
لجنة
الطب و الجراحة

شكر وعرفان

تتقدم لكم لجنة الطب والجراحة – جامعة مؤتة بهذا الملف المُعد لدراسة

امتحان الـ Mini-OSCE لمادة الباطني

والذي ساهم بإعداده نخبة من زملائكم في السنة السادسة من دفعة وتين (2014)

بإشراف وتنسيق : طارق أبولبدة & بيان عودة الله&عبدالرحمن بدير

حيث يحتوي هذا الملف على أهم النقاط التي قد تأتي في الامتحان ..

ويوجد في آخره جميع أسئلة السنوات للدفع السابقة ؛ وسيتم التحديث عليه بشكل مستمر ان شاء الله ..

- ملاحظة : آخر تعديل على هذا الملف كان بتاريخ 16/1/2024 ، حيث تمت إضافة امتحان السنة الرابعة الفصل الأول للسنة الدراسية (2023-2024)

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CVS

References

- Davidson
- Oxford
- Medstudy
- Uptodate
- John hopkins
- Step up to medicine

DONE BY :

اويس البرباوي

إبراهيم الزعبي

رغدة الرفوع

ديمة أبو محفوظ

شادن جويفل

محمود حماشا

وليد العمري

اسراء الطراونة

اشراق الحيصة

رفاه الفراجات

Infective endocarditis

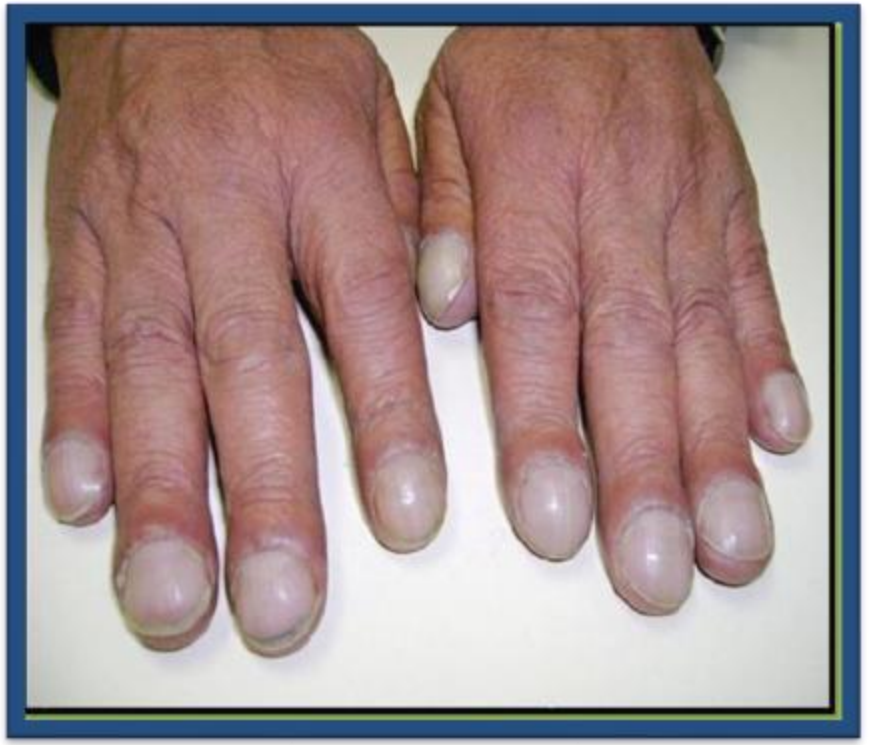
Q: A pt presented with fever, Hx of heamturia & systolic murmur at the lower left sternal border on auscultation since 8 weeks ago:

1) what is your Dx?

Subacute Infective endocarditis

2) Mention other cardiac cause for this sign?

congenital cyanotic heart disease.



Q: Patient presented with intermittent fever of 2 wks duration, he has a Hx. of dental caries & hematurea.
On P/E there was heart murmur, otherwise the exam was unremarkable!

Mention 2 tests to confirm Dx.?

1.Blood culture.

2.Echocardiography.

Give other 3 differential diagnosis ?

Trauma

Rheumatoid arthritis , SLE

VASCULITIS

Q: A patient with history of IV drug abuse presented with fever and this abnormality :

-Identify this abnormality

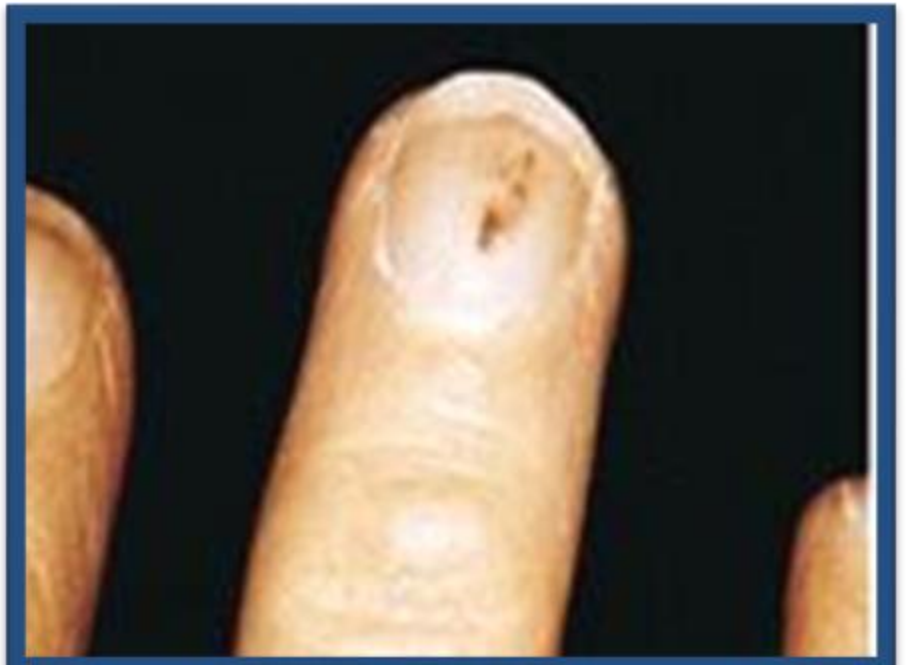
Splinter hemorrhage

-What is the most suspected diagnosis ?

Infective endocarditis

-What is the most suspected cause ?

Staph aureus infection



Q:male pt has a history of heart disease and sore throat 2 weeks ago and has murmur:

1) what is your diagnosis?

Infective endocarditis

2) Identify one of complication?

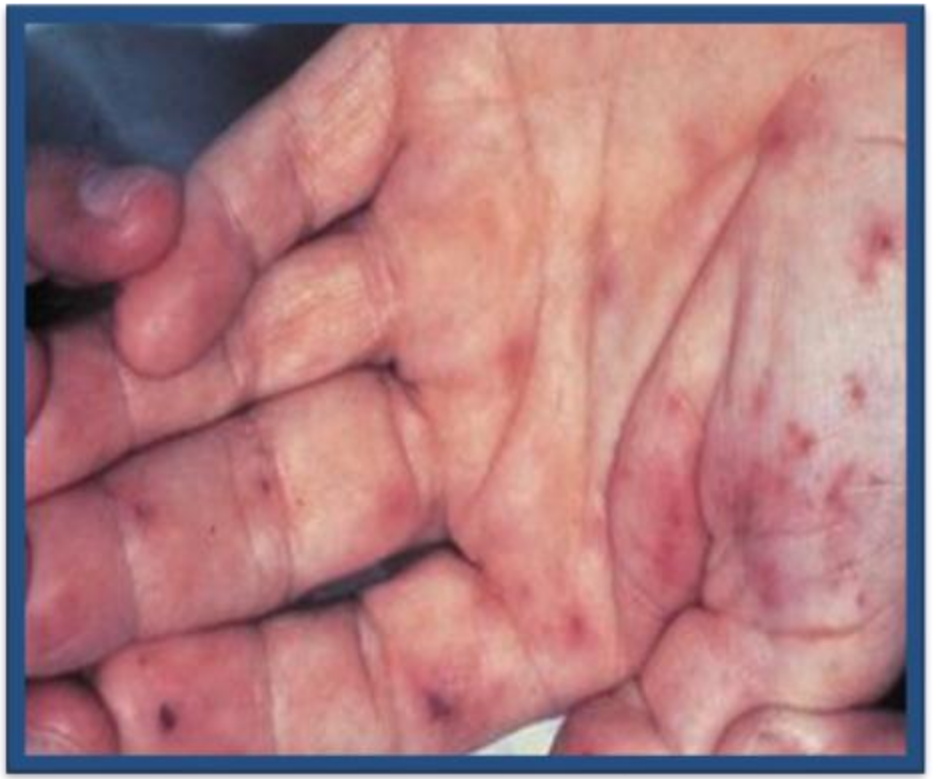
Emboli: Seen in approximately one third of patients

retinal hemorrhages

splenomegaly

Q: patient with prosthetic valves presents with prolonged fever.
Painless skin lesions seen below

- Name the skin lesions
  Jan-way lesions
- Name the underlying disease (not the same pic)
  Infective endocarditis



Q: Give 4 symptoms the patient may present with?
the patient with infective endocarditis may present with
*Fever, fatigue or failure to thrive, arthralgias
cough, chest pain, HEMATUIA
Give 3 investigations required in this case

- Blood culture (most important)
- Echocardiogram - URIN ANLYSIS
- ECG
- ESR , CRP

Give 2 ECG abnormalities you suspect in this patient

- atrioventricular block

-ST elevation (infarction)
What is the treatment ?
Extended Parenteral antibiotics
Surgery may be indicated

Q: Identify this abnormality

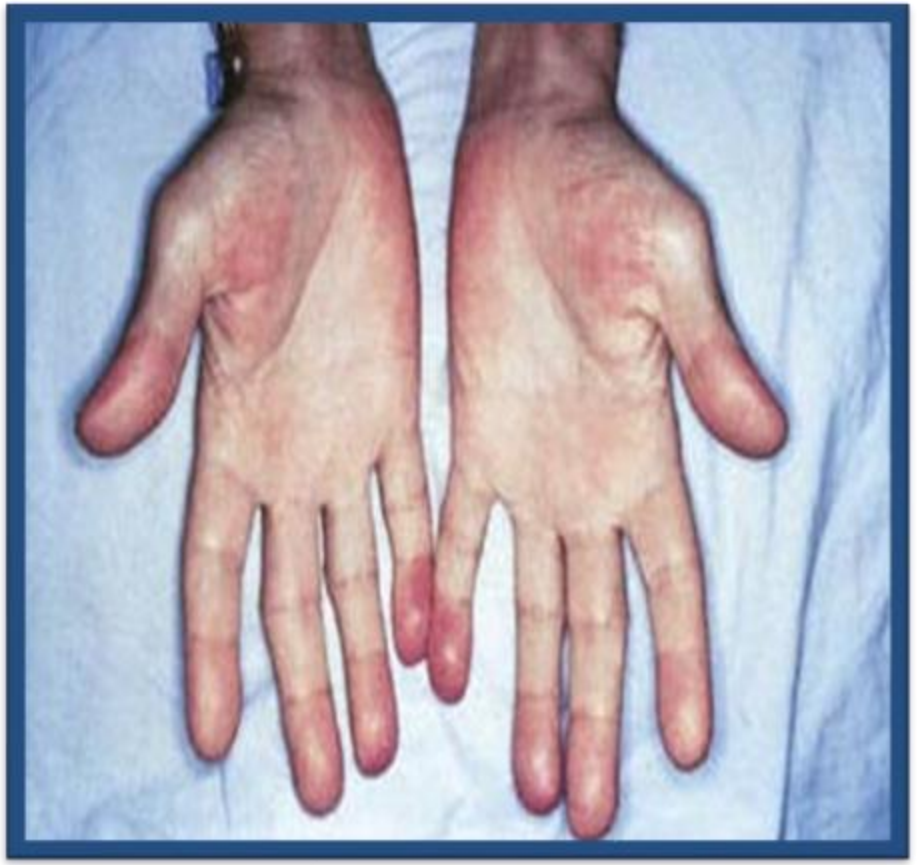
Osler's nodes

The direct cause :

vasculitis

Associated with :

Infective endocarditis

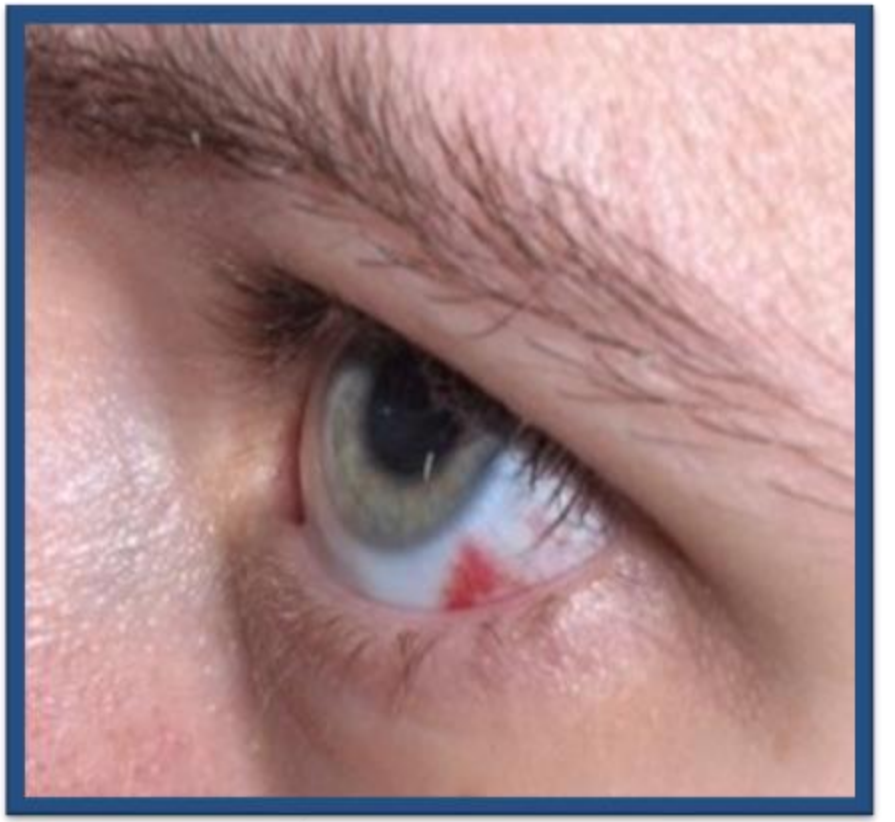


Q: Identify this abnormality

Subconjunctival hemorrhage

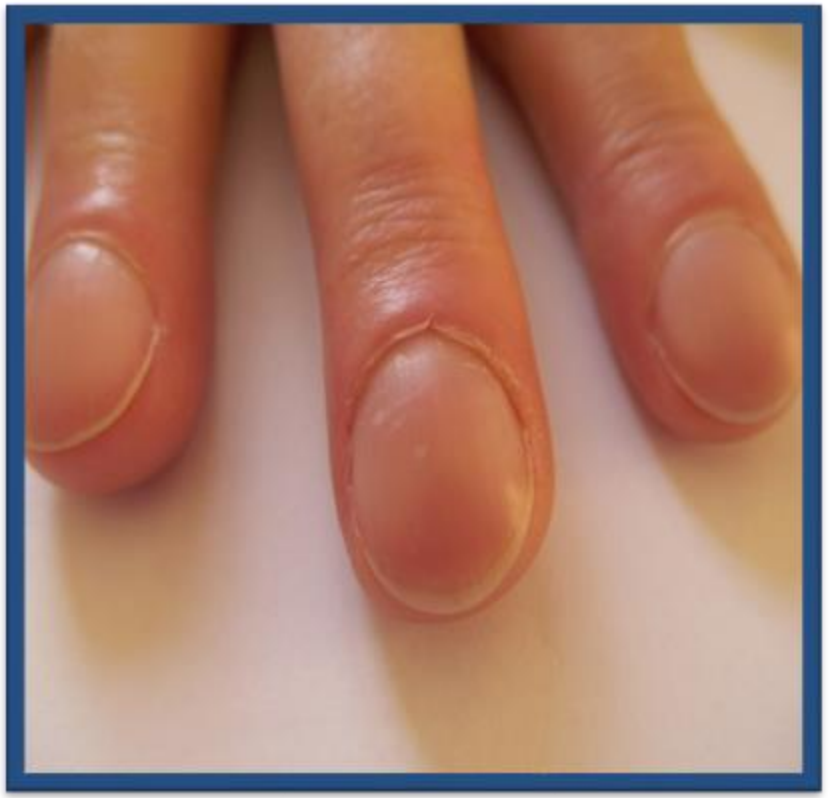
3 differential diagnosis :

- Infective endocarditis
- Trauma
- hypertension



Q: Mention 3 causes of this condition.

Congenital heart disease , cystic fibrosis ,cirrhosis, chrons and UC, lung abscess, infective endocarditis

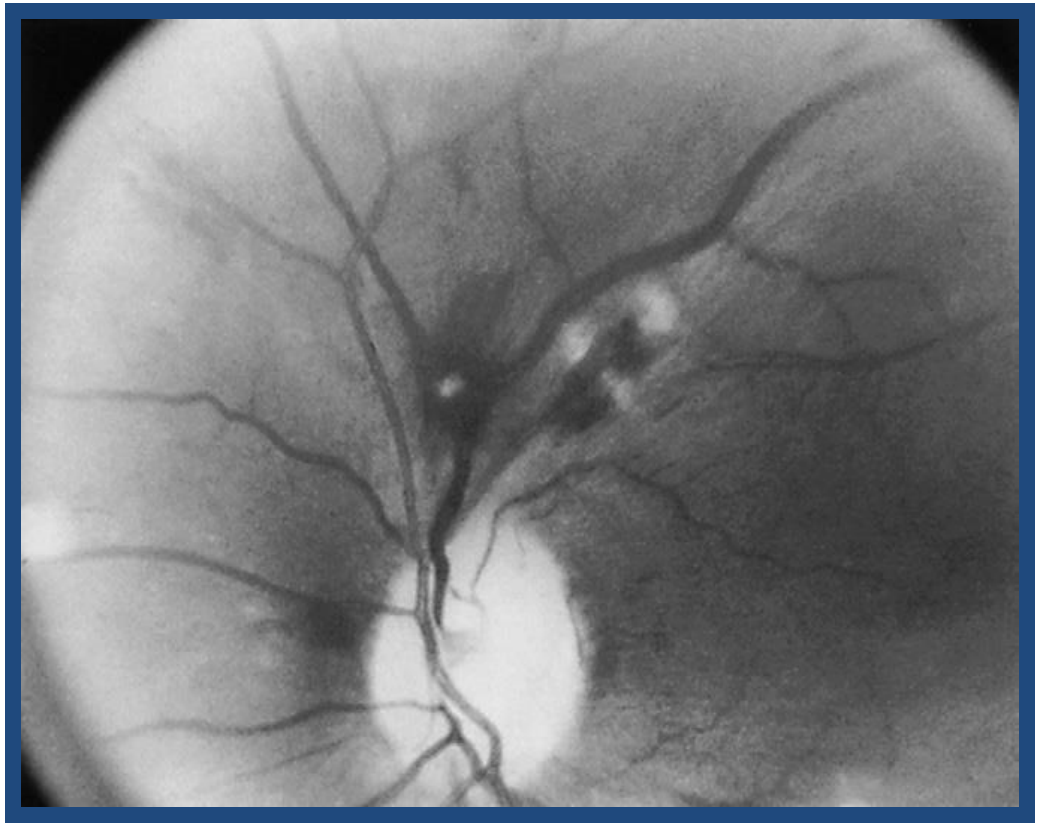


Q: Roth spots in a patient with infective endocarditis

DDX

Anima

Leukemia



A 35 year old female known case of rheumatic heart disease had a dental workup several weeks ago and is now complaining of fever, fatigue and SOB. Her physical exam reveals a murmur ……..

Some Labs were also mentioned but you won't need them to answer the question

Patient also had microscopic hematuria.

1) What is your diagnosis?

infective Endocarditis

2) Name 2 investigations to rule in your diagnosis

A. Blood Cultures B. Echocardiography (whether Transthoracic/Transesophageal it doesn't matter)

3) Name 2 physical signs seen in the patient

A. Osler's nodes B. Janeway Lesions

B. Other alternatives include: Splinter hemorrhages/Roth's spots

RISK FACTOR

- Prosthetic valve
- Congenital heart dieses
- Rheumatic heart dieses
- IV drug abuse/ *more s.auras

TABLE 16-1 *Causes of Bacterial Endocarditis*

Endocarditis Subtype	Most Common Organism	Other Common Organisms
"Typical"	Viridans streptococci Many species highly associated such as *Streptococcus mutans*	*Staphylococcus aureus* Other streptococcal species *Staphylococcus epidermidis* (uncommon) Enterococci (uncommon)
Culture-negative	No dominant organism	*Haemophilus aphrophilus* *Haemophilus parainfluenzae* *Actinobacillus actinomycetemcomitans* *Cardiobacterium hominis* *Eikenella corrodens* *Kingella kingii*
Injection drug use	*S. aureus*	*Pseudomonas aeruginosa* *Candida* Enterococci *Streptococcus viridans* *S. epidermidis* Polymicrobial
Unusual causes	No dominant organism	Fungi: *Candida, Aspergillus* *Coxiella burnetii* *Bartonella* *Chlamydia* *Legionella* *Brucella* *Mycoplasma*
Early prosthetic valve	*S. epidermidis*	*S. aureus* Gram-negative bacilli Enterococci Diphtheroids Fungi
Late prosthetic valve	*S. epidermidis*	*S. viridans* *S. aureus* Gram-negative bacilli Enterococci

**Streptococcu viridans,

4 wk penicillin or ceftriaxone

2 wk penicillin or ceftriaxone, combined with gentamicin reasonable

Staphylococcus aureus, left-sided	4 wk nafcillin combined with gentamicin for initial 3–5 days	MRSA treated with 4–6 wk daily vancomycin
S. aureus, right-sided	2 wk nafcillin combined with gentamicin	Treatment applies only to methicillin-sensitive staphylococci, with no embolic events
Prosthetic valve	6 wk therapy with penicillin derivative or vancomycin (depending on sensitivities) in combination with rifampin, plus aminoglycoside for initial 2 wk	Early surgical consultation advised Fungal infection of prosthetic valve requires surgery in most cases
Enterococcus	6 wk penicillin combined with gentamicin	If aminoglycoside resistance demonstrated, 8–12 wk penicillin indicated Other antimicrobial resistance common and should prompt consultation with infectious disease team
Fungal	Early surgery usually required	

TABLE 16-2 *Diagnostic Criteria for Endocarditis**

Major Criteria	Minor Criteria
Positive blood culture	Fever 38°C or higher
Typical microorganism of endocarditis from two separate blood cultures	Predisposing heart condition or injection drug use
Persistently positive blood cultures with a microorganism consistent with endocarditis, defined as blood cultures drawn more than 12 hr apart, or 3 of 3 positive sets of blood cultures drawn over the course of at least 1 hr	Vascular phenomena Arterial emboli Septic pulmonary infarcts Mycotic aneurysm Intracranial hemorrhage Conjunctival hemorrhage Janeway lesions
Evidence of endocardial involvement	Immunologic phenomena Glomerulonephritis Osler nodes Roth spots Rheumatoid factor
Positive echocardiogram, demonstrating oscillating intracardiac mass or abscess, or dehiscent prosthetic valve	Microbiologic evidence Positive blood cultures that do not meet major criteria
New valvular regurgitation (excludes worsening of preexisting murmur)	Echocardiographic evidence Consistent with endocarditis but not meeting major criteria

*Endocarditis is diagnosed with two major criteria, one major plus three minor criteria, or five minor criteria.

indications for surgery

- Major embolic events
- Valvular dysfunction
- Congestive heart failure
- Fungal endocarditis

Rheumatic Fever

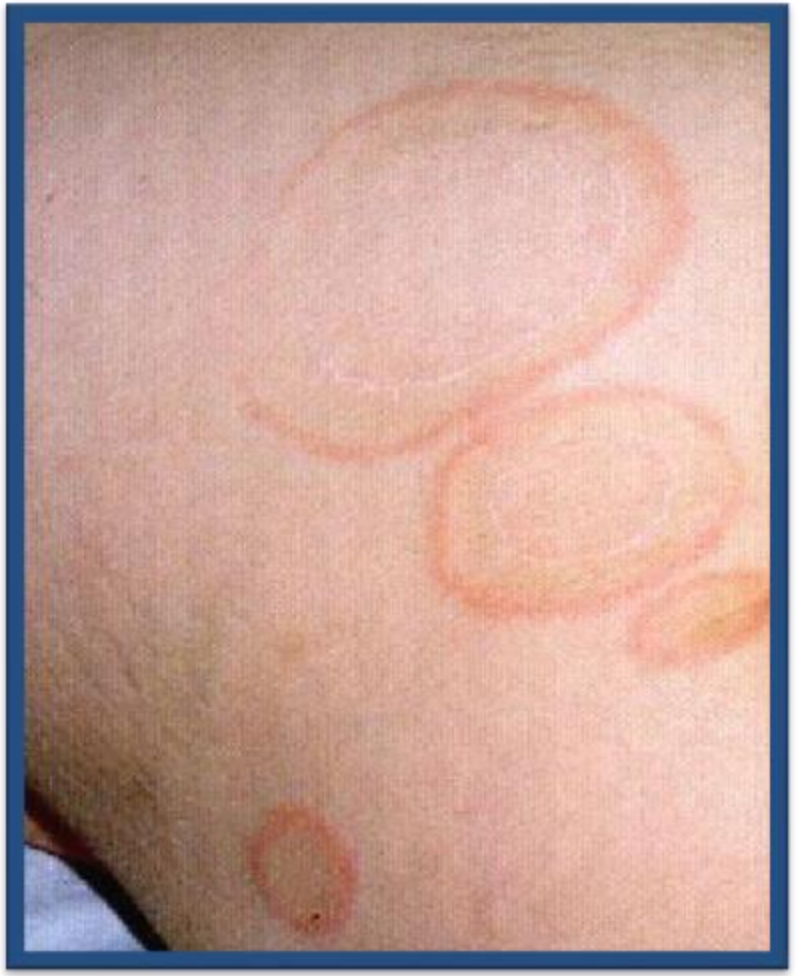
Q:

A-What is this skin lesion?

Erythema marginatum

B-What is the diagnosis?

Rheumatic fever



Q:

A young patient with recent history of upper respiratory tract infection presented this abnormality :

Identify this abnormality

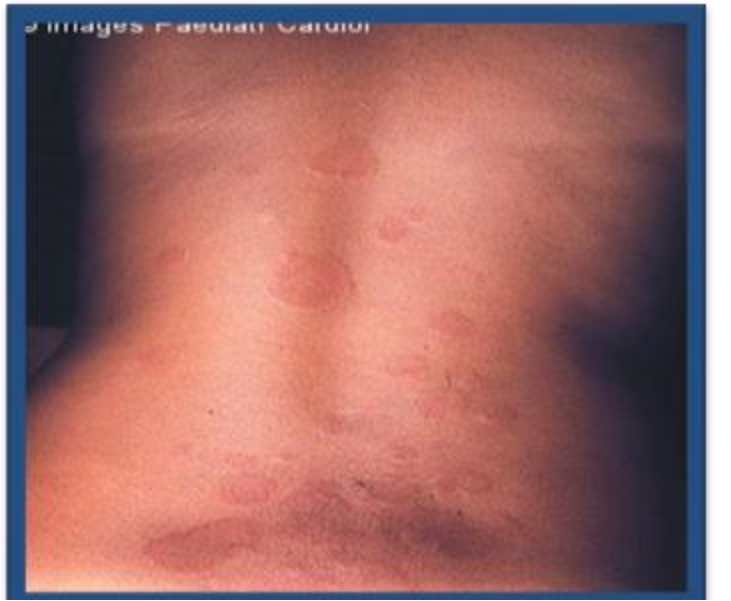
Erythema marginatum

What is the most suspected diagnosis ?

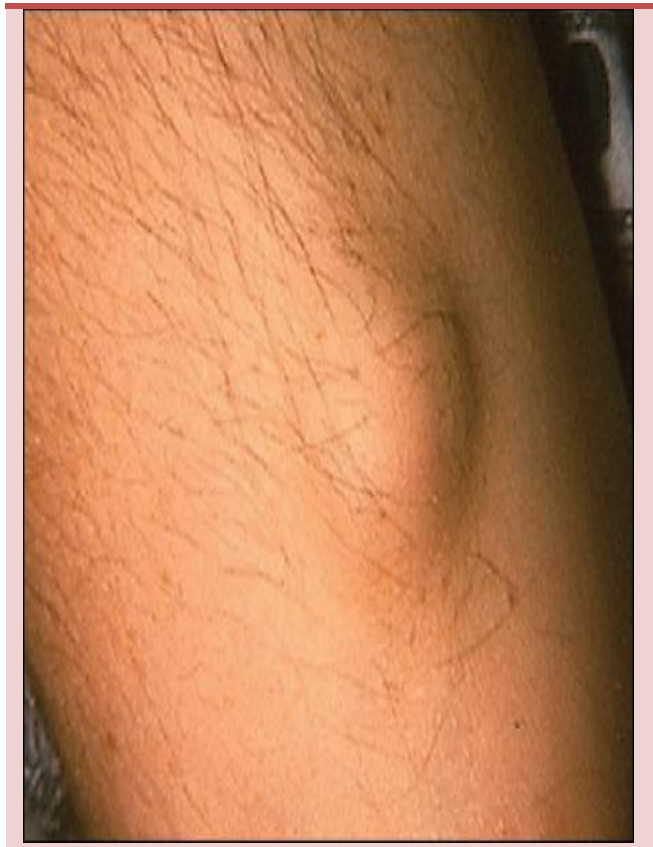
Rheumatic fever

What is the most suspected cause ?

Immune mediated delayed response to group A beta hemolytic streptococcus infection



Q:



Identify this abnormality

Subcutaneous nodules

Give 3 differential diagnosis

1- rheumatic fever
2- juvenile rheumatoid arthritis
3- neurofibromatosis

According to the ECG what's your diagnosis ?

First degree AV block

If this pt esr is elevated with migratory arthritis for 2 week duration and skin lesion as in pic

Answer the following

-your diagnosis ?

Rf

-How to confirm it ?

Aso titter

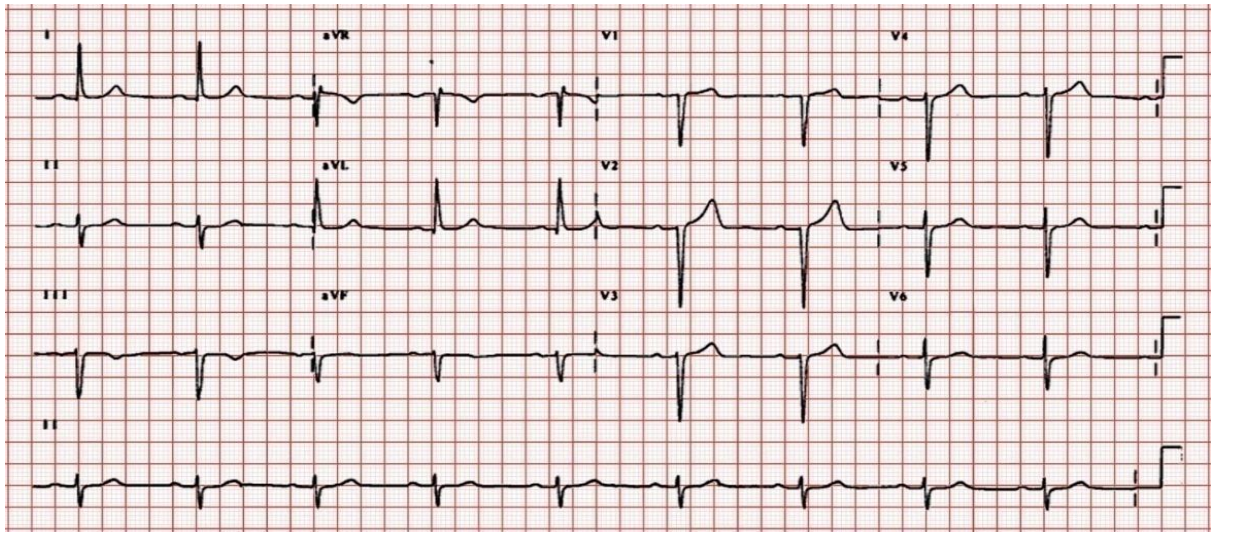
+ throat culture

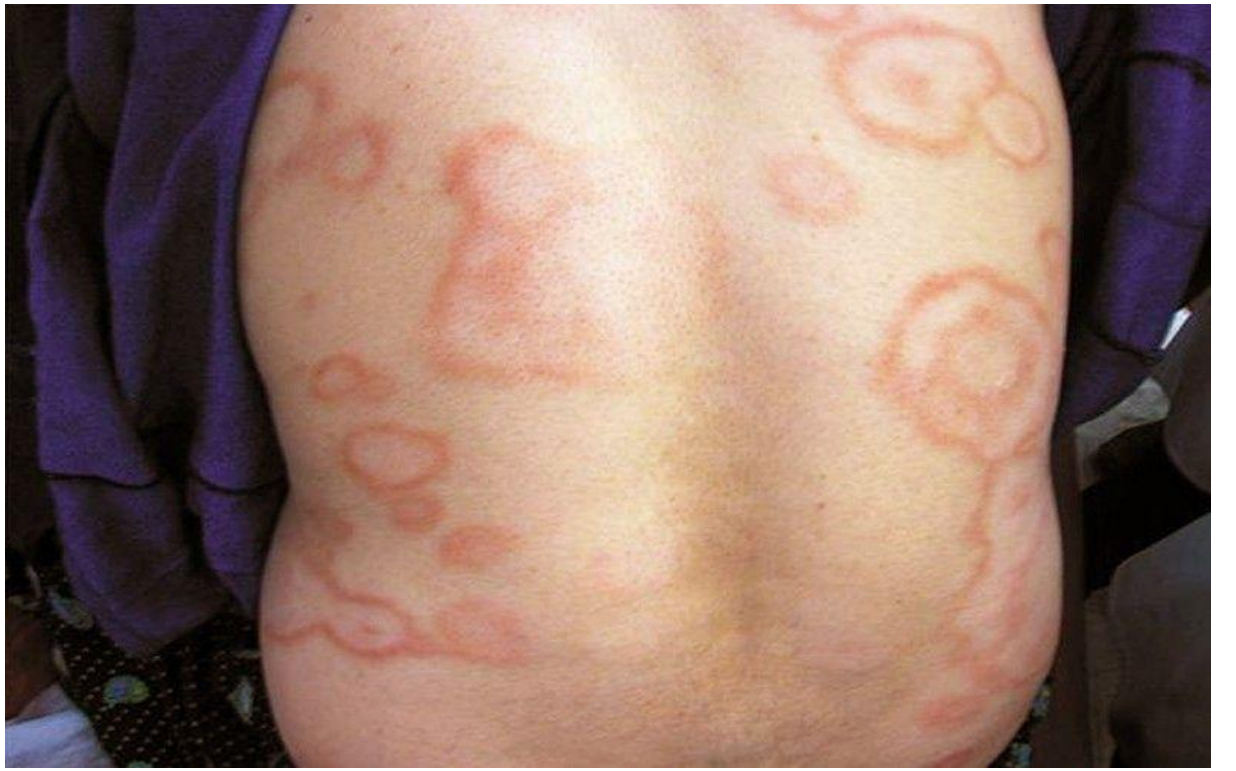
+ direct antigen test

Recent scarlet fever

-how to treat the rash ?

No treatment but may antihistamine



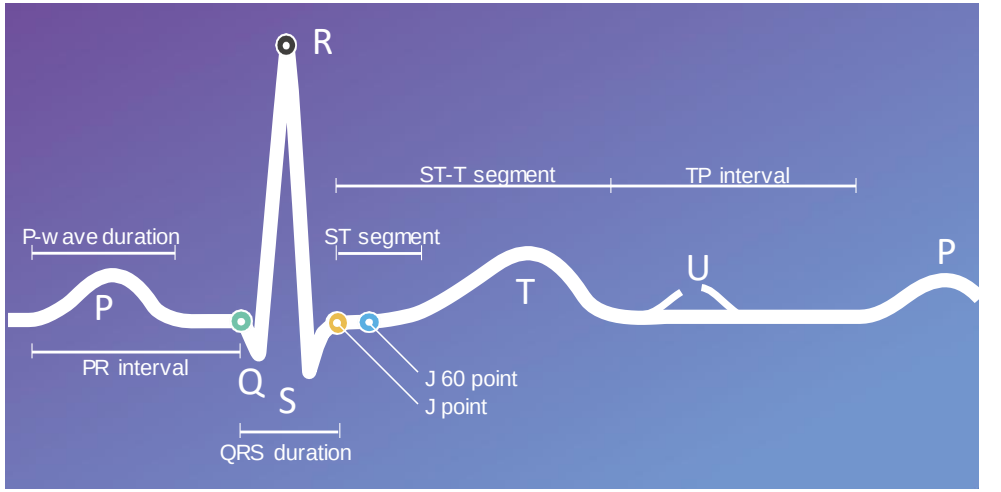


Which symptom presented is NOT a sign of rheumatic fever?
A. truncal rash
B. joint tenderness
C. changes in vision
D. nausea

Which of the following factors in the patient's history and physical is LEAST relevant to the diagnosis of Rheumatic fever?
A. sore throat from 3 weeks previous
B. living in the current conditions of Flint, MI
C. the mother's history of type-2 diabetes
D. a possible genetic factor from the father who's history is unknown

According to the Jones Criteria, which of the following sets of symptoms would indicate a positive diagnosis for Rheumatic fever? Select all that apply.
A. Carditis, fever, and an elevated WBC count
B. Positive strep throat culture, arthritis, chorea
C. Recent scarlet fever, carditis, fever, arthralgia
D. Elevated C-reactive protein, carditis, fever

ECG


R
ST-T segment
TP interval
P-w ave duration
ST segment
P
T
U
P
PR interval
Q
S
J 60 point
J point
QRS duration

ECG WAVES

Methodological ECG Interpretation

The ECG must always be interpreted systematically. Failure to perform a systematic interpretation of the ECG may be detrimental. The interpretation algorithm presented below is easy to follow and it can be carried out by anyone. The reader will gradually notice that ECG interpretation is markedly facilitated by using an algorithm, as it minimizes the risk of missing important abnormalities and also speeds up the interpretation.

1. Rhythm

ASSESSMENTS	EVALUATION
Assess ventricular (RR intervals) and atrial (PP intervals) rate and rhythm. ♥ Is ventricular rhythm regular? What is the ventricular rate (beats/min)? ♥ Is atrial rhythm regular? What is the atrial rate (beats/min)? ♥ P-waves should precede every QRS complex and the P-wave should be positive in lead II.	♥ Sinus rhythm (which is the normal rhythm) has the following characteristics: (1) heart rate 50–100 beats per minute; (2) P-wave precedes every QRS complex; (3) the P- wave is positive in lead II and (4) the PR interval is constant. ♥ Causes of bradycardia: sinus bradycardia, sinoatrial block, sinoatrial arrest/inhibition, second-degree AV block, third- degree AV block. Note that escape rhythms may arise during bradycardia. Also note that bradycardia due to dysfunction in the sinoatrial node is referred to as sinus node dysfunction (SND). If a person with ECG signs of SND is symptomatic, the condition is classified as sick sinus syndrome (SSS). ♥ Causes of tachycardia (tachyarrhythmia) with narrow QRS complexes (QRS duration <0,12 s): sinus tachycardia, inappropriate sinus tachycardia, sinoatrial re-entry tachycardia, atrial fibrillation, atrial flutter, atrial tachycardia, multifocal atrial tachycardia, AVNRT, AVRT (pre-excitation, WPW). Note that narrow complex tachyarrhythmia rarely causes circulatory compromise or collapse. ♥ Causes of tachycardia (tachyarrhythmia) with wide QRS complexes (QRS duration ≥0,12 s): ventricular tachycardia is the most common cause and it is potentially life-threatening. Note that 10% of wide complex tachycardias actually originate from the atria but the QRS complexes become wide due to abnormal ventricular depolarization (e.g sinus tachycardia with simultaneous left bundle branch block).

2. P-wave and PR interval

ASSESSMENTS	EVALUATION
♥ P-wave always positive in lead II (actually always positive in leads II, III and aVF). ♥ P-wave duration should be <0,12 s (all leads).	♥ P-wave must be positive in lead II, otherwise the rhythm cannot be sinus rhythm. ♥ P-wave may be biphasic (diphasic) in V1 (the negative deflection should be <1 mm). It may have a prominent second hump in the inferior limb leads (particularly lead II).
♥ P-wave amplitude should be ≤2,5 mm (all leads). PR interval must be 0,12–0,22 s (all leads).	♥ P mitrale: increased P-wave duration, enhanced second hump in lead II and enhanced negative deflection in V1. ♥ P pulmonale: increased P-wave amplitudes in lead II and V1. ♥ If P-wave not clearly visible: look for retrograde (inverted) P-waves, which can be located anywhere between the J point and the terminal part of the T-wave. ♥ PR interval >0,22 s: first-degree AV block. ♥ PR interval <0,12 s: Pre-excitation (WPW syndrome). ♥ Second-degree AV-block Mobitz type I (Wenckebach block): repeated cycles of gradually increasing PR interval until an atrial impulse (P-wave) is blocked in the atrioventricular node and the QRS complex does not appear. ♥ Second-degree AV-block Mobitz type II: intermittently blocked atrial impulses (no QRS seen after P) but with constant PR interval. ♥ Third-degree AV-block: All atrial impulses (P-waves) are blocked by the atrioventricular node. An escape rhythm arises (cardiac arrest ensues otherwise), which may have narrow or wide QRS complexes, depending on its origin. There is no relation between P-waves and the escape rhythm's QRS complexes, and atrial rhythm is typically faster than the escape rhythm (both rhythms are typically regular).

3. QRS complex

ASSESSMENTS	EVALUATION
♥ QRS duration must be <0,12 s (normally 0,07-0,10 s). ♥ There must be at least one limb lead with R-wave amplitude >5 mm and at least one chest (precordial) lead with R-wave amplitude >10 mm; otherwise there is low voltage. ♥ High voltage exists if the amplitudes are too high, i.e if the following condition is satisfied: S- waveV1 or V2 + R-waveV5 >35 mm. ♥ Look for pathological Q-waves. Pathological Q-waves are ≥0,03 s and/or amplitude ≥25% of R-wave amplitude in same lead, in at least 2 anatomically contiguous leads. ♥ Is the R-wave progression in the chest leads (V1–V6) normal?	♥ **Wide QRS complex (QRS duration ≥0.12 s)**: Left bundle branch block. Right bundle branch block. Nonspecific intraventricular conduction disturbance. Hyperkalemia. Class I antiarrhythmic drugs. Tricyclic antidepressants. Ventricular rhythms and ventricular extrasystoles (premature complexes). Artificial pacemaker which stimulates in the ventricle. Aberrant conduction (abberancy). Pre-excitation (Wolff-Parkinson-White syndrome). ♥ **Short QRS duration**: no clinical relevance. ♥ **High voltage**: Hypertrophy (any lead). Left bundle branch block (leads V5, V6, I, aVL). Right bundle branch block (V1– V3). Normal variant in younger, well-trained and slender individuals. ♥ **Low voltage**: Normal variant. Misplaced leads. Cardiomyopathy. Chronic obstructive pulmonary disease. Perimyocarditis. Hypothyreosis (typically accompanied by bradycardia). Pneumothorax. Extensive myocardial infarction. Obesity. Pericardial effusion. Pleural effusion. Amyloidosis. ♥ **Pathological Q-waves**: Myocardial infarction. Left-sided pneumothorax. Dextrocadia. Perimyocarditis. Cardiomyopathy. Amyloidosis. Bundle branch blocks. Anterior

♥ Is the electrical axis normal? Electrical axis is assessed in limb leads and should be between –30°
to 90°.

fascicular block. Pre-excitation. Ventricular hypertrophy.Acute cor pulmonale. Myxoma.

♥ **Fragmented QRS complexes** indicates myocardial scarring (mostly due to infarction).

♥ **Abnormal R-wave progression**: Myocardial infarction. Right ventricular hypertrophy (reversed R-wave progression). Left ventricular hypertrophy (amplified R-wave progression). Cardiomyopathy. Chronic cor pulmonale. Left bundle branch block. Pre-excitation.

♥ **Dominant R-wave in V1/V2**: Misplaced chest electrodes. Normal variant. Situs inversus. Posterolateral infarction/ischemia (if patient experiences chest discomfort). Right ventricular hypertrophy. Hypertrophic cardiomyopathy. Right bundle branch block. Pre-excitation.

♥ **Right axis deviation:** Normal in newborns. Right ventricular hypertrophy. Acute cor pulmonale (pulmonary embolism).
Chronic cor pulmonale (COPD, pulmonary hypertension, pulmonary valve stenosis). Lateral ventricular infarction. Pre- excitation. Switched arm electrodes (negative P and QRS-T in lead I). Situs inversus. Left posterior fascicular block is diagnosed when the axis is between 90° and 180° with rS complex in I and aVL as well as qR complex in III and aVF (with QRS duration <0.12 seconds), provided that other causes of right axis deviation have been excluded.

♥ **Left axis deviation**: Left bundle branch block. Left ventricular hypertrophy. Inferior infarction. Pre-excitation. Left anterior fascicular block is diagnosed if the axis is between -45° and 90° with qR-complex in aVL and QRS duration is 0,12 s, provided that other causes of left axis deviation have been excluded.

♥ **Extreme axis deviation**: Rarely seen. Probably misplaced electrodes. If the rhythm is wide QRS complex tachycardia, then the cause is probably ventricular tachycardia.

4. ST segment

ASSESSMENTS	EVALUATION
♥ The ST-segment should be flat and isoelectric (in level with the baseline). It may be slightly upsloping at the transition with the T-wave. ♥ ST segment deviation (elevation and depression)is measured in the J point.	♥ **Benign ST segment elevation** is very common in the population, particularly in the precordial leads (V2–V6). Up to 90% (in some age-ranges) of healthy men and women display concave ST-segment elevations in V2–V6 (this is called male/female pattern). ST-segment elevations which are not benign nor due to ischemia are rather common (listed below). ♥ ST-segment depression is uncommon among healthy individuals. ST-segment depression is particularly suspiciousin the chest leads. Guidelines recommend that <0.5 mm ST- segment depression be accepted in all leads.

♥ **Causes of ST-segment elevation**: Ischemia. ST segment elevation myocardial infarction (STEMI/STE-AKS). Prinzmetal's angina (coronary vasospasm). Male/female pattern. Early repolarization. Perimyocarditis. Left bundle branch block. Nonspecific intraventricular conduction disturbance. Left ventricular hypertrophy. Brugada syndrome. Takotsubo cardiomyopathy. Hyperkalemia. Post cardioversion. Pulmonary embolism. Pre-excitation. Aortic dissection engaging the coronary arteries. Left ventricular aneurysm.

♥ **Causes of ST-segment depression**: Ischemia. Non-ST segment elevation myocadial infarction (NSTEMI/NSTE-AKS). Physiological ST-segment depression. Hyperventilation. Hypokalemia. High sympathethic tone. Digoxin. Left bundle branch block. Right bundle branch block. Pre-excitation. Left ventricular hypertrophy. Right ventricular hypertrophy. Heart failure. Tachycardia.

♥ **Causes of waves/deflections in the J point (J wave syndromes)**: Brugada syndrome. Early repolarization.

5. T-wave

ASSESSMENTS	EVALUATION
♥ Should be concordant with the QRS complex. Should be positive in most leads. ♥ T-wave progression should be normal in chest leads. ♥ In limb leads the amplitude is highest in lead II, and in the chest leads the amplitude is highest in V2–V3.	♥ **Normal variants**: An isolated (single) T-wave inversion is accepted in lead V1 and lead III. In some instances the T-wave inversions from childhood may persist in V1–V3(V4), which is called *persistent juvenile T-wave pattern*. Rarely, all T-waves remain inverted, which is called *global idiopathic T-wave inversion* (V1–V6). ♥ **T-wave inversion without simultaneous ST-segment deviation**: This is not a sign of ongoing ischemia, but may be post-ischemic. One type of post-ischemic T-wave inversion is especially acute, namely Wellen's syndrome (characterized by deep T-wave inversions in V1–V6 in patient with recent episodes of chest pain). Cerebrovascular insult (bleeding). Pulmonary embolism. Perimyocarditis (after normalization ofthe ST-segment elevation, T-waves become inverted in perimyocarditis). Cardiomyopathy. ♥ **T-wave inversion with simultaneous ST-segment deviation**: acute (ongoing) myocardial ischaemia. ♥ **High T-waves:** Normal variant. Early repolarization. Hyperkalemia. Left ventricular hypertrophy. Left bundle branch block. Occasionally perimyocarditis. High (hyperacute) T-waves may be seen in the very early phase of STEMI.

6. QTc interval and U-wave

ASSESSMENTS	EVALUATION
♥ QTc duration men ≤0,45 s. ♥ QTc duration women ≤0,46 s. ♥ Prolonged QTc duration may cause malignant arrhythmias (torsade de pointes, which is a type of ventricular tachycardia). ♥ Shortened QTc duration (≤0.32 s) is rare, but may also cause malignant ventricular arrhythmias. ♥ The U-wave is seen occasionally, especially in well- trained individuals, and during low heart rate. It is largest in V3– V4. Amplitude is one fourth of T- wave amplitude.	♥ **Acquired QT prolongation:** anti arrhythmic drugs (procainamide, disopyramide, amiodarone, sotalol), psychiatric medications (tricyclic antidepressants, SSRI, lithium etc); antibiotics (macrolides, kinolones, atovaquone, klorokine, amantadin, foscarnet, atazanavir); hypokalemia, hypocalcemia, hypomagnesemia; cerebrovascular insult (bleeding); myocardial ischemia; cardiomyopathy; bradycardia; hypothyroidism; hypothermia. A complete list of drugs causing QT prolongation can be found here. ♥ **Congenital QT prolongation:** genetic disease of which there are approximately 15 variants. ♥ **Short QTc syndrome (≤ 0,32 s):** caused by hyperkalcemia and digoxin treatment. May cause malignant ventricular arrhythmia. ♥ **Negative U-wave:** high specificity for heart disease (including ischemia).

7. Compare with earlier ECG tracings

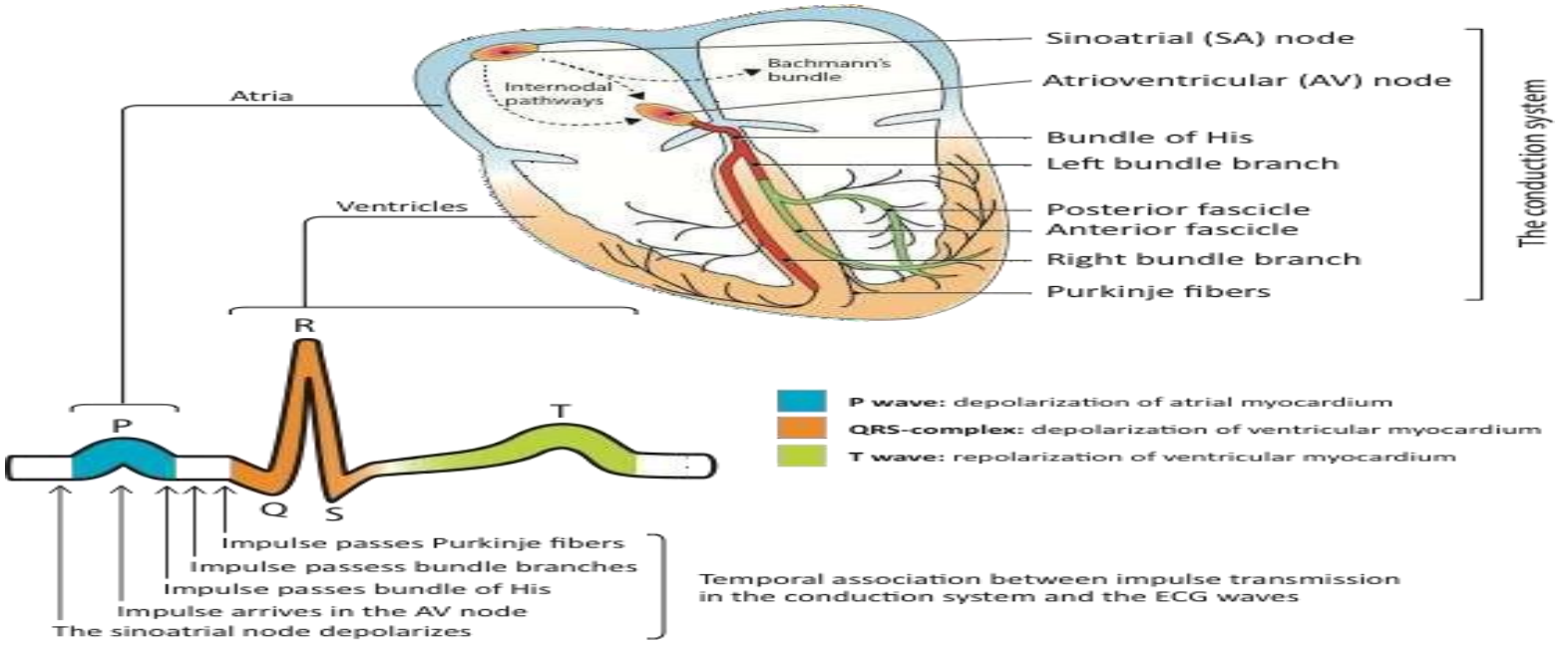
It is fundamental to compare the current ECG with previous recordings. All changes are of interest and may indicate pathology.

8. Clinical context

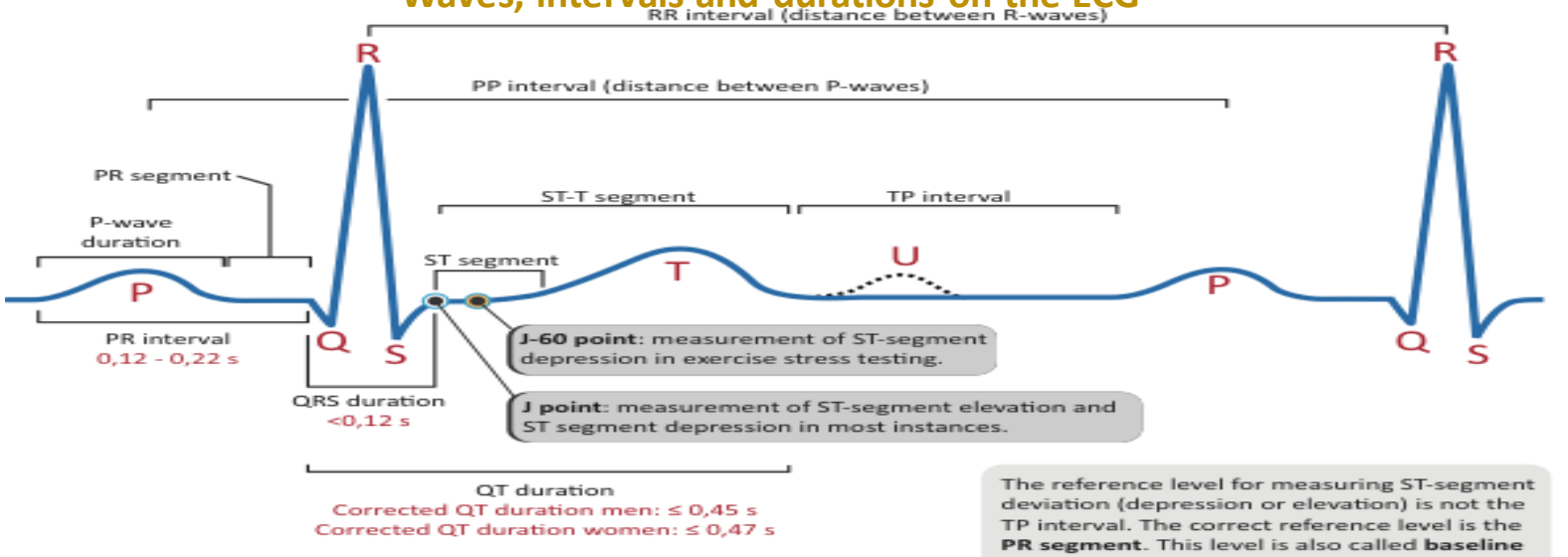
ECG changes should be put into a clinical context. For example, ST-segment elevations are common in the population and should not raise suspicion of myocardial ischemia if the patient do not have symptoms suggestive of ischemia.

The guide continues on the next page.

The cardiac conduction system

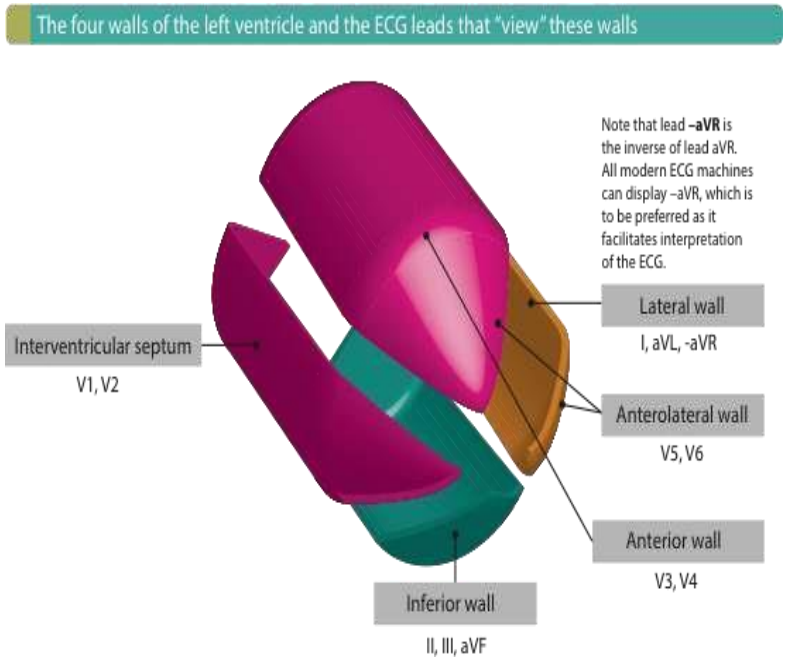


Waves, intervals and durations on the ECG

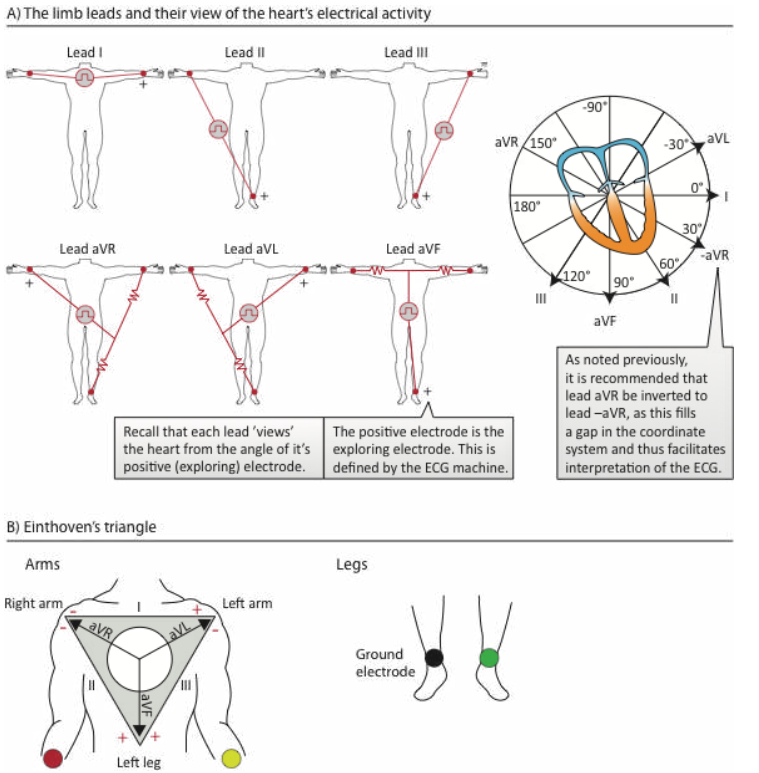


The reference level for measuring ST-segment deviation (depression or elevation) is not the TP interval. The correct reference level is the **PR segment**. This level is also called **baseline** level or **isoelectric level**.

The walls of the left ventricle and the leads that view these walls

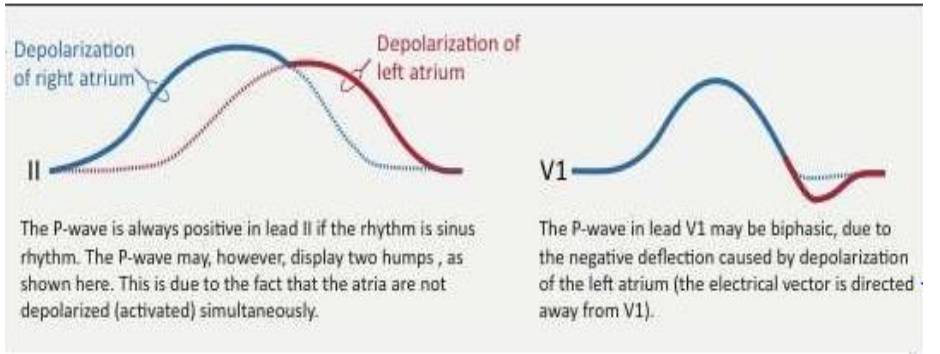


The ECG leads



P-wave changes

Contour of the normal P wave



The P-wave is always positive in lead II if the rhythm is sinus rhythm. The P-wave may, however, display two humps , as shown here. This is due to the fact that the atria are not depolarized (activated) simultaneously.

The P-wave in lead V1 may be biphasic, due to the negative deflection caused by depolarization of the left atrium (the electrical vector is directed away from V1).

Abnormal P-waves

P-mitrale P mitrale is a consequence of left atrial enlargement (often caused by mitral stenosis). Enlargement of the left atrium amplifies its contribution to the contour of the P wave.

Enhanced second hump in lead II.

Enhanced negative deflection in V1.

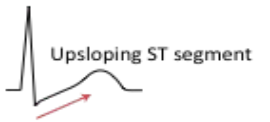
P-pulmonale P pulmonale is a consequence of right atrial enlargement. This is often a consequence of pulmonary valve stenosis or increased resistance in the pulmonary circulation. Enlargement of the right atrium causes an increased P wave amplitude in both leads.

Increased P wave amplitude.

Increased P wave amplitude.

ST segment depressions

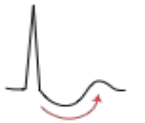
A Physiological ST-segment depressions



Upsloping ST-segment depression is a normal finding during physical exercise. It should be considered a normal finding, provided that T-waves are not inverted. Hyperventilation may cause similar ST-segment depressions.

B Non specific ST-segment depression

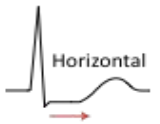
Hypokalemia and high sympathetic tone causes ST-segment depressions with flat T-waves and more marked U-waves. High sympathetic tone also causes tachycardia.



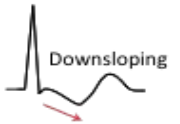
Digoxin (a drug used to treat atrial fibrillation and some caess of heart failure) causes a curved ST-segment depressions.

C ST-segment depressions caused by acute ischemia

Characteristics

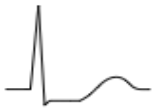


Very typical of ischemia.

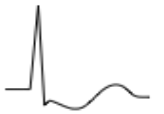


Typical of ischemia.

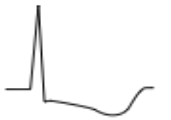
Real life examples



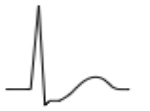
Horizontal depression with distinct ST-segment.



Downsloping with positive T-wave



Downsloping with inverted T-wave



Horizontal depression with short ST-segment.

Note

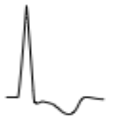
When considering myocardial ischemia, deviations in the ST-segment always indicates ongoing ischemia. ST-segment deviation may be accompanied by T-wave changes, but it is the ST-deviation that indicates acute ischemia.

de Winter's sign

de Winter's sign is an exception to the rule that upsloping ST-segment depressions are not ischemic. de Winter's sign implies the presence of upsloping ST-segment depressions with prominent T-waves in the majority of the precordial (chest) leads. This is a sign of acute ischemia, most often caused by a proximal occlusion of the left anterior descending (LAD) artery.

D Secondary repolarization abnormalities (secondary ST- and T-wave changes)

Left bundle branch block (lead V6)



Left ventricular hypertrophy (lead V6)

Right bundle branch block (lead V1)

Pre-excitation (delta wave)

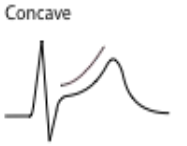
Right ventricular hypertrophy Large R-waves and ST-segment depressions in V1–V3. In case of chest discomfort, one must consider possibility of postero-lateral transmural ischemia as a differential diagnosis.

ST segment elevations

A Characteristics of ST-segment elevations caused by ischemia

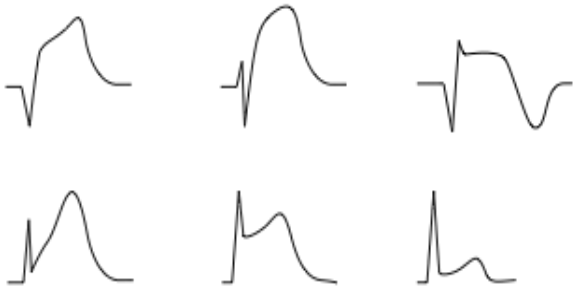
ST-segment elevations caused by ischemia typically displays a convex or straight ST-segment. Such ST-segment elevations in presence of chest discomfort are strongly suggestive of transmural myocardial ischemia. Note that the straight downsloping variant is unusual.

B Typical non-ischemic ST-segment elevation



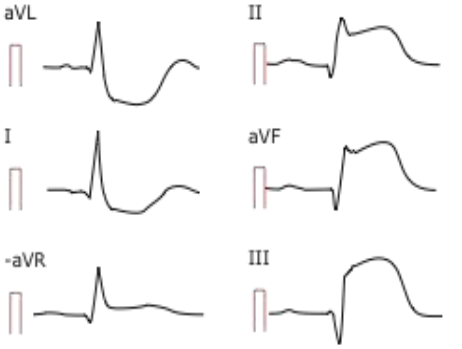
Non-ischemic ST-segment elevations are extremely common in all populations. They are characterized by a concave ST-segment and a greater distance between the J point and the T wave apex.

C Examples of ST-segment elevations caused by ischemia



ST-segment elevation can vary markedly in appearance. These six examples were retrieved from six different patients with STEMI.

D Real life example (limb leads shown)



ECG from a male patient (age 61) who experienced chest pain while driving to work. Note ST-segment elevations as well as reciprocal ST-segment depressions. There are also pathological Q-waves (leads III, aVF and perhaps II).

T-wave changes

A Normal T-waves

Normal T wave
Smooth transition from ST-segment to T wave. T wave is slightly asymmetric with a steeper downslope.

Normal variant
Large, asymmetric T wave with broad base. Often in conjunction with slight J point elevation in leads V2-V4.

B Large T-waves

Hyperkalemia
Large, symmetric, pointed with short base.

Hyperacute T wave
can be seen in transmural ischemia. High, broad based, symmetric, not pointed. Almost always seen in conjunction with ST-segment elevation.

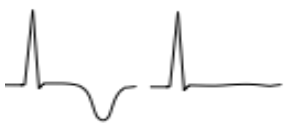
C Biphasic (diphasic) T-waves

Both these T waves are negative (inverted) since the terminal portions are negative.

This T wave is positive by definition since the terminal portion is positive.

Whenever spotting a biphasic T wave, try to determine whether it is actually a positive or negative (inverted) T-wave by viewing the terminal portion of the T wave.

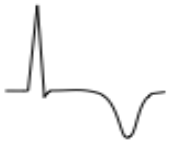
D Negative (inverted) T-waves



Post-ischemic
Symmetric T wave, with varying depth. Ranges from flat T wave to very deep T wave inversion. Inverted T waves do not equate acute (ongoing) ischemia, but rather appear after an episode of ischemia!

Acute (ongoing) ischemia
T wave inversion with simultaneous ST-segment deviation (most commonly ST-depression). Note that it is the ST-segment deviation that represents the acute ischemia!

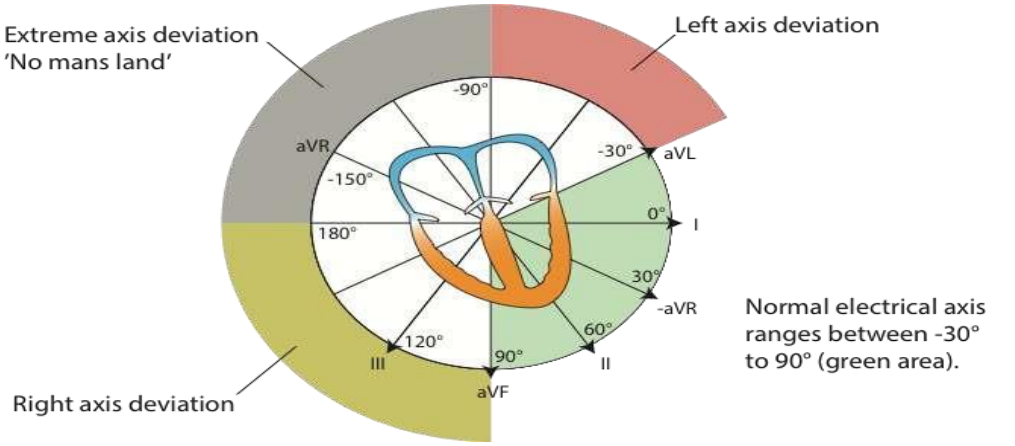
Cerebrovascular insult pattern
Very deep (gigantic) T wave inversions in the chest leads. Some studies report this finding in up to 30% of patients with intracerebral hemorrhage.



Hypertrophic cardiomyopathy
Symmetric T wave inversions, most comonly in V1–V3. Often very deep and accompanied by large R waves. Occasionally accompanied by ST-segment depression.

PERIMYOKARDIT
T wave inversions occur after normalization of ST-segment elevations in perimyocarditis. T wave inversions often seen in most leads.

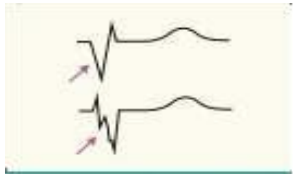
Electrical axis of the heart



As evident from the figure above, the normal heart axis is between –30° and 90°. If the axis is more positive than 90° it is referred to as right axis deviation. If the axis is more negative than –30° it is referred to as left axis deviation. The axis is calculated (to the nearest degree) by the ECG machine. The axis can also be approximated manually by judging the net direction of the QRS complex in leads I and II. The following rules apply:

- Normal axis: Net positive QRS complex in leads I and II.
- Right axis deviation: Net negative QRS complex in lead I but positive in lead II.
- Left axis deviation: Net positive QRS complex in lead I but negative in lead II.
- Extreme axis deviation (–90°to 180°): Net negative QRS complex in leads I and II.

Pro-arrhythmic ECG changes during sinus rhythm

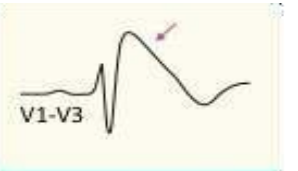


Q-waves or fragmented QRS complexes

Evidence of previous myocardial infarction and high risk of ventricular tachcyardia and ventricular fibrillation.

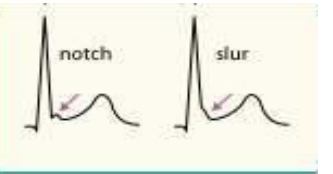
Delta wave, pre-excitation

Risk of AVRT.
May be accompanied by atrial fibrillation (pre-excited atrial fibrillation).



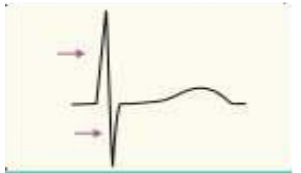
Brugada syndrome: ST elevation V1–V4. Shark tail appearance or saddle formed

Risk of ventricular tachycardia and ventricular fibrillation



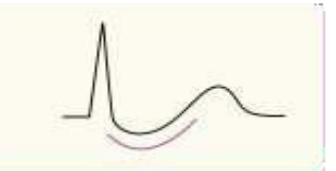
Early repolarization: slurring or notching at end-QRS

5 times increased risk of ventricular tachycardia and ventricular fibrillation.



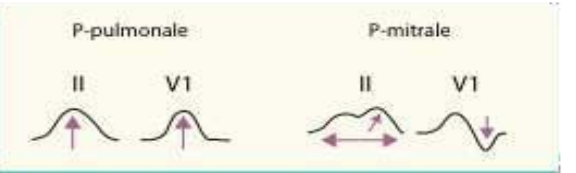
Hypertrophic cardio-myopathy: deep S, large R

Risk of ventricular tachycardia and ventricular fibrillation



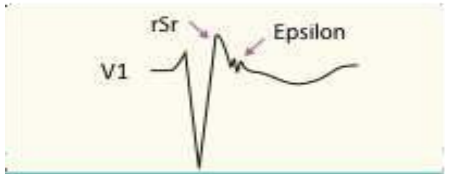
Digoxin effect: ST depression with "sagging" appearance

Digoxin may cause virtually all known arrhythmias



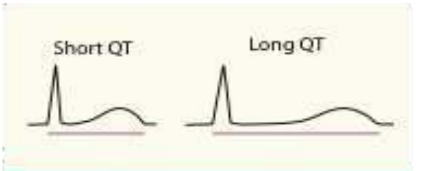
P-pulmonale (right atrial abnormality) and P-mitrale (left atrial abnormality)

Associated with atrial tachyarrhythmias.



Arrhythmogenic right ventricular cardiomyopathy: rSr-pattern in V1 with epsilon wave after QRS

Risk of ventricular tachycardia and ventricular fibrillation



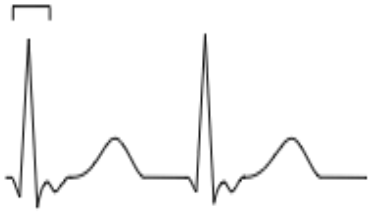
Long QT interval (LQTS) or Short QT interval (SQTS)

Long QT syndrome is fairly common and poses risk of polymorphic ventricular tachycardia referred to as torsade de pointes. Short QT syndrome is very rare but may also cause torsade de pointes.

Assessment of RP interval for tachyarrhythmias

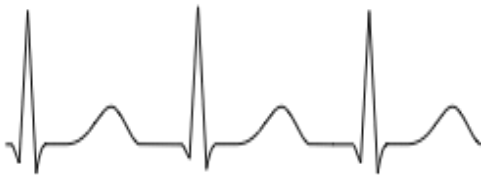
RP interval Short and <70 ms

Typical AVNRT. AVRT is unusual.



RP interval No visible P-wave

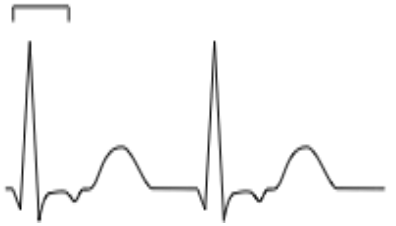
Typical AVNRT



If the P-wave is invisible, it is classified as short RP interval.

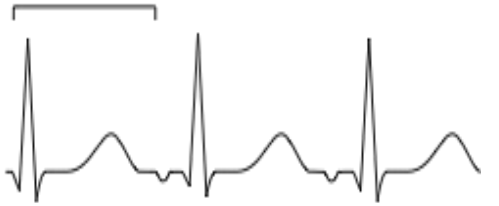
RP interval Short but >70 ms

In most cases AVRT. Occasionally atypical AVNRT or AT.

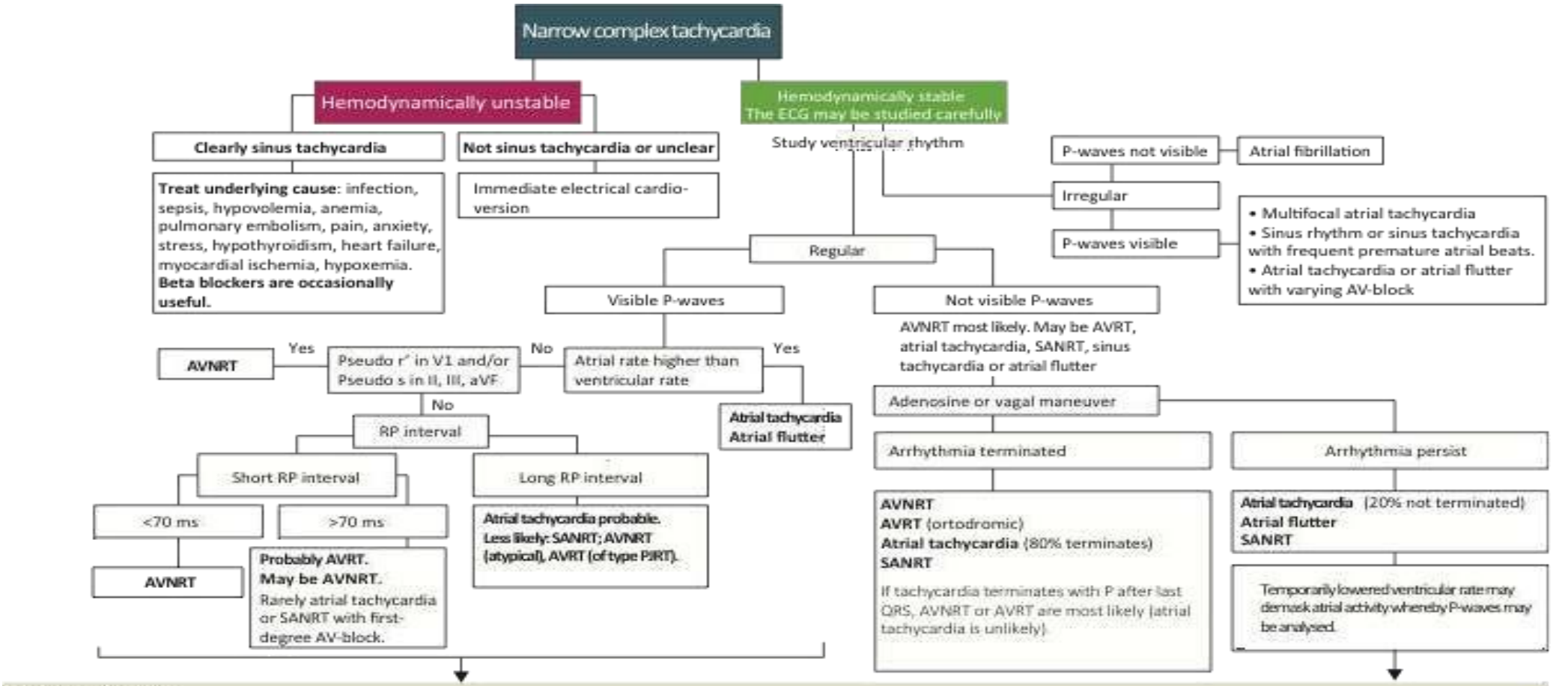


RP interval Long

In most cases AT. Occassionally atypical AVNRT. Rarely PJRT.



Diagnosis and management of tachyarrhythmias with narrow QRS complex

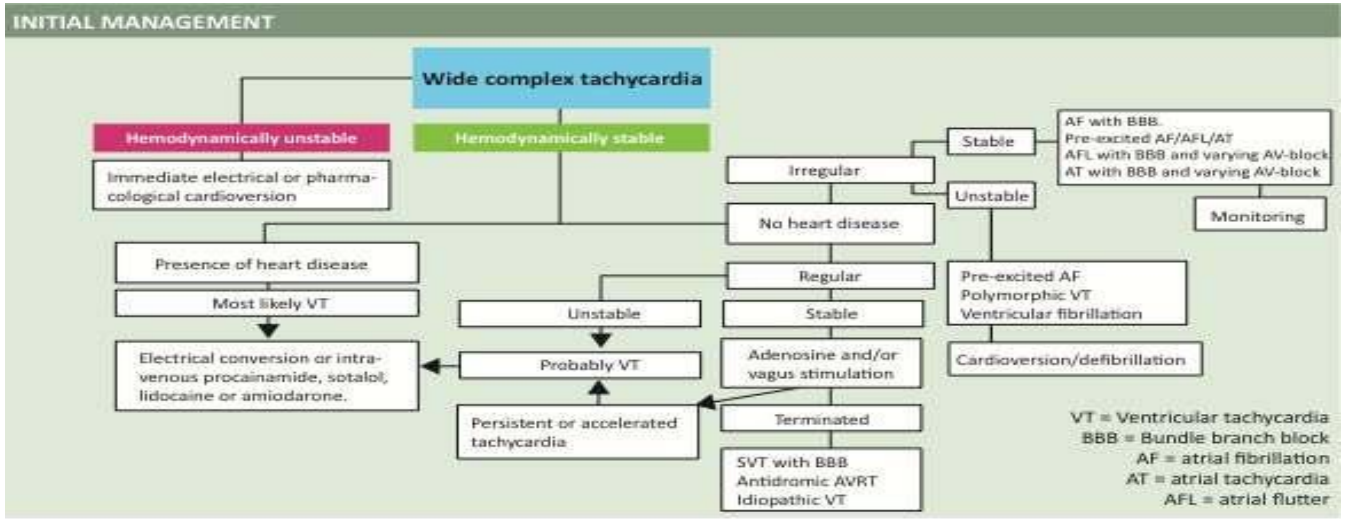


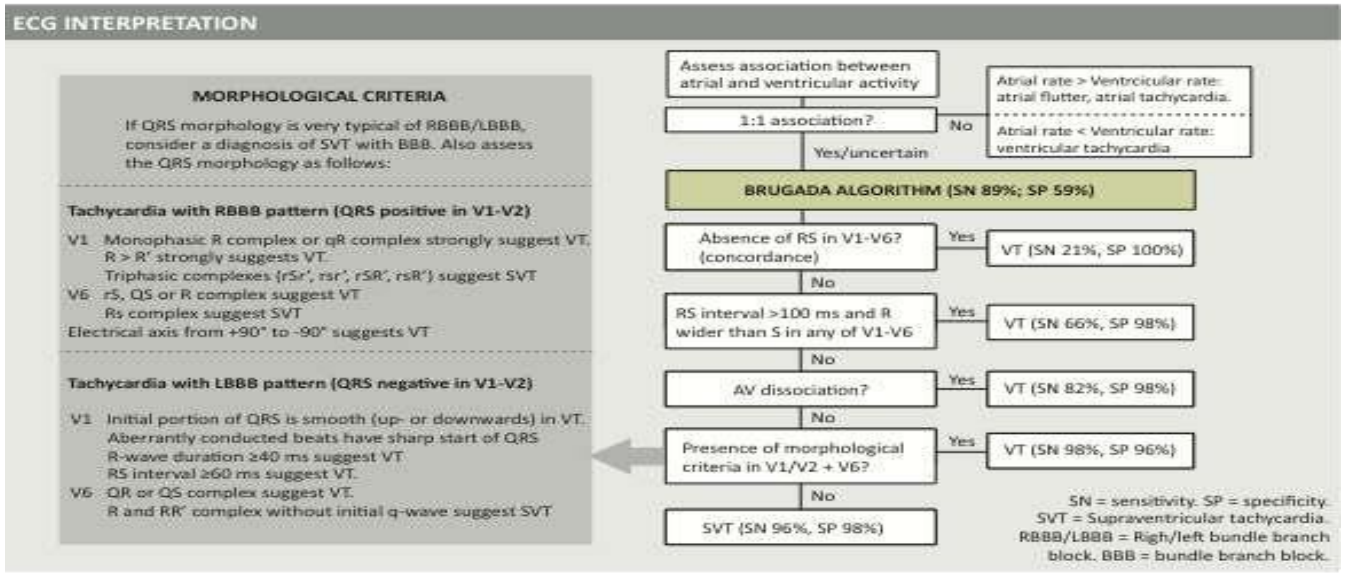
Analyse P-waves

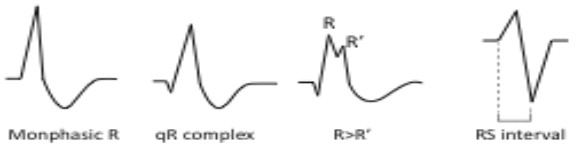
P-waves and baseline have sawtooth pattern: Atrial flutter.
Retrograde P-wave + short RP interval: typical AVNRT, AVRT, rarely AT
Retrograde P-wave + short RP interval and <70 ms: typical AVNRT most likely
Retrograde P-wave + short RP interval but >70 ms: AVRT most likely
Retrograde P-wave + long RP interval: Atrial tachycardia (focus near AV node), less likely to be atypical AVNRT or AVRT with slow accessory pathway (called PJRT).

Positive P-wave + long RP interval: sinus tachycardia, atrial tachycardia
Positive P-wave + short RP interval: atrial tachycardia with first-degree AV-block
Positive P-wave similar to sinus P-wave + long RP interval: sinus tachycardia, inapproproate sinus tachycardia, SANRT, atrial tachycardia w/ focus near the sinoatrial node.

Diagnosis and management of tachyarrhythmias with wide QRS complex

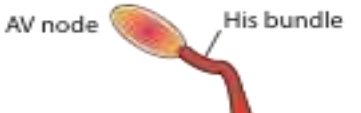






Intraventricular conduction defects

ECG changes and criteria in bundle branch blocks and fascicular blocks



Right bundle branch
Activates the right ventricle

RBBB Right bundle branch block

QRS duration ≥0,12 s.
Normal electrical axis.

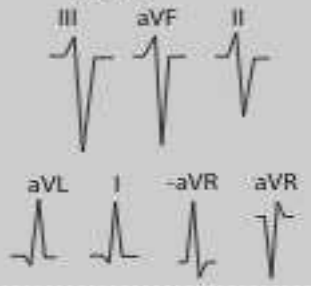
V1-V2 shows rsr', rsR' or rSR'.

V5, V6, I and aVL shows broad S-wave.

Secondary ST-T changes.

Anterior fascicle
Activates the anterior wall

LAFB Anterior fascicular block



QRS duration <0,12 s.
Left axis deviation.

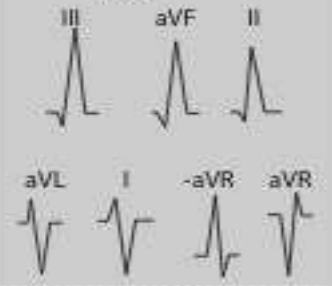
qR complex in aVL.

rS complex in II, III and aVF.

V5-V6 usually show qR complex.

Posterior fascicle
Activates the posterior and inferior wall.

LPFB Posterior fascicular block



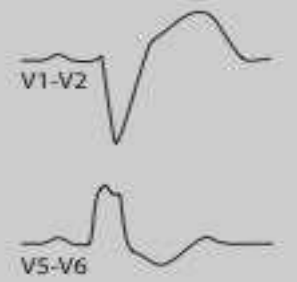
QRS duration <0,12 s.
Right axis deviation.

rS complex in lead I and aVL.
qR complex in inferior leads.

Always q-wave in III and aVF.

Left bundle branch
Provides the interventricular septum with Purkinje fibers. Divided into two fascicles which activates the left ventricle.

LBBB Left bundle branch block



QRS duration ≥0,12 s.

V1-V2: Deep and broad S-wave or QS complex.

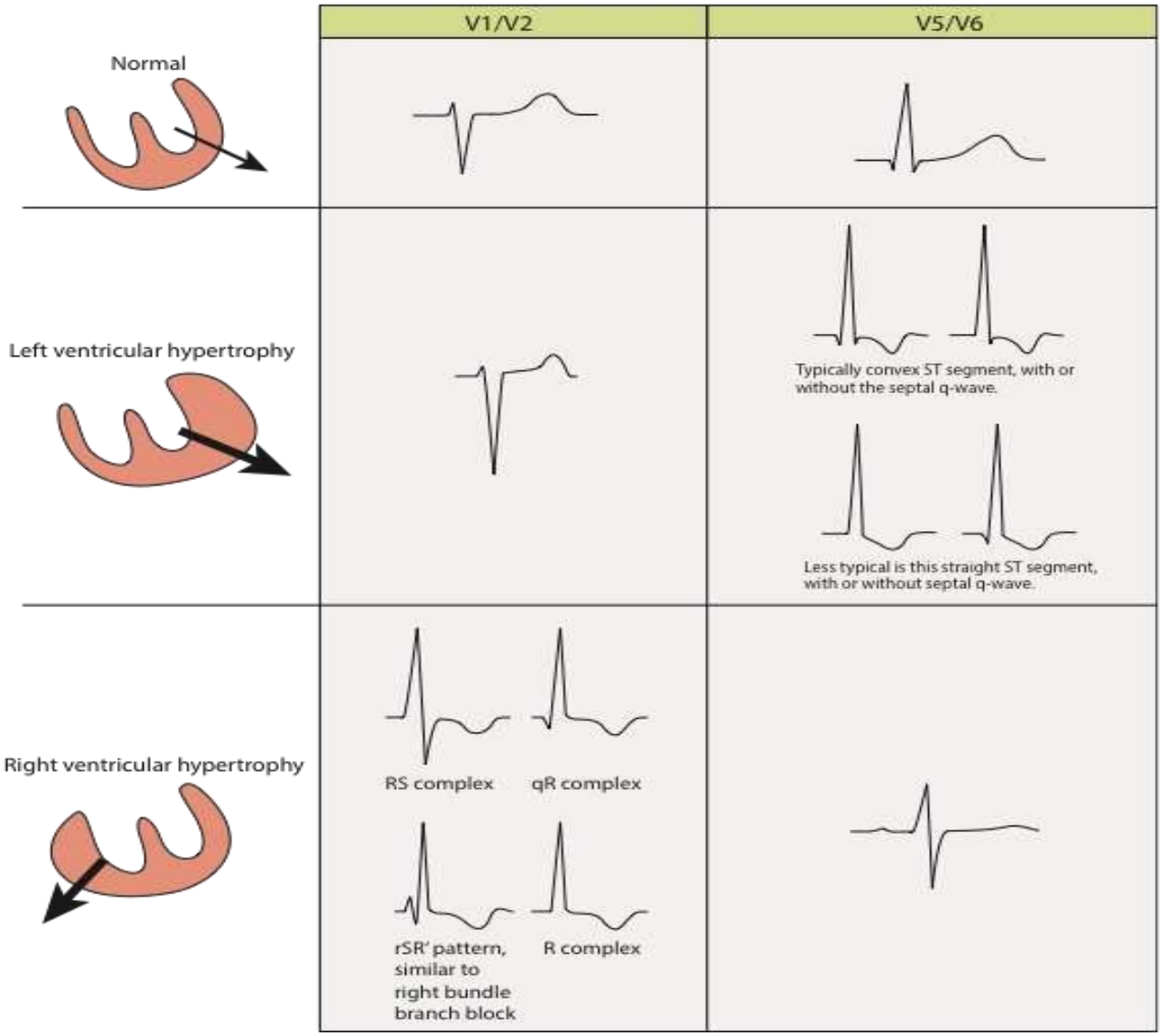
V5-V6: Broad, clumsy, positive R-wave.

Secondary ST-T changes.

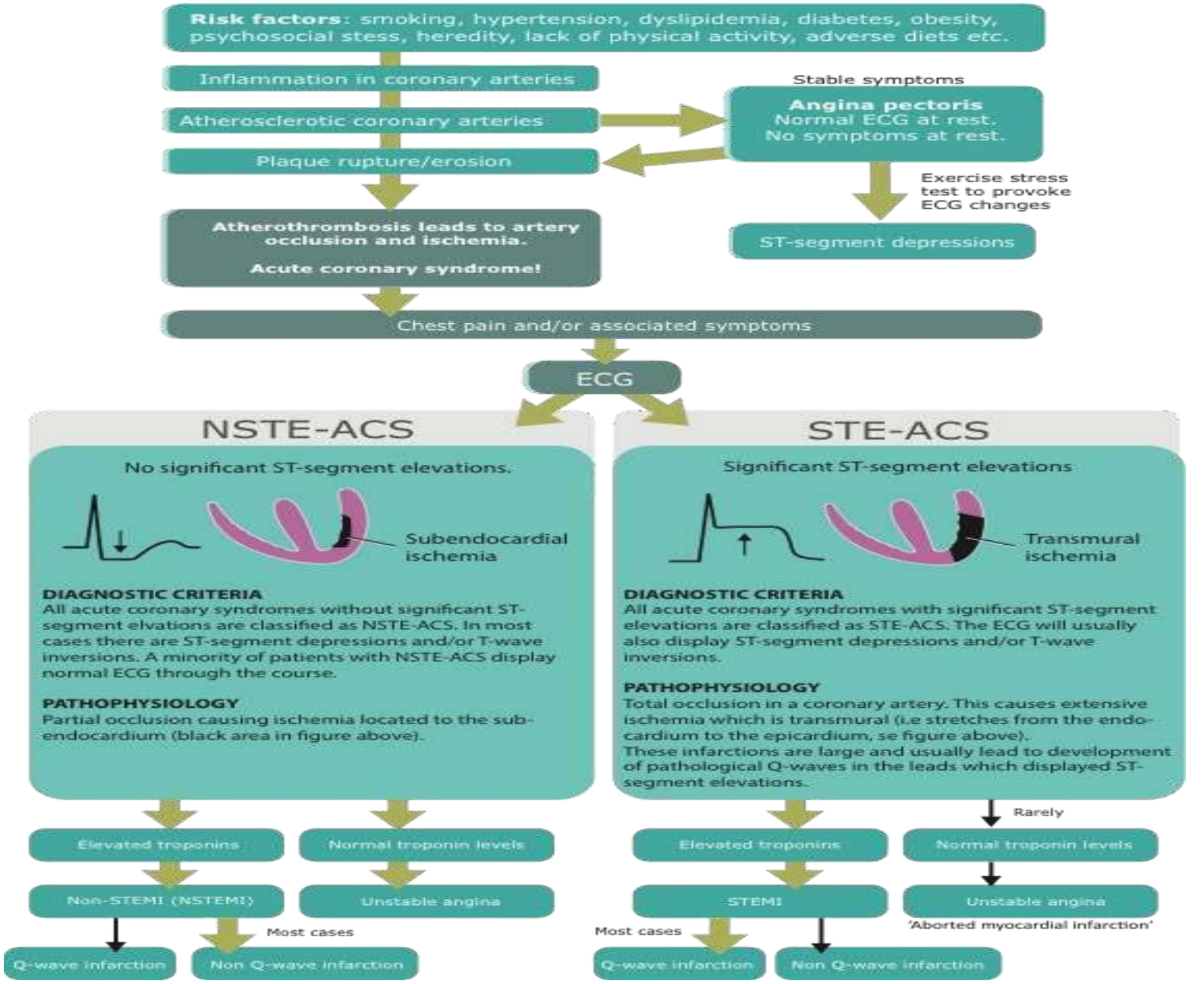
Note that both aVR and –aVR are shown.

Hypertrophy and dilatation

Use leads V1, V2, V5 and V6 to spot ventricular hypertrophy.
These leads show characteristic QRS changes in hypertrophy.



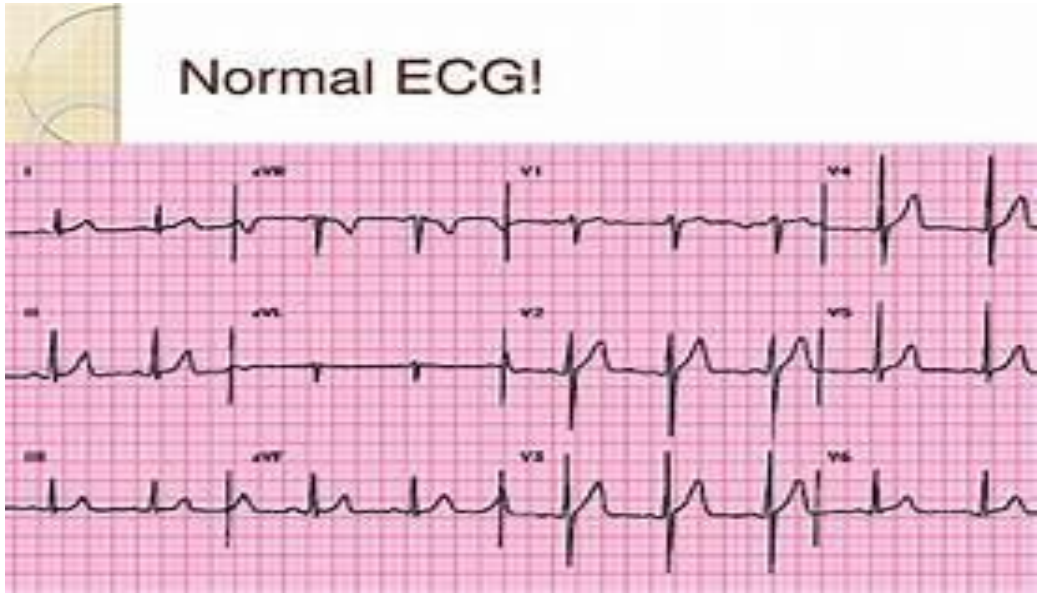
Classification of acute coronary syndromes (ACS)



NORMAL ECG

Q1:
This young patient is a smoker, presented with inflammatory, submammary chest pain, what's your interpretation of this ECG?
Normal ECG

Q2:
This is an ECG for a 22 YO male ,presented for a regular check-up.What is your interpretation?
Normal ECG



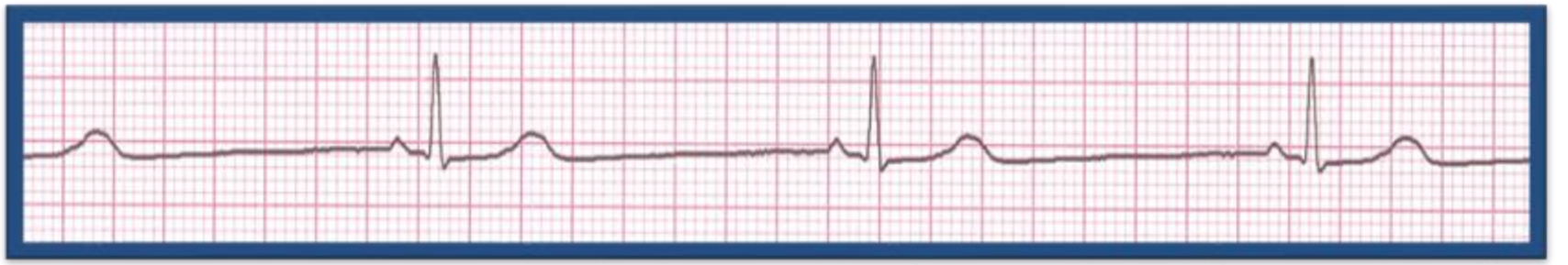
Q3:
A 35 year old female patient complaining of a retrosternal chest pain that is relieved when she stands up, what's your interpretation of this ECG?
Normal ECG

Sinus Rhythms

Sinus Bradycardia

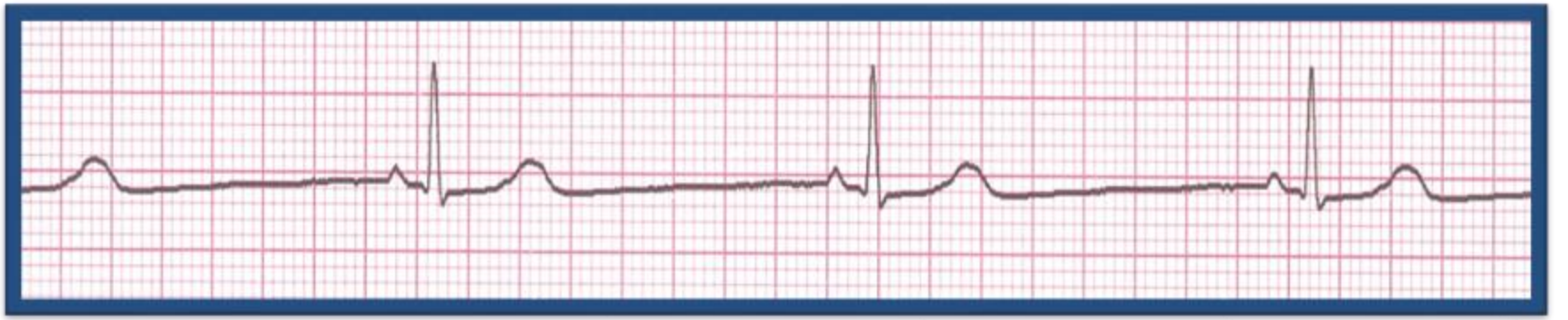
Sinus Tachycardia

Rhythm #1



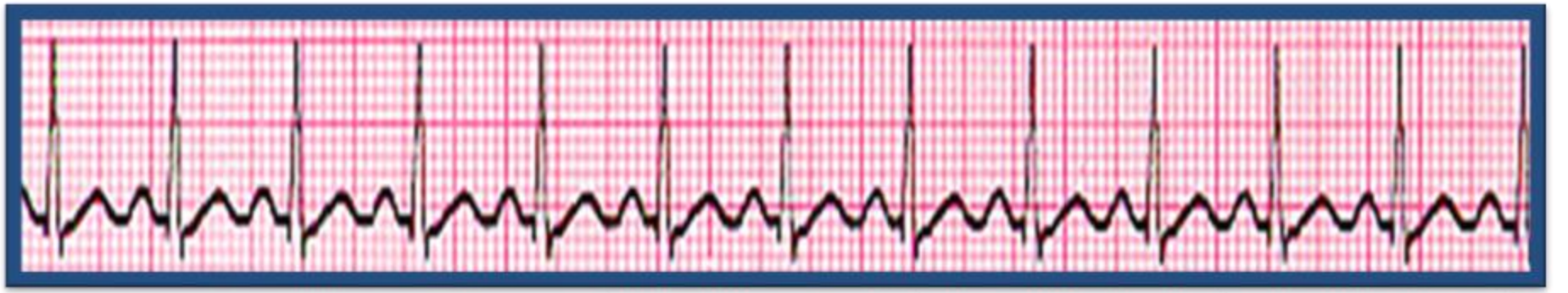
30 bpm	Rate
regular	Regularity
normal	P waves?
0.12 s	PR interval?
0.10 s	QRS duration?
Sinus Bradycardia	Interpretation

Sinus Bradycardia



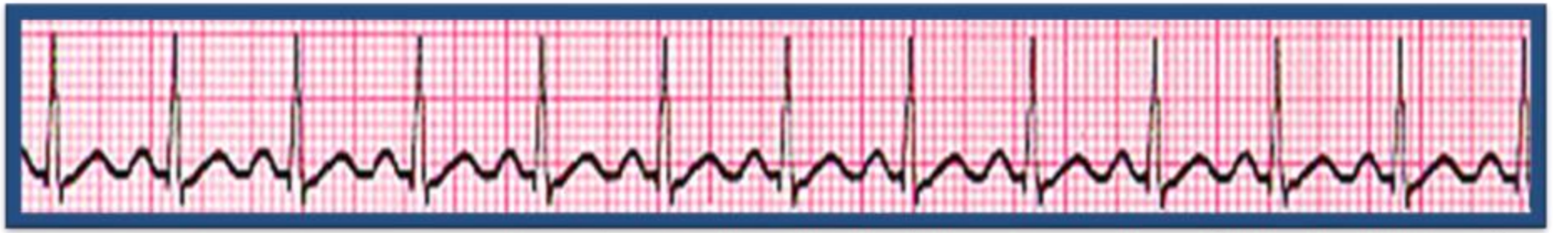
- Deviation from NSR
  - Rate < 60 bpm
- Etiology: SA node is depolarizing slower than normal, impulse is conducted normally (i.e. normal PR and QRS interval).
- causes : ischemia , increased vagal tone , anti arrhythmic drug ,may be norma in trained athletes .
- clinically : asymptomatic ,or present with inability to exercise ,angina or syncope
- treatment : atropin , cardiac pacemaker

Rhythm



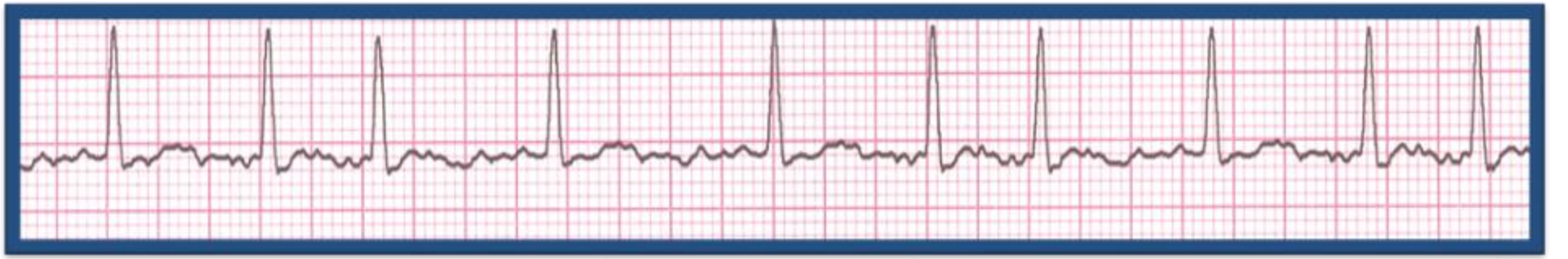
130 bpm	Rate
regular	Regularity
normal	P waves
0.16 s	PR interval
0.08 s	QRS duration
Sinus Tachycardia	Interpretation

Sinus Tachycardia



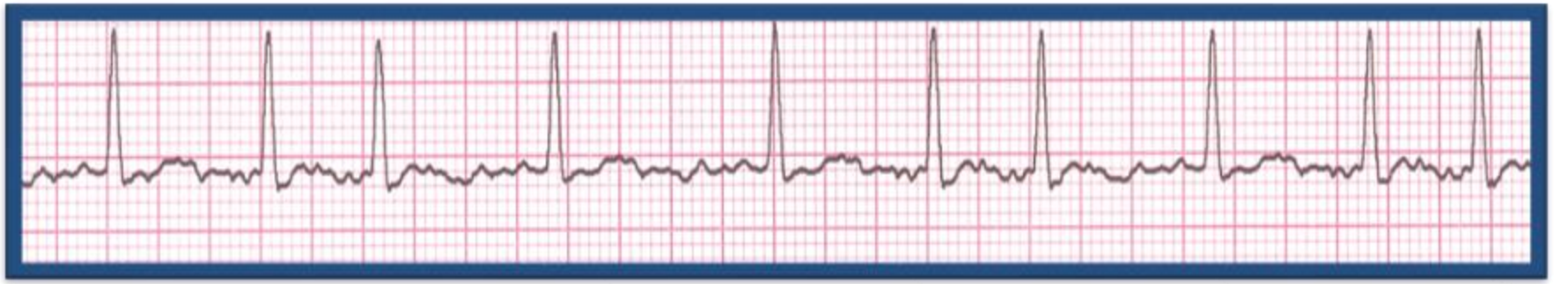
- Deviation from NSR
  - Rate > 100 bpm
- Etiology: SA node is depolarizing faster than normal, impulse is conducted normally.
- Remember: sinus tachycardia is a response to physical or psychological stress, not a primary arrhythmia.

Atrial fibrillation



100 bpm	Rate
Irregularly irregular	Regularity
None	P waves
None	PR interval
0.06 s Narrow with normal shape	QRS duration QRS complex
Atrial fibrillation	Interpretation

Atrial Fibrillation



- Etiology: Recent theories suggest that it is due to multiple re-entrant wavelets conducted between the R & L atria. Either way, impulses are formed in a totally unpredictable fashion. The AV node allows some of the impulses to pass through at variable intervals (so rhythm is irregularly irregular).
- Deviation from NSR
  - No organized atrial depolarization, so no normal P waves (impulses are not originating from the sinus node).
  - Atrial activity is chaotic (resulting in an irregularly irregular rate).
  - Common, affects 2-4%, up to 5-10% if > 80 years old

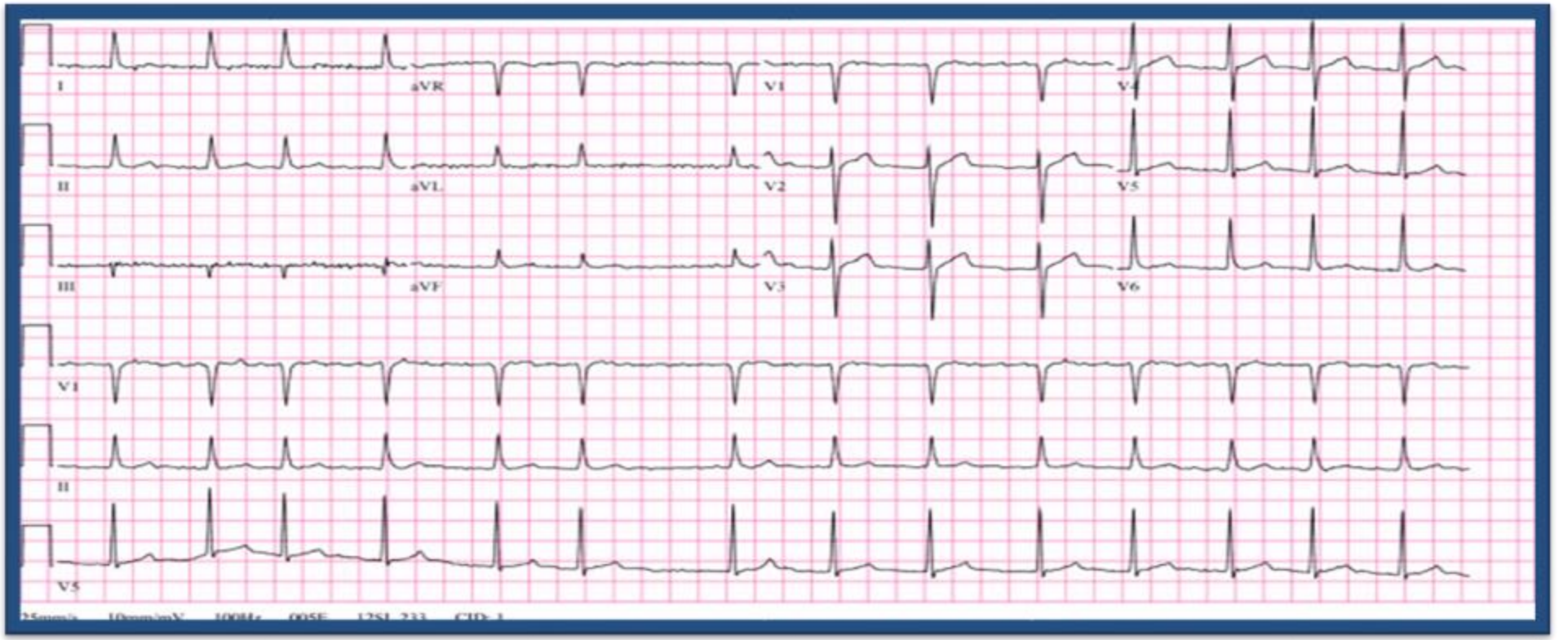
Atrial Fibrillation

- Paroxysmal AF : AF that terminates within 7 days of onset either following treatment or spontaneously
- Persistent AF : Continuous AF for > 7 days
- Long-standing persistent AF: Continuous AF for > 1 year
- Permanent AF : Long-standing persistent AF that is not treated following a joint decision by the patient and the physician

Types of Atrial Fibrillation

AF pattern	Definition
First diagnosed AF	AF that has not been diagnosed before, irrespective of the duration of the arrhythmia or the presence and severity of AF-related symptoms.
Paroxysmal AF	Self-terminating, in most cases within 48 hours. Some AF paroxysms may continue for up to 7 days.[a] AF episodes that are cardioverted within 7 days should be considered paroxysmal.[a]
Persistent AF	AF that lasts longer than 7 days, including episodes that are terminated by cardioversion, either with drugs or by direct current cardioversion, after 7 days or more.
Long-standing persistent AF	Continuous AF lasting for ≥1 year when it is decided to adopt a rhythm control strategy.
Permanent AF	AF that is accepted by the patient (and physician). Hence, rhythm control interventions are, by definition, not pursued in patients with permanent AF. Should a rhythm control strategy be adopted, the arrhythmia would be re-classified as 'long-standing persistent AF'.

- A 76-year-old man comes to the physician with palpitation , dizziness and progressively worsening fatigue over the past 3 months. He has a 50-pack-year smoking history. His pulse is 11 0/min and irregularly irregular. Initial ECG findings are shown below



A- What's your diagnosis?

AF (irregularly irregular rhythm and no p waves).

B) what is the initial investigation should be done ?

ECG (If the ECG is not present in the question)

C) give five possible causes ?

1) coronary artery disease (MI)
2) Valvular heart disease, especially rheumatic mitral valve disease
3) Hypertension
4) Sinoatrial disease
5) Hyperthyroidism, pheochromocytoma
6)Excessive Alcohol intake (holiday heart syndrome)
7) Pulmonary embolism

D) what's the valvular disorder?

Mitral stenosis

E) What is the charactaristics finding of Afib on ECG ?

- Irregularly irregular RR intervals
- P-waves are indiscernible
- Tachycardia
- Narrow QRS complex (< 0.12 seconds)

G) Mention the 3 considerations in treating AF

Rate control / Rhythm control / Anticoagulation prophylaxis

H) what is the first step in management ?

Rate control (beta blocker (the best) (propanolol, metoprolol) or CCB (diltiazem, verapamil)

I) What is the best choice for rythme control ?

electrical cardioversion

J) in patient with contraindication to take anticoagulants , what's the anatomical structure to be occluded percutaneously ?

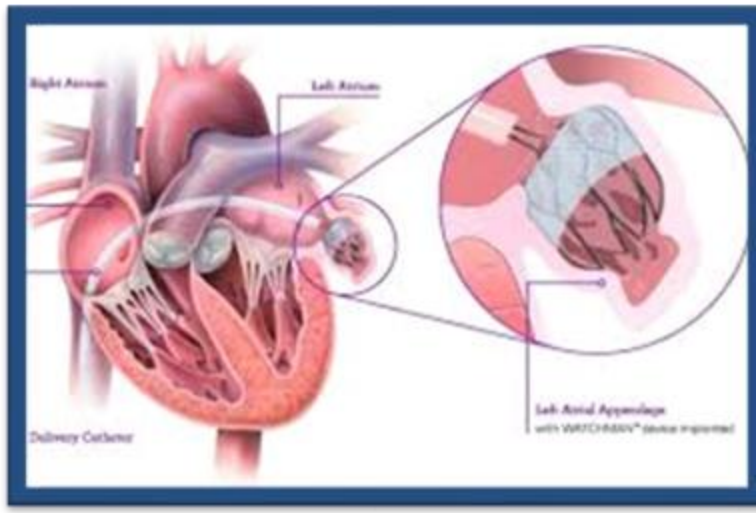
Left atrial appendage

K) Give the possible complications?

- Acute left heart failure→ pulmonary edema
- Thromboembolic events: stroke/TIA
- life-threatening ventricular tachycardia

L)AF without anatomical defect is called?

Lone AF



Name the score we depend on to consider anticoagulation therapy of AF patients

CHADS2 or CHA_2DS_2-VASc score

- Annual stroke risk
- 0 points = 0% (no prophylaxis required)
- 1 point = 1.3% (oral anticoagulant or aspirin recommended)
- 2+ points = >2.2% (oral anticoagulant recommended)

Management of A.fib

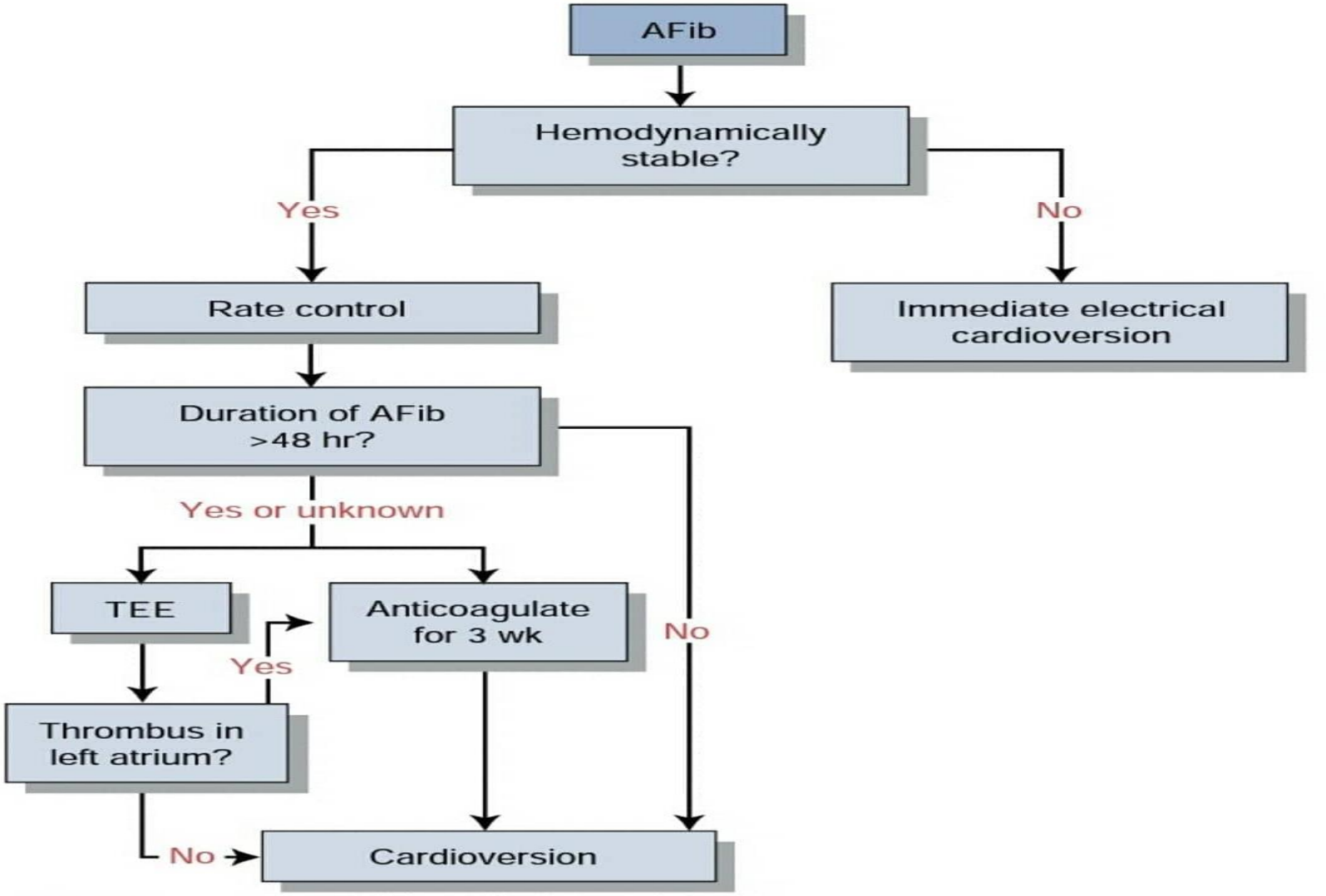


FIGURE 1-10 **Acute management of atrial fibrillation.**

- If the pt presented chest pain, confusion and his blood pressure is 90/60 pulse , what is the best initial step ? Immediate electrical cardioversion (hemodynamic unstable)

- If the pt presented chest pain, palpitation for three days and his BP is 130/95 pulse , what is the best initial step ?

  Rate control (beta blocker (the best) (propanolol, metoprolol) or CCB (diltiazem, verapamil) (hemodynamic stable)

  - What is the next step in management after we do rate control ??

    We do TEE (if present)to detect the presence of thrombus in the Lt atrium or we give anticoagulant to pateint for 4 weeks then we do cardioversion (hemodynamic stable)

- If the pt presented chest pain, palpitation for 12 hours and his BP is 130/95 pulse , what is the best initial step?

  Immediate cardioversion, after administration of intravenous heparin

- What is the preferred anticoagulant used in pt with Afib to prevent the thrombus formation ?

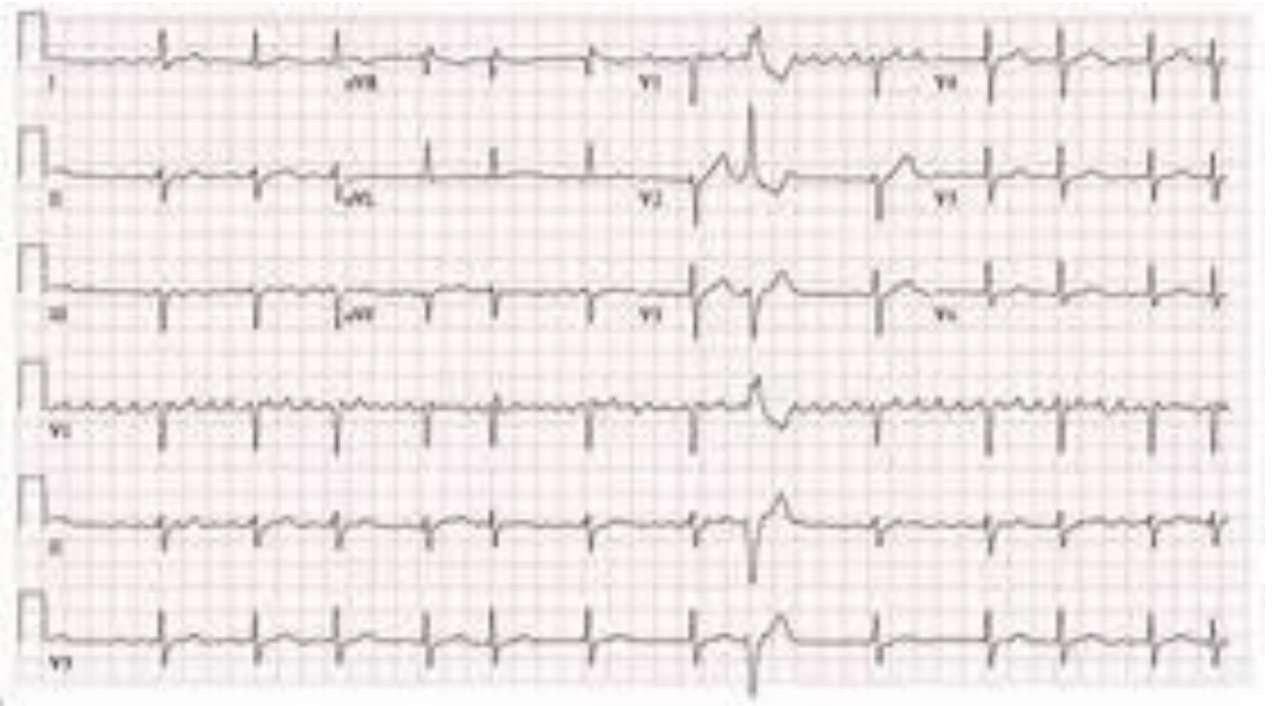
  Warfarin (INR 2-3)
- If the cardioversion is ineffictive , what is the next step ?

  Catheter-based radiofrequency ablation
- Where we should do the ablation ?

  In the atrial tissue around pulmonary vein openings

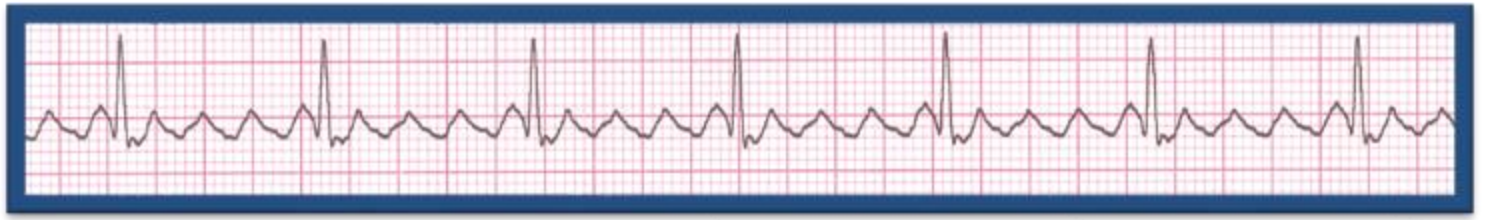
What is the abnormality in this ECG?

Irregularly Irregular Pulse, Absent p waves > Atrial Fibrillation



Atrial flutter

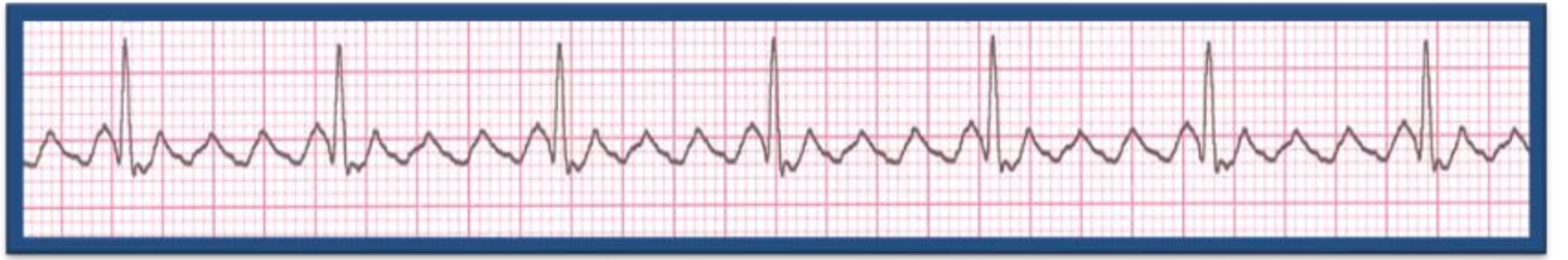
Atrial Flutter



- Deviation from NSR
  - No P waves. Instead flutter waves (note "sawtooth" pattern) are formed at a rate of 250 - 350 bpm.
  - Only some impulses conduct through the AV node (usually every other impulse).

- Etiology: Reentrant pathway in the right atrium with every 2nd, 3rd or 4th impulse generating a QRS (others are blocked in the AV node as the node repolarizes).

Atrial Flutter



70 bpm	Rate
Regular	Regularity
Flutter waves	P waves
None	PR interval
0.06 s (narrow)	QRS duration
Atrial flutter	Interpretation

- What is the best leads that we can see the flutter wave in ?

Inferior leads (II, III, aVF)

- The causes and the management of atrial flutter?

similar to that of Afib

- How can we control the ventricula rate ?

Digoxin, β-blockers (propanolol, metoprolol) or verapamil

- How can we restore the sinus rhythm ?

by direct current (DC) cardioversion or by using intravenous amiodarone

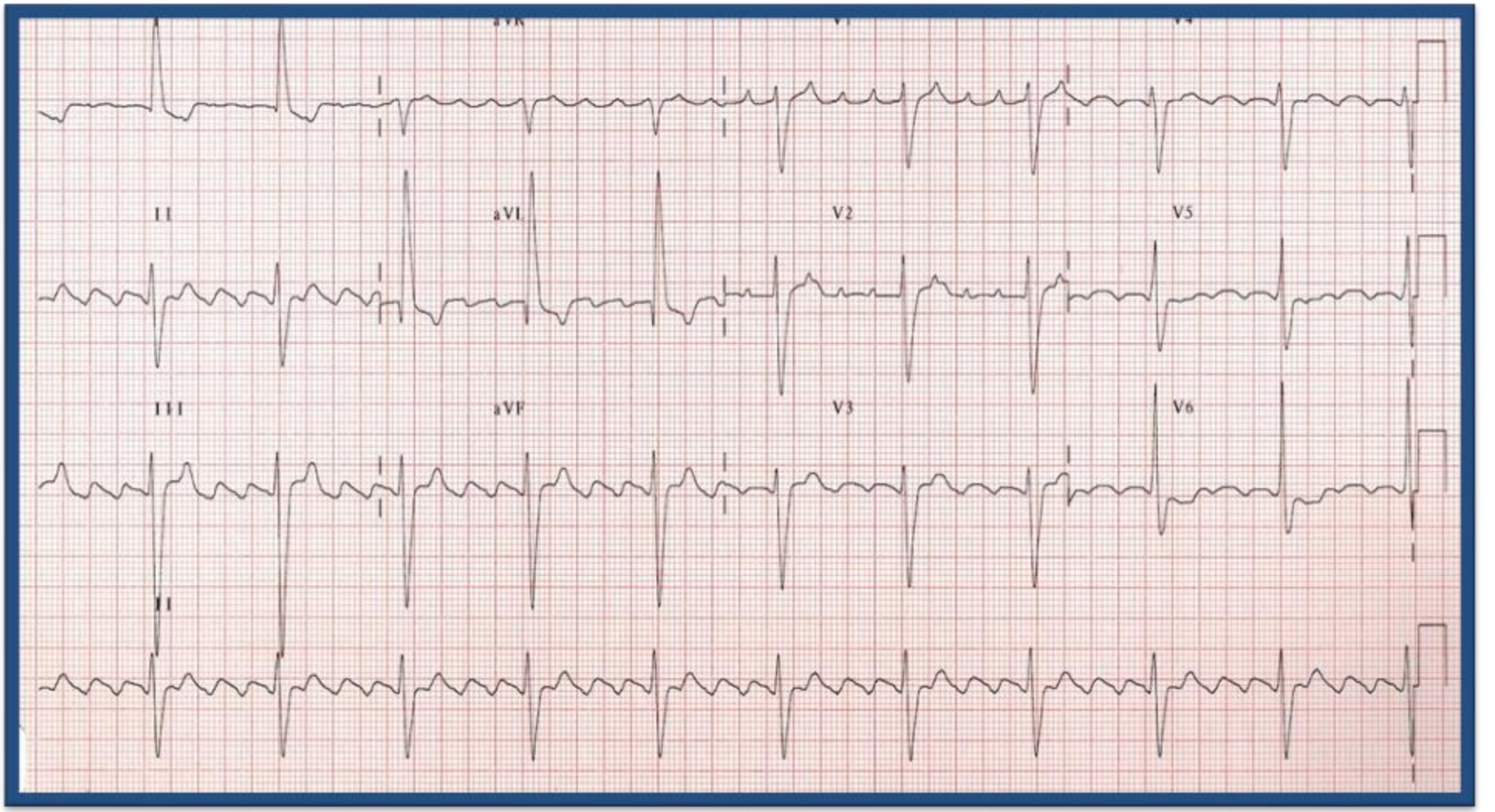
- How can we prevent the recurrent episodes of atrial flutter ?

Beta-blockers (propanolol, metoprolol) or amiodarone

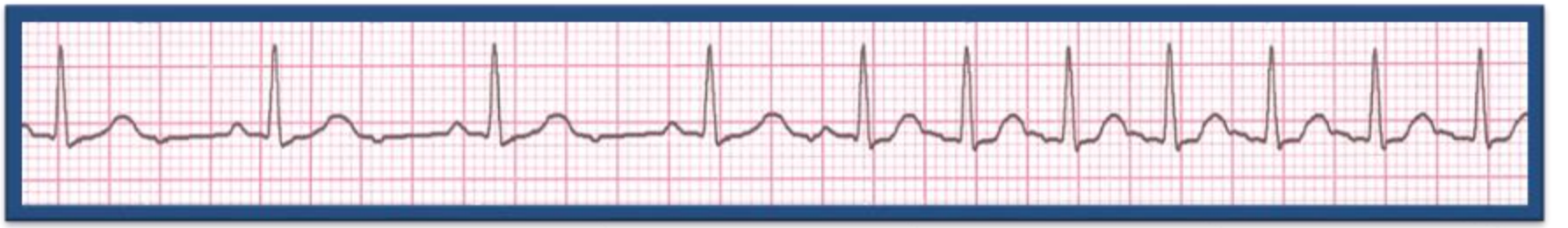
- What is the treatment of choice for patients with persistent symptoms ?

Catheter ablation

Diagnosis?
Atrial flutter
(characteristic saw tooth appearance)



PSVT

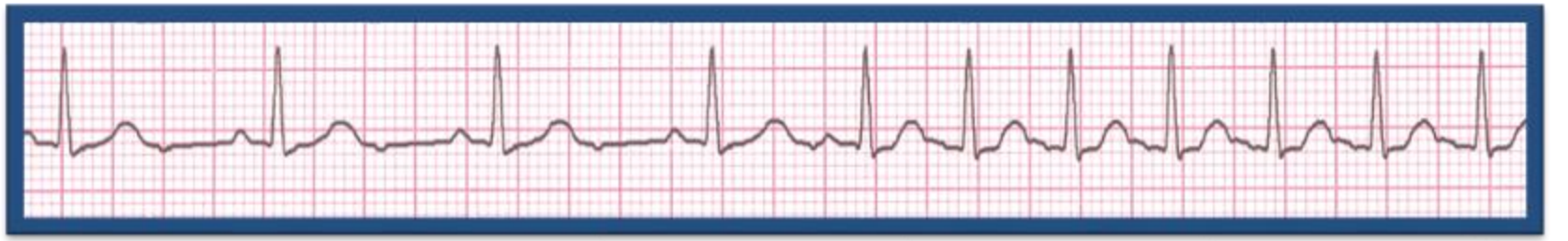


- Deviation from NSR
  - The heart rate suddenly speeds up, often triggered by a PAC (not seen here) and the P waves are lost.

- Etiology: There are several types of PSVT but all originate above the ventricles (therefore the QRS is narrow).

- Most common: abnormal conduction in the AV node (reentrant circuit looping in the AV node).

Rhythm #7



74 →148 bpm	Rate
Regular → regular	Regularity
Normal → none	P waves
0.16 s → none	PR interval
0.08 s	QRS duration
Paroxysmal supraventricular tachycardia (PSVT)	Interpretation

Q:18 YO male came to ER complaining of palpitation, depending on ECG of this pt, what is your spot Dx?

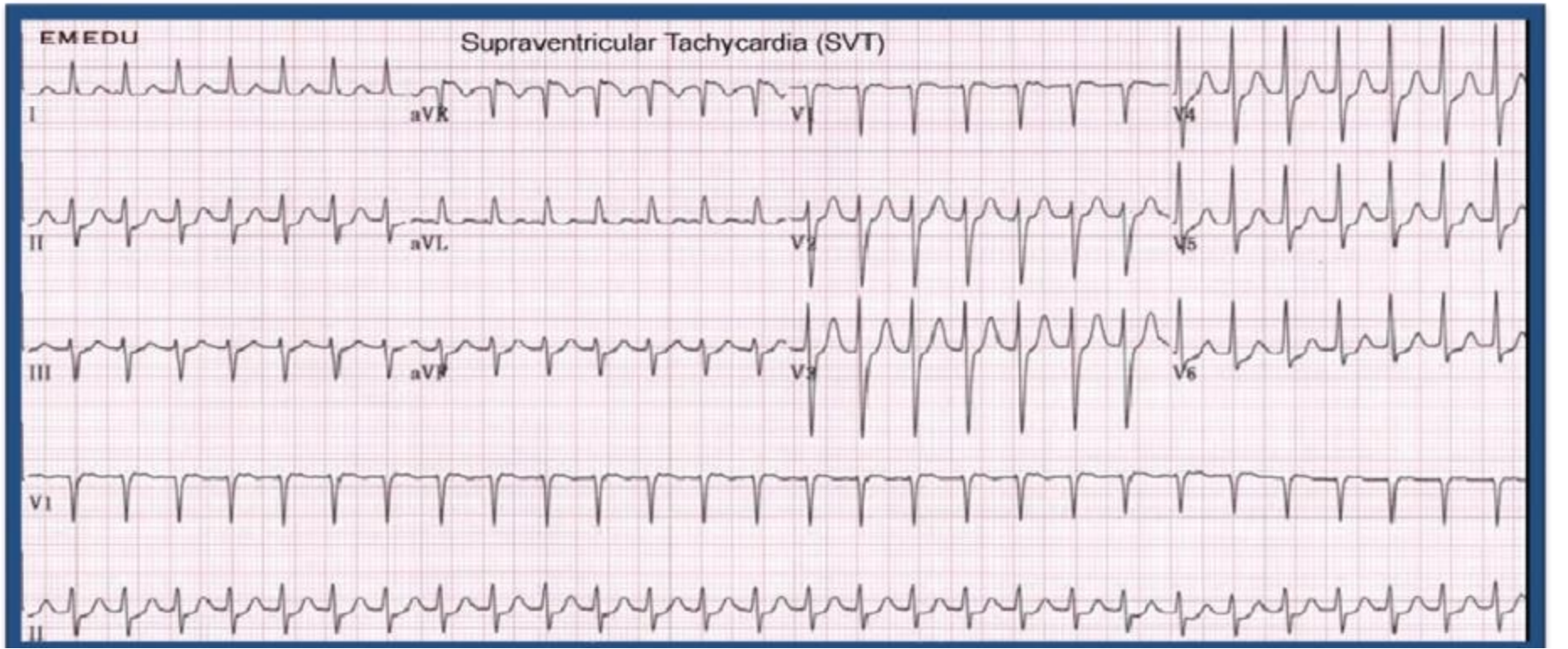
Supraventricular tachycardia (**SVT**)

Q: A pt presented with recurrent palpitation for 8 weeks, what is your Dx according to his ECG?

Paroxysmal supraventicular tachycardia.

Q: This patient came with (??) & blood pressure of , & this is his ECG, what is the treatment?

Since the patient is stable Adenosin.



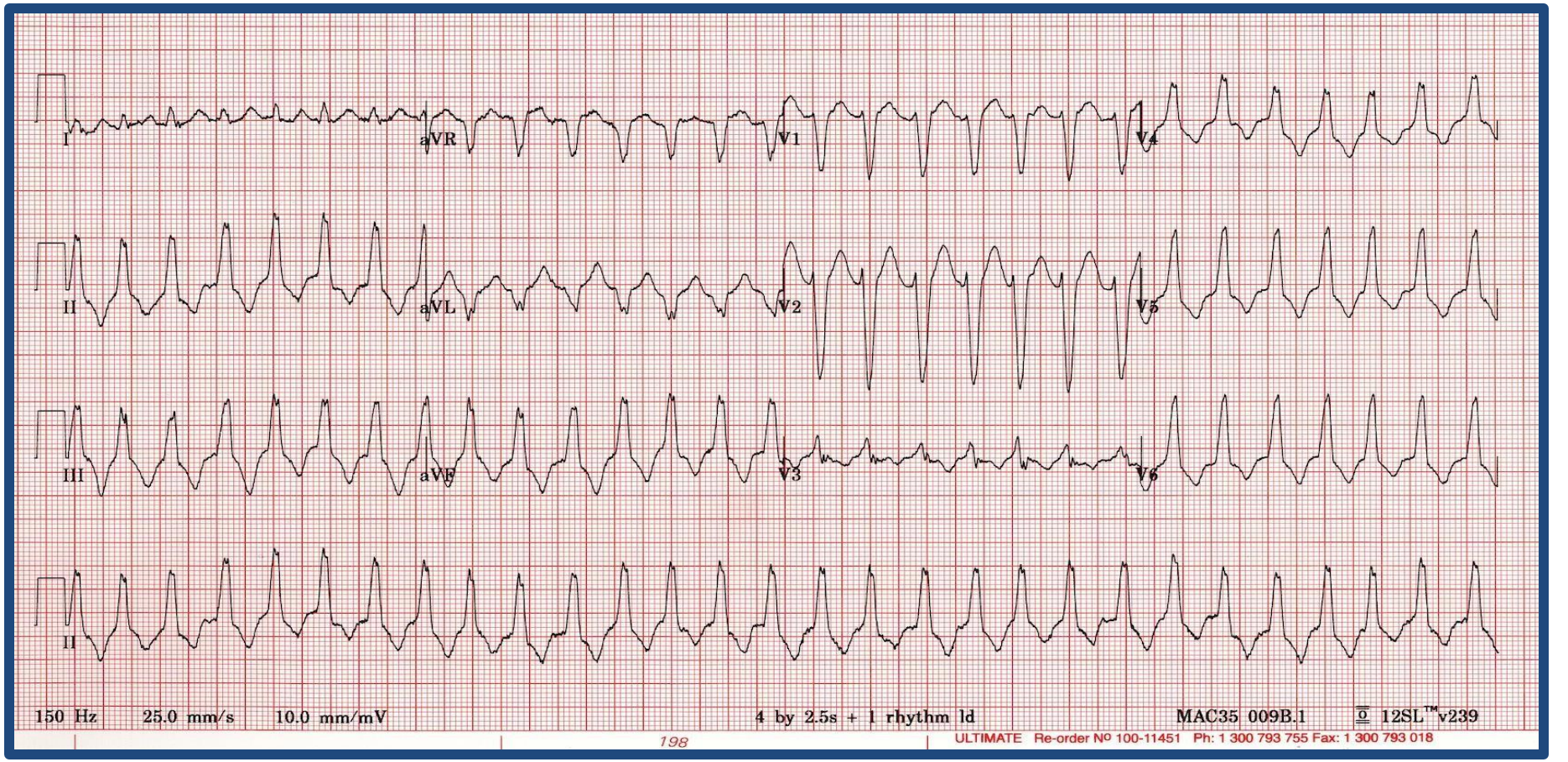
Q:A 30 year old male came to ER complaining of episodic palpitations & sweating.

what is your spot Dx?

Supraventricular tachycardia

What is the medication of choice?

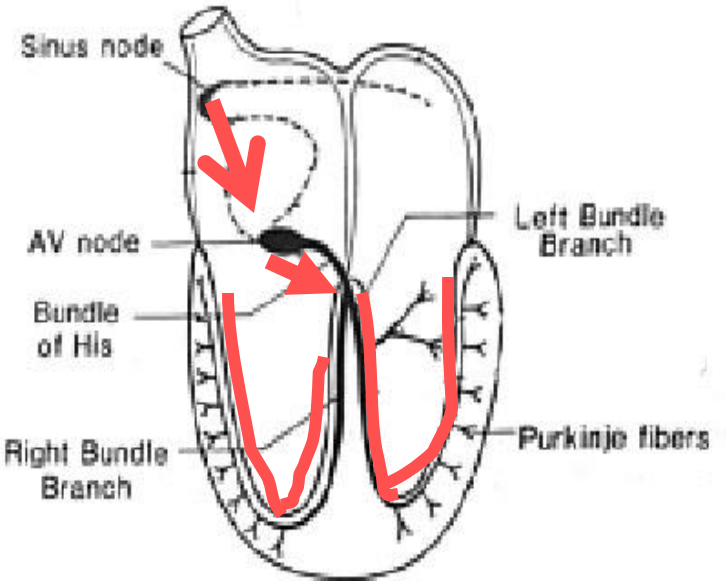
Adenosine



Ventricular Arrhythmias

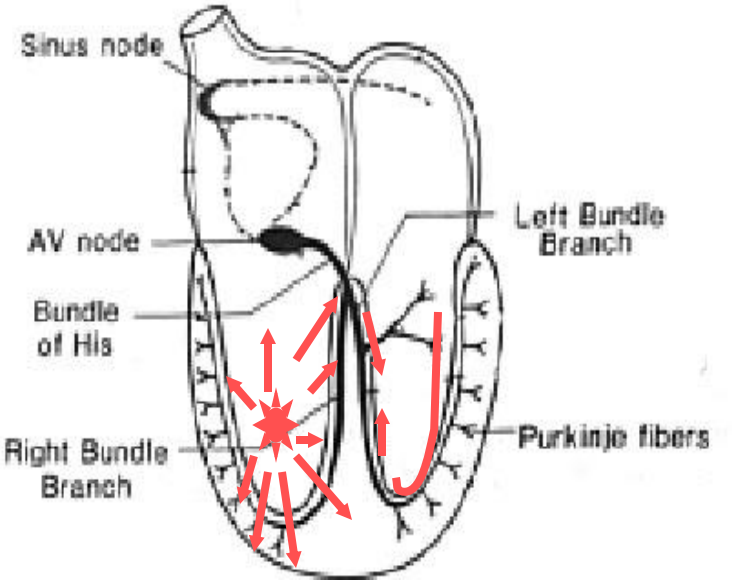
- *Ventricular Tachycardia*
- *Ventricular Fibrillation*

Ventricular Conduction



Normal

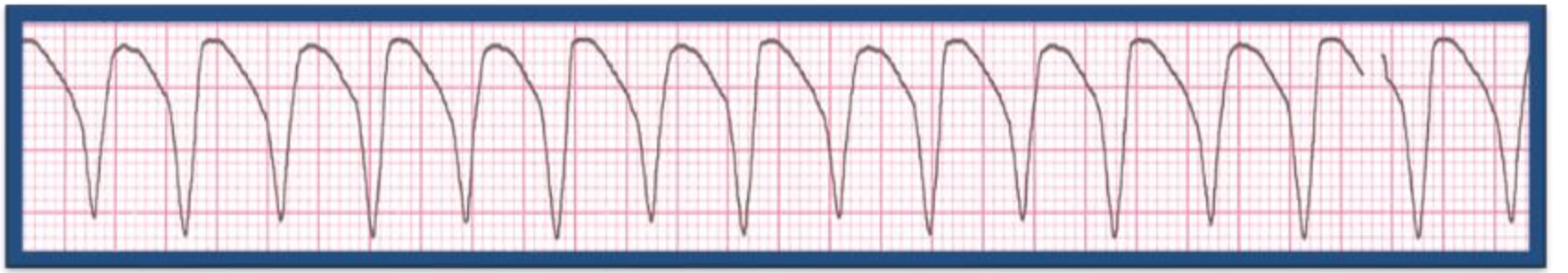
Signal moves rapidly through the ventricles



Abnormal

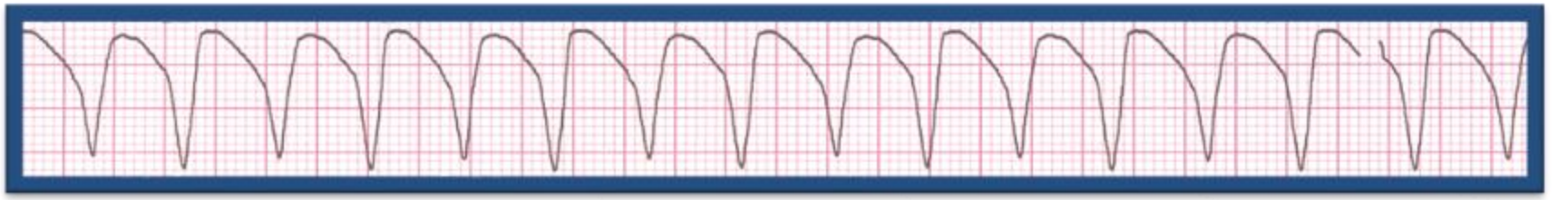
Signal moves slowly through the ventricles

Rhythm



160 bpm	Rate
regular	Regularity
none	P waves
none	PR interval
wide (> 0.12 sec)	QRS duration
Ventricular Tachycardia	Interpretation

Ventricular Tachycardia



- Deviation from NSR
  - Impulse is originating in the ventricles (no P waves, wide QRS).
- Etiology: There is a re-entrant pathway looping in a ventricle (most common cause).

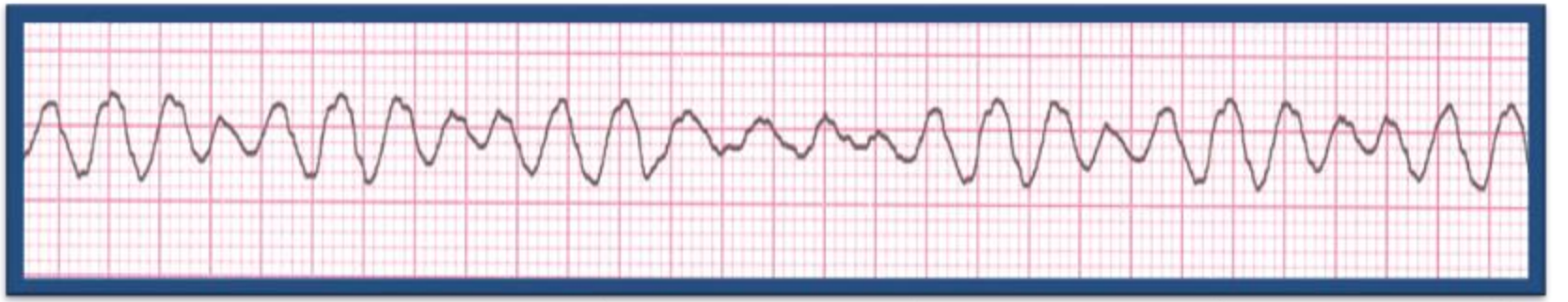
- Ventricular tachycardia can sometimes generate enough cardiac output to produce a pulse; at other times no pulse can be felt.
- 1-causes : CAD pror MI is the most common cause , active ischemia , cardiomyopathy, congenital defects ,prolonged QT syndrome ,drug toxicity .
- 2- clinically : palpitation , dyspnea , angina syncope or near syncope , signs of cardiogenic shock on PE we can see cannon A wave
- 3-dx : wide bizarre QRS

- 4-Treatment :

I. If hemodynamically stable with mild symptoms and systolic BP >90 : IV amiodaron IV procainamide or IV sotalol

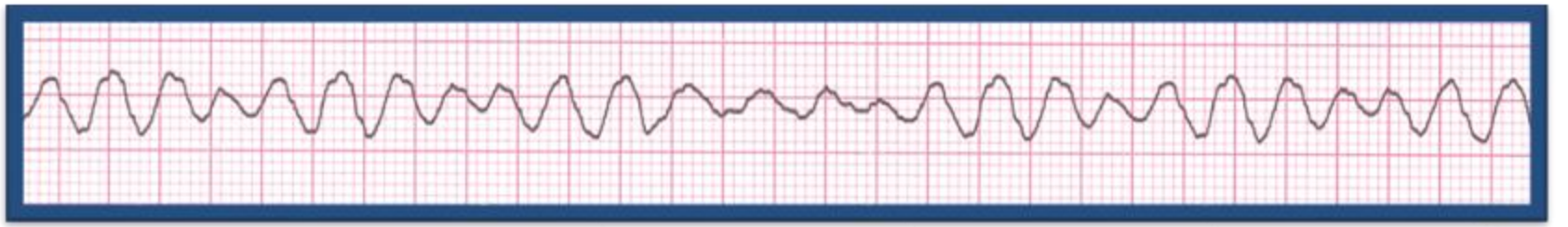
II. If hemodynamically unstable or pt with severe symptoms :immidiate synchronus DC cardioversion follow with IV amiodaron

Rhythm



none	Rate
irregularly irreg.	Regularity
none	P waves
none	PR interval
wide, if recognizable	QRS duration
Ventricular Fibrillation	Interpretation

Ventricular Fibrillation



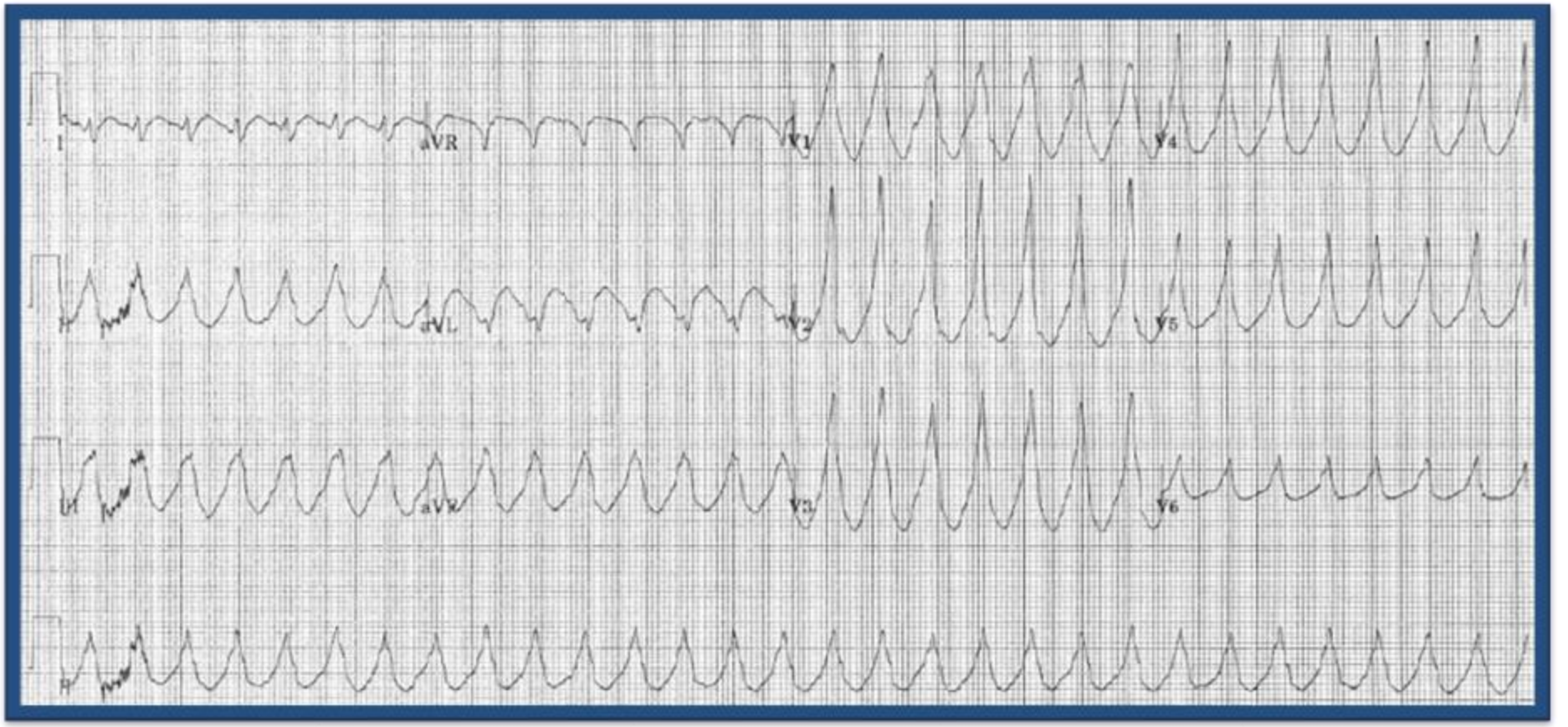
- Deviation from NSR
  - Completely abnormal.
- Etiology: The ventricular cells are excitable and depolarizing randomly.
- Rapid drop in cardiac output and death occurs if not quickly reversed
- causes : ischemic heart disease is the most common cause , anti arrhythmic drugs , Afib with a very rapid ventricular rate in pt with WPWs
- Clinically :cannot measure BP; absent heart sound and pulse , pt is unconscious if un treated lead to sudden cardiac death
- Dx : ECG findings : no QRS compexes ,no waves can be identified and irregular rhythm
- Treatment :Immidiate defibrillation and CPR is indicated

Q:This patient presented with dizziness & palpitation, normal blood pressure.

What's the treatment of this case?

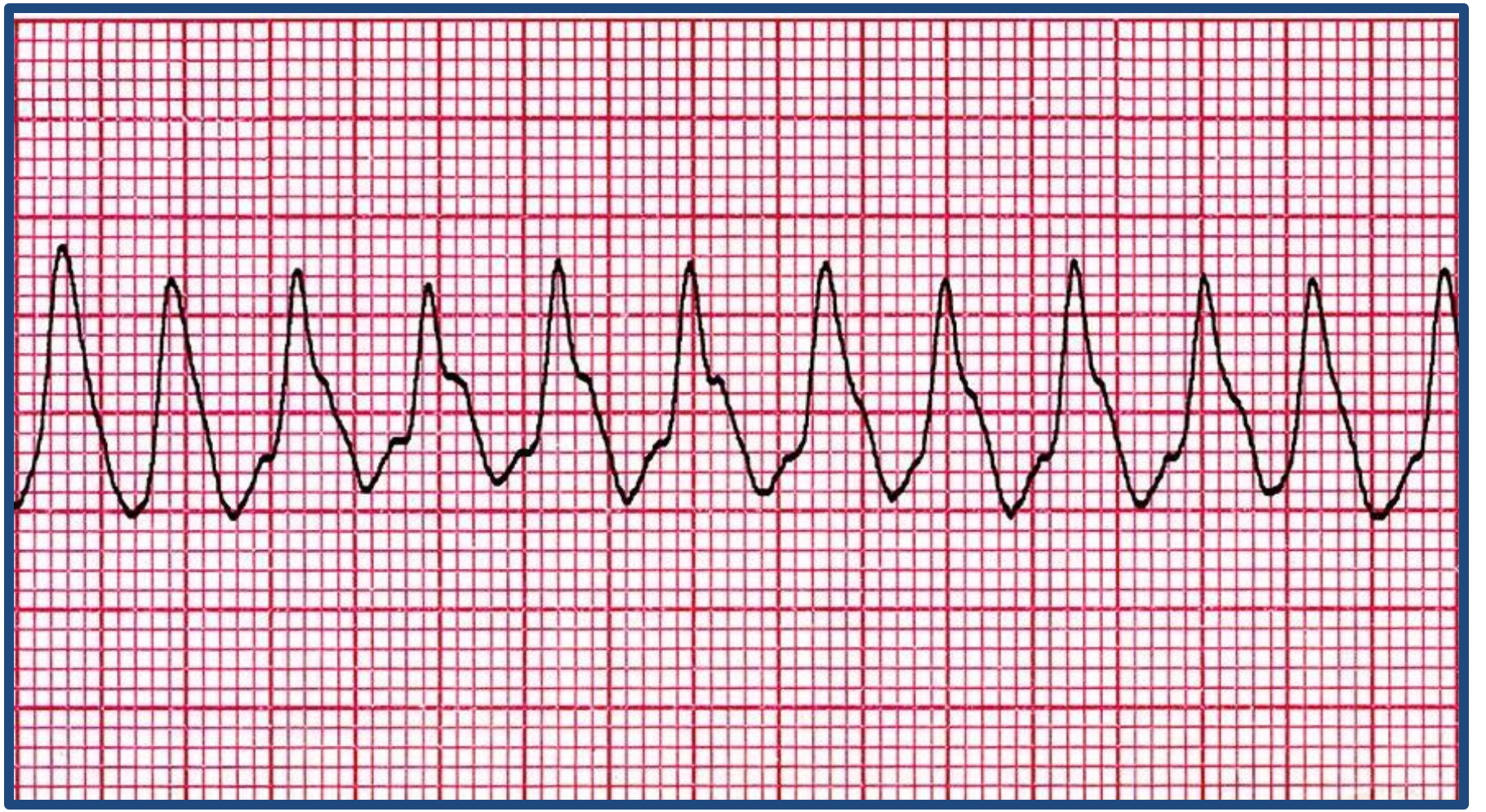
IV amiodaron or IV procainamide or Lidocain

(V. tachycardia)



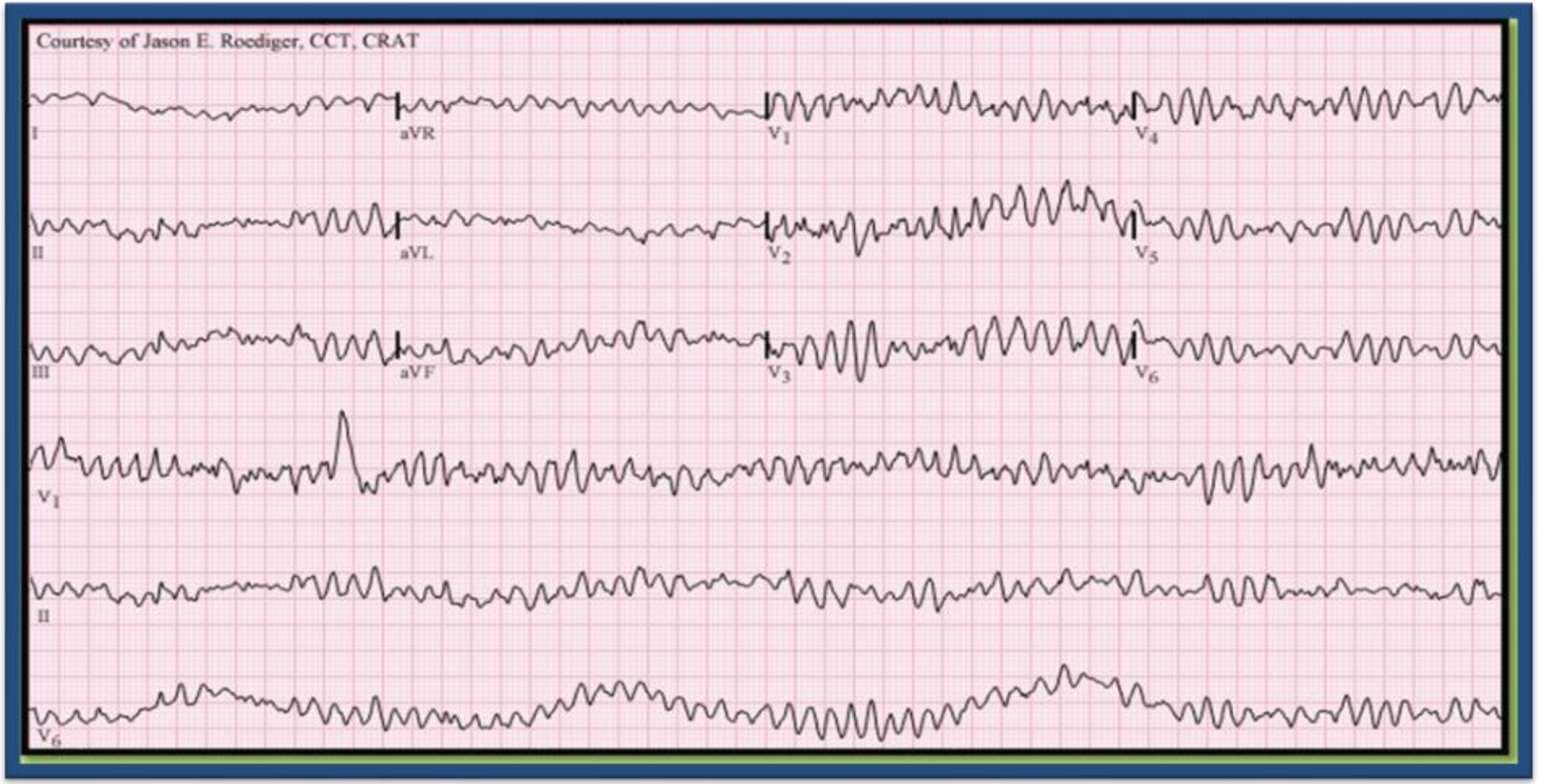
Q:A patient is hospitalized and all of the sudden he collapses, what is the ECG finding ?

Ventricular tachycardia



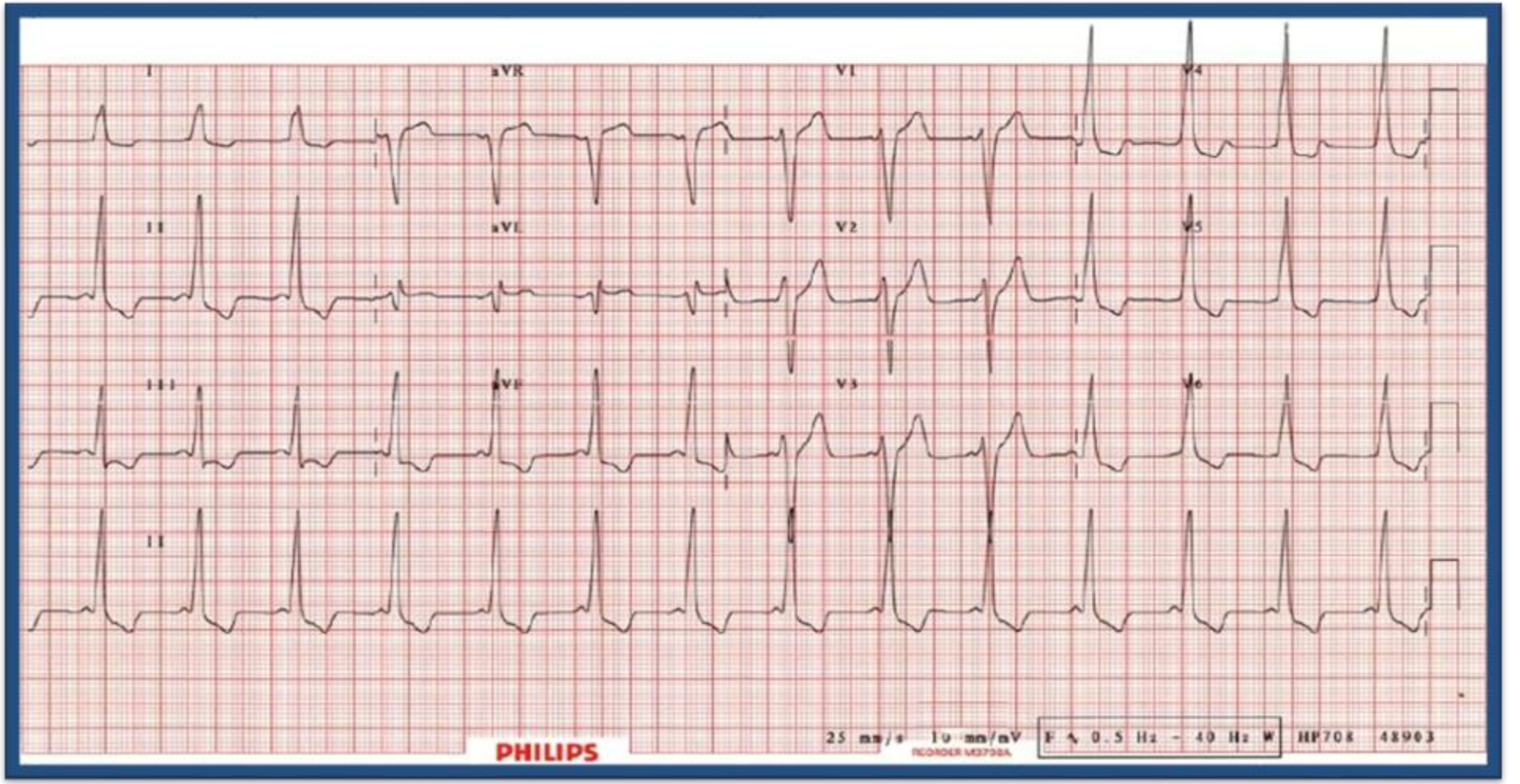
Q:50 YO male in CCU, he is waiting for cath., he lost his consciousness, with this ECG. Dx? Your management?

ventricular fibrillation >>DC shock.



Q: Patient has episodes ofpalpitation, his ECG was like this, what is your Dx.?

WPW syndrome.



I. Finding on ECG : narrow complex tachycardia, short PR interval and delta wave

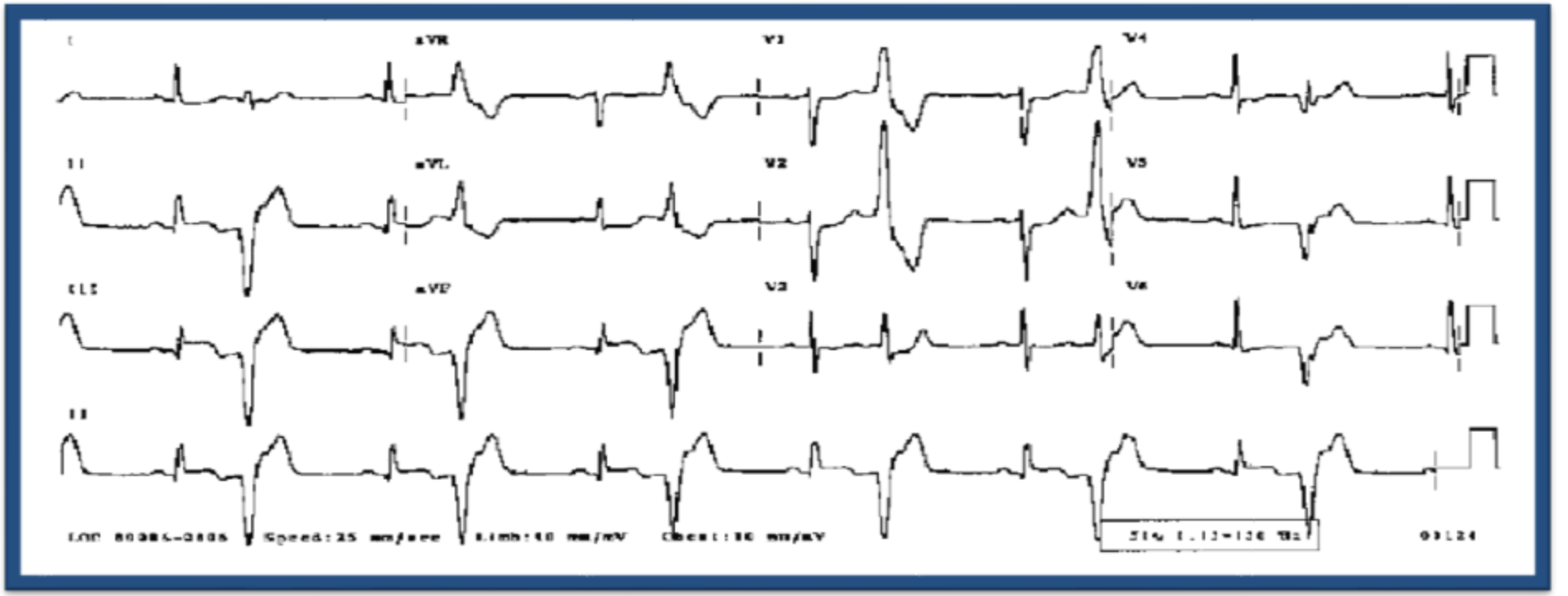
II. Treatment : radiofrequency catheter ablation

medical treatment : procainamide or quinidine

Q: What is your spot Dx?

Ventricular bigeminy

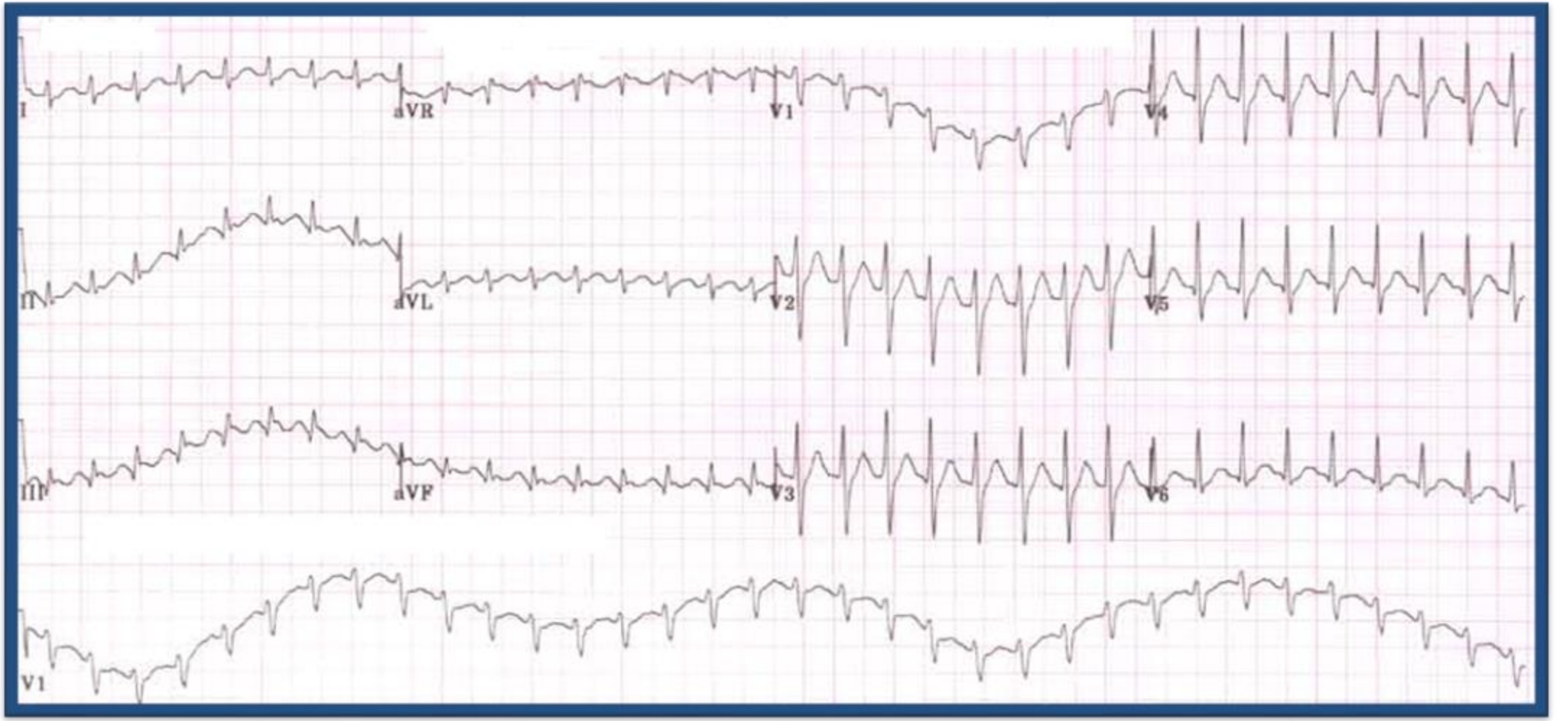
Dose not require treatment if asymptomatic but if symptomatic beta blockers can be used



Q: What is the best initial drug?

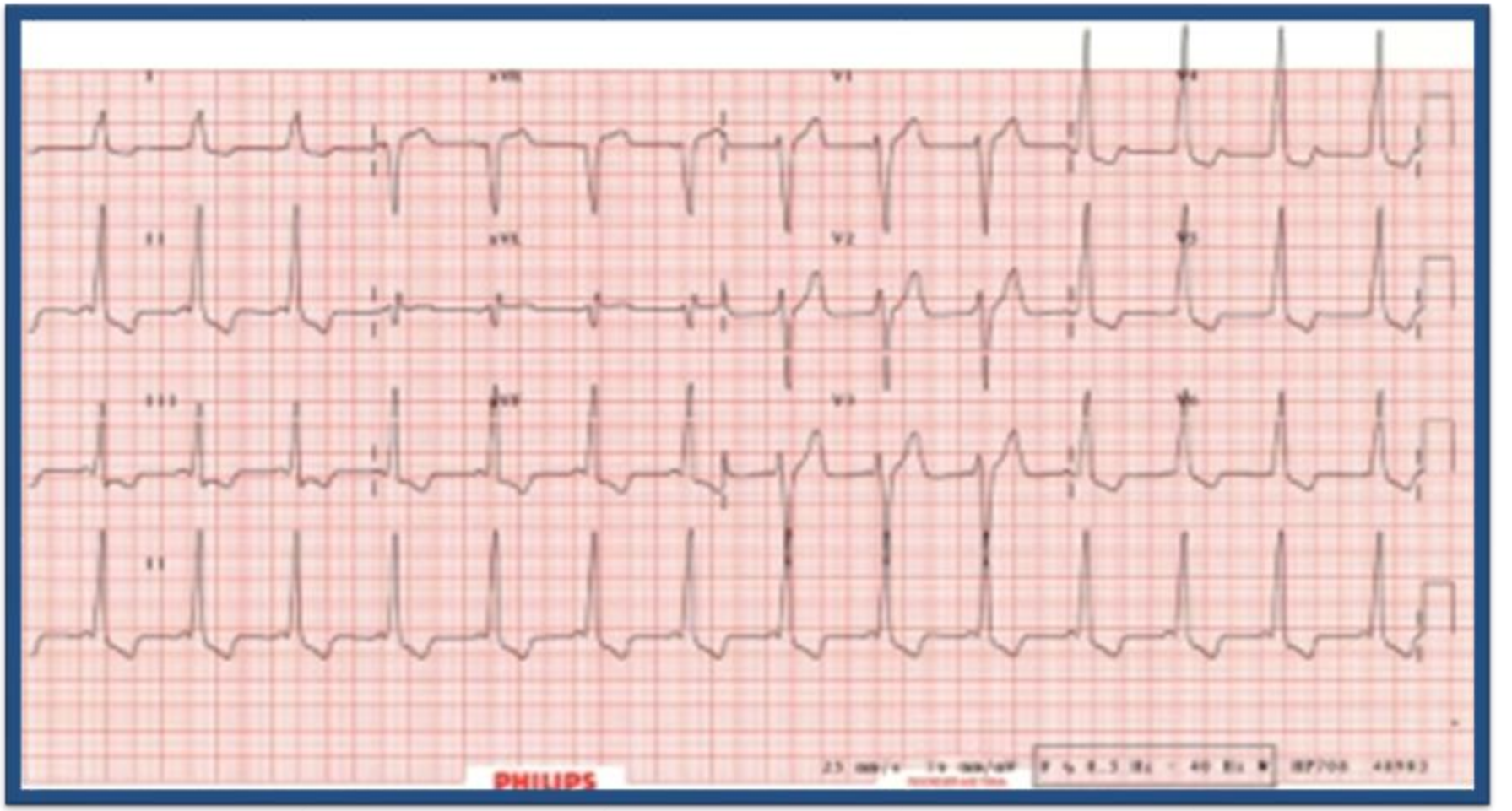
PSVT (narrow QRS with P wave which may or may not be descrinible)

IV adenosine



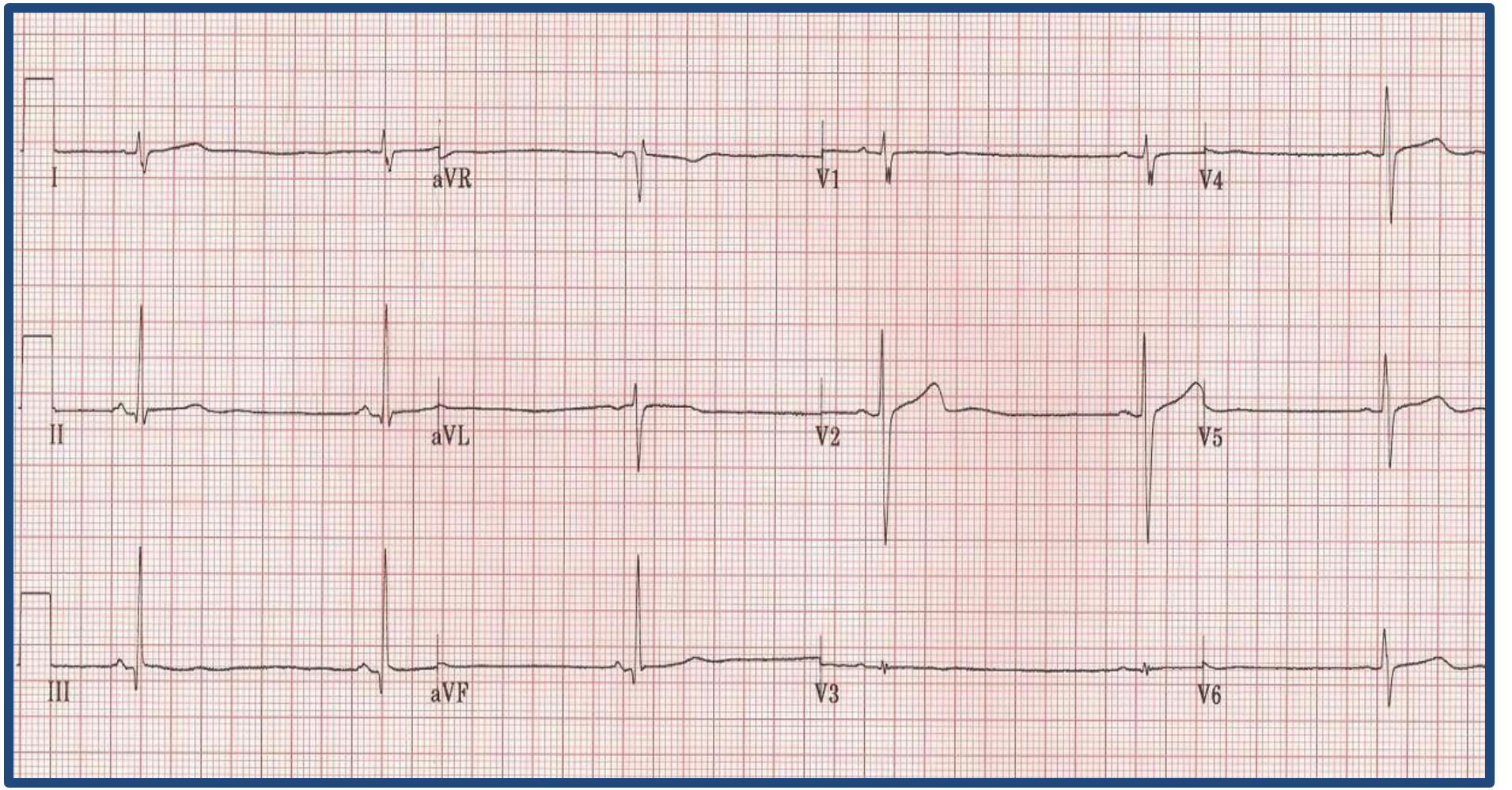
Q: Young female with recurrent episodes of palpitation.
What is the diagnosis?

WPW syndrome



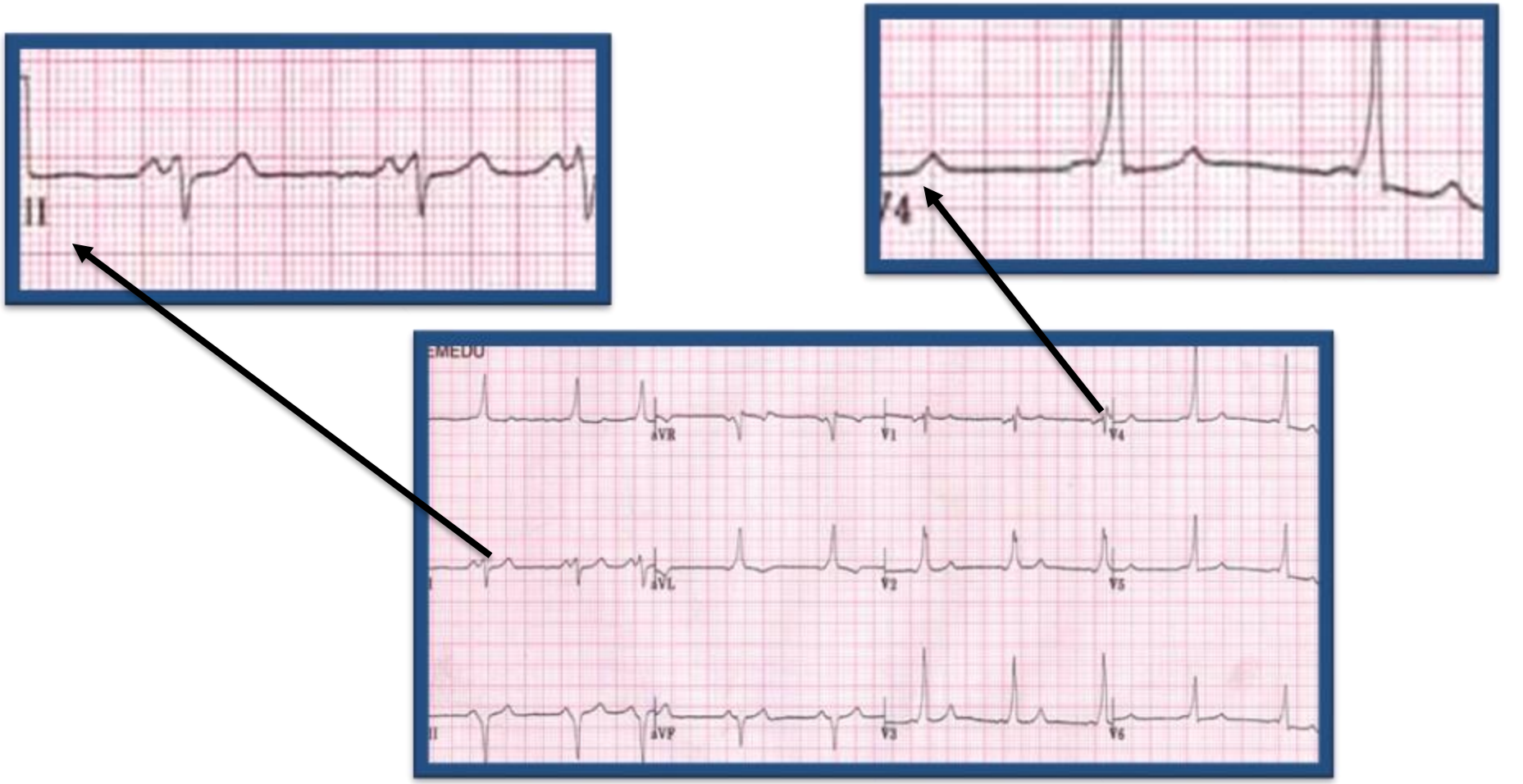
Q: this patient is on anti-hypertensive drugs with impotence, mention the abnormality here and what is the cause ?

Sinus bradycardia & Beta-blockers



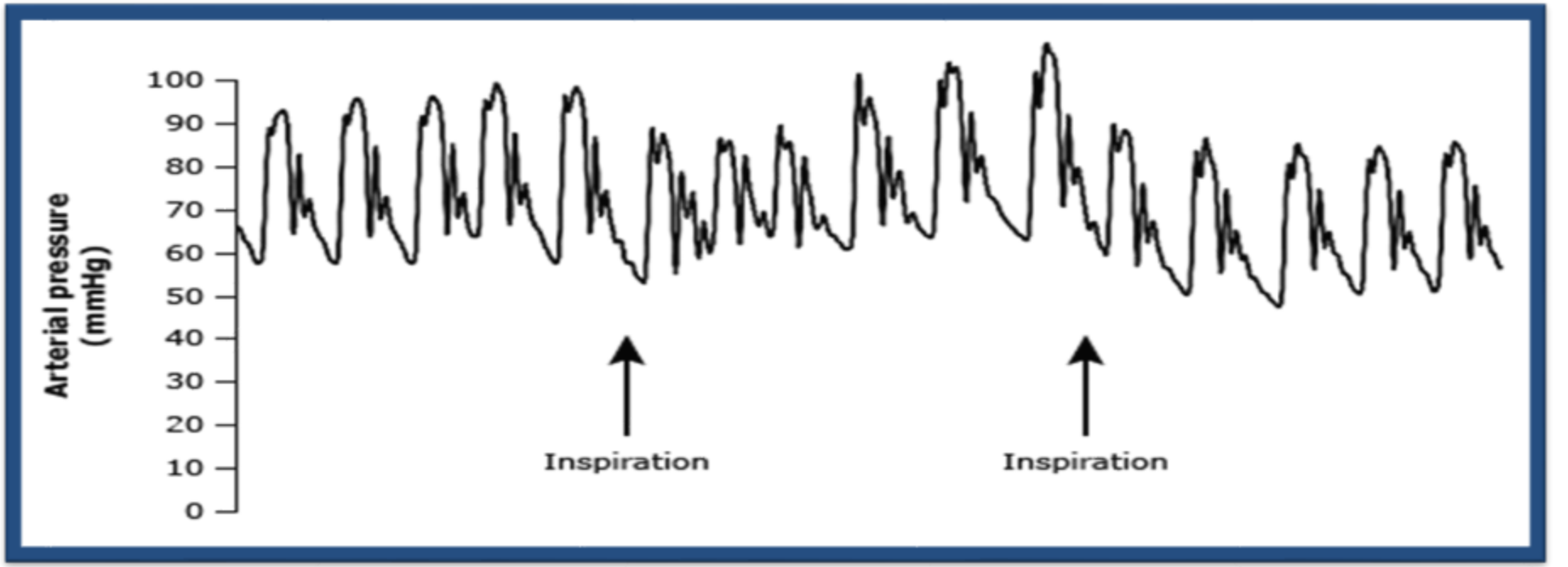
Q: A 19-year-old with recurrent dizziness and chest heaviness episodes.
- What is your diagnosis?

Wolff-Parkinson-White (WPW) syndrome



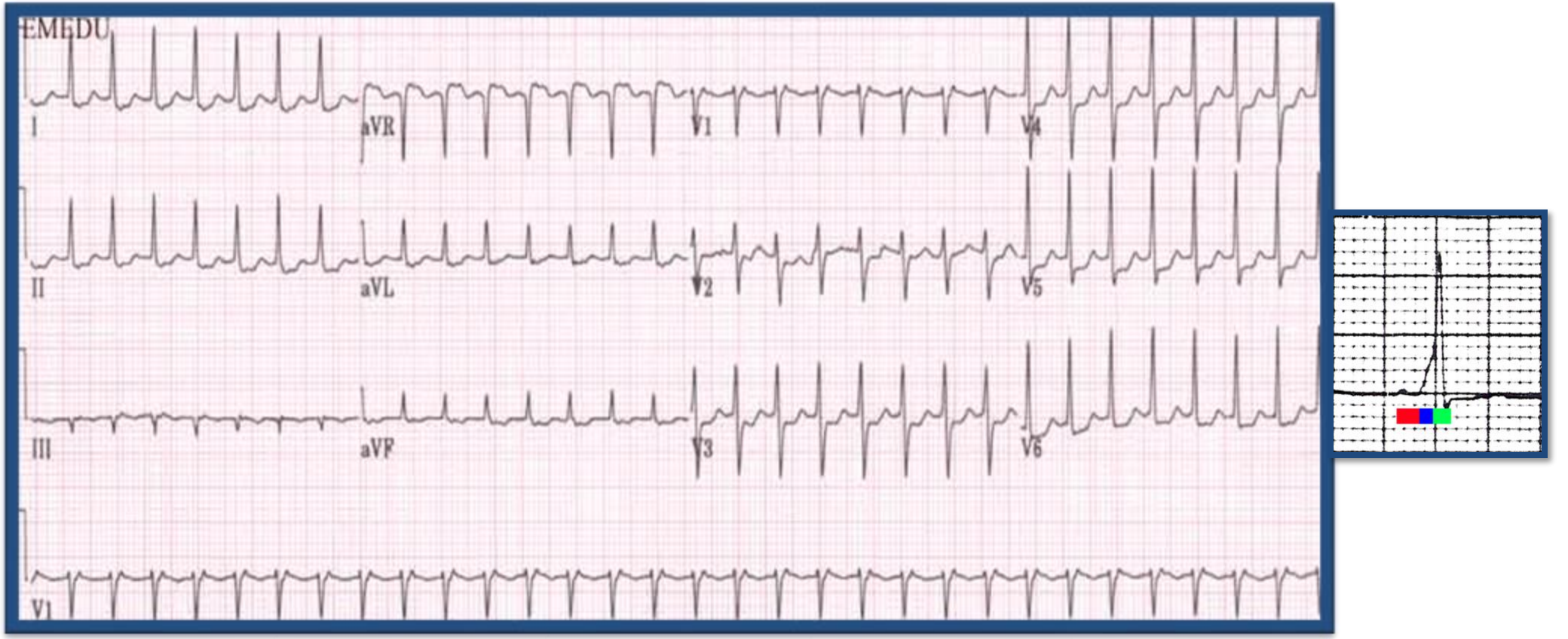
Q: Name the sign seen in this blood pressure wave form in a patient with chronic renal failure presenting with shortness of breath, distant heart sounds and wide mediastinum.

Pulsus paradoxus (is an exaggeration of the normal decrease in systolic BP with inspiration) ddx : cardiac tamponade ,constrictive pericarditis ,asthma ,tension pneumothorax



Q: 30 YO female pt presented to ER complaining of palpitation, What is the cause of her arrhythmia?

WPW'S; The arrhythmia is SVT & small delta wave was just near the ECG so on acute attack you will only find SVT after recovery delta wave can be seen on ECG.

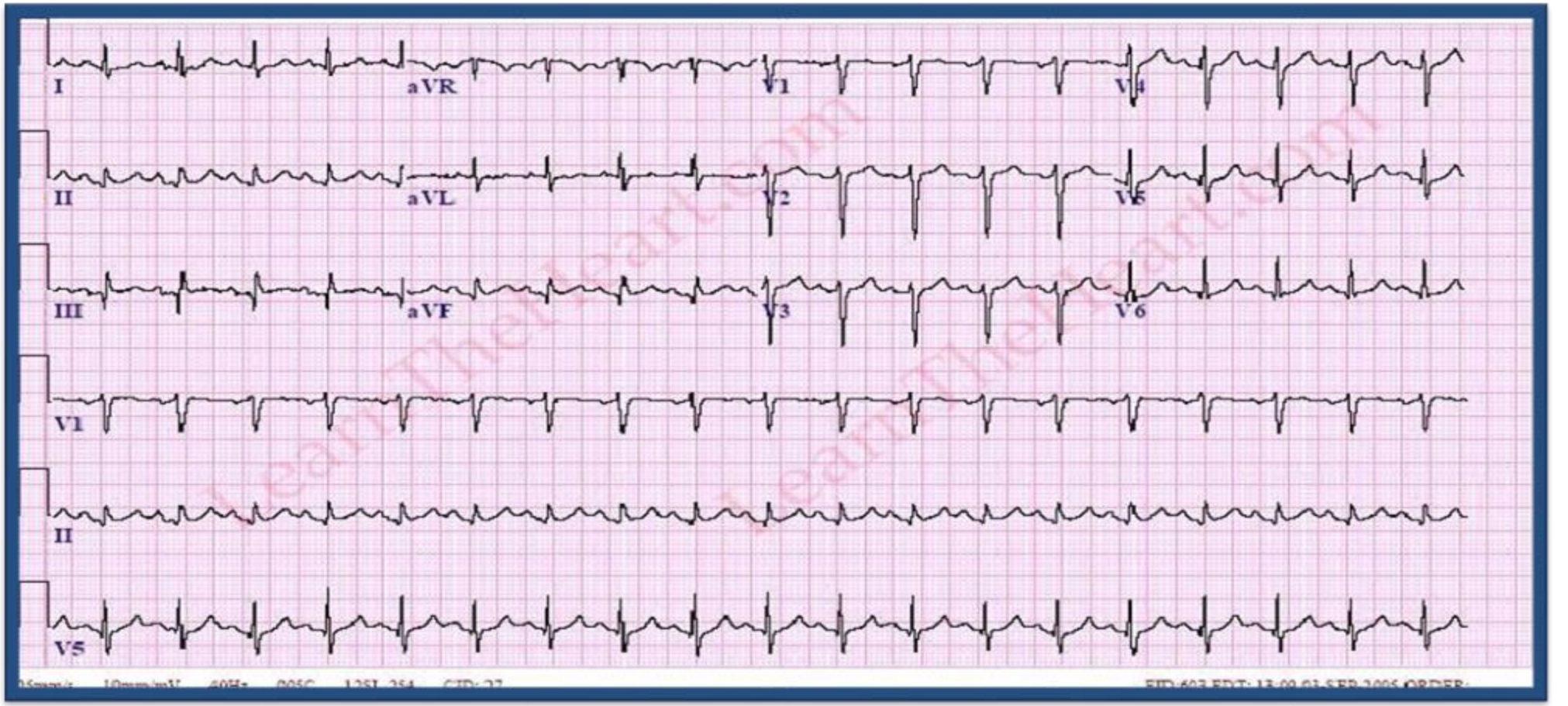


Q: What Is The Dx .

Sinus Tachycardia .

Give 3 Causes .

- Pain
- Fever
- Anxiety
- Dehydration
- Malignant hyperthermia

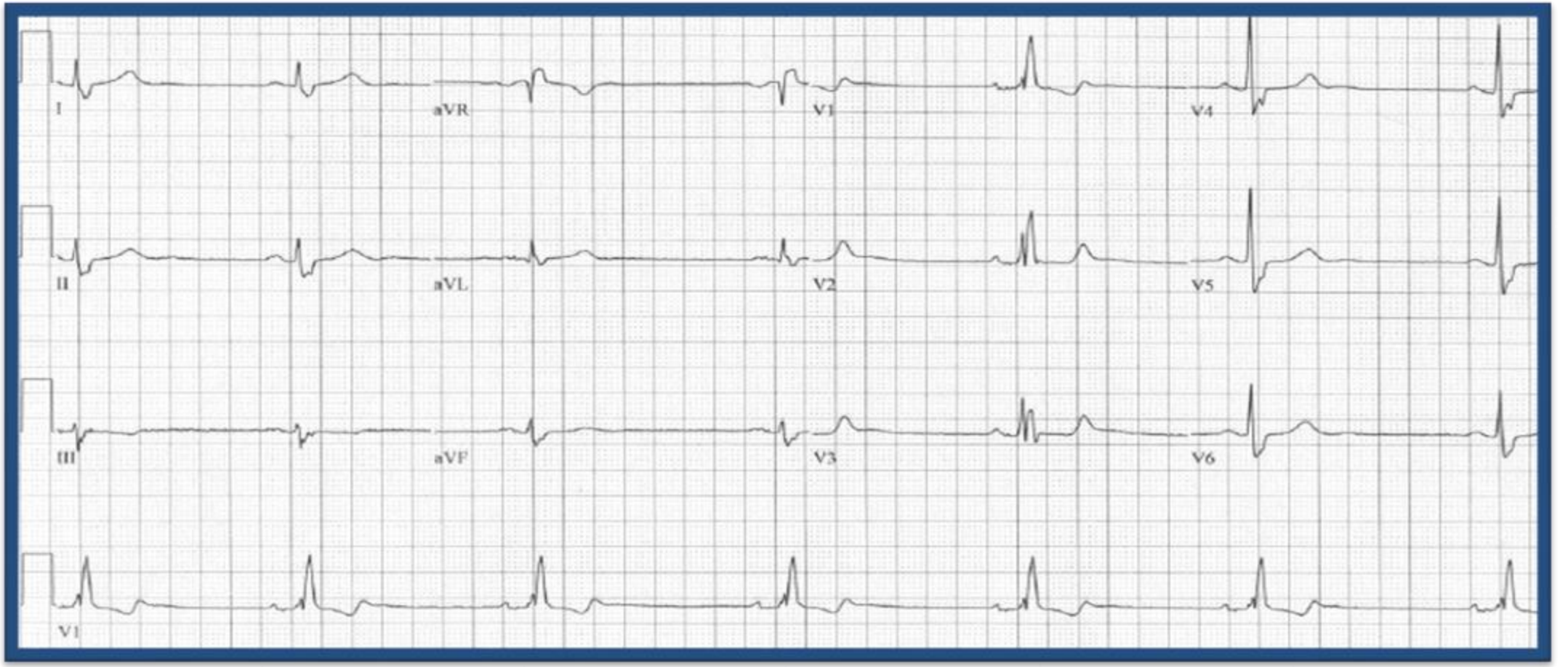


Q: What is diagnosis?

Bradycardia

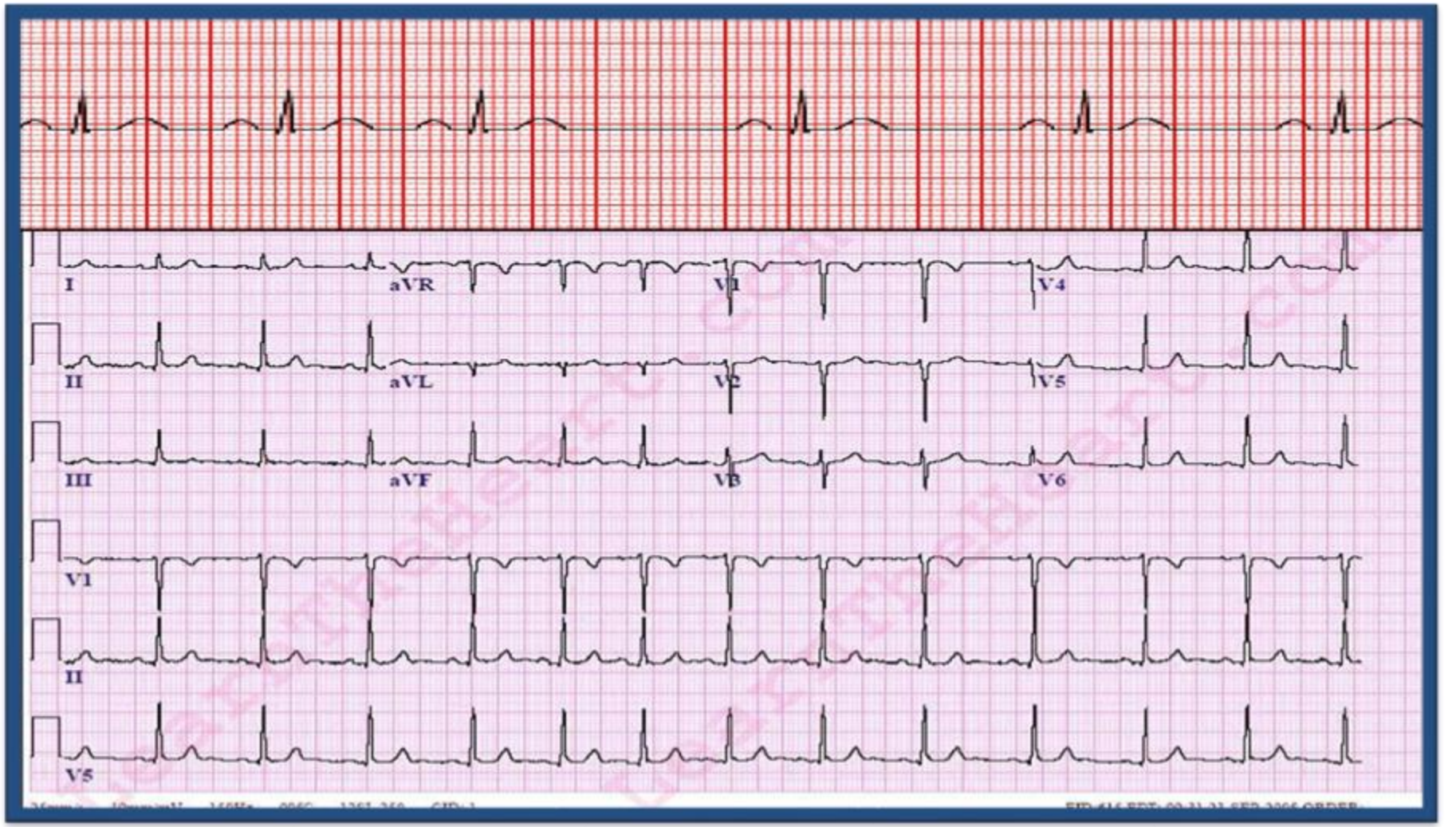
Give 3 causes

Hypothyroidism, patient on β blocker, athletes



Q: What is your diagnosis ?

Sinus Arrhythmia

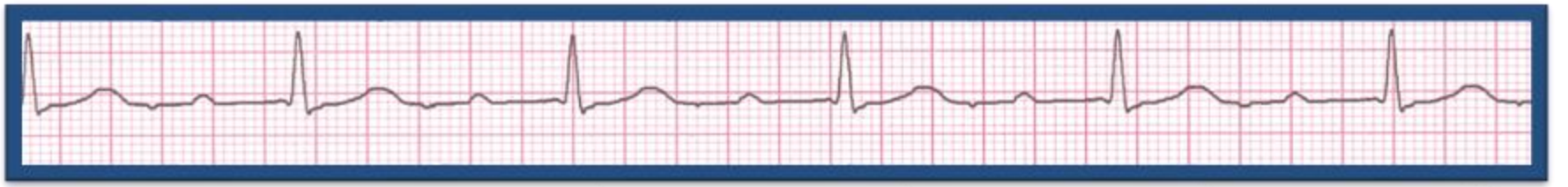


AV Junctional Blocks

AV Nodal Blocks

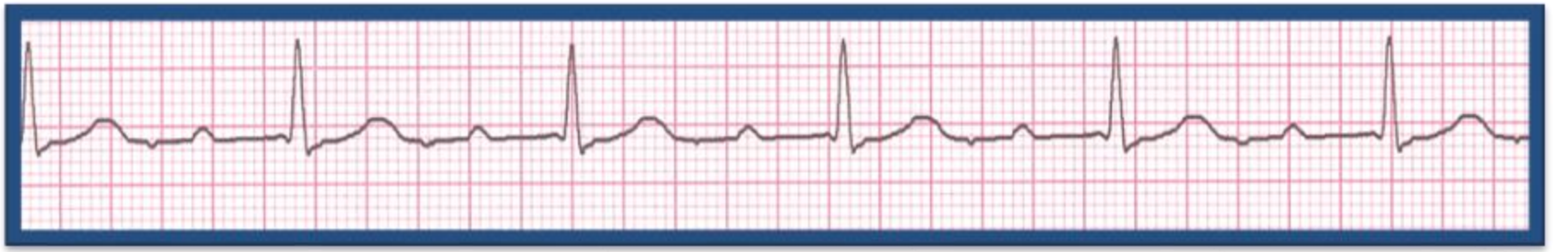
- *1st Degree AV Block*
- *2nd Degree AV Block, Type I*
- *2nd Degree AV Block, Type II*
- *3rd Degree AV Block*

Rhythm #10



60 bpm	Rate
regular	Regularity
normal	P waves
0.36 S	PR interval
0.08 s	QRS duration
1st Degree AV Block	Interpretation

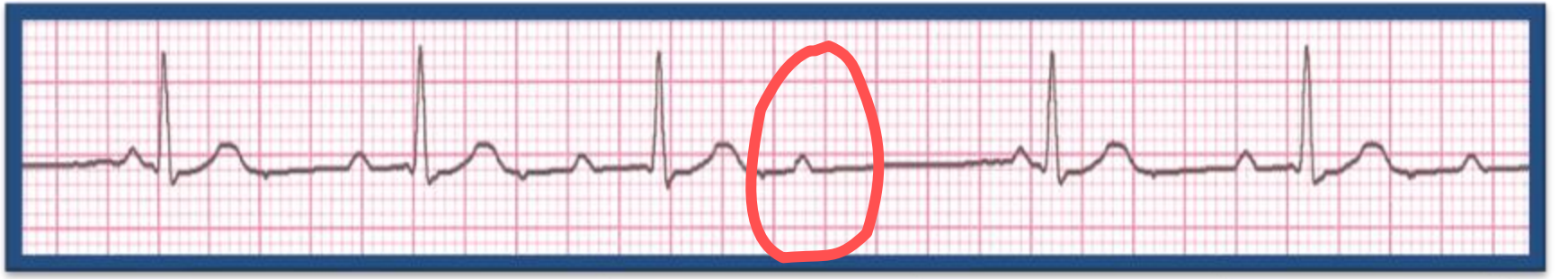
1st Degree AV Block



- A first degree AV node block occurs when conduction through the AV node is slowed, thus delaying the time it takes for the action potential to travel from the SA node, through the AV node, and to the ventricles. A first degree AV block is indicated on the ECG by a prolonged PR interval.
  - PR Interval > 0.20 s (greater than 5 small boxes)

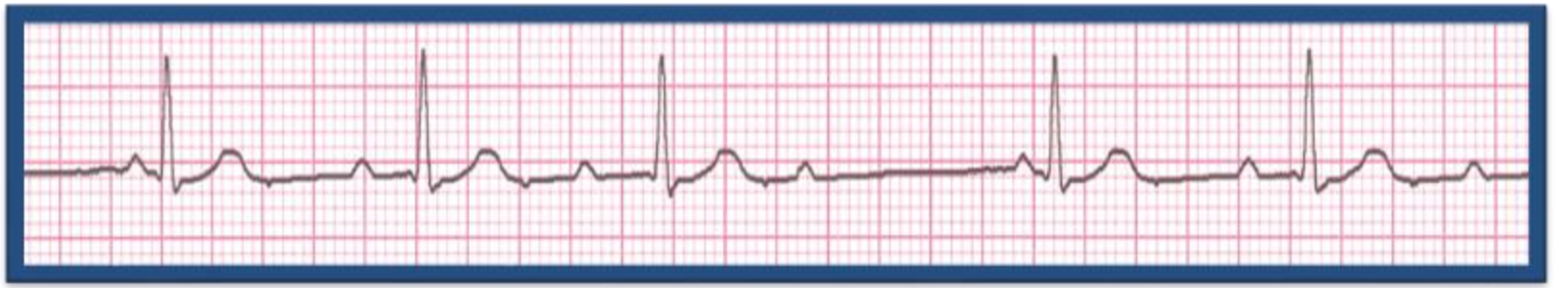
- Etiology: Prolonged conduction delay in the AV node or Bundle of His.

Rhythm #11



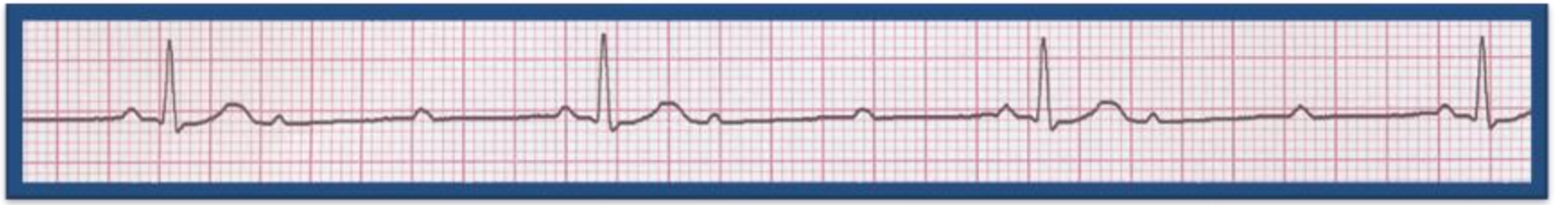
50 bpm	Rate
regularly irregular	Regularity
normall, but 4th no QRS	P waves
lengthens	PR interval
0.08 s	QRS duration
2nd Degree AV Block, Type I	Interpretation

2nd Degree AV Block, Type I



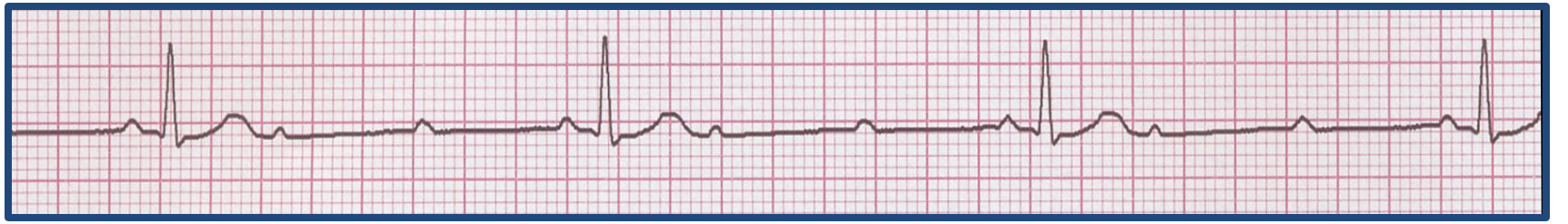
- Deviation from NSR
  - PR interval progressively lengthens, then the impulse is completely blocked (P wave not followed by QRS).
- Etiology: Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rd or 4th) fails to make it through the AV node

Rhythm #12



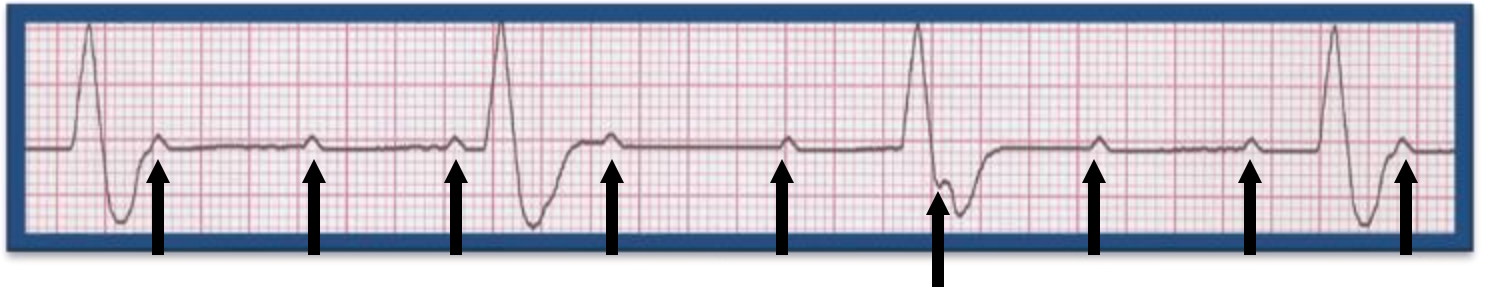
40 bpm	Rate
regular	Regularity
normal, 2 of 3 no QRS	P waves
0.14 s	PR interval
0.08 s	QRS duration
2nd Degree AV Block, Type II	Interpretation

2nd Degree AV Block, Type II



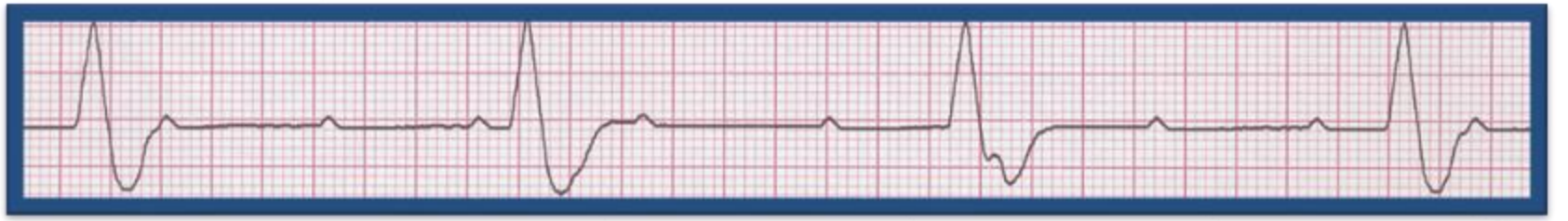
- Deviation from NSR
  - Occasional P waves are completely blocked (P wave not followed by QRS).
- Etiology: Conduction is all or nothing (no prolongation of PR interval); typically block occurs in the Bundle of His.

Rhythm #13



40 bpm	Rate
regular	Regularity
no relation to QRS	P waves
none	PR interval
wide (> 0.12 s)	QRS duration
3rd Degree AV Block	Interpretation

3rd Degree AV Block

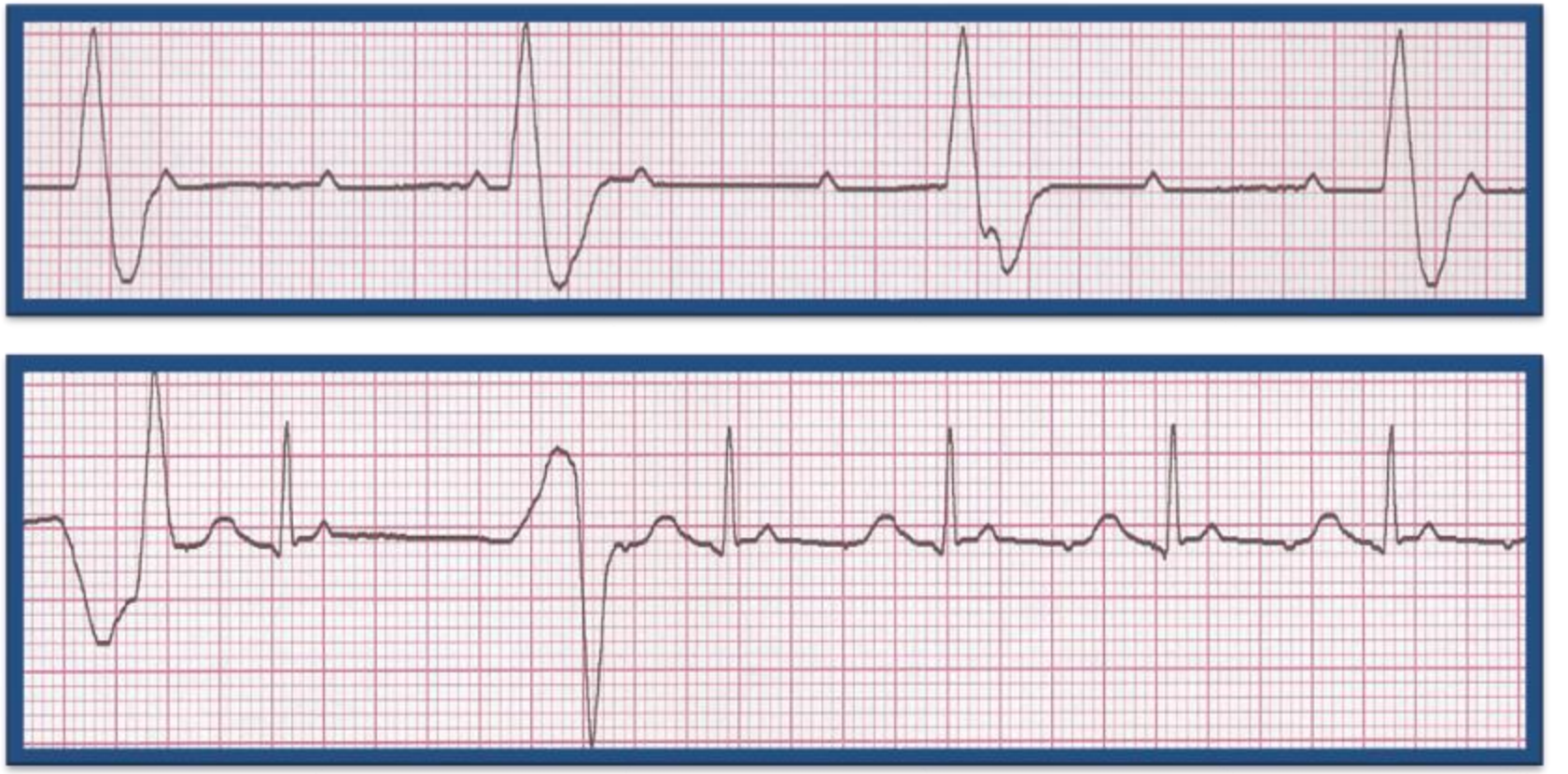


- Deviation from NSR
  - The P waves are completely blocked in the AV junction; QRS complexes originate independently from below the junction.

- Etiology: There is complete block of conduction in the AV junction, so the atria and ventricles form impulses independently of each other. Without impulses from the atria, the ventricles own intrinsic pacemaker kicks in at around 30 - 45 beats/minute.

Remember

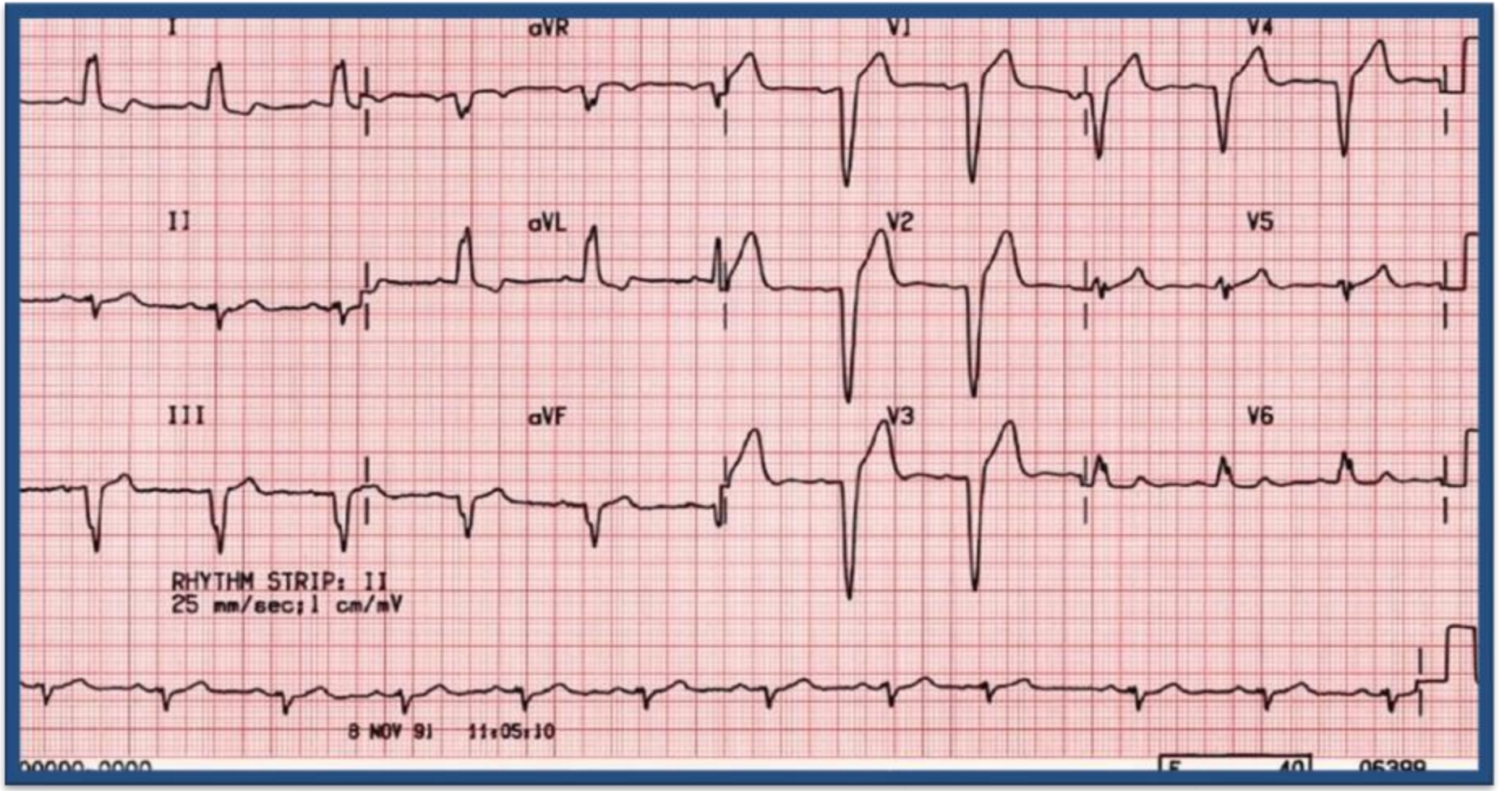
- When an impulse originates in a ventricle, conduction through the ventricles will be inefficient and the QRS will be wide and bizarre.



Q: What's the main abnormality in this ECG?

Left bundle branch block

(Notice the M shape of the QRS complex in V6).

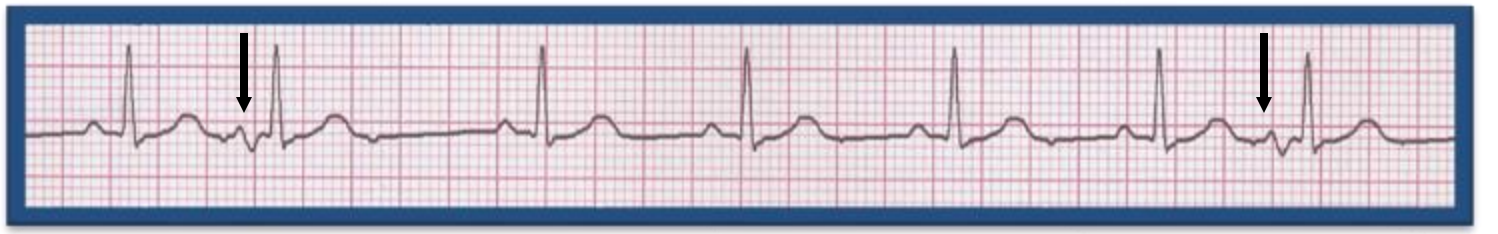


- Premature Beats

  - *Premature Atrial Contractions (PACs)*

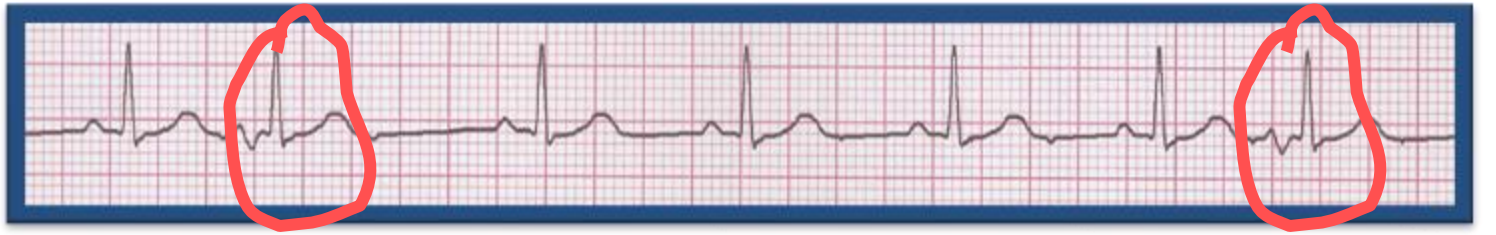
  - *Premature Ventricular Contractions (PVCs)*

Rhythm #3



70 bpm	Rate
occasionally irreg.	Regularity
2/7 different contour	P waves
0.14 s (except 2/7)	PR interval
0.08 s	QRS duration
NSR with Premature Atrial Contractions	Interpretation

Premature Atrial Contractions

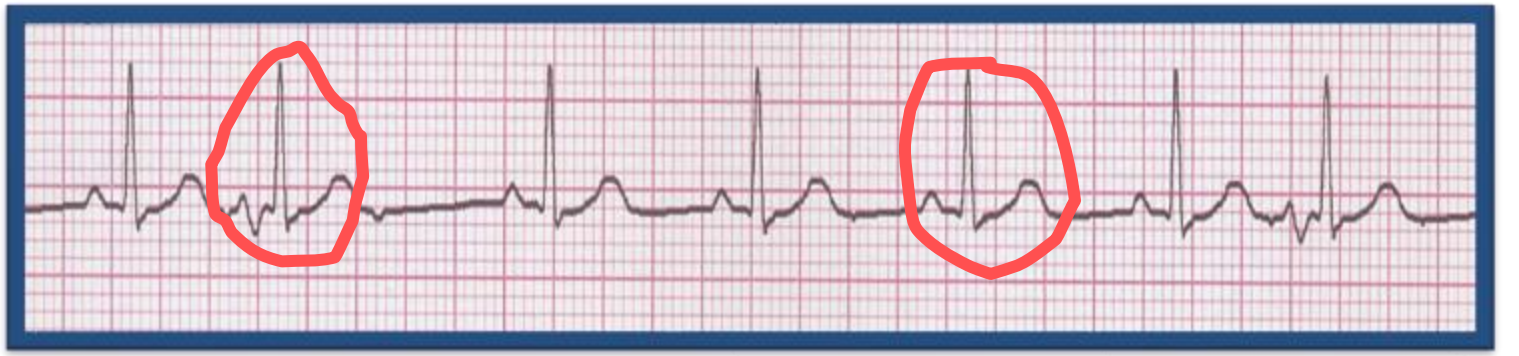


- Deviation from NSR
  - These ectopic beats originate in the atria (but not in the SA node), therefore the contour of the P wave, the PR interval, and the timing are different than a normally generated pulse from the SA node.

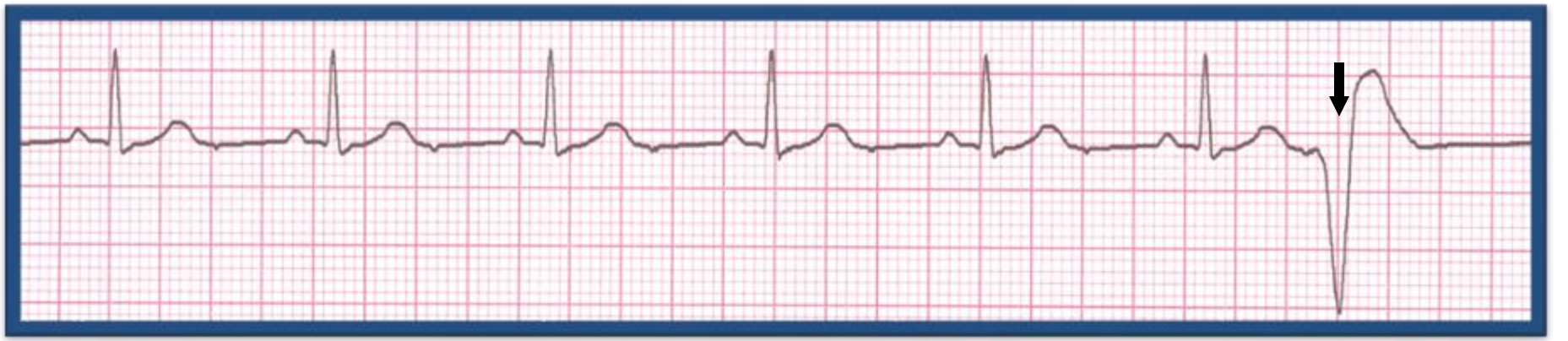
- Etiology: Excitation of an atrial cell forms an impulse that is then conducted normally through the AV node and ventricles.

Teaching Moment

- When an impulse originates anywhere in the atria (SA node, atrial cells, AV node, Bundle of His) and then is conducted normally through the ventricles, the QRS will be narrow (0.04 - 0.12 s).

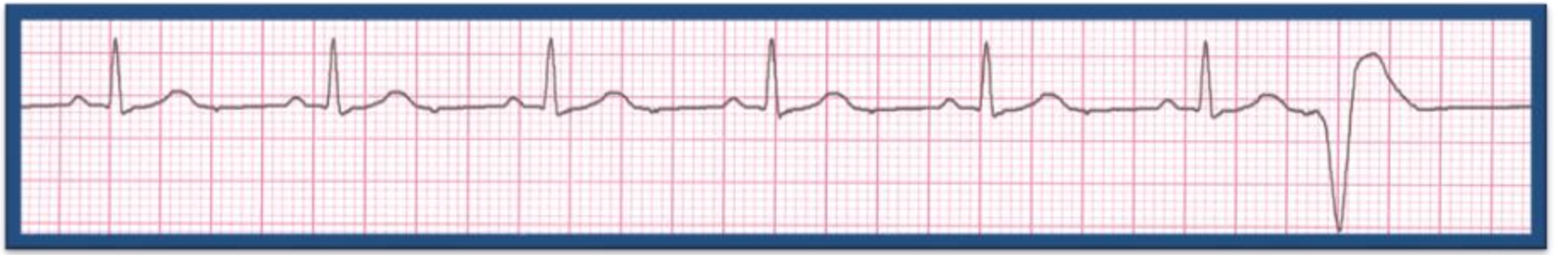


Rhythm #4



75 bpm	Rate
occasionally irreg.	Regularity
none for 7th QRS	P waves
0.14 s	PR interval
0.08 s (7th wide)	QRS duration
Sinus Rhythm with 1 PVC	Interpretation

PVCs



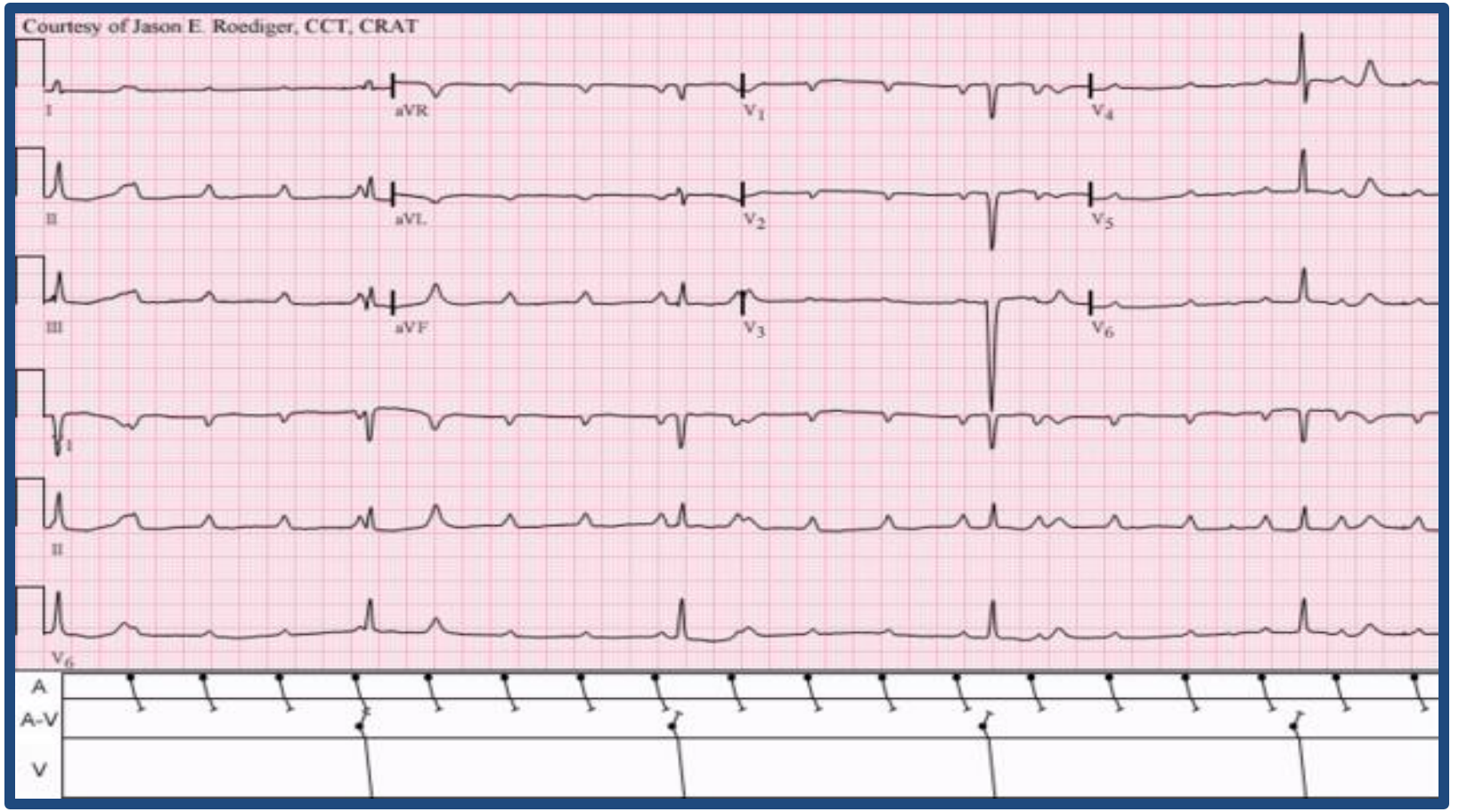
- Deviation from NSR
  - Ectopic beats originate in the ventricles resulting in wide and bizarre QRS complexes.
  - When there are more than 1 premature beats and look alike, they are called "uniform". When they look different, they are called "multiform".

- Etiology: One or more ventricular cells are depolarizing and the impulses are abnormally conducting through the ventricles.

Q: This ECG is for a 70 YO pt presented with recurrent attacks of dizziness.

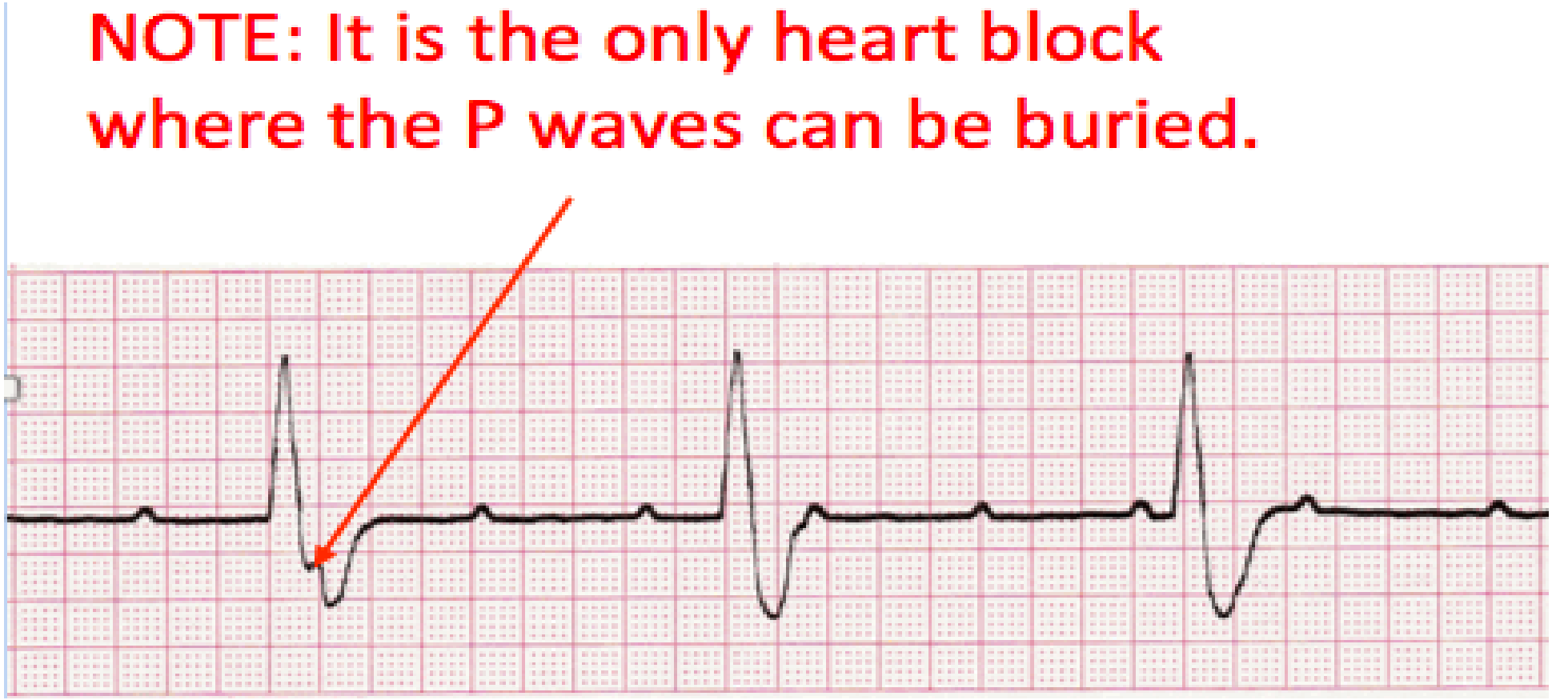
What's your Dx?

Third degree (complete) heart block.



Q: What is the diagnosis?

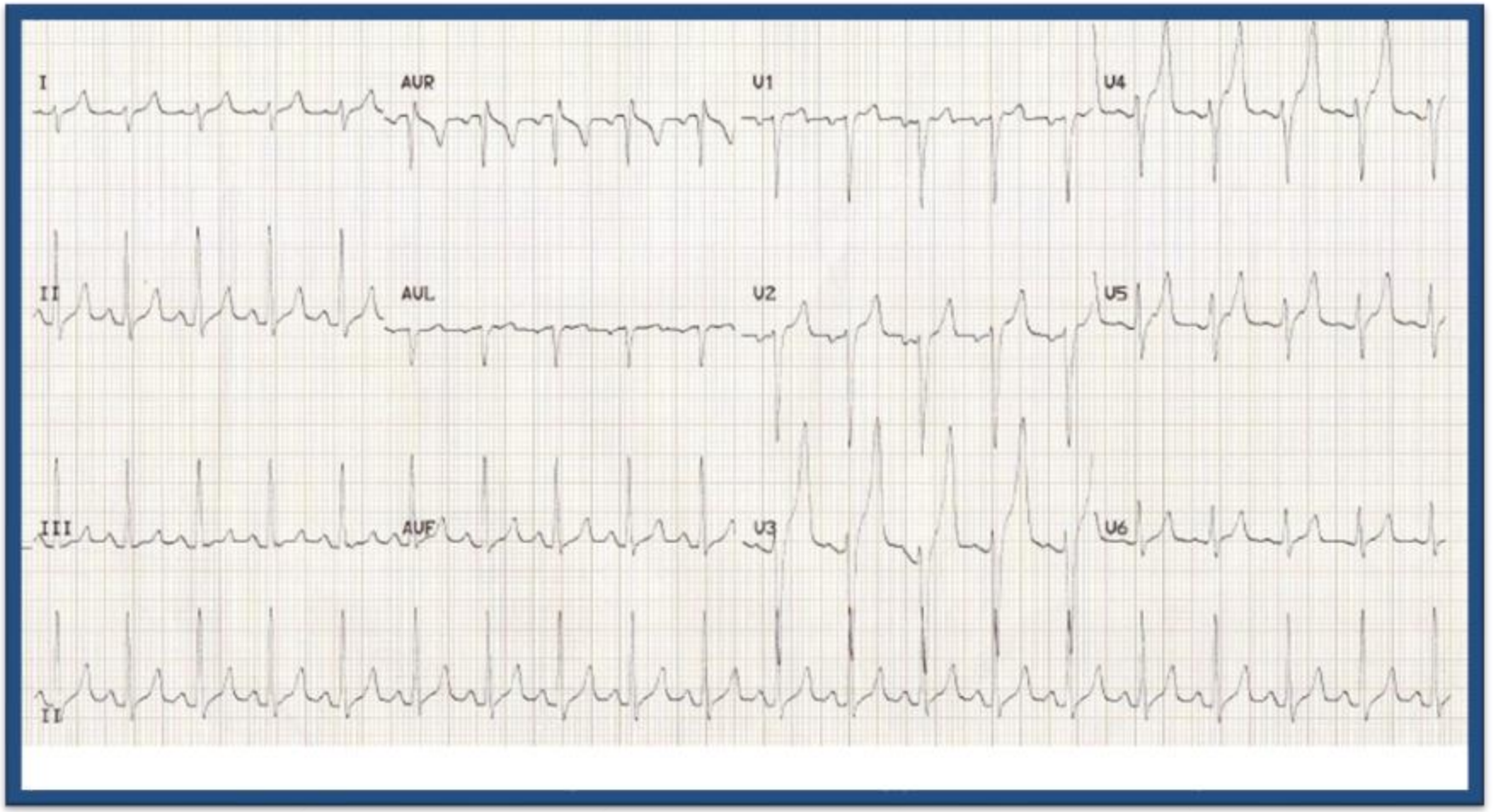
3rd degree heart block



POTASIUM Disturbance

Q: Patient with chronic renal failure presented with chest pain, what is the biochemical test you have to do?

Serum Potassium.

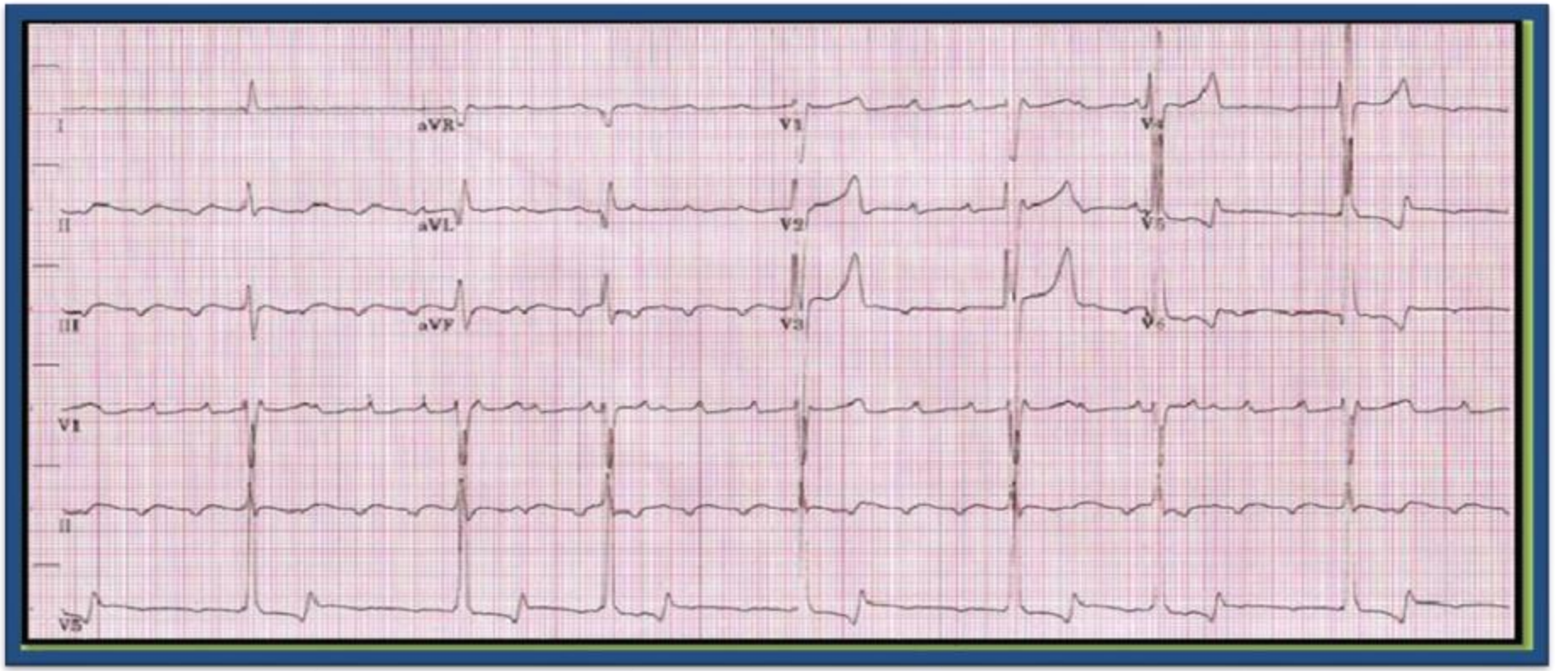


Q: This ECG is for a known case of chronic renal failure, what is your spot Dx?

Hyperkalemia

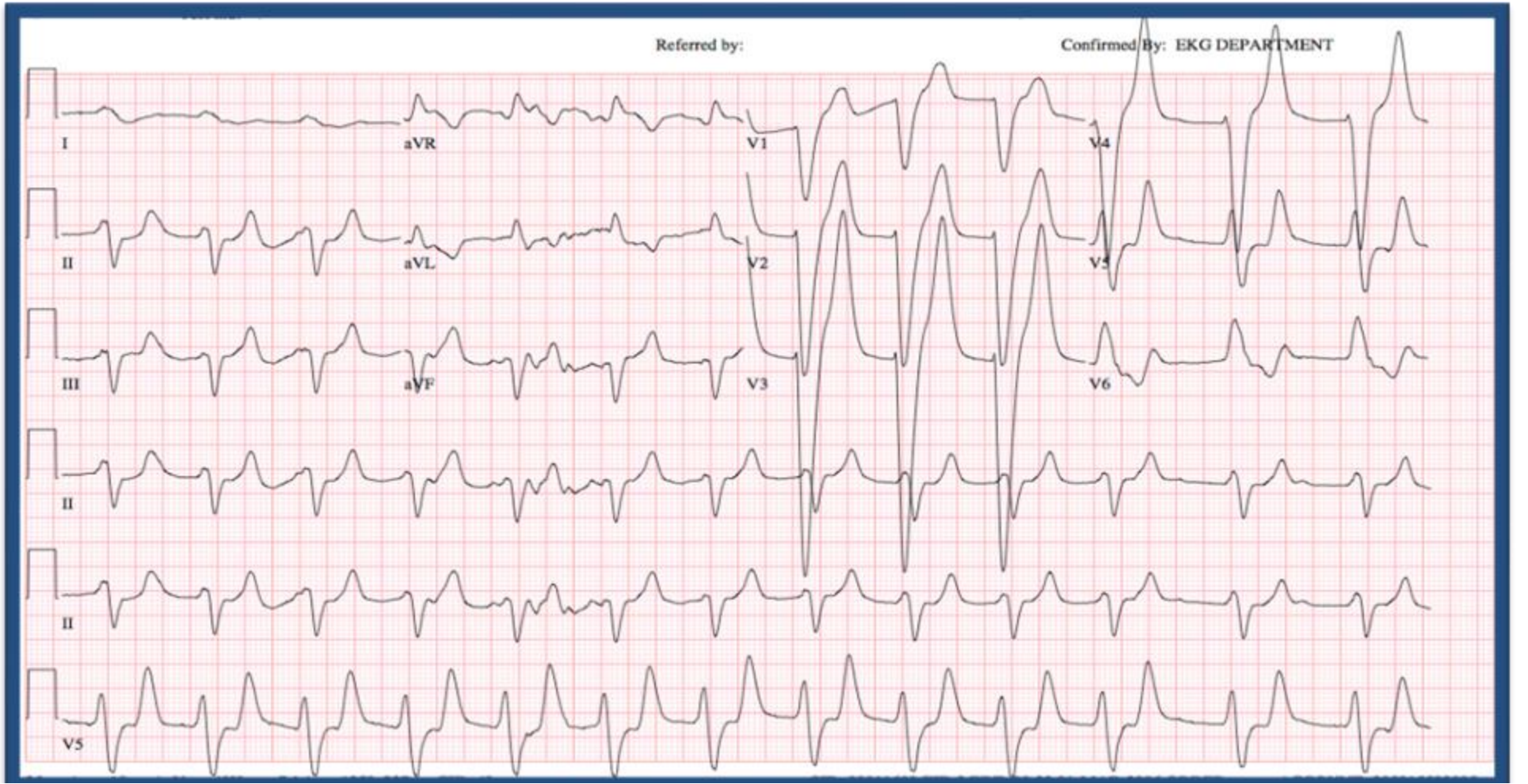
what is the most emergency ttt?

IV calcium gluconate.



Q:The patient has HTN on ACEI. Mention 2 abnormalities in this ECG and what in the underlying cause?

Wide QRS / peaked (hyperacute) T wave, Hyperkalemia (caused by ACEI)

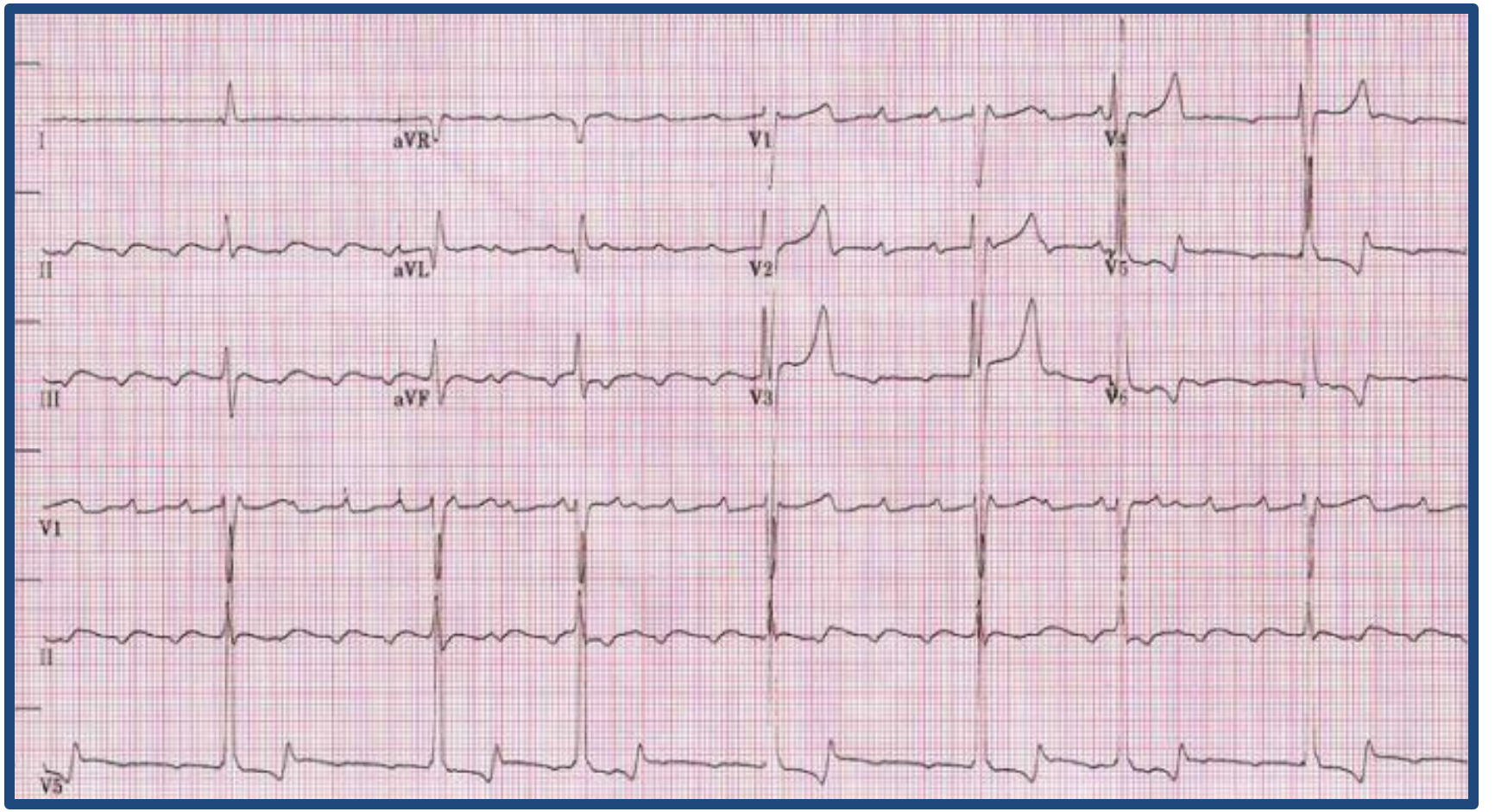


Q: This ECG is for a known case of chronic renal failure, what is your spot Dx? what is the most emergency tt?

Hyperkalemia / IV calcium gluconate.

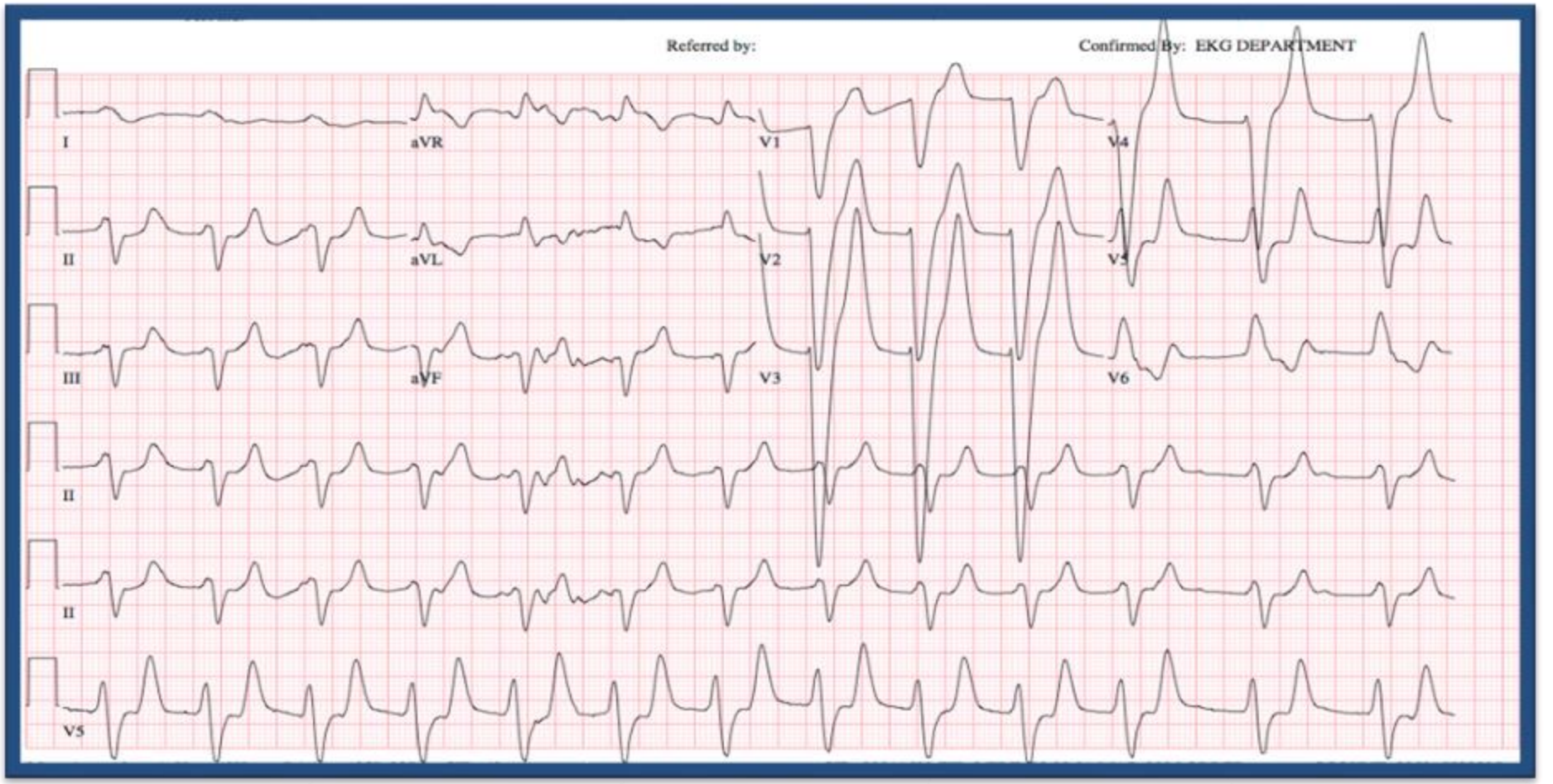
Q: known case chronic renal failure on dialysis, what is the cause of his ECG changes ?

hyperkalemia



Q: The patient has HTN on ACEI. Mention 2 abnormalities in this ECG and what in the underlying cause?

Wide QRS / peaked (hyperacute) T wave
Hyperkalemia (caused by ACEI)



Q: 60 YO DM pt with chronic dialysis came with this EKG.

1- Give 2 abnormalities in this EKG ?

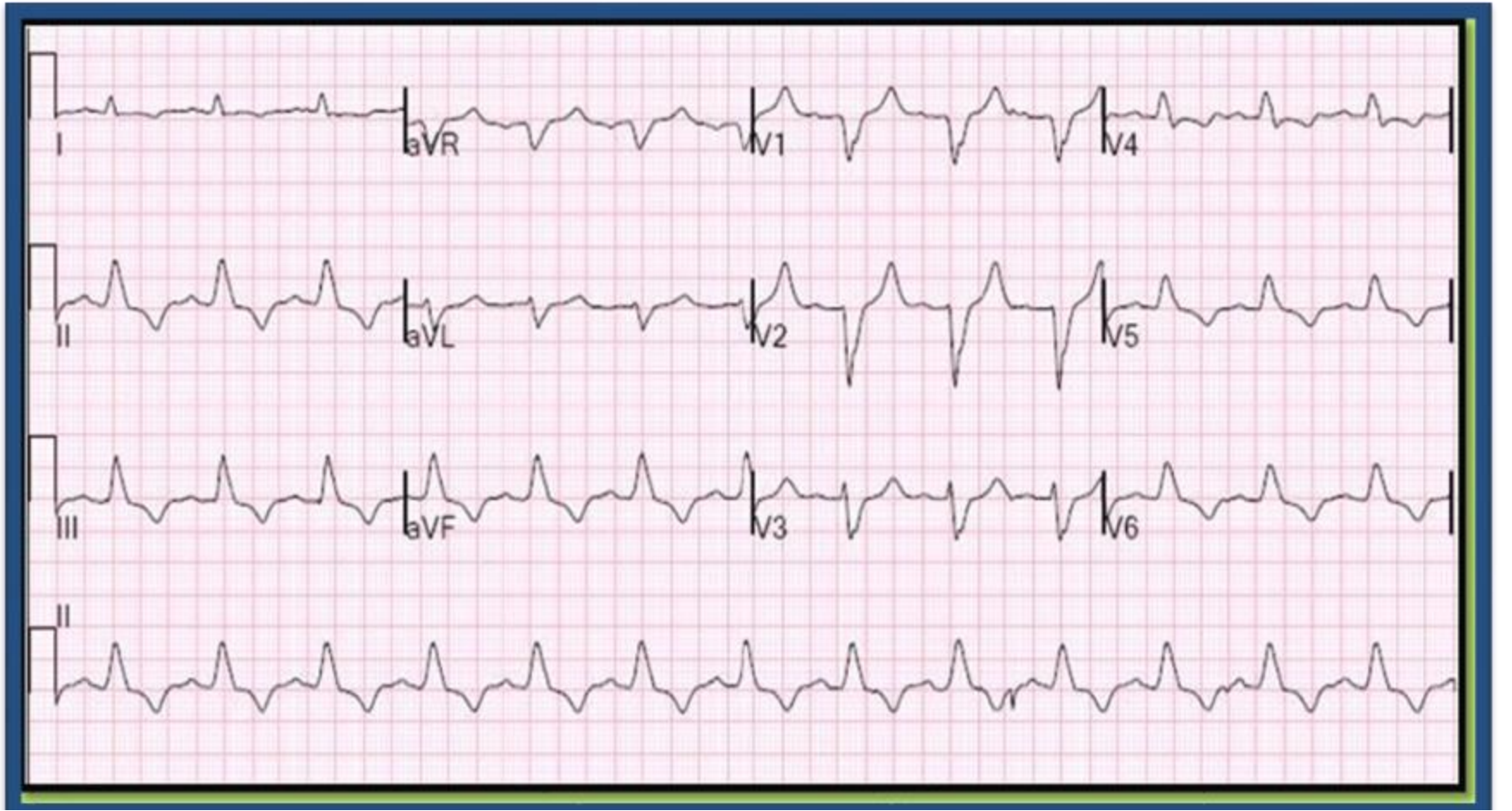
hyper acute T-waves , Wide QRS.

2- what is the cause of this EKG ?

Hyperkalemia.

3- Give 2 line of treatment.

Ca gluconate , Glucose & IV insulin.



Drug adverse effect and heart failure

Important notes for HF

- NYHA classification:

Class I: cardiac disease without limitation of physical activity

Class II: slight limitation of normal physical activity (fatigue, palpitation, dyspnea, angina)

Class III: marked limitation of physical activity(slight activity causes symptoms.

Class IV: symptom present at rest. Unable to carry on any physical activity without discomphort

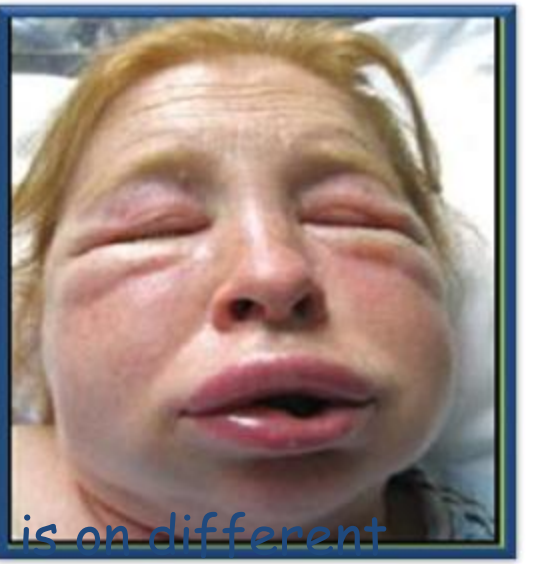
2013 ACC/AHA staging system

- STAGE A: patient at high risk for HF without structural heart disease or symptoms of HF like patient with HTN, atherosclerotic disease, dm, obesity, metabolic syndrome, cardiotoxic drugs, family hx of cardiomyopathy
- Stage B: patient with structural heart disease without signs or symptoms of Hf like patient with previous MI, LVH, asymptomatic valvular disease
- Stage C: structural heart disease with prior or current symptoms of HF (patients in stage B with symptoms)
- Stage D: marked symptoms at rest and frequent hospitalization despite medical therapy

Q1 : patient with HTN on treatment presented with face swelling.

1-According to ACC/AHA staging system of HF what is the stage of hf in this patient?

Stage A



2-What is the Dx.?

Angioedema

3-What is the cause of this?

Side effect of ACEI -(Drug-induced)

4-First step in management?

Airway monitoring

Q2: 52 years old male Patient with HX of previous MI and HF, he is on different drugs, now he has elevated creatine kinase

1) What type of drugs may be the cause ?

Statin use

2) Also electrolyte of patient show hypokalemia, high HCo3 .Give 2 DDx ?

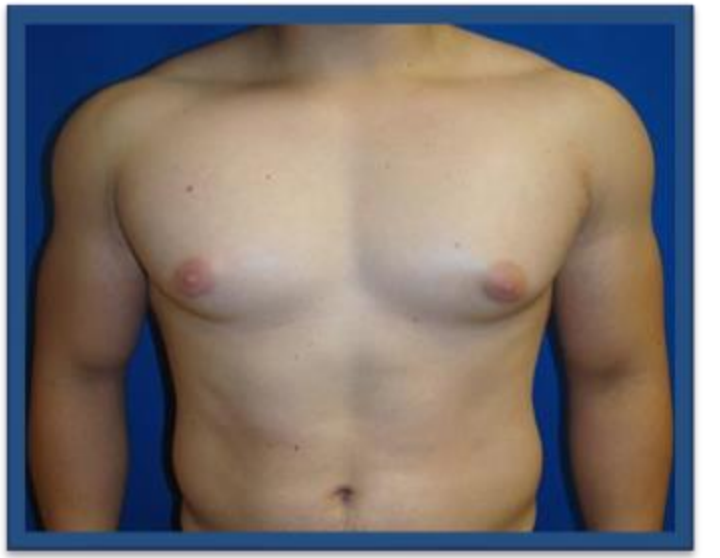
1. Metabolic alkalosis.
2. Diuretic Use.

3) What type of diuretics can be used in this patient if hypokalemia caused by drug?

potassium sparing diuretics

Q3: 30 years old male, name 2 drugs that can cause this condition.

Digoxin
Spironolactone



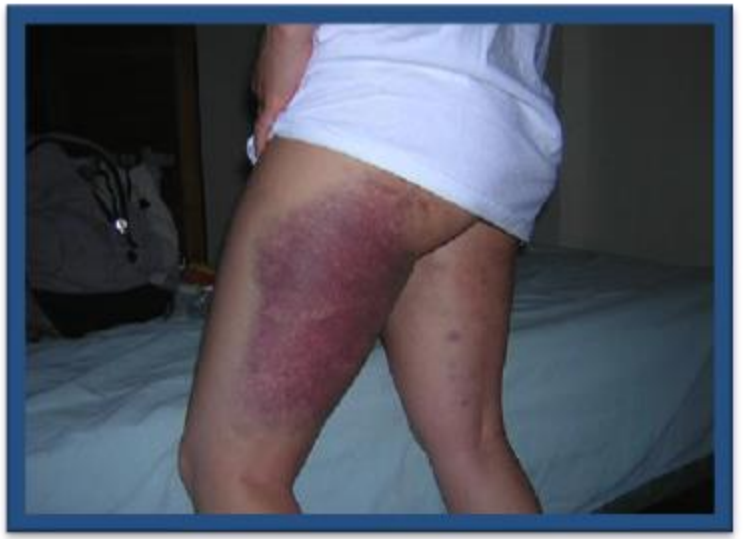
Q4: This patient with a prosthetic valve, developed this skin lesion.

A-What is the cause?

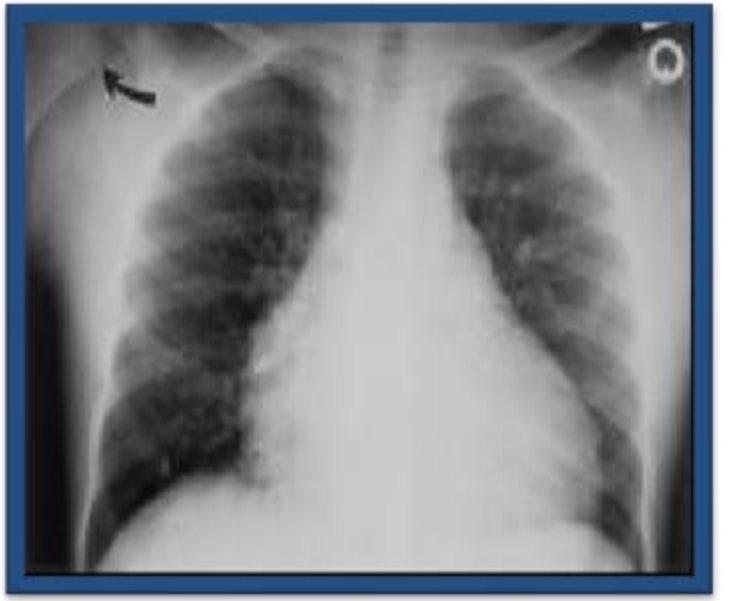
Warfarin overdose

B-What is the appropriate lab investigation?

INR



Q5 : this picture shows chest xray for 26 year old Patient



1)Write 3 Findings in this CXR.

1. Cardiomegaly.

2. Pulmonary infiltration.

3. Right-tracheal deviation.

2) What is the cause of these findings in this age?

Cardiomyopathy (but CHF in elderly patients)

Q6:Hx of a hospitalized patient with HTN , DM underwent cardiac catheterization , taking multiple medications , a contrast CT was done to him , presented with Acute kidney injury

- Mention 3 causes of hospital induced renal failure

  ATN (ischemia), Contrast nephropathy , AIN (drugs)

- True or False about Kidney Injury Molecule 1 (KIM-1)

1- novel biomarker for human renal proximal tubule injury True

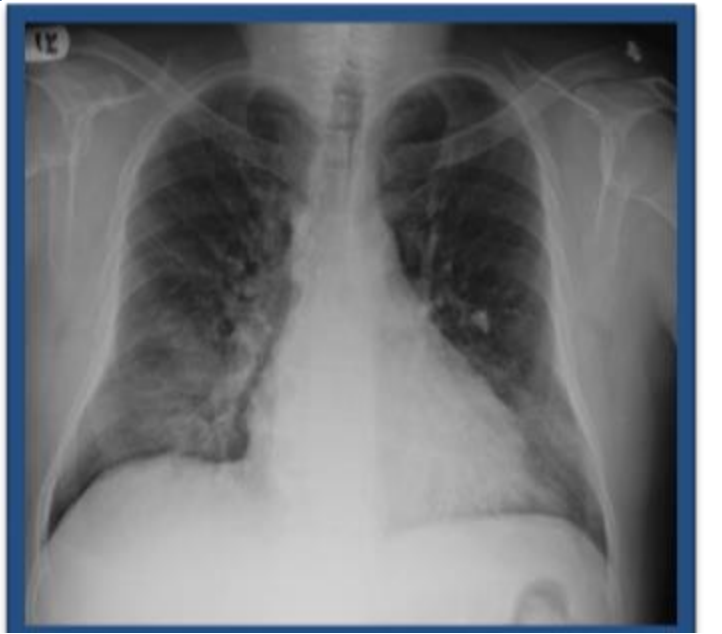
2-not affected by UTI or chronic kidney failure True

3- not affected by cardiac catheterization False

Q7: Mention 4 causes of this condition.

Heart failure
Renal failure, Nephrotic syndrome
Liver cirrhosis
Hypo-albuminemia
Fluid overload

Q8: A 65 years old male complaining of SOB this is his CXR:



- Two findings in the CXR .

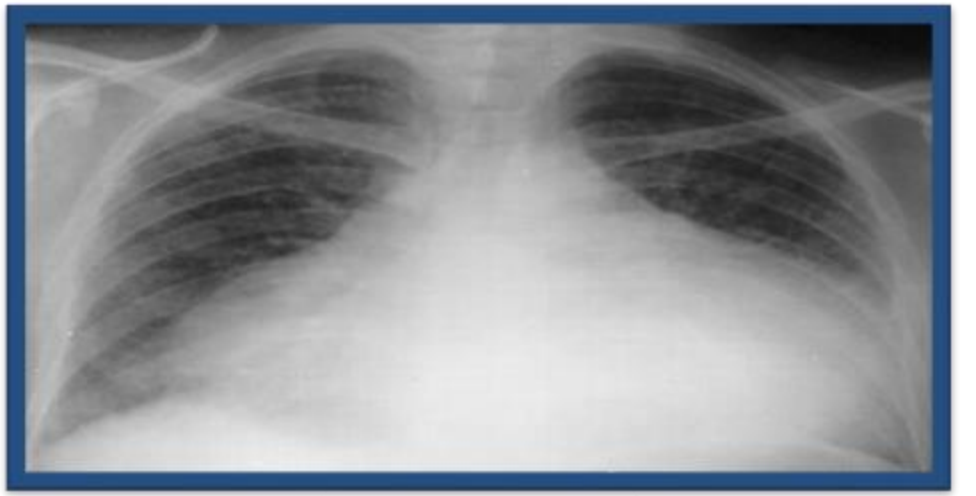
  Cardiomegaly .

  Kerly B lines

- What is your diagnosis?

  Left side heart failure

Q9 :The chest radiography of a patient Name two clinical findings on physical examination supporting the most likely diagnosis.



- Distant heart sounds
- Raised JVP
- Dilated veins
- Pulsus paradoxus

Q10: Mr.x is a known case of heart failure for 2 years presenting with increasing dyspnea and Shock (BP 80/40, and HR 130 b/m),

1) what is your diagnosis?

Sever ventricular failure

2) How to manage this patient?

Inotrops(dopamine, dobutamine, milrinone)

Mechanical circulatory support

3) What is the appropriate dose of dopamine in this case?

2-5 microgram/kg/min (this dose activate beta receptors)

Q11: Known to have HTN & IHD for long time came with SOB, orthopnea, crepitating & S3 gallop sound.

1) what is your Dx ?

acute heart failure.

2) investigations ?

x ray & echo.

3) 2 lines for the treatment?

Position and oxygen // Diuretics (IV lazix).

Coronary artery disease

Stable Angina

- Typical anginal chest pain :

  retrosternal, worse with exertion, better with rest or nitroglycerin not related to breathing or movement lasting < 15 minute
- the pathology here :

  fixed atherosclerotic lesion + increase in myocardial oxygen demand
- Management :

  1. risk factor modification , 2. aspirin, 3. b blockers 4. nitrates , 5. CCB only if b blockers and nitrates are not effective
- If ECG changes present (ST depression, abnormal T) Rx as unstable angina

Unstable angina

- Pathophysiology :

  decrease in the myocardial supply due to decrease in resting coronary flow

- Unstable angina :

  any anginal pain at rest or new onset angina that is worsening or chronic angina that is increasing in intensity, frequency or duration

How to diagnose CAD?

- ECG
- Cardiac enzymes
- Stress ECG (ST depression , onset of HF, Arrhythmia, Hypotension)
- Stress echocardiography (looking for wall motion abnormalities)
- Myocardial perfusion study
- Holter monitoring (for silent MI, arrhythmias)
- Cardiac catheterization

Case

72 YO male come to ER with chest pain for 30 min prior to admission.

Q1:what are the 2 investigations you want to order?

ECG, cardiac enzymes.

Q2: what's the most likely Dx (ST depression in anterior leads, -ve cardiac enzymes)?

Unstable angina.

Q3: what's your management?

Admission , Give O2 if hypoxic , and morphine

Give Aspirin , clopidogrel , B blockers , LMWH, replacement of deficient electrolytes .

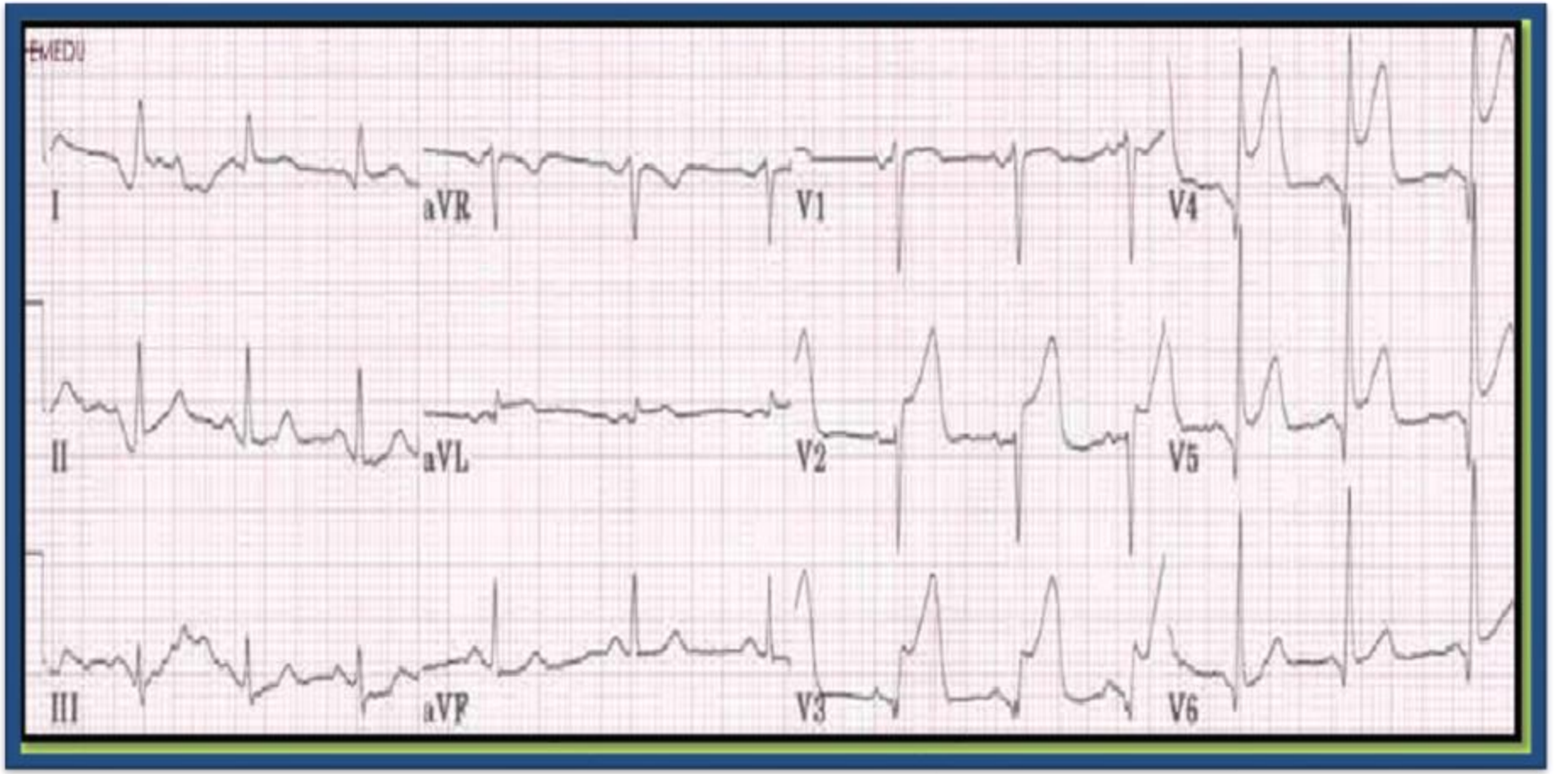
DO CATHETERIZATION

Q4: whats your management if cath. Showed 4 vessels occluded?

CABG .

Q: This ECG is for a 48 YO pt, presented with chest heaviness, diaphoresis & nausea for 2 hrs. What is your Dx?

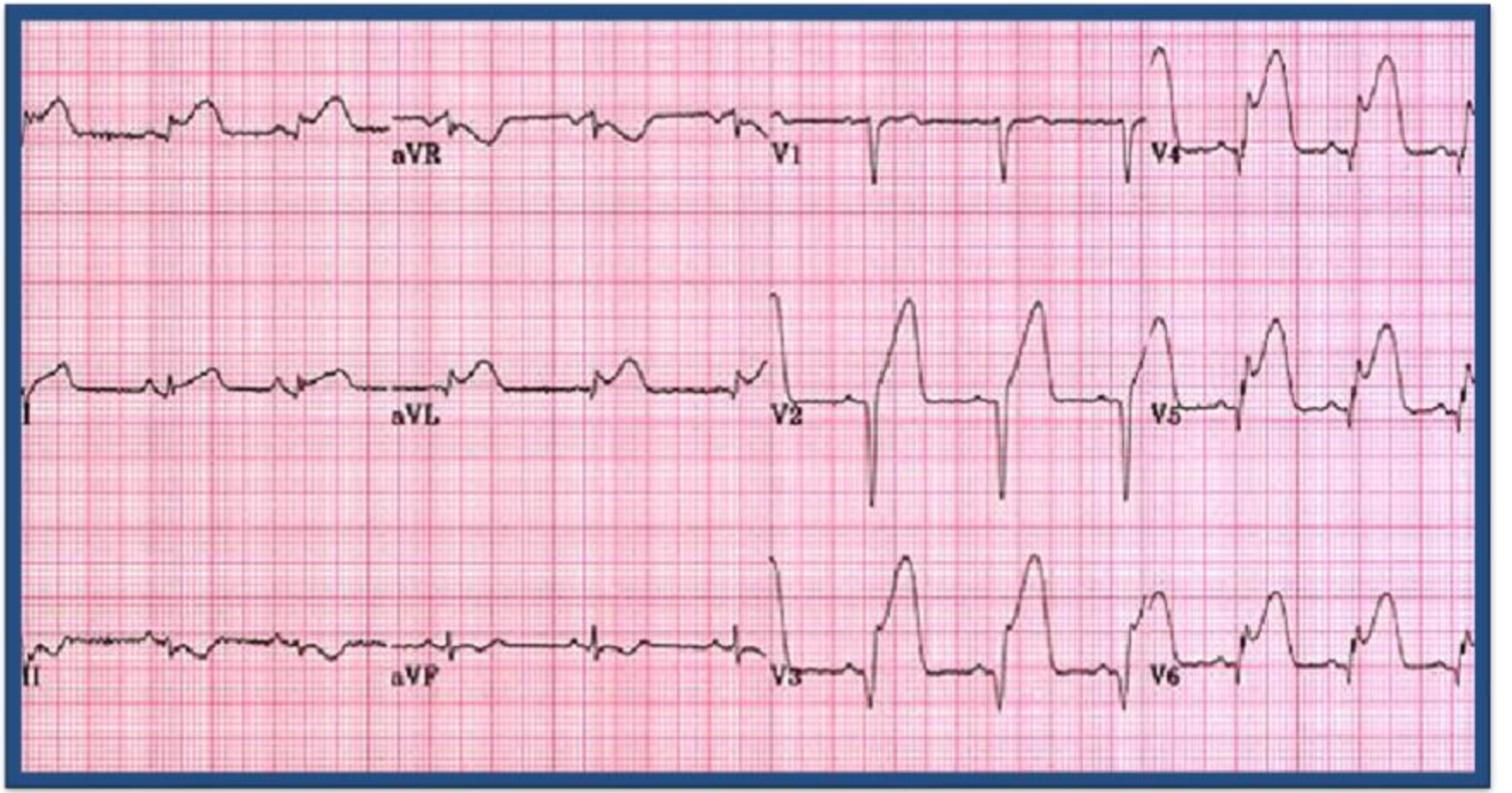
Acute Anterior wall (anteroseptal) ST elevation MI.



Q:This 40 year-old patient presented with chest pain, what's your diagnosis?

Acute Anterolateral ST elevation MI

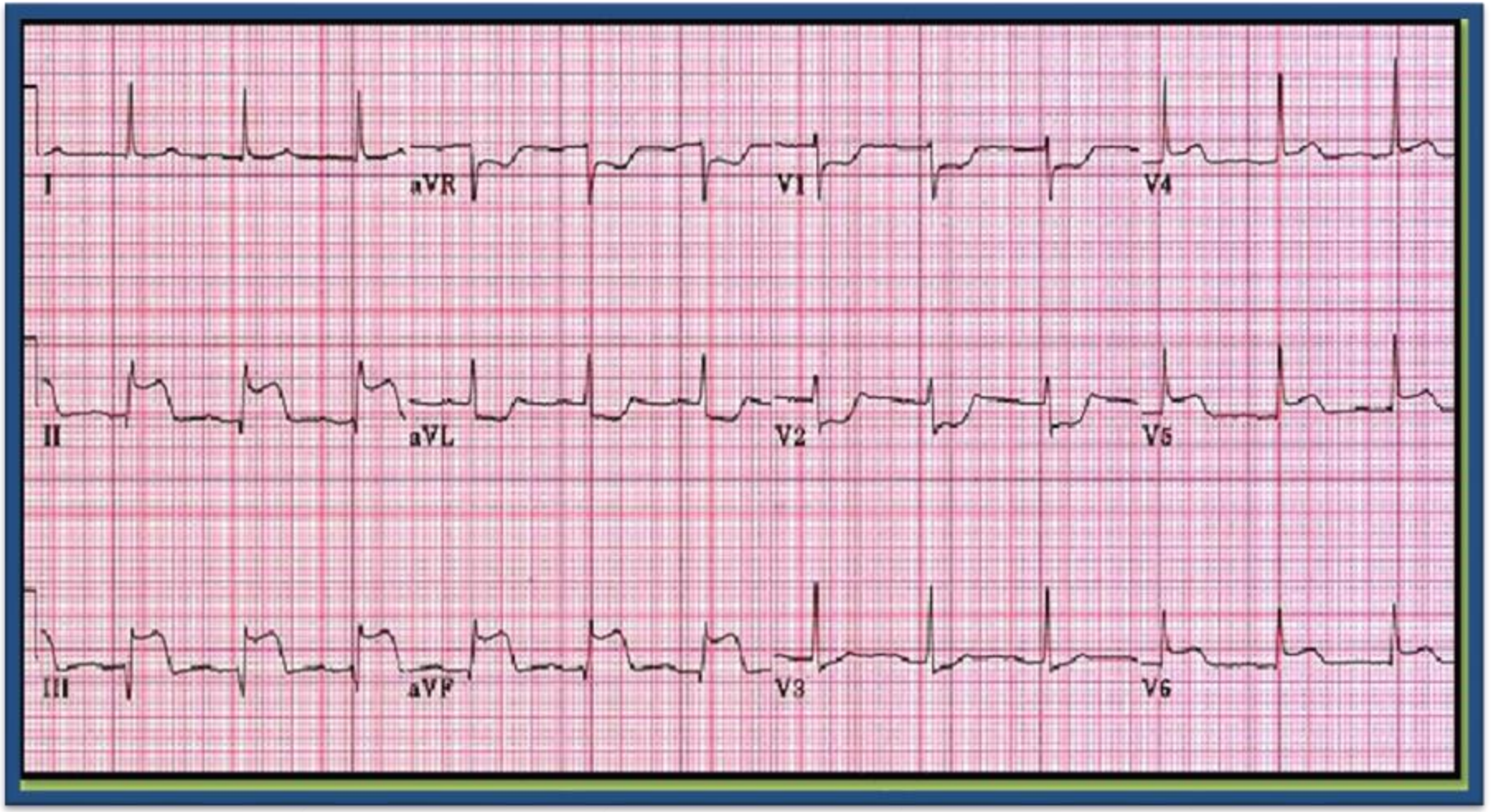
Anterior chest leads : V1-V4 … Lateral chest leads : I , AVL,V5, V6



Q: Patient presented with chest pain. what is your diagnosis ?

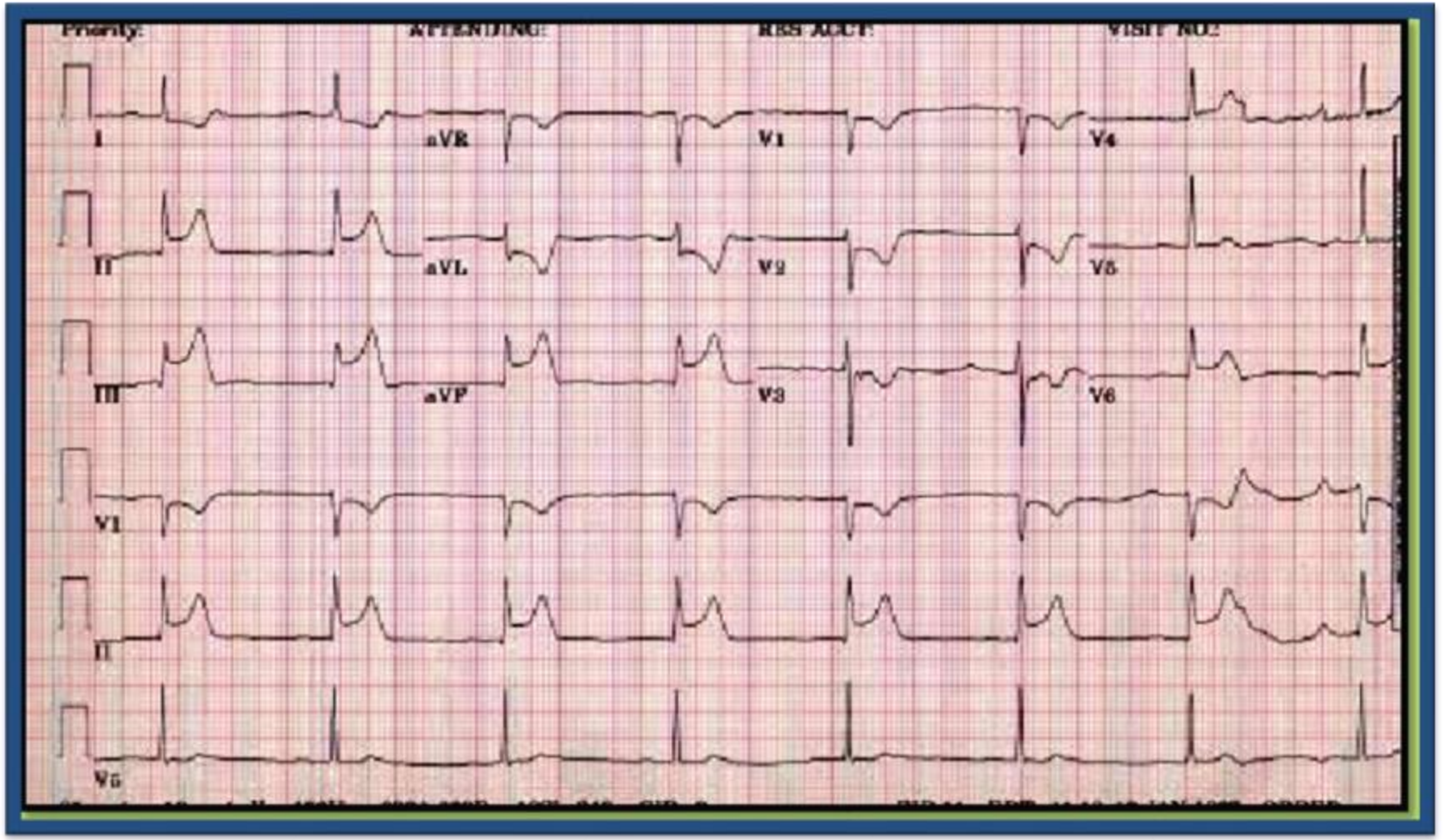
Acute inferior wall ST-elevation MI. (leads : II, III, aVF)

Note : in inferior MI the pt is hypotensive ! DO NOT give diuretics or nitrates as it may cause cardiovascular collapse



Q: 70 YO male came with palpations & chest pain. Mention 2 abnormalities in this ECG.

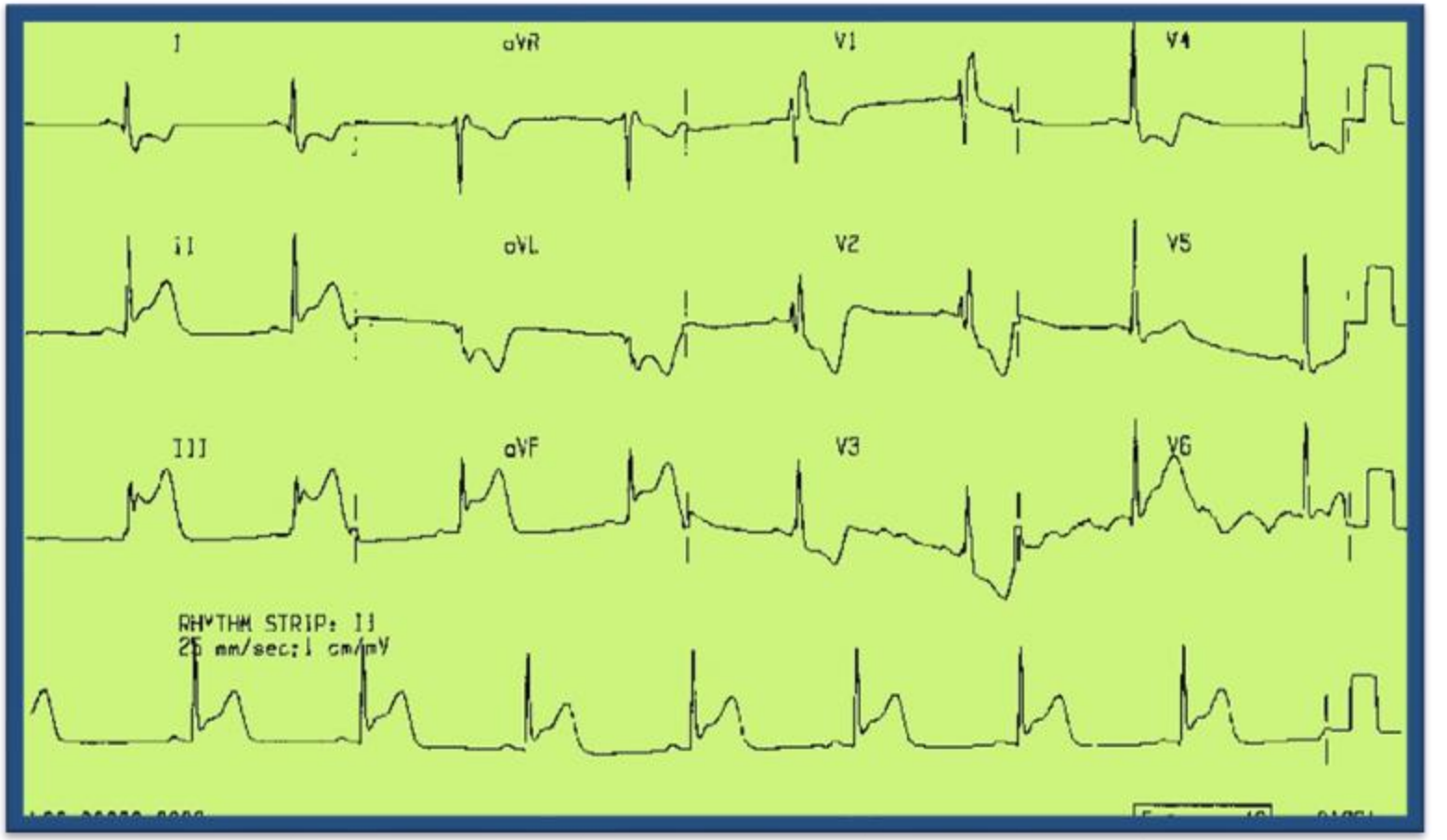
1. ST elevation leads II, III, avF.
2. ST depression in aVL and V1 (reciprocal changes).



Q:60 YO male pt, presented with acute chest pain for 30 minutes, what is the Dx?

What is your management for this pt?

Acute inferior wall myocardial infarction/Oxygen, aspirin, IV morphine , b blocker, ACE inhibitors , LMWH , streptokinase.



Q: A 79 year old female, presented with epigastric pain, N&V and sweating.

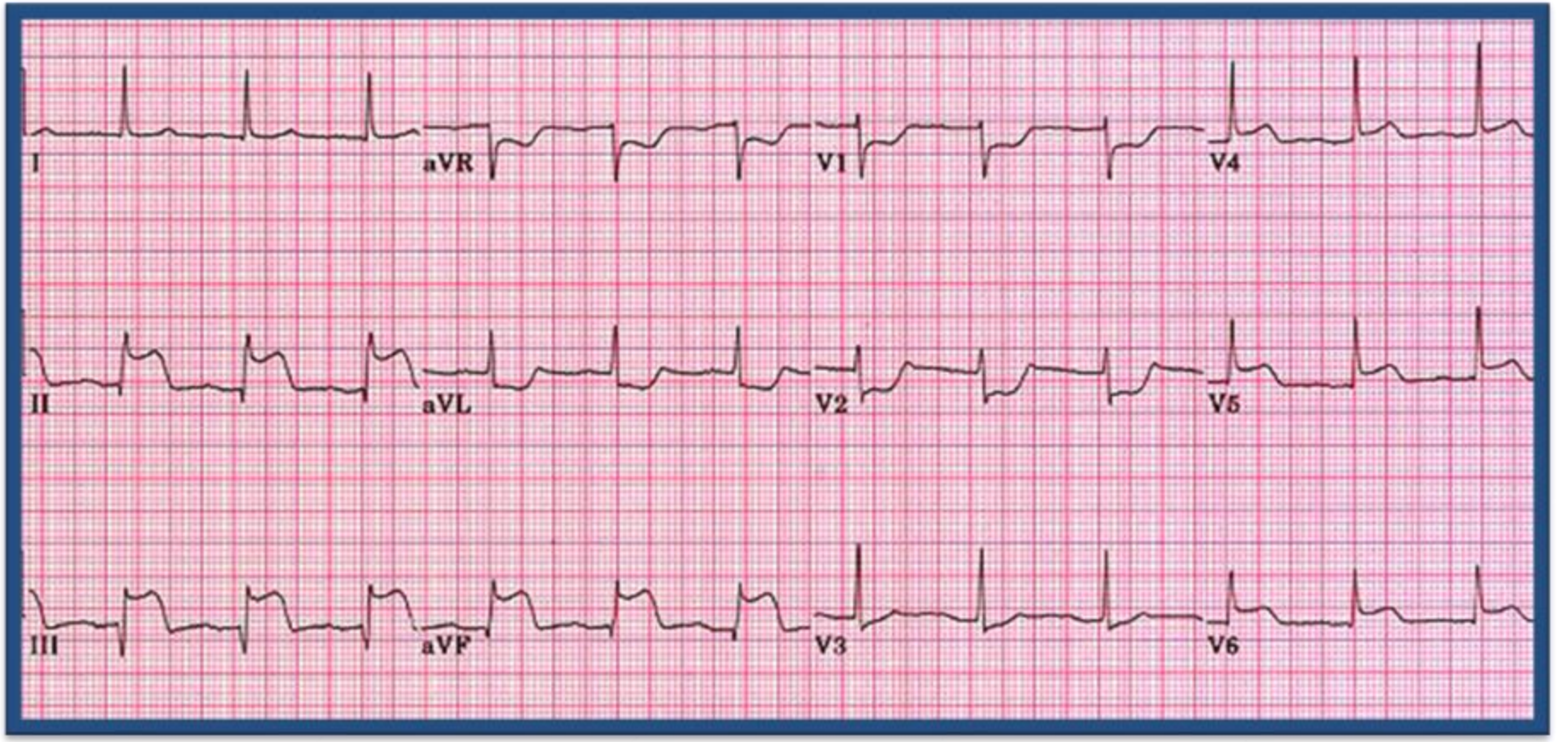
a) Mention 2 gross findings you see on the ECG.

