Dermatology Detailed Dossier

2023 edition

إعداد محمود بركات

لا أحد، شكلك أول ما شفت حجم الدوسية

ملاحظات

- شامل لأسئلة سنوات حتى نهاية 2022
- شامل ملف دكتورة ختام (Dermatology notes Dr khetam)
- شامل مقدمة عن الجلدية (1ry & 2ry skin lesions and more)
- الملف مرتب حسب المواضيع تحت كل موضوع فيه ملاحظات الدكاترة وأسئلة السنوات
- أسئلة السنوات المكررة تم جمعها بسؤال واحد ووضع عدد مرات تكرار السؤال في هامش أعلى الصفحة من جهة اليمين أو على يسار السؤال
- أي كتابة بصندوق يعتبر هامش للملاحظات
- معاني الألوان: المهم، ملاحظات أو إضافات أو أسئلة من عندي، معلومات إضافية
- الكلام الي بلغتكم فيه بدوسيه الأشعة قائم برضو على هذا الملف وأي الملفات ثانية اشتغلتها ويا ريت بس هبل

Last update: 02/Sep/2022

Basics of dermatology

Normal skin, Dermatopathology, Skin lesions, Clinical tests

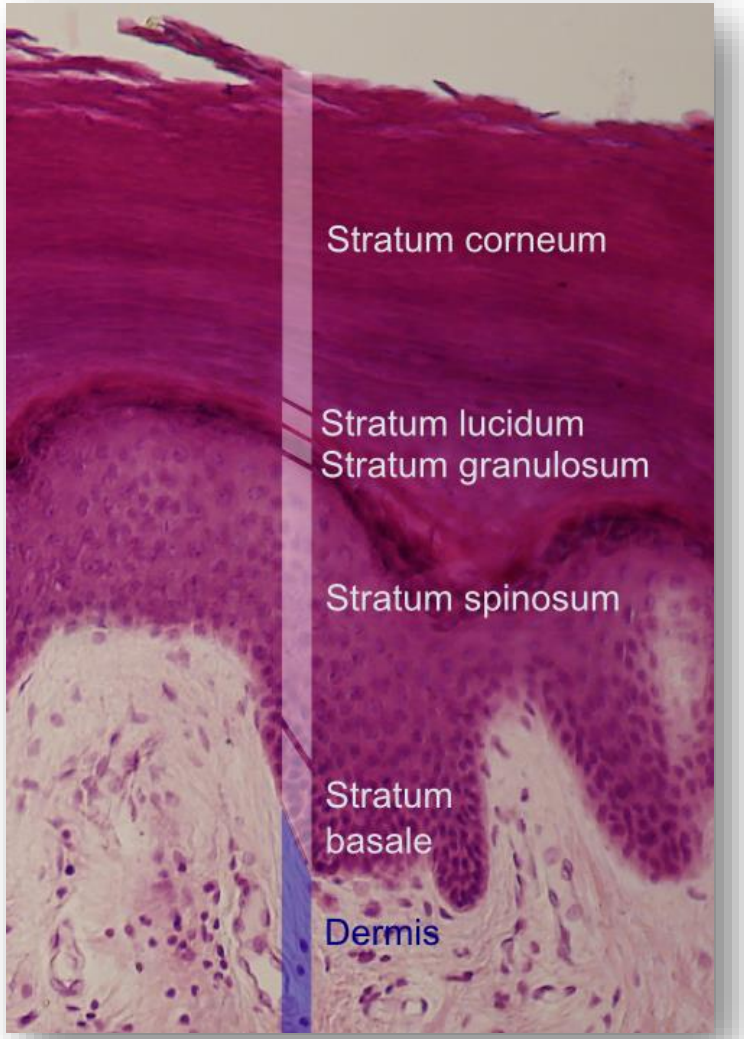
Normal skin

Three layers:

- **Epidermis**: keratinocytes (squamous epithelial cells)
  - Stratum **C**orneum
  - Stratum **L**ucidum
  - Stratum **G**ranulosum
  - Stratum **S**pinosum
  - Stratum **B**asalis
  - Mnemonic: **C**ome, **L**ets **G**et **S**un **B**urn
- **Dermis**: connective tissue, vessels
- **Subcutaneous fat** (also called hypodermis or subcutis)

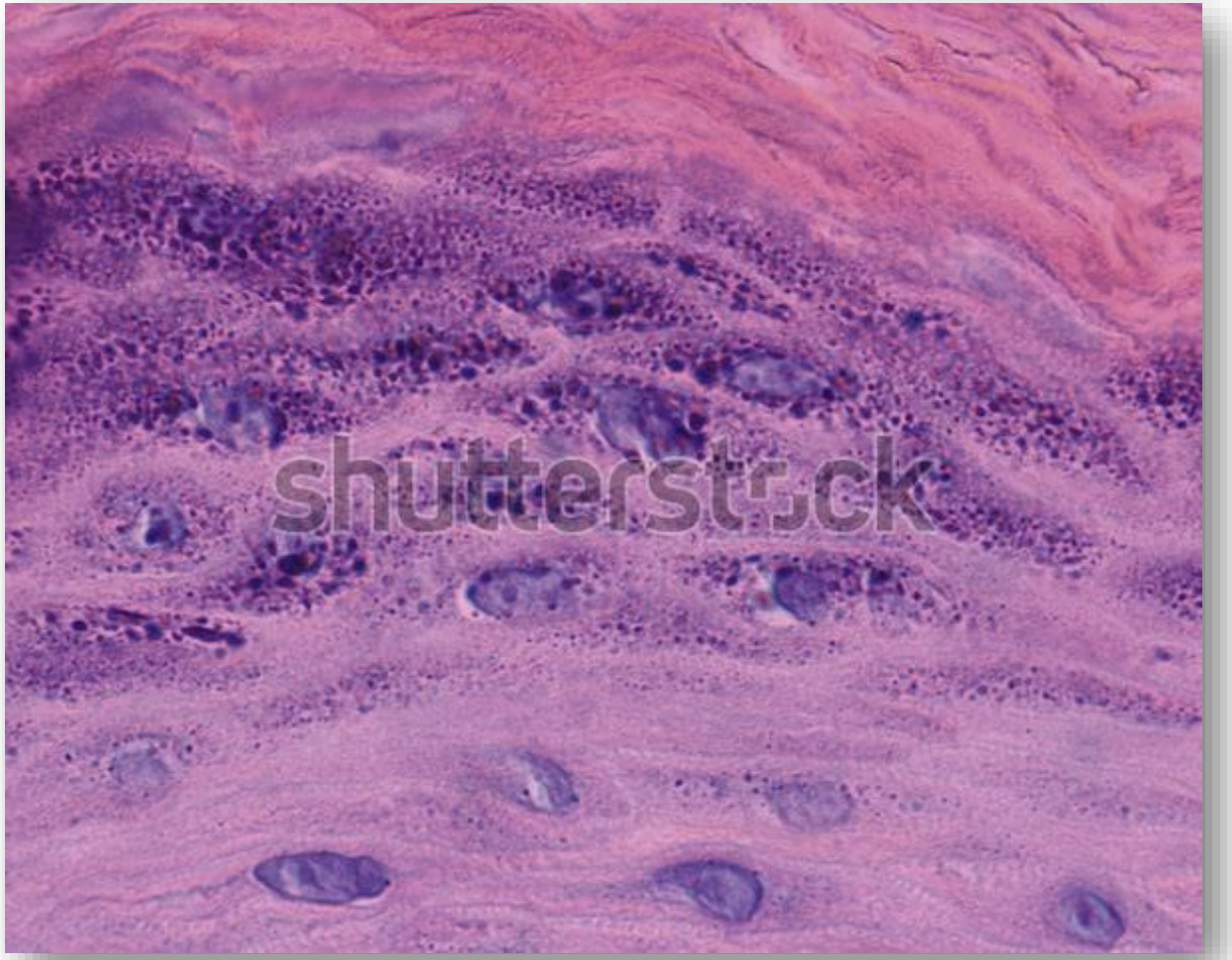
Epidermal Layers

- **Stratum Corneum**
  - Anucleated cells
  - Filled with keratin filaments
- **Stratum Lucidum**
  - Clear layer of dead skin cells
- **Stratum Granulosum**
  - Keratohyalin granules
  - Form keratin filaments
- **Stratum Spinosum**
  - Desmosomes form spines
- **Stratum Basalis**
  - Stem cells



Which skin layer is this ?

Stratum granulosum



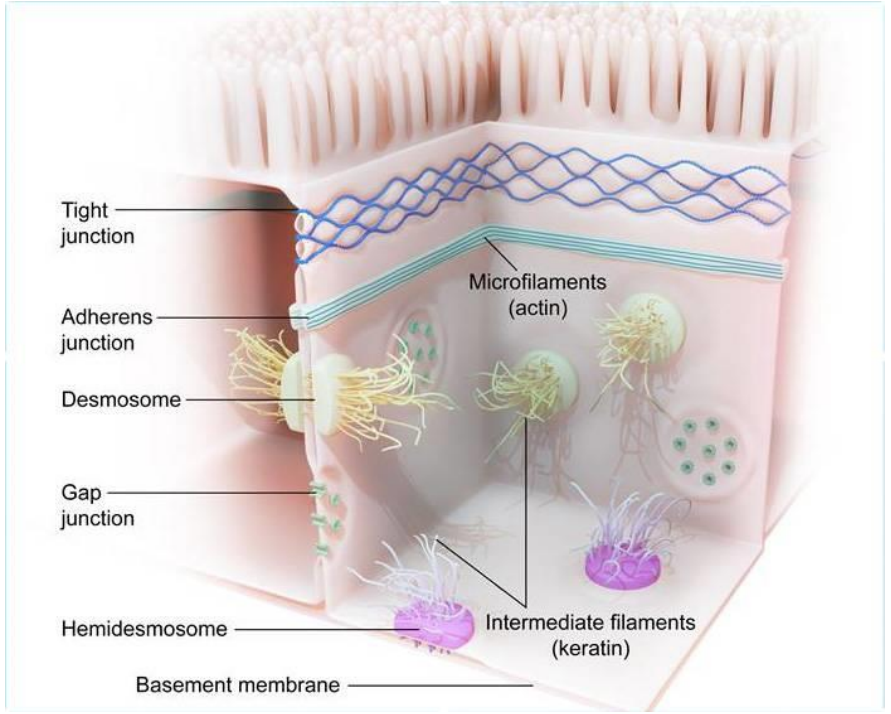
Epidermal Layers

سنوات (1)
- Mention 3 cells present in the epidermis:
  - Melanocyte, Keratinocyte and Langerhans cells

سنوات (1)
- Antigen presenting cell in skin
  - Langerhans cells

سنوات (1)
- Keratinocyte are connecting to each other by
  - Desmosomes

إضافي
- keratinocyte are connected to the basement membrane by
  - Hemidesmosomes

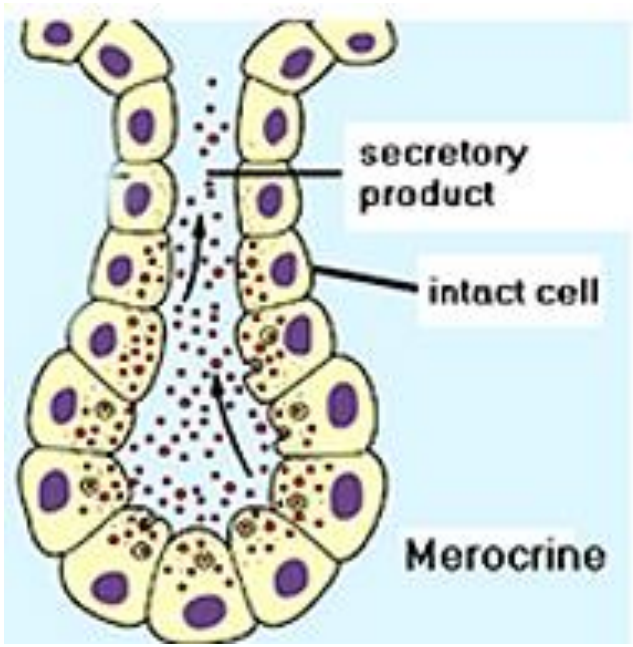


الصورة من عندي للتذكير بأنواع الcell junctions

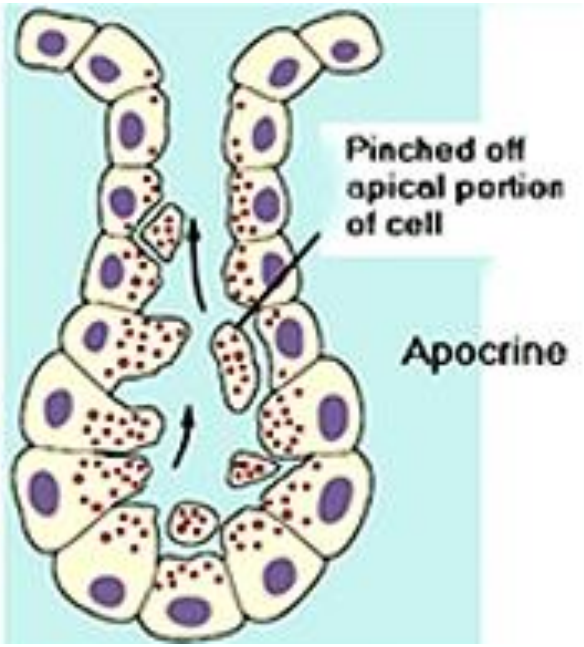
Friendly reminder

Types of glands

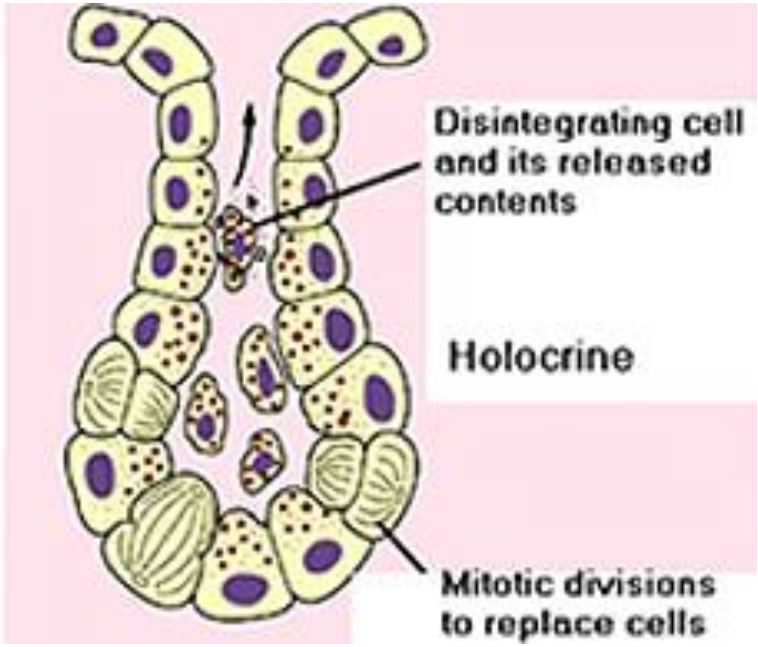
ما جاء عليهم من قبل ولكن ليطمئن قلبي



Salivary glands, eccrine sweat glands, and apocrine sweat glands



Mammary glands



Sebaceous glands and meibomian glands

مش محطوطة بالغلط انتبه !

Dermatopathology

شرح

Dermatopathology

- Terms used to describe microscopic findings
- Used in analysis of skin biopsies

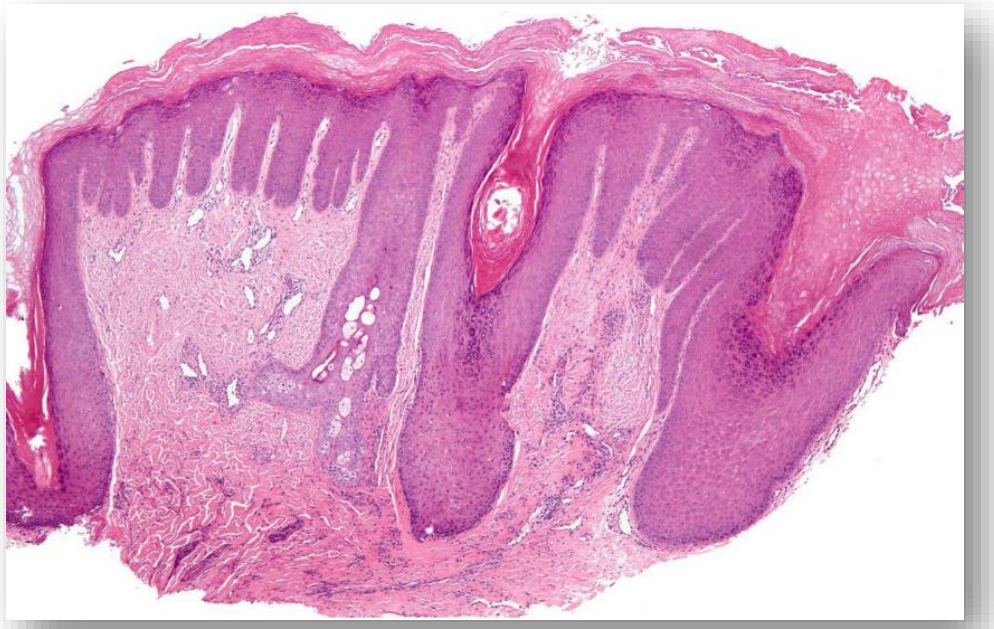
- **Hyperkeratosis**: Thickening of stratum corneum
- **Parakeratosis**: Hyperkeratosis + retained nuclei in stratum corneum
- **Hypergranulosis**: Increased thickness of stratum granulosum
- **Spongiosis**: Fluid accumulation (edema) of epidermis
- **Acantholysis**: Loss of connections between keratinocyte
- **Acanthosis**: Diffuse epidermal hyperplasia, elongated rete ridges, spinous layer thickening

سنوات (1)

Dermatopathology

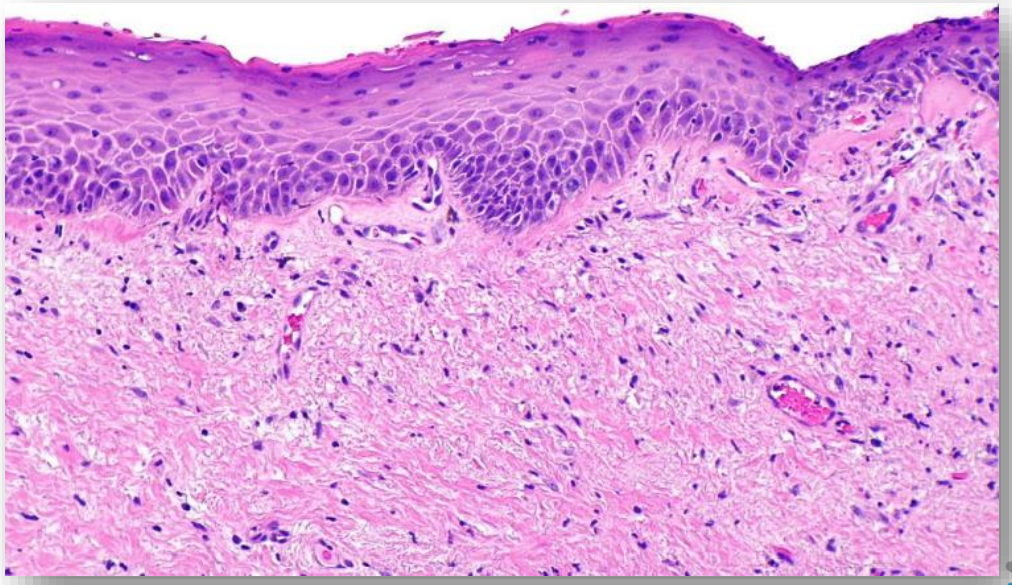
Hyperkeratosis

- Thickening of stratum corneum
- Excess quantity of keratin
- Seen in **Psoriasis** and **Callus**



Parakeratosis

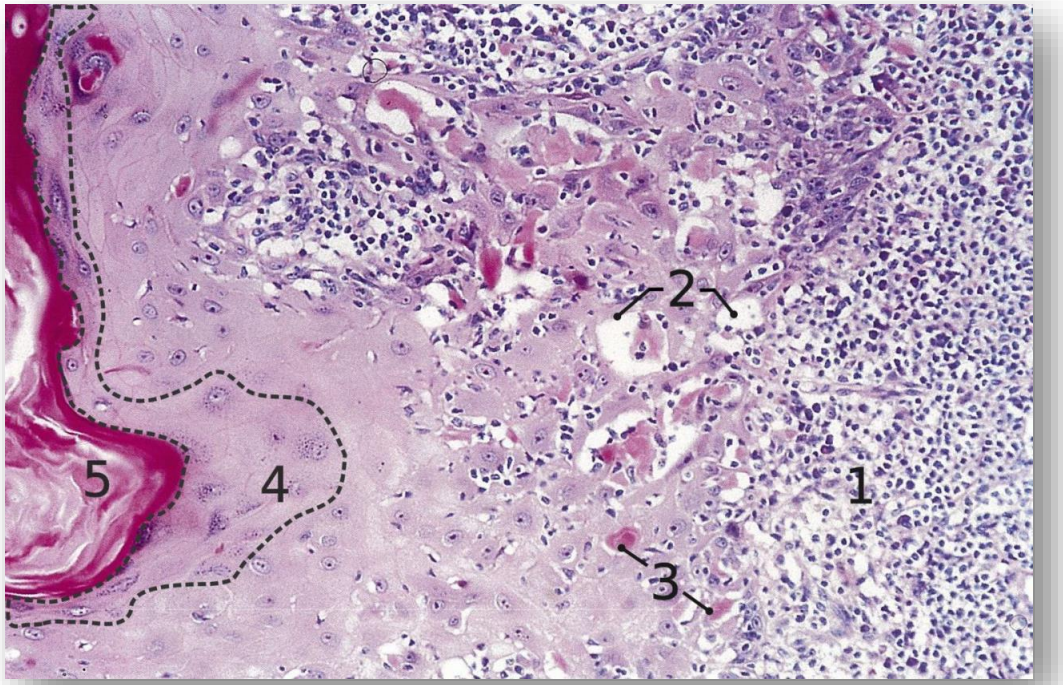
- Hyperkeratosis + retained nuclei in stratum corneum
- Indicates hyperproliferation
- Seen in **psoriasis** and **malignancies**



Dermatopathology

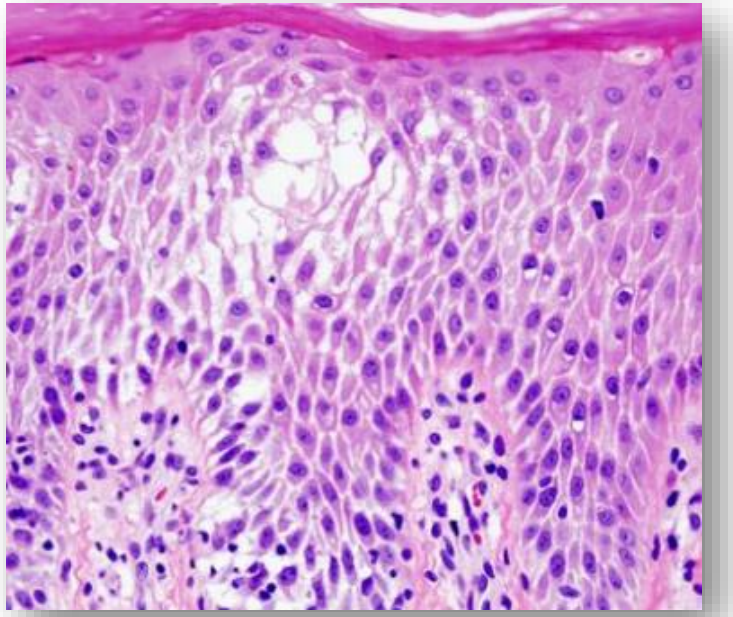
Hypergranulosis

- Increased thickness of stratum granulosum (4 in the picture)
- Classic finding in **lichen planus**



Spongiosis

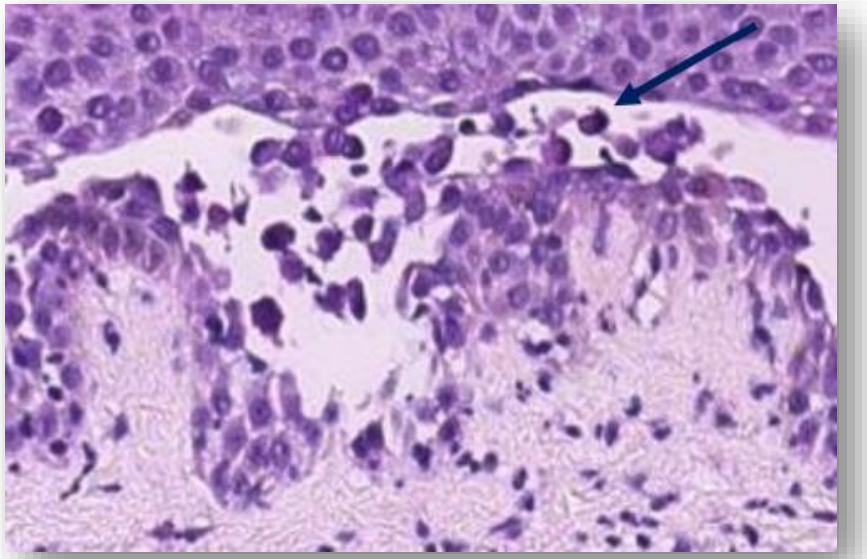
- Fluid accumulation (edema) of epidermis
- Seen in **eczema**, many other skin disorders



Dermatopathology

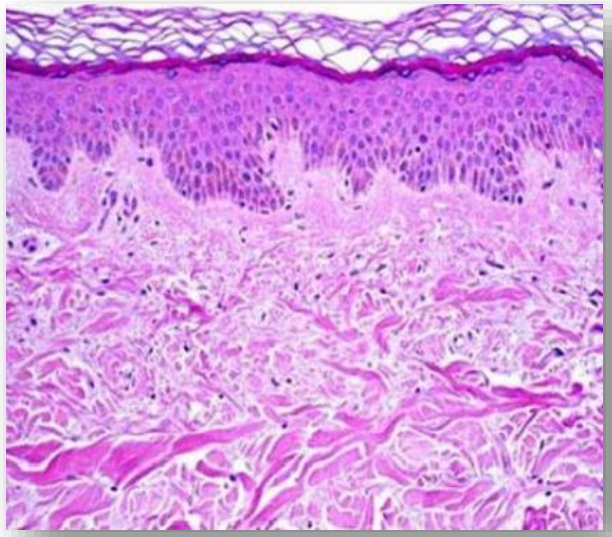
Acantholysis

- Loss of connections between keratinocyte
- Often loss of desmosomes
- Detached, floating freely in epidermis
- Key feature of **pemphigus vulgaris**

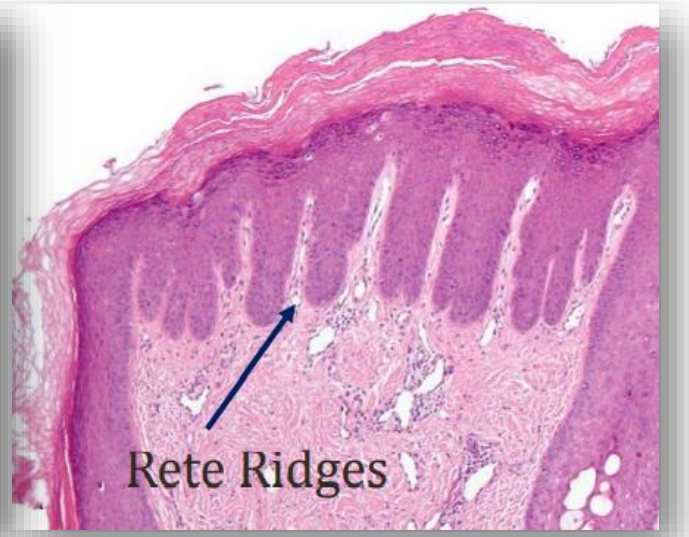


Acanthosis

- Diffuse epidermal hyperplasia
- Elongated rete ridges
- Spinous layer thickening



Normal



Acanthosis

Skin lesions

Skin lesions

- **Primary lesions**
  - Directly caused by disease process
  - Described using standard terminology
  - Macules, papules, vesicles, bulla
- **Secondary lesions**
  - Modification of primary lesion
  - Or caused by trauma, external factors
  - Scale, crust, erosion, fissure, ulcer
- **Complex skin lesions**
  - Hemorrhage, rashes, lichenification, eczema

معلومات إضافية

مشان تعرف توصف

Configuration

Configuration: refers to how lesions are locally grouped (organized)

1. **Agminate**: in clusters
2. **Annular** or **circinate**: ring-shaped
3. **Arciform** or **arcuate**: arc-shaped
4. **Digitate**: with finger-like projections
5. **Discoid** or **nummular**: round or disc-shaped
6. **Figurate**: with a particular shape
7. **Guttate**: resembling drops
8. **Gyrate**: coiled or spiral-shaped
9. **Herpetiform**: resembling herpes
10. **Linear**
11. **Mamillated**: with rounded, breast-like projections
12. **Umbilicated**: have a small depression
13. **Reticular** or **reticulated**: resembling a net
14. **Serpiginous**: with a wavy border
15. **Stellate**: star-shaped
16. **Targetoid**: resembling a bullseye
17. **Verrucous** or **Verruciform**: wart-like

معلومات إضافية

مشان تعرف توصف

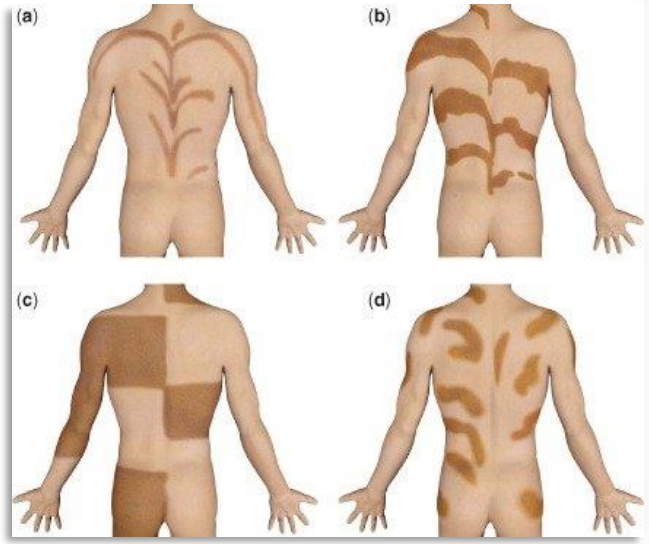
Distribution

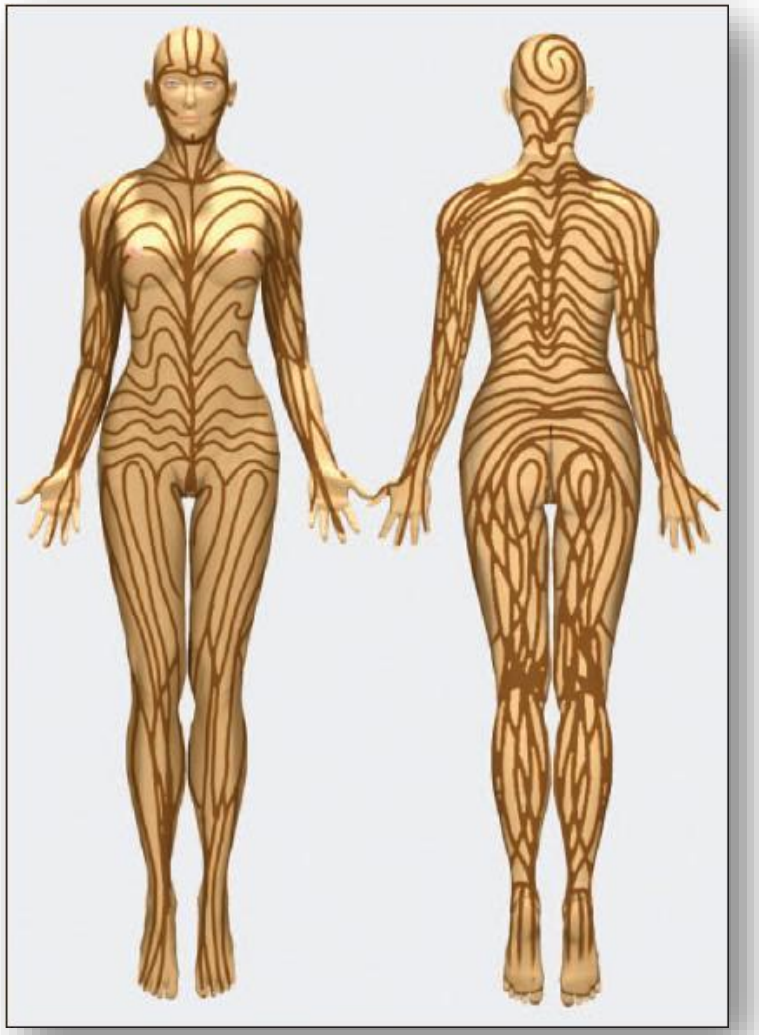
Distribution: refers to how lesions are localized. They may be confined to a single area (a patch) or may exist in several places.

1. **Generalized**
2. **Symmetric**: one side mirrors the other
3. **Flexural**: on the front of the fingers
4. **Extensor**: on the back of the fingers
5. **Intertriginous**: in an area where two skin areas may touch or rub together
6. **Morbilliform**: resembling measles
7. **Palmoplantar**: on the palm of the hand or bottom of the foot
8. **Periorificial**: around an orifice such as the mouth
9. **Periungual/subungual**: around or under a fingernail or toenail
10. **Blaschkoid**: following the path of Blaschko's lines in the skin
11. **Photodistributed**: in places where sunlight reaches
12. **Zosteriform** or **dermatomal**: associated with a particular nerve

Blaschko's lines

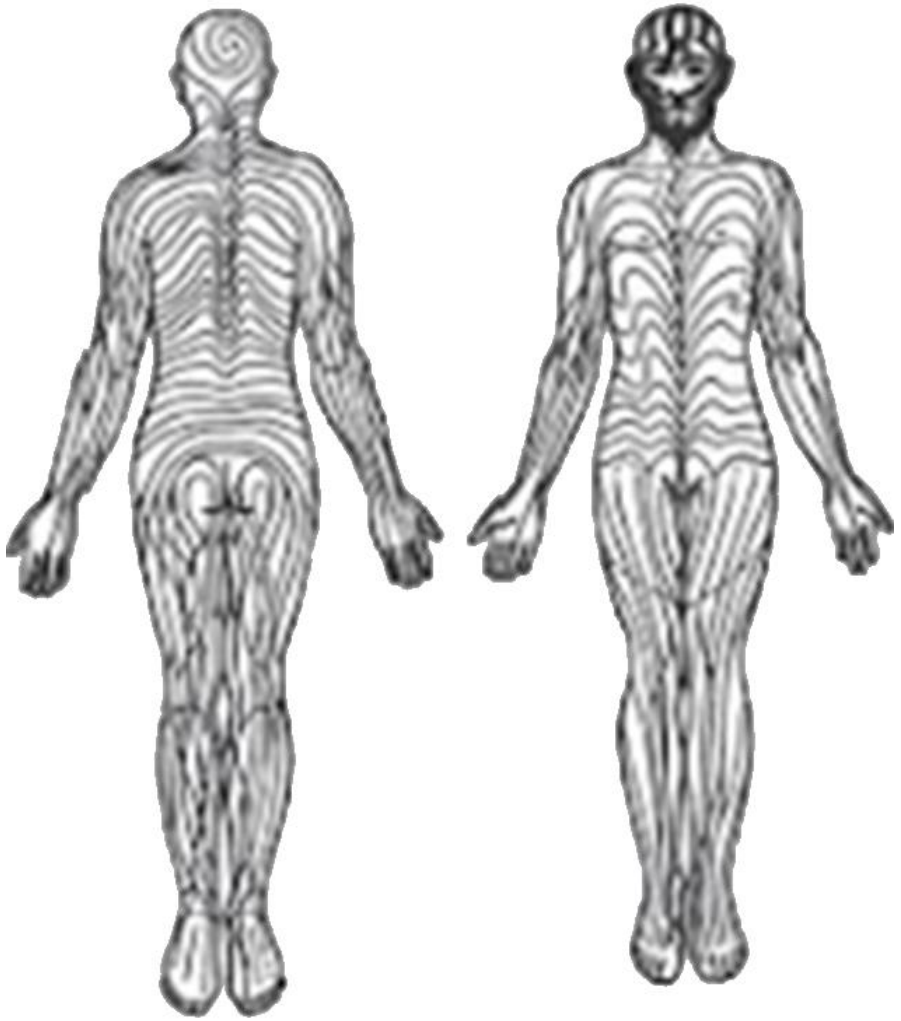
- Lines of normal cell development in the skin.
- These lines are invisible under normal conditions but can become apparent over the skin due to a mosaic skin condition.
- Many nevoid skin conditions follow Blaschko's lines, such as:
  - Melanocytic nevi
  - Achromic naevus
  - Vitiligo
  - CHILD syndrome
  - Lichen planus





One of the following dermatosis may come on these lines

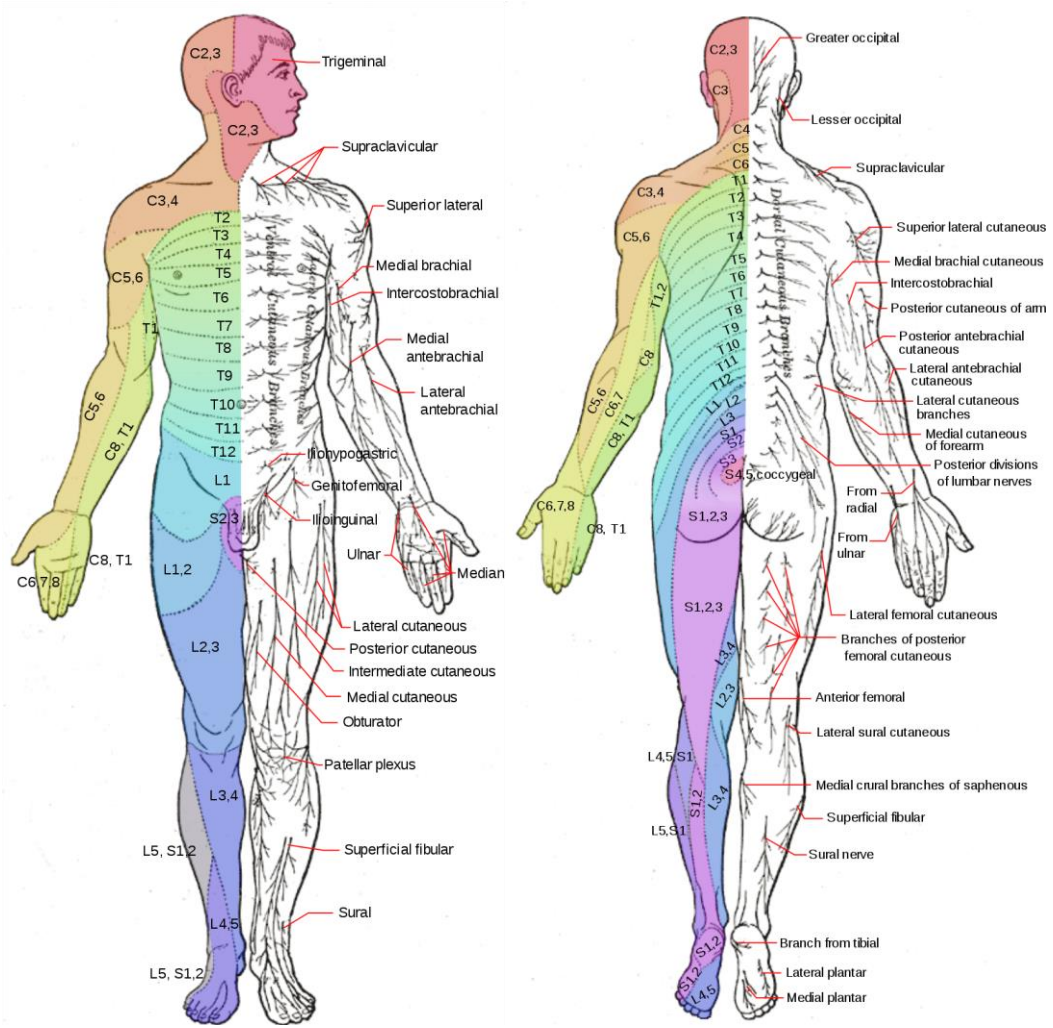
A. Vitiligo
B. Alopecia areata
C. Androgenic alopecia
D. Urticaria
E. Erythema multiforme



معلومات إضافية

Dermatome

- A dermatome is an area of skin that is mainly supplied by afferent nerve fibers from the dorsal root of any given spinal nerve.
- Some diseases can show dermatomal distribution (a zosteriform pattern) such as:
  - Varicella zoster virus (VZV)
  - Lichen planus
  - Impetigo contagiosa



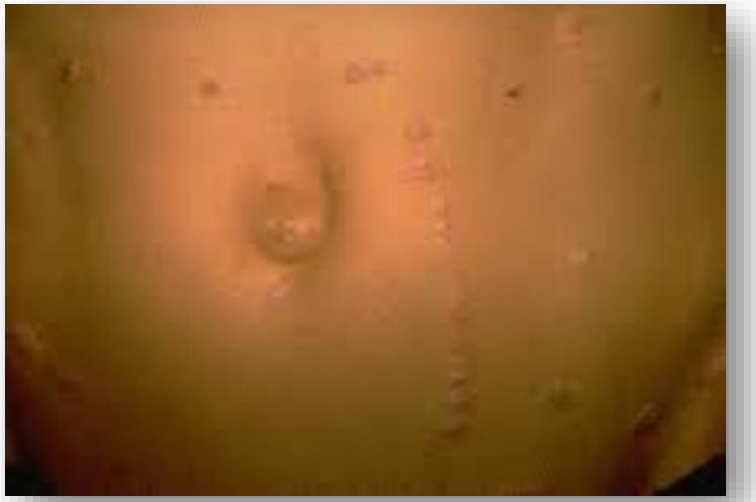
Koebner Phenomenon

- Also called isomorphic response
- Describes the appearance of new skin lesions of a pre-existing dermatosis on areas of cutaneous injury in otherwise healthy skin.
- **Causes:**
  - **Infective** & **chemical** causes; result in linear lesions after a linear exposure to a causative
    1. **molluscum contagiosum**
    2. **Warts**
    3. **Kaposi sarcoma**
    4. **Cutaneous leishmaniasis**
    5. **poison ivy**
  - Causes of the Koebner phenomenon that are secondary to scratching rather than infection or chemical
    1. **Vitiligo**
    2. **Psoriasis**
    3. **lichen planus**
    4. **Eczema**
    5. **Pityriasis rubra pilaris**

Koebner Phenomenon

دكتورة ختام

- Lesion associated with Koebner Phenomenon
  1. Psoriasis
  2. Lichen planus
  3. Vitiligo
  4. Still’s disease
  5. Small vessel vasculitis

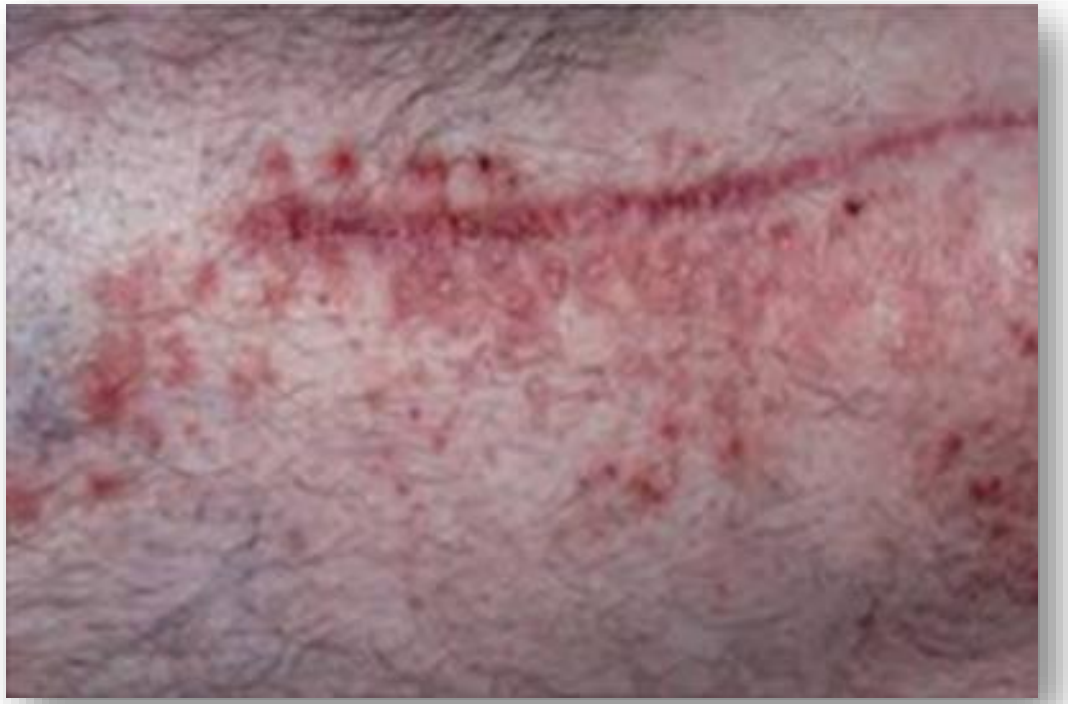


دكتور عوض

- Lesion associated with Koebner Phenomenon
  1. Psoriasis
  2. Vitiligo
  3. Lichen planus
  4. Eczema
  5. Erythema multiforme

What is the diagnosis

A. Koebner phenomena at the site of scar
B. Secondary infection
C. Impetigo
D. Fungal infection
E. Normal healing of the wound



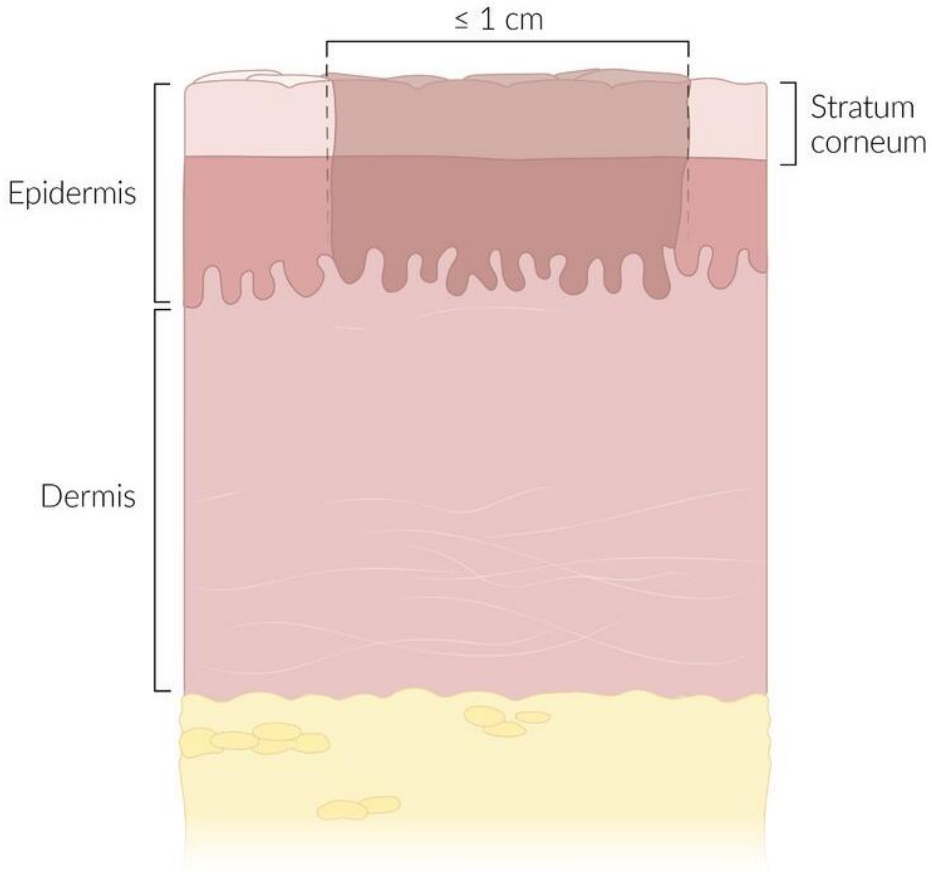
Warts distributed in a linear fashion at the site of a scar

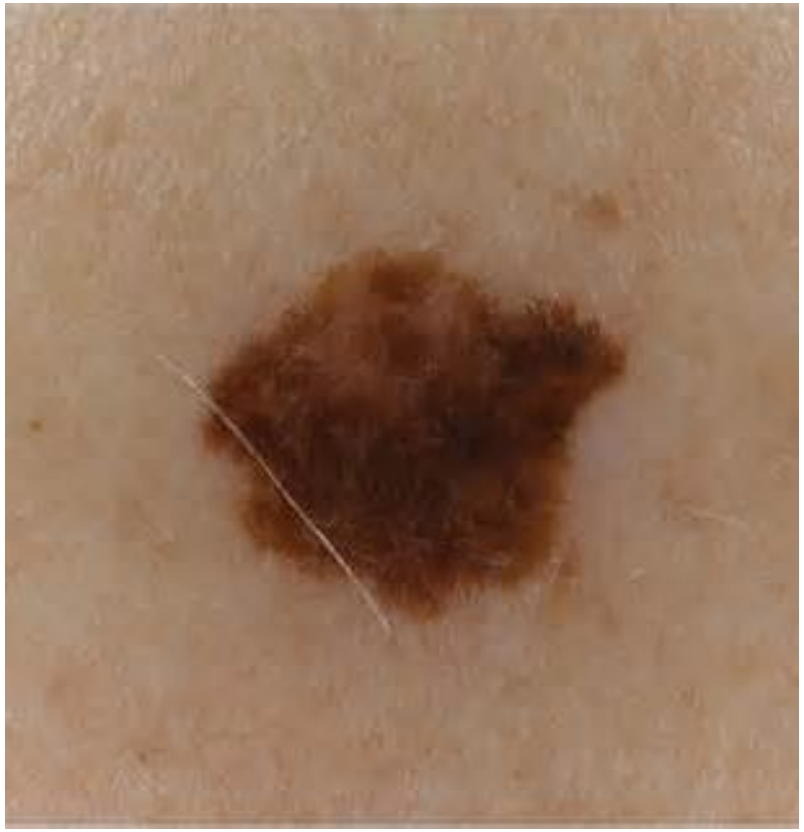
❖The linear arrangement of skin lesions in the Koebner phenomenon can be contrasted to both lines of Blaschko's and dermatomal distributions.

Primary skin lesions

1. Macule

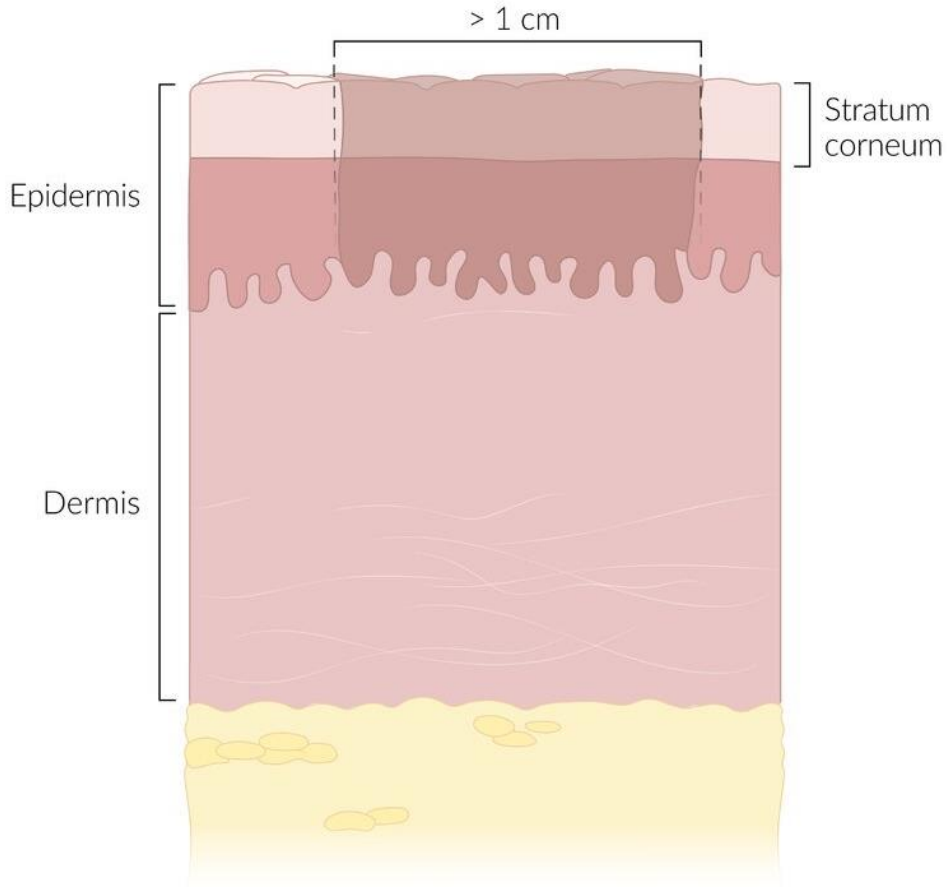
A flat (nonpalpable) skin lesion ≤ 1 cm in size that differs in color from surrounding skin (e.g., freckle; also seen in pityriasis versicolor, nevus spilus)

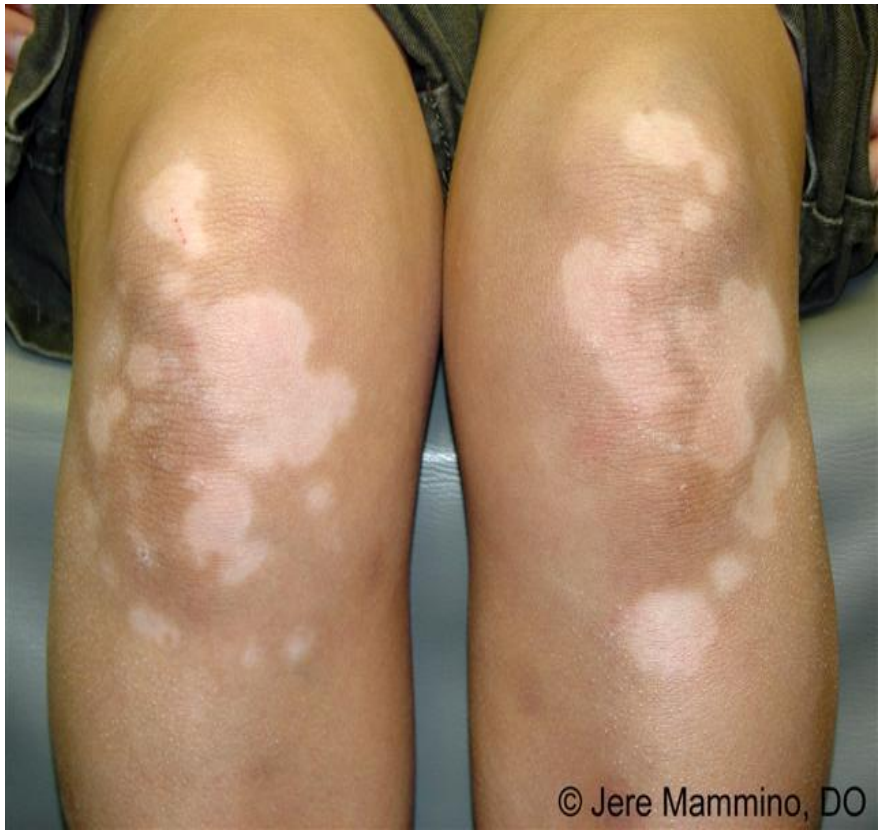




2. Patch

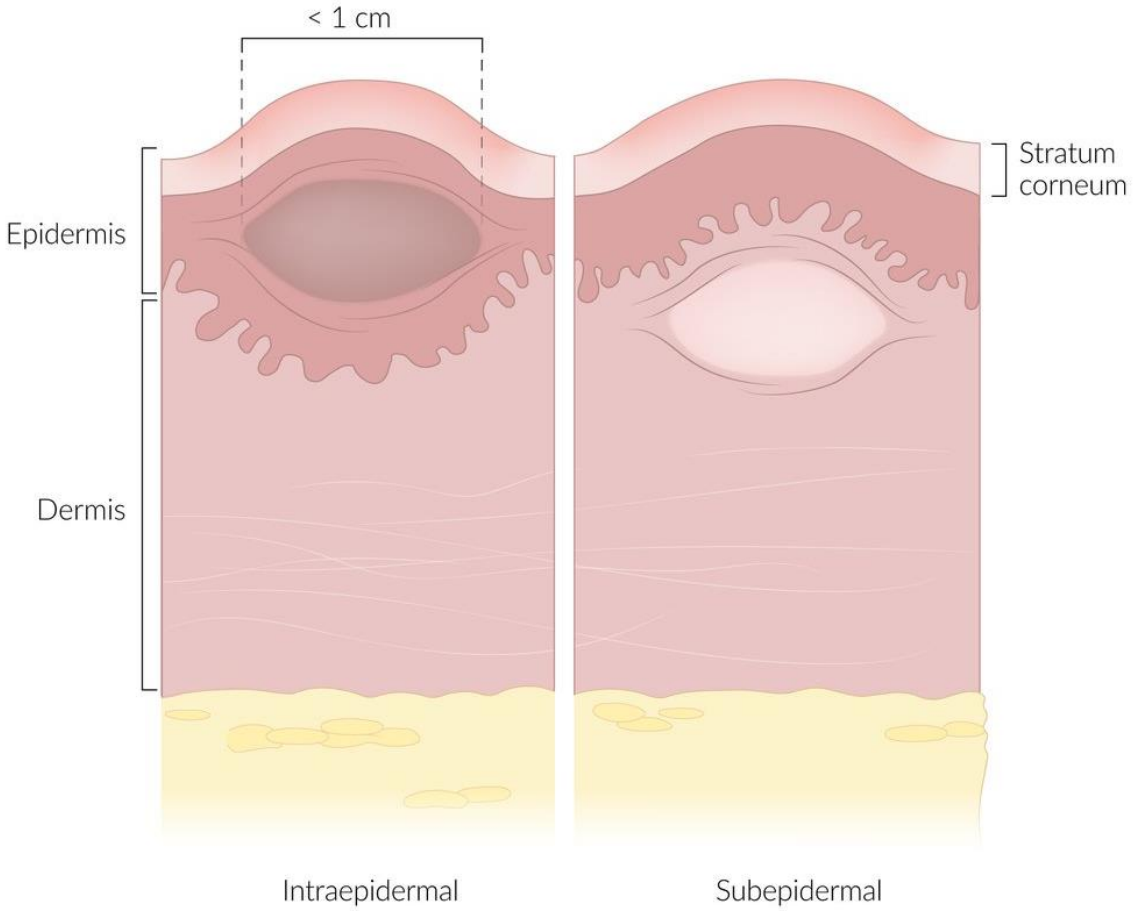
A flat skin lesion > 1 cm in size that differs in color from surrounding skin (e.g., congenital nevus)

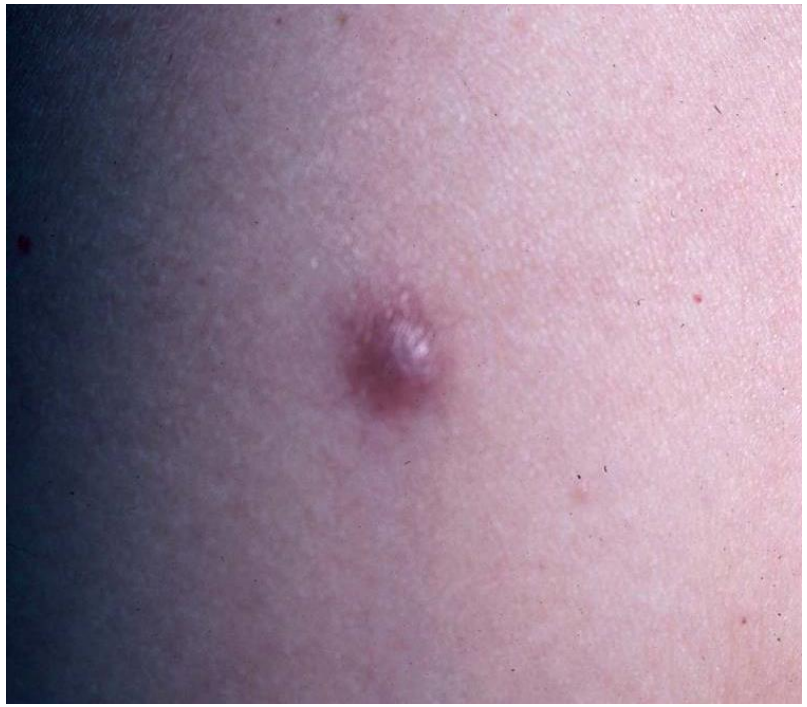




3. Papule

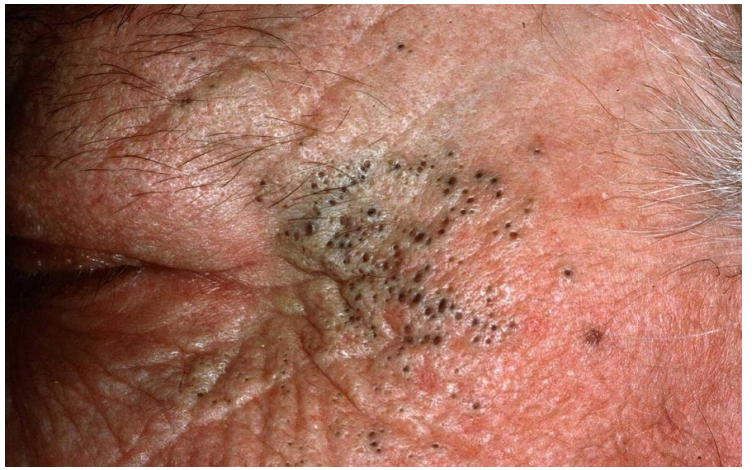
A small, palpable skin lesion ≤ 1 cm in diameter (e.g., seen in lichen planus, molluscum contagiosum, neurofibromatosis type 1, acne)

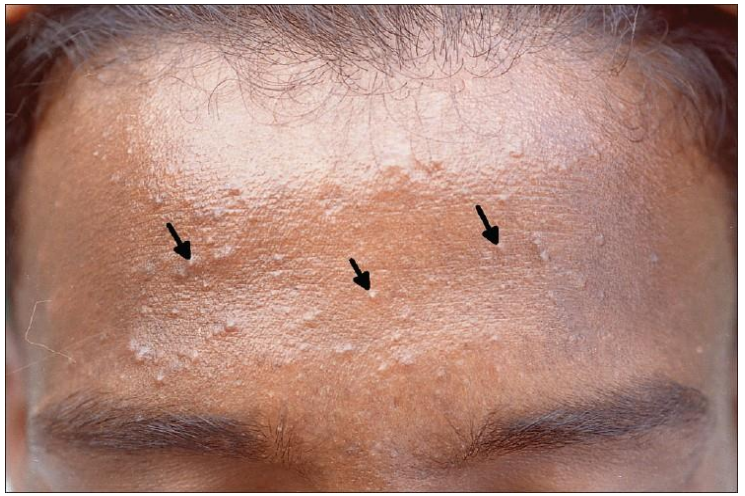




4. Comedone

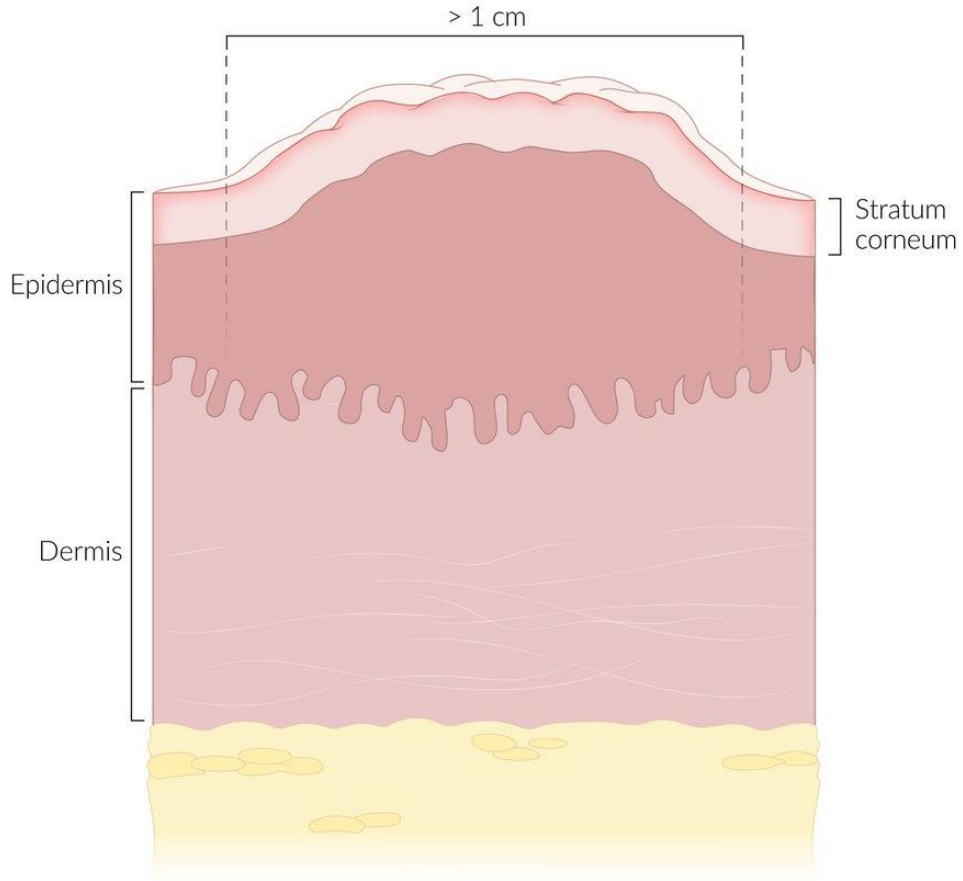
- A skin-colored papule that forms when pilosebaceous ducts become blocked with keratinaceous debris and sebum (e.g., due to acne vulgaris).
- Subtypes include closed comedones (whiteheads) and open comedones (blackheads).

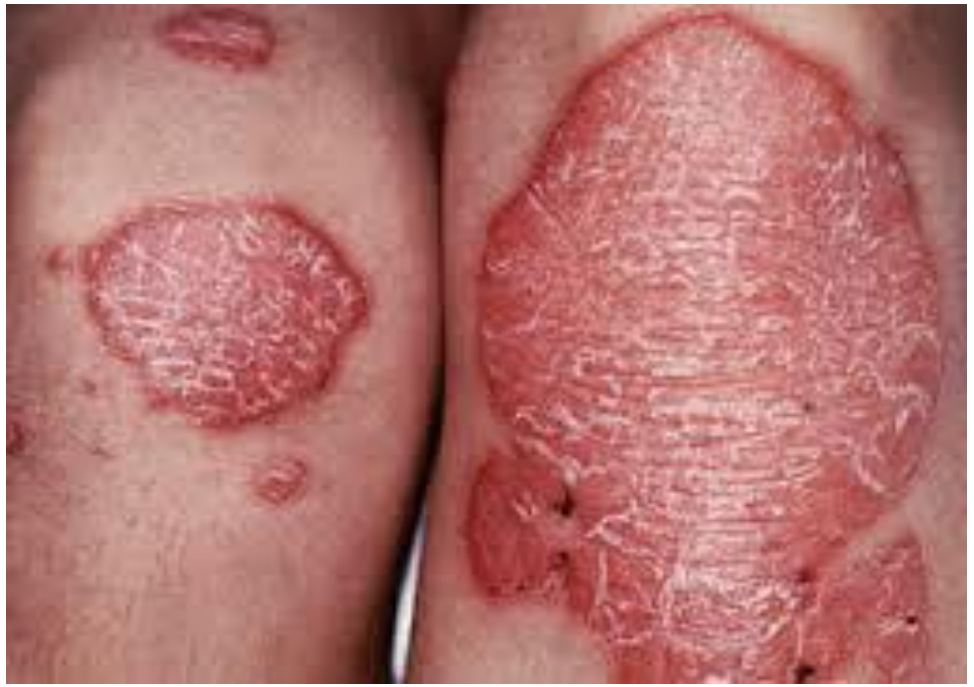




5. Plaque, Define plaque

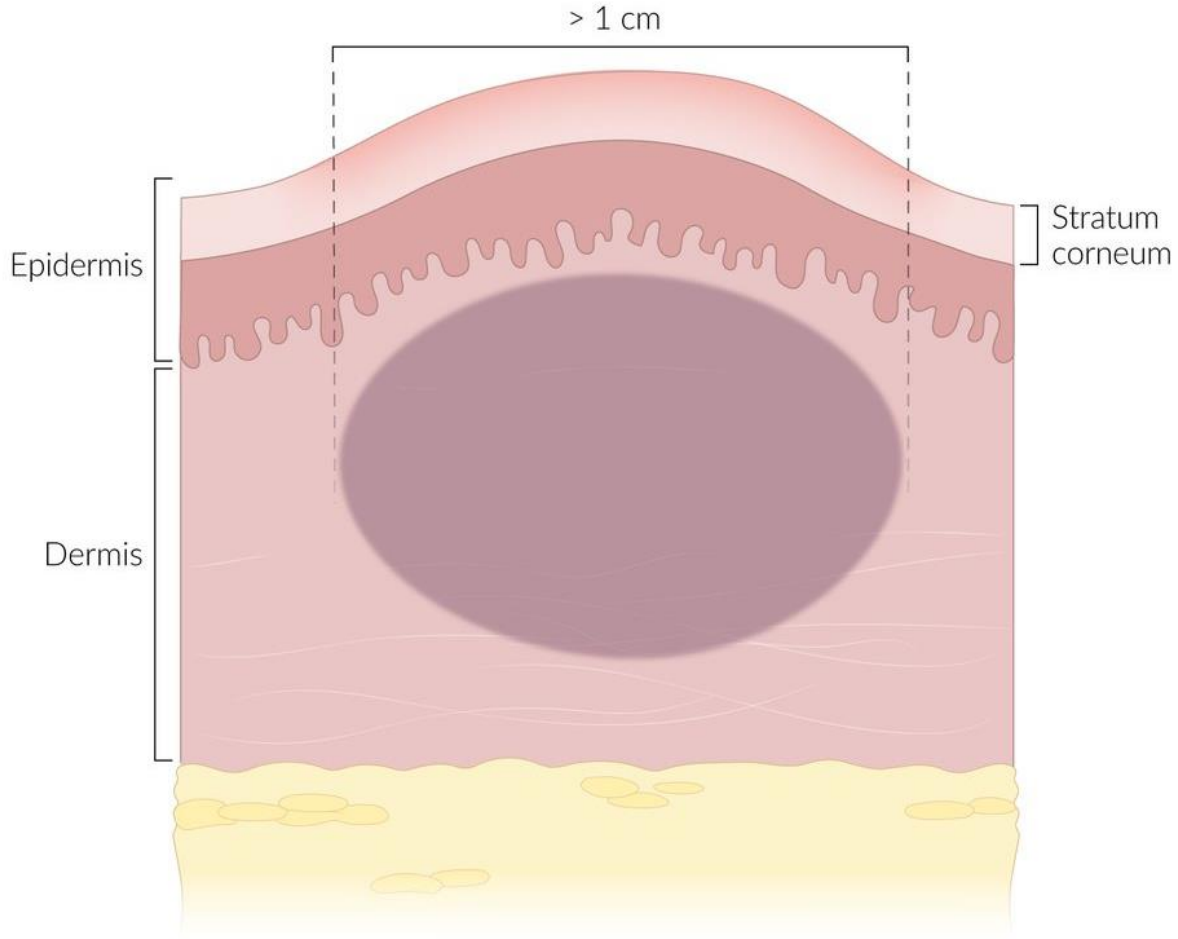
Palpable, usually raised lesion > 1 cm (e.g., seen in pigmented BCC, pityriasis rosea, necrobiosis lipoidica, psoriasis)

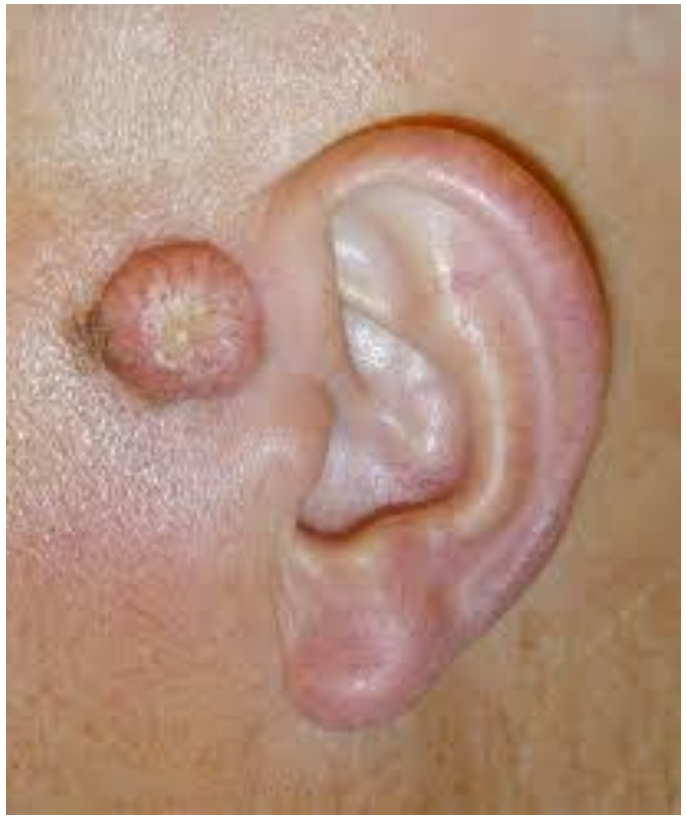




6. Nodule

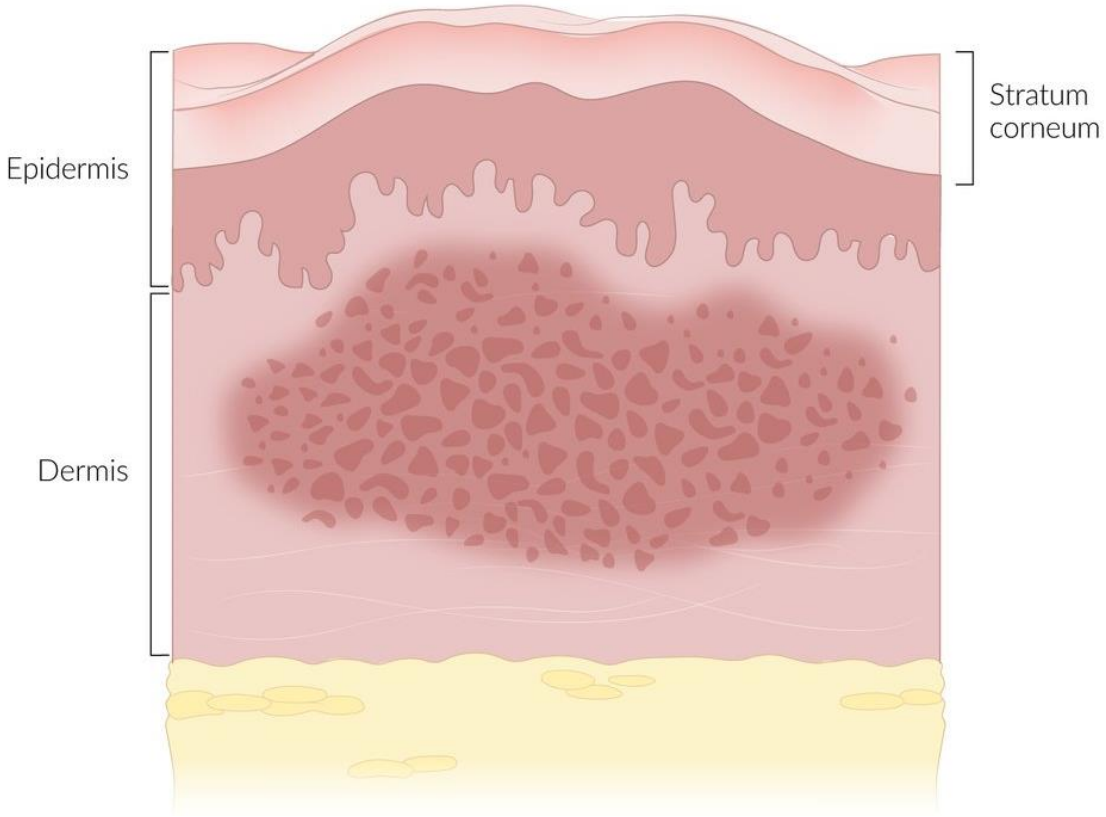
An elevated lesion, > 1 cm in both diameter and depth

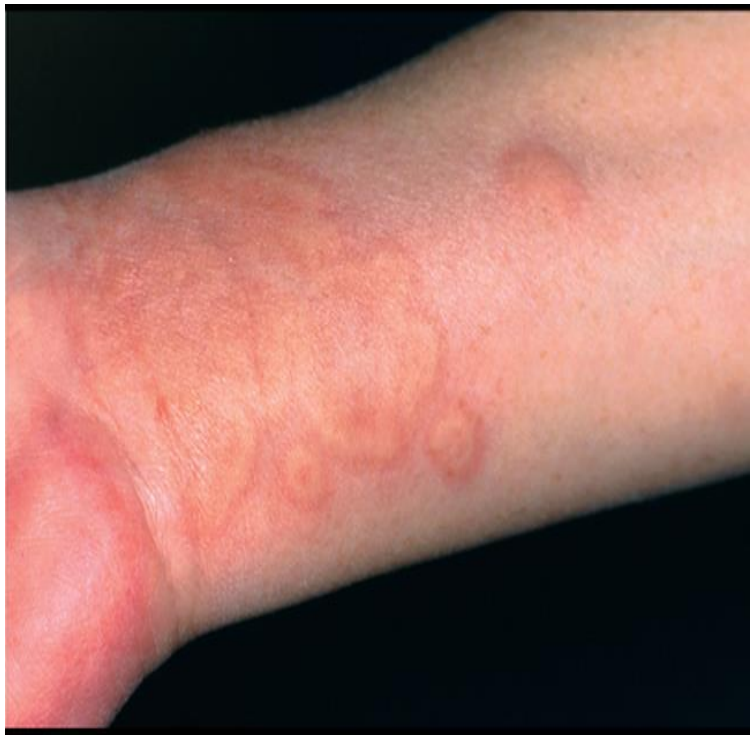




7. Wheal

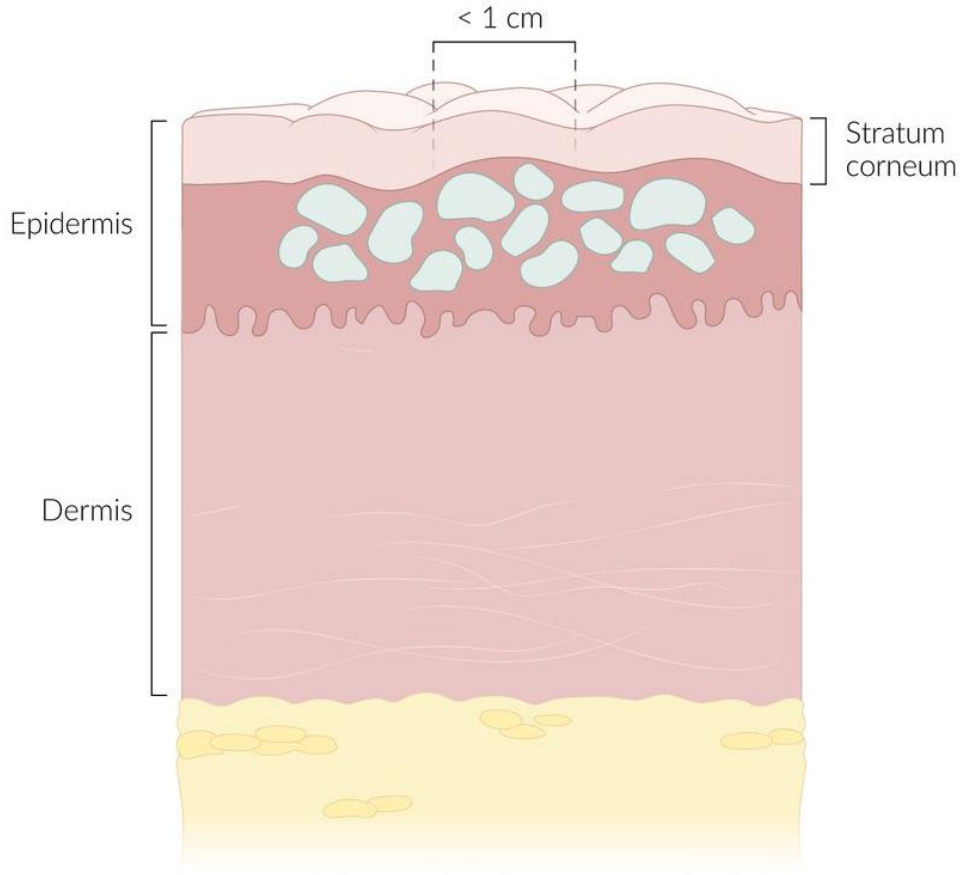
- Well-circumscribed, pruritic, and erythematous papule or plaque with dermal edema and irregular borders (e.g., seen in urticaria)
- Transient (hours to days)

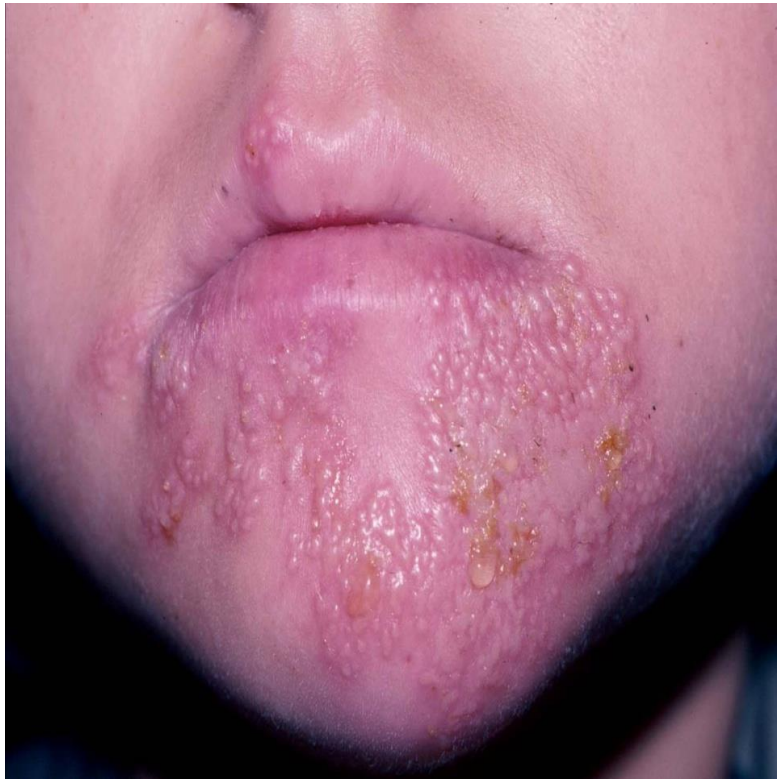




8. Vesicle

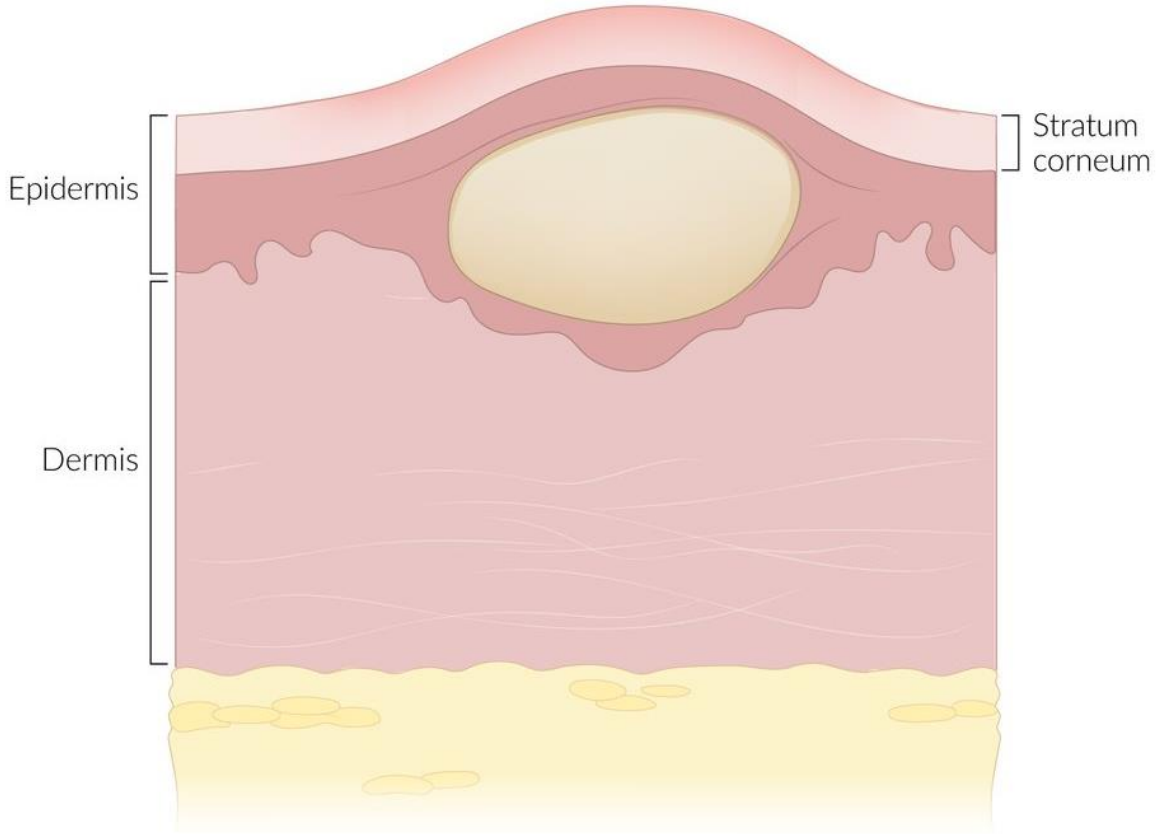
Small fluid-containing blister (collection of fluid in the skin) ≤ 1 cm in diameter (e.g., seen in eczema herpeticum, chickenpox, herpes zoster)

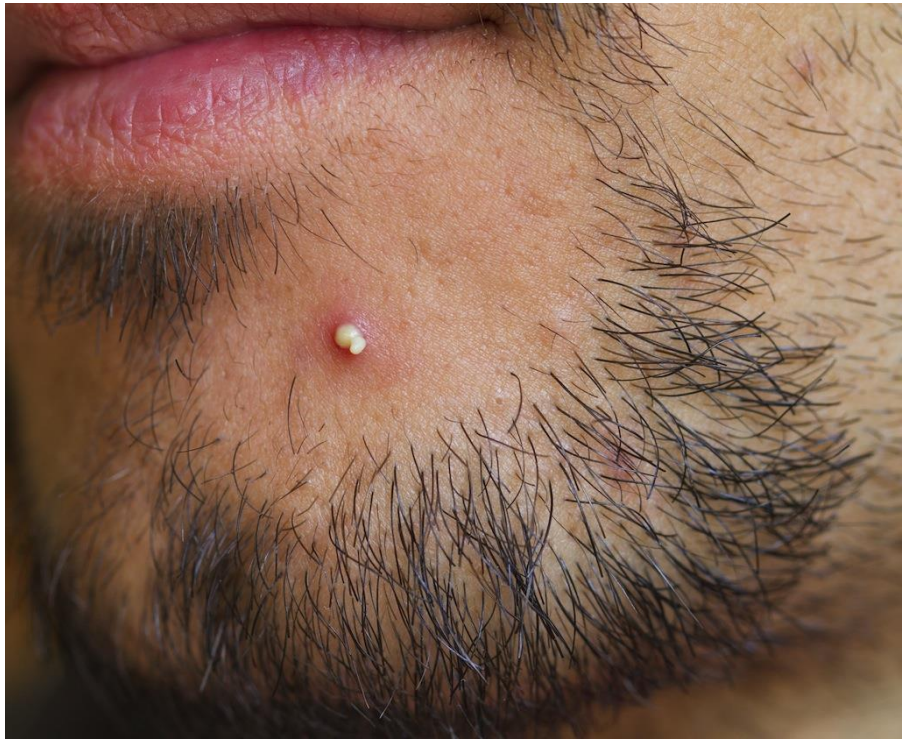




9. Pustule

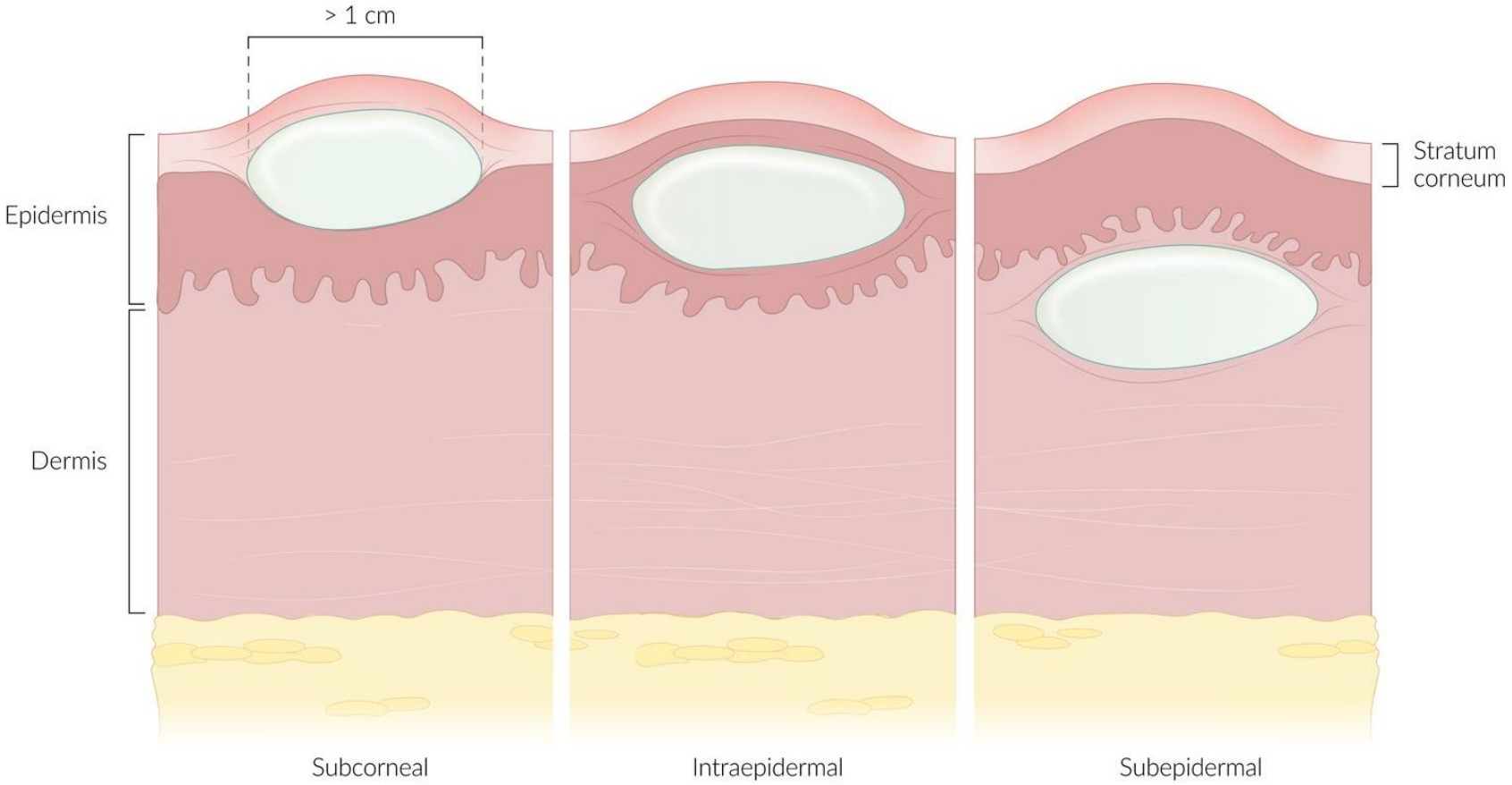
Vesicle filled with pus (e.g., seen in pustular psoriasis)

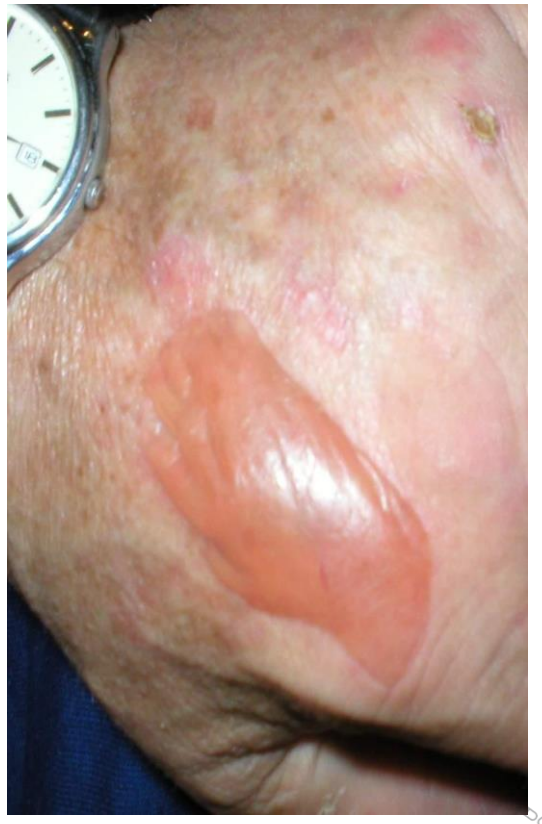




10. Bulla

❖Large fluid-containing blister > 1 cm in diameter (e.g., see in bullous pemphigoid, Stevens-Johnson syndrome)





What is the name of this primary skin lesion?

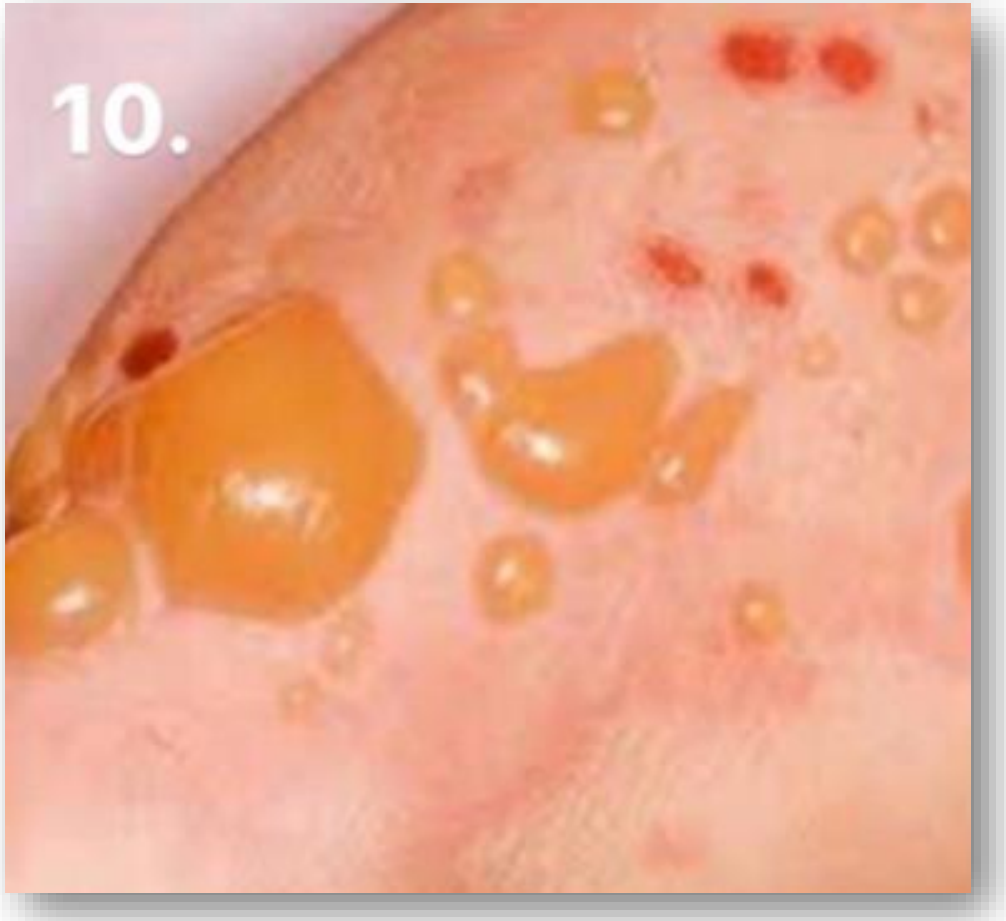
A. Bulla

B. Pustule

C. Nodule

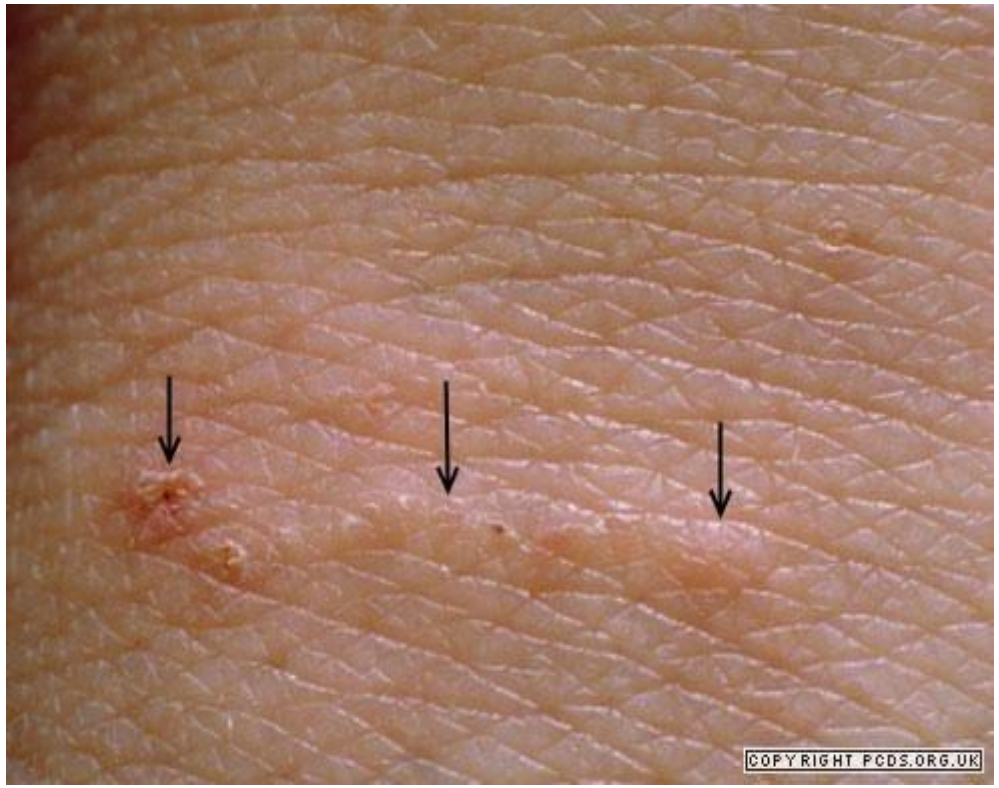
D. Papule

E. Crust



11. Burrow

- Slightly elevated, grayish, tortuous line in the skin ended by papule.
- Example : scabies



What is the primary skin lesion of the following ?

Bacterial skin infections

إضافي ❖Non-bullous Impetigo: Vesicle or pustule

إضافي ❖Bullous Impetigo: Bullae

سنوات (1) ❖Folliculitis: Pustule

إضافي ❖Erythrasma: well-defined pink or brown patches

سنوات (1) ❖Pitted keratolysis: whitish skin and clusters of punched-out pits

What is the primary skin lesion of the following ?

Viral skin infections

سنوات (1) ❖Herpes simplex: Vesicle

سنوات (1) ❖Herpes zoster: Vesicles / blisters

إضافي ❖Common warts: Papules or plaques

سنوات (1) ❖Flat (Plana) Wart: Small, smooth, flesh-colored, flattened wart

إضافي ❖Periungual wart: a cauliflower-like cluster of warts

إضافي ❖Orf: Nodule

سنوات (1) ❖molluscum contagiousm: Papule

سنوات (2) ❖Hand Foot Mouth disease: Vesicle

What is the primary skin lesion of the following ?

Infestation

سنوات (5) ❖Scabies: Burrow

Acne

سنوات (4) ❖Acne vulgaris: Comedone

سنوات (4) ❖Drug eruptive acne: Monomorphic eruption of papules and pustules

Eczema

سنوات (2) ❖Dyshidrotic dermatitis (pompholyx): Blisters on hands and feet

Psoriasis

سنوات (1) ❖Psoriasis: Plaque

What is the primary skin lesion of the following ?

Pigmentary disorders

سنوات (1) ❖Junctional nevus: Hyperpigmented macule

سنوات (1) ❖Freckle: Hyperpigmented macule

سنوات (2) ❖Lentigines: Hyperpigmented patch or macule

سنوات (1) ❖Café-au-lait: patch

سنوات (2) ❖Melasma: tan or brown patch on sun exposed area

سنوات (4) ❖Vitiligo: milky white depigmented patch

سنوات (1) ❖Halo nevus: mole surrounded by a white ring

What is the primary skin lesion of the following ?

Bullous dermatosis

سنوات (1) ❖Pemphigus vulgaris: Bulla

سنوات (1) ❖Bullous pemphigoid: Tense subepidermal bulla

سنوات (1) ❖Dermatitis herpetiformis: Vesicles

سنوات (1) ❖Erythema multiforme: Target lesions

Urticaria

سنوات (4) ❖Urticaria: Wheal or hives

سنوات (1) ❖Insect bite: Bulla or wheal

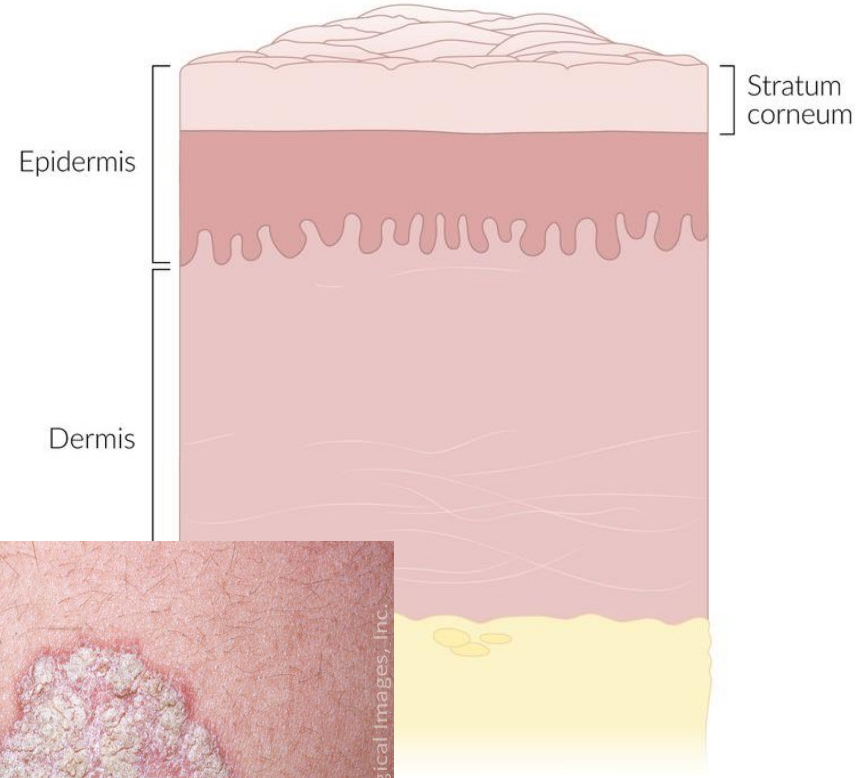
Papulosqaumous disorders

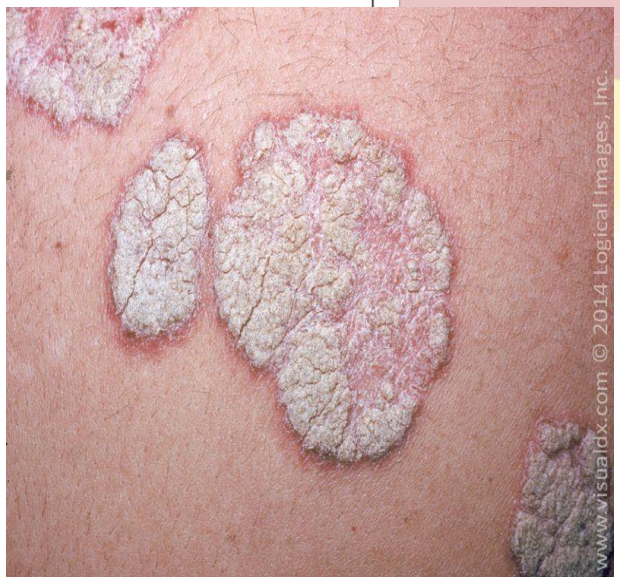
سنوات (1) ❖Lichen planus :Papules

Secondary skin lesions

1. Scale

- Thickened stratum corneum
- Scales are flaky, dry, and usually whitish.
- In contrast, crusts are more often moist and yellowish or brown.
- E.g., seen in ichthyosis vulgaris, squamous cell carcinoma, eczema, psoriasis

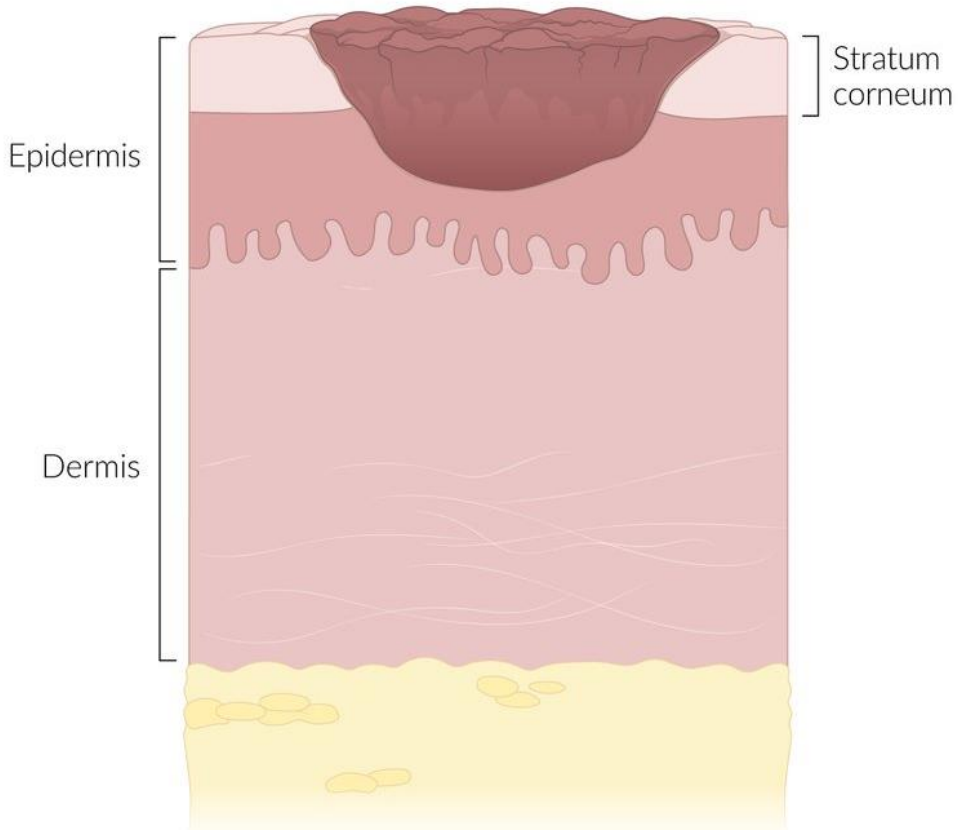


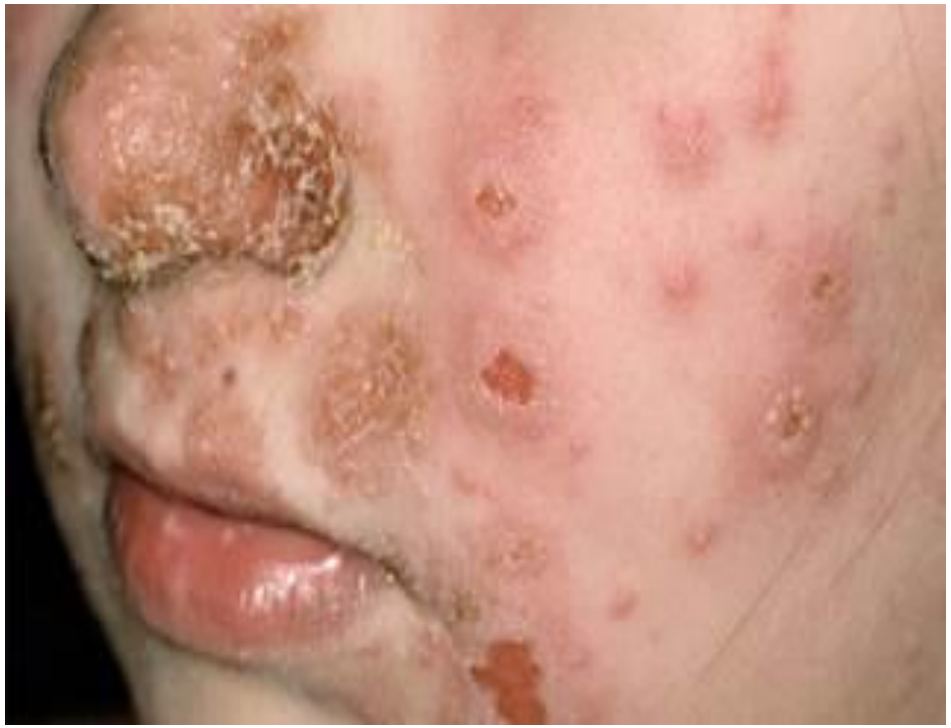


2. Crust

سنوات (1)

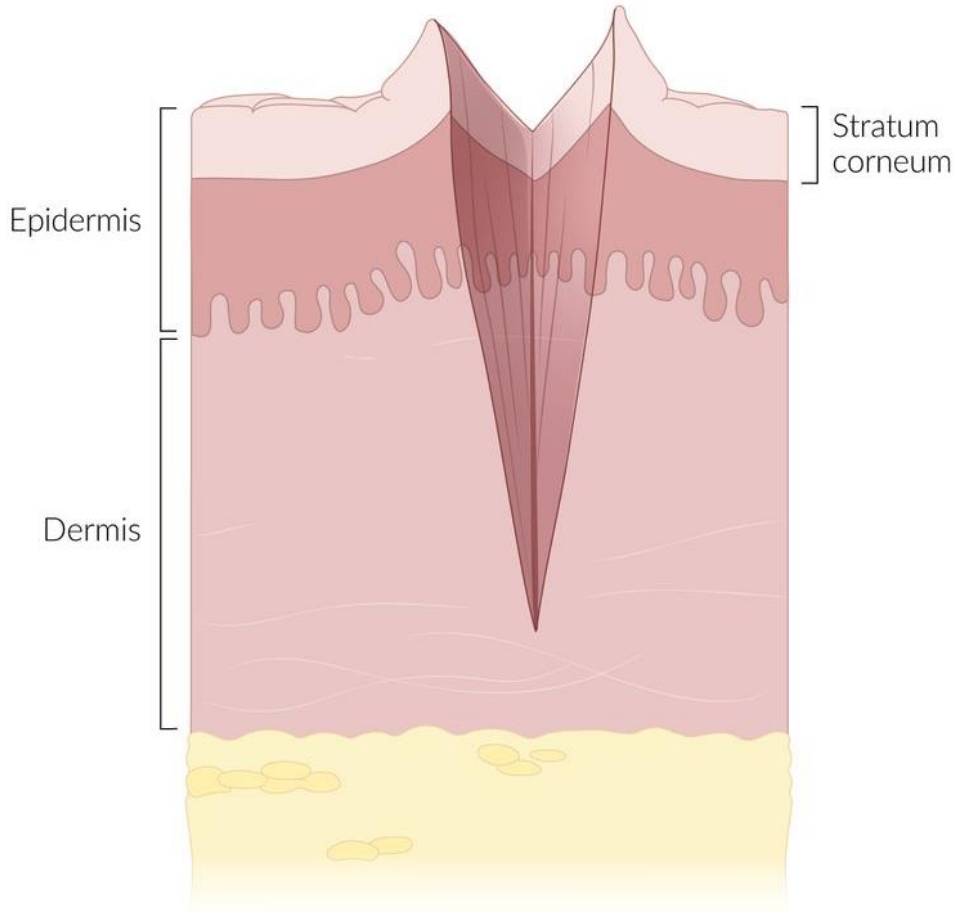
- Dried exudates such as pus or blood
- E.g., seen in atopic dermatitis, non-bullous impetigo





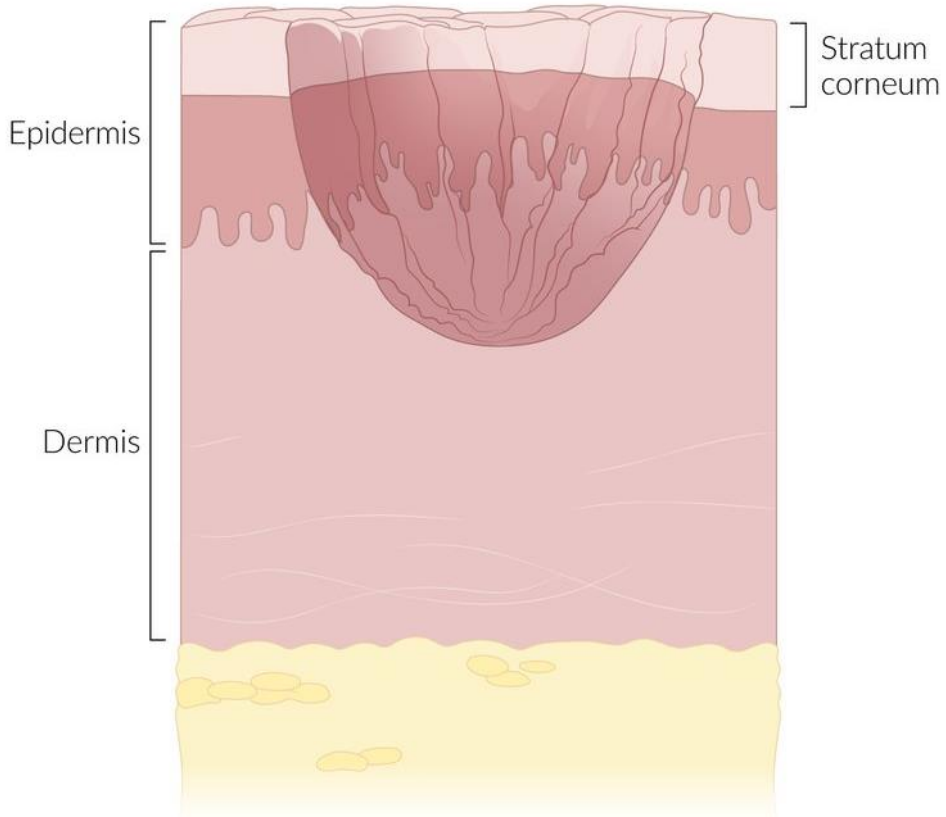
3. Fissure (cleft)

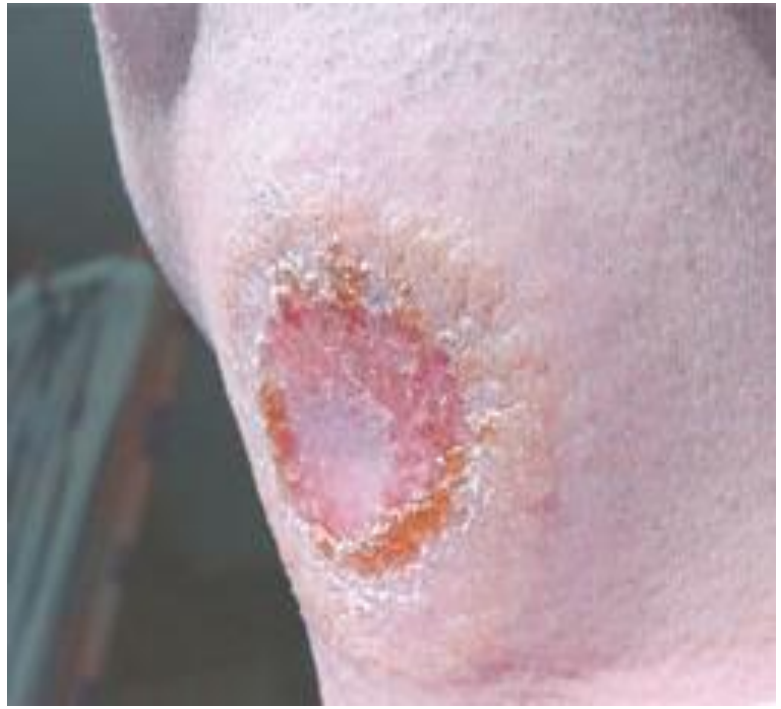
Linear crack through the epidermis that extends into the dermis



4. Ulcer

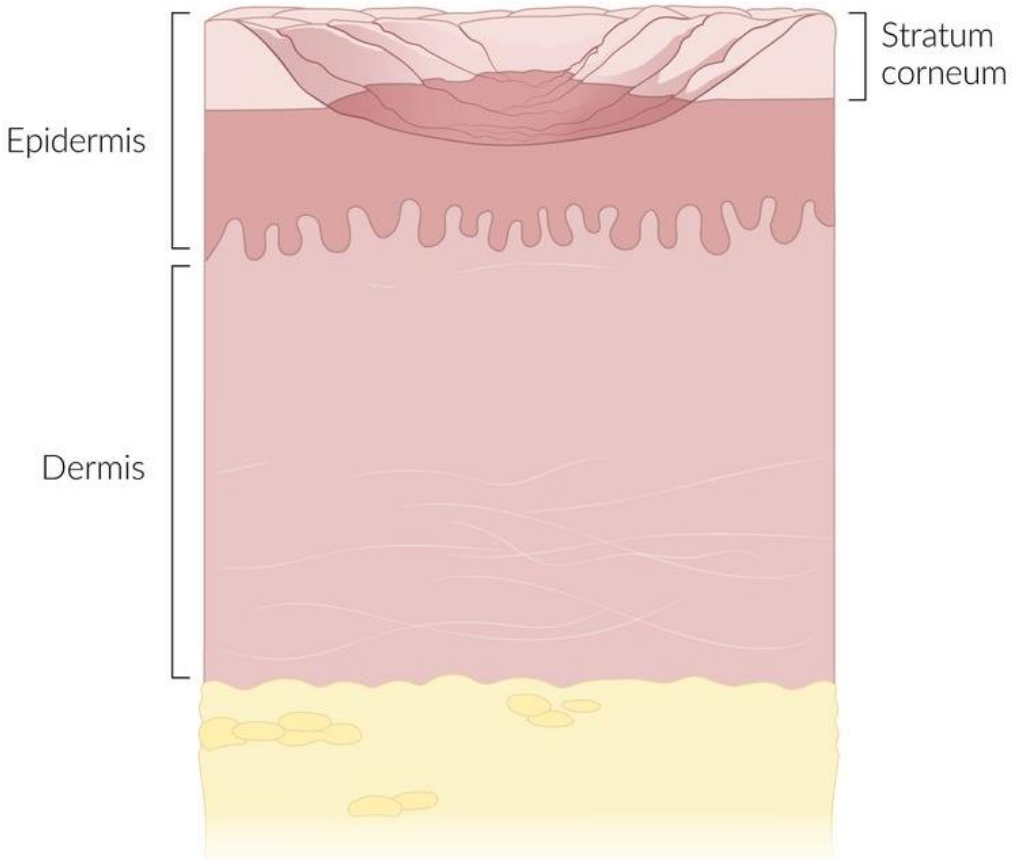
- Rounded or irregularly shaped deeper lesions that result from loss of the epidermis and some portion of the dermis.
- Ulcers usually leave a scar.





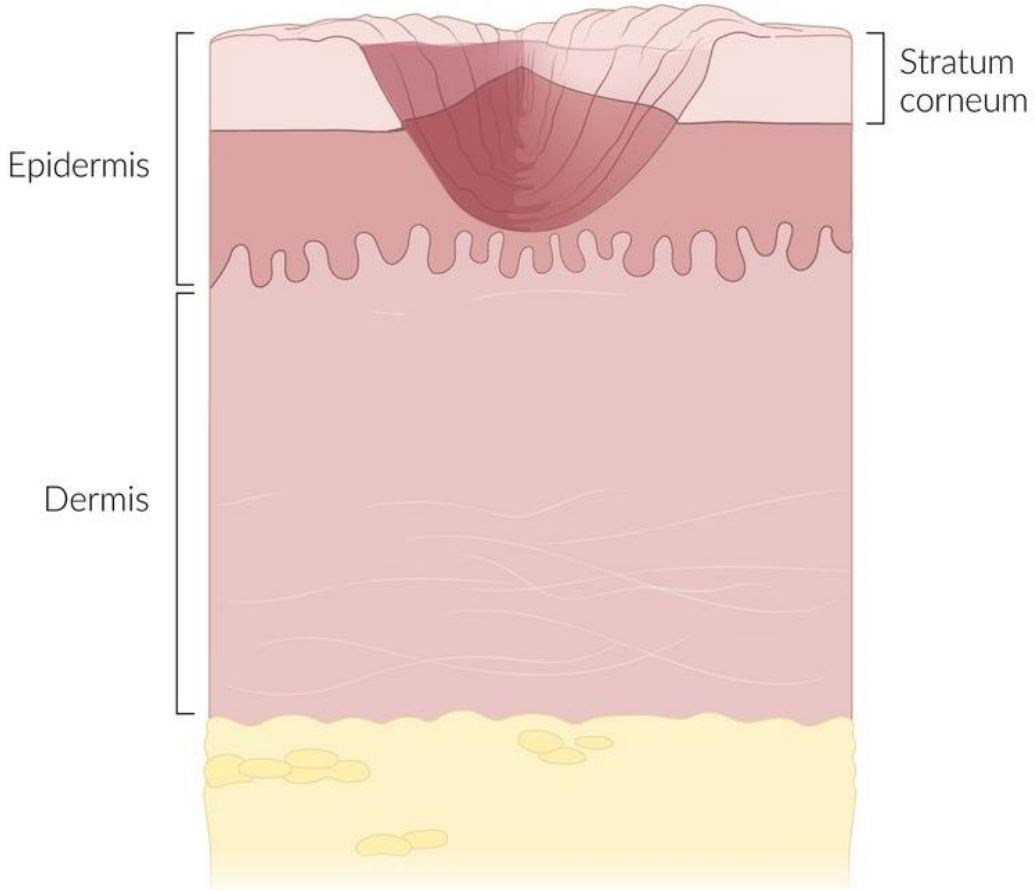
5. Erosion

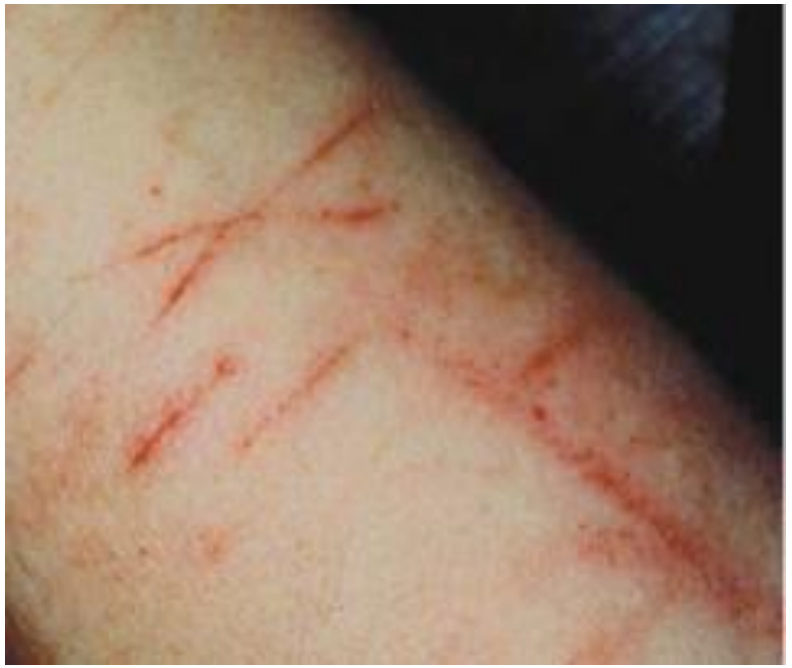
- Loss of all or portions of the epidermis
- Erosions usually heal without a scar.



6. Excoriation (scratch marks)

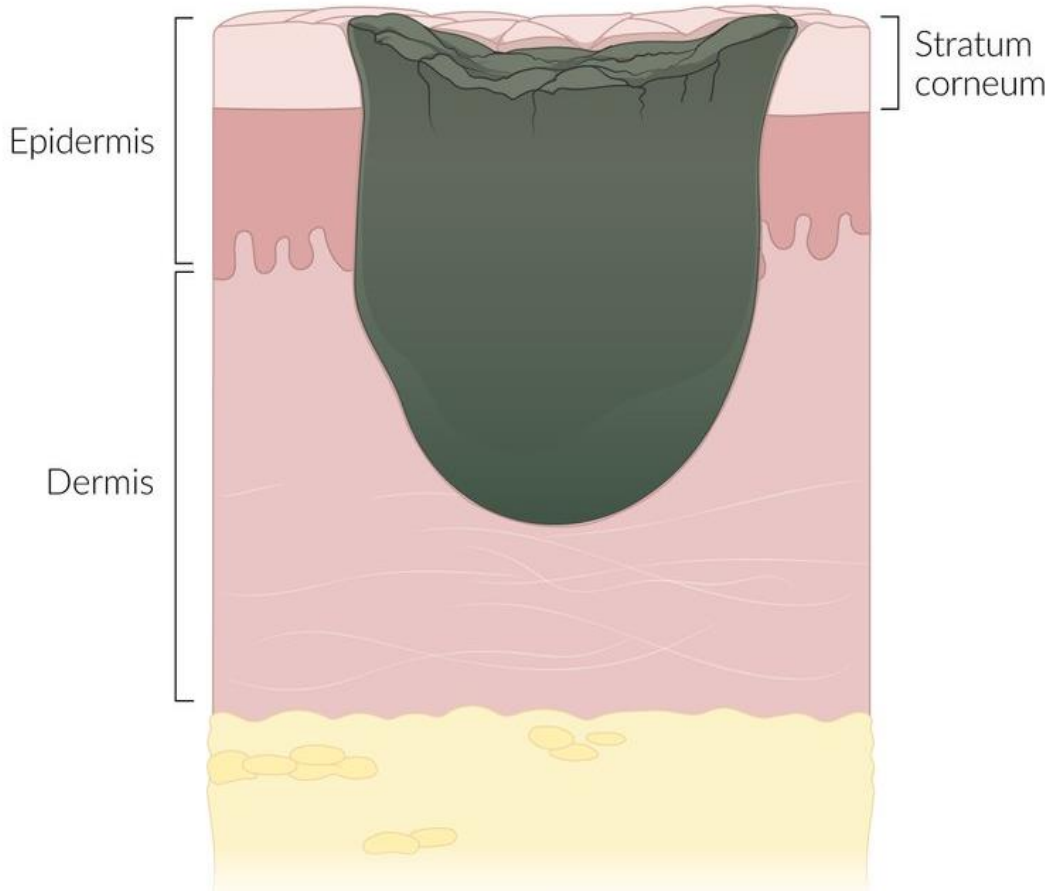
Abrasion produced by mechanical force, usually involving the epidermis (but may reach the outer layer of the dermis)





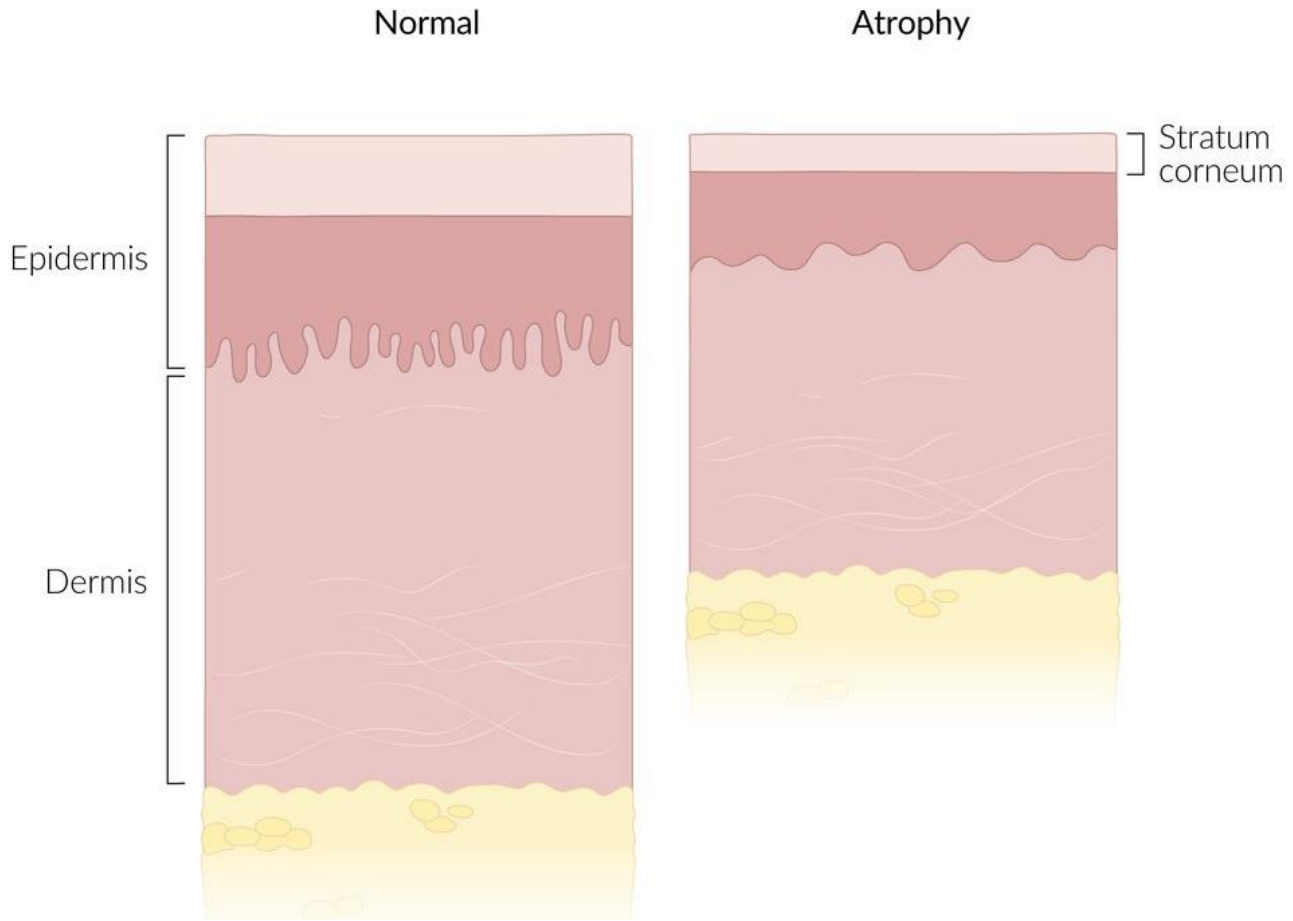
7. Necrosis

Dead skin tissue (Black or yellowish/brown)



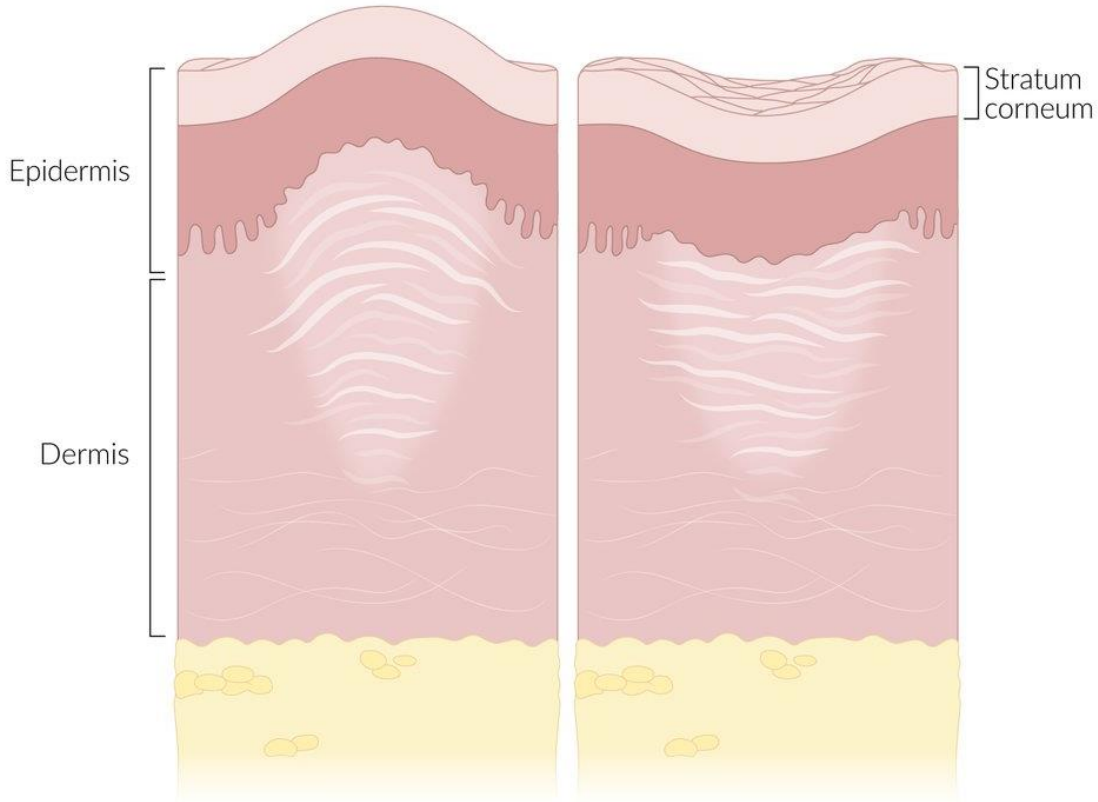
8. Skin atrophy

Thinning of skin without inflammation



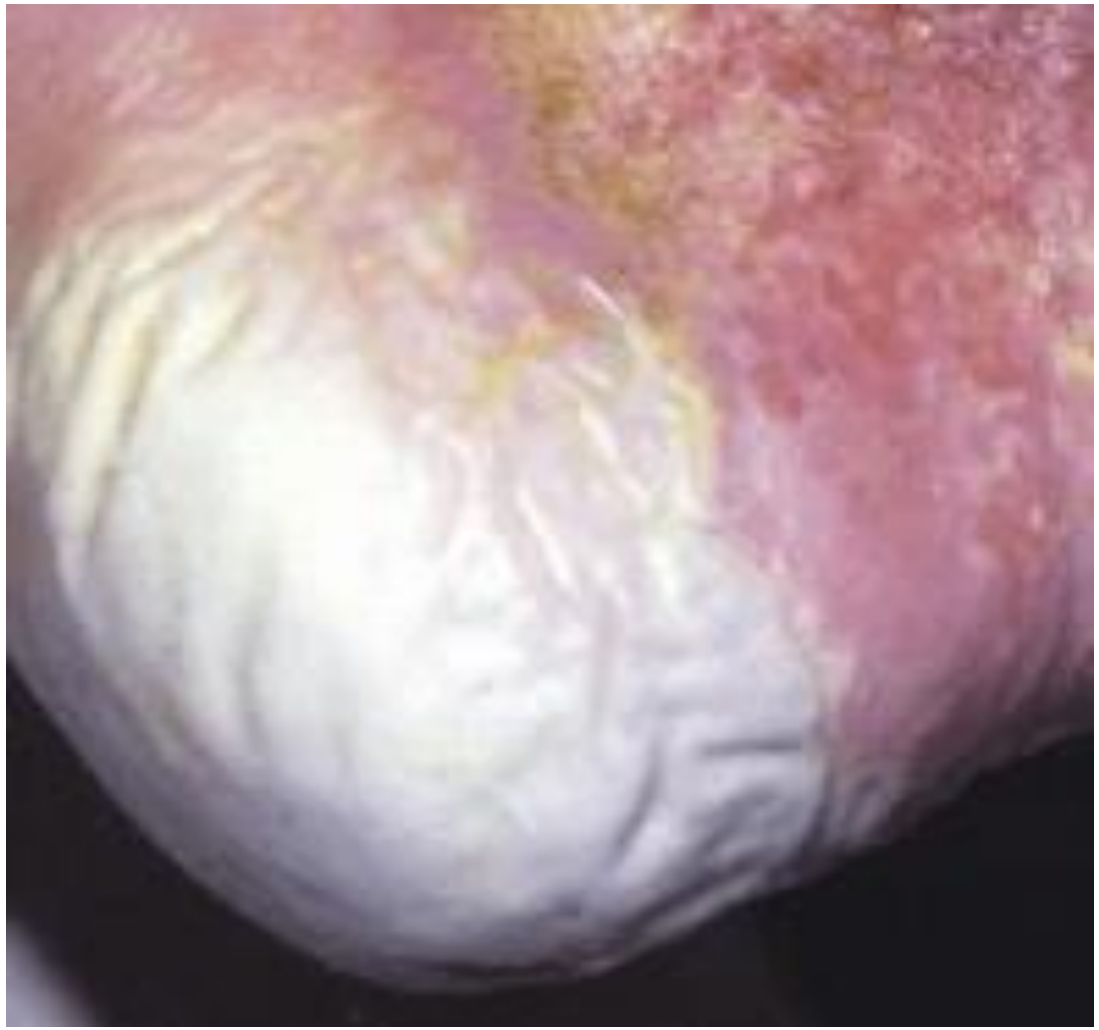
9. Scar

- Composed of new connective tissue that has replaced lost substance
- An overgrowth of scar tissue manifests as a keloid (thickened, raised tissue that grows beyond the borders of the scar and shows no regression).



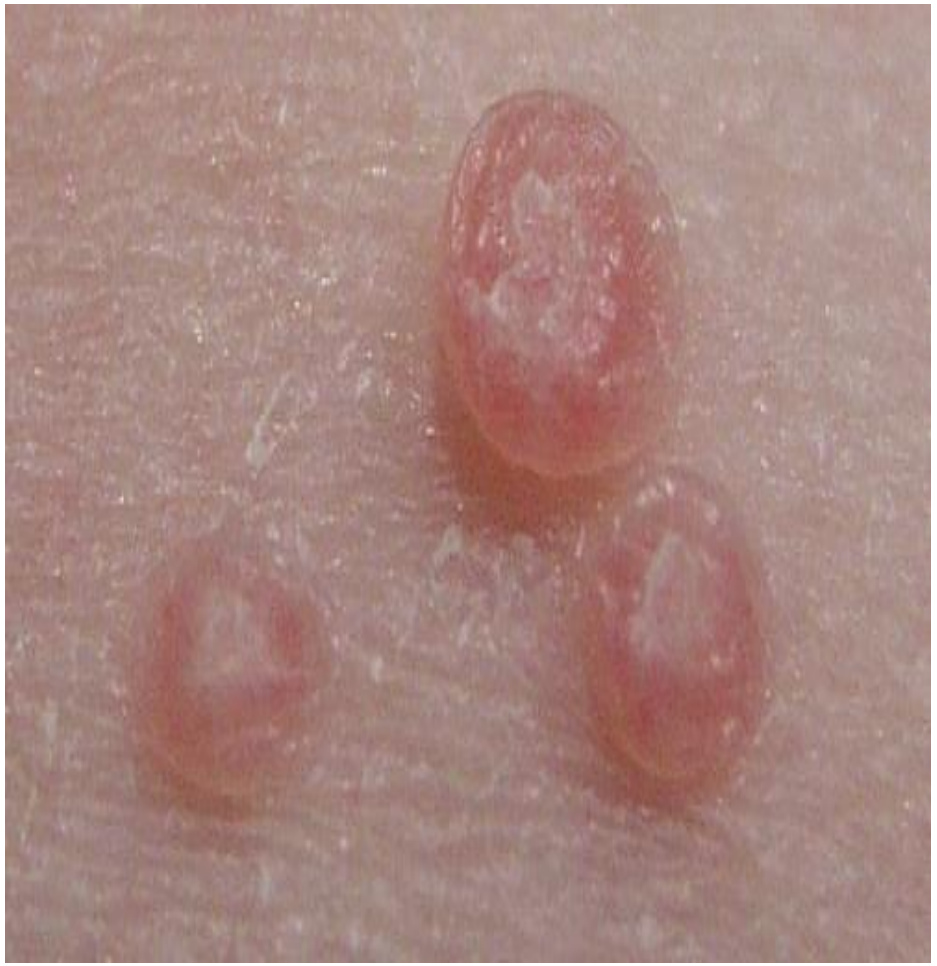
10. Maceration

- Swelling of tissue after prolonged contact with a fluid (e.g., maceration of skin after a long bath → "washerwoman skin")



11. Umbilication

- A descriptor for lesions that have a small depression (resembling the umbilicus).
- Examples include lesions of molluscum contagiosum and Penicillium marneffei infection.



Complex skin lesions

1. Hemorrhage

A. Hematoma

B. Purpura
   - A subtype of hematoma that does not blanch upon the application of pressure
   - Nonpalpable purpura
     - Petechiae
     - Ecchymosis
   - Palpable purpura

A. Hematoma

- Caused by bleeding into subcutaneous tissue, muscle, organ tissue or a cavity
  - Immediately after trauma: red
    - Cause: release of hemoglobin
  - After 24–96 h: dark red, green, blue, purple, black
    - Cause: coagulation of the blood and degradation of hemoglobin into bile pigment
  - After 4–7 days: dark green
    - Cause: breakdown of heme into biliverdin
  - After 7 days: yellow; brownish
    - Cause: breakdown of biliverdin into bilirubin

B. Purpura

Definition: a subtype of hematoma that does not blanch upon the application of pressure

- **Nonpalpable purpura**
  - **Petechiae**: Flat, red-purple, pinpoint lesions < 3 mm in size
  - **Ecchymosis**: Flat, red-purple, larger form of petechiae, > 5 mm in size
- **Palpable purpura**: Raised, red-purple lesions

2. Rashes

A. **Exanthem**: Extended uniform rash (localized or generalized)

B. **Enanthem**: Rash confined to the mucous membranes

C. **Erythema**: Reddening of the skin as a result of vasodilation (blanches if pressure is applied)

D. **Erythroderma**: Generalized reddening of the skin

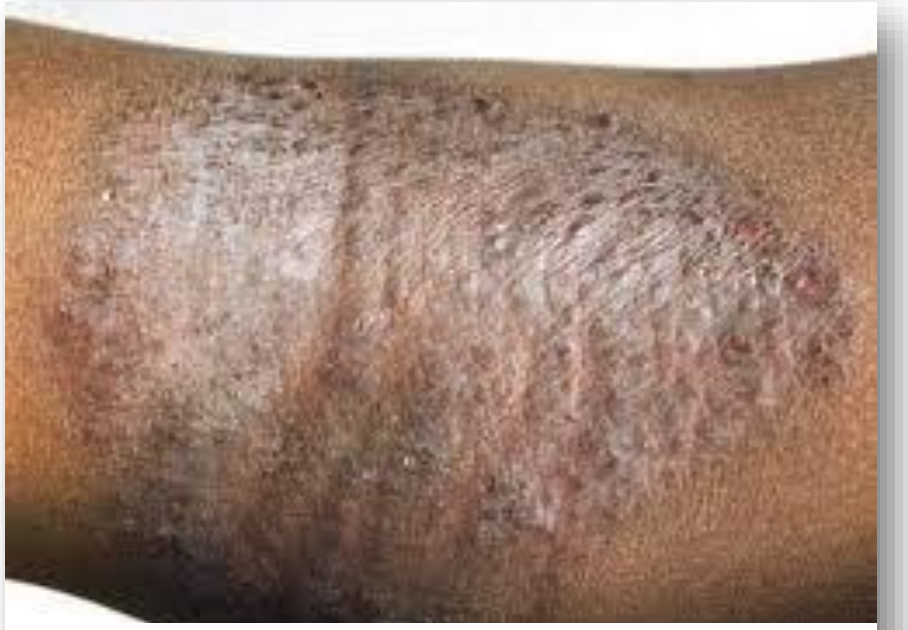
E. **Maculopapular rash**: Rash with both palpable and nonpalpable lesions ≤ 1 cm in size (e.g., seen in measles, infectious mononucleosis, secondary syphilis, fifth disease, rubella, roseola infantum)

3. Further lesions

سنوات (5)

A. Lichenification: Hard thickening of the skin with accentuated skin markings (can be considered a secondary lesion)

B. Eczema: Noncontagious dermatitis accompanied by pruritus, erythema, and papules



Lichenification

Clinical tests

KOH mount

❖Sample collection

- Skin: cleaned with alcohol, scraped with scalpel
- Hair: Plucked with forceps
- Nail: Undersurface of nail plate is scraped

❖KOH mount

- Skin & hair: 10& KOH added and heated left for half to 2 hours
- Nail: 20% KOH added and heated left for 24-48 hours

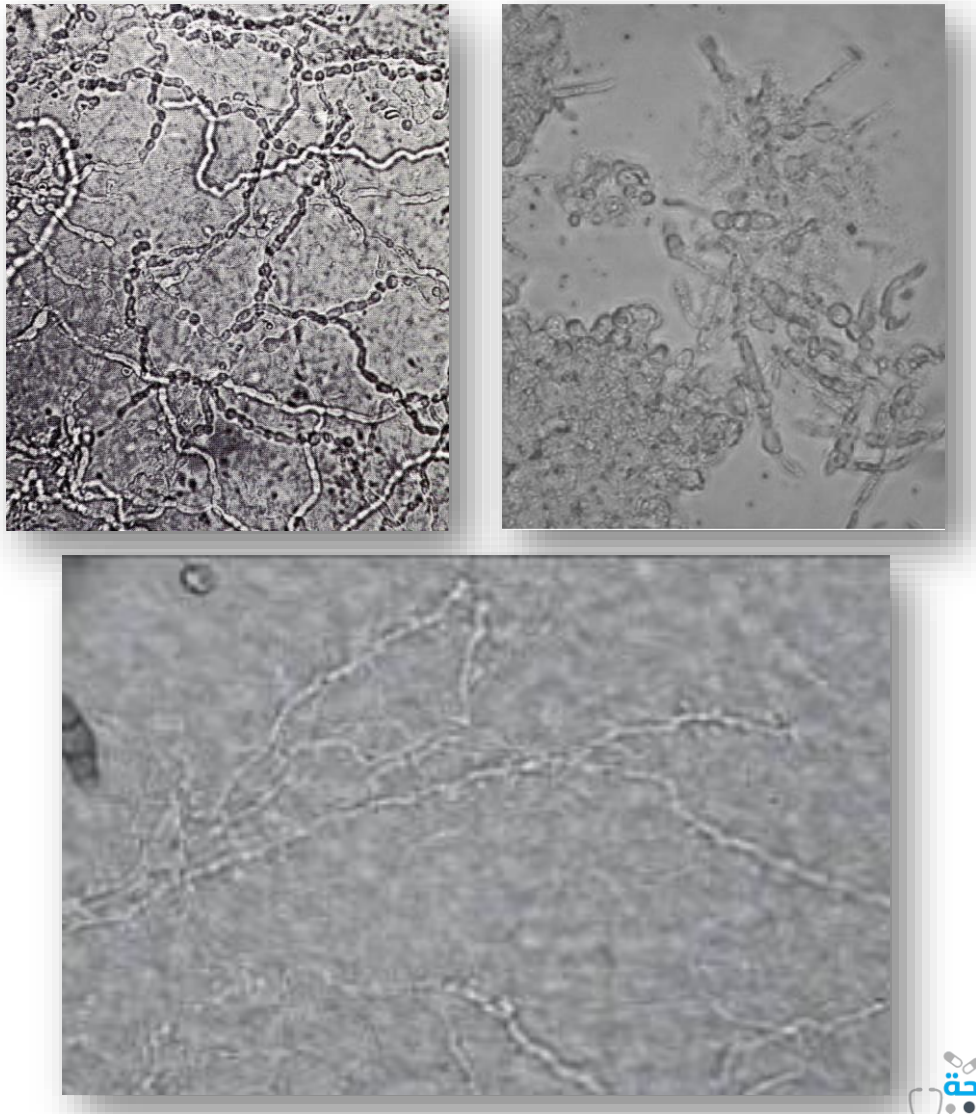
سنوات (1)

❖Findings:

- Dermatophytes: Hyphae of Tinea
- Tinea versicolor: "spaghetti and meatballs" appearance
- Candidiasis: Budding yeast with pseudo-hyphae

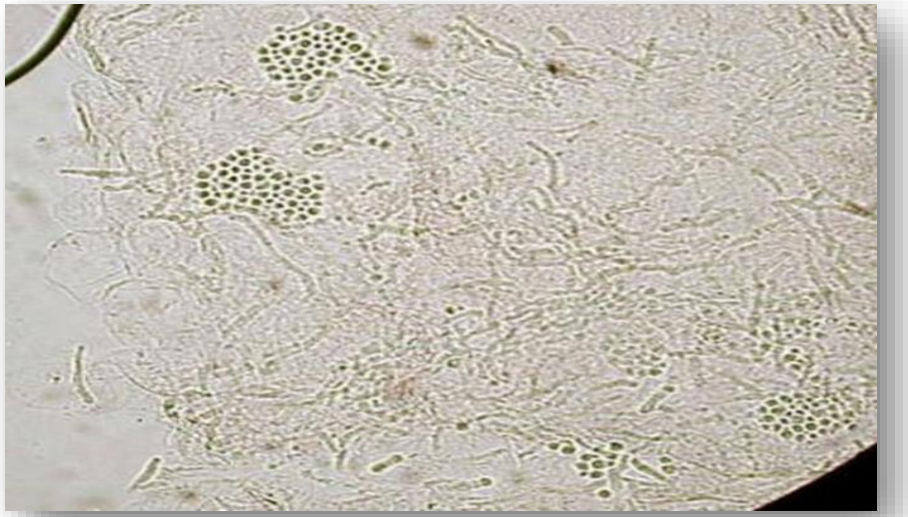
KOH test

- **What is the name of this study?**
  - KOH test
- **Define the KOH test**
  - A quick, inexpensive fungal test to differentiate dermatophytes and candida albicans symptoms from other skin disorders like psoriasis and eczema
- **What is the concentration of KOH for nail examination by this test is**
  - 20%



KOH test

- **What is the name of this study?**
  - KOH test
- **What is the characteristic findings seen in this prep**
  - Spaghetti and meatballs appearance
- **Diagnosis for this prep**
  - Tenia versicolor

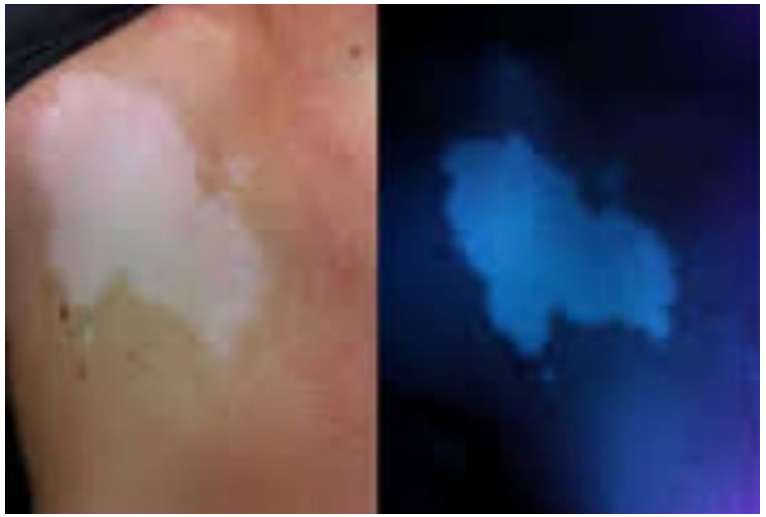


- **Mention 2 non-invasive diagnostic tests for fungal skin infection**
  - KOH prep
  - Wood's light

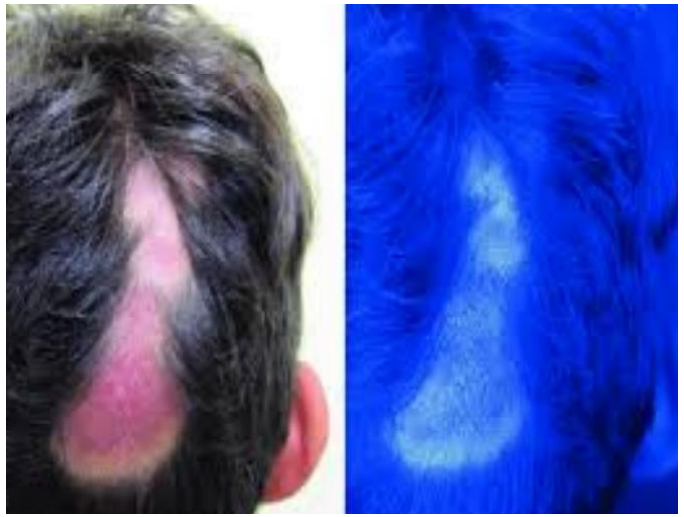
Wood's light test

- 365nm
- Why do we use it ?
  - Establish contrast between normal skin and hyper or hypo pigmented skin and detect infection
- Colors:
  - White/bright bluish: vitiligo
  - Pink: Erythrasma, pitted keratolysis
  - Green: Tinea capitis
  - Golden yellow: Tinea versicolor
  - Blue-green: Pseudomonas

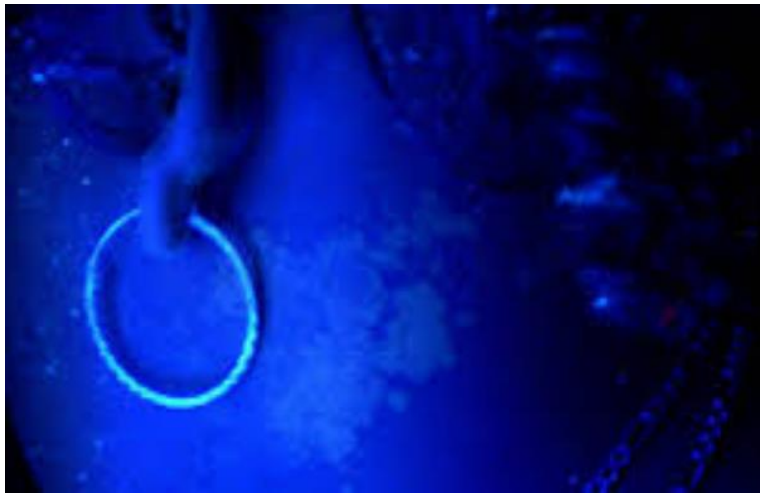
Wood's light test



Vitiligo

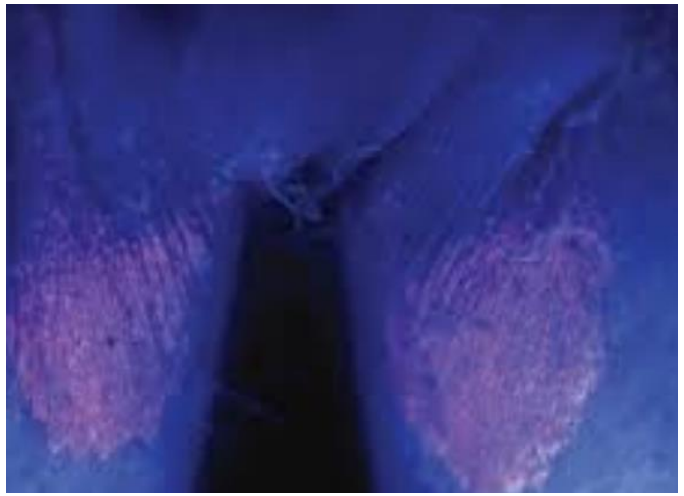


Tenia capitis



سنوات (2)

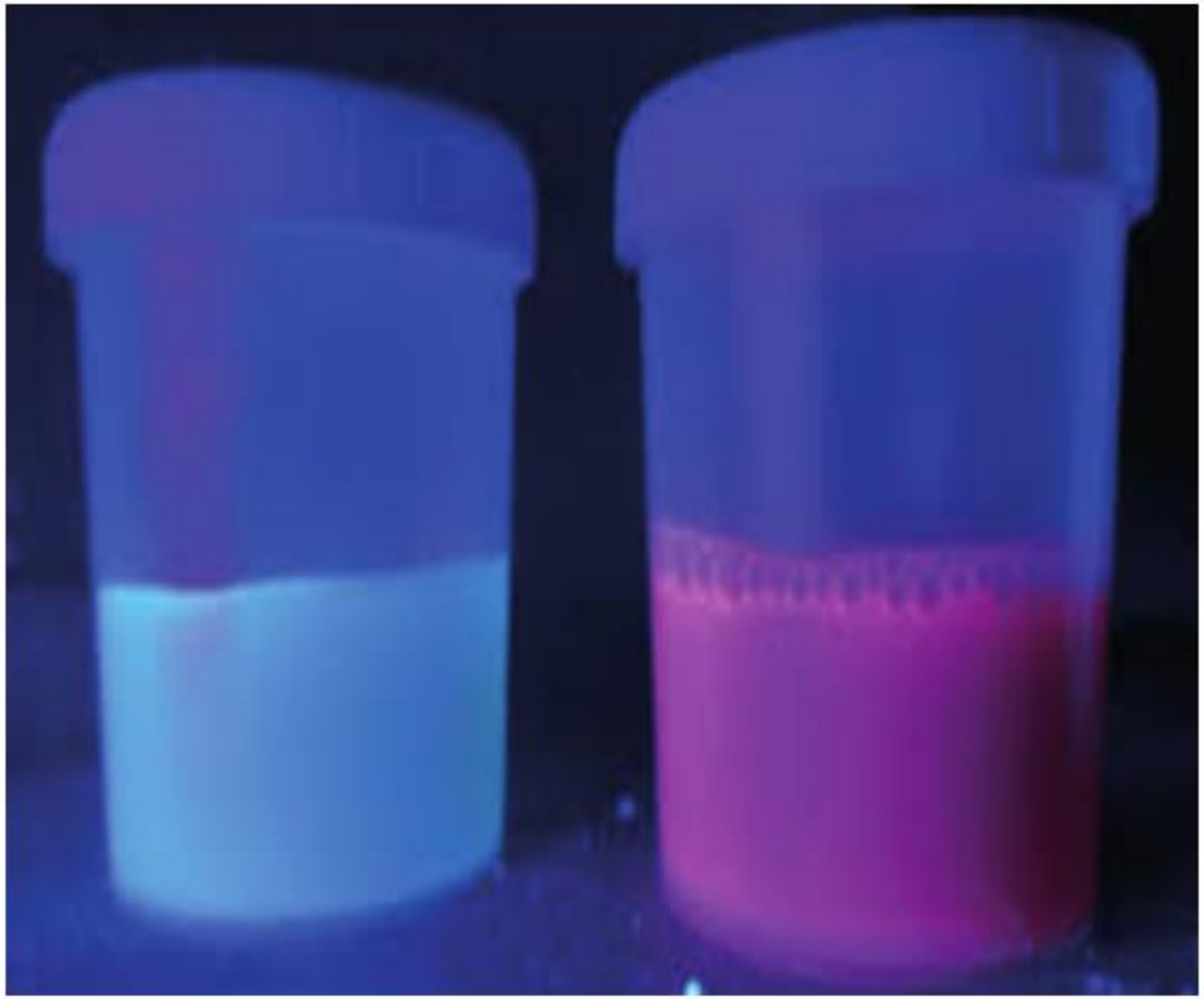
Tinea versicolor



سنوات (1)

Erythrasma

Wood's light



Wood's test on urine in case of porphyria

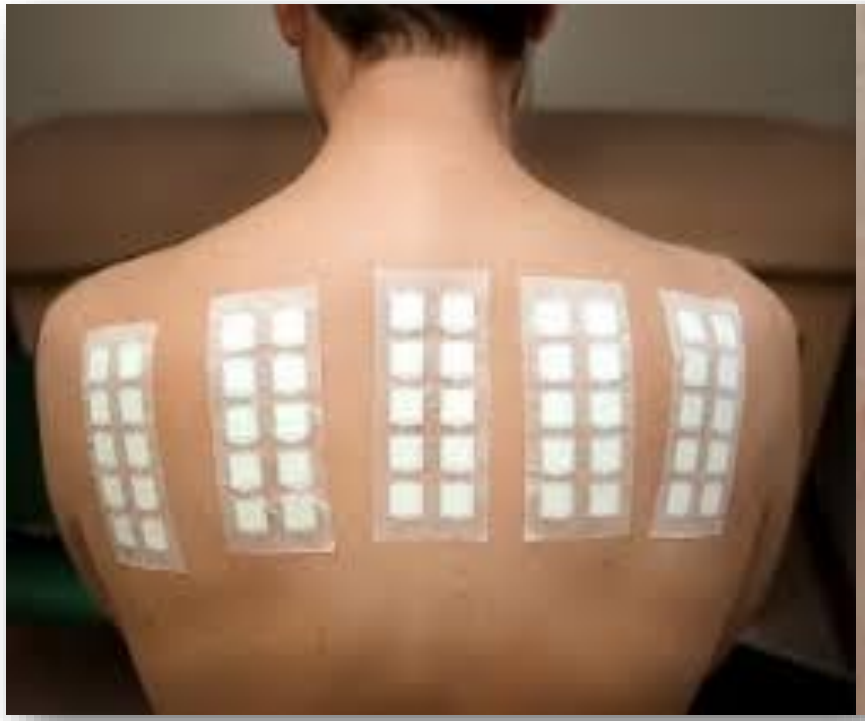
Wood's light

- Name of this devise
  - Wood's light
- Mention 4 indication
  1. Tinea capitis
  2. Tinea versicolor
  3. Vitiligo
  4. Erythrasma

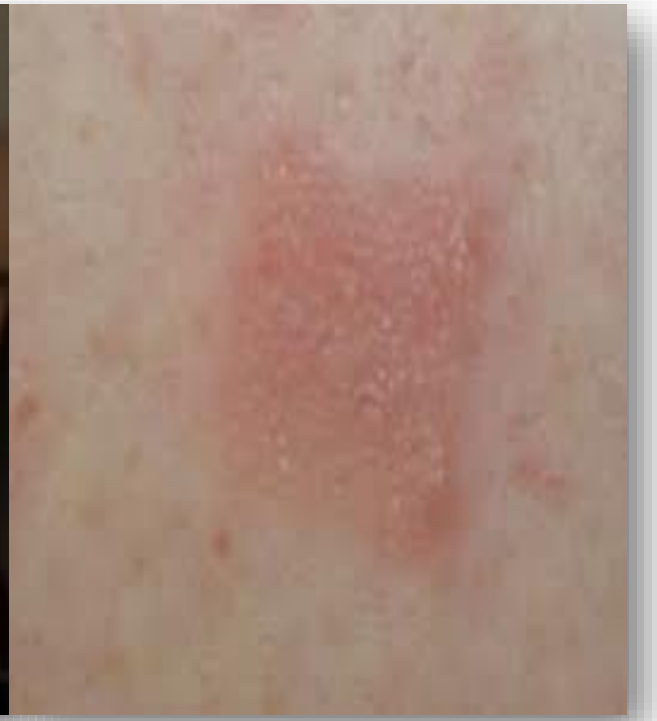
Patch test

سنوات (1) ❖Used for allergic contact dermatitis

❖Wait 24 h (type 4 hypersensitivity) but if reactions occurred before remove it



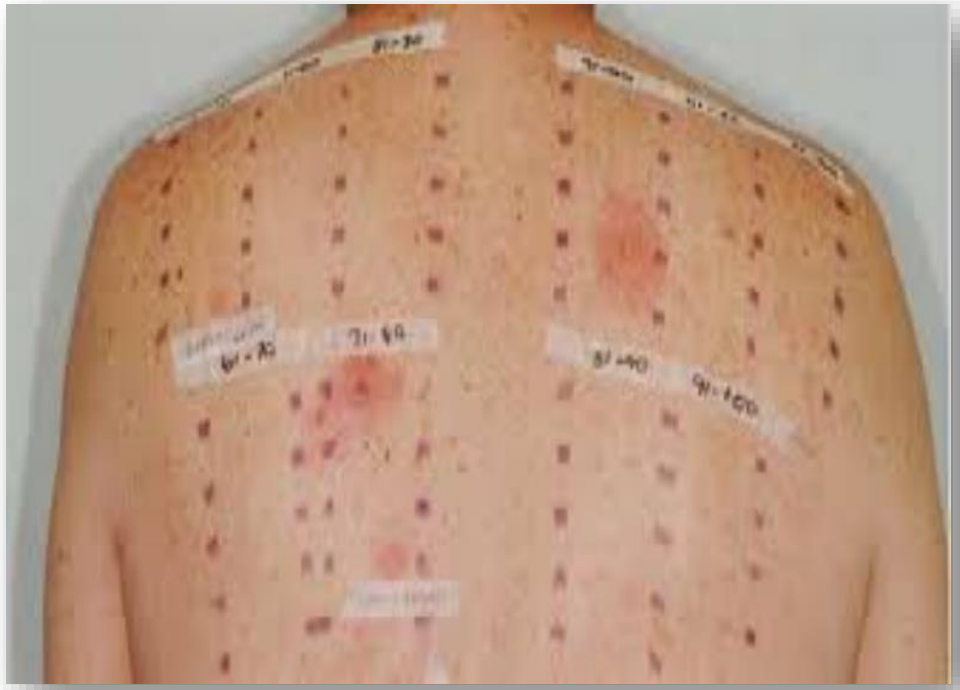
Left column contains a suspected material that the patient might be allergic to, Right column contains control that facilitates the penetration of the allergen to the skin.

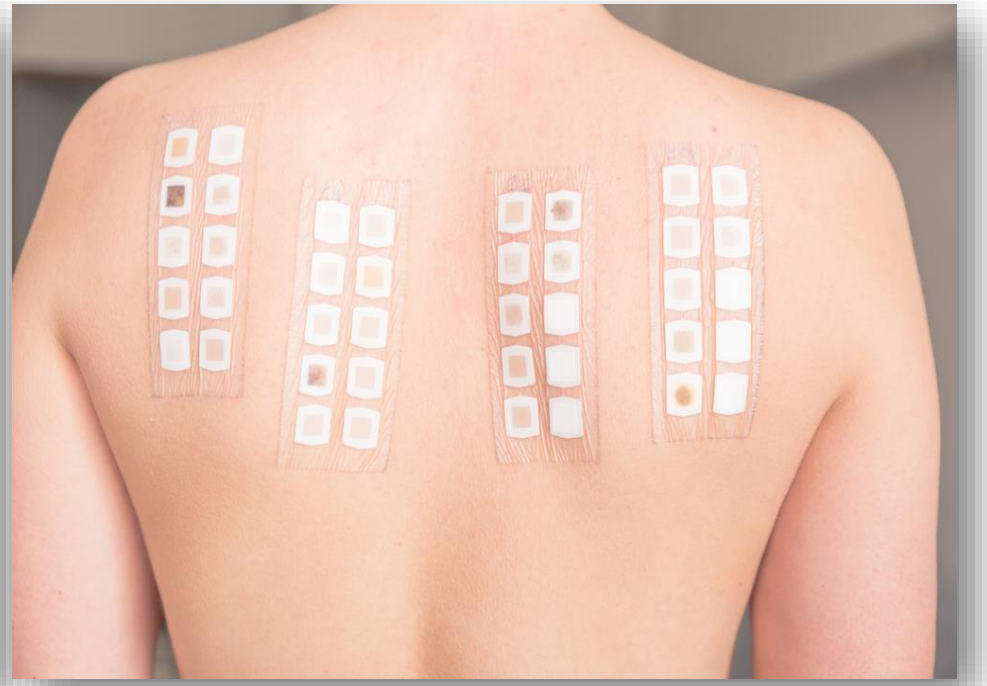


Positive patch test result : vesiculation, erythema and edema when the allergen is applied.

Define Patch test

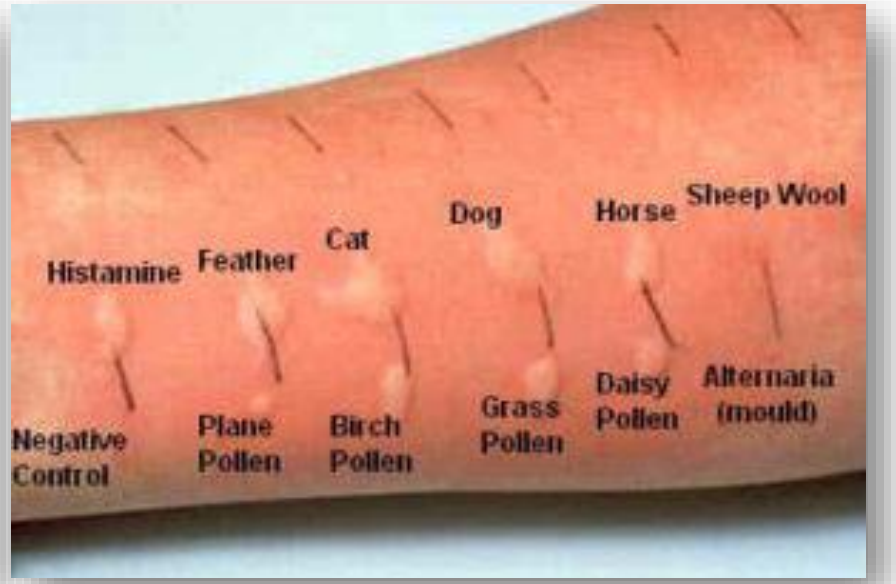
- **Patch test** is a diagnostic test to detect the allergic substance that cause the allergic contact dermatitis. Its avoidance cures the disease, and this is important in occupation related skin reactions
- **Photo patch test** is the same, but it is used for photo allergic dermatitis and the tested area needs exposure to ultraviolet light (sun light).





Skin prick test

- A test for a type I hypersensitivity reaction. Tiny amounts of various allergens are applied to the skin. A lancet is then used to prick the surface of the skin, allowing allergens to penetrate the tissue. A wheal (typically within 15–20 minutes) equal to or larger than a histamine control (or greater than 3 mm) indicates a positive reaction to that allergen.
- Used for respiratory diseases, urticaria, and atopies (atopic dermatitis, allergic rhinitis, asthma)



- What is this test ?
  - Skin prick test

سنوات (1)

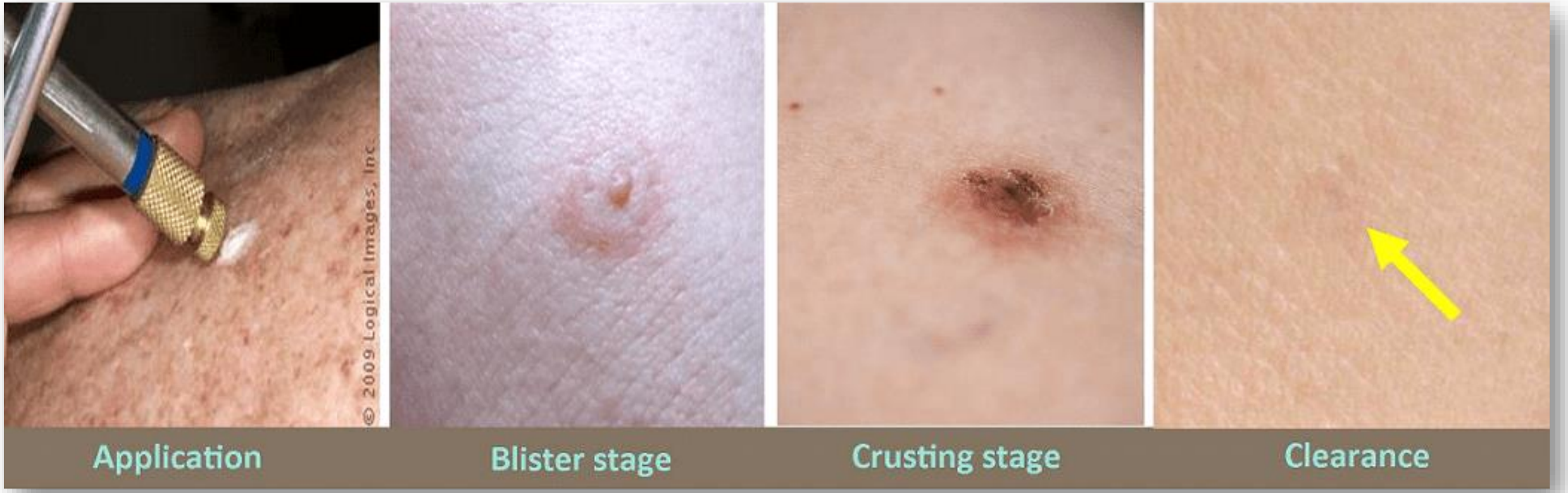
Dermoscope

Dermoscopy: A technique wherein an instrument called a dermatoscope is used to visualize and magnify skin structures in the epidermis, dermoepidermal junction, and upper dermis, e.g., to diagnose skin lesions and triage skin cancers.

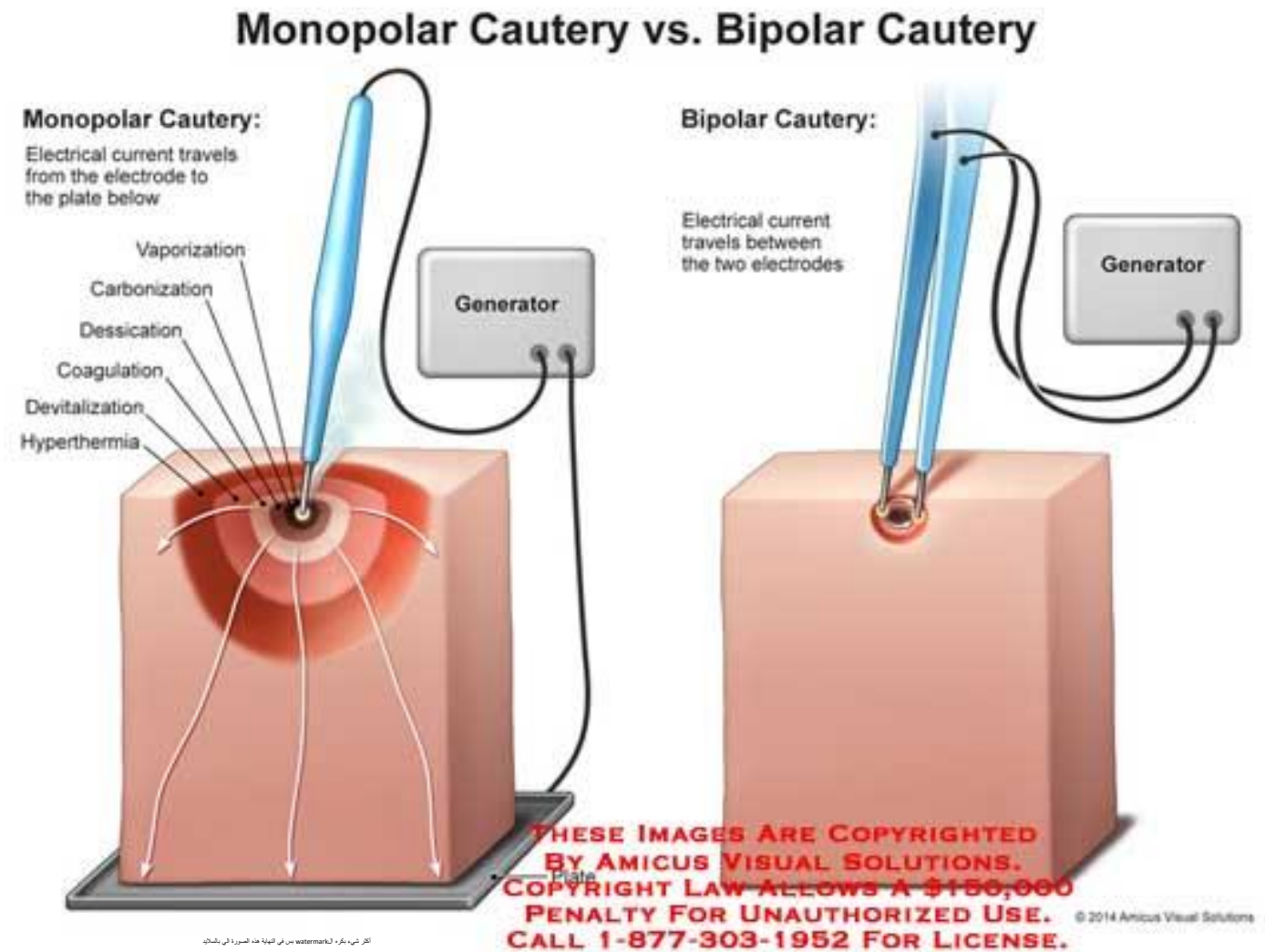
Cryotherapy

- **Name of instrument**
  - Medical cryotherapy gun
- **Name of the used gas**
  - Liquid nitrogen of -196 c
- **Indications**
  - Warts
  - Molluscum contagiosum
  - Orf
  - Callus
  - Actinic keratosis
  - Skin cancers

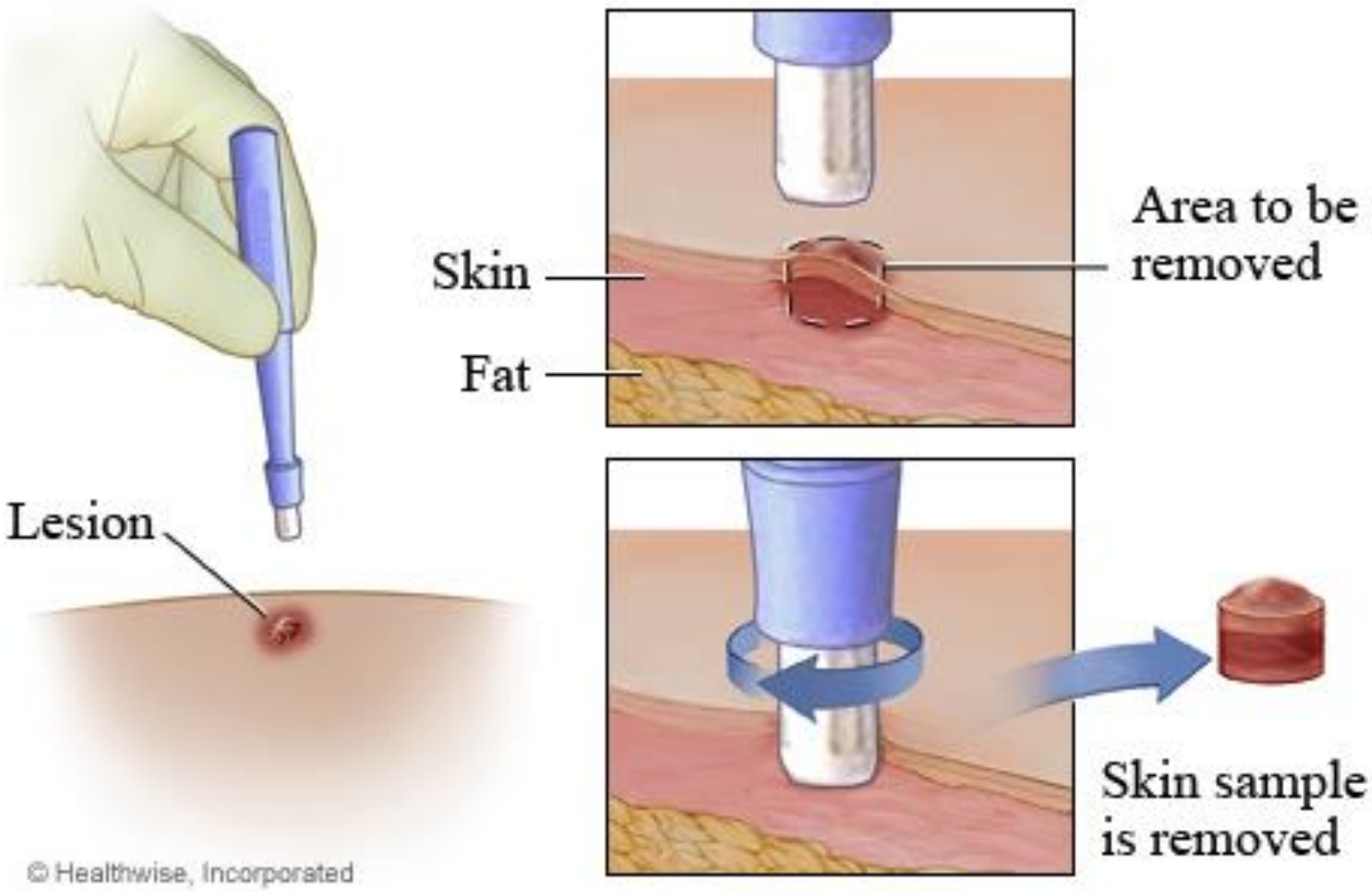
Treatment stages if cryotherapy



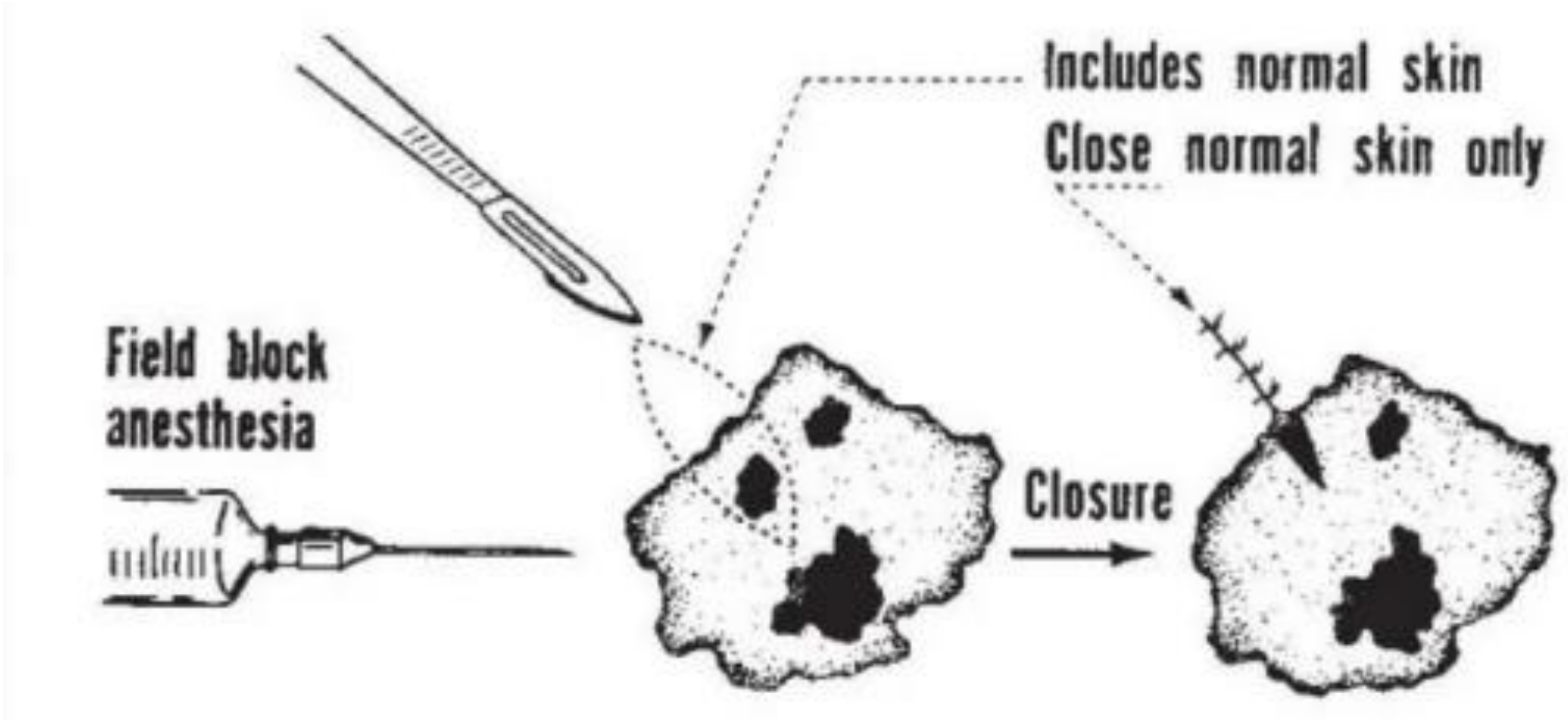
Cautery



Punch biopsy

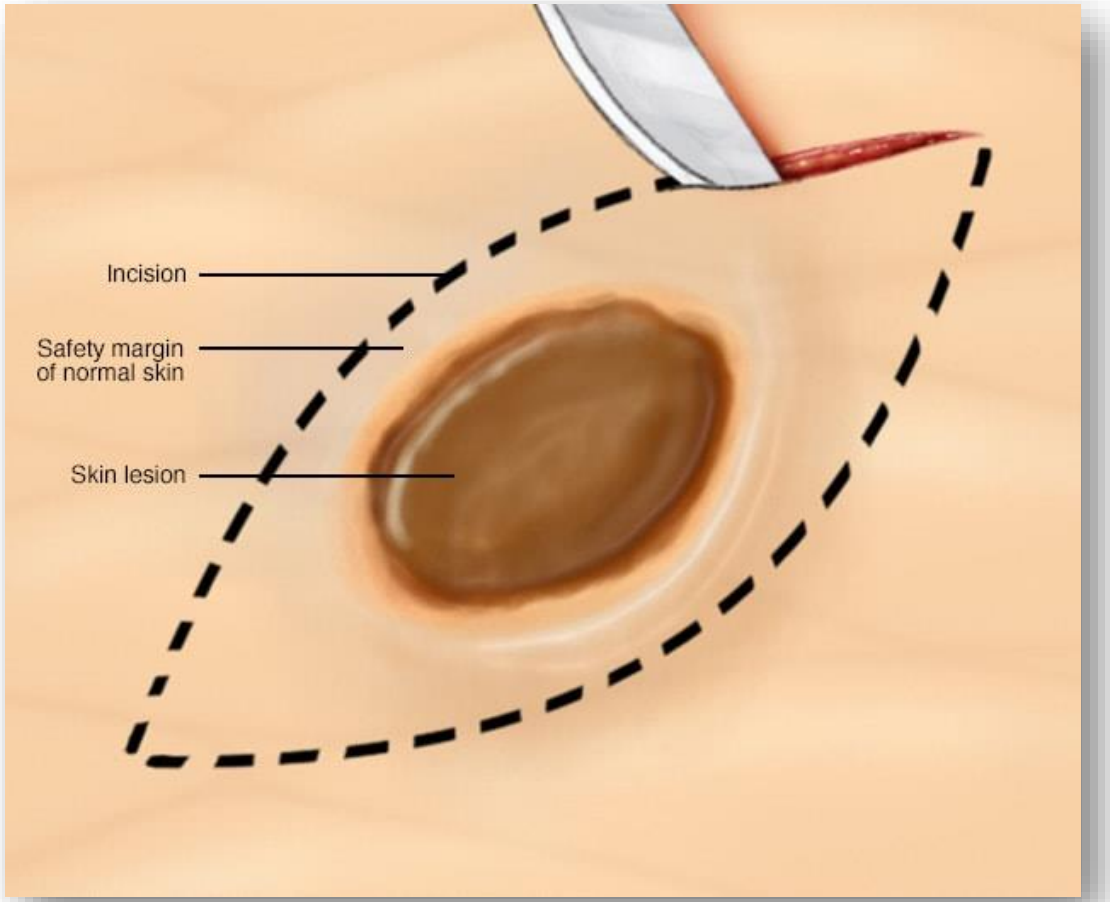


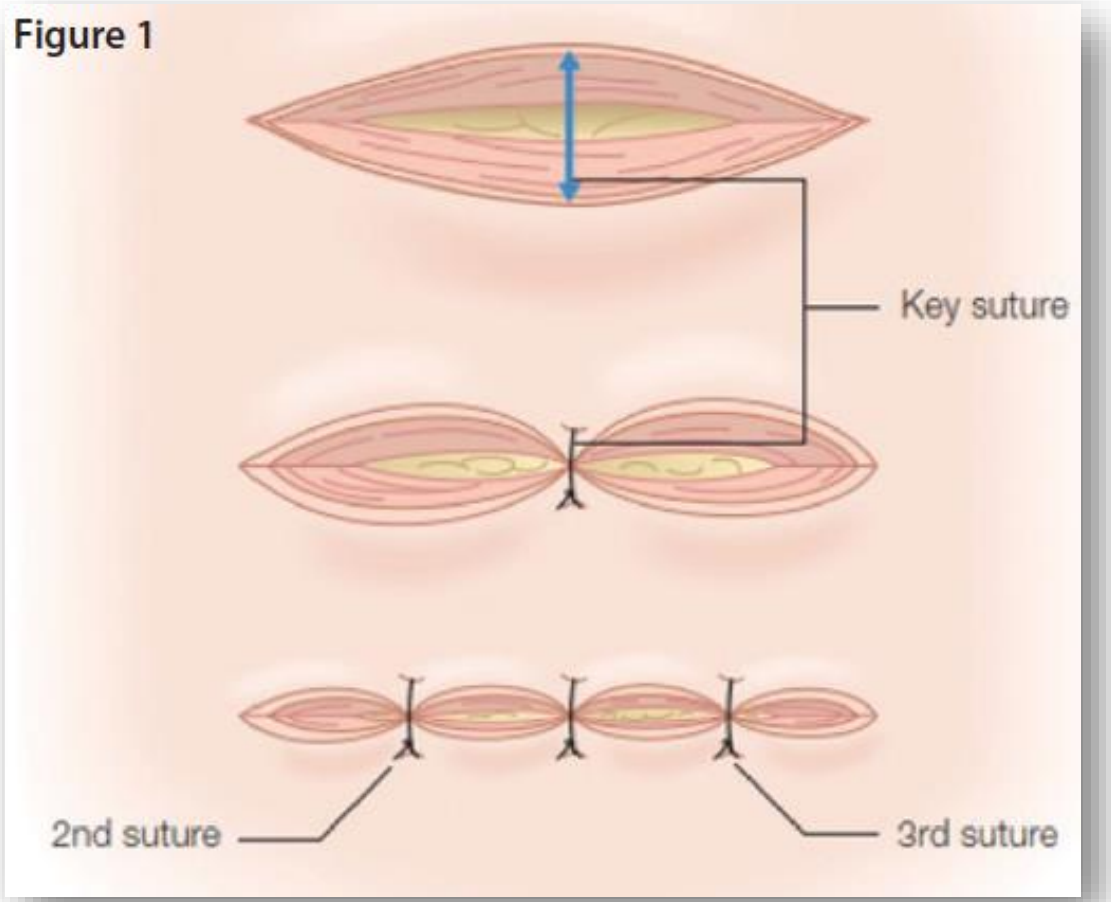
Incisional biopsy



Elliptical biopsy (مش محطوط عليه صورة بالملف)

It reaches three zones





Overview of treatment

شرح

Topical medications

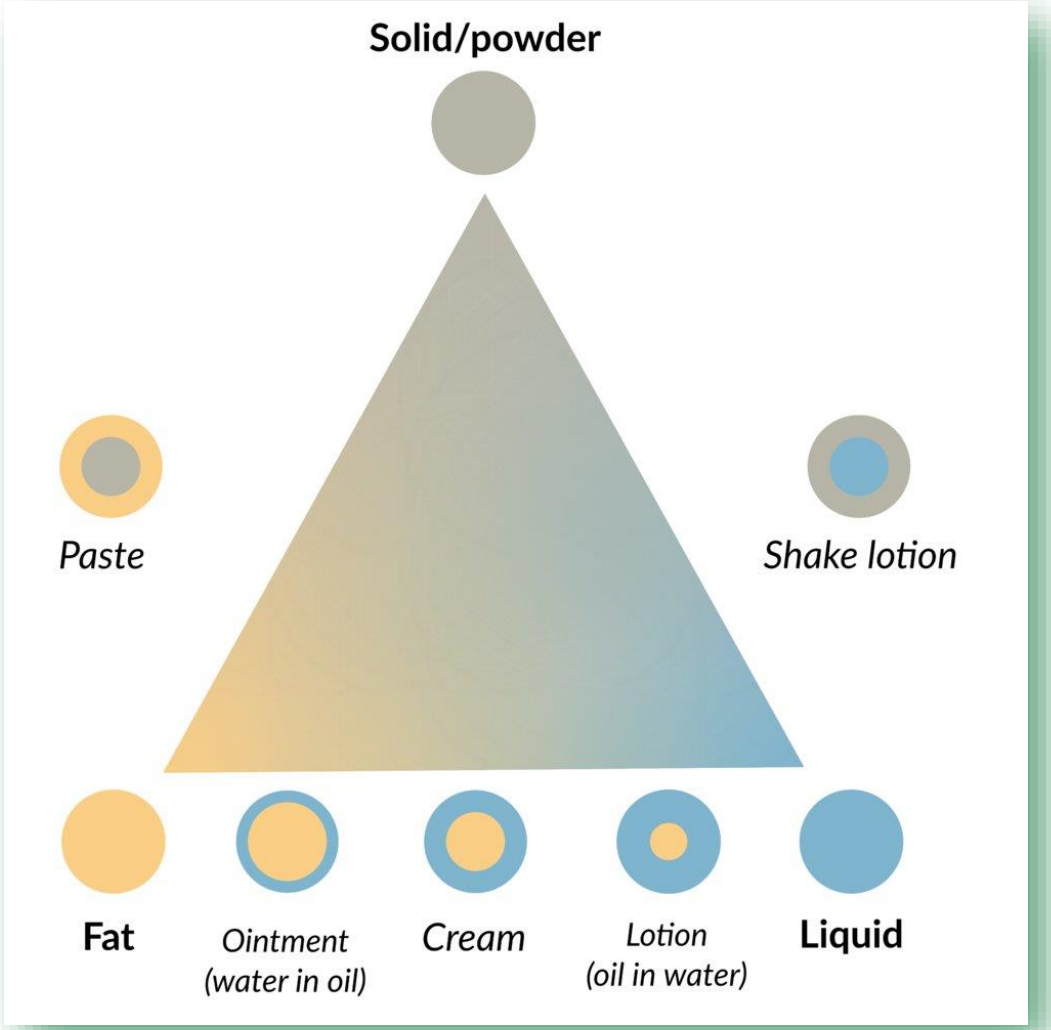
- First choice of treatment for most conditions; often preferred for treating dermatological conditions because they cause fewer systemic side effects
- **Topical steroids**:
  - Most common side effects: Skin atrophy, Steroid acne
  - Mention 2 skin diseases that topical steroid aggravates them (contraindicated)

سنوات (1)

1. Dermatophytosis as with Tinea incognita
2. Acne
3. Hypertrichosis

Types of topical preparations

- **Creams**:
  - Best for weeping eruptions
- **Ointments**:
  - Best for dry, lichenified skin
- **Lotions, foams, and gels**:
  - Best on hairy areas (e.g., scalp)



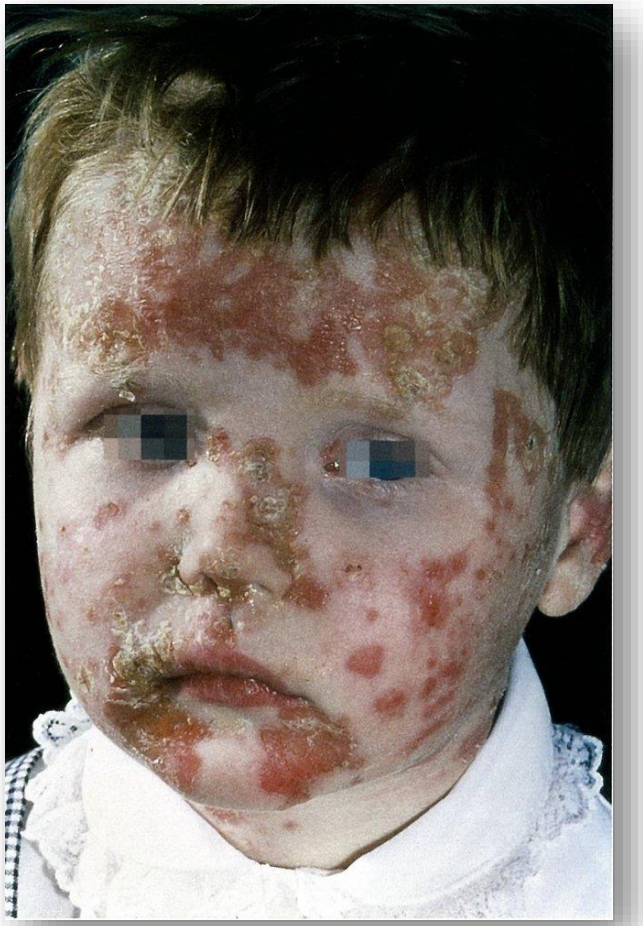

Bacterial skin infections

Impetigo

القوباء أو الحصف

Impetigo

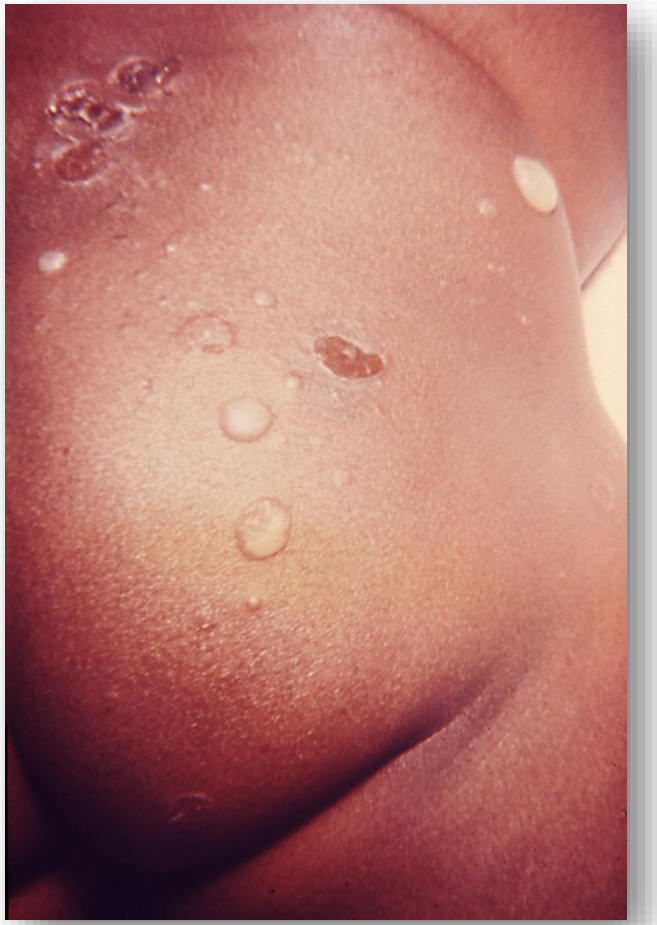
- **Types**: Non-bullous and bullous impetigo
- **Mention the causative agent of**:
  - Bullous impetigo: *S.aureus*
  - Non-bullous impetigo: *S.aureus* and *S.pyogens*
  - Ecthyma: is usually duo to *S.pyogens*, but *S.aureus* co-infection with may occur
- **What are the characteristics of impetigo lesions ?**
  - Pustules and honey-colored crusted erosions.
- **Define**
  - **Impetiginisation**: a superficial secondary infection of a wound or other skin condition
  - **Ecthyma**: An ulcerated impetigo



Non-bullous impetigo

Impetigo – Pathogenesis

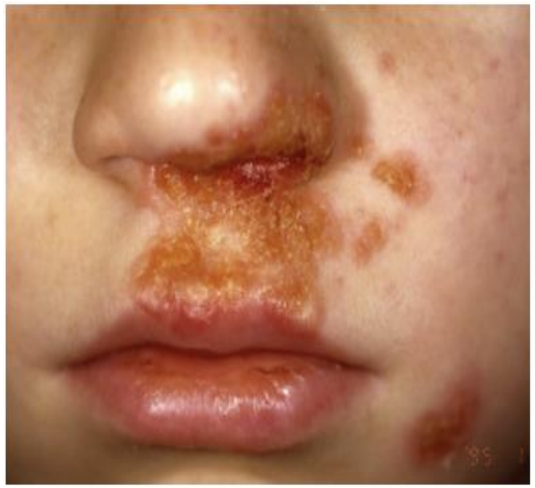
- **Non-bullous impetigo**: *S.aureus* and *S.pyogens* invade a site of minor trauma where exposed proteins allow the bacteria to adhere
- **Bullous impetigo**: is due to *staphylococcal exfoliative toxins* (*exfoliatin A-D*), which target desmoglein 1 (desmosomal adhesion glycoprotein) and cleave off the superficial epidermis through the granular layer. No trauma is required, as the bacteria can infect intact skin.



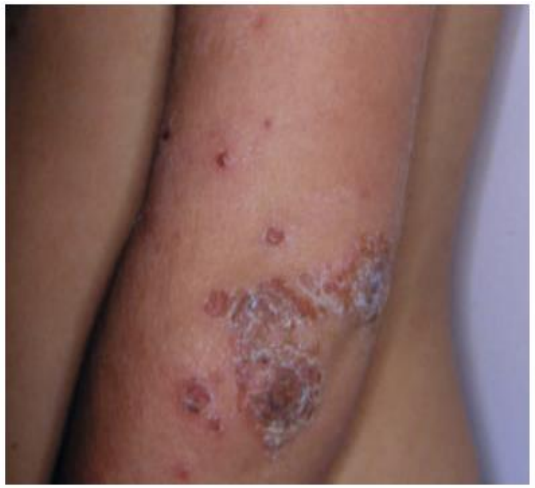
Bullous impetigo

Impetigo

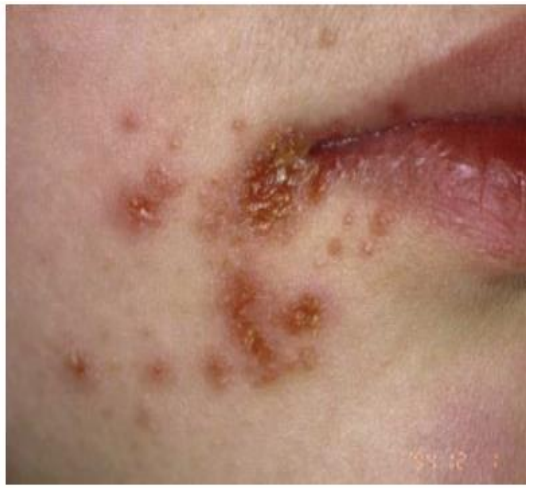
- **Impetigo is most common in which demographic ?**
  - In children (especially boys)
- **What are the factors that predispose to impetigo ?**
  - Loss of skin barrier (Atopic eczema, Scabies, Skin trauma)
- **In which area of the body impetigo most commonly occur ?**
  - Face and hands



Impetigo



Impetigo



Impetigo

Impetigo – Disease course

- **Non-bullous impetigo**:
  - Starts as a pink macule → Vesicle or pustule (1ry lesion) → crusted erosions (2ry lesion)
  - Untreated impetigo usually resolves within 2 to 4 weeks without scarring.
- **Ecthyma**:
  - Starts as non-bullous impetigo → punched-out necrotic ulcer that heals slowly, leaving a scar
- **Bullous impetigo**:
  - Small vesicles → flaccid transparent bullae (1ry lesion)
  - It heals without scarring

Impetigo

❖Mention the possible complications of impetigo

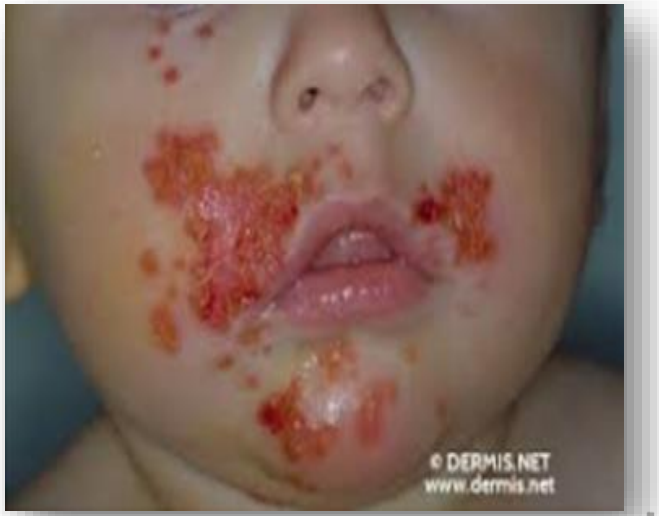
1. Soft tissue infection (cellulitis & lymphangitis)
2. Staphylococcal scalded skin syndrome (SSSS); in infants under 6 years or adults with renal insufficiency
3. Toxic shock syndrome (*S.aureus*) & Toxic shock like syndrome (*S.pyogens*)
4. Post-streptococcal glomerulonephritis (*S.pyogens*)
5. Rheumatic fever (*S.pyogens*); only if the bacteria also infect the throat

❖How is impetigo diagnosed ?

- Clinically

❖Management:

1. Cleanse the wound
2. Apply antiseptic 2-3 times daily for five days
3. Suitable oral antibiotics + **Topical anti-biotics**

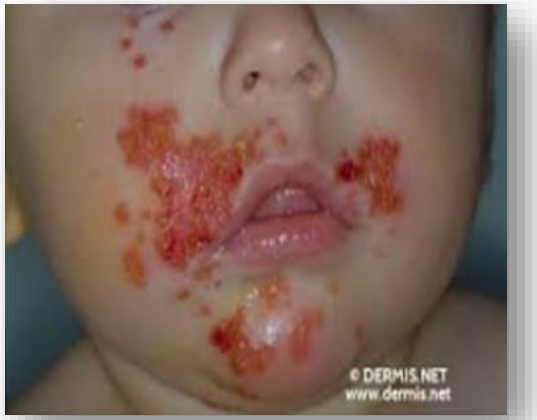


شرح

Impetigo

❖ **When are oral anti-biotics recommended in treating impetigo ?**

- Symptoms are significant or severe (fever, malaise)
- **There are more than three lesions**
- There is a high risk of complications
- The infection is not resolving or is unlikely to resolve



سنوات (1)

❖ **Mention one topical treatment for impetigo**

- Topical antibiotic such as bacitracin, mupirocin, retapamulin

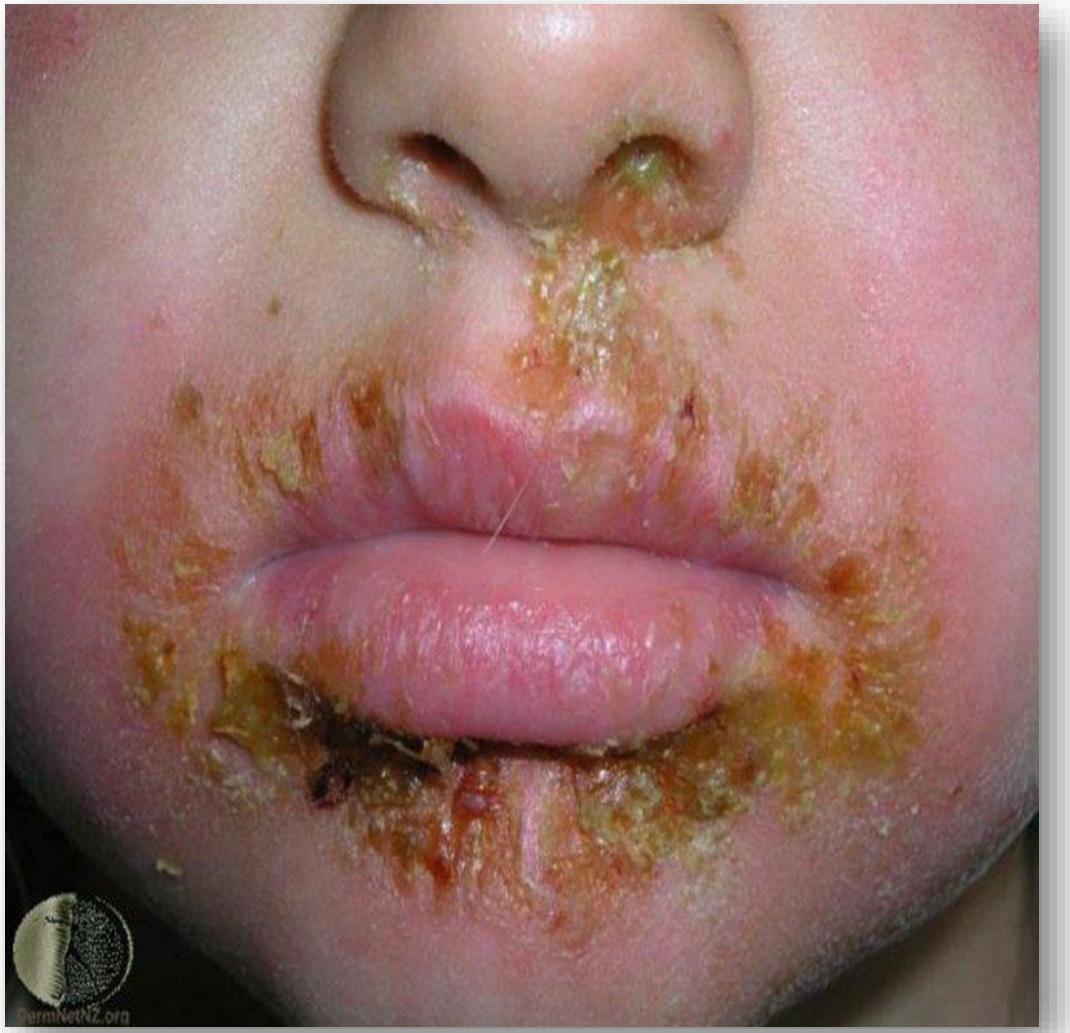
سنوات (1)

❖ **First line treatment if there is more than 5 lesions**

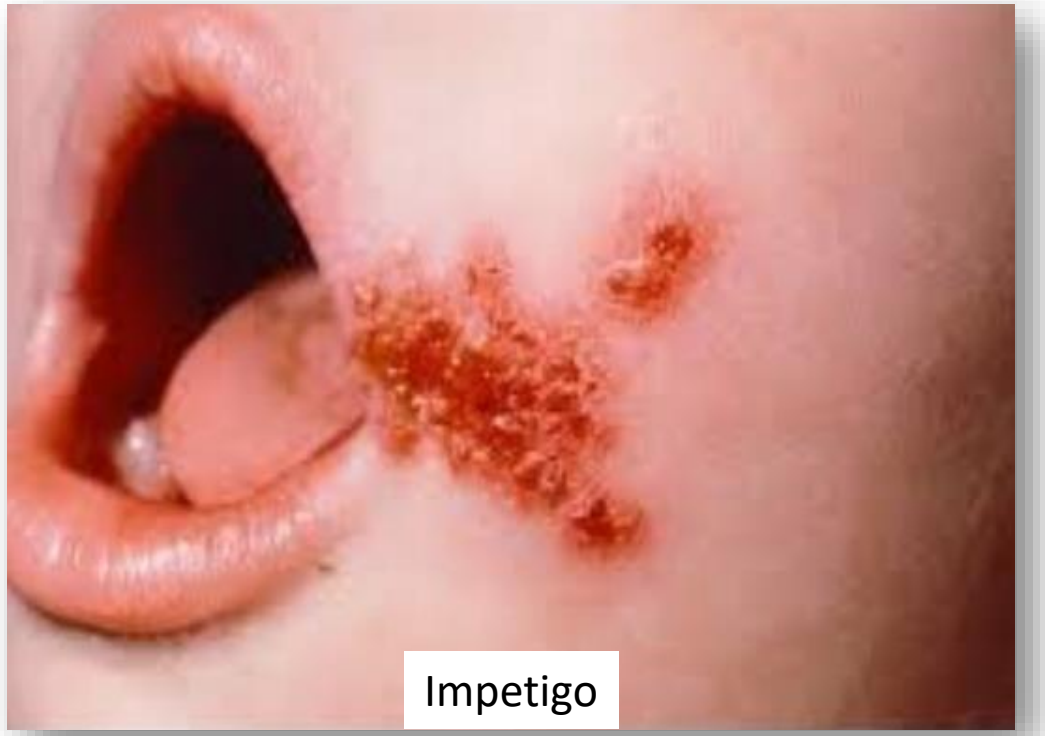
- Oral antibiotics

Impetigo

- **What is your Diagnosis ?**
  - Impetigo
- **What is the management ?**
  1. Cleanse the wound
  2. Apply antiseptic 2-3 times daily for five days
  3. Topical antibiotics
  4. Consider oral antibiotics only if there is warning signs such as fever or malaise



Impetigo



Impetigo

Severe impetigo

Cellulitis & Erysipelas

Low-yield – (ركزوا على الأحمر)

شرح

Cellulitis & Erysipelas

- **Define**:
  - **Erysipelas**: superficial skin infection involving the upper dermis
  - **Cellulitis**: local infection of the deep dermis and subcutaneous tissue
- **Mention the causative agent of**:
  - Cellulitis: *S.pyogens* (67%), *S.aureus* (33%)
- **What is the most common site affected by cellulitis**
  - Lower limbs and face
- **Management**:
  - Oral antibiotic, Analgesia, fluid intake, Management of underlying co-existing skin conditions like eczema or tinea pedis

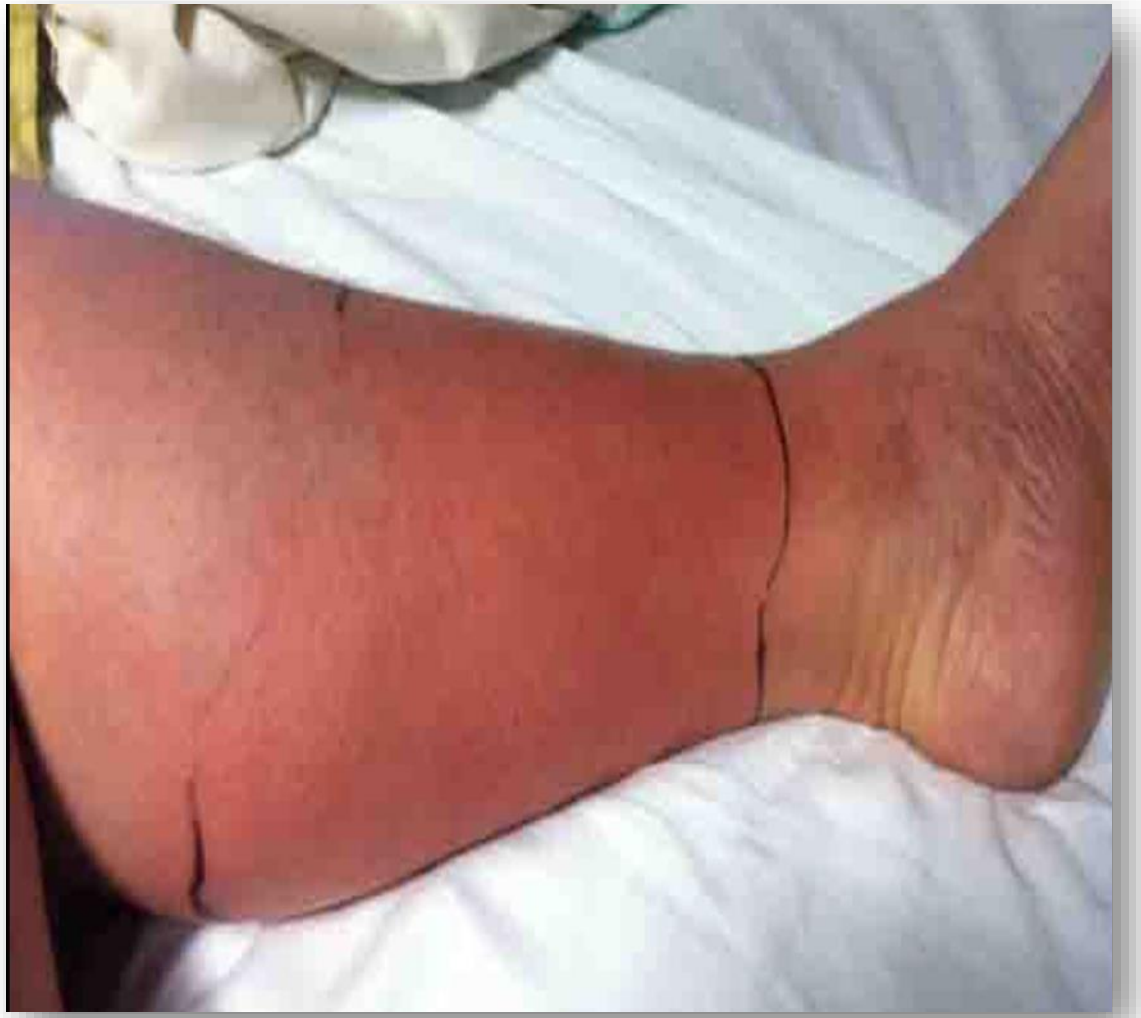
سنوات (1)

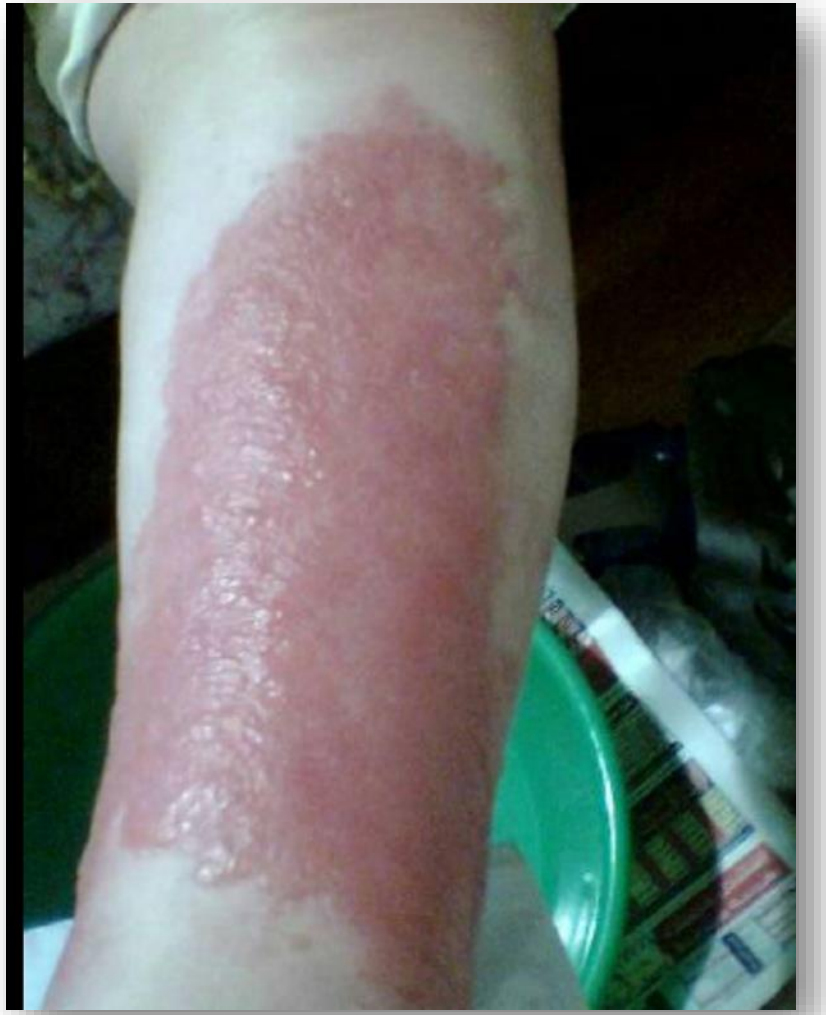
- **Mention one topical treatment for Cellulitis**
  - Antibiotic such as bacitracin

Cellulitis & Erysipelas

Cellulitis	Erysipelas
Suppurative inflammation of lower dermis and SC	Suppurative inflammation of upper dermis
Deeper infection (in subcutaneous)	More superficial (between subcutaneous and dermis)
Poorly demarcated	Well demarcated
Most commonly due to *S.pyogens* Rarely caused by *S.aureus*	

Cellulitis & Erysipelas

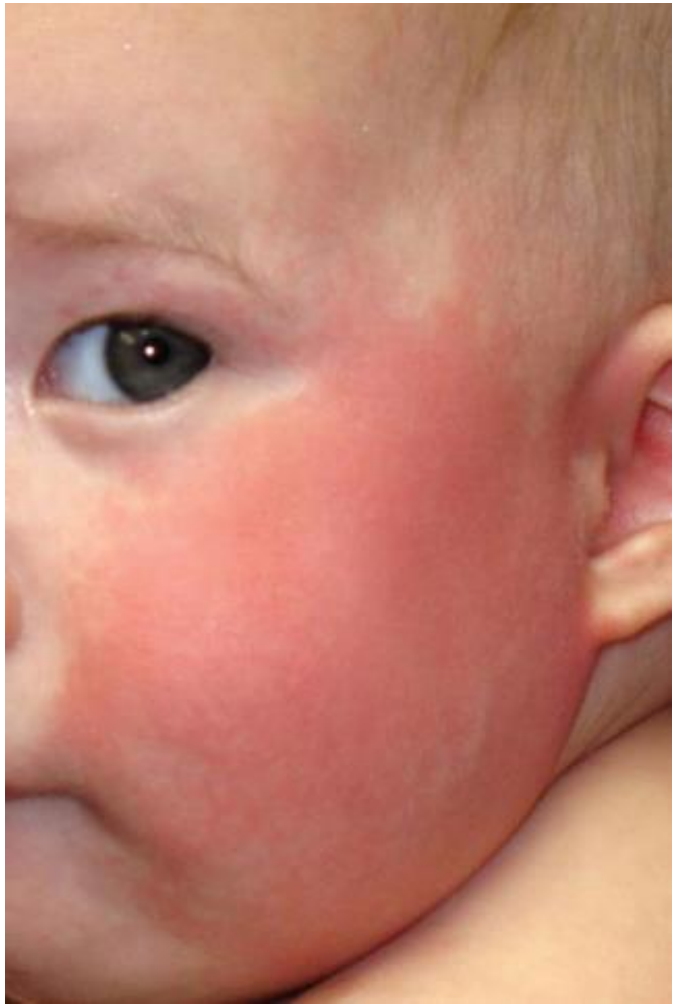


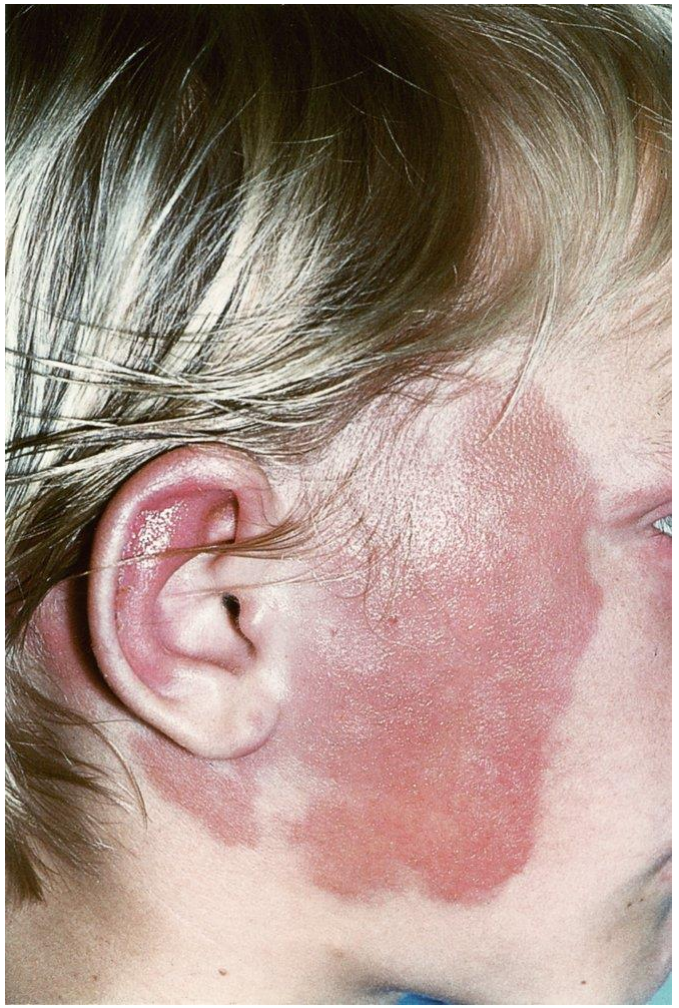


Cellulitis & Erysipelas

Milian's ear sign:

- Erysipelas can spread to the pinna, whereas cellulitis cannot, because the pinna has no deeper dermis and subcutaneous tissue
- Erysipelas involve the upper dermis, while cellulitis involve the deeper dermis and subcutaneous fat





LOW YIELD

شرح

What are the factors that predispose to cellulitis ?

1. Previous episode(s) of cellulitis.
2. Fissuring of toes or heels, e.g., due to tinea pedia, cracked heels.
3. Current or prior injury, e.g., trauma, surgical wounds,
4. Venous disease e.g., lymphedema, gravitational eczema.
5. Immunodeficiency.
6. Immune suppressive medications.
7. Diabetes.
8. Chronic kidney disease.
9. Chronic liver disease.
10. Obesity.
11. Pregnancy.

Folliculitis

ركزوا على الأحمر

شرح

Folliculitis

سنوات (1)

❖ **Describe folliculitis lesion**

- Tender red spot, often with a surface pustule.

سنوات (1)

❖ **What is the primary lesion of folliculitis**

- Pustule

❖ **The most common type of folliculitis**

- Bacterial folliculitis

❖ **The causative agent of bacterial folliculitis**

سنوات (4)

- Most common due to *S.aureus*
- Less often due to *coagulase-negative staphylococci* and *gram-negative organisms* including *anaerobes*
- Spa pool folliculitis is caused by *Pseudomonas*

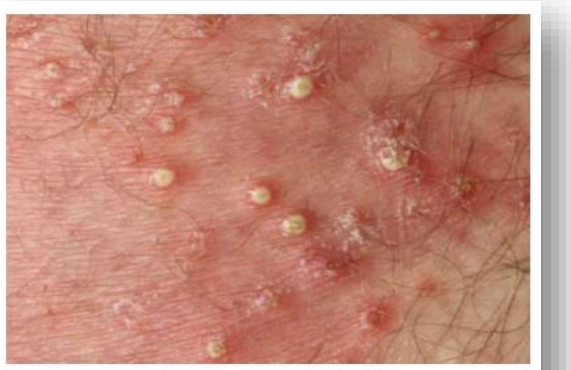
❖ **Folliculitis is most common in which demographic ?**

- Adolescents and young adult males most often infected

Folliculitis

❖Superficial folliculitis

- Superficial staphylococcal folliculitis presents with one or more follicular pustules.
- They may be itchy or mildly sore.
- Superficial folliculitis heals without scarring.



Superficial bacterial folliculitis

❖Furunculosis/boils

- Presents as one or more painful, hot, firm or fluctuant, red nodules or walled-off abscesses (collections of pus).

❖A carbuncle

- is the name used when a focus of infection involves several follicles and has multiple draining sinuses, **usually diabetic patients.**
- Causative agent: *S.aureus*
- Recovery leaves a scar.

سنوات (1)

Folliculitis

- **Gram-negative folliculitis**
  - Develops in individuals using **long term antibiotics (Doxycycline) for acne**
- **Hot tub folliculitis**
  - It settles without treatment within about 10 days without scarring
- **Pseudofollicultis**
  - Hair re-entry after shaving
- Bacterial folliculitis can lead to cellulitis, Erysipelas and lymphangitis; subsequent bacteremia might result in osteomyelitis, septic arthritis or pneumonia
- **How is folliculitis diagnosed ?**
  - Clinically

What are the factors that predispose to folliculitis ?

1. Maceration and occlusion (clothing, dressings, ointments, casts of broken bones)
2. Frequent shaving, waxing or other forms of depilation
3. Friction from tight clothing (Physical folliculitis)
4. Atopic eczema
5. Use of topical steroids
6. Previous long-term use of antibiotics
7. Chronic illness that leads to recurrent furunculosis (see nest slide)

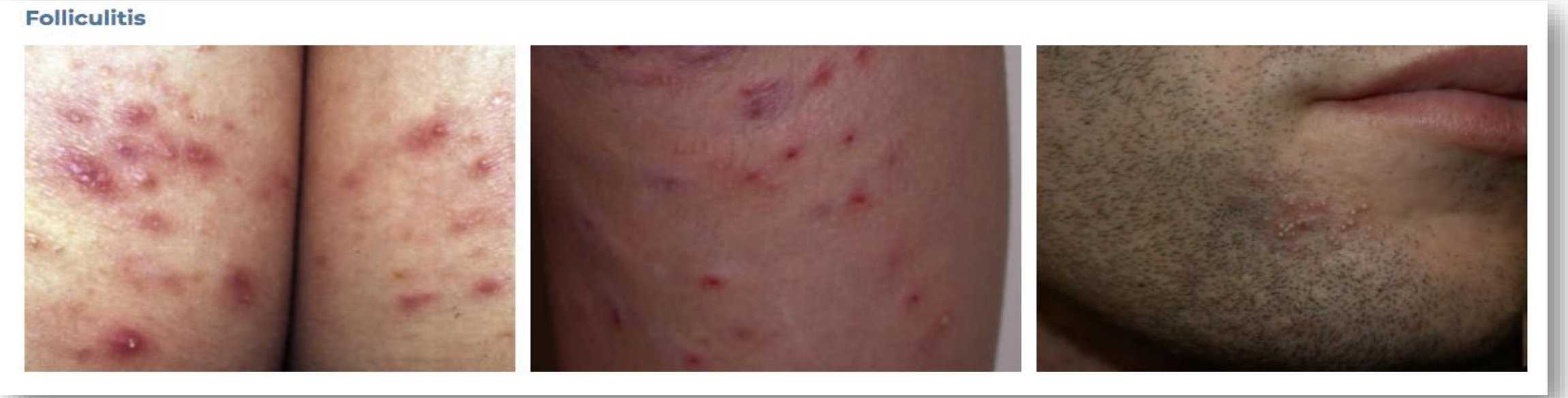
Mention 3 causes for recurrent boils (Furunculosis)

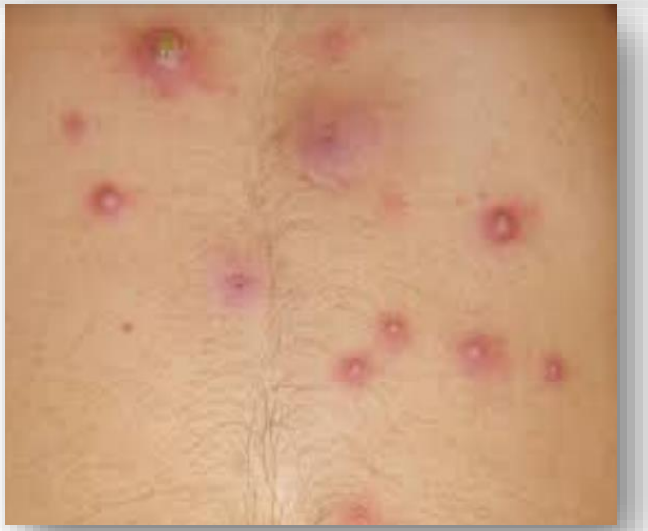
1. Health care worker carrier MRSA
2. Anemia
3. Diabetes
4. Obesity
5. (HIV)/AIDS
6. Cancer

❖Approach: Fasting blood sugar, CBC, Nasal swab

➢**You can think of them as things that weaken the immune system**

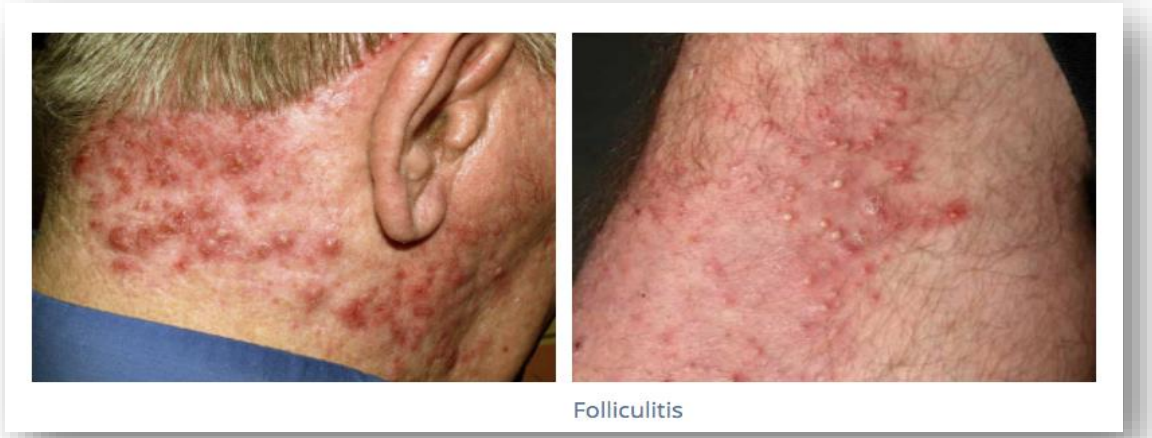
Folliculitis with boil



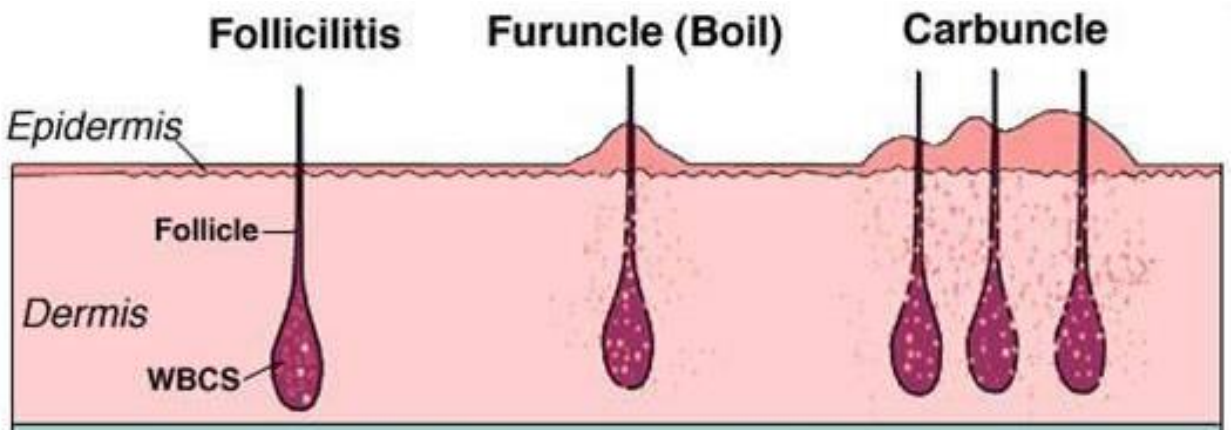


Folliculitis – Treatment

1. Warm compresses to relieve itch and pain.
2. Analgesics and anti-inflammatories to relieve pain
3. Antiseptic cleansers (e.g., hydrogen peroxide, chlorhexidine, triclosan).
4. Incision and drainage of fluctuant lesions.
5. Topical antibiotics such as erythromycin, mupirocin, Fucidic acid.
6. Oral or intravenous antibiotics for more extensive or severe infections.


Folliculitis

Folliculitis – Furuncle – Carbuncle



Folliculitis	Furuncle (Boil)	Carbuncle
1mm perifollicular red papule or pustule	About 1cm tender red papule or fluctuant nodule	Several cm diam red plaque
Areas of sweat & abrasion	Areas of sweat & abrasion	Nape of neck
Rx: Tetracycline or erythromycin 500 mg 2x/day	1. Incise & curettage. 2. Dicloxicillin 250mg 4x/d for 10 days, or Augmentin 500mg 2x/day for 10+ days	1. Incise and curettage or excise 2. Dicloxicillin 250mg 4x/day for 10+ days or rampin 300mg 2x/day for 10+ days (Orange body fluids)

سنوات (1)

Folliculitis

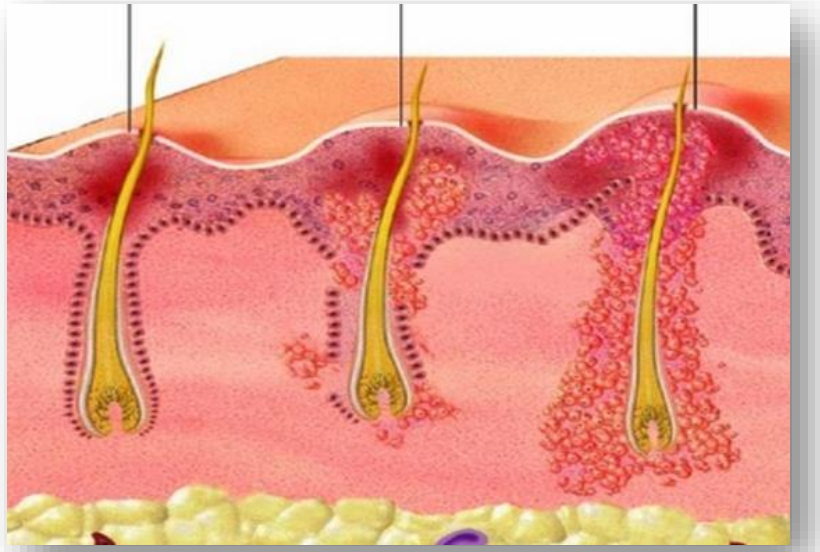
- **Describe**:
  - Tender red spot, often with a surface pustule
- **What is this lesion ?**
  - Folliculitis

سنوات (3)

- **What is the causative agent ?**
  - S.aureus

سنوات (2)

- **Mention 1 line of treatment**:
  - Any of the earlier mentioned managements
  - Ex. Warm compresses to relieve itch and pain
- **Mention the stages of this lesion**
  - folliculitis, carbuncle, furuncle



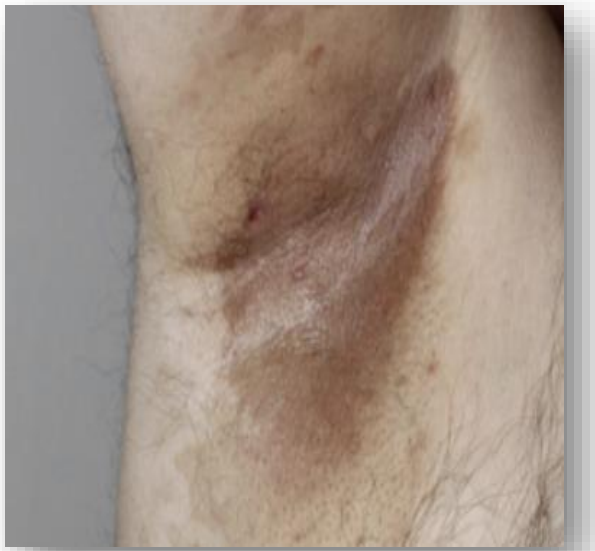
Erythrasma

الوذح

شرح

Erythrasma

❖**Erythrasma** is a common skin condition affecting the skin folds under the arms, in the groin and between the toes.



سنوات (1)

❖**Site of infection**:

- Males: more common in the groin
- Females: more common between the toes

سنوات (1)

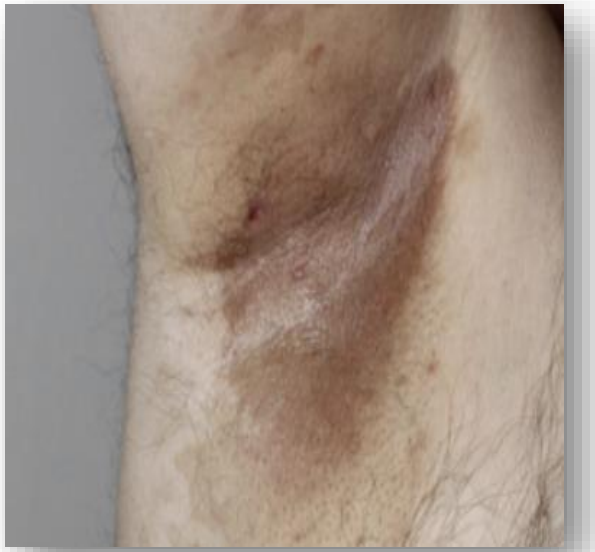
❖**Causative agent**:

- Corynebacterium minutissimum

❖**Lesion description**:

- Erythrasma presents as well-defined pink or brown patches with fine scaling and superficial fissures. Mild itching may be present.

Erythrasma



- Is more prevalent in the following circumstances:
  1. Warm climate
  2. Excessive sweating
  3. Diabetes
  4. Obesity
  5. Poor hygiene
  6. Advanced age
  7. Other immunocompromised states
     - Widespread infections are most often associated with diabetes
- Erythrasma is usually self-limiting. But it can be complicated by
  - Contact dermatitis, Lichenification, post inflammatory hyperpigmentation, and coinfection with other yeast and bacteria

شرح

Erythrasma

سنوات (1)

- **The best diagnostic test for erythrasma**
  - Wood light

سنوات (1)

- **Appearance on woods light**
  - Coral-pink color

- **Differential diagnosis**
  - Erythrasma (Pink on wood's light)
  - Pseudomonas (Green on wood's light)
  - Fungal infection

سنوات (1)

- **Treatment**:
  - antiseptic or topical antibiotic such as clindamycin solution, erythromycin creams
  - Extensive infection can be treated with oral antibiotic and usually responds promptly

What is the diagnosis

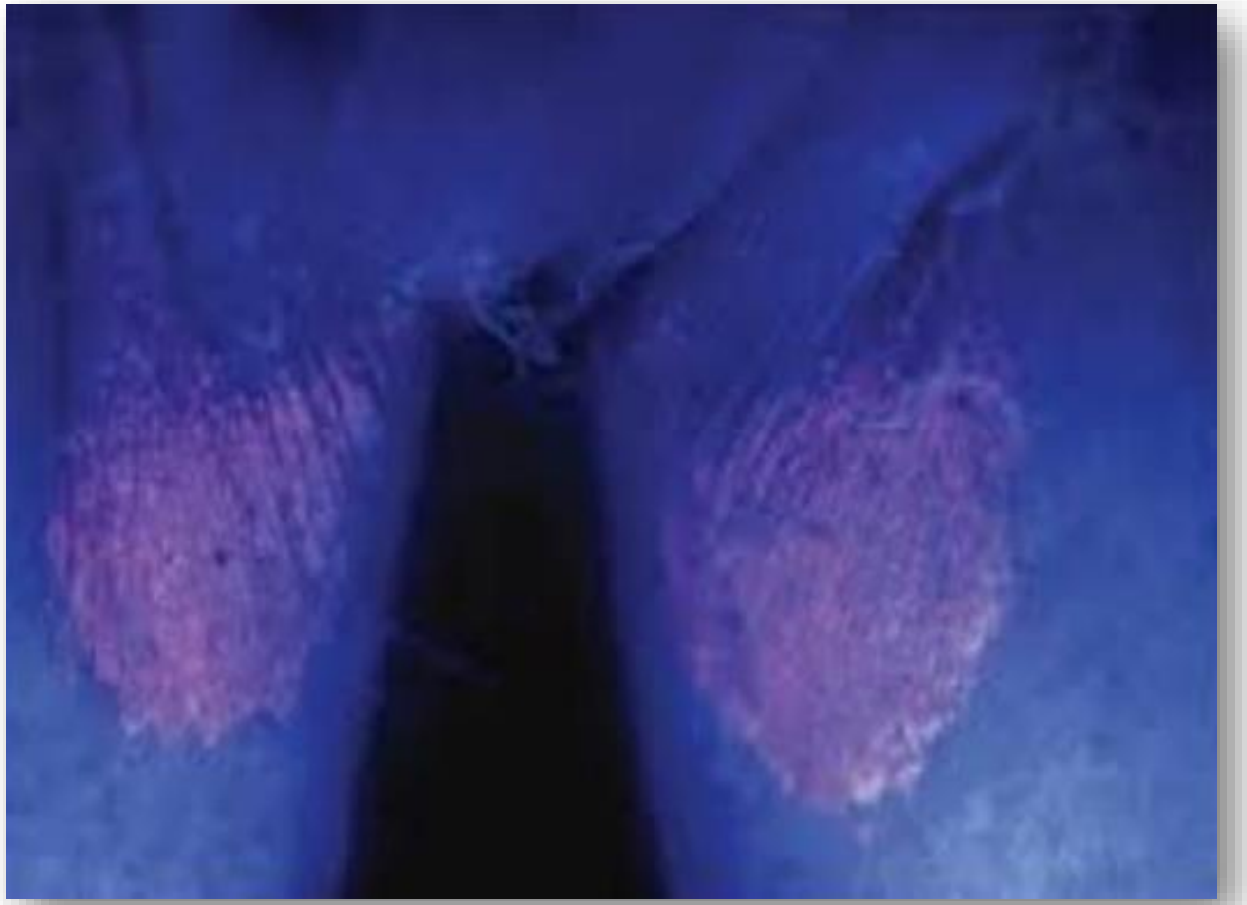
A. vitiligo

B. erythrasma

C. Tenia versicolor

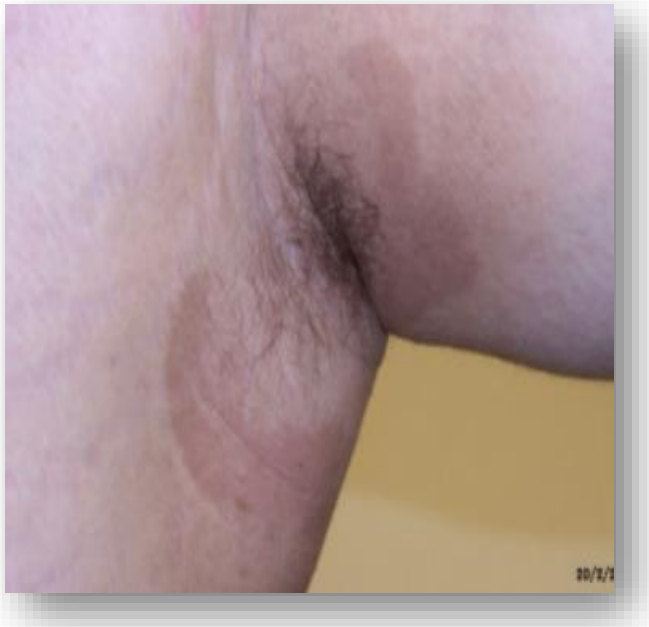
D. Pitted keratolytic

E. Tinea corporis



Erythrasma

- Brawny scaly hyperpigmentation on axilla female positive woods light



Appearance on woods light

- Coral-pink color

Diagnosis

- Erythrasma

Cause

- Corynebacterium minutissimum

One line of treatment

- antiseptic or topical antibiotic (clindamycin solution, erythromycin creams)

Another site

- groin, submamary

Pitted keratolysis

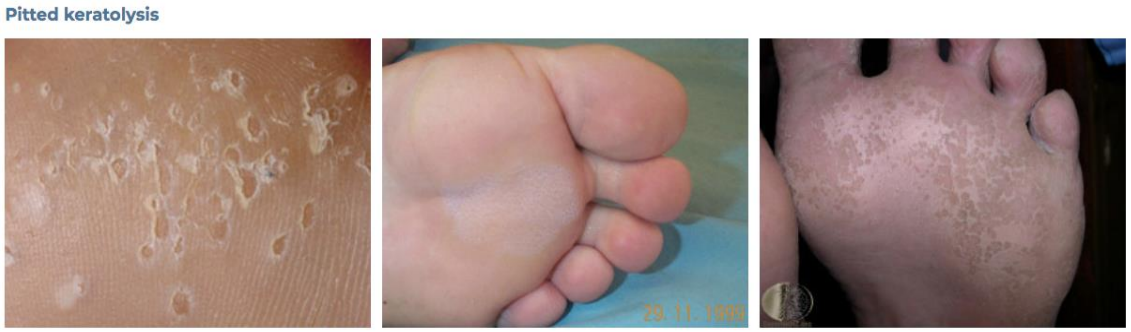
ركزوا على الأحمر

Pitted keratolysis

- **Pitted keratolysis** is a descriptive title for a superficial bacterial skin infection that affects the **soles of the feet, and less often, the palms of the hands**.
- It is one of the causes of **smelly feet**.
  - The bad smell is due to sulfur compounds produced by the bacteria
- **Lesion description**:
  - whitish skin and clusters of punched-out pits
  - The pitting is due to destruction of the horny cells (stratum corneum) by protease enzymes produced by the bacteria.
- **Causative agents**:
  - ***Corynebacteria***, *Dermatophilus congolensis, Kytococcus sedentarius, actinomyces and streptomyces*
- Much more common in males than in females

Pitted keratolysis

- Factors that lead to the development of pitted keratolysis include: (Hot, humid weather, occlusive footwear, excessive sweating of hands and feet, thickened skin of palms and soles, diabetes, advanced age)
- Pitted keratolysis is usually diagnosed clinically.
- **Appearance on woods light**
  - Coral- red color fluorescence in some cases
- **Treatment**
  - Topical antiseptics and antibiotics (Erythromycin + Clindamycin)



سنوات (1)

Pitted keratolysis

سنوات (3)

❖**Diagnosis**: Pitted keratolysis

❖**Describe what you see:**

- Whitish skin and clusters of punched-out pits on the sole of the foot.

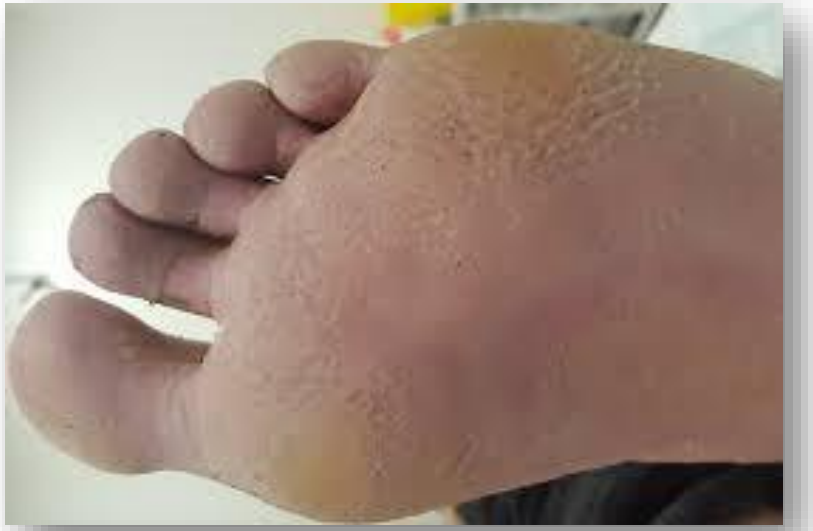
سنوات (2)

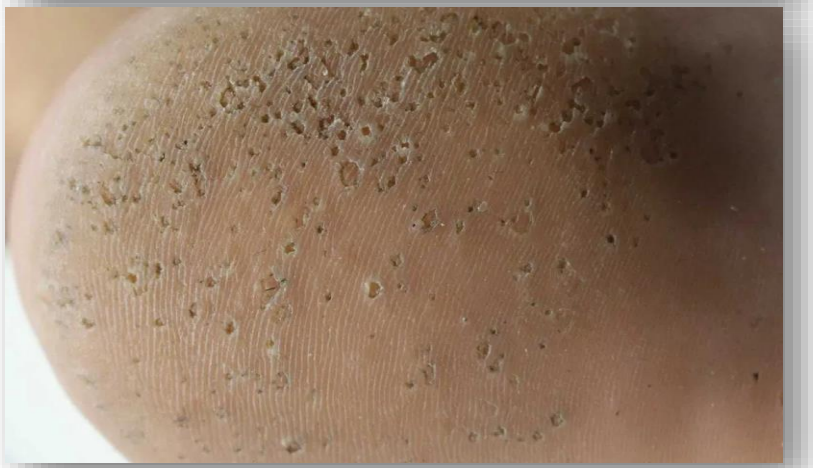
❖**What medication can be used?**

- Topical antibiotics (erythromycin, clindamycin)

❖**What is the causative organism? (Mention one only)**

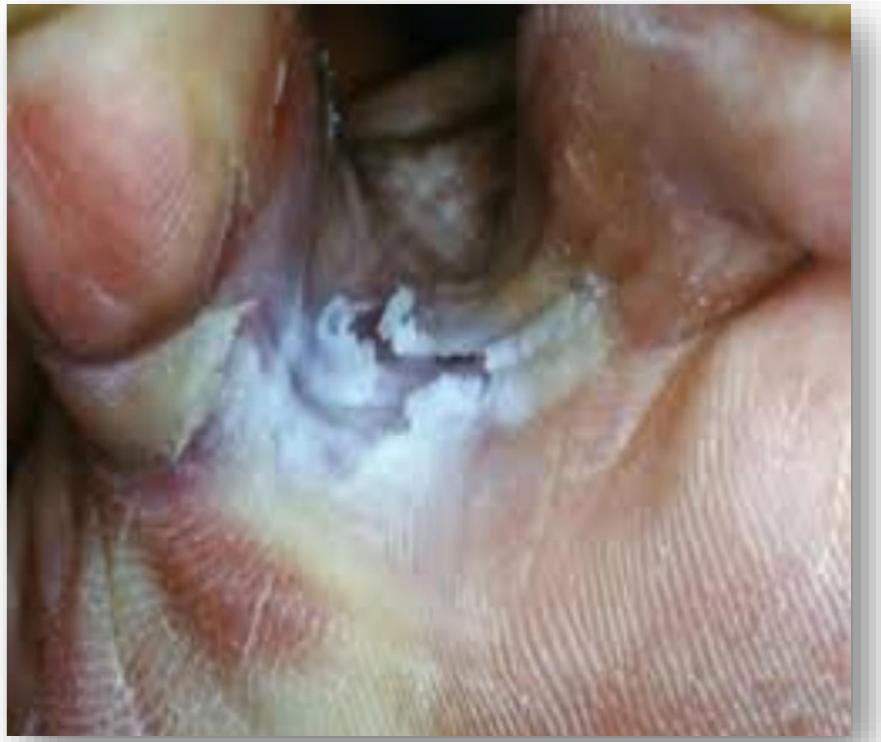
- Corynebacteria, *Dermatophilus congolensis*, *Kytococcus sedentarius*, actinomyces and streptomyces





This lesion’s color on woods lump is red

- **What is your diagnosis ?**
  - Pitted keratolysis or erythrasma
- **Mention other Ddx**
  - Tenia pedis
  - Pseudomonas
  - Psoriasis
  - Eczema
- **What is the treatment ?**
  - Topical antibiotics

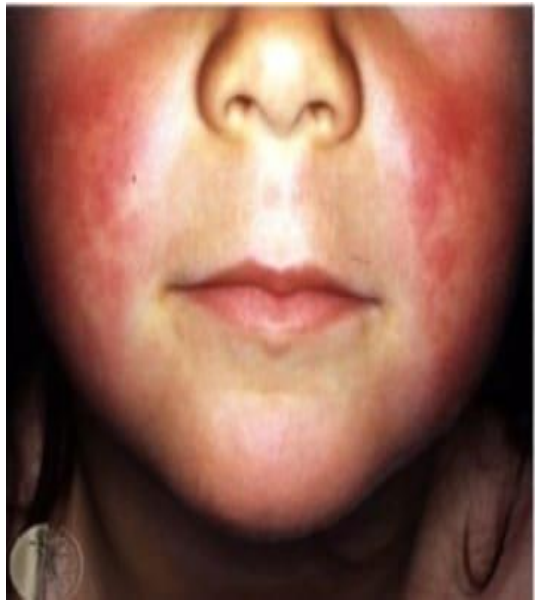


Scarlet fever (حمى قرمزية)

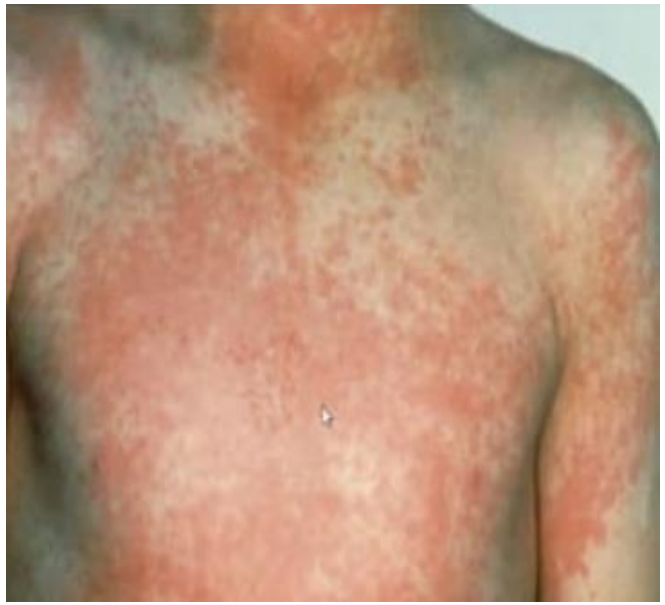
Source: Dermatology cases (1)

Scarlet ever

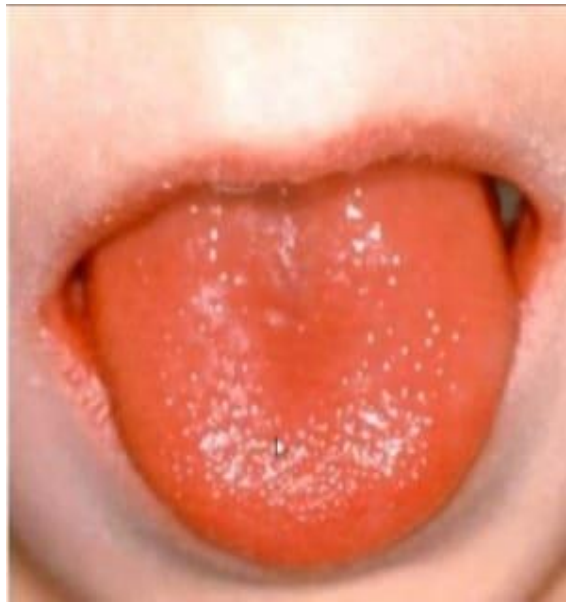
Skin rash on the face and trunk with fever in a child, caused by strep throat



Erythema on cheeks with perioral pallor

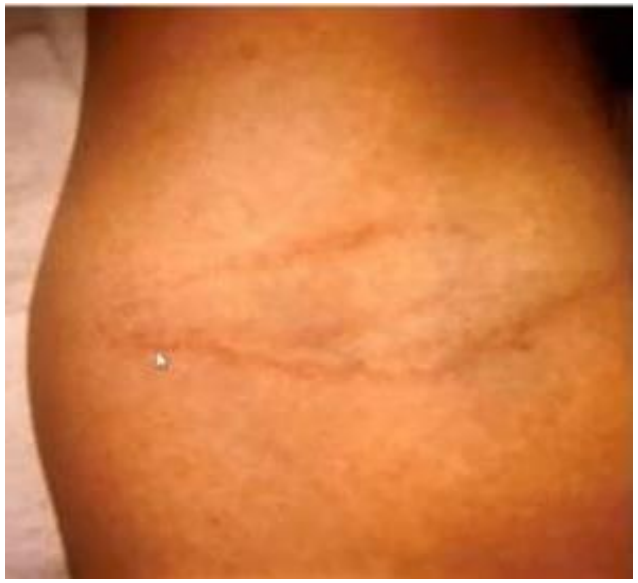


Maculopapular rash on the chest and extremities (Sand-paper rash)

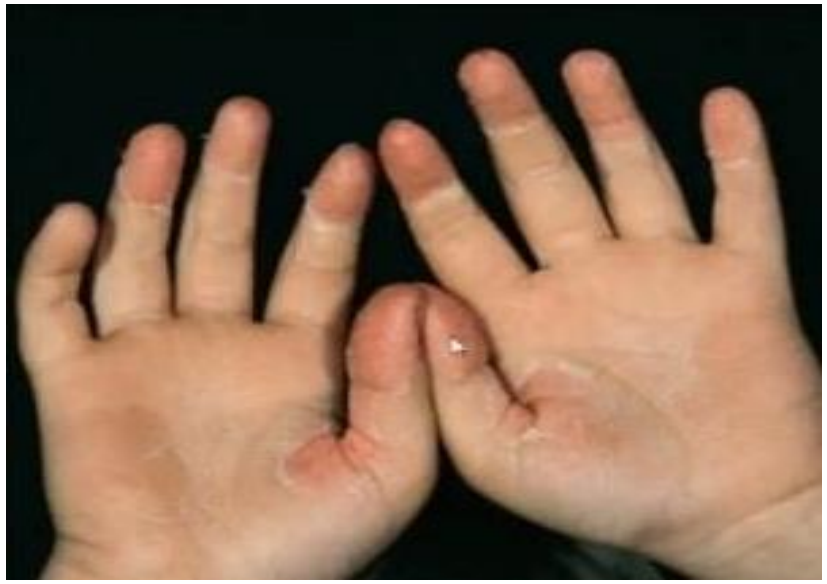


Strawberry tongue

Scarlet ever



Linear purpura on the flexural areas.
(Pastia's lines)



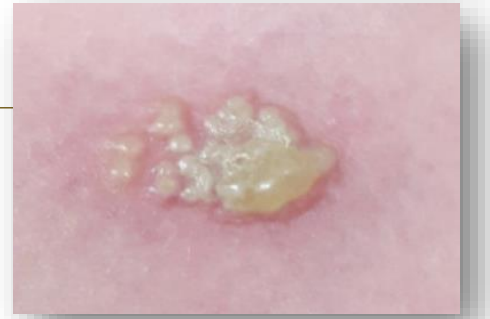
Desquamation and scaling in the limbs.

- **Investigations**:
  - Anti-ASO titer
- **Treatment**:
  - Antibiotic + Anti-Pyretic

Viral skin infections

Herpes simplex viruses

Herpes simplex



سنوات (1)

- **Primary lesion of herpes simplex**: Vesicle
- **Types of herpes simplex virus**
  - **HSV-1** is mainly associated with oral and facial infections but can occur in genital areas.
  - **HSV-2** is mainly associated with genital and rectal infections (anogenital herpes) and can be transmitted sexually.
- **Herpes simplex infection (Primary and recurrent)**
  - **Primary HSV-1**: Asymptomatic or gingivostomatitis; intend to be more severe than recurrences
  - **Recurrent HSV-1**: Cold sores
  - **Primary HSV-2**: Genital herpes after the onset of sexual activity
  - **Recurrent HSV-2**: Recurrent infections are common

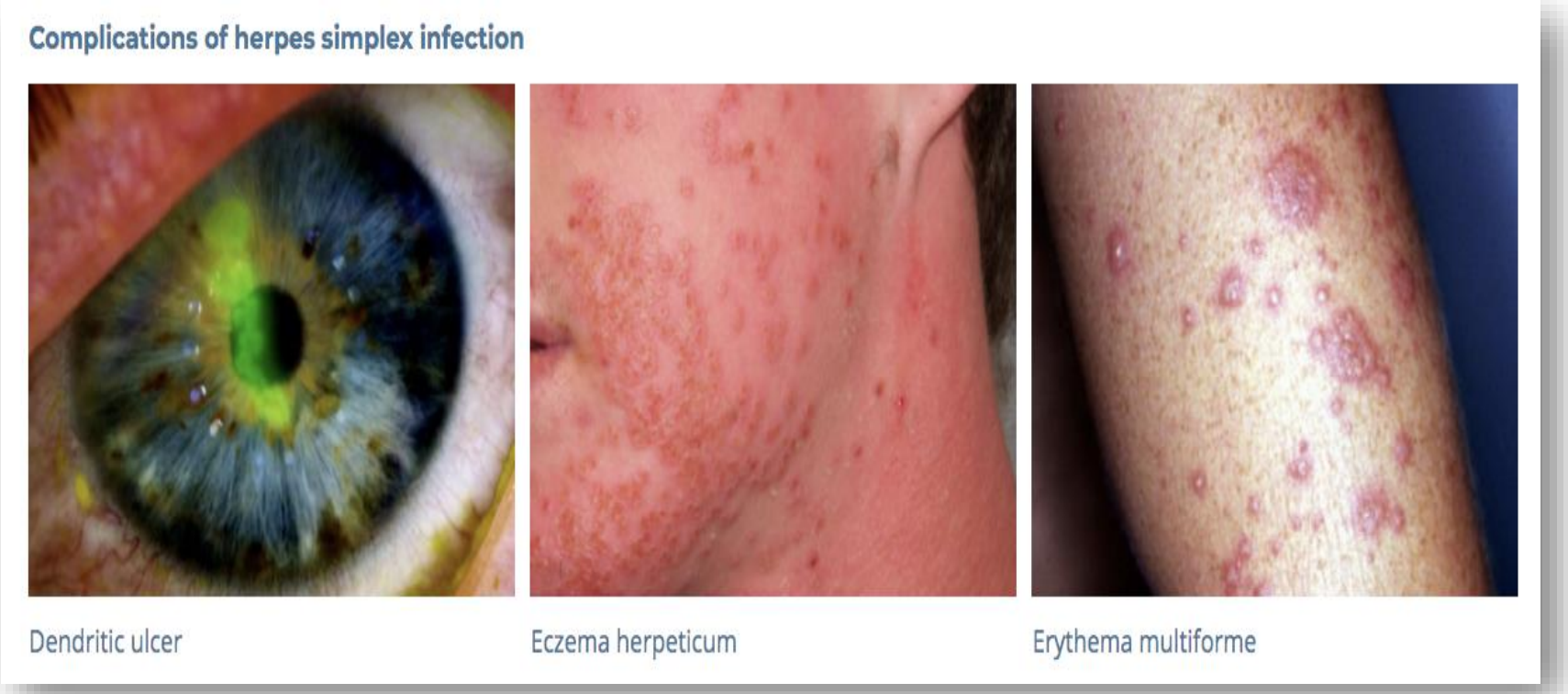
Herpes simplex

- Note: Every female which has been infected with HSV 2 or HPV should make PAP smear every 6 months to check for cervical intraepithelial neoplasia (CIN).
- **Herpes simplex recurrences can be triggered by:**
  1. Minor trauma, surgery or procedures to the affected area (must do serology to detect whether its HSV-1 OR HSV-2).
  2. Upper respiratory tract infections.
  3. Sun exposure.
  4. Hormonal factors (in women, flares are not uncommon prior to menstruation).
  5. Emotional stress.
  6. In many cases, no reason for the eruption is evident.

Herpes simplex

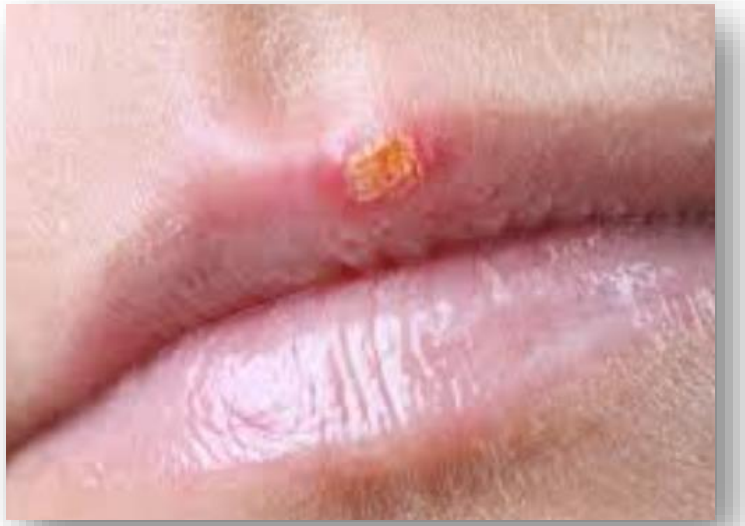
❖What are the complications of herpes simplex?

1. Eye infection (Keratoconjunctivitis)
2. Throat infection
3. Eczema herpeticum
4. Erythema multiforme
5. Encephalitis (temporal lobe)
6. Meningitis is rare
7. Widespread infection (immune deficient patients)



Herpes simplex

❖What is the causative agent of

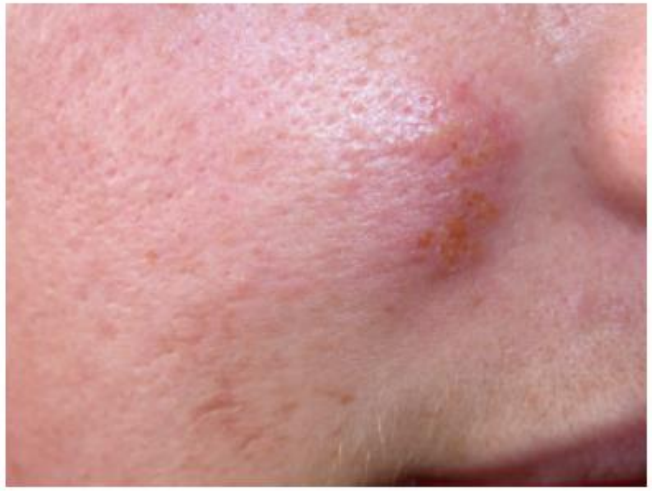


- **Cold sores**: mainly HSV-1
- **Anogenital herpes**: mainly HSV-2
- **Herpetic whitlow**: Both HSV-1 and HSV-2
- **Eczema herpeticum**: HSV (سنوات (1))
- **Keratoconjunctivitis (as a complication)**: HSV-1
- **Encephalitis (as a complication)**: HSV-1
- **Meningitis (as a complication)**: HSV-2
- **Erythema multiforme**: Most common cause is HSV specially HSV-2, 2nd most common is *Mycoplasma*

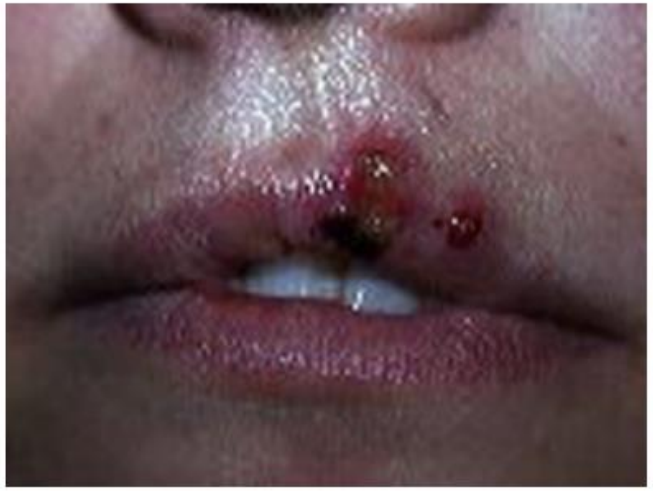
❖How is herpes simplex diagnosed

- Clinically
- Tzanck smear
- If in doubt, Culture or PCR can confirm the diagnoses

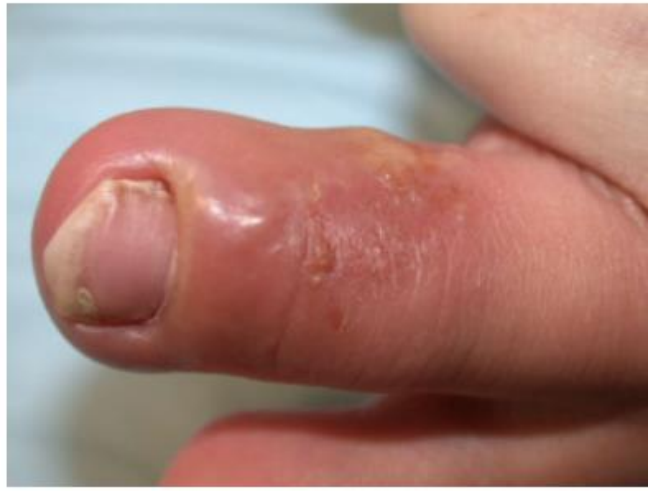
Herpes simplex



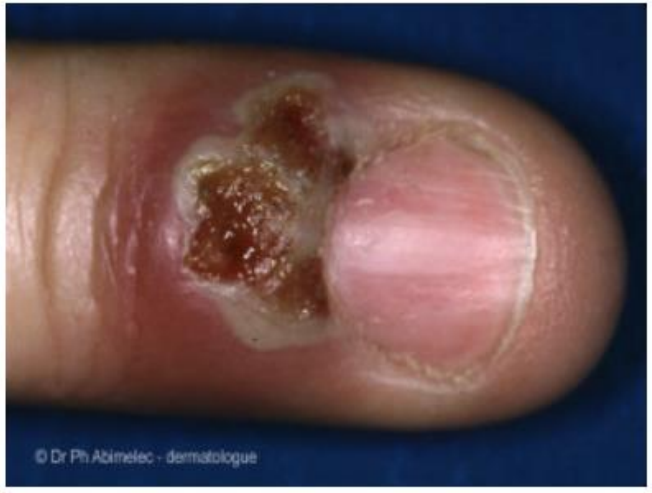
Herpes in a netball player



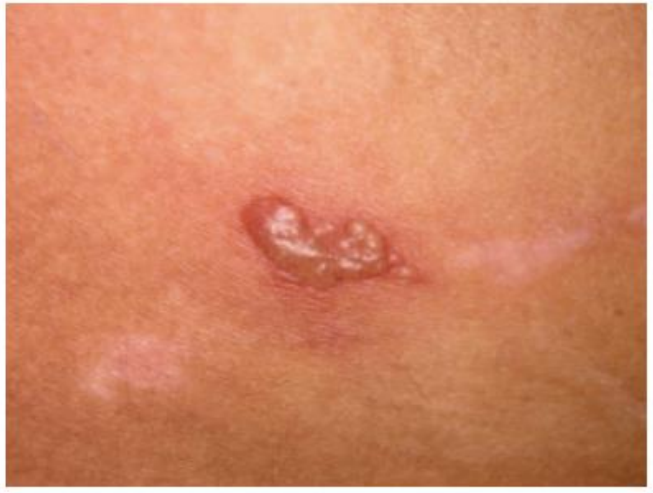
First episode of herpes



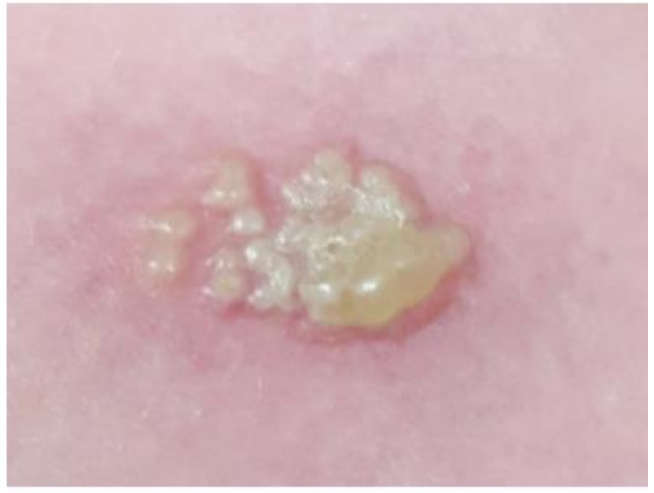
Herpetic whitlow



Herpes simplex paronychia



Scarring and blistering on buttock



Cluster of vesicles due to HSV2

Herpes simplex

❖What is the treatment for herpes simplex?

- Mild, uncomplicated eruptions of herpes simplex require no treatment.
- Severe infection may require treatment with an antiviral agent
- Fucidic acid if there is superimposed impetigo

❖Mention 2 antiviral used in the treatment of herpes simplex

- Acyclovir & Valacyclovir

❖Can herpes simplex be prevented?

- Sun protection
- Antiviral drugs (shorten and prevent attacks but a single course cannot prevent future attacks)
- Stop Oral contraceptive pills

Herpes Simplex

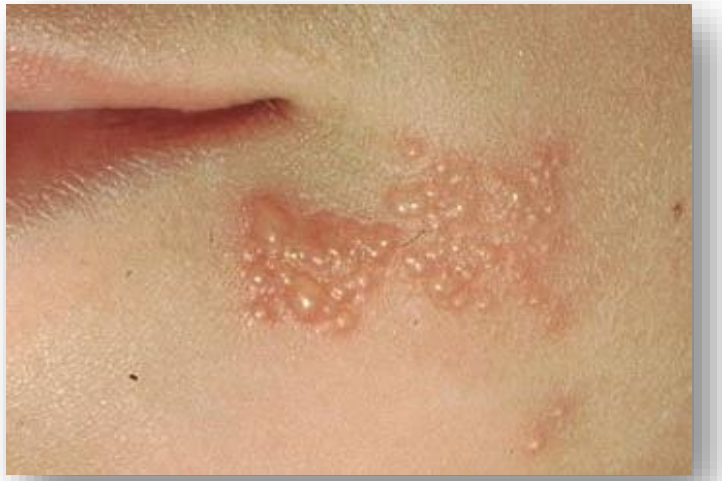
- **Describe**
  - Clear vesicles sitting on top of an erythematous base "dew drops on a rose petal appearance"
- **Diagnosis**
  - herpes simplex

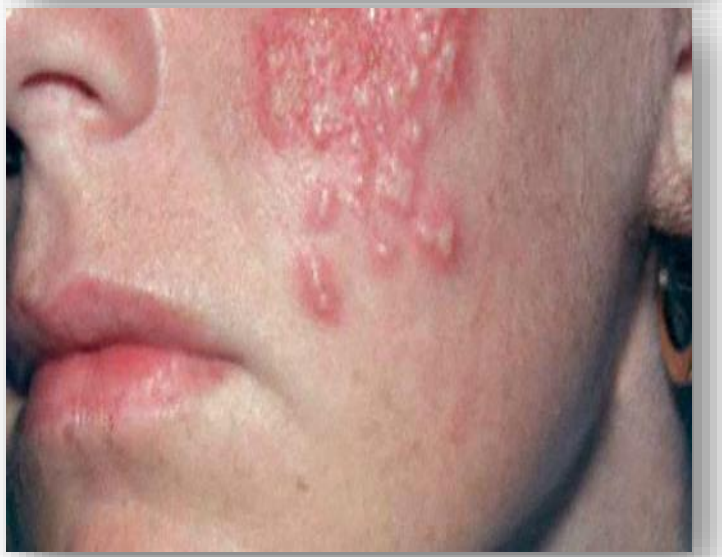
إضافي

- **Mention 2 complications**
  - Keratoconjunctivitis
  - Eczema herpeticum

إضافي

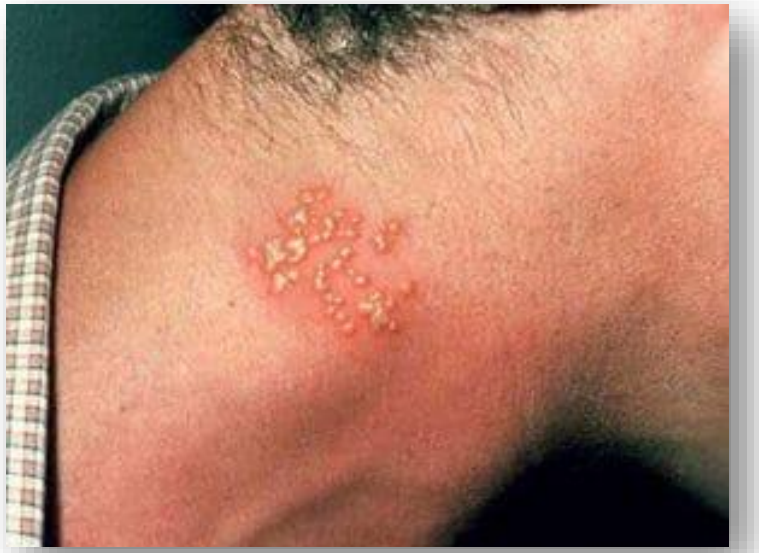
- **Mention 2 antiviral used in the treatment**
  - Acyclovir & Valacyclovir

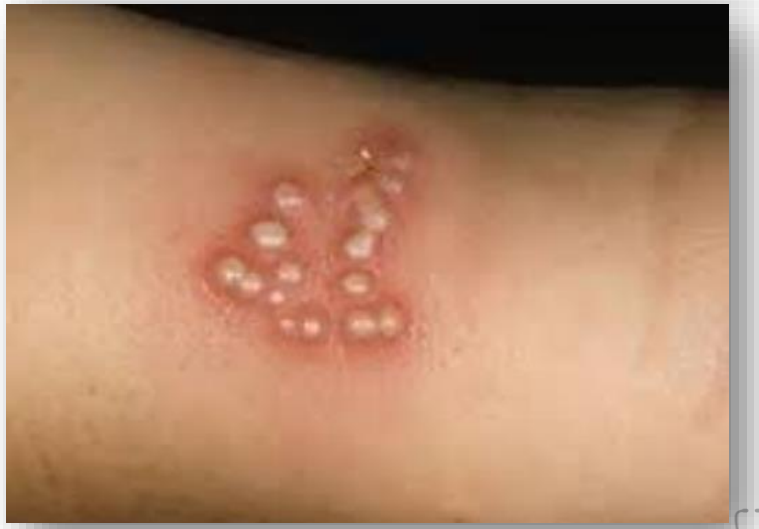




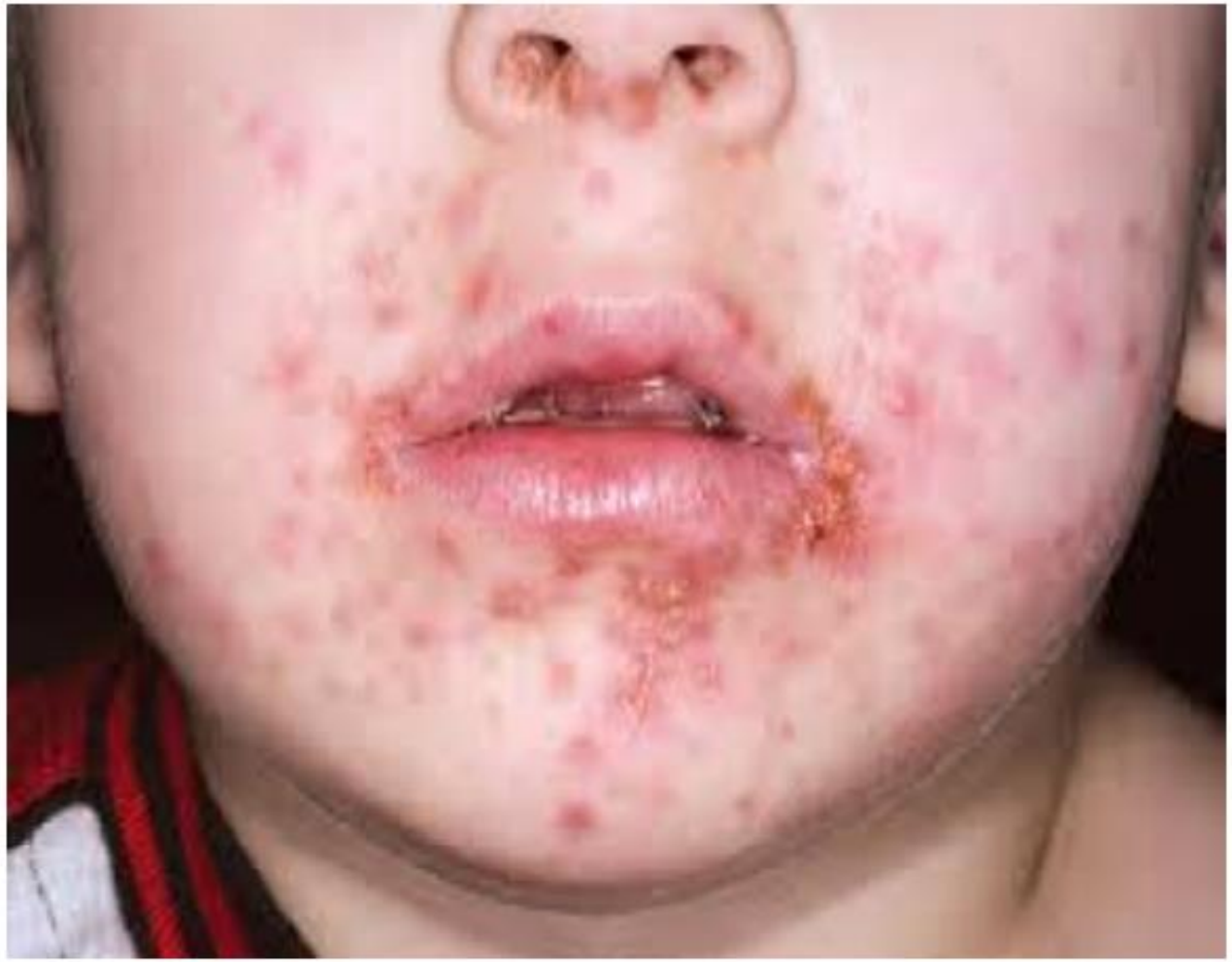
Herpes Simplex

- **Describe**
  - Clear vesicles sitting on top of an erythematous base "dew drops on a rose petal appearance"
- **Differential diagnosis**
  - Herpes simplex
  - Herpes zoster
  - Eczema herpeticum





Eczema herpetica



If only on the face
Impetigo contagiosa or
eczema herpetica

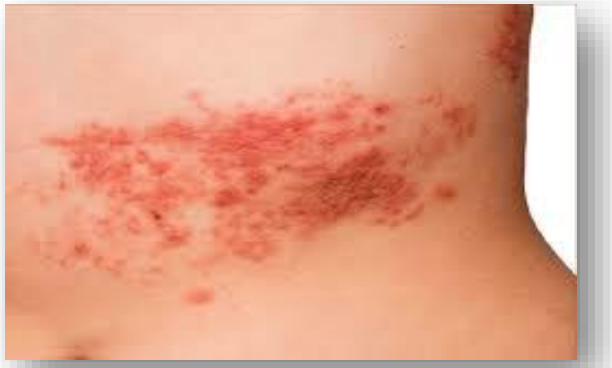
Varicella zoster virus

شرح

Varicella zoster virus

سنوات (1)

- **Primary lesion of herpes zoster**: Vesicle & blisters
- **What comes first in shingles the pain, fever and malaise or the rash ?**
  - Pain, fever and malaise precede the rash
- **What is the type of distribution of shingles ?**
  - Dermatomal



- **What is the cause of:**

سنوات (1)
  - **Chicken pox**: VZV primary infection

سنوات (2)
  - **Shingles (Herpes zoster):** VZV reactivation
  - **Ramsay Hunt syndrome**: VZV; shingles affecting the facial nerve

- **What does shingles with multi-dermatomal, extensive and hemorrhagic lesions suggest ?**
  - Underlying immunodeficiency such as HIV

شرح

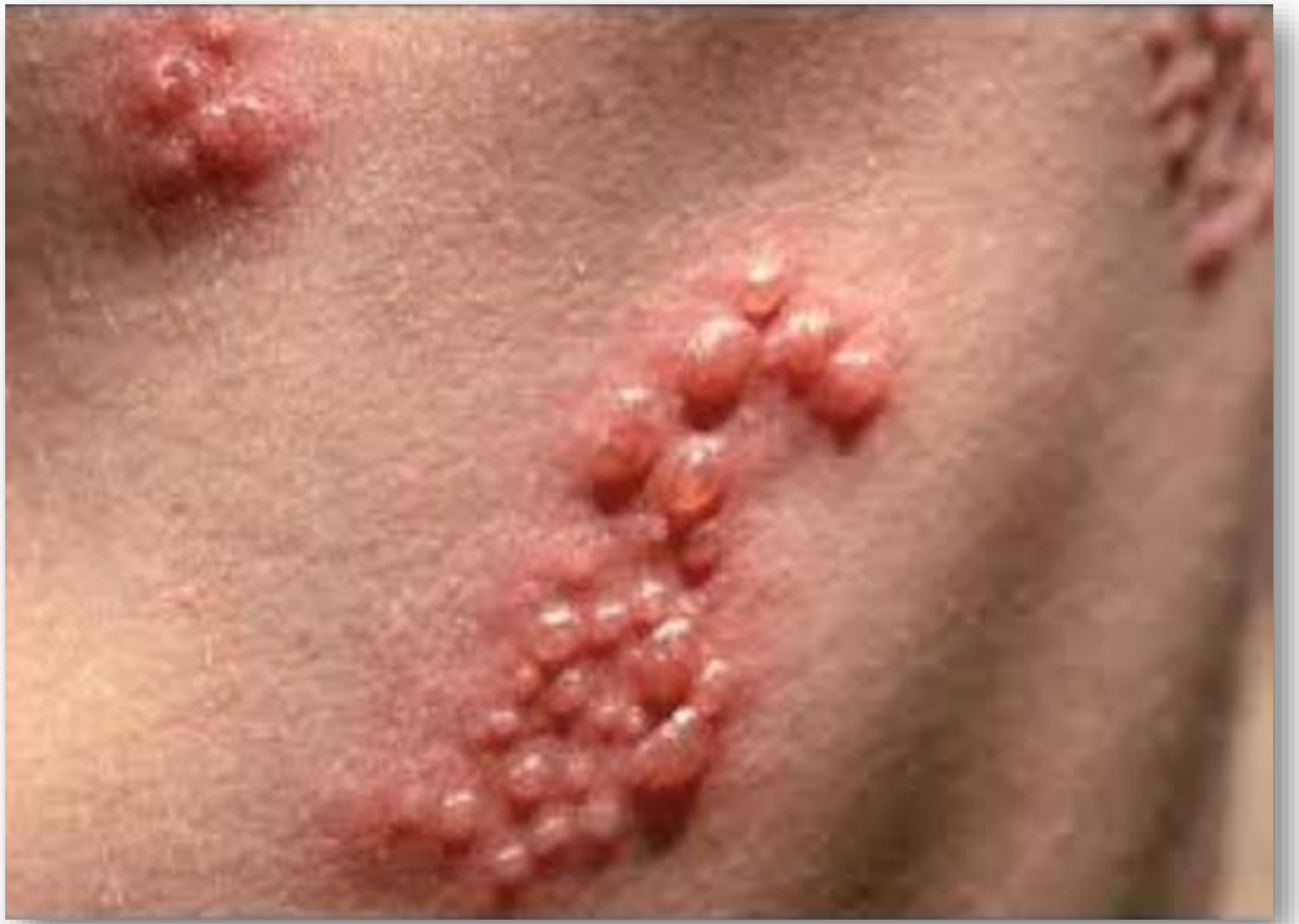
Varicella zoster virus

- **The infection of the following nerves by VZV can present with what ?**
  - **The ophthalmic nerve**: severe conjunctivitis
  - **The Maxillary nerve**: vesicles on the uvula or tonsils
  - **The mandibular nerve**: vesicles on the floor of the mouth and on the tongue
  - **The facial nerve**: lesions in the external auditory canal (Ramsay Hunt syndrome)
- **What is the most effected nerve**
  - The thoracic nerves
- **How is it diagnosed ?** Clinically, Tzanck smear
- **First line treatment**

سنوات (1)

  - Adult with chickenpox presented after 10 Hours: oral Valaciclovir 500mg / 3 times per 5 days
  - Post-herpetic neuralgia: gabapentin or carbamazepine

Herpes zoster (الحزام الناري)



Warts

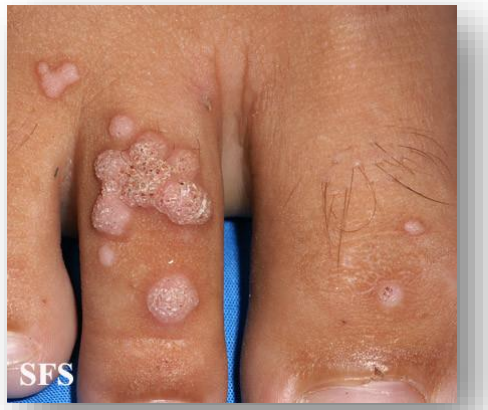
شرح

Warts

- What is the causative agent of
  - **Warts**: HPV 1-4 infection of keratinocytes
  - **Condyloma acuminata**: HPV6; HPV11
  - **CIN, Cervical Carcinoma and penis SCC**: HPV 16, 18, 31, 33 (high risk HPV)

سنوات (1)

- Plantar warts (verrucae) form painful plaques (mosaic) containing black 'dots' that **represent** thrombosed capillaries.



سنوات (2)

- **Mention the treatment modalities of warts**
  1. Topical agents such as salicylic acid and lactic acid
  2. Cryotherapy
  3. Curettage and cautery for very large warts
  4. Immune response modifier Imiquimod for the treatment of genital warts

Mention 5 variants of warts

1. **Common wart (verruca vulgaris)**:
   - Raised wart with roughened surface
   - Most common on hands
2. **Flat wart/plane warts**:

سنوات (2)

   - Small, smooth flattened wart, flesh-colored, which can occur in large numbers
   - Most common on the face, neck, hands, wrists and knees
3. **Filiform or digitate wart**:
   - Thread-like or finger-like wart,
   - Most common on the face, especially near the eyelids and lips
4. ***Condyloma acuminata***:
   - Wart that occurs on the genitalia
5. **Periungual wart**:
   - Cauliflower-like cluster of warts
   - Occurs around the nails,painful
6. **Plantar wart (verruca plantaris)**:
   - Hard, sometimes painful lump, often with multiple black specks in the center;
   - Usually only found on pressure points on the soles of the feet
7. **Mosaic wart**:
   - Group of tightly clustered plantar-type warts,
   - Commonly on the hands or soles of the feet

Types of warts

1

Common warts

2

Plane warts

3

Filiform warts

4

Periungual warts

5

Plantar warts

6

Mosaic wart

Warts

- What is the cause of this lesion?
  - HPV
- Mention 2 other clinical form?
  - Common wart
  - Genital wart
- Mention 2 line of treatment?
  - Cryotherapy
  - Curettage and cautery
- Cause of these black dots
  - Thrombosed veins

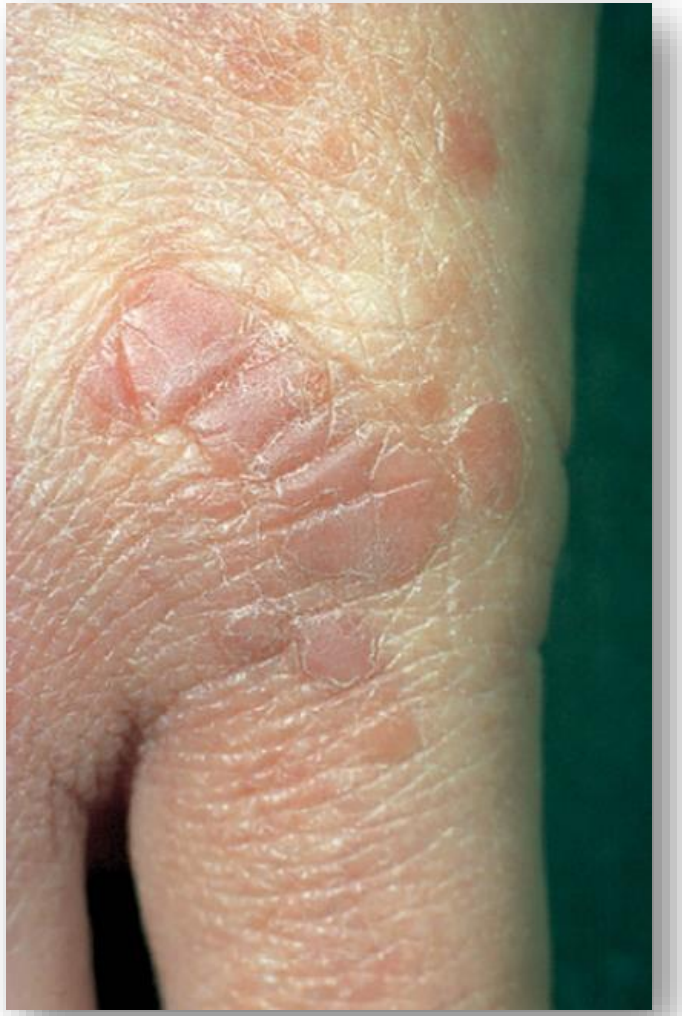
What is your diagnoses ?

Planter warts

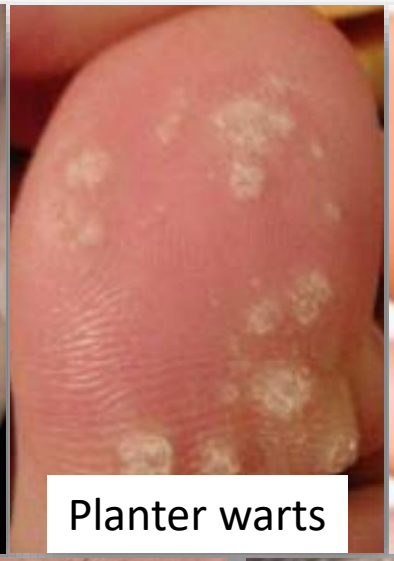
Which type of warts can resolves spontaneously ?

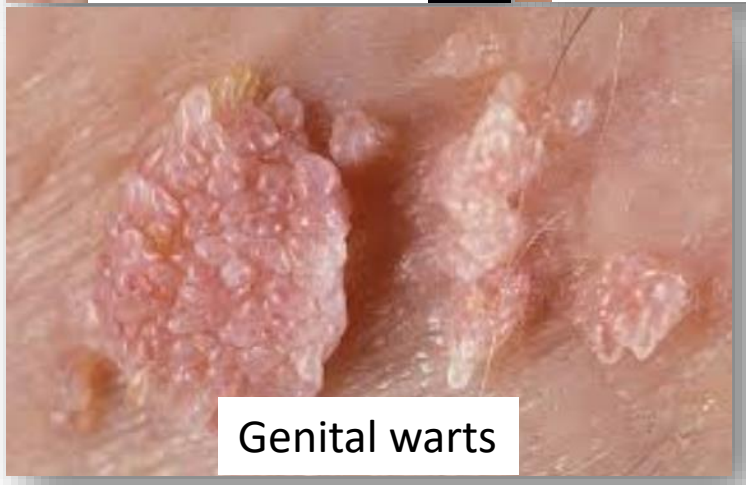
❖**Answer**: Plane warts

- Note: Plane warts are extremely difficult to treat effectively and attempts at treatment may do more harm than good. They will resolve spontaneously eventually and are best left alone.



Warts





Molluscum contagiosum

المليساء المعدية

شرح

Molluscum contagiosum

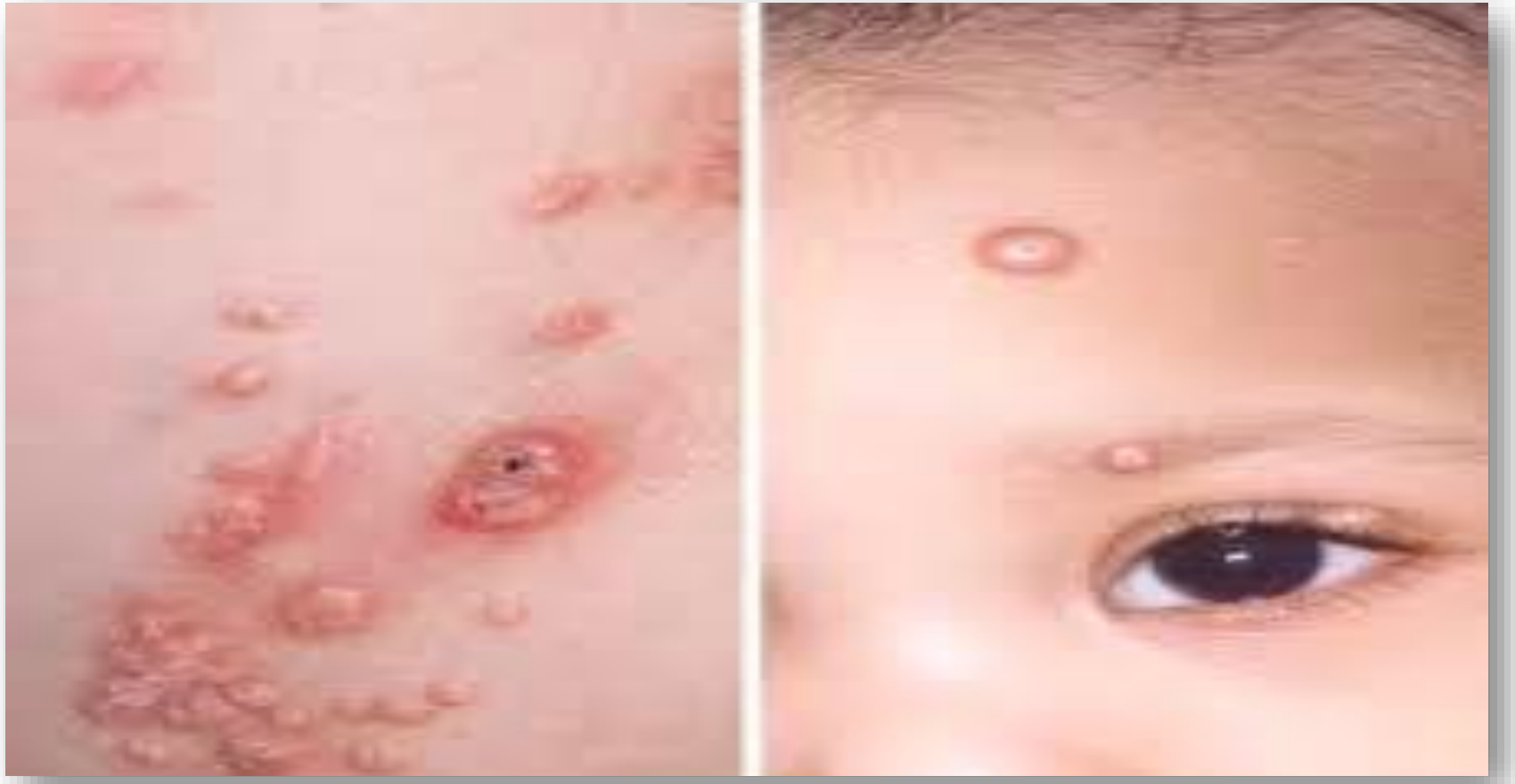
- **The primary lesion of molluscum contagiousm**: papule
- **Causative agent of molluscum contagiosum**: Poxvirus

سنوات (1)

- **What is the form of transmission ?**
  - Direct contact; specially in children
  - Sexually transmitted if present in the genital area
- **What does florid molluscum in adults indicate ?**
  - Underlying immunodeficiency such as HIV
- **Treatment of molluscum contagiosum**
  - Cryotherapy
  - Imiquimod in immunocompromised patients not responding to destructive methods

Molluscum contagiosum

- **Describe the lesion seen in the picture**
  - Shiny pearly papules with central umbilication.