-ST elevation in leads II, III, AVF

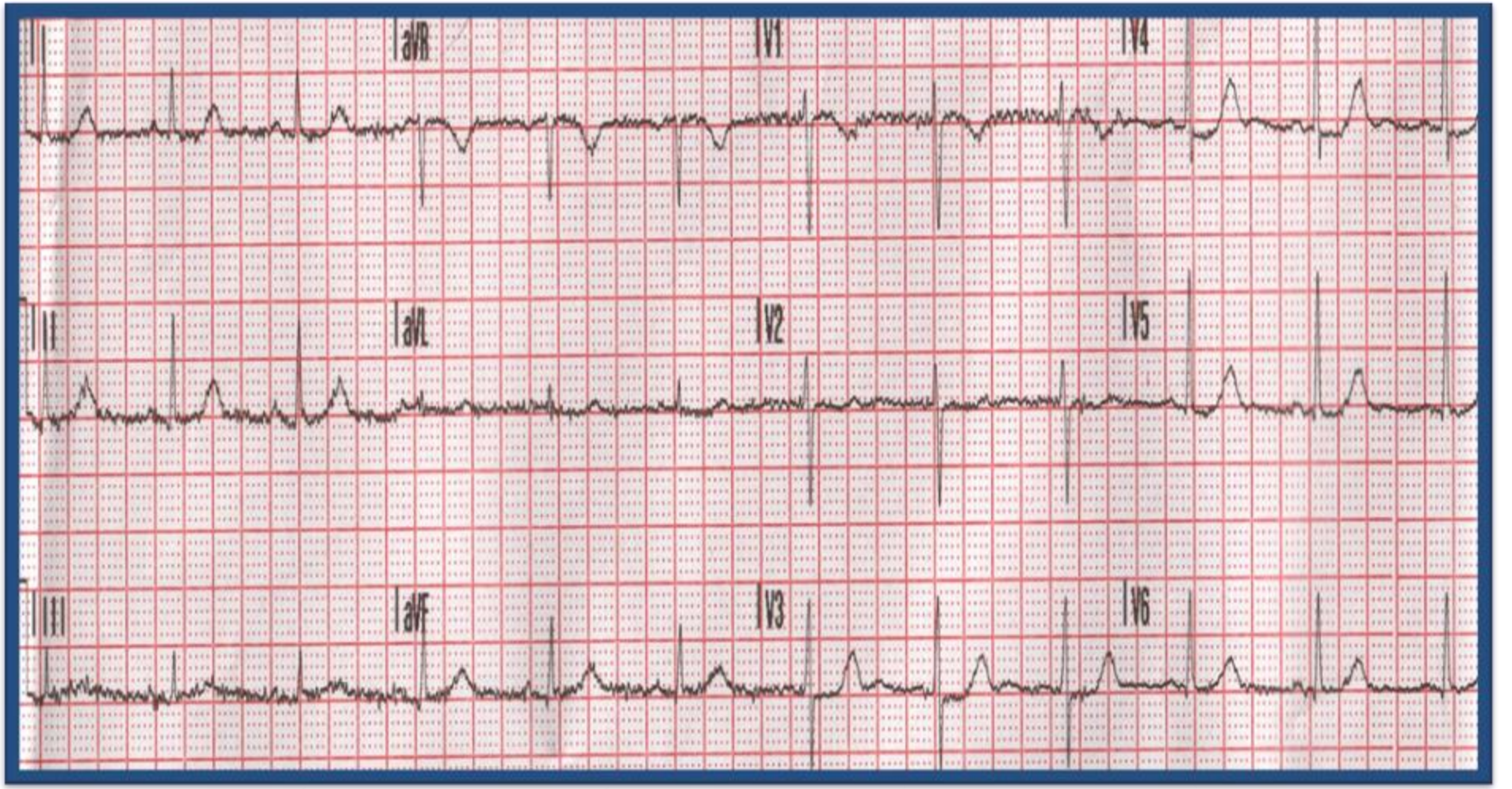
-ST depression and T inversion in leads aVL,V1,V2

b) What is the diagnosis?

Acute inferior STEMI



Q: A 48 year old male , who developed rapidly worsening chest pain on minimal exercise or even at rest,for less than 15 minitues , no vomiting was associated with , the following ECG changes :



1) Identify the abnormality in the previous ECG .

Inverted T-wave with no ST- Elevation

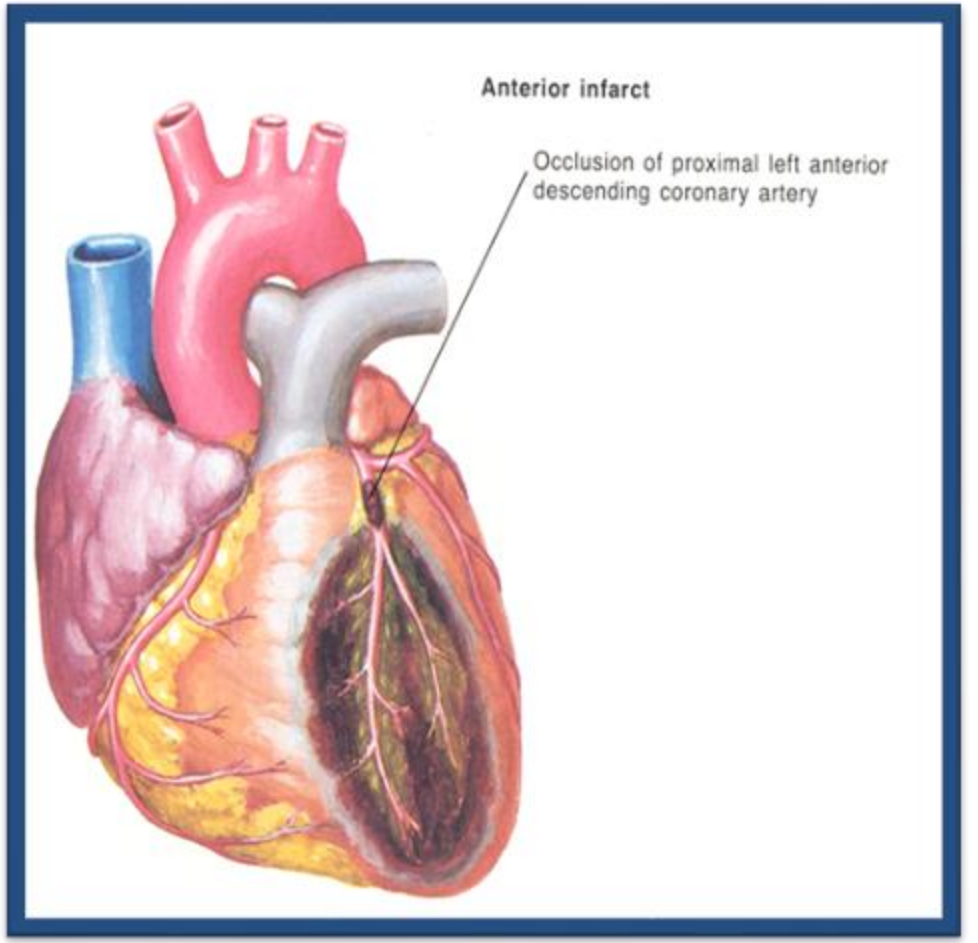
2) According to the History (rapidly worsening chest pain on minimal exercise or even at rest, for less than 15 minutes , no vomiting was associated , with Non ST-elevation & T-wave inversion ECG changes) , Give the spot diagnosis .

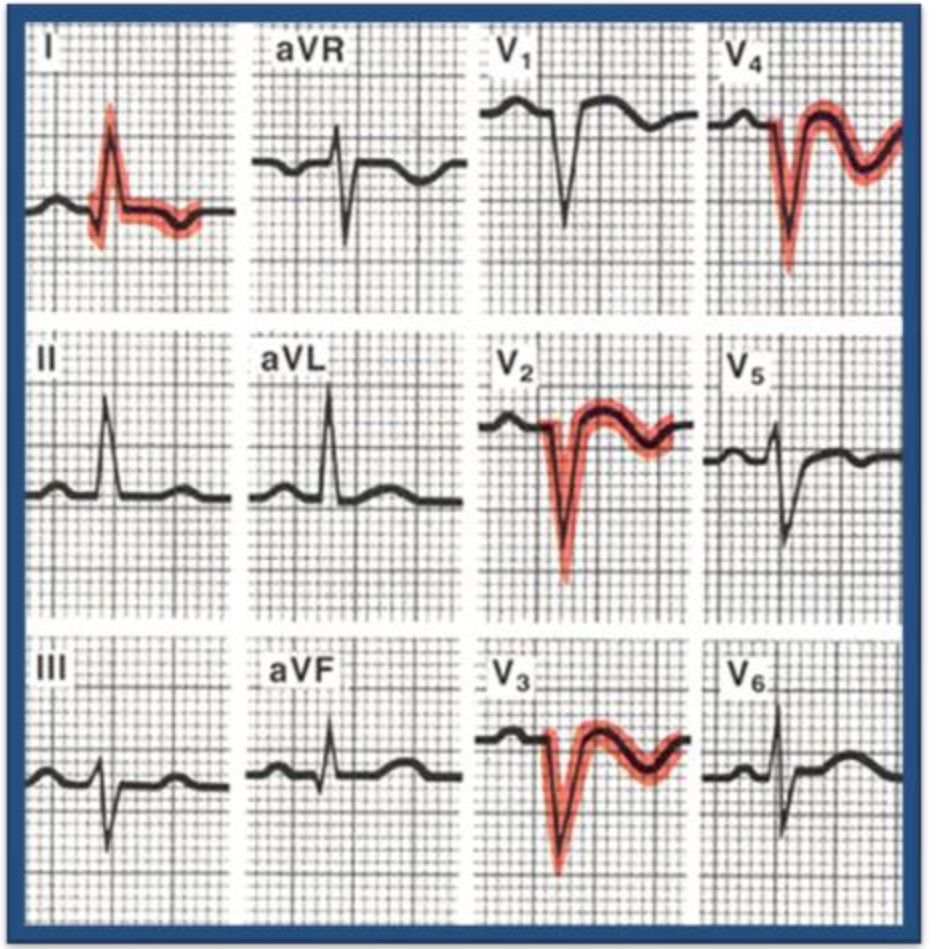
Unstable angina but we must do cardiac enzymes to R/O NSTEMI

Q: In the following ECGs , you have to Identify the abnormality & the occluded artery (location of infarction) :

1. Identify The Abnormality :

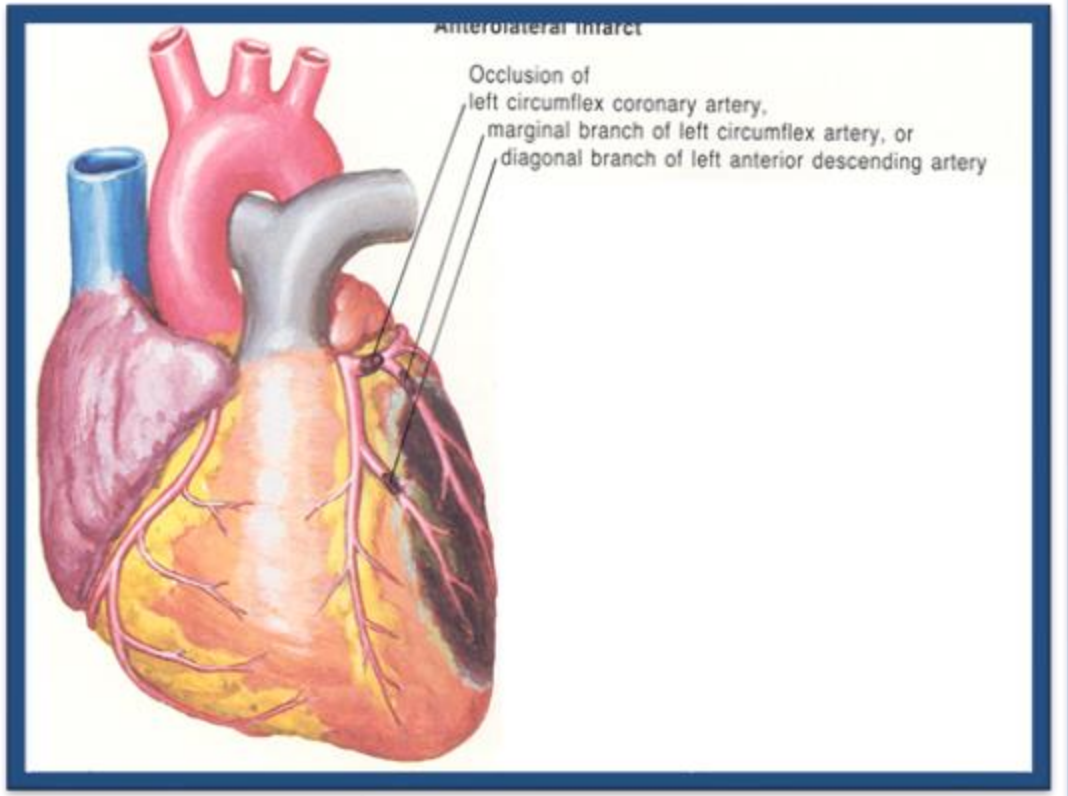
Significant Q waves , with T waves inversions in lead 1 , V2 , V3 , V4 , ST-elevation

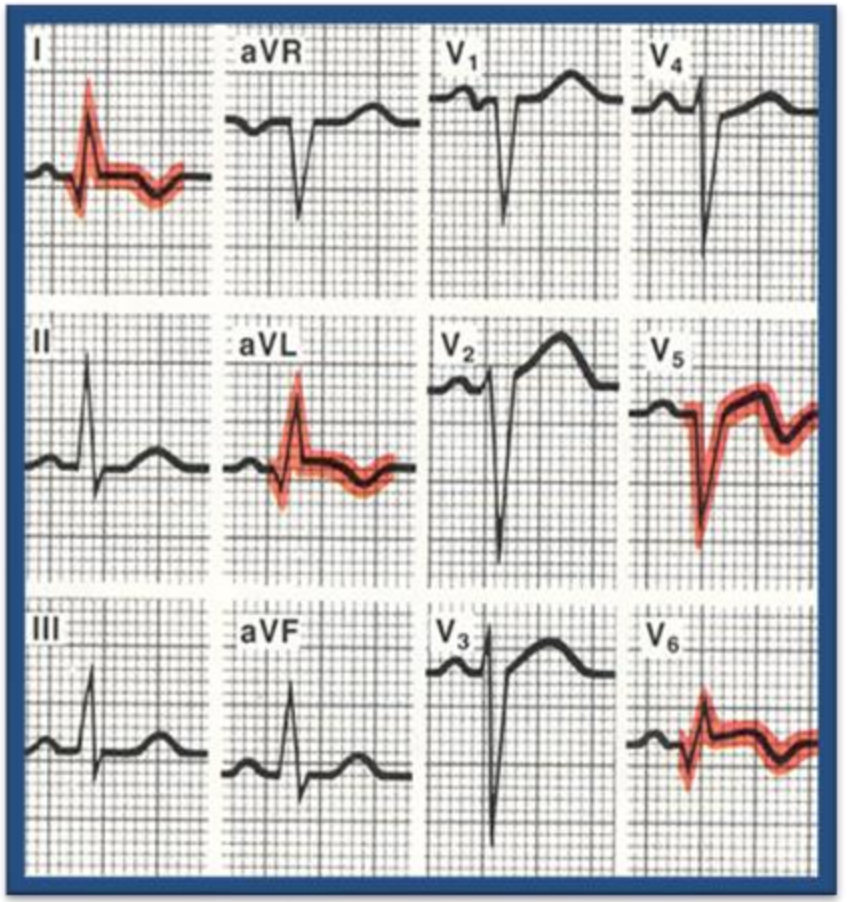




2. Identify The Abnormality :

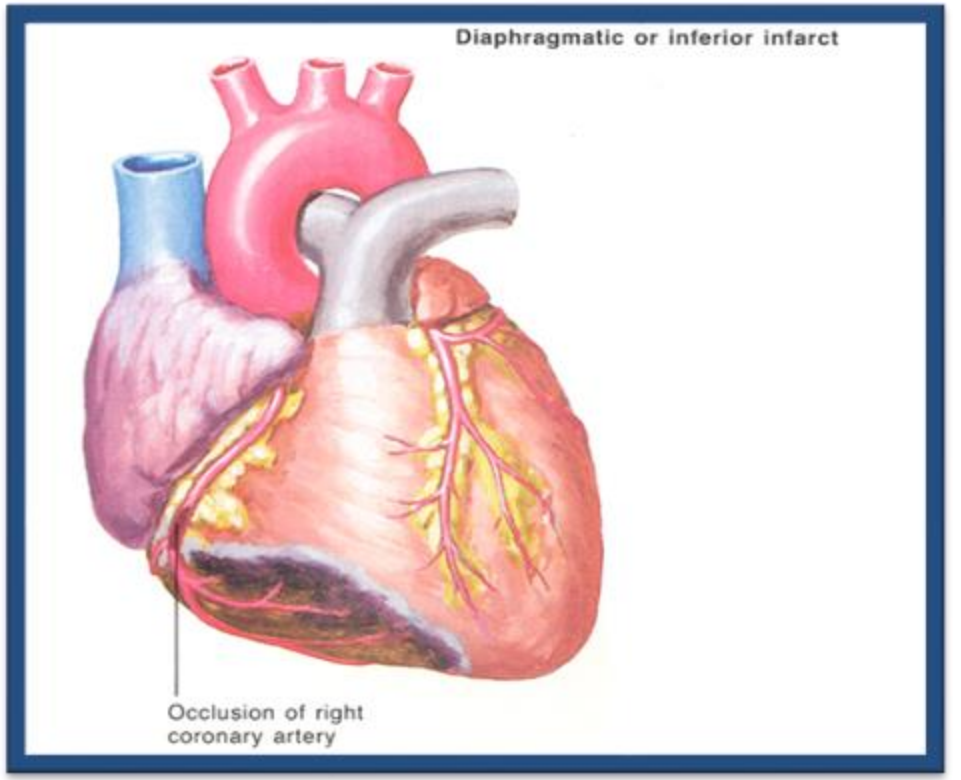
Significant Q waves , with T waves inversions in leads 1 ,aVL, V5 V 6
ST-elevation

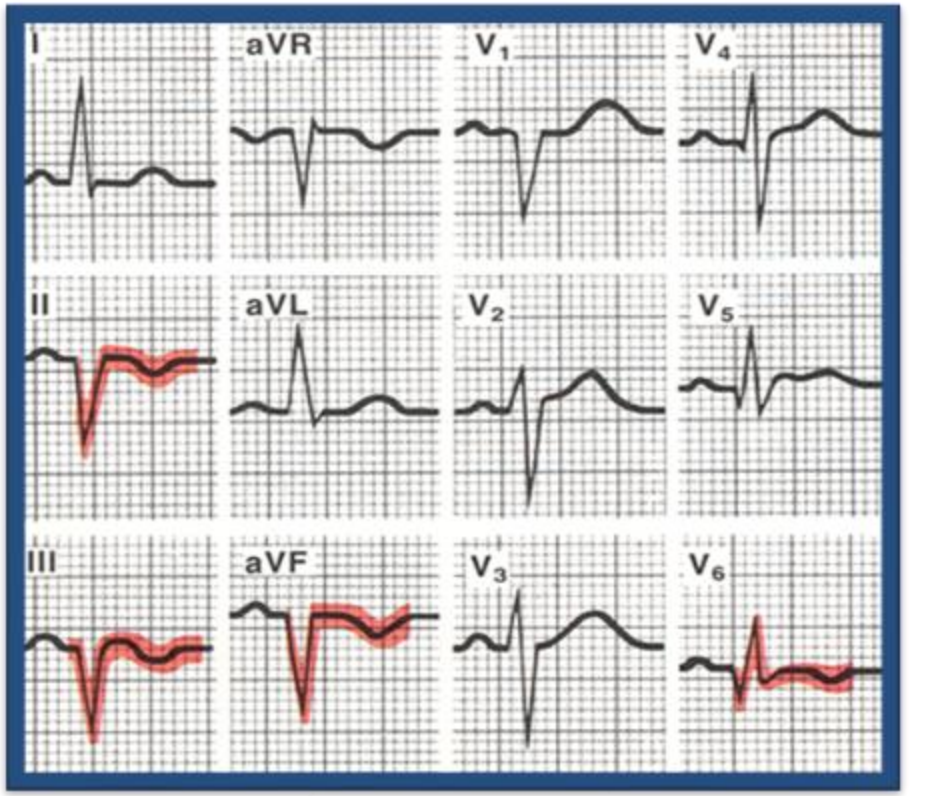




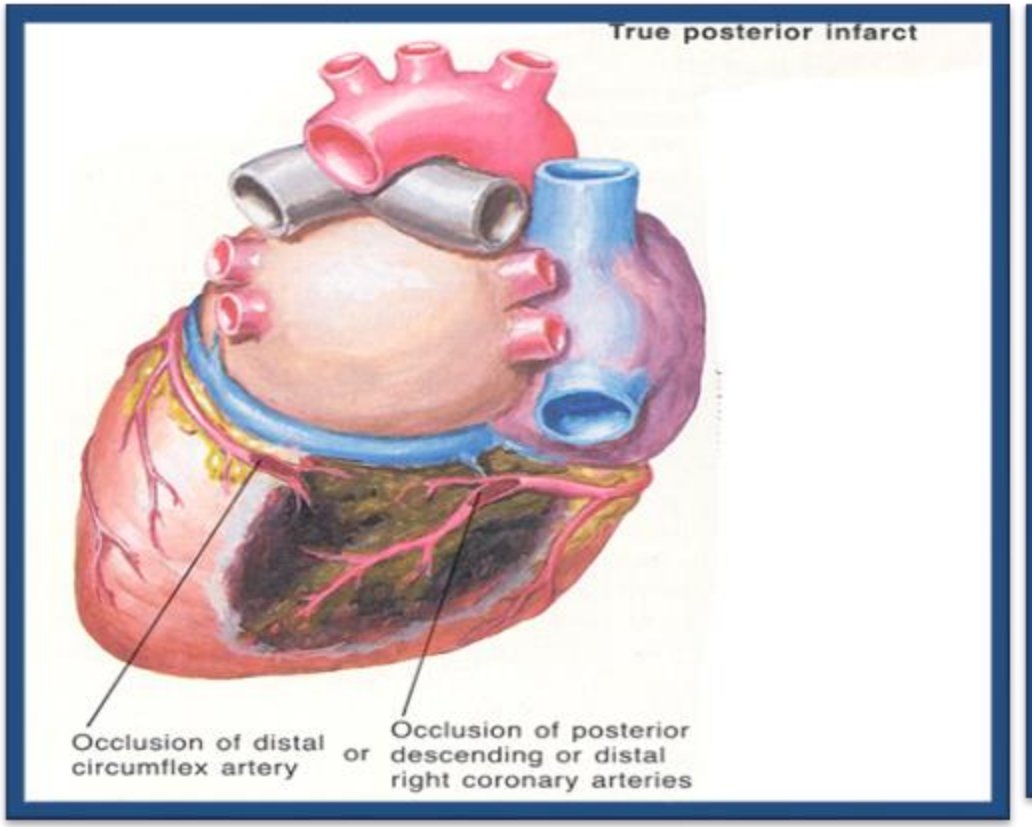
3. Identify The Abnormality :

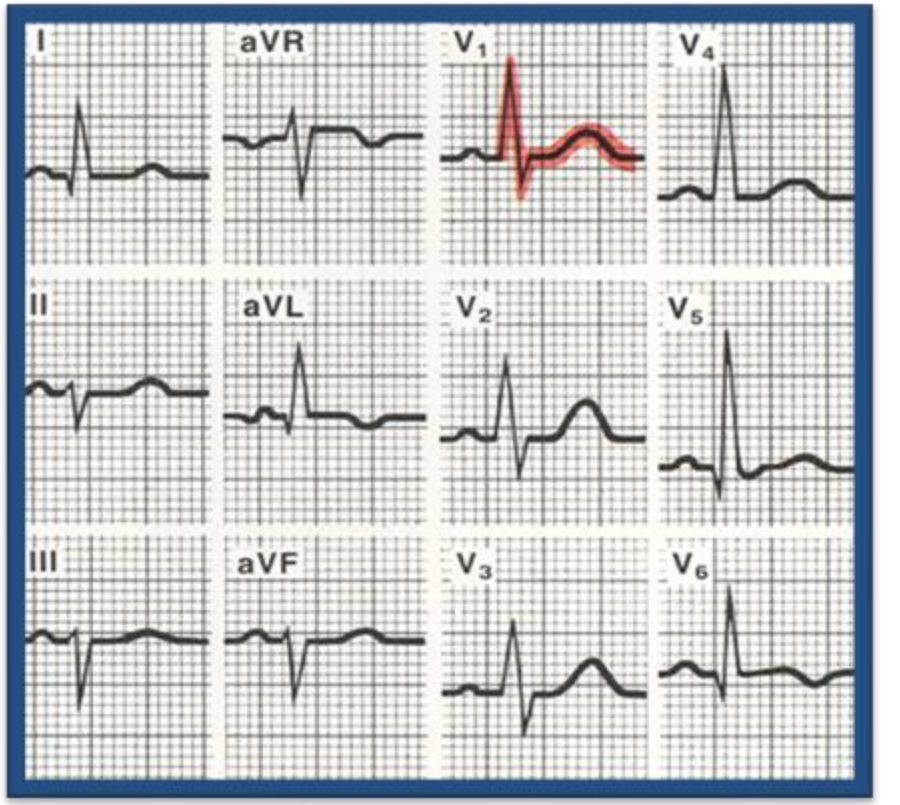
Significant Q waves , with T waves inversions in leads II , III & aVF . With Lateral damage changes in V5,V6 .
With ST elevation





4. Since no ECG lead reflects posterior electrical forces, changes are reciprocal of those in anterior leads. Lead V_1 shows unusually large R wave (reciprocal of posterior Q wave) and upright T wave (reciprocal of posterior T wave inversion)



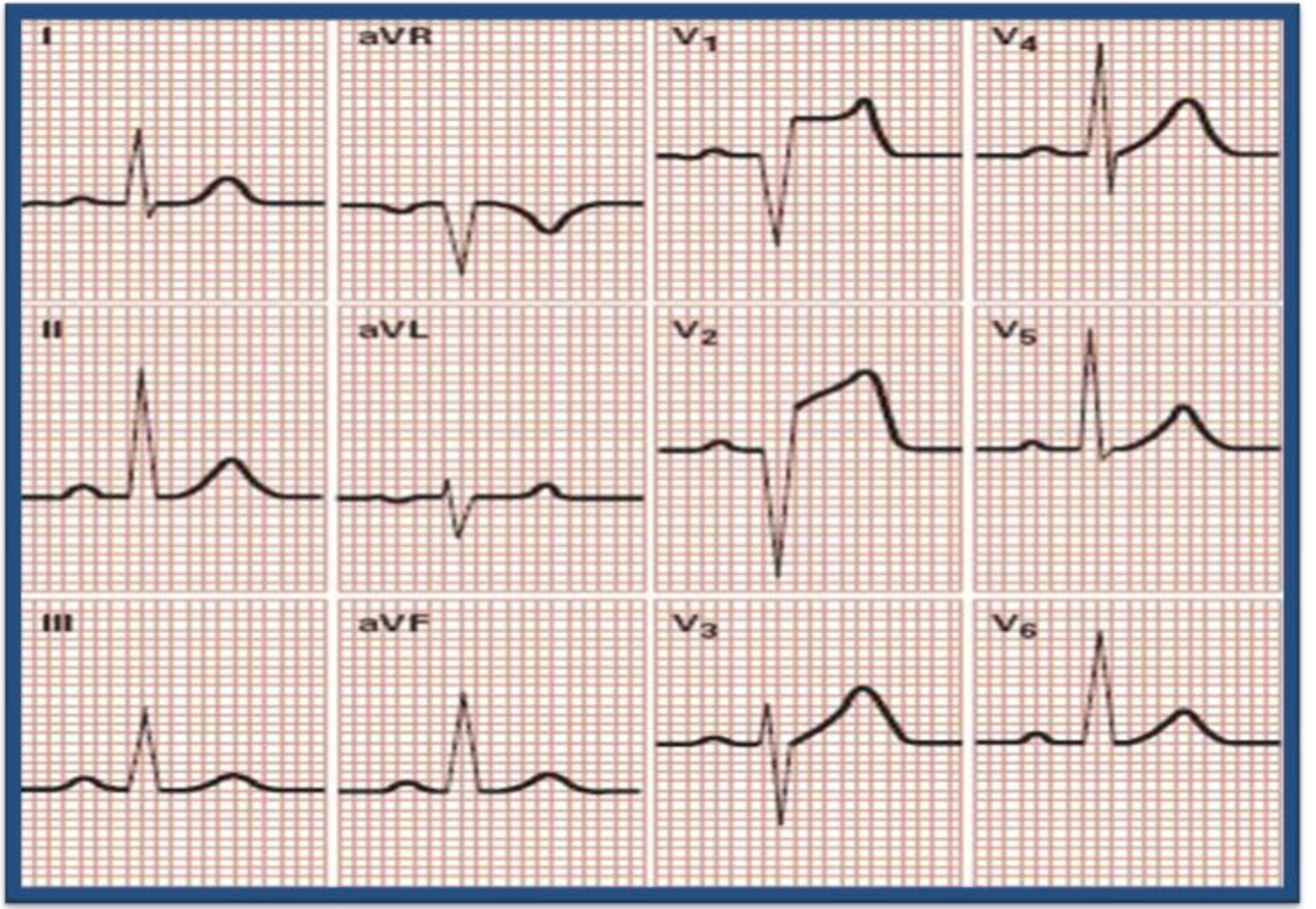


5. Identify The Abnormality :

STEMI in leads V1-V4

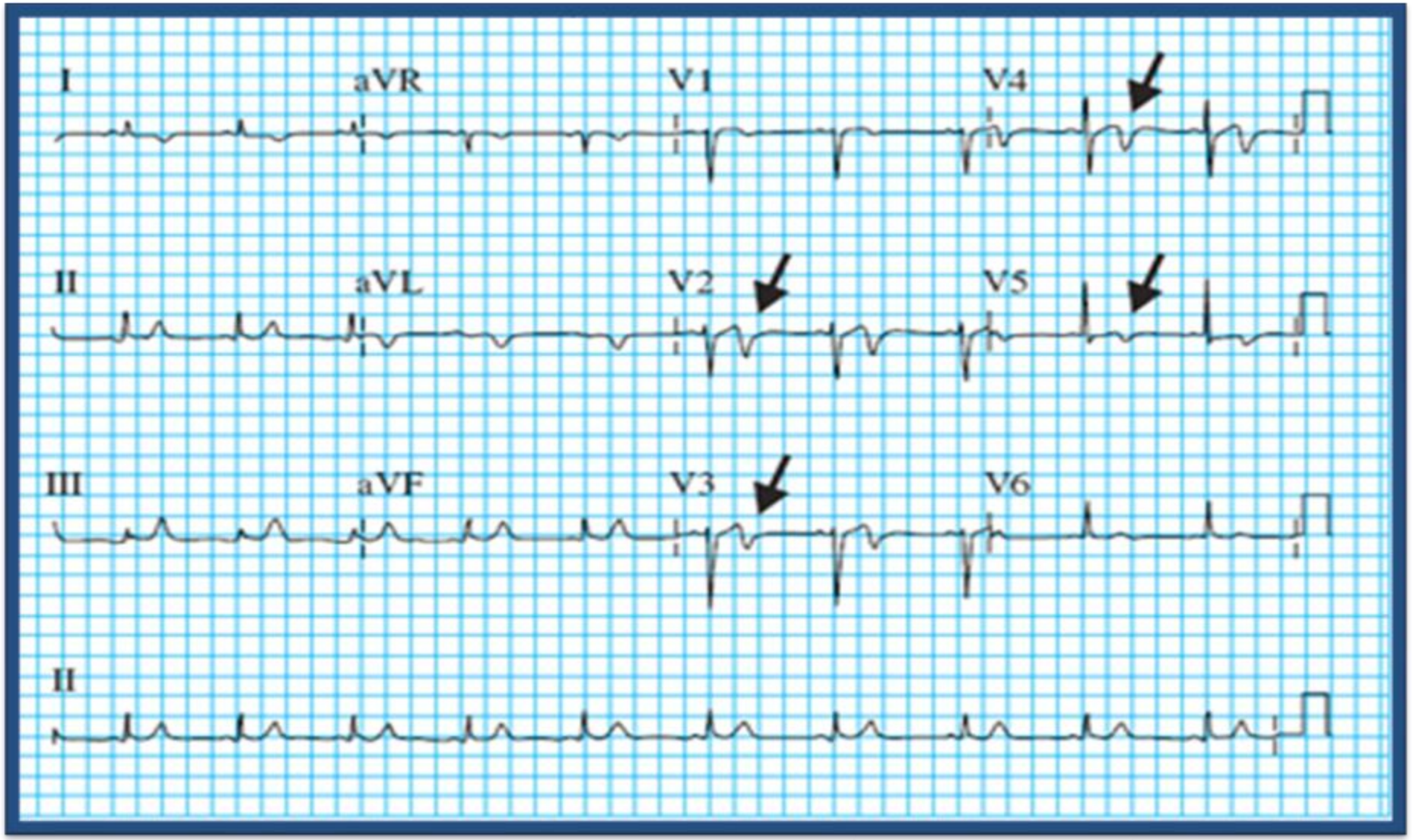
The occluded artery is :

left coronary artery – left coronary descending



Q: Identify the abnormality in the ECG .

ST- Elevation & Inverted T-wave



Q: According to the History (rapidly worsening chest pain on minimal exercise or even at rest, for **less than 15 minutes** , no vomiting was associated , with non ST-elevation & T-wave inversion ECG changes and negative cardiac enzymes).

1) Give the spot diagnosis .

Unstable Angina (Since No elevated enzymes, so it's **not MI**)

2) Give Differential Diagnosis:

1.For ST-Elevation :

1- Acute MI
2- prinzmetal Angina
3- Pericarditis

2. For inverted T-waves :

1- Ischemia (angina)
2- Ventricular hypertrophy
3- Bundle branch block
4- Digoxin Treatment

3) Investigations:

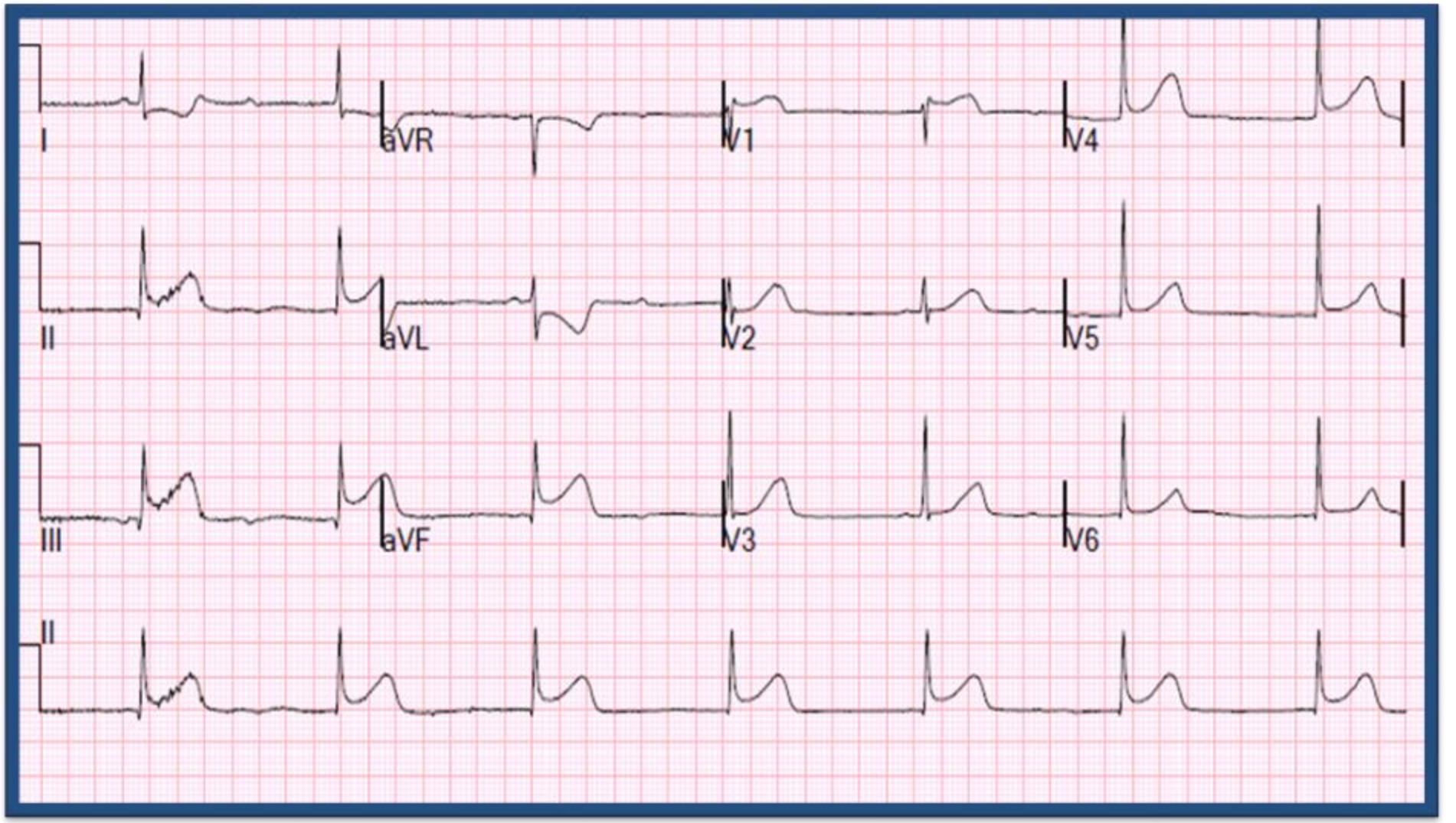
-ECG: no prominent Q-waves
-Cardiac enzymes are normal
-Coronary angiography

4) Treatment:

: The same for stable angina with two additional points
1-**Admission** to the hospital: because there's a significant risk of death or myocardial infarction during the acute phase.
2-Anticoagulants: unfractionated or LMW **heparin** should be given

Q: Mention two major ECG Findings?

a. ST elevation in the inferior leads (II , III , aVF)
b. AV block

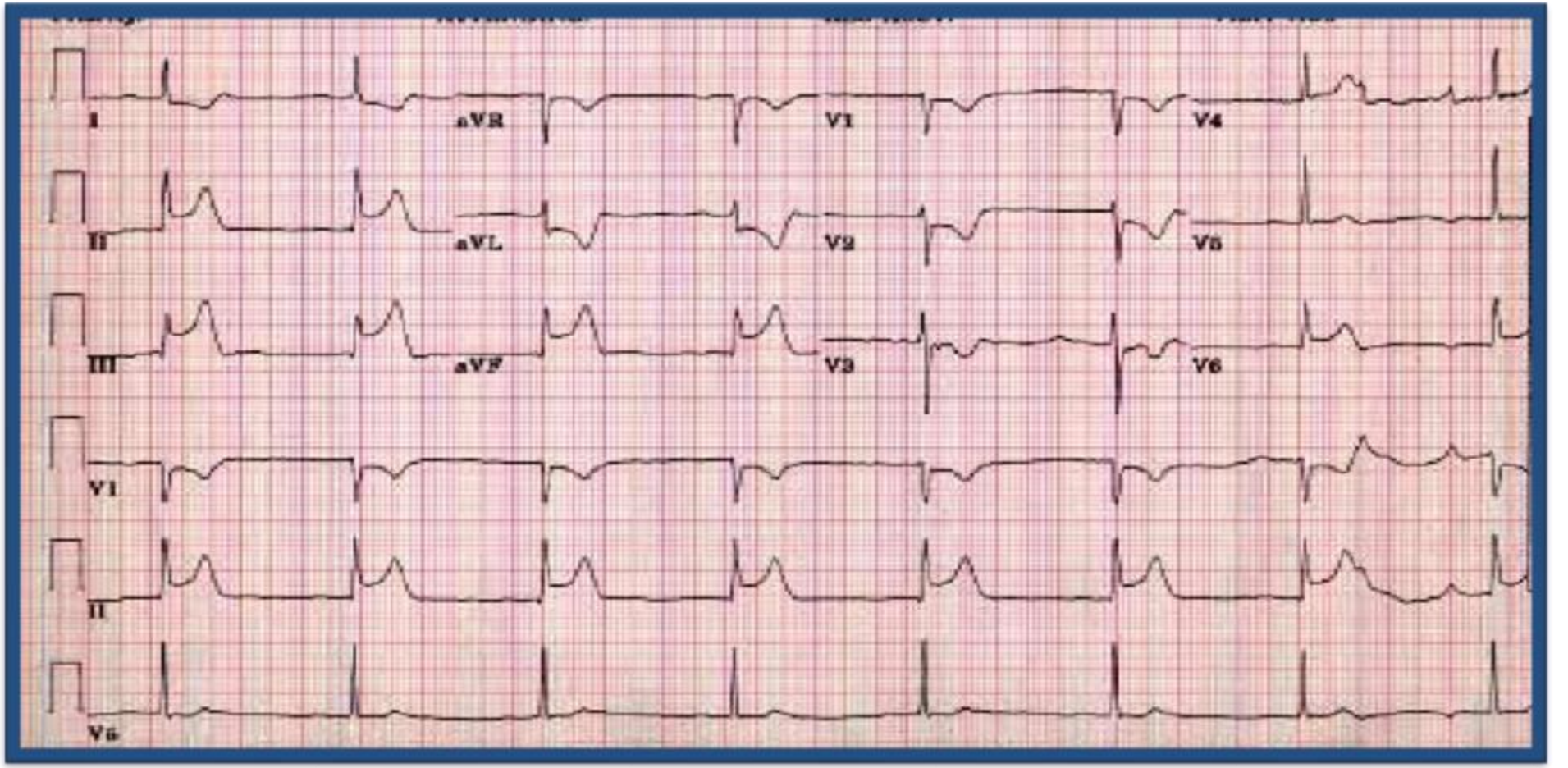


Q: Mention 2 findings.

1. ST elevation on leads II, III, & aVF.
2. ST depression in leads I, AVL , V1, V2 (reciprocal changes)

• Your Diagnosis.

Acute Inferior wall STEMI



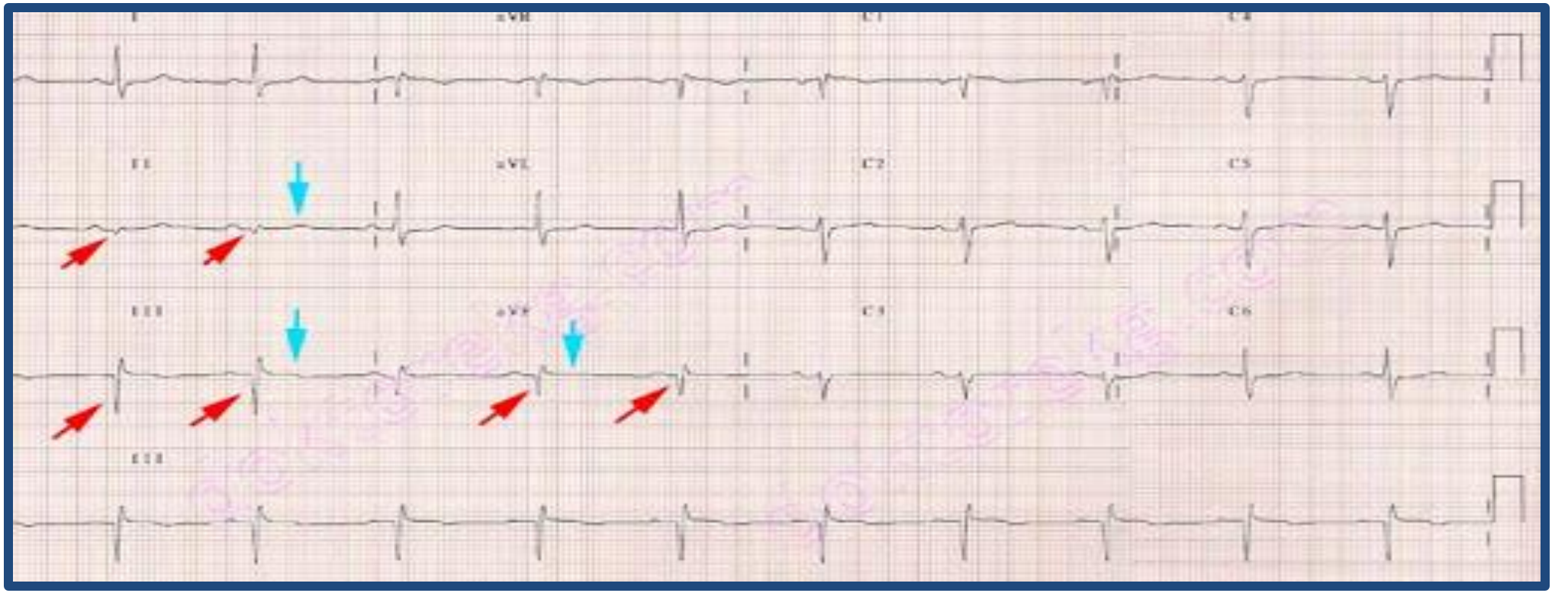
Q: 70 years old man who had underwent coronary bypass graft operation after inferior wall
myocardial infarction. The ECG was recorded when the patient was asymptomatic.

•Mention pathological findings on the ECG .

Q waves in leads II, III and aVF

•What is your diagnosis?

Old inferior MI



complications of acute MI

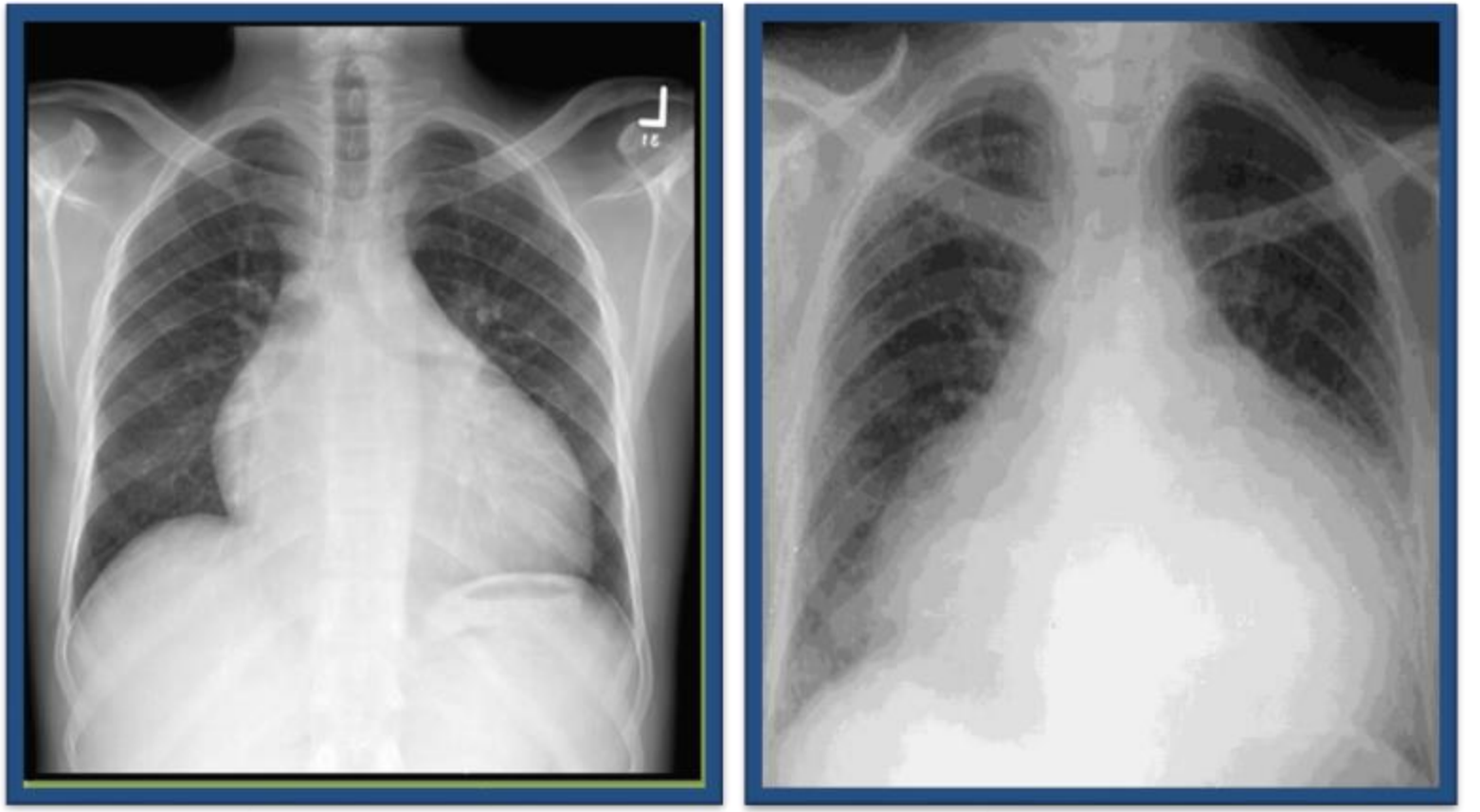
- CHF
- Arrhythmias
- Recurrent infarction (diagnosed by elevation of CK , CK-MB)
- Mechanical complications : free wall rupture, interventricular septum rupture , papillary muscle rupture , ventricular pseudoaneurysm , ventricular aneurysm
- Acute Pericarditis
- Dressler syndrome

Pericardial and valvular dx

Q1: This patient had SOB & chest pain for 2 weeks, and a normal blood pressure.

What's your diagnosis?

Pericardial effusion(enlarged cardiac silhoutte)



-What is the ECG abnormality :

Electrical alternans

-What is the next investigation you request ?

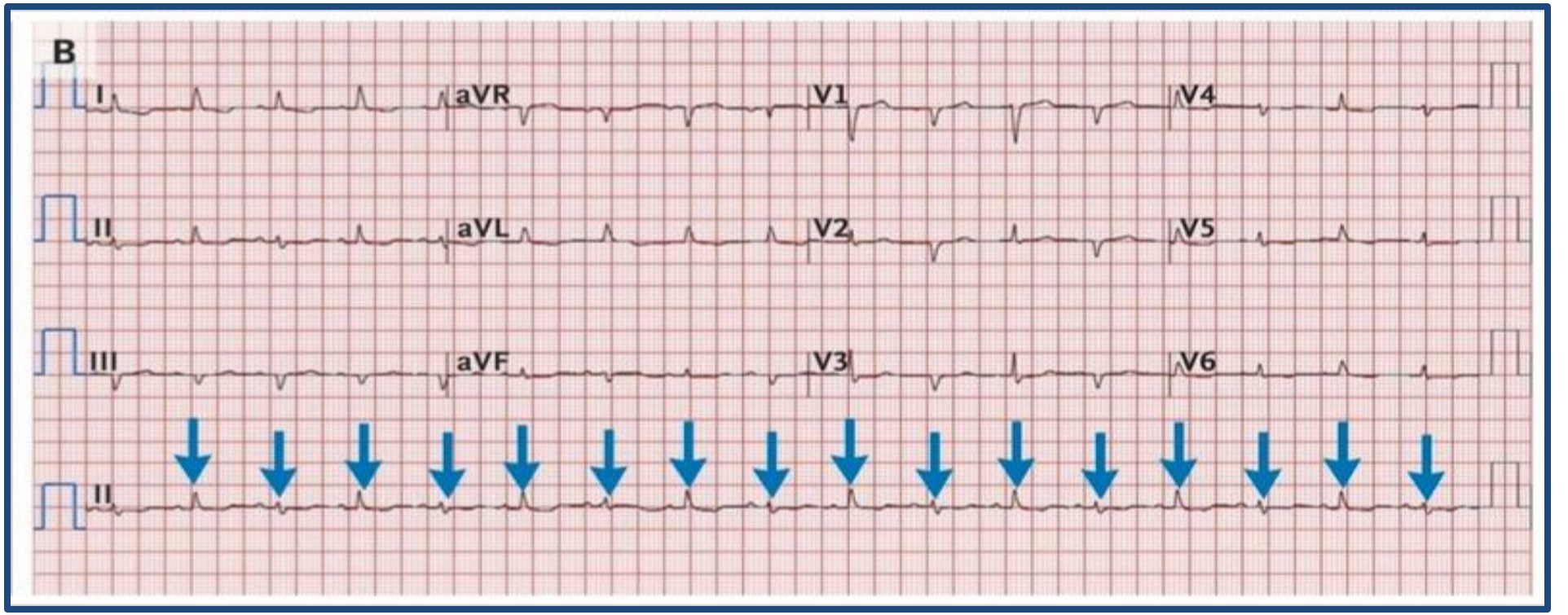
Echocardiogram

-What is the most accurate inves. ?

CT and MRI (especially in localized pockets of effusion)

-What is your diagnosis ?

Pericardial effusion



Managment

Pericardial drainge is preferable in :

-Traumatic hemopericardium

-post surgical effusion

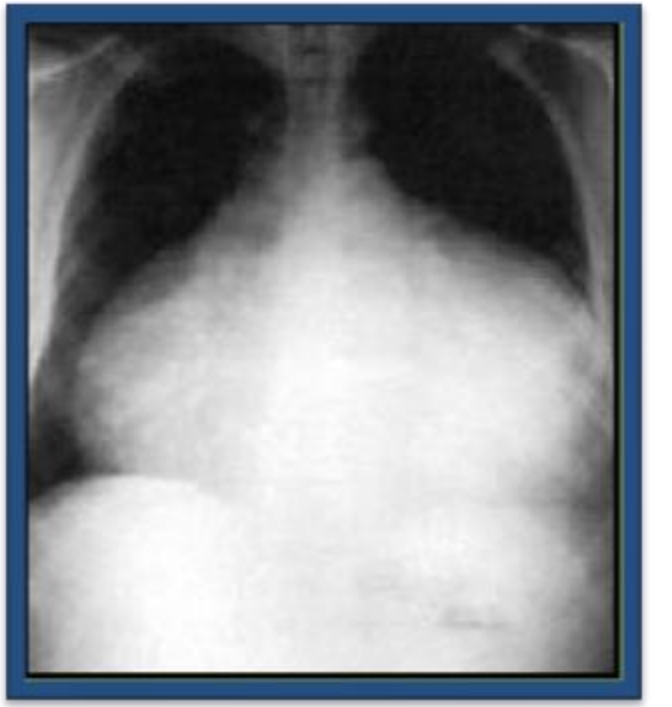
-susp. of Bacterial or TB

Pericardiocentes is used to treat :

- Viral
- Idiopathic
- Neoplastic
- Hypothyroid
- Renal failure related tamponade

- Tamponade occurs when pericardial effusion leads to critical cardiovascular compromise

Q2:This CXR is for a pt who is a known case of chronic renal failure, presented with sudden chest pain, SOB, BP 85/60 , pulsus paradoxus, dilated neck veins and soft,distant heart sound.



What's your Dx.?

Cardiac Tamponade

What is your immediate management?

Precordiocentesis.

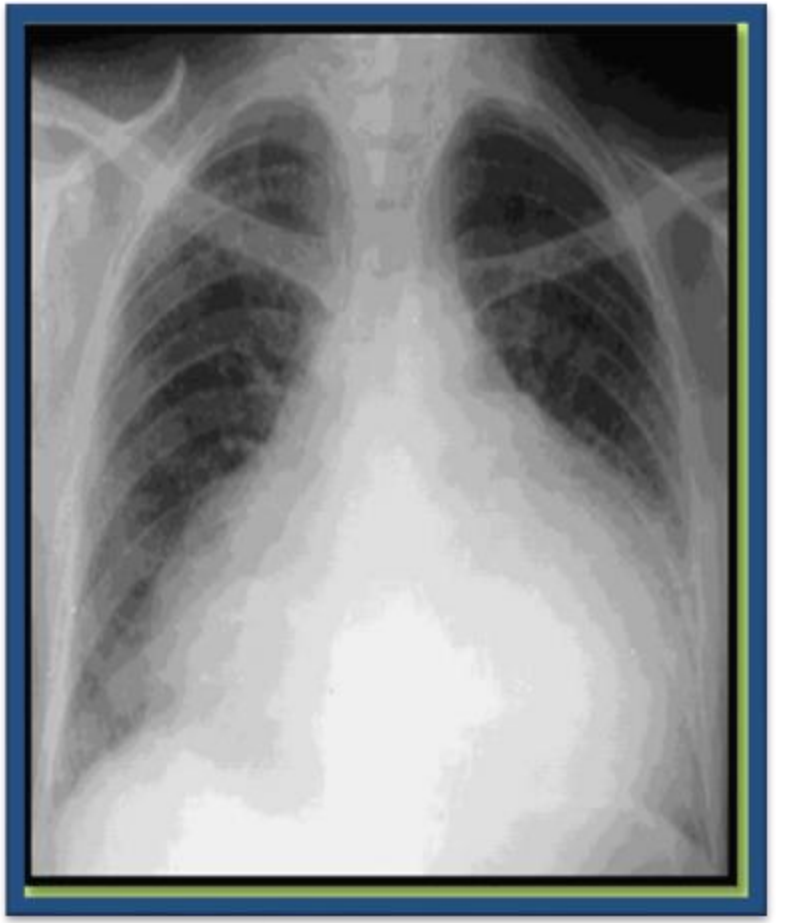
Case

Q: 50 YO male pt presented to ER 1 hour ago complaining of chest pain, diagnosed as having acute anterior wall MI, while he's in the ER he suddenly collapse,

BP=30/0, with raised JVP.

What's your Dx.?

Cardiac Tamponade



In case of Rupture of free wall of the heart (Post MI or trauma) the tamponade develops quickly ,otherwise it develops slowly)

Q: Male patient had a sore throat 3 weeks ago, he has sever retrosternal chest pain that is reduced by leaning forward, referred to the neck and left shoulder with mild fever and tachycardia

On neck examination :

- Brisk collapse of jugular vein during diastole (prominent x and y descent)
- Kussmaul sign (a lack of normal decrease in JVD during inspiration)

On CVS auscltation : Diastolic knock heard

1) What is the diagnosis ?

Acute pericarditis

2) What is the sign you should found when you do physical exam to the pt ?

Pericardial friction rub(not always present)

3) Mention 3 investigations .

a.Echocardiogram

b.ECG

c.Chest Xray(lateral CXR shows calcification over the Rt ventricle > pathognomic for constrictive pericarditis)

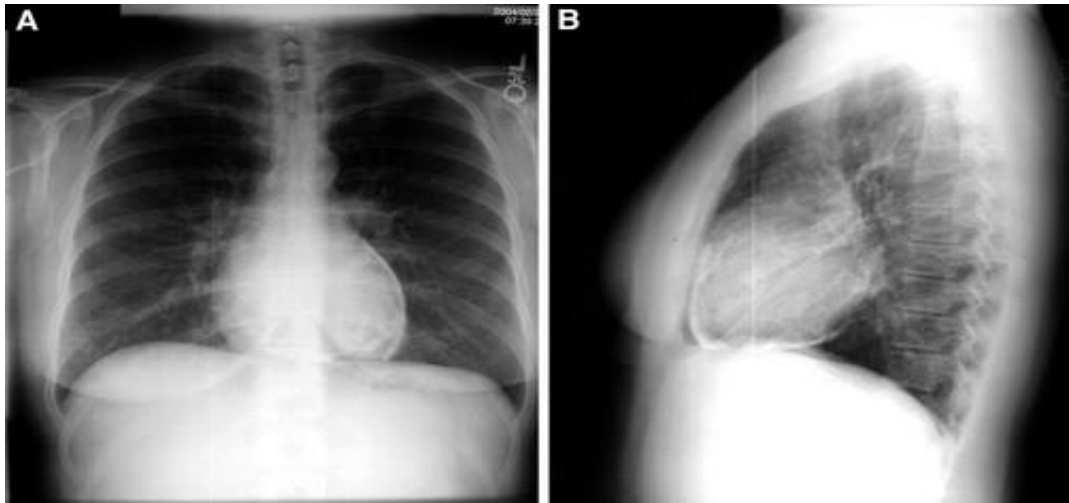
d.CT /MRI (thickened pericardium >5mm)

4) DDx of constrictive pericarditis ?

Restrictive cardiomyopathies (can be differentiated by BNP >increased in case of restr.)

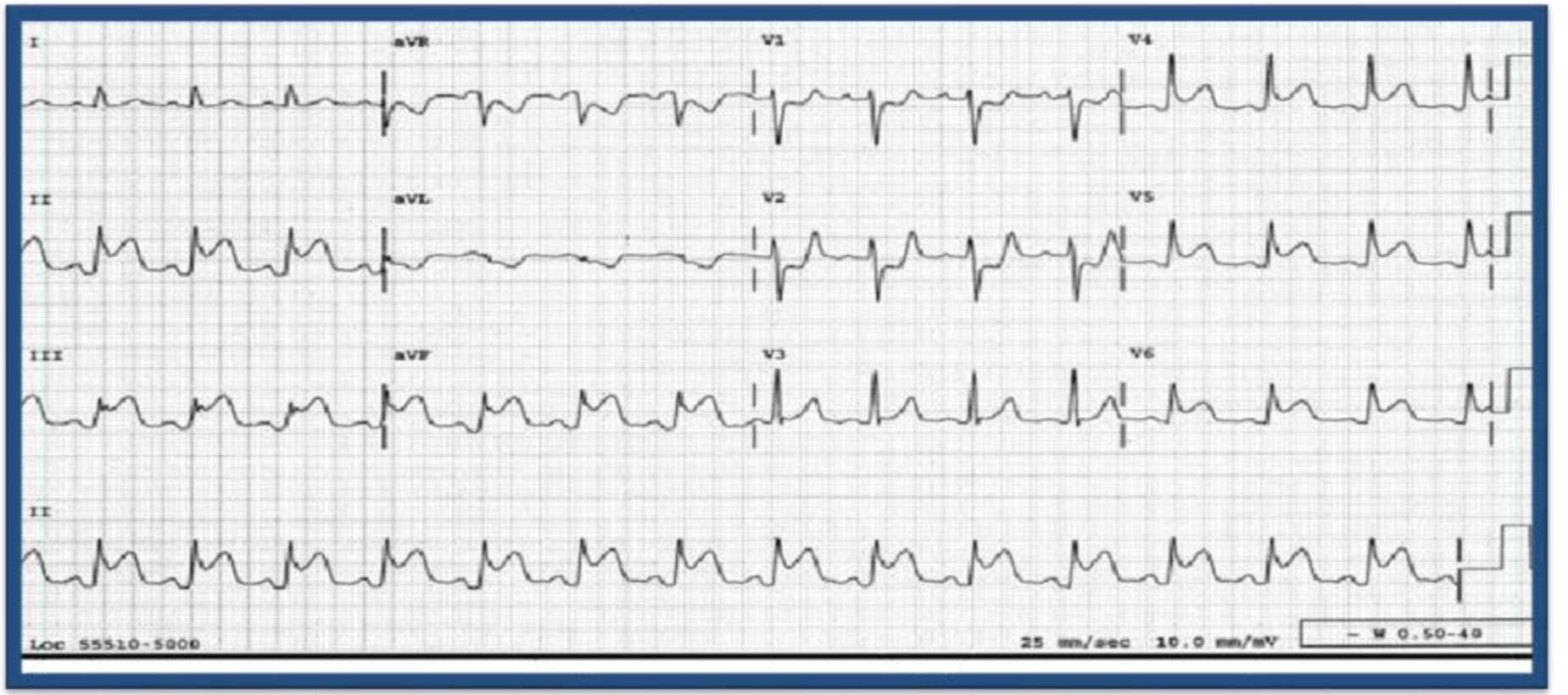
5)Treatment of pericarditis? Colchicine, Bed rest & NSAIDs.

Don't treat idiopathic pericarditis with steroids ,because the risk of relapse when stopped



Q: The pt came to the ER with chest pain of a 6-hour duration. What is the Dx. depending on his ECG?

Acute Pericarditis (diffuse cocave-up ST elevation and occasionally depressed PR segment especially in lead II)



Q:A history of myocardial infarction a week ago A murmur is heard and current ECG is shown

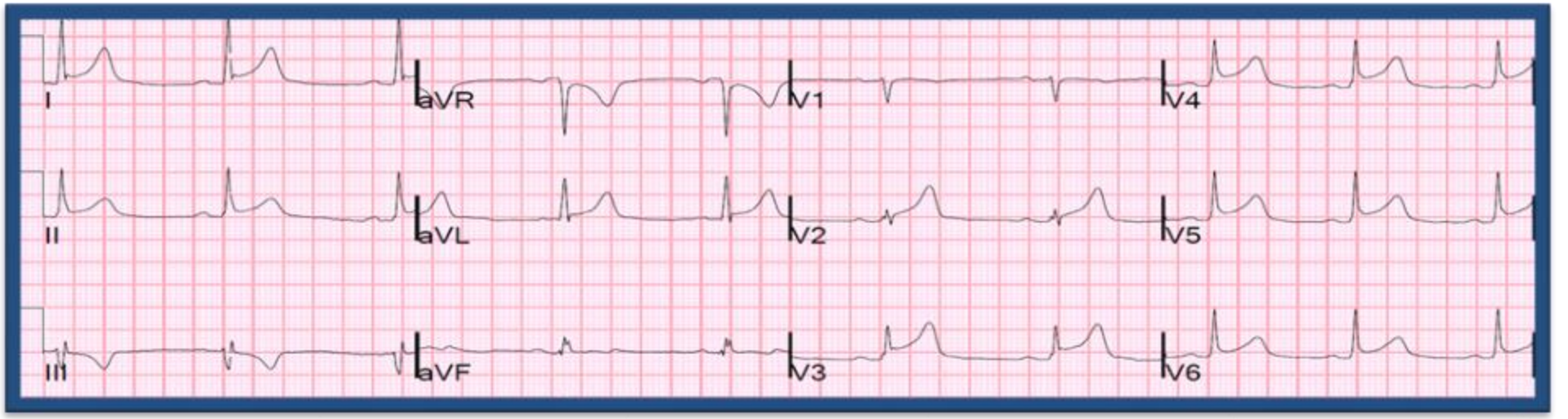
1- What is the ECG finding

Pericarditis as a post myocardial infarction complication

ST-elevation was an accepted answer

2- What is the cause of the murmur

Mitral regurgitation due to papillary muscle rupture



Q: 50 YO male, smoker, has HTN, & hyperlipidemia came to you with chest pain, effort dizziness or lightheadedness, easy fatigability, & progressive inability to exercise.

On neck examination: Parvous et tardus seen (Slowed carotid upstroke)

After Chest examination you found mid-systolic ejection murmur & you felt in left systolic thrill in left mediastinum.

1. What is Your spot Dx. ?

Aortic Stenosis

2. What is Your investigation?

Doppler Echo (very accurate in sever AS)

S&S include Classical triad of HF(LVF) ,angina, syncope with exercise

3. What are the possible Complications?

1) infective endocarditis.

2) Heart failure.

3) Cardiac arrest.

4)Coronary artery disease

4. On auscultation of the heart what is the abnomalities?

-Mid systolic ejection murmur at the (RUSB) that's radiates to the neck

-S4 gallop

-a paradoxical S2 split with sever AS

-decreased or abscent S2(Occ.)

5. What is the possible abnormality on CXR ?

LVH

6. What are The Causes?

-Congenital bicuspid valve calcification (400-70Y)

-Age related calcific degeneration of normal tricuspid valve(>75Y)

-Rheumatic heart disease (less freq.)

7.What Is the Treatment?

Aortic valve replacement(AVR)

8. Indication of AVR :

-All symptomatic pt.

-asymptomatic sever AS

Notes

- The systolic ejection murmur of AS is louder with squatting whereas the murmur of hypertrophic cardiomyopathy decreases
- The ejection click is common in bicuspid aortic valve pt but not heard with age related AS
- The severity assessed by mean valve pr. gradient or maximum velocity across the valve or low flow states (Sever AS >40mmHg ,>4 m/s , <=1cm2)

30 YO pt came to the ER suffering from SOB, palpitations, sweating & productive cough with hemoptesis , irregular irregular pulse & mid-diastolic murmur heard on the apex of the heart.

1. What the cause of the murmur?

Mitral stenosis (Diastolic rumble (low flow) with opening snap)

2. Mention the cause of the SOB.

Acute pulmonary edema (Pulmonary venous HTN)

3. What caused the irregular pulse?

AF. (most common arrhythmia seen in MS)

4.What is the best diagnostic radiological test in this case?

ECHO

5. What is your management ?

-If symptomatic , or asymptomatic sever MS (<=1.5cm2 or pulmonary HTN) >>
Percutanious mitral balloon valvotomy(PMV) is recommended
-All nonpregnant pt with AF due to MS should be on warfarin

- MS almost always due to rheumatic fever
- MS complication : AF (common) , (2ry pulmonary HTN (the main comp.) , HF
- Associated with Lt atrial enlargment
- S1 is enhanced ,sometimes snaping
- On ECG, biphasic P wave (enlarged Lt atrium)
- The pt may be present with embolic event

Vascular disease

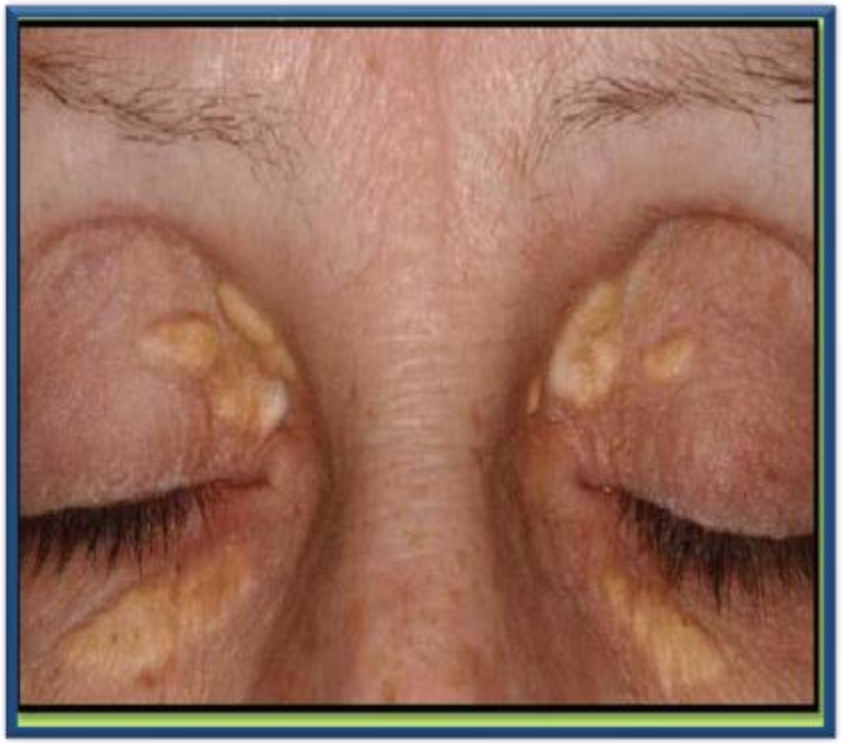
Q1:

A) What is this sign?

Xanthelasma.

B) what is the cause of it?

Hypercholesterolemia



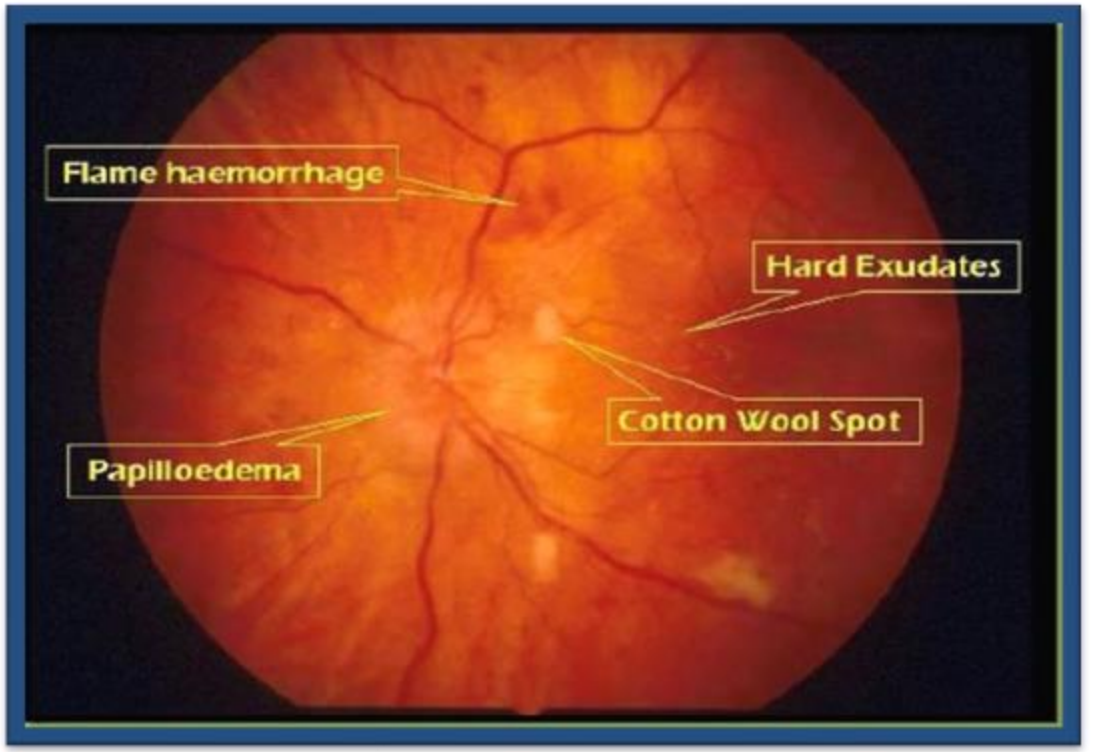
Q2: 60 YO male pt, diabetic & hypertensive. Mention pathologies seen by ophthalmoscope.

1-flame shape hemorrhage.

2-hard exudate..

3-cotton wool spot

4-papilloedema



Hypertensive retinopathy

Grade 1: **Arteriolar thickening, tortuosity and increased** reflectiveness ('silver wiring')

Grade 2: **Grade 1 plus constriction of veins at arterial crossings** ('arteriovenous nipping')

Grade 3: **Grade 2 plus evidence of retinal ischaemia (flame-shaped** or blot haemorrhages and 'cotton wool' exudates)

Grade 4: Grade 3 plus papilloedema

Q3: A pt presented to ER with severe chest pain. On P/E he had some Marfanoid features& this was his Chest X-Ray.

1-What is your Dx?

Dissecting Aortic Aneurysm.

2-predisposing conditions?

A-Aortic atherosclerosis and hypertension

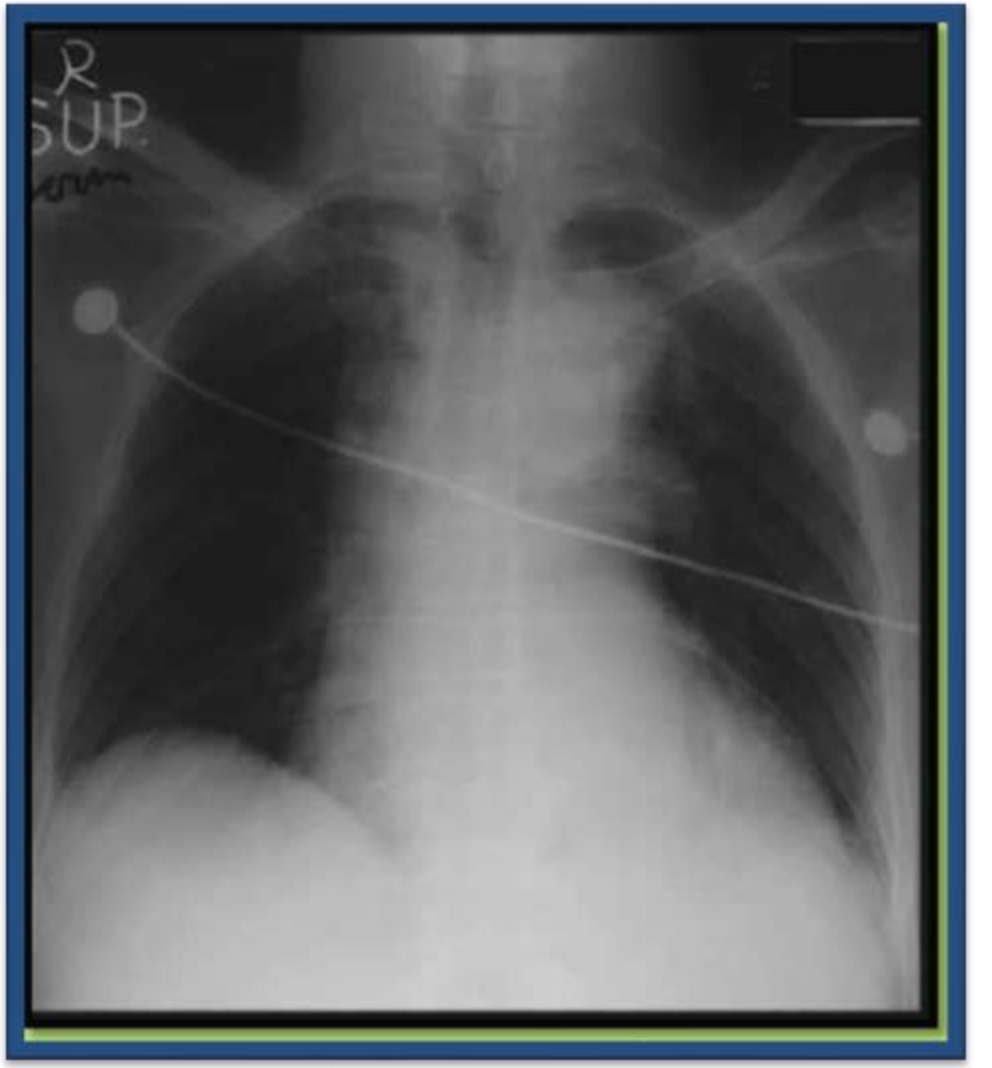
B-thoracic aortic aneurysm

C-aortic coarctation

D-previous aortic surgery

F-Marfan's syndrome

D-trauma and pregnancy



Q4:

This patient presents with sudden onset stabbing retrosternal chest pain.

1-what is your diagnosis?

Aortic Dissection.

2-This condition is further classified into:

Type A: Involving the ascending aorta

Type B:sparing the ascending aorta

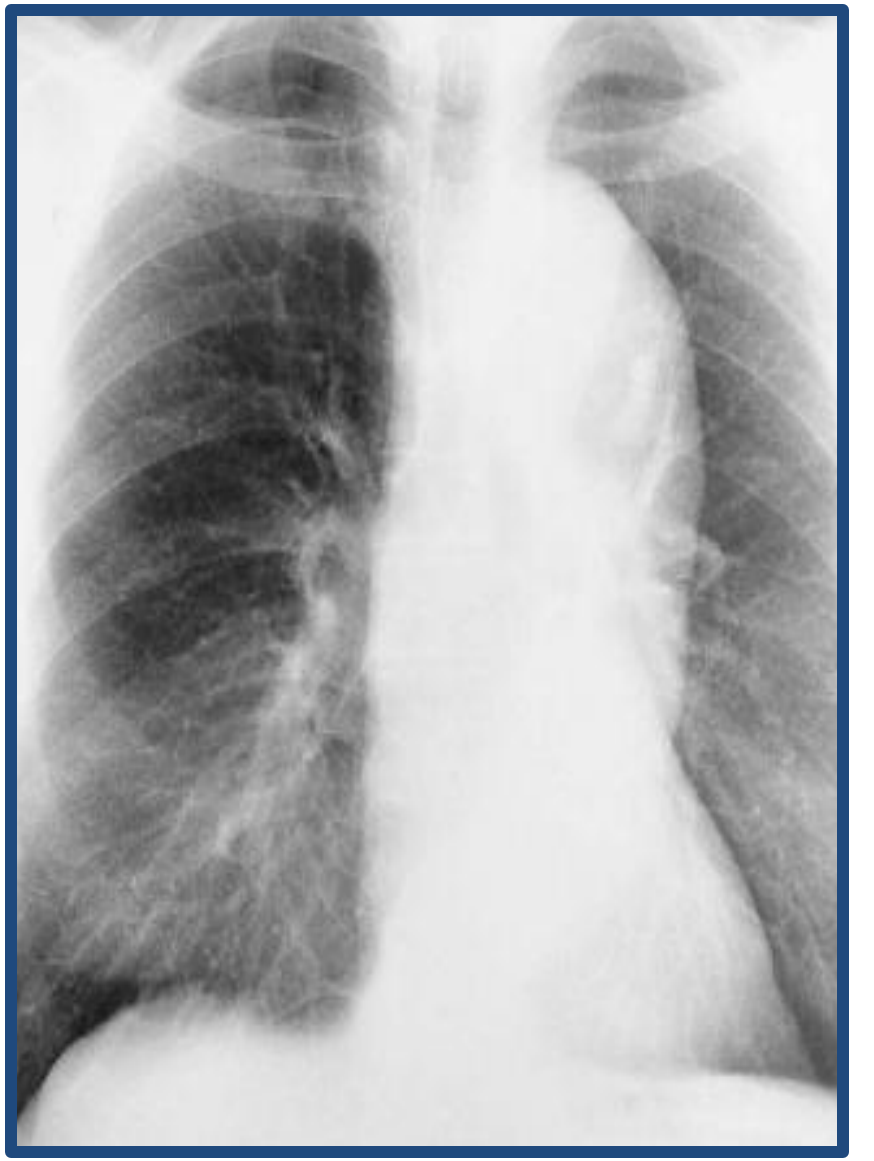
3-investigation:

A- CXR: may show broadening of the upper mediastinum and distortion of the aortic 'knuckle', but these findings are absent in 10% of cases.

B-Transthoracic echocardiography can only image the first 3–4 cm of the ascending aorta

C-transoesophageal echocardiography,

CT and MRI are all very useful.



4-Early mortality of acute dissection is?

1–5%/hr.

5-Initial management:

1-pain control and IV labetalol (target systolic BP < 120 mmHg)

2-Endoluminal repair with fenestration of the intimal flap or insertion of a stent-graft may be effective.

Q5: A 25-year old male with history of hypertension.

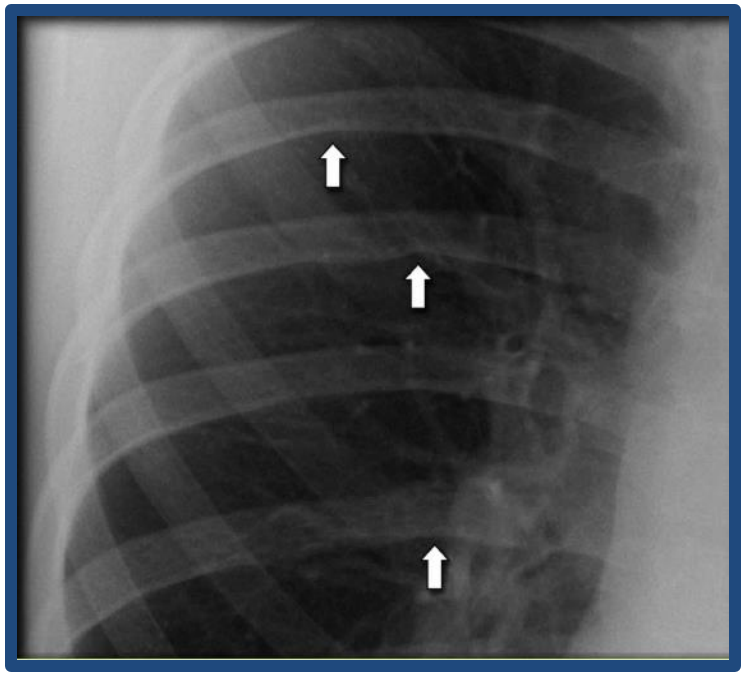
1-What is the radiological finding? Rib notching sign .

2-indicate what :

Coarctation of the aorta

3-This condition is associated with other abnormalities, including:

bicuspid aortic valve and 'berry' aneurysms of the cerebral circulation



4-Finding:

1-BP: raised in the upper body but normal or low in the legs.

2-Femoral pulses: weak, and delayed in comparison with the radial pulse.

3-Systolic murmur: usually heard posteriorly, over the coarctation.

5-Investigation:

1-CXR: may show changes in the contour of the aorta and

notching of the under-surfaces of the ribs from collateral vessel

Development.

2-MRI: ideal for demonstrating the lesion

6-Management:

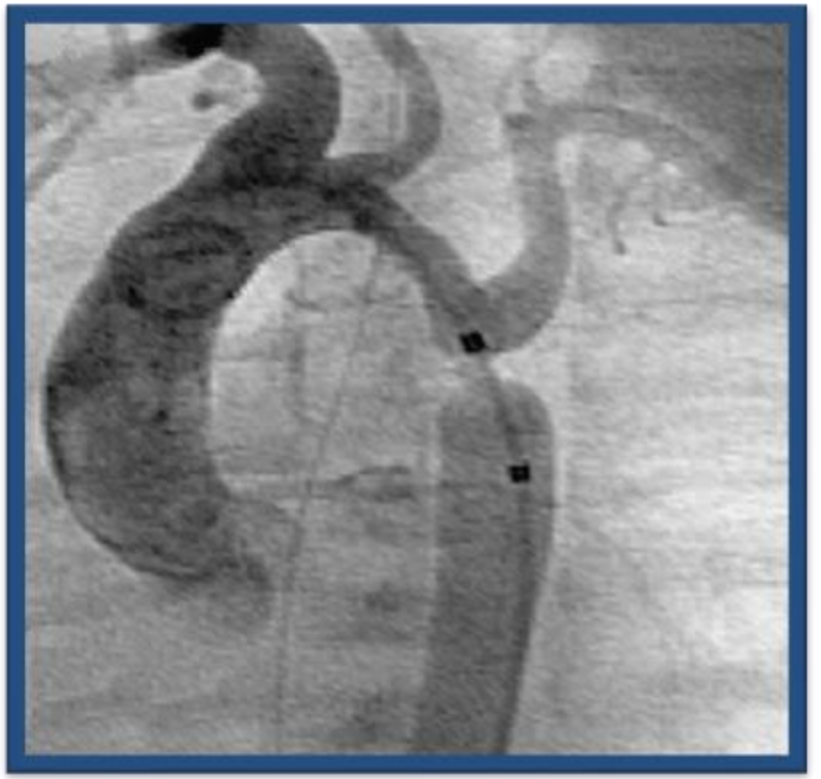
1-Surgical correction: advisable in all but the mildest cases. If this is done sufficiently early in childhood, persistent hypertension can be avoided but patients repaired in late childhood or adult life often remain hypertensive.

2-balloon dilatation.

3-Recurrence of stenosis: may be managed by balloon dilatation.

Q6: The patient is hypertensive, what sign the doctor discovered while examining this patient?
(not the same picture)

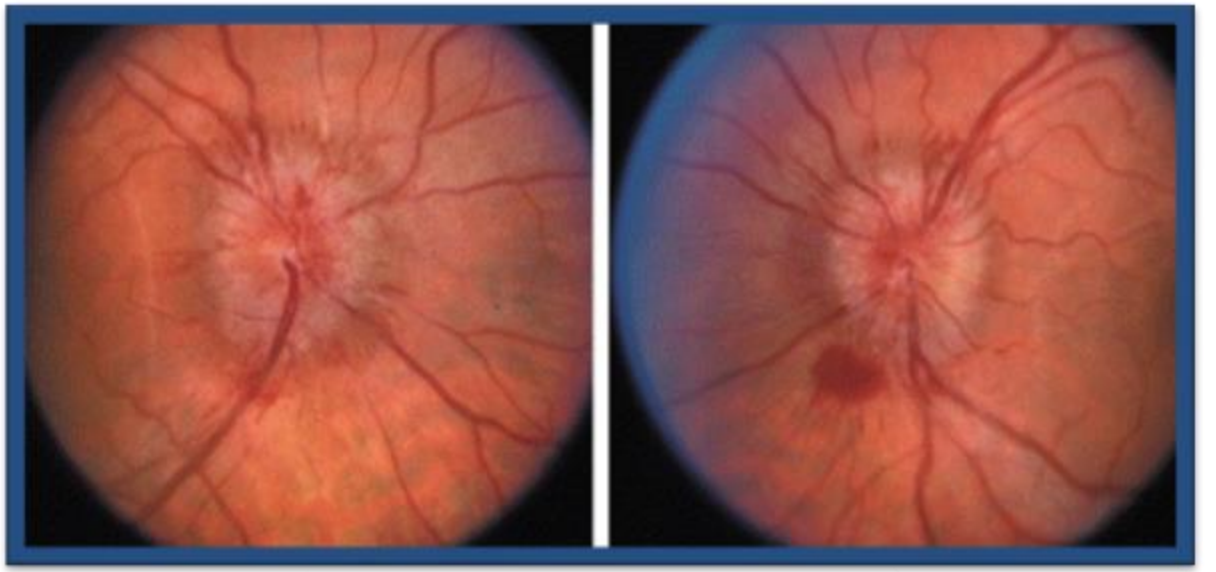
Radio-Femoral delay>> Coarctation of aorta



Q7: The patient presented with early morning headache. What is this sign and what is the underlying cause?
(not the same picture)

Papilloedema

Increased ICP



Q8:A- what are the findings?

dots and blots, neovascularizton

B- mention 2 complications

vitreous hemorrhage, RD, loss of vision, decrease visual acuity

Q9:Patient with hx of long standing HTN

A- what's your finding?

LVH.

B-symptoms :

1-Effort-related symptoms (angina and breathlessness)

2-arrhythmia(may lead to sudden death)

C-signs:

1-Harsh ejection systolic murmur radiating to the neck (often with a thrill).

2-Soft second heart sound.

3-Heaving but undisplaced apex beat.

4-arterial pulse is jerky.

D-Investigations:

1- ECG :usually abnormal and may

show features of LV hypertrophy or deep T-wave inversion

2-Echocardiography is usually diagnostic.

E-Management:

1-(Beta-blockers and rate limiting calcium antagonists)can help to relieve symptoms and sometimes prevent syncopal attacks but no pharmacological treatment is definitely known to improve prognosis.

2-partial surgical resection or by iatrogenic infarction of the basal septum using a catheter-delivered alcohol solution(to improve outflow obestruction).

3-ICD should be considered in patients with clinical risk factors for sudden death.

Risk factors for sudden death in hypertrophic cardiomyopathy

- A history of previous cardiac arrest or sustained VT
- Recurrent syncope
- An adverse genotype and/or family history
- Exercise-induced hypotension
- Non-sustained VT on ambulatory ECG
- Marked increase in LV wall thickness

Q10: This patient had unilateral lower limb swelling & redness.

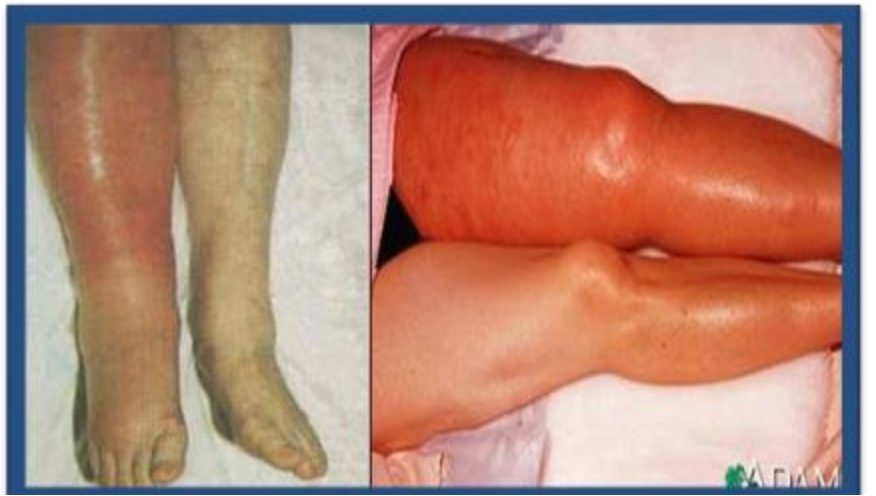
- What's the investigation that you'll do to diagnose this case?

1- CBC
2- D dimer
3- Venous Doppler Ultrasound.

- What is your differential diagnosis?

A-DVT
B-Cellulitis
C-ruptured baker cyst
D-Musculoskeletal injury

- Management: ادرسوه من المحاضرة



Q11: This afebrile patient presents with an acute episode of shortness of breath

1-what is your ddx:

PE

2-symptoms of this condition:

A- Faintness or collapse
B-central chest pain
C-apprehension
D-severe dyspnoea

3-signs:

A-tachycardia
B-hypotension
C-↑JVP
D-right ventricular (RV) gallop rhythm
E-split P2
F-severe cyanosis
G-↓urinary output

4-INVISTIGATION:

A-CXR :Usually normal

B-ECG:S1Q3T3, anterior T-wave inversion, right bundle branch Block

C-ABGs:Markedly abnormal with ↓*PaO2 and* ↓*PaCO2; metabolic* acidosis

D-CT pulmonary angiogram (definitve diagnose)

E-*Ventilation/perfusion scanning: seldom used nowaday*

D-*Echocardiography*

5-Management:

1-Sufficient oxygen should be given to all hypoxaemic patients to Restore *SpO2 to > 90%.*

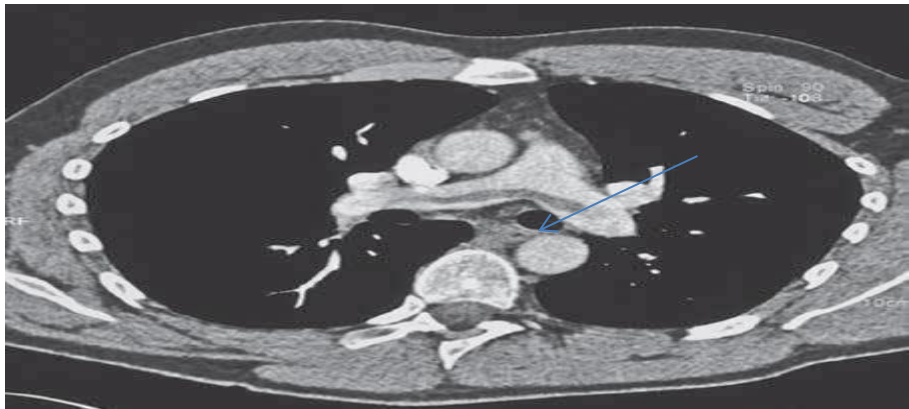
2-Hypotension should be treated using *IV fluid* or plasma expander.

3-Opiates may be necessary to relieve pain and distress but should be used with caution.

4- *Anticoagulation: This should be commenced immediately in* patients with a high or intermediate probability of PE, but can be safely withheld from patients with a low clinical probability.

5-External cardiac massage may be successful in the moribund patient by dislodging and breaking up a large central embolus.

CT pulmonary angiogram. The arrow points to a saddle embolism in the bifurcation of the pulmonary artery



Risk factors for venous thromboembolism

1-Surgery: Major abdominal/pelvic surgery; hip/knee surgery; post-operative intensive care
2-Pregnancy/puerperium
3-Cardiorespiratory disease: COPD, congestive cardiac failure or other disabling
Disease
4-Lower limb problems :Fracture; varicose veins; stroke/spinal cord injury
:5-Mailignant dx: Abdominal/pelvic; advanced/metastatic; concurrent chemotherapy
6-Miscellaneous: Increasing age, previous proven VTE, immobility, thrombotic disorders, truma.

Q12: 65 year.old male, was diagnosed with DM (for 15 years), HTN (for 10 years), he is taking atenolol and glimepiride, his blood pressure in sitting is 130/85, and in standing is 110/70, in the last month he developed dizziness.

1.What is the cause of the dizziness?

Posturalhypotension .

2. Mention two causes for this condition in this patient

1-Side effect of bata blocker drugs

2-sympathetic degeneration (e.g. diabetes mellitus, ageing)

3.Other causes for this condition:

1-hypovolaemia (e.g. excessive diuretic therapy)

2-drug therapy (e.g. vasodilators, antidepressants)

4.Management:

1-Withdrawal of unnecessary Medication.

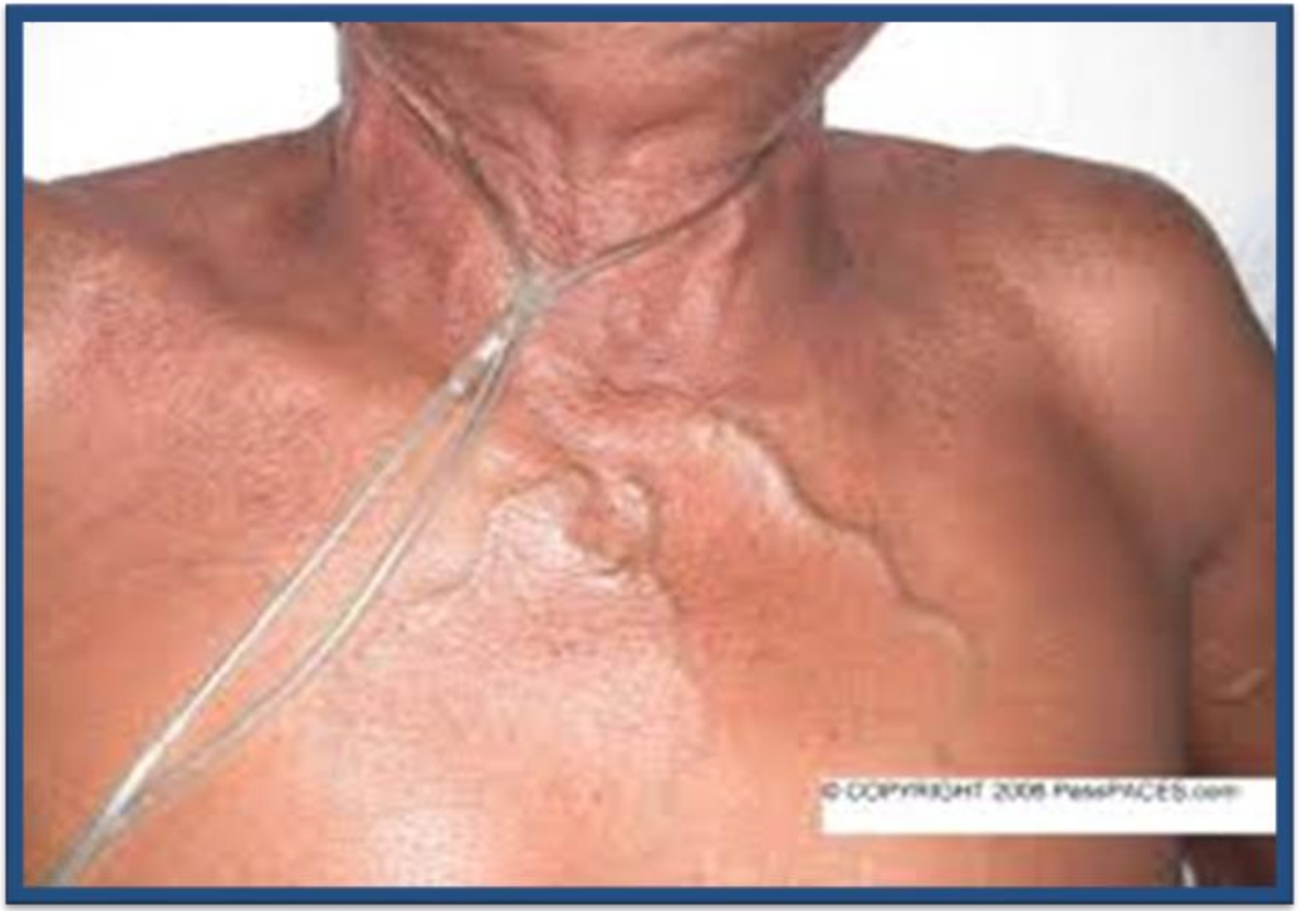
2-graduated elastic stockings.

3-in some cases, treatment with fludrocortisone may be helpful.

Q13:A 75 year-old man presented with hoarseness of voice, cough and weight loss
What is the most likely cause of his appearance?

Superior Vena Cava compression

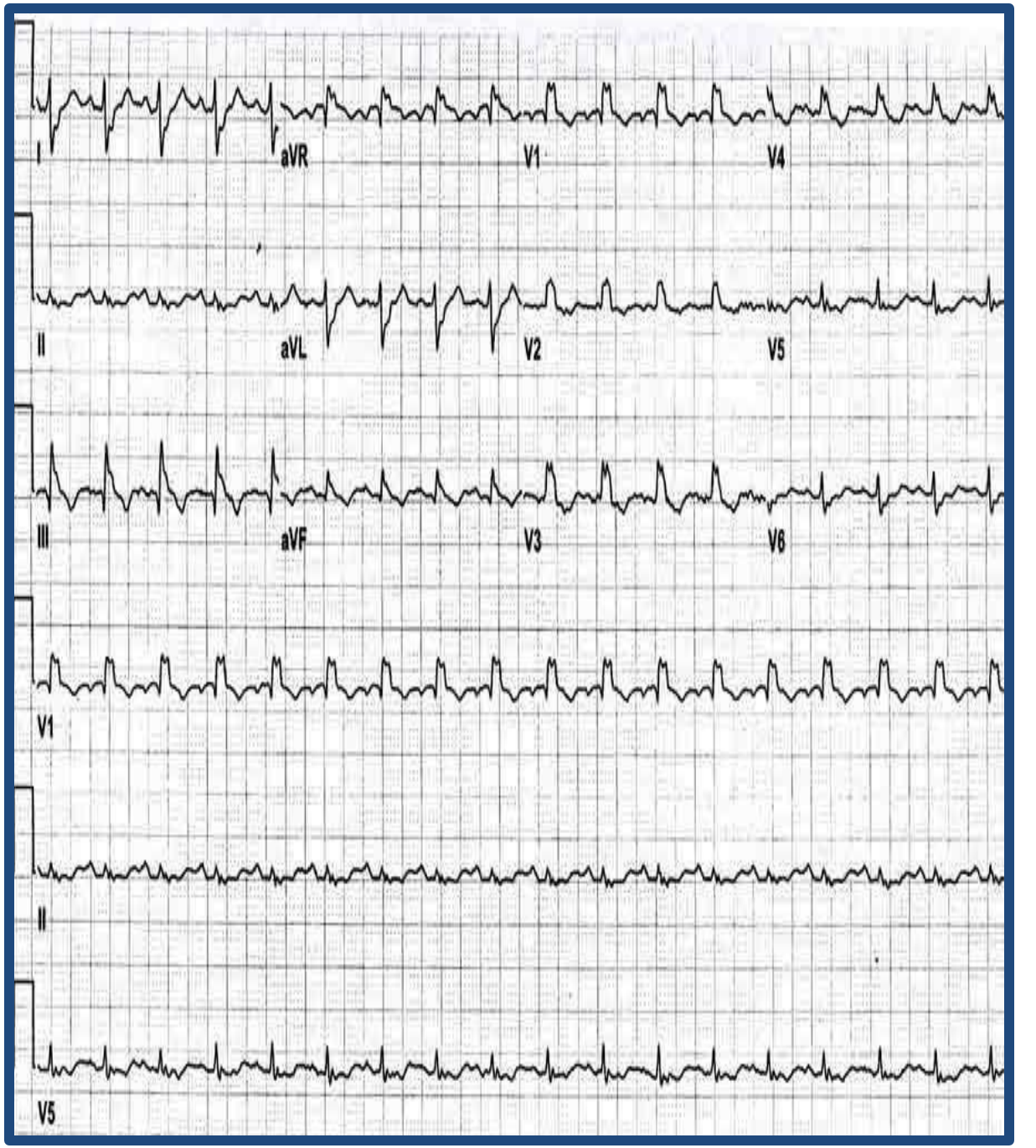
[This is dilation of the collaterals is due to superior vena cave compression, which may be caused by thyroid cancer or lung cancer].



Pulmonary embolism and edema

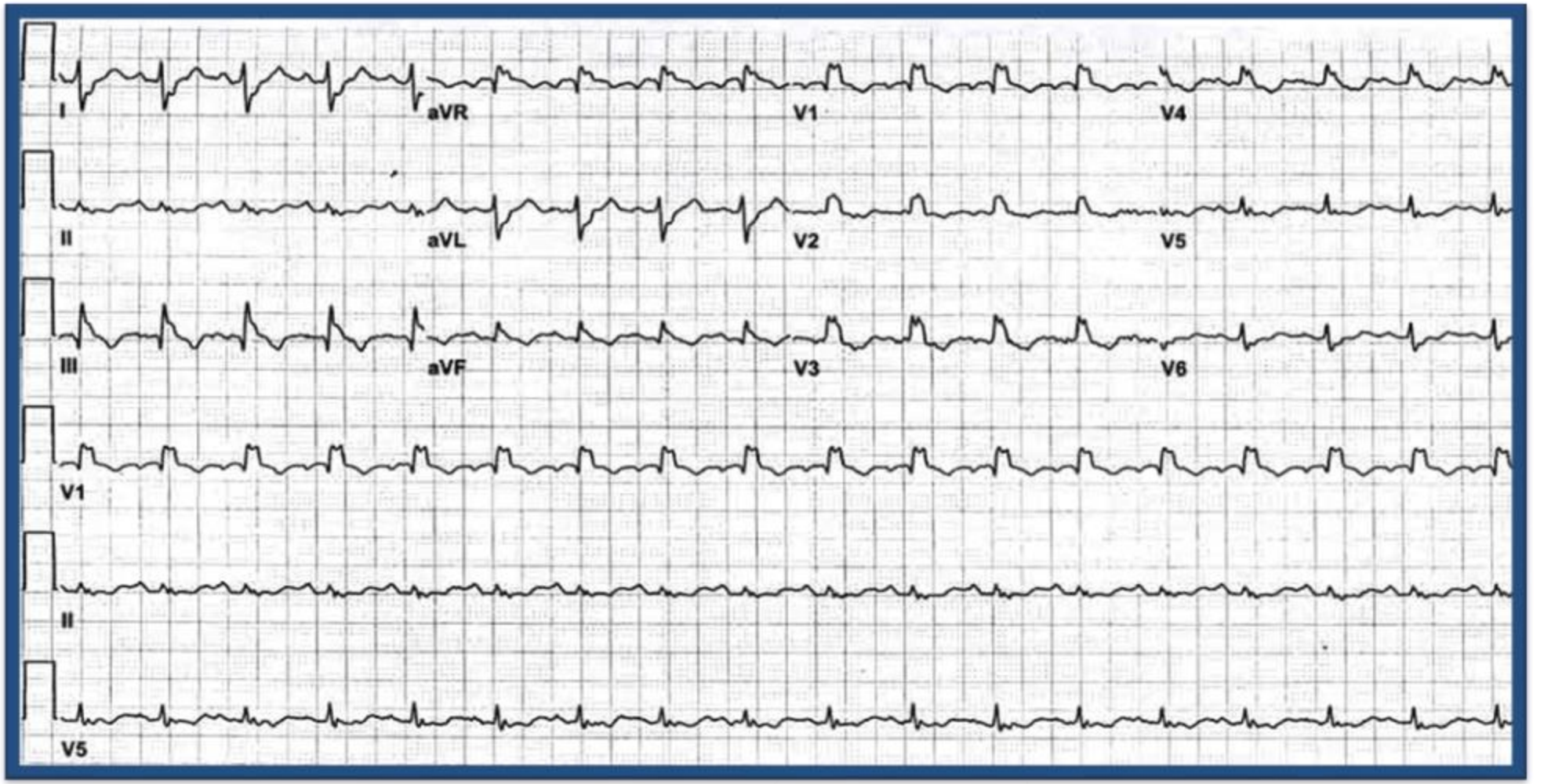
Q: YO female, bedridden , presented with sudden sob and chest pain, give two abnormalities in this ECG ?

S1Q3T3, With Wide QRS Complex And RBBB (inverted (T)from v1 to v4 (Pulmonary Embolism),



Q:This pt presented with palpitation, he is known case of recurrent attacks of DVT. Give 2 abnormalities in this ECG?

S1Q3T3, RBBB pattern which is suggestive of pulmonary embolism.



Q: An ECG for a 70 years old woman, bedridden, hospitalized for surgery and suddenly developed severe SOB.

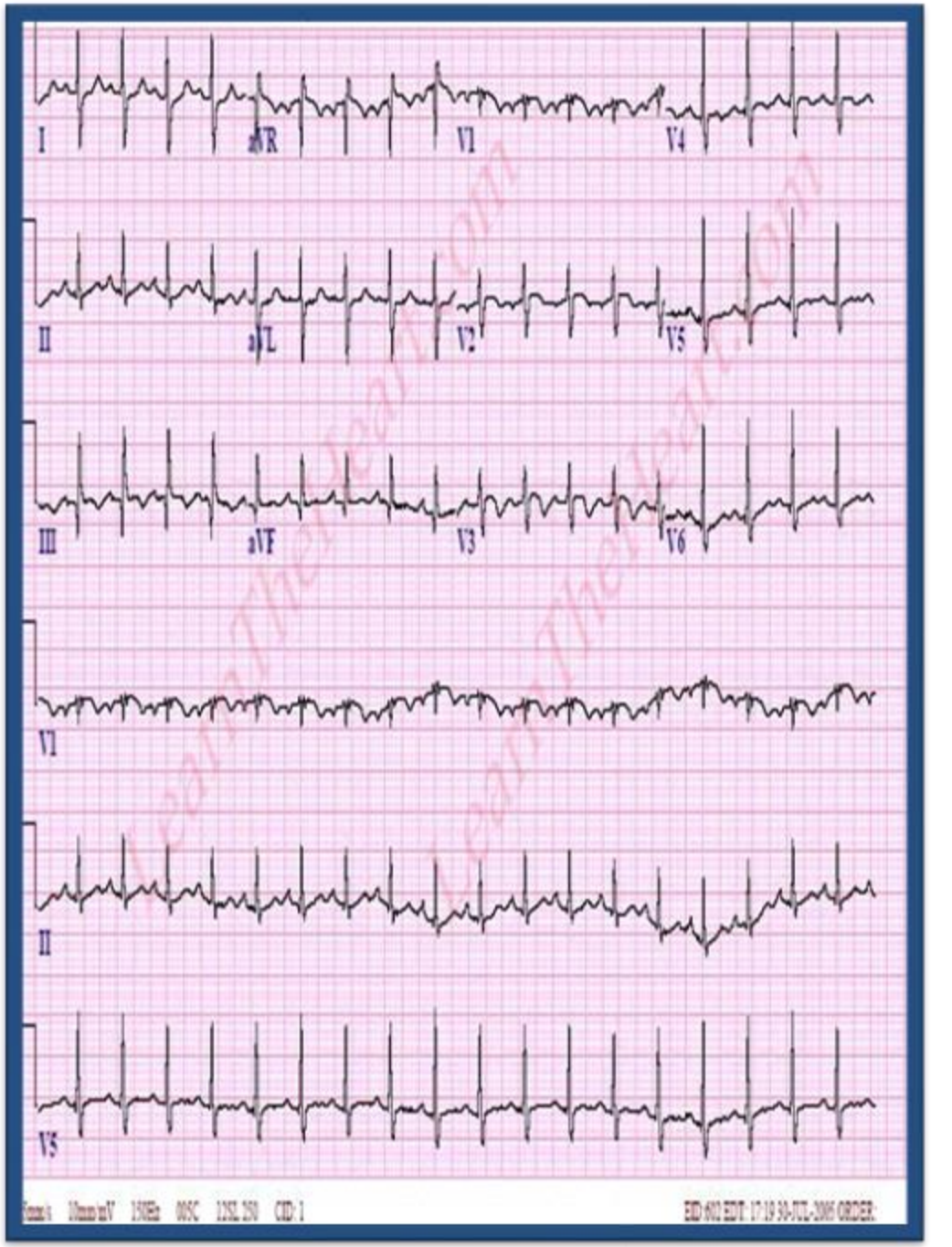
a) What's your diagnosis?

PE

b) What changes will you find on the chest x-ray?

usuallly Normal

atelectasis or plural effusion may be present, dilated pulmonary artery, widge shape opacites or cavitation



Q:

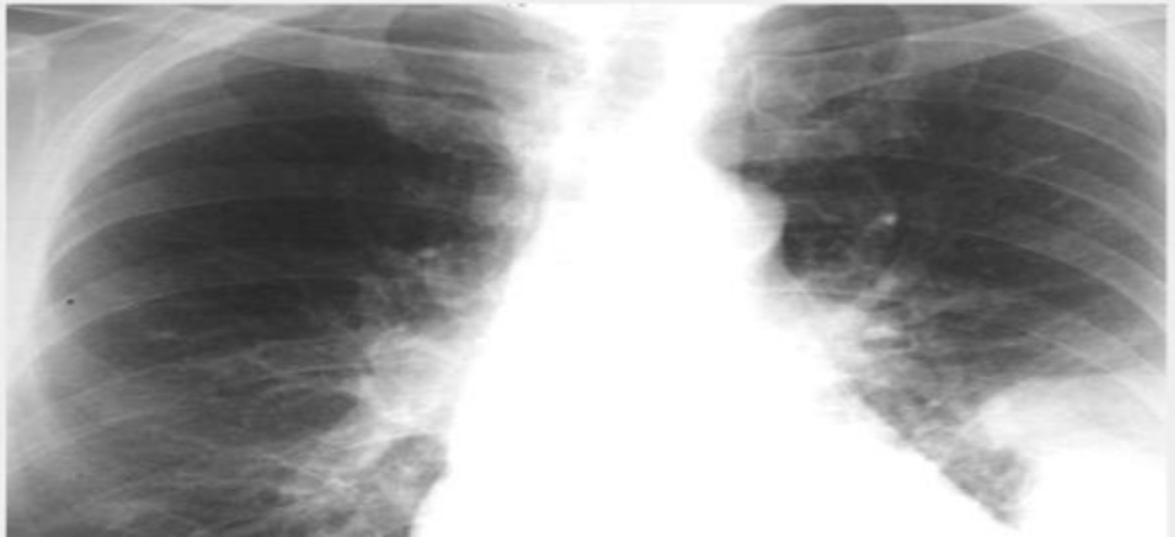
What is your diagnosis (most likely)?

Pulmonary embolism

What is the differential diagnosis ?

1. Unstable angina/acute MI
2. Pneumonia
3. Pneumothorax
4. Pulmonary edema
5. Pericarditis
6. Dissecting aortic aneurysm

What is the clinical presentation of PE ?



What is the SYMPTOM of PE ?

Dyspnea 73%

Pleuritic chest pain66%

Cough 37%

Hemoptysis13%

1/3 will have sign and symptom of DVT

What is the sign of PE?

Tachypnea 70%,rales 15%,tacyhycardia51%,S4,increased of p2

What are the investigation required in this case ?

- V-Q Scan
- Spiral CT

Most common tests for pulmonary embolism

- Angiography " venography" → golden standerd
- Chest X-Ray
- Electrocardiogram
- D-dimer
- Venous Duplex US (most common test for DVT)
- Arterial Blood Gas

What is the treatment ?

The same as DVT

What is the treatment of PE?

-supplemental oxygen to correct hypoxemia

-acute anticoagulant therapy with either unfractionated or low molecular weight heparin the goal ptt 1.5-2.5

-Oral anti coagulant with warfarin or one of novel oral anti coagulant(rivaroxaban) for long term treatment used after 24h of heparin

***Goal INR2-3 ,continue 3-6 months ,some pt at significant risk for recurrent PE,may considered for life long anticoagulant ,thrombolysis for massive PE

Q:Patient has clubbing, What is your radiological diagnosis?

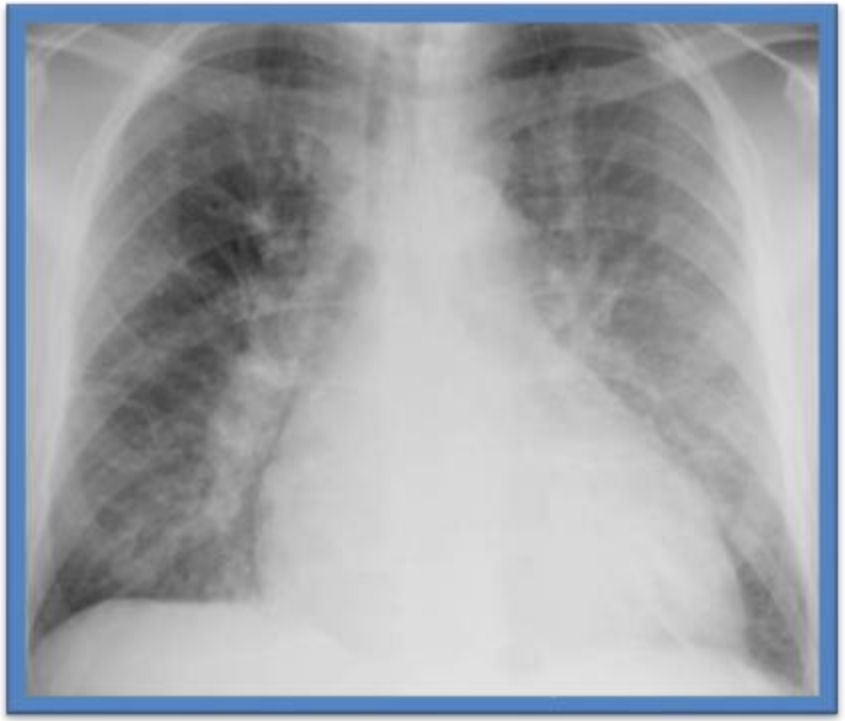
Pulmonary Edema

What's your finding on x ray?

Cardiomegaly, prominent interstial marking

What is the clinical presentation for this picture?

dyspnea,orthopnea,PND,nocturnal cough

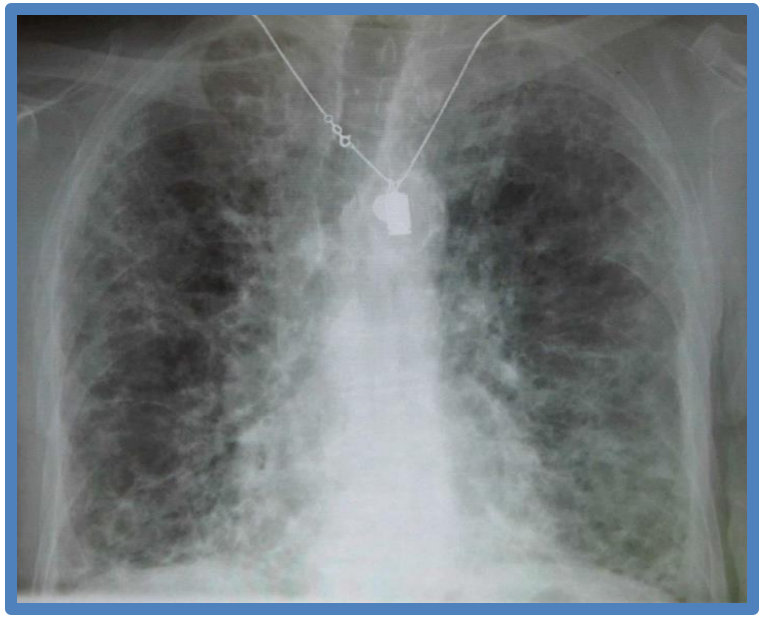


Q:A known case of Rheumatoid arthritis presents with progressive shortness of breath, describe your finding in this X ray

Diffuse Reticulonodular infiltrates indicative of pulmonary fibrosis secondary to Rheumatoid arthrities

What are the clinical feature based on this x-ray?

Dyspnea, non productive cough,fatigue, symptom of connective tissue disorder, rales at base of lung



RS

DONE BY :

همام الليمون فرح الكساسبة

رانيا الطراونة سبأ النعيمات

بهاء الشمالي علاء أبازيد

مهدي سليحات محمود التوبة

محمود الشرمان اسامة طعامنة

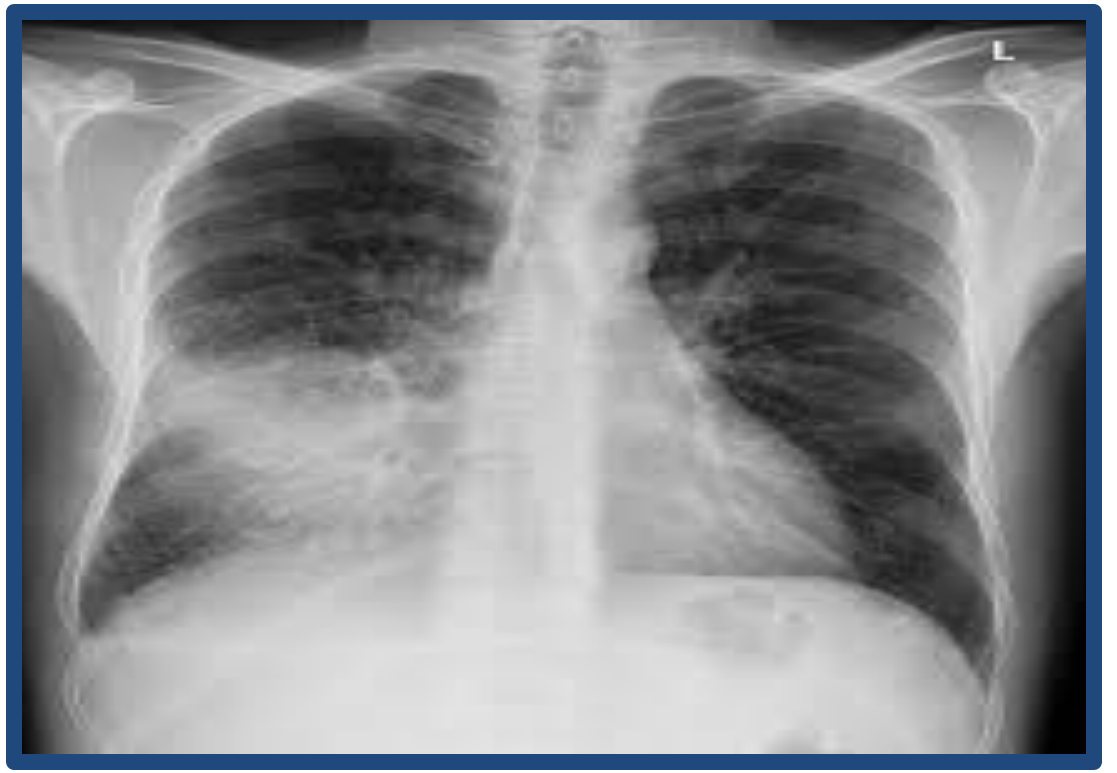
REFERENCES :

- DAVIDSON
- STEPUP

Lung Cancer

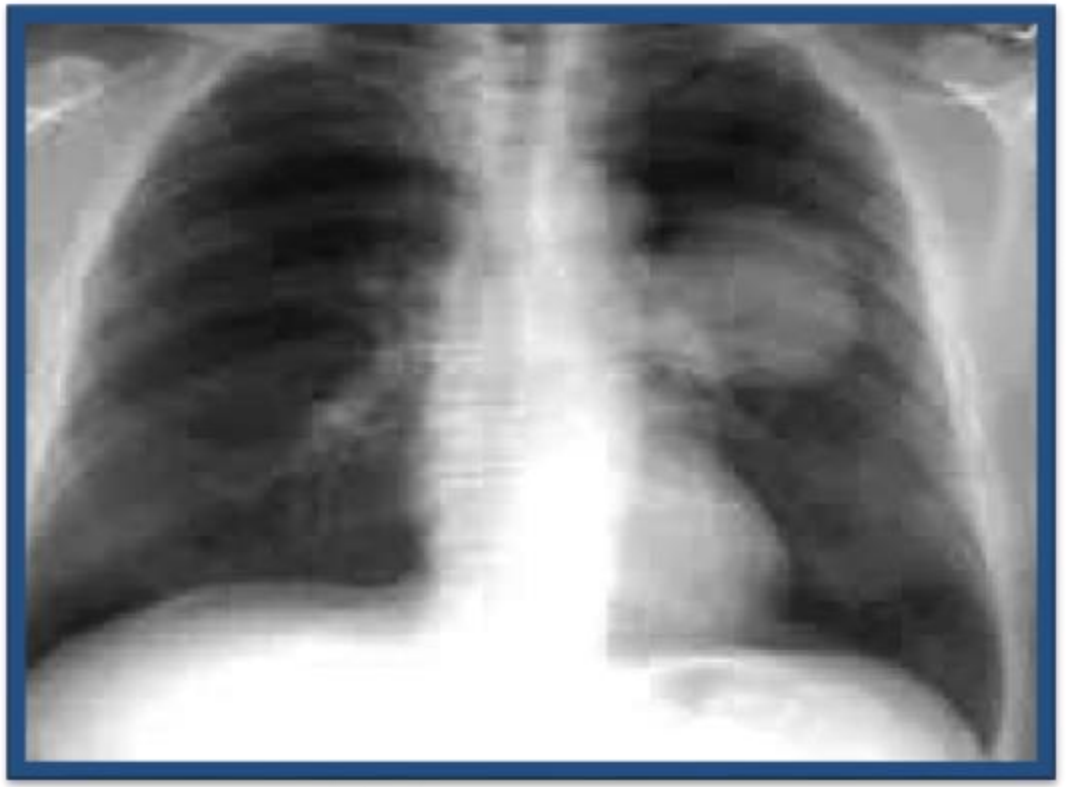
Q:55 year old patient presented with hemoptysis and weight loss of 2 weeks duration and has the following chest xray.
Your diagnosis is?

Bronchogenic Carcinoma (Lung cancer)



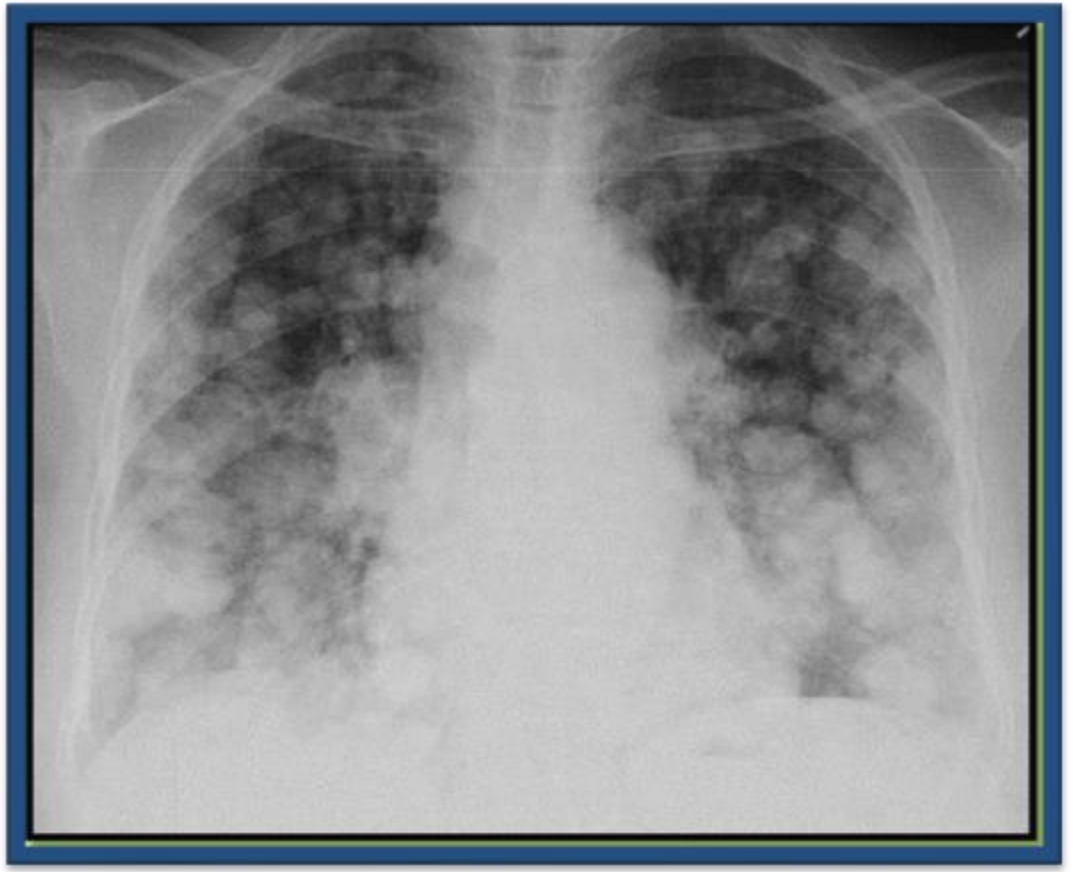
Q: This patient presented with hemoptysis.

1. What's your diagnosis?
   Lung cancer
2. What's your next investigation?
   Bronchoscopy & biopsy



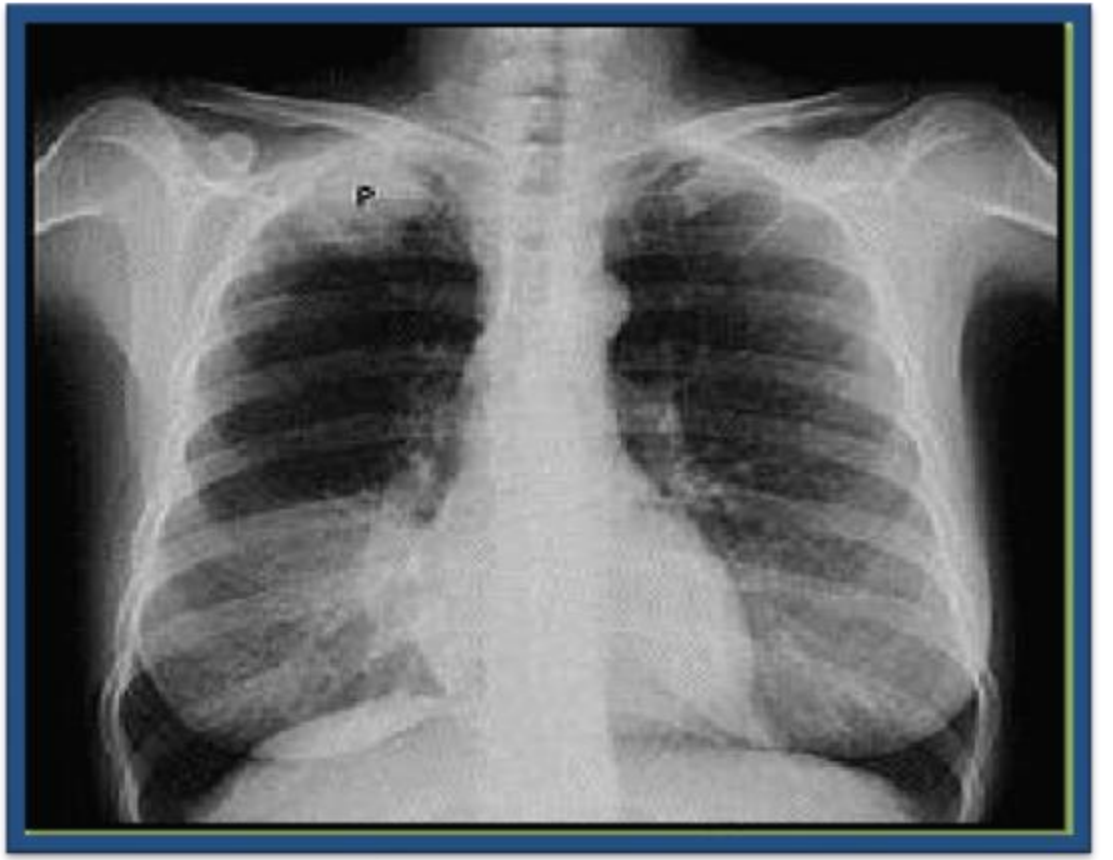
Q: Patient with back pain, hematurea, Weight loss, anorexia & general weakness. What is the Dx.?

Lung metastasis.



Q: This pt presented with ptosis & miosis on the right side of his face. Mention 2 findings can be seen in this pt's hand.

1. Clubbing. 2. Muscle weakness. 3. Numbness/Parasthesia. 4..muscle atrophy



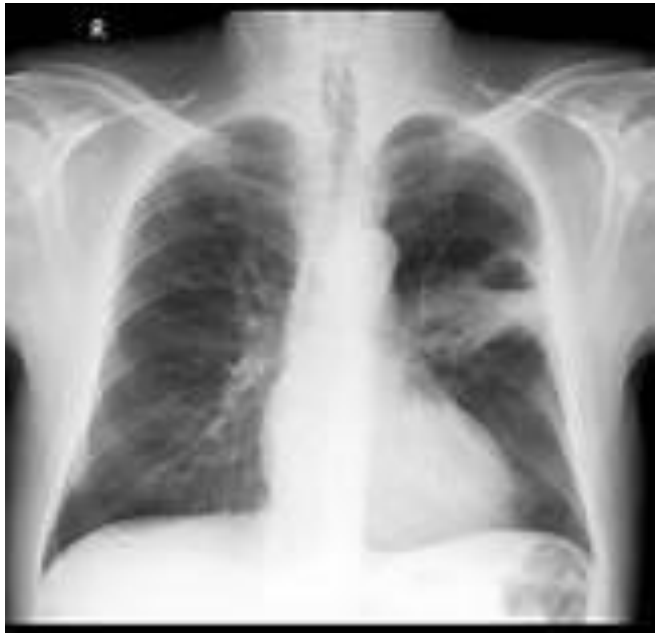
Q: 65 YO male smoker came with cough, hymoptosis, loss appetite, polyurea & polydepsia.

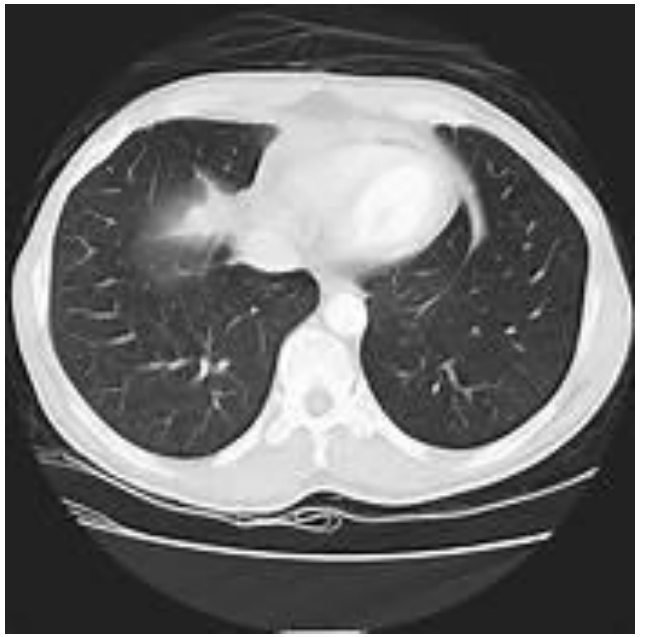
What is the dx?

Squamous cell carcinoma(lung cancer)

what's the cause of polyurea?

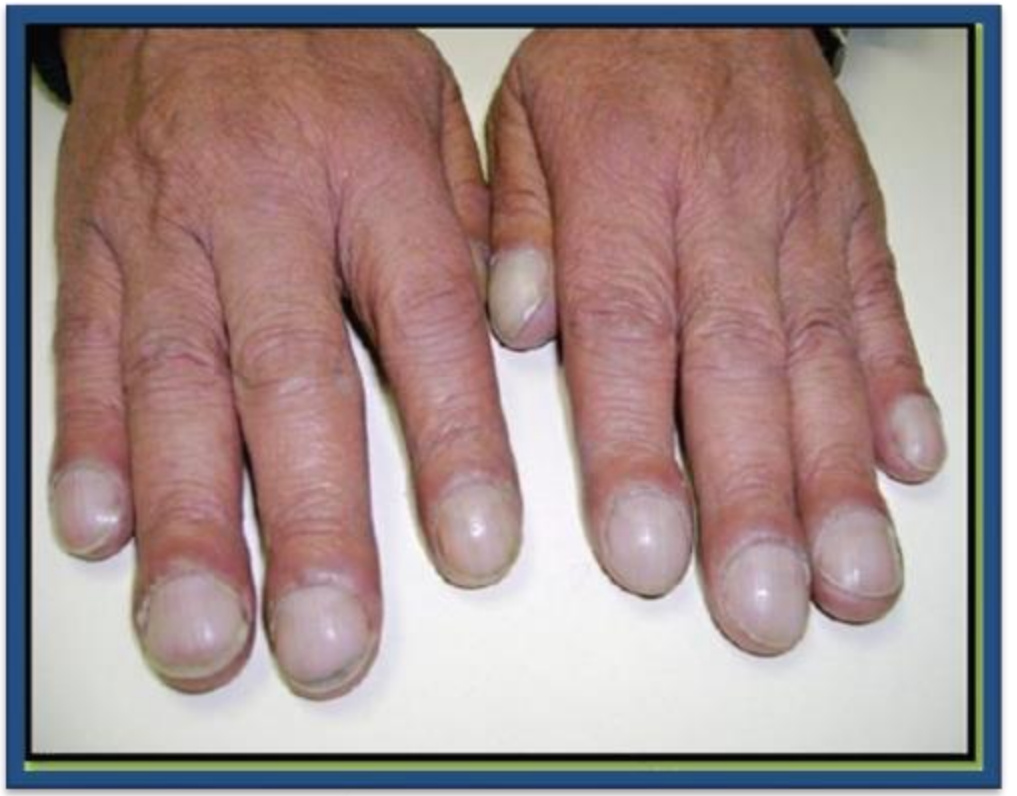
Hypercalcemia.





Q: Hx. of patient with bronchogenic carcinoma, what is the cause of his constipation ?

Hypercalcemia.



Q: A patient with significant weight loss and 100 pack year smoking history.

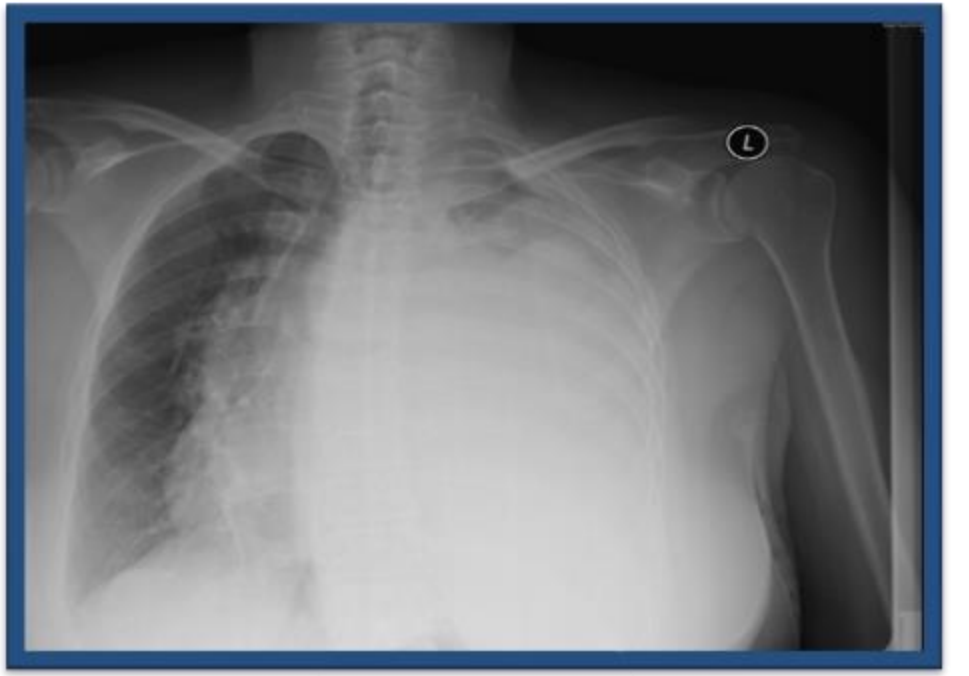
1- Diagnosis

Lung cancer

2- Cause of the finding

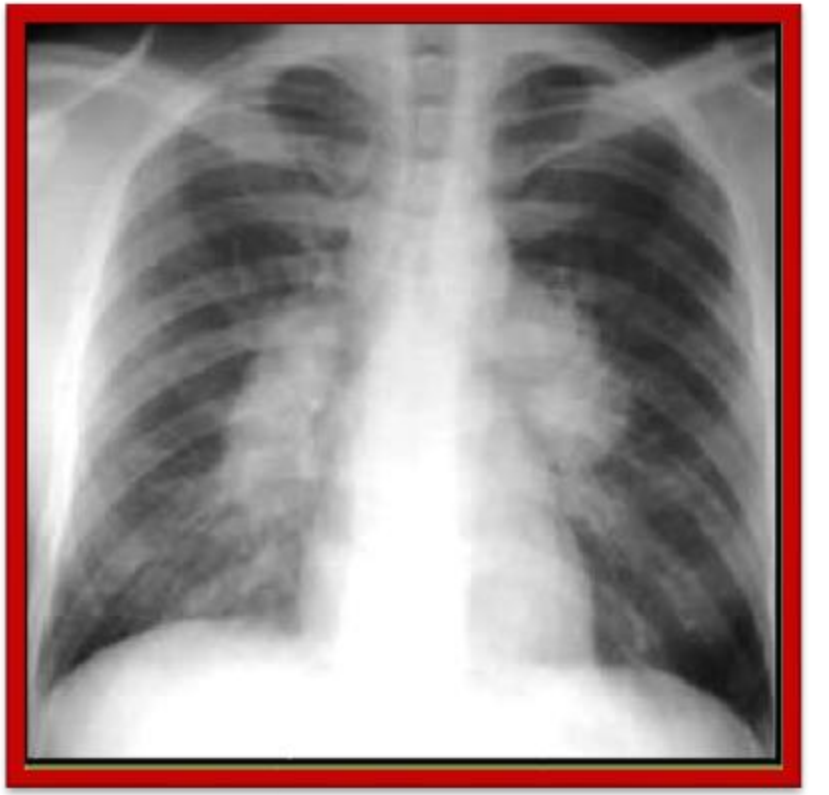
malignant pleural effusion

****Note left lung opacity and tracheal deviation away from the opacity (vs atelectasis).**



Restrictive Lung Disease

- **This pt came with red nodule on lower limbs.**
- **Mention 2 findings.**
- 1.bilateral hilar lymphadenopathy.
- 2.reticulonodular infiltration
- **What is the Dx?**
- Sarciodosis.
- **Best method for diagnosis ?**
- Flexible bronchoscopy with bronchial wall biobsies



- **Treatment** : 75% recover without treatment

-Inhaled steroid if the disease primarily in the bronchi

-Respiratory corticotropin injection (Acthar) is an FDA approved treatment of respiratory symptoms of sarcoidosis

-Systemic steroid indicated in (persistent hypercalcemia / evidence of other organ involvement)

-Others: hydroxychloroquine/infliximab/methotrexate/thalidomide

Q: 35 YO female, known case of AF, on amiodarone. Chiefly complaining of dyspnea FEV1\FVC >80%, FVC 60%, TLC 55%,

DLCO low

1. what is this ventilatory pattern?

Restrictive pattern.

2. what is the cause of her dyspnea?

Amiodarone induced lung fibrosis.

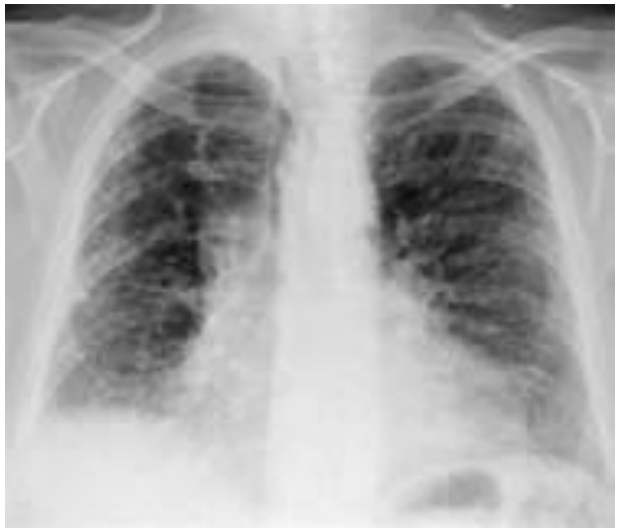
Q: A 45 y old Female was complaining of shortness of breath for ... And these painful nodules on the lower limbs ,

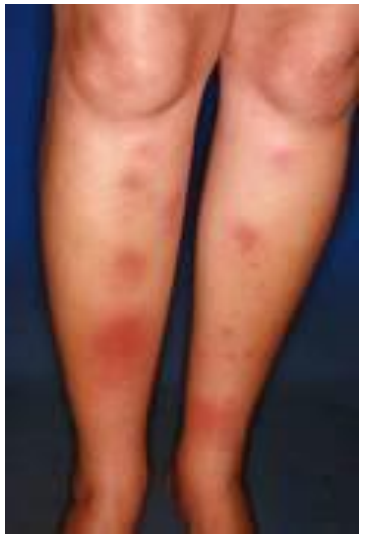
what is your diagnosis ?

Sarcoidosis

What is the name of these lesions ?

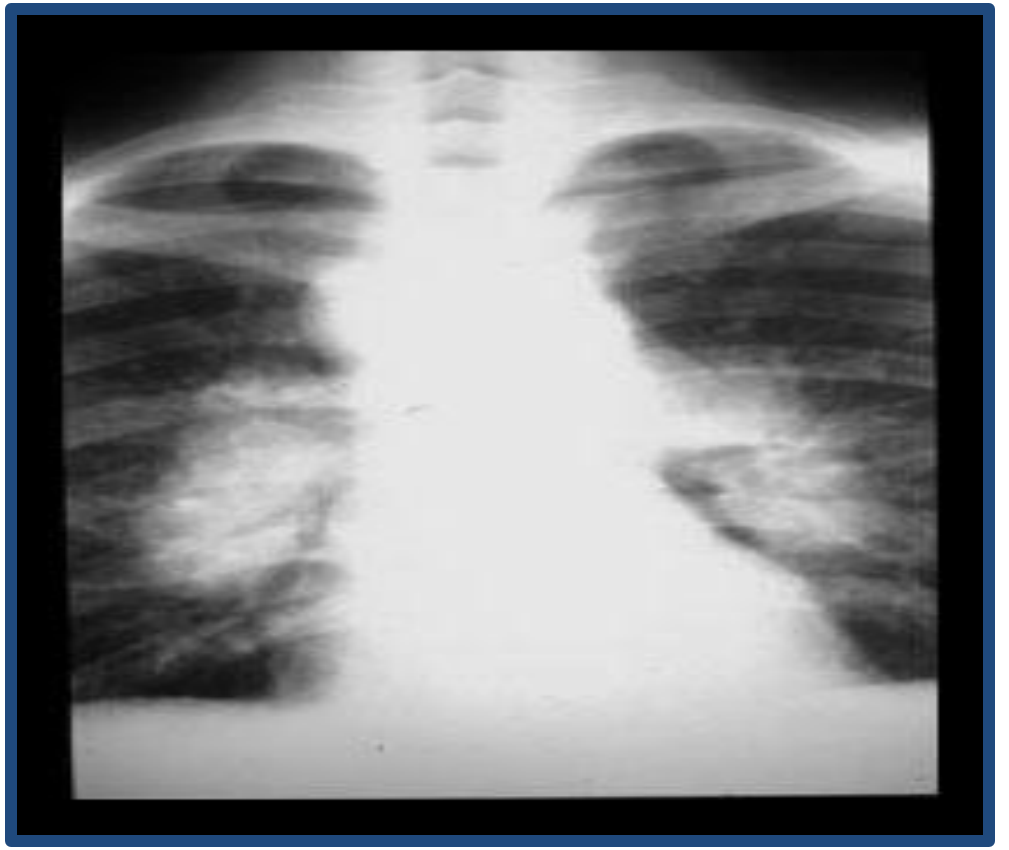
Erythema nodosum





Q: A female pt presented with eye manifestations, sob, and this cxr, what is your diagnosis ?

Sarcoidosis (Lymphadenopathy on the cxr)



Q: A 28 year old female patient presents with those painful nodules on her legs. Write down two possible diagnoses

A. Sarcoidosis
B. Inflammatory Bowel Disease(Ulcerative Colitis /Crohn's Disease)

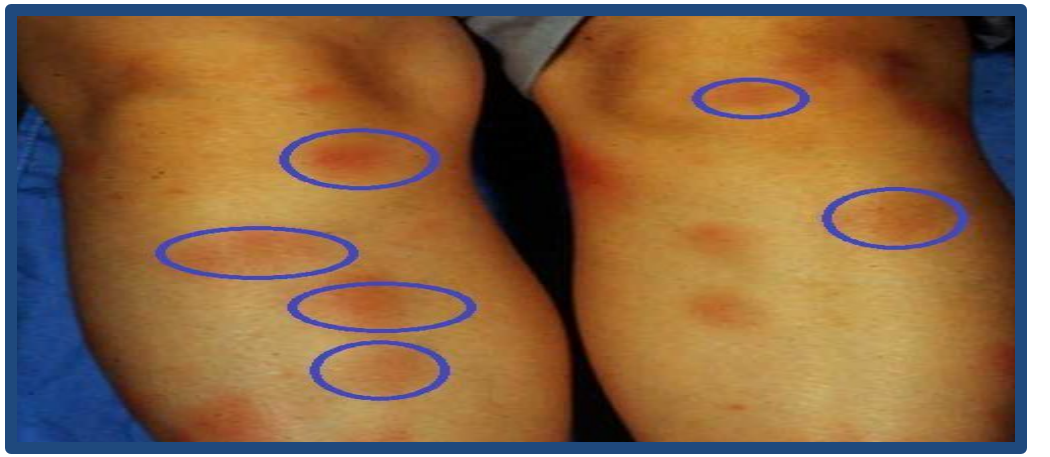
Other ddx :

Post strep infection (most common)

TB

Bahcet disease

Takayasu vasculitis



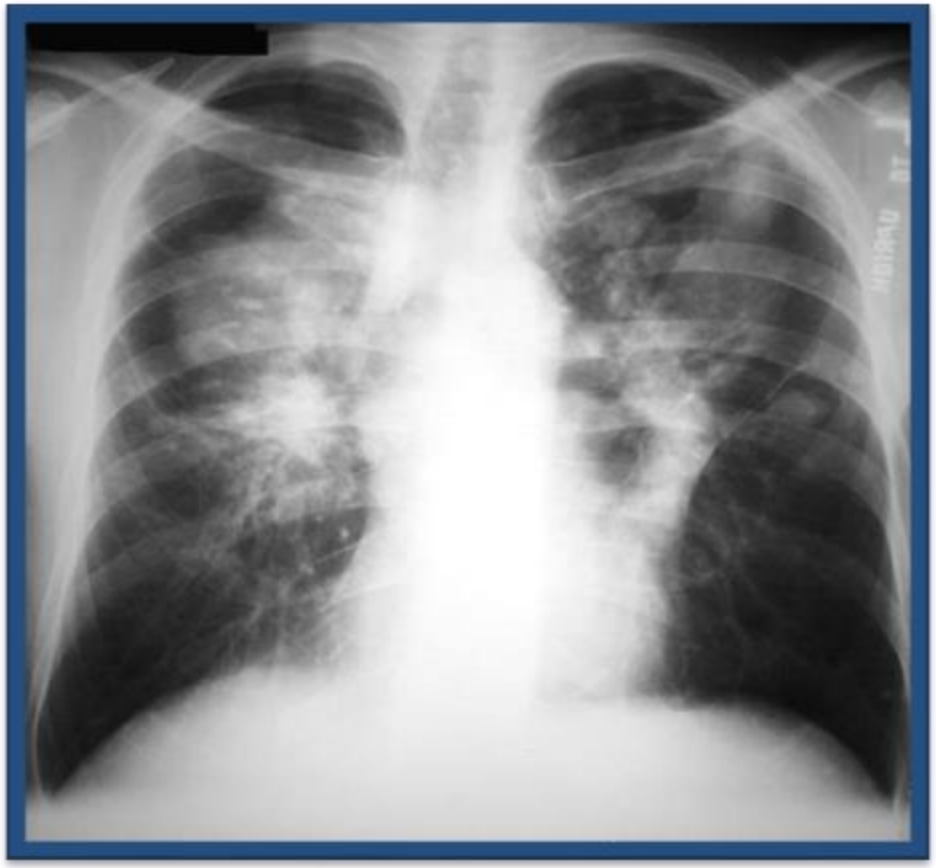
Q: A patient with hypercalcemia.

a) What are the findings in this x-ray?

Bilateral hilar lymphadenopathy

b) What's your diagnosis?

Sarcoidosis



Q: 44 year old male pt present with progressive SOB and chronic cough with history of long standing exposure to coal dust

Mention x ray findings ?

Bilateral opacities with irregular outlines in upper zones

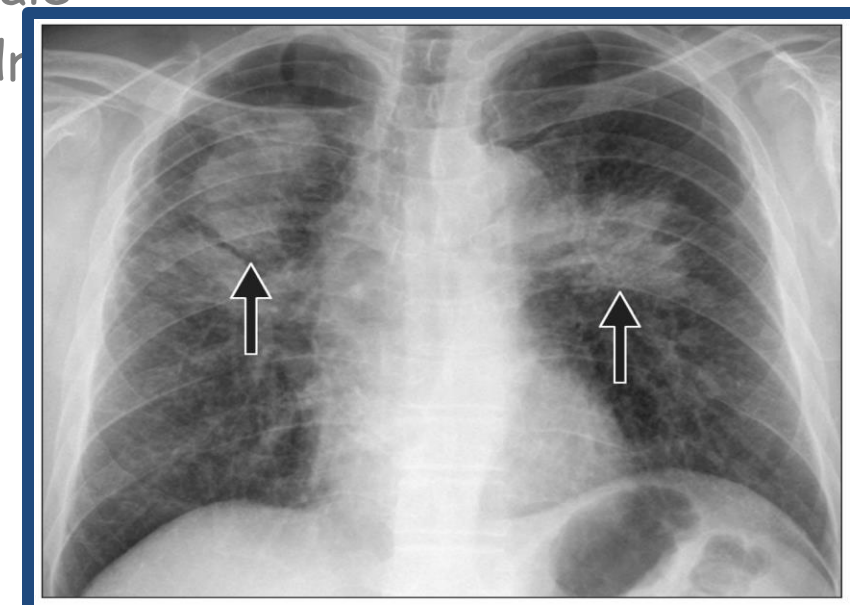
What is your diagnosis?

Coal worker pneumoconiosis

Mention two complications ?

cor pulmonale

Caplan syndr



Q: 40 year old male pt, mining worker came with SOB and cough

Mention x ray findings ?

Egg shell hilar calcification

What is your diagnosis ?

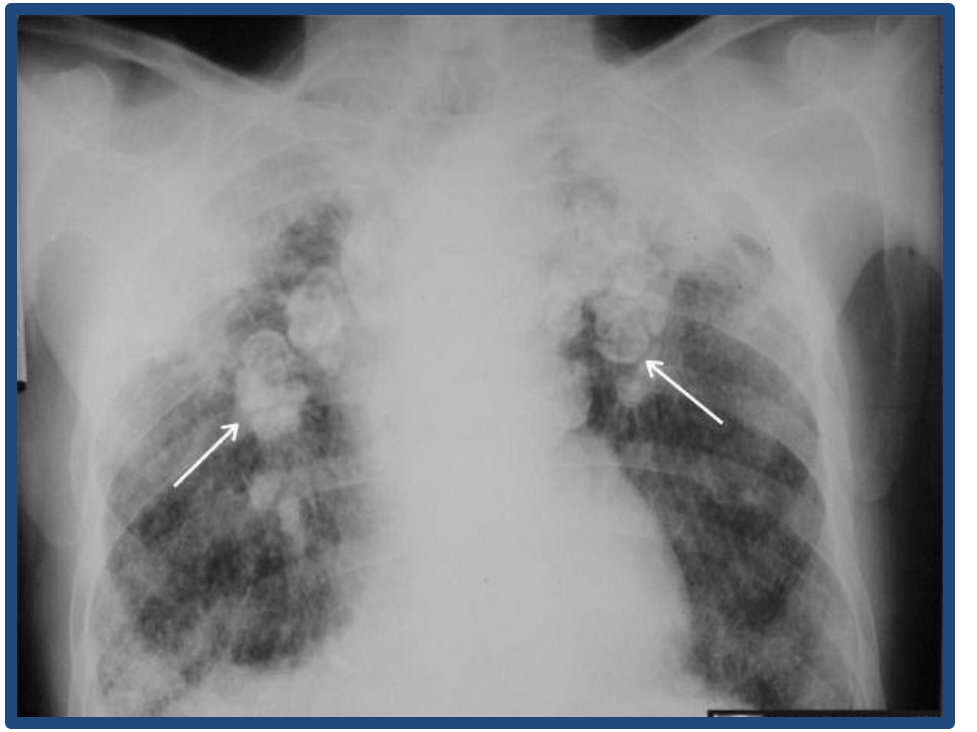
Silicosis

Mention three complications?

TB (may coexist with silicosis)

Chronic bronchitis

Cor pulmonale



Note : asbestosis involves the lower lung , while silicosis involves the upper lung

Pneumonia

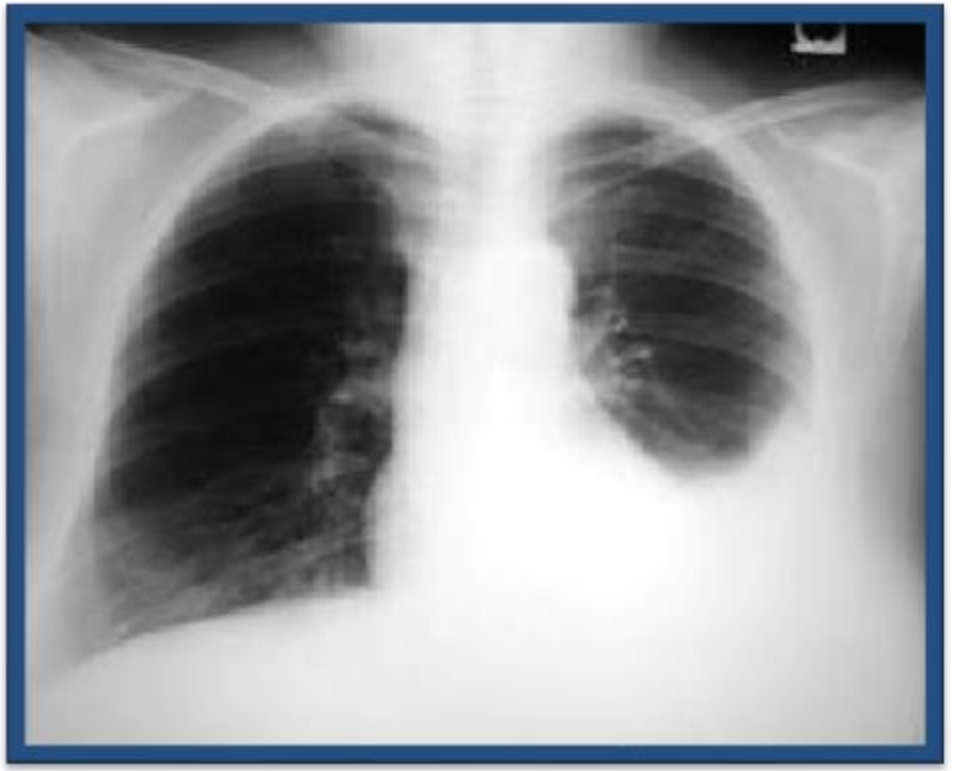
Q: A young patient presented with fever & chest pain.

1. What's the X-ray diagnosis?

Left pleural effusion.

2. What's the underlying cause?

Left lower lobe pneumonia



3.Diagnostic test ?

Diagnostic thoracentesis is the preliminary investigation of choice in the management of pleural effusion,

4. Management of plural effusion:

Bed rest , treatment underlying cause , AB , chest tube

5. Indication for aspiration of fluid:

1- large effusion

2- cardiac or respiratory embarrassment

3-secondary infection of the effusion

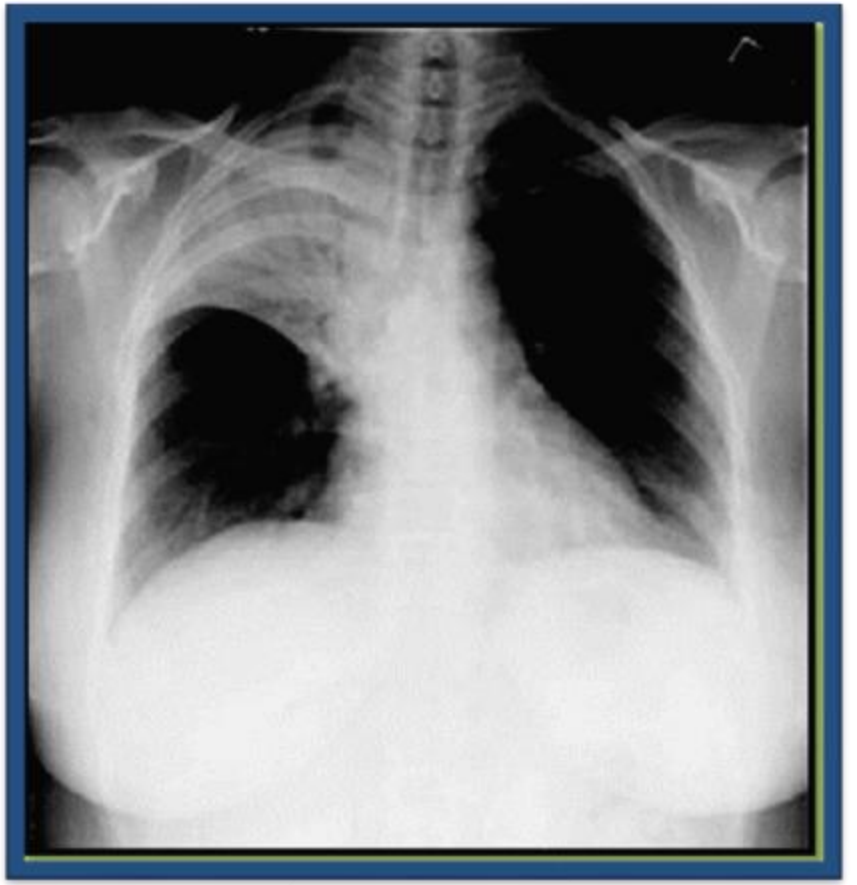
Light criteria for pleural effusions		
	Transudate	**Exudate**
Protein (pleural/serum)	≤0.5	>0.5
LDH (pleural/serum)	≤0.6	>0.6
	Pleural LDH ≤ two-thirds upper limit of normal serum LDH	Pleural LDH > two-thirds upper limit of normal serum LDH
Common causes	• Hypoalbuminemia (cirrhosis, nephrotic syndrome) • Congestive heart failure	• Infection (parapneumonic, TB, fungal, empyema) • Malignancy • PE

LDH = lactate dehydrogenase; **PE** = pulmonary embolism; **TB** = tuberculosis.

Q: Diabetic patient with productive cough of 3 days duration associated with fever & chills. What is the diagnosis?

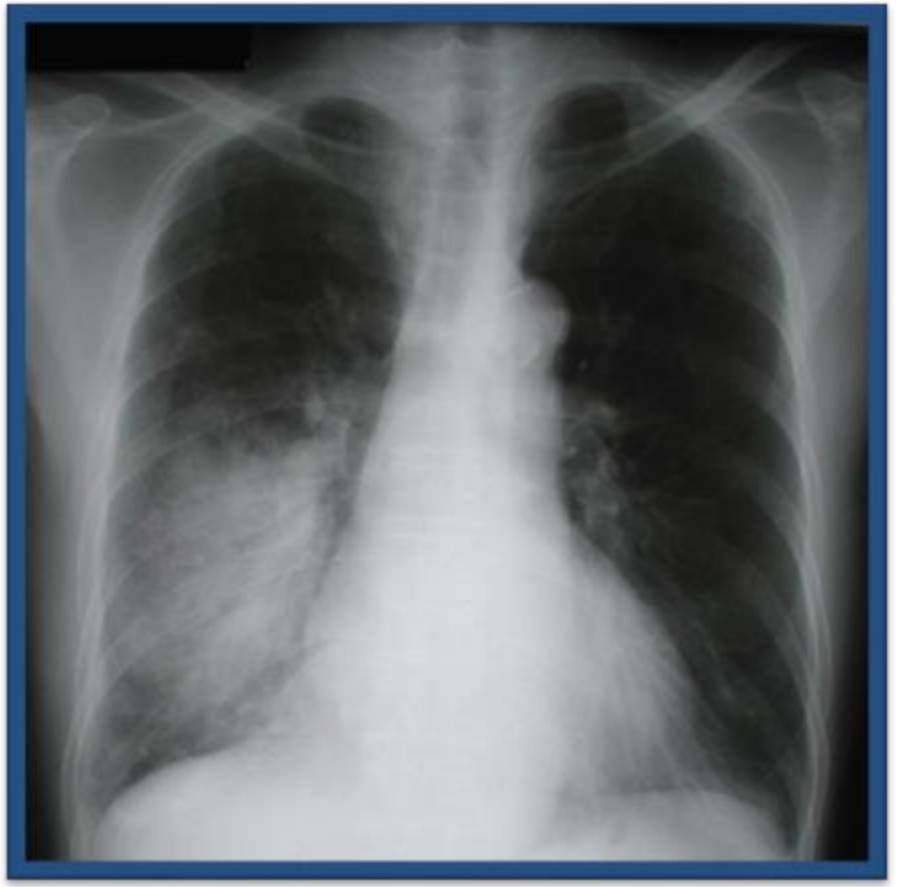
RUL pneumonia

Opcification on the upper right lobe .



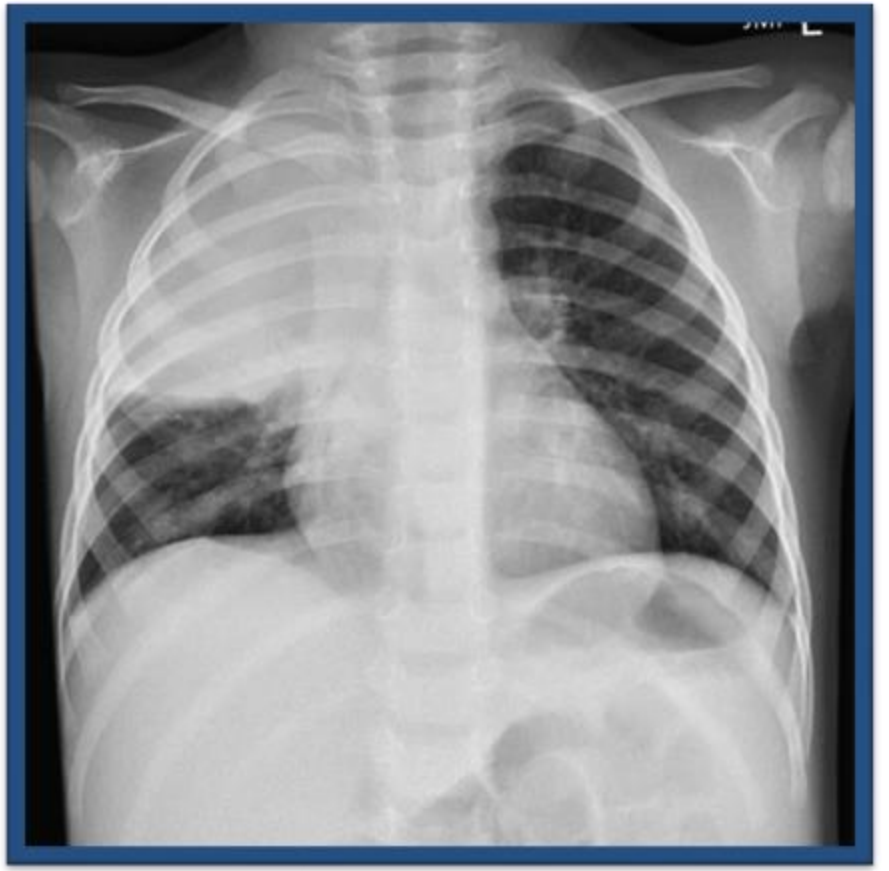
Q: 35 YO male pt, previously healthy presented complaining of cough of greenish sputum & fever, What's the most likely micro-organism?

Strep. Pneumonia



Q: Patient presented with cough, fever and SOB what's your diagnosis?

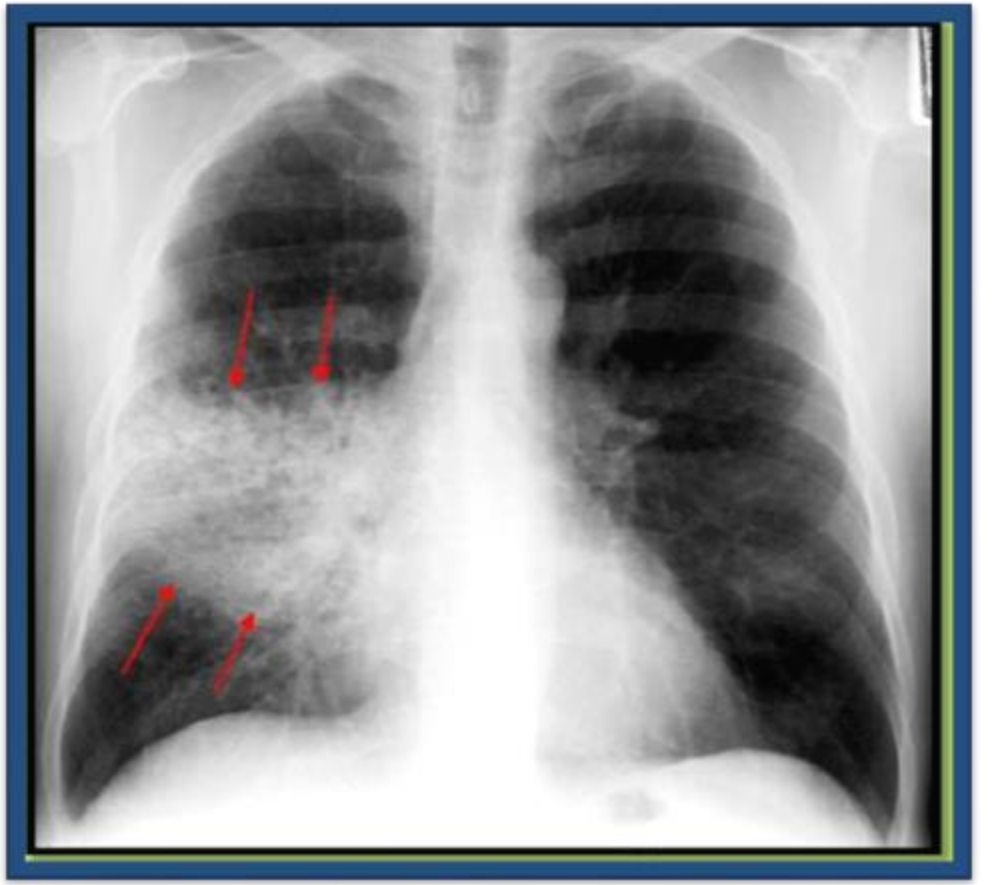
Right upper lobe pneumonia



Q: Mention 2 auscultatory findings in the pts with this X-ray.

A. Crackles, pleural rub.

B. Bronchial breathing



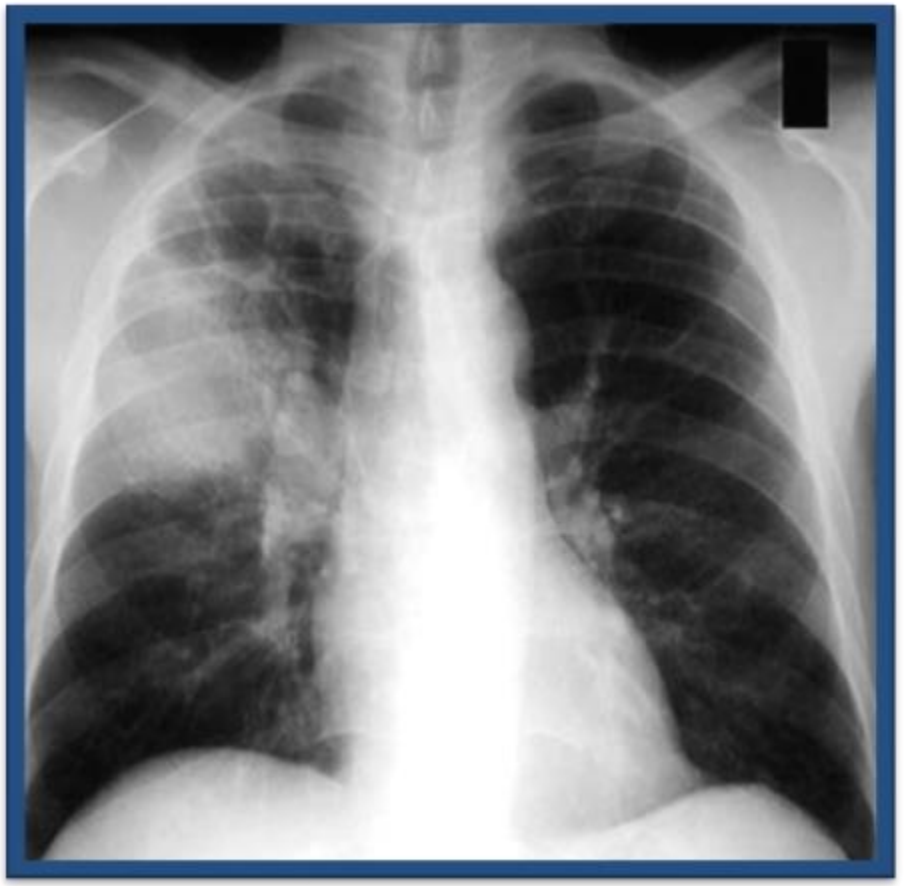
Q: Patient with fever & cough

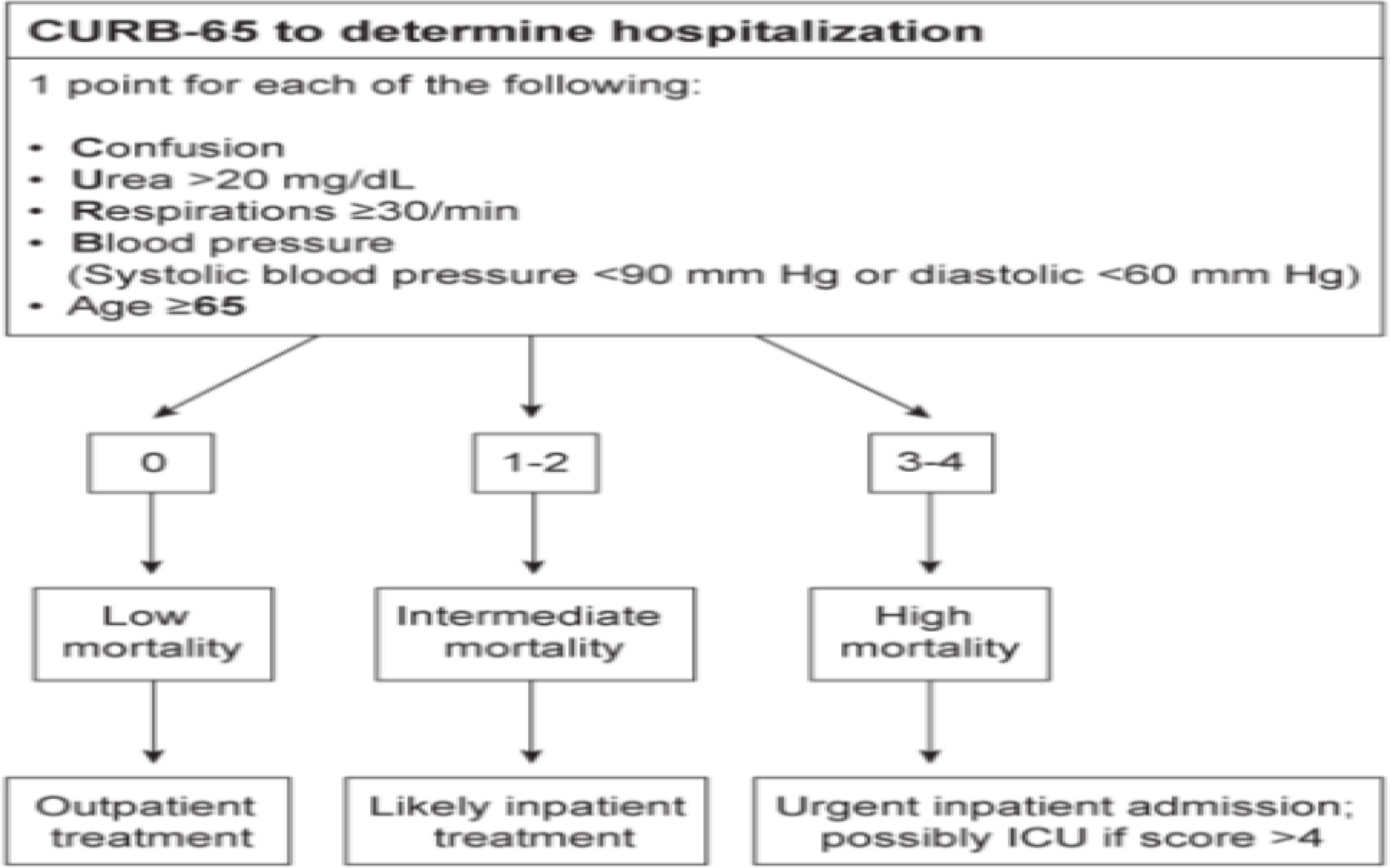
A- what's your diagnosis?

RUL pnemonia

B-What's the most common microorganism.

S.pneumonia





ICU = intensive care unit.

Overview of *Legionella* pneumonia	
Clinical features	• High fever with relative bradycardia • Headache & confusion • Watery diarrhea
Laboratory findings	• Hyponatremia • Sputum Gram stain showing many neutrophils, but few or no organisms
Diagnosis	• *Legionella* urine antigen test
Treatment	• Respiratory fluoroquinolones or newer macrolides

Empiric treatment of CAP	
Outpatient	• Macrolide or doxycycline (healthy) • Fluoroquinolone* or beta-lactam + macrolide (comorbidities)
Inpatient (non-ICU)	• Fluoroquinolone* (IV) • Beta-lactam + macrolide (IV)
Inpatient (ICU)	• Beta-lactam + macrolide (IV) • Beta-lactam + fluoroquinolone* (IV)

*Respiratory fluoroquinolones (eg, levofloxacin, moxifloxacin) are required.
CAP = community-acquired pneumonia; ICU = intensive care unit; IV = intravenous.

- Treatment of Hospital-Acquired Pneumonia:
- Those patients who develop pneumonia after 5-7 days in the hospital are at increased risk of infection from drug-resistant, Gram-negative bacilli (Pseudomonas, Klebsiella, E. coli, etc.) or gram-positive cocci such as methicillin-resistant Staphylococcus aureus (MRSA).
- o Empiric therapy of hospital-acquired pneumonia is with third generation cephalosporins with antipseudomonal activity (such as ceftazidime) or carbapenems (such as imipenem) or with beta-lactam/beta-lactamase inhibitor combinations (such as piperacillin/tazobactam) and coverage for MRSA with vancomycin or linezolid.
- Aminoglycosides (gentamicin, tobramycin, amikacin) are often added to empiric gramnegative coverage for synergy and to ensure that the patient might be getting at least one drug if the bacteria is multidrug resistant.

Infiltrate Patterns and Pathogens

CXR Pattern	Possible Pathogens
Lobar	S.pneumo, Kleb, H. influ, Gram Neg.
Patchy	Atypical, Viral, Legionella
Interstitial	Viral, PCP, Legionella
Cavitatory	Anaerobes, Kleb, TB, S.aureus, Fungi
Large effusion	Staph, Anaerobes, Klebsiella

risk for aspiration : confuse , loss of cons , operated with general anesthesia , epileptic

- Hx:

Fever/chills	**85%**	
Dyspnea	**70%**	
Purulent sputum	**50%**	
Chest pain	**40%**	

- P/E: most useful in predicting severity. Physical exam may reveal fever, tachypnea, tachycardia.

Lung exam; increased tactile fremitus, dullness to percussion, increased breath sounds, presence of crackles

- CXR is gold standard - may be normal in up to 7% on admission; assume pneumonia present if convincing hx and focal P/E

Pneumothorax

Q: A-What is the diagnosis?

Right-sided tension pneumothorax .

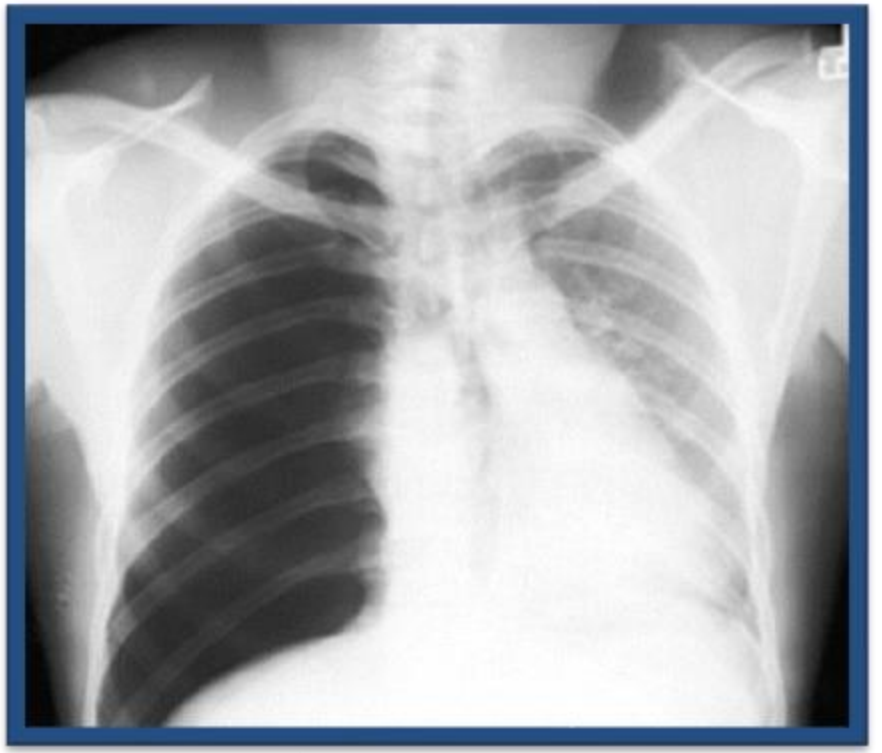
B-How to manage?

Insertion of a chest tube .

C- two findings in x ray ?

1.Viceral pleural line with absent of vascular markings beyound it

2.Mediastinal shift to the opposite side

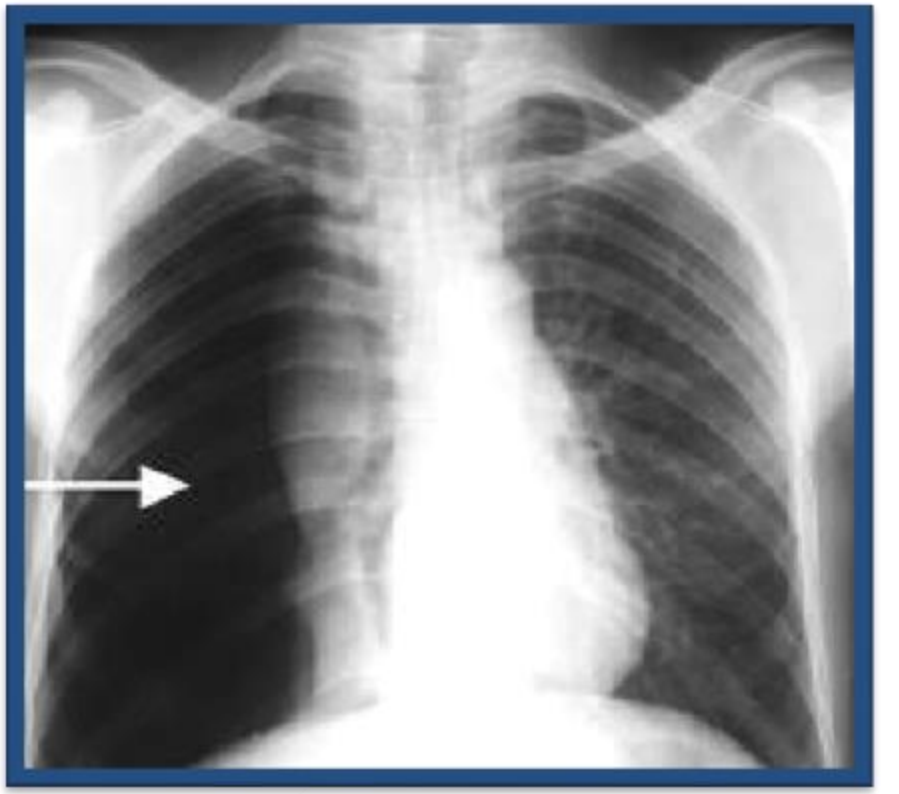


Q: This patient presented with a sudden SOB. What's your diagnosis?

Right sided Pneumothorax

Whats your management ?

Emergent chest tube replacement (if available) or needle thoracostomy

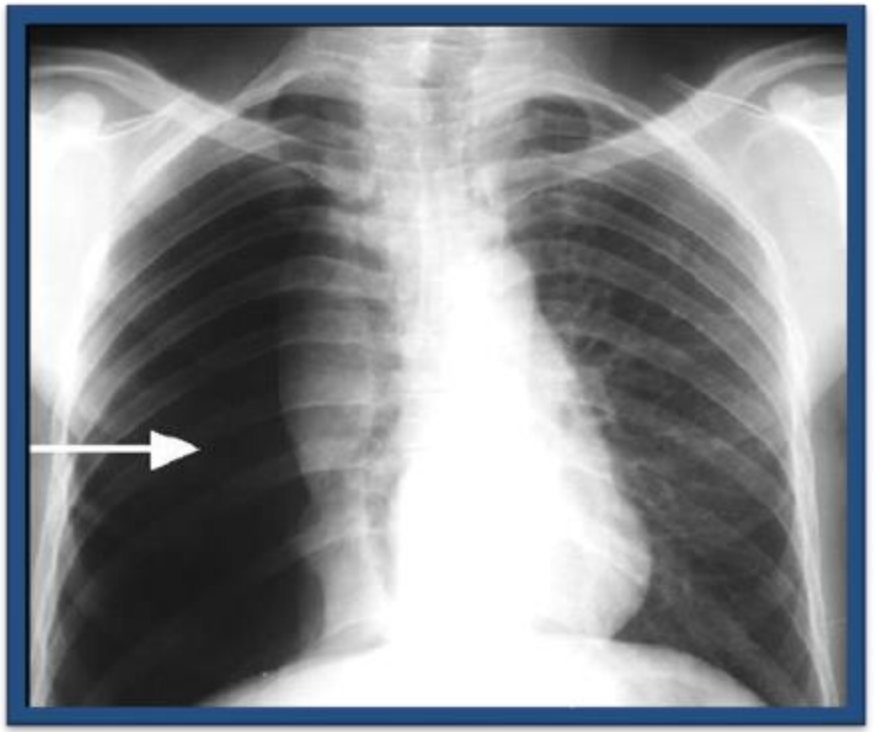


Q: Patient presented with sudden onset chest pain & SOB. What is the 1st step in management?

Chest-tube.

Mention two clinical signs in this pt

1) Severe respiratory distress.
2) Distended nick veins.
3) Absent breathing sounds.
4) Deviated trachea.to rt
5) Deviated apex pulse.

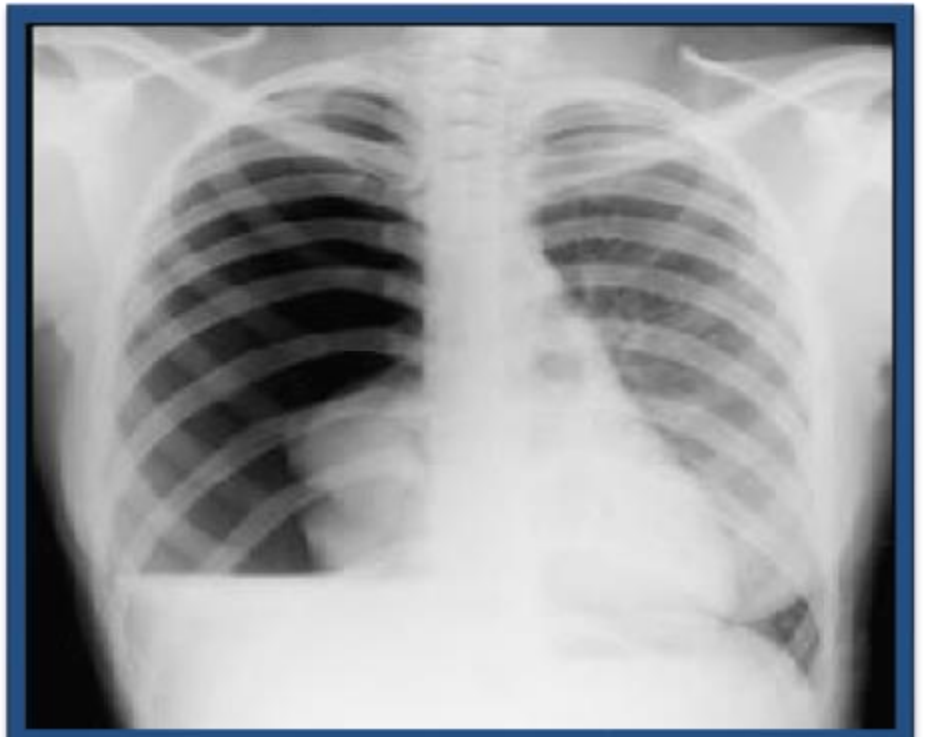


Q: A 42 YO pt is presented with sudden onset breathlessness, SOB. An urgent CXR was done for him & showed the following. What is your spot Dx?

Pnuemothorax.rt sided

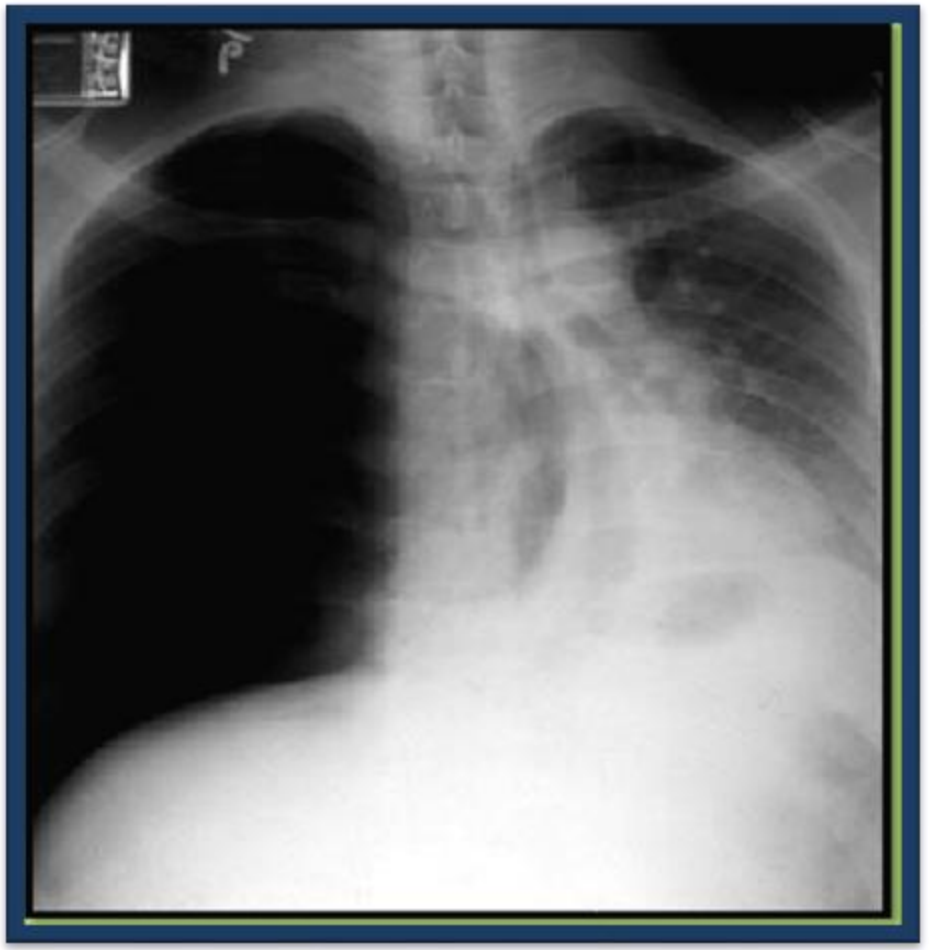
Where to insert the needle ?

Second intercostal space in midclavicular line or if failed , 5th intercostal space in midaxillary line



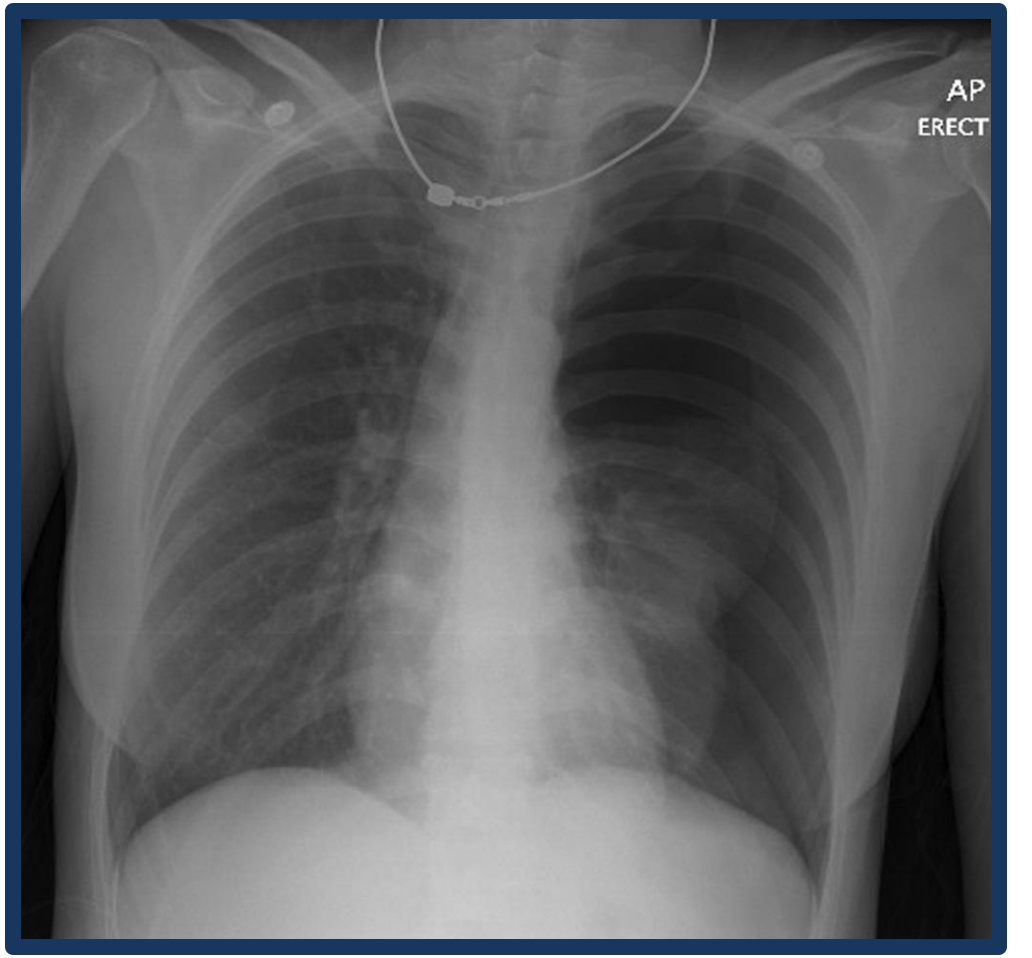
Q: What is the immediate treatment for this patient?

(Chest tube).



Q:What's your Dx.?

Right tension pneumothorax



Pneumothorax		
	Spontaneous pneumothorax	**Tension pneumothorax**
Associated features	• **Primary:** No preceding event or lung disease; thin, young men • **Secondary:** Underlying lung disease (eg, COPD)	• **Life-threatening** • Often due to trauma or mechanical ventilation
Signs & symptoms	• Chest pain, dyspnea • ↓Breath sounds, ↓chest movement • Ipsilateral hyperresonance to percussion	Same as spontaneous with: • **Hemodynamic instability** • Tracheal deviation away from affected side
Imaging	• Absent lung markings • Visceral pleural line	Same as spontaneous with: • Contralateral **mediastinal shift** • Ipsilateral hemidiaphragm flattening
Management	• **Small (≤2 cm):** Observation & oxygen • **Large & stable:** Needle aspiration or chest tube	• Urgent needle decompression or chest tube placement

COPD = chronic obstructive pulmonary disease.

PFT

Abbreviations

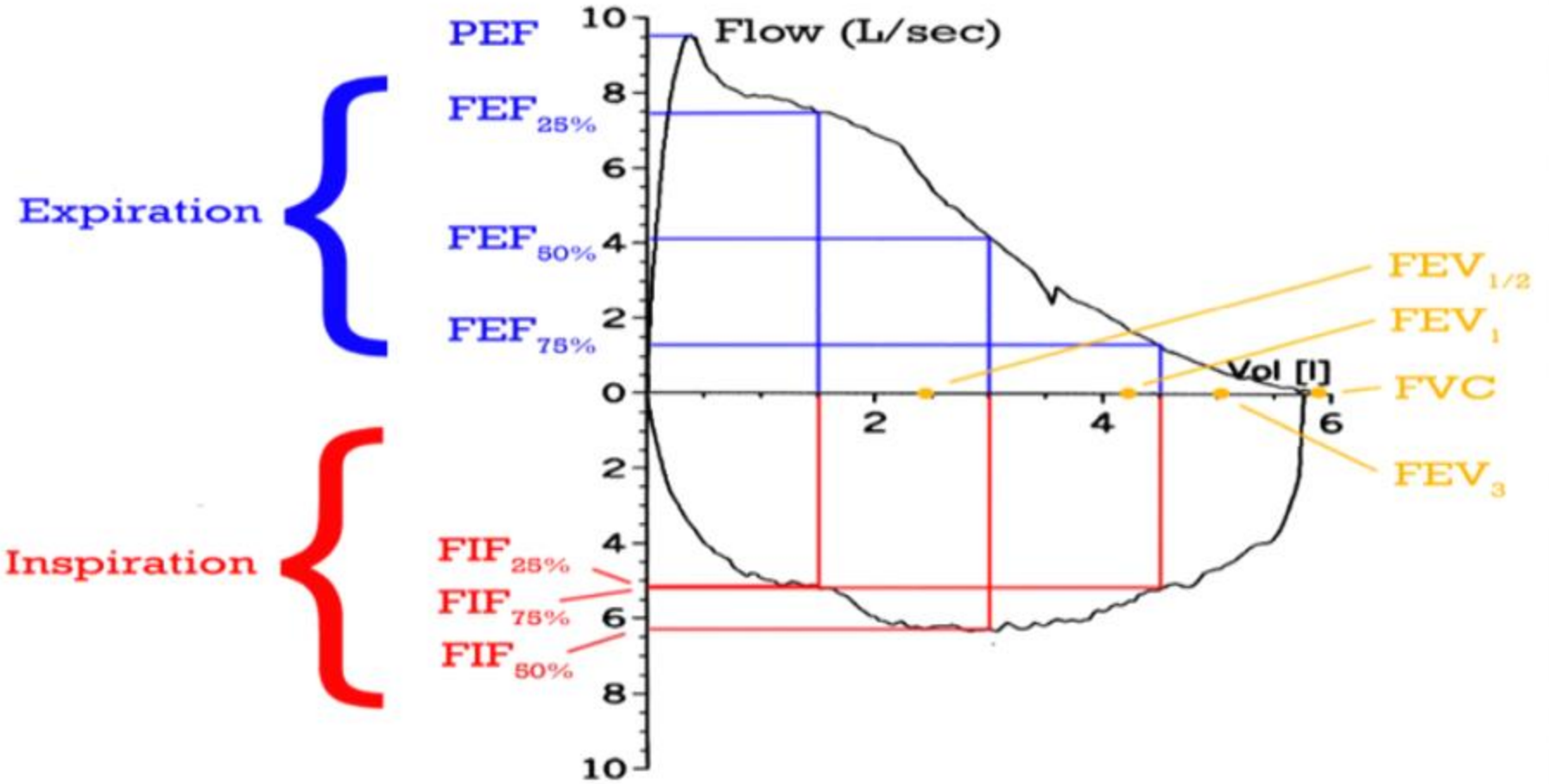
- FVC: Forced Vital Capacity
- FEV1: Forced Expiratory Volume in One Second
- TLC: Total Lung Capacity
- RV: Residual Volume
- DLCO: Diffusion Capacity for Carbon Monoxide
- BD: Bronchodilator

Severity of airflow limitation

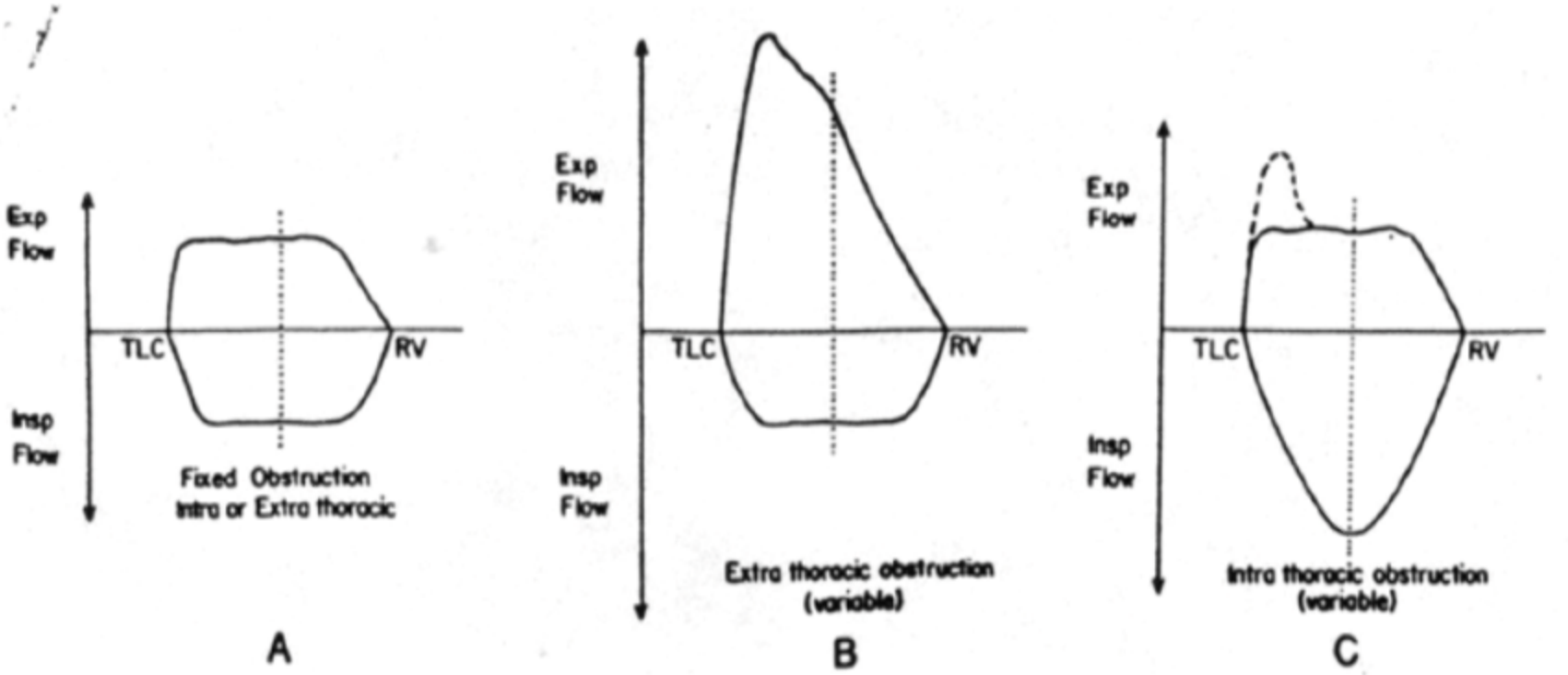
Category/Severity Stage	FEV_1/FEV	FEV_1 (% Predicted)
Normal (healthy patients)	0.80	~100
I: Mild	<0.70	≥80
II: Moderate	<0.70	50 to <80
III: Severe	<0.70	30 to <50
IV: Very Severe	<0.70	<30[a]

- FEV1/FVC ratio
- Reversibility : FEV1 > 200ml, > 12%
- TLC, RV
- FEV1 & FVC > 20% (supine & upright): diaphragmatic weakness
- Air-trapping RV
- Hyper-inflated TLC > 120
- Restrictive TLC < 80%

Flow Volume Loop

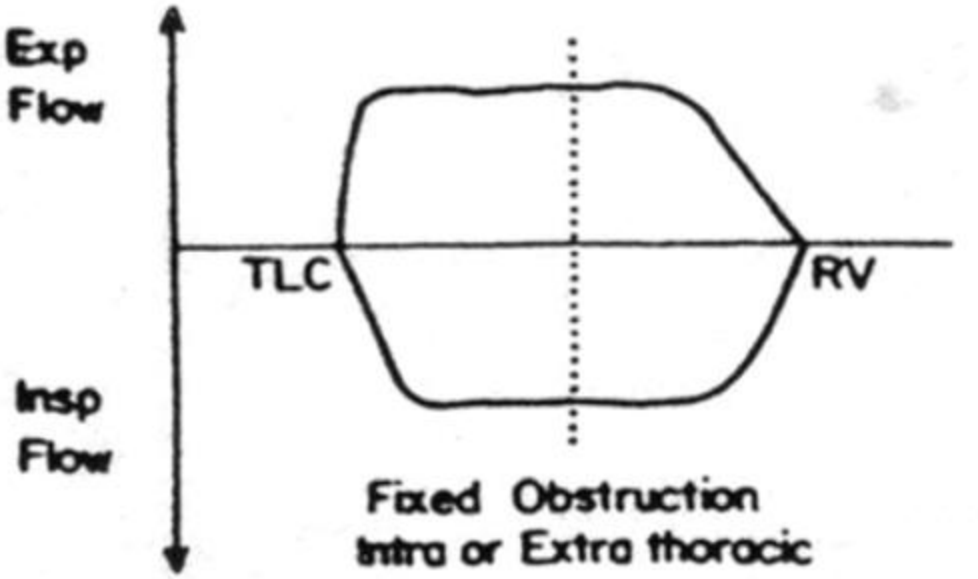


Pattern of airway obstruction



A 25 y/o man presents to his physician with complaints of dyspnea and wheezing.
He had a tracheostomy because he remained on ventilator for a total of 7 weeks after motor vehicle accident, His tracheostomy was removed 2 months after his discharge from the hospital.
flow volume loop was done as shown

- What is the most likely Diagnosis?

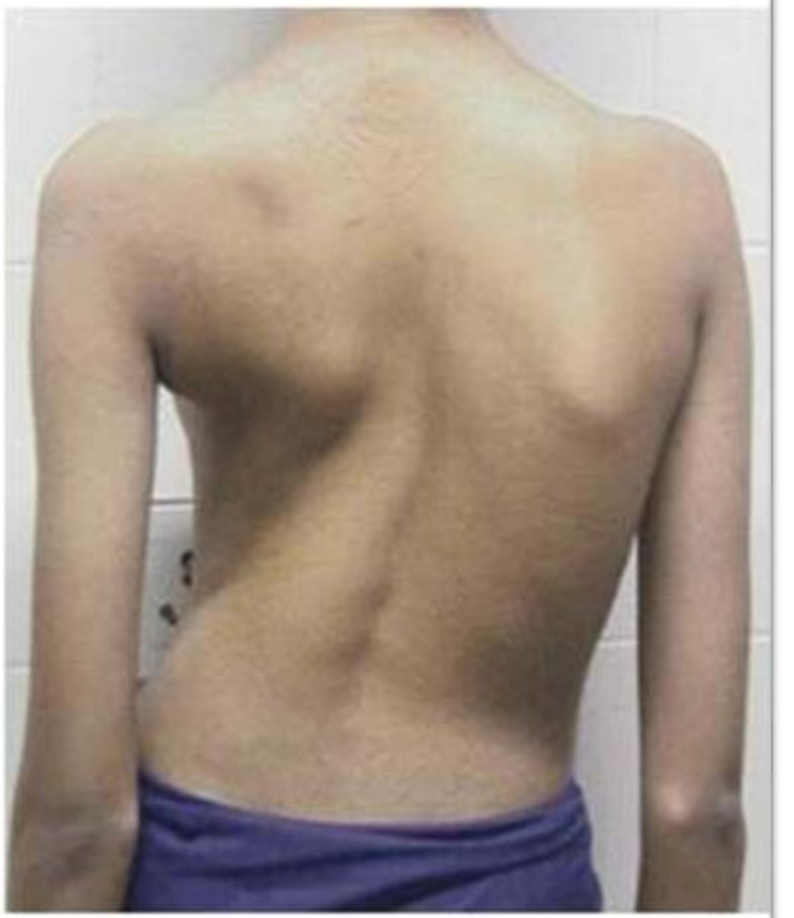


if we ask for a pulmonary function test for this patient, what are the changes that you expect to find in the:

1- TLC: decreased

2- FEV1/FVC: increased

3- DLCO: normal



A 36 year-old woman presents with a several month history of worsening dyspnea on exertion and exercise limitation, non smoker, no past history of pulmonary disease, Her pulmonary function testing is as follows:

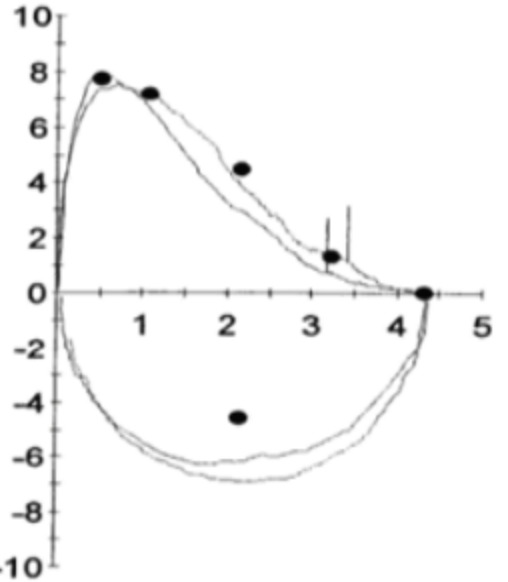
Extra thoracic restrictive disease

- What is the cause of her dyspnea ?

	Pre-Bronchodilator (BD)		
Test	Actual	Predicted	% Predicted
FVC (L)	0.88	3.34	26
FEV_1 (L)	0.87	2.87	30
FEV_1/FVC (%)	99	86	
RV (L)	1.61	1.40	115
TLC (L)	2.49	4.73	53
RV/TLC (%)	65	29	
DLCO corr	26.14	31.28	84

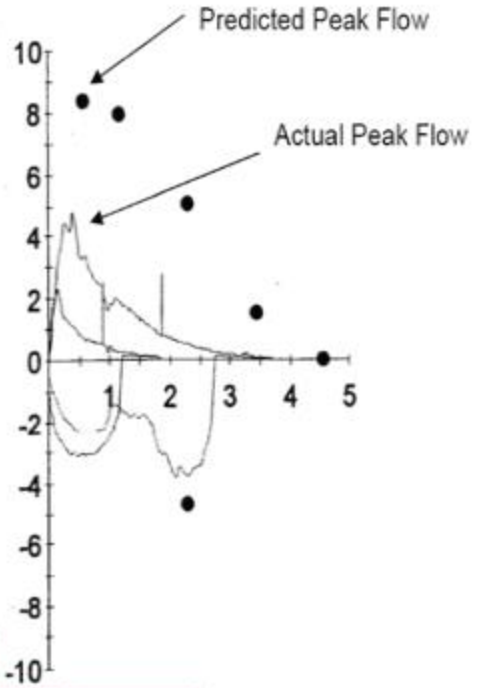
Questions

1. A 65 year-old man undergoes pulmonary function testing as part of a routine health-screening test. He had no pulmonary complaints. He is a lifelong nonsmoker and had a prior history of asbestos exposure while serving in the Navy. His pulmonary function test results are as follows:



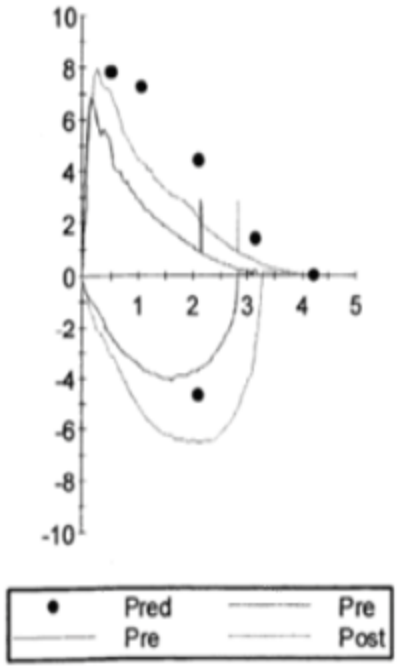
	Pre-Bronchodilator (BD)			Post- BD
Test	Actual	Predicted	% Predicted	% Change
FVC (L)	4.39	4.32	102	-1
FEV_1 (L)	3.20	3.37	95	7
FEV_1/FVC (%)	73	78		8
FRC (L)	3.17	3.25	98	
ERV (L)	0.63	0.93	68	
RV (L)	2.54	2.32	109	
TLC (L)	6.86	6.09	113	
DLCO uncorr	25.69	31.28	82	
DLCO corr	26.14	31.28	84	

2. A 54 year-old man presents to his primary care provider with dyspnea and a cough. He is a non-smoker with no relevant occupational exposures.



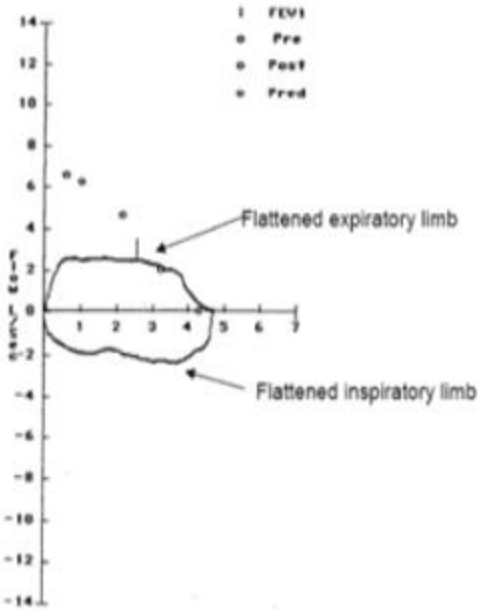
	Pre-Bronchodilator (BD)			Post- BD	
Test	Actual	Predicted	% Predicted	Actual	% Change
FVC (L)	3.19	4.22	76	4.00	25
FEV_1 (L)	2.18	3.39	64	2.83	30
FEV_1/FVC (%)	68	80		71	4

3. A 60 year-old man presents to his primary care provider with complaints of increasing dyspnea on exertion. He has a 40 pack-year history of smoking and is retired following a career as a building contractor. His pulmonary function testing is as follows:



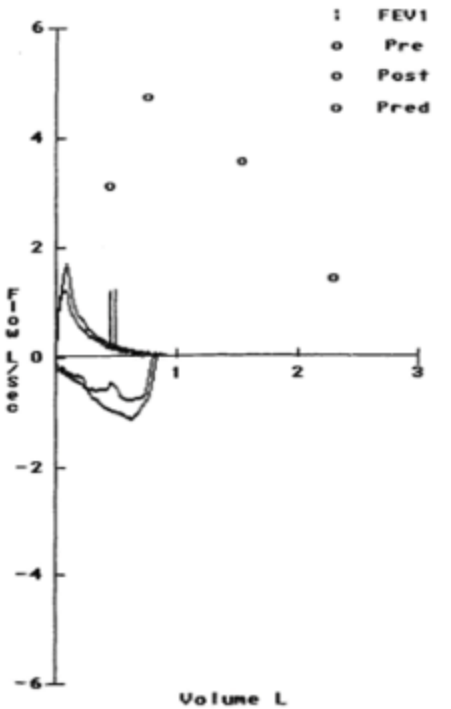
	Pre-Bronchodilator (BD)			Post- BD	
Test	Actual	Predicted	% Predicted	Actual	% Change
FVC (L)	1.89	4.58	41	3.69	96
FEV_1 (L)	0.89	3.60	25	1.89	112
FEV_1/FVC (%)	47	79			
RV (L)	5.72	2.31	248		
TLC (L)	7.51	6.41	117		
RV/TLC (%)	76	37			
DLCO corr	20.73	33.43	62		

4. A 25 year-old man presents to his physician with complaints of dyspnea and wheezing. He is a non-smoker. Two years ago, he was in a major motor vehicle accident and was hospitalized for 3 months. He had a tracheostomy placed because he remained on the ventilator for a total of 7 weeks. His tracheostomy was removed 2 months after his discharge from the hospital. His pulmonary tests are as follows:



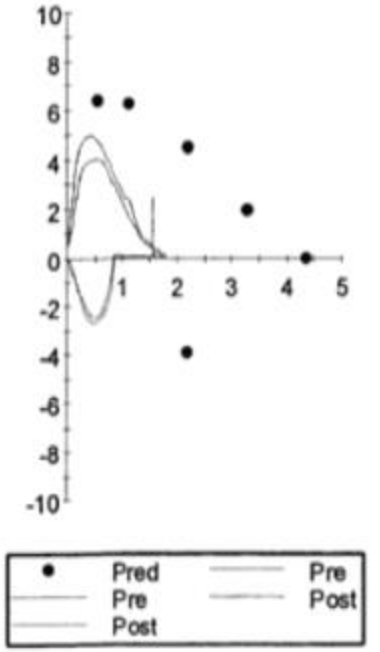
	Pre-Bronchodilator (BD)		
Test	Actual	Predicted	% Predicted
FVC (L)	4.73	4.35	109
FEV_1 (L)	2.56	3.69	69
FEV_1/FVC (%)	54	85	

5. A 41 year-old woman presents to the General Internal Medicine Clinic at Harborview Medical Center complaining of dyspnea with mild exertion. She has a 10 pack-year history of smoking and a history of using intravenous drugs including heroin. Her pulmonary function tests are as follows:



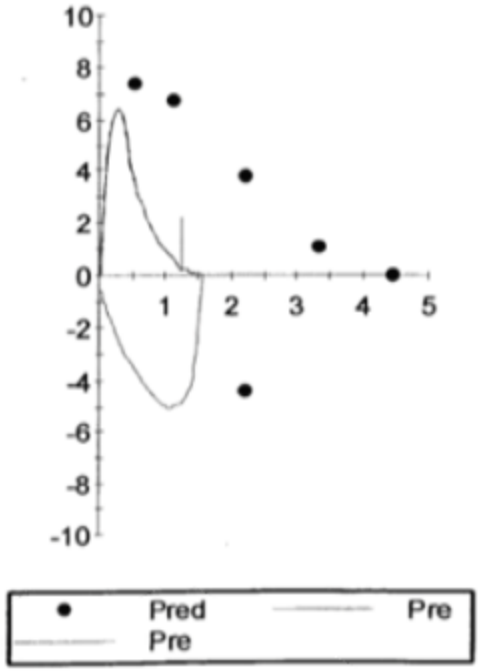
	Pre-Bronchodilator (BD)			Post- BD	
Test	Actual	Predicted	% Predicted	Actual	% Change
FVC (L)	0.90	3.09	29	0.74	- 17
FEV_1 (L)	0.49	2.57	19	0.44	-10
FEV_1/FVC (%)	54	83		59	8
RV (L)	3.83	1.49	257		
TLC (L)	4.78	4.44	108		
RV/TLC (%)	80	33			
DLCO corr	0.75	24.85	3		

6. A 30 year-old woman presents for evaluation of dyspnea on exertion, which has been present for 2 months. She is a life-long non-smoker with no prior history of asthma or other pulmonary problems. She works as a receptionist at a publishing company. She has two cats and several parakeets at home. Her pulmonary function testing is as follows:



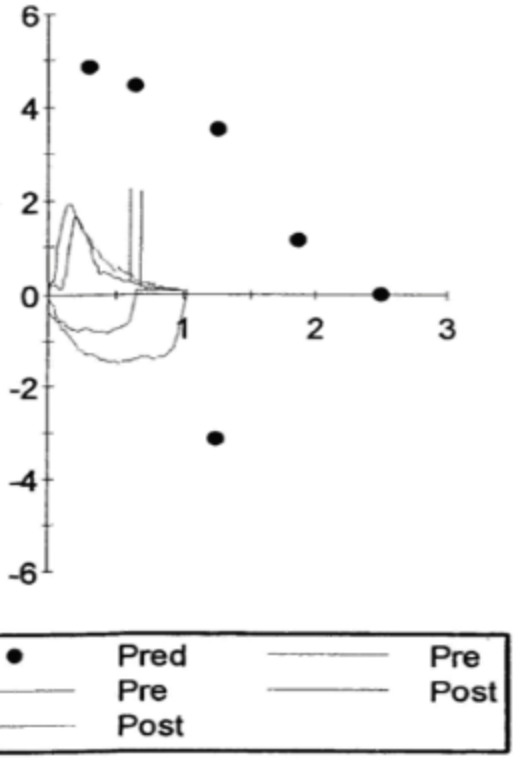
	Pre-Bronchodilator (BD)			Post- BD	
Test	Actual	Predicted	% Predicted	Actual	% Change
FVC (L)	1.73	4.37	40	1.79	4
FEV_1 (L)	1.57	3.65	43	1.58	0
FEV_1/FVC (%)	91	84		88	-3
RV (L)	1.01	1.98	51		
TLC (L)	2.68	6.12	44		
RV/TLC (%)	38	30			
DLCO corr	5.13	32.19	16		

7. A 73 year-old man presents with progressive dyspnea on exertion over the past one year. He reports a dry cough but no wheezes, sputum production, fevers or hemoptysis. He is a life-long non-smoker and worked as a lawyer until retiring 3 years ago. He likes to hunt and fish in his leisure time. His pulmonary function testing is as follows:



	Pre-Bronchodilator (BD)		
Test	Actual	Predicted	% Predicted
FVC (L)	1.57	4.46	35
FEV_1 (L)	1.28	3.39	38
FEV_1/FVC (%)	82	76	
FRC	1.73	3.80	45
RV (L)	1.12	2.59	43
TLC (L)	2.70	6.45	42
RV/TLC (%)	41	42	
DLCO corr	5.06	31.64	16

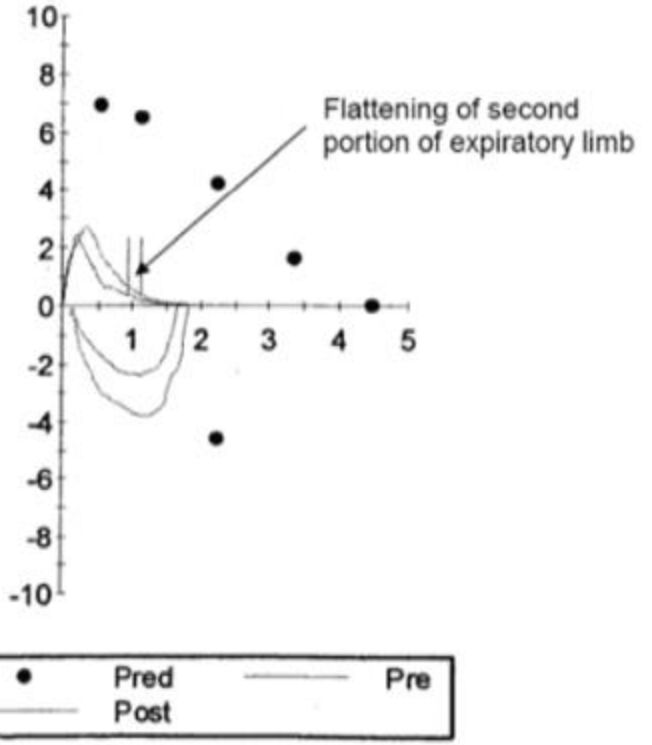
8. A 64 year-old woman presents with complaints of dyspnea and orthopnea. She is a life-long non-smoker. Her pulmonary function testing is as follows:



	Pre-Bronchodilator (BD)			Post- BD	
Test	Actual	Predicted	% Predicted	Actual	% Change
FVC (L)	1.00	2.51	40	1.02	3
FEV_1 (L)	0.61	2.00	30	0.69	13
FEV_1/FVC (%)	61	80		67	10
RV (L)	1.15	1.55	74		
TLC (L)	2.08	4.04	52		
RV/TLC (%)	55	39			

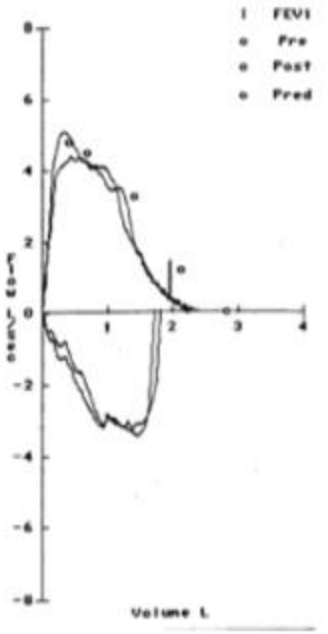
Test	Upright	Supine
FVC (L)	0.49	0.37
FEV_1 (L)	0.82	0.68
FEV_1/FVC (%)	0.60	0.54

9. A 35 year-old previously healthy man presents with dyspnea, fevers, chills and night sweats for the past 2 months. He is a non-smoker with no concerning habits or occupational exposures. His pulmonary function tests are as follows:



	Pre-Bronchodilator (BD)		
Test	**Actual**	**Predicted**	**% Predicted**
FVC (L)	1.66	4.48	37
FEV_1 (L)	0.94	3.67	26
FEV_1/FVC (%)	57	82	
RV (L)	1.39	1.66	84
TLC (L)	3.06	5.96	51
RV/TLC (%)	45	29	

10. A 53 year-old woman presents with increasing dyspnea on exertion. She denies cough, fevers, hemoptysis, weight loss or sweats. She was previously an active runner but has had to cut back significantly because of her symptoms with exercise. She does note occasional chest pain with exercise but has not had any syncope or palpitations. Her pulmonary function tests are as follows:



	Pre-Bronchodilator (BD)			Post- BD	
Test	Actual	Predicted	% Predicted	Actual	% Change
FVC (L)	2.38	2.87	83	2.23	-6
FEV_1 (L)	1.95	2.31	84	1.93	-1
FEV_1/FVC (%)	82	81		87	
RV (L)	1.69	1.58	107		
TLC (L)	4.26	4.36	98		
RV/TLC (%)	40	36			
DLCO corr	9.96	23.25	43		

11. A 36 year-old woman presents with a several month history of worsening dyspnea on exertion and exercise limitation. She is a life-long non-smoker and has no history of asthma or other known pulmonary diseases. She has had to stop going out with her weekly running group because she can no longer keep up with her friends. Her pulmonary function testing is as follows:

	Pre-Bronchodilator (BD)		
Test	**Actual**	**Predicted**	**% Predicted**
FVC (L)	0.88	3.34	26
FEV_1 (L)	0.87	2.87	30
FEV_1/FVC (%)	99	86	
RV (L)	1.61	1.40	115
TLC (L)	2.49	4.73	53
RV/TLC (%)	65	29	
DLCO corr	21	26.6	78

12. A 44 year-old woman with cirrhosis secondary to chronic alcohol abuse and Hepatitis C presents with complaints of increasing dyspnea. She reports that her dyspnea is worse when she is sitting upright or walking but improves when she is lying flat. She is an active cigarette smoker. On exam, she has a room air oxygen saturation of 88% in the sitting position and a room air oxygen saturation of 96% in the supine position. Her pulmonary function testing is as follows.

	Pre-Bronchodilator (BD)			Post- BD	
Test	Actual	Predicted	% Predicted	Actual	% Change
FVC (L)	3.94	3.69	107%	3.86	-2
FEV_1 (L)	2.76	3.03	91%	2.85	3
FEV_1/FVC (%)	70	82			
RV (L)	1.89	1.86	102		
TLC (L)	5.67	5.40	105		
RV/TLC (%)	33	33			
DLCO corr	10.22	28.22	36		

Answers

1. Normal
2. Moderate airflow limitation with reversibility
3. Severe Airflow limitation with reversibility, with air trapping (RV high)
4. Moderate airflow limitation, flattening of both inspiratory & expiratory arm, fixed upper airway obstruction (tracheal stenosis)
5. Severe airflow limitation, no reversibility, air-trapped, not hyper inflated, decrease diffusion, low PEF (alpha 1 AT deficiency)
6. Severe Restrictive pattern, with decrease diffusion (intrathoracic)
7. Severe Restrictive airway
8. Obstructive & Restrictive , Diaphragmatic weakness.
9. Obstructive (severe) & restrictive (moderate), flat 2nd part of expiratory arm: unequal emptying of both lung (mass cause obstructive & restrictive)
10. No obstructive nor restrictive airway, but isolated decreased diffusion, most likely vascular element (pulmonary hypertension)
11. No obstructive, restrictive, high RV, extra- thoracic restriction (neuromuscular)
12. No obstruction, no restriction, isolated decreased diffusion, with platypnea, orthodeoxia (intrpulmonary shunt), hepato-pulmonary

Q: What's the Dx. depending on this pulmonary function test?

Obstructive Lung Disease (Asthma).

Age: 59	Height (cm): 172		Weight (kg): 92.0		BMI: 31.10	Gender: male
	Ref	**Pre Meas**	**Pre %Ref**	**Post Meas**	**Post % Chg**	**CI**	**LLN**
FEV_1 (L)	3.11	**2.00	**64	2.85	42	1.00	
FVC (L)	4.35	3.40	78	4.10	21	1.36	
FEV_1/FVC %	72	59		69			
PEF (L/sec)	8.17	4.45	54	6.81	53	3.87	
FEF25-75 (L/sec)	4.06	**1.23	**30	2.24	82	2.67	
FET100% (sec)		7.46		10.62	42		
FEV_6	4.22	3.40	81	3.97	17		3.34
FEV_1/FEV_6	79	59		72			70

Q: What is the most likely dx?

Most likely obstructive lung disease.

Gender: Male
Age: 49 Race: Caucasian
Height(in): 70 Weight(lb): 211
Any Info:

Date: 03/21/07
Temp: 20 PBar: 712
Physician: D.Musa Malkawi
Technician: R.T RAED BASHTAW

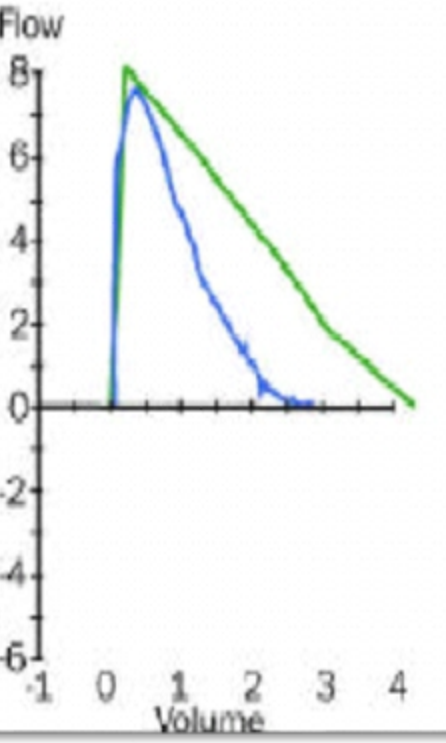
Spirometry	(BTPS)		PRE-RX		POST-RX		
		PRED	BEST	%PRED	BEST	%PRED	% Chg
FVC	Liters	4.57	4.52	99	4.59	100	2
FEV1	Liters	3.70	2.34	63	2.75	74	17
FEV1/FVC	%	78	52		60		
FEF25-75%	L/sec	4.03	1.07	27	1.56	39	46
FEF50%	L/sec	4.84	1.34	28	1.84	38	37
PEF	L/sec	8.93	4.61	52	5.92	66	28
MVV	L/min						

Q: Patient with this Spirometry result, what is his ventilatory defect ?

Restrictive lung disease (suggesting lung fibrosis).

Age: 49 Height (cm): 167 Weight (kg): 146.5 BMI: 52.53 Gender: male

	Ref	Pre Meas	Pre %Ref	Post Meas	Post % Chg	CI	LLN
FEV_1 (L)	3.24	2.27	70			1.00	
FVC (L)	4.30	**2.85	**66			1.36	
FEV_1/FVC %	75	80					
PEF (L/sec)	8.05	7.59	94			3.87	
FEF25-75 (L/sec)	4.09	2.72	67			2.67	
FET100% (sec)		14.86					
FEV_6	4.23	2.69	64				3.43
FEV_1/FEV_6	80	84					72

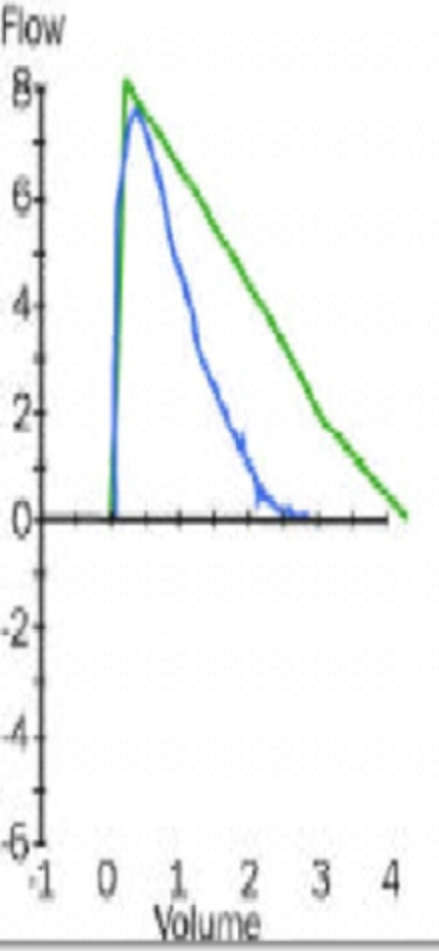


Q: Give 2 causes for this pattern.

Sarcoidosis, IPF.

Age: 49 Height (cm): 167 Weight (kg): 146.5 BMI: 52.53 Gender: male

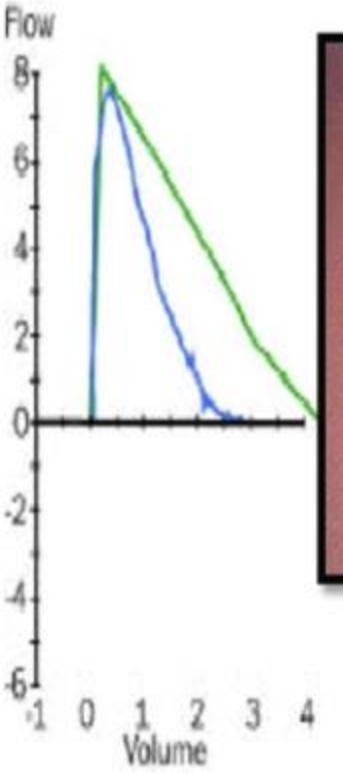
	Ref	Pre Meas	Pre %Ref	Post Meas	Post % Chg	CI	LLN
FEV_1 (L)	3.24	2.27	70			1.00	
FVC (L)	4.30	**2.85	**66			1.36	
FEV_1/FVC %	75	80					
PEF (L/sec)	8.05	7.59	94			3.87	
FEF25-75 (L/sec)	4.09	2.72	67			2.67	
FET100% (sec)		14.86					
FEV_6	4.23	2.69	64				3.43
FEV_1/FEV_6	80	84					72

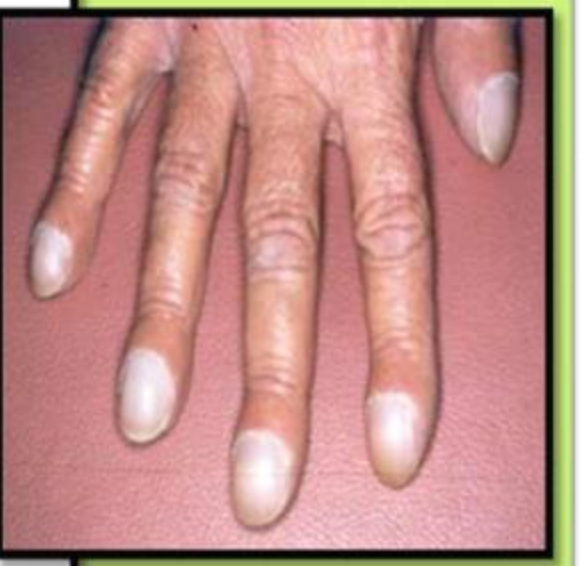


Q: what is the most likely Dx?

Age: 49 Height (cm): 167 Weight (kg): 146.5 BMI: 52.53 Gender: male

	Ref	Pre Meas	Pre %Ref	Post Meas	Post % Chg	CI	LLN
FEV_1 (L)	3.24	2.27	70			1.00	
FVC (L)	4.30	*2.85	*66			1.36	
FEV_1/FVC %	75	80					
PEF (L/sec)	8.05	7.59	94			3.87	
FEF25-75 (L/sec)	4.09	2.72	67			2.67	
FET100% (sec)		14.86					
FEV_6	4.23	2.69	64				3.43
FEV_1/FEV_6	80	84					72





Tuberculosis

Q:This pt presented with cough for 8 weeks, fever, Hemoptysis, wt loss, night sweats & anorexia.4.

What is the finding in this CXR?

Right upper lobe consolidation.

What is your Dx.?

Tubercolosis.

Investigation ?

-CXR

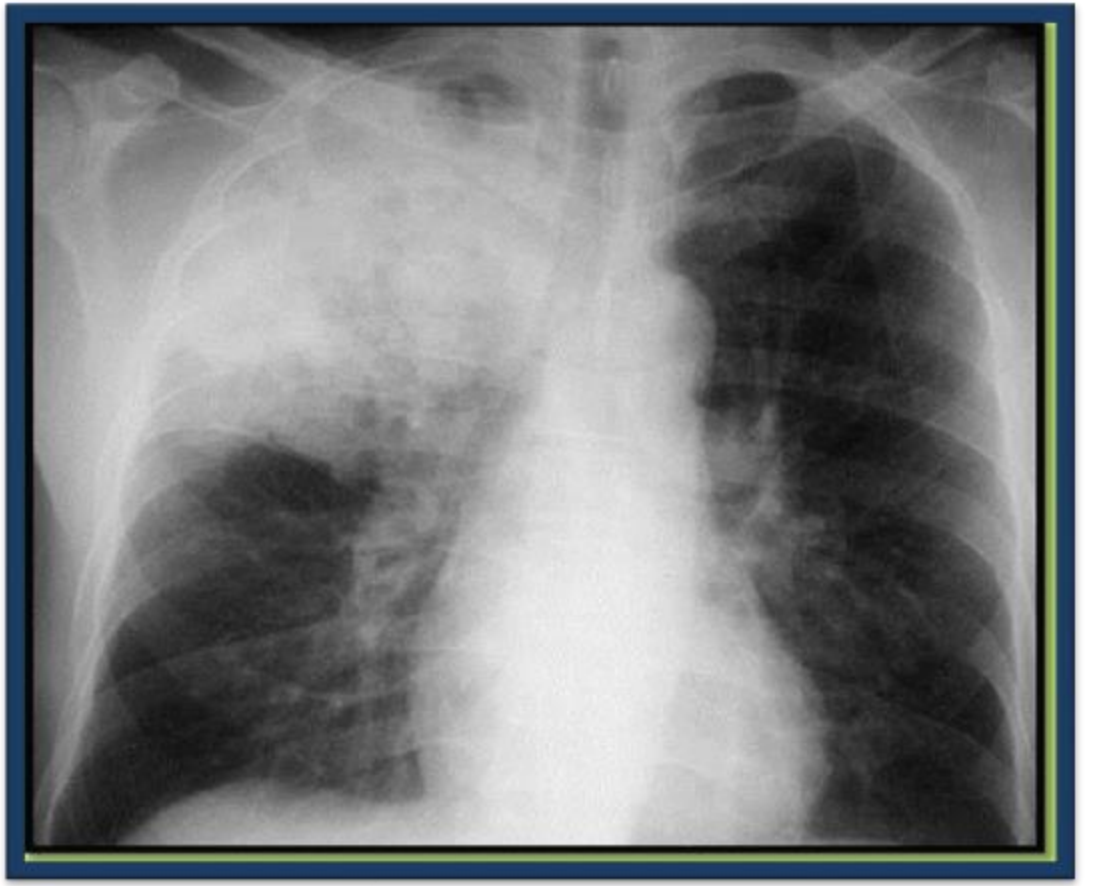
-sputum acid fast testing

-tuberculin skin test

Treatment ?

First line therapy is a four drug regimen:

isoniazid , rifampin, pyrazinamide, ethambutol or streptomycin <<< for 2 months , then for 4 months << use : INH and rifampin.



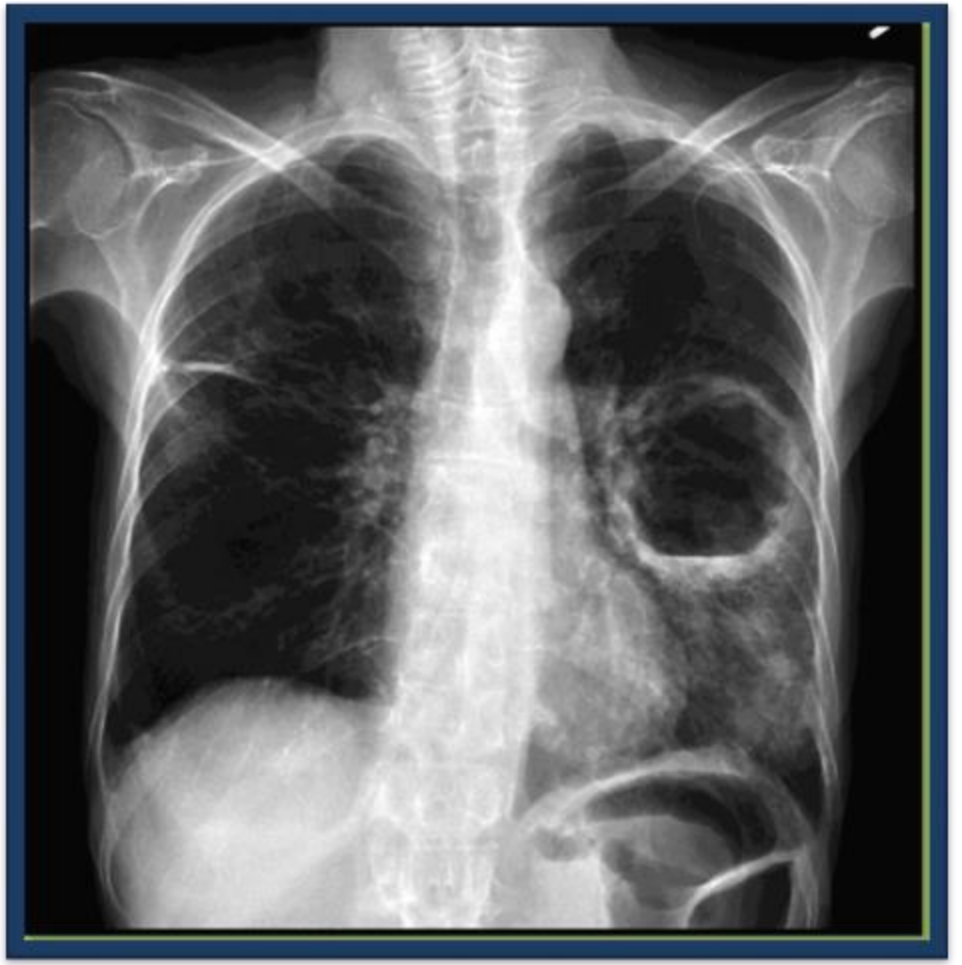
Q: What is the finding in this CXR?

Cyst with fluid level in the Lt.

Lower zone;

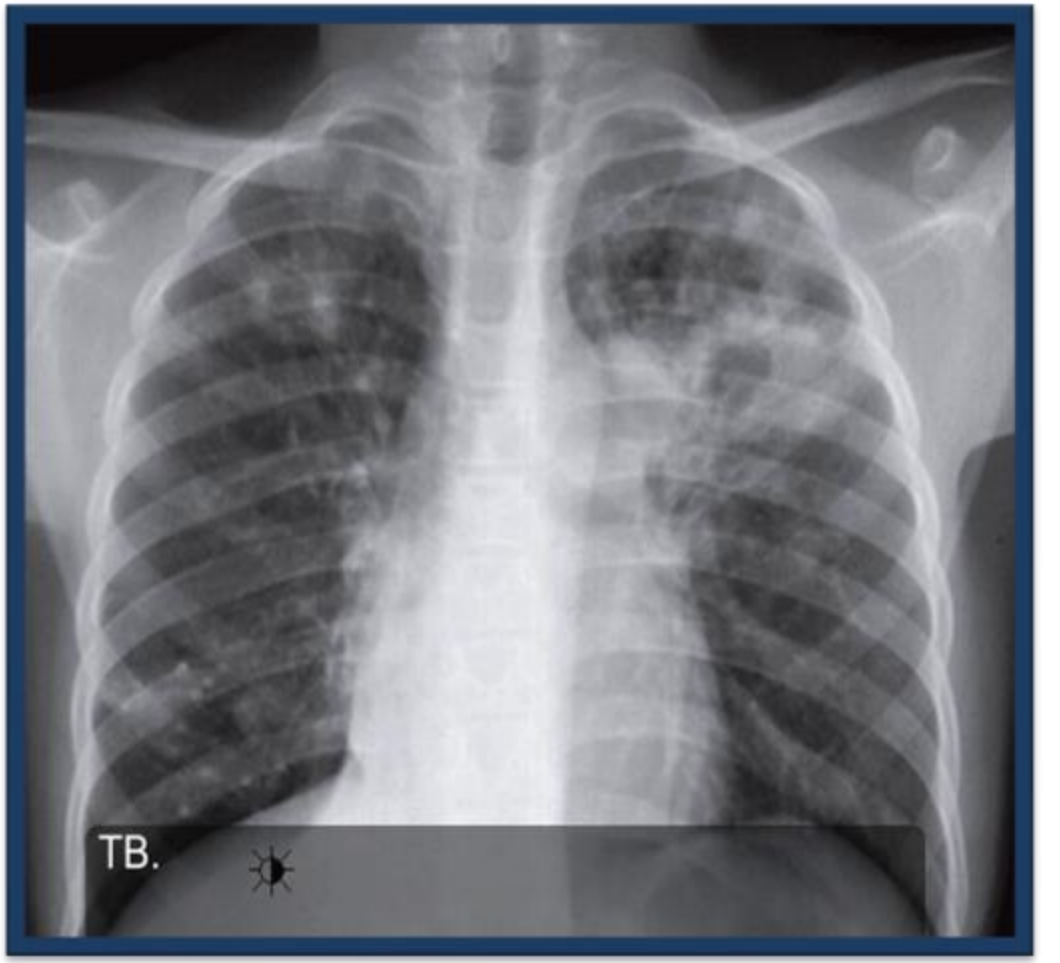
Give 2 DDx?

TB abscess, hydatid cyst.



Q:This Alcoholic pt presented with productive cough, hemoptysis, fever, night sweats, & weight loss. What is your diagnosis?

Active tuberculosis



Q: A patient with suspected TB (or something like that is being tested). Name of test ??

tuberculin skin test

Name the substance injected and how long do you wait before you check the test for the result?

Purified Protein Derivative(PPD)

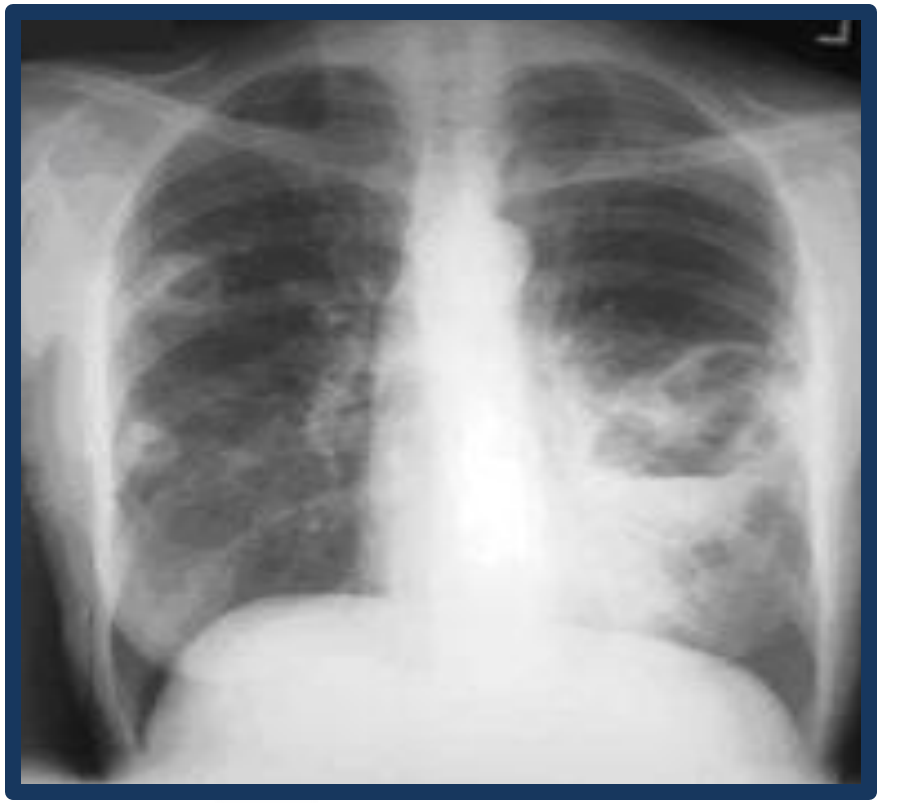
48-72 hours

Q: A 22 year old female patient presents with cough, fatigue, hemoptysis and weight loss of 2 weeks duration.

Mention 2 differential diagnosis

A: Tuberculosis

B:Lung Abscess



≥ 5 mm

- HIV positive
- Recent contact with an active TB patient
- Nodular or fibrotic changes on chest X-ray
- Organ transplant

≥ 10 mm

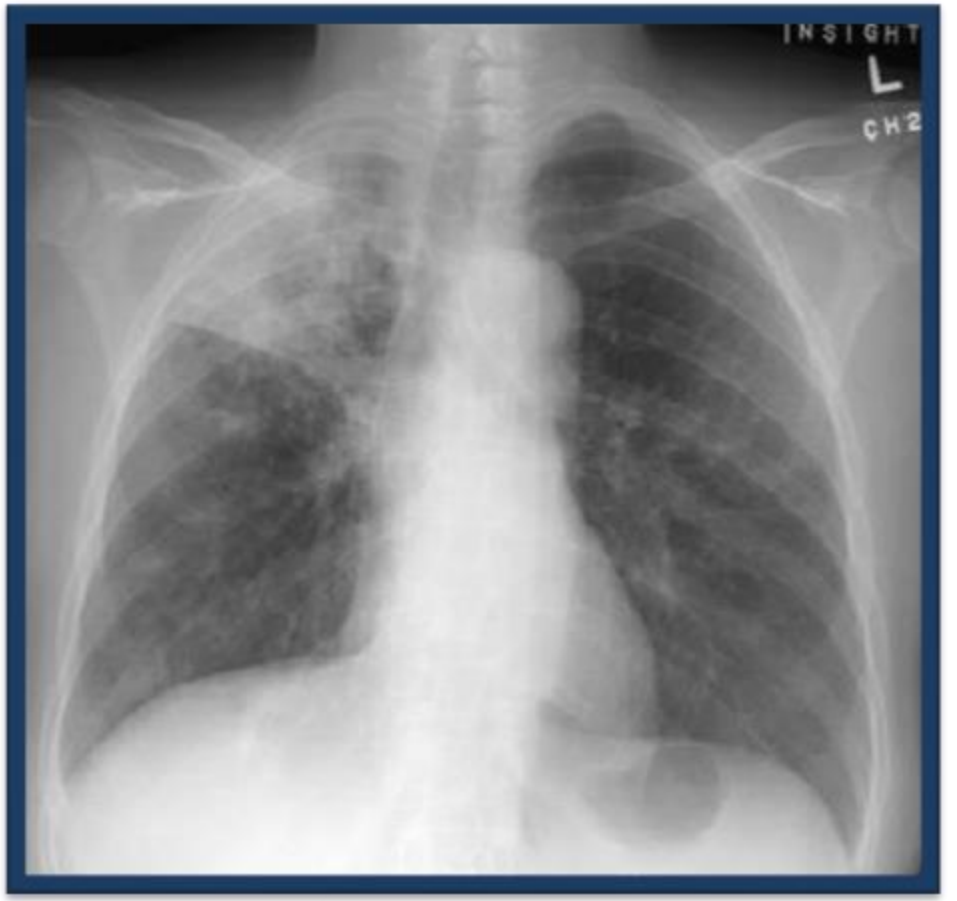
- Recent arrivals (< 5 yrs) from high-prevalence countries
- IV drug users
- Resident/employee of high-risk congregate settings
- Mycobacteriology lab personnel
- Comorbid conditions
- Children < 4 yrs old
- Infants, children, & adolescents exposed to high risk categories

≥ 15 mm

- Persons with no known risk factors for TB

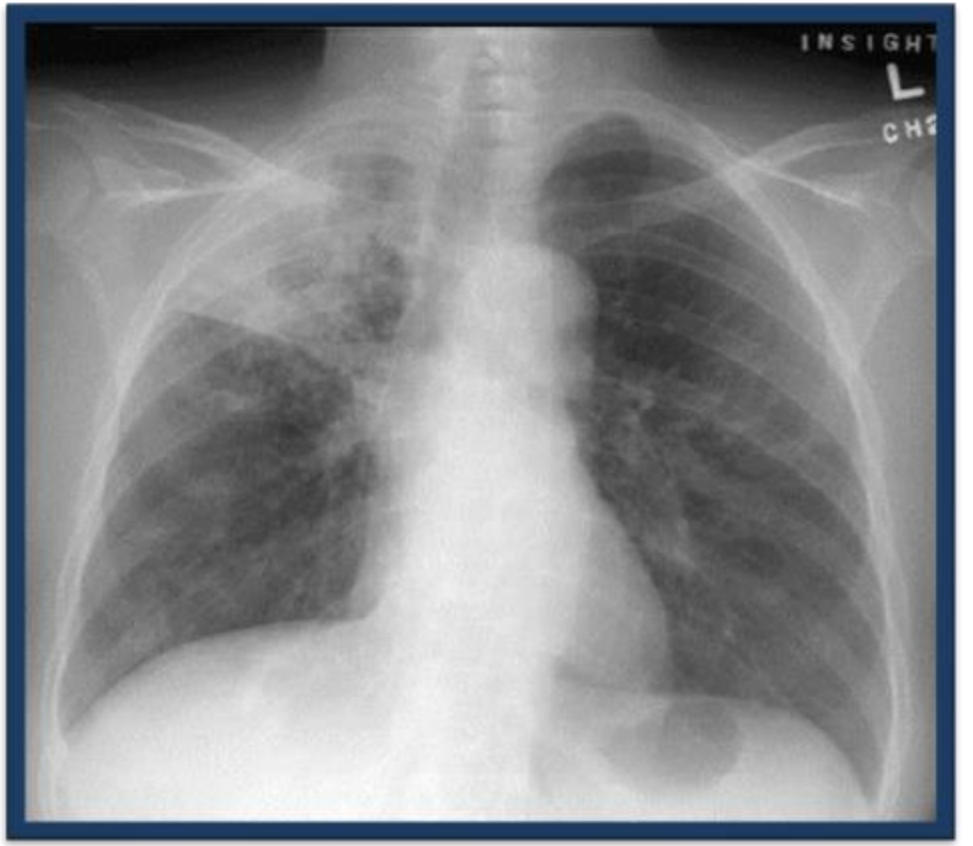
Q: This CXR is for a 30 YO farmer complaining of fever & night sweats 2 weeks prior to admission.
What is your Dx?

Tuberculosis



Q: This pt presented with productive cough, associated with hemoptysis & intermittent fever, resistant to levofloxacillin. what are CXR findings? Investigations?

Rt upper lobe consolidation (TB) >>PPD, Sputum analysis ,Bronchoscopy.



Q: A rheumatoid arthritis patient on adalimumab presented with weight loss and lymph node enlargement, biopsy is shown.

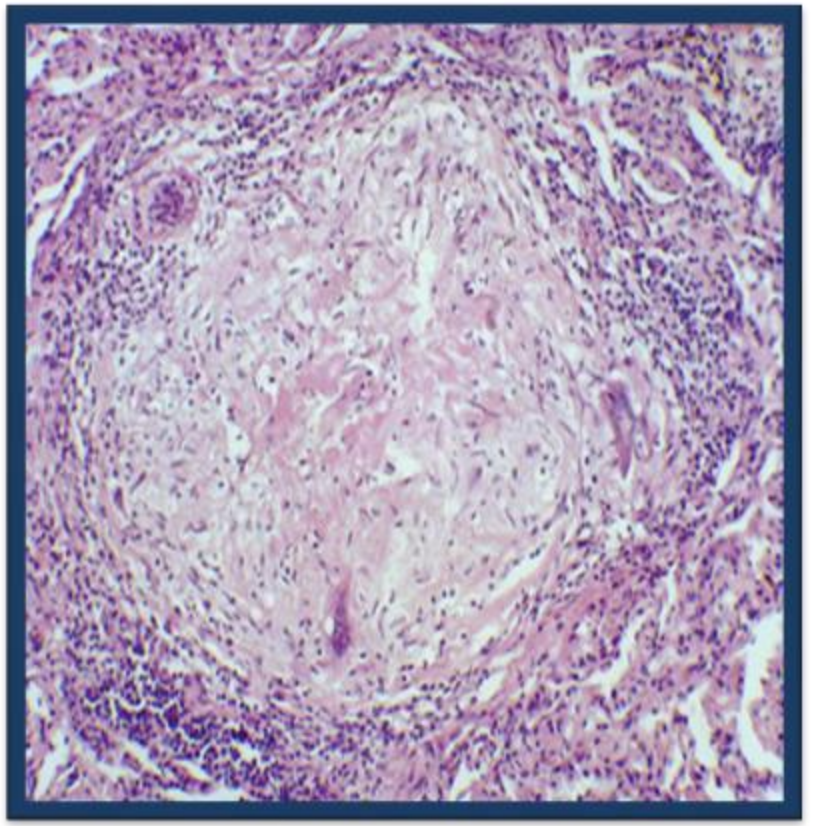
1- What is the diagnosis?

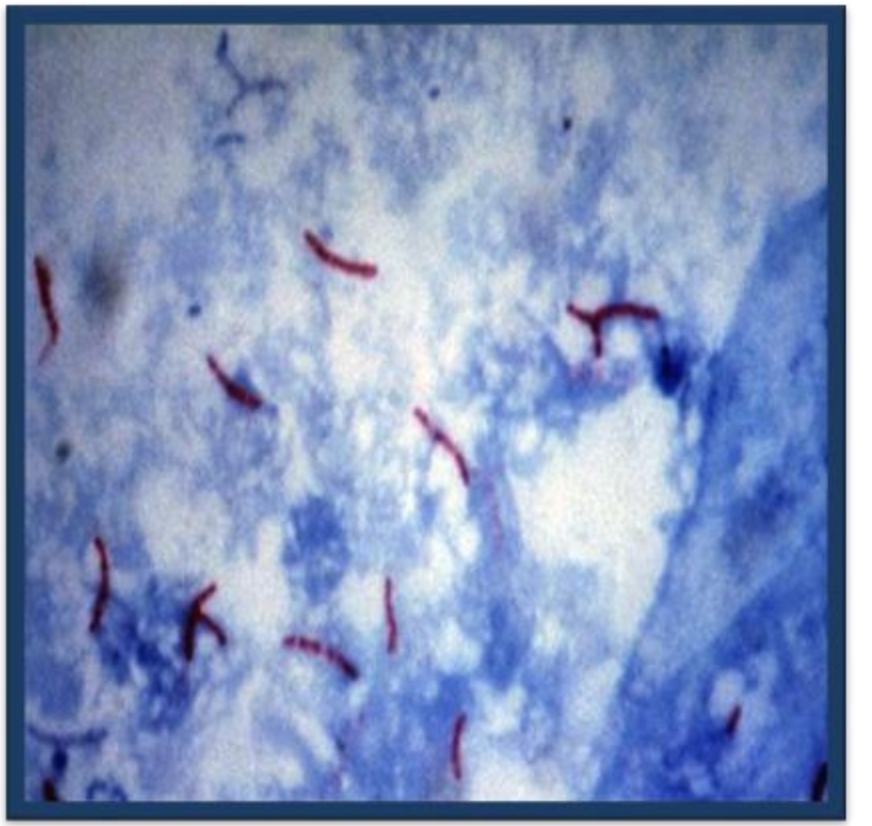
Caseating granuloma and acid fast bacilli, so:

Tuberculosis

2- 2 drugs to manage

Pyrazinamide – Ethambutol – Rifampin - Isoniazid





Others

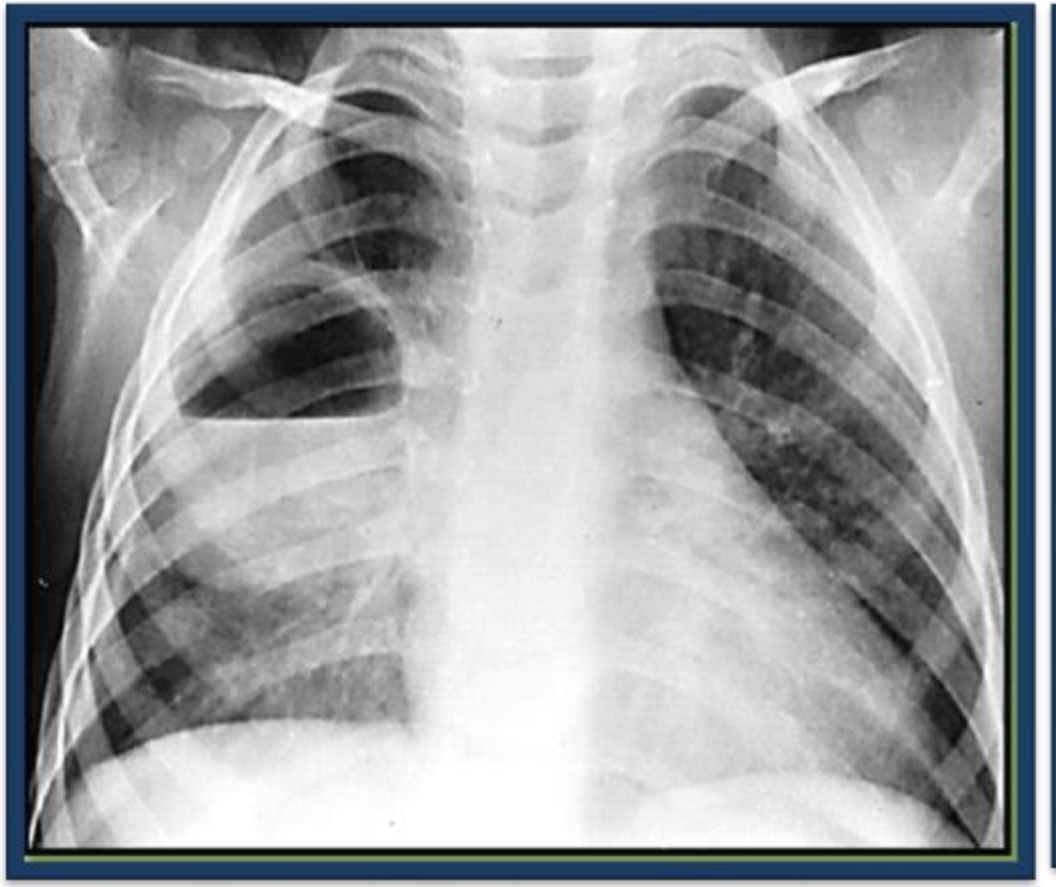
Q:This patient had a 2-week history of fever, rigors and chills.

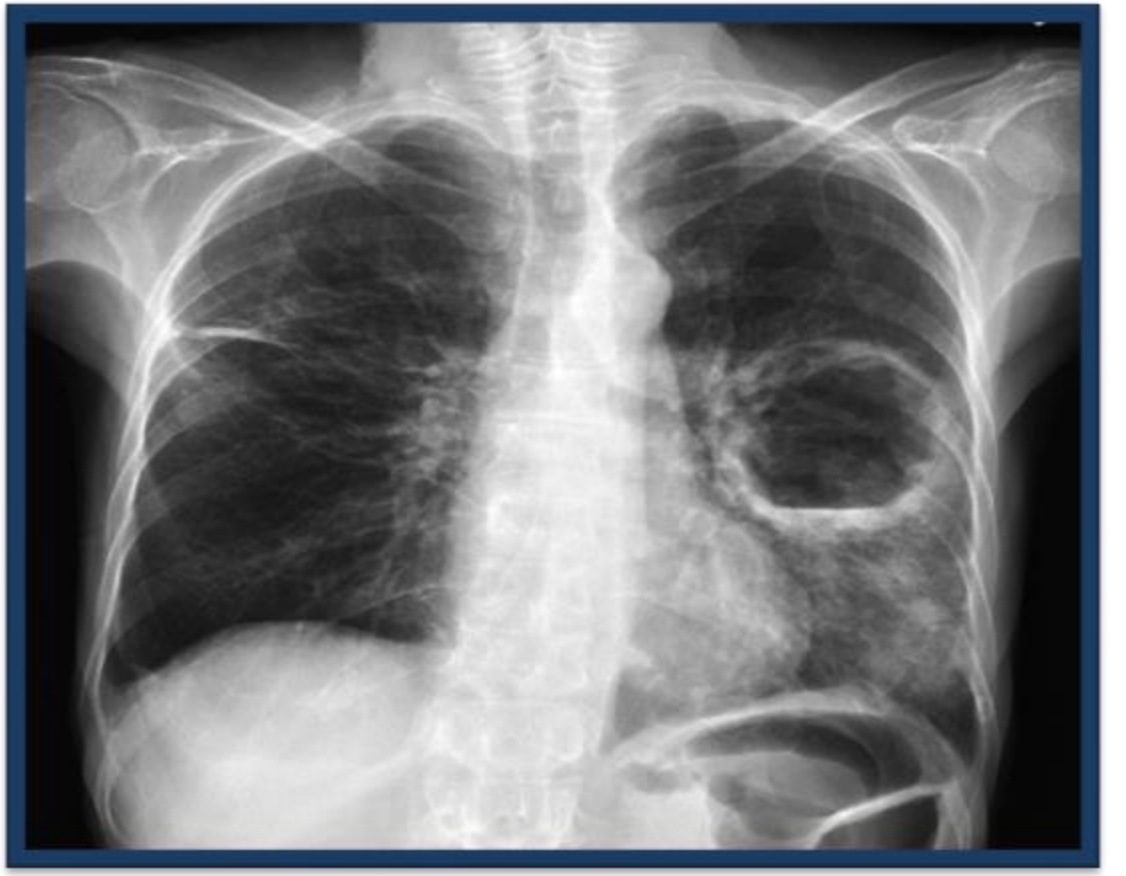
A- What is the diagnosis?

Lung abscess

B- Mention two lines of management.

Antibiotics, Surgical drainage





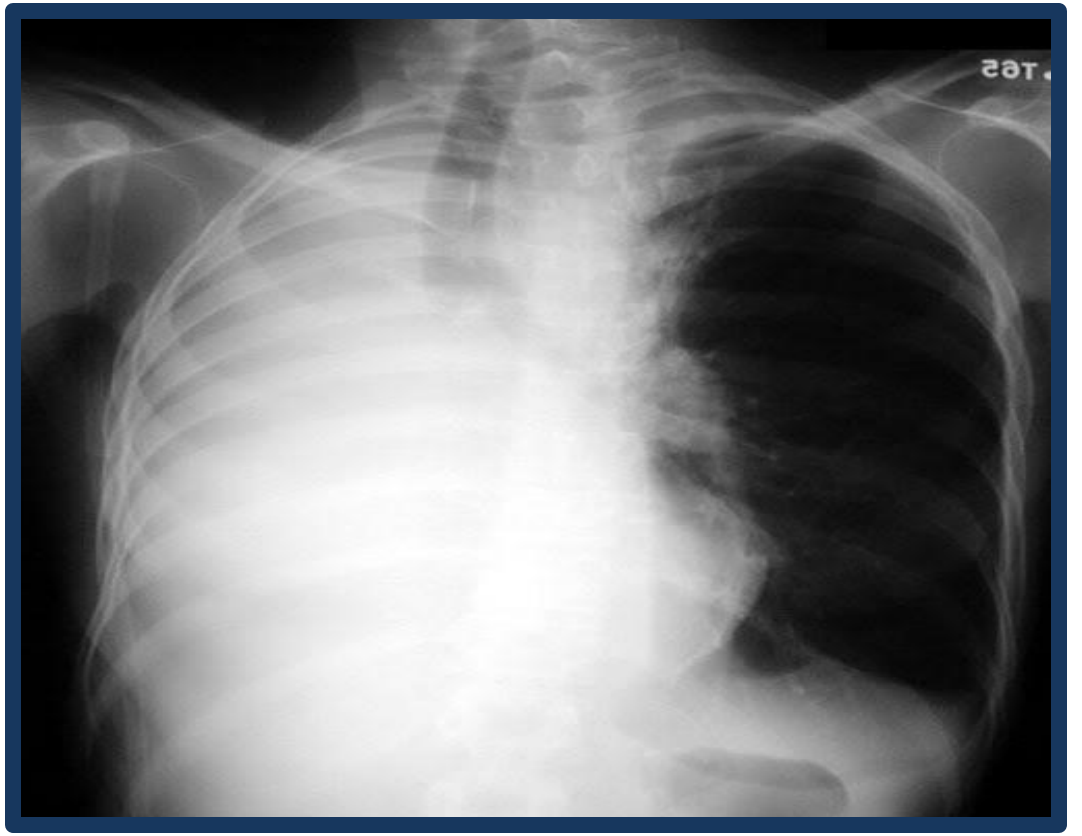
Q: The pt presented with SOB. On physical exam, his chest was dull to percussion.

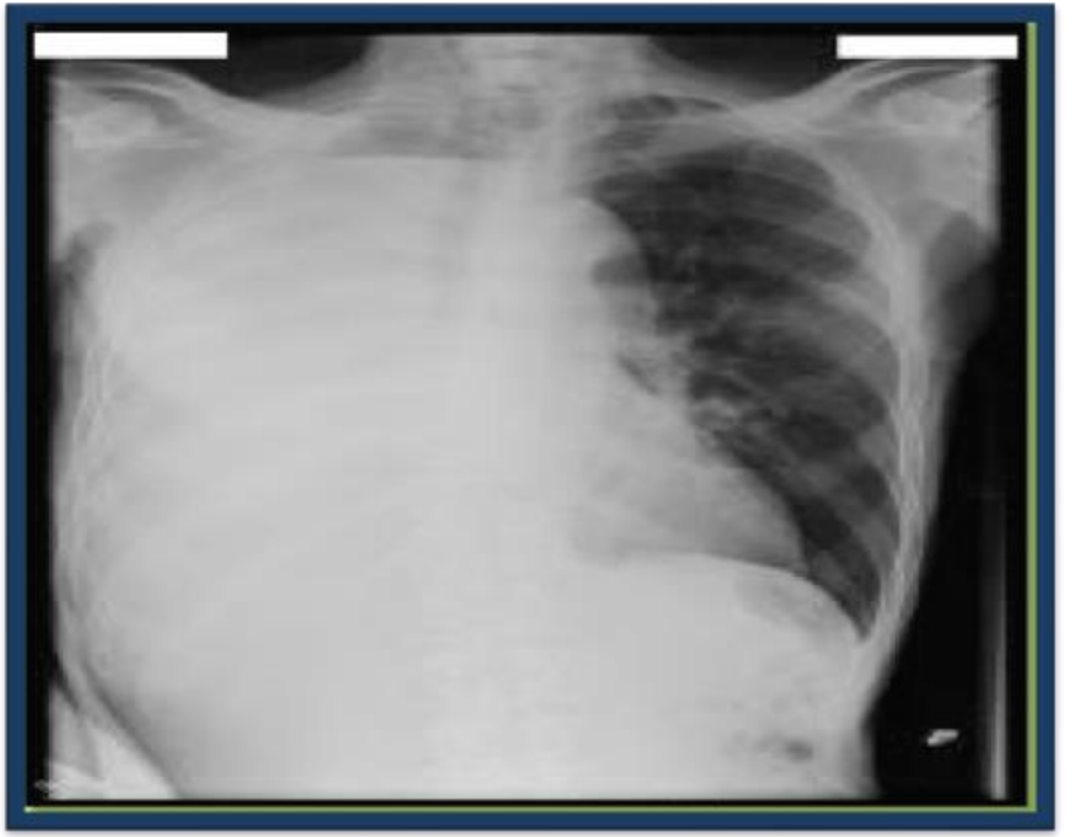
Dx?

right lung collapse/atelectasis .

Possible causes ?

Haemothorax , post operative , pulmonary fibrosis , bronchial carcinoma , massive pleural effusion





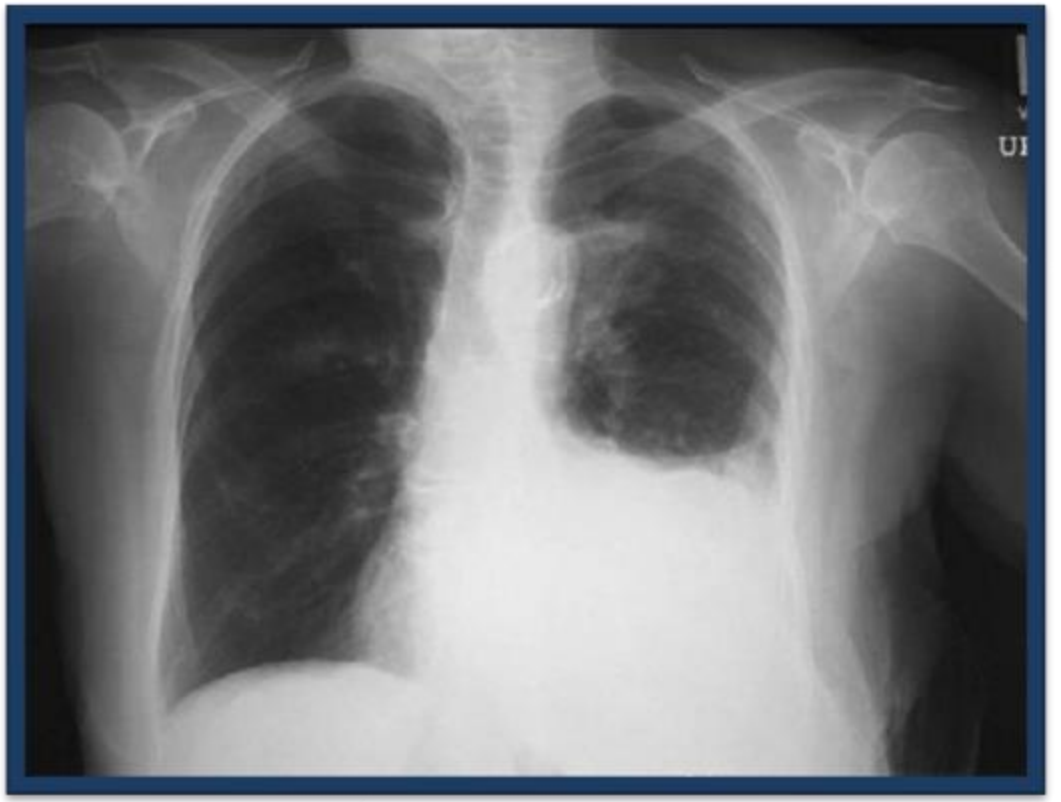
Q: This X-ray is for a pt admitted with SOB, he has stony dullness on percussion, diminished breath sounds, decreased vocal resonance & fremitus over the left side.

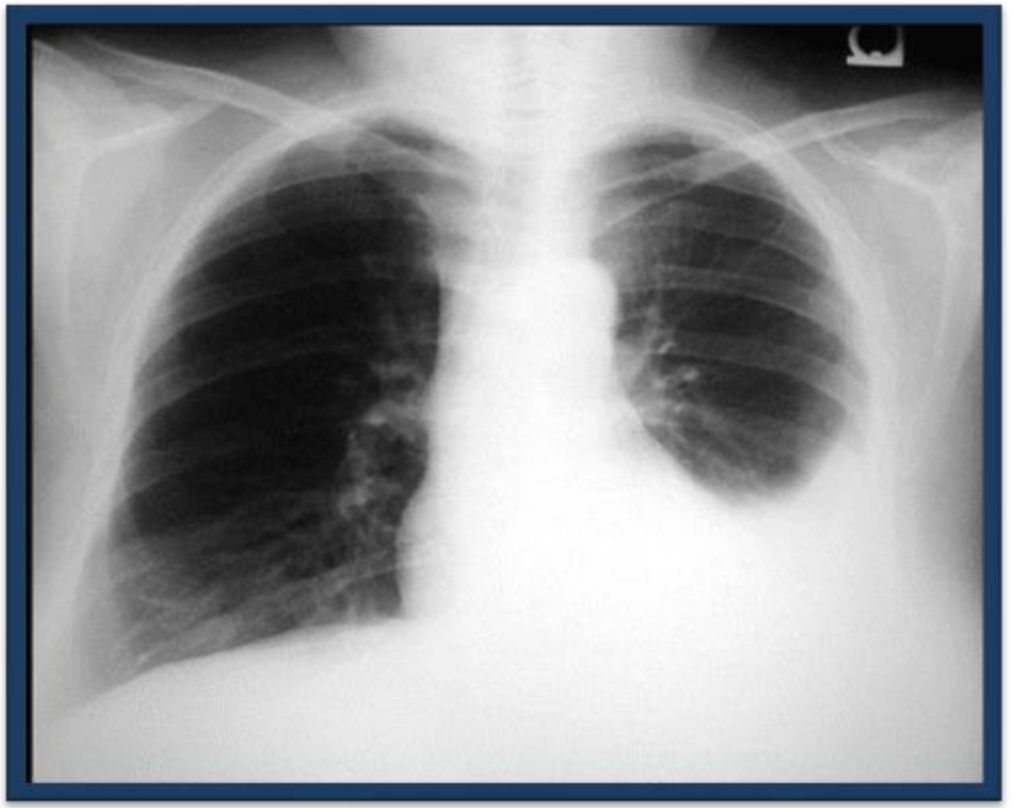
What is your Dx?

Left pleural effusion

Possible causes ?

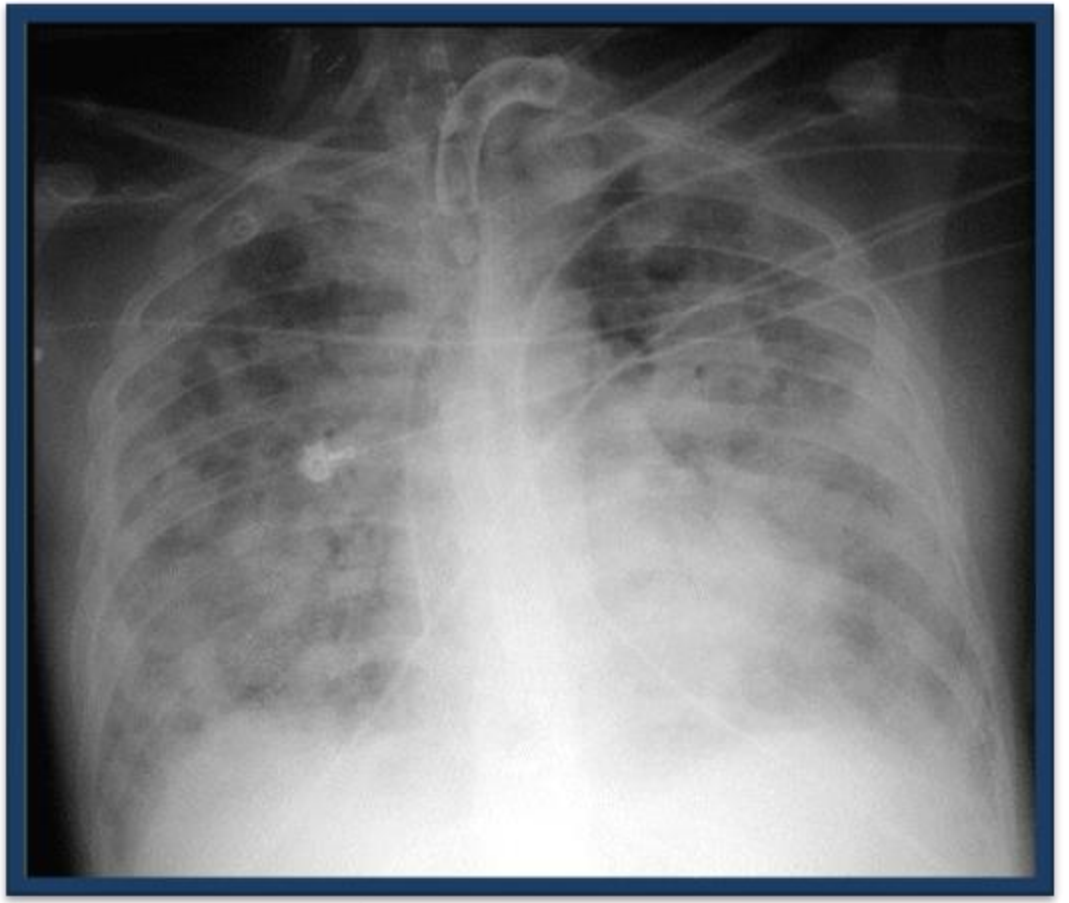
Pneumonia(para Pneumonic effusion) ,CHF , malignancy , ...





Q: 35 YO male pt, known case of pancreatitis only, presented to ER complaining of SOB, What's the cause of his SOB?

ARDS.



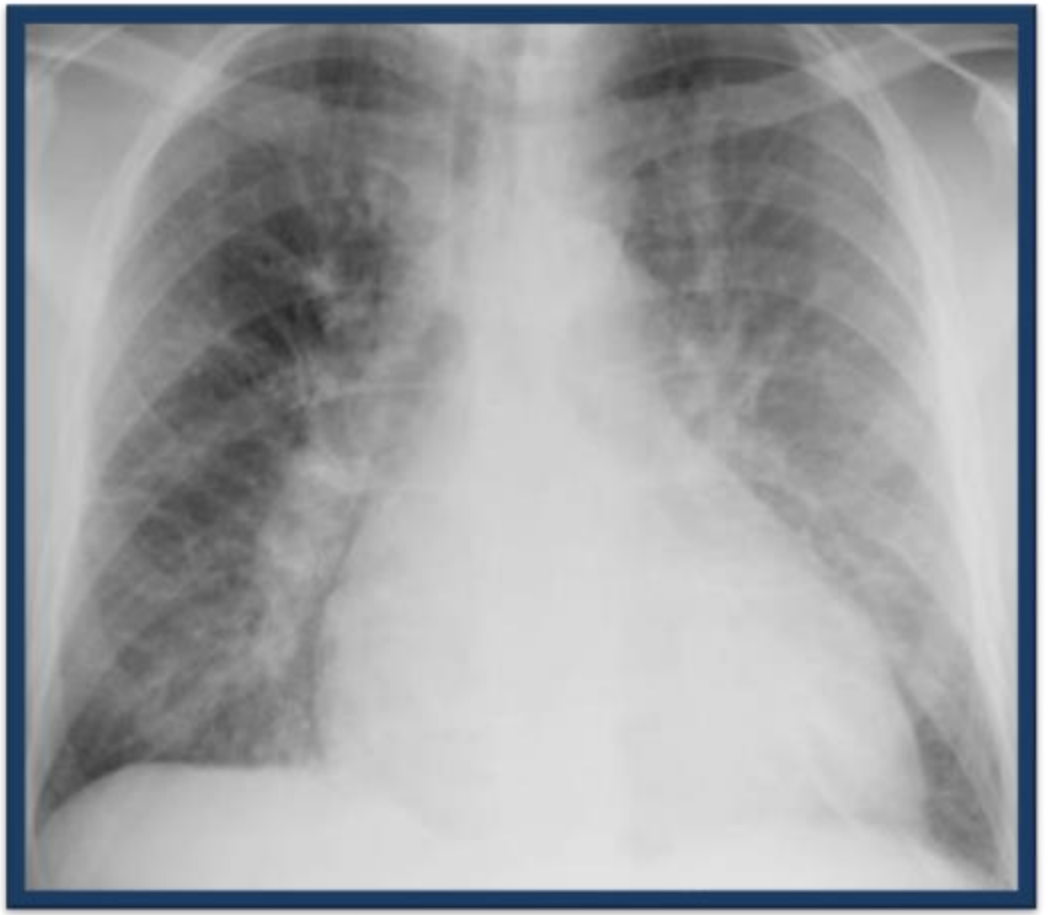
Q: Patient with SOB..

A- What's the most affected valve?

mitral.

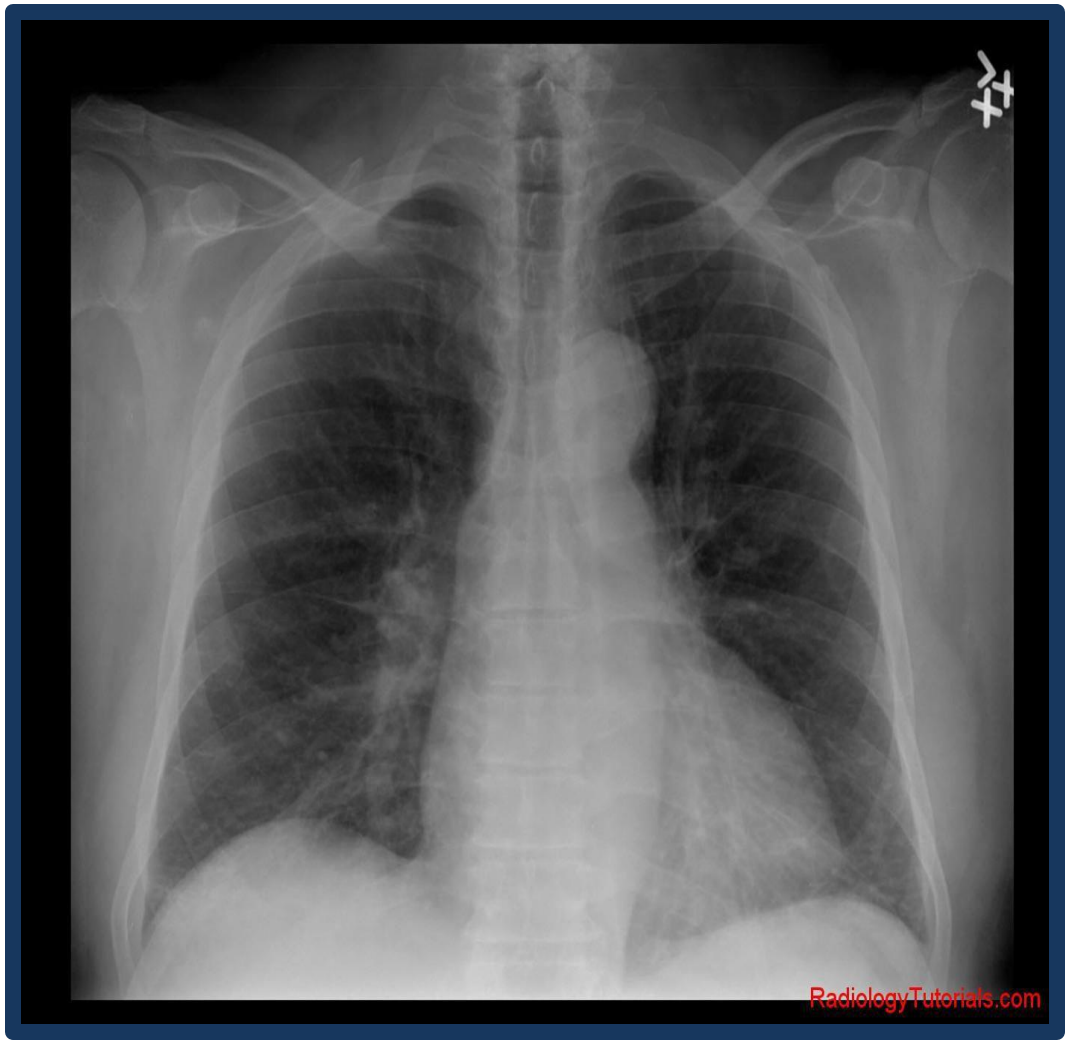
B- What's the cause of SOB?

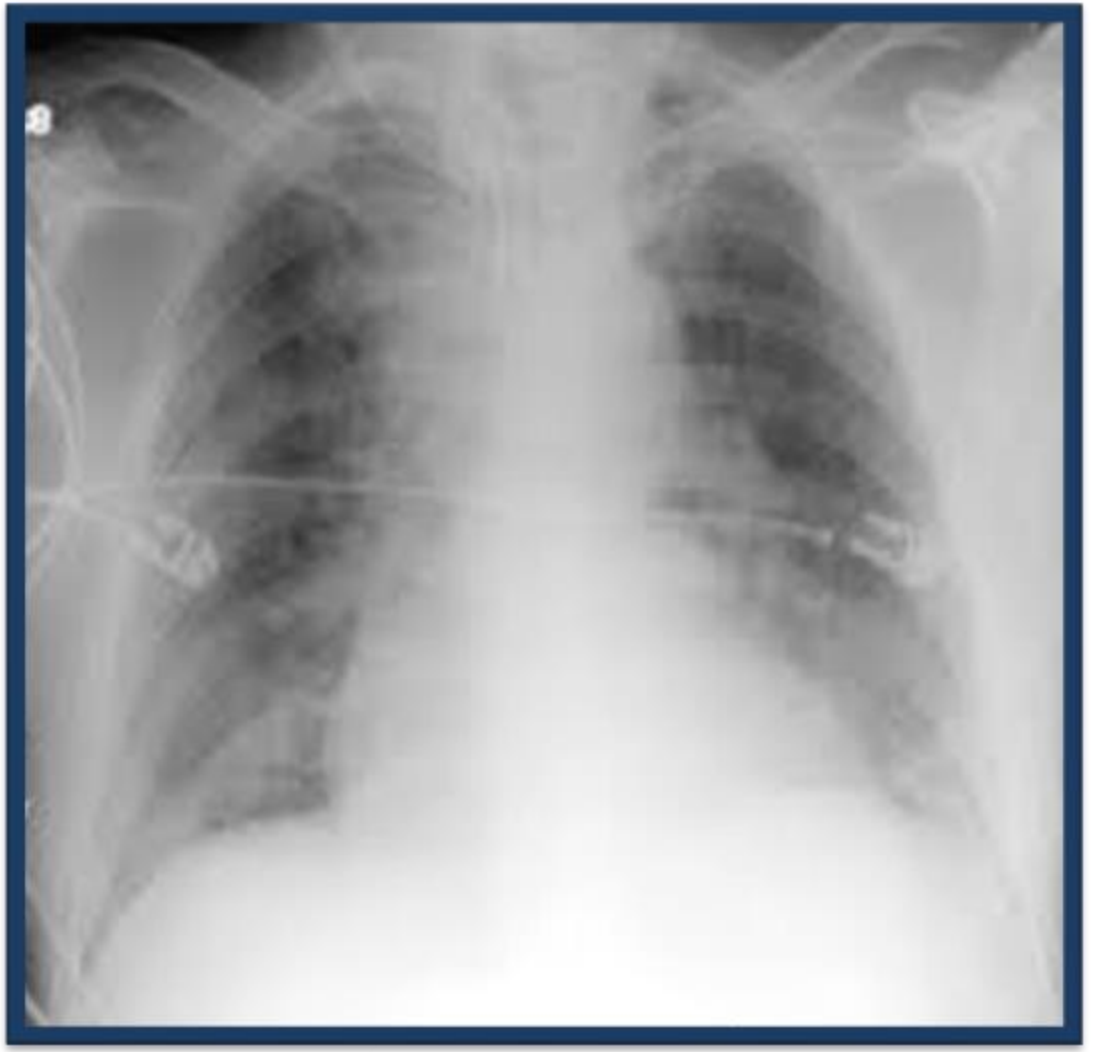
acute pulmonary edema



Q: A known case of hypertension presents with increasing shortness of breath, what is your diagnosis?

Pulmonary edema

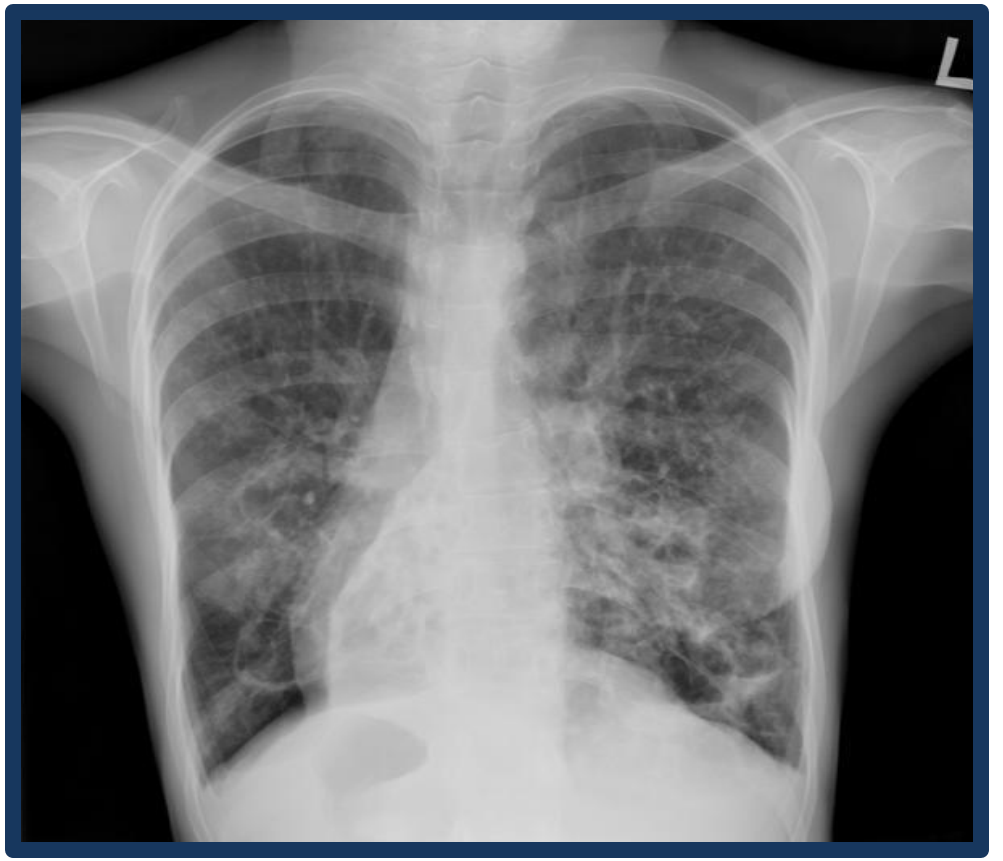




Q: this pt is presented with cough sob and large amounts of sputum ... give two abnormal findings according to the pictures ?

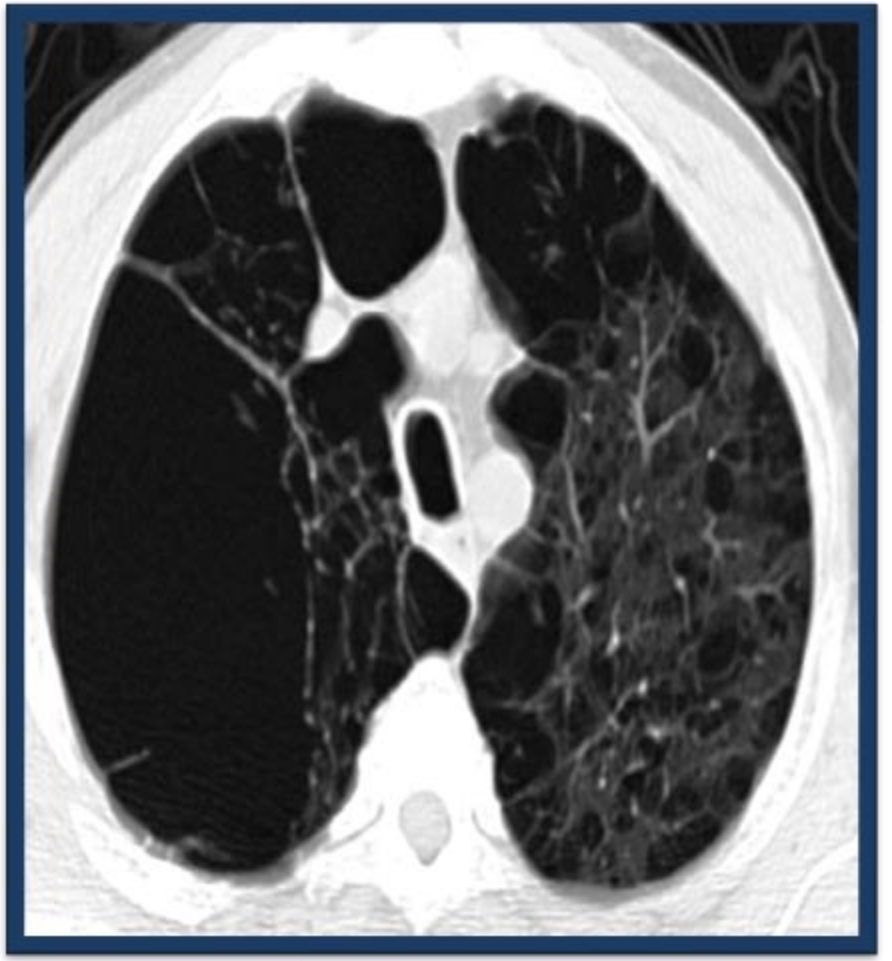
dextrocardia and reticular infiltration on x-ray , finger clubbing .... (kartagener syndrome)

Note : Kartagener syndrome triad : situs inversus , bronchiectasis , chronic sinusitis



Q: Mention the abnormal radiological finding in this picture.