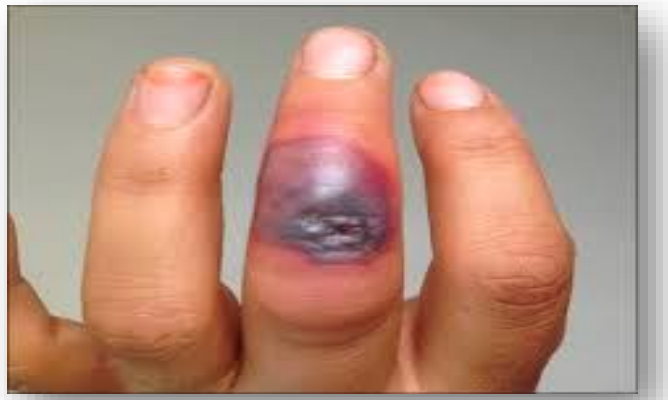
Orf

Orf

- Orf is a zoonotic viral skin infection that is contracted from sheep and goats
- **The primary lesion of orf**: Nodule
- **Causative agent of orf**: Parapoxvirus
- **Risk group**: Butchers, meat porters and housewives
- **Site of lesion**: most commonly on the fingers, hands or forearms but can appear on the face
- **Description**: small, firm, red or reddish-blue lump enlarges to form a flat-topped, blood-tinged pustule or blister
- **How is it diagnosed**: Clinically
- **Treatment**:

سنوات (1)

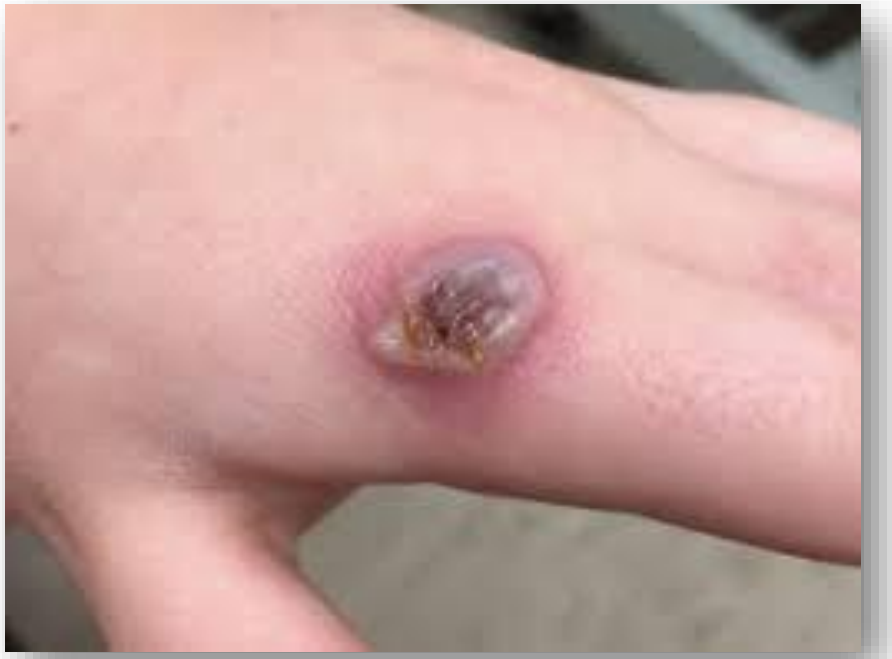
  - Cryotherapy (first line)
  - Antibiotics for secondary bacterial infections



Orf

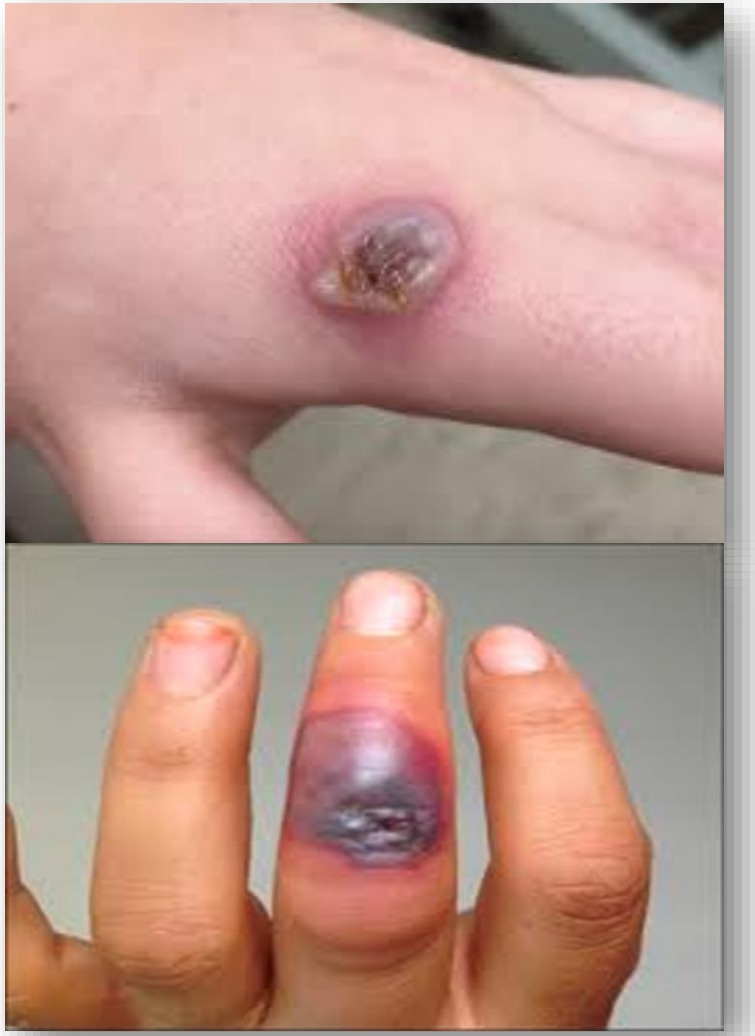
❖Complications:

1. Secondary bacterial infection can occur
2. Erythema multiforme
3. Lymphangitis with lymphadenopathy
4. There may be a mild fever

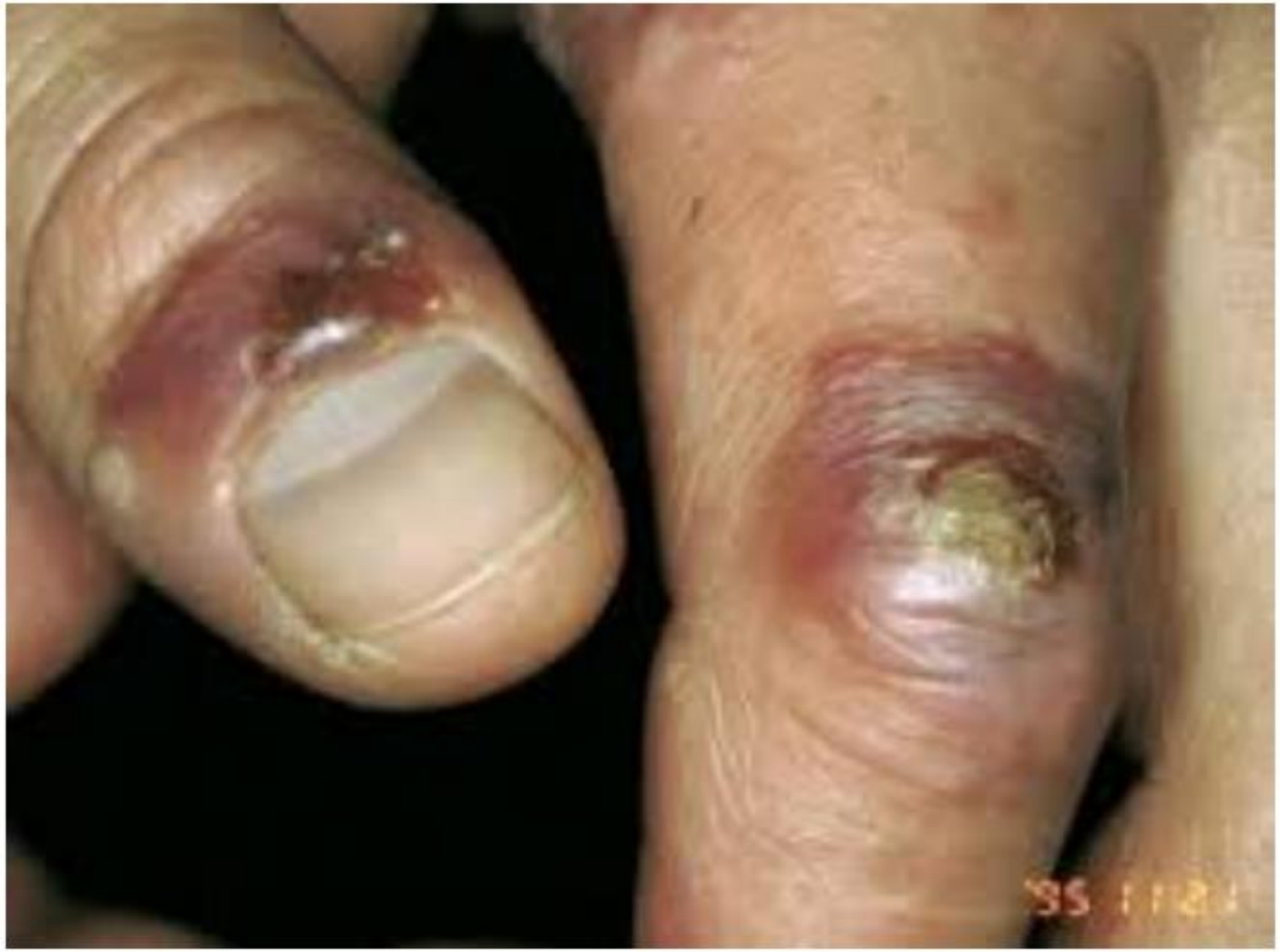


Orf

- **Describe what you see**
  - A solitary, inflammatory nodule of granulation tissue
- **Diagnosis**
  - Orf
- **Differential diagnosis**
  - Insect bite
  - Infected wound
  - Leishmania
  - Tinea manuum



Orf



Ddx:
Insect bite
Infected wound
orf (viral infection from meat)

Hand foot mouth disease

شرح

Hand foot mouth disease

- Also called enteroviral vesicular stomatitis
- سنوات (1) **The primary lesion of hand foot mouth disease**: Vesicle
- سنوات (1) **Causative agent of hand foot mouth disease**: Coxsackie A16 virus
- **Lesion description**: Vesicles and blisters on the hands, feet and in the mouth

شرح

Hand foot mouth disease

❖**Mention 3 differential causes of rash on the palms and soles?**

- 2° syphilis, Rickettsia rickettsii, Coxsackievirus A

❖**How is it diagnosed**: Clinically

❖**Treatment**: Adequate fluid intake, antiseptic mouthwashes, topical and oral analgesics, the blisters should not be ruptured, to reduce contagion

سنوات (1)

❖**Complications**

- Dehydration due to inadequate fluid intake
- Fingernail and toenail changes are often noted about 2 months after infection

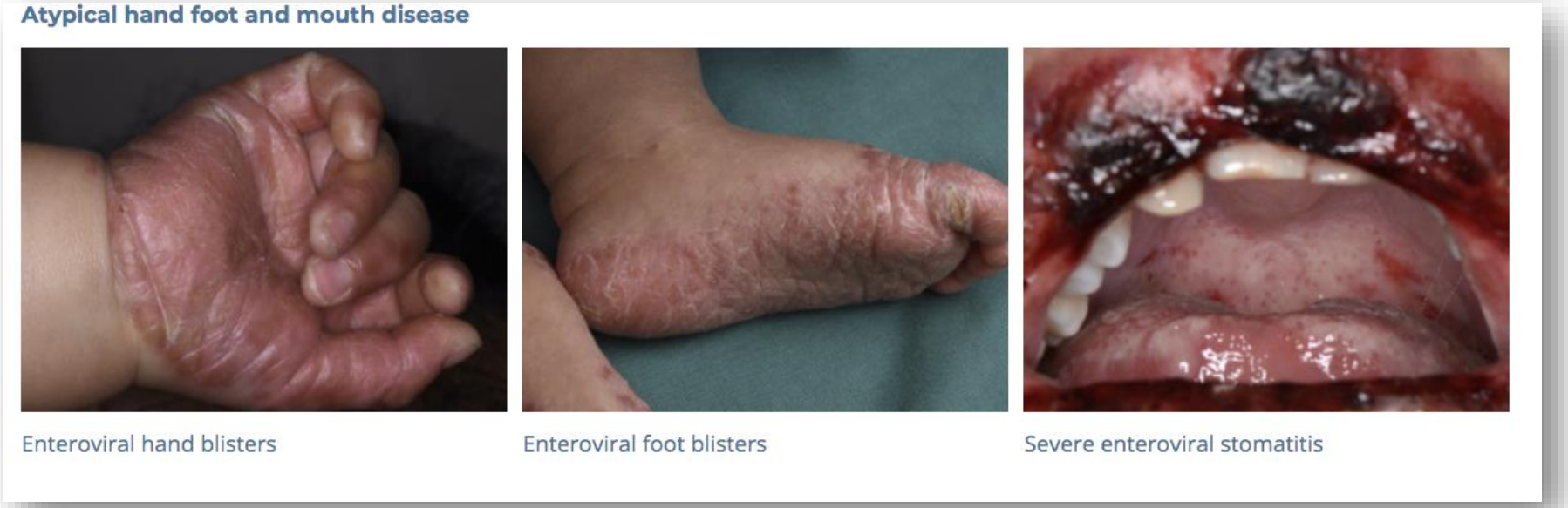
1. Transverse lines that slowly move outwards

سنوات (1)

2. Nail shedding (onychomadesis) about 2 months after the illness.
3. Eventually, the nails return to normal

Hand foot mouth disease

- Atypical disease (more widespread rash) features:
  1. Red, crusted papules
  2. No blisters or very large ones
  3. Targetoid lesions
  4. Involvement of unusual sites such as the ear
  5. In children with atopic dermatitis, lesions may select skin affected by eczema (eczema coxsackium)



Hand, foot and mouth disease

❖**What is the causative organism of this disease ?**

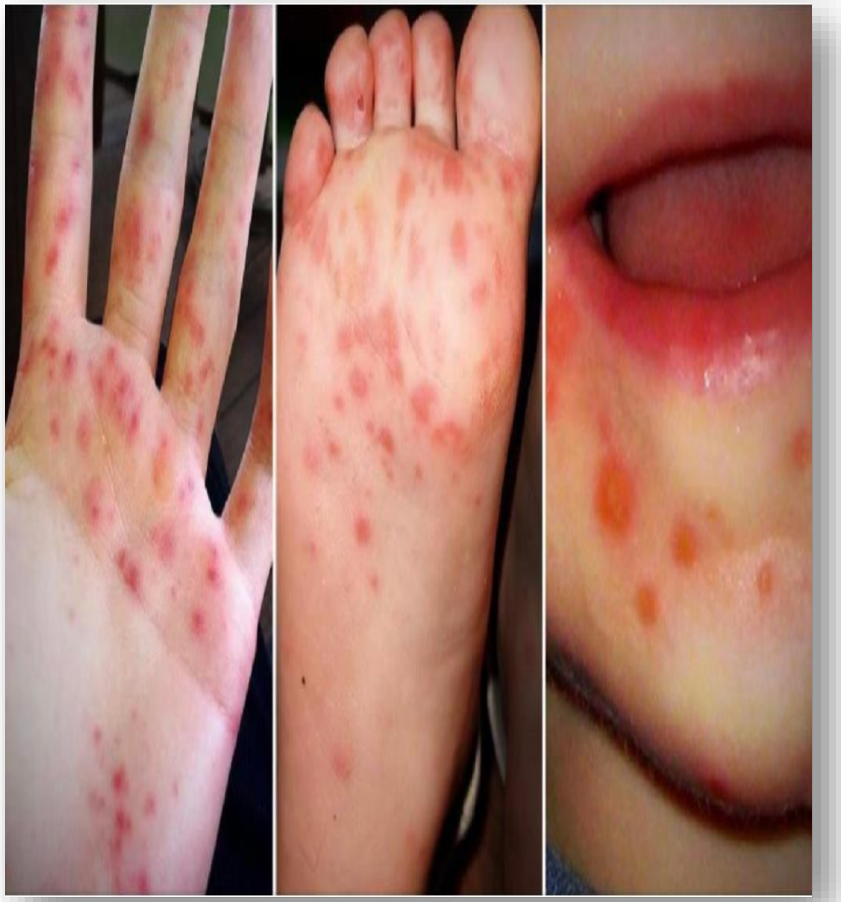
- Coxsackie virus A16

❖**Mention the complications**

1. Dehydration due to inadequate fluid intake
2. Transverse lines that slowly move outwards
3. Nail shedding (onychomadesis) about 2 months after the illness

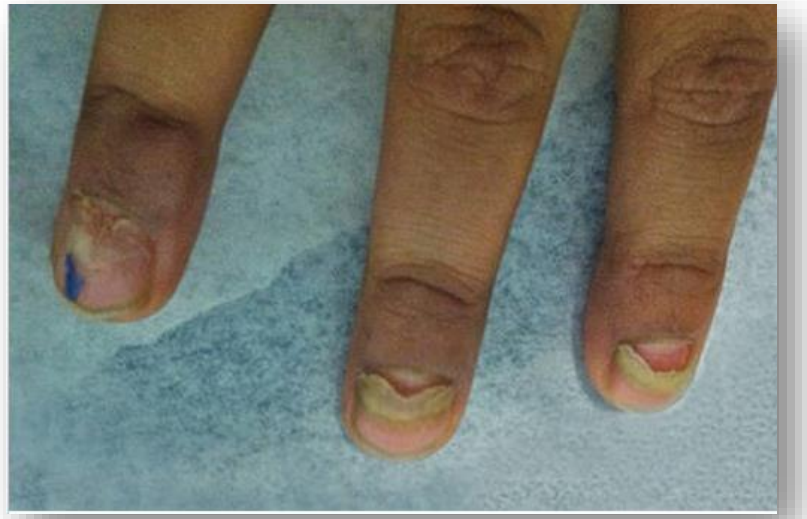
❖**Mention 2 other differential causes ?**

- 2° syphilis, *Rickettsia rickettsii*



Hand, foot and mouth disease

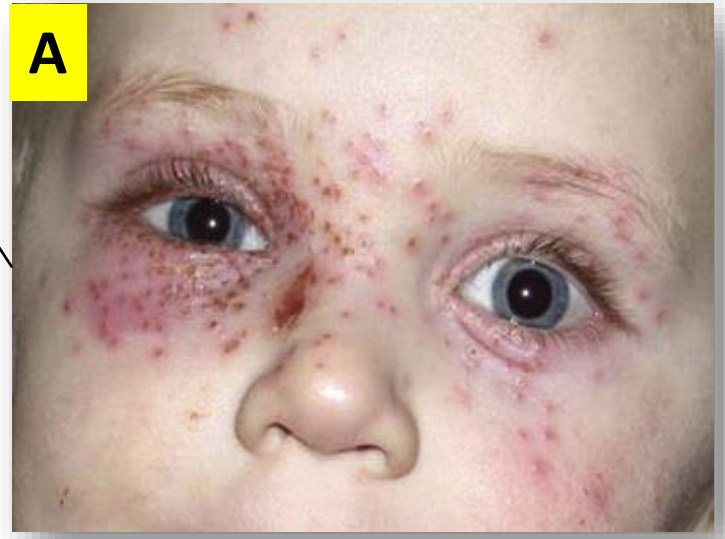
- **Describe this sign**
  - Onychomadesis (Nail shedding)
- **In which disease is this sign seen**
  - Hand, foot and mouth disease

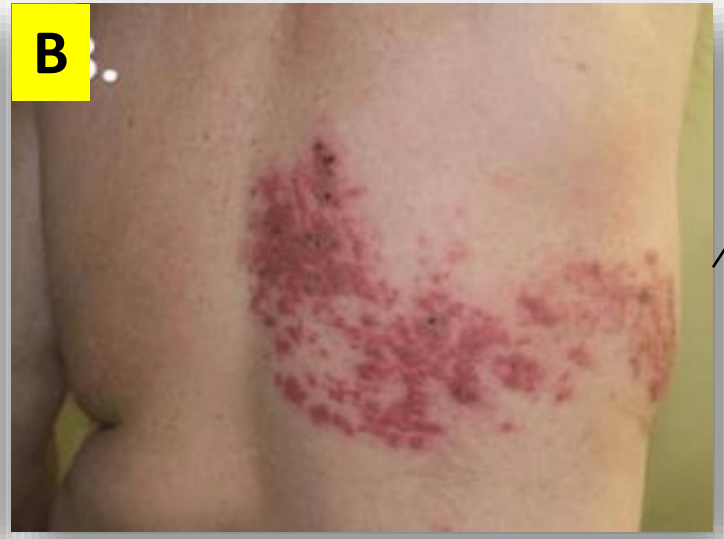


Other Question

What is your diagnosis ?

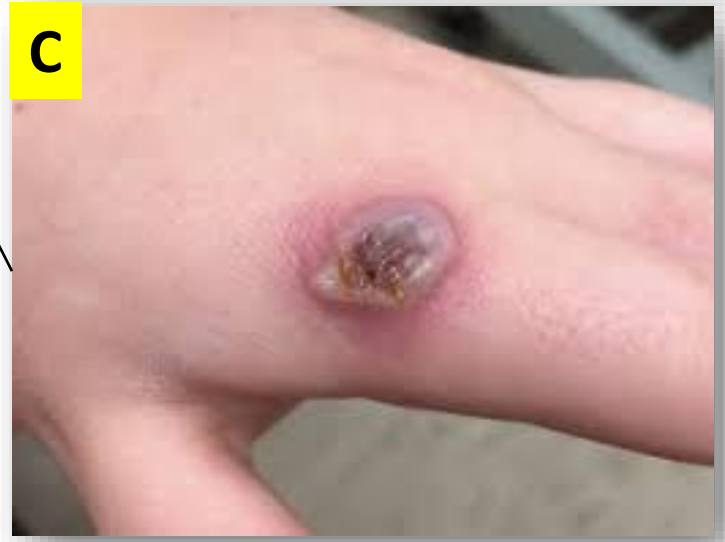
Eczema herpeticum

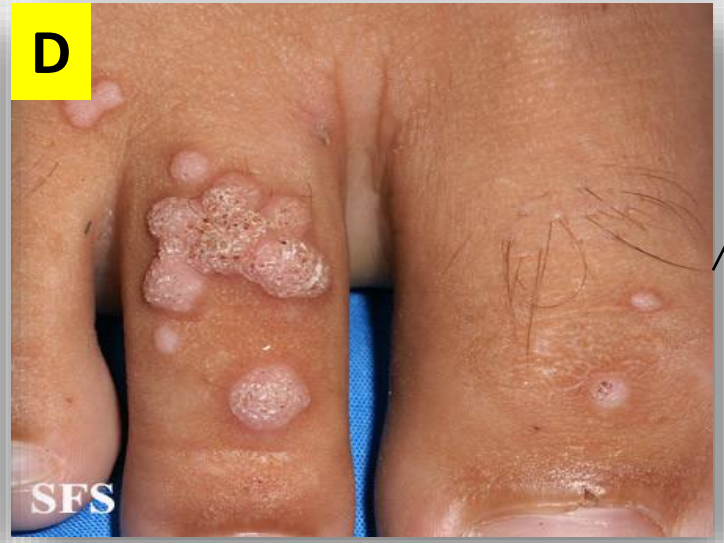




Herpes zoster

Orf





Warts

What is your possible diagnosis

- 50-years old female with pink shiny smooth papule on the genital area
  Dx: Molluscum contagiosum
- Child with multiple pink shiny papule on his face
  Dx: Molluscum contagiosum
- Child presented with maculopapular rash which had resolved after few days associated with lymphadenopathy
  Dx: Rubella
- Child come with erythema of the soft palate and lymphadenopathy
  Dx: Rubella

- **Note**: read more about childhood exanthems here, if you wish to know more about rubella and the other exanthems


Fungal skin infections

Fungal skin infections

- **Mention 3 common superficial Fungal Infection:**
  - Dermatophytosis
  - Tinea versicolor
  - Candidiasis
- **Which fungi commonly cause inflammatory response ?**
  - Dermatophytosis infections
  - Candidiasis infections
- **Which fungi cause minimal if any inflammation ?**
  - Tinea versicolor
  - Tinea Nigra
  - Black Piedra
  - White Piedra

Dermatophytosis

❖**There are three genera of Dermatophytes:**

- Microsporum
- Trichphyton
- Epidermophyton

❖**Types of Dermatophytes by mode of transmission**

- Anthropophilic (Human to human); mild & chronic
- Geophilic (Soil to human or animal); moderate
- Zoophilic (Animal to human); marked & acute

❖**Clinical presentations of dermatophytosis**

1. Tinea capitis
2. Tinea barbae
3. Tinea corporis
4. Tinea Cruris
5. Tinea Pedis
6. Tinea manum
7. Tinea Unguium

Tinea capitis

❖ **Demographic**:

- Most commonly seen in children

سنوات (1)

❖ **Mention 3 clinical variants**

- Black dot

سنوات (6)

- Kerion (due to Cattle ringworm (*T. verrucosum*))
- Favus (due to *T. Schoenleini*) bluish fluorescence by Wood`s light examination

❖ **How to prove the diagnosis of Tinea Capitis ?**

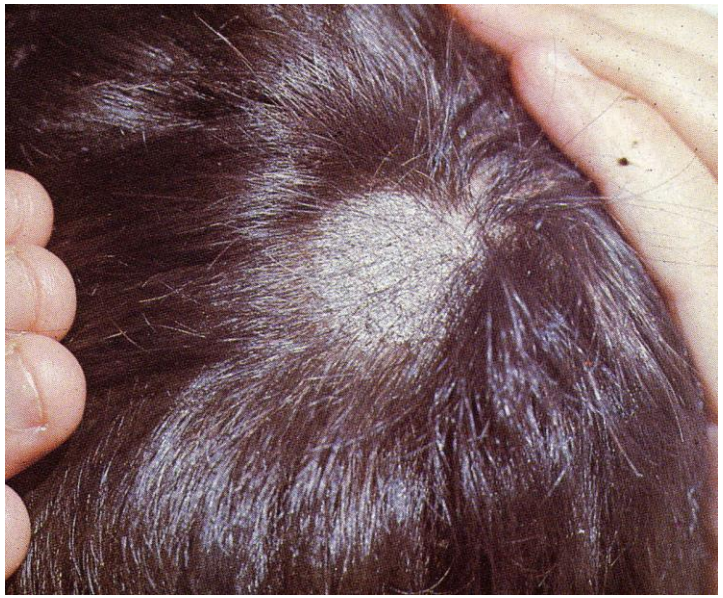
سنوات (1)

- KOH preparation (Best diagnostic tool for Tinea Capitis)
- Easily plugged hairs from the affected area

❖ **Treatment**:

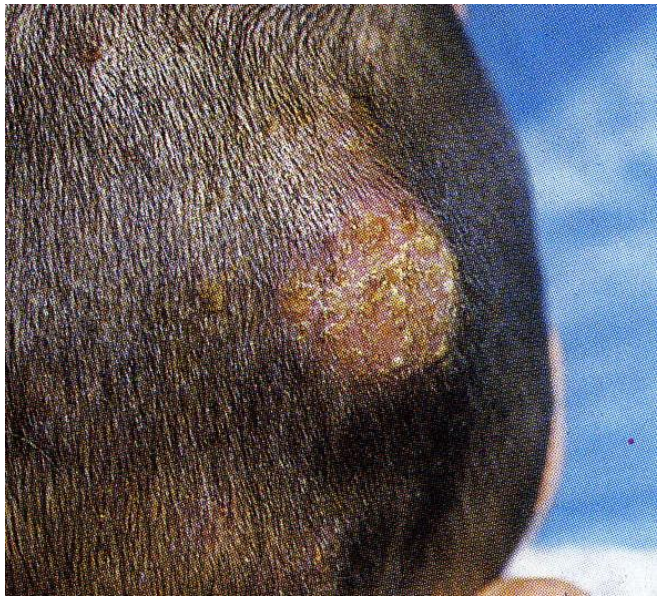
- Oral + topical antifungal (cream or shampoo)

Tinea capitis



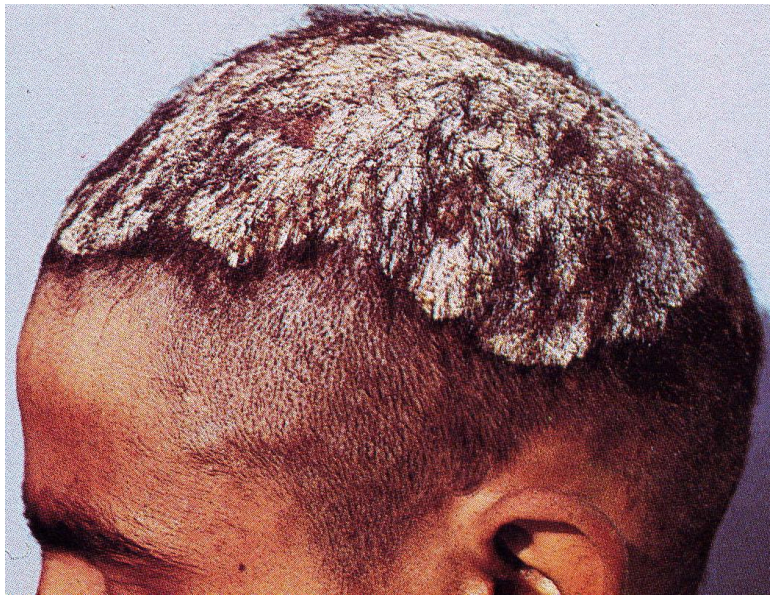
Black dot Tinea capitis

Localized hair loss with scaling



Kerion Tinea capitis

Localized hair loss with scaling and Inflamamtion of the scalp



Favus or Scutula

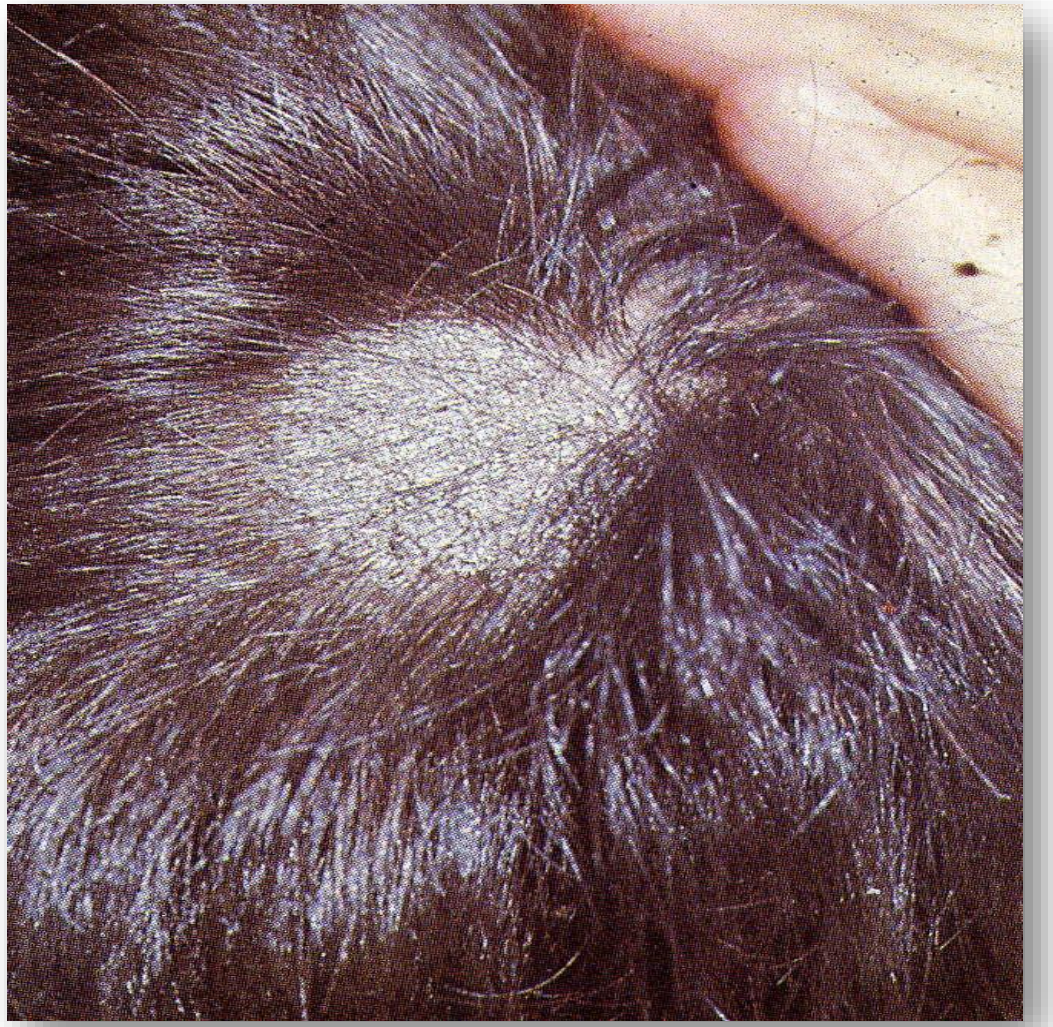
Child with scaly Scalp lesion

سنوات (3)

- **Probable diagnosis**
  - Tinea capitis
- **Mention 2 other causes of patchy alopecia in children**
  - Alopecia areata
  - Chronic traction

سنوات (2)

- **Mention 2 Investigations**
  - Wood's light
  - KOH prep
- **Treatment**
  - Oral imidazole
  - Fucidic acid

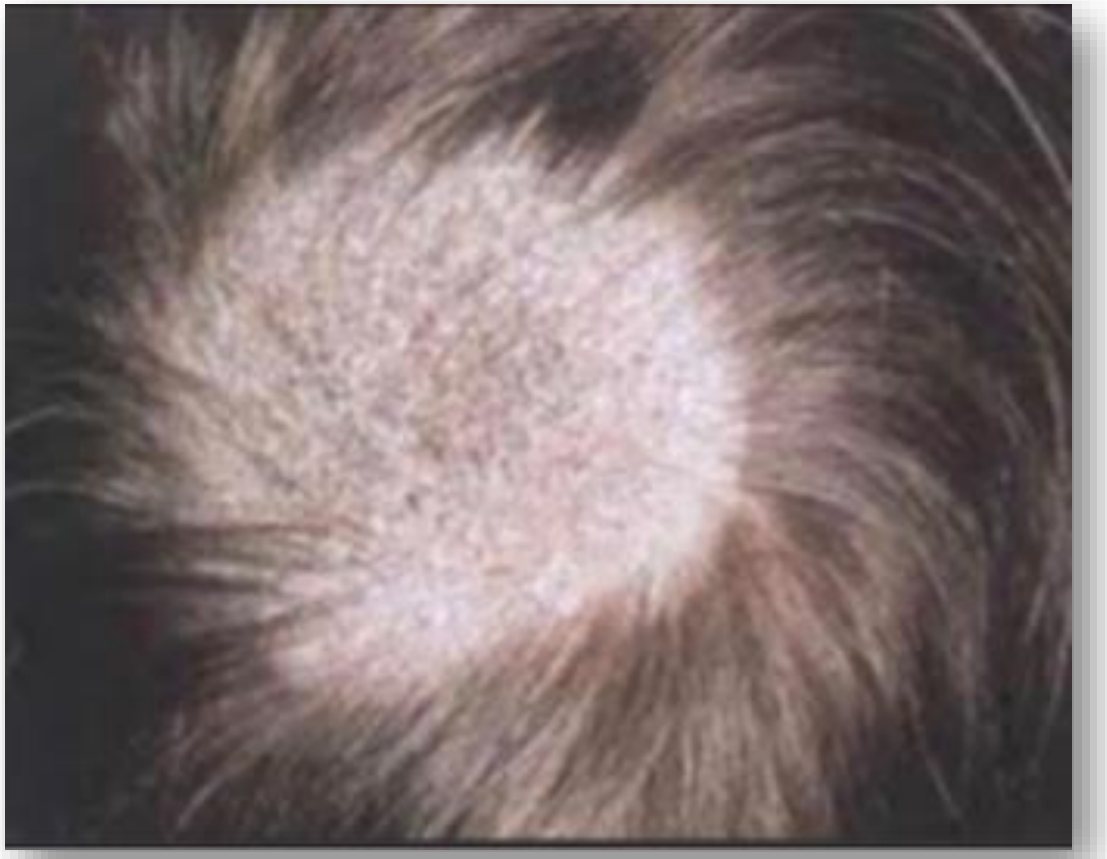


Black dot Tinea capitis

Patchy hair loss with erythema and scales

- **Probable diagnosis**
  - Tinea capitis
- **Mention 2 differential diagnosis**
  - Psoriasis
  - Atopic dermatitis
  - Seborrheic dermatitis
  - Tinea amiantacea
  - Pityriasis rubra pilaris
- **Mention 2 Investigations**
  - Wood's light
  - KOH prep

- **What type of drug is used ?**
  - Systemic and topical antifungal



Black dot Tinea capitis

Patient With Annular scaly lesion

سنوات (1)

❖You should exclude what ?

- Tinea corporis

سنوات (2)

❖Mention the variants of this disease:

1. Black dot
2. Kerion
3. Favus

❖What is the cause of each variant?

- Black dot: Dermatophyte

سنوات (6)

- Kerion: Dermatophyte (Cattle ringworm)

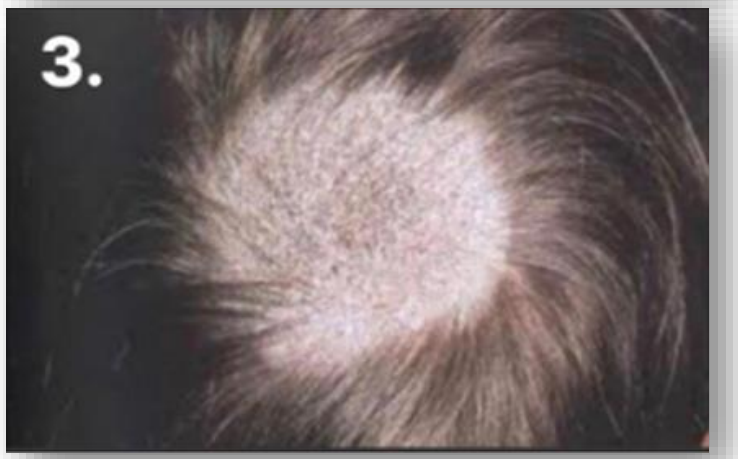
سنوات (2)

- Favus: Dermatophyte (*T. Schoenleini*)

أنك تجاوب (dermatophytes) كافي

Tinea capitis

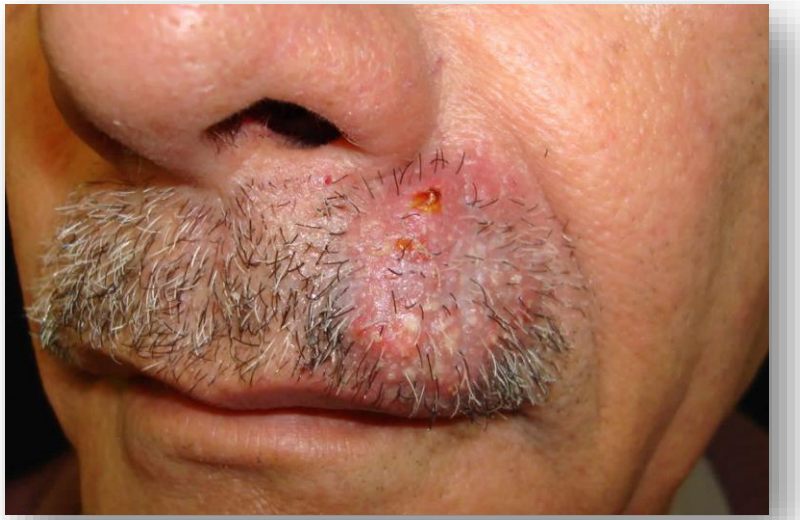
- **What is the cause ?**
  - Dermatophyte
- **Mention 2 systemic drugs used in treatment**
  - Fluconazole
  - itraconazole
- **What is name of the test in the first picture ?**
  - Wood's light test
- **Color on the wood's light ?**
  - Green



Dermatophytosis

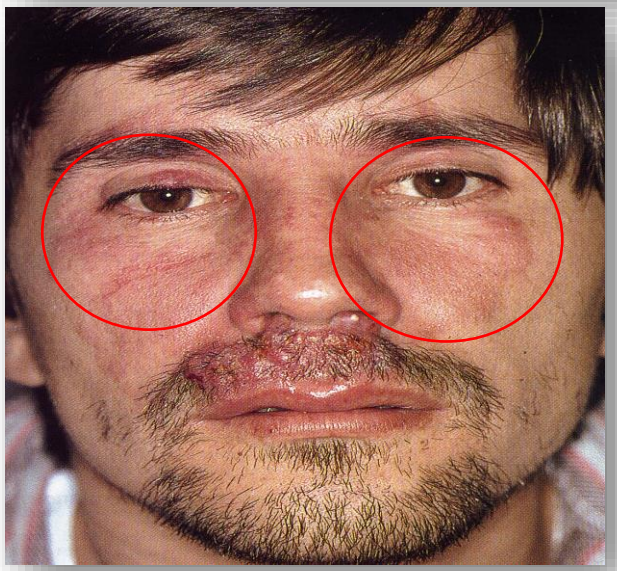
Tinea barbae

- Dermatophyte infection of the beard area
- Not a common disease
- Usually unilateral on the face or neck of a man
- DDx: Bacterial folliculitis
- **Treatment**: Oral + topical antifungal

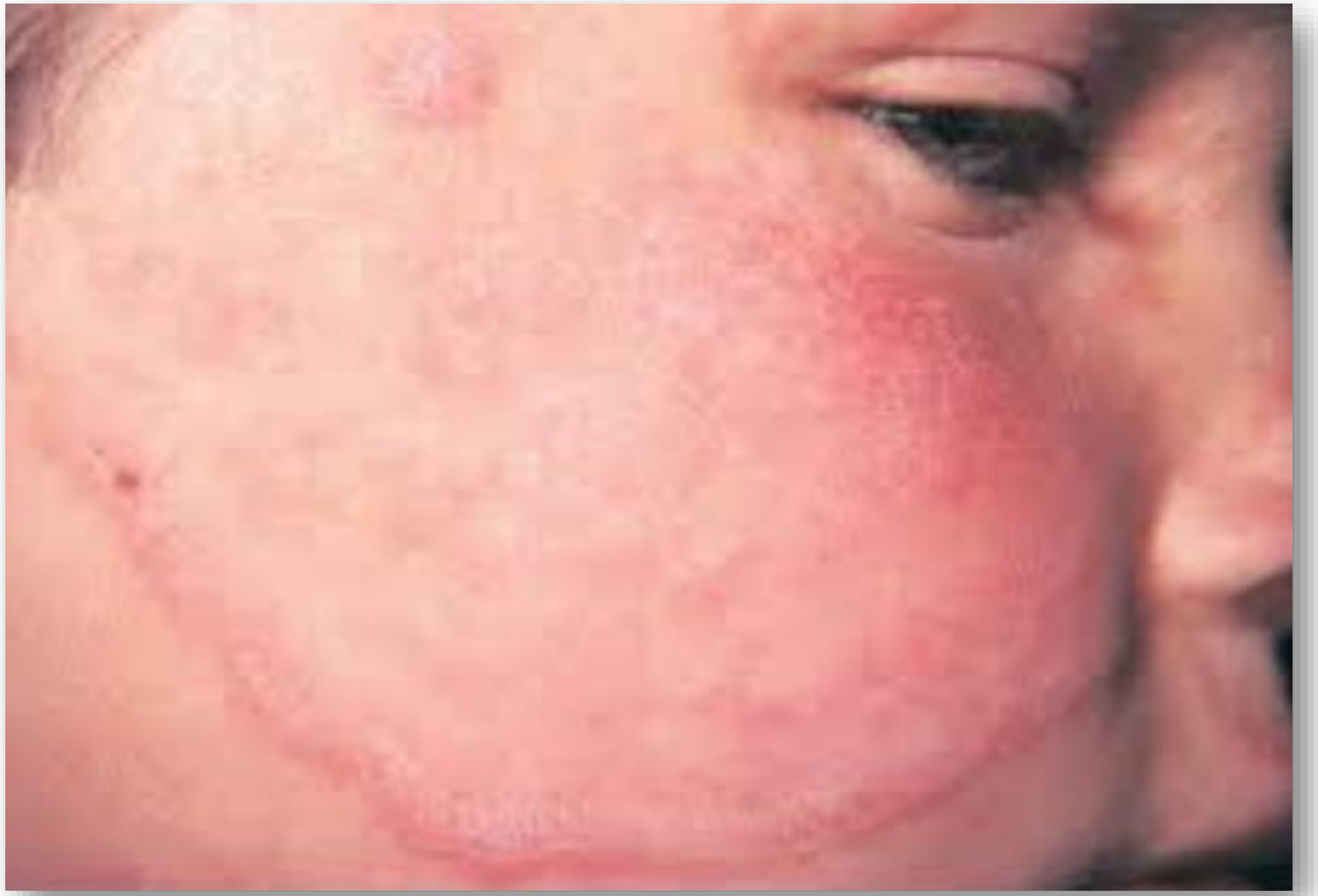


Tinea faciei

- Dermatophyte infection of the facial skin
- Not a common disease
- Erythematous area with active border
- **Treatment**: Topical antifungal



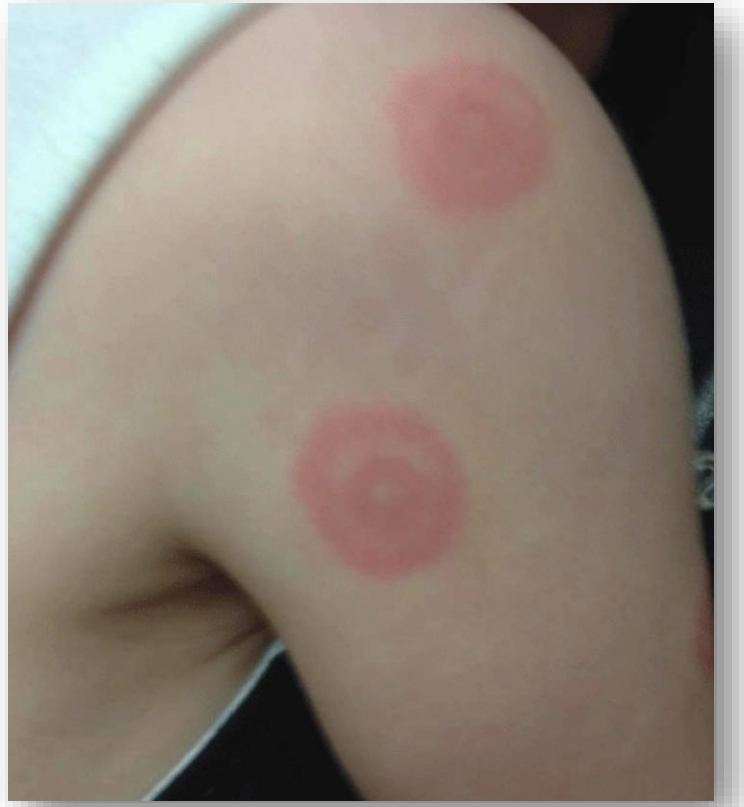
Tinea faciei



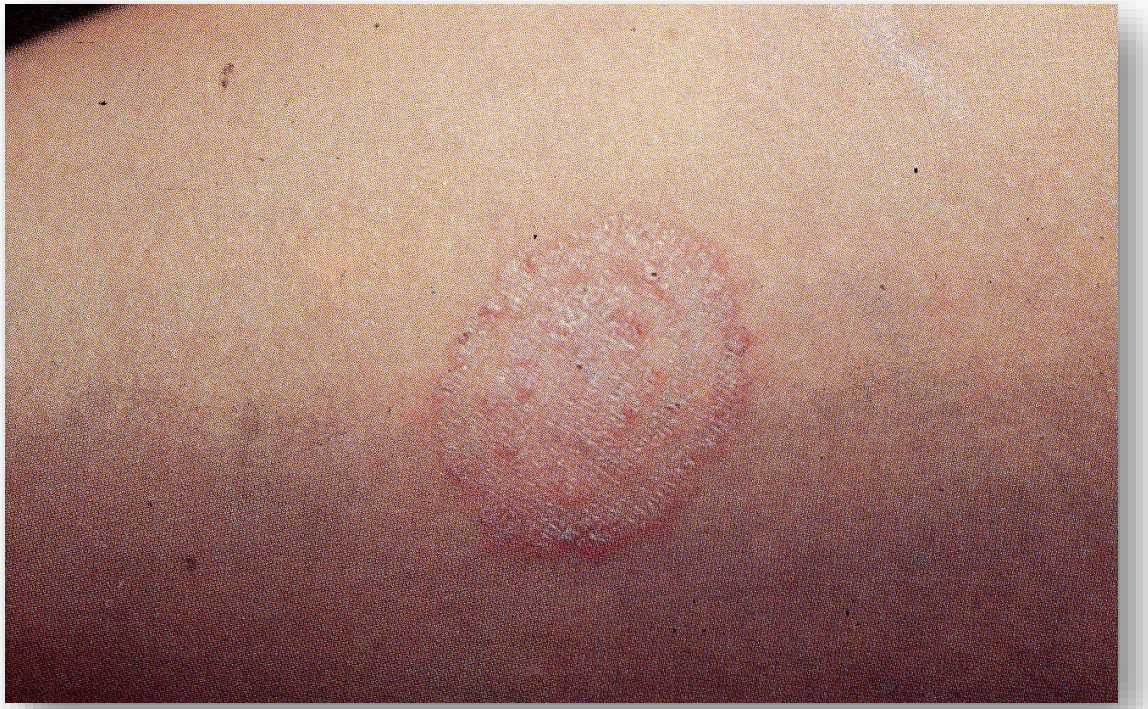
Tinea corporis

- Tinea circinate: Annular lesions with central clearing and elevated scaly border.
- **Treatment**: Topical antifungal
- **Differential diagnosis (5 أعرف لك حوالي):**

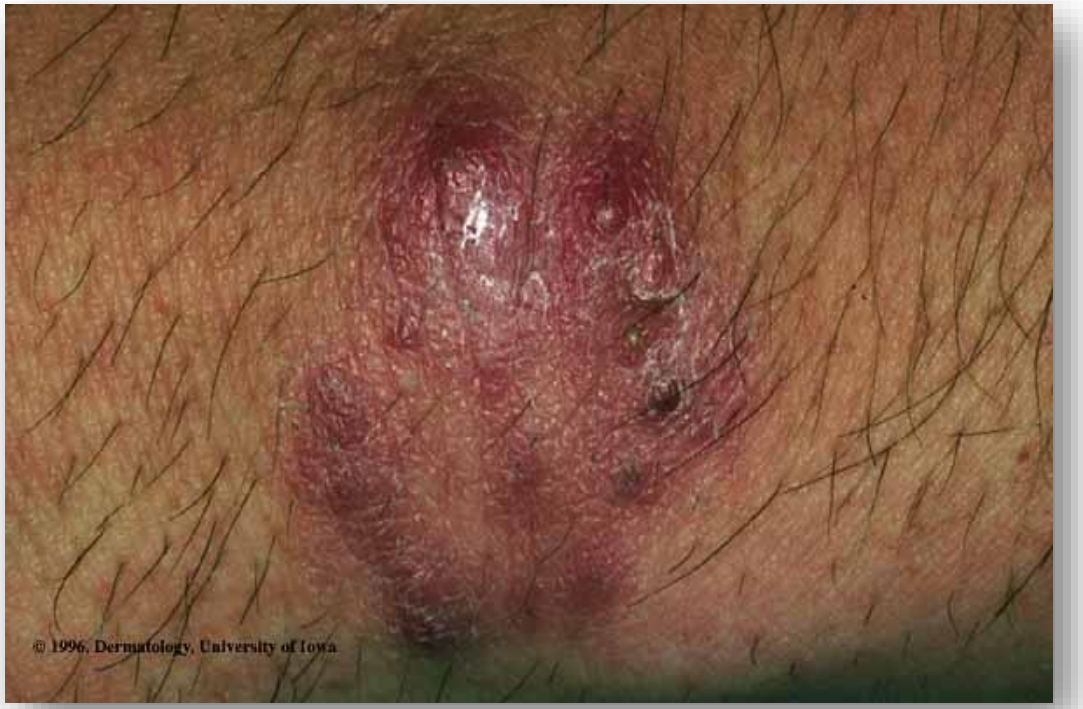
1. Psoriasis annular type.
2. Annular lichen planus.
3. Granuloma annulare.
4. Discoid eczema.
5. Leprosy.
6. Mycosis fundgoides.
7. Sarcoidosis.
8. Necrobiosis lipoidica Diabeticorum.
9. Bowens disease.



Tinea corporis



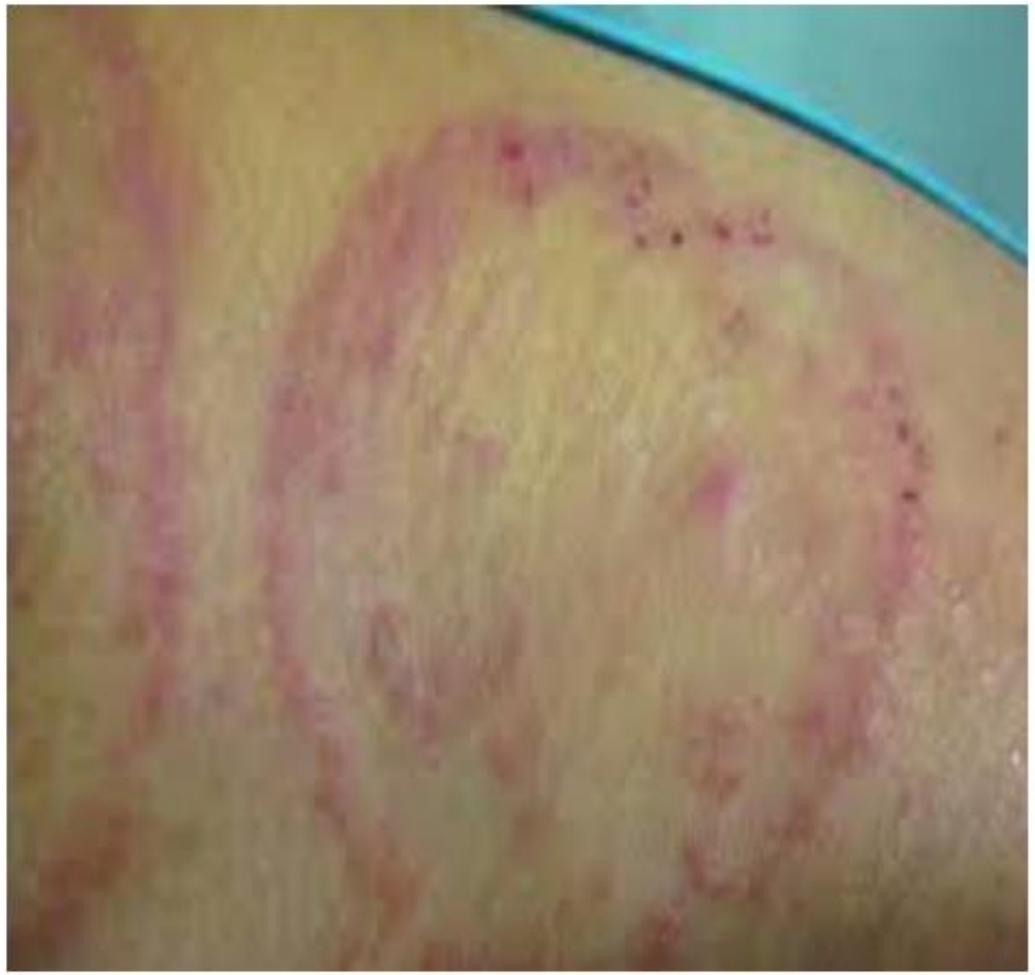
Annular lesion with expanding clear center and active margin.



Magicchi granuloma

Inflammatory type of Tinea corporis, deeply affecting the hair follicle,can be missed as bacterial infection, confirmed by biopsy or KOH preparation.

Tinea corporis

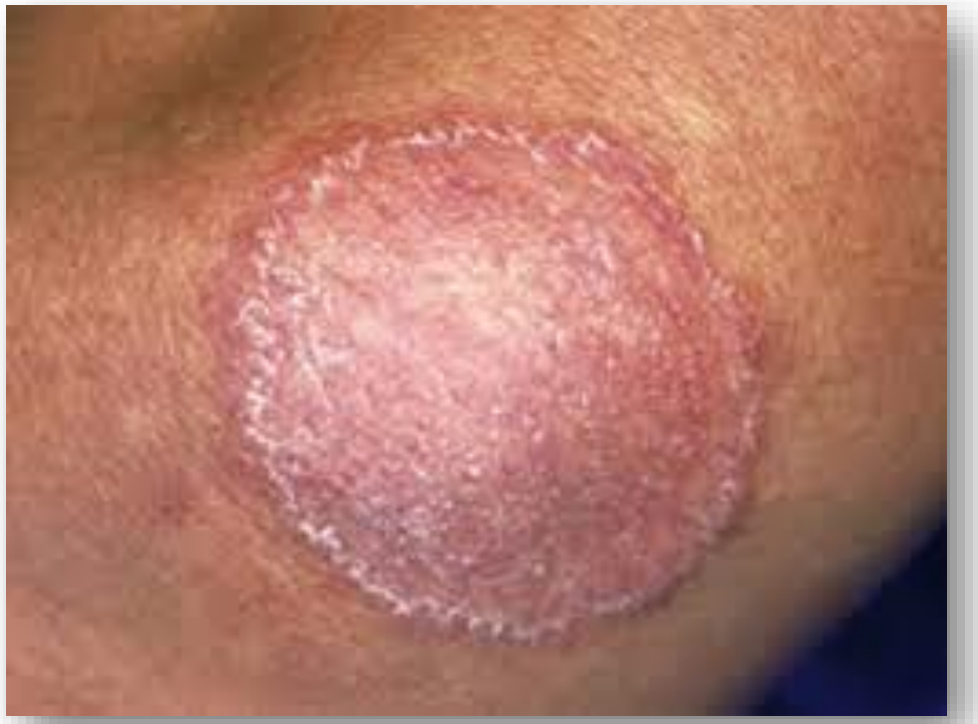


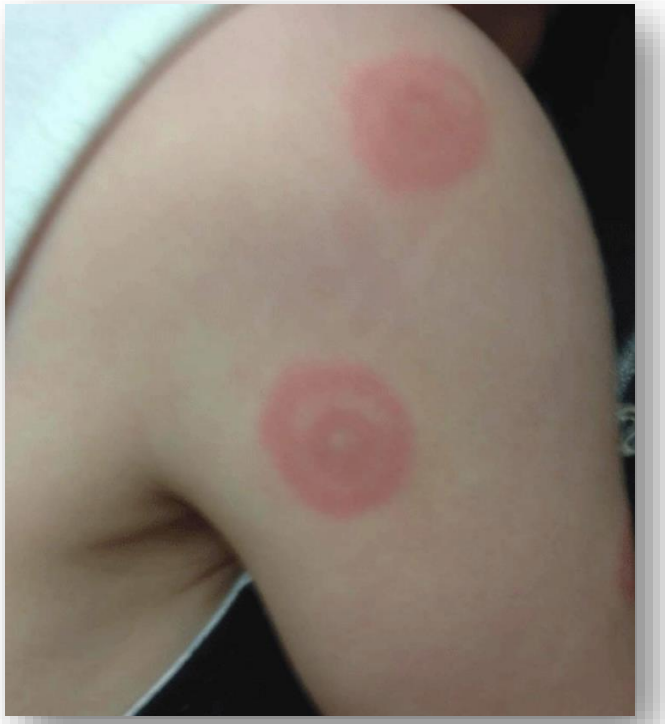
Well demarcated lesion
Tinea corporis

Tinea corporis

❖**What is the diagnosis ?**

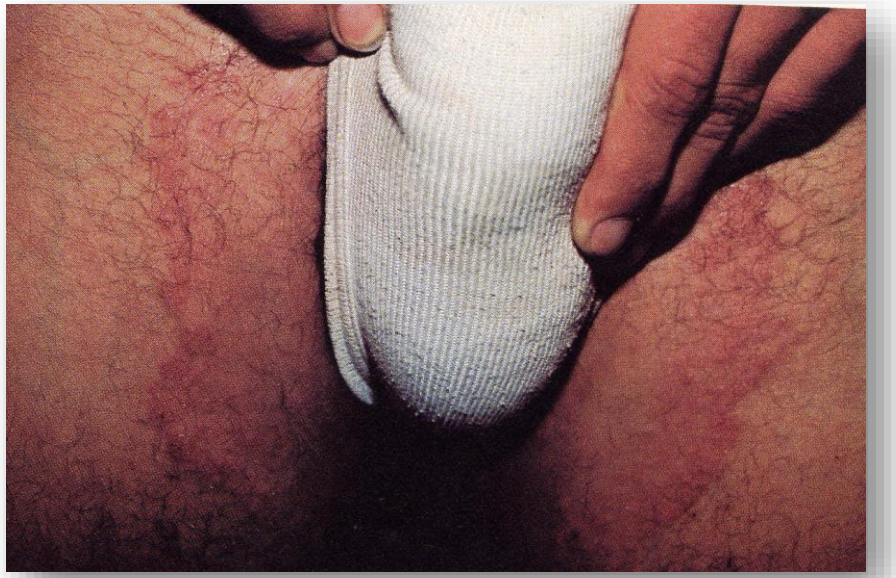
- Tinea corporis





Tinea cruris

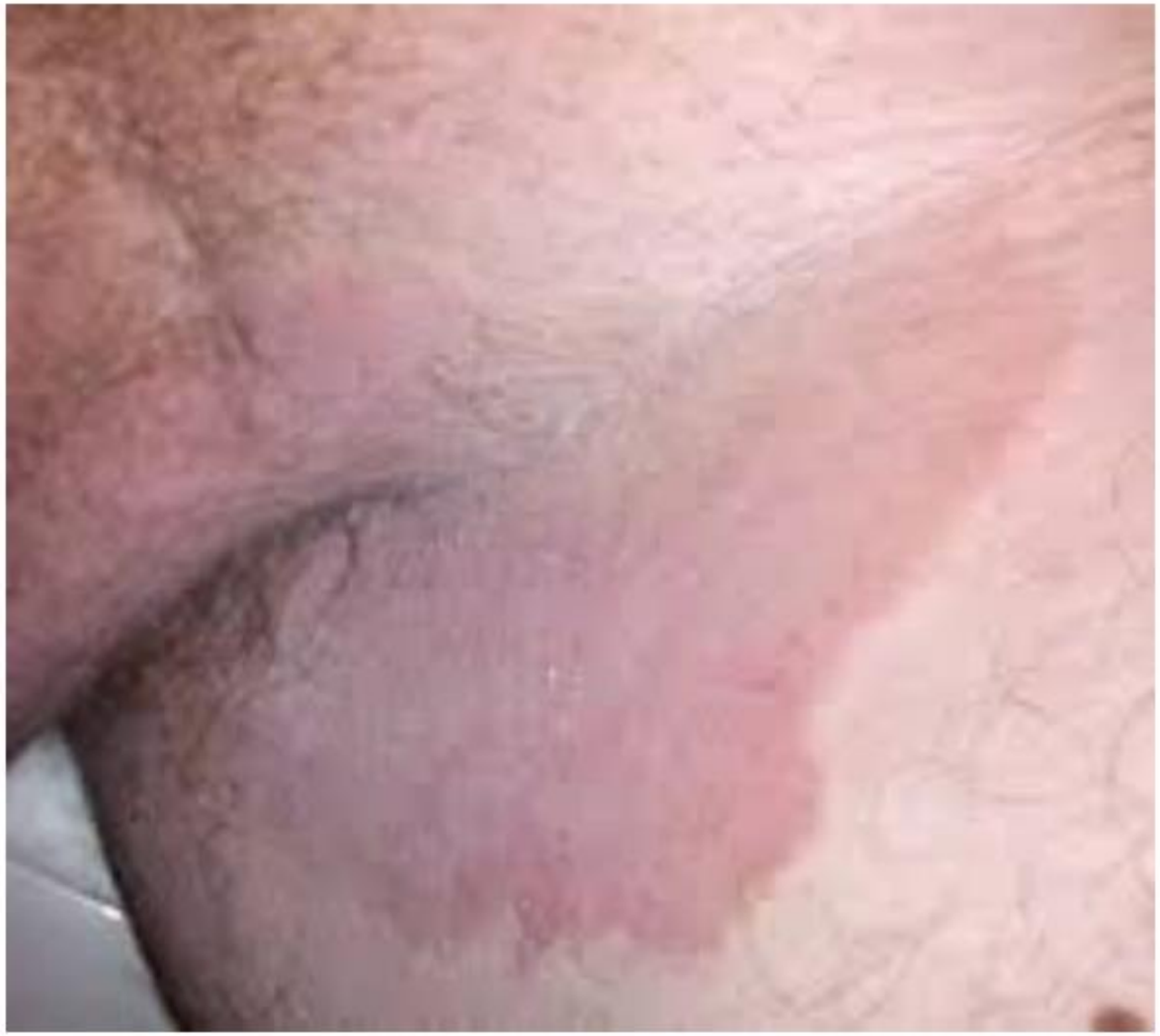
- Dermatophyte infection of the groin area
- Most common in men.
- **Treatment**: Topical antifungal
- **Differential diagnosis:**
  1. Candidiasis.
  2. Erythrasma.
  3. Psoriasis.
  4. Seborrheic Dermatitis



Erythematous areas in the groin with active scaly border.

Active = Gradually expanding

Tinea cruris



Tinea cruris

Tinea pedis

سنوات (1)

❖Most Common Tinea in ADULT ?

- Tinea peds

سنوات (1)

❖Mention the variants of tinea peds:

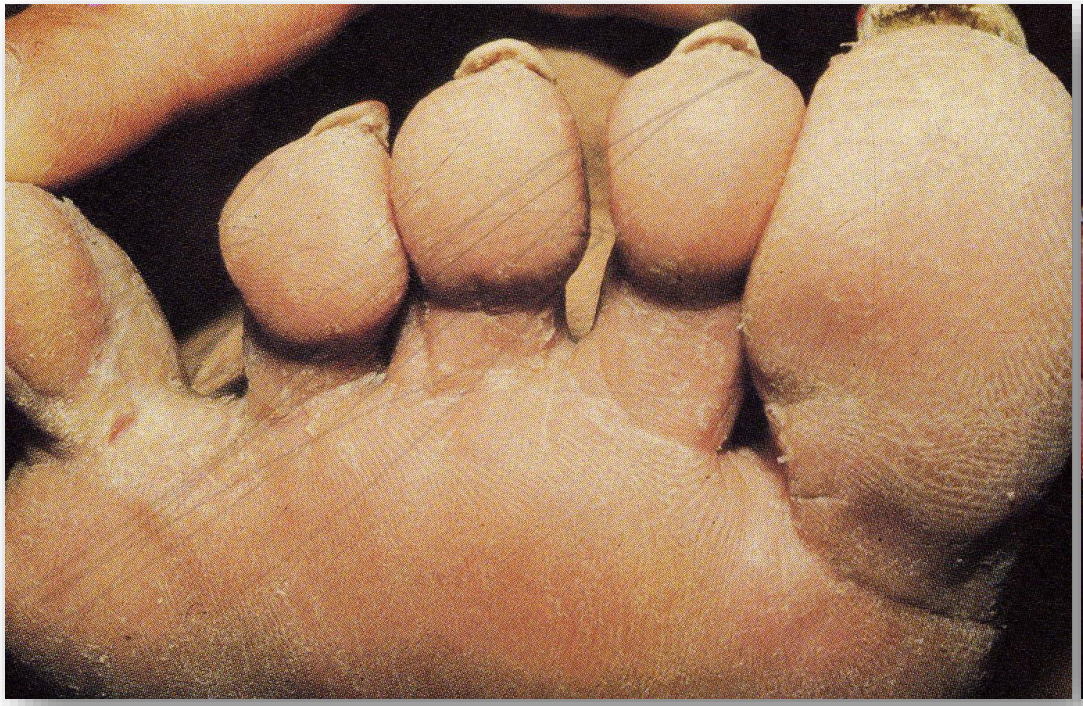
1. interdigital
2. moccasin
3. vesicular or bullus

❖**Treatment**: Topical antifungal

❖Differential diagnosis:

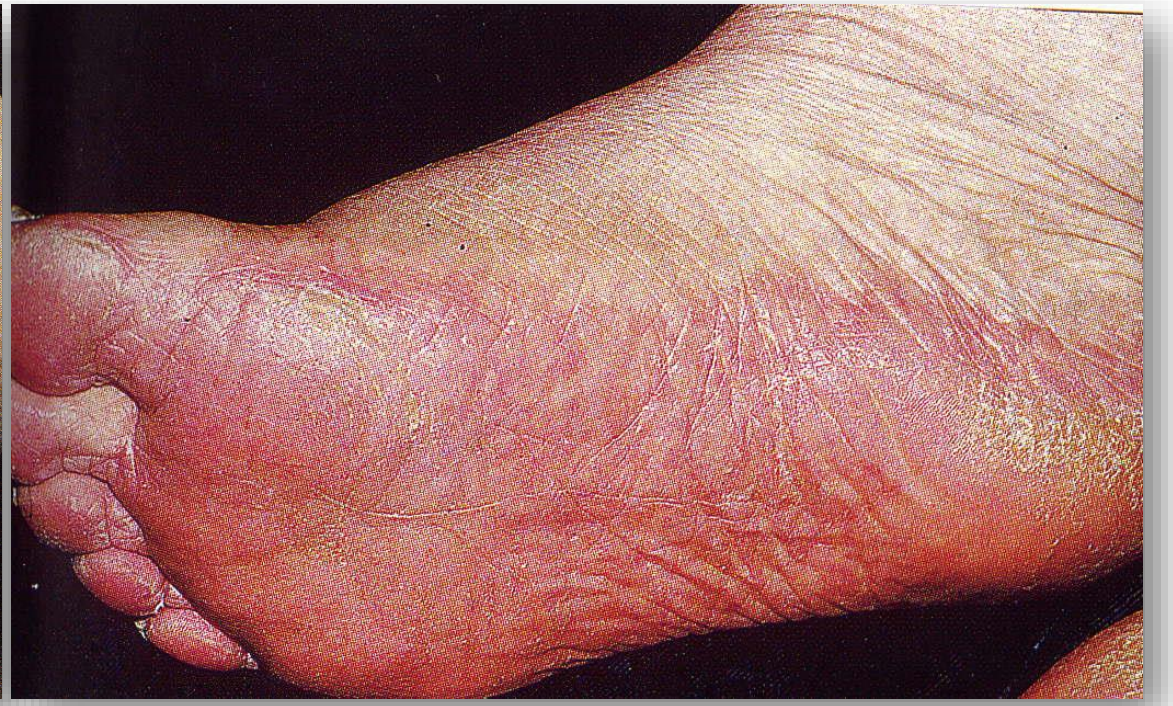
1. Psoriasis.
2. Eczema.
3. Pompholyx

Tinea pedis



Onychomycosis and Tinea Pedis

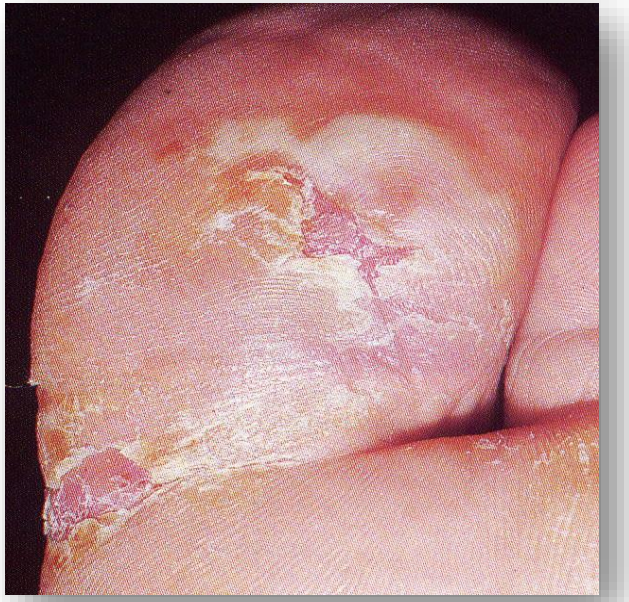
- **Interdigital type tinea pedis**: Scaling and maceration between the toes.
- Onychomycosis: there is nail involvement (subungual debri)



Moccasin type

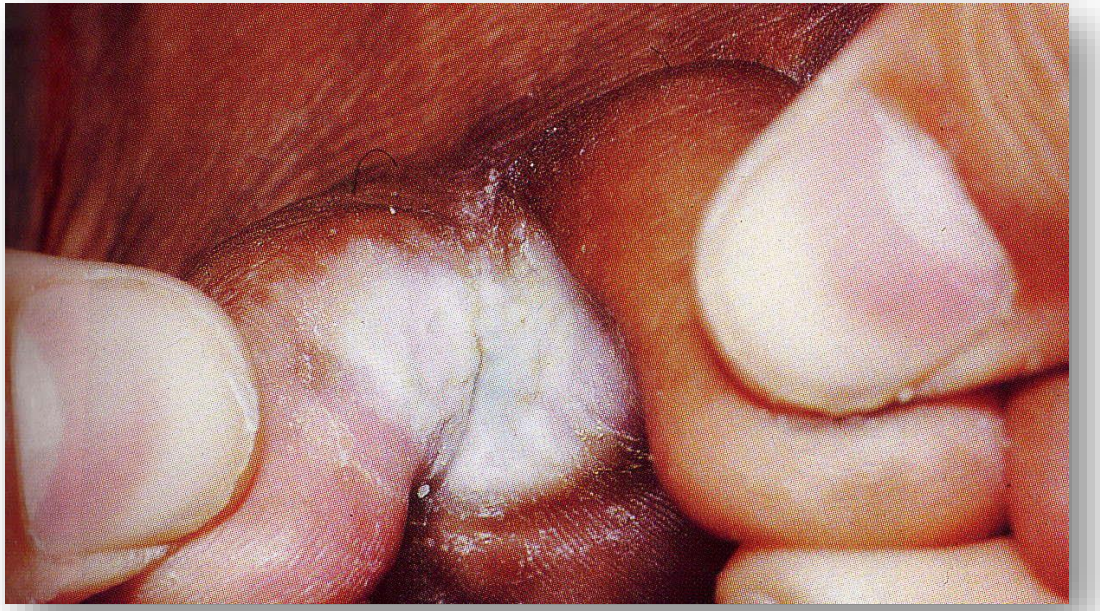
The whole plantar surface of the foot is involved with erythema and scaling.

Tinea pedis



Vesiculobullous type

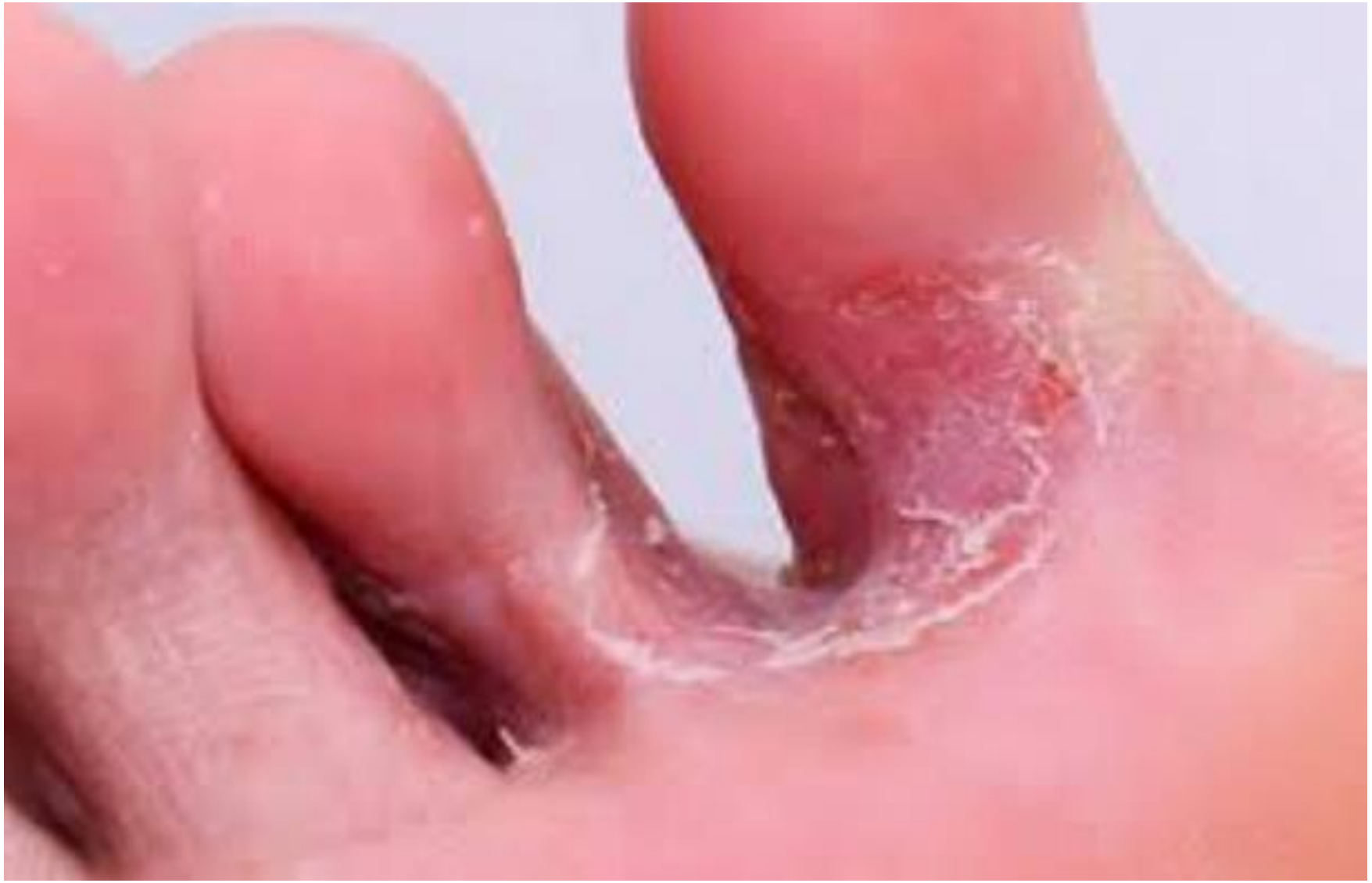
Localized area of blisters and vesicles.



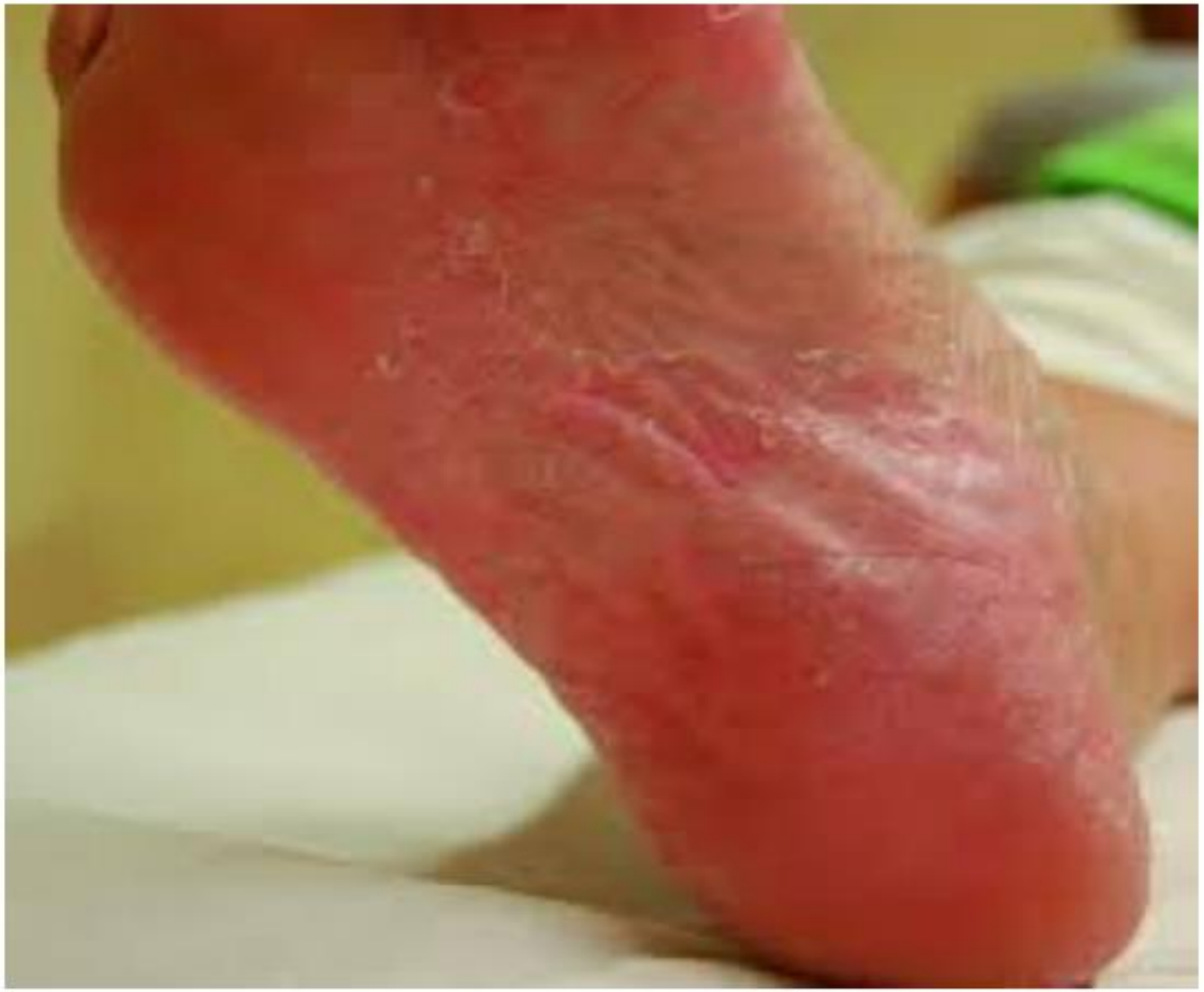
Interdigital type

Sometimes called Athlete's foot.

Tinea pedis



Tinea pedis



Tinea pedis with hyperkeratosis

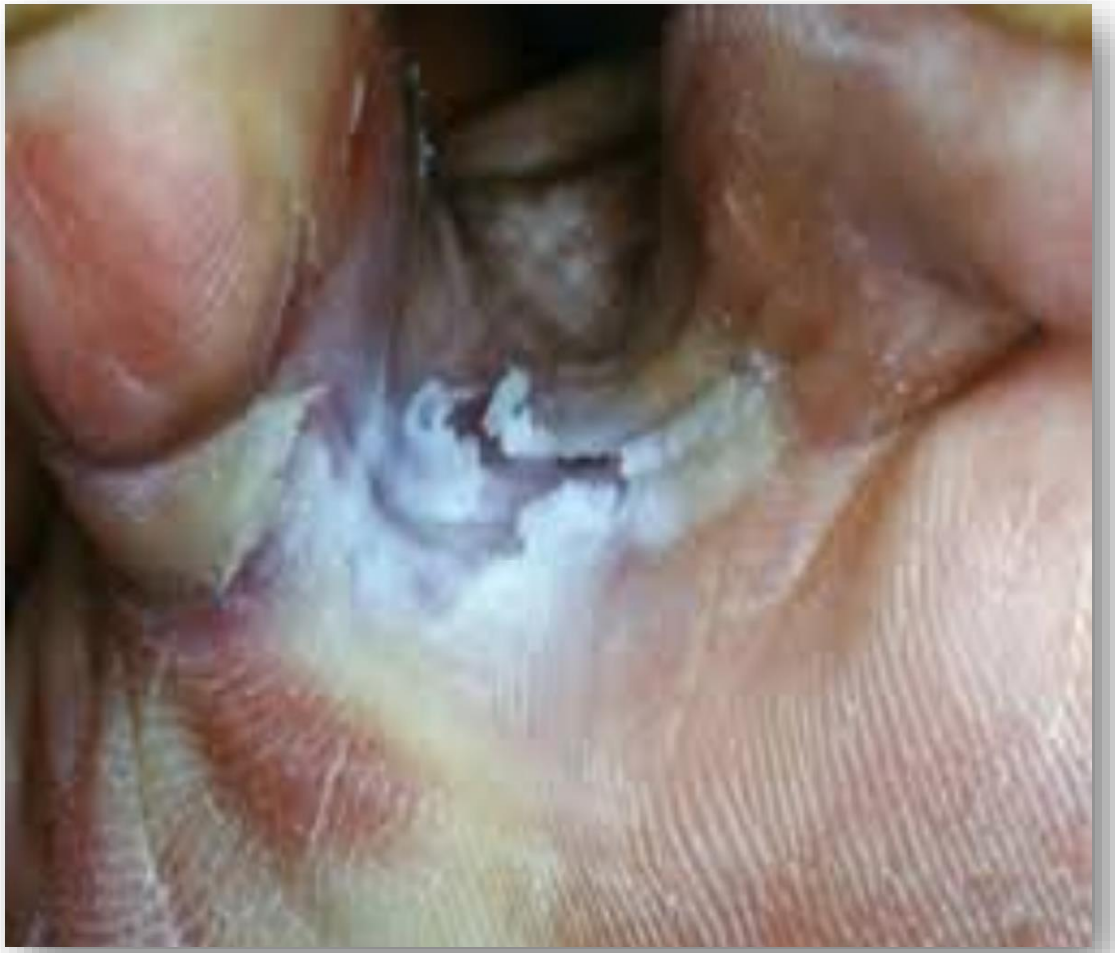
Tinea pedis

❖ **Mention 4 Ddx**:

1. Tinea peds
2. Psoriasis
3. Eczema
4. Pompholyx
5. Pseudomonas

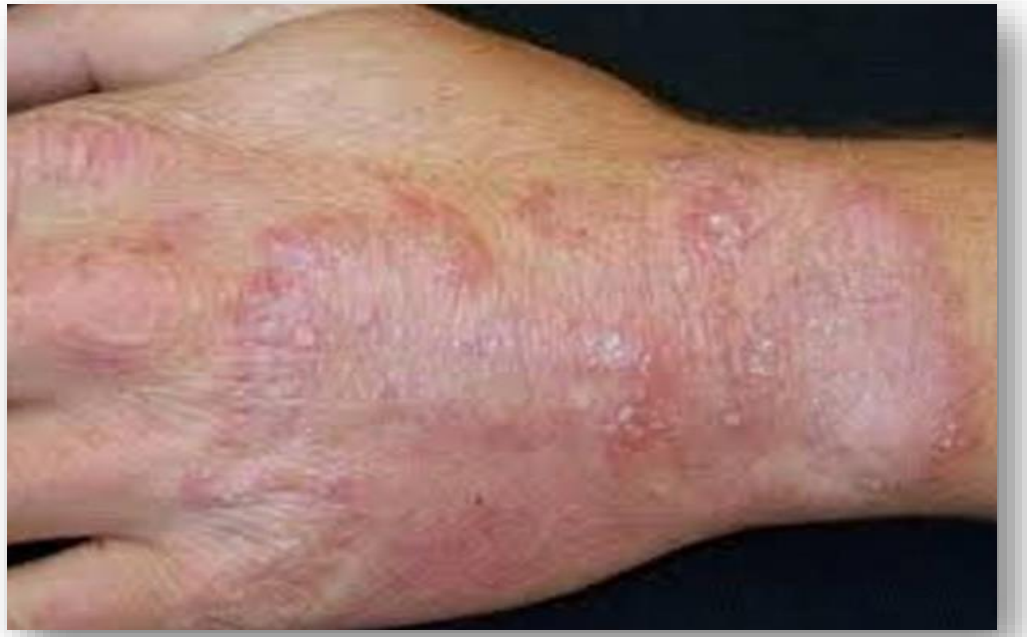
❖ **Mention 2 diagnostic tests**:

- KOH prep
- Wood's light



Tinea manum

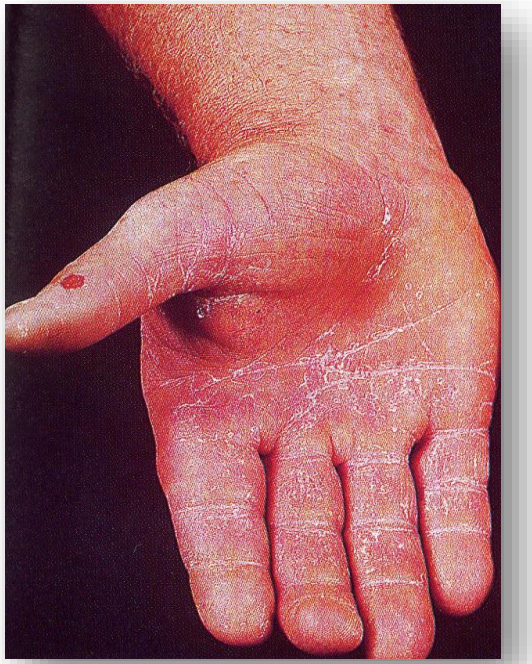
- Present with erythema and scaling involving usually one hand
- **Treatment**: Topical antifungal
- **Differential diagnosis:**
  - Hand eczema
  - Psoriasis
  - Xerosis



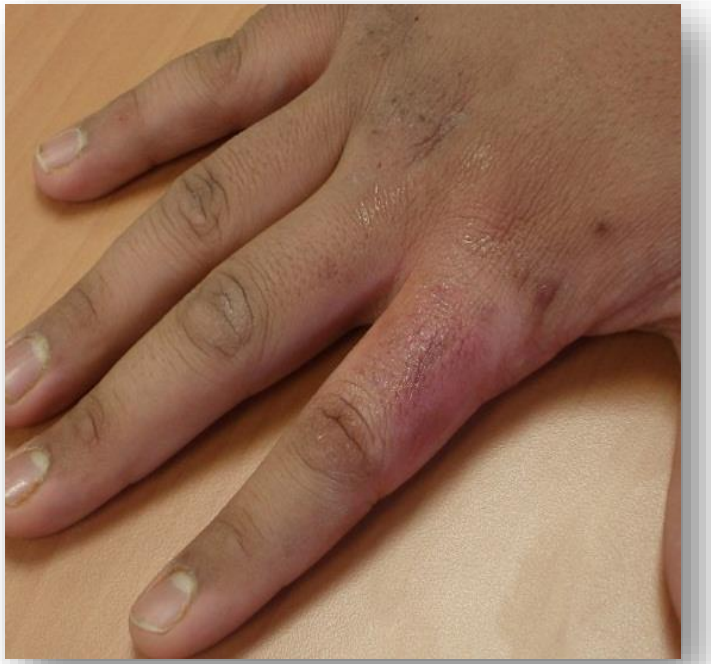
What is the diagnosis ? سنوات (3)

Tinea manum

Tinea manum

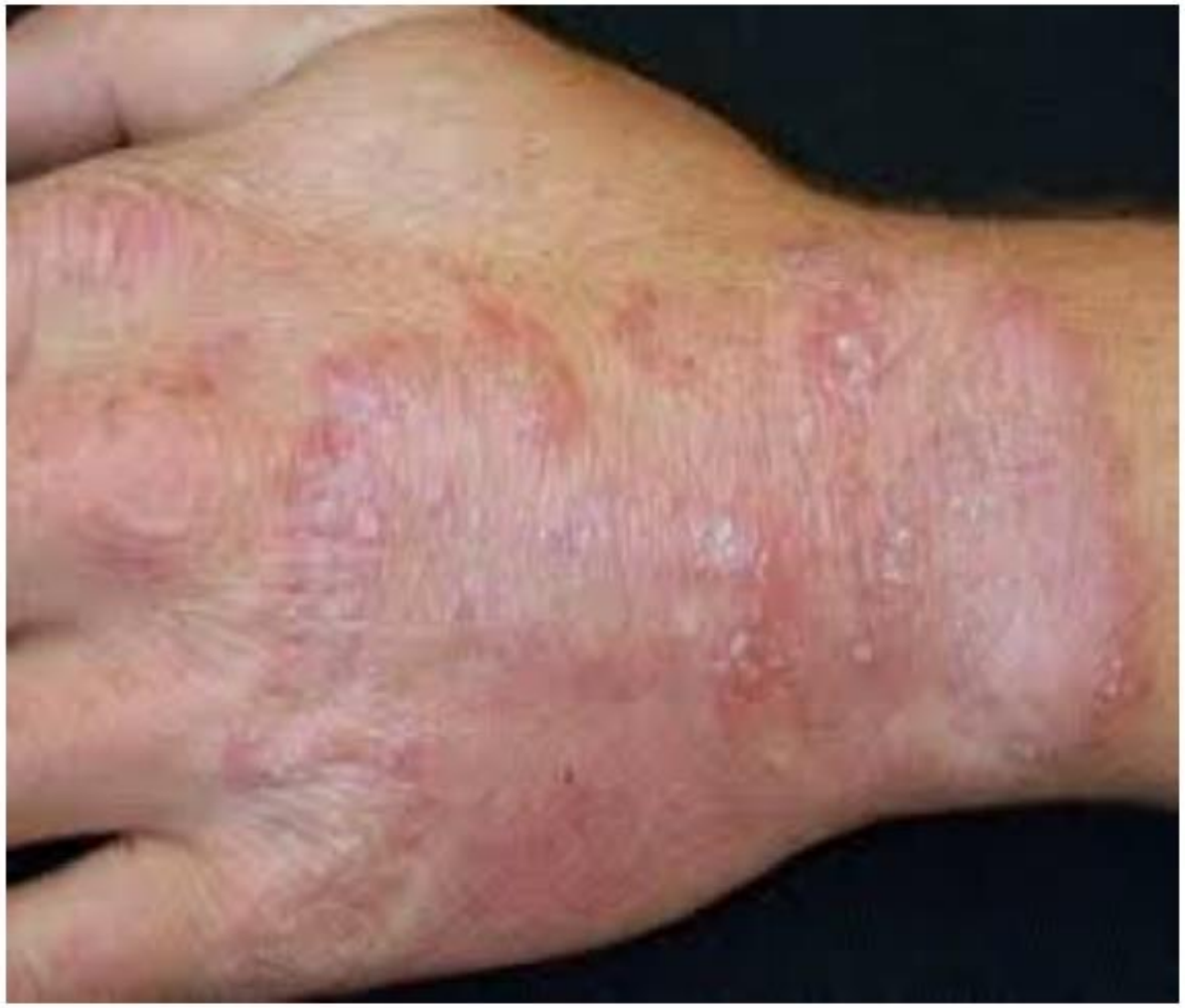


Erythematous hand with scaling, prominent creases of the hand.



Can affect the dorsal surface of the hand (this case is treated)

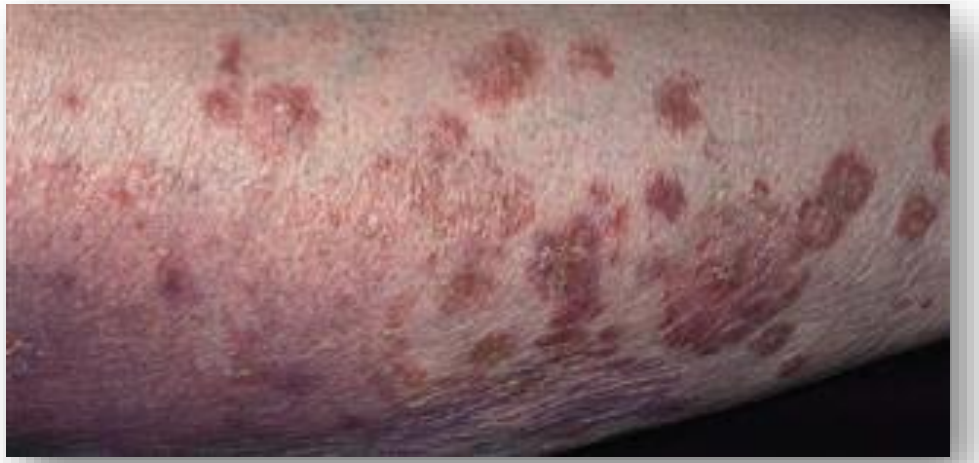
Tinea manum

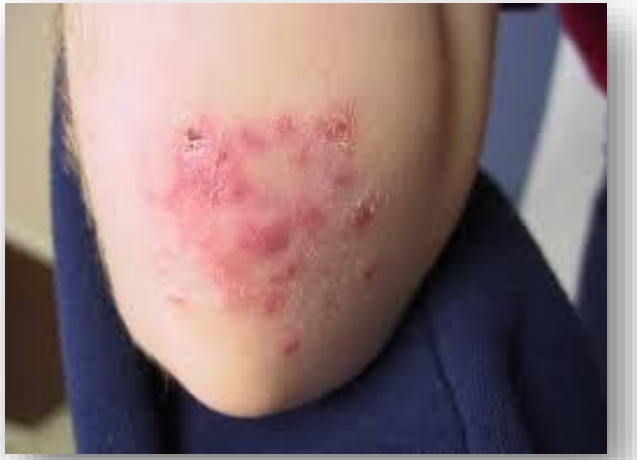


Tinea mania
Unilateral, well demarcated, not itchy

Tinea incognito

- Term used to describe a tinea infection modified by topical steroids.
- It is caused by prolonged use of topical steroids, sometimes prescribed as a result of incorrect diagnosis.
- Topical steroids suppress the local immune response and allow the fungus to grow easily.
- **Treatment**: Oral + topical antifungal





شرح

Onychomycosis

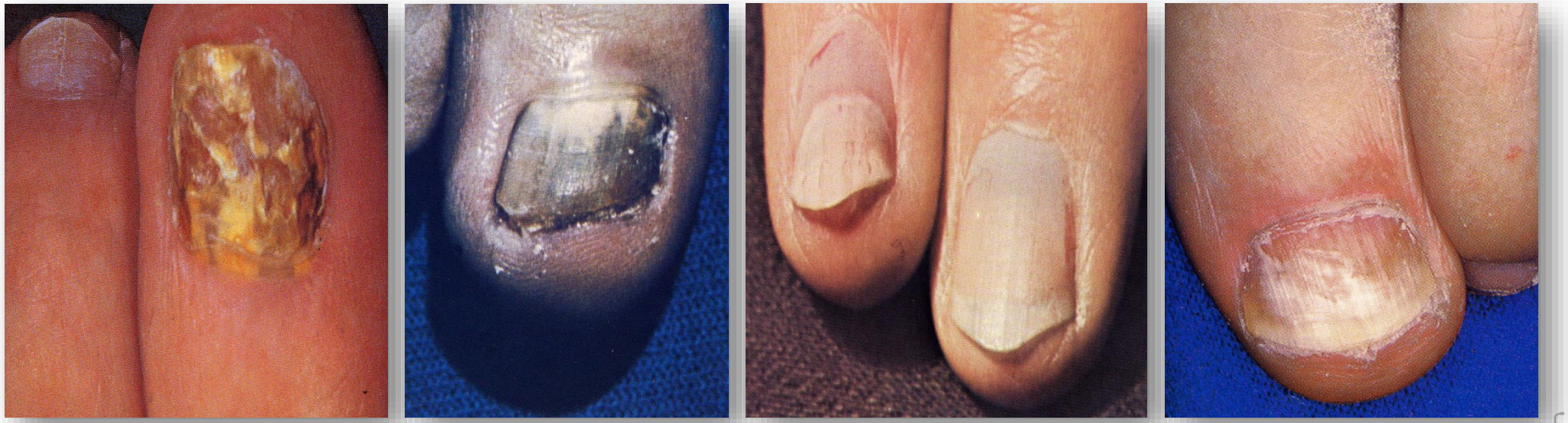
- Fungal infection of the nails
- Causes
  - Dermatophytes (Tinea unguium)
  - Candida
  - Scopulariopsis brevicaulis, Hendersonula toruloidea, Scytalidium hyalinum
- **Treatment**: Oral + topical antifungal
- **Differential diagnosis:**
  - Psoriasis
  - Lichen planus
  - Trauma
  - Eczema

سنوات (1)

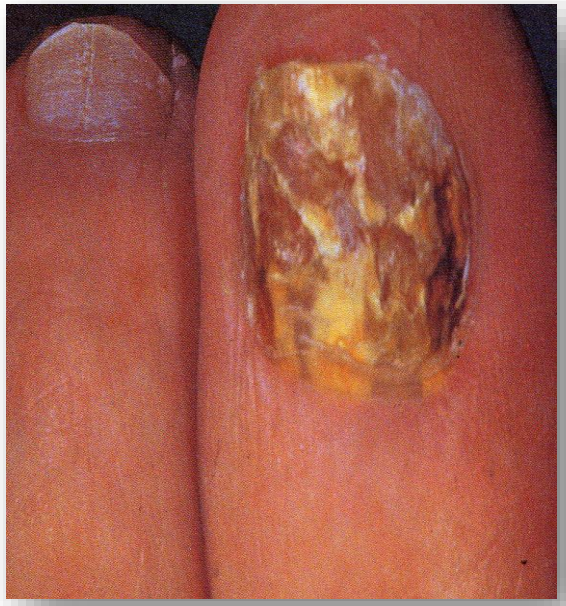
- Give 2 cases where superficial fungal infection treated with systemic antifungals
  - Nail involvement
  - Hair involvement

Onychomycosis presentations

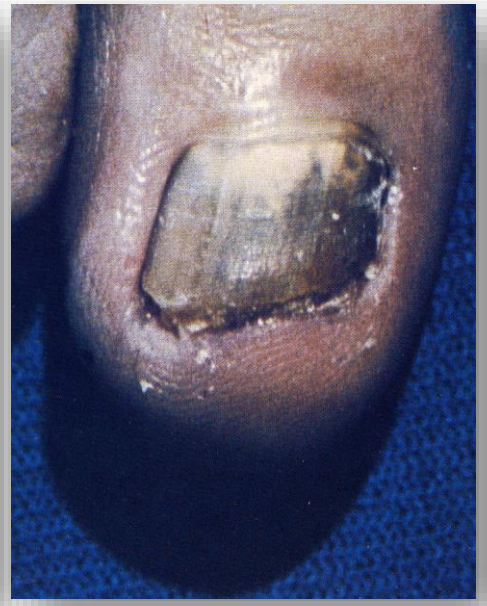
- White superficial Onychomycosis
- Distal subungual Onychomycosis
- Proximal subungual Onychomycosis
  - (mostly Immunocompromised pts and occurs in AIDS)
- Candida Onychomycosis, involves all nail plate



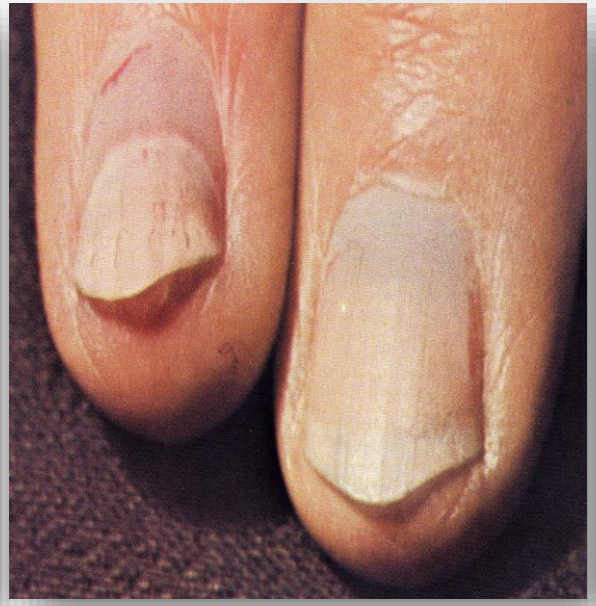
Onychomycosis presentations



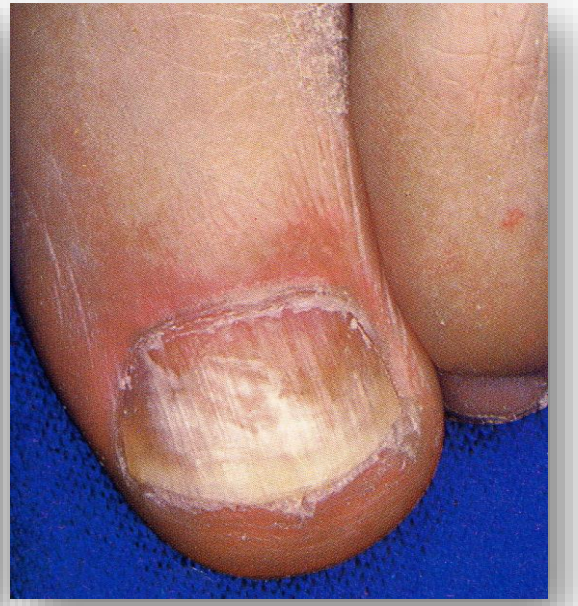
Thickened, discolored, dystrophic nail.



Thickening, hyperpigmentation. Starting from the distal lateral end of the nail and extending proximally.



Thickening, discoloration of the nail, starting distally then extending proximally.

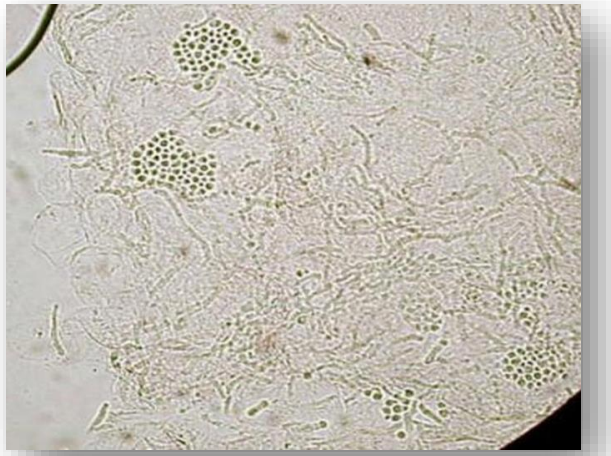


Thickening, discoloration of the nail, starting distally then extending proximally with Onycholysis.

Tinea versicolor (النُّخَالِيَّة المُبَرْقَشَة)

سنوات (6)

- **Caused by**: *Malassezia furfur* (*pityrosporum orbiculare*)
- **Description**: Scaly, mildly itchy, thin round to oval plaques usually on the back, chest and trunk.
- **Differential diagnosis:**
  - Seborrheic Dermatitis
  - Vitiligo
  - Postinflammatory hypopigmentation
- **Diagnosis**:
  - KOH preparation: meat balls and spaghetti
  - Woods light: golden yellow florescence
- **Treatment**:
  - Topical: -azoles, Selenium sulfide shampoo
  - Systemic: Fluconazole, Itraconazole



Tinea versicolor

سنوات (4)

- **What is the cause of this lesion**
  - *Malassezia furfur* (*pityrosporum orbiculare*)

سنوات (1)

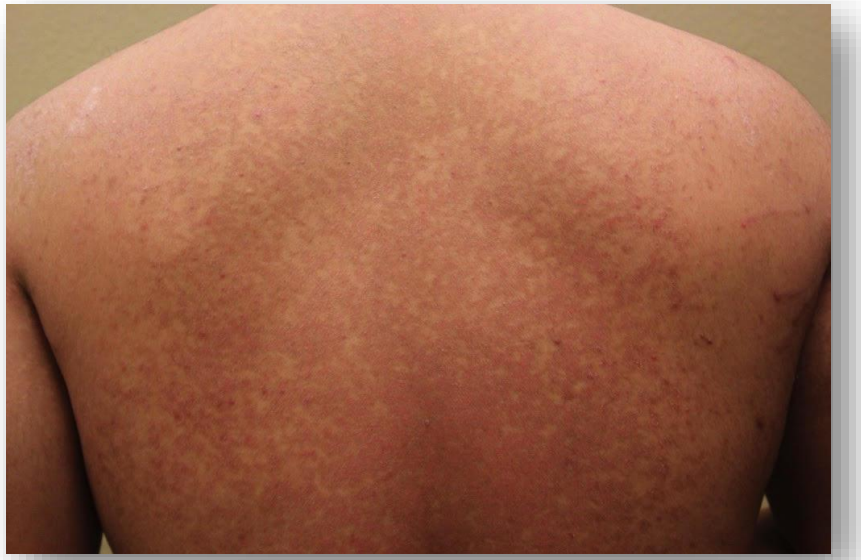
- **What is the best diagnostic test for**
  - Wood's lamp

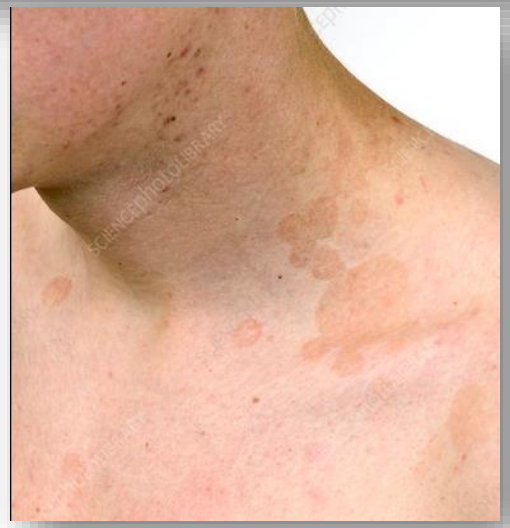
سنوات (1)

- **Wood's light of tinea versicolor**
  - Golden yellow

سنوات (1)

- **Why tinea versicolor cause hypopigmentation?**
  - This fungus produce azeliac acid which is tyrosinase inhibitor and this led to depigmentation





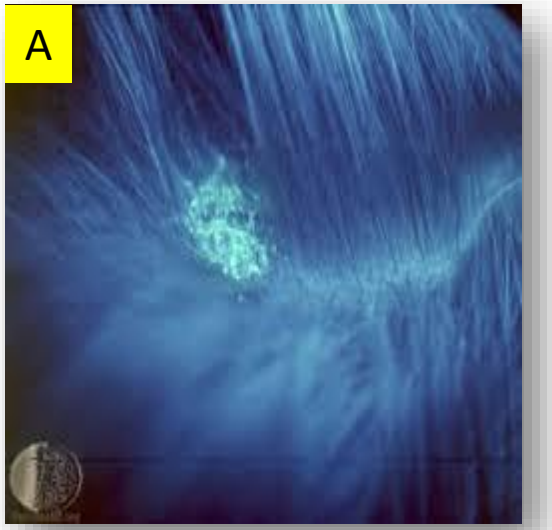
Describe; what is the diagnosis

❖**Describe**:

A. Green lesion on wood's lamb
B. Gold yellow lesion on the wood's lamb

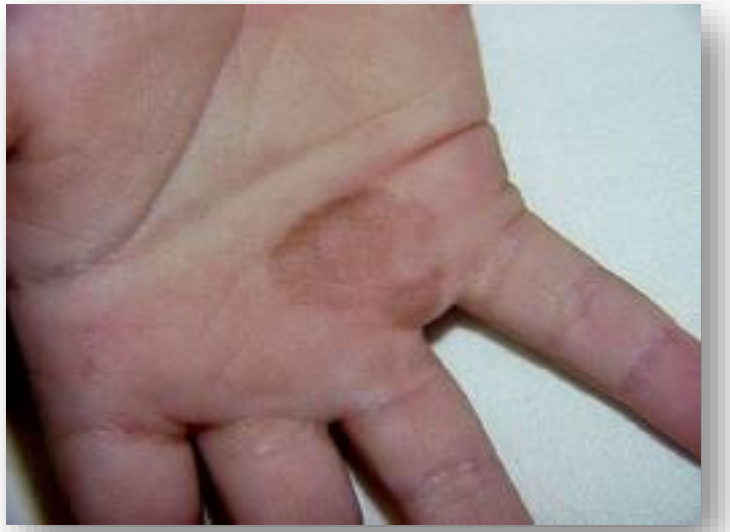
❖**Diagnosis**:

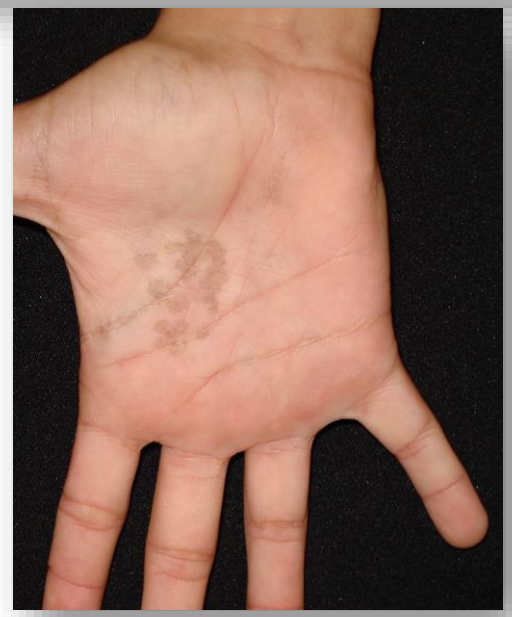
A. Tinea capitis
B. Tinea versicolor



Tinea nigra

- Single sharply marginated brown to gray to green macule or patch that can be velvety or have mild scale. No pruritus
- Common on palms, can appear on sole, neck and trunk.
- **Differential diagnosis**:
  - Pigmented lesions
  - Postinflammatory hyperpigmentation
- **Treatment**:
  - Keratolytic (Whitfield ointment; 6% Benzoic acid +3%S.A)
  - Topical antifungal
  - No need for systemic treatment





Candidiasis

- Causative agent: *candida albicans*; it's a normal flora
- It becomes a pathogen with:
  - Increased moisture
  - Administration of Antibiotics
  - Steroids
  - Pregnancy
  - DM and other debilitated conditions
- Mention 5 Mucocutaneus clinical presentation of Candidiasis
  1. Oral Candidiasis (oral thrush and perleche dermatitis)
  2. Vaginal Candidiasis
  3. Balanitis and balanoposthitis
  4. Erosio interdigitalis blastomycetic
  5. Chronic Paronychia
  6. Candidiasis intertrigo: Napkin, Submamary, Balanitis
  7. Neonatal Candidiasis
  8. Chronic mucocutaneous candidiasis

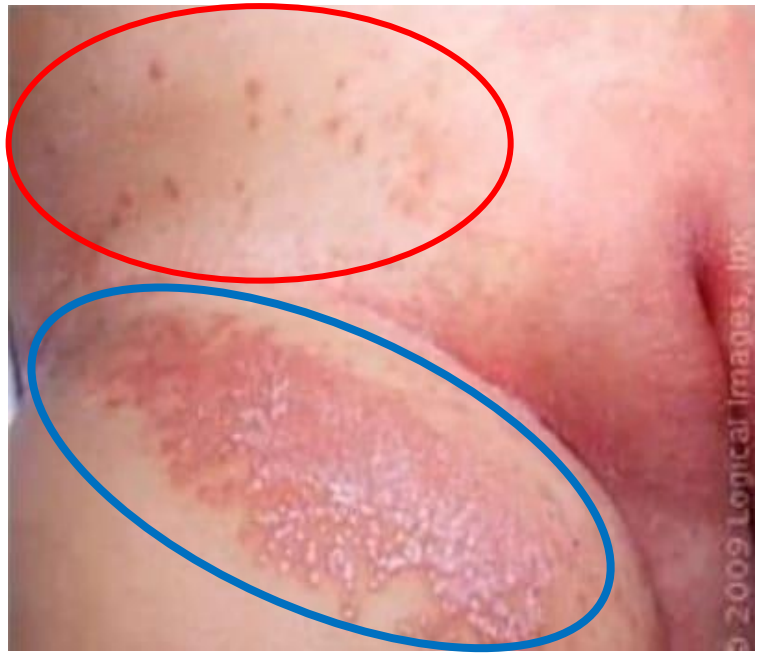
Candidiasis

سنوات (1)

- **What is the cause of angular cheilitis**: *Candida albicans*
- **Diagnosis**:
  - KOH preparation- budding yeast
  - Culture on Sabourauds medium- 4 days (creamy gray mat).
- **Treatment**:
  - Topical antifungal
  - For chronic mucocutaneous candidiasis long term imidazole is the Rx. of choice

Napkin Candidiasis

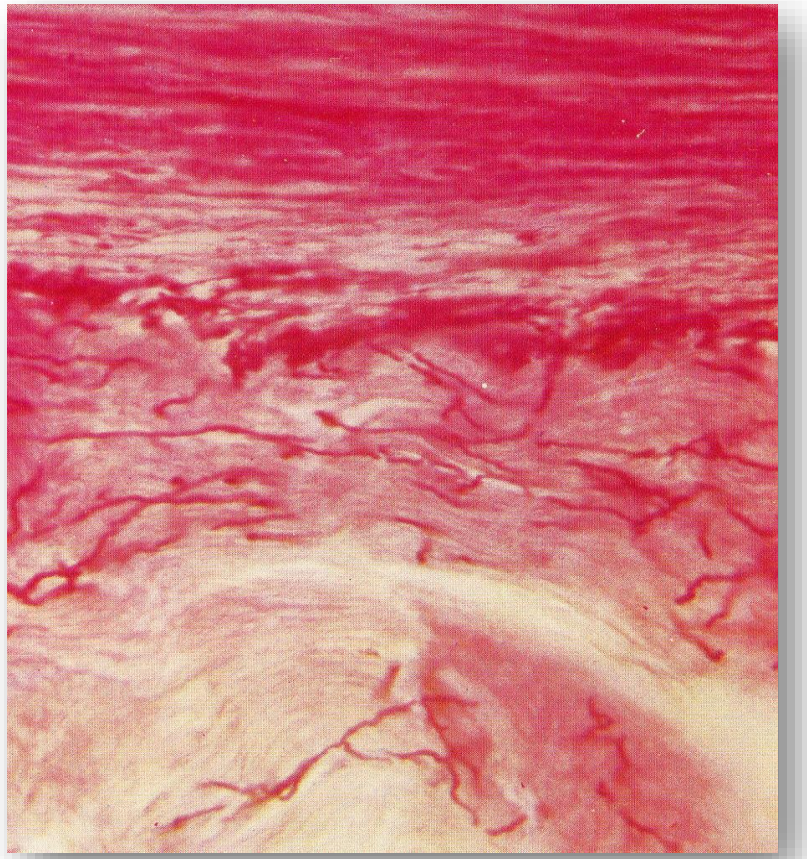
- Erythematous skin rash in the napkin area
- **Differential diagnosis (Napkin dermatitis)**:
  1. Seborrheic dermatitis.
  2. Atopic dermatitis.
  3. Contact dermatitis from diapers.
  4. Napkin candidiasis.
- **Treatment**:
  - Topical antifungal (Miconazole or Clotrimazole cream)



Erythematous scaly skin rash in the napkin area.
Satellite lesions (Papules and Pustules) away from the erythematous rash (Specific for Candida).

Diagnosis of superficial fungal infections

- KOH preparation
- Woods light exam
- Skin biopsy: to differentiate them from other dermatosis (PAS stain).
- Nail clipping in Onychomycosis can be prepared with PAS stain to identify hyphae.
- Culture on Sabourauds or dermatophyte test medium



Nail clipping stained with PAS stain showing the fungal Hyphae (Red lines)

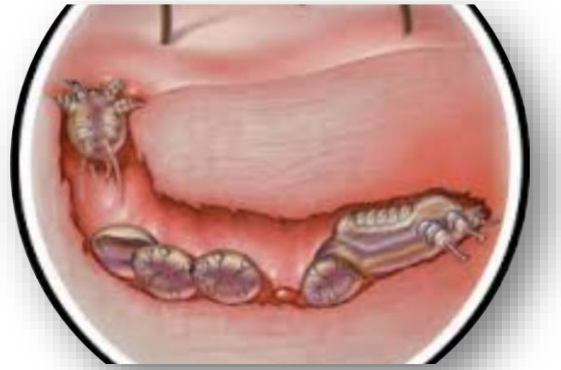
Treatment of superficial fungal infections

- **General measures**: avoid moisture, keep the area dry and clean.
- **Topical Rx**:
  - Miconazole, Clotrimazole, Econazole, Ketoconazole, Whitfield ointment. (6%benzoic acid + 3% SA).
  - Topical Rx. is not enough in cases of hair and nail involvement and in Tinea incognita.
  - Must be given for an enough period of time (3-6 Wks.) according to the site.
- **Systemic Rx**:
  - Griseofulvin, Ketoconazole, Itraconazole, Fluconazole and Terbinafine.
  - t must also be given for an enough period of time (2Wks.-6 to 9 Mo.) according to the site and the used drug.
- **Pulse Rx**:
  - e.g., Itraconazole (1Wk. Rx and 3Wks. Off Rx. For the recommended period, Fingernails need 2 pulses, Toenails need 3 pulses).


Infestation

Scabies

Scabies



سنوات (1) ❖**Is scabies a highly contagious disease ?**

- NO

سنوات (5) ❖**What is the primary lesion of scabies**

- Burrow

سنوات (3) ❖**Define burrow**

- Slightly elevated, grayish, tortuous line in the skin ended by papule.

سنوات (4) ❖**What is the cause of scabies**

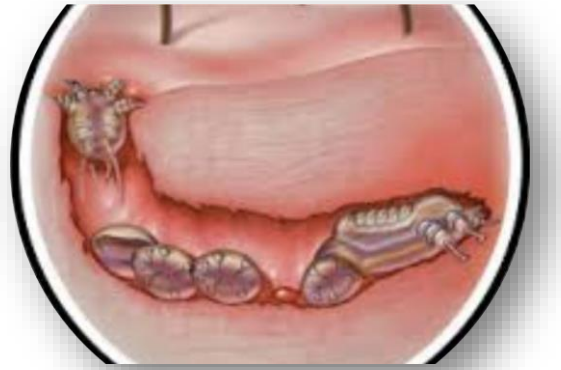
- Sarcoptes scabiei var. hominis

❖**Transmission**: skin-to-skin contact

❖**Complications**: Secondary infection

Scabies

❖Scabies itching



- Scabies causes a very itchy rash
- Itch is characteristically more severe at night, disturbing sleep
- It affects the trunk and limbs, sparing the scalp

❖Burrows

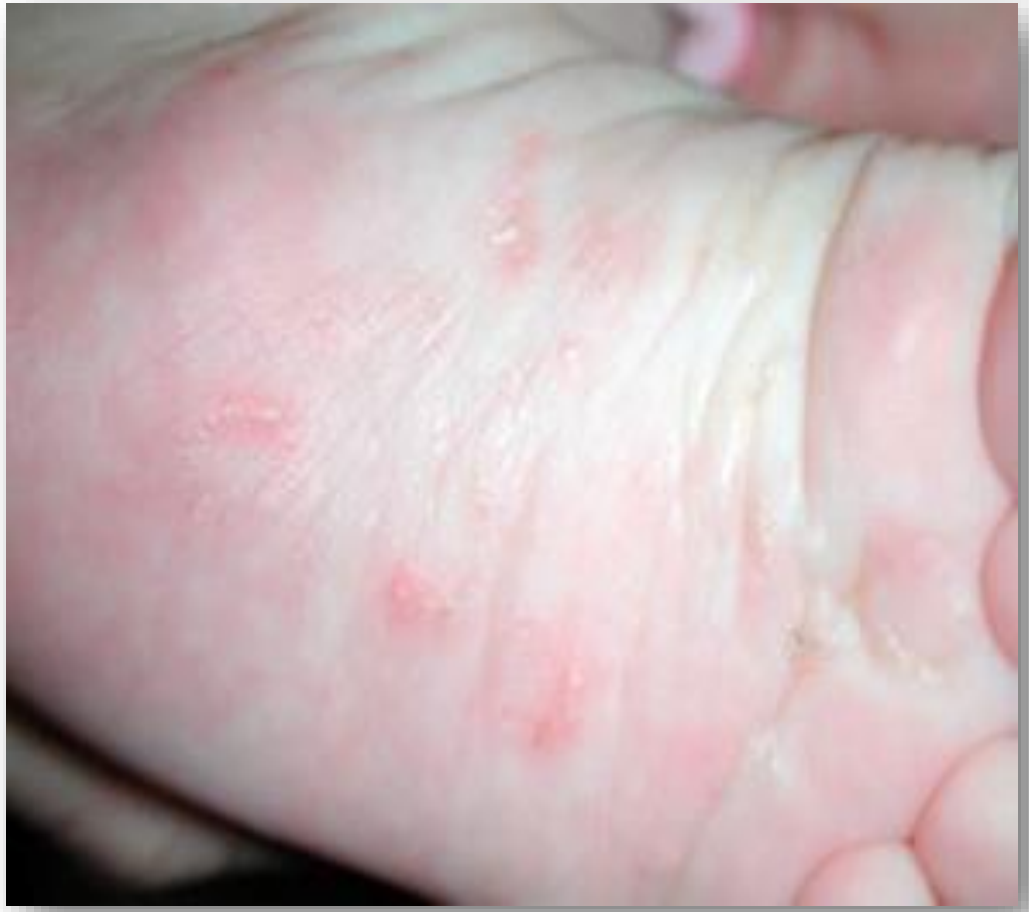
- Scabies burrows appear as 0.5–1.5 cm grey irregular tracks in the web spaces between the fingers, on the palms and wrists sparing the **face** and **back** (due to sebaceous glands activity)

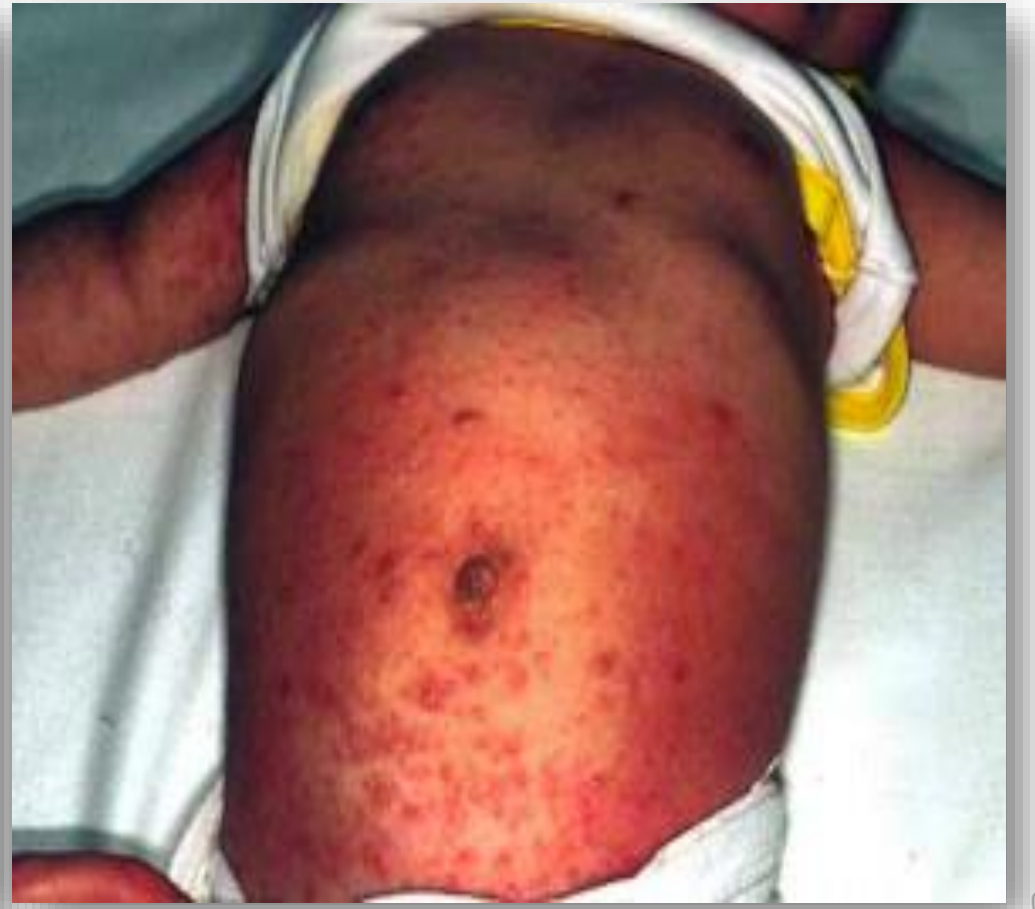
❖Generalized rash

- Erythematous papules on the trunk and limbs, often follicular
- Acro-pustulosis (sterile pustules on palms and soles) in infants
- Rare involvement of **face**, **back** and **scalp** in adults, but it can affect the face in children

Acro-pustulosis in infants

- Papules or nodules in the armpits, groins, buttocks, scrotum and along the shaft of the penis.

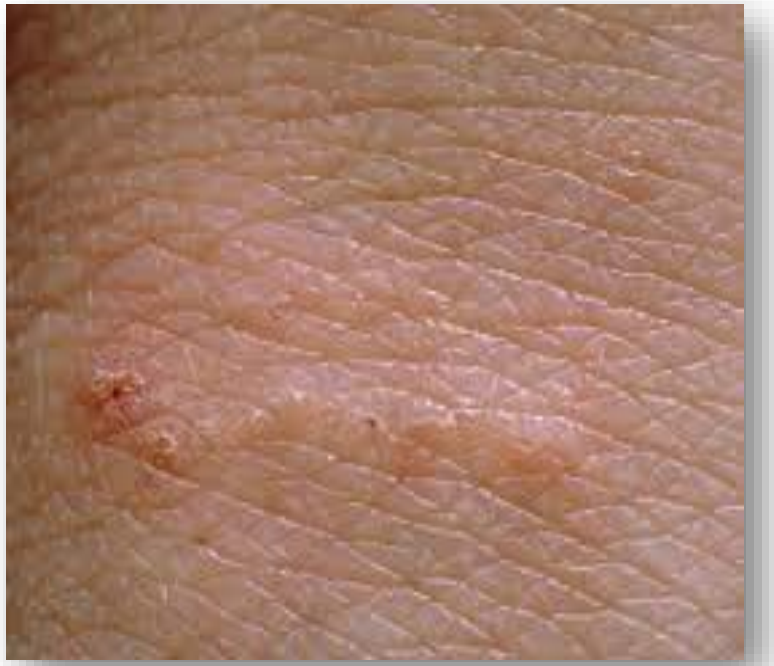




Scabies

سنوات (2)

- **Describe the lesion:**
  - Slightly elevated, grayish, tortuous line in the skin ended by papule.
- **One line of treatment**
  - 25% benzyl benzoate lotion, applied daily every 12 hours for 3 days for adults
- **Which treatment could be used in children?**
  - Crotamitone
  - Crotophile
- **What is the causative organism ?**
  - Sarcoptes scabiei var. hominis



What is this called ?

Burrow

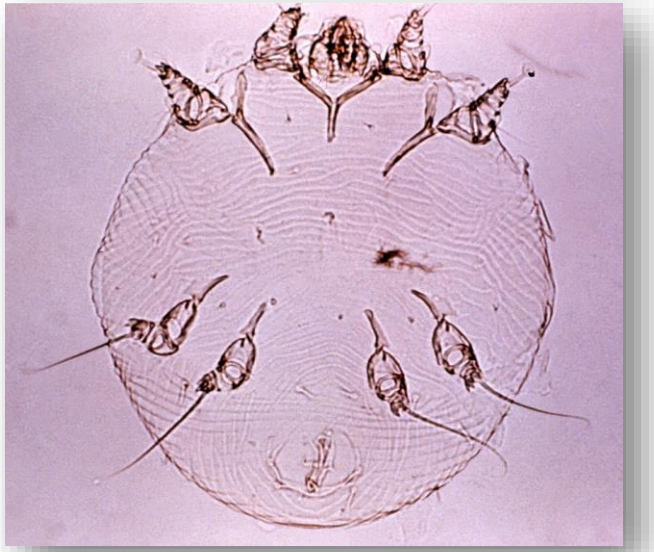
سنوات (1)

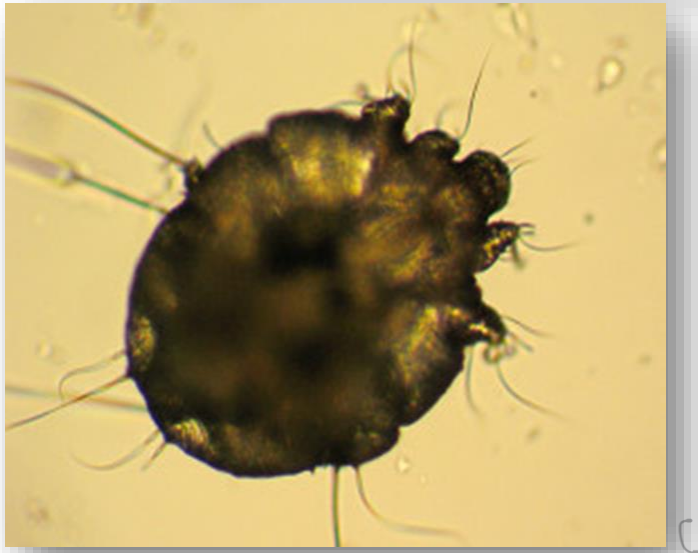
Scabies

- **What is this mite ?**
  - Sarcoptes scabiei var. hominis

سنوات (2)

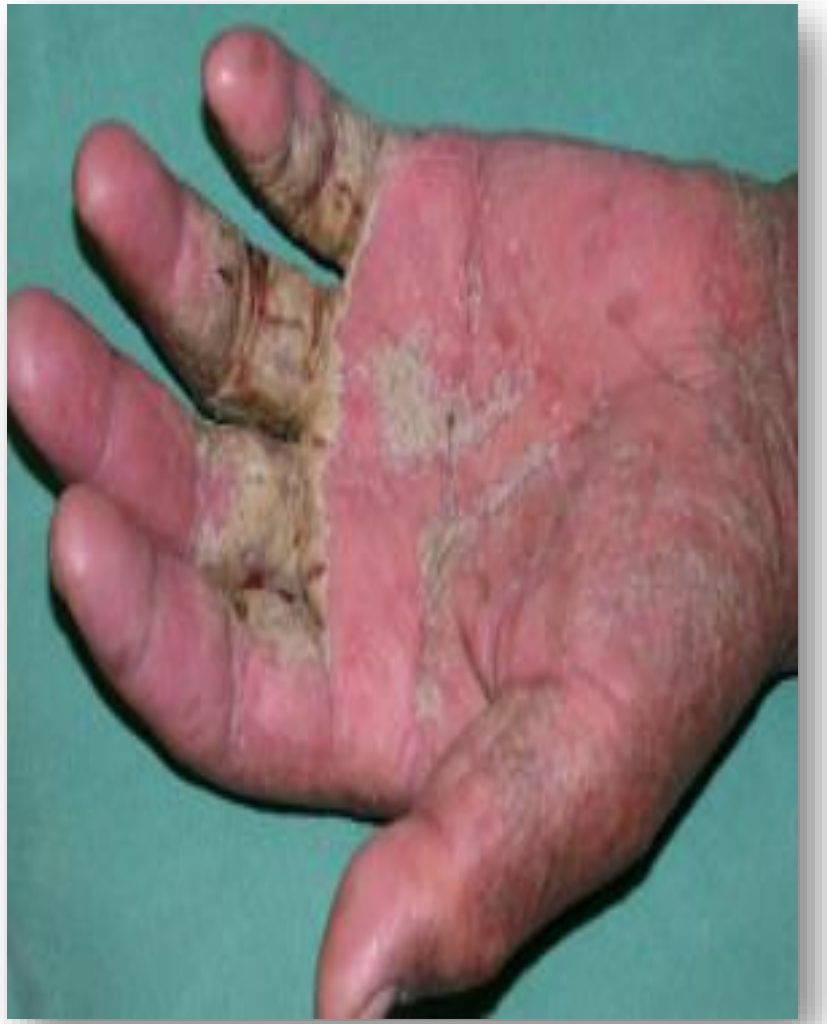
- **What is the least site to be infested by this organism ?**
  - Face, back and scalp
- **What is the incubation period for re-infection with this disease after treatment ?**
  - 8-10 days





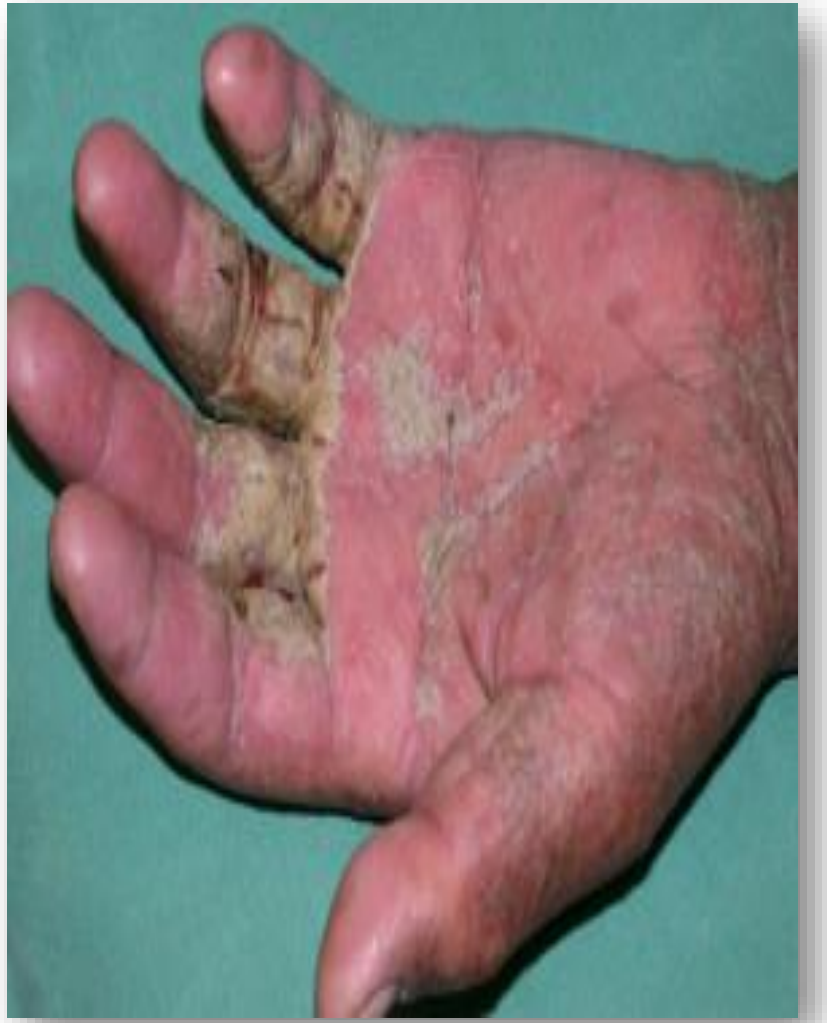
Crusted scabies

- Previously called Norwegian scabies
- Individual is infested by thousands or millions of mites living in the surface of the skin
- The patient presents with a generalized scaly rash. This is often misdiagnosed as psoriasis or eczema
- Scale is often prominent in the finger webs, on wrists, elbows, breasts and scrotum.
- Itch may be absent or minimal.
- Crusted scabies may affect the scalp.



Crusted scabies – risk factors

1. Very old age
2. Malnutrition
3. Immune deficiency
4. Intellectual deficit
5. Neurological disease
6. A specific inherited immune defect in some otherwise healthy people

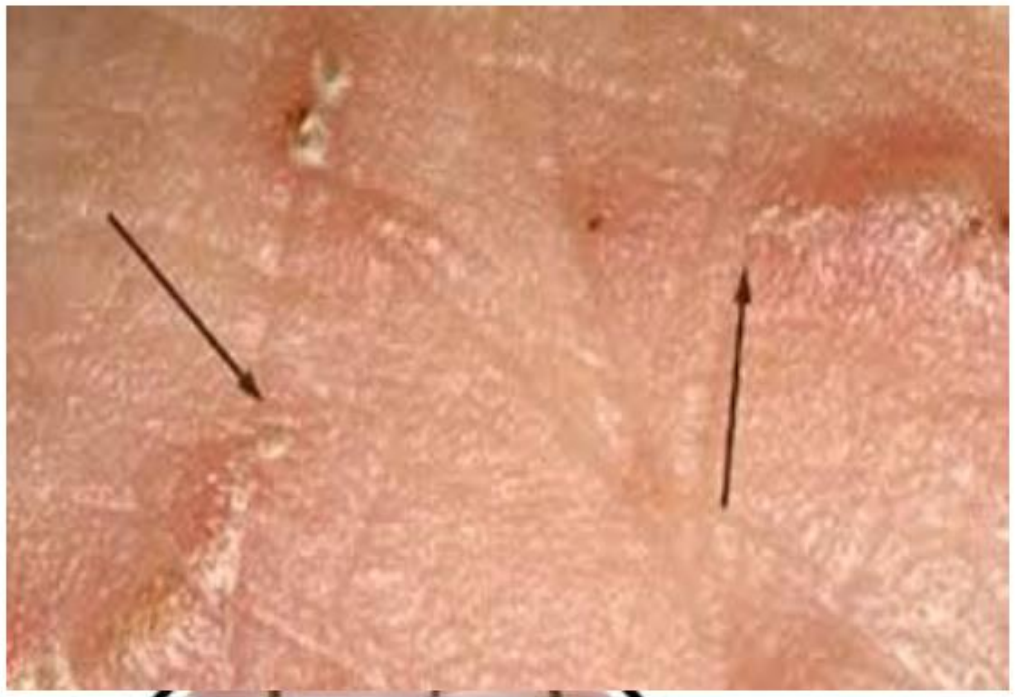


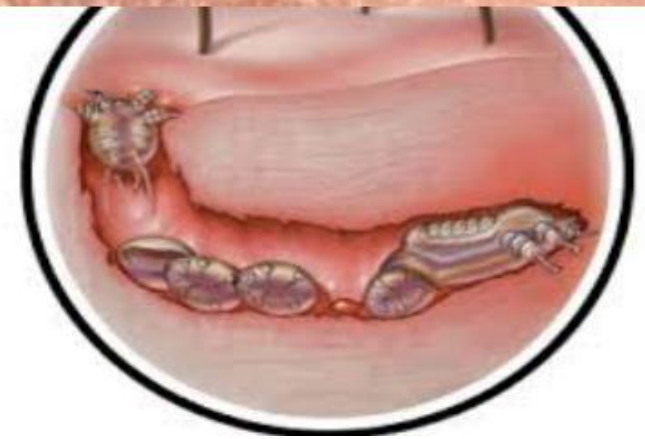
شرح

Scabies

- **The diagnosis**:
  - Dermatoscopy: the mite at the end of a burrow has characteristic jet-plane or hang-glider appearance
  - Microscopic examination of the contents of a burrow
- **Treatment**: (الأحد الأمور الي بركزوا عليها بالراوندات)
  - 25% benzyl benzoate lotion, applied daily every 12 hours for 3 days for adults. This is irritant and should not be used in children. (1st line in adult) سنوات (1)
  - Crotamiton cream once daily for 10 days for pregnant women and children <5 years of age.
  - Oral antibiotics for secondary infection.
  - Treatment should be repeated after 8–10 days after the first application to catch mites that have newly hatched.
  - Patients with crusted scabies may need repeated oral and topical treatments over several weeks or longer.

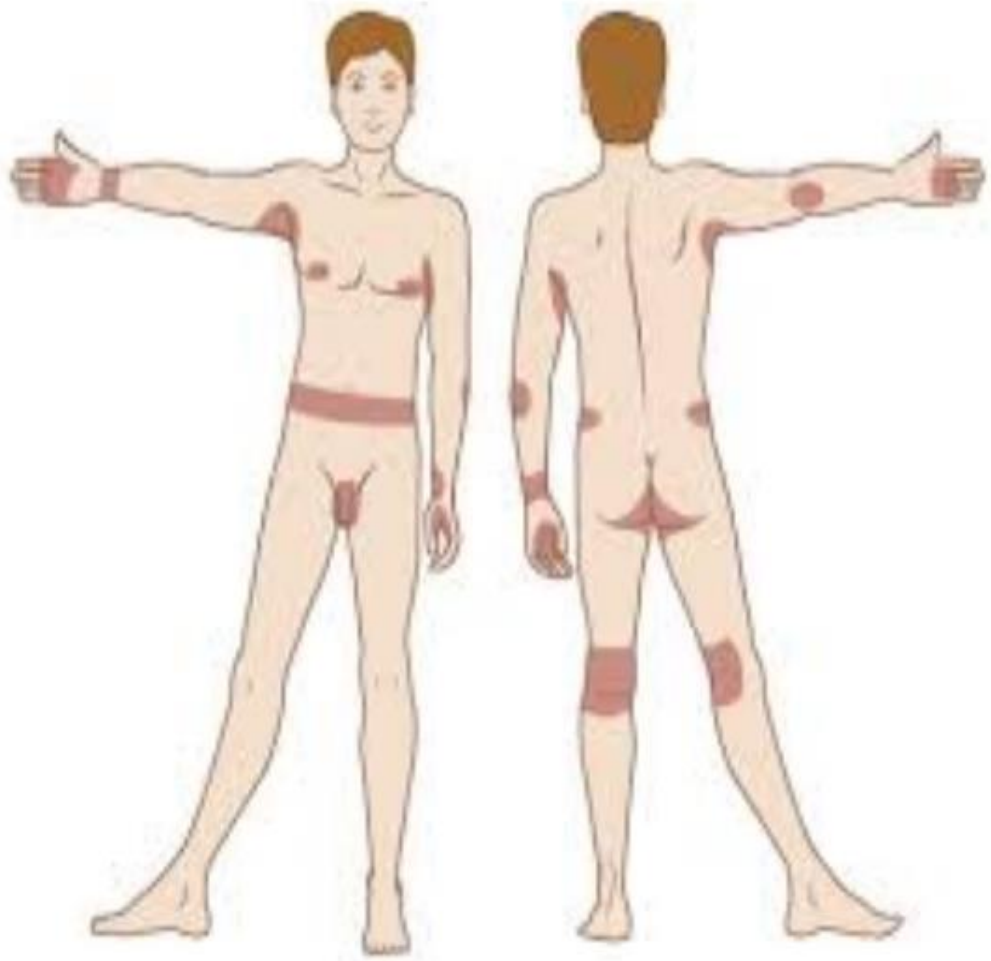
Scabies





Burrows
Few mm
Egg is cemented

Scabies



Scabies in adults spares the face and back due to the cidal effect of sebum

Scabies

But it can affect the face in children

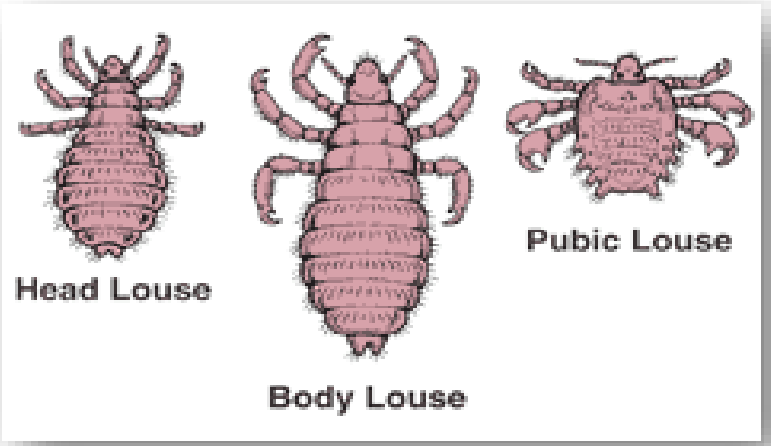
Pediculosis

Pediculosis

- **Pediculosis** is an infestation of the hairy parts of the body or clothing with the eggs, larvae or adults of lice. The crawling stages of this insect feed on human blood, which can result in severe itching
- **Type of transmission**: from person to person during direct contact
- **The three species of louse that infest humans are**:
  1. Pediculus humanus var. capitis - The head louse
  2. Pediculus humanus var. humanus - The body louse.
  3. Phthirus pubis - The pubic louse.

سنوات (1)

  - Head lice, the most common infestation in humans, are colloquially known as cooties and their eggs are called nits.
  - Pubic lice are smaller with a short body resembling a crab.



شرح

Pediculosis

- **Head lice (Pediculus humanus var. capitis)** سنوات (1) | What is the causative agent of head lice ?
  - Head lice infestations are frequently found in school settings or institutions.
  - Most common area for head lice: Occipital area.
  - Head lice is the most common cause of itching in children.
- **Body lice (Pediculus humanus var. humanus)**
  - Body lice infestation can be found in people living in crowded, unsanitary conditions where clothing is infrequently changed or laundered.
  - Body lice tend to infest people in extreme states of poverty or personal neglect.
  - The eggs of body lice are laid and glued to cloth fibers instead of hair, and the lice feed off the skin.
- **Pubic (Crab) lice (Phthirus pubis)**
  - Crab lice infestations can be found among sexually active individuals (sexually transmitted)
  - most commonly affect the pubic hair, but lice can spread to other hairy parts of the body
  - Infestation presents as itching, but blood specks on underclothes and live lice moving in the pubic hair are occasionally noted.

شرح

Pediculosis

(2) سنوات

❖Nymph:

- Eggs (brown in color containing the louse).

(1) سنوات

❖Nits:

- Hatched nymph leaves an empty capsule which is white in color.
- Head lice nits are flask-shaped

❖Hair cast:

- Thin, elongated, firm, whitish cylindrical concretions which ensheath the hair shaft and can be easily dislodge.

❖Hair cast vs pediculosis nits

- Nits are found firmly attached to hair shaft & are glued to the hair
- Hair cast slide up the hair shaft

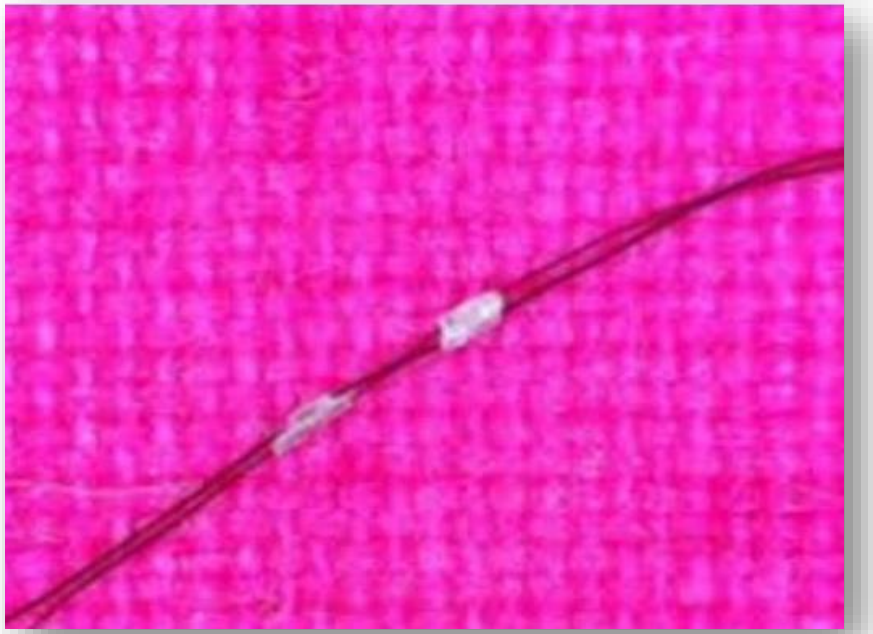
Nits

Hair cast

Lice: cemented

Pediculosis & Hair casts

- **Describe**:
  - Thin, elongated, firm, whitish cylindrical concretions which ensheath the hair shaft and can be easily dislodge.
- **Mention 2 Ddx**:
  - Nits & Hair casts
- **How to differentiate between the 2 ddx**:
  - **Nit**: firmly attached to hair shaft & are glued to the hair
  - **Hair cast**: can be easily dislodge and slide up the hair shaft



Pediculosis

- **Treatment (Head lice)**:
  - Application of insecticide foam, shampoo or liquid, repeated in one week.
  - Wet hair with vinegar to loosen nits.
  - Vigorous and repeated combing using a fine-toothed comb.
  - Regular scalp inspections.
  - Hot wash towels, sheets, pillowcases, clothing, brushes.
  - Isolate stuffed toys and other non-washable fomites for one week.
- Topical insecticides are neurotoxic and are not effective against young nits. They include
  1. Gamma benzene hexachloride: neurotoxic, increasing levels of resistance
  2. Pyrethroids: safe, may irritate
  3. Permethrin: if necessary, extend time of application to overnight treatment under a shower cap
  4. Malathion: flammable.

Pediculosis

- **Treatment (Body lice)**:
  - Similar insecticides used in the treatment of head lice are used in the treatment of body lice. Hot washing of clothes and bathing should be emphasized
- **Treatment (Pubic lice)**:
  - An insecticide such as Prioderm Cream Shampoo (maldison 1%) should be applied to all hairy parts of the body apart from the eyelids and scalp. It is washed off after 5 to 10 minutes and any remaining nits should be removed by using a fine-toothed comb. A repeat application is advisable 7 days later.
  - Lice and nits can be removed from eyelashes by using a pair of fine forceps. Alternatively, petroleum jelly, such as Vaseline can be smeared on the eyelashes twice a day for at least 3 weeks.
  - Underwear and bed linen should be washed thoroughly in hot water to prevent recurrences. Sexual partners need to be treated even if they deny itching and do not appear to be infected.