Bullous Emphysema

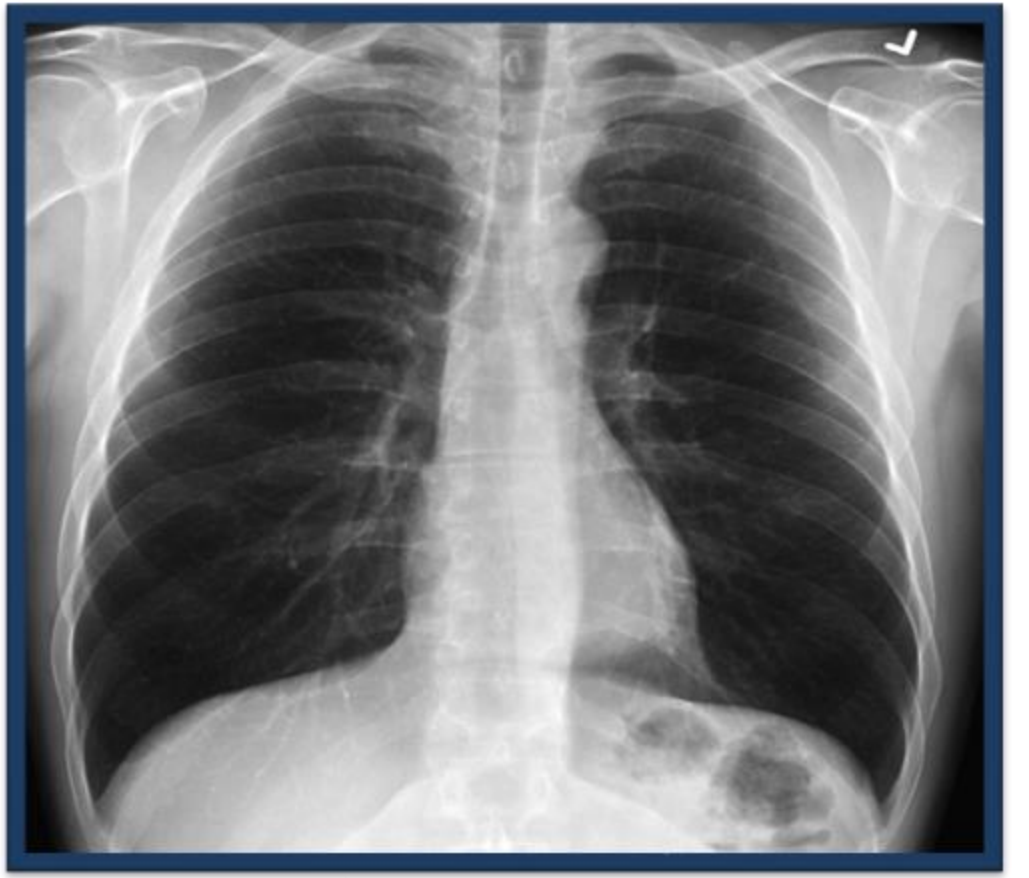


Q: Long history of smoking, presented with this chest X-ray, what is your radiological diagnosis?

Emphysema

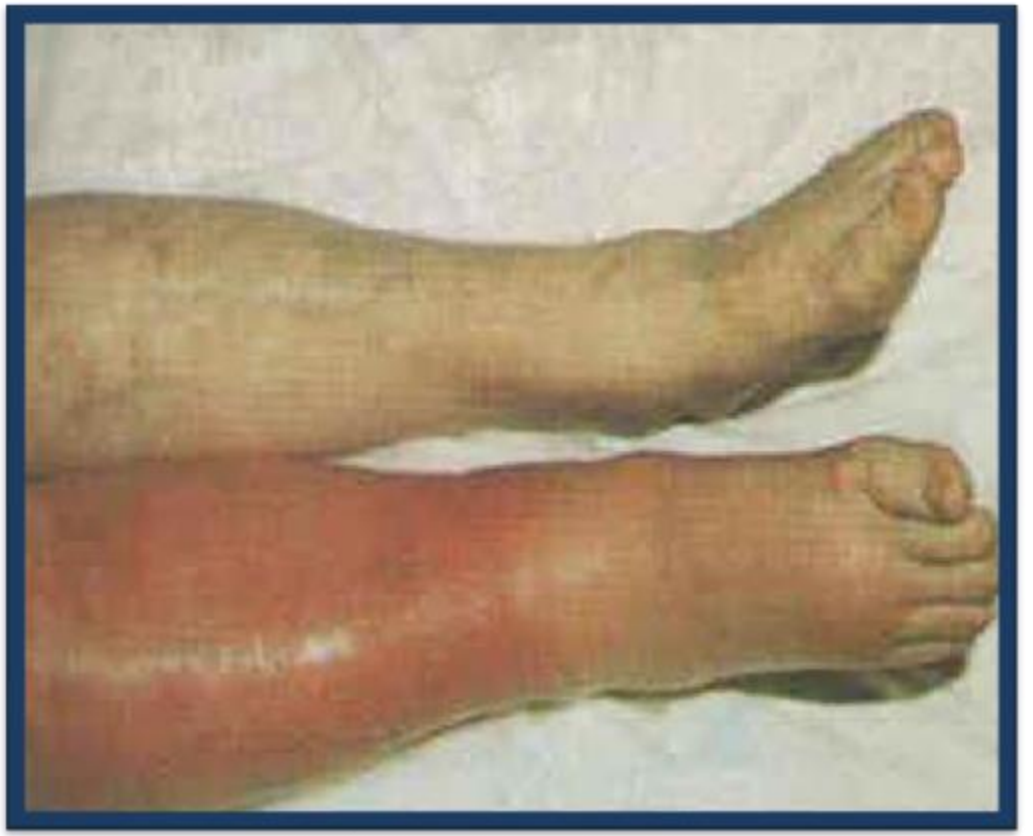
What is the most common presentation?

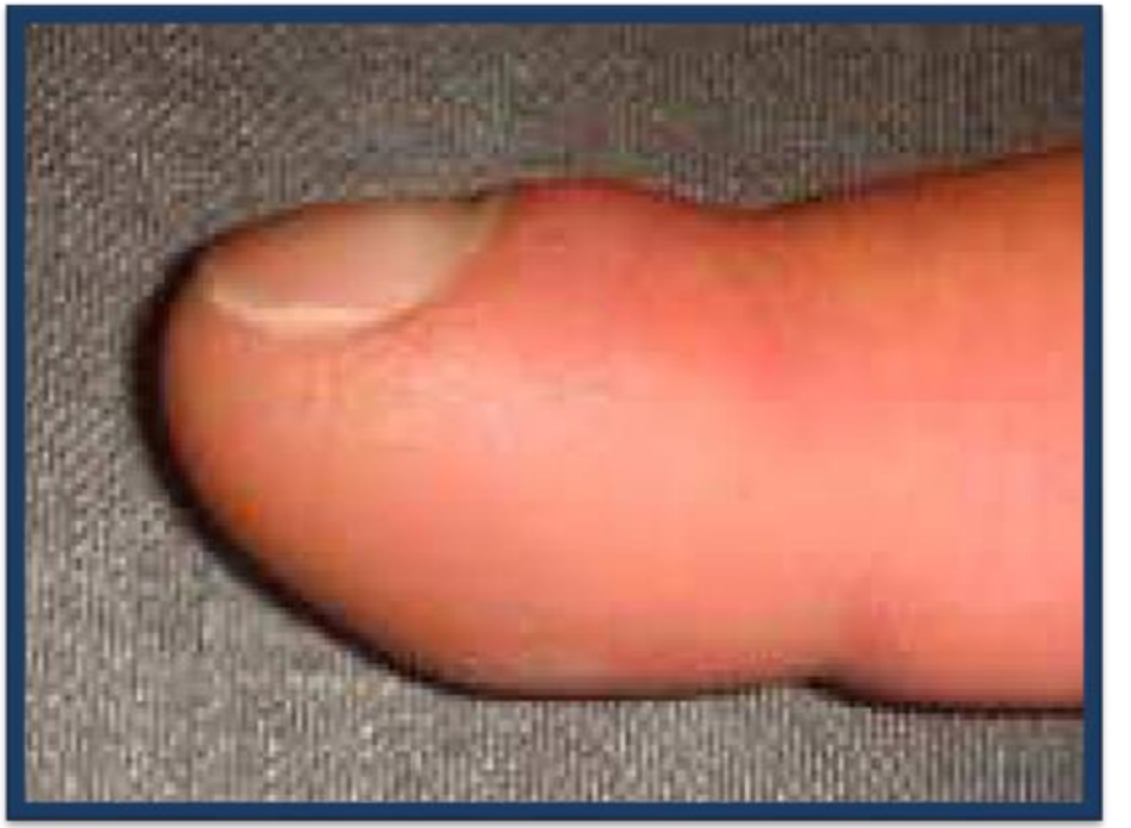
Shortness of breath



Q: This patient was presented with sudden onset chest pain with S.O.B.
A-What is the cause of his presentation?
Pulmonary embolism
B-How to diagnose
CT-Angiography

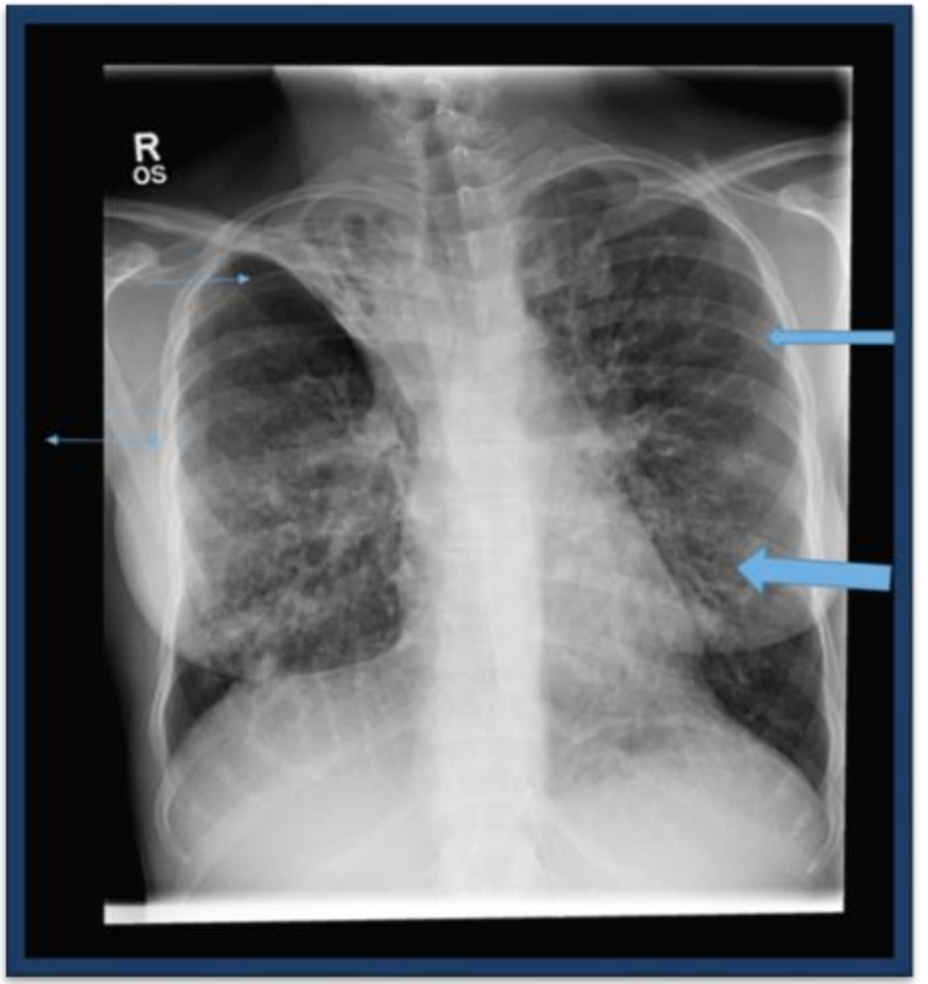
Q: Mention two respiratory causes for this condition?
Cystic Fibrosis, Bronchiectasis Lung Carcinoma, …





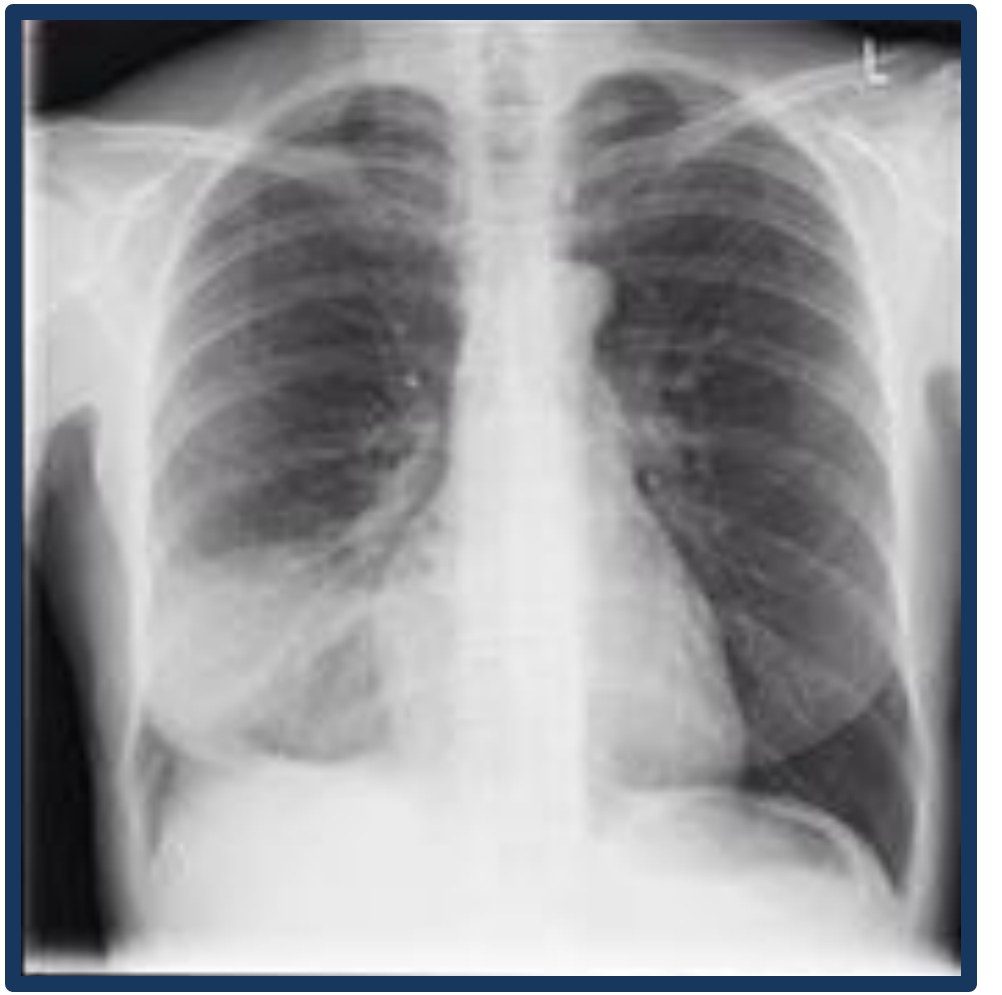
Q:This young patient has large amounts of sputum production with recurrent infections, what's the diagnosis?

Bronchiectasis



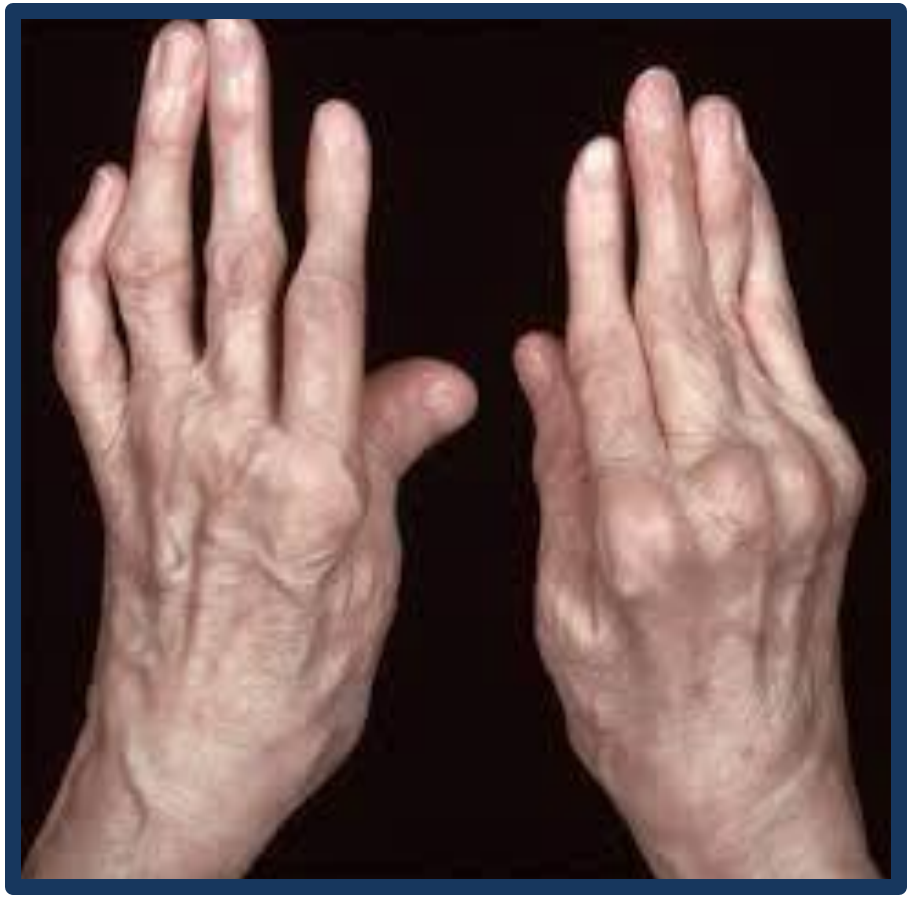
Q: Name 2auscultatory findings ?

1 bronchial breathing
2 crepetations
3 increased vocal resonance



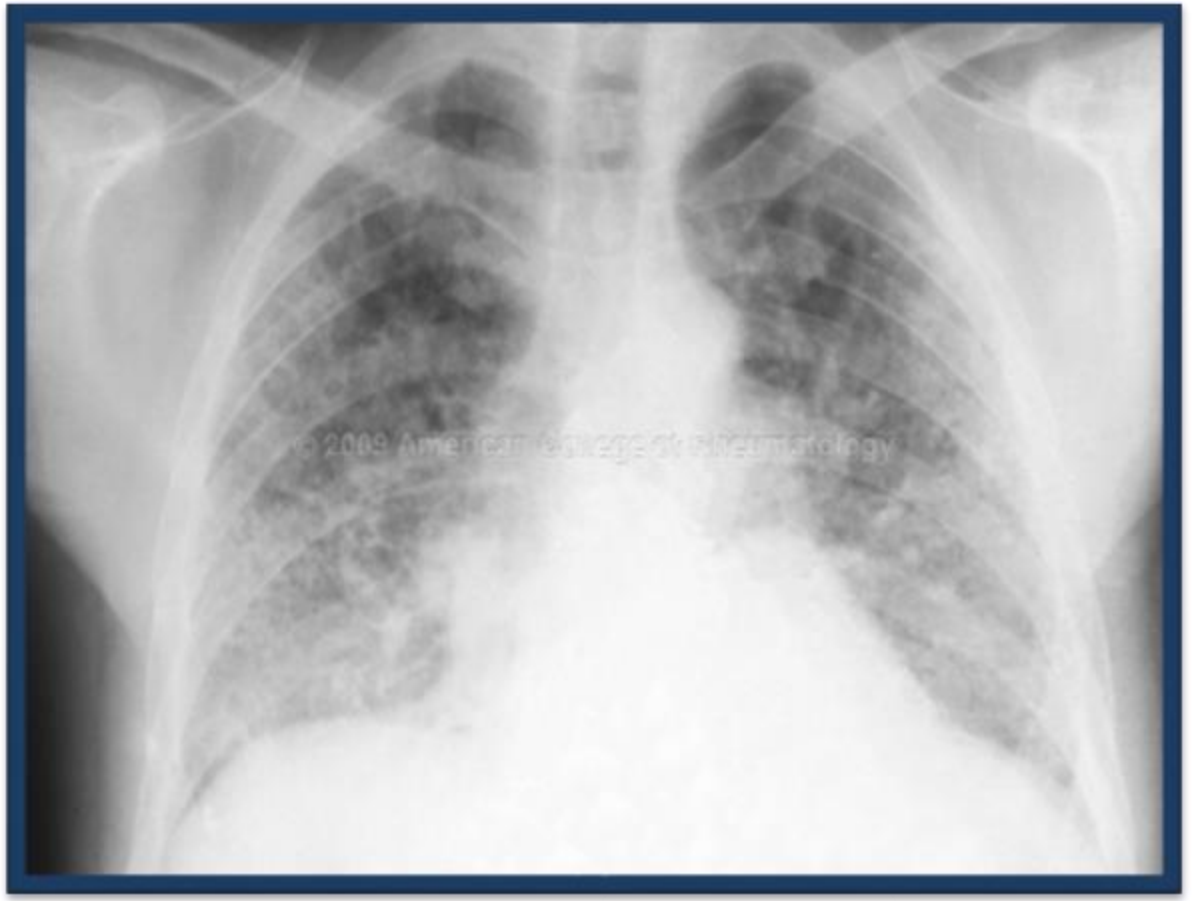
Q: This pt has developed gradual SOB , what's the Cause?

Pulmonary fibrosis



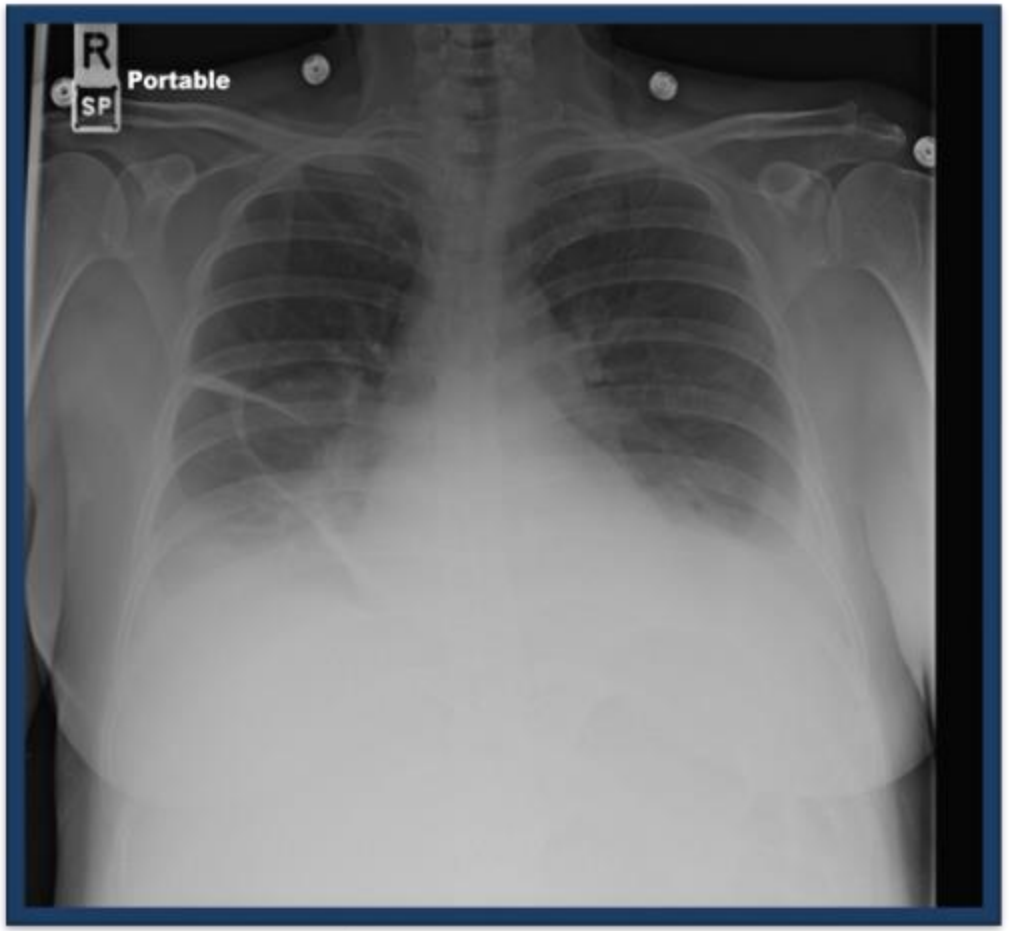
Q:Presented with progressive SOB

Lung Fibrosis



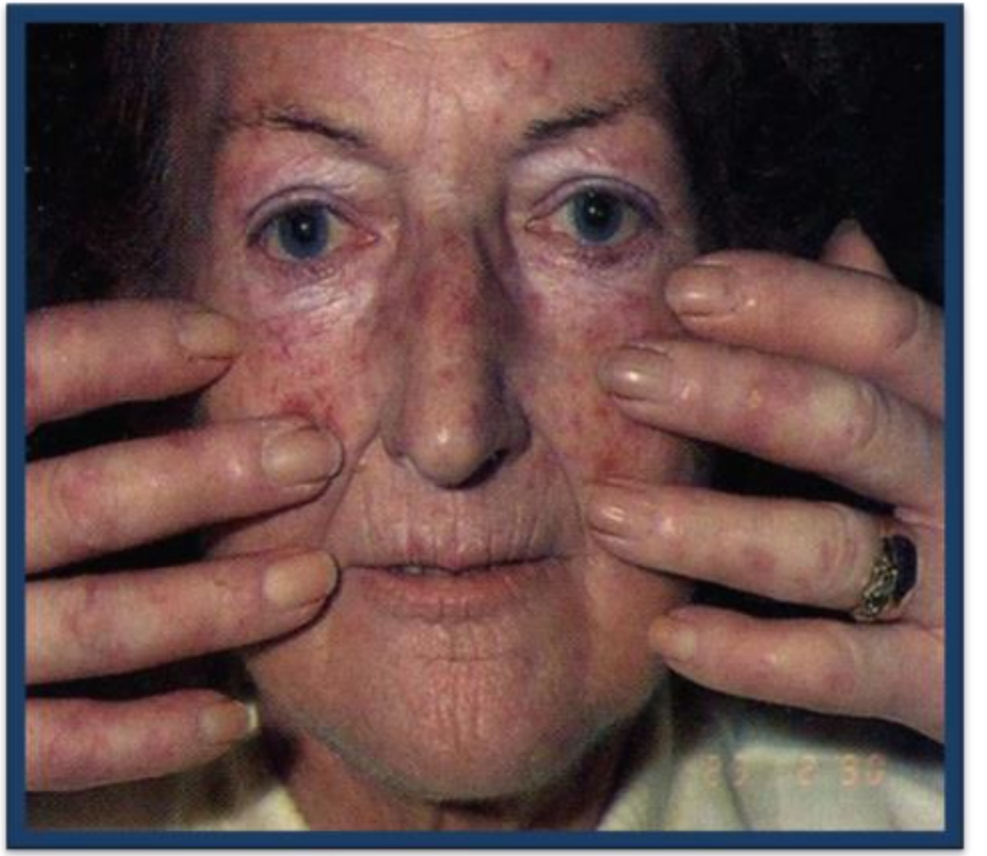
Q: Dyspneic patient, chest exam reveals both sided dullness and basal decreased air try, you obtain this chest radiography.
Name the Diagnosis?

Bilateral lower pleural effusion



Q: This patient has Raynaud Phenomenon, severe heart burning sensation and dysphagia presents with chronic hypoxia.
- Name 2 possible causes of chronic Hypoxia.

1. lung fibrosis
2. pulmonary hypertension



Cases

(the history was about acute asthmatic exacerbation , the following are the main points):

27 YO pt presented with SOB associated with fever, chills & cough with yellow sputum, the patient was unable to talk & uses his accessory muscles, RR=30, BP = 100/70, T=39.5 , he had Hx. of previous attacks.

1. Mention 2 signs indicating the severity from Hx.

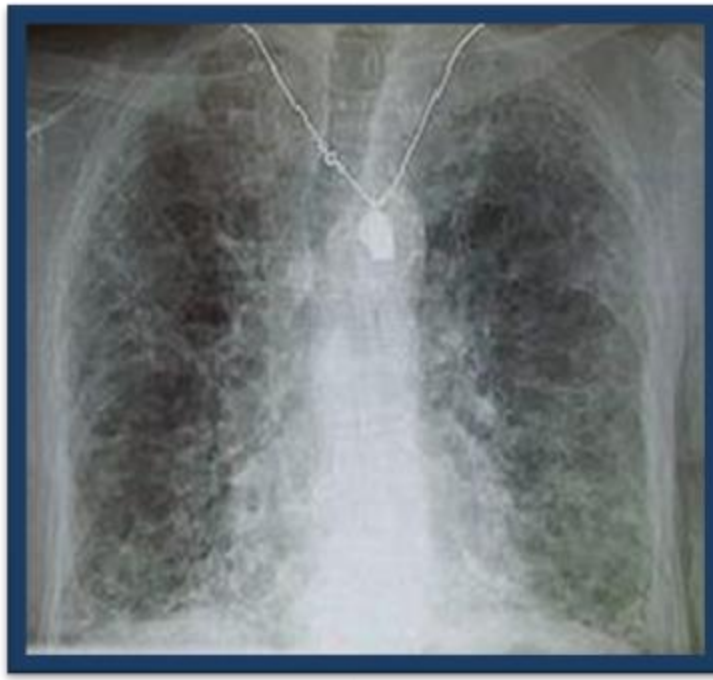
patient was unable to talk & BP = 100/70 .

2. Mention 3 lines of management.

1. Initial Management of Asthma Exacerbation:1. Oxygen therapy to maintain O2 saturation of 94-98%.
2. Nebulized B2-agonist (salbutamol 5mg or terbutaline 10mg).
3. Systemic corticosteroids (oral prednisolone 30-60mg or IV hydrocortisone 200mg).
4. Antibiotics if evidence of infection on chest X-ray, purulent sputum.
5. IV fluids if necessary.

Q: A 55 year old male patient presented with progressive SOB for 3 months. On examination he had raised JVP, lower limb oedema, & clubbing. And this is his chest X ray. Lab results:

- ABG: pH 7.46 / CO2 30 / O2 60
- PFT: FEV/FVC 90 / FVC 60



What is the Dx ?

Idiopathic pulmonary fibrosis with corpulmonale.

What is the Acid base abnormality in his ABGs?

Chronic respiratory alkalosis.

What is the interpretation of his ABG?

Hypoxia without hypercapnia (Type I respiratory failure).

What is the interpretation of his spirometry?

Restrictive lung disease.

What is the treatment ?

Supportive measures, O2 supplement.

Q:A Patient presented with cough, SOB , fever, arthritis , painful Lower limb lesions

What is your diagnosis ?

Sarcoidosis

mention one investigation to confirm your diagnosis

Excisional lymph node biopsy

mention 3 extra pulmonary manifestations of the disease

-arthritis

-Lupus pernio

-hypercalcemia

-bells palsy

Q: 32 YO female pt, presented with sudden onset of dyspnea, she has Hx of pregnancy 2 weeks ago

1. What is the most probable Dx? "2 marks"

Pulmonary embolism.

2. Give 2 diagnostic tests for this pt?

CT angio, D-dimer, V\Q scan.

3. What is the treatment?

LMWH (Anticoagulant).

30 year old female patient, presented with progressive SOB over the last 3 months. On examination she has clubbing, raised JVP & lower limb edema. There was ABG result & PFT results.

1. What's your diagnosis?

Right sided heart failure.

2. what's the best diagnostic test?

Biopsy.

3. what's the cause of her condition?

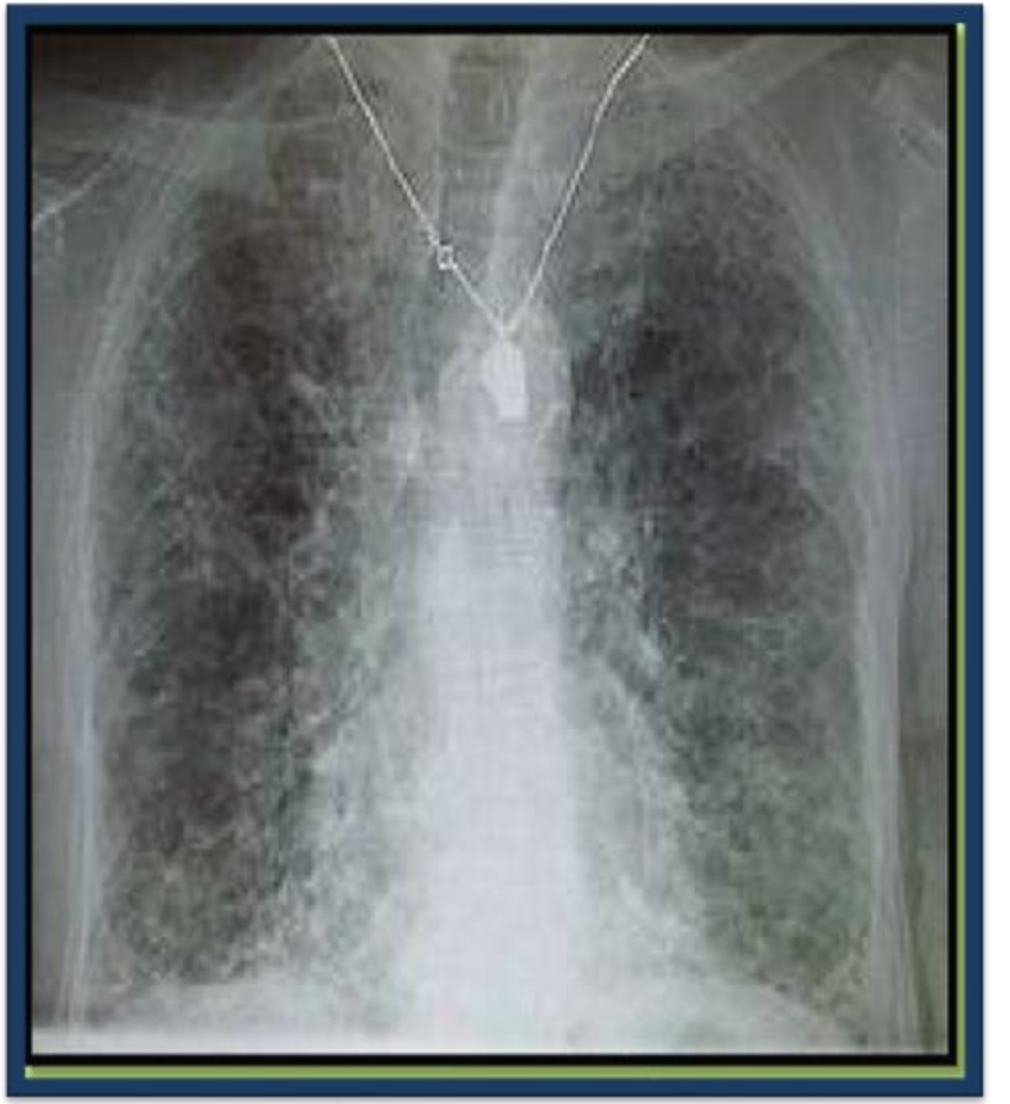
Pulmonary fibrosis.

4. Interpretation for ABG ?

Respiratory Alkalosis.

5. interpretation for PFT ?

Restrictive lung disease.



Q: A 60 year old male, known case of poorly controlled Hypertension, came to your clinic complaining of excessive somnolence & fatigue. He has a short neck, his Body Mass Index > 35.

What is your most likely diagnosis?

Obstructive Sleep Apnea

What is the confirmatory test?

Polysomnography

What complications is the patient expected to have
(Mention 2)

Pulmonary Hypertension, cor pulmonale

Mention one line of management (other than smoking cessation)

nCPAP

Q1) what is the abg showed?

-Metabolic acidosis

Q2) calculate the anion gap

Na – (CL+HCO3) = 145 – (100+10) = 35

Wide anion gap

Q3) mention 3 causes for this condimon?

Dka , lactic acidosis , methanol , etc.

- ABG Case
- Ph: 7.29
- Co2: 22
- hco3: 10
- Cl: 100
- Na: 145
- + other labs , normal values was given

This ABG is from patient presented to ER C/O vomiting & SOB?
What are the metabolic disturbances ?
Mixed alkalosis

PH	7.62
PCO2	28.5
HCO3	30
PO2	234 (FIO2 50%)

Patient presented to ER c/o vomiting what is the metabolic disturbance ?
Mixed alkalosis

PH	7.62
PCO2	28.5
HCO3	30
PO2	234 (FIO2 50%)
HCO2 excess	8.2
Na	132
Cl	90
K	2
Glucose	12.7 (X18)
Lactate	1.1 (<1.3)

40 y/o RA, complain of epigastric pain & vomiting, she is already on Aspirin?
Mention 2 metabolic disturbance caused by Aspirin ?

aspirin toxicity causes initial respiratory alkalosis then later metabolic acidosis

PH	**7.7**
PaCO2	**25**
PaO2	**85**
HCO3	**30**
Na+	**135**
Cl-	**88**
ALBUMIN	**4**

- 40 y/o RA, complain of epigastric pain & vomiting, she is already on Aspirin.
- What is the metabolic disturbances in this patient?

PH	**7.7**
PaCO2	**25**
PaO2	**85**
HCO3	**30**
Na+	**135**
Cl-	**88**
ALBUMIN	**4**

18 year-old comatose, quadriplegic patient who has the following ABG done as part of a medical workup:

- What is the Acid base disturbance?

Respiratory alkalosis with metabolic compensation

pH	7.48
C02	22
p02	96
HC03	16
Sa02	98%

GIT

Done By :

حمزة وادي & أنس حسونة

إسلام وادي & عمر كفاية

هاجر بني دومي & ديمة الحجايا

فرح العلي & مرح البعول

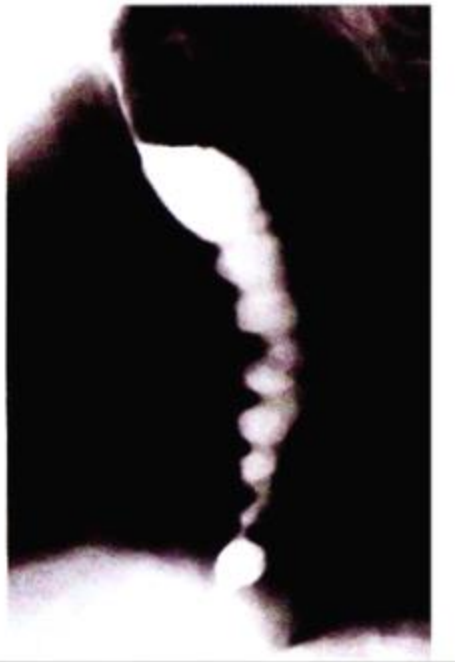
أحمد العزام

References are:

- DAVIDSON
- STEP UP
- DOCTORS' LECTURES AS WELL

1.Esophagus

- 32 year old male complaining of (crushing) chest pain precipitated by cold drink, no sweating, no vomiting, ECG normal, cardiac enzyme negative, barium swallow was done and show:



- What is the diagnosis ?
- What is the test that confirm Diagnosis ?

1)Diffuse esophageal spasm
2) esophageal manometrey

Diffuse esophageal spasm	
Pathophysiology	• Uncoordinated, simultaneous contractions of esophageal body
Symptoms	• Intermittent **chest pain** • **Dysphagia** for solids & liquids
Diagnosis	• Esophagram: "**Corkscrew**" pattern • Manometry: Intermittent peristalsis, multiple **simultaneous contractions**
Treatment	• Calcium channel blockers • Alternate: Nitrates, tricyclics

Barium esophagram

Q: A 30 year-old male patient comes with difficulty of swallowing food and drinking water for 10 years. Associated with foul breath smell and weight loss. Above is the x-ray with barium (Ba) meal showing a stricture. What is this condition?

Achalasia

-What other differential diagnosis of this condition?

1- diffuse esophageal spasm
2- Gastroesophageal reflux disease
3- esophageal carcinoma
4- Scleroderma

-What causes/mechanisms of this condition?

1- Absent peristalsis in the lower 2/3 of the esophagus (most. Imp.)
2- Failure relaxation of LES
3- Decreased or absent intramural esophageal ganglion cells

-What are the clinical presentation of this condition

1- Dysphagia (long standing, to both food and fluid)
2- Regurgitation of foods (cause halitosis)
3- Chest pain (could be mistaken with MI)
4- Aspiration pneumonia (may cause lung abscess, bronchiectasis, or hemoptysis)
5- Weight loss

Achlasia.

-What investigation(s) should be done to confirm the diagnosis?

1- Esophageal manometry (uncoordinated or abscent peristalsis with high LES resting pressure) (to confirm the Diagnosis)

2- Barium esophagram(bird peak sign)

3- Endoscopy (with biopsy to rule out esophageal malignancy)

-What are the treatment of this condition

1- medication(nifedipine,nitrate,nitroglycerate)

2- Balloon(pneumatic) dialtation

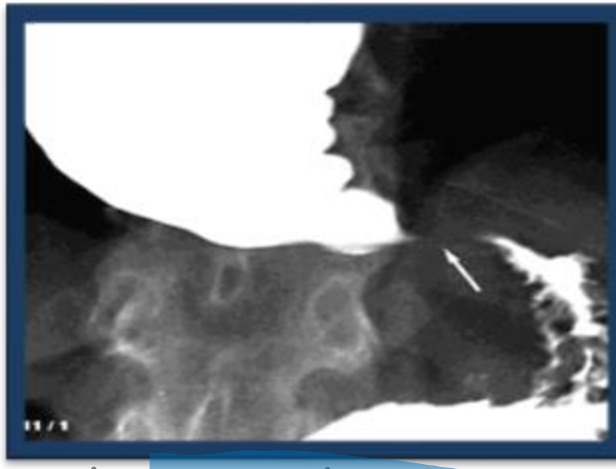
3- Surgical myotomy

4-Botulinum toxin injection(to relax lower esophageal sphincter)

Q: This patient presented with intermittent dysphagia.

What's your diagnosis?

Achalasia



Absolute criteria for diagnosis of Achalasia:

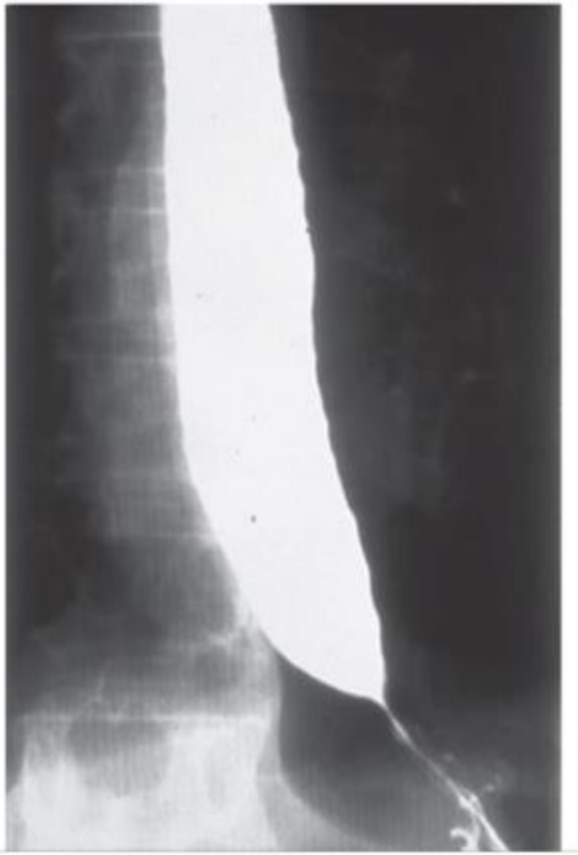
a. Incomplete relaxation of the LES

b. Aperistalsis of esophagus

Causes:

1. The majority are idiopathic.
2. In the United States, adenocarcinoma of proximal stomach is the second most common cause.
3. Worldwide, Chagas disease is an important cause

12. This patient presented with dysphagia for both solids and liquids, what is the cause and what is the diagnostic test for it?



- Achalasia
- Esophegeal manometry

Q: A pregnant woman comes with retrosternal sensation of burning associated with regurgitation of the food and chronic cough. What is this condition?

Gastroesophageal reflux disease (GERD)

What causes this condition?

1- Inappropriate relaxation of LES
2- Hypotensive LES
3- Decreased esophageal acid clearance
4- Impaired salivation
5- Hiatus hernia

What other differential diagnosis of this condition?

1- Esophagitis
2- Gastritis
3- Coronary Atery sclerosis
4- Irritable bowel syndrome
5- Esophageal cancer
6- Peptic ulcer disease

What complications could happen due to this condition?

1- Stricture formation
2- Chronic blood loss
3- Barrett's epithelium
4- Adenocarcinoma

What investigation should be done to diagnose this condition?

1- Barium esophagogram
2- Esophagogastroduodenoscopy
3- Esophageal manometry
4- Ambulatory 24-hour pH monitoring
5- Bernstein test
6- Barium Swallow

What other symptoms might come with this condition?

1- Esophageal

Dysphagia
Chest pain
Water brash
Nausea and vomiting
Belching
Hiccup

2- Extraesophageal

Recurrent pneumonitis
Nocturnal choking
Hoarseness of voice
Sore throat
Dental disease
Globus sensation

What are treatment of this condition?

1- Lifestyle modifications
2- Pharmacology (Antacids/ H2-blockers /PPIs)
3- Endoscopy therapy (Sterrata procedure / Entyrex / Gate keeper anti-reflux repair / Gastric placation)
4- Anti-reflux surgery

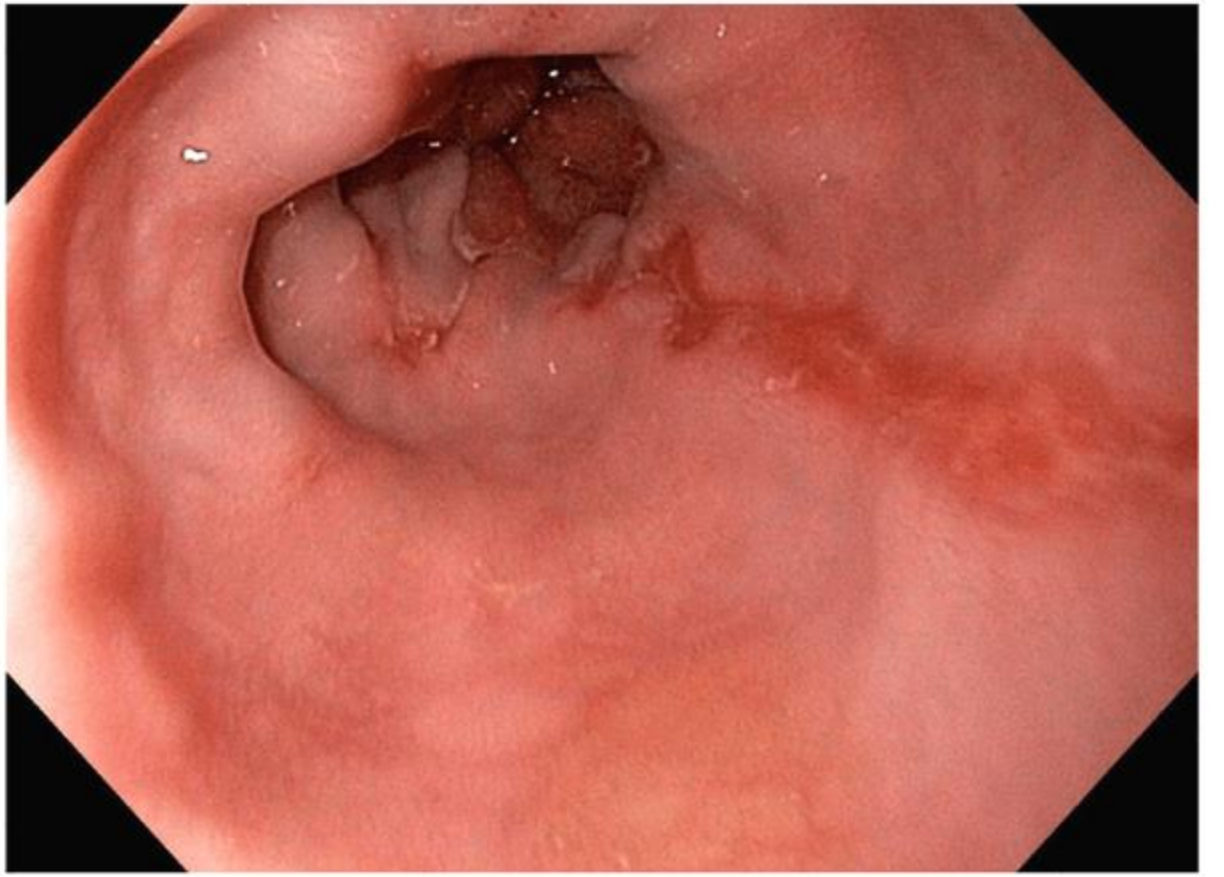
Name 3 common group of people to have this disease.

1) Pregnant women
2) Obese
3) Smokers

2. This patient came with regurgitation and heart burn, what is your diagnosis and treatment?

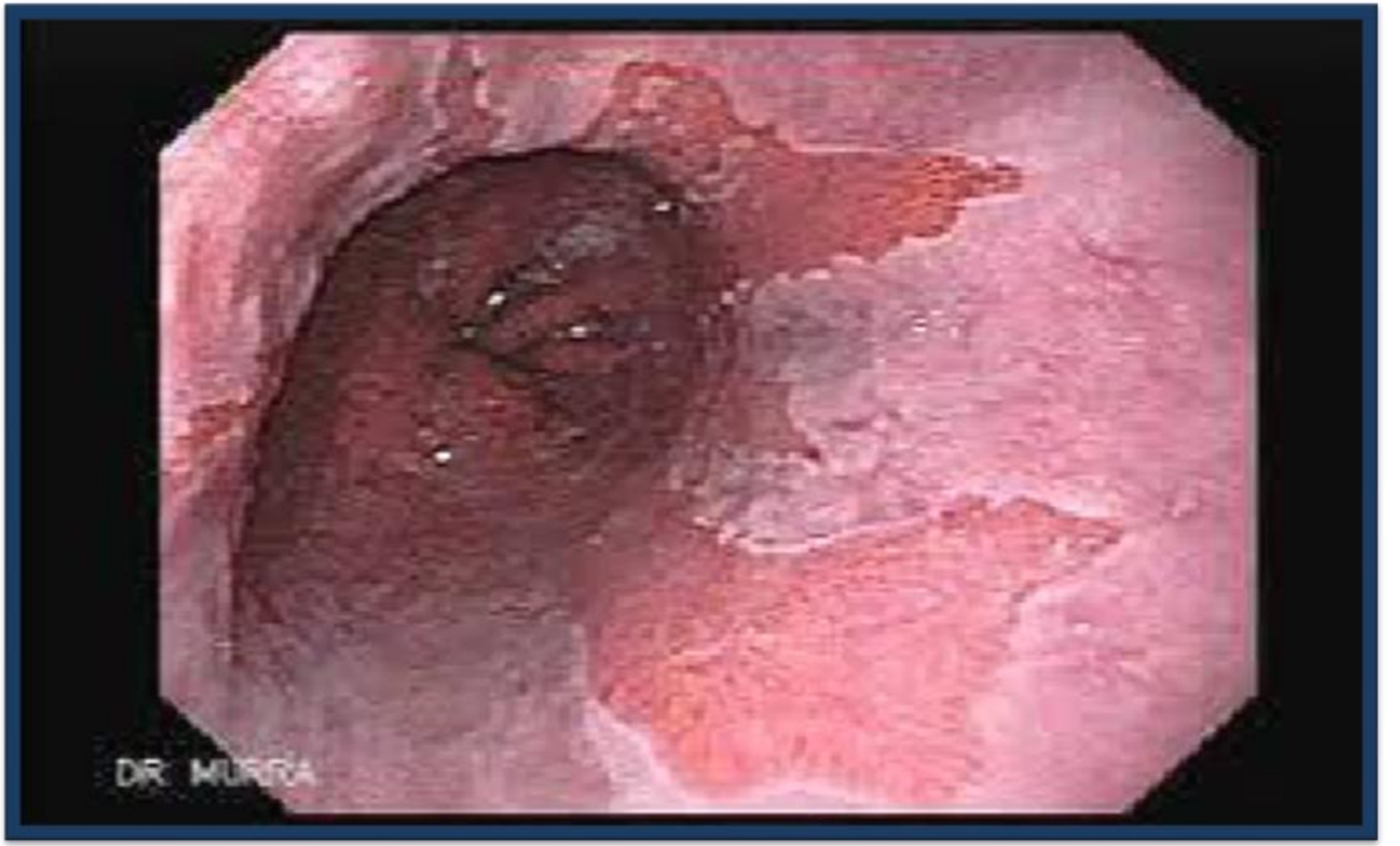
1.Esophagitiscaused by GERD

2. PPI



Q: This patient had GERD for 10 years, what's your diagnosis?

Barrett's esophagus



2.Peptic ulcer

What is peptic ulcer disease ??

break in the superficial epithelial cells penetrating down to the muscularis mucosa

Erosions: are superficial breaks in the mucosa alone

PUD devided into

1 – duodenal ulcer : most commonly PUD

2 – gastric ulcer : most commonly seen in lesser curviture

Causes of PUD

1 – H.pylori : most common cause of PUD

(spiral Gram-negative flagellate urease-producing bacterium)

2 – NSAIDs (can cause DU or gastric ulcer but mainly gastric ulcer)

3 – high acid state . Eg. ZES

4 – crohn's disease in stomach and duodenum

- Presentation :

1 – epigastric pain burning in nature

DU : pain increase when patient is hungery

GU : pain increase while patient eating

2 – anorexia and weight loss may occur espicially with GU

3 – nausea

4 – vomiting : less frequent , but when occur it relieve pain

5 – UGI bleeding or perforation : may occur without preceding any symptom

6 – weight gain with DU and weight loss with GU

Diagnosis of H.pylori infection

*non-invasive teste

1- 13C-urea breath test :most sensetive non invasive test

is suitable for testing for eradication of the organism

False –ve if patient on PPI

2 – fecal antigen test : patient should be off PPI(2-4 weeks) but can be continue on H2 blockers

3 – serological test : detect IgG antibodies , Non suitable for testing for eradicatin or presence of current infection because it still be positive after one yeare of eradication

* if you suspected ZES do fasting serum gastrin level

*Invasive tests (endoscopic)

1 – biopsy urease test : false –ve if patient on PPI or antibiotics

2 – culture

3 – histology : from mucosa of antrum and fundic body

- Patient under 55 years with typical symptoms of PUD and +ve H.pylori can start eradication therapy without more investications
- Endoscopy is gold standard for diagnosis of PUD
- All GU most be biopsied to exclude gastric Ca espically in elderly paeints
- Endoscopy is required in all patients with alarm symptoms:

1. Dysphagia or odonophagia
2. protracted vomiting
3. Anorexia and weight loss
4. hematemesis or melena
5. persistent symptoms despite of treatment
6. abnormal barium swallow or CT
7. Family history of PUD or gastric malignancy
8. older patient
9. Early staiety
10. IDA

Treatment of non bleeding PUD by 3 main strategies

1 – treat H.pylori

2 – decreast acid secretion

3 – stop exacerbating processes(smoking , NSAIDs)

* don't use one drug alone

therapy always shoud contain 2 antibiotics and poweful acid suppression agent

* start trible therapy

(clarithromycin 500 mg + amoxacillin 1g + omeprazole 20 mg) all twice daily from 1-2 weeks …… or

(clarithromycin 500 mg + metronidazole 400 mg + omeprazole 20 mg) all twice daily from 1-2 weeks

if triple therapy eradication failed … start with quadrable therapy

(bismuth chelate 120 mg 4 times daily + metronidazole 400 mg 3 times daily + tetracyclie 500 mg 4 times daily + omeprazole 20 mg 3 times daily)

- All patients with duodenal and gastric ulcers and positive H. pylori testing should have H. pylori eradication therapy
- Gastric ulcer not associated with H.pylori … treated by PPI and misoprostol for 3 months

- The effectiveness of treatment for uncomplicated duodenal ulcer should be assessed symptomatically
- If symptoms persist, breath or stool testing should be performed to check eradication
- Patients with a risk of bleeding or those with complications, i.e. hemorrhage or perforation, should always have a 13C urea breath test or stool test for H. pylori 6 weeks after the end of treatment to be sure eradication is successful
- Complications of PUD

1 – hemorrhage

2 – perforation : more common in DU

3 – gastric outlet obstruction

- Surgical treatment now only used for complications including:

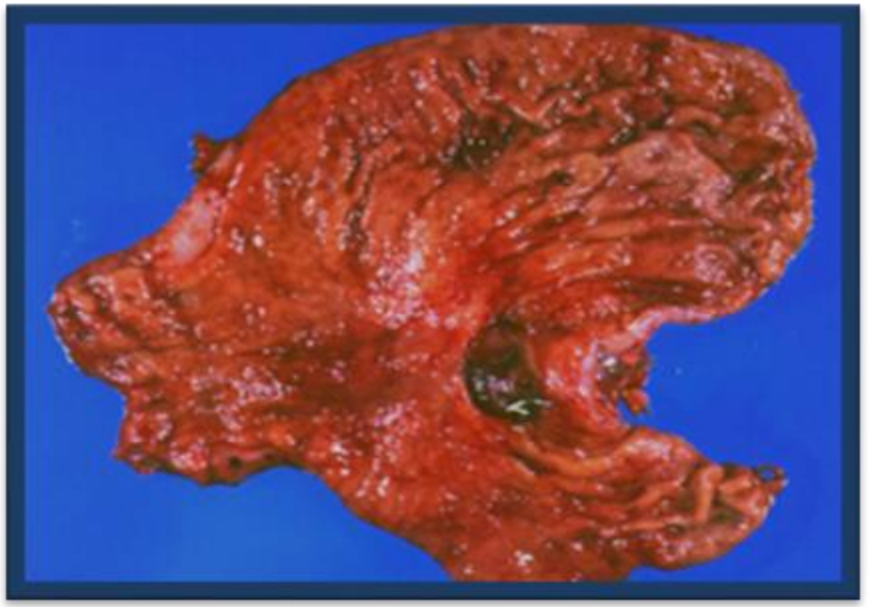
1. recurrent uncontrolled hemorrhage where the bleeding vessel is ligated
2. Perforation

- Long-term complications include

1. Recurrence of ulcers
2. Dumping
3. Diarrhea
4. Nutritional deficiencies: Iron, Folate, Vitamin B12

Q: A 40 years old male patient complaint of epigastric pain and vomiting for 3 weeks duration. The pain never relieved, aggravated by taking a meal. He noticed that his is weight slightly decrease. After done the proper investigation, the result shows below. What is this condition from this picture?

Gastric Ulcer



Name the most common site to found this condition for this organ.

Lesser curvature of stomach

What are the most important signs and symptoms for this condition?

1) Epigastric pain
2) Pain aggravated by taking a meal/food
3) Vomiting (patient like to self induced vomiting)
4) Lose weight (afraid to eat 3shan al-pain)
5) Hematemesis/Melena

Give 2 ways to diagnose this condition.

1) invasive test by Endoscopy
2) take biopsy (to exclude malignancy)

Name 2 maneuvers to diagnose the microorganism cause this disease.

A) Invasive maneuver:
   1) Endoscopic biopsy
   2) Rapid urase test
   3) Culture

B) Non-invasive maneuver:
   1) Urea breath test
   2) Fecal Ag test

What are the complications of this disease?

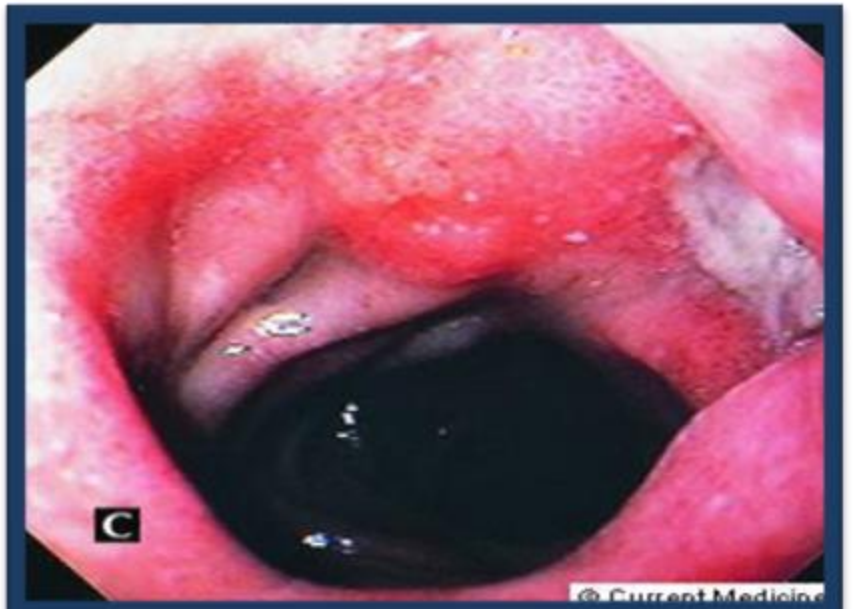
1) Hemorrhage
2) Perforation
3) Gastric Outlet Obstruction

What are the treatment for this condition?

1) H2-blockers e.g. Cimetidine/ Ranitidine
2) Antacids
3) PPI e.g. Omeprazole/ Tenatoprazole
4) Prostaglandins
5) if H.pylori positive start with eradication therapy
(clarithromycin 500 mg + amoxacillin 1g + omeprazole 20 mg) all twice daily from 1-2 weeks

Q: A 35 years old male patient complaint of epigastric pain for 3 weeks duration. The pain relieved by food and antacids,. aggravated while the patient is hungry. He noticed that his weight increase, recently. After done some investigations, one of the results showed below. What is the disease from this picture?

Duodenal Ulcer



Name the most common site to found this condition in this organ.

1st part of duodenum

What are the risk factors for this condition?

1) Genetics (in Gastric Ulcer plays no role)
2) Smoking
3)NSAIDs

What are signs and symptoms of this condition?

1) Epigastric pain
2) Pain reoccur 3 hours after eating
3) Relieved by food and antacids
4) Aggravated while hungry
5) Gain weight

Give 2 ways to diagnose this condition.

1) Endoscopy
2) take biopsy (to exclude malignancy)

What are the complications of this disease?

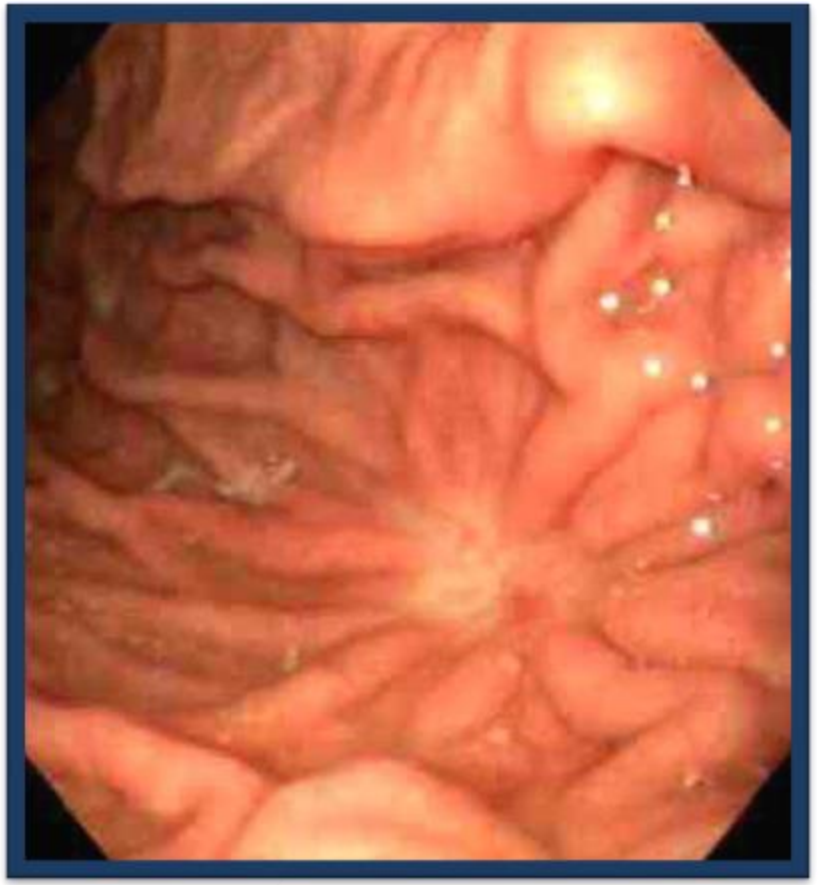
1) Hemorrhage (the worst)
2) Perforation
3) Gastric Outlet Obstruction

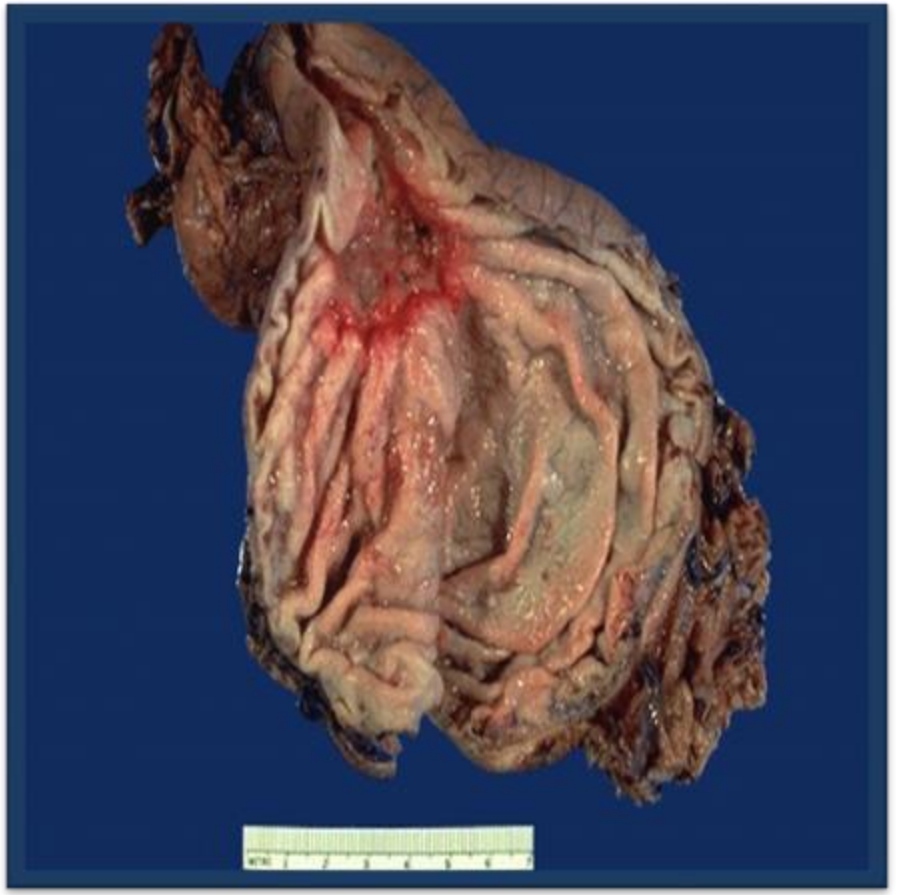
What are the treatment for this condition?

1) H2-blockers e.g. Cimetidine/ Ranitidine
2) Antacids
3) PPI e.g. Omeprazole/ Tenatoprazole
4) Prostaglandins
5) if H.pylori positive start with eradication therapy
(clarithromycin 500 mg + amoxacillin 1g + omeprazole 20 mg) all twice daily from 1-2 weeks

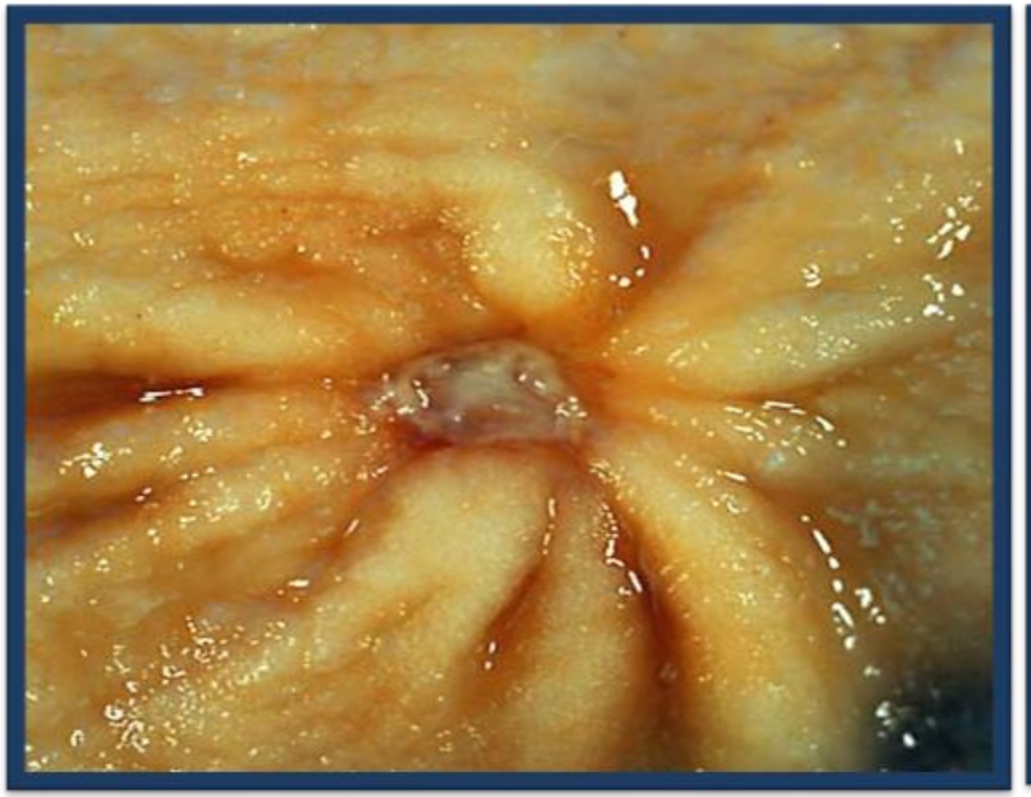
*Some pictures to differentiate between gastric and duodenal ulcer.

Gastric Ulcer



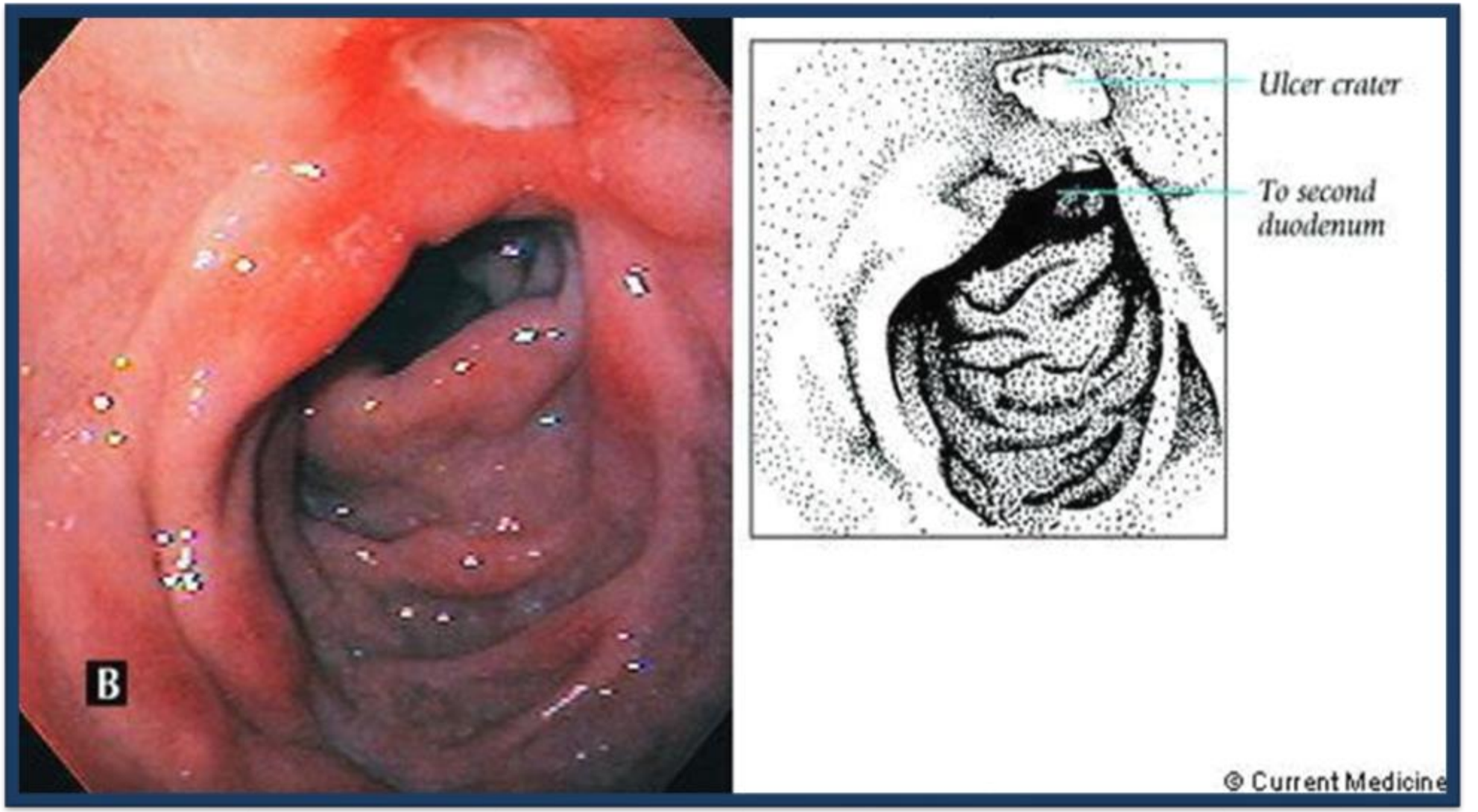


Gastric Ulcer

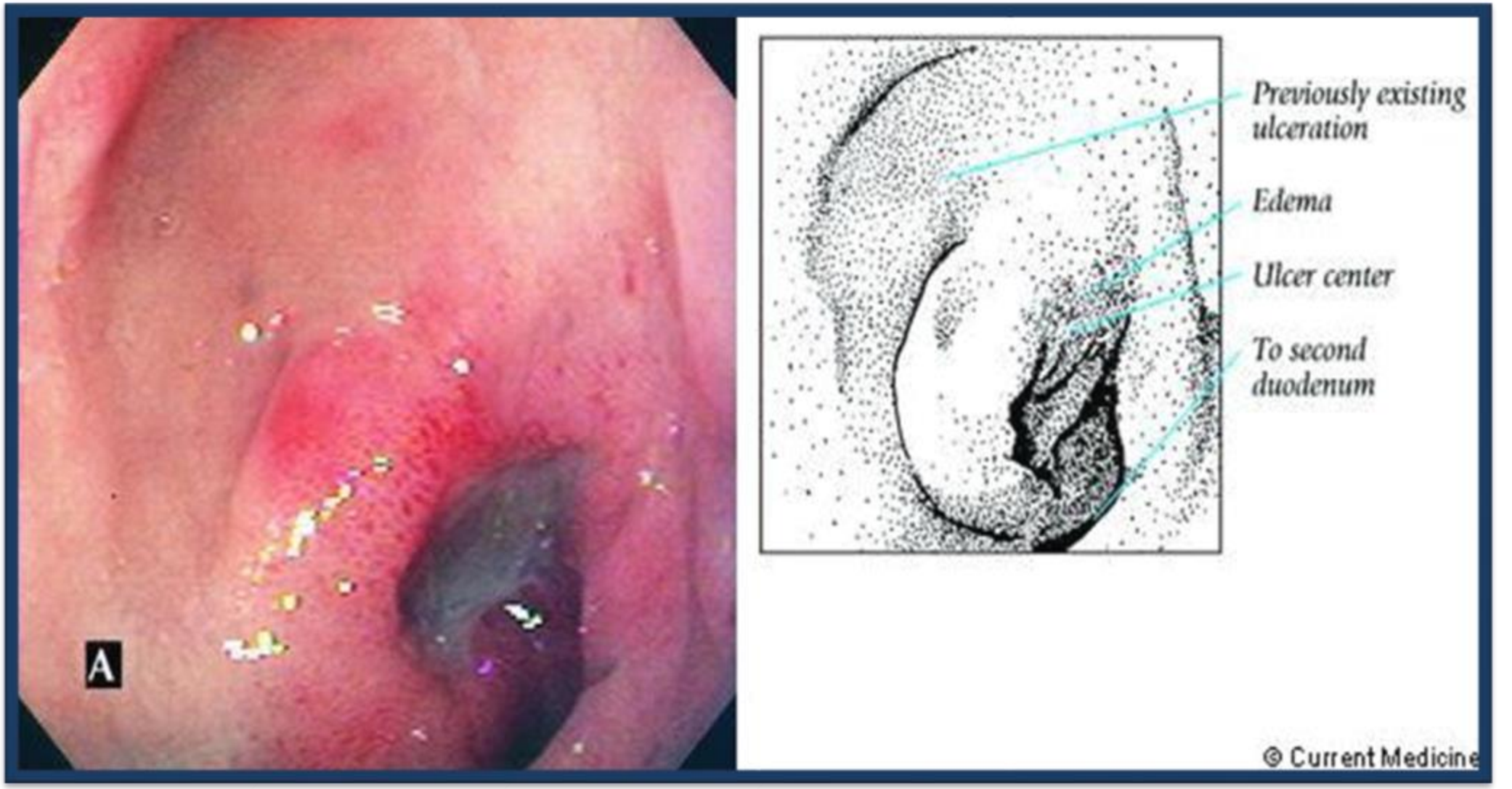


The picture can't be displayed.

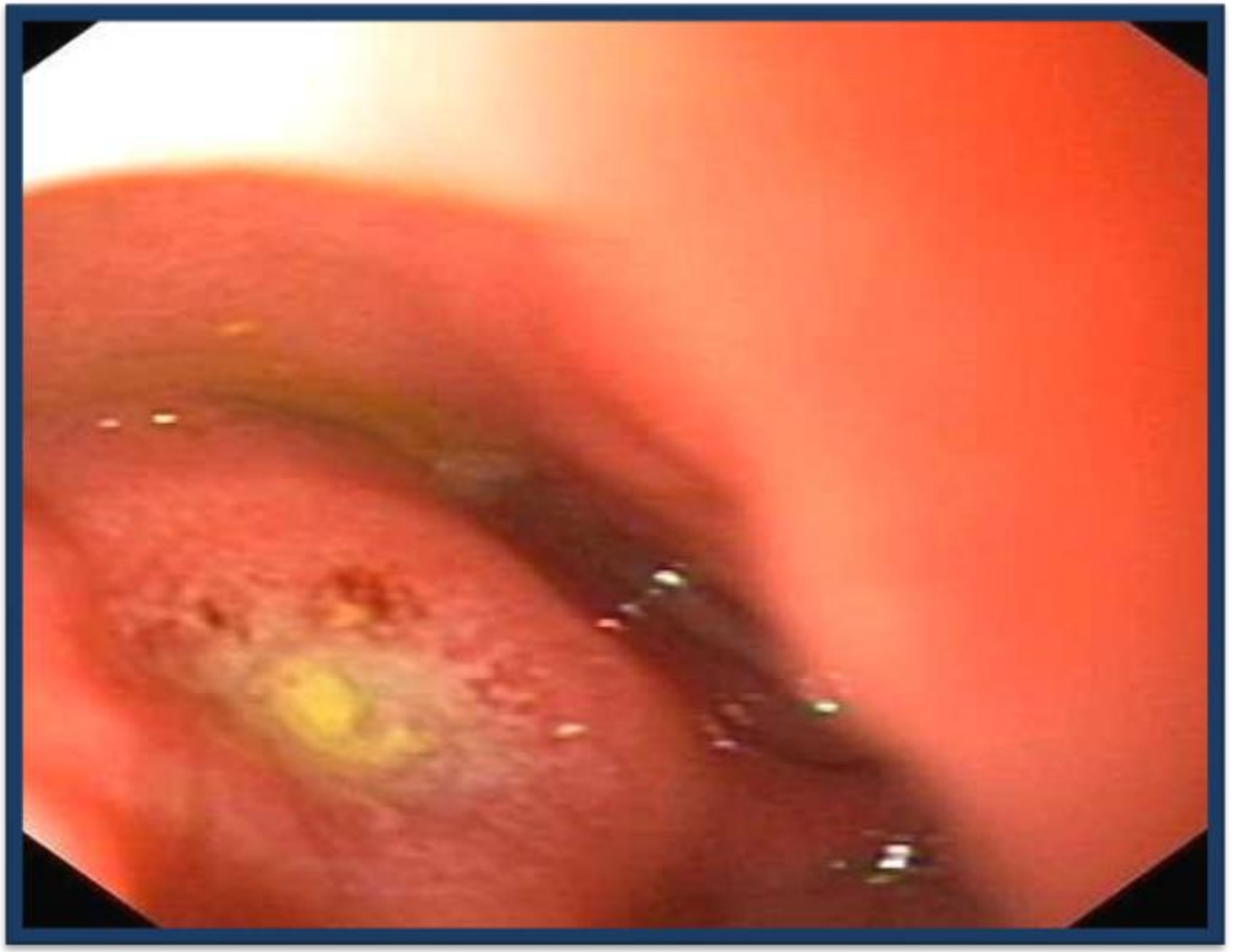
Duodenal Ulcer



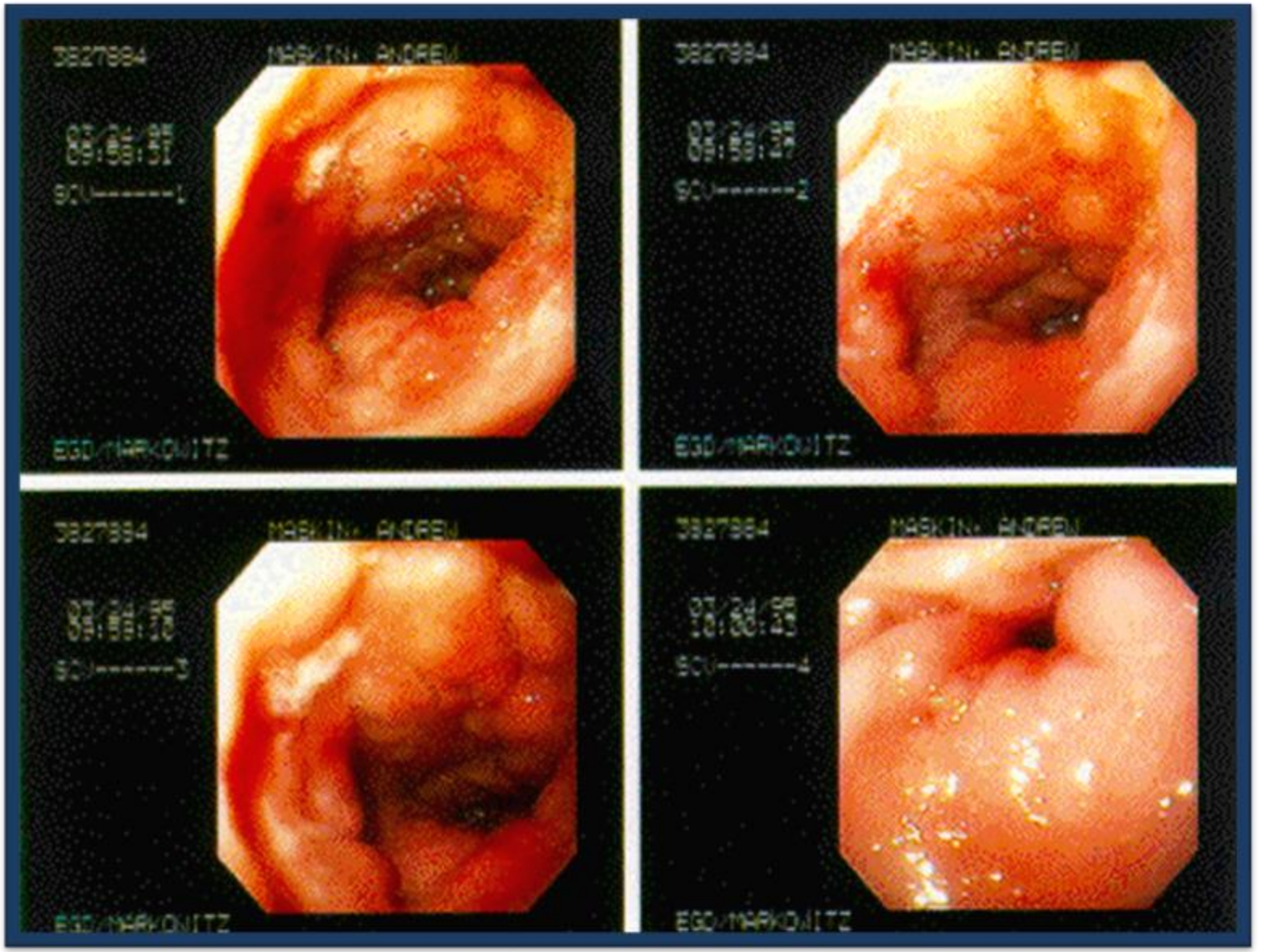
Duodenal Ulcer



Duodenal Ulcer



Duodenal Ulcer



3.Liver Cirrhosis

A-What is the finding?

Palmar Erythema

B-Mention two causes.

Thyrotoxicosis,

Liver Cirrhosis causes

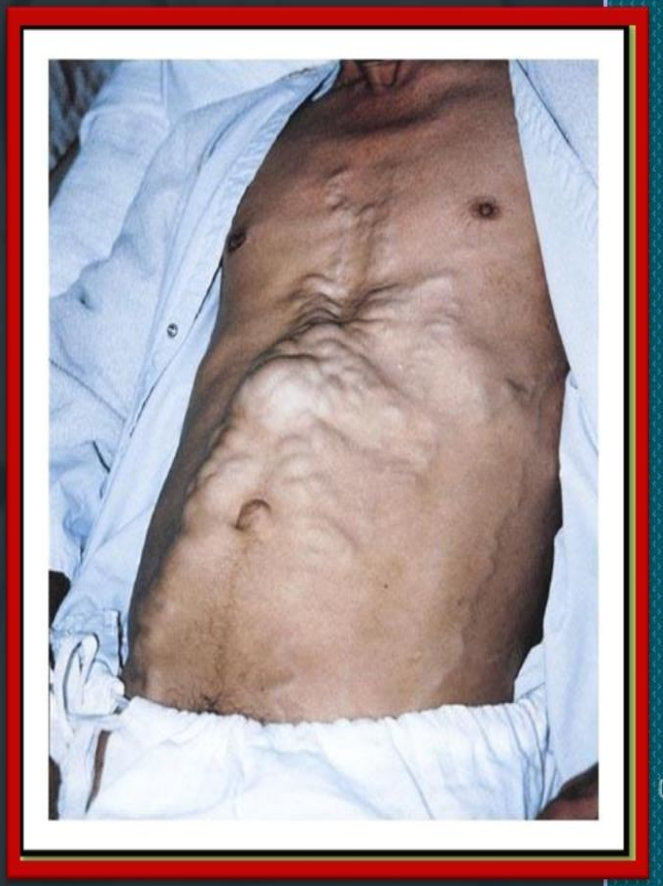
)alcoholic liver disease, chronic hepatitis B AND C, methotrexate, autoimmune hepatitis, primary biliary cirrhosis (,

Pregnancy

1. Give the cause of this condition?

Portal hypertension

2. name this pathology?



Caput medusa.

Causes of PHT

SUPRAhepatic

cardic disease

Hepatic vein thrombosis

Inferior venacava thrombosis

Hepatic cause

Liver cherosis

Posthepatic portal vein thrombosis

Name this physical finding in a patient with portal hypertension and spider Naevi.

Palmer Erythema,

Signs of chronic liver disease

1scites

2 Varices

3 GynecomastiA

4 Palmer Erythema ,

5 Hemorrhoids

6 Caput medusa

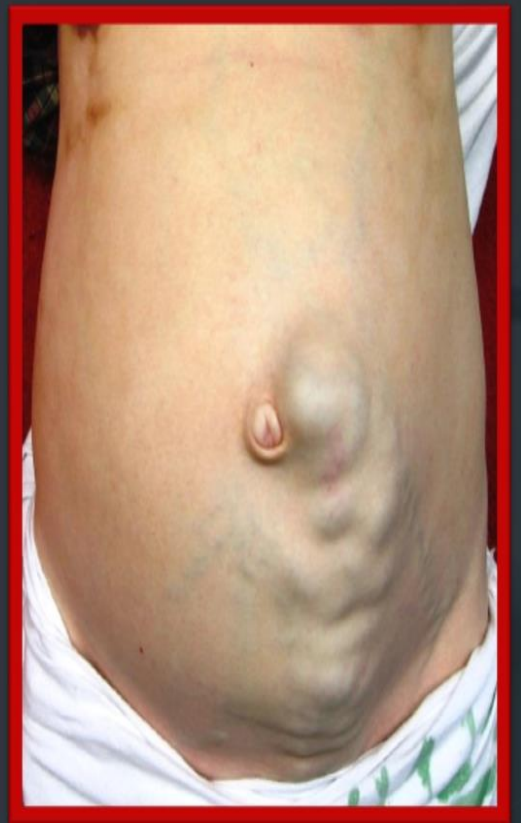
Pt with cirrhosis .

- What the most imp. Organomegaly you look for in examination ?

Splenomegly .

- What is the technique you do if you can't feel it?

abdominal ultrasound(my answer)/some answered it: tapping on the lower left ribs .



This patient has chronic liver disease.

Name 2 visible abnormal findings on his abdominal inspection.

Ascites
Dilated veins
Inverted umbilicus0

Complication of this disease

portal HTN

Varices

Ascites

Hepatic encepbalopathy

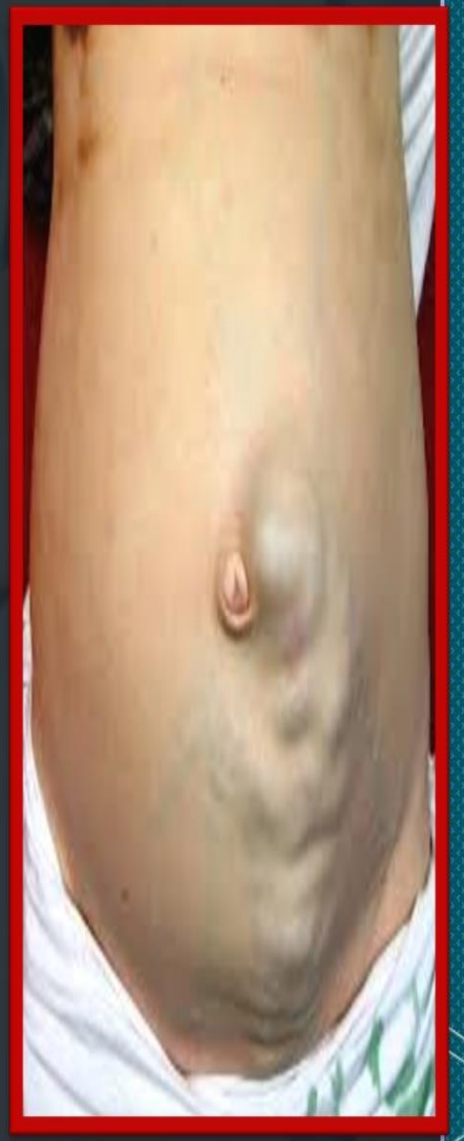
Hepatorenal syndrome

Infectedascitic fluid

Hyperestrinism

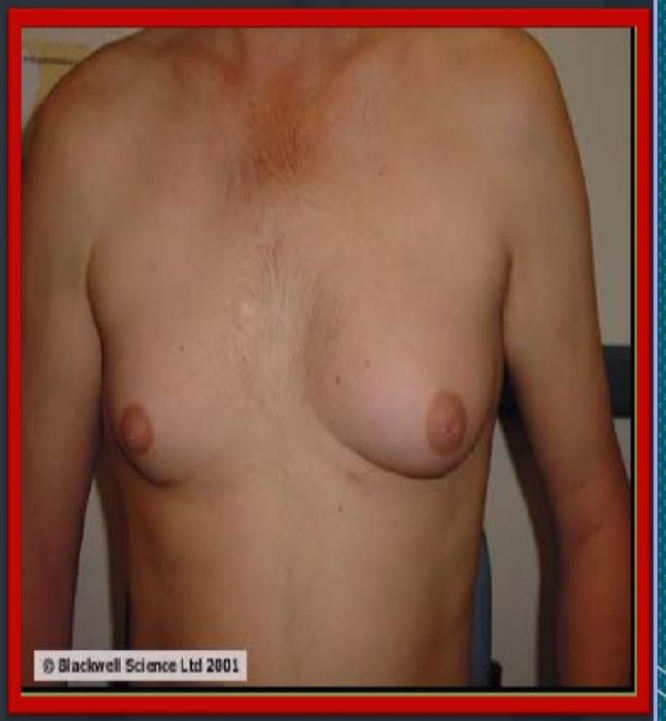
Coagulopathy

Hepatocellular carcinoma



Pt with CHRONIC hepatitis B. what is the cause of this picture?

liver cirrhosis



An endoscopy was done for a patient withliver cirrhosis and showed the following.

A-What is the diagnosis?

Esophageal varices

B-Mention a line of management

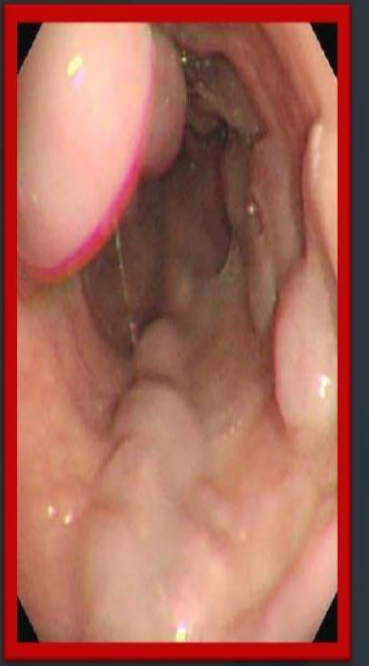
Esophageal band ligation

Endoscopic sclerotherapy .

Iv vasopressin

IV octreotide

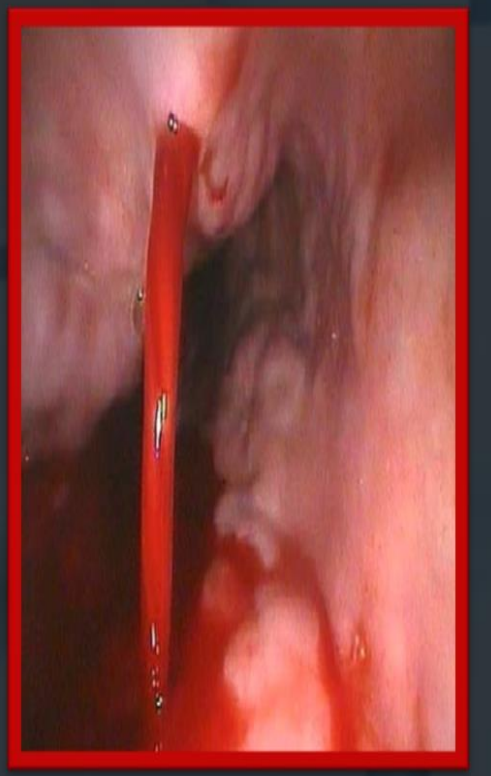
TIPS



This patient presented with massive hematemesis. This is the picture of his endoscopy. What's your diagnosis?

Esophageal varices

They are graded according to their size, as follows: Grade 1 – Small, straight esophageal varices. Grade 2 – Enlarged, tortuous esophageal varices occupying less than one third of the lumen. Grade 3 – Large, coil-shaped esophageal varices occupying more than one third of the

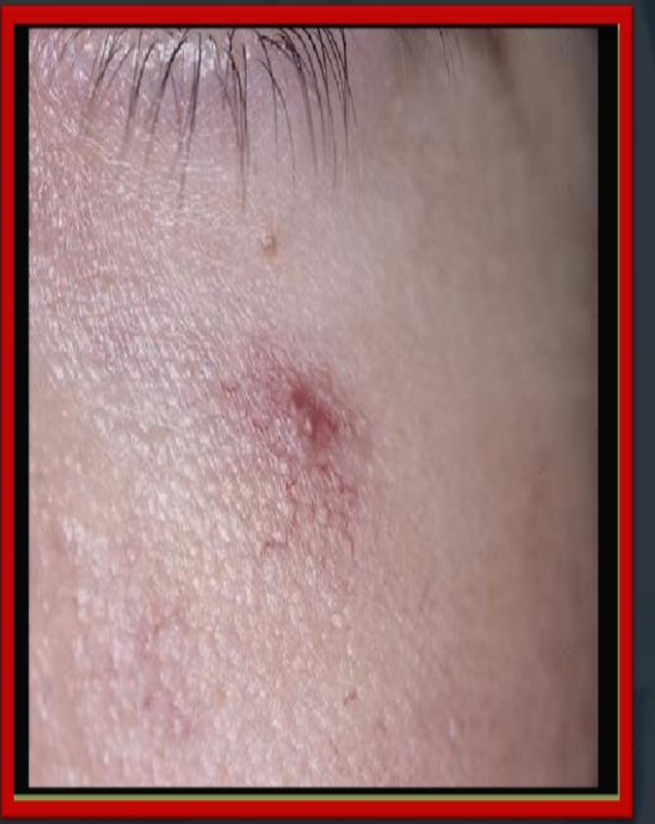


Mention the endoscopic finding for this patient?

Esophageal varices.

2 Minute Medicine®	Child-Pugh Score		2minutemedicine.com
Factor	1 point	2 points	3 points
Total bilirubin (μmol/L)	<34	34-50	>50
Serum albumin (g/L)	>35	28-35	<28
PT INR	<1.7	1.71-2.30	>2.30
Ascites	None	Mild	Moderate to Severe
Hepatic encephalopathy	None	Grade I-II (or suppressed with medication)	Grade III-IV (or refractory)
	Class A	Class B	Class C
Total points	5-6	7-9	10-15
1-year survival	100%	80%	45%

Table 1. Child-Pugh score.



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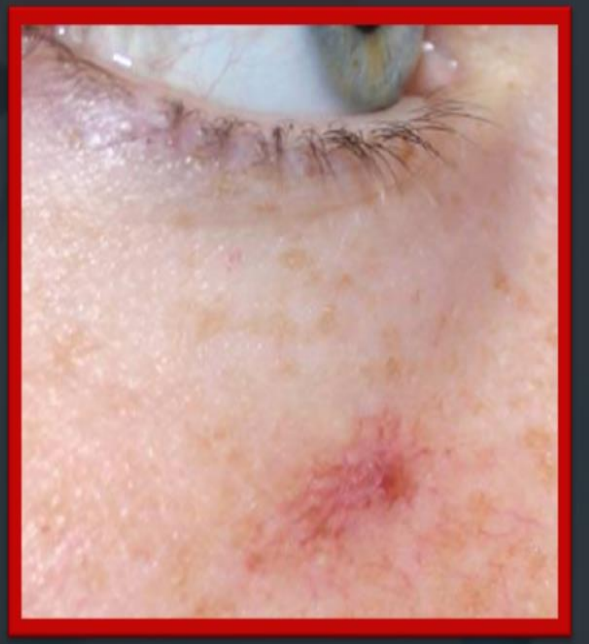
	Class A	**Class B**	**Class C**
Total points	5-6	7-9	10-15
1-year survival	100%	80%	45%

Table I. Child-Pugh score.

Patient pfeaturesd with agitation & confusion, now he comes complaining of Hematemesis, on endoscopy he has bleeding varices .
What is the cause of his confusion?
Hepatic encephalopathy.

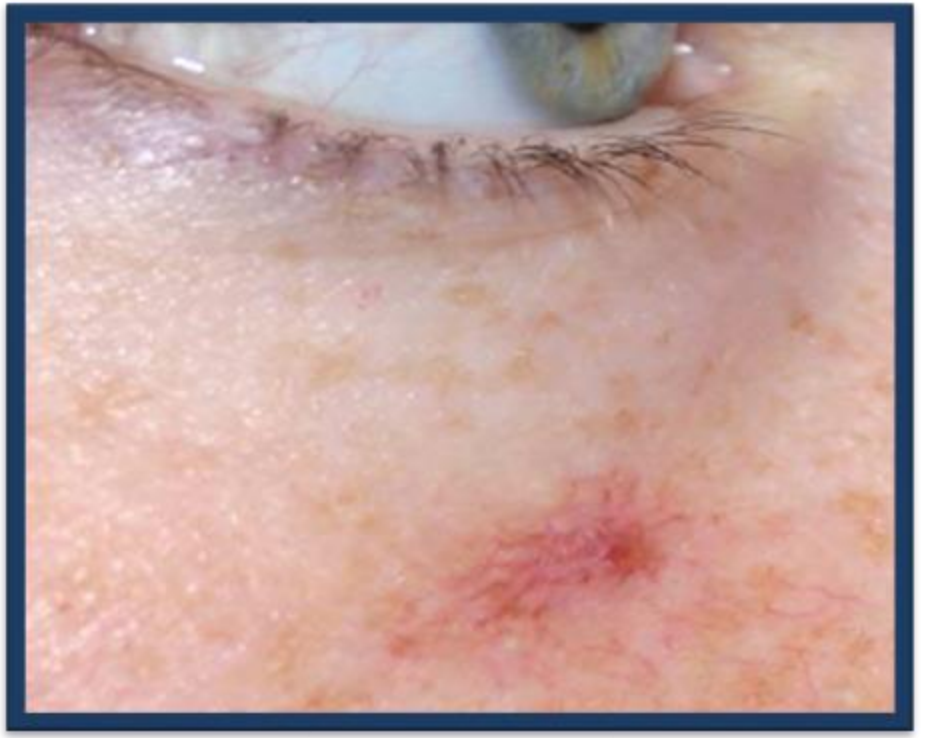
Precipitants (alkalosis, hypokalemia, GI bleeding, hypovolemia) .

Clinic al feature
dcreased mental function
asterixis.
Rgidity, hyperreflexia.
Fetor Hepaticus
Tretmant.
Lactulose
rifaximin.
Dite limit protine to 30g/day.



Q: Patient presented with agitation & confusion, now he comes complaining of Hematemesis, on endoscopy he has bleeding varices . What is the cause of his confusion?

Hepatic encephalopathy.

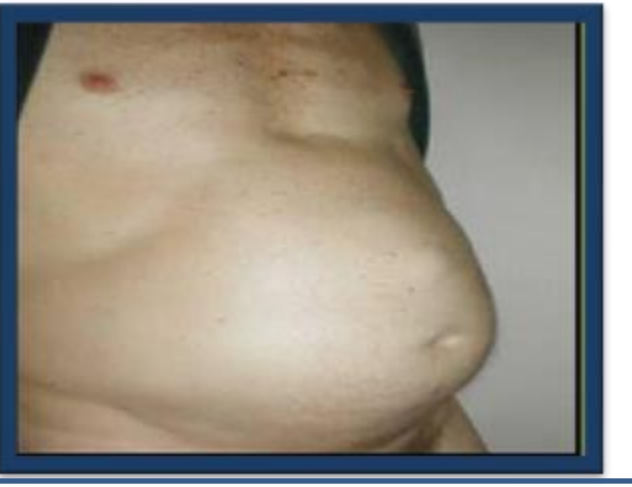


Q: Pt with liver cirrhosis & ascites , presented with fever & abdominal pain , P/E shows rigid abdomen, what is the most likely Dx?

Spontaneous bacterial peritonitis (SBP) .

How to confirm?

Diagnostic paracentesis .



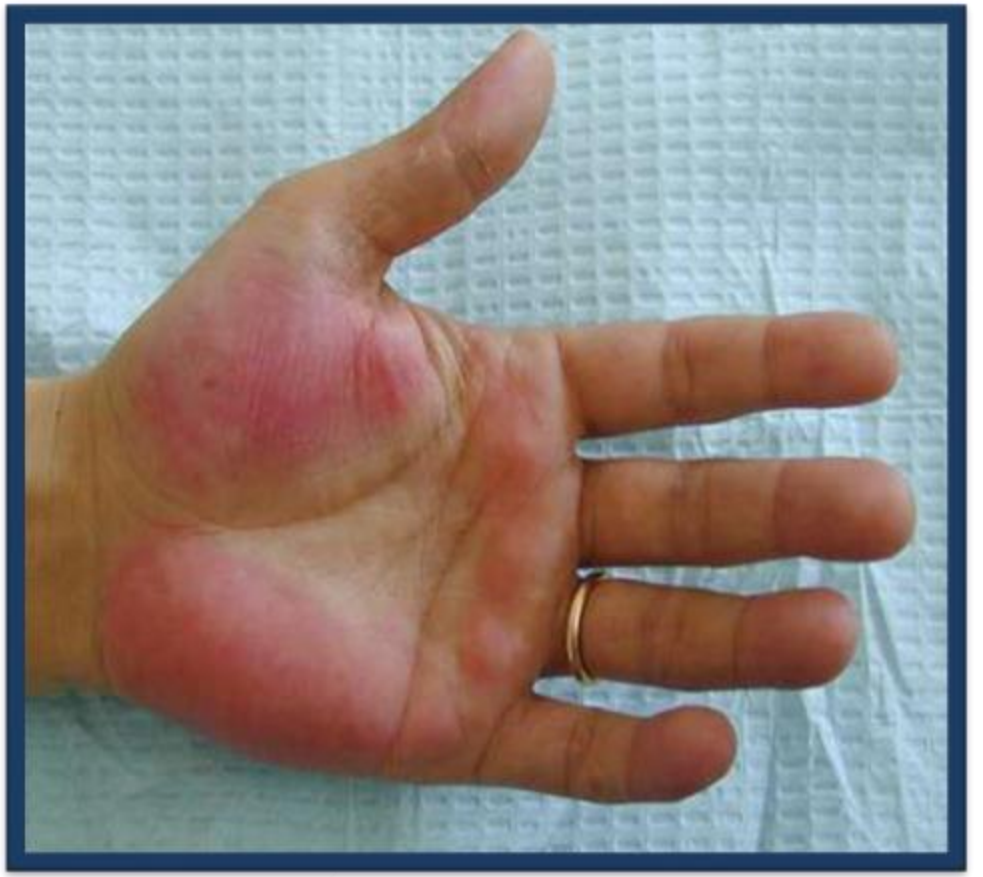
*SBP occur in 20% of pts hospitalized for ascites
M.C organism is E.COLI
*High mortality rate (20-30%)
*High recurrence rate (up to 70% in the first year)

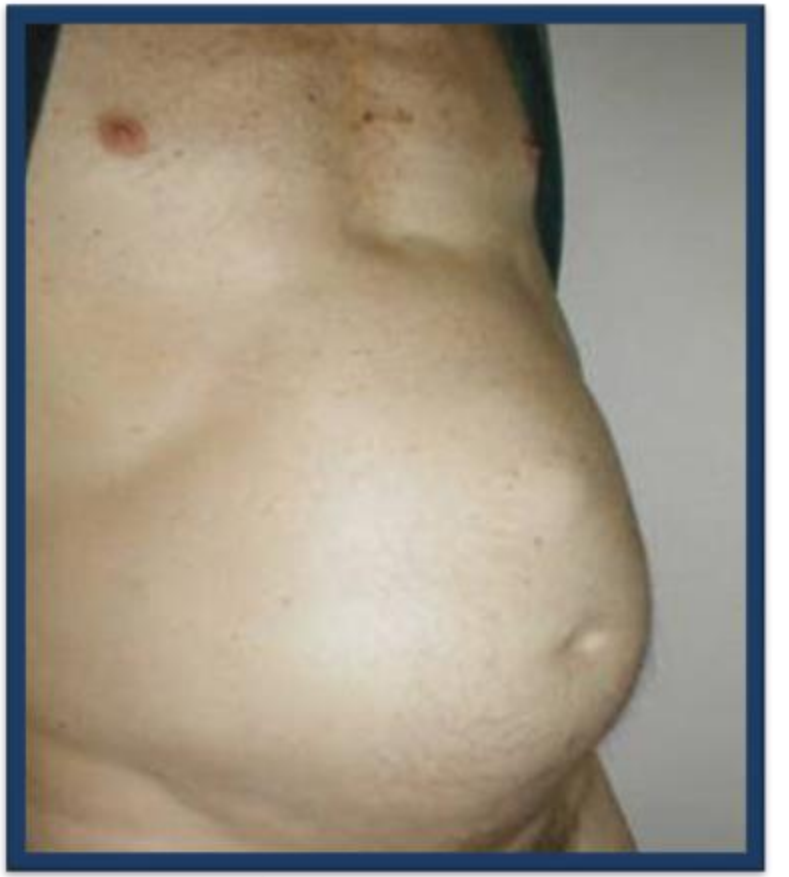
Q: A- what do you expect to see by gastroscope?

esophageal varices.

B- what's the cause of distended abdomen

due to PORTAL HTN (increased hydrostatic pr.) and HYPOALBUMINEMIA (decreased oncotic pr.)



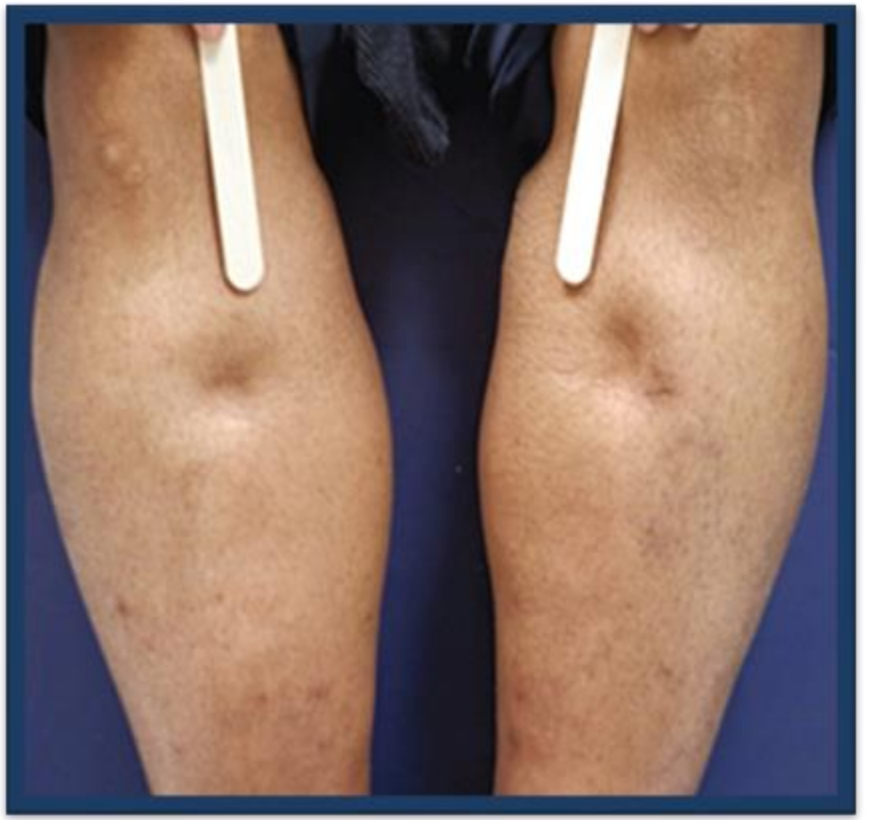


Q: 1.What is your spot dx?

Bilateral lower limb pitting edema

2. Name two conditions associated with this.

Nephrotic syndrome, liver cirrhosis, right heart failure



Q: Mention 4 causes of this condition.

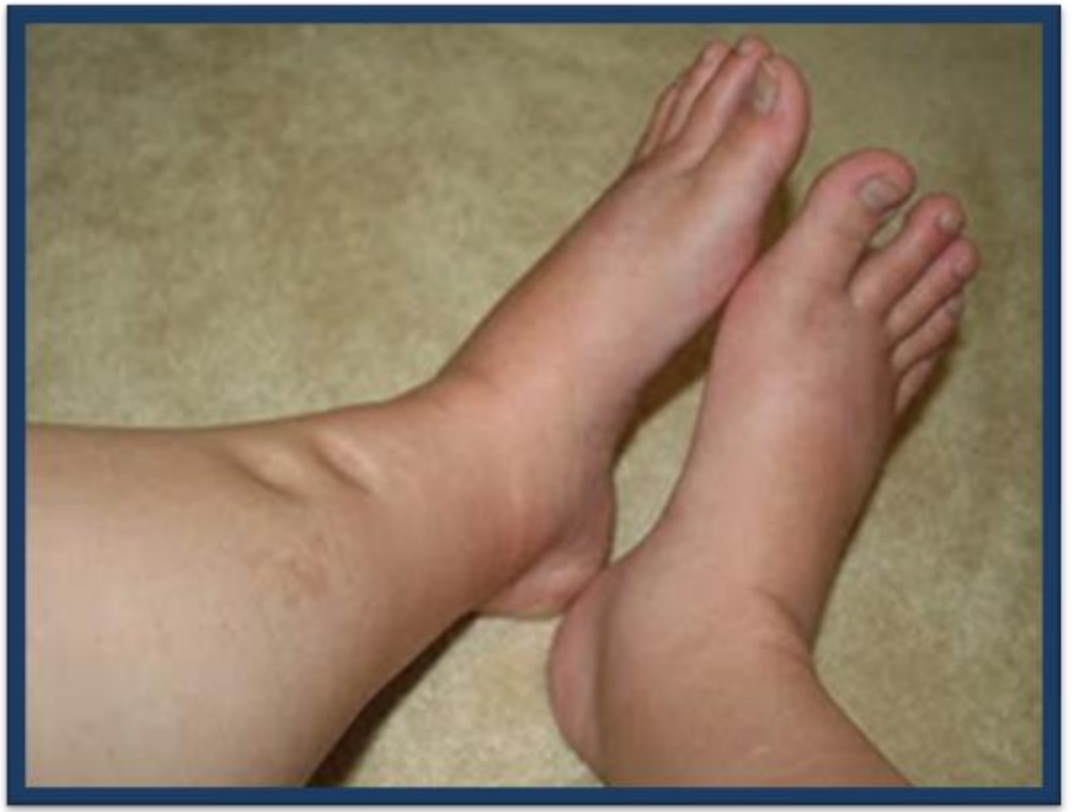
Heart failure

Renal failure, Nephrotic syndrome

Liver cirrhosis

Hypo-albuminemia

Fluid overload

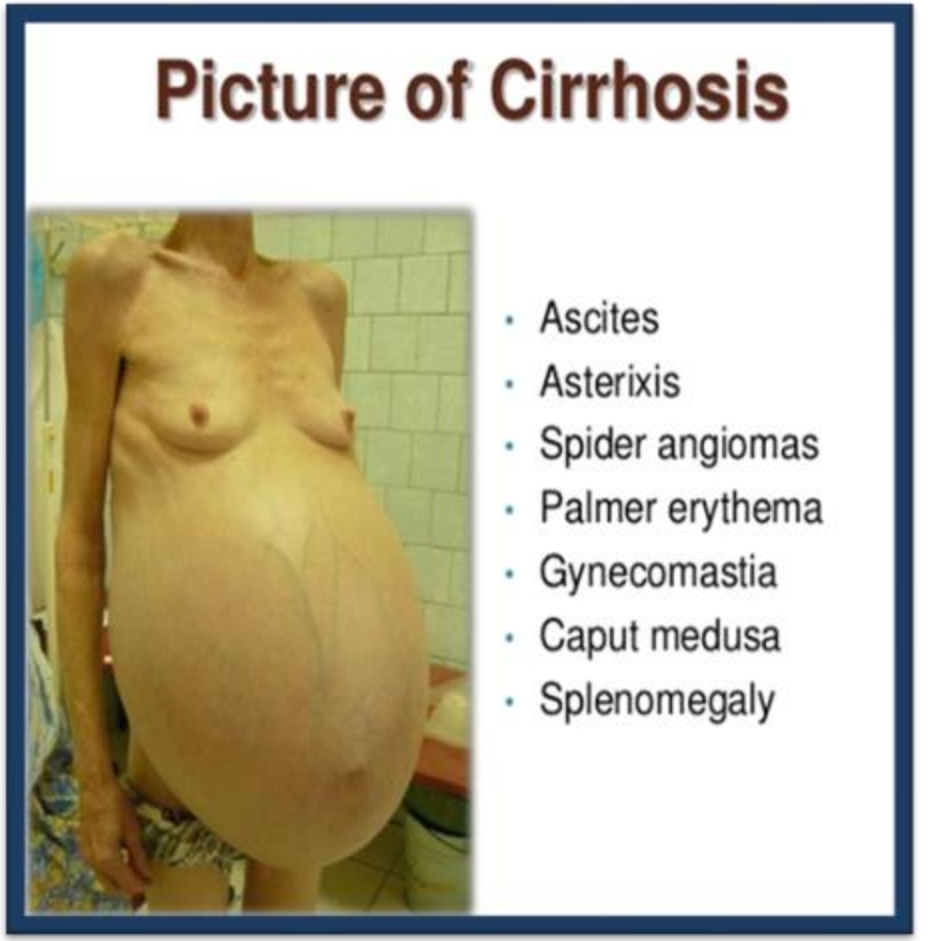


Q: Name three sign seen in this picture.

Ascites, dilated veins, gynecomastia

What is your spot dx?

Liver cirrhosis

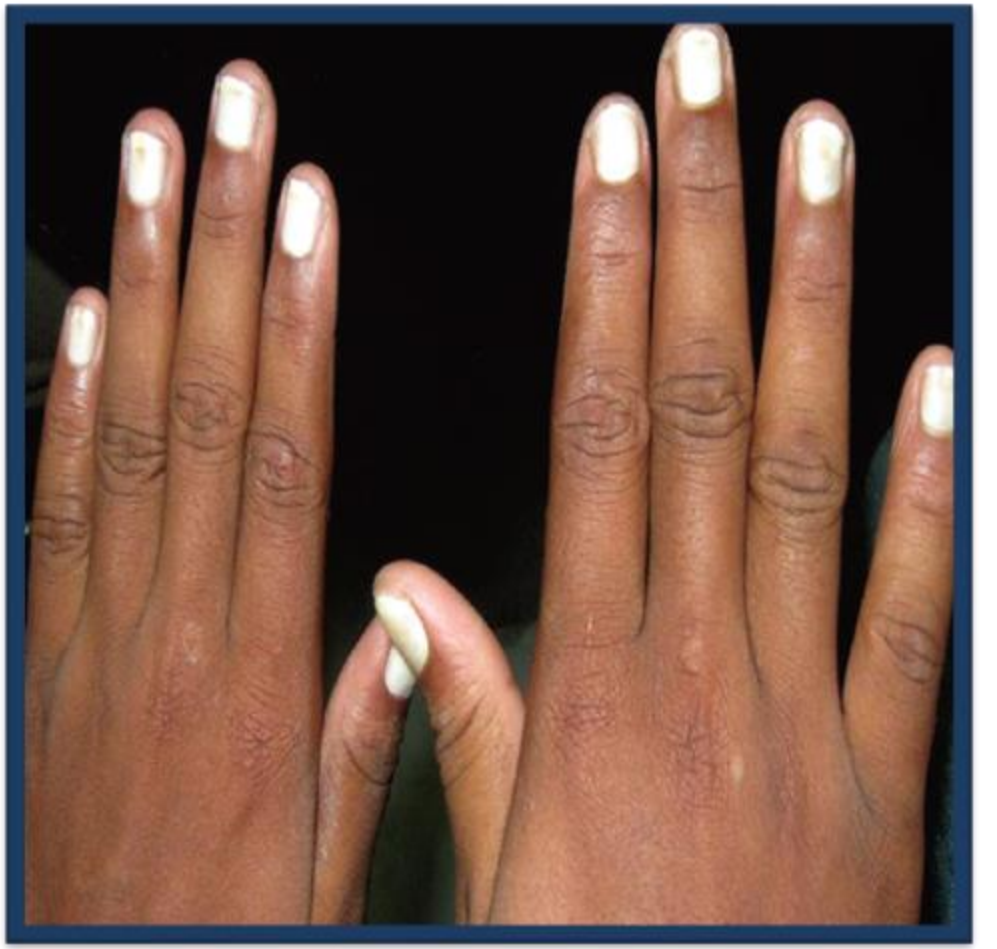


Q:What is your finding?

Leukonychia

What blood test would you order?

serum albumin level



Q:Mention 3 causes of this condition.

CLUBBING

- G: GI malabsorption $ (Celiac disease)
- N: Neoplasm (Lung Ca)
- I: Infective endocarditis
- B: Biliary cirrhosis
- B: Birth defects
- U: Ulcerative colitis (IBD)
- L: Lung disease
  - Abscess
  - Bronchiactasis
  - Cystic Fibrosis
  - Dont say COPD
  - Emphysema
  - Fibrosis / Fibroma
- C: Cyanotic Conginital Heart disease

4.IBD

Introduction

* As you know, IBDs have GI and extra-GI manifestations and I will try to include them all in this presentation. We will start with typical GI presentations of IBDs then the extra-GI manifestations. After that I will put the common complications of the IBDs.

Let's begin…

Clinical Features of IBD

1-Diarrhea

2-Rectal bleeding (more in UC)

3-Abdominal pain/Cramps

4-Tenesmus (rectal dry heaves)

5-Fever

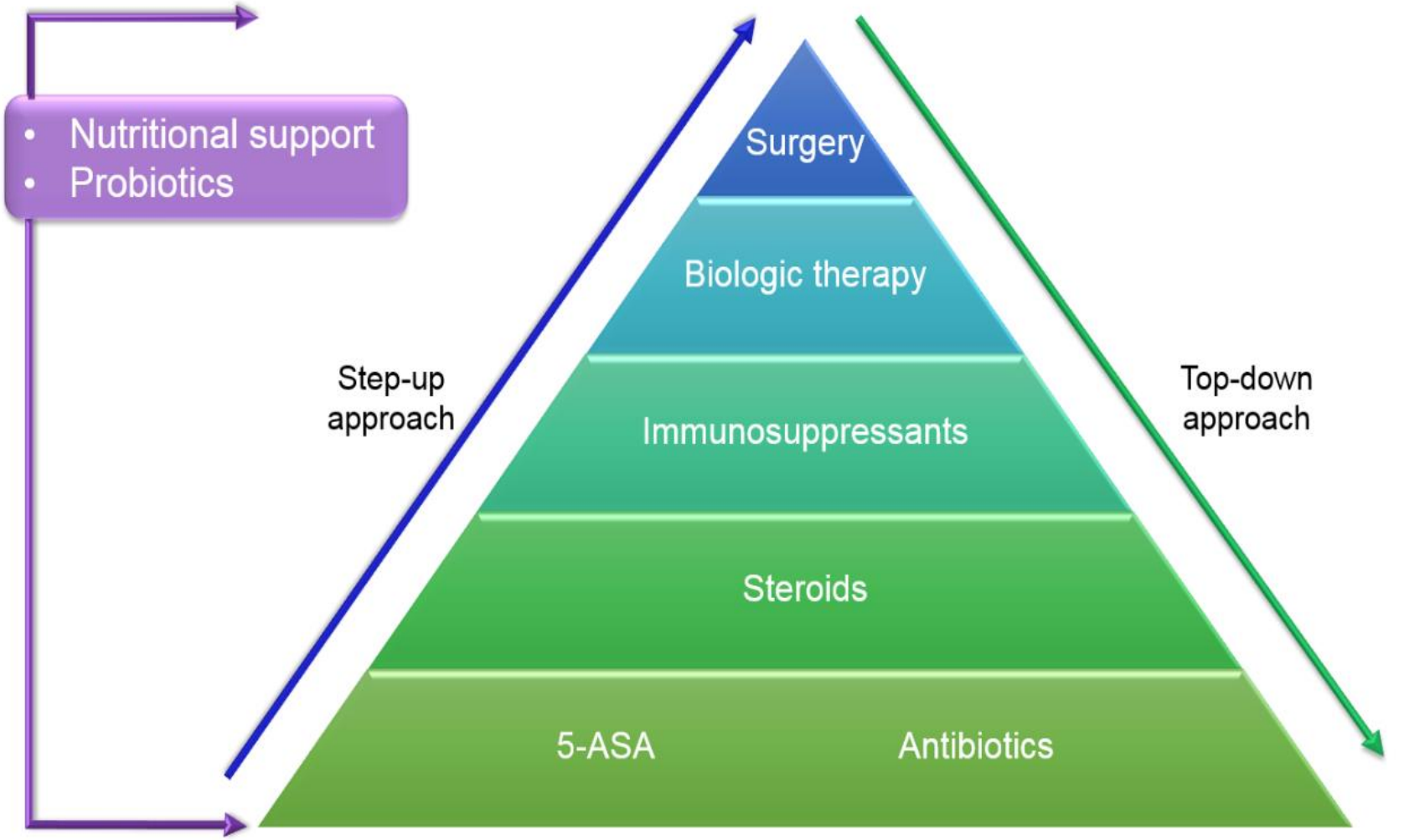
6-Weight loss

7-Vomiting

8-Muscle spasm

	ULCERATIVE COLITIS (UC)	CROHN DISEASE
Wall Involvement	Mucosal and submucosal ulcers	Full-thickness inflammation with knife-like fissures
Location	Begins in rectum and can extend proximally up to the cecum (involvement is continuous, Fig. 10.21A); remainder of the GI tract is unaffected.	Anywhere from mouth to anus with skip lesions; terminal ileum is the most common site, rectum is least common.
Symptoms	Left lower quadrant pain (rectum) with bloody diarrhea	Right lower quadrant pain (ileum) with non-bloody diarrhea
Inflammation	Crypt abscesses with neutrophils (Fig. 10.21B)	Lymphoid aggregates with granulomas (40% of cases)
Gross Appearance	Pseudopolyps; loss of haustra ('lead pipe' sign on imaging, Fig. 10.21C)	Cobblestone mucosa (Fig. 10.22A), creeping fat, and strictures ('string-sign' on imaging, Fig. 10.22B)
Complications	Toxic megacolon and carcinoma (risk is based on extent of colonic involvement and duration of disease; generally not a concern until > 10 years of disease)	Malabsorption with nutritional deficiency, calcium oxalate nephrolithiasis, fistula formation, and carcinoma, if colonic disease is present
Associations	Primary sclerosing cholangitis and p-ANCA positivity	Ankylosing spondylitis, sacroiliitis, migratory polyarthritis, erythema nodosum, and uveitis
Smoking	Protects against UC	Increases risk for Crohn disease

Treatment of IBD



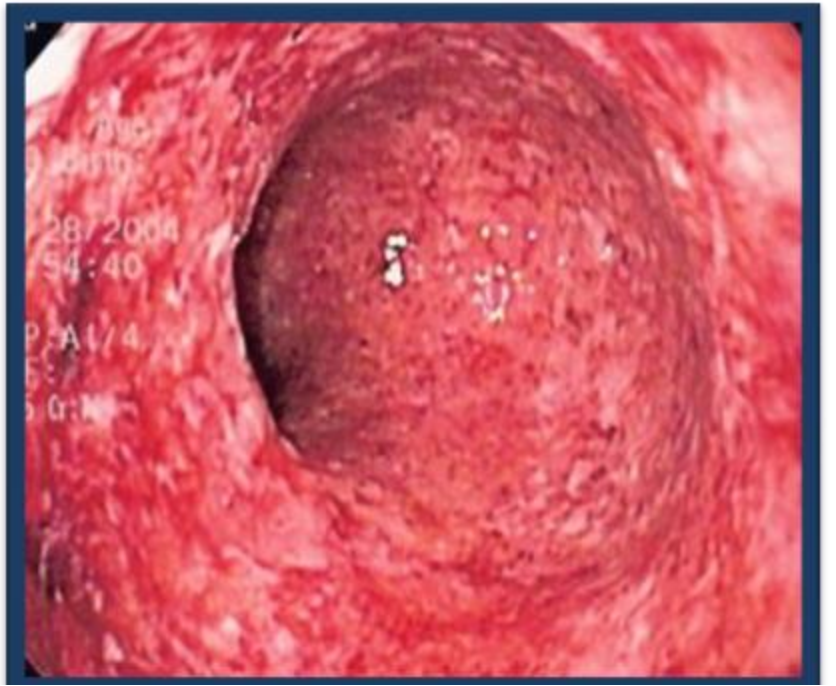
Major endoscopic features for UC :

1. Diffuse involvement
2. Rectum always diseased
3. Superficial ulcerations
4. Friability/bleeding
5. Flattening/disappearance of *haustral folds
6. Pseudopolyps
7. No cobblestoning

Q: A 25 y.o. non-smoker female presented to the ER with bloody diarrhea, mixed with mucus and tenesmus…after performing colonoscopy this how her colon looked like… What's her condition?

This is typical endoscopic picture for Ulcerative Colitis

Note the diffuse involvement and the SAND PAPER appearance



Mention 2 serological test for diagnosis:

1- Saccharomyces cerevisiae antibody (Negative)

2- P-ANCA (positive)

If this pt came with jaundice what is your ddx?

1-Sclerosing cholangitis

2-Cholangiocarcinoma

Leading cause of death in this disease?

Toxic Megacolon

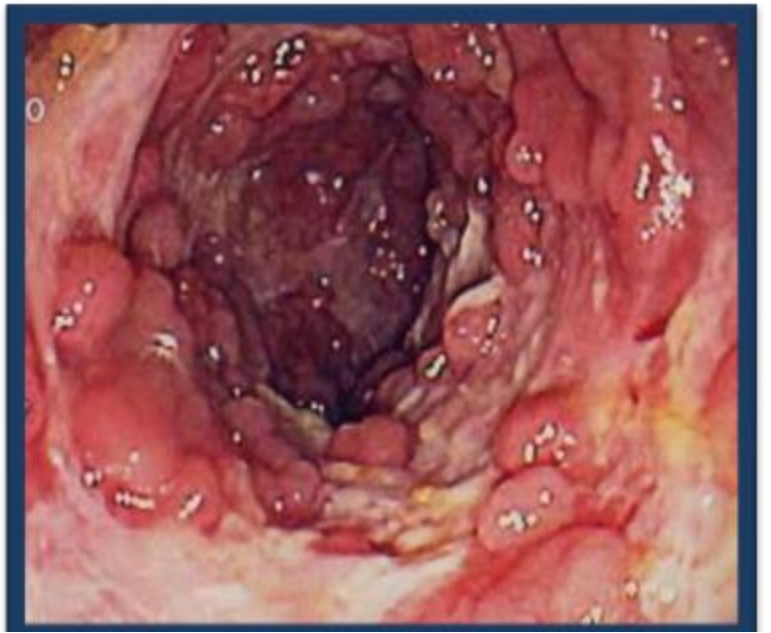
Major endoscopic features for CD

1. Asymmetric patchy *inflammation
2. Skip lesions
3. Rectal sparing
4. Ulcerations-deep/serpiginous
5. Cobblestoning-common
6. Pseudopolyps-rare

Q:A 30 y.o. smoker male presented at the clinic with watery diarrhea ,abdominal pain and weight loss...these are the pictures of his colonoscopy...What is his condition?

Crohn's Disease

Note the patchy involvement and the COBBLE STONE appearance



Q:This ileum appearance is in a young patient with weight loss, chronic diarrhea and right lower abdominal pain.

Name the underlying autoimmune disorder.

Crohns disease

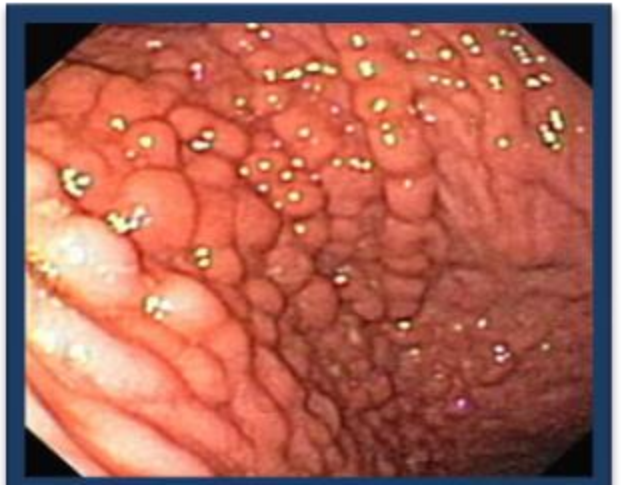
Mention 2 serological test for diagnosis:

1- Saccharomyces cerevisiae antibody (positive)

2- P-ANCA (Negative)

Most Common indication for surgery in this disease?

- Small bowel obstruction



Q: A Patient has bloody diarrhea & this skin lesion, What is your Dx.?

Inflammatory Bowel Disease: (Mostly Ulcerative colitis).

DDX:

1-IBD

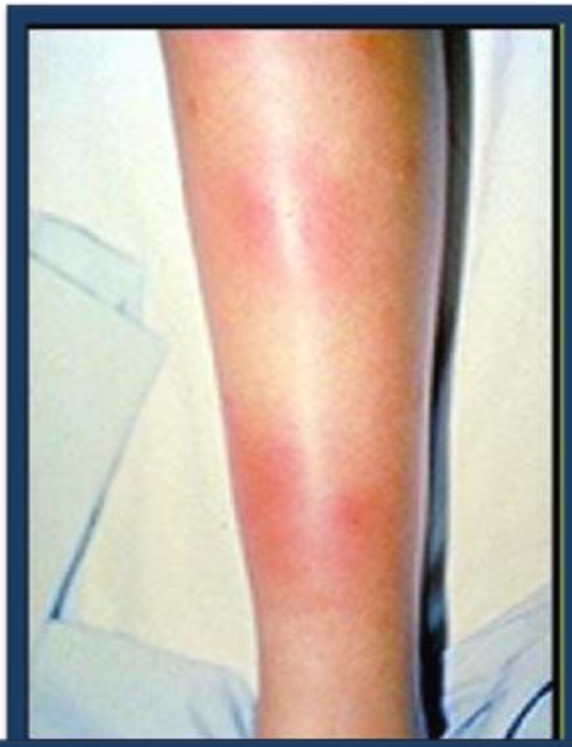
2-Sarcoidosis

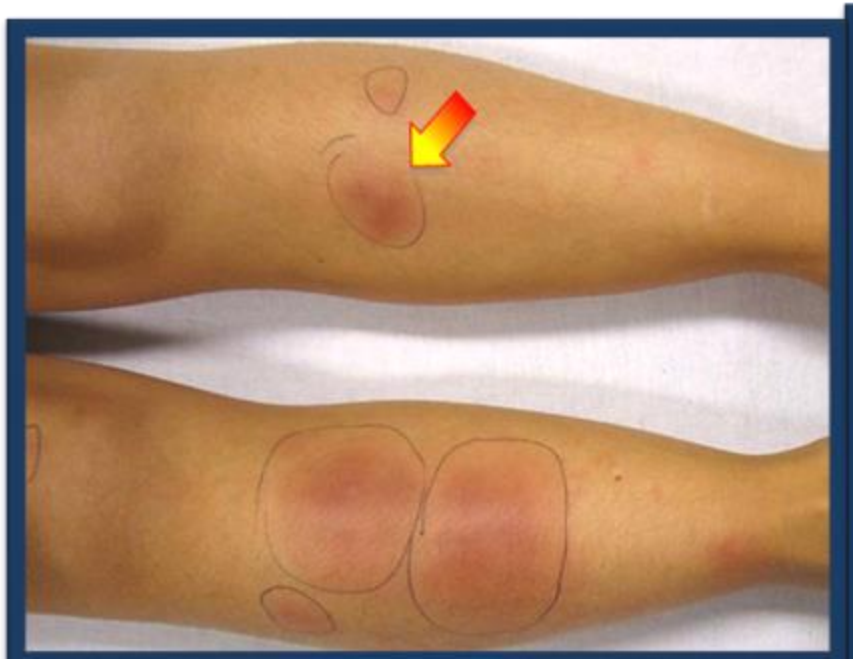
What is the name of this lesion?

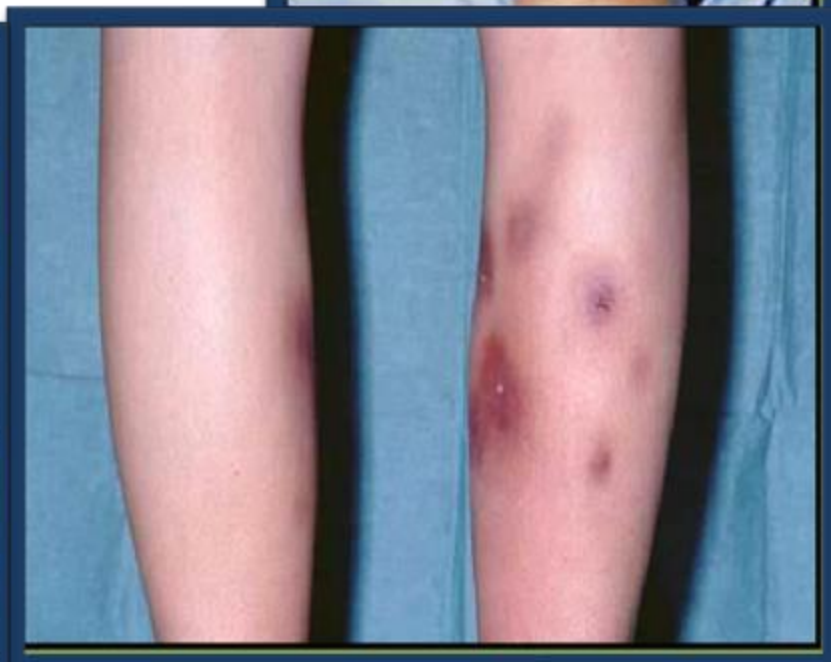
Erythema nodosum.

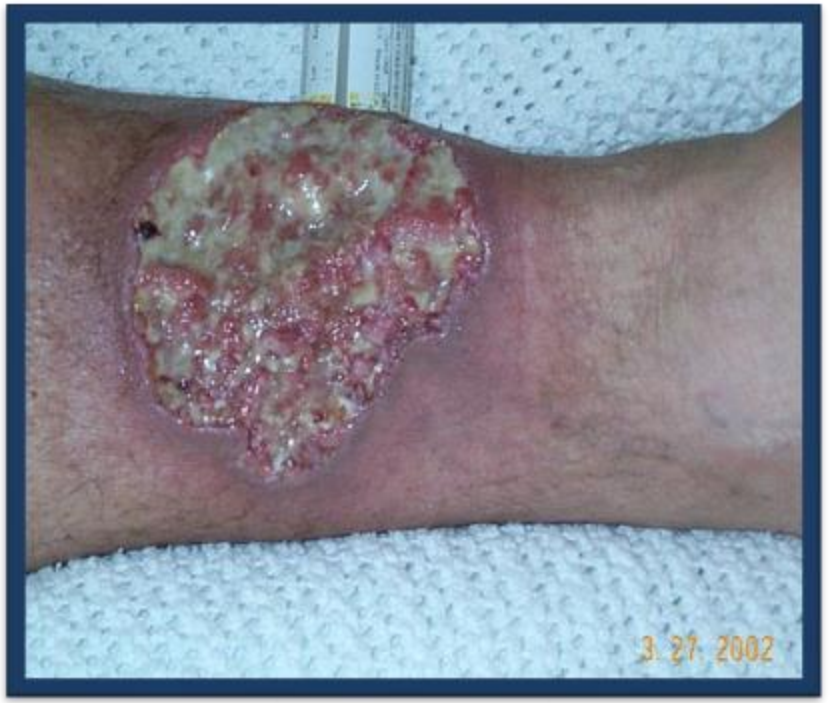
What is the best treatment for this condition?

Steroids



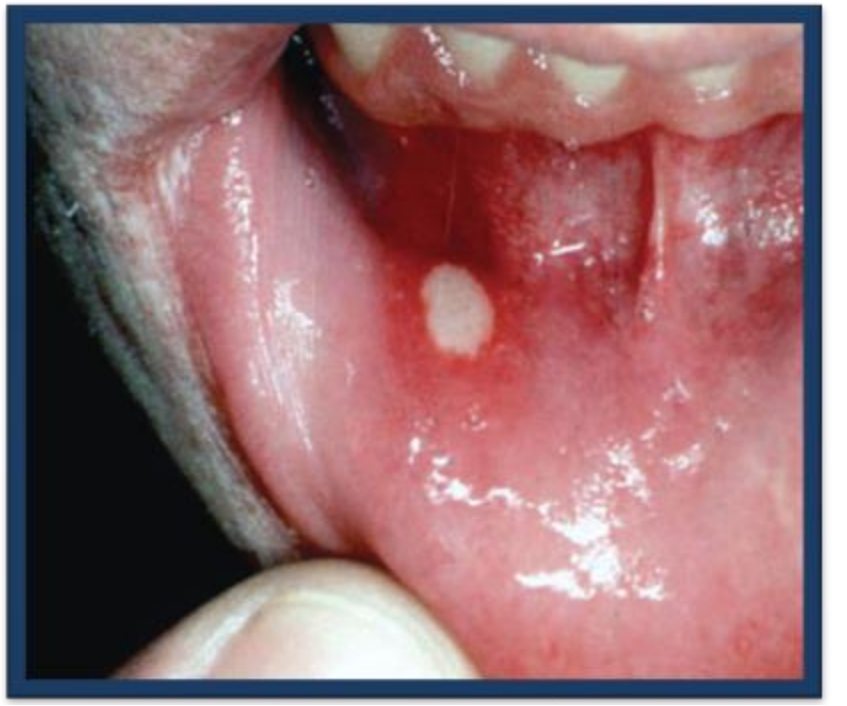






pyoderma gangrenosum
in UC , parallels bowel disease activity in 50% of cases

This is Aphthous Stomatitis
Painful ulcer in the mouth that everyone of us had experienced



Q:

A- A known case of crohns disease came with this oral lesion identify this lesion?

aphthus ulcers

Note: some said it was candida infection (Pic was not that clear)

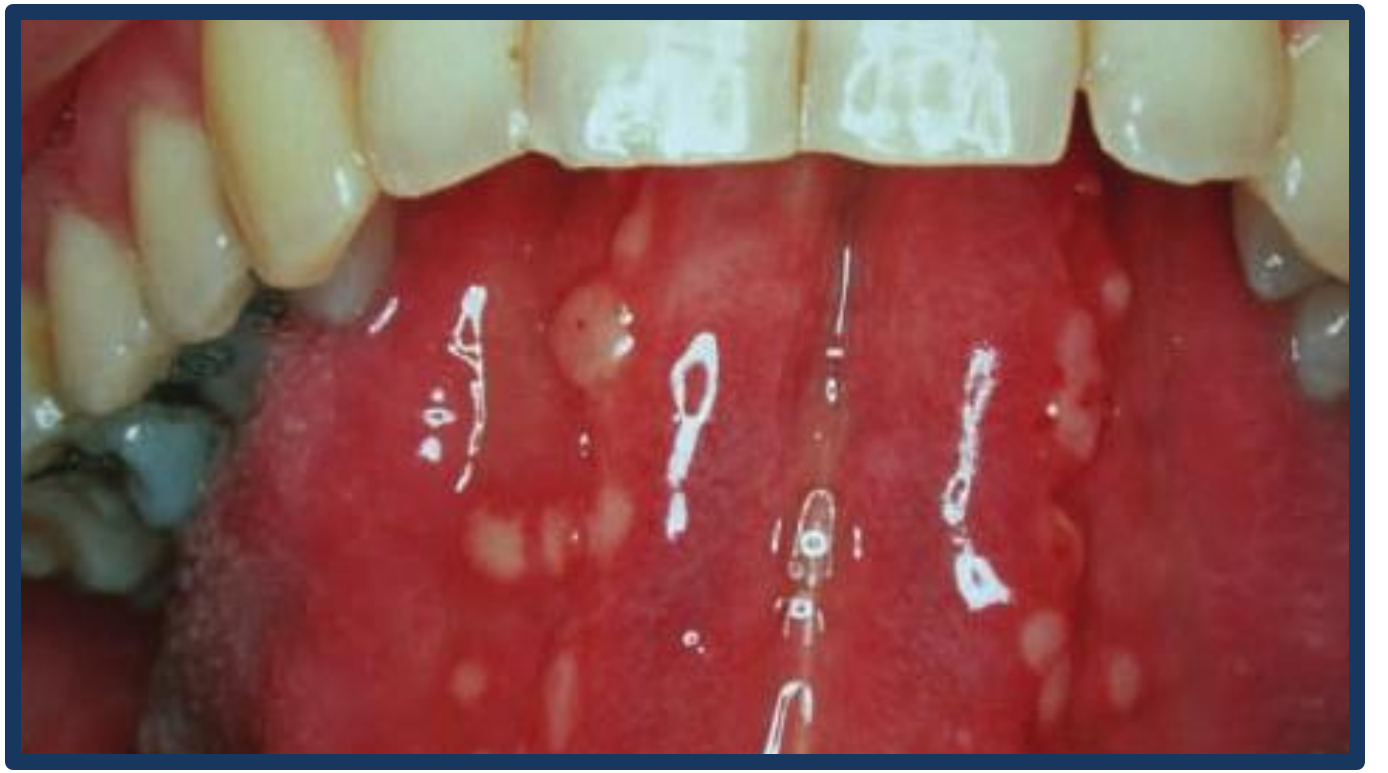
B- Do you think the anus will be affected??

Yes anus can be affected

c. Mention 2 DDx?

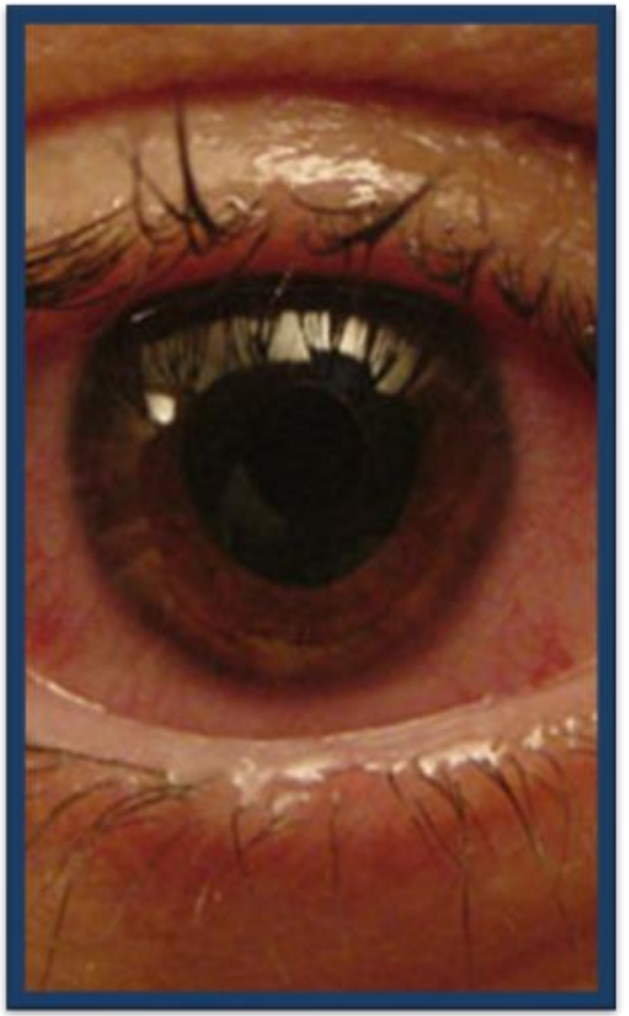
1- Behcet disease

2- IBD



Now we will move to the lesions of the eyes…

Uveitis : (Doesn't parallel bowel disease activity)

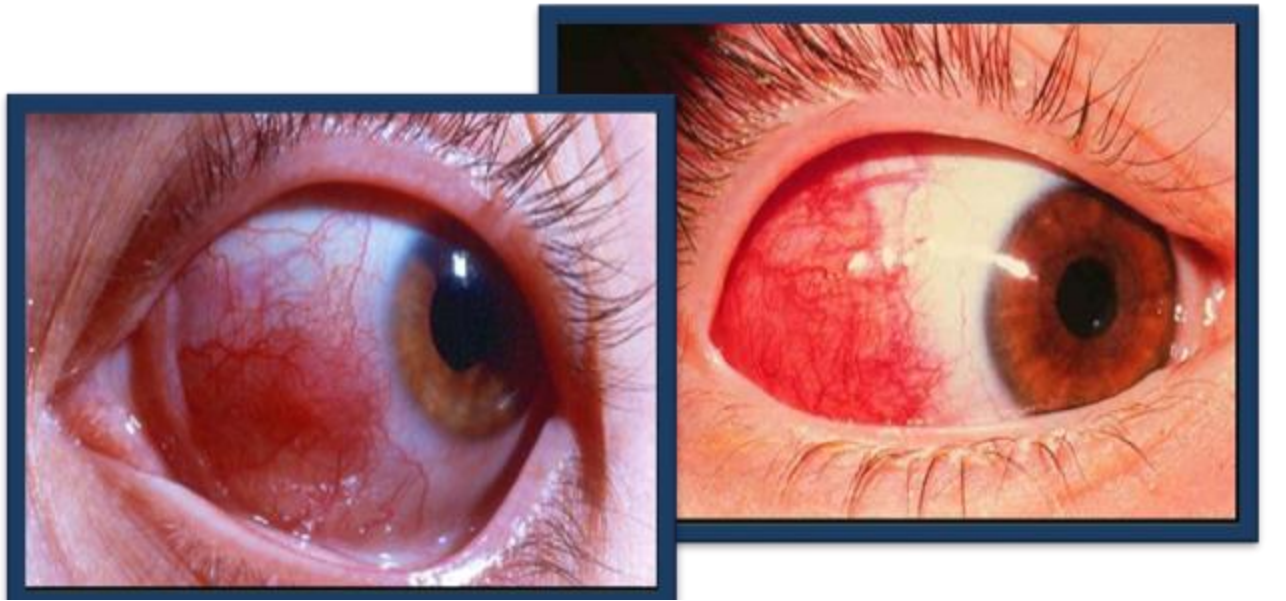


Q:A pt presented with bloody diarrhea & tenesmus as well as this painless eye lesion. what is your diagnosis?

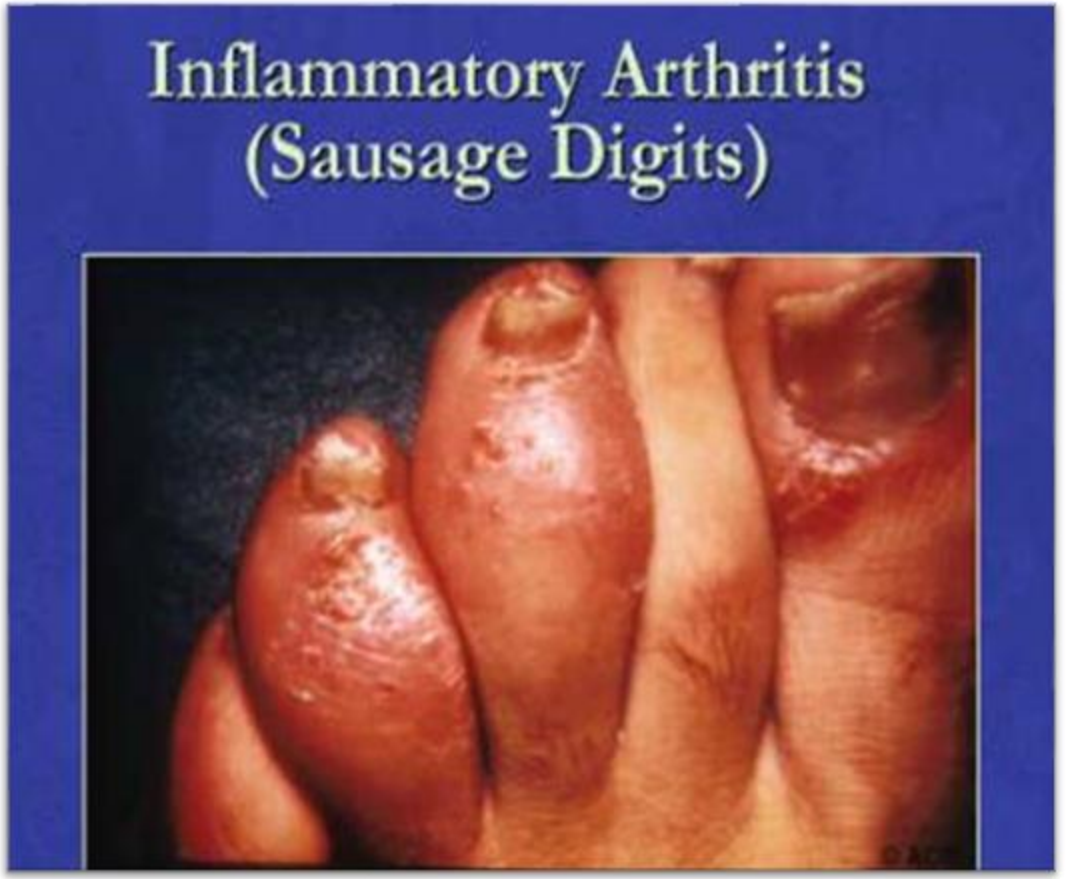
Ulcerative colitis.

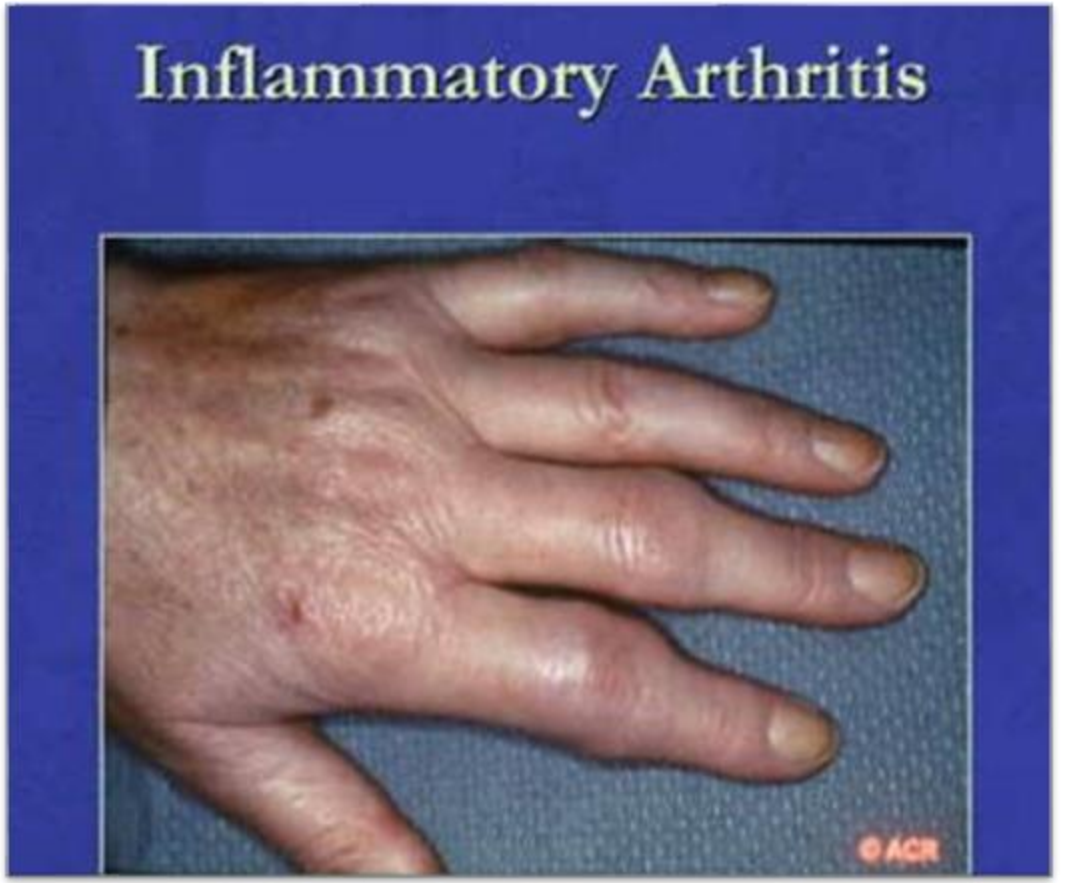
what is this eye lesion

Episcleritis. (parallel bowel disease activity)



Now we move to the rheumatological lesions that accompany IBD…





Q: This old-aged male has back pain that's relieved with exercise.

What the name of his condition?

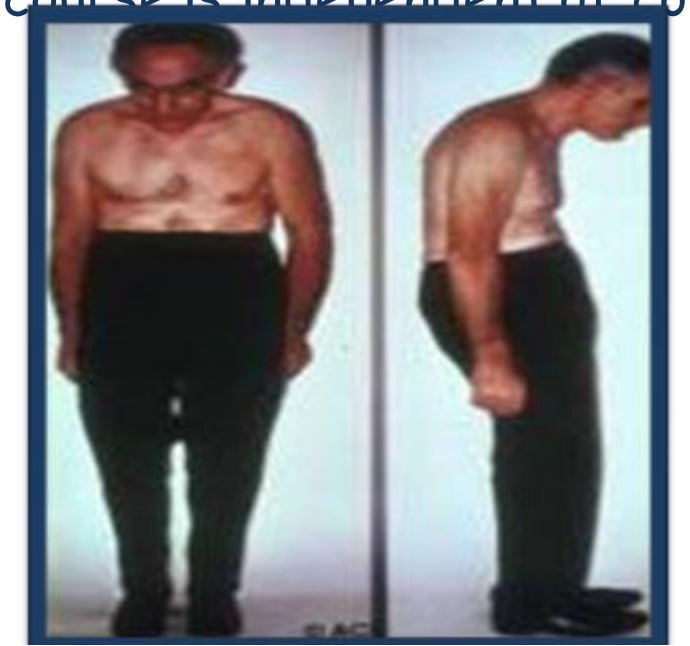
Ankylosing Spondylitis

What's the other disease that could accompany this condition?

could come along with (Crohn's with HLA-B27)

*Patients with UC have a 30 time greater incidence of AS.

*The course is independent of colitis .



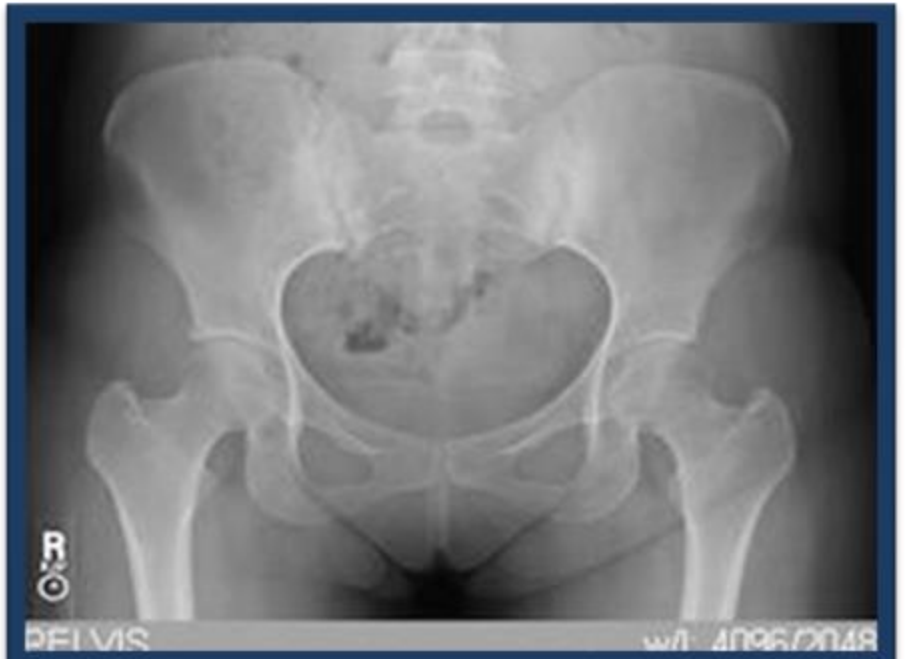
Q: This is an X-ray of a CD patient that came with lower back pain.

What is the name of this condition?

Sacroiliitis (note the loss of demarcation of sacroiliac joint)

Also accompanies Crohn's with HLA-B27

- Does not parallel bowel disease activity.

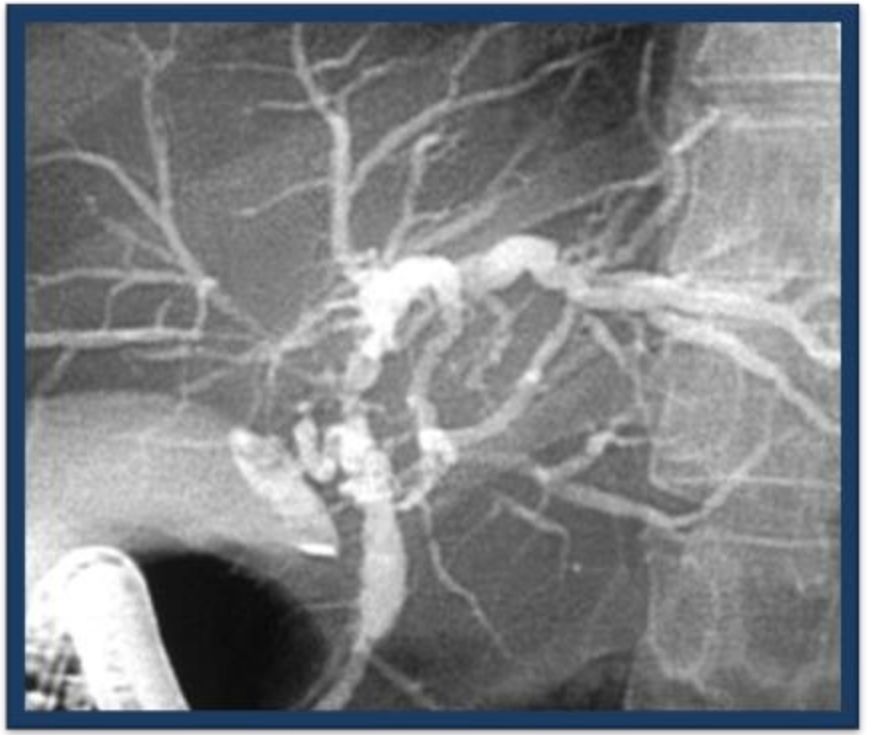


Q: This ERCP belongs to a patient who was presented with bloody diarrhea.

What is this condition?

Primary Sclerosing Cholangitis

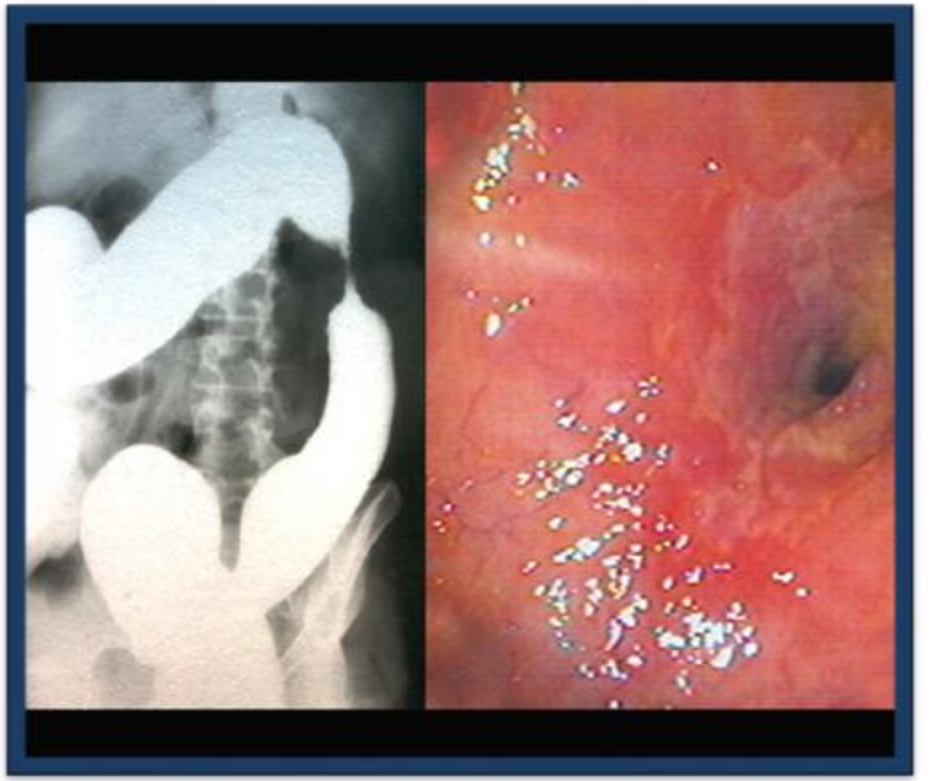
This is commonly a manifestation of UC and it is unrelated to the disease's activity…



These are an X-ray and an endoscope of a CD patient that now complains of constipation…

This is colonic stricture

This is a complication of Crohn's disease (usually)

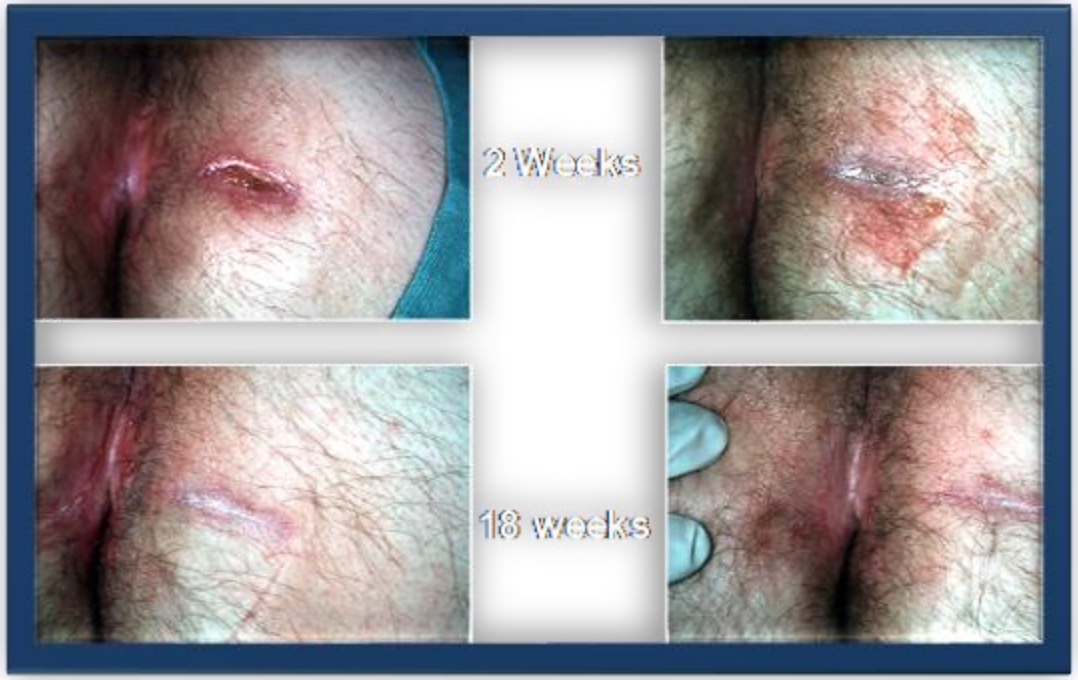


Q: This patient is presented with this condition.

What is the most likely underlying disease?

This is a perianal fistula

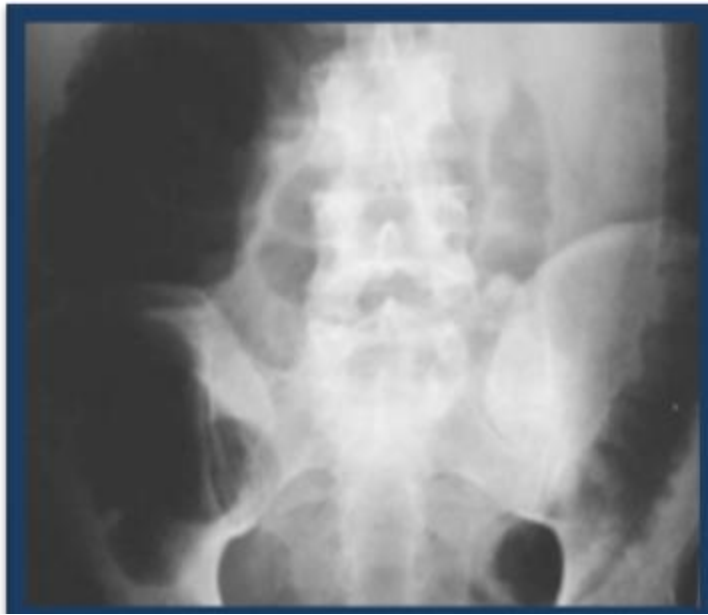
Fistulas such as perianal, enteroenteric, enterovesicular, enterovaginal are complications of Crohn's disease.



Q: A middle aged patient known to have UC and was brought to the ER looking shocked with distended abdomen. After performing an abdominal xray this was the result. What is this condition?

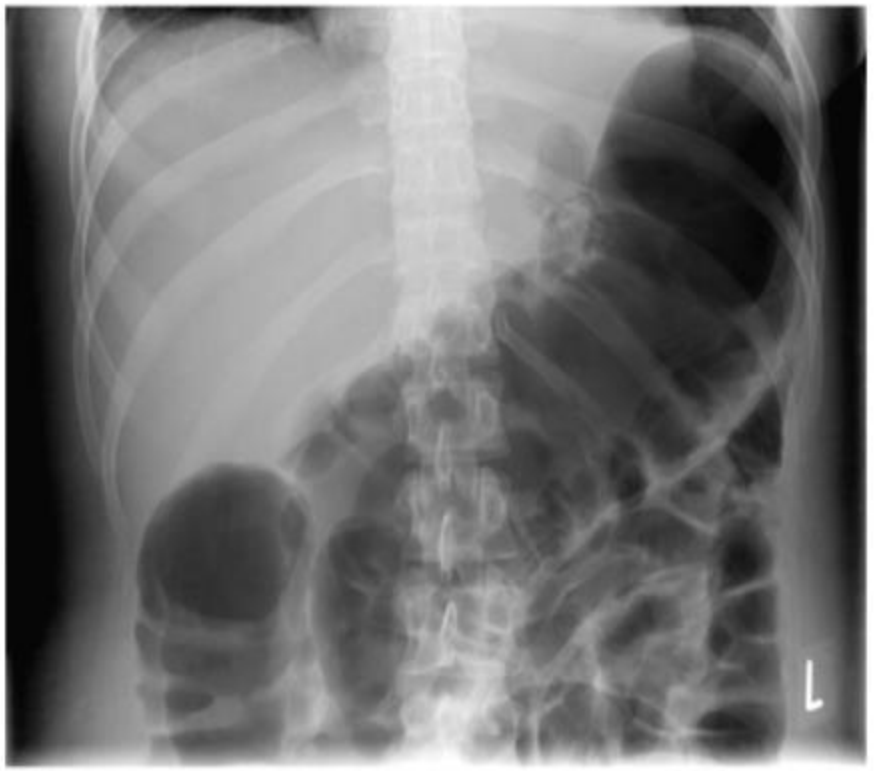
Toxic Megacolon (note the big black shadow on the left of the screen)

This is a known complication of UC as it the wall of the bowel thinner.



36 years old patient with IBD, present with abdominal pain & distension
- What complication is shown in this Abdomen X ray?

Toxic Megacolon



Q:A 65 y.o. patient known to have UC with remission and relapse. Now he complains of anorexia and weight loss with alternating bowel habits. What should we think about in our DDx?

Colon cancer.

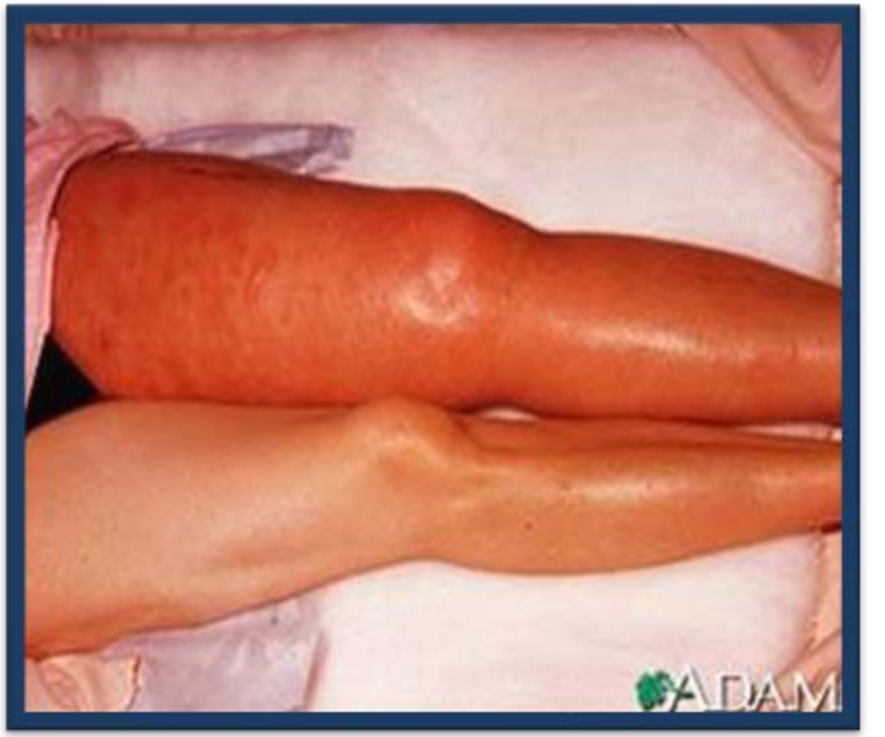
Colon cancer is one of UC complications

Q:Now, if a patient known to have IBD and he was presented to the ER with swollen erythematous tender unilateral lower limb what is your explanation?

Venous Thrombosis

- can lead also to :

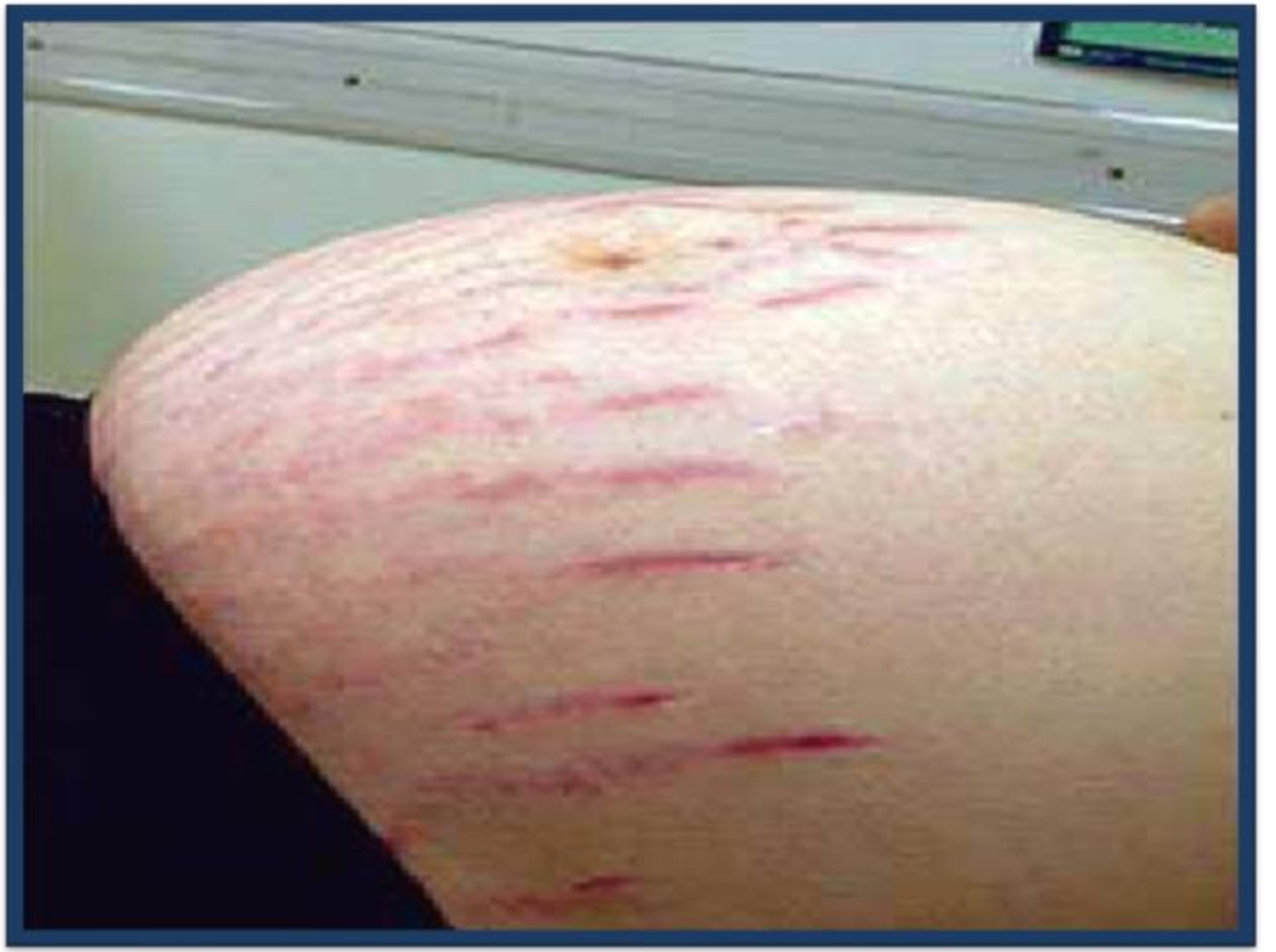
1-PE 2-CVA 3-ITP



Q: pt of Crohn's disease presented with these lesions on his abdomen.

What's the name of these lesions & what is the cause?

Abdominal Stria due to Steroid Therapy in IBD.



5.Celiac Disease

Q: A 3 year old boy presented with diarrhea for one month,
Name 3 histological findings ?

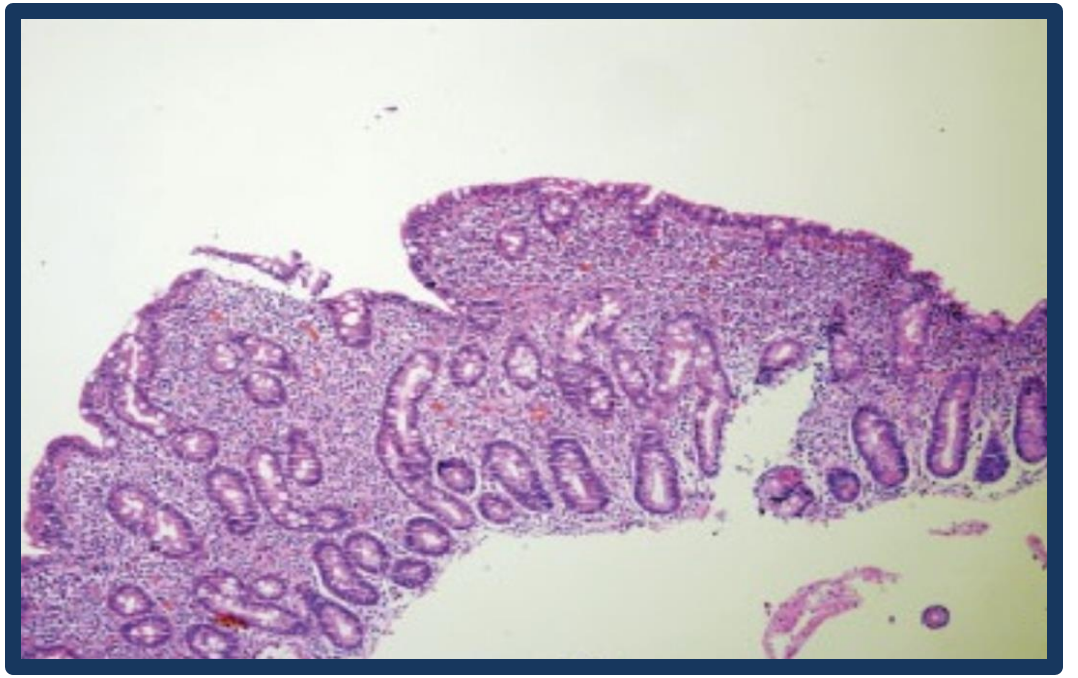
1-lympocytic infiltrate
2-flattening of the villi(atrophy)
3- villus to crypts ratio less than 3:1

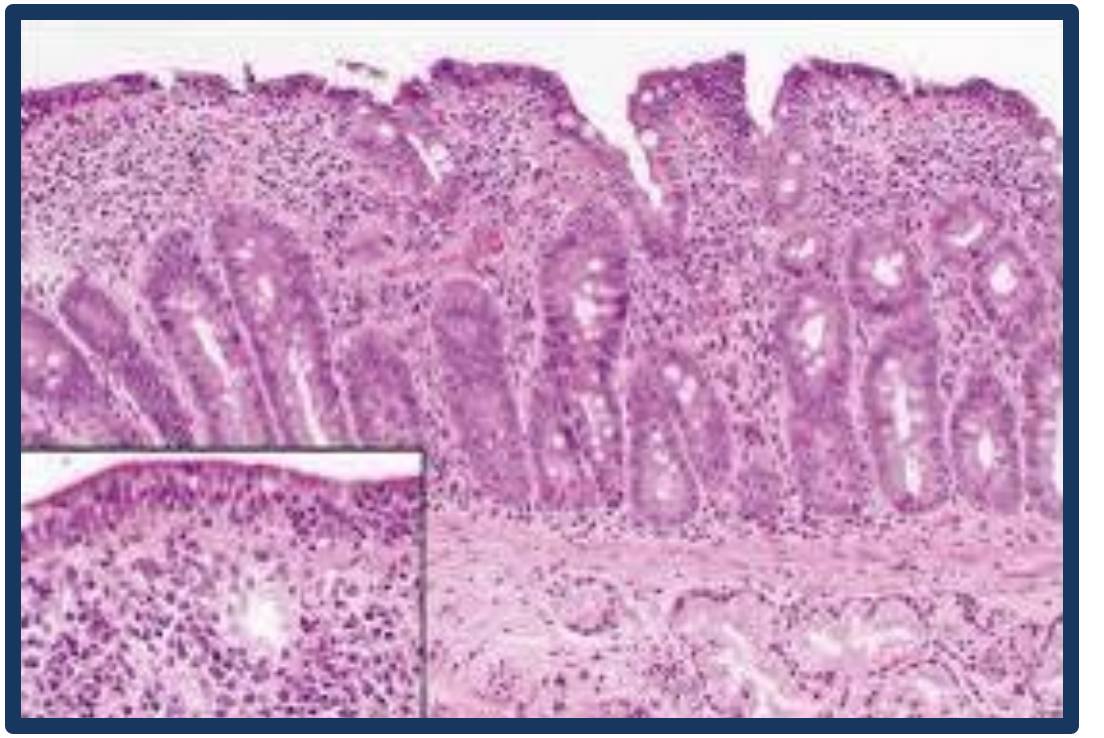
Your Dx?

Celiac Disease

Treatment?

free diet, Fluid & nutrients Glutein replacement





*Classical presentation:

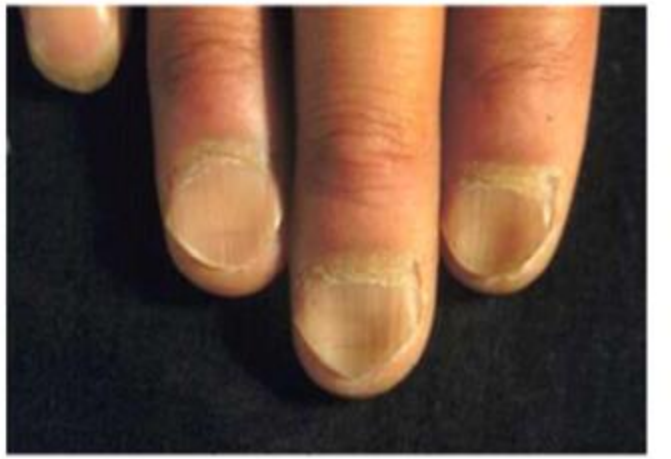
-Abdominal distention
-Wasted extremites
-Chronic diarrhea
-Abdominal pain
-Aphthous ulcer
-Weight loss

*Extraintestinal manifestation:

-anemia(iron,b12,folate)
-Rickets
-Peripheral neuropathy(b12,b6)
-Seizure (occipital calcification)
-Dermatitis herpitiformis
-Short stature

- In patient with celiac disease you found this nail change, what is the main cause ?

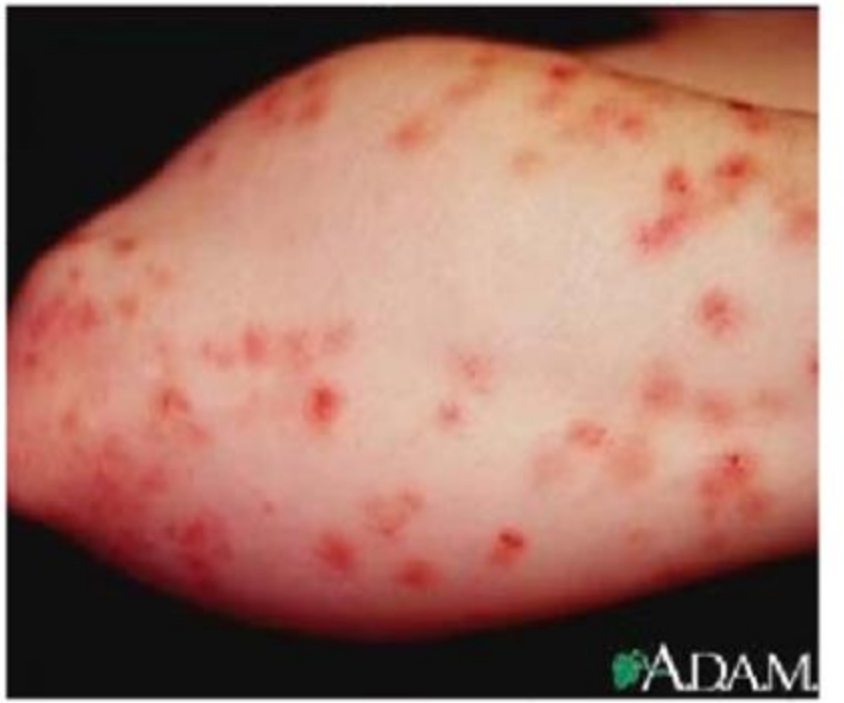
- **Koilonychia**
- **Most common cause is iron deficiency anaemia**



A 60 lady has symptoms of intermittent abdominal pain and loose stool which have occurred over 1 year, Iron & folate Deficiency anemia, TTG antibodies positive.

- What is this skin lesion ?

Dermatitis Herpetiformis In Celiac Disease



Patient with diarrhea, abdominal pain and other symptoms and lab findings, anti TTG +ve.

1- what's your diagnosis?

celiac disease

2- what's the most common cause of anemia?

IDA

3- what's the HLA type?

HLA DQ8, DQ2 (99% of patients)

4- what's the best diagnostic investigation?

intestinal biopsy

5- what's the treatment?

glutein free diet

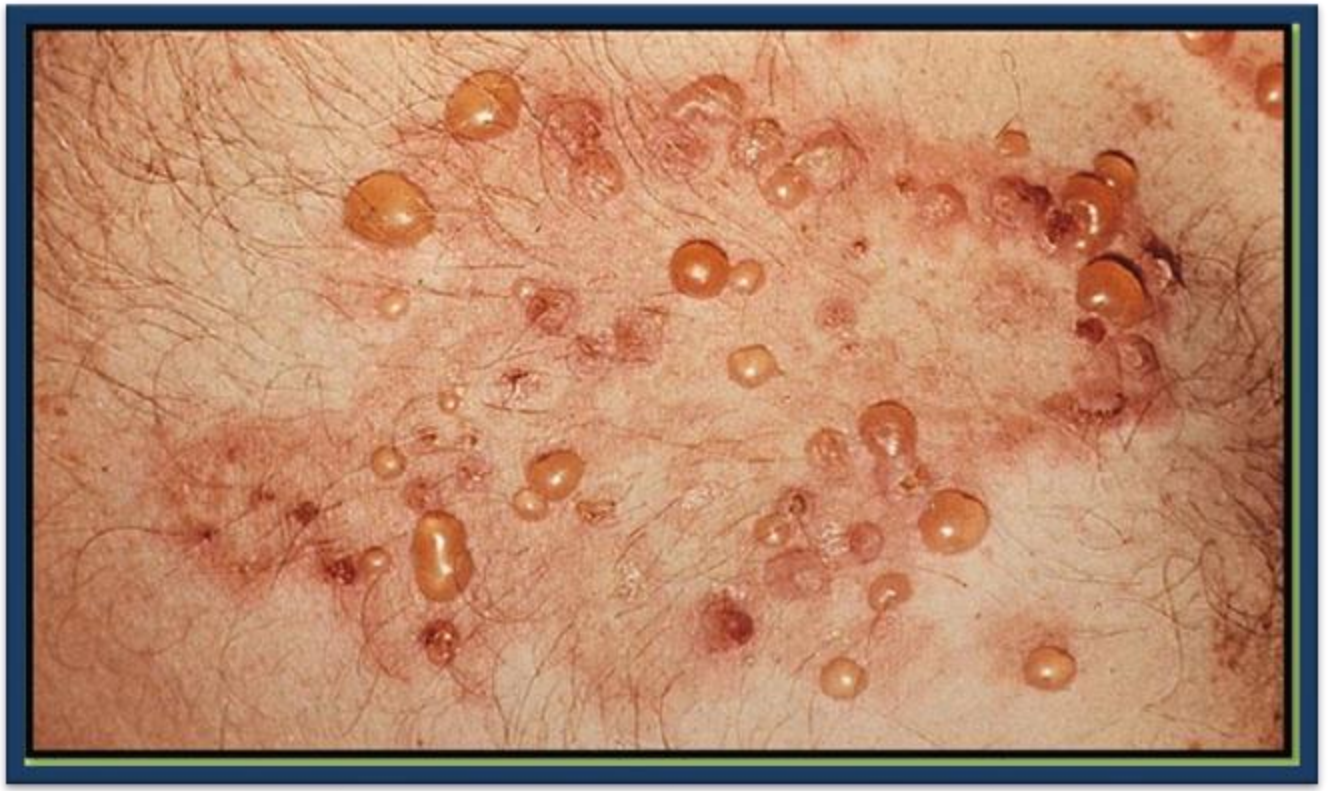
Q: Over a period of 6 weeks, the 18 YO pt began to develop abdominal bloating, pain, & Diarrhea. in CBC: she was anemic.

1) what is the pathology seen in the picture?

dermatitis herpetiformis.

2) what is the most likely Dx?

celiac disease.



6.Others

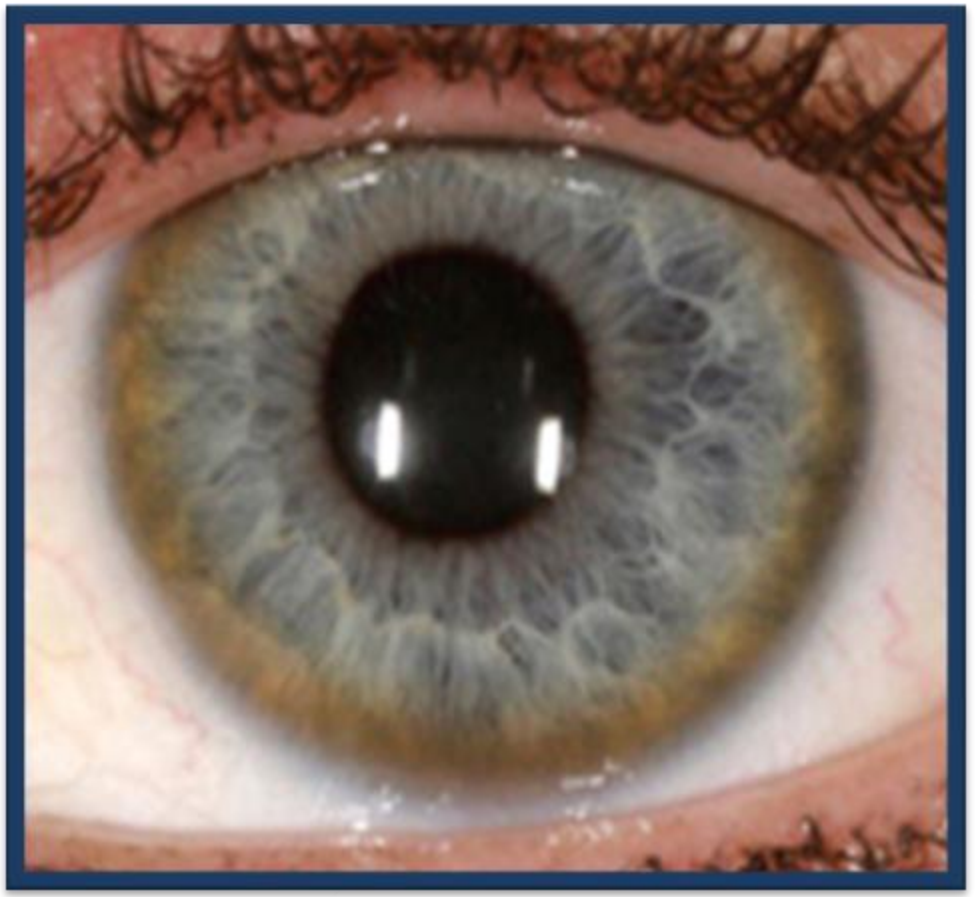
Q: Patient die due to liver failure.

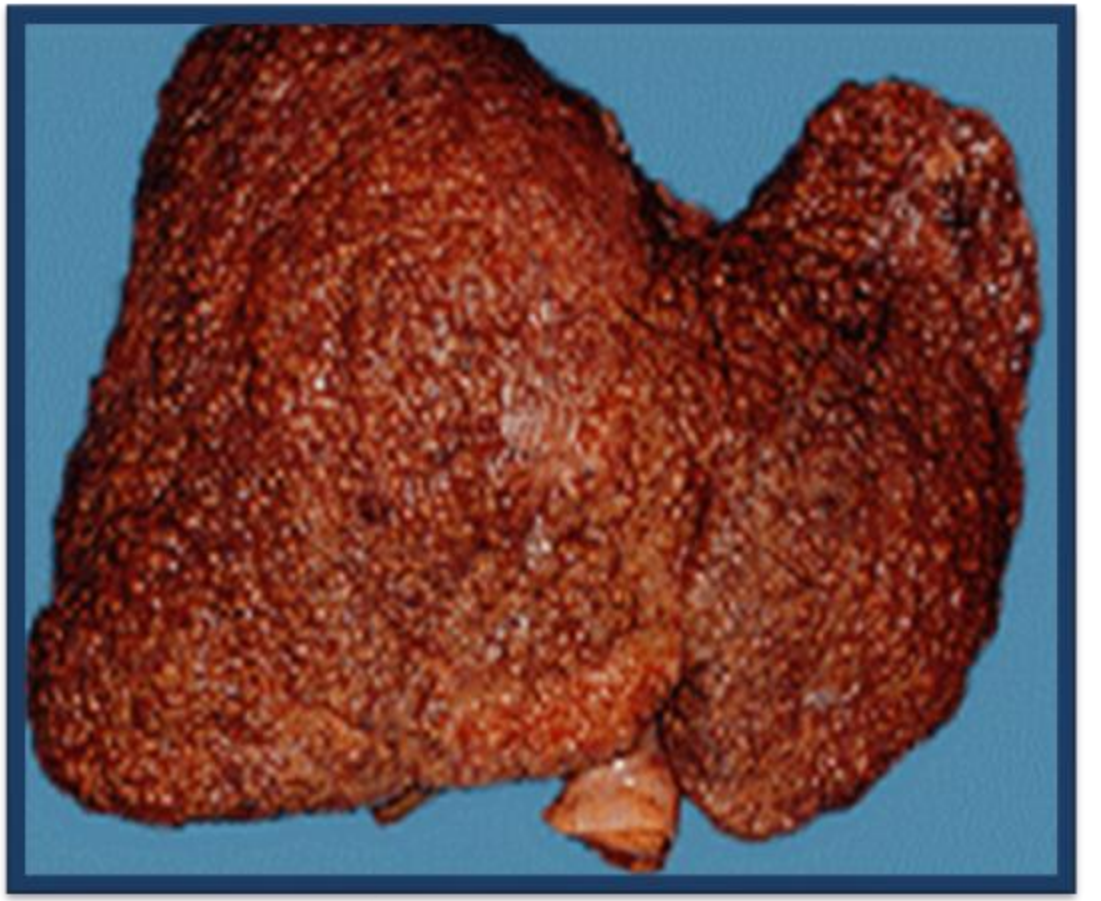
A- what's your diagnosis?

wilson disese

B- What's the eye finding?

kayser fleisher ring





*Cause:

Mutations in the ATP7B gene lead to impairment of copper excretion into bile

*The disease is most often apparent during childhood/adolescence (after age 5), and the majority of cases present between ages 5 and 35.

*Clinical Features:

1- Liver disease (most common initial manifestation): Manifestations vary and may include acute hepatitis, cirrhosis, and fulminant hepatic failure.

2- Kayser–Fleischer rings (yellowish rings in cornea) are caused by copper deposition in cornea; they do not interfere with vision

3- CNS findings are due to copper deposition in the CNS. a. Extrapyramidal signs—parkinsonian symptoms (resting tremor, rigidity, bradykinesia), chorea, drooling, incoordination due to copper deposition in basal ganglia.

b. Psychiatric disturbances—depression, neuroses, personality changes, psychosis.

4- Renal involvement—aminoaciduria, nephrocalcinosis

*diagnosis: presence of:

a. Hepatic disease—elevated aminotransferases; impaired synthesis of coagulation factors and albumin.

b. Decreased serum ceruloplasmin levels (seen in 90% of patients), although ranges within normal do not exclude the diagnosis.

c. Liver biopsy

*If diagnosed, first-degree relatives must be screened as well.

*treatment:

1-Chelating agents like: D-penicillamine

2-Zinc

3-Liver transplantation

Q:A 79 YO, is admitted to the hospital with CC: intermittent rectal bleeding for 3 days. What is the diagnosis?

Diverticulosis

Mention one complication of the diagnosis.

Bleeding, infection (diverticulitis), perforation.

The most common location:

sigmoid colon.

Diagnosis:

1. Barium enema is the test of choice.
2. Abdominal x-rays are usually normal and are not diagnostic for diverticulosis.

Treatment:

1. High-fiber foods (such as bran) to increase stool bulk
2. Psyllium (if the patient cannot tolerate bran)

complications:

1. Painless rectal bleeding (up to 40% of patients).
2. Diverticulitis (15% to 25% of patients), presentation: (fever, LLQ pain, leukocytosis.)

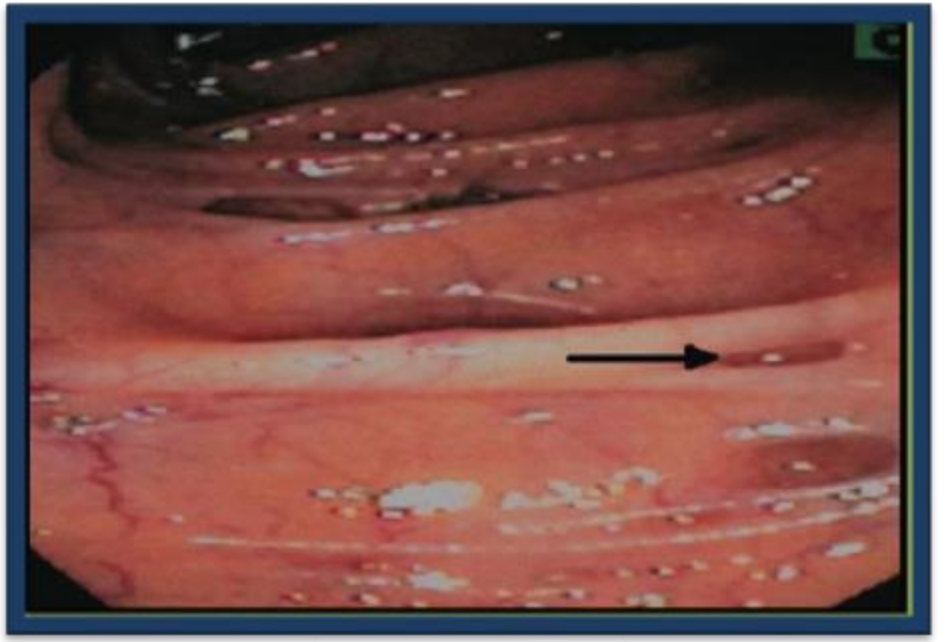
Diverticulitis:

- **Diagnostic tests:**

  CT scan (abdomen and pelvis) with oral and IV contrast is the test of choice; Barium enema and colonoscopy are contraindicated in acute diverticulitis due to the risk of perforation.

- **Treatment of diverticulitis:**

- Uncomplicated diverticulitis is managed with IV antibiotics, bowel rest (NPO), IV fluids
- Complicated diverticulitis—surgery indicated.



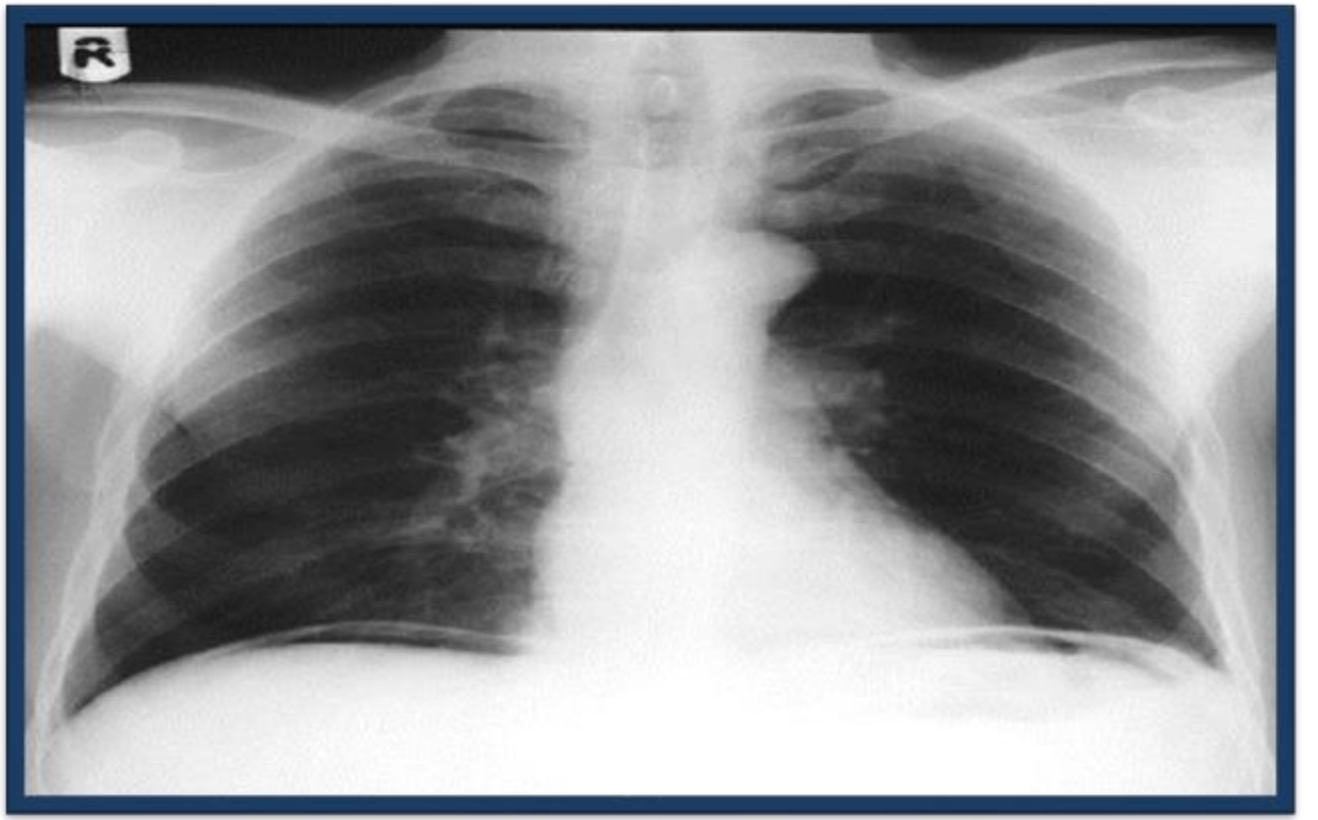
Q: The pt presents with sudden & severe abdominal pain.

What the abnormal finding shown on CXR?

Air Under The Diaphragm .

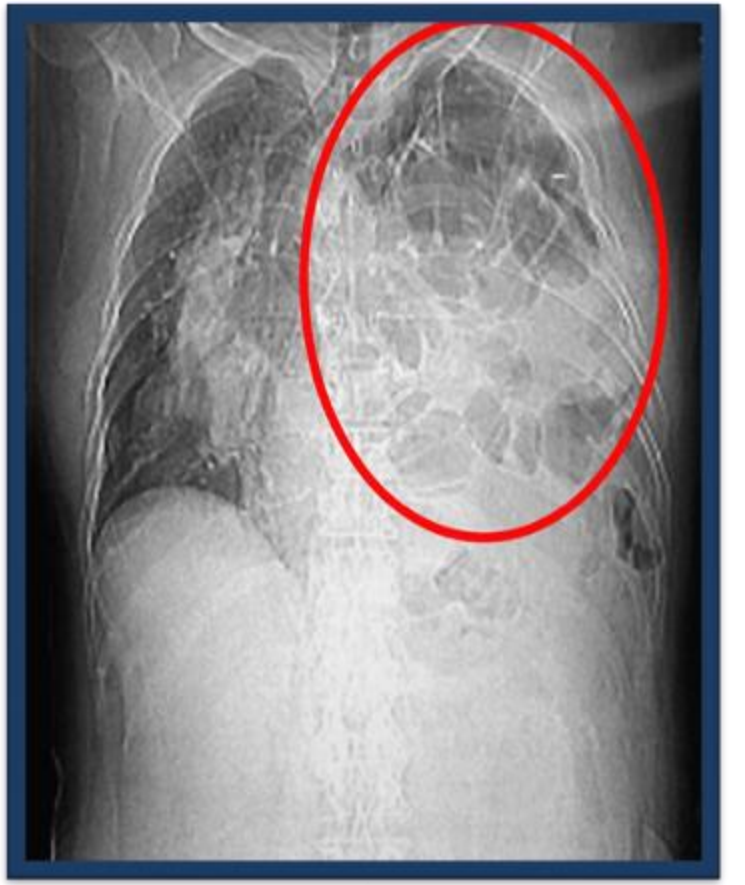
What is the Dx?

Perforated Viscous.



Causes of air under diaphragm:

- Perforated duodenal ulcer – The most common cause of rupture in the abdomen.
- Perforated peptic ulcer.
- Ruptured diverticulum.
- Penetrating trauma.
- Ruptured inflammatory bowel disease (e.g., megacolon)



Q: Whats the diagnosis ?
Diaphragmatic Hernia

Most comon cause of this condition in adult is?
Trauma

Radiological signs?
1-Abdominal contents in the thorax
2-Distortion of diaphragmatic margin

Clinical Features:
Marked respiratory distress
Decreased breath sounds on the affected side
Palpation of abdominal contents upon insertion of a chest tube
Auscultation of bowel sounds in the chest
Paradoxic movement of the abdomen with breathing
Diffuse abdominal pain

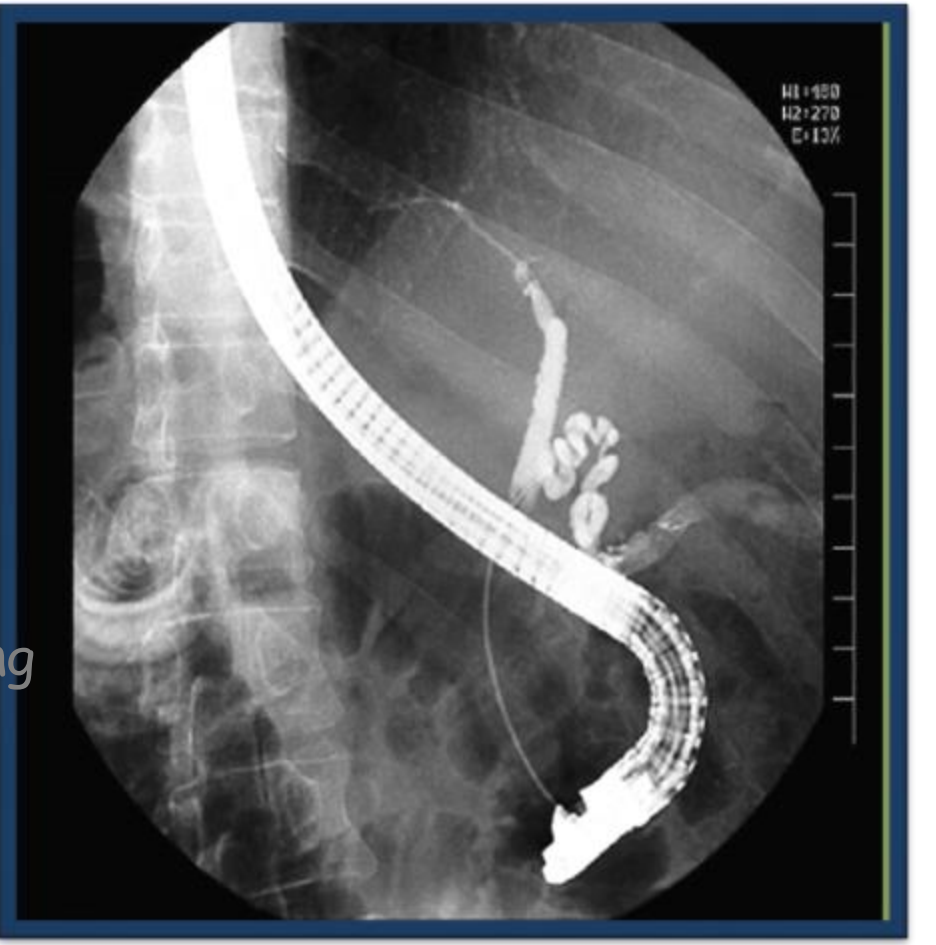
ERCP: It's diagnostic and theraputic procedure

Diagnostic uses :

1- obstructive jaundice
2- bile duct tumors
3- bancreatic tumors

Theraputic uses:

1- endoscopic sphincterotomy
2- removal of stones
3- insertion of a stent
4-dilatation of a stricture as in primary sclerosing cholangitis



Containdication of ERCP

1-acute pancreatits
2- previous pancreatoduodenectomy
3- coagulation disorder if sphincterotomy planned
4- recent MI
5- hx of contrast dye anaphylaxis

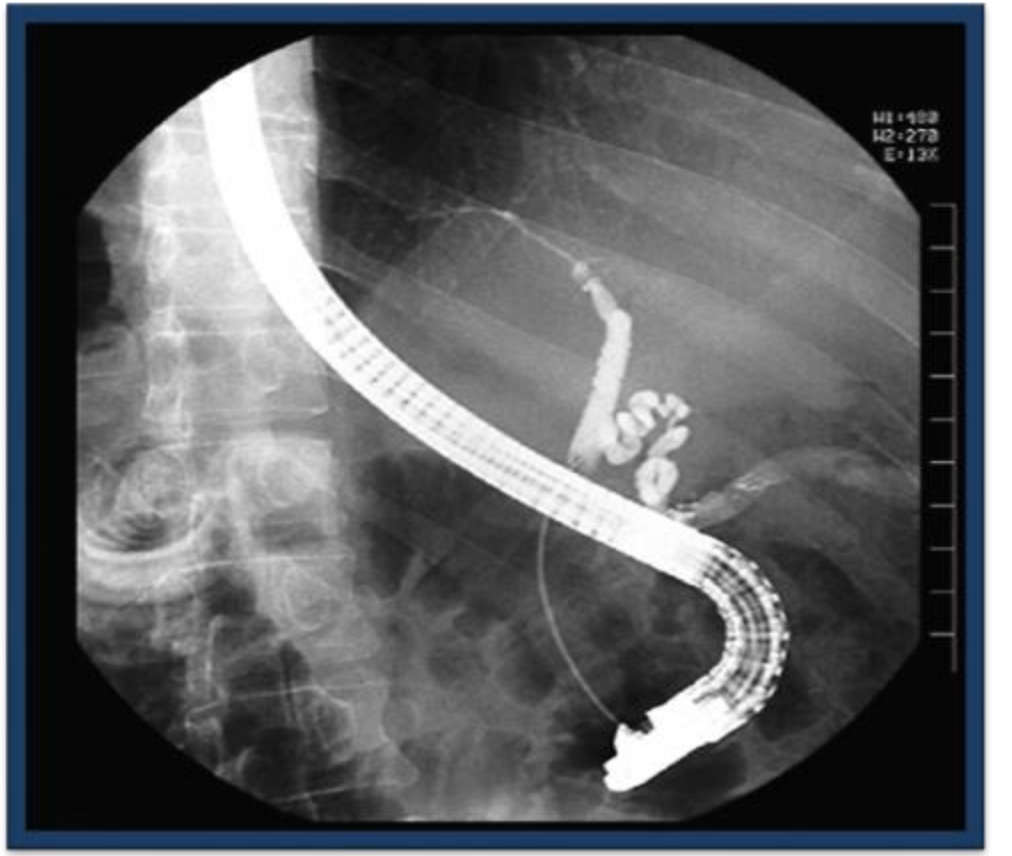
Preparation for ERCP:

1 - Npo >> for six to eight hours

2 - Nrophylactic AB

3 - Iv fluids

4 - Vit. k IM 10mg



Complications of ERCP:

1 - Duodenal perforation

2 - Haemorrhage after insertion or sphincterotomy

3 - Pancreatitis (there is some evidence for the use of periprocedural nitroglycerin or rectal NSAIDs after high risk procedure to prevent this complication)

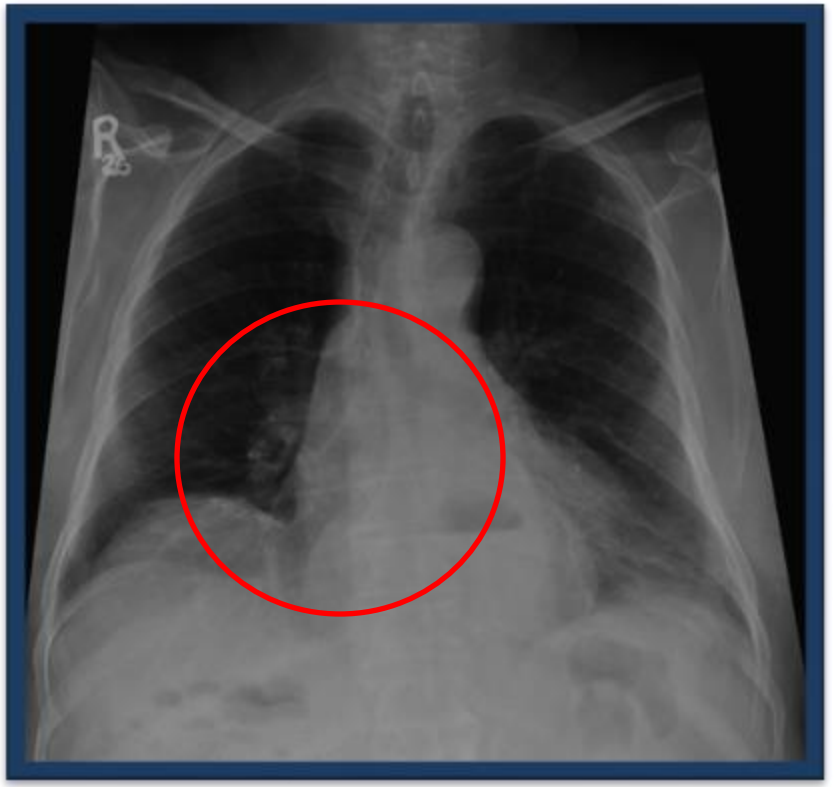
4 - sepsis

Q:What's your diagnosis?

Hiatal hernia

Radiological sign?

Rounded density
with air-fluid level
superimposed over
the cardiac silhouette.



Q: What is the finding in upper GI endoscopy?

gastric atrophy

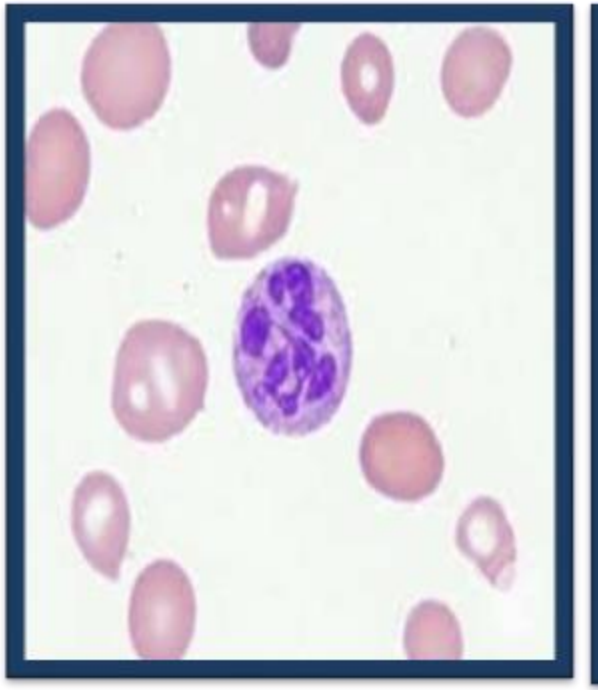
What are the findings in these pics?

Vitiligo

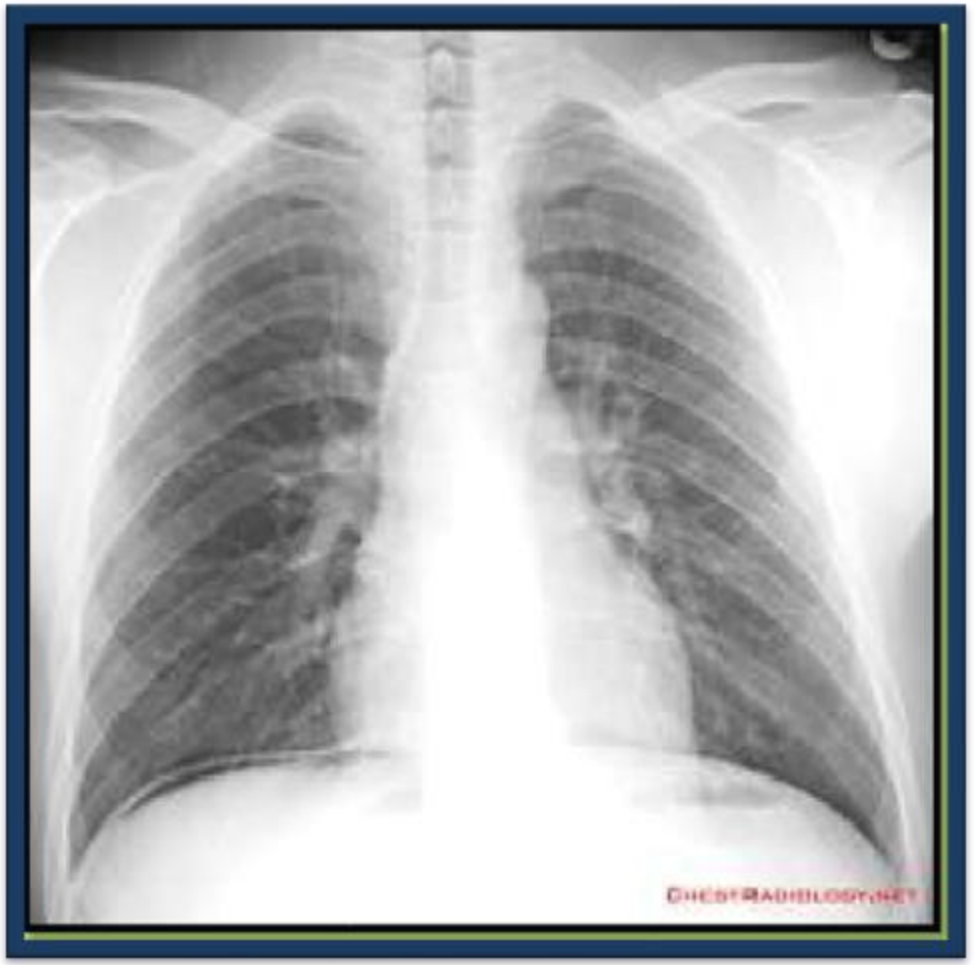
Hyper-segmented neutrophils

What is your Diagnosis?

Pernicious anemia (Autoimmune Disease)



Q: 23 year old male patient came with severe abdominal pain,
What is your diagnosis?
Perforated viscous (air under diaphragm).



*Causes :
1-Perforated duodenal ulcer – The most common cause of rupture in the abdomen. Especially of the anterior aspect of the first part of the duodenum.
2-Perforated peptic ulcer
3-Bowel obstruction
4-Ruptured diverticulum
5-Penetrating trauma
6-Ruptured inflammatory bowel disease (e.g., megacolon)
7-Necrotising enterocolitis/pneumatosis coli
8-Bowel cancer
9-Ischemic bowel
10-Steroids
11-After laparotomy
12-After laparoscopy
13-Breakdown of a surgical anastomosis
14-Bowel injury after endoscopy

Differential diagnosis

1- A subphrenic abscess
2- Bowel interposed between diaphragm and liver (Chilaiditi syndrome)
3- Linear atelectasis at the base of the lungs
All those can simulate free air under the diaphragm on a chest X-ray.

Treatment:

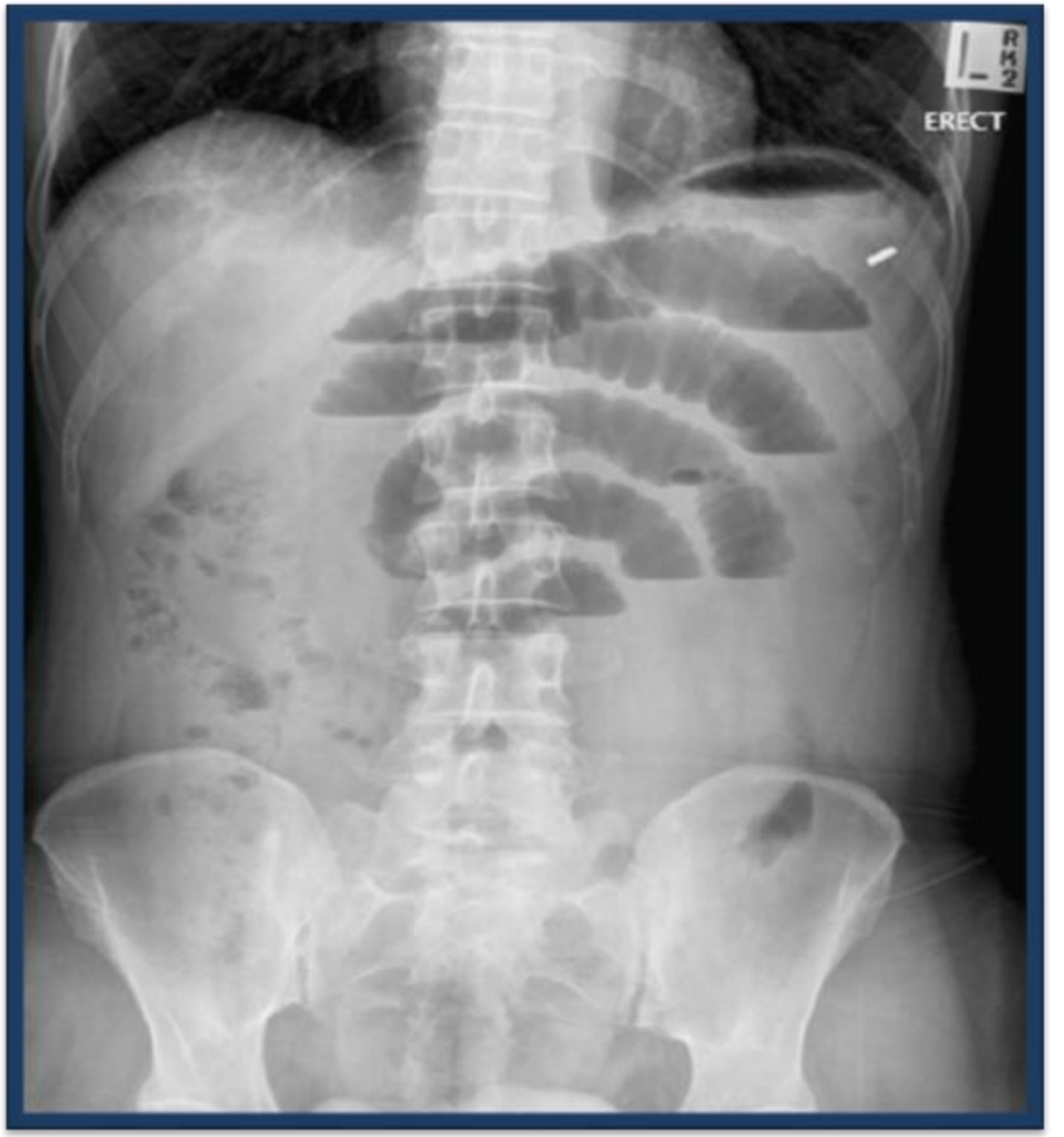
Depends on cause
Usually a surgical consultation is indicated

Q: What's your diagnosis?

Intestinal Obstruction

Clinical features?

1- Vomiting
2- Abdominal Pain
3- Constipation
4- Abdominal distension



Causes of intestinal obstruction according to the site:

Duodenum	Small bowel	Colon
Stenosis	Adhesions	Carcinoma
Foreign body (bezoars)	Hernia	Fecal impaction
Stricture	Intussusception	Ulcerative colitis
Superior mesenteric artery syndrome	Limphoma	Volvulus
		Diverticulitis
		Intussusception
		Pseudo-obstruction

Tinitinalli J, Kelen GD, Stapczynski JS (eds) (2004) Emergency medicine: a comprehensive study guide, 6th edn. McGraw-Hill, New York

7.Cases

*46 YO male pt comes vomiting coffee ground blood & black stools. Pulse: 96, RR: 24, BP:100\60. He had dizziness, general fatigue & weakness, SOB , & palpitation at rest.

UGIB

The first physical sign u want to look for?

postural hypotension .

Indications of severity?

hematochezia, sign and degree of shock (check vital sign).

Management?

IV fluid, Blood.

Mention3 causes related to your case:

Peptic ulcer gastric or esophageal varix ,esophagitis

2 confirmatory test:

Upper GI endoscopy, coloscopy

2 complications related to your case:

Shock , sepsis , DIC

Q: A previously healthy 36 YO male applied for a job in KSA, his application was refused because of abnormal liver function test. He drinks Alcohol occasionally, he was asymptomatic. his AST and ALT were mildly elevated. (numbers were mentioned in all the following tests, so you should know the normal ranges), his ALP was in normal range, +ve for Hbs IgG, -ve for Hbc antigen & Hbs antigen, –ve for other hepatophilic viruses. There was increase in LDL, Triacylglicerides, and a high BMI. Tests for metabolic and inherited liver diseases were normal.

HBV

1- Mention 3 DDx ?

chronic hepatitis B infection, steatohepatits, Autoimmune diseases.

2- Mention 2 tests to confirm your diagnosis ?

(definite Dx) >> Ds-DNA of hepatitis B, Liverbiopsy .

3- Mention 5 health problems associated with his BMI.

DM, HF, HTN, OSA, Atherosclerosis

Was about known case of liver cirrhosis who presented with loss of concoiusness and painful abdominal distention , the dx was spontoneous bacterial peritonitis.

Q: A 47 YO pt, known case of liver cirrhosis, presented with decreased level of consciousness. He takes propronolol, furosemide, spironolactone, lansoprazole, lactulose. He has been constipated for the last 2 weeks. His wife noticed abdominal distension. On P/E he is jaundiced, has ascites but no tenderness, paracentesis revealed clear fluid with 55 neutrophils per ml, gram stain was -ve. Lab results showed hyponatremia, hypokalemia, high creatinine.

1- What's the Dx?

Hepatic encephalopathy.

2- What's the cause of his hypokalemia?

Furosemide.

3- Give 2 possible causes for his condition?

Constipation, Hypokalemia (= diuretics).

Q: A male patient presented complaining of itching for 3months not responding to antihistamine. His lab data:

-Total protein 85 / Albumin 35 / Bilirubin 80 / Direct 20
-GGT and ALP high
-Antimitrochondial titer positive 1/280.
-ALT and AST normal.
-Ultrasound normal

Mention two signs on the examination of this patient.
Jaundice / spider nevi … etc
What is the Diagnosis ?
Primary biliary cirrhosis.
What is the finding expected on ERCP?
Some said obstruction, others answered normal. We're not sure?
Diagnostic confirmatory test?
Liver biopsy.
What's the treatment for his itching?
Cholestyramine

Q: A 30 YO female patient presented with jaundice & itching. Can't recall the rest of the case!

In lab results there was direct hyperbilirubinemia, AST & ALT were slightly high, ALP = 800, +ve anti-mitochondrial antibody, biliary tree is normal (on US).

1.What's your diagnosis ?
Primary Biliary Cirrhosis.
2. Mention 2 serological test ?
ANA , AMA (antimitochondrial antibodies).
3. Best diagnostic test ?
Liver biopsy
4. Treatment?
Ursodeoxycholic acid or ursodiol fist line of treatment
Liver transplant if less aggressive treatment have failed or develops liver failure.

Q: A 55 year-old woman presents complaining of fatigue for the last 2-3 months, Yellowish discoloration of her sclerae, Arthralgia, & itching. She doesn't have fever, hx is negative for a recent infection, ill- contacts, or blood transfusion.

On examination, HR: 74/min, BP: 128/76 mmHg. Liver is not palpable but the spleen is felt 2 cm under the left costal margin. It is not tender.

All lab investigations for Hepatitis viruses were negative.

Total bilirubin 84 mmol/L (3–17 mmol/L) Direct bilirubin 2 mmol/L (1.0–5.1 mmol/L), ALP 794 IU/L (30–300 IU/L), Gamma-glutamyl transpeptidase 568 IU/L (11–51 IU/L), ALT 63 IU/L (5–35 IU/L), AST 50 IU/L (10 to 34 IU/L).

Ultrasound reveals normal liver, biliary tract, & pancreas., No gall bladder stones, & no dilatation in intra- or extra- hepatic bile ducts.

What is your diagnosis?

Primary biliary cirrhosis

What additional tests will you order?

Antimitochondrial antibodies (AMA), ANA, anticentromere antibodies

What is the diagnostic confirmatory test?

Liver biopsy (Although it's not routinely used to confirm the diagnosis!).

What other diseases are you expecting to accompany this condition?

Sjogrens syndrome, systemic sclerosis, lupus , rheumatoid arthritis, hypothyroidism

Q: A pt presented with pallor, fatigue, cold intolerance, ... The pt also had Vitiligo. [They gave us the result of the pt's CBC which showed that the pt had pan-cytopenia; all the blood elements are low].

What is the most probable diagnosis?

Pernicious anemia.

What's the cause of the patient's "cold intolerance"?

Hashimoto's thyroiditis.

What finding can you see in an upper GI endoscopy for this patient?

Chronic atrophic gastritis.

What is the drug used to treat this condition?

Vit B12 supplements.

Mention the route of administration for this drug.

Intramuscular.

Q: patient presented with bloody diarrhea , fever , cramping abdominal pain:

-mention 3 important investigations to be done in the emergency room .

CBC
urea,creat,electrolytes
stool analysis, stool culture

- What's your first line management ?

IV fluid

Q: A 55 year-old woman presents complaining of fatigue for the last 2-3 months, Yellowish discoloration of her sclerae, Arthralgia, & itching. She has xanthelasma, doesn't have fever, hx is negative for a recent infection, ill-contacts, or blood transfusion.

On examination, HR: 74/min, BP: 128/76 mmHg. Liver is not palpable but the spleen is felt 2 cm under the left costal margin. It is not tender.

Total bilirubin 84 mmol/L (3–17 mmol/L) Direct bilirubin 2 mmol/L (1.0–5.1 mmol/L), ALP 794 IU/L (30–300 IU/L), Gamma-glutamyl transpeptidase 568 IU/L (11–51 IU/L), ALT 63 IU/L (5–35 IU/L), AST 50 IU/L (10 to 34 IU/L). And Hypothyroidism.

Ultrasound reveals normal liver, biliary tract, & pancreas., No gall bladder stones, & no dilatation in intra- or extra- hepatic bile ducts.

What is your diagnosis ?

PBC (primary biliary cirrhosis)

Do you expect ANA to be positive or negative?

Positive in half of the patinets

What is the diagnostic confirmatory test?

Liver biopsy

What are ERCP findings?

Normal ?

What is the cause of xanthelasma?

Hypercholestremia

PBC

-Autoimmune disease

-Middle aged female

-Characterized by destruction of intrahepatic bile duct with portal inflammation and scarring

-Positive antimitochodrial abs in 90% _95% of pts and this is the hallmark of the disease

-Do liver biopsy to confirm the diagnosis

-Treatment is symptomatic + ursodeoxycholic acid (has been shown to slow progression of the disease)

-Liver transplant is the only curative tx

Q: A 40 Year old Woman , with 1 month history of Upper abdominal discmofort , Fatigue , pruritis , on examination she is found to be icteric , and the liver is palpated 3 cm
below costal margin a liver function test ordered and the result was as following :
Albumin 30 g/l
AST 167 u/l
ALT 189 u/l
ALP 170 u/l
Total Bilirubin 30 umol/l
direct bilirubin 12 umol/l

1) what is best test to screen for hepatitis B inf?
Hep ds-DNA?
2)what is the best test to screen for hepatitis C inf?
Hep RNA?
3) if both hepatitis B and C were negative what is the most likely diagnosis ?
Autoimmune
4) Mention 2 seromarkers for this diagnosis ?
ANA, antismooth muscle antibodies

Nephrology

Done by:

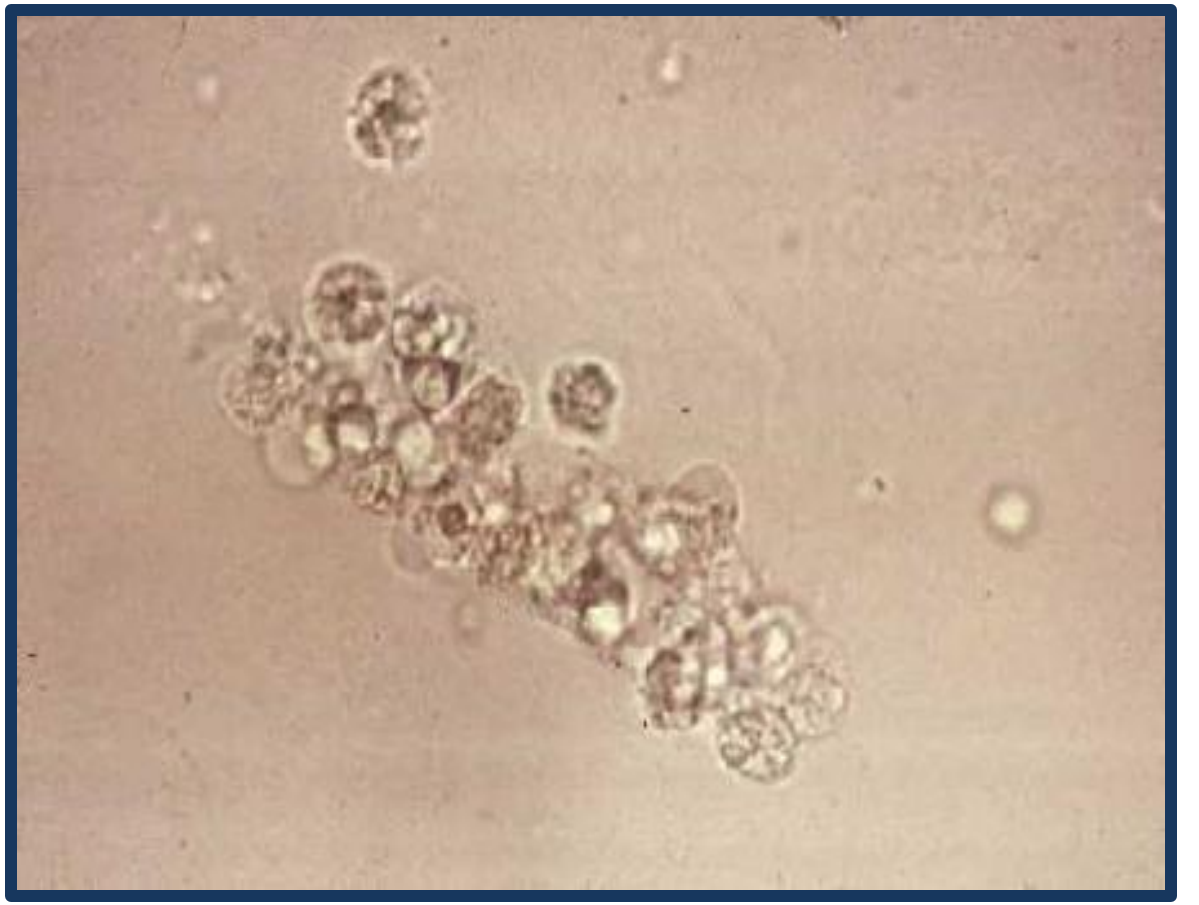
عبدالله بسيسو & محمد عقيلي

حسام عدس & محمود الأسود

لاميس أبوزيد & فاطمة العجارمة

سماح السليحات

- 35-year old patient diagnosed with epilepsy 2 years ago , he came to emergency room complaining of fever and rash , the CBC was done and showed elevated WBC and elevated eosinophils , among his investigations that were done urine microscope result is shown :



Q1 : what does the urine microscope show ?

WBC cast

Q2 : what is the most likely diagnosis that interprets his complains ?

Interstitial nephritis

Q3 : what is the most likely cause of his disease ?

His anti-epileptic drug (phenytoin)

Q4 : what is the most common antibiotic that can cause his disease ?

Cloxacilin

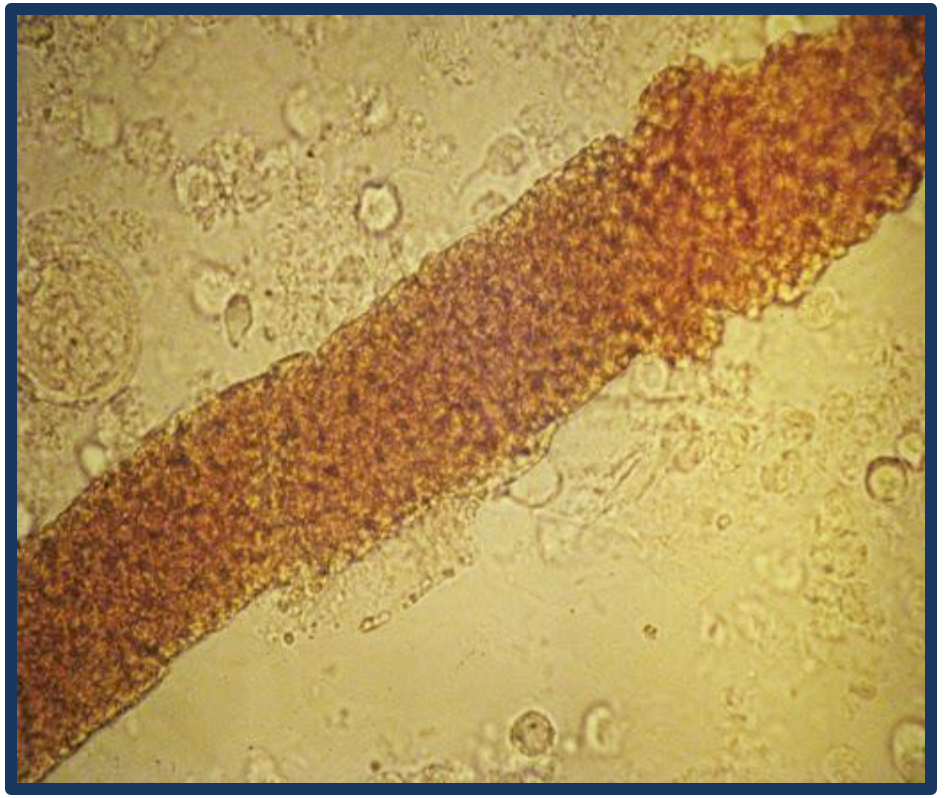
Q5 : what are the other investigations that you may need ?

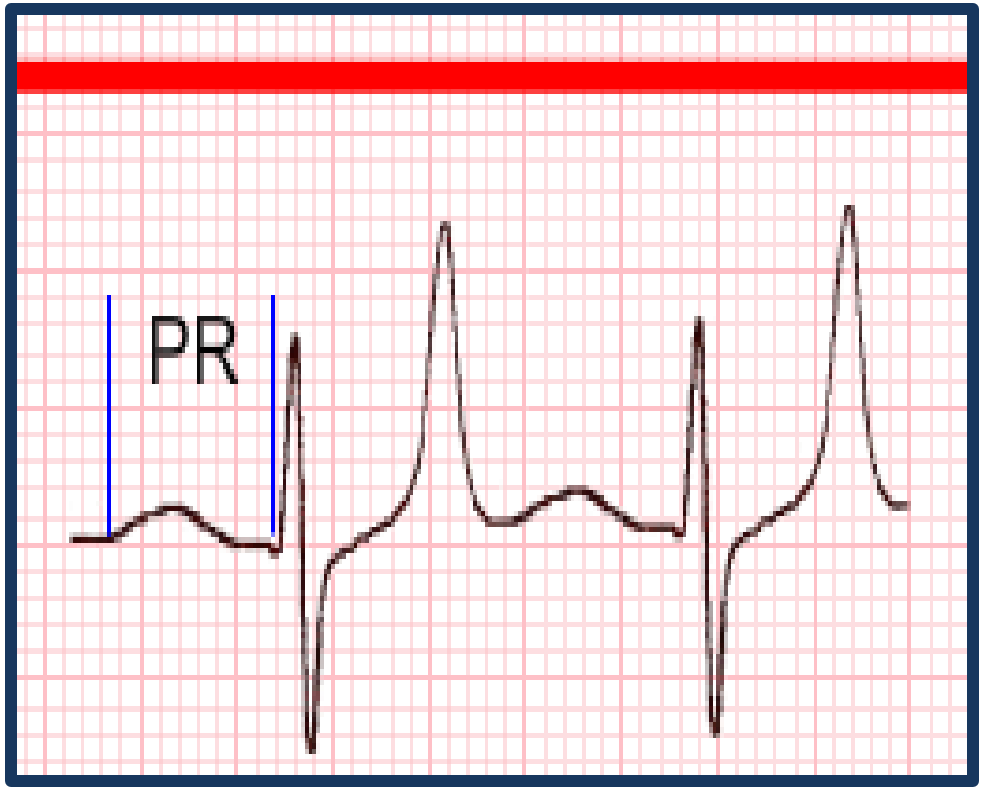
KFT (cr , urea) , electrolytes (K , na) , biopsy (not done usually)

Q6 : what is your management in this case ?

Stop phenytoin and choose other anti-epileptic drug , and then re-assess the cr if it continues to increase give steroid

- 45-year old patient is a known case of hepatitis C (two months ago) , he came to emergency room complaining of oliguria and palpitations , among his investigations that were done urine microscope and ECG results are shown :





Q1 : what does the urine microscope show ?

RBC cast

Q2 : what are the ECG findings ?

Peaked T wave , flattening of P wave (usually it is above this level), prolonged PR interval

Q3 : what do you suspect the most likely cause of urine microscope result in this case ?

Membranoproliferative glomerulonephritis (most common type of glomerulonephritis that associated with hepatitis C)

Q4 : what do you suspect the cause of ECG findings in this case ?

Hyperkalemia due to acute kidney impairment

Q5 : what are the investigations that you may need in this case ?

KFT , electrolytes , kidney biopsy

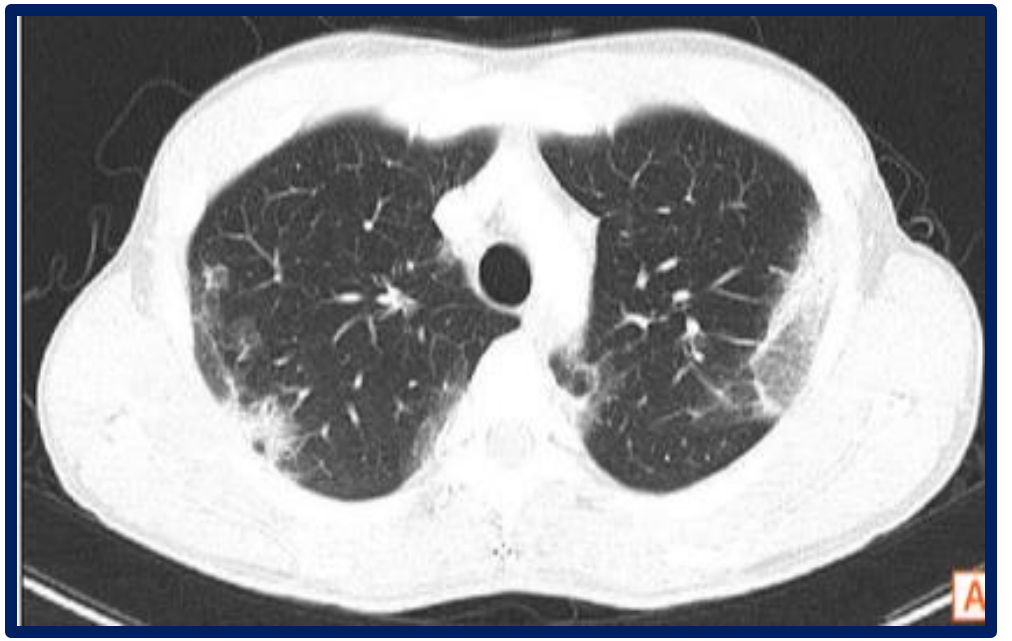
Q6 : what is your management in this case ?

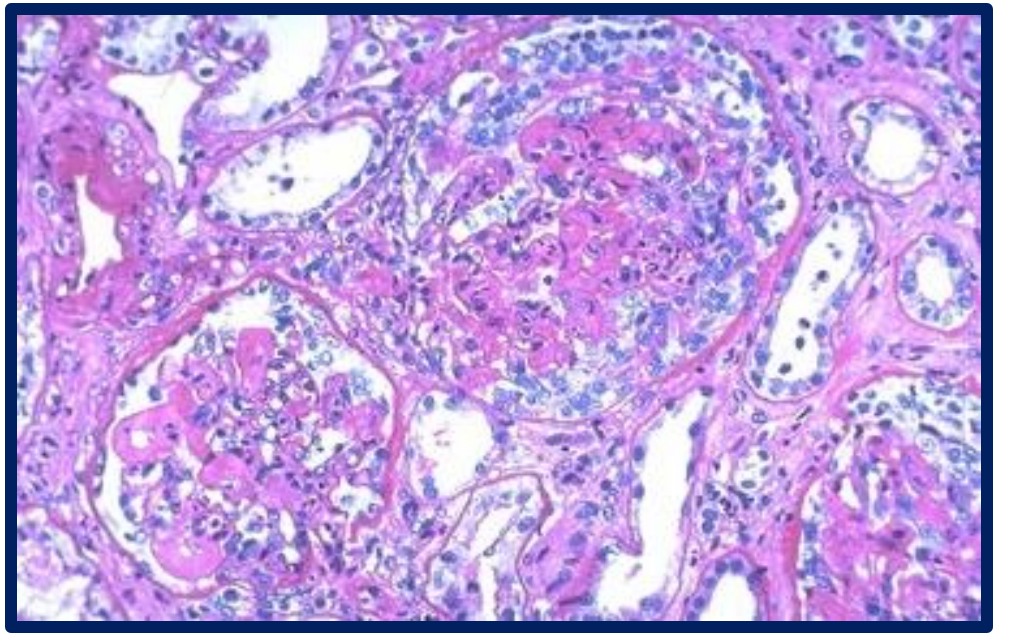
Treat the underlying cause (hepatitis C) , steroid for glomerulonephritis , IV calcium gluconate with cardiac monitoring , IV glucose and short acting insulin , inhalational albuterol may be needed

Q7 : if his ECG findings not improved upon your initial management , what is your last step in management options ?

Hemodialysis

- 27 year-old female patient diagnosed with bronchial asthma and she is compliant to her treatment , but inspite of that her complains not improved , she came to emergency room complaining of SOB and chest tightness , among her investigations that were done P-ANCA is (+) chest CT and kidney biopsy are shown :





Q1 : what is the findings in patient chest CT ?

subpleural opacities

Q2 : what does renal biopsy show ?

crescent proliferation suggesting rapidly progressive glomerulonephritis

Q3 : what is the most likely diagnosis in this case ?

churg-strauss syndrome (eosinophilic granoulomatosis with poliangiitis)

Q4 : what do you suspect to see in her CBC ?

eosinophilia

Q5 : what are the other investigations that you may need ?

urinanalysis , KFT , electrolites

Q6 : what is your management in this case ?

systemic glucocorticoids

- Note :

If glomerulonephritis come after some DAYS of URTI it is IgA glomerulonephritis

If glomerulonephritis come after some WEEKS of URTI it is post streptococcus glomerulonephritis

Case 1

Clinical case scenario

26-year-old female previously healthy, her weight 45kg and use a dighragm as a method of contraception on routine physical exam :

blood pressures was 166/100 mmHg another reading was taken and bp was 158/94

Pulse 72 beat per minute regular ,good volume no radio-radial or radio – femoral delay

Abdominal and chest exam was unremarkable

What to do next for her ?

Consider it as hypertension but you need to confirm that by 2 measurement in the next visit

Note :diagnosis of hypertension require two or more properly measured, seated BP readings On each of two or more office visits.

On the next visit the blood pressure measured twice and still elevated so hypertension is confirmed and because she is a young take it seriously (secondary hypertension).

What investigation to do for her ?

1. investigation for effect of HTN on organs (ecg for LVH ,urinalysis ,fudal exam for retinopathy)
2. Routine screening lab :glucose ,electrolytes ,creatinine,GFR ,total cholestrol ,HDL , TFT)

The previous investigations was normal except for stage 2 hypertensive retinopathy on fundal exam K: 3 meq/l NA :145

What further investigation you will order ?

Serum level of renin , aldosterone , renin / aldosterone ratio , CT SCAN

Result : aldosterone elevated , low plasma renin

so you think of primary hyperaldosteronism(conns or bilateral adrenal hyperplasia)

How to differentiate between them ?

Saline infusion test (its called suppression test) or stimulation test (captopril)

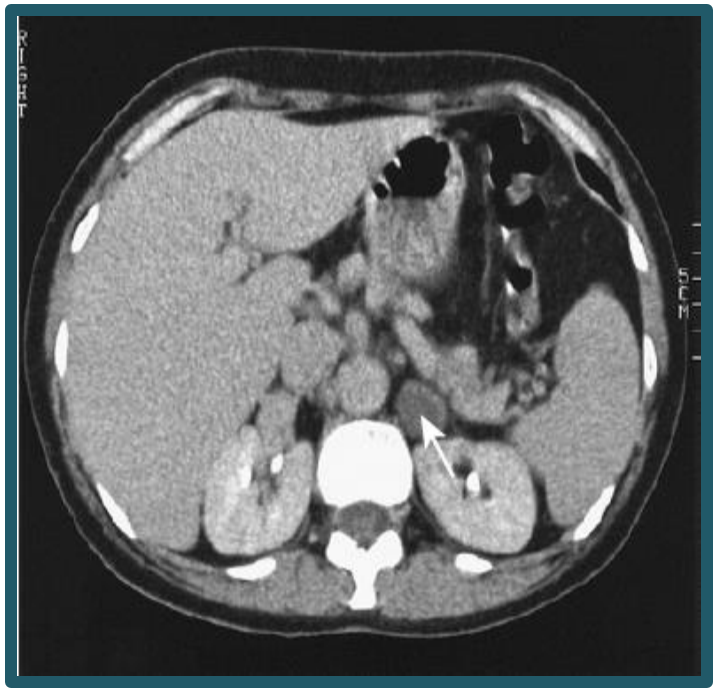
Result of saline suppression :

persistent elevation of aldosterne so its conns (aldosternoe secreting adenoma) order CT SCAN to localize tumor

Note if the cause of primary hyperaldosteronism is bilateral adrenal hyperplasia saline suppression test will decrease the serum aldosterone while in conns not (because its autonomous secretion)

Q: Result of CT scan

adenoma in left adrenal gland



Q: Treatment for this patient ???

1. Mineralocorticoid receptor anagonist prior to surgery(to correct electrolyte abnormality)
2. Adrenal vein catheterization and adrenalectomy
3. after surgery 70% has persisitent HTN so you should give minralocorticoid receptor antagonist

Notes

**Mineralocorticoid receptor antagonist like :spironolactone ,eplerenone

**adrenal vein cathetarization is important during surgery to confirm site of adenoma right or left because CT may visualize non functioning adenoma as aldosterone secreting adenoma (false positive) .

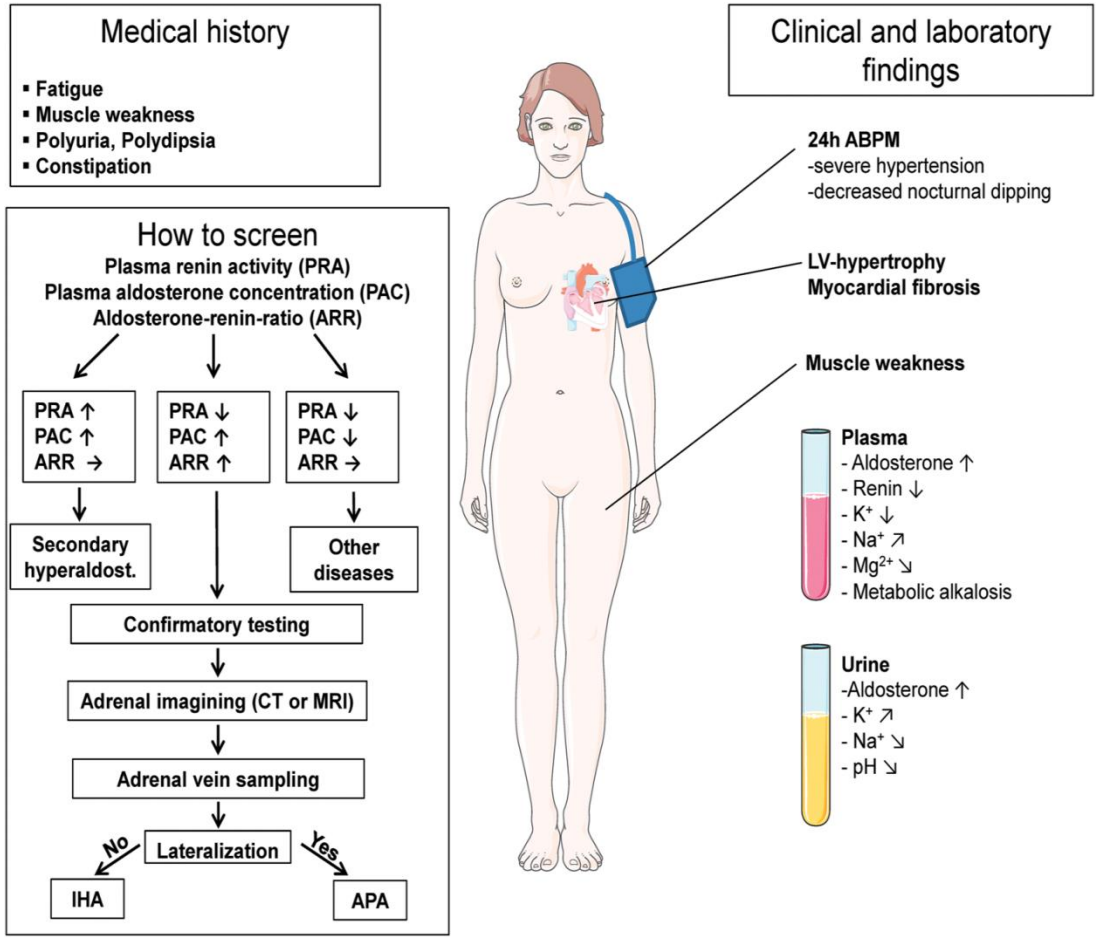
**Dr mdallal said that :in primary hyperaldosteronism potassium not always low it could be at lower normal limit .

**Saline suppression test and stimulation test (captopril ,furesemide) used to differentiate causes of primary hyperaldosteronism is it conns or bilateral adrenal hyperplasia .

If there is decrease in aldosterone after suppression test it means that secretion not autonomous (not conns) so its bilateral adrenal hyperplasia

If the cause of hyperaldosteronism is bilateral adrenal hyperplasia we don't do bilateral adrenalectomy we give only mineralocorticoid receptor antagonist

Figure 5 Medical history, clinical findings, and screening work-up in patients with suspected primary aldosteronism. ...



European Heart Journal, Volume 35, Issue 19, 14 May 2014, Pages 1245–1254,
https://doi.org/10.1093/eurheartj/eht534

New case

- A 15-year-old gypsy girl student with a history of migraines diagnosed 2 years before the current clinical picture, began having symptoms of non-pulsatile frontal headaches 9 months before hospital admission, with worsening symptoms in the last 2 months associated with palpitations. She turned to the emergency room (ER) where she presented AHT (blood pressure (BP) 160/123 mm Hg) for which she was then treated, discharged and referred to her treating physician (without medication).
- On 9 February 2012, she returned to the ER because of worsening headaches and new visual symptoms (blurred vision of the left eye). She was evaluated by ophthalmology, which observed the following changes in visual acuity: right eye 9/10 and left eye 4/10. Funduscopy and angiography revealed small venous occlusions with sparing of the macula. The patient was medicated (eye drops) and referred to ophthalmology, paediatrics and neurology consults, which she failed to attend.
- On 10 February 2012, she turned to the ophthalmology ER because of worsening visual symptoms: bilateral 'cloudy' vision most pronounced in the left eye, presenting aggravated retinopathy with macular detachment and superficial peripapillary haemorrhages. She was referred to a consult and observed 6 days after with worsening visual acuity (right eye: 1/10, left eye with the capability of only counting fingers). Funduscopy with cotton wool spots, haemorrhages and macular oedema were most pronounced in the left eye, without oedema of the optic disc. The patient was transferred to the general ER and referred to Internal Medicine with the diagnosis of hypertensive crisis. The patient denied relevant pathological medical history including smoking, alcohol use or drug consumption, and had had no previous hospital admissions. She was on no regular medication, including oral contraceptives.
- Regarding her family history, the patient's mother was diagnosed with HTN at the age of 38 years and chronic kidney disease. No familial hereditary diseases were known.

Physical exam

Medical examination showed BMI of 17.5 kg/m^2 and no skin lesions. BP was 187/139 mm Hg—overlapping values in all four limbs with no asymmetric pulse. Heart rate was 130 bpm—rhythmic and without heart murmurs, abdomen without murmurs or palpable masses and without oedema. Funduscopy with grade II retinopathy, was without papillary oedema. The remaining neurological examination was normal.

Labs

Blood work with complete blood count, renal function and hepatic enzymes, was normal. Urine was without proteinuria. ECG showed sinus tachycardia, HR of 126 bpm and signs of left ventricular hypertrophy. Chest radiograph and cerebral CT were normal.

The patient was admitted with the diagnosis of severe HTN and retinopathy. She began perfusion of labetalol until BP control was achieved, with progressive clinical improvement, and regression of headaches and visual symptoms.

The clinical picture of recurrent HTN in a young patient made us suspect underlying secondary hypertension.

According to history and physical exam what's the most likely diagnosis ?

Pheochromocytoma

Findings support diagnosis in history and physical exam :

1-episodic attack of HTN

2- sweating

3- tachycardia

What further lab investigation you order to confirm the diagnosis?

- serum, urinary catecholamines metanephrines
- parathyroid hormone
- Renal u/s
- Thyroid , parathyroid u/s (maybe men syndrome)
- CT scan (chest ,abdomen to exclude extradrenal sites of pheochromocytoma)

Lab result

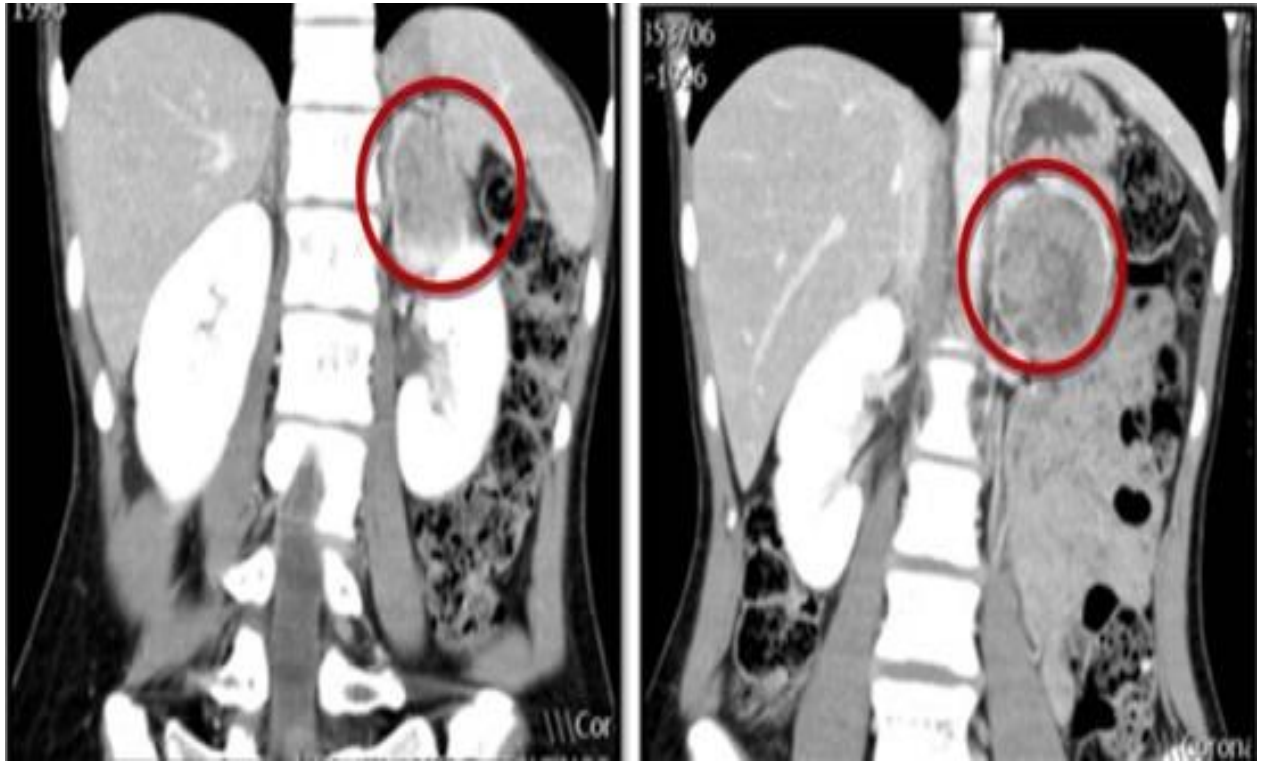
Table 1

Laboratory tests

		Ref.
Fasting glucose (mg/dL)	**97**	74–106
PTHi (pg/mL)	**111.5**	7.8–53.8
Renin (pg/dL)	16.7	1.1–16.5
Aldosterone (ng/dL)	11.8	5–14.5
Cortisol (µg/dL)	11.8	8.7–25
ACTH (pg/mL)	22.8	<46
Calcitonin (pg/mL)	<2	<5
Phosphorus (mg/dL)	5.5	2.8–4.8
Calcium corrig (mg/dL)	9.8	8.4–10.3
Chromogranin A (nmol/L)	**47.7**	<6
Serum catecholamines (ng/L)		
Total	**22 326**	<598
Norepinephrine	**20 591**	<420
Epinephrine	**1318**	<84
Urinary metanephrines (µg/24 h)		
Metanephrines	**2570**	<90
Normetanephrines	**>4800**	<180

Values in bold

- Renal Doppler ultrasound revealed a solid nodule in left adrenal gland and normal permeability of the renal arteries.
- Abdominal CT scan confirmed the presence of a heterogeneous nodular 6.5×5.3×6.0 cm mass in the left adrenal gland, compatible with a pheochromocytoma

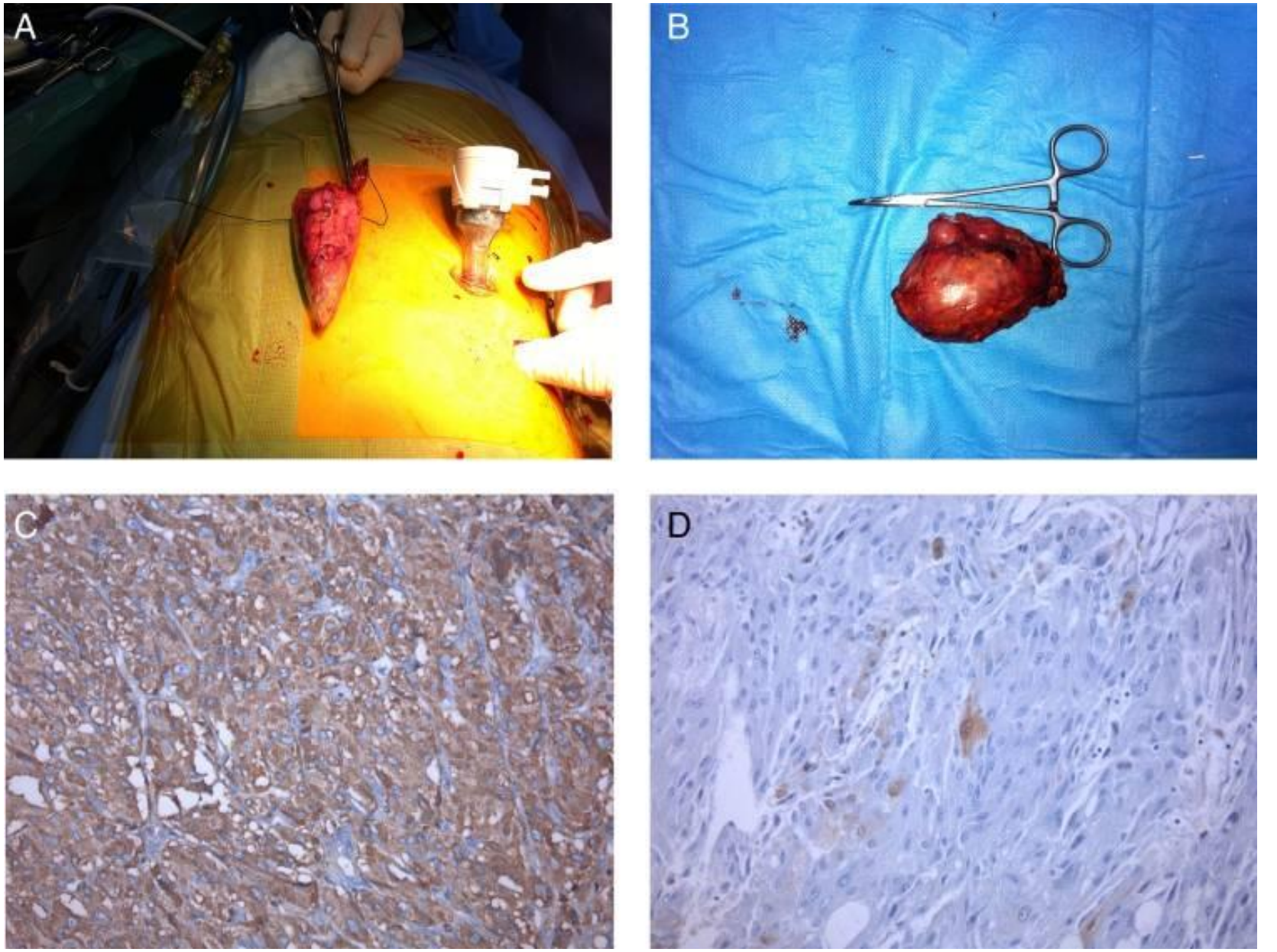


Thyroid and parathyroid ultrasound revealed a small nodular 6×9×4 mm formation posteroinferior to the left lobe, suggestive of a probable parathyroid.

With the diagnoses of pheochromocytoma and associated hyperparathyroidism in a young patient, although no lesions suggestive of thyroid carcinoma existed, possible MEN was admitted, so genetic testing was performed, which revealed a negative RET gene.

Treatment ??

- Therapy with a and posteriorly β-adrenergic blockers was started (intradermal phenoxybenzamine 10 mg and propranolol 20 mg) with adequate BP control.
- The patient was submitted to laparoscopic left adrenalectomy from a retroperitoneal approach on 28 March 2012.
- A 6 cm tumour was removed and the pathological examination confirmed the diagnosis of pheochromocytoma


A
B
C
D

New case

- John is a 35 years old male work as industrial engineer comes to clinic complaining of general fatigue ,poor concentration ,excessive sleep during day , he said that he sleep 8hours at night despite that he awake fatigued , his wife also said that he has excessive snoring during sleep .

On physical exam :

BMI :40

short neck

blood pressure 150/95 mmhg

- Whats the most likely diagnosis?

Obstructive sleep apnea

- What is the diagnostic test

Overnight oxygen saturation trace

- Treatment ?

Weight reduction

If alcoholic advice to stop

CPAP by nasal mask to keep airway patent (improve day time performance ,quality of life and survival)

- What is risk factor for this condition

Obesity

Gender :male

Recessed mandible

Short neck

Acromegaly ,hypothyroidism

Alcohol sedative

Familial :maxilla-mandible back set

Case

- A patient with poorly controlled IDDM missed his insulin for 3 days.

pH 7.1 HCO3 8 mEq/l PaCO2 20 mmhg Na 140 mEq/l CL 106 mEq/l and urinary ketones +++

- Diagnosis

Metabolic acidosis

- The anion gap expected to be

Anion gap = na – (HCO3 + cl)

140-(106+8)= 26

(high anion gap) in case of high AG can calculate

Gap_gap = raising in AG =?Falling in HCO3

(26-12 < 24 -8)

so its coexisting with normal anion gap metabolic acidosis

- Expected compensation

Exp Pco2 = 1.5*actual HCO3 +8 -/+2

= 1.5*8+8=20

So it match with actual Pa co2 >> primary metabolic acidosis not associated with other respiratory disorder

High Anion Gap Metabolic Acidosis

M	METHANOL
U	UREMIA -ARF/CRF
D	DIABETIC KETOACIDOSIS & other KETOSIS
P	PARALDEHYDE, PROPYLENE GLYCOL
I	ISONIAZIDE, IRON
L	LACTIC ACIDOSIS
E	ETHANOL, ETHYLENE GLYCOL
S	SALICYLATE

Metabolic Acidosis

Causes of normal Anion Gap metabolic acidosis

H	Hyperalimentation
A	Acetazolamide
R	Renal tubular acidosis
D	Diarrhoea
U	Uretero sigmoidostomy
P	Pancreatic fistula

- A case of hepatic failure has persistent vomiting

pH 7.54 HCO3 38 mEq/L PaCO2 44 mmhg

- What is the ABG showed ?

Metabolic alkalosis

- Expected compensation (rise in PaCO2) will be

Every 1 mEq change in Hco3 will change PaCo2 0.6

Rise in PaCO2= 0.6 X rise in HCO3= 0.6 X (38-24) =0.6 X14=8.4

- So expected PaCO2 will be 40+8.4 =48.4 mmhg. But actual value of PaCO2 is lesser than expected PaCO2 (44 vs 48.4 mmhg) which suggests presence of additional respiratory disorder (respiratory alkalosis ... actual value of PaCO2 is lesser than expected PaCO2 , if its higher >> respiratory acidosis) SO pateint have Mixed disorder metabolic alkalosis and respiratory alkalosis **

Causes of Acid-Base Balance

Metabolic Acidosis Diabetic ketoacidosis Diarrhea Renal failure Shock Aspirin overdose Sepsis	**Metabolic Alkalosis** Loss of gastric secretions Overuse of antacids K+ wasting diuretics
Respiratory Acidosis Hypoventilation COPD Airway obstruction Drug overdose Chest trauma Pulmonary edema Neuromuscular disease	**Respiratory Alkalosis** Hyperventilation Hypoxia Anxiety High altitude Pregnancy Fever

- Following sleeping pills ingestion, patient presented in drowsy state with sluggish respiration with respiratory rate 4/min.

pH 7.1 HCO3 28 mEq/L PaCO2 80 mmhg PaO2 42 mmhg

Respiratory acidosis

- Is it Acute OR chronic respiratory disorder???

Acute: Every 10 mmHg change in PaCo2 leads to change pH 0.08.

Chronic: Every 10 mmHg change in PaCo2 leads to change pH 0.03.

Δ pH = 7.4 – 7.1 = 0.3..... So It is Acute Disorder

** Expected Hco3 : Every increase Co2 (10 mmHg) leads to increase Hco3 (1 mEq)

= 24 + 4 = 28 mEq/ L which matches with actual HCO3,

which is 28mEq/l, suggestive of simple ABD.

- So, the patient has primary respiratory acidosis due to respiratory failure, due to sleeping pills.

** If HCO3 lower than expected >> associated with metabolic acidosis , if it higher than

- A 15 year old boy is brought from examination hall in apprehensive state with complain of tightness of chest.

pH 7.54 HCO3 21 mEq/L PaCO2 21 mm of hg

** Respiratory alkalosis

- Is it Acute OR chronic respiratory disorder???

Acute: Every 10 mmHg change in PaCo2 leads to change pH 0.08.

Chronic: Every 10 mmHg change in PaCo2 leads to change pH 0.03.

Δ pH = 7.54 – 7.40 = 0.14… So It is Acute Disorder

** Expected Hco3 : Every ↓ Co2 (10 mmHg) leads to ↓ Hco3 (2 mEq)

= 24 – 4 = 20 mEq/ L which almost matches with actual HCO3, which is 21 mEq/l, suggestive of simple ABD

** So the patient has primary respiratory alkalosis due to anxiety….

If HCO3 lower than expected >> associated with metabolic acidosis , if it higher than expected >> metabolic alkalosis

CASE1

You are called to see a 19-year-old woman in complaining of a 2-day history of frequency, dysuria and urgency. She has a temperature of 39.8°C with some right loin pain.Yesterday she had a rigor. She tells you that this is her first episode of an UTI. She has no vaginal discharge and has never had a history of sexually transmitted diseases

Q1:what is the diagnosis,and most common organism?

uncomplicated pyelonephritis,E.coli

Q2:what are the initial investigations for this patient?

urinanalysis,gram stain+urine culture,CBCwith differential ,RFT

Q3:what is the treatment and for how long?

single parenteral dose of ceftriaxone, or of gentamicin, followed by oral fluorquinolones orTMP\SMX for gram-ev and amoxicillin for gram+ev, for 10-14day.

Q4:Mention Prophylactic measures to prevent further UTIs that should be advised.

- A 2 L daily fluid intake
- Voiding before bedtime and after intercourse
- Avoidance of spermicidal jellies and bubble baths and other chemicals
- in bathwater
- Avoidance of constipation

CASE2

An 84-year-old woman, a nursing home resident with Alzheimer disease,is brought to the emergency room for agitation and confusion. She is found to be febrile, tachycardic, and hypotensive. Examination shows flat neck veins, clear lung fields, and no cardiac murmur or gallops; her limbs are warm and well perfused.Her hemodynamic status has improved with a fluid bolus. Laboratory examination shows evidence of a urinary tract infection (UTI).

Q1:what is the diagnosis?

Shock, most likely as a consequence of urosepsis.

Q2:what is the initial the management of this patient?

- intravenous (IV) fluids or vasopressors as necessary. Broad-spectrum

antibiotics should be started as soon as possible

Q3:which investigations should you order?

urinanalysis,gram stain+urine culture,CBCwith differential ,RFT,blood culture

Q4:which antbiotics could be used for her UTI, and for how long?

IV Ampicillin+gentamicin OR fluorquinolones (cipro or levo)for 2-3weeks

CASE3

a 45 -year -old woman who presents with a 4 -day history of urinary frequency and dysuria. On examination, her temperature is 38 ° C, pulse 90 bpm and BP 125/75 mmHg. She is mildly tender in the right flank and suprapubically but there is no rebound or guarding. This is the third UTI in 9 months.

Q1:what is the most likely dignosis?

complicated UTI

Q2:what are the initial tests for this patients?

urinanalysis,gram stain+urine culture,CBCwith differential ,RFT

Q3:mention three causes for her recurrent UTI.

Diabetes mellitus• Immunosuppression• Pregnancy(or others)

Q4:how would you further investigate her ?

screen for diabetes,KUB X-RAY,US,IVP,CT...etc

Q5:how would you prevent her recurrent UTIs?

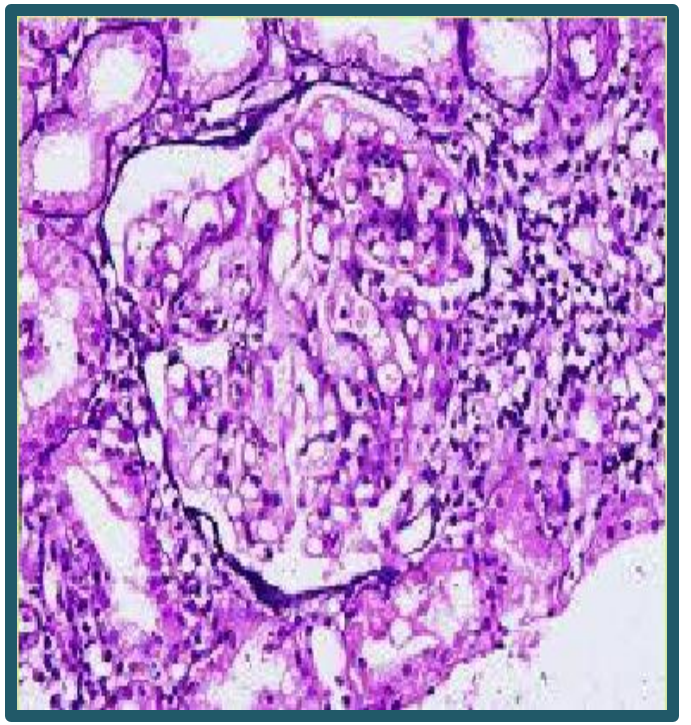
- Single dose of TMP/SMX after intercourse or at first signs of symptoms.
- Alternative low-dose prophylactic antibiotics (e.g., TMP/SMX) for 6 months.

Q: 1. This biopsy is taken from which organ?

Kidney

2. Mention 1 indication.

Nephrotic syndrome (extereme ages, resistant to steroids .. Etc)/ Nephriticsyndrome … etc

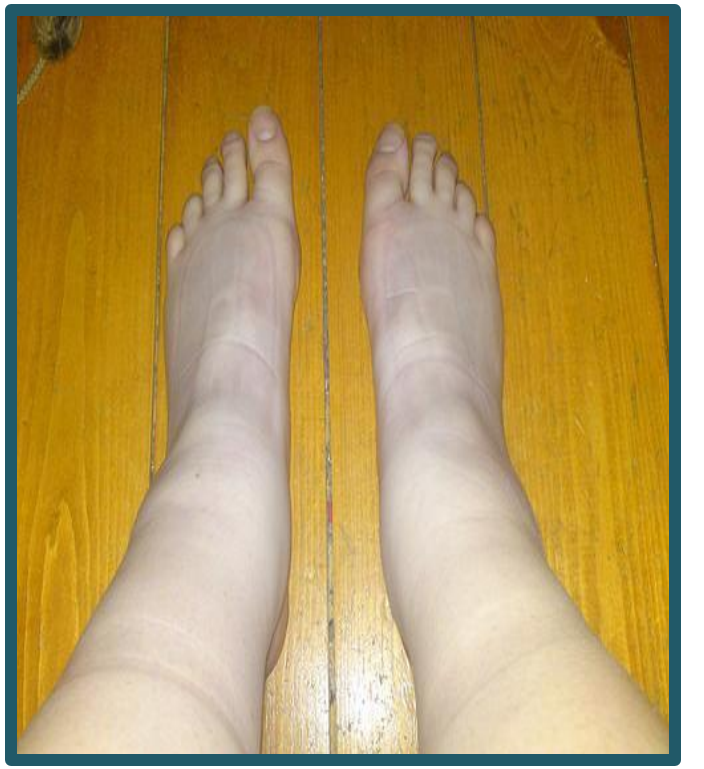


Q: This pt admitted with of bilateral lower limb pitting edema, & puffy eyes. He is a known case of Diabetes. What do you think this pt have?

nephrotic syndrome

What is the best test to start with in this case?

urinalysis, 24 hour urine collection.



Q: A known case of diabetes presented complaining of bilateral lower limb edema & facial puffiness.

What is your diagnosis?

Nephrotic Syndrome due to Diabetic nephropathy

What is the confirmatory test?

24- hour urine protein collection

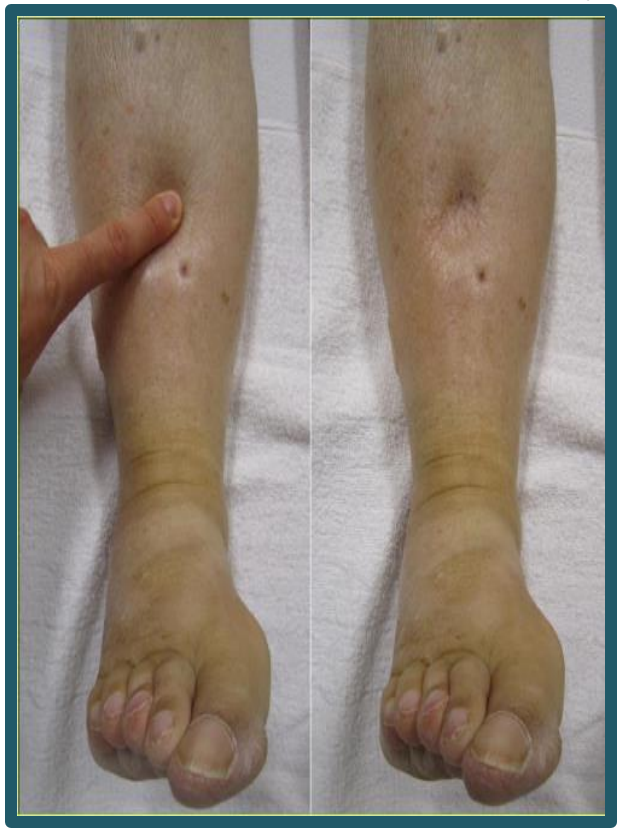
Q: A 50-year old diabetic patient developed the following.

A-What is your diagnosis?

DM nephropathy

B-What is the first lab investigation to be done?

24-hour urine collection for protein

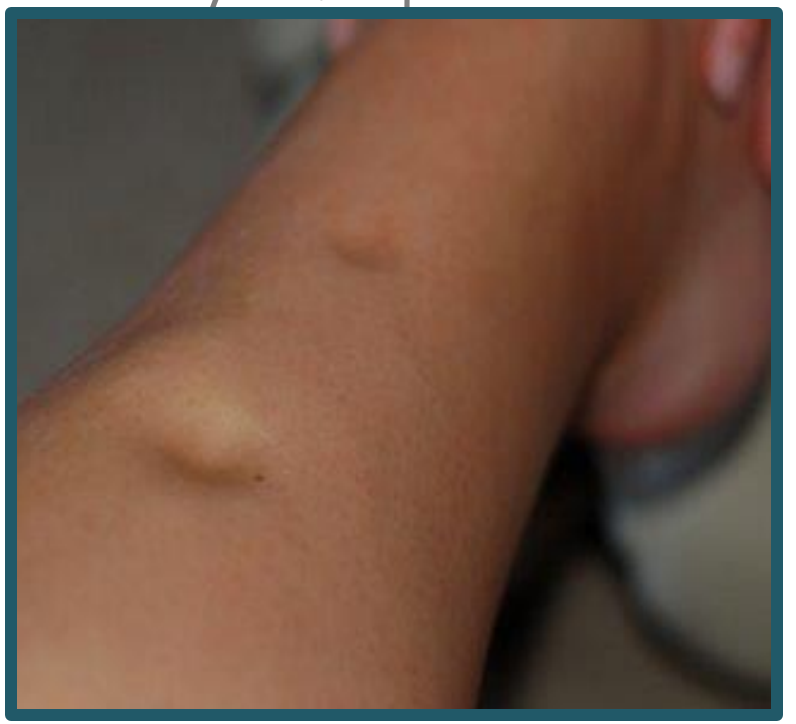


Q: A 48 year old diabetic patient presented with bilateral lower limb edema and frothy urine. What is the cause of his condition

Nephrotic Syndrome (Due to Diabetic Nephropathy)

What is the test you want to do for him?

Urine Analysis for proteinuria.



Q: 34 YO male presented with bilateral lower limb edema, puffiness of face, peri-orbital edema. 24-hour urine collection sample showed 5.4g protein

1- What other 2 findings you suspect to have in the serum of this patient ?

Hypoalbumenia/Hyperlipidemia.

2- write 2 causes that would lead to his condition.

Amyloid , Diabetic nephropathy

3- what is the diagnostic test that will give you the etiology & guide your treatment ?

Kidney biopsy

4-What is your diagnosis?

diabetic nephropathy.

5-Mention 2 other possible lab findings in this case.

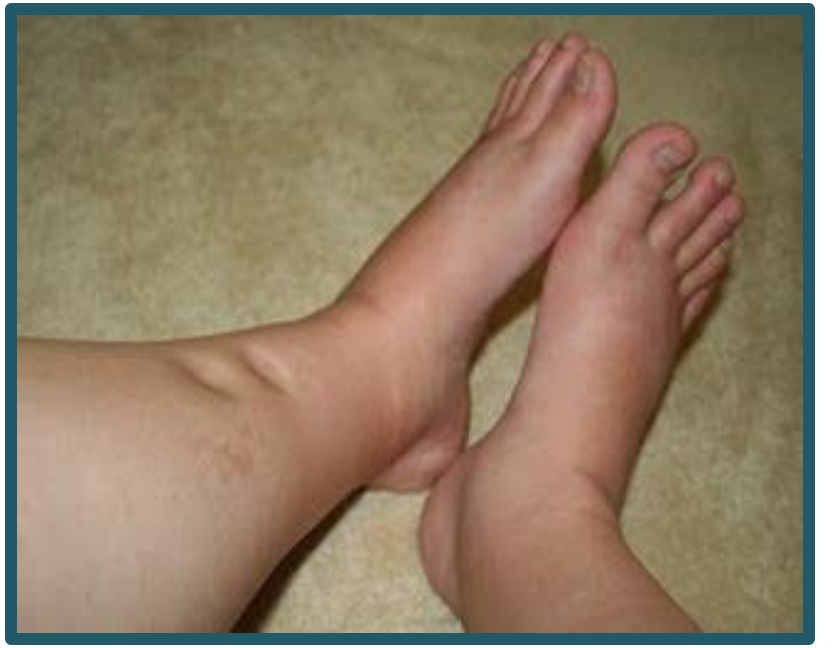
Hyperlipidemia and hypoalbuminemia

6-What is the most appropriate treatment in his case?

control HTN and diabetes , give ACEI for example

Q: A female pt visited your clinic complaining of bilateral leg swelling & peri-orbital edema. She is a known case of DM which was controlled until 3 months ago. She developed HTN 3 months ago, but was not controlled even with 2 drugs. On examination she has mild respiratory distress & large edema in her legs.

A- What is your most likely Dx?
Nephrotic Syndrome.
B- Mention 2 confirmatory tests.
24h urine collection for albumin (> 3.5 gm) / Serum albumin (dec.) /Serum lipids profile(inc.).
C- Mention 2 lines of management for this pt.
Steroids /Prophylactic Anticoagulants/ Diuresis
D- Mention 4 causes of this condition.
Heart failure
Renal failure,
Nephrotic syndrome
Liver cirrhosis
Hypo-albuminemia
Fluid overload

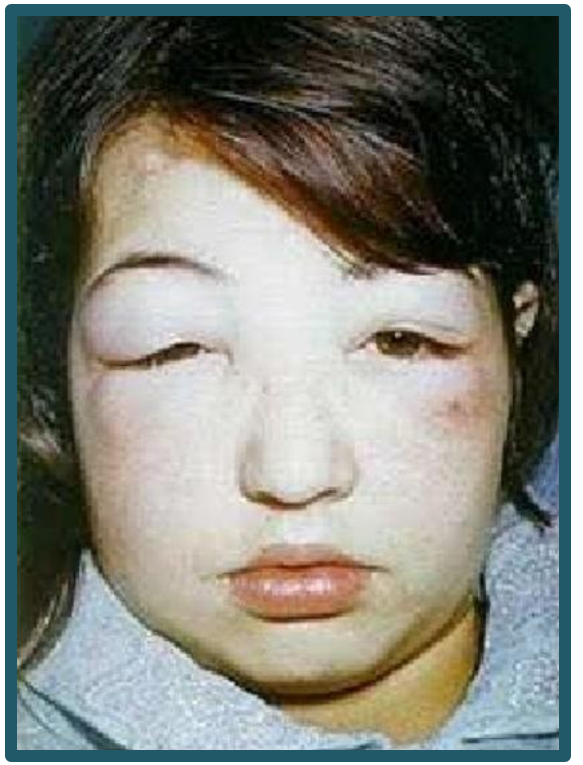


Q: female pt with frothy urine , DM , edema around eyes, what is your 2 lab findings?

hypoalbuminemia

Hyperlipidemia

Proteinuria



Q: 67 YO woman presents with SOB on exertion &bilateral ankle edema that she noticed just today. UA/ 24 hour urine 3+ Protein, low Albumin-3.4 g/dL (3.5-5g/dL).

Q1: What is the most likely diagnosis?

Nephrotic syndrome.

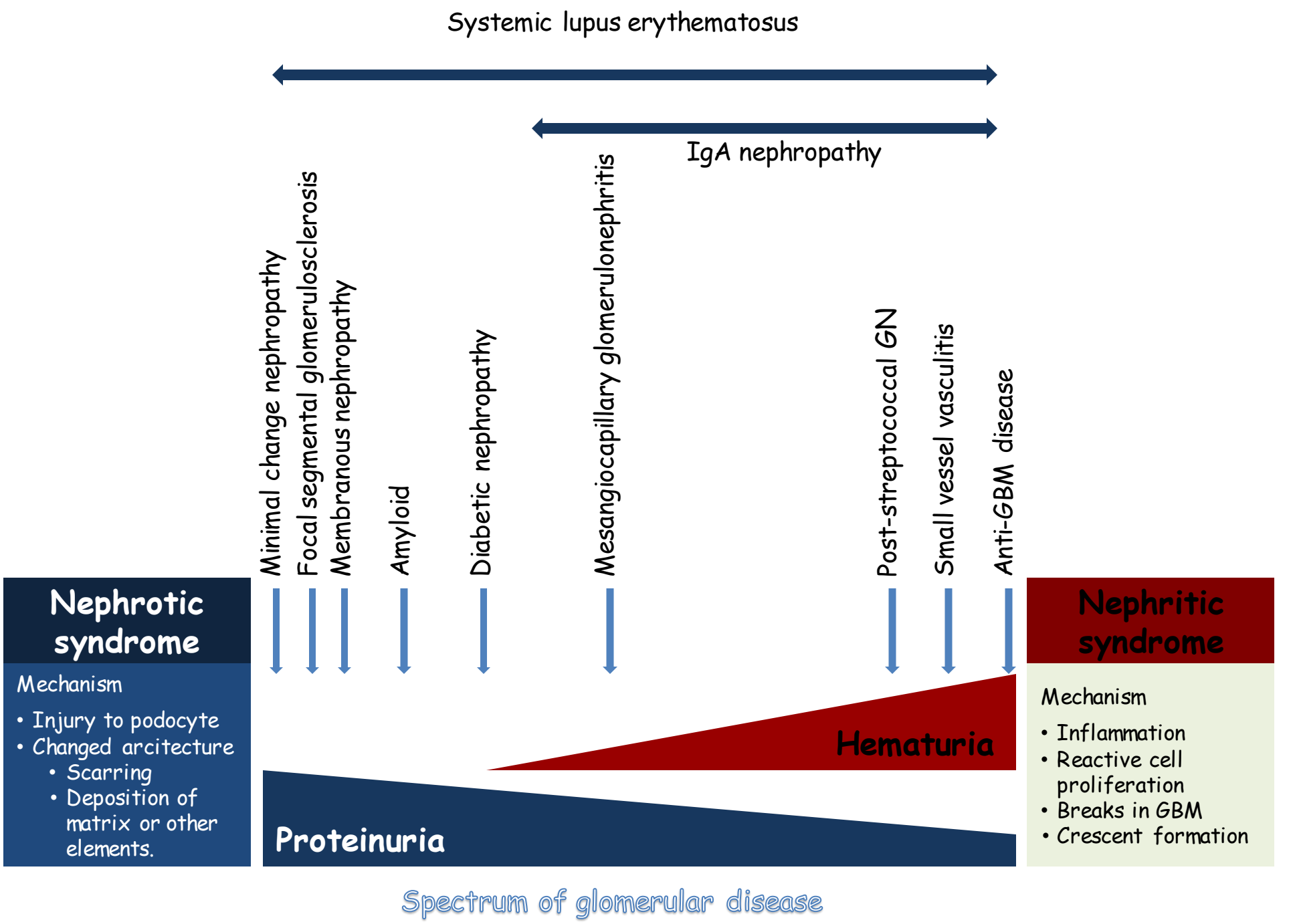
Q2: mention 2 common secondary causes of Dx?

DM, SLE , lymphoma.

Q3: mention 2 complications related to the Dx?

Increased chances of infection,

Hypercoagulability.



Spectrum of glomerular disease

Q: Patient x , 67 years old , with 10 years history of HTN and DM , present with bleeding gum , and epistaxis , pruritus , arrythmia , on exam has astrexis , labs indicate metabolic acidosis and hyperkalemia

Q1 what is ESRD ??

that form of kidney failure so severe as to need dialysis or renal transplantation.

- ESRD is not defined as a particular BUN or creatinine. ESRD is defined as the loss of renal function leading to a collection of symptoms and laboratory abnormalities also known as uremia.

- Uremia is a term interchangeable with the conditions for which dialysis is the answer as therapy.

Q2 what are the etiology ??

The most common causes of end-stage renal disease (ESRD) requiring dialysis are diabetes and hypertension. The next most common cause is glomerulonephritis (15% of cases), followed by cystic disease and interstitial nephritis (each 4–5%).

- ESRD usually implies disease that has been present for years; however, rapidly progressive glomerulonephritis is so named because it can lead to ESRD over weeks.

- Q3 what are the manifestations ??

anemia

hypocalcemia

hyperphosphatemia

hypermagnesemia

osteodystrophy

bleeding

infection

pruritus

- Q4 what treatment for manifestation ??
- Anemia Erythropoietin replacement and iron supplementation
- Hypocalcemia and osteomalacia Replace vitamin D and calcium
- Bleeding Desmopressin (DDAVP) increases platelet function; use only when bleeding
- Pruritus Dialysis and ultraviolet light
- Hyperphosphatemia Oral binders
- Hypermagnesemia Restriction of high-magnesium foods, laxatives, and antacids
- Atherosclerosis Dialysis
- Endocrinopathy Dialysis, estrogen and testosterone replacement

- **Q5 what are the indications for dialysis ??**

1 metabolic acidosis ph < 7.1

2 symptomatic hyperkalemia , K > 6.5 mEq/L

3 ingestion of toxic alcohols salicylate lithium

4 volume overload

5 symptomatic uremia (enchephalopathy , pericarditis , bleeding)

- **Q6 what are the advantages of renal transplantation ??**

The advantages of renal transplantation over dialysis are: - Better survival and quality of life.

- Anemia, bone disease, and hypertension persist in spite of dialysis: these are better controlled with transplantation.
- Transplant patients have a return of normal endocrine, sexual, and reproductive functions, and enhanced energy levels; thus, returning to fulltime employment and more strenuous physical activity is possible.
- In diabetics, autonomic neuropathy persists or worsens after dialysis; whereas, it stabilizes or improves with transplantation.
- Expected survival rate after transplantation is 95% at one year and 88% at five years.

Acute kidney injury

sudden and often reversible loss of renal function, which develops over days or weeks.

Defined as an increase in serum creatinine by 0.3ml/dl or by 1.5 fold over baseline within 48 hrs or by oliguria (< 0.5ml/kg/hr) for at least 6 hrs . Anuria(< 50ml/day).

AKI can be either non-oliguria ,oligouria or anuria.

Elderly patients are at higher risk of developing AKI and have a worse outcome

Cause:

1- Pre-renal 70% : when perfusion to the kidney is reduced, (If the insult is not corrected, this may lead to 'renal' injury: namely, acute tubular necrosis (ATN)).

2- Intrinsic renal 20%:ATN ,interstitial and glomerular disease.

3- Post-renal: when there is obstruction to urine flow at any point from the tubule to the urethra (external compression of urinary tract or intraluminal/ intratubular).

Q: Hx of a hospitalized patient with HTN , DM underwent cardiac catheterization , taking multiple medications , a contrast CT was done to him , presented with Acute kidney injury .

1. Mention 3 causes of hospital induced renal failure.

ATN (ischemia), Contrast nephropathy , acute interstitial nephritis (AIN) (drugs: PPIs is the most common cause)

2. True or False about Kidney Injury Molecule 1 (KIM-1)

1- novel biomarker for human renal proximal tubule injury. True

2-not affected by UTI or chronic kidney failure. True ?????

3- not affected by cardiac catheterization. False

3. What to see in labs can be differentiated between cause of AKI:

Table 4-11: Acute Kidney Injury Labs and Clues

Category	Causes	FE_{Na}	$FE_{Uric\ acid}$	FE_{Urea}	U_{Osm}	Urine Na^+	Urine Sediment	Suspect in Patient with ...
Prerenal	Volume depletion Decreased EABV* NSAIDs ACEI	< 1%	< 12%	< 35%	> 400 mOsm/L	< 20	Normal Granular casts Hyaline casts	Bleeding CHF Cirrhosis/hepatorenal Abdominal compartment syndrome (ACS) Nephrotic syndrome GI fluid loss (nausea/vomiting/diarrhea)
Intrinsic renal	Diseases of, or damage to, the glomeruli, tubules, or interstitium	ATN* > 2% GN* < 1%	> 20%	> 50%	300–350 mOsm/L	> 20	Red cell casts and/or protein (GN) Dirty brown casts (ATN*) Eos (AIN*)	Infections SLE Vasculitis Drugs (aminoglycosides, amphotericin, cisplatin, NSAIDs) Contrasts/IV dyes Atheroembolism Heroin Myeloma Diabetes HTN Hypotension, shock
Postrenal	Obstruction	Varies	Varies	Varies	Normal	Normal	Hematuria	Elderly males Colicky pain

Fractional excretion**	Level indicating prerenal AKI	Changed by diuretics
FE_{Na}	< 1	Yes
FE_{Urea}	< 35	No
$FE_{Uric\ acid}$	< 12	No

*EABV = effective arterial blood volume
ATN = acute tubular necrosis
GN = glomerulonephritis
AIN = acute interstitial nephritis
**Recent diuretics use can alter the FE_{Na} and, in this setting, FE_{Urea} and $FE_{Uric\ acid}$ are more reliable.

4. If we assume that this case is a renal, what makes you suspect that by history and examination well?

Renal			
ATN	Prolonged pre-renal state Sepsis Toxic ATN: drugs (aminoglycosides, cisplatin, tenofovir, methotrexate, iodinated contrast) Other (rhabdomyolysis, snake bite, *Amanita* mushrooms)	Vital signs Fluid assessment Limbs for compartment syndrome	Urine Na > 40 mmol/L Fractional excretion Na ≥ 1% Dense granular ('muddy brown') casts Creatine kinase
Glomerular	Rash, weight loss, arthralgia Chest symptoms (pulmonary renal syndromes) IV drug use	Hypertension Oedema Purpuric rash, uveitis, arthritis	Proteinuria, haematuria Red cell casts, dysmorphic red cells ANCA, anti-GBM, ANA, C3 and C4 Viral hepatitis screen, HIV Renal biopsy
Tubulo-interstitial	Interstitial nephritis: drugs (PPIs, penicillins, NSAIDs) Sarcoidosis Tubular obstruction: 1. Myeloma (cast nephropathy) 2. Tubular crystal nephropathy: Drugs (aciclovir, indinavir, triamterene, methotrexate) Oxalate (fat malabsorption, ethylene glycol) Urate (tumour lysis)	Fever Rash	Leucocyturia Eosinophiluria (and a peripheral eosinophilia) White cell casts Minimal proteinuria Paraprotein Calcium (myeloma, sarcoidosis) Urine microscopy for crystals Serum urate Urine collection for oxalate
Vascular (including renal infarction, renal vein thrombosis, cholesterol emboli, malignant hypertension)	Flank pain, trauma Anticoagulation Recent angiography (cholesterol emboli) Nephrotic syndrome (renal vein thrombosis) Systemic sclerosis (renal crisis) Diarrhoea (HUS)	BP (malignant hypertension) Fundoscopy Livedo reticularis (cholesterol emboli) Sclerodactyly	Normal urinalysis or some haematuria C3 and C4 (cholesterol emboli, TMA) Doppler renal ultrasound CT angiography Platelets, haemolytic screen, LDH Consider ADAMTS13 and complement genetics (if TMA)

5.If we assume that this case is a pre-renal, what makes you suspect that by history and examination well?

15.25 Categorising acute kidney injury based on history, examination and investigations

Type of AKI	History	Examination	Investigations
Pre-renal	Volume depletion (vomiting, diarrhoea, burns, haemorrhage) Drugs (diuretics, ACE inhibitors, ARBs, NSAIDs, calcineurin inhibitors, iodinated contrast) Liver disease Cardiac failure	Low BP (including postural drop) Tachycardia Weight decrease Dry mucous membranes and increased skin turgor JVP not visible even when lying down	Urine Na <20 mmol/L Fractional excretion Na <1% High urea:creatinine ratio Urinalysis bland

6. If we assume that this case is a post- renal, what makes you suspect that by history and examination well?

Post-renal	Prostate cancer history Neurogenic bladder Cervical carcinoma Retroperitoneal fibrosis Bladder outlet symptoms	Rectal examination (prostate and anal tone) Distended bladder Pelvic mass	Urinalysis frequently normal (may reveal haematuria depending on cause) Renal ultrasound (hydronephrosis) Isotope renogram (delayed excretion) if ultrasound inconclusive

(ACE = angiotensin-converting enzyme; ANA = antinuclear antibody; ANCA = antineutrophil cytoplasmic antibody; ARBs = angiotensin receptor blockers; BP = blood pressure; GBM = glomerular basement membrane; HIV = human immunodeficiency virus; HUS = haemolytic uraemic syndrome; JVP = jugular venous pulse; LDH = lactate dehydrogenase; Na = sodium; NSAIDs = non-steroidal anti-inflammatory drugs; PPIs = proton pump inhibitors; TMA = thrombotic microangiopathy)

6. What the management of AKI?

15.26 Management of acute kidney injury

- Assess fluid status as this will determine fluid prescription:
  - If hypovolaemic: optimise systemic haemodynamic status with fluid challenge and inotropic drugs if necessary
  - Once euvolaemic, match fluid intake to urine output plus an additional 500 mL to cover insensible losses
  - If fluid-overloaded, prescribe diuretics (loop diuretics at high dose will often be required); if the response is unsatisfactory, dialysis may be required
- Administer calcium resonium to stabilise myocardium and glucose and insulin to correct hyperkalaemia if $K^+ > 6.5$ mmol/L (see Box 14.17, p. 363) as a holding measure until a definitive method of removing potassium is achieved (dialysis or restoration of renal function)
- Consider administering sodium bicarbonate (100 mmol) to correct acidosis if H^+ is >100 nmol/L (pH <7.0)
- Discontinue potentially nephrotoxic drugs and reduce doses of therapeutic drugs according to level of renal function
- Ensure adequate nutritional support
- Consider proton pump inhibitors to reduce the risk of upper gastrointestinal bleeding
- Screen for intercurrent infections and treat promptly if present
- In case of urinary tract obstruction, drain lower or upper urinary tract as necessary

Infectious diseases

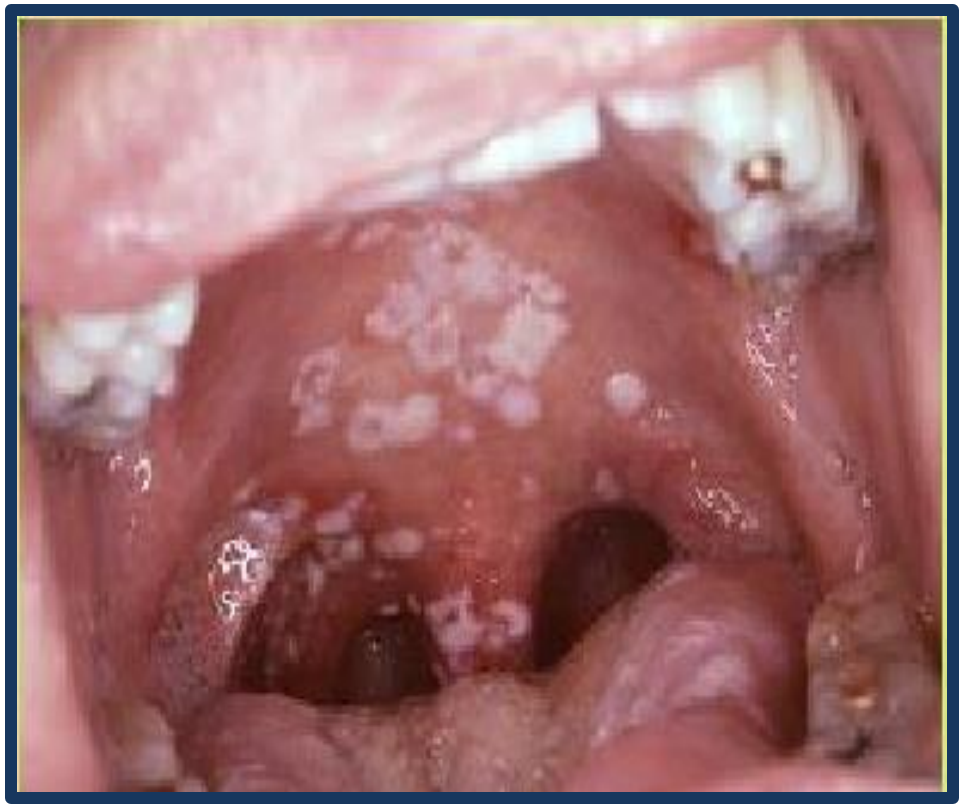
Done by :

ماجدة يوسف

رنا الشرع

Q: This patient is receiving inhaled steroids, what's your diagnosis?

Oral Candidiasis



Q: Who are the patients mostly affected by this ?

Immunocompromizes

Patients that have uncontrolled DM

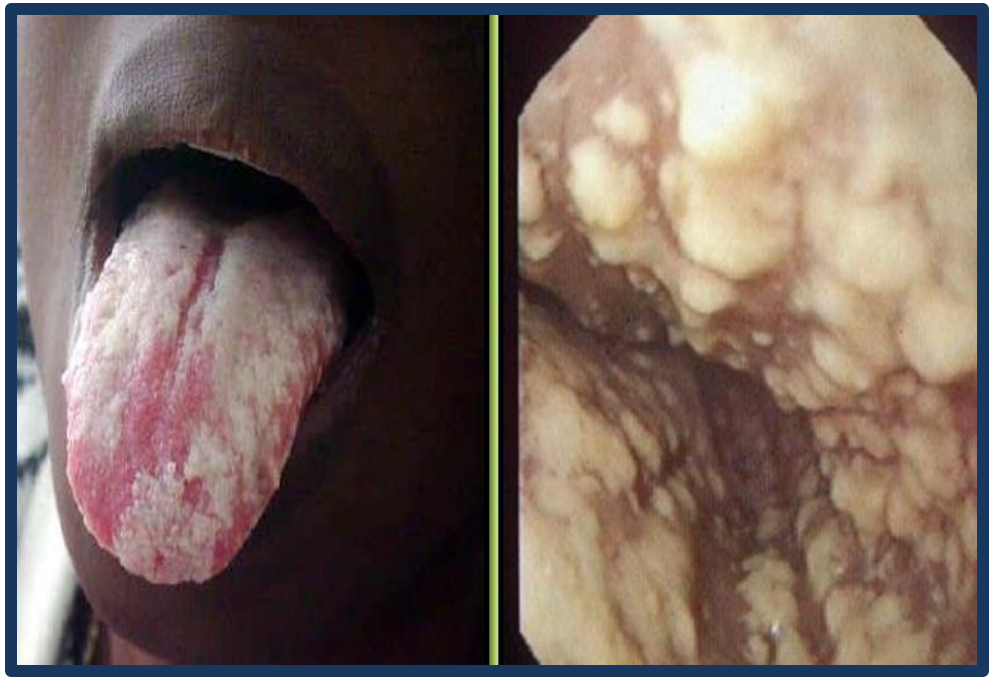
Patients have HIV infection

How to treat such case ?

Echinocandin is the first line therapy in all patients

Q: 34 YO pt with HIV presented with these lesions, what is your Dx?

Candidiasis.



Q: This patient had unilateral lowerlimb swelling &redness. What's the investigation that you'll do to diagnose this case?

Venous Doppler Ultrasound

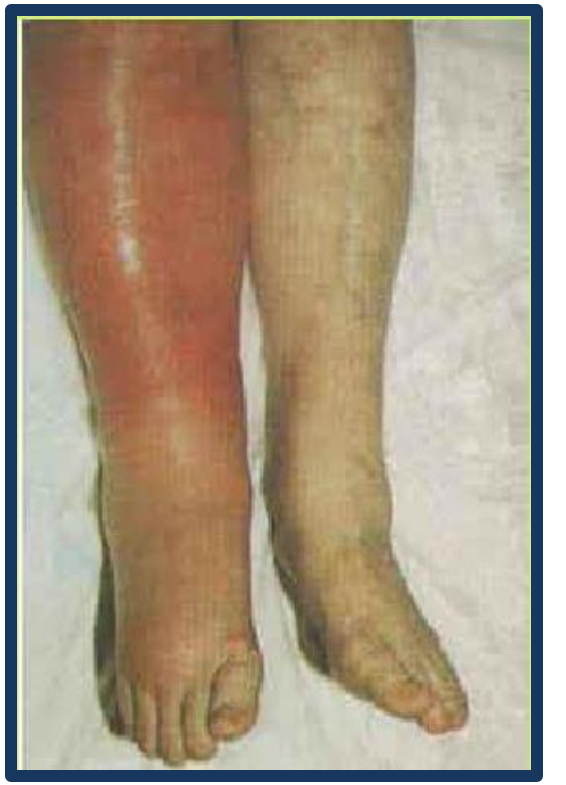
What are the posible causes causes for this ?

Dvt

Celluitis

Comatment syndrome

Lymphadema



Q: This pt was presented with swollen, red, warm & painful right leg. WBCs = 17.000, what is your spot Dx.?

Cellulitis.

Mention another differential

DVT

How to differentiate between them ?

D dimer and Doppler u/s

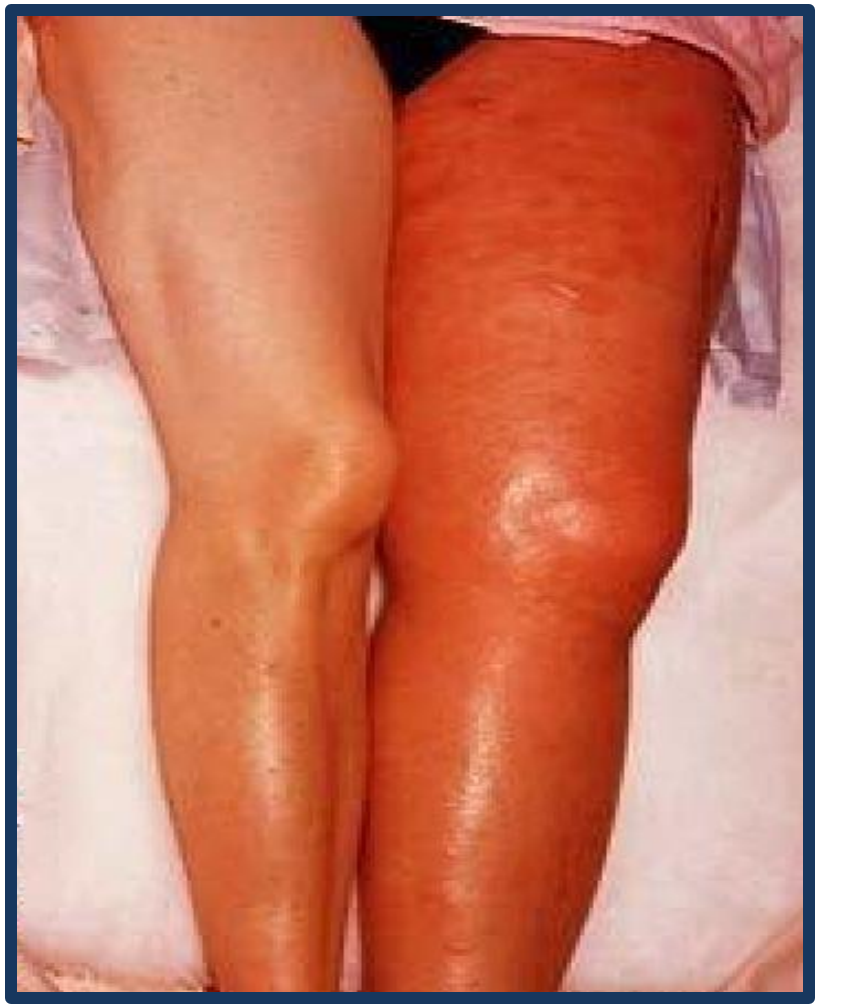
How to treat based on your spot DDX ?

Treat with staphylococcal penicillin or cephalosporin iv till symptoms improve follow up with oral antibiotics till symptoms improve

Q: Pt with DM & HTN, give 2 DDx?

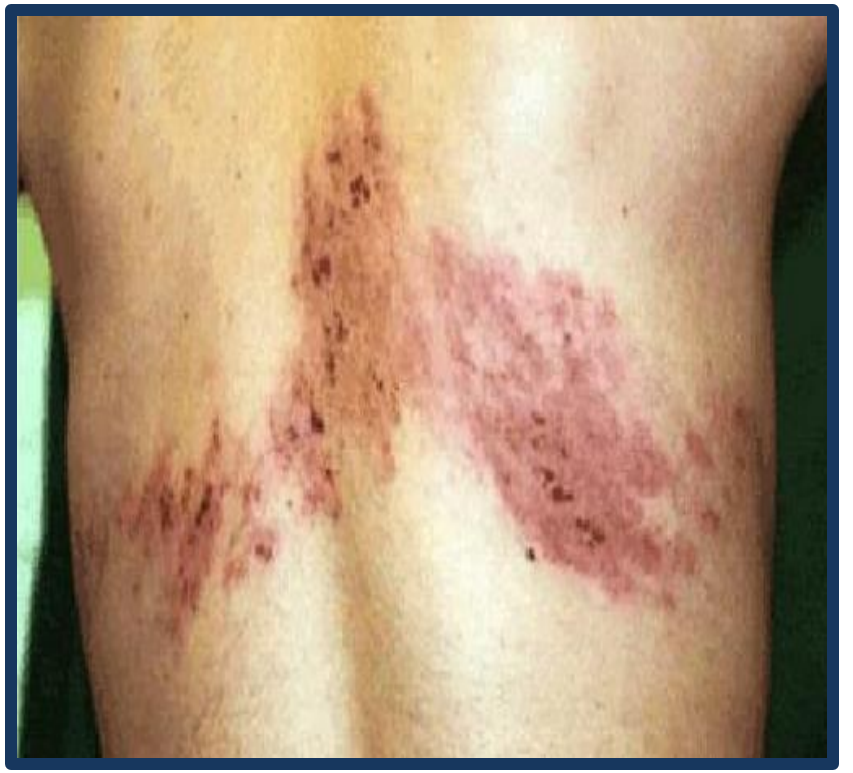
A. DVT .

B. Cellulites.



Q: what is the diagnosis?

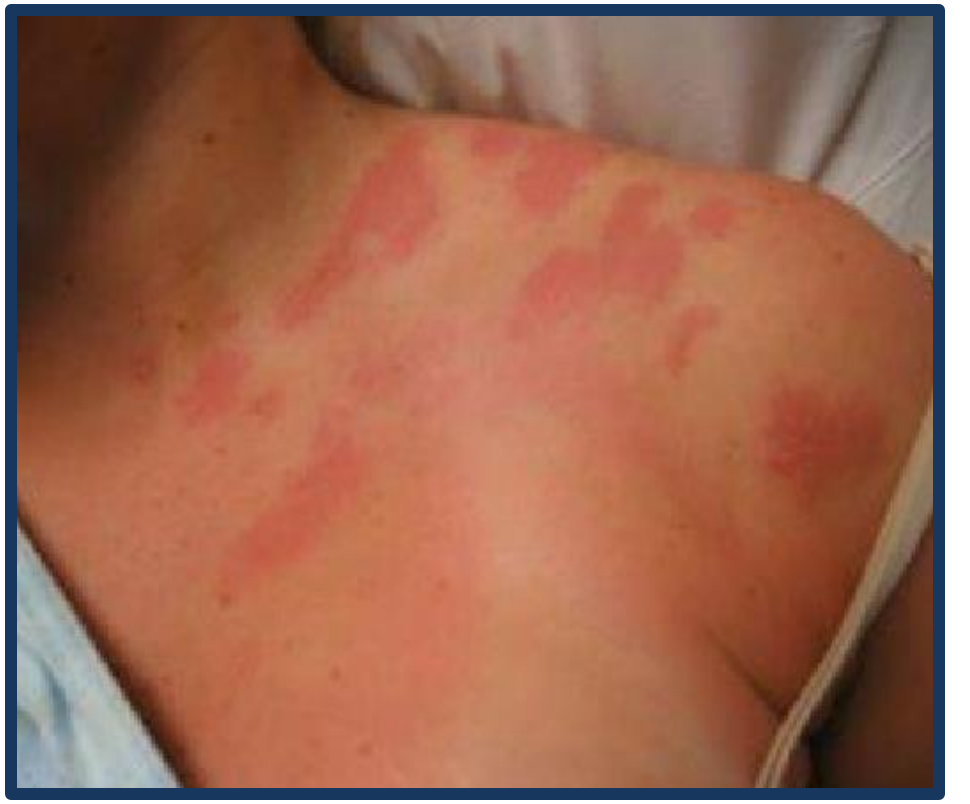
Herpes zoster.



Q: a pt with skin lesions on a Dermatological distribution.

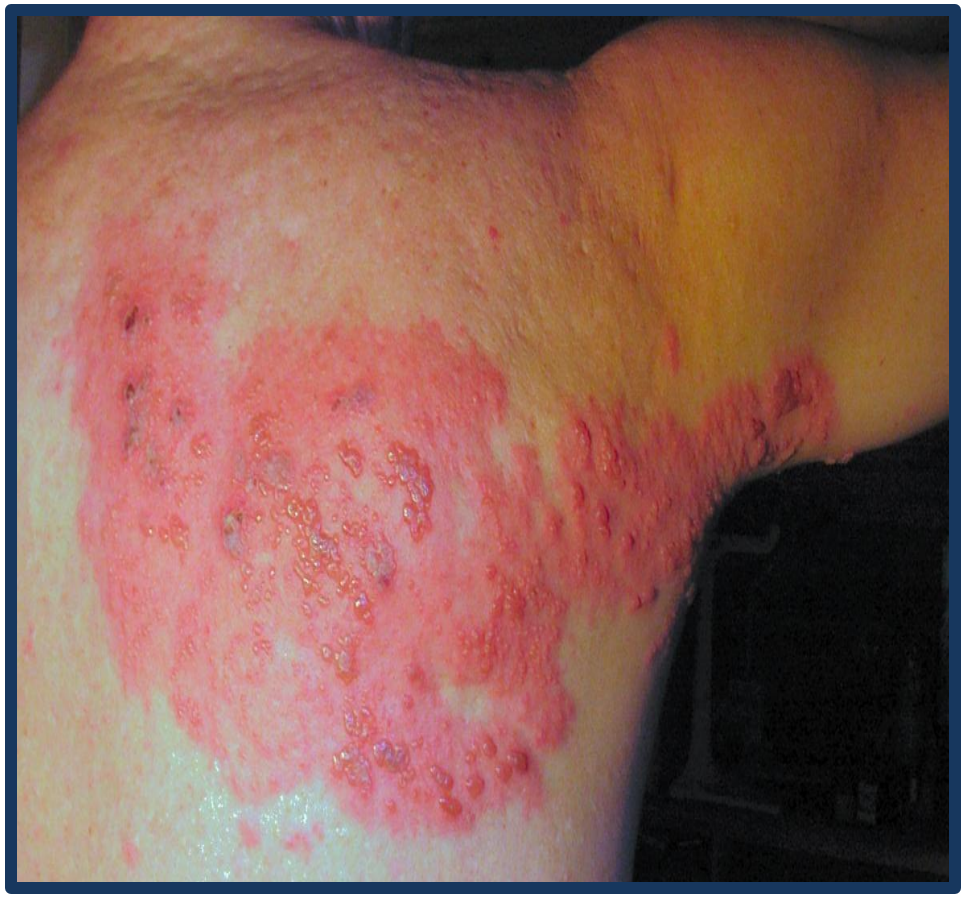
What is your Dx?

Herpes zoster.



Q: What is your spot diagnosis ?

Herpes zoster



Q: 24 YO female, presented with headache, fever, & deterioration in level of consciousness, brain CT was free, the L.P s (values shows high WBS, LOW glucose).

Q1: what is the Dx?

Acute meningitis.

Q2: give 2 lines of treatment.

IV antibiotics , Anti-pyretics .

Q3: give one major complication.

brain abscess, seizure, encephalitis .

Q :a known case of crohns disease came with this oral lesion

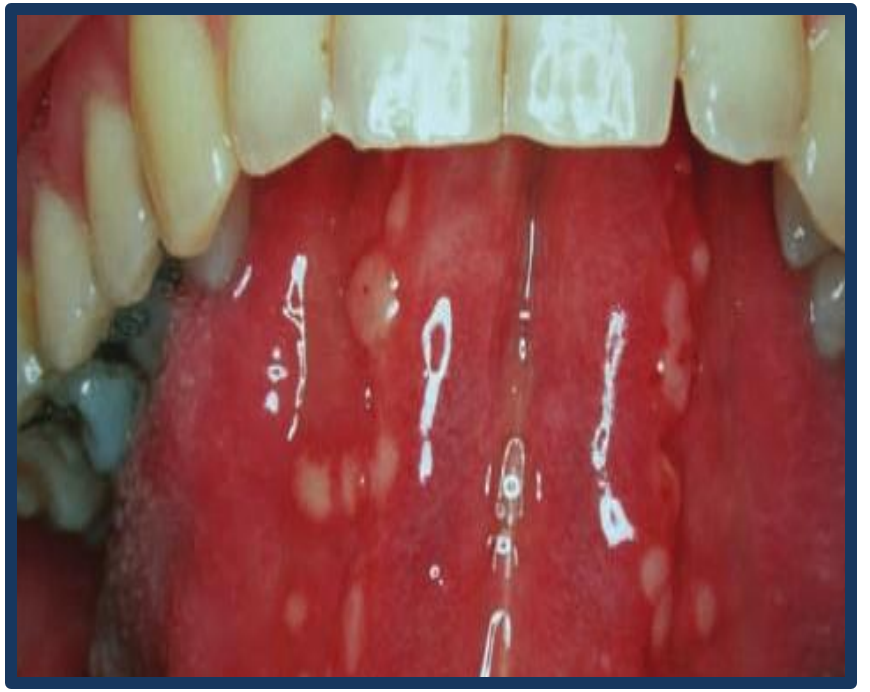
a. identify this lesion ?

apthus ulcers

Note* some said it was candida infecon (Pic was not that clear)

B. do you think the anus will be affected ?

Yes anus can be affected



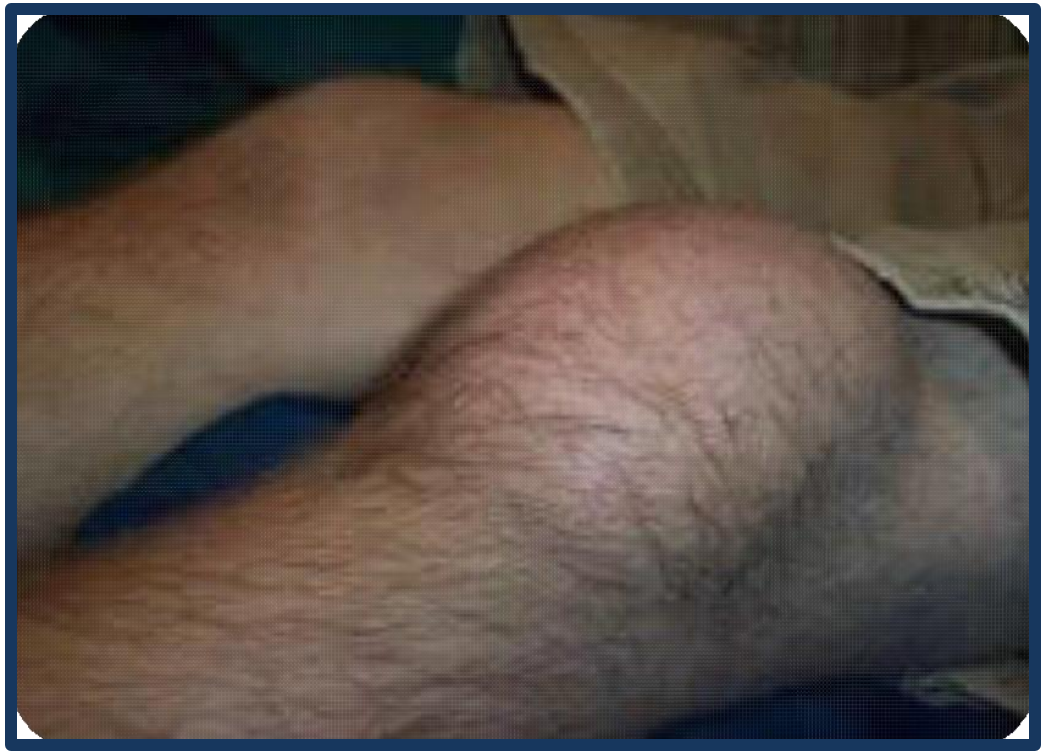
Q: A man comes to the ER after3 hours of severe pain in his knee, on examination his left knee is swollen, warm, & very tender to palpation .

What is the Most likely diagnosis?

Septic Arthritis

What is the investigation of choice?

Synovial Fluid Analysis



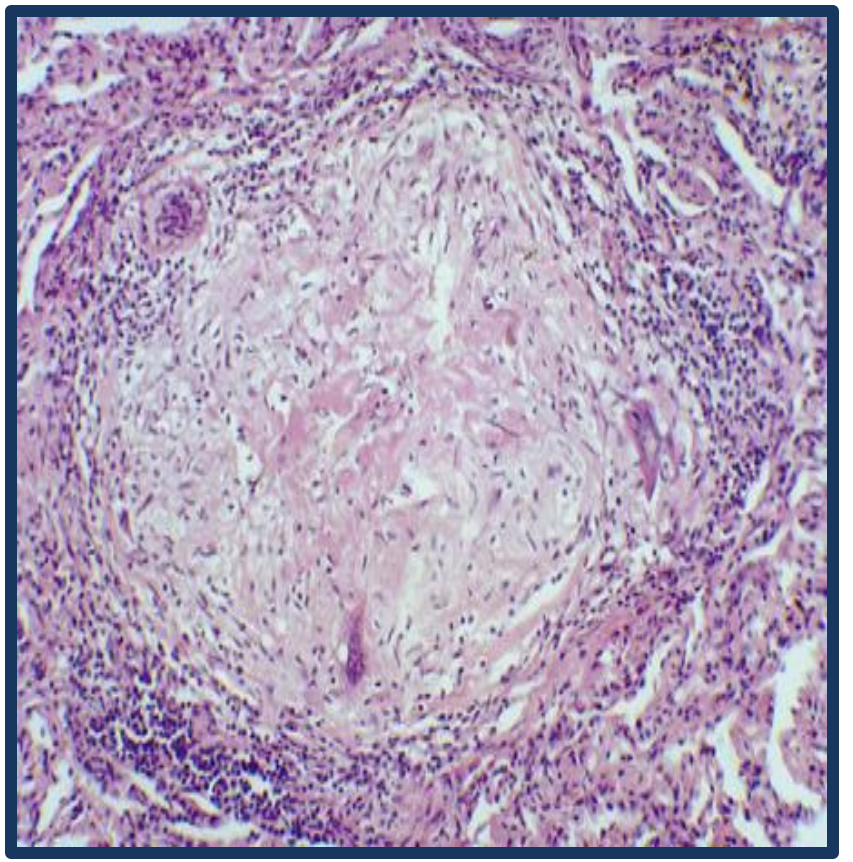
Q: A rheumatoid arthritis patient on adalimumab presented with weight loss and lymph node enlargement, biopsy is shown.

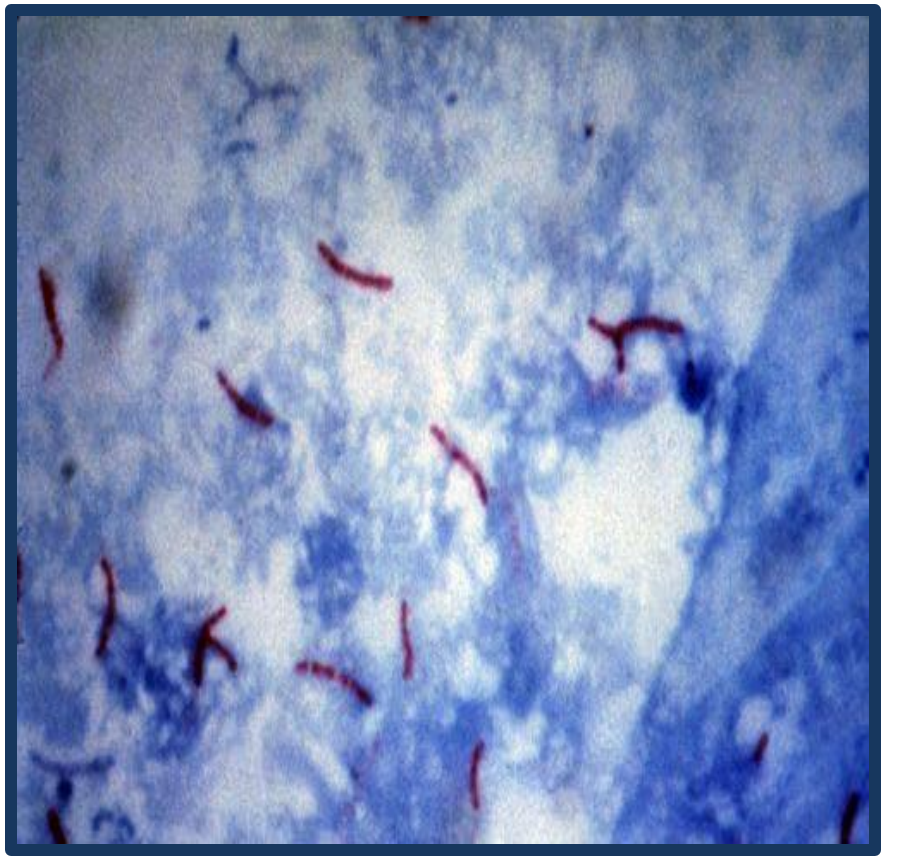
1- What is the diagnosis?

Caseating granuloma and acid fast bacilli, so: Tuberculosis

2- 2 drugs to manage

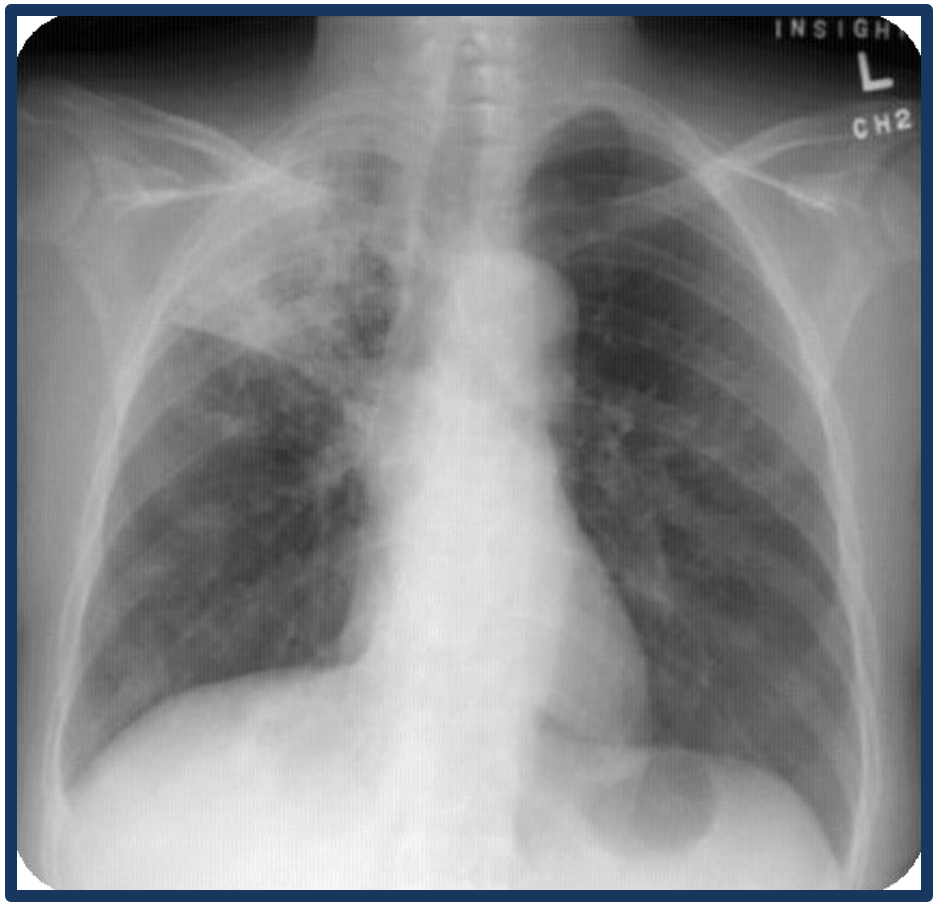
Pyrazinamide – Ethambutol – Rifampin - Isoniazid





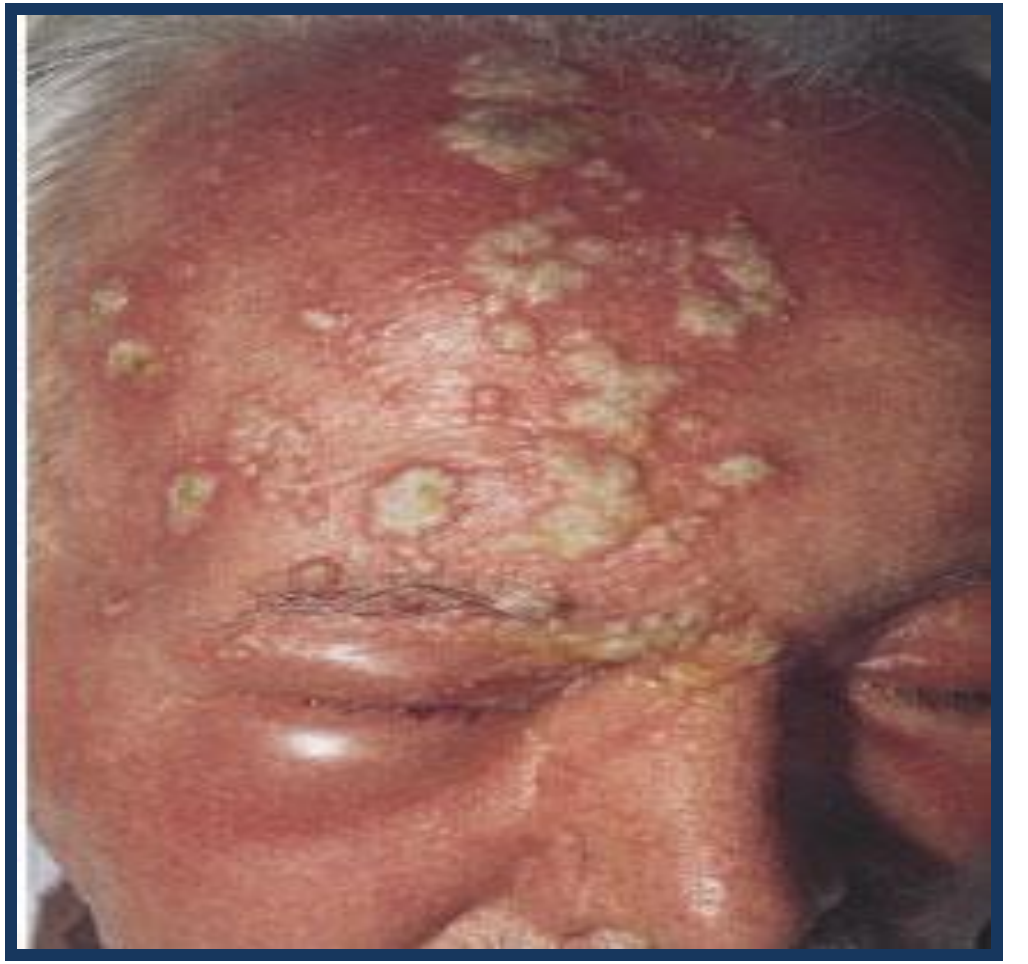
Q: This Alcoholic pt presented with productive cough, hemoptysis, fever, night sweats, & weight loss. What is your diagnosis?

Active Tuberculosis



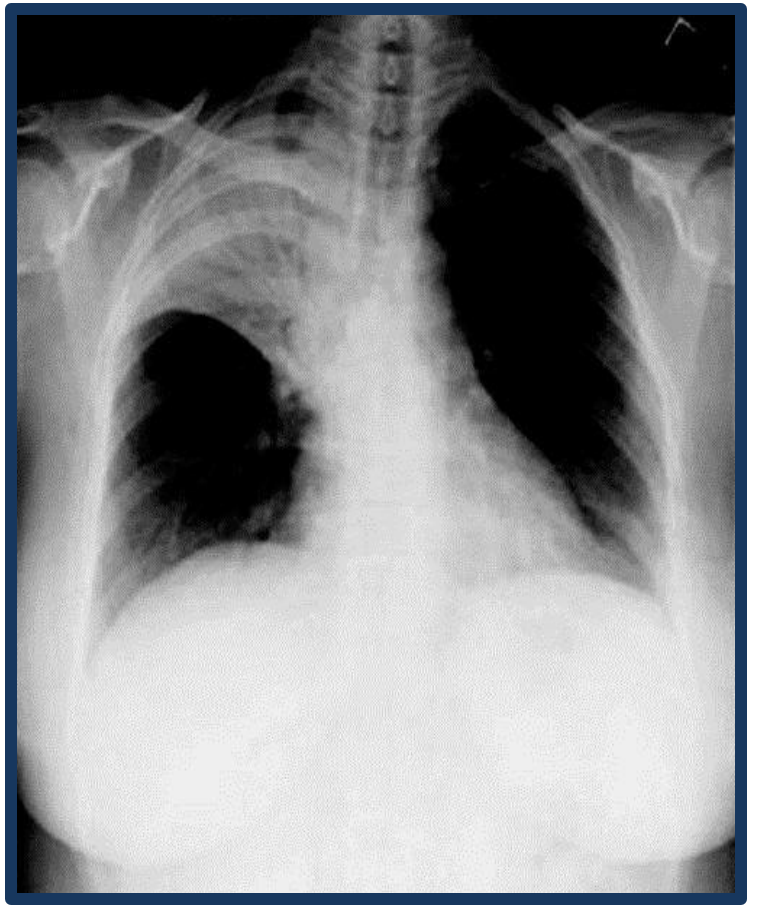
Q: A 55 Year old man with Hx of Lymphoma What is your diagnosis ?

Zoster ophthalmicus



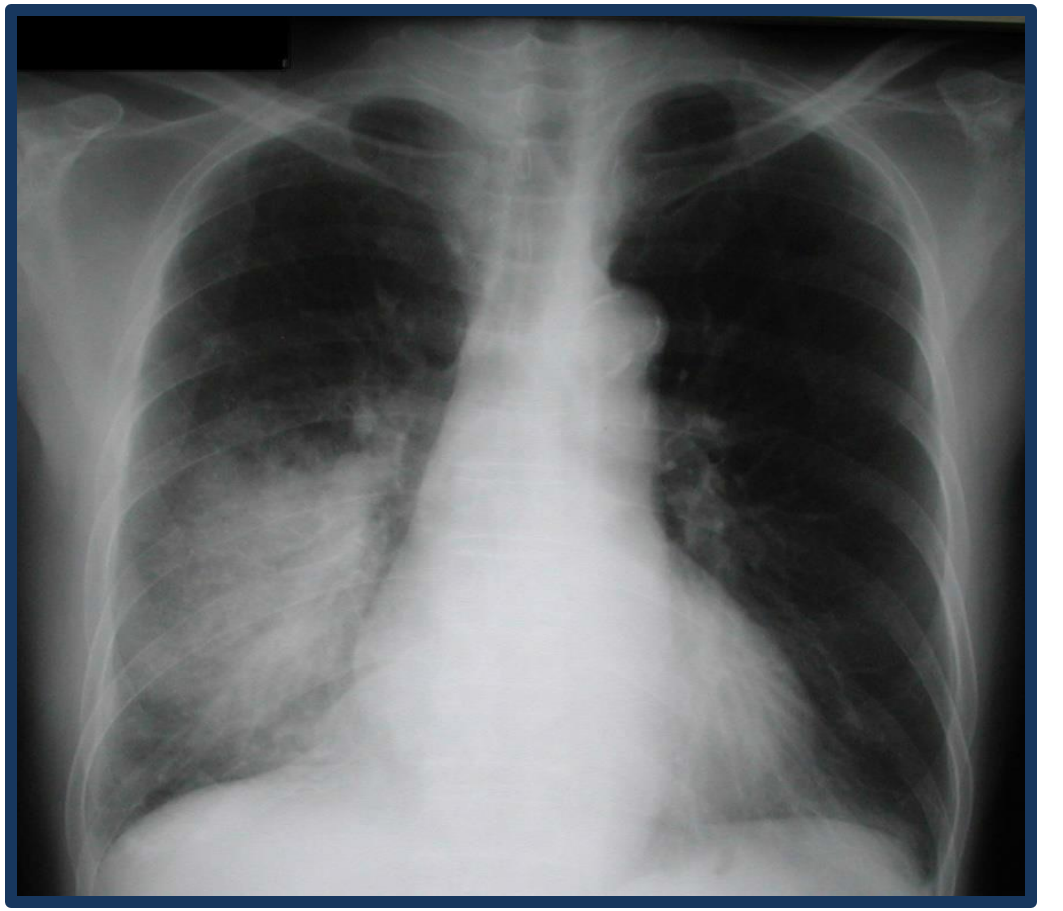
Q: Diabetic patient with productive cough of 3 days duration associated with fever & chills. What is the diagnosis?

RUL pneumonia



Q: 35 YO male pt, previously healthy presented complaining of cough of greenish sputum & fever, What's the most likely micro-organism?

Strep. Pneumonia

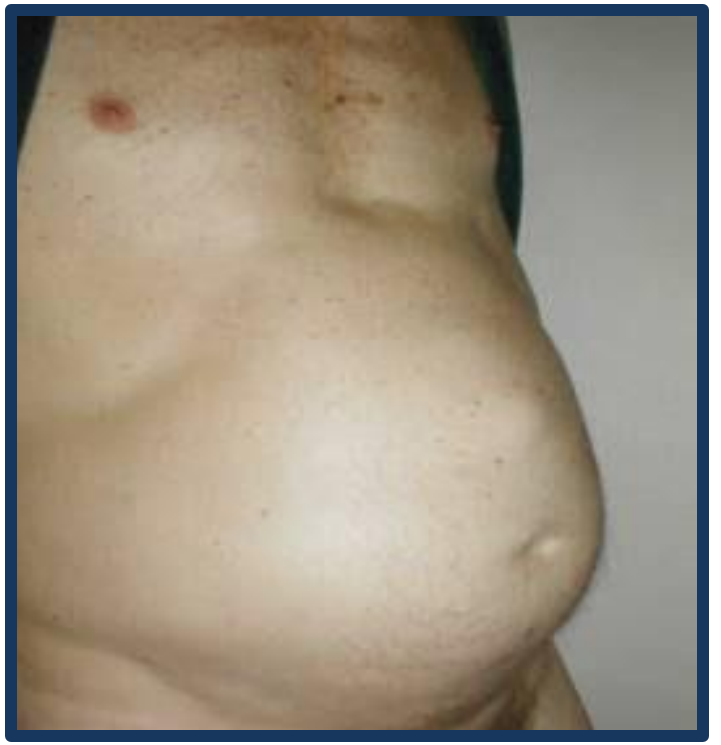


Q: Pt with liver cirrhosis & ascites , presented with fever & abdominal pain , P/E shows rigid abdomen, what is the most likely Dx?

Spontaneous bacterial peritonitis .

How to confirm?

peritoneal fluid analysis & cultur.



Q: A man is suffering from haematuria after 2 days of having Streptococcal infection in his throat.

What's your Dx?

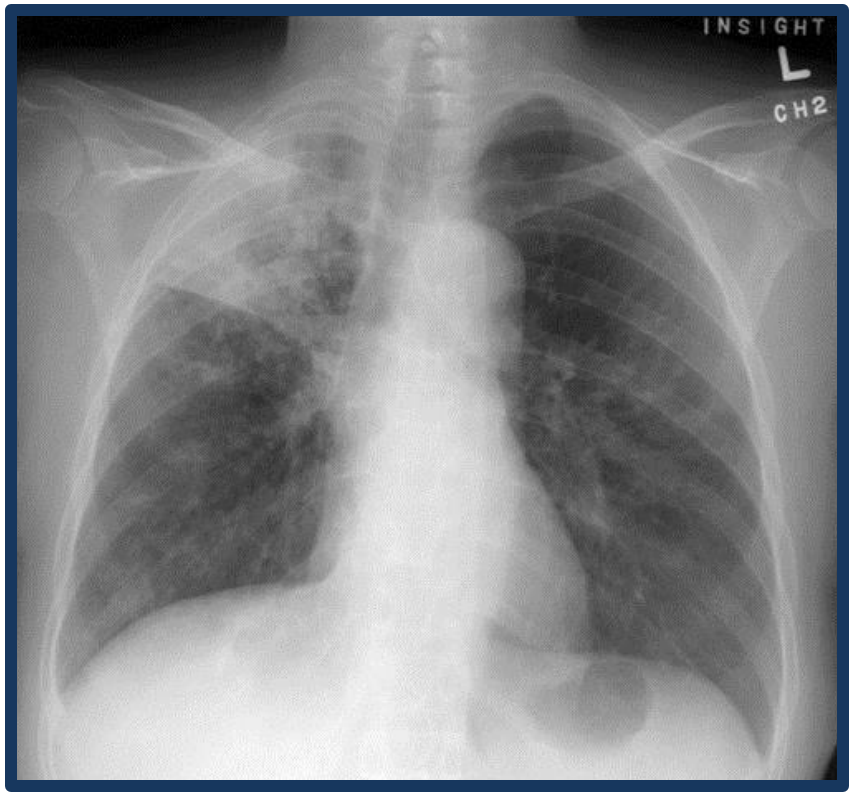
IgA glomerulonephritis

Q: This pt presented with productive cough, associated with hemoptysis & intermittent fever, resistant to levofloxacillin. what are CXR findings?

Rt upper lobe consolidation (TB)

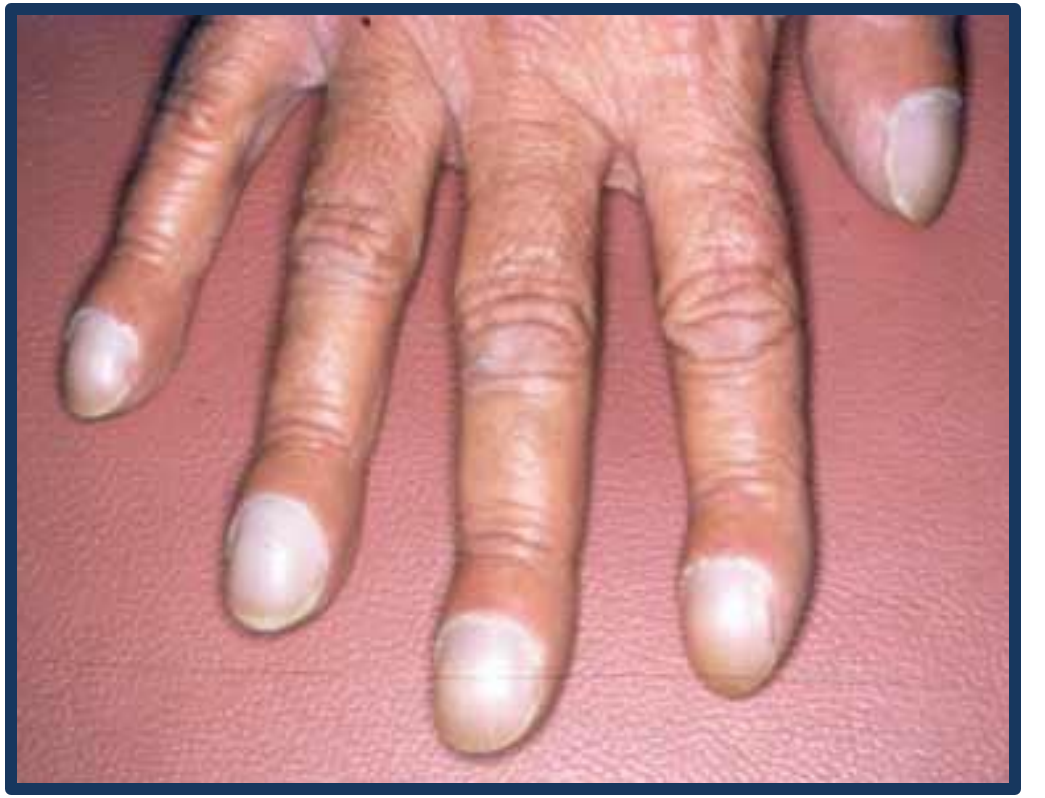
Investigations?

PPD, Sputum analysis , Bronchoscopy.



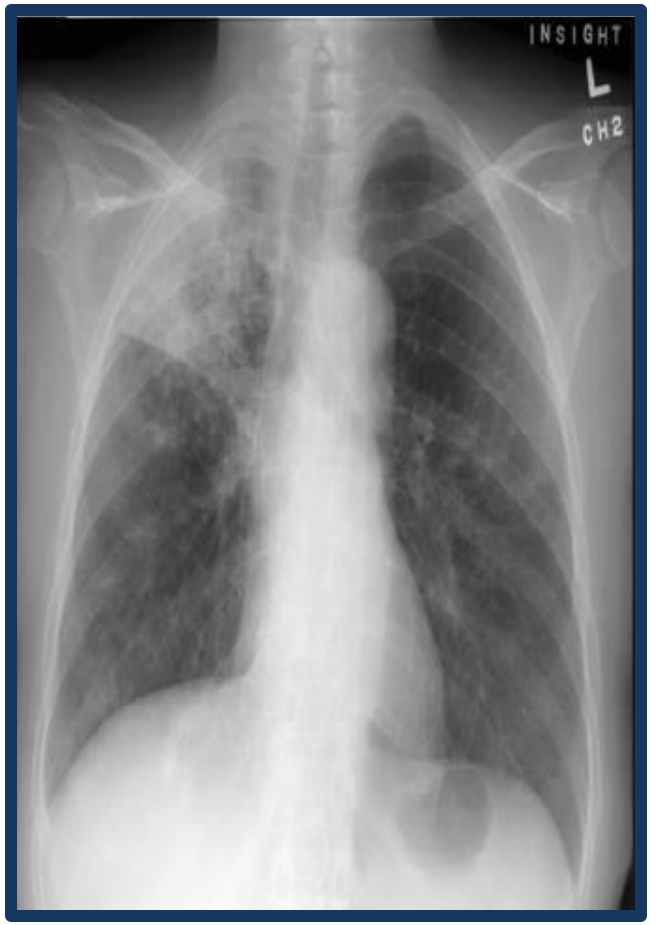
Q: A 40 YO man is brought, to the hospital because of fever. He has Hx of heamturia. On exam, there is a systolic murmur, at the lower left sternal border. What is the Dx?

Infective endocarditis



Q: This CXR is for a 30 YO farmer complaining of fever & night sweats 2 weeks prior to admission. What is your Dx?

Tuberculosis

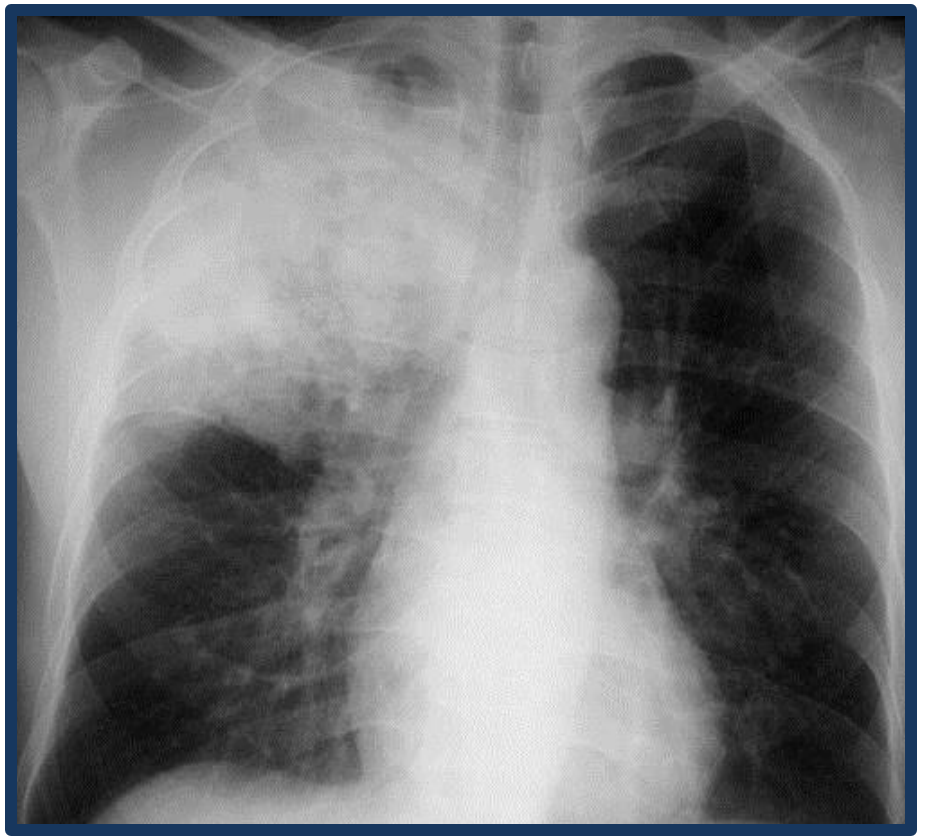


Q: This pt presented with cough for 8 weeks, fever, Hemoptysis, wt loss, night sweats & anorexia. What is the finding in this CXR?

Right upper lobe consolidation.

What is your Dx.?

Tubercolosis.



Q: A previously healthy 36 YO male applied for a job in KSA, his application was refused because of abnormal liver function test. He drinks Alcohol occasionally, he was asymptomatic. his AST and ALT were mildly elevated. (numbers were mentioned in all the following tests, so you should know the normal ranges), his ALP was in normal range, +ve for Hbs IgG, -ve for Hbc antigen & Hbs antigen, –ve for other hepatophilic viruses. There was increase in LDL, Triacylglicerides, and a high BMI. Tests for metabolic and inherited liver diseases were normal.

- Mention 3 DDx?

chronic hepatitis B infection, steatohepatits, Autoimmune diseases

- Mention 2 tests to confirm your diagnosis ?

(definite Dx) >> Ds-DNA of hepatitis B, Liver biopsy .

- Mention 5 health problems associated with his BMI

DM, HF, HTN, OSA, Atherosclerosis.

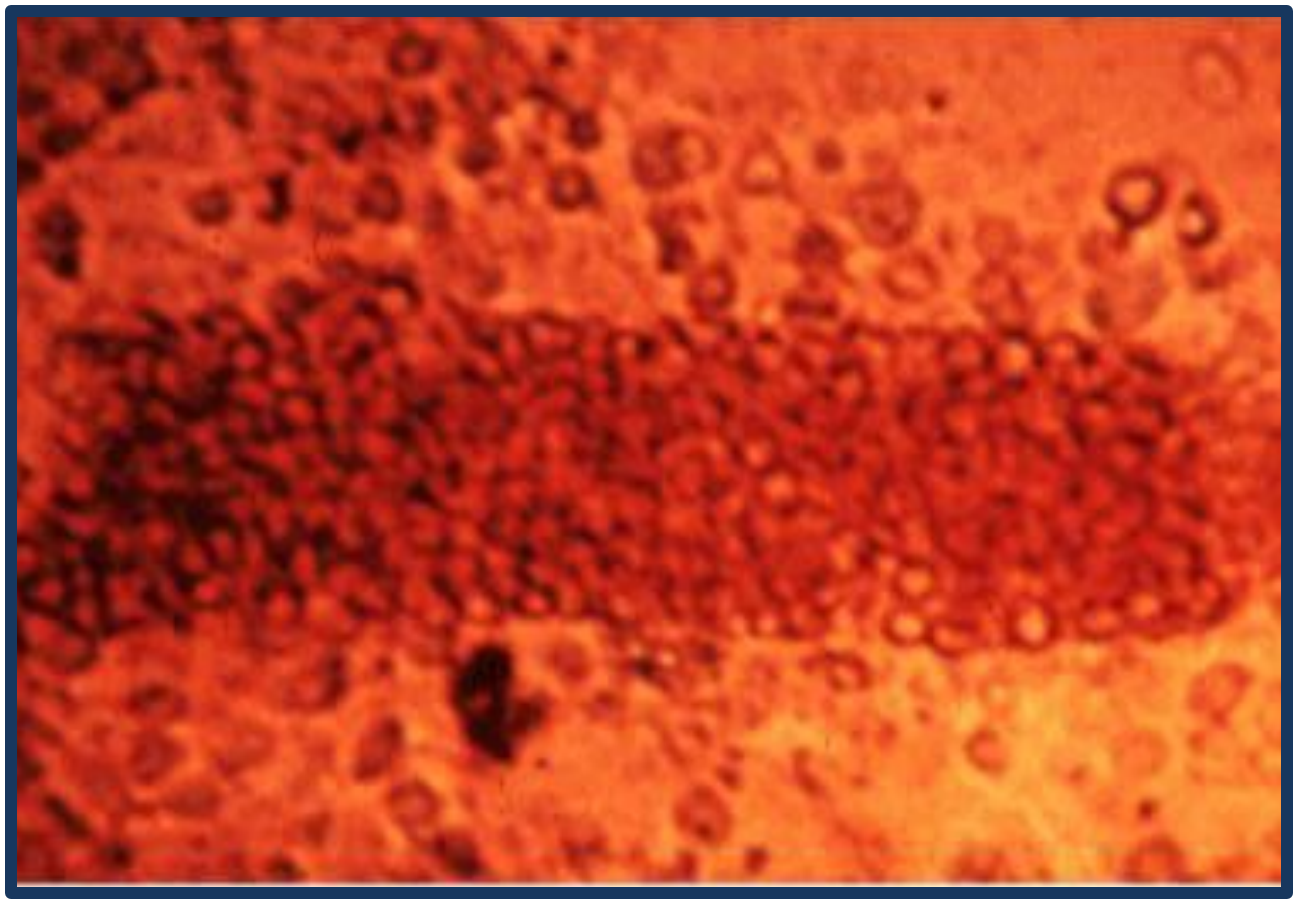
Q: A pt presented with red urine. The picture shows a microscopical view of his urine sample. Mention 2 causes for this condition.

This is an RBC cast seen in nephritic syndrome.

Causes are :

1- IgA Nephropathy. 2- SLE. 3- Cryoglobulinemia.

4- Post-Strep infection



Q: A pt with hypertension (or DM) presented with right ankle swelling & pain. He had 2 previous similar conditions; one was in the same site, the other was on the left ankle. His CBC showed leukocytosis (WBC count = 10,000).

1- What is the most probable Dx?

Gout

2- Mention another DDx.

Septic arthritis, Cellulitis, Pseudogout.

3-If a sample from the synovial fluid was aspirated, what is your confirmatory test?

Identification of monosodium urate crystals under polarized light microscopy; they have a needle-like morphology & strong negative birefringence.

4-Mention 2 drugs for the treatment of the acute attack.

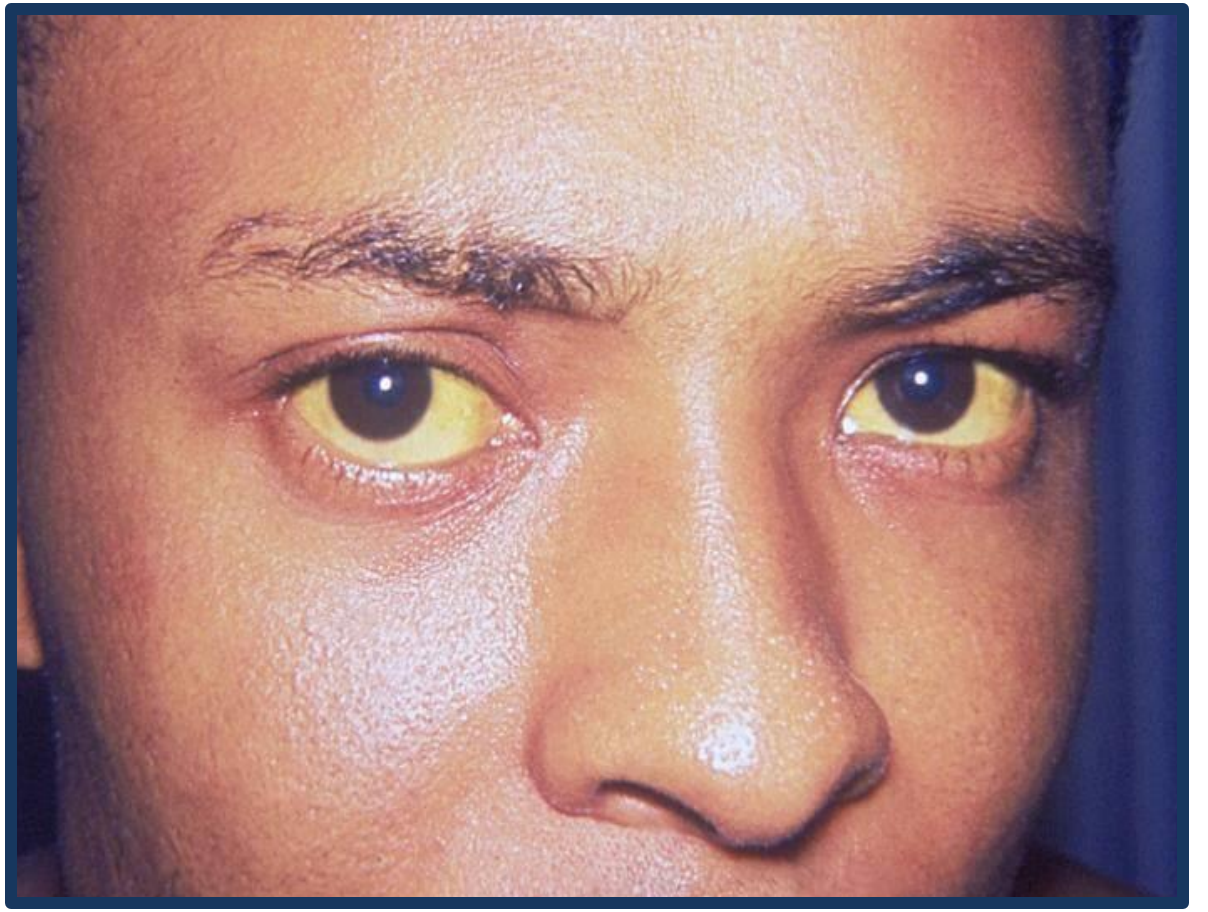
Steroids, NSAIDs, Colchicine.

Q: A pt presented with fever, abdominal pain, dark urine & nausea. Three of his classmates had similar condition. What is your Dx?

Acute Hepatitis A.

Q: Sicke cell pt ,Recurrent RUQ abd pain for a week ,Now comes with this:
2 investigations to confirm your diagnosis?

CBC
LFT
Retic

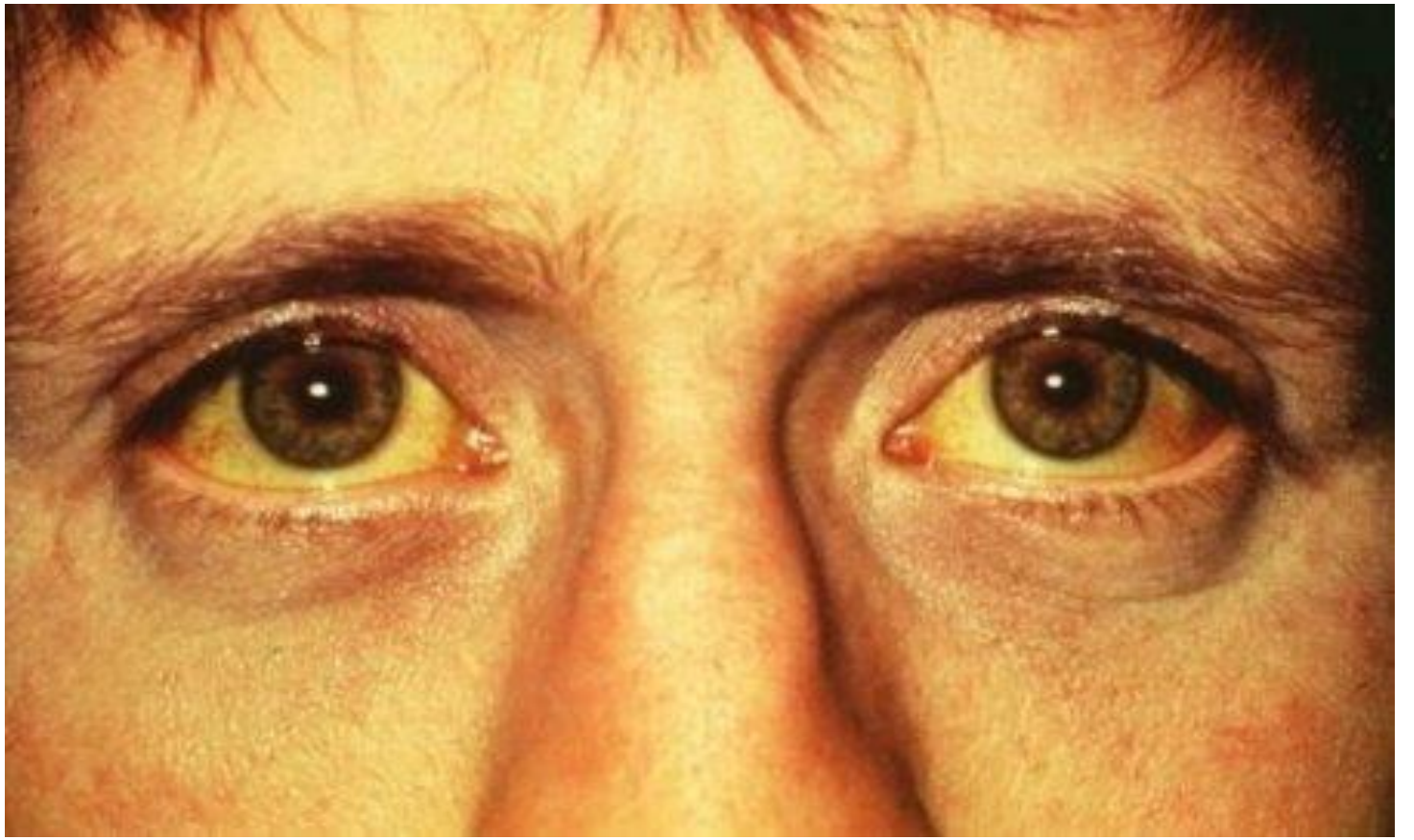


Q: This pt presented with RUQ pain, diarrhea, anorexia, & nausea. His sister has similar condition.

Acute Hepatitis A.

Q: This patient came with intermittent abdominal pain of 1 weeks duration, what is the best initial diagnostic test to order for him?

Don't know exactly! The answer could be LFT .. Ultrasound .. IgM for hepatitis A.



Q: Mention complications for this procedure

Complication of Tattoo

- Allergic reactions
- Skin infections
- Bloodborne diseases like hepatitis B and c and MRSA

Q: A pt came to ER complaining of swelling in his left knee. He has no Hx of trauma or bleeding diathesis.

•What is your most likely Dx?

Septic Arithritis

•How to diagnose ?

Joint fluid analysis and blood tests

Imaging test to assess damage To joint

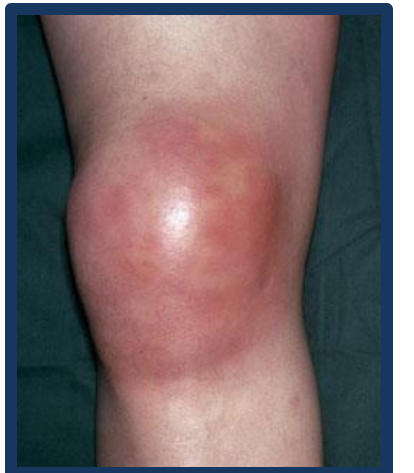
•Most common organism to cause This ?

•Bacterial infection with staphylococcus

• aurus is the most common cause

•Who are the most susceptible patients To this insult ?

Patients with damaged or prosthetic Joint



Q: How to treat such case ?

- Systemic antibiotic nafcillin or vancomycin for gram positive cocci
- For negative gram stain use vancomycin plus ceftazidim
- It may be necessary to repaetedly drain the joint
- Patients who don't improve with antibiotic and repeated aspiration should undrgo surgical lavage and or arthrotomy

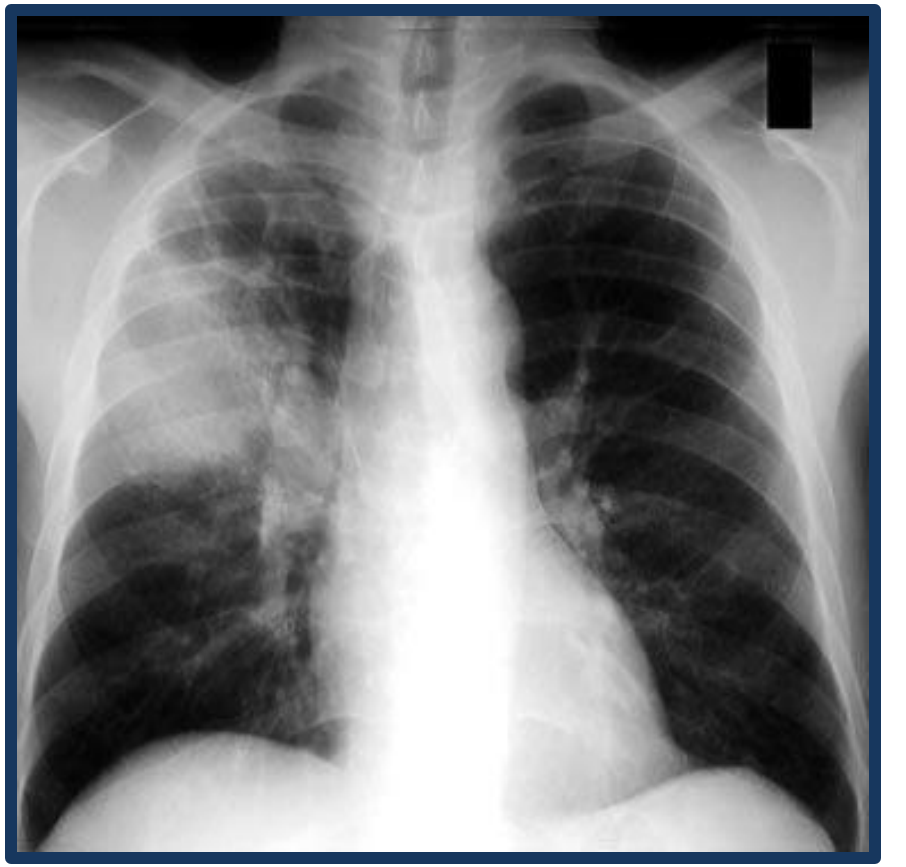
Q: Patient with fever & cough

A- what's your diagnosis?

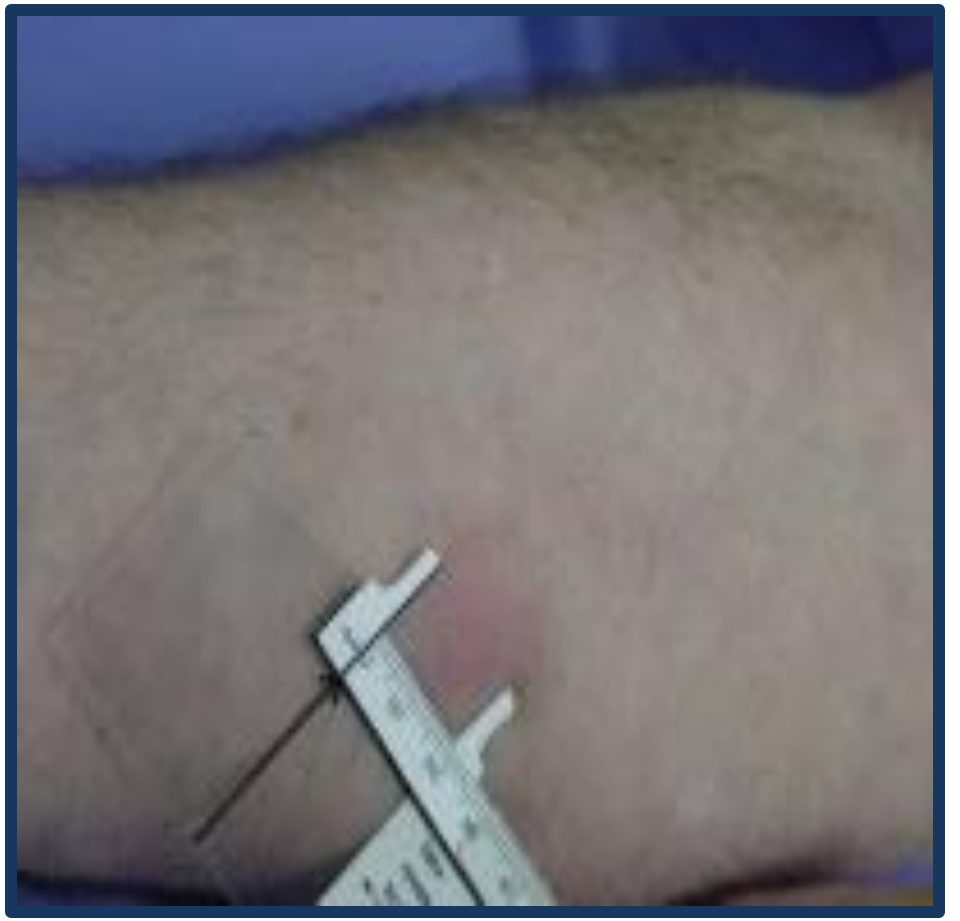
RUL pnemonia

B-What's the most common microorganism.

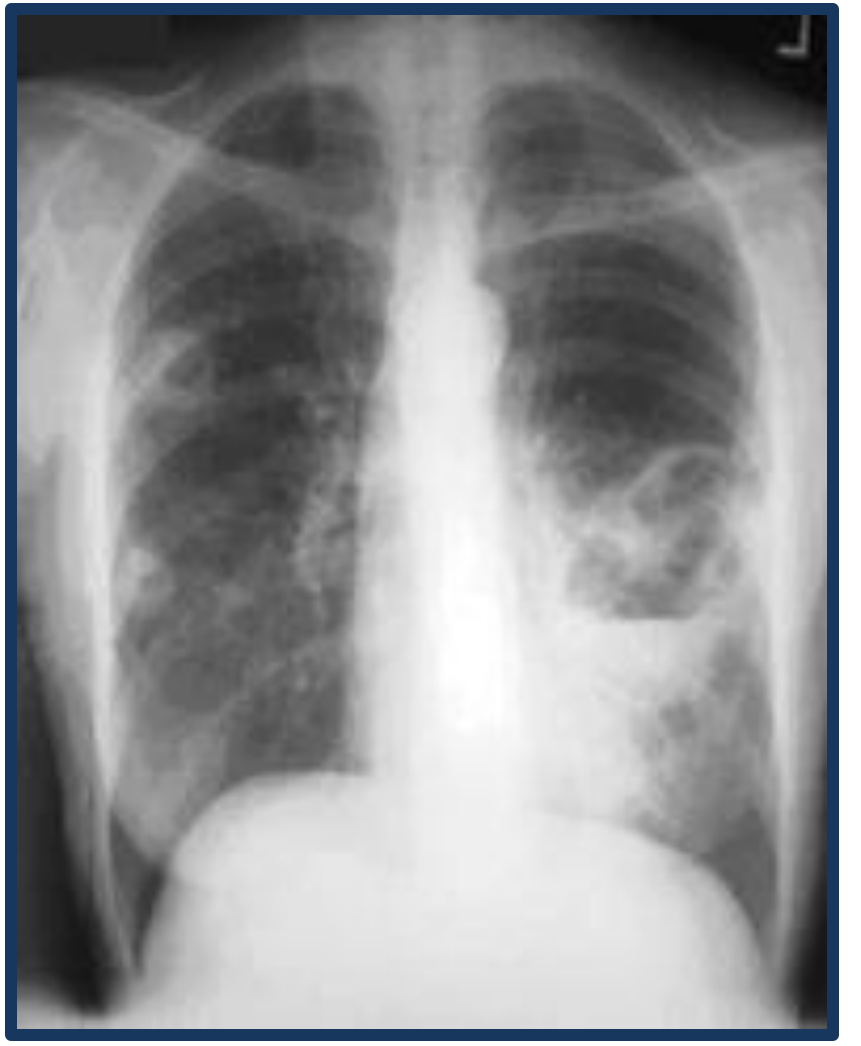
S.pneumonia



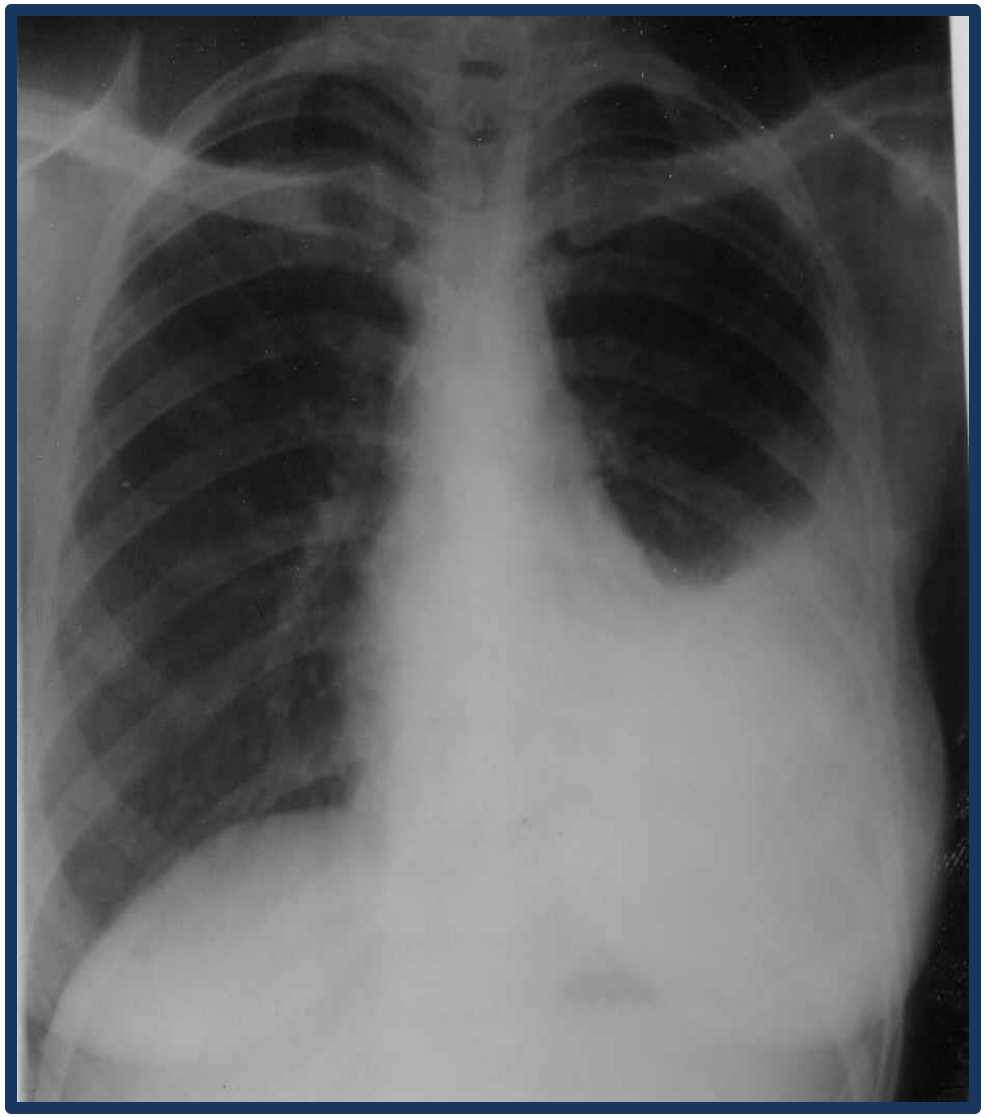
Mantoux Test



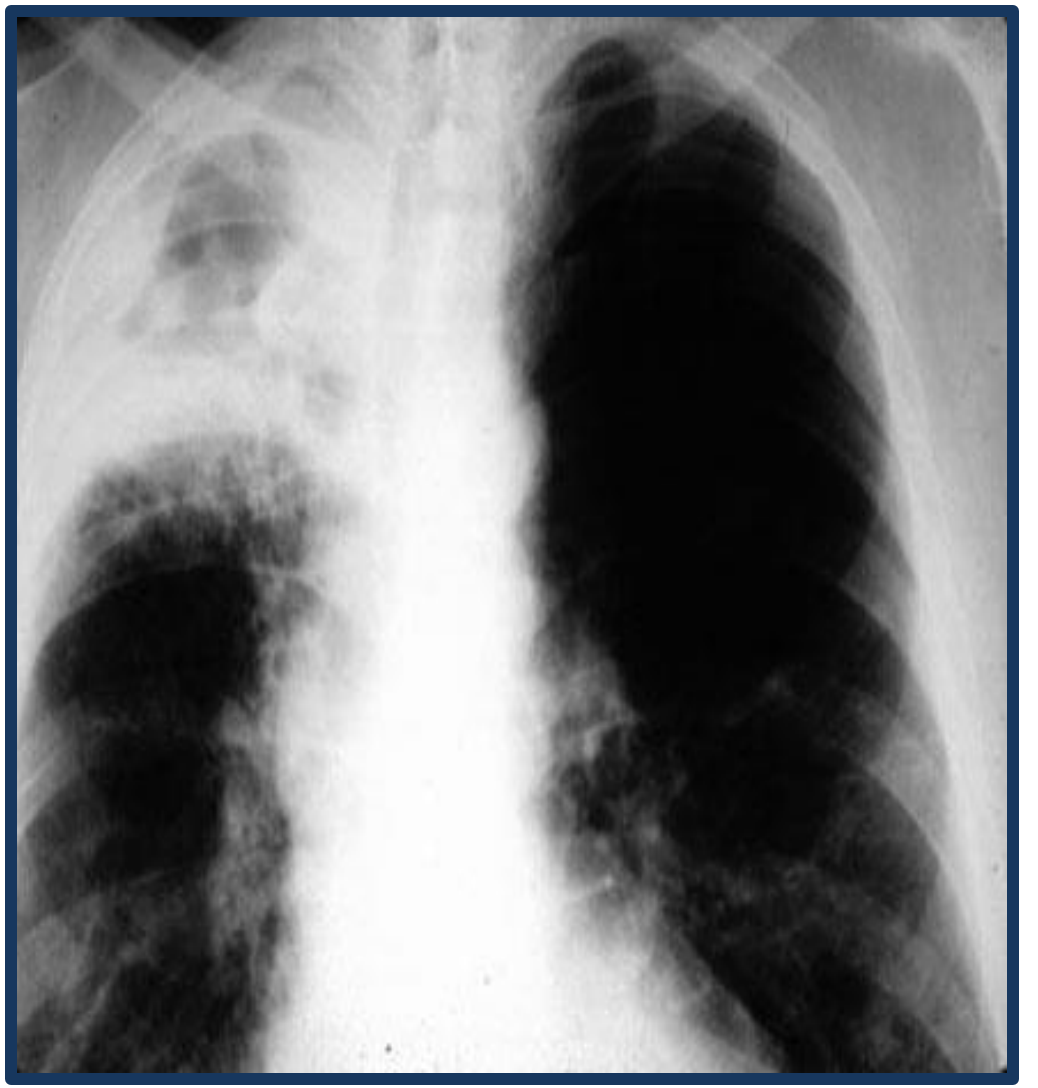
A cavitory TB (primary or reactive tb)



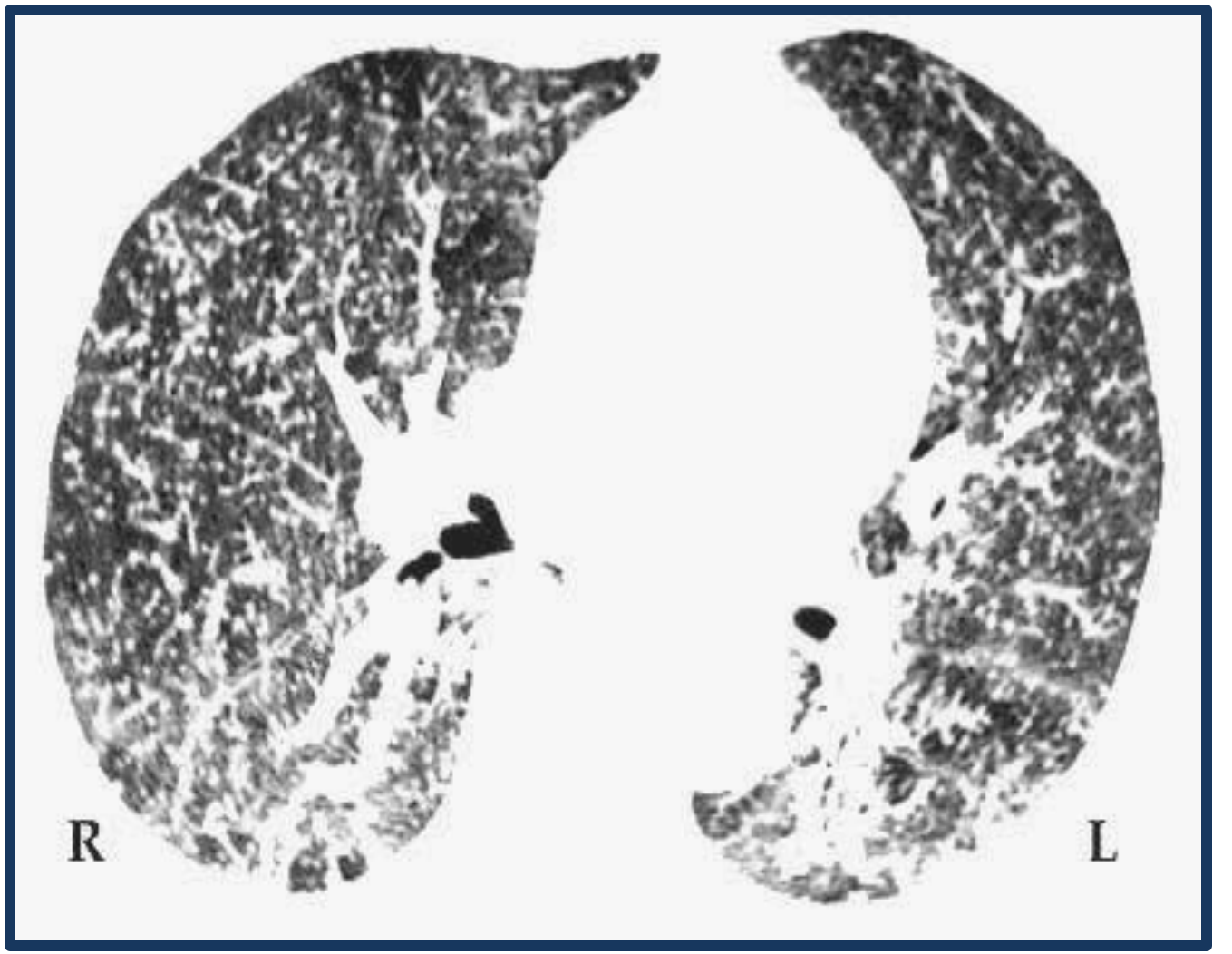
Primary or reactive tb)(Pleural Effusion



Consolidation(reactive tb)



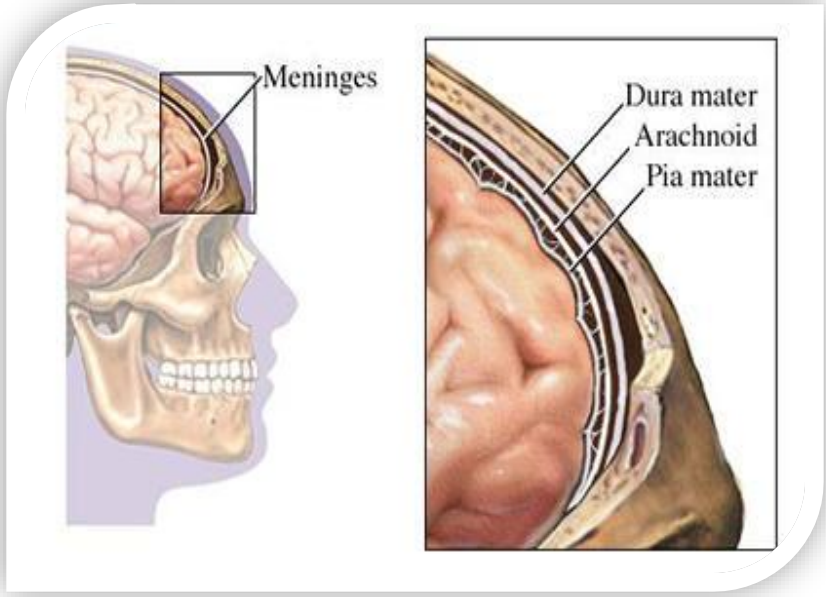
CT scan Miliary TB



Meningitis

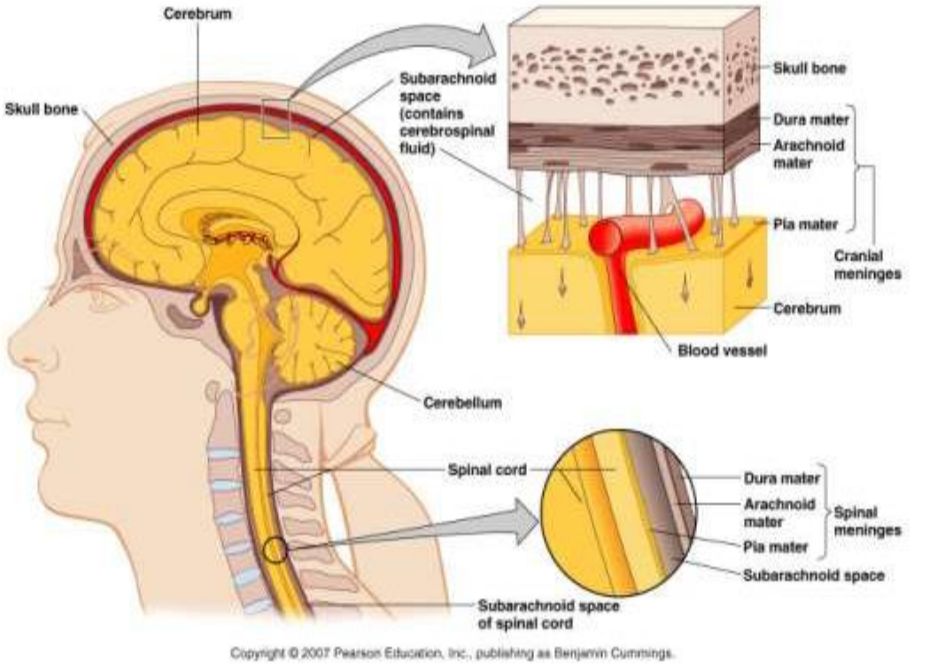
Meningitis

- Meningitis is a disease caused by the inflammation of the protective membranes covering the brain and spinal cord known as the meninges.
- The inflammation is usually caused by an infection of the fluid surrounding the brain and spinal cord.
- Meningitis can be life-threatening because of the inflammation's proximity to the brain and spinal cord; therefore the condition is classified as a medical emergency.



Meninges

- The meninges is the system of membranes which envelops the central nervous system.
- It has 3 layers:
- 1. Dura mater
- 2. Arachnoid mater
- 3. Pia mater
- Subarachnoid space - is the space which exists between the arachnoid and the pia mater, which is filled with cerebrospinal fluid.



Causes of Meningitis

- Bacterial
- - Viral
- - Fungal
- - Rickettsia
- - Parasitic/ protozoal
- - Physical injury
- - Cancer
- - Certain drugs (mainly, NSAID'S)

❖ Severity/treatment of illnesses differ depending on the cause. Thus, it is important to know the specific cause of meningitis.

Epidemiology

- Major risk factor for meningitis

lack of immunity to specific pathogens associated with young age.

- Additional risks:

recent colonization with pathogenic bacteria close contact (household, day care centres) with individuals having invasive disease caused by N. meningitides and H. influenza type b, crowding, poverty

- Mode of transmission

Probably person-to-person contact through respiratory tract secretions or droplets.

- Defects of the complement system (C5-C8) have been associated with recurrent meningococcal infection.
- Splenic dysfunction (sickle cell anemia) orasplenia (due to trauma, or congenital defect) is associated with an increased risk of pneumococcal, H. influenzae type b (to some extent), and, rarely, meningococcal sepsis and meningitis.

Pathogenesis

- Bacterial meningitis most commonly results from haematogenous dissemination of microorganisms from a distant site of infection; bacteremia usually precedes meningitis or occurs concomitantly.

- Usual source of bacteremia: bacterial colonisation of naso-pharynx with potentially pathogenic microorganism.

- Common Causes of bacterial meningitis

Newborn(0-6 MO)	Children (6MO – 6 YR)	Young Adults (6-60 yr)	Elderly 60Y+
GBS	S pneumoniae	S pneumoniae	S pneumoniae
E coli	N meningitides	N meningitidis	Gram - rods
Listeria	H influenza type b	Enteroviruses	Listeria
	Enteroviruses	HSV	

Streptococcus pneumoniae

- Most common cause of meningitis in all ages
- Gram positive cocci in pairs
- Can follow strep respiratory infection
- Risk factors:
  - Asplenic patients
  - Sickle cell anemia
- Otitis media(children), pneumonia, sinusitis.

N meningitidis

- Gram negative diplococci
- Hematogenous spread
- Transmitted by respiratory droplets
- Collage students and military recruit

Listerias

- Gram positive rod
- Immunocompromised
- neonates, elderly, HIV, steroids, hematological malignancies, organ transplants and pregnancy

Viral

- Enterovirus (coxsackie, echovirus)
- - Arboviral (mosquito-borne diseases)
- - Influenza
- - Herpes simplex virus type2 (especially in infants)
- - Varicella zoster
- - HIV
- - Mumps
- - measles

Viral Meningitis

- Incubation period : 3 to 6 days.
- Duration of the illness : approx 7 to 10 days.
- Milder and occurs more often than bacterial meningitis.
- Affects children and adults under age 30. Most infections occur in children under age 5.
- Most viral meningitis is due to enteroviruses, that also can cause intestinal illness.

Fungal

- Cryptococcus
- Coccidiodes
- Histoplasma
- Mucormycosis
- Aspergillus
- Candida (yeasts)

Parasitic/protozoal

- Angiostrongylus
- Toxoplama
- Hydatid
- Amoeba
- Plasmodium
- Cysticercosis

Clinical Manifestations

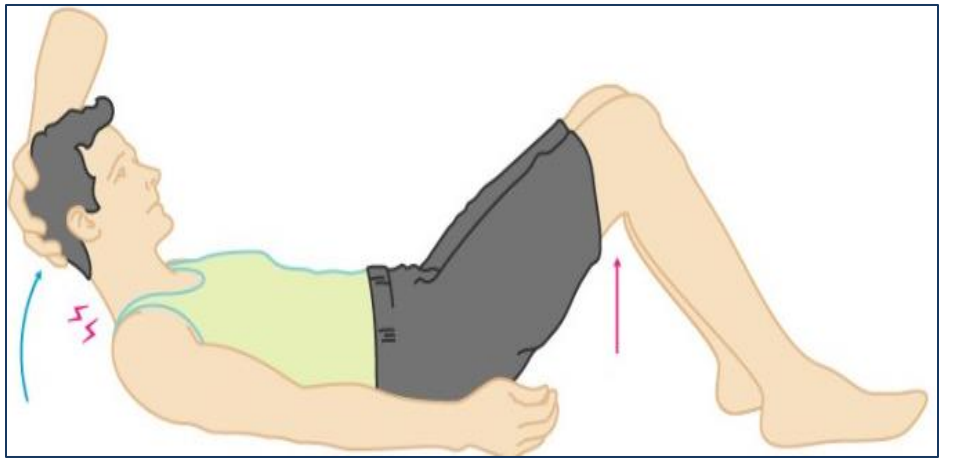
- Common presentation is
- sudden onset
- rapidly progressive manifestations of shock
- Purpura
- disseminated intravascular coagulation (DIC)reduced levels of consciousness often resulting in progression to coma or death within 24 hr. More often, meningitis is preceded by several days of fever accompanied by upper respiratory tract or gastrointestinal symptoms, followed by nonspecific signs of CNS infection such as increasing lethargy and irritability.

The signs and symptoms of meningitis are related to the nonspecific findings associated with a systemic infection and to manifestations of meningeal irritation.

Nonspecific findings include:

- Fever
- Anorexia
- Poor feeding
- Headache
- Symptoms of upper respiratory tract infection
- Myalgias
- Arthralgias
- Tachycardia
- Hypotension
- Various cutaneous signs, such as petechiae, purpura, or an erythematous macular rash

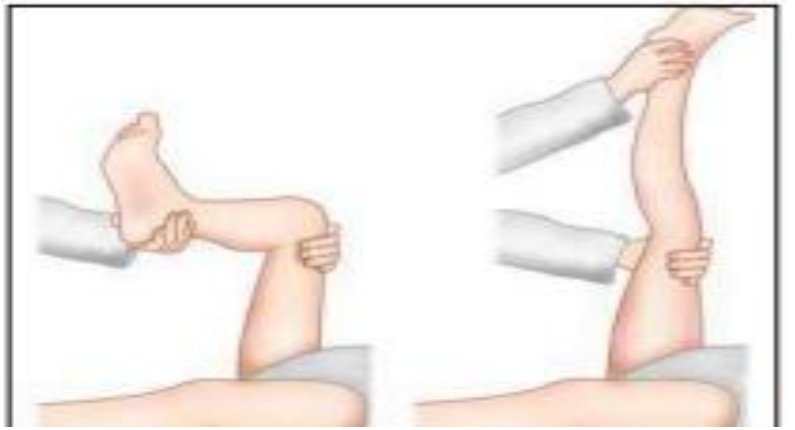
- Meningeal irritation is manifested as:
- Nuchal rigidity- impaired neck flexion resulting from muscle spasm (not actual rigidity) of the extensor muscles of the neck; usually attributed to meningeal irritation.
- Back pain
- Kernig sign (flexion of the hip 90 degrees with subsequent pain with extension of the leg)
- Brudzinski sign (involuntary flexion of the knees and hips after passive flexion of the neck while supine)



Brudzinski's sign

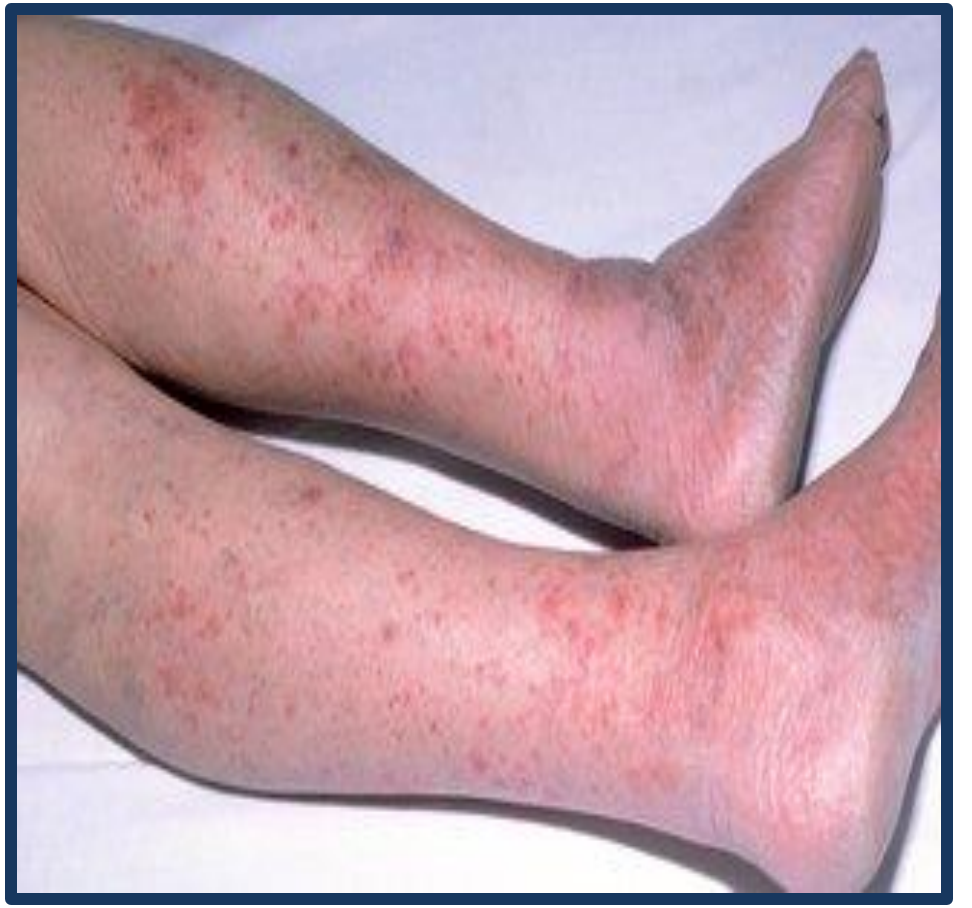
Brudzinski's contralateral reflex sign
hip and knee are passively
flexed on one side

Testing for meningeal irritation (neck rigidity)



Testing for meningeal irritation (Kernig's test

Skin findings: Nonspecific blanching, erythematous, maculopapular rash to a petechial or purpuric rash.
**Approximately 6% of affected infants and children show signs of disseminated intravascular coagulopathy and endotoxic shock.
These signs are indicative of a poor prognosis.



Diagnosis

1. CSF Study

- Confirmed by analysis of the CSF, which typically reveals microorganisms on Gram stain and culture.
- Lumbar Puncture is done for CSF collection.

Contraindications for an immediate LP include:

- evidence of increased ICP, HTN.
- in patients in whom positioning for the LP would further compromise cardiopulmonary function.
- infection of the skin overlying the site of the LP.

Spinal Tap

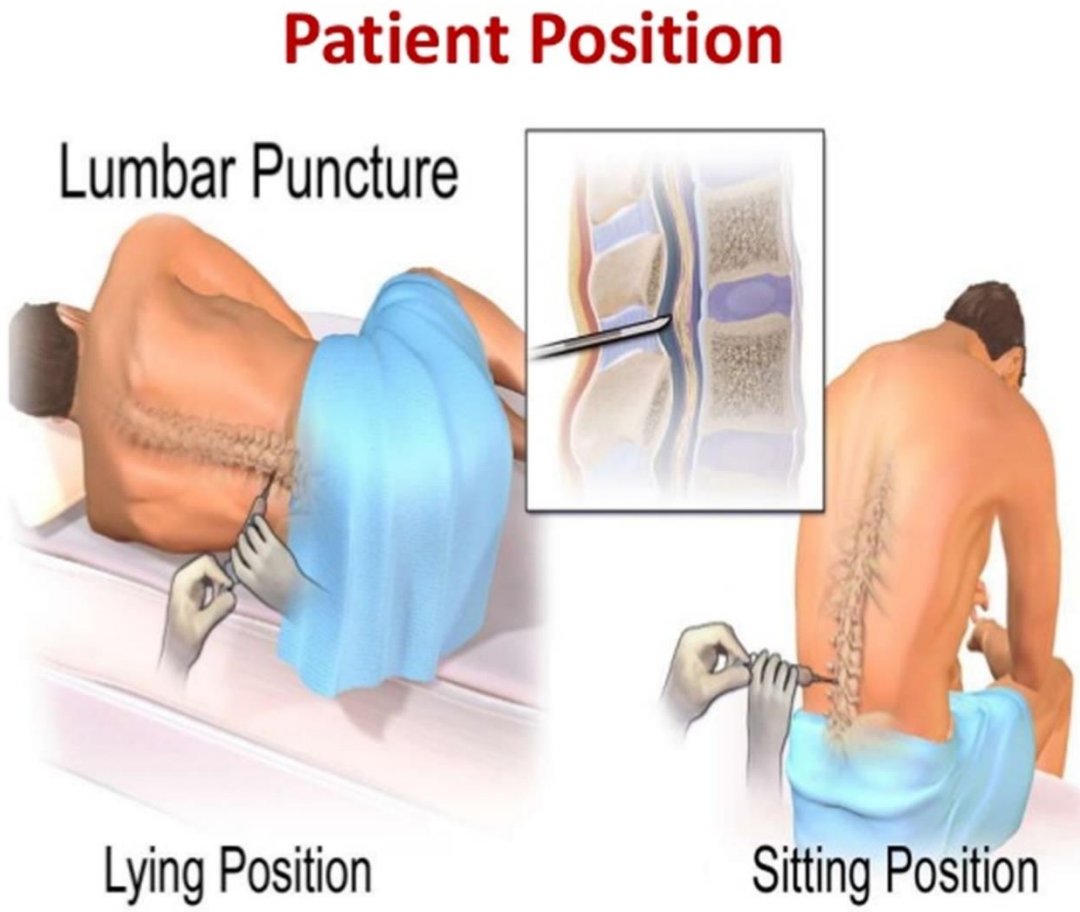
-left lateral position with neck flexion and knee in full extension (fetal position)

-Determine L4 –L5 depend on post. iliac crest .

-Sterile the area in circular manner .

-Inject local anesthesia under the skin .

-By spinal needle enter to subarachnoid space.



Opening pressure

- Patient must lie on their side
- Normal pressure up to 250 mm H2O
- Elevated (> 250 mm H2O):

    bacterial, fungal or TB

    rarely Viral

- We have to take 4 tubes of CSF, for :

1- Cytology

2- Chemistry

3- Gram stain

4- Culture

CSF analysis

	Appearance	RBCs (per mm3)	WBCs (per mm3)	Protein (mg/dL)	Glucose (mg/dL)	Opening Pressure (cm H2O)
Normal	clear	0	0-5 Lymphocytes	15-45	50-80 2/3 of serum glucose	10-20
Bacterial	Cloudy/purulent	↔	↑ (> 1000 PMNs)	↑	↓	↑
Viral	Clear	↔	↑ (monos/ lymphs)	↔ or ↑	↔	↔
Fungal/TB	Fibrin web	↔	↑ (monos/ lymphs)	↑	↓	↑

2. Blood cultures

- Blood cultures reveal the responsible bacteria in up to 80-90% of cases of meningitis. Elevations of the Creactive protein,erythrocyte sedimentation rate, and procalcitonin have been used to differentiate bacterial (usually elevated) from viral causes of meningitis.

3. CT Scan

Cranial computed tomography (CT) is of limited use in acute bacterial meningitis . CT in cerebral oedema may show slitlike lateral ventricle and areas of low attenuation.

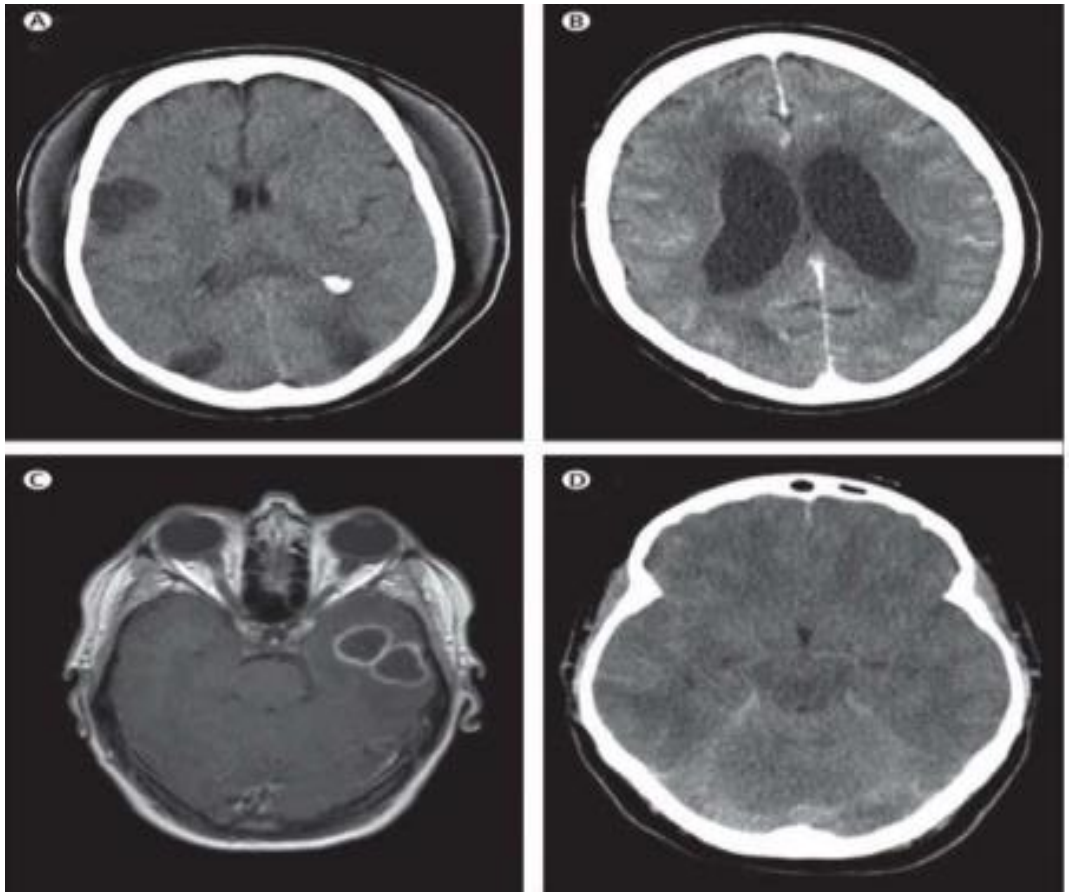


Table 1. Differential Diagnosis of Meningitis

Common	**Uncommon** *(continued)*
Bacterial meningitis	Human immunodeficiency virus
Viral meningitis	Leptomeningeal carcinomatosis
Uncommon	Lyme disease (neuroborreliosis)*
Behçet syndrome	Neoplastic meningitis
Benign recurrent lymphocytic meningitis (Mollaret meningitis)	Neurosarcoidosis
Central nervous system abscess	Neurosyphilis*
Drug-induced meningitis (e.g., non-steroidal anti-inflammatory drugs, trimethoprim/sulfamethoxazole)	Parasitic meningitis*
Ehrlichiosis	Systemic lupus erythematosus
Fungal meningitis	Tuberculous meningitis*
	Vasculitis

**—More common in geographic areas with higher incidence of these infections.*

Treatment

- Antibiotics should be administered rapidly and may be given empirically up to 2 hours before an LP.
- Dexamethasone may be beneficial in bacterial meningitis, especially S pneumoniae or H influenzae, if given 15–20 minutes before antibiotics.

Recommended Empiric Antibiotics for Suspected Bacterial Meningitis

Age or Predisposing Feature	Antibiotics
Age 0-4 wk	Ampicillin plus either cefotaxime or an aminoglycoside
Age 1 mo-50 y	Vancomycin plus cefotaxime or ceftriaxone
Age >50 y	Vancomycin plus ampicillin plus ceftriaxone or cefotaxime plus vancomycin
Impaired cellular immunity	Vancomycin plus ampicillin plus either cefepime or meropenem
Recurrent meningitis	Vancomycin plus cefotaxime or ceftriaxone
Basilar skull fracture	Vancomycin plus cefotaxime or ceftriaxone
Head trauma, neurosurgery, or CSF shunt	Vancomycin plus ceftazidime, cefepime, or meropenem

Complications

- Death
- Hydrocephalus
- Hearing loss
- Seizures
- SIADH

- Bacteremia can complicate meningitis
- Meningococcemia
  - septic shock, fever, chills, tachycardia and hypotension
  - purpuric rash
  - DIC
  - Waterhouse-Friderichsen syndrome

Close contacts must receive prophylaxis

- Rifampin
- Ceftriaxone

Q: 24 YO female, presented with headache, fever, & deterioration in level of consciousness, brain CT was free, the L.P s (values shows high WBS, LOW glucose).

Q1: what is the Dx?

Acute meningitis.

Q2: give 2 lines of treatment.

IV antibiotics , Anti-pyretics .

Q3: give one major complication.

brain abscess, seizure, encephalitis .

Rheumatology

Done by:

سندس الدهيسات & إيناس الريان

طارق أبو لبدة & مهند العقيل

ربى العمرو & رزان الرفوع

مؤيد جرادات

Autoantibodies

- RF: IgM against IgG (RA, Sjögrens)
- Anti CCP: specific for RA
- ANA (SLE)
- Anti ds DNA (SLE)
- Anti RO (SS-A), LA (SS-B): Sjögrens
- Anti U1-RNP: MCTD
- Anti Scl70 (limited), Anti centromere (diffuse): scleroderma
- Anti histone: drug induced LE
- ANCA (p-, c-): vasculitis
- Celiac: anti endomyseal, TTG antibodies
- DM type 1: Anti GAD

Antibodies in Medicine

- RF: IgM antibody against Fc portion of IgG. Sensitive but not specific for RA.
- Anti-CCP (ACPA): Specific for RA.
- ANA: non specific for rheumatoid diseases (associated more with SLE).
- Anti-dsDNA, anti-Smith antibodies: specific for SLE
- Anti-Ro (SS-A) and Anti-La (SS-B): Specific for Sjogrens Syndrome.
- Anti-U1 RNP (ribonucleoprotein): Mixed connective tissue diseases.
- Anti-Histone antibodies: Drug induced lupus.
- P-ANCA (anti-myeloperoxidase-antineutrophil cytoplasmic antibodies), or C-ANCA (anti-proteinase-antineutrophil cytoplasmic antibodies): Vasculitis, P-ANCA may also be seen in ulcerative colitis.

- Anti-GAD (glutamic acid decarboxylase): Type I DM
- Islet cells antibodies: Type I DM
- Anti-TTG (tissue transglutaminase): Celiac disease
- Anti-endomysial antibodies: Celiac disease
- Anti-scl-70 (anti-topoisomerase): diffuse scleroderma
- Anti-centromere: CREST syndrome (limited scleroderma)
- Anti-Jo-1, Anti-Mi-2, anti-SRP: polymyositis/dermatomyositis
- Anti-smooth muscle antibodies: autoimmune hepatitis
- P-ANCA (anti-myeloperoxidase-antineutrophil cytoplasmic antibodies), or C-ANCA (anti-proteinase-antineutrophil cytoplasmic antibodies): Vasculitis, P-ANCA may also be seen in ulcerative colitis.

- The previous list does not include a full list of antibodies that is present in Medicine, but hopefully it covers the most significant diseases and syndromes.

Synovial fluid

1. Cell:

- Normal: 0-200
- Non inflammatory: 200-2000
- Inflammatory: 2000-2,000
- Septic: > 50,000

Crystal

Culture

1.Rheumatoid arthritis (RA)

RA 2010 criteria for diagnosis

The 2010 rheumatoid arthritis classification criteria

Involvement of swollen and tender joints	Points
1 medium-large joint	0
2-10 medium-large joints	1
1-3 small joints	2
4-10 small joints	3
Greater than 10 joints (at least 1 must be small)	5
Serology	
Neither RF nor ACPA positive	0
One low-positive titer on at least one test	2
One high-positive titer on at least one test	3
Duration of synovitis	
Less than 6 weeks	0
6 weeks or longer	1
Acute-phase reactants	
Neither CRP nor ESR abnormal	0
Abnormal CRP or abnormal ESR	1

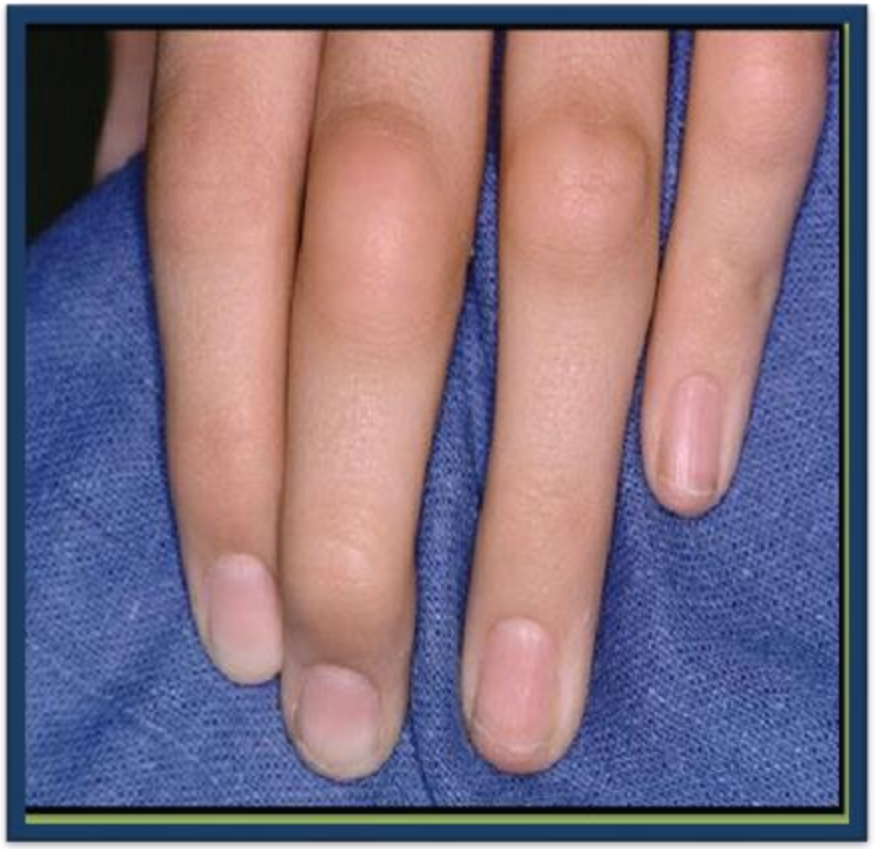
Source: Arthritis Rheum. 2010;62:2569-81

IMNG Medical Media

A score of 6 or more is likely to be RA

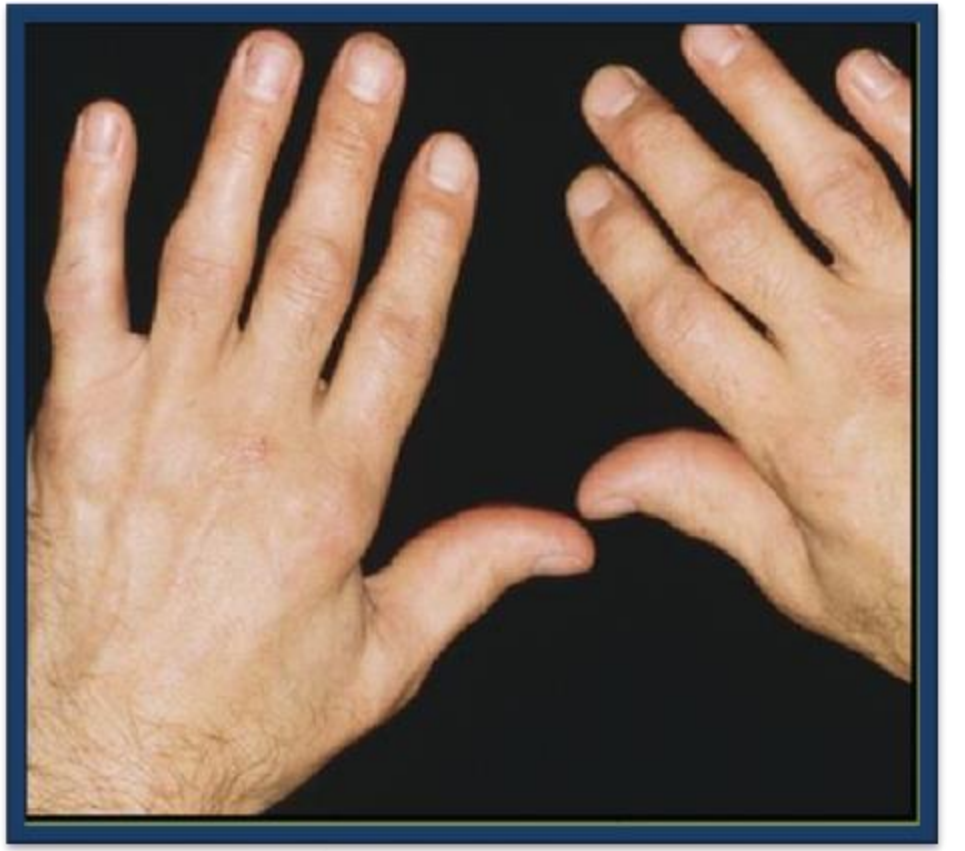
Q: What is Your Spot Dx?

Rheumatoid arthritis (RA)



Q: The pt complains of morning stiffness & pain in the joints of his hands. What's the Dx.?

Rheumatoid arthritis (RA)

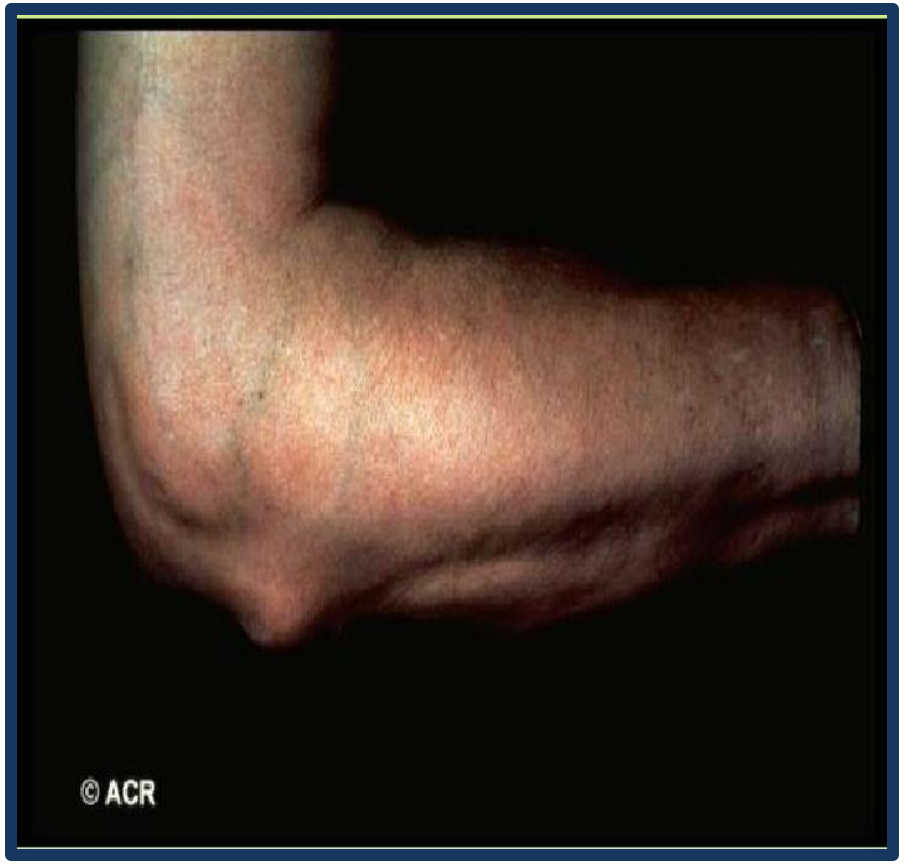


Q: A-What is this finding?

Rheumatoid nodule

B-How to confirm the diagnosis?

clinical diagnosis ,RF, Anti-CCP, x-rays(erosive changes ,deformities ,etc)



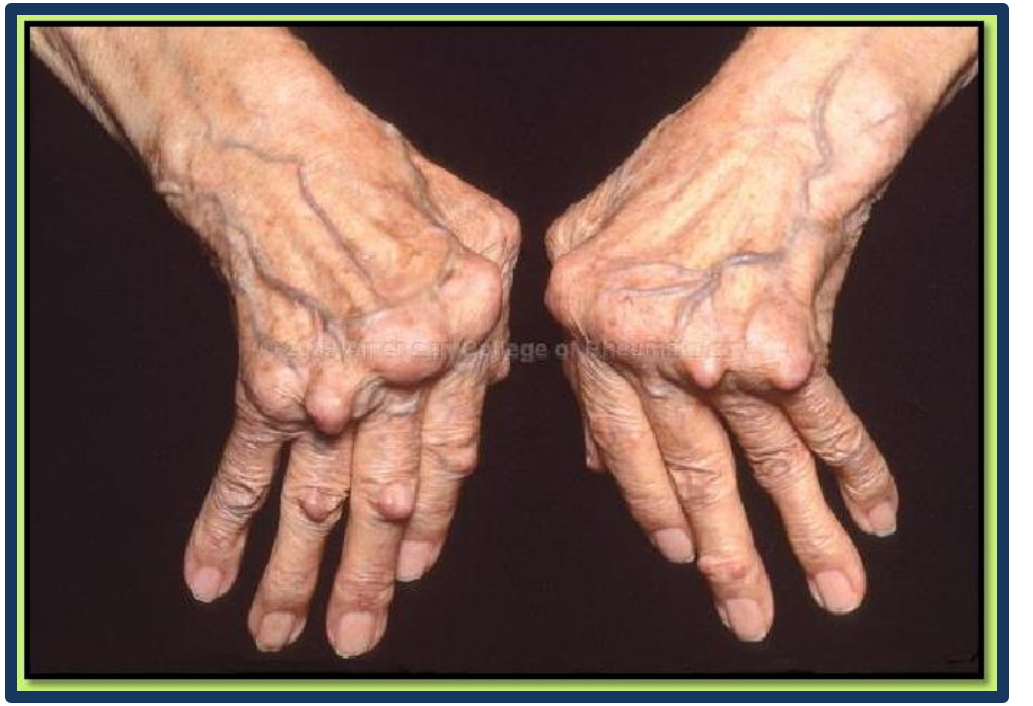
Q: Female with joints pain in both hands & dyspnea.

- What is the diagnosis?

Rheumatoid arthritis

- What is the sign you look for on olecranon fossa?

Subcutaneous rheumatoid nodules.



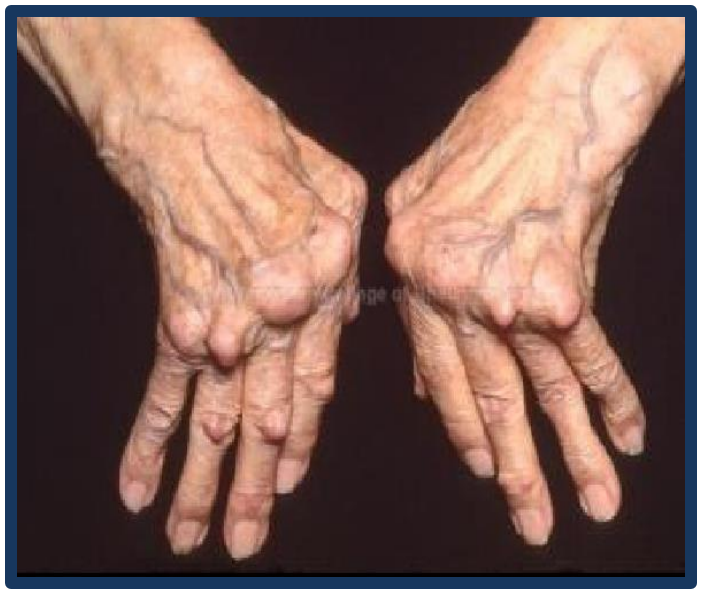
This picture shows ulnar deviation

Dyspnea: caused by lung fibrosis or pleural effusion

Q: Mention 3 drugs which stop the progression of this disease.
1.Methotrexate 2.Infliximab 3.Hydroxychloroquine 4. Etanercept.

•Gold salts and penicillamine are no longer used in RA

•New molecular biological agents for treatment of RA (bDMARDs)
Tofacitinib
Abatacept (not sure)



DMARD's

Disease Modifying Anti Rheumatoid Drugs

Cute - Chloroquine

P - Penicillamine

A - Azathioprine

G -Gold Salts

L - Leflunomide

I - Immunosuppresant drugs

Malika - Methotrexate (DOC)

Sherawat - Sulfasalazine

TNF-@ Inhibitors
1) Adalizumab
2) Etanarcept
3) Infliximab

IL-1 Receptor Antagonist
Anakinra

IL-6 Inhibitor

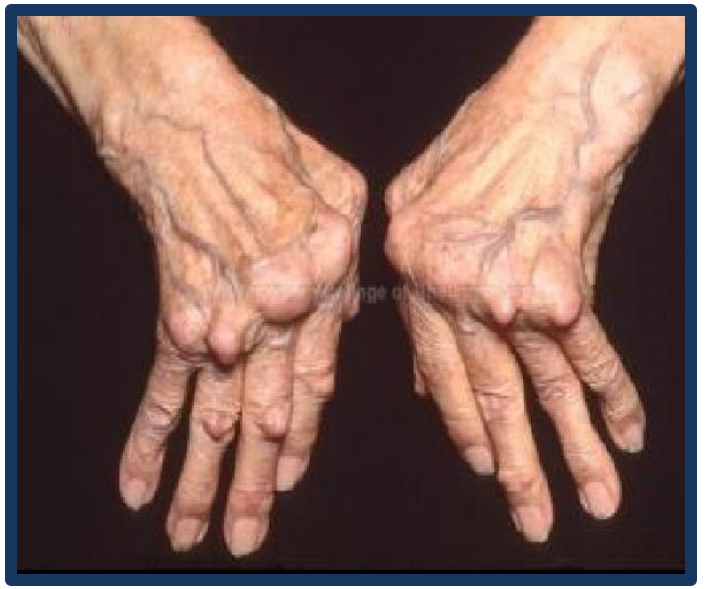
Tocilizumab

Q: This photo is for the hand of a female pt who was diagnosed previously with Rheumatoid Arithritis. What deformity can you see in this photo?

Ulnar Deviation.

What pulmonary manifestations can you expect in this pt?

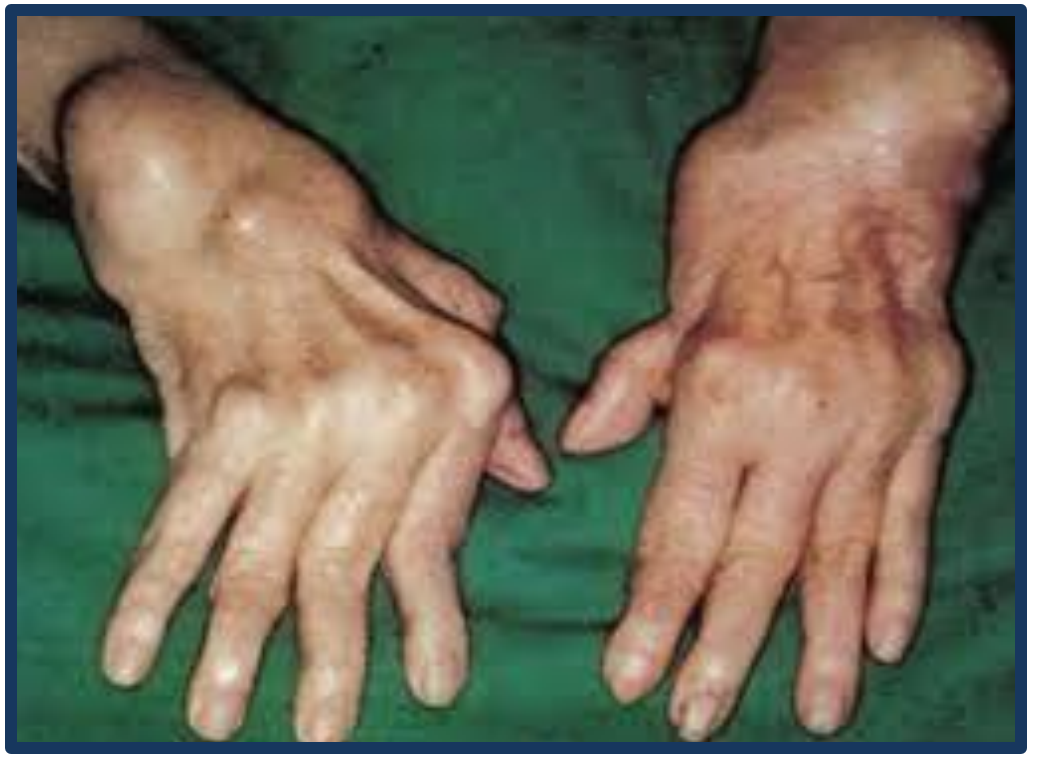
Interstitial Lung Diseases [Lung Fibrosis]; Caplan's Syndrome - [Intrapulmonary Nodules].



Q: The following patient has been complaining of joint pain for several years and was diagnosed with rheumatoid arthritis.

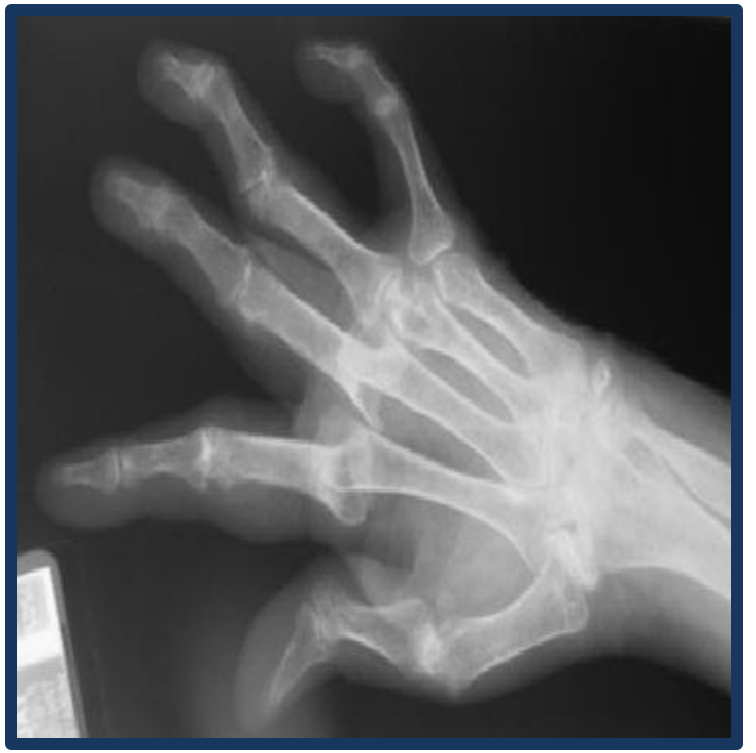
Name 2 deformities in the image

1. Swanneck deformity
2. Ulnar deviation



Q: 56 YO pt complaining of general aches & pain, but also some stiffness & swelling in her both hands for the past 2 months that is worse in the morning. What's Your Dx.?

rheumatoid arithritis (Swan neck and butonniere deformities are both present).



Q: A 30 years old female patient comes to the clinic complaining of morning stiffness, pain at the MCPs and PIPs, and stiffness of joints that is more pronounced after prolonged inactivity. What is the Diagnosis?

RA

What is the explanation of joint stiffness after prolonged inactivity?

Gel phenomenon

The same patient comes again after 5 years, but is now complaining of dryness of mouth and blurred vision. What is your diagnosis?

Secondary Sjogren Syndrome (keratoconjunctivitis sicca)

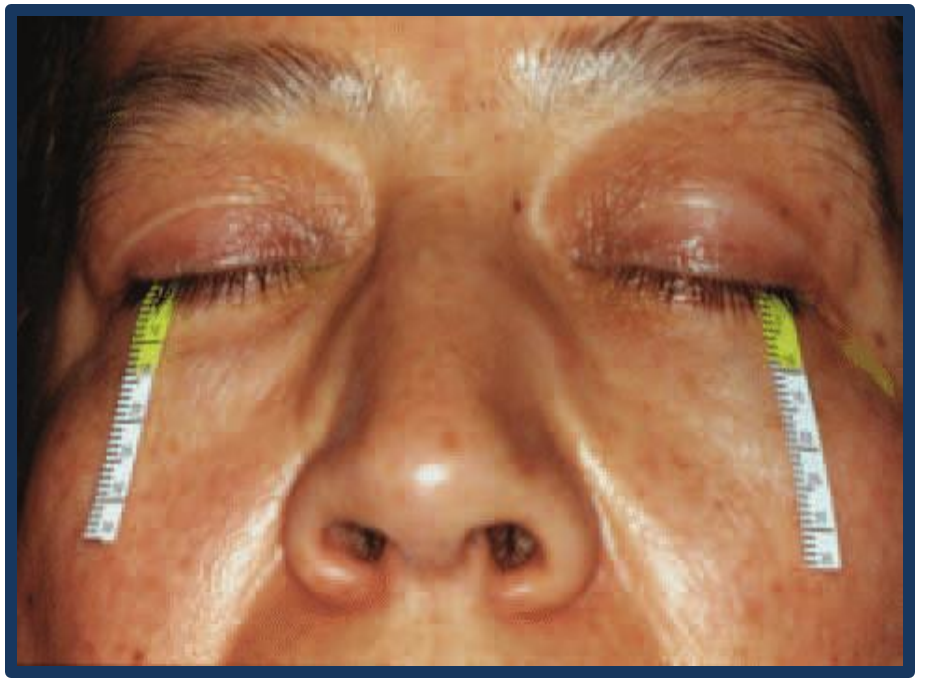
Continue

This test was done to the patient, what is the name of this test?

Schirmer test

What is it used for?

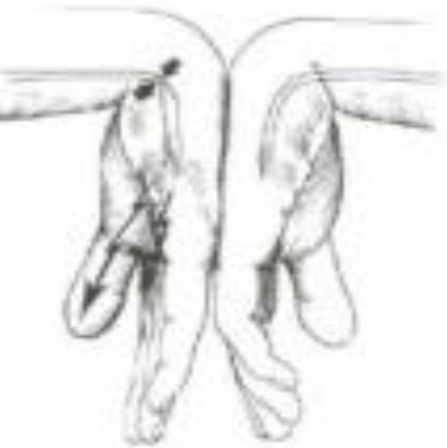
To measure tears production in each eye to diagnose Sjogren Syndrome



Q: A patient who was previously diagnosed with RA comes to the clinic complaining of numbness and paresthesia in her wrists. The numbness is exacerbated during activity. Tinel's sign and Phalen's test are postivie. What is the diagnosis?

Carpal tunnel syndrome secondary to RA

Phalen's Test

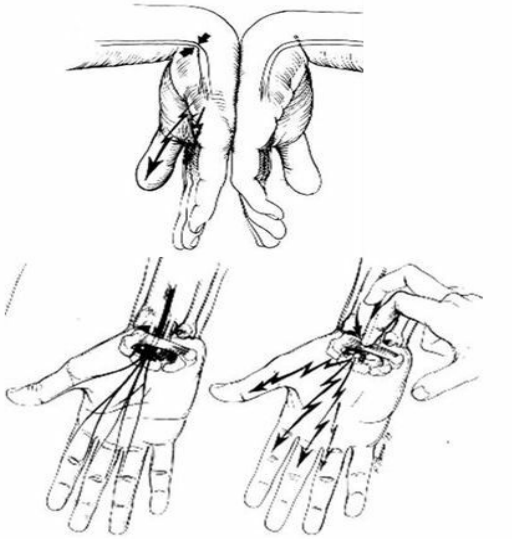


- Therapist flexes client's wrists manually and holds together for one minute. Positive test elicits tingling in thumb, index finger, and middle and lateral half of the ring finger and is indicative of Carpal Tunnel Syndrome.

Special Tests

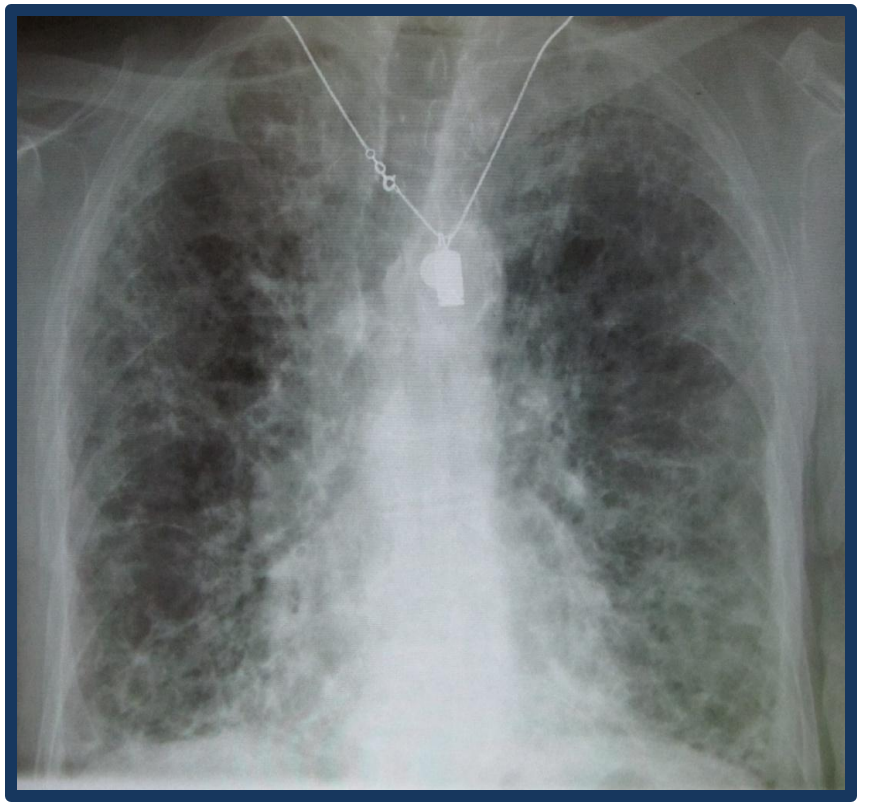
Phalen's & Tinel's Tests

- Phalen's
  - Wrist flexion to maximum for 60 sec
- Tinel's
  - Tapping over transverse carpal ligament
- Symptoms
  - Pain
  - Anesthesia
  - Paresthesia



Q: A known case of Rheumatoid arthritis presents with progressive shortness of breath, describe your finding in this X ray

Diffuse Reticulonodular infiltrates indicative of pulmonary fibrosis secondary to Rheumatoid arthritis

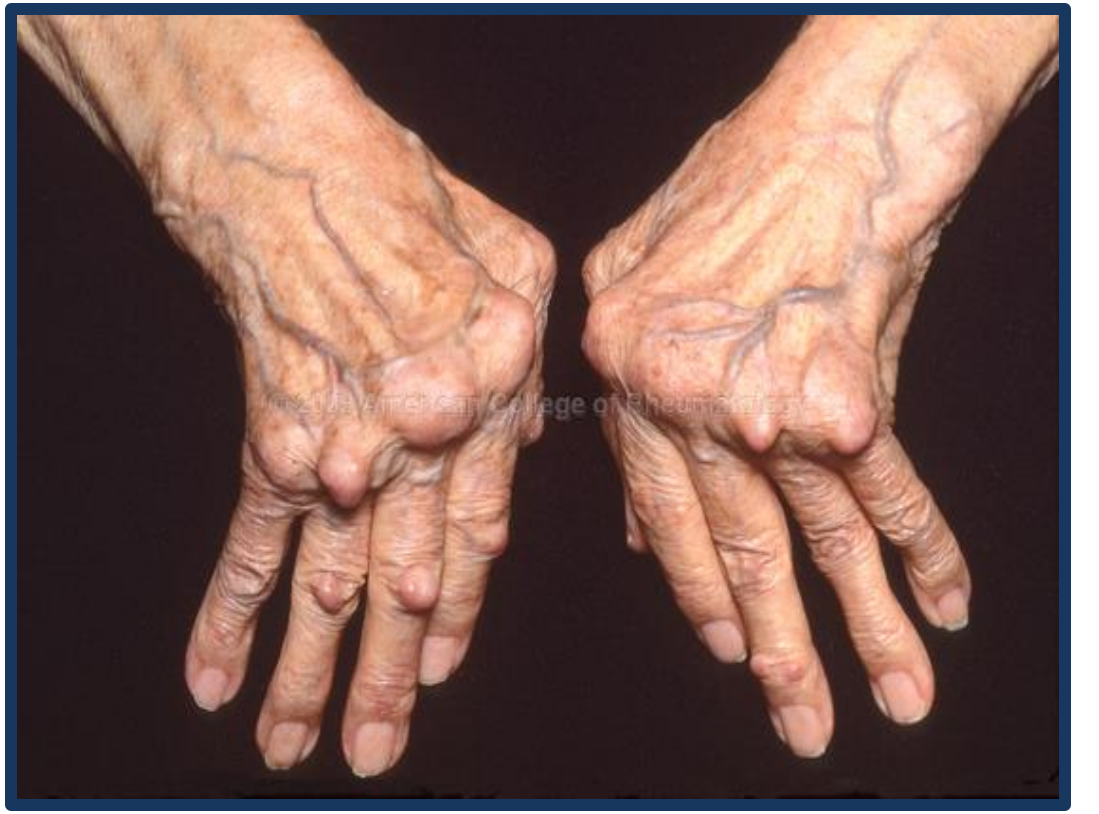


Q: This patient had high creatinine. What's the cause?

One of tofacitinib sides effects , NSAIDS

Mention 2 drugs that modify the progression of this condition?

Methotrexate, Hydroxyurea

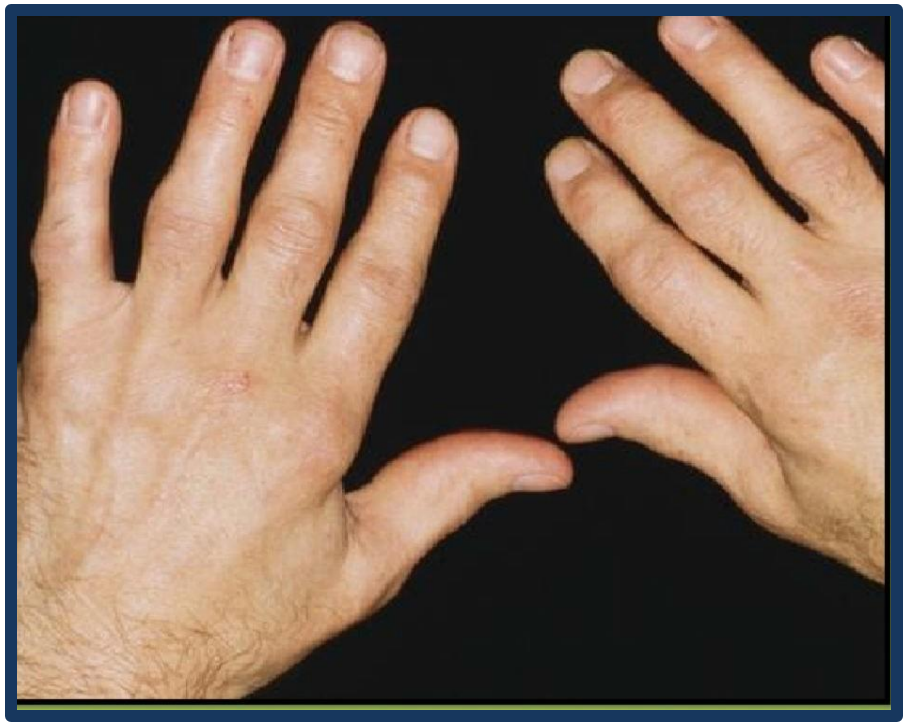


Q: mention 2 findings in this RA pt

Swan neck deformity

Rheumatoid nodules

Note* some said ulnar deviation , I don't think there was ulnar deviation on the exam pic



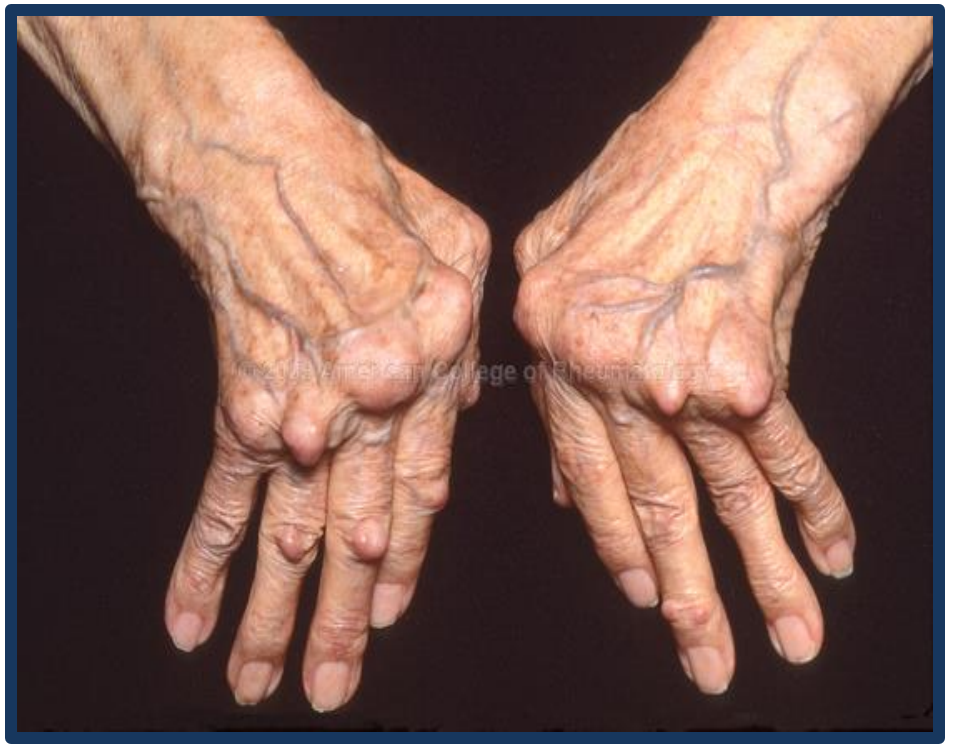
Q: female with joints pain in both hands and dyspnea.

1- what is the diagnosis?

RA.

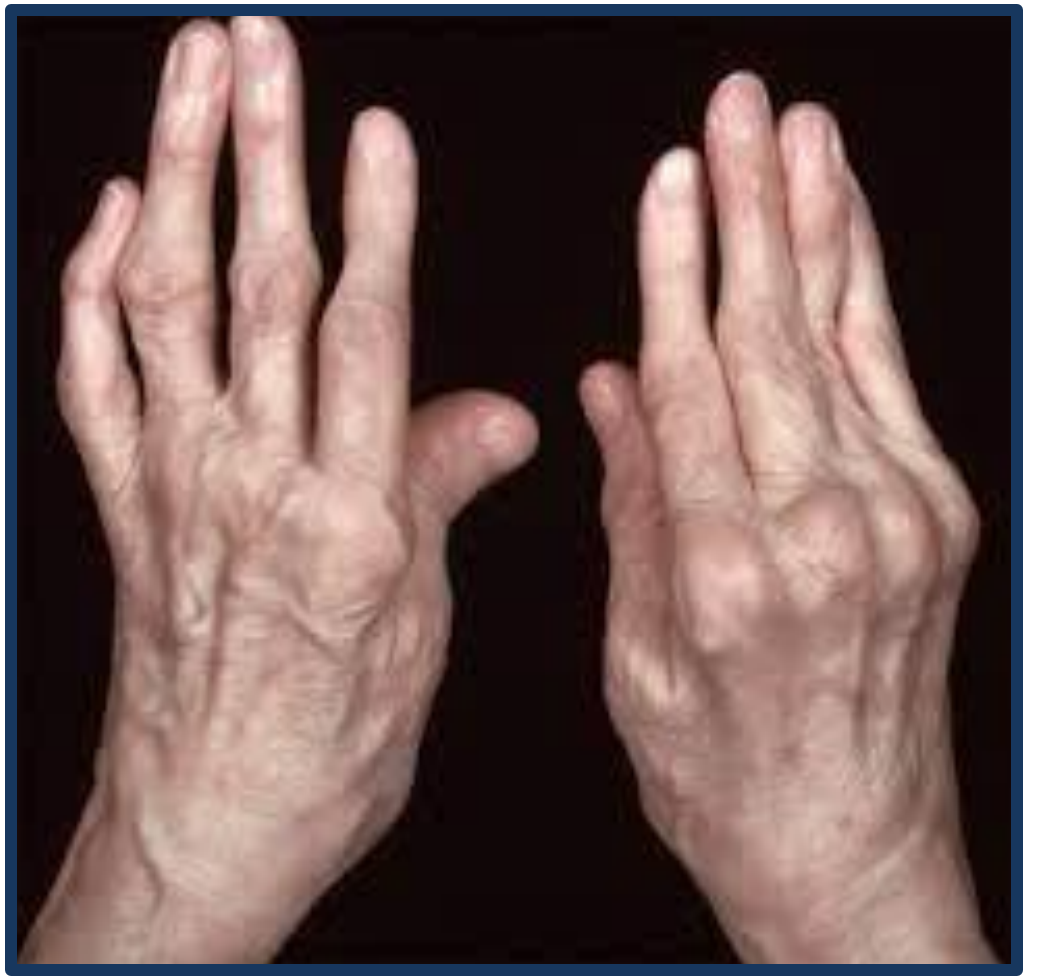
2- what is the cause of Dyspnea?

lung fibrosis and nodules



Q:This pt has developed gradual SOB , what's the Cause ?

Pulmonary fibrosis



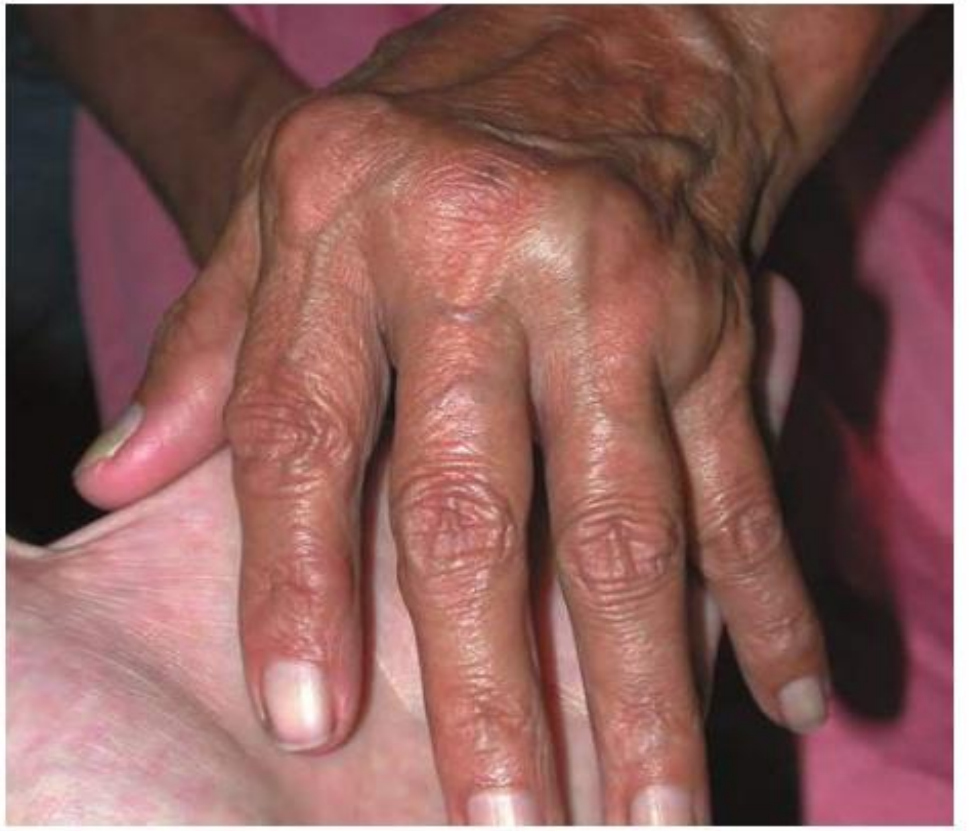
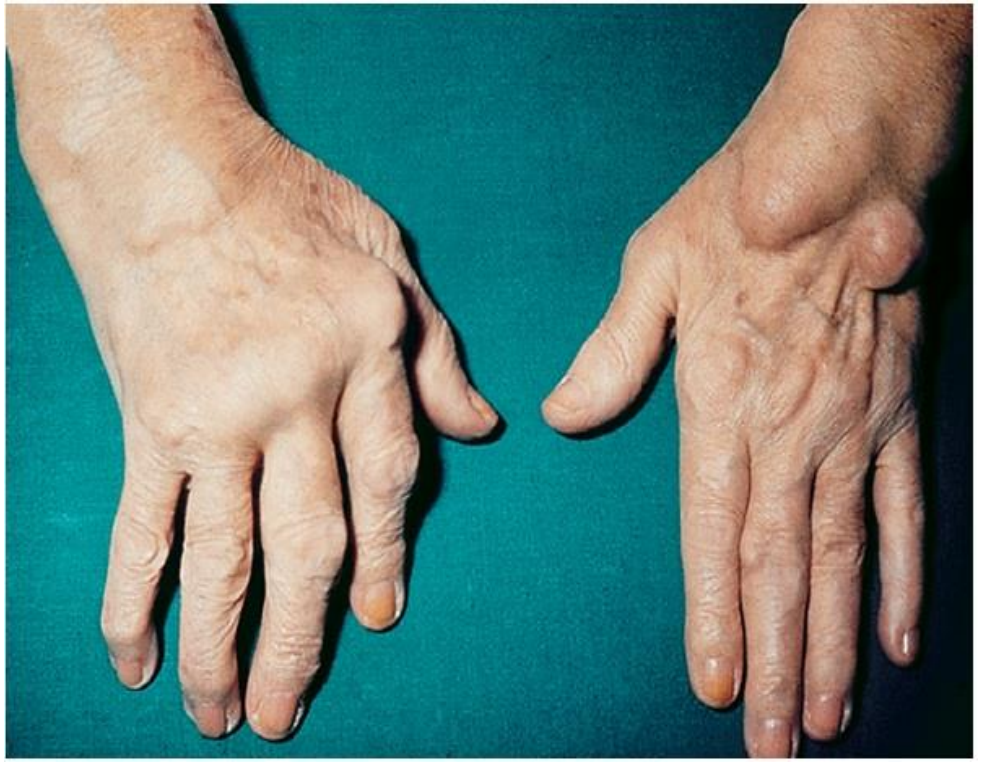


FIGURE 98-6 Rheumatoid arthritis showing ulnar deviation of the fngers at the metacarpophalangeal joints. (Reproduced with permission from Richard P. Usatine, MD.)

Finding :

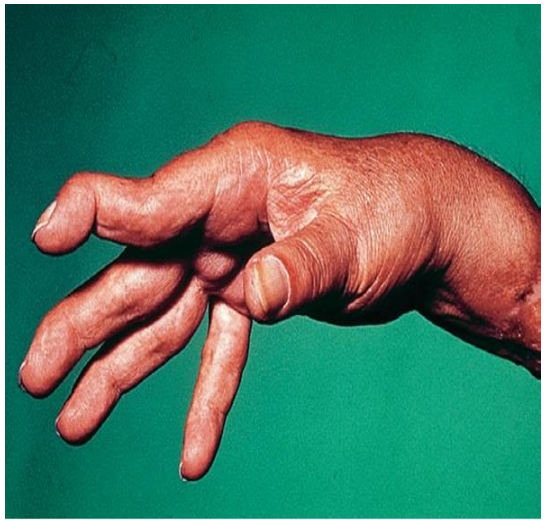
Ulnar deviation of the fingers, wasting of small muscles and synovial swelling at the wrists, extensor tendon sheaths , PIPjs and MCPjs.

Diagnosis: Rheumatoid Arthritis

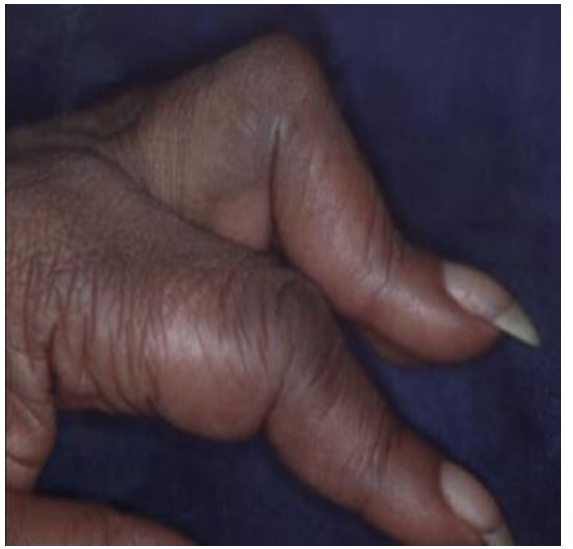


DX: Rheumatoid Arthritis

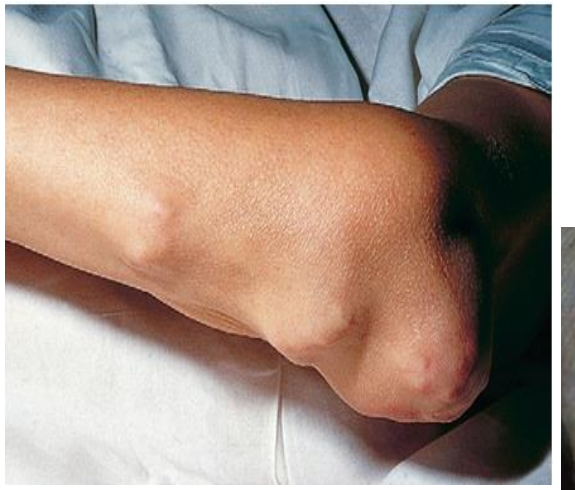
Swan neck deformity



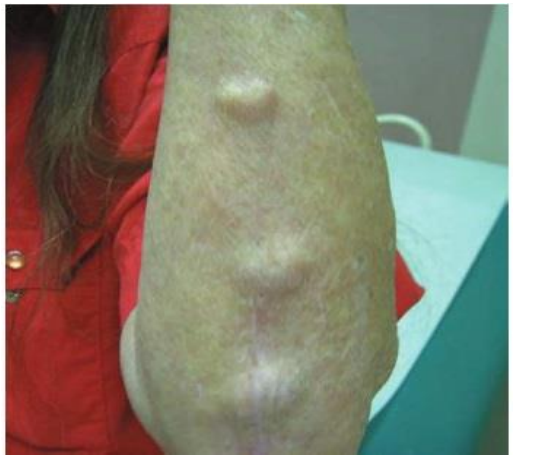
Button hole deformity
(boutonniere deformity)

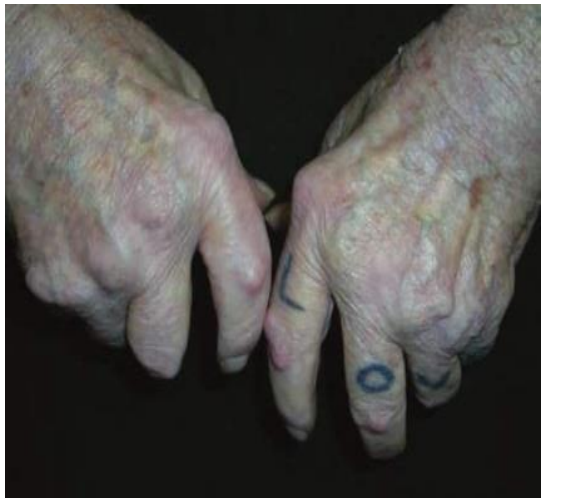


Olecranon bursitis



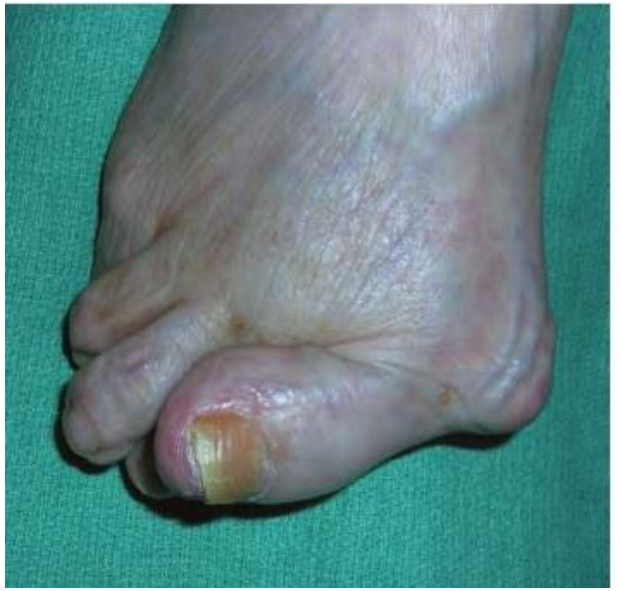
Rheumatoid Nodules

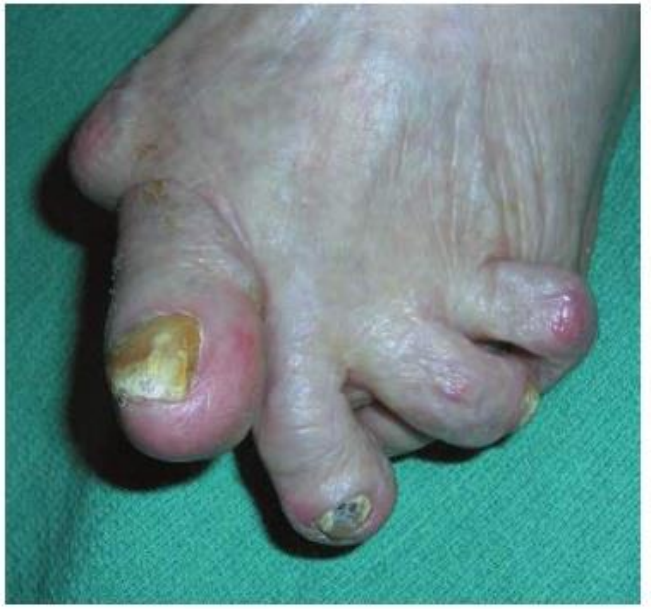




DX: Rheumatoid Arthritis

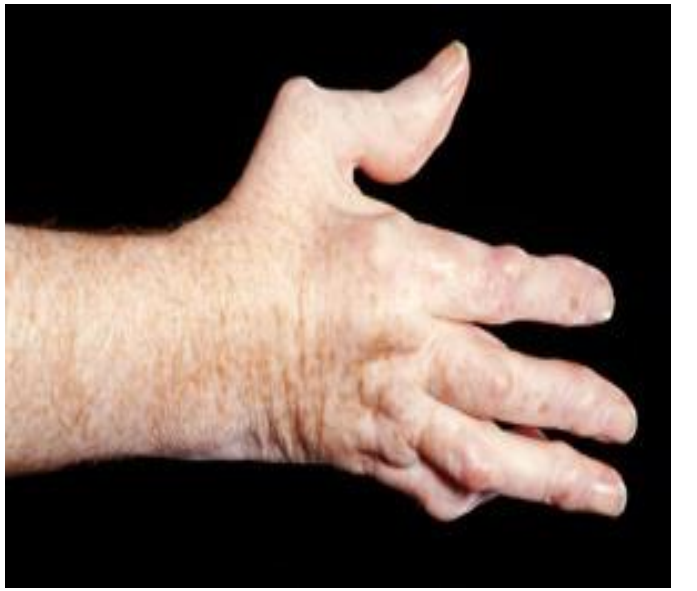
Deviation at the metatarsophalangeal joints



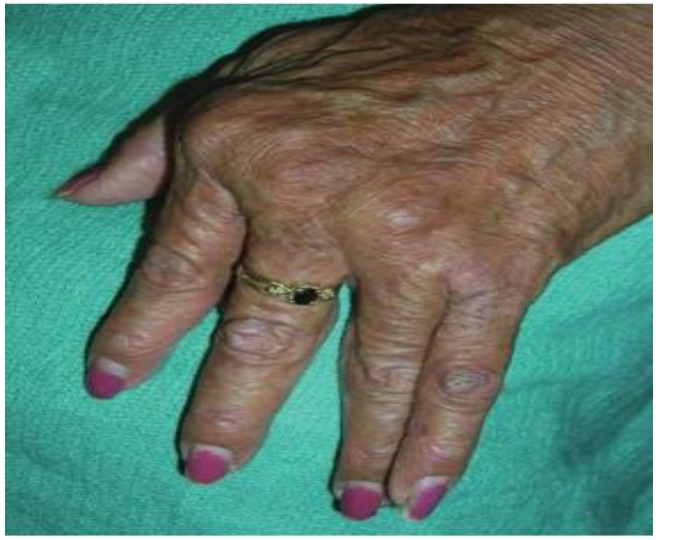


subluxation of the first metatarsophalangeal joint

"Z" deformity of the thumb

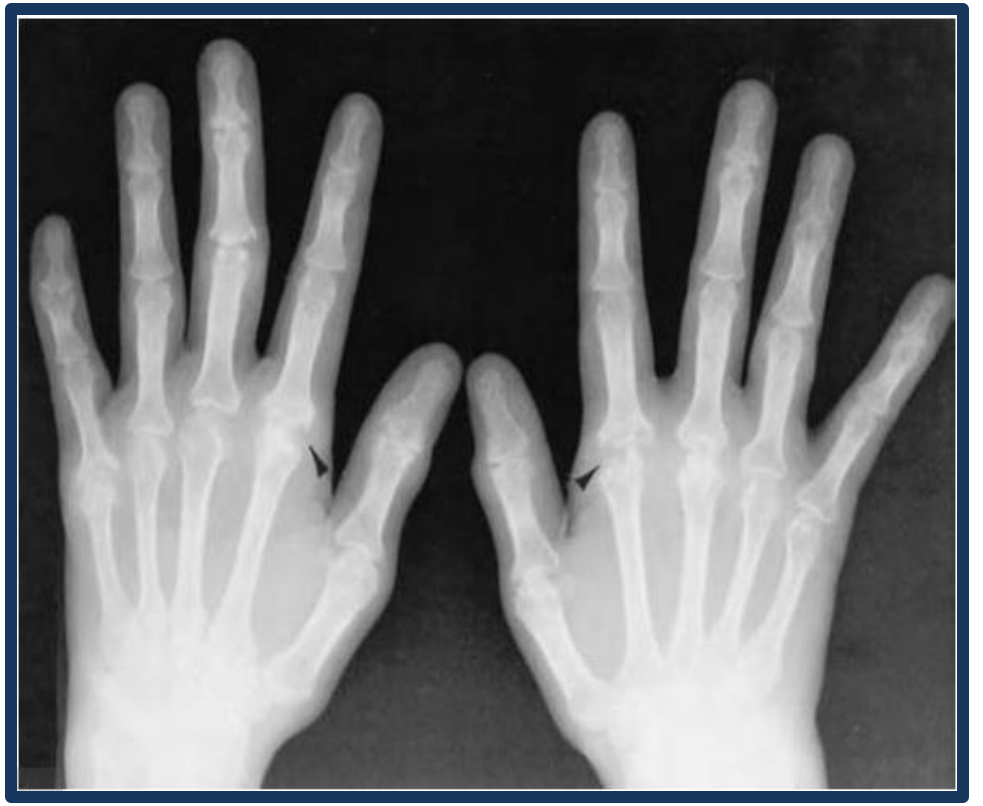


Ulnar deviation



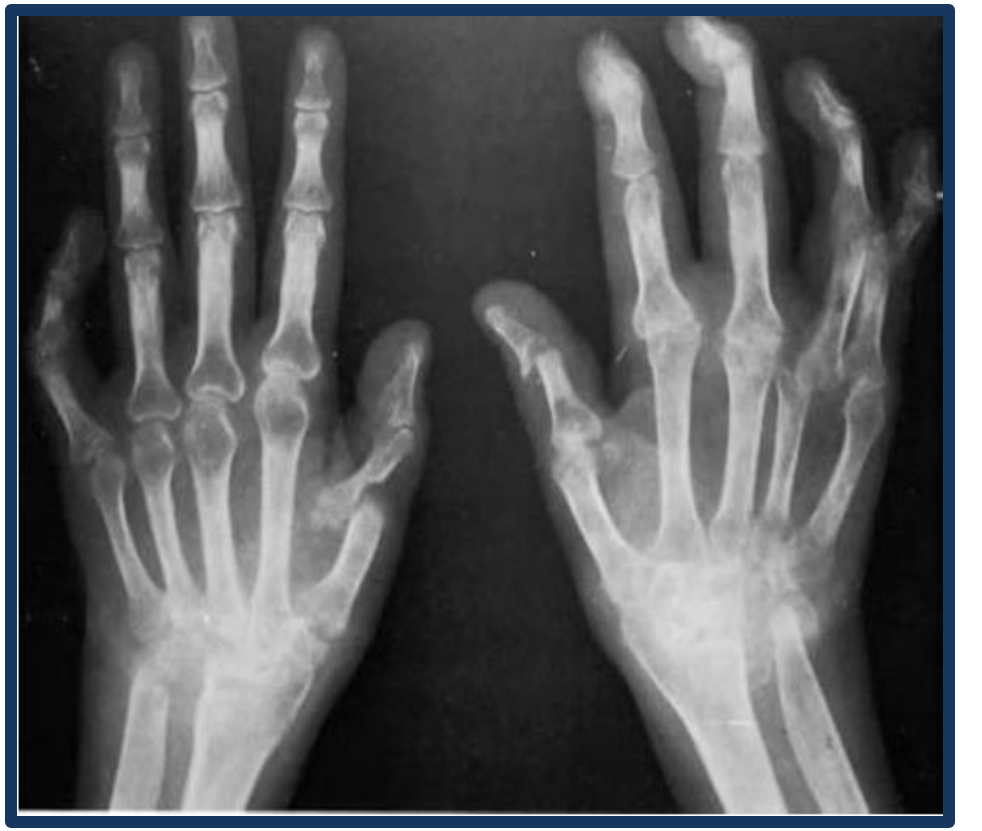
DX: Rheumatoid Arthritis

F: Hand radiographs in long-standing rheumatoid arthritis demonstrating carpal destruction, radiocarpal joint narrowing, bony erosion (arrowheads), and soft-tissue swelling



DX: Rheumatoid Arthritis

F: Radiocarpal joint destruction, ulnar deviation, erosion of the ulnar styloid bilaterally, dislocation of the left thumb PIP joint, and dislocation of the right fourth and fifth MCP joints

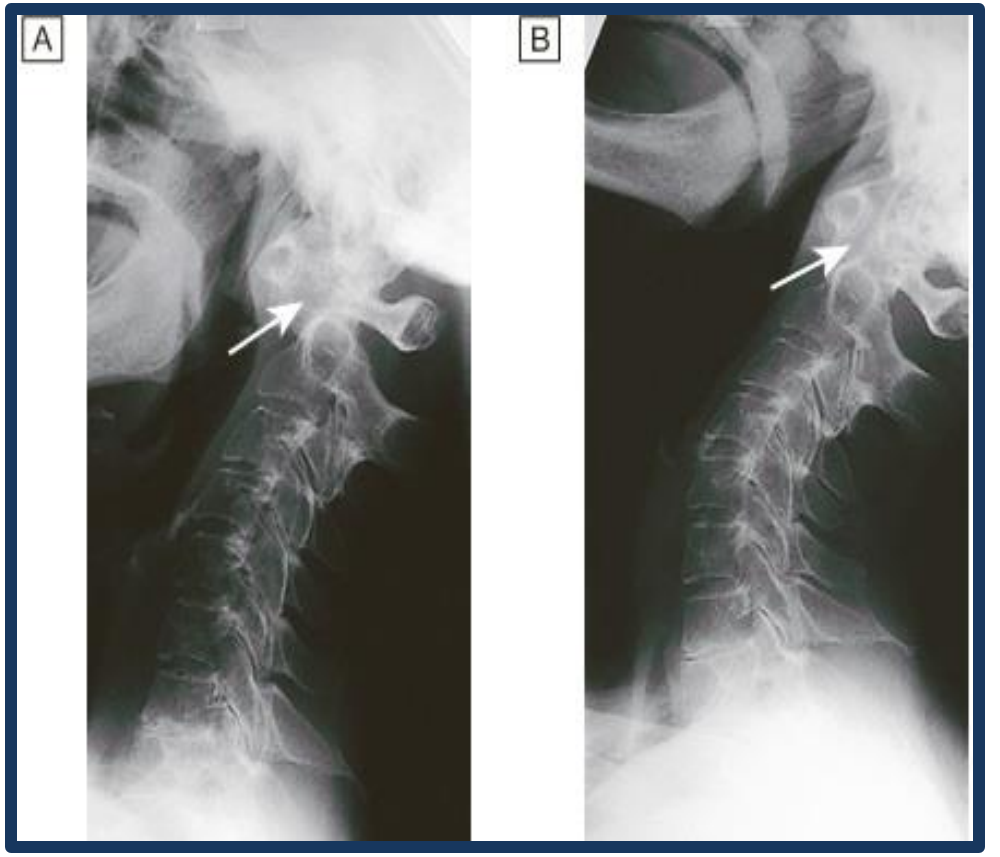


DX: Rheumatoid Arthritis

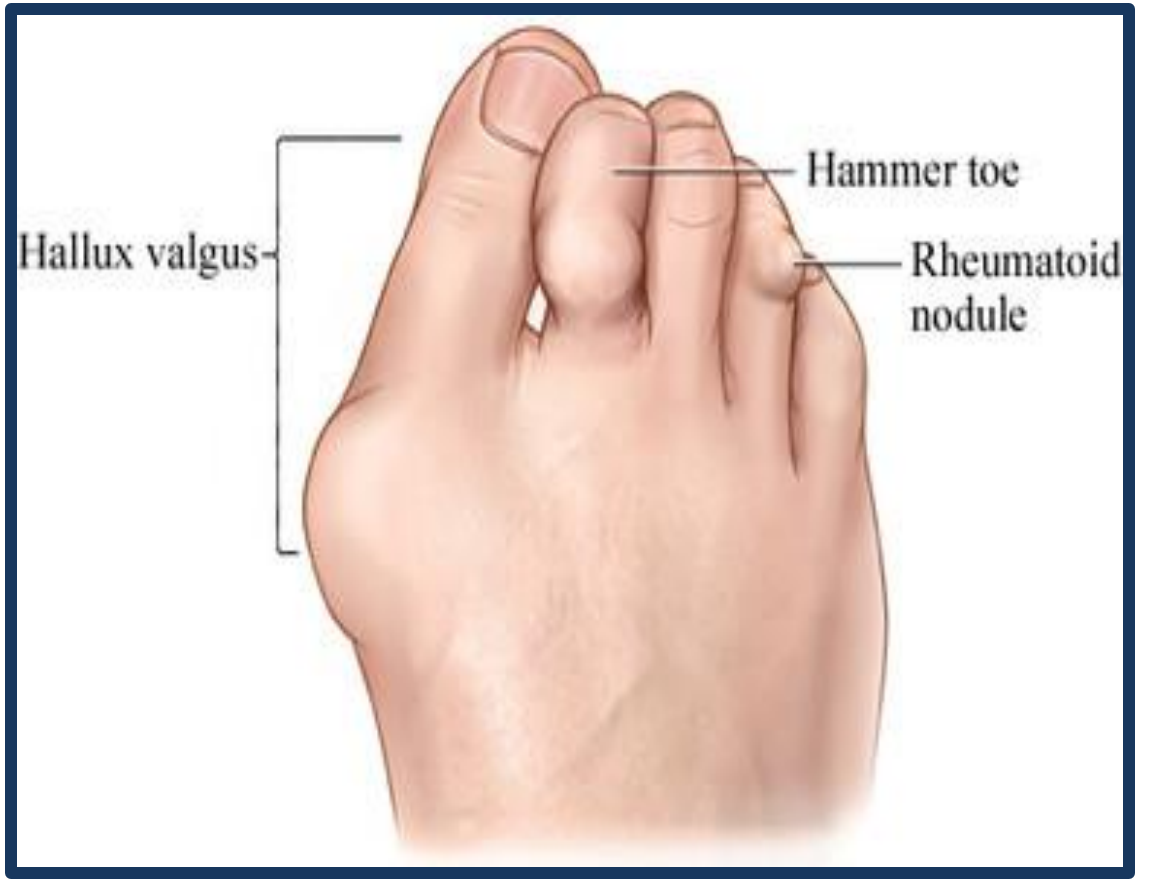
F: subluxation of cervical spine

A. flexion, showing widening of the space(arrow)

B. extension, showing reduction in this space



Changes of RA affecting the foot



RA	Osteoarthritis
—	Osteophytes
Juxtarticular osteopenia	
Joint space narrowing	Joint space narrowing
—	Subchondral cysts
Subchondral sclerosis	Subchondral sclerosis
Soft tissue swelling	—

2.SLE

Q:Sam is 24 yr old male, presents to his GP complaining of symmetrical small joint pain, with prolonged fatigue and fever.

Identify the condition

SLE

Differential diagnosis

SLE , Rheumatoid Arthritis

Causes

- *the cause is unknown ,but there are Environmental and genetic factors*
- Env factors (infections, antibiotics such as sulfa ,other drugs e.g procainamide and hydralazine ,UV radiation ,extreme stress ,hormones)

Clinical Presentation

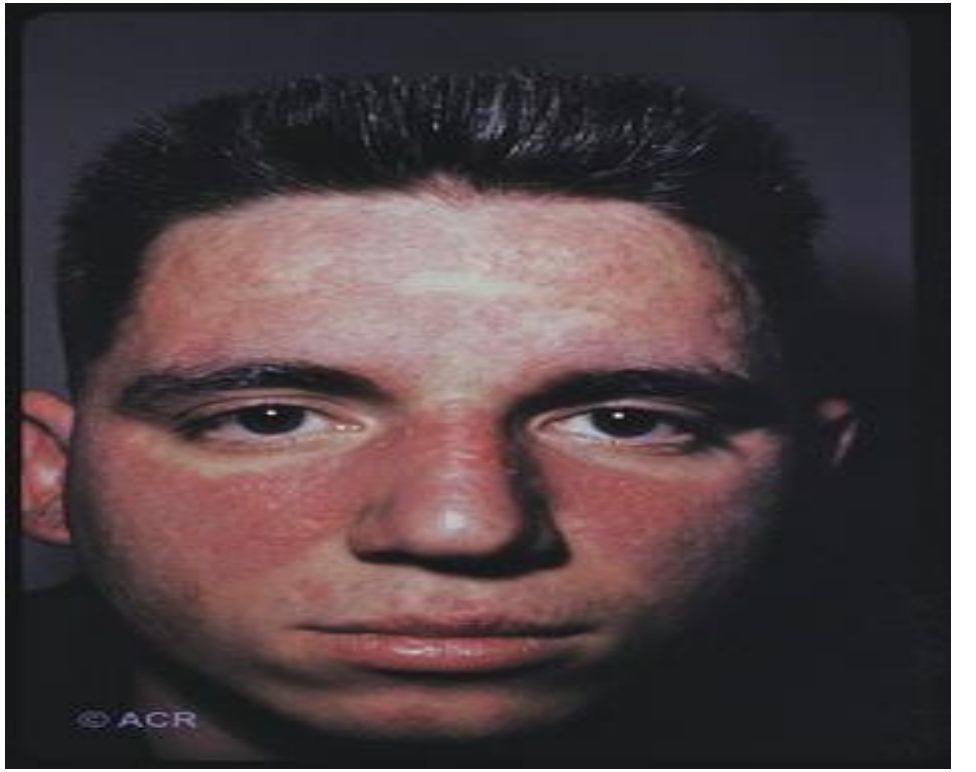
Small joints arthralagia, extreme or prolonged fatigue, fever ,butterfly rash, photosensitivity, anemia , vasculitis, , raynaud's phenomenon, oral ulcers, discoid lupus, , leucopenia, lymphopenia, thrombocytopenia, ,pericarditis, myocarditis endocarditis, pleuritis, pulmonary hypertension, pulmonary emboli, pulmonary hemorrhage , glomerulonephritis, seizures, psychosis, polyneuropathy , alopecia

Investigations

serological tests : Anti -nuclear antibody (ANA),Anti-dsDNA antibody ,Anti-smith antibody , anticardiolipin antibodies ,serum complement levels , Depending on organ involved (CBC,KFT, liver enzymes)

Treatment or Management

NSAIDs, hydroxychloroquine , corticosteroids, immunosuppressive drugs (cyclophosphamide,azathioprine)

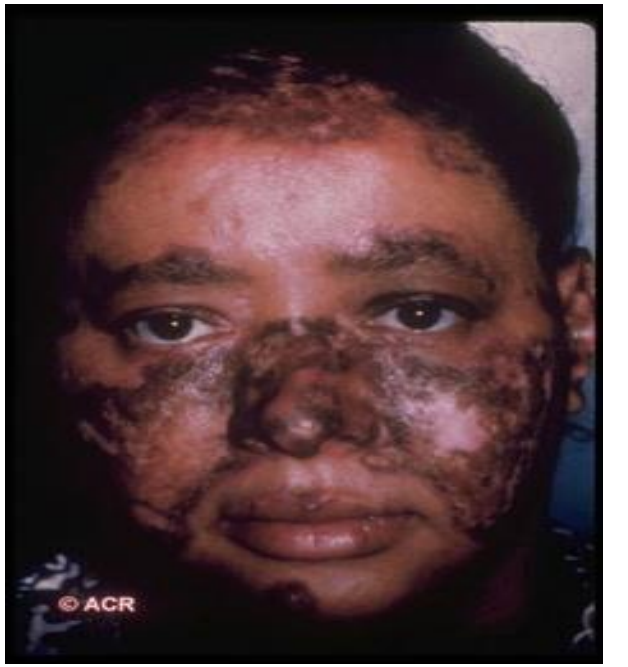


Q: *24 yr old female pt with history of extreme fatigue and arthralagia ,*
What do you see?
Photosensitivity rash over the Sun exposed areas
Differential diagnosis?
- Skin manifestation of SLE,
- Idiopathic inflammatory myopathies
How do you treat this patient according to the Hx?
-Anti-malarial drugs (hydroxychloroquine)
-NSAIDs , steroids
- Immunosuppressive drugs

In SLE , we have a special type of endocarditis we call it **Libman-Sacks endocarditis** and its a form of **nonbacterial** endocarditis that is seen in systemic lupus erythematosus. It is the most common cardiac manifestation of lupus. Libman-Sacks lesions rarely produce significant valve dysfunction and the lesions only rarely embolize. The Valvular abnormalities are often clinically silent, without significant valvular dysfunction. Valvular regurgitation is more common than stenosis, which is rare. Valvular dysfunction can result in cardiac failure. Embolic phenomena and secondary infective endocarditis are uncommon but can result in neurological and systemic complications.

Q: 1-describe what you see?

flat and circular scarred hypopegminted areas ,with raised hyperpigmented margins we call it ***discoid lupus***



2-differntial diagnosis?

skin manifestation of SLE

Pure cutaneous lupus: here the body's immune system incorrectly attacking normal skin, leading to create various kind of skin lesion, some patients go on to develop SLE

3- how to treat such patients?

1. **NSAIDs** ➔ aspirin and ibuprofen
2. **Corticosteroids** ➔ Prednisone
3. **Anti-malarial drugs** ➔ such as Plaquenil , hydroxychloroquine, are prescribed for skin and joint symptoms of lupus. It may take months before these drugs demonstrate a beneficial effect.
4. Immunosuppressive agents: **Azathioprine (*Imuran*) , cyclophosphamide (*Cytoxan*)** .
5. **Life style change** : avoidance of (excessive) sun exposure, Maintaining a healthy lifestyle – get plenty of rest, reduce stress, eat a balanced diet, and quit smoking.

Q:A 22 year old female presents with a 6 week history of fatigue and facial rash. Her rash seems to be exacerbated by sun exposure. She has recently developed pain and swelling in her fingers and wrists.. By examination , She has an erythematous maculopapular rash over her malar areas spanning the bridge of her nose , *Erythema of hard and soft palate and erythematous rash of the tongue* (as you see), Joint exam reveals mild swelling and tenderness to palpation and range of motion in the proximal interphalangeal joints of several of her fingers and both wrists.

1- Identify the condition??

SLE

2- investigations?

1. **CBC :**

a) Haemolytic anaemia b) Leukopaenia

c) Lymphopaenia d) Thrombocytopaenia

2. **Serology:**

a)Anti-DNA antibodies ,ANA b) Anti-Sm antibodies c) Anti-phospholipid antibodies

3. **urinalysis:**

a) Proteinuria (> 3+ or 0.5 g/day) b) Cellular casts in urine

4. **Elevated ESR and CRP**

3-The Hgb in this pt was 10 ,how can you explain that??

Anemia may be secondary to chronic disease (normocytic, hypochromic) or due to autoimmune hemolysis with a positive Coombs test.

Q: Suppose you have a female pt with SLE , presents with swollen ,painful leg and history of miscarriages ,

Identify the condition ?

-Secondary anti - phospholipid syndrome.

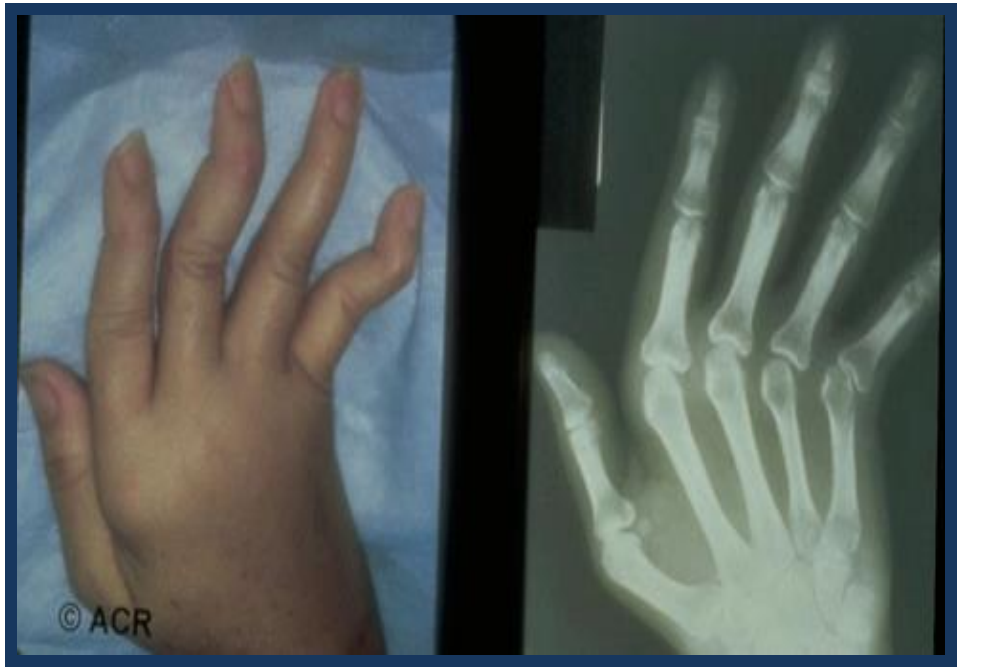
Investigations??

Lupus anticoagulant , Anti-cardioliptin antibodies ,prolonged PTT, thrombocytopenia

SLE arthropathy

-Non erosive arthritis

-Hand may show diffuse soft tissue swelling, ulnar deviation, swan neck deformity , MCP subluxation.



Diagnostic criteria of SLE. A person is said to have SLE if he/she meets any 4 of these 11 criteria simultaneously or in succession

Criterion	Definition/examples
1. Malar rash	Fixed erythema over the malar eminences, tending to spare the nasolabial folds
2. Discoid rash	Erythematosus raised patches, may scar
3. Photosensitivity	Skin rash as a result of unusual reaction to sunlight
4. Oral ulcers	Usually painless
5. Arthritis	Non-erosive: Jaccoud's arthropathy
6. Serositis ECG	a) Pleuritis – pleuritic pain, pleural rub, pleural effusion b) Pericarditis – changes rub, pericardial effusion
7. Renal disorder	a) Proteinuria (> 3+ or 0.5 g/day) b) Cellular casts in urine
8. Neurological disorder	a) Seizures b) Psychosis
9. Haematological disorder	a) Haemolytic anaemia b) Leukopaenia c) Lymphopaenia d)Thrombocytopaenia
10. Immunological disorder	a) Anti-DNA antibodies b) Anti-Sm antibodies c) Anti-phospholipid antibodies
11. Anti-nuclear antibody	Exclude drug causes

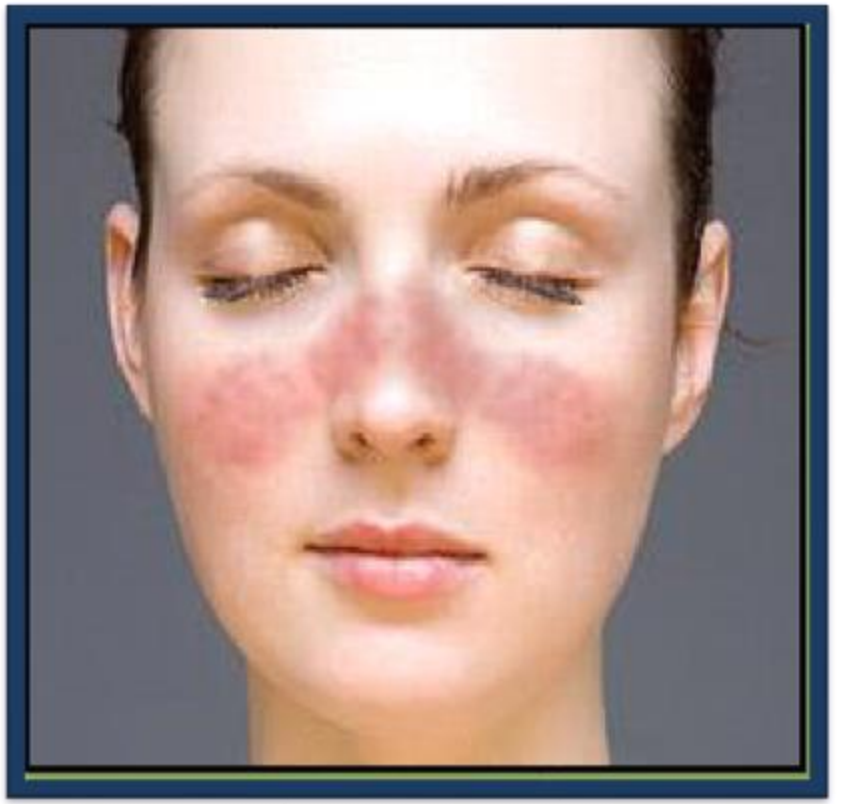
Q:24 YO female patient, presented with Hematurea & Hemoptysis, what is the diagnosis?

SLE.

Q:What is the most specific test to diagnose this disease?

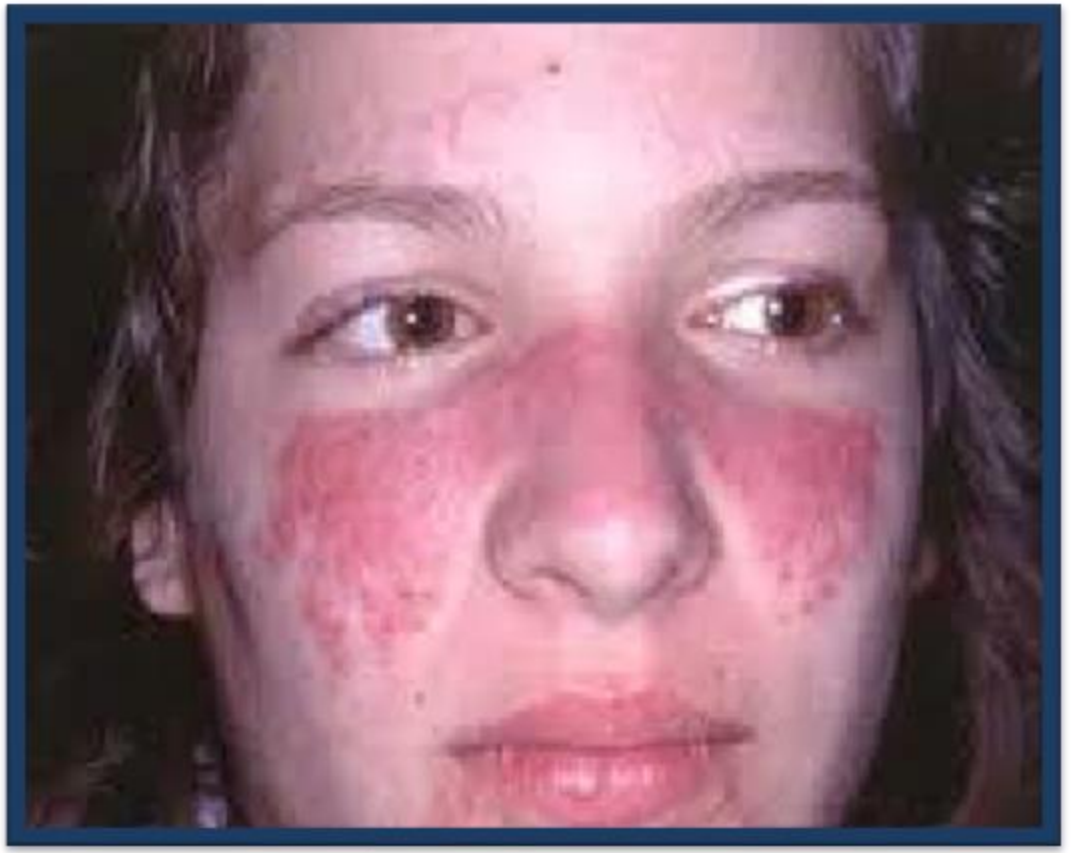
Anti- ds DNA

Anti Smith



Q: This patient had fever & joint pain. Mention a specific test for the diagnosis.

Anti ds-DNA antibodies



Q: A. What is your spot Dx?

SLE.

B. What is the cause of her respiratory problems?

Serositis/ Lung fibrosis

C. Write the name of a blood test.

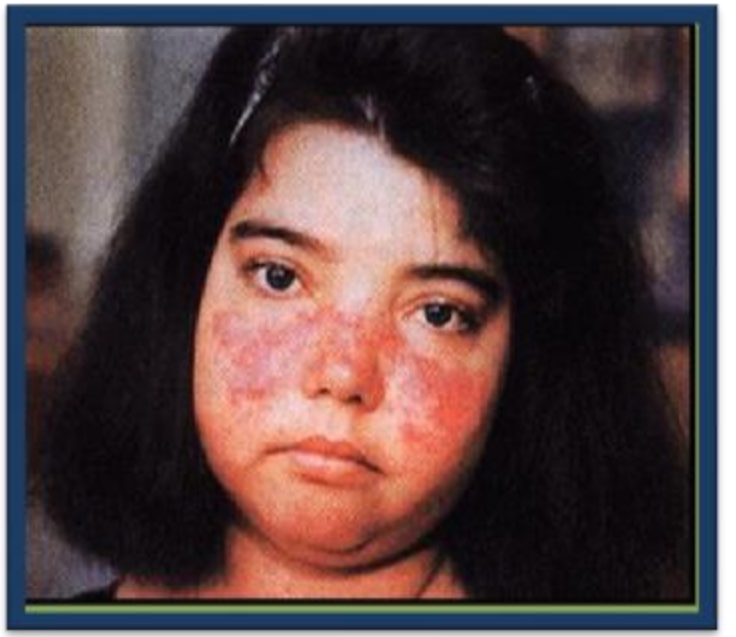
ANA, anti-dsDNA & anti-smth.

D. Mention 2 other manifestations for this disease. (Signs or Symptoms)

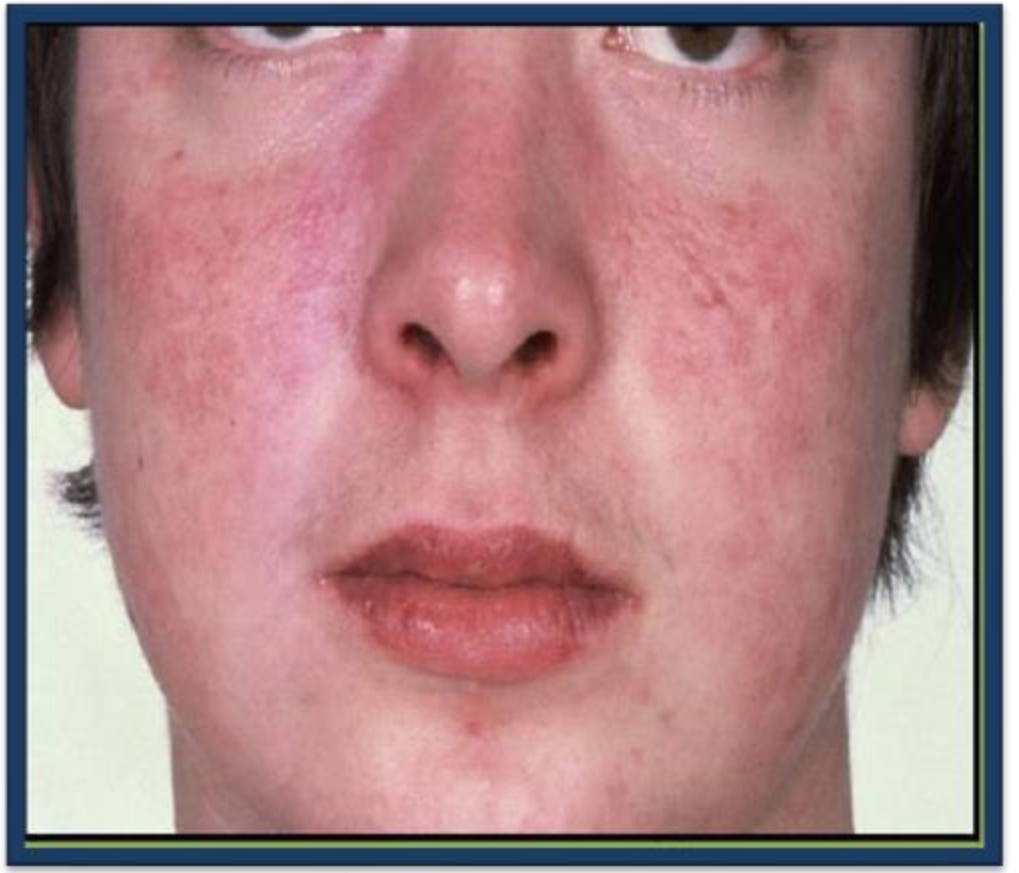
photosensitivity, discoid lupus, Neurological (psychosis, seizures), …

Q: mention two hematological manifestations ?

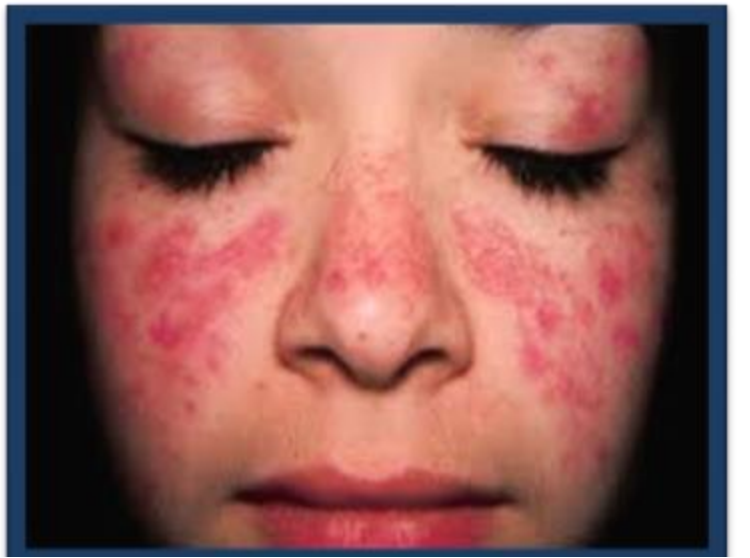
Hemolytic Anemia & thrombocytopenia



Q: This pt presented with joint pain, proteinurea, & anemia. What blood test are you going to order for her?
ANA, Anti-dsDNA, Anti-Smith.



Q: a) Diagnosis?
SLE
b) What's the most specific test? Anti ds-DNA
c)Give 3 antibodies for diagnosis.
ANA, Anti dsDNA antibody, anti smith antibody….
d) Name 2 possible diagnostic hematologic abnormalities in this patient with arthralgia.
Hemolytic anemia
Thrombocytopenia
Leukopenia



Q: Female pt presents with chest pain and this rash.
Give to physical findings

discoid rash - photosensitivity – malar rash

cause of chest pain?

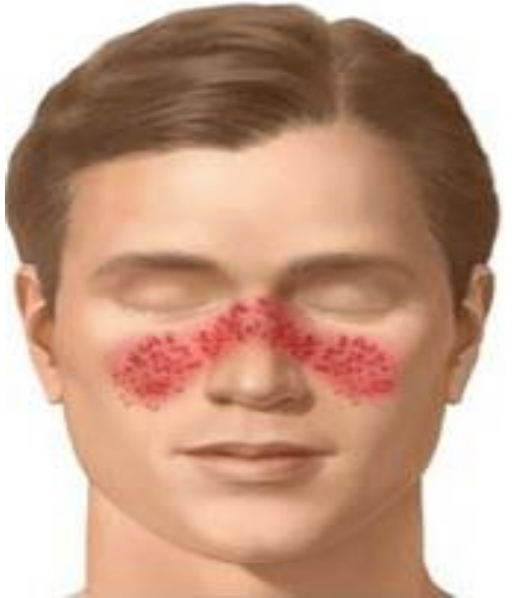
Serositis

What is your finding?

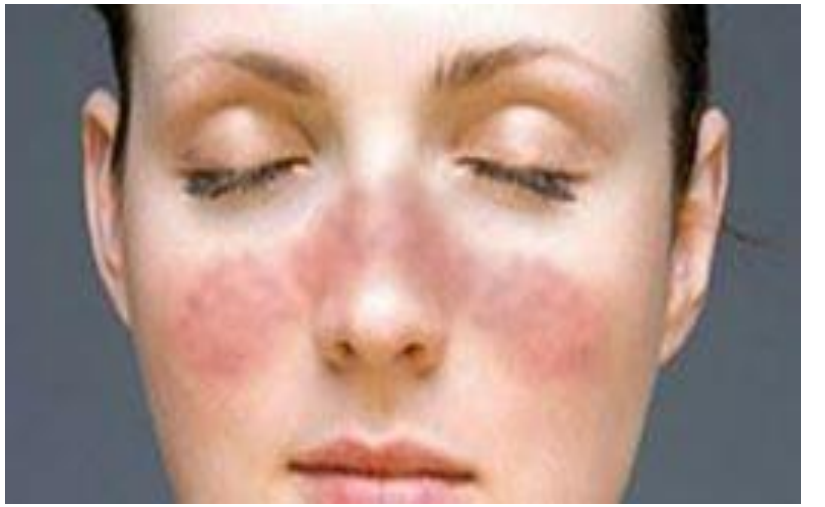
Malar rash

Name one disease associated with it

SLE



Patient with murmur of mitral stenosis malar rash

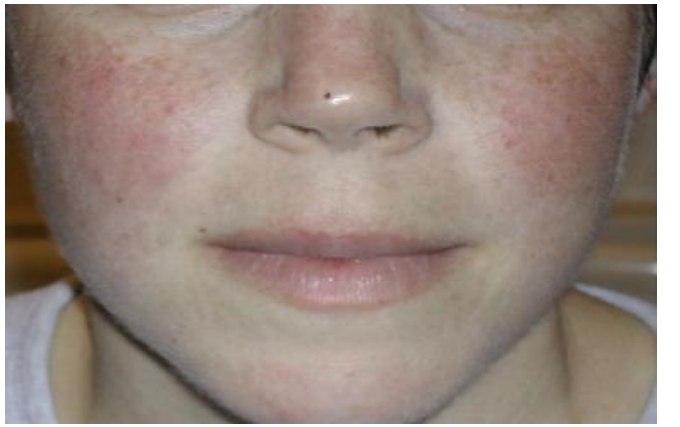


Q:Name the following sign?

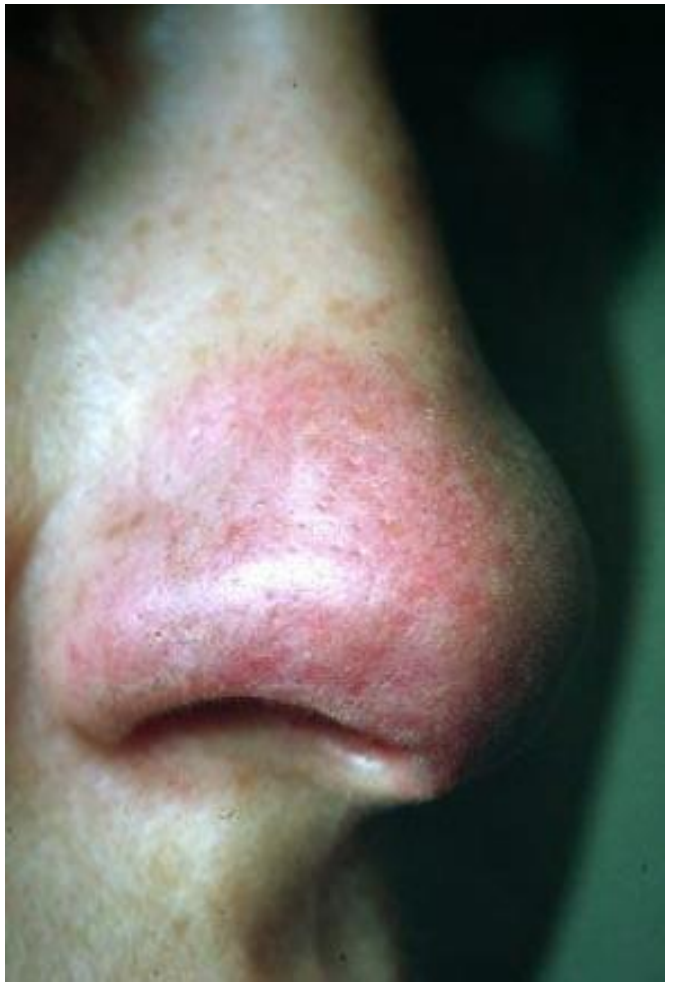
Malar (butterfly) rash

Your Dx?

SLE



Q: Diagnosis?
SLE (lesion is lupus pernio)



Q: History and lab tests suggesting SLE with elevated KFTs

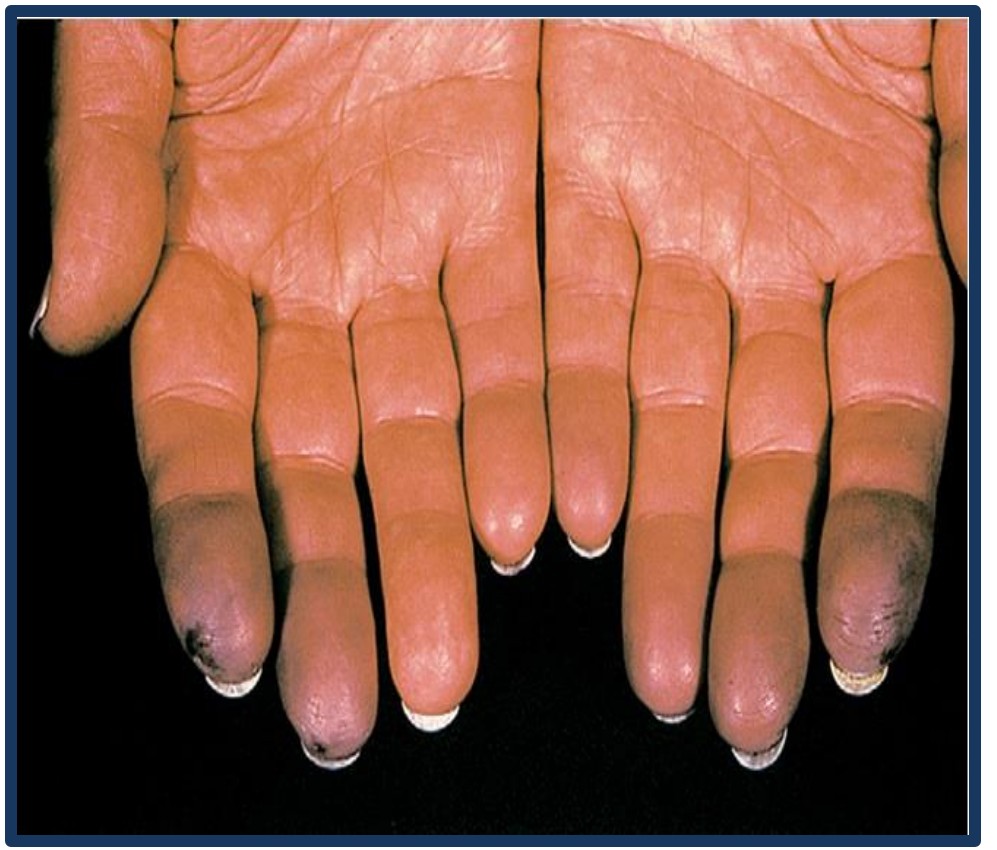
1- Diagnosis?
SLE / SLE nephropathy

2- 2 lab test to confirm the diagnosis?
Anti-dsDNA antibodies / Anti-smith antibodies

3- 2 lab tests for follow up
KFT? Serum complement level? ESR? CRP?

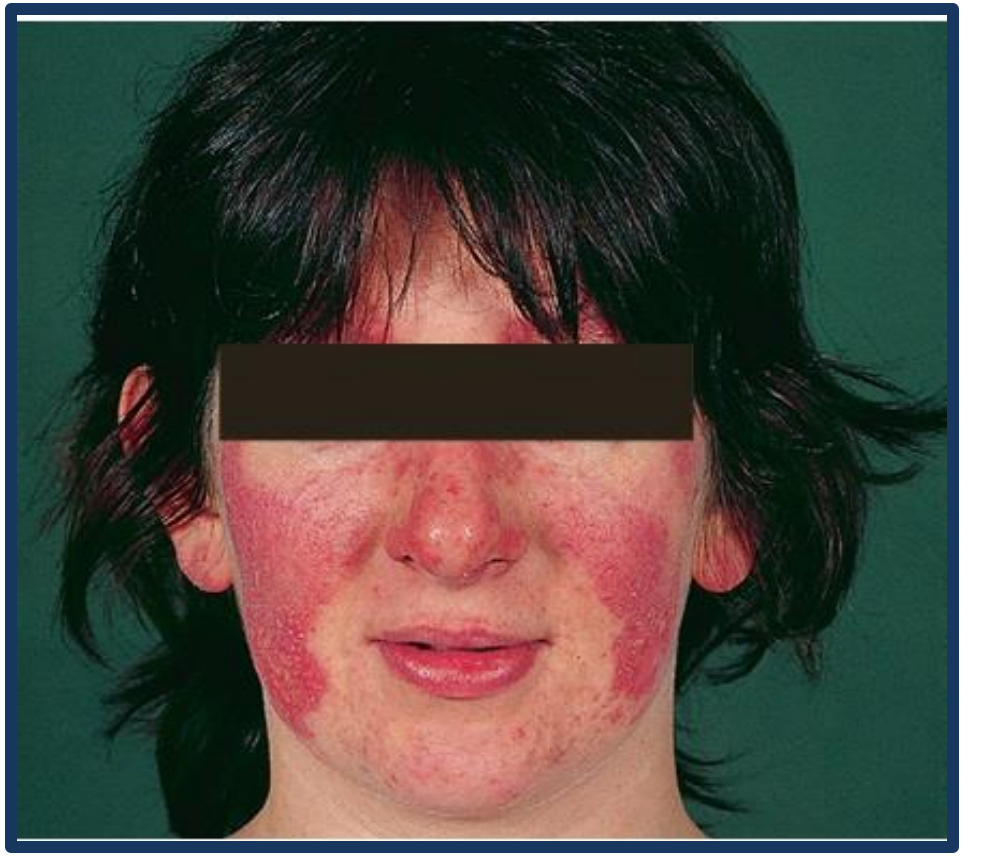
DX: SLE

F: Secondary raynaud's phenomenon leading to digital ulceration



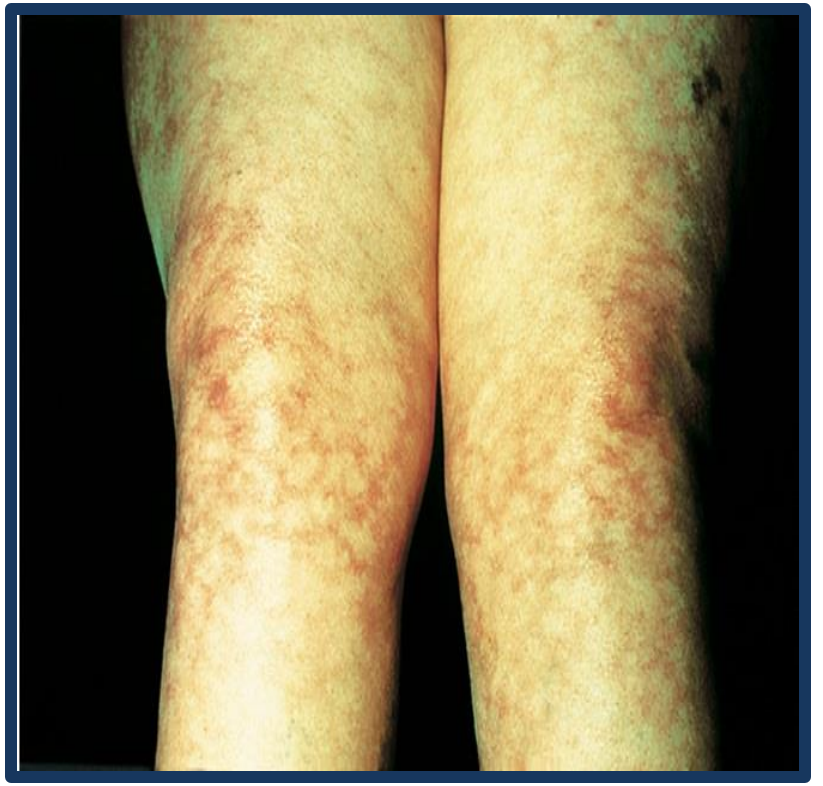
DX: SLE

F: butterfly (malar) rash with sparing of nasolabial folds.



DX: SLE/ Behcet Syndrome/Systemic Cholesterol embolism/ Amantadine drug side effect

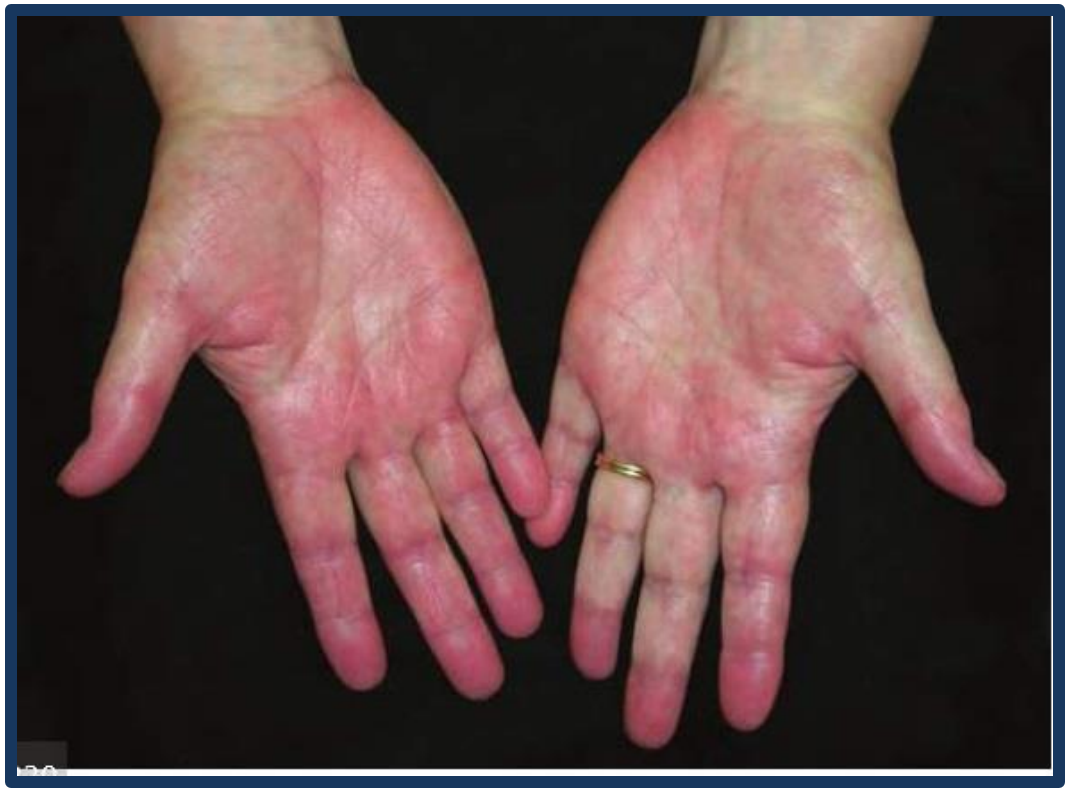
F: Livedo reticularis



DX:

SLE/RA/Thyrotoxicosis/pregnancy /familial

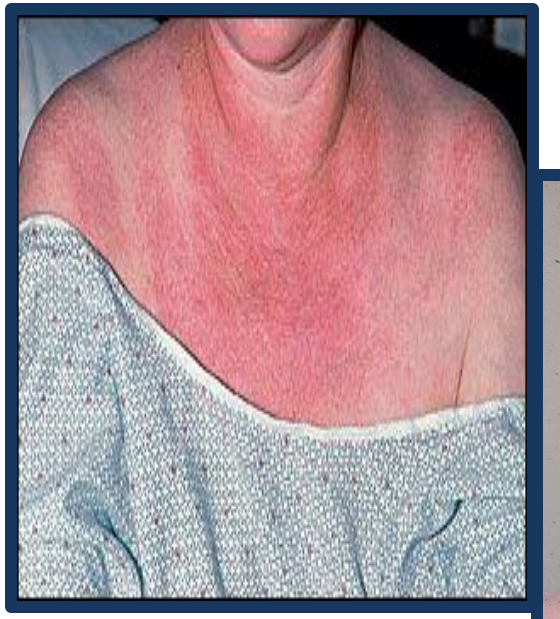
F: palmar erythema.



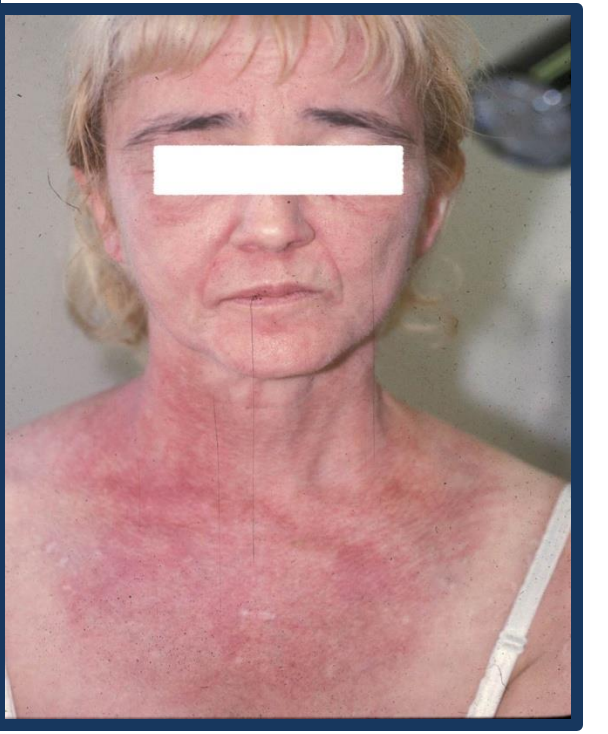
DX: SLE

F: Subacute cutaneous lupus in sun-exposed areas of the face and neck..

I think the previous picture can also be seen in Dermatomyositis as in "shawl and face" sign



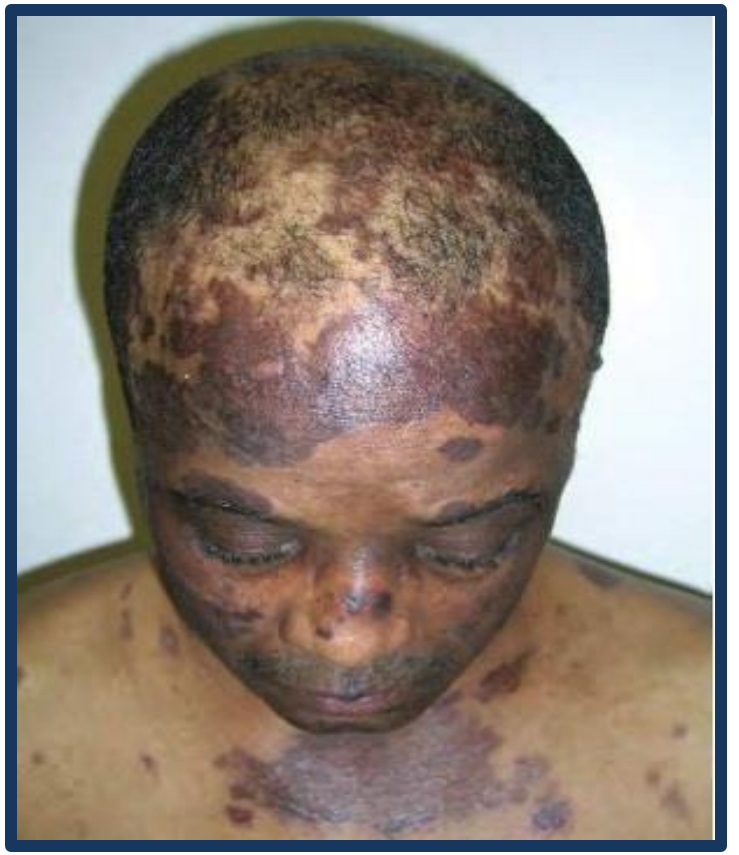
"shawl" sign



"Shawl and face" sign

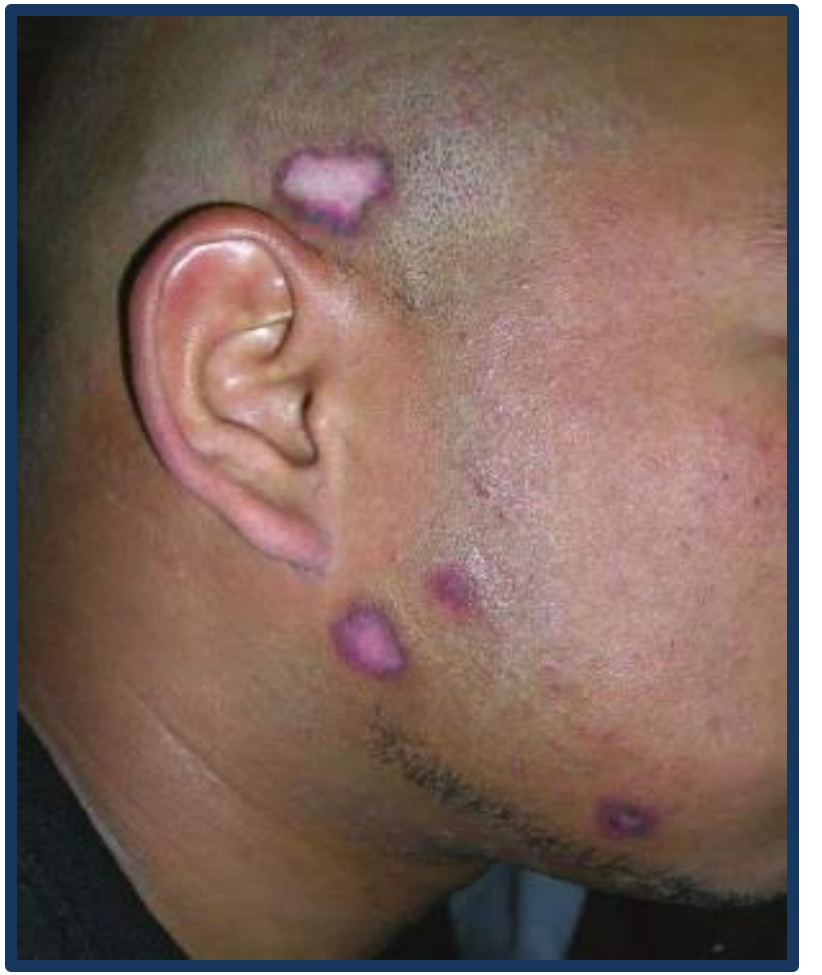
DX: SLE

F: Severe chronic cutaneous lupus with hyperpigmentation, hypopigmentation, and scarring alopecia. Sun-exposed areas of the face and neck are heavily involved.



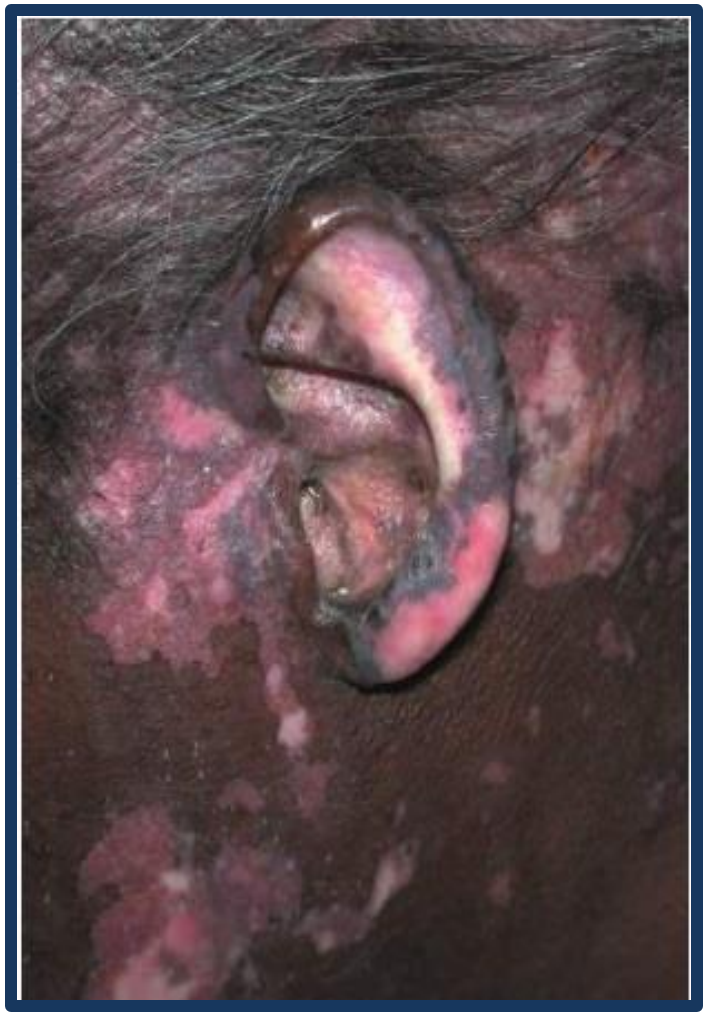
DX: SLE

F: discoid lupus with central hypopigmentation and peripheral hyperpigmentation



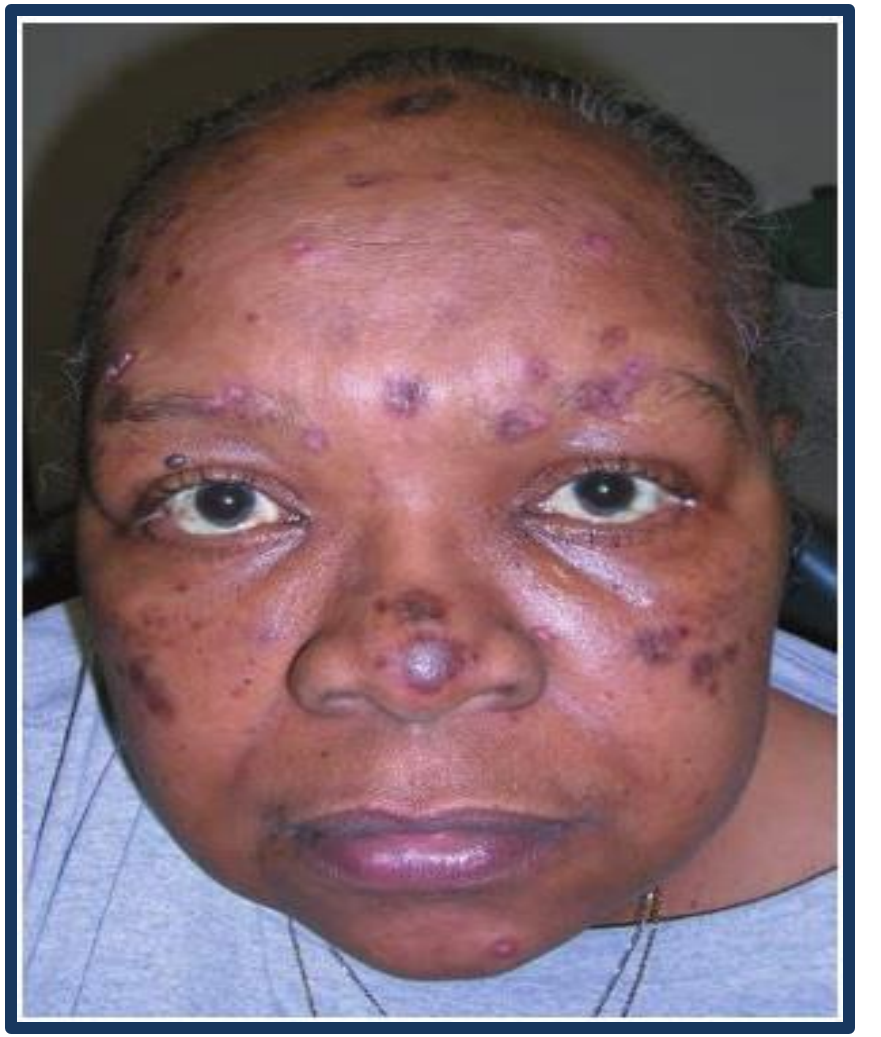
DX: SLE

F: Discoid lupus with hypopigmentation and scarring of the pinna



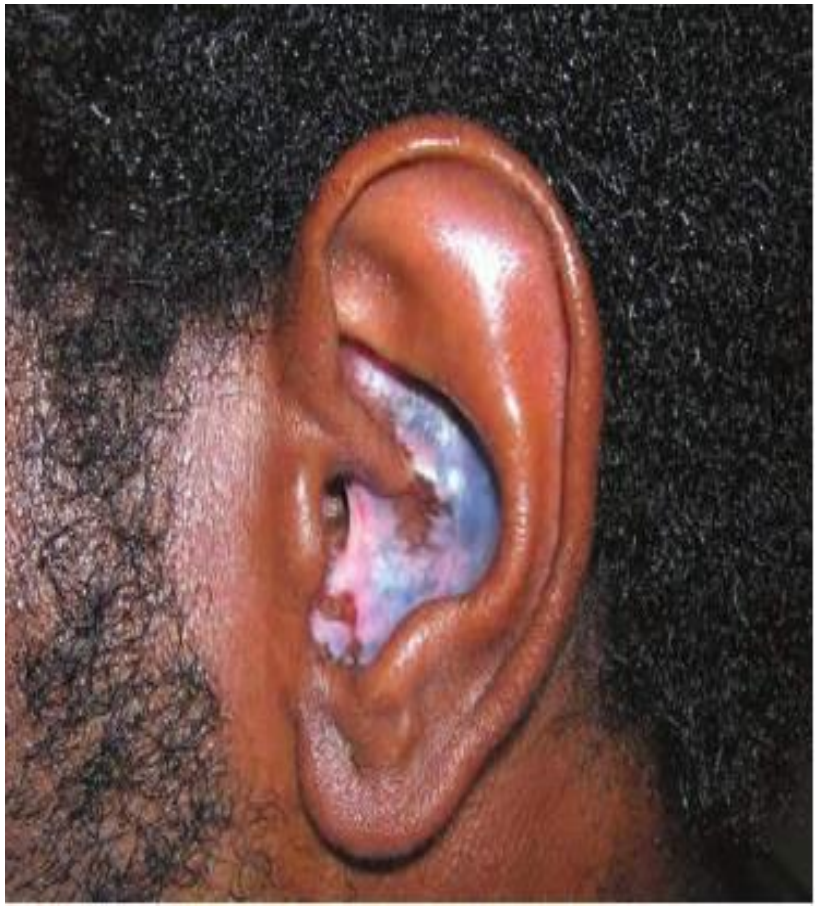
DX: SLE

F: Discoid lupus on the face and scalp with hyperpigmented lesions that are indurated and atrophic, there is also scarring alopecia



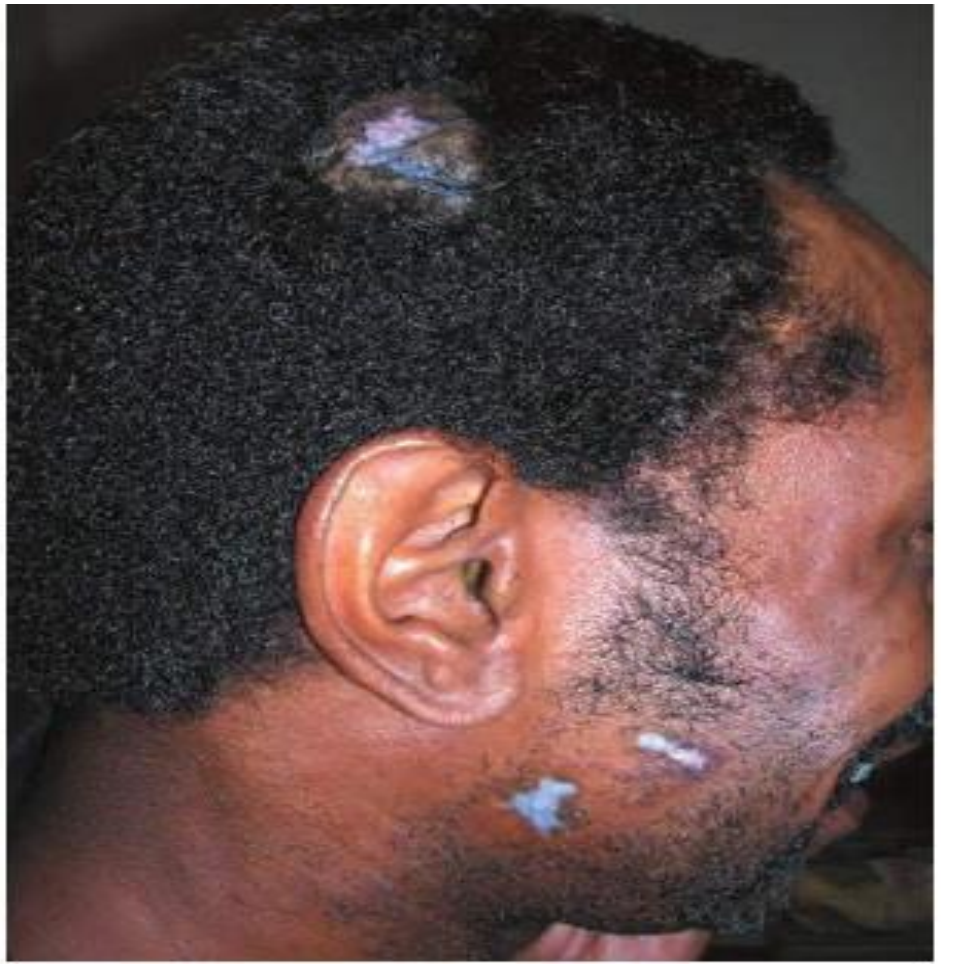
DX: SLE

F: Discoid lupus with hypopigmentation and scarring inside the pinna



DX: SLE

F: Discoid lupus with scarring alopecia and hypo pigmentation on the scalp and face

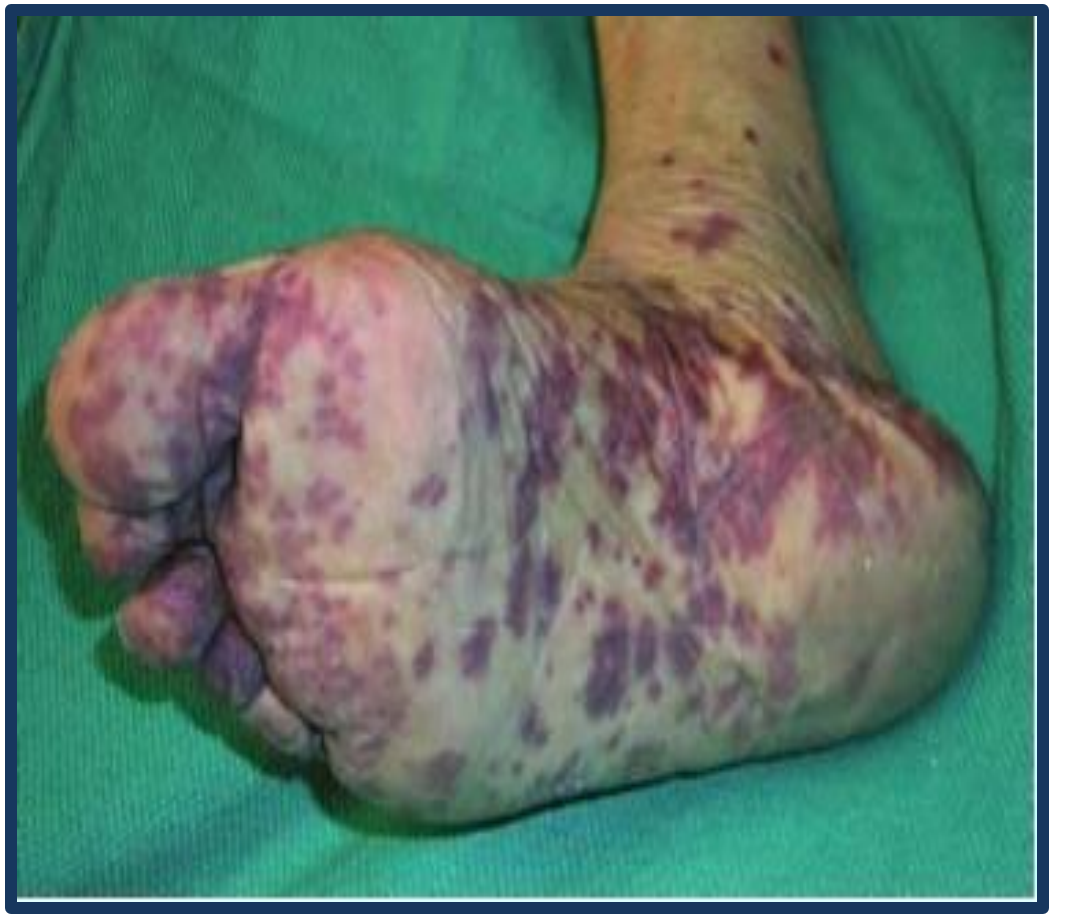


DX: SLE

F: Erythema, swelling, and hyperpigmentation on the cheeks and lips

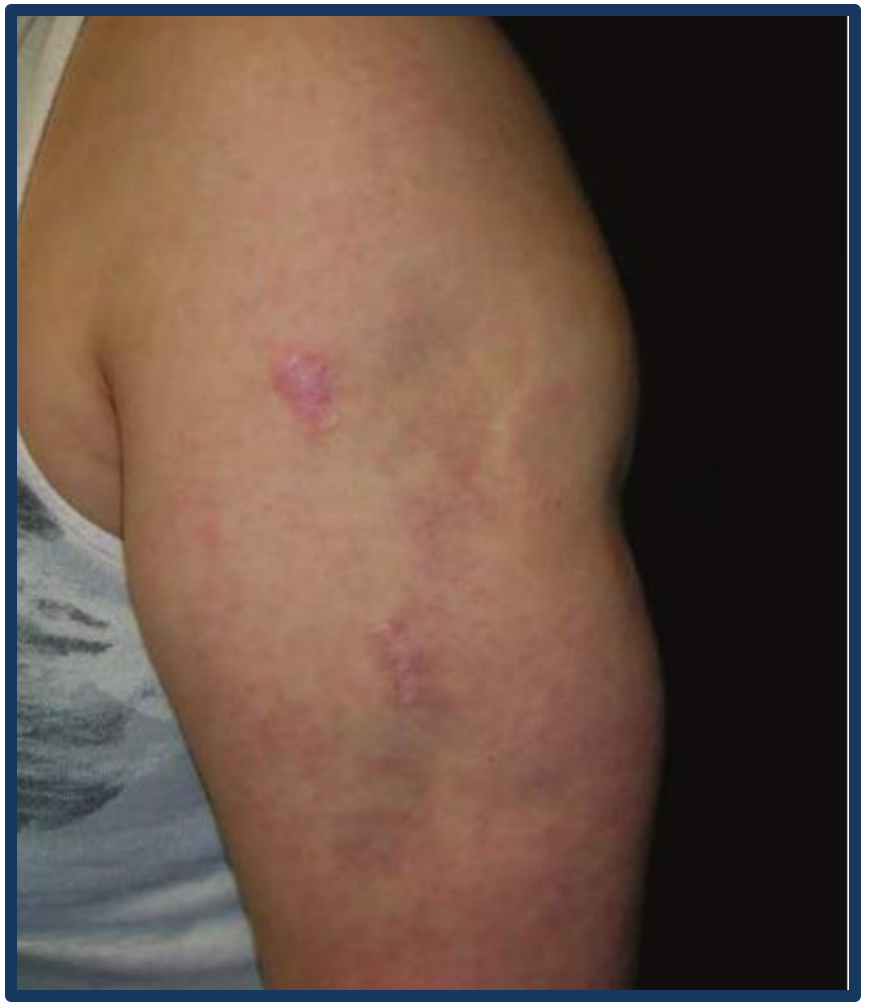
DX: SLE

F: Leukocytoclastic vasculitis on the foot.



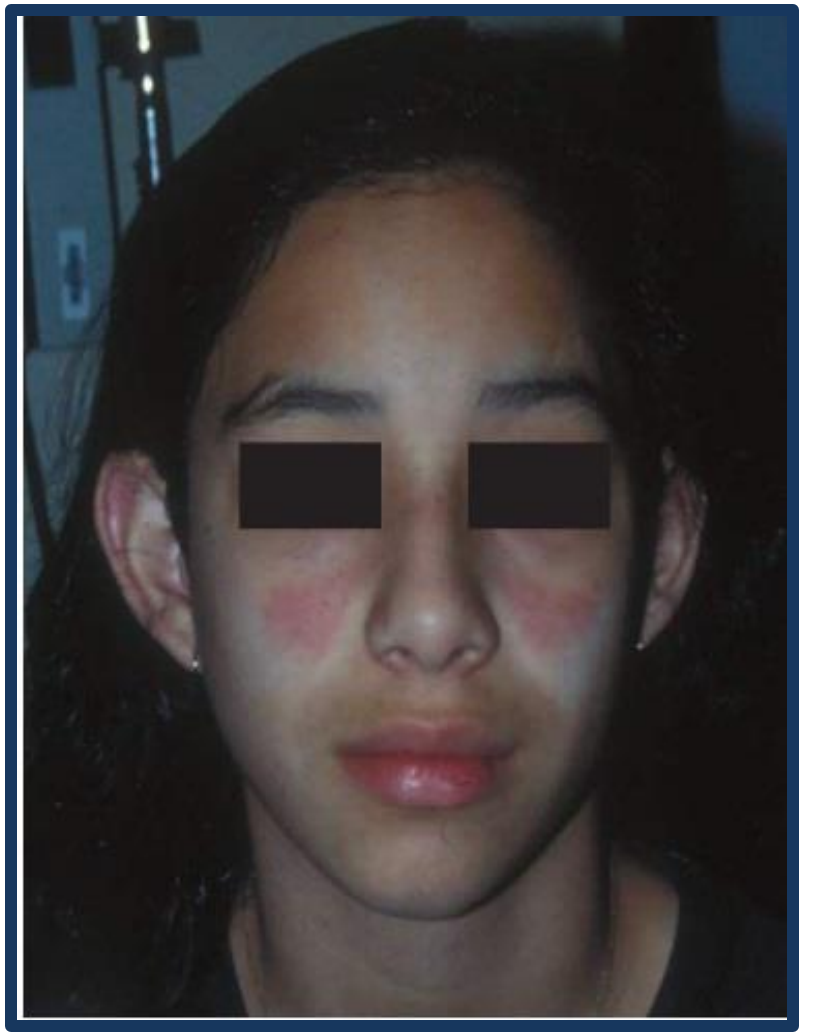
DX: SLE

F: Lupus profundus showing localized atrophic changes of the arm secondary to the panniculitis.



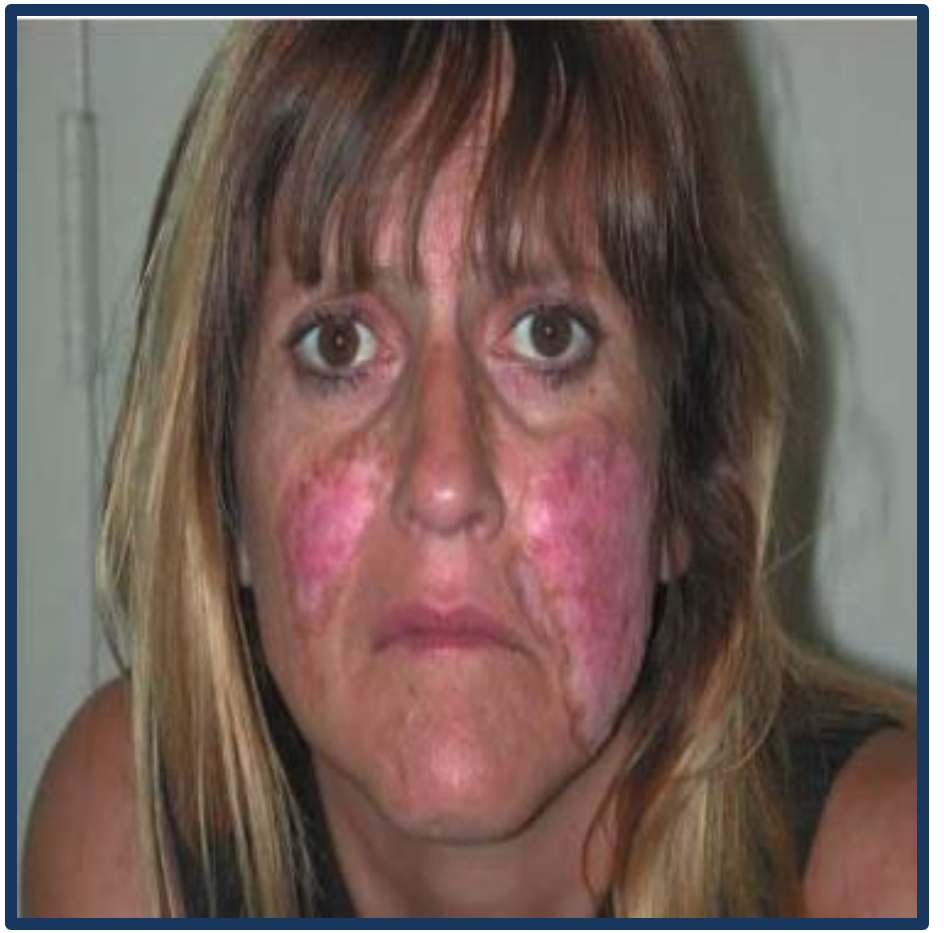
DX: SLE

F: Malar rash with relative sparing of the nasolabial fold



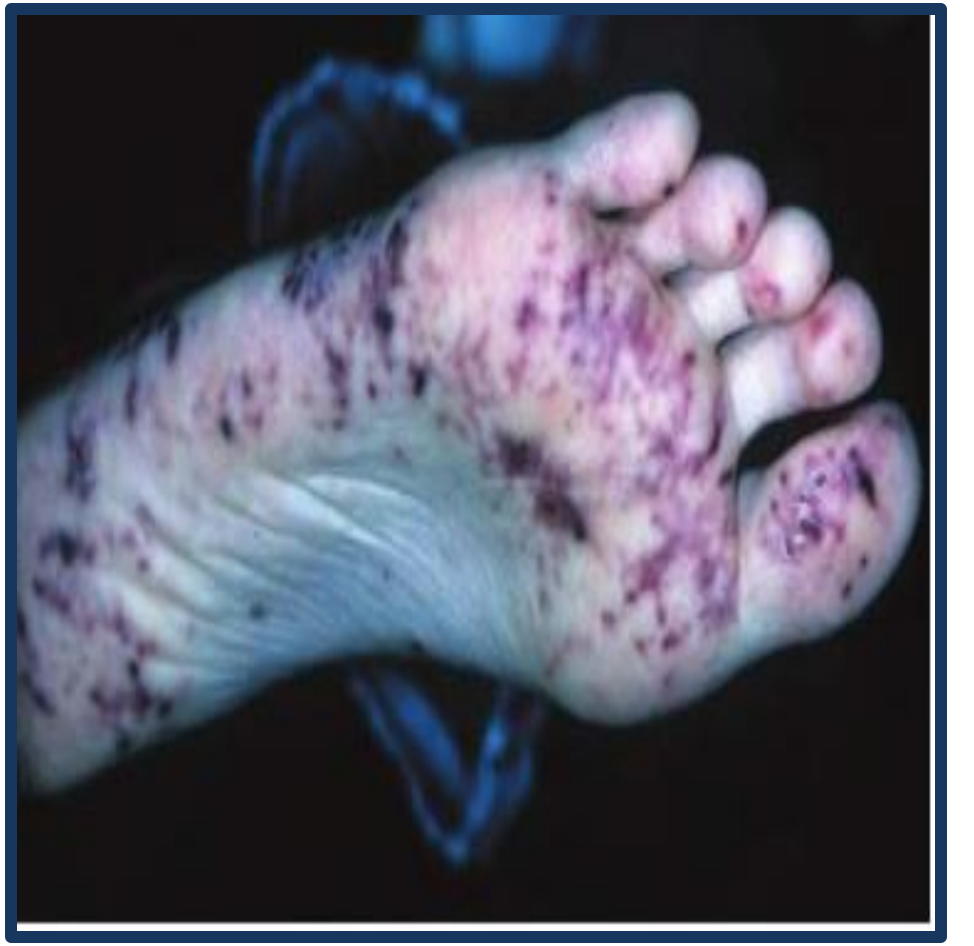
DX: SLE

F: Malar rash with severe atrophy, scarring, and hypopigmentation. The facial lesions are more typical of discoid lupus

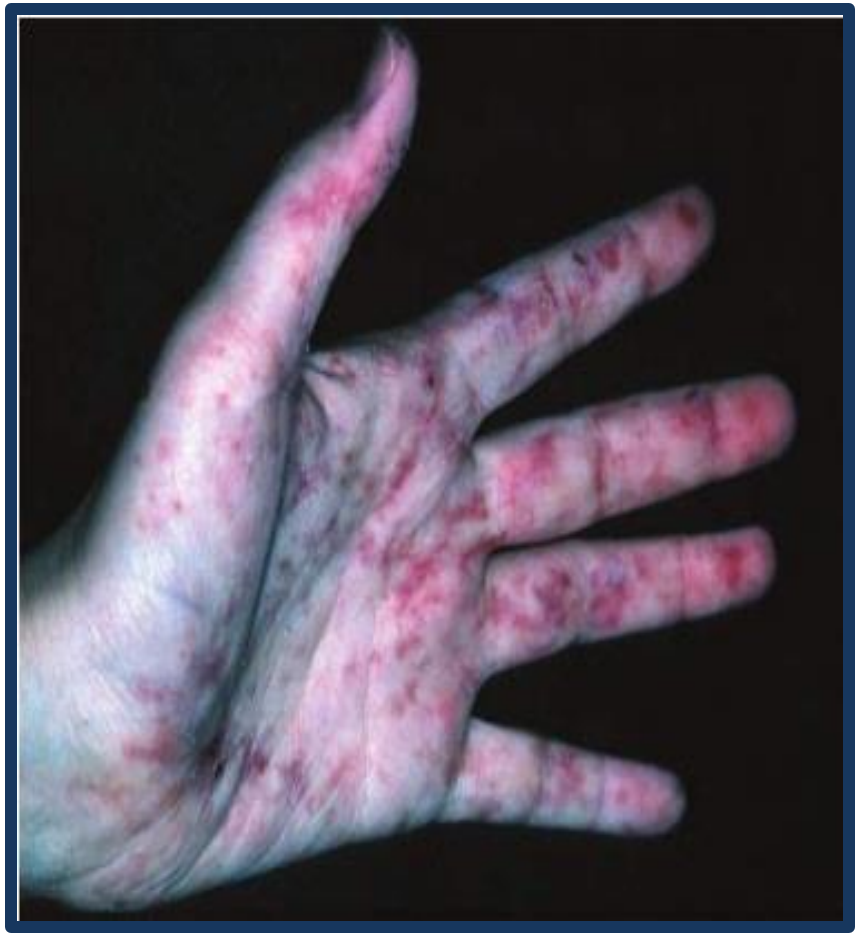


DX: SLE

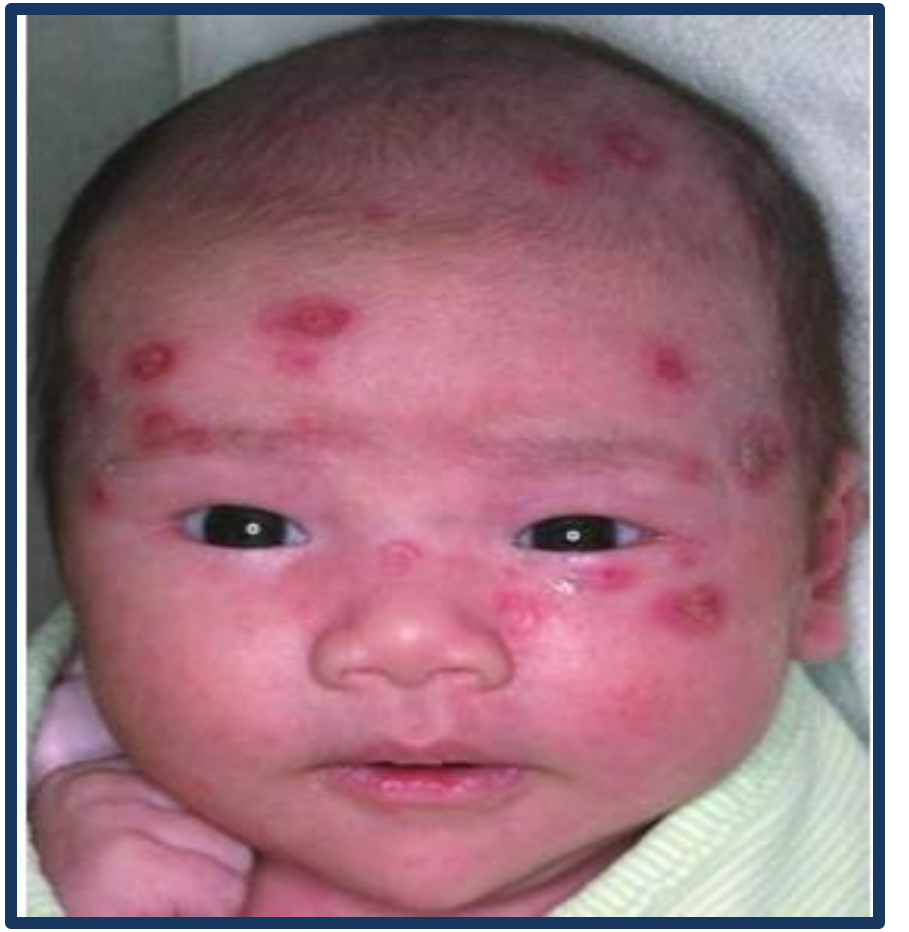
F: Necrotizing angiitis . Palpable purpura was evident on both feet and hands



DX: SLE
F: Necrotizing angiitis.

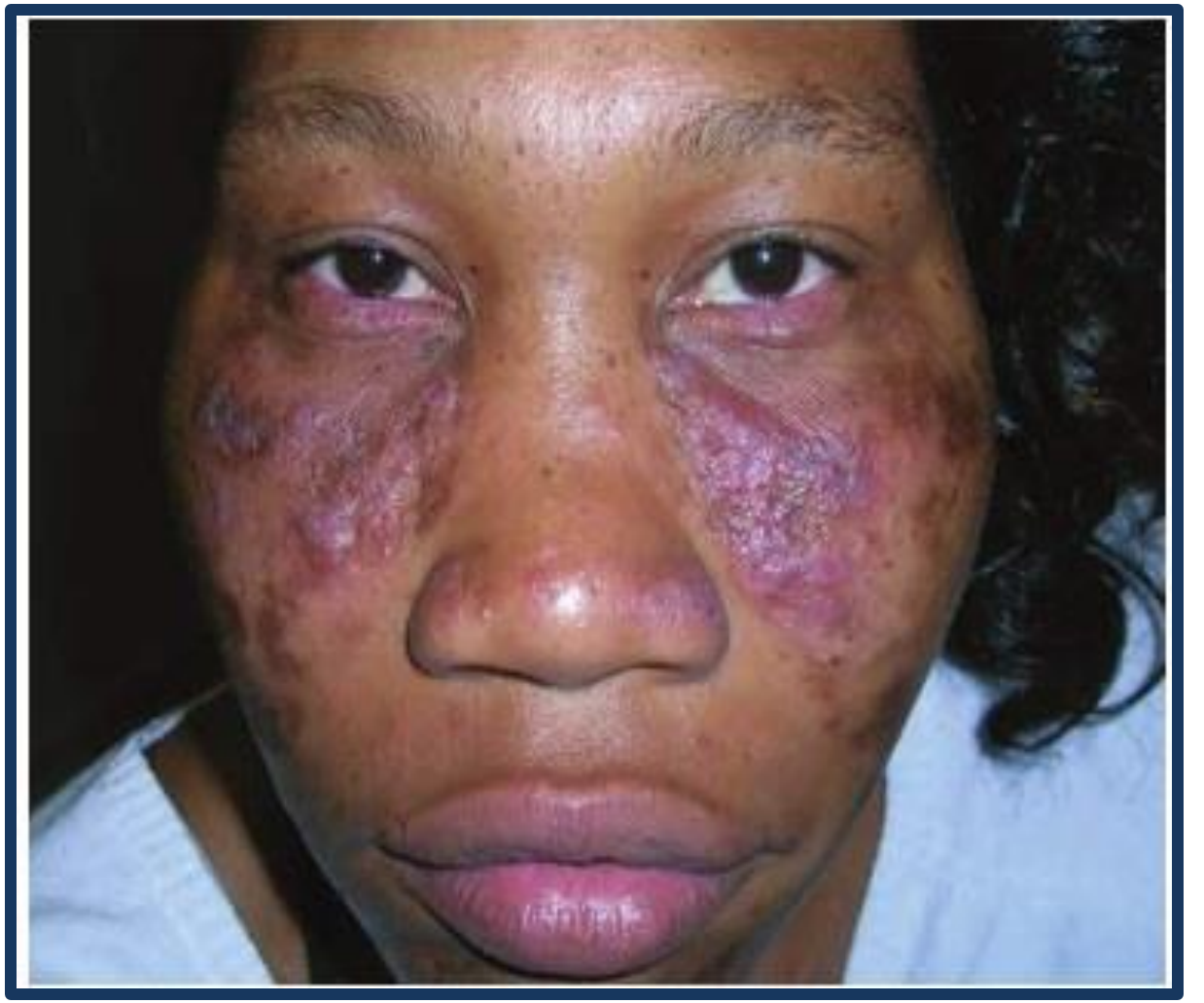


DX: SLE
F: Neonatal lupus from acquired antibodies through transplacental transmission from the mother with active SLE



DX: SLE

F: Severe discoid lupus in a malar distribution on the face. Note this chronic cutaneous lupus has caused permanent scarring



3. Scleroderma

Q: 40 yr old female patient, presented with marked induration of both hands and arms and associated with limitation of movement and , moderate induration of her face chest and legs ,she denied GI and RS symptoms.

1 - Identify the condition

Reynaud's phenomena, according to history its one clinical feature of scleroderma.

2 - Differential diagnosis

- CTDs (scleroderma /systemic sclerosis ,SLE,CREST syndrome ,RA),vibration tools
- occlusive arterial disease , Repetitive vascular injury , Polycythemia , Cryoglobulinemia

3 - Clinical Presentation

-Reynaud's phenomena

a) Pallor phase: the skin turns white becomes cold and numb

b) Cyanotic phase: it turns blue but remains cold and num

c) Hyperemic phase: it turns red and becomes hot and painful

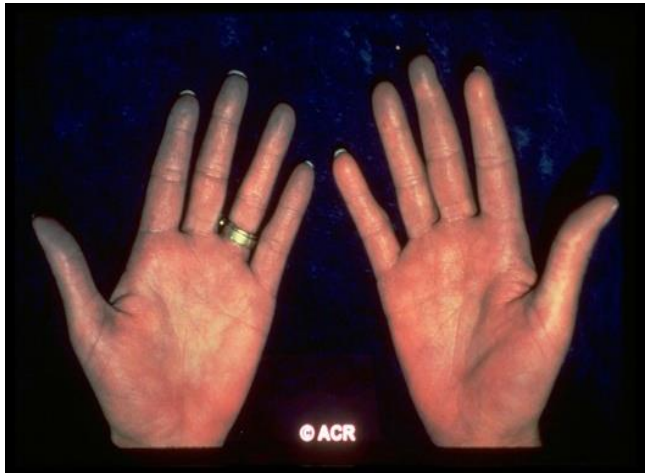
-Typical skin changes

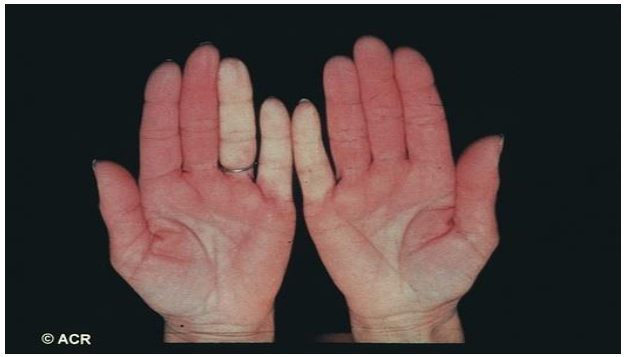
-Esophageal and small bowel dysfunction

-Interstitial lung disease

-Pulmonary hypertension

-Renal crises





4 – Investigations

according to history

- ANA positive 90%
-Anti topoisomerase 1 antibody positive (scl-70) 30% in diffuse scleroderma (systemic sclerosis)
-Anticentromere antibody positive in CREST and localized scleroderma

5 - Treatment or Management

Management is symptomatic,

Skin: no effective treatment, 60% improve with time, Calcium channel blockers may help Reynaud's phenomena .

ACE is the drug of choice to treat hypertension and to prevent further kidney damage.

Q: 4 month later, she started complaining of GI symptoms ,SOB and dry cough

Describe what you see?

-tight thick skin

-pursed mouth

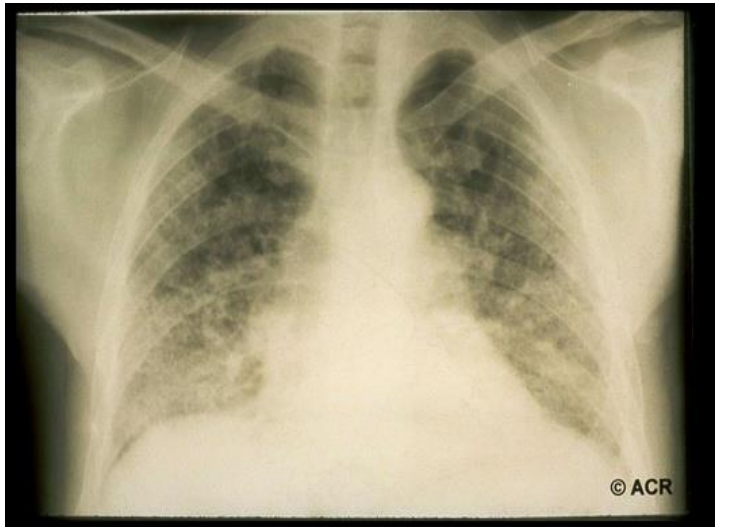
-peaked nose

What can you find mainly on PFTs, why?

-↓ FVC, ↓ TLC, ↓ DL_{CO}

-due to interstitial lung disease (look at the X-ray)

-and the pt may have pulmonary hypertension, so the DL_{CO} will decrease .



What are the GI manifestations that associated with SD?

- Scleroderma can decrease motility anywhere in the gastrointestinal tract, leading to:
- heart burn
- reflux symptoms
- Dysphasia
- Bacterial overgrowth, mal absorption
- pseudo obstruction

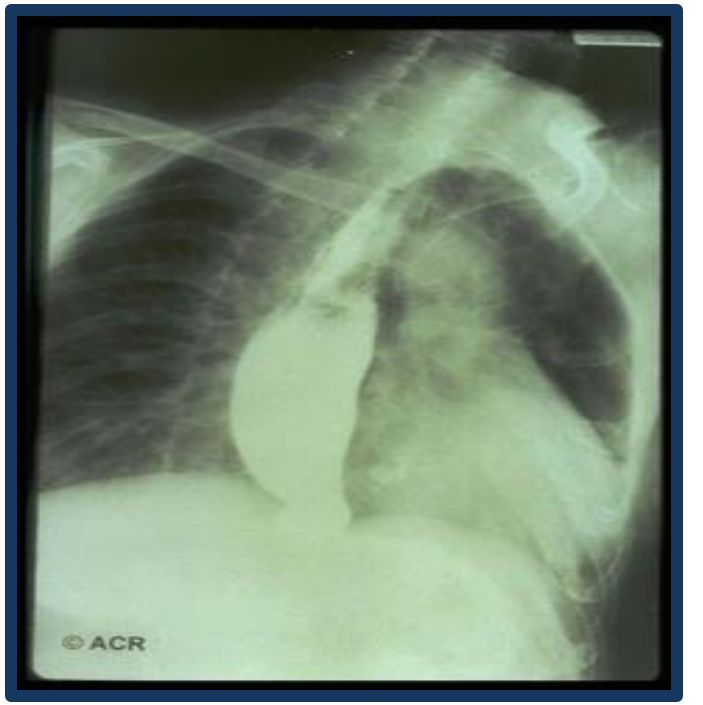
What do you see?

- Dilated lower part of esophagus

What is the name of the investigation?

- esophagram (barium swallow): A series of x-rays of the Esophagus.
- The x-ray pictures are taken after the patient drinks

a solution that coats and outlines the walls of the esophagus



Q: 25-year-old female presented with history of Raynaud's phenomenon over fingers and toes since 3 years, recurrent painful ulcers over fingers and toes since $2\frac{1}{2}$ years, tightening of the skin since 2 years, postprandial odynophagia to liquids since one year. There was no history of palpitations, dyspnea, and syncope, cough or pain chest. The course was progressive and unremitting.

Describe what you see?

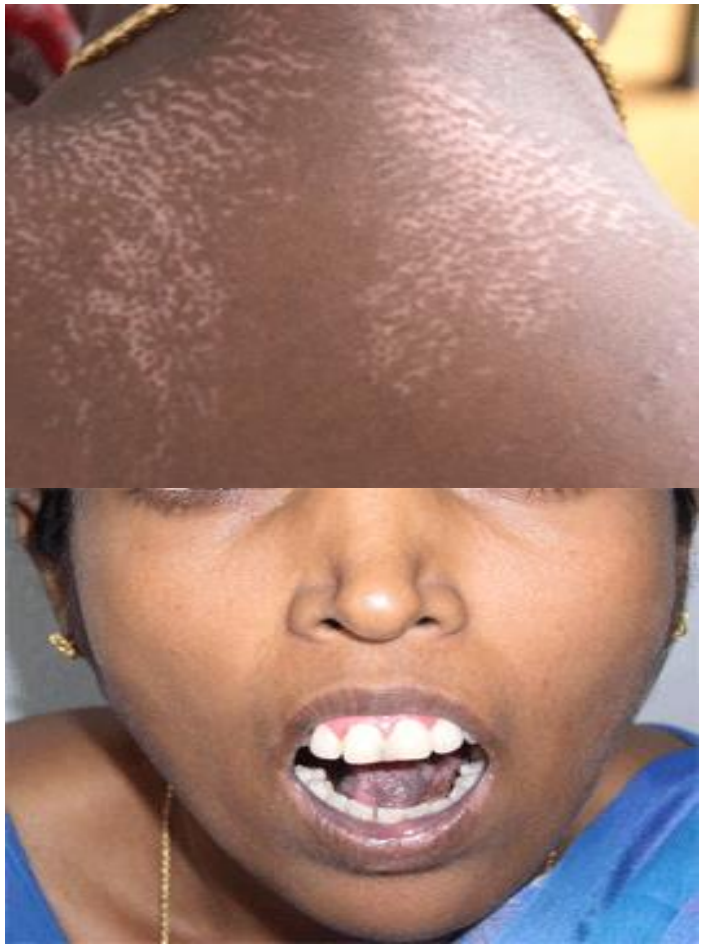
a) Salt and pepper like pigmentation: areas of Hyper-pigmentation alternating with hypo-pigmentation

b) Microstomia: the condition of having an abnormally small mouth.

What is your differential diagnosis?

Diffuse scleroderma

Localized scleroderma



What do you expect to find on examination?

-normal vital signs

- Mask like face with a pinched nose, microstomia.

-shinny, waxy and inelastic skin of face and scalp, and hands

- There was also generalized hyperpigmentation of the skin.

- Acrosclerosis with semi flexed fingers, loss of finger tip

Pulp, multiple stellate scars and small ulcers over the fingertips , cyanosed fingers and toes.

- Salt and pepper pigmentation.

-RS: maybe normal , sign of interstitial lung disease

- CVS: Normal or you can find sign of right sided heart failure due to pulmonary hypertension.

Diagnostic Testing for Scleroderma?

-Skin biopsy to look for collagen fibers (connective tissue) in the skin layers especially in systemic scleroderma

-Antinuclear antibodies (ANA) - over 90% of people with systemic scleroderma show elevated ANA in the blood. This antibody is a marker of autoimmune disease. If the ANA test is positive, further antibody testing can be conducted to determine the type of SSc. These include:

-Anti-centromere antibodies - commonly seen with CREST and limited scleroderma.

-Anti-topoisomerase antibodies - commonly seen in diffuse scleroderma.

-CBC (complete blood count).

-ESR - this is rarely elevated unless the scleroderma is diffuse.

-CRP may be elevated

-Urinalysis - to evaluate the presence of hematuria (blood cells in the urine) or proteinuria (elevated levels of proteins in the urine) this would indicate kidney involvement.

-PFTs

- Upper GI endscopy , barium swallow

-ECG and Exercise Stress Test (cardiac arrhythmias, conduction defects of the heart)

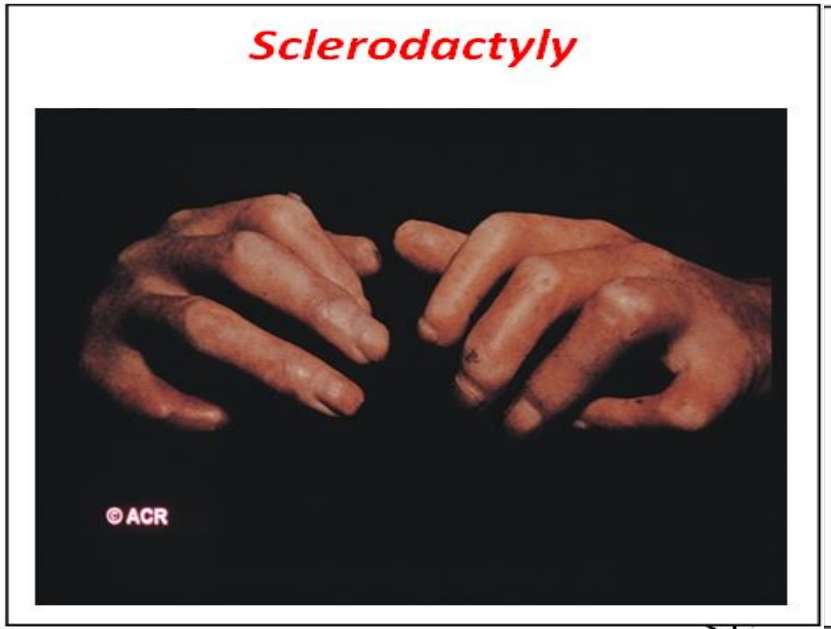
-Echocardiogram (pericardial involvement)

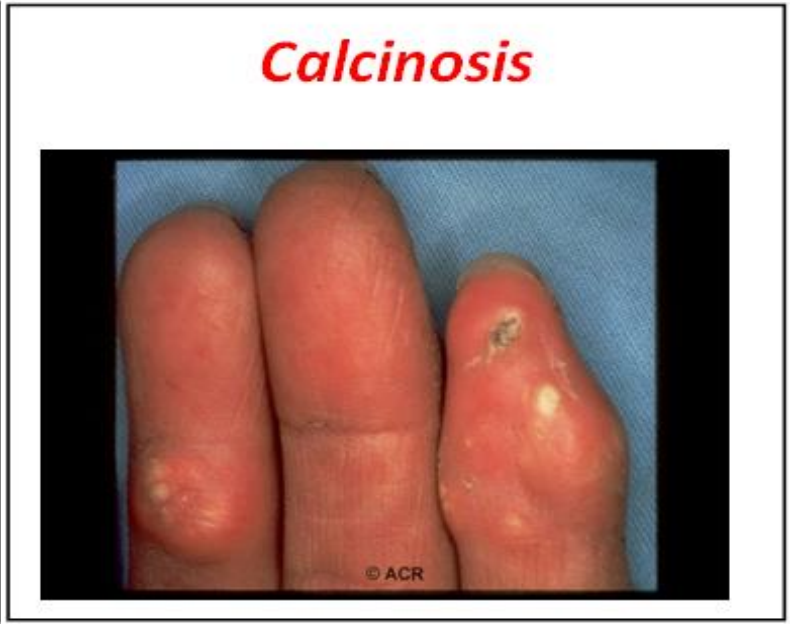
-cardiac enzymes (myocardial involvement)

REM: Investigations as appropriate, (depending on the systems involved

CREST syndrome

- Calcinosis
- Raynaud's phenomenon
- Esophageal motility dysfunction (dysphagia)
- Sclerodactyly (acrosclerosis)
- Telangiectasia





1 - Calcinosis - deposits of calcium crystals under the skin
Around the joints and organs. Skin ulcers may form over these areas.

2 - Raynaud's phenomenon - numbness, pain or color changes in the extremities brought on by cold temperatures or emotional stress. This is caused by changes in the small arteries and capillaries resulting in constriction and a temporary disruption of circulation, usually in the extremities (fingers, toes, nose and ears). This is often the first symptom of systemic scleroderma.

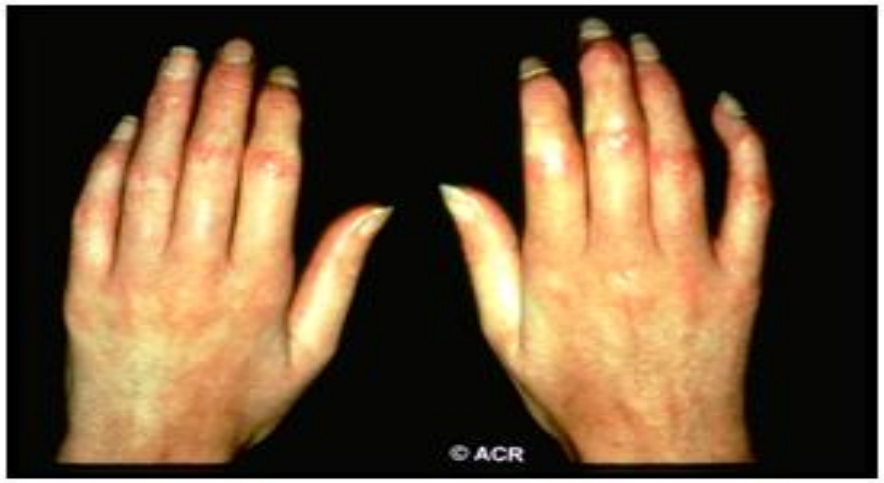
3 - Sclerodactyly (acrosclerosis) - stiffness and tightening of the Skin of the fingers. Bone loss may also occur in the fingers and toes. This symptom is usually found distal to the elbows and knees and may or may not involve the face.

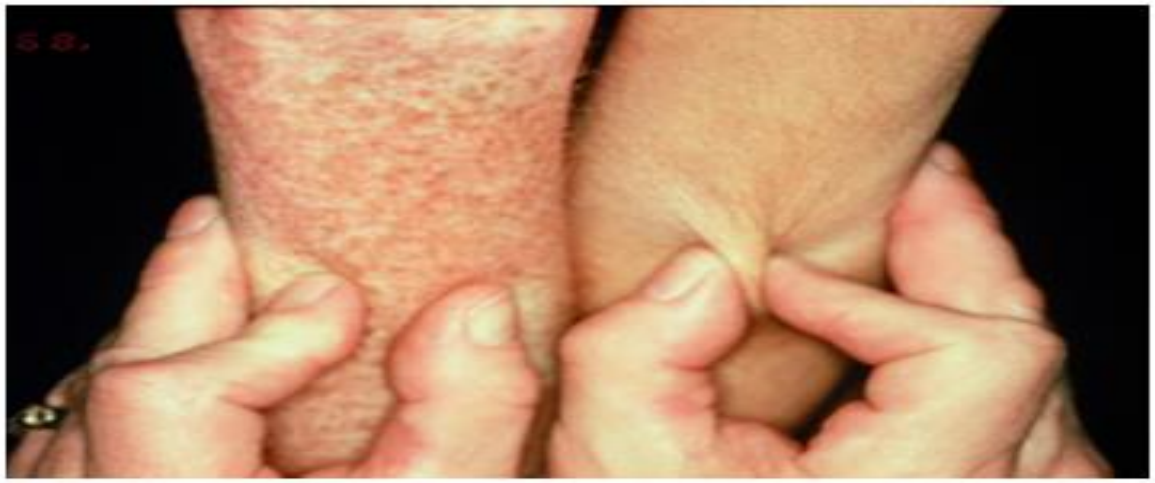
4 - Esophageal motility dysfunction (dysphagia) - muscles in the esophagus are unable to contract normally due to scarring. This can cause heartburn or a sensation of food being stuck in the throat or chest. It is estimated that up to 90% of patients with systemic scleroderma have esophageal involvement

5 - Telangiectasia - dilation of the small vessels and capillaries near the skin surface causing flat red marks on the palms of the hands, face, and tongue.

Thick skin of fingers, hand and forearms

(Proximal scleroderma)





Thick skin of torso and face

(Proximal scleroderma)

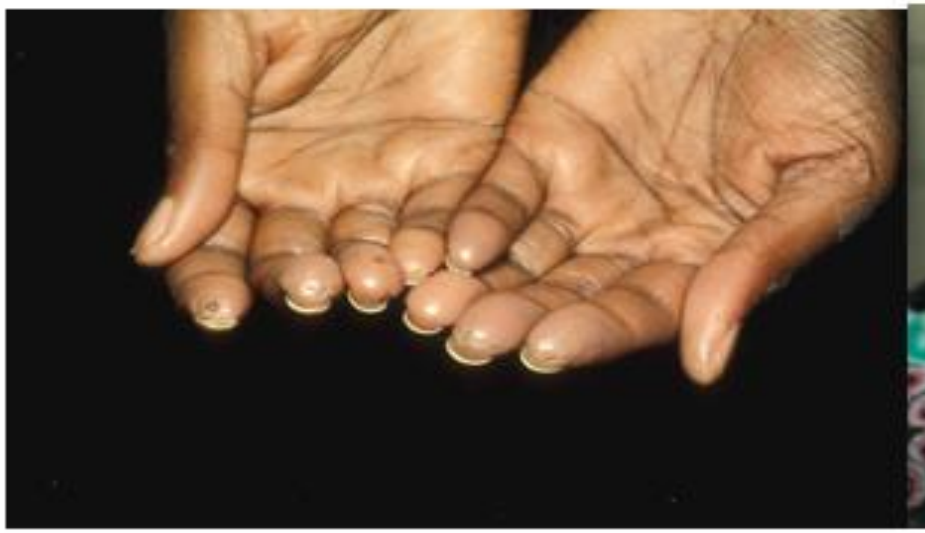
The American College of Rheumatology classification of scleroderma requires one major or two minor criteria for the diagnosis.

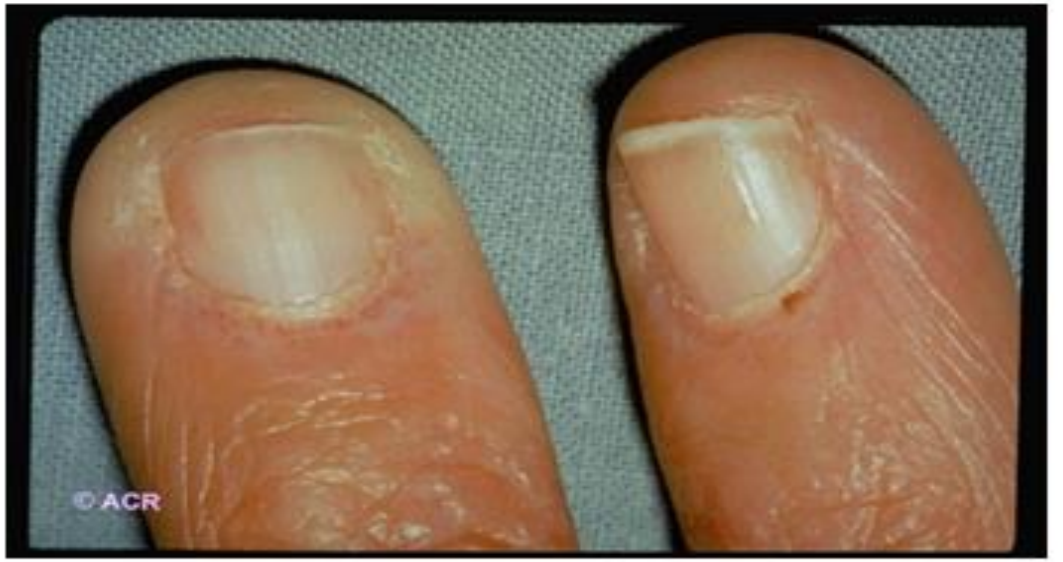
- **Major Critera -** ***Proximal scleroderma*** where the skin proximal to the metacarpophalangeal joints in the hand or the metatarsophalangeal joints in the foot is indurated, thickened, and hard and is often shiny with loss of skin surface markings. Loss of skin elasticity also occurs. A "salt and pepper" pattern of *hyperpigmentation* (excess pigmentation of the skin) and *hypopigmentation* (reduced pigmentation of the skin) is common.

- **Minor Criteria**
- **sclerodactyly -** stiffness and tightening of the skin of the fingers
- **digital pitted scars or loss of substance of the finger pad**
- **bibasilar pulmonary fibrosis** - fibrosis of the base of both lungs that is evident on a chest X-ray.

The diagnosis of scleroderma is approximately 97% accurate with **one** major or **two** minor criteria present.

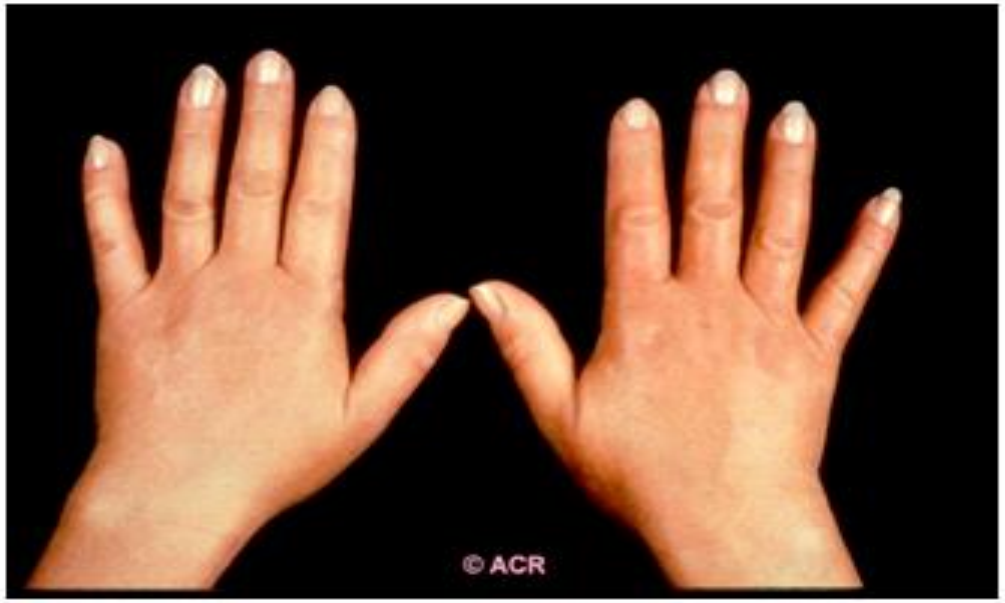
Digital tip pitting scars

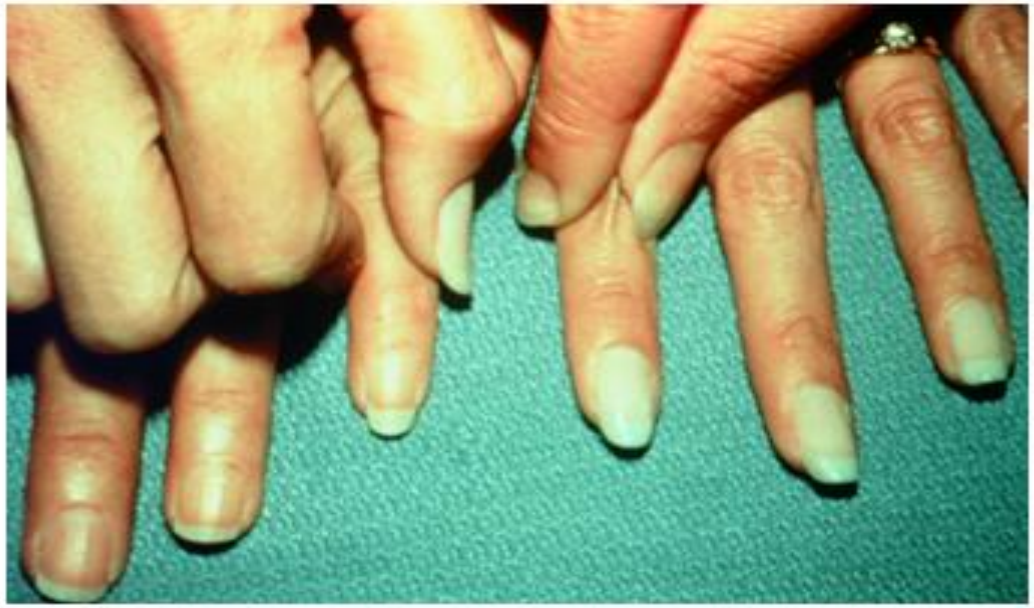


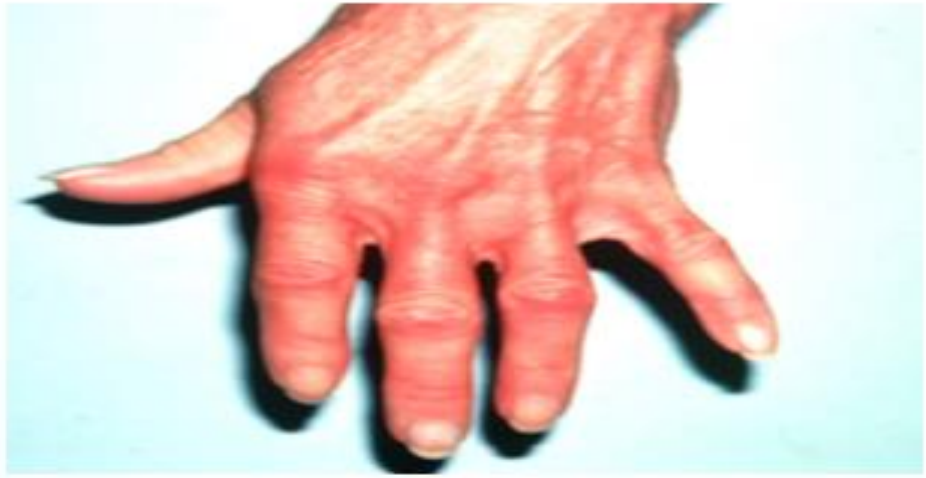


Nailfold capillary abnormalities

Taut, thin skin of fingers
sclerodactyly





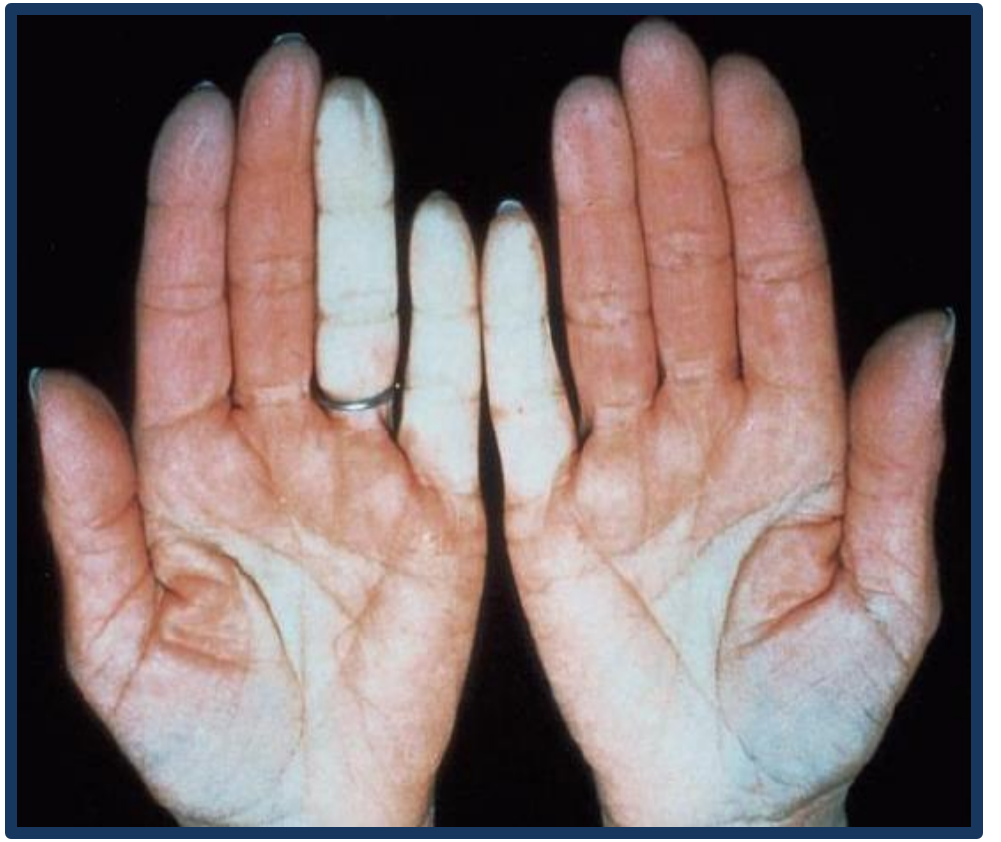


Q: What's your diagnosis?