What is your diagnosis ?

Pediculosis capitis

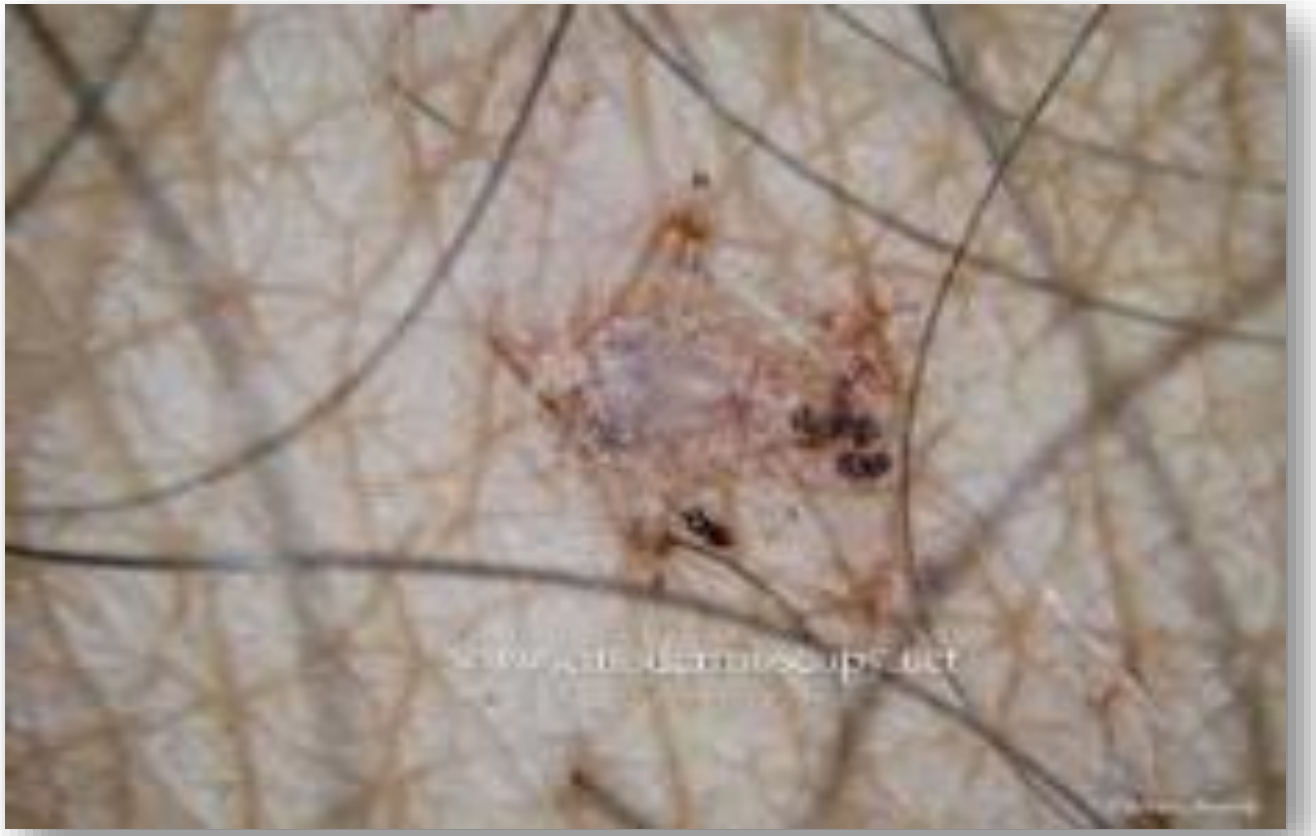
Pubic lice (crabs)

Body lice

Lice on clothes

What is the diagnosis ?

A. Crabs
B. Scabies
C. Pediculosis capitis
D. Tinea corporis
E. Lyme disease



Cutaneous leishmaniasis

مش مطلوب للامتحان بس اكتشفت بعد ما قعدت ساعة وانا أكتب فاحلم احذفه

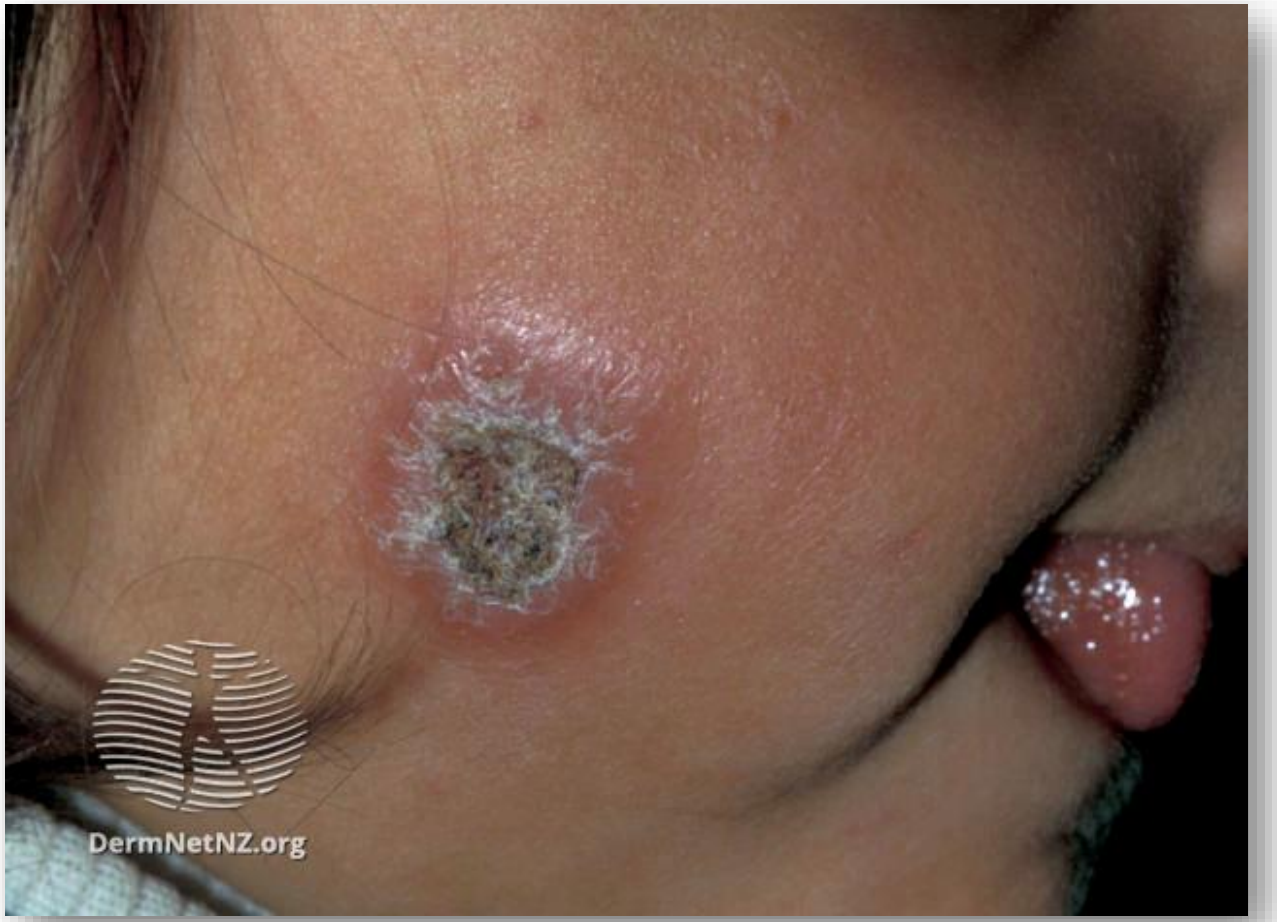
Leishmaniasis

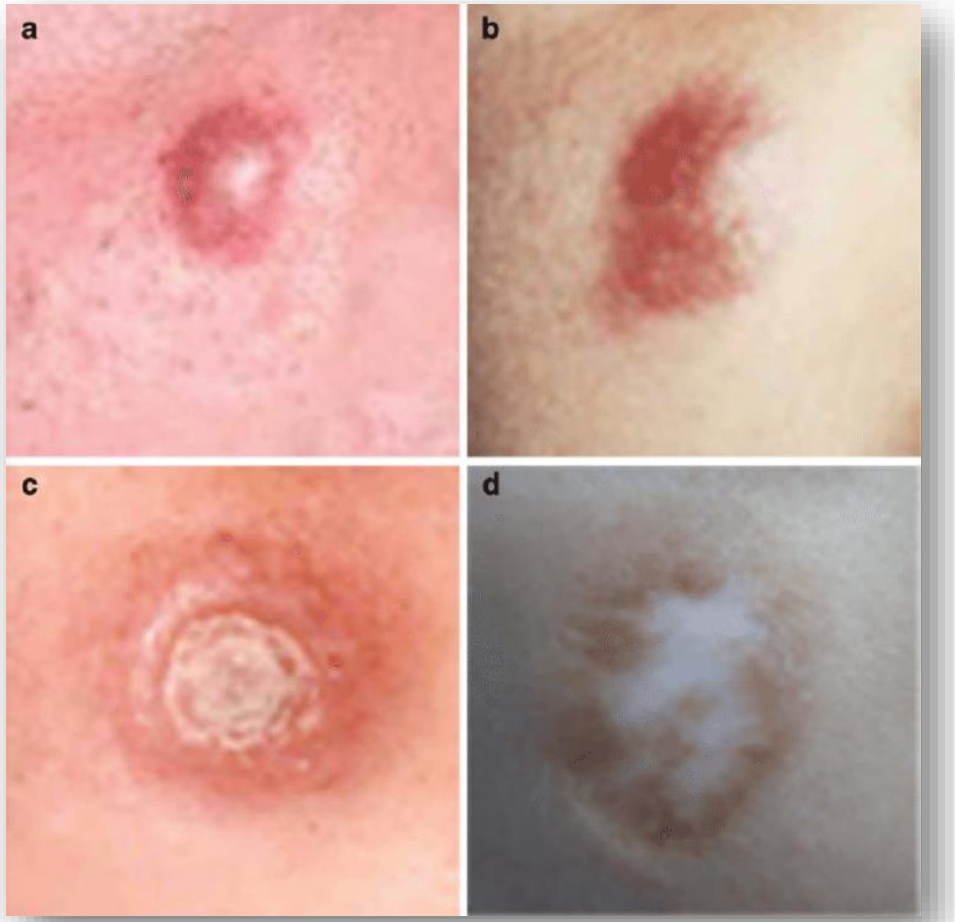
- **Leishmaniasis** is a parasitic disease transmitted by sandflies infected with the protozoa Leishmania.
- Forms of leishmaniasis:
  1. Cutaneous leishmaniasis
  2. Mucocutaneous leishmaniasis
  3. Diffuse cutaneous leishmaniasis resulting from an anergic response to the parasite by the host
  4. Visceral leishmaniasis results from the involvement of the internal organs and is usually fatal if untreated. It is also known as kala-azar or Dumdum fever.
  5. **Leishmaniasis recidivans** is a rare, cutaneous form of leishmaniasis, occurring in patients with a good cellular immune response. It is also known as lupoid leishmaniasis

Cutaneous leishmaniasis

- Most common form of leishmaniasis
- Typically, solitary lesion with central ulceration, but multiple lesions do occur
- **Primary lesion**: Painless small red papule or nodule
- Lesions are usually painless, and most resolve spontaneously often leaving residual atrophic scarring
- Chronic disease can occur, and there is a risk of dissemination in immunodeficient patients
- **Treatment**:
  1. Cryotherapy + Intralesional Pentostam (Sodium Stibogluconate) injection
  2. If multiple : IM Pentostam (Cardiotoxic)

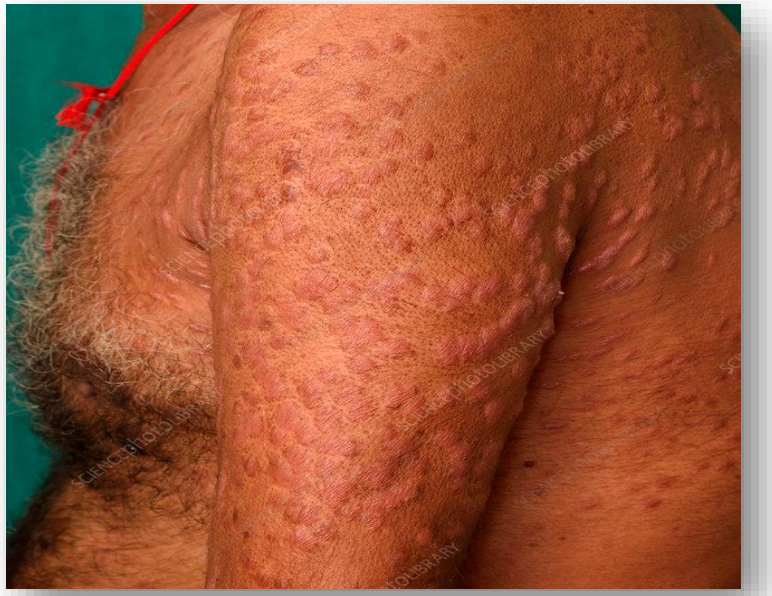
Cutaneous leishmaniasis





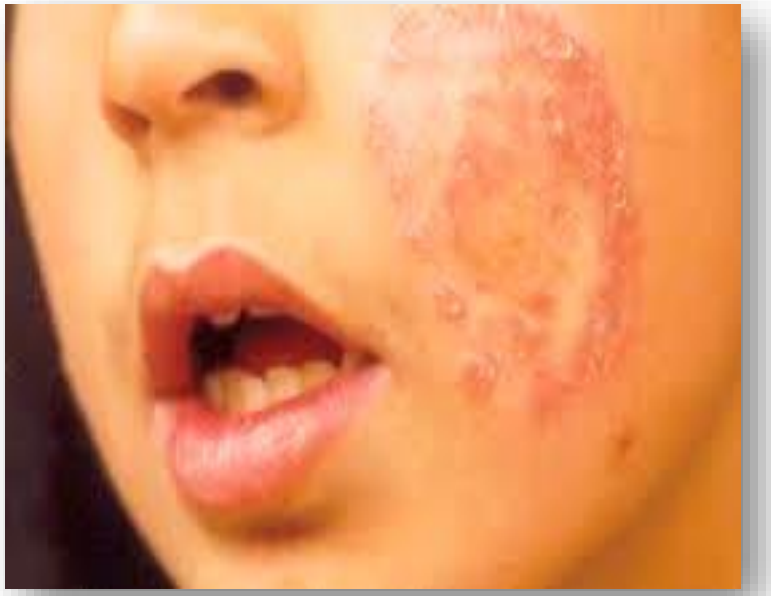
Clinical course of the cutaneous leishmaniasis.

What is your diagnosis ?



Diffuse cutaneous leishmaniasis

- Following the primary cutaneous leishmaniasis lesion, non-ulcerative nodules and plaques develop
- Lesions may be numerous and may extend over the whole body
- Follows a chronic relapsing or progressive course
- Often difficult to treat



Leishmaniasis recidivans

- Spontaneous resolution of the primary cutaneous lesion is followed by the development of new lesions around the edge of the primary scar
- The lesions typically ulcerate then heal
- The cycle continues with a chronic recurrent course, usually over decades

Sexually Transmitted Diseases

Sexually Transmitted Diseases

❖**Risk factors for STDs**:

1. Sexually active age (25-35 Yr.)
2. Sexual promiscuity
3. History of sexually transmitted disease
4. Sexual abuse
5. Alcohol and drug abuse
6. Multiple partners (Extramarital sexual contacts)

❖**Causes of STDs**:

- Bacterial (*N.gonorrhea*, *T.pallidum*, *H.ducreyi*, and others)
- Viruses (HIV, HPV, HSV, Molluscum contaginosum virus, and others)
- Protozoa (*T.vaginalis*, *G.lamblia* , *E.histolytica*, and others)
- Fungi (*Candida albicans*)
- Ectoparasites (*Sarcoptes scabiei*, *phthirus pubis*)

Urethritis

Non-gonococcal & gonococcal urethritis

شرح

1. Non-gonococcal urethritis

- The most common sexually transmitted disease

سنوات (1)

- **Causative agents**: Chlamydia trochomatis (Mostly), Ureaplasma urealyticum, Trichomonas vaginalis and rarely by others
- **Clinical presentations**: mild watery, mucoid or mucopurulent urethral discharge and dysuria.
- **Diagnosis**: Clinical presentations, Urethral discharge smear, urine analysis, PCR.
- **Incubation period**: 1-2 weeks
- **Treatment**:
  - Doxycycline 100mg twice daily for 1-2 weeks
  - Partner should be treated in all STDs and should be examined for other possible STDs
  - Doxycycline or azithromycin + Ceftriaxone for possible N.gonorrheae coinfection

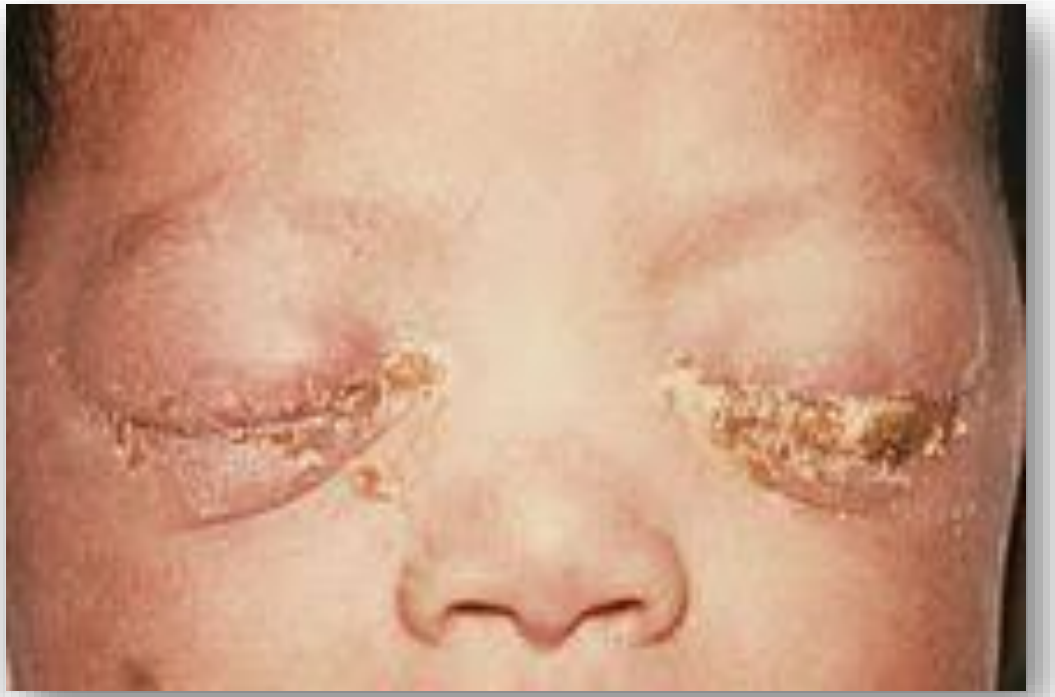
2. Gonococcal urethritis (Gonorrhea)

- Second most common STD
- **Causative agent**: Neisseria gonorrhea (Gram negative diplococci)
- **Clinical presentations**:
  - It can present as urethritis, cervicitis, proctitis, pharyngitis and conjunctivitis in newborns because Neisseria gonorrhea affects the columnar epithelium
  - Men usually present with heavy purulent (pussy) discharge and dysuria. In women as cervicitis , the discharge is less
  - In women 50% of cases are asymptomatic
- **Diagnosis**: Clinically, urethral discharge smear and culture (Thayer-Martin medium (VPN medium)) for antibiotic sensitivity
- **Incubation period**: 3-5 days

VPN media
Vancomycin
Polymyxin
Nystatin

2. Gonococcal urethritis (Gonorrhea)

Heavy discharge in men, while its mild in women.



Ophthalmia Neonatorum

Gonococcal conjunctivitis, neonate gets infection from his mother during birth, prevented by giving Erythromycin eye drops soon after birth.

شرح

2. Gonococcal urethritis (Gonorrhea)

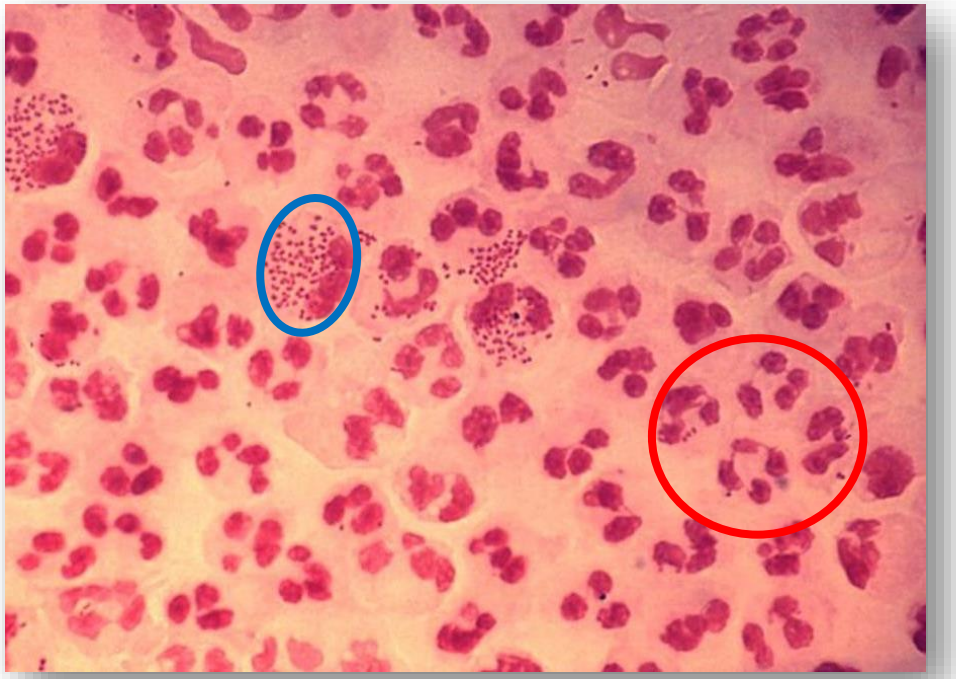
❖**Treatment**:

- Single dose of ceftriaxone 250mg IM and Doxycycline (to treat any associated non-gonococcal urethritis) 100g orally twice daily for 2 weeks, alternative therapeutic agents also present for some cases.

سنوات (1)

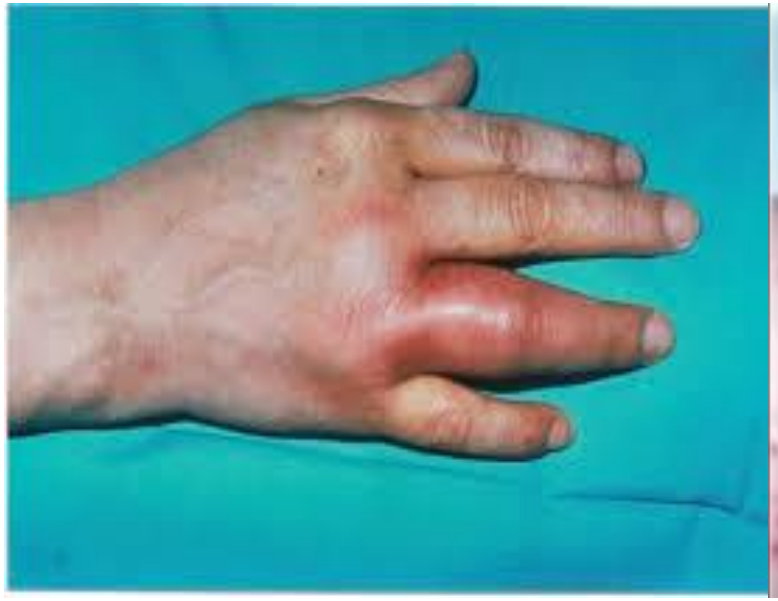
❖**Complications**:

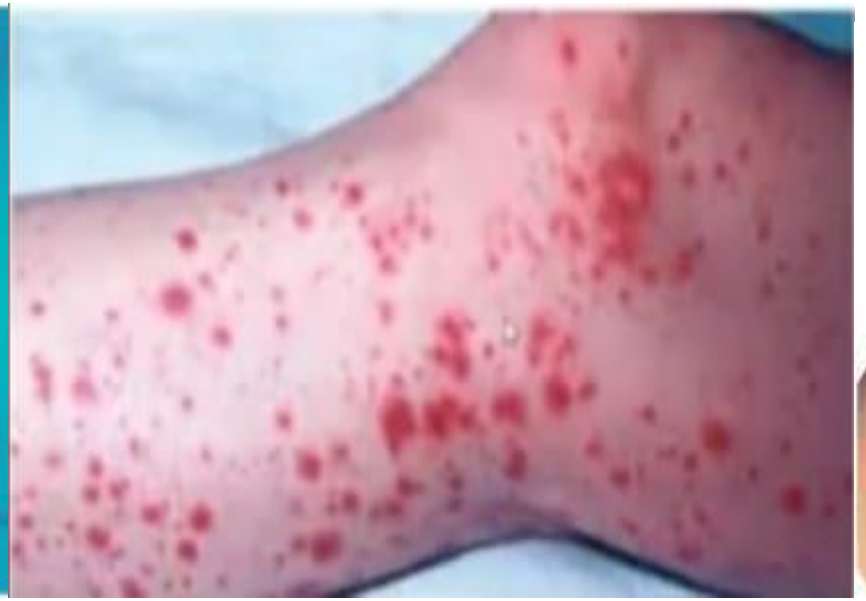
- **Males**: Epididymitis, orchitis, proststitis
- **Females**: Salpingitis and PID
- **Both**: Infertility and gonococcemia (Arthritis dermatitis syndrome)

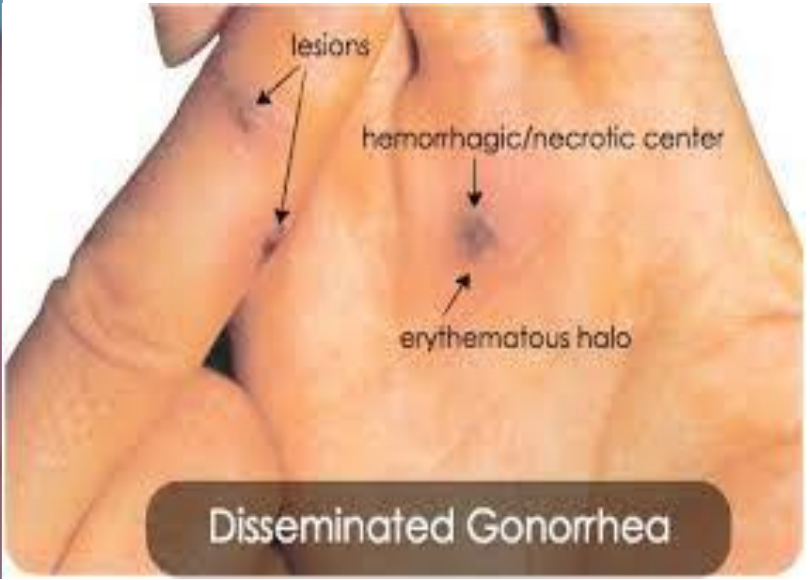


Urethral discharge smear will show gram negative diplococci and neutrophils

2. Gonococcal urethritis (Gonorrhea)







Gonococcemia manifested as Arthritis and Dermatitis

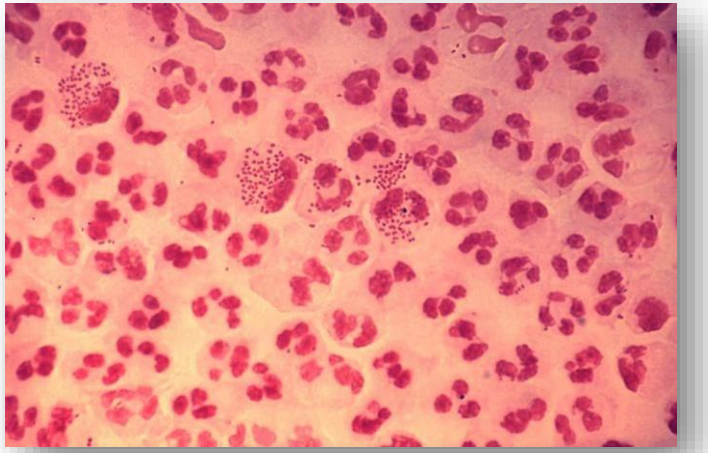
(Necrotic and vasculitic lesions)

Urethritis

سنوات (4)

❖**What is your diagnosis of the photos ?**

- Gonorrhea



سنوات (1)

❖**What is the cause of**

- **Non-gonococcal urethritis**: *Chlamydia trachomatis* (Mostly), *Ureaplasama urealyticum*, *Trichomonas vaginalis* and rarely by others
- **Gonorrhea** : *Neisseria gonorrhoeae*

سنوات (1)

❖**The best diagnostic test for gonorrhea**

- Urethral discharge smear and culture

❖Treatment:

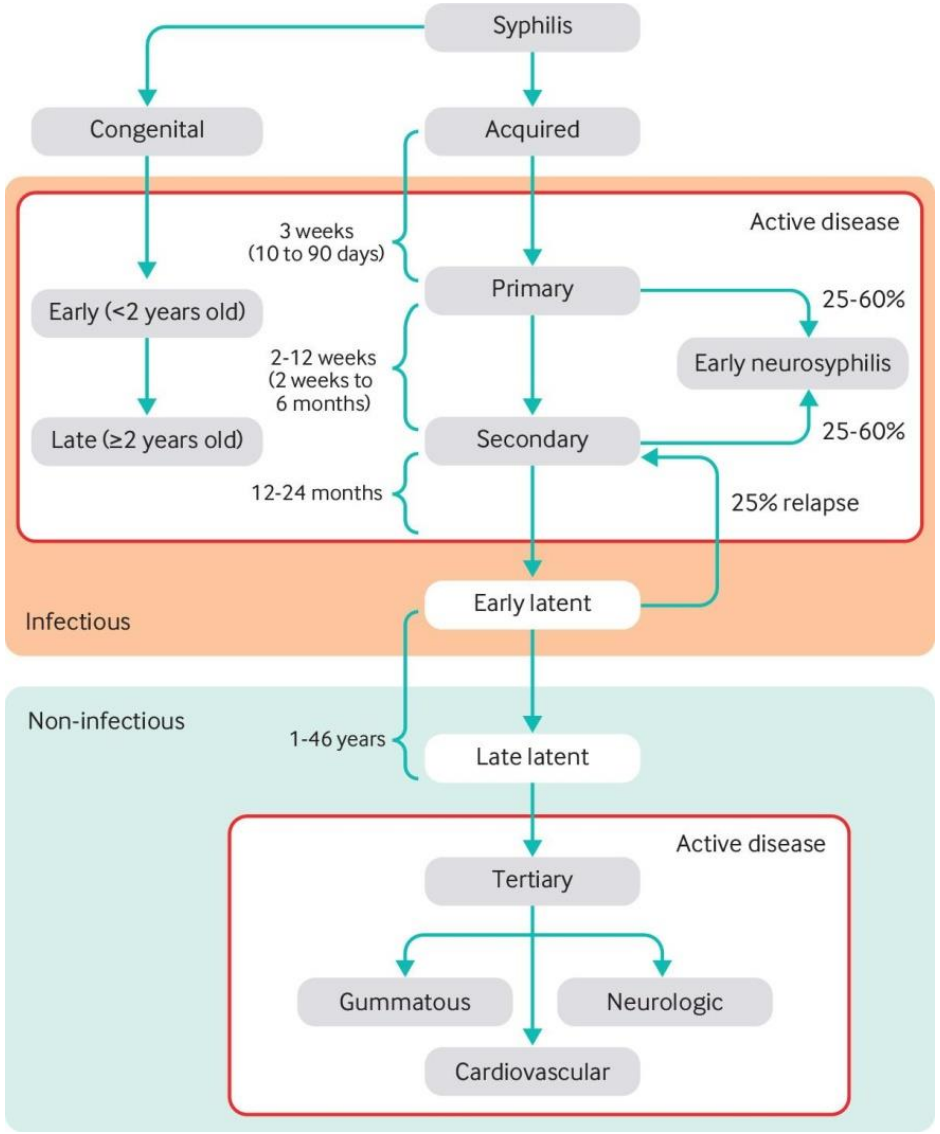
- Ceftriaxone + azithromycin or doxycycline for possible non-gonococcal coinfection

Syphilis

Syphilis

- **Causative agent**: Treponema pallidum spirochete
- **Clinical presentations**: Primary syphilis classically presents as a single, painless, indurated genital ulcer (chancre)
- **Diagnosis**: Usually based on serology, using a combination of treponemal and non-treponemal tests
- **Drug of choice**: Penicillin (**First line**: Benzathine penicillin G)
- Incidence rates of syphilis have increased substantially around the world, mostly affecting men who have sex with men and people infected with HIV.
- Patients with syphilis should be screened for HIV, gonorrhea, and chlamydia.

Stages and classifications



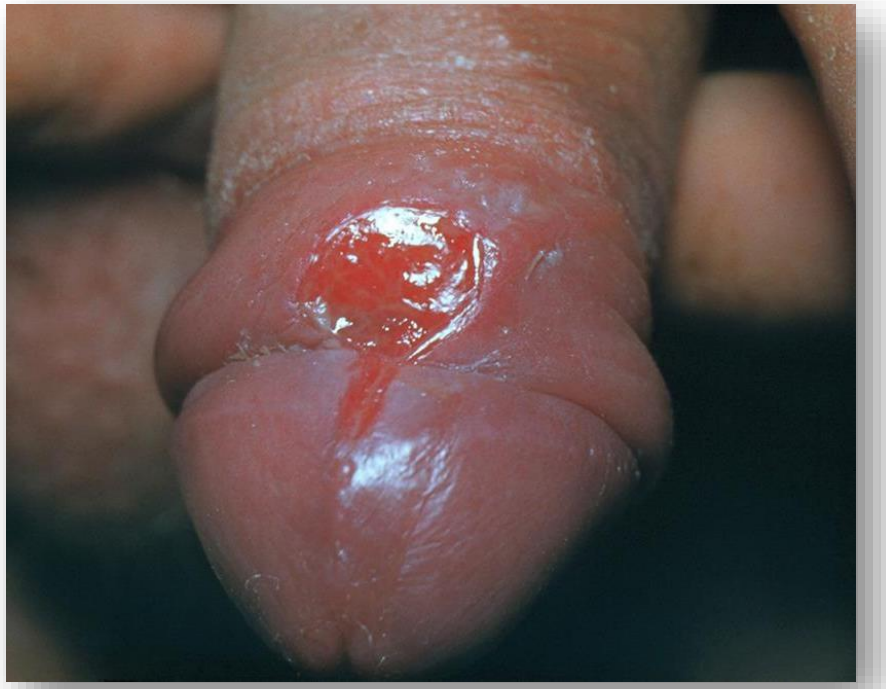
- Symptomatic only in primary, secondary, and tertiary stages.
- Syphilis is infectious only during primary, secondary, and early latent stages.

Primary syphilis

- **Timing**: Symptoms appear 10-90 days (mean 21 days) after exposure
- **Manifestations**:
  - **Main symptom**: <2 cm chancre
  - **Progress**: macule to papule to ulcer over 7 days
  - **Characters**: Painless, solitary, indurated, clean base (98% specific, 31% sensitive)
  - **Site**: On glans, corona, labia, fourchette, or perineum
  - A third are extragenital in men who have sex with men (MSMs) and in women
  - Localized painless adenopathy
- Chance resolve within 3-10 weeks and 60% of patients do not recall this lesion because its asymptomatic sometimes.

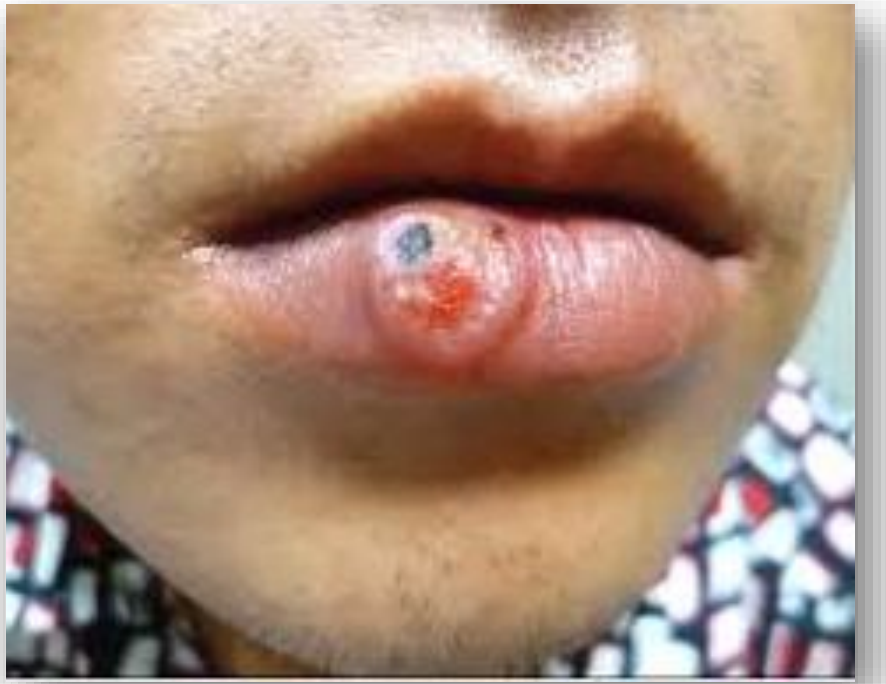
سنوات (1)

Primary syphilis



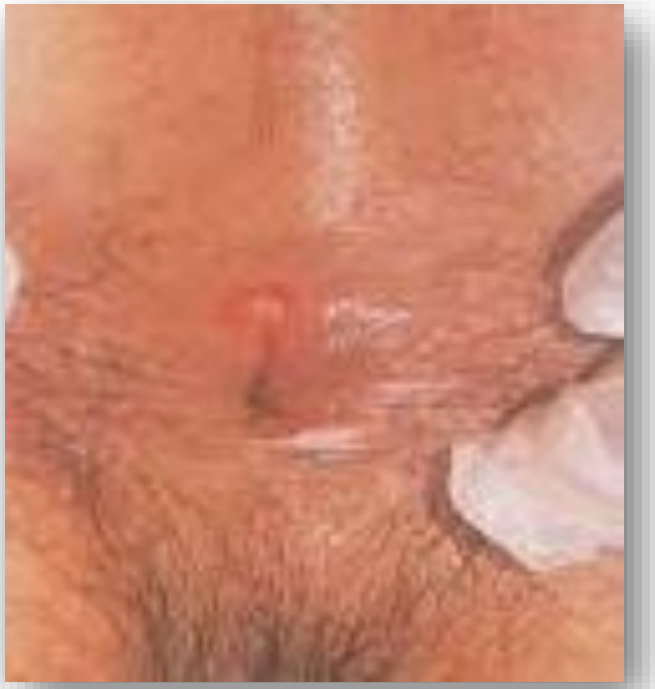
Chancre

Painless, Indurated ulcer



Extra-genital Chancre

Extra-genital Chancre with clean base on the area of contact (on the lips)



Peri anal Chancre

Risk group are MSMs

Secondary syphilis

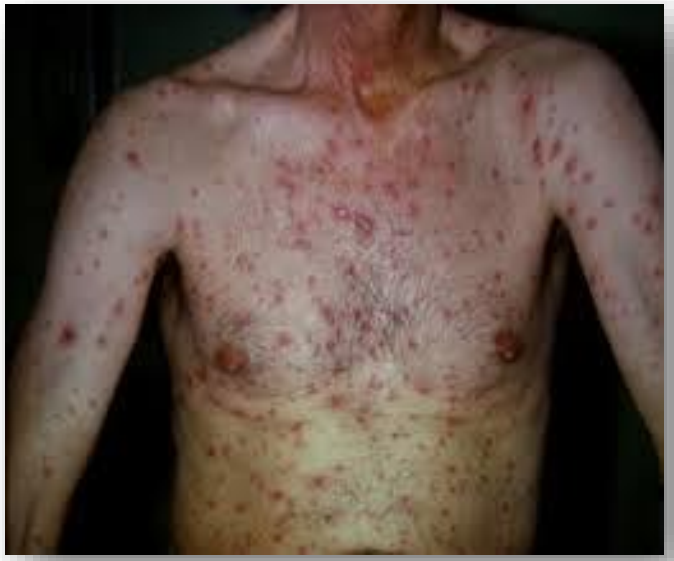
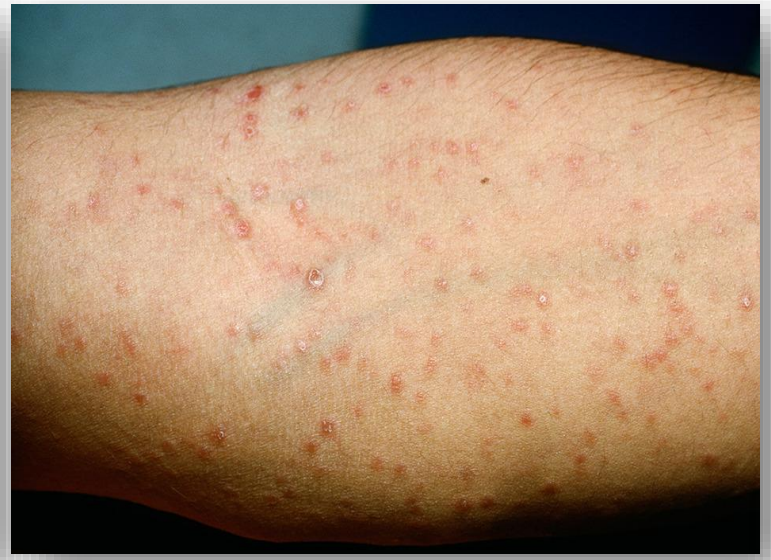
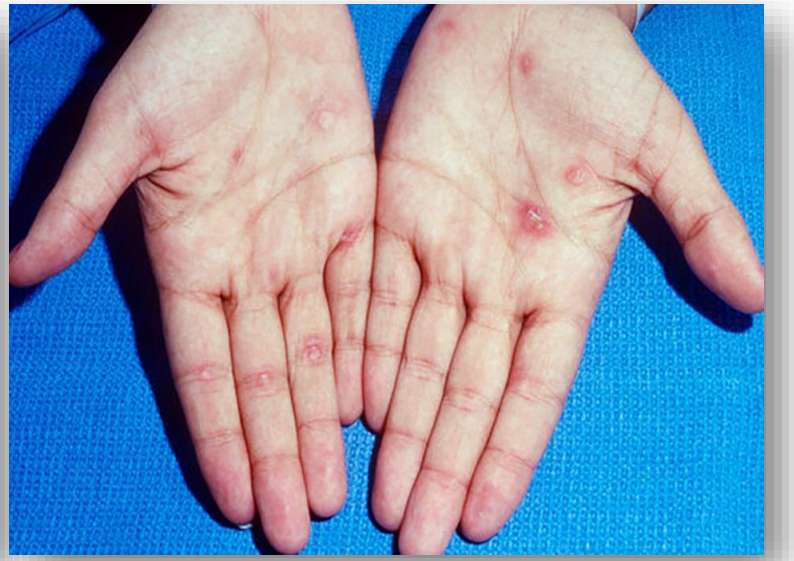
❖**Timing**:

- Symptoms appear 2 weeks to 6 months (mean 2-12 weeks) after exposure.
- Can be concurrent with, or up to 8 weeks after the chancre.

❖**Manifestations**:

1. **Rash**: in about 90% of cases. Diffuse, symmetric, on trunk (often subtle or atypical), usually asymptomatic.
2. **Condylomata lata (fleshy moist papules)**: in about 20% of cases (in moist areas = groin and flexural areas); The characteristic lesion in 2ry syphilis
3. **Mucous patches-oral mucosa** in about 30% of cases.
4. **Patchy alopecia** (4-11%).
5. **Generalized painless lymphadenopathy** in about 75% of cases.
6. **Fever, night sweats and headaches**.
7. **Neurologic symptoms** in about 25% of cases. Cranial nerve palsies (II,VIII), eye redness or pain, meningitis, changes to mental status or memory.

Secondary syphilis

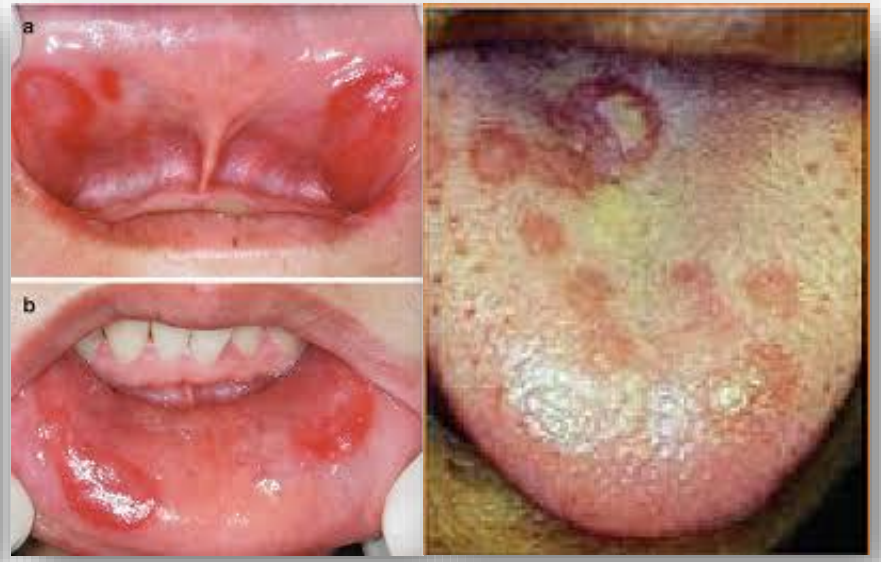




Symmetrical, asymptomatic, scaly papulosquamous rash on the trunk, extremities, palms and soles, **any asymptomatic rash on these regions should urge us to do serological test for syphilis**. سنوات (1)

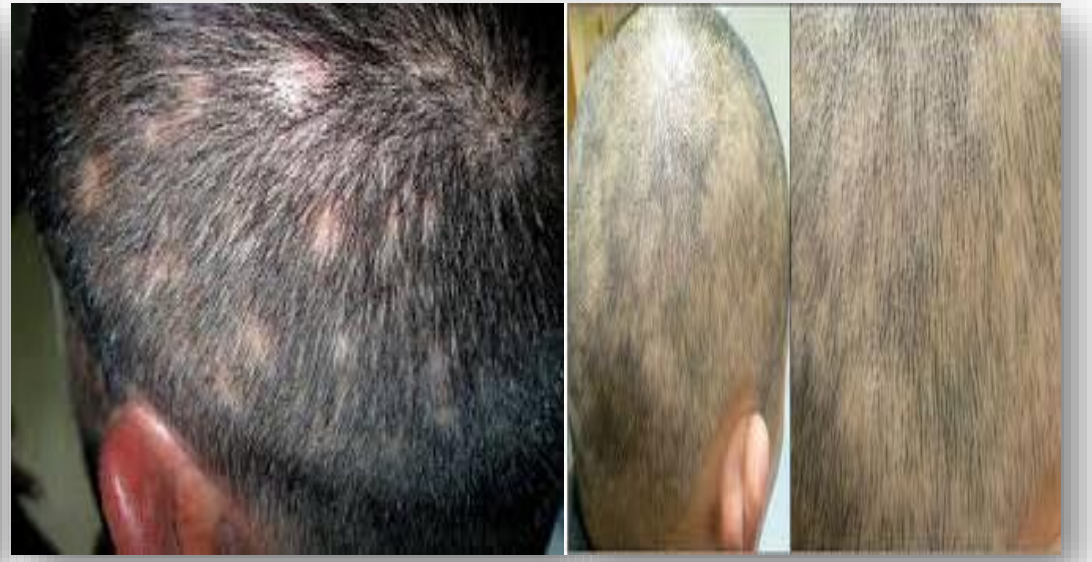
Secondary syphilis

Condylomata lata

Moist papules on the genital area, very infectious



Mucous patches with erosions on the oral mucosa and the tongue



Moth eaten alopecia

Multiple patchy alopecia

Essay Questions

سنوات (7)

❖What is the cause of

- Syphilis: treponema pallidum
- Condyloma lata: treponema palladium

سنوات (1)

❖Characteristic of lesion in 2ry syphilis

- Condyloma (Condylomata) lata

سنوات (3)

❖Mention 4 skin lesions in 2ry syphilis

- Condylomata lata
- Patchy alopecia
- Rash (Generalized, maculopapular rash)
- Mucous patch-oral mucosa

Latent syphilis

❖ **Timing**:

- **Early latent** (<12 months or <24)
  - About 25% of subjects relapse to secondary syphilis and they are infectious
- **Late latent** (>12 months or >24 months) no relapse and not infectious
  - About 25% 0f cases in late latent syphilis develop tertiary syphilis

❖ **Manifestations**: No symptoms with positive serology

Notes:

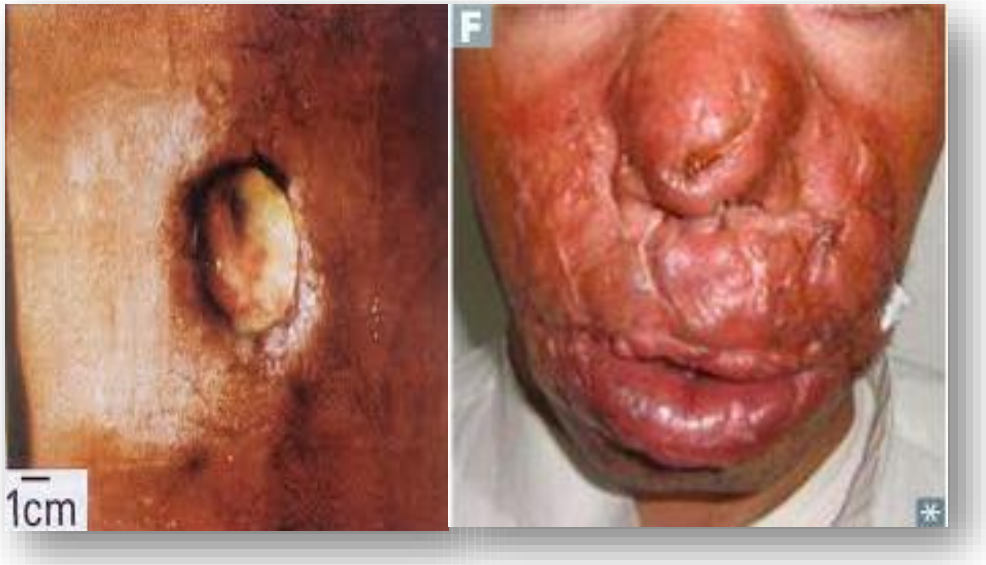
❖ <12, >12 months system = USA, UK, Canada guidelines

❖ <24, >24 months = WHO guidelines

❖ In early latent stage 90% of those who relapse, relapse in first year, 94% relapse within 2 years

Tertiary syphilis

- **Timing**: Around 25% of late latent cases can develop 3ry within 1-46 years after exposure
- **Manifestations**:
  - **Neurologic**: Paresis, tabes dorsalis, Argyll Robertson pupils (about 6%)
  - **Cardiovascular**: aortitis (about 10%)
  - **Gummatous**: necrotic granulomatous lesions in the bones and skin (about 20%)



Gumma

Necrotic tissue

Argyll Robertson pupils

Light near dissociation (irregular pupils, not reactive to light but reactive to near object)

Diagnosis of syphilis

- **Nontreponemal tests**:
  - **Indications**: screening, evaluation of disease activity, monitoring response to treatment
  - **Commonly used tests**: RPR, VDRL
- **Treponemal tests**:
  - **Indication**: confirmatory test after a positive or inconclusive nontreponemal test
  - **Commonly used tests**: FTA-ABS, TPPA, TPHA, EIA, CLIA
- **Direct detection of the pathogen**:
  - Definite tests to detect primary and secondary syphilis when a specimen can be obtained
  - **Tests**: Dark field microscopy, direct fluorescent antibody testing, or PCR

Abbreviations

- **RPR**: Rapid Plasma Reagin
- **VDRL**: Venereal disease research laboratory test
- **FTA-ABS:** Fluorescent treponemal antibody absorption
- **TPPA**: Treponema pallidum particle agglutination
- **TPHA**: The Treponema pallidum hemagglutination
- **EIA**: Enzyme Immunoassay
- **CLIA**: Chemiluminescence Immunoassay
- **PCR**: Polymerase Chain Reaction

Diagnosis of syphilis

❖ **Approach**:

- **General approach**: Use nontreponemal serological tests to screen for syphilis, then treponemal tests and PCR to confirm the diagnosis. If both tests are positive, infection with syphilis is confirmed.
- Although, algorithms varies, some examples:
  - **Algorithm 1**: First, we run a screening treponemal test (EIA or CLIA), if positive we run a confirmatory treponemal test (TPPA). If both tests are positive, infection with syphilis is confirmed.
  - **Algorithm 2:** First, we run a screening nontreponemal test (RPR or VDRL), if positive we run a confirmatory treponemal test (FTA-ABS or TPHA).
- When a specimen can be obtained (e.g., exudative chancre, condyloma) we Directly detect the pathogen by dark field microscopy, direct fluorescent antibody testing, or PCR

Essay Questions

سنوات (1)

❖ **Which test should be used to measure disease activity and track response treatment in syphilis ?**

- Nontreponemal tests: RPR, VDRL

سنوات (1)

❖ **Patient with asymptomatic rash in palms and soles, what test you should do to confirm secondary syphilis ?**

- Write any of the following: TPPA, TPHA, FTA-ABS

سنوات (1)

❖ **Patient presented with single ulcer on penis from 7 days, what is the best investigation used to rule out syphilis ?**

- Directly detect the pathogen by dark field microscope, direct fluorescent antibody testing, or PCR

Treatment of syphilis

- **First-line therapy**: Benzathine penicillin G
  - **Primary, secondary, or early latent**: IM, a single dose is sufficient
  - **Late latent, tertiary, or date of transmission unknown**: weekly IM injections over a 3-week course
  - Sexual contacts should also be treated
- **Neurosyphilis**:
  - IV penicillin G every 4 hours for 10–14 days
- **Alternatively**:
  - Procaine penicillin G
  - In the case of allergy to penicillin, treat with doxycycline or ceftriaxone
  - In neurosyphilis and during pregnancy: desensitization and treatment with penicillin

Jarish-Herxheimer Reaction

- Self-limited phenomenon after first dose of treatment of syphilis.
- **Timing**: Occurs within 4-6 hours of giving the penicillin and subside within < 24 hours.
- Only appears after the first dose.
- **Symptoms**: Fever, chills, headache, malaise , arthralgia and myalgia and may be exacerbation of skin or mucous membrane lesions.
- It is more common in early and seropositive syphilis.

سنوات (1)

27-year-old male with asymptomatic skin rash

سنوات (1)

- **What investigation should you do ? And why ?**
  - Write any of syphilis screening tests (RPR, VDRL, EIA, CLIA)
  - To rule out syphilis

سنوات (5)

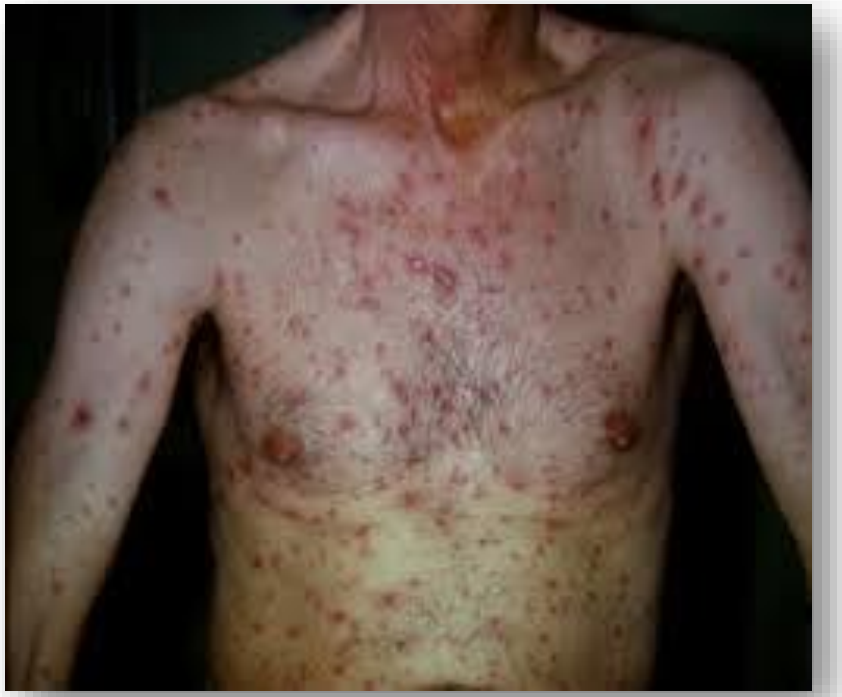
- **Mention 2 confirmatory test for your diagnosis**
  - FTA-ABS test, TPHA, TPPA

سنوات (2)

- **What is your treatment of choice ?**
  - Benzathine penicillin IM

إضافي

- **Mention 2 alternative treatments**
  - doxycycline or ceftriaxone



Congenital syphilis

سنوات (1)

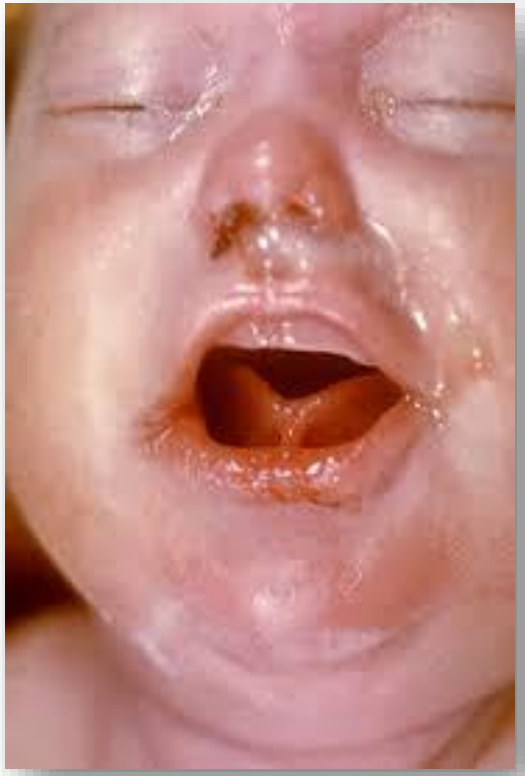
❖Early congenital syphilis:

1. Hepatomegaly most common findings and may associated with splenomegaly.
2. Jaundice, may or may not present.
3. Rhinitis, one of the first clinical presentation. (Snuffles)
4. Generalized non-tender lymphadenopathy-common finding.
5. Maculopapular skin rash appears 2 weeks after rhinitis.

❖Late congenital syphilis:

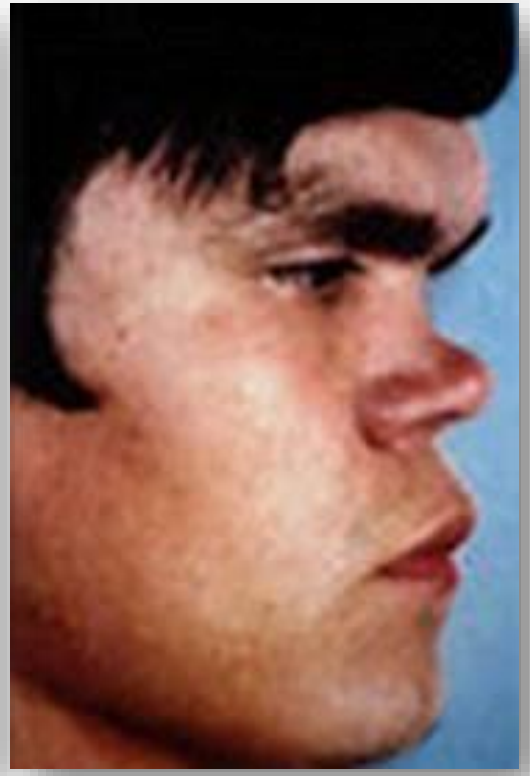
1. Skin and mucous membrane Gumma.
2. Facial changes: frontal bossing, saddle nose, prominent maxilla.
3. Anterior bowing of shin (saber shin).
4. Hutchinson teeth-hypoplastic notched permanent teeth(upper central incisors).
5. Nerve palsies, Sensorineural hearing loss and changes in vision.
6. Eye involvement.

Congenital syphilis



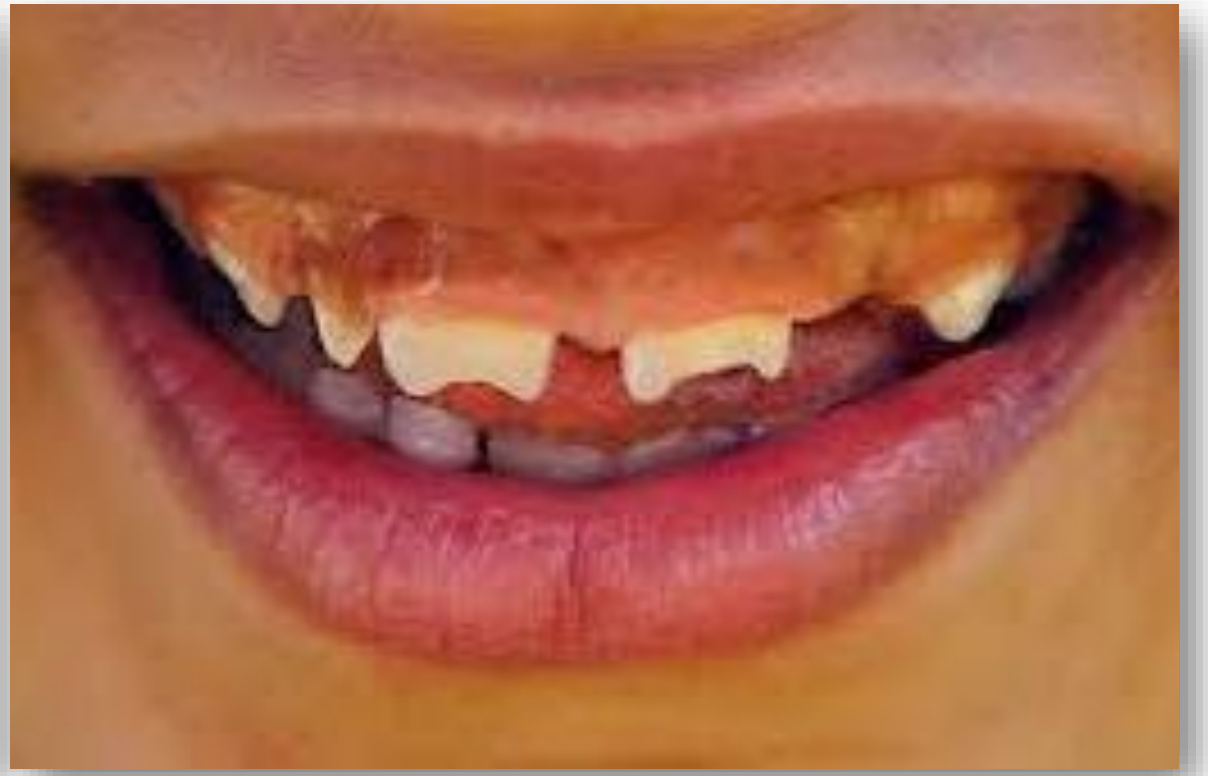
Rhinitis with snuffles

(Early congenital syphilis)



Saddle nose and frontal bossing

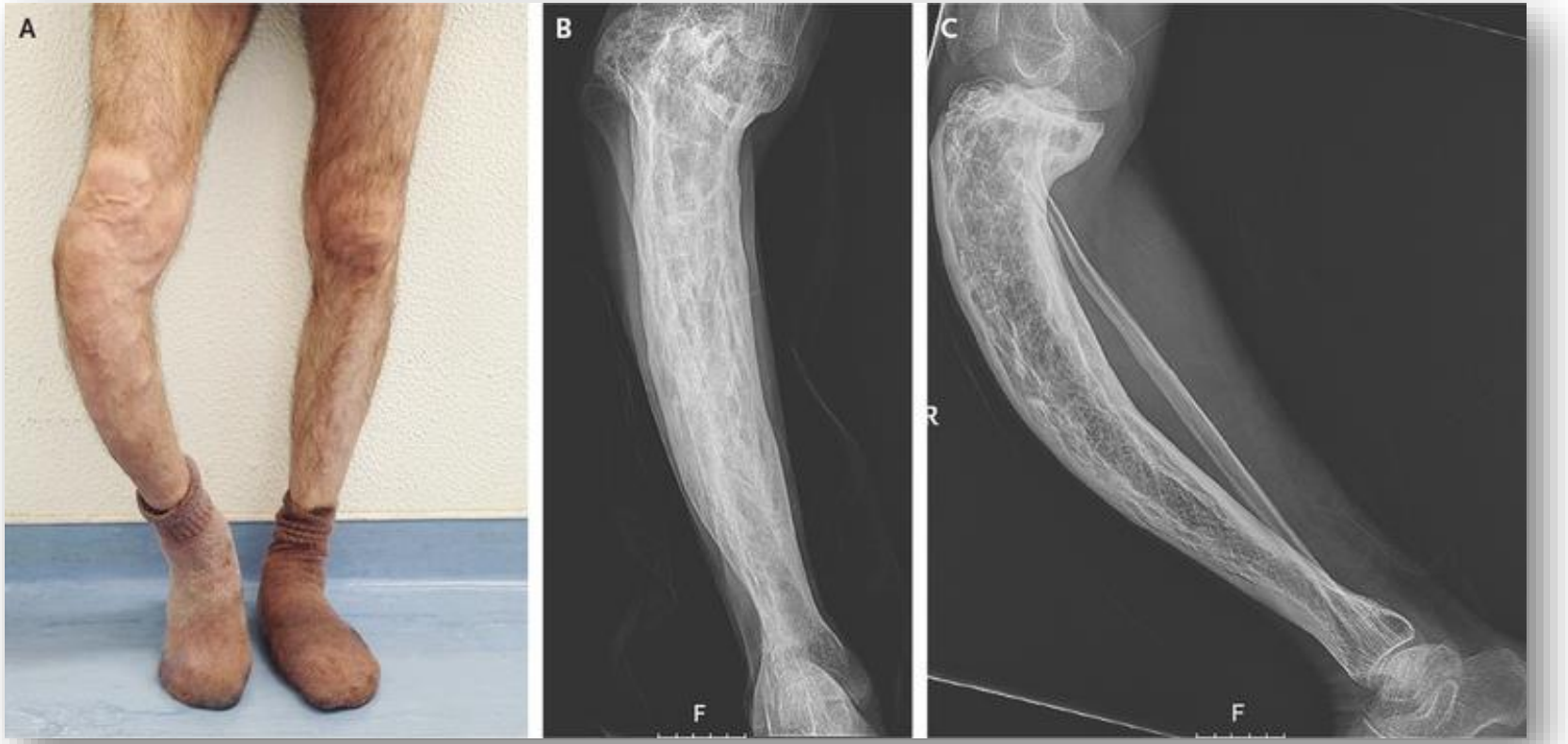
(Late congenital syphilis)



Hutchinson teeth

(Late congenital syphilis)

Congenital syphilis



Saber shins

(Late congenital syphilis)

Treatment of congenital syphilis

- **Infants up to 4 week of age**:
  - Aqueous crystalline penicillin G, 50,000 units/kg per dose IV every 12 hours in the first 7 days of life.
  - After 7 days of life, 50,000units/kg per dose every 8 hours for 10-14 days.
  - Alternatively, procaine penicillin G 50,000 units/kg/day IM for 10-14 days
- **Infants older than 4weeks and older children**:
  - Aqueous penicillin G 50,000 units/kg per dose every 6 hours IV for 10-14 days
- **Notes**:
  - We should treat both the child and the mother
  - In case of penicillin allergy, the patient should be desensitized and then given penicillin.
  - For patients with neurosyphilis, longer treatment with penicillin G is recommended.

Congenital Syphilis

❖**What is the name of this sign ?**

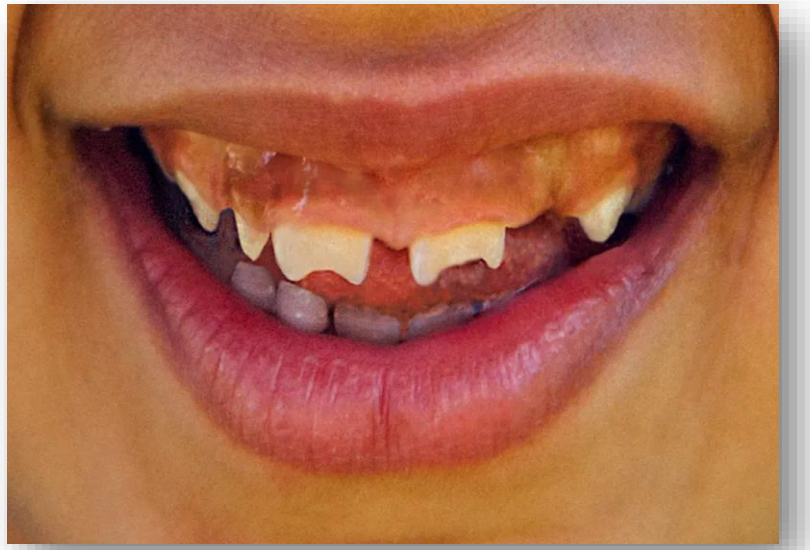
- Hutchinson's teeth (notched-incisors)

❖**What is the diagnosis ?**

- Congenital Syphilis

❖**Mention 3 manifestations of early congenital syphilis**

1. Hepatomegaly
2. Jaundice
3. Rhinitis

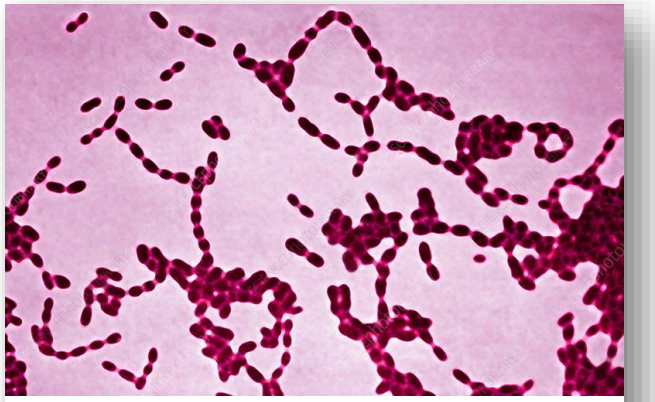


Chancroid

Chancroid

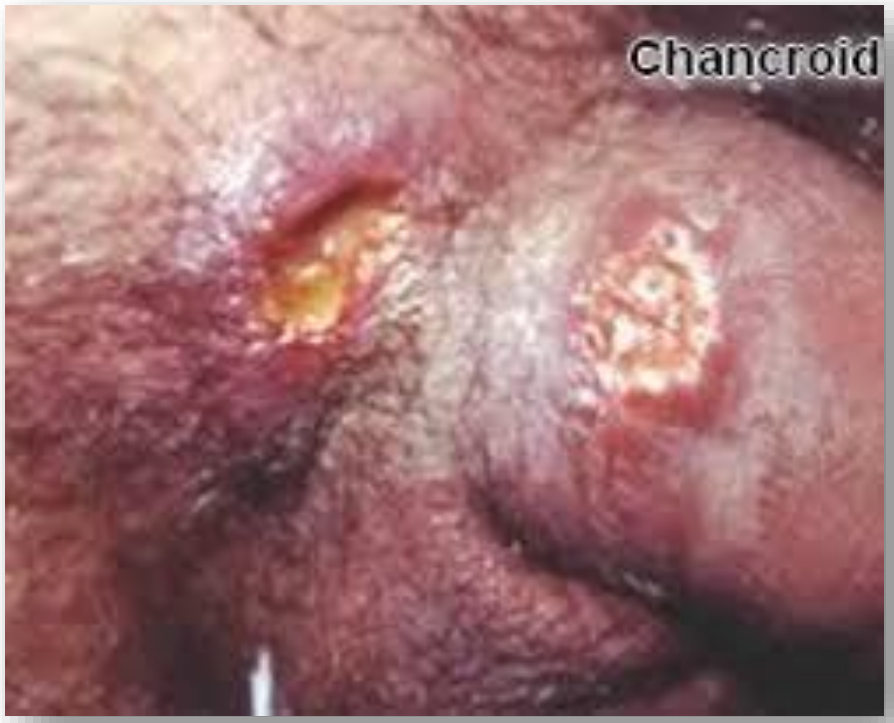
سنوات (6)

- **Causative agent**: Haemophiles ducreyi – Gram negative bacteria
- **Clinical presentations**: mostly painful genital ulcers, often multiple with tender, painful lymphadenopathy mostly unilateral (bobbo)
- **Diagnosis**:
  - Smear with Gram stain (appears as school of fish under microscope)
  - Culture
  - Laboratory tests to rule out other ulcerative sexually transmitted diseases like syphilis and herpes
- **Incubation period**: 3-7 days
- Uncommon sexually transmitted disease



school of fish under microscope

Chancroid



Chancroid

Painful, tender papules and ulcers with pus

Bubo

Tender, unilateral Lymphadenopathy

Treatment of Chancroid

- Azithromycin 1g orally single dose or
- Ceftriaxone 250mg IM single dose or
- Ciprofloxacin 500mg orally twice daily for three days or
- Erythromycin 500mg orally t.i.d for 7 days
- Partner must be treated, and the patient should be examined for other STDs.

سنوات (1)

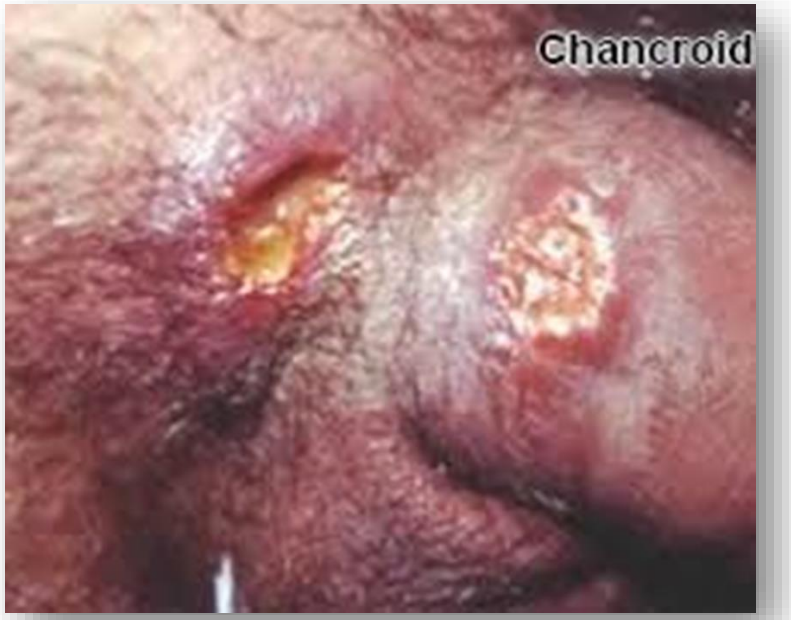
Patient present with painful unilateral tender inguinal lymph node

- What is the diagnosis
  - Chancroid

سنوات (1)

- What is the causative agent of the lesion
  - *Haemophilus ducreyi*

سنوات (6)

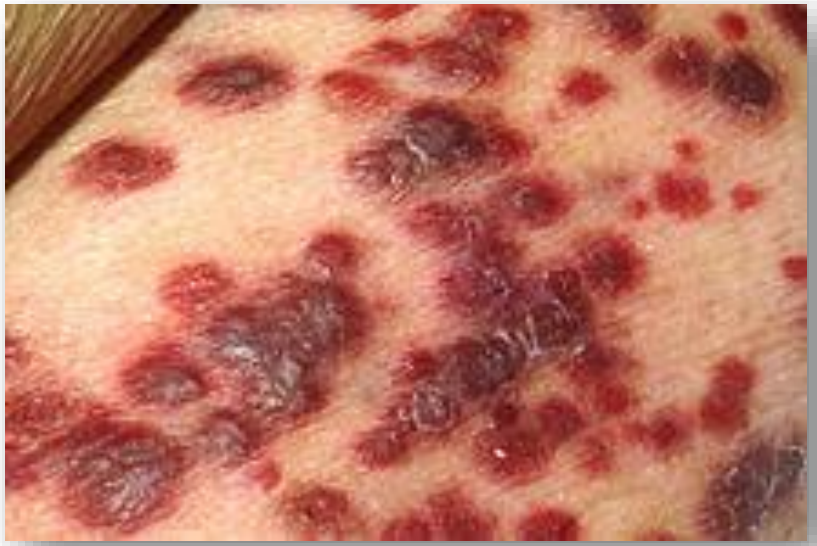


AIDS

Mention 5 skin manifestation with ADIS

1. Oral candidiasis extending into the oesophagus
2. Kaposi’s sarcoma
3. Hairy leukoplakia
4. Eosinophilic folliculitis of AIDS
5. Proximal onychomycosis
6. Severe seborrheic dermatitis
7. Opportunistic infections
8. Severe bacterial ,viral and fungal infections
9. Pre-existing psoriasis may become severe and extensive in AIDS patients

Skin manifestations of AIDS

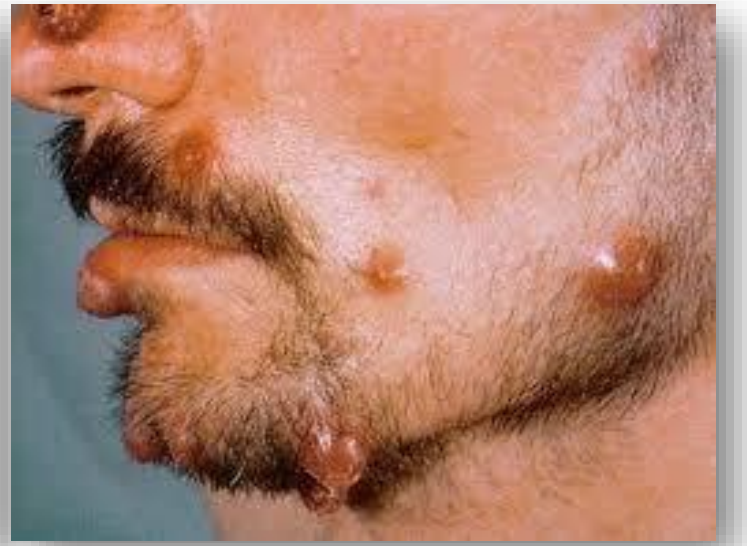


Kaposi’s sarcoma

Vascular tumor appearing as dull red plaques, diagnosed by skin biopsy.

Hairy leukoplakia

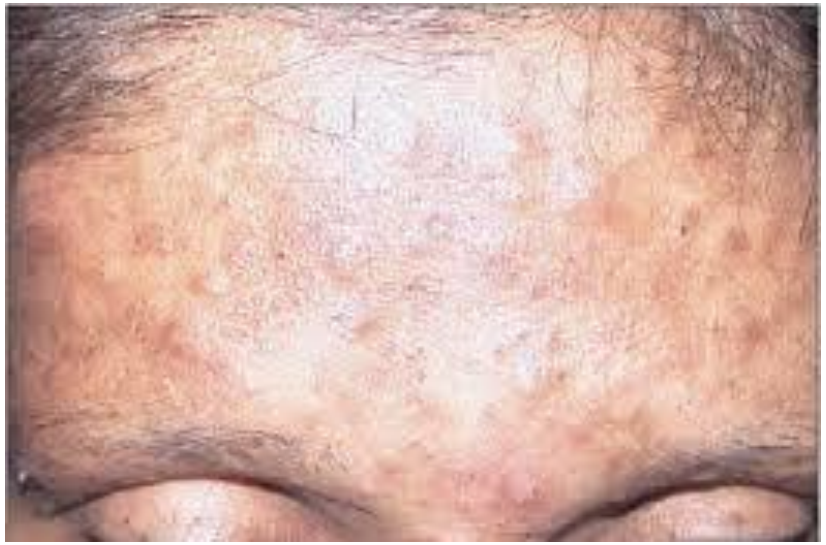
Whitish verrucous at the edge of the tongue due to EBV or HPV in AIDS patients.



Bacillary angiomatosis

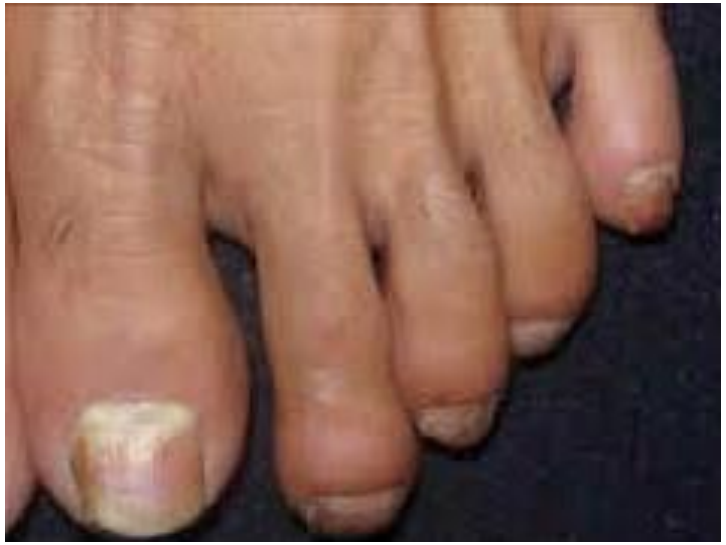
due to opportunistic infection by Bartonella.

Skin manifestations of AIDS

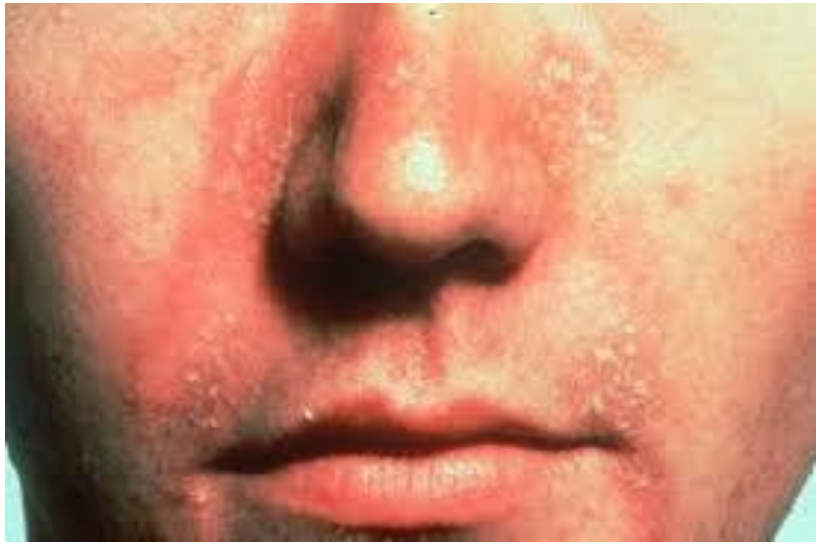


Eosinophilic folliculitis

Very itchy inflammatory infiltrate which is seen under the microscope occurring in the face , upper chest and upper back



Proximal onychomycosis



Severe seborrheic dermatitis

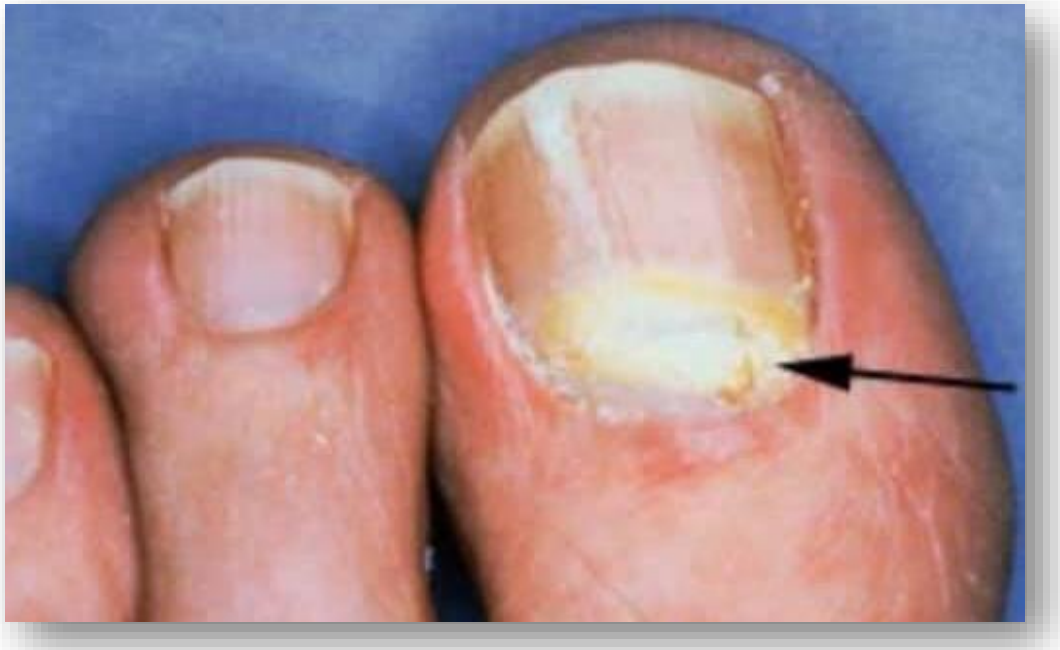
Erythema on the nasolabial folds and face

What disease should be ruled out in this case

A. Syphilis

B. AIDS

C. Psoriasis



Proximal onychomycosis

Acne & Rosacea

Acne

شرح

Acne

سنوات (2)

❖Factors that induce the pathogenesis of Acne vulgaris

1. Hormonal role, androgens, Testosterone, DHEAS
2. Increased sebum production.
3. Hyper-cornification (increase in keratin formation) of the pilosebaceous duct (infundibulum).
4. Role of *Propionibacterium acne* - enzyme production (lipase).

سنوات (4)

❖Primary lesion of acne vulgaris

- Comedone

❖Evolution of acne

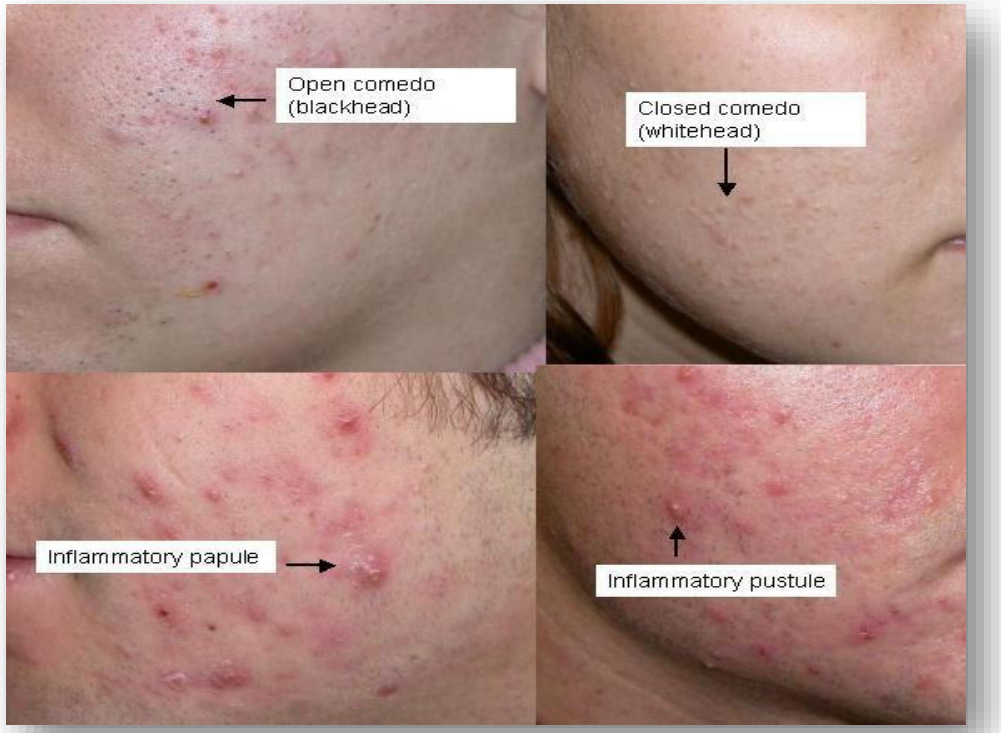
- Non inflammatory lesion (Comedones) ⟶ inflammatory lesion (Papule ⟶ Pustule ⟶ Cyst) ⟶ Sequelae (Scar, hyperpigmentation, erythema)
- Acne must be treated in its early stage to avoid those Sequelae.

شرح

Acne

❖Comedones

- White head comedon (closed comedon), will develop into inflammatory lesions
- Black head comedon (open comedon), will not develop into inflammatory lesions because they are open

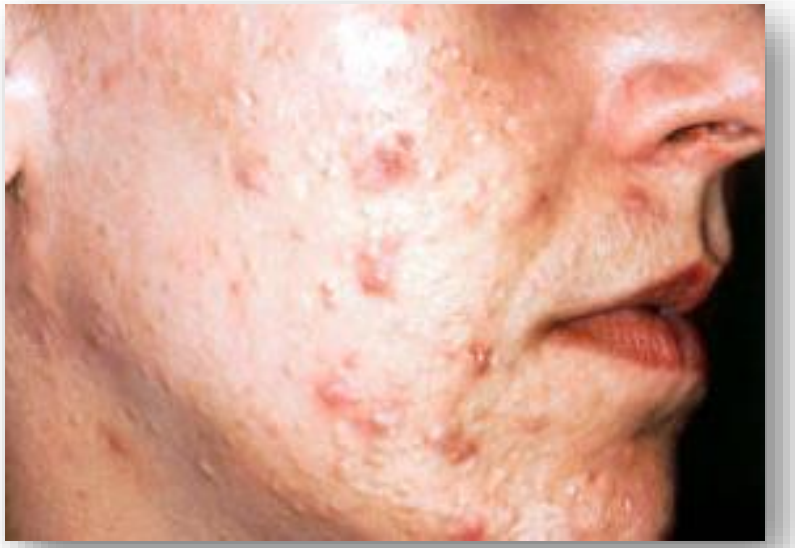


سنوات (3)

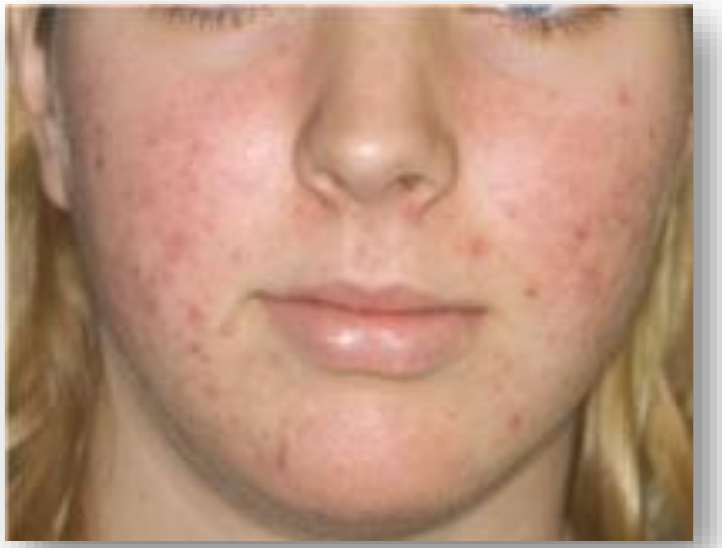
❖Mention 3 topical treatments in acne vulgaris

1. Retinoids
2. Antibiotic
3. Keratolytic
4. Benzoyl peroxide

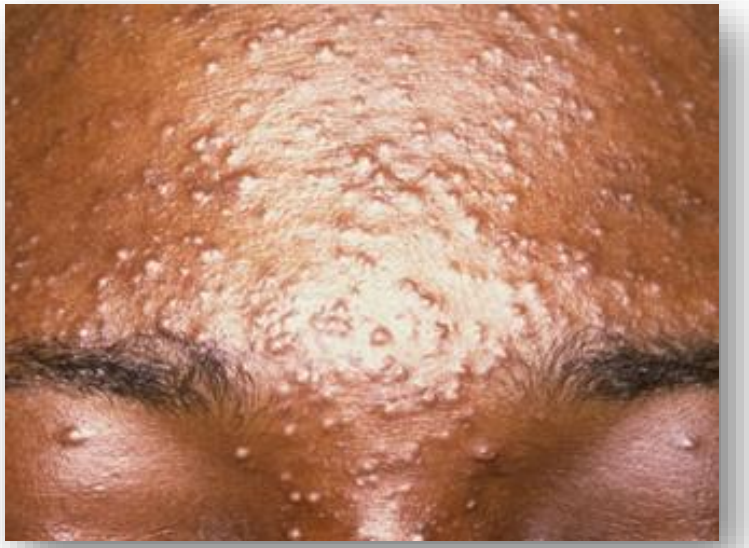
Acne



White and black comedones, and inflammatory papules and pustules.

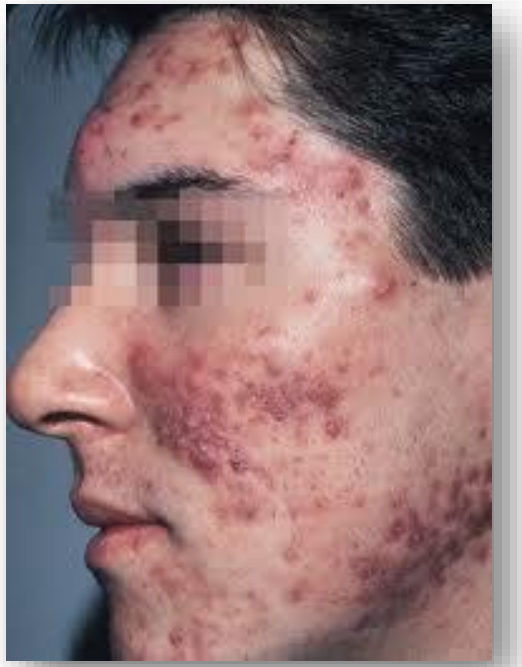


Erythema and hyperpigmentation.

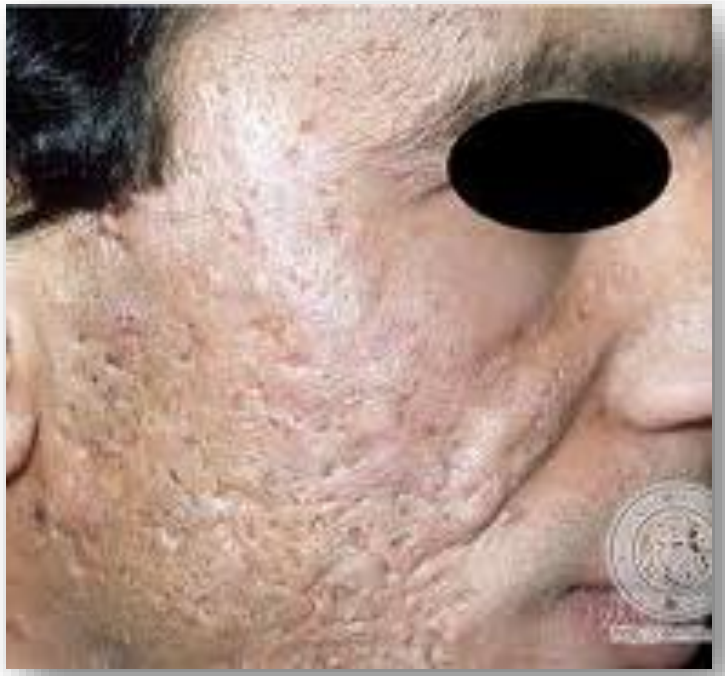


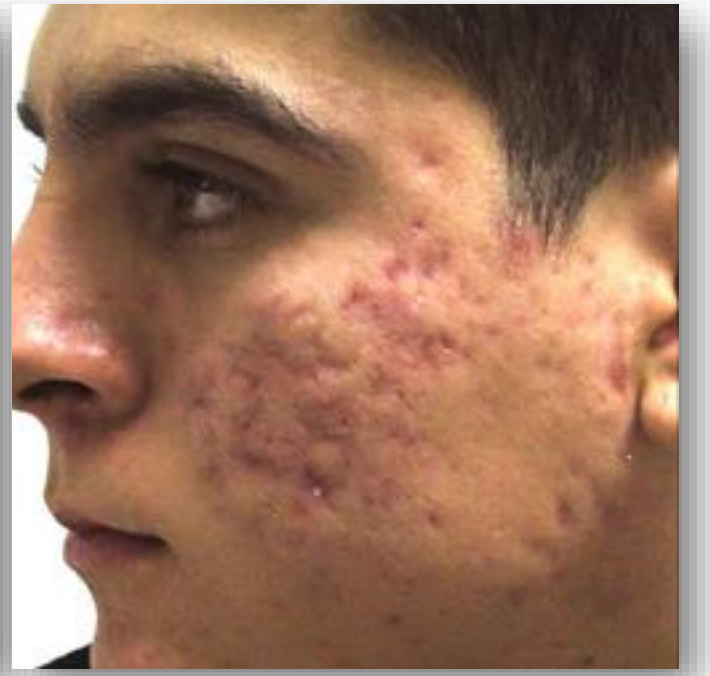
White Comedonal acne.

Acne



Inflammatory lesions:
papules and nodules.
Some scars and cysts.



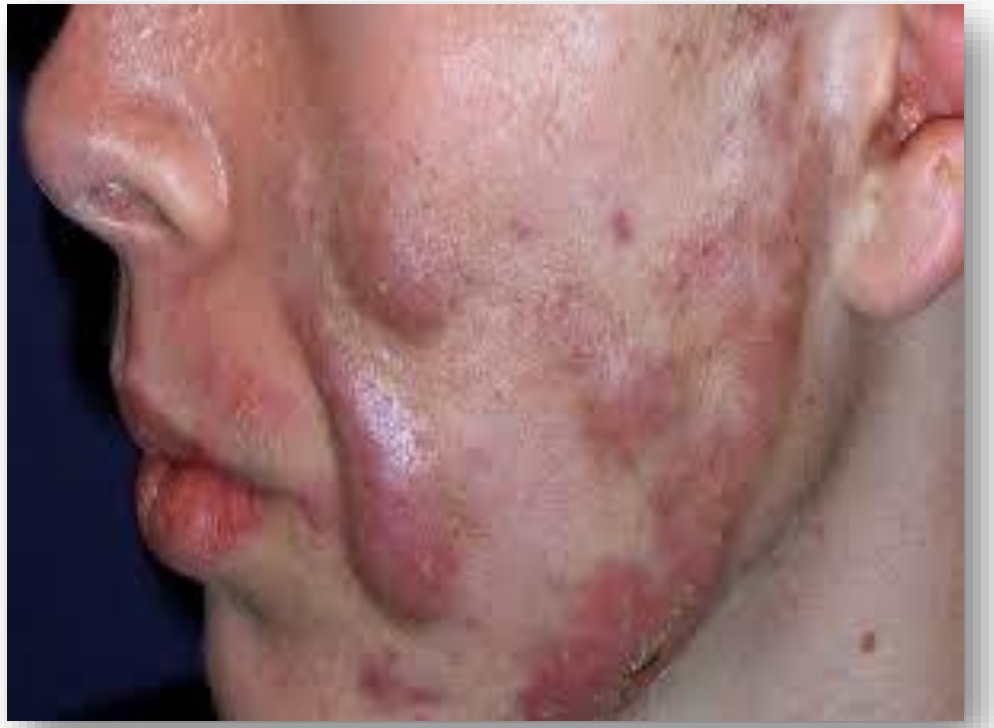


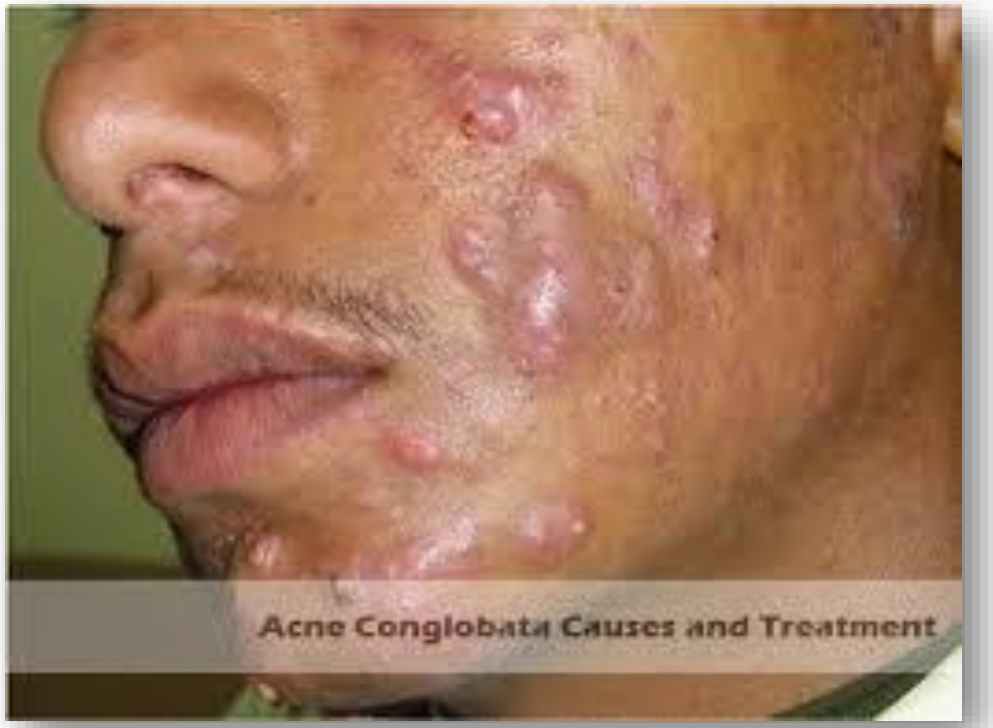
Scarring acne.
Indication for Vitamin A derivatives use.

Mention 5 variants of acne

1. Acne vulgaris.
2. Acne conglobata (Nodulocystic).
3. Acne fulminans.
4. Acne mechanica.
5. Acne excoriee.
6. Drug induced acne.
7. Occupational acne.
8. Neonatal acne.
9. Infantile acne.
10. Late onset acne (adult type)

2. Acne conglobata-Nodulocystic acne



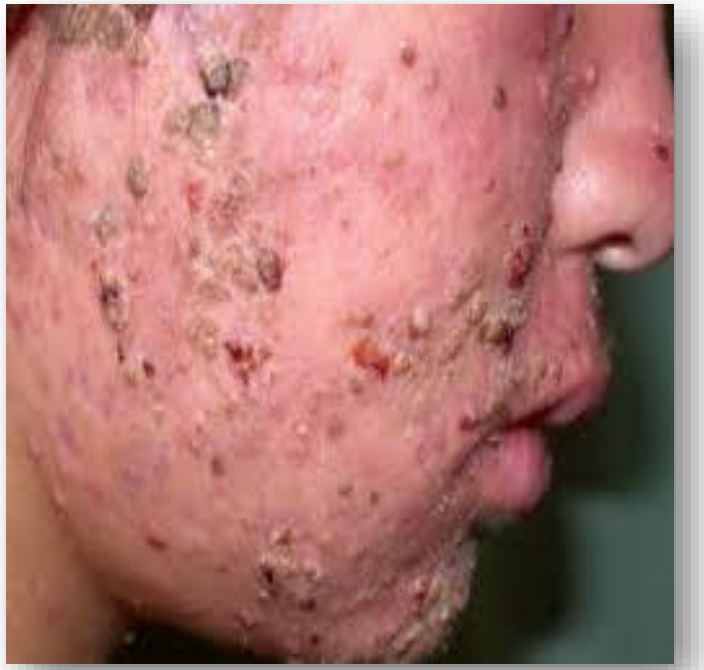


Nodulocystic lesions

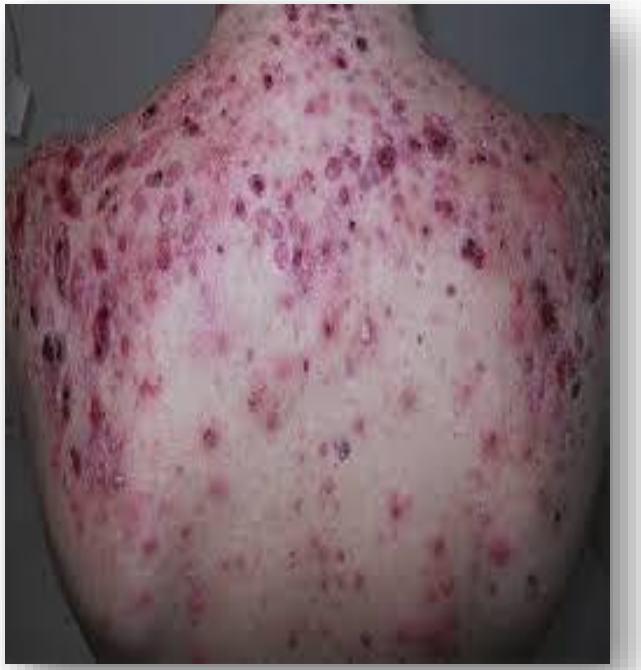
Severe form of acne

3. Acne Fulminans

- Severe rare form with cystic lesions and systemic symptoms.
- Young men 13-16 yr.
- **Treatment**: Isotretinoin with systemic steroid (the only type of acne that is treated with steroids).



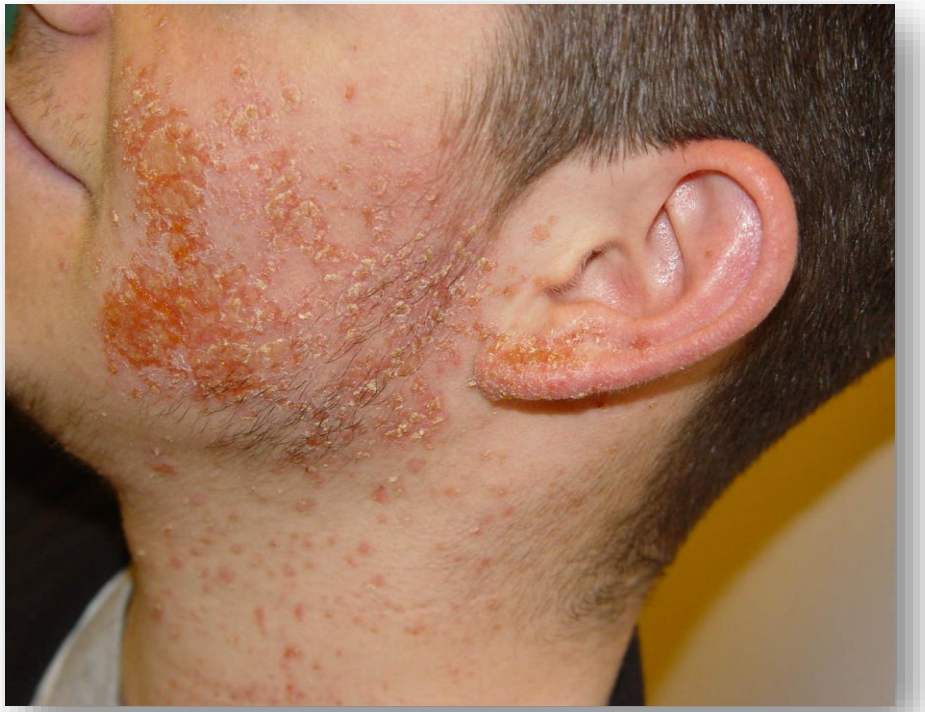
Young man with inflammed cystic lesions.



Inflammed cystic lesions on the back.

4. Acne Mechanica

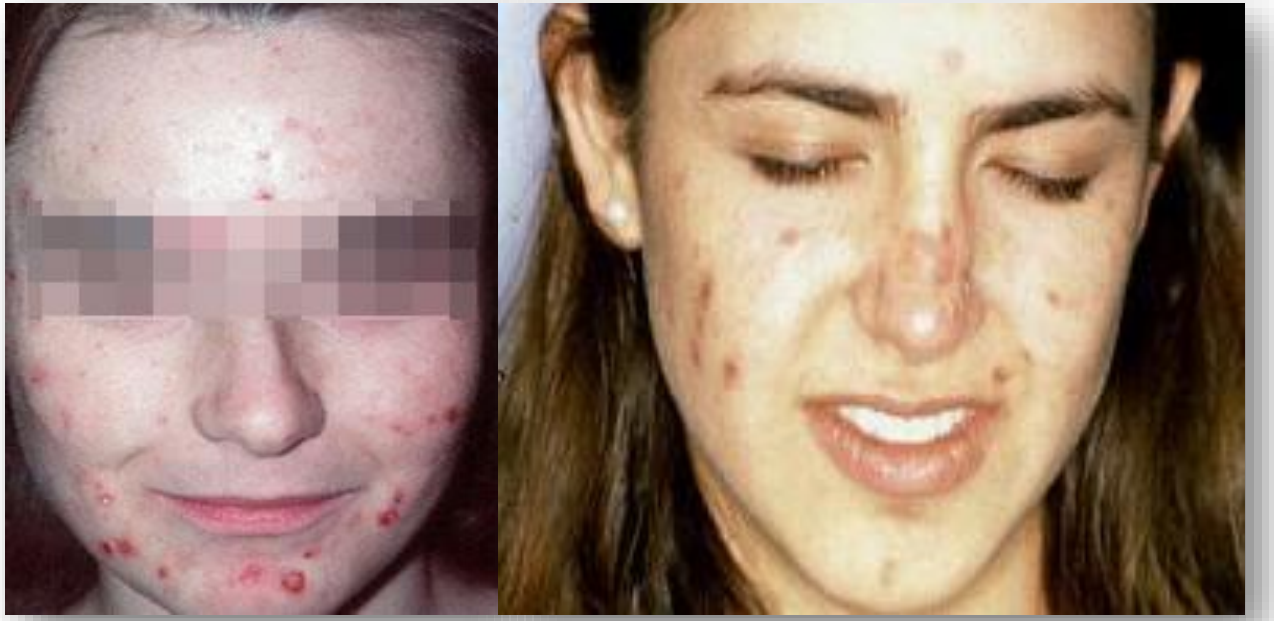
- Repeated mechanical and frictional obstruction.
- Rubbing by helmets, chin straps, masks.
- **Treatment**: Eliminating these factors.



Pustules and papules at the site of contact.
Yellowish discoloration indicates secondary infections.

5. Acne Excoriee

- Young women.
- Underlying psychiatric components.
- Antidepressants or psychotherapy may be indicated.



Excoriations only.
No comedones, papules or pustules.

شرح

6. Drug Induced Acne

سنوات (1)

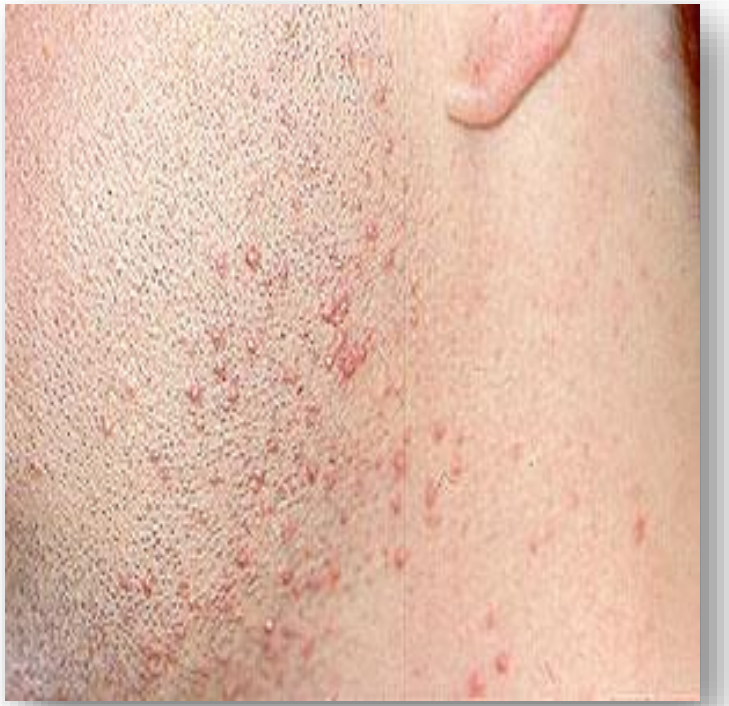
❖**Mention 2 drugs that can induce acne**

- Anabolic steroids – Danazole, Stanazole
- Corticosteroids
- phenytoin
- Lithium
- Iodides, bromides, Vit. Supplements, cough compounds and sedatives
- Azathioprine,Vit.B12, cyclosporine

سنوات (1)

❖**Primary lesion of drug induced acne**: Monomorphic eruption of papules and pustules

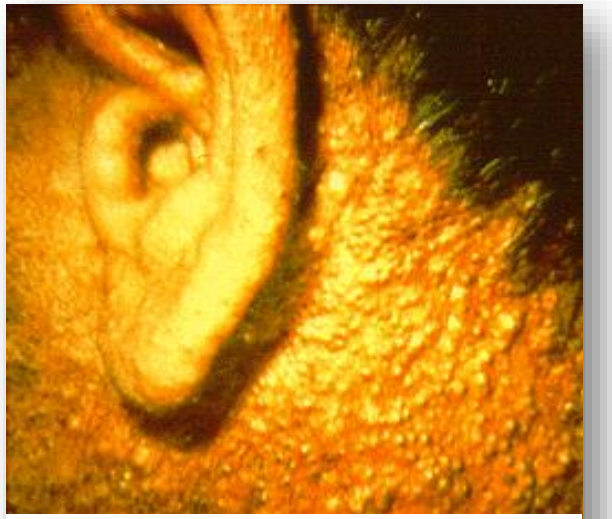
❖Steroid induced acne (in spring): after oral or topical use of steroids



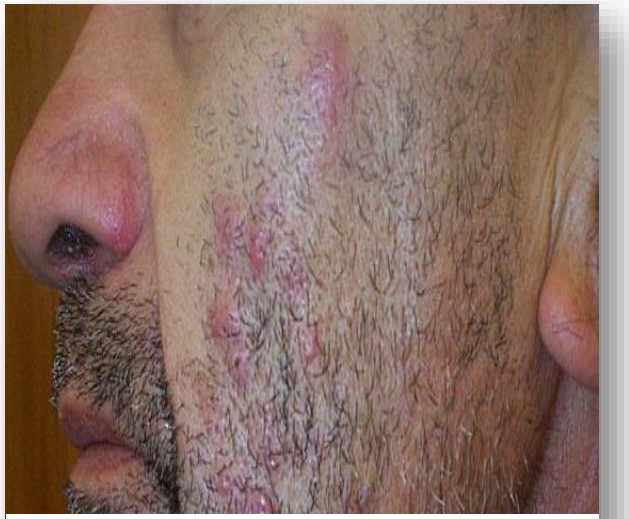
Monomorphic papules and pustules due to steroids.

7. Occupational Acne

- Comedones dominate usually.
- **Chloracne**:
  - Acne-like eruption of blackheads, cysts, and pustules associated with exposure to certain halogenated aromatic compounds, such as chlorinated dioxins and dibenzofurans
  - Malar, retro-auricular, mandibular areas, axillae, sacrum, buttock are involved



Comedone predominant, some papules.

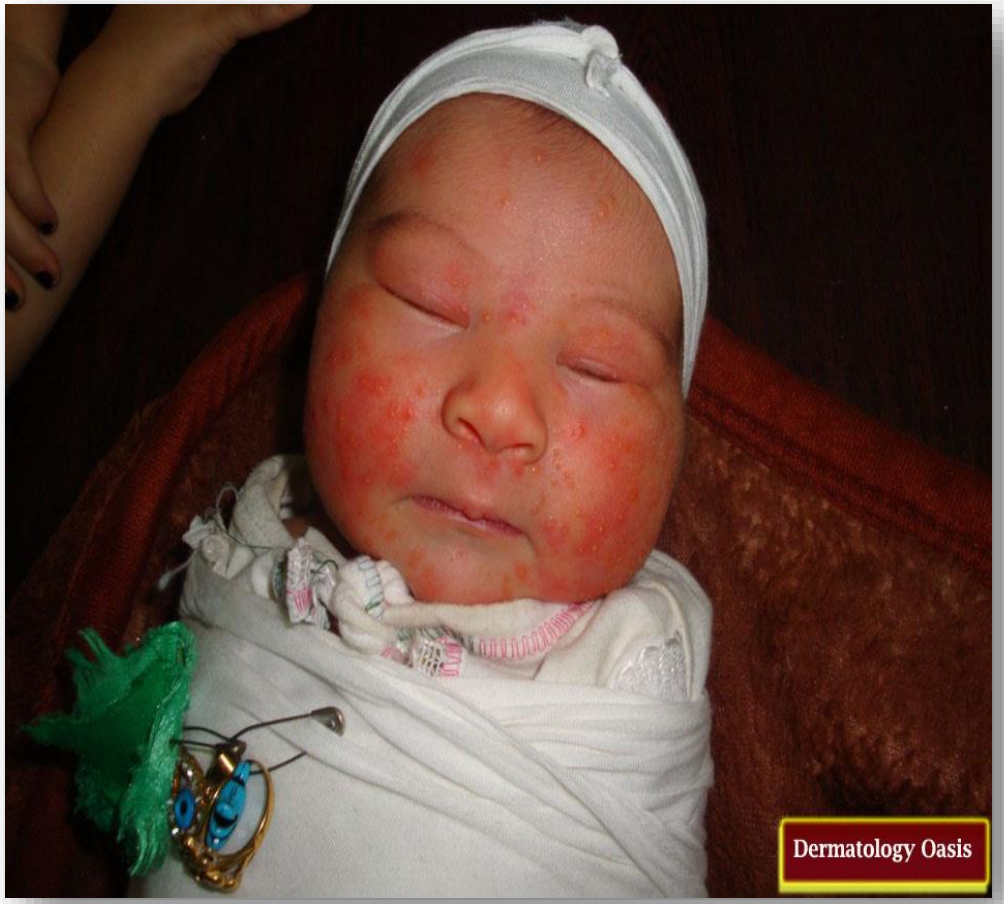


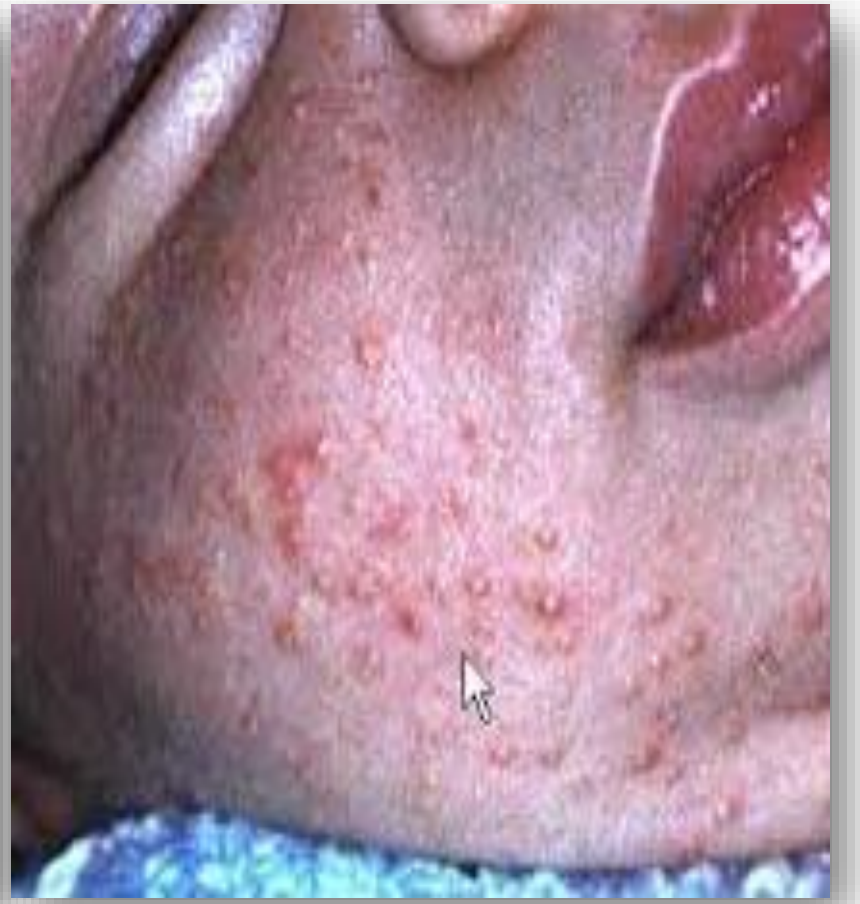
Inflammatory lesions on the malar area and cheeks.

8. Neonatal Acne & 9. Infantile Acne

8. Neonatal Acne	9. Infantile Acne
Hormonal factors from mother and Malassezia	Hormonal factors DHEA
Appears at the ages of 2 weeks	present at 3-6months of age
resolves at the age of 3 months	Resolves within 1-2 years
Small papules on cheeks	More comedones than in neonatal
Treatment: 2% ketoconazole, Benzoyl Peroxide	**Treatment**: Tretinoin, Benzoyl Peroxide

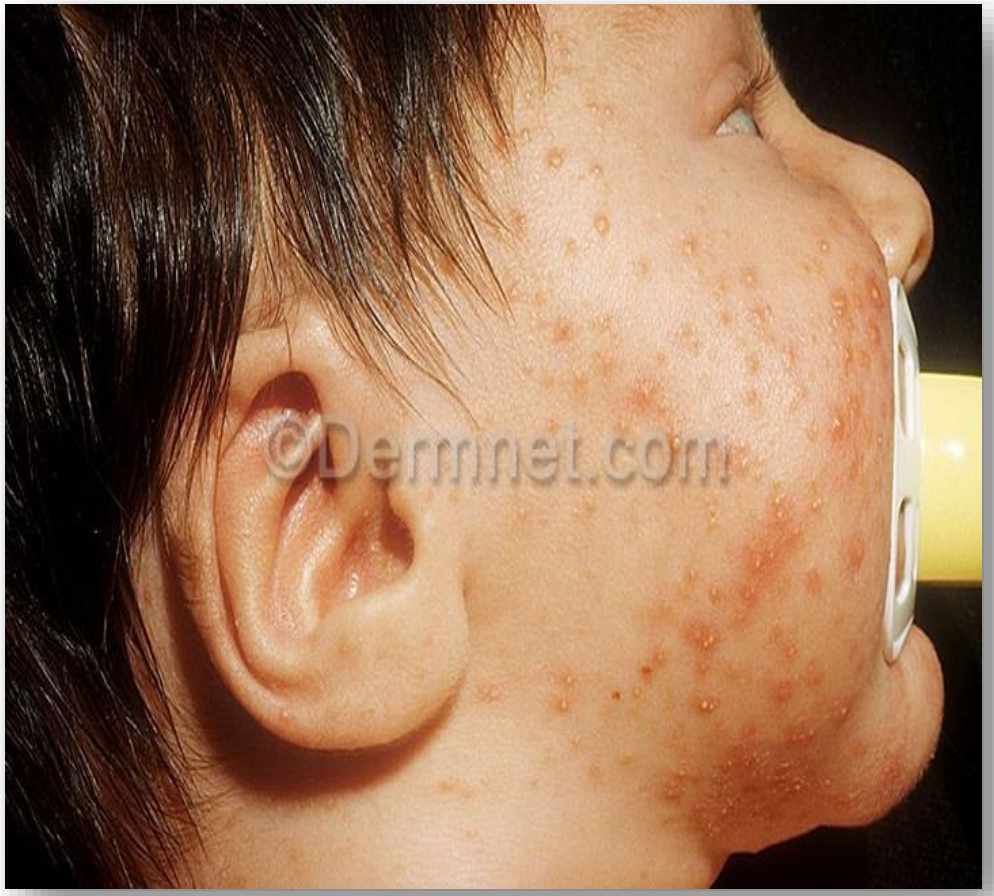
8. Neonatal Acne



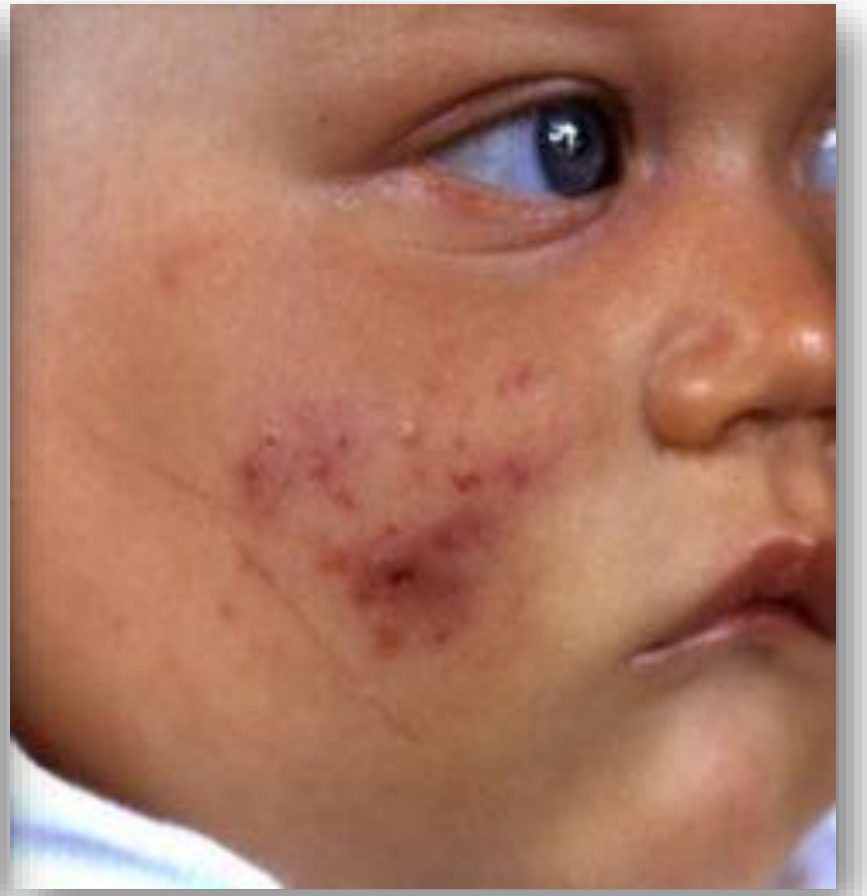


Papules and few comedones on the cheeks.

9. Infantile Acne



Comedones, papules, erythema and hyperpigmentation.



Comedones and papules.

10. Late onset Acne (Adult acne)

Papules, erythema and Hyperpigmentations.
Must do investigations to rule out causes of androgen excess.
Usually affect the jaw area and the neck.

Mention 3 investigations for late onset acne:

1. Hyperandrogenism should be suspected in female with hirsutism, irregular cycles, severe acne, abrupt onset, coarse voice.
2. Androgenetic alopecia: Free testosterone, DHEAS, 17-hydroxyprogesterone.
3. AM serum cortisol level if hypercortisolism is suspected.
4. Elevated DHEAS and 17-OH-progesterone suggest adrenal source of excess androgen.
5. DHEAS 4000-8000g\ml or 17-OH progesterone level > 3ng\ml congenital adrenal hyperplasia.
6. Elevated testosterone suggest ovarian source.
7. Increased LH /FSH ratio to > 2-3 in polycystic ovary syndrome.
8. Serum testosterone >200ngIdl indicates ovarian tumor.
9. Ovarian US: Ovarian cysts.

Differential diagnosis of acne

1. Milia (small facial cysts)
2. Sebaceous hyperplasia (appear as papules)
3. Folliculitis
4. Pseudofolliculitis
5. Trichoepithelioma, syringoma
6. Seborrheic dermatitis
7. Rosacea
8. Perioral dermatitis

شرح

Treatment of Acne

- Topical: 1st line of treatment
  - Retinoids (the best choice), Benzoyl peroxide
  - Antibiotic (Erythromycin, Clindamycin)
  - Keratolytic: Salicylic acid

سنوات (1)

- Systemic:
  - Antibiotics (Antibacterial + Anti inflammatory) : 1st line
    - Tetracycline, Doxycycline, Erythromycin, Azithromycin
  - Retinoids : 2nd line
    - Isotretinoin (the best treatment but last choice)

- Lesions may flare in the first 2 weeks of treatment.

شرح

Treatment of Acne

Overview of acne treatment	
Severity	**Treatment**

Mild (e.g., comedonal)	• **Topical benzoyl peroxide** (comedolytic and bactericidal effects secondary to a release of oxygen free radicals) OR topical retinoids
	• Topical combination therapies
	  ◦ Benzoyl peroxide AND antibiotic/retinoid
	  ◦ Benzoyl peroxide AND antibiotic AND retinoid

Moderate (e.g., papular/pustular)	• Combination therapy:
	  ◦ Topical benzoyl peroxide AND **topical retinoids**/ antibiotics
	  ◦ Oral antibiotic (tetracycline-class) may be added
	  ◦ Combined oral contraceptives may be added (in females)

Severe (e.g., conglobata)	• **Oral isotretinoin** • Or oral antibiotics (tetracycline-class) AND topical combination therapy • Combined oral contraceptives may be added (in females)

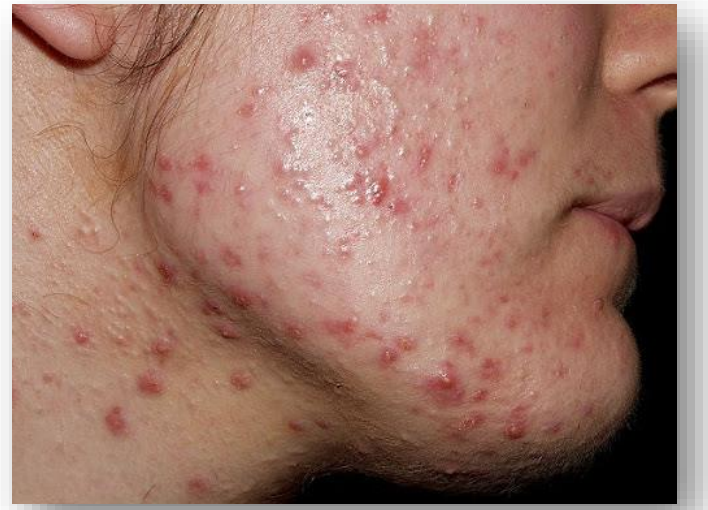
شوفوا صورة الحالة وبناءا عليها قرروا قديش الseverity وبناءا عليها بكون العلاج

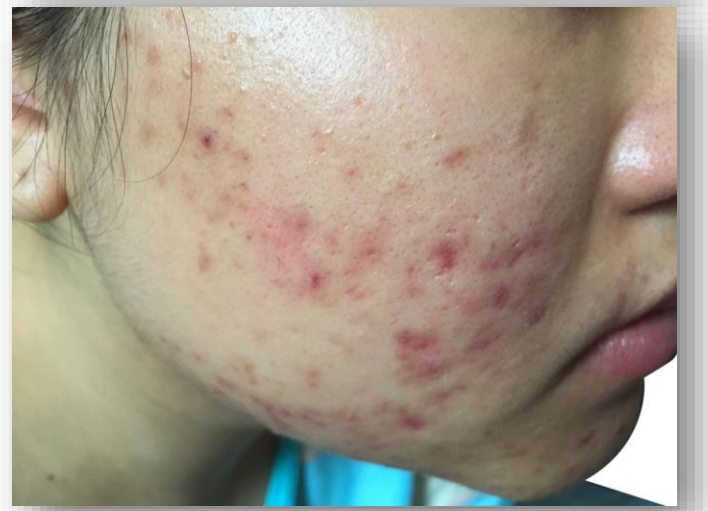
Isotretinoin (Roaccutane)

- For severe cases :
  1. Nodulocystic acne not responding to first line measurements
  2. Scarring acne
  3. Dysmorphophobic acne
- Effective but toxic.
  - Teratogenic – cause fetal anomaly in > 95% of cases (abortion or fetal anomalies).
  - Needs close follow up (liver enzymes, serum lipids, bleeding, papilledema).
  - Dose 0,5-1mg\kg, used alone without any combination with other drugs.
  - Duration 5-6 months or total accumulative dose 120mg\kg\course.

Acne vulgaris

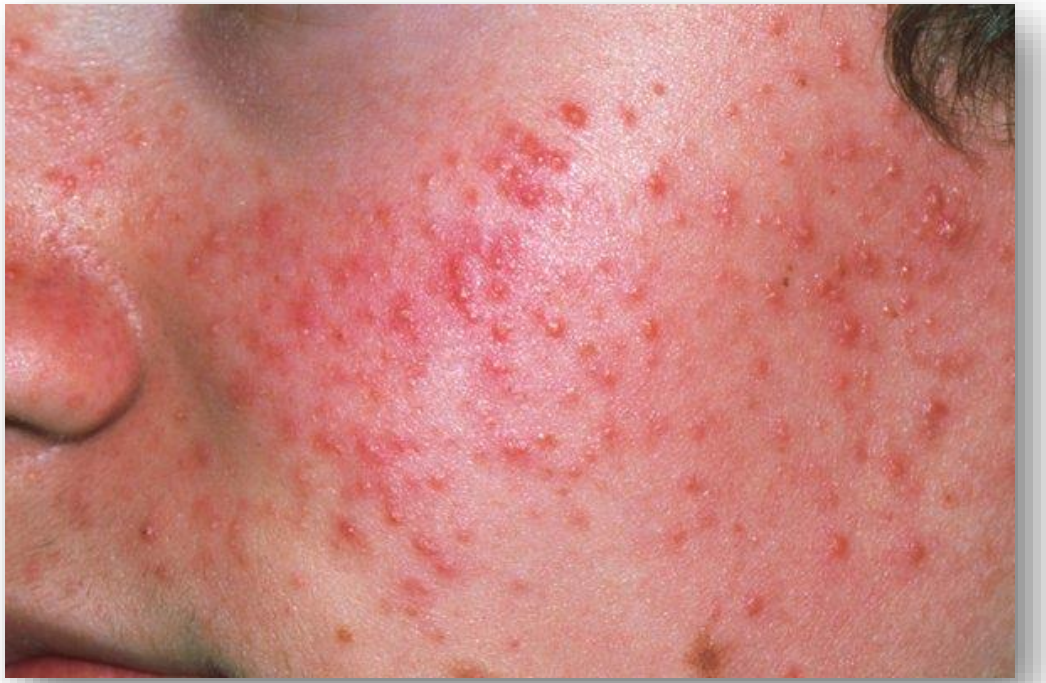
- **First line treatment**
  - Oral antibiotic, topical benzoyl peroxide and topical retinoids
- **Which drug is contraindicated ?**
  - Steroid



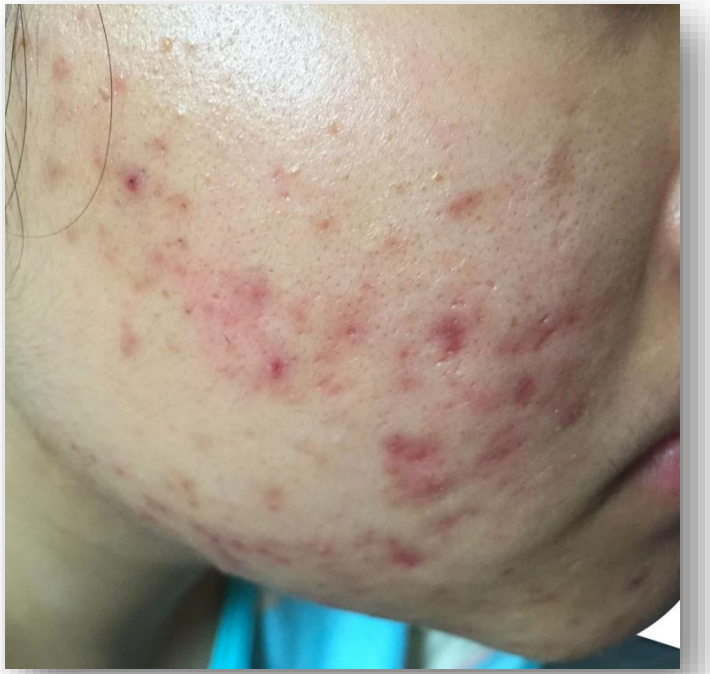


A 27-year-old married female presented with these lesions

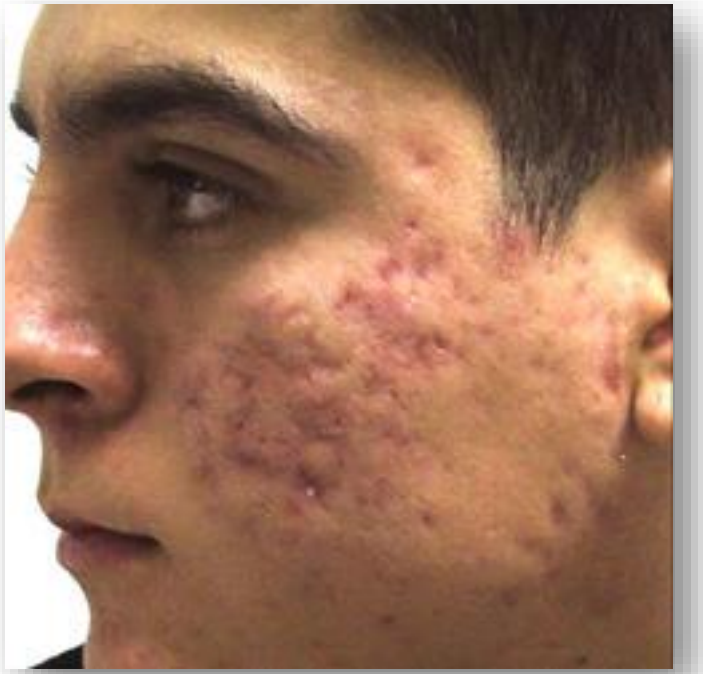
- Which drug shouldn’t be used for her case
  - Oral Isotretinoin
- Because she is married and might want to get pregnant, also the question and the picture didn’t show any of the indications for using roaccutane (isotretinoin) (scarring, failure of medical therapy,…)



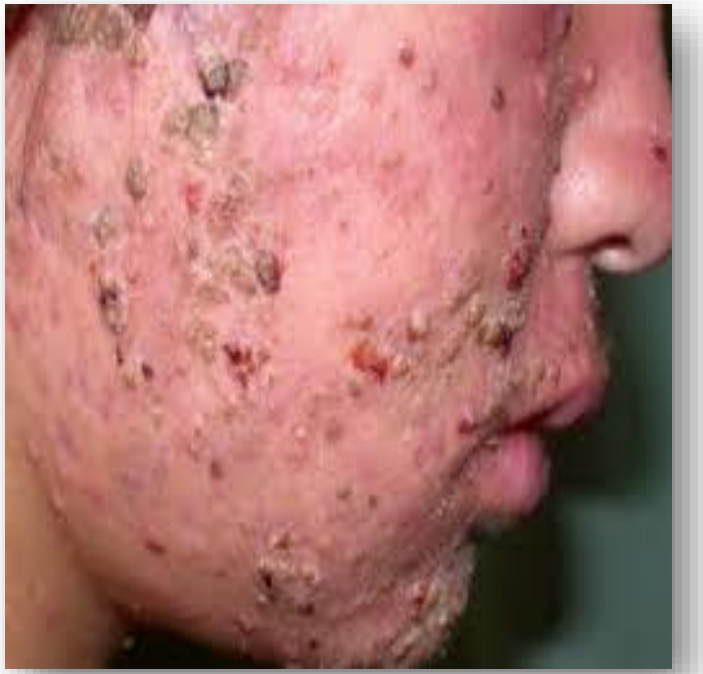
First line treatment of each of the following cases



Oral antibiotic, topical benzoyl peroxide and topical retinoids



Oral isotretinoin



Isotretinoin with systemic steroid

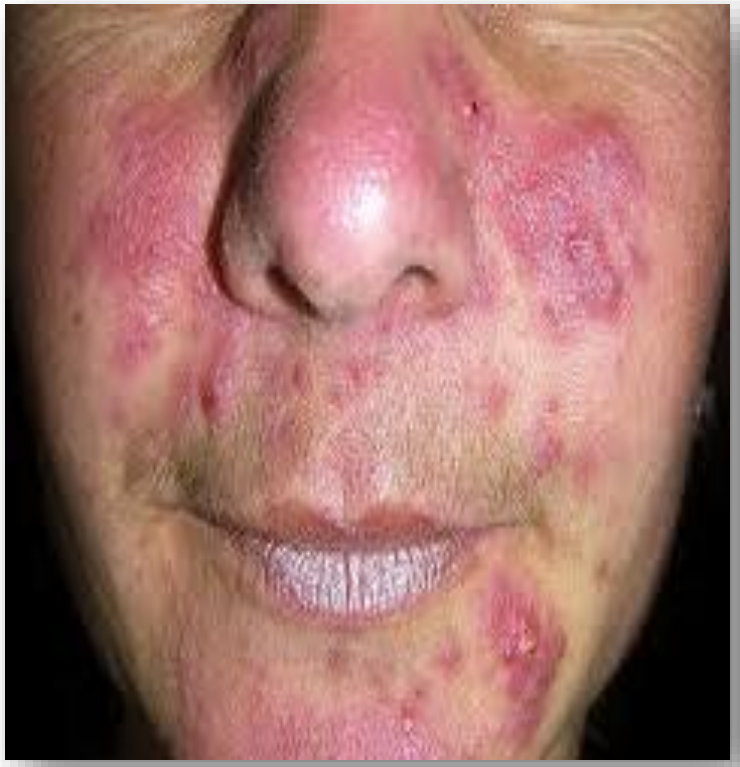
Rosacea

Rosacea

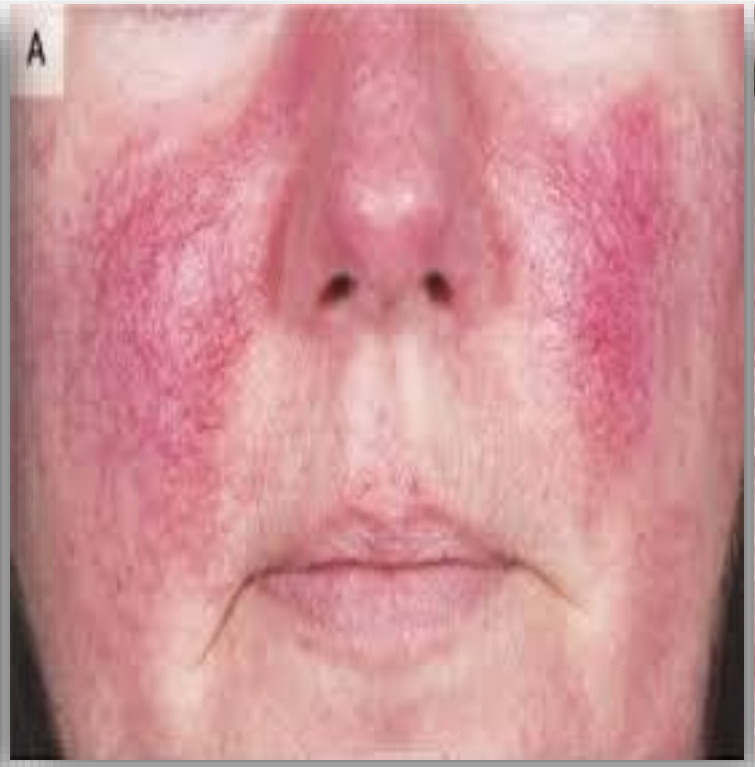
- Chronic inflammatory skin disease with characteristic Lesions on the face
- Affect mostly middle-aged women (female:male 9:1)
- **Characteristic skin lesions**:
  - Telangiectasia, erythema, papules, pustules on cheeks, forehead, nose and chin; no comedones
- **Complications**: Rhinophyma and Otophyma
- **Risk factors**:
  - Female + Fertile + Fair skin + Fatty + Forty
- **Treatment**: topical antibiotics

Rhinophyma Complication of rosacea

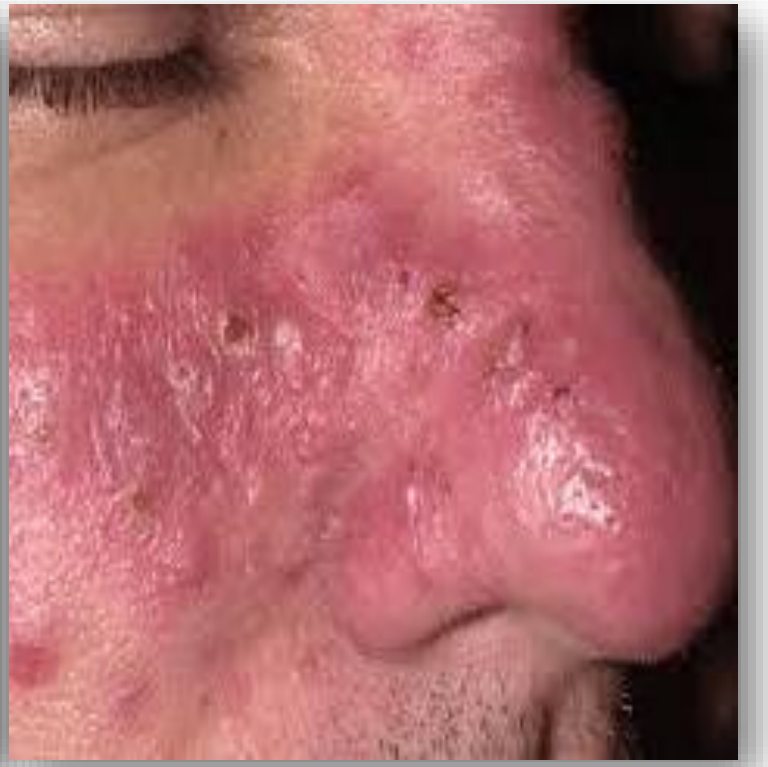
Rosacea



Erythematous papules on the cheeks.

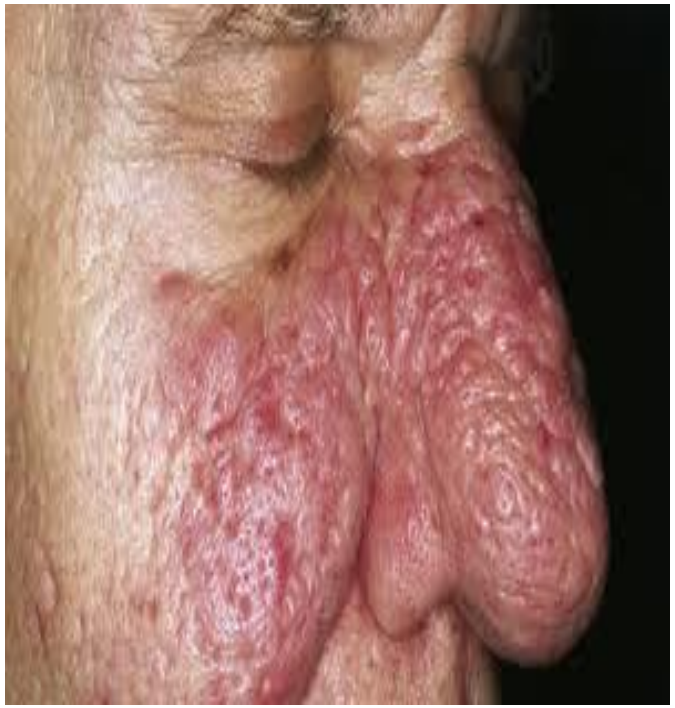


Pink papules with Telengectasia on the cheeks.

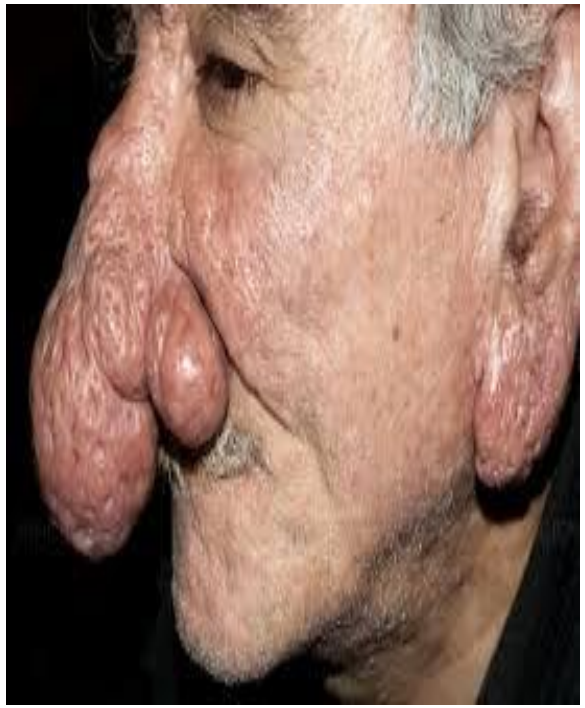


Bilateral pink papules on the cheeks, nose and chin.

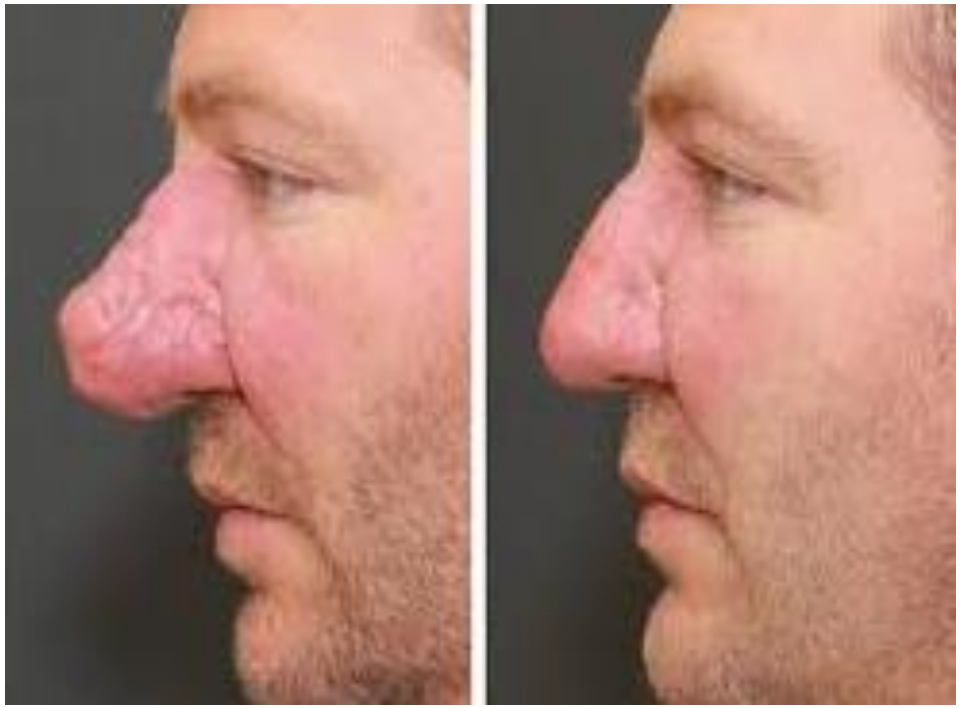
Rosacea – Complications



Rhinophyma



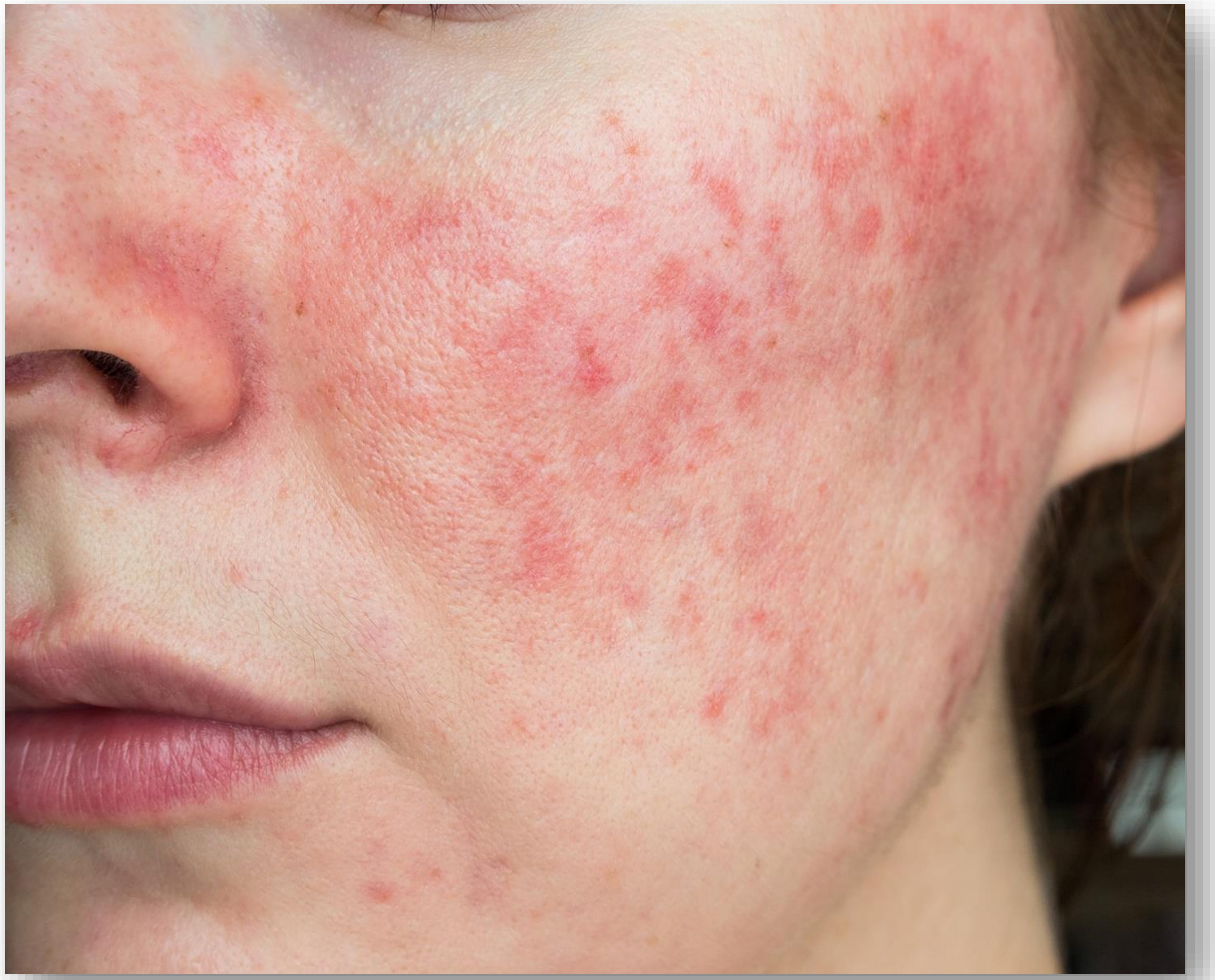
Rhinophyma & Otophyma



Rhinophyma before and after the surgical treatment.

Fair skin female, 40Y/O, no other complaint

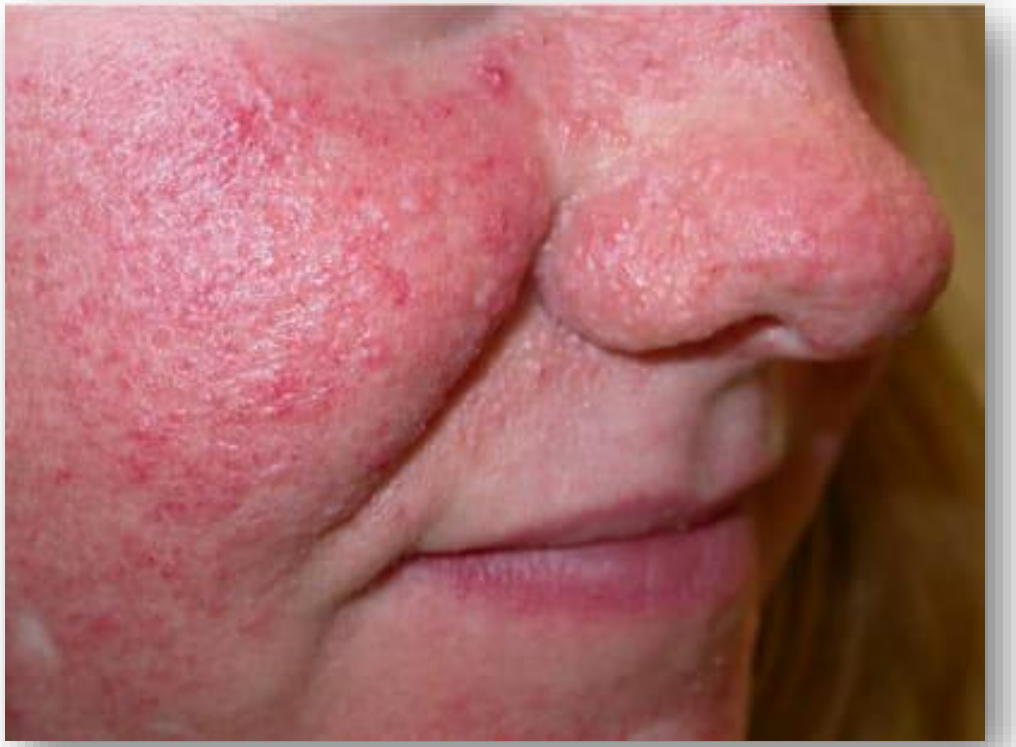
- Diagnosis
  - Rosacea
- One topical treatment
  - Metronidazole
- Mention 2 complications
  - Rhinophyma and Otophyma



سنوات (2)

Erythematous lesion for 2 years, diagnosis ?

- Diagnosis
  - Rosace
- Differential diagnosis
  - Folliculitis
  - Systemic lupus erythematosus
  - Pityriasis rosacea
  - Dermatomyositis



سنوات (1)

- **Define Rhinophyma**: enlarged, bulbous nose, seen in rosacea; due to repeated inflammation and edema

Rosacea

Rosacea
Papules and pustules with background erythema but no scaring

Acneiform skin rash

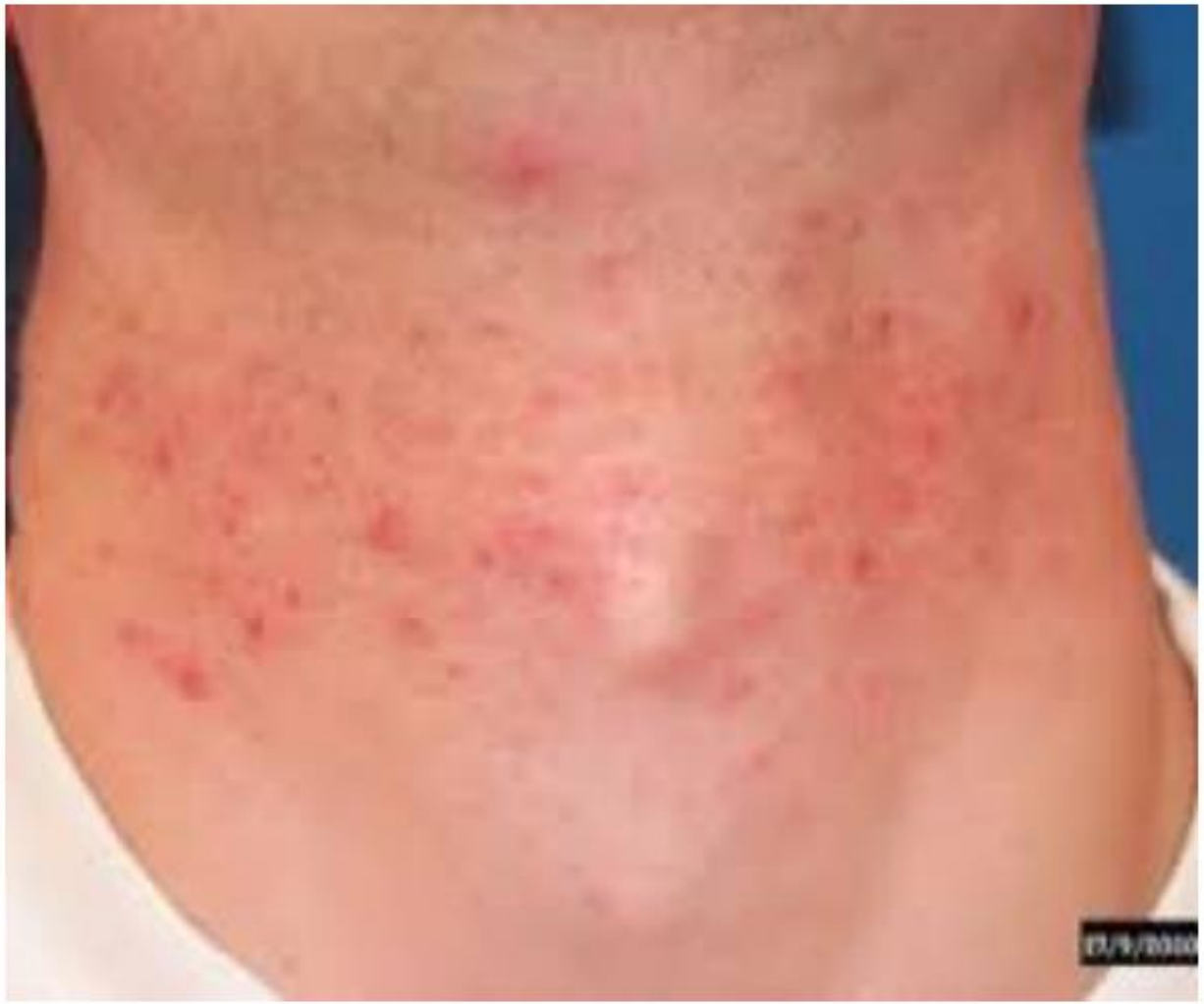
Acneiform skin rash

1. **Pseudofollicultis Barbae**: Skin reaction to hair as foreign body.
2. **Pityrosporum folliculitis**
3. **Acne keloidalis nuchae**: inflammatory keloidal lesions on the nape of the neck

Pseudofollicultis barbae

- Not a true folliculitis (not due to infection) but due to a reaction against the hair itself when its plugged inside (foreign body reaction).
- Occurs in Negroid people, Curley hair.

Pseudofollicultis



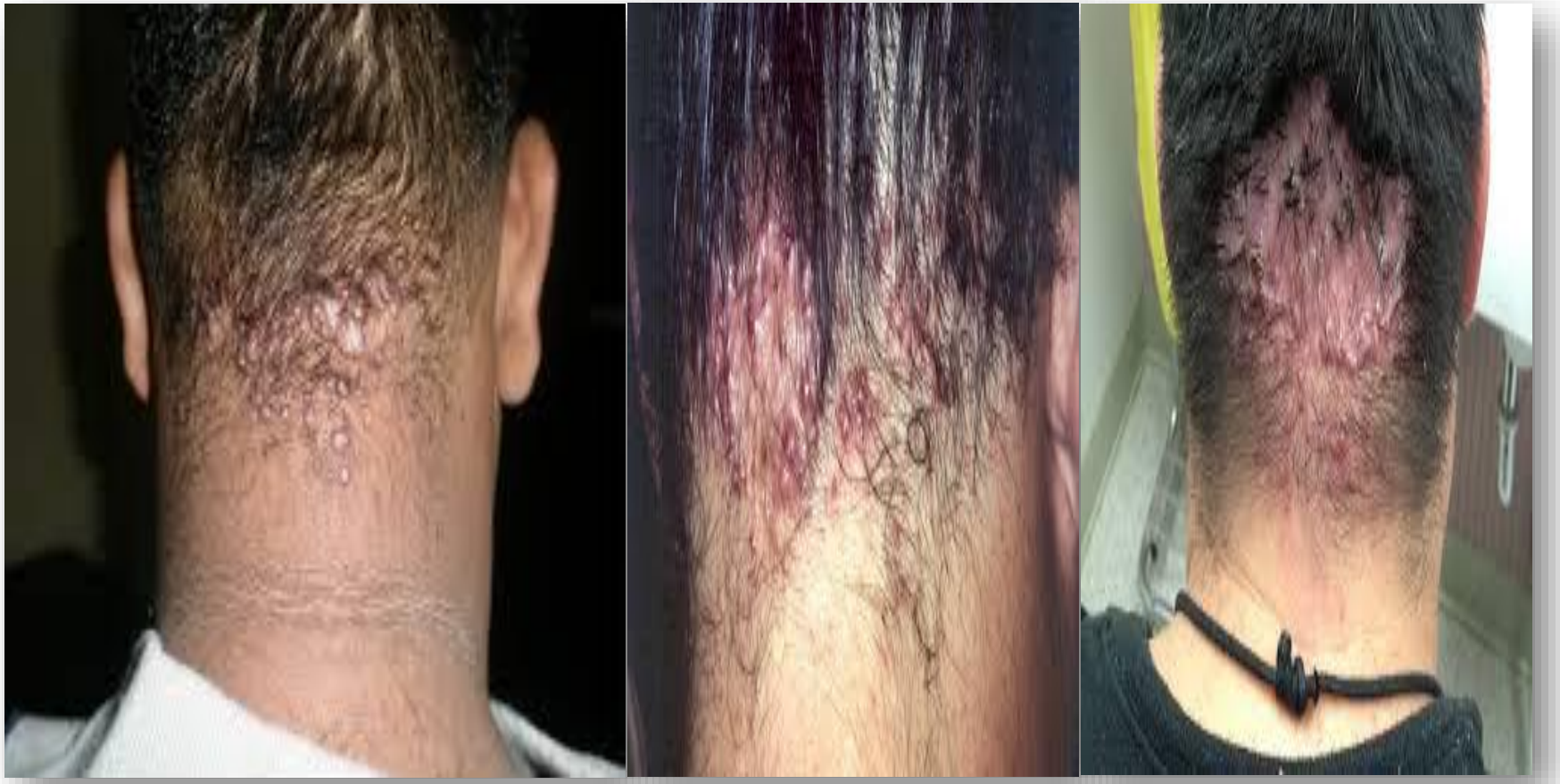
Pseudofolliculitis

Pityrosporum folliculitis

Acne form rash due to fungal infection.

Acne keloidalis nuchae

Acne form rash with scarring on the nape of the neck, could lead to permanent hair loss.



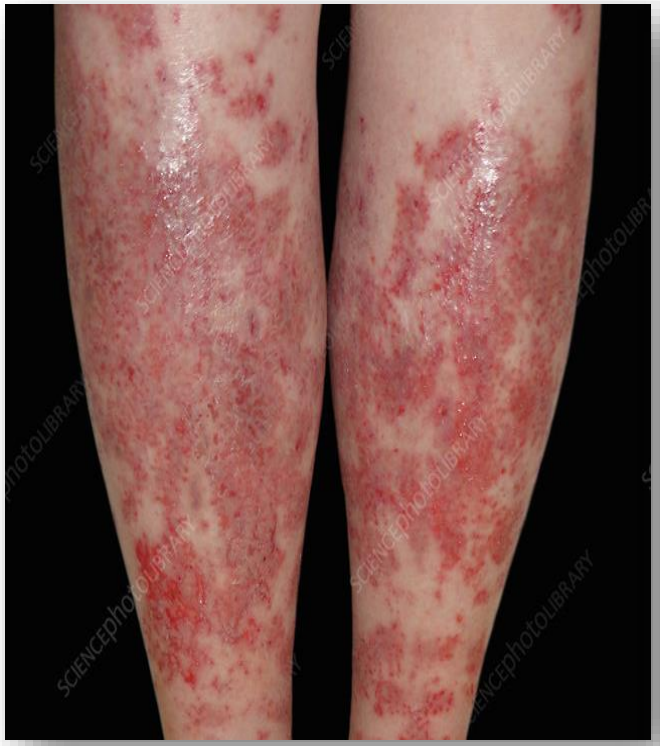
Eczema

Pruritis are discussed later

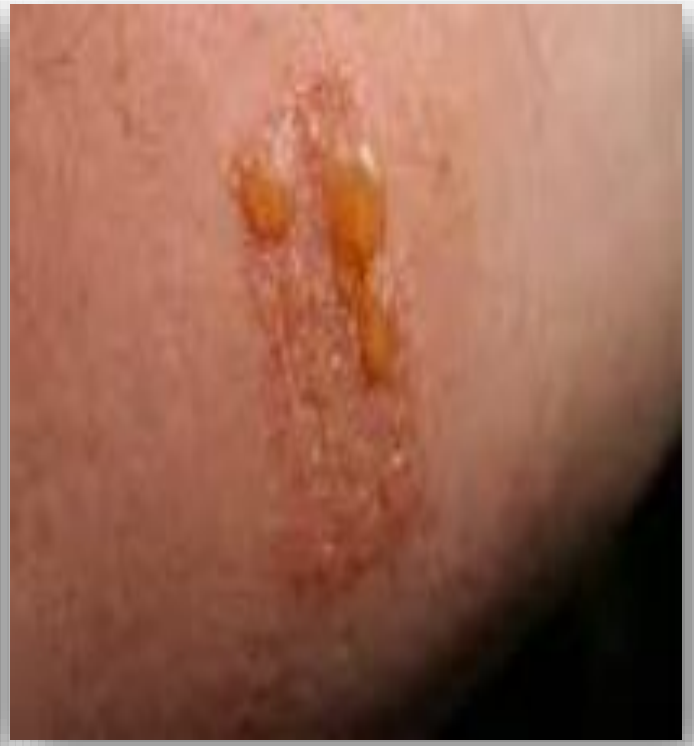
Eczema (Dermatitis)

- **Eczema can be classified according to the onset and duration into**
  - **Acute**: Recent marked erythema, marked edema with vesicle formation and oozing, marked itching
  - **Chronic**: Thick, dry scaly skin, itching during exacerbation, less edema but more thickening of the skin (Lichenification)
- **Eczema also can be classified according to the cause into**
  - **Exogenous**, such as contact allergic dermatitis
  - **Endogenous**, such as atopic dermatitis

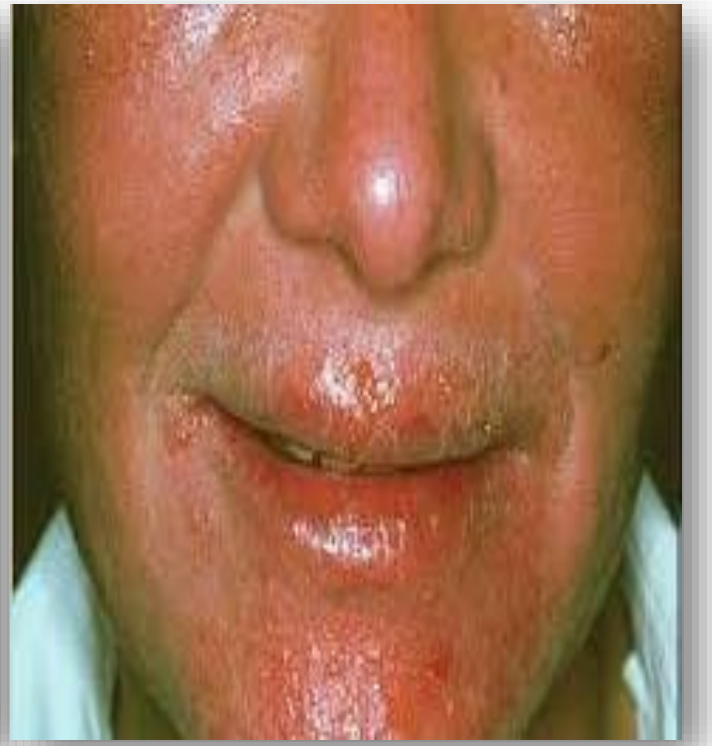
Acute eczema



Ill defined, Wet erythema with oozing

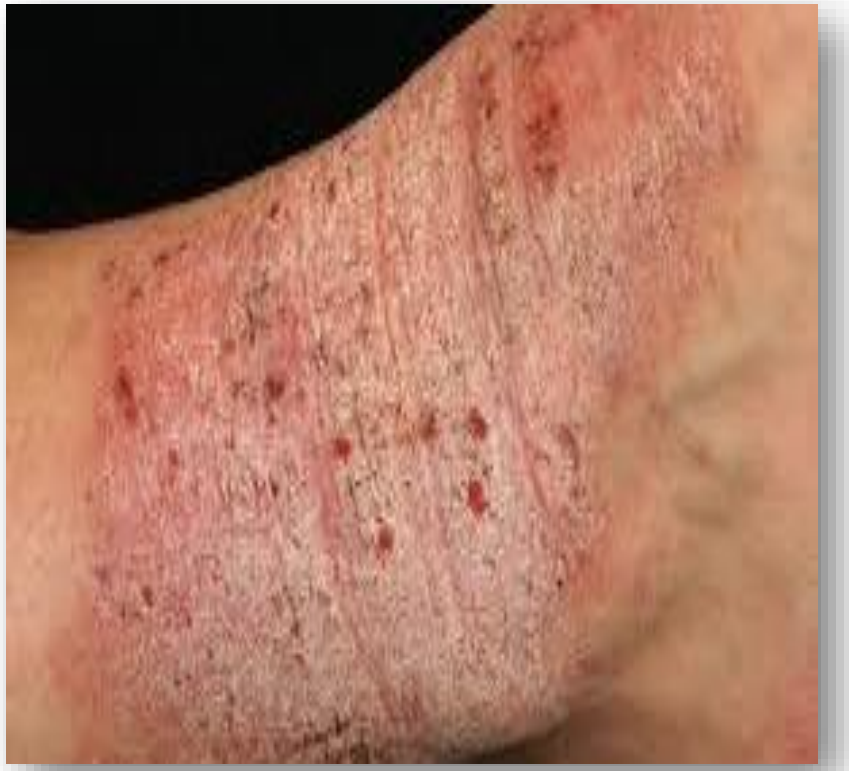


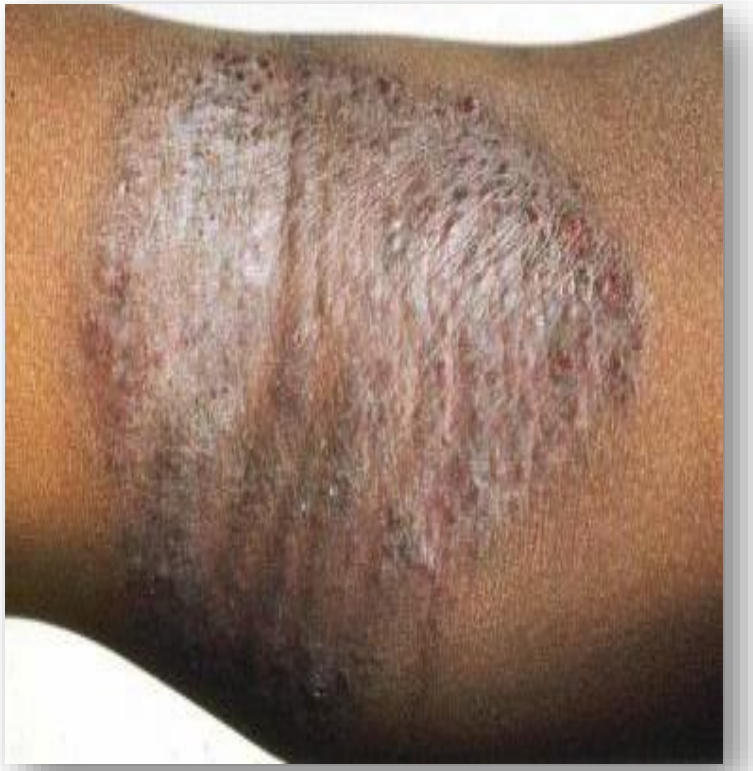
Well defined erythema with blister formation



Erythematous rash with oozing, vesiculation and edema

Chronic eczema





Dryness of the skin with thickening and scaling due to chronic itching.

(Lichenification)

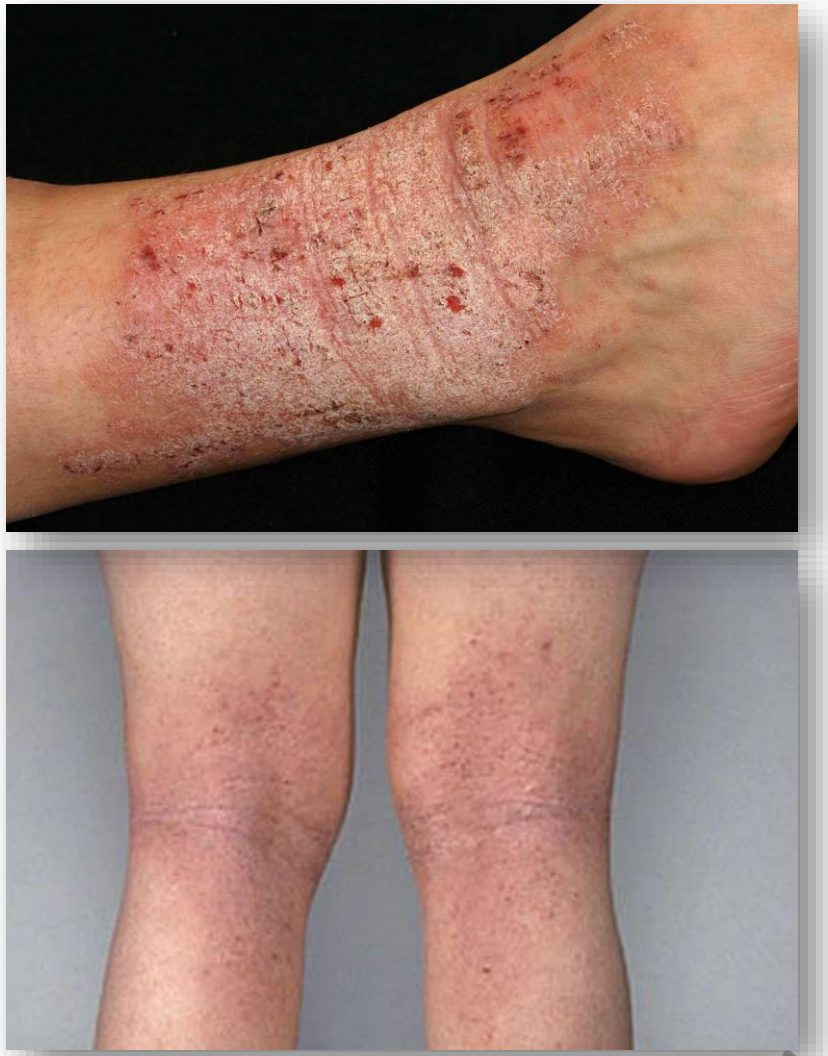
Chronic eczema

سنوات (6)

- **What is the 2ry lesion is seen ?, define it**
  - **Lichenification**: Hard thickening of the skin with accentuated skin markings which sign in chronic itching

سنوات (1)

- **It is diagnostic for what disease ?**
  - Chronic eczema



Causes of eczema

سنوات (1)

❖Mention 4 types of exogenous eczema (outside cause)

1. Contact allergic dermatitis
2. Contact irritant dermatitis
3. Contact allergic photodermatitis
4. Contact irritant photodermatitis

سنوات (2)

❖Mention 5 types of endogenous eczema (inside cause)

1. Atopic dermatitis
2. Seborrheic dermatitis
3. Discoid (nummular) eczema.
4. Stasis dermatitis
5. Aesteatotic eczema
6. Dyshidrotic eczema (Pompholyx)
7. Gravitational (varicose) eczema.
8. lichen simplex
9. Juvenile plantar dermatosis

سنوات (1)

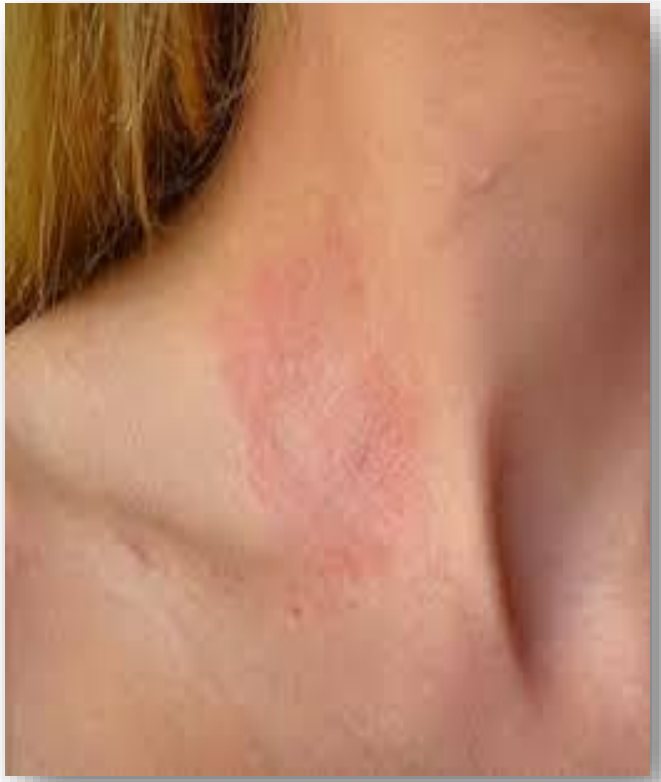
❖Mention the most common 2 type of endogenous eczema:

1. Atopic dermatitis.
2. Seborrheic dermatitis

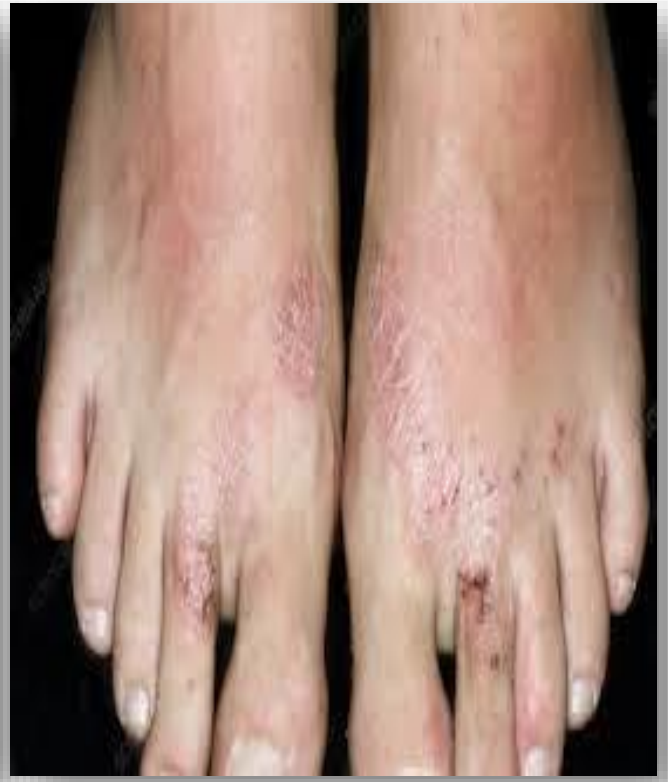
Contact dermatitis

Contact allergic dermatitis	Contact irritant dermatitis
Predisposed persons only	All persons
Needs previous sensitization	No need for sensitization
Allergic substances include nickel, cement, rubber, dyes and others	Chemicals,detergents
In some persons with some substances, regardless the concentration of the substances or the duration of the exposure	In all persons if they expose to the substance for long duration or with high concentration, even after first exposure
Contact allergic photodermatitis	**Contact irritant photodermatitis**
It is a contact allergic dermatitis, but it needs sun exposure to occur	It is a contact irritant dermatitis, but it needs sun exposure to develop
Sun exposure is needed for the eczematous reaction to develop	

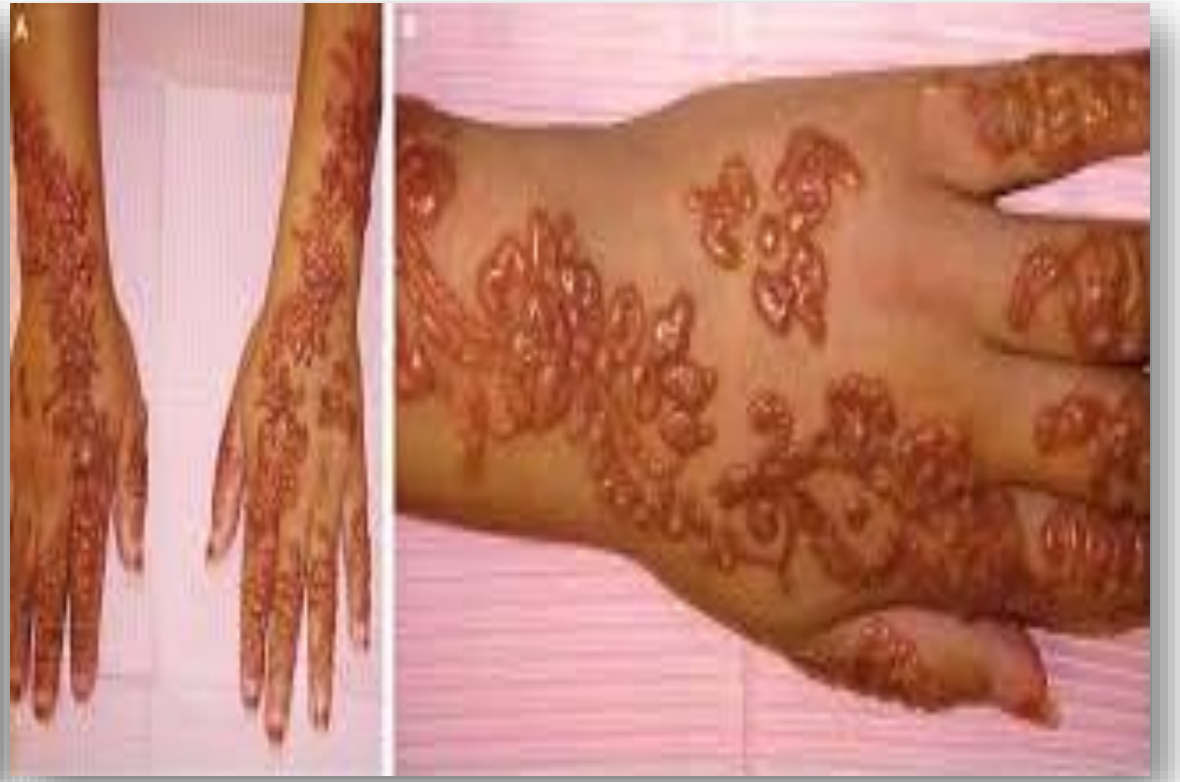
Contact allergic dermatitis



Contact allergic dermatitis due to the necklace (contains nickel)

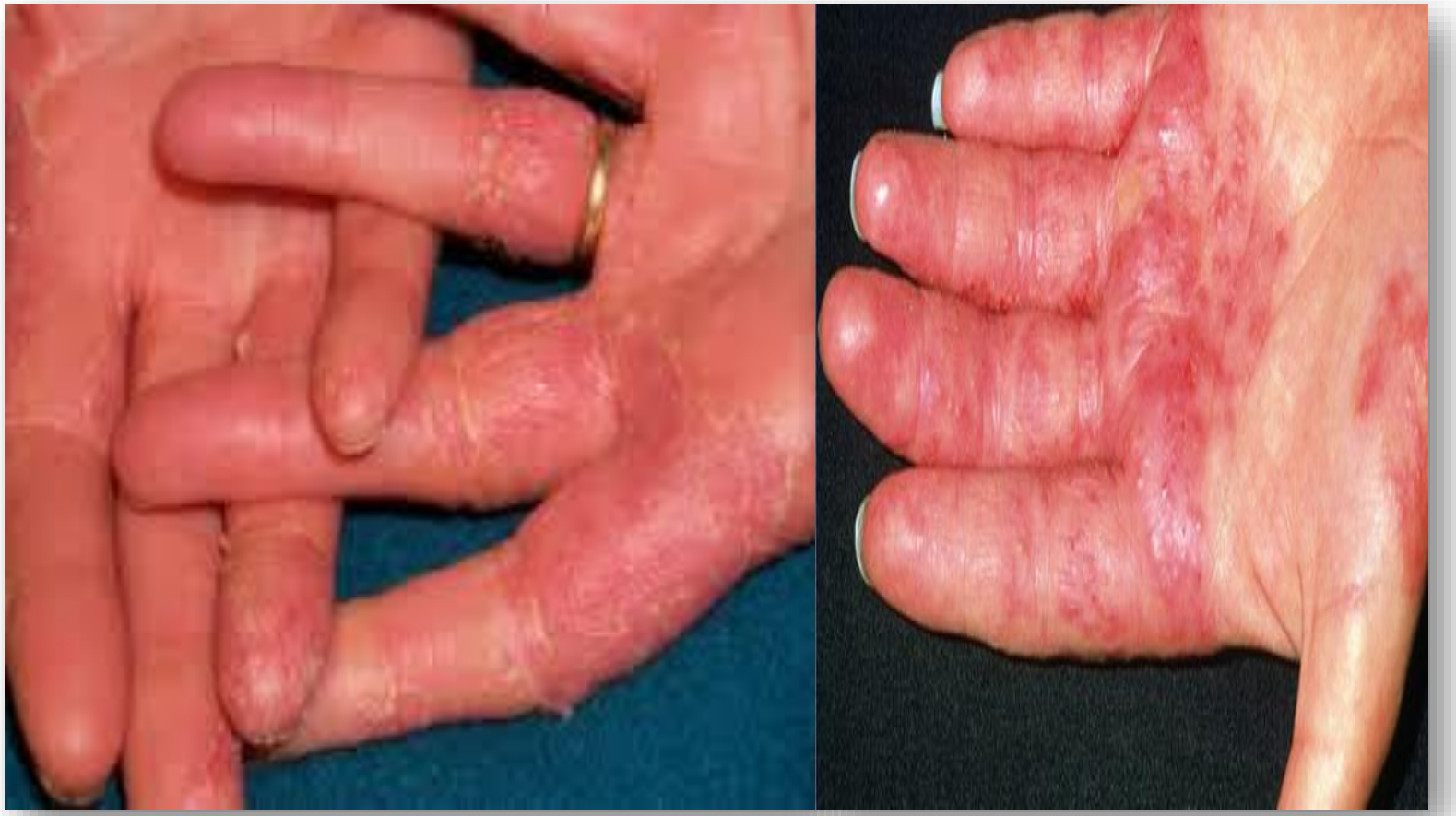


Contact allergic dermatitis due to shoes (contains dyes and rubber)



Contact allergic dermatitis due to Henna.

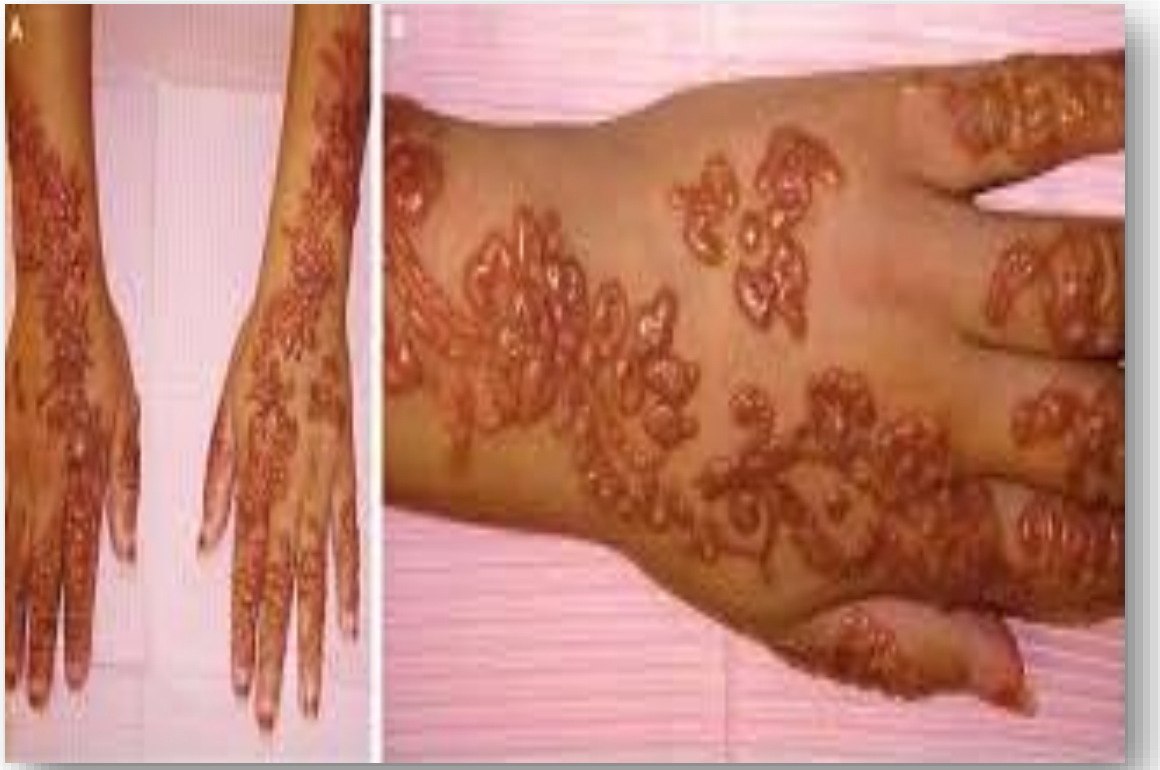
Contact irritant dermatitis



Contact irritant dermatitis due to detergents exposure

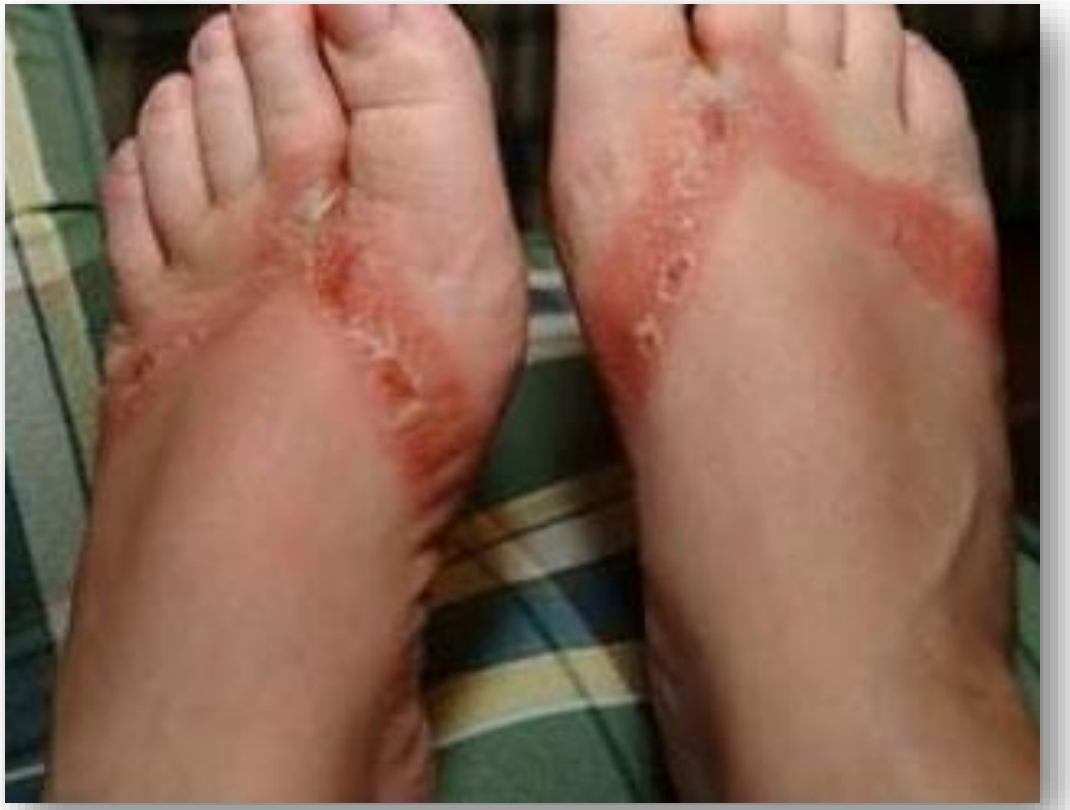
What is the diagnosis

A. Contact irritant dermatitis
B. Contact allergic dermatitis
C. Psoriasis
D. Koebner phenomena
E. Auto-sensetization



Contact allergic dermatitis

- What is your diagnosis ?
  - Contact allergic dermatitis
- What is the confirmatory test ?
  - Patch test

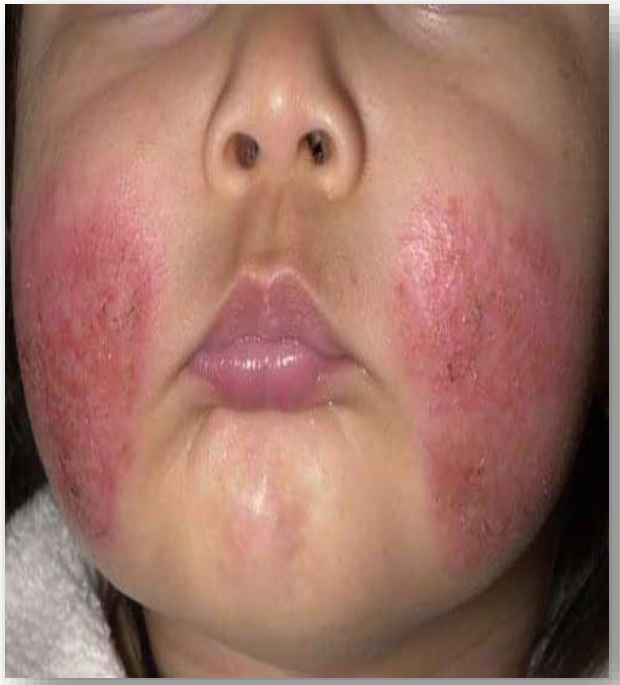


Atopic dermatitis

- Affects 20% of children and 1-3% of adults
- 85% of patients are less than the age of 5 years
- Diagnosis is clinically, a triad of:
  1. dry skin,
  2. itching
  3. specific eczematous lesions especially in flexures
- Cheeks is a common sites of skin lesions in infants and flexures is a common sited in children and adults.
- It can be a part of atopic state that includes atopic eczema, hay fever, allergic rhinitis, allergic conjunctivitis and bronchial asthma

سنوات (1)

Atopic dermatitis



Infant with ill –defined, scaly, erythematous patches over the cheeks.

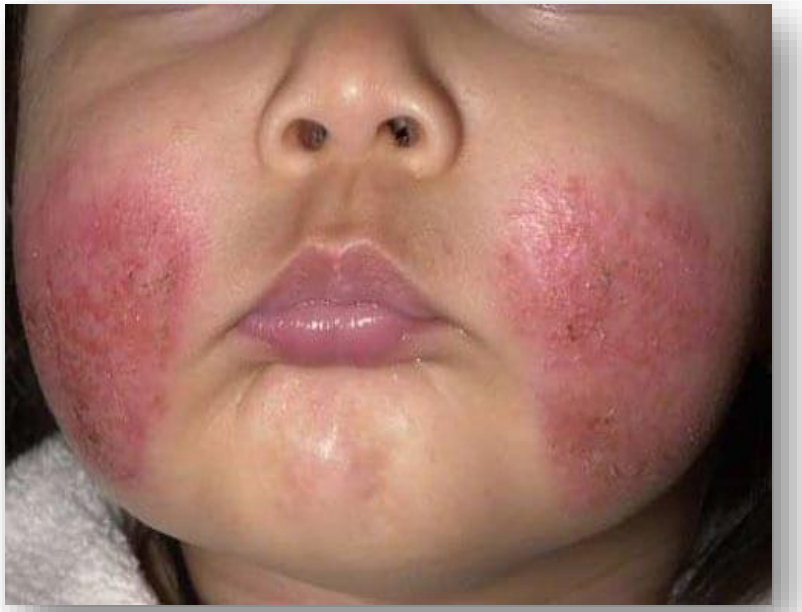
Child with ill –defined, scaly, erythematous patches with Lichenification over the popliteal fossa (Flexural area)

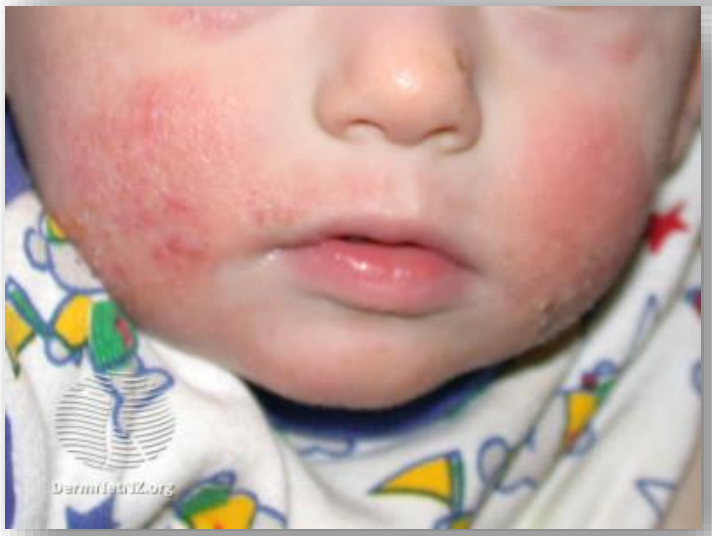
Child with ill –defined, scaly, erythematous patches with Lichenification over the popliteal fossa (Flexural area)

Adult with signs of eczema and Lichenification on the flexural site.

Recurrent lesion in multiple occasion

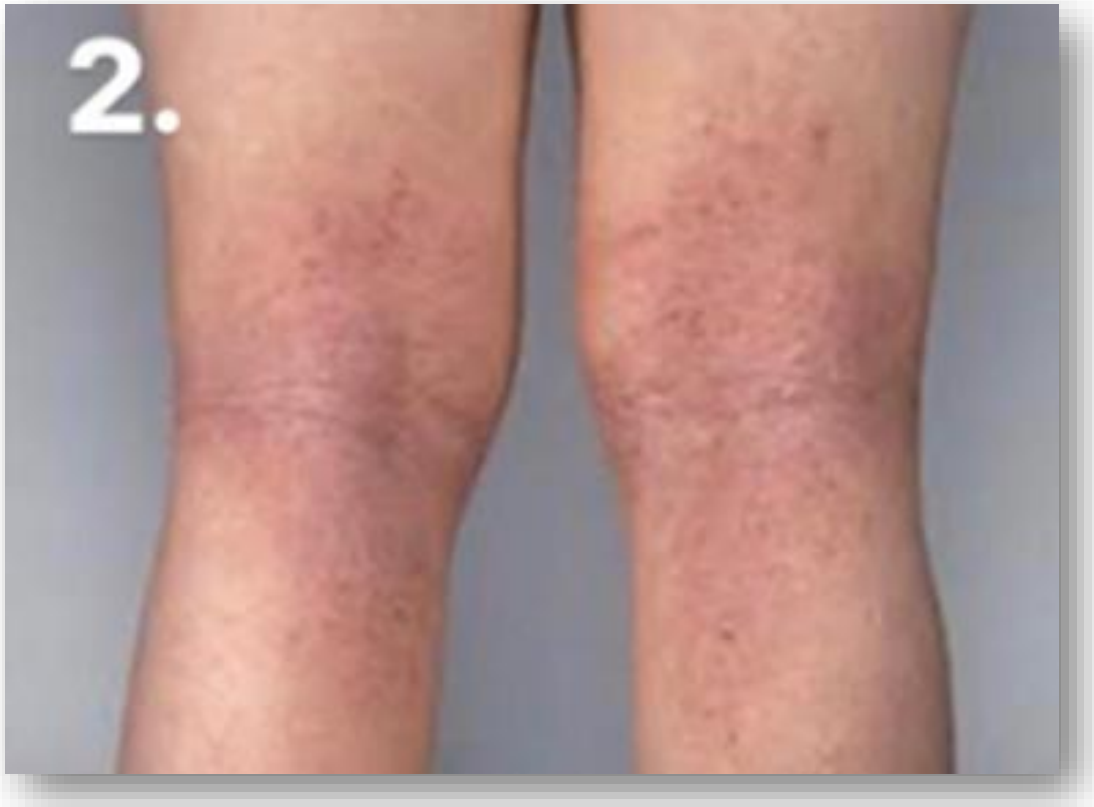
- **Spot diagnosis**:
  - Atopic dermatitis on children
- **Mention the 3 characteristic features of it**
  1. Dry skin
  2. Itching
  3. Specific eczematous lesions especially in flexures





Atopic dermatitis

- **What is the diagnosis ?**
  - Atopic dermatitis
- **Mention one symptom that patients suffer from**
  - Dryness, itchy, specific eczematous lesions especially in flexures



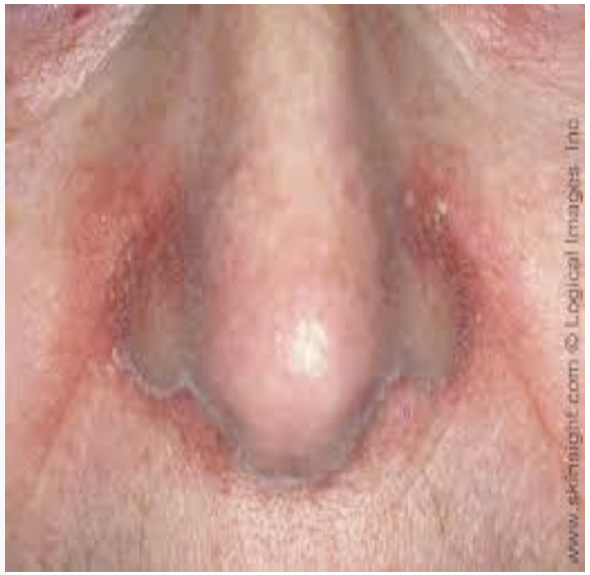
Seborrheic dermatitis

- Common itchy chronic inflammatory skin disease
- **Demographic**: mainly newborns and adults due to sebum production
- There is a possible role for *Pityrosporum ovale (Malassezia)* (yeast)
- **Appearance**:
  - In newborns it can appear as cradle cap.
  - In adults appears as erythematous lesions with greasy scales on the Face and/or scalp, anterior chest upper back and skin folds.
- **Differential diagnosis** includes psoriasis.
- **Treatment**: Low-potency topical steroids or a selenium-based shampoo

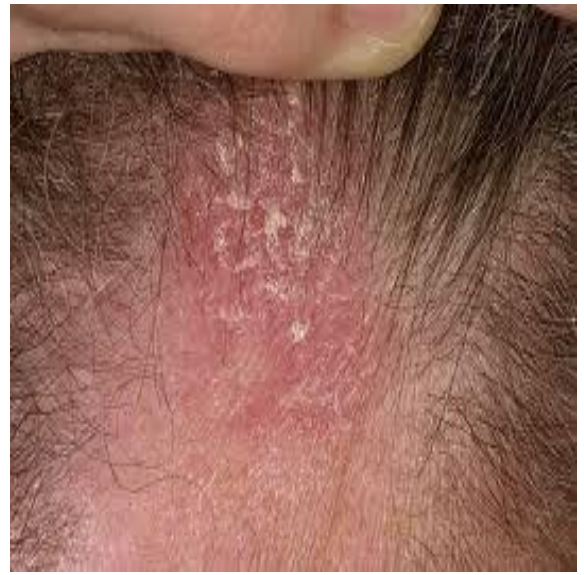
Seborrheic dermatitis

Infant with erythematous scalp with greasy scales and, eyebrows and nasolabial folds involvement

(Cradle cap = adherent yellowish scales on the scalp)



In adults, nasolabial folds are involved, greasy scales on the face.



Greasy scales on the scalp and the lesions respect the hair margin.

(Psoriasis doesn't respect the hair margins)

50-year-old male, what is the diagnosis

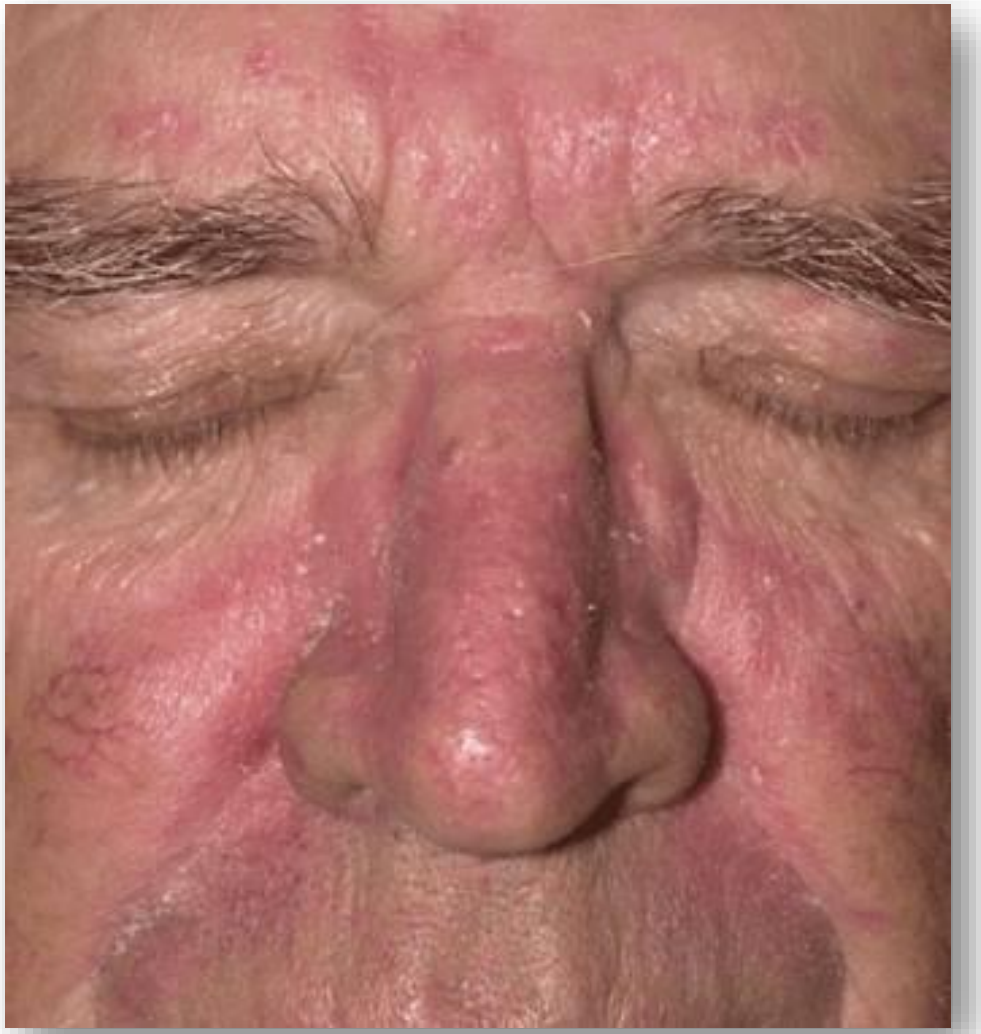
A. SLE

B. Photo dermatitis

C. Tinea faciei

D. Seborrheic dermatitis

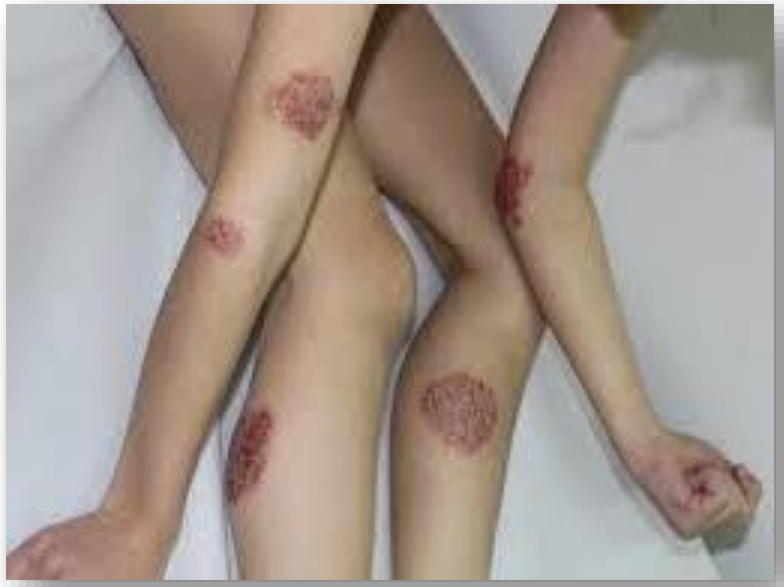
E. Lichen planus

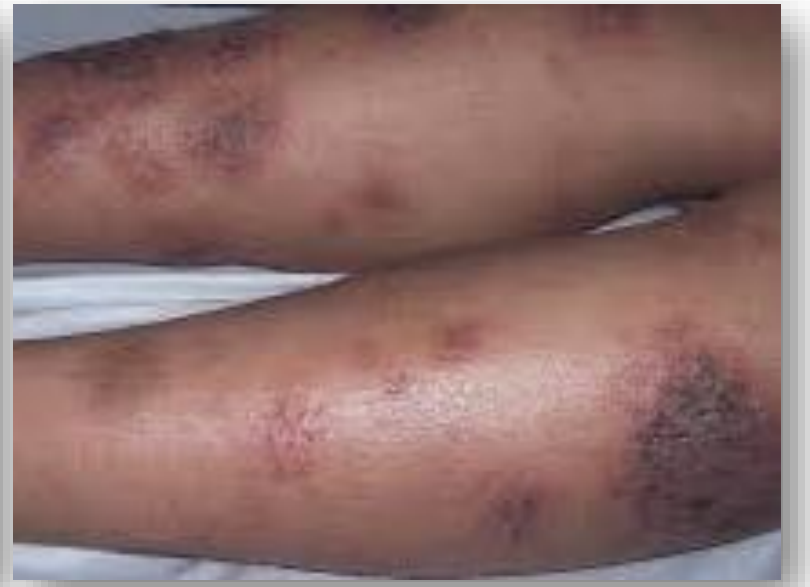


Discoid (nummular) eczema

- Chronic itchy inflammatory skin disease that can affects children and adults
- **Characterized by** disc shape lesions bilaterally with sometimes mirror image distribution
- **Lesion Site**:
  - Commonly lesions involve the extremities more than the trunk
  - Usually does not affect the face and scalp
- **Differential diagnosis**
  - Psoriasis
  - Fungal infection

Discoid (nummular) eczema



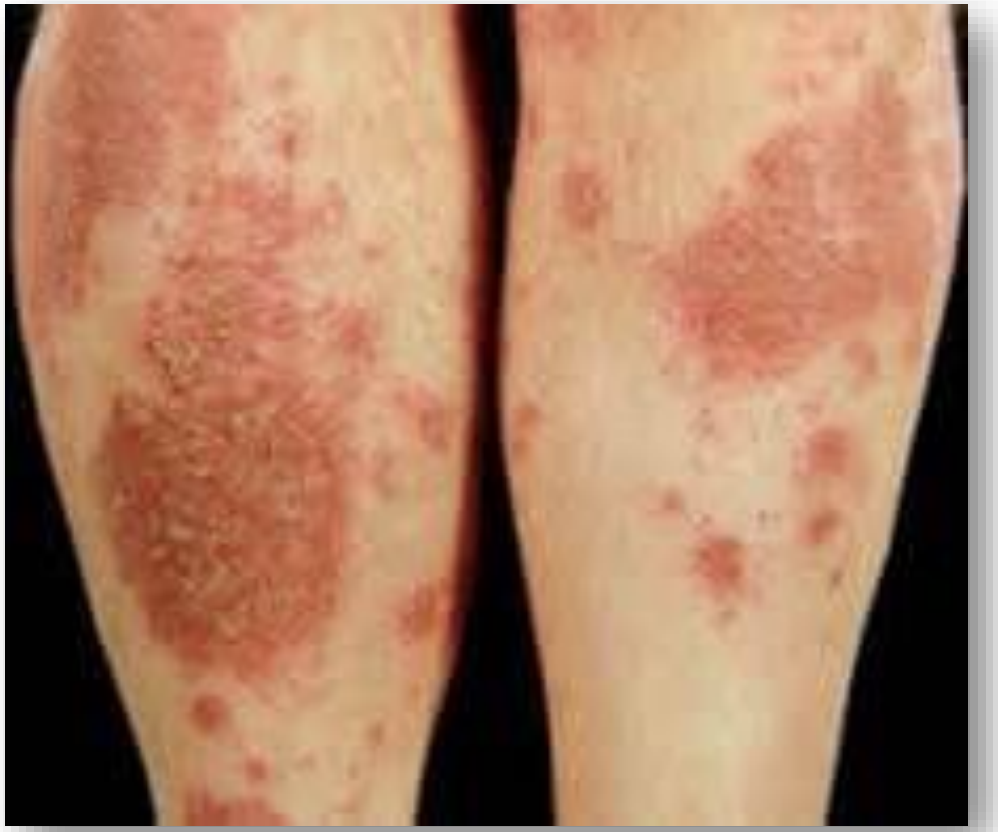


Young patient with discoid lesions, bilaterally distributed in mirror image pattern, itchy.

DDx: Tinea corporis by KOH

Bilateral symmetrical lesion, what is the diagnosis

A. Psoriasis
B. Atopic dermatitis
C. Lichen planus
D. Tinea corporis
E. Discoid eczema

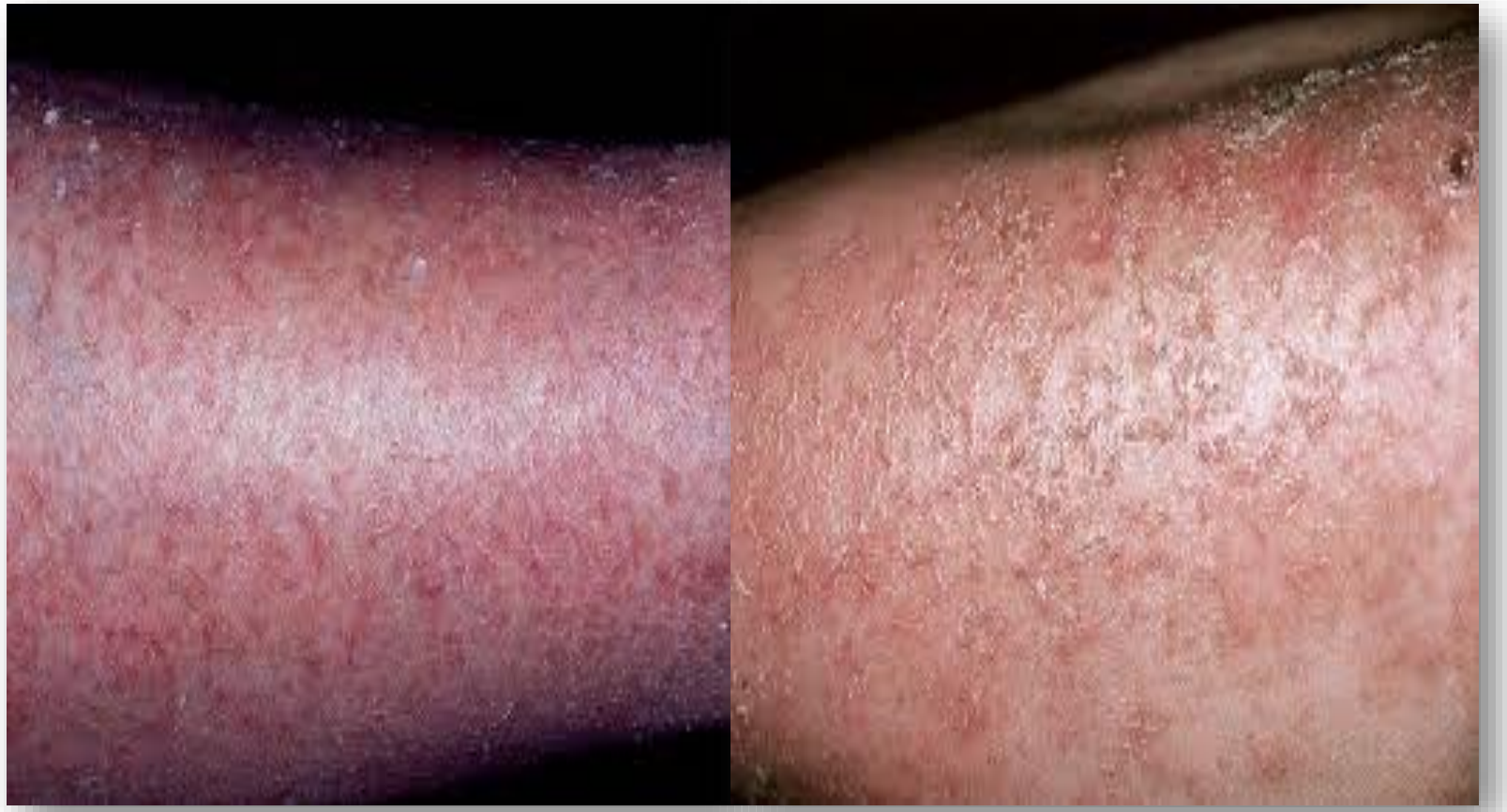


Aesteatotic eczema

- Chronic itchy inflammatory skin disease often affects elderly.
- Mostly due to water loss from the stratum corneum because of genetic and environmental factors (desert, winter, excessive bathing).
- Starts on the shins and then spreads.
- **Appearance**: dry and cracked skin appearance of crazy paving.
- **Differential diagnosis**
  - Acquired ichthyosis
  - Skin changes

Crazy paving

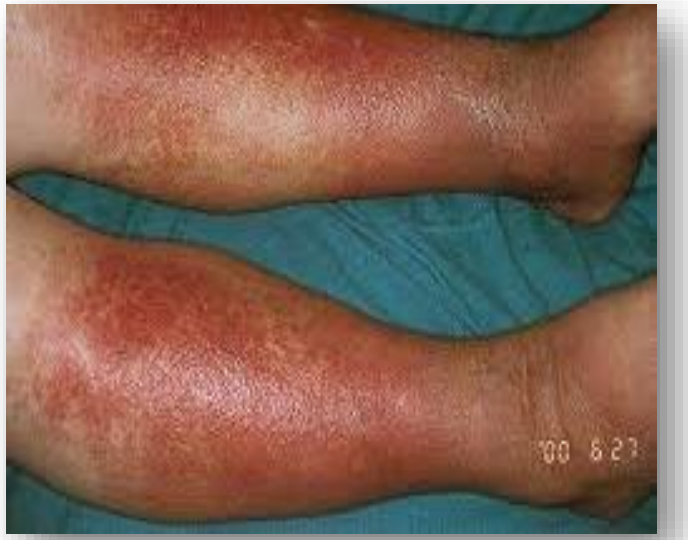
Aesteatotic eczema

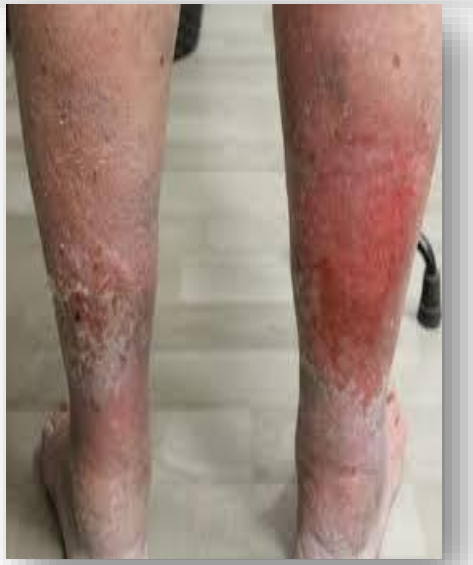


Dry, cracked skin like crazy paving.

Stasis eczema (Gravitational dermatitis)

- Occur mostly in people aged 50 years or older with lower limb stasis.
- Chronic itchy inflammatory skin disease due to stasis.
- Occur in women more than in men.
- Lower limbs usually affected with scaly erythematous and Hyperpigmented (due to hemosiderin deposition) ill defined lesion.
- **Differential diagnosis**
  - DVT,
  - Erysipelas
  - Cellulitis





Lower limb edema with oozing and ill-defined area of hyperpigmen tation.

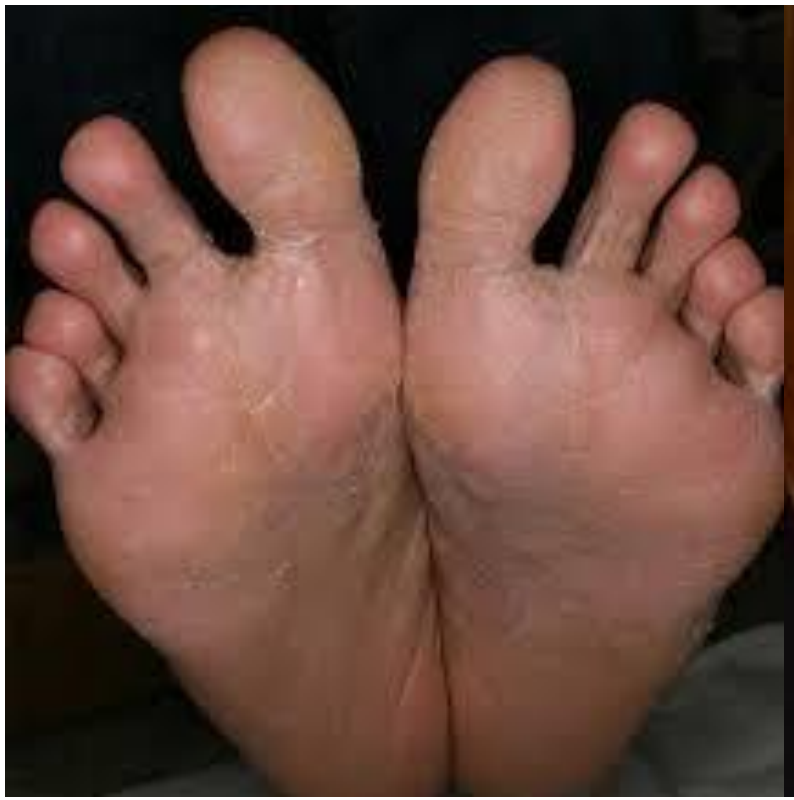
Dyshidrotic eczema

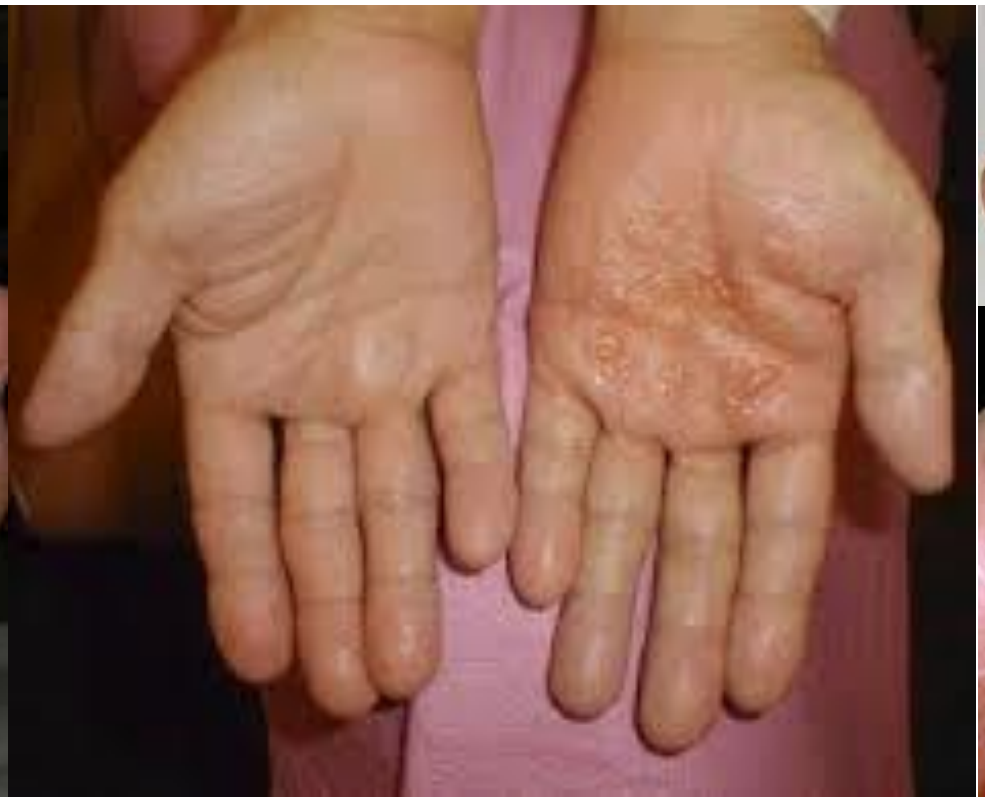
- Chronic itchy inflammatory skin disease affecting the hands (cheirpompholox) and/or feet (podopompholox).
- Most often affects young adults.

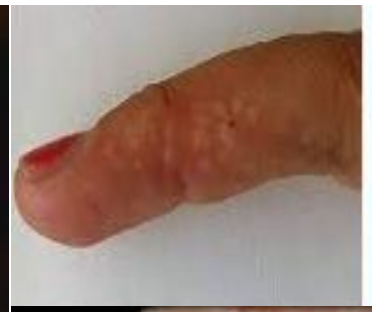
سنوات (1)

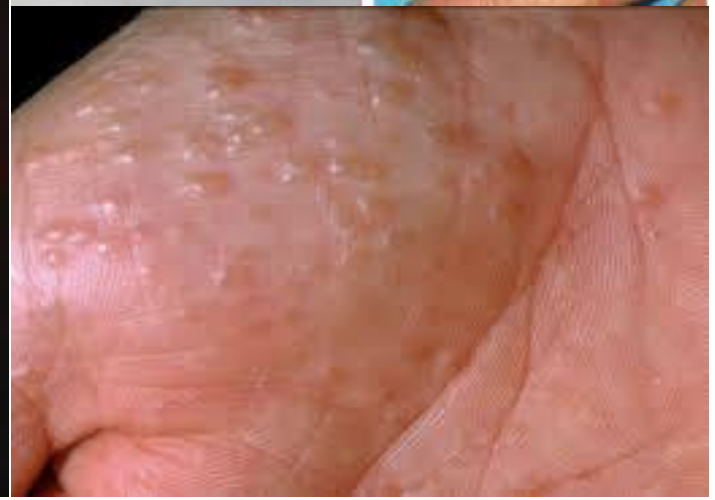
- **Primary lesion**: deep seated vesicles and blisters on the palms, fingers, soles and toes.
- Many patients report palmoplantar hyperhidrosis.
- **Differential diagnosis**
  - Psoriasis
  - Contact dermatitis and
  - Id-reaction (An allergic reaction to an inflammatory dermatophyte fungal (Tinea Pedis / Tinea Cruris) infection elsewhere)

Dyshidrotic eczema









Erythema, scaling and deep-seated vesicles.

Deep-seated vesicles.

Management of eczema

1. Prophylactic measures to avoid exacerbating factors like harsh clothes (Contact dermatitis), irritants (Irritant contact dermatitis), infections and stress especially in atopic and Seborrheic eczema
2. Treatment of stasis in stasis dermatitis
3. Emollient especially important for atopic and aesteatotic eczema
4. Topical steroids
5. Topical Calcineurin inhibitors (Tacrolimus)
6. Antihistamines to relieve itching
7. Sometimes short course of systemic steroids in severe cases especially in acute forms or sever exacerbation

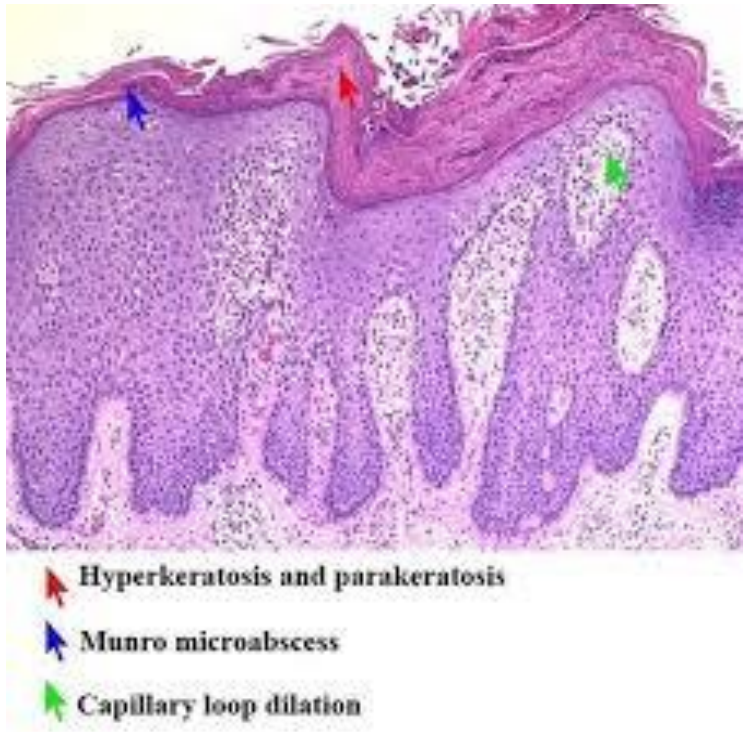
Psoriasis

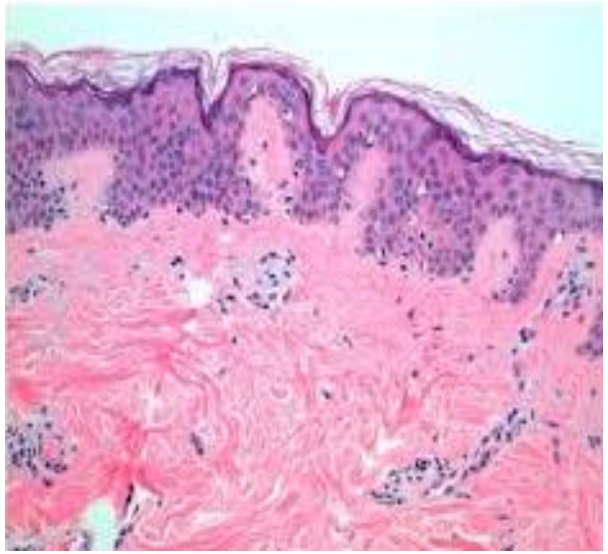
الصدفية

شرح

Histopathological features of psoriasis

1. Hyperkeratosis
2. Parakeratosis
3. Munro`s microabscess
4. Acanthosis
5. Hypogranulosis
6. Lymphocytic inflammatory infiltrate





❖**Mention the cells that are involved in psoriasis:** سنوات (1)

1. keratinocytes
2. Dendritic cells
3. T-cells

شرح

Clinical presentation of psoriasis

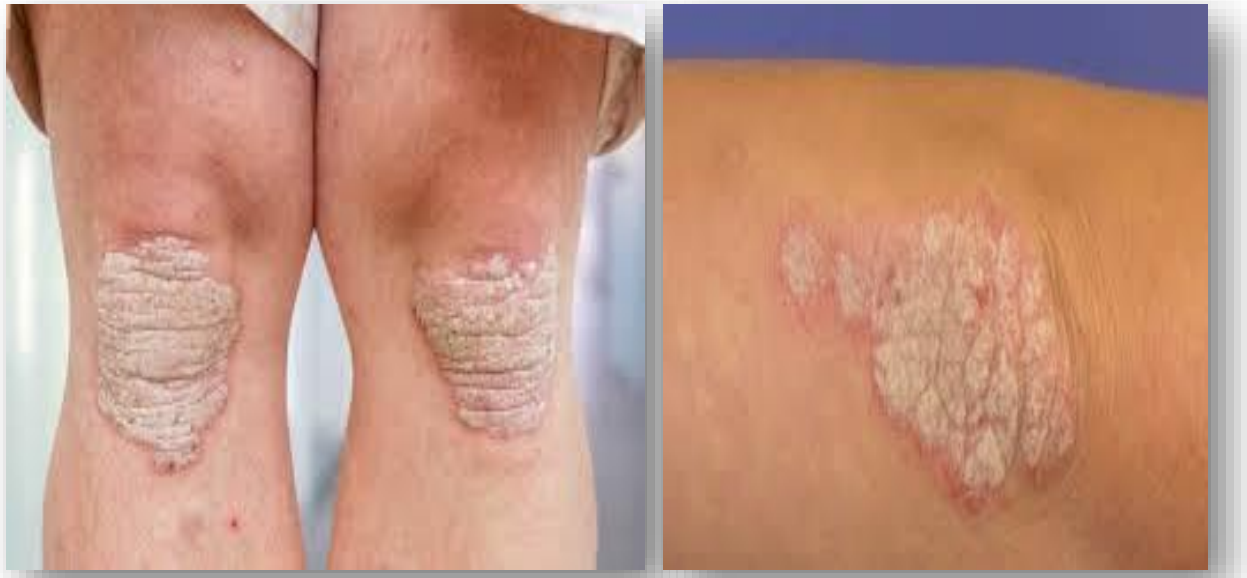
- Skin:
  - (1) سنوات **Primary lesion**: Well demarcated erythematous plaques covered with dry silver scales on extensor surfaces (in psoriasis vulgaris)
  - (1) سنوات Positive auspitz sign: pinpoint bleeding when scale is picked off
- Nail changes seen in psoriasis:
  - (1) سنوات Nail pitting, onycholysis, oil spot, and discoloration and thickening
- Scalp:
  - Thick scaly plaques covered with silvery dry scales that may extend beyond the hair margin (vs seborrheic dermatitis which respect the margin)
- Mouth:
  - Geographical tongue

Mention 5 subtypes of psoriasis

1. Plaque psoriasis (psoriasis vulgaris)
2. Scalp psoriasis
3. Nail psoriasis
4. Flexural (inverse) psoriasis
5. Acute pustular psoriasis
6. Chronic palmoplantar pustulosis
7. Erythrodermic psoriasis
8. Guttate psoriasis
9. Unstable or ‘brittle’ psoriasis
10. Arthropathic psoriasis

1. Psoriasis vulgaris

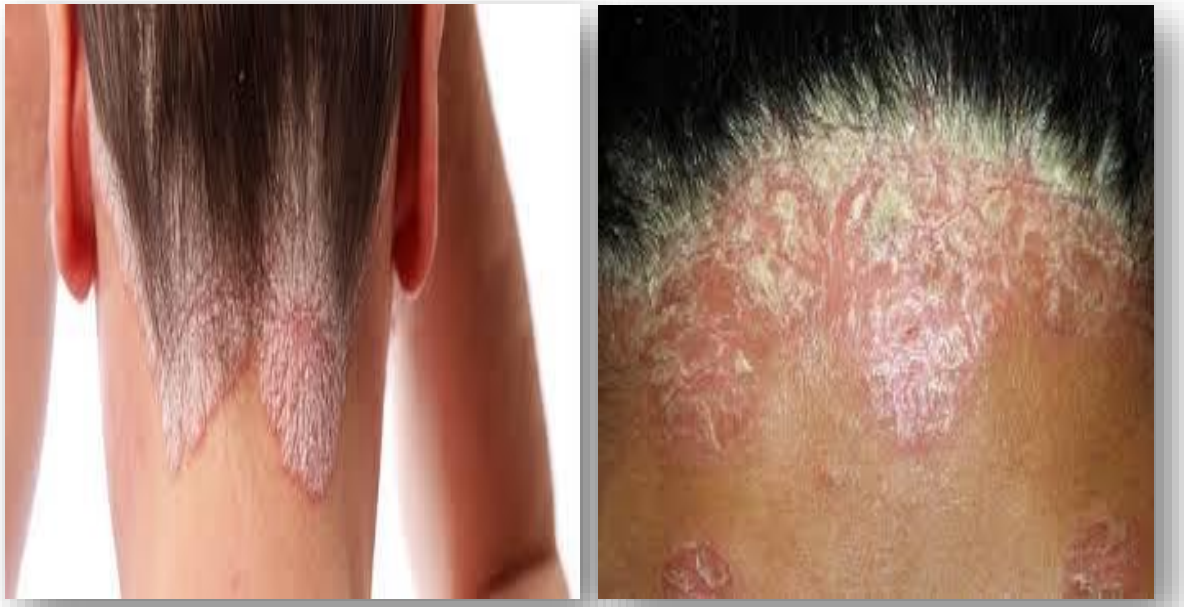
- It is characterized by well-defined erythematous plaques that may have adherent dry silvery scales
- Symmetrical plaques on elbows, knees, and lower trunk, with scalp involvement and it can be pruritic



Well demarcated, erythematous plaques covered with silvery dry scales on the knees.

2. Scalp psoriasis

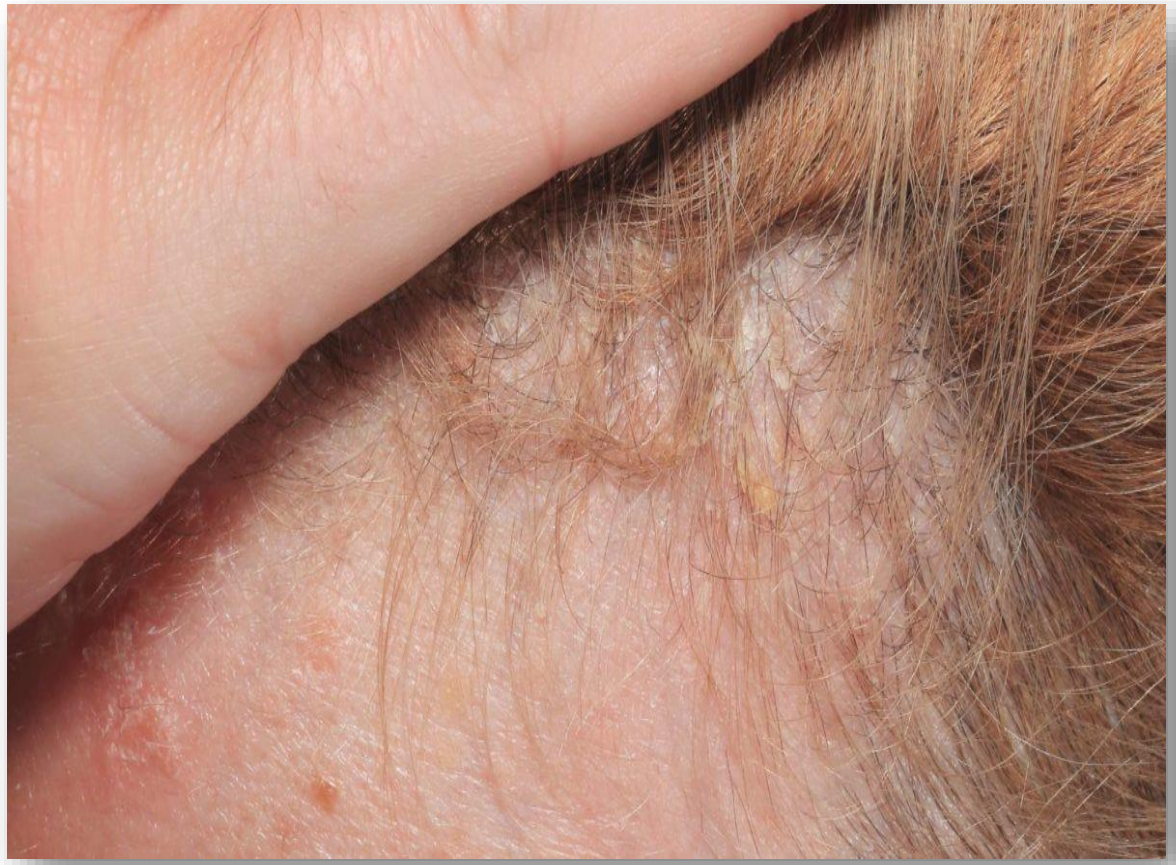
- Between 50% and 80% of patients with psoriasis develop lesions on their scalp.
- If it occur without skin lesions it is called scalp psoriasis.
- The scales are dry and silvery, and the lesions can be felt.
- lesions may extend onto facial skin or posterior neck (do not respect the hair margin)



Well demarcated erythematous plaques covered with silvery scaly and extending beyond the hair margin.

What is the diagnosis ?

Scalp psoriasis (do not respect the hair margin)



3. Nail disease (Nail psoriasis)

- Nail involvement is common in all forms of psoriasis, affecting an estimated 80% of patients with the disease especially in pustular, Erythrodermic and palmoplantar forms and with psoriatic arthritis.
- Nail pitting, oil drop–like patterns of yellow or salmon discoloration, nail thickening, Onycholysis and discoloration.

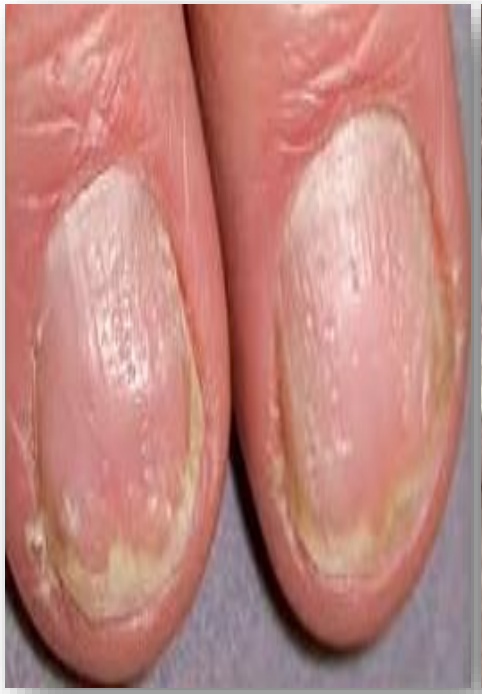
سنوات (1)

- **Define oil spot sign**: yellowish brown spots that result from nail bed parakeratosis
- Nail disease can occur without any skin involvement (nail psoriasis) Which is sometimes difficult to diagnose.

Onycholysis, oil spots and pitting.

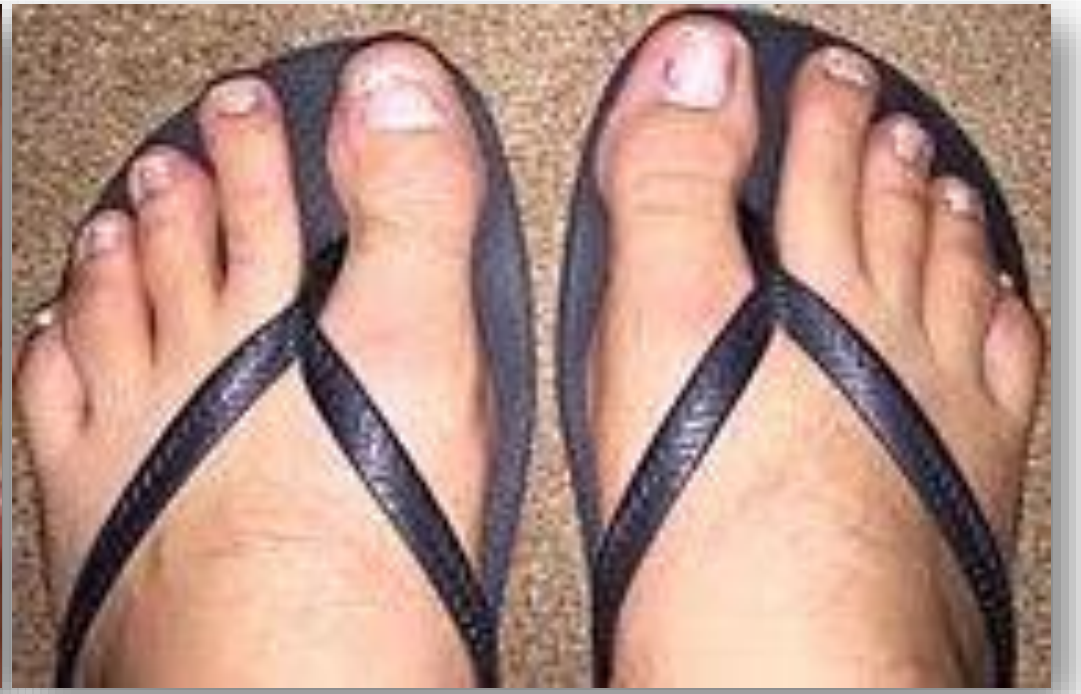
Onycholysis, discoloration, oil spots.

Nail disease (Nail psoriasis)



Pitting and Onycholysis

Pitting, Onycholysis and oil spots.

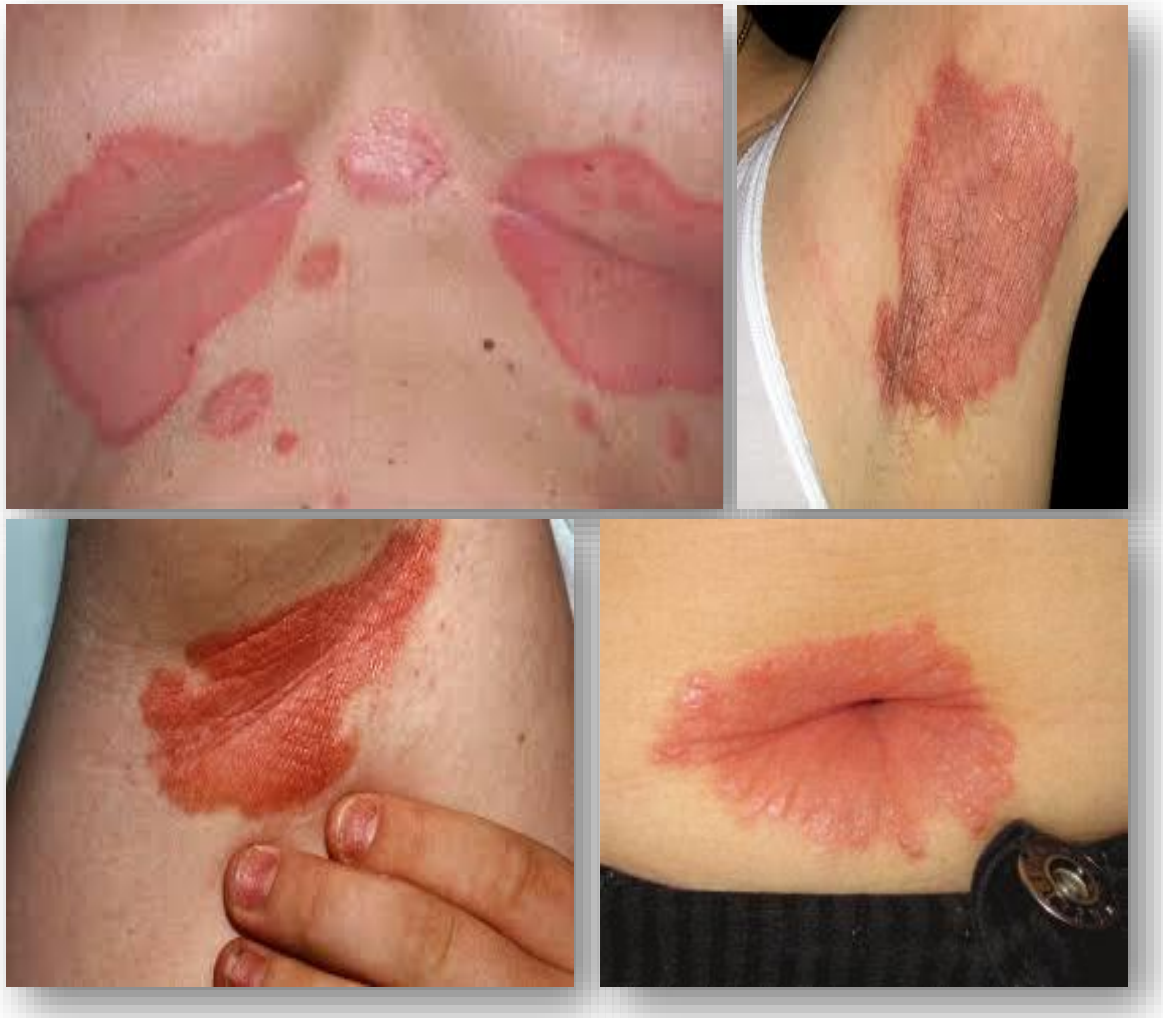


Onycholysis, pitting and discoloration.

Plaques which form around the nail plate can cause pitting. Those which form beneath the nail plate can cause Onycholysis

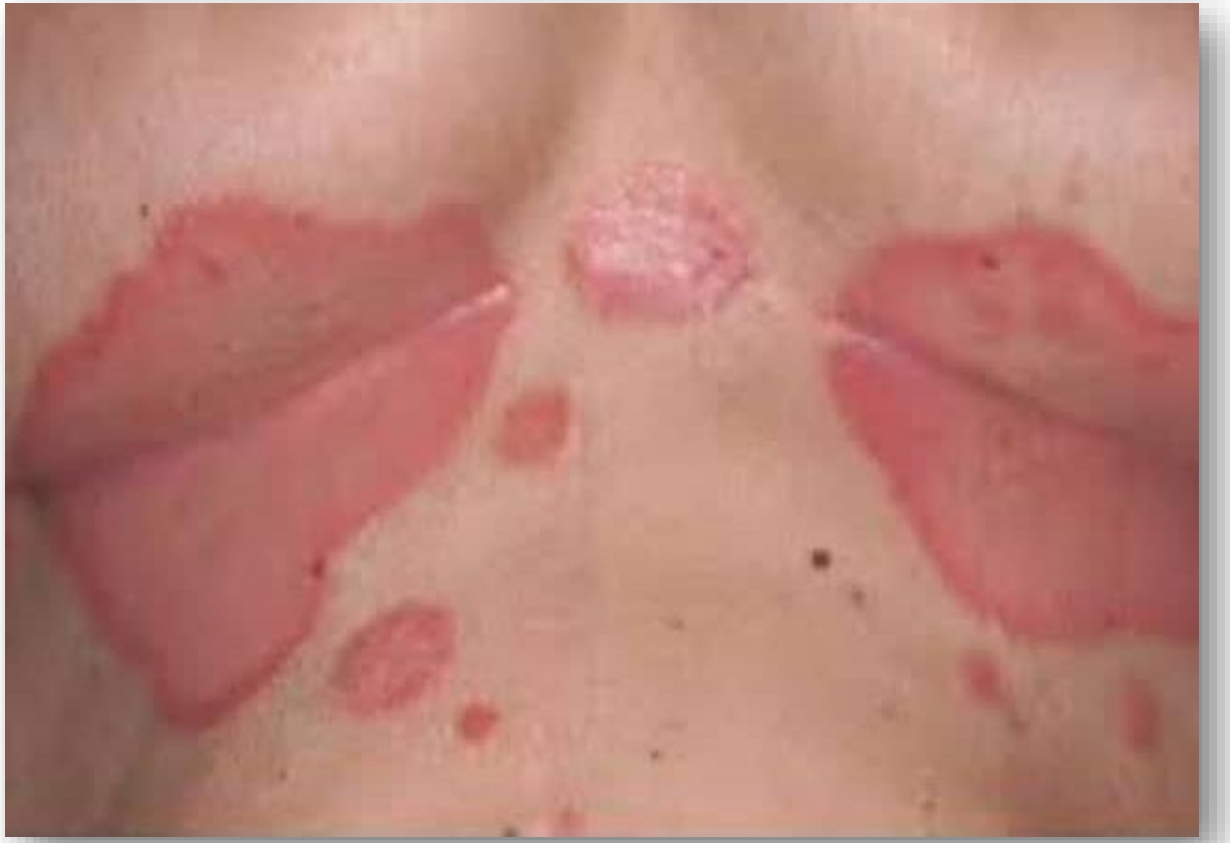
4. Inverse (flexural) psoriasis

- Involves the groin and/or other intertriginous areas, such as the armpits, under the breasts, or in abdominal skin folds
- **Characterized by**
  - well-defined, shiny, erythematous plaques with minimal scaling (due to friction of the opposed skin leading to scale removal)
- **Differential diagnosis**:
  - Fungal infection and Seborrheic dermatitis



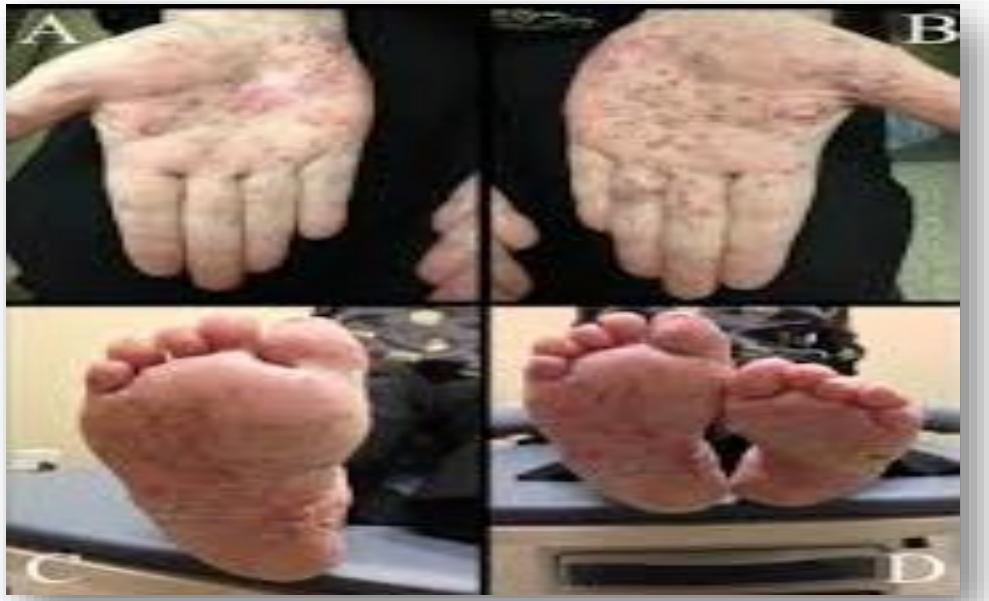
Flexural psoriasis

- Which type of psoriasis that doesn’t present with scales ?
  - Flexural (inverse) psoriasis



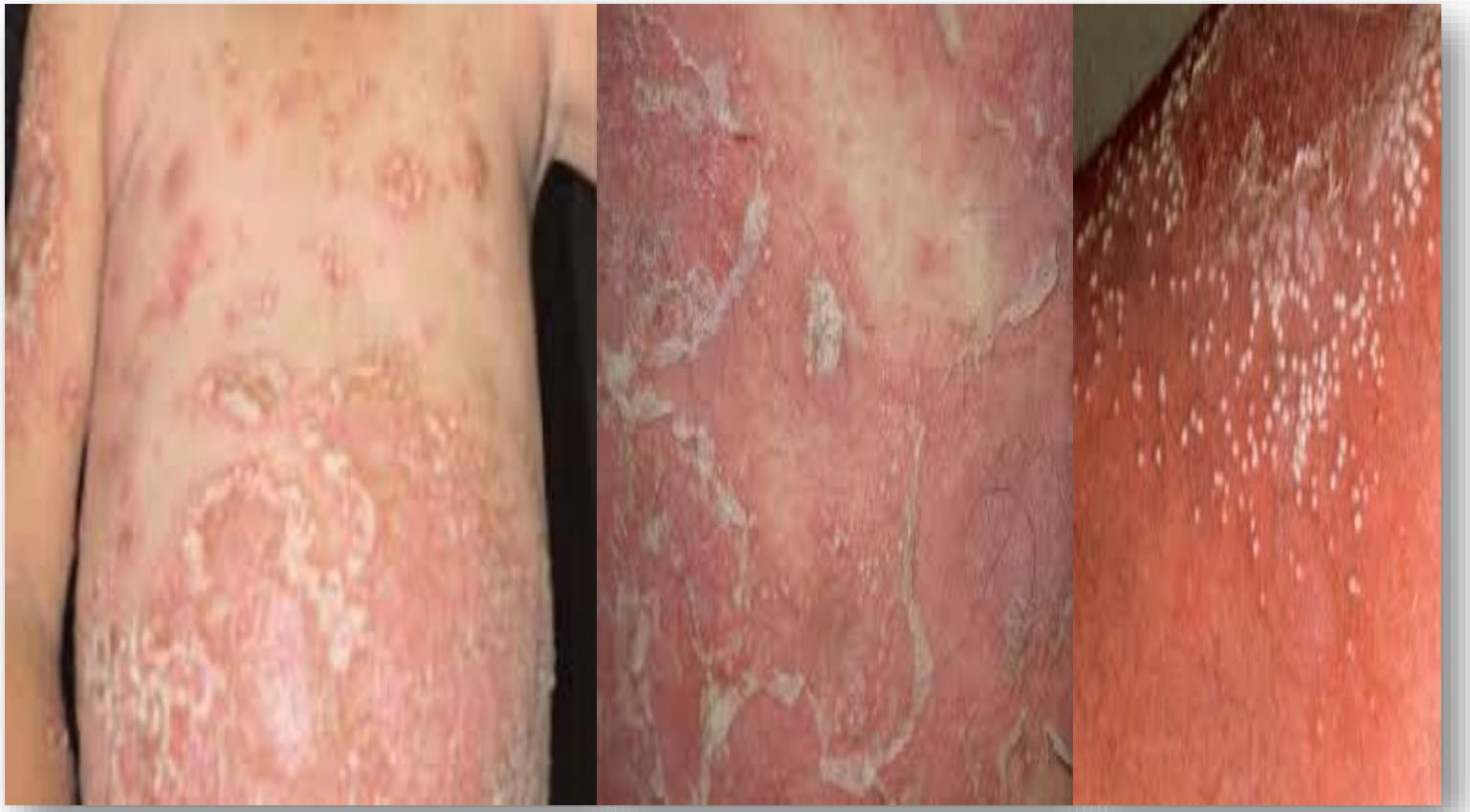
5. Pustular psoriasis

- Eruption of sterile pustules that can be generalized and extensive or localized to existing plaques
- **von Zumbusch variant**: Acute generalized pustular psoriasis, an uncommon, severe form of psoriasis that may be accompanied by edema and fever and may require hospitalization
- It needs systemic treatment



Pustular psoriasis-Localized (palmoplantar)

Pustular psoriasis – Generalized

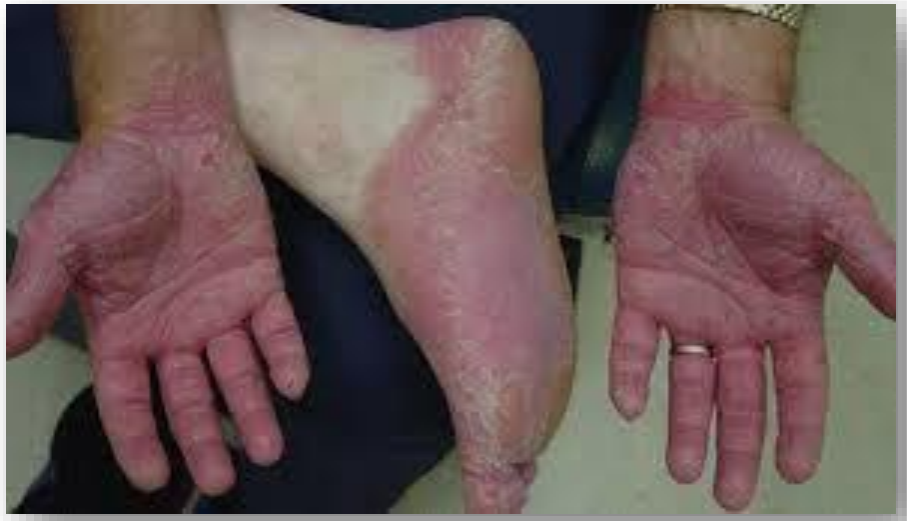


Generalized erythema studied with sterile pustules.

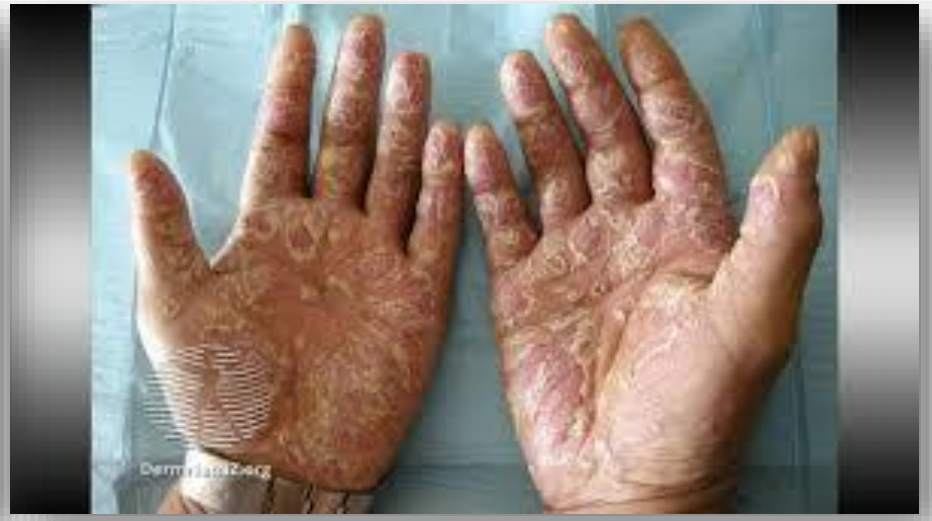
6. Palmoplantar psoriasis

- **Characterized by** yellow-brown sterile pustules on the hands and feet
- Nail changes are more frequent in this variant
- Patients may also experience scaling and severe pruritus, making this variant difficult to differentiate from hand eczema
- This form of psoriasis is more common in women
- Smoking is a risk factor for this variant
- Differential diagnosis:
  - Eczema (biopsy is helpful for making the diagnosis of psoriasis)
  - Fungal infection

Palmoplantar psoriasis



Well-defined Itchy erythema with scaling



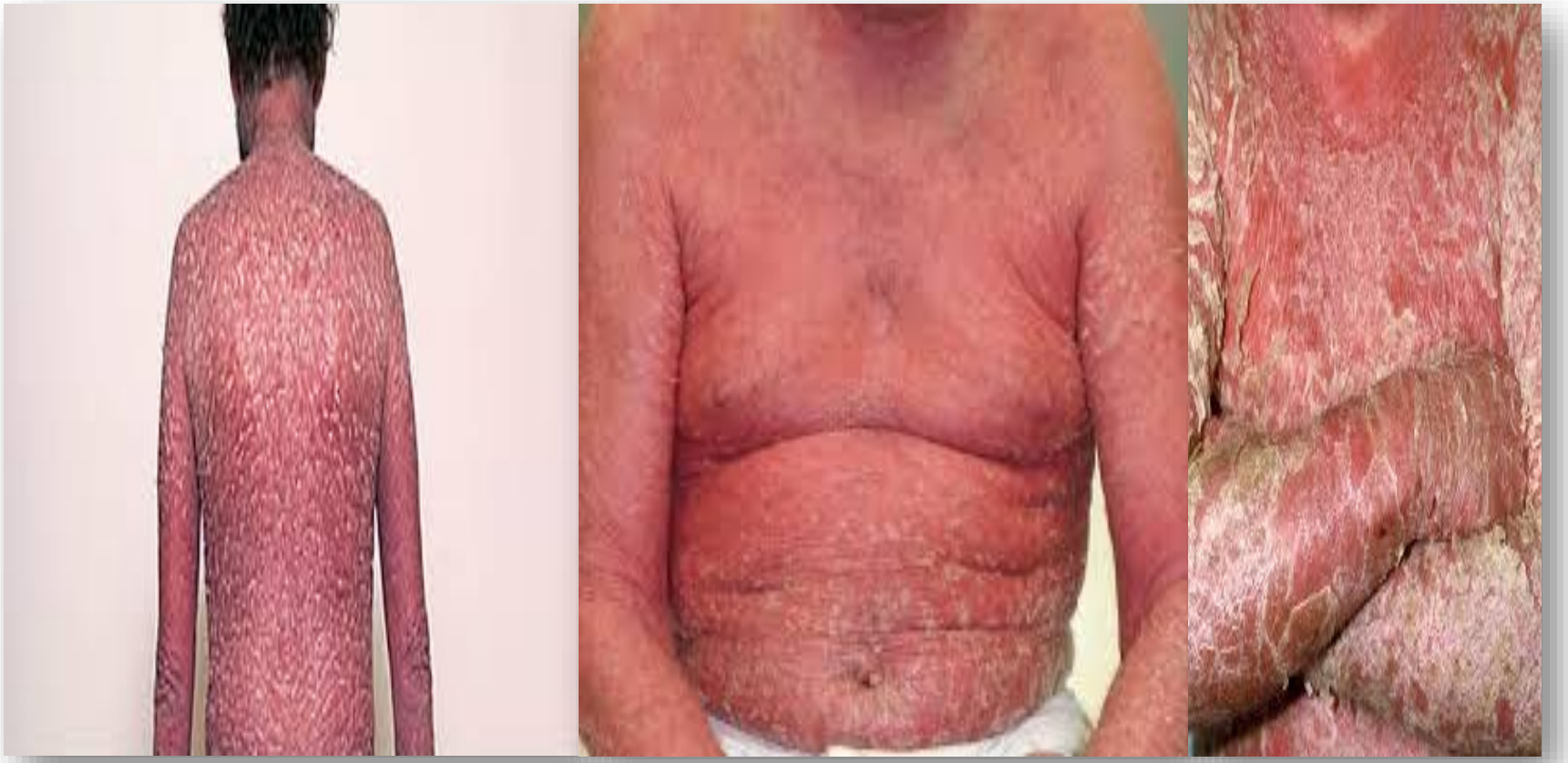
Well-defined itchy erythema with scaling, some pustules.

Well-defined itchy erythema with scaling, some pustules and nail changes.

7. Erythrodermic psoriasis

- Erythrodermic psoriasis appears as generalized exfoliative dermatitis that can affect a large percentage of a patient's body surface area
- **Erythrodermic**: affection of more than 90% of BSA (body surface area)
- Hair loss and nail dystrophy are common with this type
- Patients may experience fever, chills, and/or fatigue
- Erythrodermic psoriasis can be life-threatening and require hospitalization

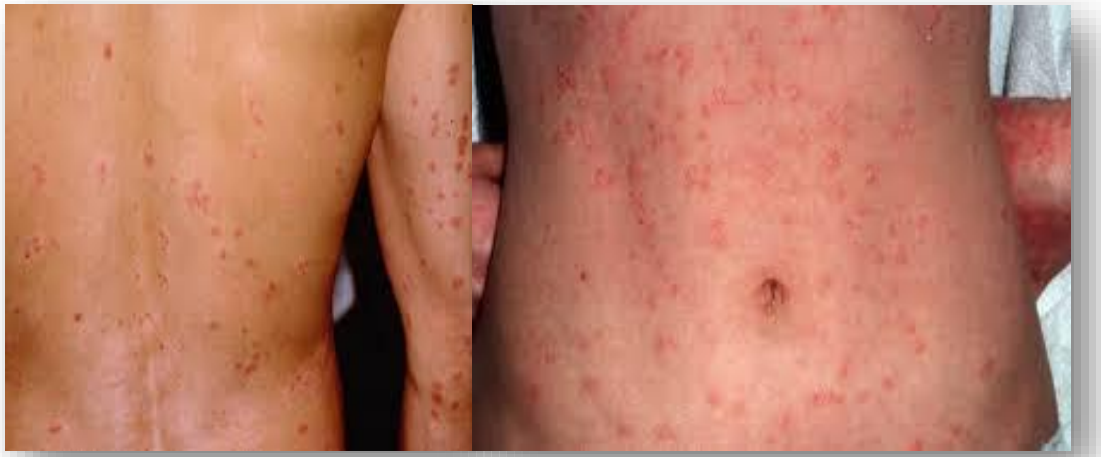
Erythrodermic psoriasis



Generalized erythema with scales, can appear due to maltreated psoriasis vulgaris

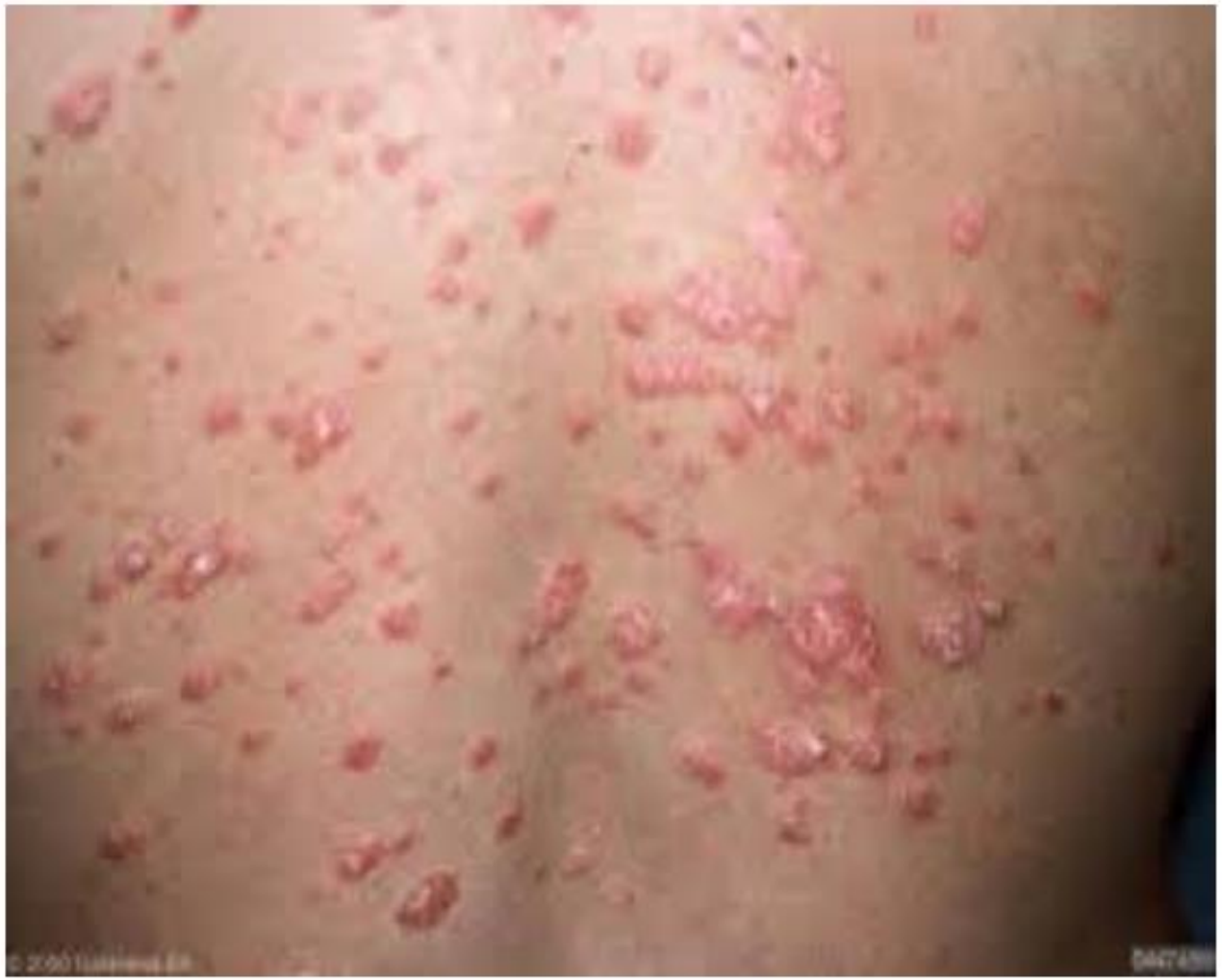
8. Guttate psoriasis

- **Characterized by** small, scattered, pink, oval (drop-shaped) papules with silvery scaling that usually appear on the trunk and extremities
- It typically occurs as new onset psoriasis in patients under 30 years of age
- Guttate psoriasis is often triggered by strep throat infections.
- Systemic antibiotic should be given
- **Differential diagnosis**: pityriasis rosea, lichen planus, pityriasis lichenoides
- Good prognosis



Small, erythematous papules (drop shaped lesions) covered with scales on the trunk and extremities.

Guttate psoriasis



Guttate psoriasis

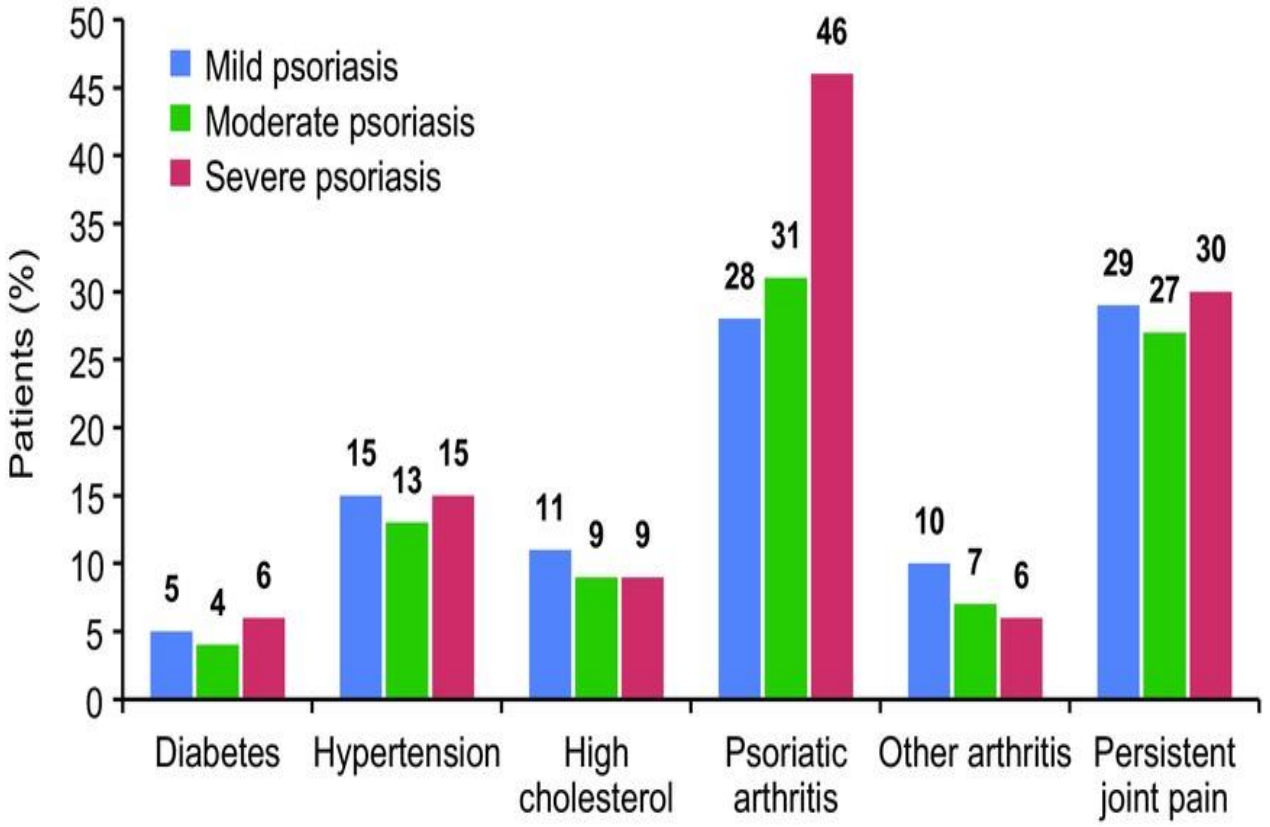
شرح

Exacerbating factors

- Infections, particularly strep throat
- Smoking, alcohol consumption, obesity
- **Drugs**: lithium, synthetic antimalarial drugs, tetracycline antibiotics, beta blockers, and NSAIDs (سنوات (2))
- Skin trauma
- Emotional stress
- In women, psoriasis severity often fluctuates with changes in hormone levels (High levels of disease activity are often observed during puberty, postpartum, and during menopause psoriasis often improve during pregnancy when levels of estrogen are increased)

Associated co morbidities

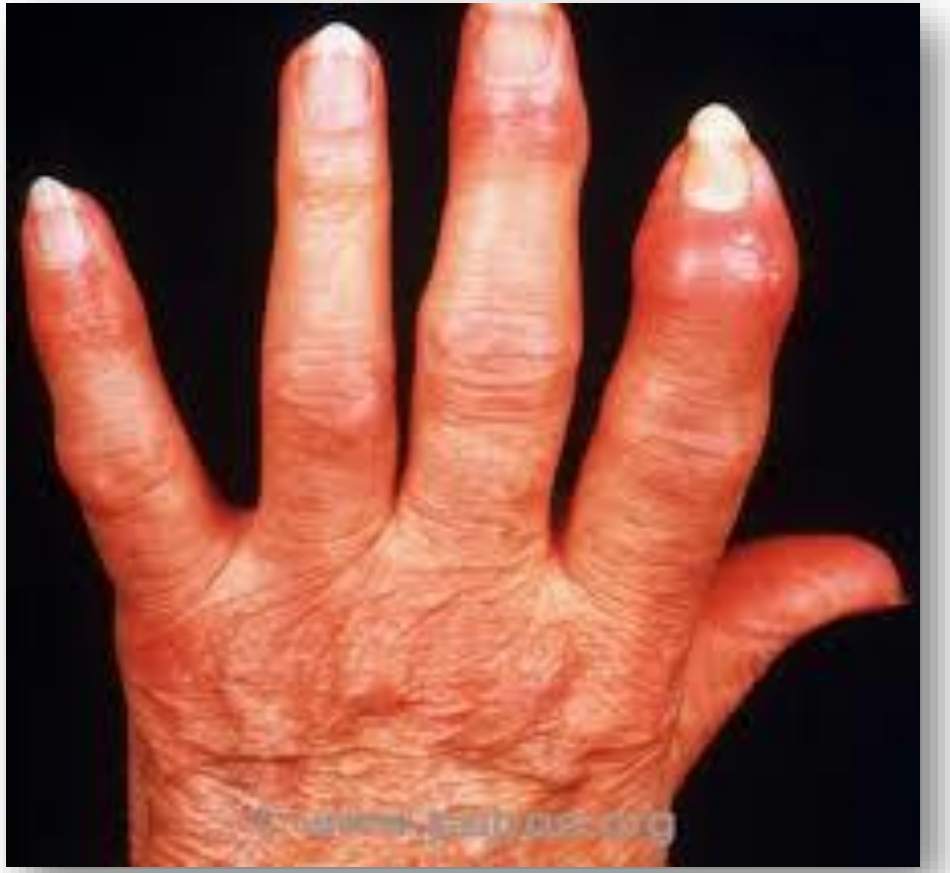
1. Psoriatic arthritis
2. Hyperlipidemia
3. Obesity
4. Hypertension
5. Hyper metabolic syndrome
6. Increased risk for cardiovascular disease



الصورة بس منظر

Psoriatic arthritis

- One in five patients with psoriasis has psoriatic arthritis (20%).
- It is a Seronegative arthritis.
- Nail changes is seen more with psoriatic arthritis.
- Dactylitis is a clinical feature.
- X-ray is helpful in the diagnosis.



Differential diagnosis for psoriasis

1. Eczema
2. Lichen planus
3. Fungal infection
4. Pityriasis rubra pilaris
5. Pityriasis lichenoides
6. Mycosis fundgoides
7. Secondary syphilis (especially in Guttate psoriasis)

شرح

Psoriasis Treatment

Topical treatment agents

1. Crude coal tar (Carcinogenic & smelly)
2. Emollients (Petrolatum / Vaseline)
3. Dithranol
4. Topical steroids
5. Topical calcipotriol (Vit.D derivative)
6. Topical Calcineurin inhibitor (Tacrolimus)
7. Topical retinoids (Vit. A derivatives)
8. Local phototherapy
9. Local laser treatment

Systemic treatment Options

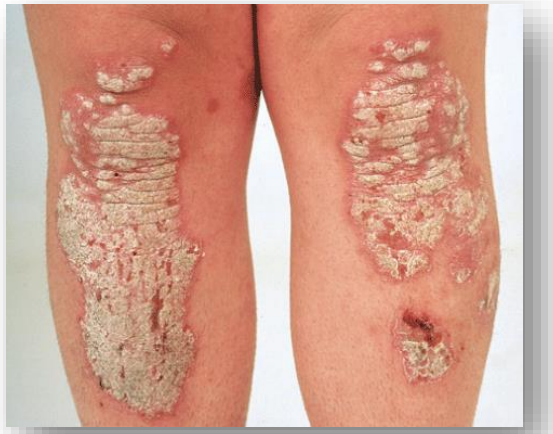
سنوات (5)

1. Phototherapy (PUVA and NB-UVB)
2. Methotrexate (low weekly dose)
3. Retinoids (Vit. A derivatives)
4. Cyclosporine
5. Apremilist
6. Biological treatment

- **Topical treatment** for less severe (<10% BSA) as first line of treatment
- **Systemic treatment** for more extensive and severe disease and failure of topical treatment

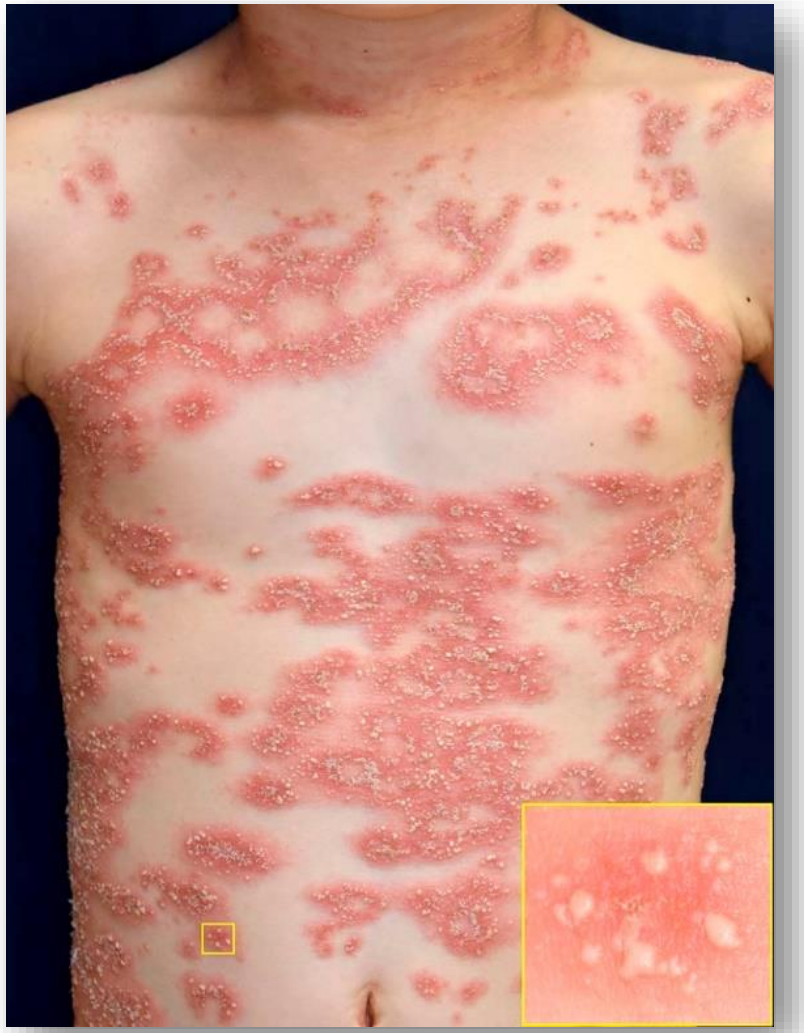
Psoriasis vulgaris

- **What is your diagnosis ?** Psoriasis vulgaris
- **What is this primary skin lesion ?** Plaque
- **Which drug MUST be avoided ?** Systemic steroid
- **Mention 3 drugs the exacerbate this condition**
  1. Lithium
  2. Synthetic antimalarial drugs
  3. Tetracycline antibiotics
  4. Beta blockers
  5. NSAIDs
- **Mention 3 nail changes associated with this disease**
  - Oil spot, Onycholysis, Pitting

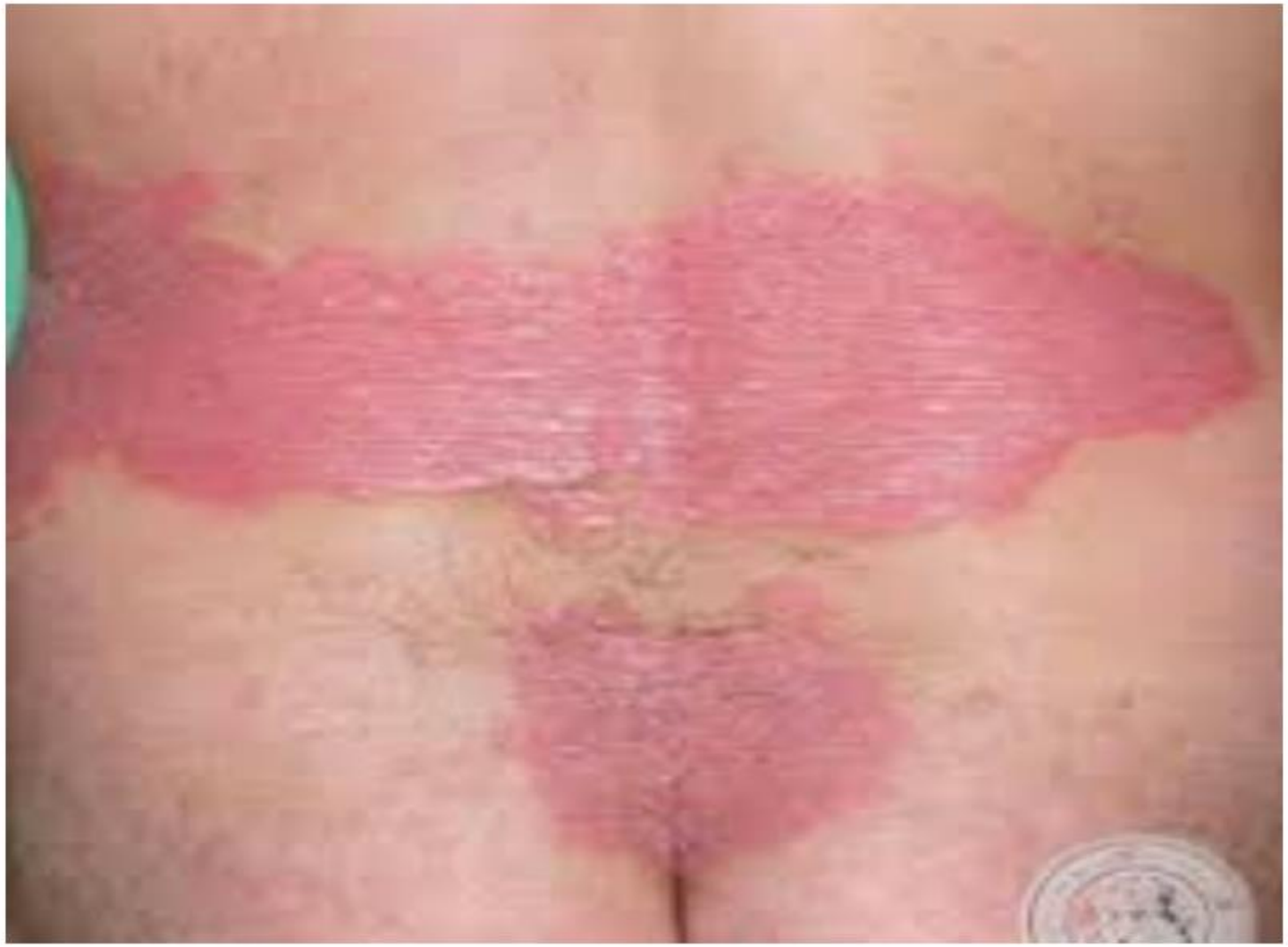


Child presented with itchy scaly lesions with positive auspitz sign

- **Define auspitz sign**
  - Pinpoint bleeding when scale is picked off
- **This signs is associated with what disease ?**
  - Psoriasis
- **Which of drug is contraindicated ?**
  - Systemic steroids
- **Mention 3 systemic treatments**
  1. Cyclosporine
  2. Methotrexate
  3. Biological Agents



Psoriasis



Salmon like colored plaque with silver scales
Psoriasis

Erythrodermal psoriasis is extensive psoriasis

Ichthyosis

السُماك

شرح

Ichthyosis

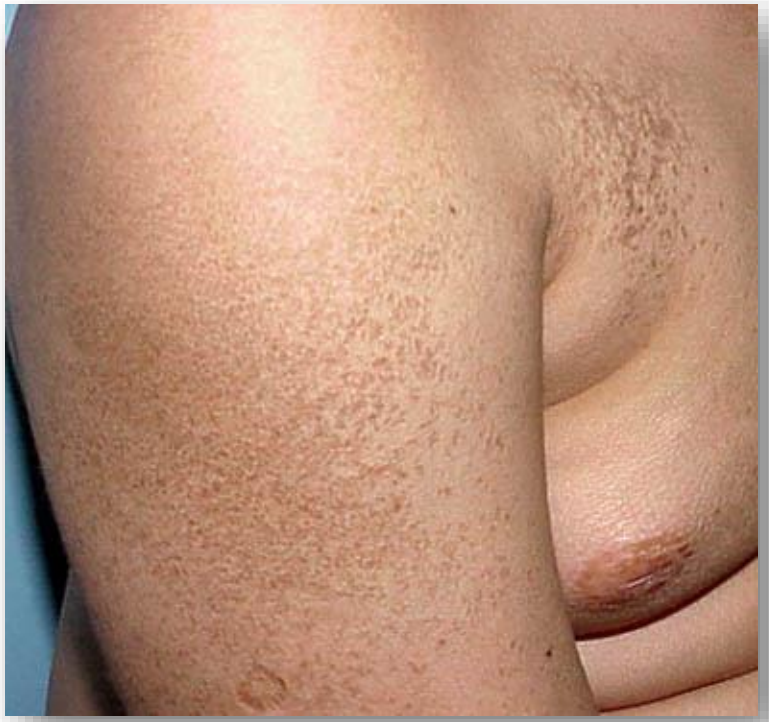
- **Definition**: A heterogeneous group of skin conditions characterized by dry and scaly skin due to impaired keratinization.
- Characterized by generalized scaling of the skin
- Can be **congenital** or **acquired**
- **Mention 3 variant of congenital ichthyosis** سنوات (4)

1. Ichthyosis vulgaris
2. Steroid sulfatase deficiency
3. Collodion baby
4. Lamellar ichthyosis
5. Congenital Ichthyosiform erythroderma
6. Bullous-congenital Ichthyosiform erythroderma
7. Netherton syndrome
8. Harlequin Ichthyosis

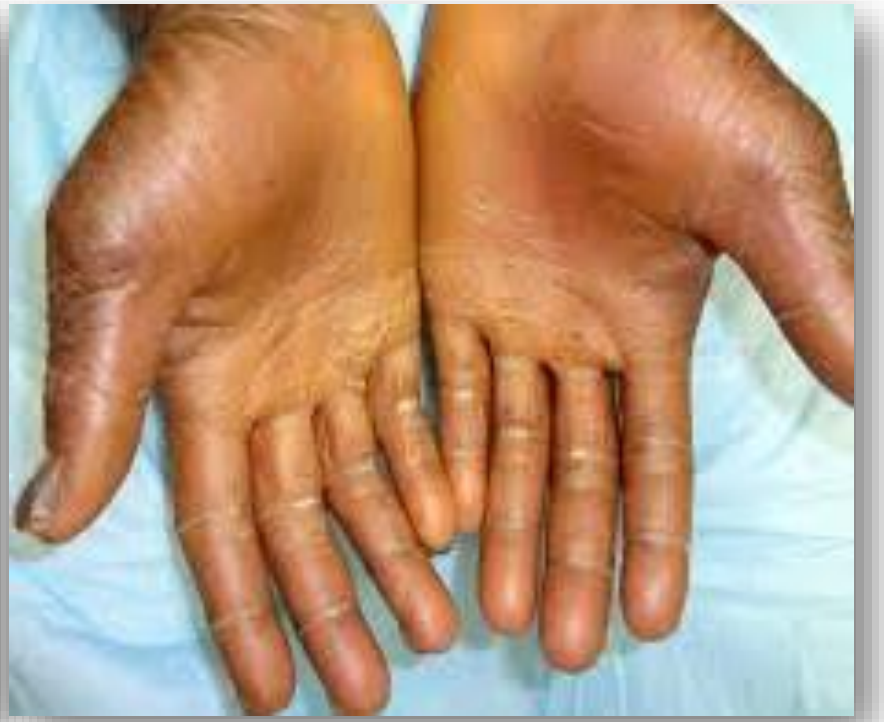
1. Ichthyosis vulgaris

- **Inheritance**: Autosomal dominant
- **Defect**: Loss of function mutations in the filaggrin gene (FLG)
- **Age on presentation**: Not present at birth, appear later after few months
- **Clinical presentation**:
  - Dry skin, fine scales on extensor surfaces sparing the groin and flexural area
  - Increased skin marking of palms and soles (Thickening of skin), mild hyperkeratosis.
  - Improves in summer, worsens with cold and dry weather
  - Improves with age
- **Associations**: Keratosis pilaris, Atopic dermatitis and Asthma
- **Treatment**: Emollients, keratolytics, ceramide containing lipid cream, urea

1. Ichthyosis vulgaris



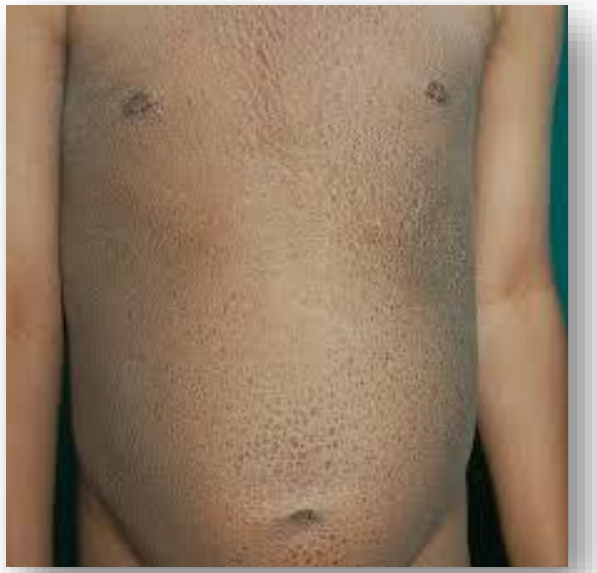
Fine scales with dryness sparing the flexural area.

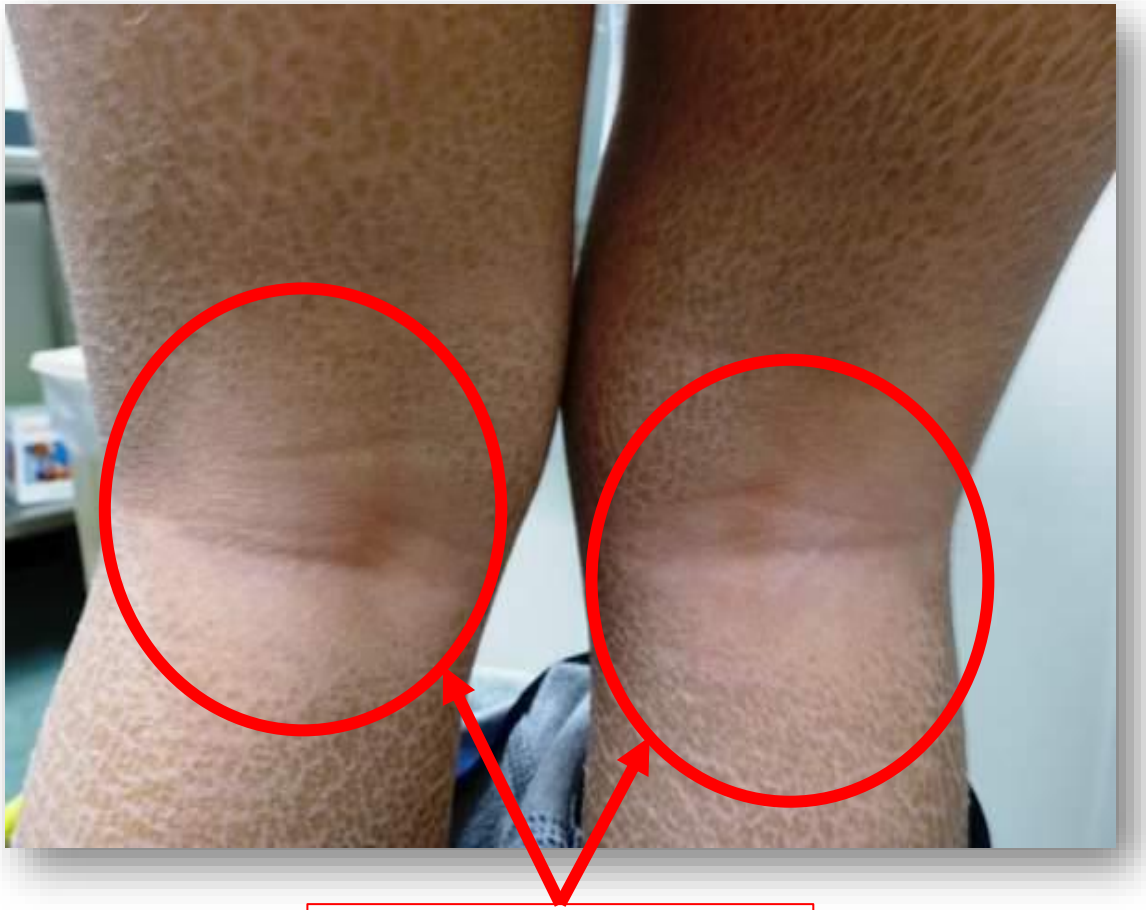


Palmoplantar thickening with increased palmar markings.