Scleroderma

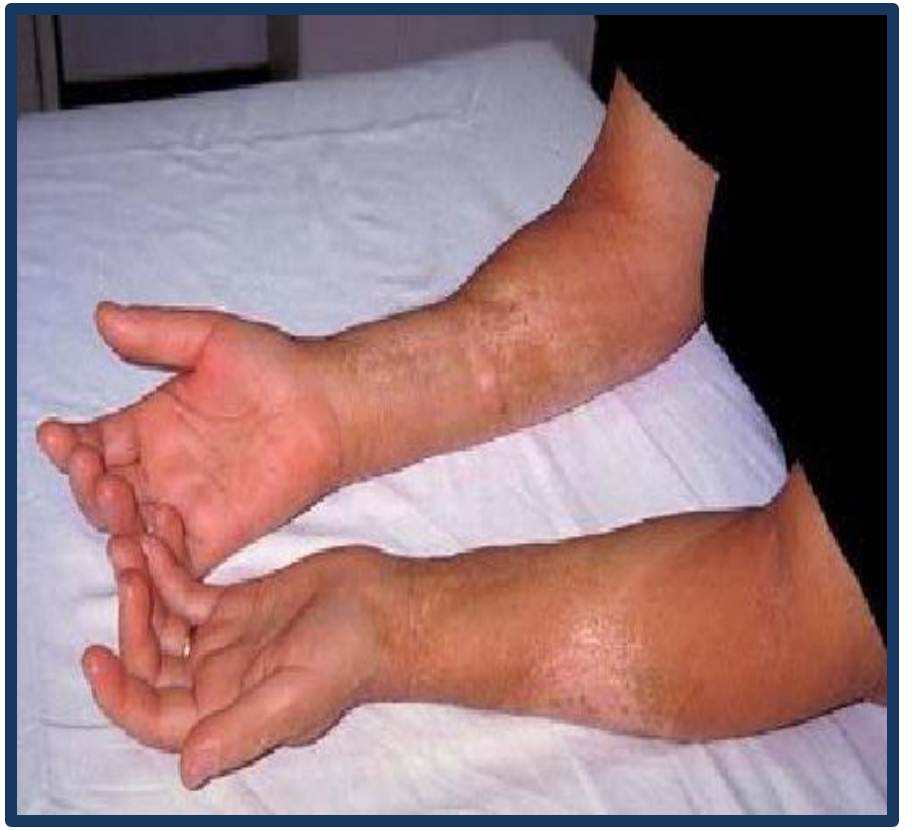
Q: What is the name of this sign?

Raynaud's phenomenon.



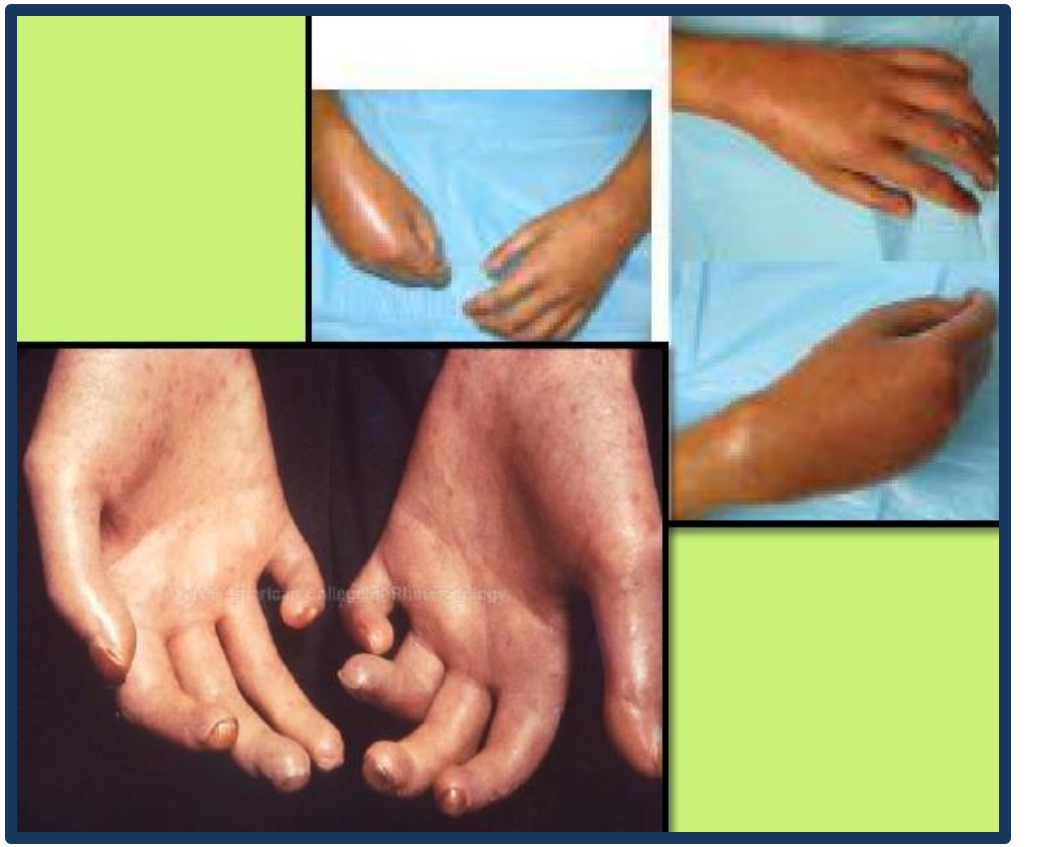
Q: What is your spot Dx?

Scleroderma.



Q: A pt presented with difficulty swallowing & chest pain, what is your Dx?

Scleroderma.



Q: this patient started to complain from progressive SOB, mention the cause ?

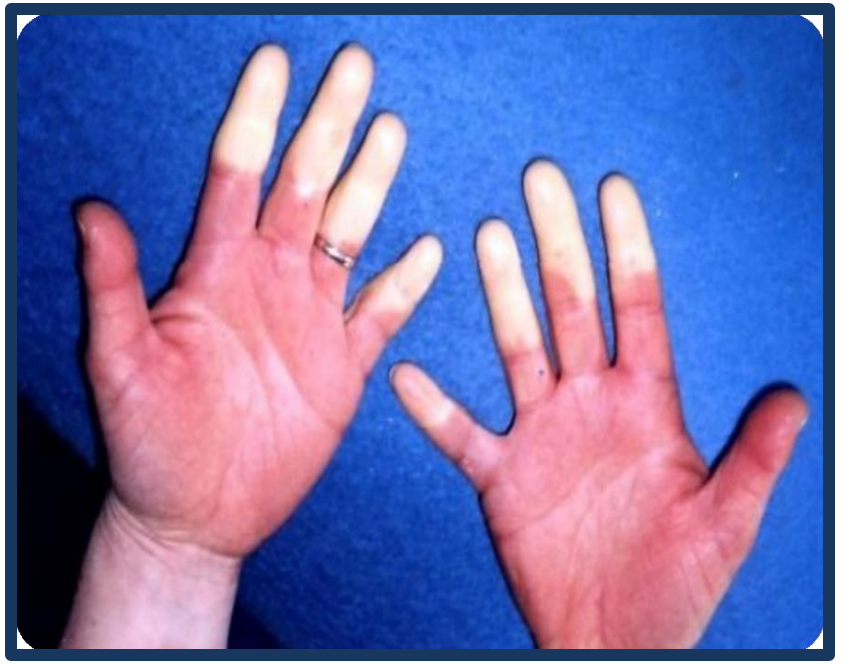
lung fibrosis

What is this sign?

Raynaud's phenomena

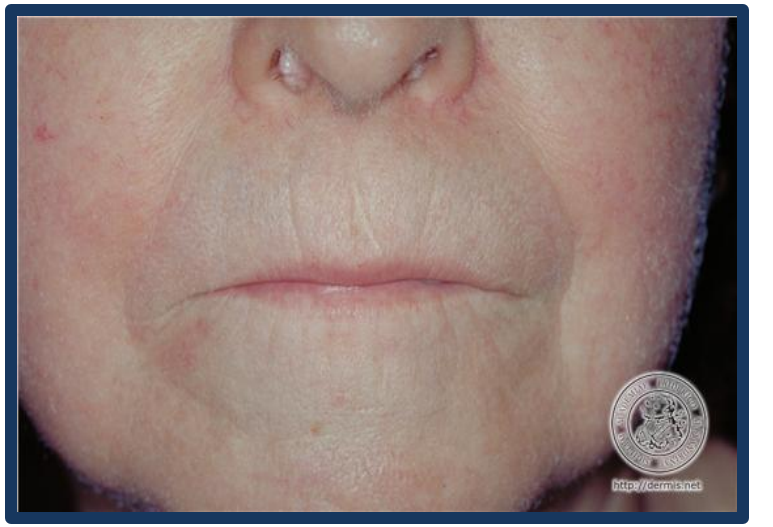
Name one disease causing it?

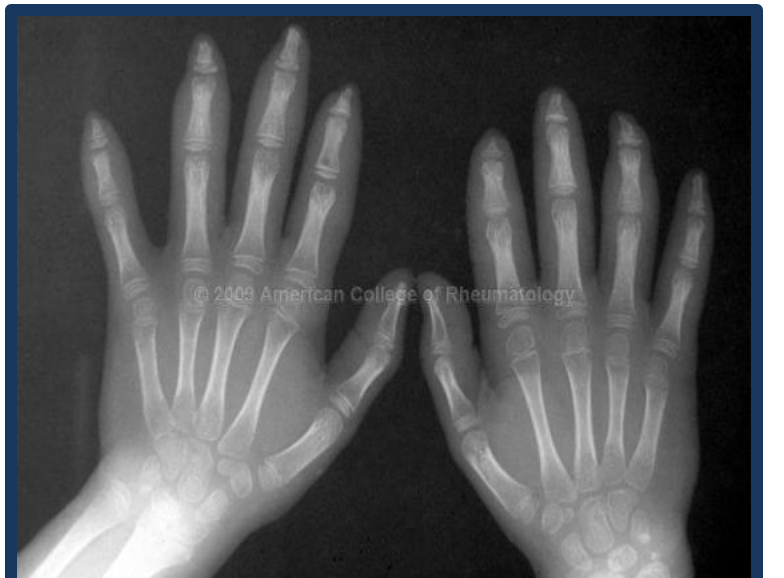
Systemic lupus erythrematosus , scleroderma



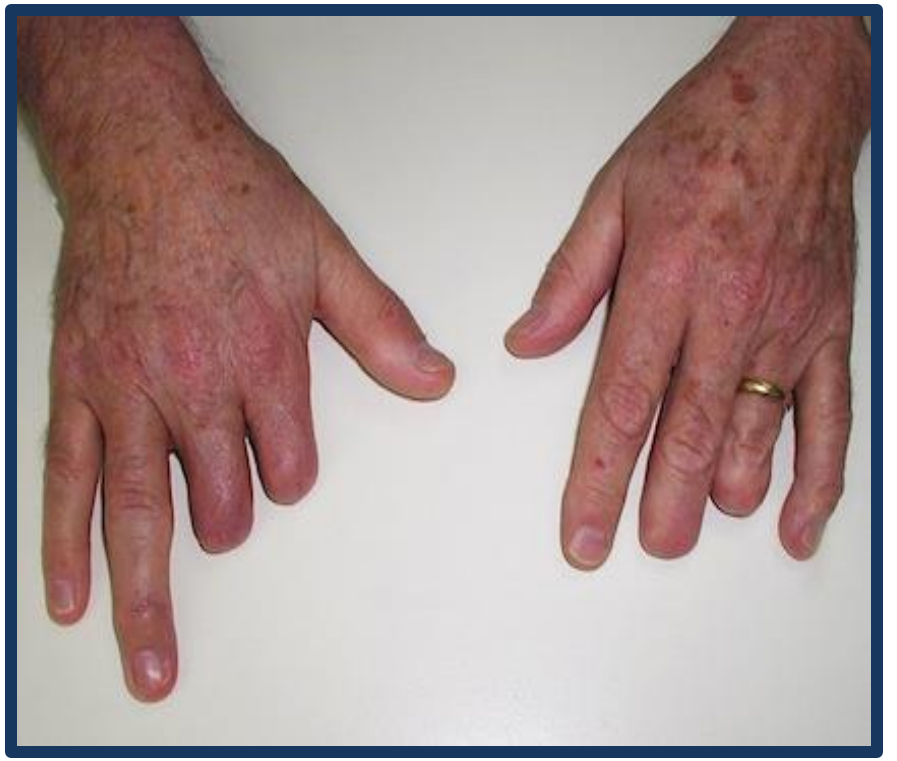
Q: Your diagnosis, the most common GI abnormality associated with this condition?

Scleroderma, Dysphagia

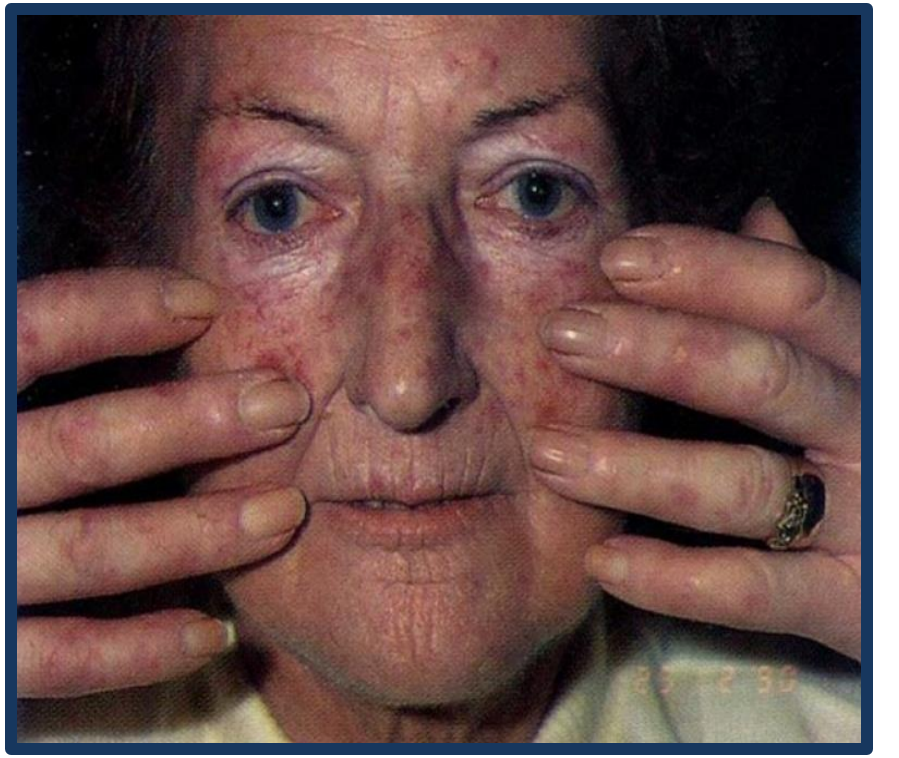




Q:A scenario asking for
1- diagnosis
Scleroderma (don't know if CREST syndrome is acceptable as well)
2- Two causes of shortness of breath
Lung fibrosis / Pulmonary hypertension



Q: This patient has Raynaud Phenomenon, severe heart burning sensation and dysphagia presents with chronic hypoxia.
- Name 2 possible causes of chronic Hypoxia.
1. lung fibrosis
2. pulmonary hypertension

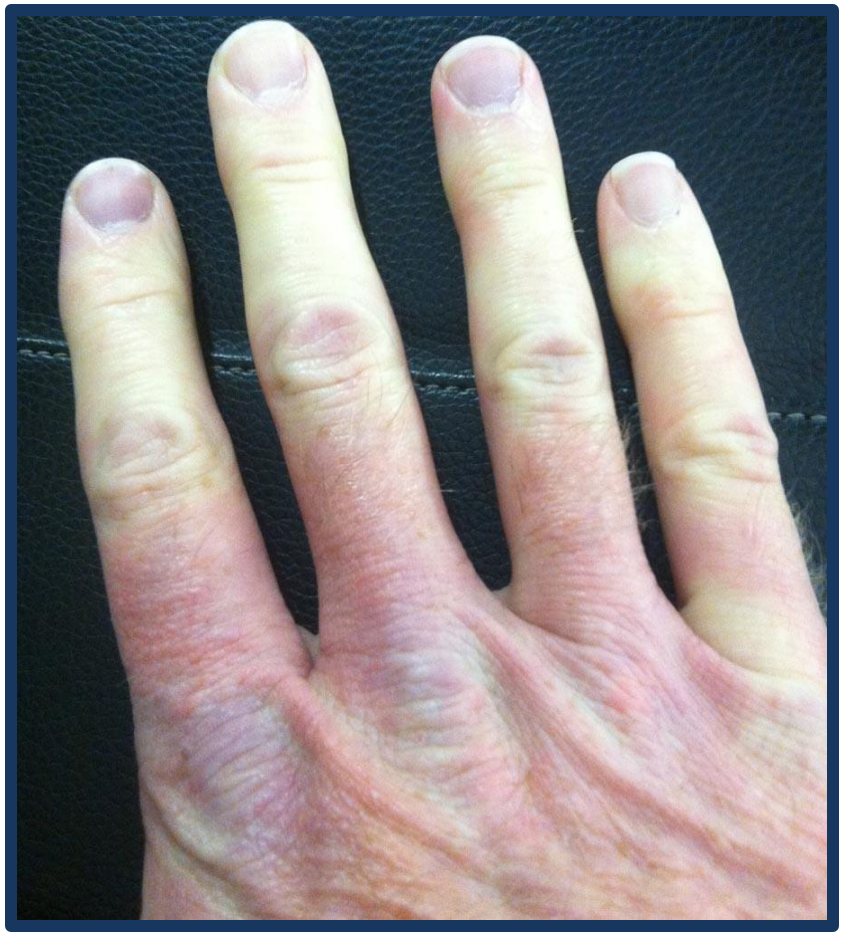


Q: What is your spot diagnosis?

Raynaud's phenomenon

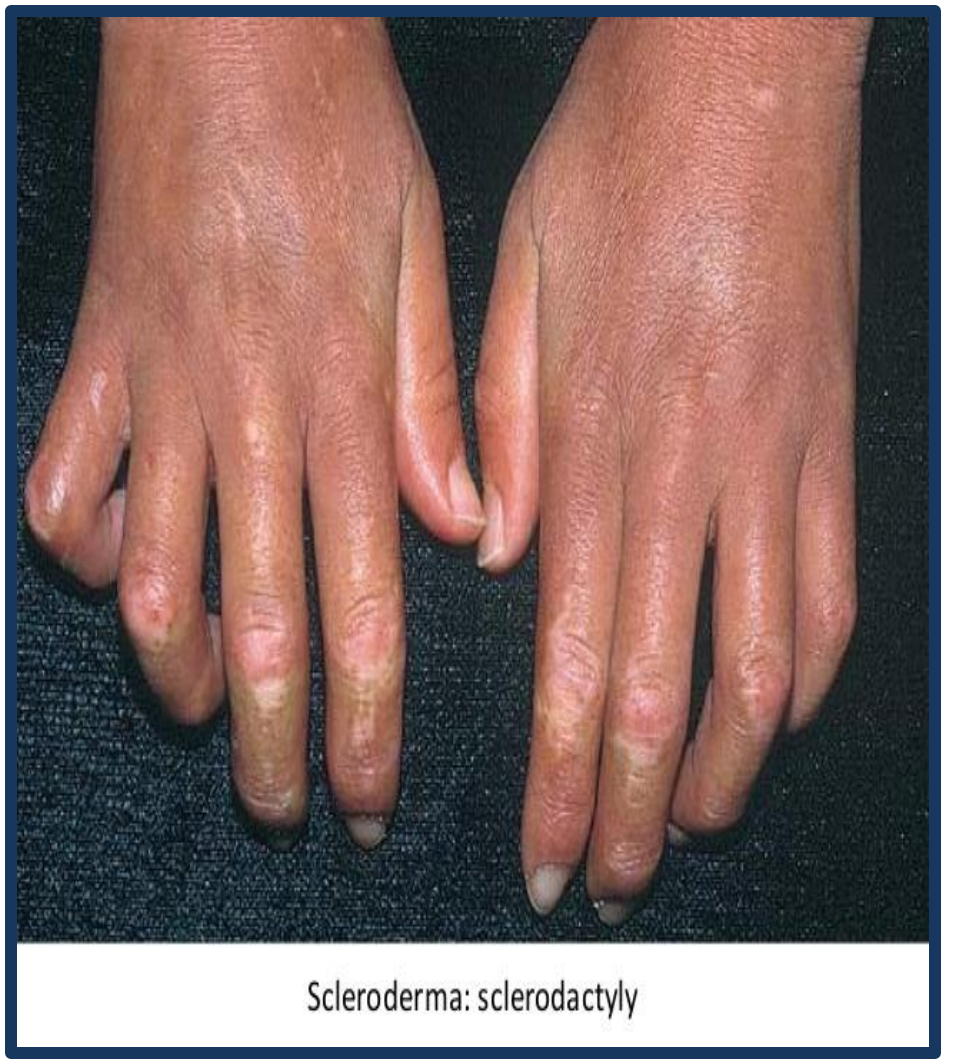
Give one associated disease with this condition.

Systemic sclerosis



Q: What is the diagnosis in this pt?

Scleroderma

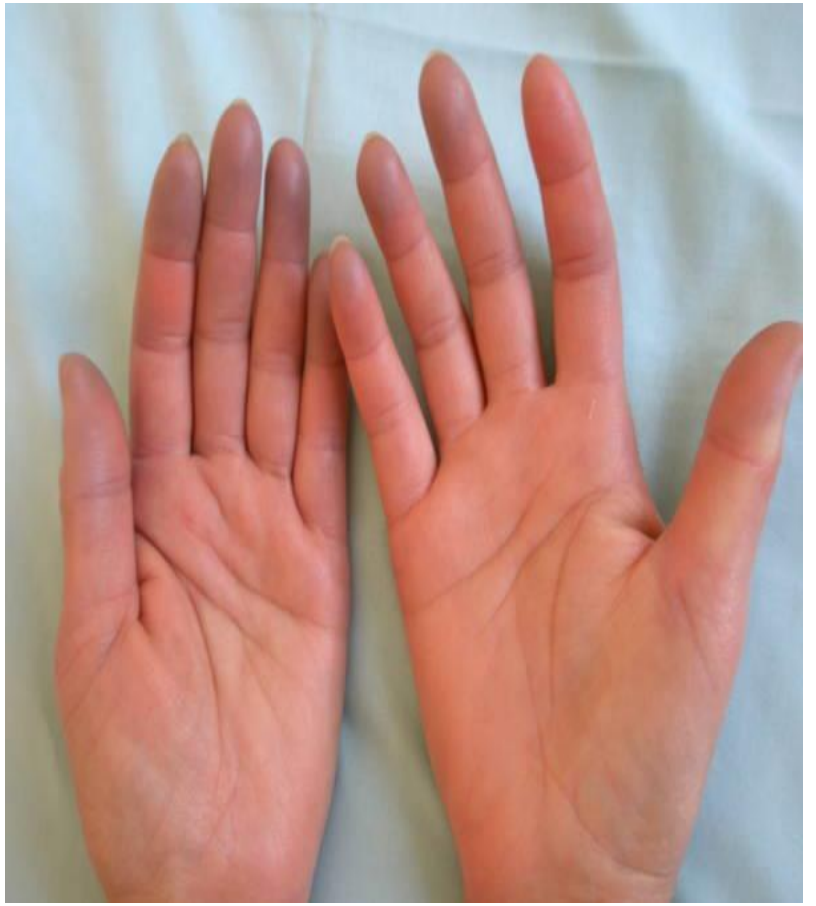


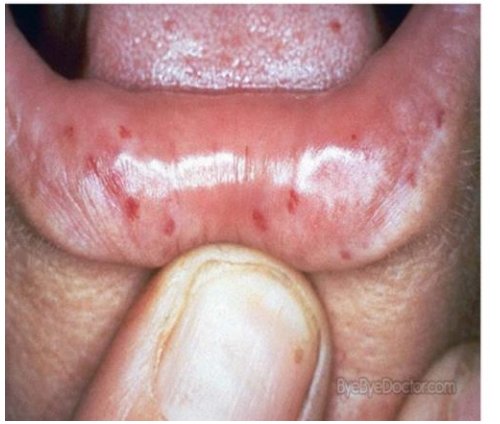
Scleroderma: sclerodactyly

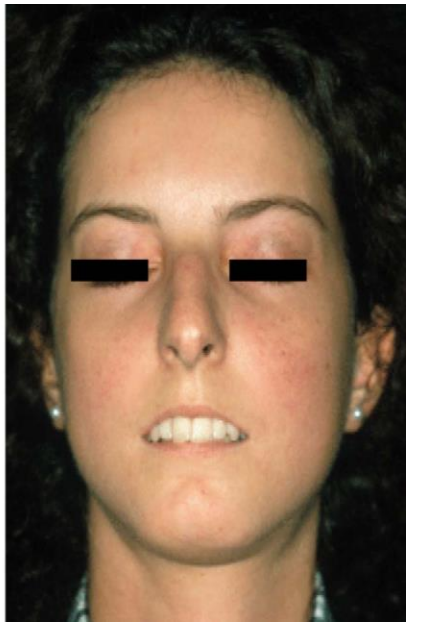
Scleroderma

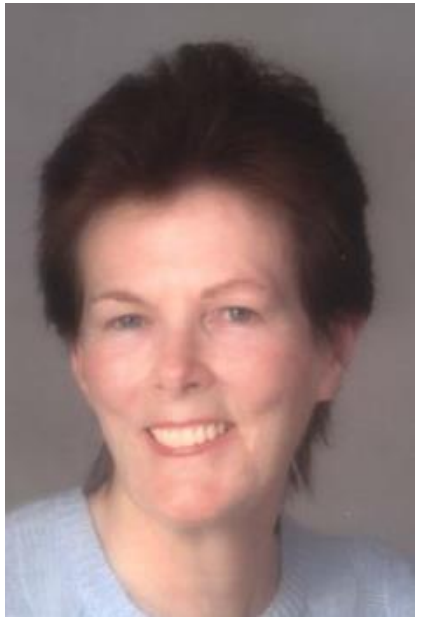
Q: Name this sign mention one association

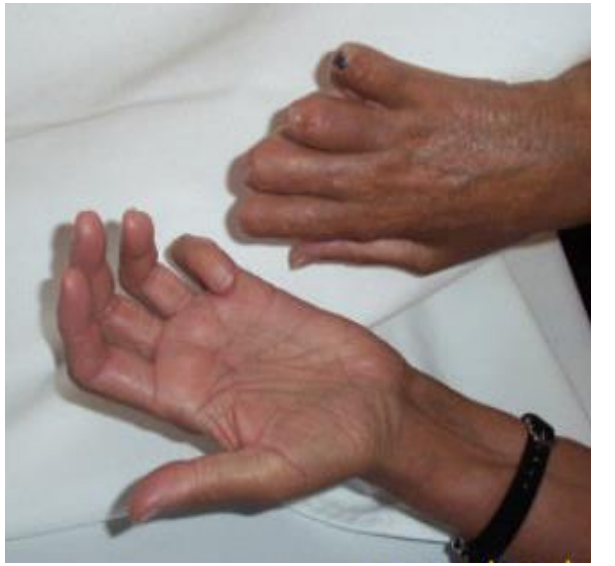
Ryanoud's → Scleroderma



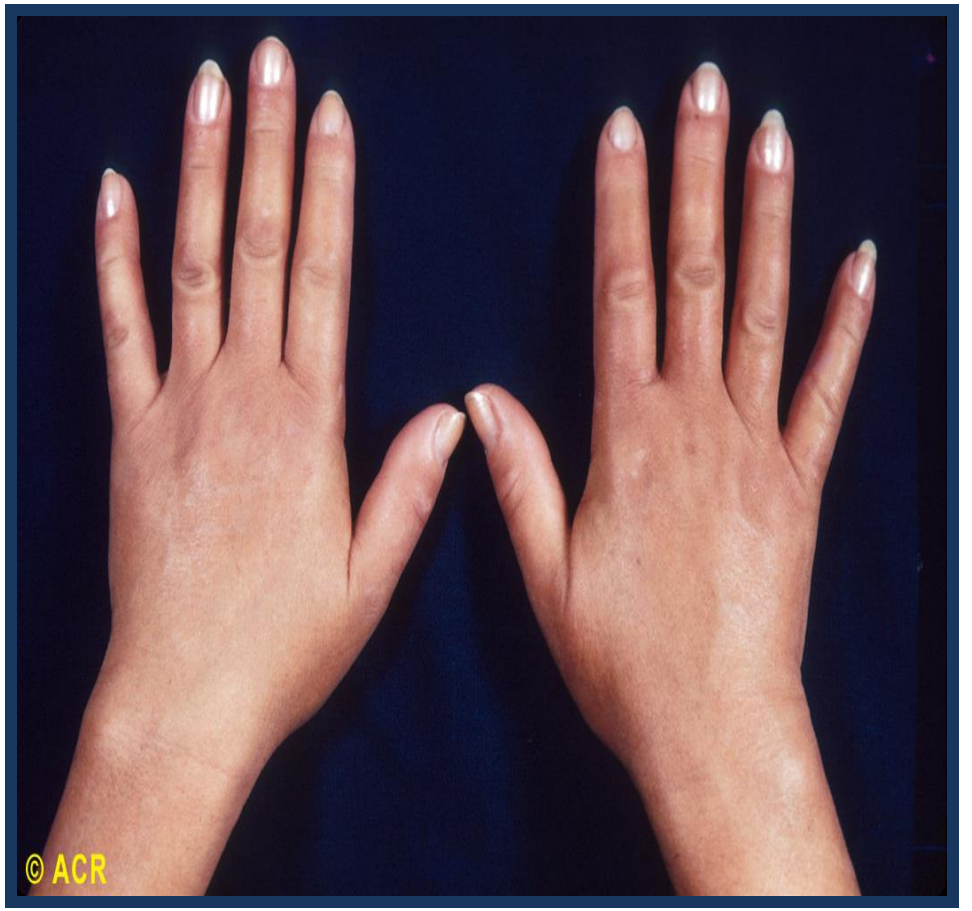




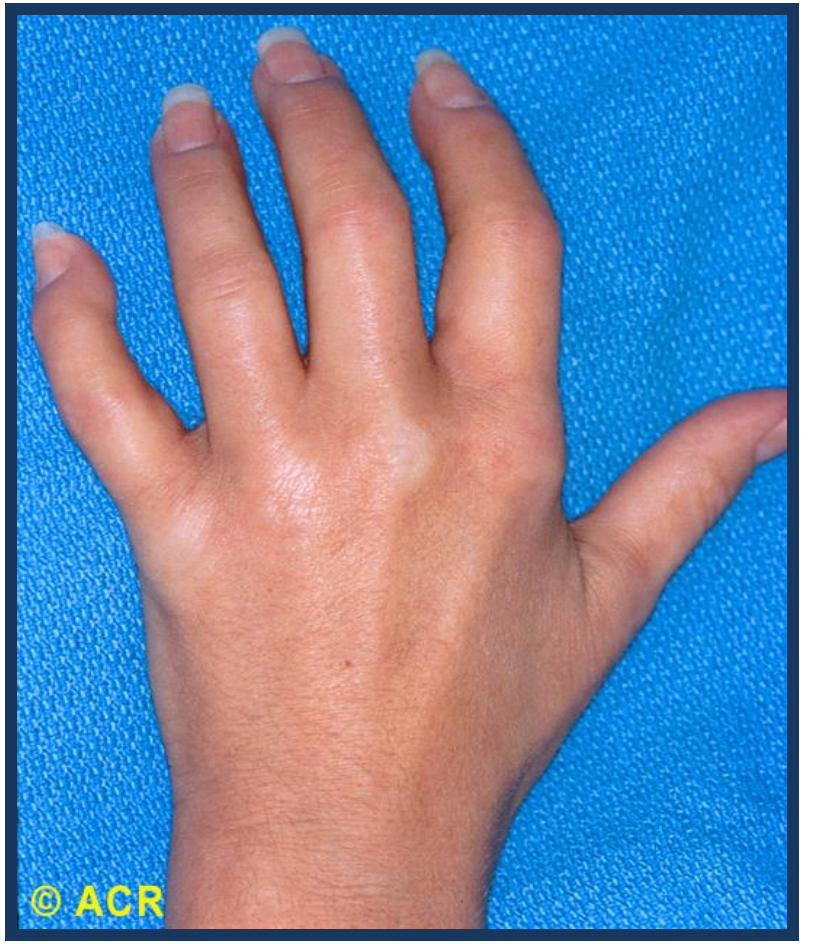




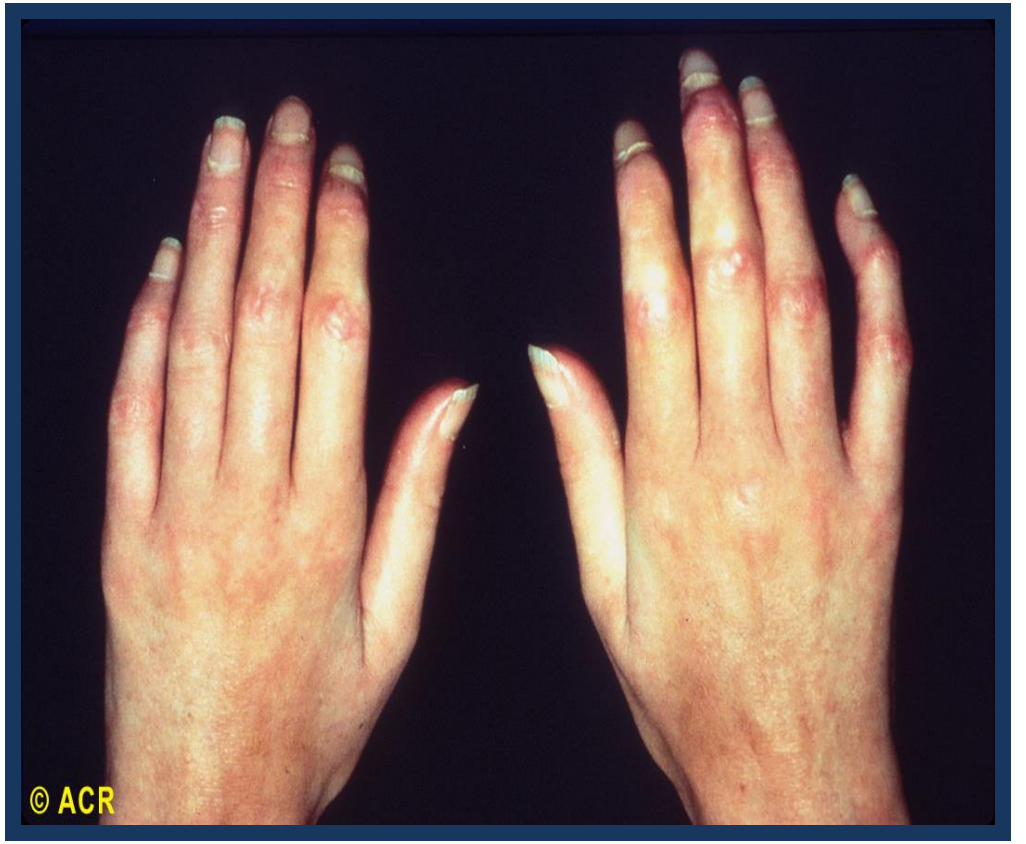
Scleroderma: edematous changes, hands



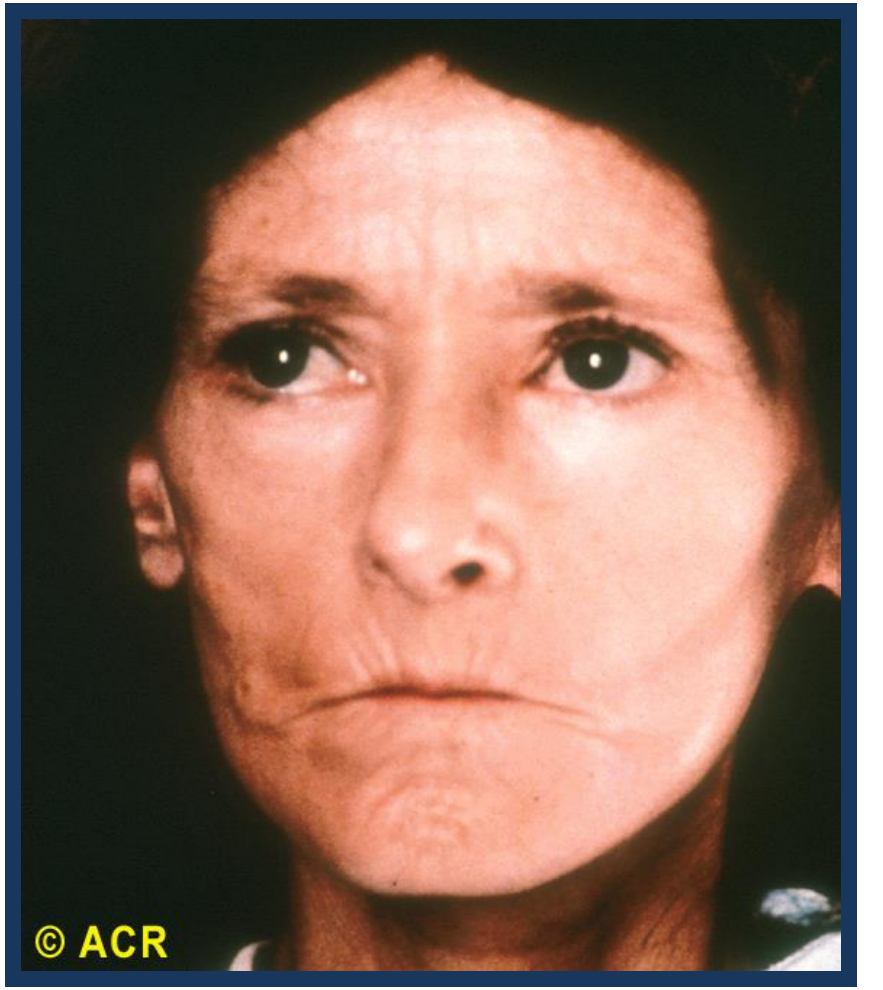
Scleroderma: puffy phase, hand



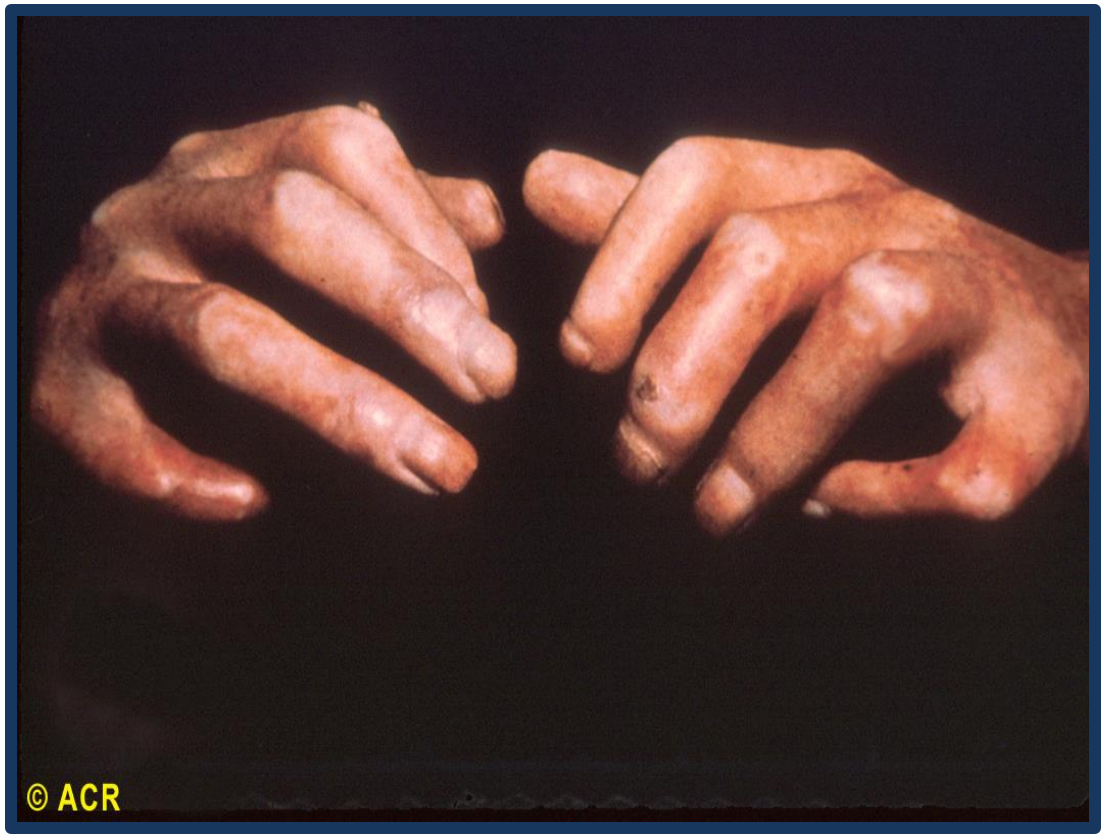
Scleroderma: skin induration, hands



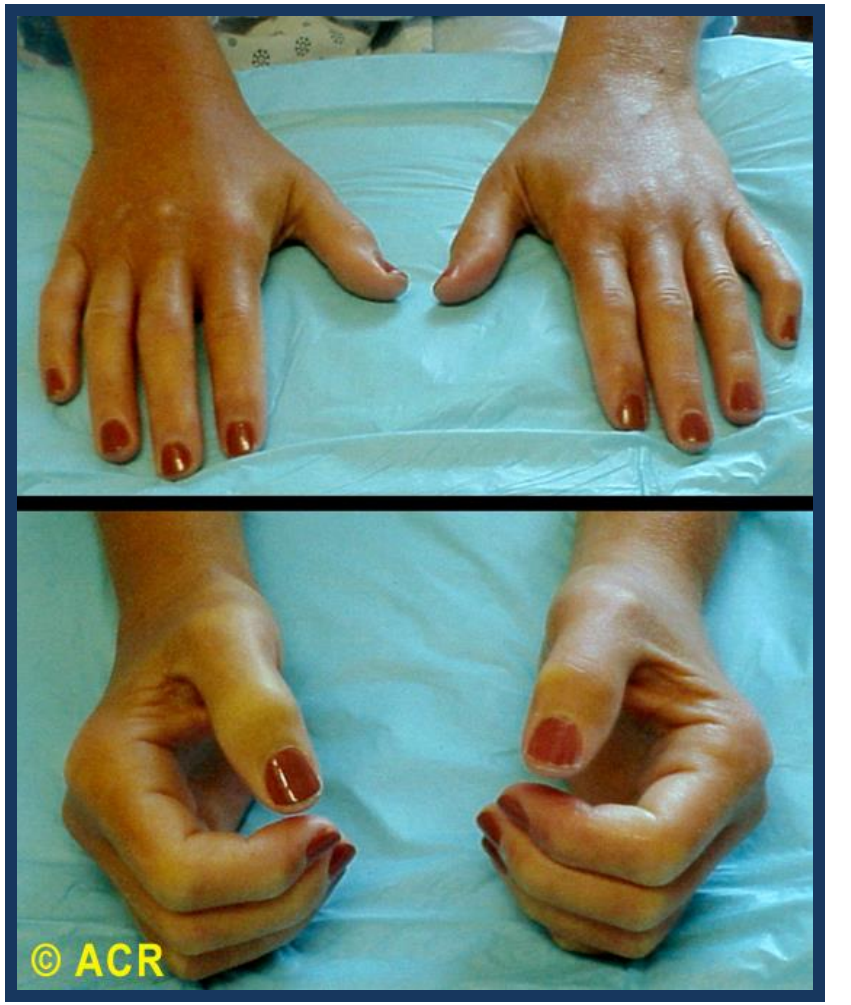
Scleroderma: Mauskopf, facial changes



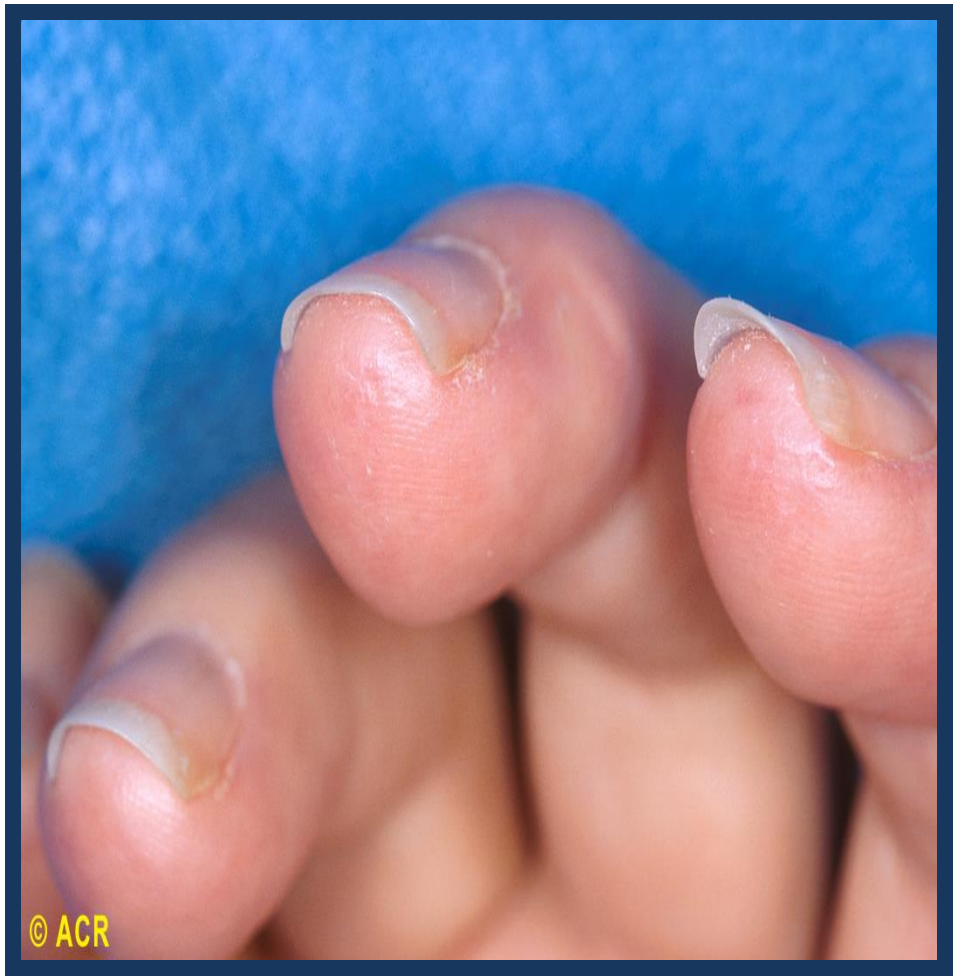
Scleroderma: acrosclerosis



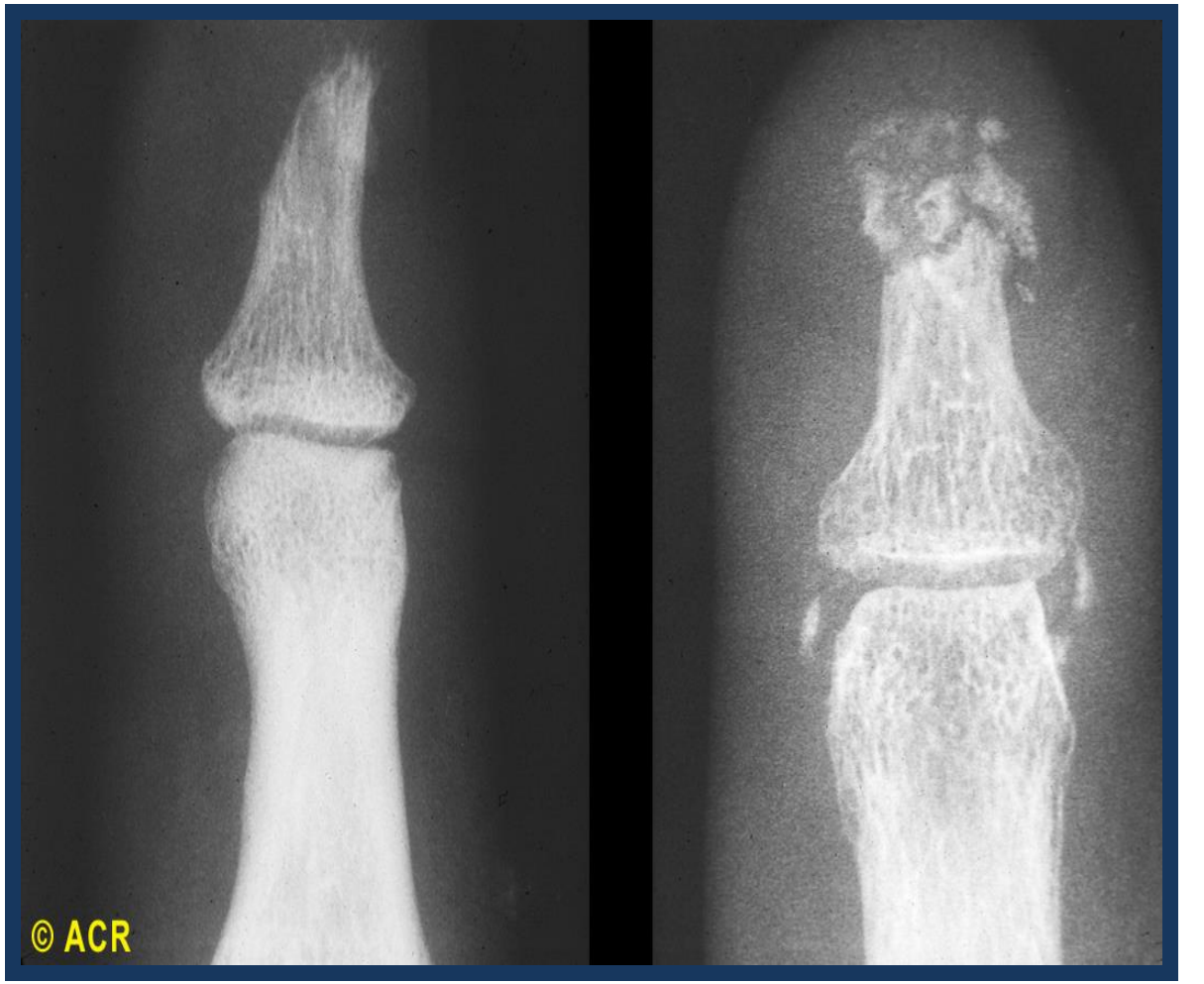
Scleroderma: hands



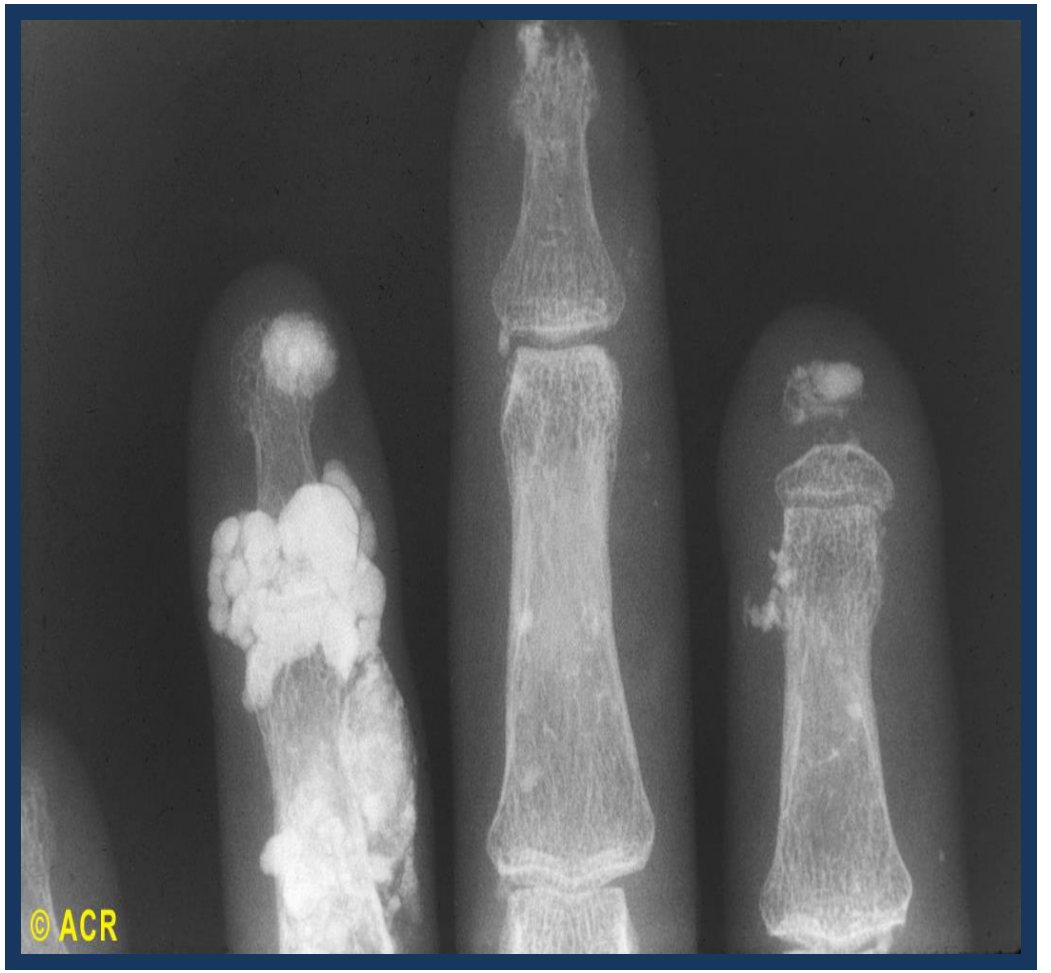
Scleroderma: digital pitting scars



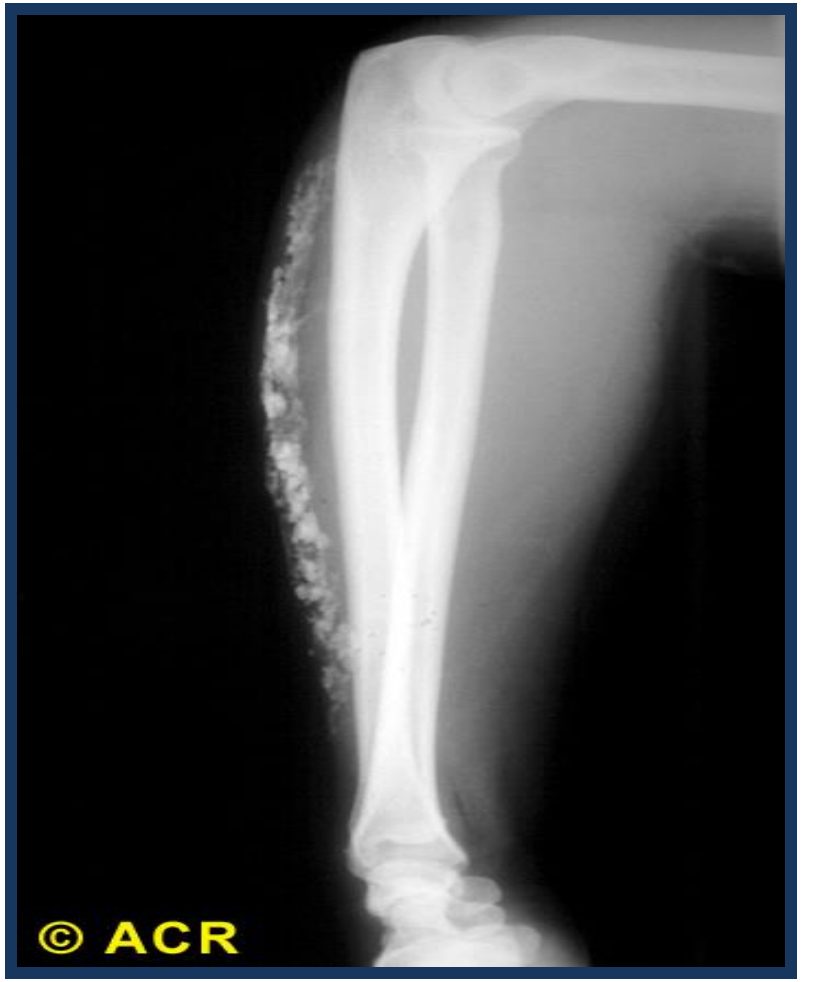
Scleroderma: acrolysis (radiographs)



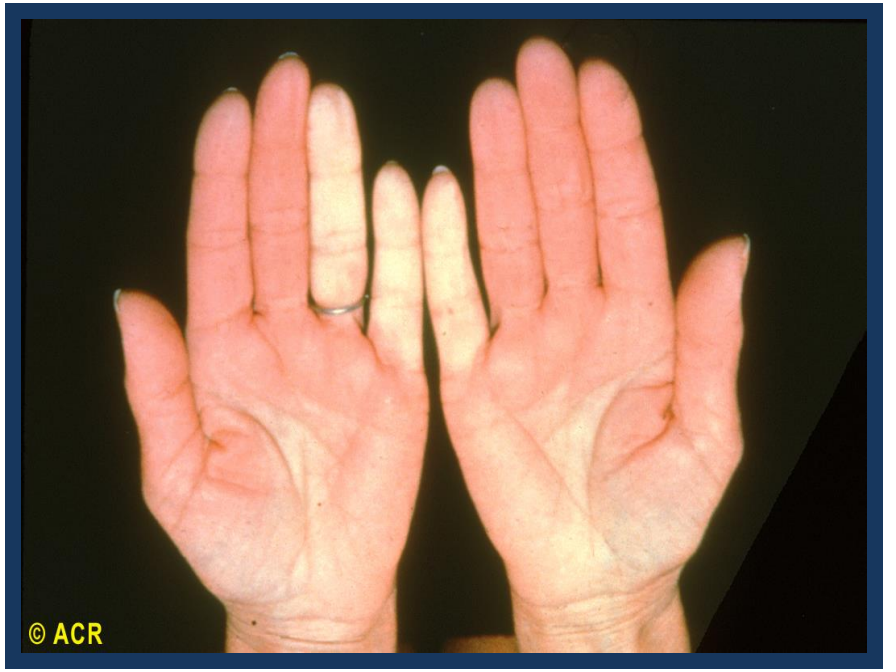
Scleroderma: calcinosis and acrolysis (radiograph)

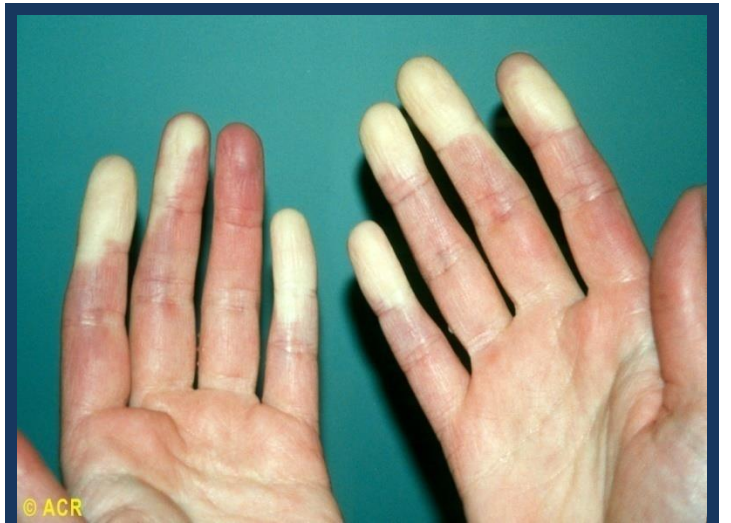


CREST syndrome: arm (radiograph)

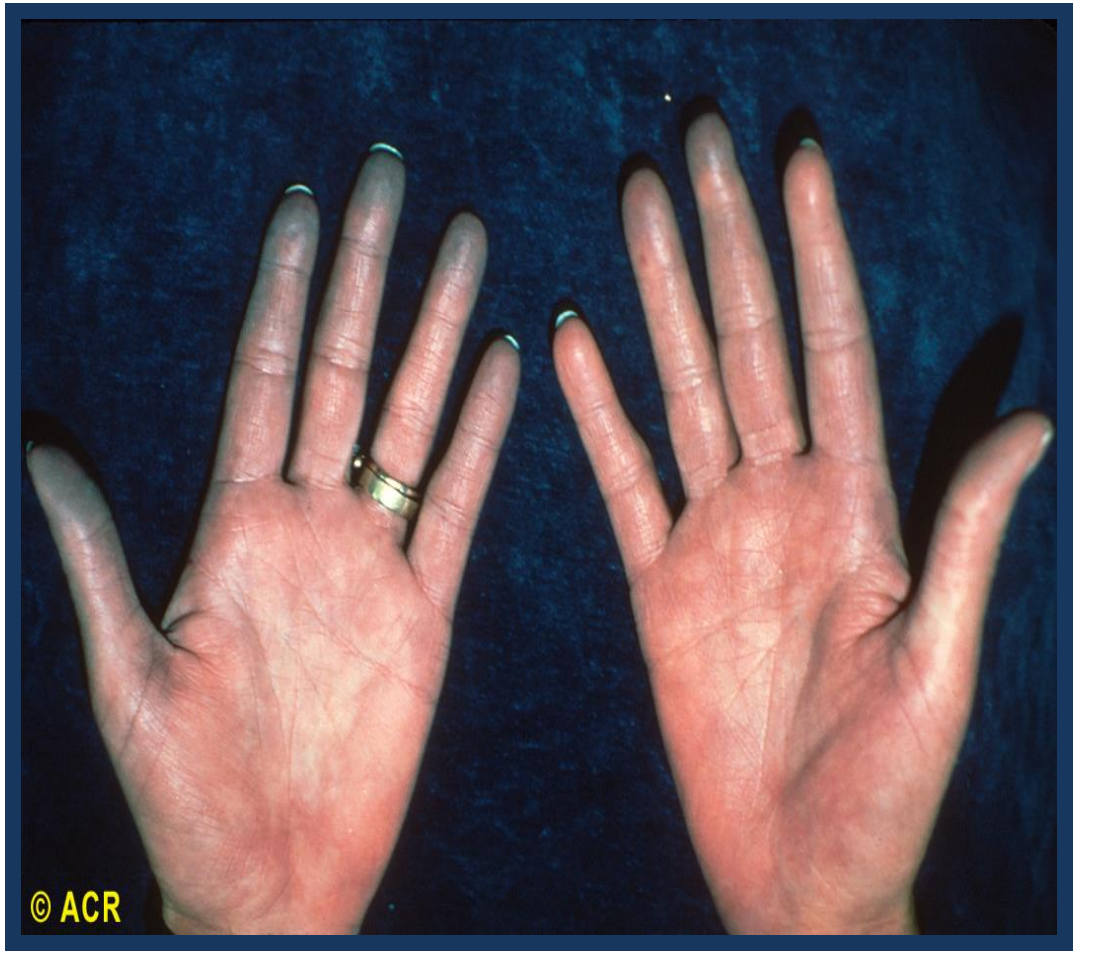


Scleroderma: Raynaud's phenomenon, blanching of hands

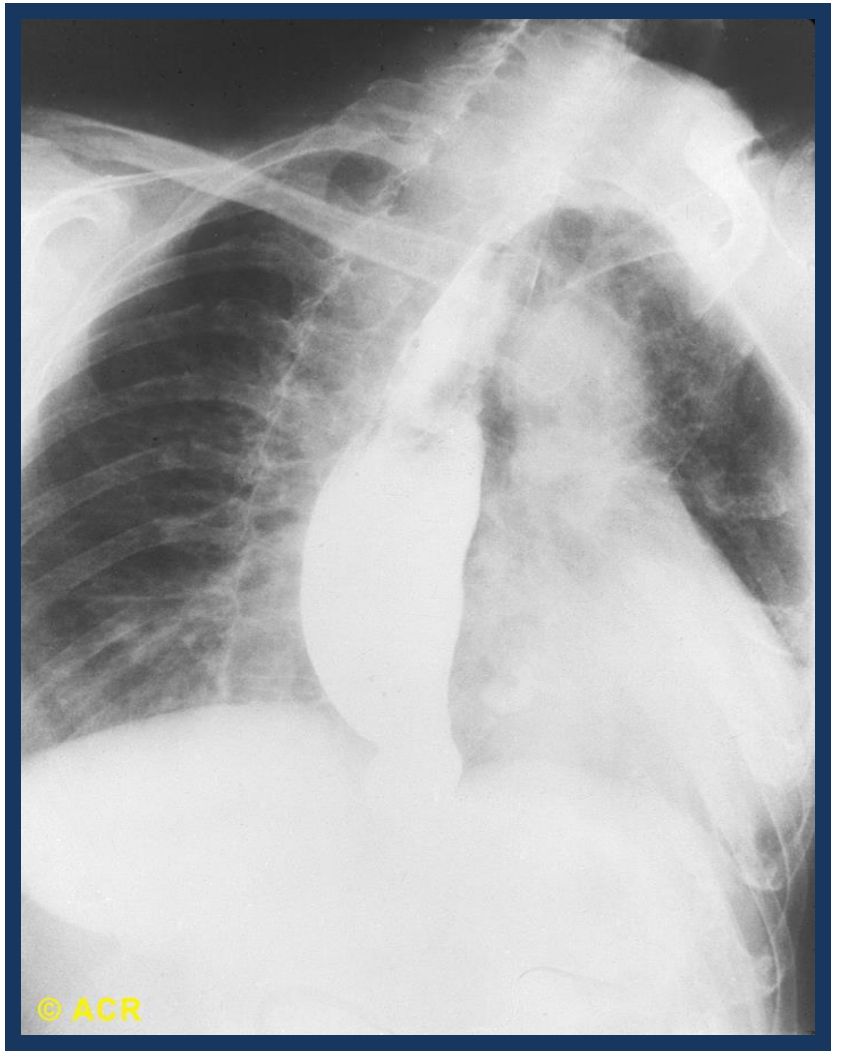




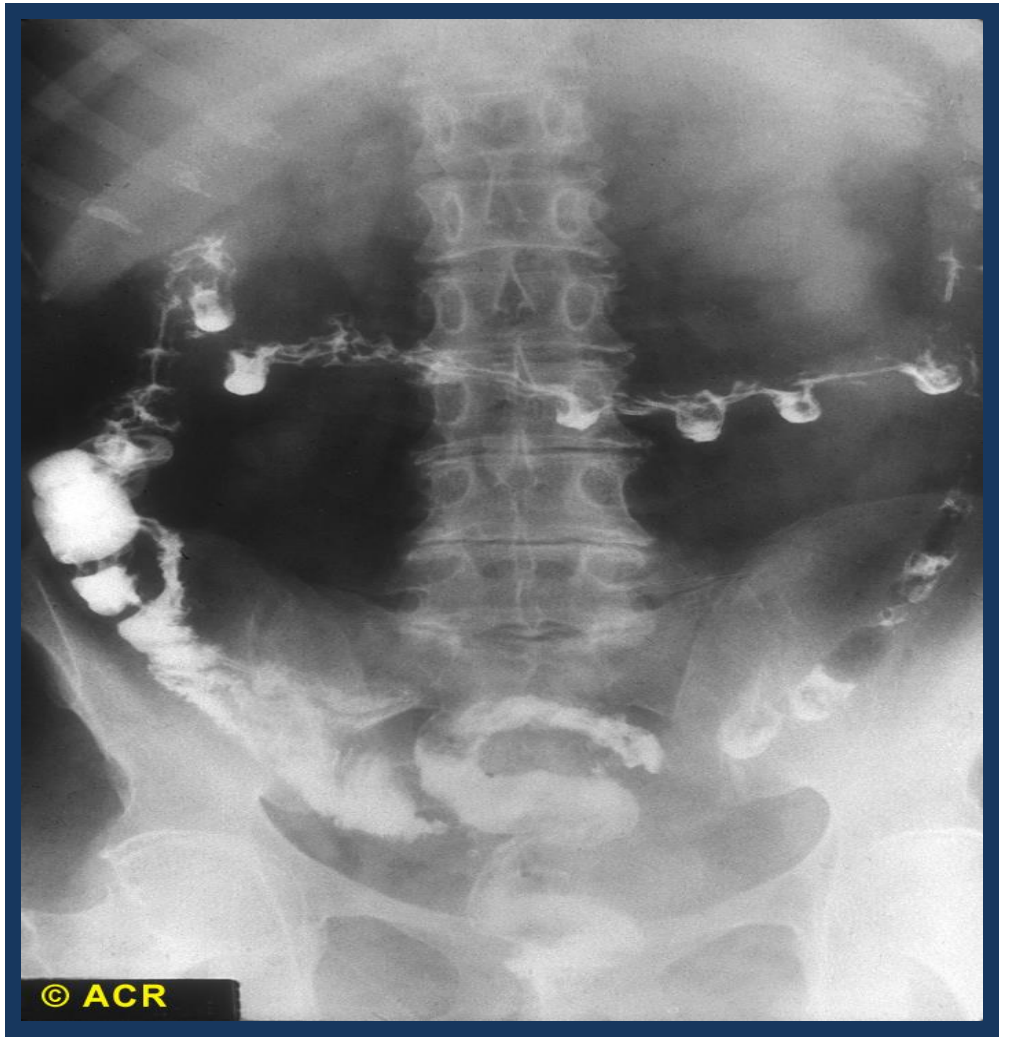
Scleroderma: Raynaud's phenomenon, cyanosis of the hands



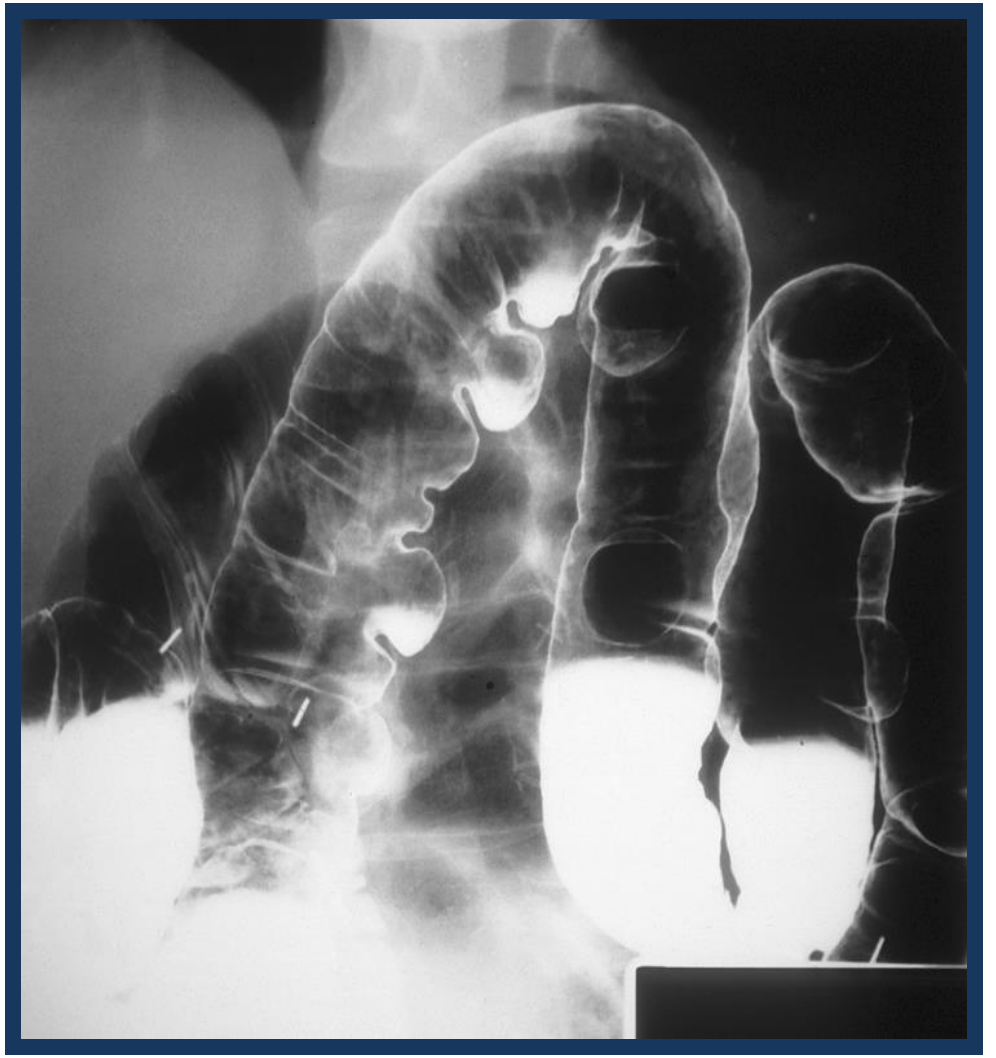
Scleroderma: abnormal motility, esophagus (radiograph)



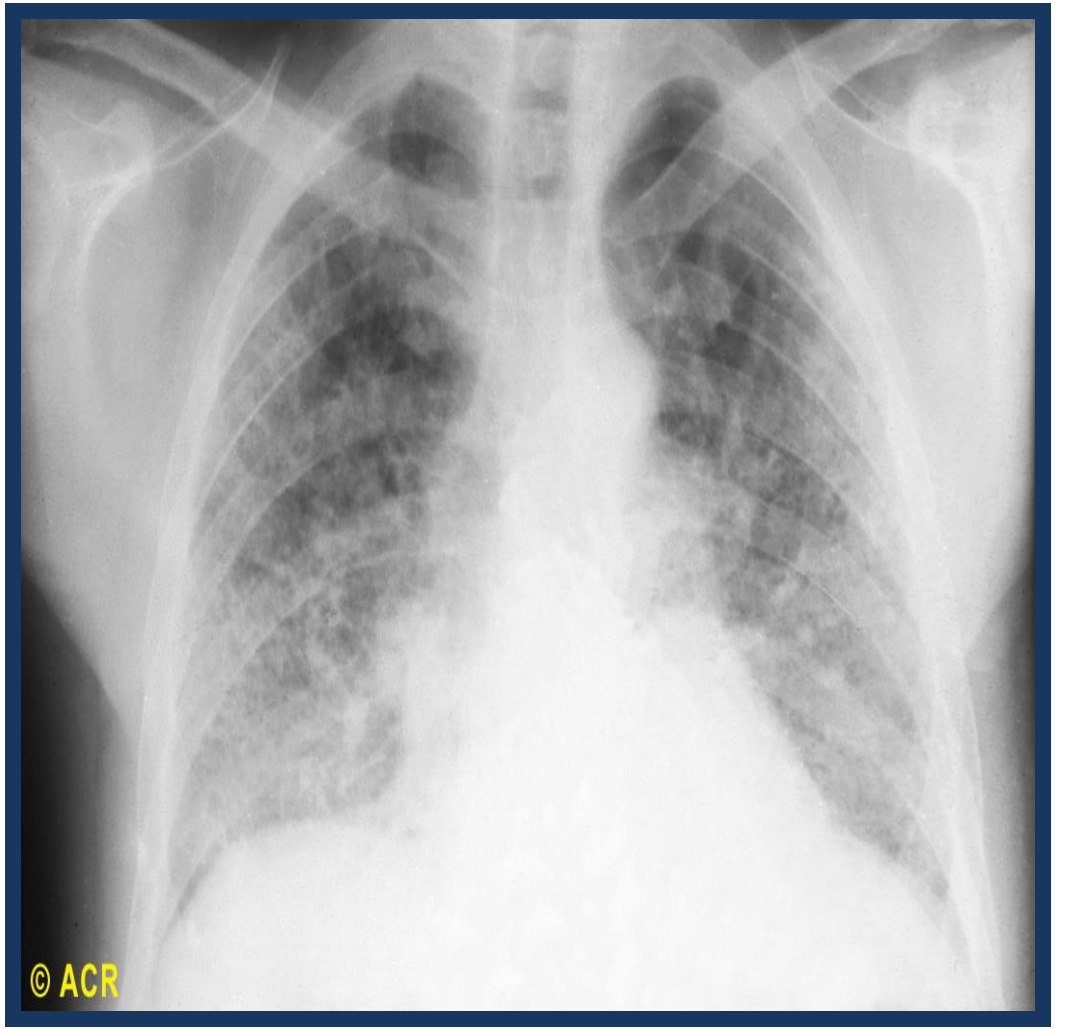
Scleroderma: wide-mouthed diverticula, colon (radiograph)



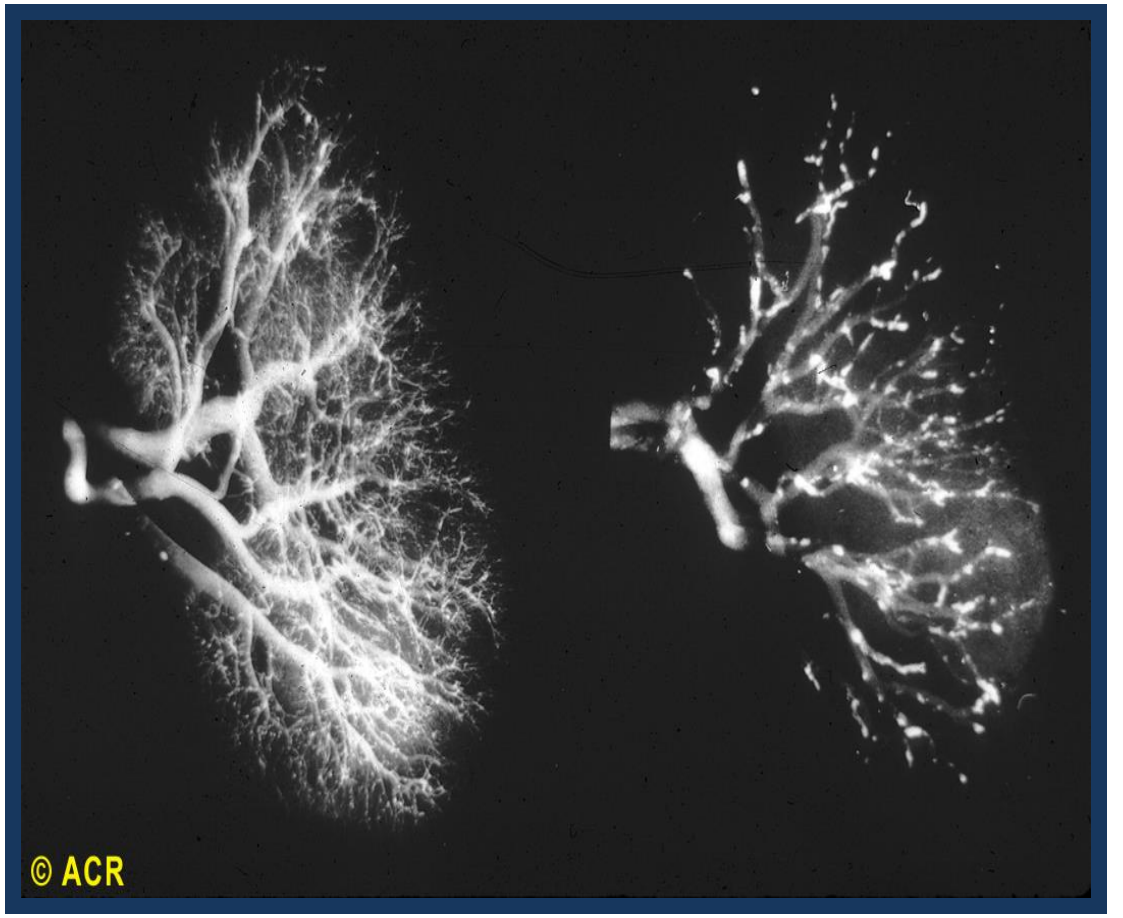
Scleroderma: large-mouth diverticula (radiograph)



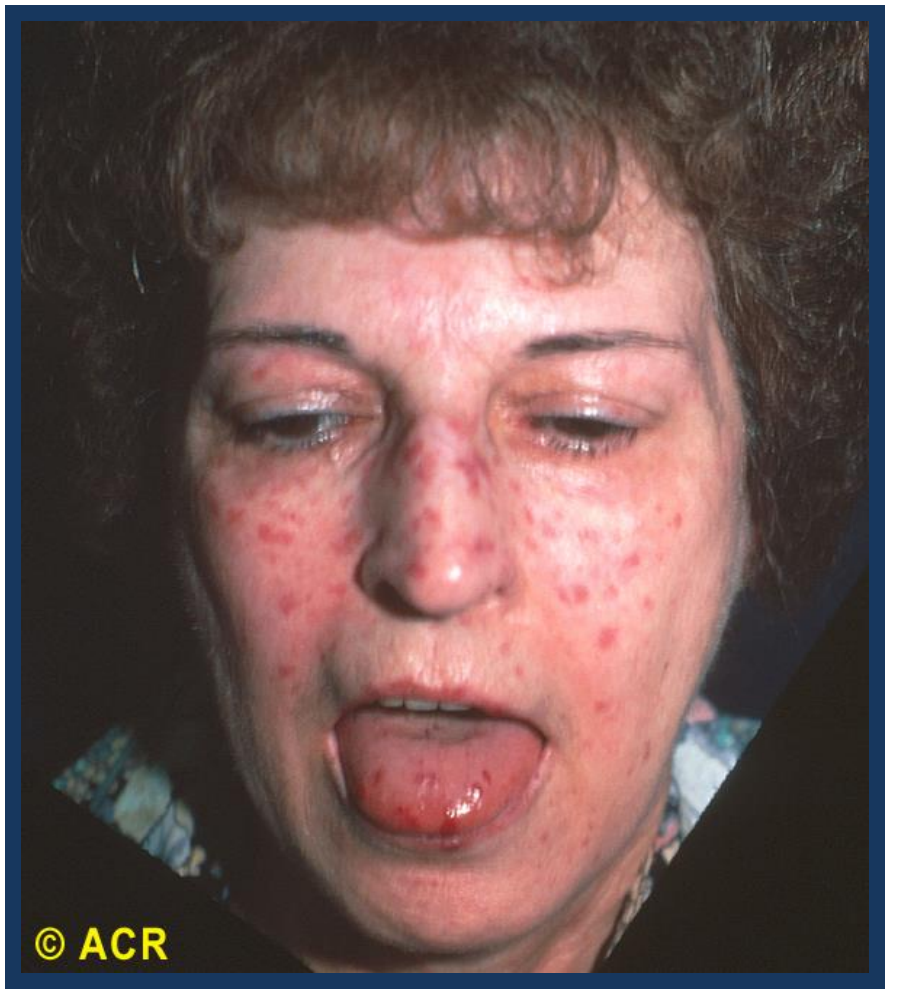
Scleroderma: pulmonary fibrosis (radiograph)



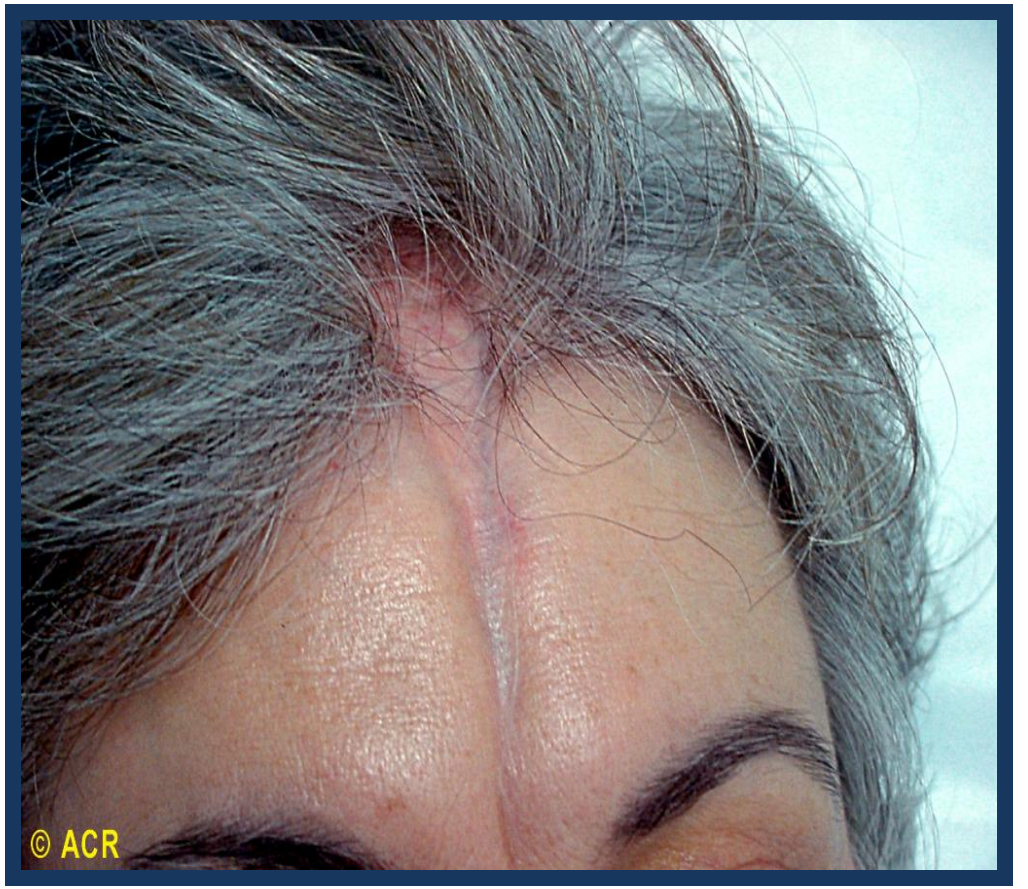
Scleroderma: kidney (arteriograms)



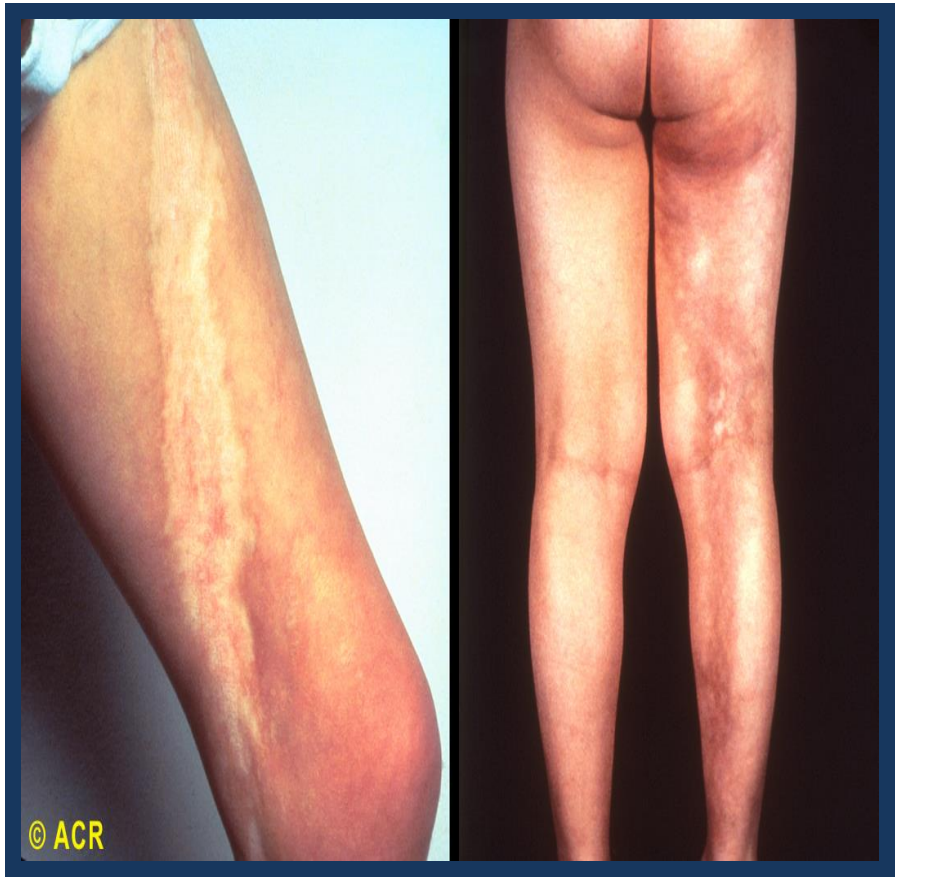
Scleroderma: Mauskopf, facial changes



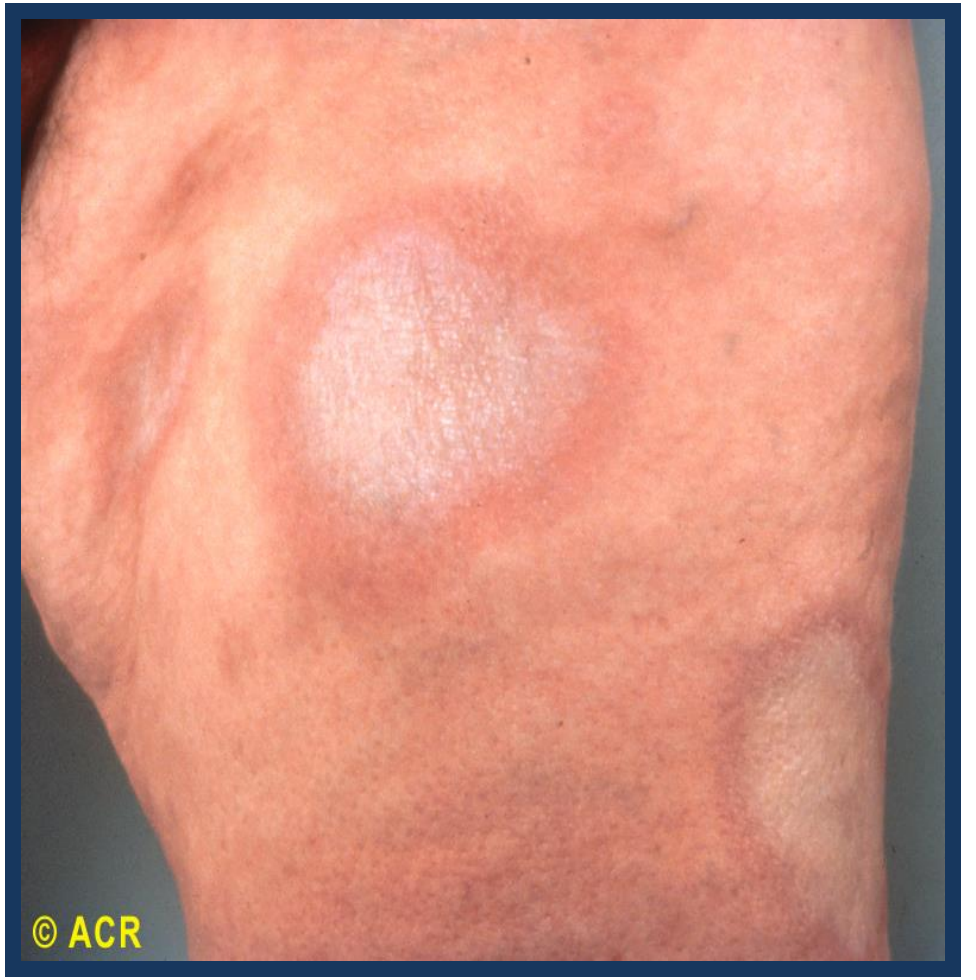
Linear scleroderma: en coup de sabre, scalp and forehead



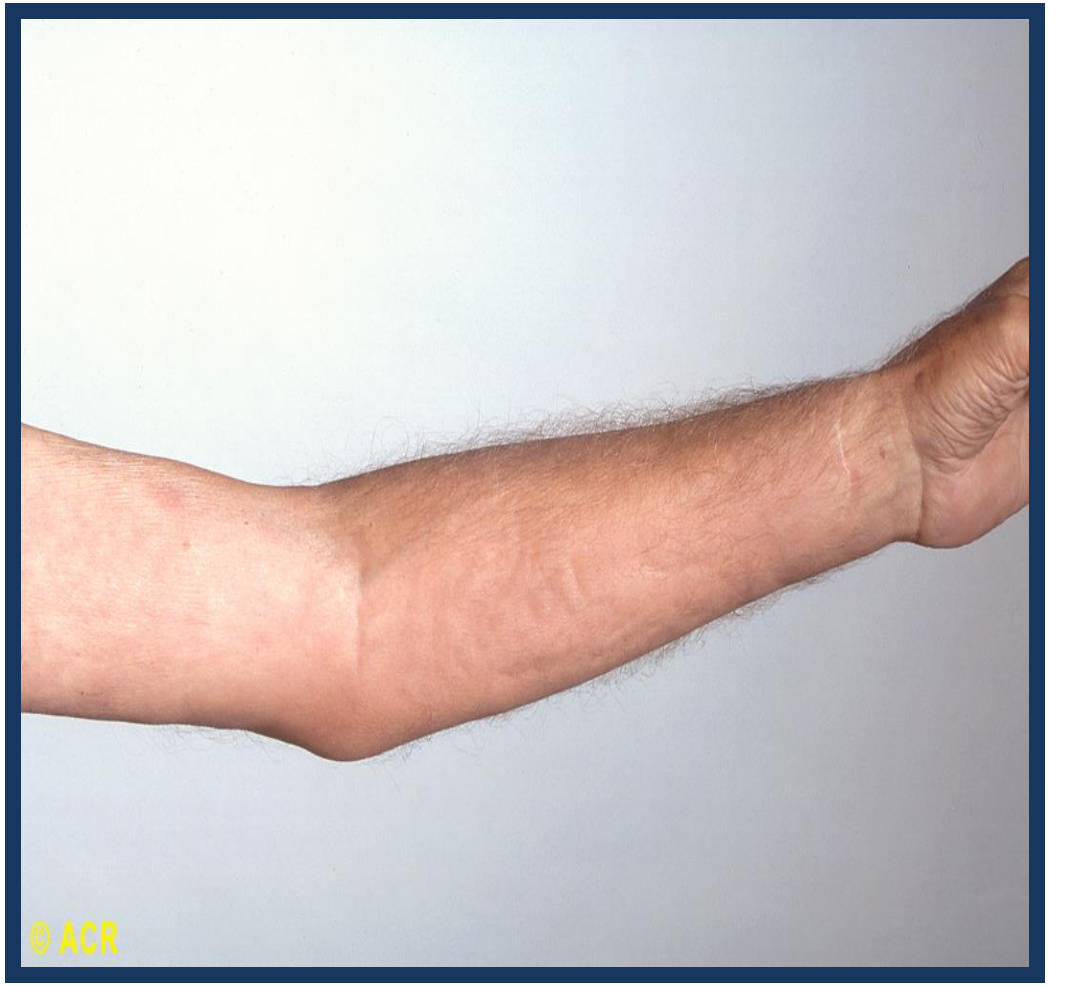
Linear scleroderma: thigh and leg



Morphea: leg



Eosinophilic fasciitis: cutaneous lesions, arm

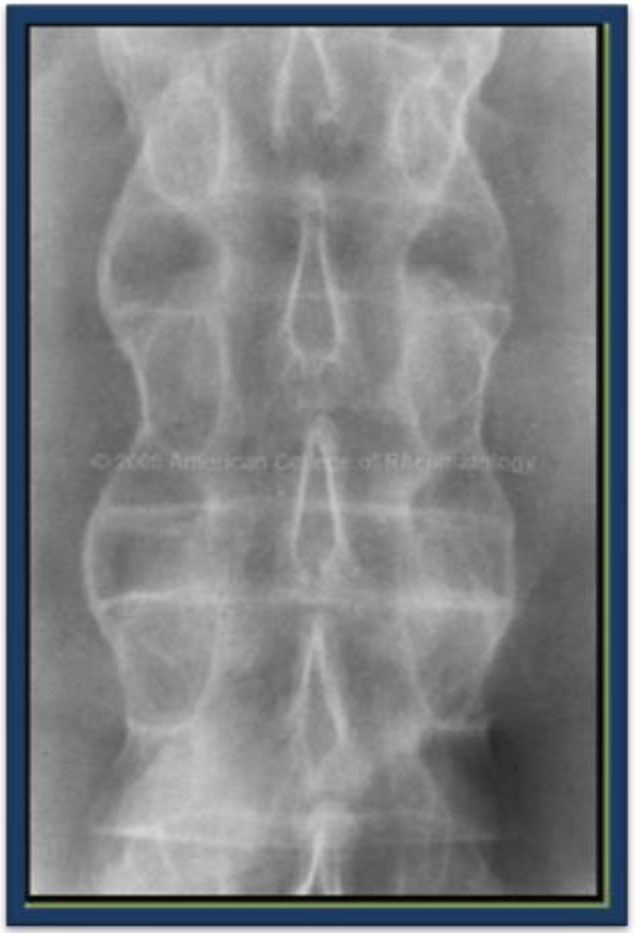


4.Ankylosing Spondylitis

Q:Male patient presented with unilateral uveitis. This is x-ray for his spine. What is your Dx.?

Ankylosing Spondylitis

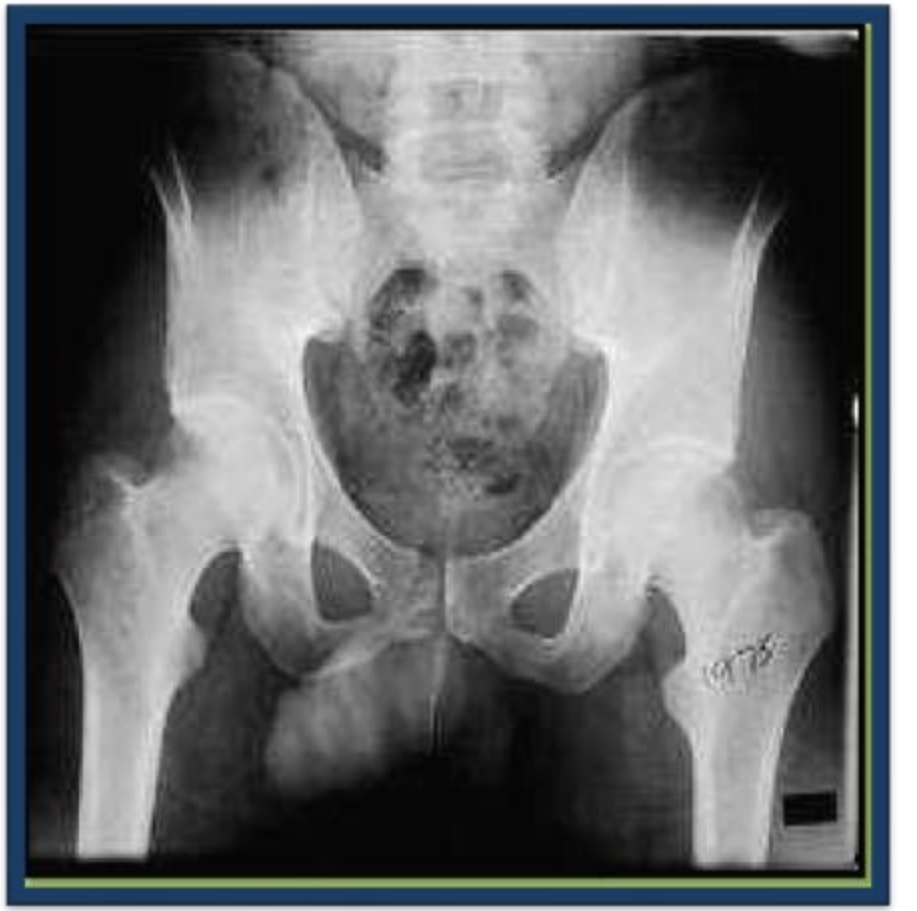
"Bamboo spine"



Q: a 28 YO male pt had chronic lower back pain with morning stiffness which improves with exercise. What is your Dx

Ankylosing Spondylitis.

Bilateral sacroiliiteis



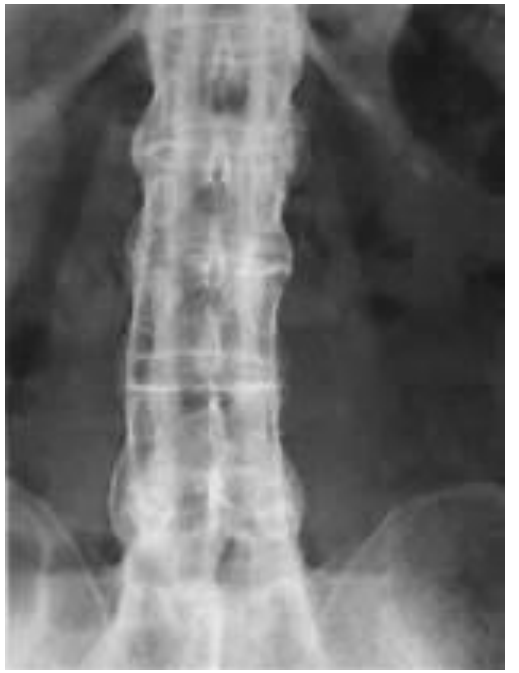
Q: Diagnosis?

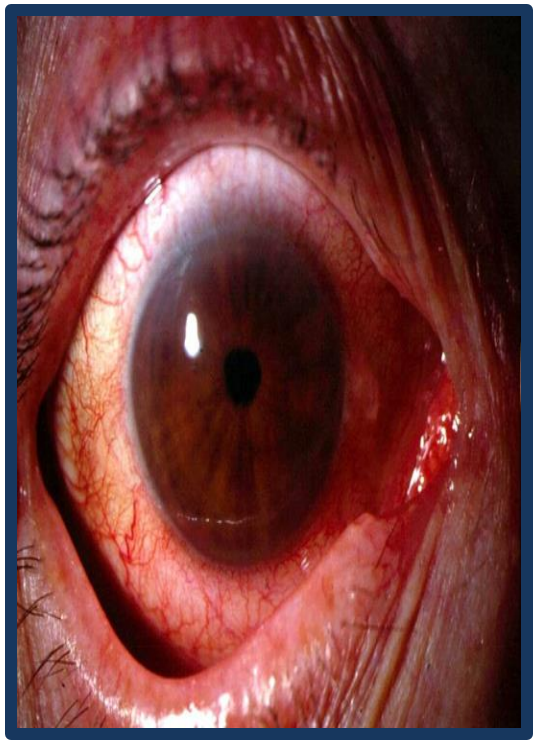
Bamboo spine and Anterior uveitis

So diagnosis is ankylosing spondylitis

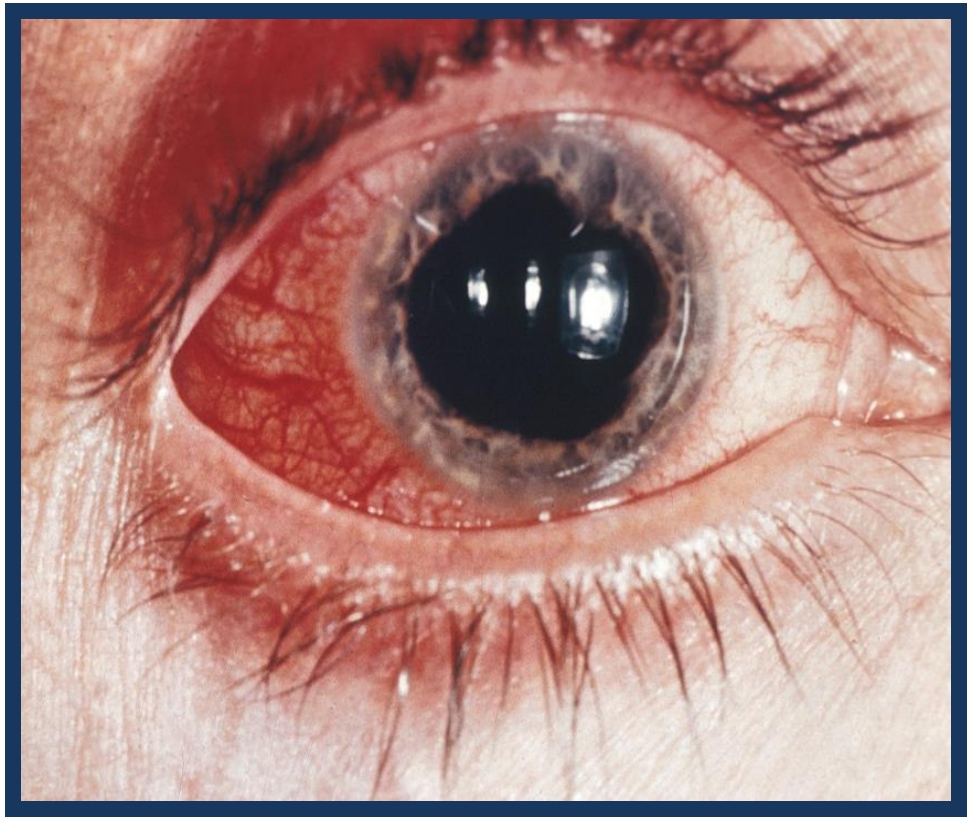
Note that a 2 lines scenario was given

Ankylosing spondylitis: ankylosis, lumbar spine

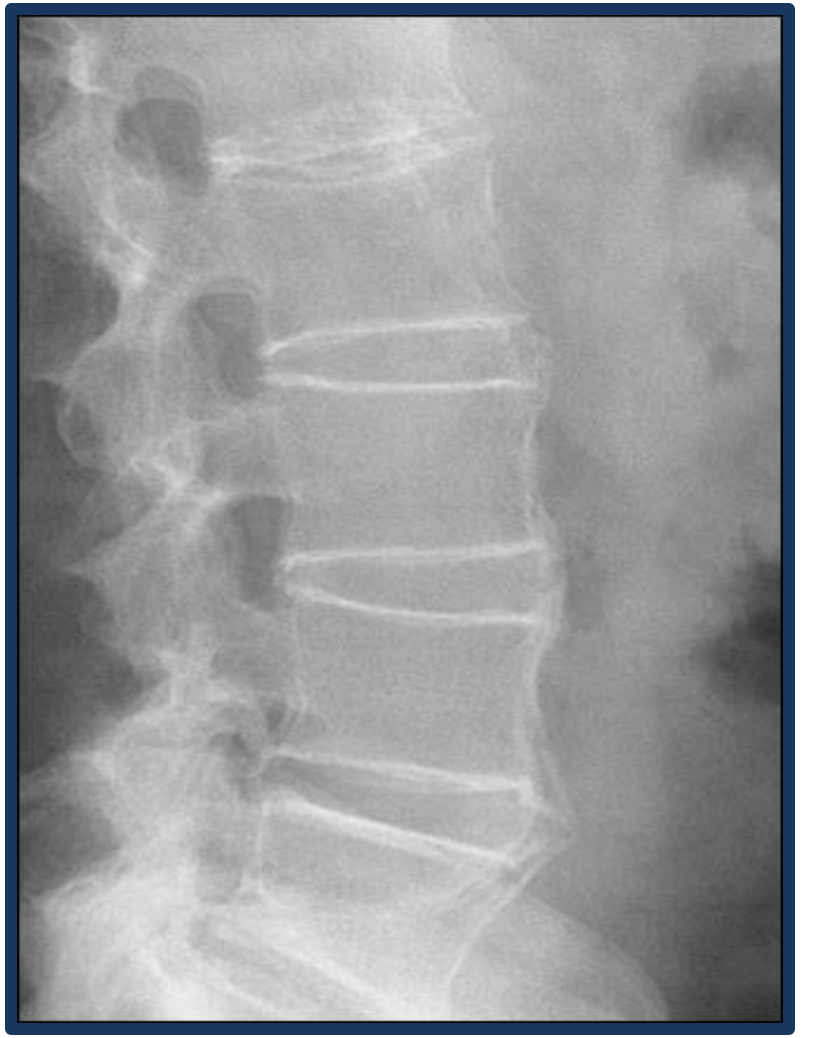




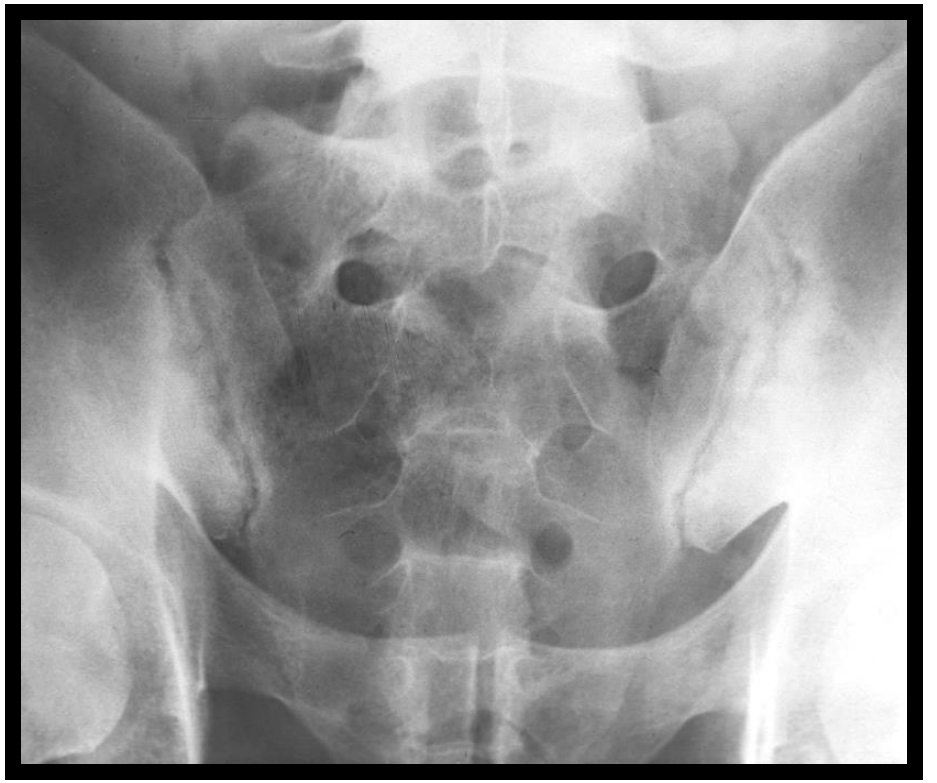
Ankylosing spondylitis: iridocyclitis with synechiae



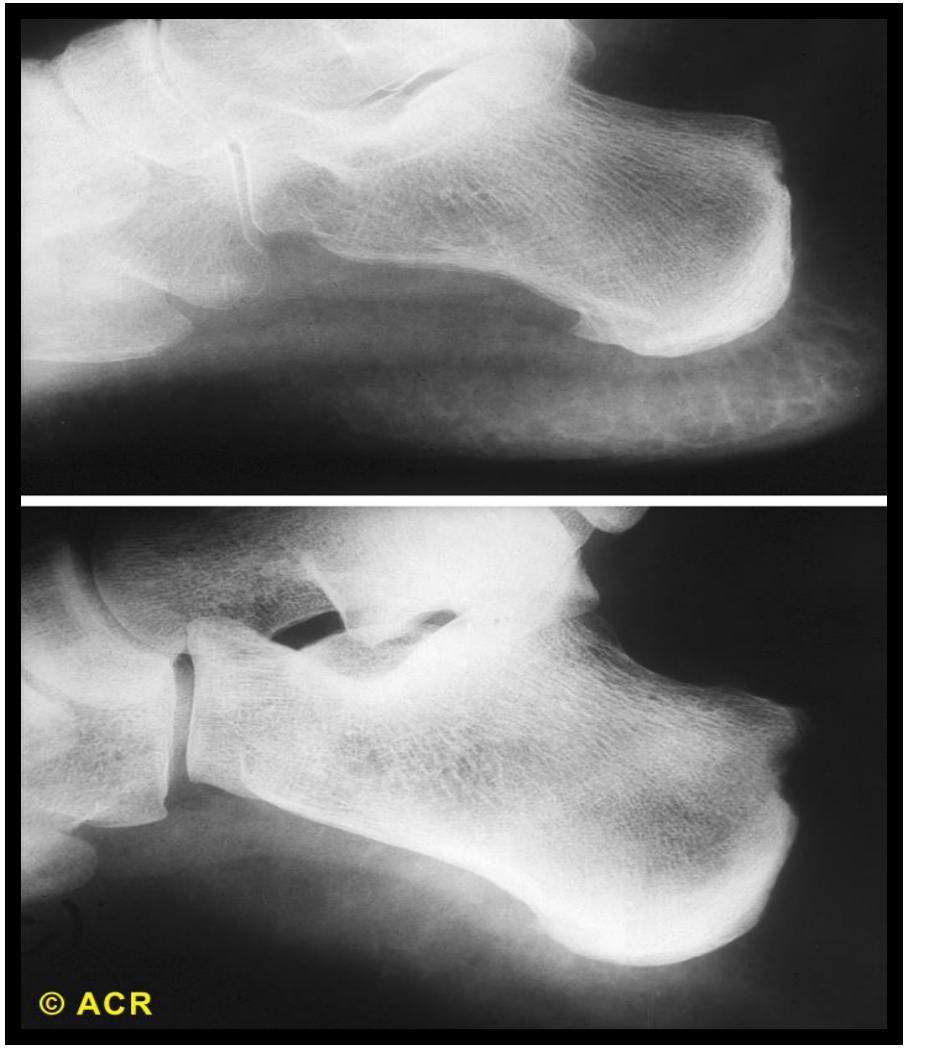
Bamboo spine of ankylosing spondylitis



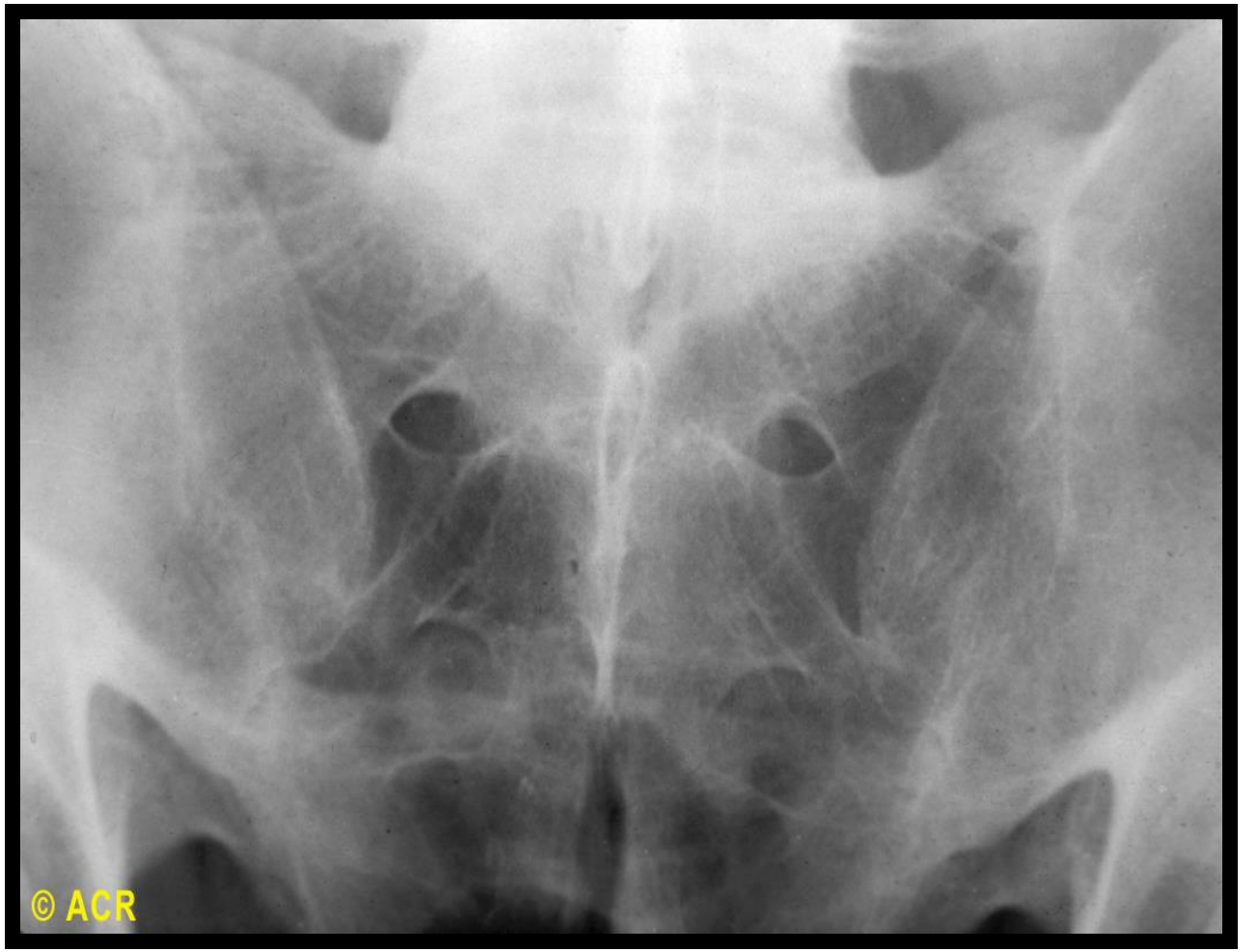
Ankylosing spondylitis: early sacroiliitis (radiograph)



Ankylosing spondylitis: calcaneal erosion and spur (radiographs)

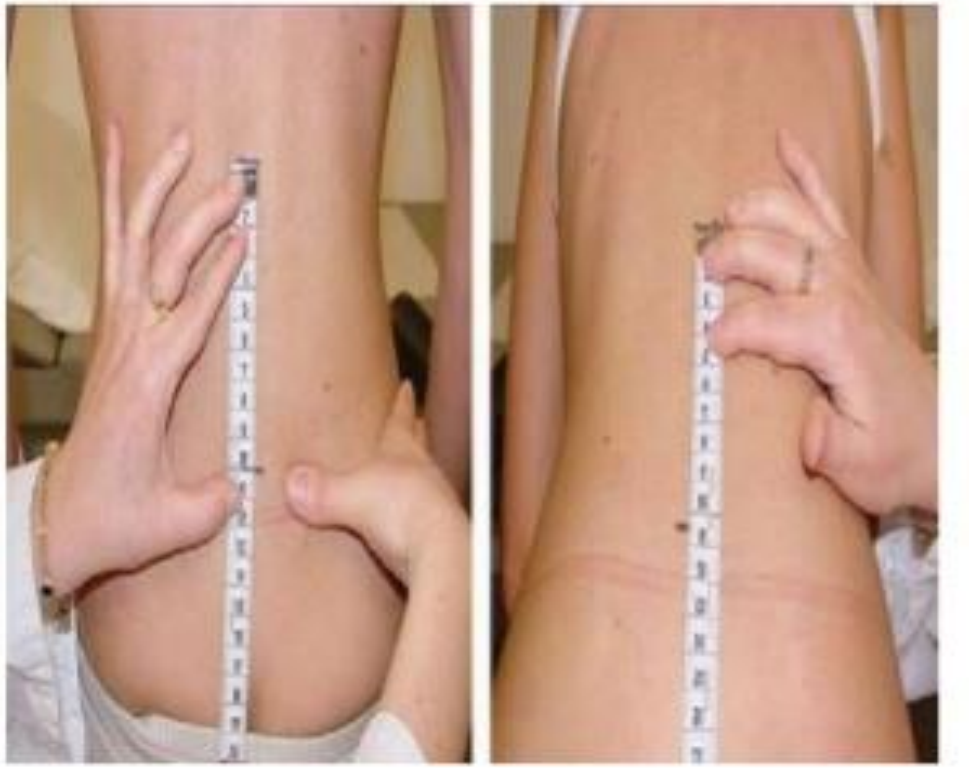


- **Ankylosing spondylitis: advanced sacroiliitis** (radiograph)
- The sacroiliac joints are almost completely obliterated. Bony trabeculae cross the residual sacroiliac joint space. There is no gross sclerosis at this time. A moderate degree of osteopenia is present.



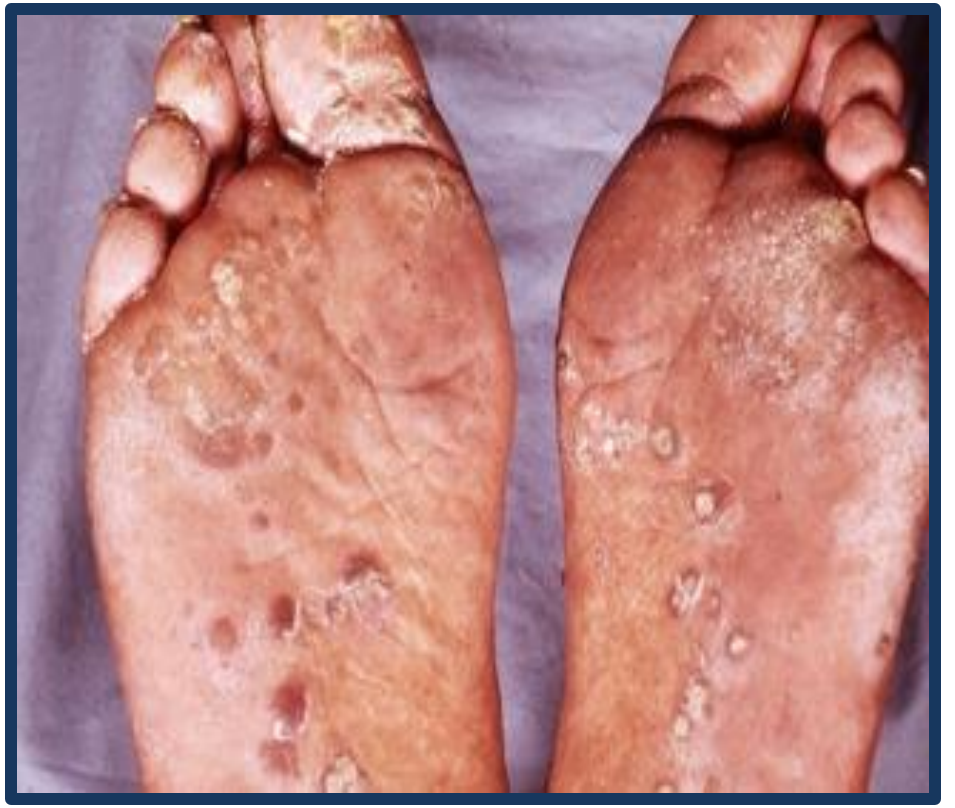
Schober test

- Patient standing upright
- Two marks are made on the patient's back: one at the level of the sacral dimples (at the fifth lumbar spinous process) and the other 10 cm above.
- The patient then bends forward as far as possible (ie, attempts to touch toes with knees extended), and the distance between the two marks is again measured.
- Normally the overlying skin will stretch to 15 cm
- Values less than this can be indicative of reduced lumbar mobility.

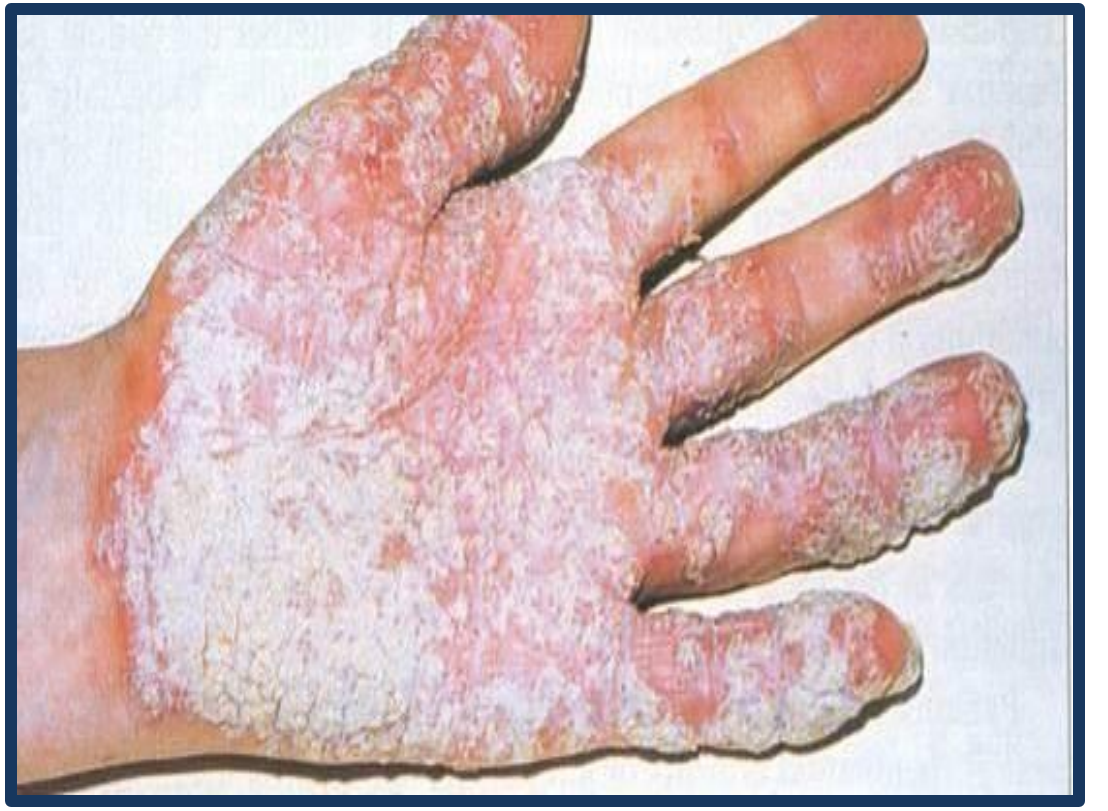


Which is seen in ankylosing spondylitis

Keratoderma balanorrhagicum, seen in seronegative arthropathies



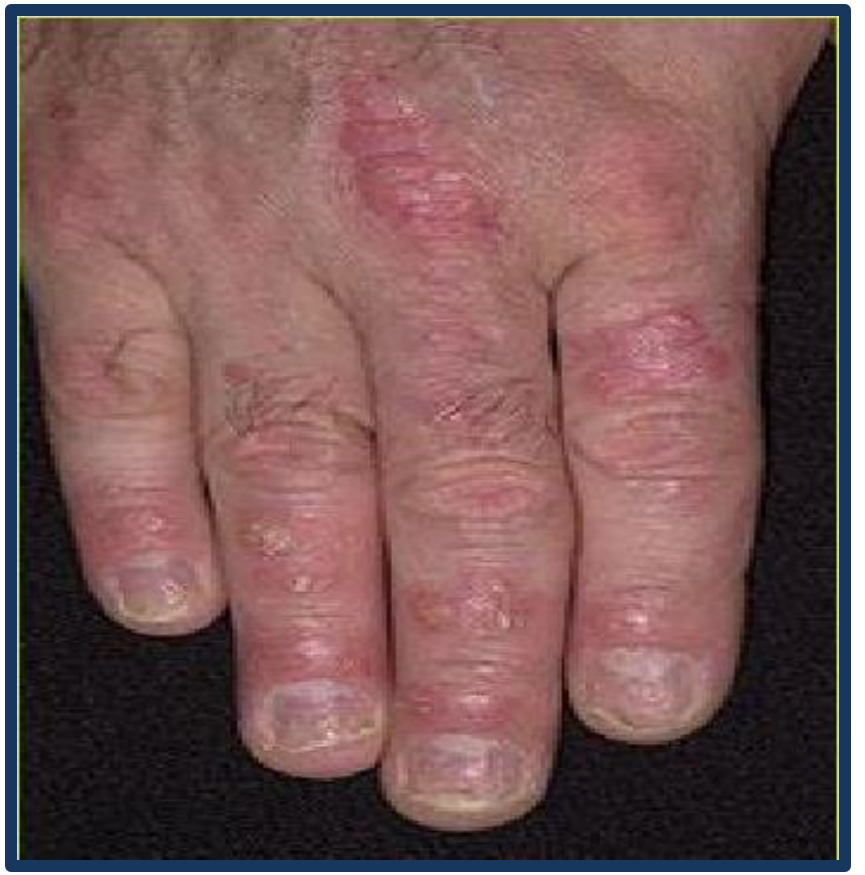
Keratoderma balanorrhagicum, seen in seronegative arthropathies



5. Psoriatic Arthritis

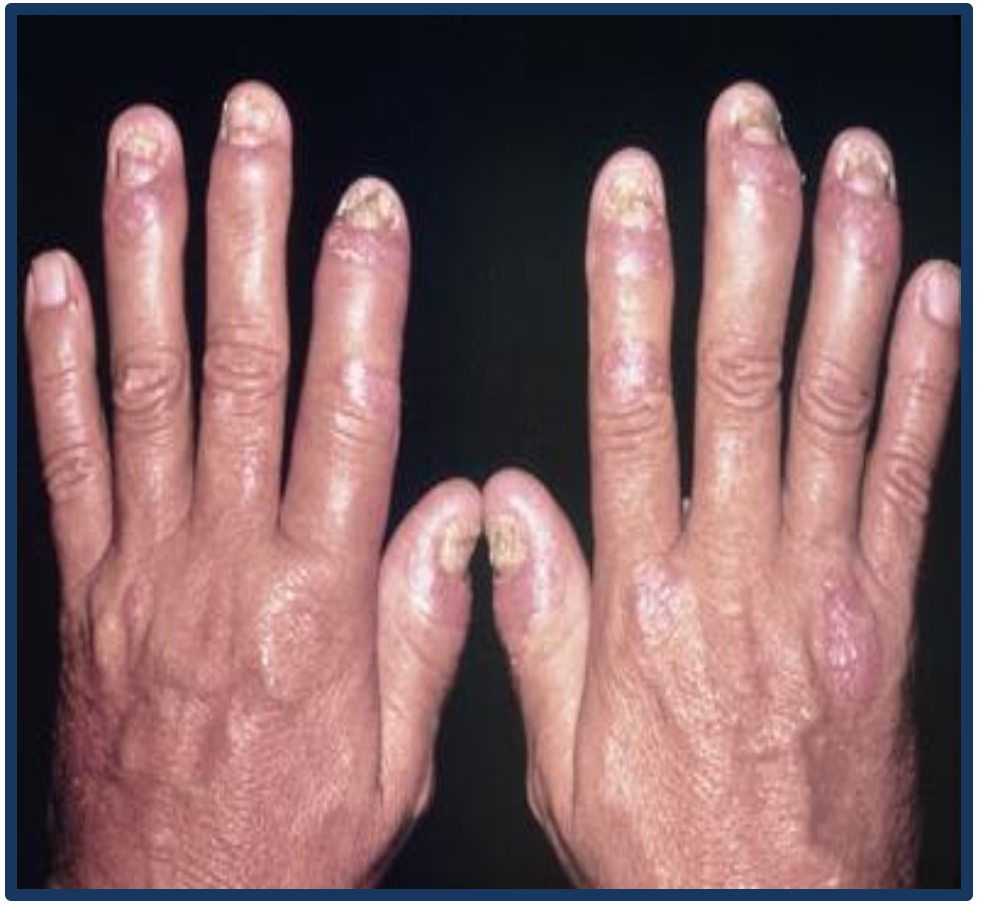
Q: This patient also has non itchy scaly rash on both knees, what's your diagnosis?

Psoriatic Arthritis.



Q: What is the finding in this picture?

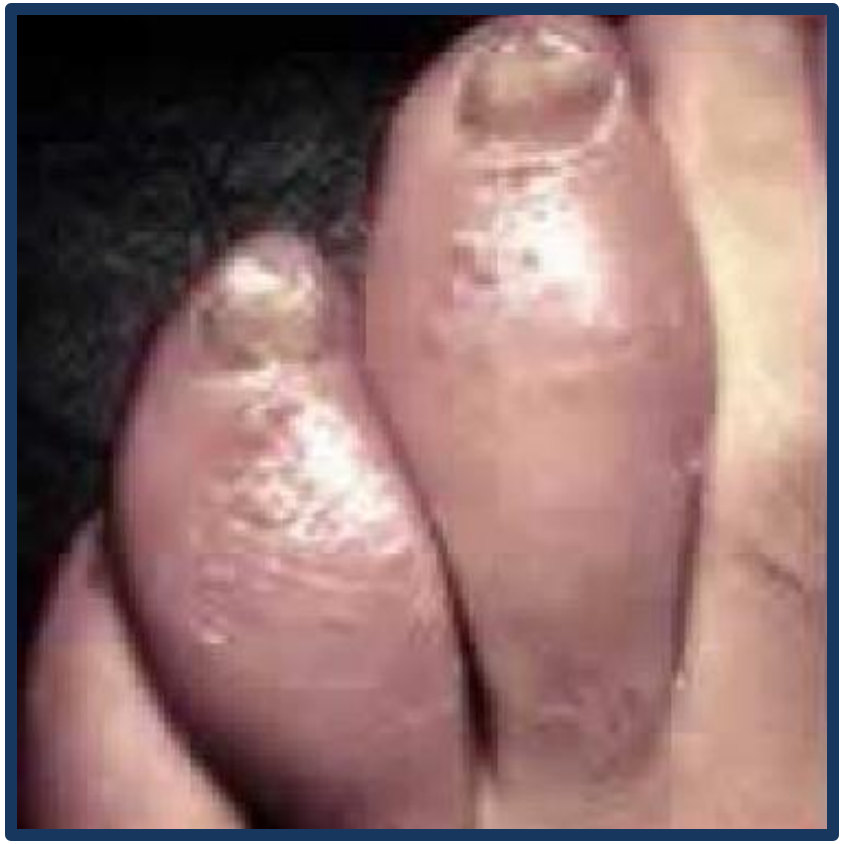
Dactylitis (sausage fingers)



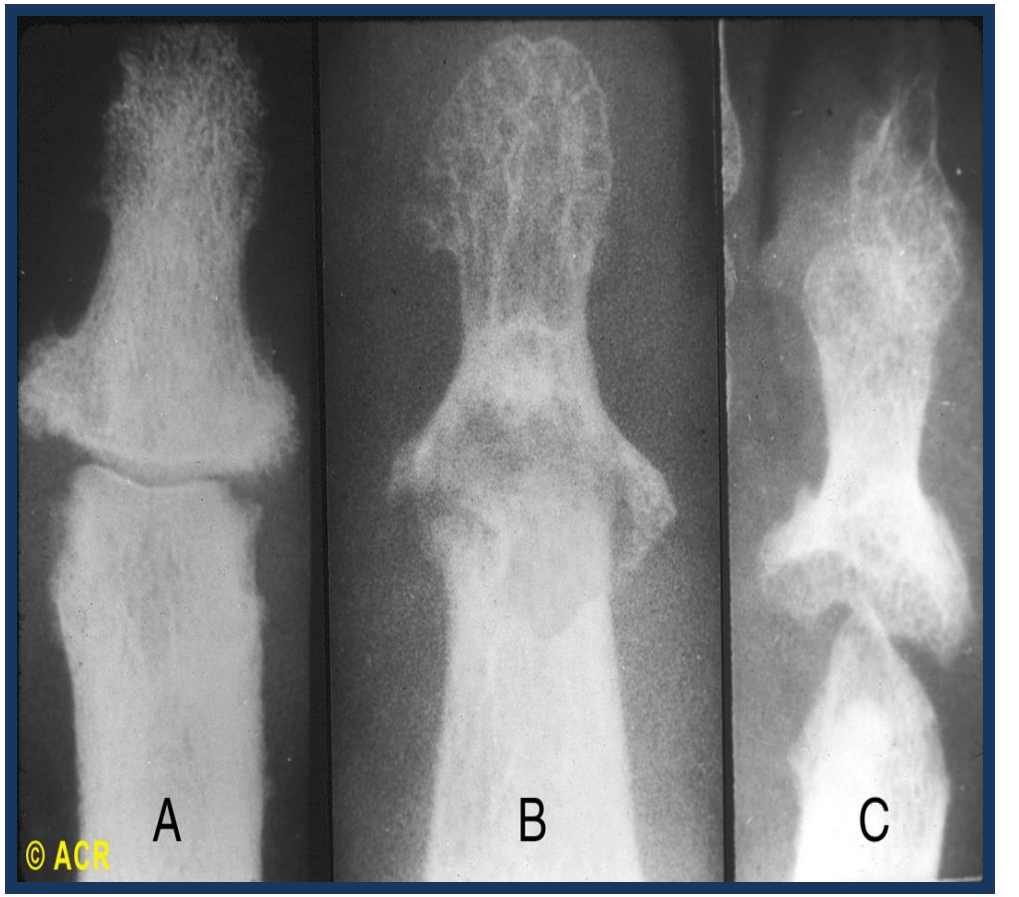
Q: In which disease could we see this lesion?

Psoriatic Arthritis

Sausage digit and rash



Psoriatic arthritis: progressive joint changes (radiographs)



Q: Dx?

Psoriatic arthritis

Nail lesion?

Onycholysis nail pitting

Affected joint?

I think it was DIP

Q: This patient also has non itchy scaly rash on both knees, what's your diagnosis?

Psoriatic arthritis

Q:This patient came also with itchy non-scaly rash on both knees, what is the sign you see on the nails and what is the diagnosis?

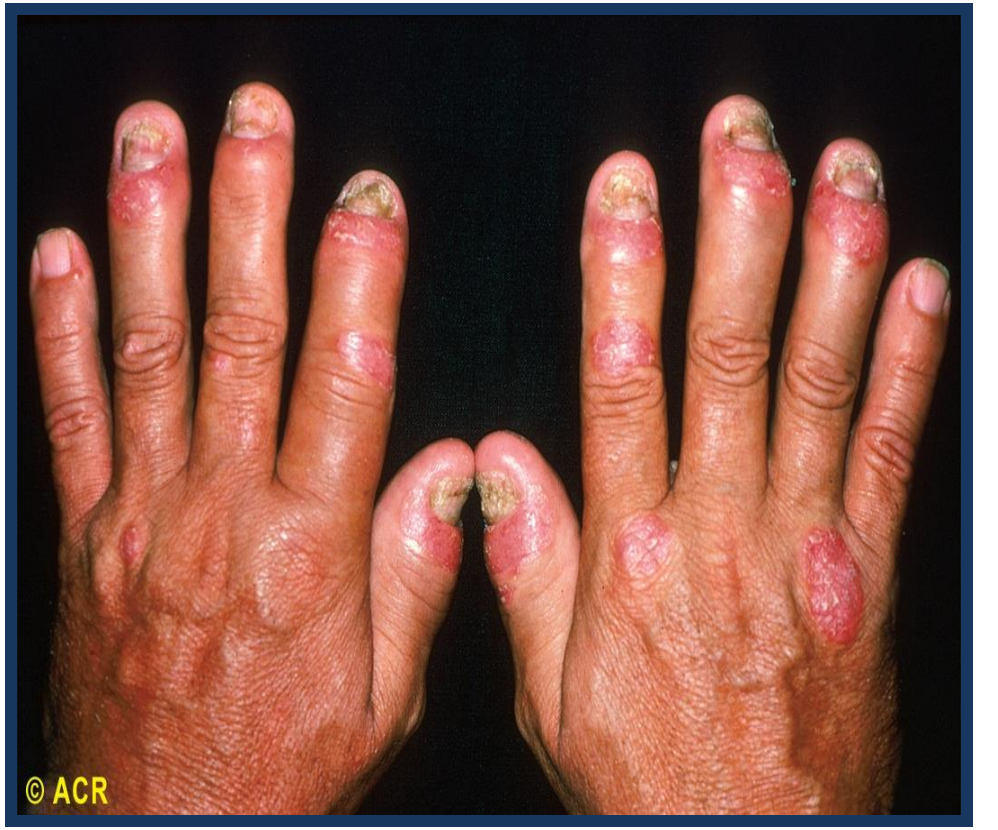
Pitting Nails

Psoriatic arthritis

Psoriatic arthritis

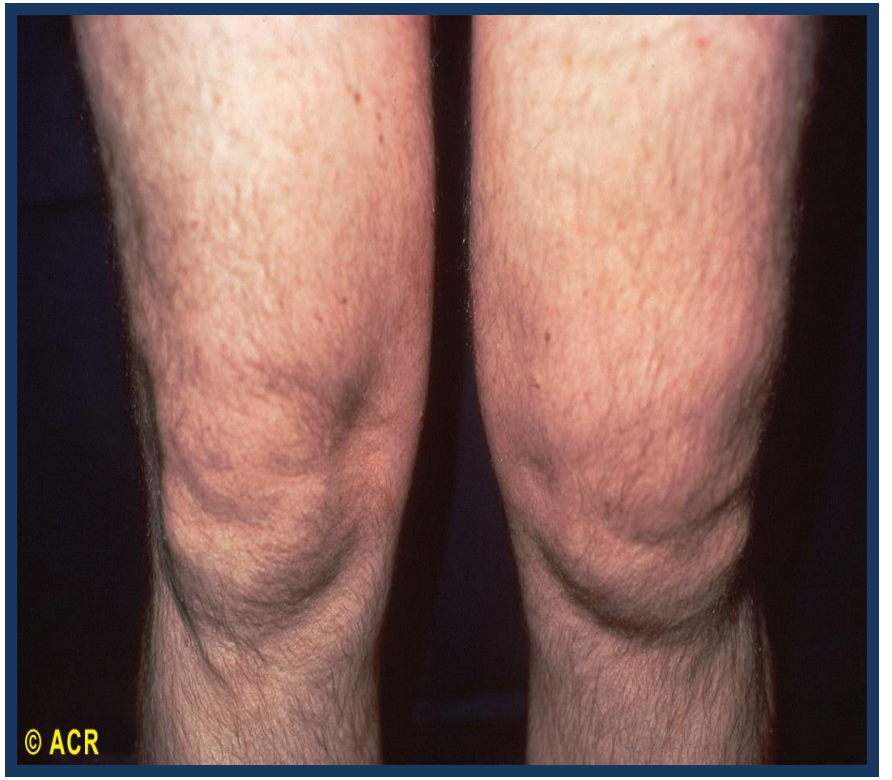
Predominant involvement of Dip's

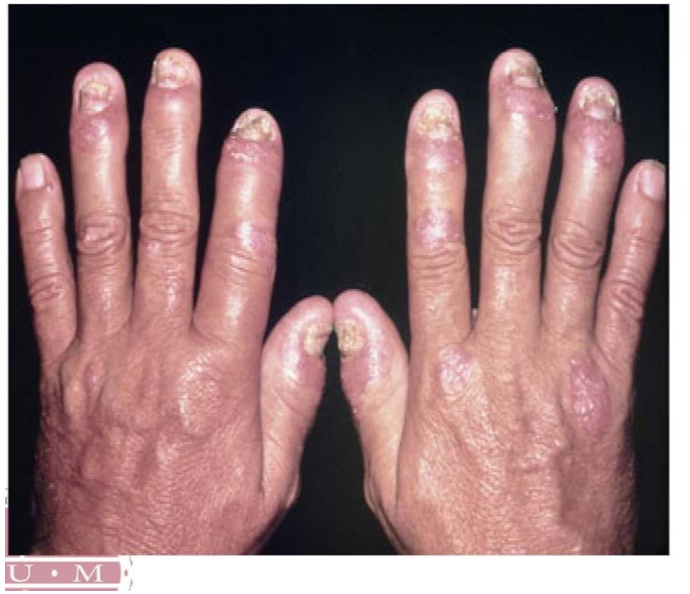
8-16%.

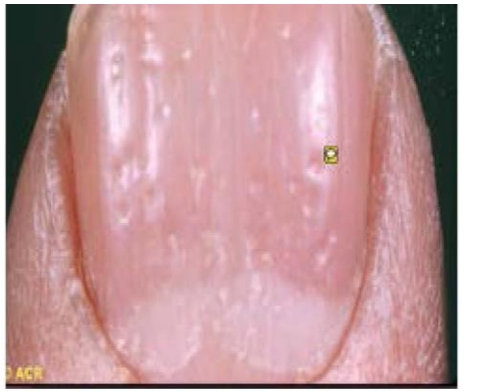


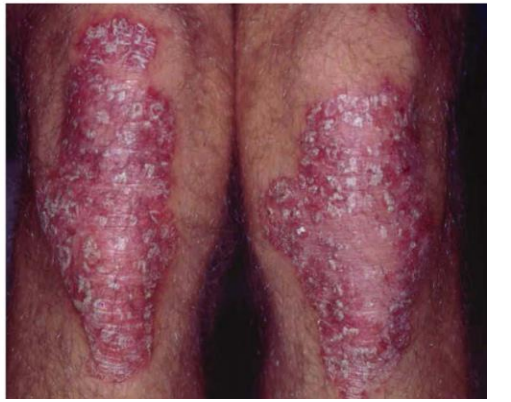
Psoriatic arthritis: asymmetric synovitis, knees

Nail pitting
Sausage digit
Non itchy scaly rash

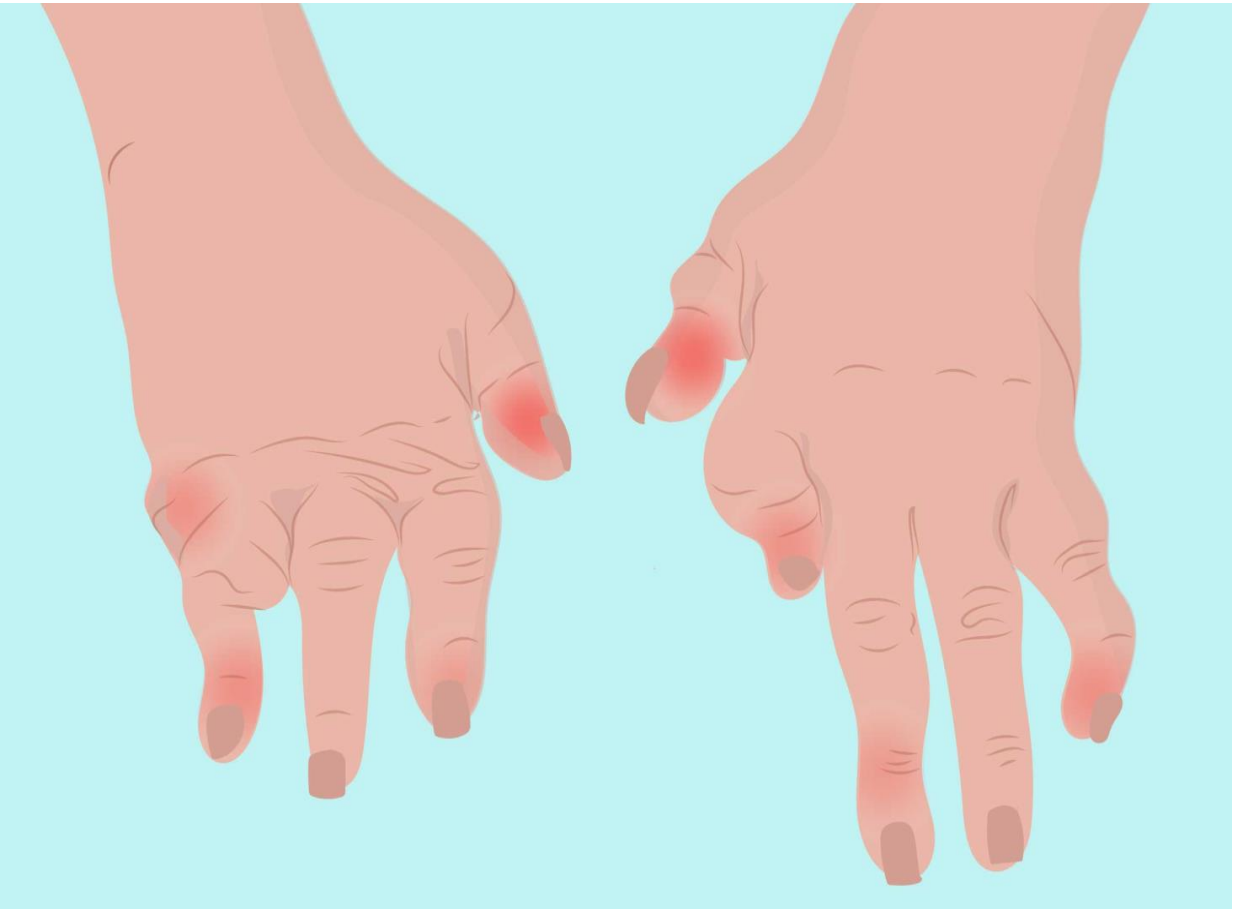








Arthritis mutilans telescoping



6.Gout

Q: This patient was prescribed an antihypertensive medication.

a) What is the diagnosis?

Acute gouty attack -podegra

b) What was the drug?

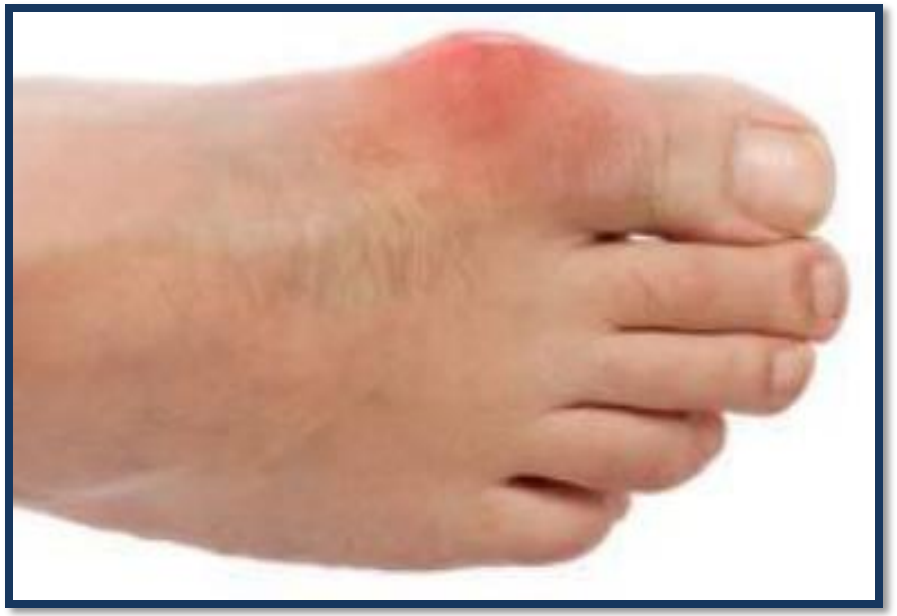
Thiazide

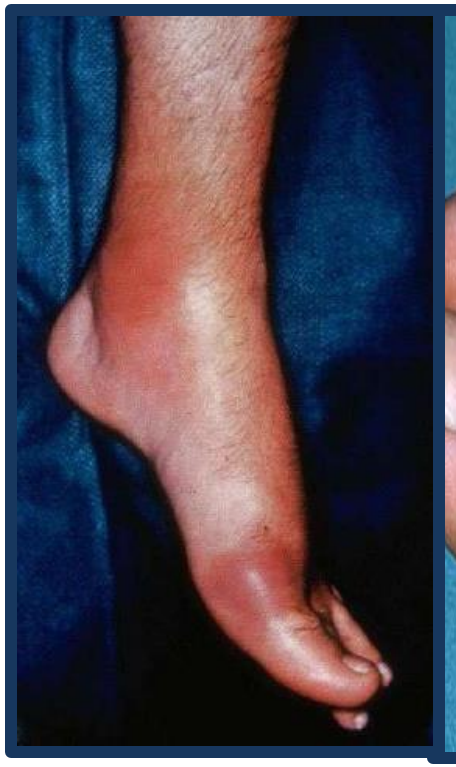
Q:1- Diagnosis?

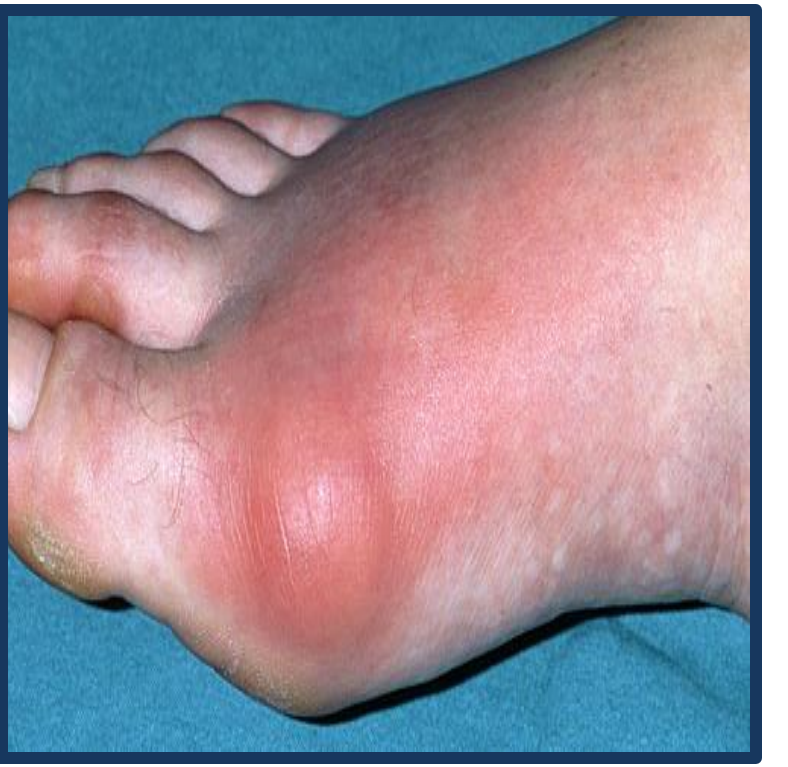
Gout

2- A blood test to confirm it?

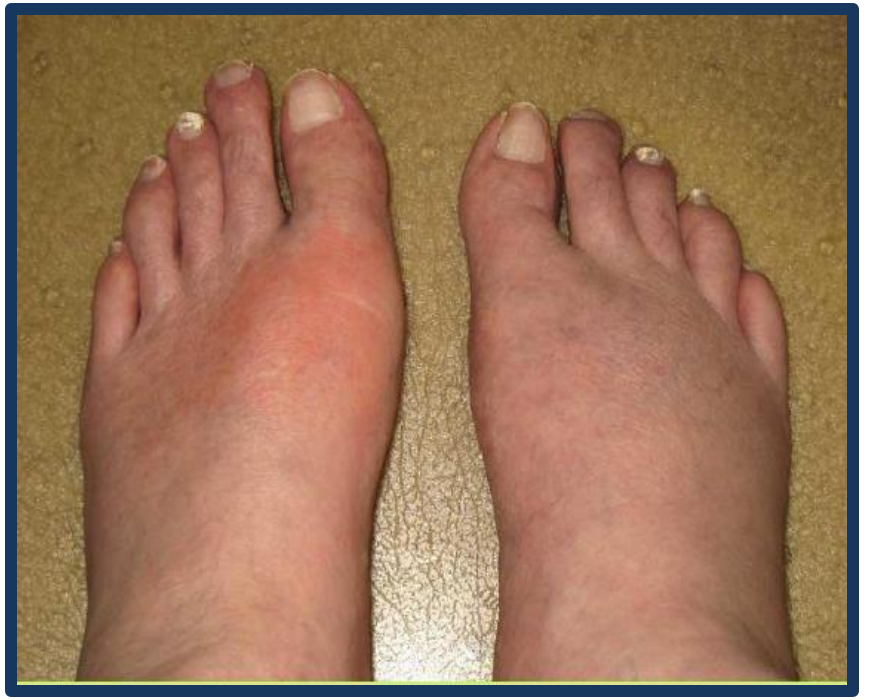
Serum uric acid level (not sure, they may appear normal even during attacks)



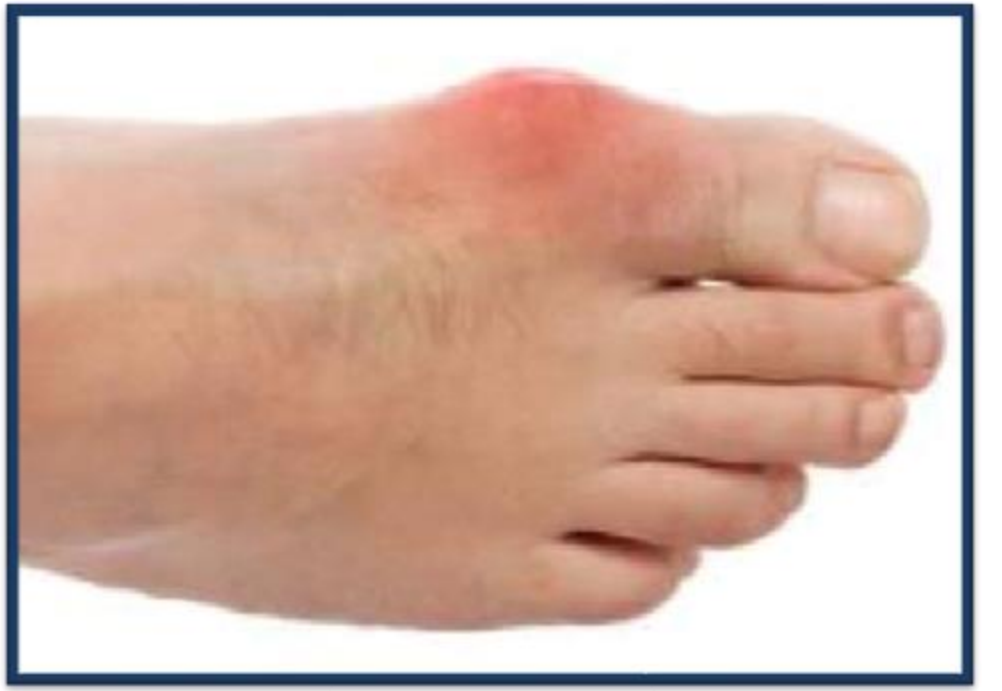




Q:This patient presented with sudden onset pain in his big toe.
A-What is the diagnosis?
Gout (Acute gouty arithritis)
B-Mention a line of management
Steroids, NSAIDS, …

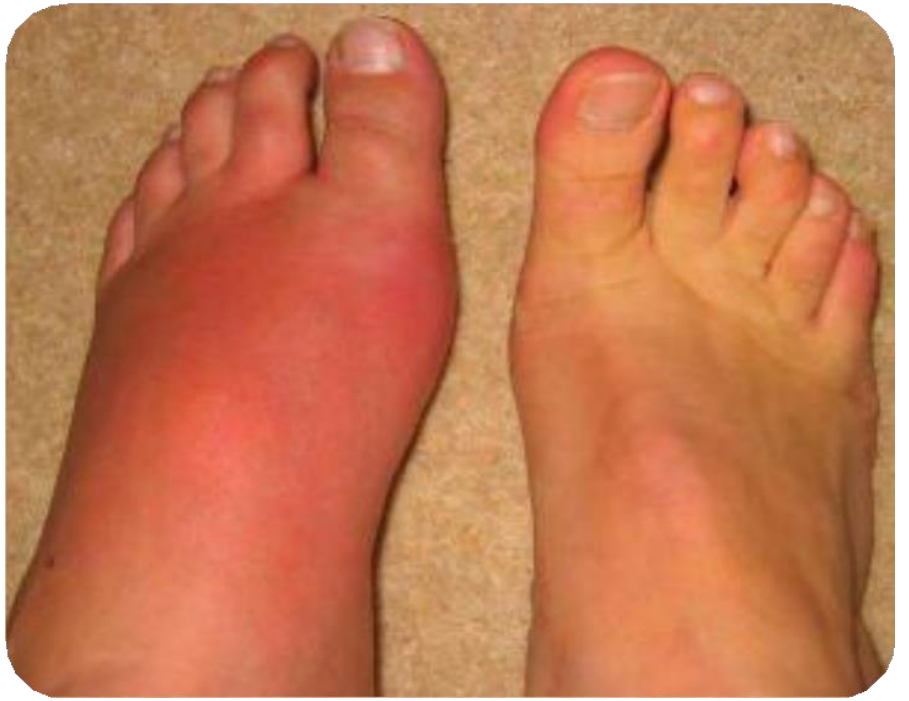


Q: DM patient started taking thiazide recently, What is the blood test you want to do for him?
Serum uric acid levels



Q:A patient recently diagnosed with hypertension was started on diuretics, presents to the ER with severe big toe pain, it's the third attack of such pain, what is your diagnosis?

Acute Gouty Arthritis



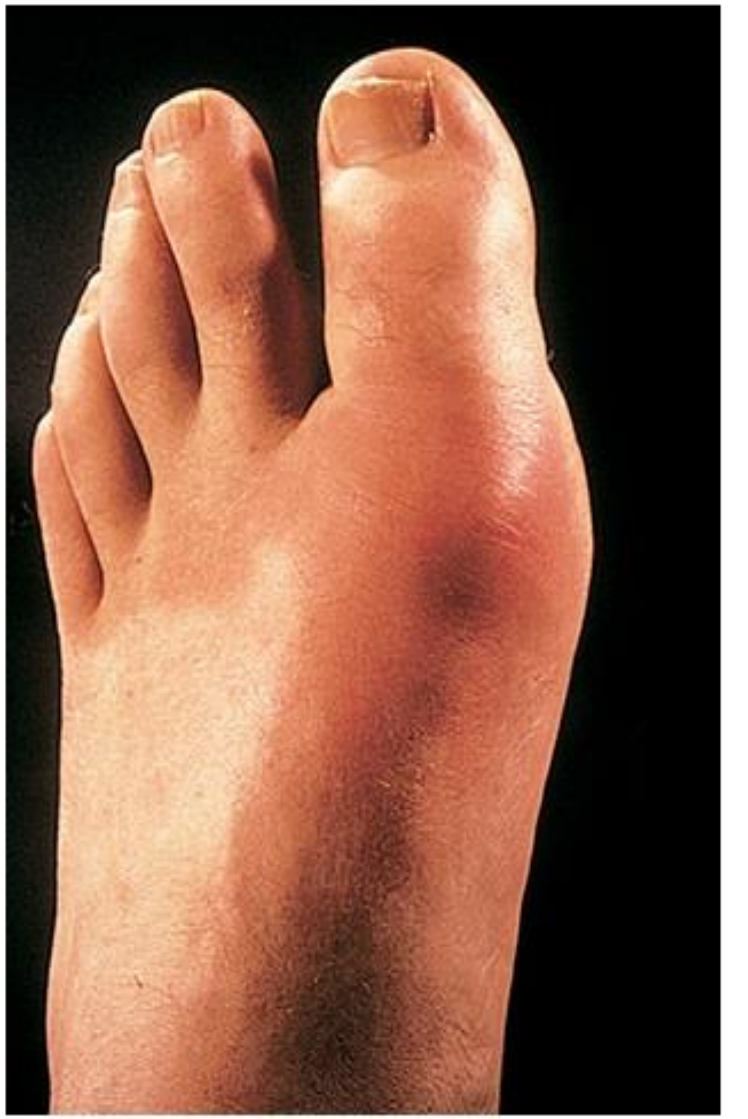


Fig. 25.24 Podagra. Acute gout causing swelling, erythema and extreme pain and tenderness of the first metatarsophalangeal joint.

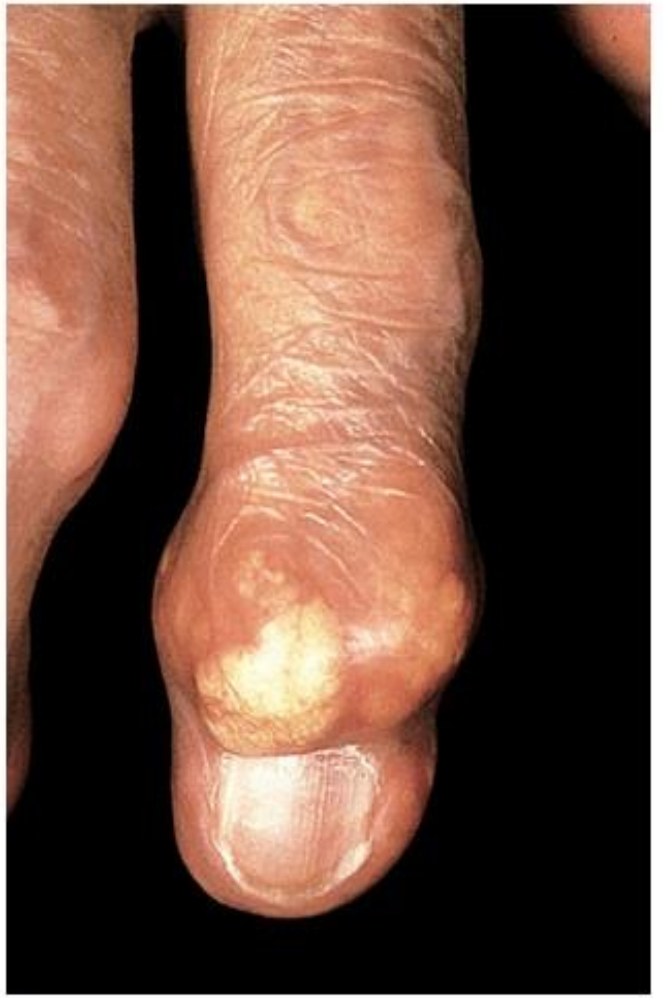


Fig. 25.25 Tophus with white monosodium urate monohydrate crystals visible beneath the skin. Diuretic-induced gout in a patient with pre-existing nodal OA.

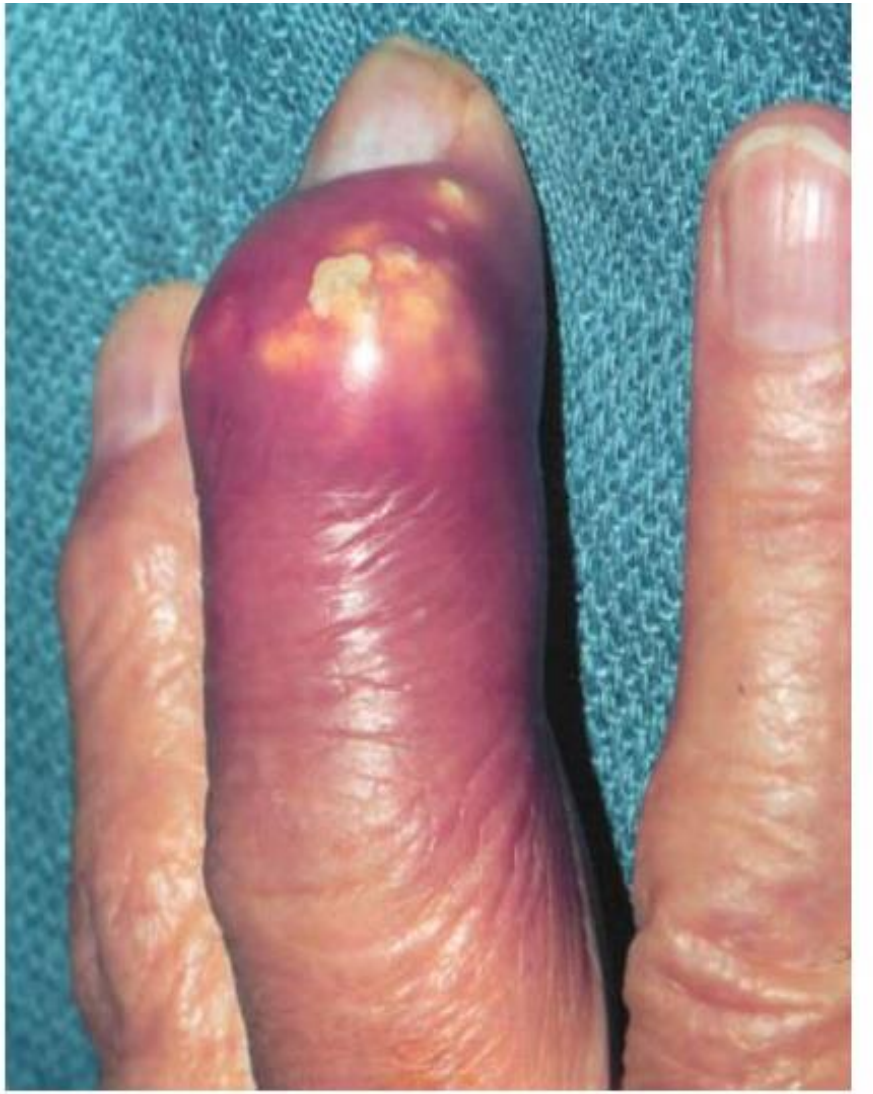


FIGURE 105-1 Acute gouty arthritis superimposed on tophaceous gout. (Reproduced with permission from Geiderman JM. An elderly woman with a warm, painful finger. West J Med. 2000;172(1):51-52.)

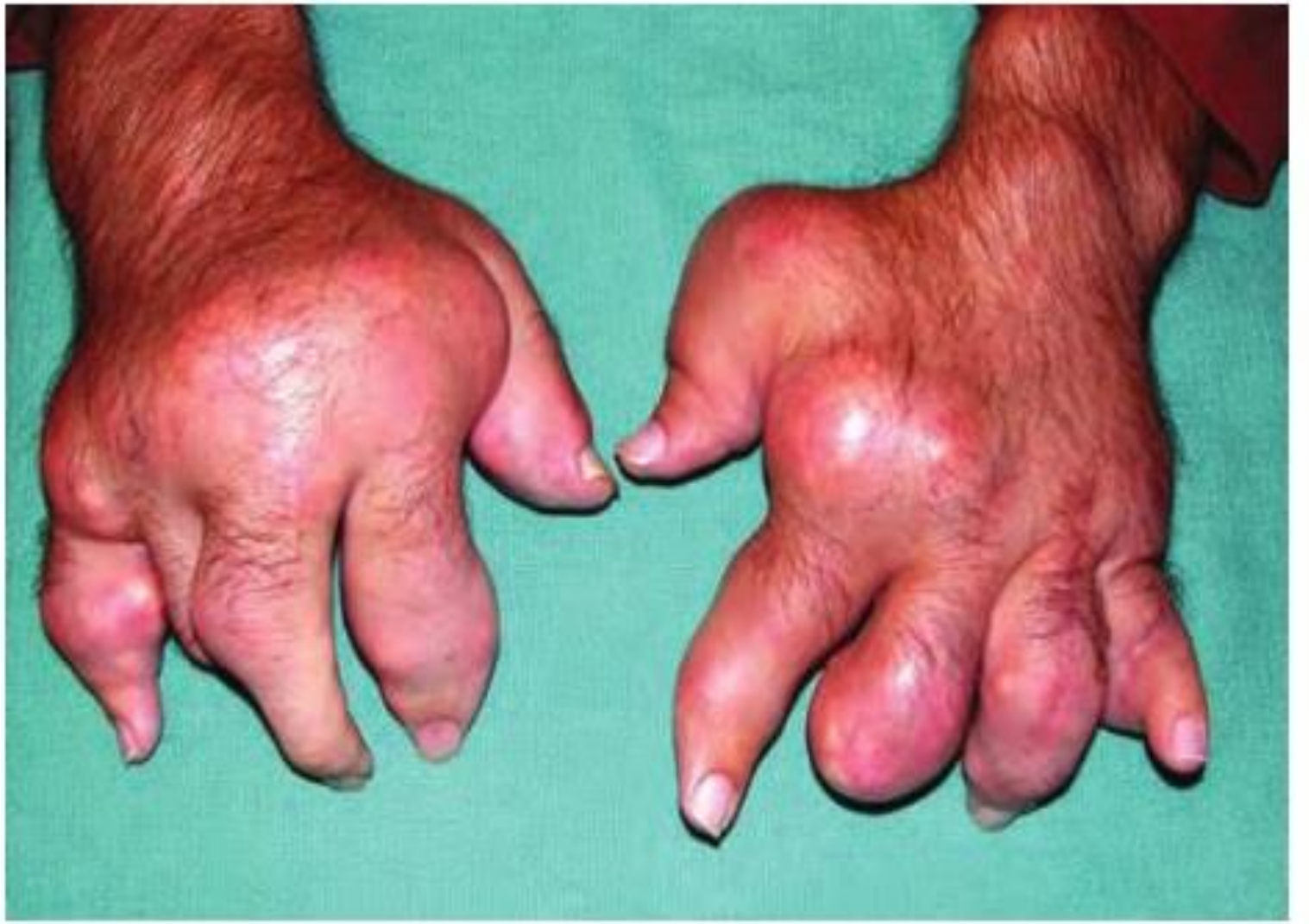


FIGURE 105-5 Severe tophaceous gout causing major deformities in the hands. (Reproduced with permission from Eric Kraus, MD.)

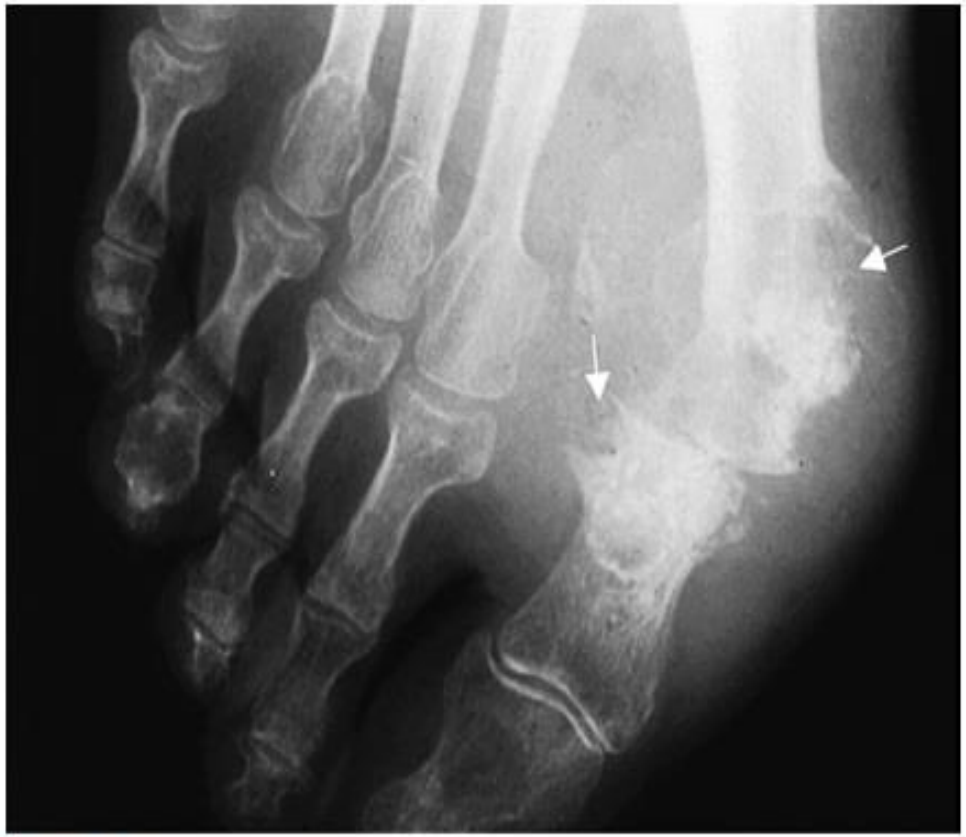


Fig. 25.26 Erosive arthritis in chronic gout. Punched-out erosions are visible (arrows), in association with a destructive arthritis affecting the first metatarsophalangeal joint.

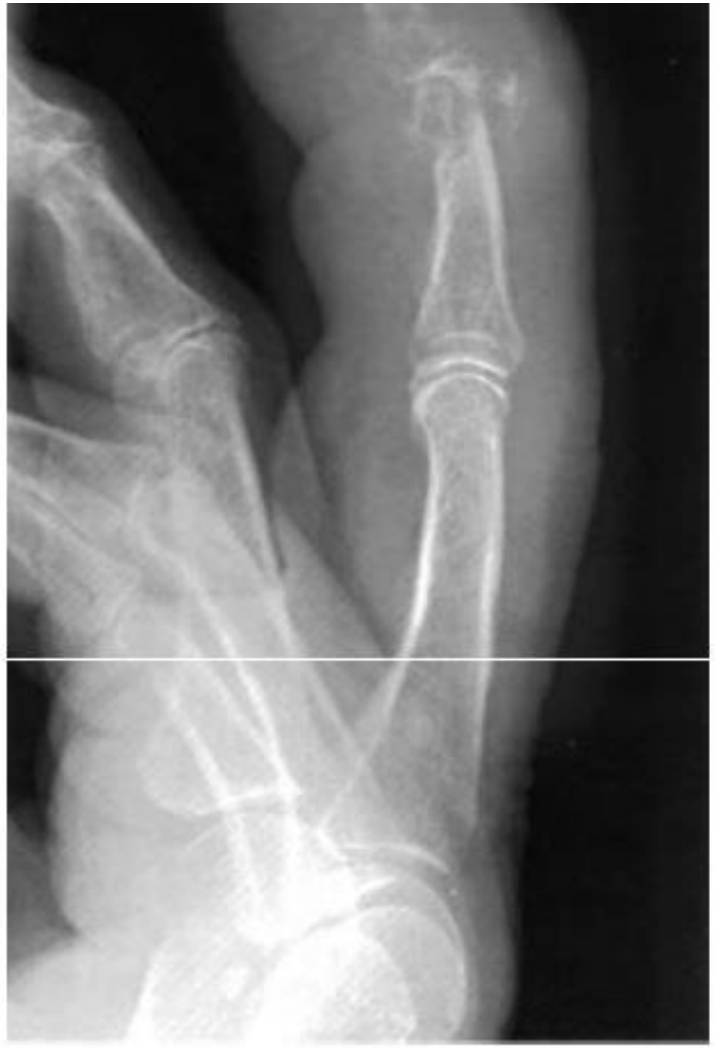


FIGURE 105-2 This X-ray of the finger in Figure 105-1 shows several tophi (monosodium urate [MSU] deposits) in the soft tissue over the third distal interphalangeal joint. Note the typical punched out lesions under the tophi. This is subchondral bone destruction. (Reproduced with permission from

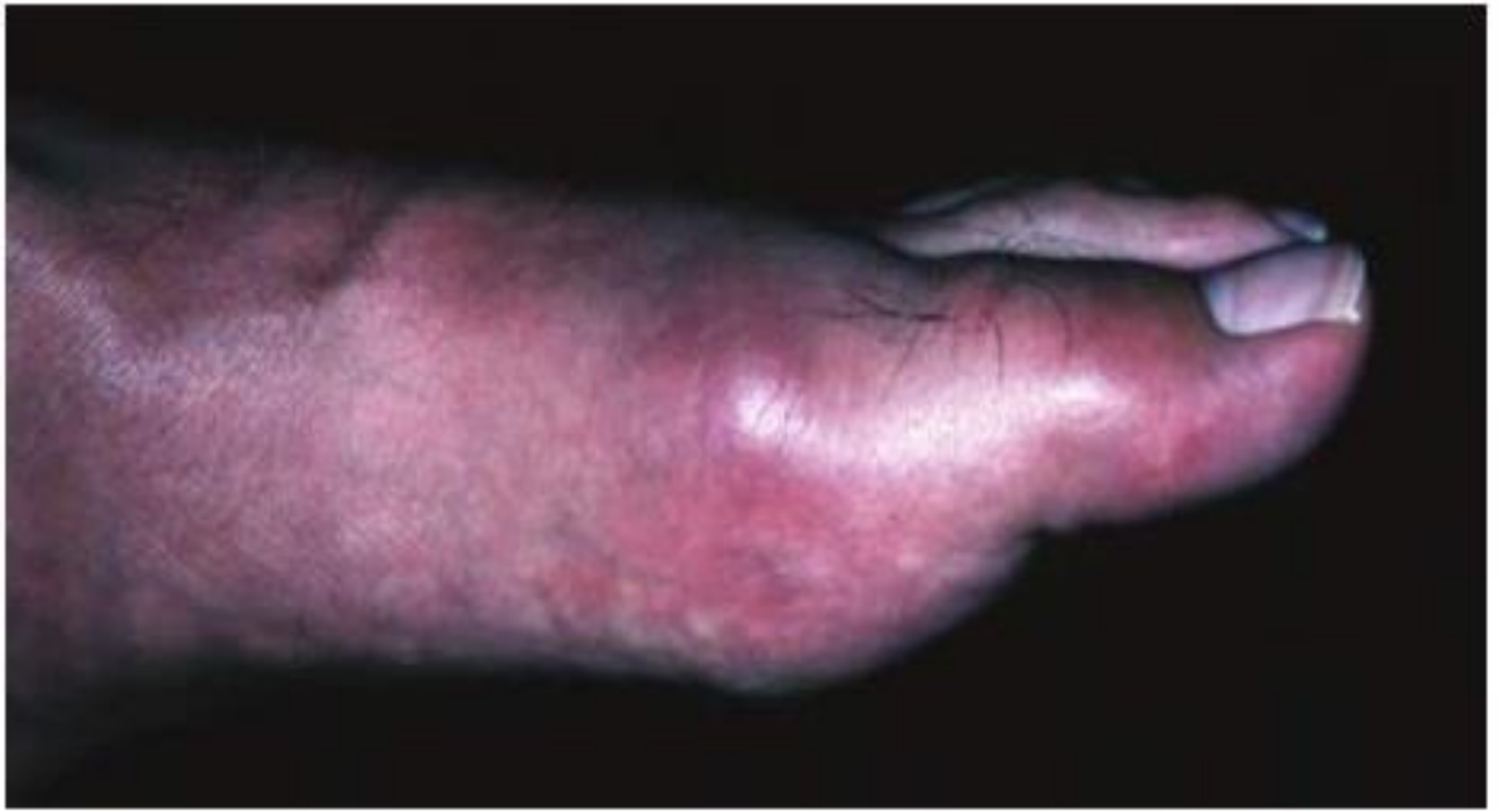


FIGURE 105-3 podagra. Typical inflammatory changes of gout at first MTp joint. (Reproduced with permission from Richard P. Usatine, MD.)

Q: A pt with hypertension (or DM) presented with right ankle swelling & pain. He had 2 previous similar conditions; one was in the same site, the other was on the left ankle. His CBC showed leukocytosis (WBC count = 10,000).

1- What is the most probable Dx?

Gout.

2- Mention another DDx.

Septic arthritis, Cellulitis, Pseudogout

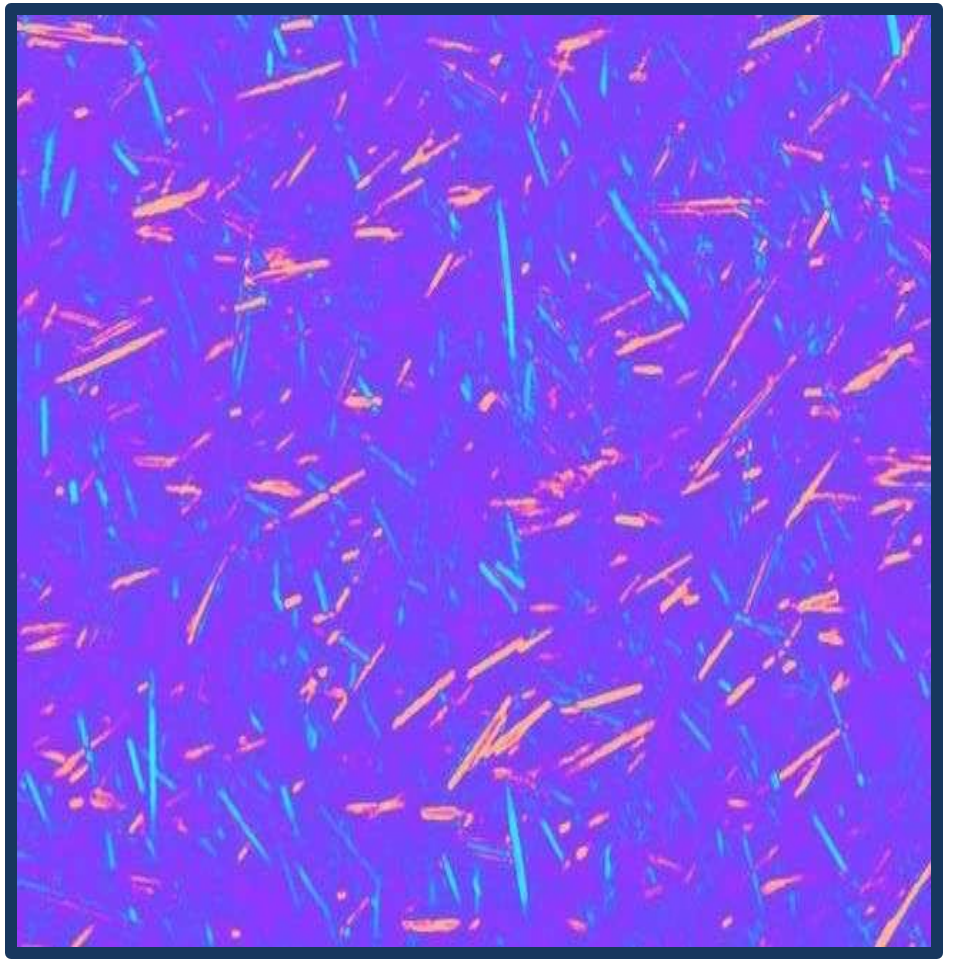
3-If a sample from the synovial fluid was aspirated,
what is your confirmatory test?

Identification of monosodium urate crystals under polarized light microscopy; they have a needle-like morphology & strong negative birefringence.

4-Mention 2 drugs for the treatment of the acute attack.

Steroids, NSAIDs, Colchicine.

Needle shaped monosodium urate crystals. Has a negative birefringence under polarized light.

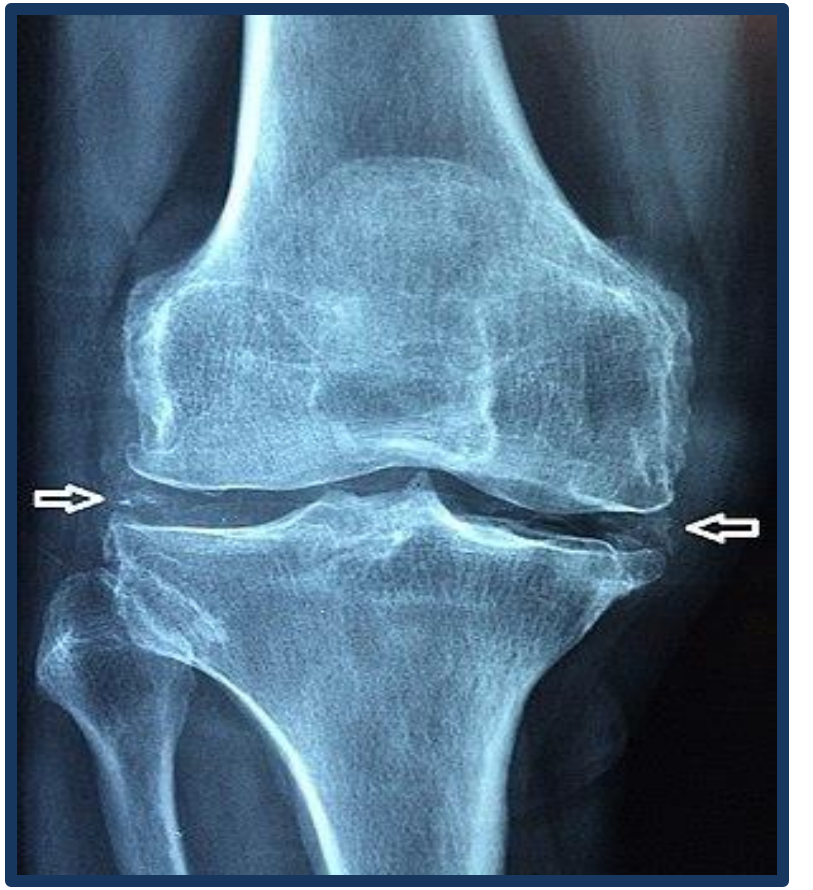


Q: What is the abnormality in this x-ray?

Linear calcification of the joints cartilage

Diagnosis?

Pseudogout (CPPD)



Q: What is the finding?

Weakly positive birefringence of rhomboidal crystals of calcium pyrophosphate dihydrate under polarized light

What is the diagnosis?

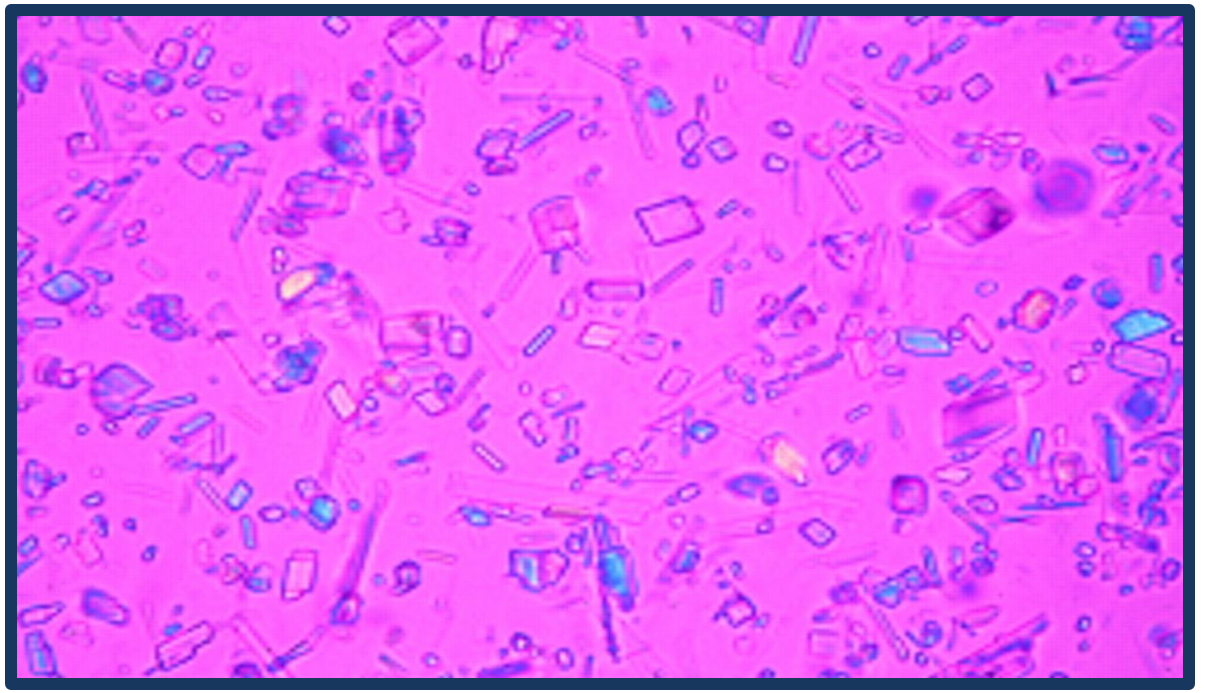
CPPD (calcium pyrophosphate dehydrate deposition disease)

What is the most commonly involved joint?

The knee joint

What is the treatment?

Same as gout. NSAIDS, Corticosteroids, colchicine.



7.Behcet's disease

7.Behcet's disease

*The pathological lesion is systemic peri-vasculitis

*HLA-B51 association

*Clinical feature for behcet disease:painful non-scaring oral ulcer,

Painful scaring genital ulcer ,uveitis or iritis(red painful eye),arthritis or (arthralgia), DVT(recurrent) , skin lesion (folliculitis, erythema nodosum , Pyoderma gangrenosum)

*diagnostic criteria: recurrent oral ulcer+ any two of the following :-

1-recurrent genital ulcer

2-ocular involvement (uveitis,iritis,optic neuritis)

3-skin lesion(as above)

4-positive pathergy test

Pathergy test :pricking skin with needle or intra-dermal normal saline injection and wait 48 hrs if there is skin reaction it is +ve

*the only serious complication of behcet's disease is blindness

*investigation : CBC,ESR,CRP, dx is clinical

*Tratment of behcet disease :

1-topical glucocorticoid for oral ulcer(soluble prednisolone mouth wash)

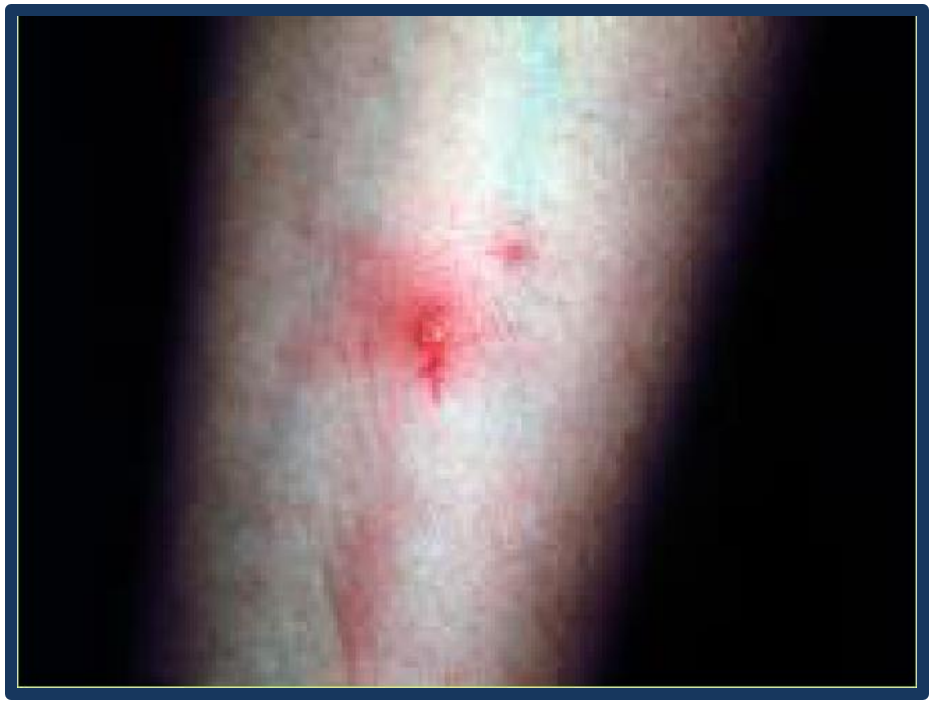
2-colchicine for arthritis and skin lesion

3-systemic glucocorticod and azathioprine(immunosuppresnt) for uveitis

4- thalidomide for resistant oral and genital ulcer

*DDX of behcet's disease :- IBD(inflammatory bowel disease { UC and CD}) , SLE ,apthous stomatitis

Q: This 23-year old patient developed this skin lesion after a needle prick.
A-What is your diagnosis?
Behcet's disease
B-Mention two clinical manifestation of this disease.
Recurrent oral and genital ulcers
C- what is the name of the test:
pathergy test

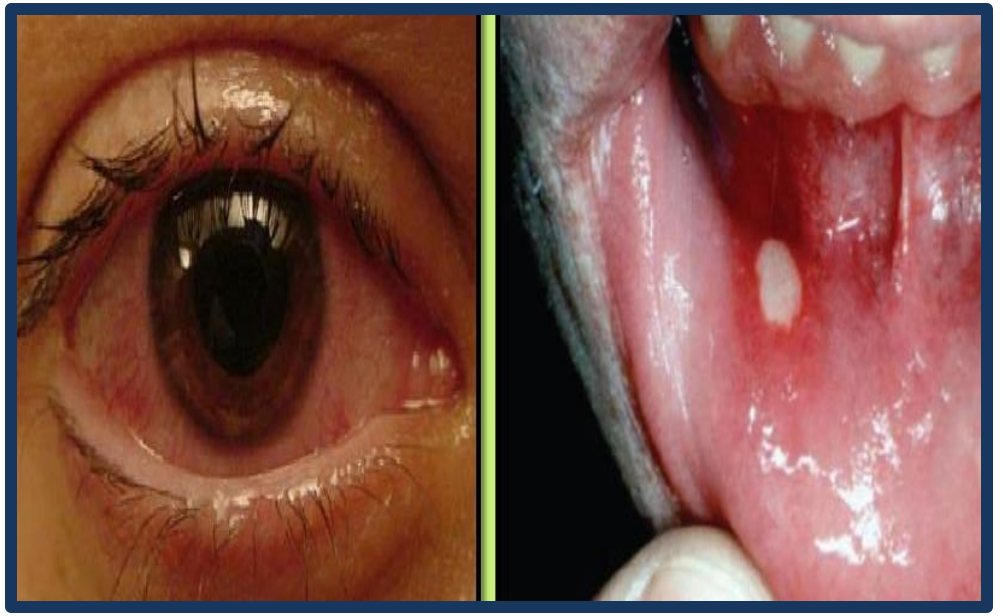


Q: This patient had this mouth lesion, and we did this test for him
What's the name of the test?
Pathergy test.
What's your diagnosis?
Behcet'sdisease.

Q: A 25 YO non-smoker female presented to the ER with bloody diarrhea, mixed with mucus & tenesmus and with this. Mention 2 DDx?

A. Behchet's disease .

B. IBD.



Q: 35 this patient has oral ulcer, arthritis and recurrent DVTs what's your diagnosis?

behcet's disease

mention one complication of this disease :

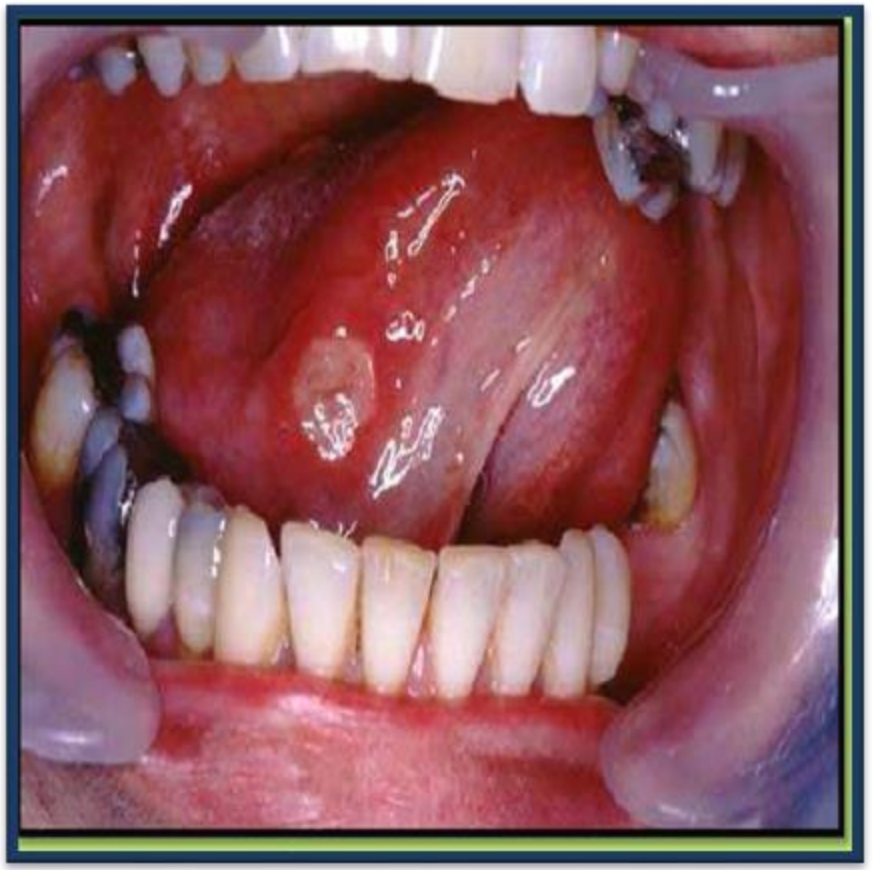
blindness

Q36: A young male who have this lesion with haemoptysis & other symptoms of DVT, what's your Dx?

Bahcet's disease.

Mention one of the ocular manifestation of this disease?

uveitis, iritis



Q: Pt came to your clinic complaining of painful red eye and joint pain

On history the pt had recurrent mouth ulcers

On examination you noticed this skin lesion

1-what is your dx ?

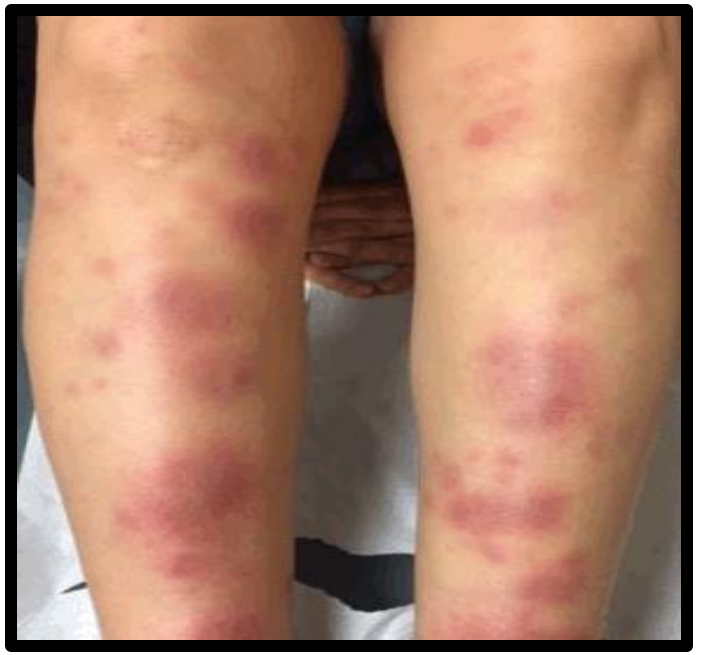
behcet disease

What is the name of this lesion ?

erythema nodosum

What is your management mention 3?

Systemic glucocorticoid (oral prednisolone) +Azathioprine +cochicine



Q: What is the most likely diagnosis in this 23 year old male pt with this painful lesion, red eyes and recurrent DVT?

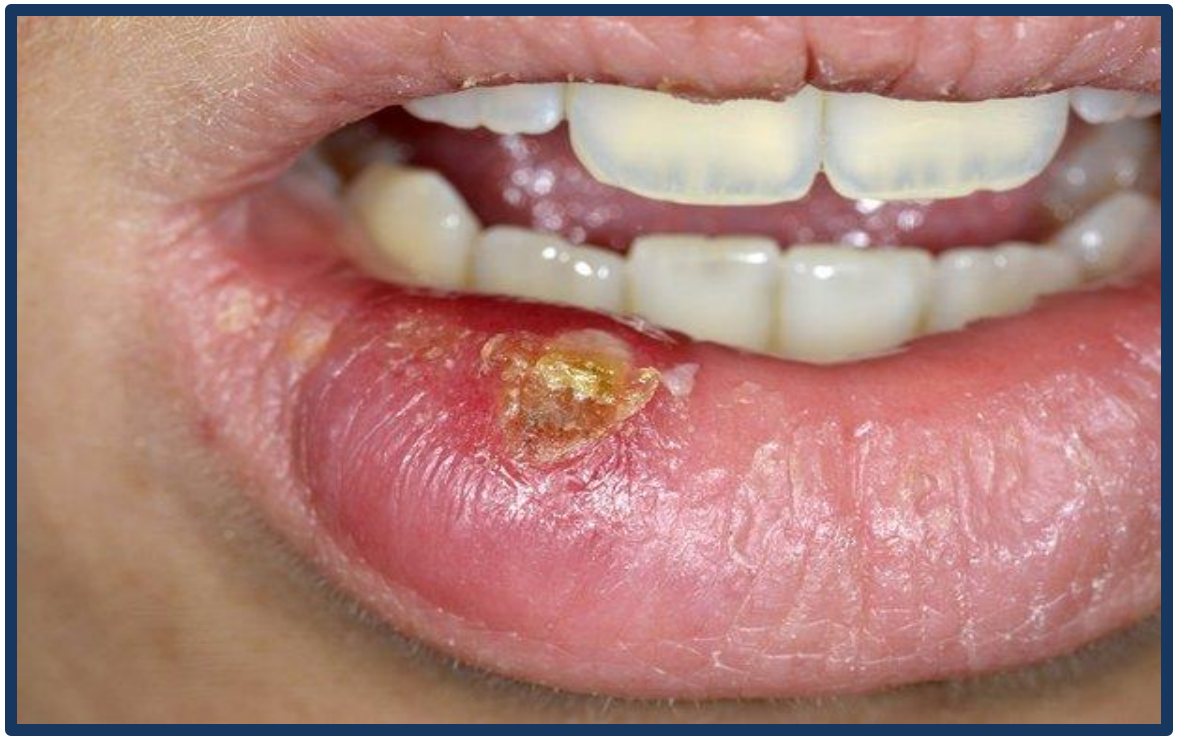
Bahcet's disease.

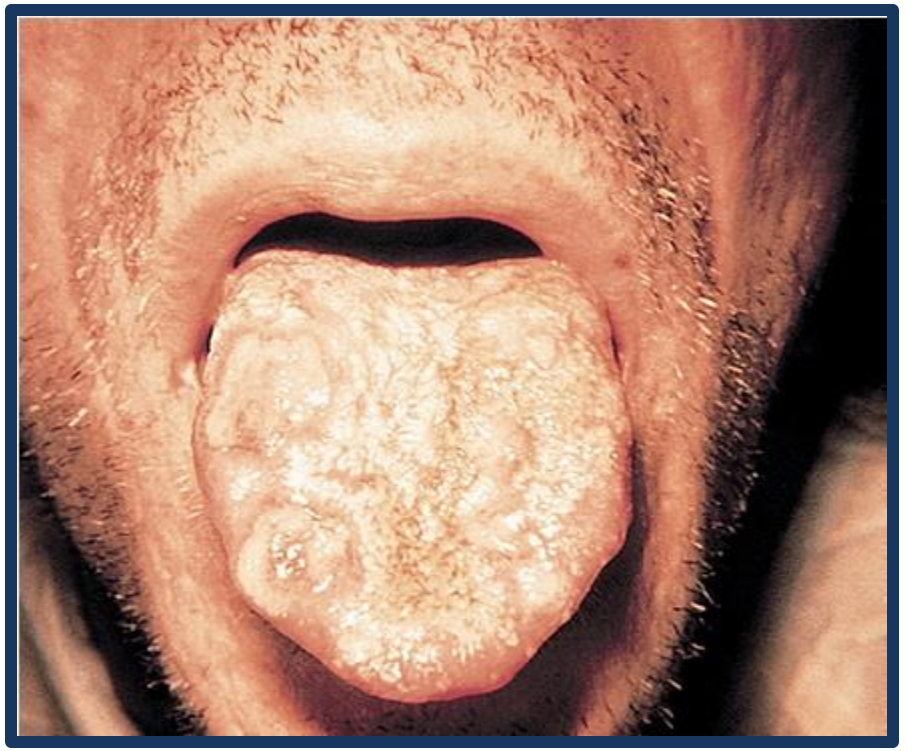
What is the HLA type associated with this disease?

HLA-B51

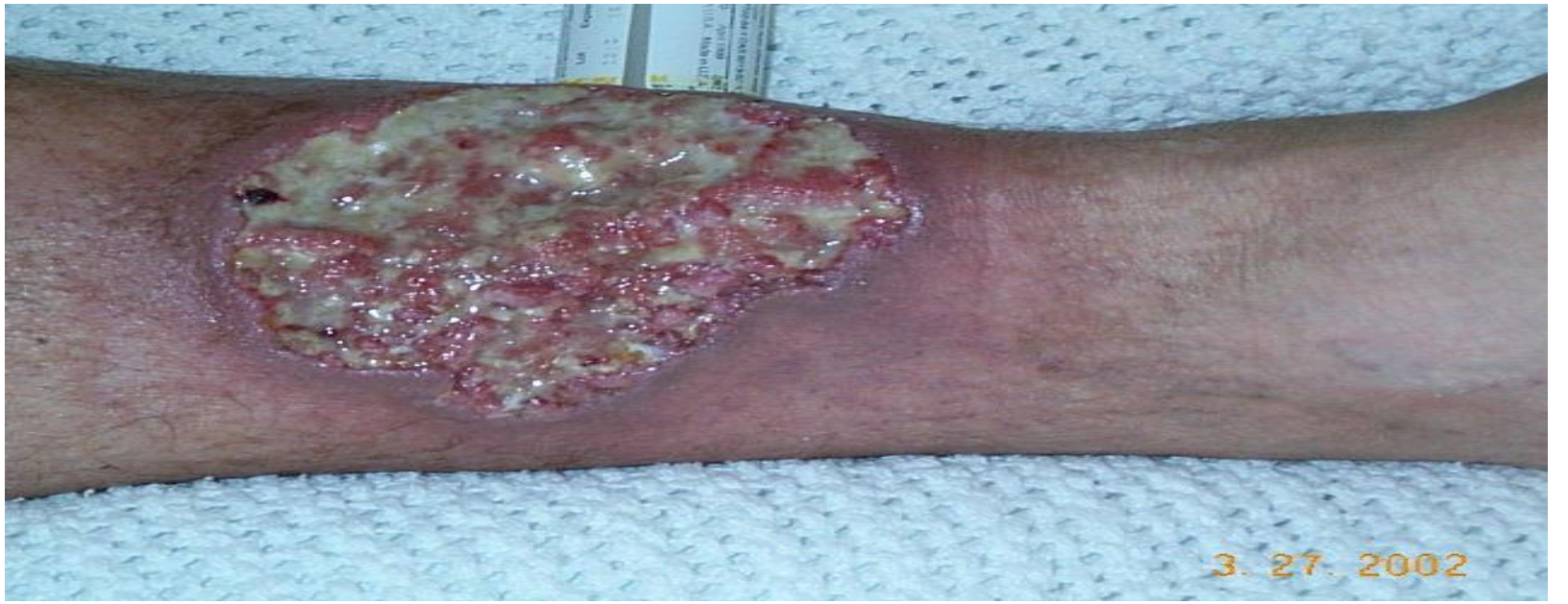
mention two line of management?

Local and systemic glucocoricod +azathioprine



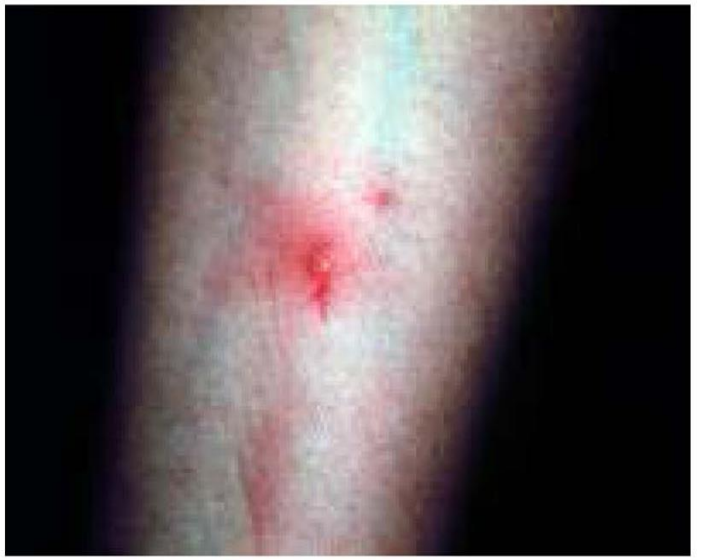


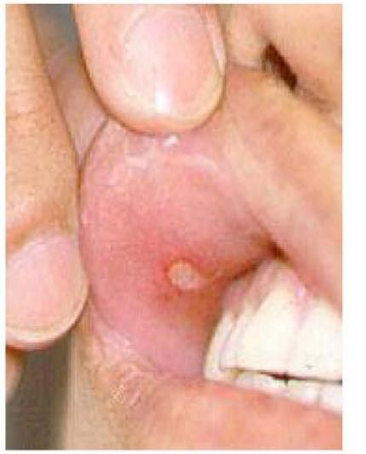
Q: Patient came to the clinic complaining of joint pain and this skin lesion
On history the patient had recurrent mouth and genital ulcers
what is your diagnosis?
Behcet disese
What is the name of this skin lesion ?
pyoderma gangrenosum
mention other skin lesion of this disease?
erythema nodosum, folliculitis



Patient with painful mouth lesion, and we did this test for him (below), 3months later he developed left leg swelling & calf pain that diagnosed as DVT.

What is the diagnosis ?





Rheumatic fever (RF)

***Cauesd by** group A streptococcus infection of certain M-protein types

***Natural history of the disease**

(group A strep-----URTI-------2-3 weeks later rheumatic fever -----many years later rhematic heart disease)

***Clinical feature of rheumatic fever**:

1- high grade fever (more than 39)

2- arthritis:-in 60%-75% of cases
(polyarticular ,large joints, disabling, migratory in hours)

3- skin lesion (erythema mrginatum on the trunk…limb..face),
(sub cutaneous nodule ….painless and mobile overlying bony prominence ….. In hand , feet, elbow)

4- heart involvement : in 50%-60% of cases
(carditis with valvular damage, heart failure , …..mitral valve is almost always is affected , aortic valve may be involved also) ----
early valvular damage lead to regurgitation

5- evidence of preceding group A strep infection

6- abnormal movment (Sydenham Chorea)

Investigations of acute rheumatic ferver:

ESR , CRP, echo, CBC , blood culture , anti-strptolysin o (ASO) titer, anti-DNAase B (ADB) titer, throat swap

Jone's criteria of diagnosis of rheumatic fever :

Dx of intial rheumatic ferver:

2 major criteria or 1 major crireria+2minor

Dx of recurrent rheumatic ferver:

(2major)or(1 major+2 minor)Or(3minor)

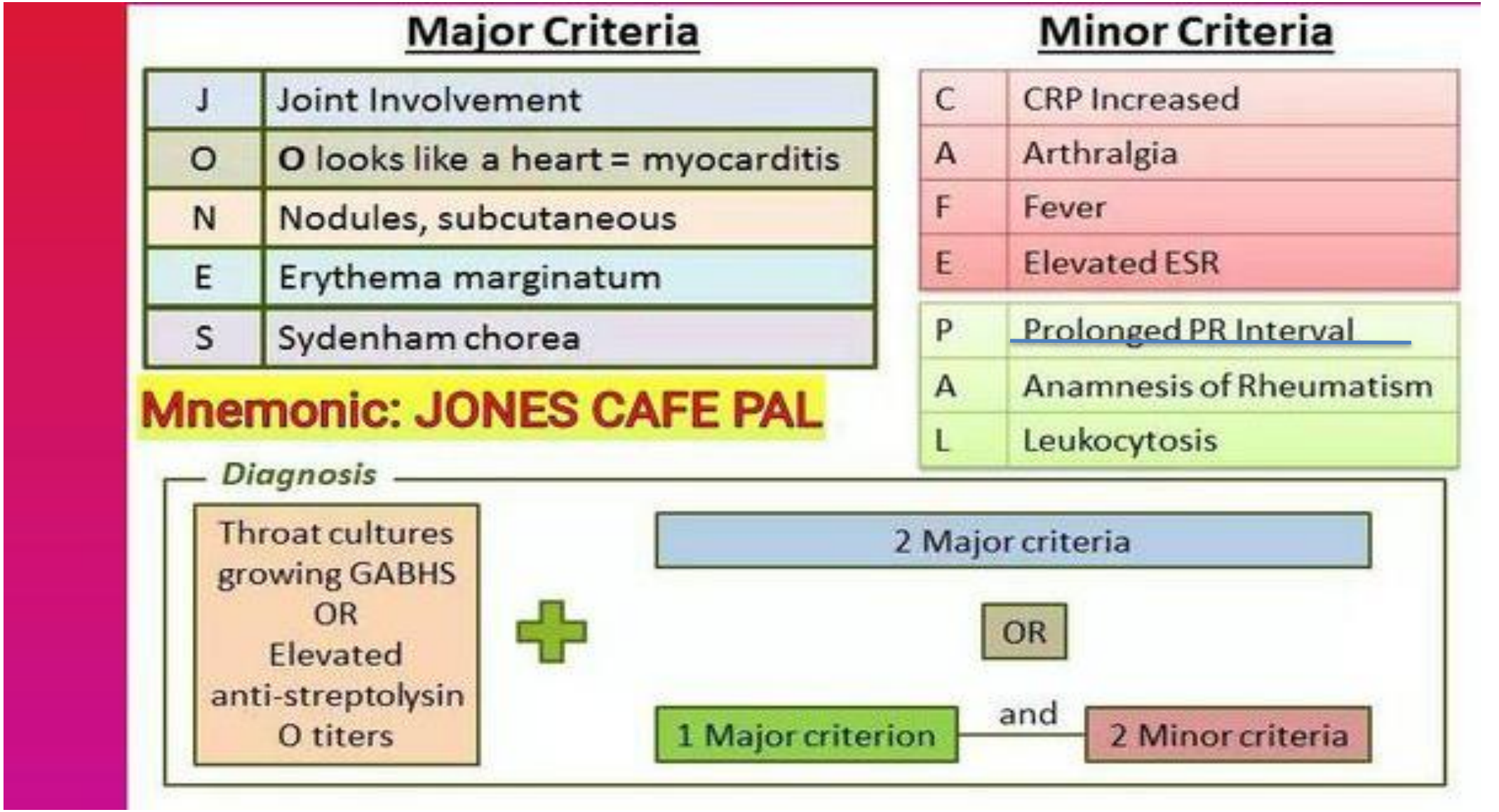
1-Major criteria :

Arthritis , carditis , erythema marginatum , Sydenham Chorea , sub-cutaneous nodule

2-minor criteria:

ferver , high ESR , highCRP , poly-arthralgia

HOW TO DIAGNOSE ?



Q: A young patient with recent history of upper respiratory tract infection presented this abnormality

Identify this abnormality

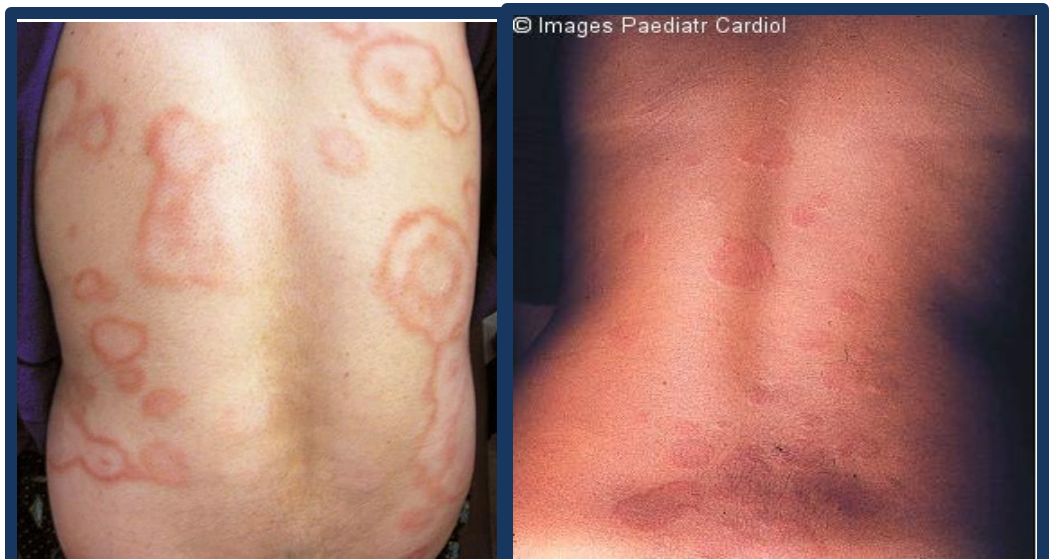
Erythema marginatum

What is the most suspected diagnosis?

Rheumatic fever

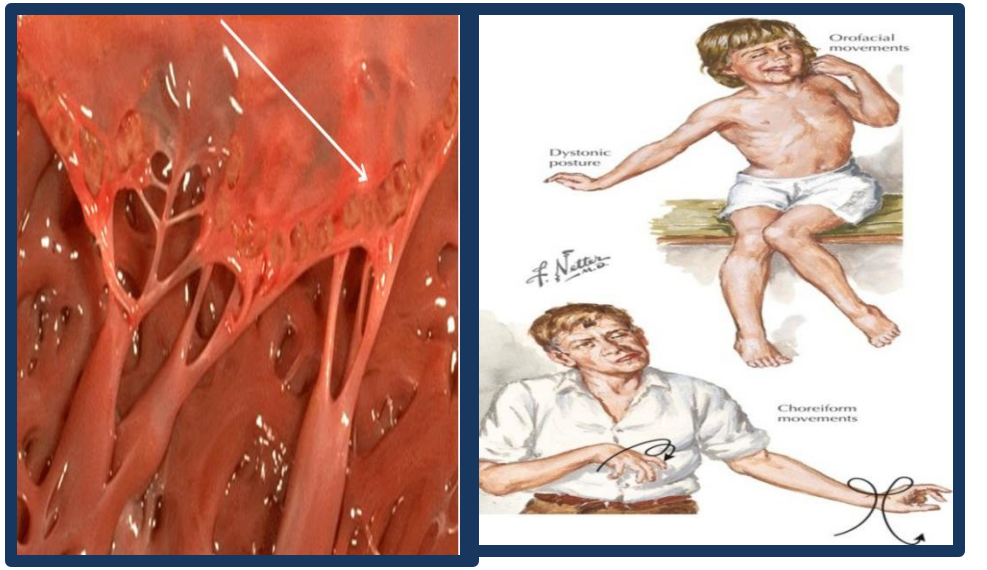
What is the most suspected cause ?

Immune mediated delayed response to group A beta hemolytic streptococcus infection



Q: Give 4 symptoms the patient may present with ?

- Painful joints (Flitting polyarthritis)
- Dyspnea (pancarditis)
- Abnormal movements , worsening of handwriting (Sydenham Chorea)
- Painless nodules (subcutaneous nodules)



Q: Give other 3 signs can be seen in this patient ?

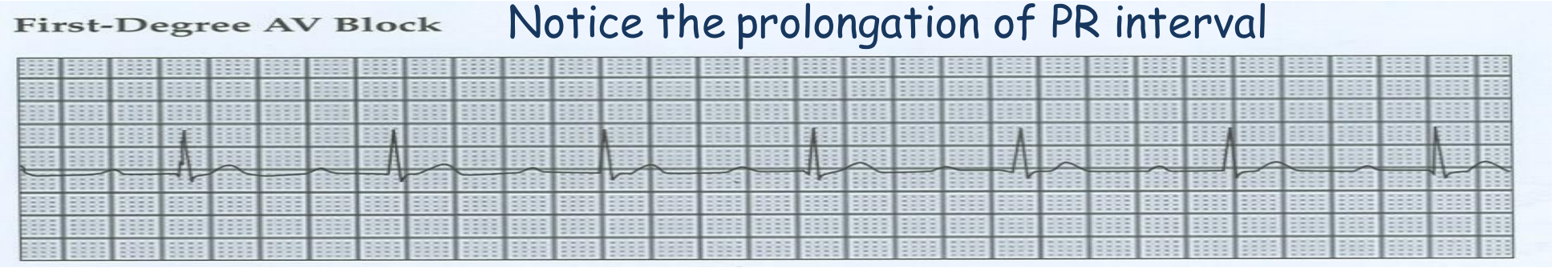
- Pronator sign : pronation of the patient hand when the arm is raised
- Jack in the box sign : when protrude the tongue, the patient is unable to keep it out
- Milking sign : by asking the patient to squeeze the examiner's hands; the pressure of the patient's grip increases and decreases continuously and capriciously, a phenomenon known as relapsing grip or "milking sign."

Q: Give 5 investigations required in this case

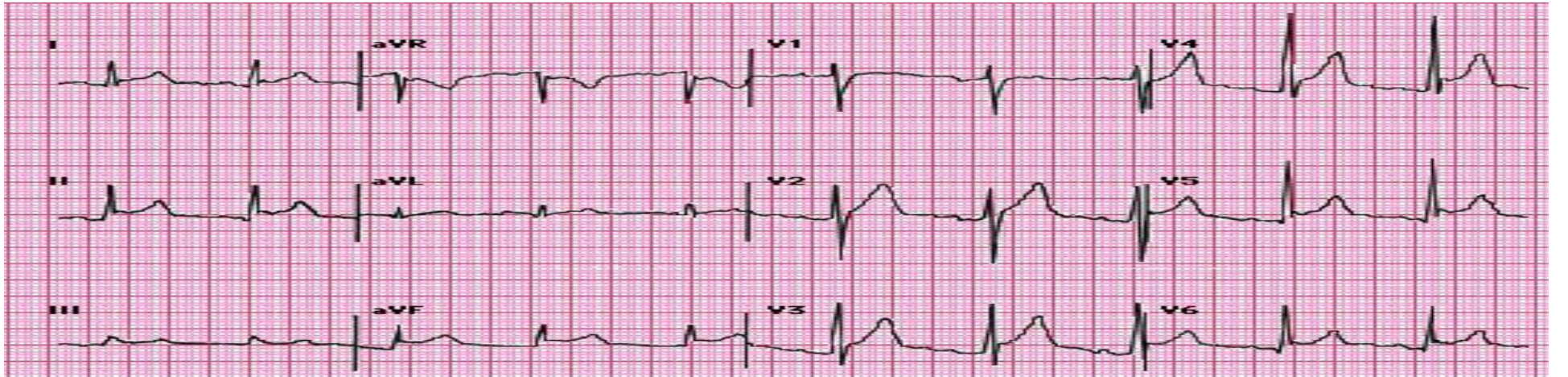
- Acute phase reactant (ESR, CRP)
- ASO titer
- Anti DNAse B titer
- Throat culture
- ECG , Echocardiogram

Q: Give 2 ECG abnormalities you suspect in this patient

- atrioventricular block
- PR depression, ST elevation , T wave inversion
  (pericarditis and pericardial effusion)



Pericarditis :
ST elevation (lead II , lead III, aVF , V3- V6)
PR depression in most leads (except aVR)



Q: What is the treatment ?

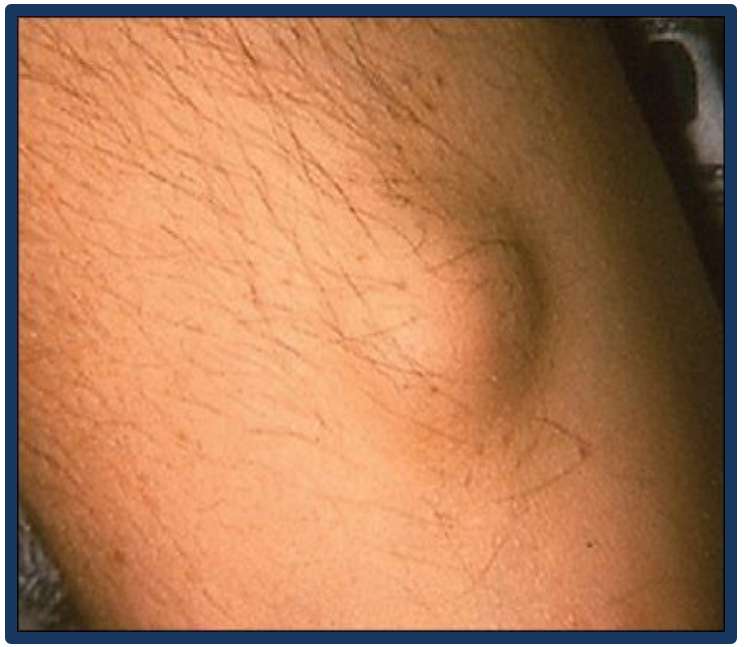
- Eradication of GABS (mainly Benzathine penicillin G)
- Anti inflammatory treatment (aspirin, steroid)
- Supportive, treat complications
- prevention

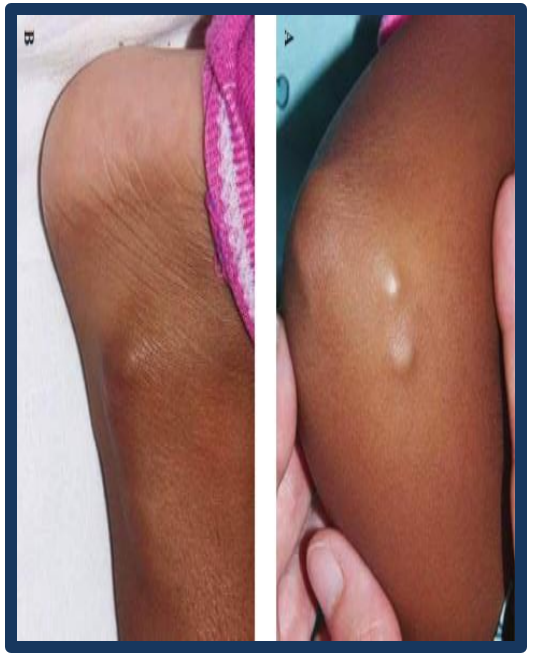
Q: Identify this abnormality

Subcutaneous nodules

Give 3 differential diagnosis

- rheumatic fever
- juvenile rheumatoid arthritis
- neurofibromatosis





Q: Young patient presented with this abnormality

Identify this abnormality

Arthritis of ankle joint

Give 5 differential diagnosis

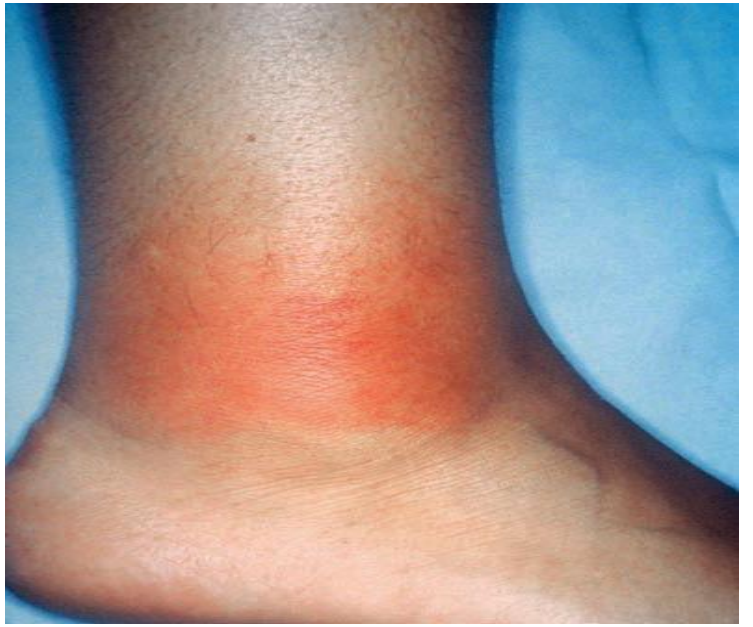
rheumatic fever

juvenile rheumatoid arthritis

septic arthritis

sickle cell arthropathy

Kawasaki disease



Q: Do these results go with the diagnosis of rheumatic fever ?

Lab results:	
ASO titer	280 todd
ESR	120 mm/ hr.

Yes

- High ASO titer (more than 200 todd is significant)
- high ESR (nl. Up to 30 mm/hr.)

8.septic arthritis

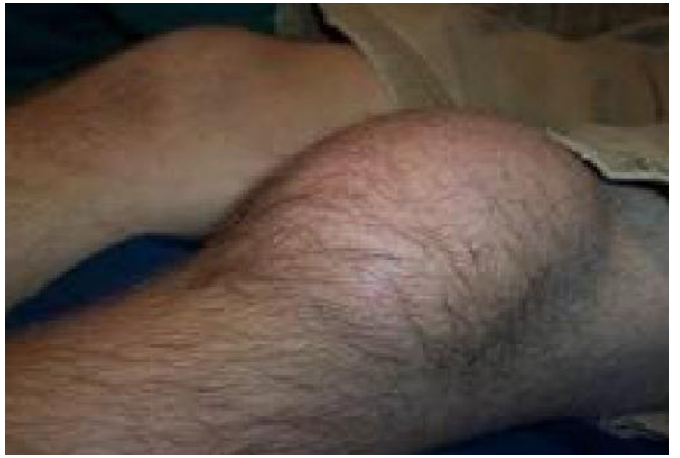
Q: A 34 YO man comes to the ER after 3 hours of severe pain in his knee, on exam is left knee is swollen, warm, & very tender to palpation.

What is the Dx?

septic arthritis

Give one investigation?

synovial fliud aspiration.



Tx: The patient should be admitted to hospital for pain relief and administration of parenteral antibiotics. Pending the results of cultures

Q: A man comes to the ER after3 hours of severe pain in his knee, on examination his left knee is swollen, warm, & very tender to palpation. What is the Most likely diagnosis?

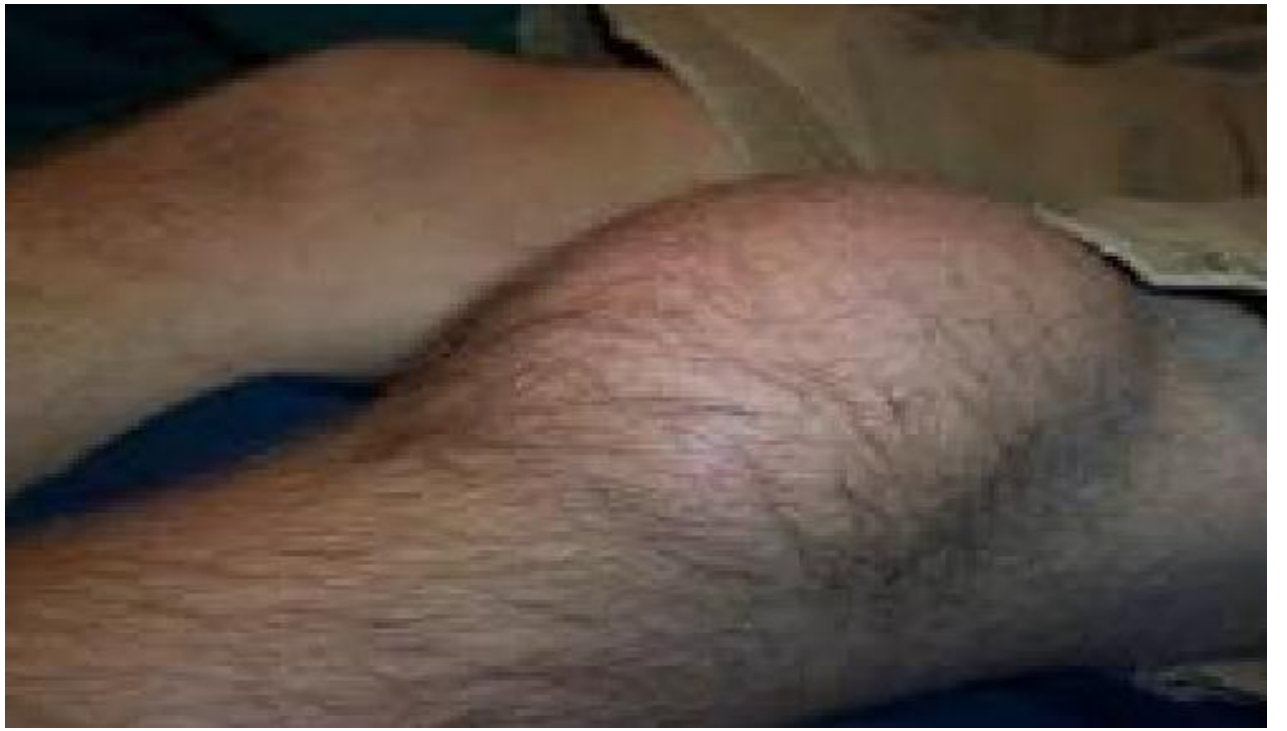
Septic Arthritis

What is the investigation of choice?

Synovial Fluid Analysis (should be sent of gram stain, culture and sensitivity)

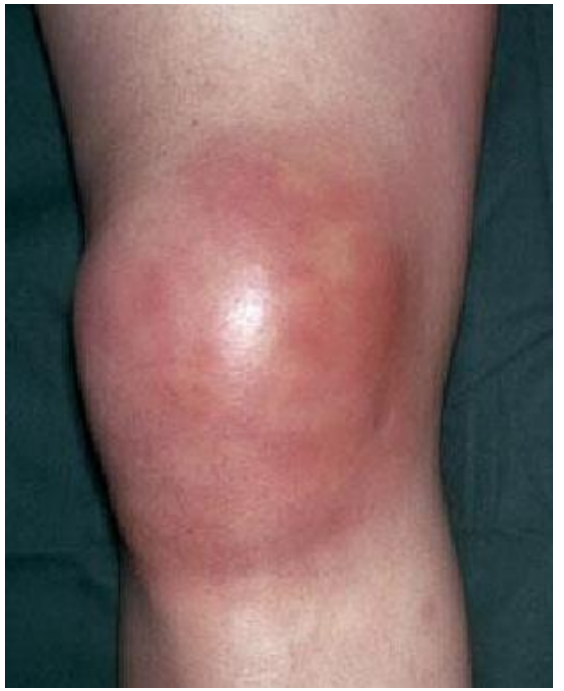
What other investigation can be done?

Blood culture, CRP and ESR, CBC (leukocytosis)



Q: A pt came to ER complaining of swelling in his left knee. He has no Hx of trauma or bleeding diathesis. What is your most likely Dx?

Septic Arthritis.



Most common orgenism in old:staph aureus while in adult (sexaul active) :gonorrhea

Common cause of monoarthritis

- Gout
- Pseudogout
- Trauma
- Haemarthrosis
- Spondyloarthritis
- Psoriatic arthritis
- Reactive arthritis
- Enteropathic arthritis
- Palindromic arthritis

Less common

- Rheumatoid arthritis
- Juvenile idiopathic arthritis
- Pigmented villonodular
- Synovitis
- Foreign body reaction
- Tuberculosis
- Leukaemia*
- Gonococcal infection
- Osteomyelitis*
- *In children, both leukaemia and osteomyelitis may present with monoarthritis

Dermatomyositis

Q: This patient complained of shoulder and hip weakness. What is your diagnosis?

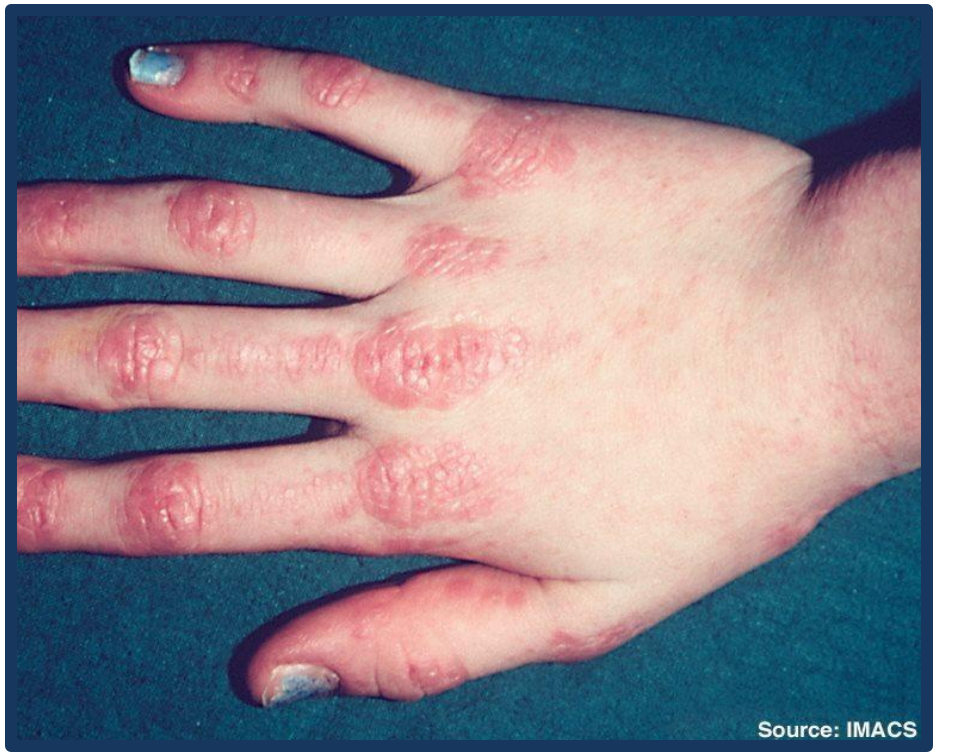
Dermatomyositis (Idiopathic inflammatory myopathy) .

Q: This pt was presented with proximal muscle weakness, dysphagia , and this skin rash, what is your diagnosis ?

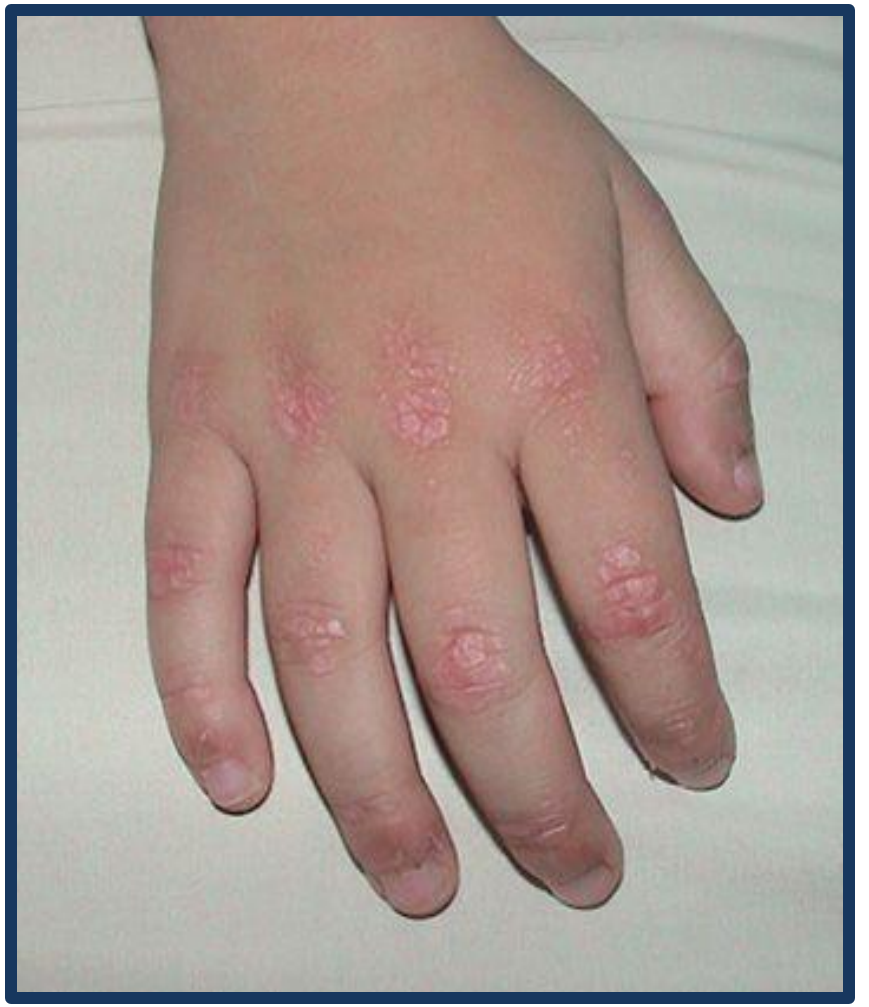
dermatomyositis

what's the rash seen in this patient?

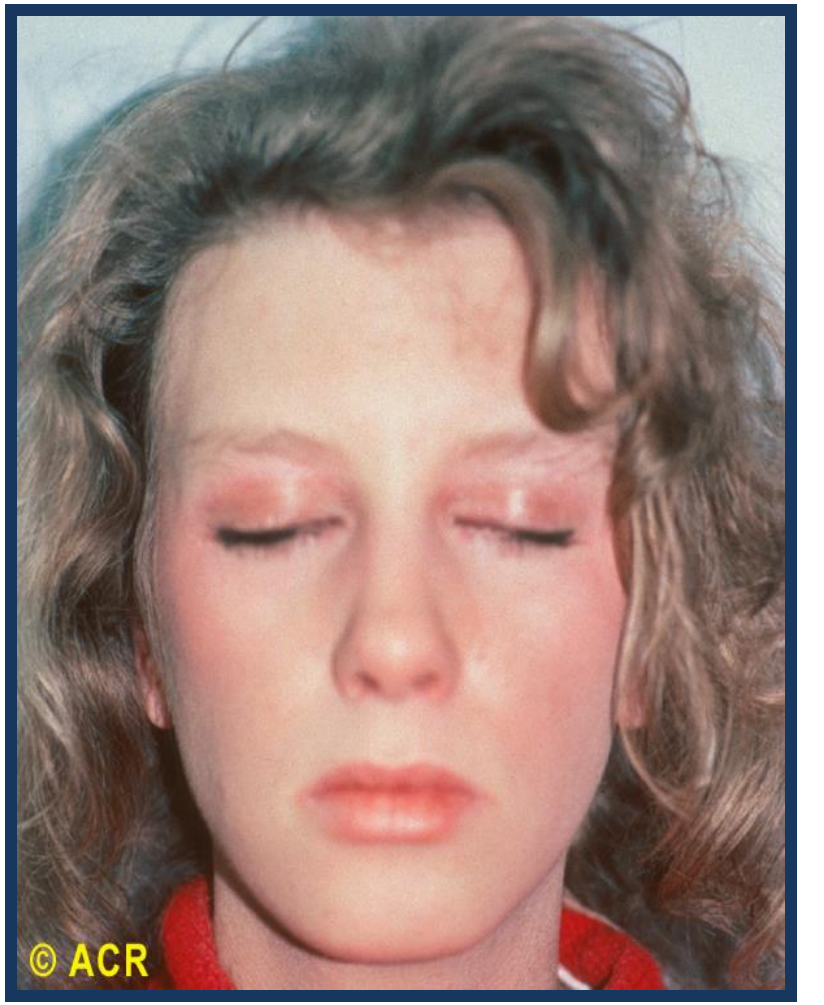
Gottron's papules



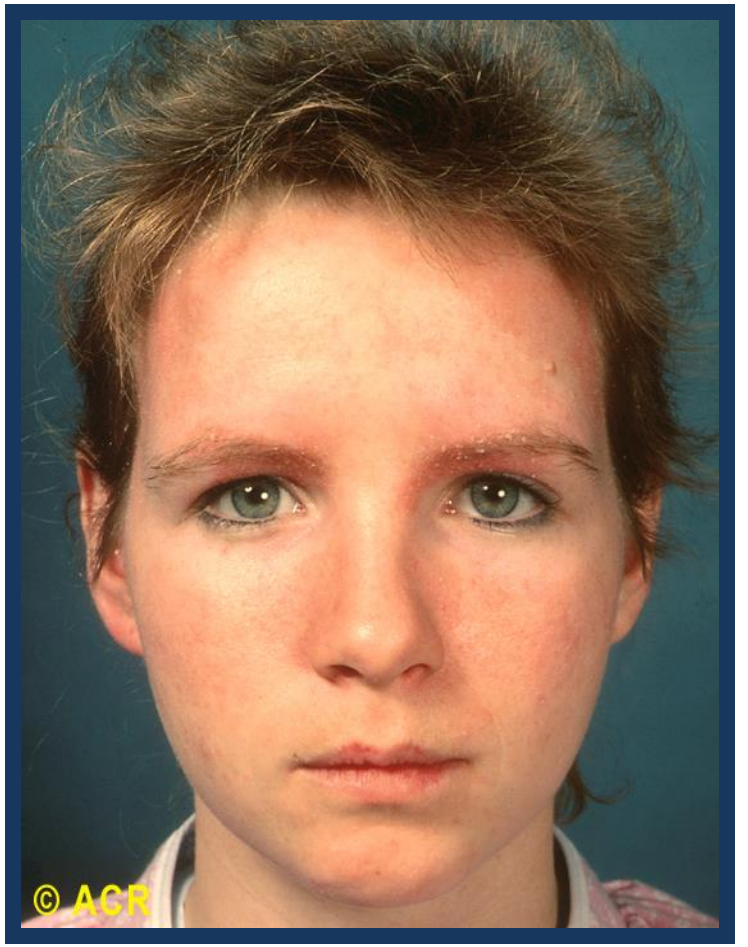
Gottrons papules



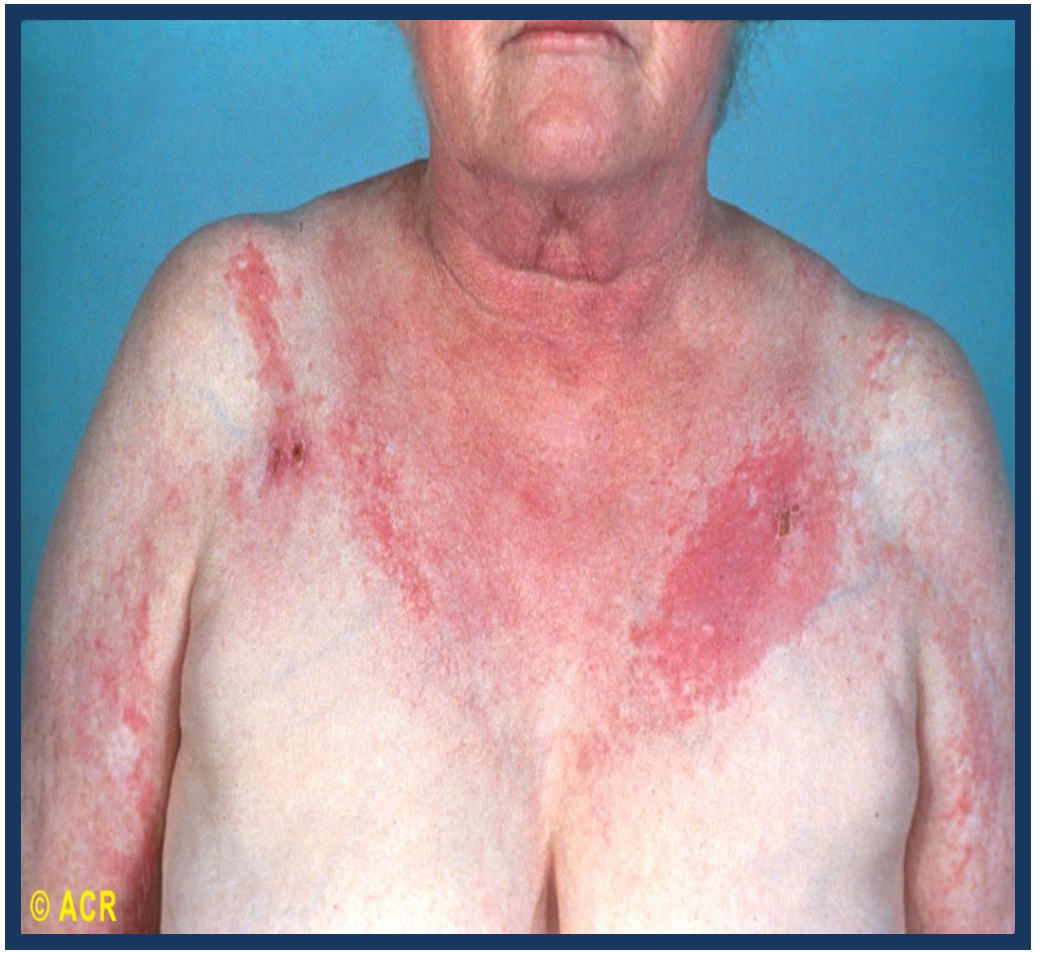
Dermatomyositis: heliotrope rash



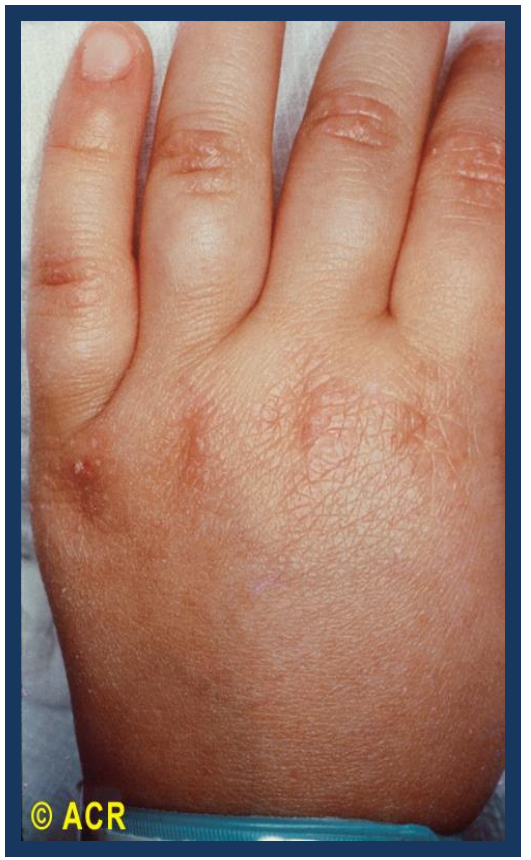
Dermatomyositis: diffuse facial erythema



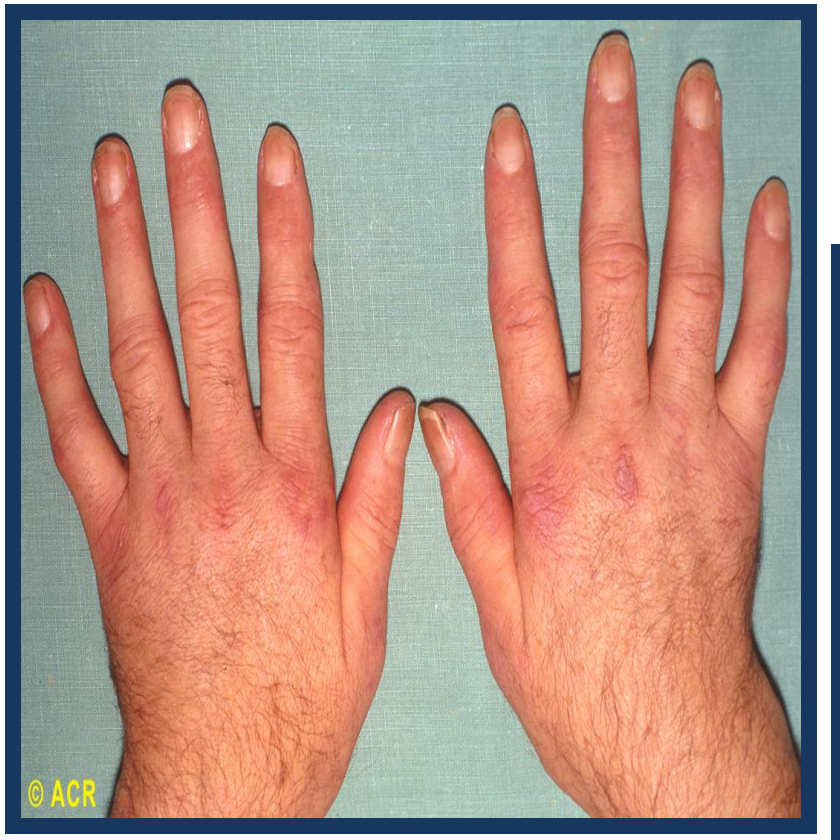
Dermatomyositis: rash, chest



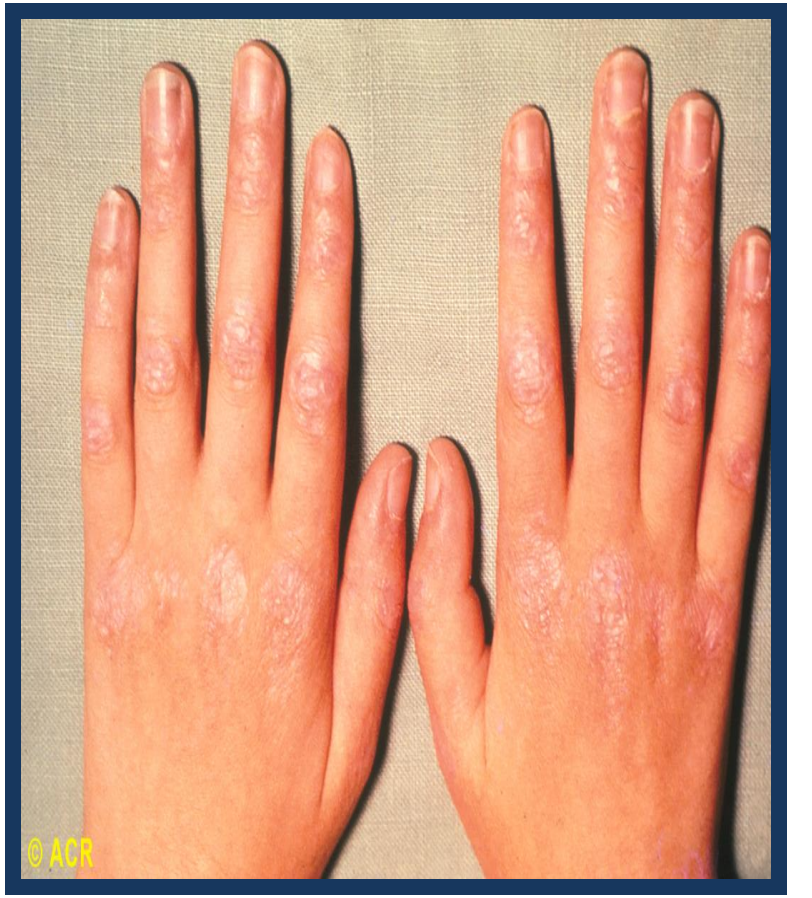
Dermatomyositis: edema and rash, hand

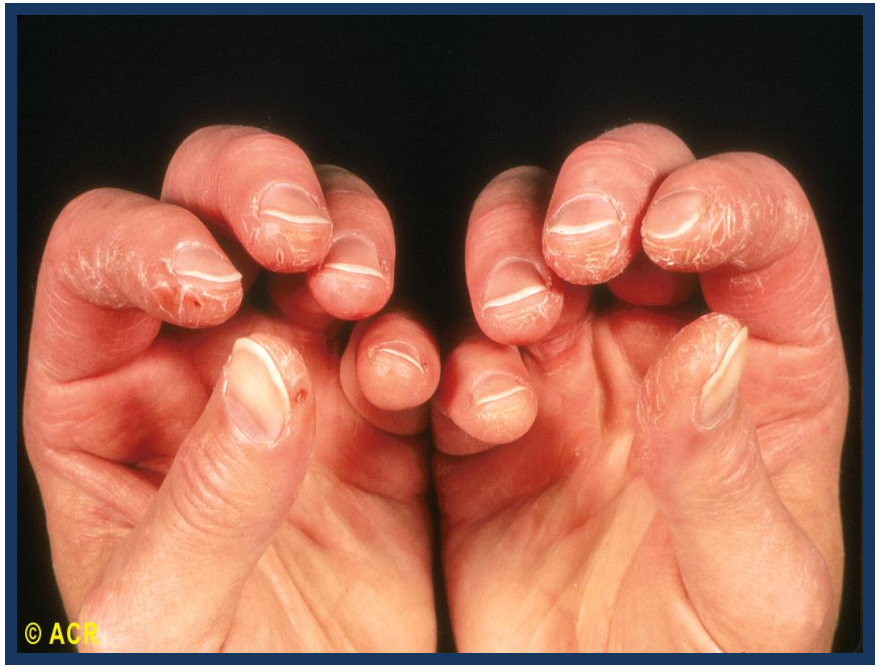


Dermatomyositis: erythematous lesions, hands

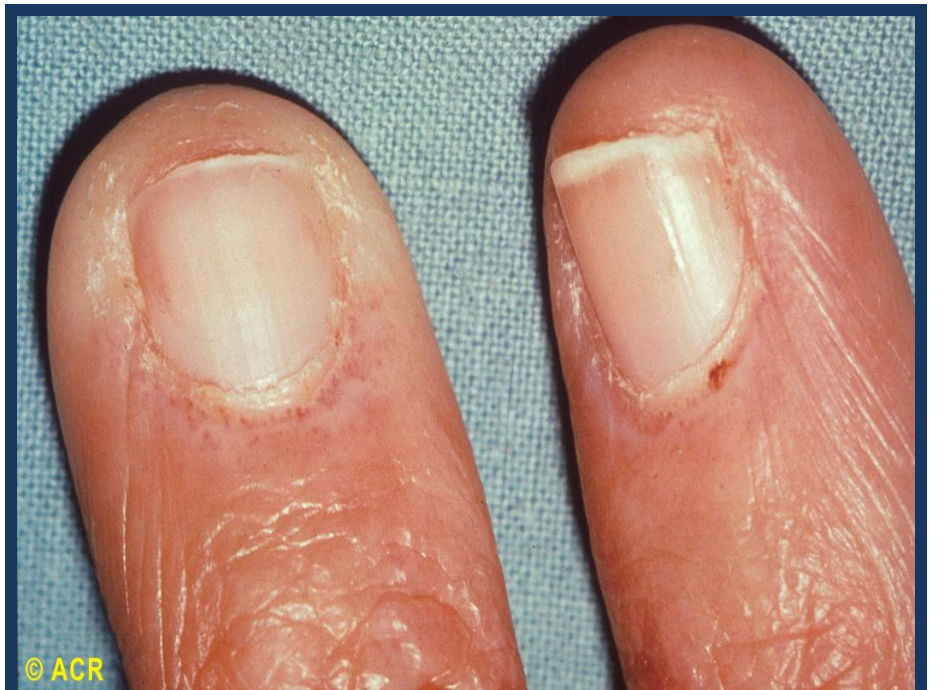


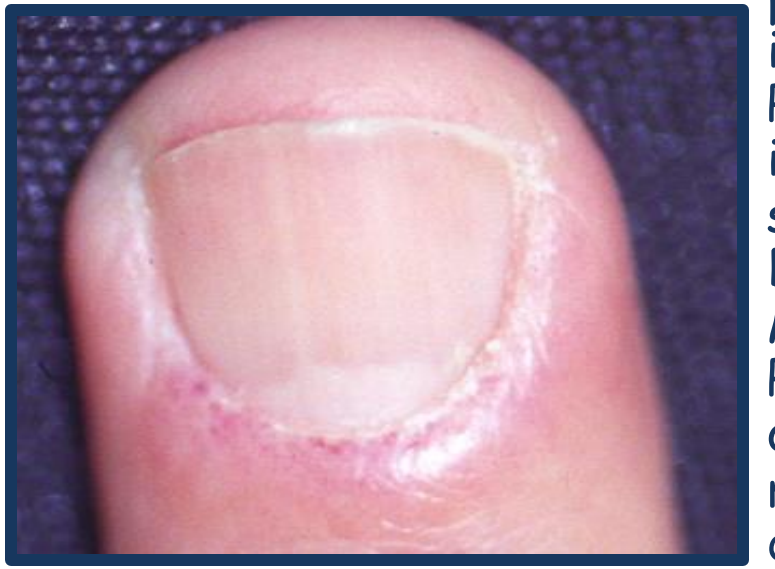
Dermatomyosistis: rash, hands





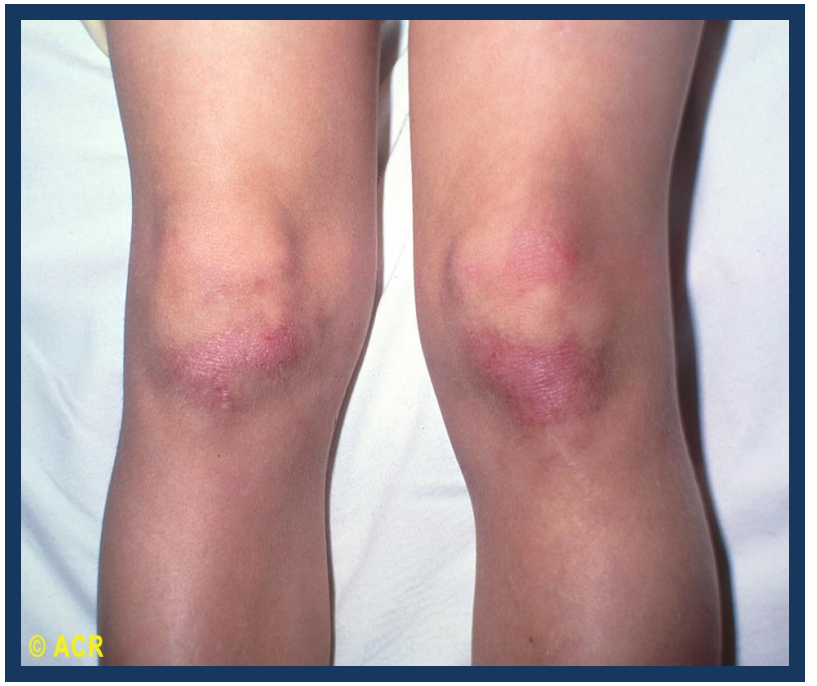
Dermatomyositis:
"mechanic's hands"



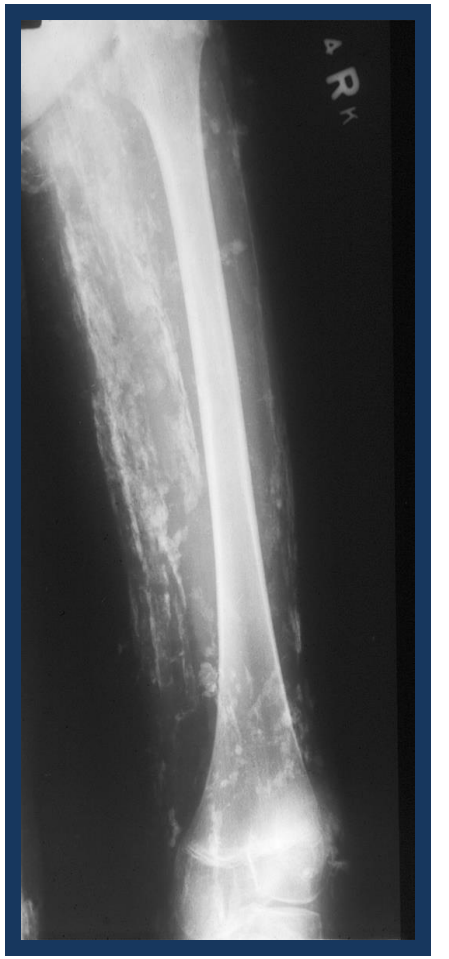


Dermatomyosistis: periungual involvement
Periungual involvement of nail, seen in Dermatomyositis. May be also seen in Polymoysitis and other forms of rheumatologic disorders

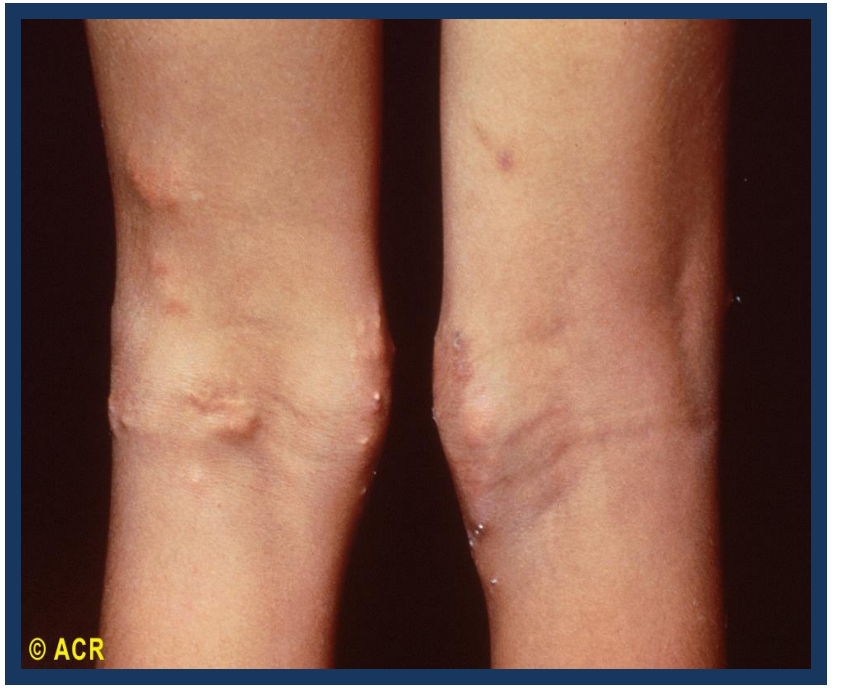
Dermatomyositis: rash, knees



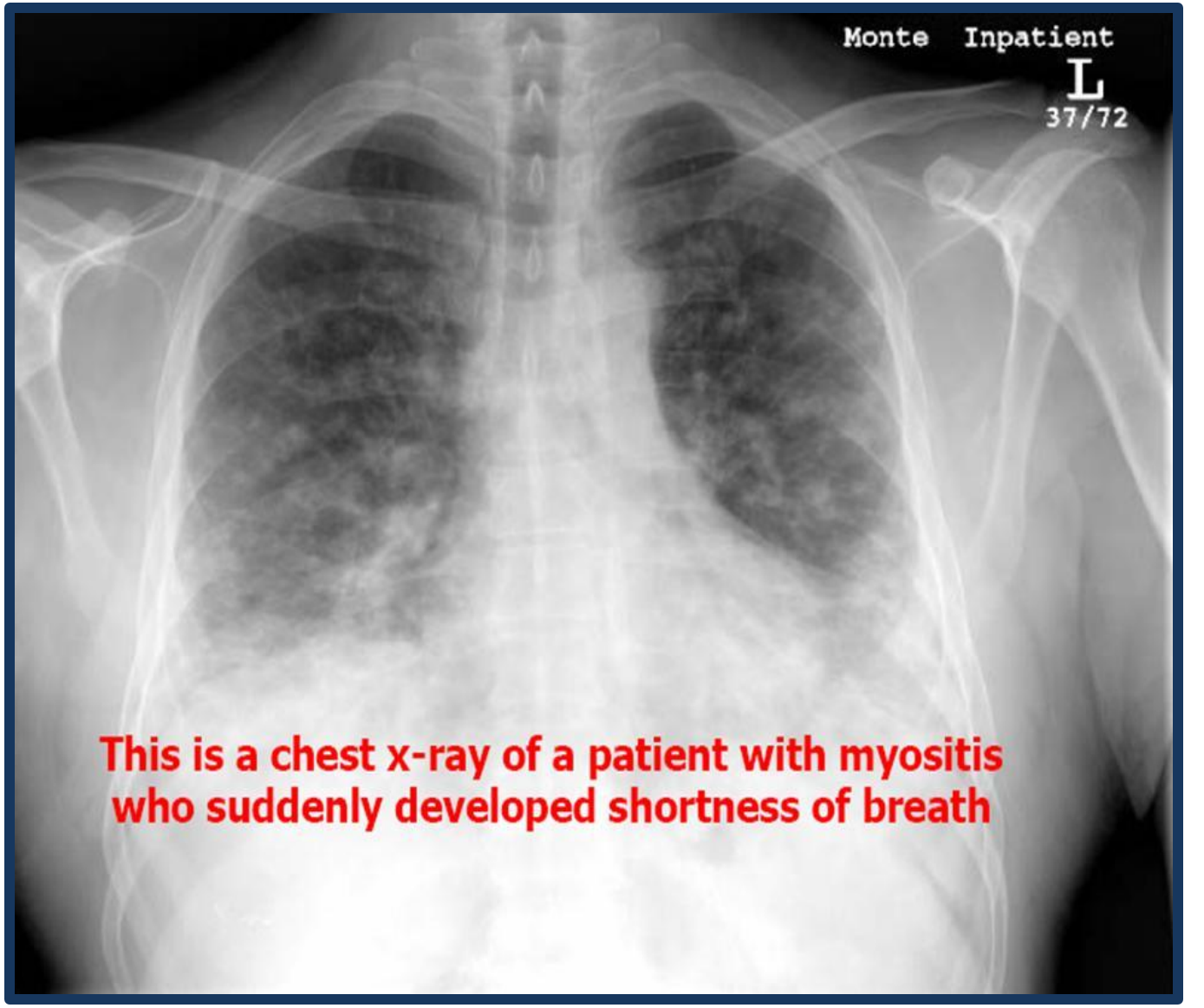
Dermatomyositis: calcinosis, thigh (radiograph)



Dermatomyositis: subcutaneous calcification, knees



Mechanic's hand seen in dermatomyositis



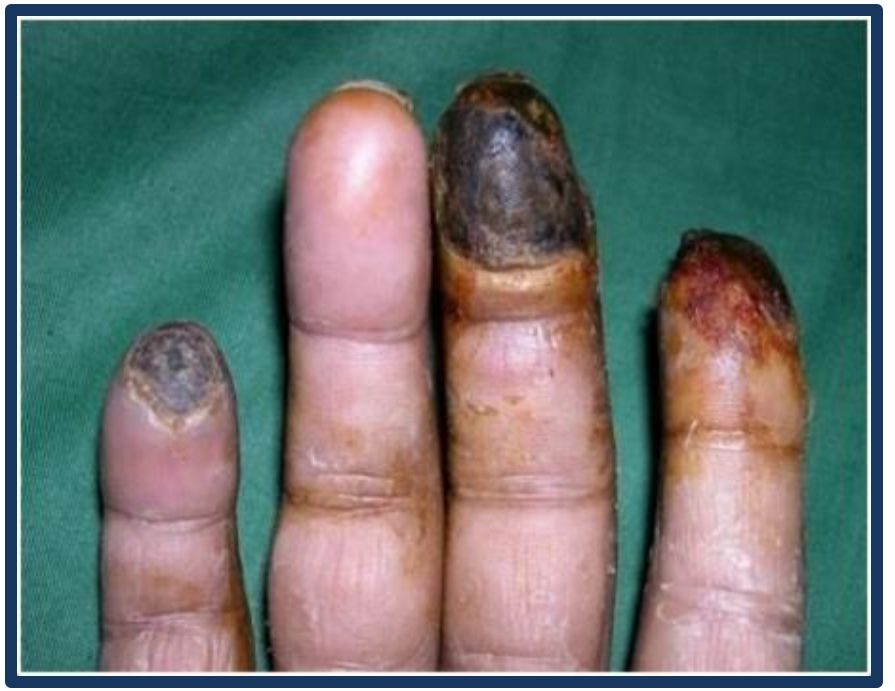
Inclusion body myositis

- Presents in an old male with distal rather than proximal muscle weakness (although both can happen), asymmetrical, and not responsive to steroids.

Vasculitis

Q: This patient is a young male smoker who presented to the ER with this picture, what is the diagnosis ?

Dx: Buerger Disease (thromboangiitis oblitirans)



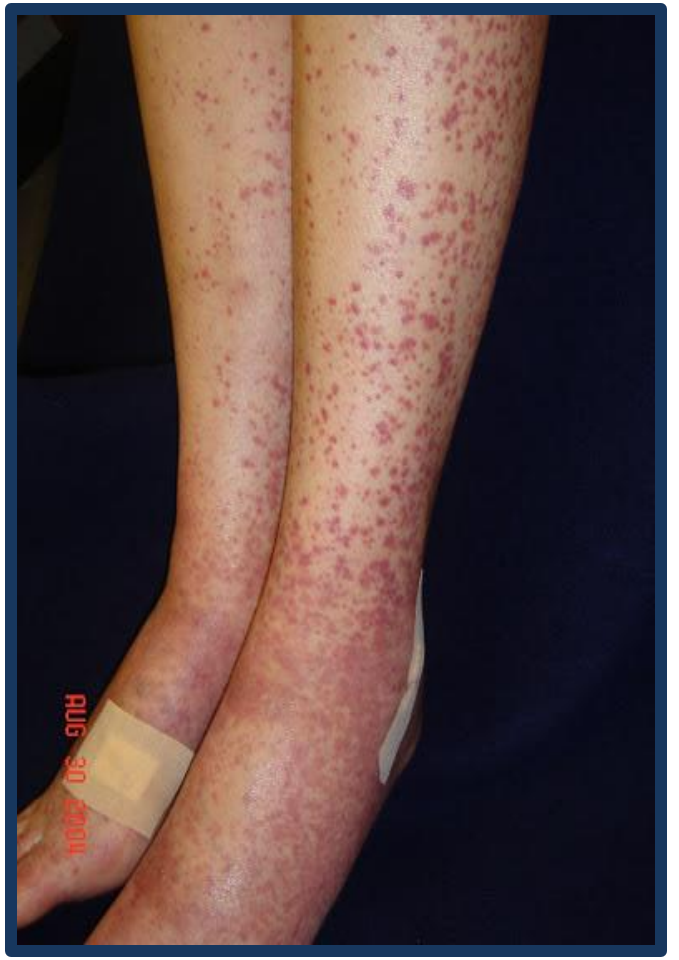
Q: An old male patient presented with progressive eye redness and pain, and now is having tinnitus. What is the most probable diagnosis ?

Cogan syndrome, which is a form of large vessel vasculitis. It presents with sensorineural deafness and ocular abnormality (such as: uveitis, scleritis (as in this patient), episcleritis, vertigo, oscillopsia). The patient may also have constitutional symptoms.

Tx: Topical steroids for keratitis, and systemic steroids for other symptoms.

Q: This patient presented with this picture. She has a history of hepatitis C infection. What is the most likely diagnosis?

Cryoglobulinemic vasculitis

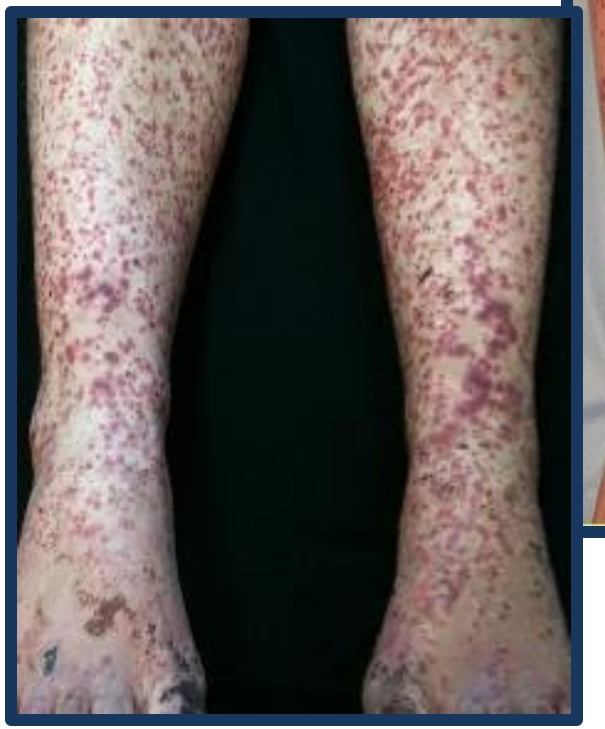


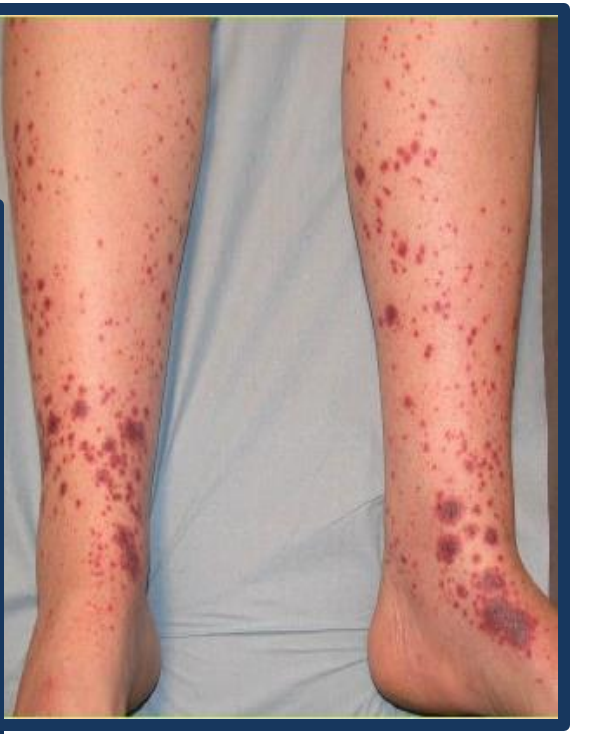
Q: This patient had abdominal pain, hematuria & this picture. What's your diagnosis?

Henoch-Schönlein purpura (HSP)

what's the other system to be involved ?

Joint pain





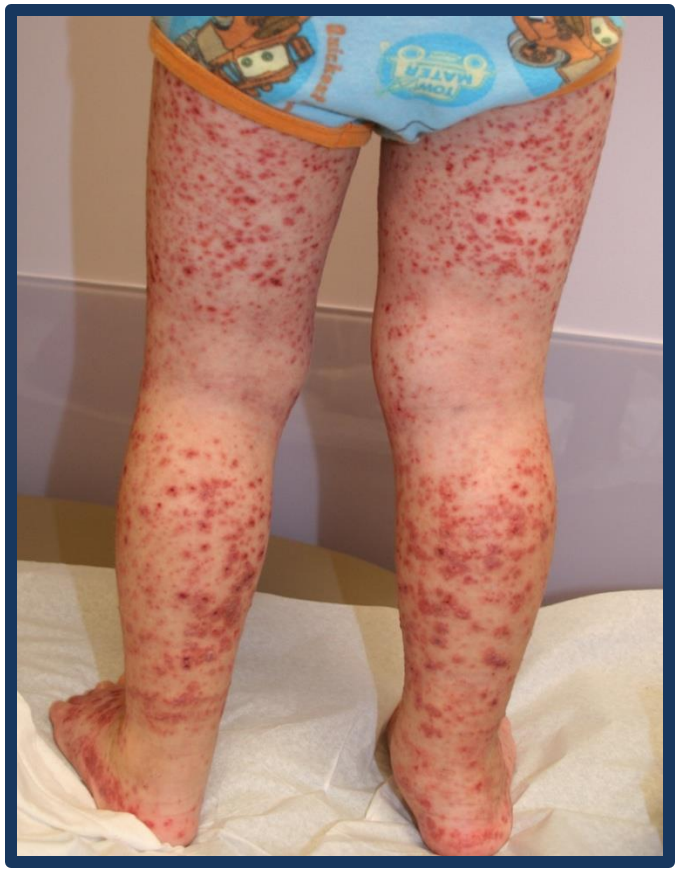
Q:A 12 years old boy.

a) What is your diagnosis?

Henoch schonlein purpura

b) What's the major cause of morbidity and mortality in this patient?

Renal failure



Q: Patient with hx of headache and high ESR

A- What's your diagnosis?

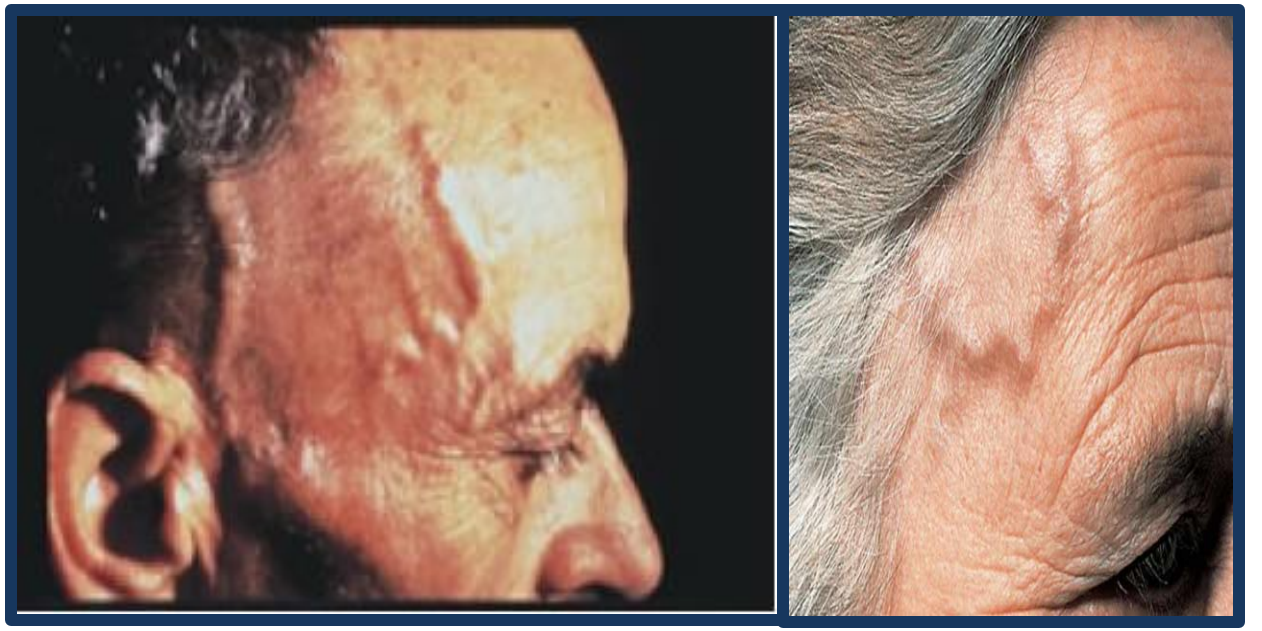
temporal arteritis

B- Give one complication?

vision loss, stroke, hemorrhage

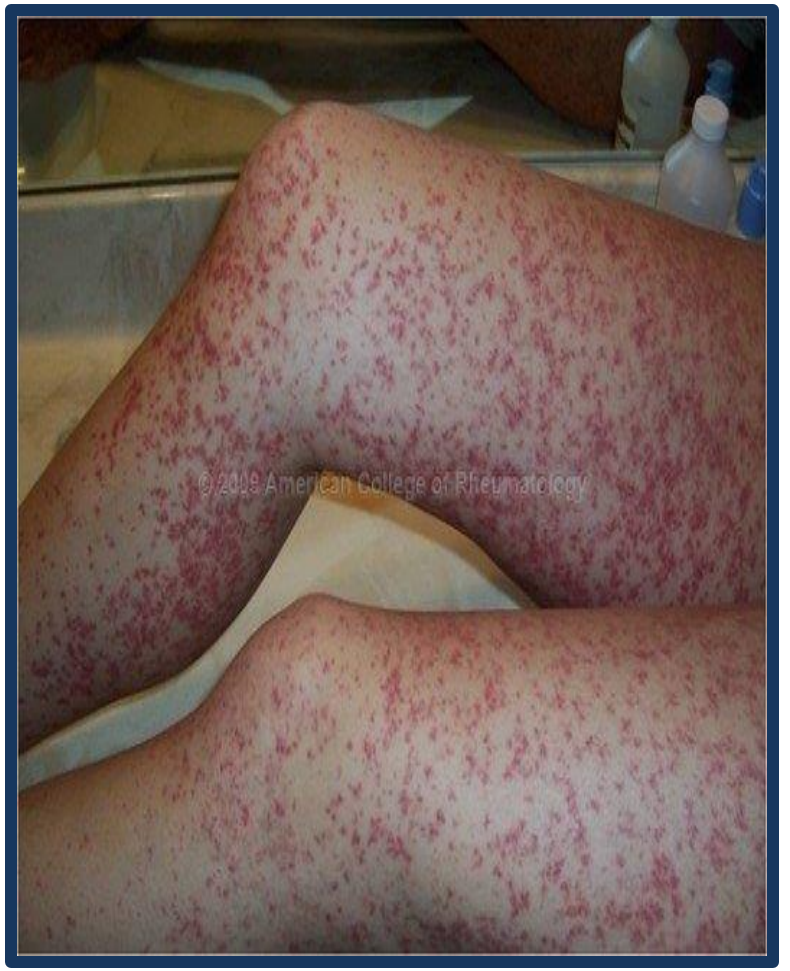
C- What is the best next step in management?

High dose IV corticosteroids [it is preferable to write: intravenous since it is an emergency case]



Q:A previously healthy 31-year-old female presented with this rash with a normal platelets count, Diagnosis?

HSP



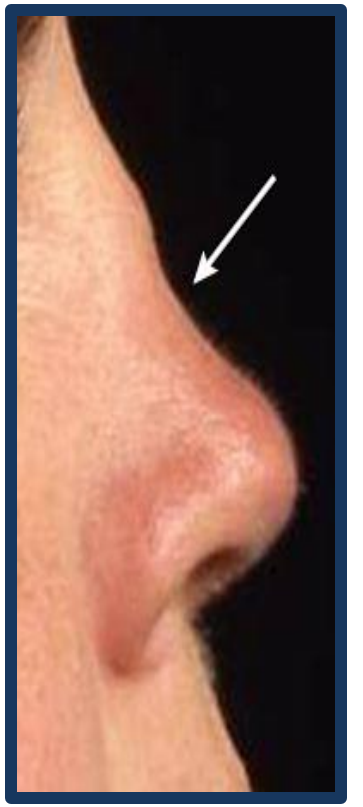
Q: This patient presents with history of recurrent sinusitis. He presents with hemoptysis and acute renal failure.

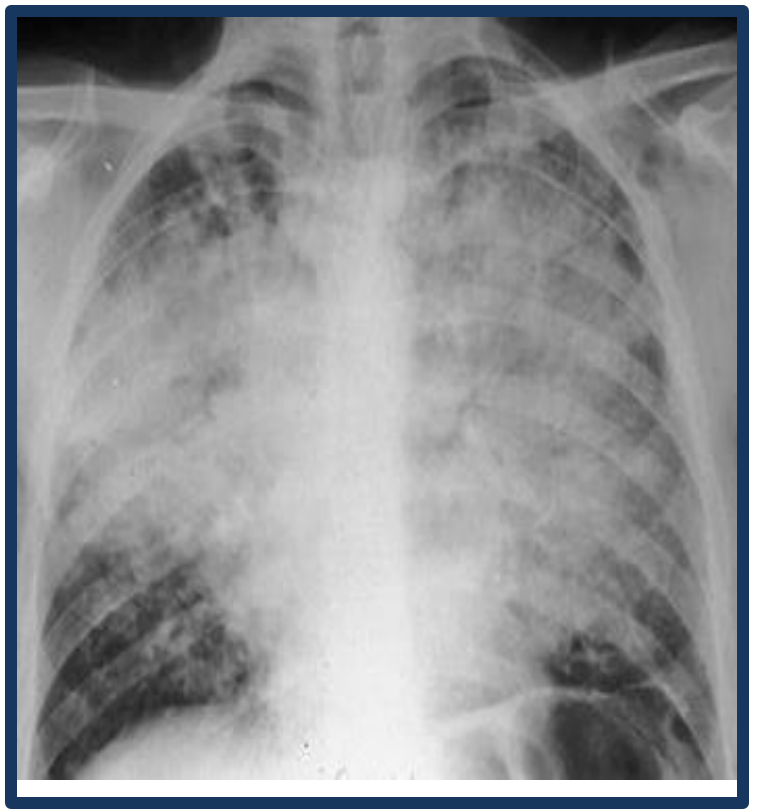
What is the most likely underlying diagnosis?

Wegener's granulomatosis (granulomatosis with polyangiitis).

What is the likely cause of hemoptysis?

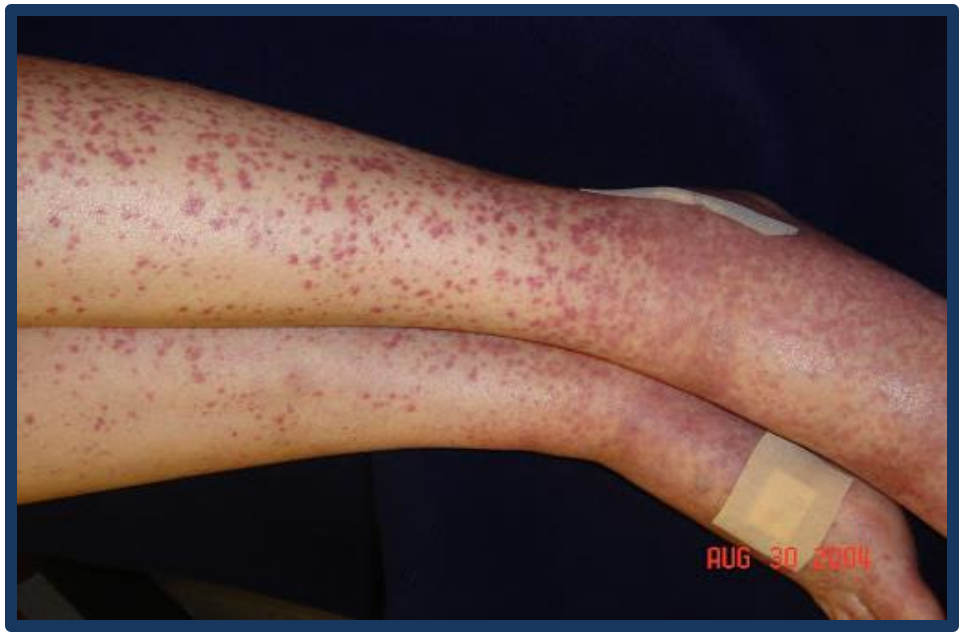
vasculitis in the pulmonary blood vessels.





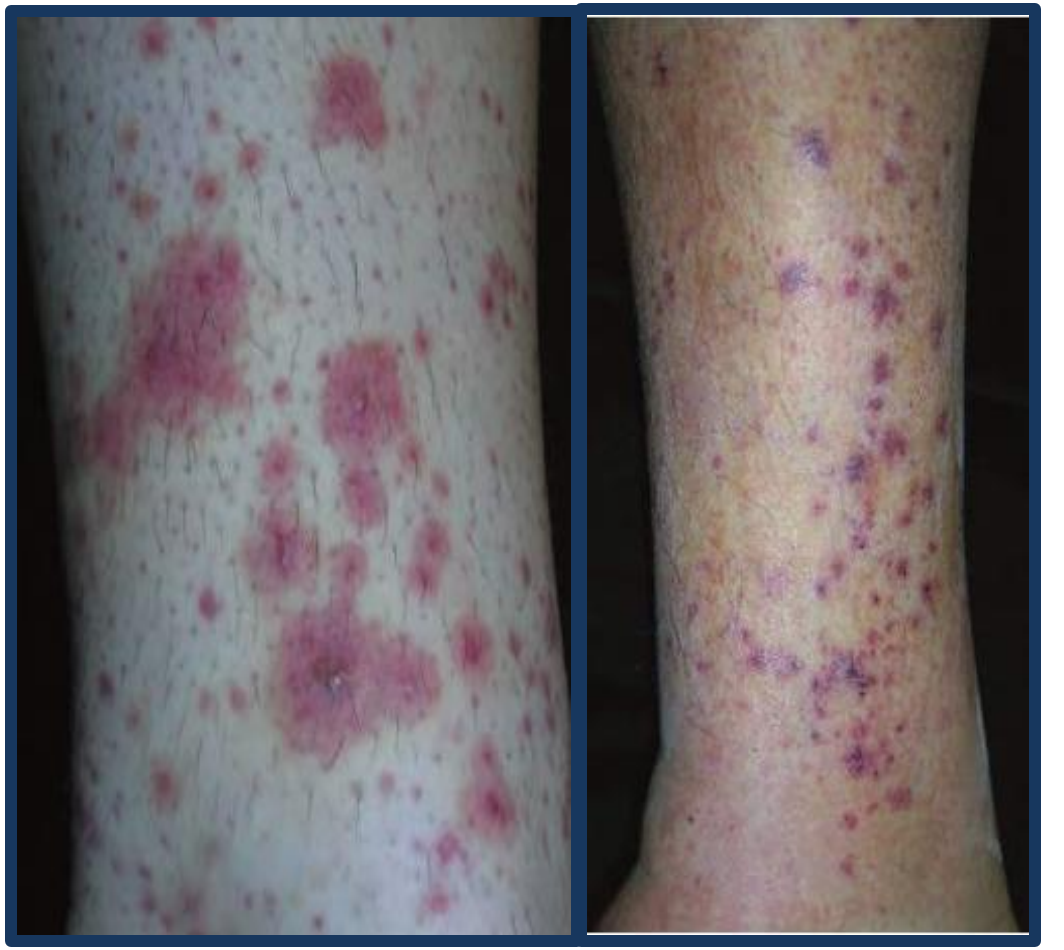
Q: This patient presented with this palpable rash, she had a history of URTI (upper respiratory tract infection) and she took antibiotics 1 week ago. What is the most likely diagnosis?

Dx: Hypersensitivity vasculitis, may be seen 7-10 days after infections or drugs intake.