Ichthyosis vulgaris

- **What is the diagnosis ?**
  - Ichthyosis vulgaris
- **Mention the spares area in ichthyosis vulgaris:**
  - Groin and flexural area

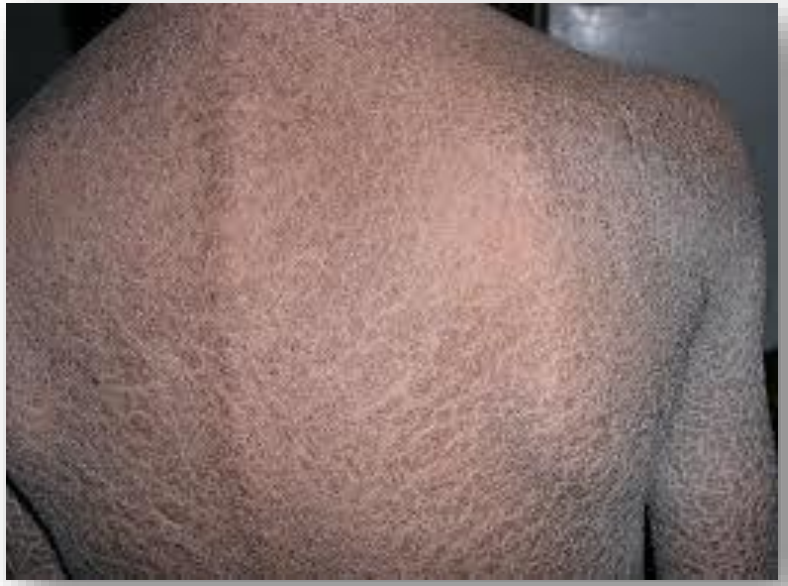


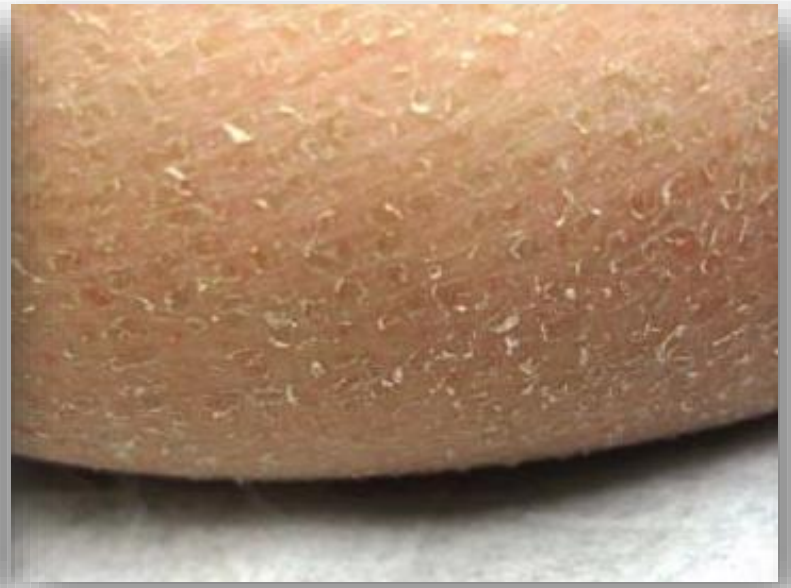


2. Steroid sulfatase deficiency

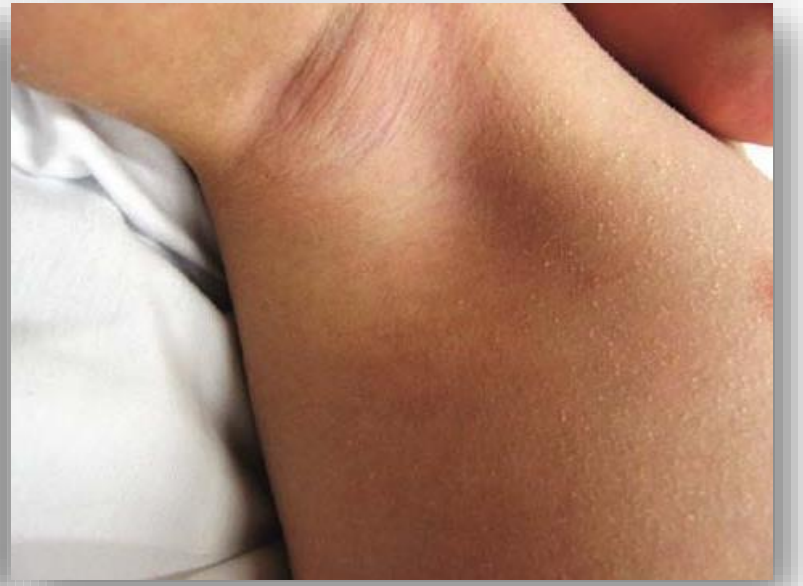
- **Inheritance**: X-linked recessive; 90% are boys
- **Defect**: Complete absence of steroid sulfatase caused by complete deletion of the STS gene
- **Age on presentation**: Present within the first weeks after birth
- **Clinical presentation**:
  - Typical large polygonal dark-brown scale with tight adherence to the skin develop later during infancy.
  - Symmetrical involvement,Sparing the palms and soles.
  - May spare the flexures except the neck which is always involved (dirty neck).
  - Do not improve with age
- **Associations**: Cryptorchism, Asymptomatic corneal opacities 10-50%
- ~~**Treatment**~~

2. Steroid sulfatase deficiency





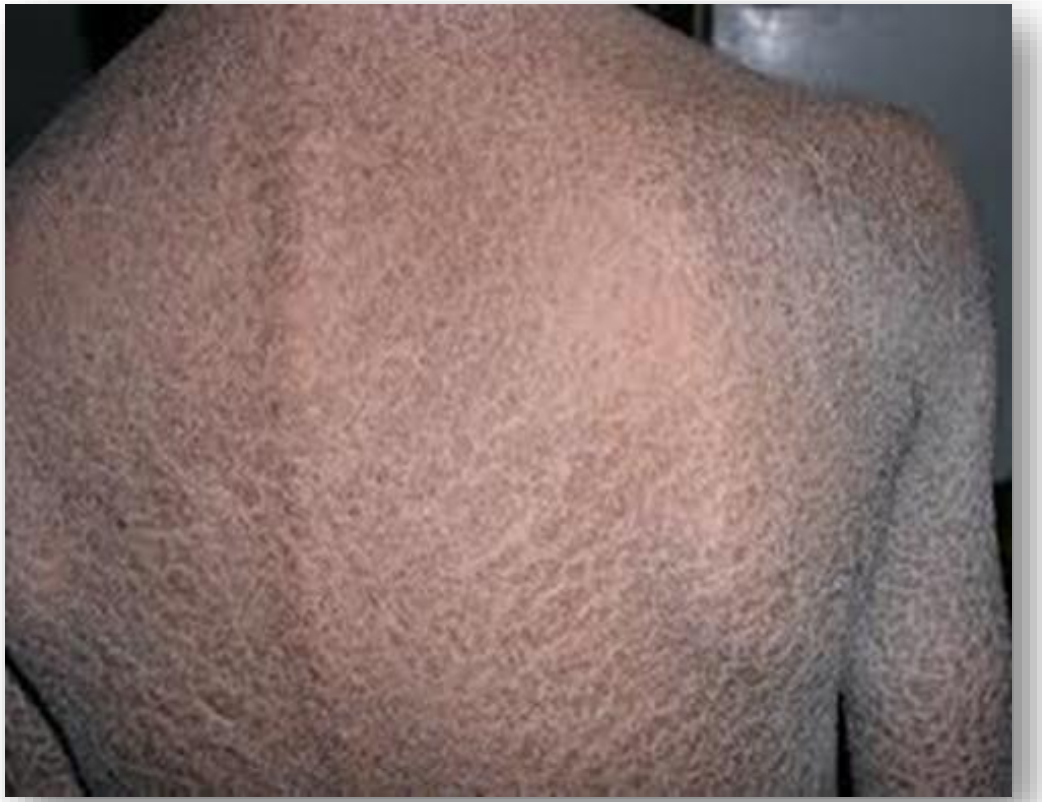
Dark adherent scales.



Dark adherent scales sparing the flexural areas except the neck

This pt has history of corneal opacity & Cryptorchism, diagnosis

- **What is your diagnosis ?**
  - X-linked recessive ichthiosis
- **What enzyme absence is the cause of this case ?**
  - Steroid sulfatase



3. Collodion baby

- **Inheritance**: Commonly Autosomal recessive
- **Defect**: Premature birth
- **Age on presentation**: At birth covered with a taut shiny and transparent membrane that resemble a plastic wrap.
- **Clinical presentation**:
  - Ectropion, Eclabium, hypoplasia of nasal and auricular cartilage.
  - After birth the membrane dries, cracks and breaks up, fissure develop.
  - Dehydration, hypoxia, malnutrition and pulmonary infection may result (need special care)
  - Within 2 wks. the membrane peels off → congenital Ichthyosiform erythroderma or lamellar ichthyosis.
  - In some cause normal skin appear or mild exfoliation.
- **Treatment**: Topical antibiotics and emollients

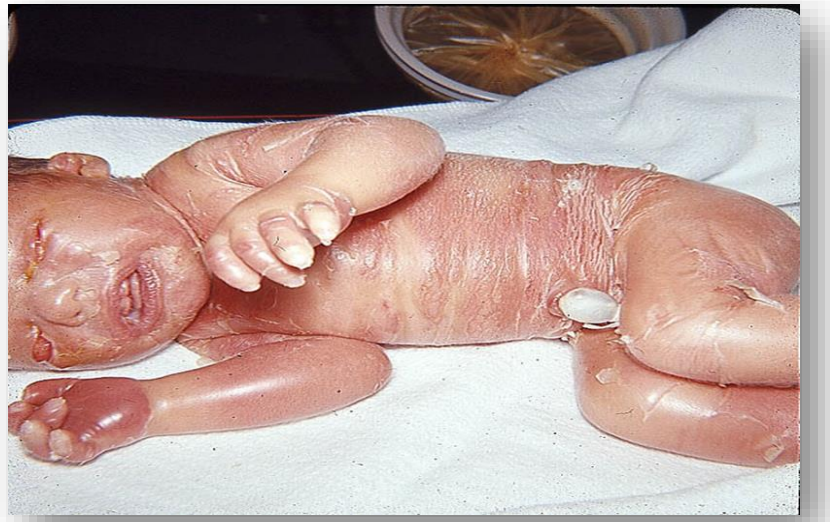
Baby born encased with transparent rigid membrane

سنوات (2)

- What is your diagnosis ?
  - Collodion baby

سنوات (1)

- What happen next ?
  - Can transform into either
    - Congenital Ichthyosiform erythroderma
    - Lamellar ichthyosis



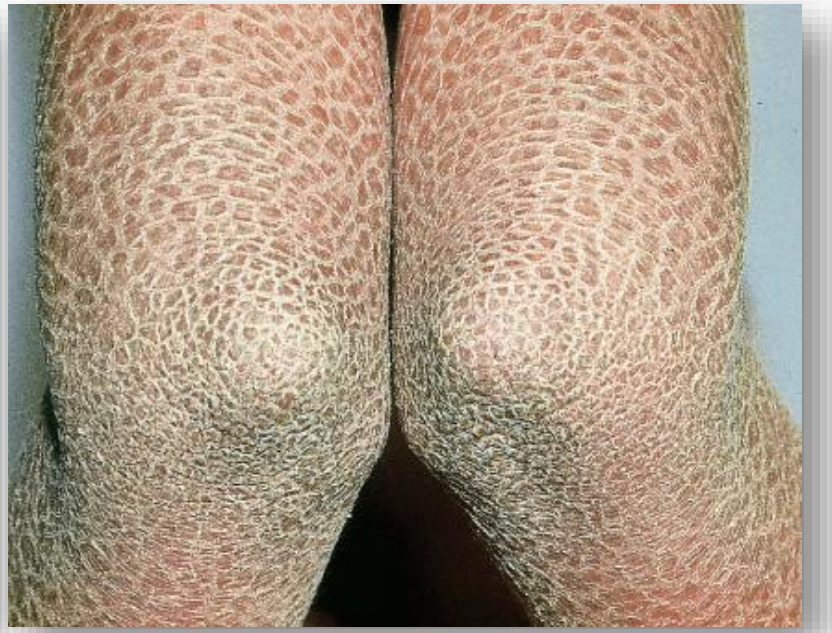
4. Lamellar Ichthyosis

سنوات (1)

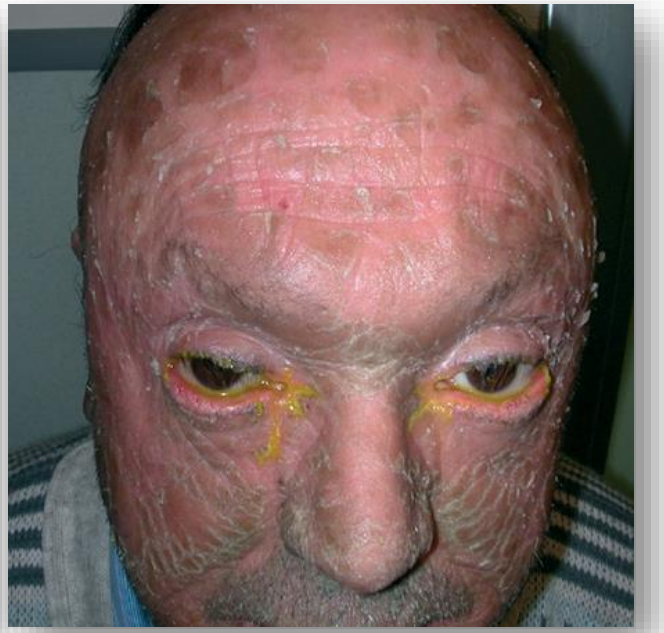
- **Inheritance**: Autosomal recessive
- **Defect**: Transglutaminase deficiency due to mutation of TGMI gene
- **Age on presentation**: Appears at birth as collodion baby
- **Clinical presentation**:
  - Severe disorder.
  - Large, dark-brown plate – like scale.
  - Ectropion, eclabium, hypoplasia of nasal and auricular cartilages.
  - Scarring alopecia because of taut skin.
  - Mild to sever palmoplantar keratoderma
- **Treatment**: Acitretin from early childhood

4. Lamellar Ichthyosis

Very large dark scales, Ectropion, eclabium, deformed nose and ears and hair loss



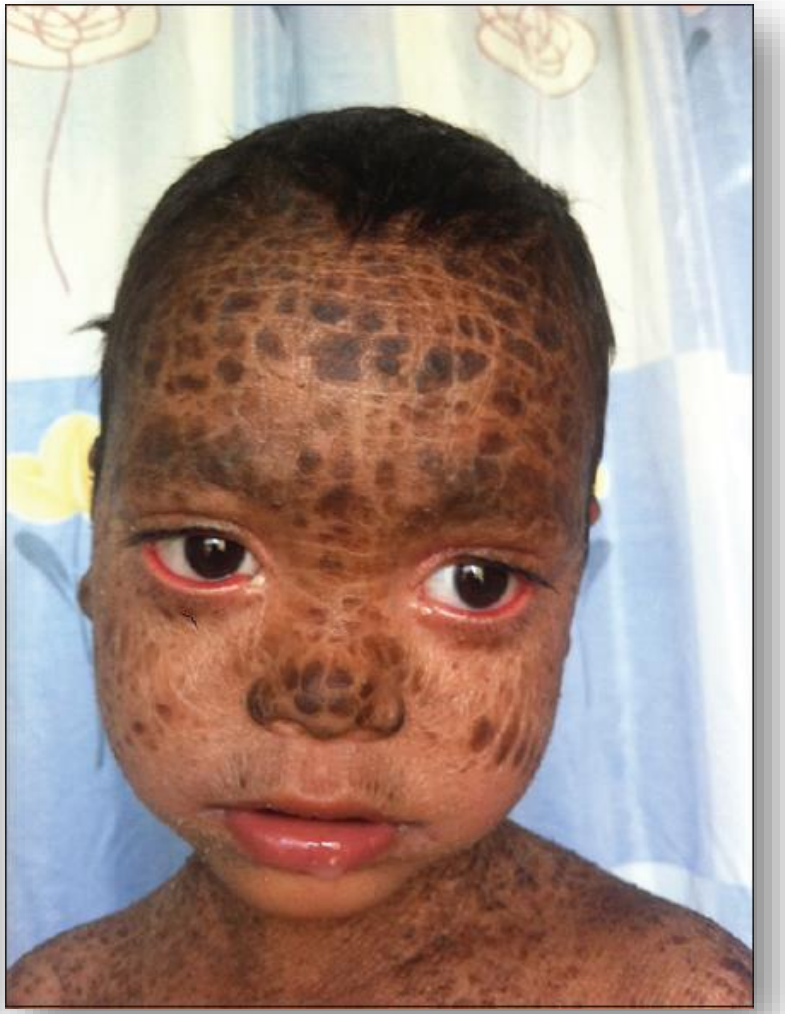
Very large disfiguring scales



Ectropion, with exposure keratitis as a complication

This child was born as collodion baby, diagnosis

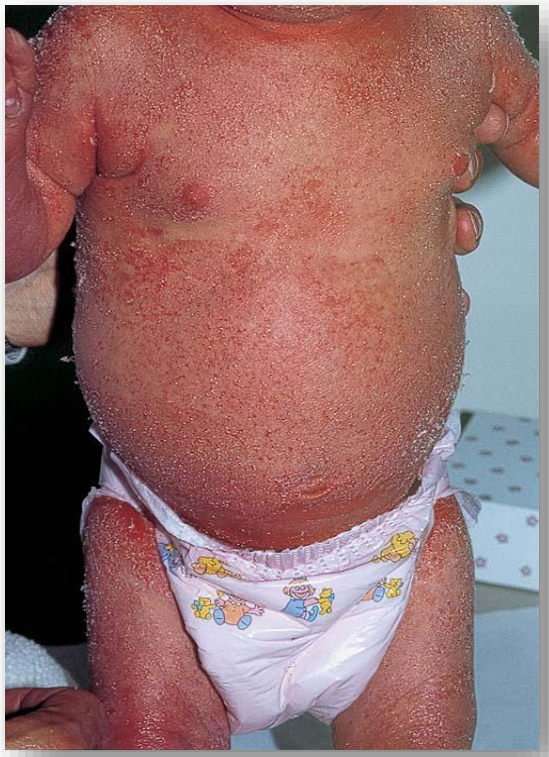
A. Bullous congenital ichthyosifor erythroderma
B. X-linked icthyosis
C. Lamellar icthyosis
D. Icthyosis vulgaris

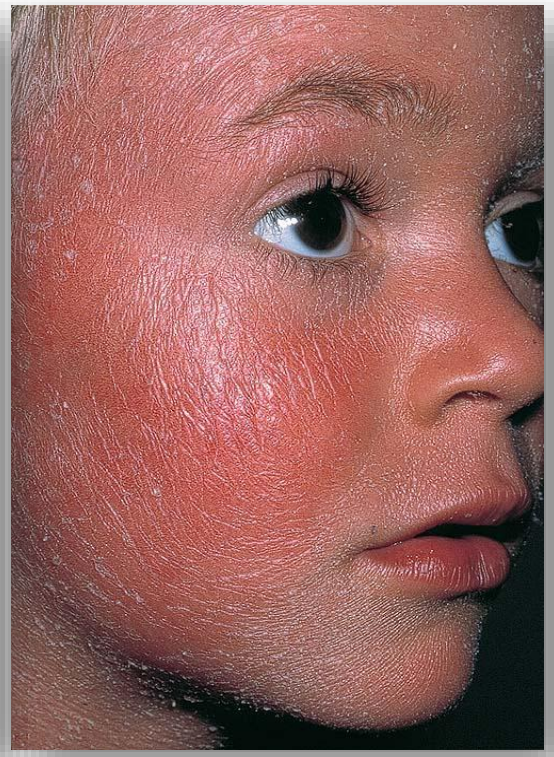


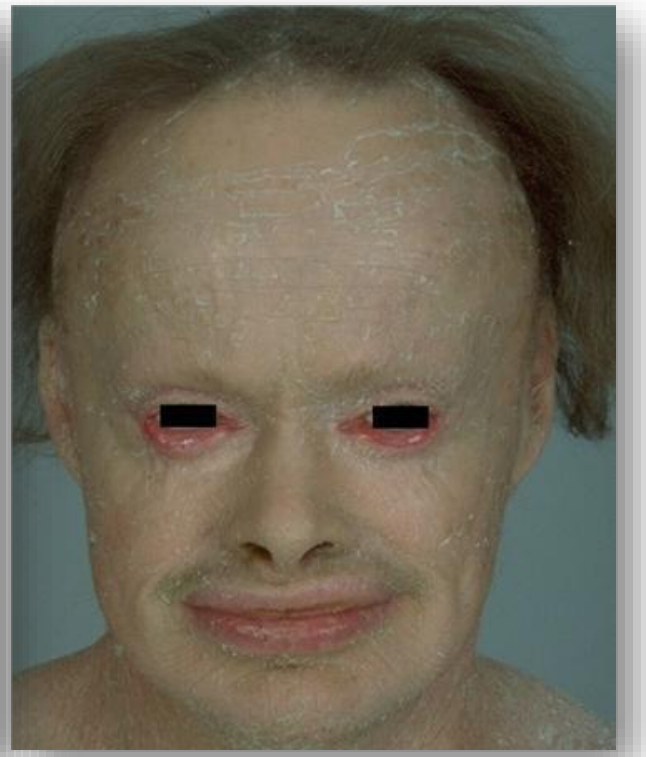
5. Congenital Ichthyosiform erythroderma

- **Inheritance**: Autosomal recessive
- **Age on presentation**: Present at birth as collodion baby
- **Clinical presentation**:
  - Generalized erythroderma with persistent scaling throughout life.
  - Milder presentation than lamellar ichthyosis.
  - Ectropion.
  - Scarring alopecia.
  - Bright erythroderma, generalized, white powdery scales.
  - Severe palmoplantar keratoderma with fissuring.
- **Treatment**: Acitretin as lamellar ichthyosis

5. Congenital Ichthyosiform erythroderma







Erythroderma with mild scaling.

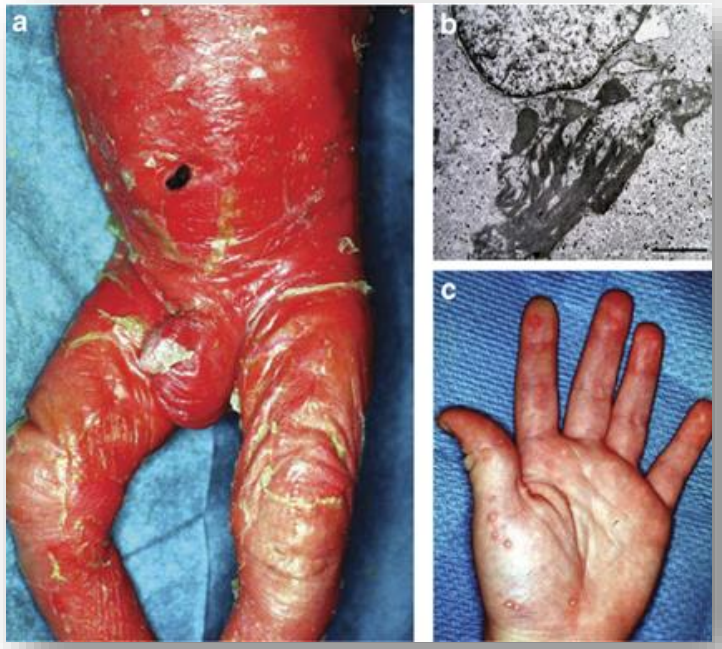
Scaling, scarring, Ectropion, Eclabium, deformity of the ears

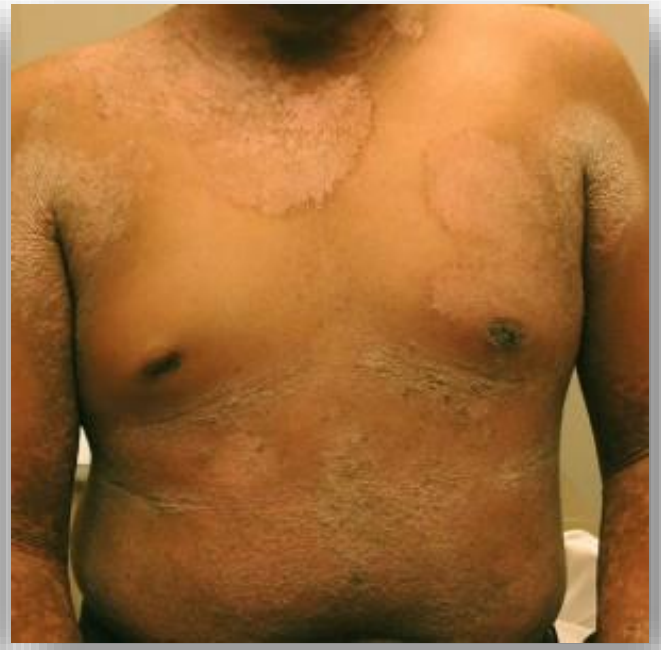
6. Bullous congenital Ichthyosiform erythroderma

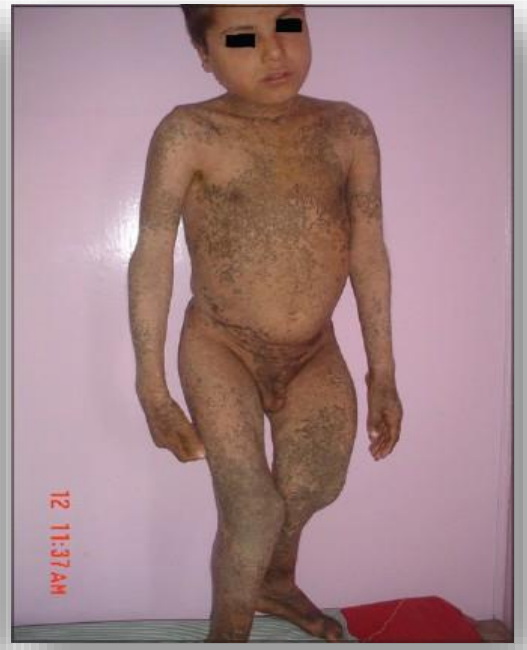
(Epidermolytic hyperkeratosis)

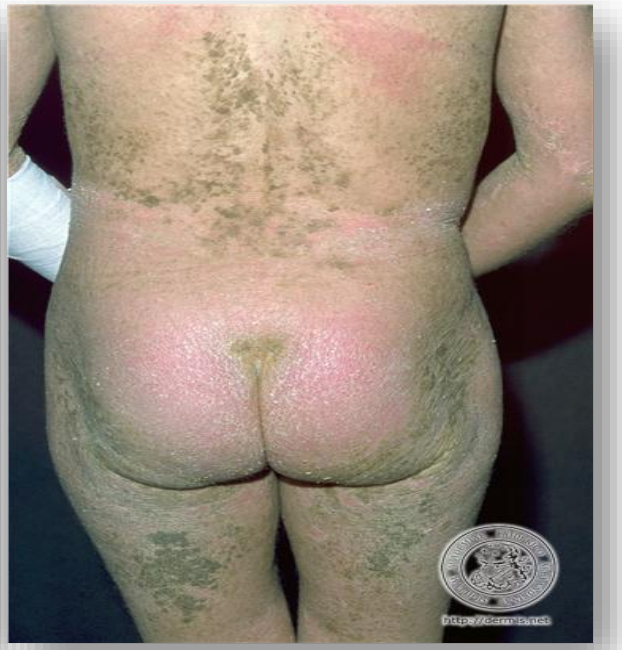
- **Inheritance**: Autosomal dominant, 50% are sporadic
- **Defect**: heterogeneous mutation in the gene encoding keratin 1(KRT1) and keratin 10(KRT10)
- **Age on presentation**: At birth with erythroderma and erosions, Over time erythroderma decrease and hyperkeratosis prevails
- **Clinical presentation**:
  - Different clinical forms of presentation in different families.
  - Chronic, disfiguring with great impact on social life
- **Treatment**: According to the age, emollients, keratolytic, Retinoids

6. Bullous congenital Ichthyosiform erythroderma









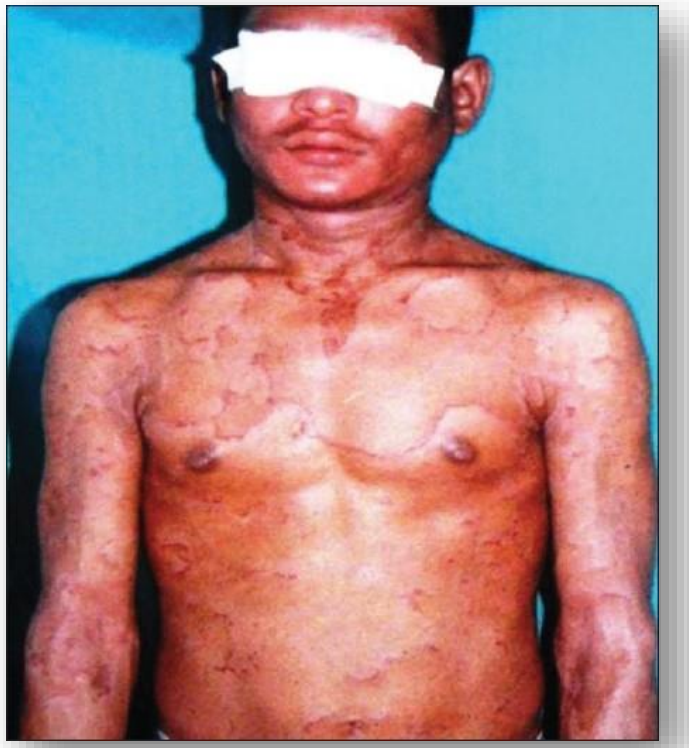
Erythroderma

Later on, hyperkeratosis, thickening of the skin and disfiguring lesions appear.

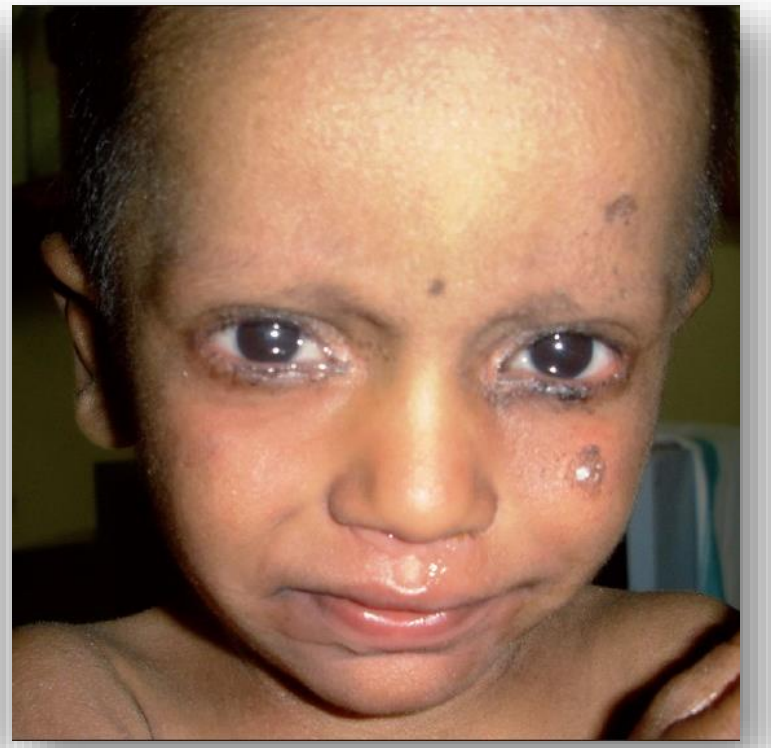
7. Netherton syndrome

- **Inheritance**: Autosomal recessive
- **Triad of** congenital ichthyosis, Trichorrhexis invaginata and atopy
- **Age on presentation**: Present at or soon after birth with erythroderma and scaling, no collodion baby
- **Clinical presentation**:
  - Gradually evolves into circinate scaling and erythematous plaques (Ichthyosis linearis circumflexa) over trunk and extremities and change over time.
  - Eczematous pruritic plaques due to atopy.
  - Scalp involvement.
  - Hair shaft abnormality since infancy (bamboo hair) improves with age.
- **Associations**:
  - Elevated serum IgE due to atopy.
  - Increased susceptibility to infection.
  - Mental retardation can occur
- **Treatment**: If symptomatic: emollients, Retinoids and phototherapy

7. Netherton syndrome



Ichthyosis linearis circumflexa.



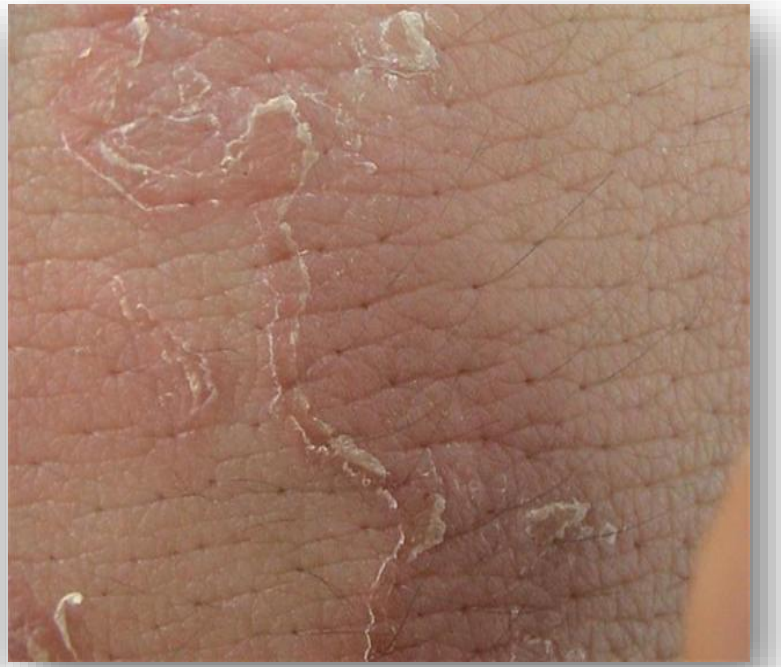
Scalp alopecia

Hair defects with eczematous features on the face

7. Netherton syndrome

Bamboo hair which is a feature of Trichorrhexis invaginata.

Trichorrhexis invaginata under light microscopy.



Double edge scaling which is a feature of Ichthyosis linearis circumflexa.

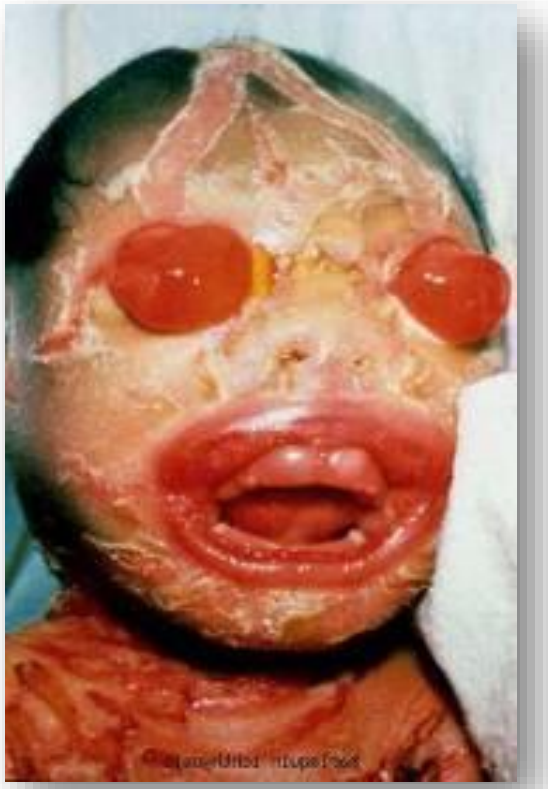
7. Netherton syndrome

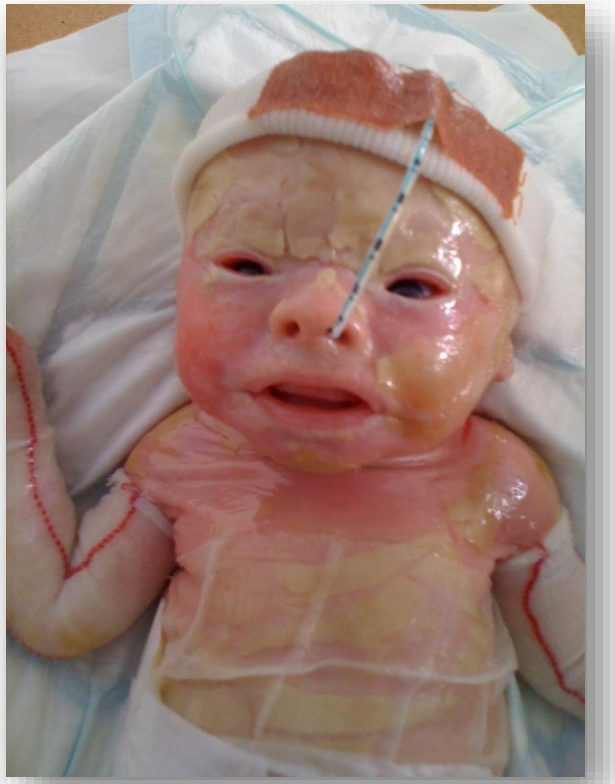
- What is this finding ?
  - Bamboo hair
- It is seen in what ?
  - Netherton syndrome

8. Harlequin Ichthyosis

- Most extreme and distinct form of congenital ichthyosis.
- **Age on presentation**: Premature baby, die within few days or weeks After birth
- **Clinical presentation**:
  - Encased in a hard, armor-like thick stratum corneum that severely immobilizes the baby.
  - After birth this taut cast cracks and form large, yellow adherent plates with deep fissures resembles a harlequin`s costume.
  - Ectropion – eversion of eyelids.
  - Eclabium – eversion of lips.
  - Microcephaly.
  - Edematous hand and feet, digits are well developed.
  - Eyelashes and eyebrows are missing.
- **Treatment**:
  - Need special care for water and electrolyte balance and prevention of sepsis
  - Retinoids

8. Harlequin Ichthyosis



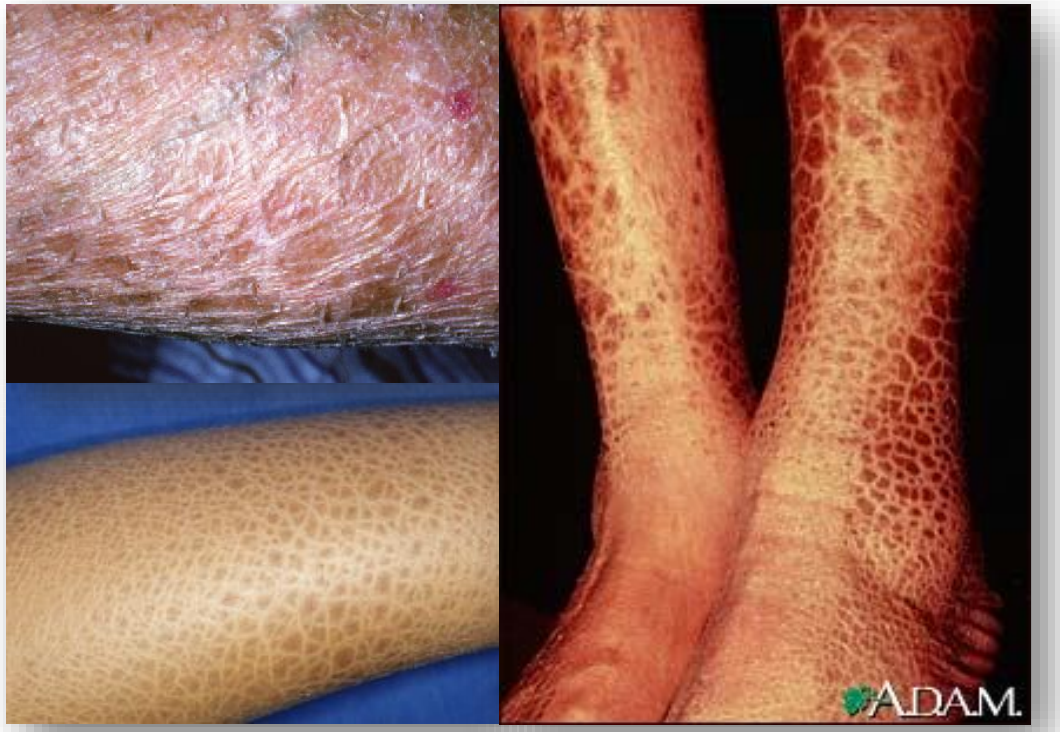


Armor plate like lesions, Ectropion and eclabium

Acquired Ichthyosis

❖Any patient presented with acquired Ichthyosis must be investigated to rule out these causes (Mention the causes of acquired Ichthyosis):

1. Sarcoidosis.
2. Polycythemia rubra Vera.
3. Cutaneous T cell lymphoma
4. Leprosy.
5. TB.
6. Hodgkin`s lymphoma.
7. Lupus erythematosus.
8. Dermatomyositis.
9. Carcinomas.
10. Thyroid disease.



Dry, cracked, scaly skin.

Pigmentary disorders

Skin color

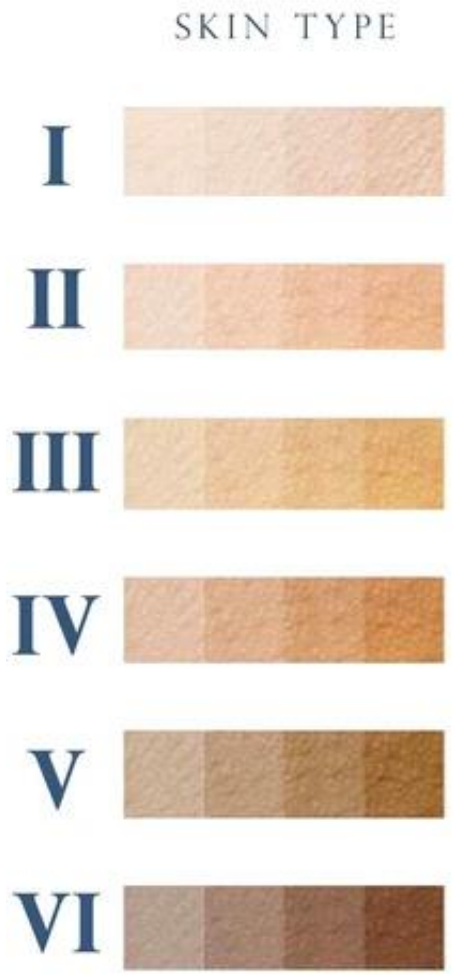
❖**Skin color factors**:

- Hemoglobin (Pallor in anemia)
- Exogenous pigments in or on the skin surface
- Endogenously produced pigments (e.g., bilirubin)
- The pigments produced in the skin itself: melanin and phaeomelanin
- Carotenemia (Orange in color)

❖The different skin colors result from the size and number of melanosomes not number of melanocytes. (i.e., Negro skin contains no more melanocyte than fair people).

Mention and describe the skin types

- Type I
  - Skin burns very easily and doesn't tan. Likely to have light blonde or red hair.
- Type II
  - Skin will usually burn in the sun. and has difficulty tanning.
- Type III
  - Skin will sometime burn and will tan gradually.
- Type IV
  - Skin will tan easily and rarely burn.
- Type V
  - Skin will tan without burning.
- Type VI
  - Skin never burns and will tan very quickly.



Melanin

- Produced by melanocytes in the epidermal basal layer
- Synthesized from tyrosine in melanosomes (in melanocytes) by tyrosinase enzyme
- Melanosomes is the site of synthesis and storage of melanin. It can be passed from melanocytes to keratinocytes
- Its function to protect cell nuclei from damage by UV
- Types:
  - Eumelanin: deep brown-black
  - Pheomelanin: red mainly in hair

موجود بالمحاضرة لكنه مش شيء رئيسي ممكن ينسأل عنه حطيته كونه بساعد بالفهم

Disorders of pigmentations

1. Disorders of pigmentation can result from migration abnormalities of melanocytes from the neural crest to the skin during embryogenesis (Albinism)
2. In addition, impairment of melanosome transfer to the surrounding keratinocytes
3. An alteration in melanin synthesis
4. A defective degradation or removal of melanin may lead to abnormal skin pigmentation
5. Immunologic or toxic mediated destructions of melanocytes can end in pigmentation disorders (Vitiligo)

❖Disorders of pigmentation are classified in hypo- or hyperpigmentation which can occur as a genetic or acquired disease

❖They can manifest locally or diffuse

Hyperpigmentation

Freckles

سنوات (1)

❖**Primary lesion in freckles**:

- Hyperpigmented macule

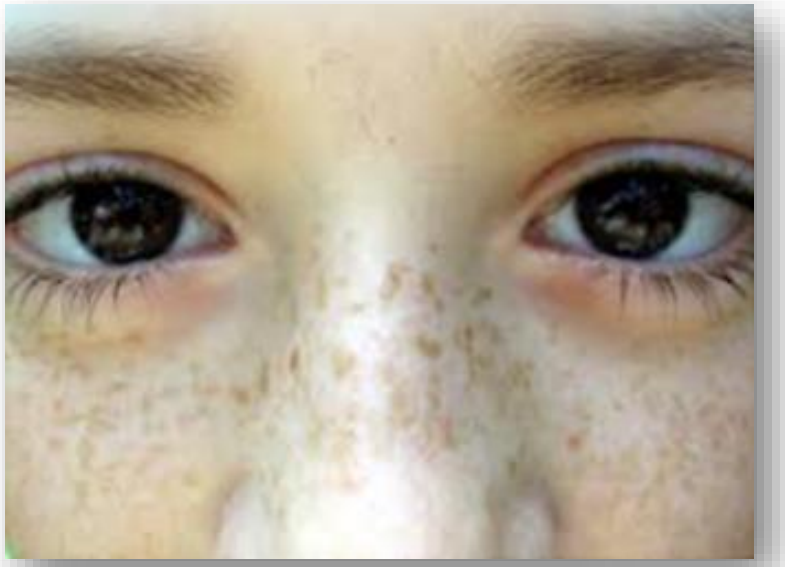
❖**Predisposing factors**:

- Genetics and sun exposure are the primary causes of freckles
- People with red, blonde, or light brown hair and who have light-colored skin and eyes usually produce mainly Pheomelanin and are more likely to develop freckles.

❖People can **prevent** or **reduce** the appearance of freckles by protecting their skin from the sun

Freckles

سنوات (2)
- **Describe:**
  - Hyperpigmented macules over the checks and nose



سنوات (2)
- **Mention 2 ddx**
  - Freckles, Melasma

سنوات (7)
- **What is the best treatment**
  - Sunblock

سنوات (1)
- **What is the topical treatment**
  - Sun protection

Melanocytic nevi (moles)

سنوات (1)

❖**Primary lesion of junctional nevus**: Hyperpigmented macule

❖Rarely can develop into melanoma, especially the junctional type because they are more superficial

- For this reason, moles should be watched for bleeding, pain, itch, color, shape, symmetry, even borders, and size changes.
- No moles occur after 45 years of age, so after this age is considered melanoma until proven otherwise.

❖Laser can treat the junctional type only.

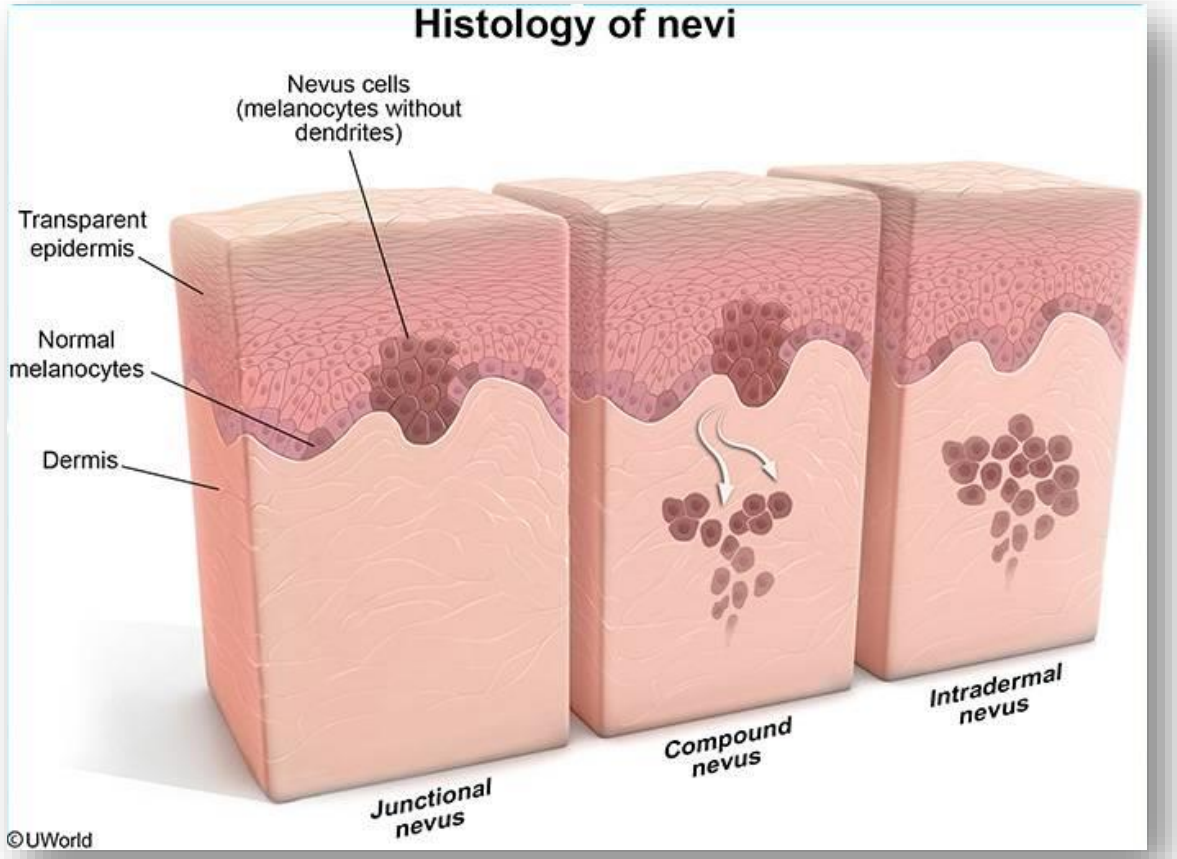
❖The **ABCDE**s of melanoma are:

- **A**: Asymmetry, If you divide your mole in half, both sides should look the same
- **B**: Border irregularity, The border of your mole should be even
- **C**: Color variation, Your mole should be one color
- **D**: Diameter > 6 mm
- **E**: Evolution or elevation over time

Melanocytic nevi (moles)

Mention the types of nevi

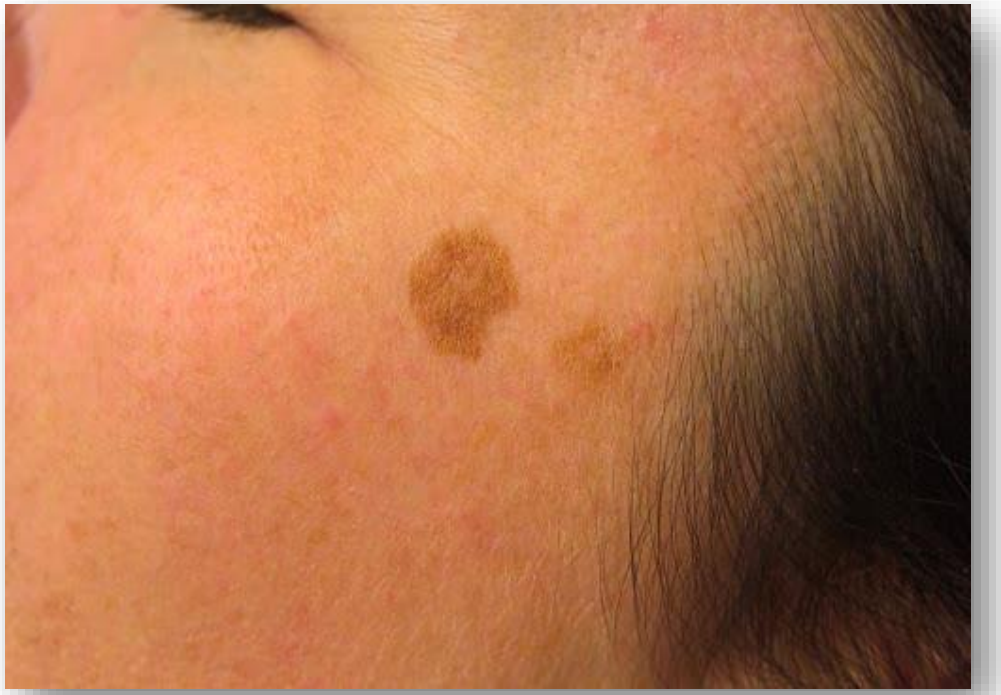
- **Junctional nevi**: flat macules, most commonly seen in children
- **Compound nevi**: slightly elevated, when junctional nevi extend to the dermis
- **Intradermal nevi**: raised papules, most commonly seen in adults



Simple lentigines

سنوات (1)

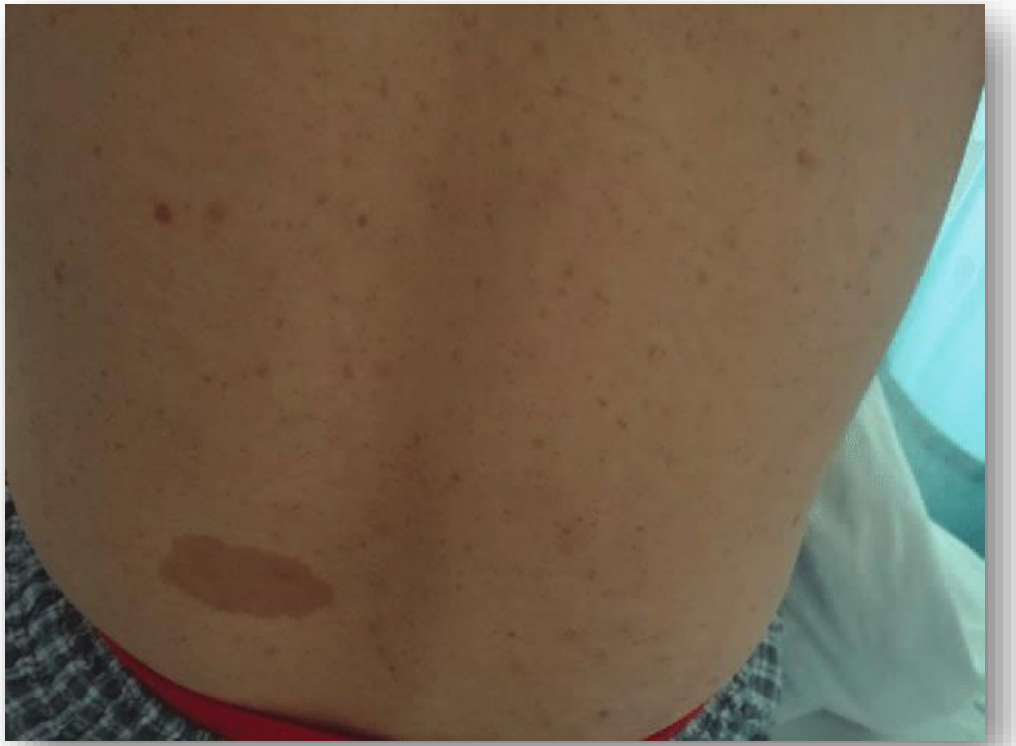
- **Primary lesion of lentigines**: Hyperpigmented patch or macule
- Simple lentigines are the result of increased melanocytes in the stratum basale layer of the skin and sometimes increased melanin content in the upper layers of the epidermis and stratum corneum, brown in color.
- They can occur anywhere on the skin and also involve the lips, inside the mouth, and genitalia.



Café-au-lait spots

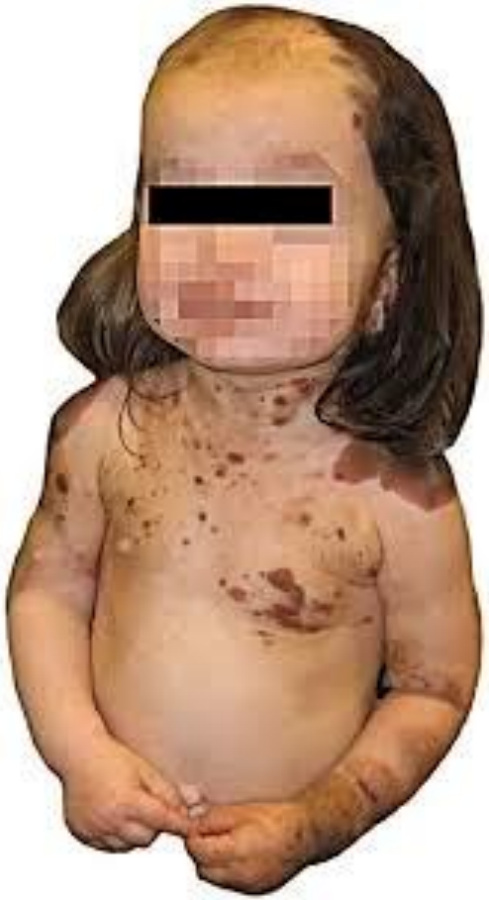
سنوات (1)

- **Primary lesion of Café-au-lait**: patch
- **Which systemic disease is Café-au-lait associated with ?**
  - Neurofibromatosis
- Six or more spots of at least 5mm in diameter in pre-pubertal children or 15mm in post-pubertal individuals is one of the major diagnostic criteria for diagnosing **Neurofibromatosis**



Giant congenital nevi

- Congenital melanocytic naevi are usually classified by their size in an adult.
- There are several different classifications.
  - A small congenital melanocytic naevus is < 1.5 cm in diameter.
  - A medium congenital melanocytic naevi is 1.5–19.9 cm.
  - A large or giant congenital melanocytic naevus is ≥ 20 cm in diameter, treated by Plastic surgery.



Acanthosis Nigricans

- **Definition**: Thick velvety skin in a skin fold.
- **Description**:
  - Hyperpigmented (dark) plaques on skin
- **Location**:
  - Intertriginous sites (folds)
  - Classically neck and axillae
- **Dermatopathology**
  - Hyperkeratosis
  - Mild acanthosis
- **Associated with** سنوات (2)
  - Insulin resistance
  - Rarely associated with malignancy

Melasma (Chloasma) (الكلف)

- **Melasma (Chloasma)**: is marked by tan or brown patches that may appear on the forehead, cheeks, upper lip, nose, and chin
- Can occur during pregnancy ("pregnancy mask"), women who are taking birth control pills or postmenopausal estrogen

سنوات (1)

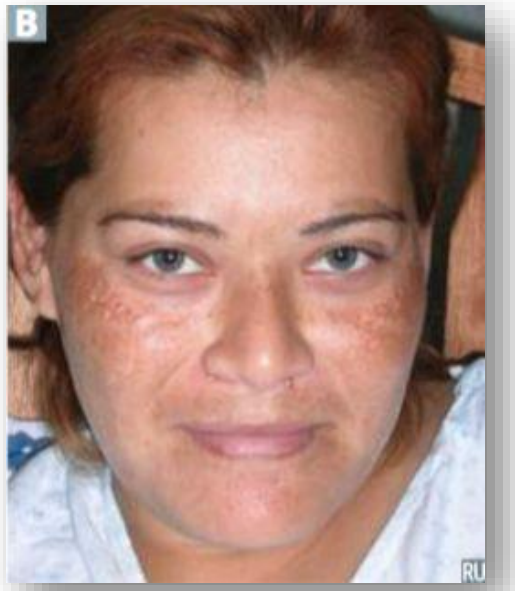
- **Primary lesion of melasma**: tan or brown patch on sun exposed area

سنوات (1)

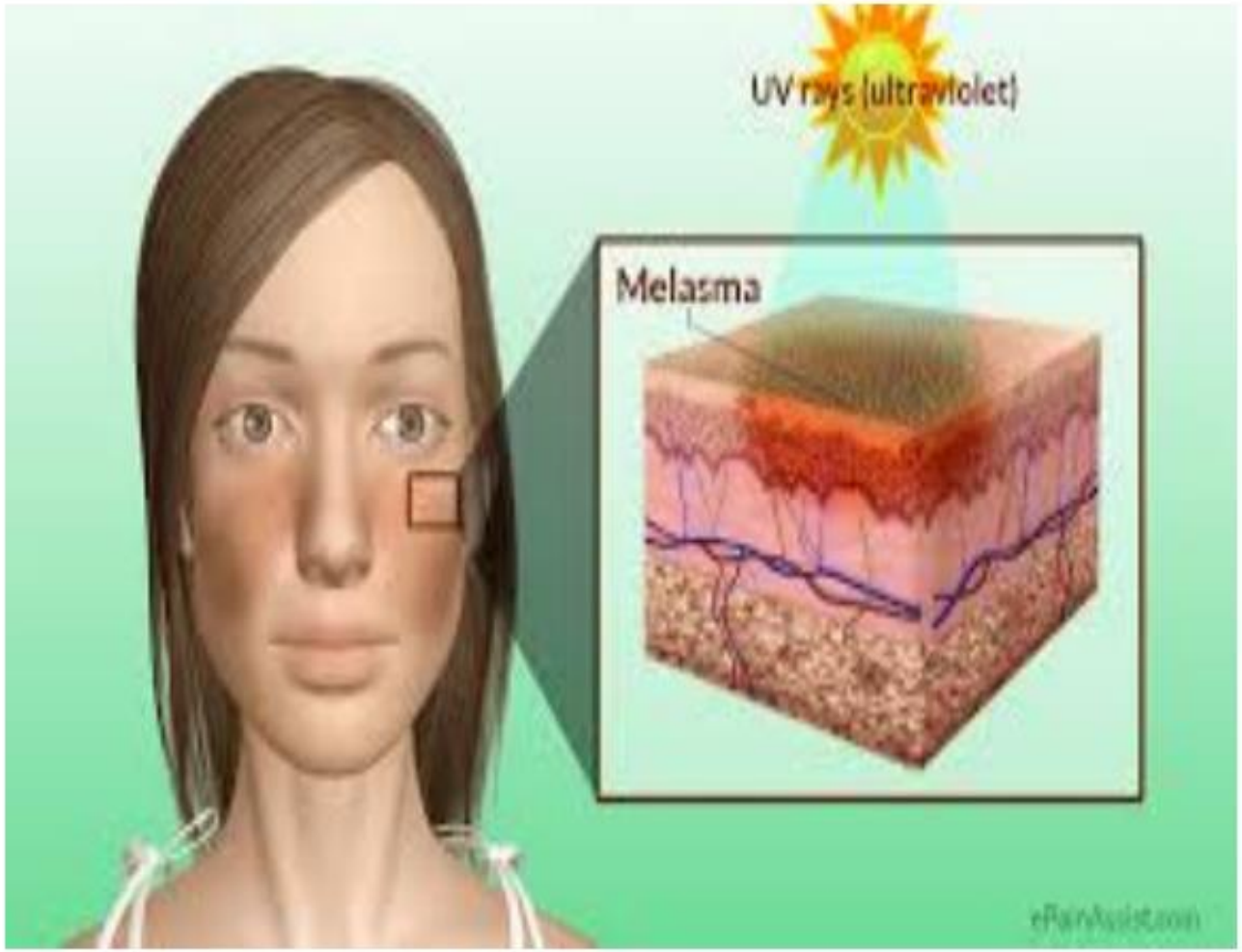
- **What is the topical treatment of melasma ?**
  - Sun protection, Azelaic acid, mild cleanser

سنوات (1)

- **Management Approach For Melasma**
  1. Sunblock
  2. Emollient creams
  3. Leaser Therapy
  - Melasma may go away after pregnancy



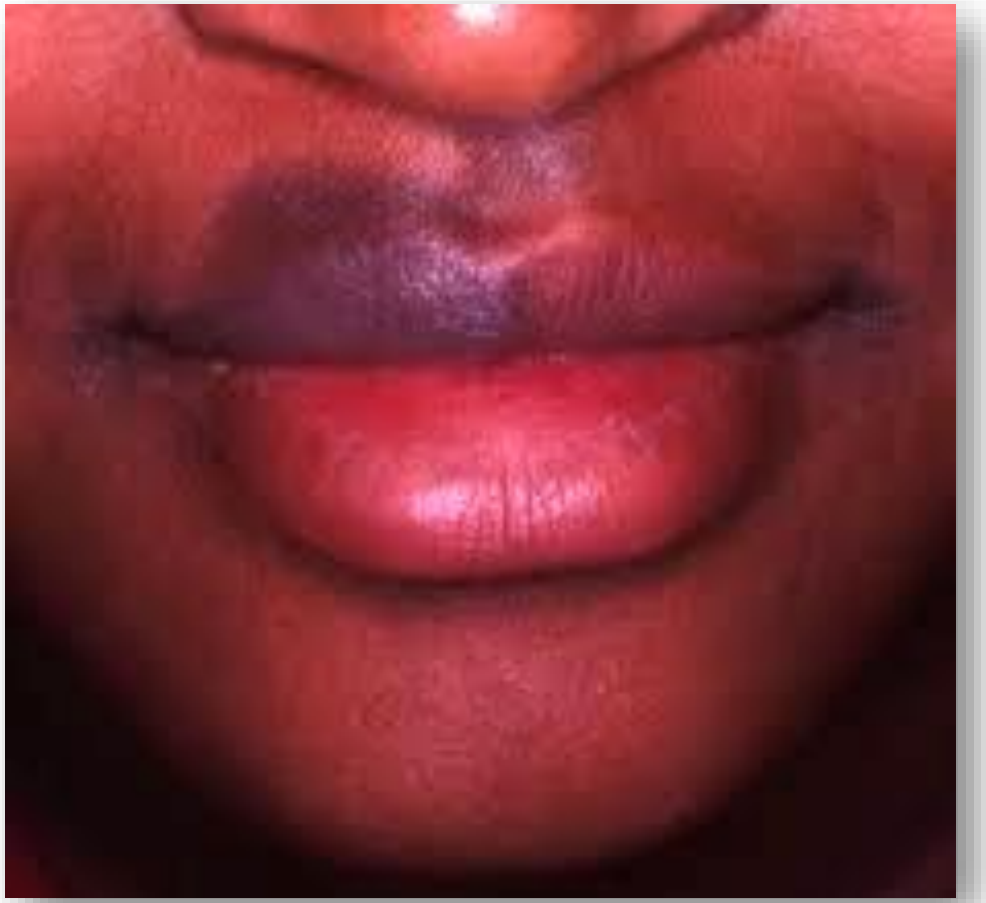
Melasma (Chloasma)



Melasma
Hyperpigmentation

Post inflammatory hyperpigmentation

- In post-inflammatory hyperpigmentation, disruption of the epidermis results in deposition of melanin granules in the dermis (pigmentary incontinence).
- Many skin disorders do this, particularly in pigmented skin.
- There is no useful treatment, but the pigmentation gradually fades with time.
- Causes of post inflammatory hyperpigmentation
  1. Lichen planus.
  2. Fixed drug eruption.
  3. Eczema.



Hypopigmentation

Albinism (البرص)

- Defect of melanin production results in little or no color in skin, hair and eyes
- Due to congenital inability to form melanin patients have fair skin blonde hair and pink irises.
- Have poor vision photophobia.
- Increase risk of developing skin Cancer.
- **Oculocutaneous albinism**: severe form, disease affect the eyes causing vision problems
- **Ocular albinism**: rare form where the disease affect only the retina

Piebaldism

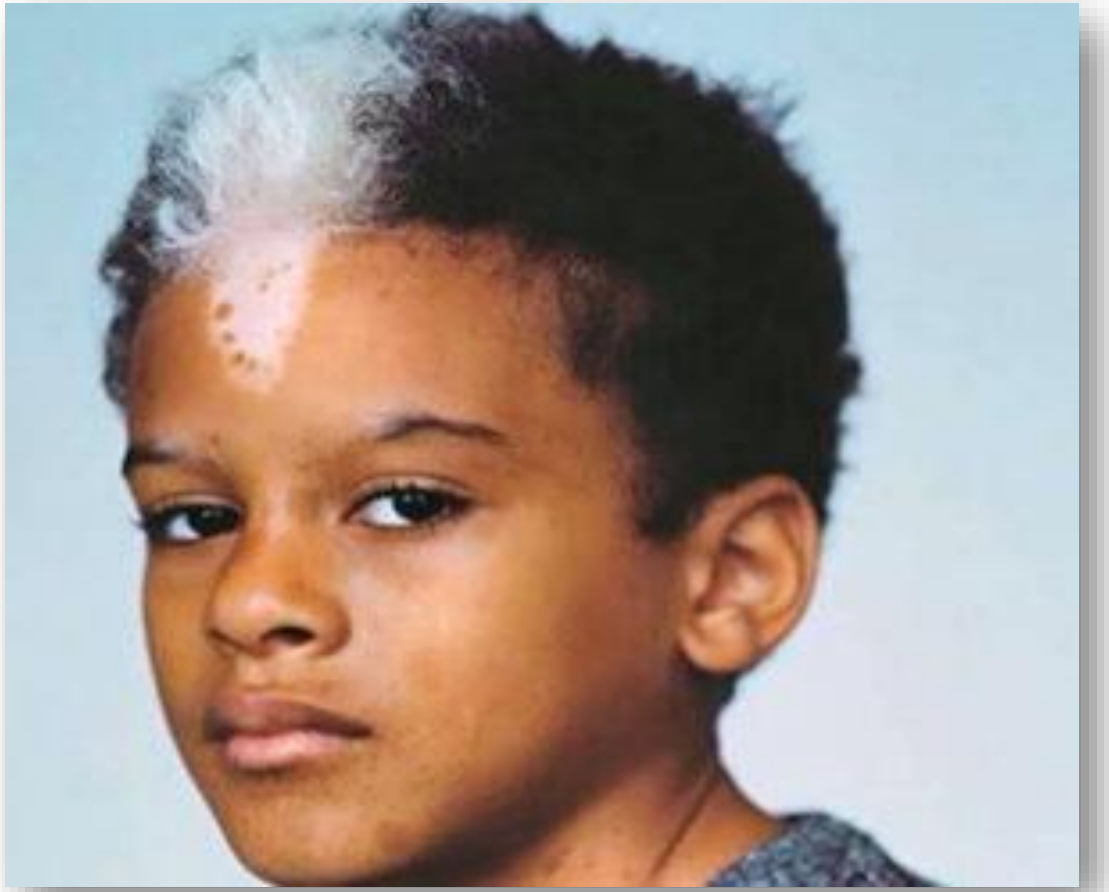
سنوات (1)

❖**What is your diagnosis ?**

- Piebaldism

شرح

❖**Piebaldism**: is a genetic condition, typically present at birth, in which a person develops an unpigmented or white patch of skin or hair.



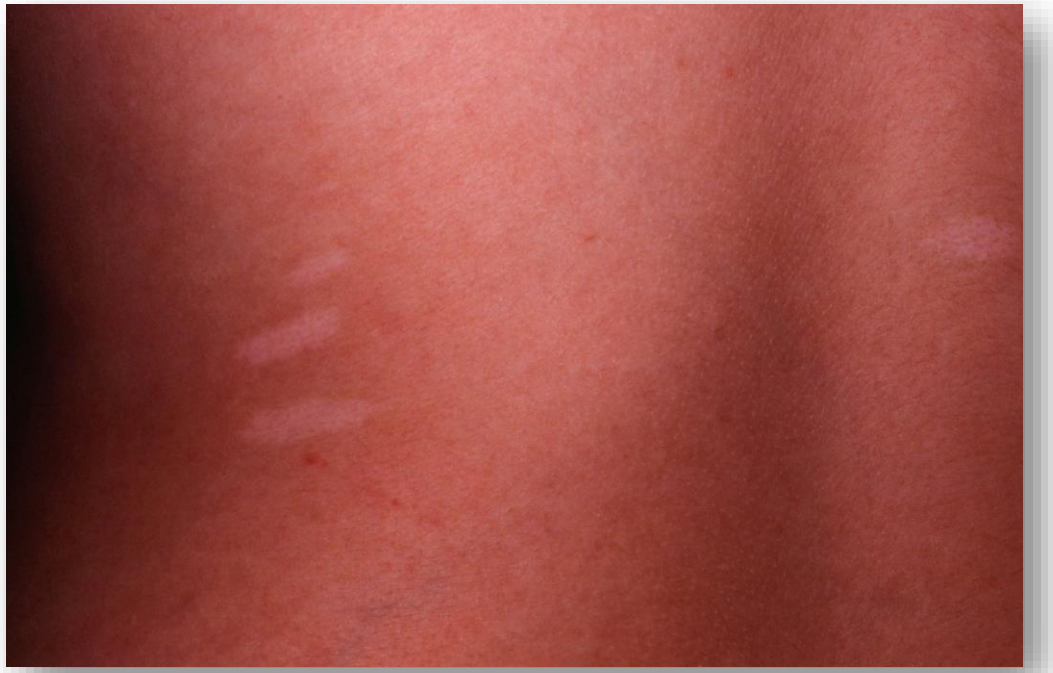
404

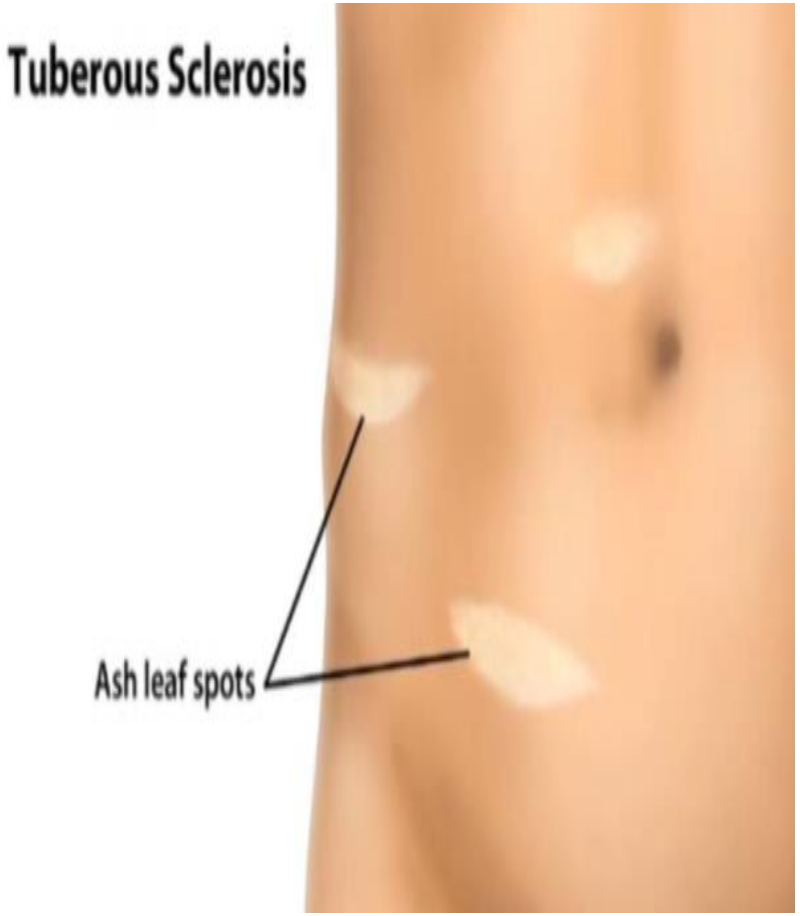
Not Found

The resource requested could not be found on this server!

Ash-leaf hypopigmentation

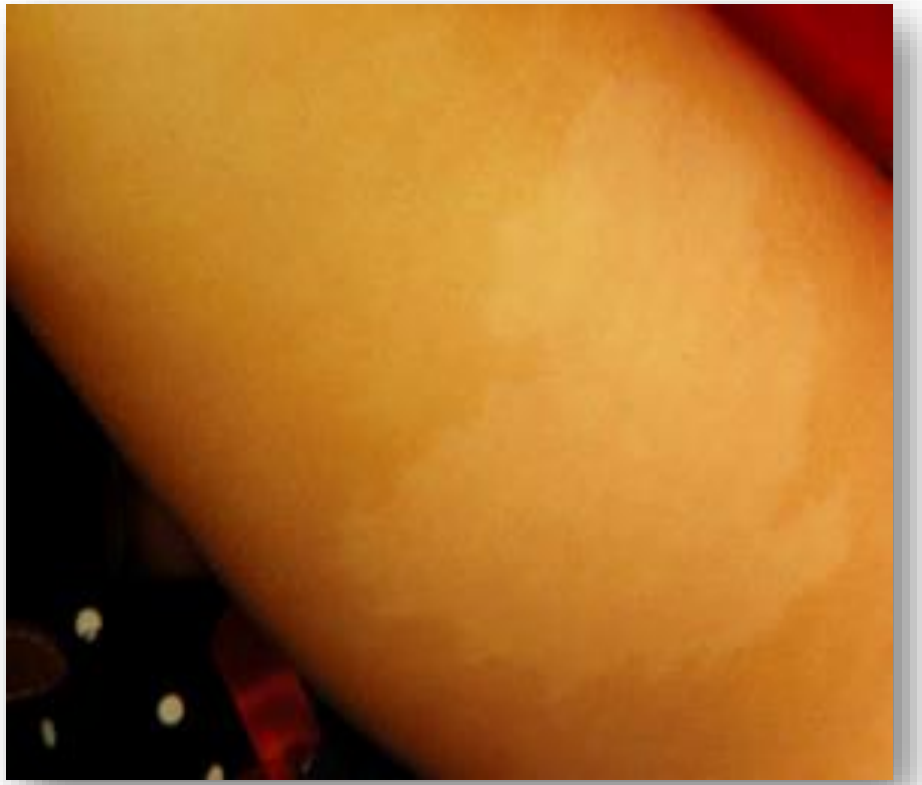
- The presence of more than three ash-leaf spots is characteristic of tuberous sclerosis





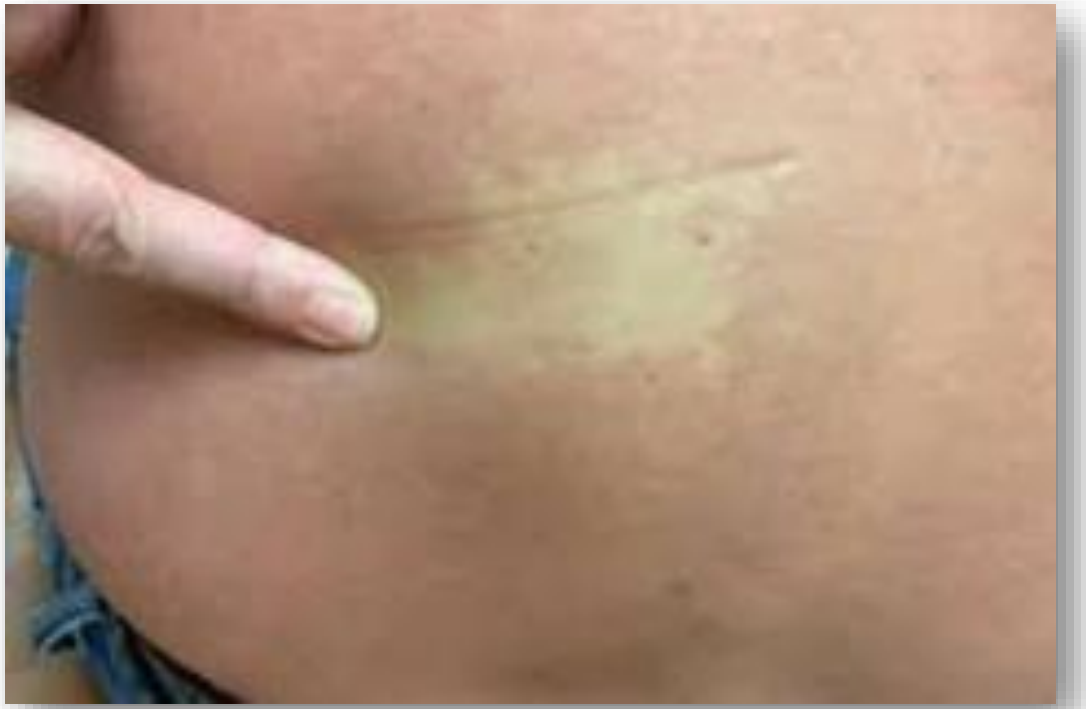
Achromic naevus (hypochromic nevus)

- **Achromic naevus (nevus anemicus)** is an uncommon birthmark characterized by a well-defined pale patch.
- **Characteristic**: This is usually several centimeters in diameter, with an irregular but well-defined border.
- Shape and size varies.
- Often, smaller hypopigmented macules arise around the edges, resembling a splash of paint.
- Negative in wood's light.



Lesion since birth, what is the diagnosis

A. Vitiligo
B. Pityriasis alba
C. Psoriasis
D. Neurofibromatosis
E. Nevus anaemicua



Vitiligo (بهاق)

- **Vitiligo**: Skin condition resulting from loss of pigment which produces white patches
- **Primary lesion of vitiligo**: milky white depigmented patch (سنوات (1))
- **Common areas**: face, lips, hands, arms, legs
- Vitiligo often begins with a rapid loss of pigment; this may continue until for unknown reasons the process stops
- **What investigations should be dome for patient with vitiligo ?**
  - Investigations to rule out other autoimmune diseases

Vitiligo

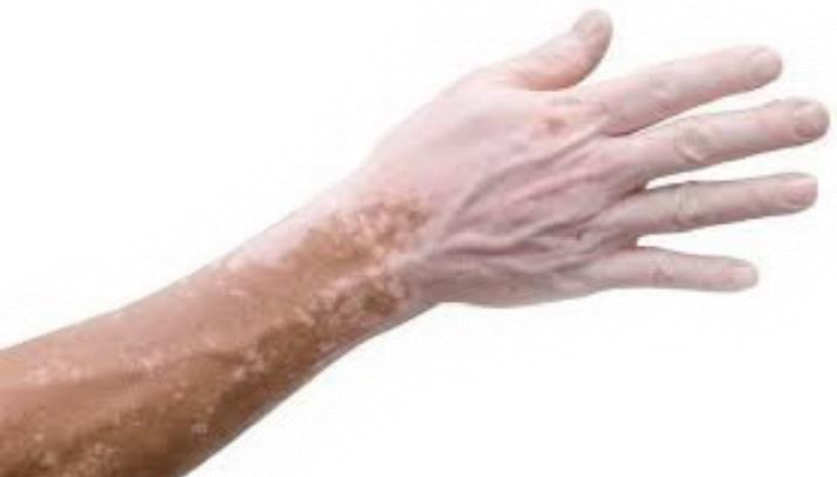
❖Management of vitiligo

- Sometime the best treatment for vitiligo is no treatment at all, Because these area are easily sun burned.
- Repigmentation therapy (for small areas of vitiligo): topical corticosteroid, PUVA
- Depigmentation therapy (For extensive involvement): Monobenzylether

سنوات (1)

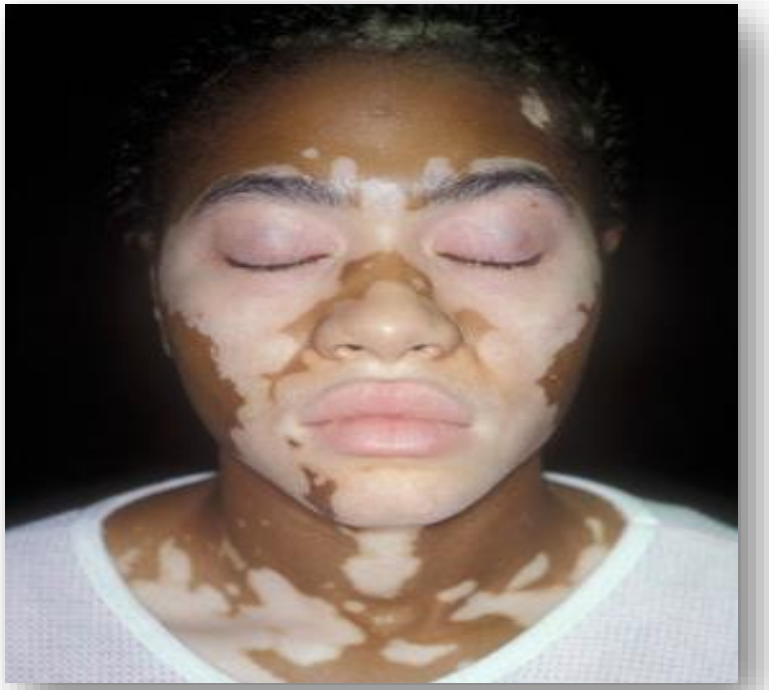
❖What is the topical treatment of vitiligo

- Topical steroids



Vitiligo

- **Describe this lesion**
  - Milky white depigmented patch
- **Mention 3 systemic diseases may be associated with**
  - Diabetes Meletus
  - Hypothyroidism
  - Celiac disease



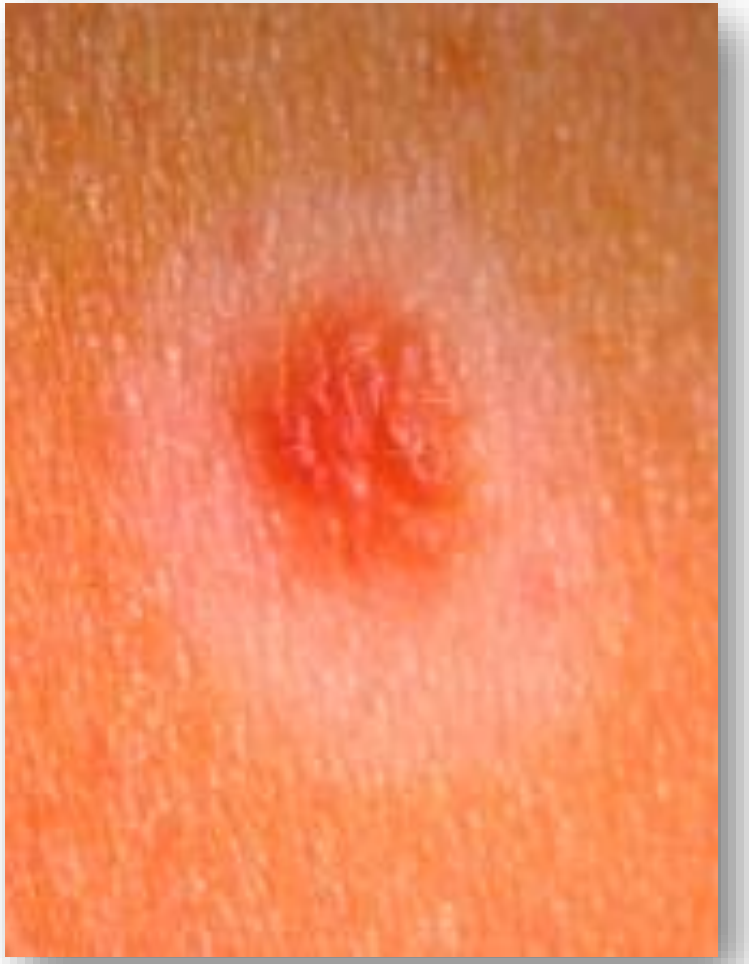
Sutton’s halo naevi

سنوات (2)

- **Define Halo naevus**: An otherwise normal mole with a white ring, or halo, around it.

سنوات (1)

- **Primary lesion of Halo nevus**: mole surrounded by a white ring
- The central dark brown naevus fades from dark brown to light brown to pink, eventually disappearing completely, needs follow up for melanoma.
- Halos can be seen as part of a more generalised pigment loss, vitiligo elsewhere, and halo naevi may also be associated with another autoimmune disease.

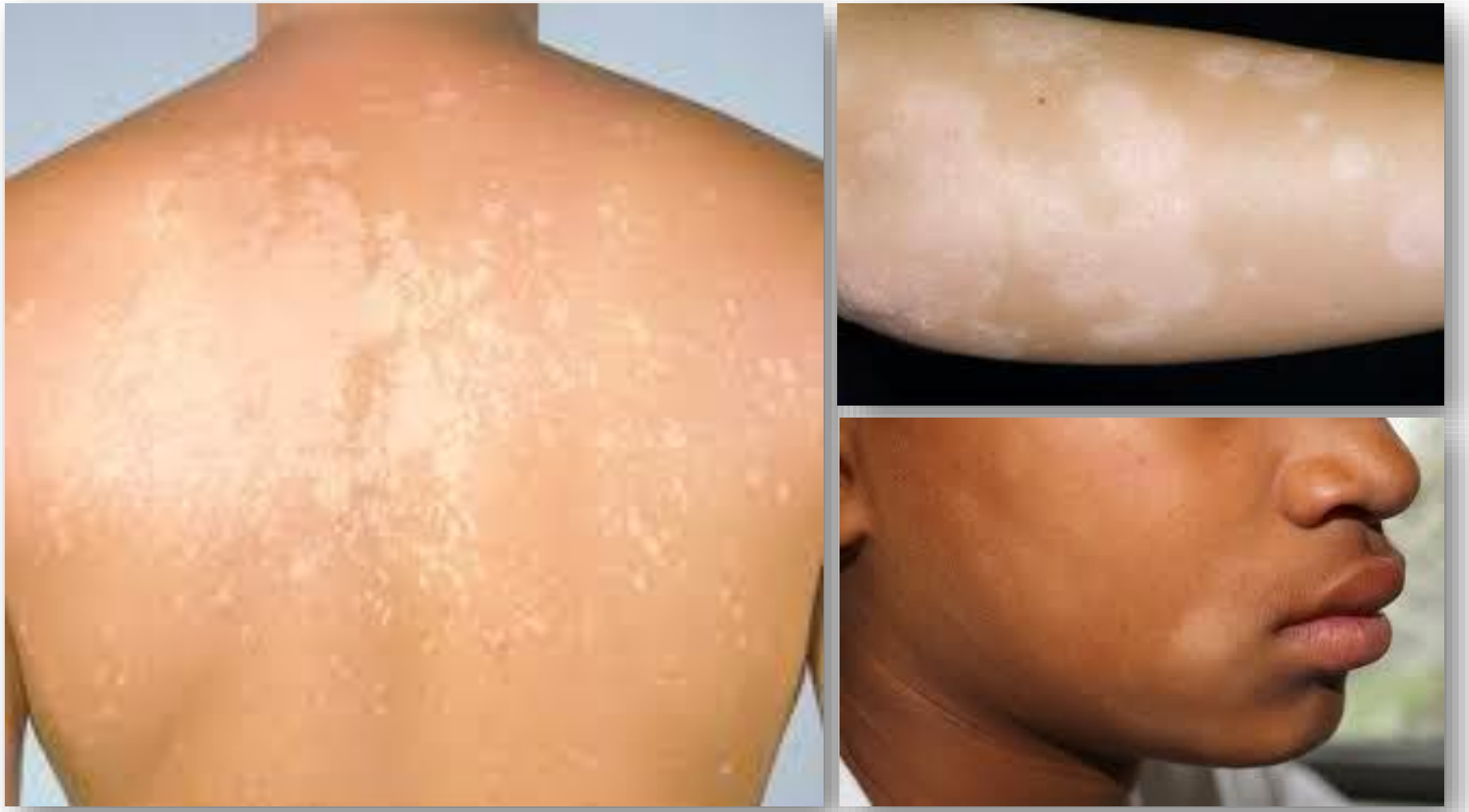


Post inflammatory hypopigmentation

سنوات (1)

1. **Tinea versicolor**: *M.furfur* destroys lipids releasing fatty acid (Azelaic acid) leading to irritation of melanocytes
2. **Psoriasis**: Short Melanocyte transit time, also topical steroids can lead to hypopigmentation
3. **Pityriasis alba**:
   - **Description**: Ill-defined hypopigmented patch, more in dark skin and atopic dermatitis.
   - **Treatment** : Emollient + Avoid sun exposure, steroid if inflammatory type.

Pityriasis alba (النخالية البيضاء)



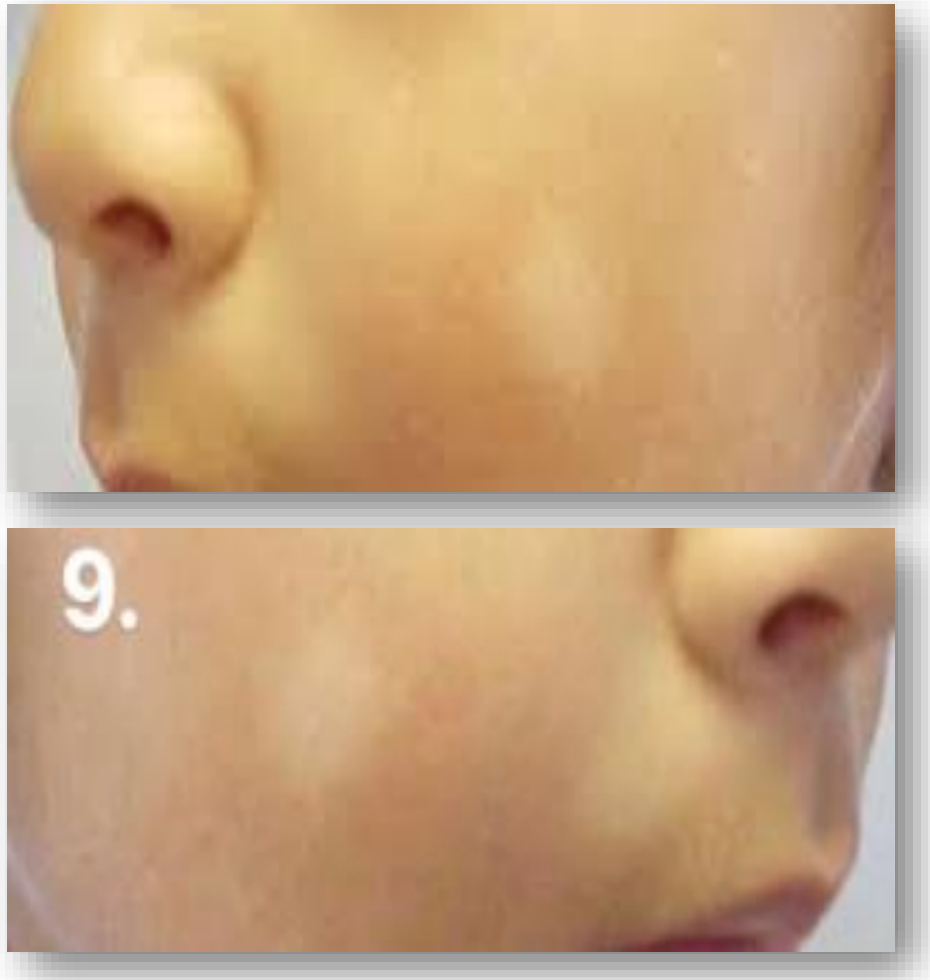
Pityriasis alba

سنوات (3)
- What is your diagnosis
  - Pityriasis alba

سنوات (2)
- Describe what you see:
  - ill-define hypopigmented area on the face

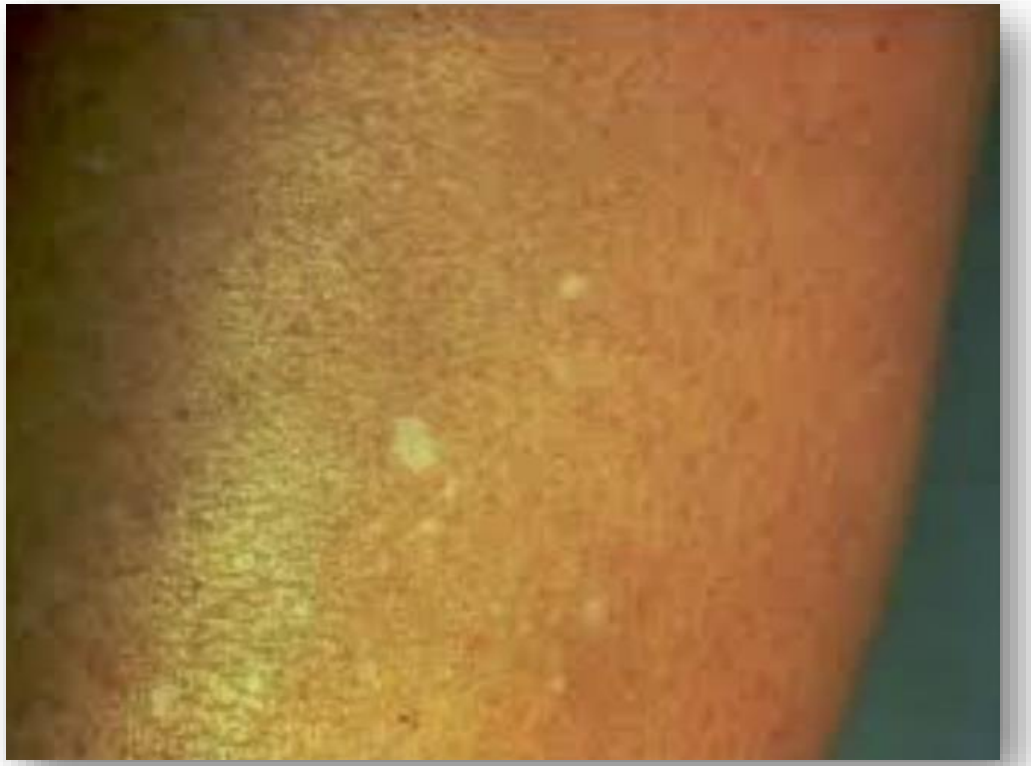
سنوات (2)
- Mention 3 differential diagnosis
  - Pityriasis alba
  - Vitiligo
  - Tinea versicolor

سنوات (1)
- What bedside test you would to do for this patient ?
  - Woods light examination



Depigmented macule

- **Differential diagnosis**:
  - Vitiligo (Koebner's phenomenon)
  - Nervous depigmentosa (since birth, globular pattern on dermoscope)
  - Idiopathic guttate hypopigmentation



Hair & its disorders

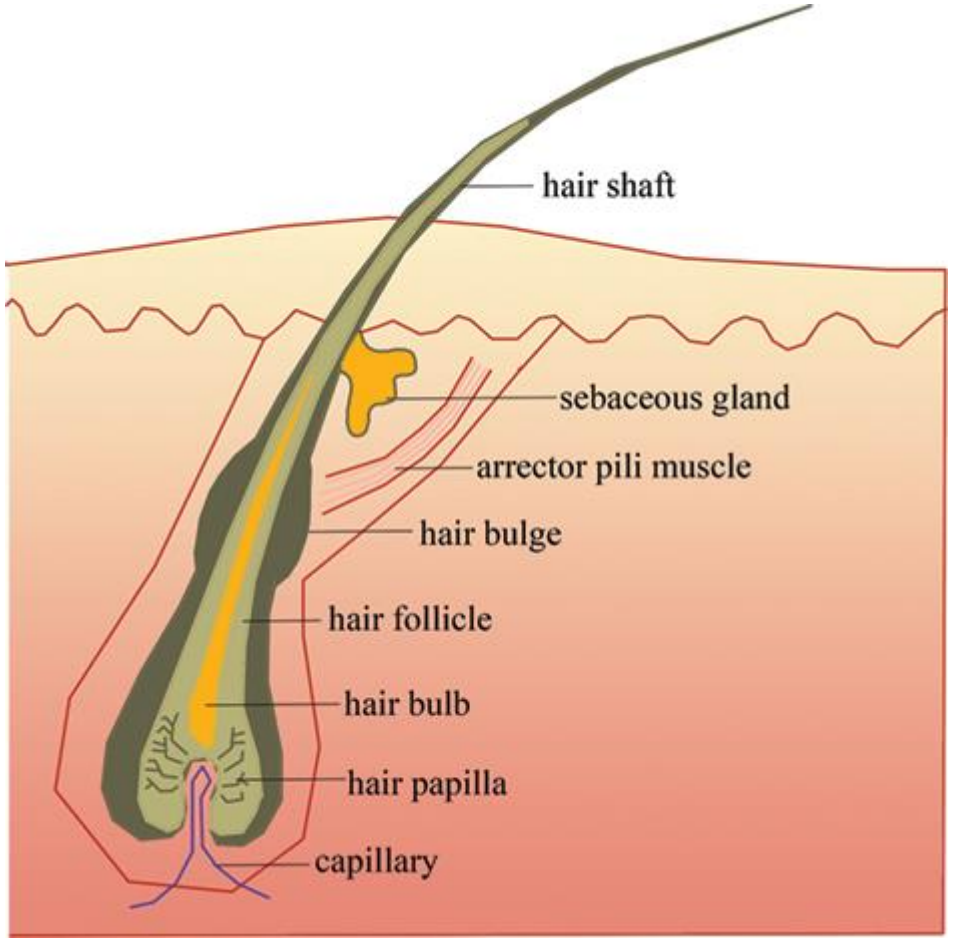
Anatomy of the hair

❖Hair is composed of:

1. **Shaft (Exposed part above the skin):** Composed of Medulla, Cortex and Cuticle
2. **Hair follicle (Under the skin):** Composed of Hair bulb (Dermal papilla and the Matrix) and the root Sheath

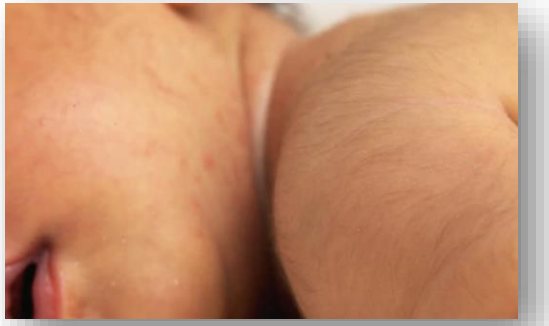
❖Accessory structures of Hair:

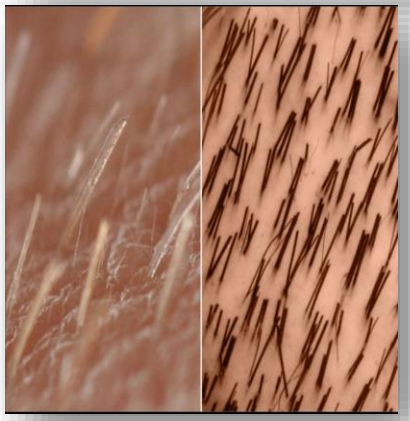
1. Arrector pilli muscle.
2. Sebaceous glands.
3. Hair root plexus.



Types of hair

- **Lanugo hair**:
  - Very thin, soft, usually unpigmented and long hair, produced by fetal hair cells and is usually shed before birth.
- **Vellus hair**:
  - Short, thin, light colored and barely noticeable hair that develops on most of a person's body childhood sparing the palms and soles.
- **Terminal hair**:
  - Thick, long and dark, it is limited to the eyebrows, eyelashes and scalp until puberty.
  - During puberty, the increase in androgenic hormone levels causes vellus hair to be replaced with terminal hair in certain parts of the human body, also secondary terminal hair develops in the axillae, pubic region and central chest in men in response to androgens.





Hair cycle

إجابة الأرشيف للسؤال الأحمر
فقط وان شاء الله كافية

❖ **Write the hair cycle and the definition of each one**

- **Anagen (growing phase)**:
  - The active growth phase, which typically lasts 1000 days depending on predetermined genetic factors, it determines the length of our hair
- **Catagen (transition phase)**:
  - The short growth arrest phase, of approximately 10 days; due to cessation of protein and pigment production and regression of the follicle due to detachment from the dermal papilla
- **Telogen (resting phase)**:
  - The resting phase, lasting approximately 100 days irrespective of location
  - Whilst the old hair is resting, a new hair begins the growth phase
- **Exogen (new hair phase)**:
  - This is part of the resting phase where the old hair sheds and a new hair continues to grow

سنوات (1)

إضافي

Hair cycle

سنوات (1)

❖Active phase of hair growth is **anagen**

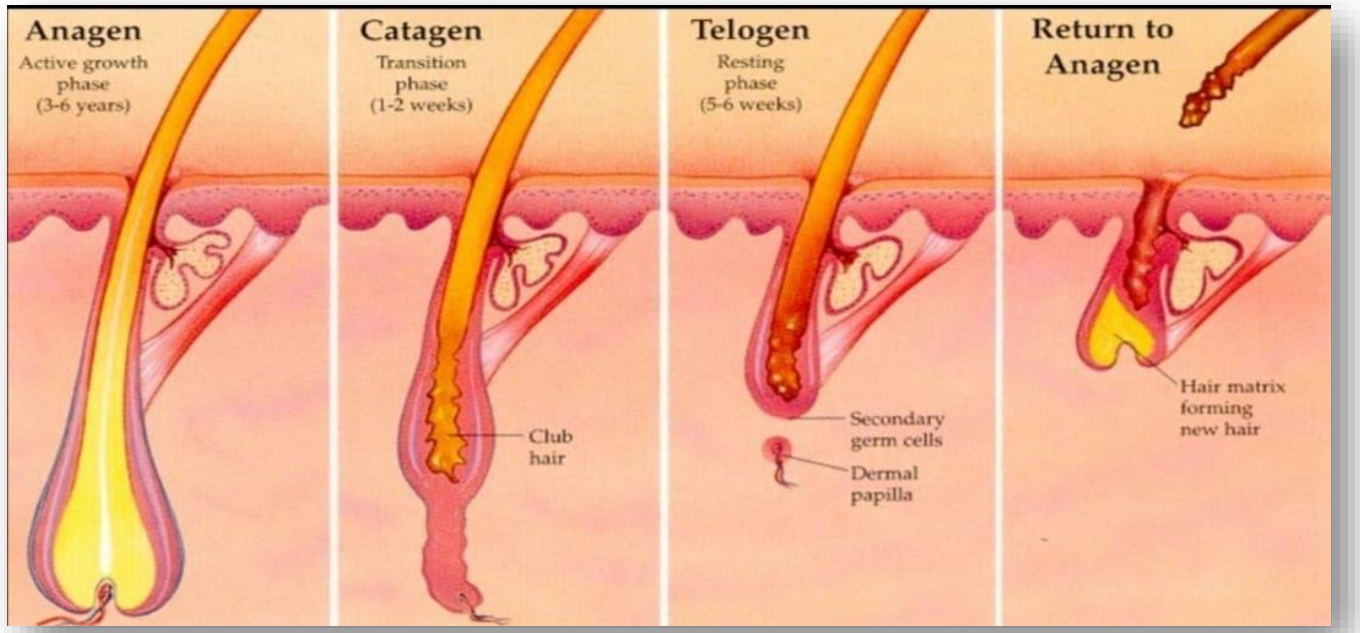
Hair growth rate 1 cm / month

سنوات (2)

❖The transition state of hair is **catagen**

❖The resting state of hair is **telogen**

❖The new hair phase of hair is **exogen**



Hair loss

Hair loss (Alopecia)

Localized (Patchy)

سنوات (3)

- Non-scarring
  - Tinea capitis
  - Alopecia areata
  - Androgenetic alopecia
  - Traumatic (trichotillomania, traction, cosmetic)
  - Syphilis
- Scarring
  - Idiopathic
  - Developmental defects
  - Discoid lupus erythematosus
  - Herpes zoster
  - Pseudopelade
  - Kerion

Diffuse

سنوات (1)

1. Androgenetic alopecia
2. Telogen effluvium
3. Metabolic
4. Hypothyroidism
5. Hyperthyroidism
6. Hypopituitarism
7. Diabetes mellitus
8. HIV disease
9. Nutritional deficiency
10. Liver disease
11. Post-partum
12. Alopecia areata
13. Syphilis
14. Discoid lupus erythematosus
15. Radiotherapy
16. Folliculitis decalvans
17. Lichen planus pilaris

Hair loss (Alopecia)

سنوات (1) ❖ Mention 3 cause if **patchy** hair loss

سنوات (1) ❖ Mention 3 cause if **patchy** hair loss **without scarring**

إضافي ❖ Mention 3 cause if **patchy** hair loss **with scarring**

سنوات (1) ❖ Mention 3 DDx of **diffuse** hair loss in **female** patient

- Any of the 17 points

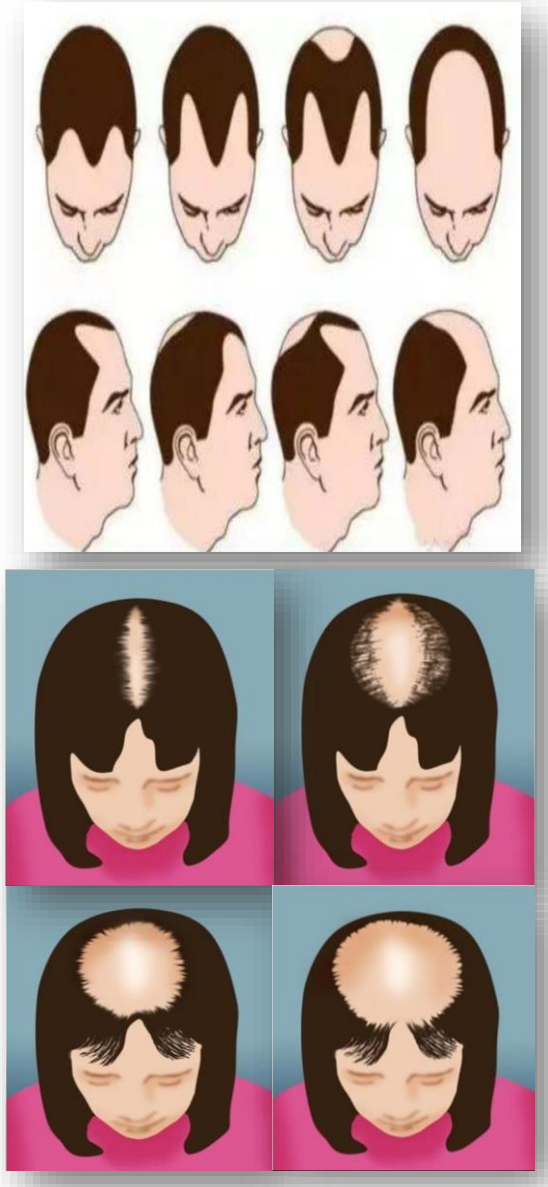
إضافي ❖ Mention 3 DDx of **diffuse** hair loss in **male** patient

- Any of the 17 points except postpartum

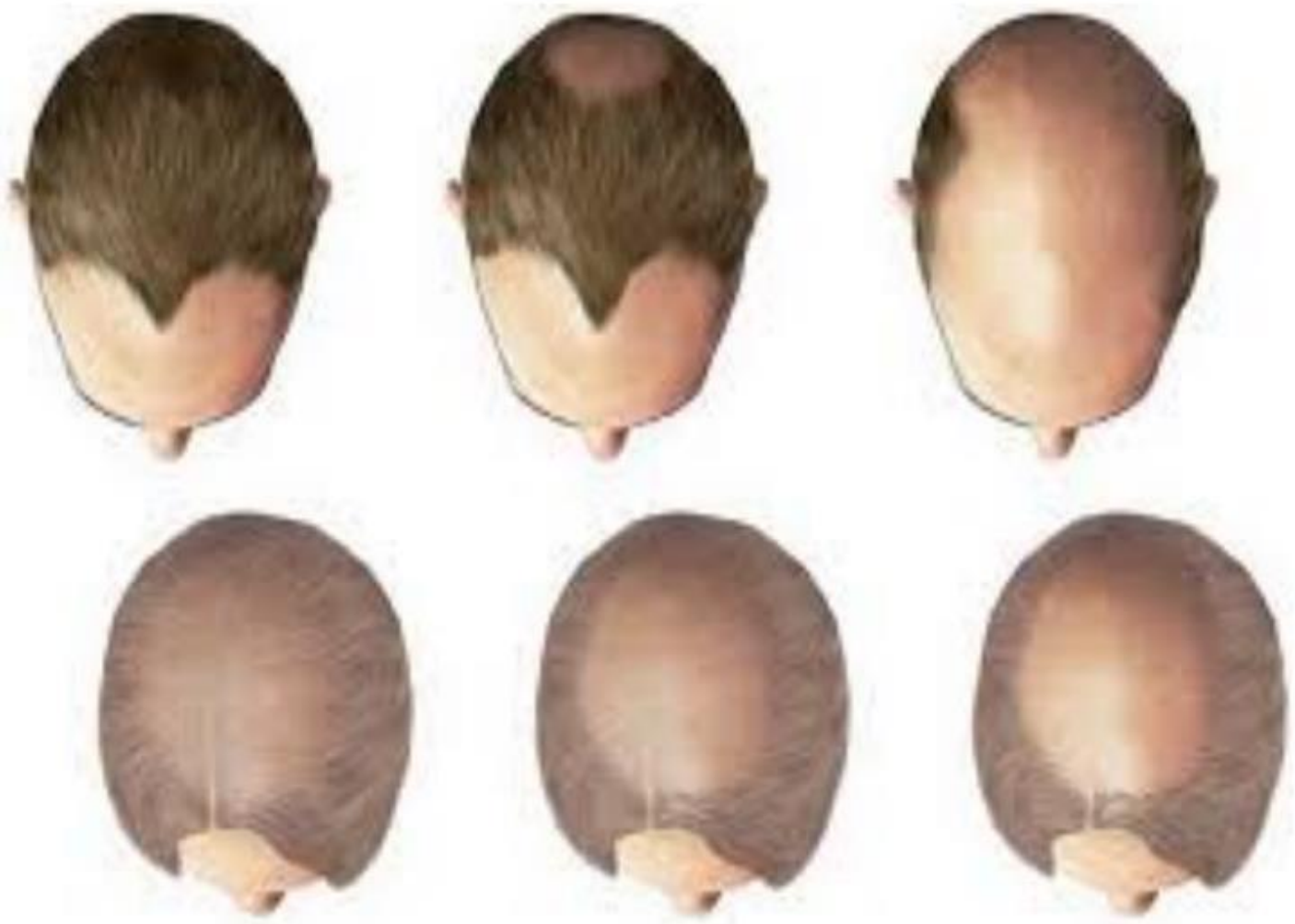
شرح

Androgenetic alopecia

- Androgen dependent, non-scaring
- The most common cause of hair loss in both males and females
- It affects both men and women in a different specific pattern of hair loss of each sex
- **Treatment male pattern**
  - Minoxidil lotion or cream (first line treatment) (سنوات (3))
  - Oral Finasteride
- **Treatment female pattern**
  - Minoxidil
  - Anti-androgens such as spironolactone
- Hair transplant when medical therapies fail



Androgenetic alopecia



Male pattern
Female pattern
Androgenetic alopecia
difference in the
frontal part

سنوات (2)

Androgenic alopecia

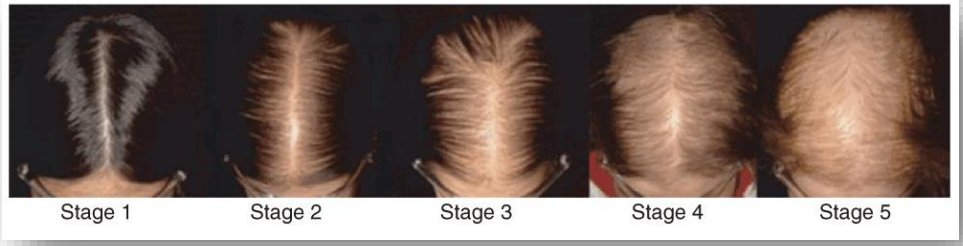
- **In what disease do we see this pattern of hair loss?**
  - Androgenic alopecia

سنوات (3)

- **First line treatment in male pattern**
  - Minoxidil lotion

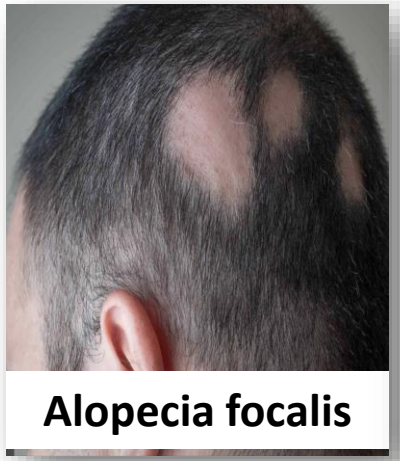
سنوات (1)

- **What is the pattern of androgenetic alopecia in female ?**
  - Thinning over the central scalp, usually preservation of the frontal margin

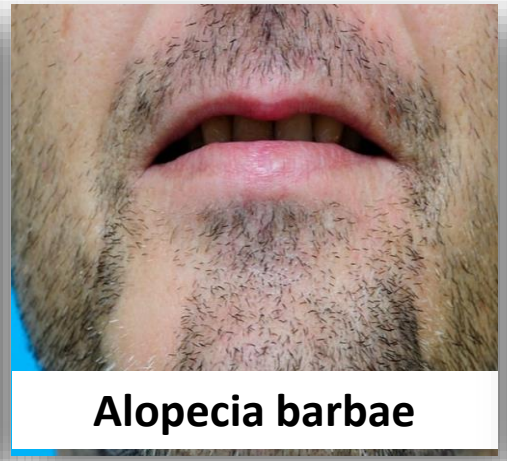


Alopecia areata (الثعلبة)

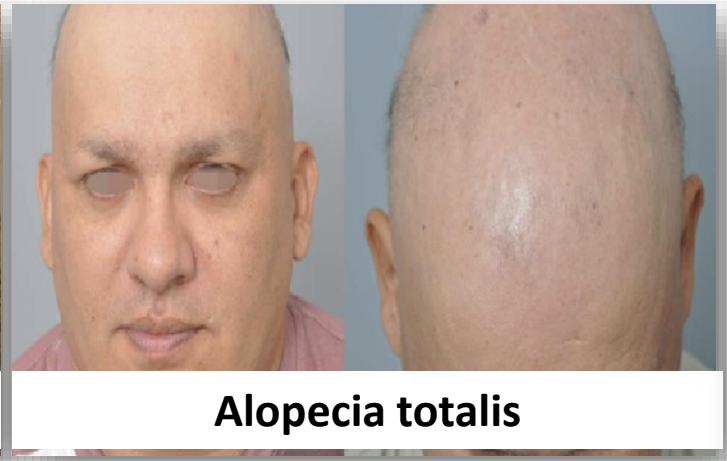
- ❖It is an organ-specific auto-immune disease affecting the hair follicles. Type of non-scarring alopecia
- ❖Typically presents with smooth round or oval patches of no-scaring hair loss on the scalp without itching, erythema or scaling
- ❖Extensive involvement may lead to total scalp hair loss (alopecia totalis), total body hair loss (alopecia universalis) or localized hair loss along the scalp margin (Ophiasis)



Alopecia focalis



Alopecia barbae



Alopecia totalis

Alopecia universalis

Ophiasis

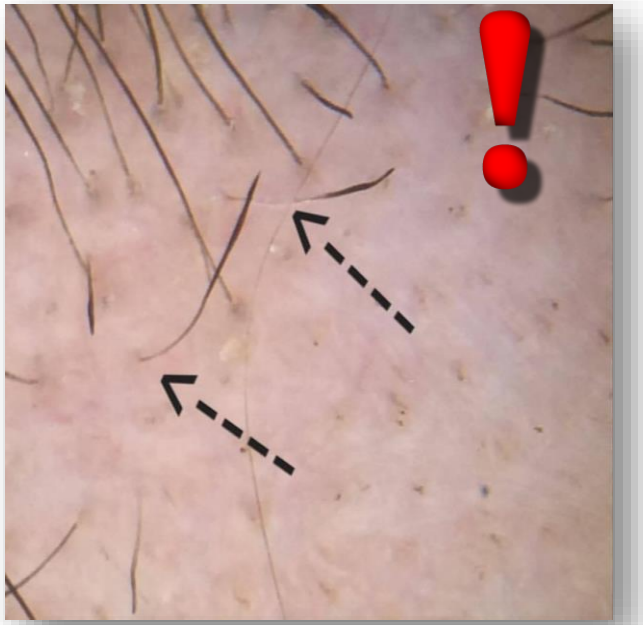
Alopecia areata

سنوات (1)

- **Exclamation mark** hairs indicate that the process is active, thus when present, are diagnostic of alopecia areata

سنوات (4)

- **Define exclamation mark**: short hairs that taper towards the base, indicate active growth diagnostic for alopecia areata
- Nail abnormalities, predominantly pitting or roughening, may occur in association with this condition
- Could be associated with Atopic dermatitis, Vitiligo, Thyroid disease, Collagen vascular diseases, Celiac disease, Diabetes mellitus, Down syndrome, Anxiety, Depression and stressful life events in the 6 months before onset.
- Investigation of associated diseases is usually indicated if symptomatic



Alopecia areata

- The age of onset is usually in the first two decades. The course of AA is difficult to predict

- **Poor prognostic markers include:**
  - Childhood onset of disease.
  - Atopy.
  - Ophiasis (band of alopecia in occipital region).
  - Nail dystrophy.
  - Family history of other autoimmune disorders.
  - Presence of autoantibodies.

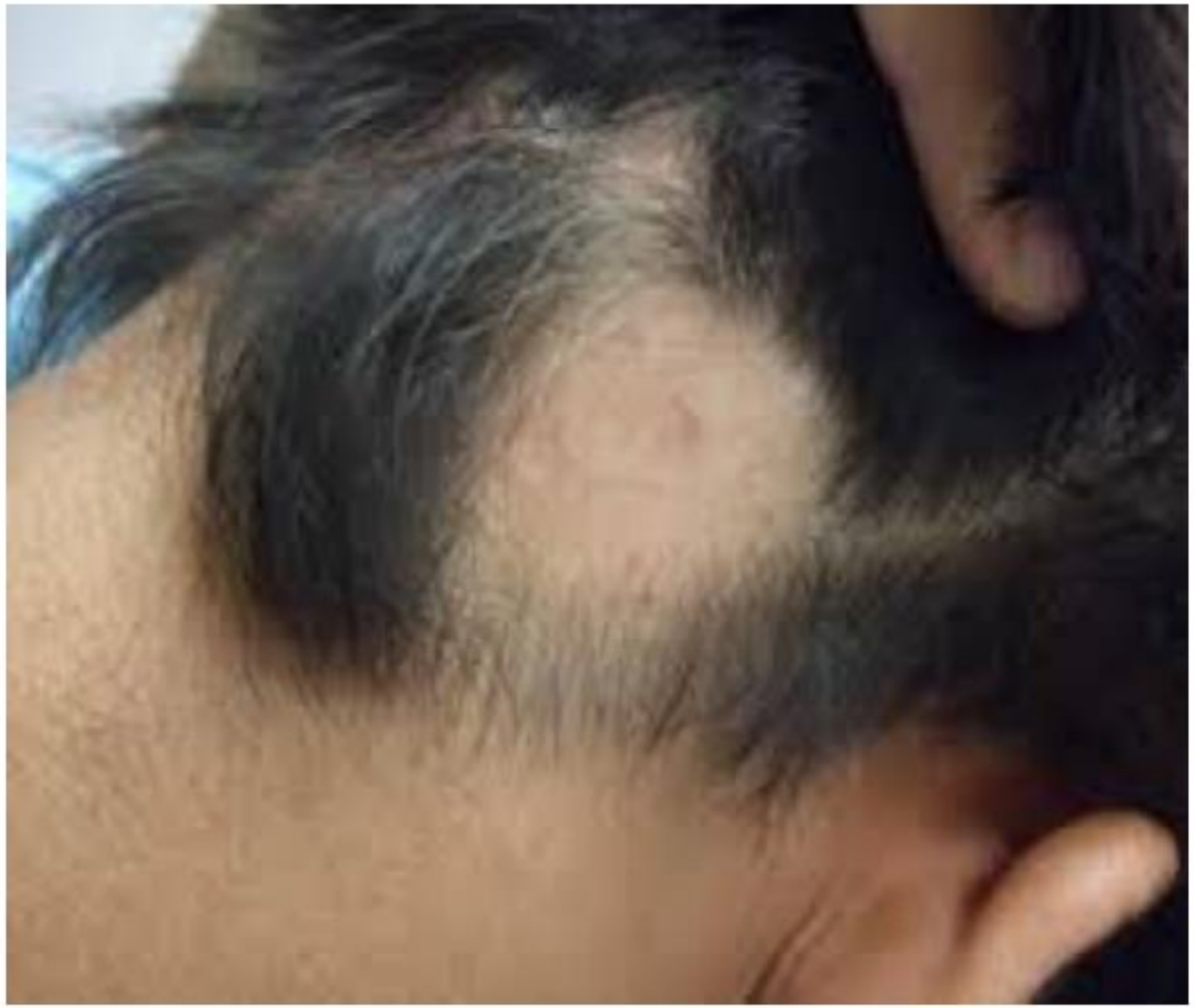
سنوات (1)

- **Differential diagnosis**
  - Trichotillomania, Traction alopecia, Telogen effluvium, androgenetic alopecia and Tinea capitis

Alopecia areata

- In alopecia areata, the hair follicle is not injured and maintains the potential to regrow hair. Indeed, the majority of isolated patches spontaneous regrow if given enough time
- There is no cure for alopecia areata
- **Management**:
  - **Potent topical steroids** (Clobetasol propionate 0.05%): used on the scalp for 2-3months on localized patches of alopecia.
  - **Topical Calcineurin inhibitors** as an alternative of for long term use of the potent topical steroids.
  - **Intralesionsal corticosteroids** (Triamcinolone diluted with local anesthetic)
  - **Systemic steroids**: for more extensive loss when Intralesionsal steroids cannot manage
  - **PUVA therapy**: not recommended (high relapse rate and the risk of cancer)
  - **Contact sensitization** using either irritants (dithranol or retinoids) or allergens (diphencyprone).
  - **Topical Minoxidil** (also used in combination with corticosteroids).

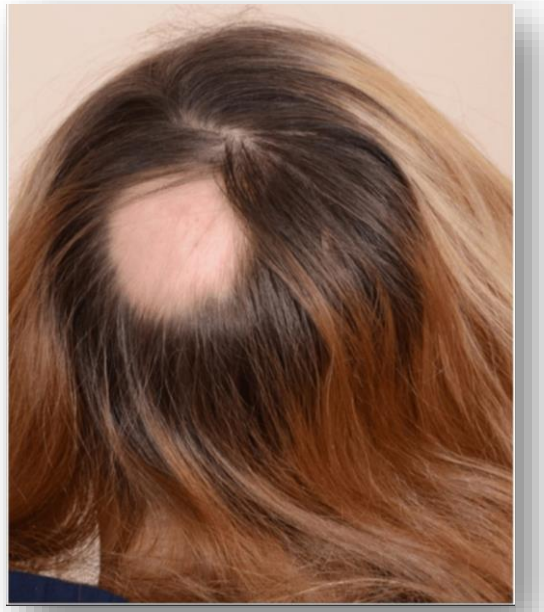
Alopecia areata

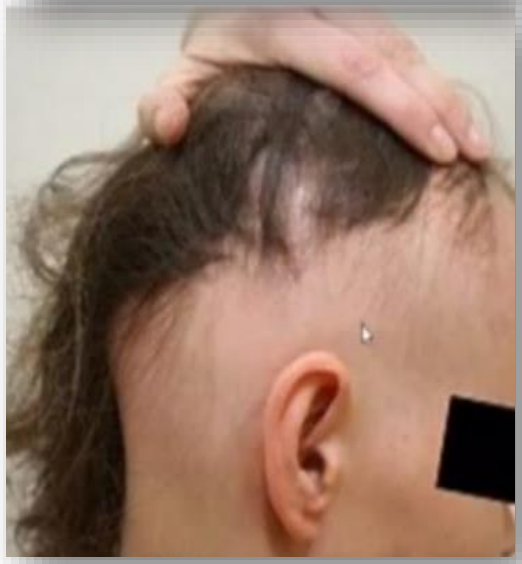


Alopecia areata
Dx by dermoscope
Tests: CBC (pernicious anemia), thyroid function (Hashimoto) , ANA, HbA1c , anti-TPO , celiac

10Y/O female with smooth round patch of hair loss on the scalp

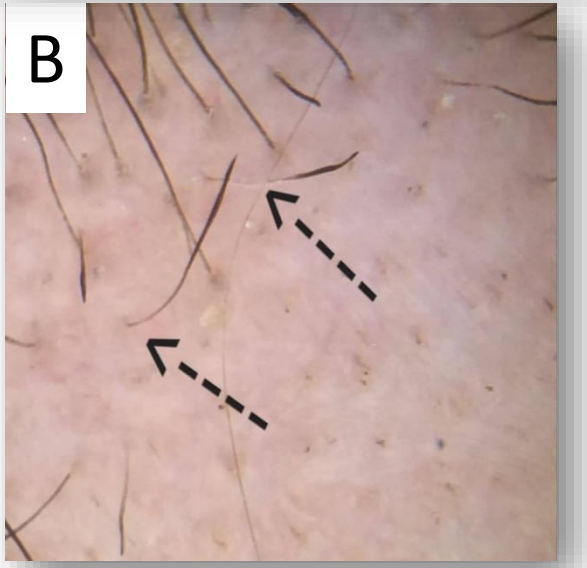
- **Describe**
  - Round patch of hair loss
- **Mention 2 differential diagnosis**
  - Alopecia areata
  - T. Capitis
  - trichotillomania
- **Mention 1 topical treatment**
  - Topical and intralesional steroids
  - Topical Calcineurin inhibitors
  - Topical Minoxidil
- **First line treatment in adult**
  - Intralesional topical steroid





17Y/O male with smooth oval patches of hair loss on the scalp

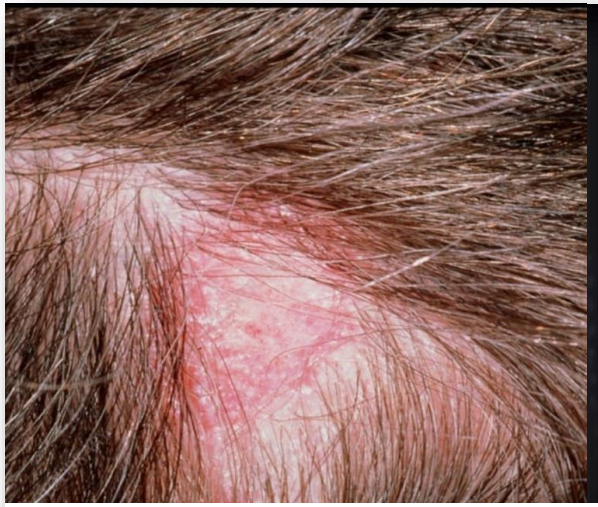
- **Describe what do you see in A**
  - Round patch of hair loss
- **What is the name of the sign seen in B, what does it indicate ?**
  - Exclamation mark, indicates active alopecia areata



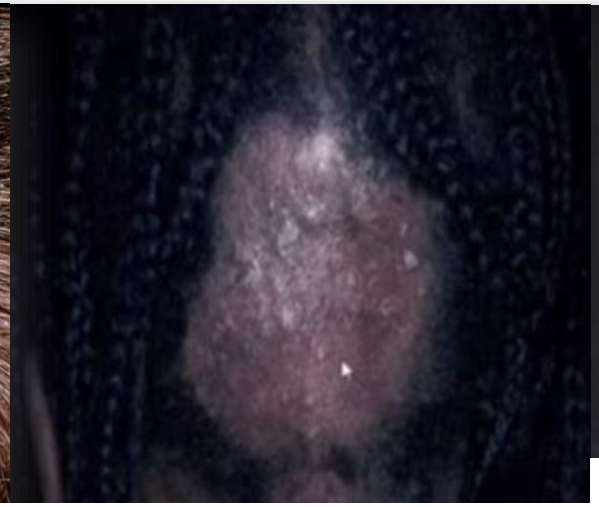
- **What is a possible diagnosis**
  - Case: 17-years old male with smooth round or oval patches of non-scarring hair loss on the scalp ⟶ Alopecia areata

For more details see fungal infection section

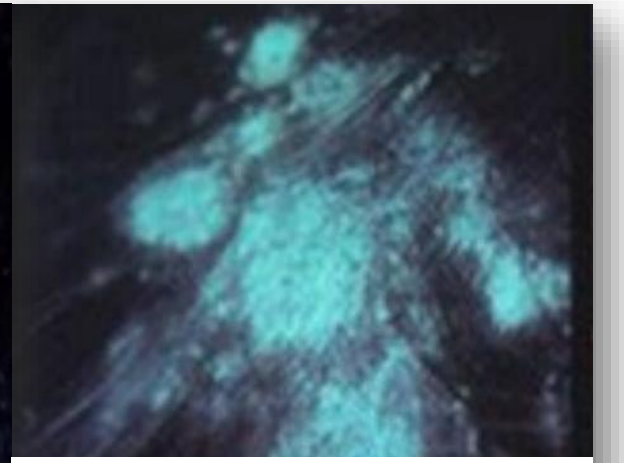
Tinea capitis



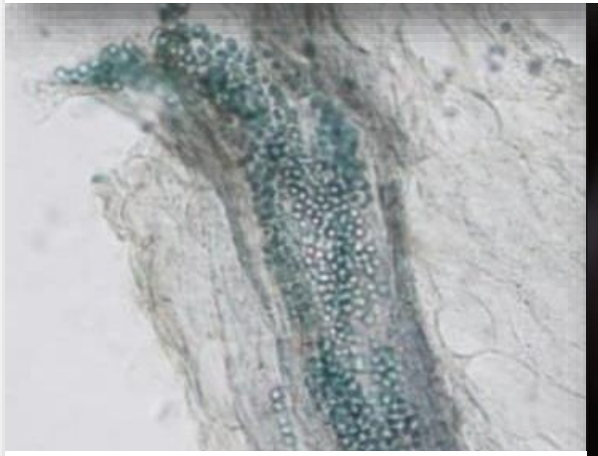
Tinea capitis



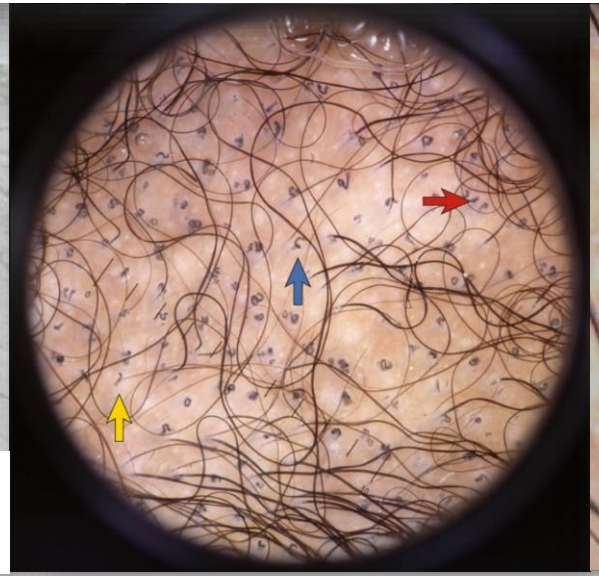
Kerion

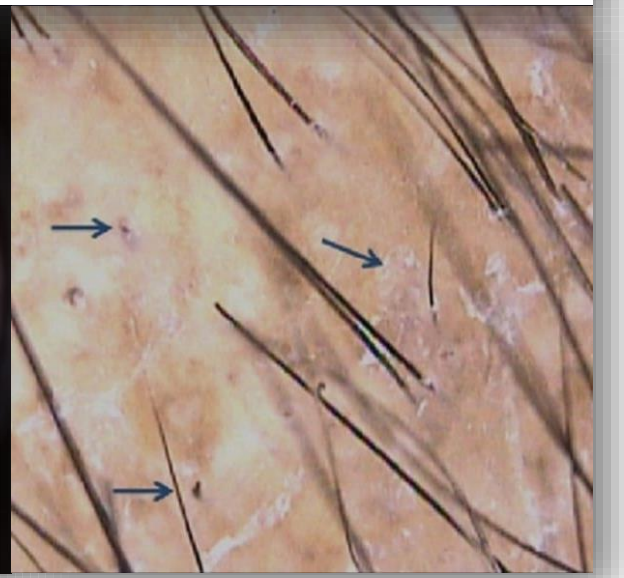


Green appearance on Wood's lamb

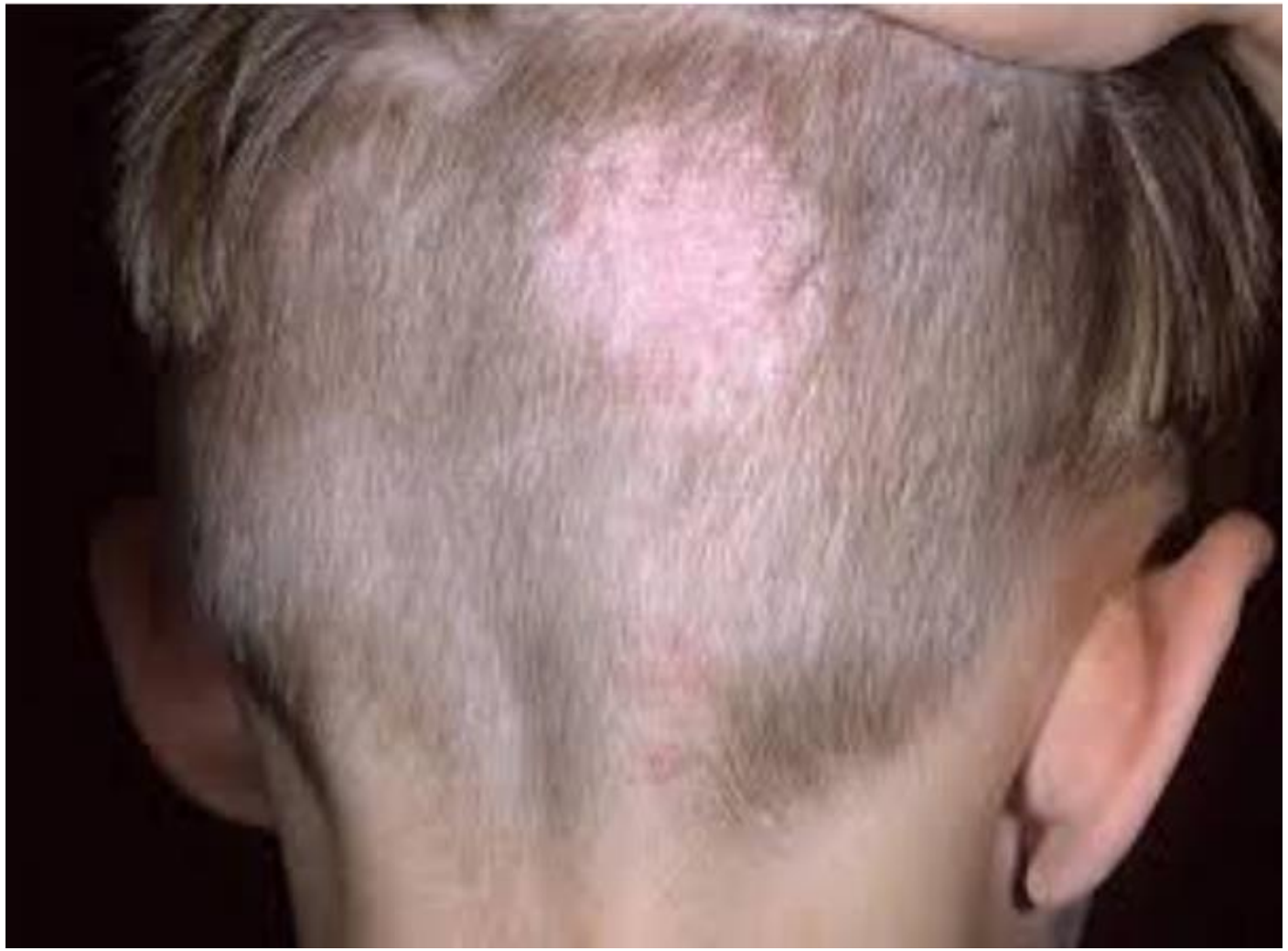


KOH preparation showing the fungal hyphae

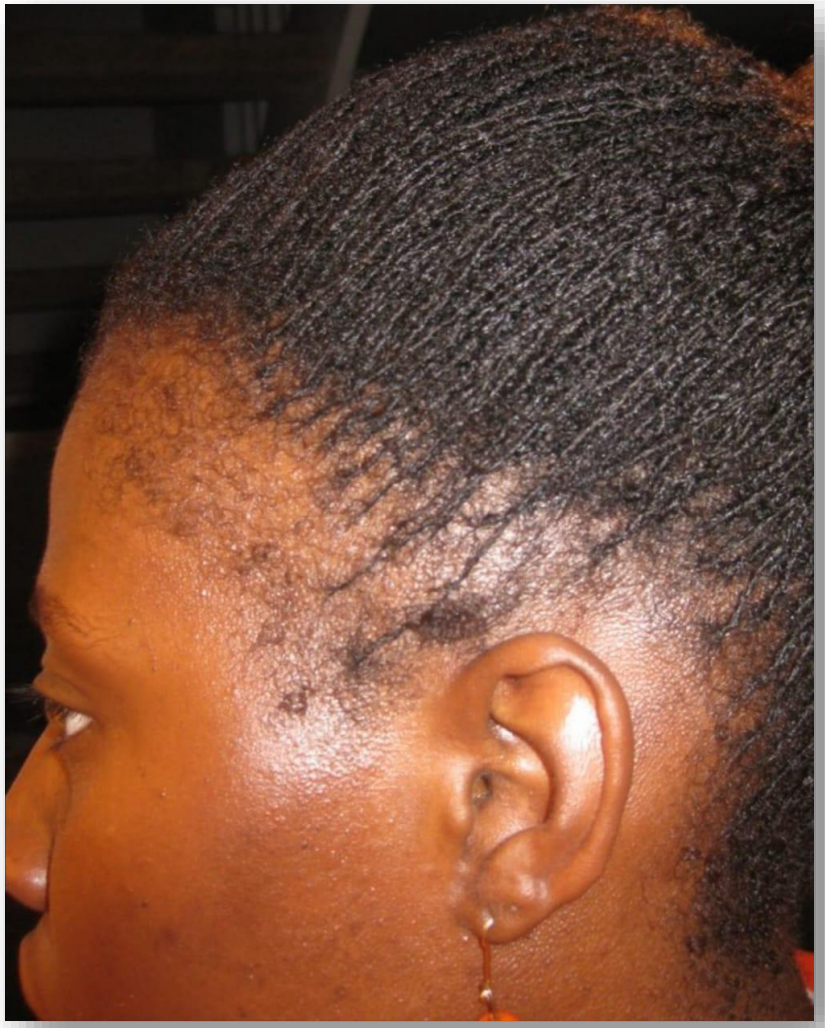


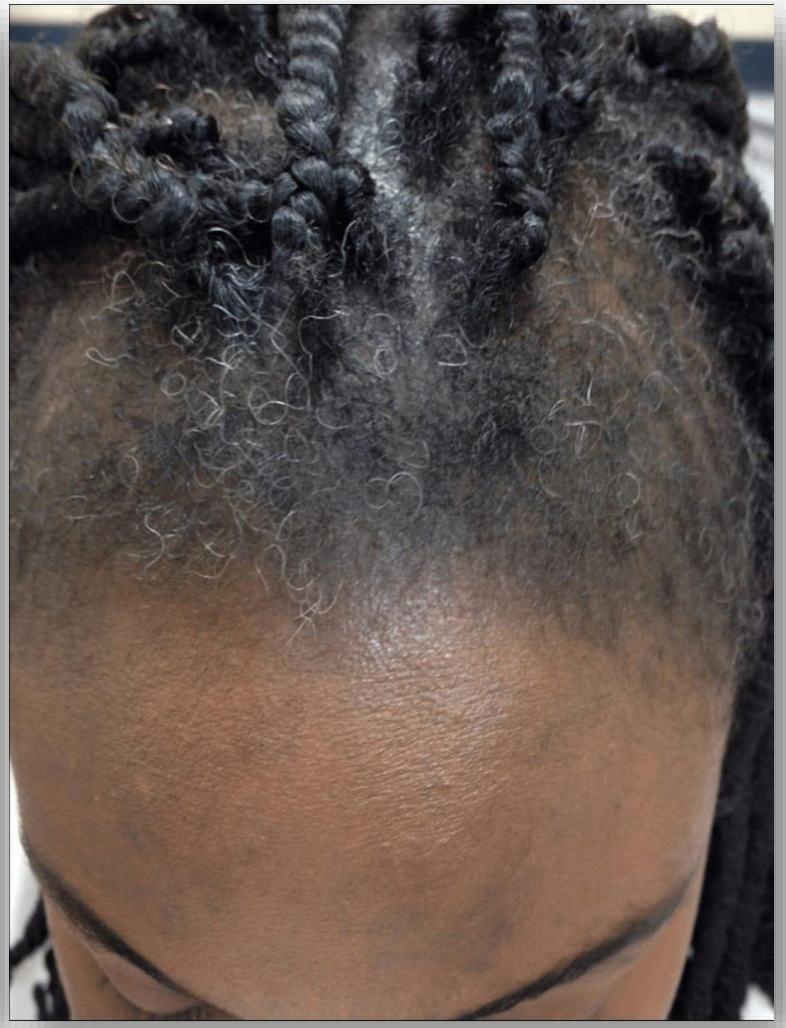


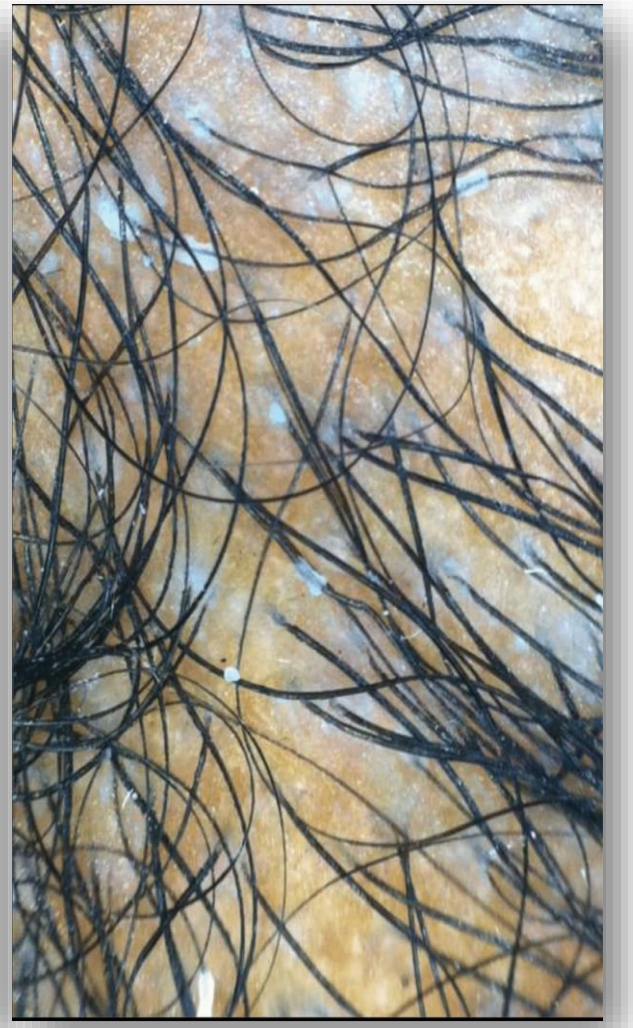
Tinea capitis



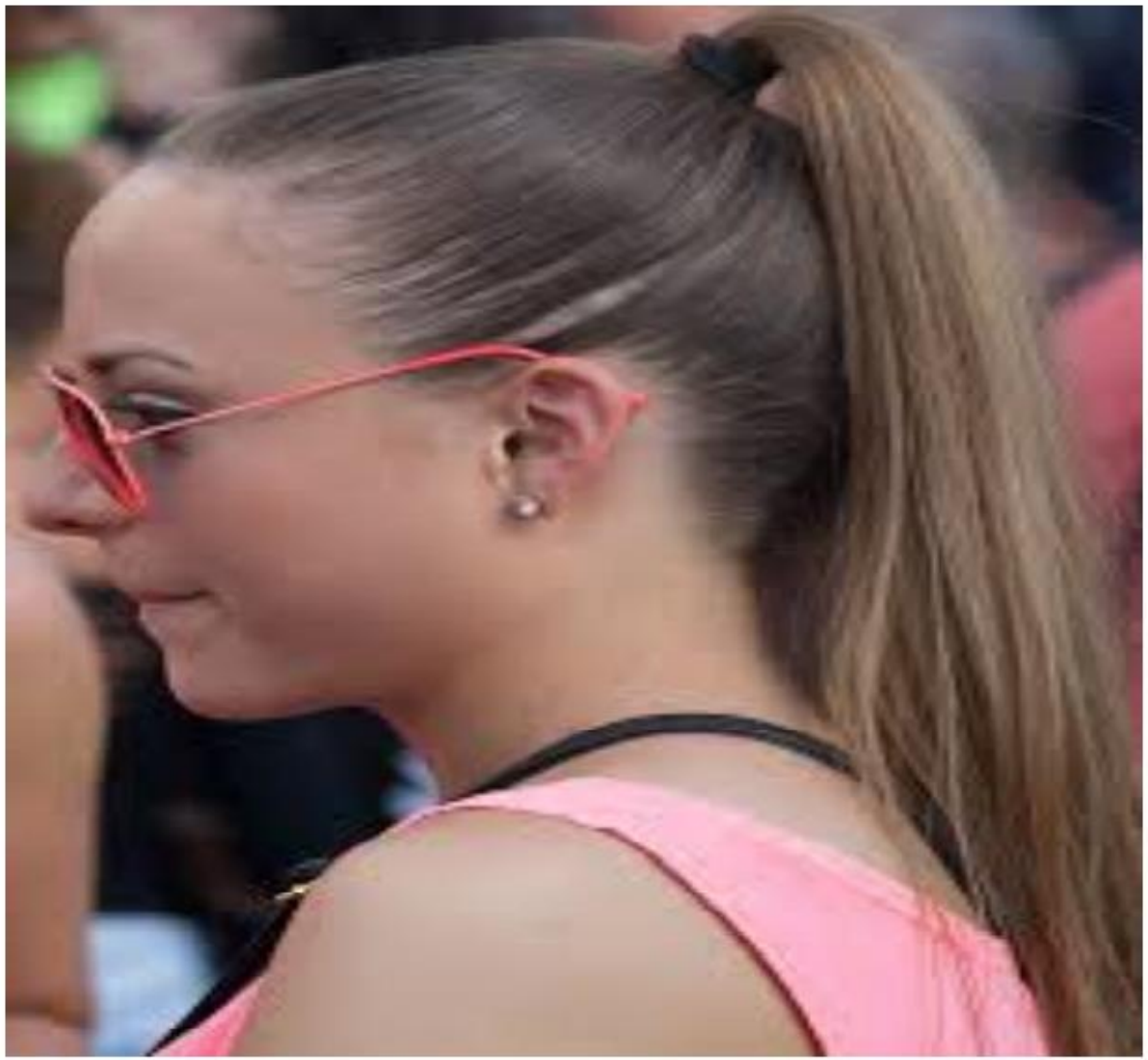
Traction alopecia/traumatic







Traction alopecia

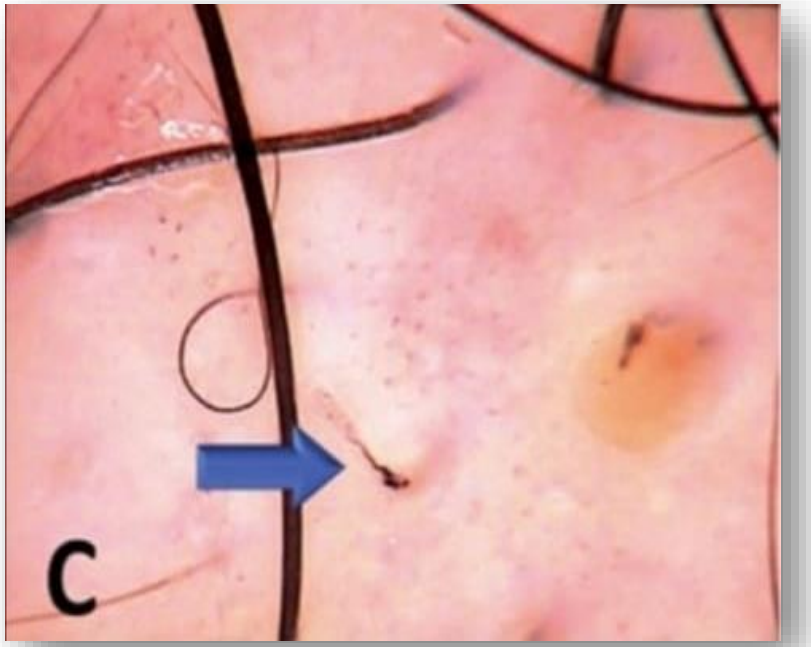


Traction
Fibrosed hair follicles

شرح

Trichotillomania

- In trichotillomania, hair is pulled, twisted or rubbed out, and affected site(s) are covered in broken hairs of different lengths
- There may be psychological factors



سنوات (1)

- 5 years old child came with hair loss due to trichotillomania mention 2 ddx.
  - Alopecia areata
  - Traction alopecia.