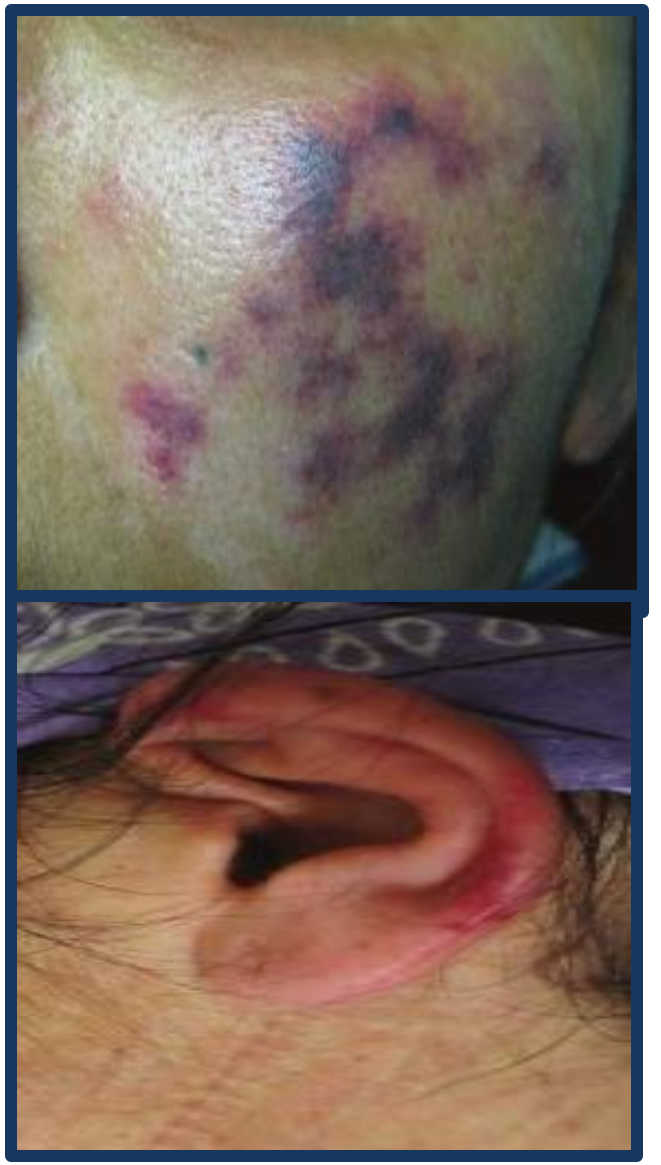


DX: Henoch-Schönlein purpura

F: Close-up of palpable purpura. Some lesions look like target lesions.



F: Cutaneous vasculitis in (net-like) pattern



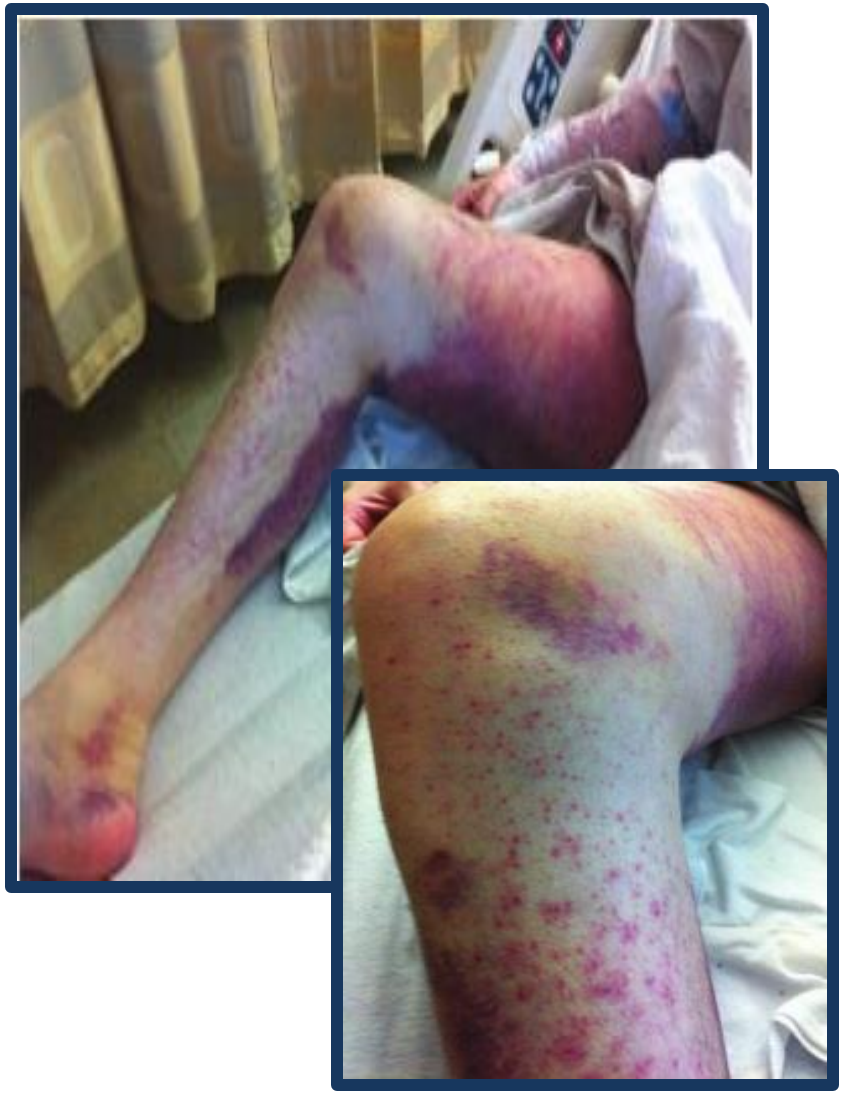
Q: This patient presented with this picture and dyspnea. What is the diagnosis?

Granulomatosis with Polyangiitis (Wegener Granulomatosis). The nose of this patient shows saddle nose deformity.

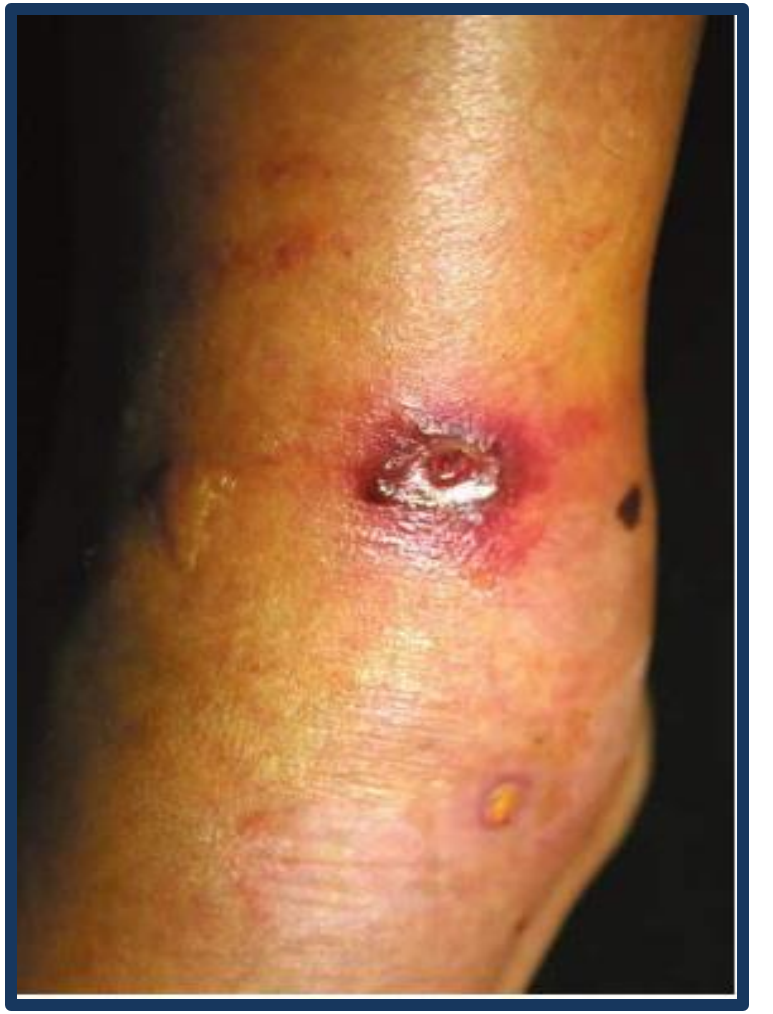
What is the most common antibody to be positive in this patient?

C-ANCA (anti-PR3 antibodies)

DX: Scurvy
F: Ecchymosis and petechial rash

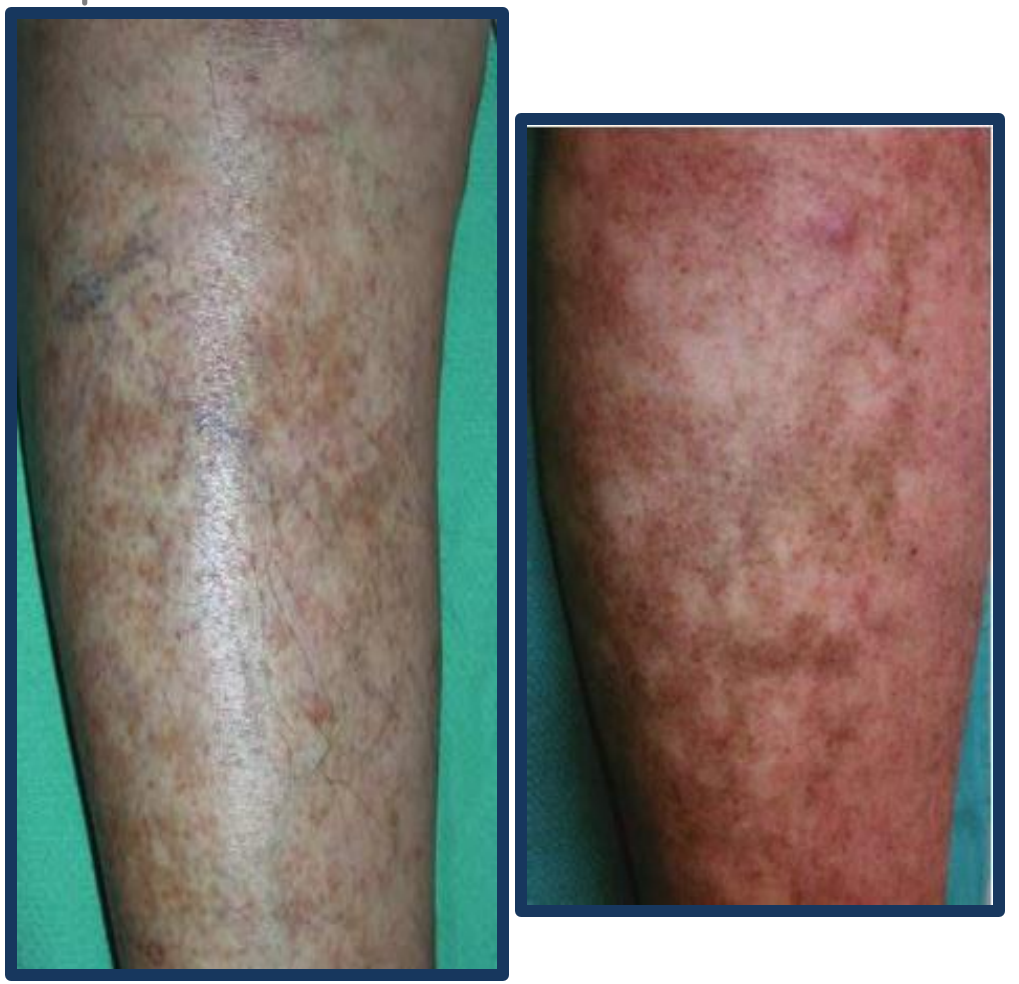


DX: SLE
F : Vasculitic ulcer



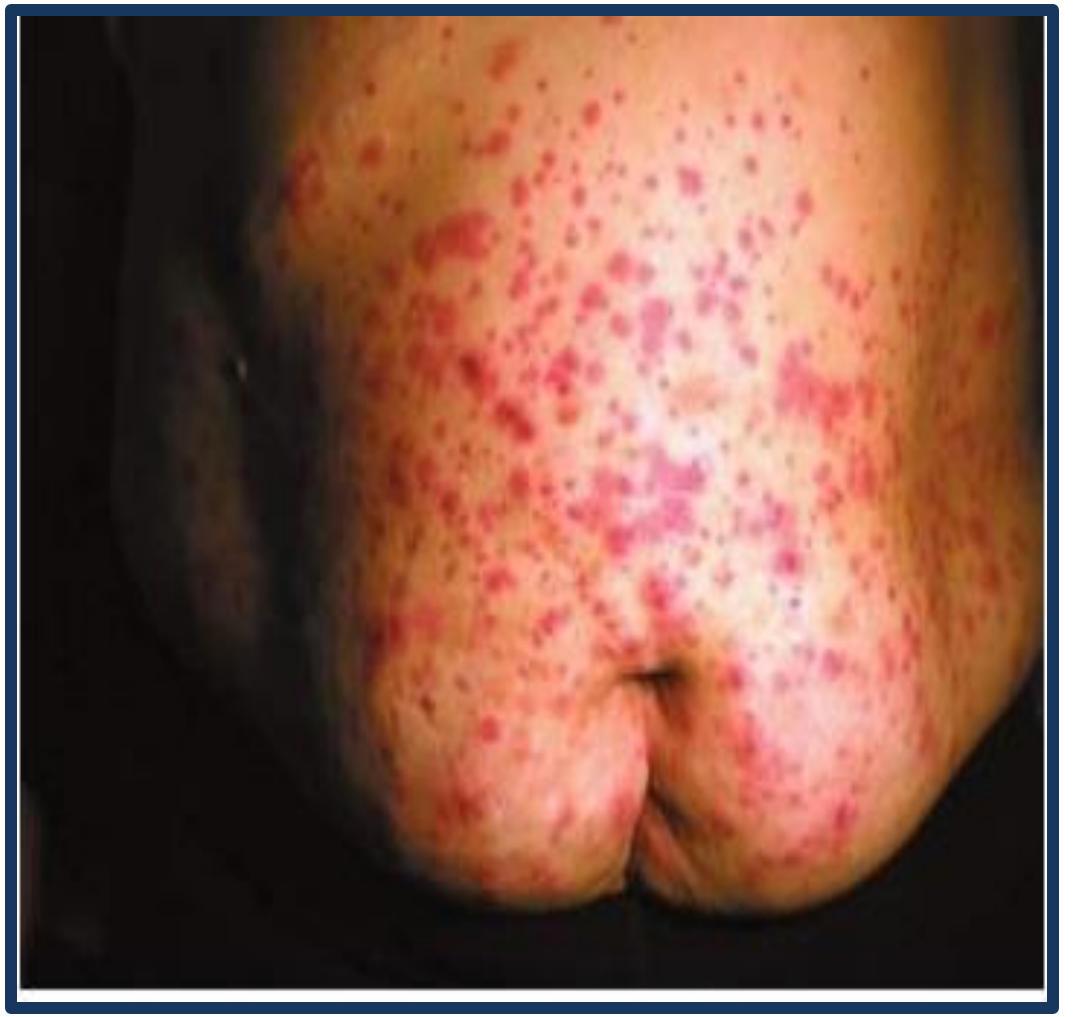
DX: Schamberg disease

F: pigmented purpuric dermatosis on the lower leg showing hemosiderin deposits and a cayenne pepper capillaritis



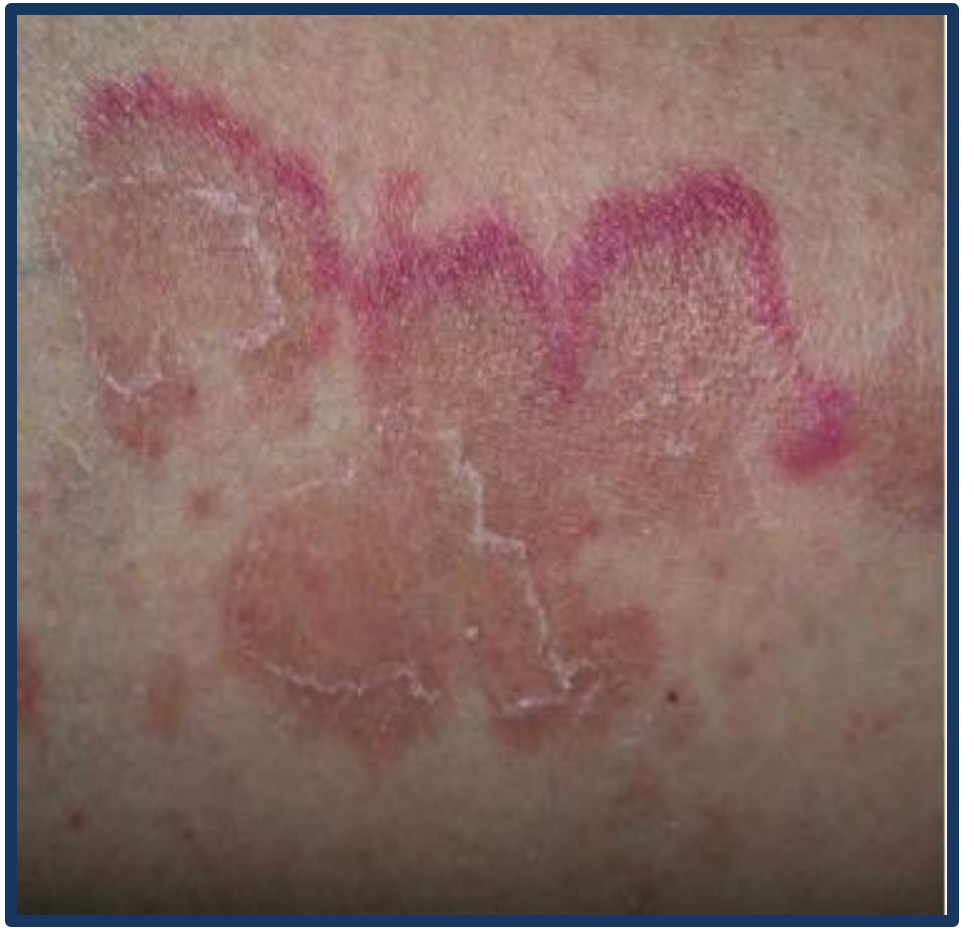
DX: Vasculitis

F: rash on the abdomen



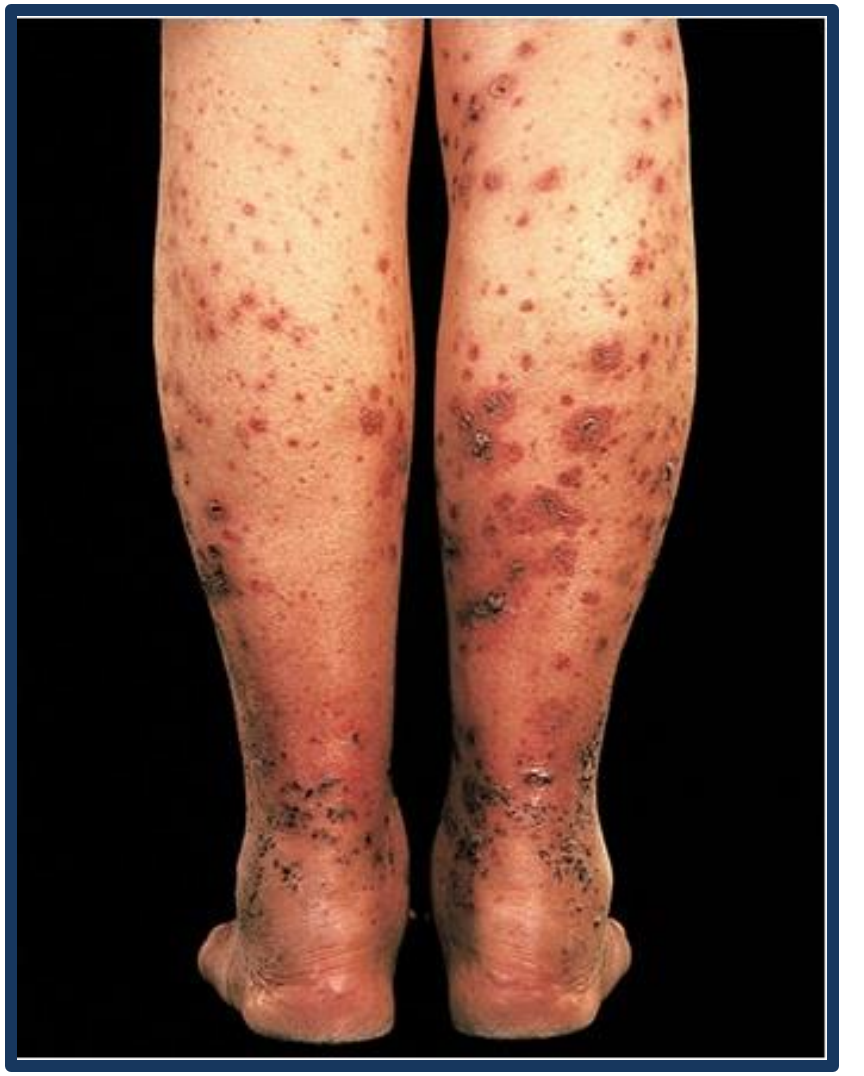
DX: Vasculitis

F: Pigmented purpuric dermatosis of the Majocchi type. Note the annular appearance and the prominent elevated erythematous .borders

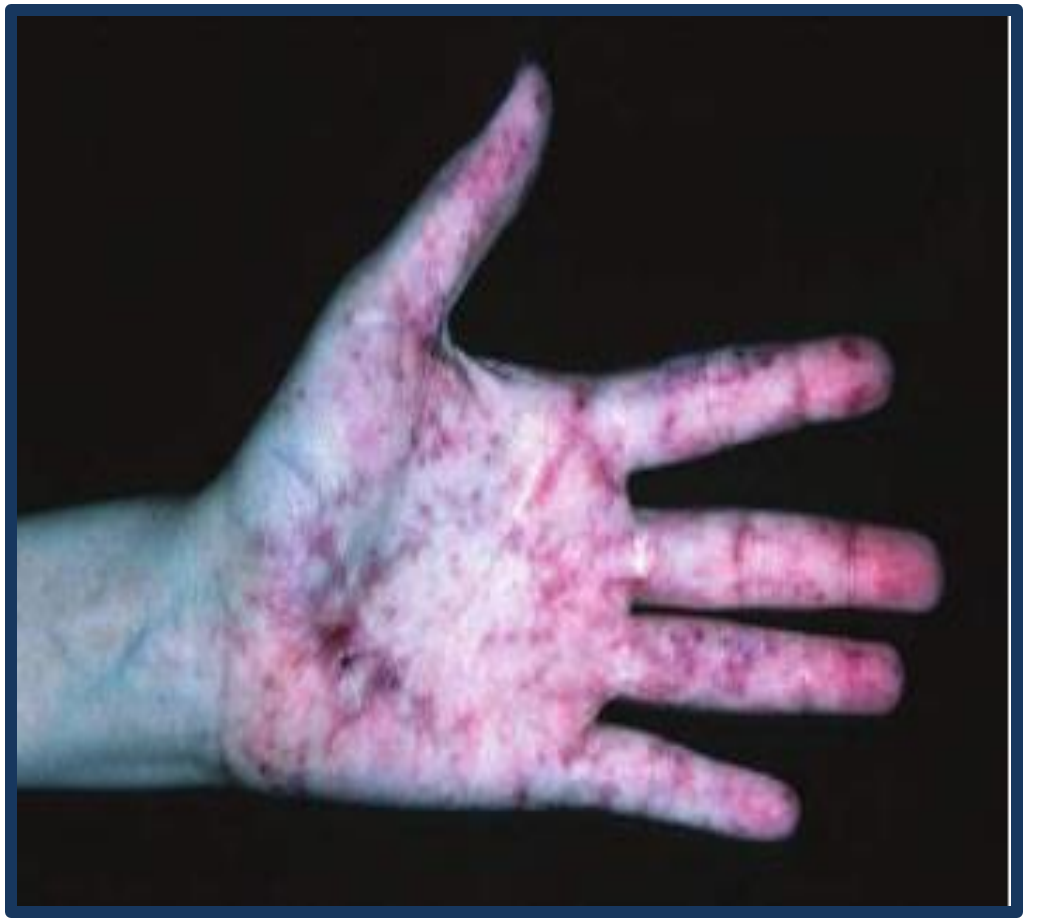


DX: Systemic vasculitis

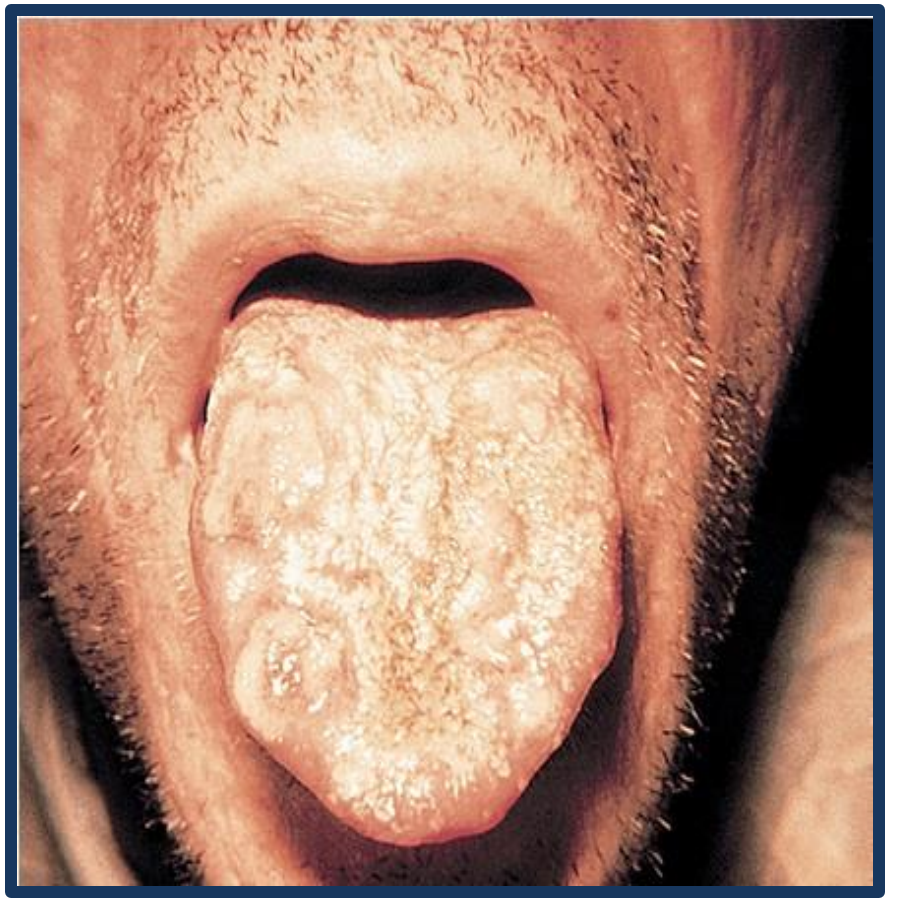
F: Rash (palpable purpura)



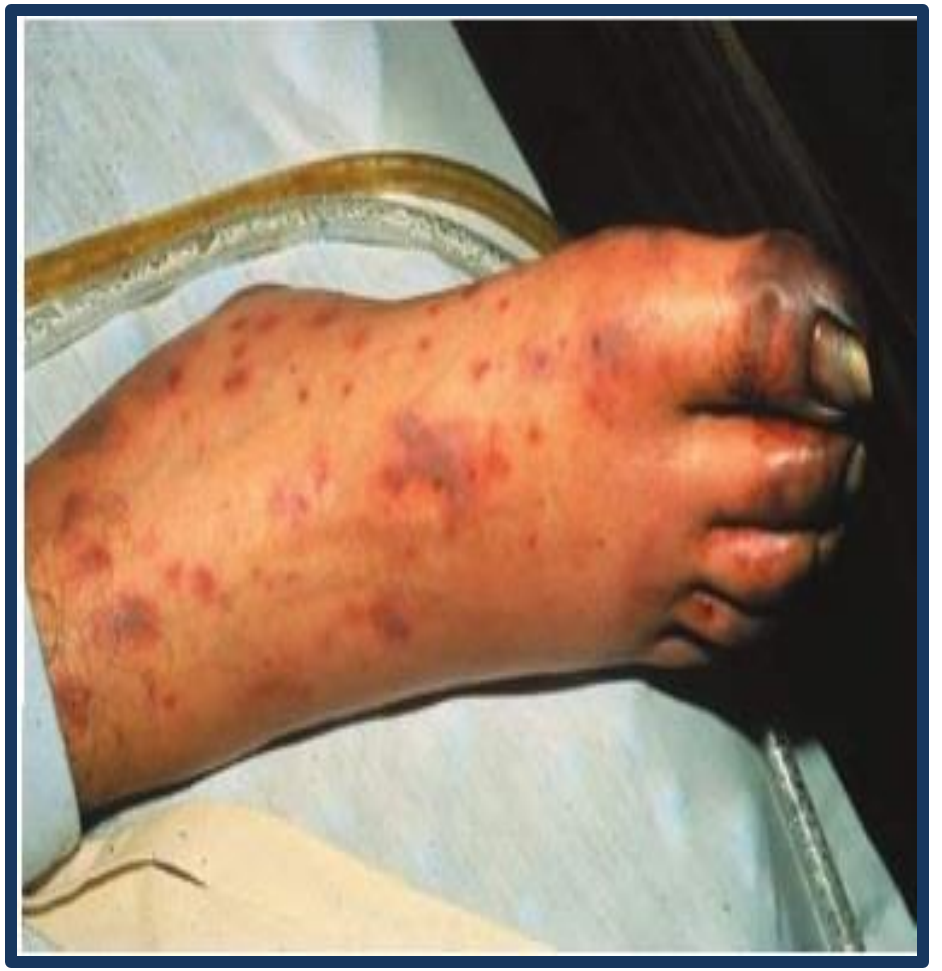
DX: SLE
F: Necrotizing vasculitis



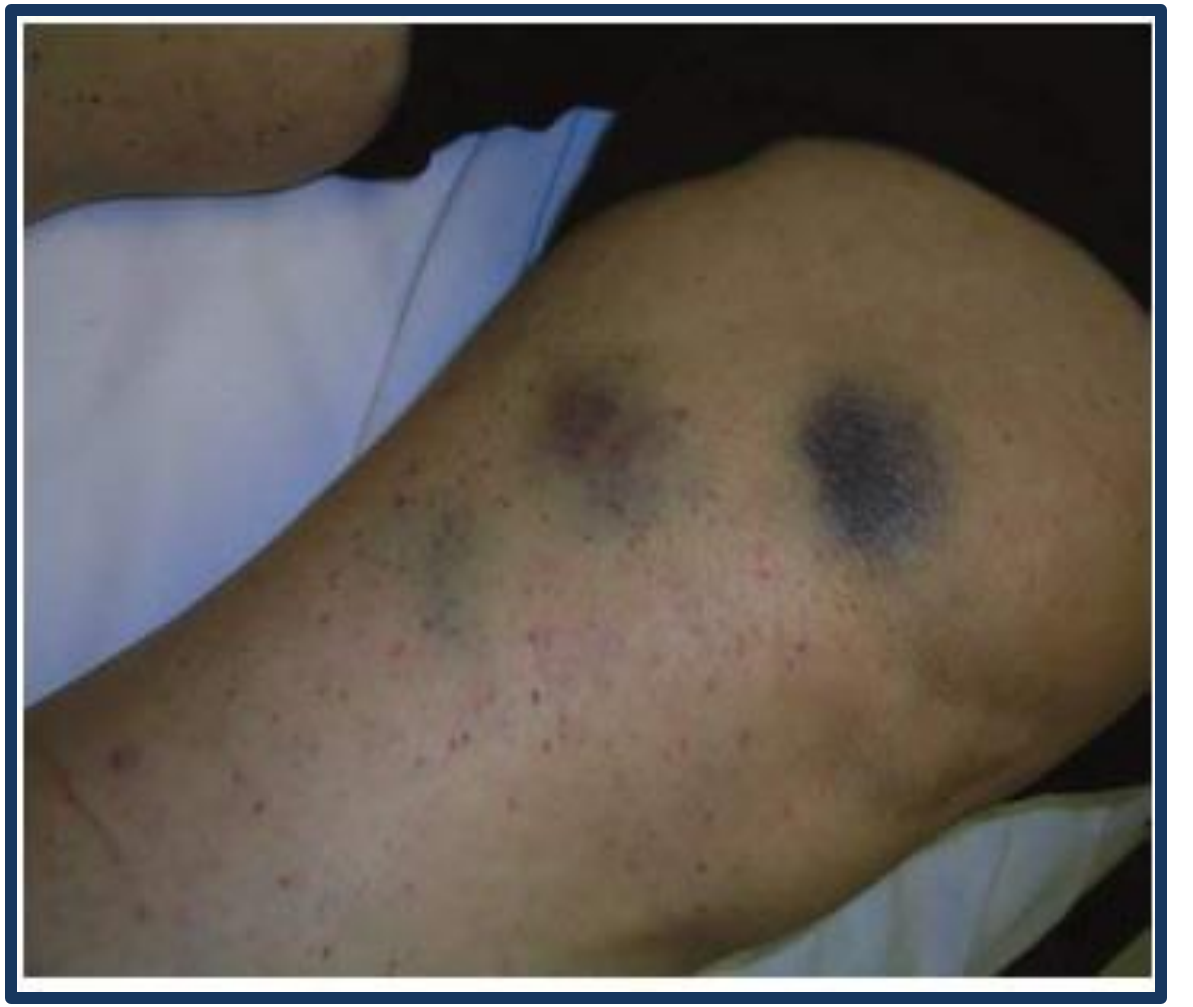
DX: Behcet's syndrome
F: oral ulceration



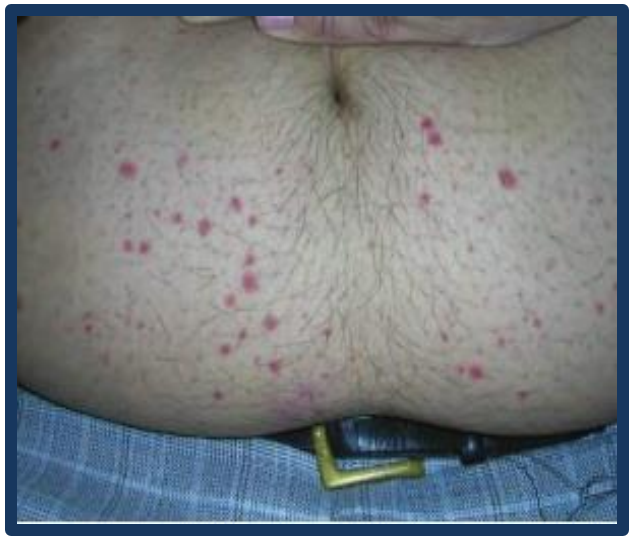
DX: Vasculitis
F: Petechiae , purpura, and acrocyanosis

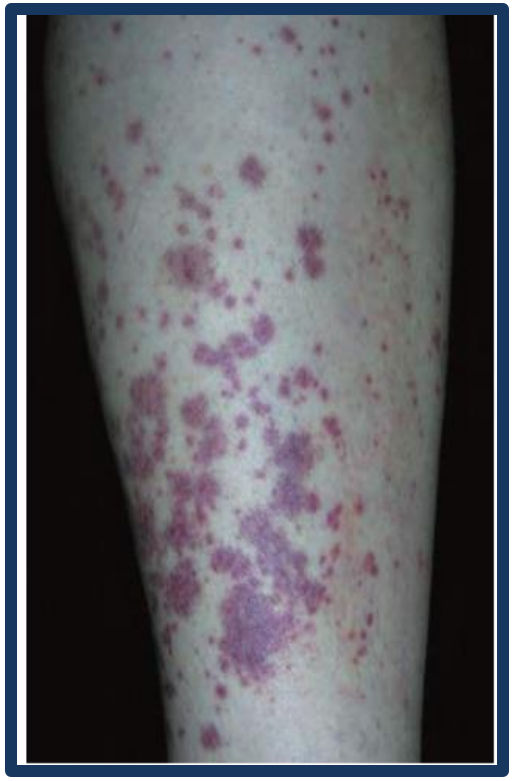


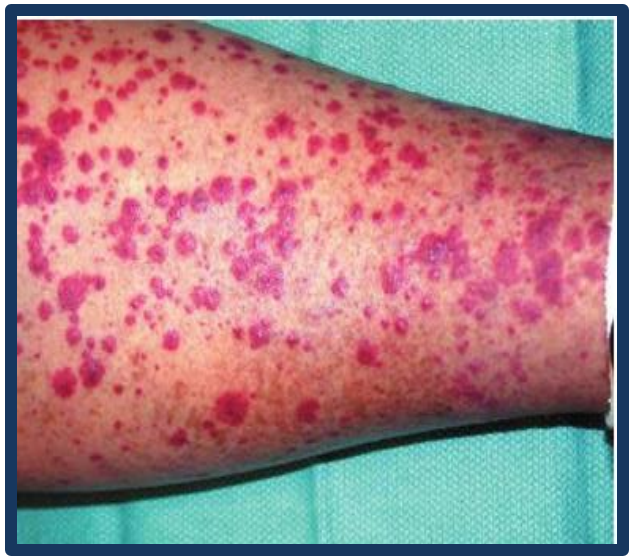
DX: thrombotic thrombocytopenic purpura
F: Petechiae and purpura (not palpabale)



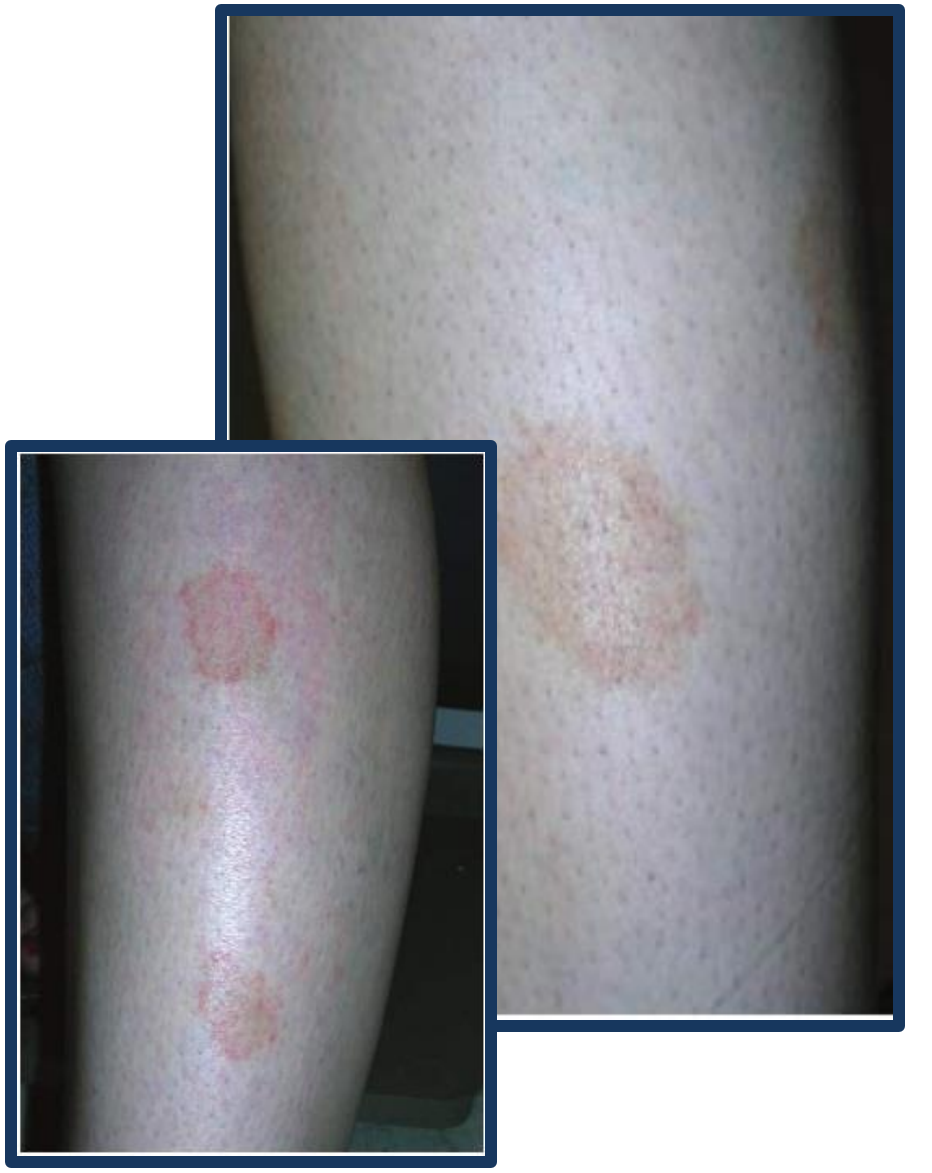
DX: Leukocytoclastic vasculitis
F: Palpable purpura



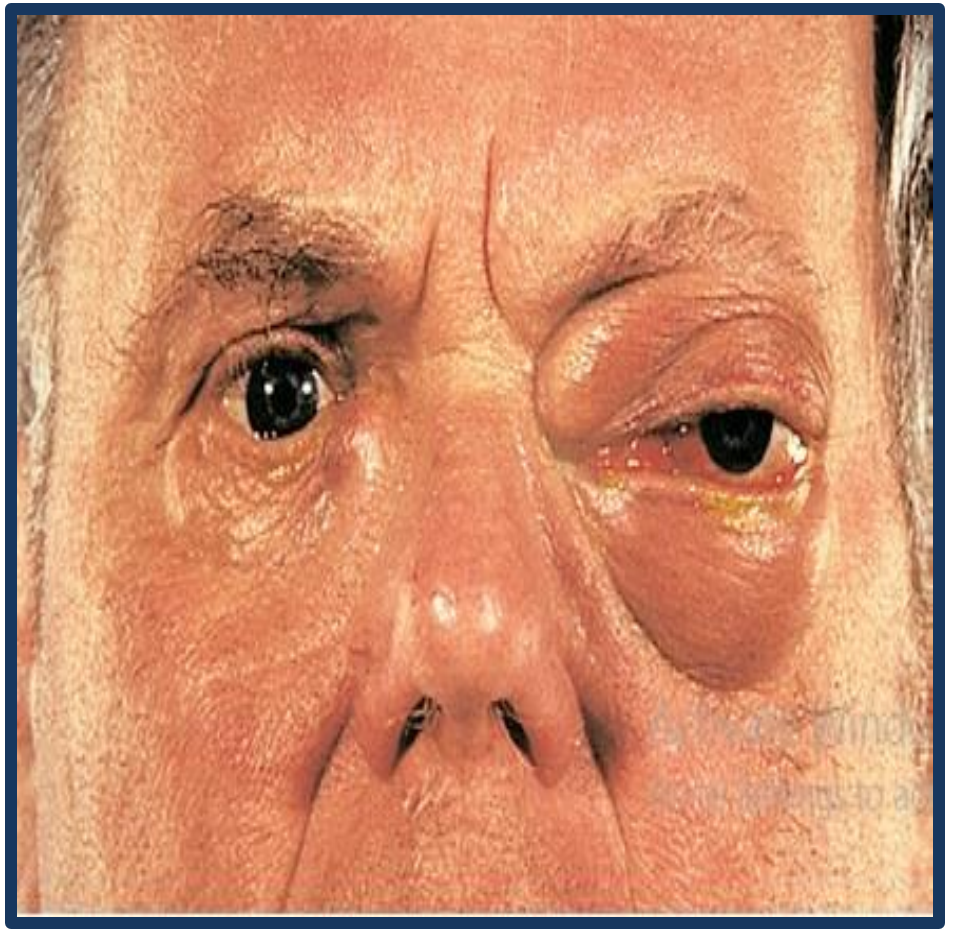




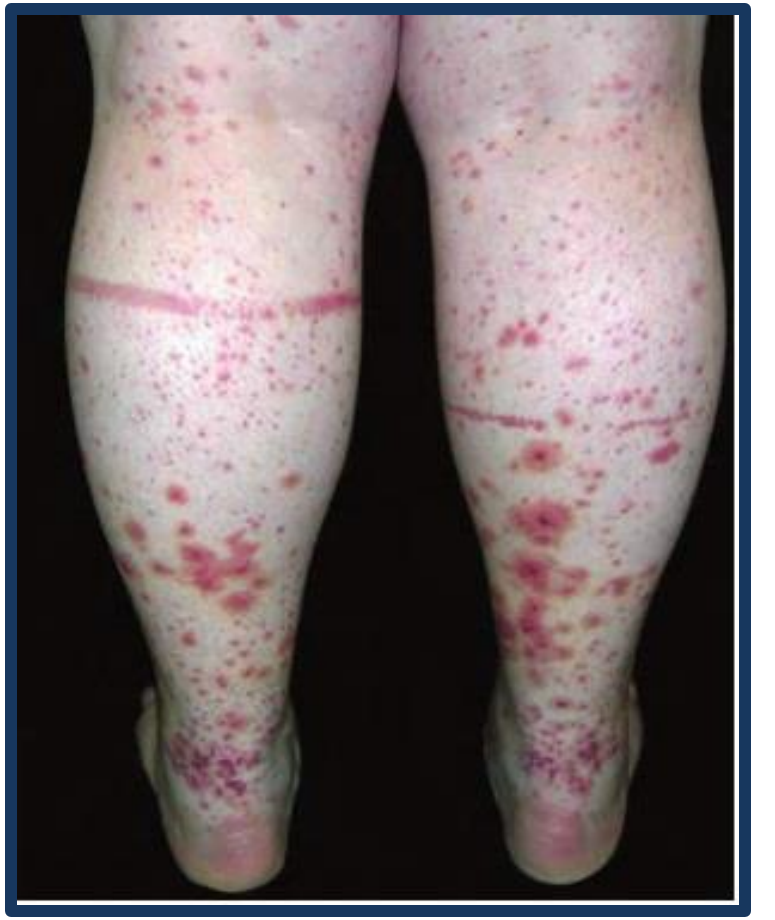
DX: Vasculitis
F: Lichen aureus



DX: wegener's granulomatosis
F: proptosis



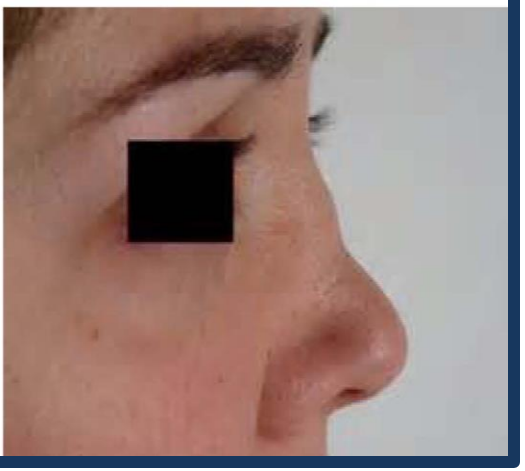
DX: Henoch-Schönlein purpura ,Vasculitis
F: palpable purpura on the lower extremity

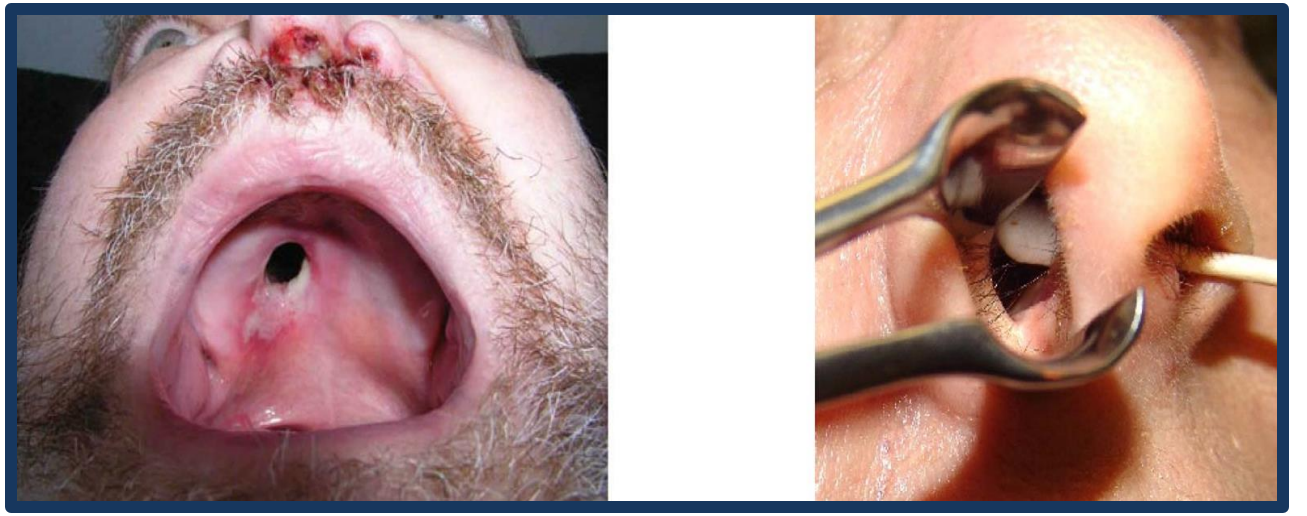


35 y/o male patient, c/o of cough, hematuria and presented with saddle nose.

- What are the autoantibodies associated with this disease?

C-ANCA (PR3-ANCA), or may also rarely be associated with P-ANCA (MPO-ANCA)



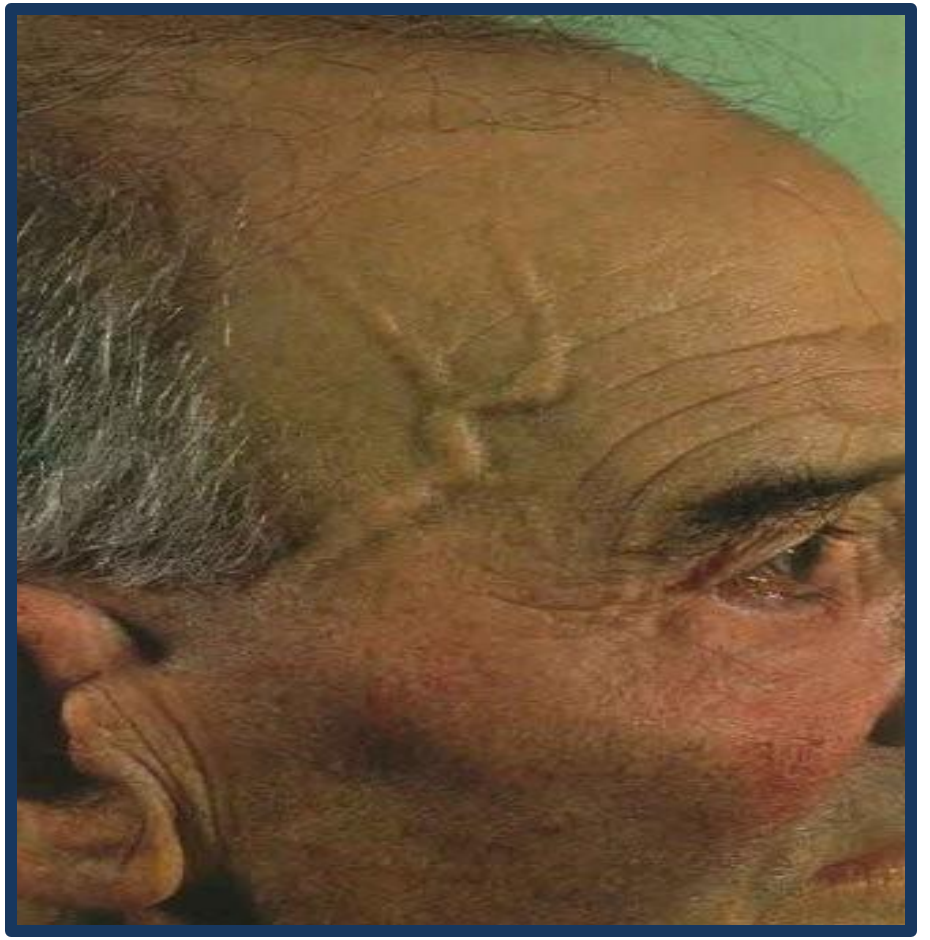


Granulomatosis with polyangiitis (Wegener vasculitis), a similar picture can be seen in patients who take cocaine through inhalation by nose.

Q: A 65 years old male pt complaining of headache.

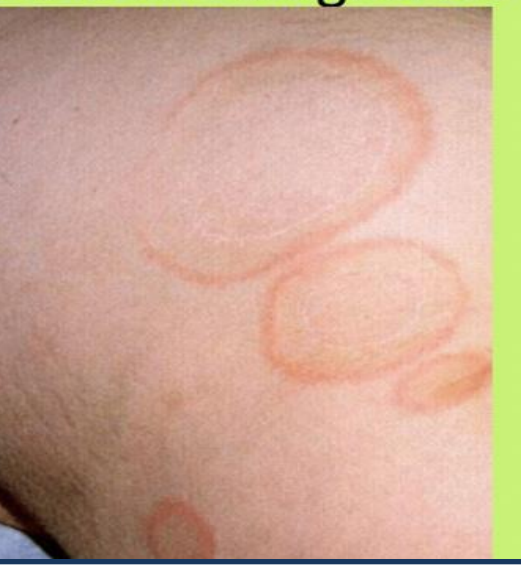
Dx?

Giant cell arteritis.



Q13: A-What is this skin lesion?

B-What is the diagnosis?



A. Erythema marginatum

B. Rheumatic fever

12.Osteoarthritis

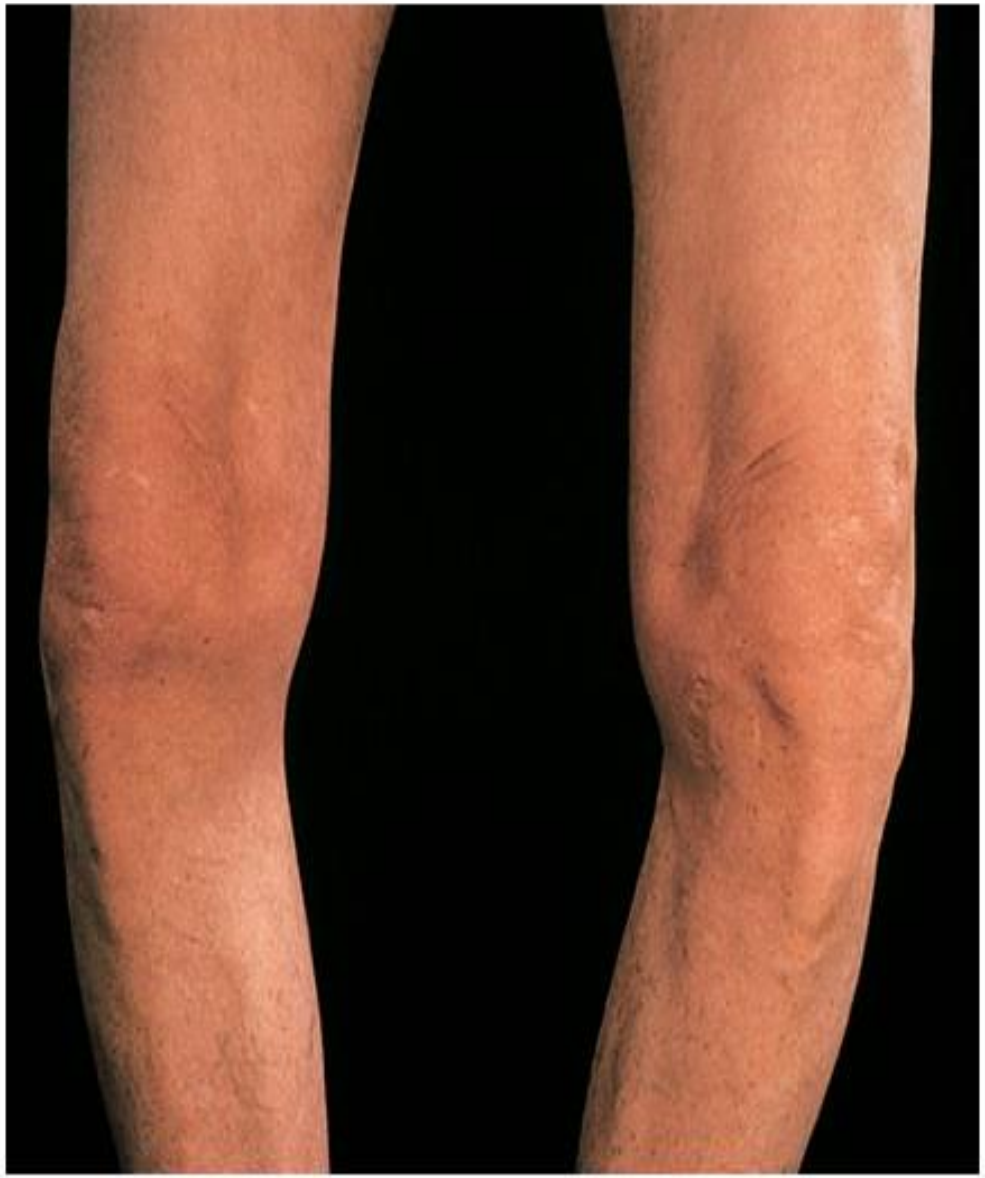


Fig. 25.19 Typical varus deformity resulting from marked medial tibio-femoral osteoarthritis.

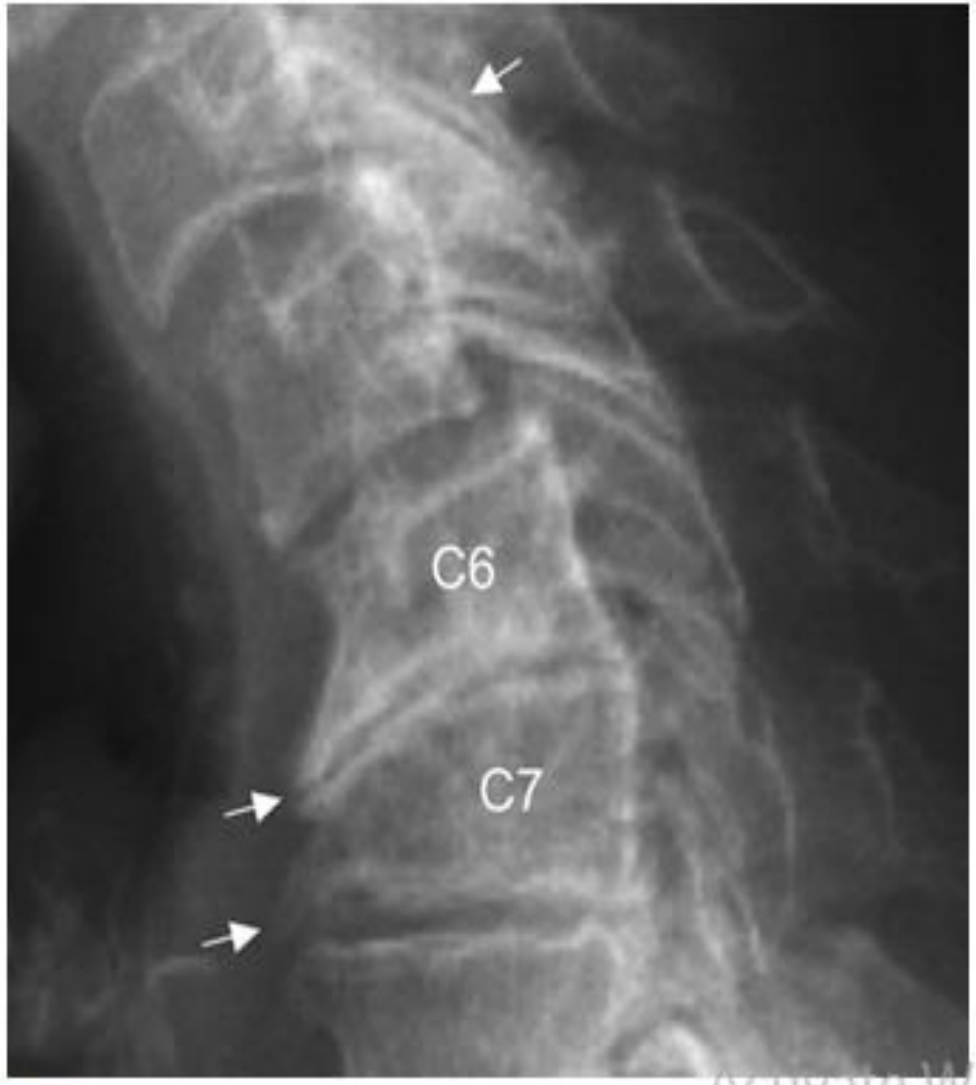


Fig. 25.21 **X-ray of spine showing typical changes of osteoarthritis.** Cervical spondylosis showing disc space narrowing between C6 and C7, osteophytes at the anterior vertebral body margins (arrows) and osteosclerosis at the apophyseal joints.

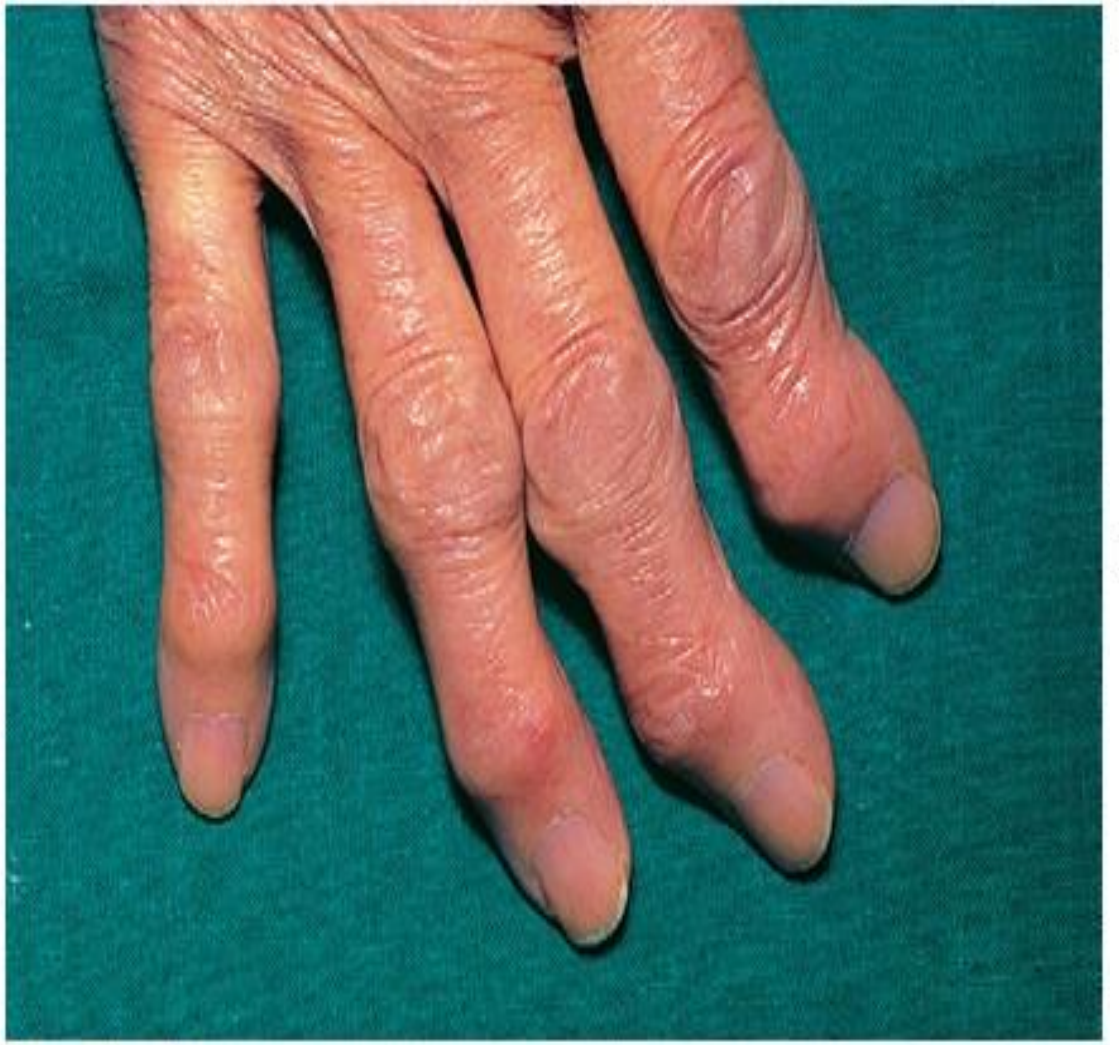


Fig. 25.16 Nodal osteoarthritis. Heberden's nodes and lateral (radial/ulnar) deviation of distal interphalangeal joints, with mild Bouchard's nodes at the proximal interphalangeal joints.

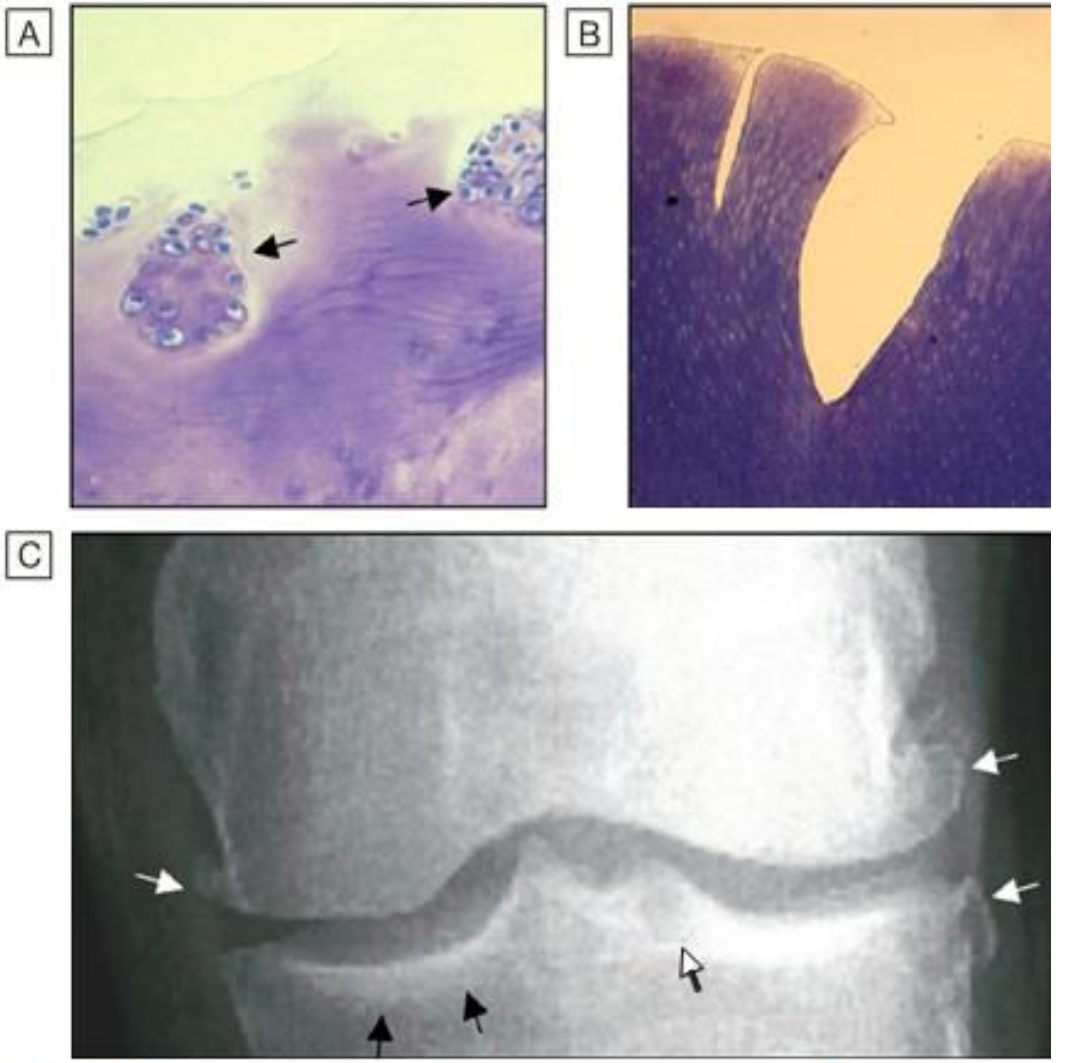


Fig. 25.15 Pathological changes in osteoarthritis. A Abnormal nests of proliferating chondrocytes (arrows) interspersed with matrix devoid of normal chondrocytes. B Fibrillation of cartilage in OA. C Radiograph of knee joint affected by OA, showing osteophytes at joint margin (white arrows), subchondral sclerosis (black arrows) and subchondral cyst (open arrow).

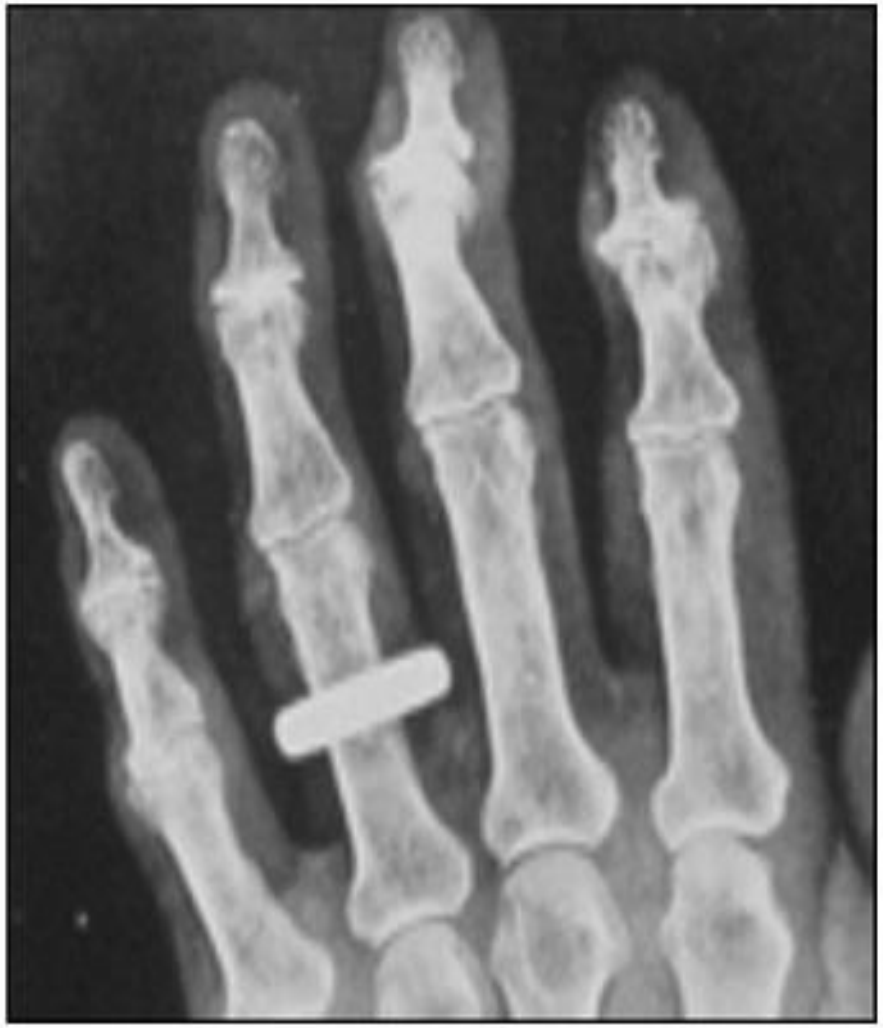


Fig. 25.17 X-ray appearances in hand osteoarthritis. There is marked loss of joint space at all of the distal interphalangeal joints, with osteophyte formation most marked at the first and second DIP joints. The fifth proximal interphalangeal joint also shows loss of joint space with osteophyte formation.

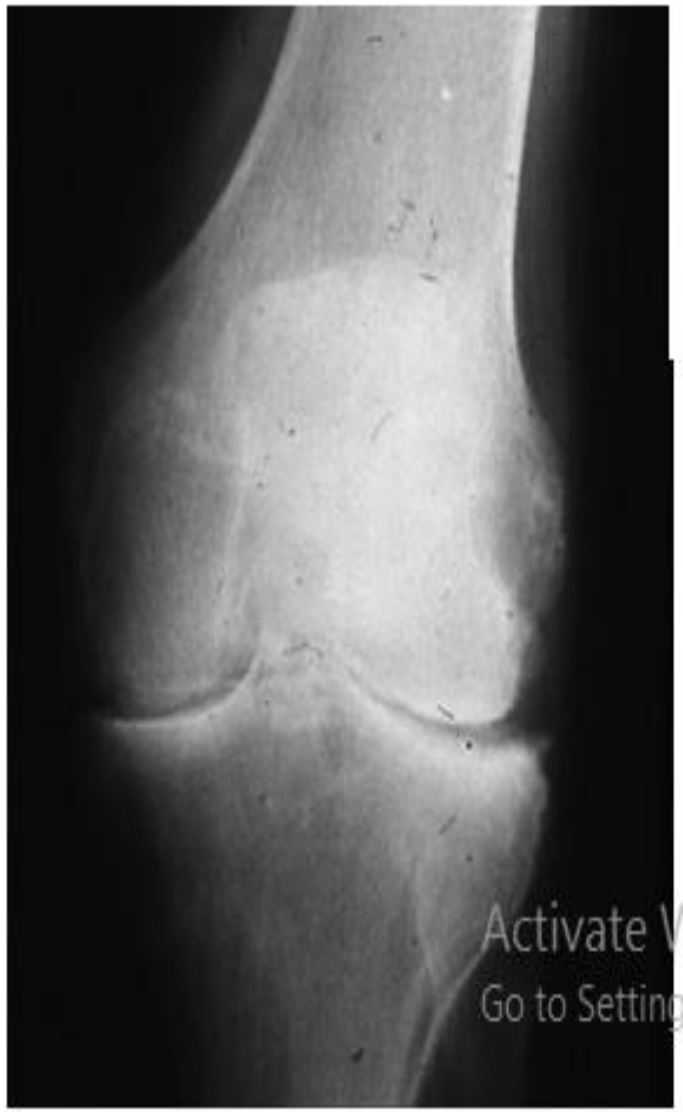


Fig. 25.18 X-ray appearances in knee osteoarthritis. There is almost complete loss of joint space affecting both compartments, and sclerosis of subchondral bone.

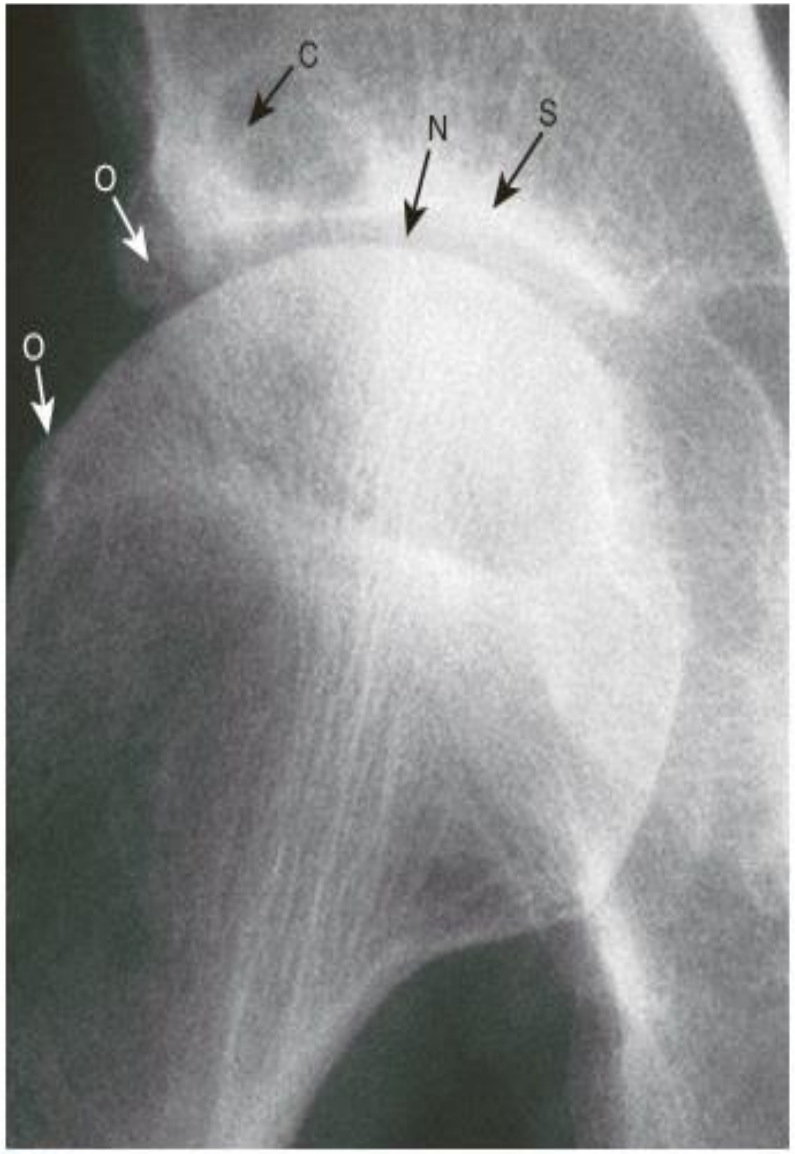


Fig. 25.20 X-ray of hip showing changes of osteoarthritis. Note the superior joint space narrowing (N), subchondral sclerosis (S), marginal osteophytes (white arrows) and cysts (C).

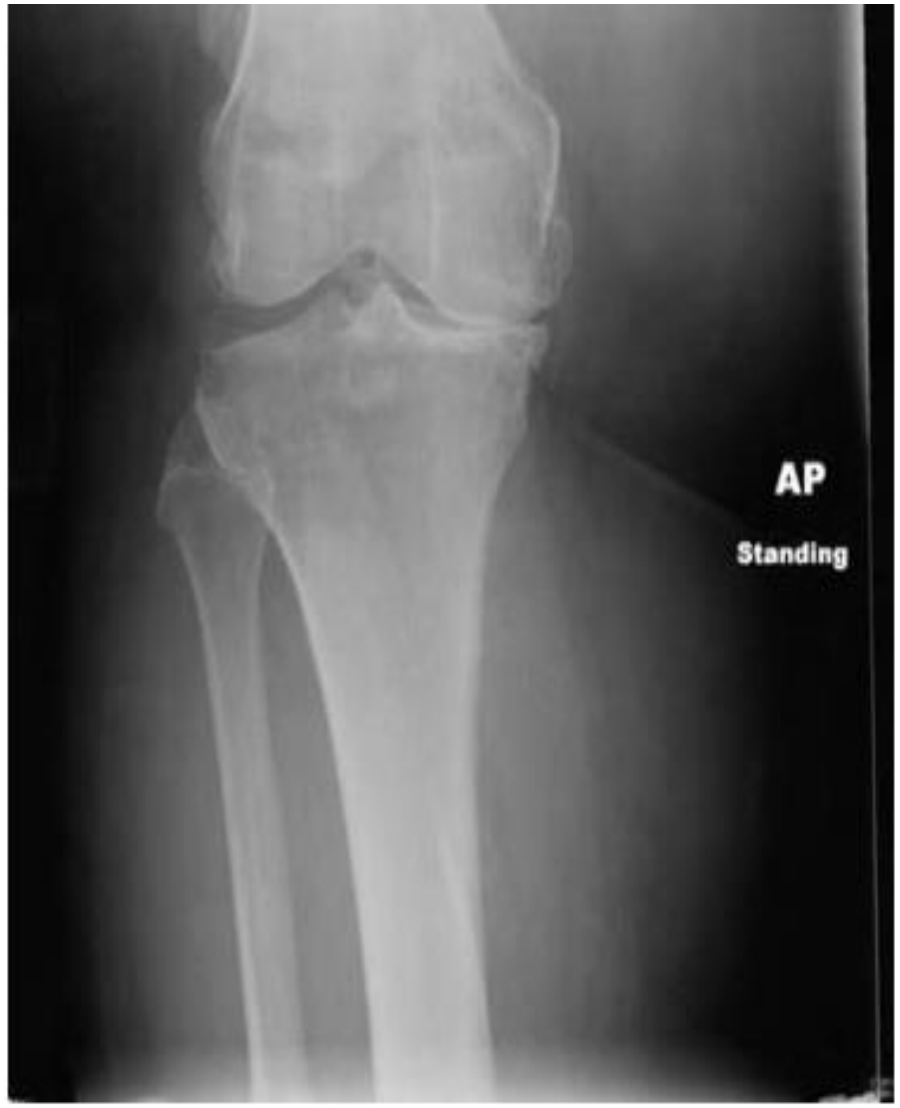


FIGURE 98-3 Osteoarthritis of the knee causing joint space narrowing, sclerosis, and bony spurring in all 3 compartments of the right knee, most pronounced in the medial compartment. (Reproduced with permission from Heidi Chumley, MD.)

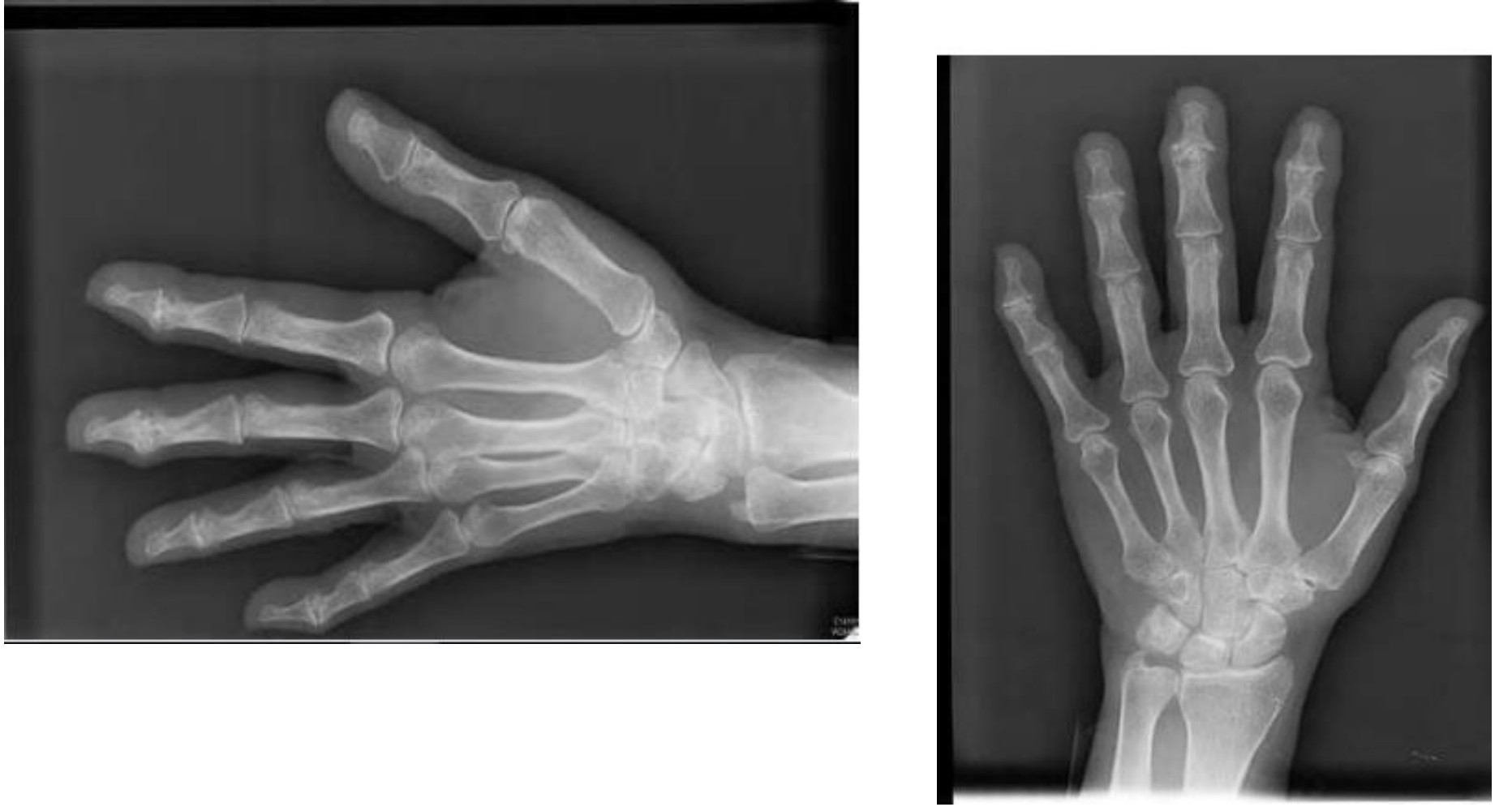


FIGURE 98-2 Joint space narrowing, marginal osteophytes, and Heberden nodes at the distal interphalangeal joints of the second through fifth fingers. (Reproduced with permission from Heidi Chumley, MD.)

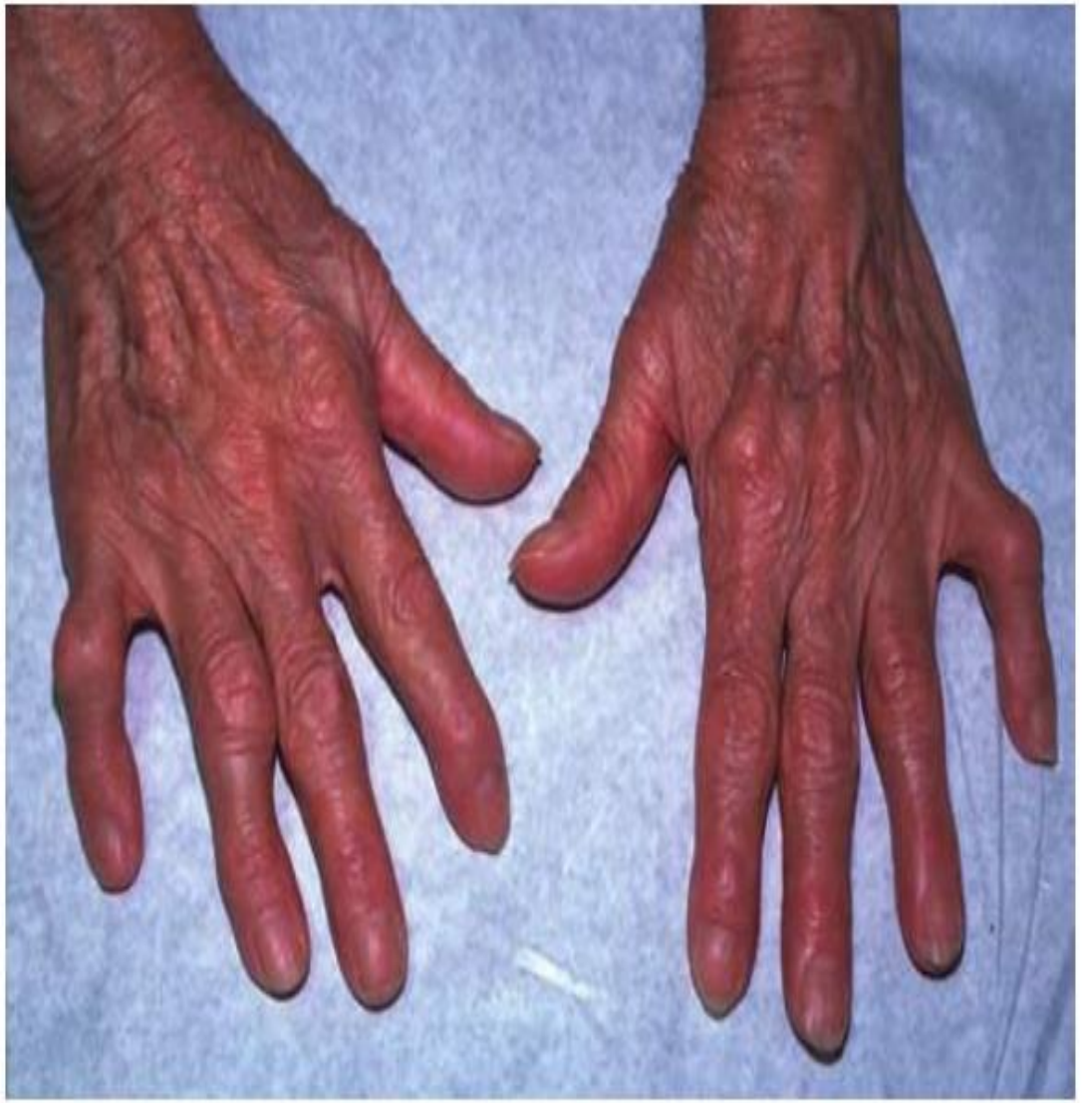


FIGURE 98-1 Bony enlargement of some distal interphalangeal (DIP) and proximal interphalangeal (PIP) joints consistent with Heberden (DIP) and Bouchard (PIP) nodes. (Reproduced with permission from Richard P. Usatine, MD.)

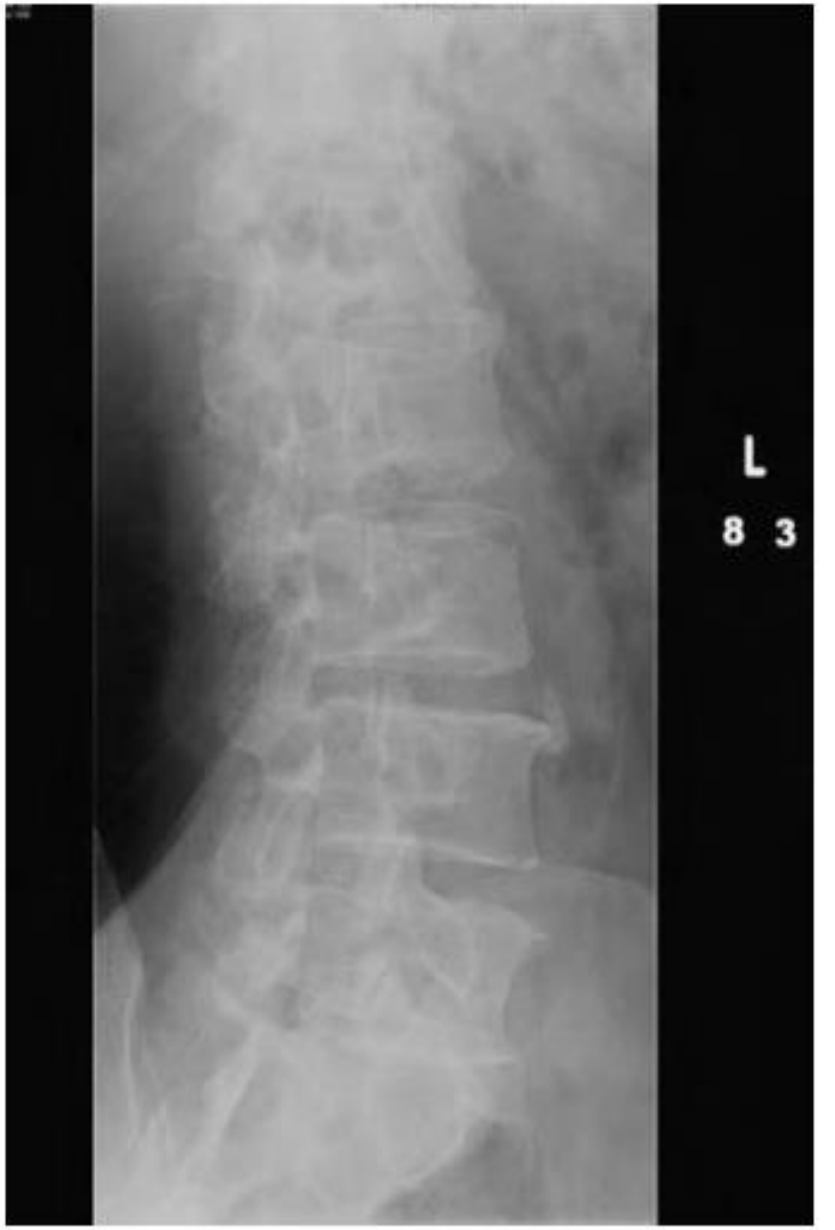


FIGURE 98-5 Loss of disc space and facet arthropathy at L5-S1 and small osteophytes, best seen on L4 and L5. These changes are caused by osteoarthritis. (Reproduced with permission from Heidi Chumley, MD.)

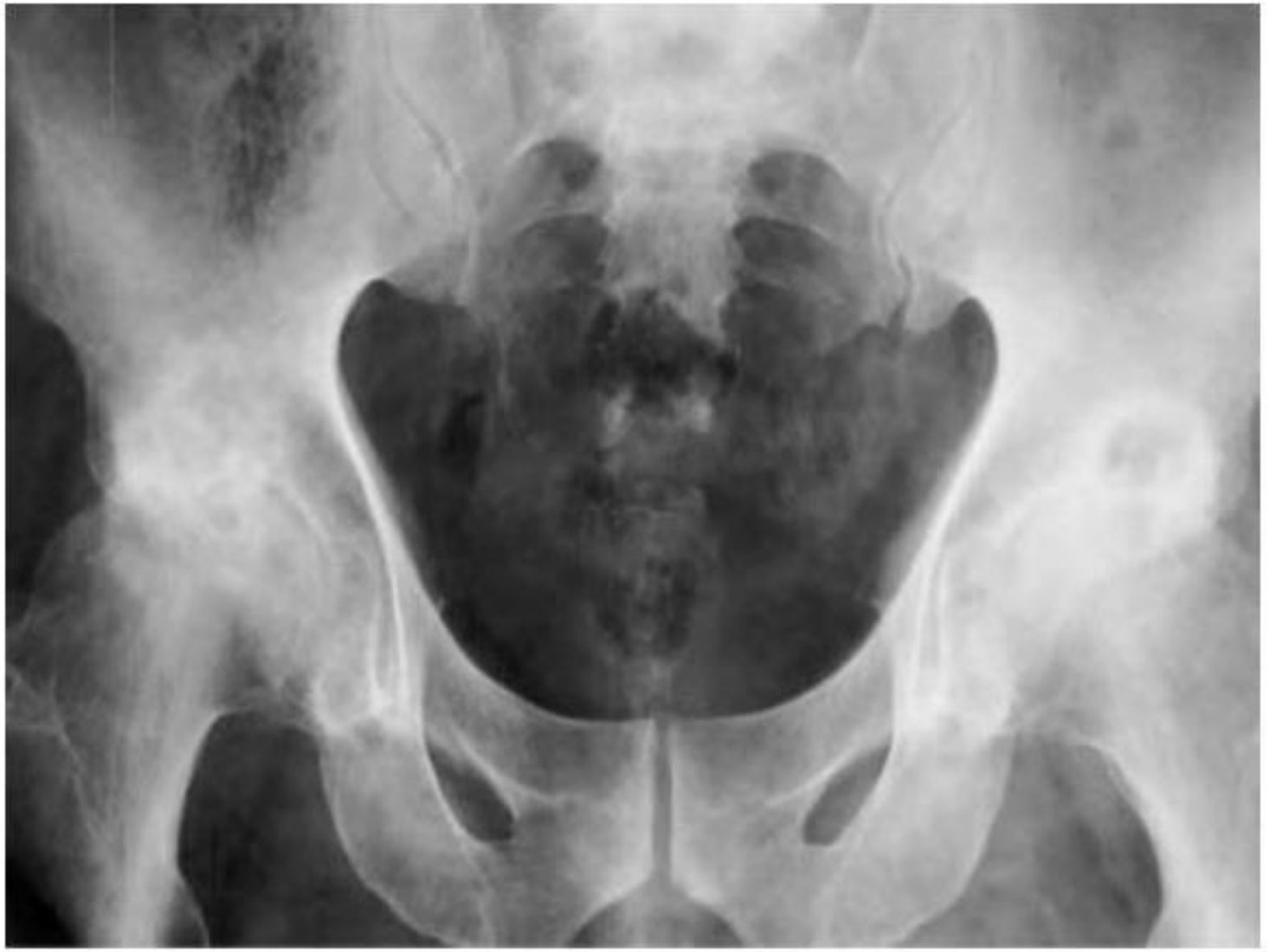
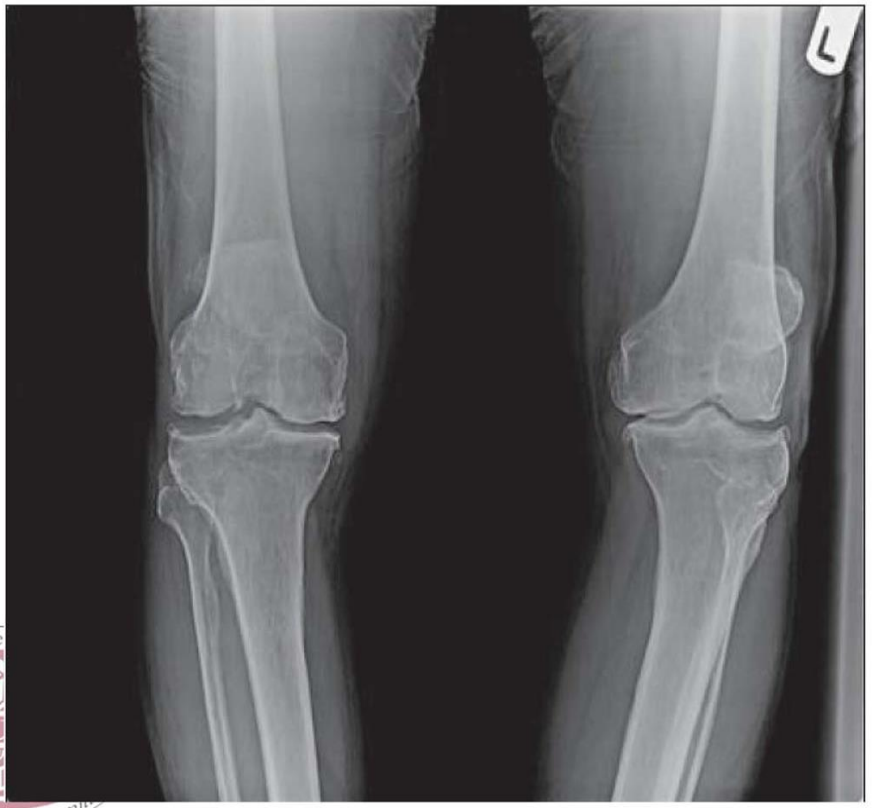
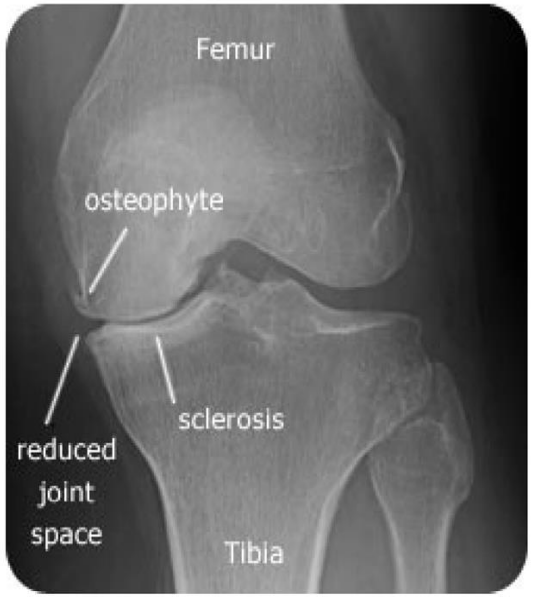


FIGURE 98-4 Articular space narrowing, sclerosis, and subchondral cyst formation of both hips because of osteoarthritis. (Reproduced with permission from Chen MYM, Pope TL Jr, Ott DJ. Basic Radiology. McGraw-Hill; 2004:189, Figure 7-34.)

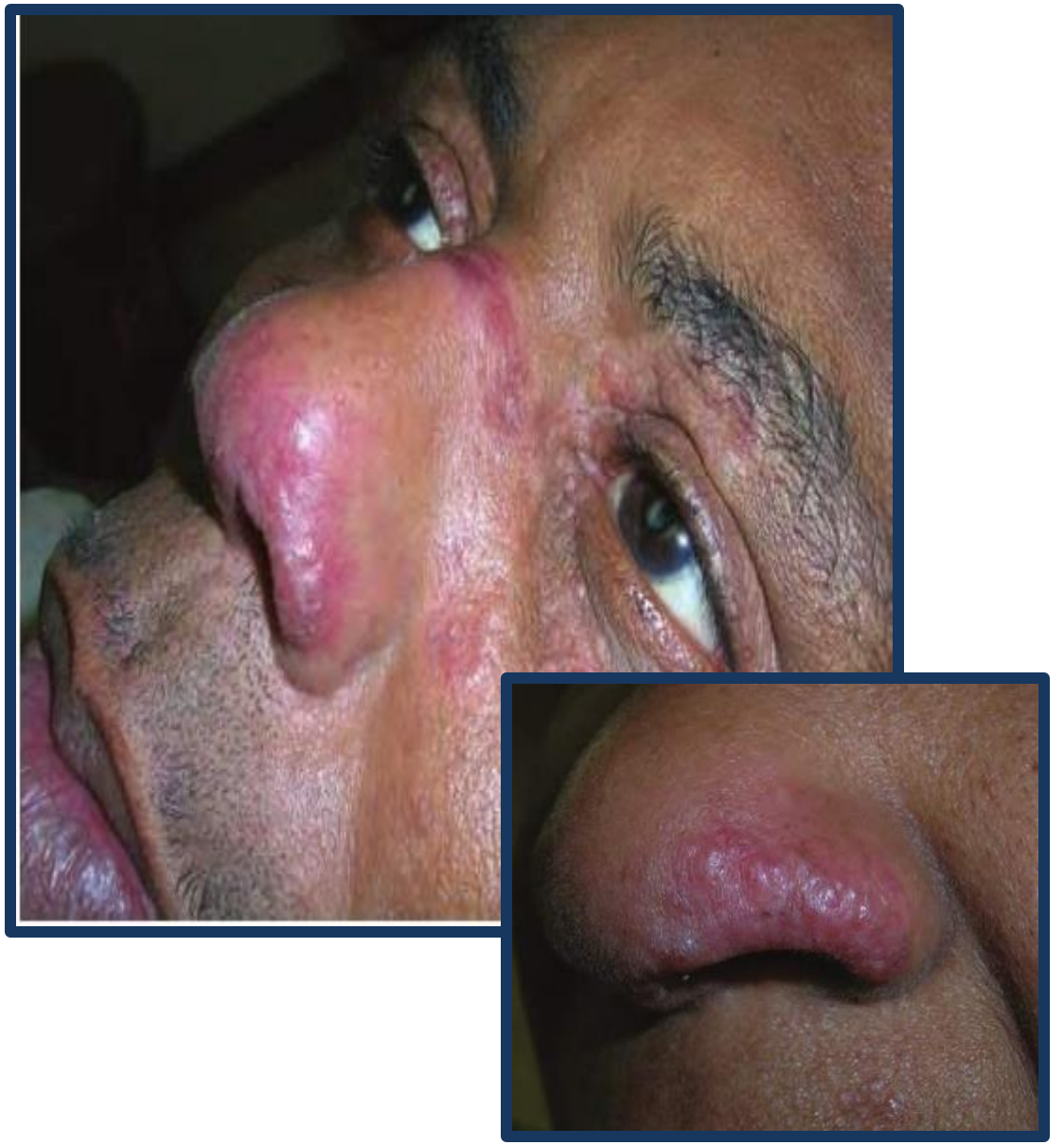
Osteoarthritis



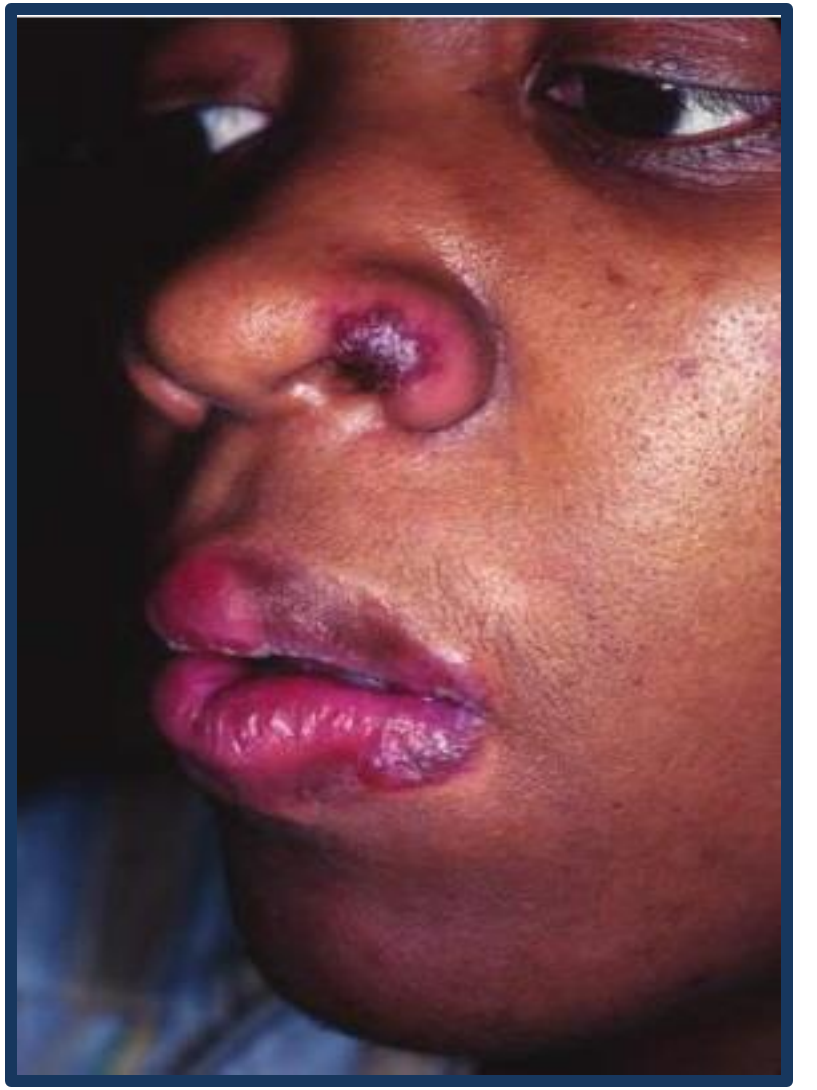


13.Sarcoidosis

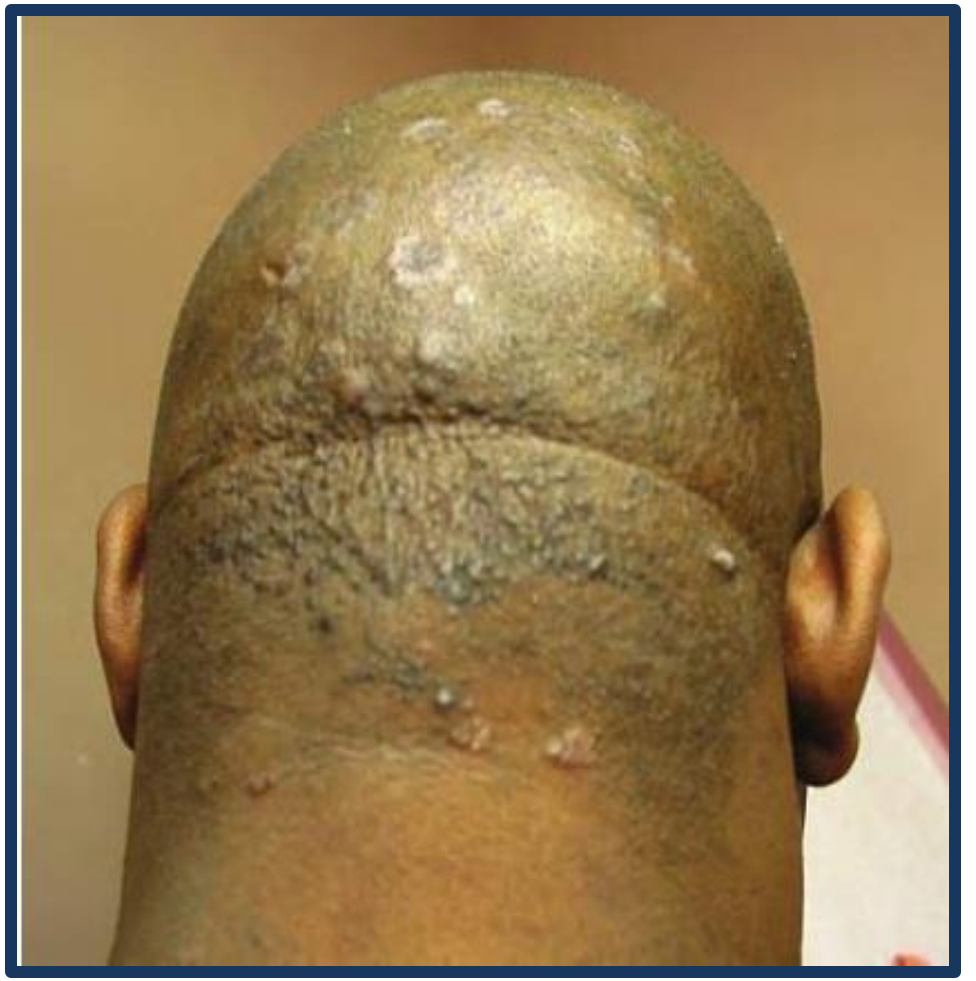
DX: Sarcoidosis
F: lupus pernio involves the nasal rim.



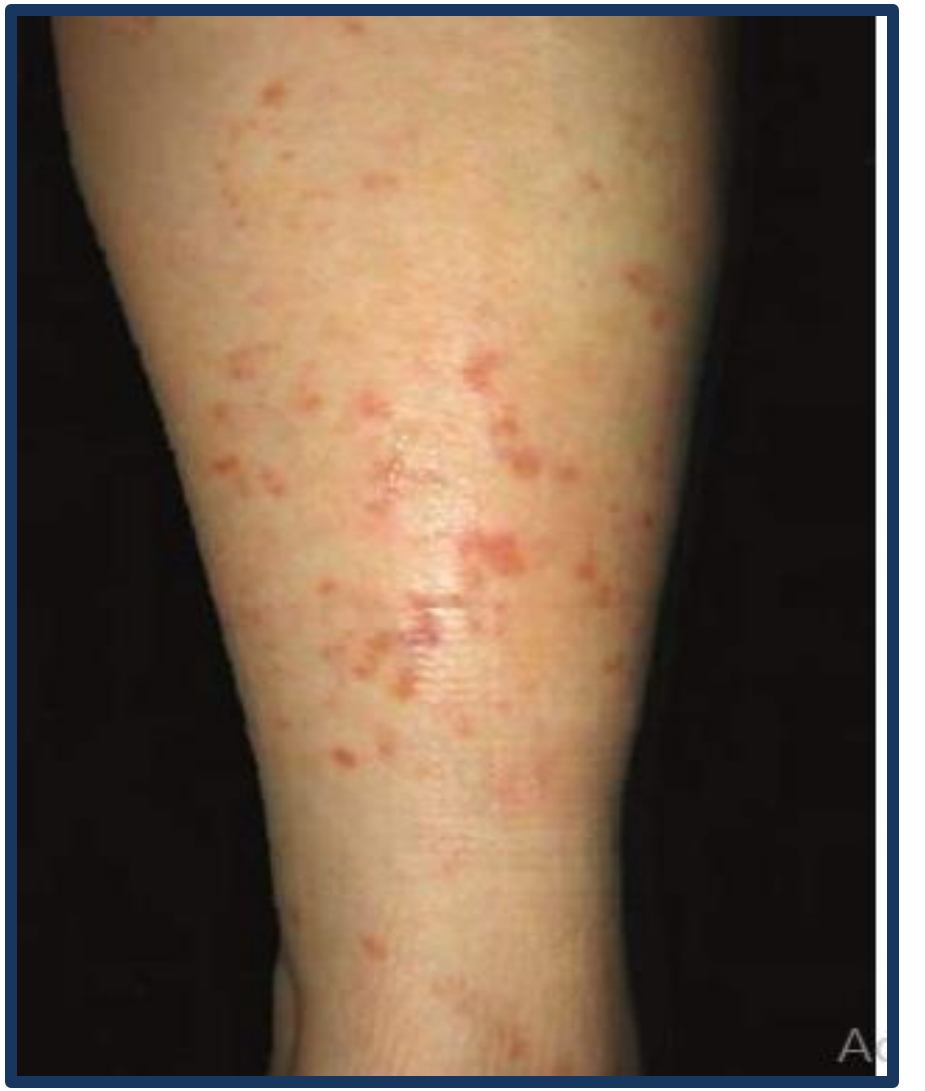
DX: Sarcoidosis
F: Lupus pernio with red papules and plaques on the nose and lips.



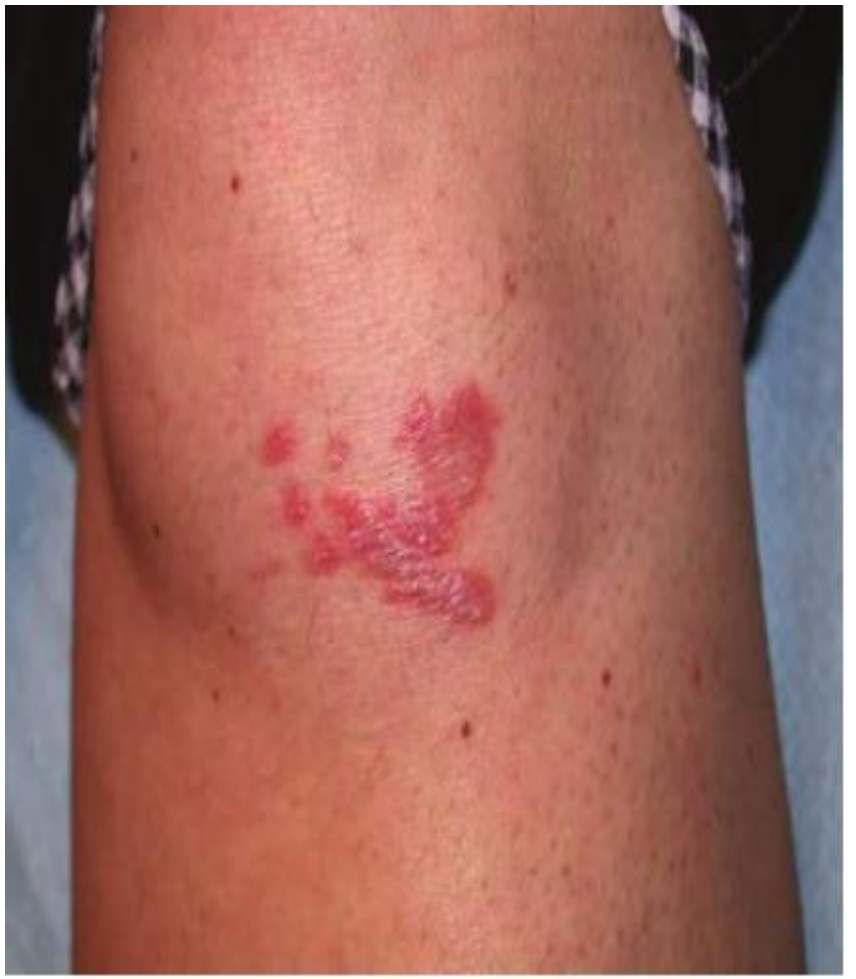
DX: sarcoidosis
F: papular and annular lesions on the scalp and neck.



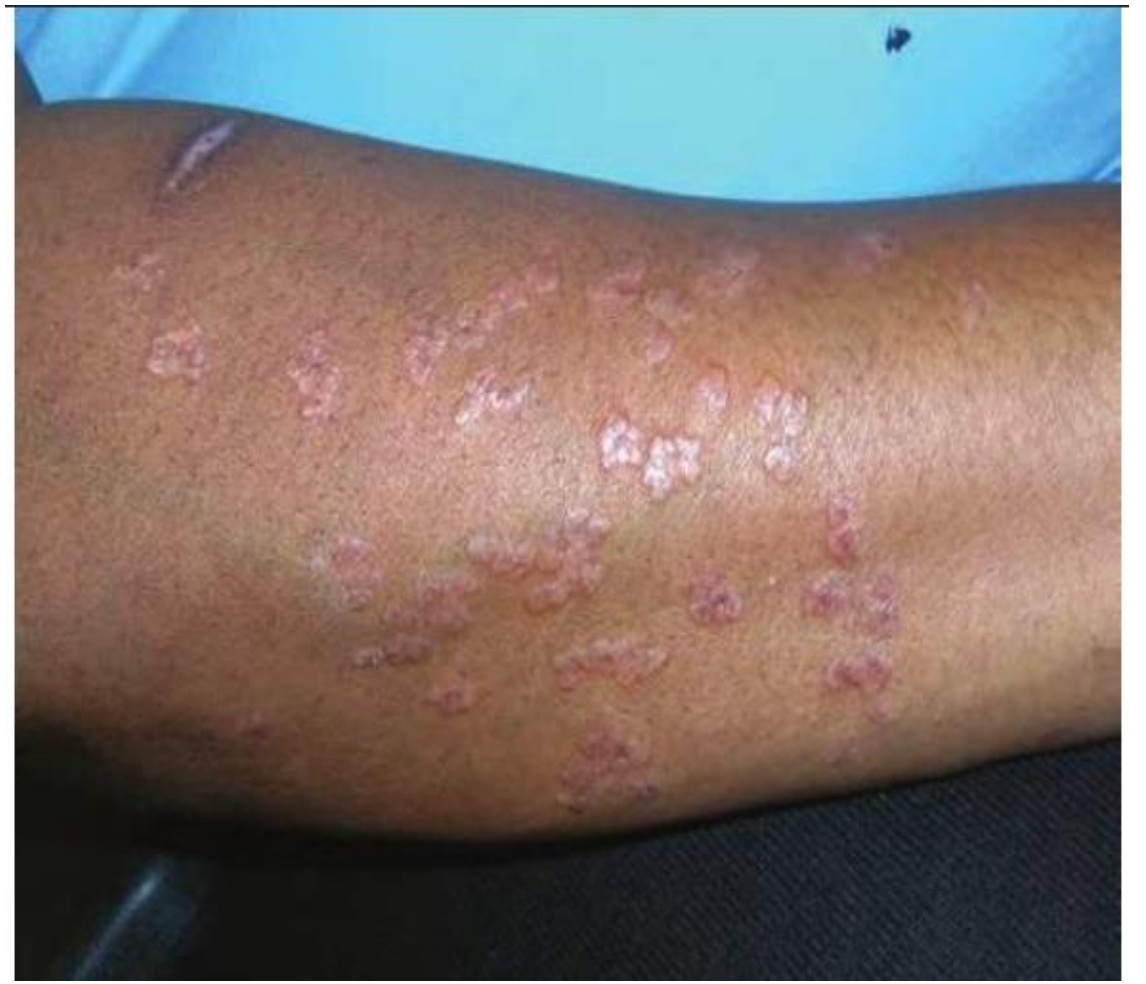
DX: Sarcoidosis
F: maculopapular lesions.



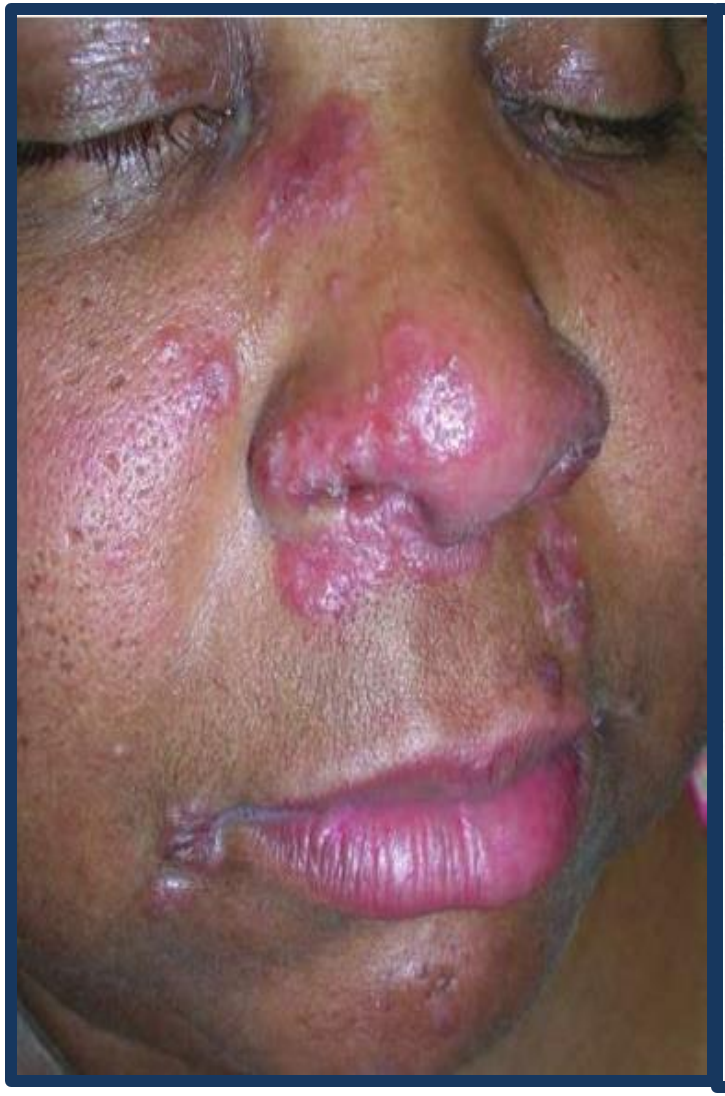
DX: sarcoidosis
F: red plaque of the knee after trauma



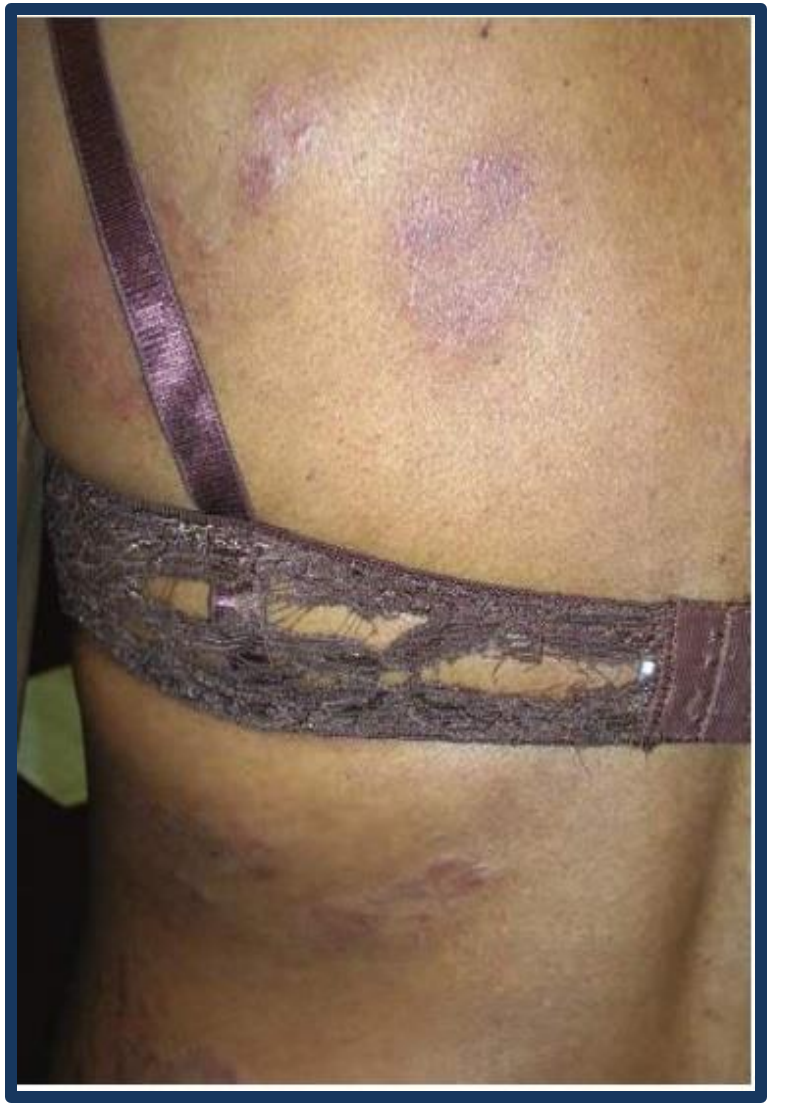
DX: sarcoidosis
F: granulomatous plaques



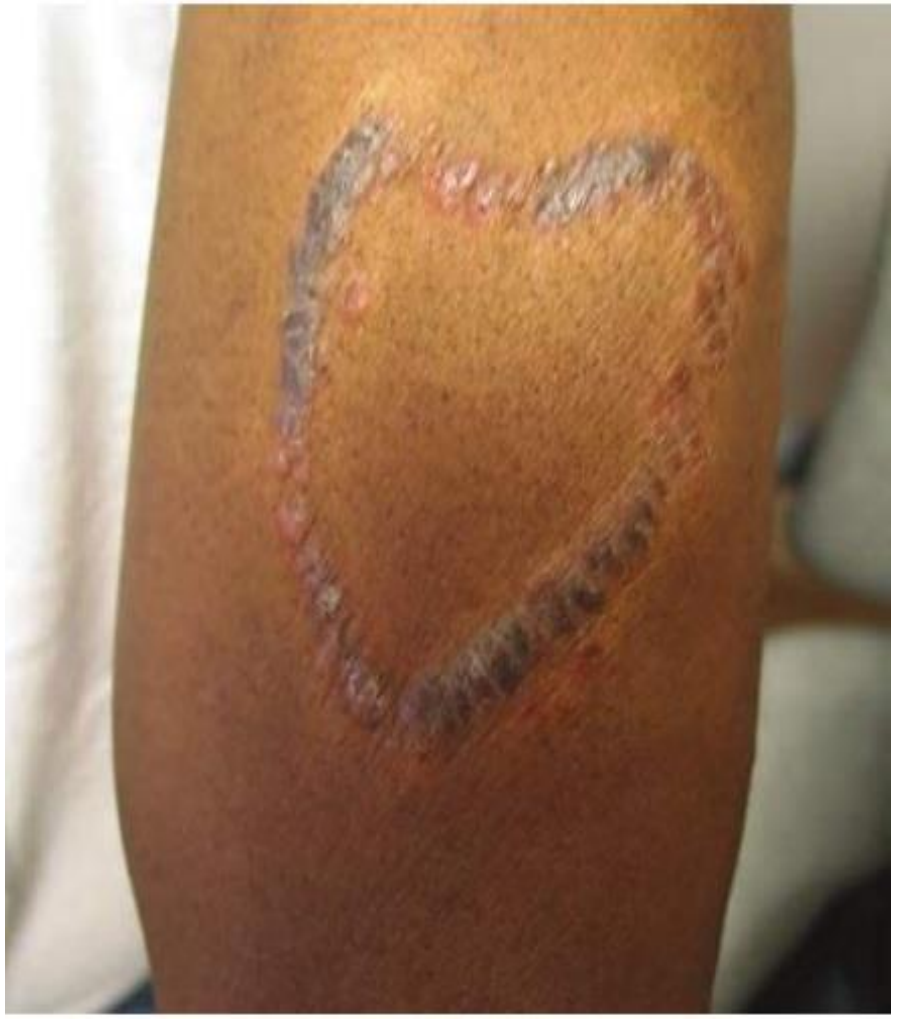
Dx: sarcoidosis
F: lupus pernio



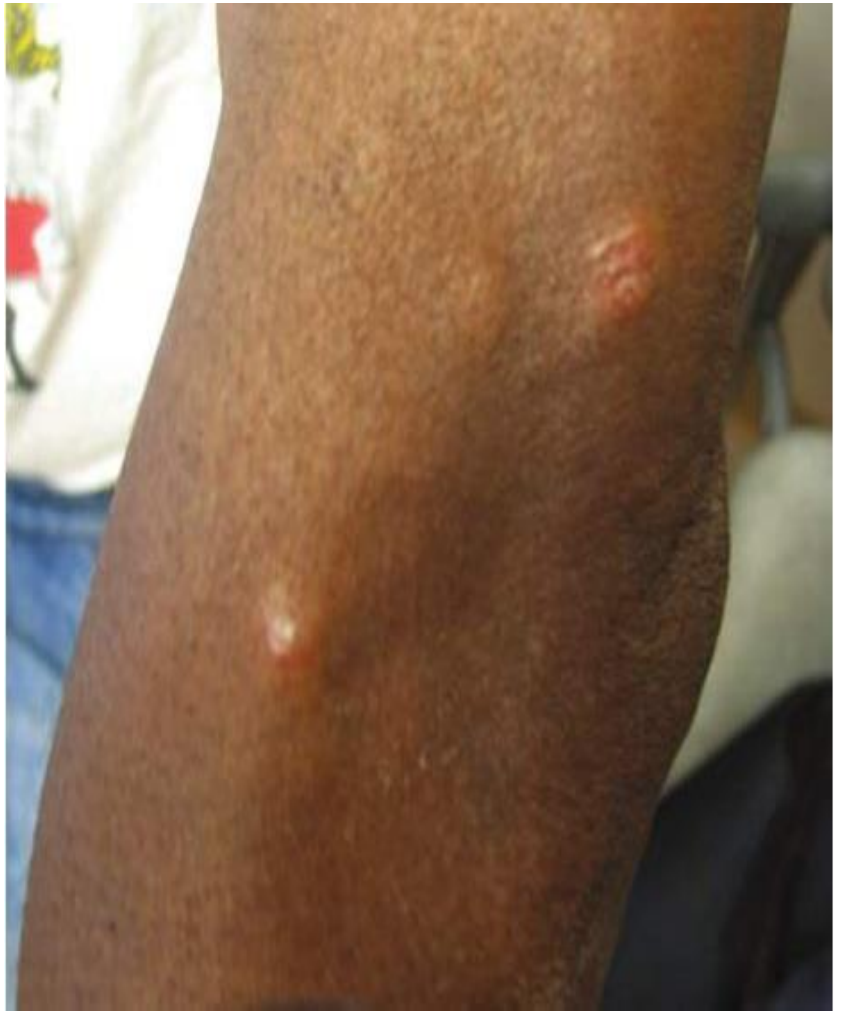
DX: sarcodosis
F: violaceous papules coalescing into annular plaques on the back



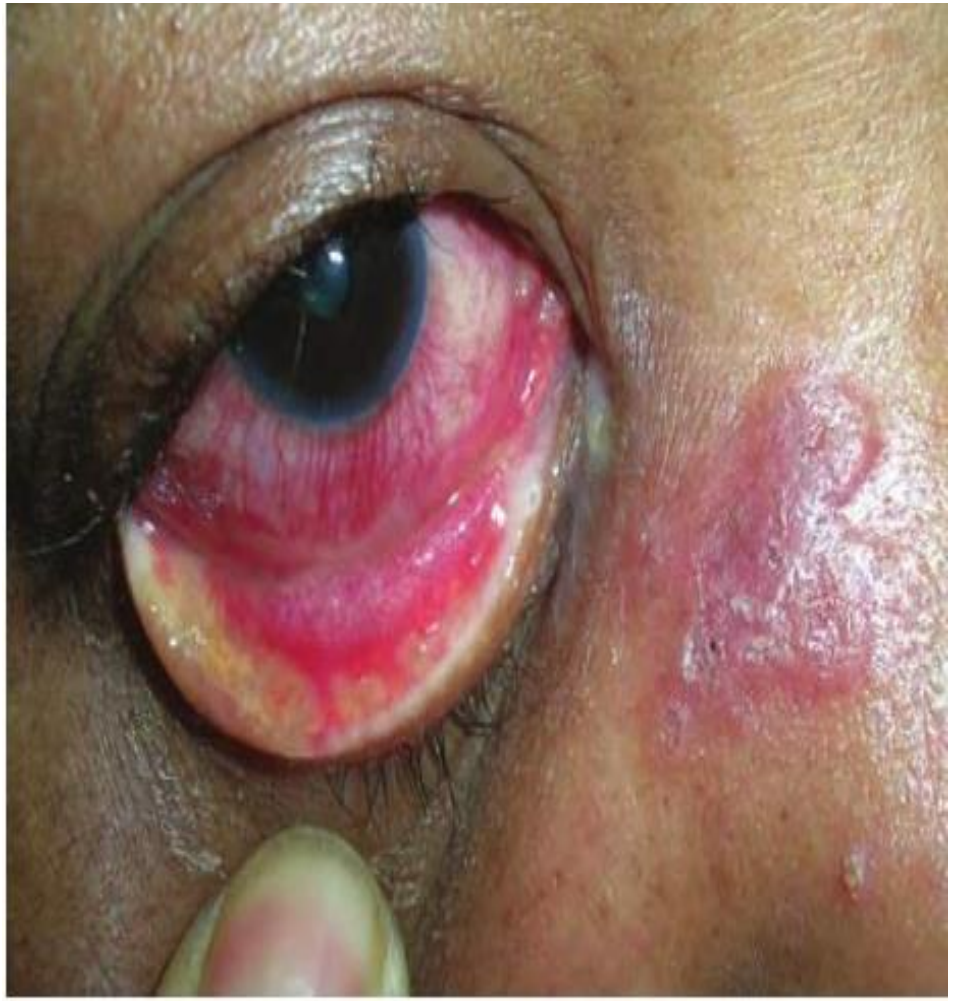
F: sarcoid on a heart-shaped homemade tatto over the knee



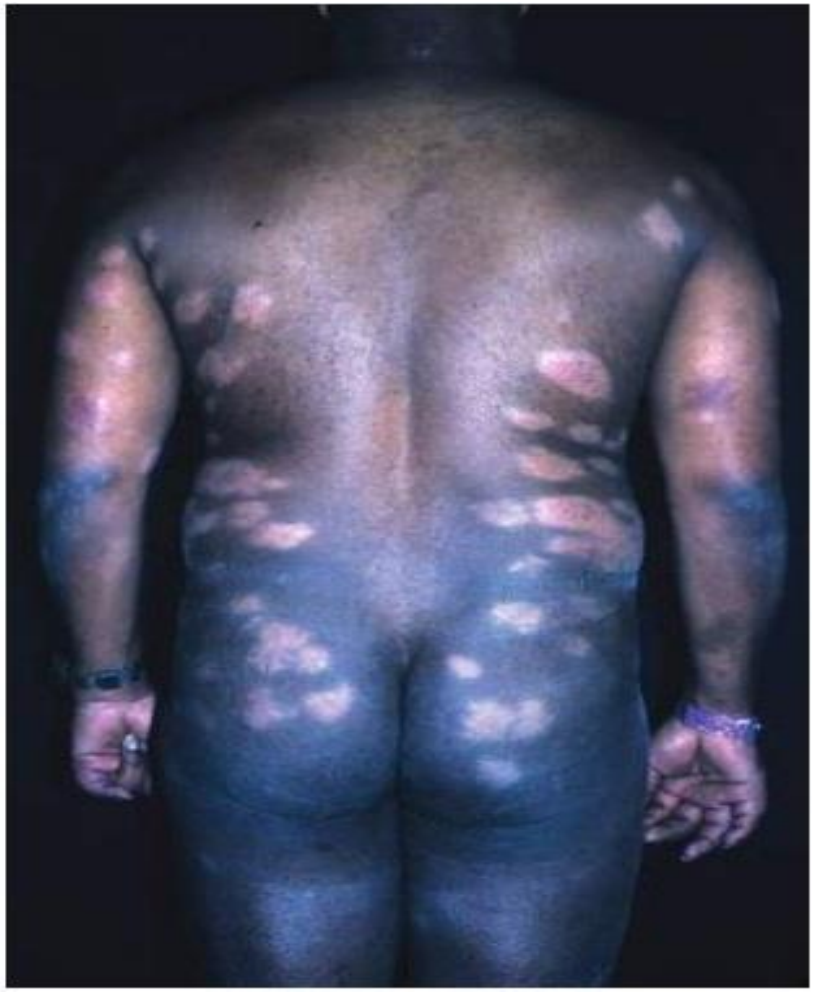
F: subcutaneous sarcoid (darier-Roussy syndrome) in advanced sarcoidosis



Dx: sarcoidosis
F: involvement of the conjuctiva and inner lower eyelid.

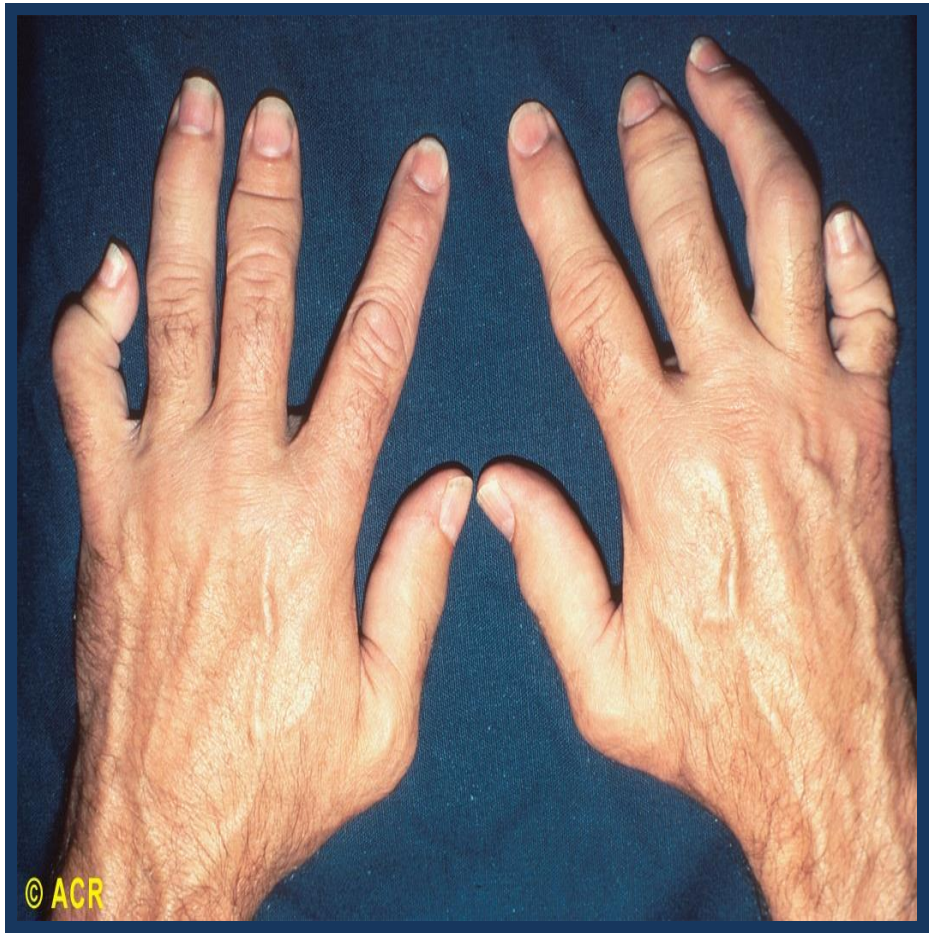


DX: sarcoidosis
F: Hypopigmented cutaneous plaques.



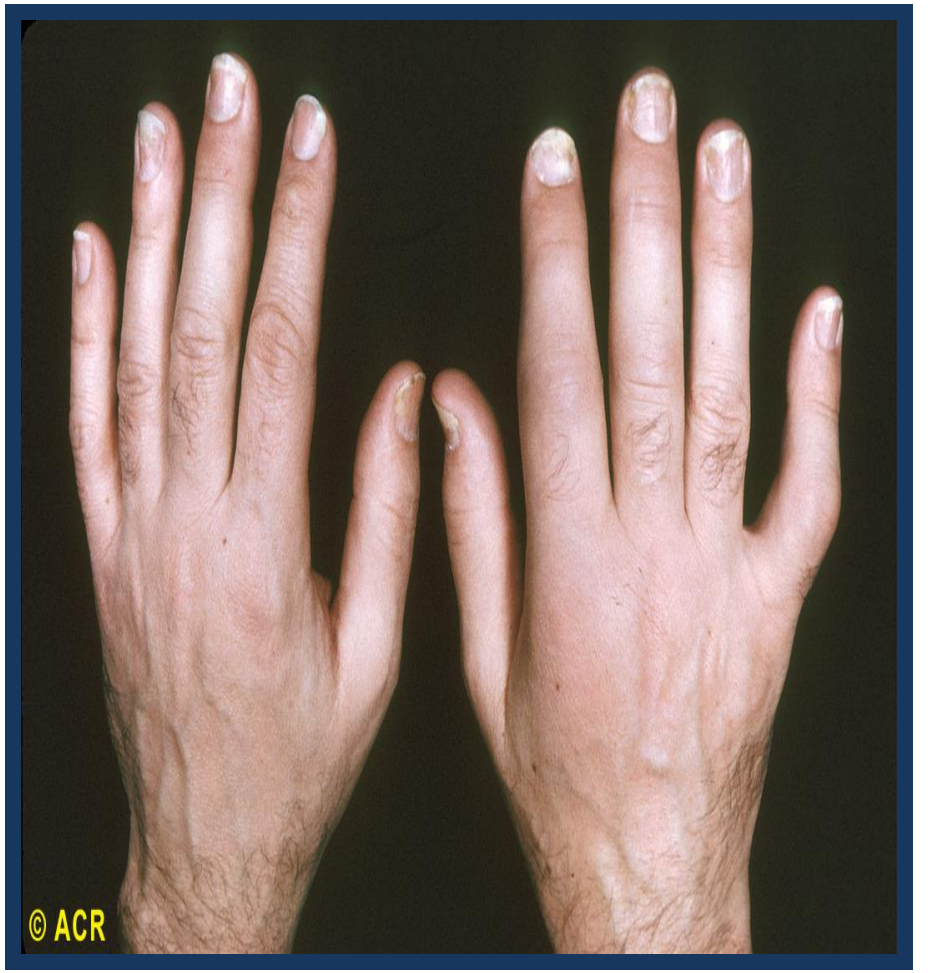
Arthritis Mutilan's

- Osteolysis of the phalanges and Metacarpal's 5%.
- Often associated with sacorilities.



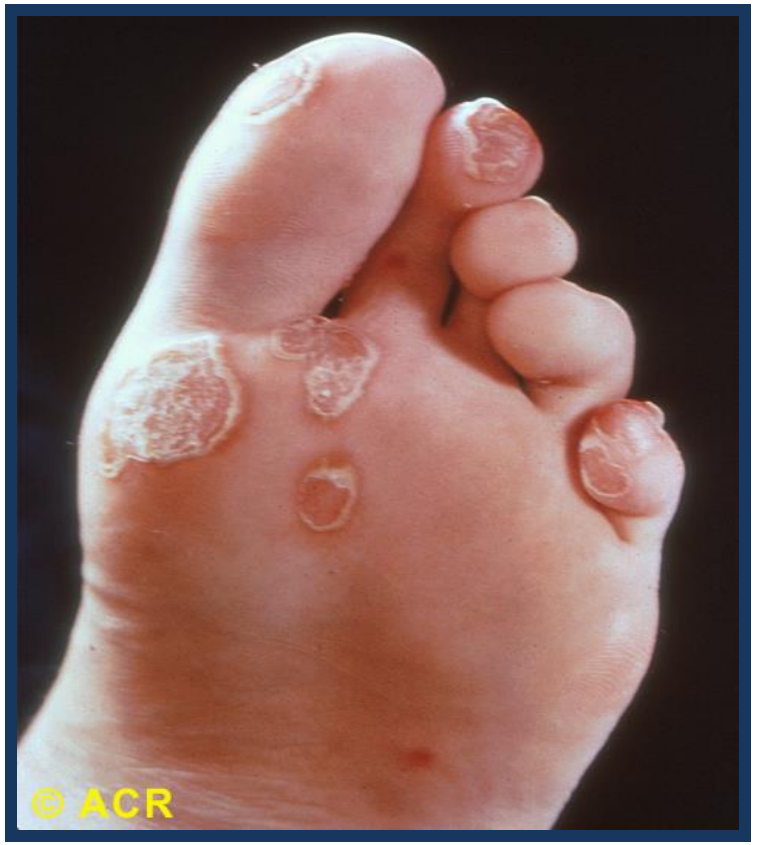
Symmetric polyarthritis

- Dip
- Tendency for bony ankylosing
- R A

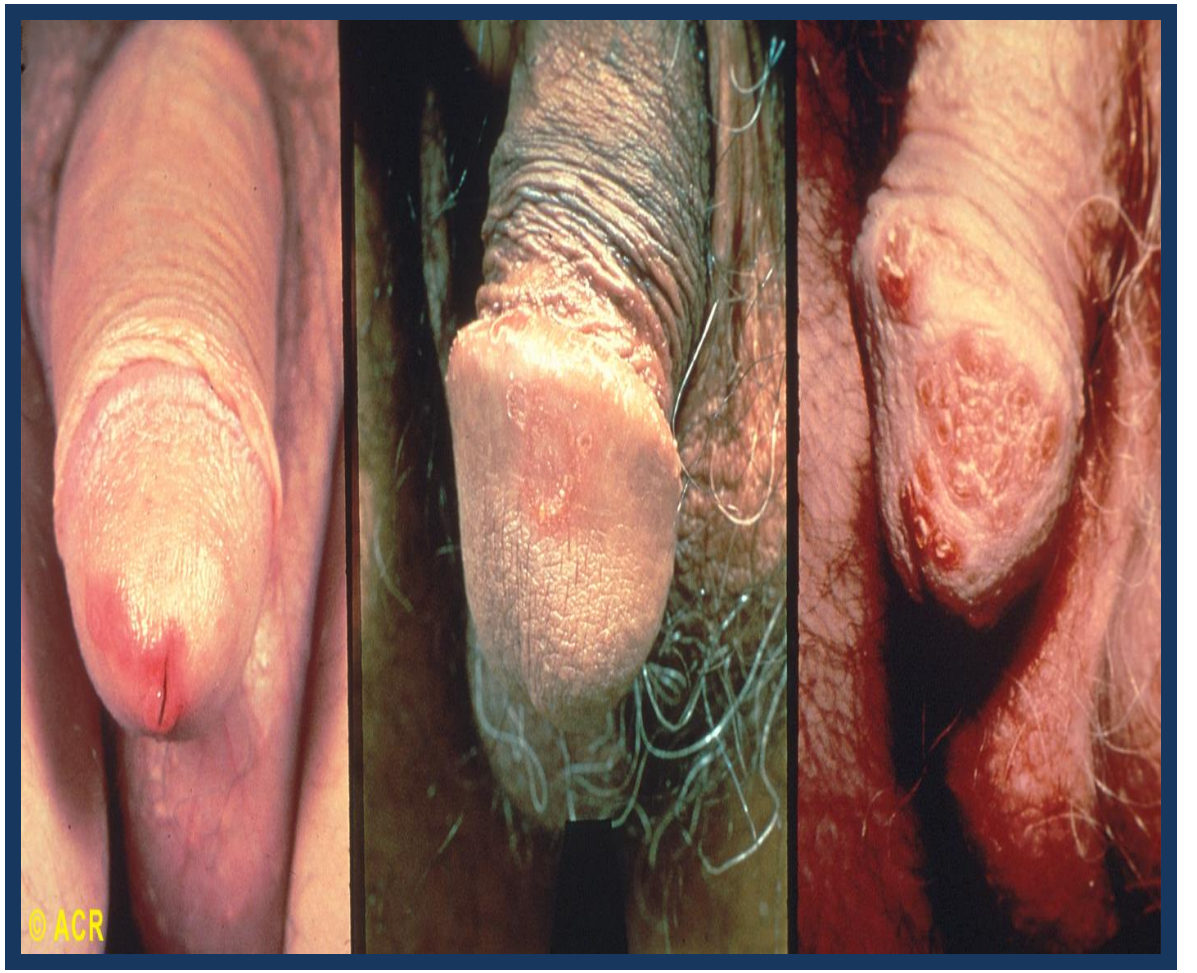


Reactive arthritis: keratoderma blennorrhagica, foot

Discrete, circinate, scaly, and plaque-like lesions on the foot in reactive arthritis resemble secondary syphilis and psoriasis. Note the two small lesions are in an early phase of keratoderma.

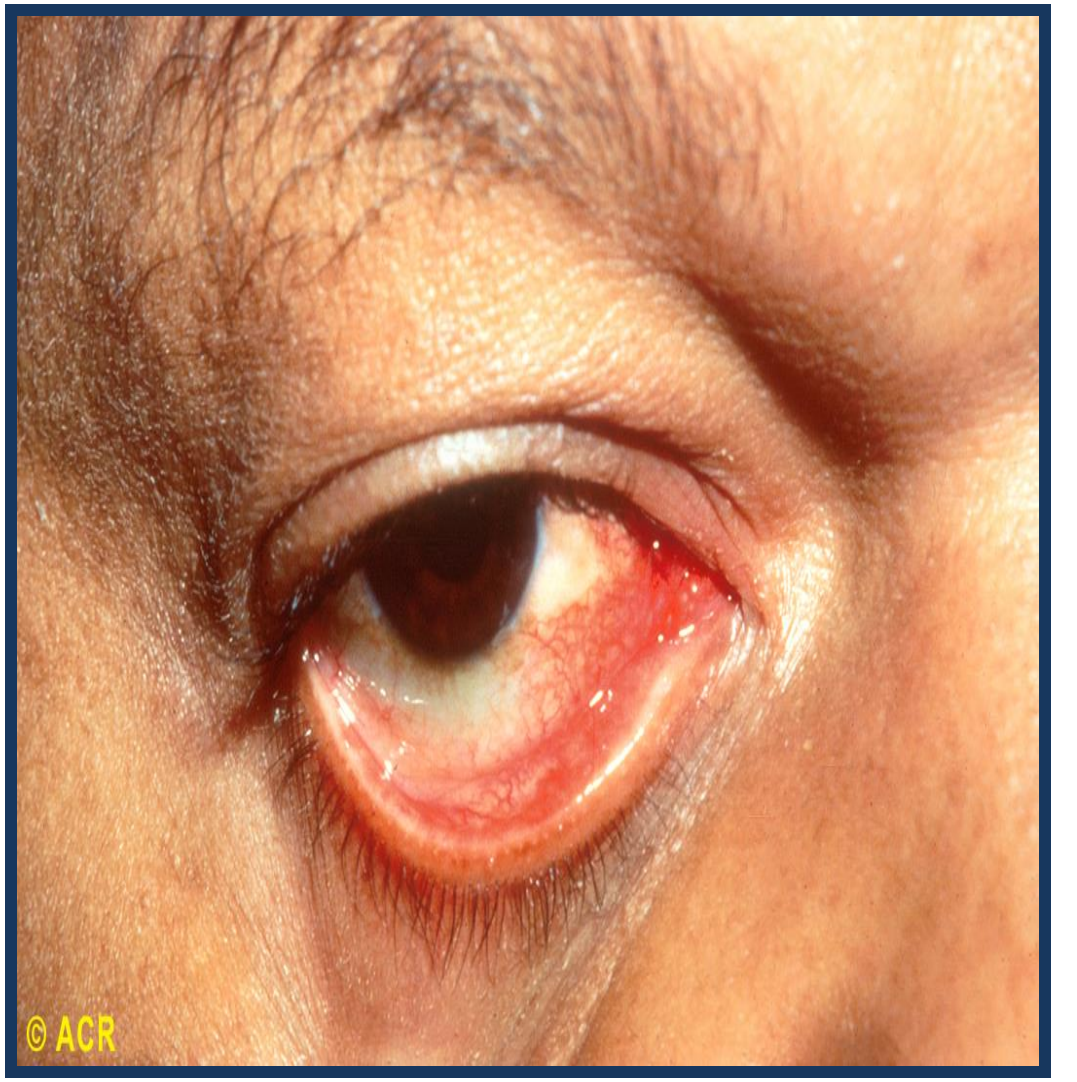


Reactive arthritis: balanitis circinata



Reactive arthritis: conjunctivitis

The erythema and exudate on the bulbar and palpebral conjunctivae are characteristic of the acute transient conjunctivitis of reactive arthritis. The reaction is often mild and easily overlooked, but photophobia, excessive lacrimation, burning, and intense hyperemia may occur. Iritis and episcleritis usually do not involve the palpebral conjunctivae.



14.CASES

Q: A 24 year old female recently married has had 3 abortions. She also complains of general fatigue, polyarticular arthritis, and has had recurrent oral ulcerations……
CBC shows an anemic patient with Anemia of
Chronic Disease(Normocytic)
Lots of other labs that are useful to exclude differentials if you're seriously thinking about them.

Q1. What is your diagnosis
Systemic Lupus Erythematosus
Q2. Two tests you would carry out to confirm
A. ANA B. Anticardiolipin antibodies (Alternatives include anti-dsDNA/anti-SM/lupus anticoagulant)
Q3. Two physical signs you would see in the patient
A. Malar Rash B. Discoid Rash (Alternatives include: Photosensitivity or any other sign in a lupus patient)

Q:A pt with hypertension (or DM) presented with right ankle swelling & pain. He had 2 previous similar conditions; one was in the same site, the other was on the left ankle. His CBC showed leukocytosis
(WBC count = 10,000).

1- What is the most probable Dx?

Gout.

2- Mention another DDx.

Septic arthritis, Cellulitis, Pseudogout.

3- If a sample from the synovial fluid was aspirated, what is your confirmatory test?

Identification of monosodium urate crystals under polarized light microscopy; they have a needle-lik morphology & strong negative birefringence.

4- Mention 2 drugs for the treatment of the acuteattack.

Steroids, NSAIDs, Colchicine.

15.Summary slides
- Dr. Walid Wadi

Monoarthritis

Differential Diagnosis of Acute Monoarthritis

Infection
- Bacterial
- Fungal
- Mycobacterial
- Viral
- Spirochete

Crystal-induced
- Monosodium urate
- Calcium pyrophosphate dihydrate
- Hydroxyapatite
- Calcium oxalate
- Lipid

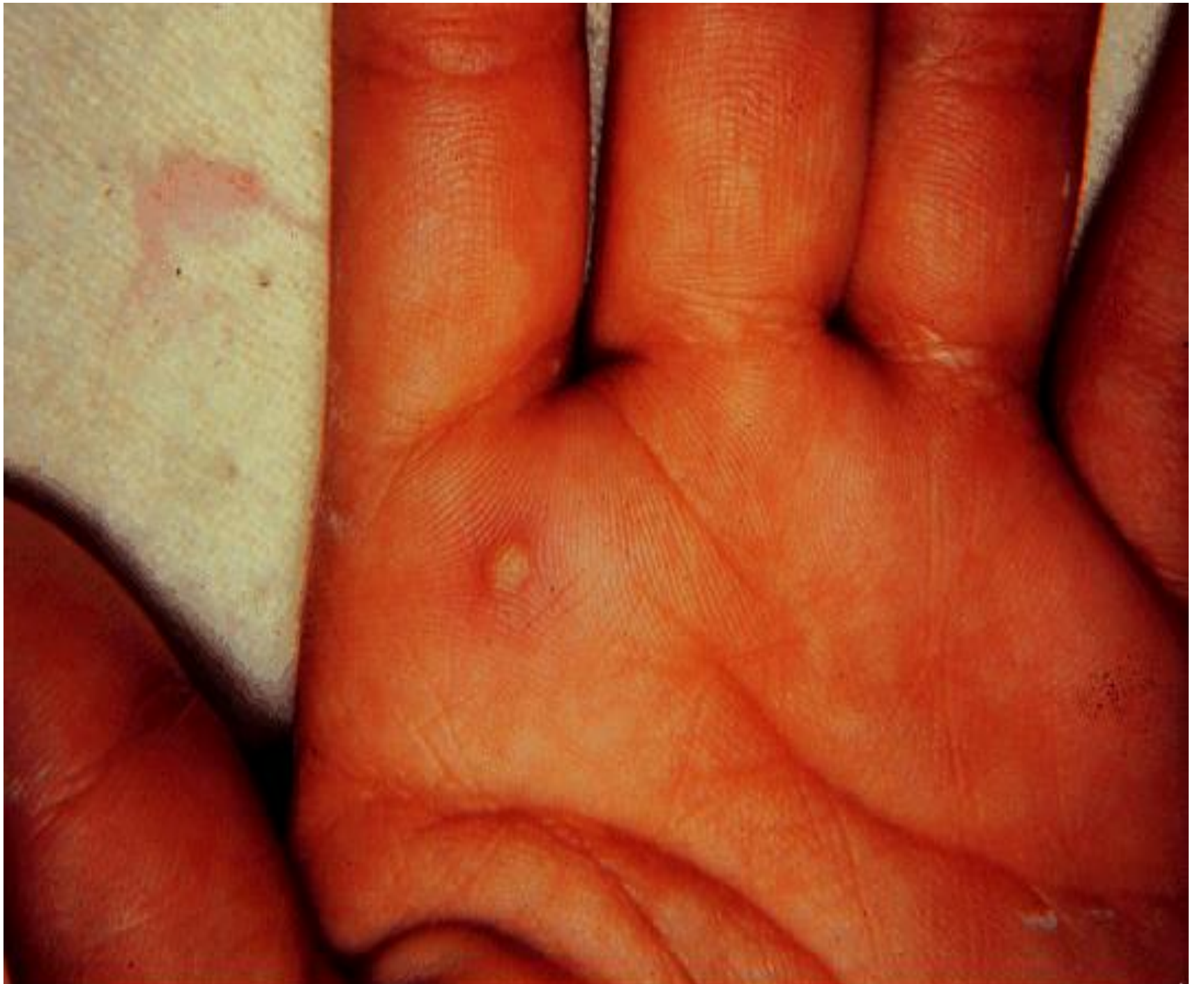
Hemarthrosis
- Trauma
- Anticoagulation
- Clotting disorders
- Fracture
- Pigmented villonodular synovitis

Tumor
- Pigmented villonodular synovitis
- Chondrosarcoma
- Osteoid osteoma
- Metastatic disease

Systemic rheumatic disease
- Rheumatoid arthritis
- Spondyloarthropathy
- Systemic lupus erythematosus
- Sarcoidosis

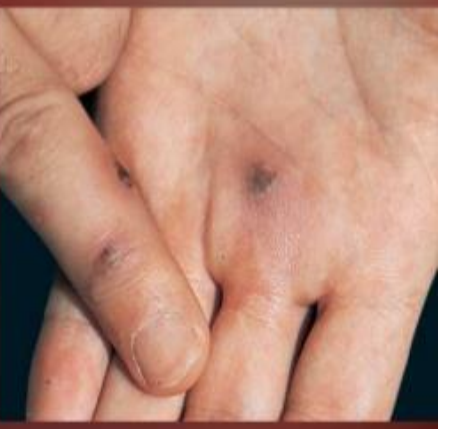
Osteoarthritis
- Erosive variant

Intraarticular derangement
- Meniscal tear
- Osteonecrosis
- Fracture



Disseminated Gonococcal Infection

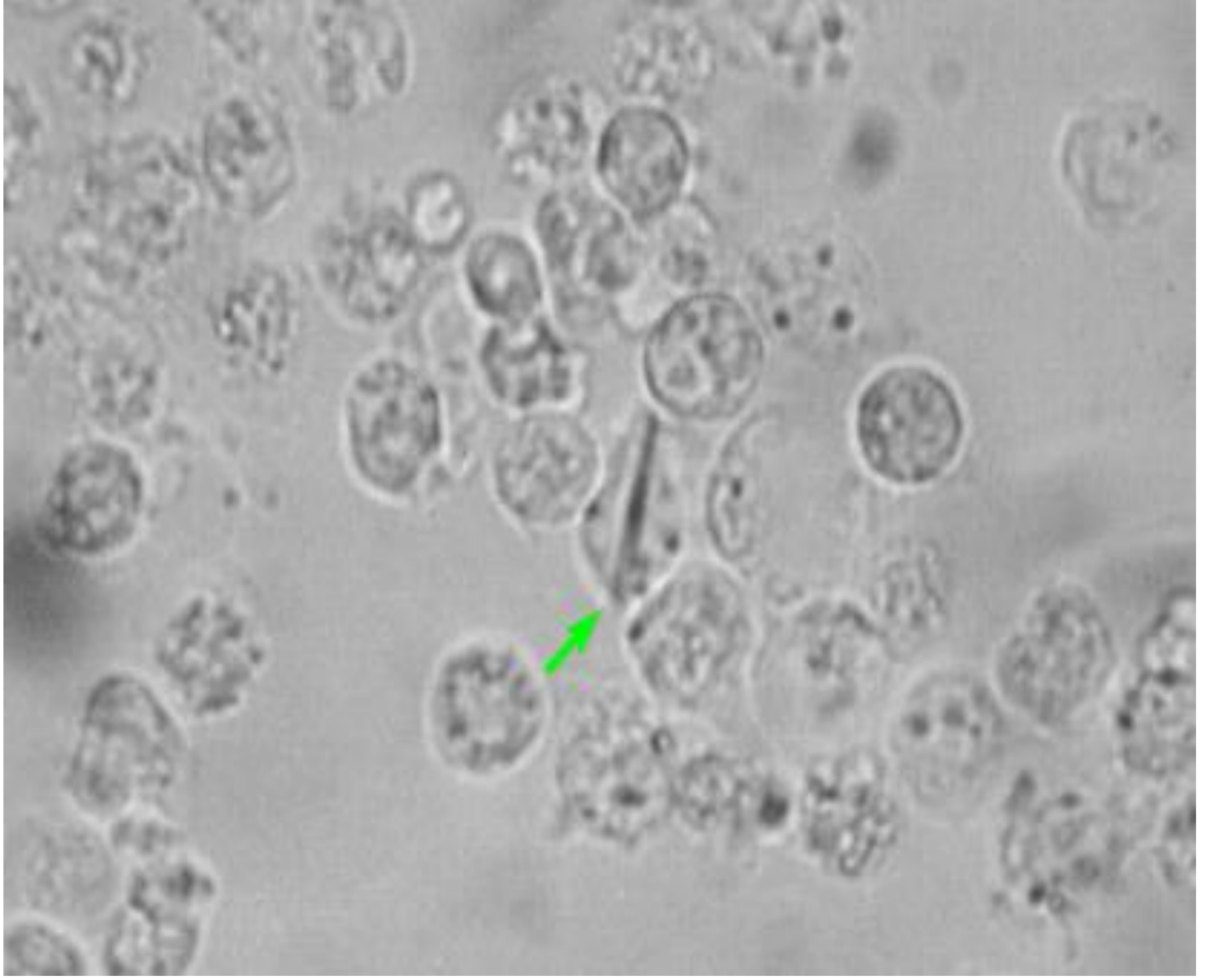
- Rash
  - Hemorrhagic pustules on erythematous base
- Bactermemia
  - Meningitis, endocarditits
- Oligoarticular arthritis
  - Knees most common
- Tenosynovits
- ADMIT IV ABX
  - And treat partner



Categories of Synovial Fluid Based Upon Clinical and Laboratory Findings

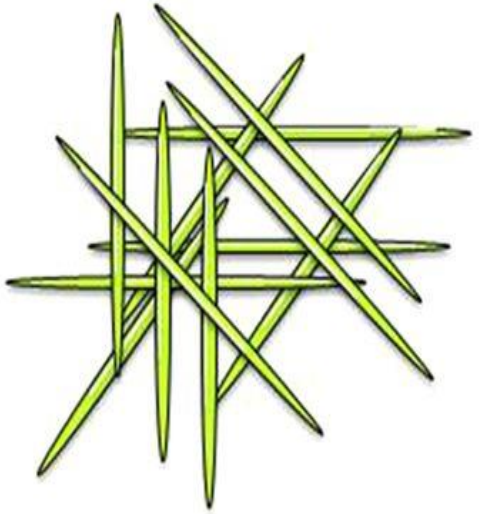
Measure	Normal	Noninflammatory	Inflammatory	Septic	Hemorrhagic
Volume, mL (knee)	<3.5	Often >3.5	Often >3.5	Often >3.5	Usually >3.5
Clarity	Transparent	Transparent	Translucent-opaque	Opaque	Bloody
Color	Clear	Yellow	Yellow to opalescent	Yellow to green	Red
Viscosity	High	High	Low	Variable	Variable
WBC, per mm3	<200	200-2,000	2,000-10,000	>100,000†	200-2,000
PMNs, percent	<25	<25	≥50	≥75	50 - 75
Culture	Negative	Negative	Negative	Often positive	Negative
Total protein, g/dL	1-2	1-3	3-5	3-5	4-6
LDH (compared to levels in blood)	Very low	Very low	High	Variable	Similar
Glucose, mg/dL	Nearly equal to blood	Nearly equal to blood	>25, lower than blood	<25, much lower than blood	Nearly equal to blood

†Lower with infections caused by partially treated or low virulence organisms



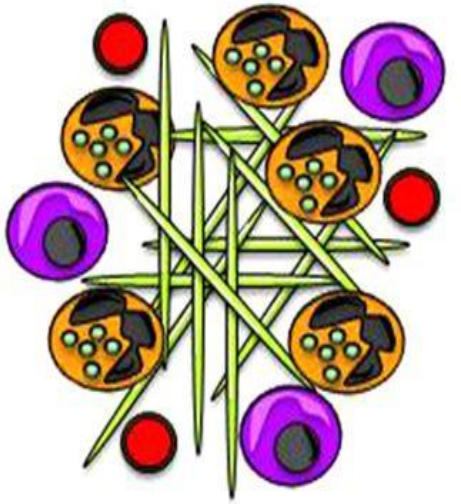
1. Formation of MSU Crystals

- Hyperuricaemia
- **Precipitation** of MSU crystals
- **Deposition** in articular and periarticuar tissue



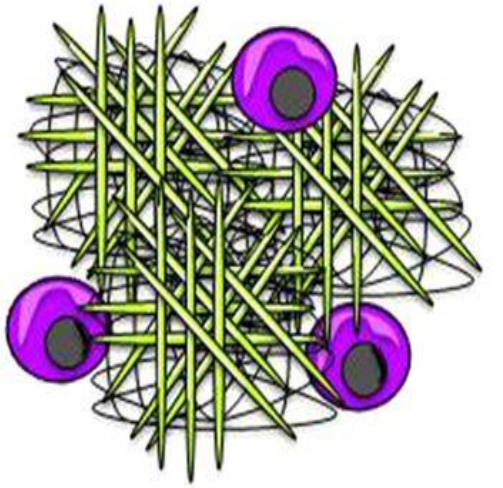
2. Acute Gout Attack

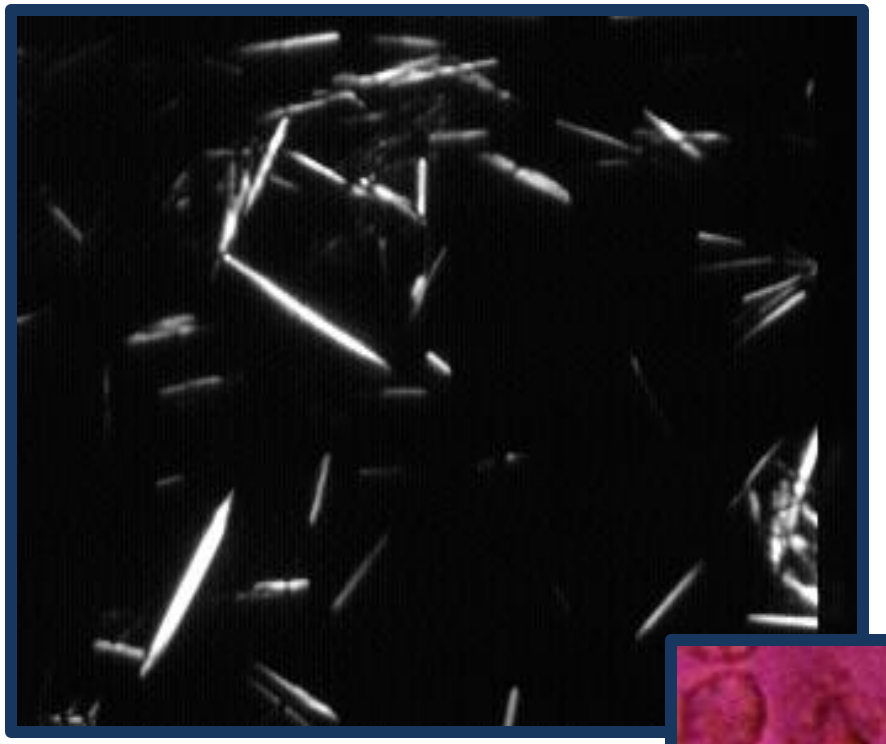
- **Phagocytosis** of Crystals
- Cell Swelling and **Inflammasome** Activation
- **Cytokine p**roduction and vasodilatation
- Neutrophil and monocyte influx

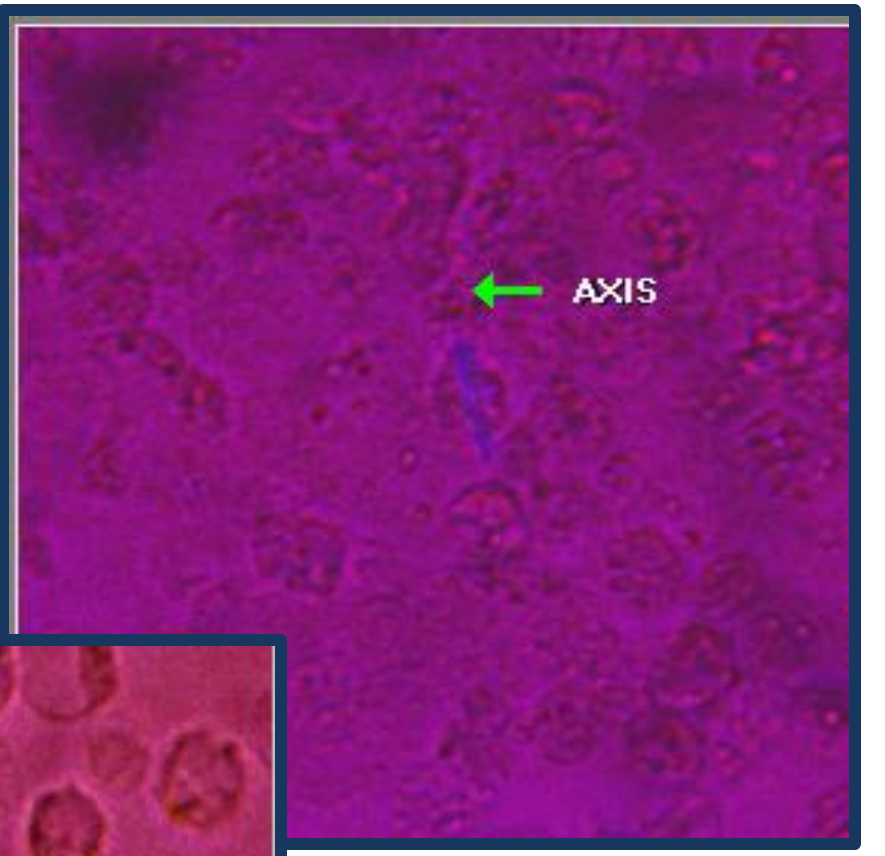


3. Chronic Tophaceous Gout

- Neutrophil death by **NETosis**
- Packaging of MSU crystals
- Inactivation of inflammatory cytokines
- **Resolution** of Inflammation



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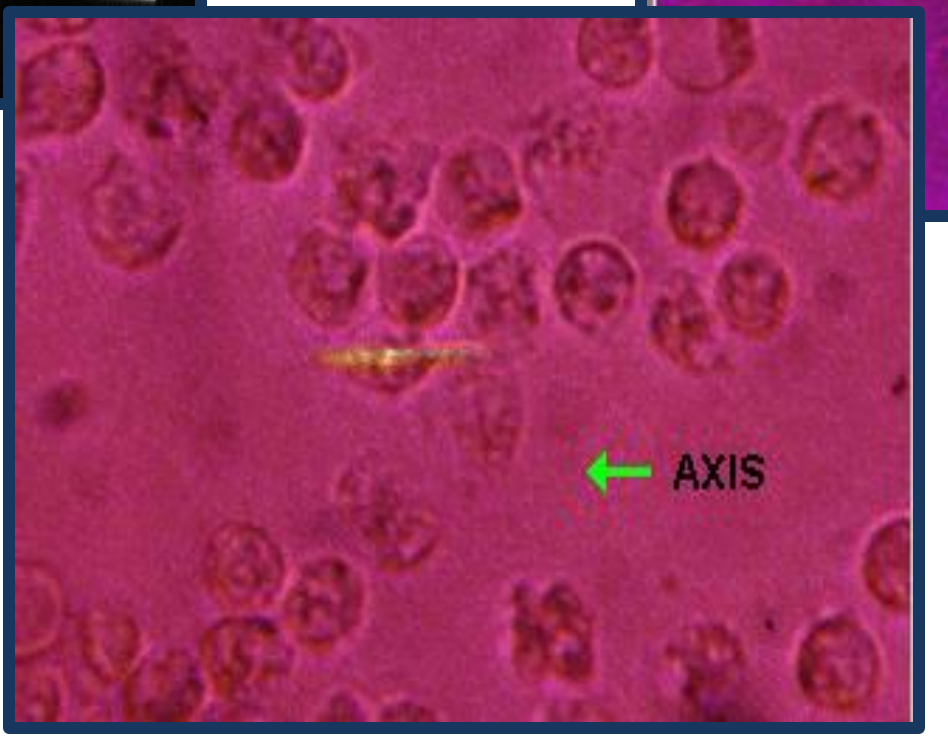
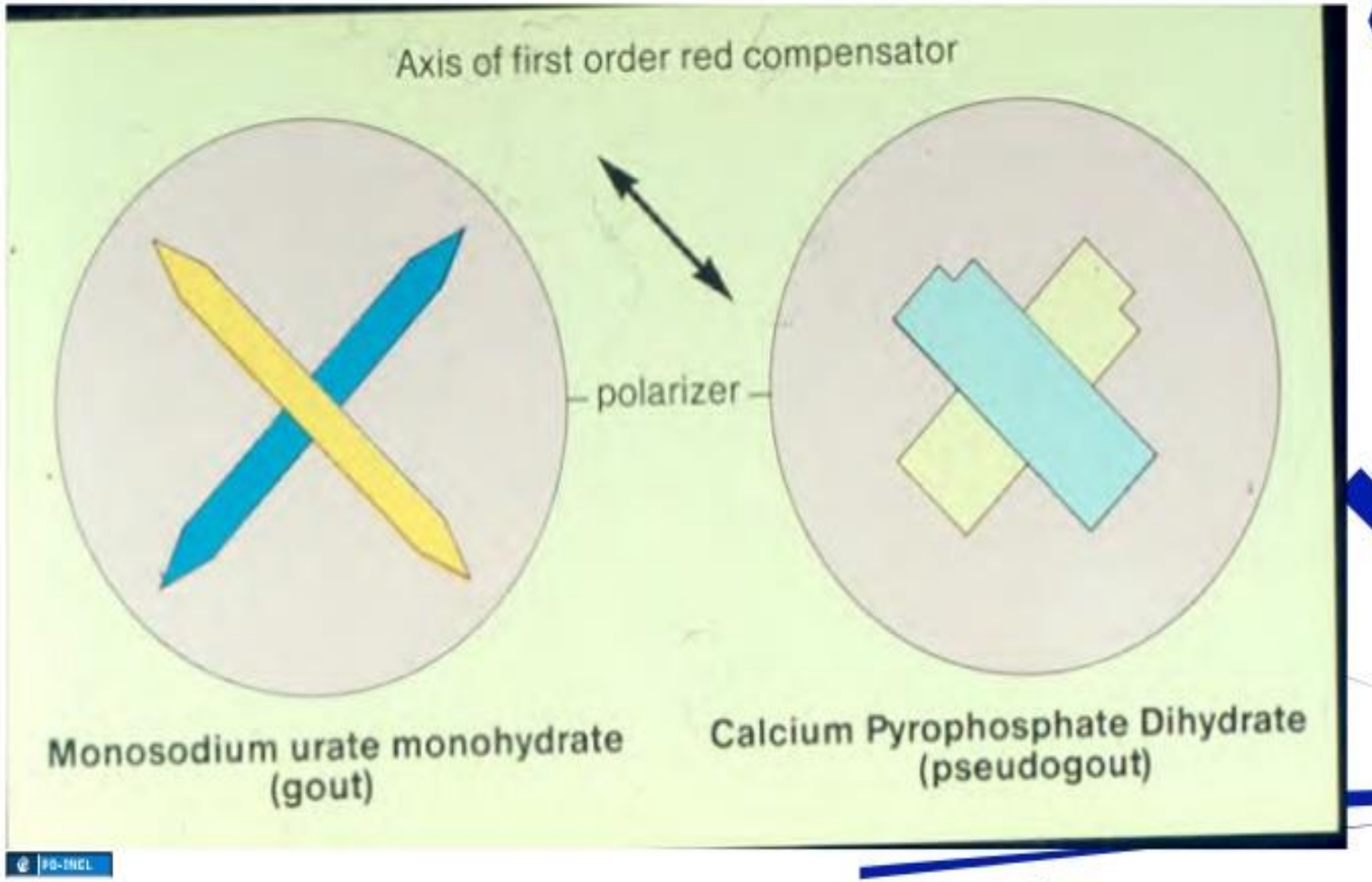
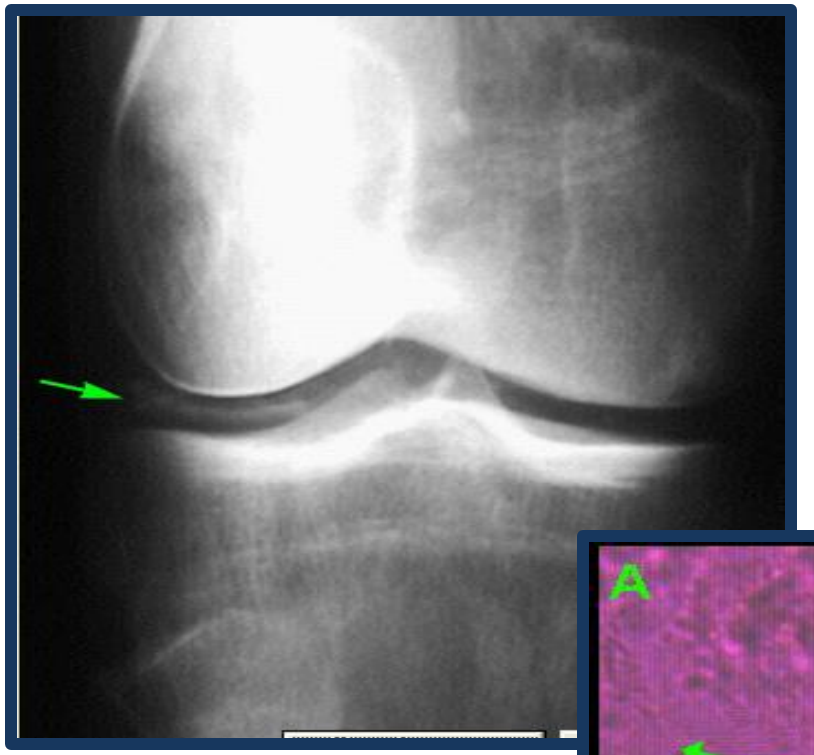

AXIS

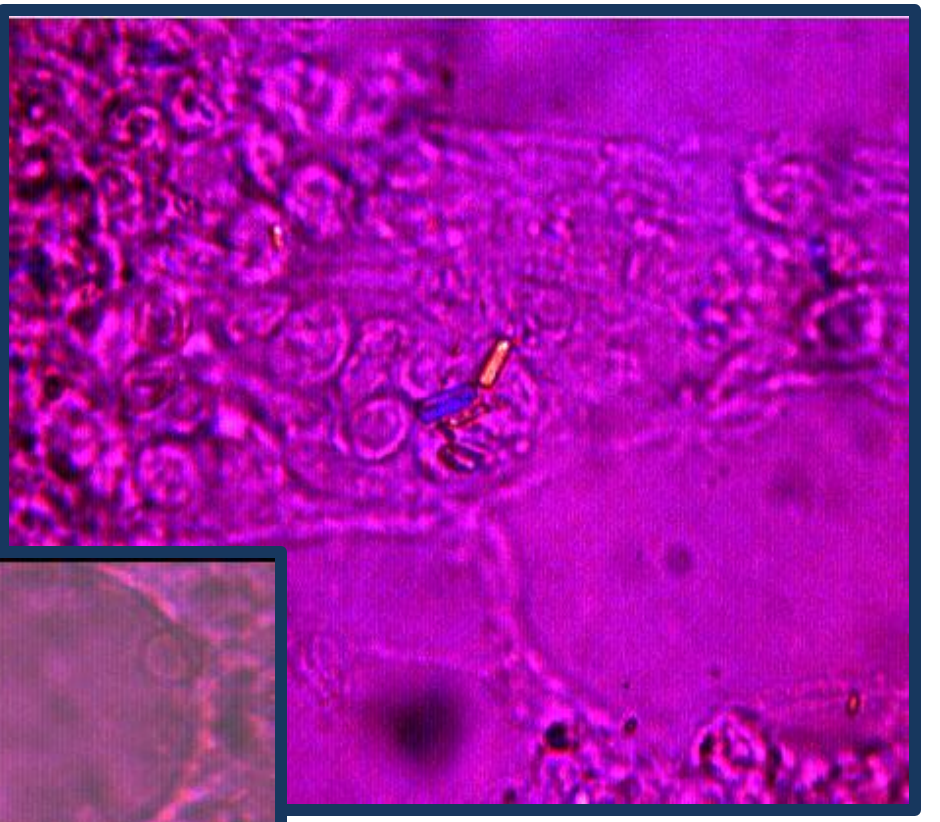
Polarizing Microscopy

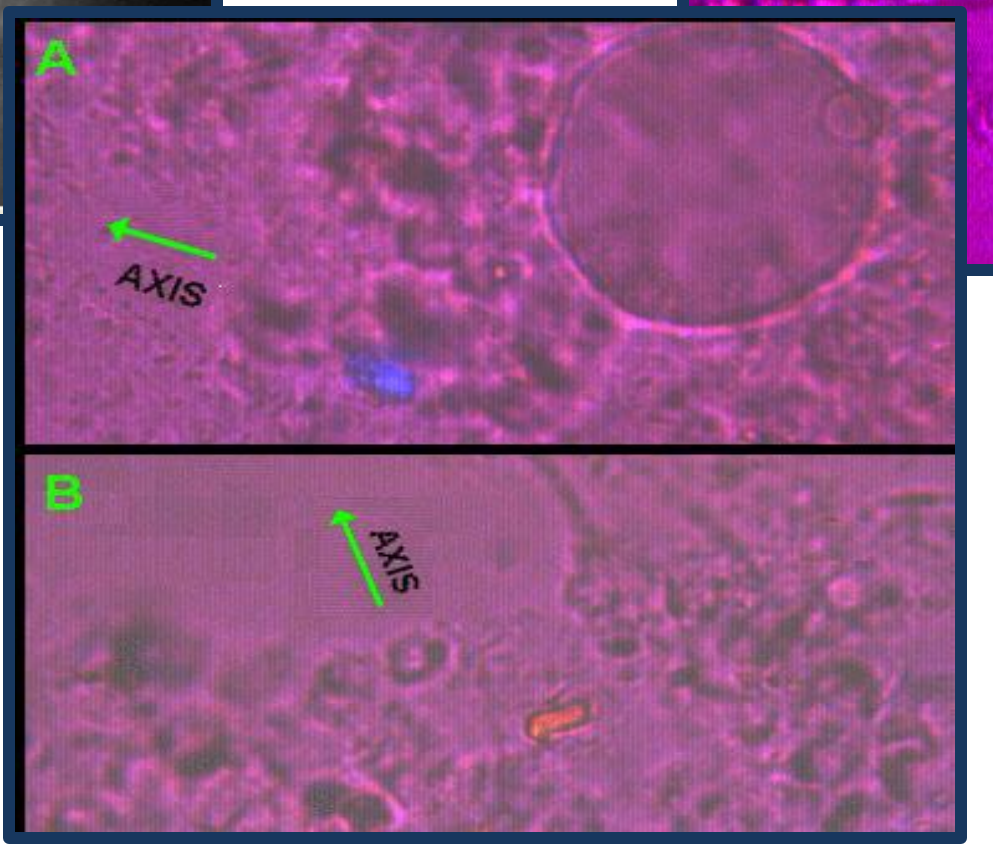


CPPD

- Calcium pyrophosphate deposition disease
- Can be confused with septic arthritis.
- Modes of presentation:
    - -A symptomatic: most common
    - -Pseudogout.
    - -Pseudorheumatoid.
    - -Pseudoosteoarthritis
    - -Pseudoneuropathic joint



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AXIS

Rheumatoid arthritis

American Rheumatism Association Revised Criteria For Rheumatoid Arthritis Classification

Criterion	Description
Morning stiffness	Morning stiffness in and around the joints, lasting at least one hour before maximal improvement.
Arthritis of 3 or more joint areas	At least 3 joint areas (out of 14 possible areas; right or left PIP, MCP, wrist, elbow, knee, ankle, MTP joints) simultaneously have had soft-tissue swelling or fluid (not bony overgrowth alone) as observed by a physician.
Arthritis of hand joints	At least one area swollen (as defined above) in a wrist, MCP, or PIP joint.
Symmetric arthritis	Simultaneous involvement of the same joint areas (as defined above) on both sides of the body (bilateral involvement of PIPs, MCPs, or MTPs, without absolute symmetry is acceptable).
Rheumatoid nodules	Subcutaneous nodules over bony prominences or extensor surfaces, or in juxta-articular regions as observed by a physician.
Serum rheumatoid factor	Demonstration of abnormal amounts of serum rheumatoid factor by any method for which the result has been positive in less than 5% of normal control subjects.
Radiographic changes	Radiographic changes typical of rheumatoid arthritis on posteroanterior hand or wrist radiographs, which must include erosions or unequivocal bony decalcification localised in, or most marked adjacent to, the involved joints (osteoarthritis changes alone do not qualify).

Note: For classification purposes, a patient has RA if at least four of these criteria are satisfied (the first four must have been present for at least six weeks).

2010 criteria of RA

Joint involvement (0-5)

1 med / large joint	0
2-10 med / large joints	1
1-3 small joints	2
4-10 small joints	3
>10 joints (at least 1 small)	5

Serology (0-3)

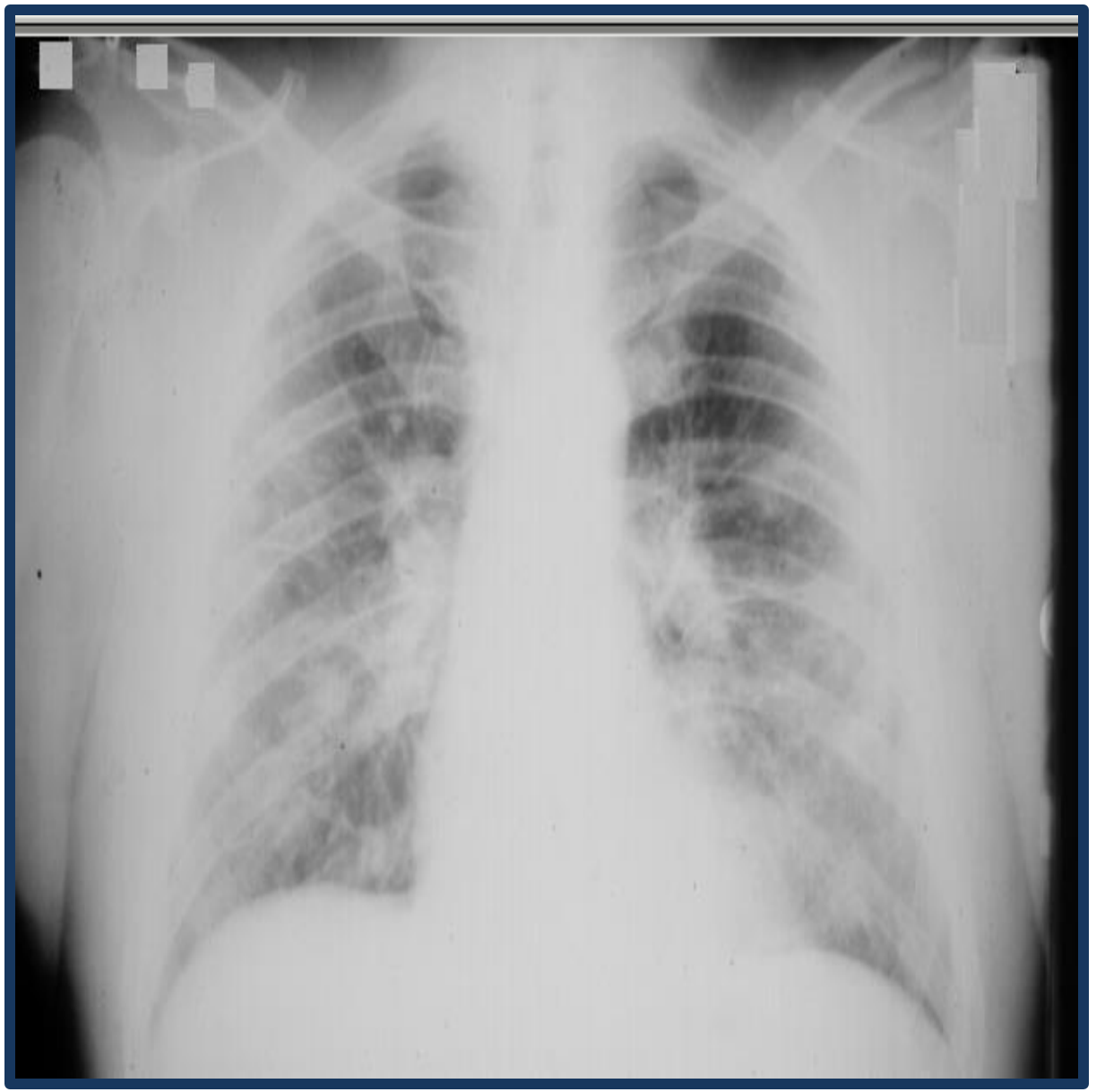
Neither Rf nor ACPA positive	0
At least one test low postive	2
At least one test high postive	3

Duration of synovitis (0-1)

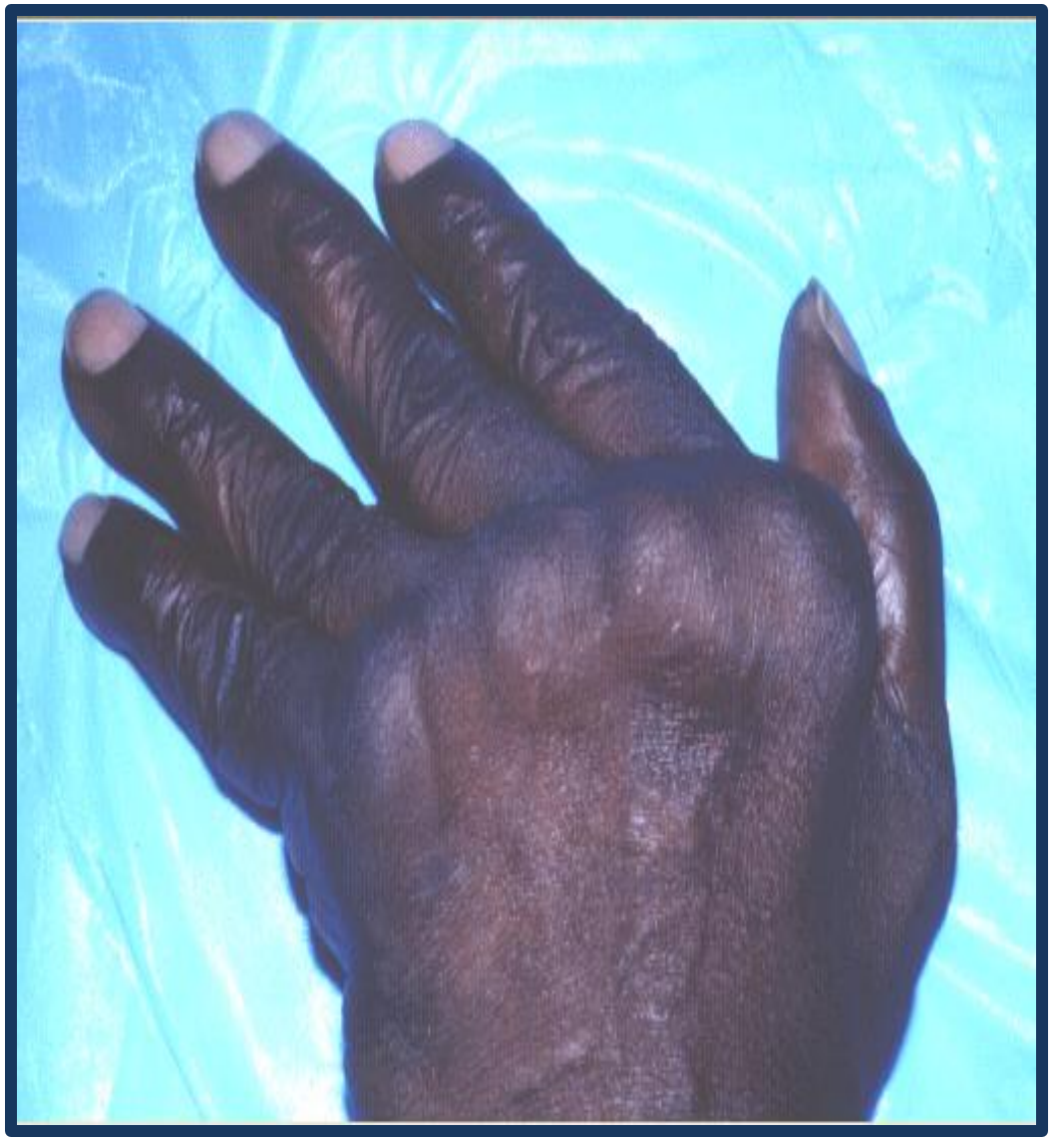
<6 weeks	0
>6 weeks	1

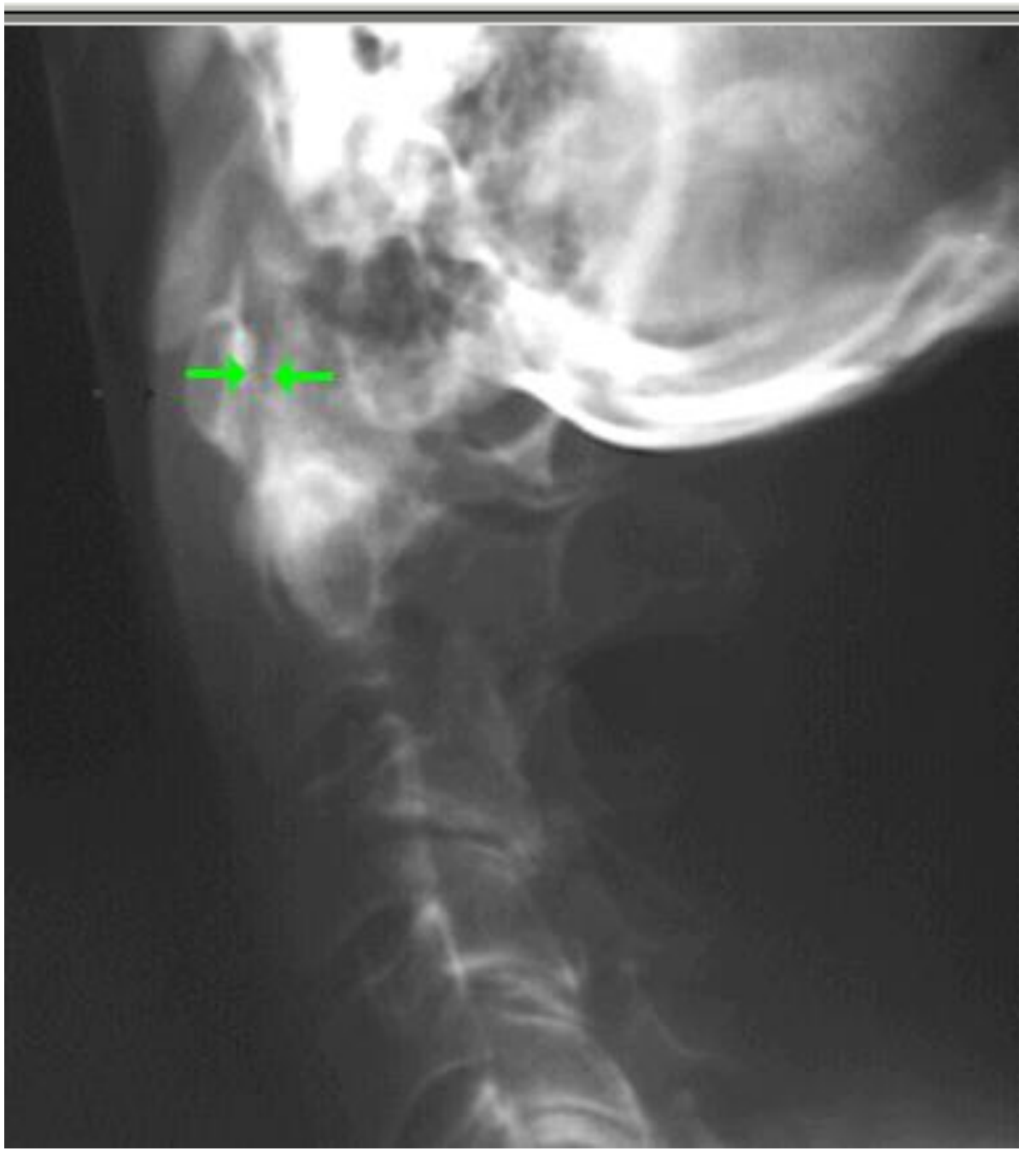
Accute phase reactants (0-1)

Neither CRP nor ESR abnormal	0
Abnormal CRP or abnormal ESR	1

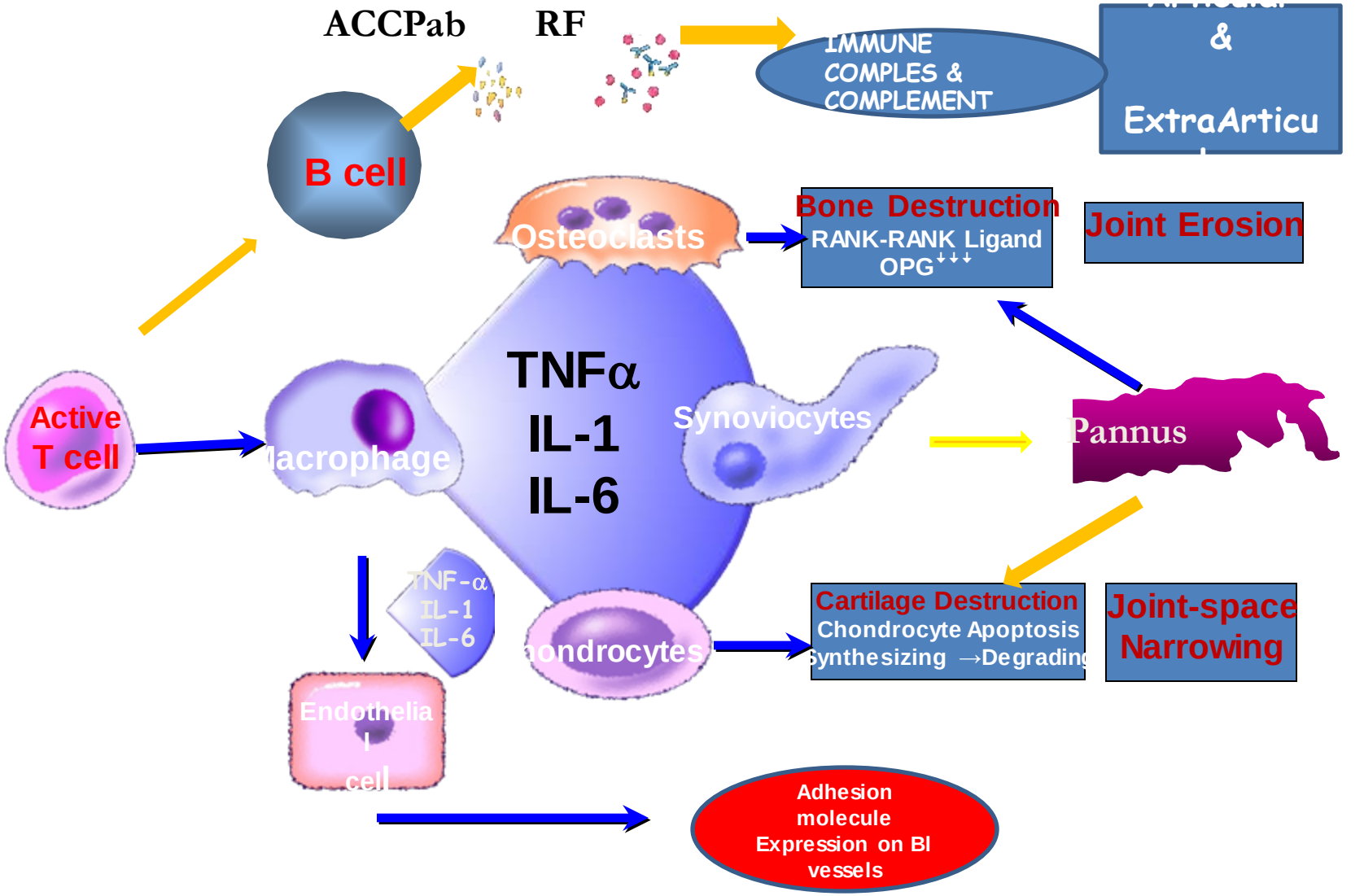


Rheumatoid nodules



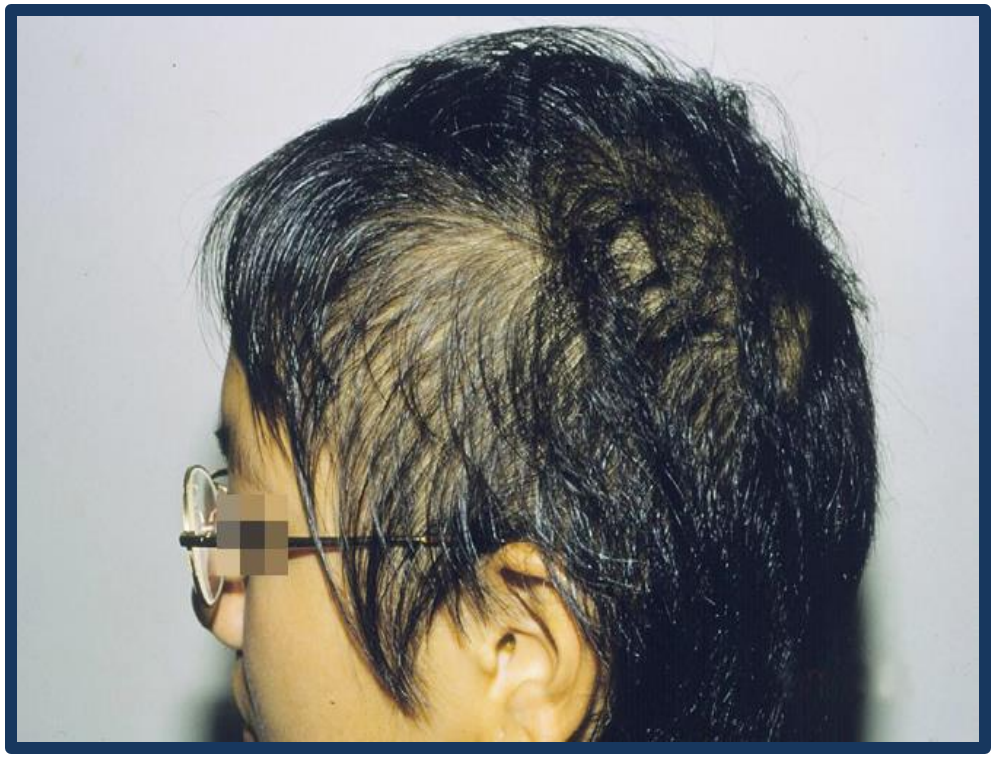


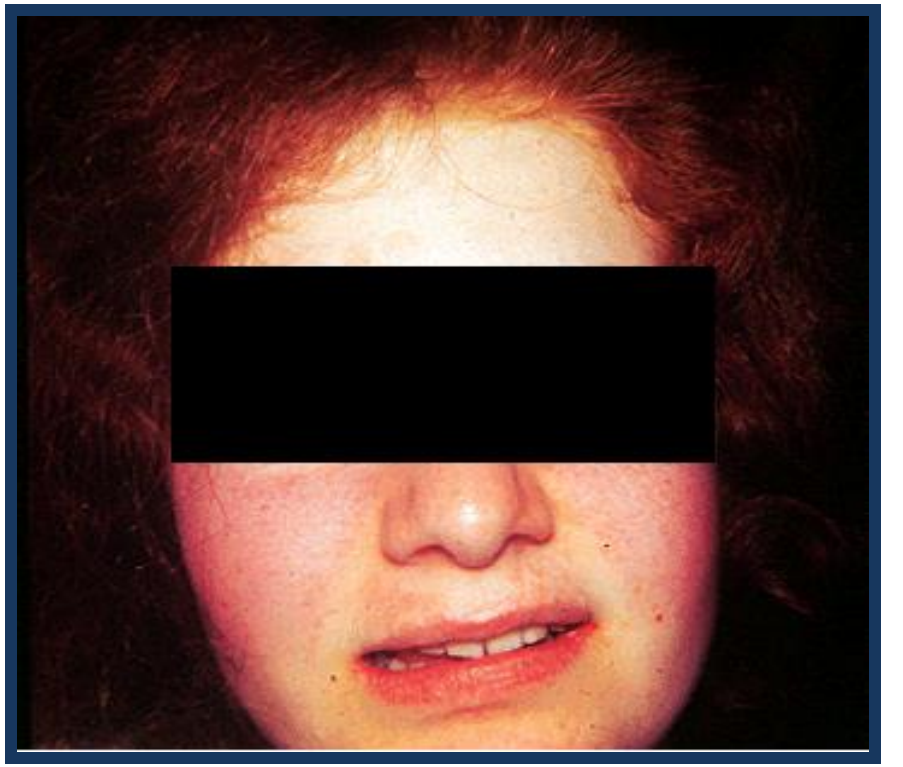
RA Pathogenesis & Structural Damage

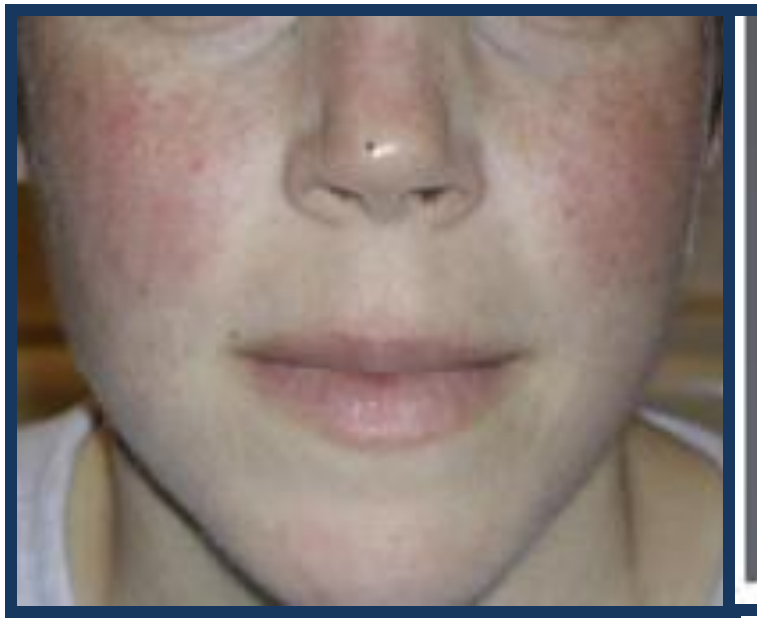


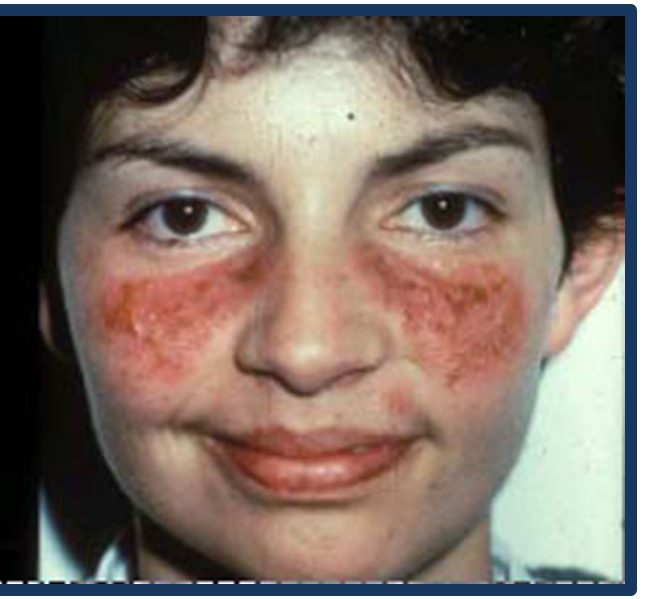
Adapted from Arend WP. *J Rheumatol* Suppl. 2002;65:16-21. Permission to reproduce granted by *Journal of Rheumatology* and Dr WP Arend.

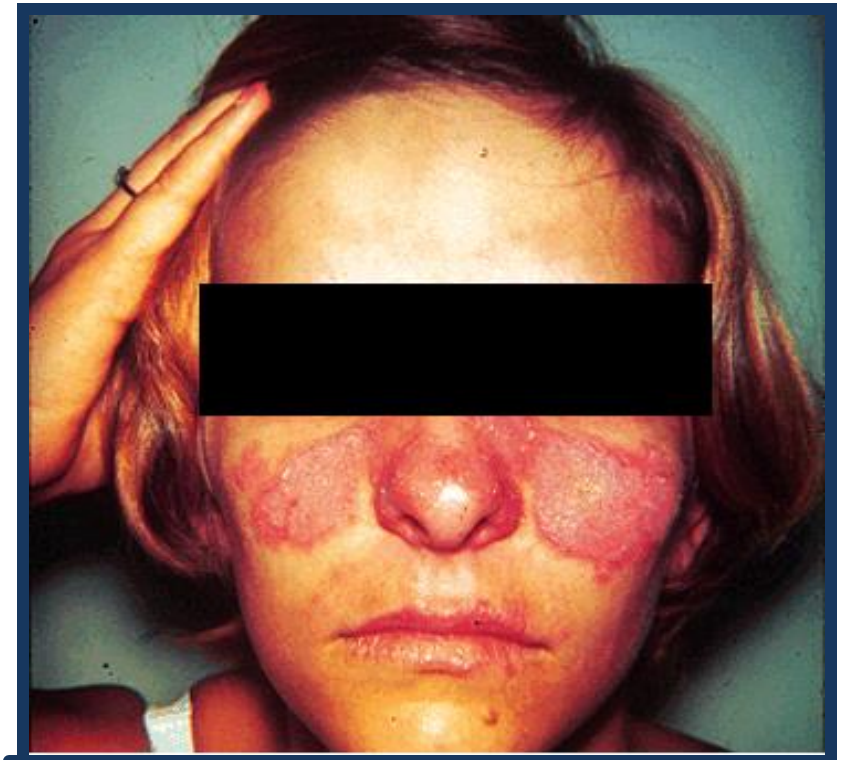
Systemic lupus erythematosus

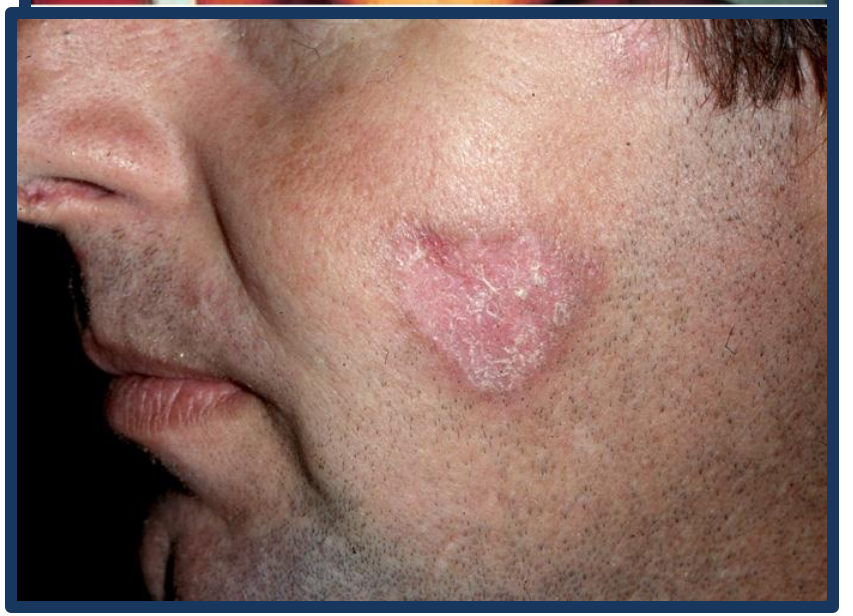


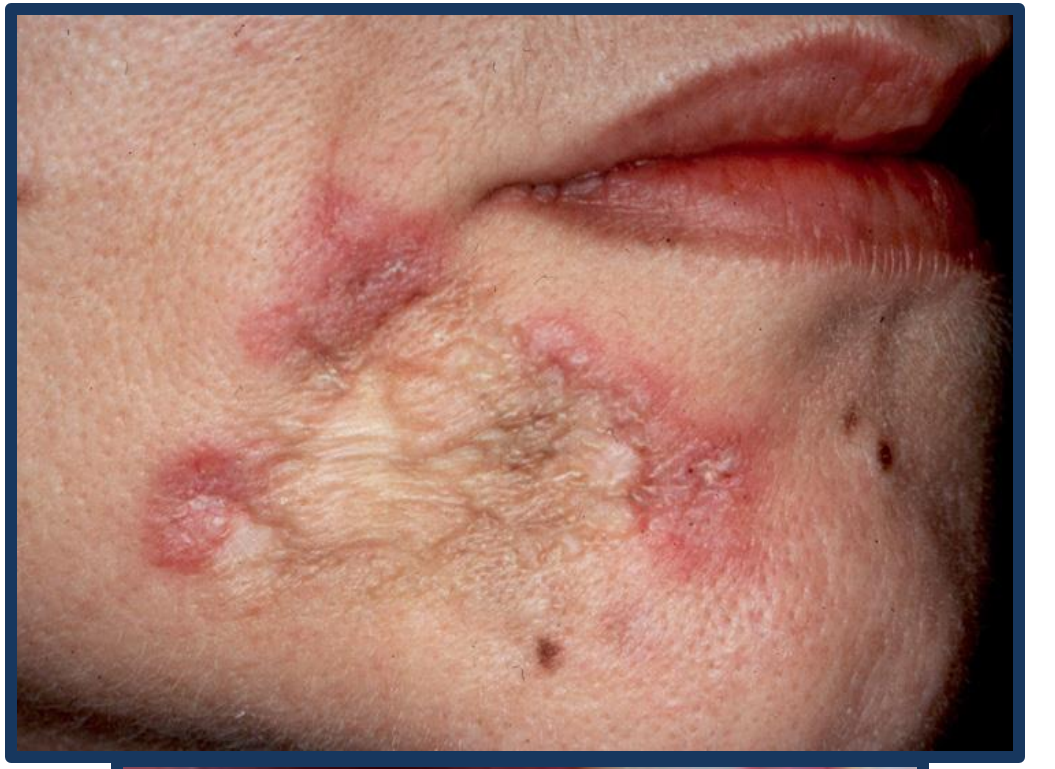


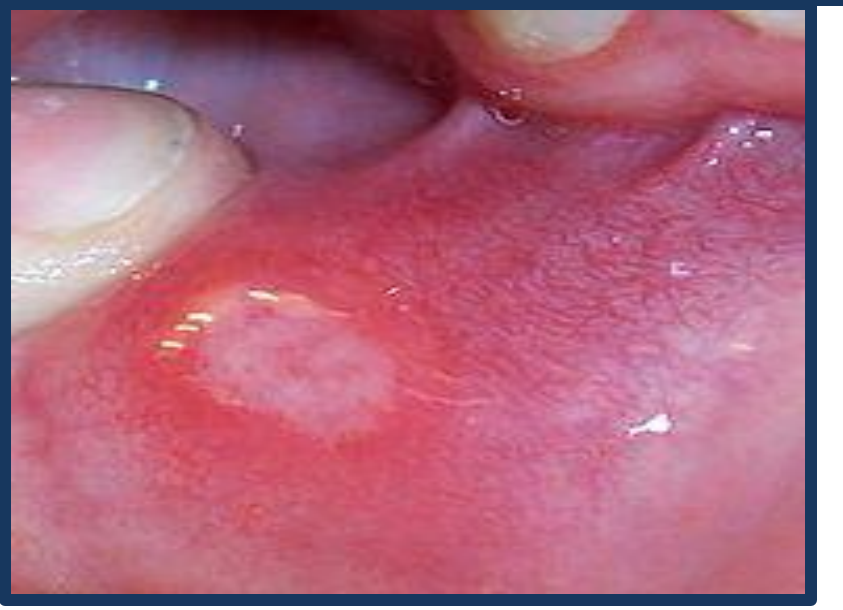


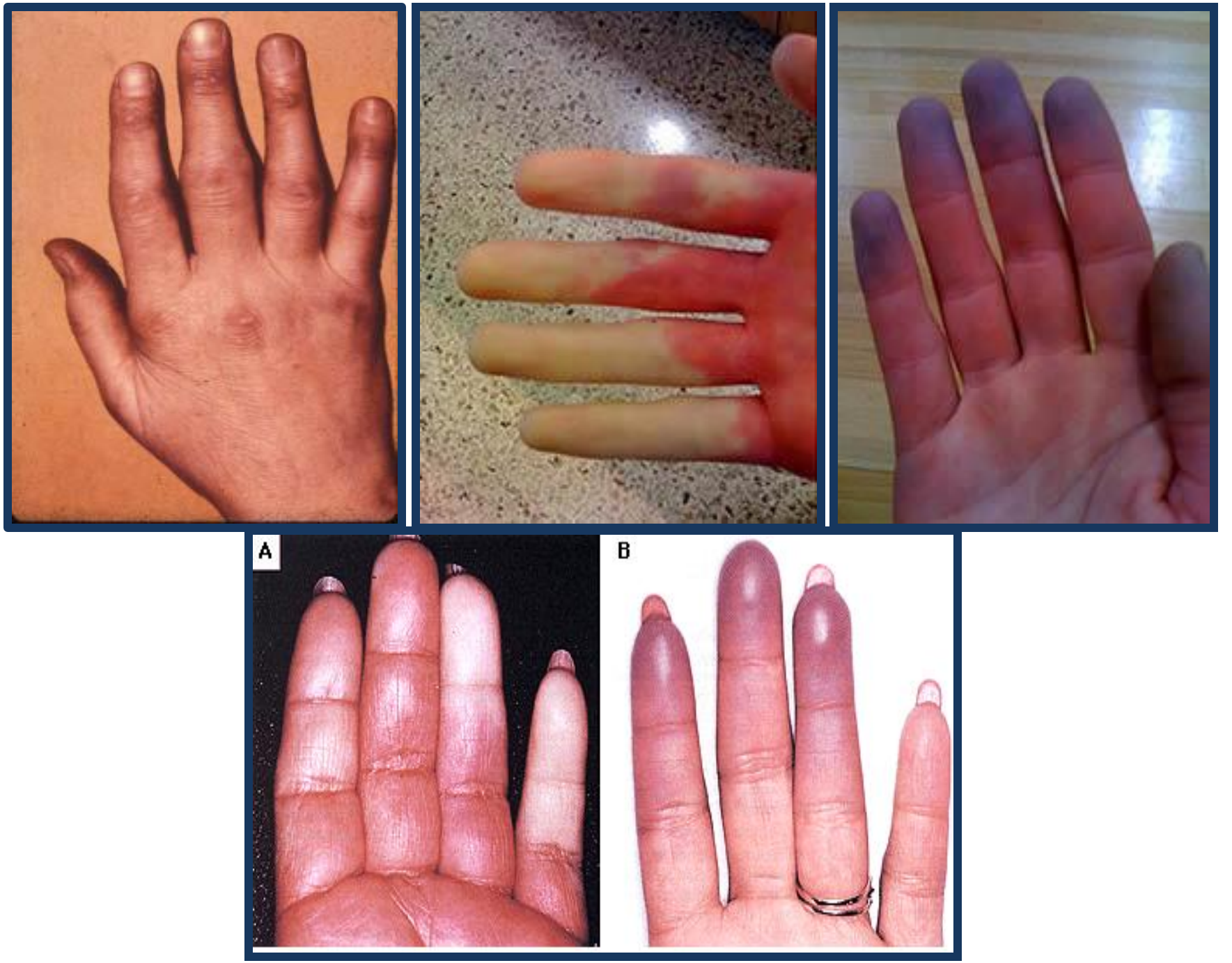








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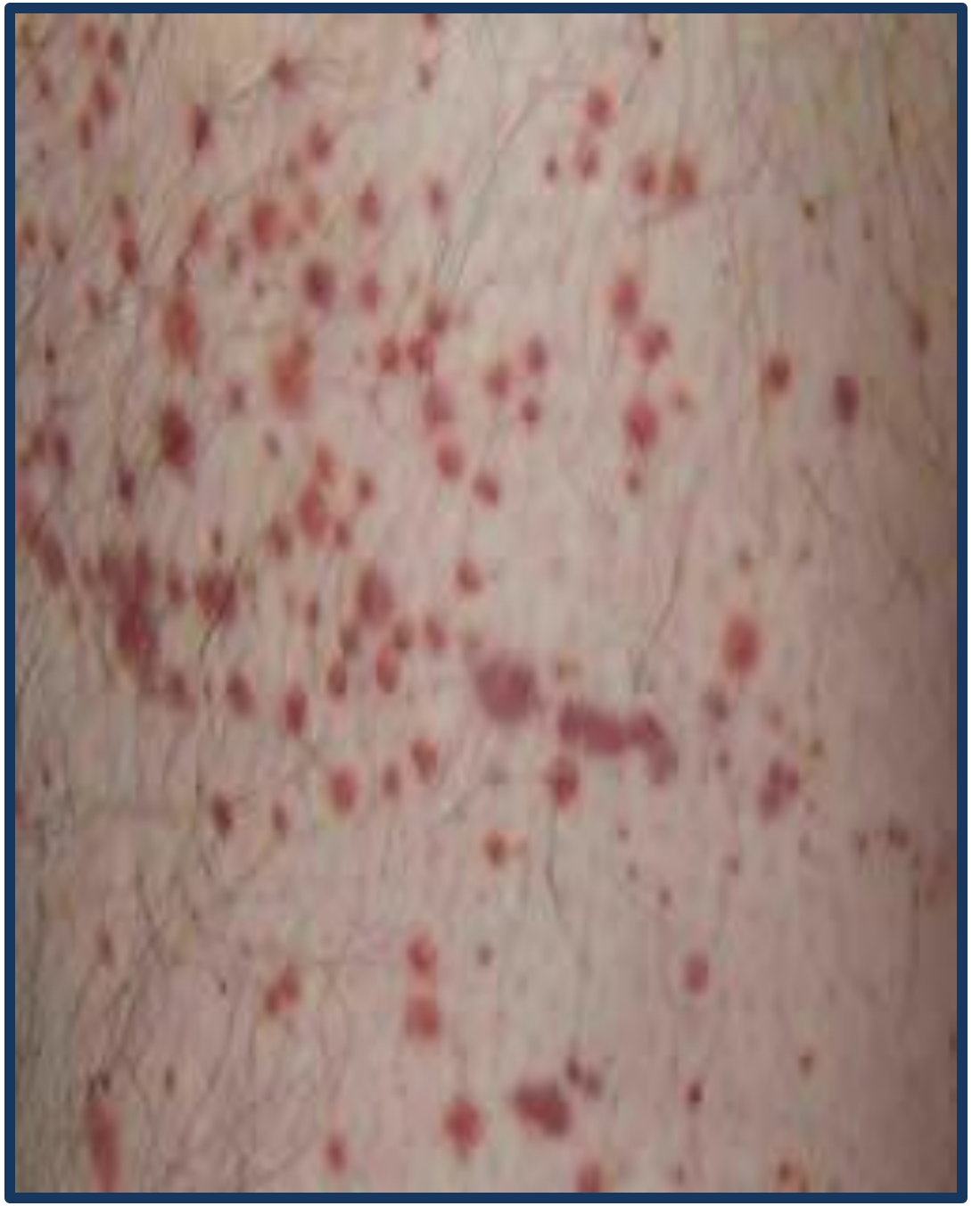
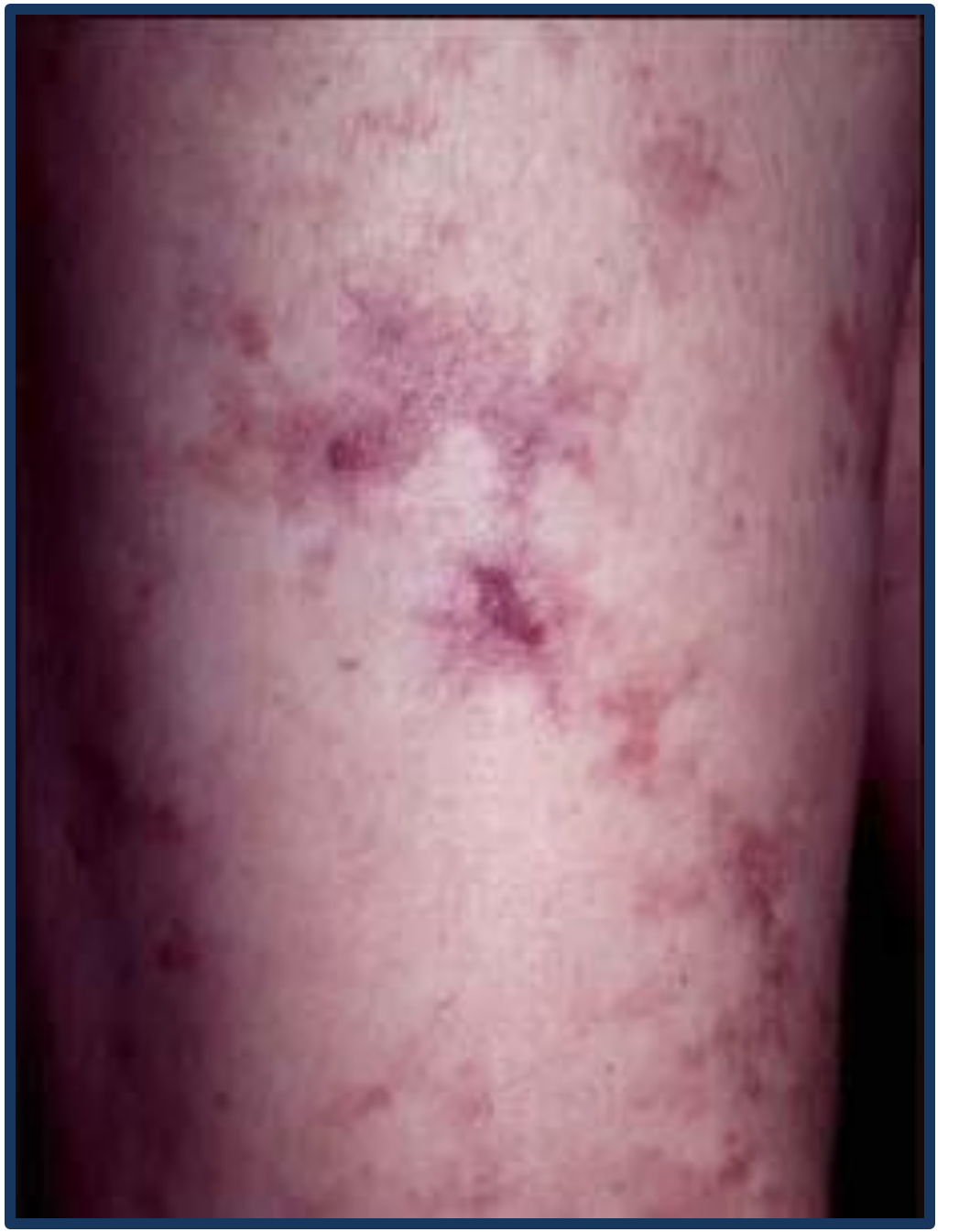




Frequency of Symptoms of Systemic Lupus Erythematosus[†]

Symptoms	Percent at onset	Percent at anytime
Fatigue	50	74-100
Fever	36	40-80+
Weight loss	21	44-60+
Arthritis or arthralgia	62-67	83-95
Skin	73	80-91
Butterfly rash	28-38	48-50+
Photosensitivity	29	≤60
Mucuous membrane lesion	10-21	27-52
Alopecia	32	55-71
Raynaud's phenomenon	17-33	30-71
Purpura	10	15-34
Uticaria	1	4-8
Renal	16-38	50-73
Nephrosis	5	11-18
Gastrointestinal	18	38-44
Pulmonary	2-12	24-98
Pleurisy	17	30-45
Effusion		24
Pneumonia		29
Cardiac	15	20-46
Pericarditis	8	8-48
Murmurs		23
ECG changes		34-70
Lymphadenopathy	7-16	31-50
Splenomegaly	5	9-20
Hepatomegaly	2	7-25
Central nervous system	12-21	25-75
Functional		Most
Psychosis	1	5-52
Convulsions	0.5	2-20

[†]Adapted from Von Feldt, JM, Postgrad Med 1995; 97:79.

Criterion	Definition
1. Malar rash	Fixed erythema, flat or raised, over the malar eminences, tending to spare the nasolabial folds
2. Discoid rash	Erythematous raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur in older lesions
3. Photosensitivity	Skin rash as a result of unusual reaction to sunlight, by patient history or physician observation
4. Oral ulcers	Oral or nasopharyngeal ulceration, usually painless, observed by physician
5. Arthritis	Nonerosive arthritis involving 2 or more peripheral joints, characterized by tenderness, swelling, or effusion
6. Serositis	a) Pleuritis--convincing history of pleuritic pain or rubbing heard by a physician or evidence of pleural effusion *OR* b) Pericarditis--documented by ECG or rub or evidence of pericardial effusion
7. Renal disorder	a) Persistent proteinuria greater than 0.5 grams per day or grater than 3+ if quantitation not performed *OR* b) Cellular casts--may be red cell, hemoglobin, granular, tubular, or mixed

8. Neurological disorder	a) Seizures--in the absence of offending drugs or known metabolic derangements; e.g., uremia, ketoacidosis, or electrolyte imbalance *OR* b) Psychosis--in the absence of offending drugs or known metabolic derangements, e.g., uremia, ketoacidosis, or electrolyte imbalance
9. Hematological disorder	a) Hemolytic anemia--with reticulocytosis *OR* b) Leucopenia--less than 4,000/mm<>3<> total on 2 or more occasions *OR* c) Lymphopenia--less than 1,500/mm<>3<> on 2 or more occasions *OR* d) Thrombocytopenia--less than 100,000/mm<>3<> in the absence of offending drugs
10. Immunological disorder	a) "Positive finding of antiphospholipid antibodies based on 1) an abnormal serum level of IgG or IgM anticardiolipin antibodies, 2) a positive test result for lupus anticoagulant using a standard method, or 3) a false-positive serologic test for syphilis known to be positive for at least 6 months and confirmed by*Treponema pallidum* immobilization or fluorescent treponemal antibody absorption test." Standard methods should be used in testing for the presence of b) Anti-DNA: antibody to native DNA in abnormal titer *OR* c) Anti-Sm: presence of antibody to Sm nuclear antigen *OR* d) False positive serologic test for syphilis known to be positive for at least 6 months and confirmed by *Treponema pallidum* immobilization or fluorescent treponemal antibody absorption test
11. Antinuclear antibody	An abnormal titer of antinuclear antibody by immunofluorescence or an equivalent assay at any point in time and in the absence of drugs known to be associated with "drug-induced lupus" syndrome

New SLICC criteria

Classify a patient as having SLE if:

A.The patient has biopsy-proven lupus nephritis with ANA or anti-dsDNA OR the patient satisfies four of the criteria, including at least one clinical and one immunologic criterion.

B.Clinical Criteria

1. Acute or subacute cutaneous lupus
2. Chronic cutaneous lupus
3. Oral/Nasal ulcers
4. Nonscarring alopecia
5. Inflammatory synovitis with physician-observed swelling of two or more joints OR tender joints with morning stiffness
6. Serositis
7. Renal: Urine protein/creatinine (or 24 hr urine protein) representing at least 500 mg of protein/24 hr or red blood cell casts
8. Neurologic: seizures, psychosis, mononeuritis multiplex, myelitis, peripheral or cranial neuropathy, cerebritis (acute confusional state)
9. Hemolytic anemia
10. Leukopenia (<4000/mm^3 at least once)

OR

Lymphopenia (<1000/mm3 at least once)

11. Thrombocytopenia (<100,000/mm3) at least once

Immunologic Criteria

1. ANA above laboratory reference range
2. Anti-dsDNA above laboratory reference range (except ELISA: twice above laboratory reference range)
3. Anti-Sm
4. Antiphospholipid antibody

lupus anticoagulant

false-positive test for syphilis

anticardiolipin–at least twice normal or medium-high titer

anti-b2 glycoprotein 1

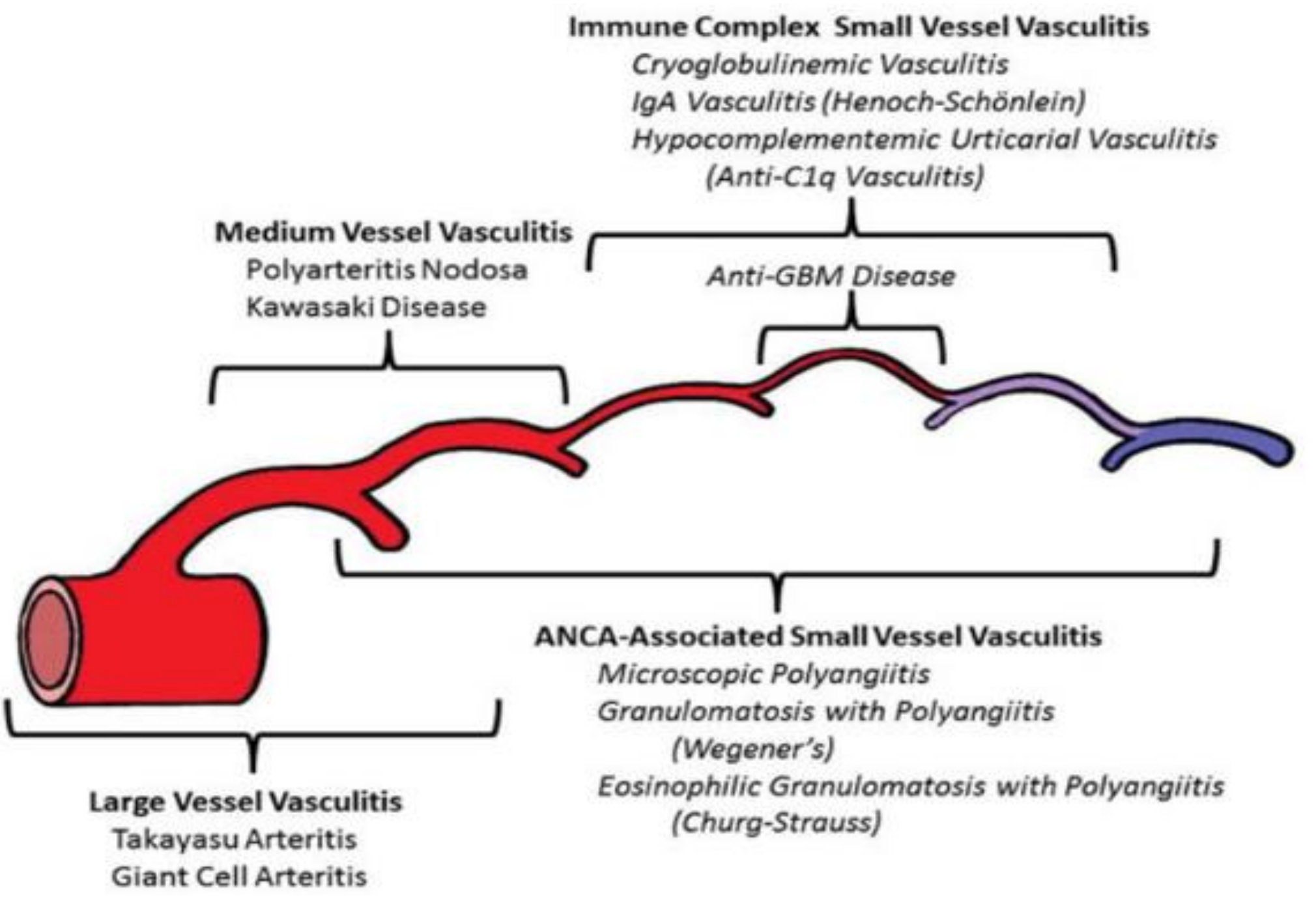
5. Low complement

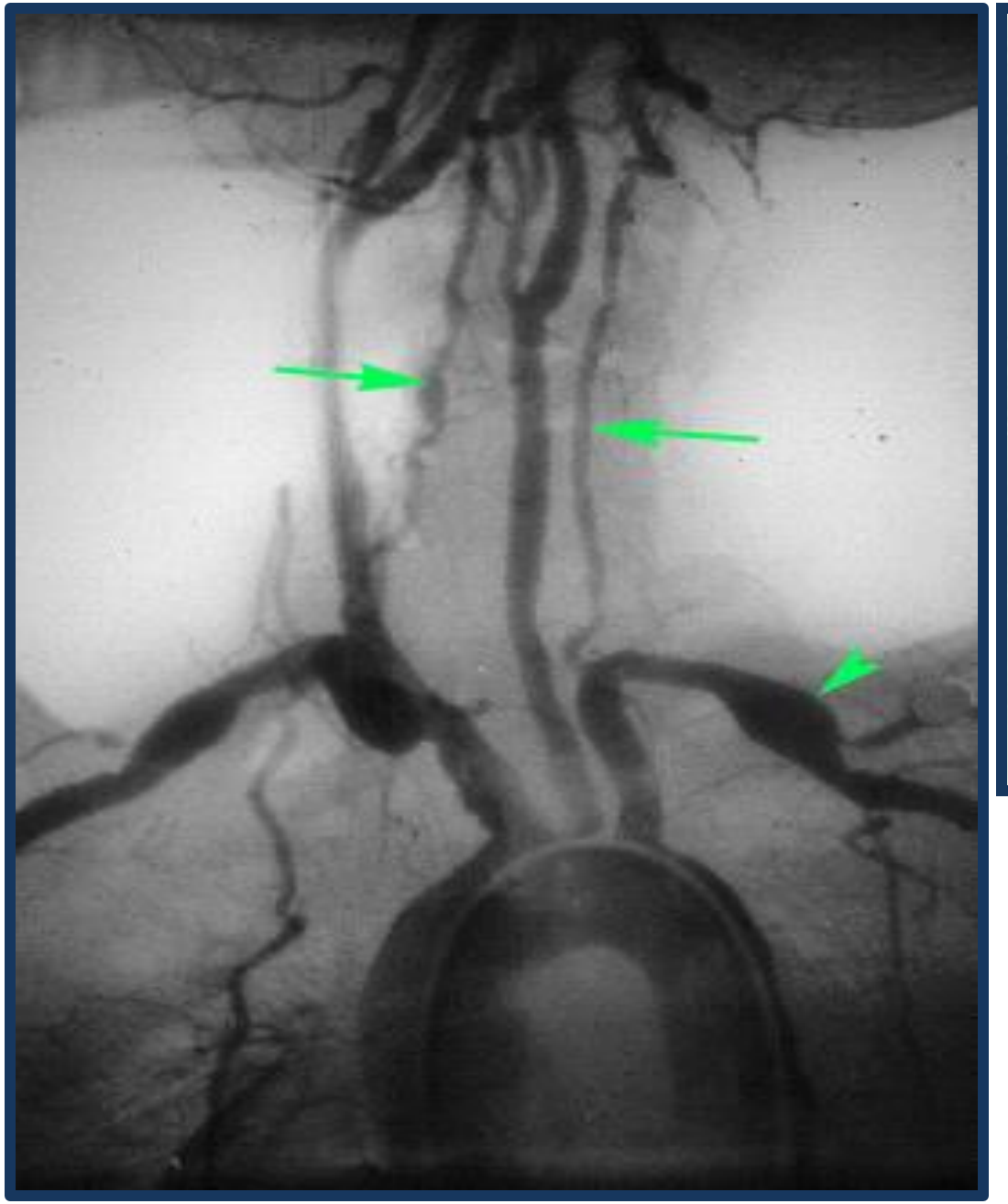
low C3

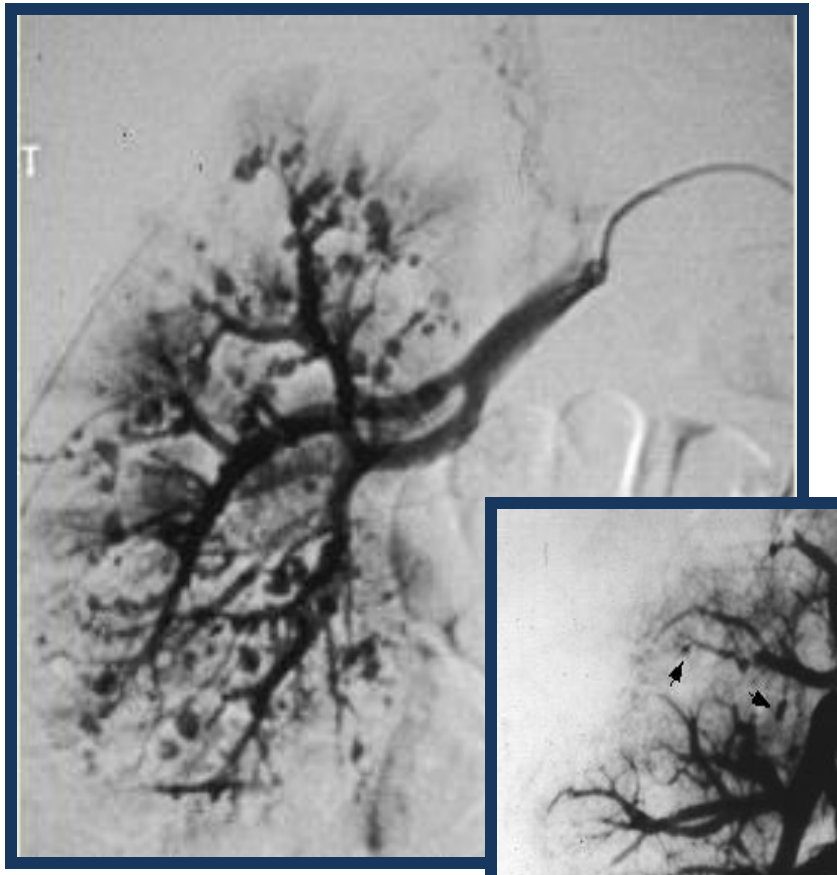
low C4

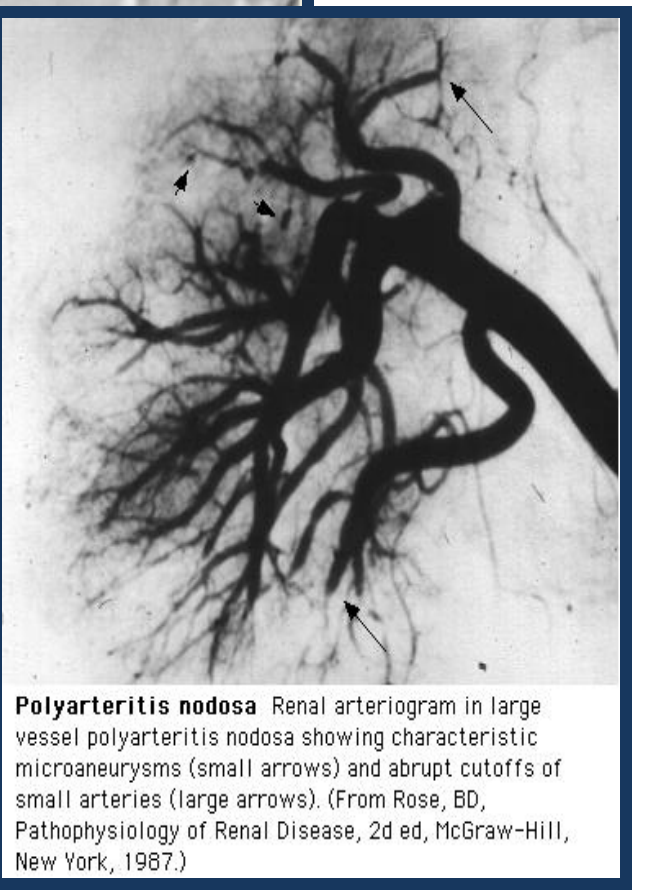
low CH50

6. Direct Coombs test in absence of hemolytic anemia.

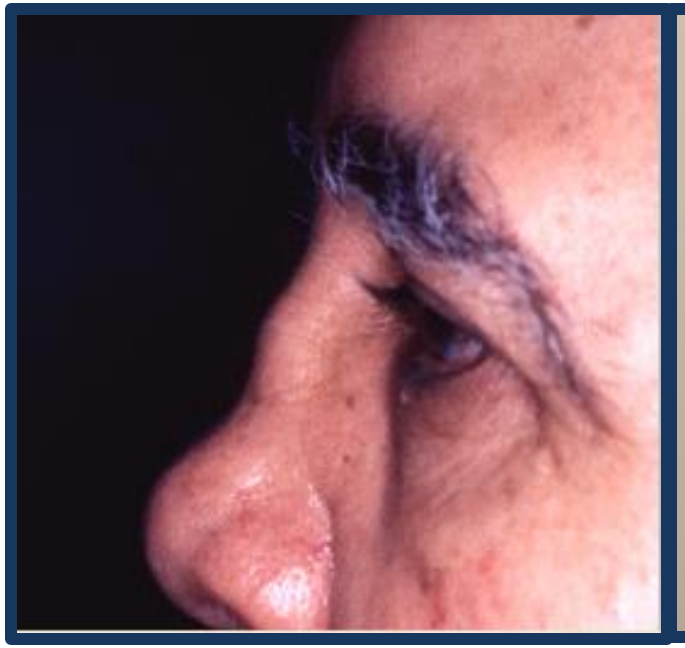

Immune Complex Small Vessel Vasculitis
Cryoglobulinemic Vasculitis
IgA Vasculitis (Henoch-Schönlein)
Hypocomplementemic Urticarial Vasculitis
(Anti-C1q Vasculitis)
Medium Vessel Vasculitis
Polyarteritis Nodosa
Kawasaki Disease
Anti-GBM Disease
ANCA-Associated Small Vessel Vasculitis
Microscopic Polyangiitis
Granulomatosis with Polyangiitis
(Wegener's)
Eosinophilic Granulomatosis with Polyangiitis
(Churg-Strauss)
Large Vessel Vasculitis
Takayasu Arteritis
Giant Cell Arteritis

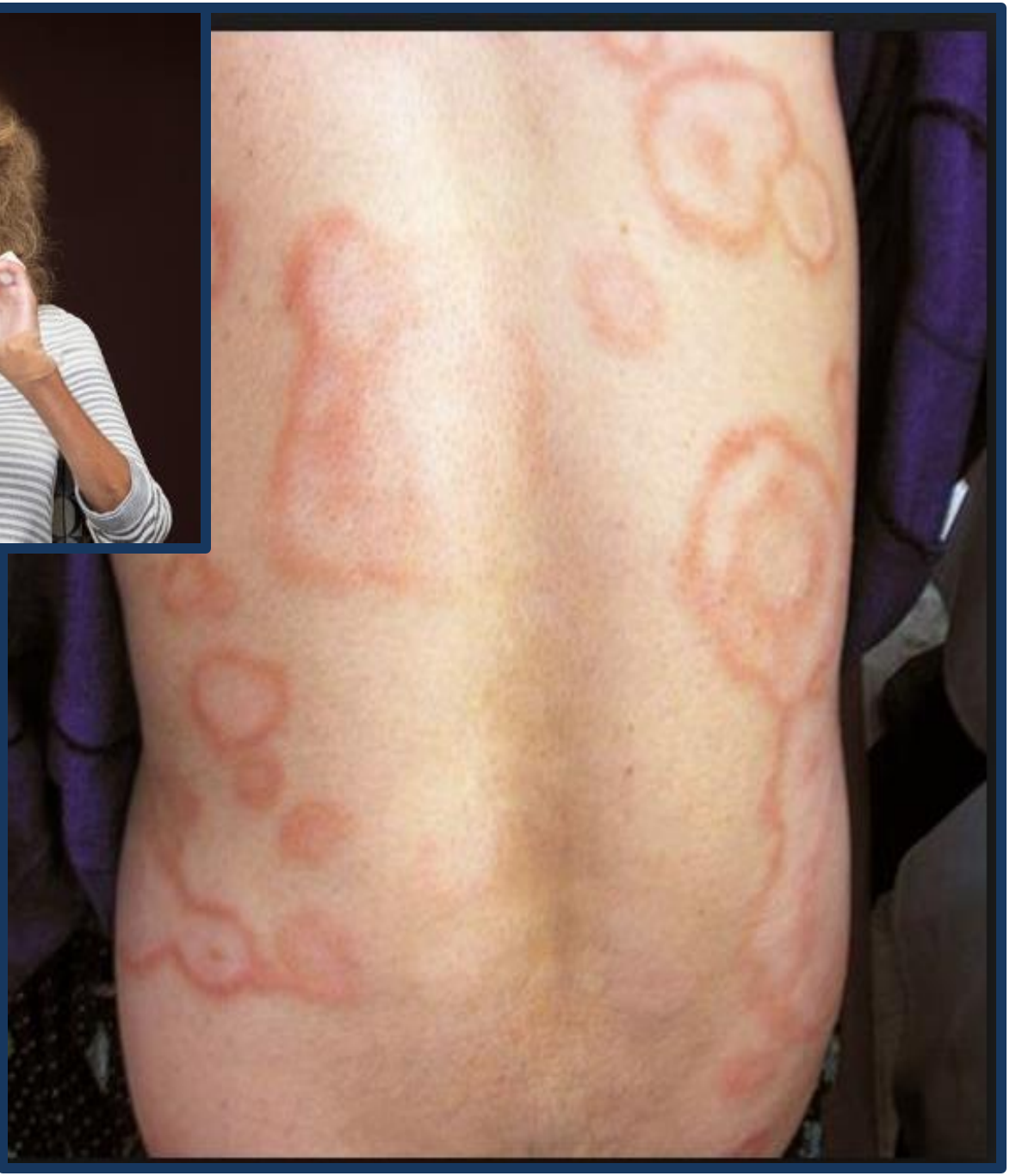


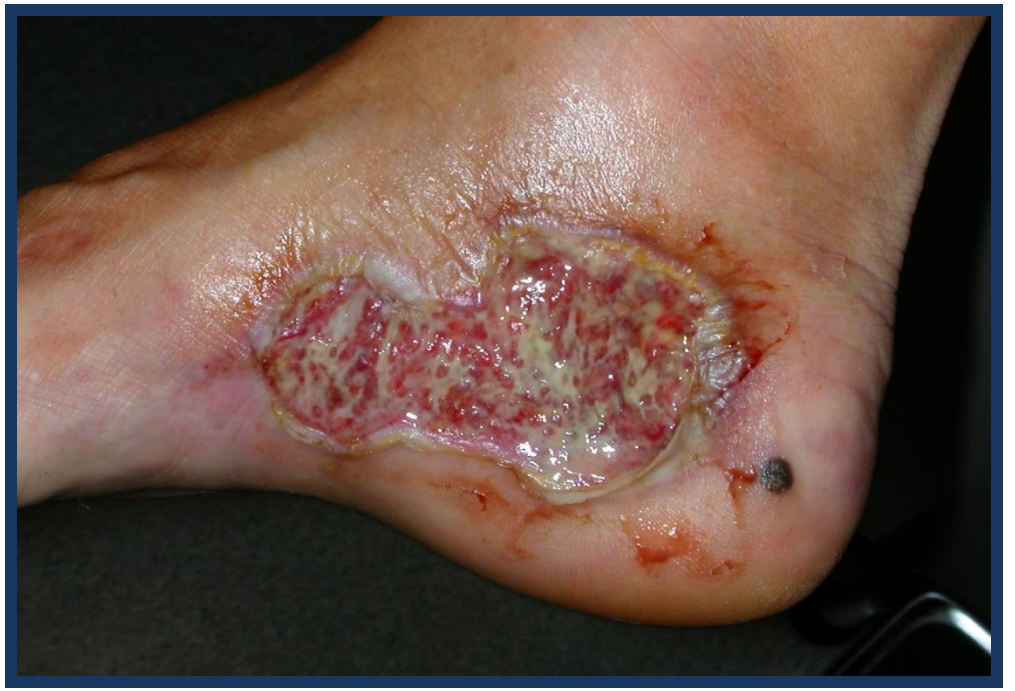


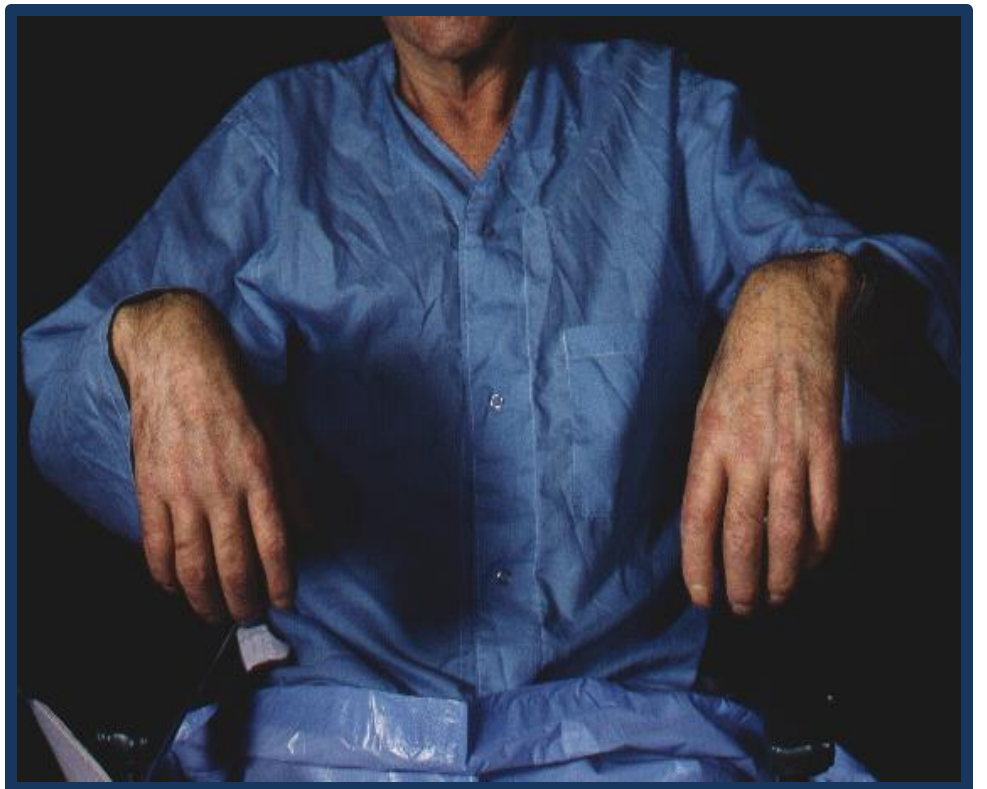


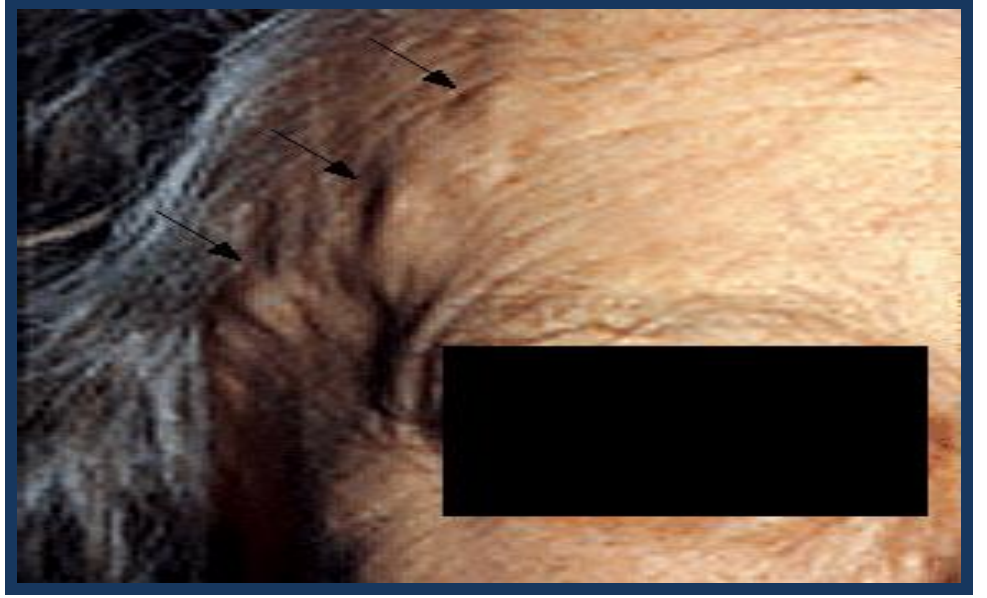
Polyarteritis nodosa Renal arteriogram in large vessel polyarteritis nodosa showing characteristic microaneurysms (small arrows) and abrupt cutoffs of small arteries (large arrows). (From Rose, BD, Pathophysiology of Renal Disease, 2d ed, McGraw-Hill, New York, 1987.)

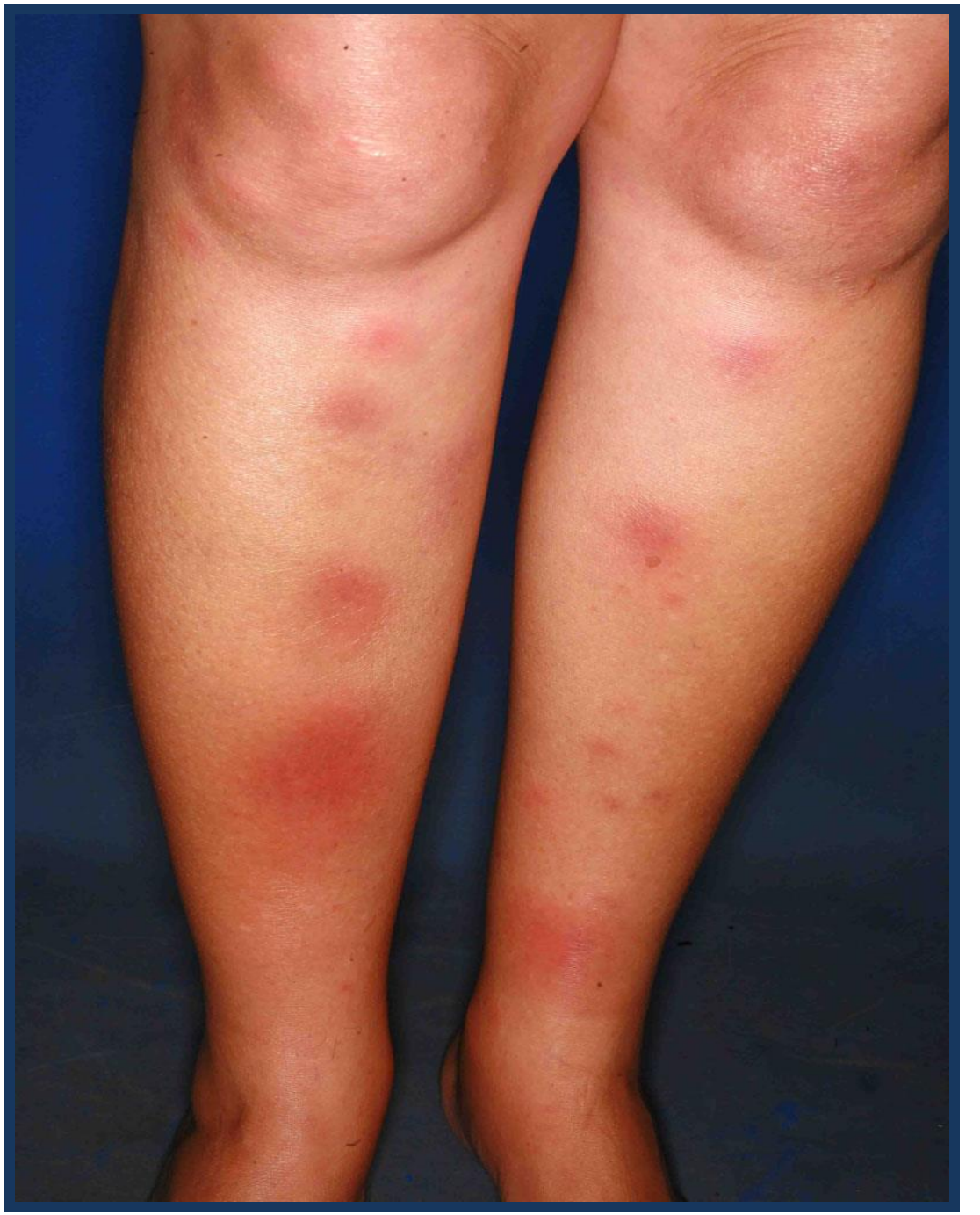


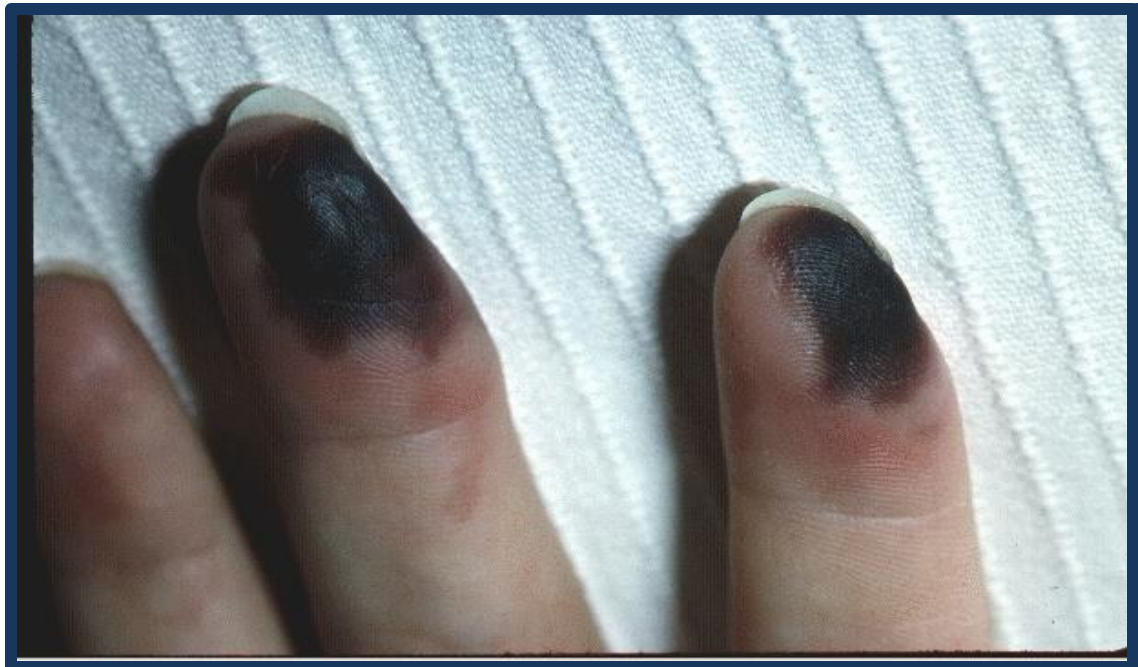


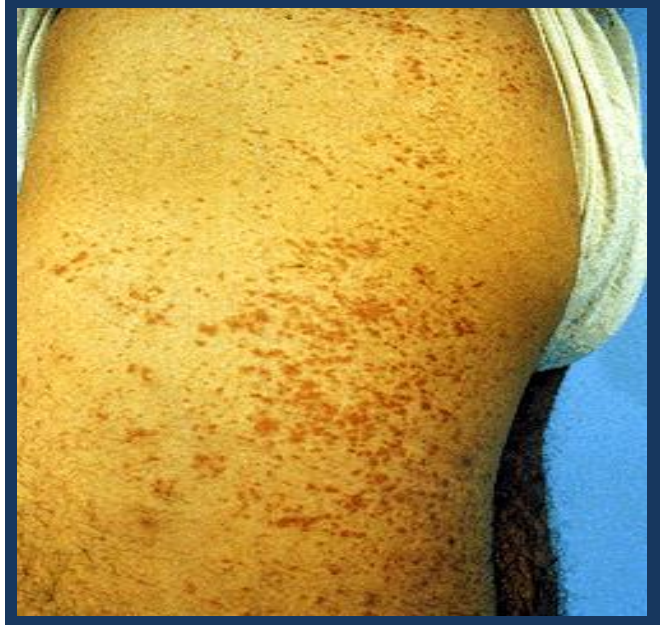


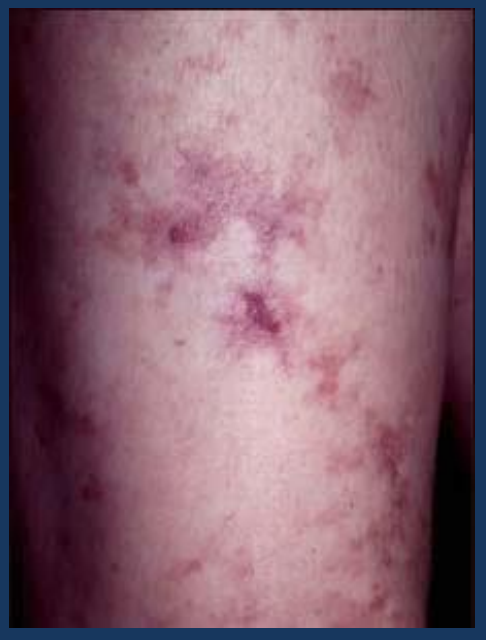


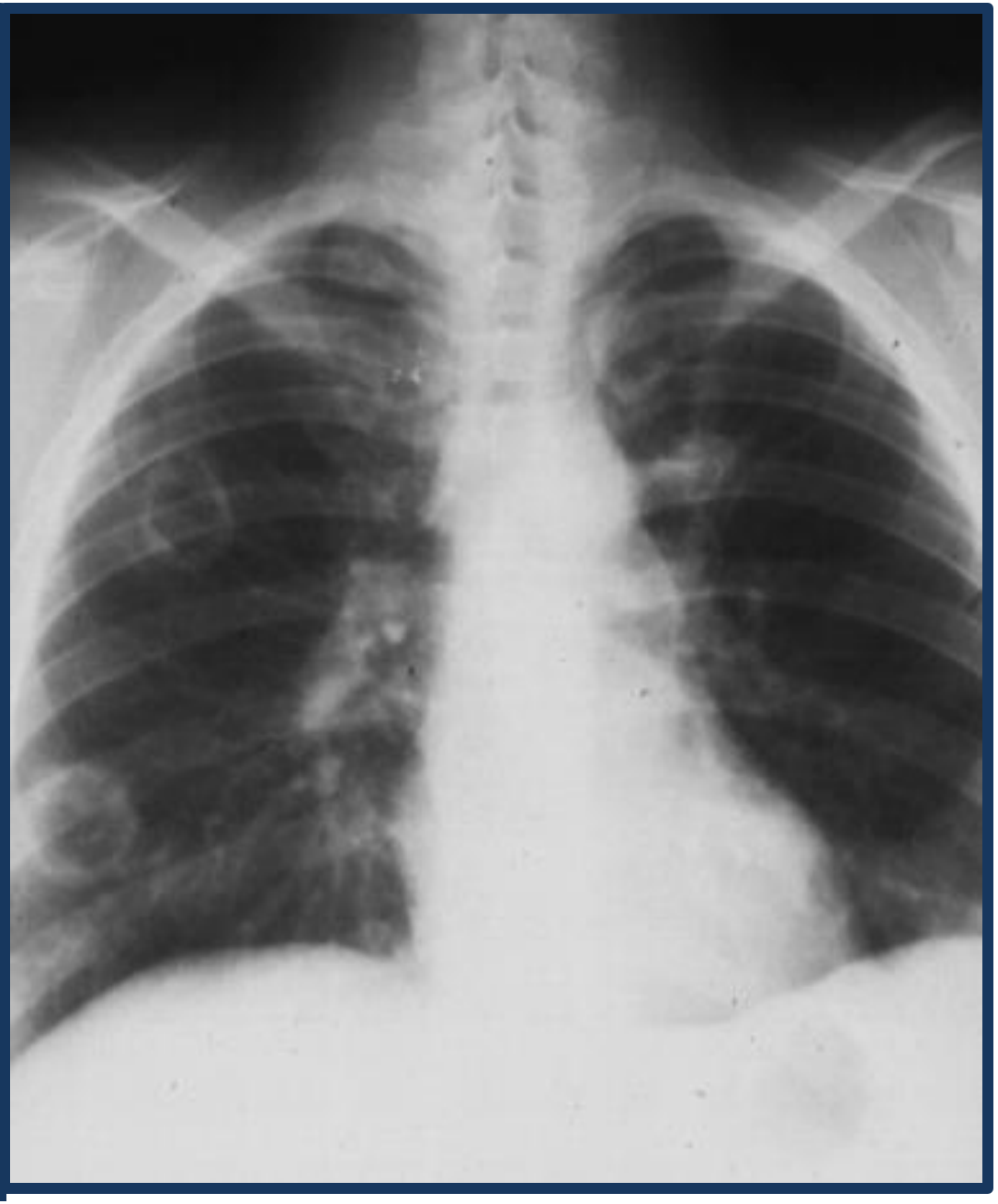






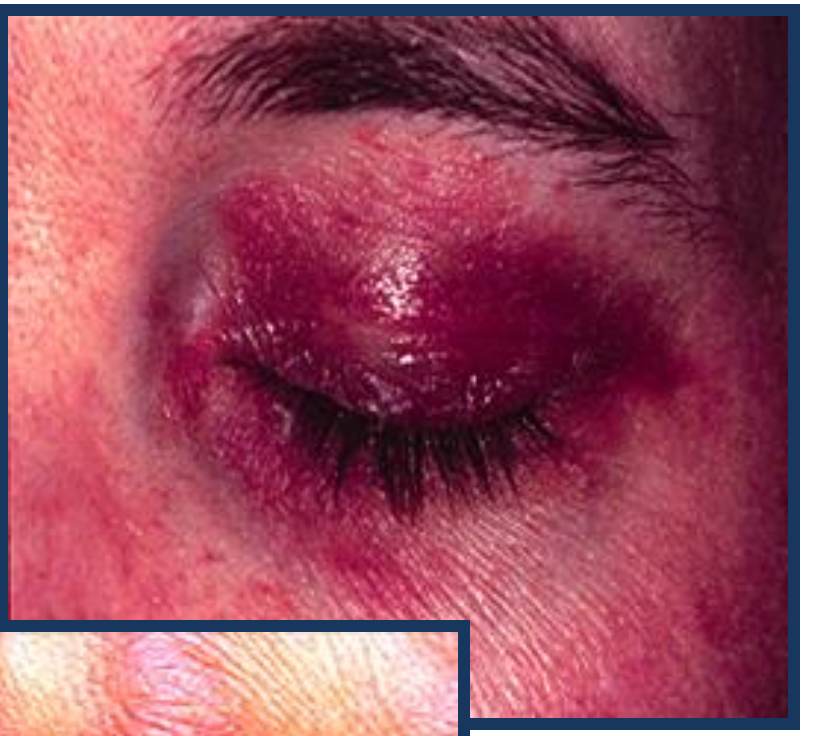


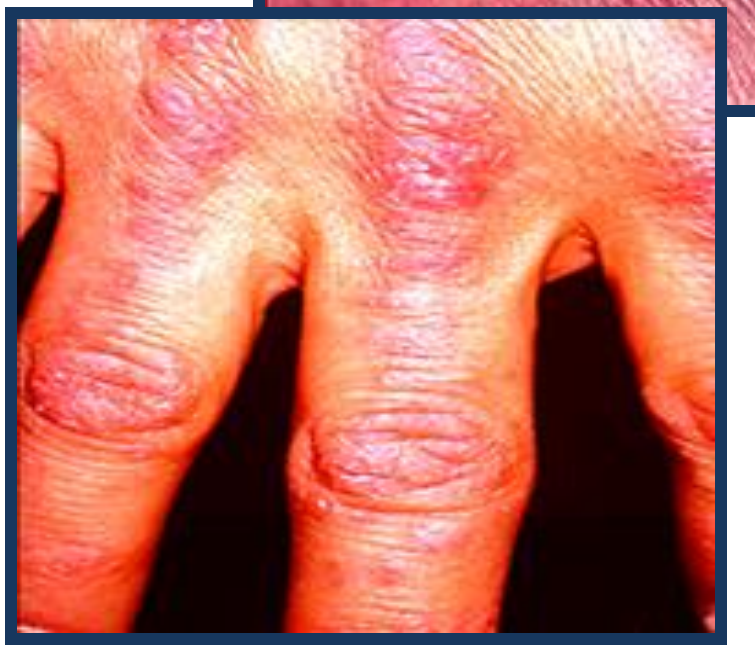




Raynaud's Phenomenon and Digital Ulcers (Pain at the tip of fingers)

Dermatomyositis





16.Others

Rheumatoid arthritis

Q: Mary is a 56 year old secretary who presents to her GP complaining of general aches and pain, but also some stiffness and swelling in her both hands for the past two months that is worse in the morning and lasts for more than 45 minutes, this was associated with low grade fever and fatigue, if she was (anti-citrulline-containing peptide antibodies) positive.

1. Identify this condition?

Rheumatoid Arthritis (which is a common autoimmune connective tissue disorder of unknown etiology which primarily affects the distal joints (result in inflammatory synovitis in a symmetrical distribution) joints but we consider it as a systematic disease with many systematic presentation.).

2. Give 2 risk factor to that precipitate this condition?

it is possible to develop rheumatoid arthritis with or without the risk factors listed below. However, the more risk factors you have, the greater your likelihood of developing rheumatoid arthritis:

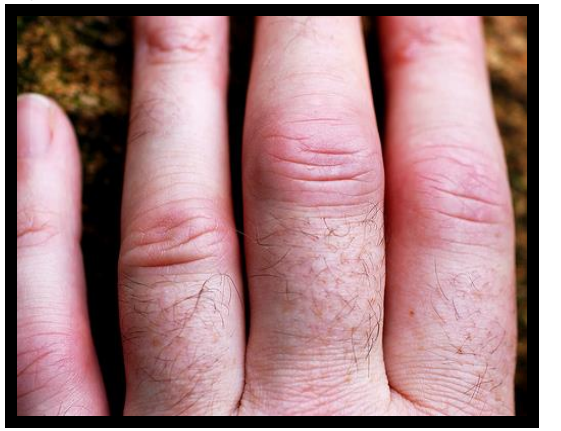
a) Age between 30 to 55 is at higher risk.

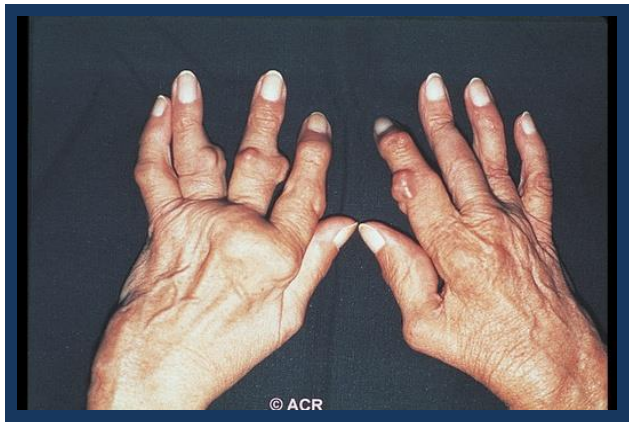
b) Women are more affected with a ratio of 3:1 to men.

c) Presence of +ve family history of RA.

d) More common in white people.

e) Less important….obesity & cigarette smoking.

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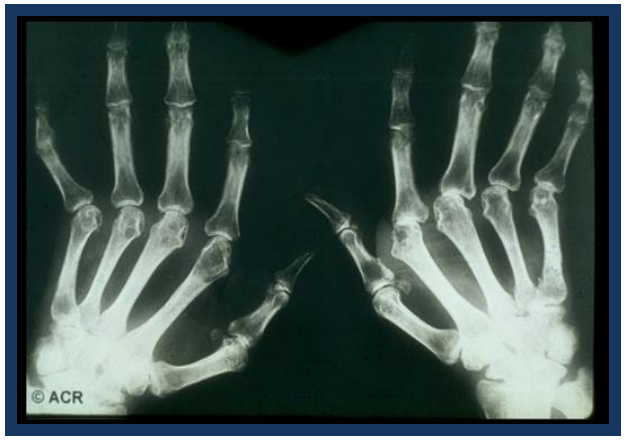
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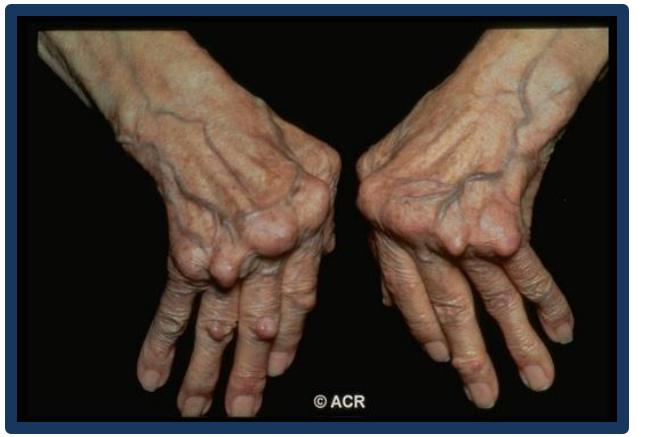
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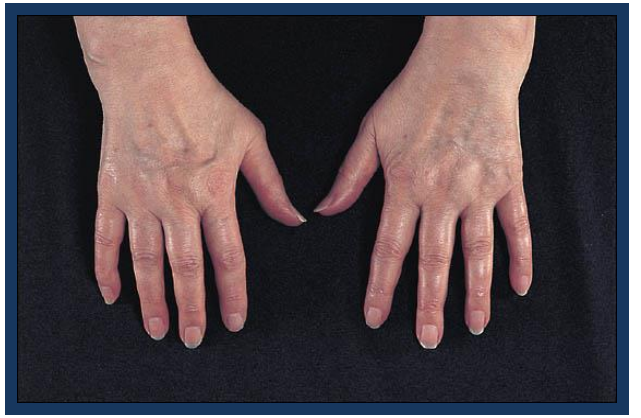

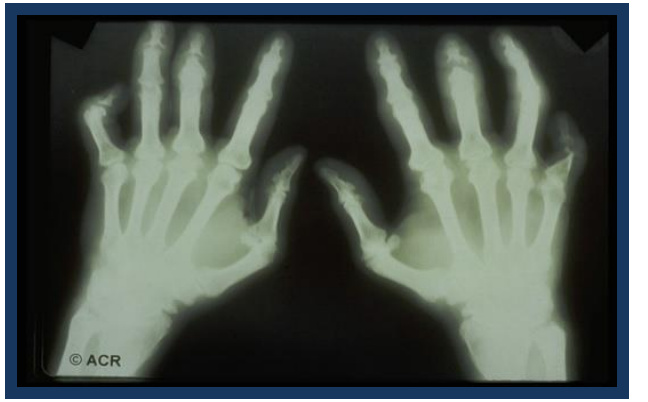
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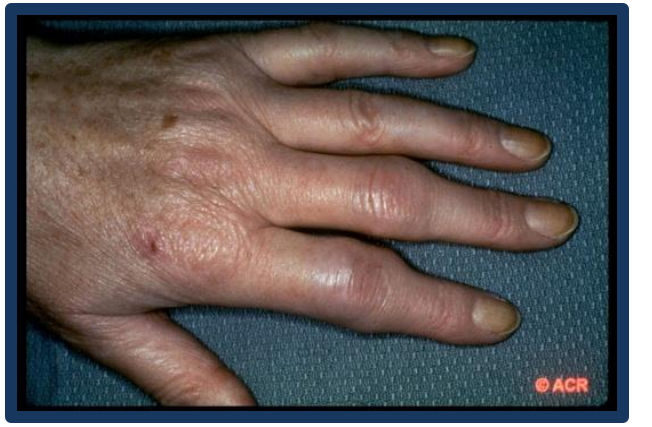
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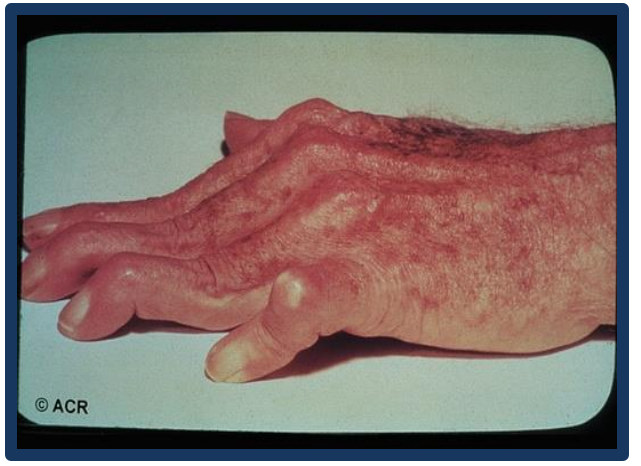
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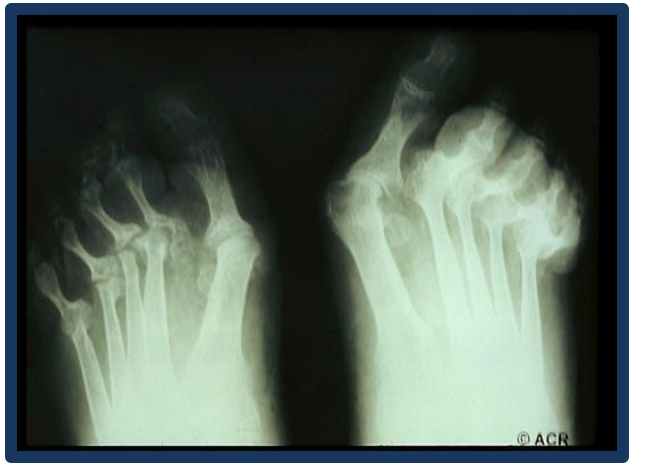
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3) Give 2 deferential diagnoses?

a) SLE is the most important one.
b) Psoriatic arthritis (which associated with skin rash, assymetrical joint involvement & cause whole digital inflammation (ductylitis)).
c) Septic arthritis.
d) Viral syndromes such as parvo virus.
e) Bacterial syndromes such as endocarditis, poststreptococcal, lyme disease…
f) Ankylosing Spondylitis.
g) Mixed Connective Tissue Diseases.
h) Gout (crystal arthopathy).

4) What is the commonest joint to be affected by this condition?

the most common joints to be affected are the MCP joints especially the 2nd & 3rd of the dominant hand.

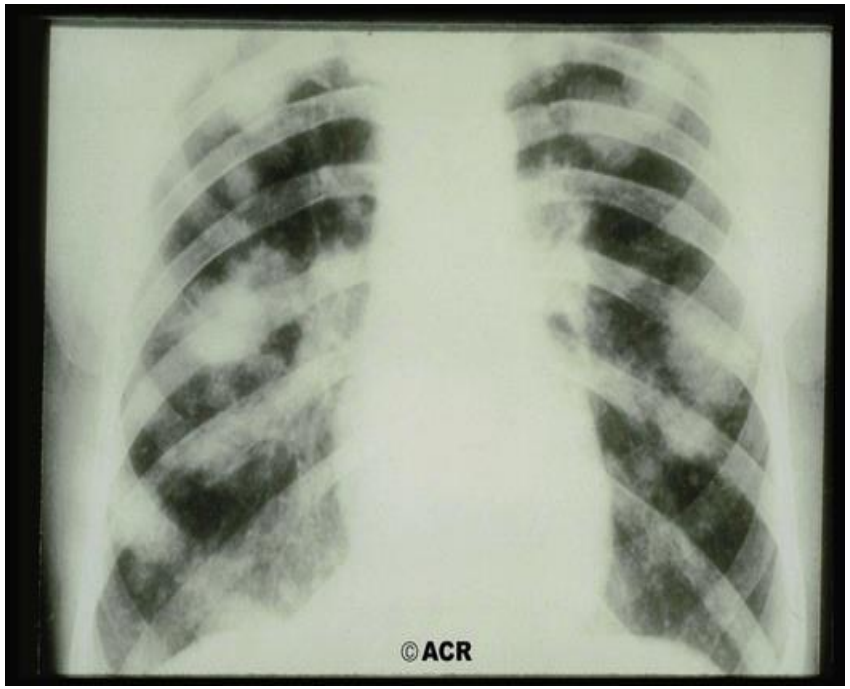
5) What is the clinical presentation of her condition?

usually the patent come with general symptoms of fatigue, wt. loss, myalagia, low grad fever could be associated with lymphoadenopathy. & local pain, morning stiffness, excess swelling & hotness at the affected joints.

6) Give 2 systematic menfistitation of such condition?

RA could involve multiple systems in the body such as:
Lungs→ →→ nodules, lower interstitial lung disease & effusion

Lung nodules in RA



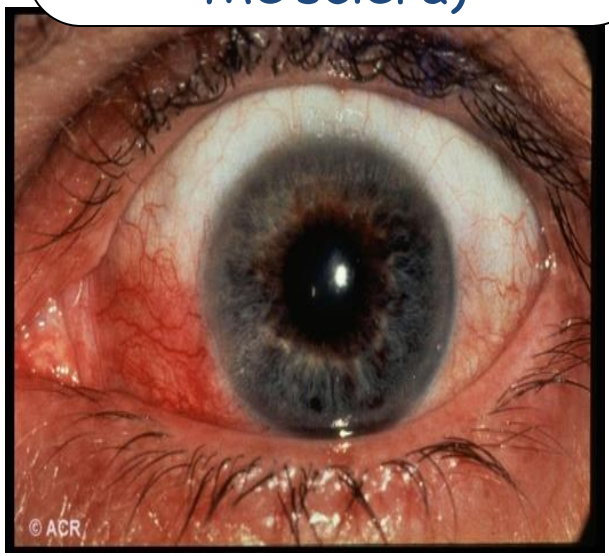
Keratoconjunctivitis

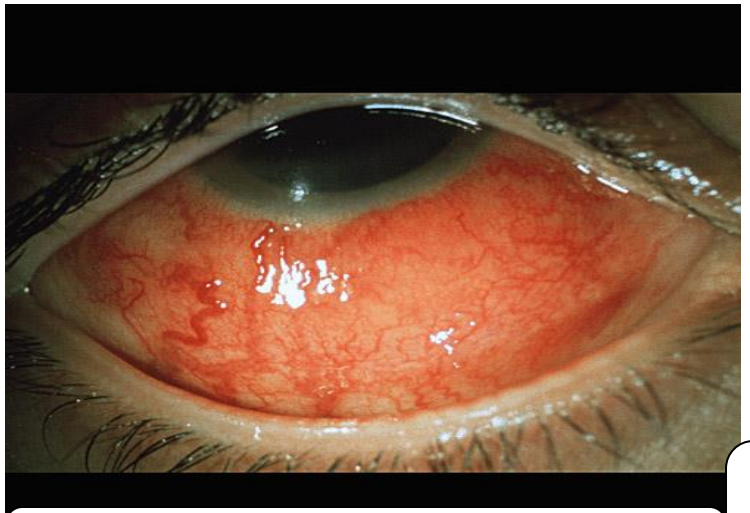
(Dryness of the eye)


Inflamed or irritated conjunctiva

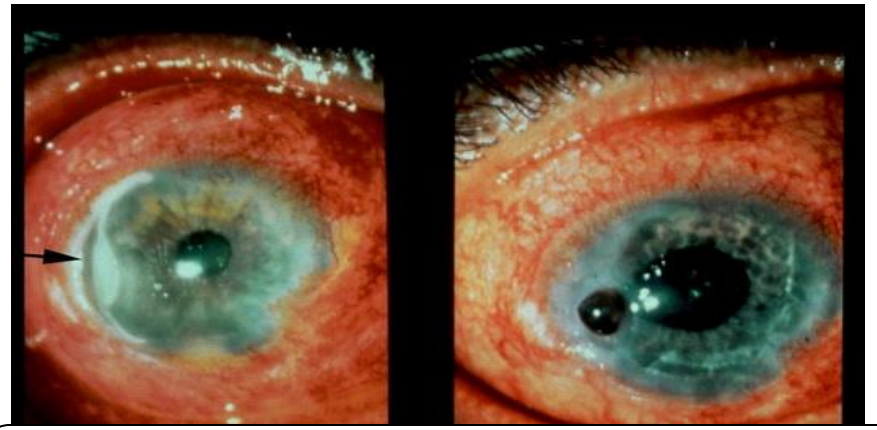
Episcleritis

(Inflammation of the superficial layer of the sclera)





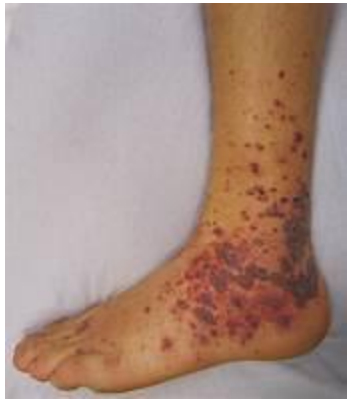
Corneal milk & perforation

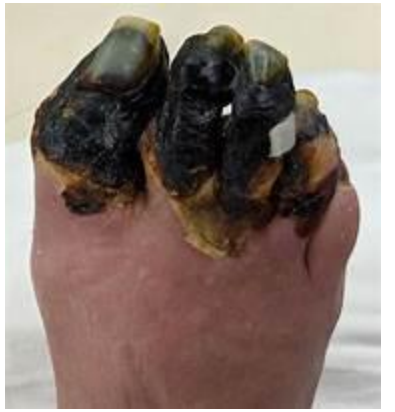


Scleritis

(Can result in scleromalacia & rupture)

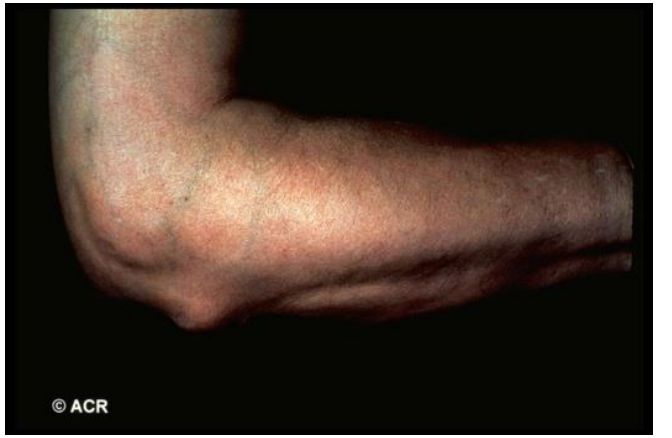
Vascular manifestation→→→ vasculaitis & gangrene.





Nervous system→→→ carpel tunnel syndrome, tarsal tunnel syndrome & spinal cord injury.

Subcutaneous nodules→→→



7) What are the other investigations you have to do to confirm your diagnosis?

a) the most sensitive test is anti-citrulline-containing peptide antibodies which mensitioned in the case above.
B) X-ray to affected joint.
c) synovial fluid analysis.

8) What is the treatment of this woman?

a) NSAIDs.
b) Steroids.
c) Hydroxychloroquine.
d) Methotrexate.
e) Surgery.

Hematology

Done by:

ابراهيم غياظة & محمد المدهون

منى أبا زيد & بيان نوافلة

زيد رطروط & آمنة الصالح

Plasma cell disorder

Q: 70 years old patient with low back pain and chronic anemia?

What is your radiological finding?

Multiple scattered lytic lesions

What is your diagnosis?

Multiple Myeloma

Mention 2 tests to confirm the diagnosis?

1.Serum protein electrophoresis.

2.Bone marrow biopsy

Two lab findings?

- hypercalcemia

-Bence jones protien in urine

Q: This X-ray was done for a 60-year old male who was C/O hypercalcemia.

What is your diagnosis?

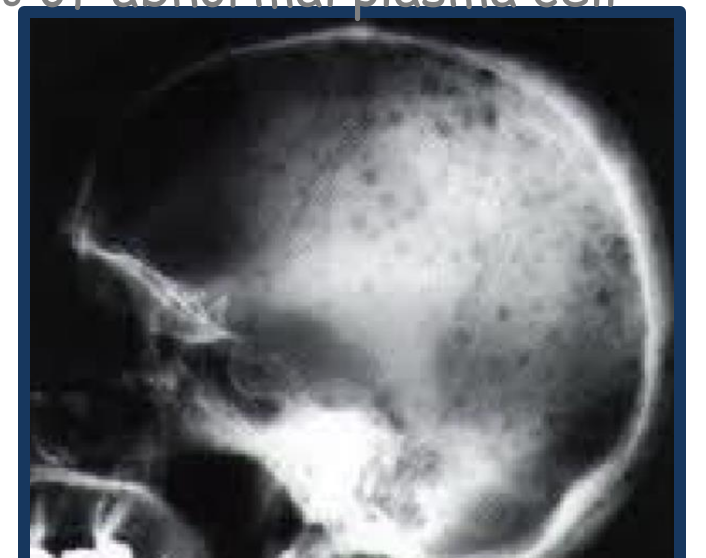
Multiple Myeloma

What is the abnormality seen in his serum protein electrophoresis

plasma cells synthesizing a single Ig (usually IgG) called monoclonal M-protein].

What you expect to find on bone marrow aspiration?

At least 10% of abnormal plasma cell



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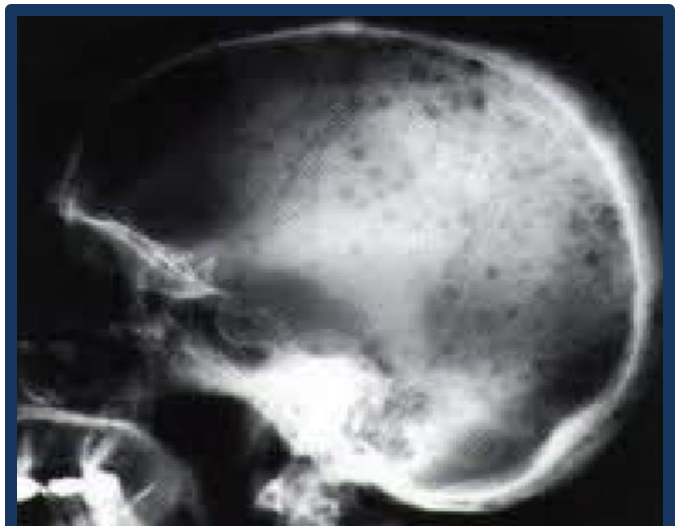

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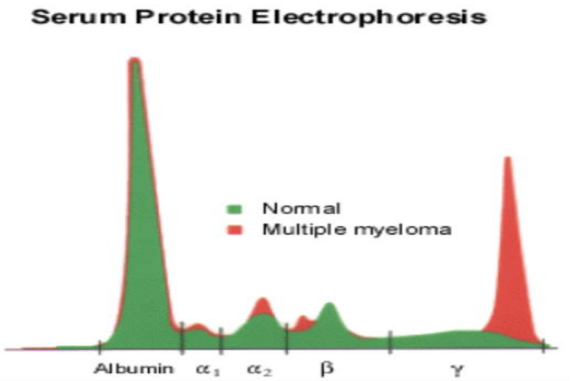


Q: Bone marrow of patient with elevated serum and urine monoclonal protein

What is the abnormality in blood film?

Rouleaux formation

What is your diagnosis?

Multiple myloma

What is the most common cause of death?

Infection (lung or urinary tract)

complications ?

-Pathological fracture

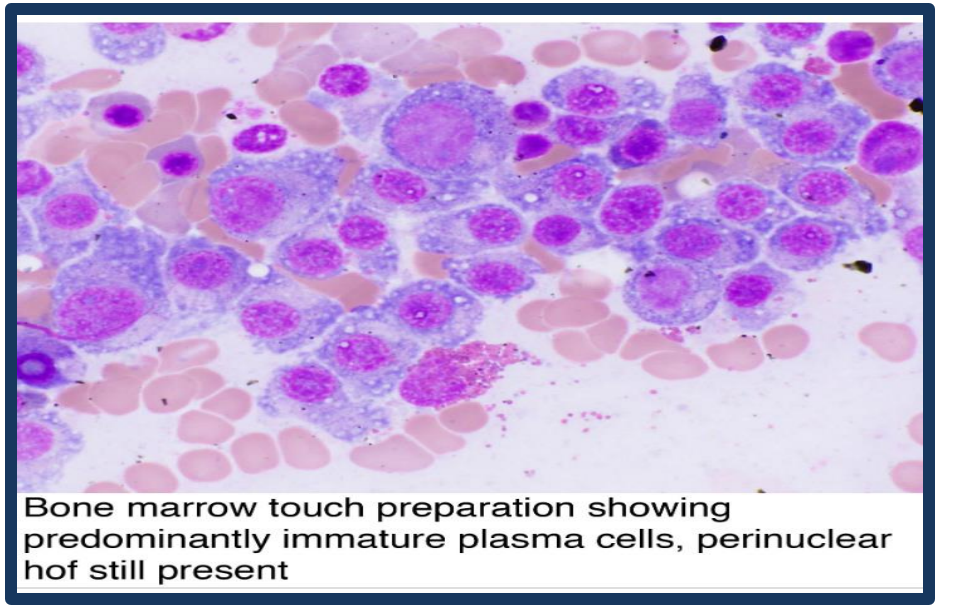
-Loose of hight secondary to collaspse of vertebrae

-amyloidosis

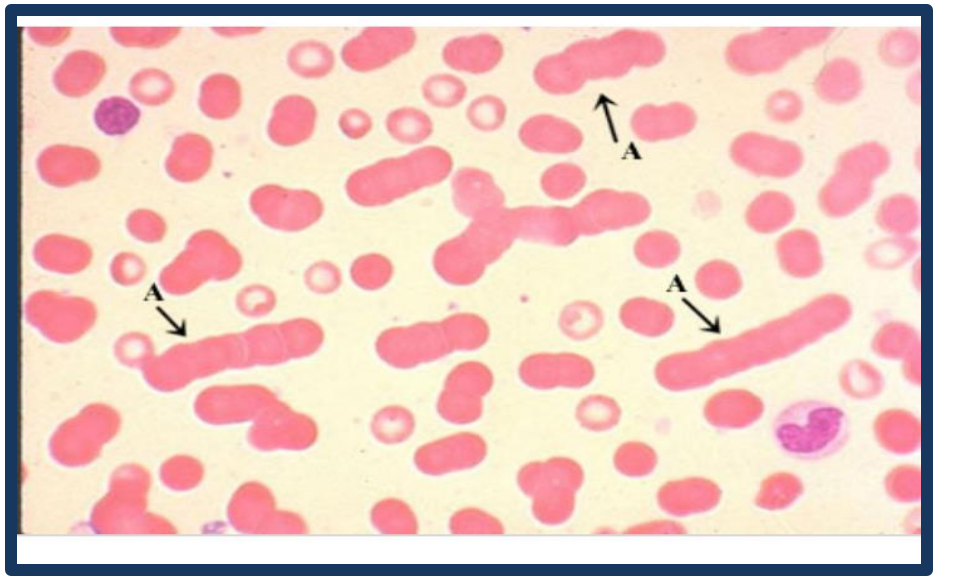
-Rena stones , renal failure

-Anemia thrombocytopenia leukemia

-Cord compression due to plasmacytoma or fractured bone fragment (rare)



Bone marrow touch preparation showing predominantly immature plasma cells, perinuclear hof still present

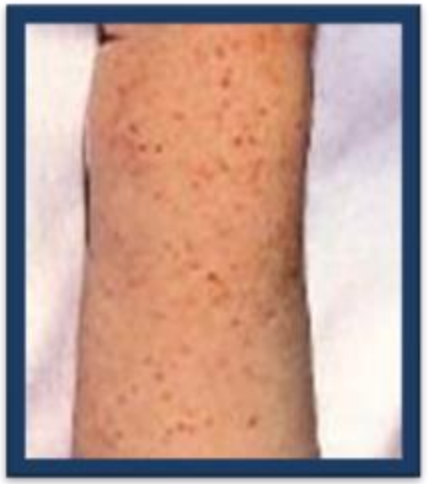


Platelet and coagulation disorder

Q: Patient present with this skin manifestation and

His investigation is :

Hb:9g\d WBC:6 x 10^9\L MCV:80fl(nl) LDH:350iu\l Bilirubin:3mg\dl
Platelete: 18,000\µl PT:12sec PTT:30sec



urea:10mmol\l Potassium:7mmol\l sodium:120mmol\l

1-What your diagnosis?