Trichotillomania

Trichotillomania
Dx by dermoscope
If large area consult GI on trichobezoar

شرح

Telogen effluvium

سنوات (1)

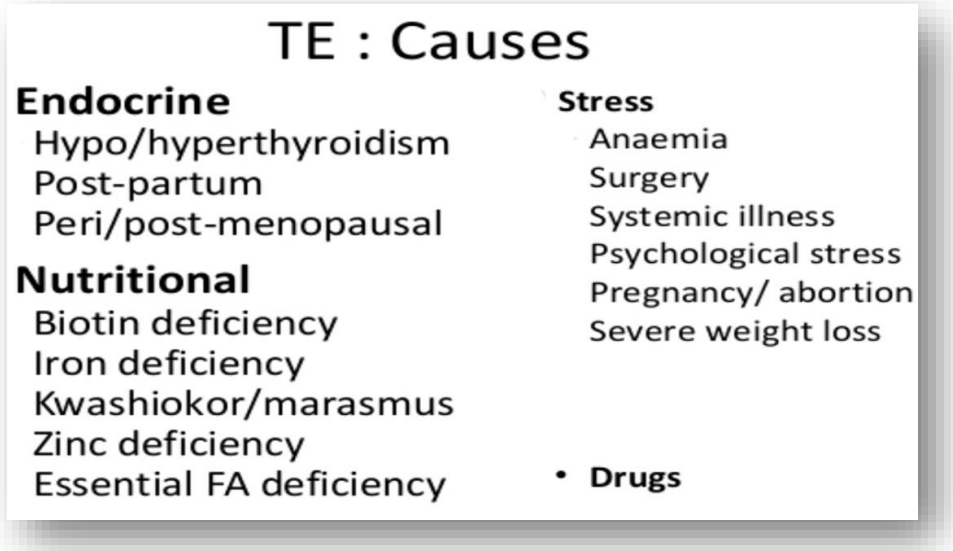
❖Define telogen effluvium:

- following a number of stimuli the majority of hair follicles may enter the resting phase (telogen) at the same time (synchronously) resulting in diffuse shedding approximately 2 months after the triggering event, often described as the hair ‘falling out by the roots. This is usually an acute self-limiting phenomenon, usually resolving within 6 months

سنوات (1)

❖Mention 3 causes of telogen effluvium

1. Childbirth
2. severe trauma or illness
3. marked weight loss
4. major operations



Acute telogen effluvium

Acute telogen effluvium :
due to stress which is sudden or prevents the person from eating, weight loss, diet , surgeries, general anesthesia, fever, acute medical illnesses, shock
Chronic is due to vit deficiencies or chronic medical illnesses
Anogen effluvium is due to chemotherapy

Symptoms & signs of Telogen Effluvium

Noticeable hair loss during washing or combing hair

Hair loss that is temporary

A generalised hair thinning

Losing hair that has white bulb & lack of shiny sheath

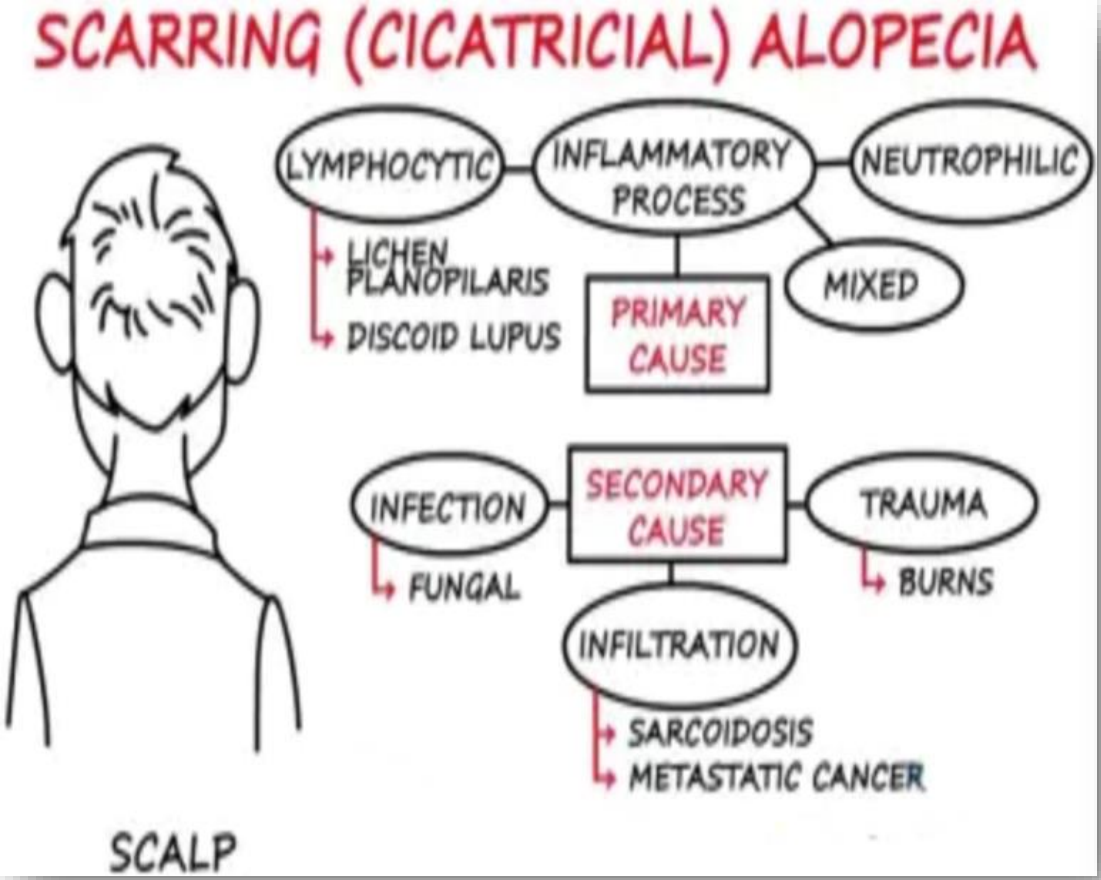
Dry, lusterless hair that are sparse & easily pluckable

Hair colour changes from dark to brown or red, brown to blond

What is the diagnosis ?

A. Telogen effluvium
B. Alopecia areata
C. Traction alopecia
D. Androgenetic alopecia
E. Trichotillomania

Scarring alopecia



Primary
Lichen planopilaris
Discoid lupus erythematosus
Pseudopelade of Brocq
Central centrifugal cicatricial alopecia and traction
Dissecting folliculitis
Folliculitis decalvans
Secondary
Post-traumatic
Burns
Radiotherapy
Neoplasia (e.g. squamous cell carcinoma, lymphoma, and sarcoma)
Infection
Bacterial (e.g. folliculitis, acne keloidalis and syphilis)
Viral (e.g. herpes zoster)
Fungal (e.g. with kerion formation)

Excessive hair

شرح

Two patterns of hair overgrowth are recognized

سنوات (1)

Define

❖**Hirsutism**: excessive growth of hair in a female, which is distributed in a male secondary sexual pattern (androgen-sensitive areas)

- **Treatment**:
  - Treat underlying cause
  - Treatments include suppression of androgens, peripheral androgen blockade and mechanical or cosmetic treatment (FDA approved drug: **eflornithine**) سنوات (1)

سنوات (1)

Define

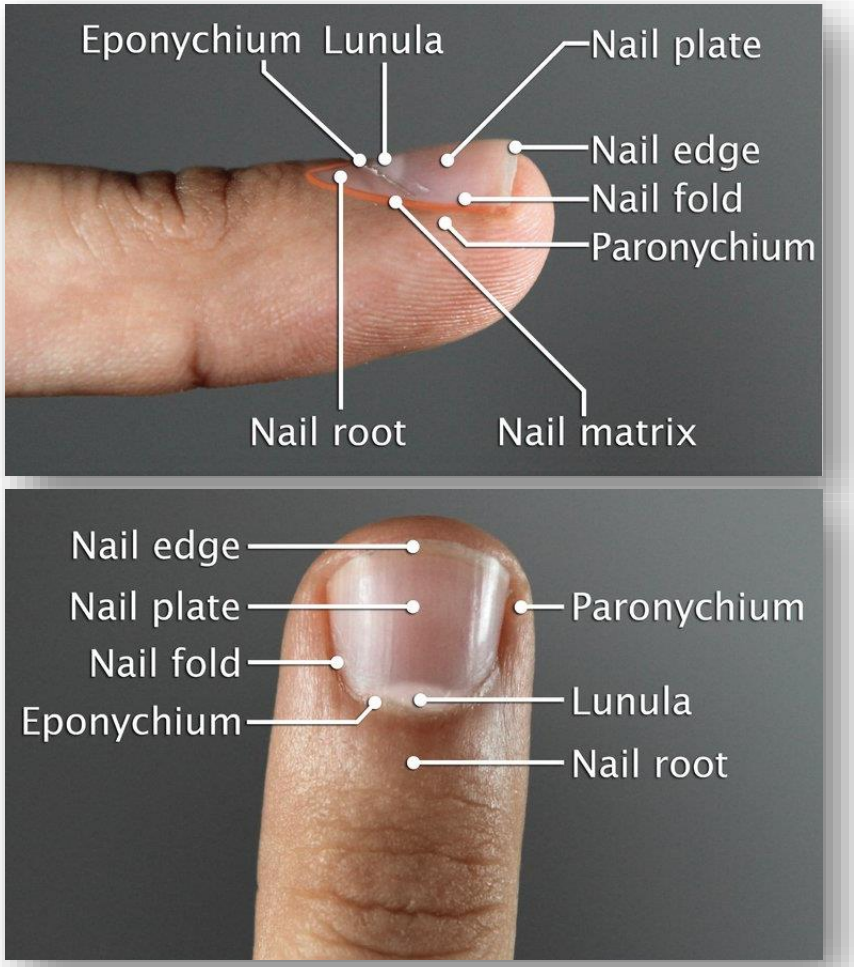
❖**Hypertrichosis**: excessive hair growth in a nonsexual distribution may occur in both sexes

- Causes may be **congenital** or **acquired**; important systemic diseases associated with hypertrichosis include hyperthyroidism, porphyria and anorexia nervosa, could be caused **by drugs**
- Treatment is directed at the underlying cause and stopping any implicated drug, where possible.

Nails & their disorders

Nail anatomy

- **Perionychium**: epidermal tissue surrounding the root and base of the nail
- **Eponychium**: proximal layer of epidermis extending over the nail base
- **Hyponychium**: epidermal tissue immediately underlying the free distal edge of the nail
- **Nail plate (nail body)**
  - Covers the nail bed
  - **Proximally**: consists of the matrix unguis or onychostroma (responsible for new nail growth) and the lunula (the white, crescent-shaped, poorly vascularized portion of the nail)
  - **Distally**: sterile matrix (provides the nail with bulk and strength)
- **Nail fold**: depression proximal to the nail plate from which the nail grows



Findings

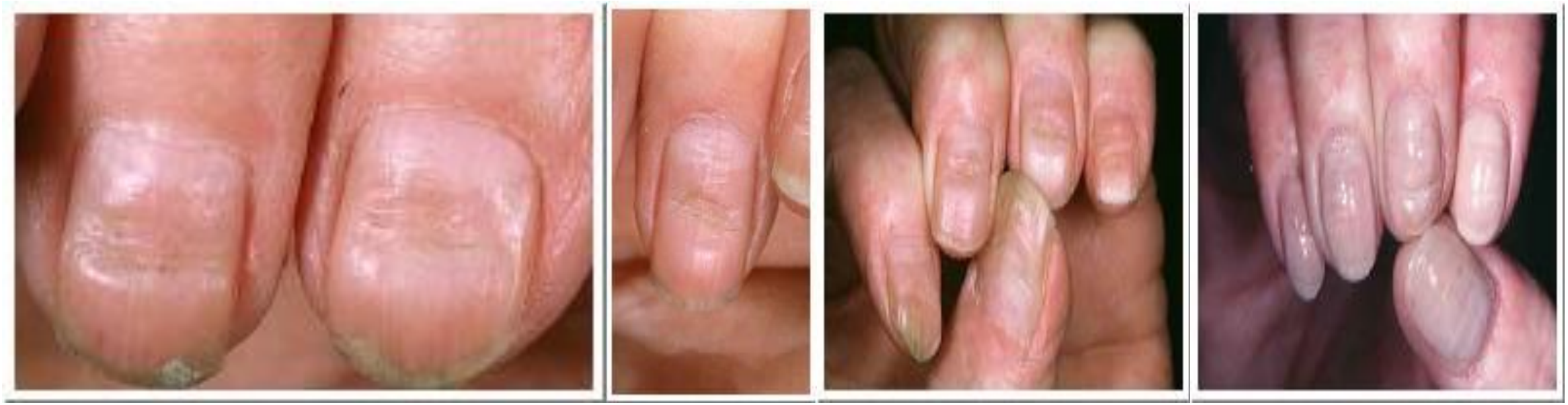
شرح

1. Beau’s lines

سنوات (2)

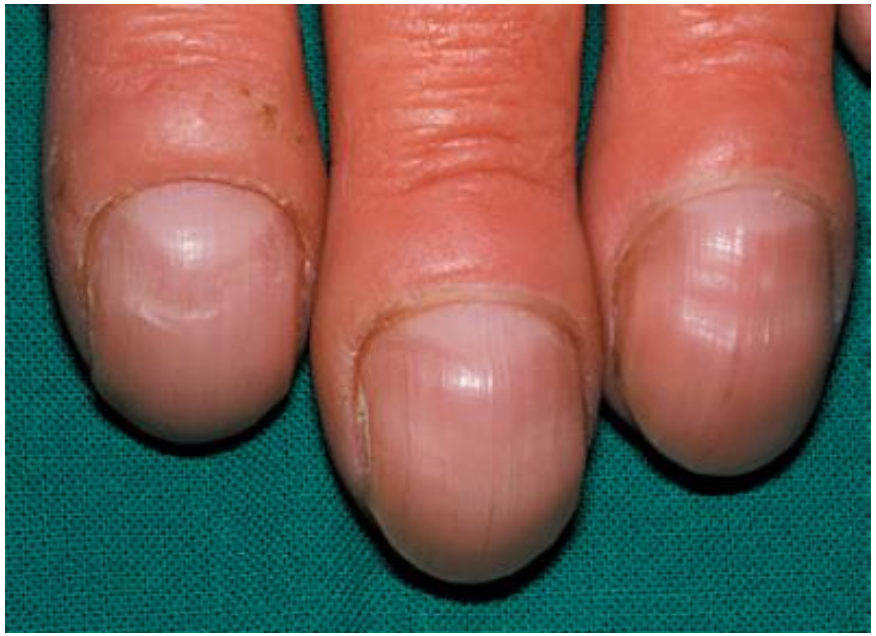
Define

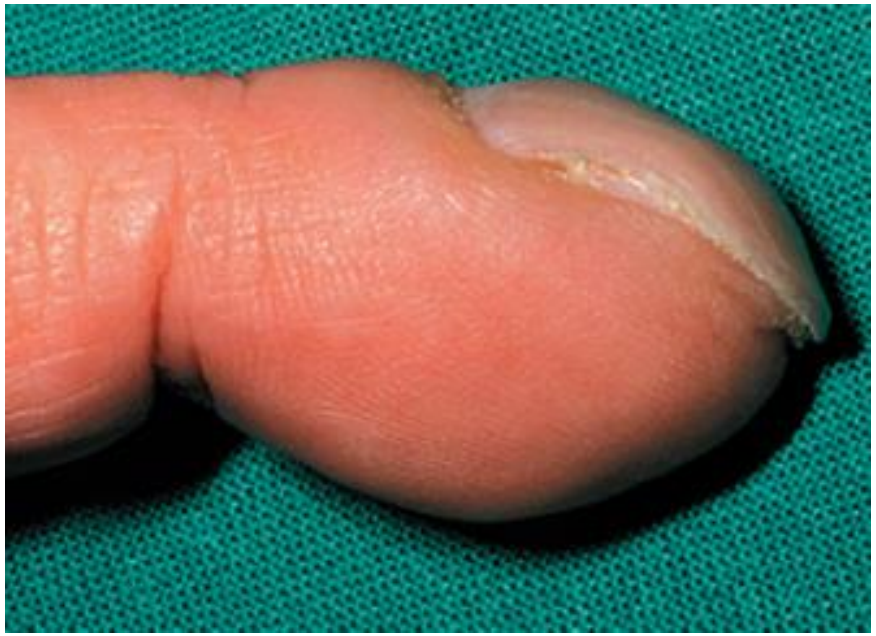
- **Lesion description**: Single horizontal ridge, all fingers involved
- **Underlying cause**: Sequela of any severe systemic illness, such as a heart attack, measles that affects growth of the nail matrix
- **Differential diagnosis**: Nail biting (usually one nail is involved)



2. Clubbing

- **Lesion description**: Loss of angle between nail fold and nail plate
- **Underlying cause**: CLUBBING mnemonic

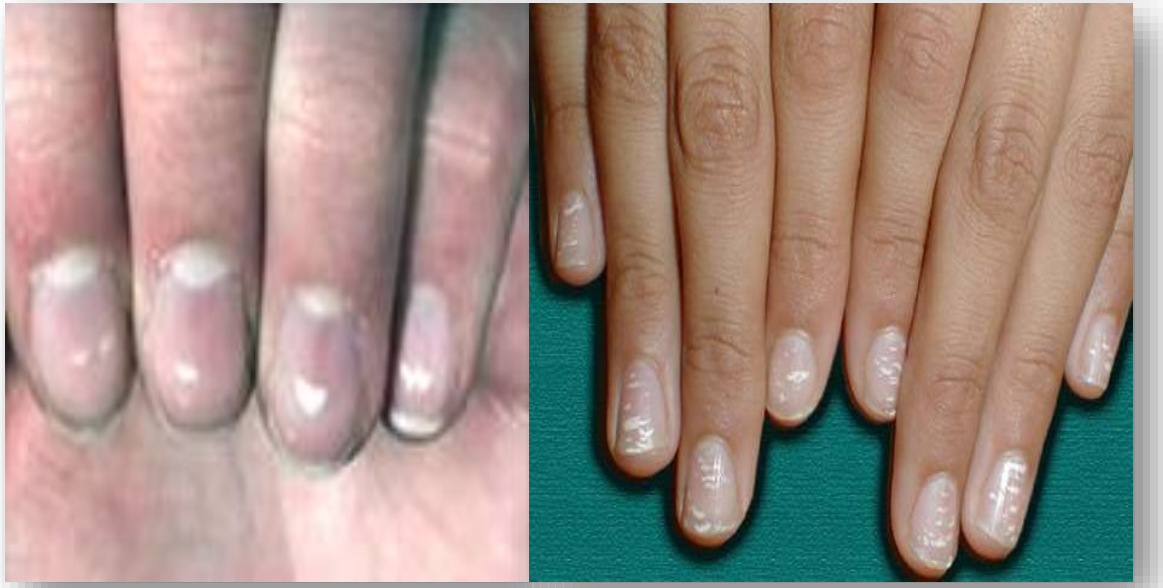




Mentioned in systemic diseases lecture – Hyperthyroidism

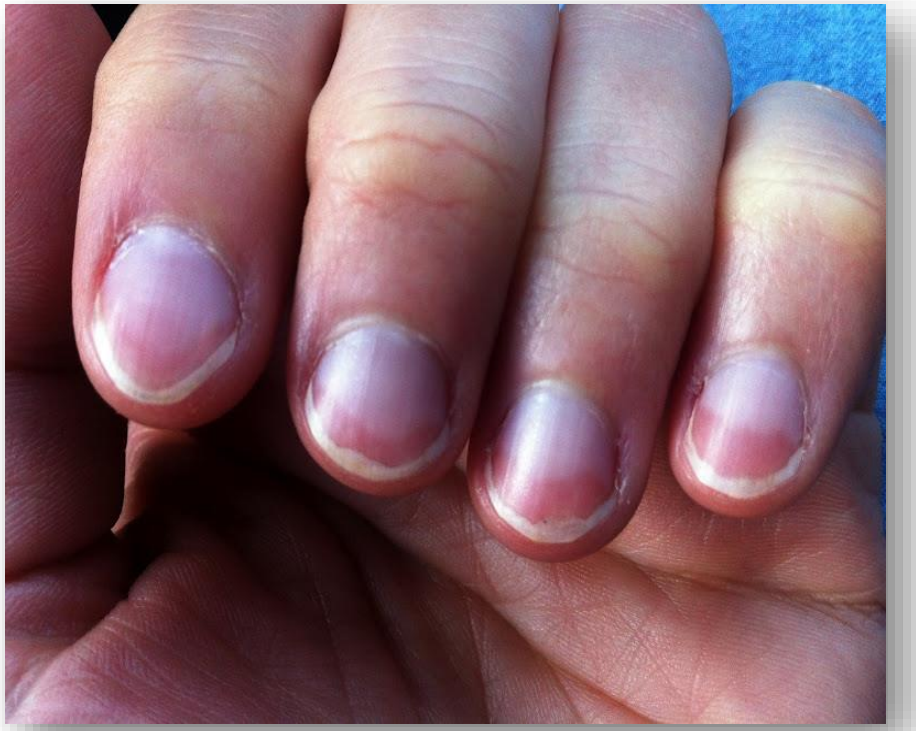
3. Leukonychia spots

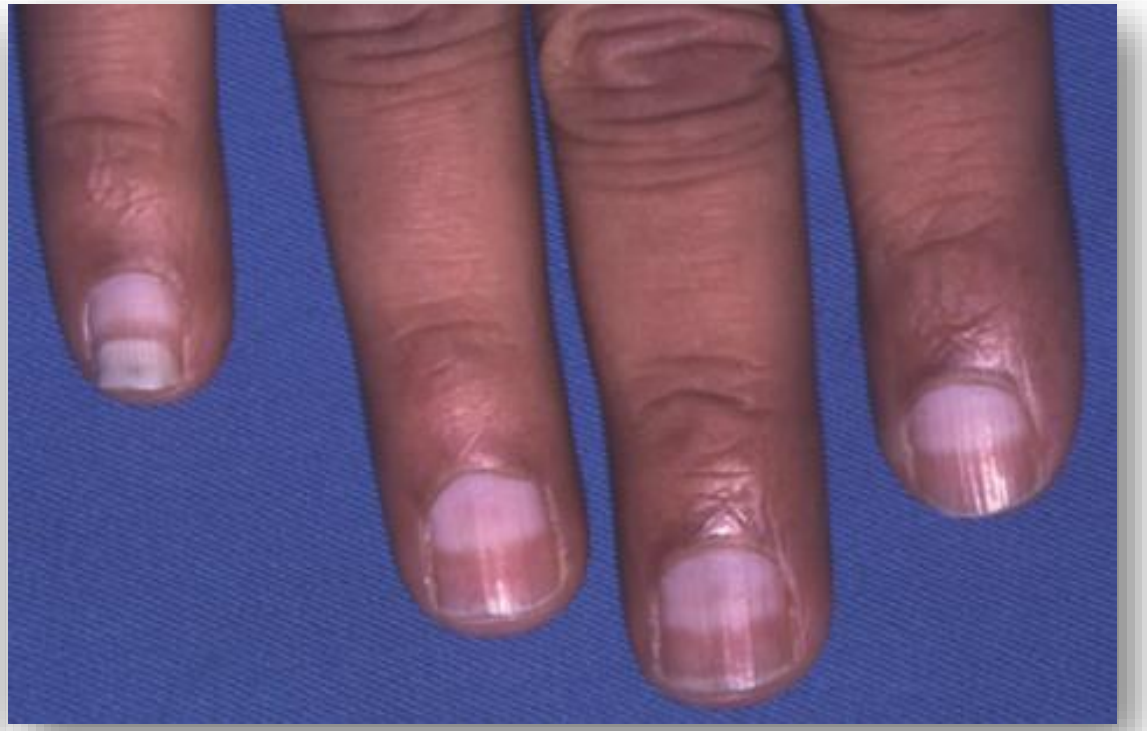
- **Lesion description**: Small white spots are groups of whitish nail cells trapped inside the nail plate
- **Underlying cause**: Minor trauma to the Matrix
- **Pseudoleukonychia**: surface layers develop a whitish flaky appearance due to a lack of moisture in the nail plate sometimes caused by picking off or removal of nail polish



4. Lindsay's nails

- **Lesion description**: White/brown 'half-and-half' nails
- **Underlying cause**: Chronic kidney disease

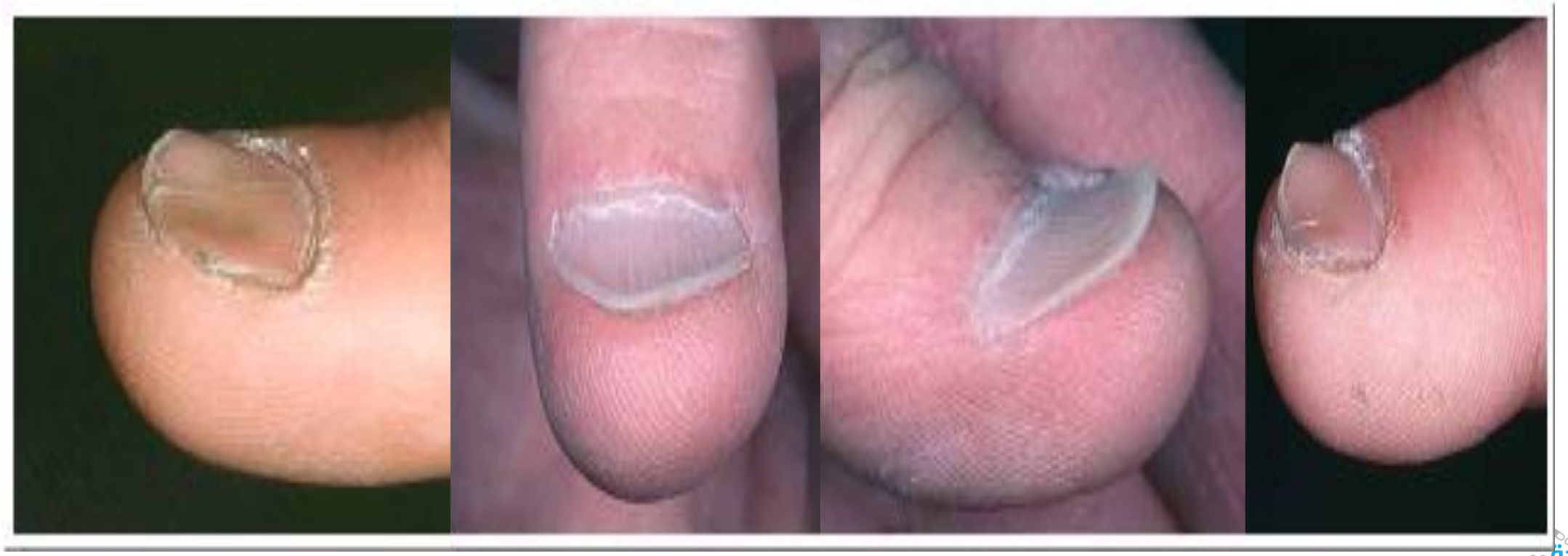




Mentioned in systemic diseases lecture – Skin and kidney diseases

5. Koilonychia (spoon nail)

- **Lesion description**: Flat or spoon shaped nail often thin and soft
- **Underlying cause**: iron deficiency (anaemia), excessive exposure to harsh chemicals etc., or is a congenital condition

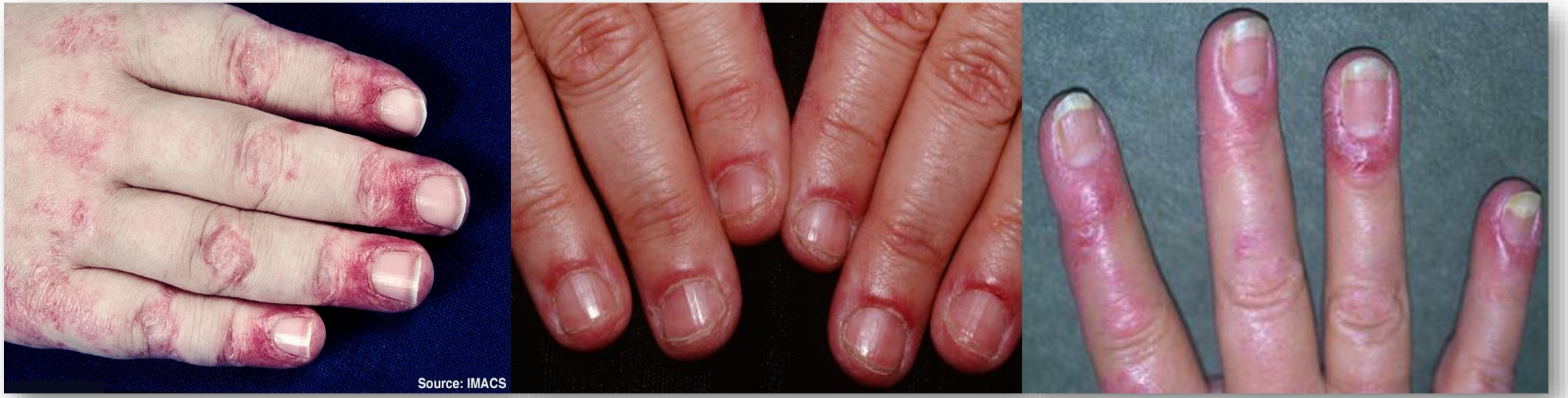


6. Muehrcke’s lines

- **Lesion description**: Narrow, white transverse lines
- **Underlying cause**: Decreased protein synthesis or protein loss

7. Nail-fold telangiectasia (Periungual telangiectasia)

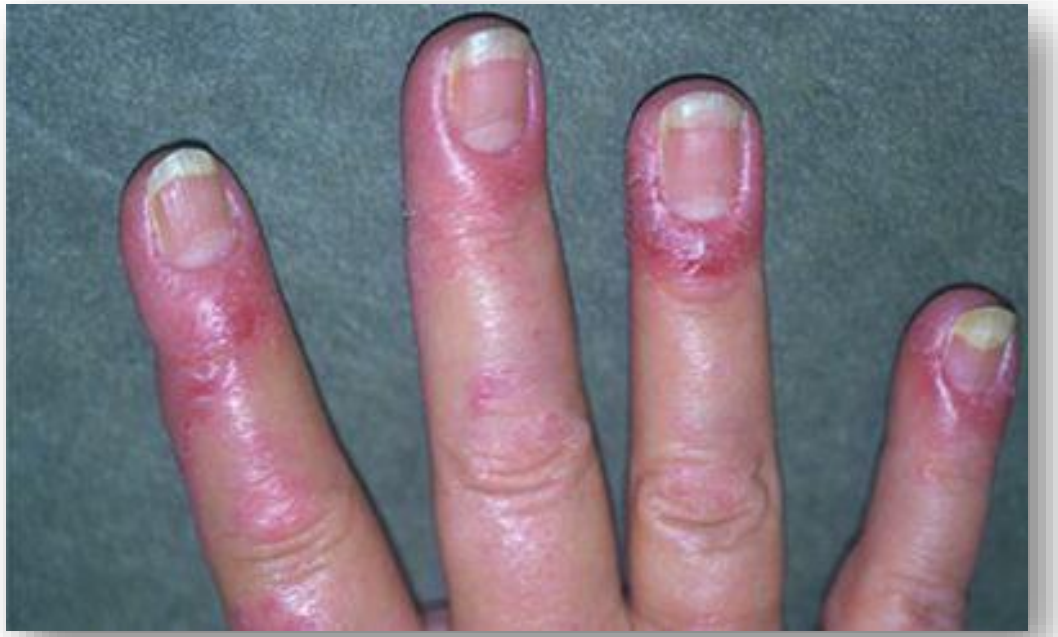
- **Lesion description**: Dilated capillaries and erythema at nail fold
- **Underlying cause**: Connective tissue disorders, including systemic sclerosis, systemic lupus erythematosus, dermatomyositis



Mentioned in systemic diseases lecture – Skin and connective tissue diseases

This presentation is seen in which of the following

A. Syphilis
B. Connective tissue disease
C. Tinea unguium
D. Kidney disease



Findings

شرح

8. Onycholysis

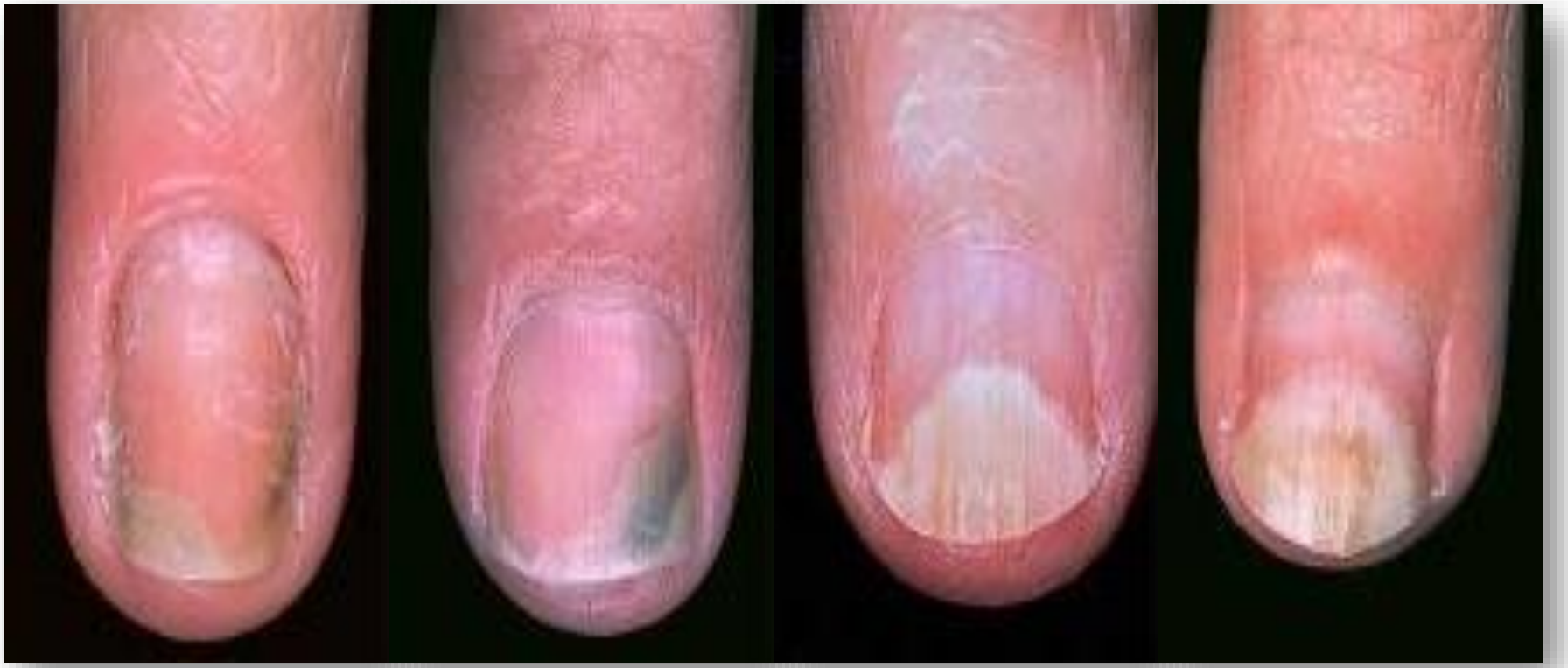
❖**Lesion description**: Nail plate separates from the nail bed

سنوات (2)

Define

❖**Underlying cause**: سنوات (1)

- Most commonly associated with external trauma to the nail (e.g., fungal infection)
- Can also be associated with an internal disorder (e.g., psoriasis)



For more details see fungal infection section

شرح

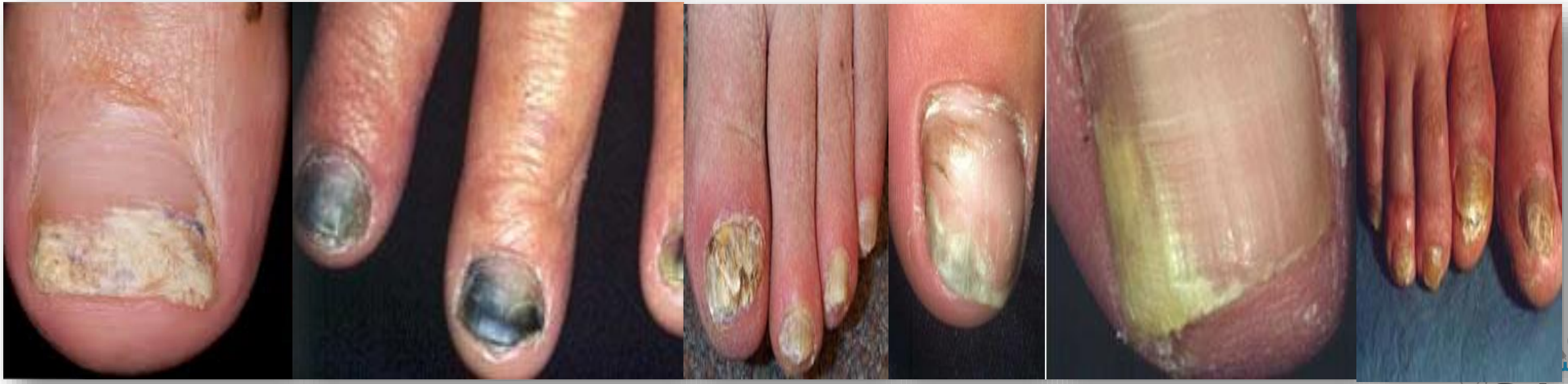
Disease

9. Onychomycosis

❖**Lesion description**:

- white spots that can be scraped off the surface, or long yellowish streaks within the nail substance
- The disease attacks the free edge and moves its way to the matrix.
- The infected portion is thick and discolored.

❖**Underlying cause**: Fungal infection most commonly Tinea unguium



Findings

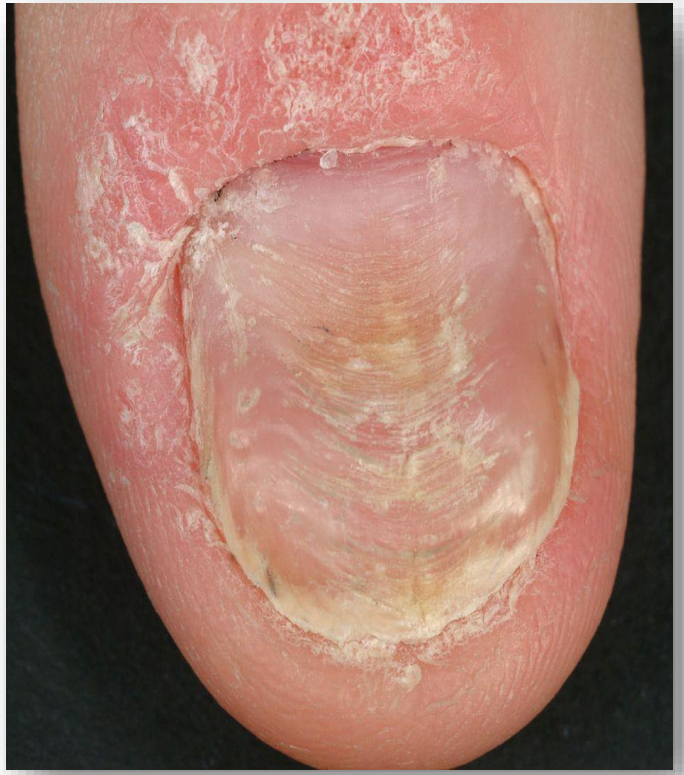
شرح

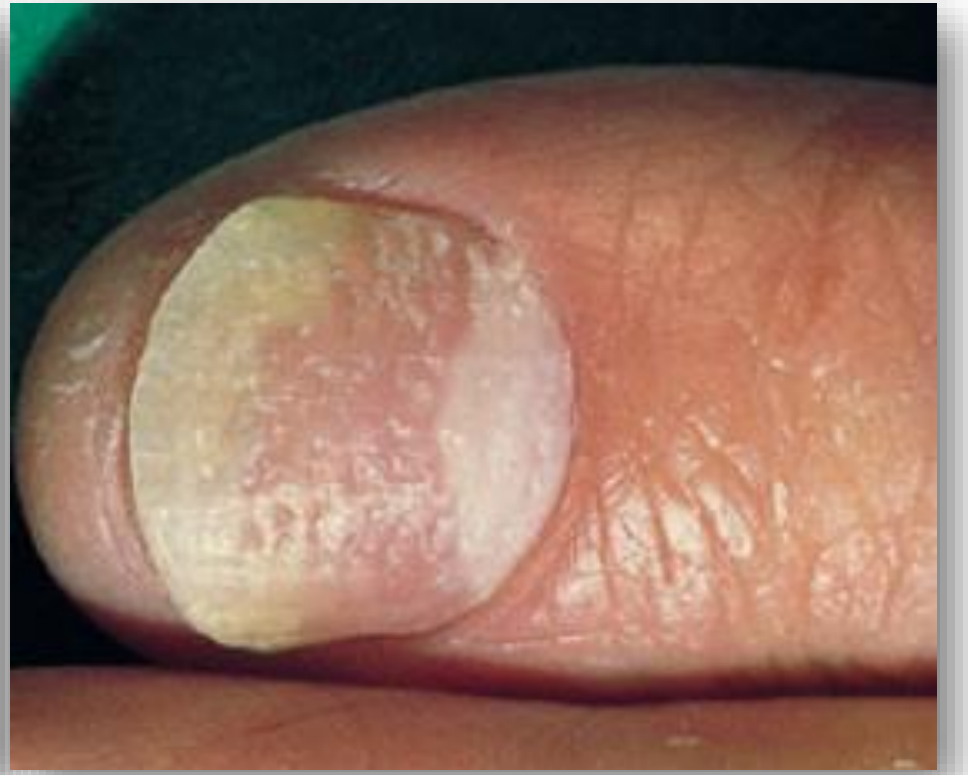
10. Pitting

❖**Lesion description**: Fine or coarse pits in nail

سنوات (1)

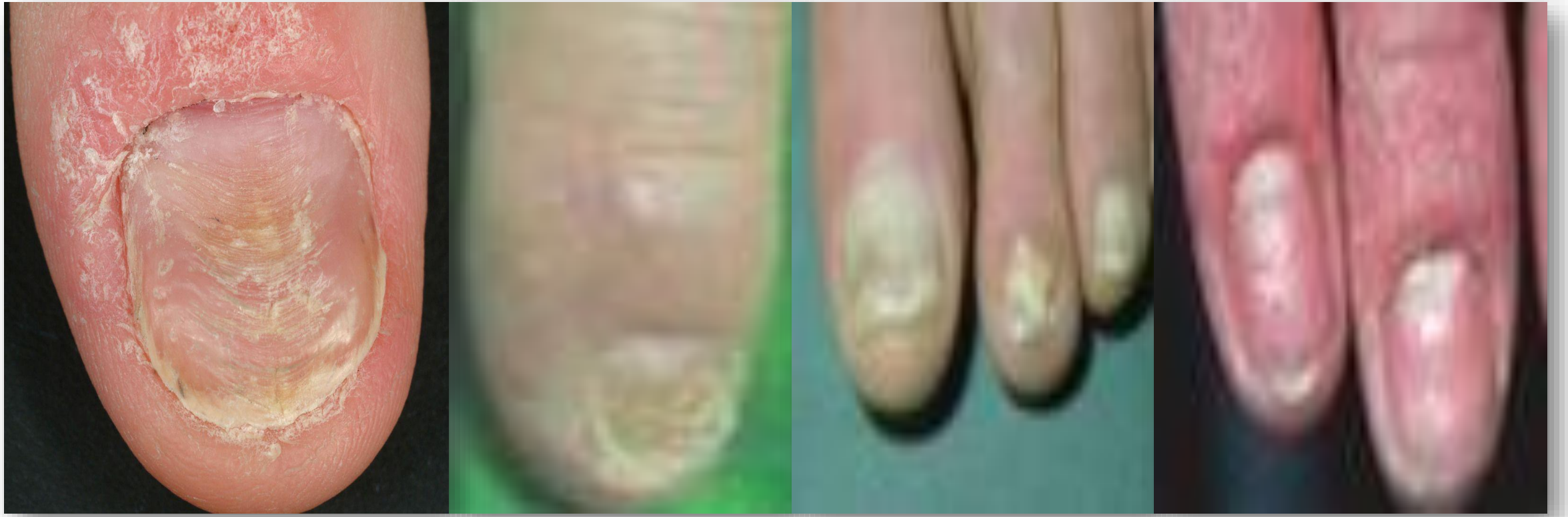
❖**Underlying cause**: Psoriasis, eczema, alopecia areata, lichen planus





Eczema of the nail

❖**Lesion description**: Can affect the eponychium, nail plate and bed causing pitting and onycholysis



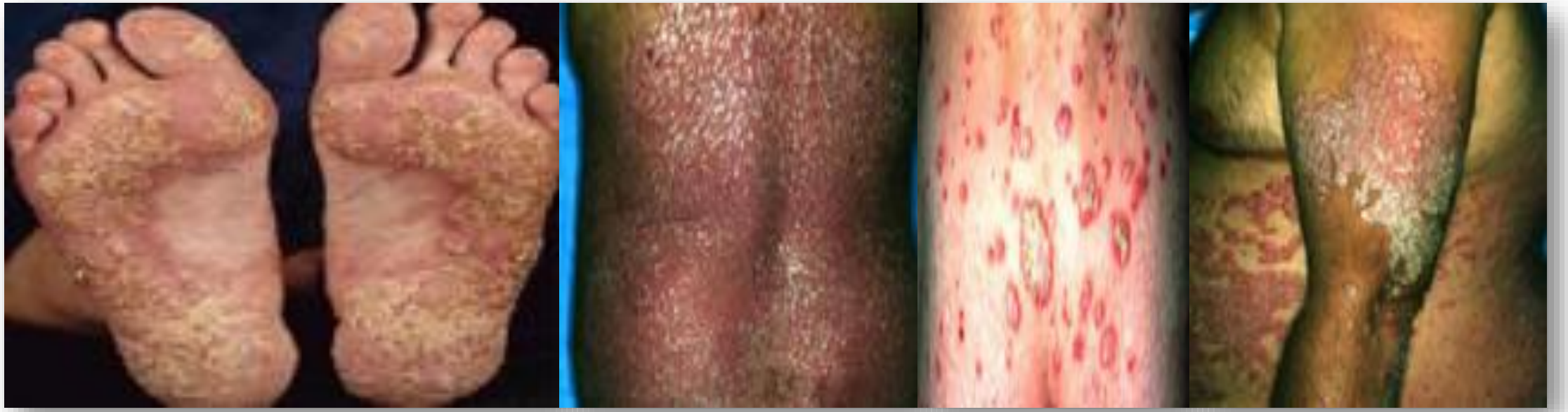
Finding: Pitting

Disease

شرح

Psoriasis

- **Lesion description**: Nail pitting, oil drop–like patterns of yellow or salmon discoloration, nail thickening, Onycholysis and discoloration
- **Underlying cause**: Psoriasis
- Plaques which form around the nail plate can cause pitting. Those which form beneath the nail plate can cause Onycholysis



هذول الصور الي كانوا مع السلايد

Findings

شرح

11. Splinter haemorrhages

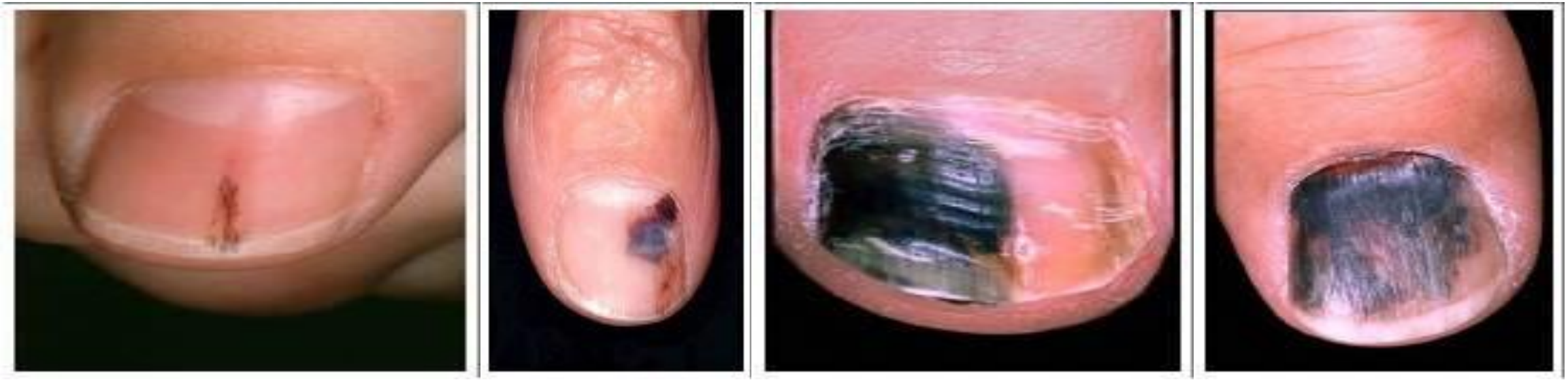
سنوات (1)

❖**Lesion description**: Small red streaks that lie longitudinally in nail plate

❖**Underlying cause**: Trauma, infective endocarditis

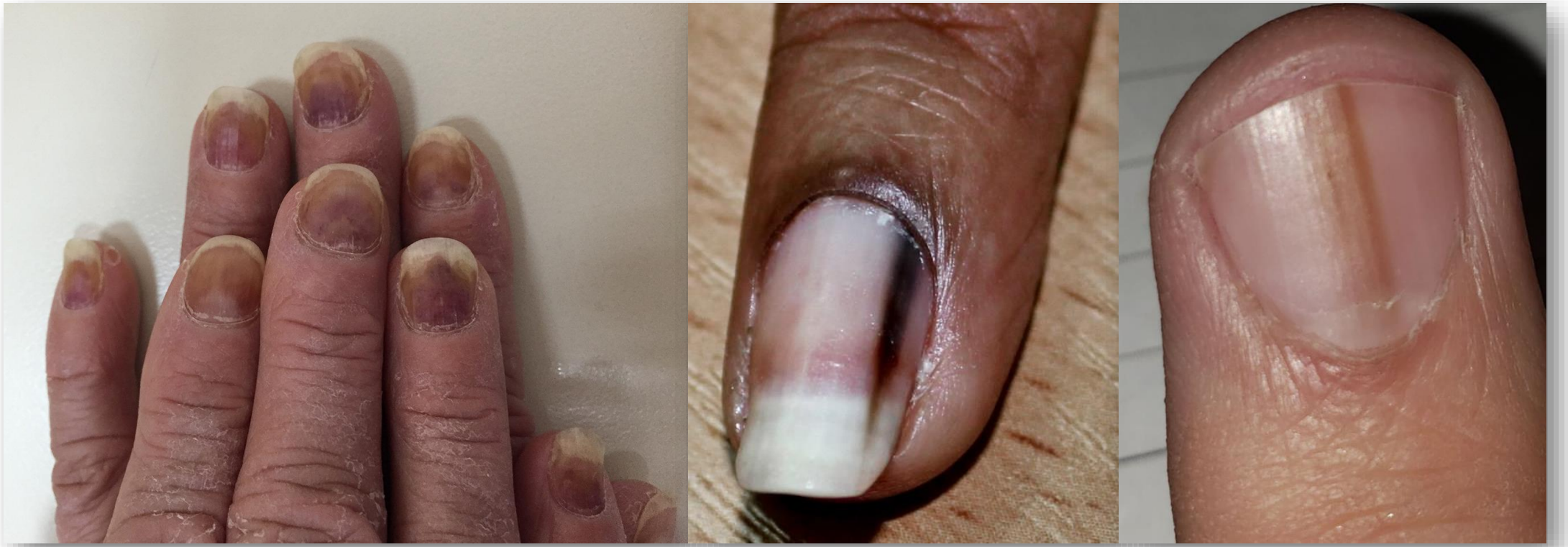
12. Bruised nail

- **Lesion description**: Dark, congealed spots of blood between nail plate and bed, extension of the pigment due to elongation of the nail
- **Underlying cause**: Crush injury, blunt trauma, repetitive microtrauma
- **Differential diagnosis**: Melanoma (the pigment is fixed)



13. Discolored nails

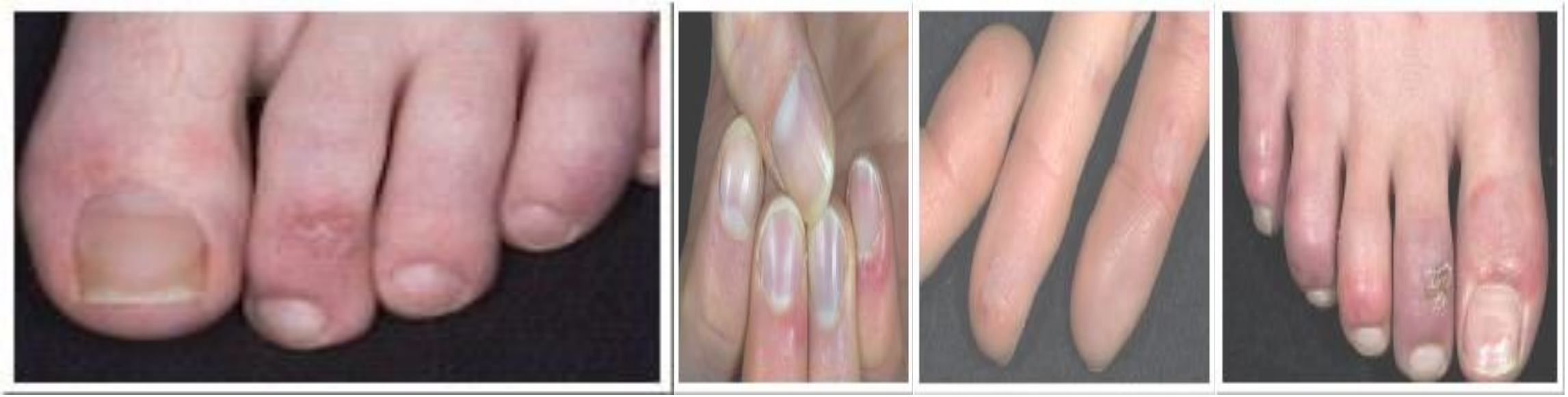
- Due to exposure to chemicals including dyes & nicotine, some medications, bacterial infections and systemic disorders.



السلايد ما كان معه صور، الصور هذه عشوائية من النت فمش مطلوبة

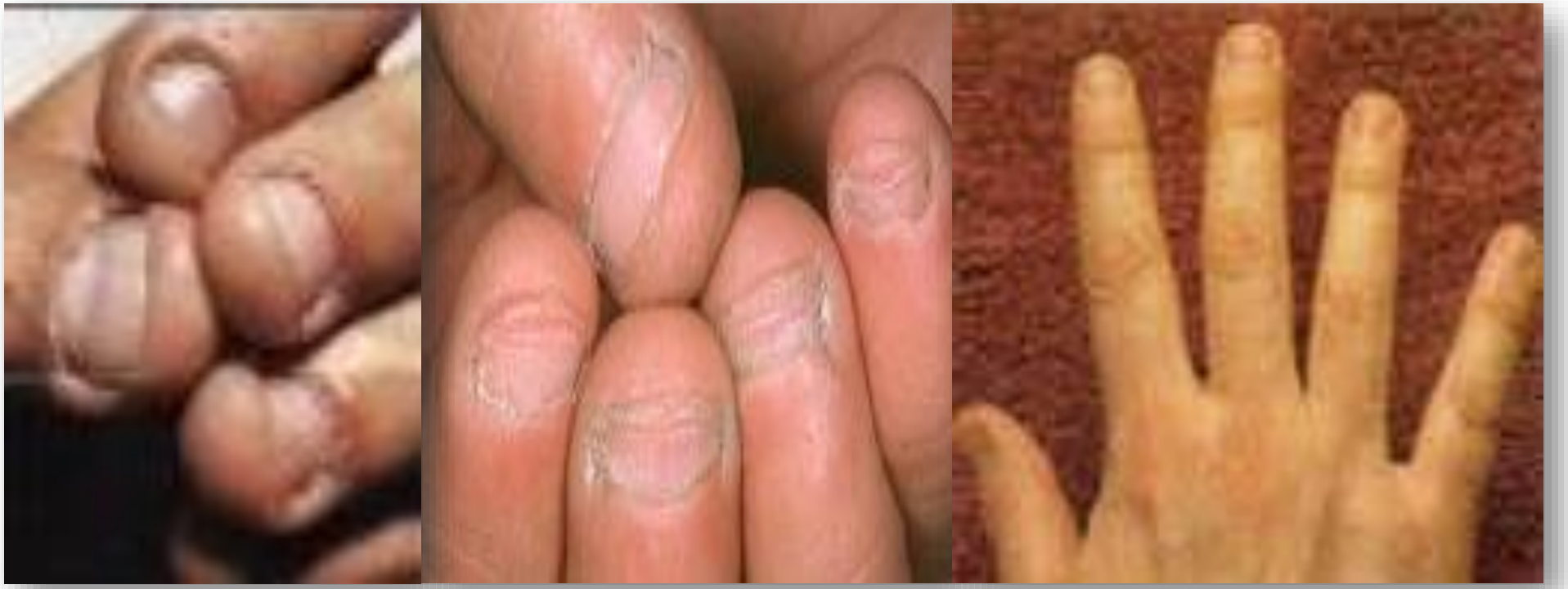
14. Chilblains

- **Lesion description**: An itchy, sore, tingly, red area resulting in broken skin
- **Underlying cause**: Prolonged exposure to cold and poor circulation
- **Differential diagnosis**: Raynaud's phenomenon



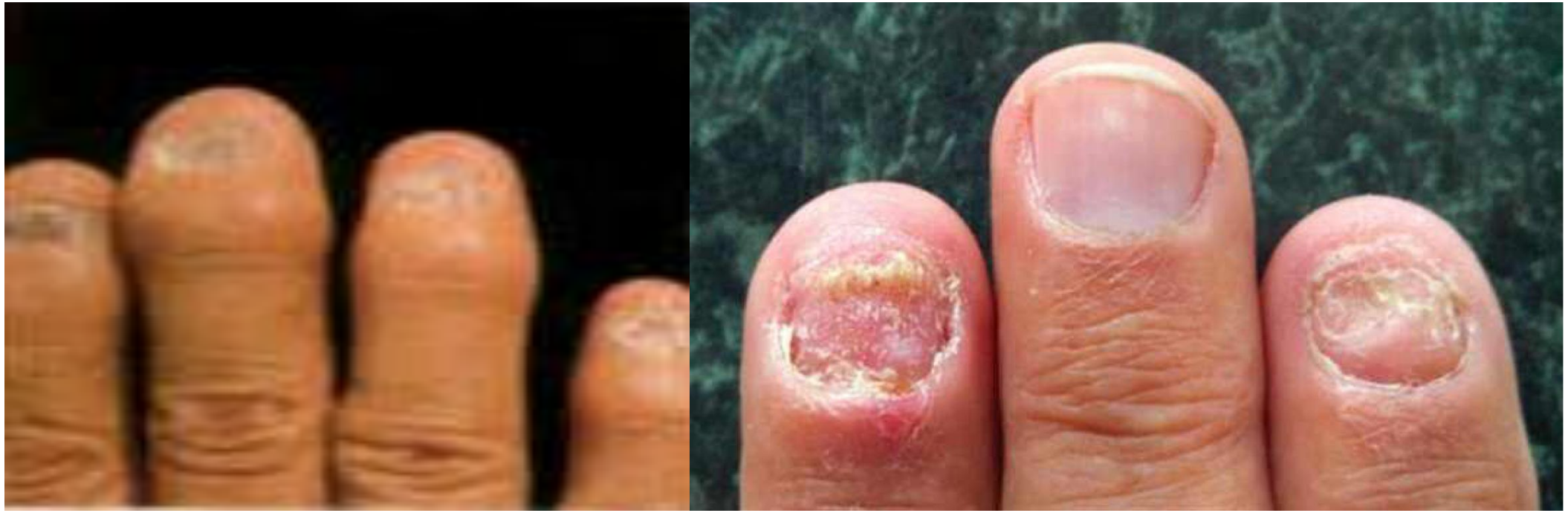
15. Onychophagy

- **Lesion description**: Bitten nails, often no free edge is visible. Nails look ragged and distorted; skin and nail bed can be exposed and raw
- **Management**: Regular manicure or apply nail enhancements to discourage the client from biting their nails



16. Onychatrophia

- **Lesion description**: The wasting away of the nail, causing it to lose its luster and become smaller. The nail can also shed completely.
- **Underlying cause**: injury or disease

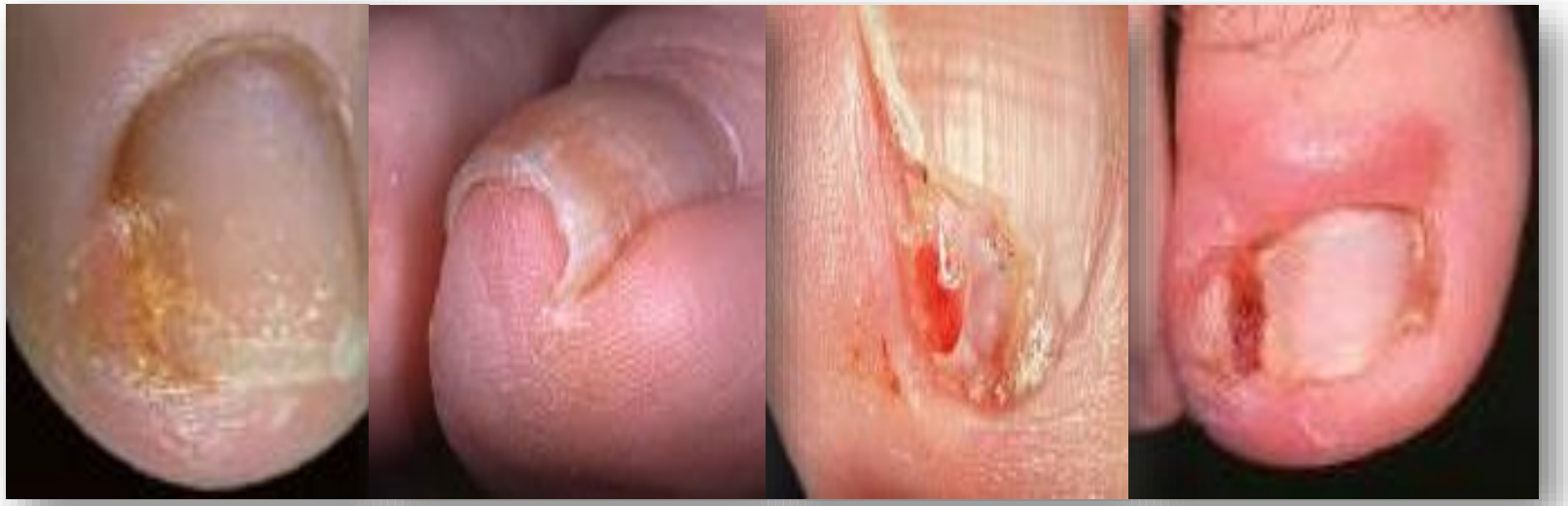


What is the diagnosis

A. Onychatrophia
B. Onychauxis
C. Paronychia
D. Onychophagy
E. Onychorrhexis

17. Ingrown nails

- **Lesion description**: The nail grows into the sides of the flesh and may cause infection
- **Underlying cause**: Nail grows into the sides of the flesh
- **Differential diagnosis**:
  - Filling the nails too much in the corners
  - Failing to correct hang nails

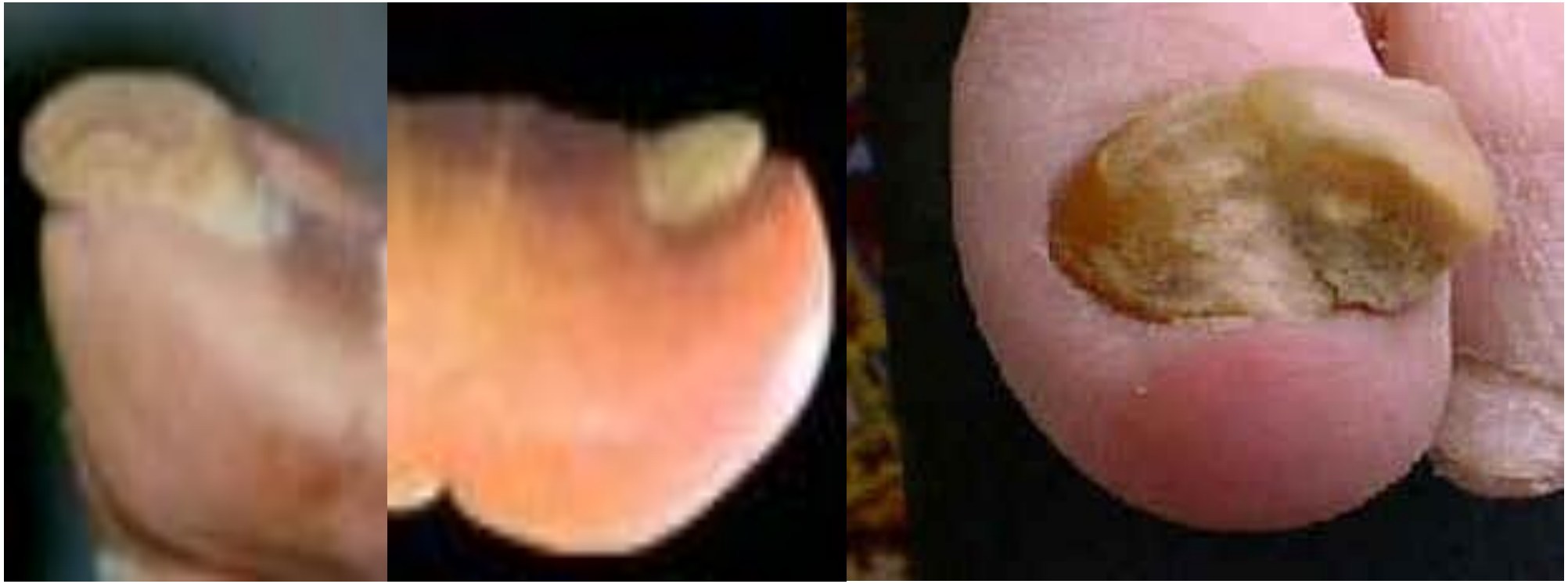


18. Onychorrhexis

- **Lesion description**: Split or brittle nails
- **Underlying cause**: injury to the finger or exposure to harsh chemicals

19. Onychauxis

- **Lesion description**: An overgrowth of the nail, in thickness rather than in length
- **Underlying cause**: Internal disturbance, such as a local infection



Findings

شرح

20. Nail pterygium

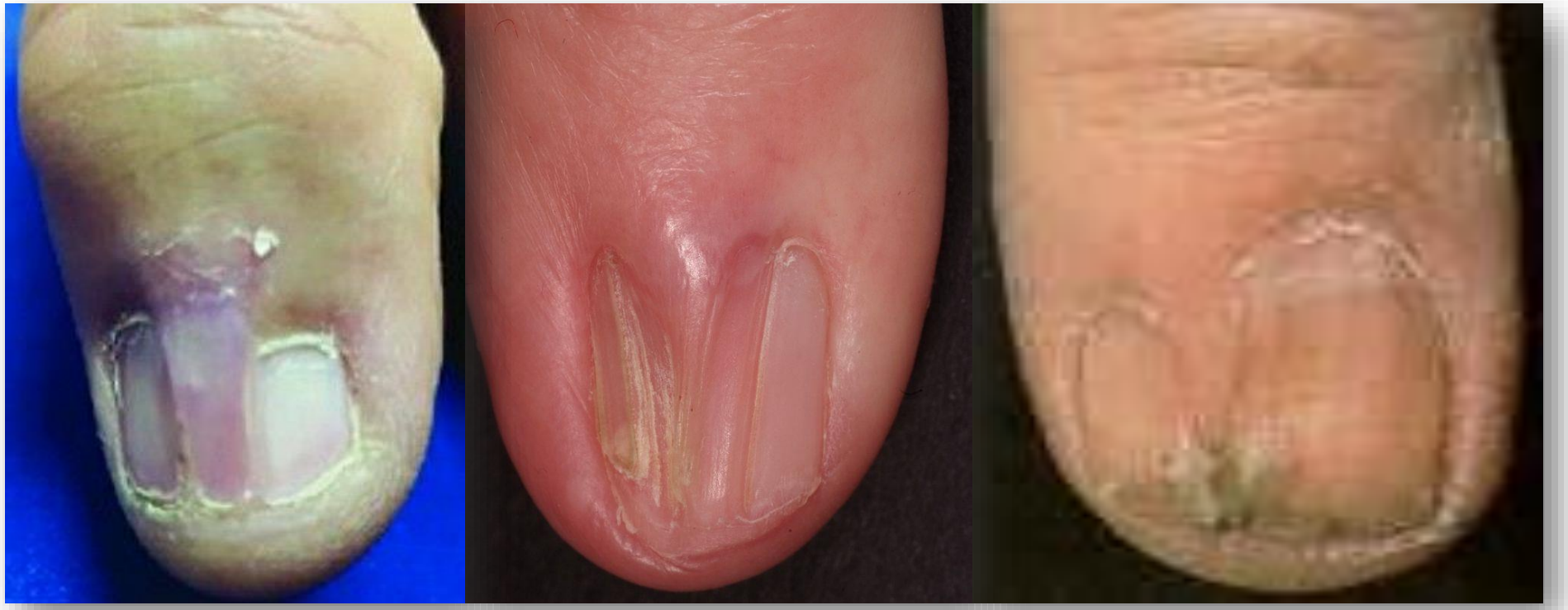
(2) سنوات

Define

❖**Lesion description**: An abnormal winged like growth of cuticle on the nail plate. The skin is slowly stretched and dragged along the bed.

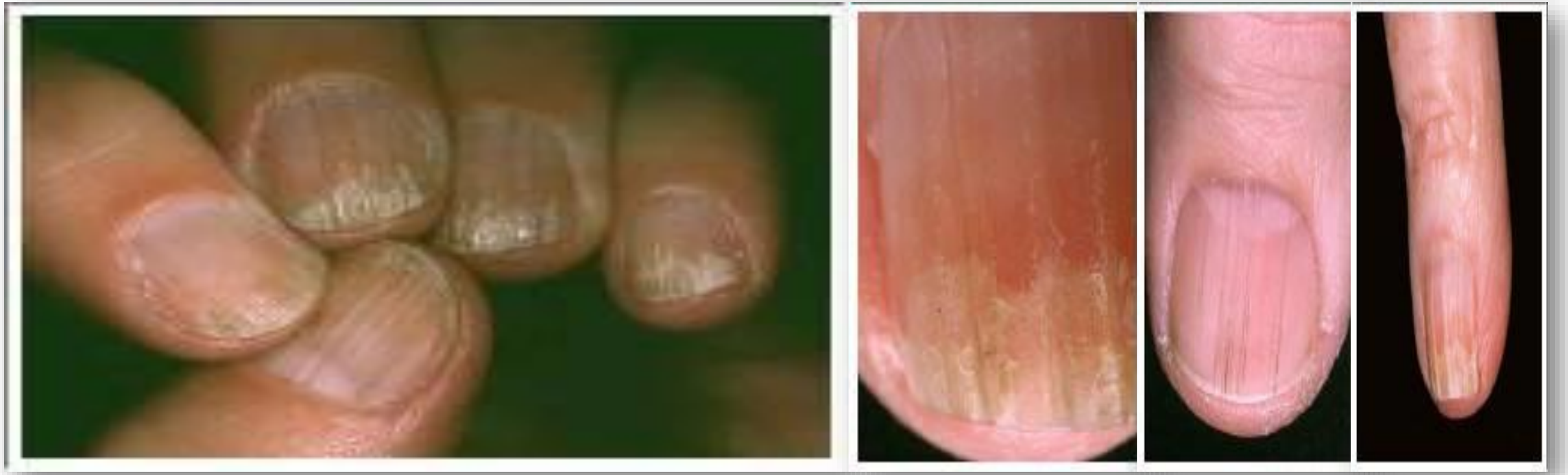
(3) سنوات

❖**Underlying cause**: Most commonly caused by severe trauma such as warts, burns & blood circulation disorders and lichen planus



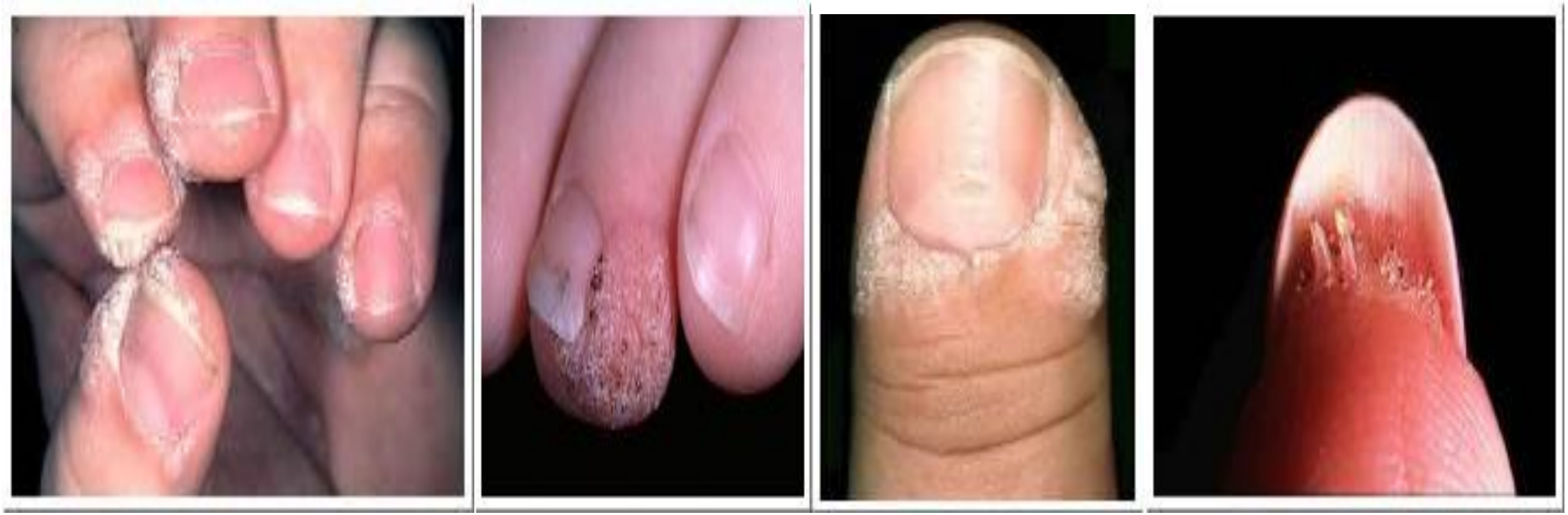
21. Ridges, furrows, corrugations

- **Lesion description**: Multiple shallow/deep ridges
- **Underlying cause**: illness or injury, excessive dieting, incorrect removal of nail enhancements, pregnancy, etc.



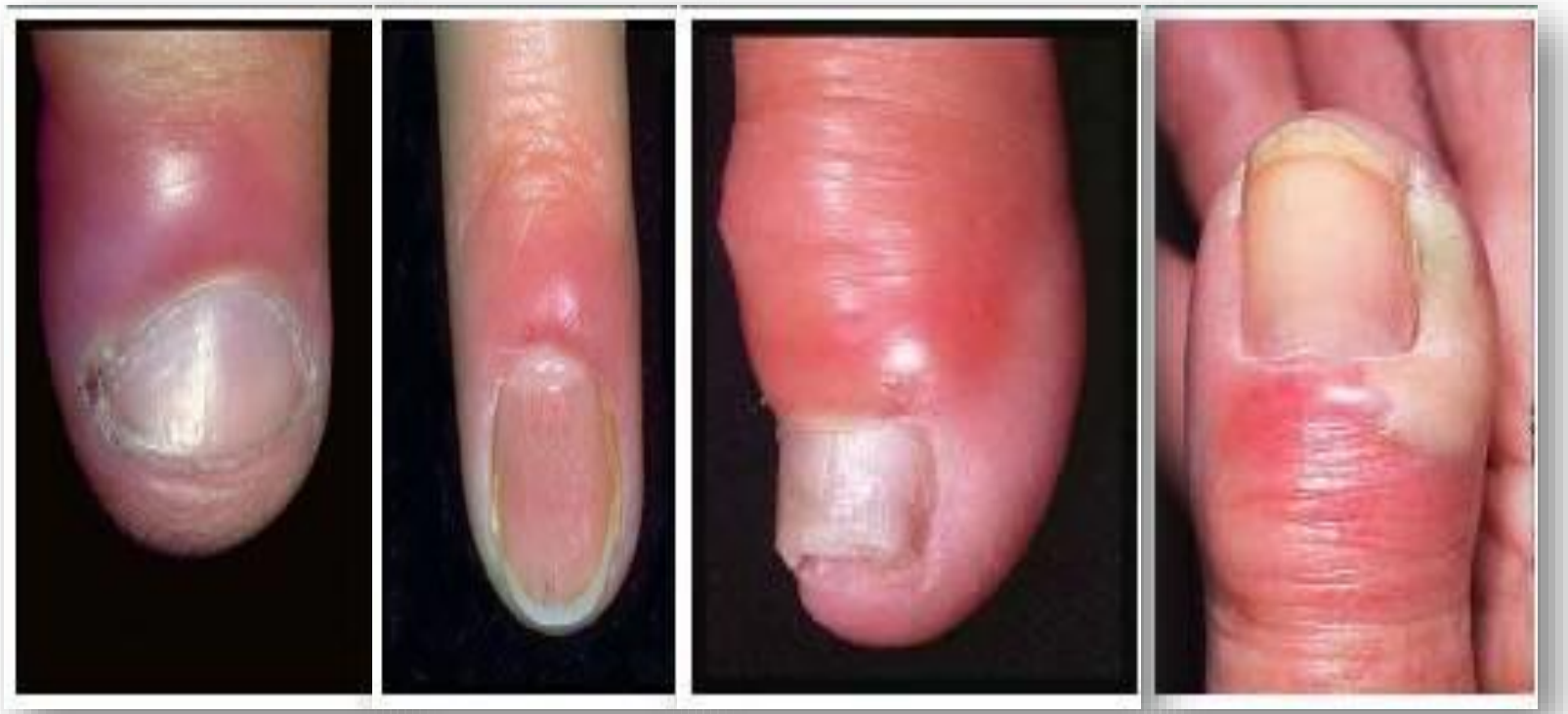
22. Verruca vulgaris (common warts)

- **Lesion description**: Raised lumps of horny tissue in areas of pressure
- **Underlying cause**: HPV 1-4
- **Note**: Cryotherapy may damage the nail matrix



23. Paronychia

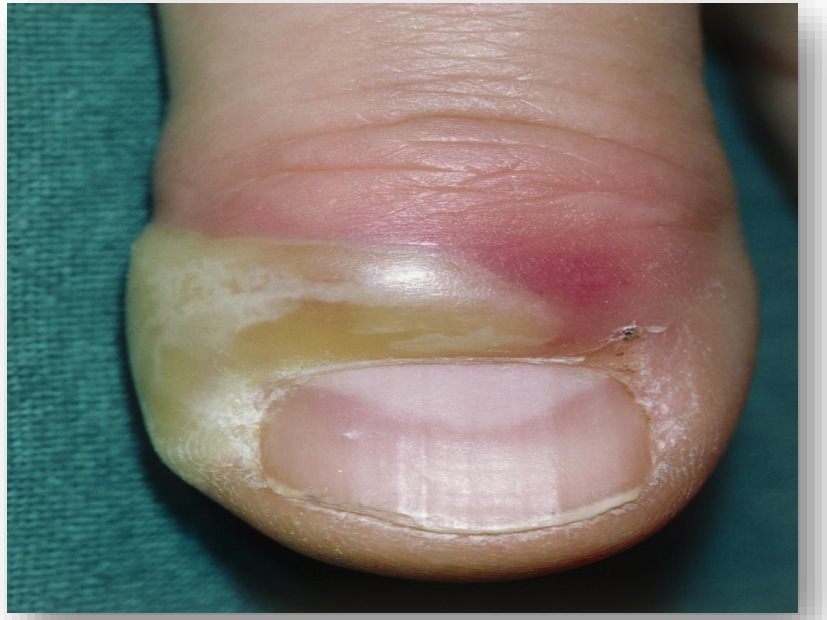
- An infectious and inflammatory condition of nail folds.
- Chronic paronychia may weaken defenses and increase the risk of developing a fungal infection of the nail or may permanently deform the nail plate.
- سنوات (3) **Acute paronychia**: Staphylococcal; **Chronic paronychia**: Candidiasis



سنوات (2)

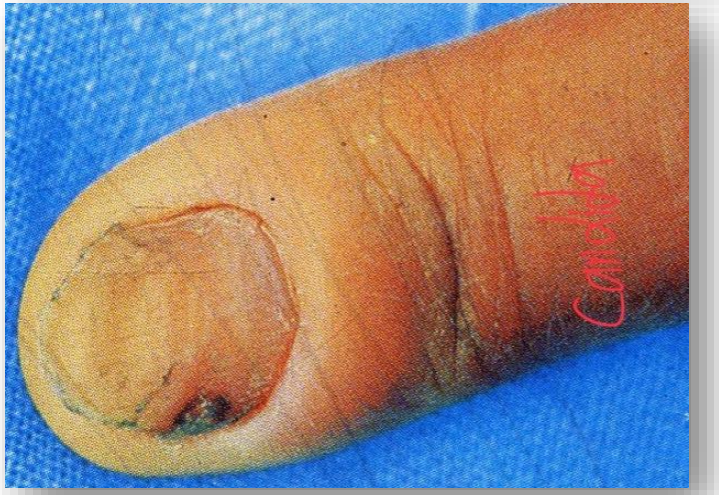
Paronychia

- **Describe this lesion:**
  - Pustule at the nail fold surrounded by erythema (acute paronychia)
- **What is the most common organism**
  - S.aureus
- **Treatment**
  - Incision and drainage with topical antibiotics



- **What is your diagnosis ?**
  - Chronic paronychia
- **What is the most common organism**
  - Candida albicans

سنوات (1)



What is the diagnosis

A. Lichen planes nails

B. Chronic paronychia

C. Psoriasis nails

D. onychomycosis

Chronic paronychia Loss of cuticle

Others

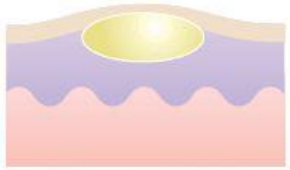
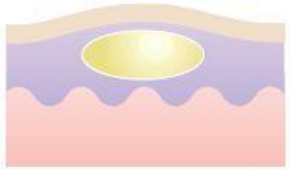
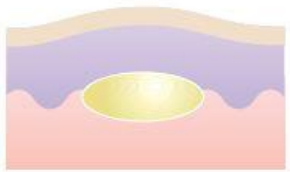
سنوات (1)

❖**Define Anonychia**: absence of nail

Bullous dermatosis

Types of blisters

- **Subcorneal** blisters:
  - Just beneath the stratum corneum
  - Have the **thinner** roofs.
  - Rupture easily & leave an **oozing** denuded surface
- **Intra-epidermal** blisters:
  - Within the **prickle cell layer** of the epidermis
  - Have thin roofs
  - Rupture easily & leave an **oozing** denuded surface
- **Subepidermal** blisters:
  - Between the **dermis & epidermis**
  - Their roofs are relatively **thick**
  - Tend to be **tense**
  - May contain **blood**

Location of bullae	Diseases
 Subcorneal bulla	Bullous impetigo Miliaria crystallina Staphylococcal scalded skin syndrome
 Intra-epidermal bulla	Acute eczema Viral vesicles Pemphigus Miliaria rubra Incontinentia pigmenti
 Subepidermal bulla	Bullous pemphigoid Cicatricial pemphigoid Pemphigoid gestationis Dermatitis herpetiformis Linear IgA disease Bullous erythema multiforme Bullous lichen planus Bullous lupus erythematosus Porphyria cutanea tarda Toxic epidermal necrolysis Cold or thermal injury Epidermolysis bullosa

Different mechanism to form blisters

- **Spongiosis**: keratinocyte get separated by the accumulation of edema fluid
- **Epidermal cell necrosis** seen when keratinocytes are invaded by a virus as varicella zoster or herpes simplex. The cells get swollen and vacuolated to produce an appearance called balloon degeneration.
- **Basal cell damage** seen in epidermolysis bullosa simplex, lupus erythematosus and lichen planus, and on rare occasions this can be so severe as to produce bullae.
- **Acantholysis** seen in pemphigus vulgaris.
- **Damage to the lamina Lucida** seen in bullous pemphigoid
- **Dermal damage** seen in dermatitis herpetiformis, porphyria cutanea tarda and recessive dystrophic epidermolysis bullosa

شرح

1. Pemphigus vulgaris

سنوات (1)

❖**Primary lesion of pemphigus vulgaris**: Bulla

سنوات (1)

❖**Characteristic lesion**: painful blisters (flaccid bullae) and erosions on the skin and mucous membranes, most commonly inside the mouth

❖It is an autoimmune inactivation of desmosomes between keratinocytes (IgG antibodies against desmoglein 3); Type II HSR

❖characterized histologically by a "tombstone" appearance and acantholysis

❖characterized by a "fish net" (reticular) pattern on immunofluorescence

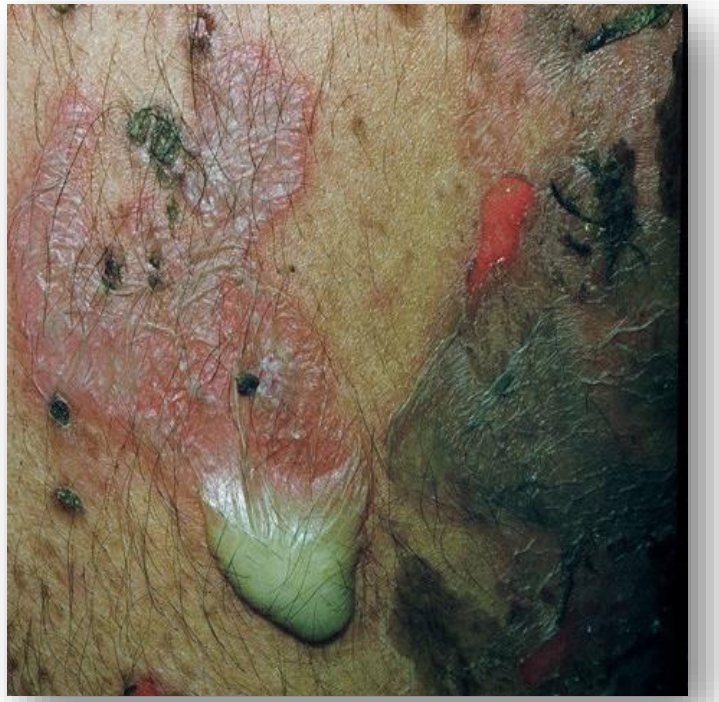
❖Drug-induced pemphigus is also recognized and is most often caused by Penicillamine, ACE inhibitors, ARBs, and Cephalosporines.

❖Pemphigus is sometimes triggered by cancer (paraneoplastic pemphigus), infection or trauma.

Pemphigus vulgaris – Clinical Presentation

- Most patients with pemphigus vulgaris first present with lesions on the mucous membranes such as the mouth and genitals. Blisters usually develop on the skin after a few weeks or months, although in some cases, mucosal lesions may be the only manifestation of the disease.

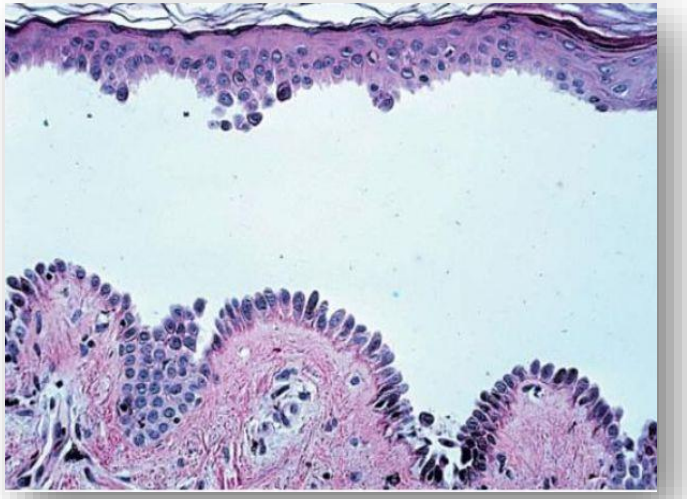
- Features of oral mucosal pemphigus include:
  1. Oral lesions in 50–70% of patients
  2. Superficial blistering and erosions
  3. Widespread involvement within the mouth
  4. Painful, slow-to-heal ulcers
  5. Spread to the larynx causing hoarseness when talking
  6. Difficulty eating and drinking.



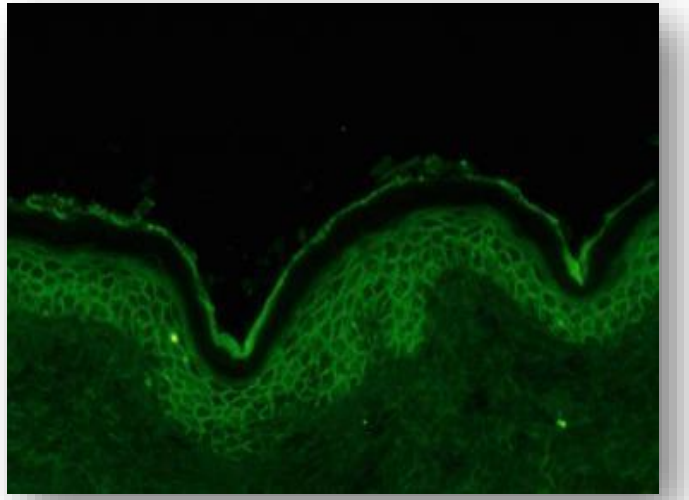
Pemphigus vulgaris

❖Diagnosis of pemphigus vulgaris

- biopsy from the skin adjacent to a lesion. Histology typically shows rounded-up and separated keratinocytes (acantholytic cells)
- Confirmed by direct immunofluorescence staining of perilesional skin biopsy
- In most cases, circulating antibodies can be detected by a blood test (anti-dsg1 and anti-dsg3 antibody titers)



"Tombstone" appearance and acantholysis



"Fish net" (reticular) pattern on immunofluorescence

Pemphigus vulgaris

❖Complications

1. Very extensive, life-threatening erosions
2. Secondary bacterial infection
3. Fungal infection, especially candida
4. Viral infection, especially herpes simplex
5. Nutritional deficiencies due to difficulty eating
6. Complications of systemic steroids especially infections and osteoporosis.
7. Complications of immune suppressive treatments
8. The psychological effects of severe skin disease and its treatment (anxiety and depression)

❖Management:

- **First-line**: High-dose topical steroids
- **Second-line**: Systemic glucocorticoids and immunosuppressants

شرح

2. Bullous pemphigoid

سنوات (1)

- **Primary lesion of Bullous pemphigoid**: Tense subepidermal bulla

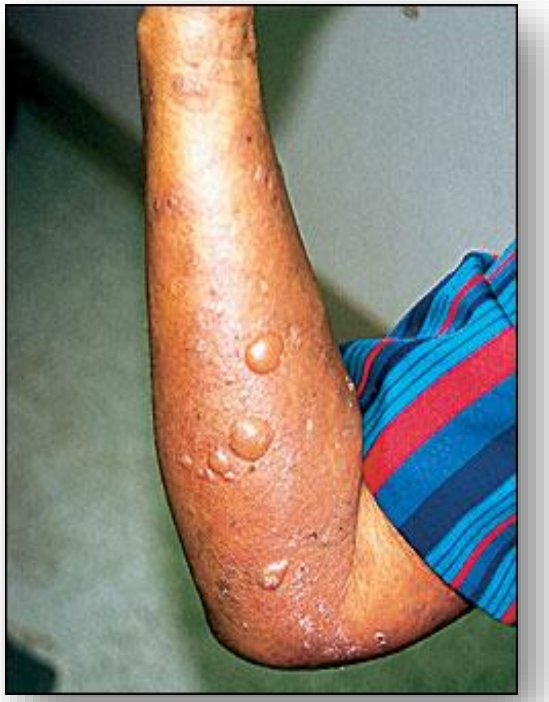
سنوات (1)

- **Characteristic lesion**: severe itch and (usually) large, tense bullae (fluid-filled blisters), which rupture forming crusted erosions
- It is an autoimmune disease due to IgG antibodies against hemidesmosome components (BP180); Type II HSR
- characterized histologically by detachment of the basal cell layer from the basement membrane
- characterized by a linear pattern on immunofluorescence
- The most common drugs associated with bullous pemphigoid are the PD1-inhibitor immunotherapies, other medications include Diuretics and Anti-diabetes drugs

Bullous pemphigoid

❖ **Diagnosis of bullous pemphigoid**:

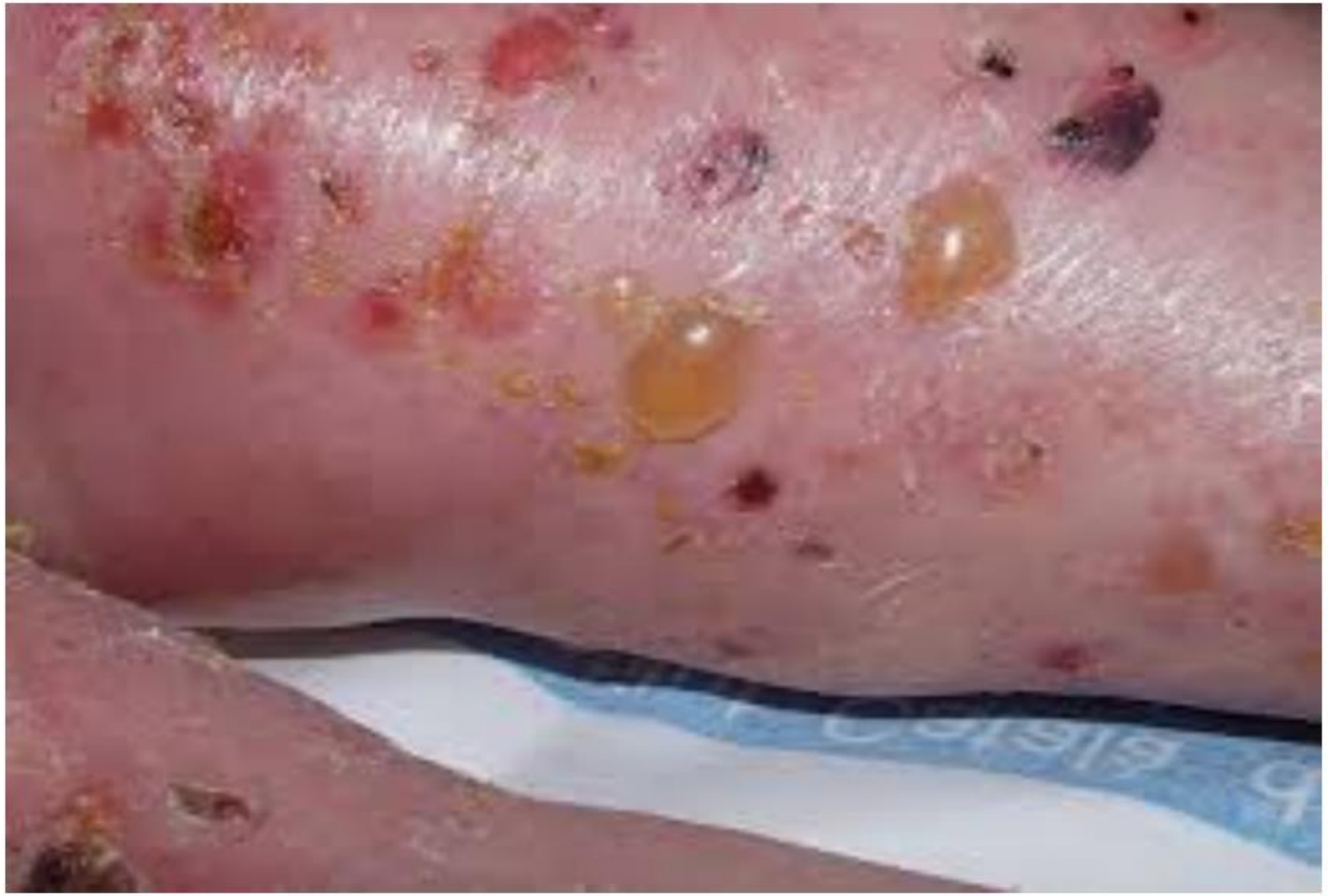
- When typical bullae are present, the diagnosis is suspected clinically
- Confirmed by a skin biopsy of an early blister (best diagnostic tool)
- Direct immunofluorescence staining of a skin biopsy
- Blood tests include an indirect immunofluorescence test for circulating pemphigoid BP180 antibodies.



❖ **Treatment**:

- **First-line**: High-dose systemic steroids
- **Second-line**: Immunosuppression

Bullous pemphigoid



Tense bullae with vesicles and erythema
Hx of pruritic rash up to 3 months
Bollous pemphigoid

شرح

3. Dermatitis herpetiformis

(3) سنوات

❖**Primary lesion of dermatitis herpetiformis**: Vesicles

(1) سنوات

❖**Characteristic lesion**: grouped (herpetiform) excoriations or vesicles symmetrically located on extensor remission; watch for signs surfaces of elbows, knees, sacrum, buttocks, and shoulders with intense **pruritus** and burning sensation

❖**Diagnosis**:

- **Light microscopy**: neutrophilic abscesses in dermal papillae, dermal infiltrates of neutrophils and eosinophils with subepidermal vesicles
- **Direct immunofluorescence**: granular IgA deposits in the tips of the dermal papillae

❖**Treatment**:

- **First-line**: Gluten-free diet + Dapsone
- Corticosteroids (prednisone) alone or with azathioprine, Mycophenolate mofetil or a tetracycline as alternatives.

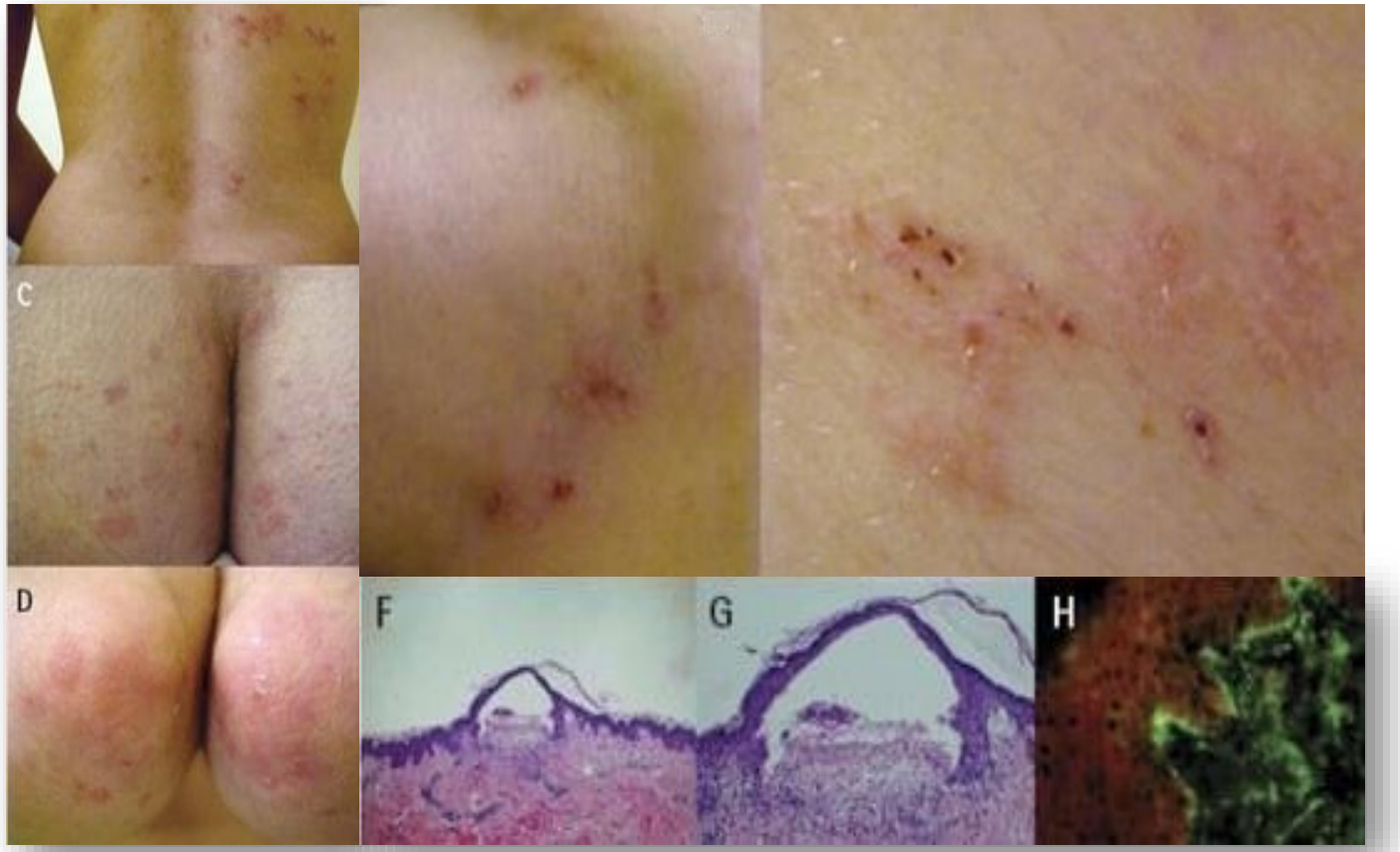
شرح

Dermatitis herpetiformis

سنوات (2)

❖What is your diagnosis ?

A. Herpes zoster
B. Dermatitis herpetiformis
C. Eczema herpeticum
D. Herpes simplex
E. Lichen planus



شرح

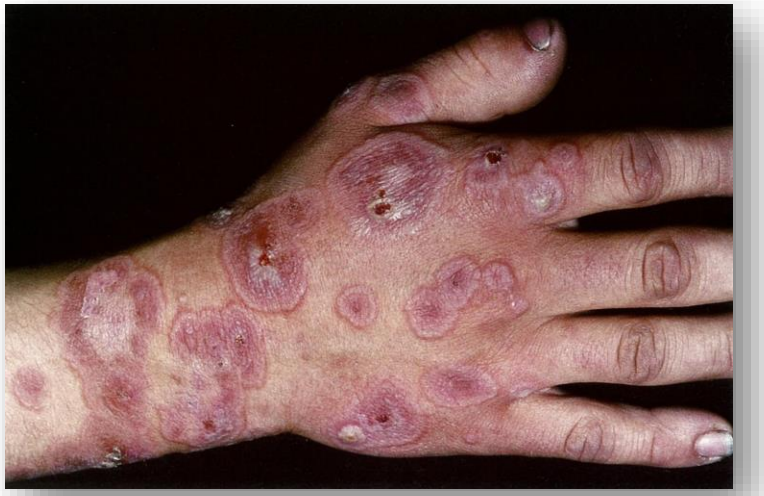
4. Erythema multiforme

❖**Erythema multiforme** is a type of hypersensitivity reaction that occurs in response to medications, infections, illness , stress , sun or cold exposure.

(3) سنوات ❖**Primary lesion of dermatitis erythema multiforme:** Target lesions

(2) سنوات ❖Define target lesion:

- central sore surrounded by pale red ring



(1) سنوات ❖Mention 2 diseases containing target lesion

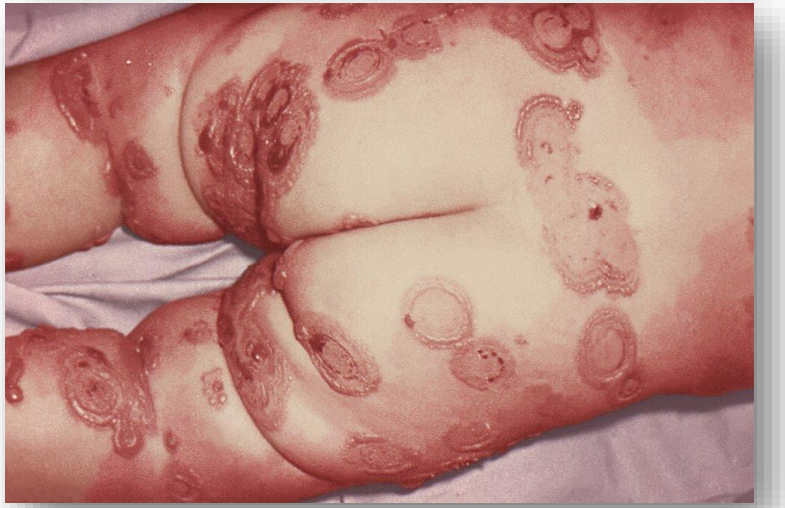
- Lyme disease, Erythema multiforme

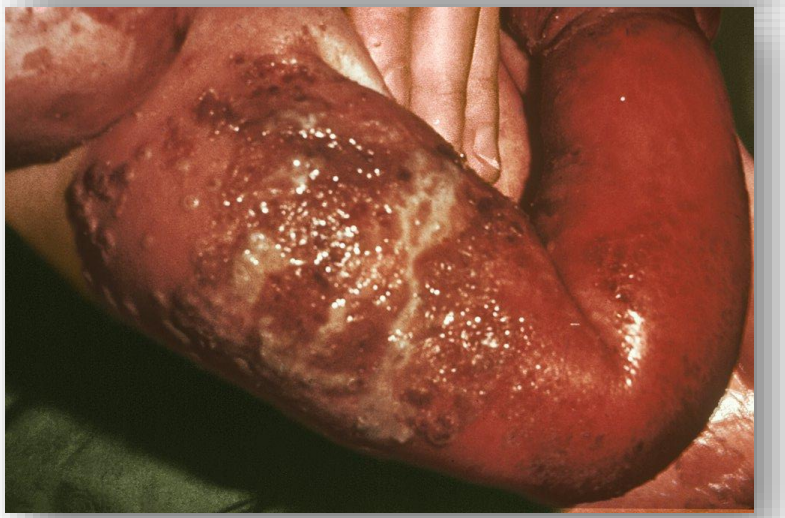
❖Causes of erythema multiforme

- **Most common cause** is herpes simplex
- **Second most common** is mycoplasma infection
- **Medications**: Barbiturates, OCPs, Penicillins, Phenytoin, and Sulfonamides
- **Other causes**: Connective tissue diseases, Pregnancy

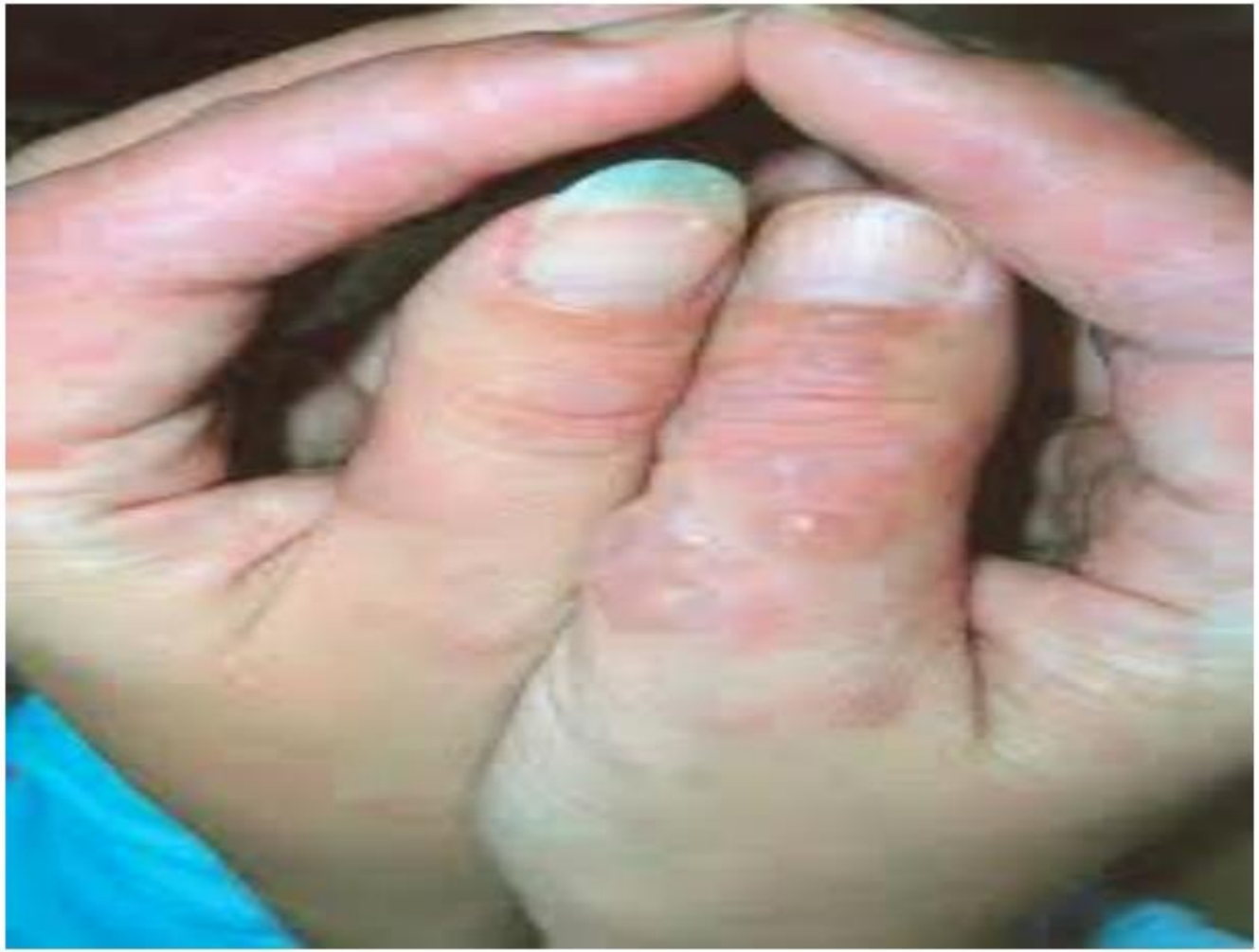
Erythema multiforme

- Erythema multiforme with lip/oral mucosal involvement and fever is termed Stevens-Johnson syndrome
- Stevens-Johnson syndrome typically arises as an adverse drug reaction
- When Stevens-Johnson syndrome involves > 30% of the body surface it is termed toxic epidermal necrolysis (TEN)
- **Treatment**:
  - Treat the primary cause.
  - **Few lesions**: Potent topical steroids + Anti-histamines for 1 week.
  - **Multiple lesions**: Short course of systemic steroids for 1 week.



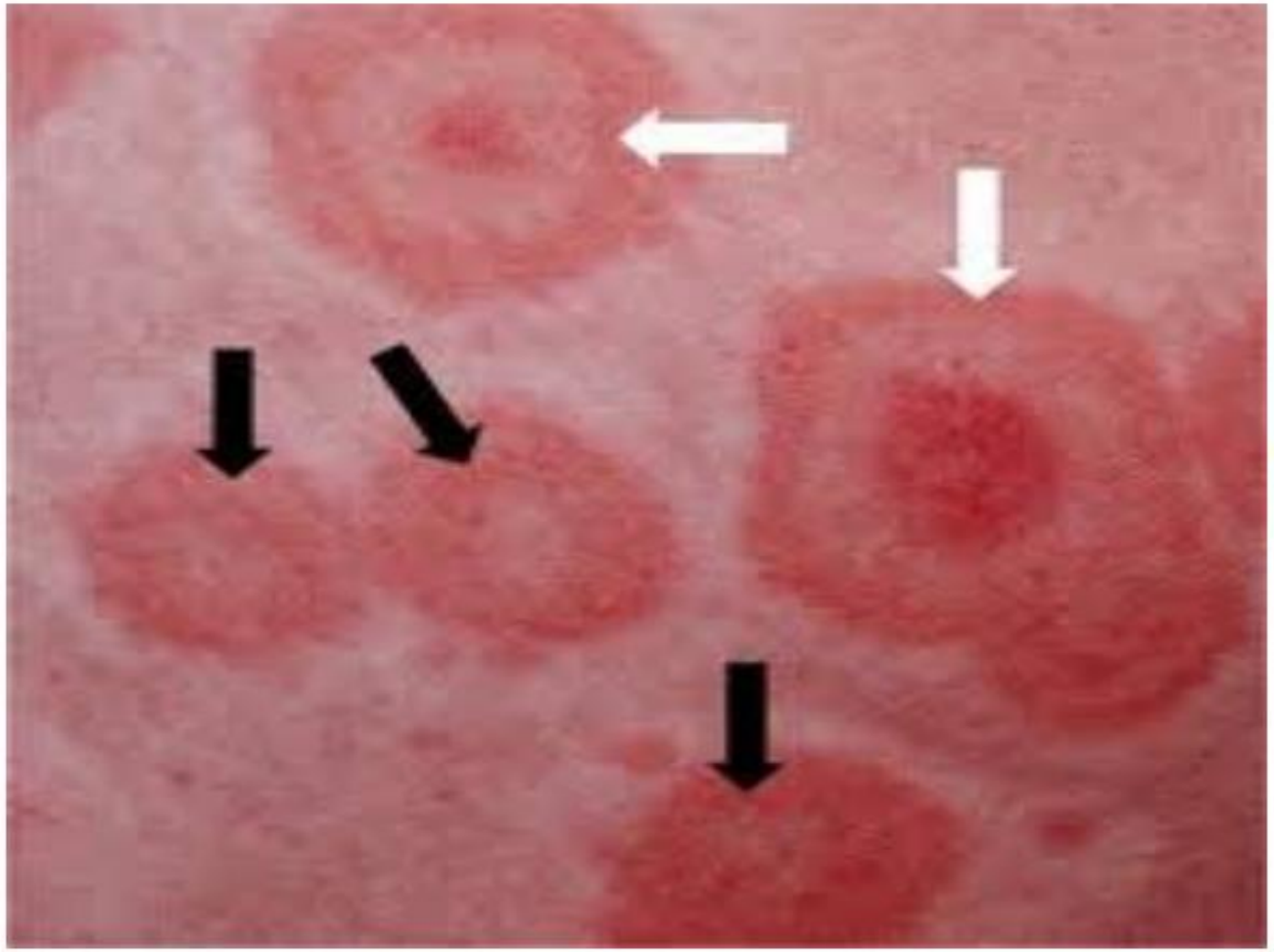


Erythema multiforme



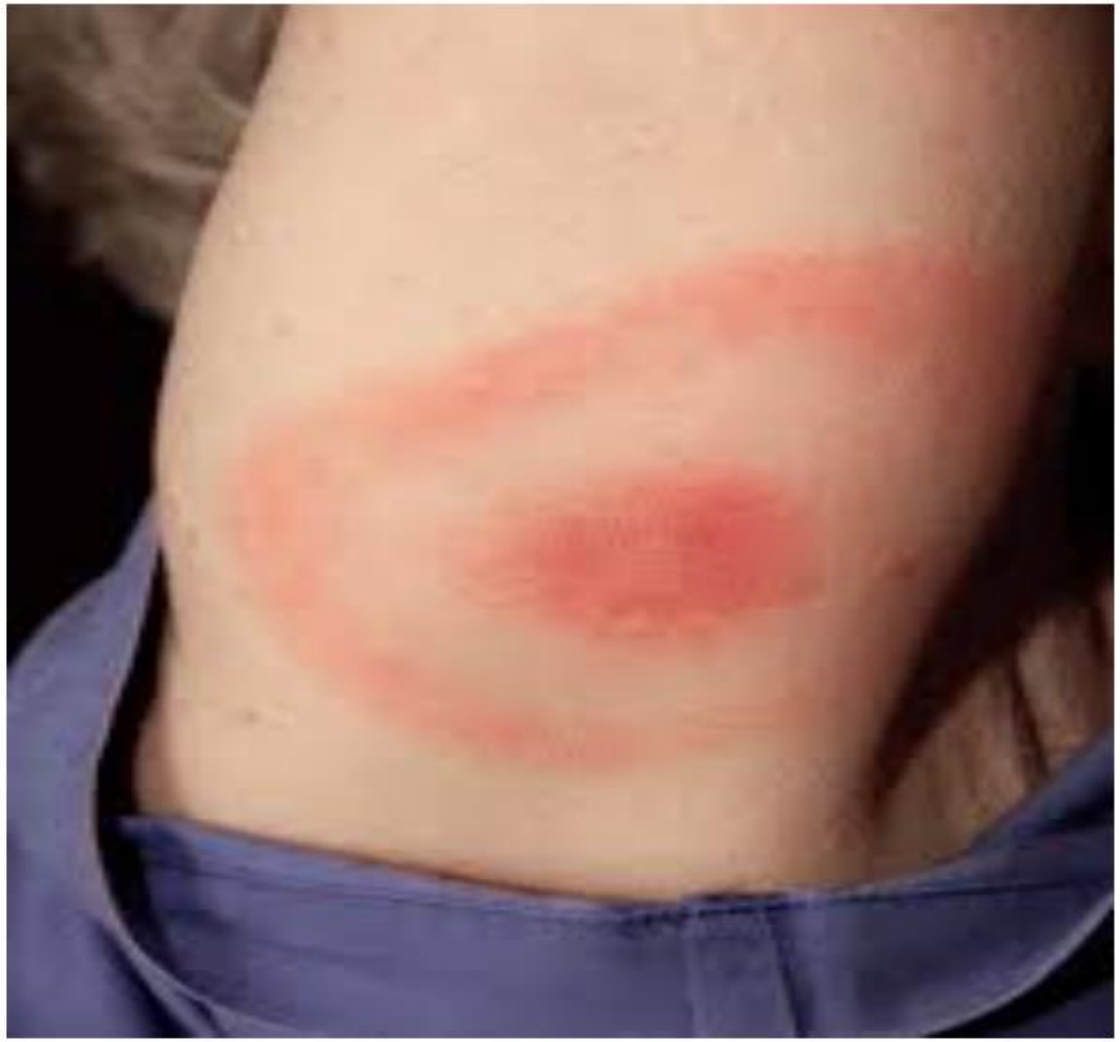
Erythema multiforme

Erythema multiforme



Target lesion of erythema multiforme

Erythema multiforme Vs Erythema marginatum



Lyme disease
Erythema marginatum

Nikolsky's sign

- **Definition**: application of slight lateral pressure to epidermal surface results in the separation of epidermis from its underlying surface.
- **Positive Nikolsky's sign is seen in**:
  - Stevens–Johnson syndrome (SJS)/Toxic Epidermal Necrolysis (TEN)
  - Staphylococcal Scalded Skin Syndrome (SSSS)
  - Pemphigus vulgaris and pemphigus foliaceus
  - Scalding
- **How to differentiate between pemphigus vulgaris and bullous pemphigoid clinically ?**
  - Nikolsky's sign positive in pemphigus vulgaris and negative in bullous pemphigoid

	Pemphigus vulgaris	Bullous pemphigoid	Dermatitis herpetiformis
Peak incidence	40-60 years	> 60 years	15-40 years
Primary lesion	Bulla	Tense subepidermal bulla	Vesicles
Antibodies	IgG antibodies against desmoglein 3	IgG against hemidesmosome components (BP180)	IgA deposits in the tips of the dermal papillae
Clinically	• painful flaccid, intraepidermal blisters • First present on the oral mucosa • Pruritus is typically absent. • Positive Nikolsky's sign	• Large, tense, subepidermal blisters • Oral involvement is rare • Intensely pruritic lesions • Negative Nikolsky's sign	• Tense, grouped subepidermal vesicles • No mucosal involvement • Intense pruritus • Bilateral, symmetrical distribution
Histology	Acantholysis and acantholytic cells "Tombstone" appearance	detachment of the basal cell layer from the basement membrane	neutrophilic abscesses in dermal papillae, dermal infiltrates of neutrophils and eosinophils with subepidermal vesicles
Immunofluore scence	Fish net" (reticular) pattern	Linear pattern	Granular IgA deposits in the tips of the dermal papillae
Serology	Specific anti-dsg1 and anti-dsg3 antibody	pemphigoid BP180 antibodies	Rule out celiac disease
Treatment	• **First-line**: High-dose **systemic** steroids • **Second-line**: Immunosuppression • Topical treatment	• **First-line**: High-dose **topical** steroids • **Second-line**: Systemic glucocorticoids and immunosuppressants	• **First-line:** dapsone • Gluten-free diet • Topical steroids

50 Y/O female patient present with the following lesion on her oral mucosa

سنوات (2)

❖**What disease is this lesion characteristic for ?**

- Pemphigus vulgaris

سنوات (1)

❖**What is the primary lesion of the possible disease** ? Bulla

سنوات (1)

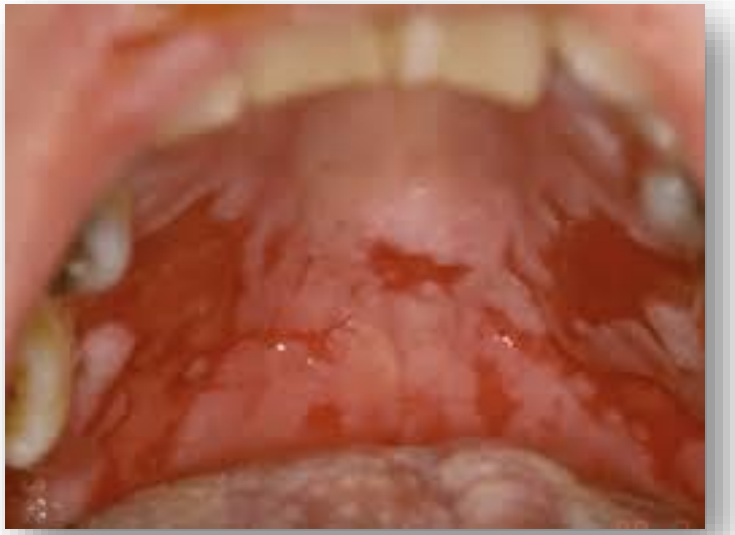
❖**What is the best diagnostic test to confirm the diagnosis ?**

- Direct immunofluorescence staining of perilesional skin biopsy sections

إضافي

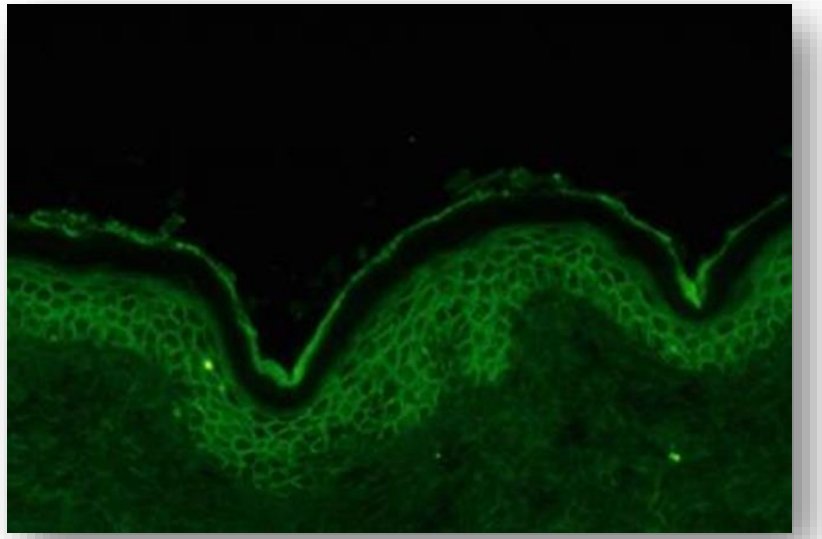
❖**What is the appearance of a perilesional skin biopsy on immunofluorescence**

- "Fish net" (reticular) pattern



Note: Most patients with pemphigus vulgaris first present with lesions on the mucous membranes such as the mouth and genitals

53 Y/O Indian male patient present with oral bullae



سنوات (3) ❖**What is your diagnosis (same photo) ?**

- Pemphigus vulgaris

إضافي ❖**What is the first line of treatment ?**

- High-dose topical steroids

إضافي ❖**What features can be seen histologically ?**

- Acantholysis and acantholytic cells

إضافي ❖**This immunofluorescence test shows antibodies most likely against what ?**

- Desmosomes between keratinocytes (IgG antibodies against desmoglein 3)

إضافي ❖**Serum test of which antibodies is likely to be positive in this patient ?**

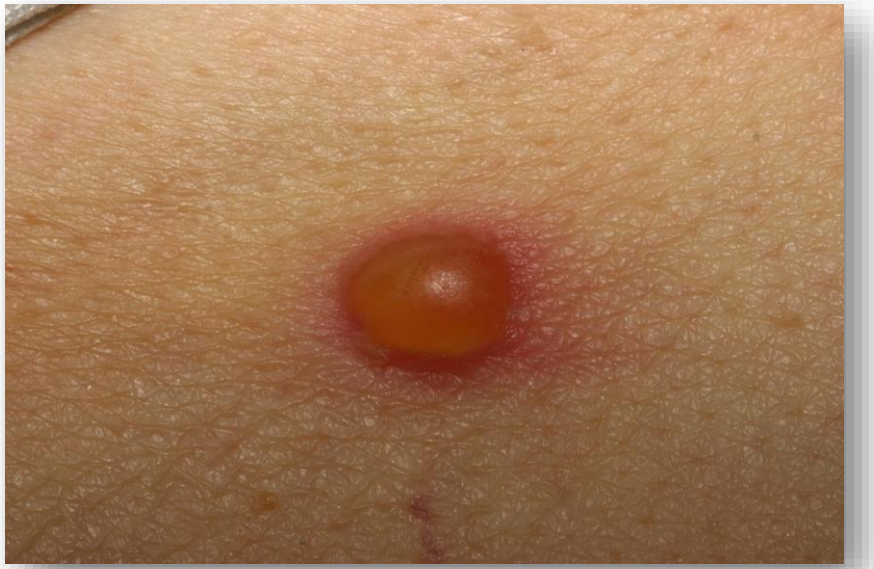
- Specific anti-dsg1 and anti-dsg3 antibody

سنوات (1)

Case w/o pic

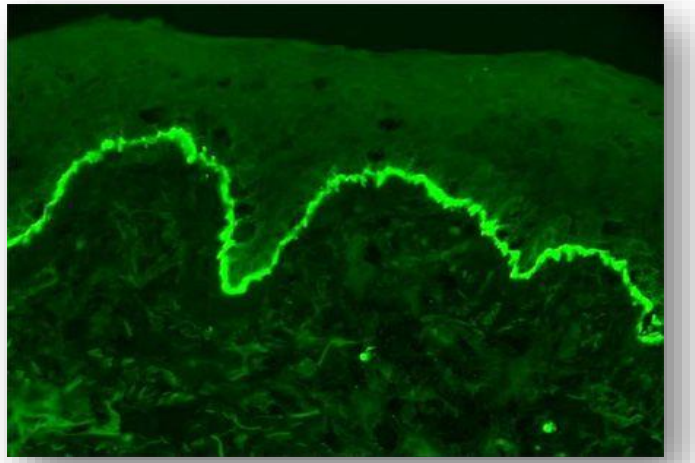
70 Y/O female patient with tense bullae on her body

- **What is your diagnosis ?**
  - Bullous pemphigoid
- **What is the primary lesion of the possible disease** ? Tense subepidermal bulla
- **What is the best diagnostic test to confirm the diagnosis ?**
  - Skin biopsy sections (حسب السلايد)
  - Direct immunofluorescence staining of perilesional skin biopsy sections (حسب رأيي)
- **What is the appearance of a perilesional skin biopsy on immunofluorescence**
  - linear pattern



Picture from Amboss

75 Y/O male patient with multiple cutaneous bullae



- **What is your diagnosis ?**
  - Bullous pemphigoid
- **What is the first line of treatment ?**
  - High-dose systemic steroids
- **What features can be seen histologically ?**
  - Detachment of the basal cell layer from the basement membrane
- **This immunofluorescence test shows antibodies most likely against what ?**
  - IgG antibodies against hemidesmosome components (BP180)
- **Serum test of which antibodies is likely to be positive in this patient ?**
  - Pemphigoid BP180 antibodies

تجميعة سنوات

سنوات (1)

Case w/o pic

Female with celiac disease present with vesicles symmetrically located on elbow and knee

سنوات (2)

❖**What is your diagnosis ?**

- Dermatitis herpetiformis

سنوات (1)

❖**What is the primary lesion ?**

- Vesicles

إضافي

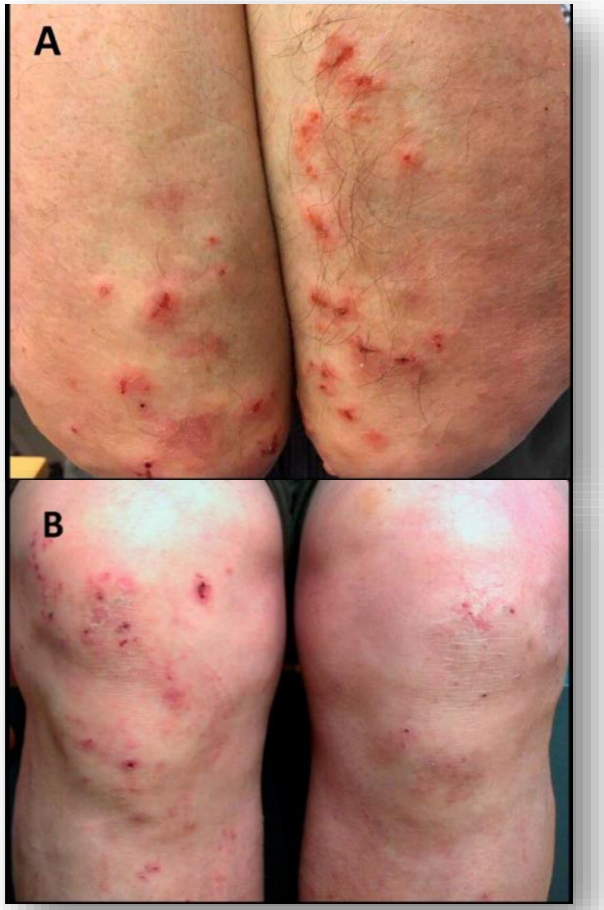
❖**First line treatment ?**

- First-line: Gluten-free diet + Dapsone

إضافي

❖**Appearance on immunofluorescence test**

- Granular IgA deposits in the tips of the dermal papillae



20 Y/O male diabetic patient present with itchy bilateral vesicles

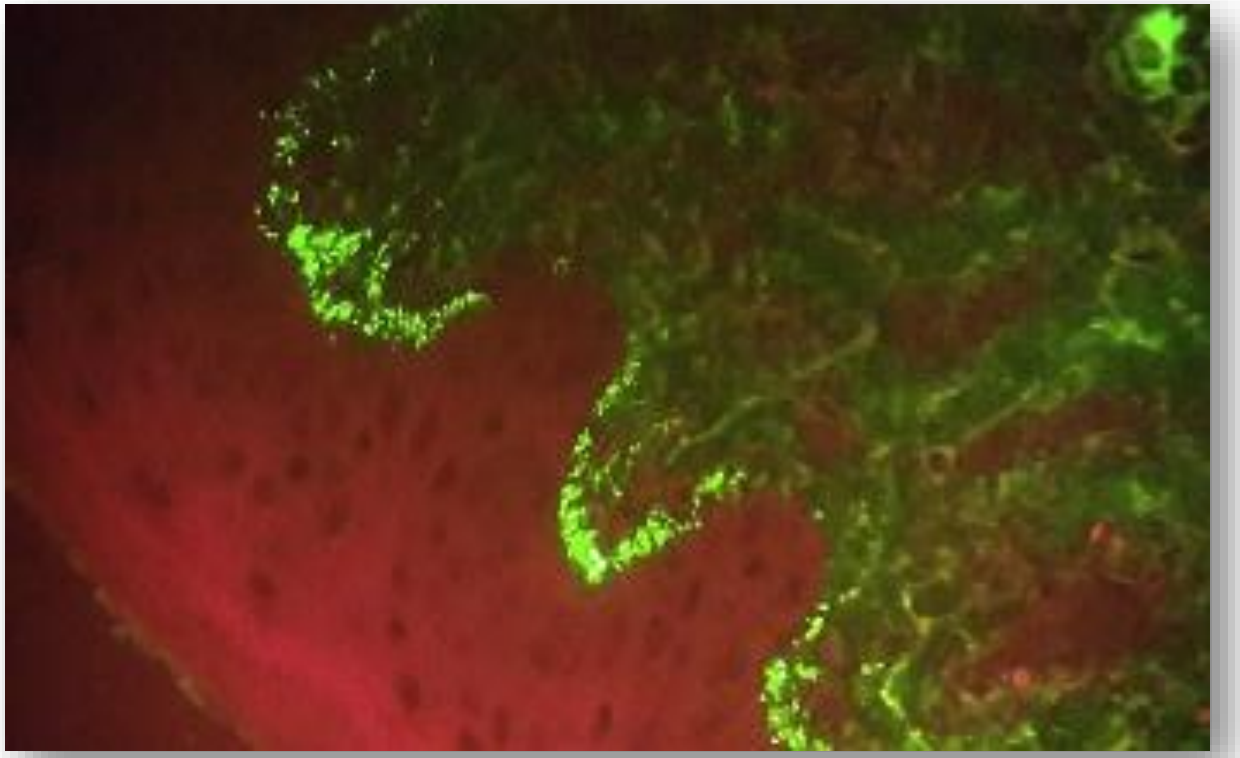
❖ **Describe what you see**

- Granular IgA deposits in the tips of the dermal papillae

سنوات (1)

❖ **What is your diagnosis ?**

- Dermatitis herpetiformis



Urticaria

شرح

Urticaria

❖**Definition**: Urticaria is characterized by wheals (hives) or angioedema (swellings, in 10%) or both (in 40%).

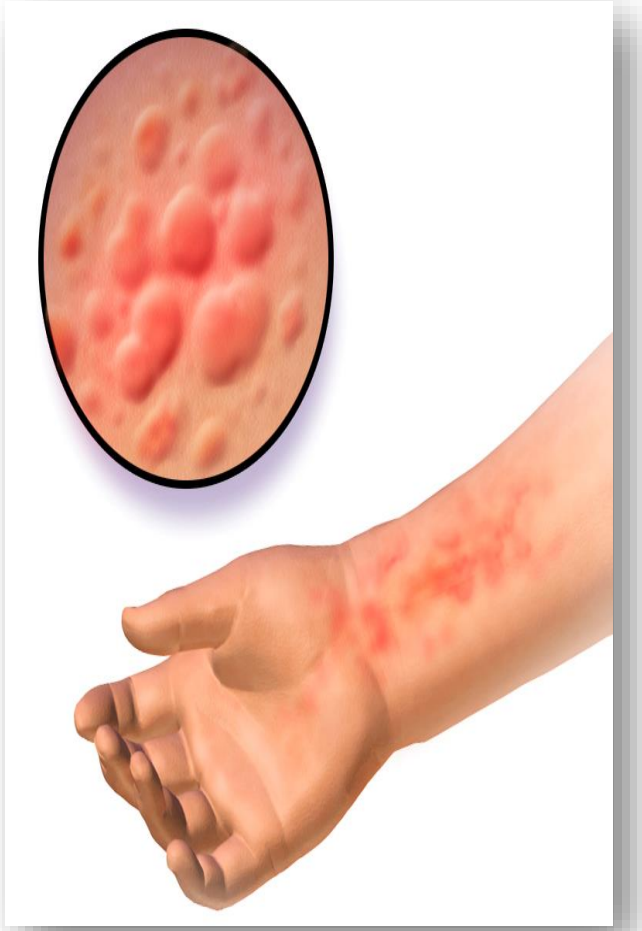
❖**Primary lesion of**

(4) سنوات
- **Urticaria**: Wheal or hives

(1) سنوات
- **Insect bite**: bulla or wheal

❖**Define wheal**: Superficial skin -colored or pale skin swelling, usually surrounded by erythema (redness) that lasts from a few minutes to 24 hours. Usually very itchy, it may have a burning sensation

❖**What are the characteristic clinical features of wheal ?**
- Linear in symptomatic dermographism.
- Tiny in cholinergic urticaria.
- Confined to contact areas in contact urticaria.
- Diffuse in cold urticaria.



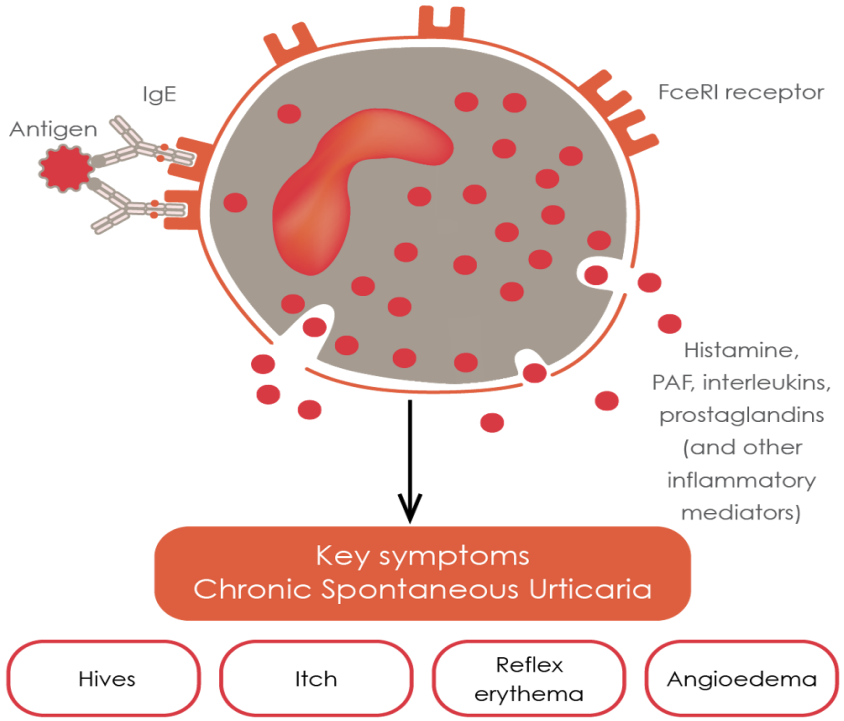
Urticaria

- **Define angioedema**: Deeper swelling within the skin or mucous membranes and can be skin-coloured or red. It resolves within 72 hours. Angioedema may be itchy or painful and sometimes need IV adrenaline but is often asymptomatic.
- **Who gets urticaria ?**
  - One in five children or adults has an episode of acute urticaria during their lifetime (25%).
  - It is more common in atopics.
  - Chronic spontaneous urticaria affects 0.5–2% of the population.
  - There are genetic and autoimmune associations.

Angioedema

Urticaria

- Wheal are due to release of chemical mediators from tissue mast cells and circulating basophils.
- These chemical mediators include
  - Histamine, platelet-activating factor and cytokines.
- The mediators activate sensory nerves and cause dilation of blood vessels and leakage of fluid into surrounding tissues.
- Bradykinin release causes angioedema.



شرح

Urticaria

- Classification (according to duration)
  - Acute urticaria (< 6 weeks duration, and often gone within hours to days)
  - Chronic urticaria (> 6 weeks duration, with daily or episodic wheals)
    - Chronic urticaria may be spontaneous or inducible

- **Mention 4 types of inducible (physical) urticaria:** سنوات (3)

1. Symptomatic dermographism
2. Cold urticaria
3. Cholinergic urticaria
4. Contact urticaria
5. Delayed pressure urticaria.
6. Solar urticaria.
7. Aquagenic urticaria.

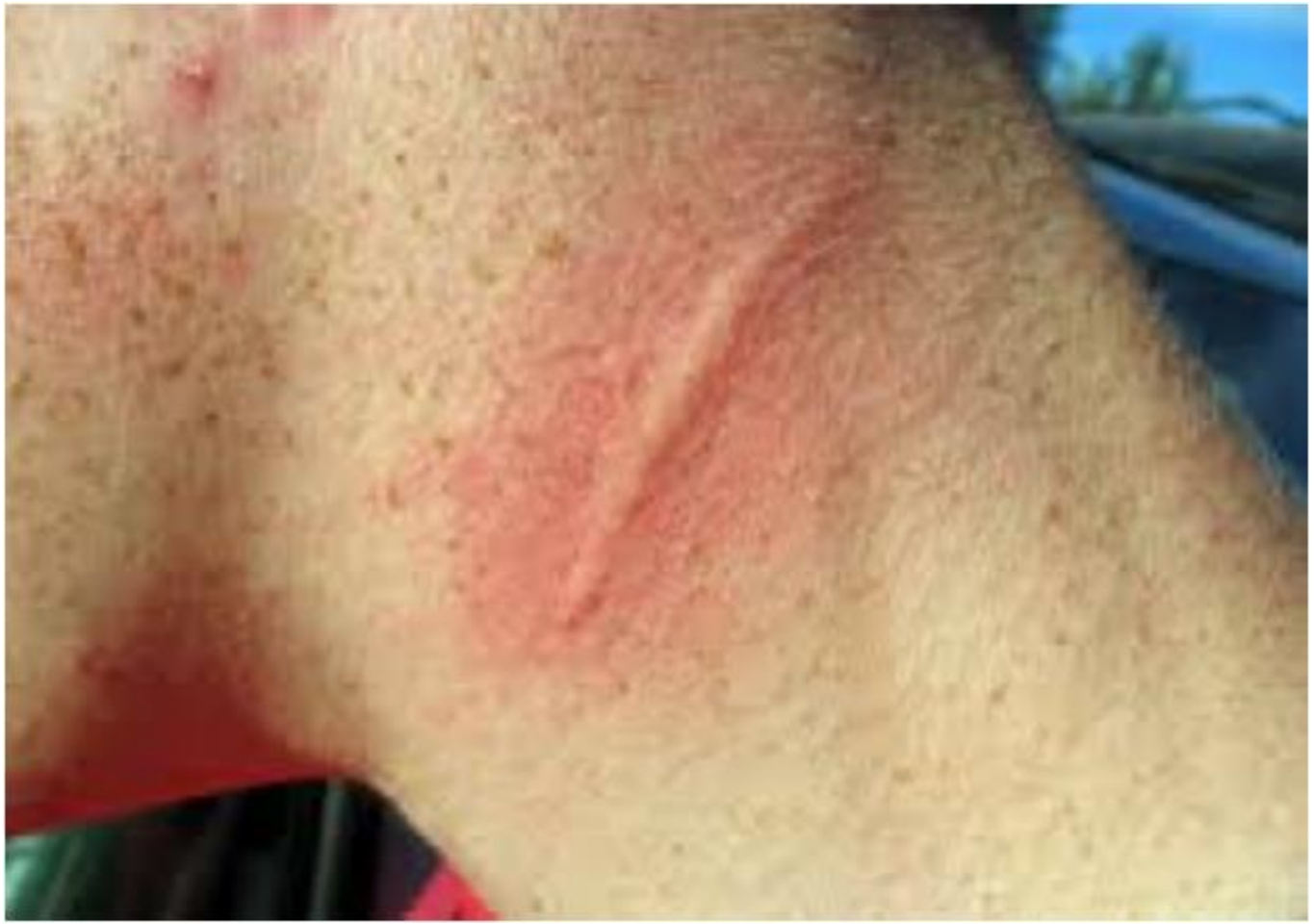
Inducible urticaria is a response to a physical stimulus.

Types of inducible

1. Symptomatic dermographism
   - The most common form of physical urticaria with a prevalence of up to 5%
   - characterized by itchy wheals that occur in response to friction, for example, after rubbing or scratching of the skin, and usually last for 1 to 2 hours

2. Cold urticaria
   - is a disorder where hives (urticaria) form on the skin after exposure to a cold stimulus.

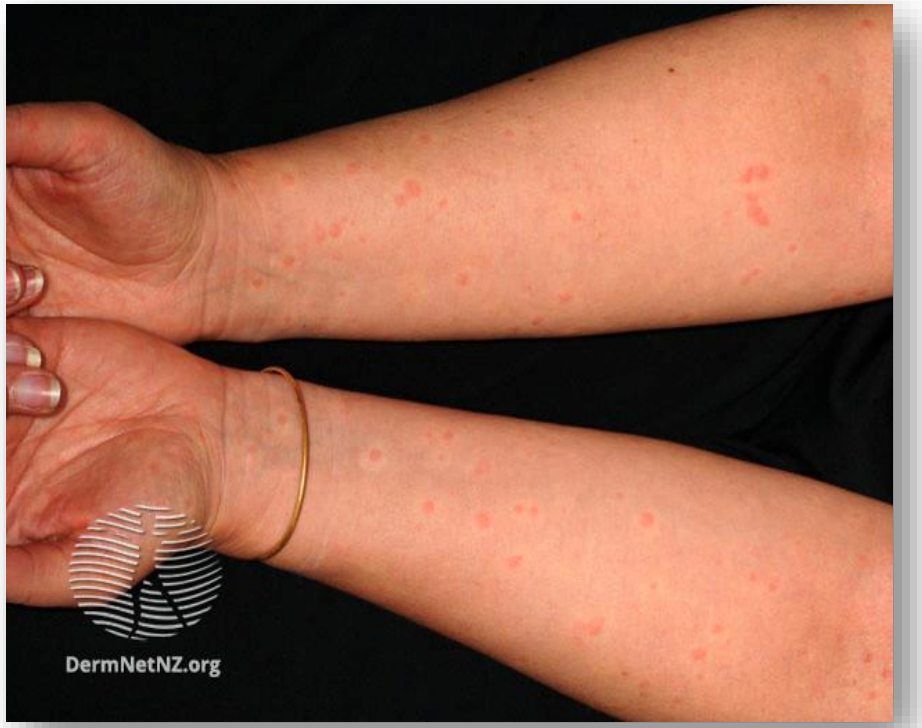
Types of inducible – Dermographism



Dermographism :
exaggerated whealing tendency

Types of inducible – 3. Cholinergic urticaria

- Common chronic inducible urticaria that is caused by **sweating**
- It is sometimes referred to as **heat bumps**
- **Presents with** very small (1–4 mm) wheals surrounded by bright red flares
- **Common triggers include**:
  - Exercise, Hot baths/showers, Fever, Occlusive dressings, Eating spicy food, Emotional stress.
- Can be associated with other types of inducible urticaria such as dermographism or pressure urticaria
- Treated by Anti-histamines.

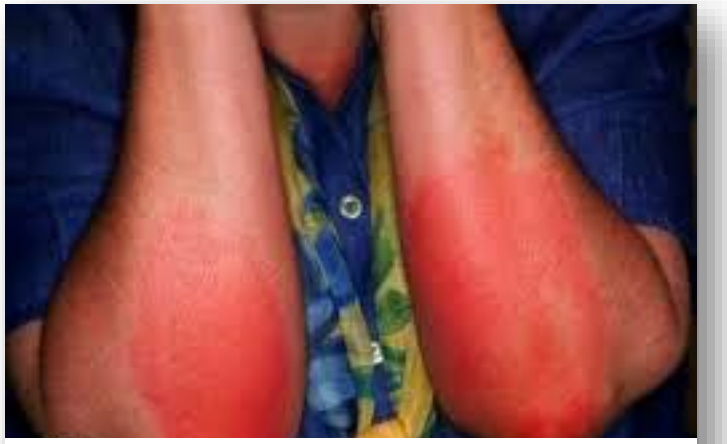


Types of inducible – 4. Contact urticaria

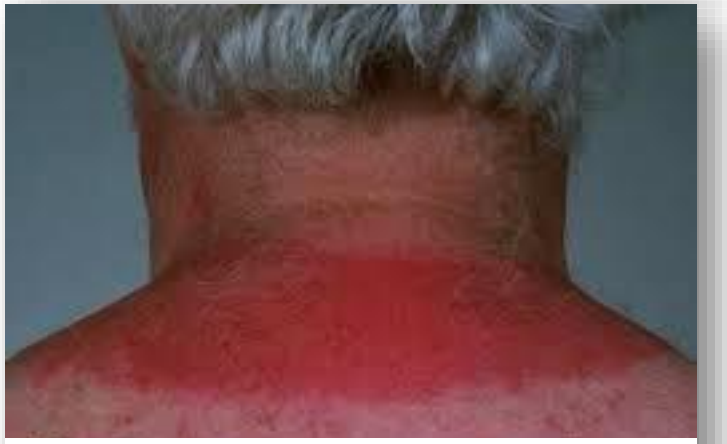
- An immediate but transient localized swelling and redness that occurs on the skin after direct contact with an offending substance.
- Should be distinguished from **contact dermatitis** where a dermatitis reaction develops hours to days after contact with the offending agent.

Types of inducible

5. **Delayed Pressure Urticaria**
   - A physical urticaria where erythematous, often painful swellings occur at sites of sustained pressure on the skin, after a delay of several hours.
   - It is present in up to 40% of patients with ordinary chronic “idiopathic urticaria ” to a varying degree.
6. **Solar urticaria**
   - A rare condition due to exposure to ultraviolet radiation, or sometimes even visible light.
   - Hives that can appear in both covered and uncovered areas of the skin.
7. **Aquagenic urticaria**:
   - Very rare.
   - Itchy rash from contact with water.



Delayed Pressure Urticaria



Solar urticaria

Inducible urticaria is a response to a physical stimulus

Type of inducible urticaria	Examples of stimuli inducing wealing
Symptomatic dermographism	• Stroking or scratching the skin • Tight clothing • Towel drying after a hot shower
Cold urticaria	• Cold air on exposed skin • Cold water • Ice block • Cryotherapy
Cholinergic urticaria	• Sweat induced by exercise • Sweat induced by emotional upset • Hot shower
Contact urticaria	• Eliciting substance absorbed through the skin or mucous membrane • Allergens (IgE-mediated): white flour, cosmetics, textiles, latex, saliva, meat, fish, vegetables • Pseudoallergens or irritants: stinging nettle, hairy caterpillar, medicines

Inducible urticaria is a response to a physical stimulus

Type of inducible urticaria	Examples of stimuli inducing wealing
Delayed pressure urticaria	• Pressure on affected skin several hours earlier • Carrying heavy bag • Pressure from a seat belt • Standing on a ladder rung • Sitting on a horse
Solar urticaria	• Sun exposure to non-habituated body sites • Often spare face, neck, hands • May involve long wavelength UV or visible light
Heat urticaria	• Hot water bottle • Hot drink
Vibratory urticaria	• Jackhammer
Aquagenic urticaria	• Hot or cold water • Fresh, salt or chlorinated water

Acute urticaria

❖ What are the Causes of acute urticaria

1. Acute viral infection, such as upper respiratory infection
2. Acute bacterial infection, such as dental abscess, sinusitis
3. Food allergy (IgE mediated), such as usually milk, egg, peanuts shellfish
4. Drug allergy (IgE mediated drug-induced urticaria) often an antibiotic
5. Drug-induced urticaria due to pseudo-allergy, such as aspirin, nonselective NSAIDS, radiocontrast media; these cause urticaria without immune activation
6. Vaccinations
7. Bee or wasp stings
8. A single episode or recurrent episodes of angioedema without urticaria can be due to an angiotensin-converting enzyme (ACE) inhibitor drug

- Severe allergic urticaria may lead to anaphylactic shock

Chronic urticaria

- **Chronic inducible urticaria**: Characterized by the recurrence of itchy wheals and/or angioedema that lasts more than 6 weeks and is induced by specific physical or environmental stimuli. Wheals appear about 5 minutes after the stimulus and last a few minutes or up to one hour
- **Chronic spontaneous urticaria**: The presence of urticaria (hives) on most days of the week, that lasts more than 6 weeks
- **Chronic spontaneous urticaria causes**
  - Mainly idiopathic (cause unknown)
  - Could be due to drugs and food
  - An autoimmune cause is likely
  - Chronic underlying infection, such as Helicobacter pylori, bowel parasites and chronic autoimmune diseases.

Urticaria – Diagnosis

- ❖ History and physical (أهم شيء)
- ❖ Skin prick test and RAST (radioallergosorbent tests) or CAP fluoroimmunoassay may be requested if a drug or food allergy is suspected
- ➢ Inducible urticaria is often confirmed by inducing the reaction
- ➢ Investigations for a systemic condition should be undertaken in urticaria patients with fever, joint or bone pain, and malaise
- ➢ Patients with angioedema without wheals should be asked if they take ACE inhibitor drugs and tested for complement C4; C1-INH levels, function and antibodies; and C1q
- ➢ Routine diagnostic tests in chronic spontaneous urticaria
  - CBC, CRP, ESR, LVT, KFT, Food allergy test, Prick test, Urinalysis, Stool analysis, *H.pylori* test, ANA

Urticaria – Treatment

❖**First-line treatment in acute urticaria**:

- Oral 2nd generation anti-histamine, until the urticaria settle down

❖**Why are 1st generation not used anymore ?**

1. They are short-lasting (2 hours).
2. They have sedative and anticholinergic side effects.
3. They impair sleep, learning and performance.
4. They cause drowsiness in nursing infants if taken by the mother.

❖**Treatment of acute refractory urticaria (refractory to anti-histamine)**

- 4-5 days course of oral prednisolone in severe acute urticaria

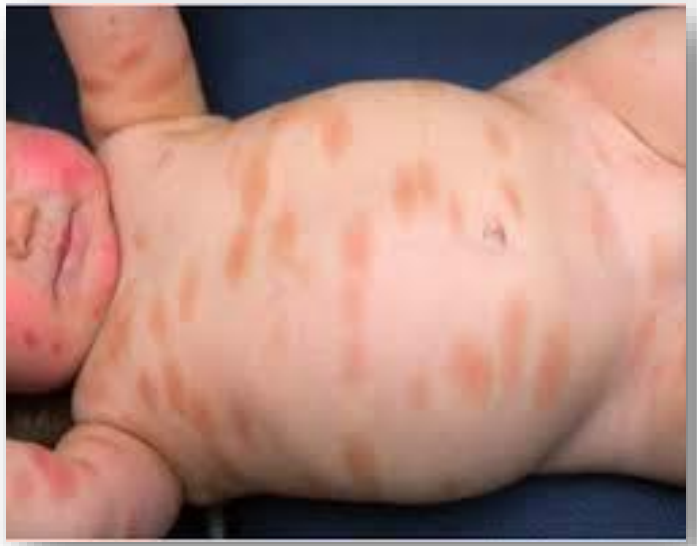
❖**Intramuscular injection of adrenaline (epinephrine) is reserved for**

- Life-threatening anaphylaxis
- Swelling of the throat (angioedema).