Thrombotic thrombocytopenic purpura

2-the most likely pathophysiology?

Lake functional ADAMTS13 lead to impaird regu;lation of von willebrand factor

3-Other manifestation associated with it

Fever , fluctuating transient neurological sign

4-Type of anemia associated with it?

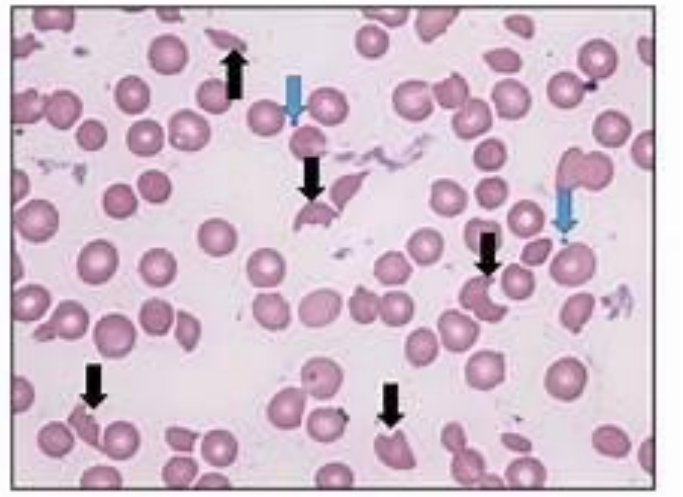
Microangiopatithic hemolytic anemia

Q: A young male patient presented complaining of bloody diarrhea for 5 days, followed by confusion, anuria, and low grade fever. Below is his blood film. His labs are:

- Platelets 55 / PT & PTT normal
- Hb 8
- Urea and creatinine high

Mention 2 findings on the blood film.

Schistocytosis (helmet cells) / spur cells …



Mention two possible DDx

TTP / HUS.

What is the Treatment ?

Plasmapheresis.

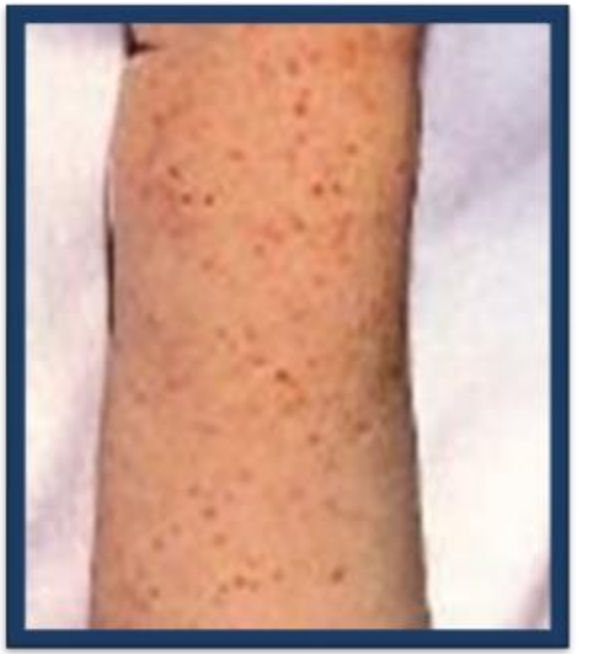
Mention two complications

- Bleeding tendency. - Multi organ failure.

Q: 30 year old female complain from easy bruise for several months and recurrent epistaxsis,, and rash(echymosis)

1) What is your first physical examination?
Palpate spleen and lymph node

On physical examination, there was no splenomegaly nor lymphadenopathy, and her CBC was:
Hb :13 WBC : 6 x 10^9\L Platelete:18,000\µl



2) What is your other investigation ?
coagulation studies (PT , PTT) , blood smear ,ANA

If all your investigation was normal ,
3) what your diagnosis and management?

diagnosis: Immune thrombocytopenic purpura

Manegment : it Depend on severity , IVIG if platelets below 20,000,immunosuppressive drugs, occasionally splenectomy

Q: 40 year female patient presented with 80.000 platlet count accidentally by doing regular CBC.

What is further investigation?

blood film to exclude pseudothrombocytopenia in which we find platelets clumps

Q: 10 YO male pt presented with this picture, with a Hx of URTI 1 week ago , what is this finding?

Petechial rash

In this case, what's the first lab test you order for this patient?

CBC (Platelet count)

Mention 3 DX?

ITP

Autoimmune vasculitis

Henoch-schonlein purpura

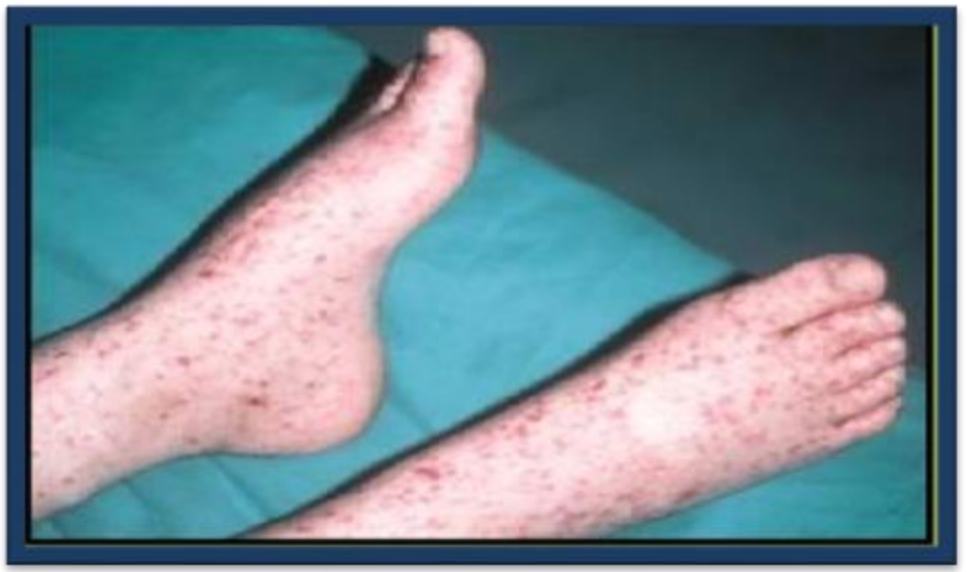
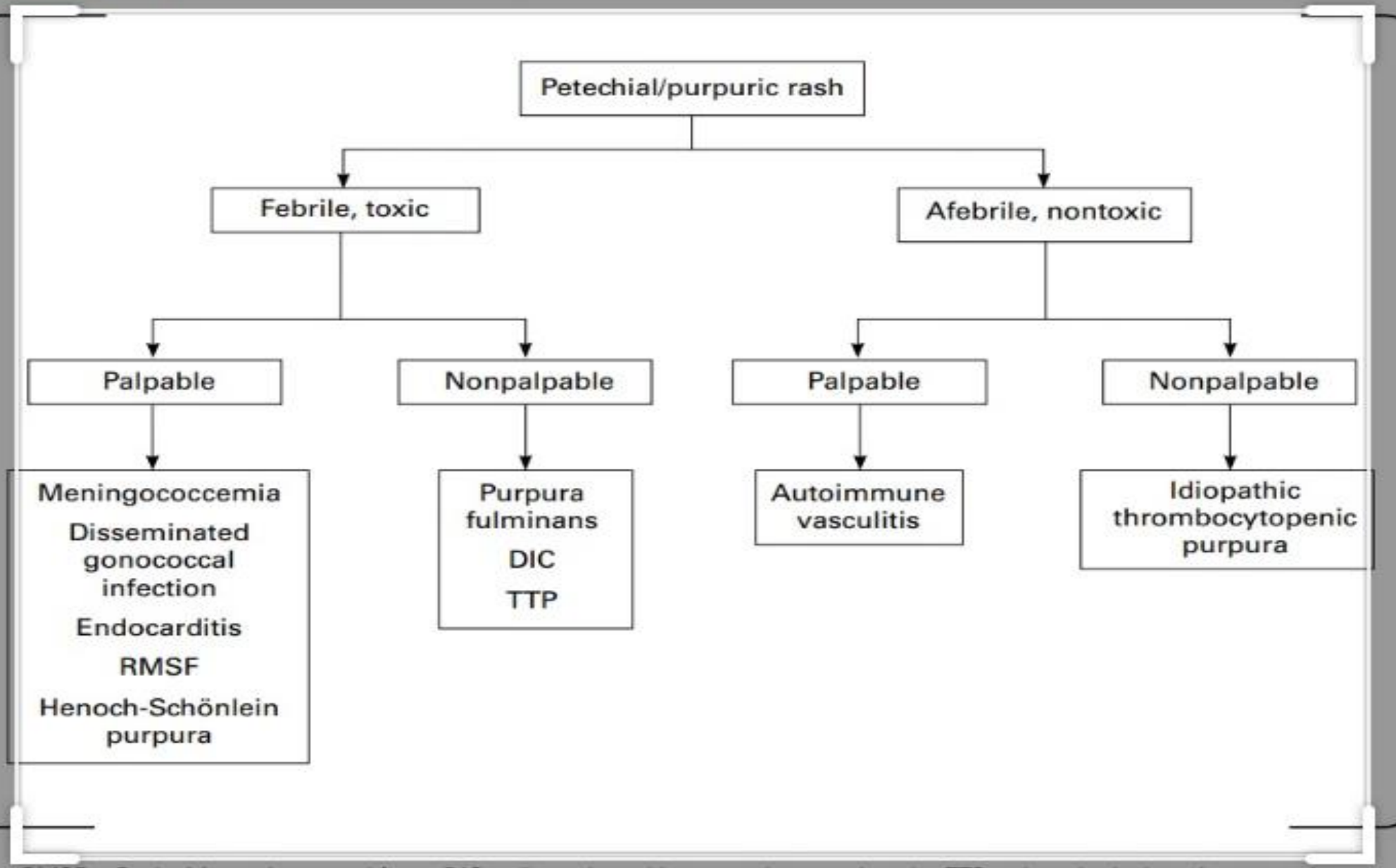


FIGURE 4. Diagnosis of the Petechial/Purpuric Rash



RMSF = Rocky Mountain spotted fever; DIC = disseminated intravascular coagulopathy; TTP = thrombotic thrombocytopenic

Q: 60-year-old female patient come to the ER with hip fracture after falling down during the admition the patient complaining from left leg swelling….DVT was diagnosed and UFH was started after 1 week the patient complain from SOB , tachycardia ,tachypnea and diagnose as a case of PE , his platelets was fallen by 50% from the base line..

1-Whats the cause that you should think about it?

HIT

2-Diagnostic test for it?

Antiplatelet factor IV antibody

3-Your management?

Stop heparin

Give a thrombin inhibitor (lepirudin,dabigatrane)

Avoid heparin in the future

Q: 6-year-old boy brought to the ER due to this painful lesion after minor trauma ,past history is significant bleeding after dental extraction

And family history of the same complain..

1)Name of this lesion ?

Hemarthrosis

2)Diagnosis should be suspected

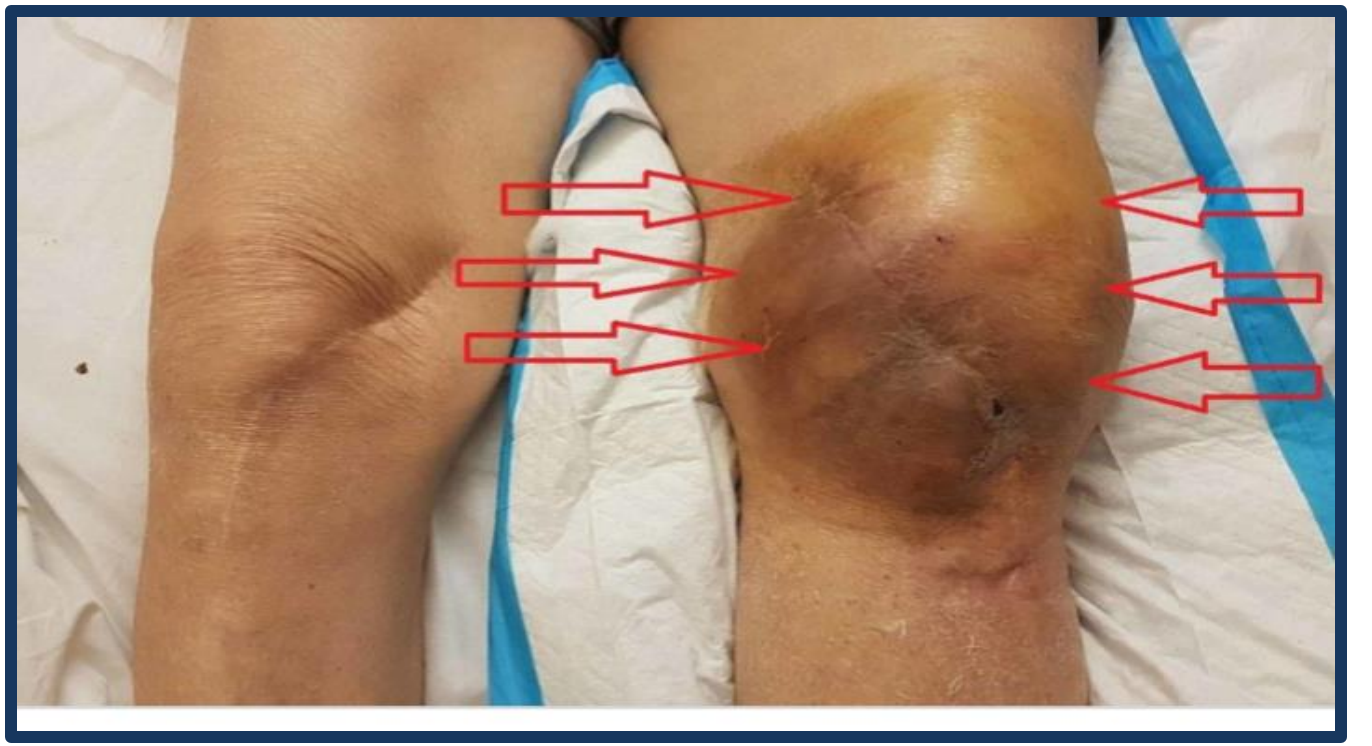
Hemophilia

3) 2 investigation?

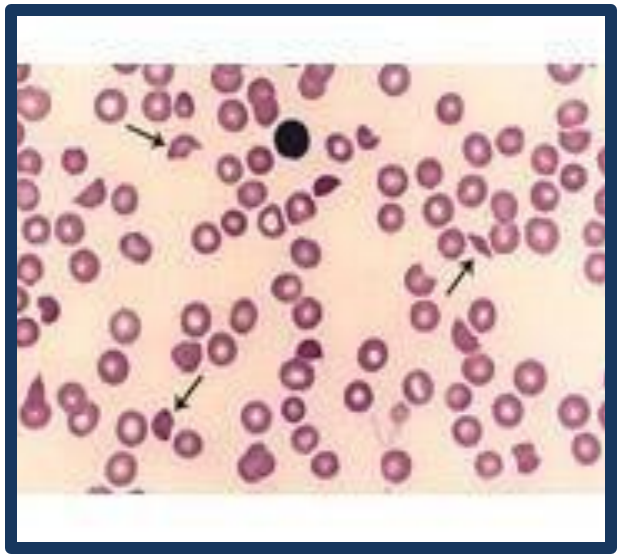
Coagulation profile

Factor VIII

vWF



Q: 37 YO pt, already admitted to ICU, sepsis had oozing from sites of cannula. A lab result: low fibrinogen Platelets count: 25,000 INR: 2.1 PTT: 49 and this is her blood film ?



What is the most likely Dx?

DIC.

Mention 2 causes.

malignancy , Obstetrical Problems, Sepsis, Massive Injury

What is the line of tt?

1-Treat Underlying Disease-(antibiotic)

2-supportive measures for sever heamorrhage (cryoprecipitate,FFP , Platelet)

3-other supportive measures (IV fluid , oxygen)

Q: Alcoholic patient comlaining from hematemesis and malena his investigations: PT 20 PTT 50 Palatelet 10,000

1-what is the cause of this pt's thrombocytopenia ??

hypersplenism due to chronic liver disease ... (not splenomegaly)

2-Most likely cause of UGIB ?

due to varices secondary to portal HTN but exacerbated by coagulopathy

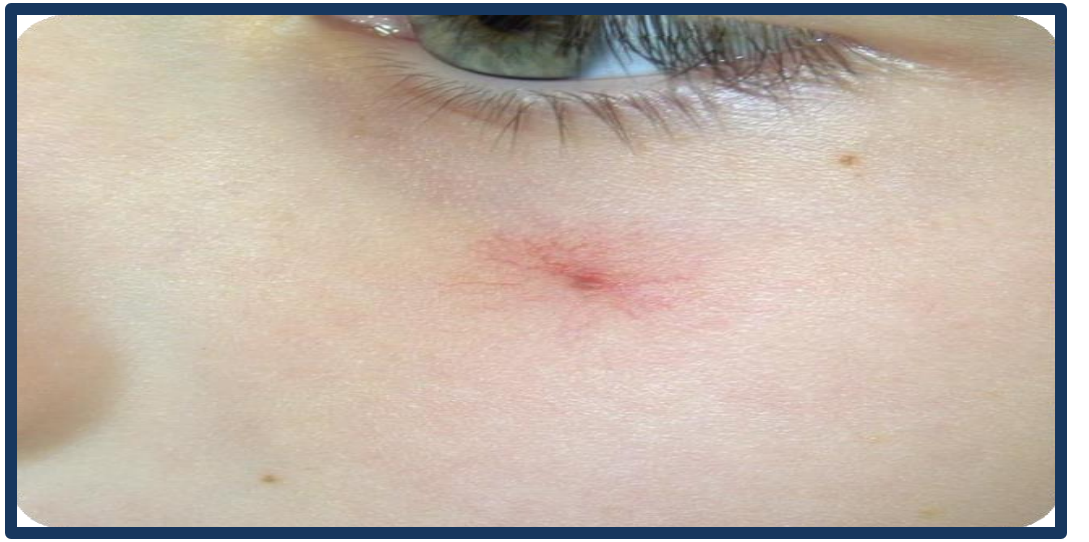
3-Management ?

1-resuscitation

2-FFP

3-Platelet

4-VK



Myeloproliferative disorders

Q: 73 YO woman with known risk factor (HTN) for cerebrovascular disease who developed a TIA like symptom & vertigo , & headache. Splenomegaly are also finding.

WBC x 109/L 18.0 [4-11], Hb g/L 200 [140-180], HCt 0.62, [80-100], Platelets x 109/L 850 [.42-.51], MCV fl 75 [150-450], Neuts x 109/L 14.6 [2-7.5], Lymphs x 109/L 2.0 [1.5-4], Monos x 109/L 0.8 [0.2-0.8], Eos x 109/L 0.1 [0-0.7], Basos x 109/L 0.5 [0-0.1].

Q1: What is the most likely Dx?
Polycythemia rubra vera.

Q2: mention 2 common secondary causes of Dx.
Tobacco abuse, Renal Cell Carcinoma, Chronic heart or lung disease.

Q3: mention 2 lines of treatment.
Phlebotomy "venesection" , low-dose aspirin.

Q: 19 year old male pt with long history of cyanosis since birth , Dx

- CBC:
  WBCs 9000
  Plt 355000
  Hgb 22
- ABGs

pH	7.41
PaCO2	33
HCO3	20
PaO2	35
O2sat	67%

2nd polycythemia rubra vera due to cyanotic heart disease

Polycythemia vera

Malignant clonal proliferation of hematopoitic stem cells
Mutation JAK2 tyrosine kinase

Clinical picture :
Symptoms of hyperviscosity (headache,dizziness,weakness ,pruritis)
Complain of severe pruritis after hot bath
Thrombotic events (DVT,CVA,MI,portal vein thrombosis)
Bleeding (GI,genitourinary,epistaxis,ecchymosis)
Splenomegaly, hepatomegaly
HTN

- Diagnosis:

  Rule out secondary polycythemia(hypoxemia,carbo monoxide)

  Cbc(⬆RBCs,⬆ Hb,⬆Hct >50%

  ⬆ PLT , ⬆ WBCs

  ⬇ serum erythropoietin

  ⬆ vit.b12 ,hyperuricemia

  Bone marrow biopsy to confirm diagnosis

- Treament :

  Repeated phlebotomyto lower Hct

- Diagnostic criteria for polycythemia vera
- 3 major or 2 major & 2 minor
- Major criteria :

  ⬆ RBCs (men >36L/kg ,women>32L/kg)

  O2 sat >92%

  Splenomegaly
- Minor criteria :

  ⬆PLT >400,000

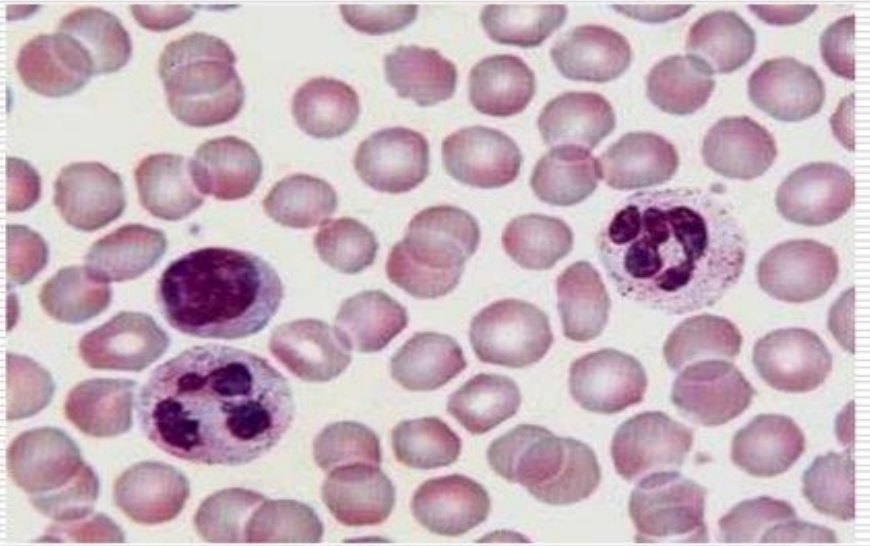
  ⬆WBCs >12,000

  ⬆leukocyte alkaline phosphatase >100 (no fever or infection)

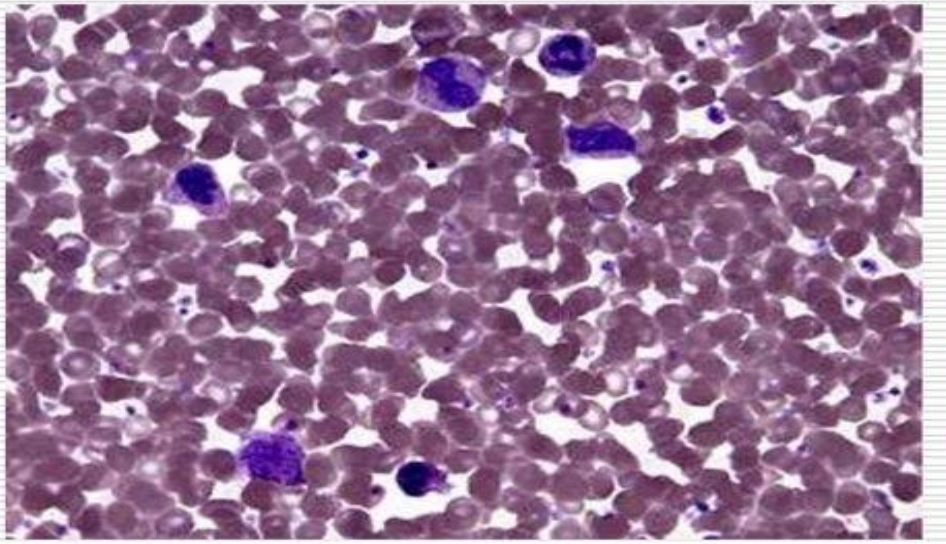
  ⬆serum b12>900

Vaquez' disease (Polycythemia vera)

Tumor induced hyperplasia of bone marrow



Normal blood smear



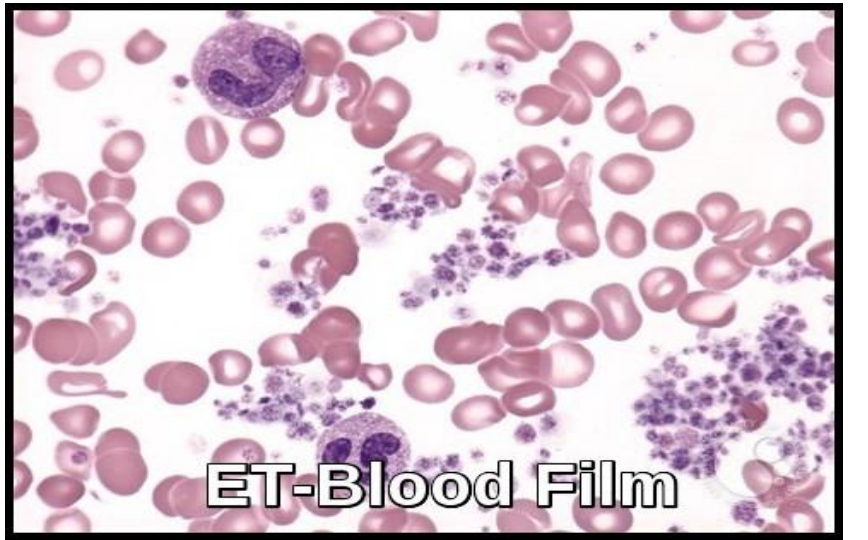
Polycytemia vera

Myelodysplastic syndrome

- Clinical picture :
- Pancytopenia , anemia , thrombocytopenia , neutropenia
- Diagnosis:
- Bone marrow biopsy show dysplastic marrow cells with blast or ringed siderobasts
- CBC : NL or mild high MCV , low reticulocyte ,Howell-jolly bodies , basophilic stippling, nucleated RBCs,hypolobulated neutrophils nuclei , large agranular PLT

Essential thrombocytosis

- PLT >600,000 , diagnosis of exclusion
- Manifested by thrombosis (CVA) or less freq bleeding due PLT dysfunction
- Splenomegaly , pseudohyperkalemia , elevated bleeding time ,erythromyalgia (burning pain and erythema of extremities due microvascular occlusion)
- Perioheral smear shows hypogranular ,abnl shape PLT
- Bone marrow biopsy shows high megakaryocytes
- JAK2 mutation 40-50% cases
- Treatment : antiplatelet agents(anagrelide & low dose of aspirin) , hydroxyurea for severe thrombocytosis



leukemia

- Atypical lymphocytes
- infectious mononucleosis
- EBV(MC) , CMV
- EBV IS transmitted by saliva
- EBV infected :

1. Pharyngitis
2. Hepatitis
3. B cell

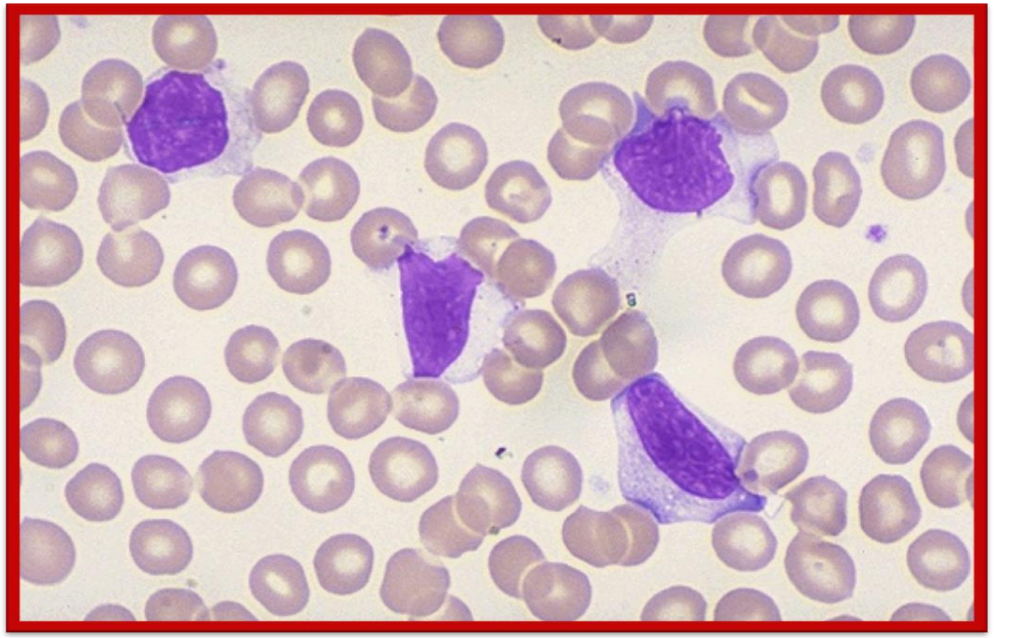
- Test for screening :

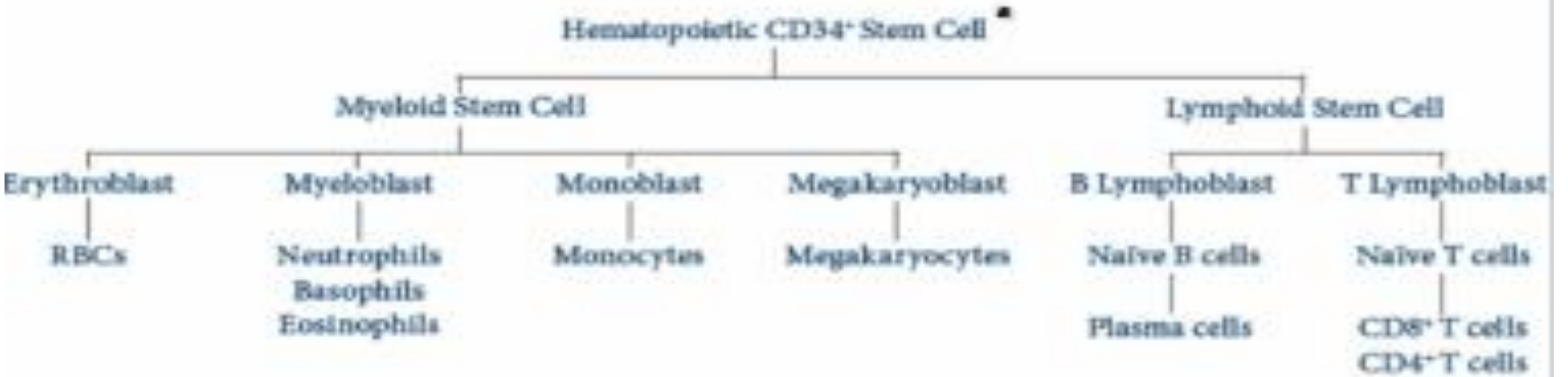
1. Monospot test (positive test within 1 week)
2. Serologic test (definitive diagnosis)

- Note : negative monospot test suggests CMV as possible cause of IM

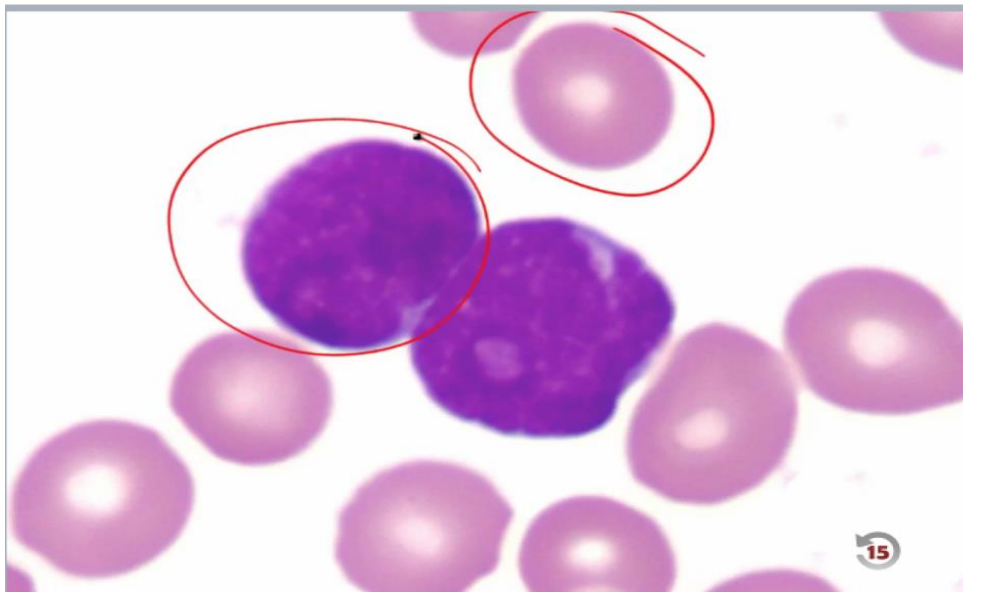
•Complication :

1. Splenic rupture
2. Rash if exposed to ampicillin
3. Dormancy of virus in B cell




Hematopoietic CD34+ Stem Cell
Myeloid Stem Cell
Lymphoid Stem Cell
Erythroblast
Myeloblast
Monoblast
Megakaryoblast
B Lymphoblast
T Lymphoblast
RBCs
Neutrophils
Basophils
Eosinophils
Monocytes
Megakaryocytes
Naïve B cells
Naïve T cells
Plasma cells
CD8+ T cells
CD4+ T cells

- Acute leukemia
- Define: neoplastic proliferation of blasts.
- Defined by: > 20% blasts in bone marrow.
- Affect on hematopoiesis :increased blasts 'crowd-out' normal hematopoiesis, resulting in "acute" presentation of anemia, thrombocytopenia, or neutropenia.



- WBC count: Blasts enter blood stream, resulting in high WBC count.

Acute lymphoblastic leukemia

- Subdivides into B-ALL & T-ALL
- Age affected commonly in children. associated with Down syndrome (usually arises after 5yo)
- Marker to distinguish a cell as a lymphoblast:
- TdT, a DNA polymerase (TdT absent in myeloid blasts and mature lymphocytes)

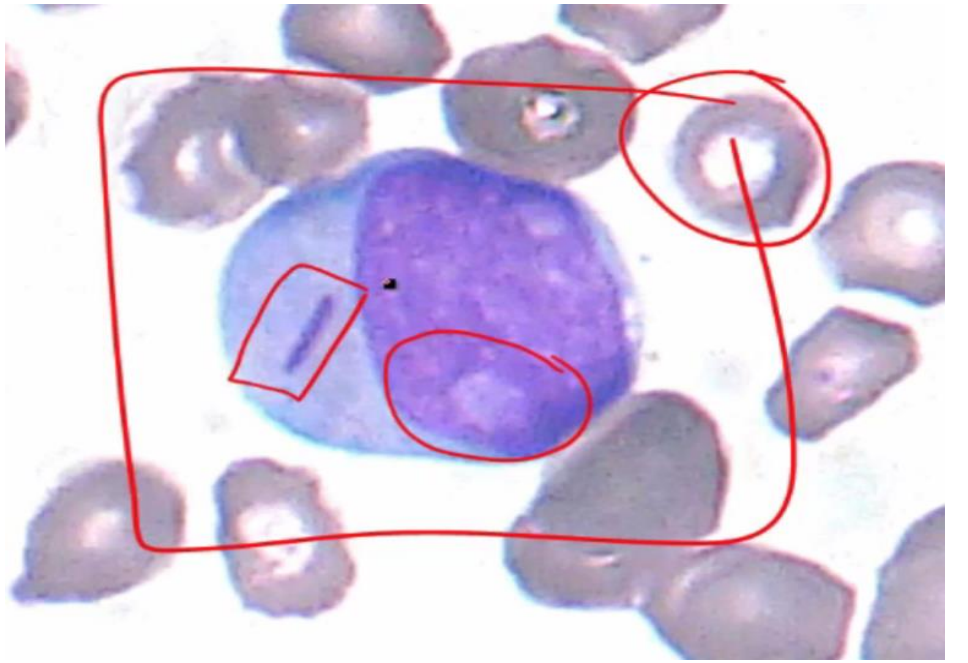
B-ALL (B-cell Acute lymphoblastic leukemia)

- CD: +CD10, +CD19, +CD20, +TdT
- Chemo: excellent response
- t(12;21) Good prognosis - more commonly seen in kids
- t(9;22) poor prognosis; commonly seen in adults. Philadelphia+ALL

T-ALL (T-cell Acute lymphoblastic LYMPHOMA)

- CD: +CD2-CD8, +TdT. NOT CD10
- presents as: thymic mass in teenager

- Define neoplastic:accumulation of immature myeloid cells (>20%) in bone marrow
- Distinguished by: +MPO (myeloperoxidase) in cytoplasm. Or, crystal aggregates of MPO seen as Auer rods
- Sub classifications (3): Based on lineage of immature myeloid cells. Acute promyelocytic leukemia, acute monocytic leukemia, acute megakaryoblastic leukemia

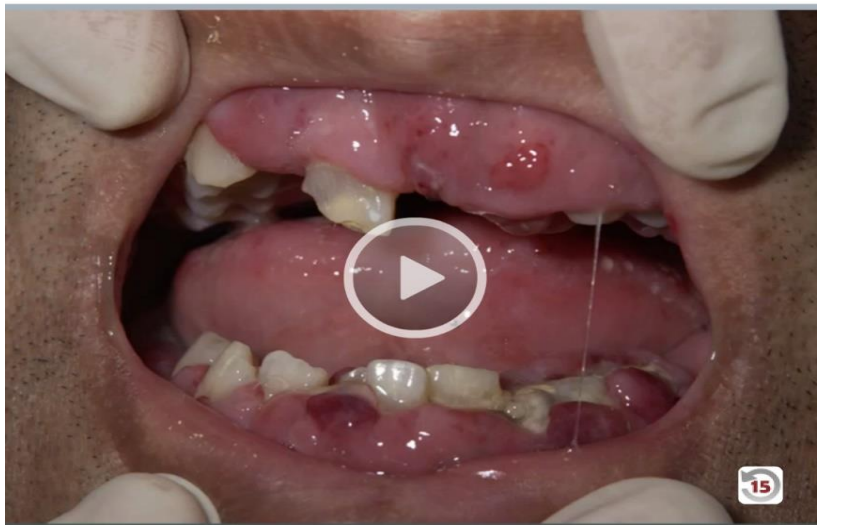


Acute promyelocytic leukemia (APL)

- Characterized by t(15;17). Retinoic acid receptor RAR) on 17 translocated to 15. RAR disruption blocks maturation and promyelocytes (blasts) accumulate
- High risk of DIC, Auer rods made of crystalized MPO (myeloperoxidase), can be released and trigger coagulation cascade
- treatment All-trans-retinoic acid (ATRA, vitamin A derivative) binds altered receptor and causes blasts to mature (and eventually die)

Acute monocytic leukemia

- Patient presents with involvement of the gums (swollen is common) b/c blasts infiltrate the gums
- Characterized by Lack MPO



Acute megakaryoblastic leukemia

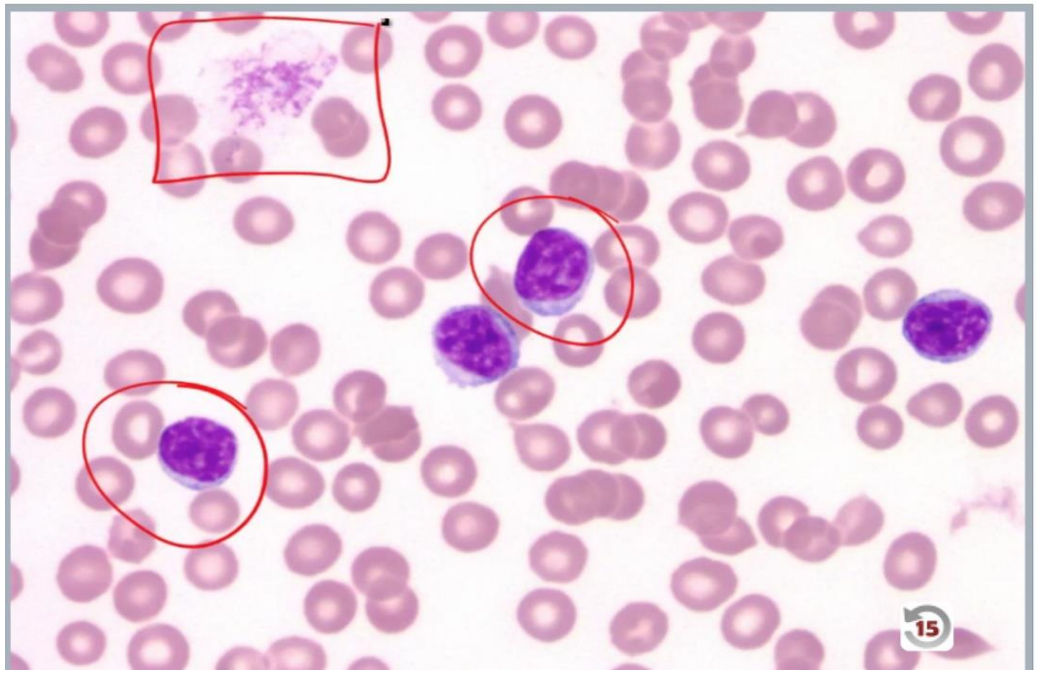
- Characterized by Lack MPO
- Associated with Down syndrome (before the age of 5)

Chronic leukemia

- Define :neoplastic proliferation of mature circulating lymphocytes

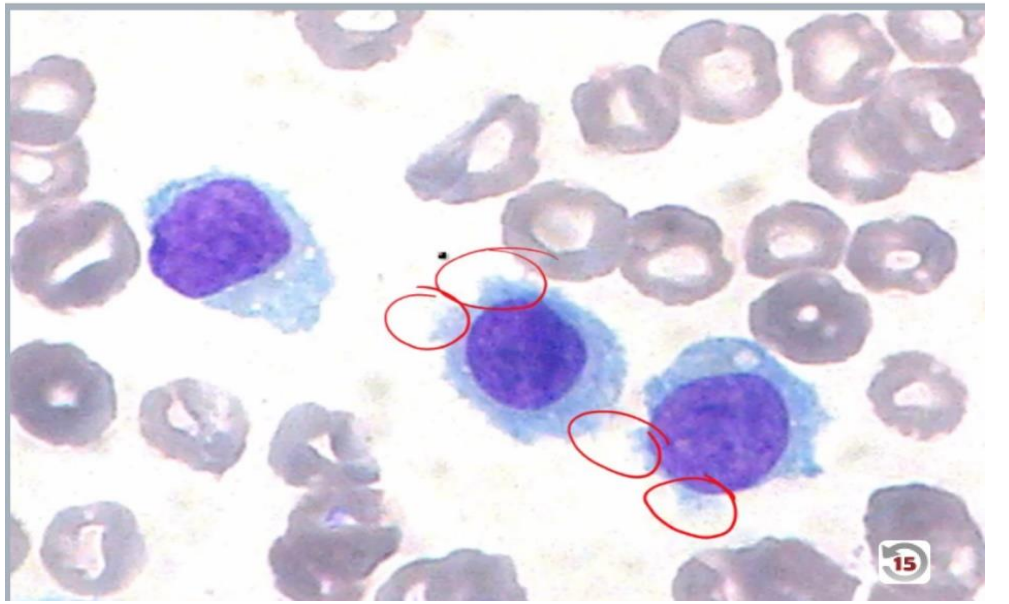
Chronic Lymphocytic leukemia (CLL)

- Define: neoplastic proliferation of naive B-cells
- CD expression: +CD5 & +CD20
- blood smear findings: smudge cell
- Clinical findings :lymphadenopathy ("small lymphocytic lymphoma")
- Complications: hypogammaglobulinemia (infection most common cause of death in CLL). Autoimmune hemolytic anemia (the Ab that are made are often recognized by self). Transformation to diffuse large B-cell lymphoma



Hairy cell leukemia

- Define: neoplastic proliferation of mature B cells.
- blood smear findings: characterized by hairy cytoplasmic processes
- Clinical findings: splenomegaly (red pulp; chronic leukemias usually due to white pulp expansion). Fibrotic bone marrow, resulting in dry tap with bone marrow aspiration. Lymphadenopathy usually absent
- Treatment: excellent response to cladribine (2-CDA), an adenosine deaminase inhibitor; adenosine accumulates to toxic levels in neoplastic B cells



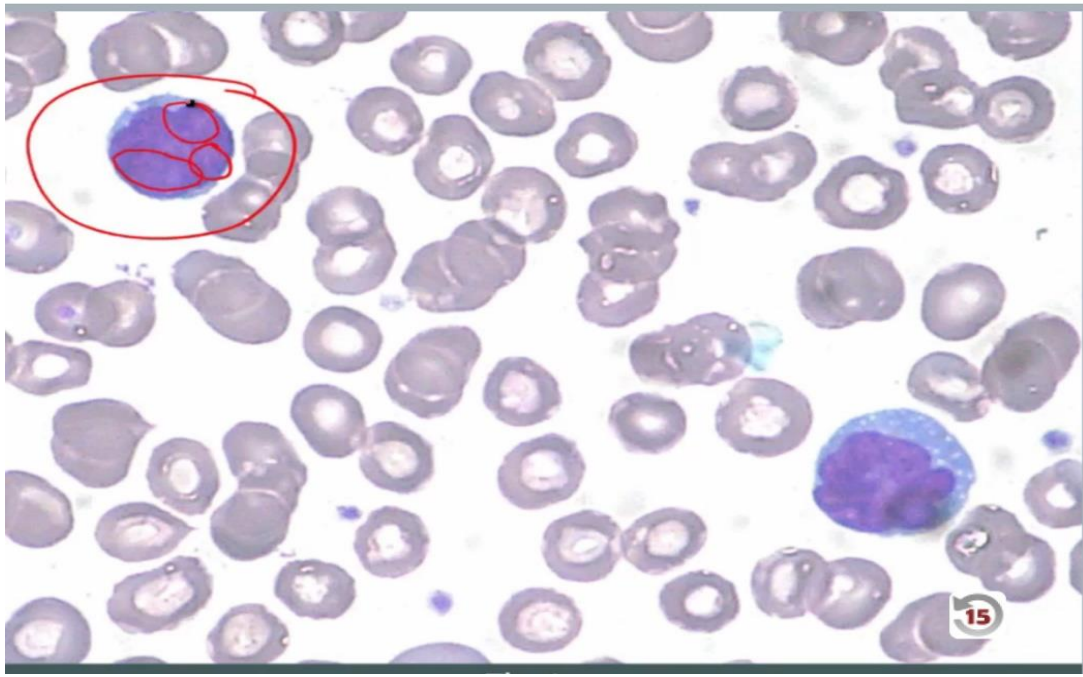
Adult T-cell leukemia/lymphoma (ATLL)

- Define neoplastic proliferation of mature +CD4 T-cells
- commonly seen in Japan and Caribbean. Associated with HTLV-1 (human T-cell leukemia virus 1)
- Clinical findings Rash, Generalized lymphadenopathy with hepatosplenomegaly, Lytic bone lesions with hypercalcemia

Mycosis Fungoides

- Define Neoplastic proliferation of mature +CD4 T-cells that infiltrate the skin, producing localized skin rash, plaques and nodules.
- Pautrier microabscesses aggregates of neoplastic cells in the epidermis

Sezary Syndrome cells spread to involve blood. Lymphocytes with cerebriform nuclei (Sezary cells) seen in smear



Myeloproliferative disorder (MPD

- Define Neoplastic proliferation of mature cells of myeloid lineage. Cells of ALL myeloid lineages are increased; classified based on dominant myeloid cell produced
- Complications increased risk of hyperuricemia & gout (uric acid in joints). Progression to marrow fibrosis. Transformation to acute leukemia
-

Chronic myeloid leukemia (CML)

- Define Neoplastic proliferation of mature myeloid cells, especially granulocytes and their precursors. BASOPHILS are characteristically increased!
- cause t(9;22) →Philadelphia chromosome. Generates BCR-ABL fusion protein with increased tyrosine kinase activity, which drives over proliferation of neoplastic cells
- treatment imatinib, blocks tyrosine kinase activity. Not a cure, but manages the disease. Resistance mutations can develop.
- Clinical findings splenomegaly is common; enlarging spleen suggests accelerated phase. Can transform to AML (2/3 of cases) or ALL (1/3) since mutation is in pluripotent stem cell
- CML distinguished from leukemoid reaction (reactive neutrophilic leukocytosis) 1) negative leukocyte alkaline phosphatase (LAP) stain (granulocytes in leukemoid reaction are LAP positive). 2) increased basophils (absent with leukemoid reaction). 3) t(9;22) which is absent in leukemoid reaction

Poycythemia vera (PV)

- Define Neoplastic proliferation of mature myeloid cells, especially RBCs. EPO independent erythropoiesis! Granulocytes and platelets are also increased
- mutation JAK2 Kinase. uncontrolled proliferation of blood cell types
- Clinical findings All are associated with hyper-viscosity of the blood. Blurry vision and headache. Increased risk of venous thrombosis. Flushed face due to congestion. Itching after bathing* (mast cells release histamine)
- treatment Phlebotomy (decrease RBCs). hydroxyurea secondly, JAK inhibitors
- PV distinguished from reactive polycythemia In PV EPO is decreased and Sao2 is normal. In reactive polycythemia due to high altitude or lung disease, Sao2 is low and EPO increased. In reactive polycythemia due to ectopic EPO production from renal cell carcinoma, EPO is high and Sao2 is normal
- bone marrow Panmyelosis. Panmyelosis is a form of myelofibrosis. It is part of the presentation in acute panmyelosis with myelofibrosis.

Essential thrombocythemia (ET)

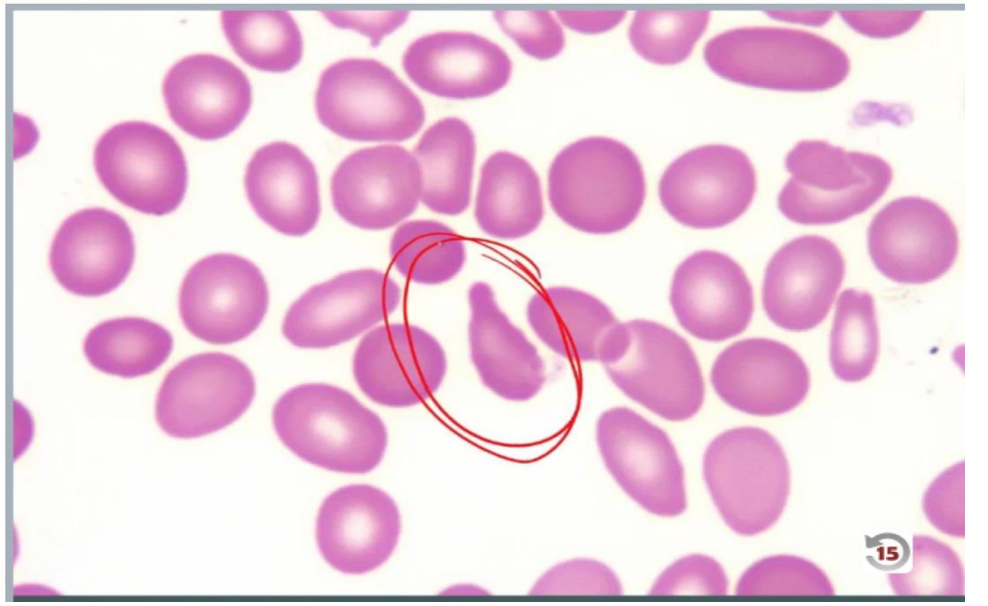
- Define Neoplastic proliferation of mature myeloid cells, especially platelets. RBCs and granulocytes are also increased
- mutation JAK2 kinase; uncontrolled proliferation of blood cell types
- symptoms related to increased risk of bleeding (ineffective platelets, though they're increased) and/or thrombosis. 1) rarely progresses to marrow fibrosis or acute leukemia. 2) no significant risk of hyperuricemia or gout (b/c platelets don't have nucleus, no purines to break down and therefore no uric acid overload)

Myelofibrosis

- Define Neoplastic proliferation of mature myeloid cells, especially megakaryocytes
- mutation JAK2 kinase (50% of cases); uncontrolled proliferation of blood cell types
- Mechanism Megakaryocytes produce excess platelet-derived growth factor (PDGF) causing marrow fibrosis
- Clinical findings Splenomegaly due to extramedullary hematopoiesis. Leukoerythroblastic smear (tear-drop RBCs, nucleated RBCs, and immature granulocytes). Increased risk of infection, thrombosis, and bleeding (marrow doesn't properly produce normal myeloid cells)
- blood smear findings Leukoerythroblastic smear (tear drop cells, RBCs, nucleated RBCs, and immature granulocytes)

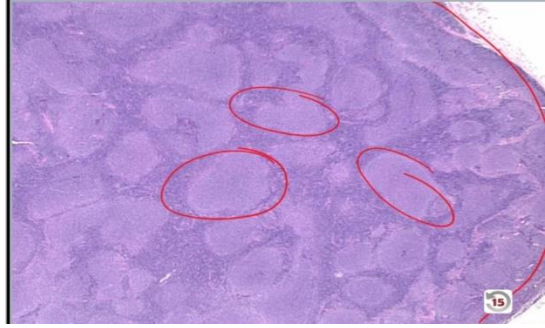
Myelofibrosis

- Leukoerythroblastic smear (tear drop cells, RBCs, nucleated RBCs, and immature granulocytes



Lymphoma

- Define Neoplastic proliferation of lymphoid cells that forms a mass. Mar arise in lymph node or in extranodal tissue



Follicular Lymphoma

- classify non-Hodgkin lymphoma
- Define Neoplastic small B cells (CD20+) that make follicle-like nodules.
- presents as painless lymphadenopathy, in late adulthood.
- Translocation t(14;18)BCL2 on chromosome 18 translocates to Ig heavy chain locus on 14. Results in over expression of BCL2, which inhibits apoptosis {BCL2 sits on BAX on mitochondria membrane and regulates cytochrome c escape into cytosol which signals apoptosis}
- treatment Reserved for patient who are symptomatic. Low dose chemotherapy or rituximab (anti-CD20 antibody)
- complication progression to diffuse large B-cell lymphoma. Presents as an enlarging lymph node
- Distinguished from follicular hyperplasia by In follicular lymphoma you see 1) Disruption of normal lymph node architecture (hyperplasia you see follicles only in cortex, not in medulla). 2) Lack of tinigble body macrophages in germinal center in (which indicates macrophages consuming dead, apoptotic B cells). 3) Expression of BCL2 in follicles (normal follicle there is not expression of BCL2 b/c apoptosis is desired). 4) Monoclonality

Mantle cell lymphoma

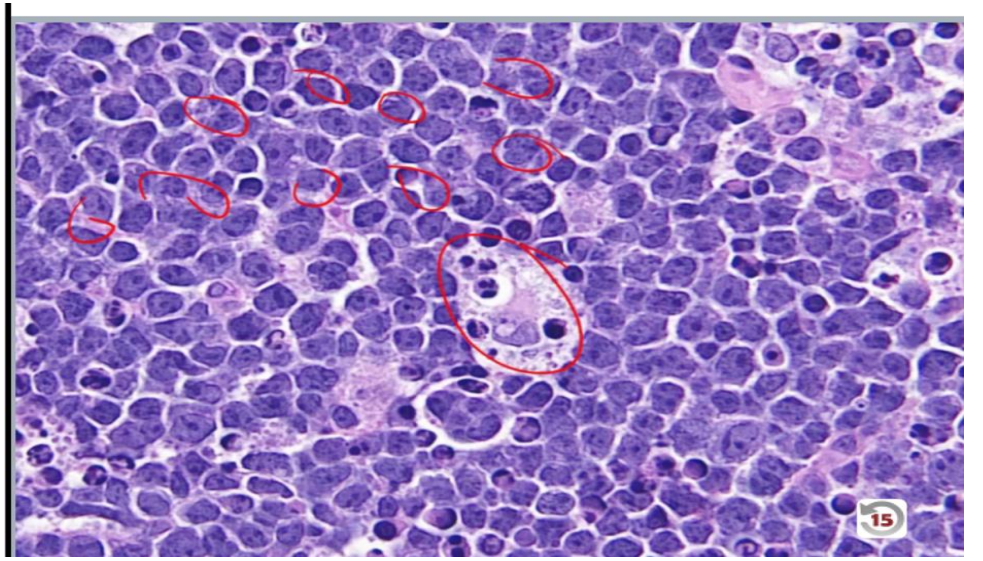
- Define Neoplastic proliferation of small B cells (CD20+) that expand the mantle zone (region immediately adjacent to follicle).
- presents as Painless lymphadenopathy in late adulthood
- Translocation t(11;14). Cyclin D1 gene on 11 translocates to Ig heavy chain locus on 14. Overexpression of cyclin D1 promotes G1/S transition in cell cycle, facilitating neoplastic proliferation

Marginal Zone lymphoma

- Define Neoplasatic small B-cells (CD20+) that expand marginal zone (region outside mantle, mantle is adjacent to follicle)
- Associated with chronic inflammatory states
- MALToma maringal zone lymphoma in mucosal sites (i.e. stomach)

Burkitt lymphoma

- Define neoplastic intermediate-sized B cells (CD20+)
- presents as extranodal mass in child or young adult
- African form usually involves jaw
- sporadic form usually involved abdomen
- Translocation t(8;14). Results in translocation of c-myc to Ig heavy chain locus on 14. Overexpression of c-myc oncogene promotes cell growth
- blood smear findings starry-sky appearance (high mitotic index; small blue cells=tumor. white=macrophages that die due to rapid growth)



Diffuse large B-cell lymphoma (DLBCL)

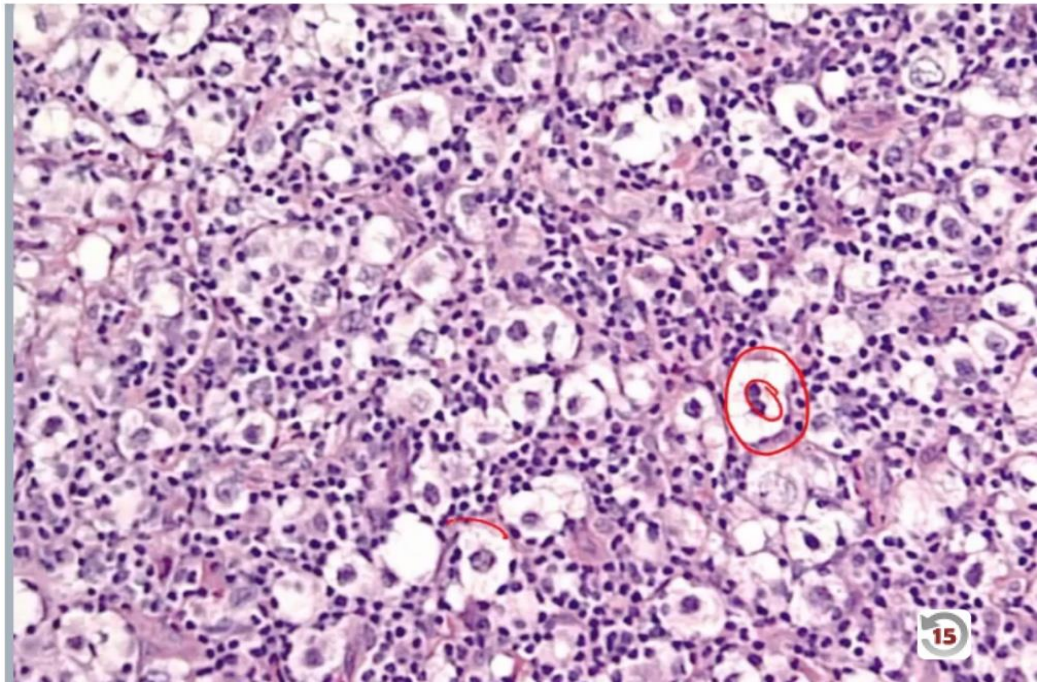
- Define Neoplastic large B-cells (CD20+) that grow diffusely in sheets. Most common form of non-Hodgkin lymphoma. Clinically aggressive
- presents as enlarging lymph node or an extranodal mass

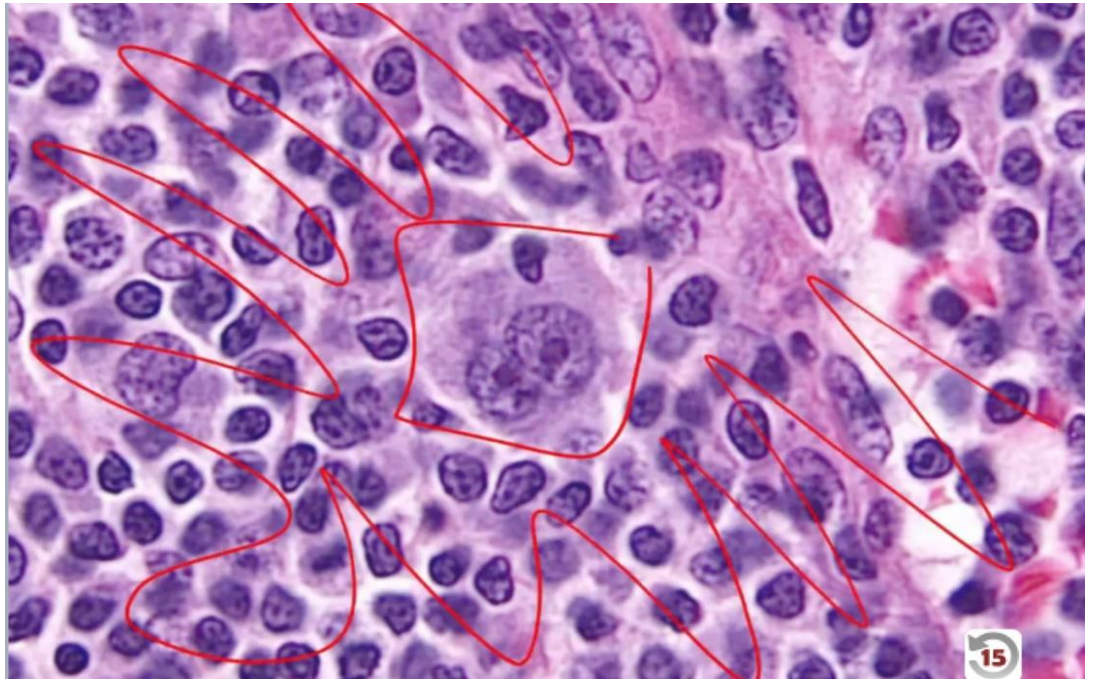
Hodgkin lymphoma

- Define Disease spreads locally to contiguous lymph nodes. neoplastic proliferation of Reed-Sternberg (RS) cells, which are large B cells with multi-lobed nuclei and prominent nucleoli ('owl-eyed' nuclei). CD15+ & CD30+ (note, no CD20+ even though it's a B cell)
- Reed-sternbery cell cytokines occasionally results in B symptoms (fevers, chills, night sweats). Attract reactive lymphocytes, plasma cells, macrophages, and eosinophils. May lead to fibrosis.
- Reactive inflammatory cells make up bulk of the tumor and form the basis for classification

- Nodular sclerosis enlarging cervical or mediastinal lymph node in young adult female. lymph node divided by bands of sclerosis. RS cells present in lake-like spaces called lacunar cells (see image)
- Important considerations regarding other subtypes lymphocyte-rich has the best prognosis. Mixed cellularity is associated with abundant eosinophils (IL-5). Lymphocyte-depleted has the worst prognosis (seen in elderly and HIV+ individuals)
- Age Distribution bimodal 15-45 and >50
- Clinical findings Painless enlargement of lymph nodes, usually in neck. Typical B symptoms=night sweats, fever, itching

owl-eyed

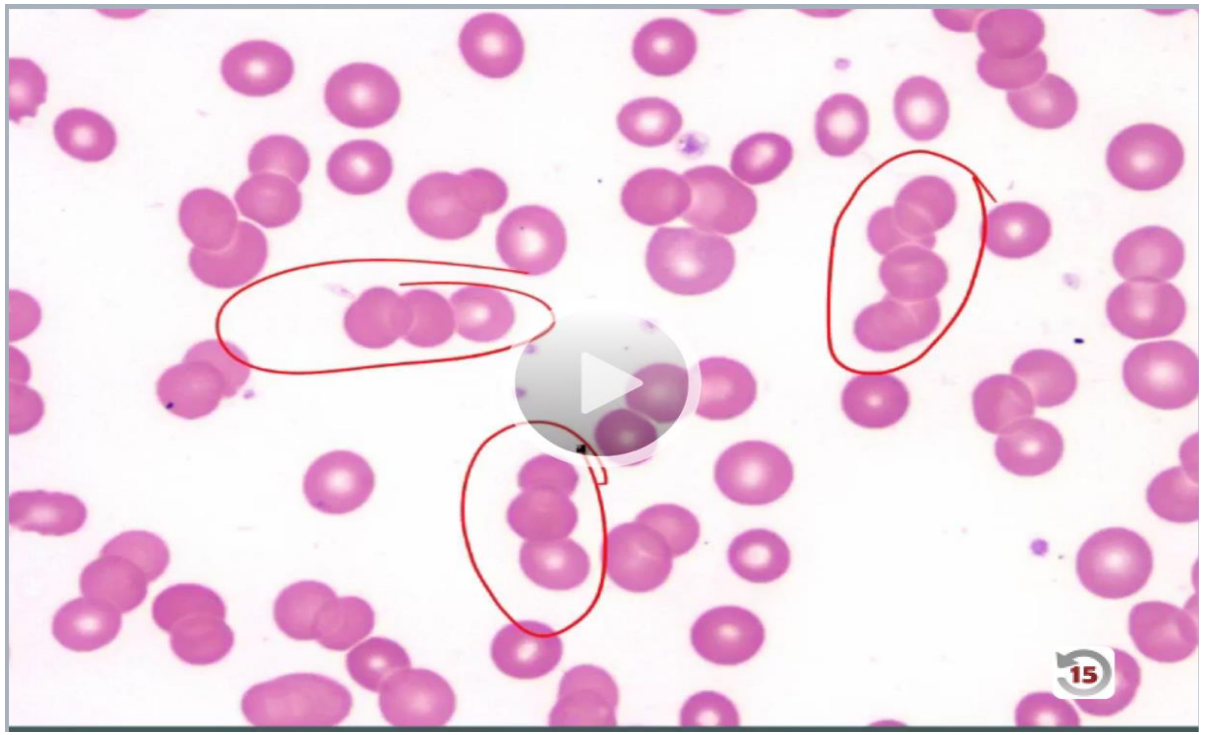




Multiple myeloma

- Define malignant proliferation of plasma cells in bone marrow. most common primary malignancy of bone (metastatic cancer is most common malignant lesion of bone overall)
- serum findings IL-6 high in serum (stimulates plasma cell growth and immunoglobulin production). Elevated serum protein ("M-spike," increased monoclonal IgG or IgA)
- Clinical findings bone pain with hypercalcemia (neoplastic plasma cells activate RANK receptor on osteoclasts leading to bone destruction.). Lytic lesions seen on XRAY (esp. in skull and vertebrae). Increased risk for fracture
- most common cause of death risk for infection, monoclonal antibody lacks antigenic diversity
- blood smear findings Rouleaux formation of RBCs (clumping together) due to increased serum protein which decreases charge b/w RBCs
- Primary al amyloidosis Free light chains (over produced in multiple myeloma) circulate in serum and deposit in tissues
- Bence-Jones proteins free light chain is excreted in urine as Bence-Jones proteins. Deposition in kidney tubules leads to risk for renal failure (myeloma kidney)

- blood smear Rouleaux formation of RBCs (clumping together) due to increased serum protein which decreases charge b/w RBCs



Langerhans cell histiocytosis

- Langerhans cells specialized dendritic cell found predominantly in skin. Derived from bone marrow monocytes. Present antigen to naive T-cells
- Define Neoplastic proliferation of Langerhans cells.
- CD CD1a+ and S100+
- Birbeck granules tennis racket granules
- Letterer-Siwe disease malignant proliferation of Lanherhans cells. Classic presentation is skin rash and cystic skeletal defects in an infant (< 2yo). Multiple organs may be involved; rapidly fatal
- Eosinophilic Granuloma Benign proliferation of Langerhans cells in bone. Classic presentation is pathologic fracture in an adolescent; skin not involved. Biopsy shows Langerhans cells with mixed inflammatory cells, including numerous eosinophils
- Hand-Schuller-Christian disease malignant proliferation of Lahngerhans cells. Classic presentation is scalp rash, lytic skull defects, diabetes insipidus, and exophthalmos in child.

tennis racket granules

Anemia

Q1: A 29 YO female has become increasingly lethargic for the past 6 months. She complains from SOB, fatigue & tachycardia. Her peripheral blood smear is shown here.

What is the Dx?

Iron deficiency anemia

Mention other 2 DDx?

Sideroblastic anemia/Thalassemia

Investigation you order and the findings that go with your Dx ?

CBC:

MCV<80/MCHC<32/RDW elevated/Reticulocyte low

Iron study:

Serum ferritin and serum iron low

TIBC and transferrin receptors increase

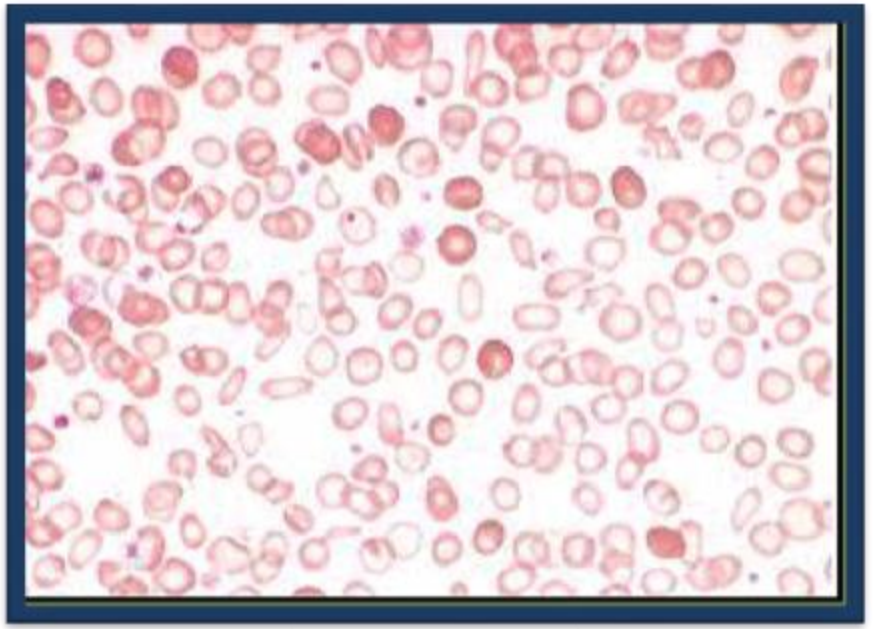
But transferrin saturation low

What is single best test to confirm your Dx?

Serum ferritin (less than 10ng/ml)

How you can manage the pt.?

Ferrous sulfate 200mg 3x daily for 3-6months



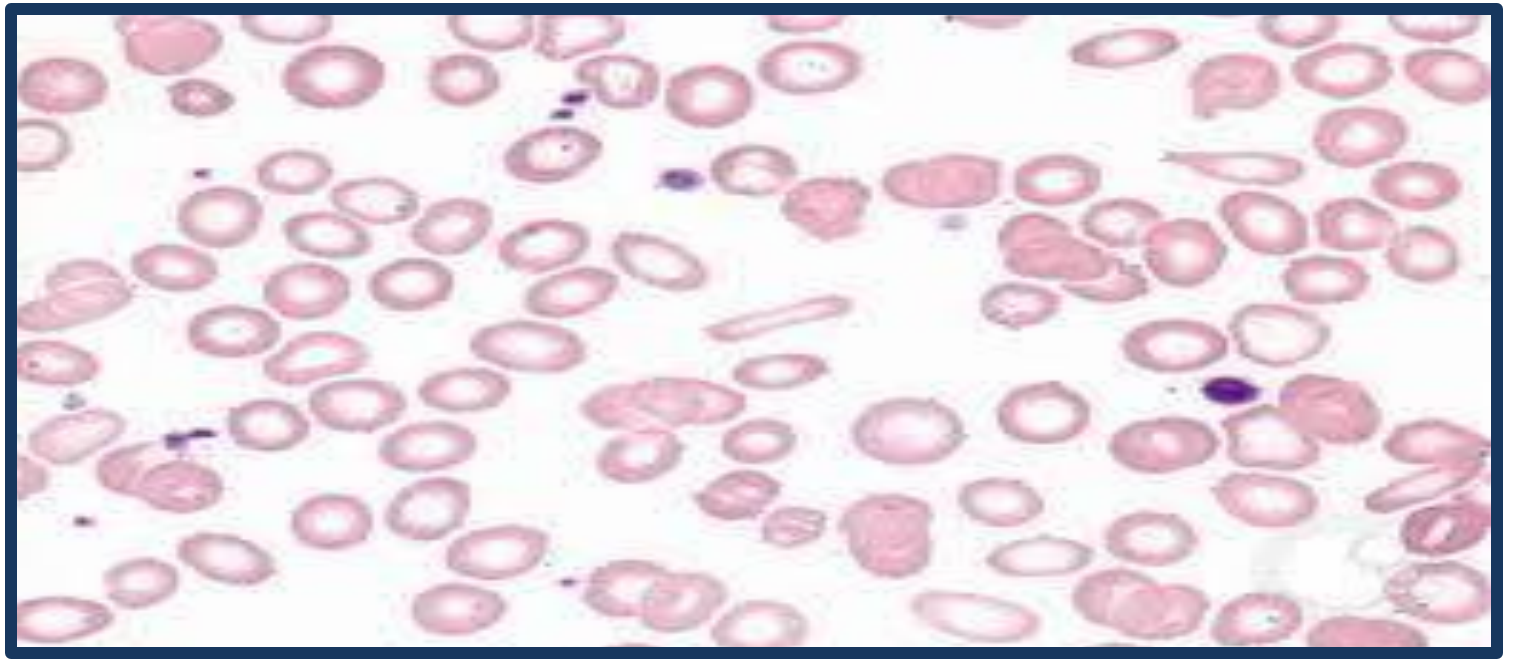
Q2 : this blood film is taken from a female pt who Had a history of heavy menses, what is the cause ?

iron deficiency anemia

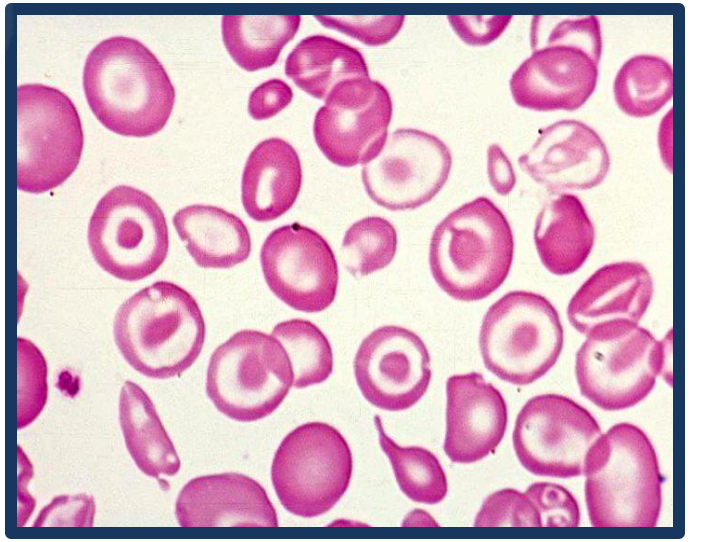
P.S. *with replacement of iron,brisk increase in reticulocyte occur within 2 week of the Rx.

*HB raised around 1g/dl every 7-10 days

*If your pt. not tolerate to oral therapy ,u can give her parenteral iron but it need monitoring because risk of anaphylaxis



Q3: A 22 year old man with an anemia with high ferritin levels and history of blood transfusion has this blood smear.



Your diagnosis ?

Thalassemia

What the findings on blood film?

Target cell

How you can confirm your Dx ?

HB electrophoresis

If beta thalassemia :increase level of HBF and HBA2

If alpha thalassemia :normal level of HBF and HBA2

Mention three findings u can found in examination?

Beta thalassemia(Hepatosplenomegly ,Jundice and bony deformities)

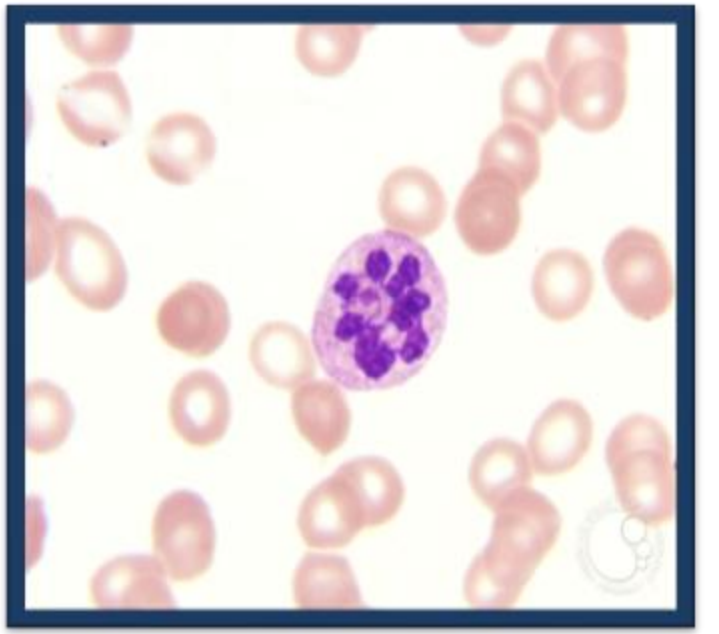
How you can manage this pt ?

Blood transfusion ,oral deferasirox

May do splenectomy

Small number can treated with BMT

Q4: This blood film is for a patient with vitiligo(pt with neurological symptom =vit. B 12).



A-What is the blood film finding?

Hypersegmented Neutrophil

B-What is the diagnosis

Vitamin b12 deficiency anemia

Mention 3 causes ?

Pernicious anemia (mcc)

Decrease dietary intake

Malabsorbtion …regional enteritis ,blind loop $

Tapeworm (diphyllobothrium latum 'rare')

Most specific test to confirm your Dx?

Blood level of b12

If it pernicious anemia , how you can confirm your diagnosis?

Look for Antibody to intrinsic factor

Other test but rarely use now schilling test

How you can manage your pt?

Replace vit. B12 lifelong …1000Mg/IM 6doses ,3 days apart

Then every 3 months one dose

Q5:What's you diagnosis?

Megaloblastic anemia

What blood film show?

Hypersegmented neutrophil

Oval macrocytes

If it folate deficiency anemia how you can confirm your Dx?

Folate RBC's level

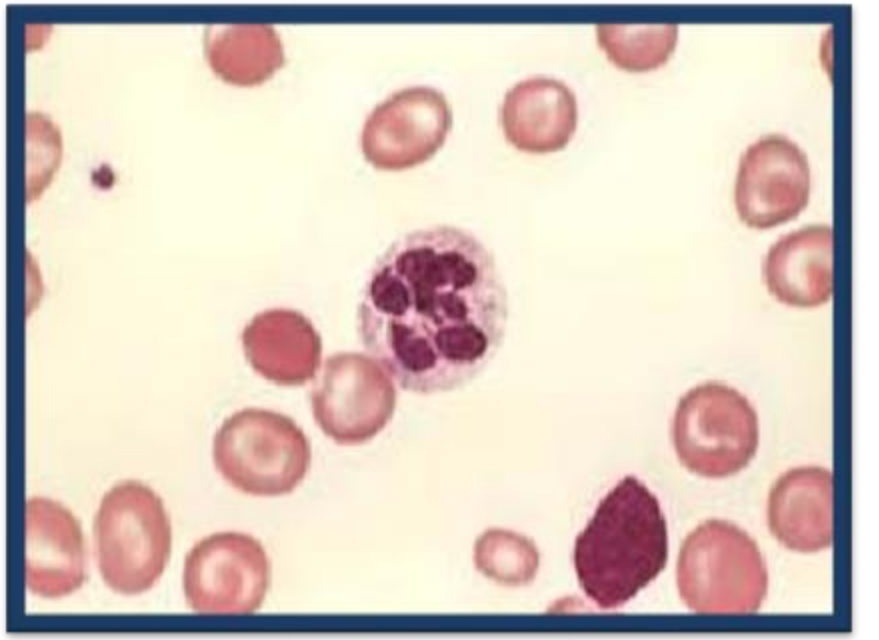
Mention 3 drugs that may cause folate deficiency anemia?

Phenytoin ,MTX ,OCP

How you can manage the pt.?

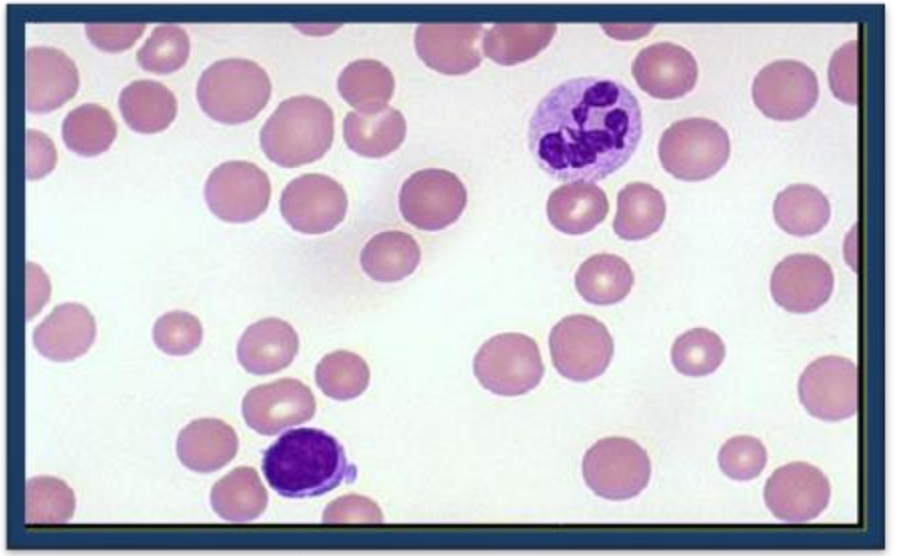
5mg daily for 3 w

Maintenance 5mg once per week



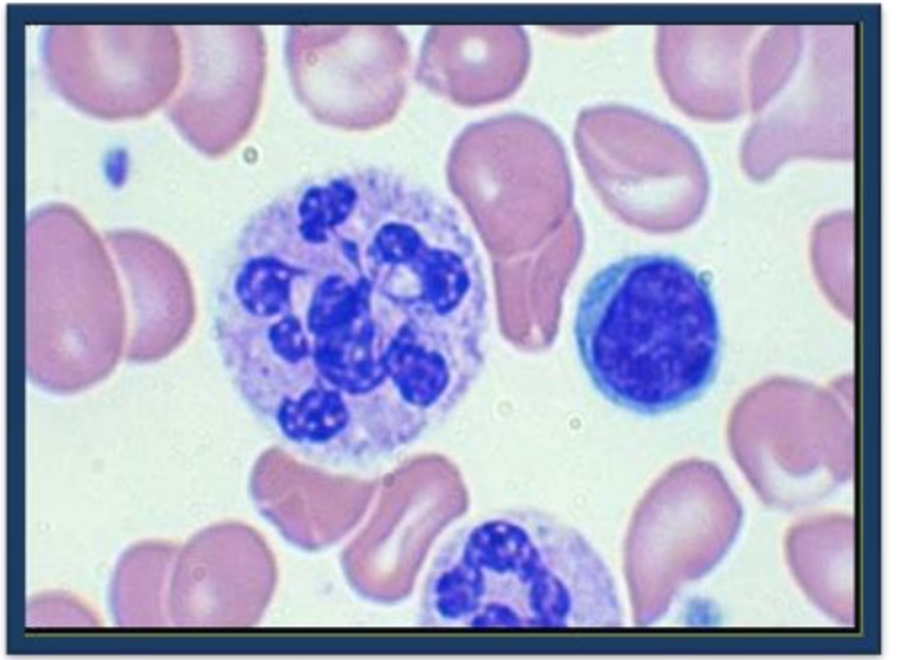
Q6: 32 YO female patient presented with pallor, lower limb numbness, & Vitiligo , what is the diagnostic test?

Serum B12 level

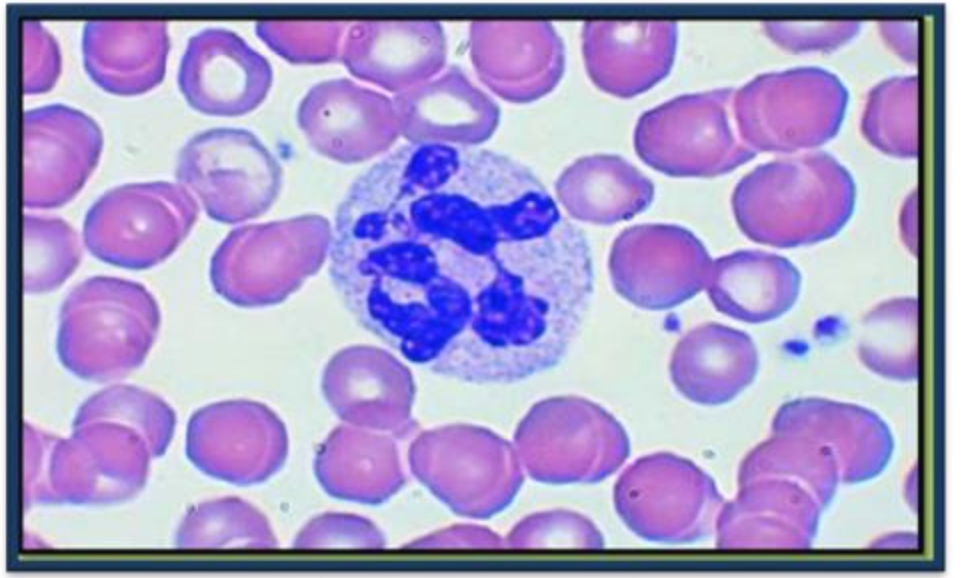


Q7: This patient suffered from parasthesia and weakness in her lower limbs, what is your diagnosis?

Megaloblastic anemia (Due vit.B12 deficiency).



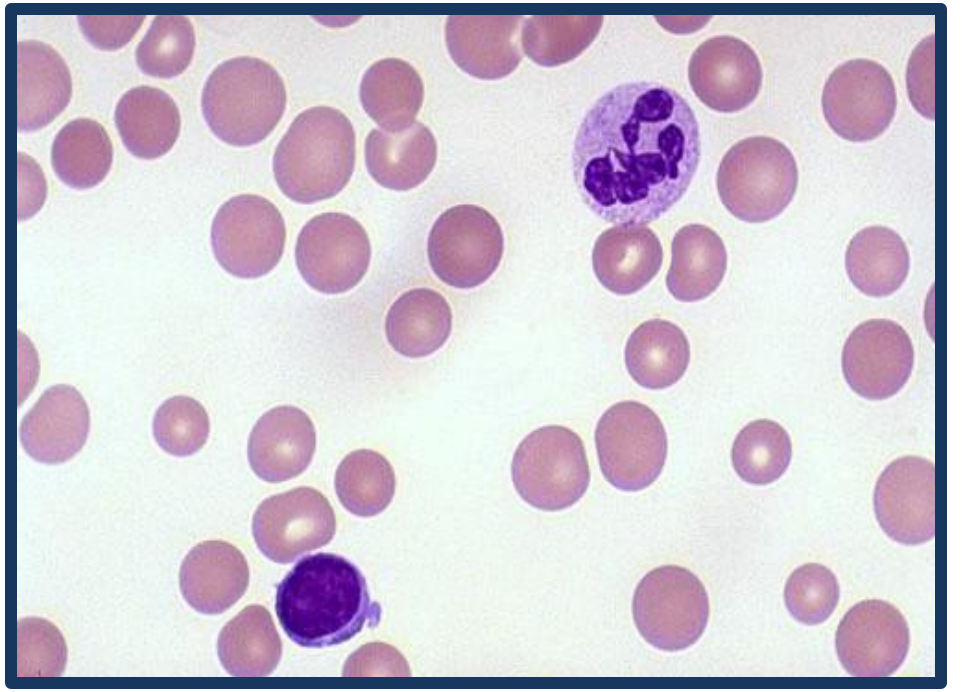
Q8: This blood film is for a pt who has terminal ileum resection in his past Hx., & now he presented with dyspnea & fatigue.
What's your Dx.?
Megaloblastic Anemia due to Vitamin B12 deficiency.
Give one abnormal finding in this blood film.
Hyper segmented neutrophils.



Q9: this is a blood film for a known case of crohn's disease
Who is presented with dizziness ...

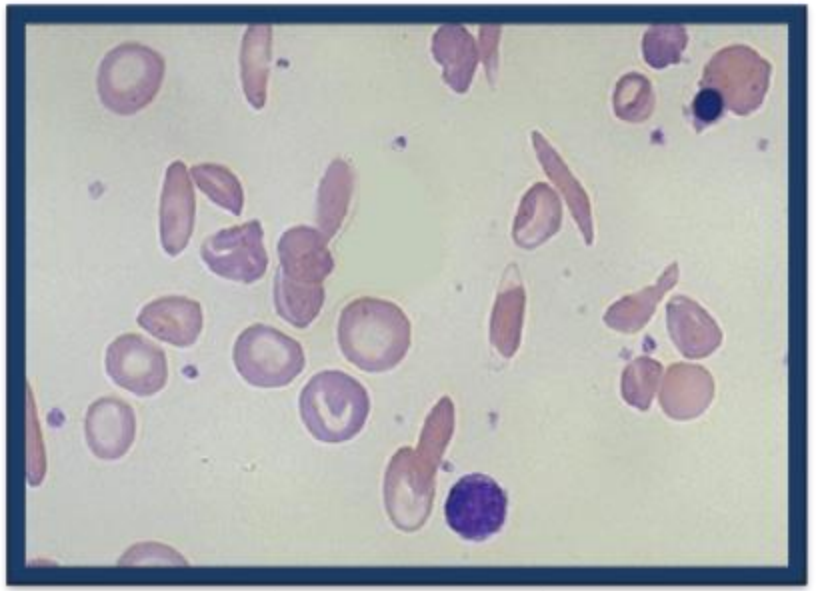
1)What is the abnormality that you see in the blood film ?
Hyper segmented neutrophil, OVAL macrocytes

2) what is the possible cause ?
Vit b12 def. Due to malabsorption



Q10:This patient is anemic, and have abdominal &lower limb pain. What's your diagnosis?

Sickle Cell Anemia



What is the most specific test to confirm the Dx?

HB electrophoresis (HBS no HBA2 ,2-20%HBF)

What other investigations?

Bcz it a hemolytic disease LDH , unconjugated bilirubin and reticulocyte will increase

leukocytois

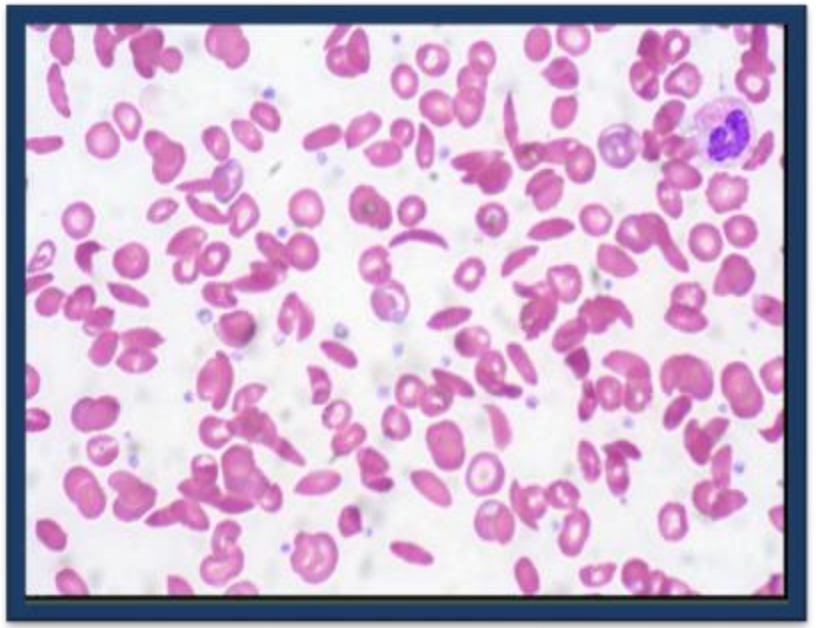
On Urine analysis microscopic hematuria

Management of acute sickle cell pain crisis?

Oxygenation ,hydration ,analgesic(opiate)

For all sickle cell pt :Abx, and vaccination against hemophilius and pneumococcal influenza

Q11: 21 YO male patient presented with dark urine & mild jaundice.
What is the diagnosis?



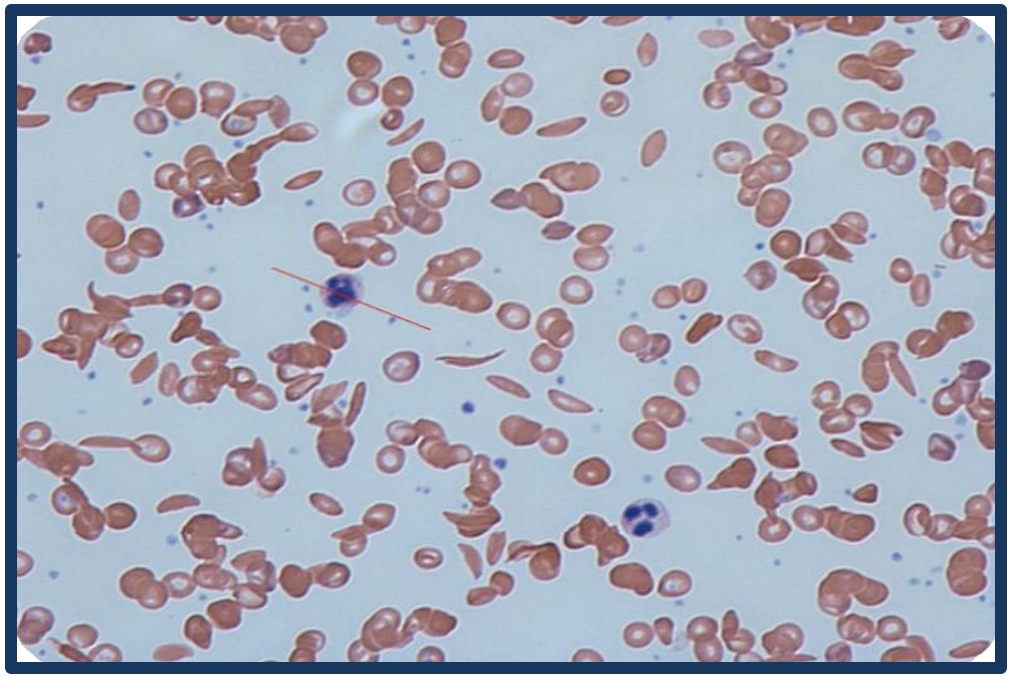
Sickle cell anemia

***clinical presentation of SCA

1. Painful vasooclusive crisis :severe bone pain (femur ,pelvis ,heumerus ,ribs) associated fever ,sweating ,tachycardia
2. Acute chest $:mcc of death in adult with SCA
3. Silent stroke
4. Sequestration crisis(thrombosis of venous outflow):massive splenomegaly ,priapism(prostatic plexus vein)
5. Aplastic crisis after parovirus b19 infection

Q12:A 25 yr old male patient complains of dyspnea on mild exertion, recurrent jaundice, back pain. He has history of chronic anemia and needs blood transfusion every ()yrs with a family history of chronic anemia, what is your diagnosis?

Sickle Cell Anemia



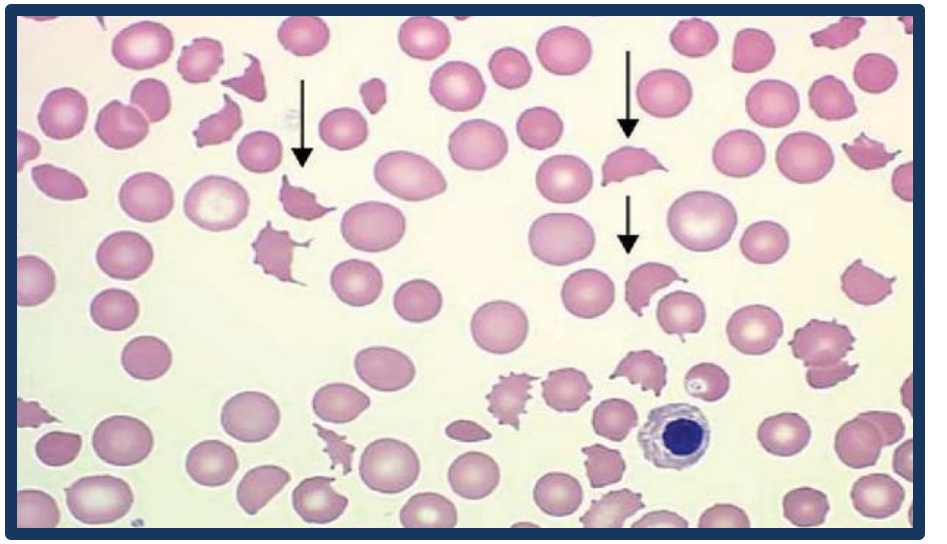
Q13: 45 YO pt complains of progressive fatigue, exertional dyspnea, jaundice, & with following picture.what the most likely Dx?

Autoimmune Hemolytic Anemia=warm or cold agglutinin

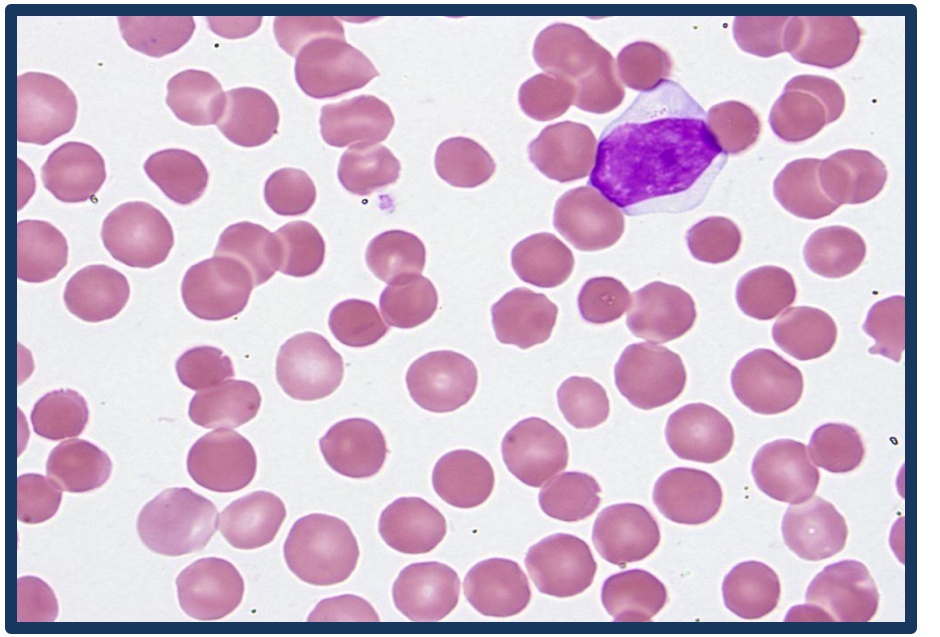
(+ve direct coombs test)

Q14: A 22 year old female with a prosthetic valve presented with this blood film. What is your diagnosis?

RBC's fragments: Microangiopathic Hemolytic Anemia

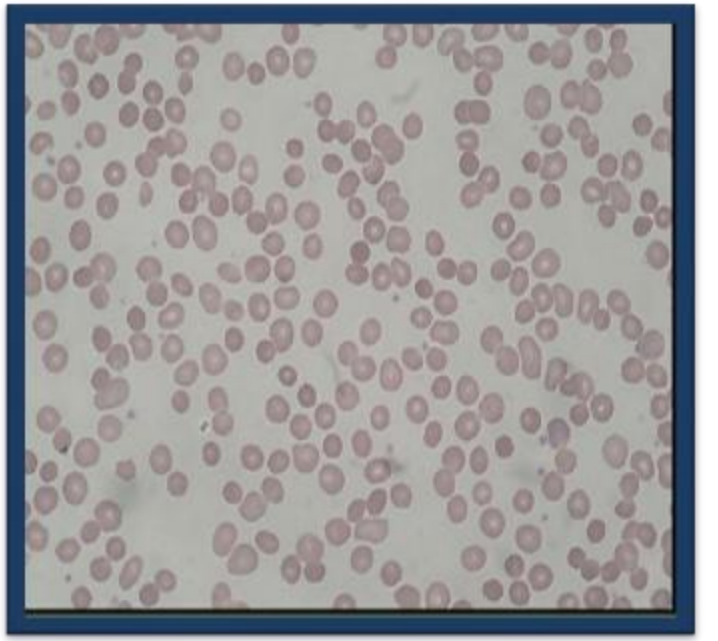


Q15: A15 year old male presented with pallor & fatigue. On examination he had splenomegaly. He has two siblings with similar complaints.
What is your diagnosis?
Hereditary spherocytosis
What if the diagnostic test?
Osmotic Fragility Test



Q16: Pt presented with anemia & splenomegaly with family Hx of Anemia, what is the Dx?

Hereditary spherocytosis.



What blood film show?

Spherocyte

Findings on investigation?

Dec. MCV

inc. MCHC

Inc. reticulocyte

Inc. unconjugated bilirubin

inc. LDH

Coombs test :negative

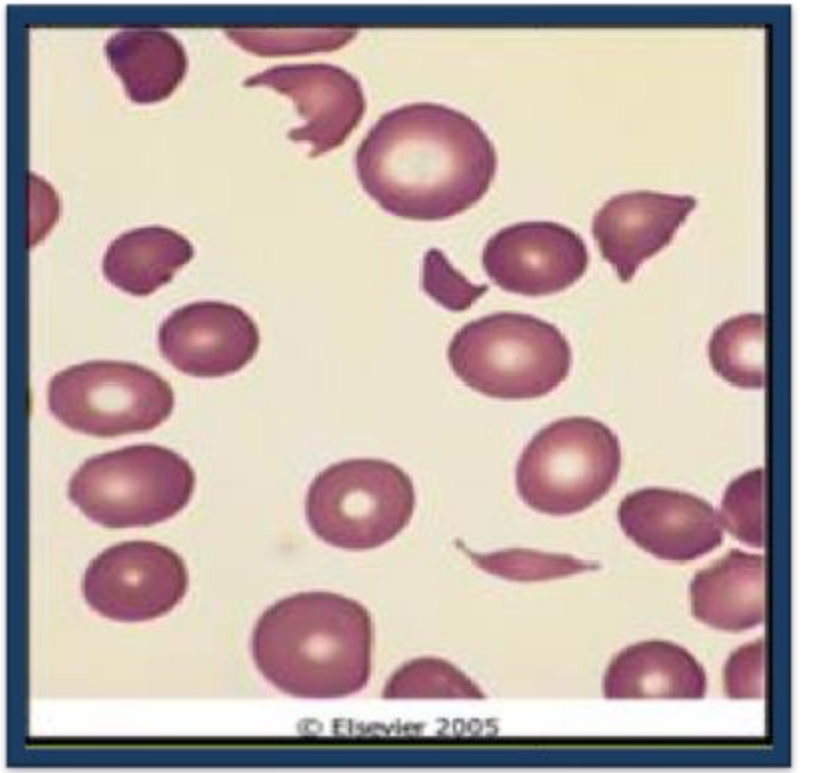
Osmotic fragility test :inc. lysis in hypotonic solution

Management?

Folate replacement , splenectomy(resolve symptom and jaundice but spherocyte will remain)

Q17: What's the hematological abnormality in this blood film?

G6PD deficiency(x-linked ,decrease NADPH which protect against oxidative stress)



Most common type of stress ?

infection

Drugs that may cause oxidative stress?

Sulfa drug ,dapsone ,quinidine

Blood film show ?

bite cells

Definitive test :G6PD level

Treatment ?hydration and transfusion if severe hemolysis

Endocrine system

REFERENCES :

- DAVIDSON
- STEP-UP

DONE BY :

وسيم التميمي & عمر المعاني

آلاء الجبالي & تالا الجرادات

قيس عويس & نزار حداد

محمد رياض & يوسف الخطيب

عبدالله ابوخيط & أسامة الربضي

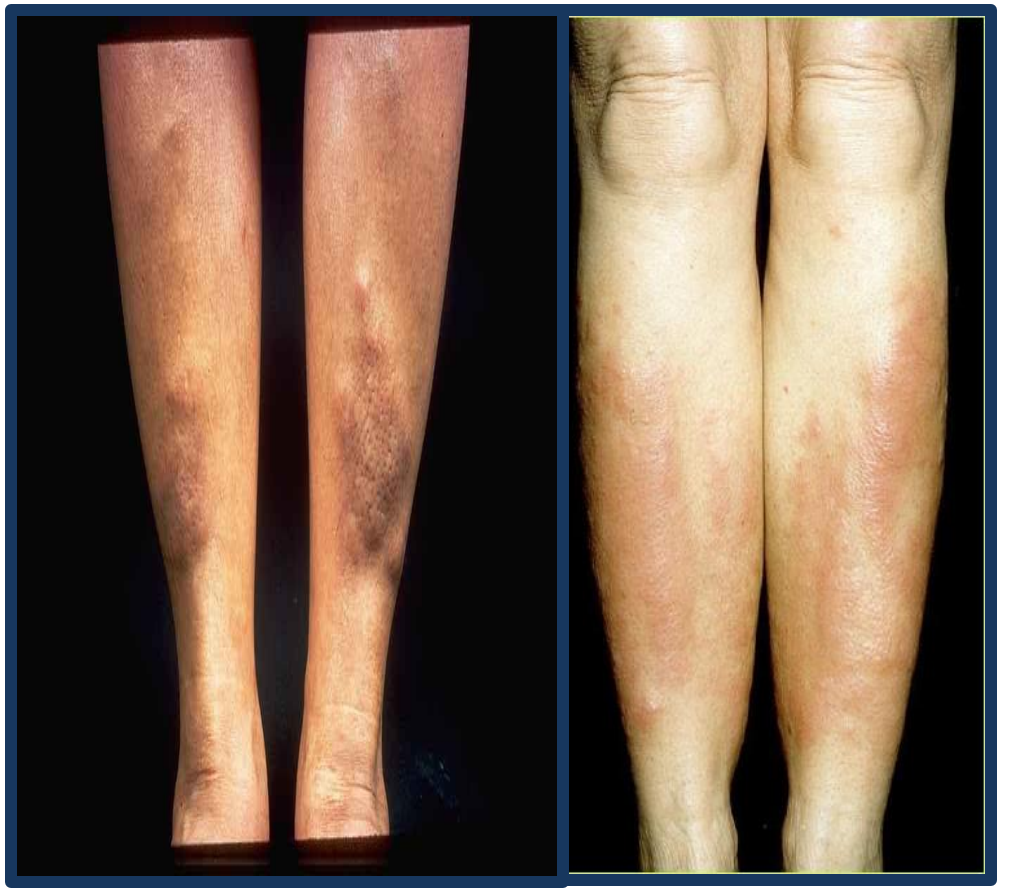
Hypo and hyperthyroidism

Q: This patient had thyrotoxicosis what is this lesion or the finding on her limbs?

Bilateral pretibial myxedema

What is your diagnosis ?

Graves disease



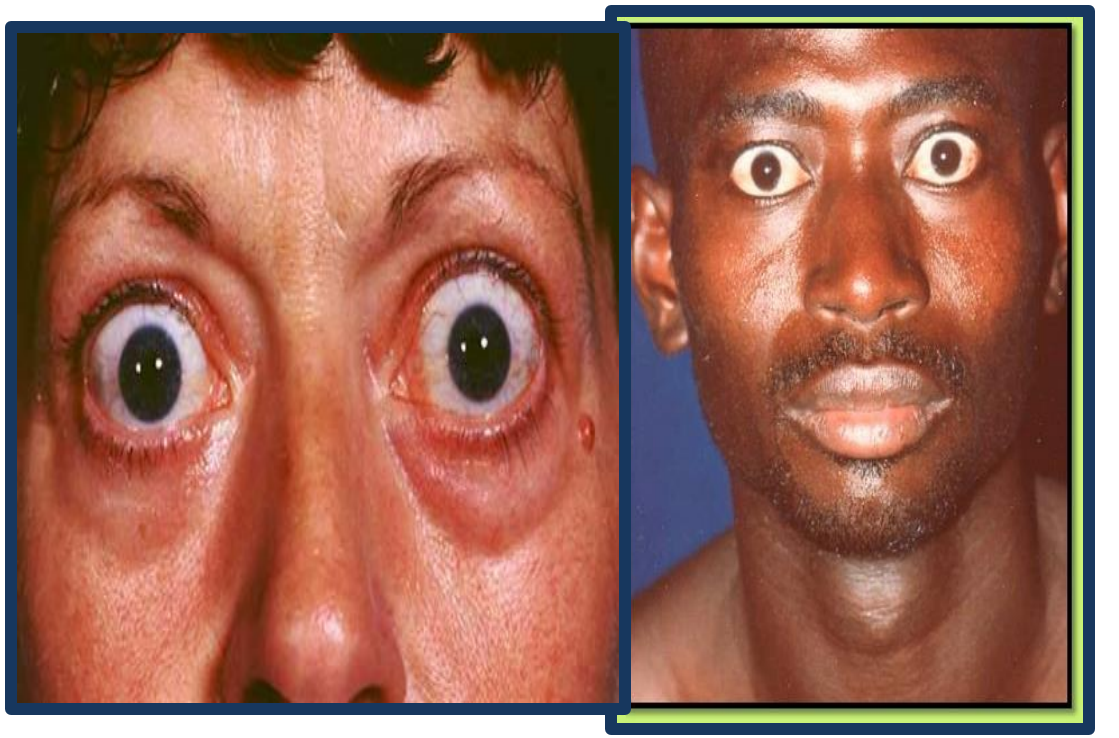
Q: A pt presented with palpitation & tachycardia, sweating & heat intolerance.

What Is the diagnostic test?

Thyroid function tests

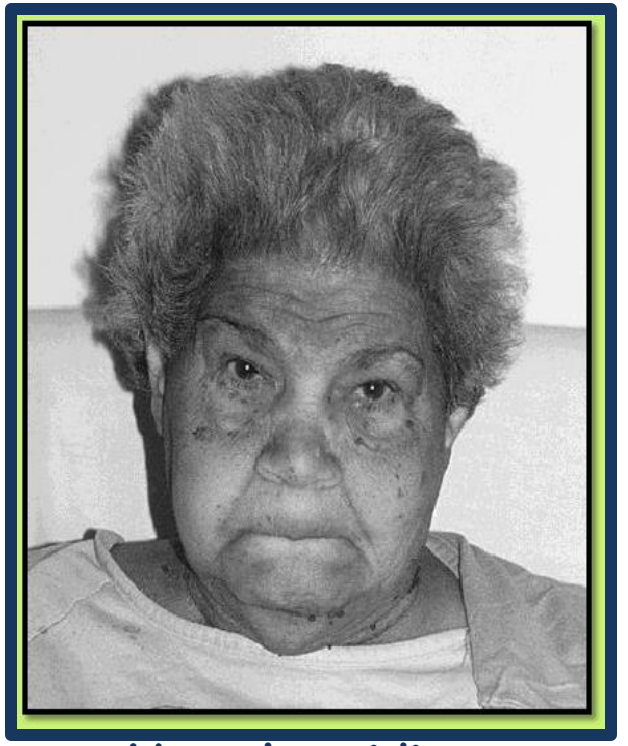
What is the main feature in this figure?

Exophthalmus.



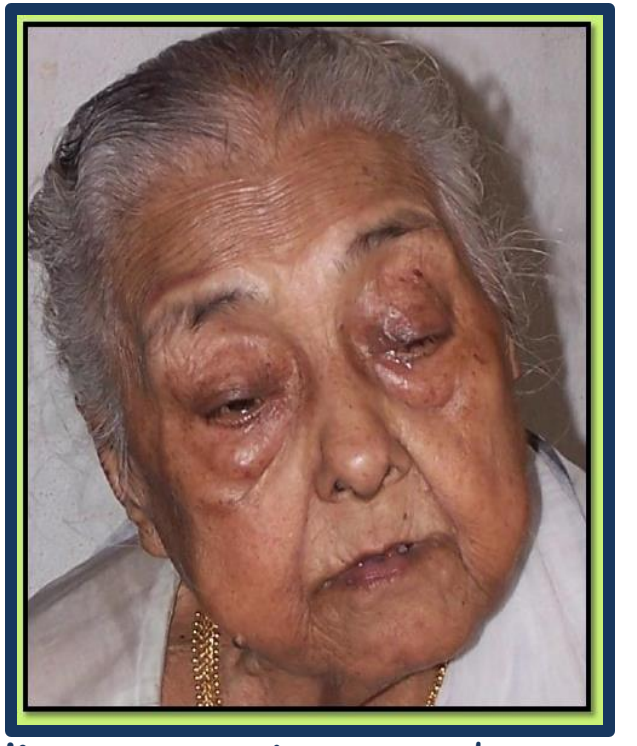
Q: This patient came with constipation & wt gain, mention 2 cardiac complications for it.

1. Hypertension.
2.Cardiomegaly.
3.Bradycardia.



Q: Patient has hair loss and weight gain. What is the test you want to do?

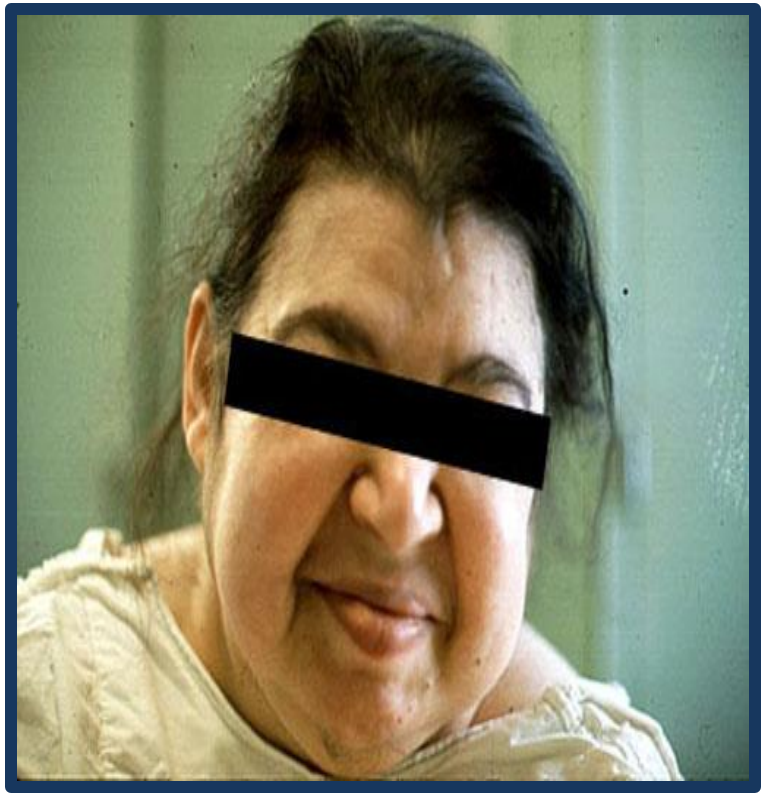
Thyroid Function test.
خاصةTSH
High TSH is the most sensitive indicator for hypothyroidism



Hypothyroidism can result in decreased **cardiac** output, increased systemic vascular resistance, decreased arterial compliance, **and** atherosclerosis. Impaired **cardiac** muscle relaxation, decreased heart rate, **and** decreased stroke volume contribute to heart failure in **hypothyroidism**.

Q: patient with fatigue , cold intolerance, what is the most diagnostic lab investigation ?

Thyroid function test



Q: patient with fatigue , hair loss, her blood pressure 130/80 , HR 12 what is the most diagnostic lab investigation ?

Thyroid function test .

Q: What is your spot diagnosis?

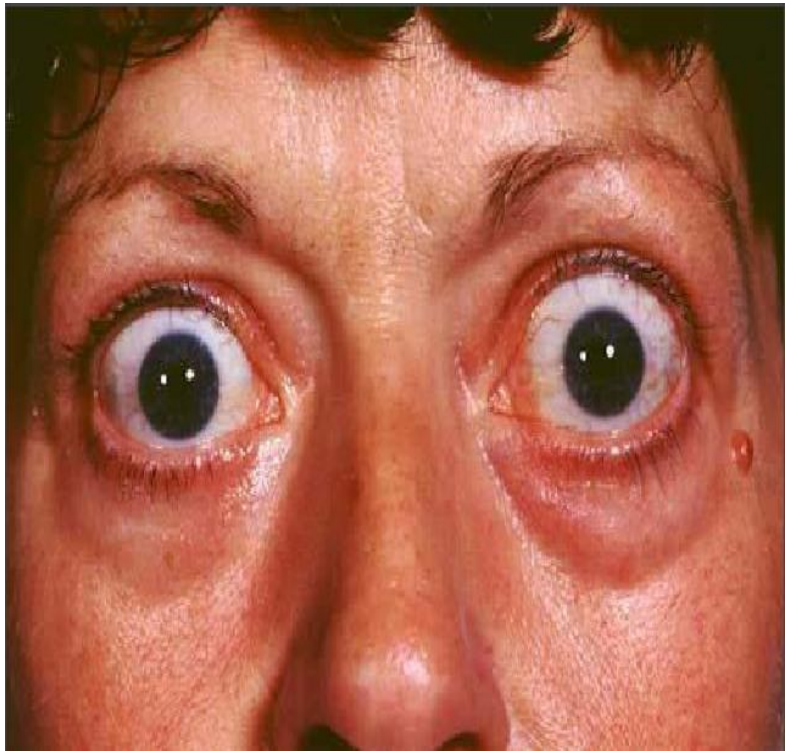
Graves' disease

Mention two lab investigations to support it.

Thyroid function test

Radioactive iodine uptake

Anti-TSH (thyrotropin)receptor antibodies



- Mention 2 abnormal physical signs
- What is the diagnosis

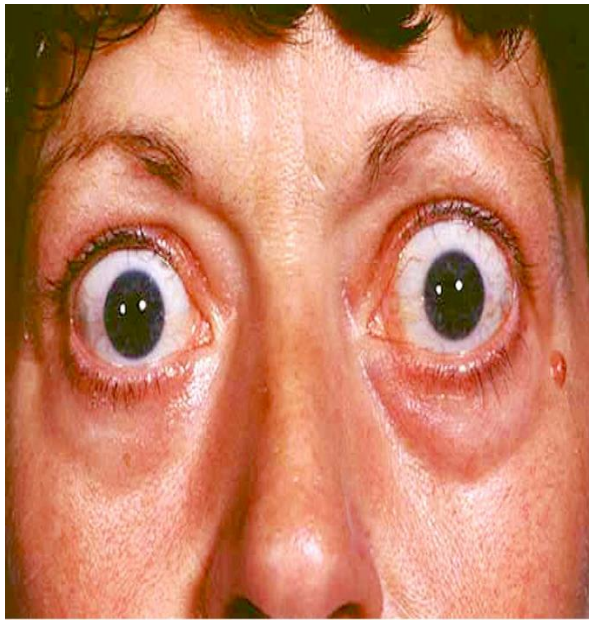
1- Neck Mass

2- Exophthalmos

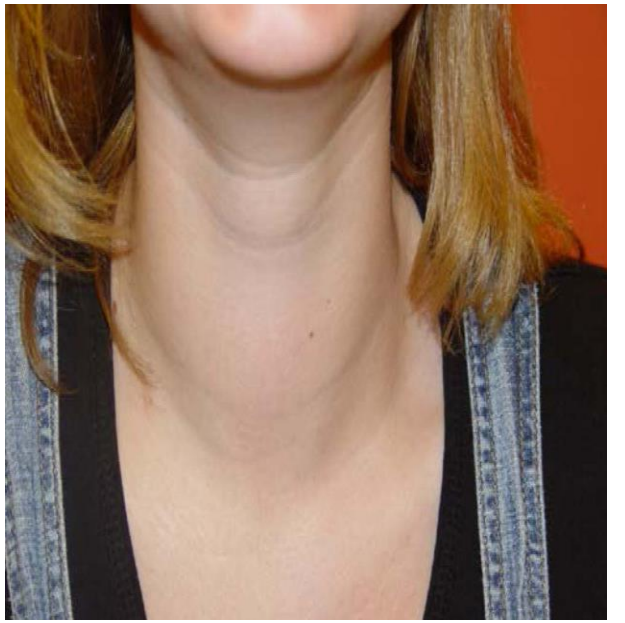
Diagnosis: Hyperthyroidism

Graves disease

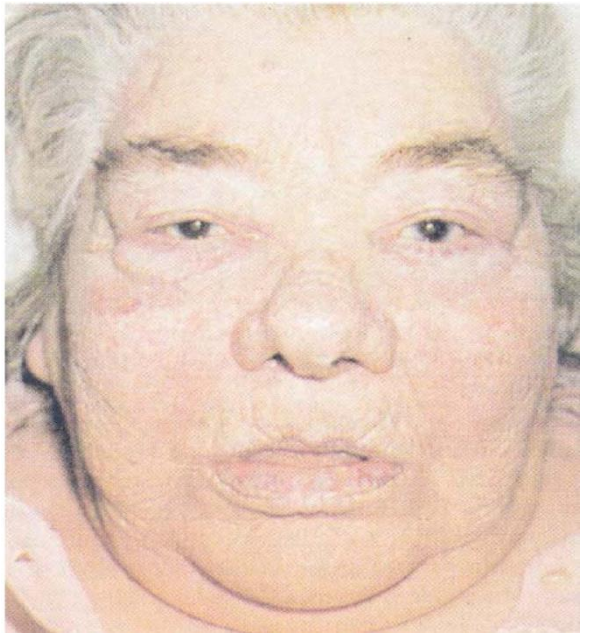
Exophthalmos



Goiter



Hypothyroidism



Q: Mention the 3 lines of treating this patient

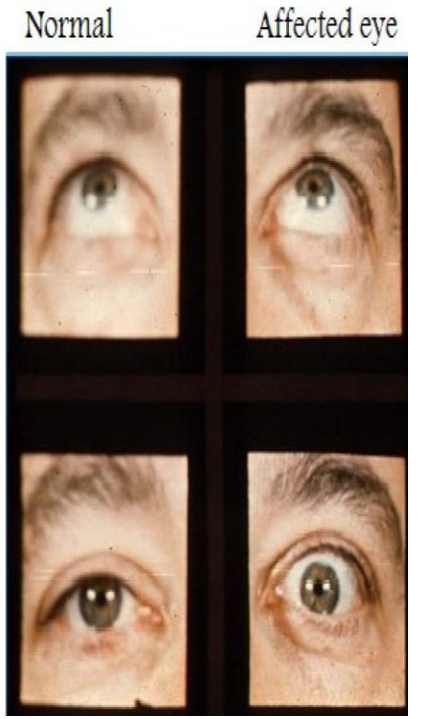
-Anti thyroid Drugs (Propythiouracil, methimazol,)
-Radio active iodine ablation
-Surgery (thyroidictomy)
-other drugs: beta blockers, sodium ipodate or iopanoic acid

Q: Mention 3 findings on pt's hands :

Tremors , moist skin , palmar erythema

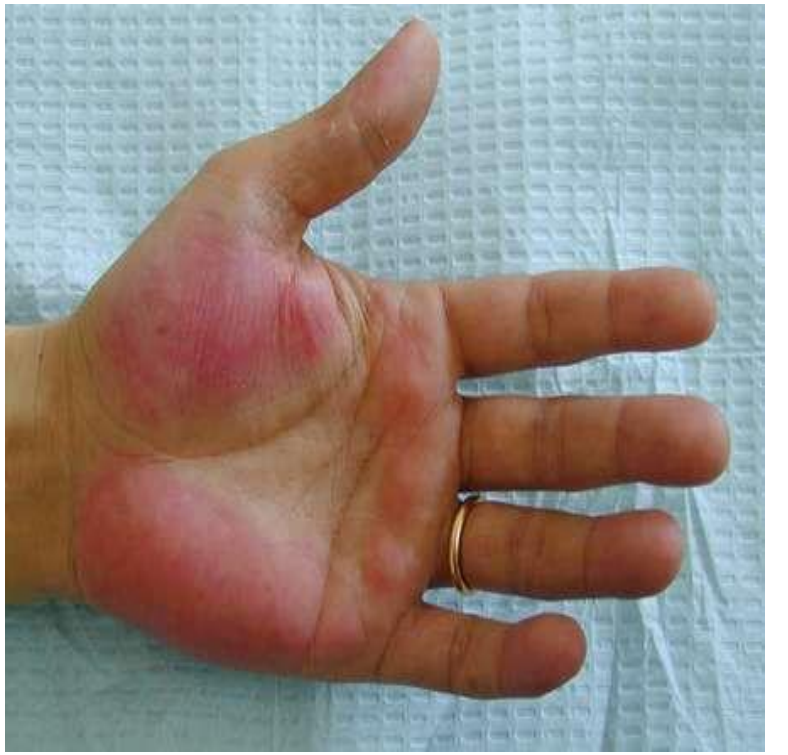
Lid Lag

- During Examination of the eyes of Thyrotoxicosis patient. What is this sign ?



Q: Mention 2 causes of this condition.

Pregnancy
Thyrotoxicosis
Liver cirrhosis
Diabetes
Autoimmune diseases: rheumatoid arthritis
Smoking



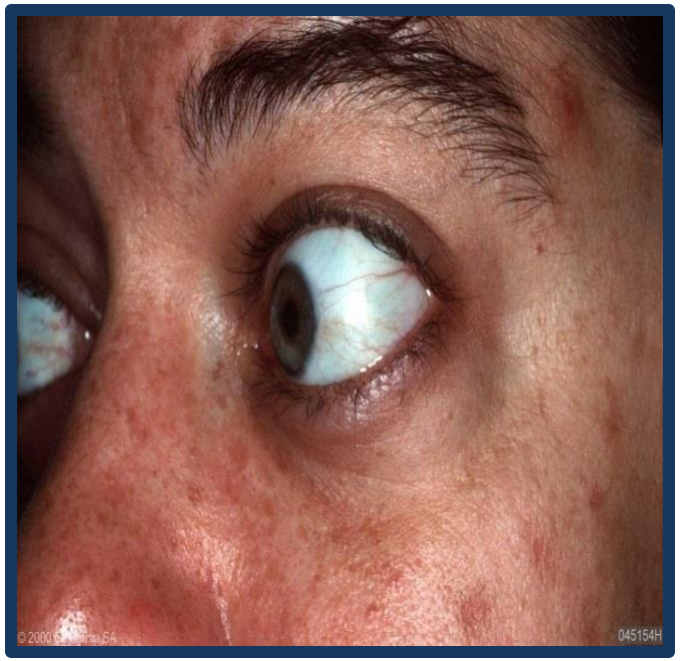
Q: This 30-year-old woman presents with weight loss sleep disorders and this orange skin rash on her lower limbs.

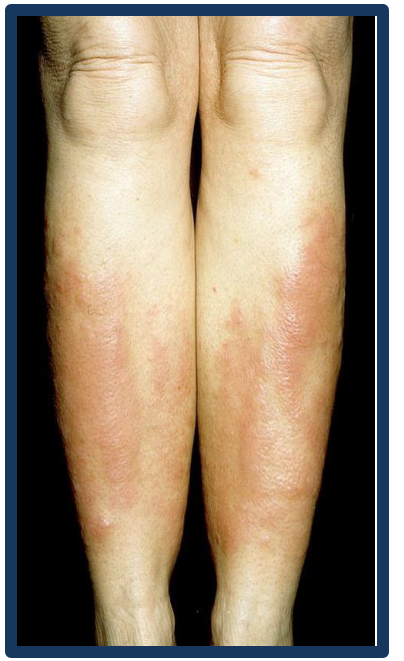
A. What is the likely cause?

Thyrotoxicosis

B. Name one specific immunologic test to confirm diagnosis

Thyroid stimulating immunoglobulins (IgG) (bind to TSH receptor causing production of thyroid hormone)





Patient c/o tremor & palpitation is trying to follow the examiner's finger, what is the sign shown in this patient?

Lid Lag

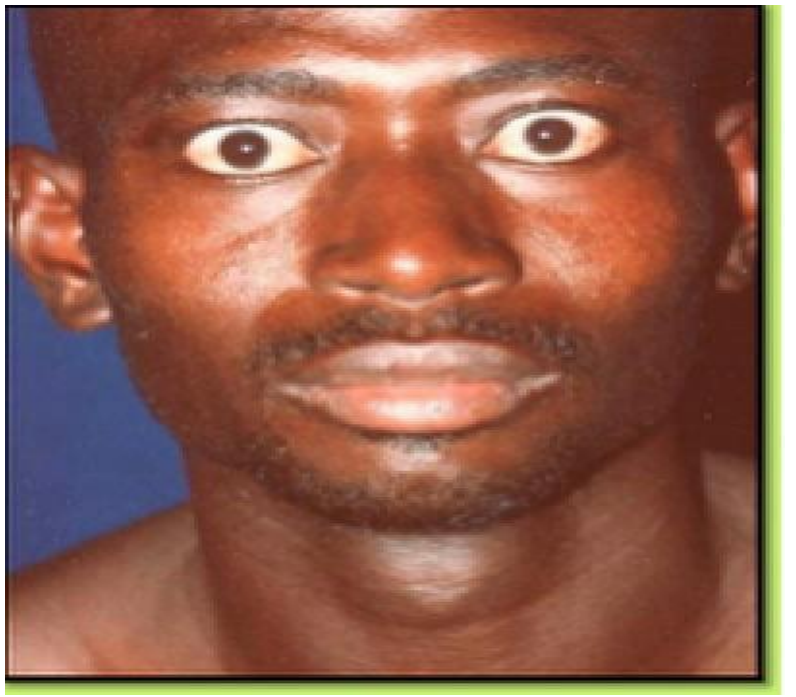
Q: thyrotoxicosis pt ,,

A. Mention 2 face features in this pt

Exophthalmos , lid lag , lid retraction , etc.

B. What is his radio iodine uptake ?

Increase diffuse uptake



Q: old women has been complaining of 3 months palpitations.

Mention 3 physical signs?

she was chacixic with exophthalmos and temporalis wasting

Dx?

Hyperthyrodism.

Graves -diffuse iodine uptake
Plummer disease – patchy uptake

Exophthalmos Peritibial myxedema Thyroid bruit - Specific to graves

Subacute thyroiditis- very tender on palpation

Thyroid storm Tx – IV fluid, cooling blanket, glucose
-antithyroid agents (PTU every 2 hrs) follow with iodine to inhibit thyroid hormone release
-beta blocker
Dexamethasone

PTU and methimazole can cause: agranulocytosis and hepatotoxicity
Initial test of choice for hyperthyroidism is TSH

Q: 34 YO female pt come to you with fatigue, hair loss, her blood pressure 130/80, HR 12

What is the Spot Dx ?

Hypothyroidism.

What is most diagnostic lab investigation ?

Thyroid function test

What is The Treatment ?

Give thyroxine & triiodothyronine

Q: 37 YO female presented with thyroid enlargement, the thyroid was firm, non- nodular & double-sized.

She is suffering from increase in weight, cold intolerance, thin dry skin & hair loss as well as menstrual irregularities.

What is your Dx?

Hypothyroidism

Give 2 causes of such condition

Iodine deficiency,

Hashimoto's thyroiditis .

What drug would you prescribe to this pt?

Thyroxin

Q: Asymptomatic 25 year-old male, T3 is normal, T4 is also normal, but TSH is 6.4 .

1.What's your diagnosis?

Subclinical hypothyroidism.

2.What's the management?

We only monitor the patient & follow him up (as long as his TSH is below 10, no need for thyroxine, except if antithyroid antibody for hashimoto is positive we start tt even TSH below 10, or elderly complains from unusual complains).

Q: Asymptomatic 25 year-old male, T3 is normal, T4 is also normal, but TSH is 9.

•1. What is the diagnosis?

It is subclinical hypothyroidism

•2. What is the management?

while the TSH remain below 10(mIU/L),monitor the patient and do T3, T4,TSH test once every year. If TSH becomes more than 10, the patient become symptomatic, infertile or has goiter; give Levothyroxine (LT4)

•3. What Is the next step ?

thyroxin

Q: patient has normal T3 , T4 but TSH : 15

1. What is your diagnosis ?

Subclinical hypothyroidism .

2. How do you treat ?

Thyroxin .

Q: Case About A Female Increase In Weight And Decrease The Activity She Had , T3 T4 Normal TSH Elevated .

•What is the Diagnose ?

Subclinical hypothyroidism

Q: Patient Came To Your Clinic And The TFT Was :

•TSH= 100

•T3= Raised

•T4= 0 normally 4.6-12(or T4 Raised And T3 =0) .

1- What Is The Dx ?

Hypothyroidism

2- What Is The Treatment?

Thyroxine

Q: Female pt presented with tremors, loss of wt & irregular irregular pulse.

- Dx?

thyrotoxicosis

- Most common rhythm you see in this case?

atrial fibrillation .

- Invistigations ?

thyroid function test .

Q: A 23 YO woman, presented to ER presenting with diarrhea, excessive sweating, & tremor. on examination RR: 32, BP 130\90, HR: 120.

What is the diagnosis? "2 points"

Thyrotoxicosis.

What is the test should be done?

Thyroid function test.

Give 2 modalities of treatment in such a case?

Radioactive iodine, Thyroidectomy

Q: A 25-year-old lady with progressive fatigue, irritability and recurrent palpitation. On exam, she appears anxious. HR= 120/min and irregular, BP 130/60. Skin is warm and moist. Noted to have fine tremor and difficulty rising up from sitting position. She has lid lag but normal ocular motility, thyroid is diffusely enlarged with a prominent impulse is bounding.

•Lab:
-Serum free T4 = 2.4 mg/dl (.8-1.8)
-Serum TSH = 0.001 (0.2-4.6)

•What is the medical condition ?
Thyrotoxicosis, Gravis disease
•What is the eye abnormality you can find in this patient ?
Exophthalmos
•Mention one ECG abnormality you can find it in this patient
Atrial fibrillation

Q: 34 YO female pt come to you with fatigue, hair loss, her blood pressure 130/80, HR 12 .

1- What is the Spot Dx?

Hypothyroidism.

2- What is most diagnostic lab investigation?

Thyroid function test .

3- What is The Treatment?

Give thyroxine & triiodothyronine.

- Q: 37 YO female presented with thyroid enlargement, the thyroid was firm, non-nodular & double-sized.
- She is suffering from increase in weight, cold intolerance, thin dry skin & hair loss as well as menstrual irregularities.
- 1- What is your Dx?
- Hypothyroidism.
- 2- Give 2 causes of such condition.
- Iodine deficiency, Hashimoto's thyroiditis, ...
- 3- What drug would you prescribe to this pt?
- Thyroxin.

Q: Patient with sweating, palpitation, heat intolerance…. Neck pain and tenderness, no goitre, increased T3, T4 and low TSH..

1- what's your diagnosis?

thyrotoxicosis/ subacute thyroiditis

2- next investigation?

RAIU/ thyroid scan

3- what's the finding in thyroid scan?

decrease uptake

4- what's the treatment?

symptomatic and supportive tt; NAID, analgesics, antipyretics, beta blockers… no antithyroid drugs since hyperthyroid state id sue toe release of previously formed thyroid hormones

Q: Patient with low t3 and t4 but TSH was high with other lab tests which were normal ,

1- what is your diagnosis

primary hypothyroidism

2- and treatment?

thyroxin (treatment)

3- mention other 2 symptoms that may the pt have?

cold intolerance, alopecia, menorrhagia, constipation

Hypocalcemia

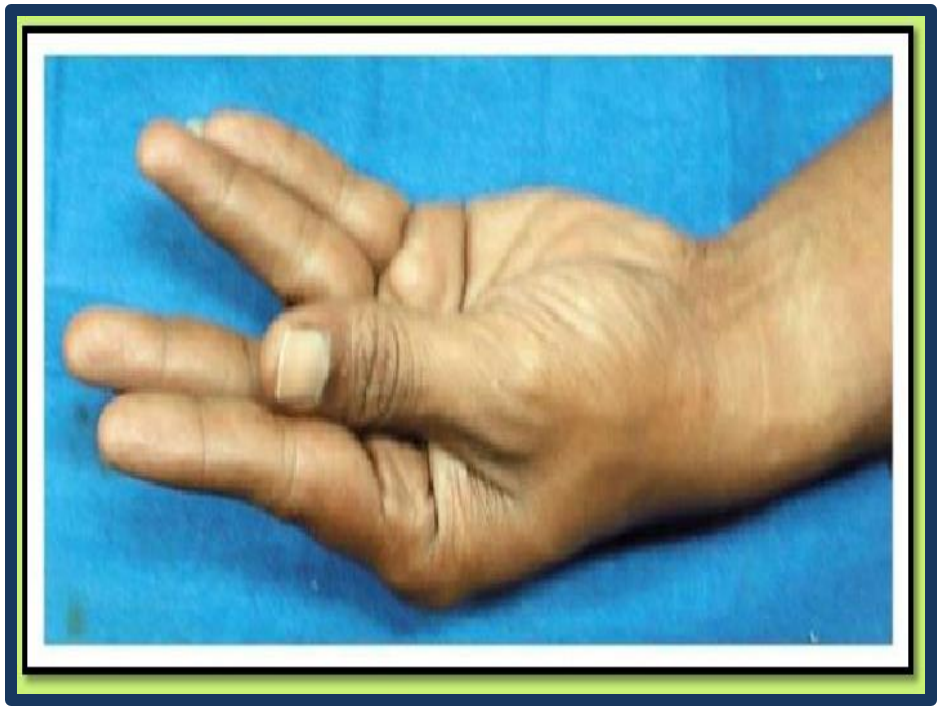
Serum ca+2 below 8.5 mg/dl

Q: pt after total thyroidectomy presented with this condition, what is the cause ?

Hypocalcemia (carpopedal spasm).

*If cuff of sphygmomanometer where present in pic…

it's called troussie sign



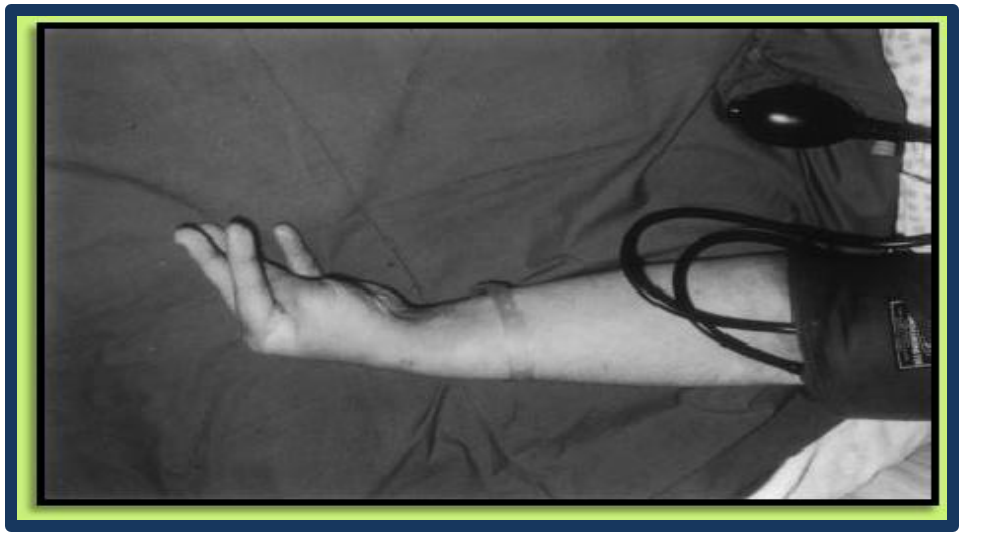
Q: 33 YO male pt, underwent subtotal thyroidectomy 5 days ago, presented with this

pic. What is this sign?

Troussie sign

What is the investigation of choice?

Ca+2 level



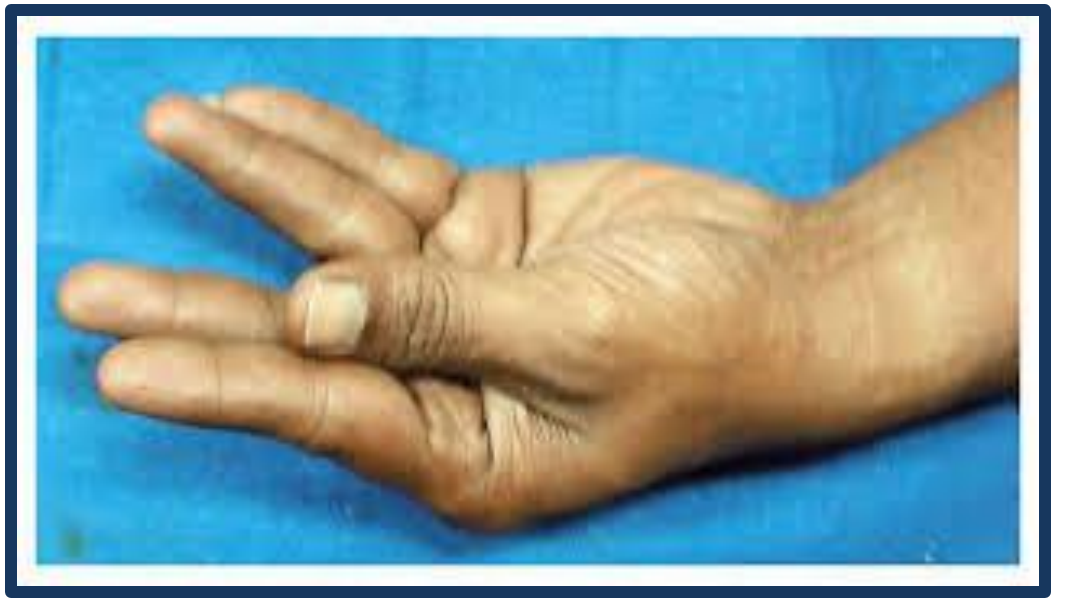
Q:A 35-year-oldman on furosemide presents with a 2-day history of cramps and paresthesia in the arms. This physical finding is reproducible by inflating a blood- pressure cuff placed on the patient's arm.

A: What is the cause of this presentation?

Hypocalcemia

B: What is the name of this sign?

Trousseau sign



Q: Name of this sign ?

Chvostek sign

cause ?

Hypocalcemia

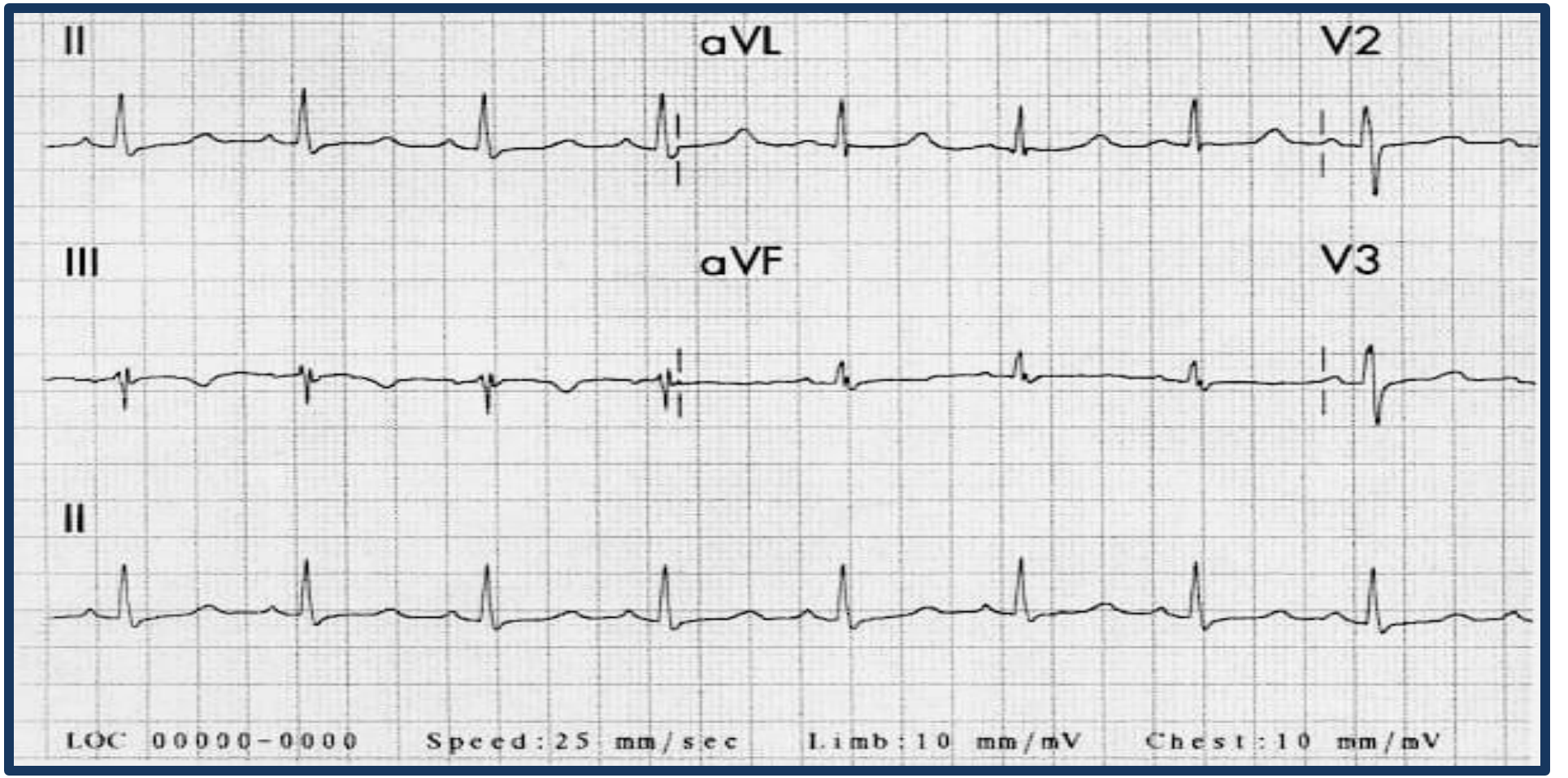
Q1 - What is the abnormality in this ECG?

1- prolonged QT segment

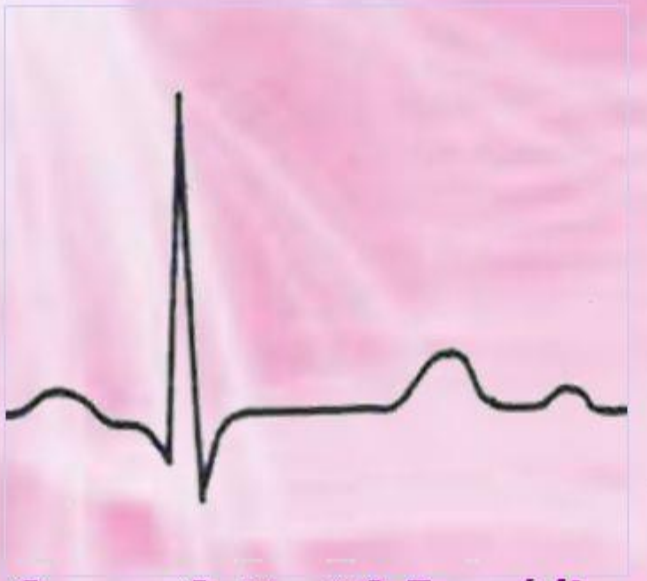
2- prolonged ST segment

Q2 - What is your dx ?

Hypocalcemia

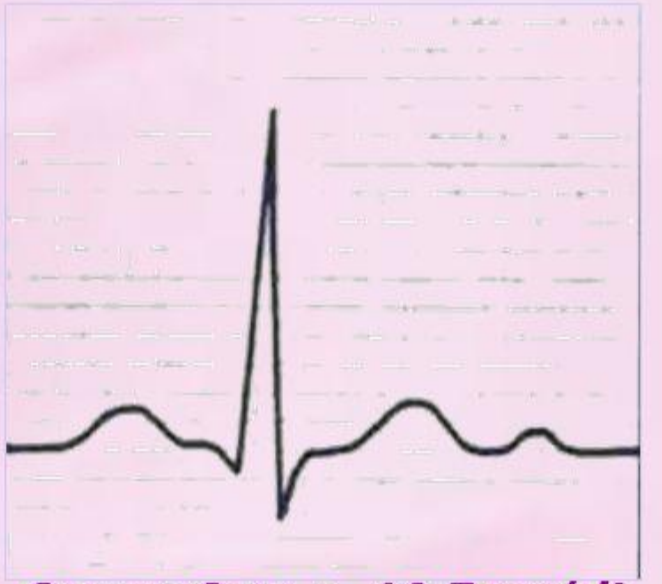


ECG Changes: Hypocalcemia/Hypercalcemia



Serum Ca^{++} < 8.5mg/dL

- *Lengthened ST*
- *Lengthened QT*
- *May cause Torsades de pointes*



Serum Ca^{++} > 10.5mg/dL

- *Shortened ST*
- *Shortened QT*

Q:Mention 3 causes of hypocalcemia :

1-Hypoparathyroidism (after neck surgery, autoimmune, infiltrative diseases)
2- Large-volume blood transfusions (citrate in transfused blood can bind calcium)
3- Magnesium depletion (decreased PTH release)
4-Acute pancreatitis (calcium binds to free fatty acids released by lipase)
5- Acute respiratory alkalosis (increases binding of calcium to albumin)

Q: Clinical Presentation of hypocalcemia :

Neurologic :

1. Numbness, paresthesias (especially perioral), muscle irritability, tetany
2. Chvostek sign (spasm of the facial nerve when tapped)
3. Trousseau sign (carpopedal spasm elicited by inflating blood pressure cuff above systolic pressure)

Cardiovascular

1. Hypotension with decreased contractility
2. Prolonged Q-T interval on electrocardiogram

Pulmonary

1. Bronchospasm

Q: Treatment of hypocalcemia:

- Severe cases (symptomatic patients):

Iv calcium gluconate or calcium chloride

- Mild cases:

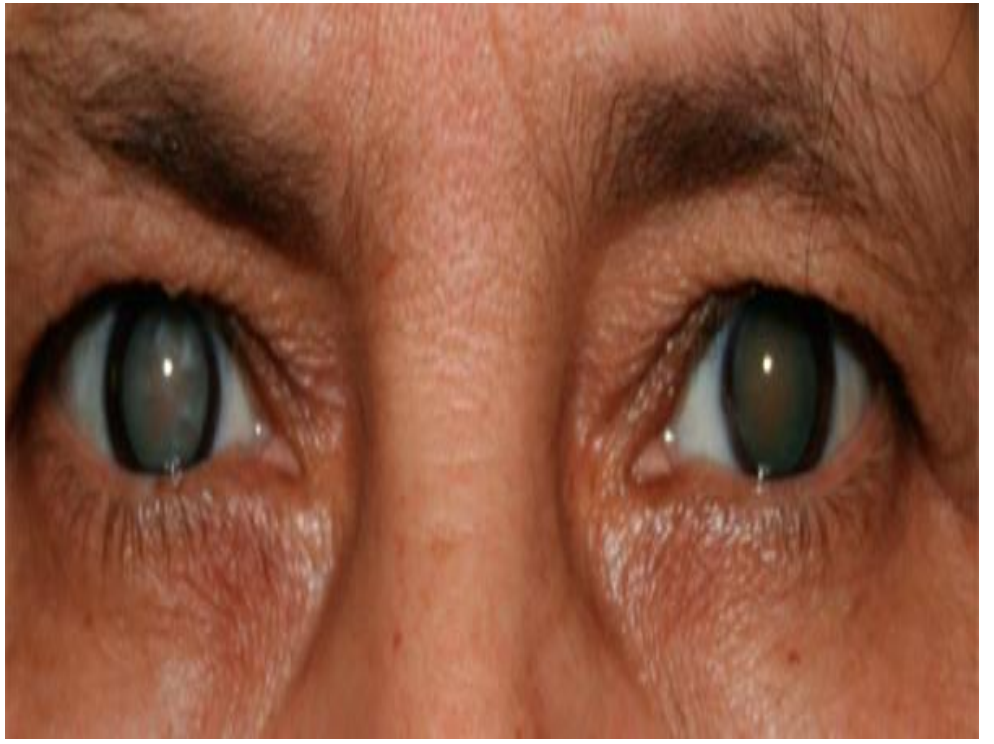
Oral calcium carbonate or calcium citrate

Vit D (oral calcitriol)

DM

Q: Pt with long history of DM

Bilateral cataract



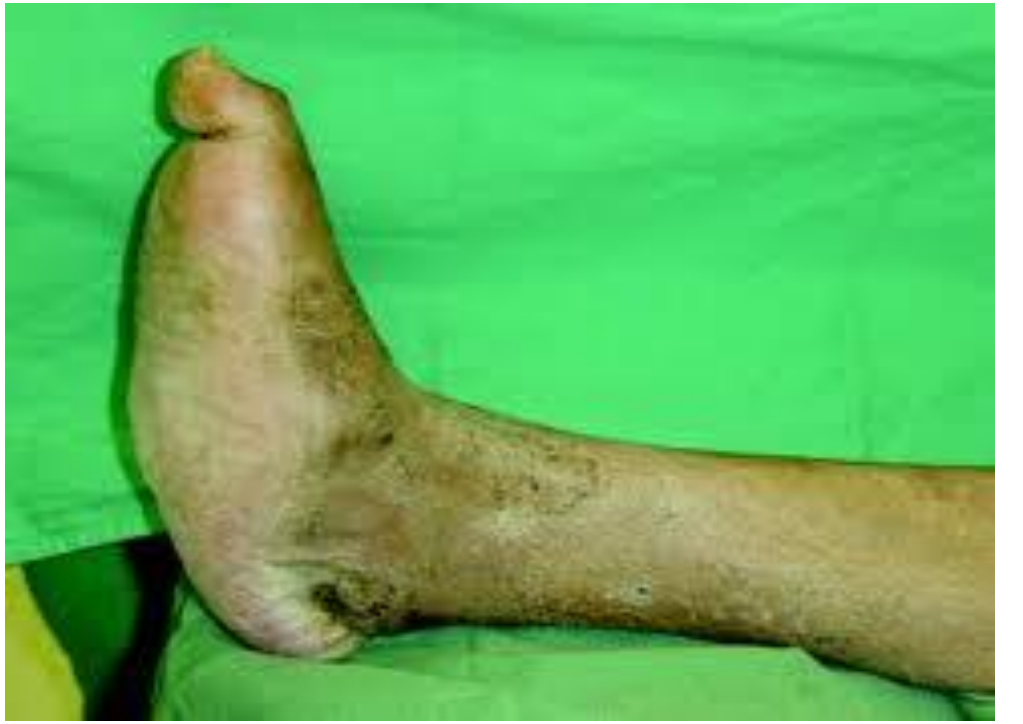
Q: Patient with uncontrolled diabetes

a) What do you see in the picture?

Charcoat joint

b) What's the cause?

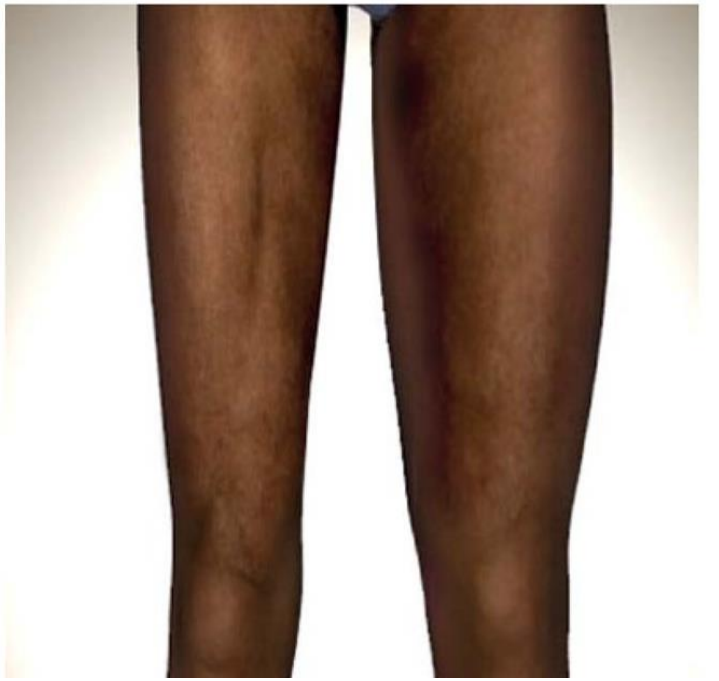
Diabetic neuropathy



Diabetic Amytrophy

- A 56-year-old man with type 2 DM (HbA1c 8.8%) of 24 years' duration presents with burning, lancinating pain in the right buttock, thigh, and legs. He had weight loss, On physical examination, there is wasting of the thigh muscles on the right side, with occasional involuntary twitching.

- What is the likely diagnosis?



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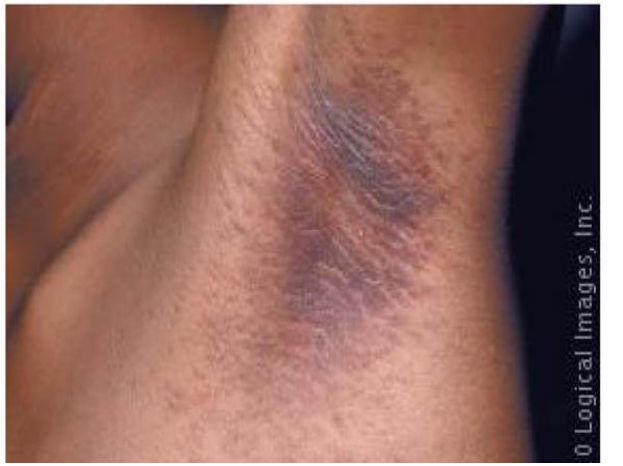
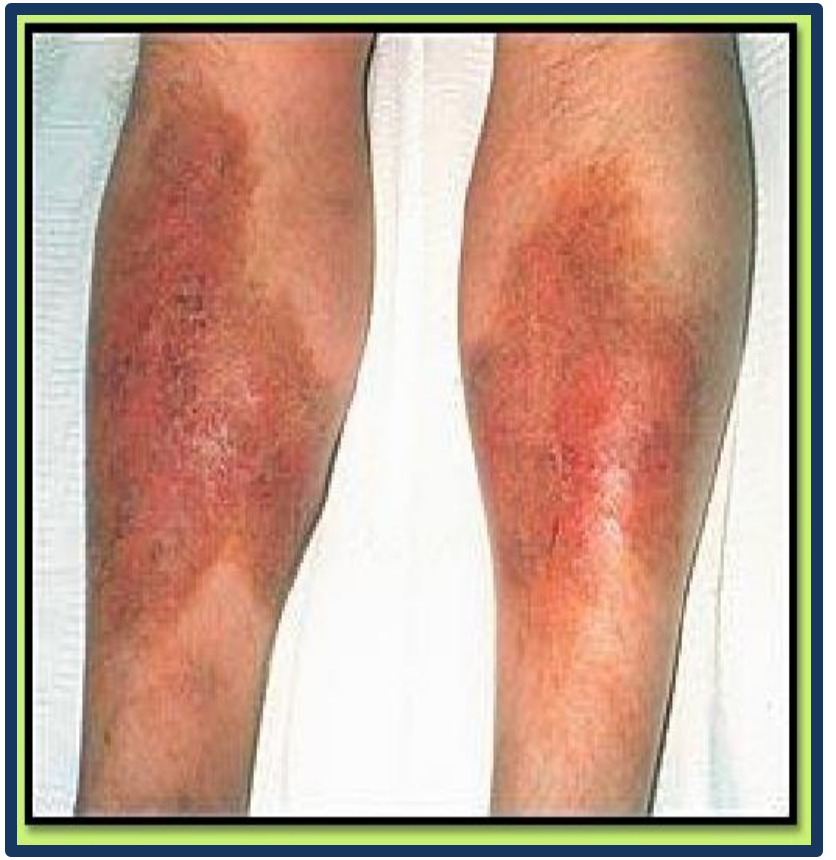
Acanthosis Nigricans

- An obese 24-year-old man presents to the emergency department (ED) with headache and fatigue. He has no previous history of DM. His blood glucose was 450 mg/dL, and his HbA1c is 12.3%. The physical examination is remarkable for this papillomatous, hyperkeratotic and pigmented lesions in both axillae. The patient had known about the lesions for at least 3 years.
- What is this lesion ?
- What is the significance of these lesions?



Q: 60 YO Pt known case of DM 30 yrs ago, presented with this asymptomatic, gradual, painless lesion. Name this lesion?

necrobiosis lipoidica.



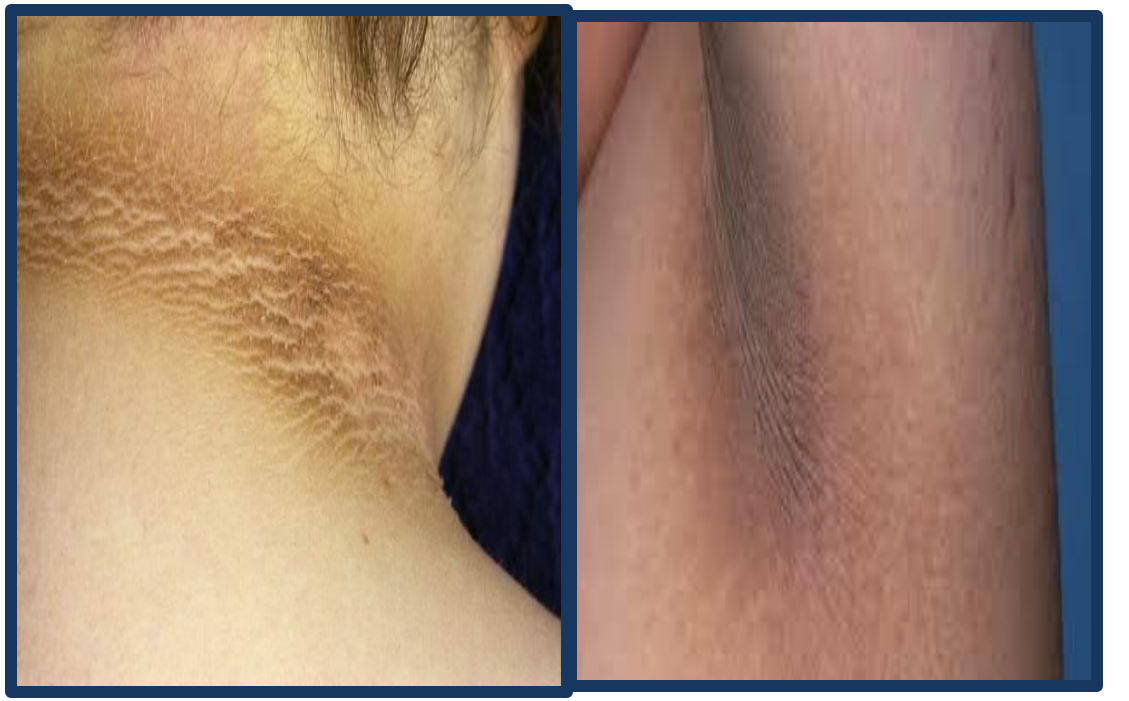
Q: 22 y/o diabetic pt:

A- name the lesion

acanthosis nigricans

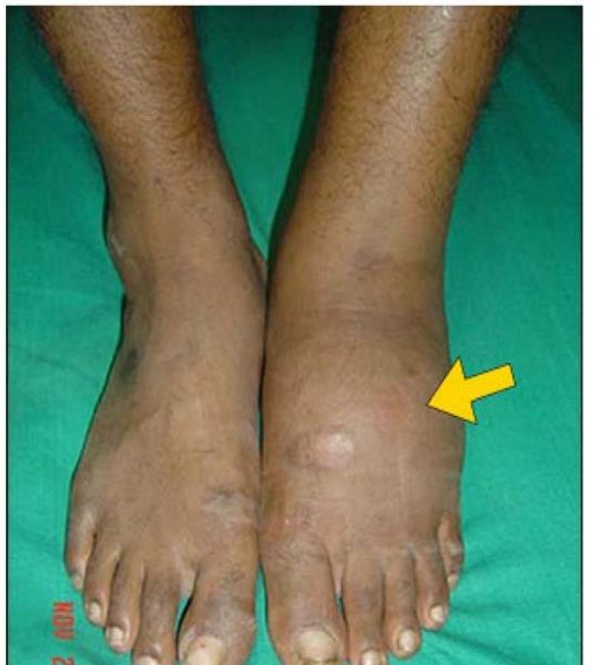
B- type of DM?

insulin resistance (type two)



Charcot Nueroarthropathy

- A 72-year-old man with long-standing uncontrolled diabetes and autonomic neuropathy presents to you with a painful and warm left foot.
- What is the most likely diagnosis for this patient?

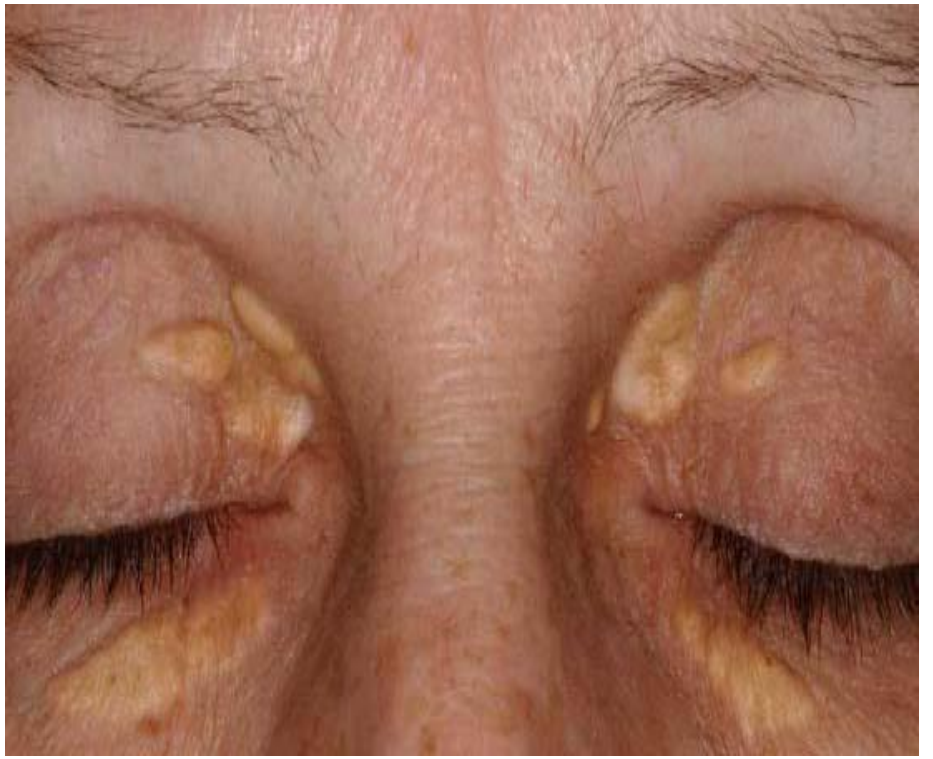


Q: What is the test you would order if you saw a patient with this sign?

Fasting SerumLipids

Patient has xanthalesma, a sign of hypercholesterolemia.

To evaluate LDL levels a fasting serum lipid profile would be needed



Q: A 54 YO male pt complaining of severe abdominal pain, nausea, vomiting. He is a known case of DM. 3 days before he came he had URTI. On P/E; there is tenderness in the epigastric area: RR: 33. investigations: Blood Sugar: 620 mg/dl, PH: 7.2, PaCO2 : 22, HCO3: 11.

What is your diagnosis?

DKA.

What type of acid-base disorder is this ?

Metabolic acidosis.

what are the most common causes of this condition? What is it in this case ?

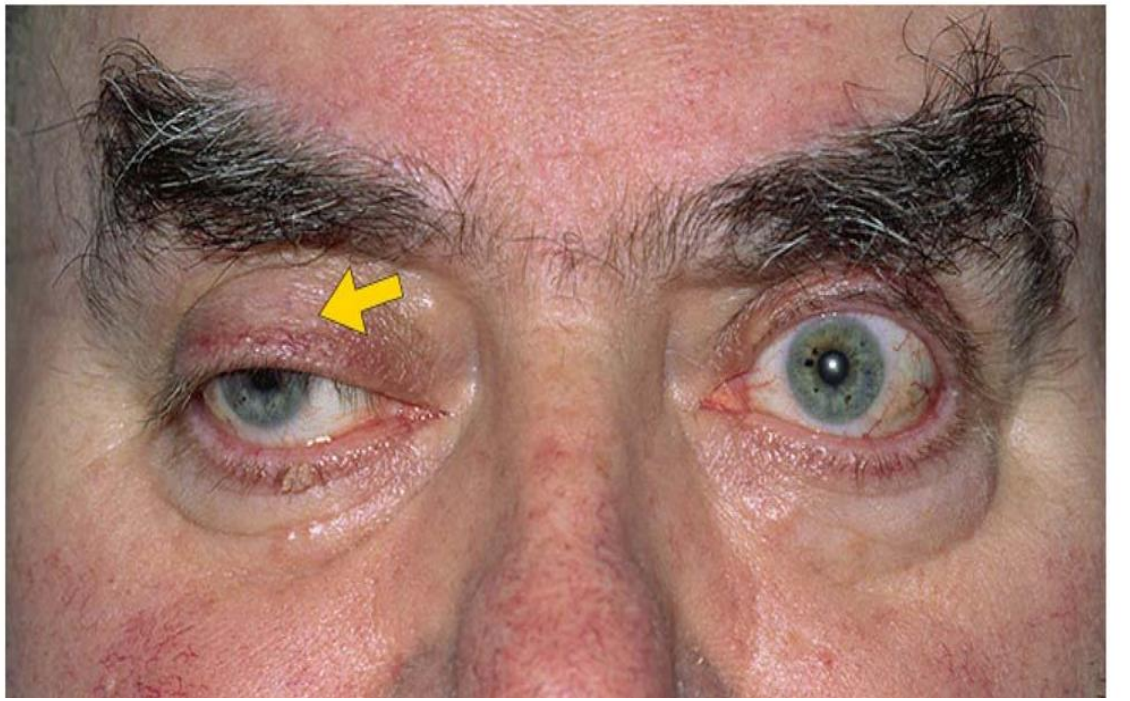
Infection, stress.

Give 2 lines of treatment in such cases.

- IV fluid -IV glucose - IV insulin

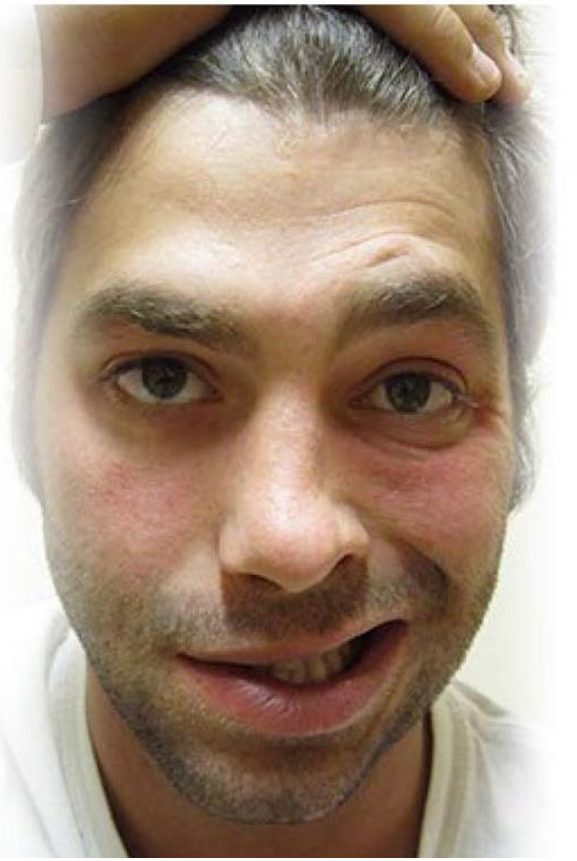
Right 3rd CN palsy

- A 72-year-old man with a history of type 2 DM and hypertension presents with a complaint of having awakened with headache and nausea. His right eye shows clinically remarkable findings (shown).
- What is the likely diagnosis?



Left Bells Palsy

- A 24-year-old male with uncontrolled type 1 DM (HbA1c 11%) presents with diabetic ketoacidosis (DKA). He also demonstrates features of cranial nerve neuropathy.
- What is the likely diagnosis, and what is the prognosis?



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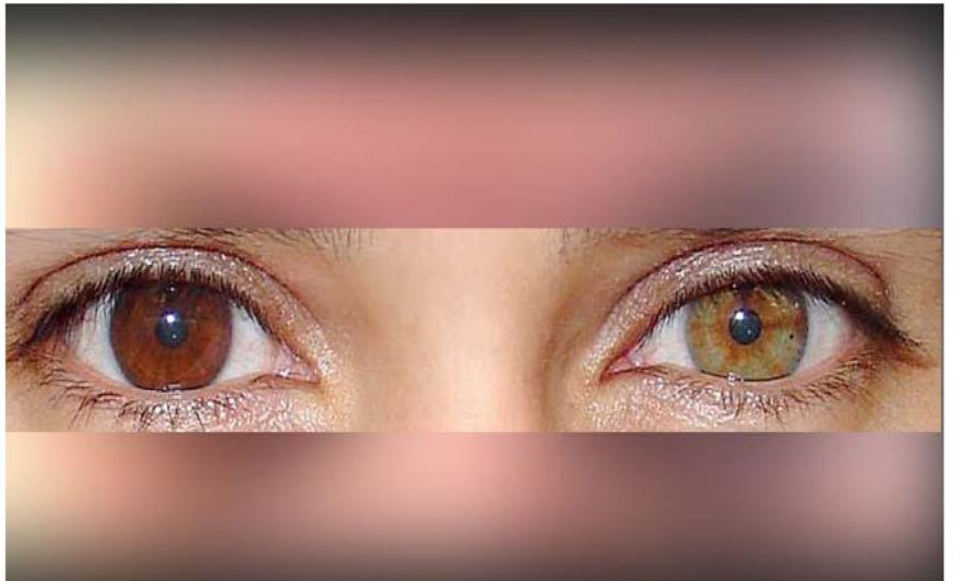
Medical School Committee

Heterochromia Iridium

- A young female patient with DM (HbA1c 8.9%) of 8 years' duration undergoes a physical examination, the results of which are completely normal. Her primary care provider asks you take a look at the patient's most recent photograph (shown), which, he thinks, demonstrates an abnormality that was not seen earlier.
- What is the diagnosis, and how is it linked to diabetes?



Q: Patient with diabetes on insulin, presented with abdominal pain, vomiting, diarrhea, & poly-urea. ABG was done (the values shows metabolic acidosis wide AG):

What is the Dx. ?

DKA .

Mention 2 lines of management.

Correction of fluid loss with intravenous fluids

Correction of hyperglycemia with insulin

Calculate the anion gap.

Na – (Cl+HCO3) .

Q :A 36yr old female pt has hypotensive and sweating and polyphagia and abdominal pain, her blood sugar was 450 HR 120, RR 25 ,temp 37.5

Has hx of polyuria, polydypsia 2 days ago but now is anuria, dry lips

The questions:

What is the diagnosis?

Diabetic ketoacidosis

Mention two important managements.

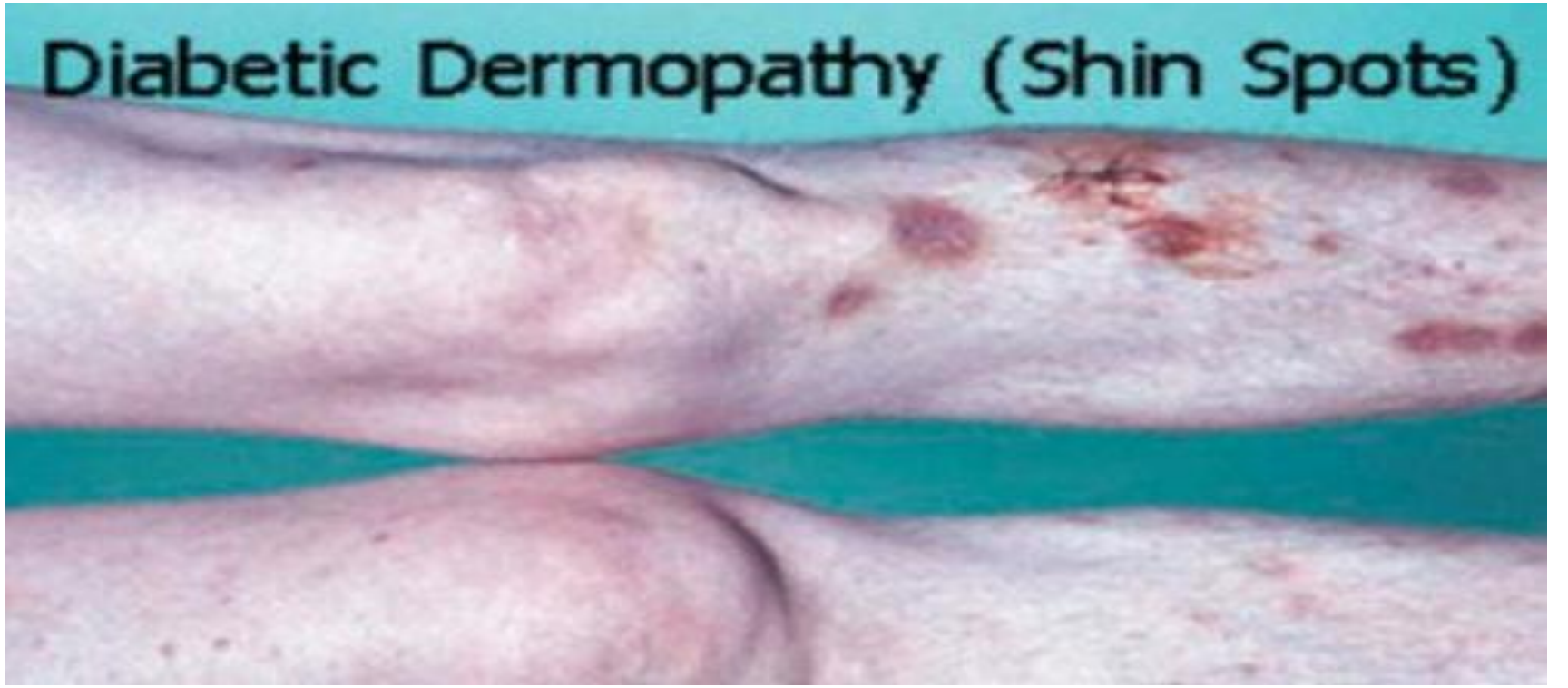
Iv fluid , iv insulin

Mention 2 investigation.

Abg, ketone bodies in the blood

Q: Name 3 ketone bodies?

acetoacetate, **beta-hydroxybutyrate, acetone**

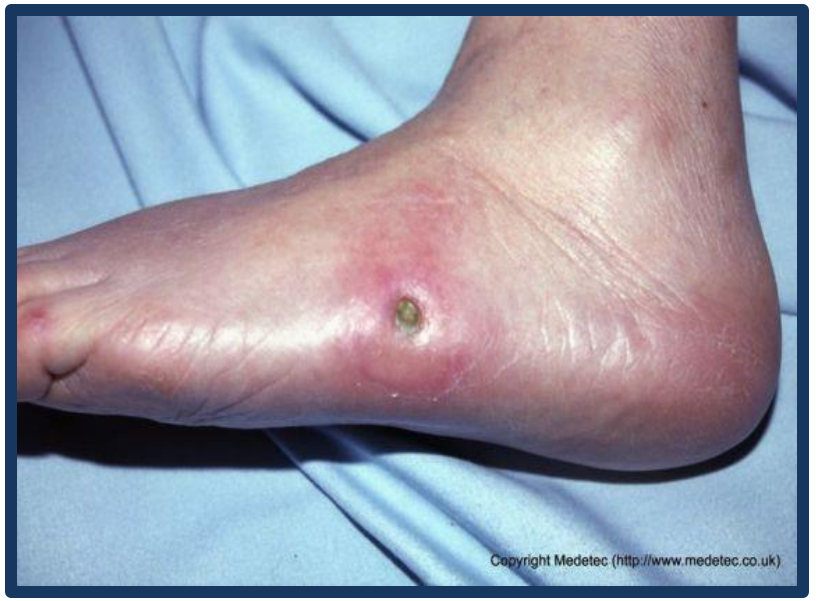

Diabetic Dermopathy (Shin Spots)

Q: 45 years old male pt came to your clinic with this lesion ,
1-name 3 history questions you would ask ?

polyphagia polydipsia and polyuria

2-Name 3 laboratory test you would order

fasting blood sugar , HB1AC , GLOUCOSE TOLERANCE TEST



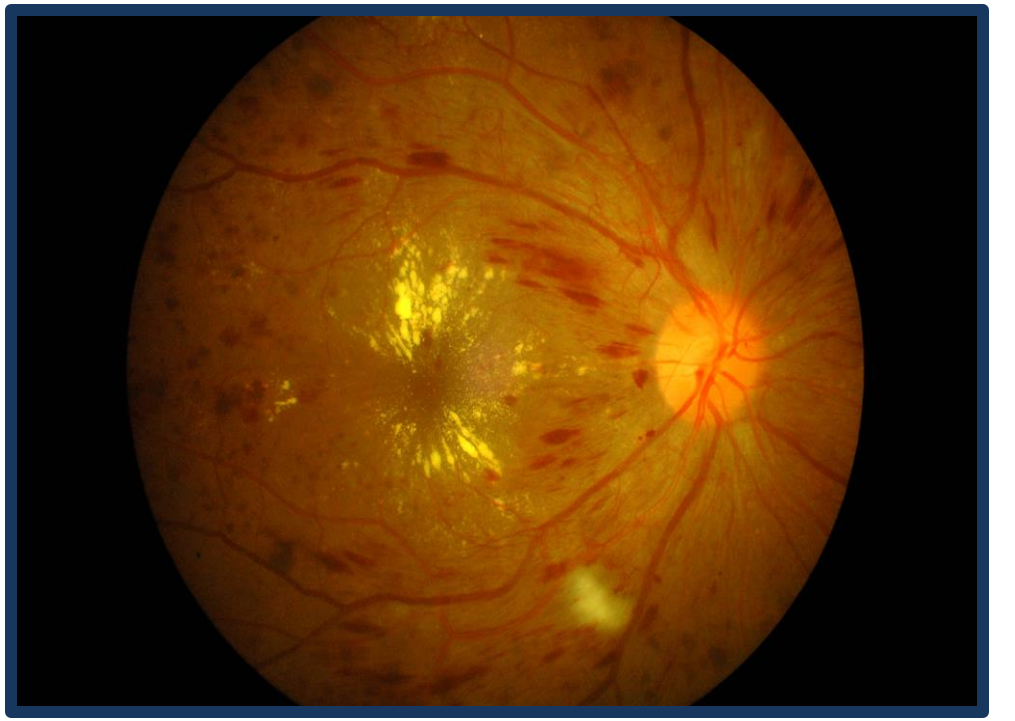
Q: 55 years old male pt came to your clinic with chef complain of decrease visual acute and recurrent UTI

1- 3 laboratory tests

HB1AC , urine analisis , CBC

2- name the abnormality in fundoscope

flare hemorrhage , cotton wool spots

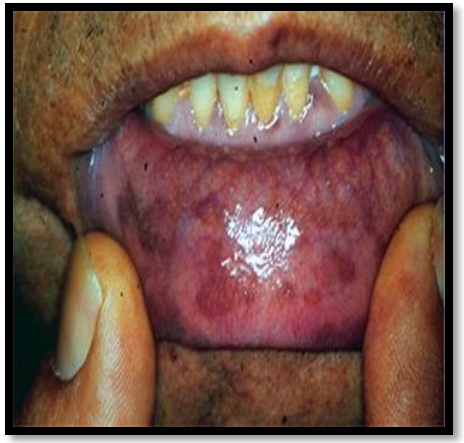


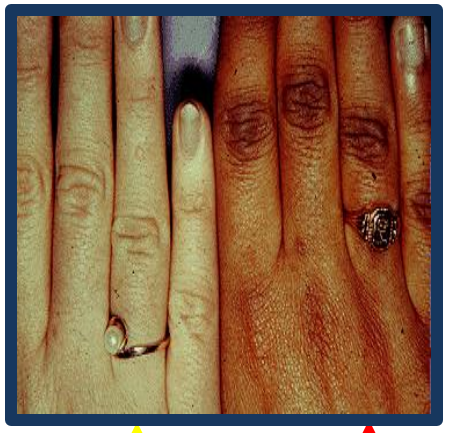
Primary chronic adrenal insufficiency (addison disease)

Q: This patient has generalized fatigue and hyperpigmentation presents with hypotension and hyponatremia.

- What is the initial screening test?

Cortisol AM (SERUM CORTISOL)



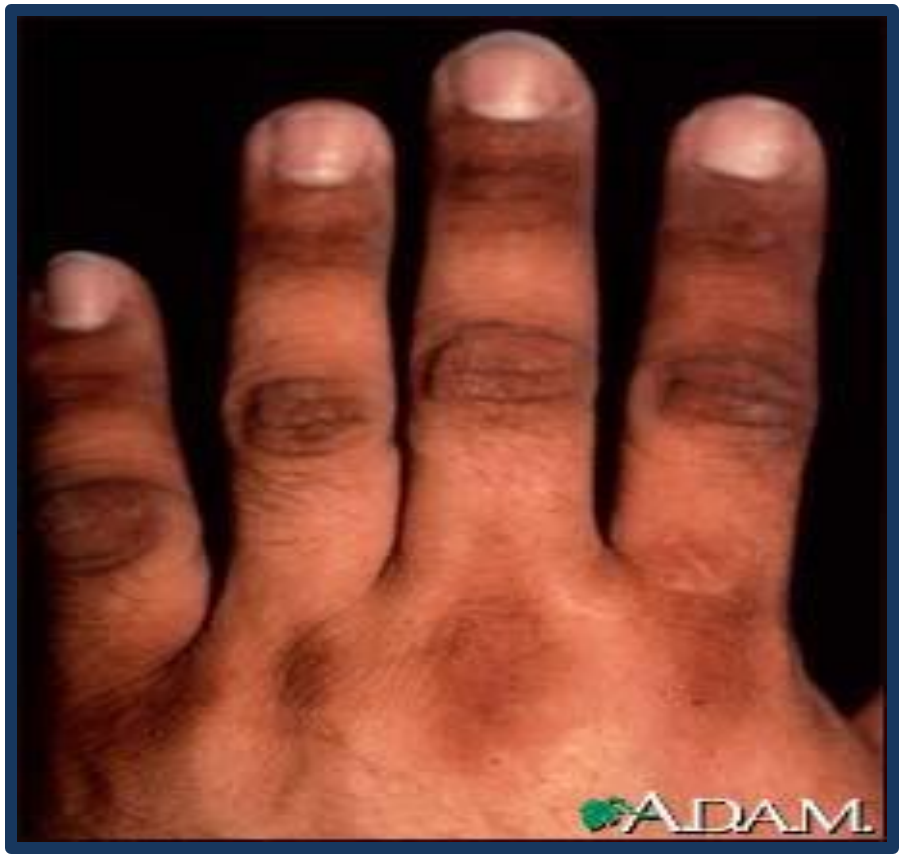


Normal sister

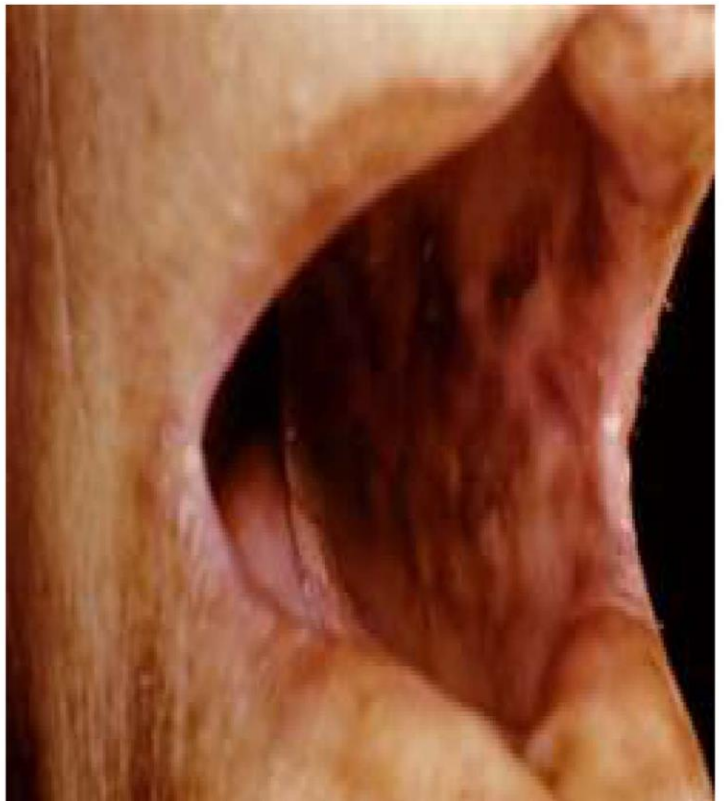
Patient

Q: What is the Diagnosis ?

adrenal insufficiency is manifested in the skin primarily by hyperpigmentation. Addison's disease or Chronic adrenal insufficiency



Mucosal pigmentation (Adrenal Insufficiency)



Q: A 17 YO male has fatigue, lightheadedness upon standing or while upright, muscle weakness, fever, wt loss, difficulty in standing up, anxiety for long period with hyper-pigmentation, this is his hand (inf.) compared to his brother (Sup.). What is your spot Dx.?

Adrenal insufficiency.

Q: SLE pt on steroids, presented with high fever, nausea, vomiting & hypotension(80\60). (There were many labs data, numerical values were given for ALL of them, normal ranges were given for some!)
Urea: high, creatinine: high, Na: 120, K: 5, HCO3: 10, Cl: 100, Glucose: 60, Ca: 2.3, urine analysis was positive for leukocyte esterase and nitrites,...that is what I remember)

What is the cause of hypotension?
Adrenal Crisis.
What is the underlying acid-base abnormality?
Metabolic Acidosis.
What is the cause of hyponatremia?
Low cortisol and aldosterone level.
Mention first two steps in management.
1. IV fluid. 2. IV Cortisone + Mineralocorticoids.

Q: A female patient known case of SLE and on steroids, presented complaining of high fever, nausea and vomiting, chills,dysuria, and hypotension 80/60 , her lab data are as follows:

- O2 sat 92%
- labs: Na 135 | K 5.9 | Cl 90 | Hco3 10 | Glucose 65
- Wbc 17,000 | Urine positive for nitrites and leukoeseterase.
- Other CBC parameters were normal.

What is your diagnosis?

Adrenal crisis / some answered sepsis or pyelonephritis (we're not sure).

What is the confirmatory investigation?

Blood culture (if sepsis) / 24 hour urine for cortisol (if adrenal crisis)

Calculate the anion gap?

AG = 35

How do you explain the bicarbonate level?

bicarbonate because of the increase in hydrogen ions that resulted from the acidosis (not sure)

What is the management?

IV fluids, IV mineralocorticoids & steroid, IV antibiotics.

Q: 30 years old lady with autoimmune hepatitis on prednisolone therapy presents with weakness ,fatigue and abdominal pain.
Important readings:
BP (95/60)
Blood glucose (65)
Hb (12)

1. What is the diagnosis?
Adrenal/Addison's crisis
2. What test you will order to confirm your diagnosis?
Serum Cortisol level

Q: Asthma pt , on oral steroids stopped his medications presented to the ER with fever fatigue his BP is 80/40
what is your diagnosis?
Adrenal crisis
What is your management other than IV fluid?
IV hydrocortisone

Q: SLE pt on steroids, presented with high fever, nausea, vomiting & hypotension(80\60).

- (There were many labs data, numerical values were given for ALL of them, normal ranges were given for some!)
- Urea: high, creatinine: high, Na: 120, K: 5, HCO3: 10, Cl: 100, Glucose: 60, Ca: 2.3, urine analysis was positive for leukocyte esterase and nitrites,...that is what I remember : S)

1) What is the cause of hypotension?

Adrenal Crisis.

2) What is the underlying acid-base abnormality?

Metabolic Acidosis.

3) What is the cause of hyponatremia?

Low cortisol and aldosterone level.

4) Mention first two steps in management.

A. IV fluid.

B. IV Cortisone + Mineralocorticoids.

Causes of addison

- Autoimmune primary adrenal insufficiency
- infection (e.g., tuberculosis)
- surgical excision
- bilateral hemorrhage of the adrenal glands
- meningococcal infection (Waterhouse-Friderichsen syndrome)

Clinical Presentation:

- skin hyperpigmentation
- hyponatremia and hyperkalemia
- Weakness
- hypotension
- nausea
- vomiting
- diarrhea
- weight loss

Diagnosis

- In primary adrenal insufficiency, plasma ACTH levels will be elevated
- cortisol (taken any time of day) is less than 15 µg/dL(unless serum albumin is less than 2.5 g/dL, as serum cortisol binding capacity is reduced)

- Gold-standard test of adrenal function is insulin induced hypoglycemia or insulin tolerance test , Test performed by administering insulin (0.1 to 0.15 U/kg) intravenously (IV), with measurement of cortisol levels during symptomatic hypoglycemia
- A normal response is considered to be a peak cortisol level greater than 18 µg/dL
- ITT contraindicated in presence of coronary artery disease, seizure disorder, or age above 60 years
- Most commonly used test is ACTH stimulation test Administer ACTH (cosyntropin) 250 mg IV or intramuscularly, Measure serum cortisol just before injection and 60 minutes following injection
- If cortisol level is 18.5 µg/dL or more at either measure, patient does not have adrenal insufficiency
- If cortisol levels stay below 18.5 µg/dL, adrenal insufficiency is present, and results are combined with ACTH levels, as described previously, to determine cause

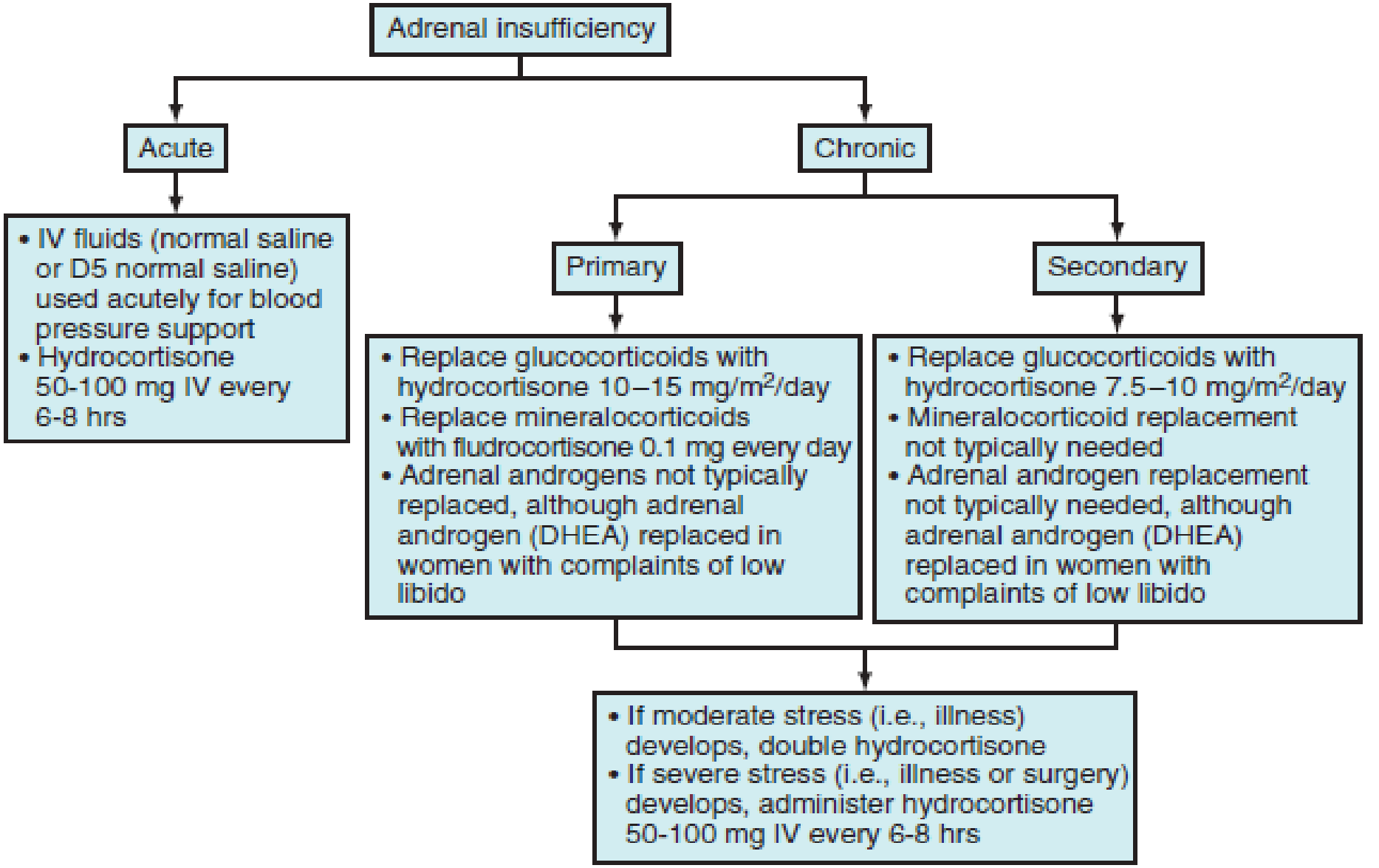


FIGURE 41-16 Treatment of adrenal insufficiency. D5, 5% Dextrose; DHEA, dehydroepiandrosterone; IV, intravenous.

acromegaly

Q: This patient has hypertension & DM ,sleep apnea ,vision problem, what's your diagnosis?

Acromegaly

Q: HTN, DM, What is the diagnosis and what is the diagnostic test?

Glocuse suppression test OGTTو, acromegaly

what's the visual field abnormality?

Bitemporal hemianopia

Treatment ?

First line surgery (trans_sphenoidal approach)

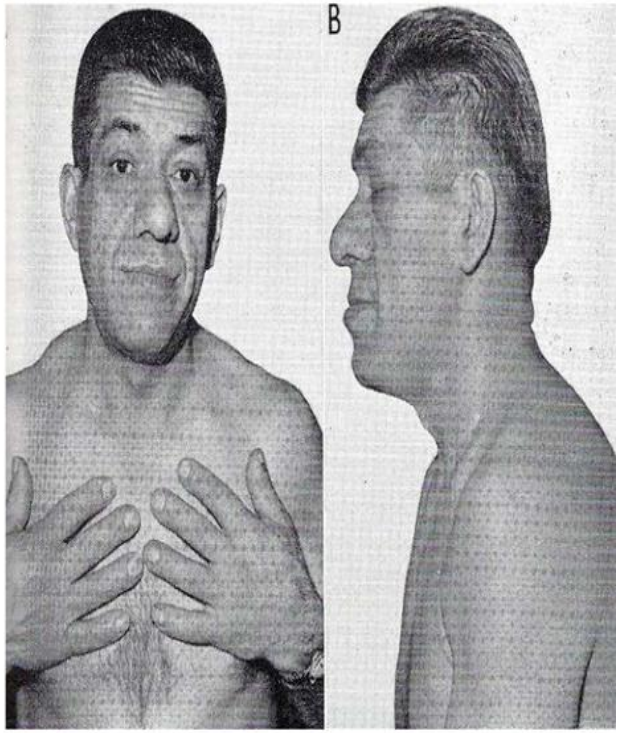
second line madication (somatostatin (lanreotide &octeriotide), dopamine agonist cabergoline , GH receptor antagonist)

Acromegaly

Other complications in patients with acromegaly is carpal tunnel syndrome

Their pressure is also usually high

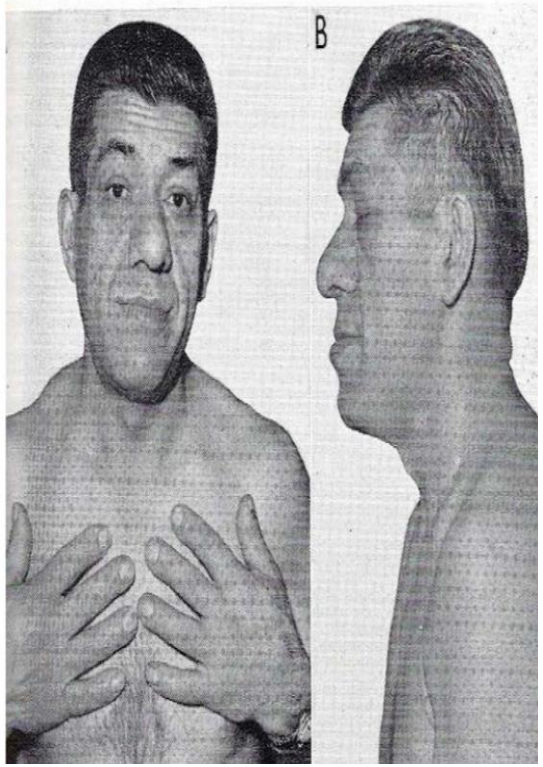
Due to excess growth hormone



This is patient with visual field defect
What is the first line of treatment of such patient presented to Endocrinology clinic ?

1st line treatment is trans sphenoidal surgery, followed by medical therapy for residual disease.
Radiation treatment usually is reserved for recalcitrant cases.

Also somatostatin and dopamine analogues and GH receptor antagonists are the mainstays of medical treatment for GH excess and are generally used when primary surgery fails to induce complete remission.



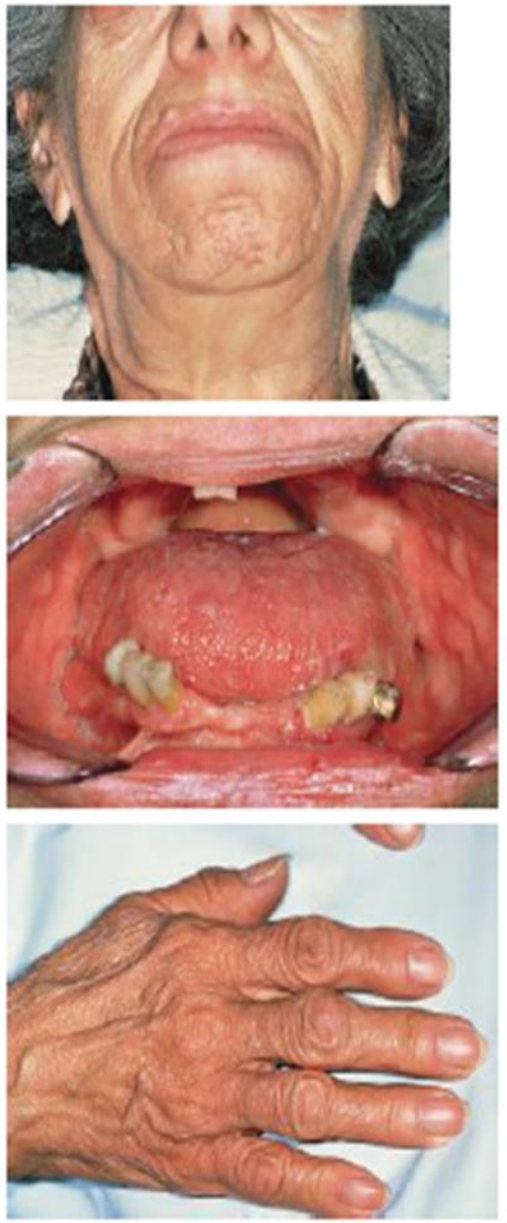


FIGURE 41-5 Acromegaly of the jaw and hand. (From Regezi JA, Sciubba JJ, Jordan RCK. *Oral Pathology: Clinical Pathological Correlations*. 4th ed. Philadelphia: Saunders; 2003: Fig. 15-8.)

Q: What is your spot dx?

Acromegaly

Name two lab tests to confirm your dx

A. Glocuse suppression test with oral glucose
B. B. serum IGF-1 level

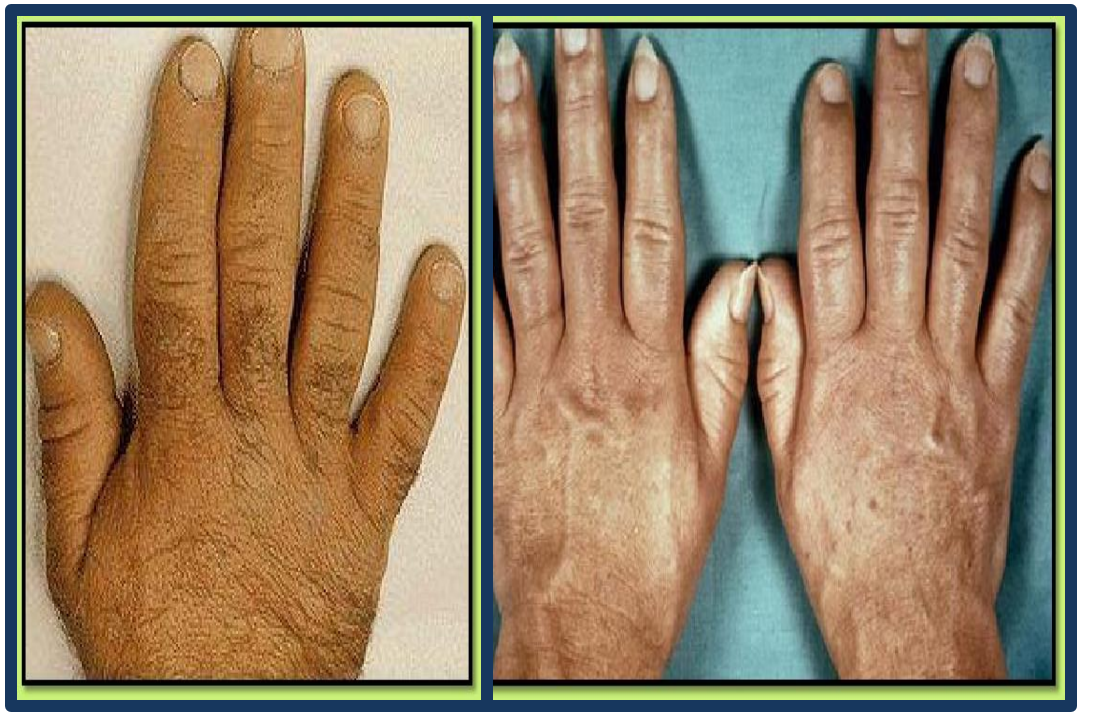
Q: This pt has HTN, diabetes insipidus, bone pain …What's your Dx.?

Acromegaly.

What's the diagnostic test?

Glucose suppression test ,OGTT

The photo is carpal tunnel syndrome



Q: what's your Diagnosis and what complications this patient might have ?

-Acromegaly

-carpal tunnel syndrome, DM,HTN, sleep apnea ,cardiomegaly

Q: Name 2 conditions that are associated with the diagnosis in this image

A: Diabetes Mellitus

B: Carpal Tunnel Syndrome

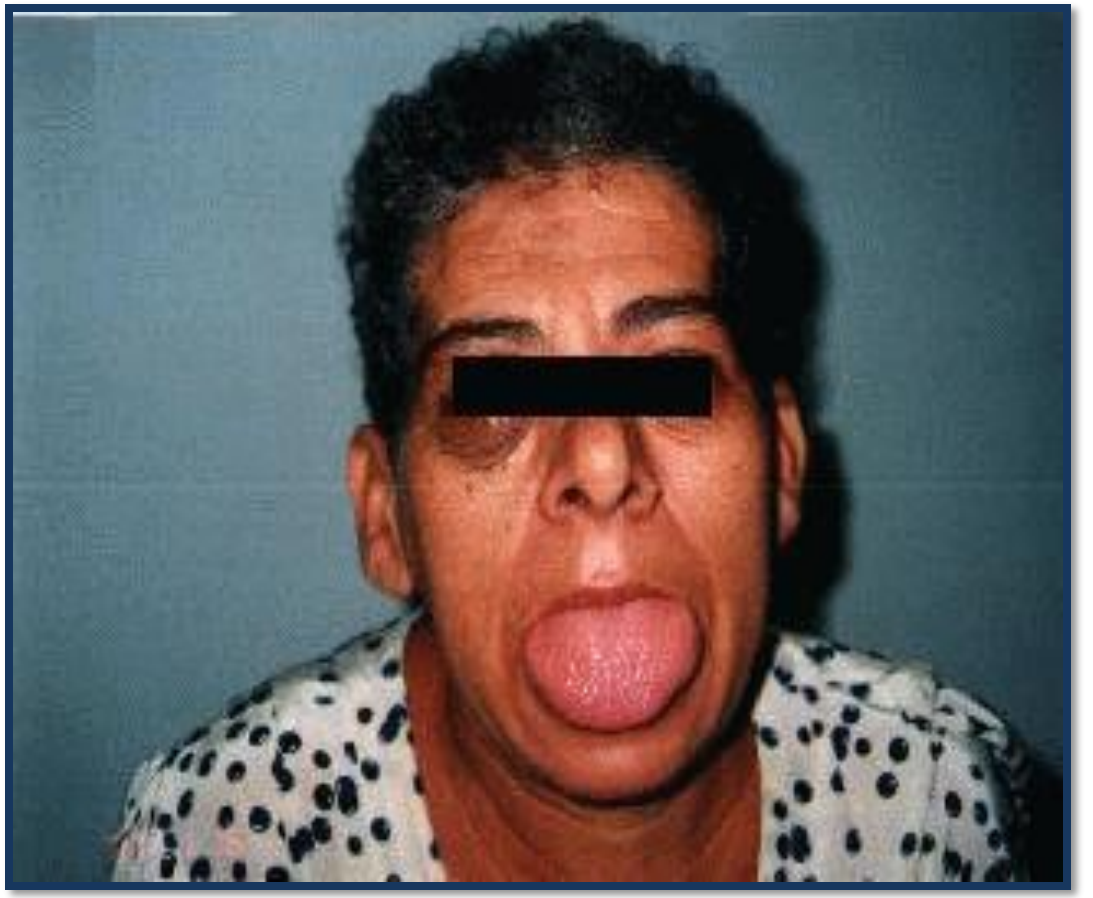
Q:This patient presents with obstructive sleep apnea, bilateral hand numbness, sweating and blurred vision.

-What is the most likely underlying disease?

Acromegaly

-What is the likely cause of her sleep apnea?

Macroglossia (enlarged tongue)



Cushing syndrome

اول 8 سلايدات هي المهمه لل Mini osce

الباقي مراجعه سريعه لل Cushing syndrome

- Background
- Cushing syndrome is caused by prolonged exposure to elevated levels of either endogenous glucocorticoids or exogenous glucocorticoids. Exogenous use of glucocorticoids should always be considered and excluded in the etiology of Cushing syndrome.
- Endogenous glucocorticoid overproduction, or hypercortisolism, can be dependent on or independent of adrenocorticotropic hormone (ACTH).

Describe the image
- rounded face (moon face)
- striae over the anterior chest

WHAT is your diagnosis
- Cushing syndrome

DDX
- obesity AS DIFFERENTIAL

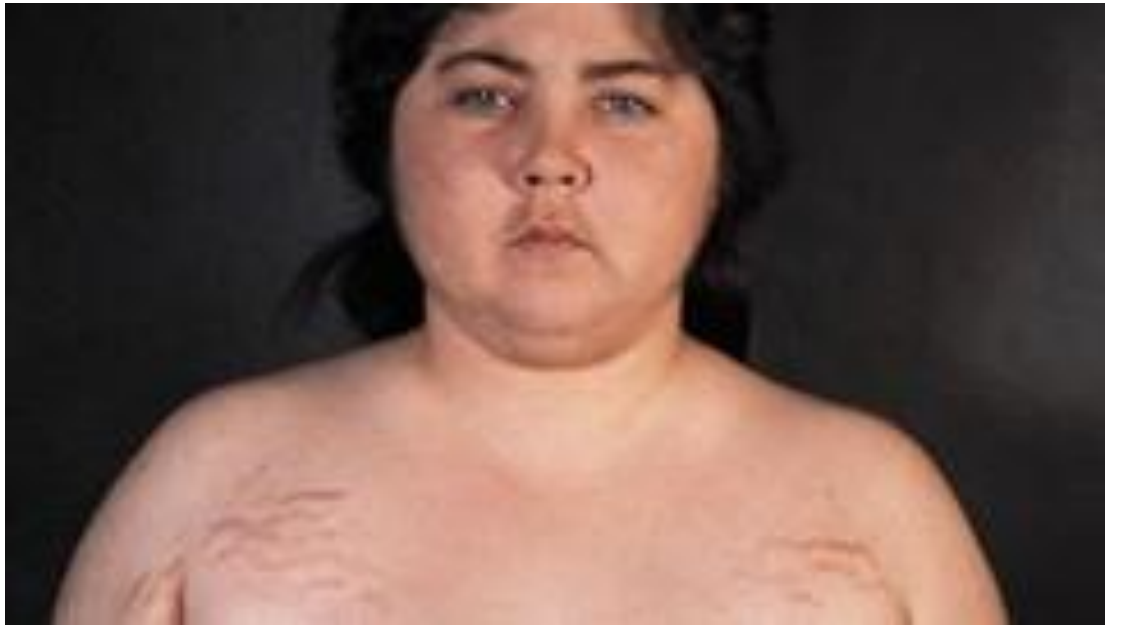
Mention # of symptoms
- weight gain
- difficulty in combing hair etc.

other are mentioned below

Mention other physical finding:
- buffalo hump
- facial lanugo hair
- acanthosis nigricans …. Etc.

Other are mentioned below

Mention 3 diagnostic test used to diagnose this patient
- 24 hrs urine free cortisol level
- low dose dexa suppression test
- mid-night serum and salivary cortisol level



Mention imaging study far this patient
- CT- brain
- CT ABDOMIN
- MRI with contrast for pituitary gland

Medication

1-Somatostatin analogs: Pasireotide

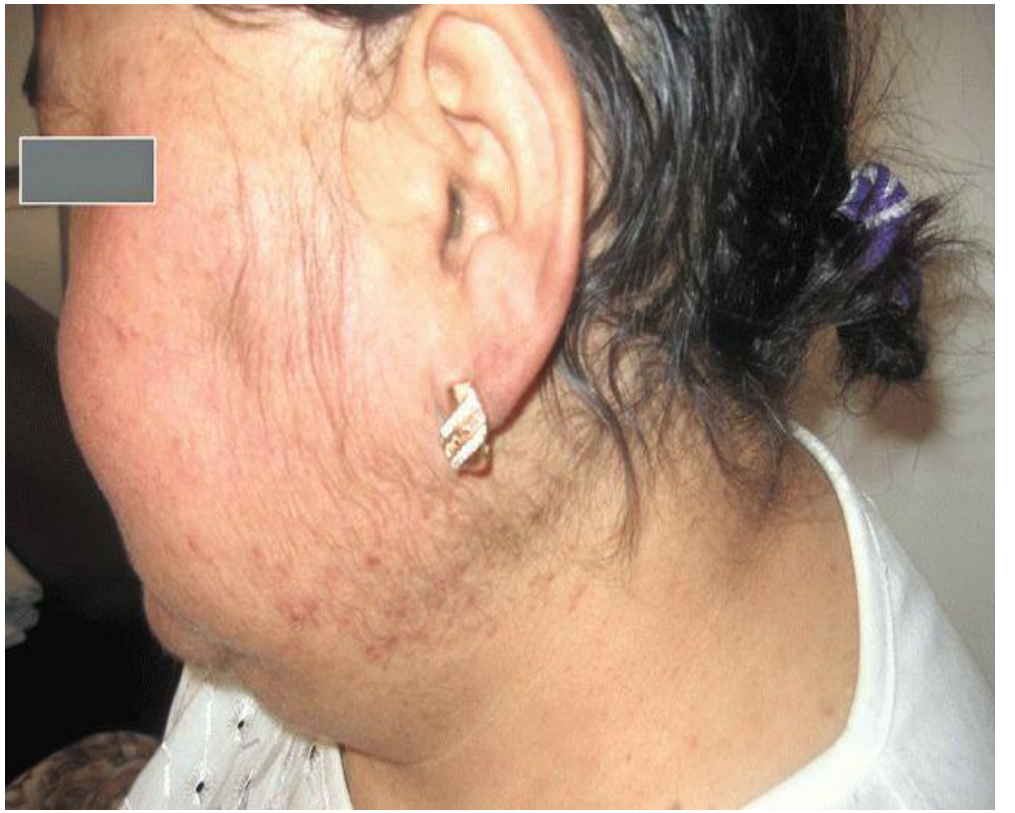
2- Adrenal steroid inhibitors: Metyrapone

3- Glucocorticoid receptor antagonist: Mifepristone

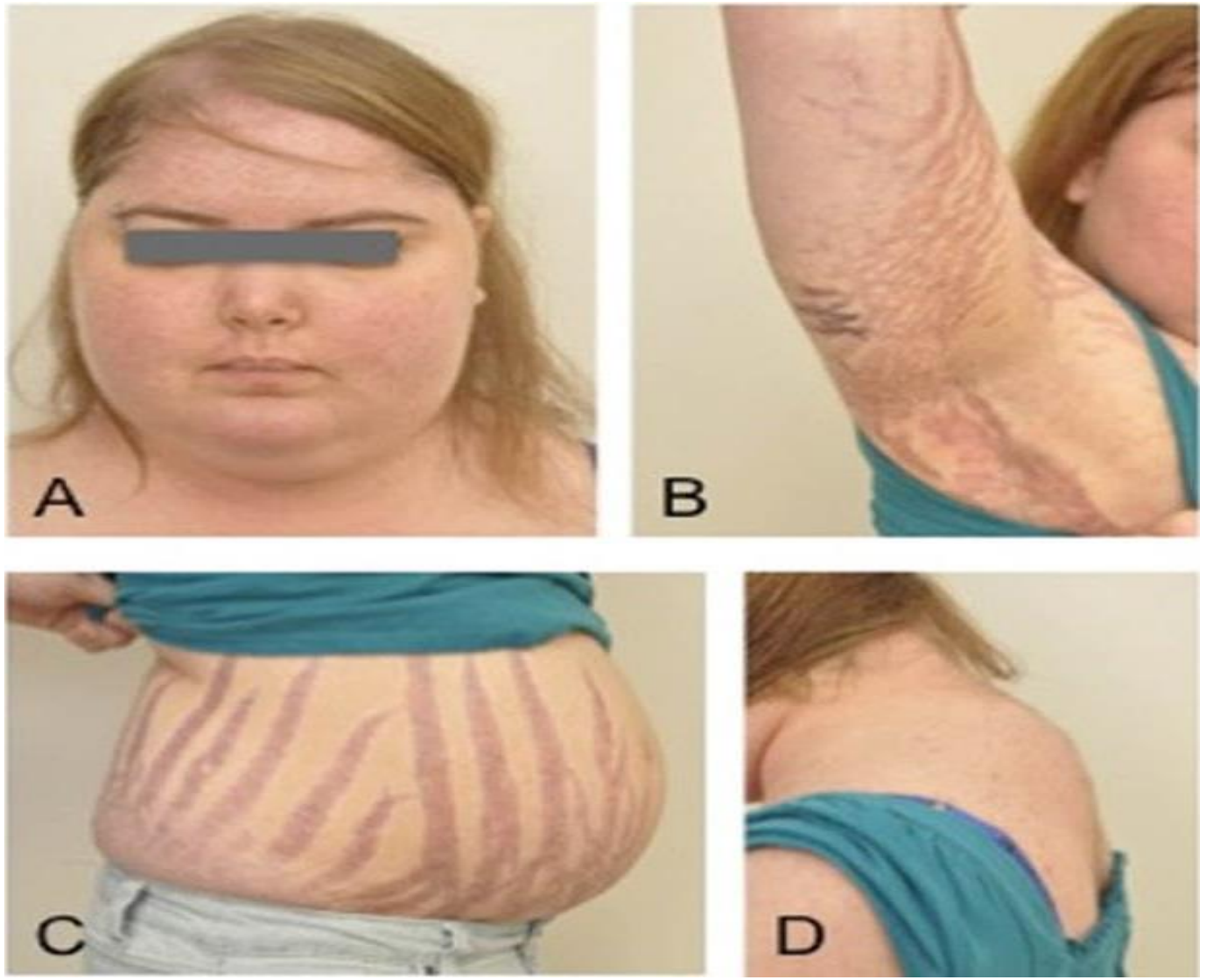
4- Adrenolytic agents: Mitotane

- Describe the image :
  increased facial hair in female(hirsutism)
- Diagnosis :
  cushing dyndrome
- Mention # of symptoms
  - as the first case
- Mention other physical finding:
  - as first case
- Mention 3 diagnostic test used to diagnose this patient
  as first case
- Mention imaging study far this patient
  - as first case

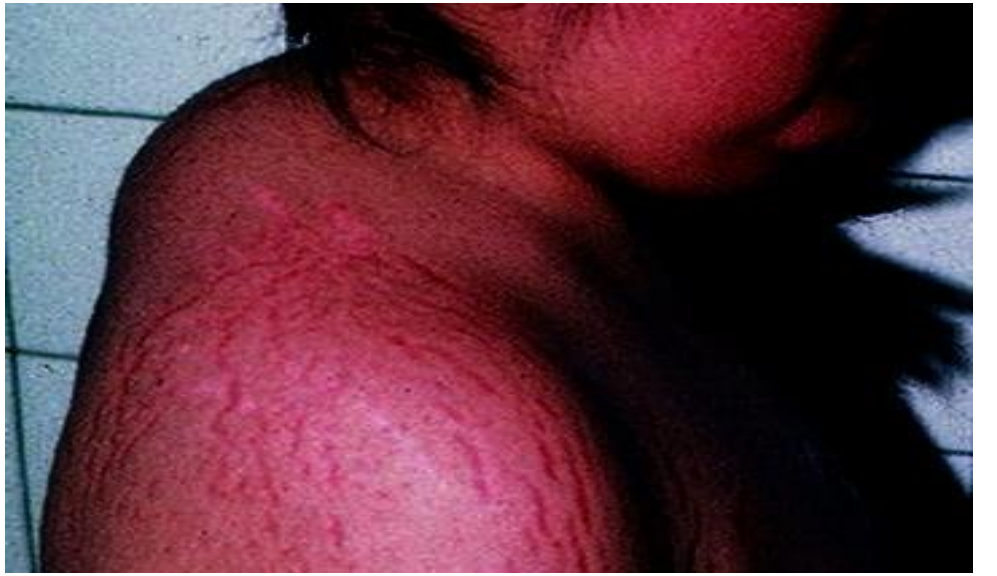
- Medication
  -as previous case



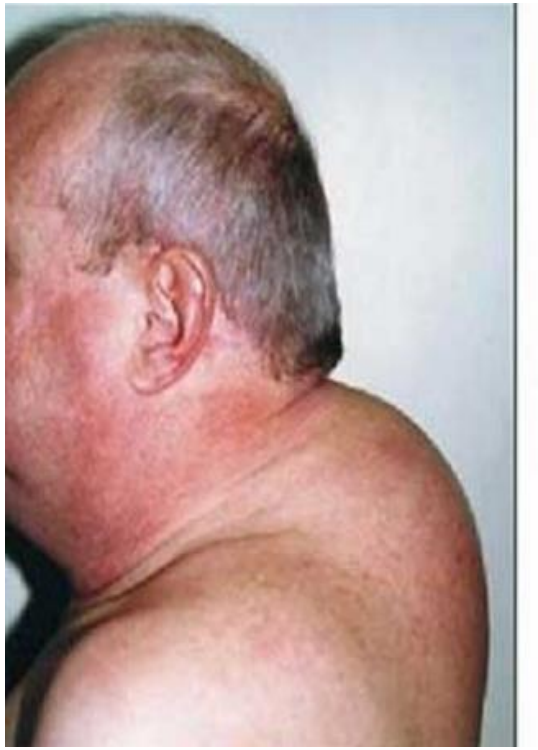
•B,C : striae rubra due to central obesity and impaired collagen synthesis

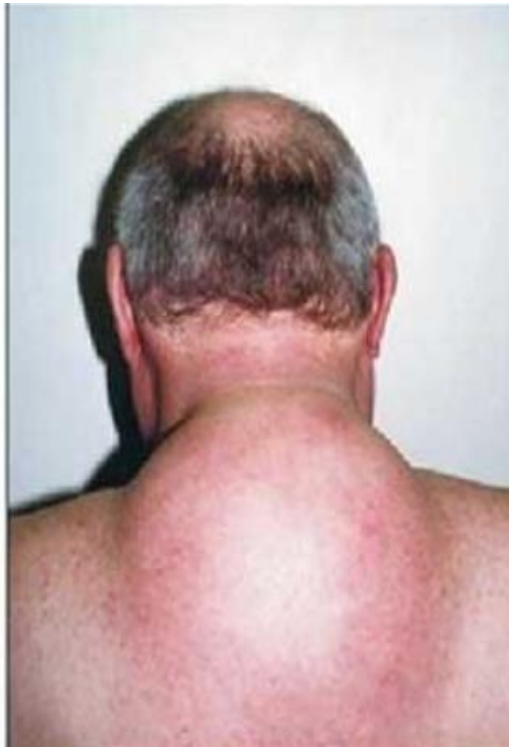


- describe image :
  - striae rubra
  - Buffalo hump
    high cortisol cause redistribution of fat from periphery to center of body
  other questions as first case

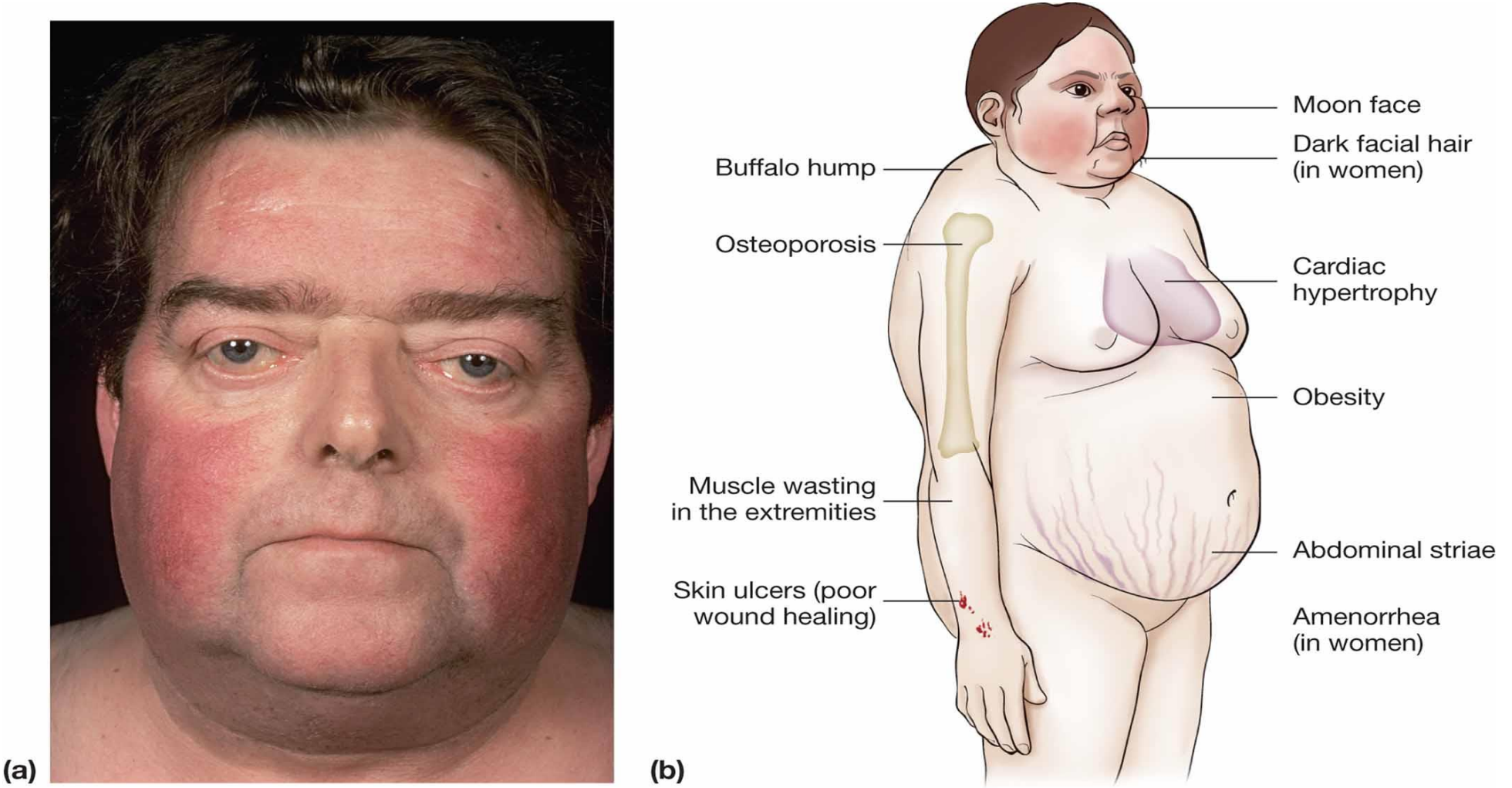


- Buffalo hump





- describe : Facial plethora ; due to degradation and atrophy of epidermis and subcutaneous connective tissue so vasculature become more obvious



- History
- Hx of

  1- weight gain (central obesity)

  2- skin thinning(easily bruising, stretch marks)

  3- difficulty combing hair , climbing stairs …etc

  due to proximal muscle weakness

  4- Menstrual irregularities, amenorrhea, infertility, and decreased libido in females

  5- decreased libido and impotence

  6- new-onset or worsening HTN , DM

  7- delayed wound healing , recurrent infection

  8- pathological fracture due to osteoporosis

**4&5 both due to inhibition of LH & FSH pulsatile secretion in both sexes

•Psychological hx :

1- depression

2- cognitive dysfunction

3- emotional lability

- Tumor specific history :

- ACTH-producing pituitary tumor: Headaches, polyuria, nocturia, visual problems (bitemporal hemianopia) , or galactorrhea
- adrenal carcinoma: Rapid onset of symptoms of hyperandrogenism presenting as virilization in women or feminization in men

- **Physical finding**
- Obesity :
  1-moon face
  2- buffalo hump
  3- supraclavicular fat pads
  4- waist-to-hip ratio;
  > 1 in men
  > 0.8 in women
- Skin :
  1- facial plethora
  2- violaceous striae > 0.5 cm
  3- ecchymosis, telangiectasia , purpura
  4- lanugo facial hair
  5- hirsutism and male pattern balding in female & steroid acne
  6- acanthosis nigricans

- CVS & renal :

1- possibly edema due to Na & water retention

- Endocrine

1- galactorrhea
2- slow deep tendon relaxation due to hypothyroidism
3- decreased testicular volume in male

- MSS :

1- proximal muscle weakness
2- pathological fracture and kyphosis
3- height loss

- Neuro :

1- bitemporal hemianopia
2- blurred vision

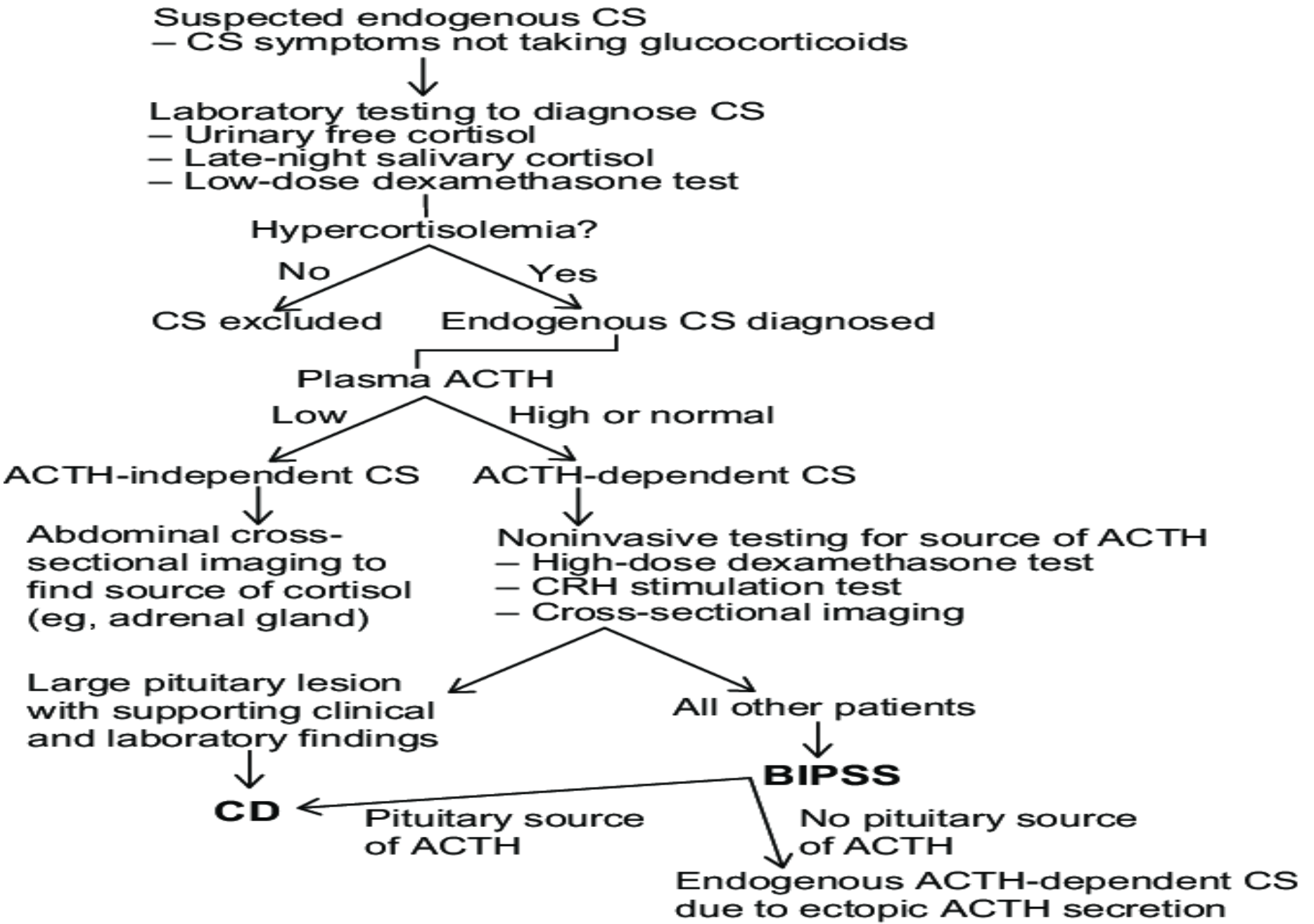
- Adrenal crisis
  1- hypotension
  2- abdominal pain
  3- vomiting
  4- mental confusion
- * Other findings include hypoglycemia, hyperkalemia, hyponatremia, and metabolic acidosis

- **DDx**
- differentialطبعا حسب الكيس كيف تيجي بنختار
- Alcoholism
- Anorexia Nervosa
- Bulimia Nervosa
- Concurrent ritonavir and inhaled fluticasone in patients with HIV - Interferes with dexamethasone suppression testing (false positive)
- Depression
- Obesity
- Pseudo-Cushing Syndrome
- Psychiatric Illness

Work up

- Four methods are accepted for the diagnosis of Cushing syndrome:
  1- urinary free cortisol level -24hrs
  2- low-dose dexamethasone suppression test
  3- evening serum and salivary cortisol level
  4- dexamethasone–corticotropin-releasing hormone test.

- BIPSS: bilateral inferior petrosal sinus sampling



- **Treatment**
- Medications :

  1-Somatostatin analogs: Pasireotide

  2- Adrenal steroid inhibitors: Metyrapone, ketoconazole, etomidate

  3- Glucocorticoid receptor antagonist: Mifepristone

  4- Adrenolytic agents: Mitotane

- Surgery : surgical removal of

  1- ectopic cortisol secreting tumor

  2- pituitary tumor

  3- adrenal tumor

- CUSHING SYNDROME

ACTH-independent (primary) :

1- adrenal cause

* primary adrenocortical neoplasm :
  - mostly adenoma
  - rarely carcinoma

* adrenocortical hyperplasia (very rare)
  - bilateral micronodular hyperplasia
  - macronodular hyperplasia

2- ectopic cortisol secretion like in ovarian Ca cases has been reported

- Female to male ratio = 5:1
  except for lung ca as cause of Cushing syndrome in which there is a male predominance

- Peak incidence : 25- 40 years
  except for ectopic cushing due to lung ca
  peak incidence is in older age

- ACTH-independent : characterized by LOW ACTH hormone due to negative feedback of high serum cortisol to corticotroph cells in the anterior pituitary gland

- ACTH-dependent (secondary) :
  * anterior pituitary tumor (more common)
    (cushing disease)
  * Non pituitary source of ACTH
    - small cell lung ca (aot cell carcinoma)
    - carcinoid tumor
    - medullary thyroid ca
    - other neuroendocrine tumors

- Tertiary ACTH-dependent

- Ectopic corticotropin-releasing hormone (CRH) secretion leading to increased ACTH secretion comprises a very rare group of cases of Cushing syndrome. [4]

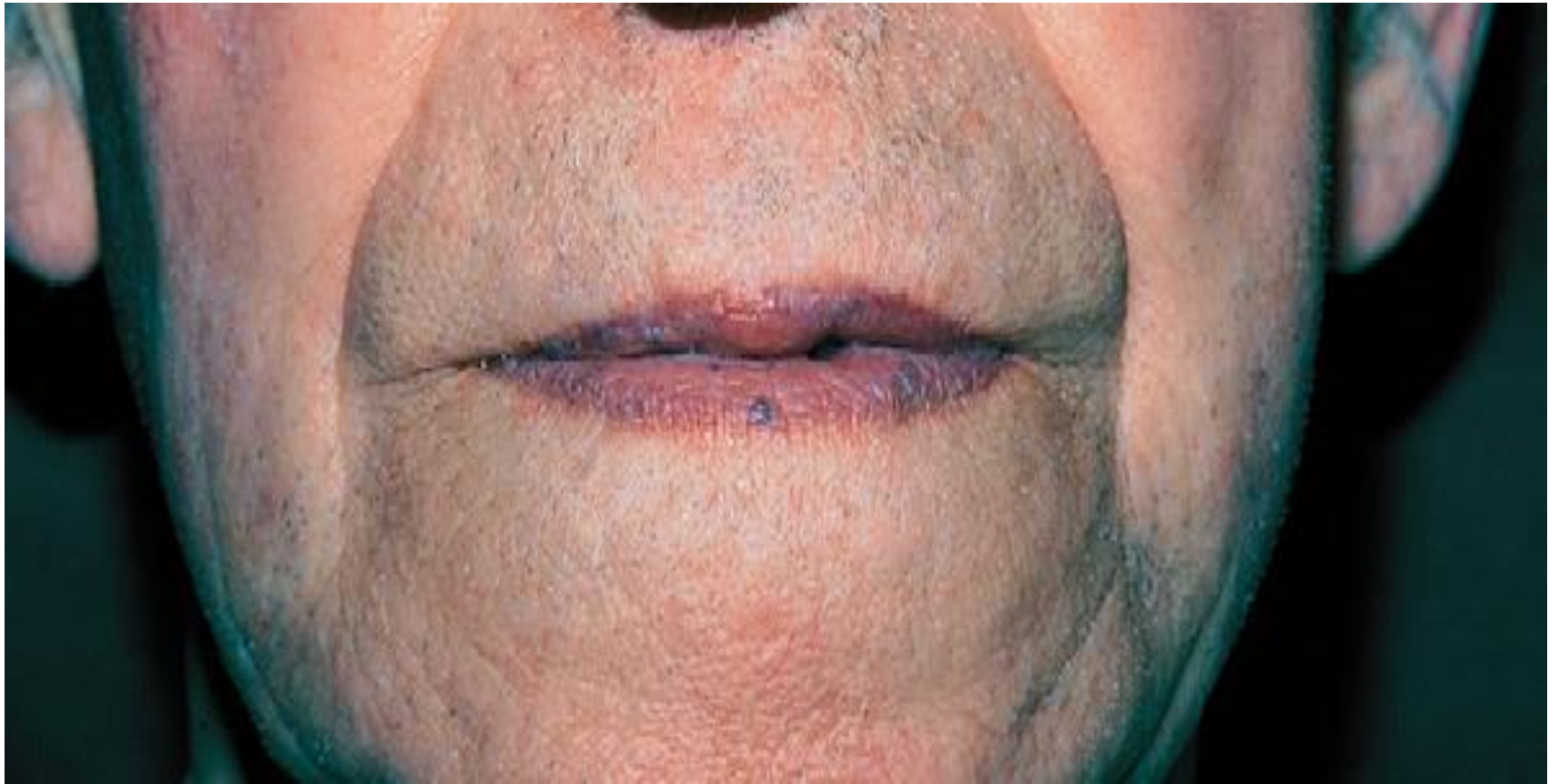
- Reference : https://emedicine.medscape.com/article/2233083-overview#a5

Macleod's pictures

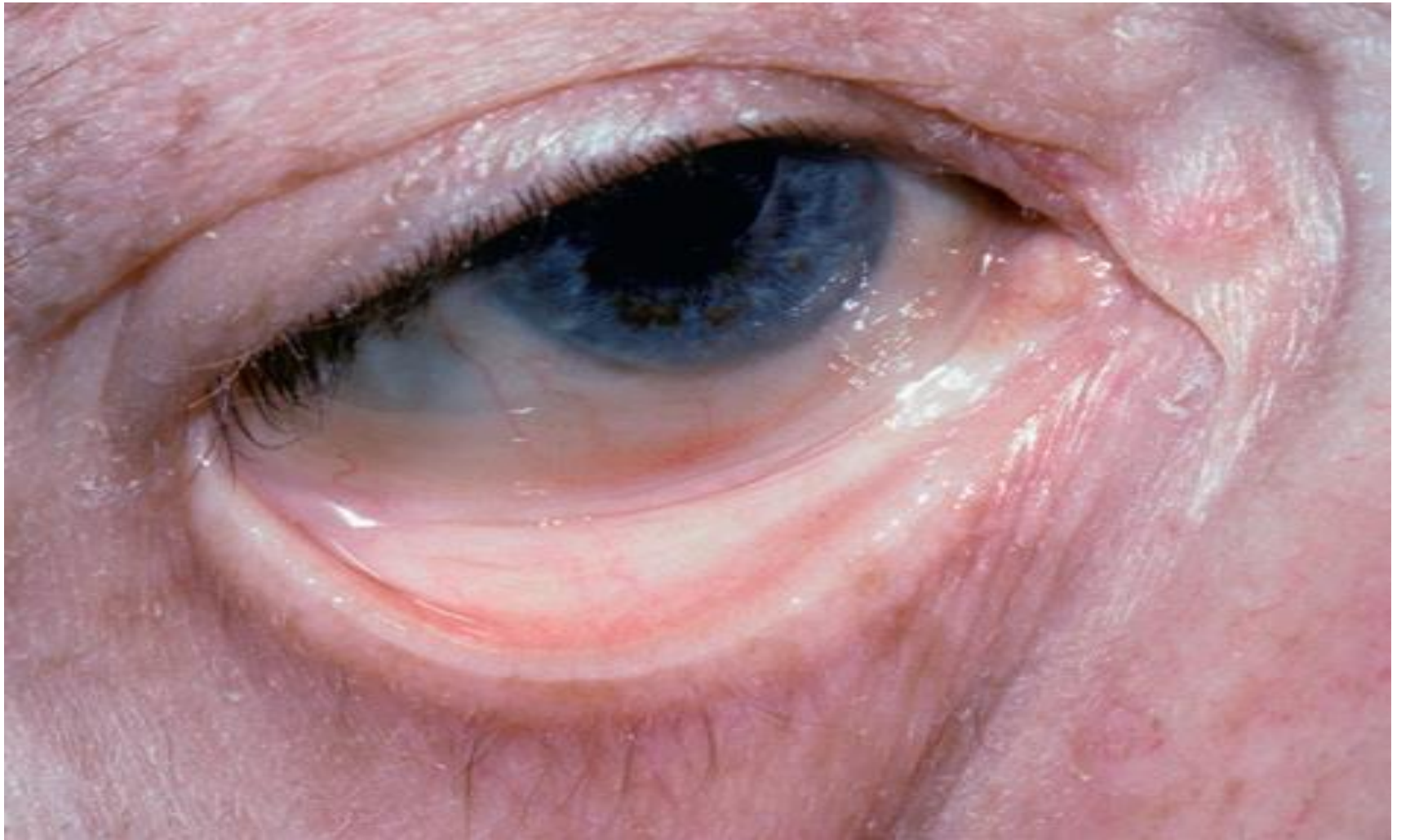
The General examination

Phenothiazine induced pigmentation

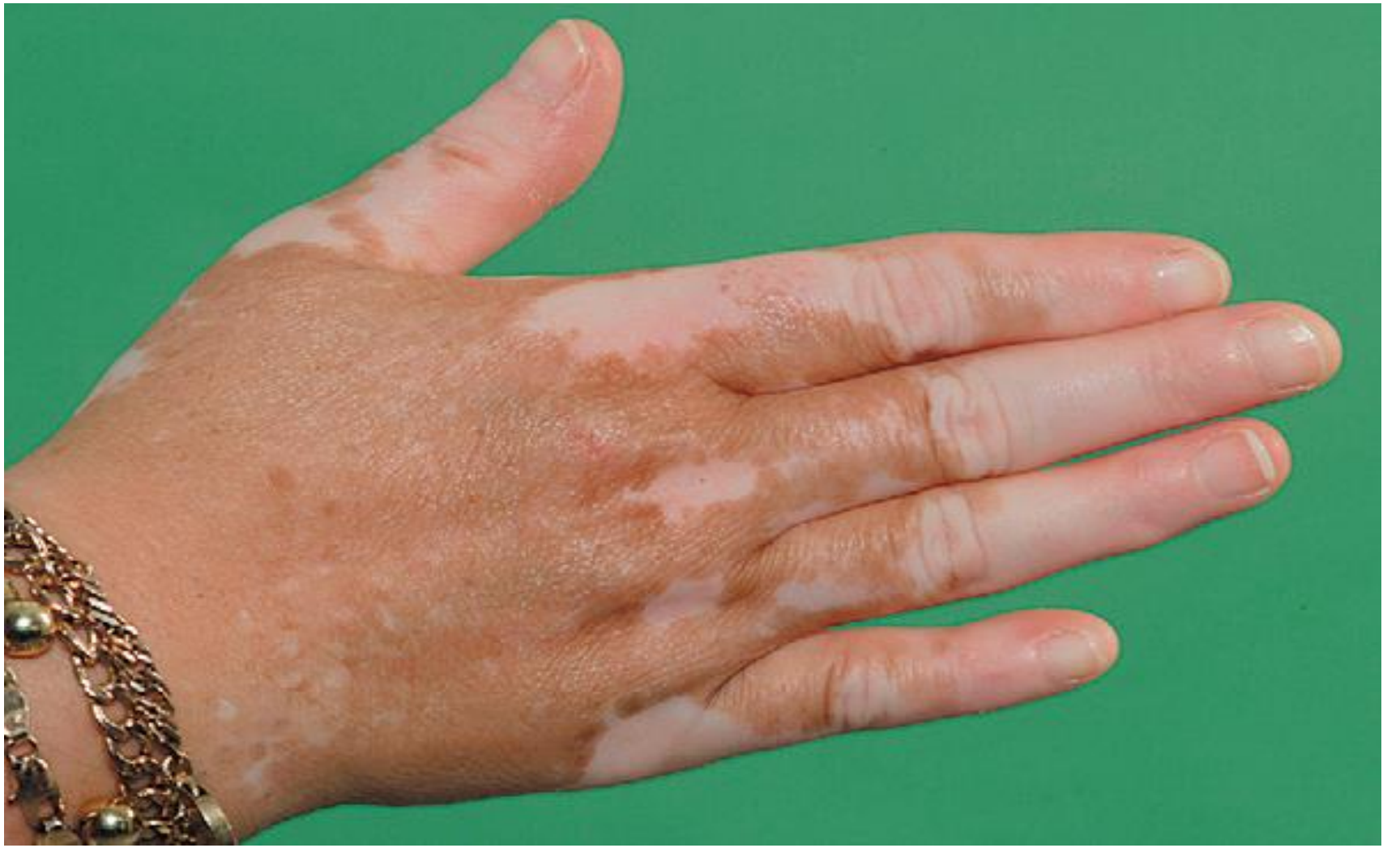
Central cyanosis of the lip



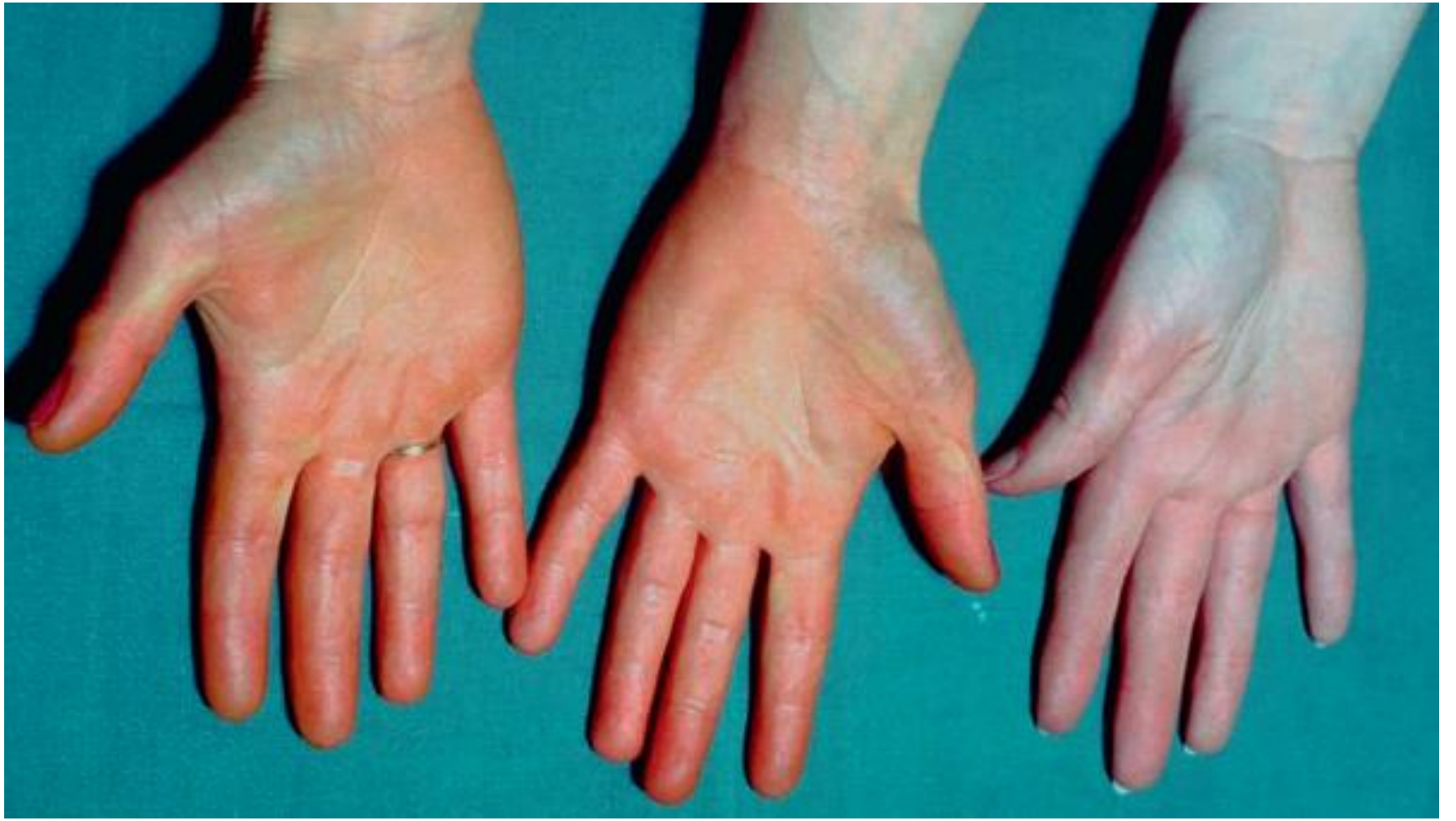
Conjunctival pallor



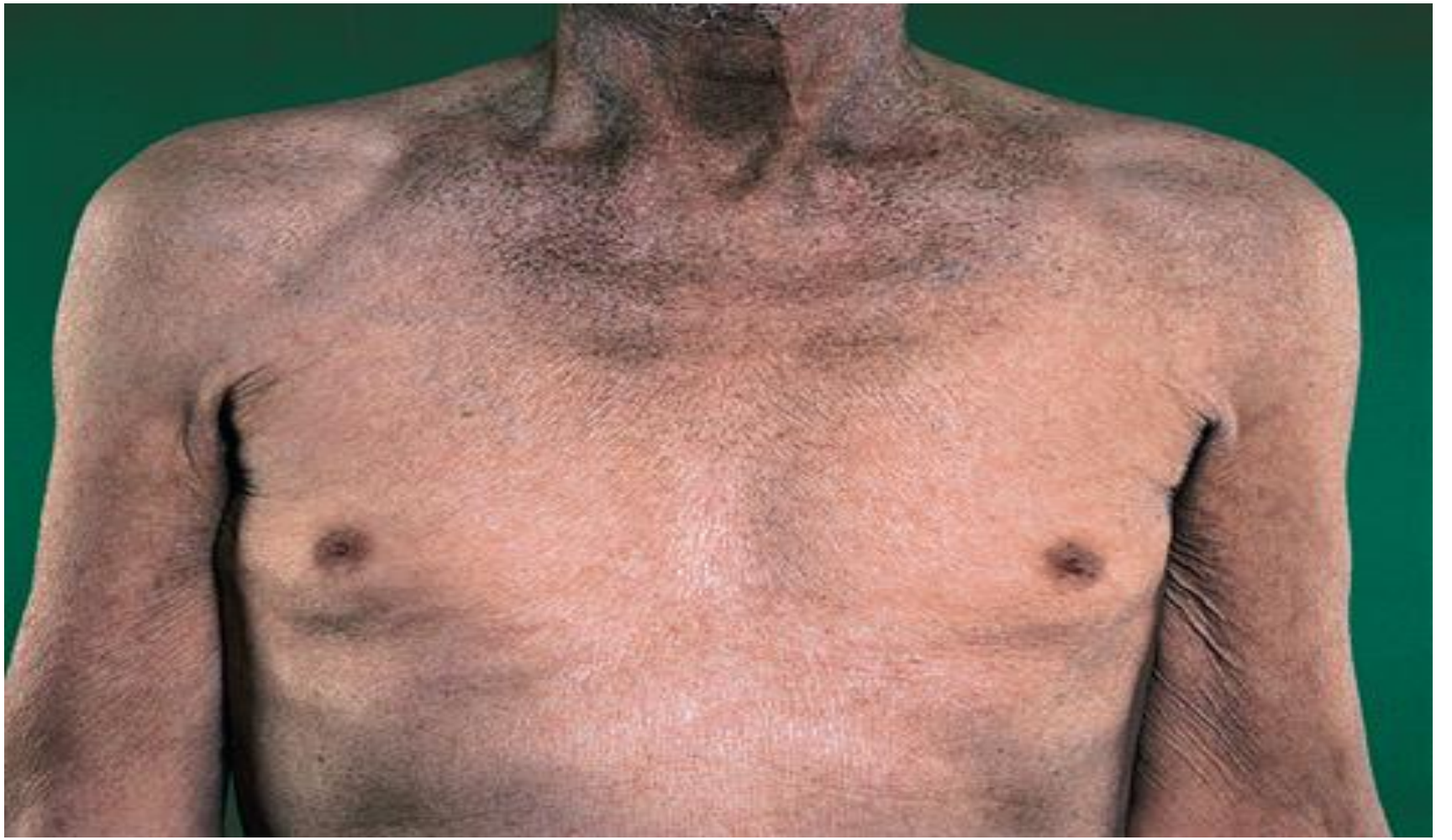
vitilligo



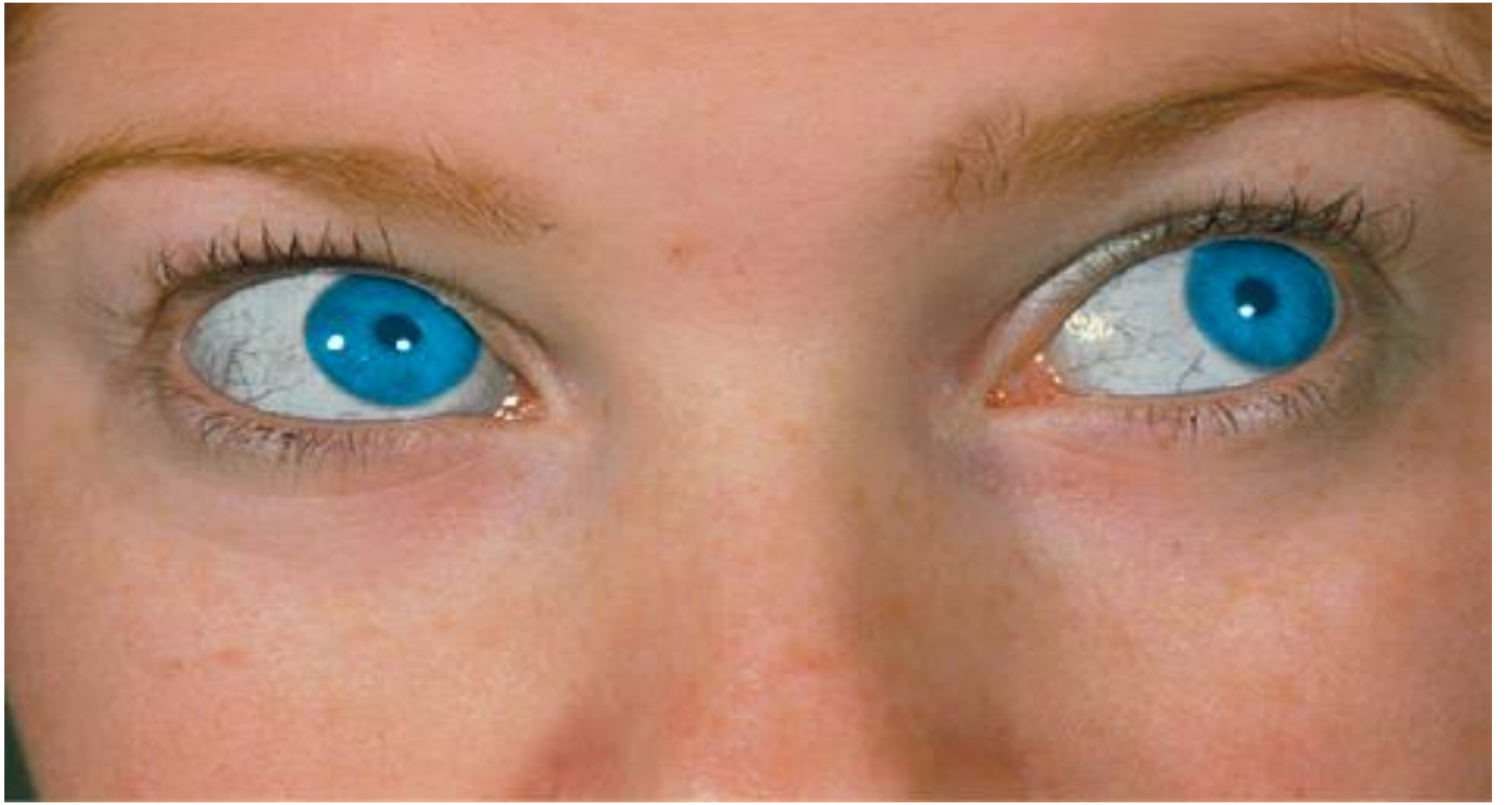
hypercarotenemia



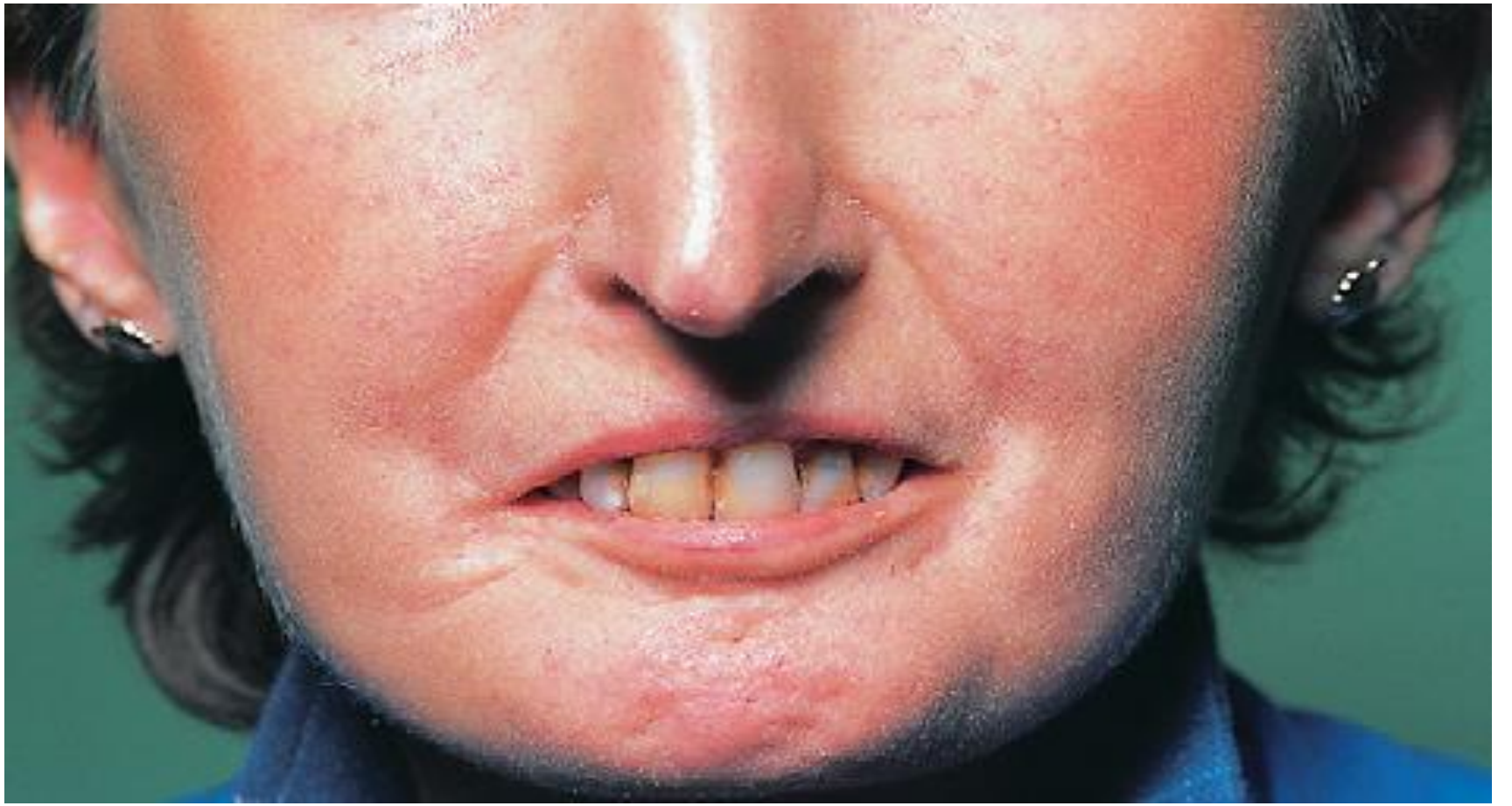
Hemochromatosis with skin pigmentation



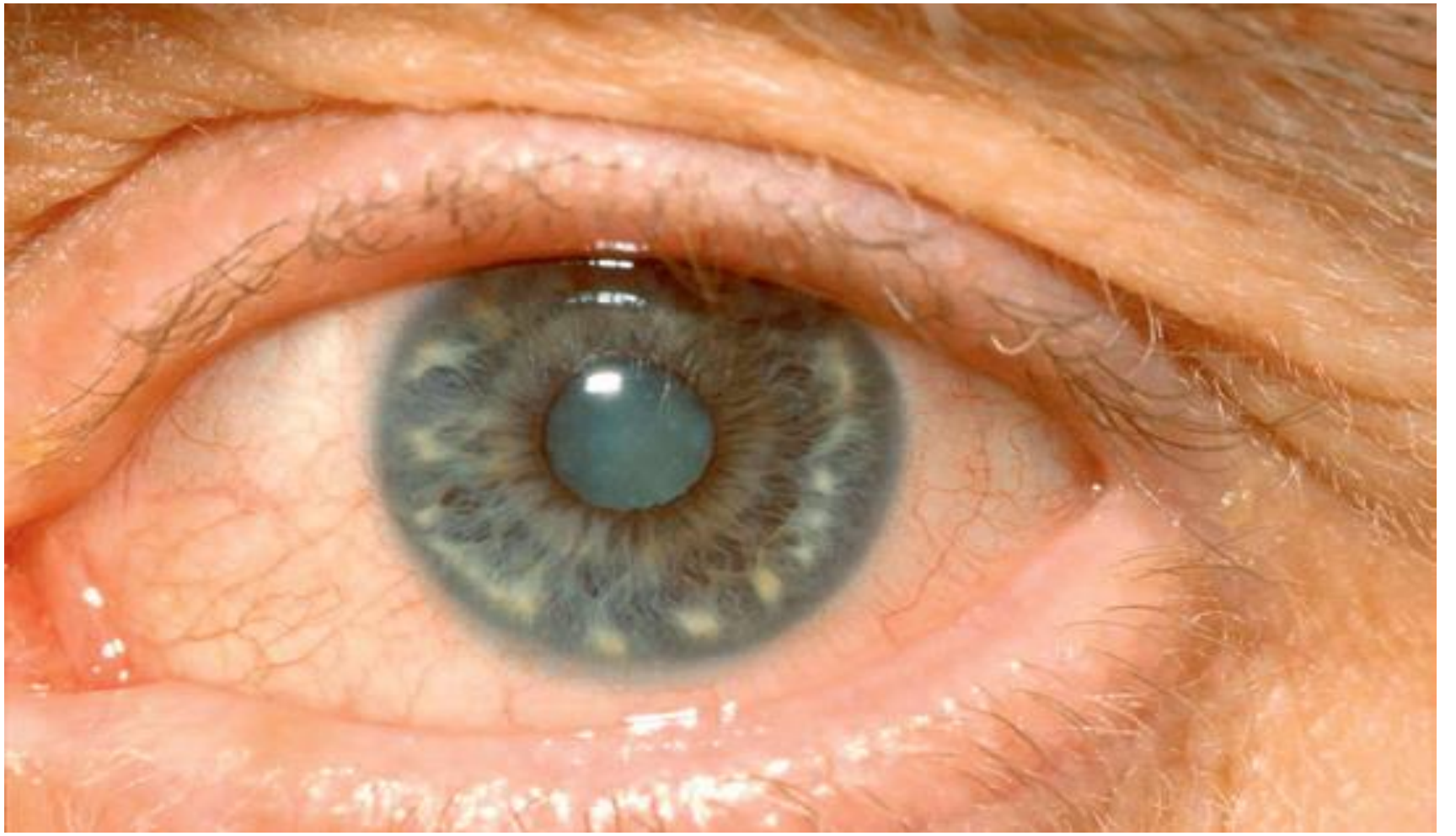
Blue sclera of osteogenesis imperfecta



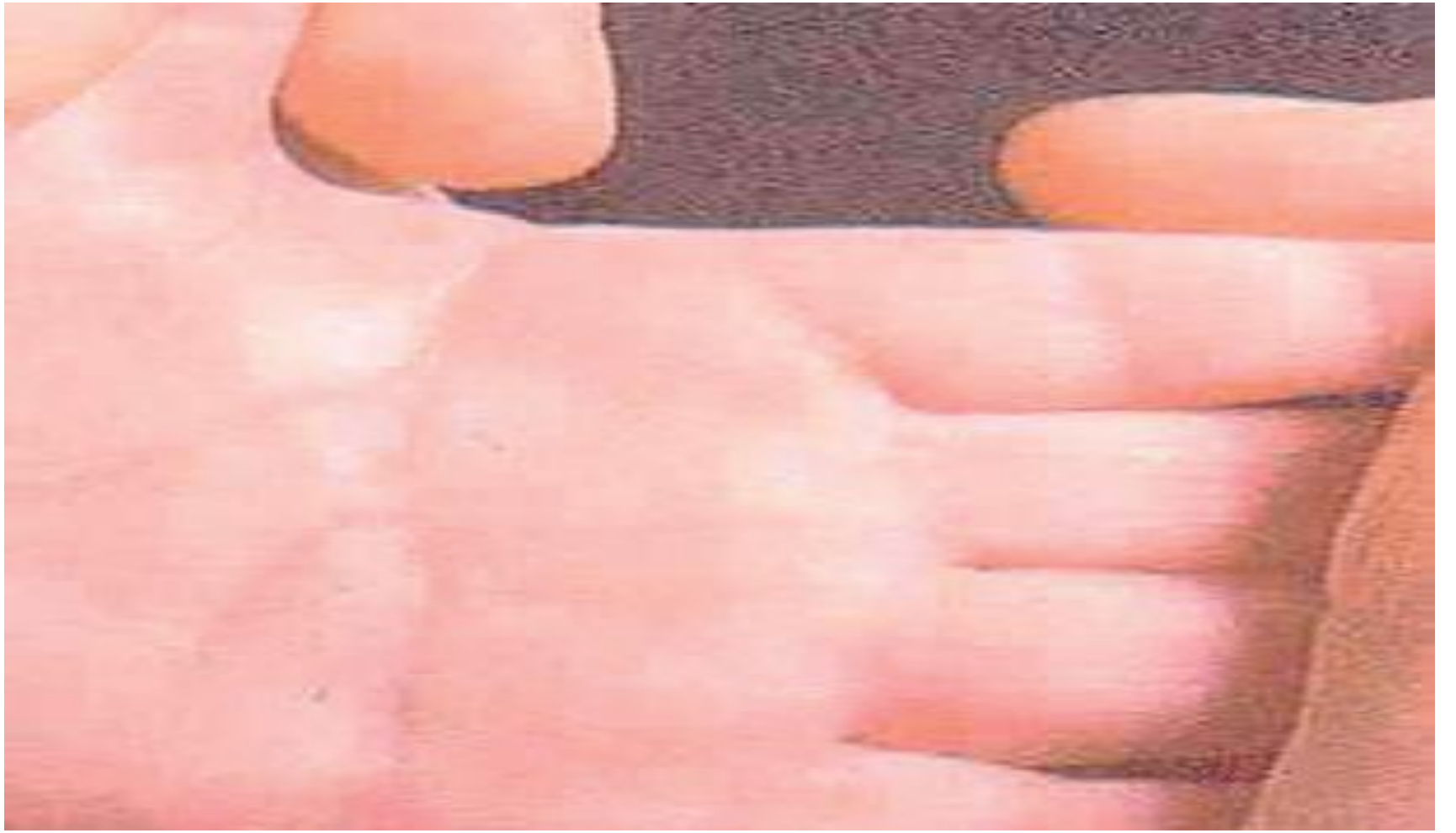
Systemic sclerosis



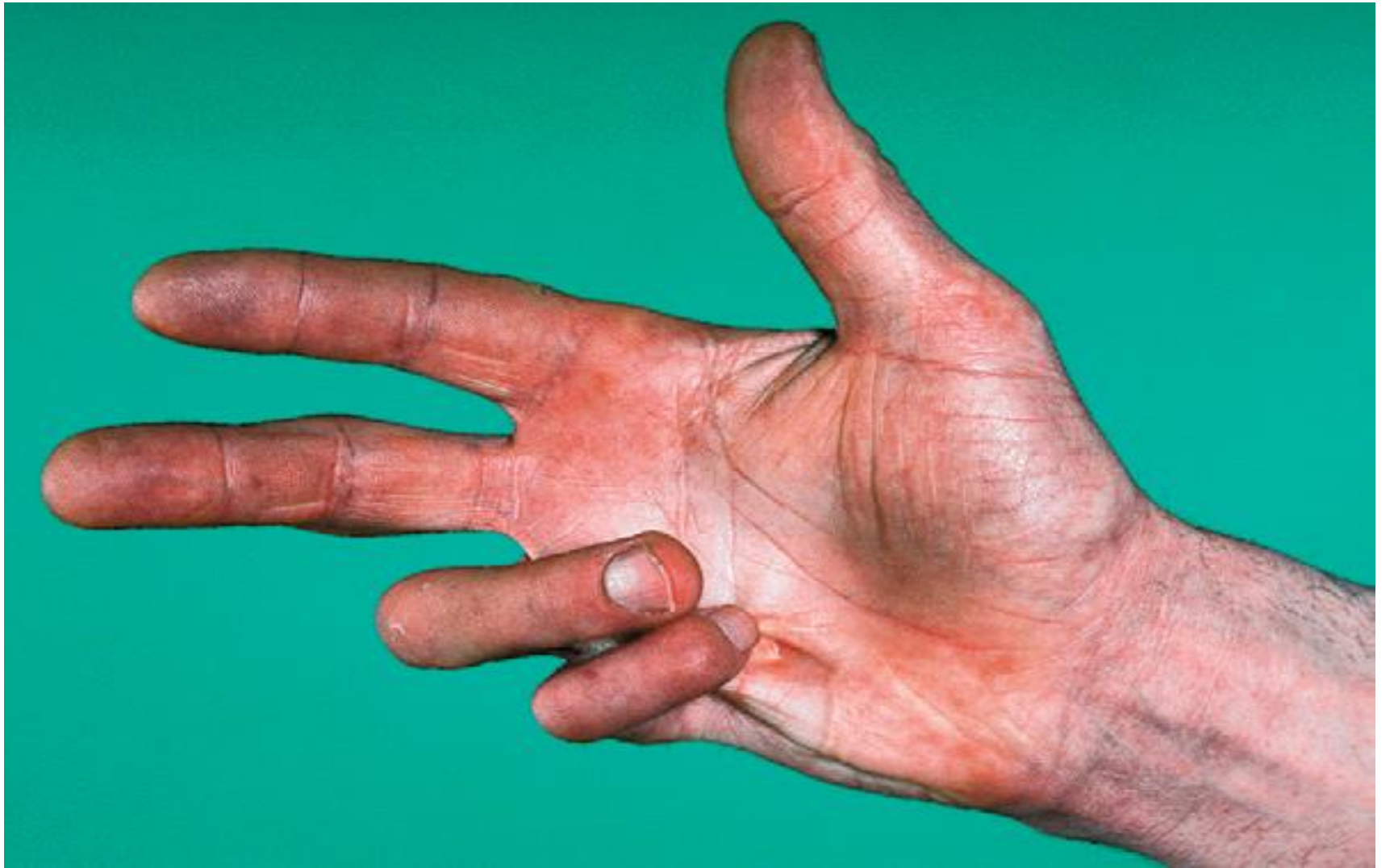
Brushfield spots: grey/white areas of depigmentation in the iris (DOWN



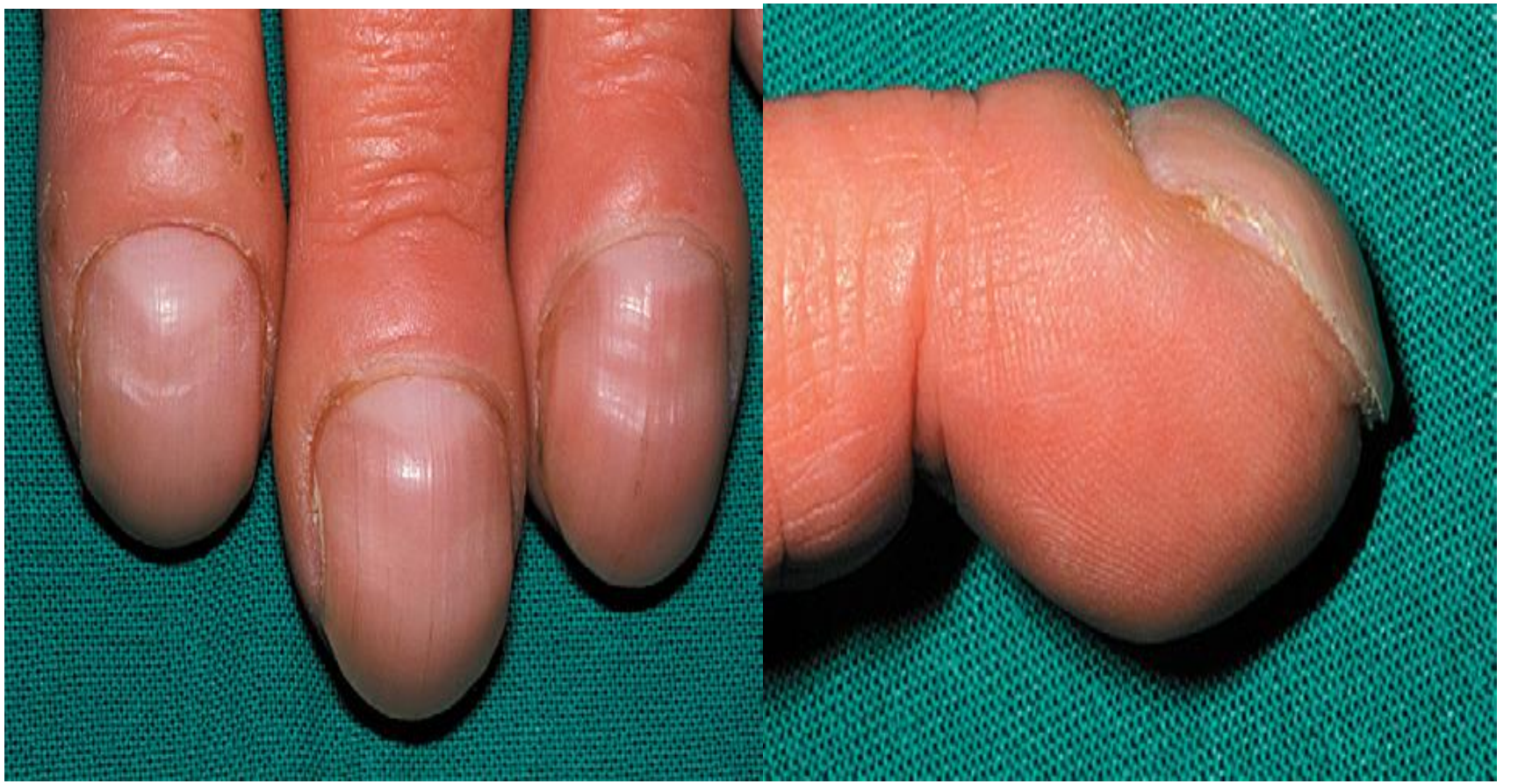
SINGLE PALMAR CREASE
(down syndrome)



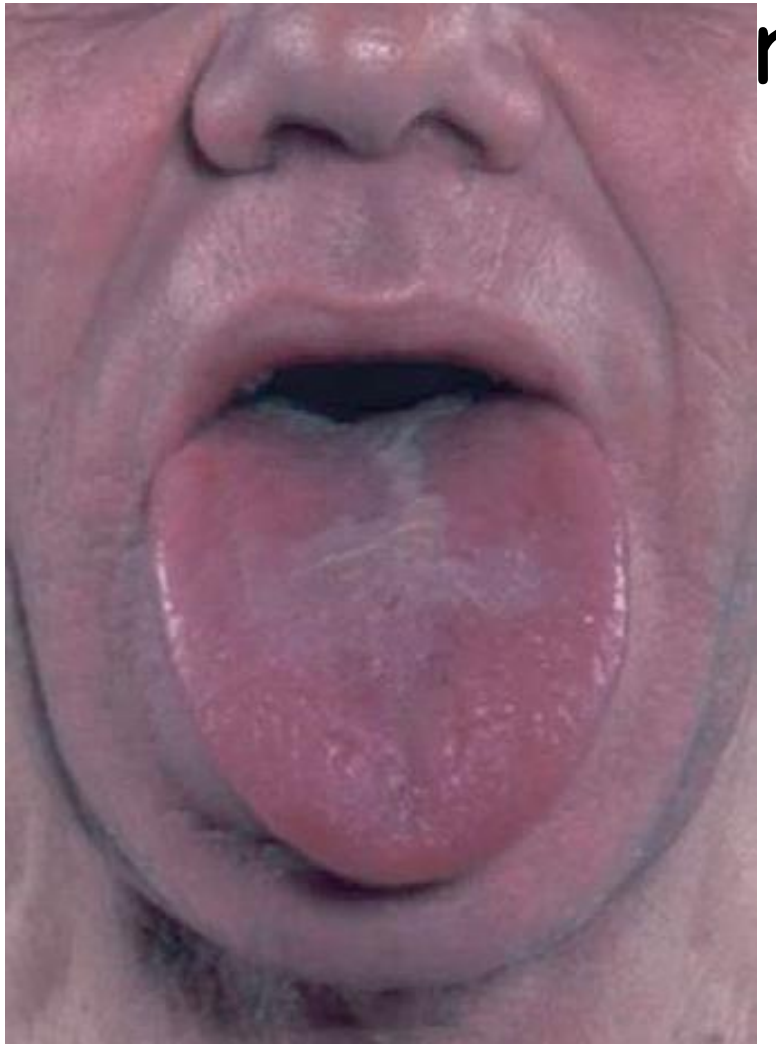
Duputyren contracture

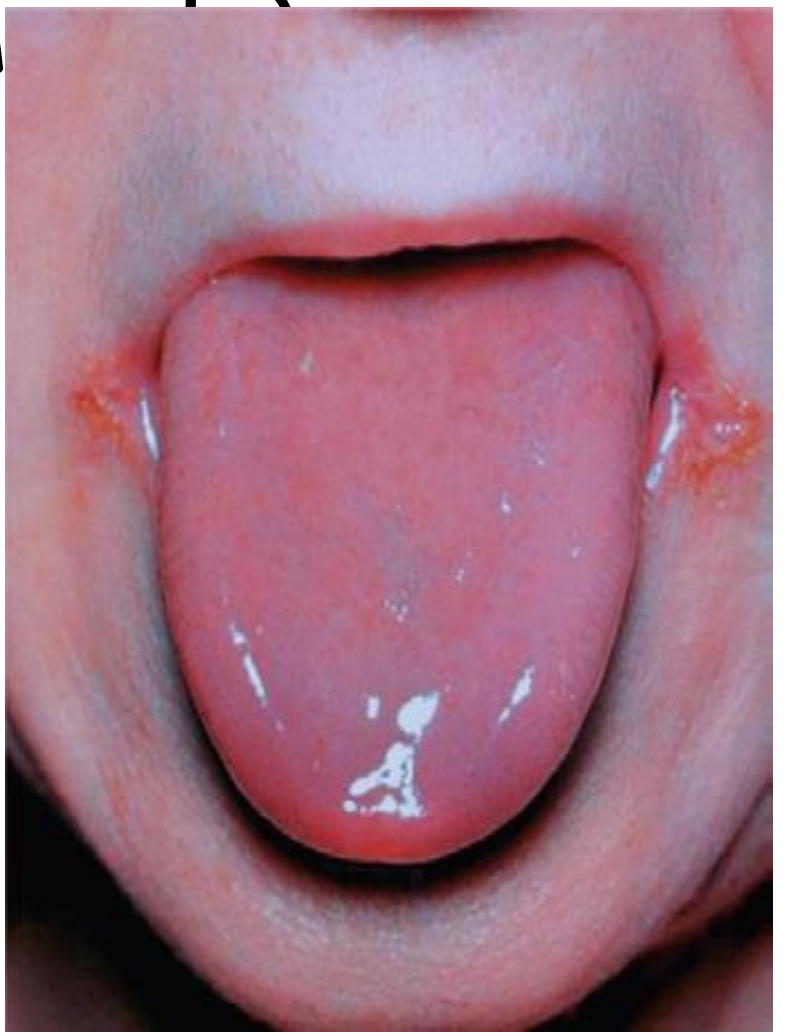


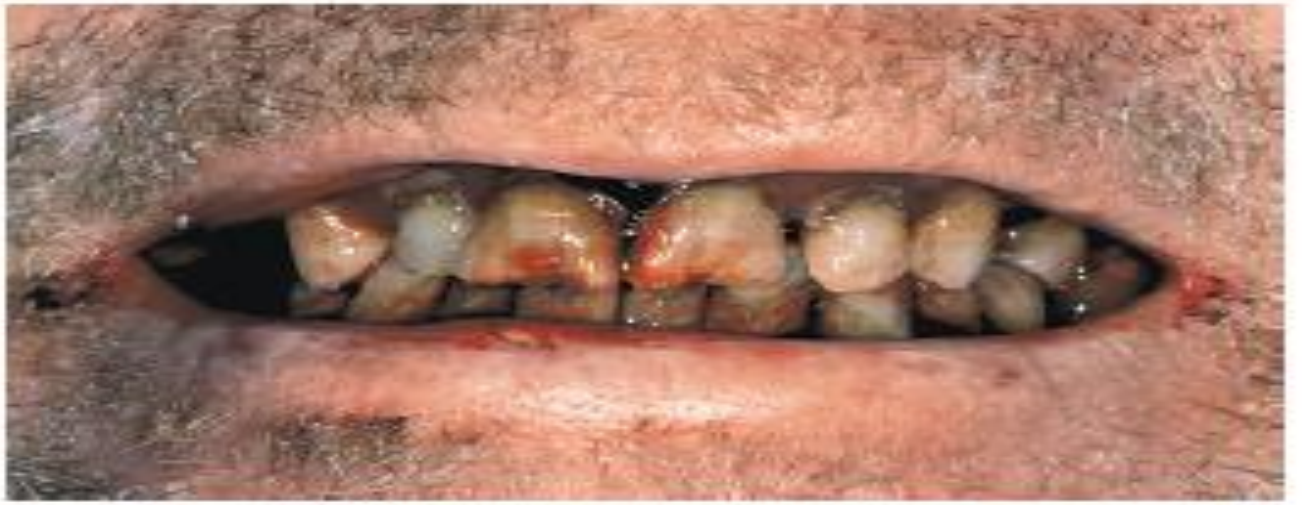
Clubbing anterior and lateral views



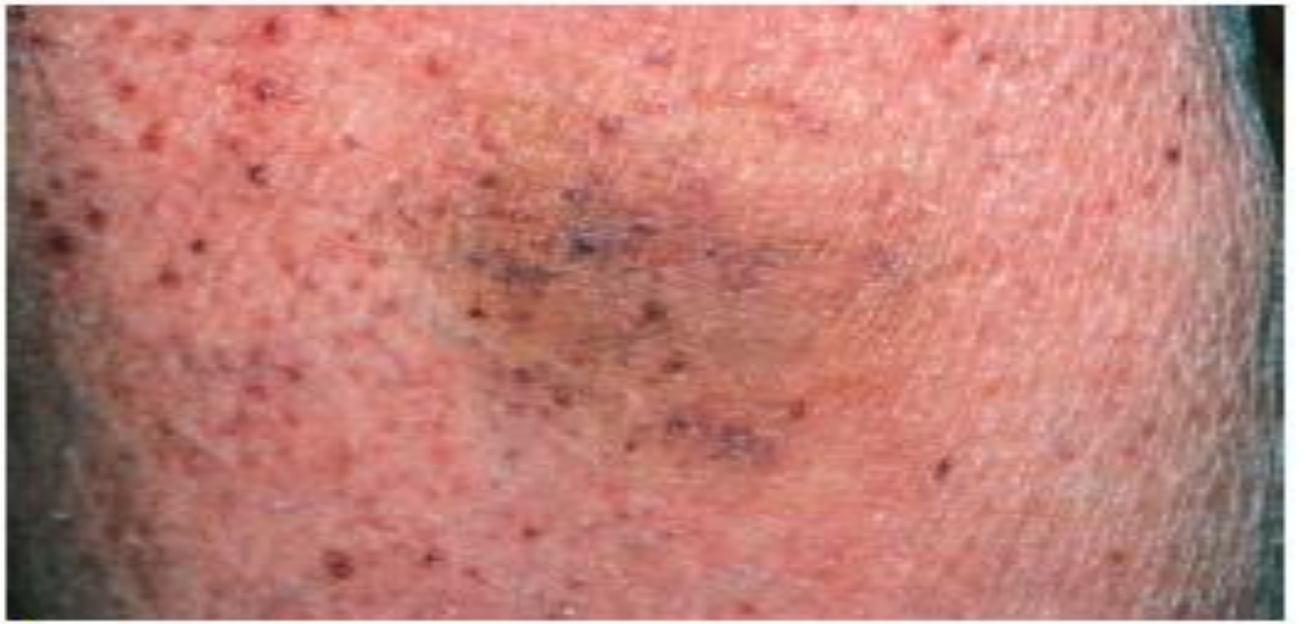
with angular stomatitis(iron deficiency) left(macroglossia of rom







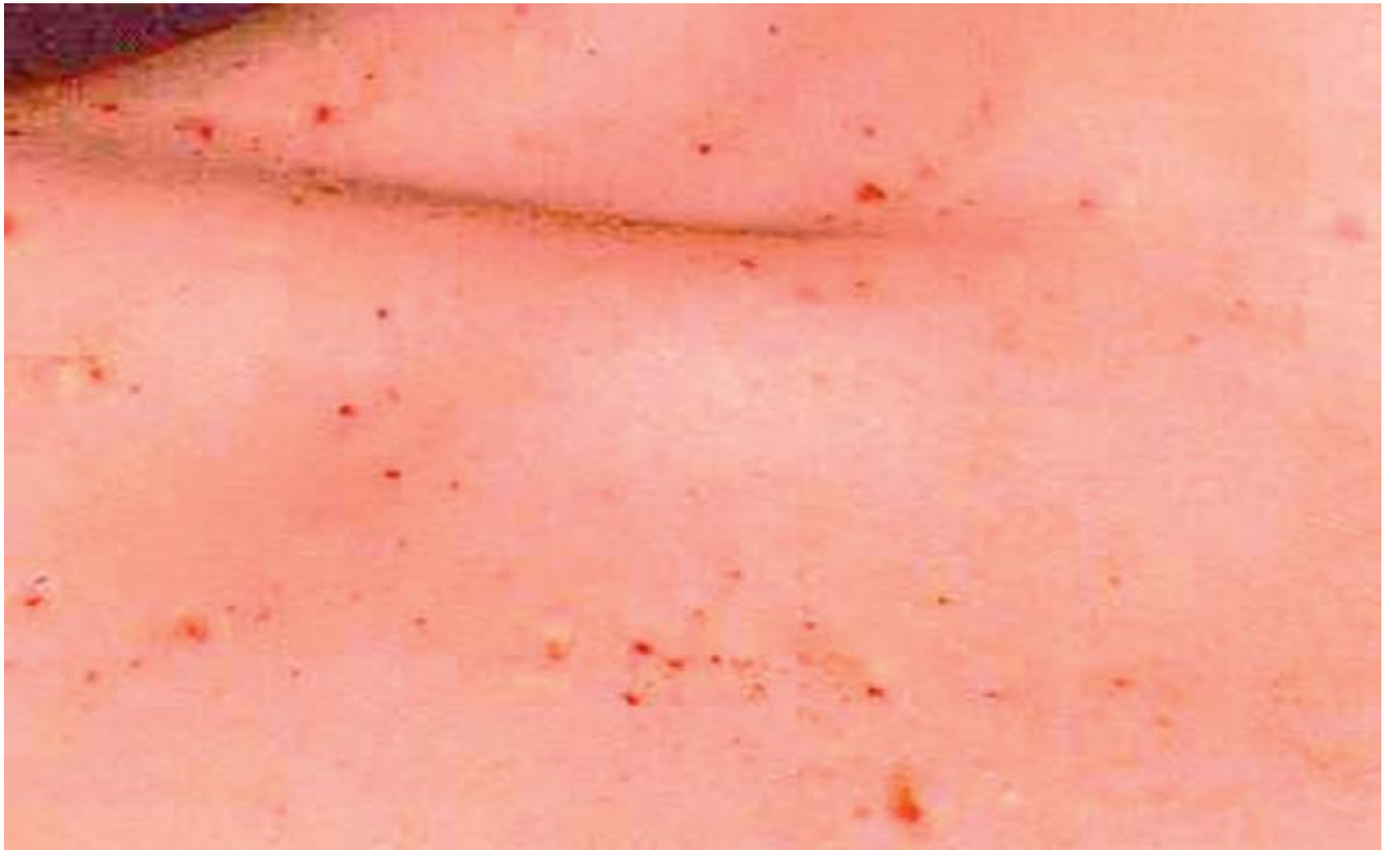
A

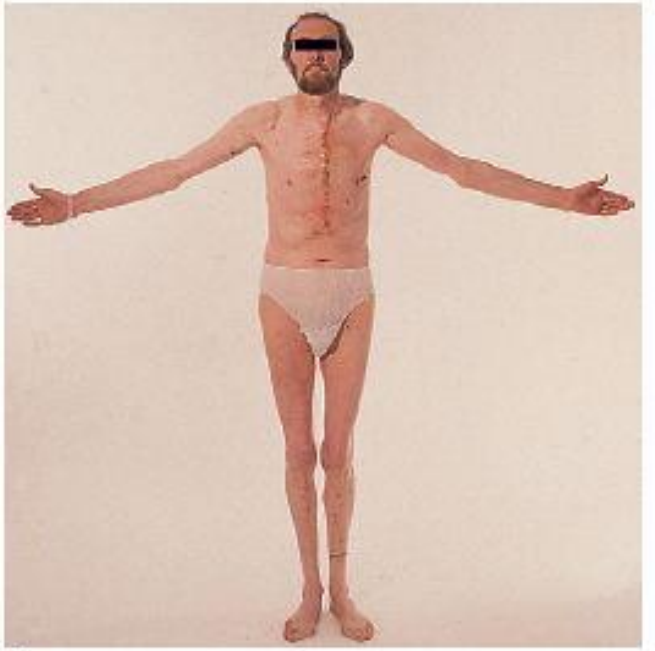


B

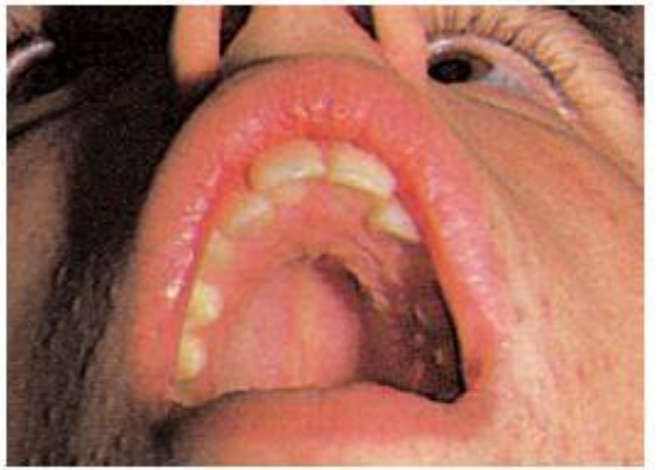
Fig. 3.26 Scurvy. (A) Bleeding gums. **(B)** Bruising and perifollicular haemorrhages.

petechiae

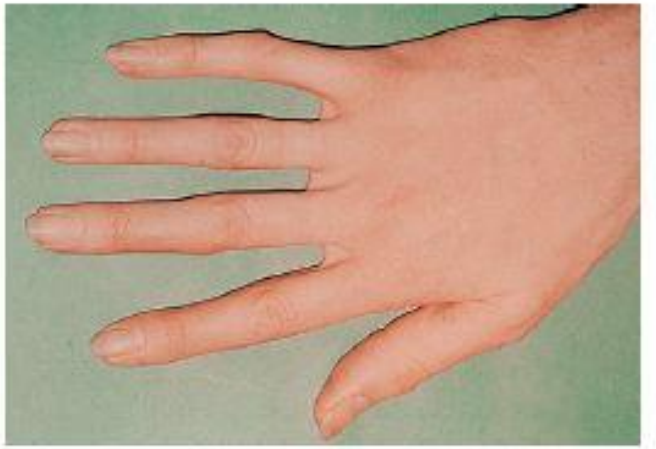




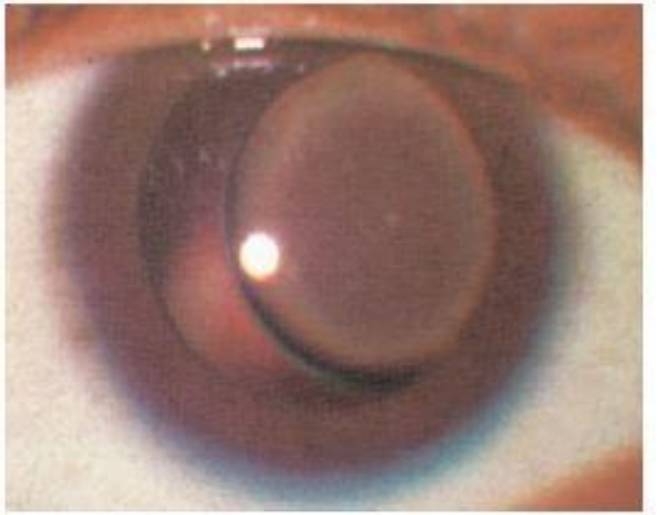
A



C



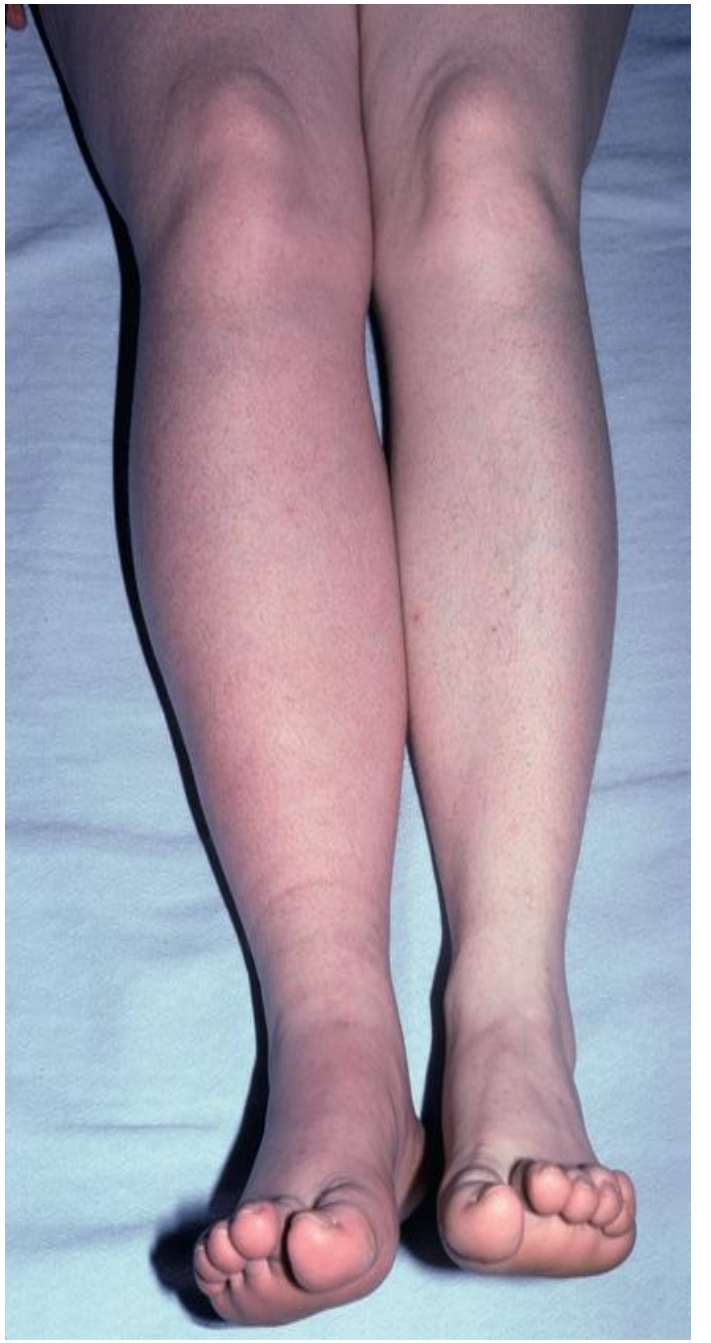
B



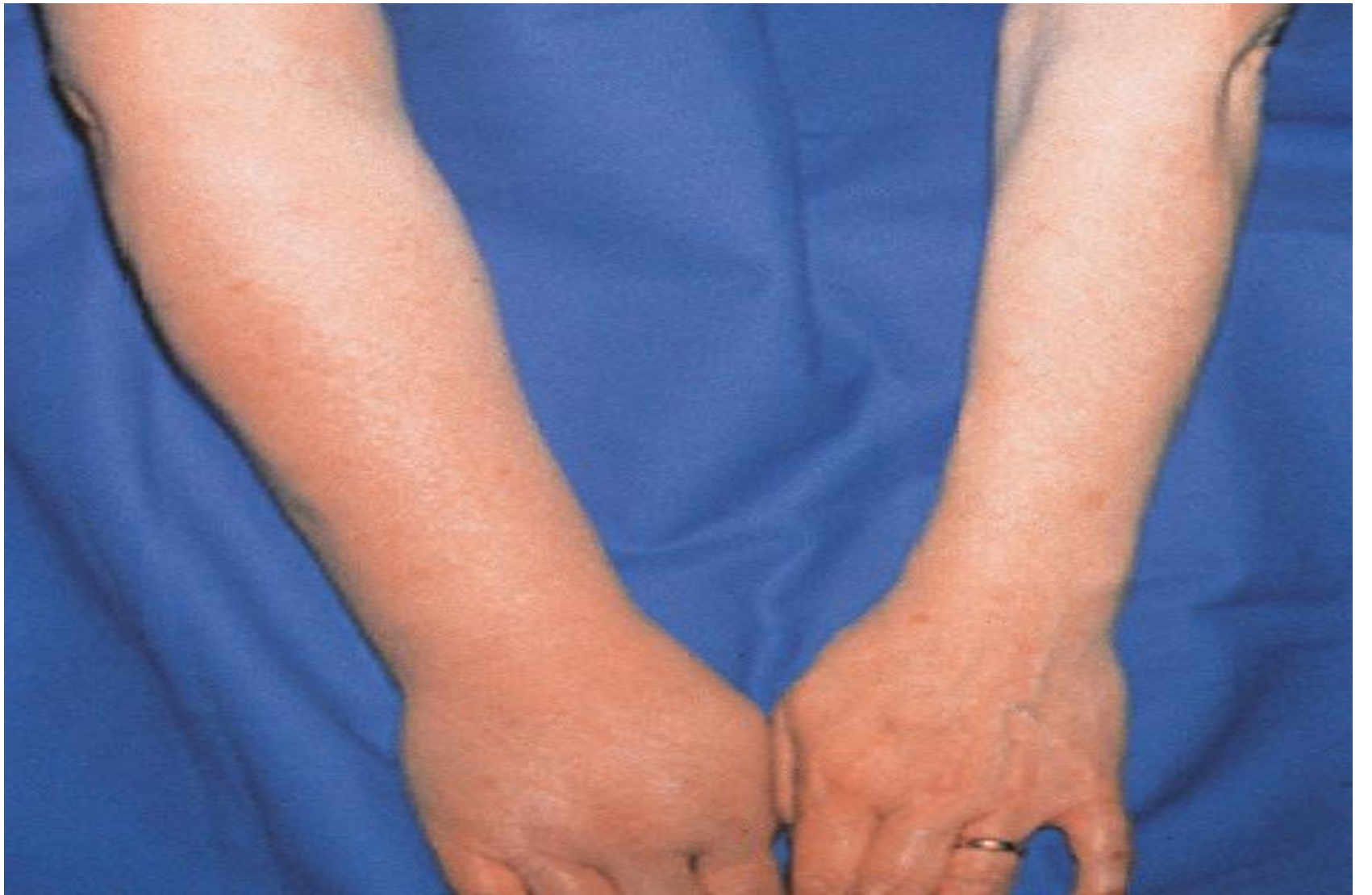
D

Fig. 3.28 Marfan's syndrome, an autosomal dominant condition. (A) Tall stature and reduced upper segment to lower segment ratio (note surgery for aortic dissection). **(B)** Long fingers. **(C)** High-arched palate. **(D)** Dislocation of the lens in the eye.

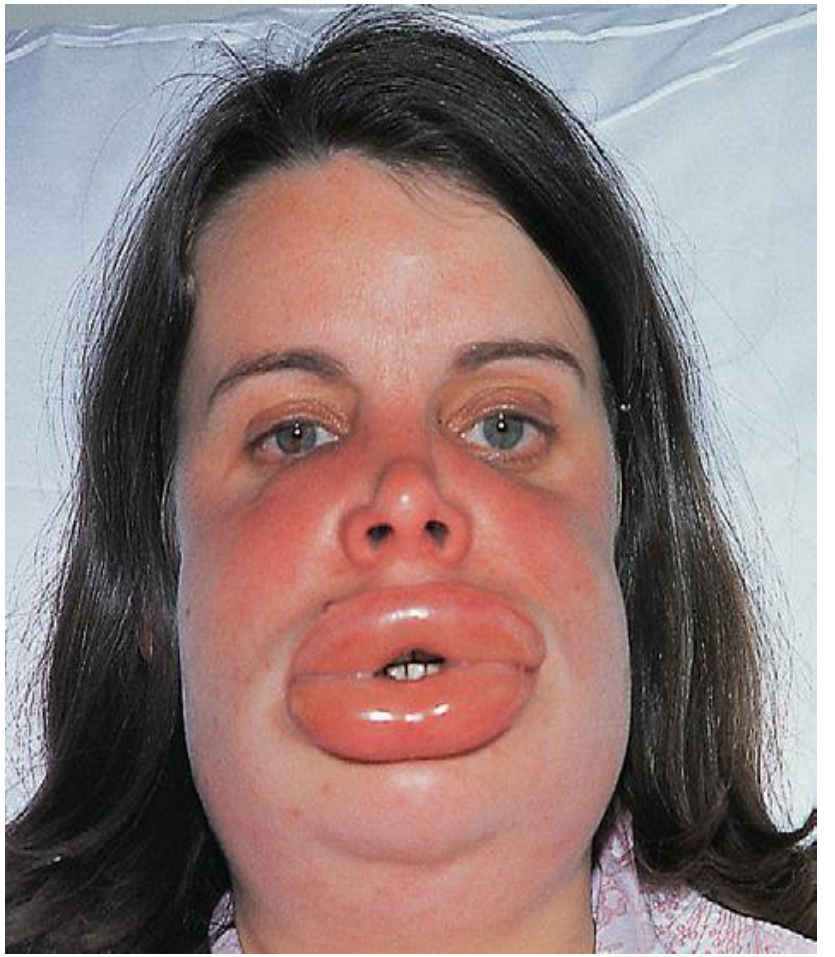
Swollen right leg, suggesting deep vein thrombosis or inflammation, e.g. soft-tissue infection or ruptured Baker's cyst.



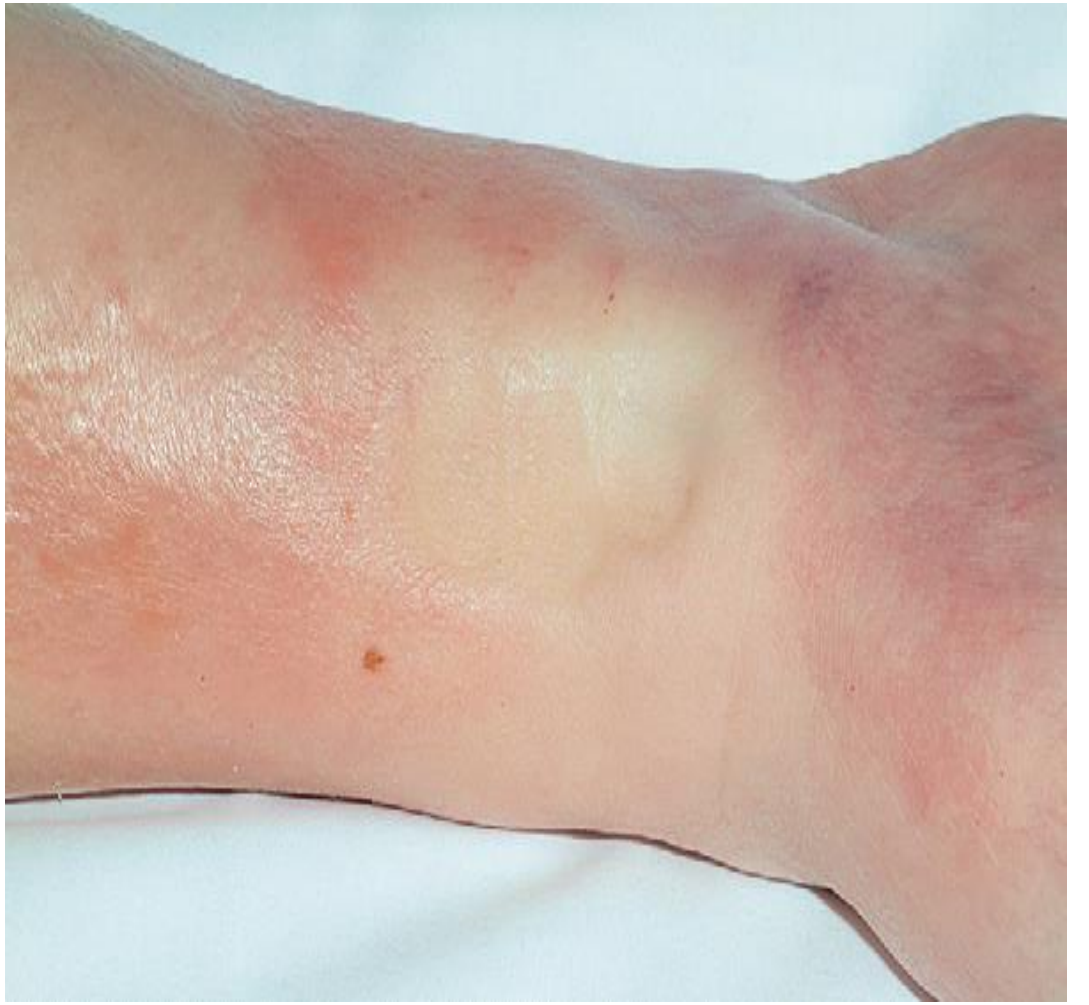
Lymphoedema of the right arm following right-sided mastectomy and radiotherapy.



angioedema

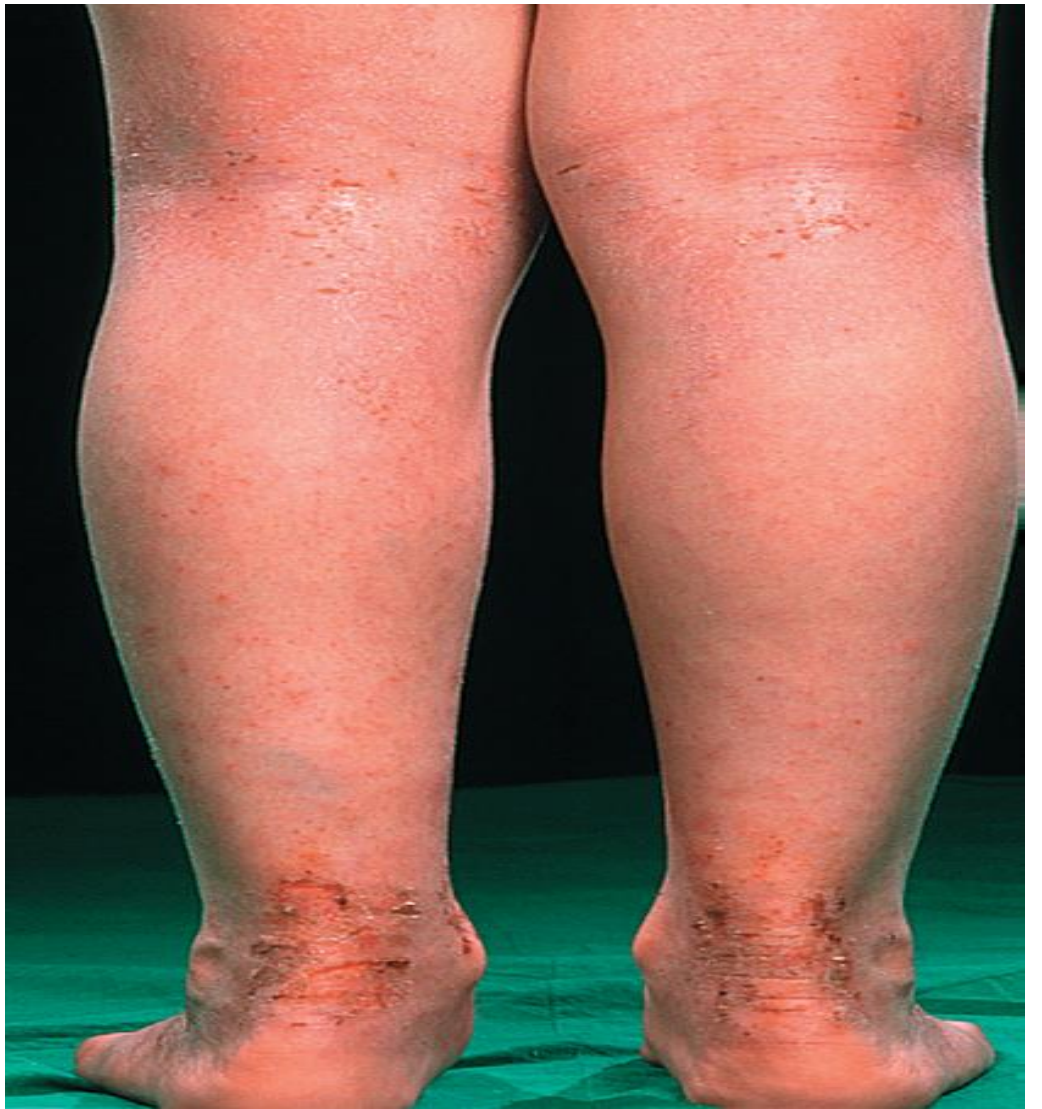


Pitting edema

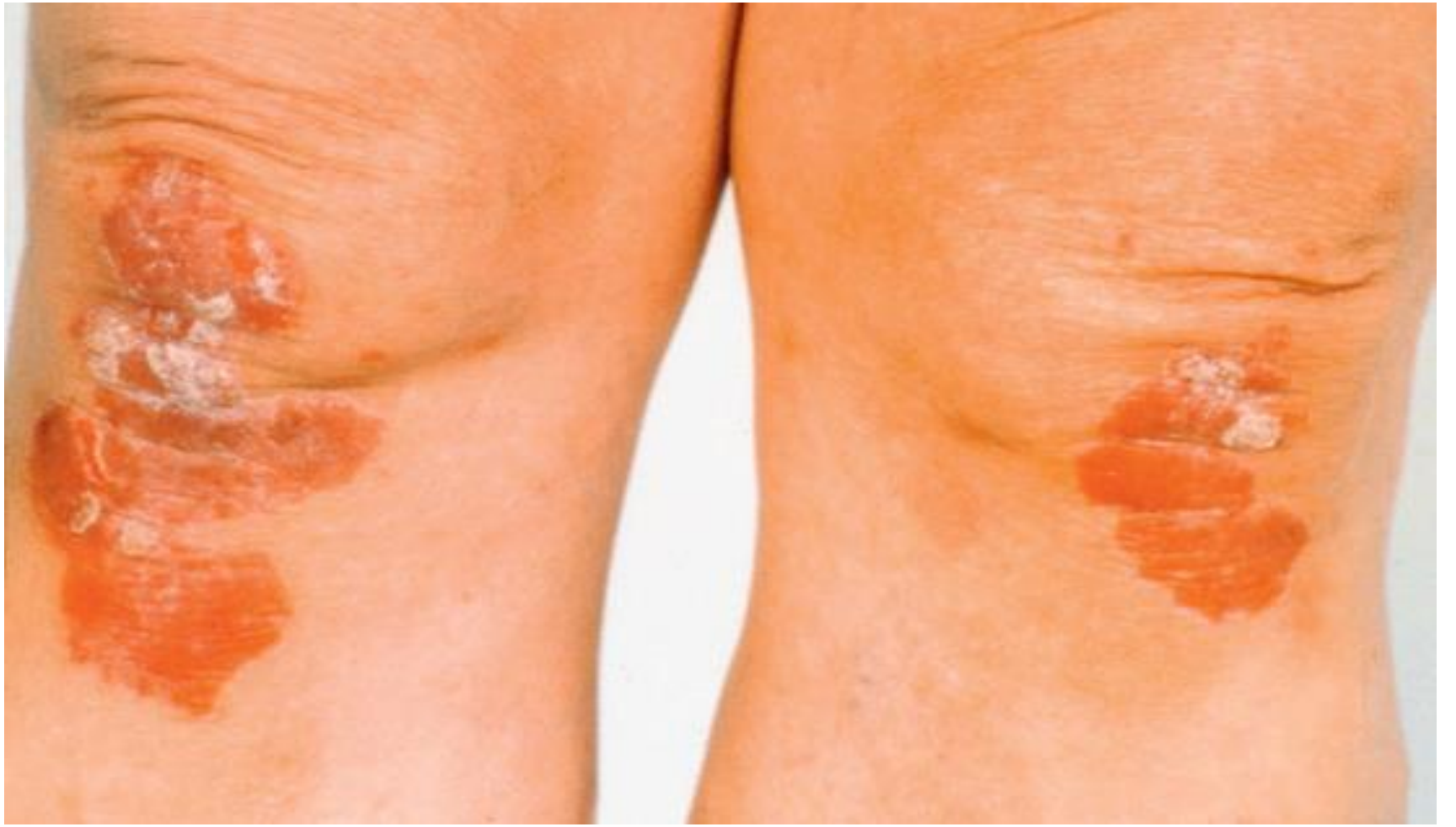


Skin and nails

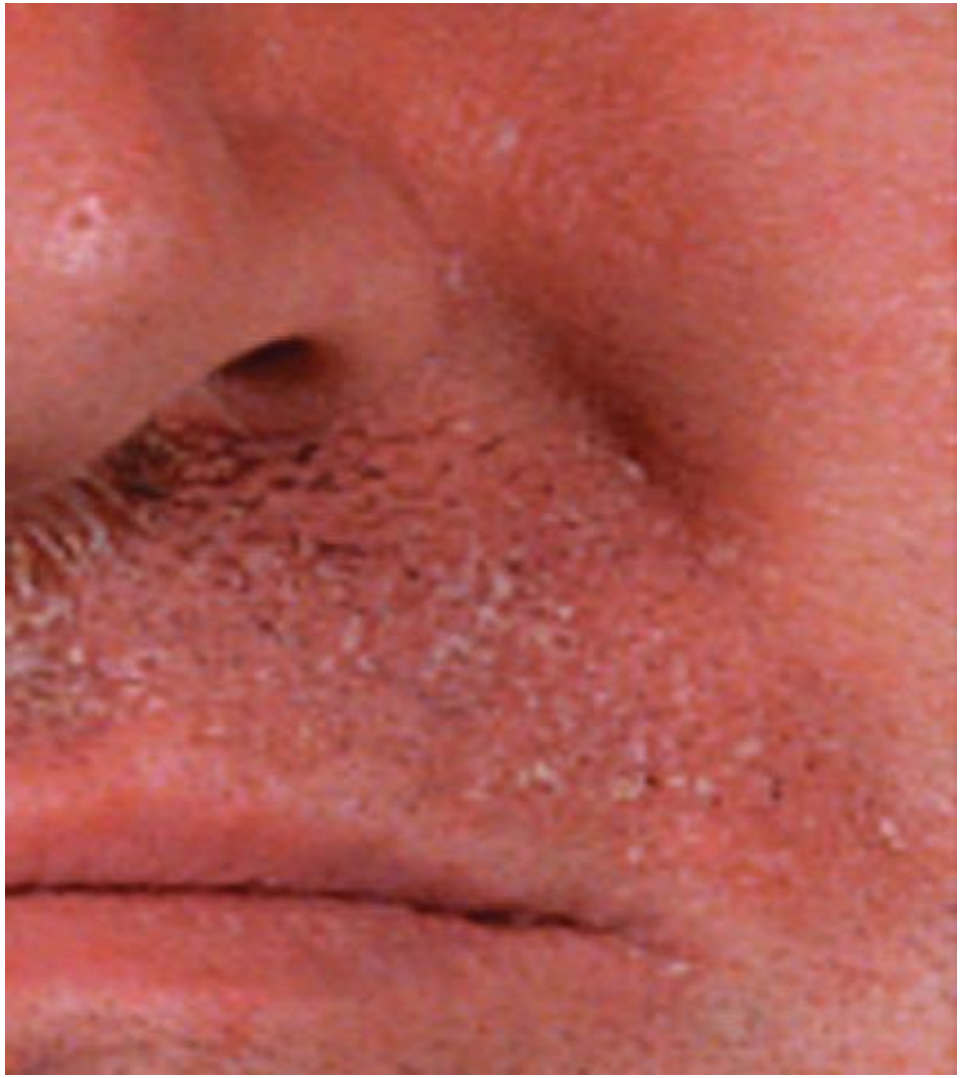
Atopic eczema in the popliteal fossae and ankles.



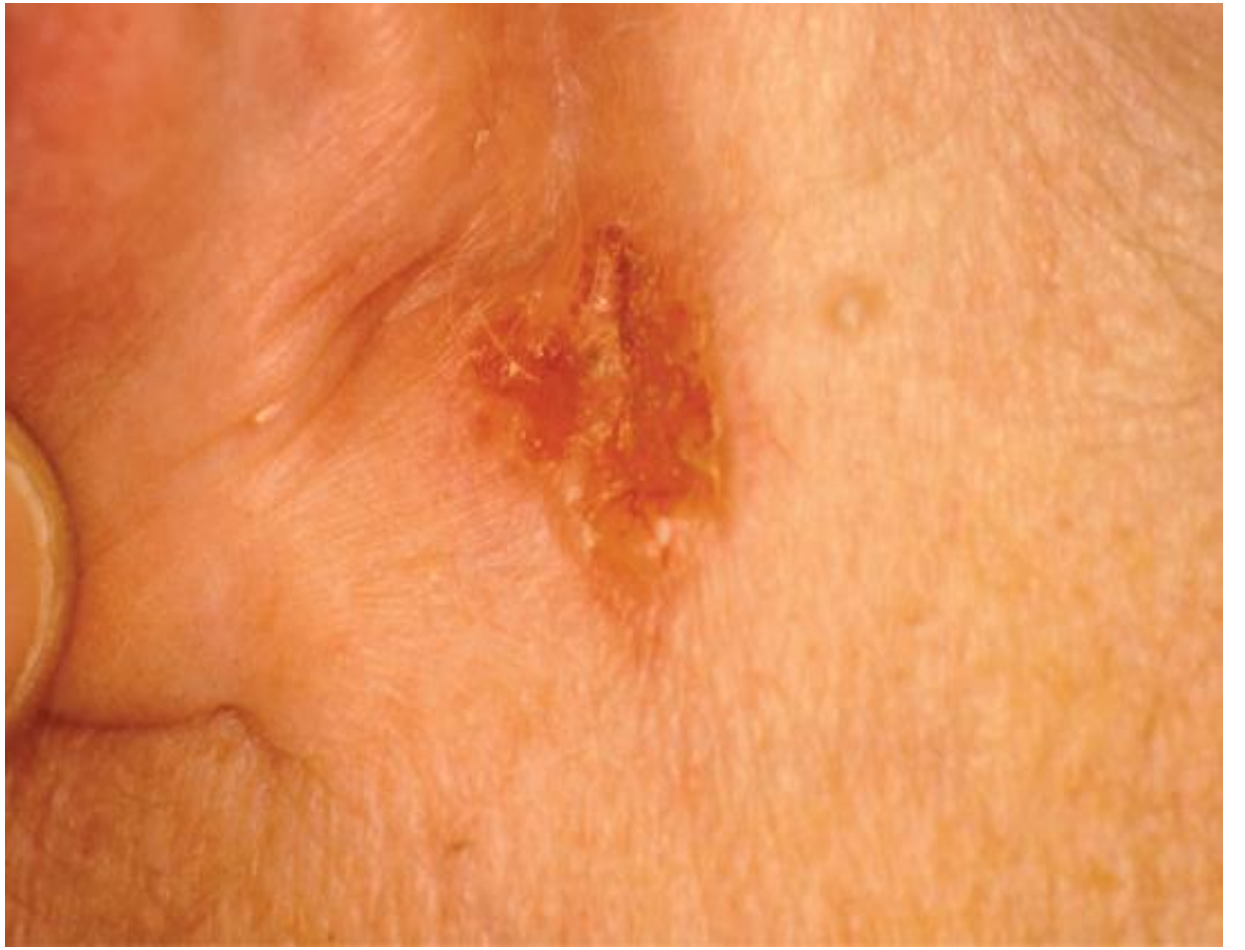
Psoriasis on the knees



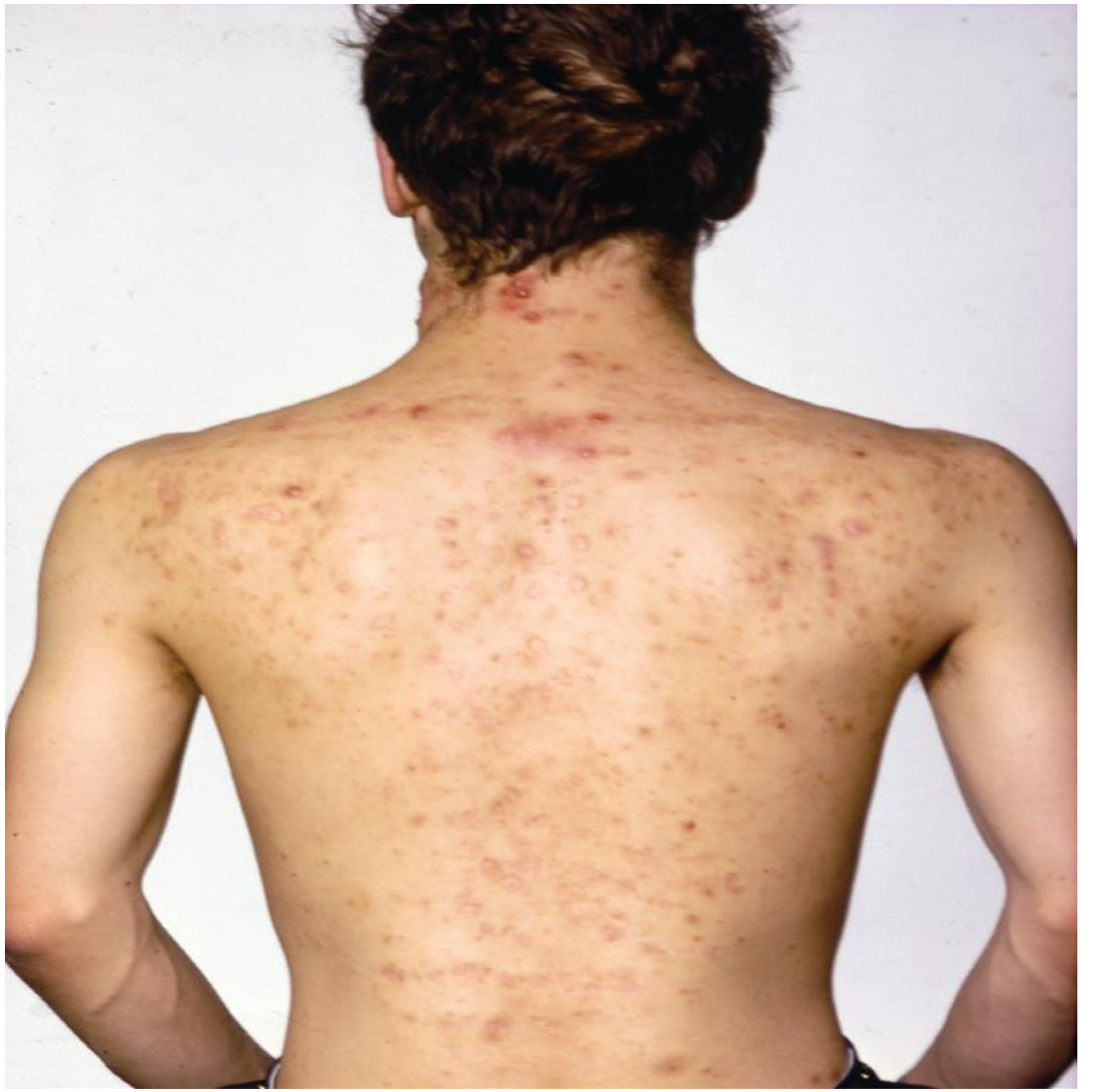
Seborrhoeic dermatitis



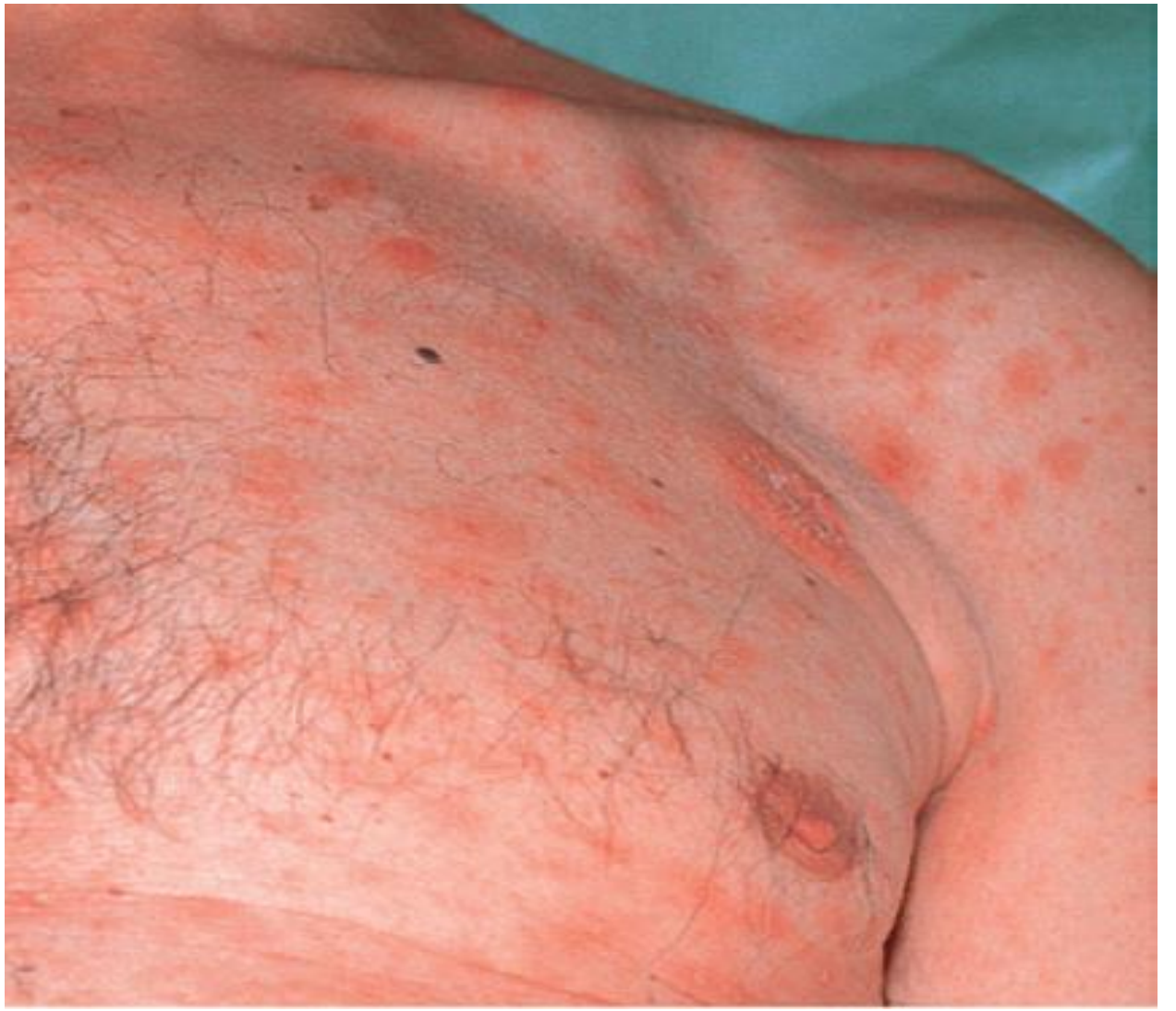
Basal cell cancer showing pearly papules and telangiectasia



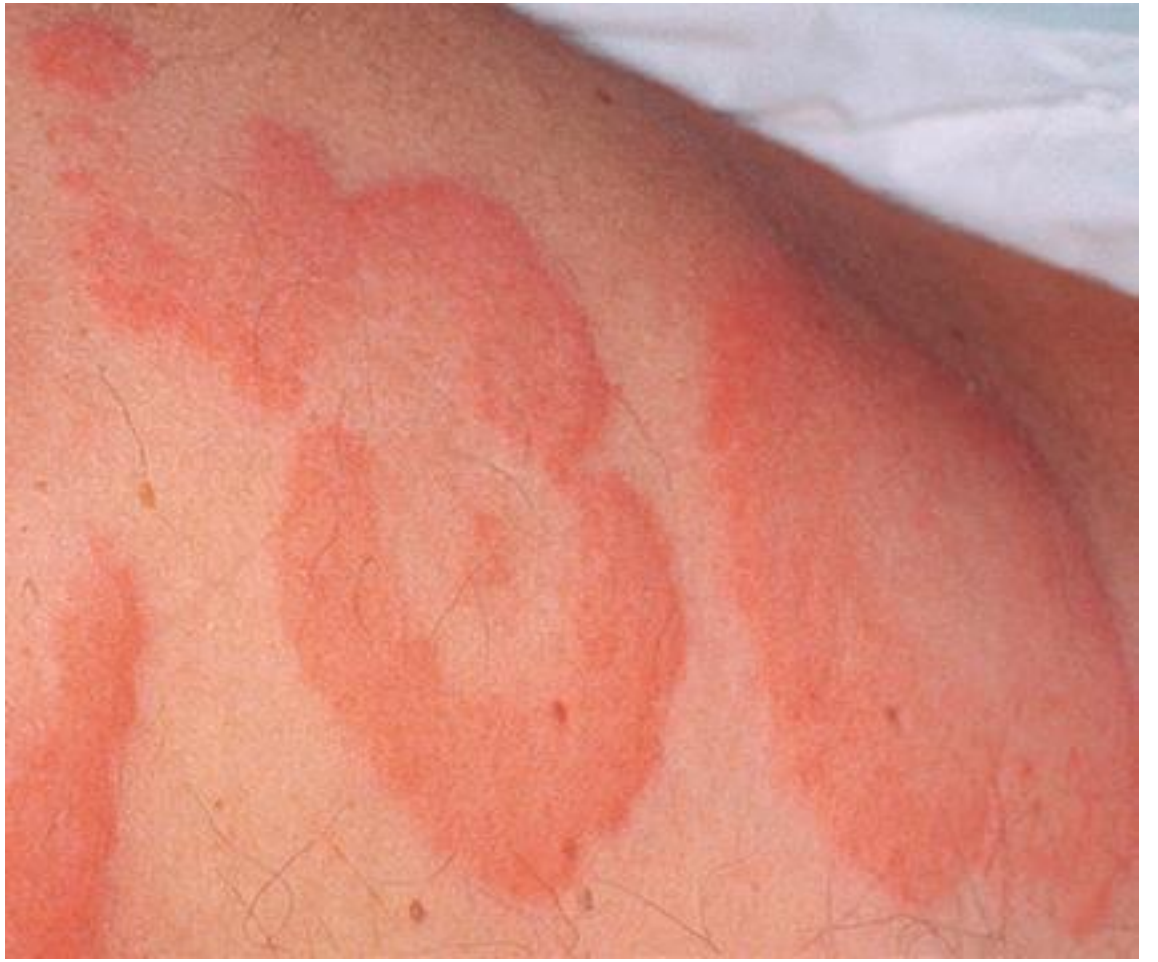
Acne vulgaris



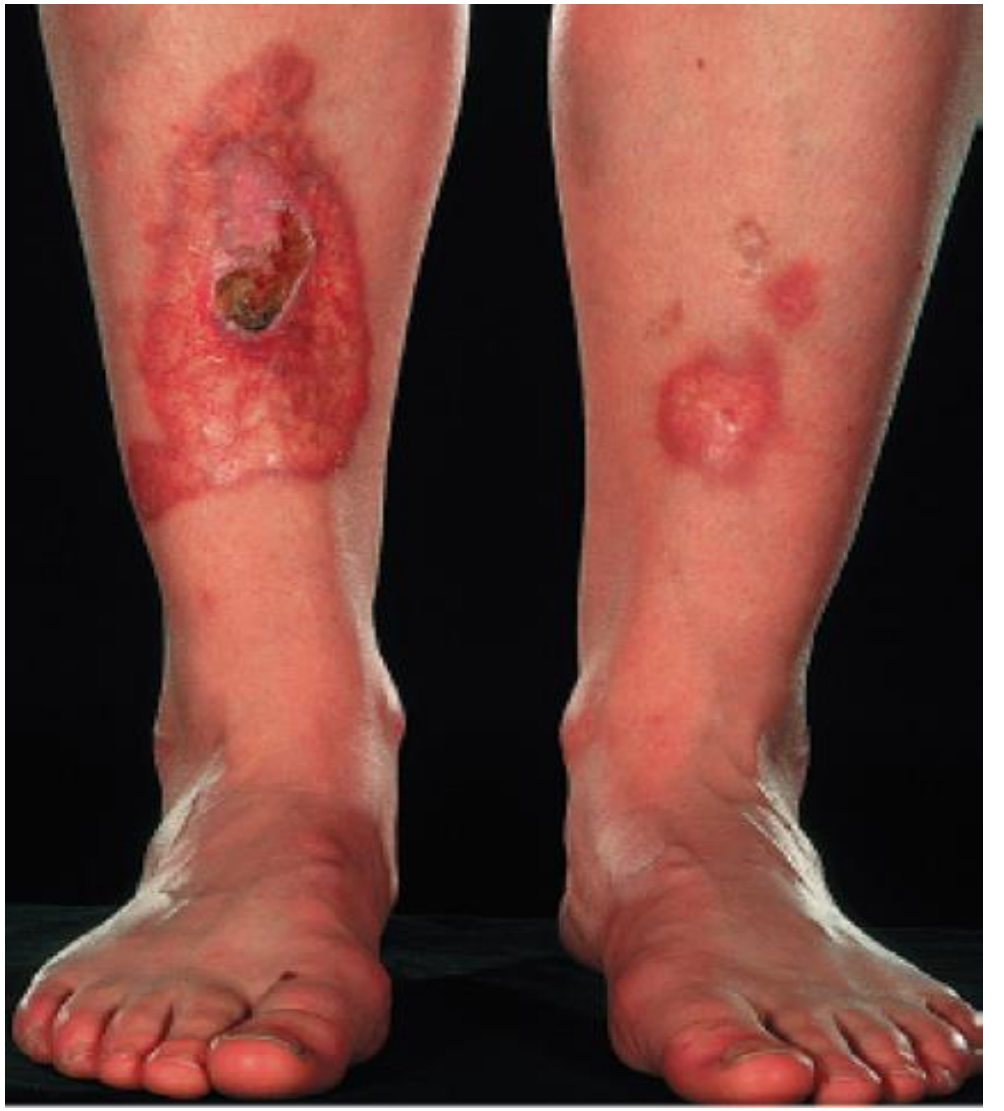
Pityriasis rosea



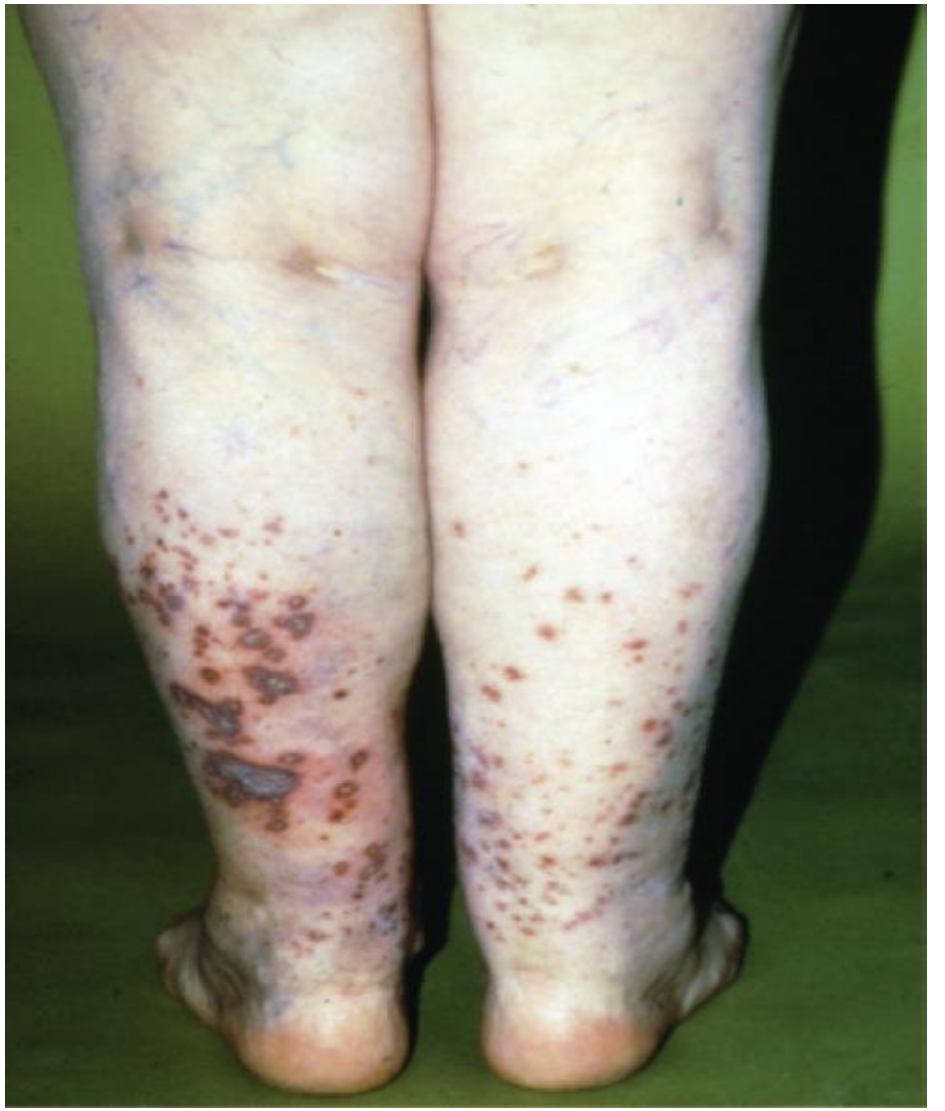
urticaria



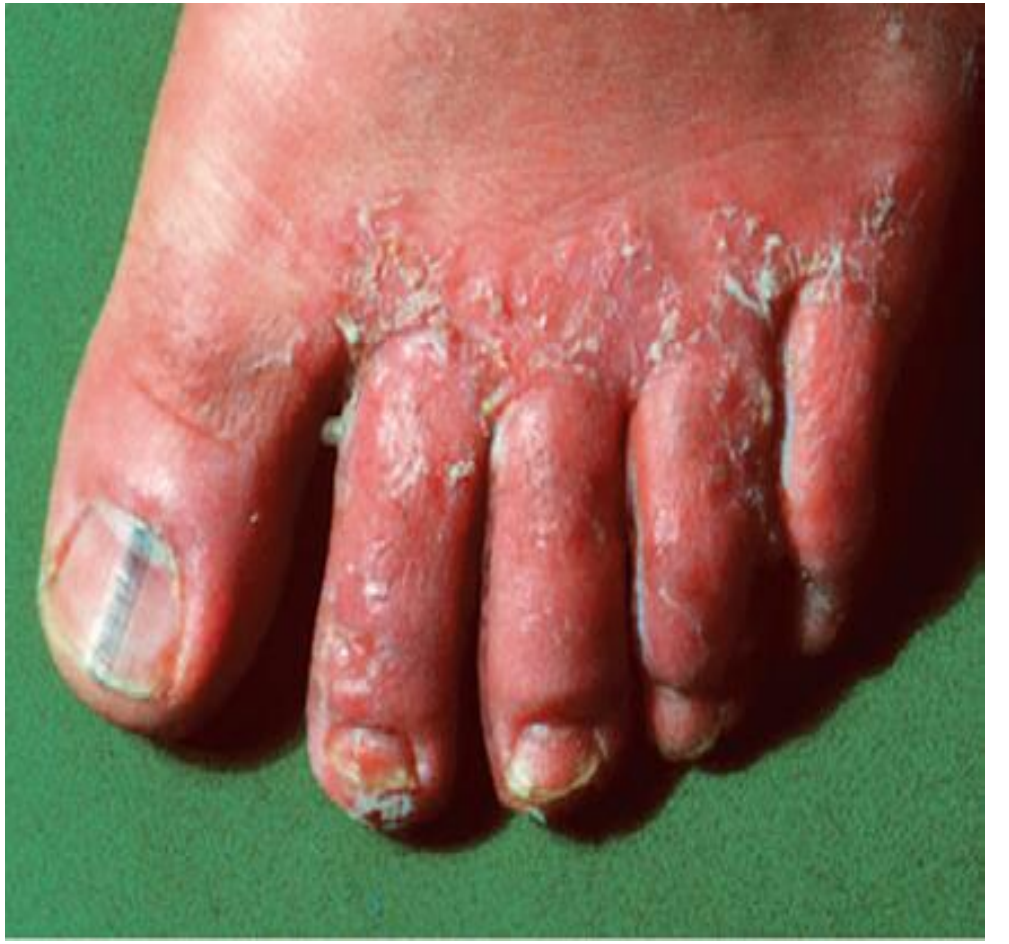
Nicrobiosis lipodica



vasculitis

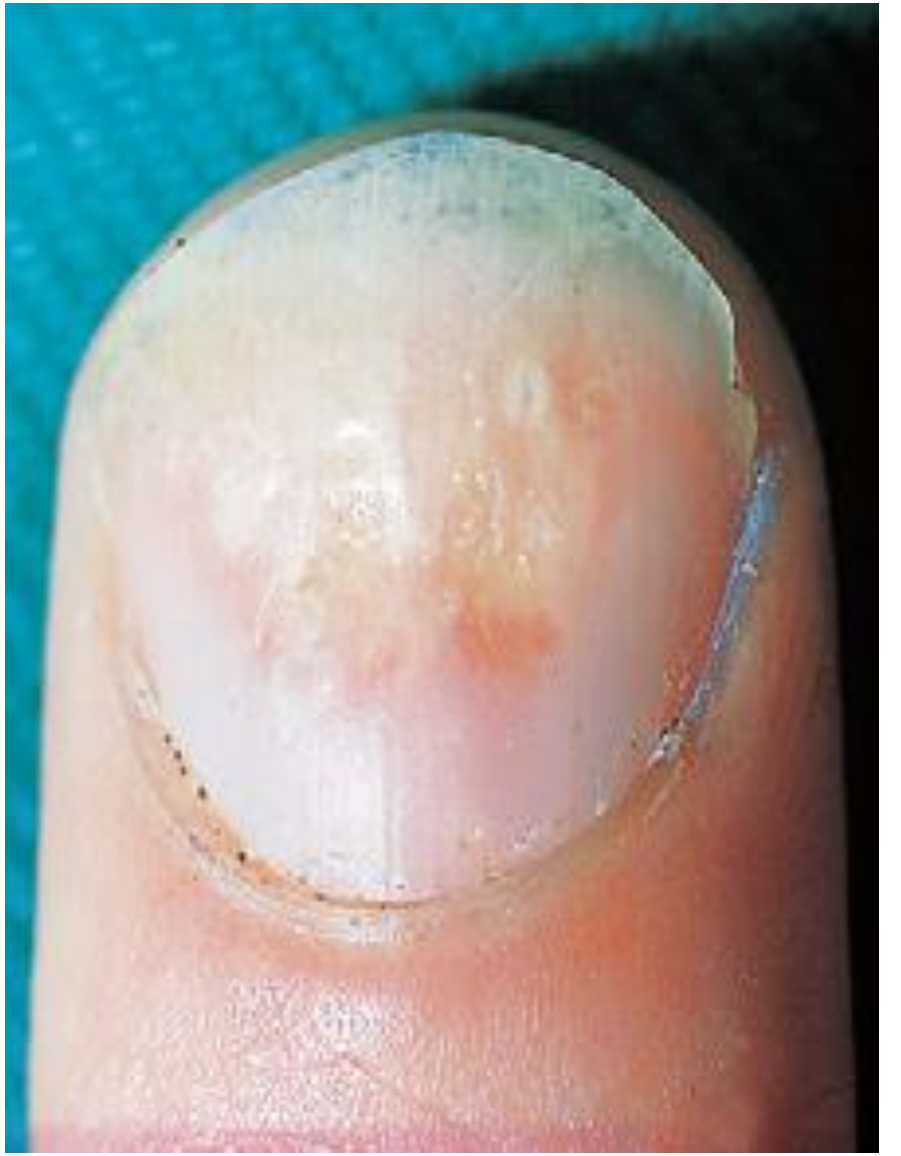


Fungal infection

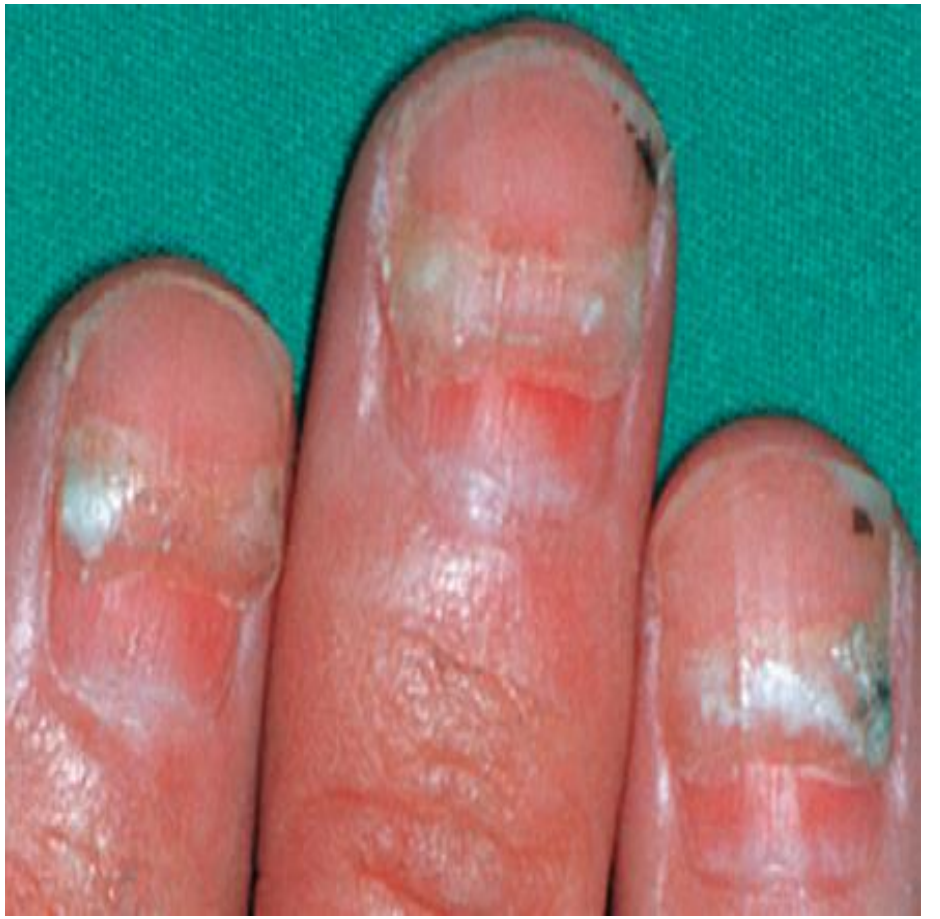


Splinter hemorrhage

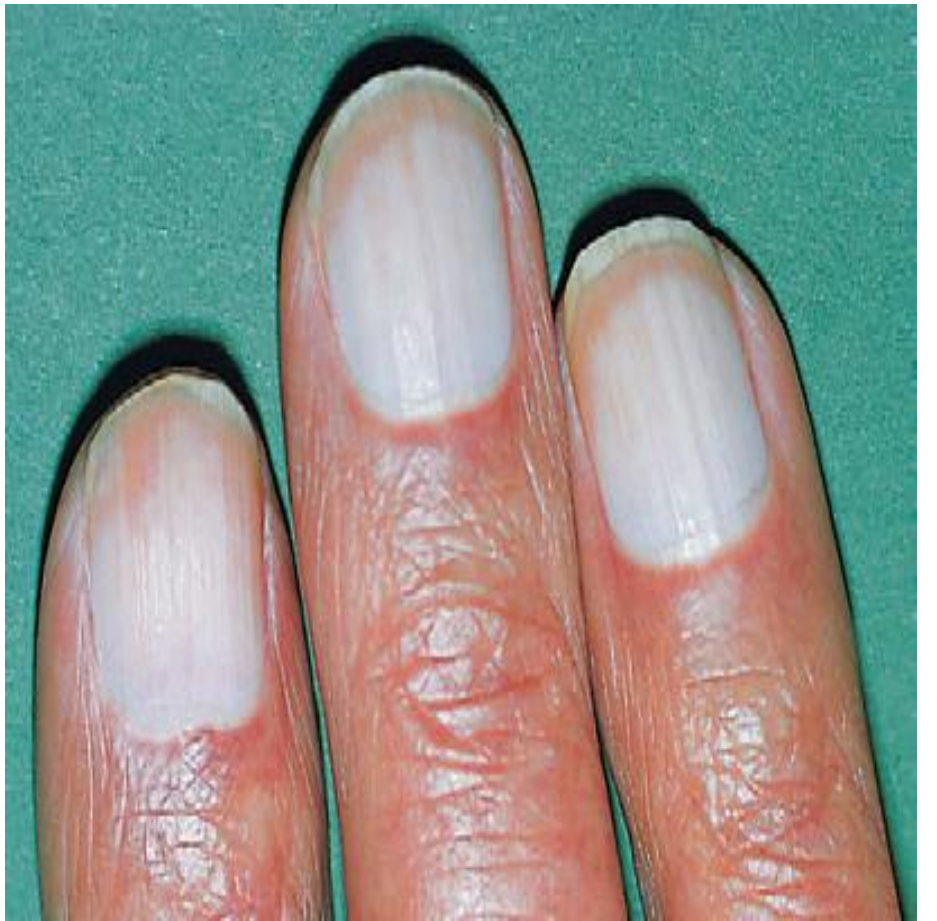
Onycholysis with pitting in psoriasis



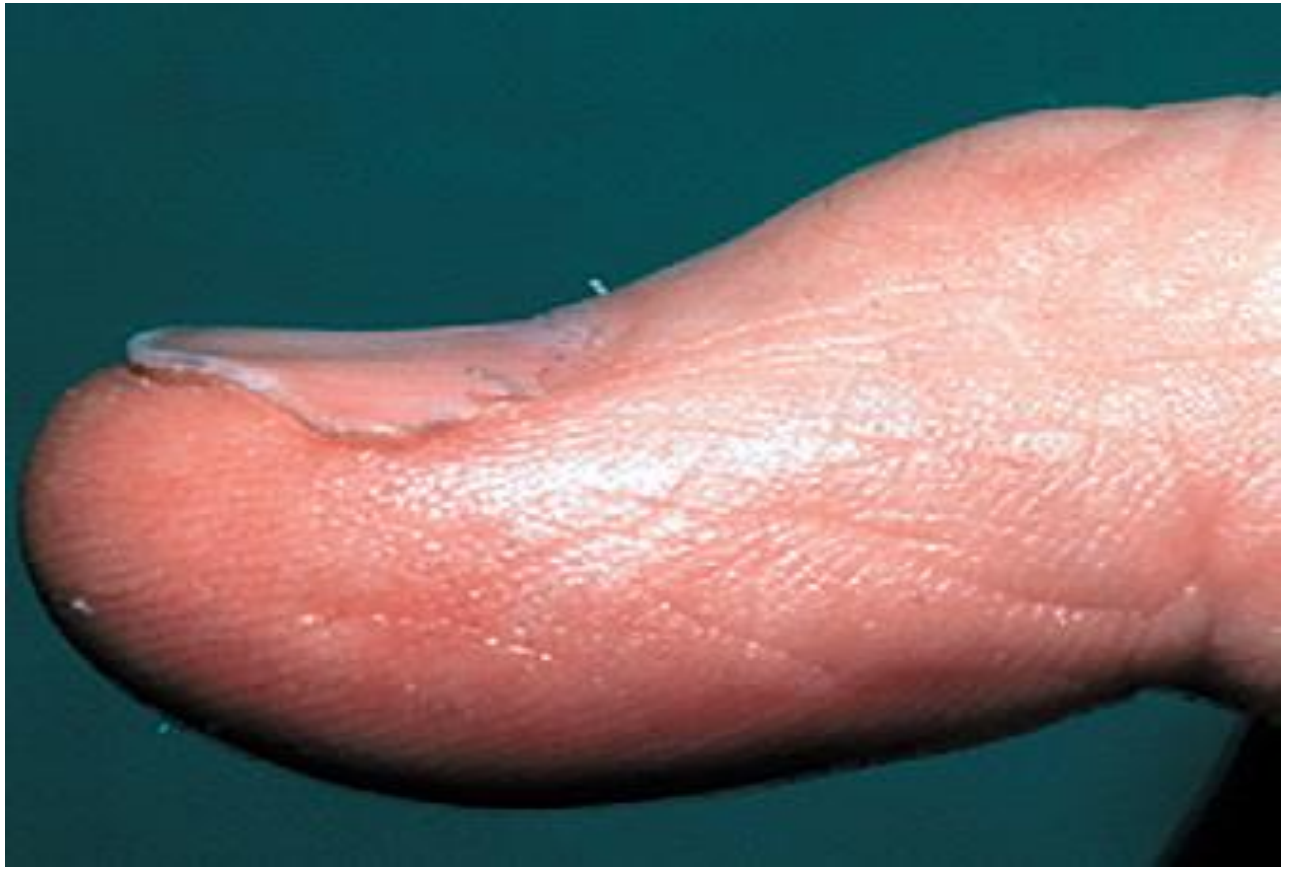
Beau's lines



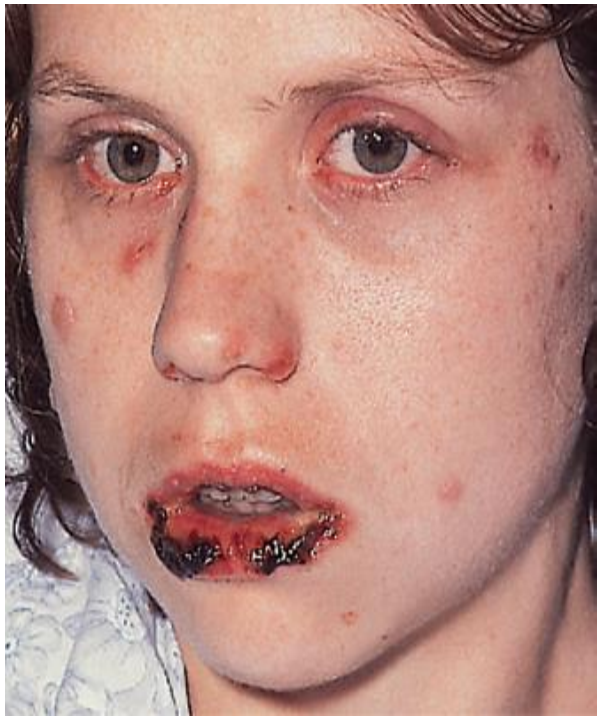
leukonychia

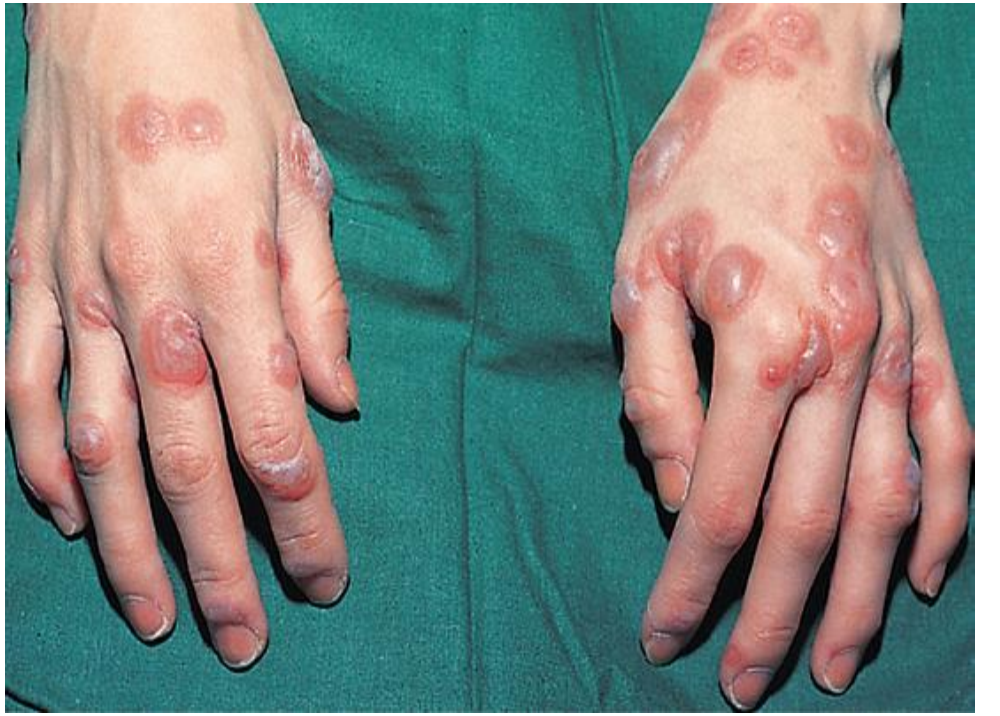


koilonychia

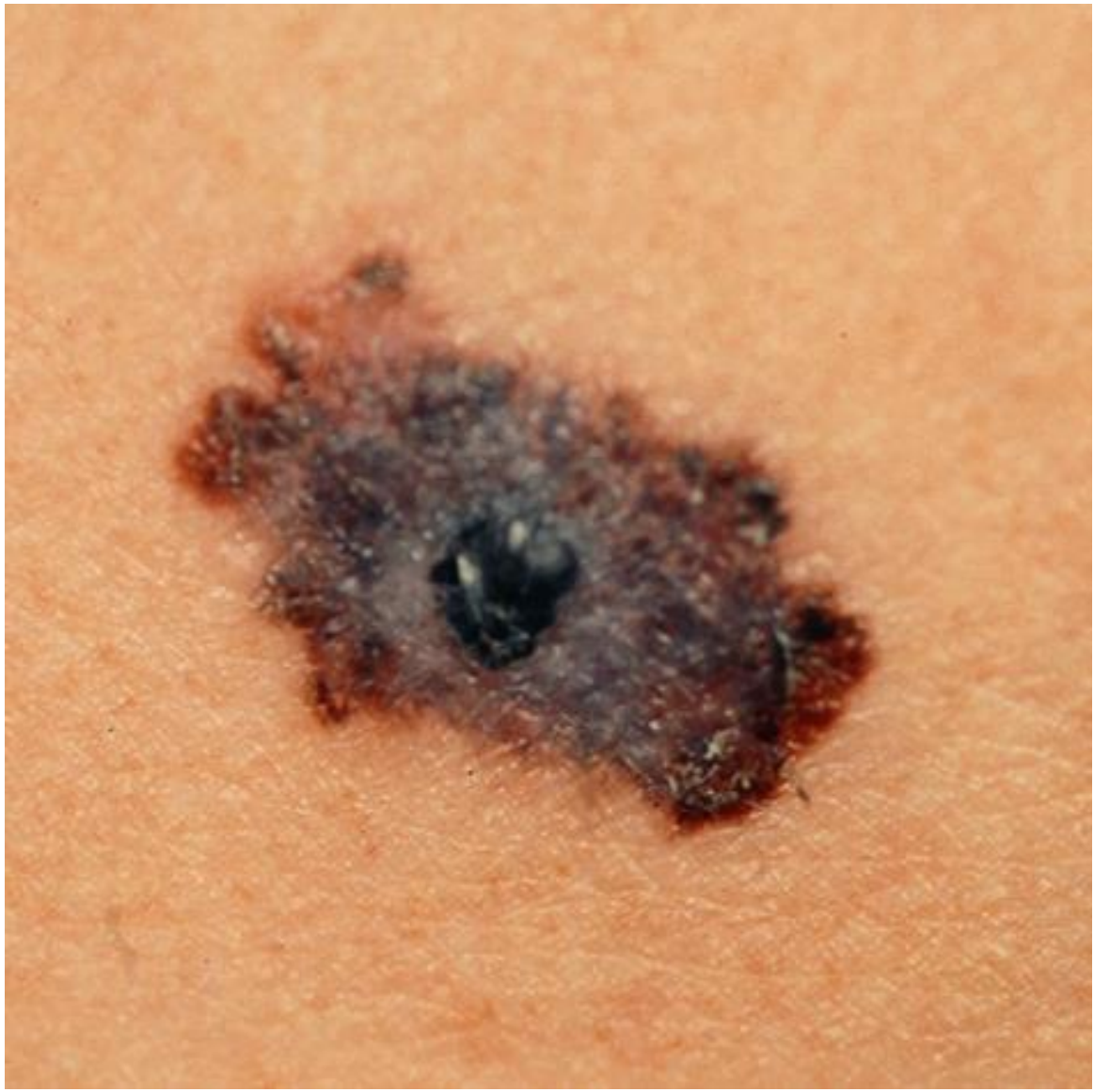


Stevens johnsons syndrome
target lesions on the hand facial and oral lesions





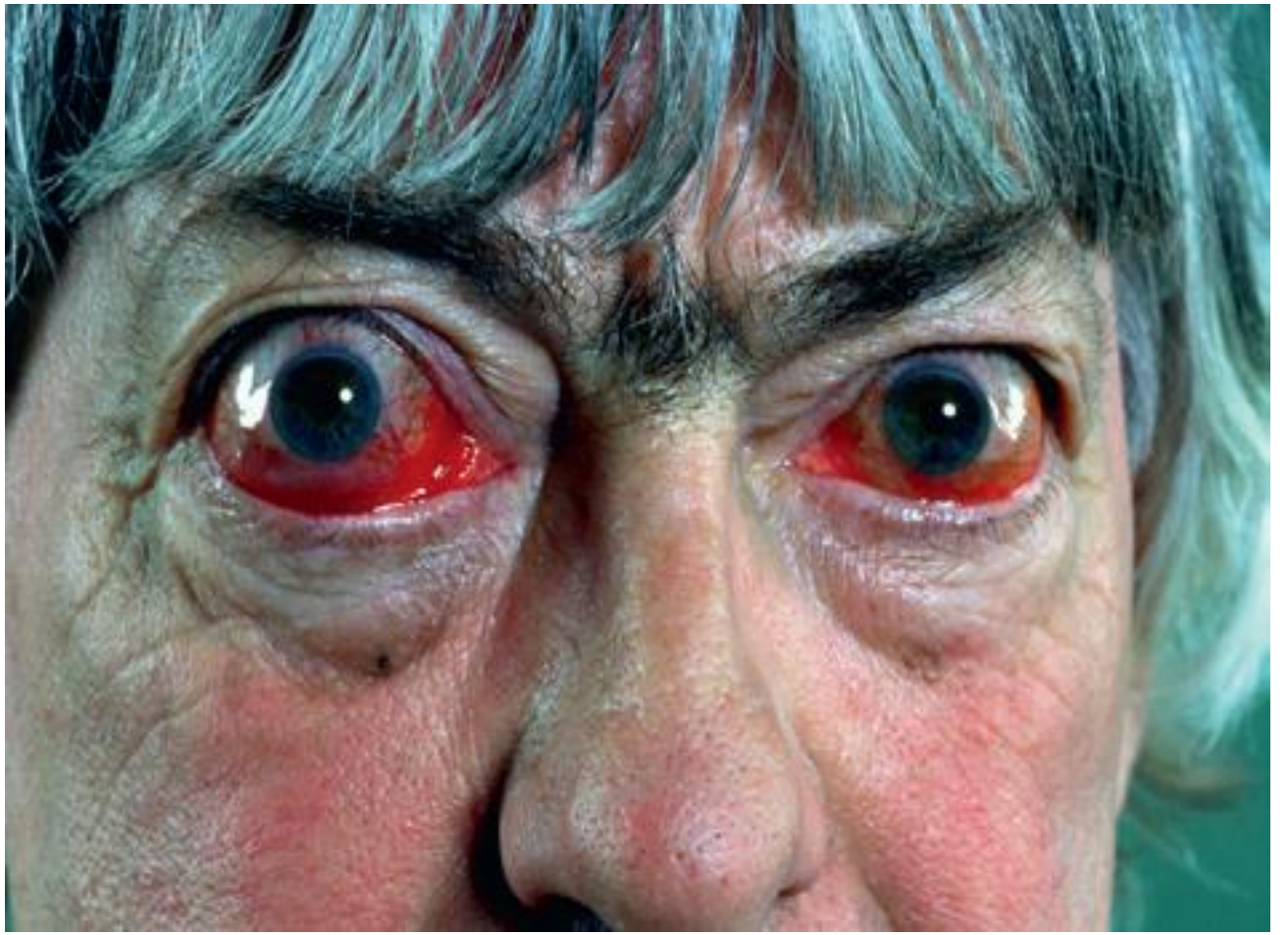
Malignant melanoma



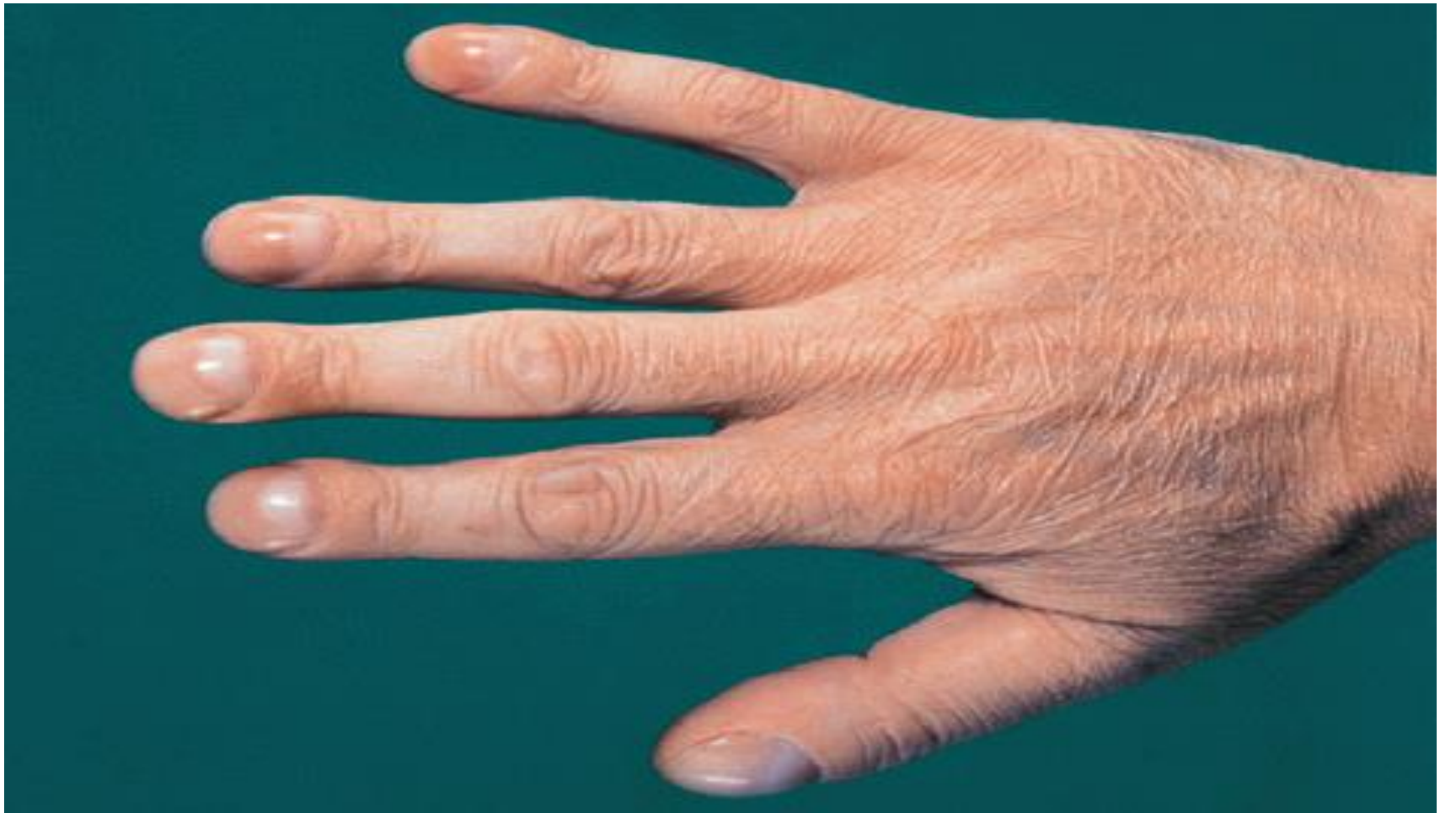
Endocrine system

Graves hyperthyroid(typical face)

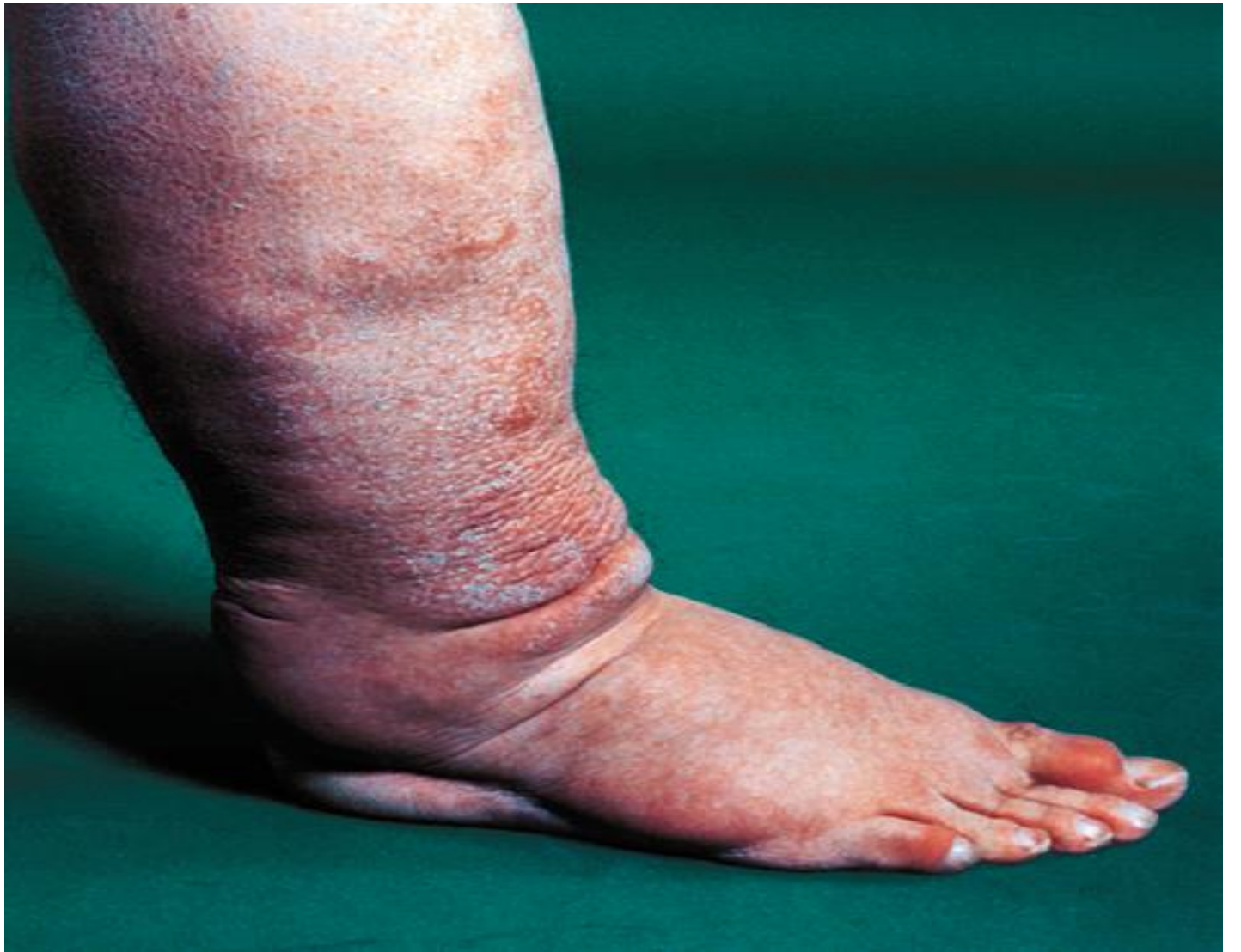
Severe inflammatory thyroid eye disease.



Thyroid achropachy



Pretibial myxedema



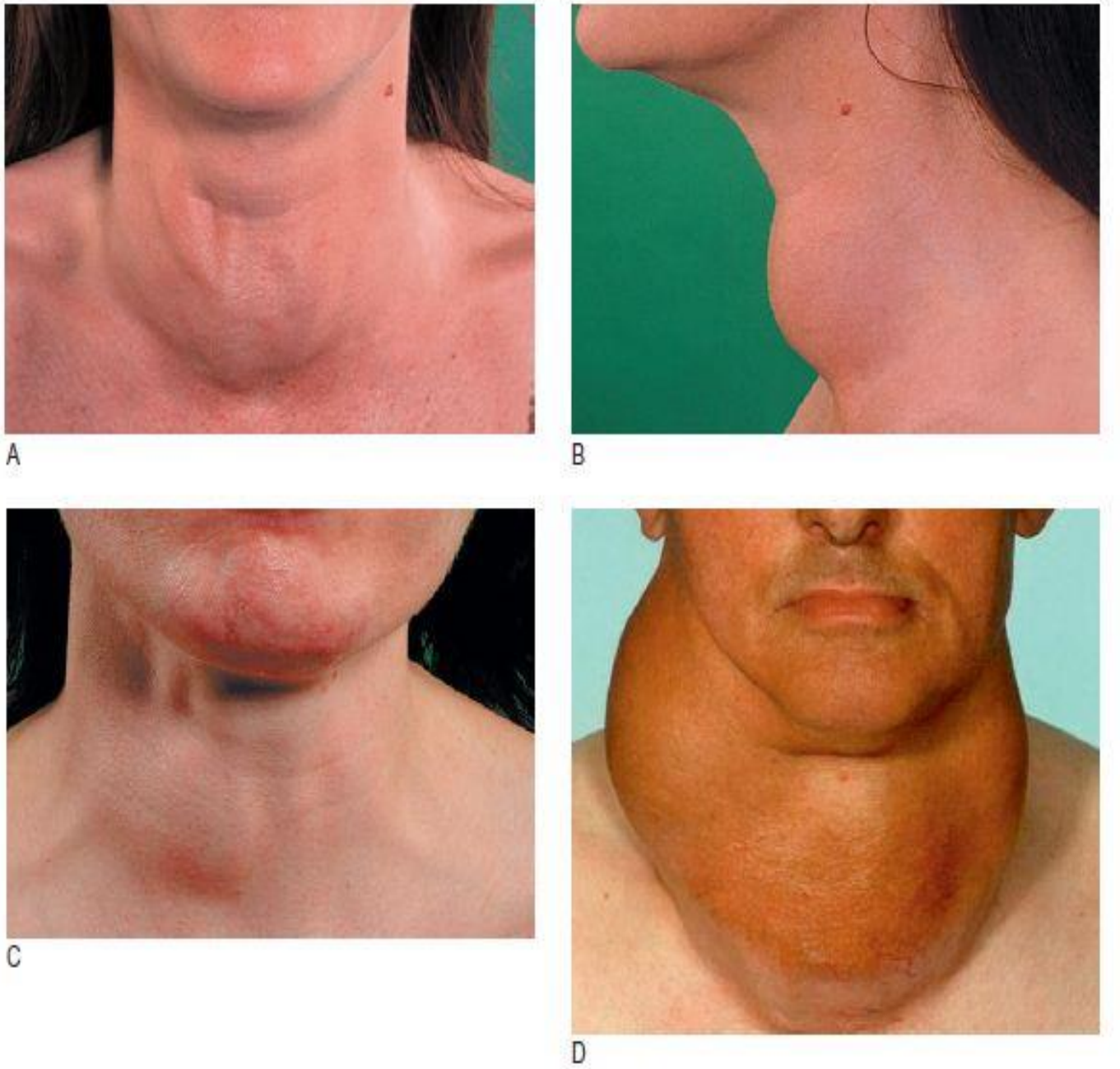
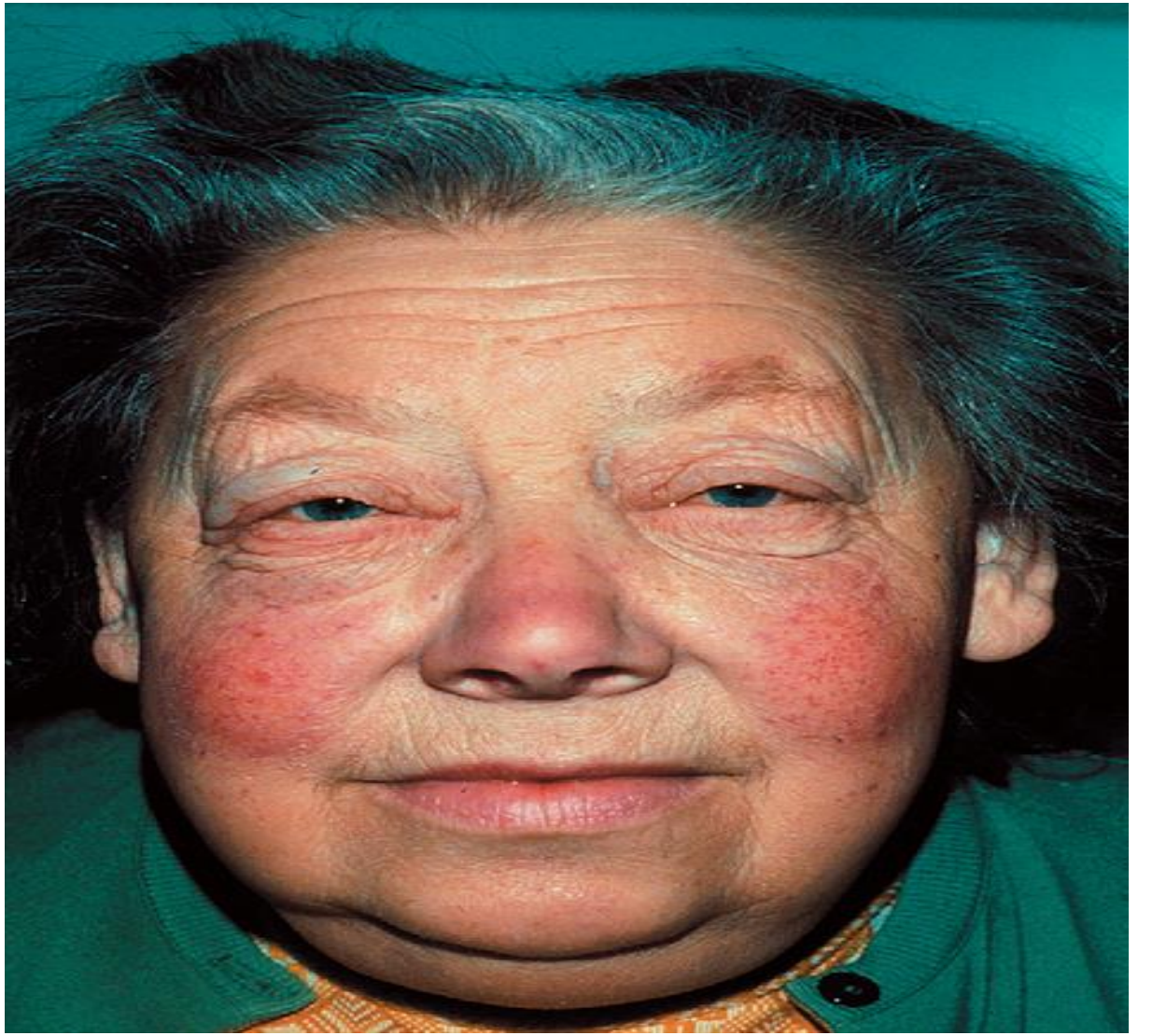
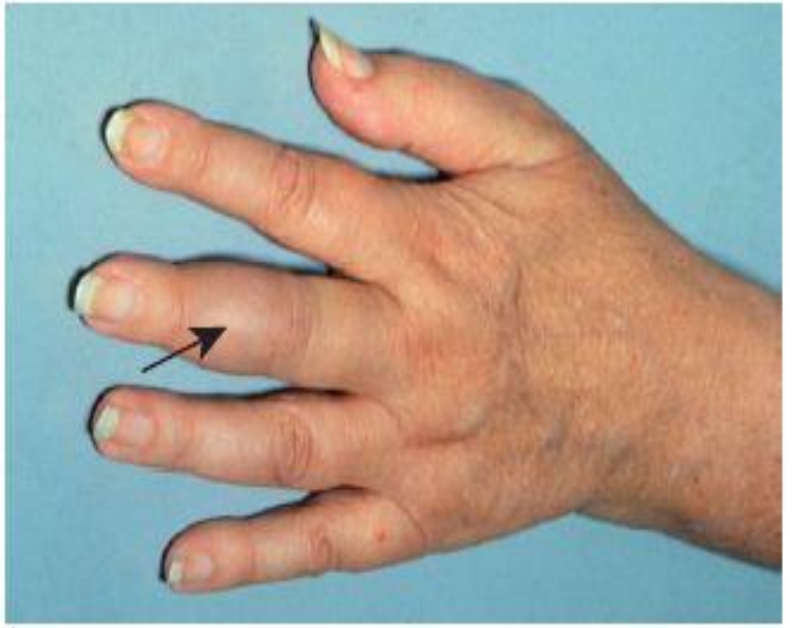


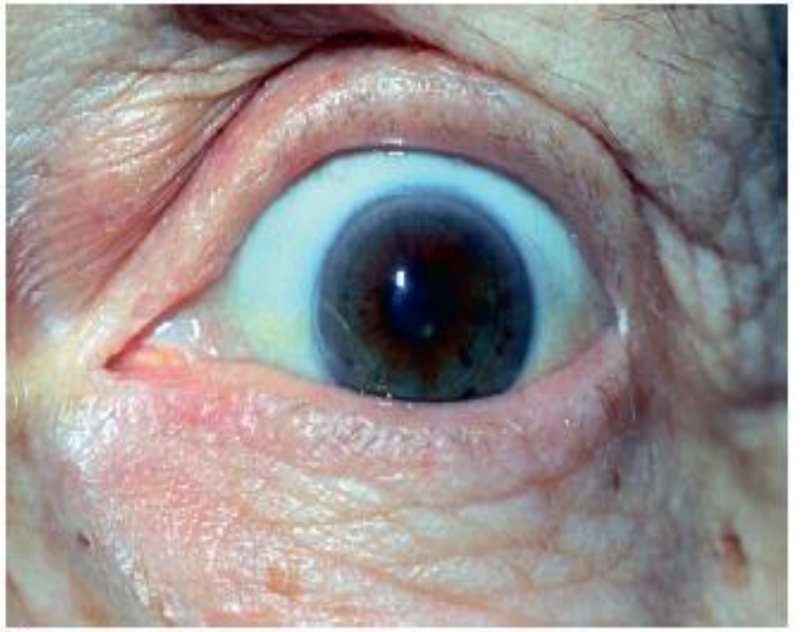
Fig. 5.5 Goitres. (A and B) Diffuse – Graves' disease. **(C)** Uninodular – toxic nodule. **(D)** Multinodular.

hypothyroidism

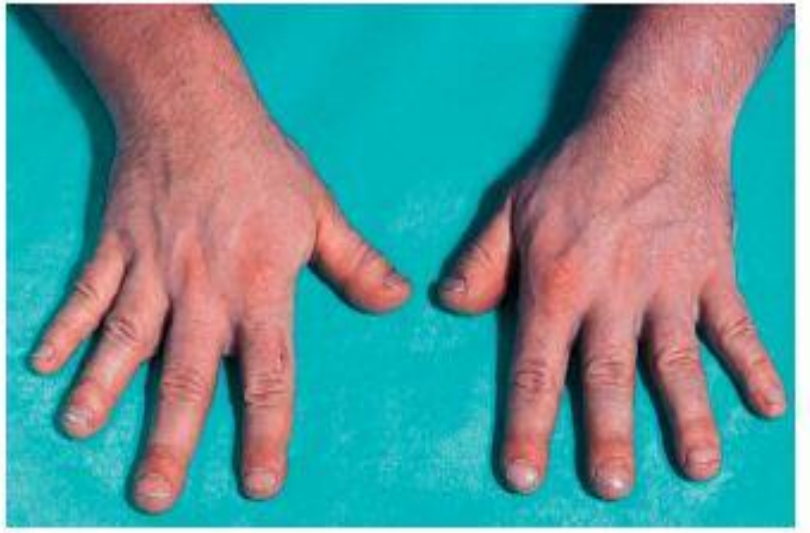




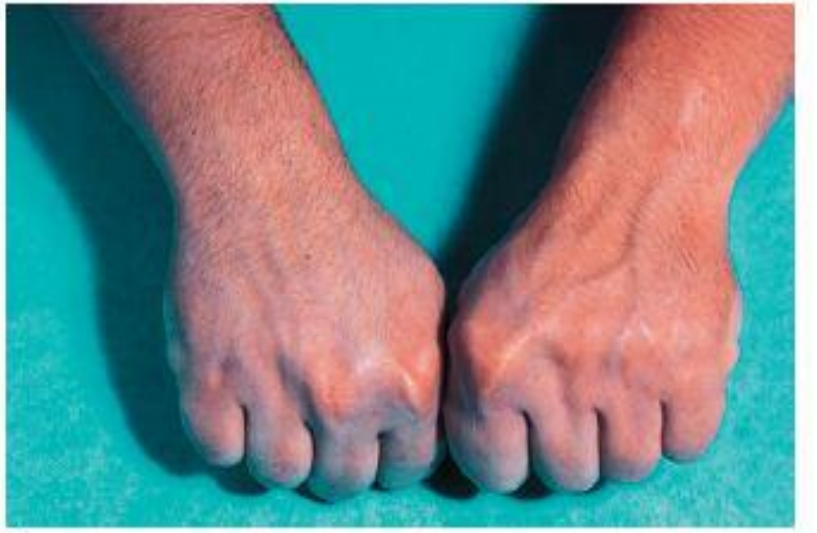
A



B



C

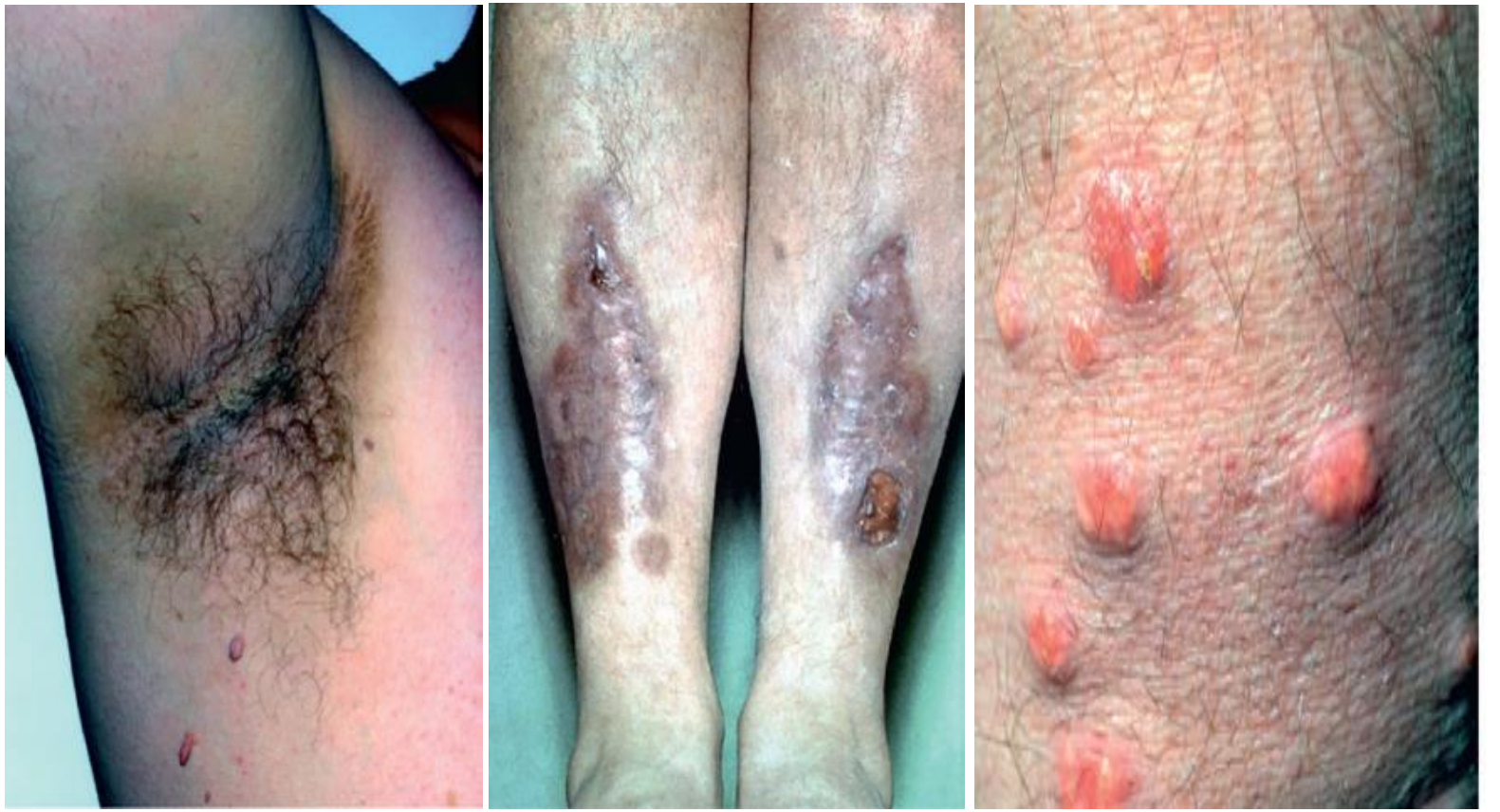


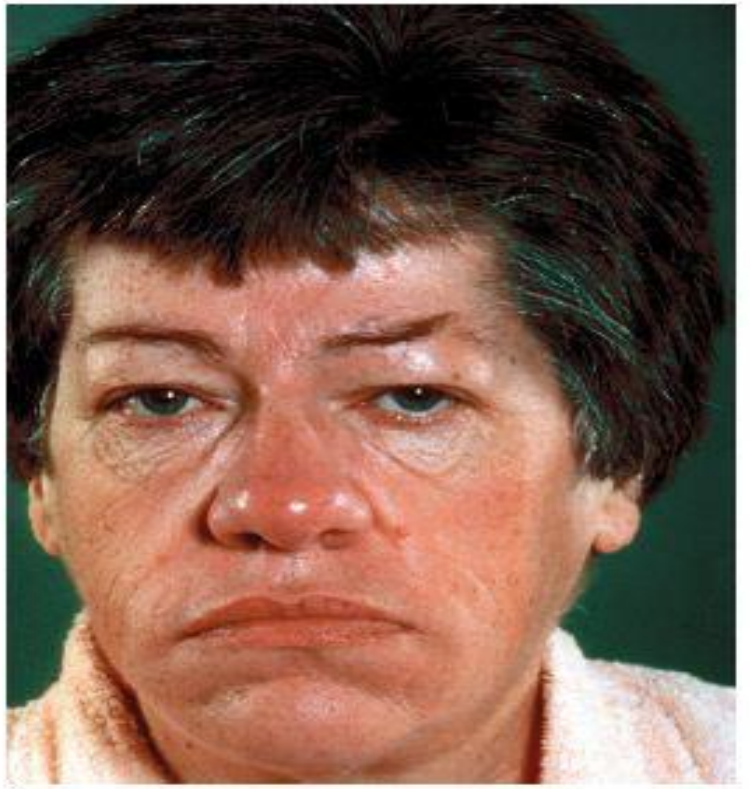
D

Fig. 5.8 Parathyroid disease. (A) 'Brown tumour' of the phalanx (middle finger) in hyperparathyroidism. **(B)** Corneal calcification in hyperparathyroidism. **(C)** Pseudohypoparathyroidism: short metacarpals. **(D)** These are best seen when the patient makes a fist.

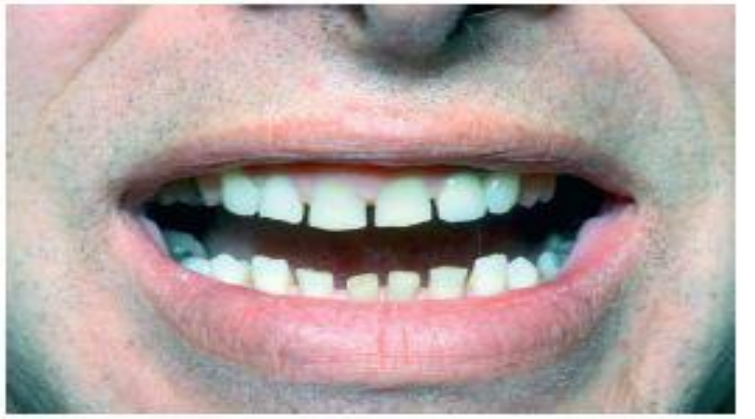
A.Acanthosis negricans
B.necrobiosis lipodica
C.eruptive xanthomata

- A B C





A



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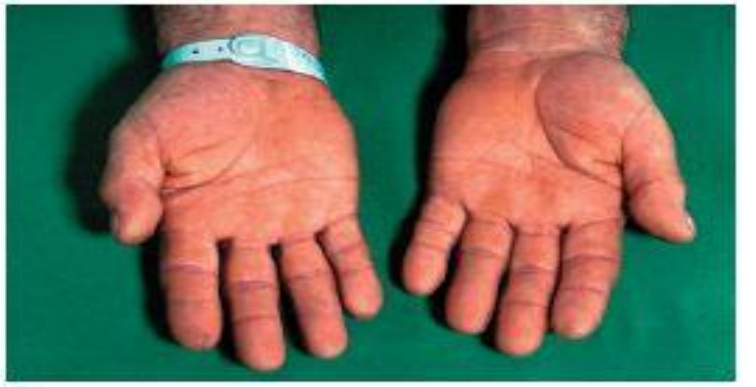
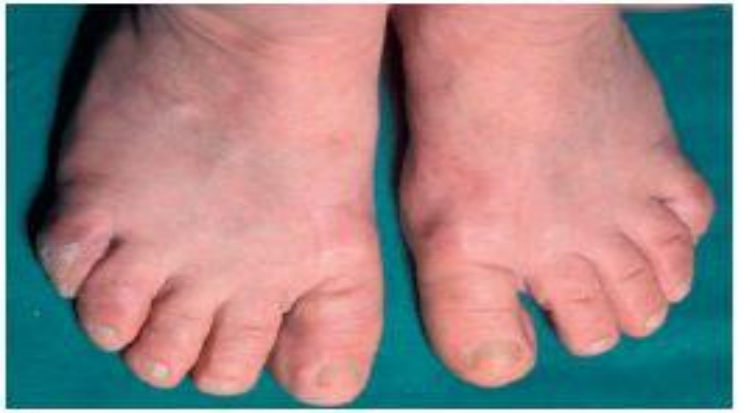
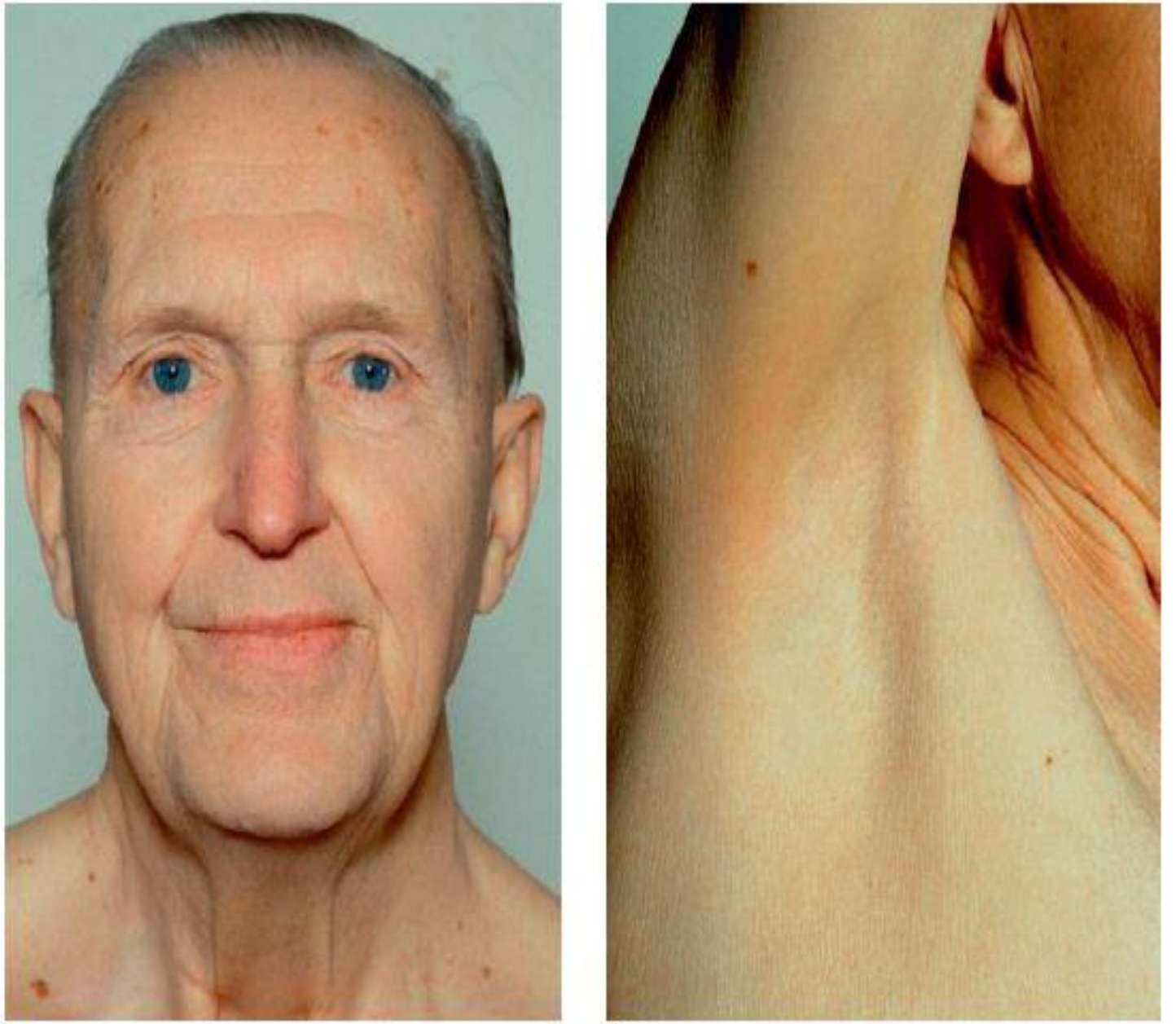


C



D

Fig. 5.14 Acromegaly. (A) Typical facies. **(B)** Separation of lower teeth. **(C)** Large fleshy hands. **(D)** Widening of the feet.

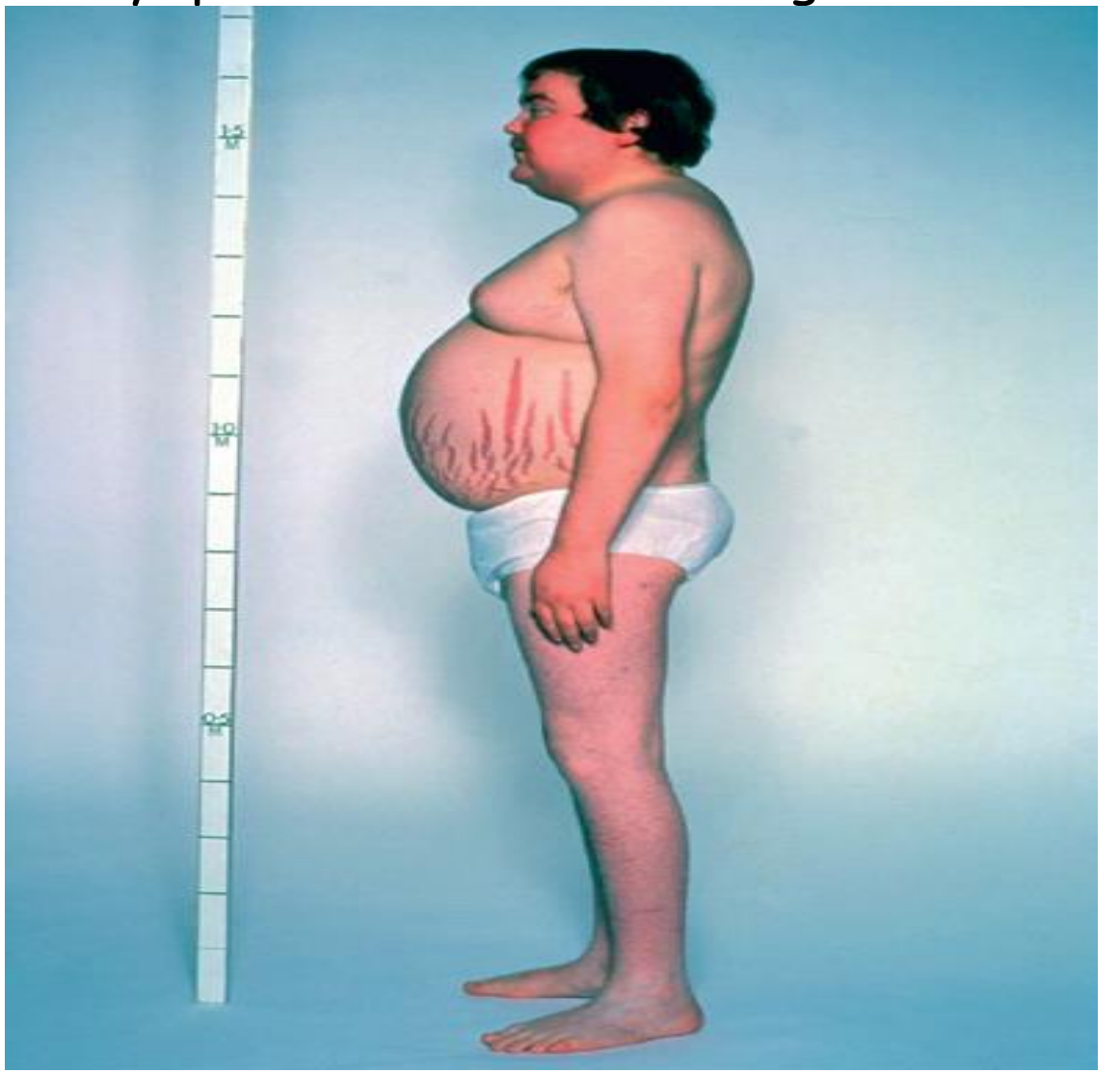


A B

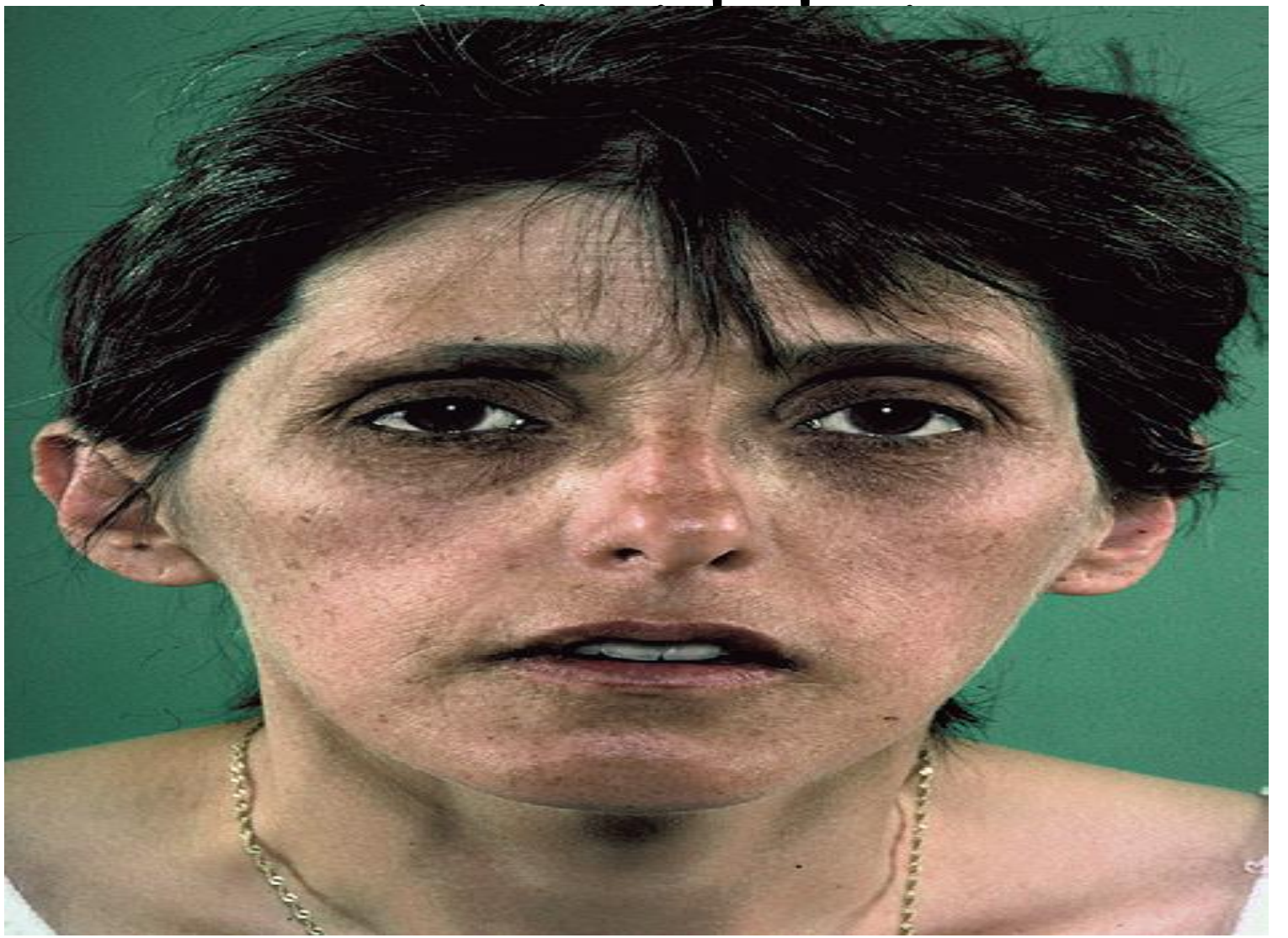
Fig. 5.15 Hypopituitarism. (A) Hypopituitarism due to a pituitary adenoma (note the fine pale skin). **(B)** Absent axillary hair.

Cushingoid face

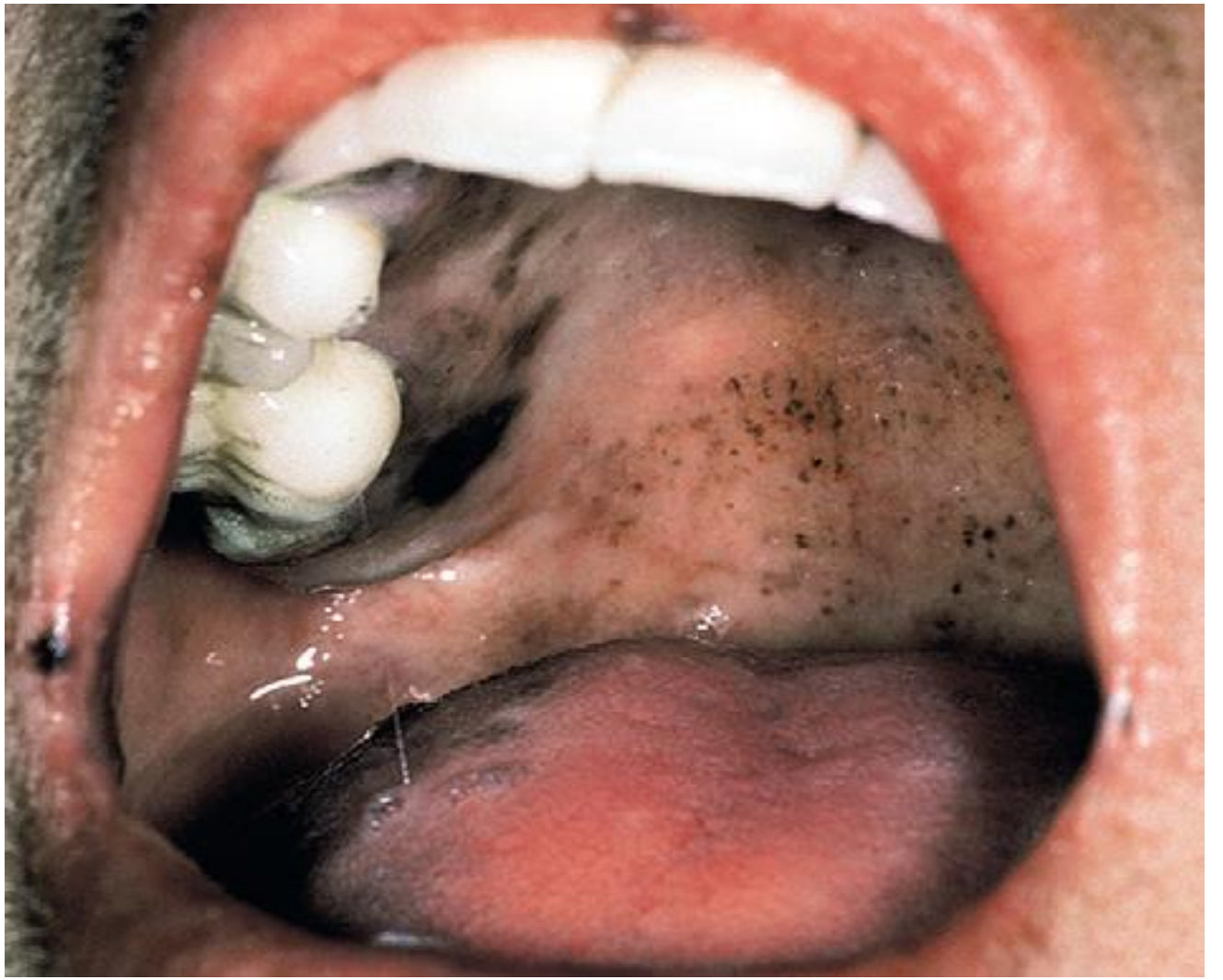
Typical features of cushing: facial rounding, central obesity, proximal muscle wasting and skin striae



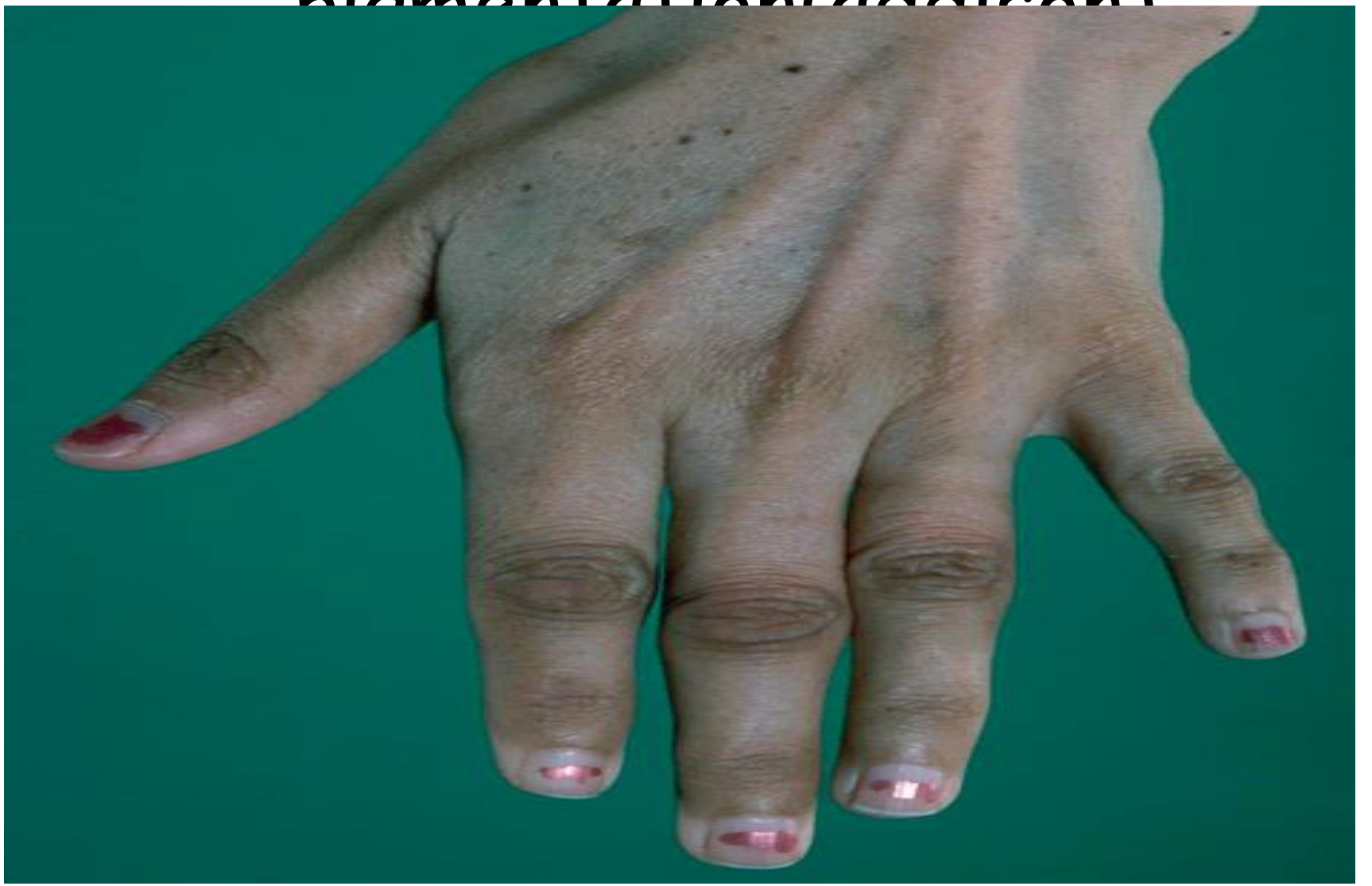
Addison disease facial



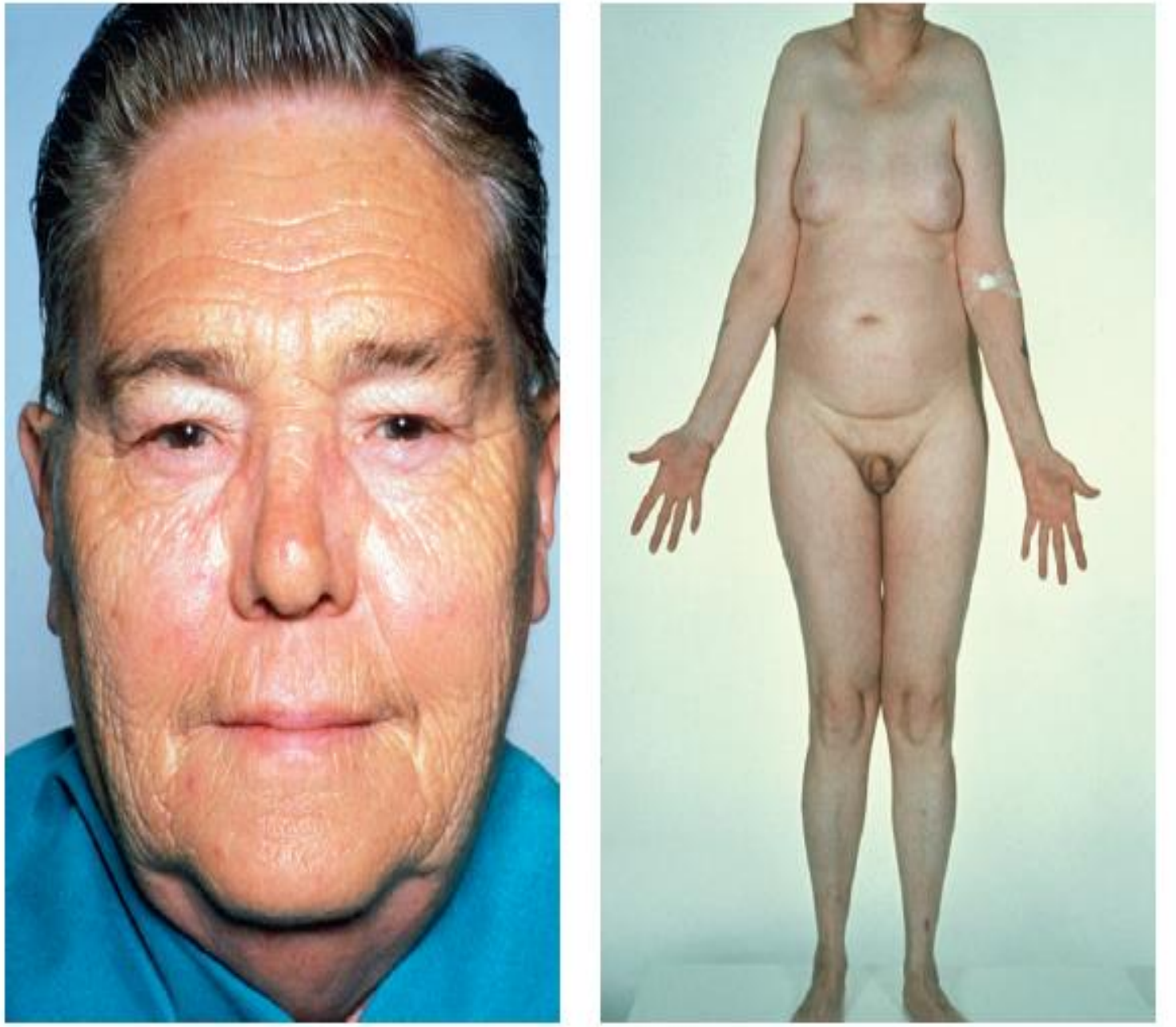
Buccal pigmentation(addison)



Skin crease pigmentation(addison)



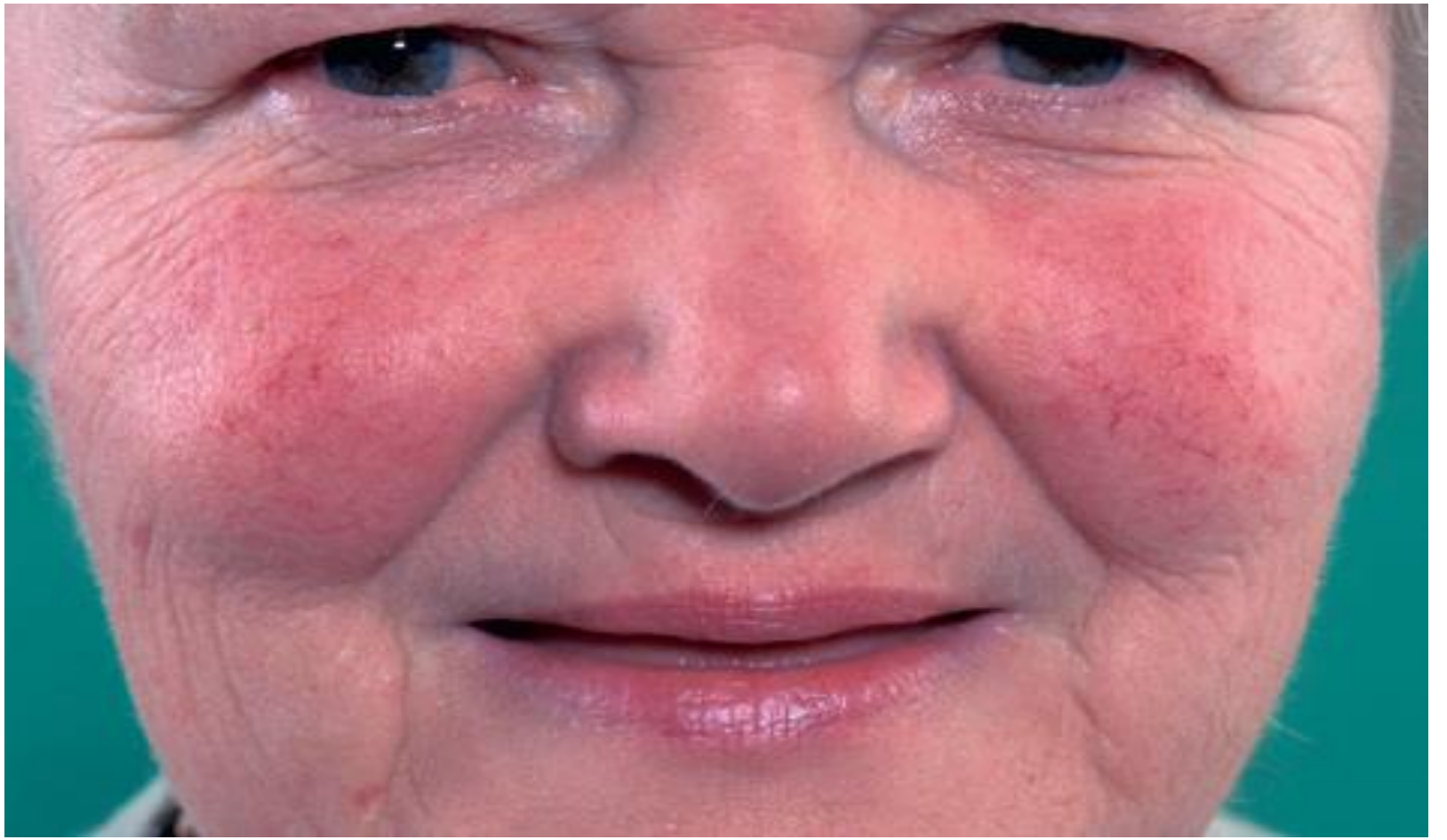
Vitilligo due to addison



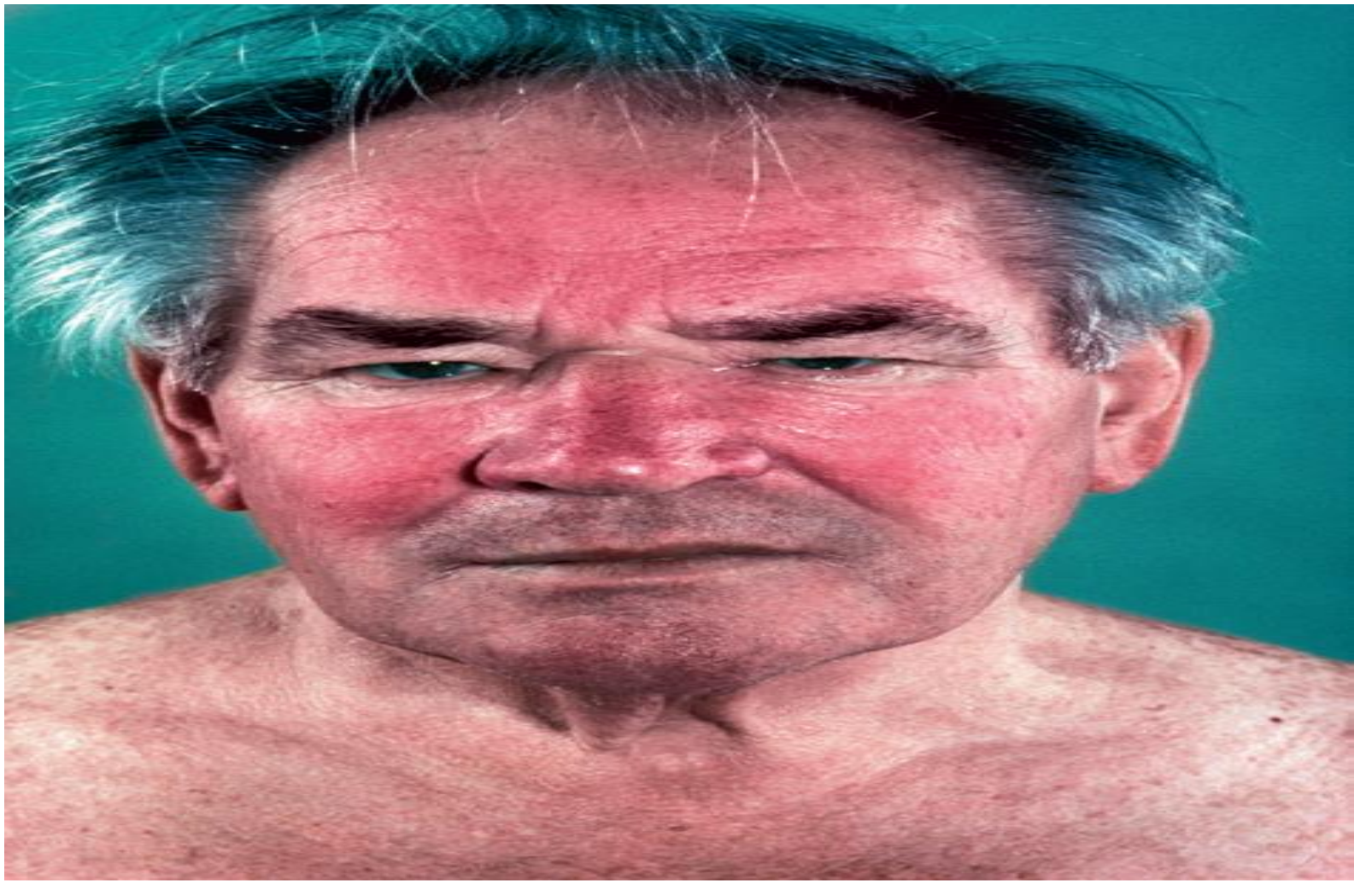
A B

Fig. 5.18 Klinefelter's syndrome. (A) Hypogonadal facial skin. **(B)** Gynaecomastia, reduced pubic hair and small testes.

Acute carcinoid flush(carcinoid syndrome)

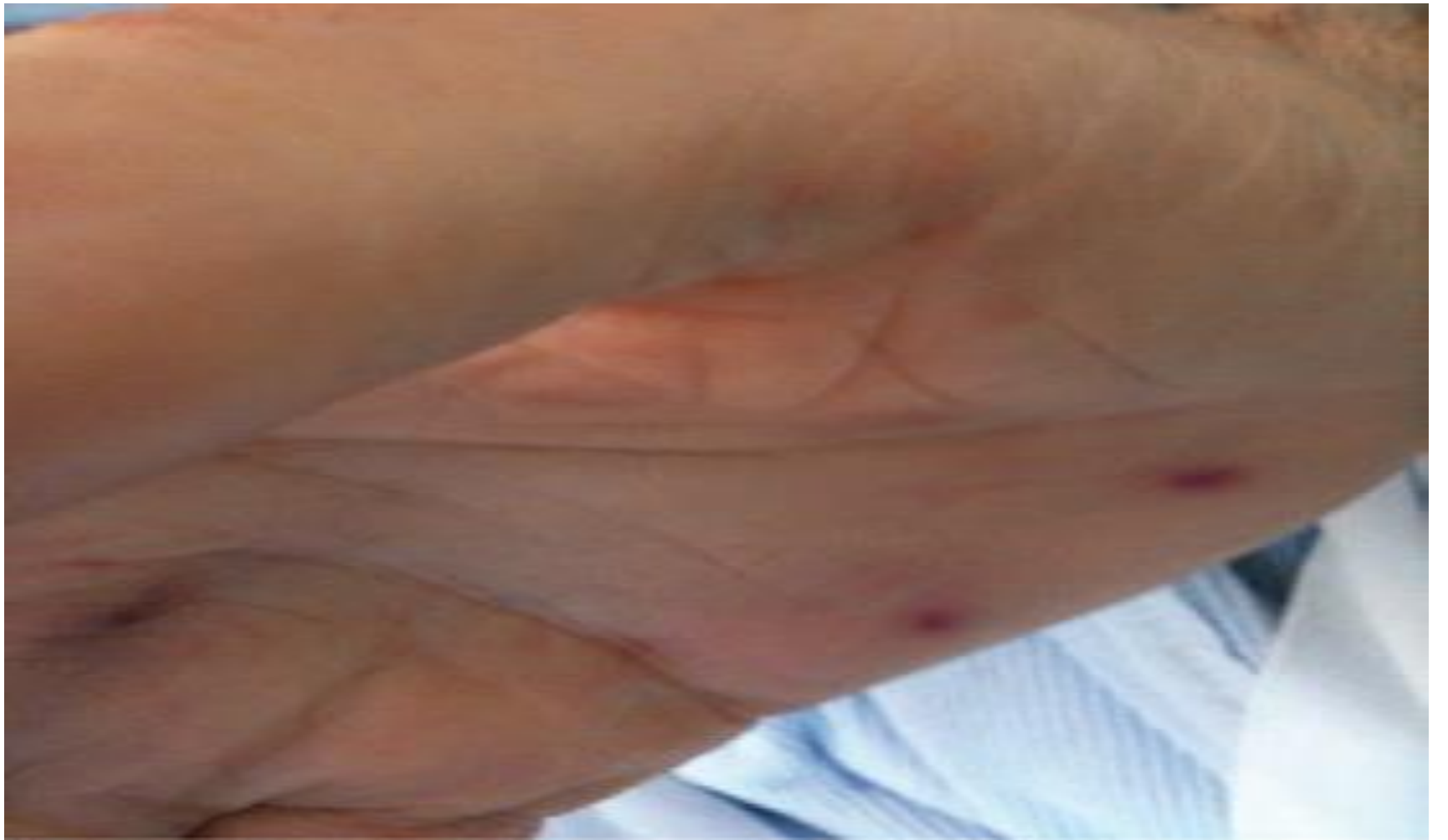


Carcinoid syndrome(chronic telangectasia)

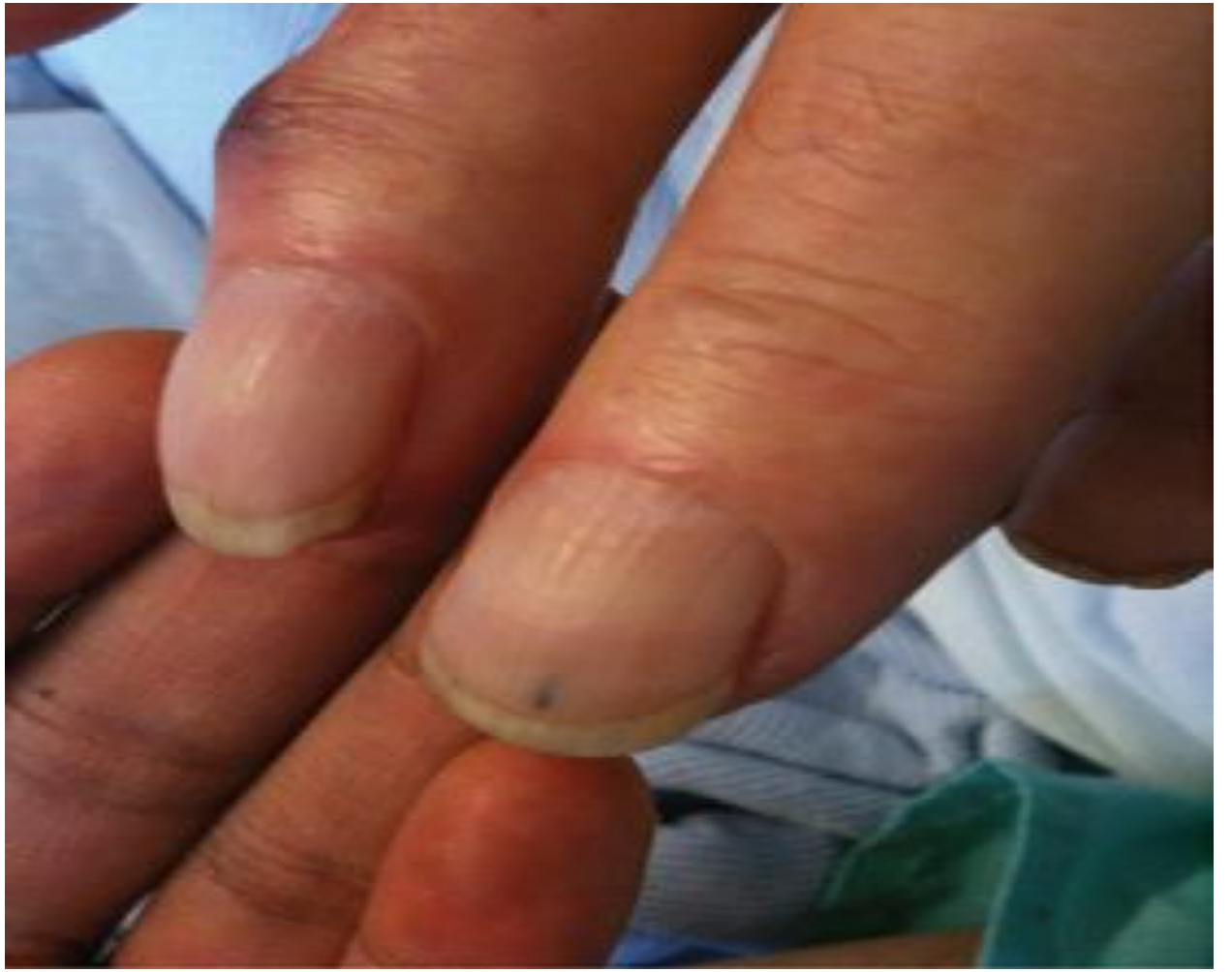


Cardiovascular system

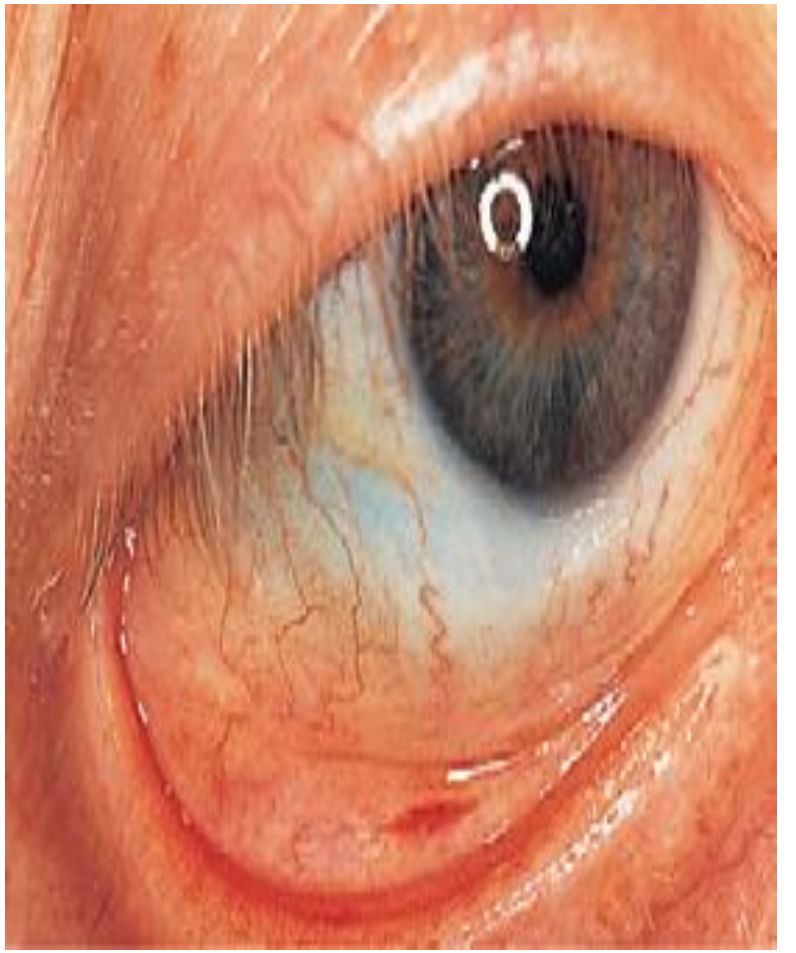
Infective endo.(janeway lesions on the hypothenar eminence)



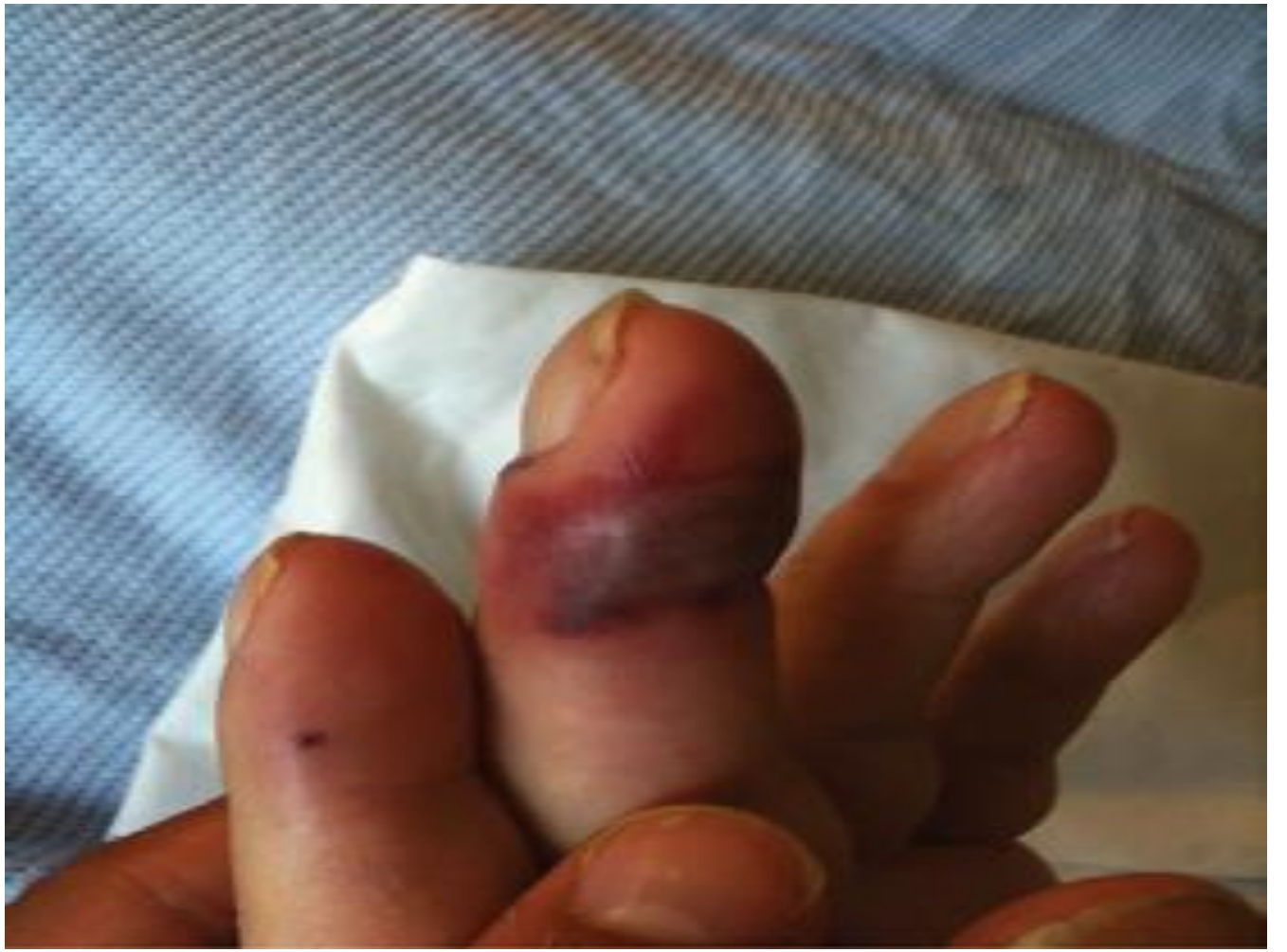
Splinter hemorrhage(infective endo)



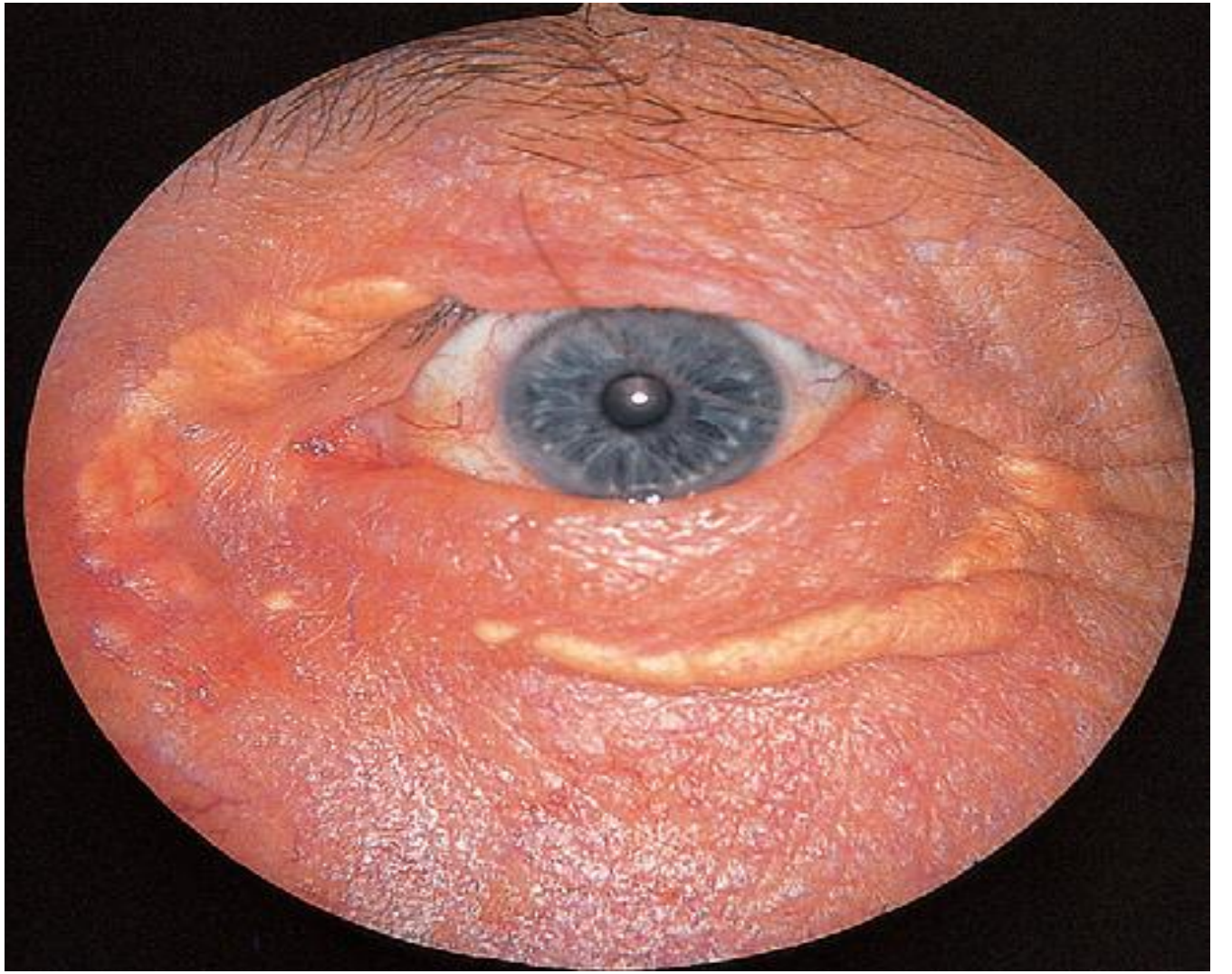
Petechial hemorrhage on conjunctiva(I.E)



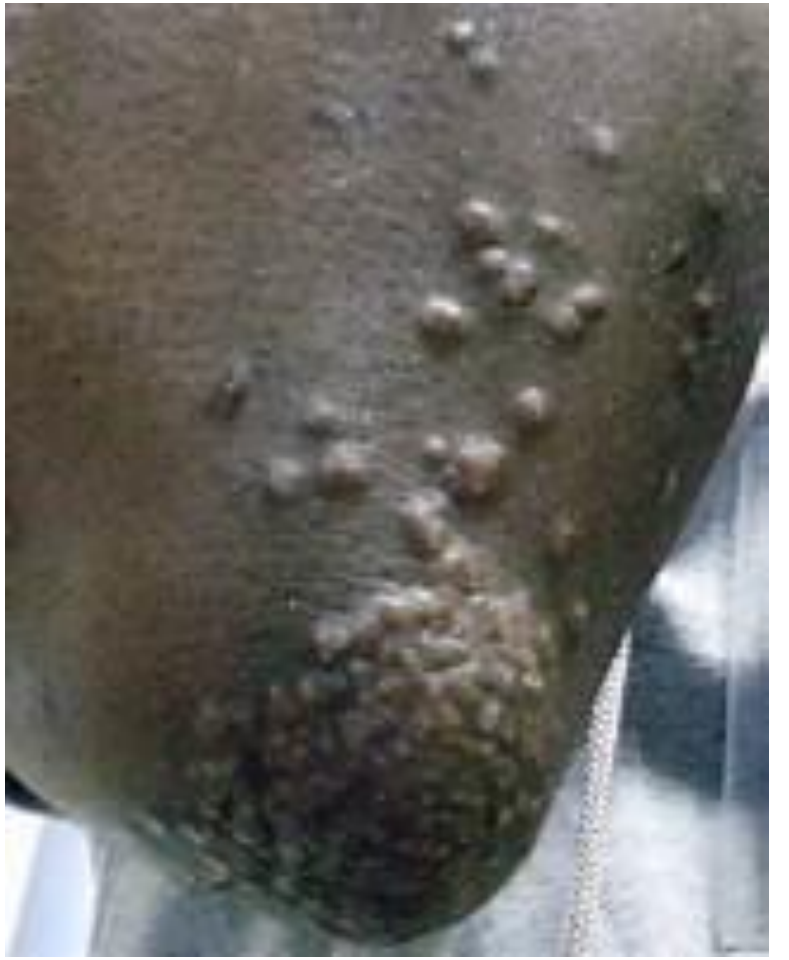
Osler nodes(IE)



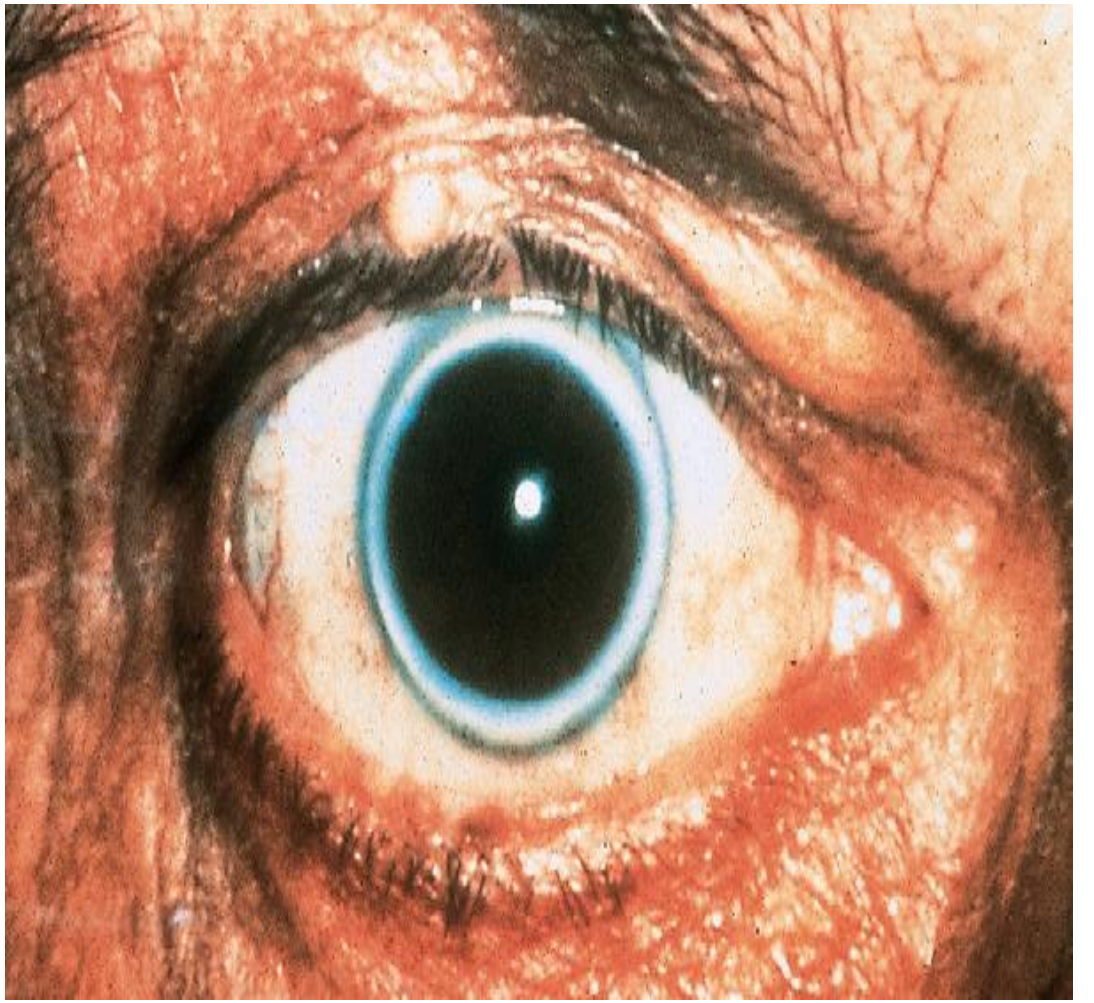
Periorbital xanthelsma



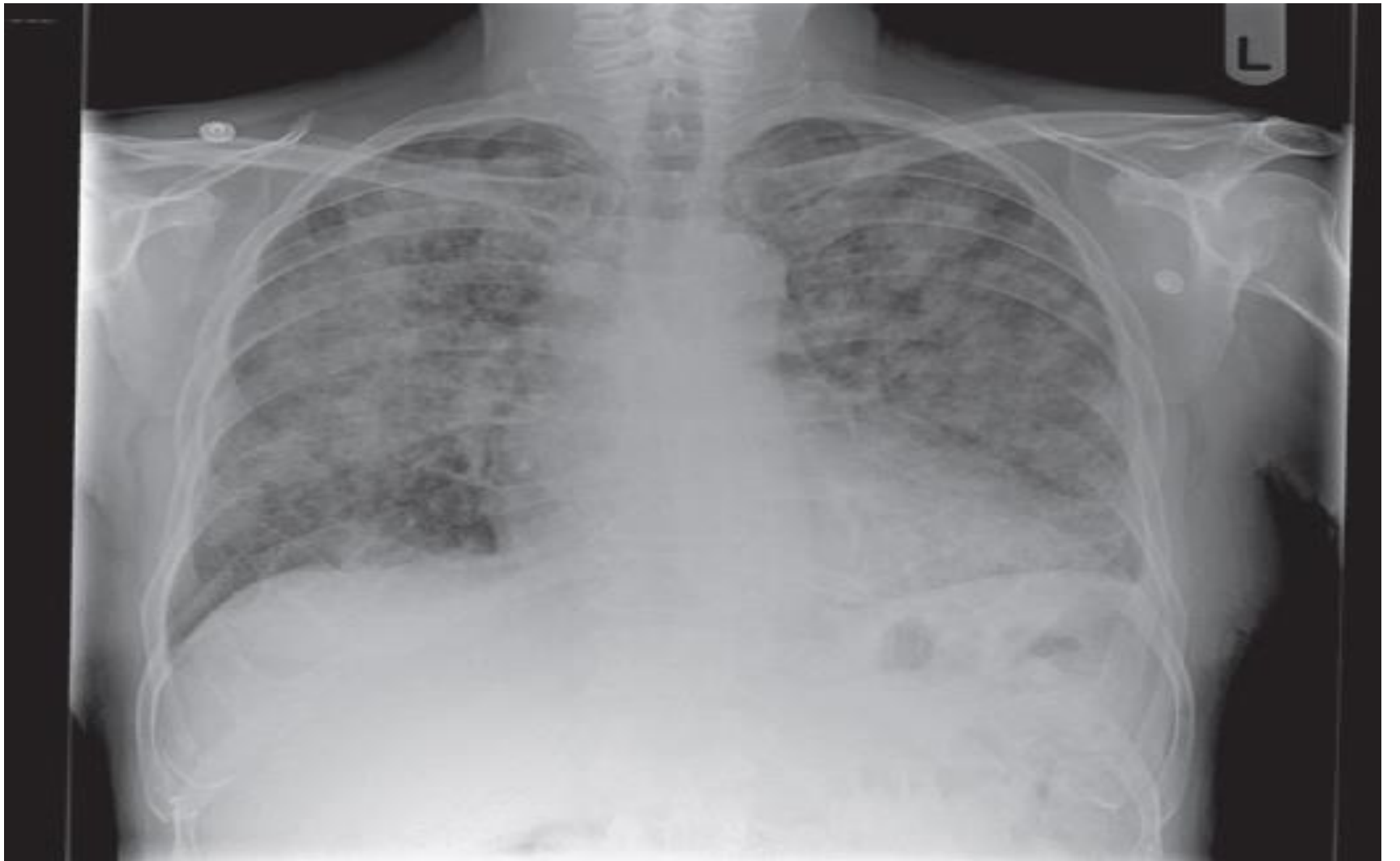
Skin xanthomata over the knee



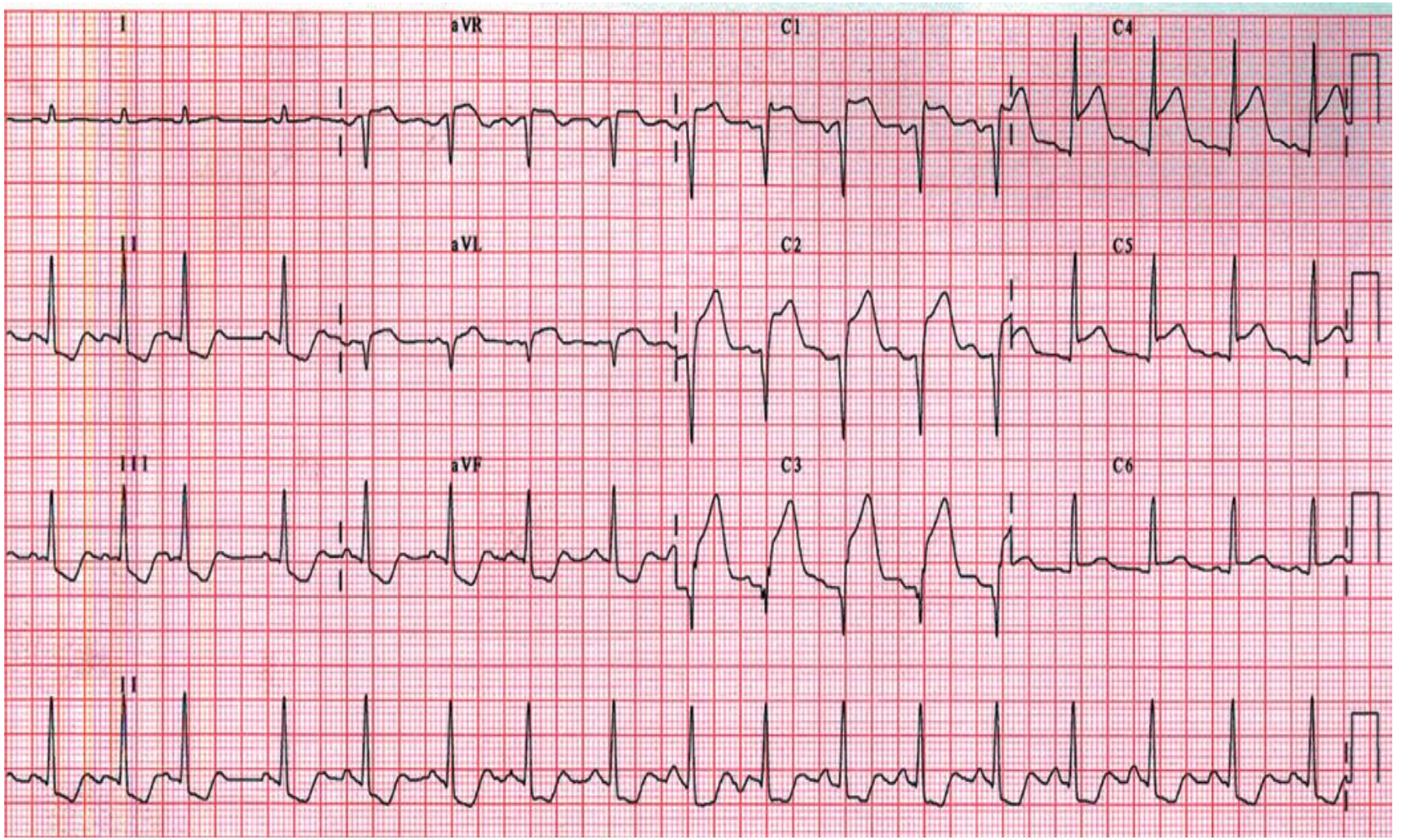
Corneal arcus



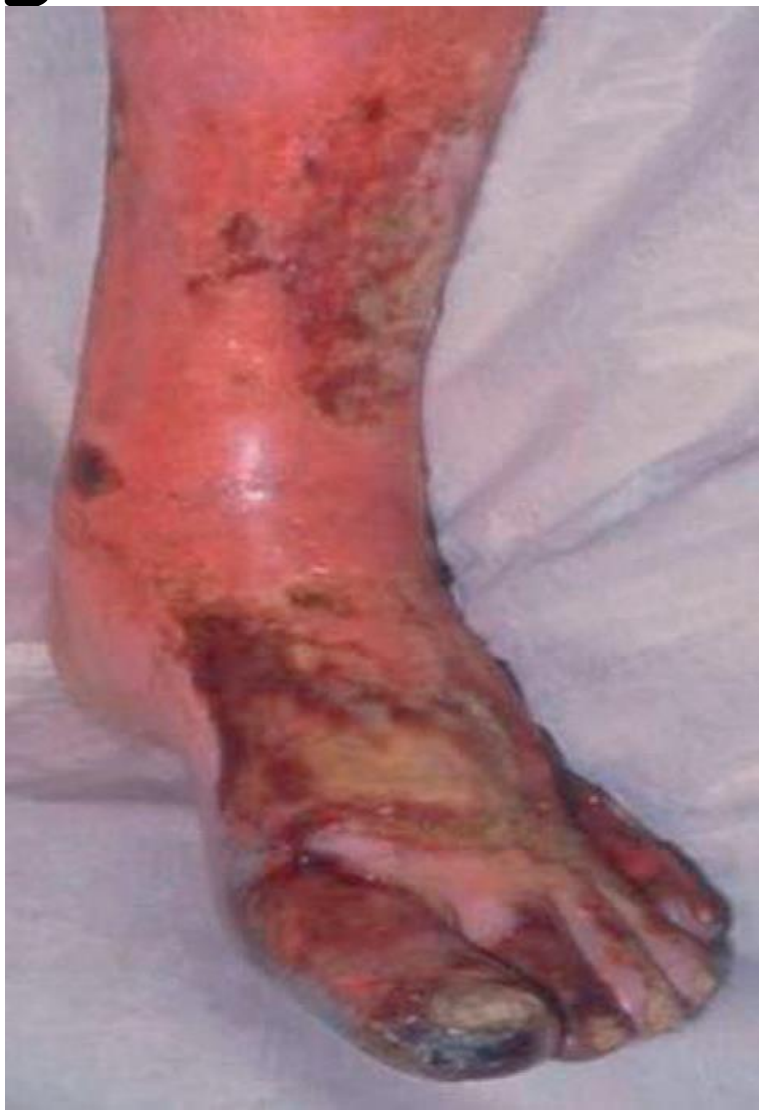
Chest X-ray in heart failure. This
shows cardiomegaly with patchy alveolar shadowing of pulmonary oedema
and Kerley B lines (engorged lymphatics) at the periphery of both lungs.



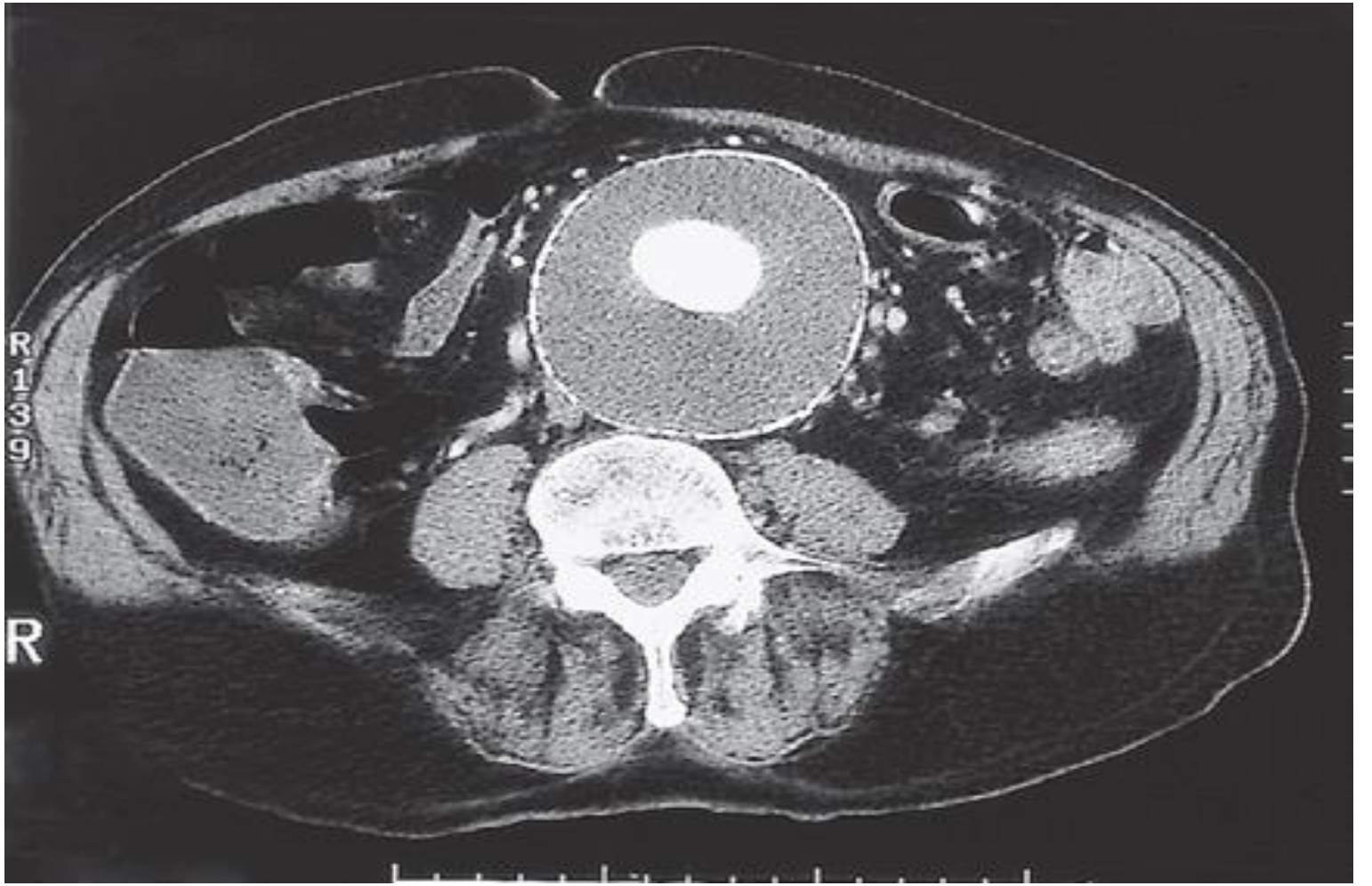
Acute anterior myocardial infarction



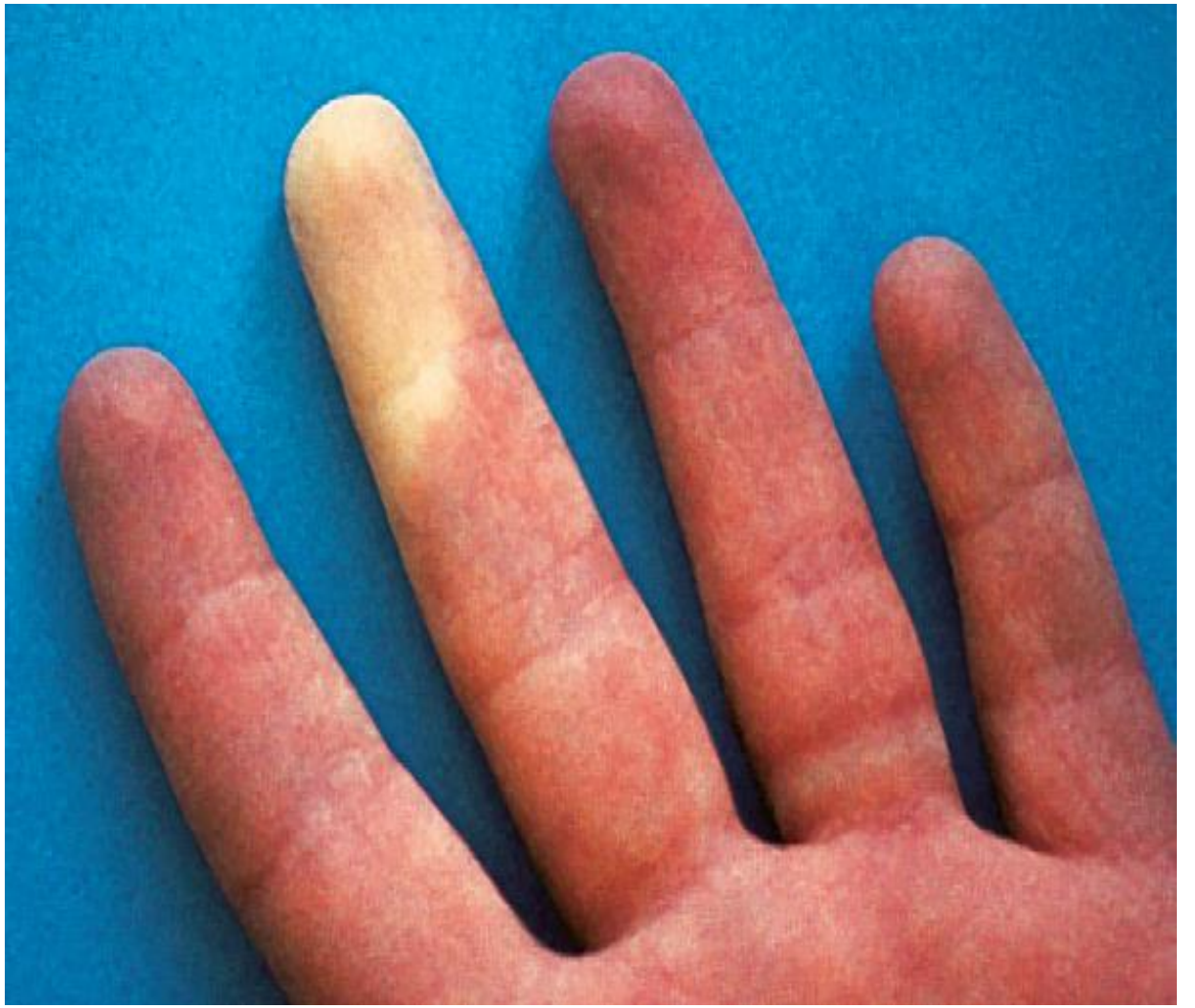
Gangrene of the foot



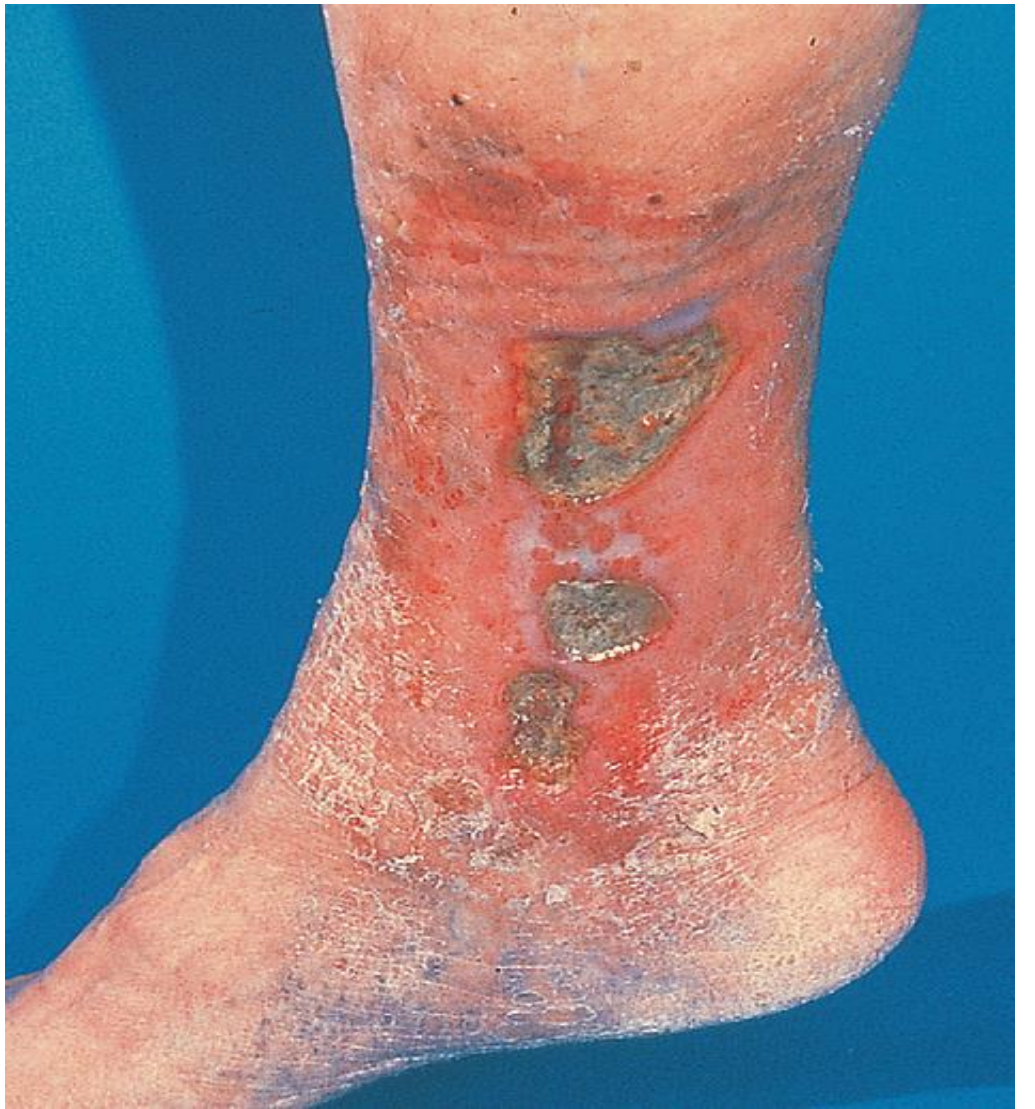
Abdominal aortic aneurysm



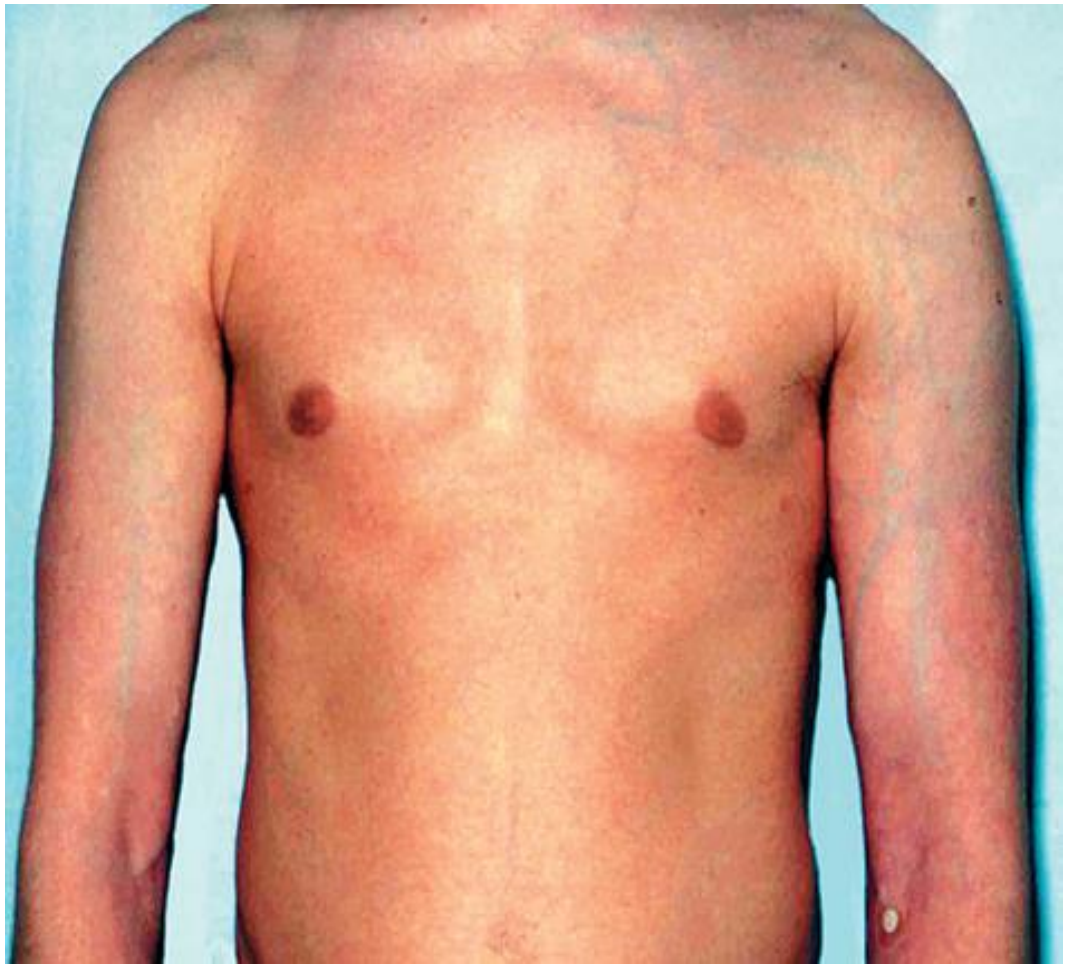
Raynaud's phenomenon



Venous ulceration

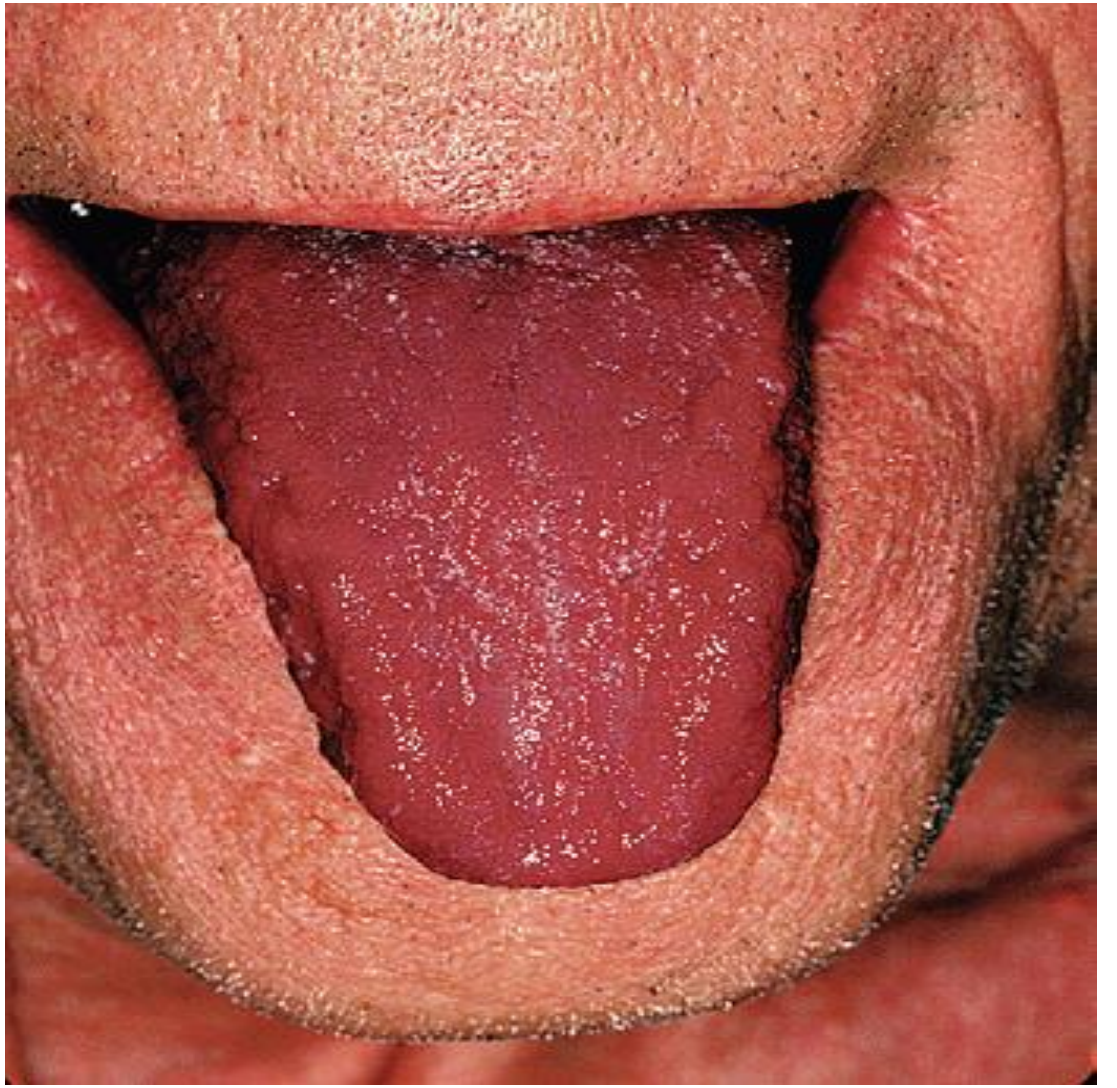


Axillary vein thrombosis
Clinical appearance with swollen left arm and dilated superficial veins.

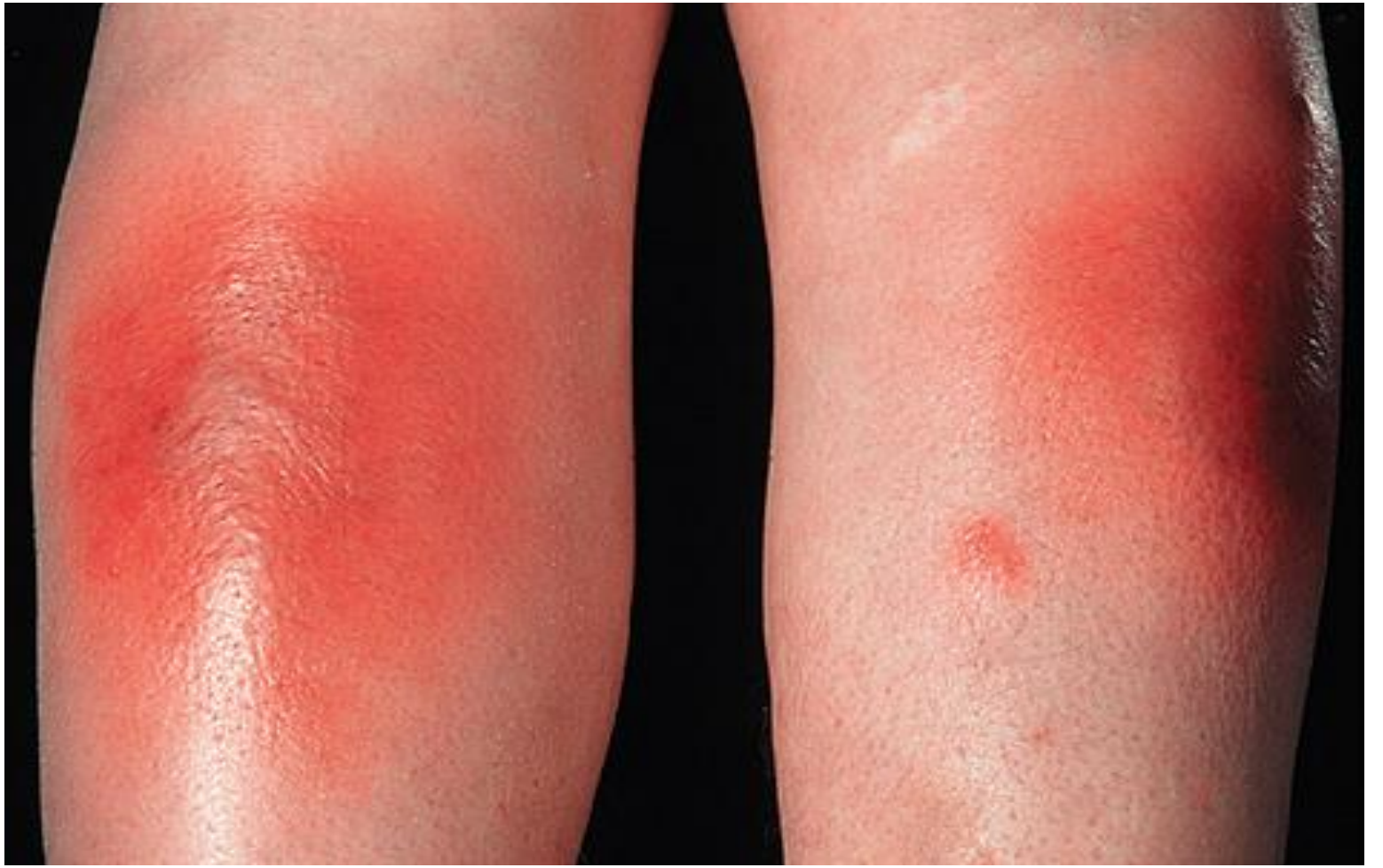


Respiratory system

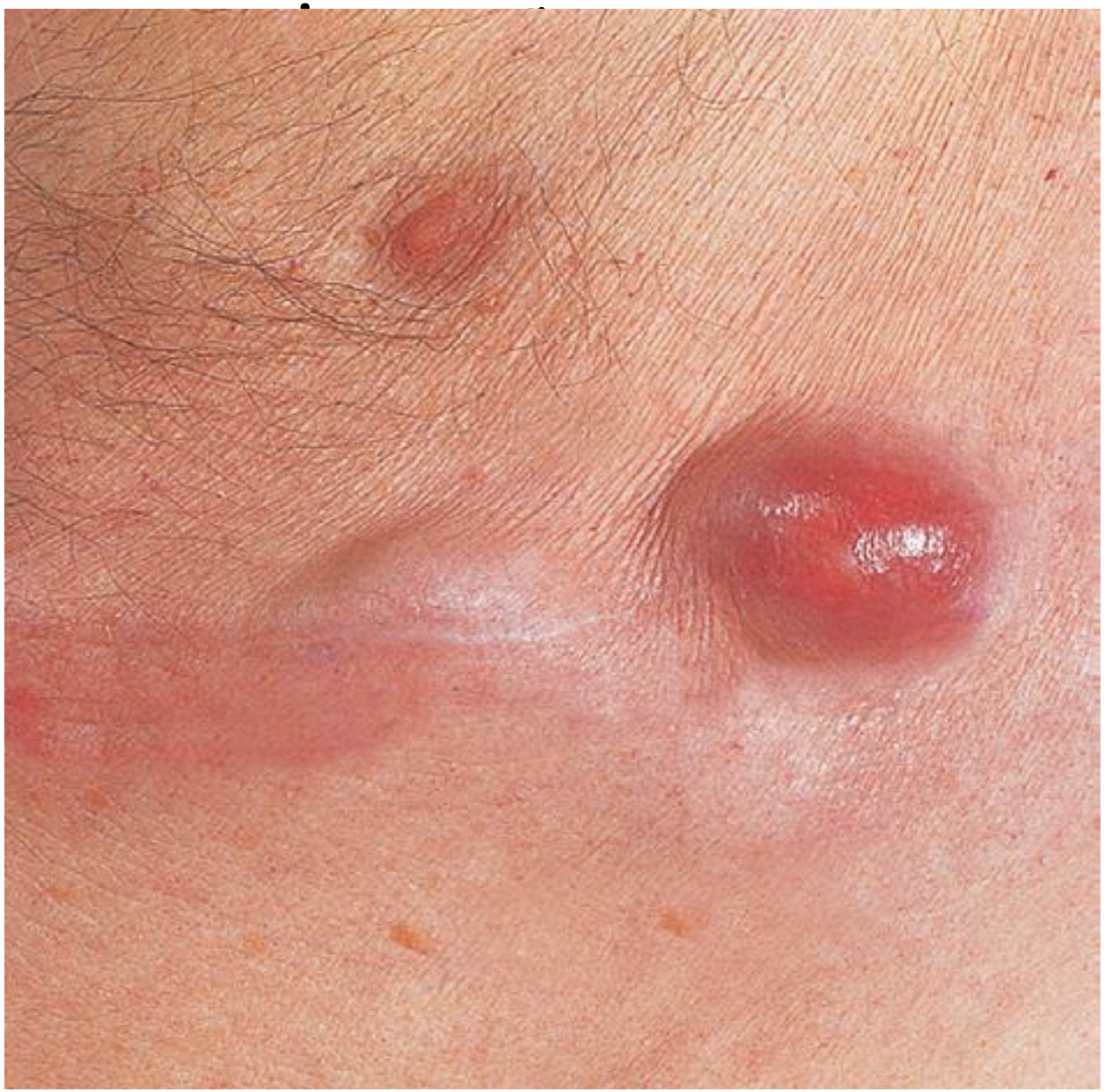
Central cyanosis of the tongue



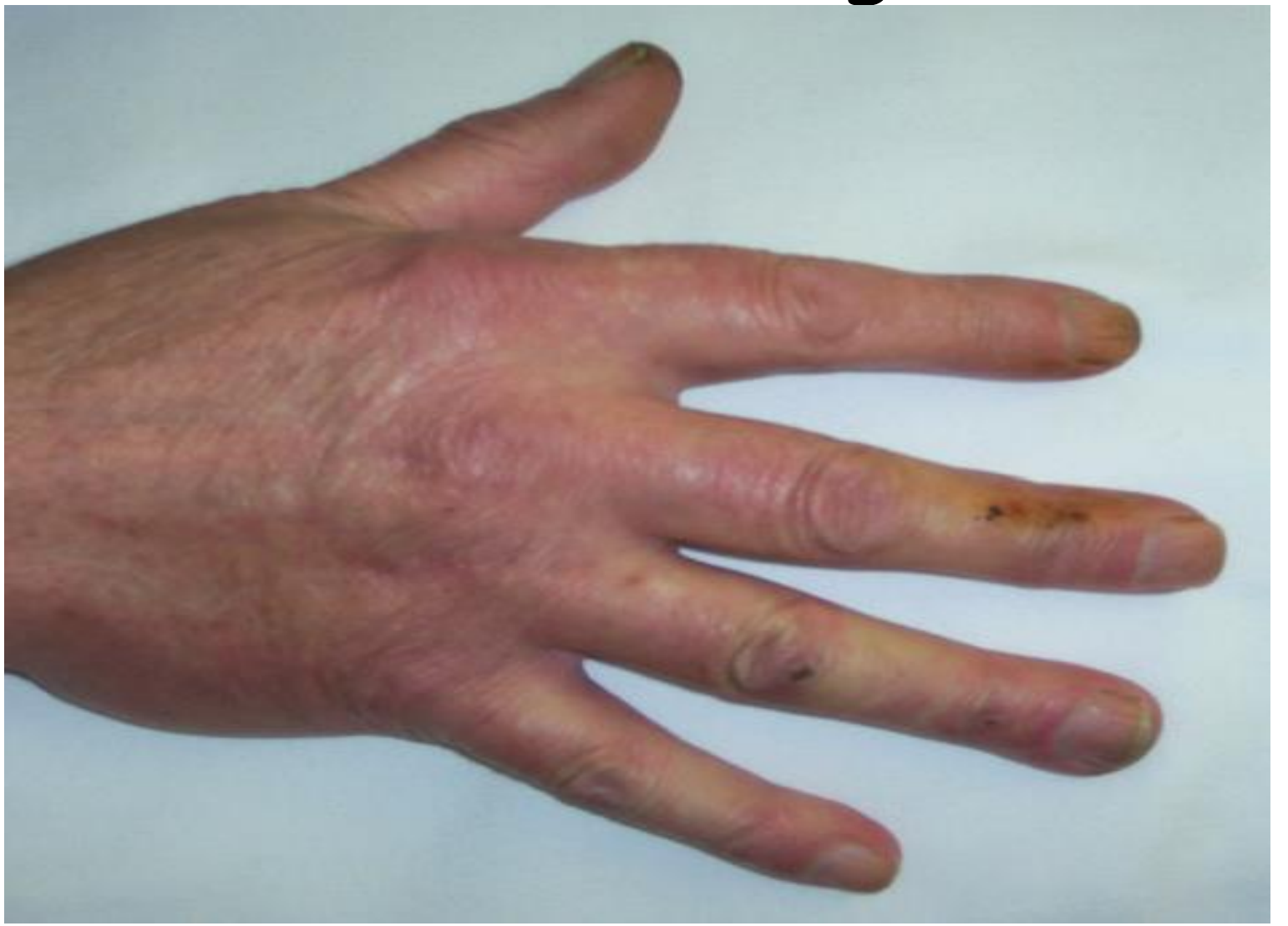
Erythema nodosum



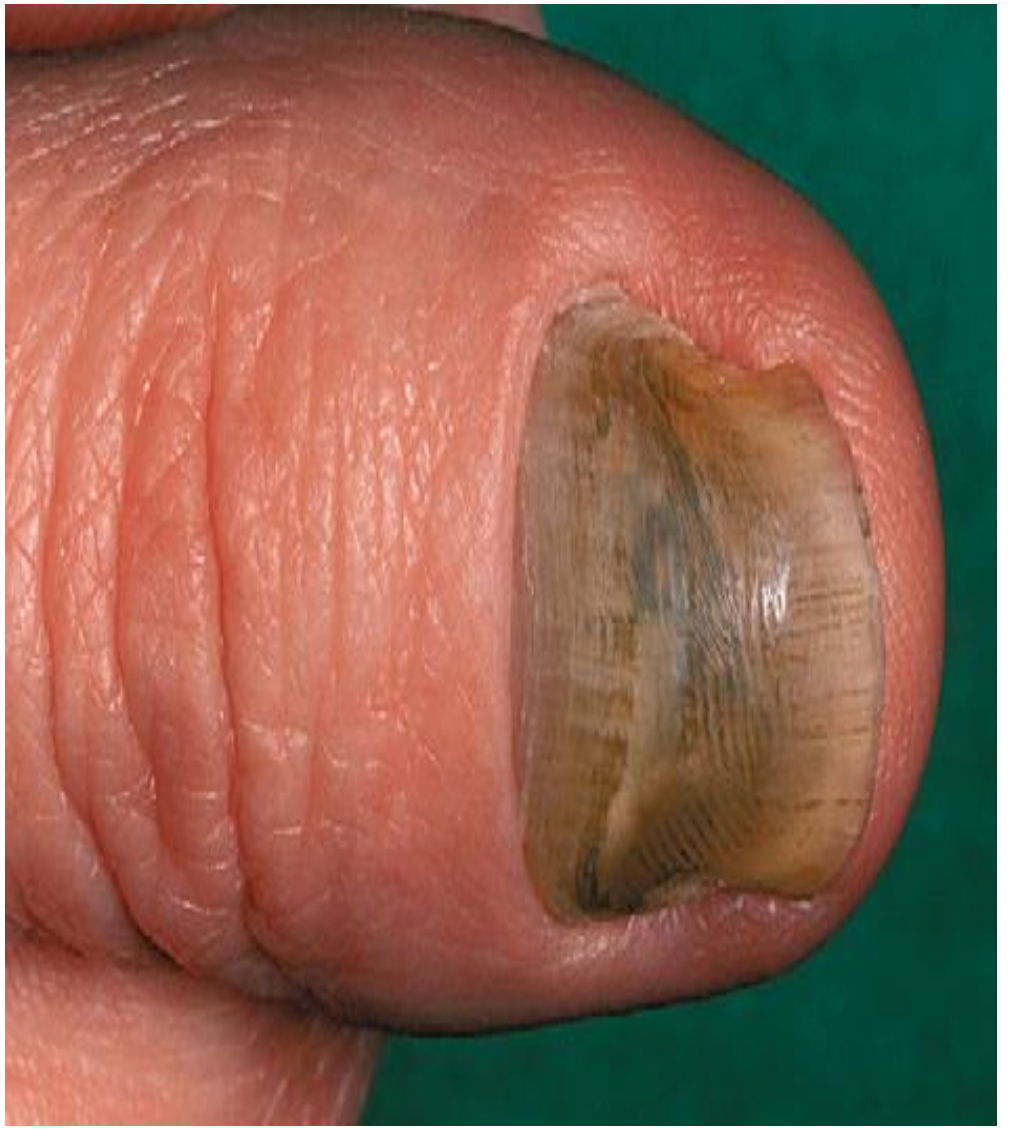
Metastatic skin nodes of

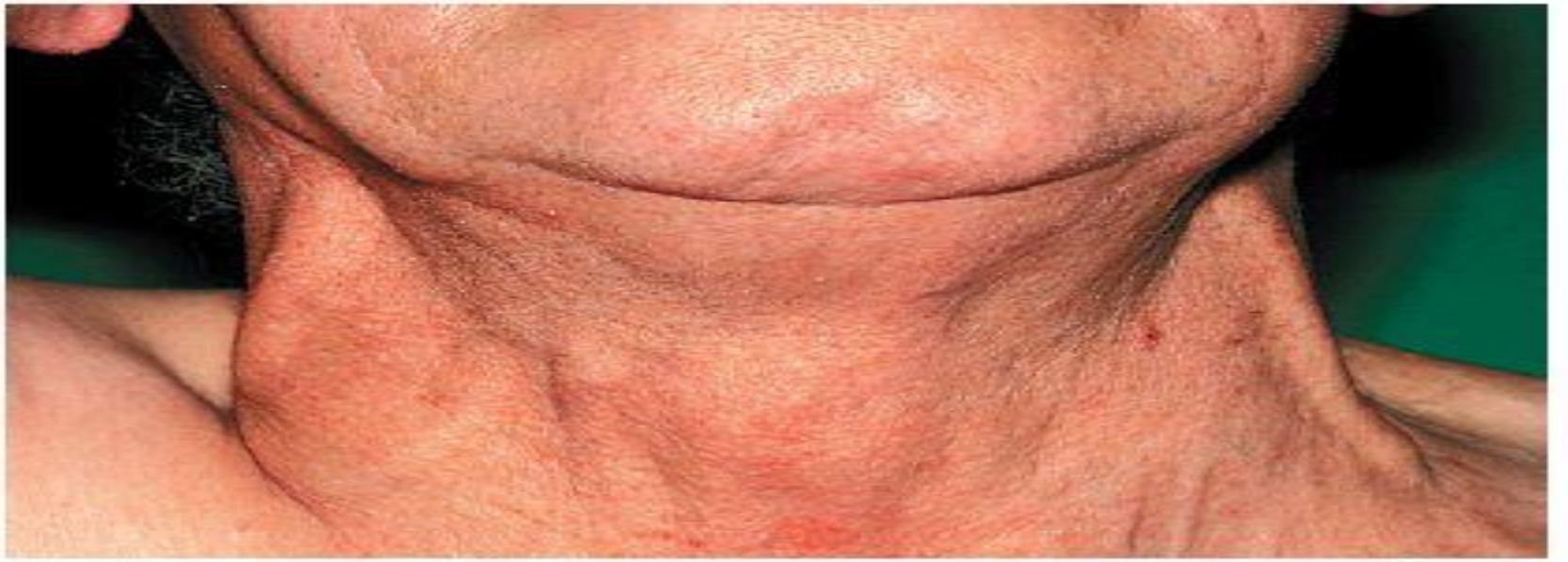


Tar staining

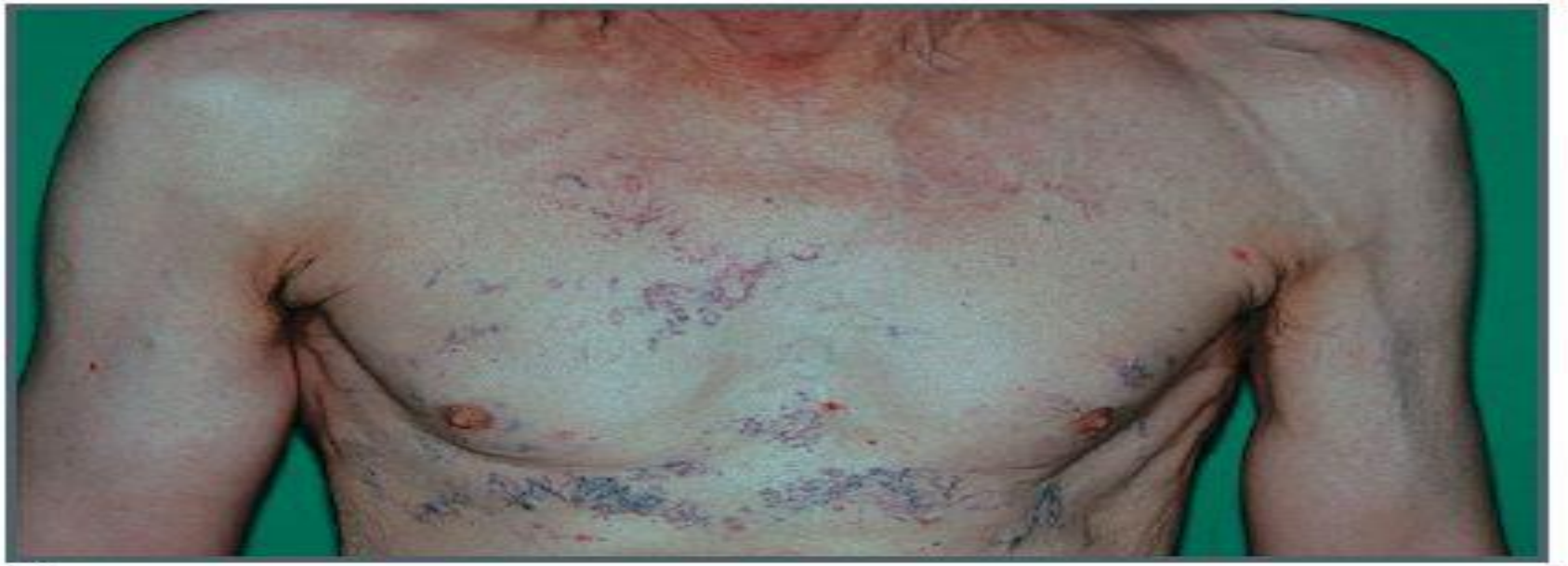


Yellow nail syndrome





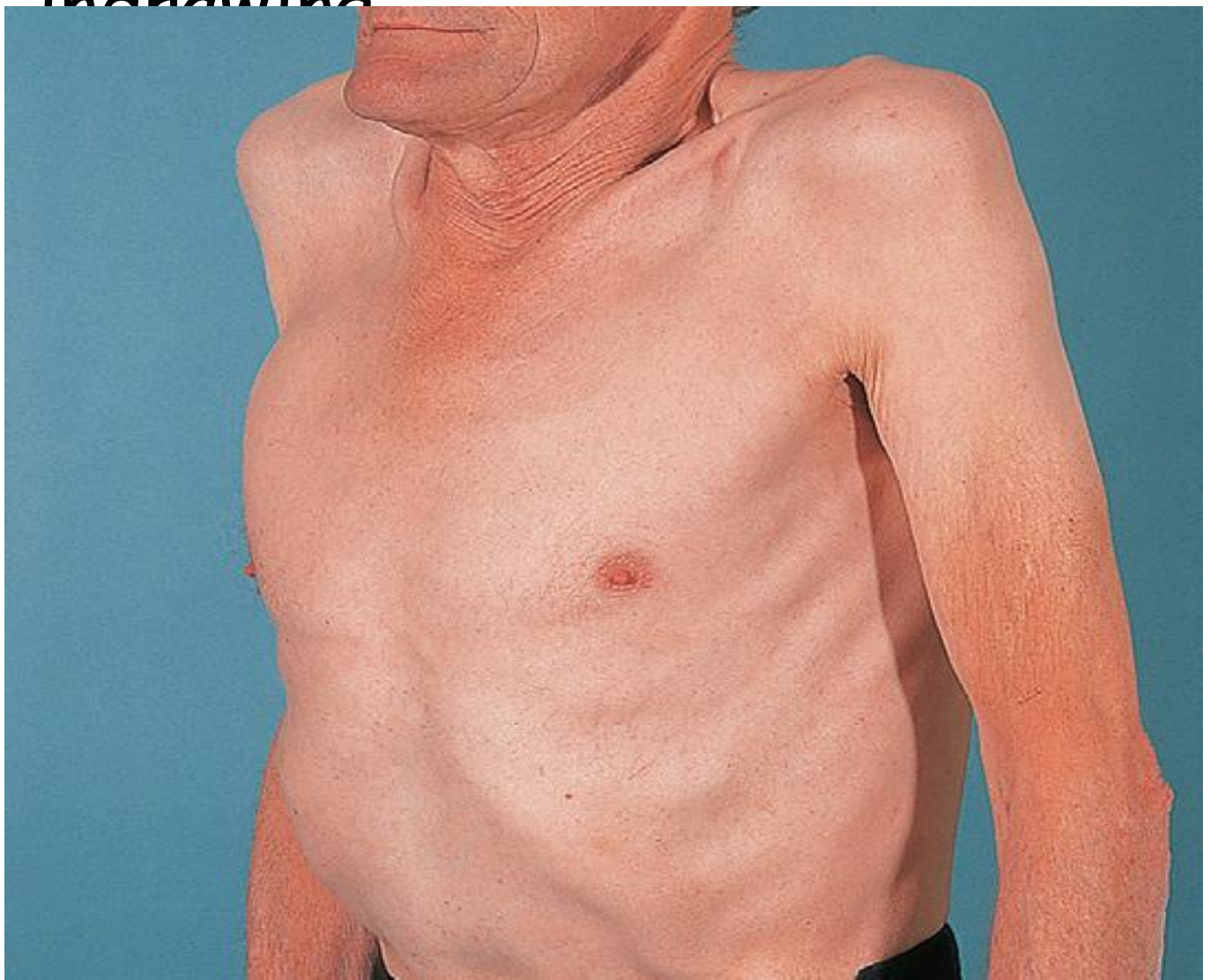
A



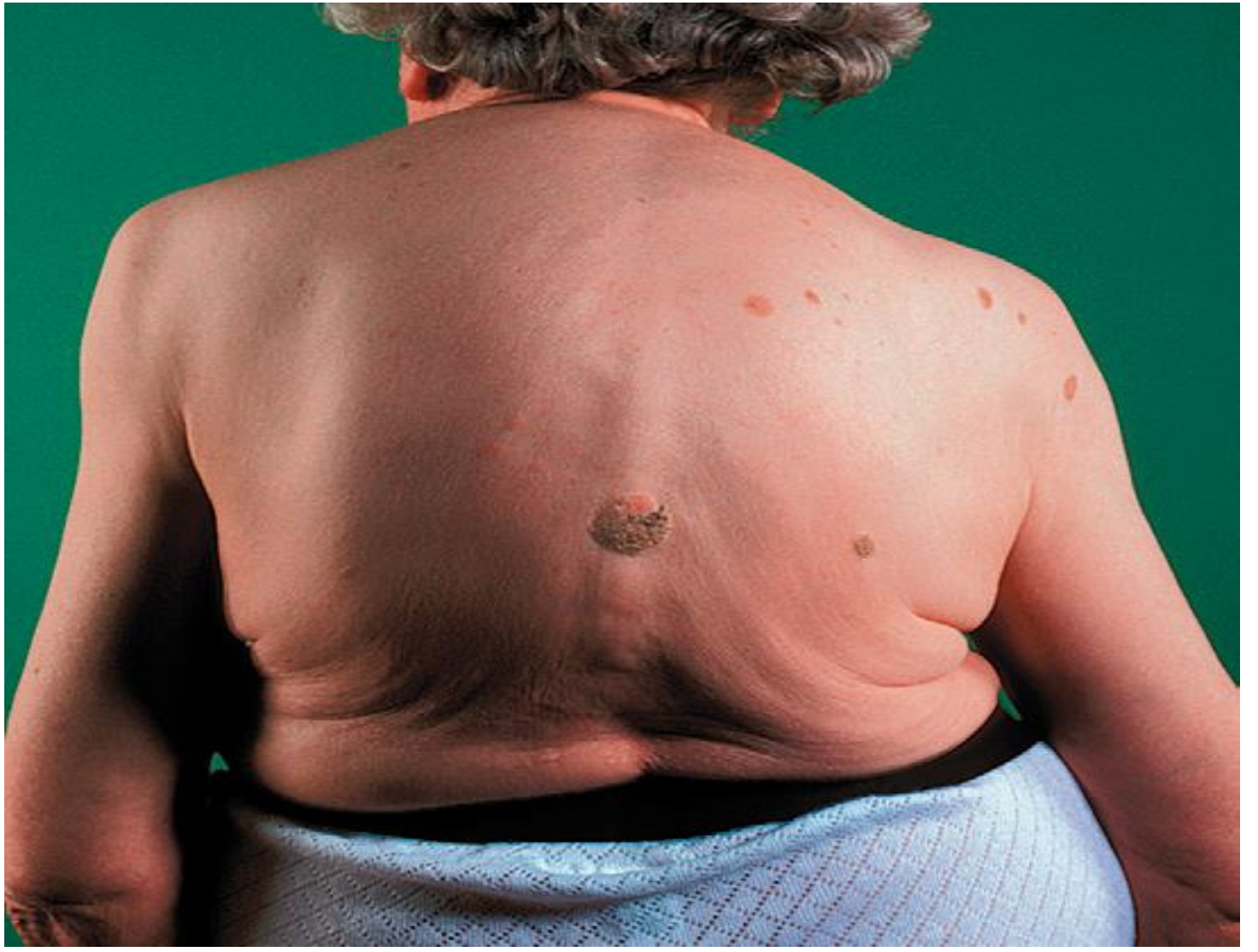
B

Fig. 7.11 Superior vena caval obstruction. (A) Distended neck veins. **(B)** Dilated superficial veins over chest.

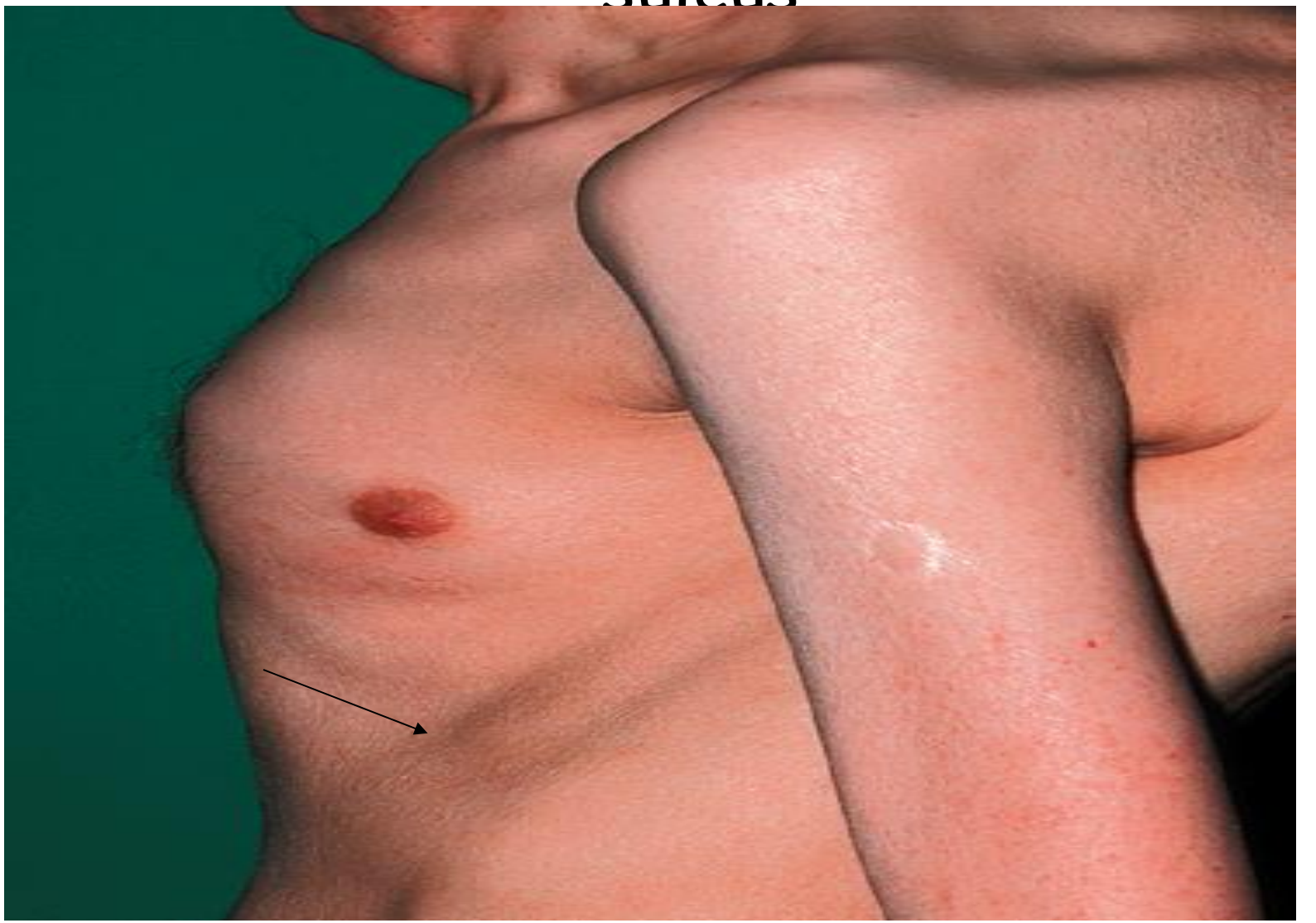
Hyperinflated chest with intercostal indrawing



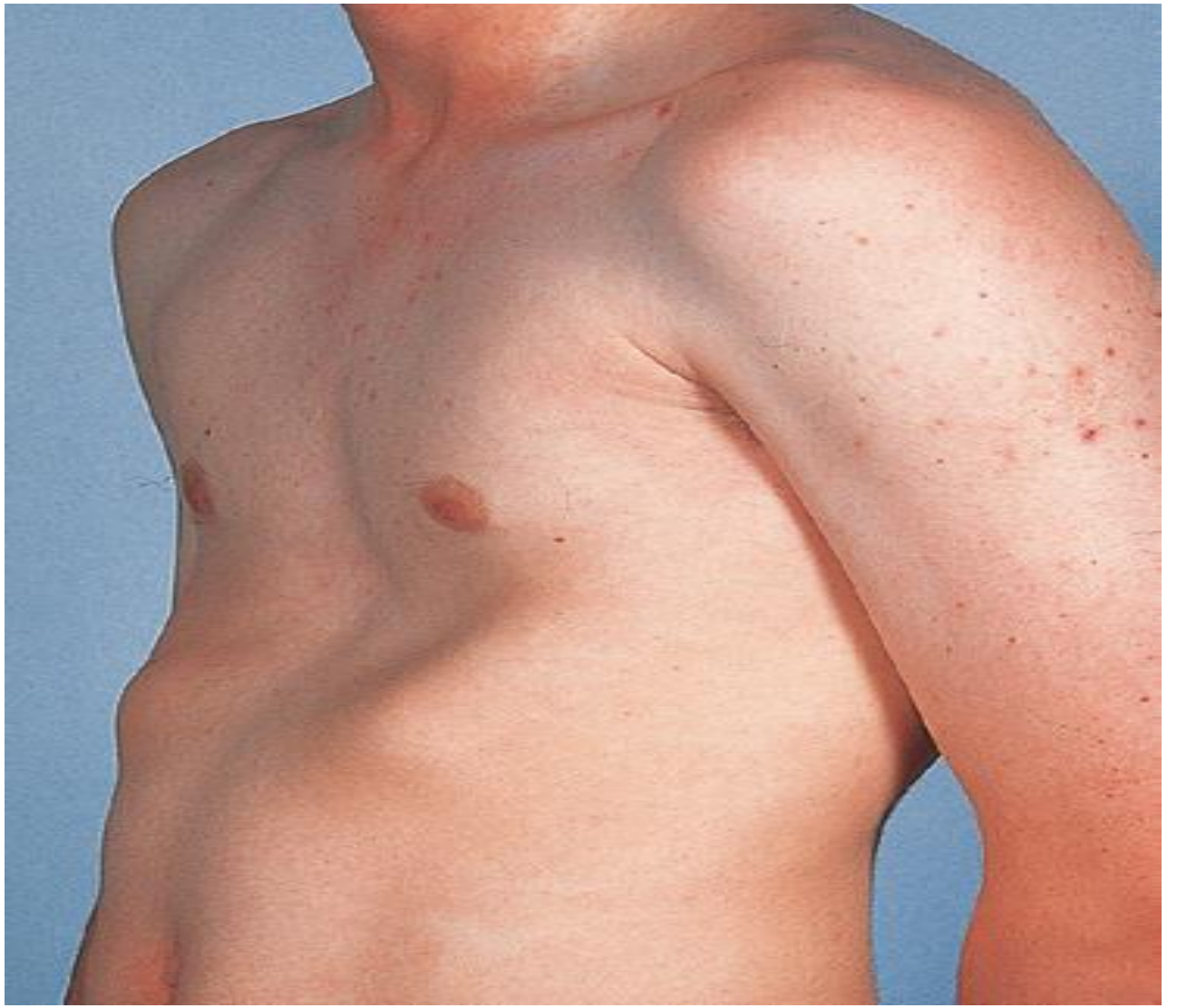
kyphoscoliosis



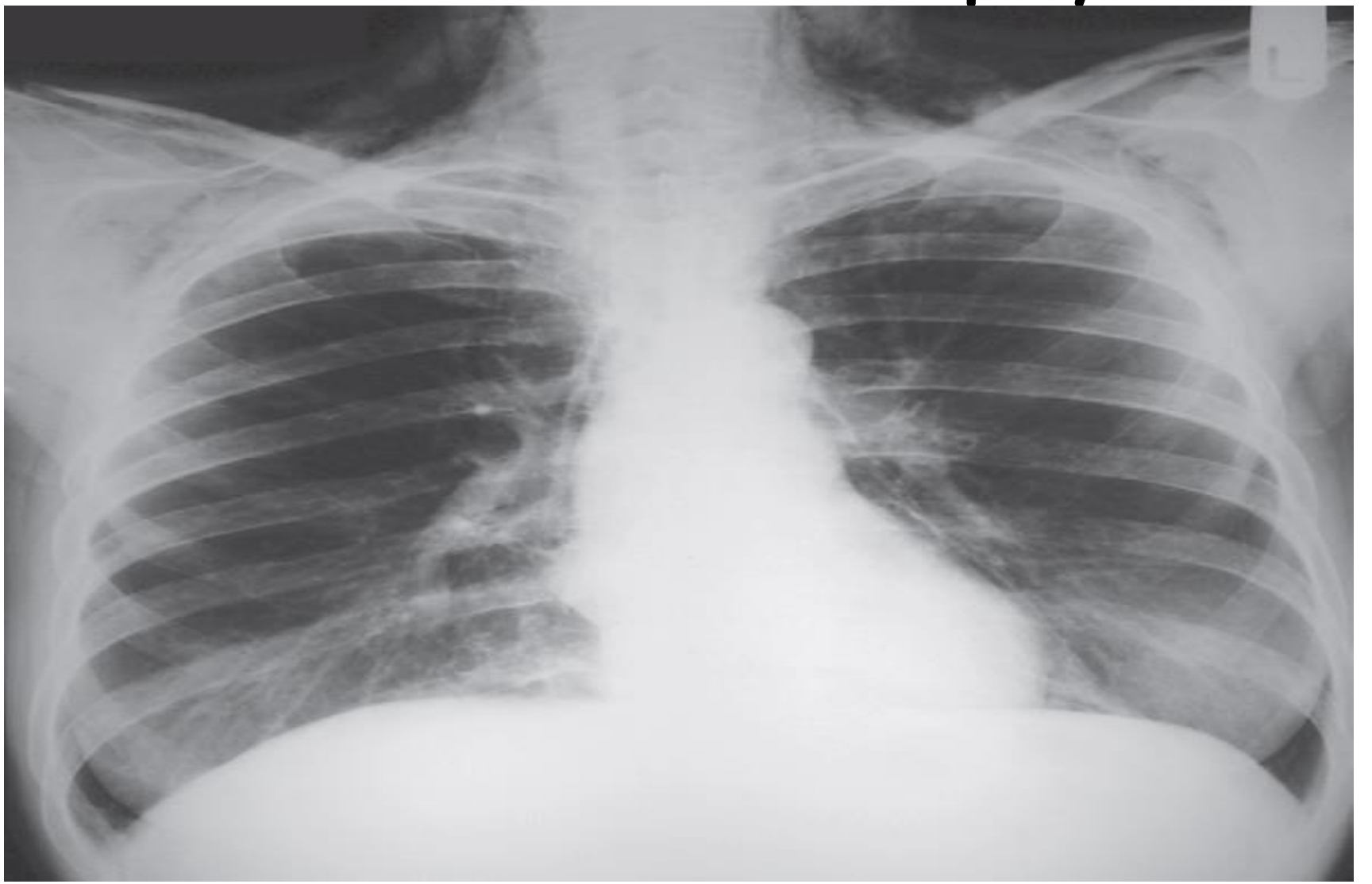
Pectus carinatum with harrison sulcus



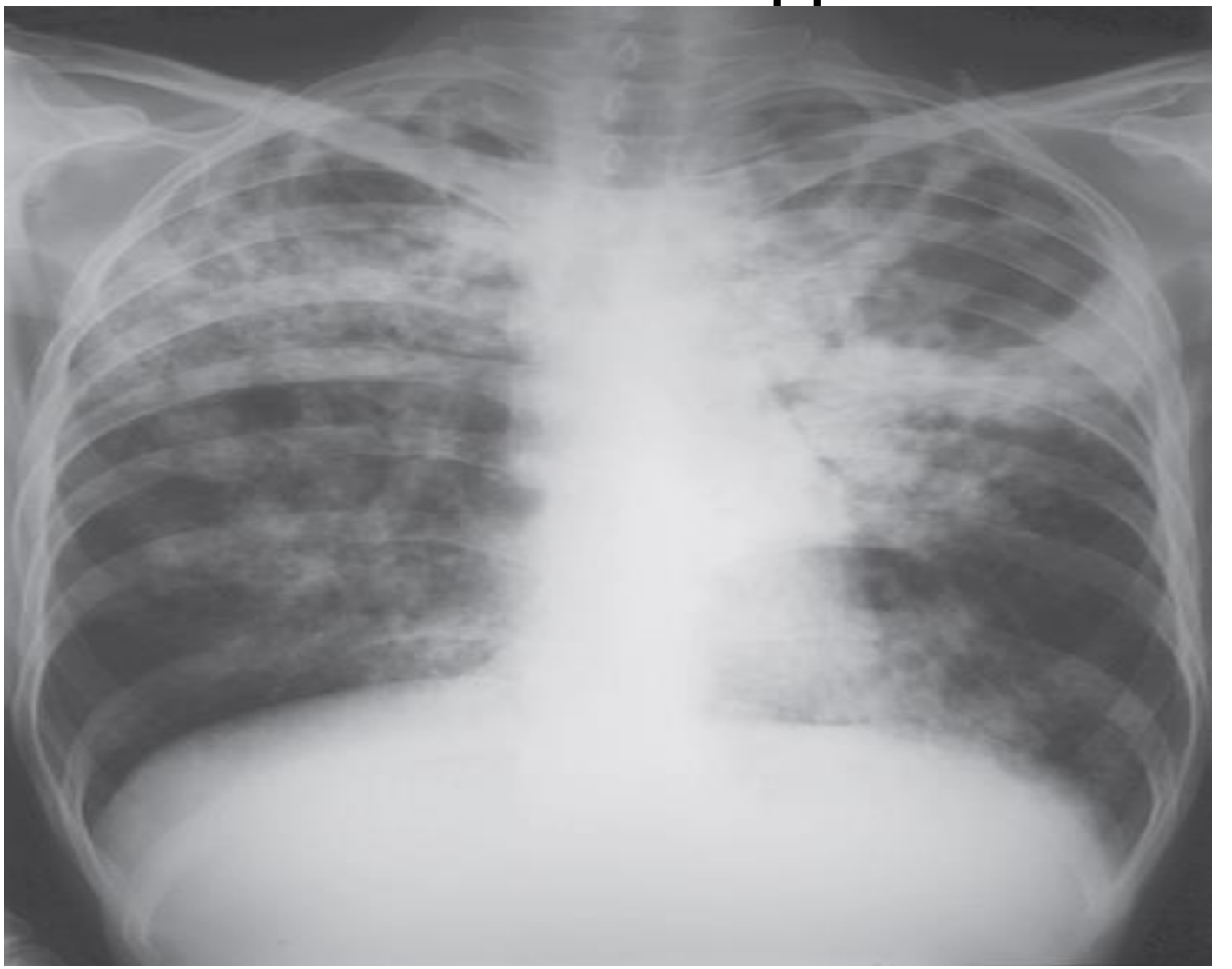
Pectus excavatum



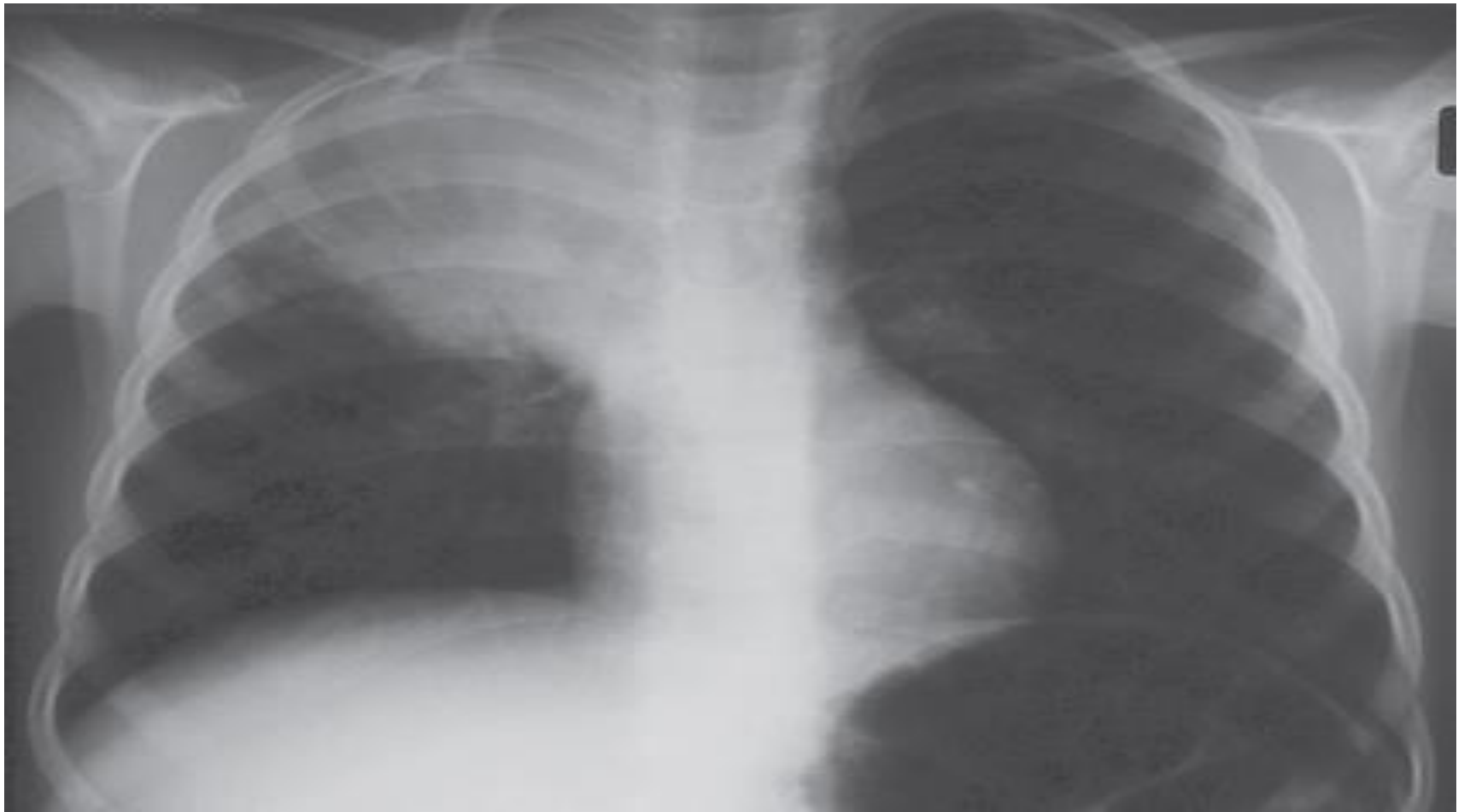
Subcutaneous emphysema



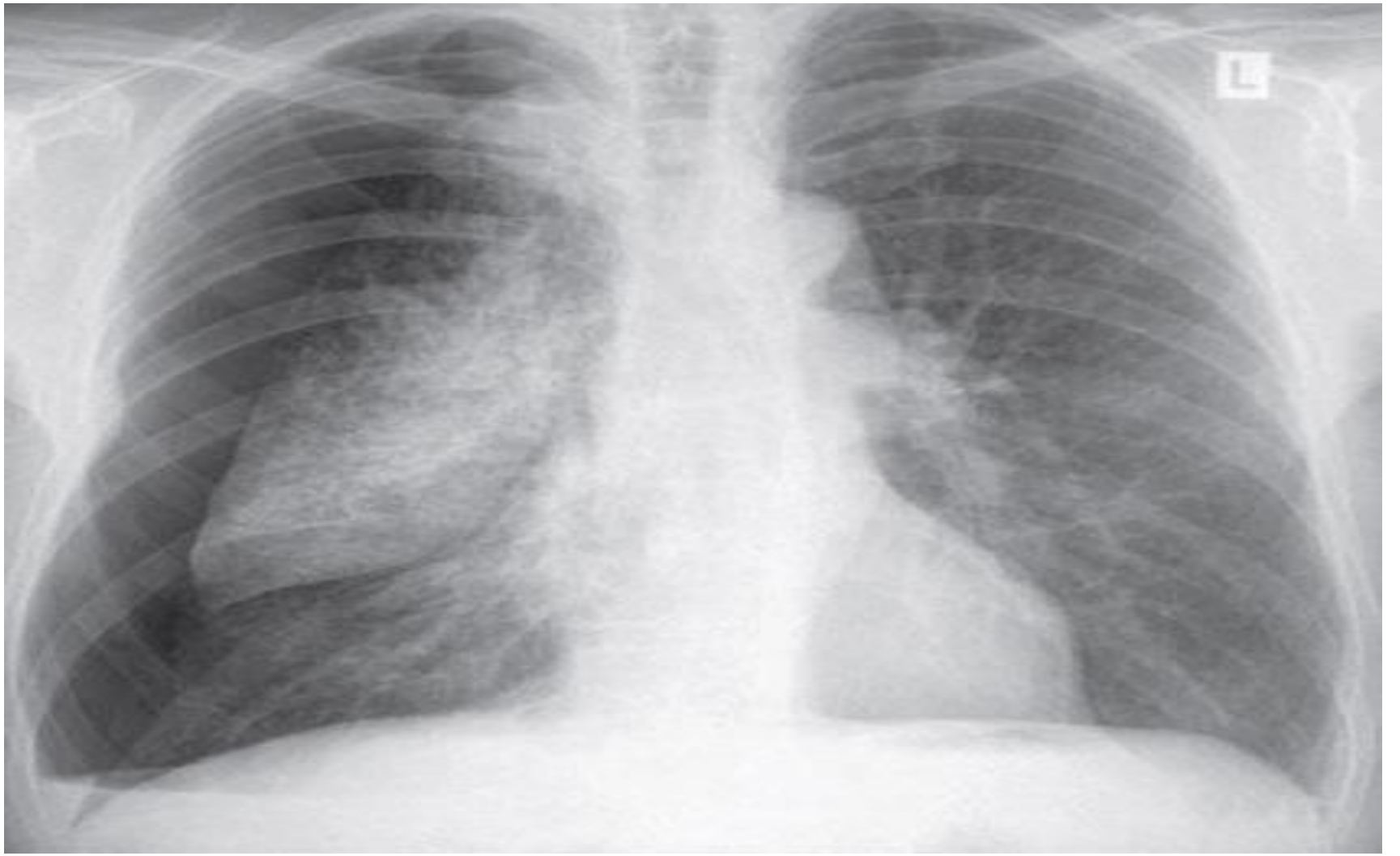
Tuberculosis: consolidation and cavitation in both upper zones.



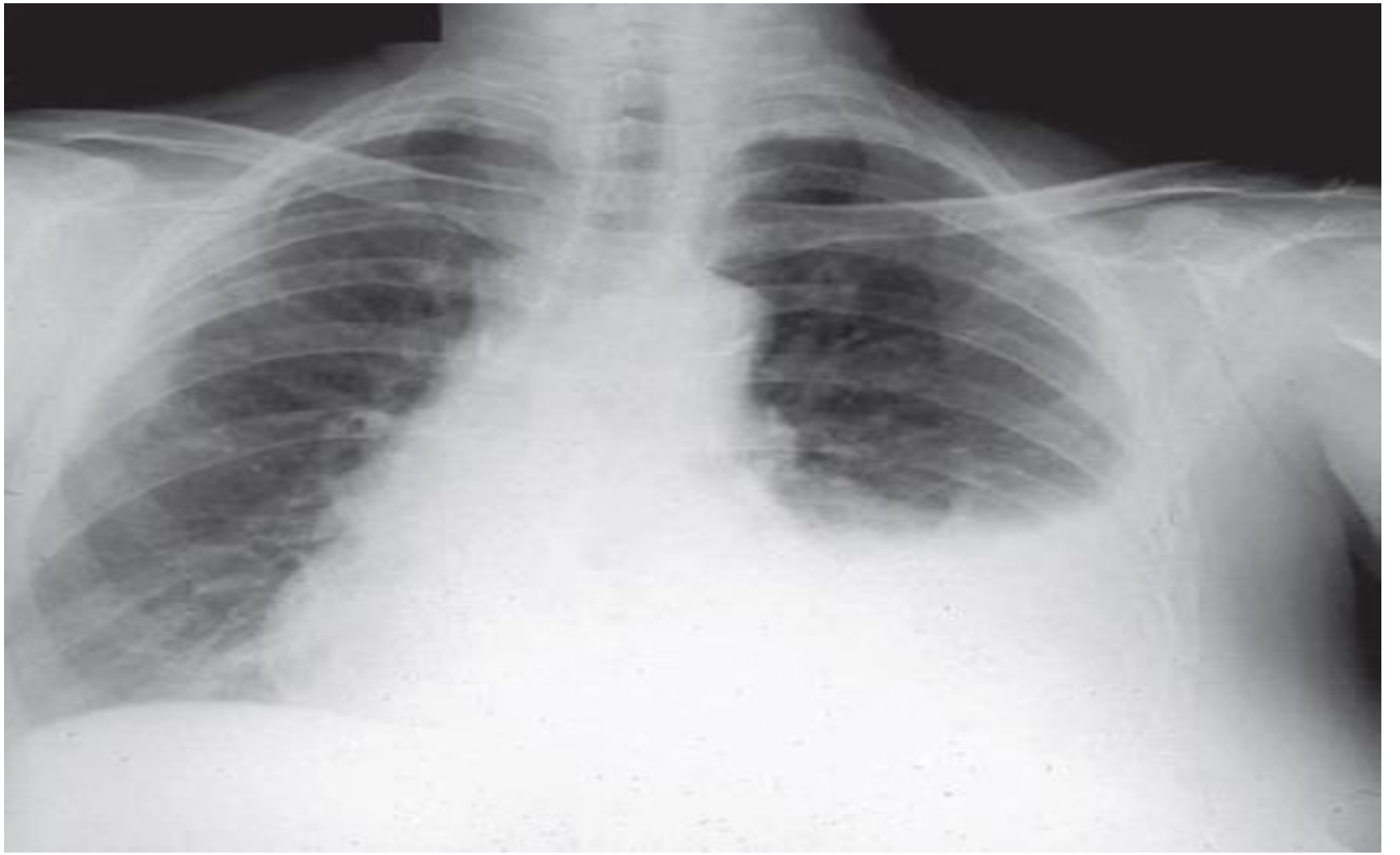
Right upper-lobe pneumonia containing air bronchograms.



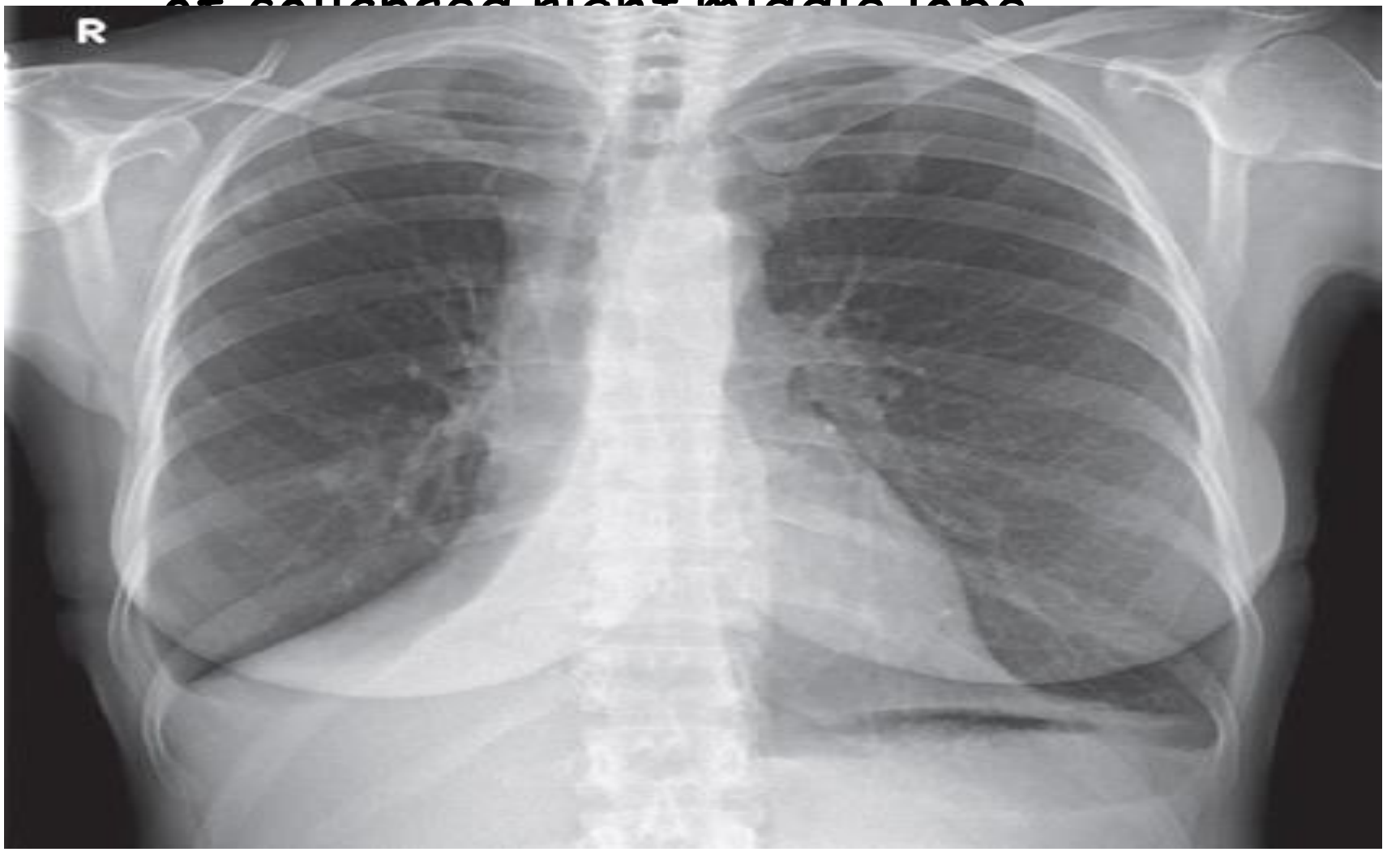
Right pneumothorax.



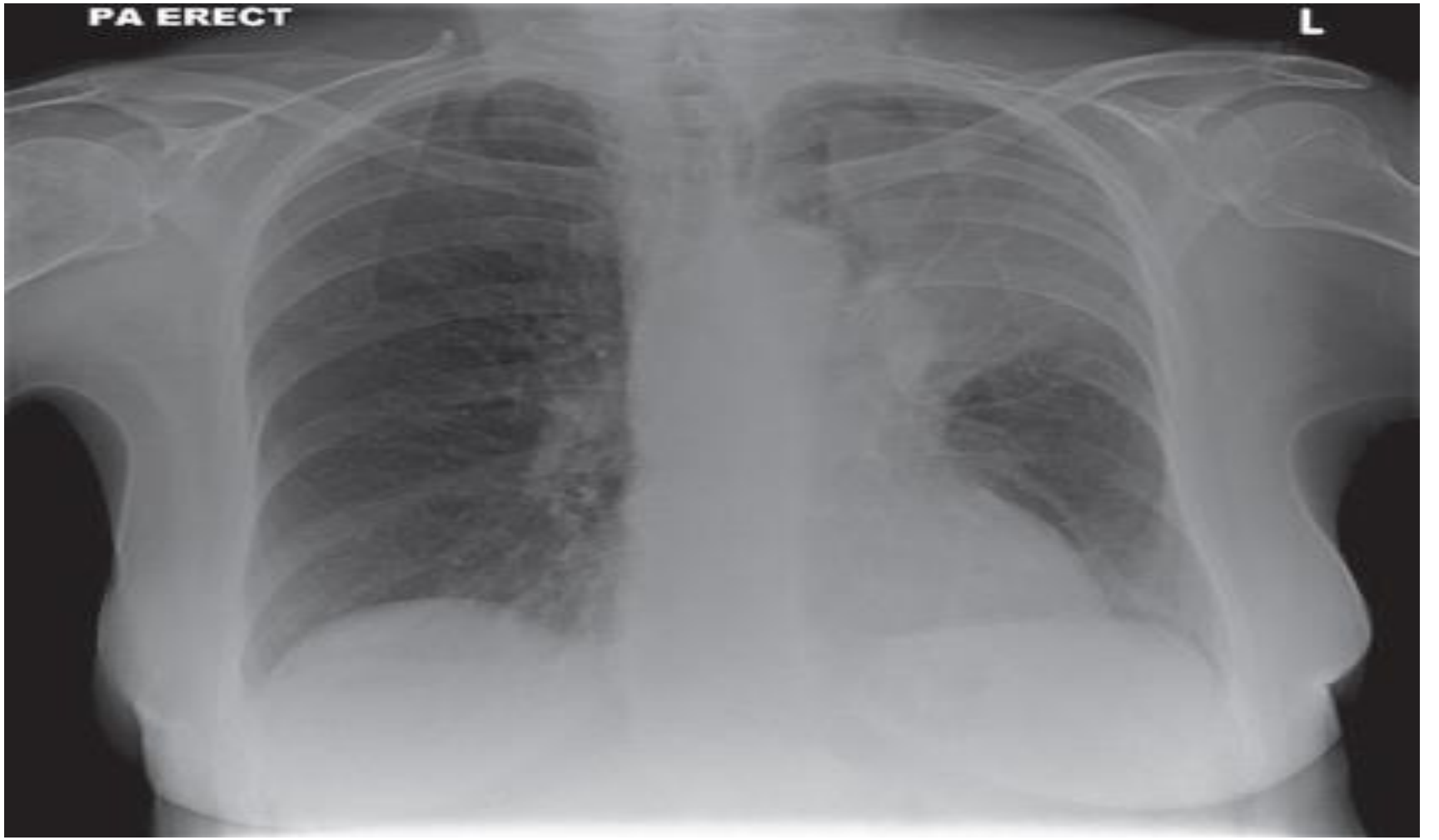
Left pleural effusion



Posteroanterior chest X-ray showing straight line
of collapsed right middle lobe

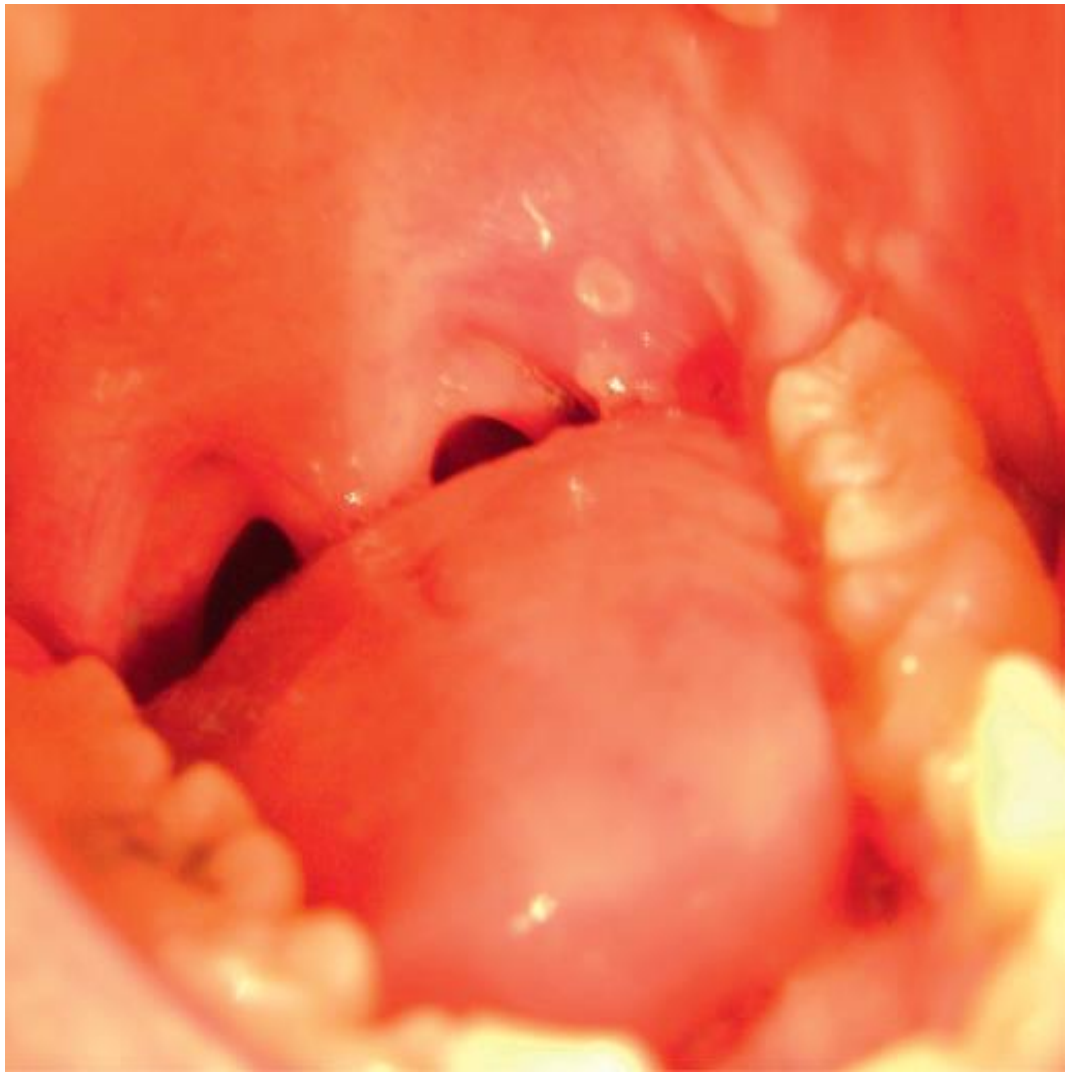


Left upper-lobe collapse.

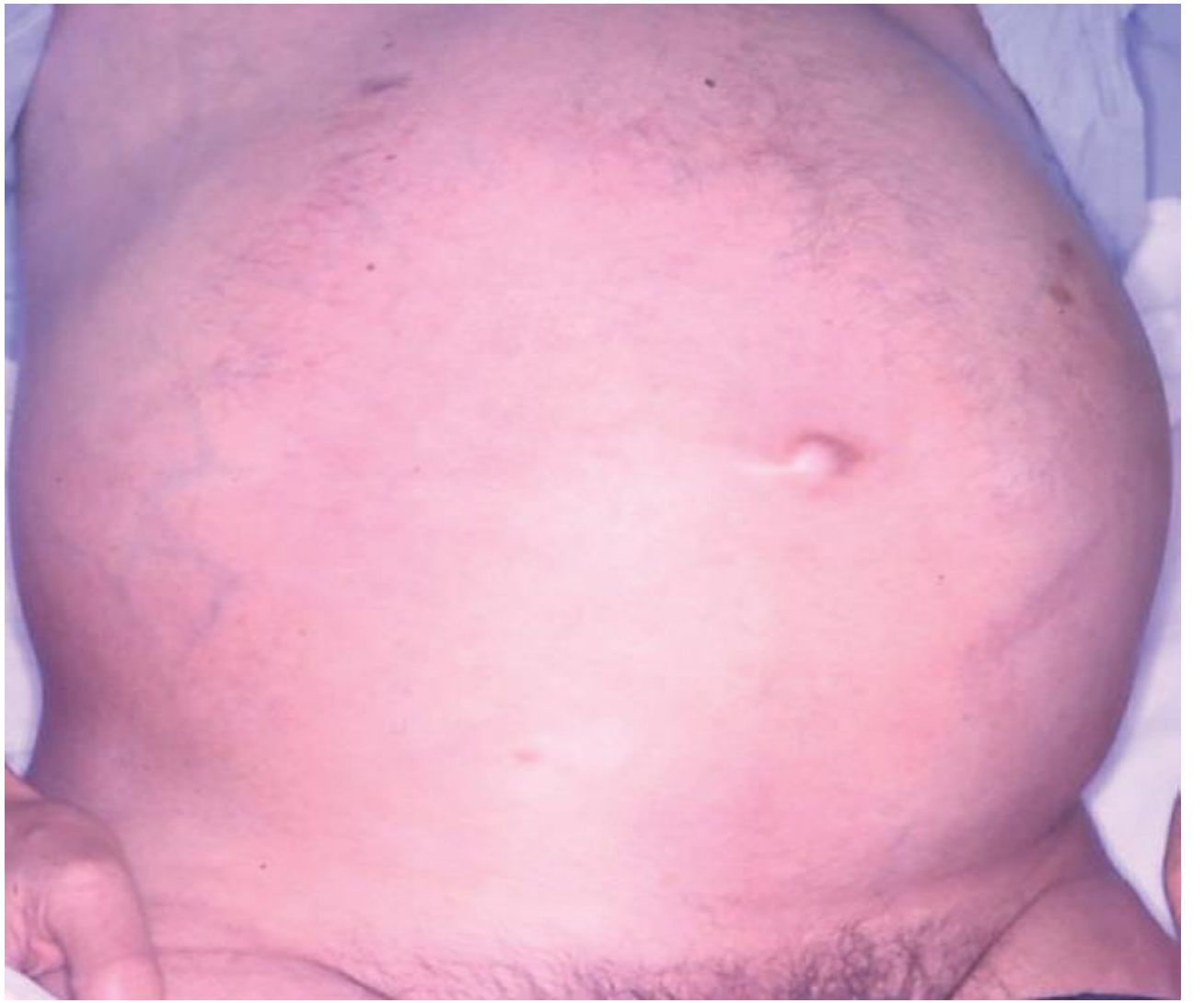


Gastrointestinal system

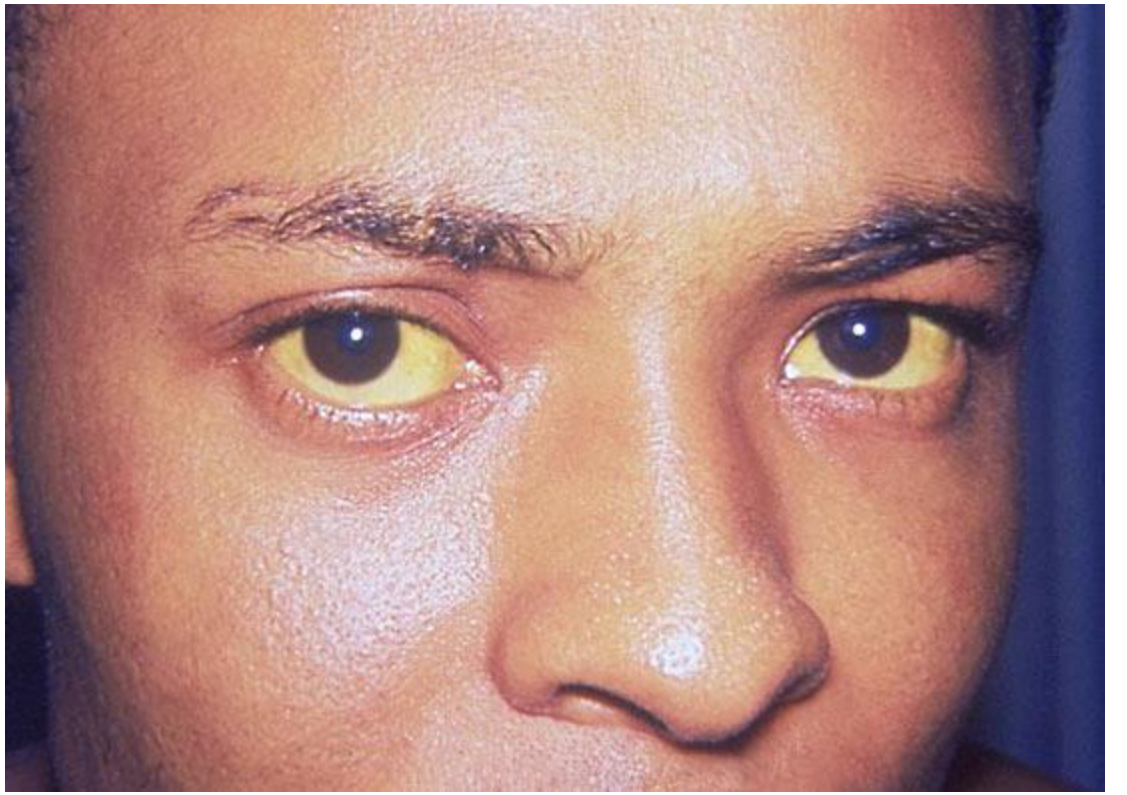
Aphthous ulcer



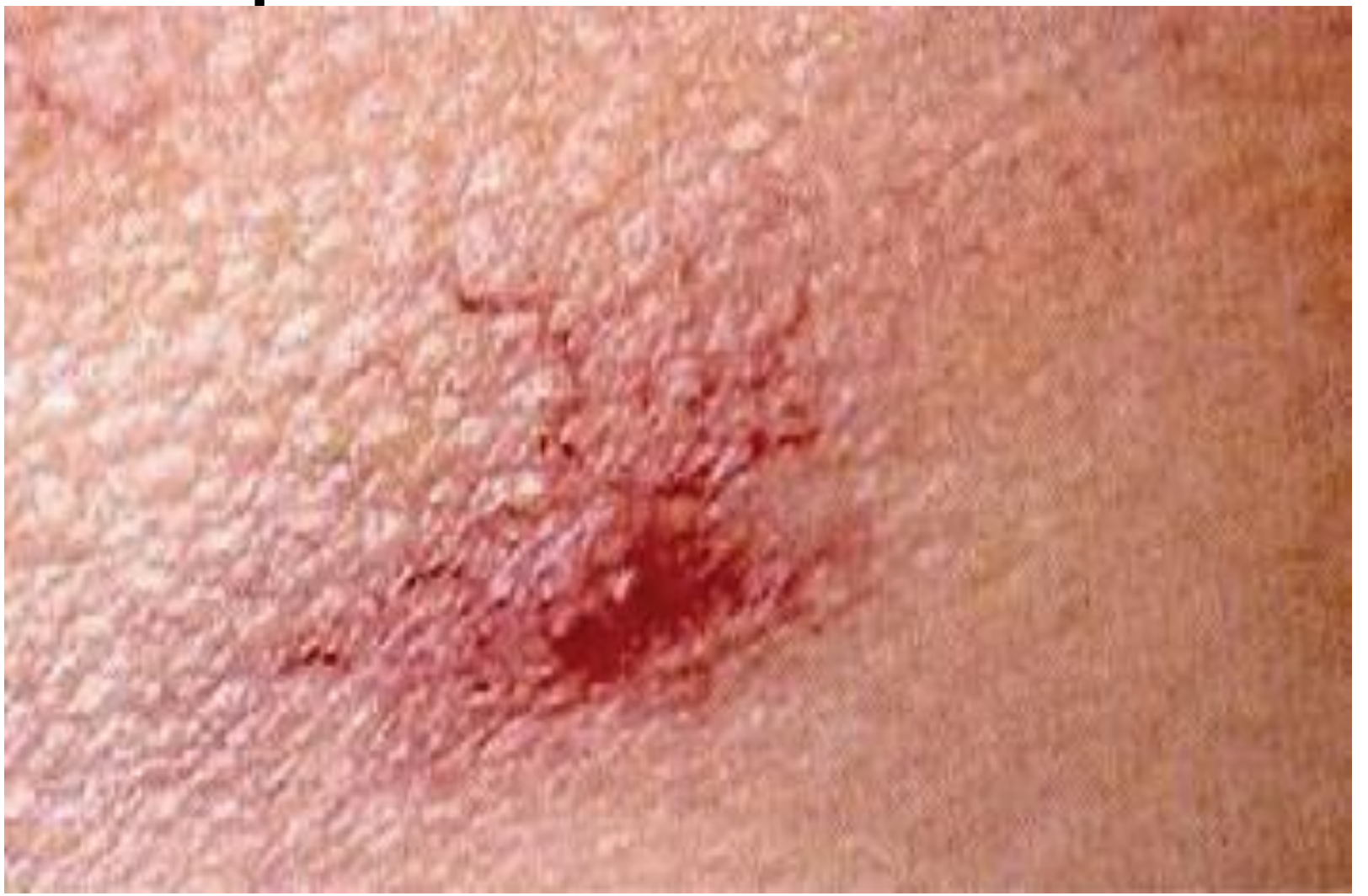
Abdominal distention due to ascites



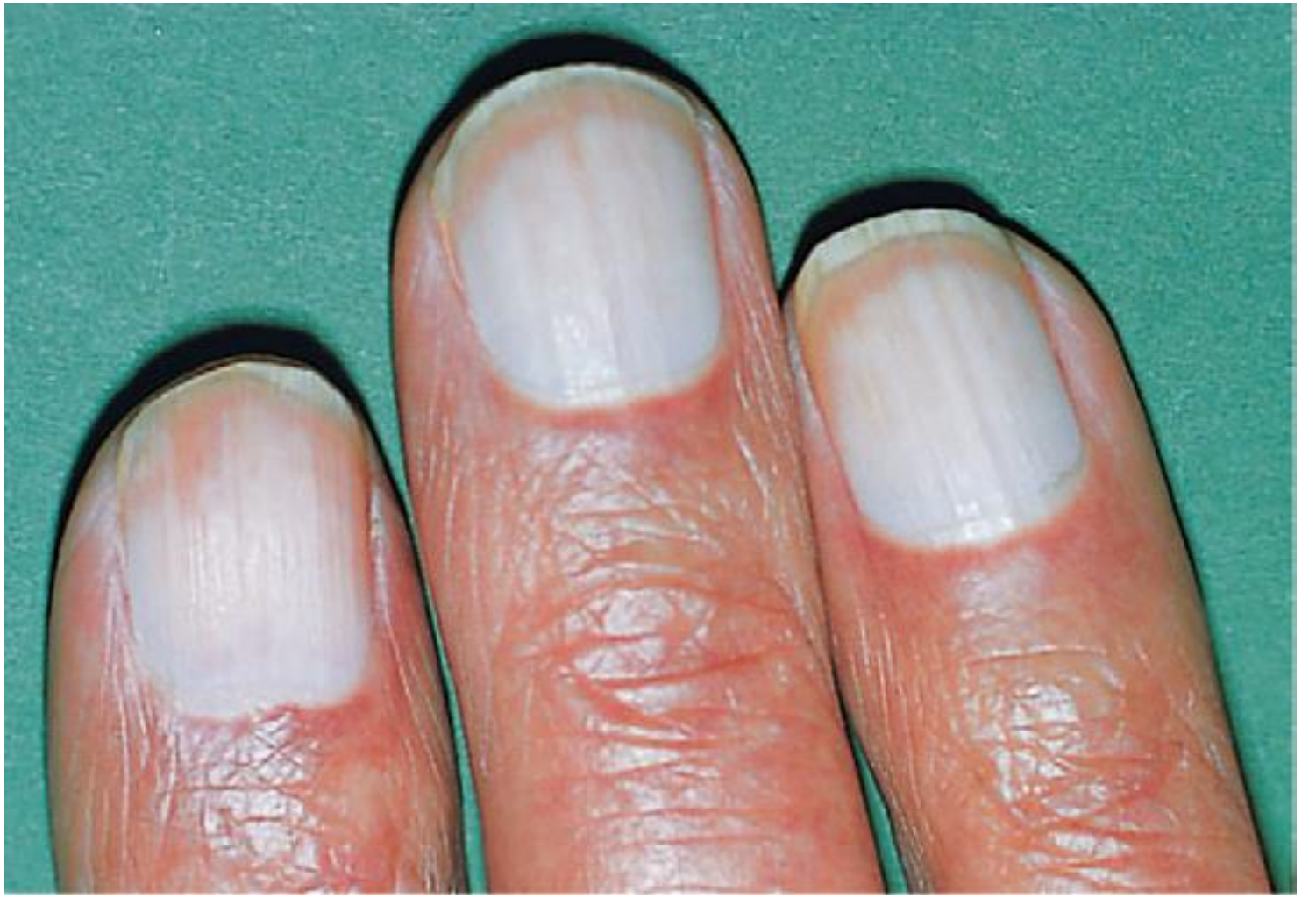
Yellow sclera of jaundice



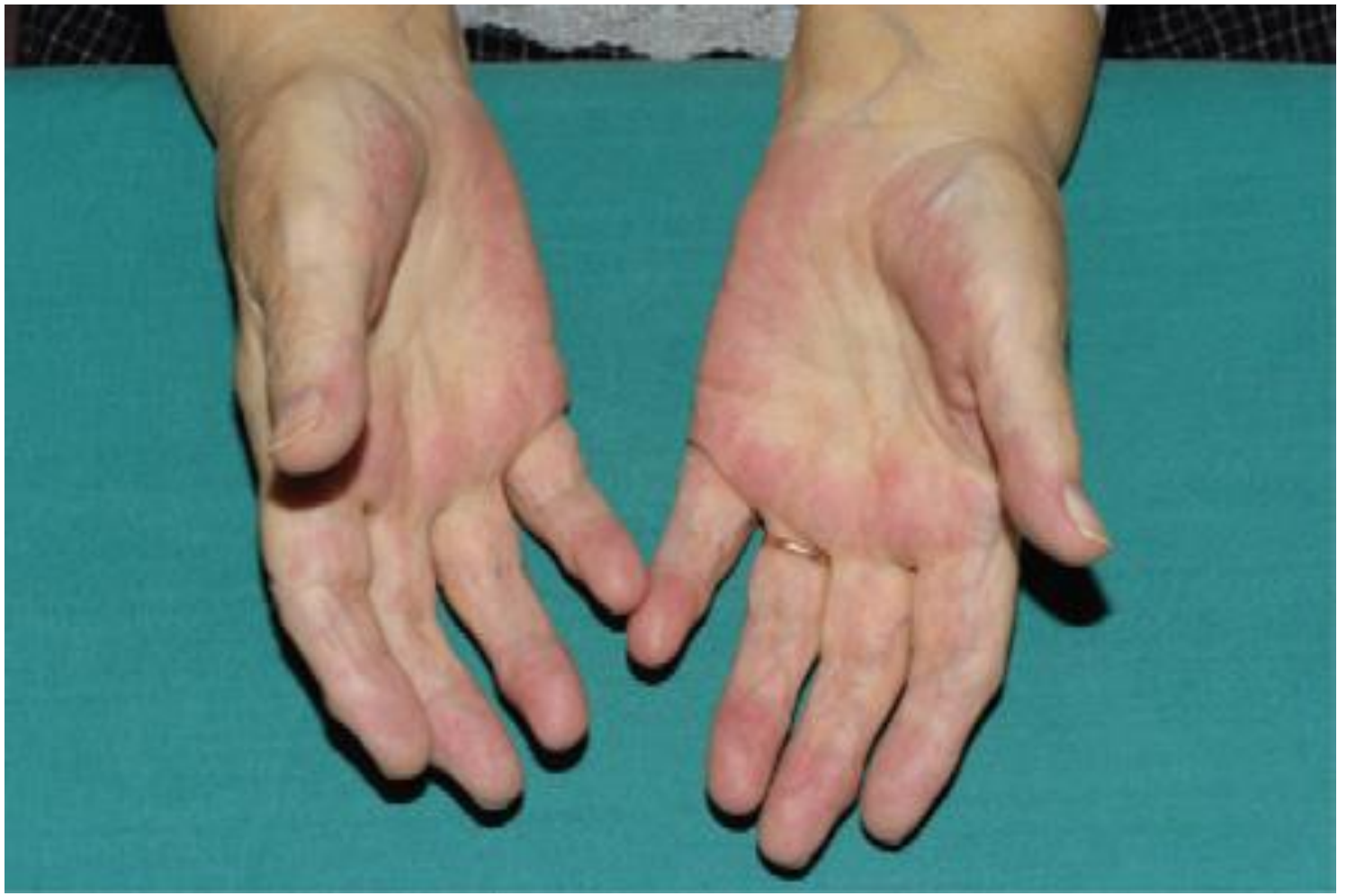
Spider naevi



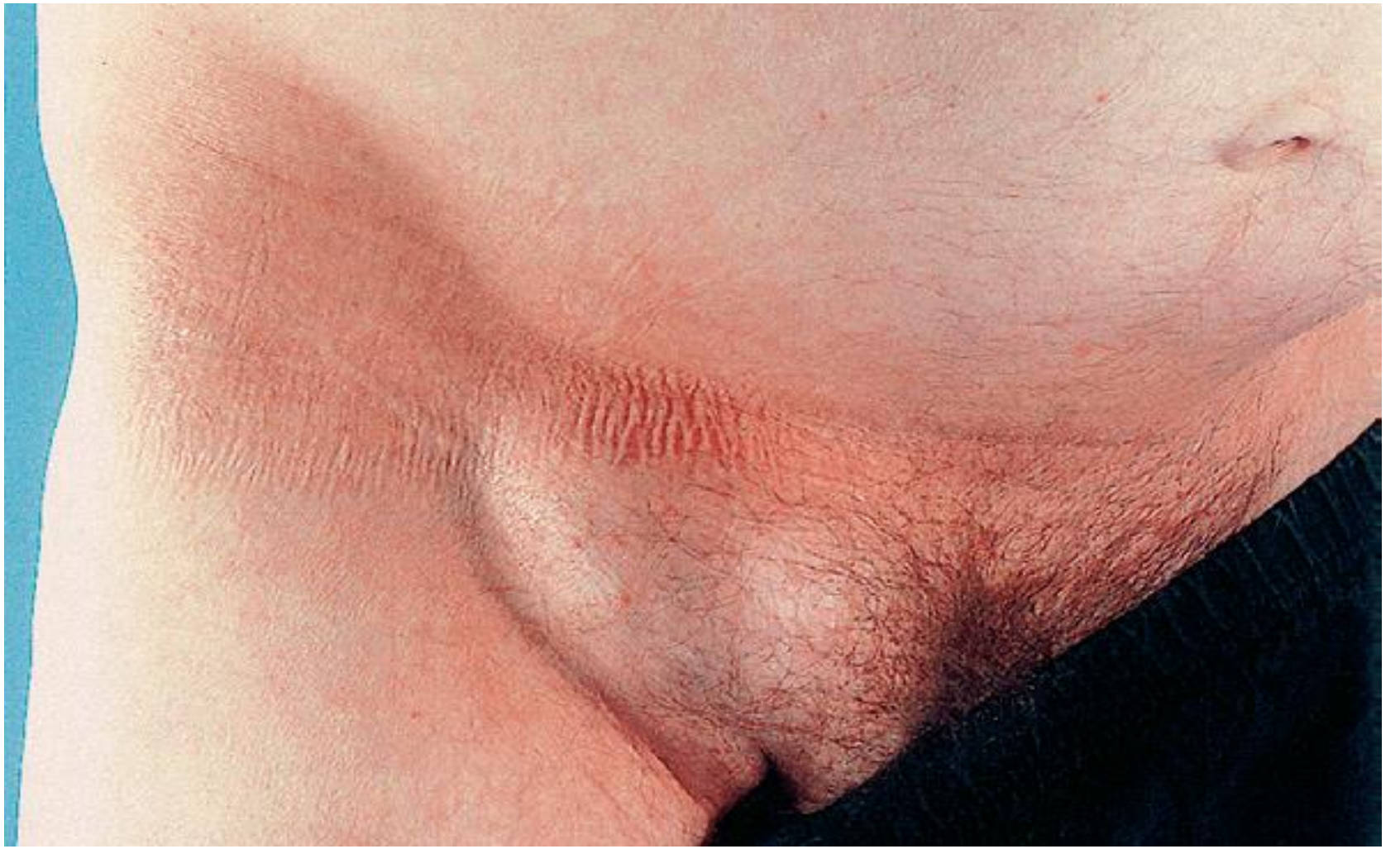
leukonychia



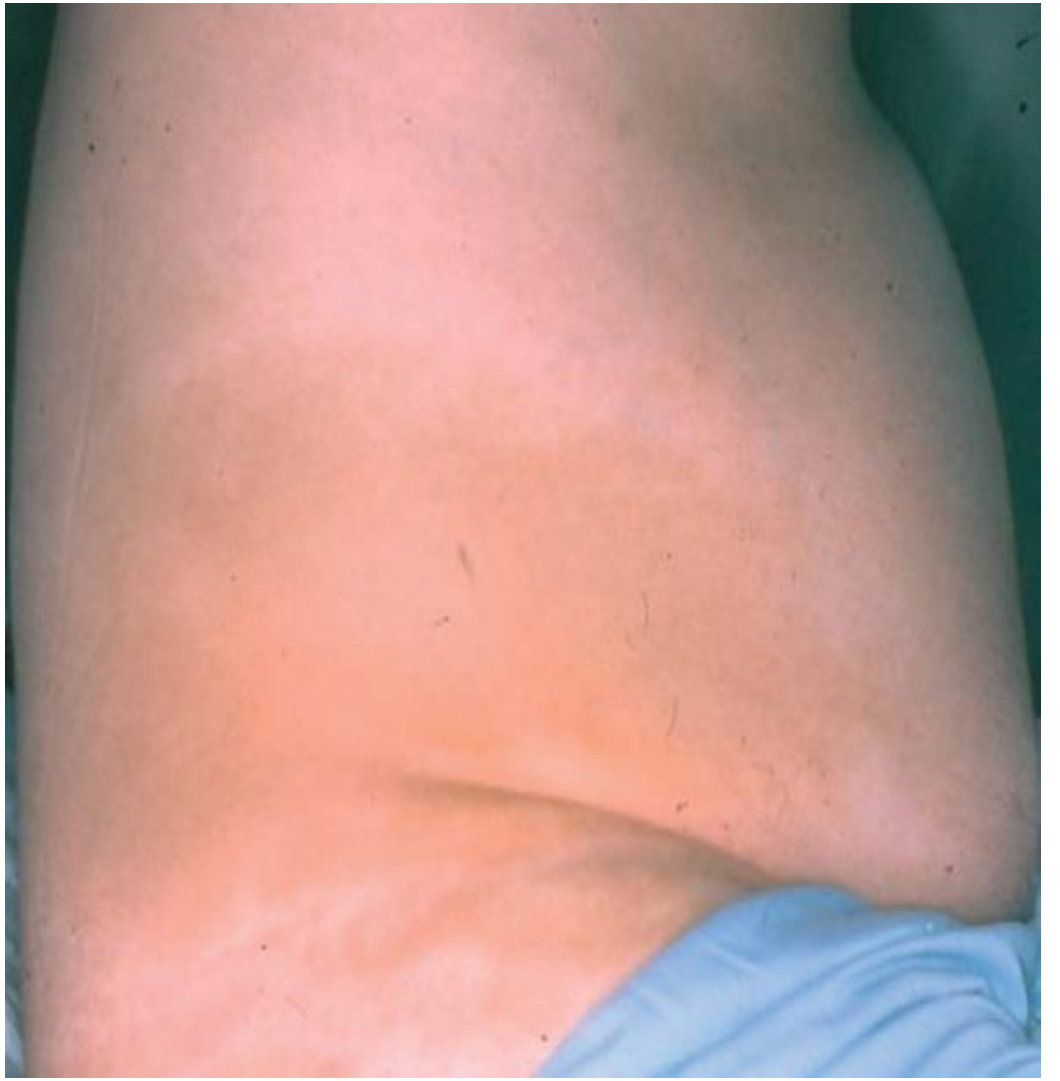
Palmar erythema



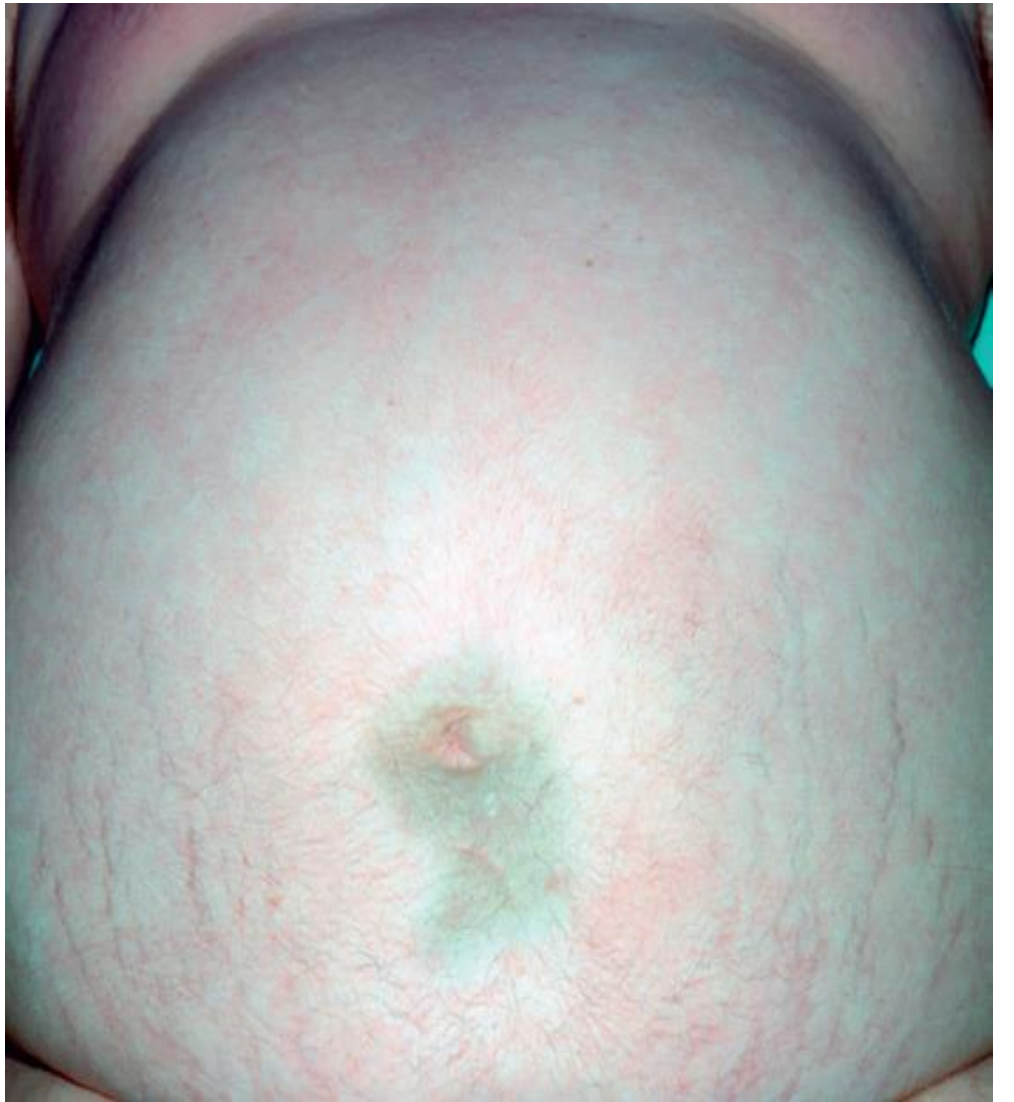
Right inguinal hernia



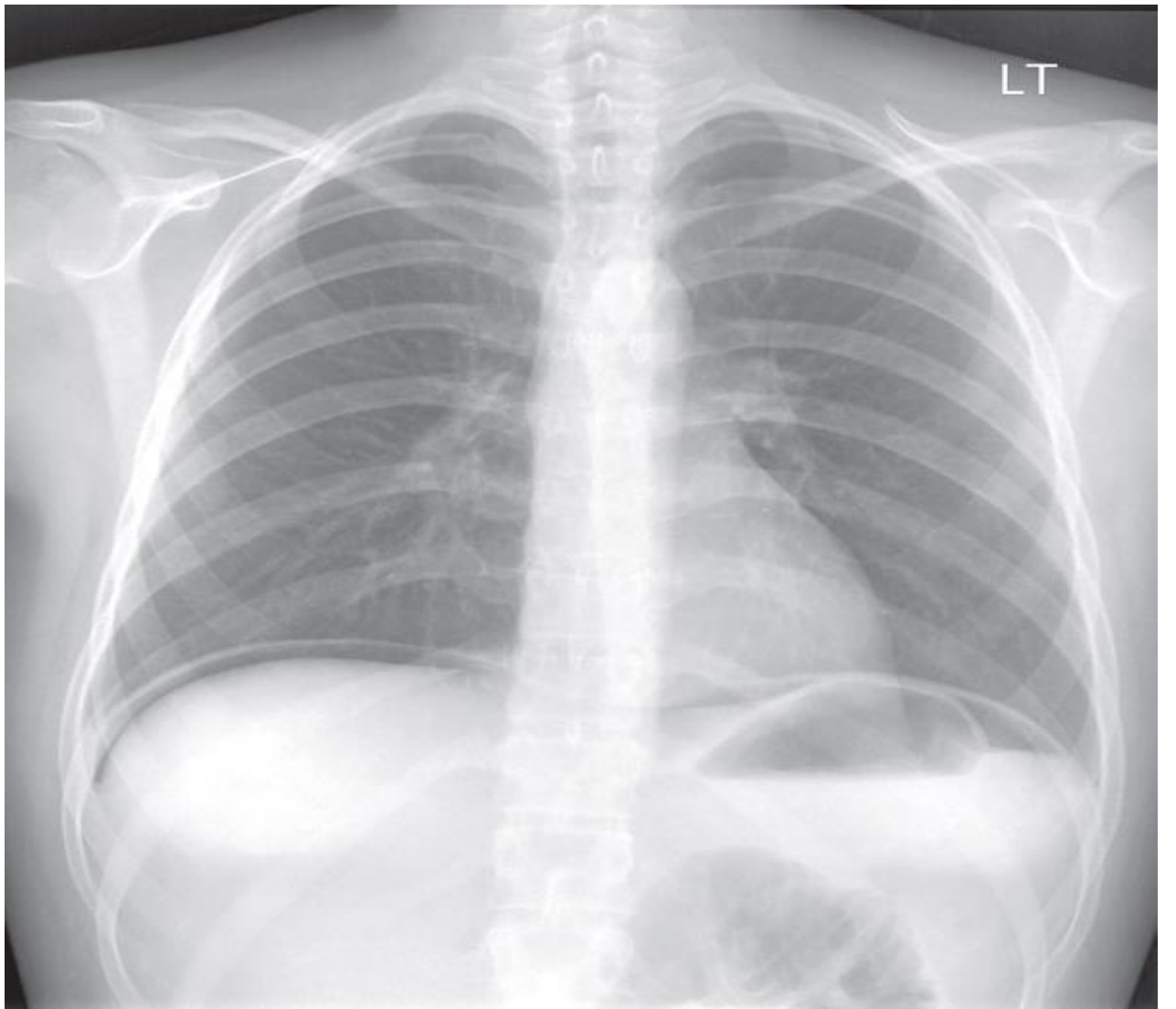
Grey-turner sign



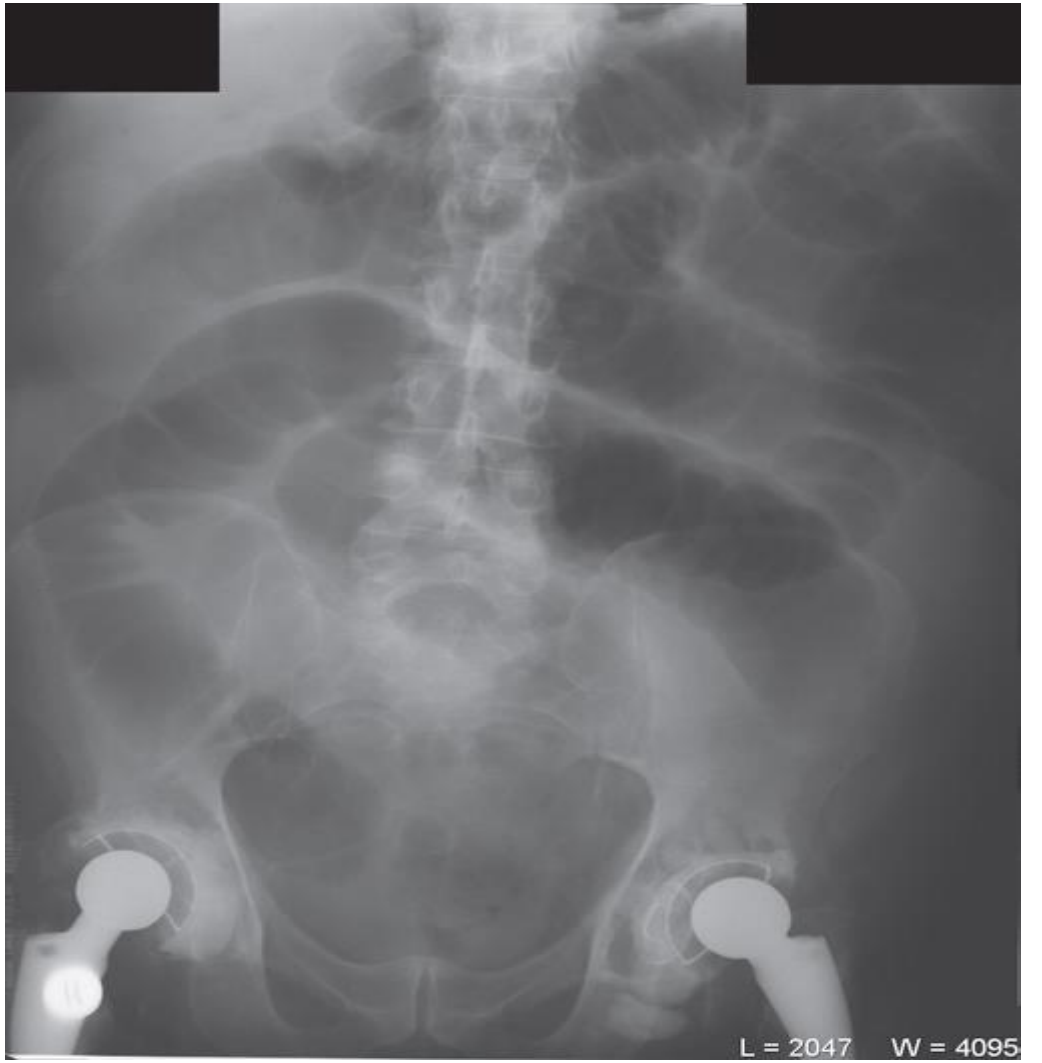
Cullen sign



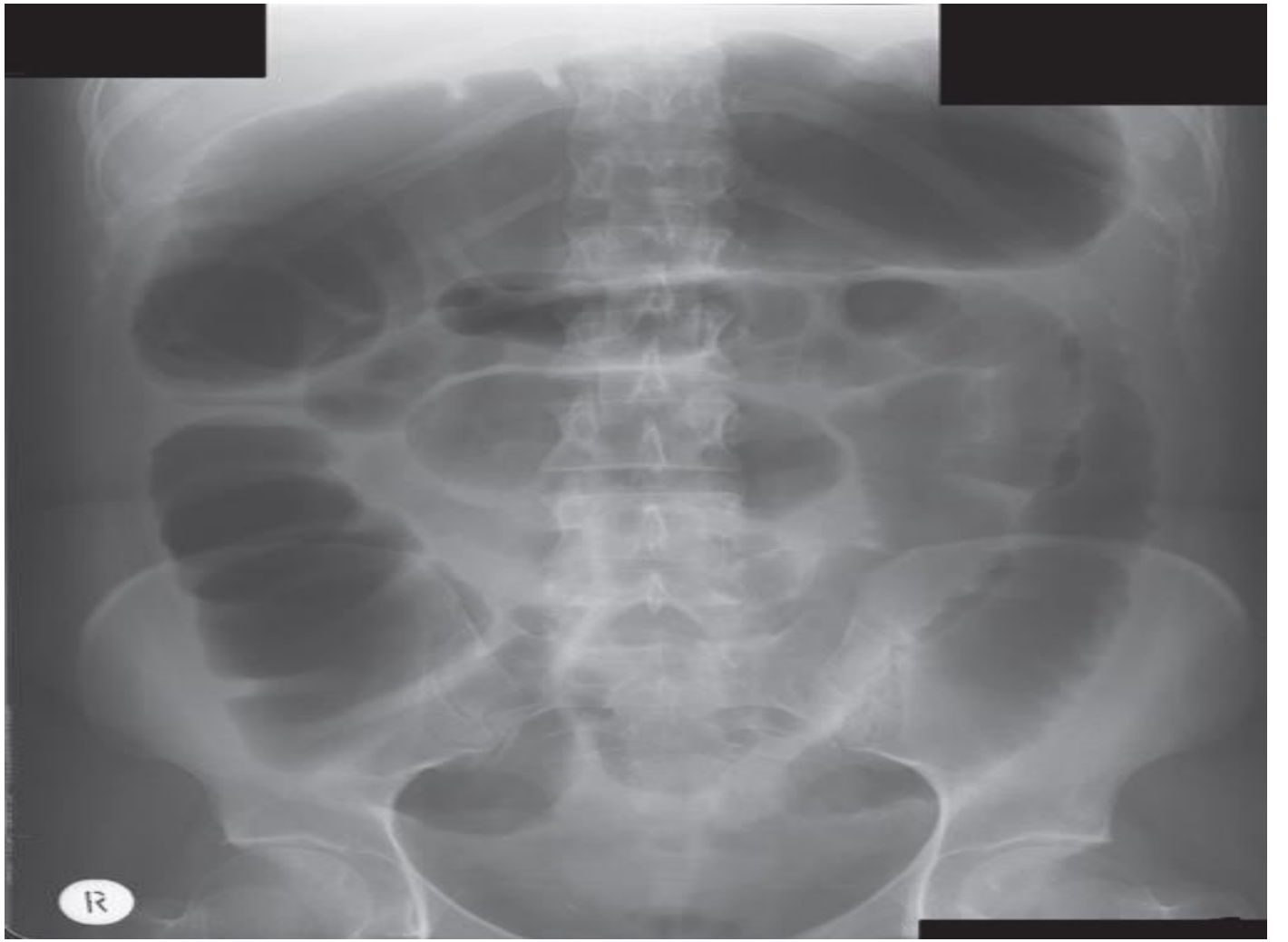
Air under diaphragm



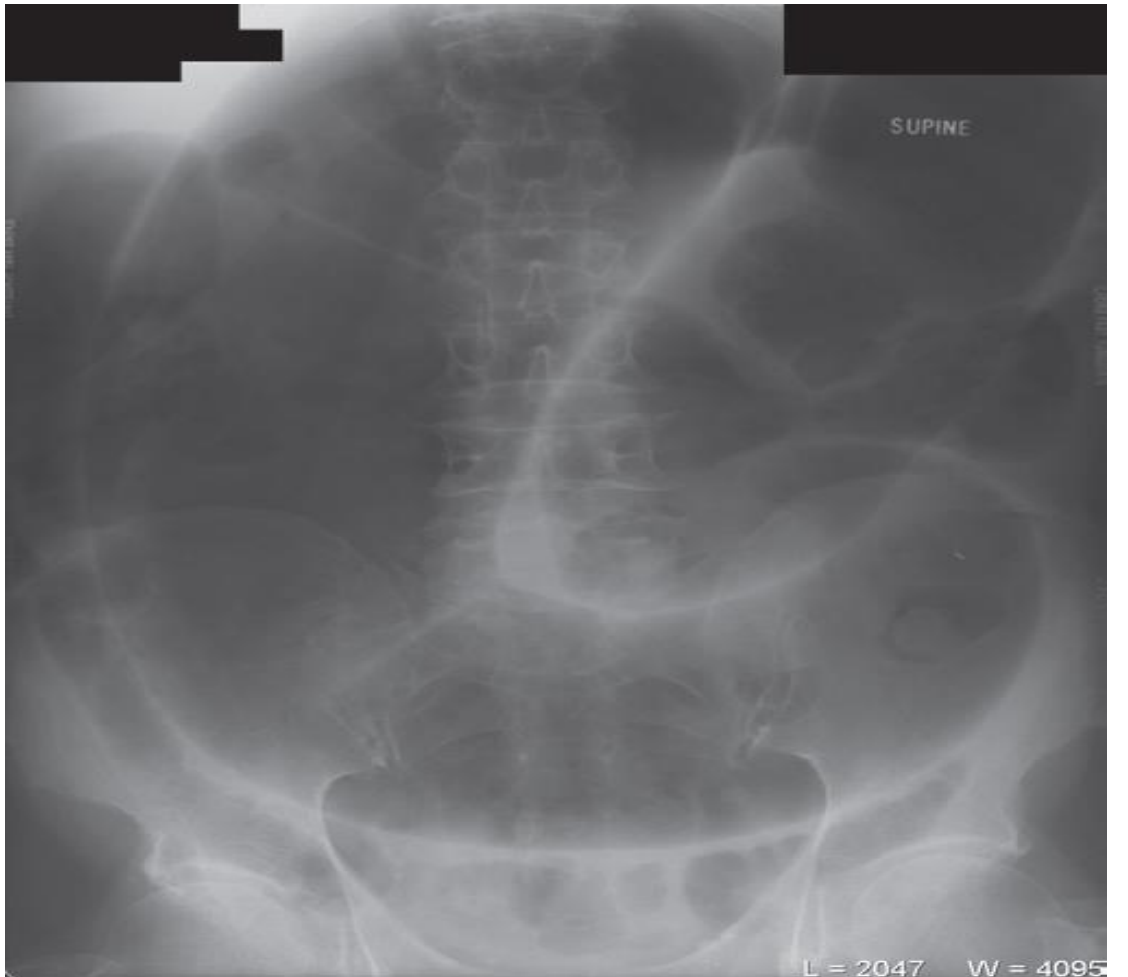
Dilated small bowel loops due to intestinal obstruction



Dilated large bowel loops due to toxic megacolon



Dilated large bowel loops due to sigmoid volvolus



Q2 : this female came with proximal Muscle weakness , She can't brush her hair , What is this lesion ?

- Gottron Papules

«وكُلُّ مَرءٍ سيَشقَىٰ دُونَما هَدَفٍ وكُلُّ عَيشٍ سَيَبلَىٰ دُونَما أمَلٍ وكُلُّ سَعيٍ سَيَجزِي اللهُ فَاعِلَهُ وكُلُّ حُلمٍ سَرَابٌ دُونَما عَمَلٍ!»

و آخر دعواهُم أن الحمدُ لله رب العالمين