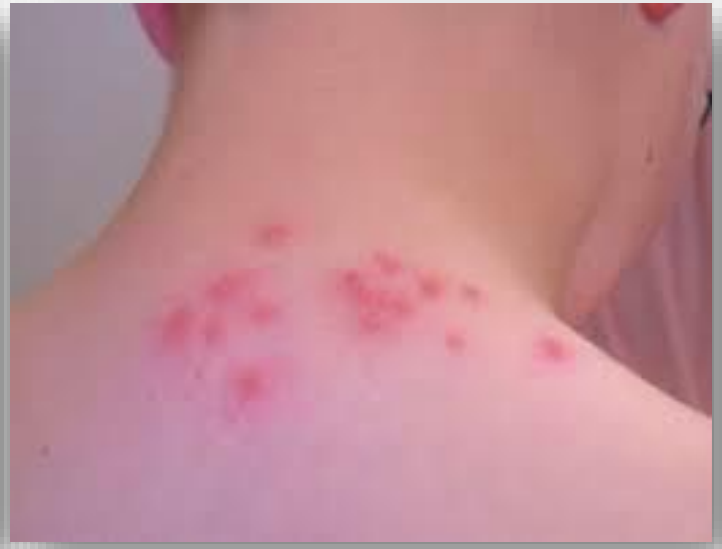
Differential diagnosis of urticaria

1. **Mastocytosis**
   - Maculopapular cutaneous mastocytosis (urticaria pigmentosa) is the most common form of mastocytosis
   - Itchy brown patches or freckles on the skin are due to abnormal collections of mast cells.
2. **Papular urticaria**
   - Insect bites are localized, often clustered in groups of 3–5 lesions, and they appear in crops. Bites persist for days. Close inspection reveals a central punctum
   - Chronic hypersensitivity to insect bites is often called papular urticaria
3. **Urticarial vasculitis**
   - Urticarial vasculitis causes persistent urticaria-like plaques that last more than 24 hours and resolve with bruising.
   - Biopsy reveals Leukocytoclastic vasculitis, connective tissue diseases have to be ruled out.
   - **Causes include** SLE, Rheumatoid arthritis, Systemic vasculitis syndromes, Cancer, reaction to drugs, infection or viruses, and glandular issues

Differential diagnosis of urticaria



Mastocytosis



Papular urticaria

Urticarial vasculitis

Urticarial vasculitis:

Painful

Persist >24 hours

VS

Other urticaria:

Painless

Less than 24 hours

Urticaria

سنوات (2)

❖ **What is the diagnosis of the pictures?**

- Cholinergic urticaria

سنوات (1)

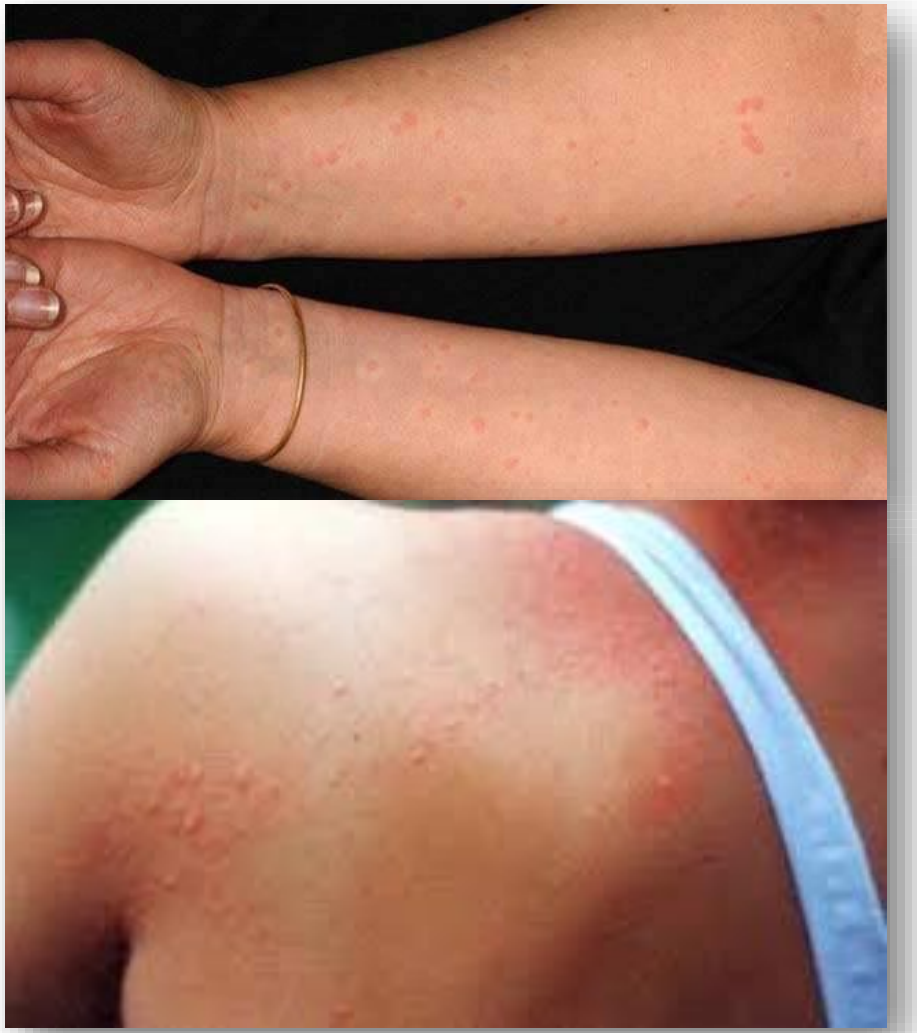
❖ **What is the first line treatment ?**

- 2nd generation anti-histamine

سنوات (2)

❖ **Mention 2 uses of oral steroids in urticaria:**

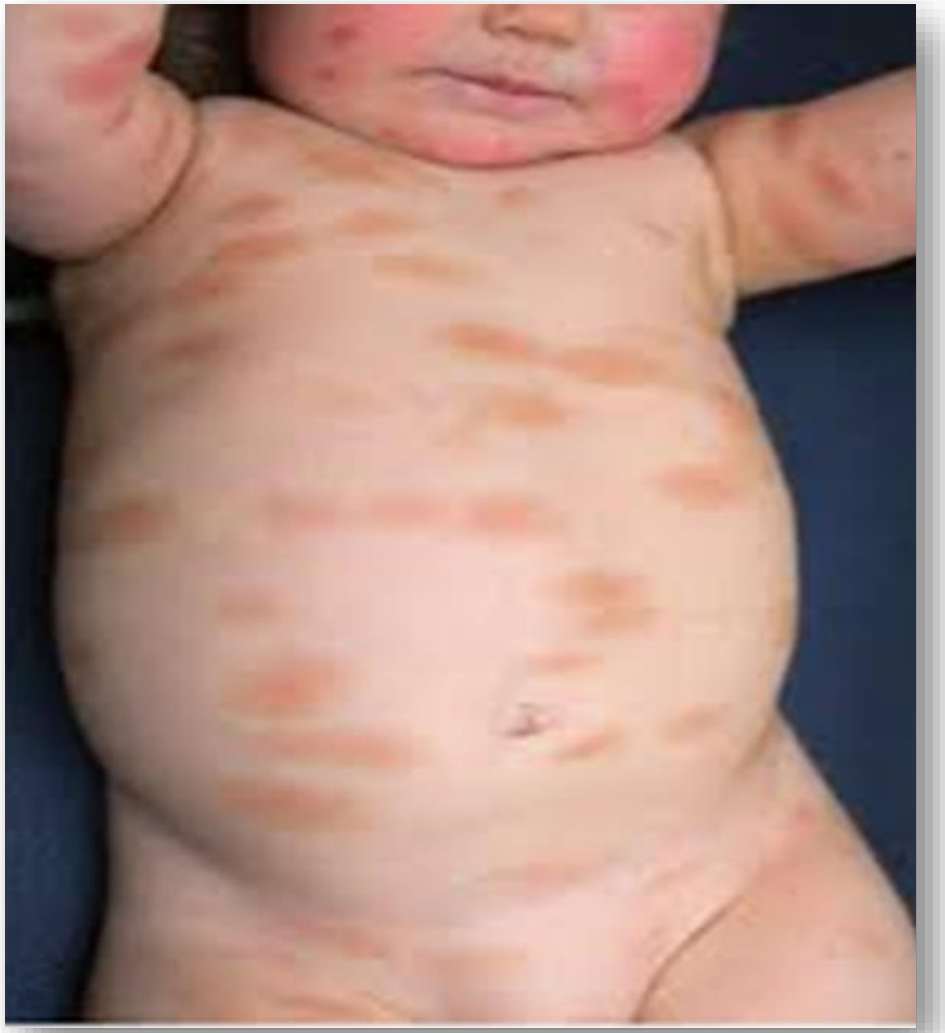
1. Severe acute urticaria
2. Vasculitic urticaria



Male infant present with itchy brown patch

سنوات (1)

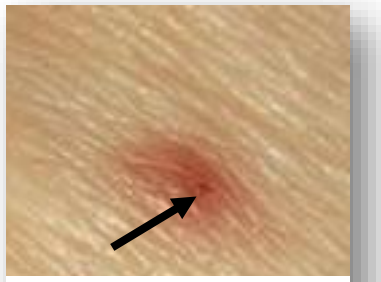
- **What is the diagnosis ?**
  - Mastocytosis
- **Describe what do you see**
  - Itchy brown patches on the skin that are due to abnormal collections of mast cells



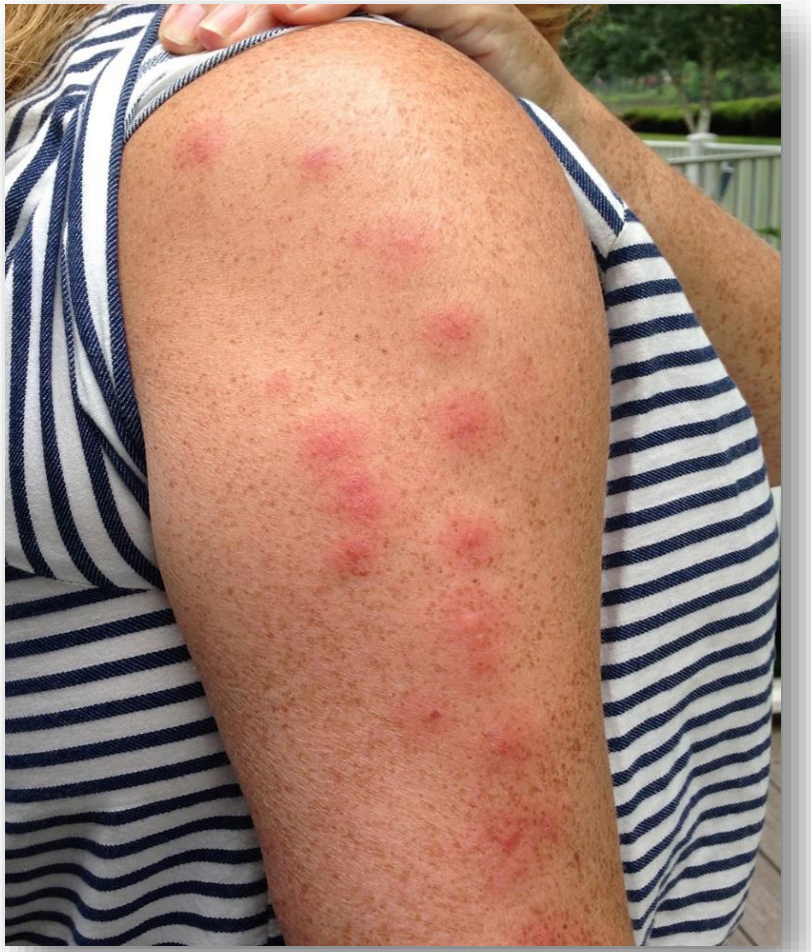
Insect's bite

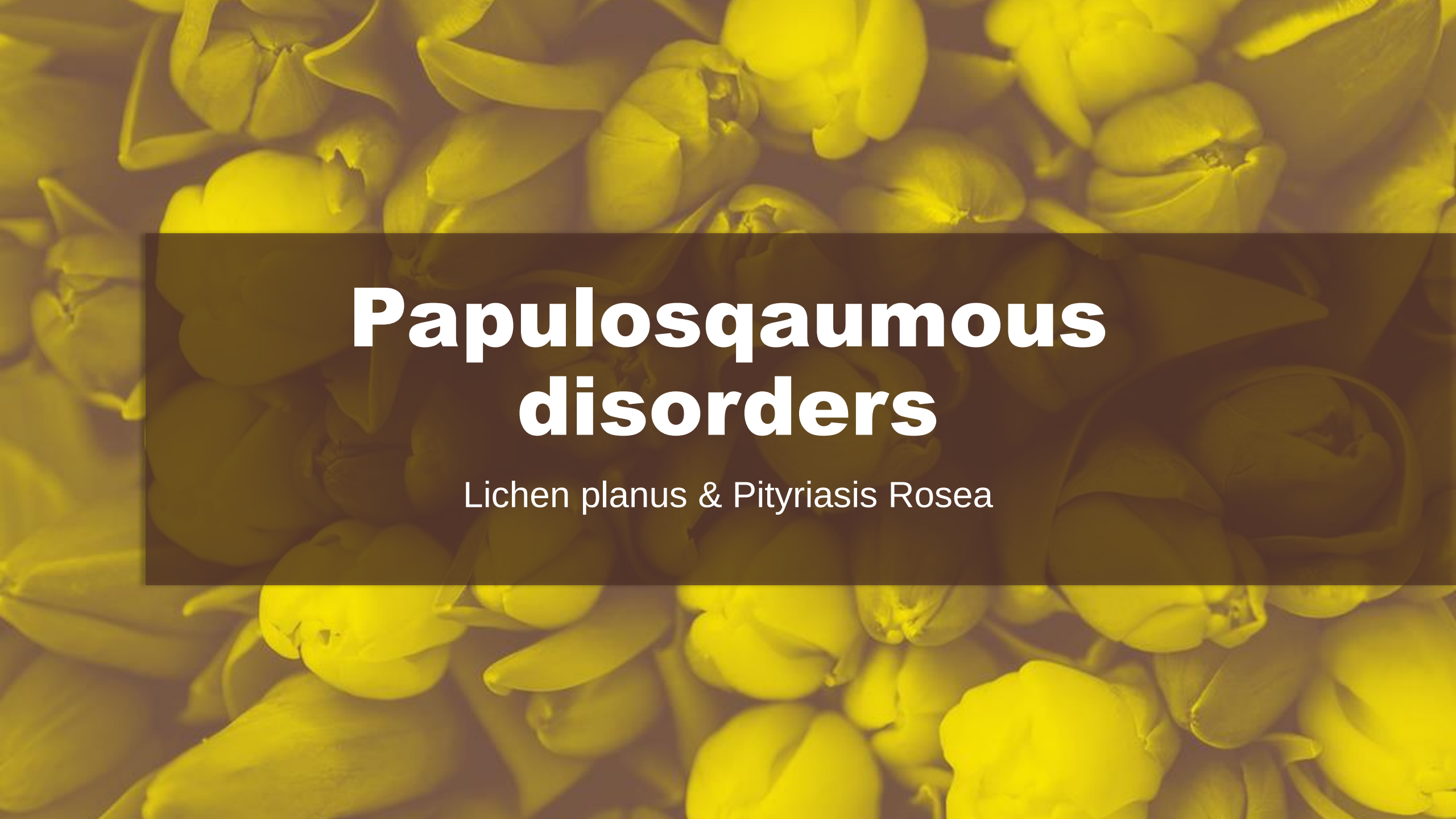
سنوات (1)

- **What is the diagnosis ?**
  - Papular urticaria
- **What is the likely cause of this lesion ?**
  - Chronic hypersensitivity to insect bites
- **Close inspection of these lesion can show what characteristic finding ?**
  - central punctum



Central punctum





Papulosqaumous disorders

Lichen planus & Pityriasis Rosea

Lichen planus

حزاز مسطح

شرح

Lichen planus

- **Lichen planus**: Idiopathic inflammatory autoimmune disease of skin, nails, hair and mucous membrane in middle aged adults with characteristic clinical and histopathological features.

سنوات (1)

- **The primary lesion of lichen planus**: Papules

سنوات (1)

- **Mention 2 systemic disorders associated with lichen planus**
  1. Chronic hepatitis C infection
  2. Diabetes

- **Clinical features**: (Mention 1 characteristic presenting symptom of LP) سنوات (1)

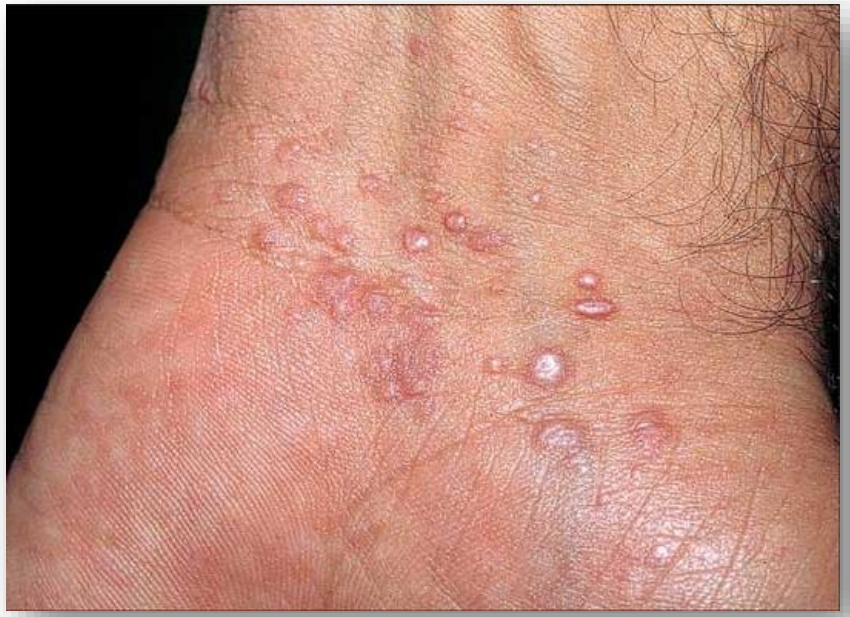
سنوات (1)

  - Small polygonal flat-topped violaceous papules and plaques with shiny surface
  - Wickham`s striae on the surface (fine white lines)
  - Pruritis
  - Koebner phenomenon (also occurs in Psoriasis and Vitiligo)
  - No excoriation (patients rub their skin instead of scratching it)

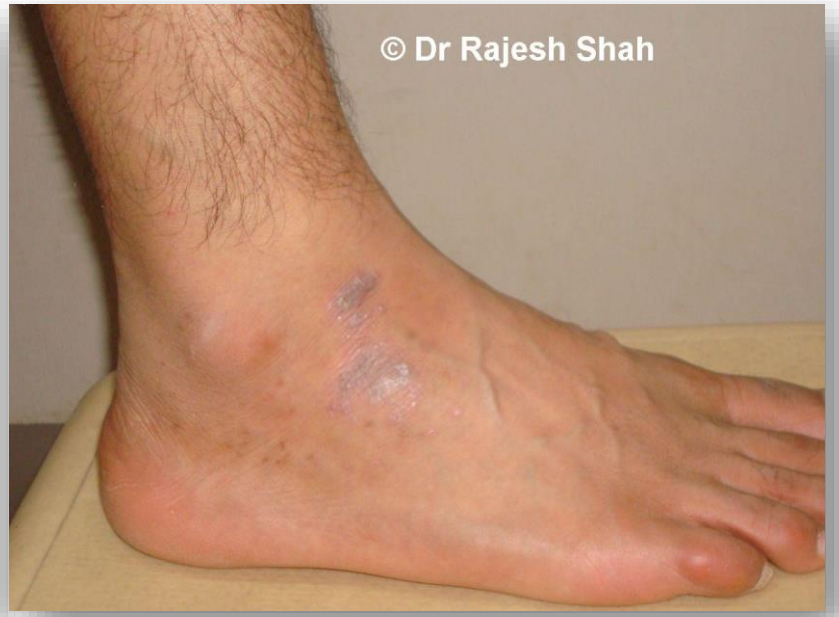
سنوات (1)

  - **Site**: Flexural surface of wrist , Forearm , Anterior leg , Neck , Presacral area and Glans penis

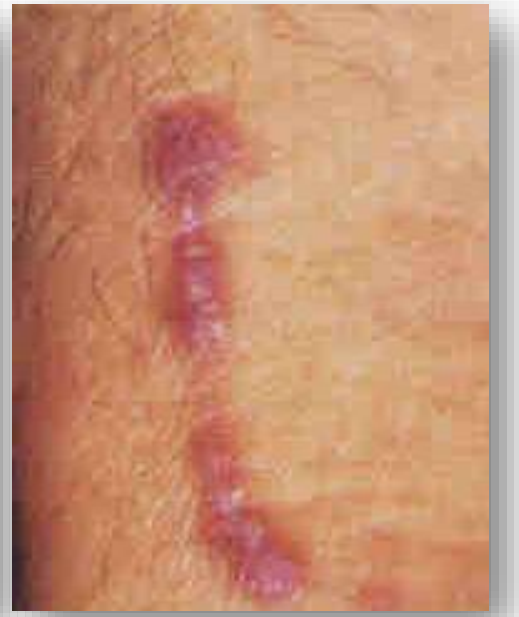
Lichen planus



Shiny violaceous flat topped polygonal papules and plaques on the wrist



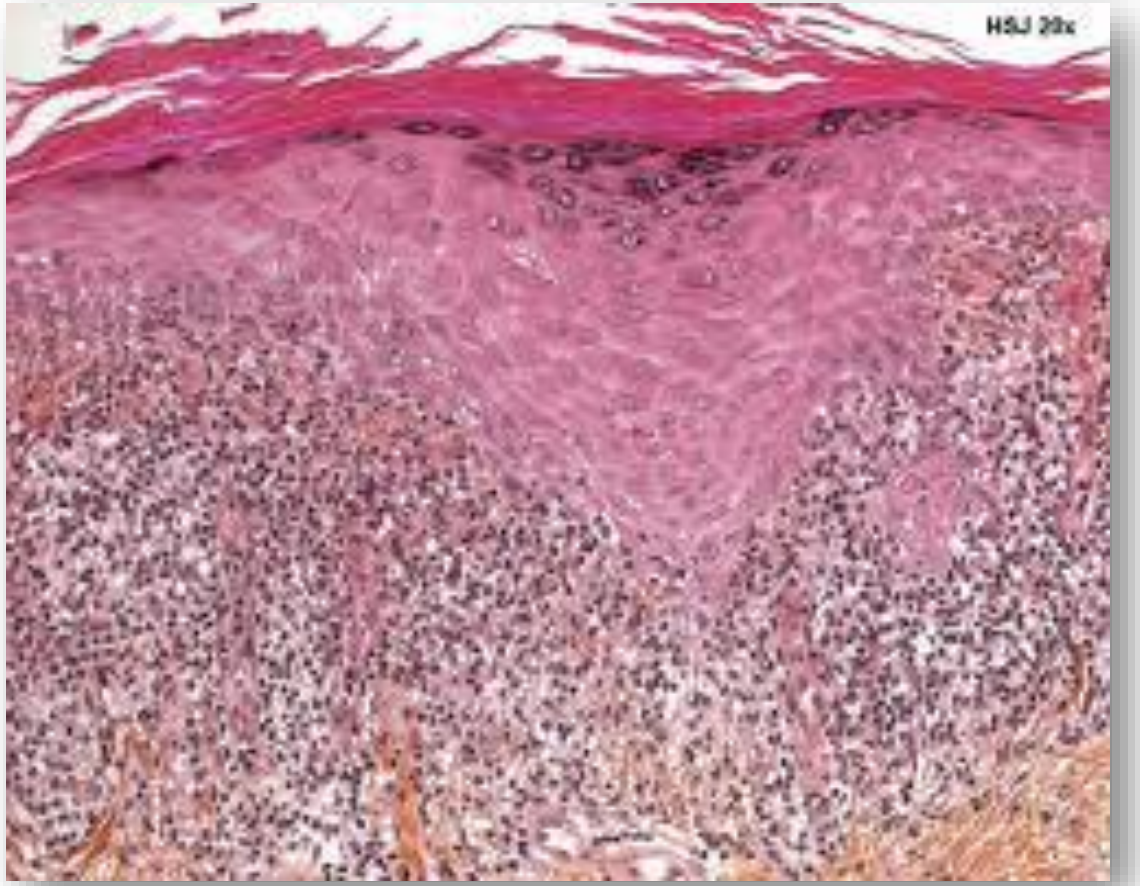
Shiny violaceous flat topped polygonal papules and plaques on the flexural areas



Koebner’s phenomenon

Histopathological changes in all types of lichen planus

1. Hyperkeratosis.
2. Hypergranulosis.
3. Epidermal hyperplasia.
4. Lichenoid inflammatory cell infiltrate.

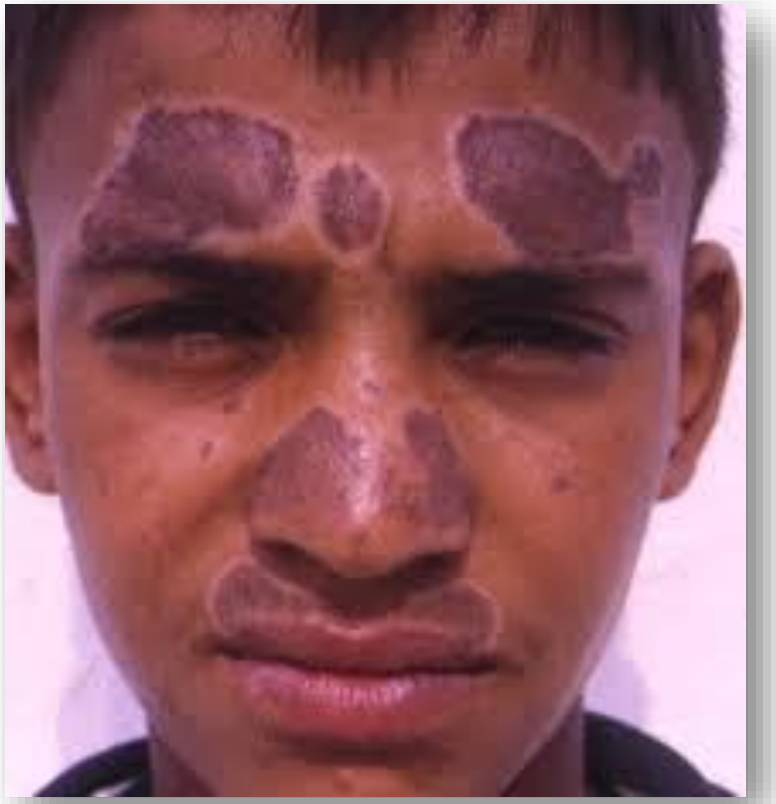


Mention 3 Lichen planus variants

1. Actinic Lichen planus
2. Acute lichen planus
3. Annular lichen planus
4. Atrophic lichen planus
5. Bullous lichen planus (Lichen planus pemphigoides)
6. Hypertrophic lichen planus (Verrucous lichen planus)
7. Inverse lichen planus
8. Linear lichen planus
9. Lichen planus pigmentosus
10. Lichen plano-pilaris (Lichen planus acuminatus)
11. Lichen planus – lupus erythematosus overlap syndrome
12. Ulcerative lichen planus
13. Vulvovaginal lichen planus
14. Lichenoid drug eruption
15. Oral lichen planus
16. Nail lichen planus

1. Actinic lichen planus

- **Description**: Red-brown annular plaques on sun exposed areas (actinic means sun radiation).
- **Site**: Common site forehead and face.
- Common in middle East.
- Onset during summer and spring.
- Young adults and children.

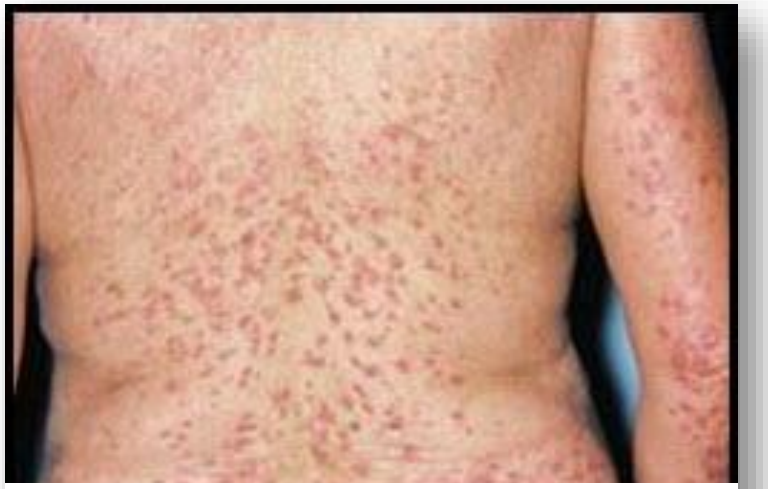


Annular dark brown lesions with rim of hypopigmentation on the forehead and face.

2. Acute lichen planus & 3. Annular lichen planus

2. Acute lichen planus

- **Description**: Exanthematous or eruptive LP.
- **Site**: Trunk, wrist, feet.
- Rapidly disseminate.
- Self-limited course within 3-9 months.

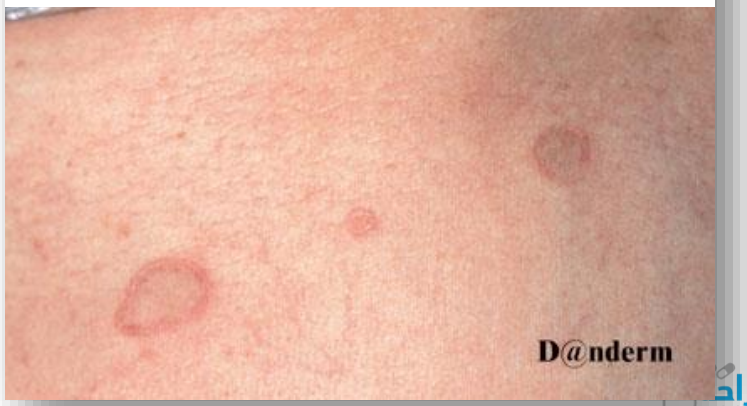


Multiple eruptive widespread lesions

3. Annular lichen planus

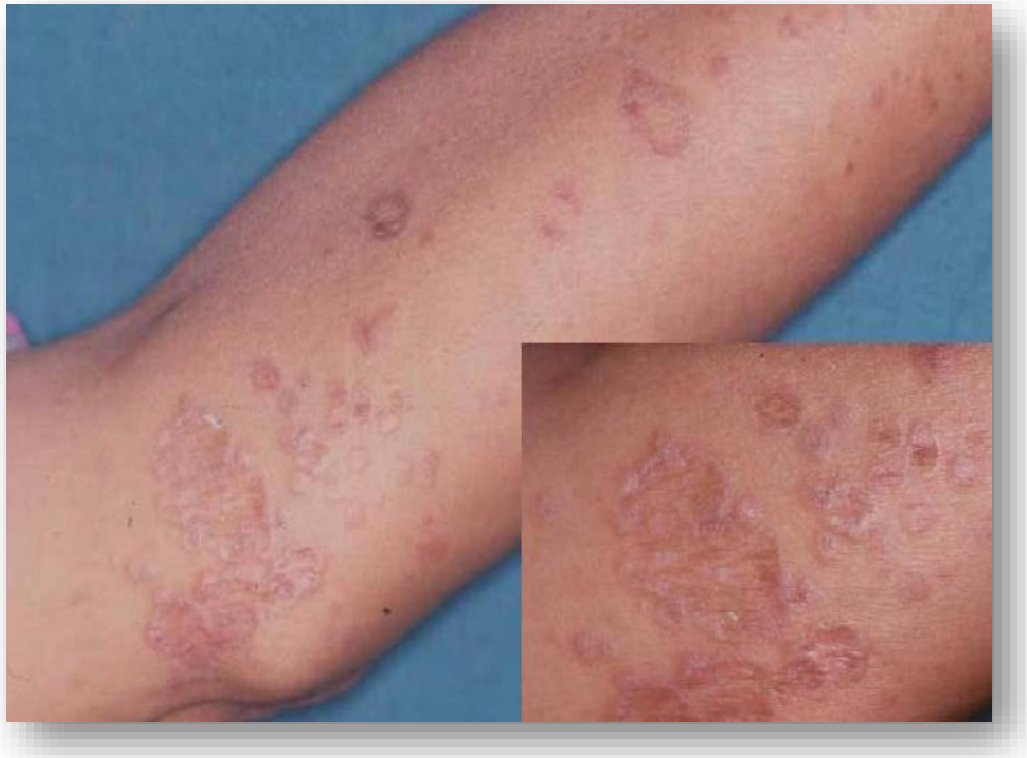
- **Description**: Annular scaly plaques with hyperpigmented center
- **Site**: Axillae and male genitalia (penis)
- Most patients are asymptomatic, but some have pruritus.

Annular lesions with Clear center and erythema at the periphery.



4. Atrophic lichen planus

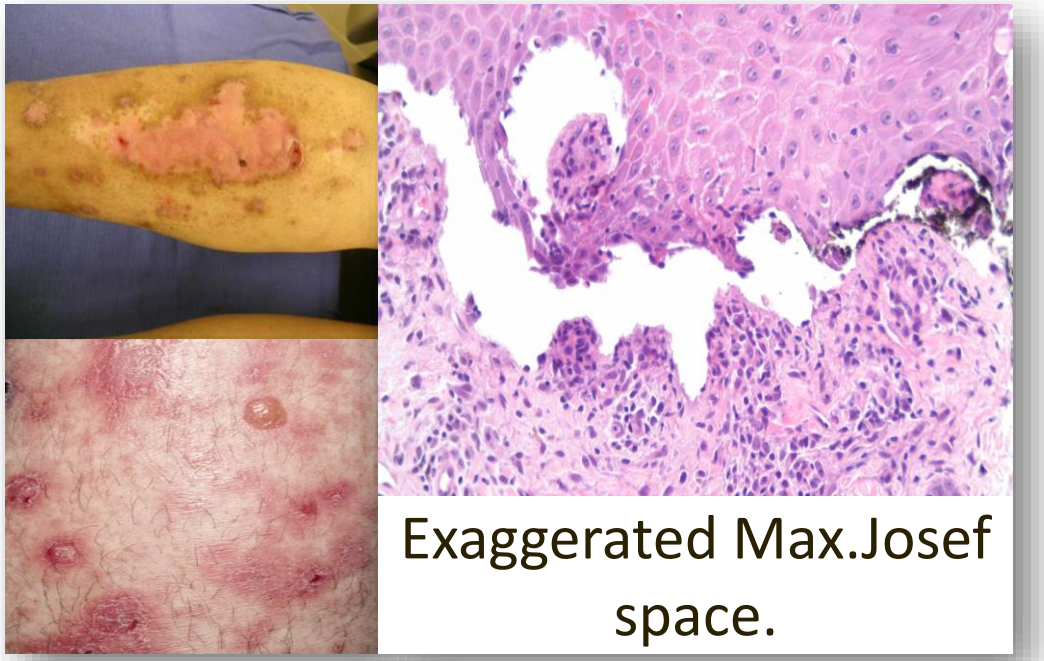
- **Description**: May represent a resolving LP.
- **Site**: Lower extremities.
- **Differential diagnosis**:
  - lichen sclerosus et Atrophicans.
  - lichen sclerosus Morphea (because of atrophy).



Atrophic lesions on the legs.

5. Bullous lichen planus (LP pemphigoides)

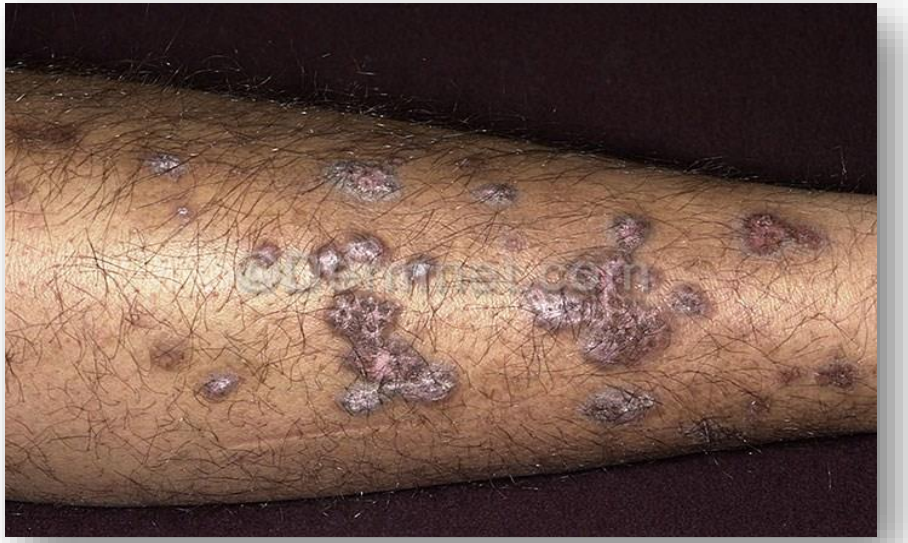
- Bullous or vesiculobullous lesions develop within pre-existing LP lesions.
- **Have 2 variants**
  1. First: exaggerated Max.Josef space which is a histopathological feature
  2. second true sub epidermal blister due to circulating IgG autoantibodies



Exaggerated Max.Josef space.

6. Hypertrophic lichen planus

- **Description**:
  - Verrucous LP (Warts like).
  - Extremely pruritic.
- **Site**:
  - Shins or dorsum of feet.
  - Symmetrical.
- **Duration**: prolonged (for 20 years).
- SCC can develop as a complication, so you need to follow up patients presenting with hypertrophic lichen planus.

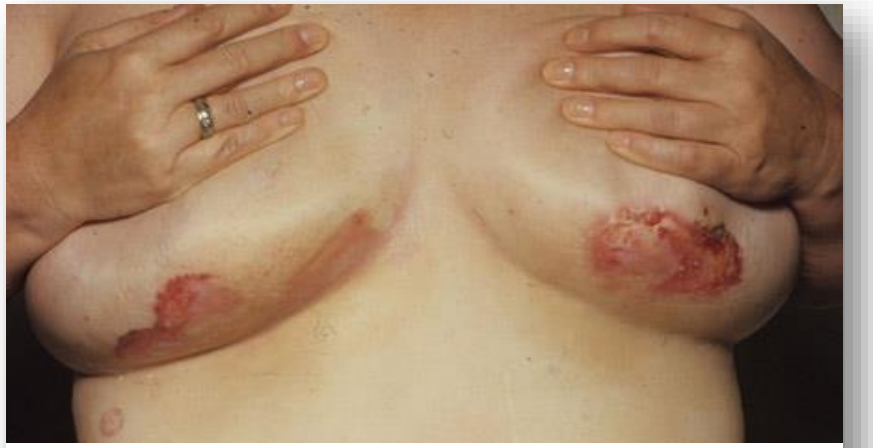


Verrucous, hypertrophic, thickened lesions.

7. Inverse lichen planus & 8. Linear lichen planus

7. Inverse lichen planus

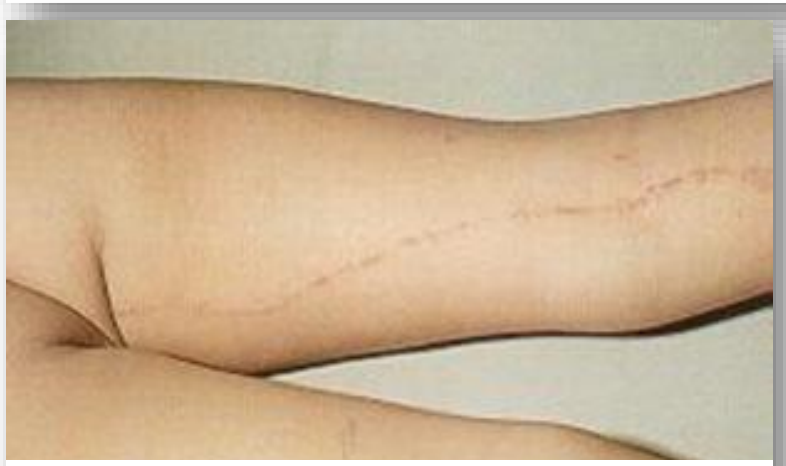
- **Description**: May present as hyperpigmentation or as violaceous papules
- **Site**: Lesions in intertriginous (flexural areas) zones (axillae, inguinal, inframammary folds)



Need a skin biopsy to make the diagnosis

8. Linear lichen planus

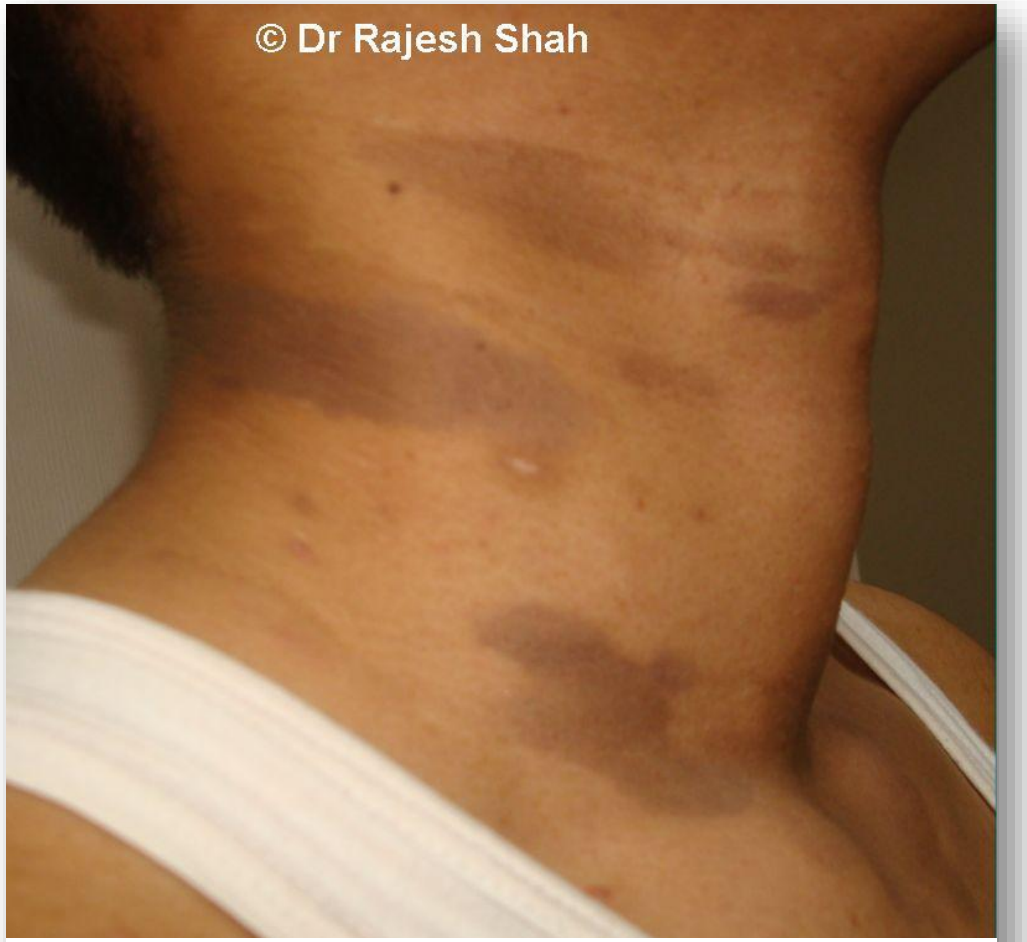
- Linear distribution of lesions within the lines of Blaschko.



Need a skin biopsy to make the diagnosis

9. Lichen planus pigmentosus

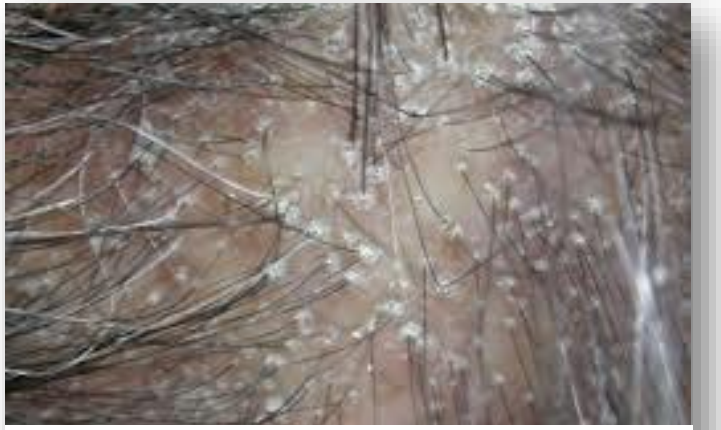
- **Description**: Brown to gray-brown macules on sun exposed areas.
- **Site**: Flexural involvement can occur
- Evolving into diffuse reticulate pigmentation.
- In type III-IV skin (our skin type).
- **Differential diagnosis**: erythema dyschromicum perstans (Ashy dermatosis)



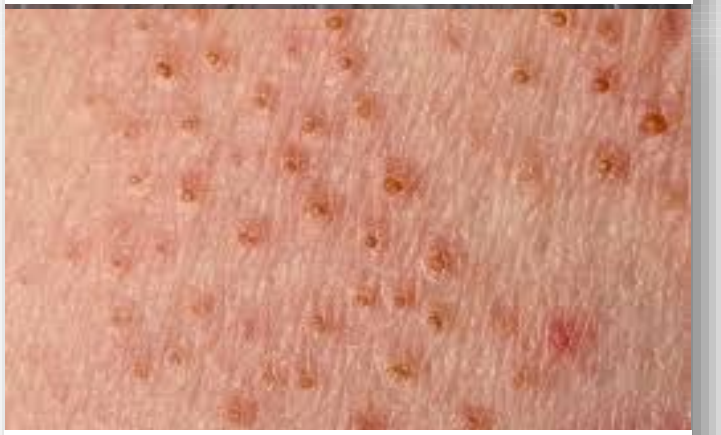
Need a skin biopsy to make the diagnosis

10. Lichen plano-pilaris (lichen planus acuminatus)

- **Description**: Keratotic plugs surrounded by a narrow violaceous rim on the scalp or other hairy areas
- **Site**: Involvement of hair follicle
- Scarring alopecia
- Women > Men
- A variant of lichen planopilaris is Graham little–Piccardi syndrome (non-scarring) axillary and pubic hair loss + scalp scarring alopecia + typical lesions of LP

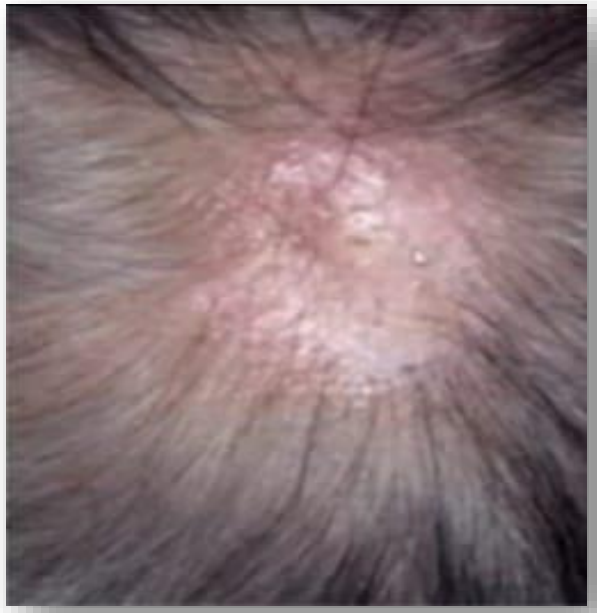


Keratotic plugs around the hair follicle with a rim of erythema and scarring alopecia.



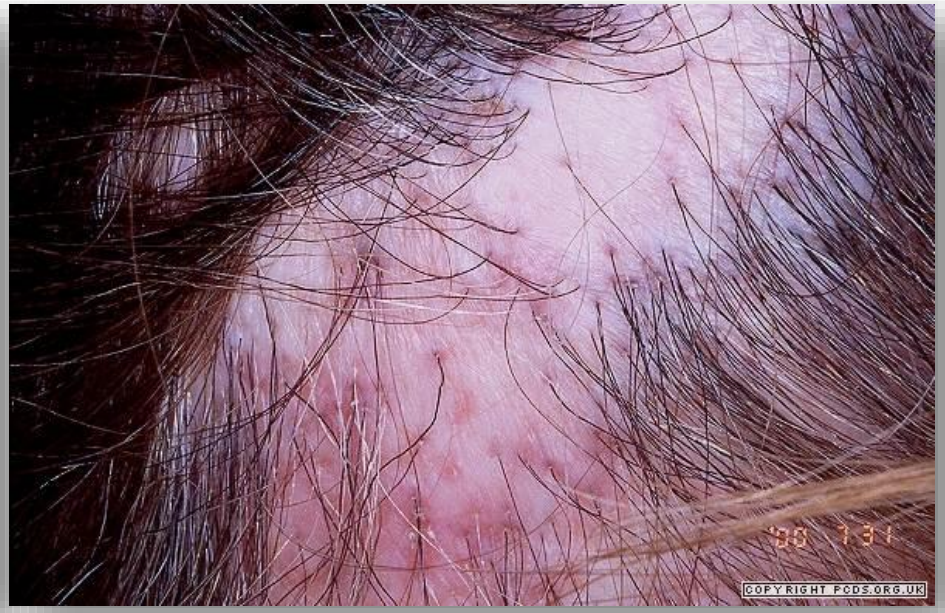
Keratotic plugs around the hair follicle with a rim of erythema.

10. Lichen plano-pilaris (lichen planus acuminatus)

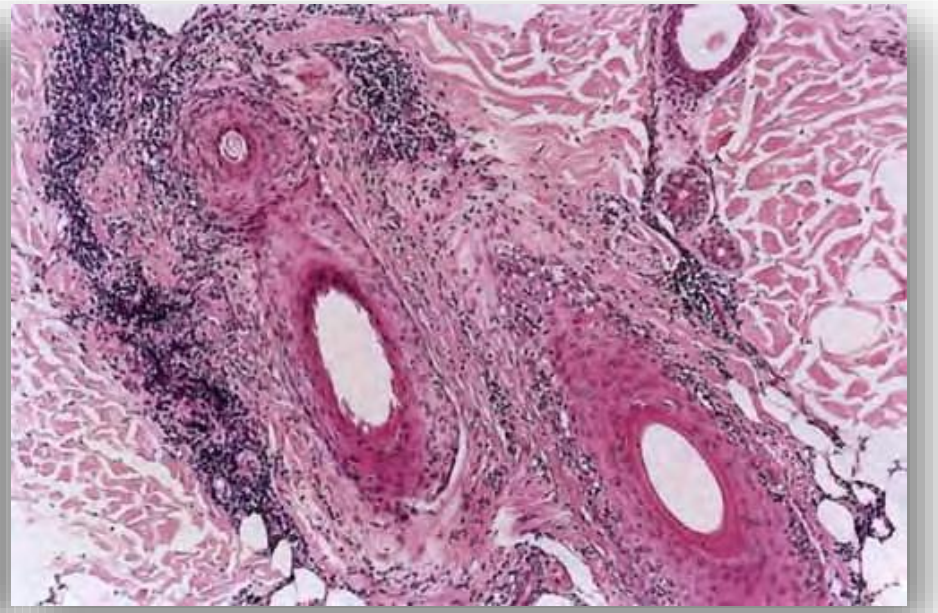


Lichen plano-pilaris with scarring alopecia.

Diagnosis is made by skin biopsy.



Permeant alopecia and disfigurement due to scarring alopecia.



Inflammatory lymphocytic reaction involving the hair follicle and destroying it.

Lichen planus variants 11, 12, 13

11. Lichen planus – Lupus erythematosus overlap syndrome

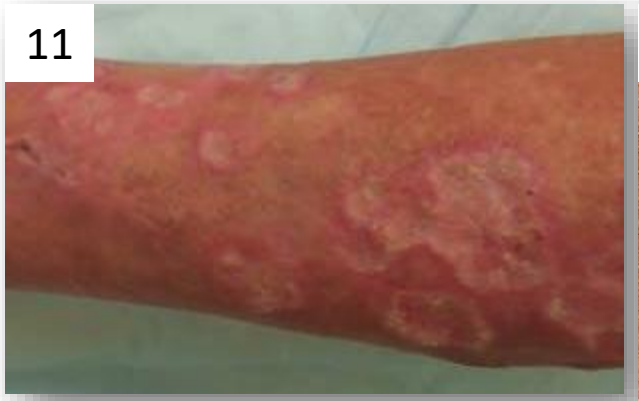
- Acral lesion
- Histological and immunofluorescence features of both LP and LE

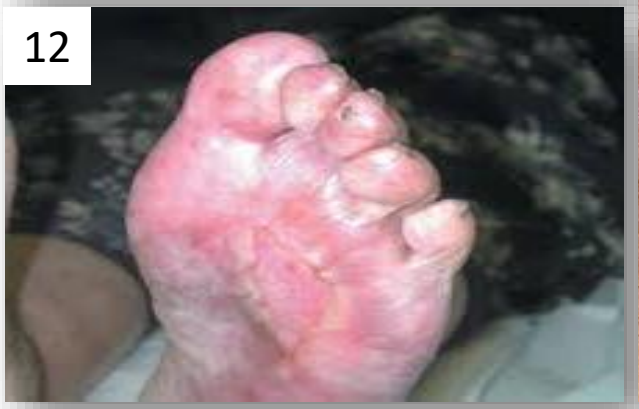
12. Ulcerative lichen planus

- **Description**: Painful ulcers
- **Site**: Palmoplantar lesions
- **Demographic**: 30-40 years of age
- Risk for SCC

13. Vulvovaginal lichen planus

- Commonly erosive.
- **Differential diagnosis**: lichen sclerosus and blistering diseases





11. Random photo from google
12. Erosions on plantar surface of the foot. Diagnosed by skin biopsy
13. Violaceous lesions streaked with white lines.

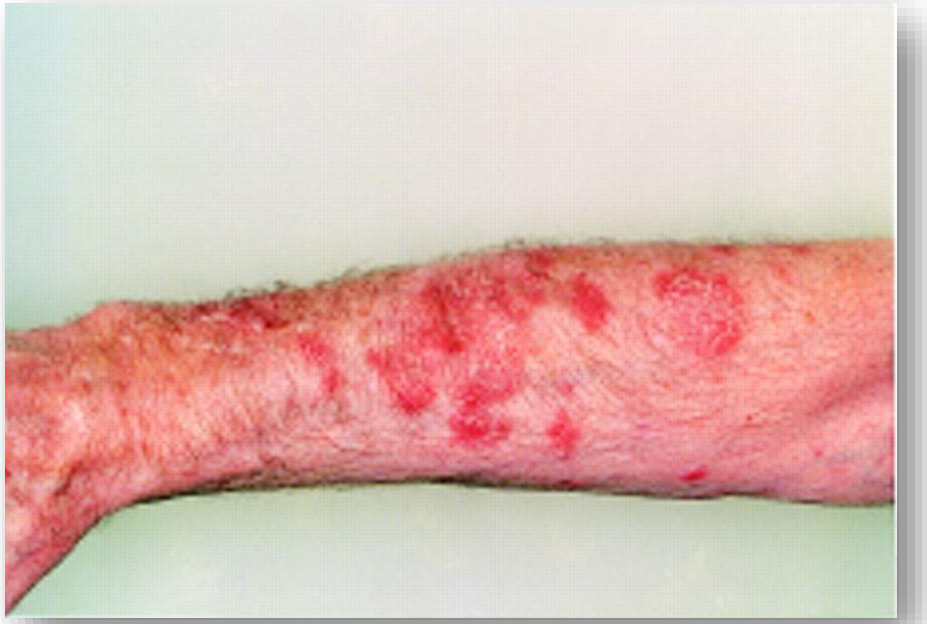
14. Lichenoid drug eruption

❖**Description**:
- More eczematous and psoriasiform lesions
- Uncommon Wickham's striae
- Spared mucosal membranes

❖**Demographic**: Older age group

❖**Causative agents**:
- Antibiotics
- ACE inhibitors
- B-Blockers
- NSAID
- Lipid lowering agents

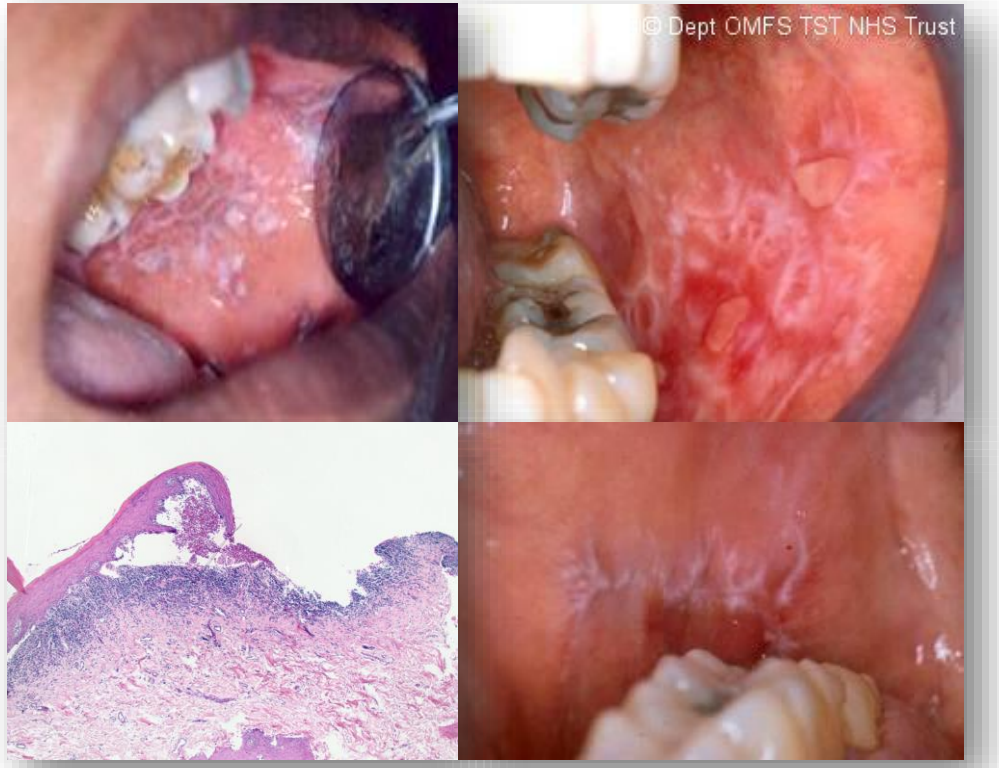
❖Usually, latent period of several months from intake to the appearance of rash



Lichenoid eczematous and psoriasiform features, diagnosed by skin biopsy or history.

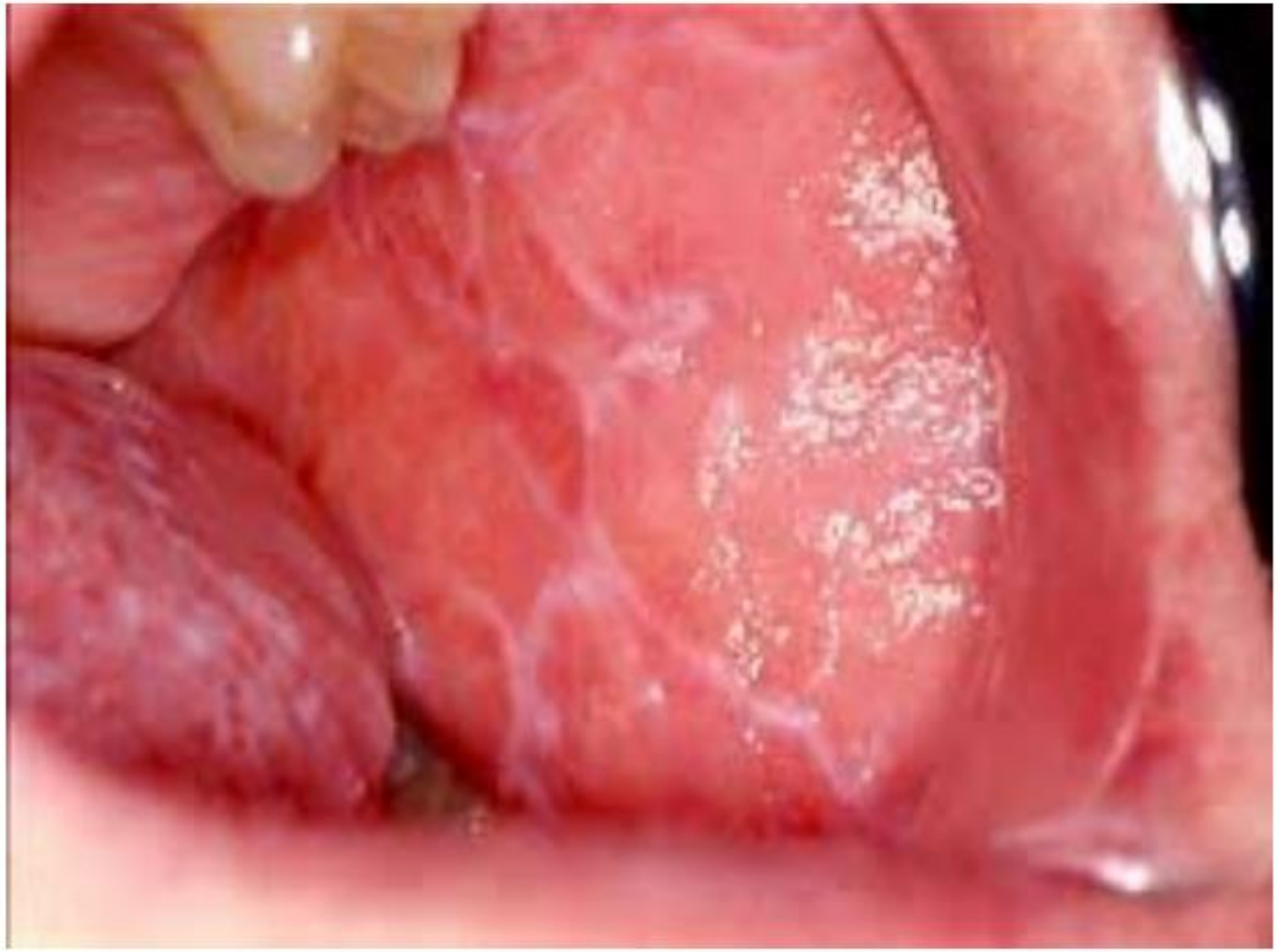
15. Oral lichen planus

- Different forms and can come in combinations:
  - Atrophic: symptomatic.
  - Bullous: symptomatic.
  - Erosive: symptomatic.
  - Papular: asymptomatic.
  - Pigmented: asymptomatic.
  - Plaque-like: among smokers.
  - Reticular: the most common as lace-like pattern, asymptomatic, symmetrical.



- Uncommon in young patients
- Women > Men
- All mucous membranes should be examined

Oral lichen planus



Mucosal lichen planus: cancerous

16. Nail lichen planus

سنوات (2)

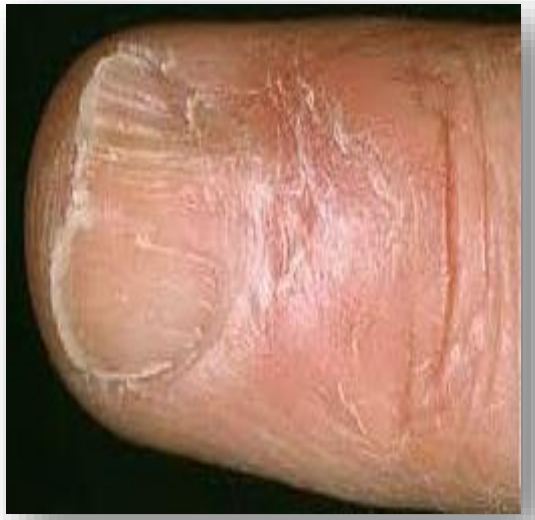
❖Mention 2 Nails changes with Lichen Planus:

1. Lateral Thinning
2. Longitudinal ridges
3. Fissuring
4. Pterygium formation
5. Twenty nail dystrophy
6. Onycholysis
7. Subungual hyperkeratosis

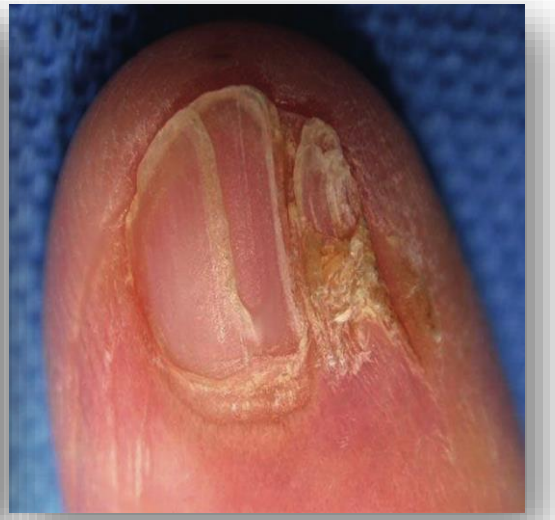
سنوات (3)

❖Pterygium is a manifestation of

- Most commonly caused by severe trauma such as warts, burns & blood circulation disorders and lichen planus

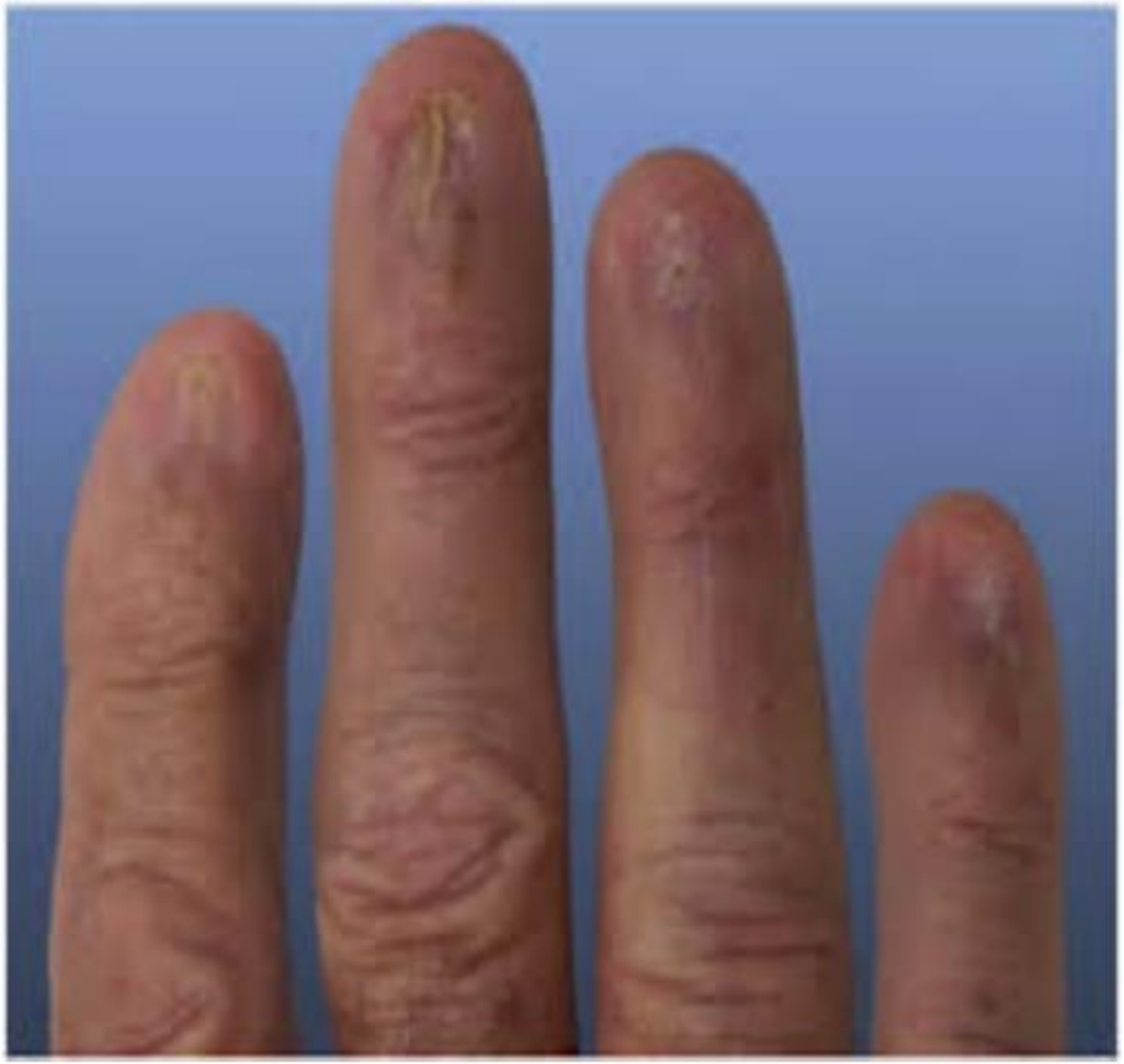


- ✓ Longitudinal ridging
- ✓ Lateral thinning
- ✓ Onycholysis
- ✓ Early Pterygium formation



- ✓ Longitudinal ridging
- ✓ Lateral thinning
- ✓ Pterygium formation

Nail lichen planus

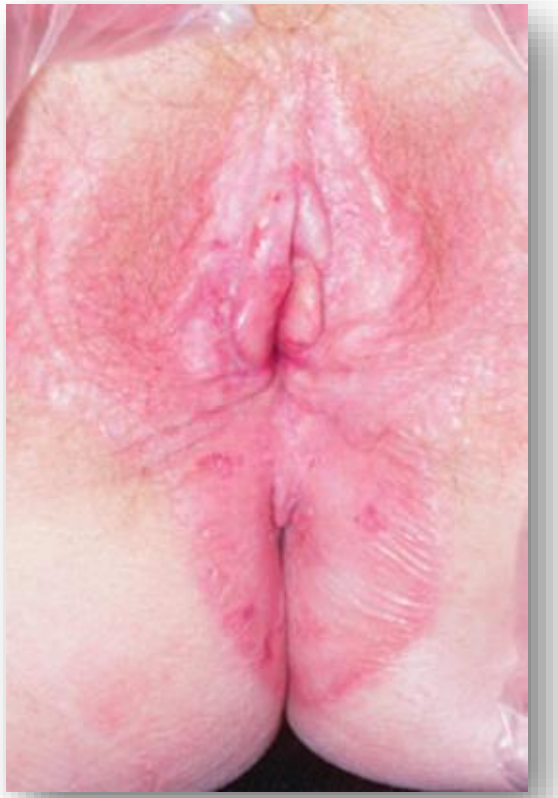


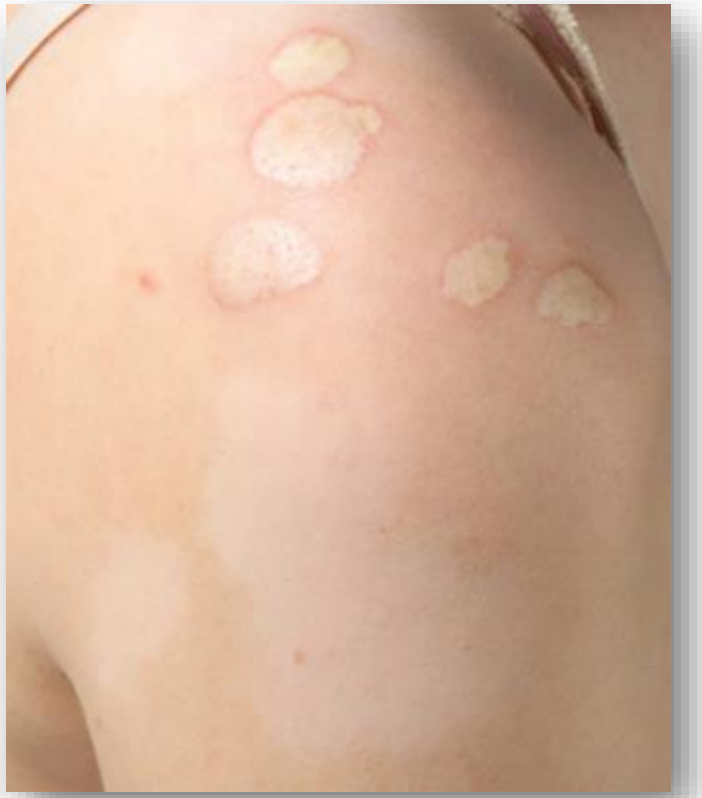
Nail changes I LP :
Pitting , nail plate thickening, multiple longitudinal splits

Lichen sclerosus 

- **Description**: White, atrophic patches appear on the vulva with severe itching
- **Site**: Most commonly on the genitalia, especially in women, but lesions also occur on the male genitalia and on extra-genital sites
- **Complications**: Vulval scarring, development of squamous cell carcinoma
- **Treatment**: Have a high recurrence rate
  - Very potent topical steroids provide symptomatic relief in vulval disease
  - Clobetasol propionate is the treatment of choice.
  - Patients should be kept under surveillance because of the risk of neoplastic change.

Lichen sclerosus





Characteristic appearance of vulval lichen sclerosus.

Well-defined white shiny skin with small haemorrhages.

Note the atrophy gives the affected skin at the bottom of the picture a creased appearance.

Treatment of Lichen planus

❖**Topical treatment**:

1. Topical steroids
2. Intralesional steroids
3. Topical calcineurin inhibitors (Tacrolimus)
4. Narrow band Ultraviolet B therapy

❖**Systemic treatment**:

1. Low dose of steroids (15-20mg for 2 weeks then taper the drug)
2. Retinoids (Acitretin 30mg daily for 8 weeks)
3. Cyclosporine

سنوات (1)

❖Itchy papule on male genetalia diagnostic for: scabies/lichen planus

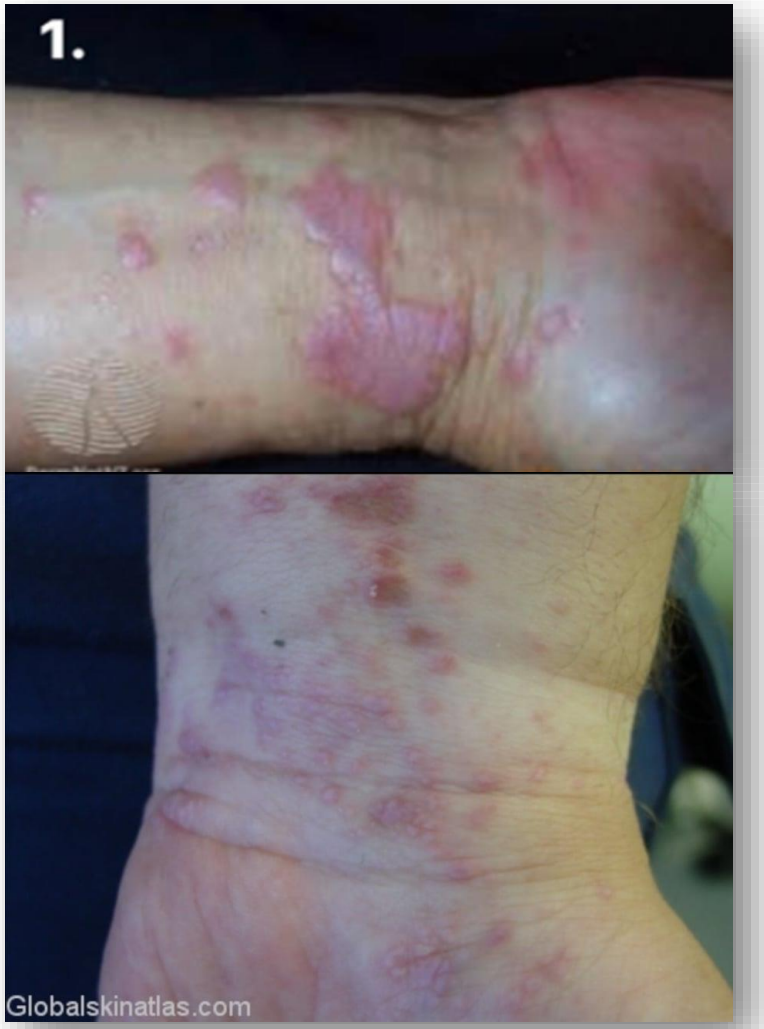
Lichen planus

سنوات (3)
- **What is your diagnosis ?**
  - Lichen planus

سنوات (1)
- **What is the primary lesion of this disease ?**
  - Papules

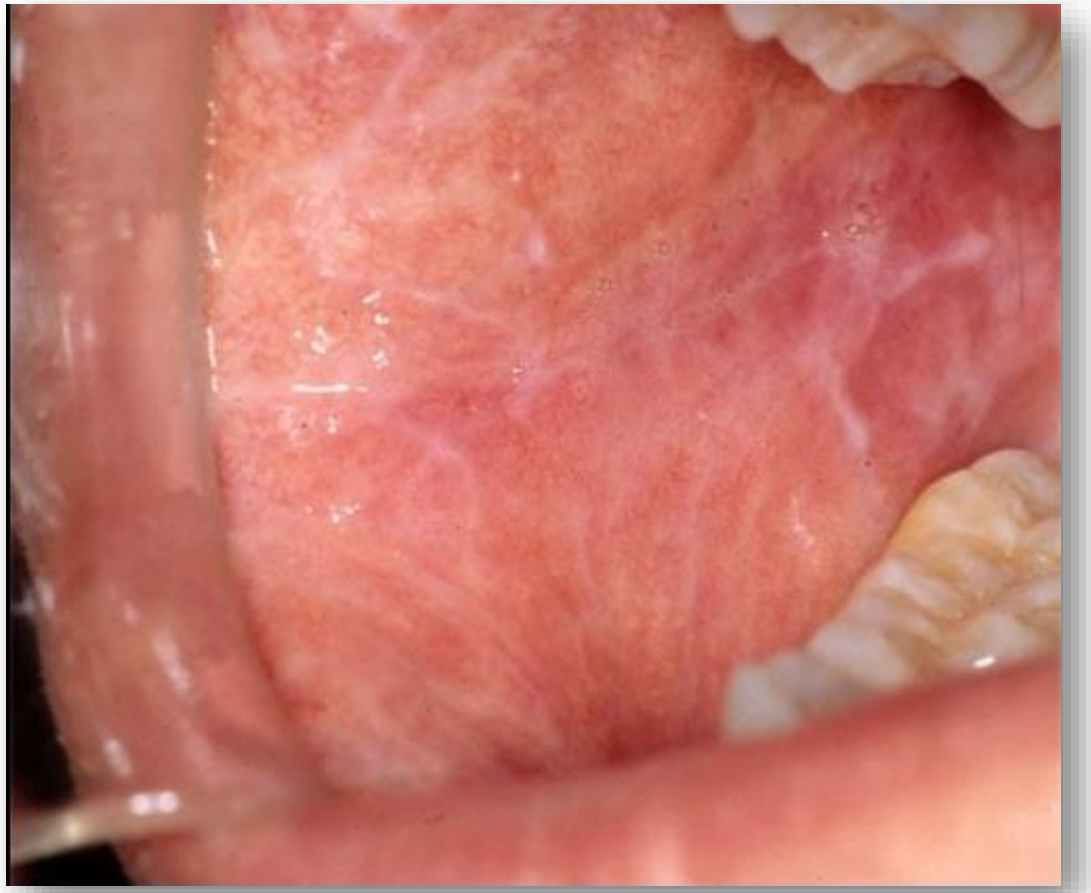
سنوات (1)
- **Mention 2 systemic disorders associated with lichen planus**
  1. Chronic hepatitis C infection
  2. Diabetes

- **Mention 3 topical and 3 systemic treatment**
  - Previous slide



Patient with white lesion on oral mucosa

- **Describe what you see**
  - Lacy, reticulate white streaks appear on the lining of the cheeks, gums and lips
- **What disease is this lesion characteristic for ?**
  - Lichen planus (Oral variant)
- **Mention 2 other forms of this oral lesion**
  1. Atrophic
  2. Bullous
  3. Erosive
  4. Papular
  5. Pigmented
  6. Plaque-like



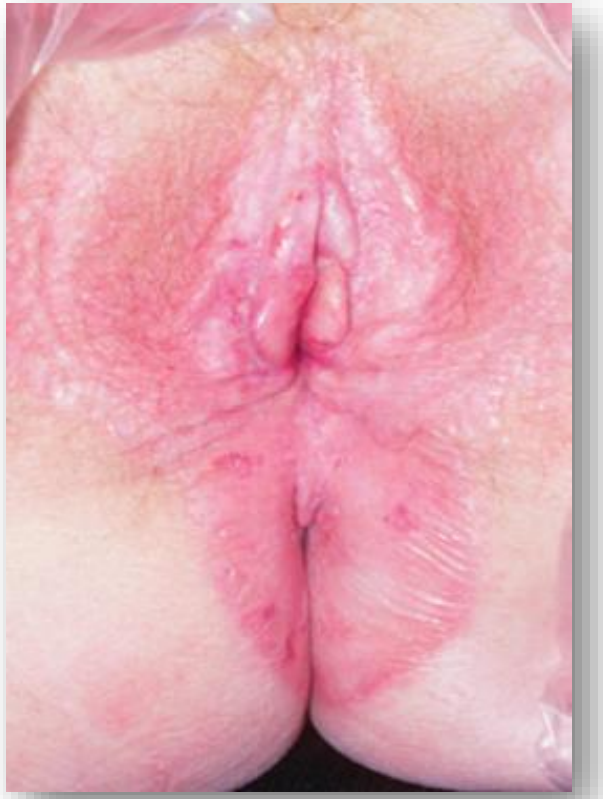
Lichen sclerosus

سنوات (1)

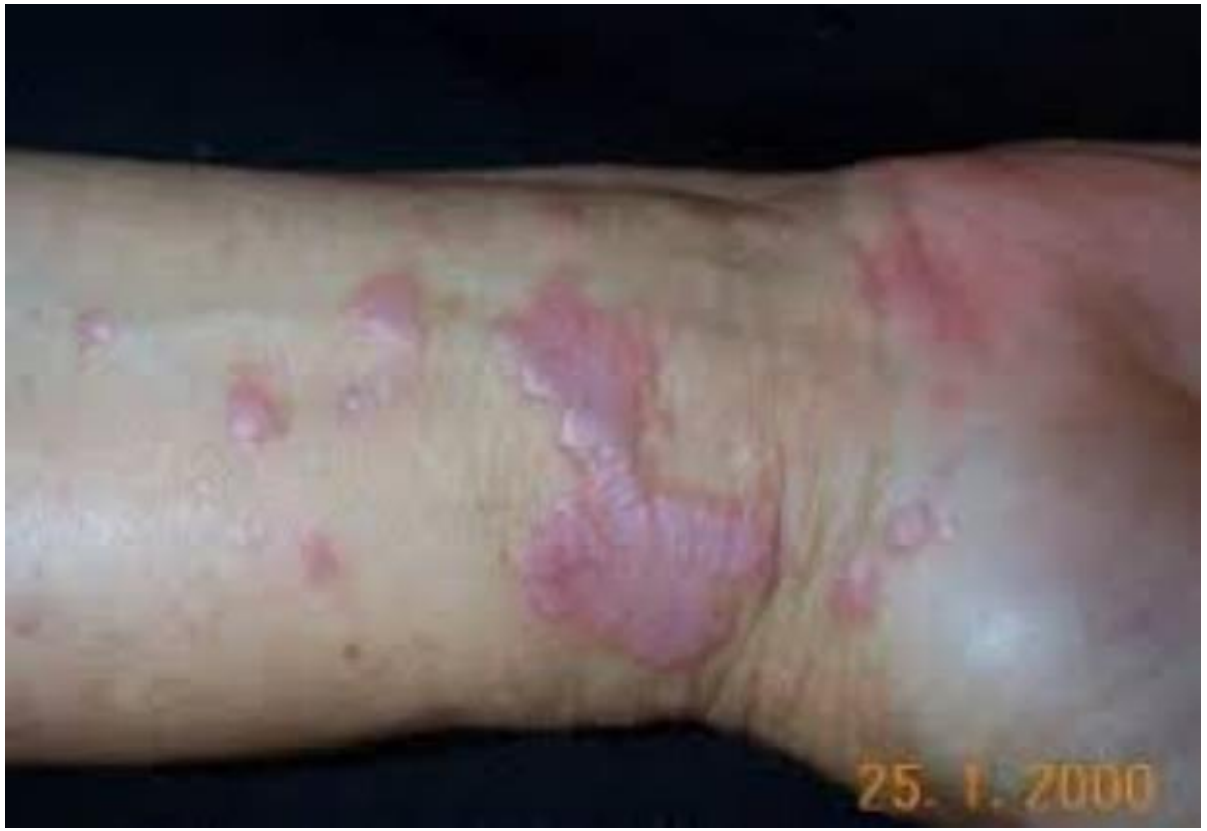
- **Presentation of patient with lichen sclerosus?**
  - White, atrophic patches appear on the vulva with sever itching

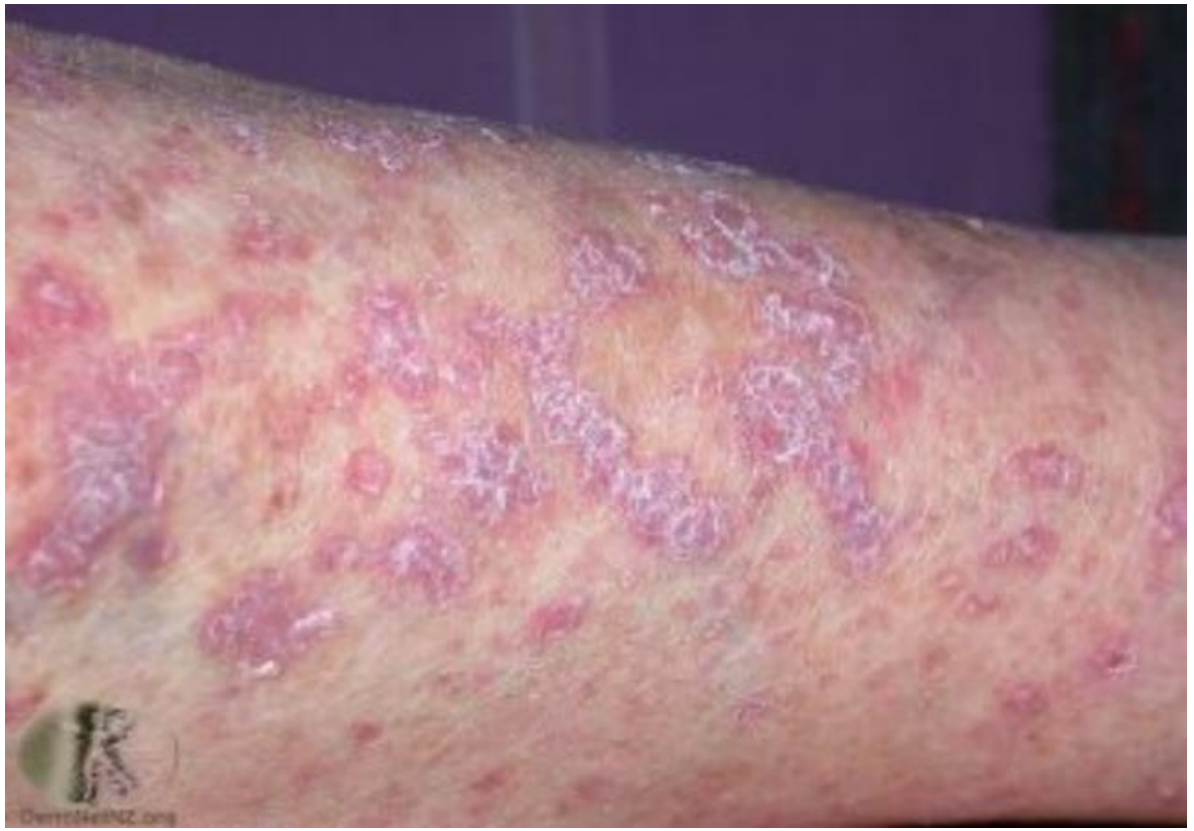
سنوات (1)

- **Patient with lichen sclerosus have increased risk for?**
  - Squamous cell carcinoma, vulval scarring
- **Differential diagnosis**
  - Atrophic lichen planus
  - Vulvovaginal lichen planus
- **Who does prepubertal girls present with this disease, it can be misdiagnosed as what ?**
  - Presents with dysuria and pain on defecation. It may be misdiagnosed as sexual abuse

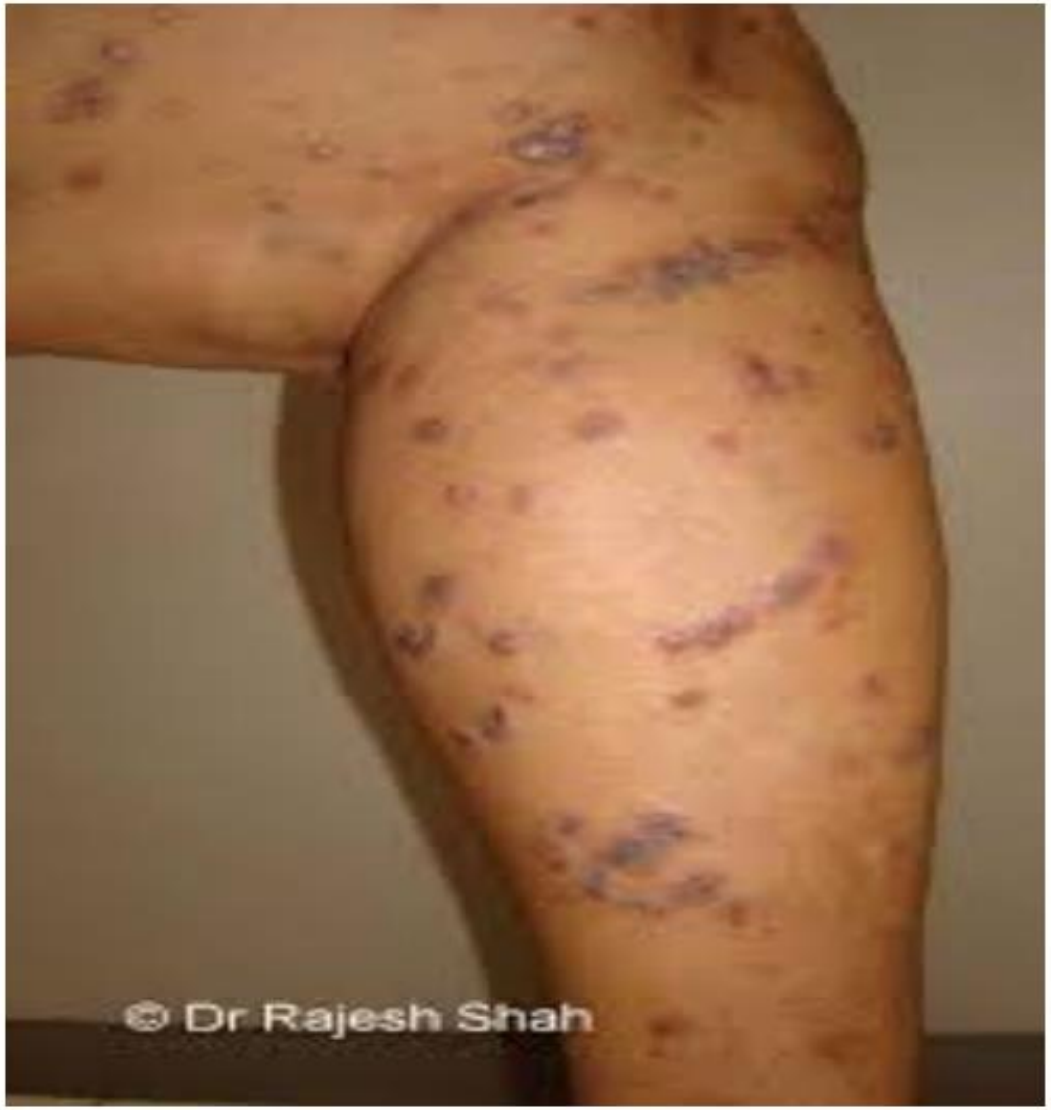


Lichen planus





Lichen planus



Koebner's

Pityriasis Rosea

نخالية وردية

شرح

Pityriasis Rosea

- Relatively common acute, self-limited papulosquamous eruption (mildly itchy or asymptomatic mainly in healthy adolescent and young adults.
- **Incidence**: 0.5-1%. **Demographic**: 10-35 years old age. F>M.
- **Duration** 6-8 weeks may be up to 5 months or more rarely.

سنوات (2)

- **Mention 2 clinical features of pityriasis rosea?**
  1. Herald patch
  2. Collarette scale
  3. Christmas tree distribution

سنوات (1)

- **Define Herald patch**: is the first lesion to appear, it appears as Pink patch or plaque with raised advancing edge 1-4cm in diameter.

شرح

Pityriasis Rosea

سنوات (1)

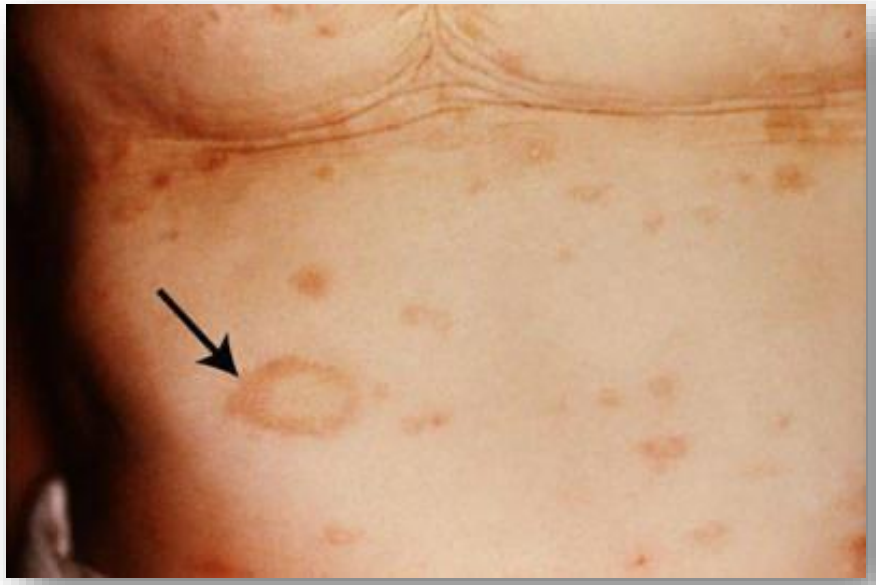
❖Clinical course of pityriasis rosea

1. Prodromal illness
2. One or more herald patches, (most common on trunk or neck)
3. Sudden eruption of pink oval patch
4. After 2-3 days numerous scaly small oval plaques and papules (daughter patches) appear along the trunk and proximal extremities with collaret scales (free edge points inward)
5. Christmas tree distribution on the back

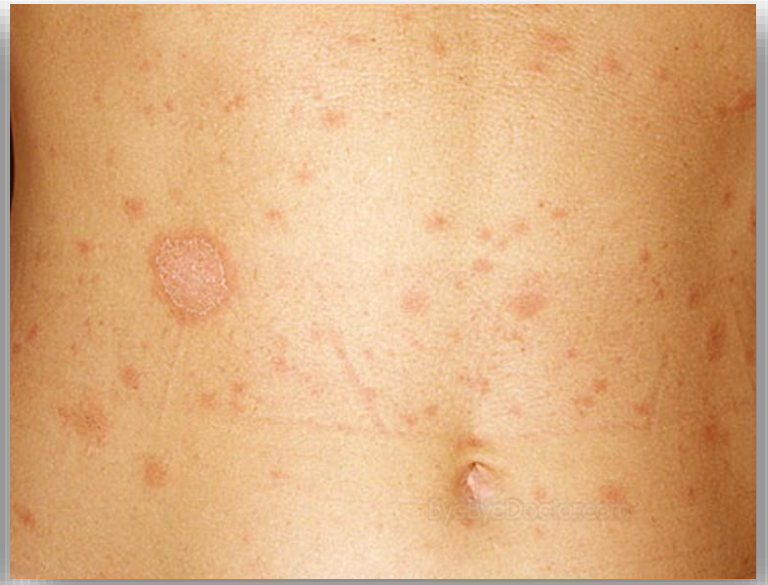
❖Treatment:

- Reassurance.
- Symptomatic.
- Phototherapy – Narrow band – UVB.
- Erythromycin (500 mg 5 times daily for 10 days to shorten the duration of the disease).

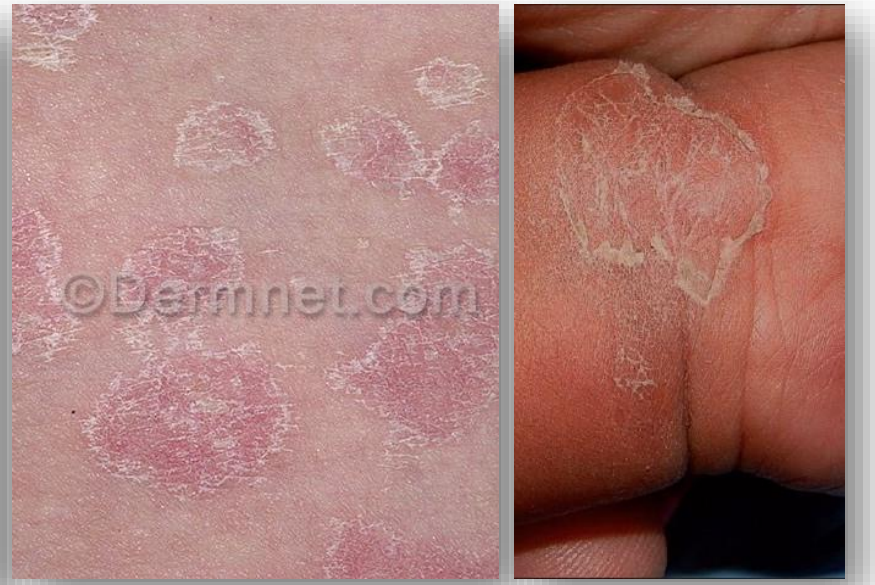
Pityriasis Rosea



Annular lesion with clear center and raised scaly margin (Herald patch) which appears first, followed by the appearance of the daughter patches.

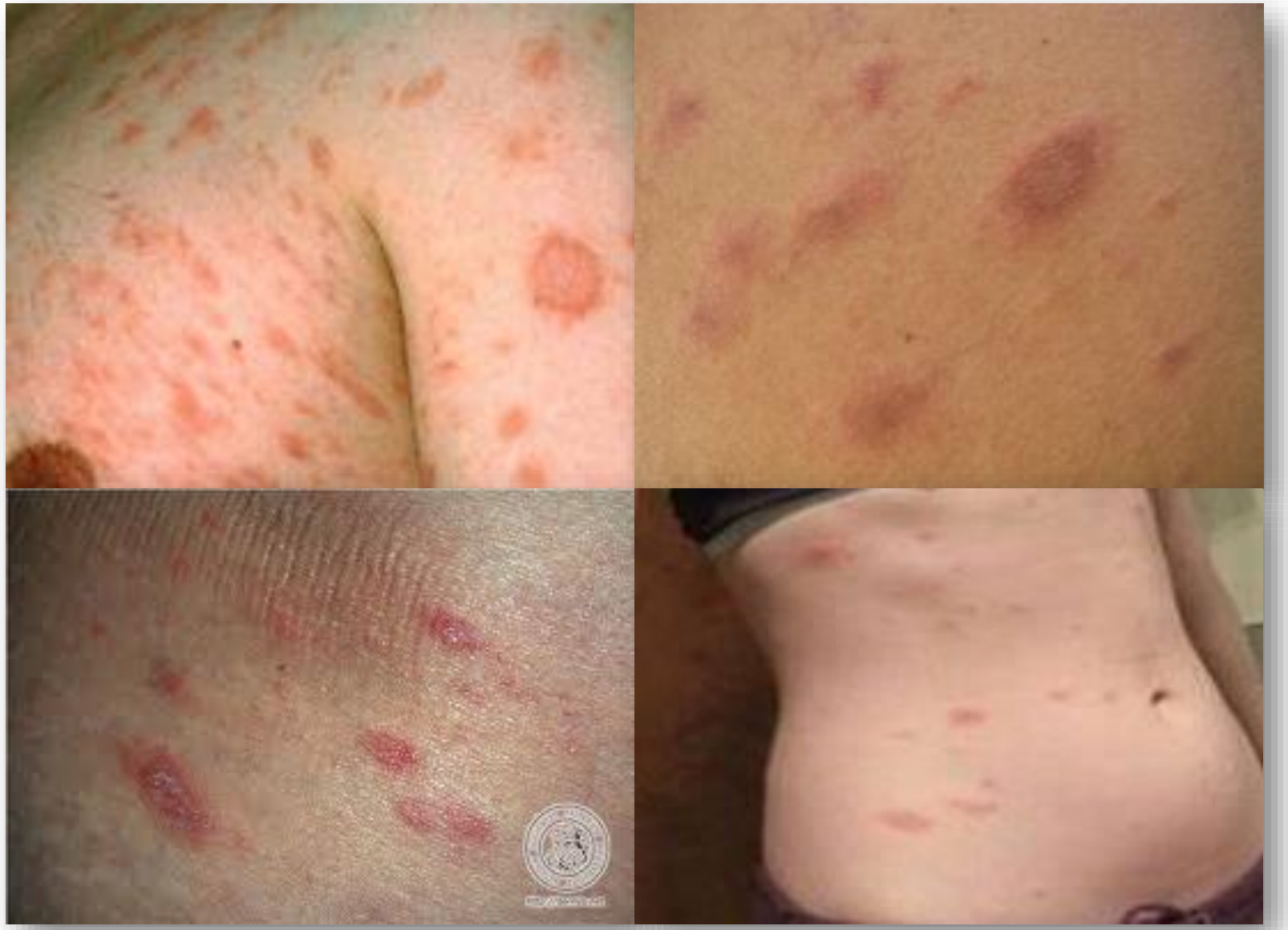


The scale is attached to the periphery of the lesion and opens toward the center (Collaret scale).

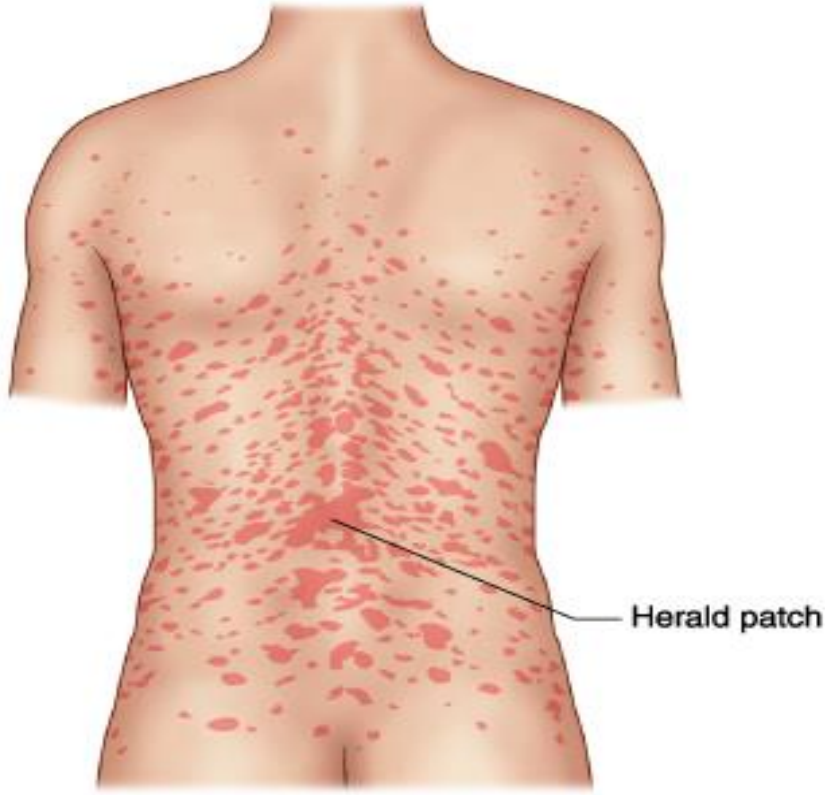


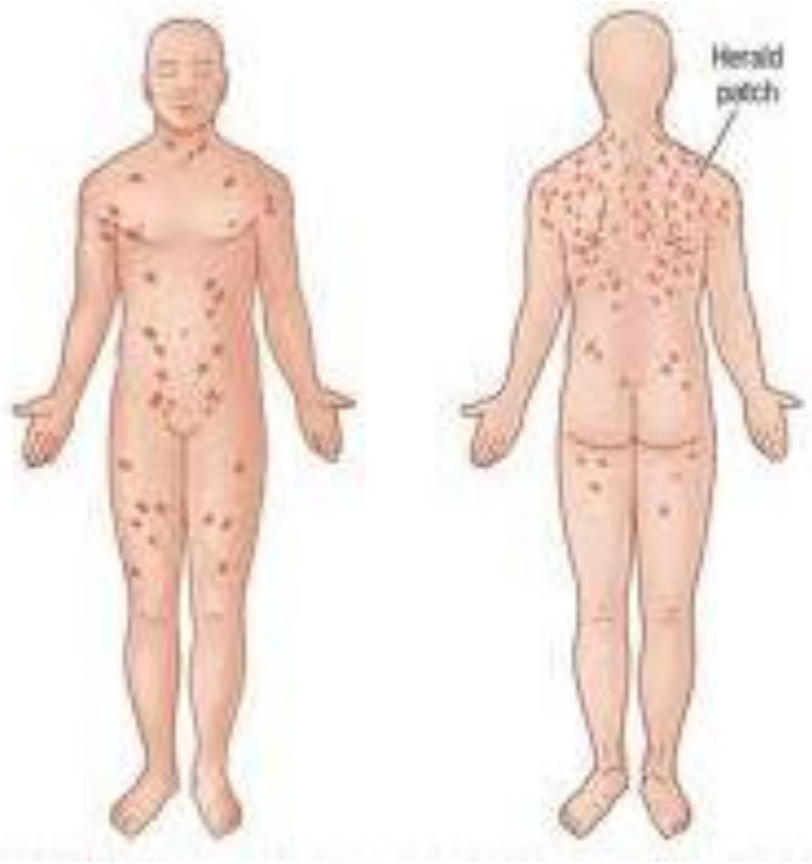
Collaret scales.

Oval Daughter patches which are parallel to each other.



Pityriasis Rosea

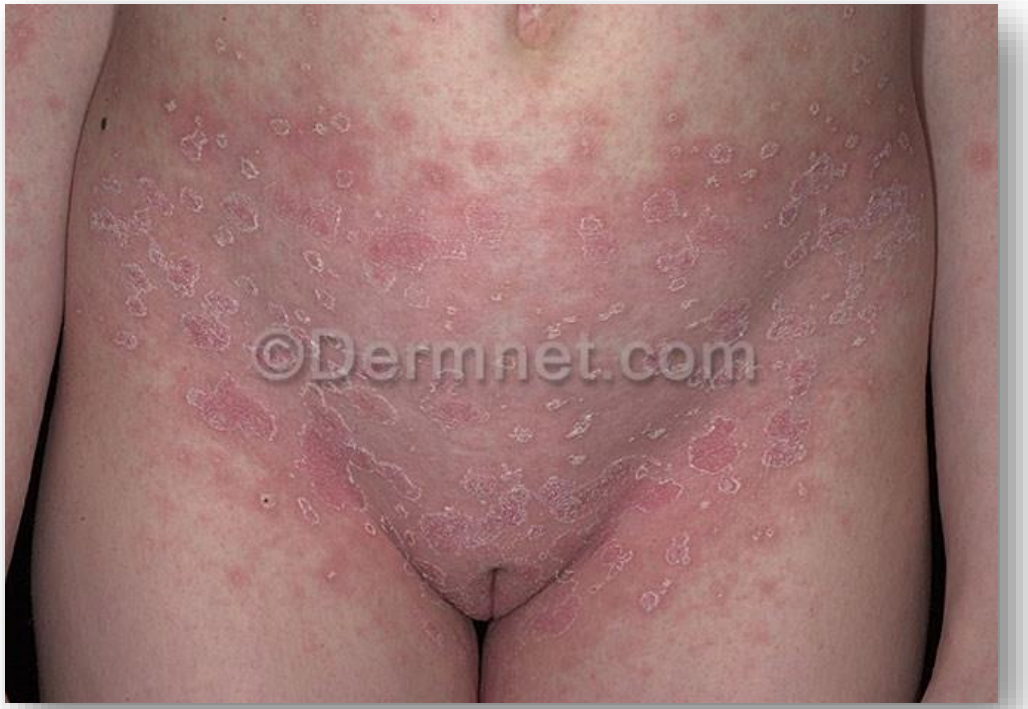




Christmas tree distribution on the trunk and proximal extremities

Pityriasis Rosea – Atypical forms

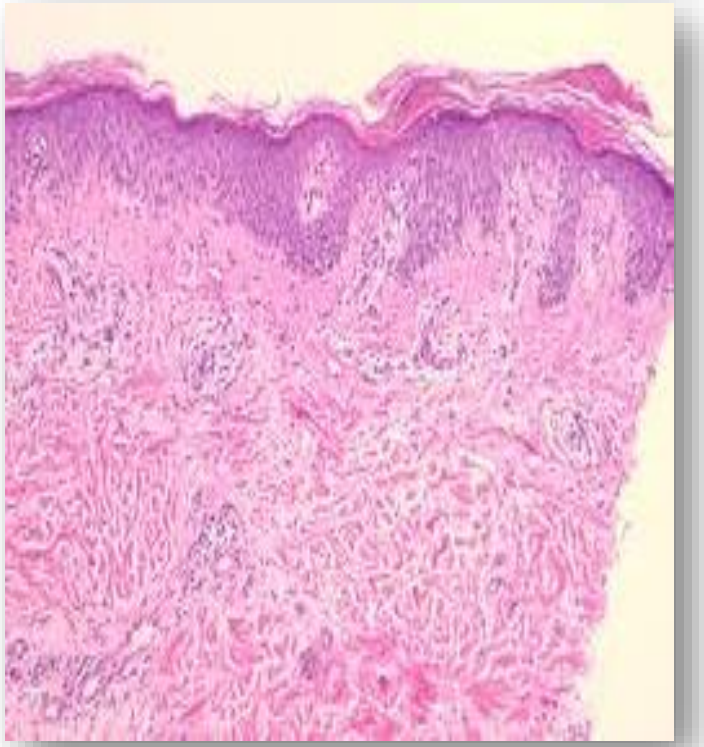
1. Inverse form (common in children)
2. Urticarial form
3. Erythema multiforme – like form
4. Vesicular form
5. Pustular form
6. Pruritic form



Inverse form on flexural areas

Pityriasis Rosea – Diagnosis

- Diagnosed clinically
- If persists > 5 months ⟶ Pityriasis lichenoides chronica (PLC) should be considered and skin biopsy should be performed
- If persists > 8 months ⟶ skin biopsy is done to look for other differential diagnosis:
  1. Secondary syphilis (in young adults)
  2. Tinea corporis
  3. Lichen planus
  4. Mycosis fungoides (Cutaneous T cell lymphoma)
  5. Drug eruption
  6. Guttate psoriasis
  7. Pityriasis lichenoides chronica



The histological appearance of Pityriasis Rosea is non-specific but the biopsy is done to rule out other possible causes and to support the clinical suspicion of Pityriasis Rosea.

سنوات (1)

Sudden patches appear after prodromal illness

سنوات (1)

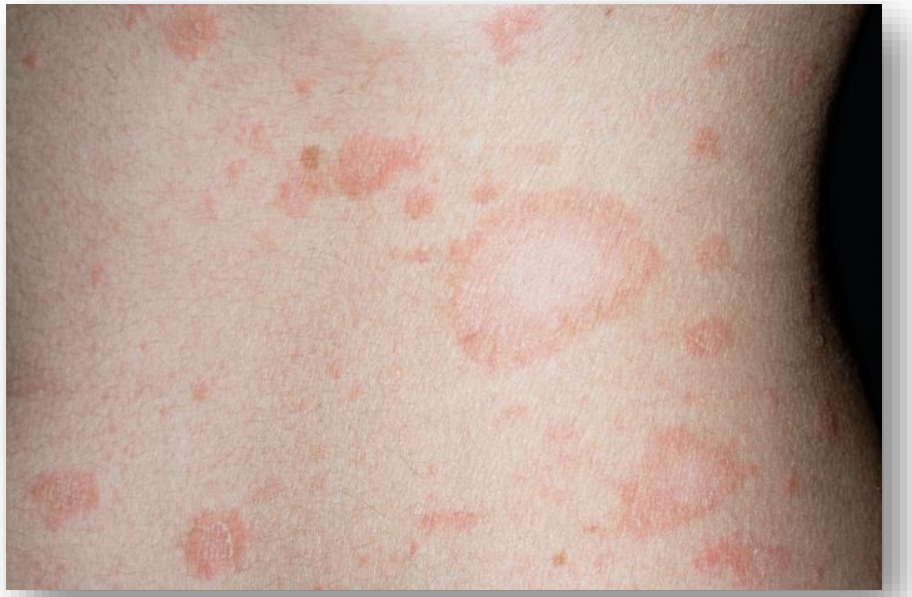
❖**What is your diagnosis ?**

- Pityriasis rosea

سنوات (1)

❖**Mention 3 clinical features of pityriasis rosea ?**

1. Herald patch
2. Collarette scale
3. Christmas tree distribution



❖**What is your management if the lesion present for more than 5 months ?**

- Skin biopsy to rule out other differential diagnosis

Pt with Christmas tree distribution on the back, diagnosis ?

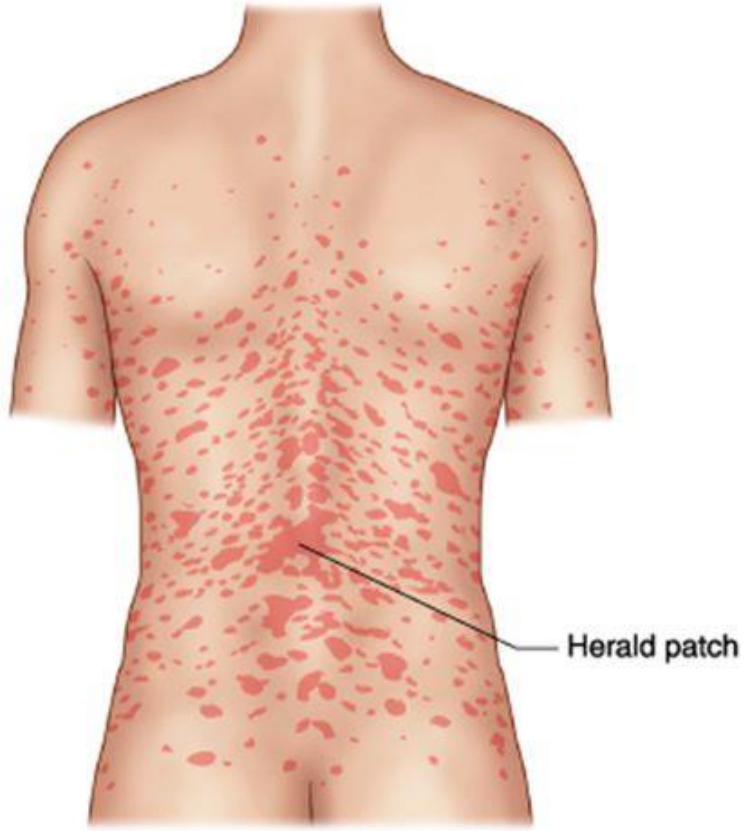
سنوات (2)

❖**What is your diagnosis ?**

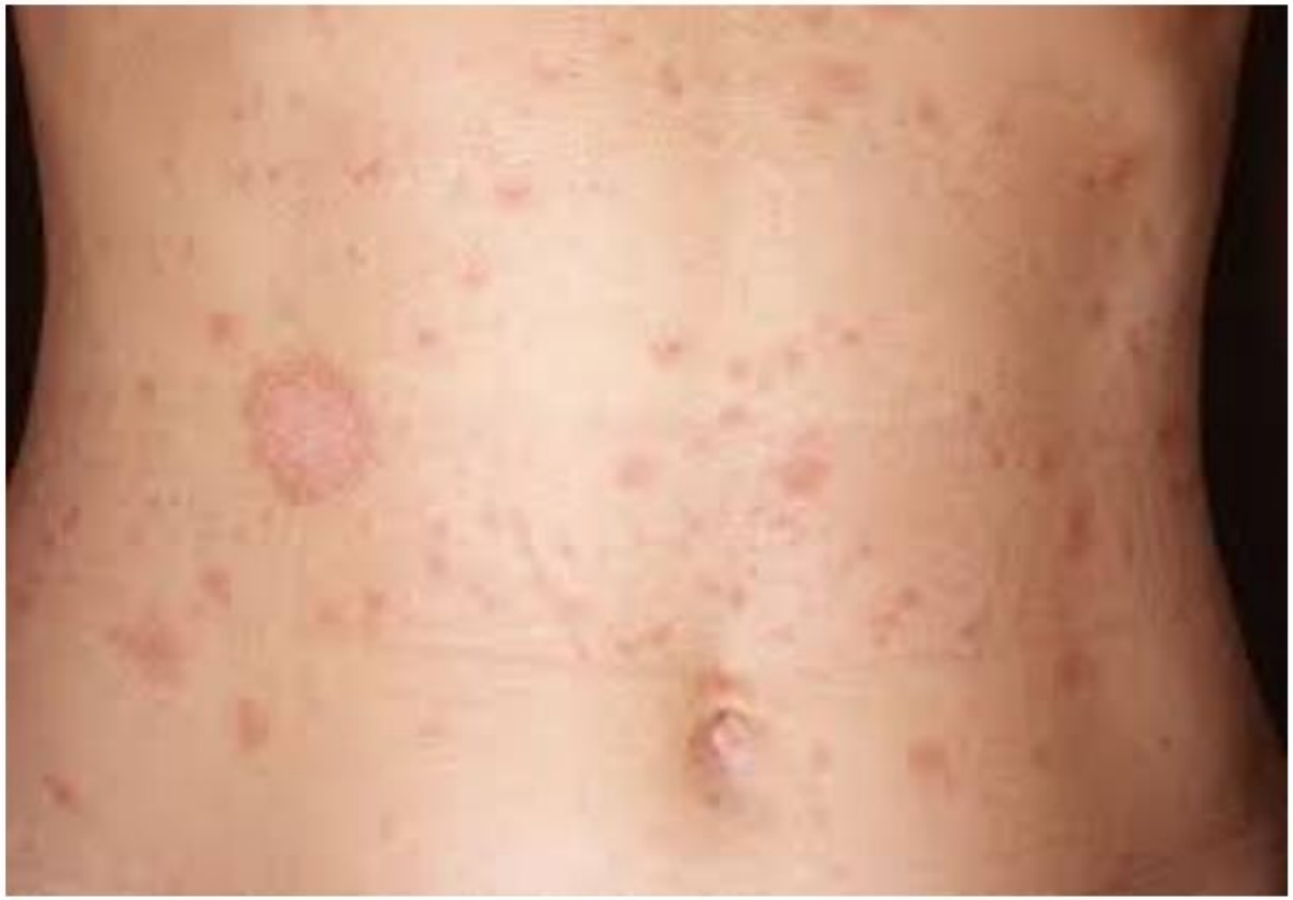
- Pityriasis rosea

❖**If this lesion present for more than 8 months mention 3 differential diagnosis**

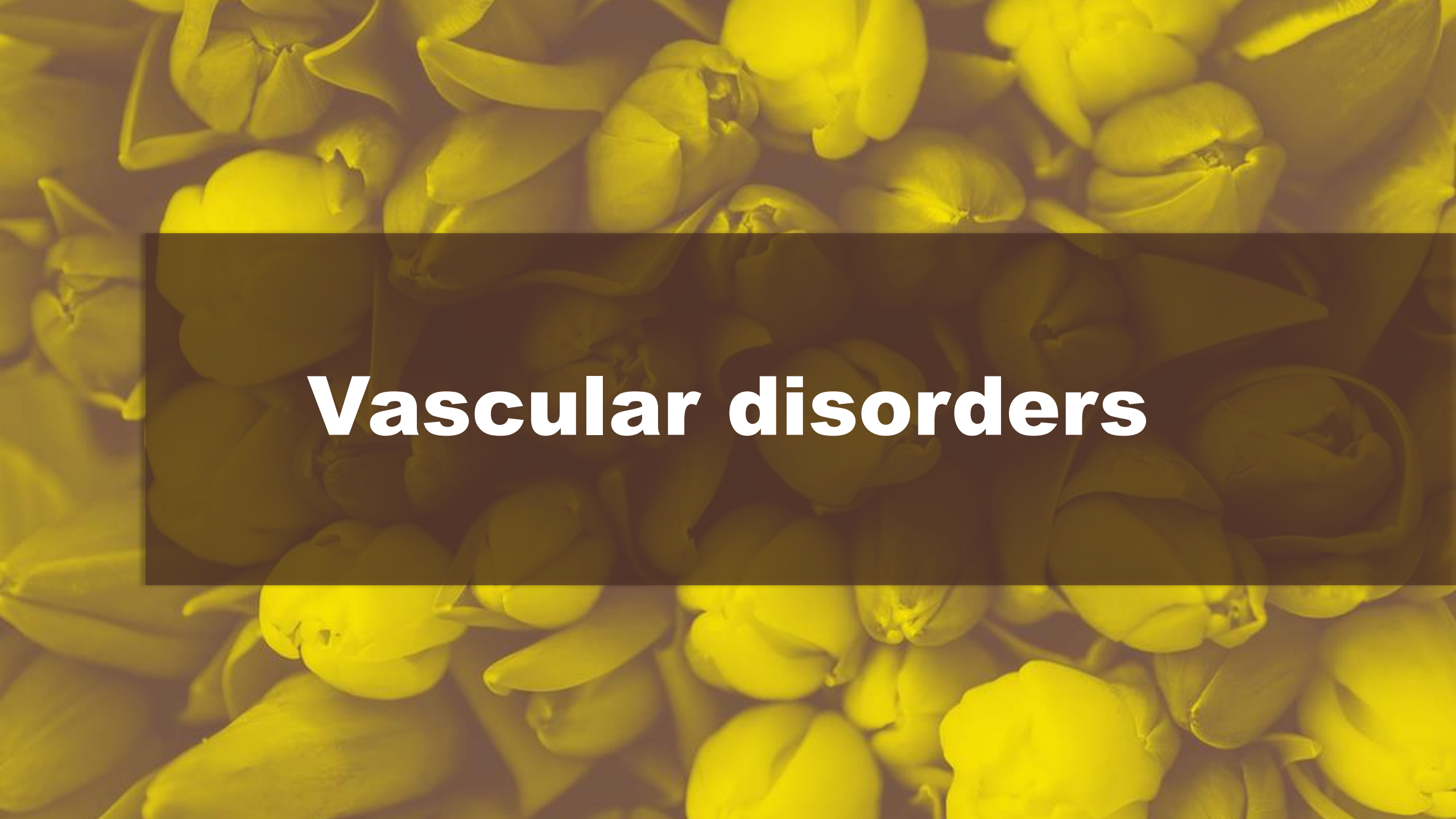
1. Secondary syphilis (in young adults)
2. Tinea corporis
3. Lichen planus
4. Mycosis fungoides (Cutaneous T cell lymphoma)
5. Drug eruption
6. Guttate psoriasis
7. Pityriasis lichenoides chronica



Pityriasis rosea

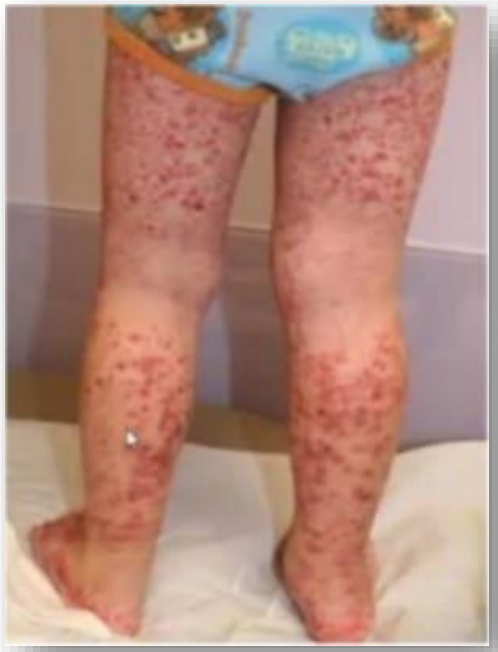


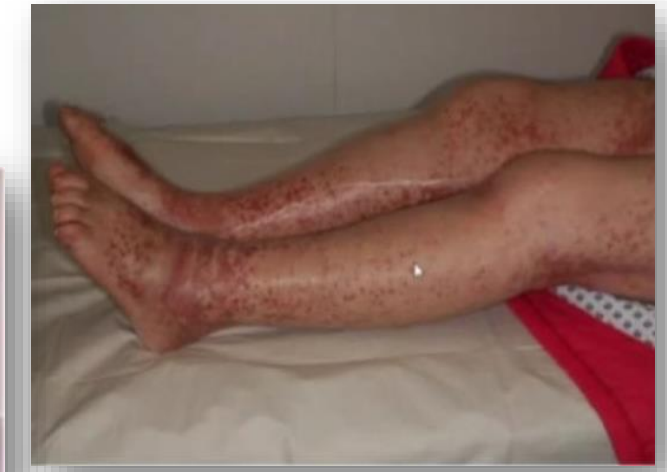
Pityriasis rosea
No recurrence, no scars , not contagious, collar scales, herald patch

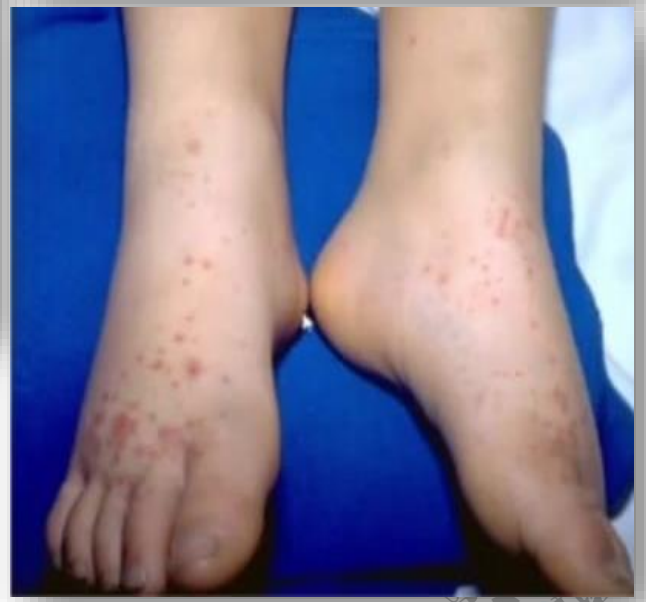

Vascular disorders

Henoch–Schönlein purpura

- **Demographic**:
  - Predominantly in children
- **Presentation:**
  - Maculopapular rash usually over the lower extremities and buttocks, Joint swelling and Abdominal pain
- **Pathophysiology**:
  - Deposition of IgA immune complexes in the skin (palpable purpura), joints (arthritis), kidney (glomerulonephritis), and GI tract (abdominal pain and gastrointestinal haemorrhage)

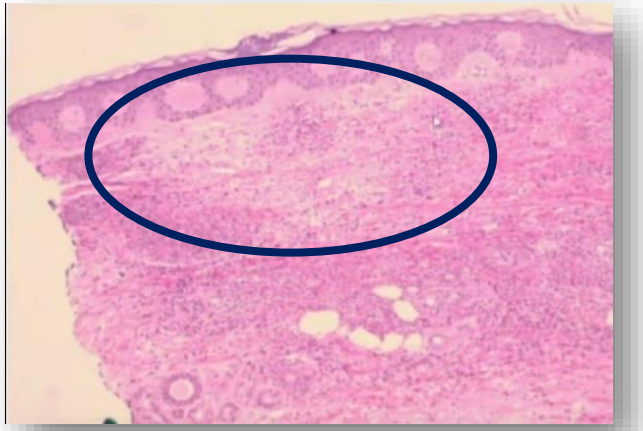


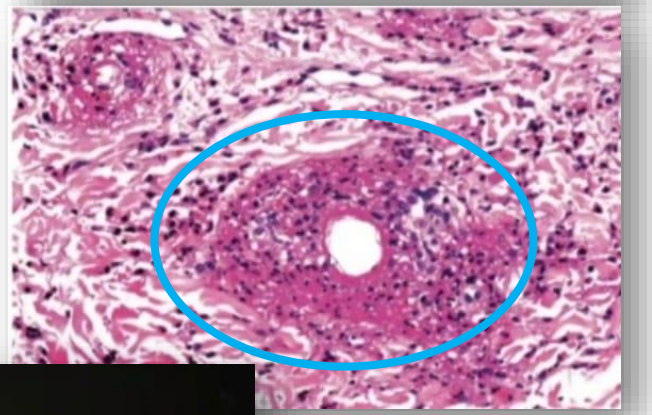


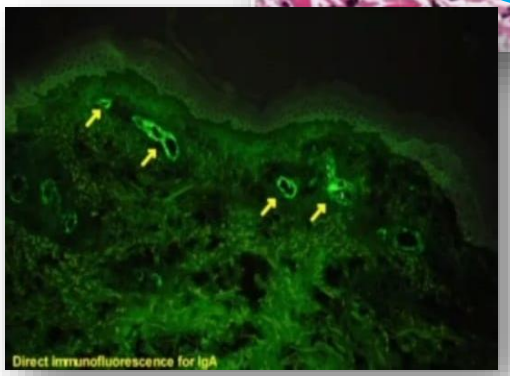


Henoch–Schönlein purpura

- **Preceding event**:
  - Usually following an Infection (URT infection), Drug allergy and insect bites
- **Diagnosis**:
  - Usually clinically.
  - Skin biopsy will show inflamed dermis and necrotic wall of the blood vessel (Fibrinoid necrosis).
  - Immunofluorescence will show IgA deposits in the wall of blood vessels.
- **Treatment**:
  - Treat the triggering factor.
  - Short course of systemic steroids for the blood vessels' inflammation.
  - Those patients need follow up for their renal involvement.





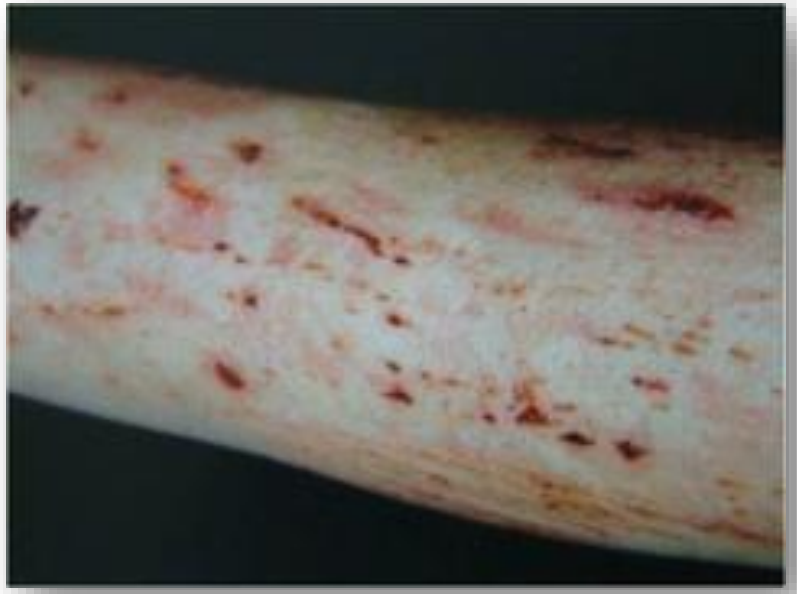


Pruritis

Pruritus

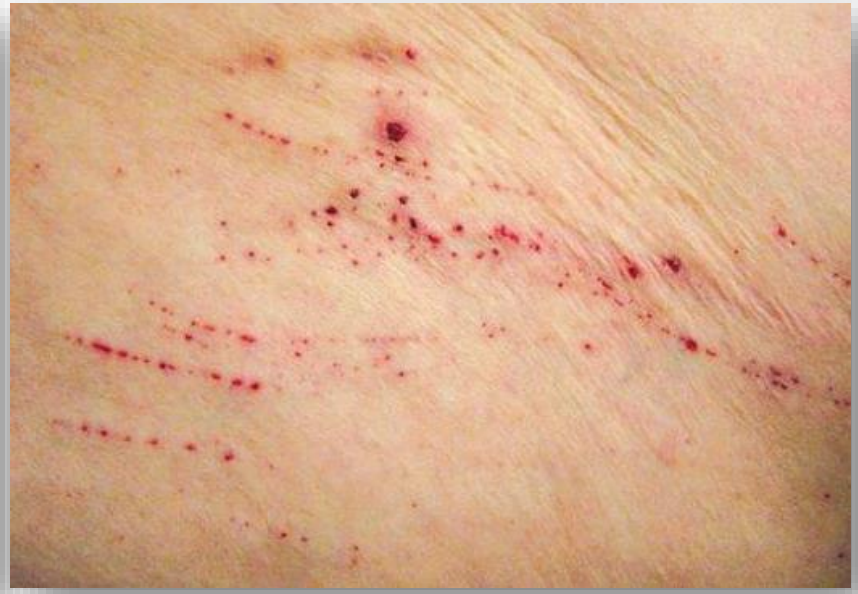
- Pruritus is a complex neurophysiological process through different mediation as a protective response to remove pruritogens.
- Pruritus is the most common dermatologic symptom.
- Few clinical clues
  - Acute pruritus with no primary skin lesions and of short duration is less suggestive of systemic cause
  - Pruritus not related to a primary skin disease where there is only excoriation and secondary changes is mostly suggestive a systemic cause
  - When multiple family members are affected think of infestation (Scabies, pediculosis)
  - Pruritus after bathing think of polycythemia rubra vera
  - Night pruritus with chills , sweating and fever think of Hodgkin`s disease

Pruritus

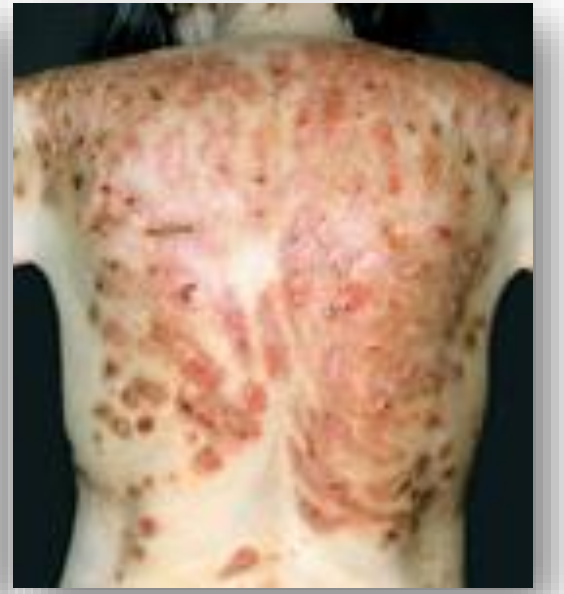


Scratch marks and excoriation due to Hodgkin's lymphoma

(Night pruritus with chills, sweating and fever)



Scratch marks and excoriation without primary skin lesions



Neurotic excoriation, may be due to psychological stress, abnormality or triggering factor.

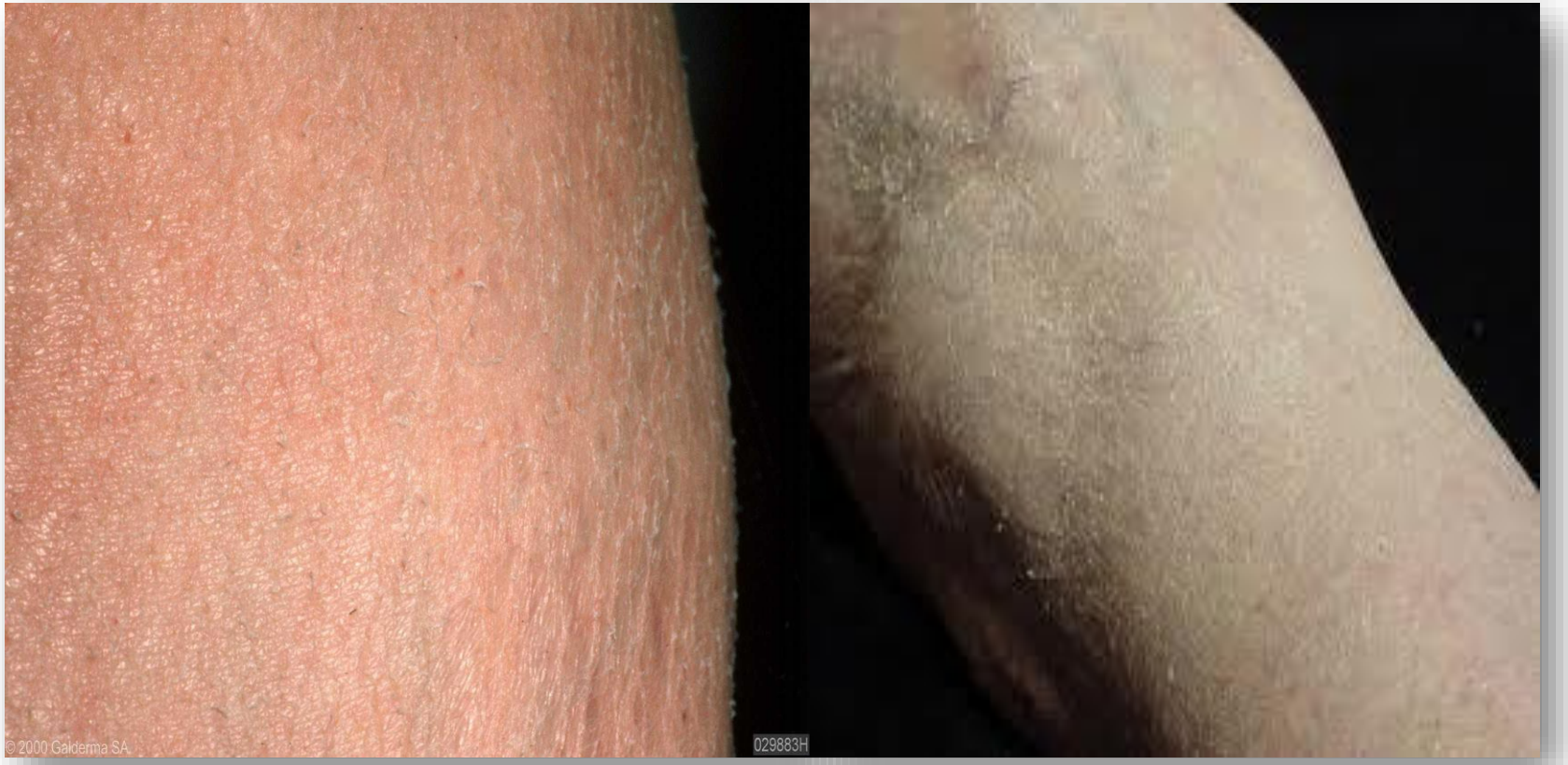
Dermatological disease that cause generalized pruritus

1. **Xerosis**
2. Infections (Folliculitis, Chickenpox, Herpes)
3. Scabies
4. Pediculosis
5. Atopic dermatitis
6. Psoriasis
7. Dermatitis herpetiformis
8. Urticaria
9. Lichen planus
10. **Mycosis fundgoides**

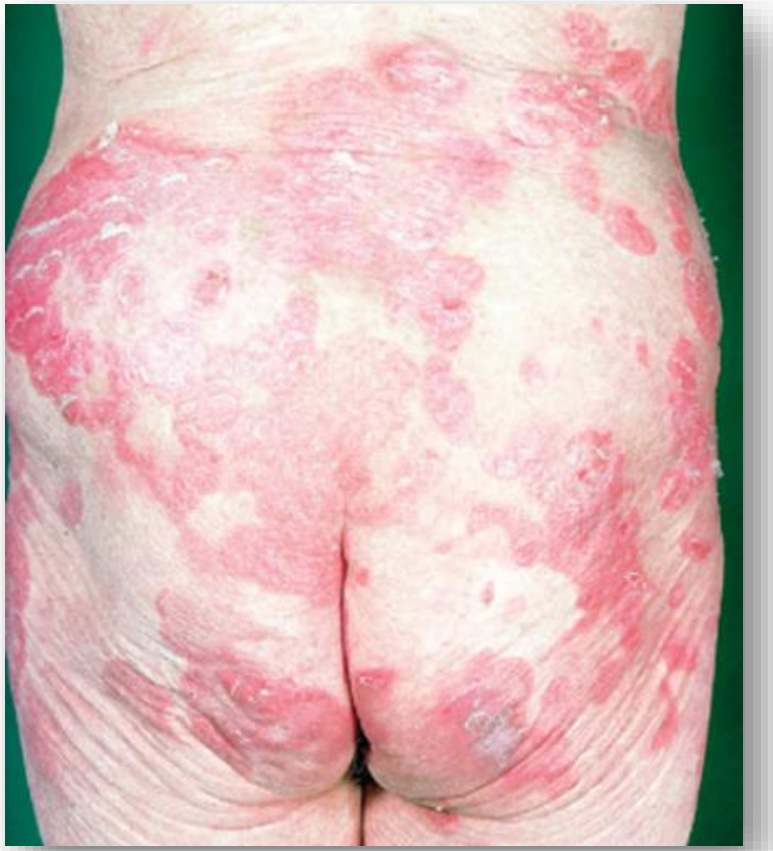
Causes highlighted with this color are discussed in the next slides the other cause are mentioned earlier

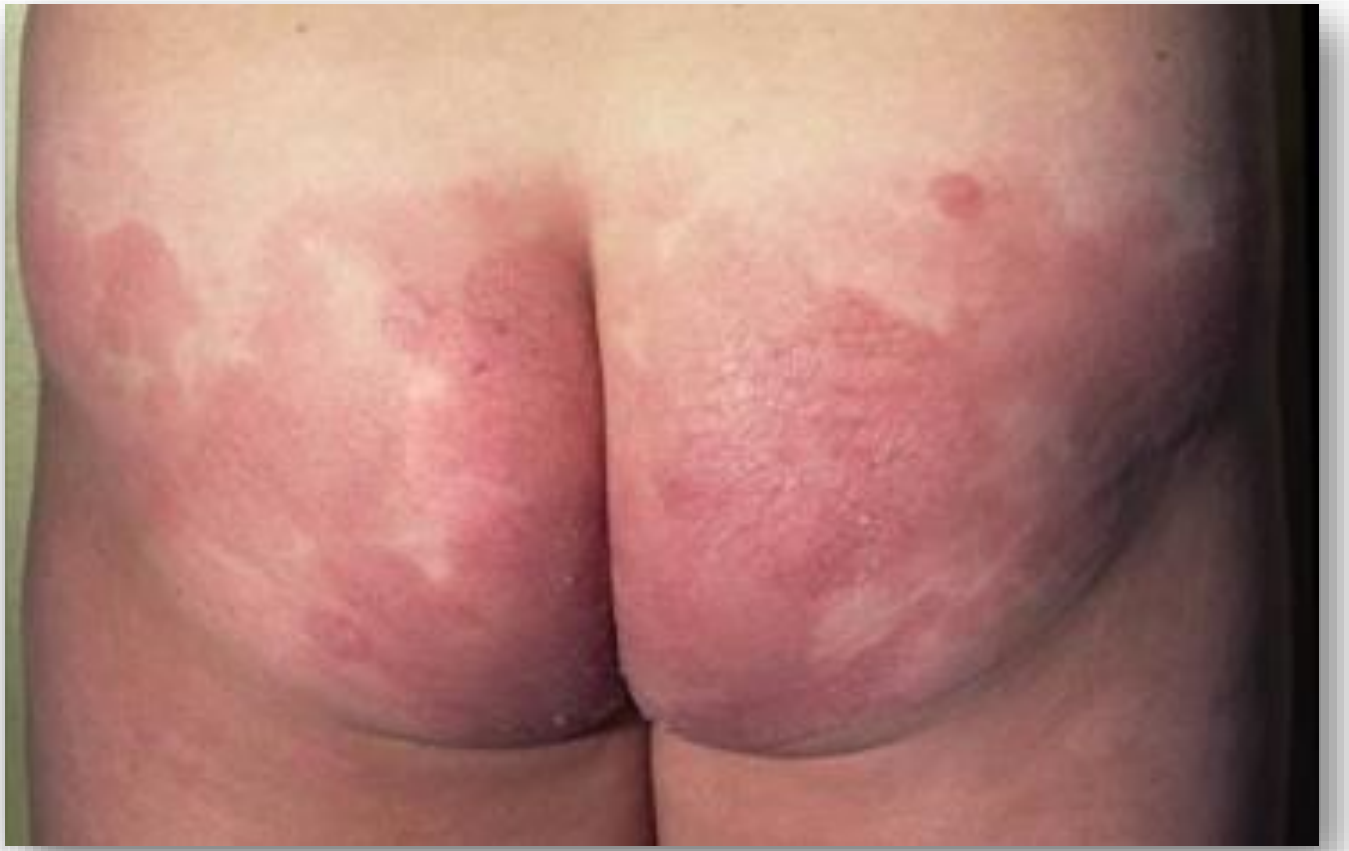
Xerosis

- Causes generalized pruritus.
- **Demographic**: Occurs on elderly due to skin dryness.
- **Treatment**: Improved by emollients.



Mycosis fundgoides (Cutaneous t cell lymphoma)





Areas of mycosis fungoides (cutaneous T-cell lymphoma). Multiple, superficial, scaly erythematous plaques of the buttocks and trunk.

Dermatological disease that cause localized pruritus

1. Lichen simplex chronicus (neurodermatitis).
2. Prurigo nodularis.
3. Pruritus ani.
4. Pruritus vulvae and scroti (Infection, Skin disease, Lichen planus, Psoriasis, Lichen sclerosus, Neoplasm, Paget`s disease, 7% psychogenic)
5. Scalp pruritus
6. Pruritus in scar

سنوات + جدعنه

Possible Questions

سنوات (1)

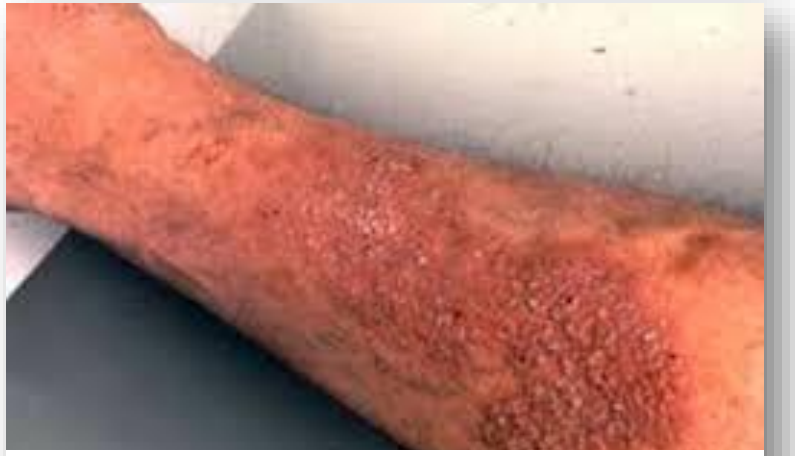
- Mention 3 skin diseases that cause pruritus
  - Any 3 from the previous slides

سنوات (3)

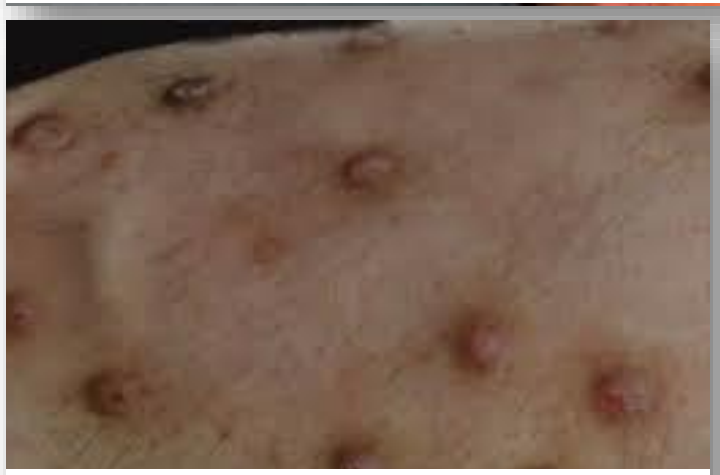
- Mention 2 causes of localized pruritus
  - Any 2 from the second slide
- Mention 2 skin diseases that cause localized pruritus
  - Any 2 from the second slide
- Mention 4 causes of generalized pruritis
  - Any 4 from the first slide
- Mention 4 skin diseases that cause generalized pruritis
  - Any 4 from the first slide

Lichen simplex chronicus and prurigo

- This difficult problem is sometimes called ‘neurodermatitis’.
- It is thought that constant irritation leads to constant scratching, which in turn leads to thickening of the skin.
- This may occur in plaques, known as ‘lichen simplex chronicus’, or in nodules, which are given the name ‘prurigo nodularis’.
- **Classic Sites**: Shins, forearms, Palms, back and neck
- **Treatment**: Potent topical steroids (sometimes under occlusive bandages) may help, but the problem often recurs.



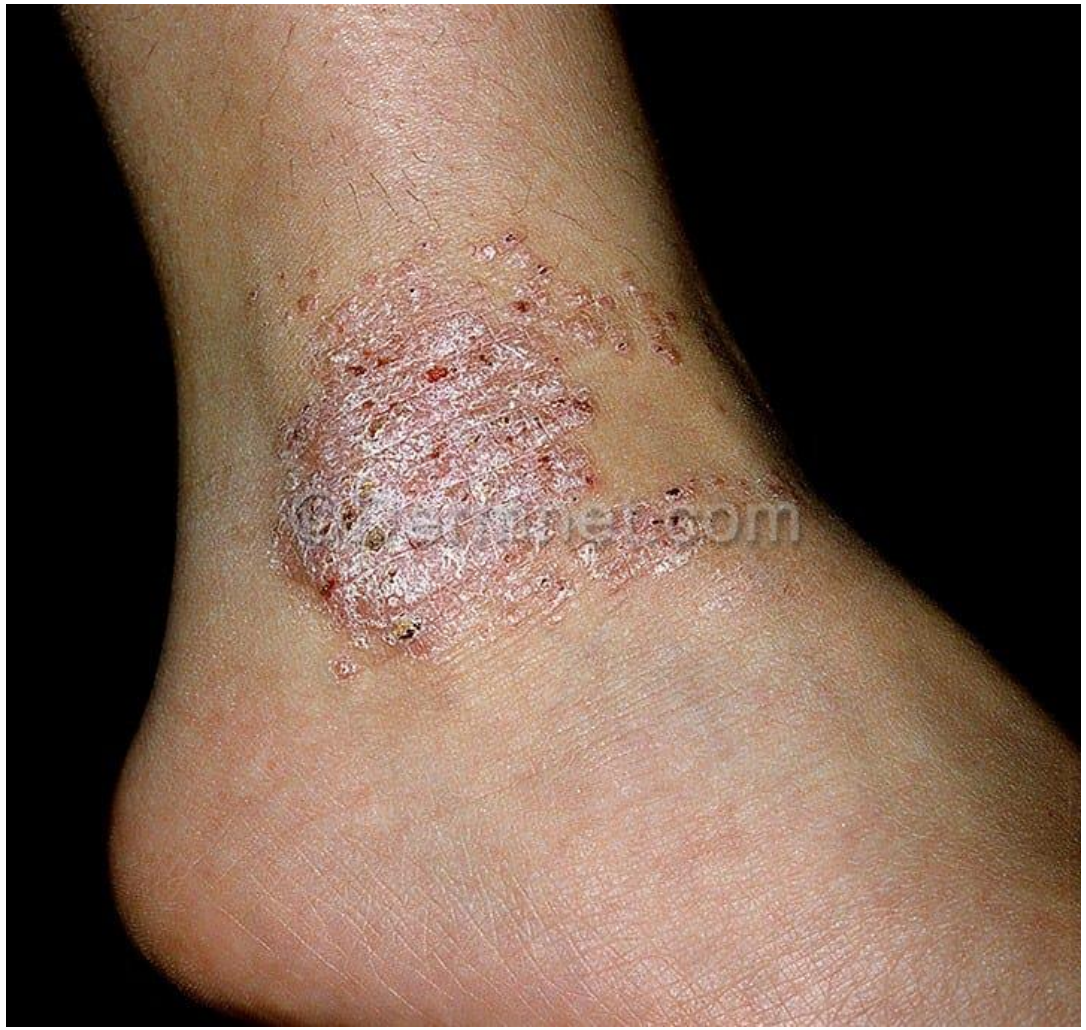
Lichen simplex chronicus



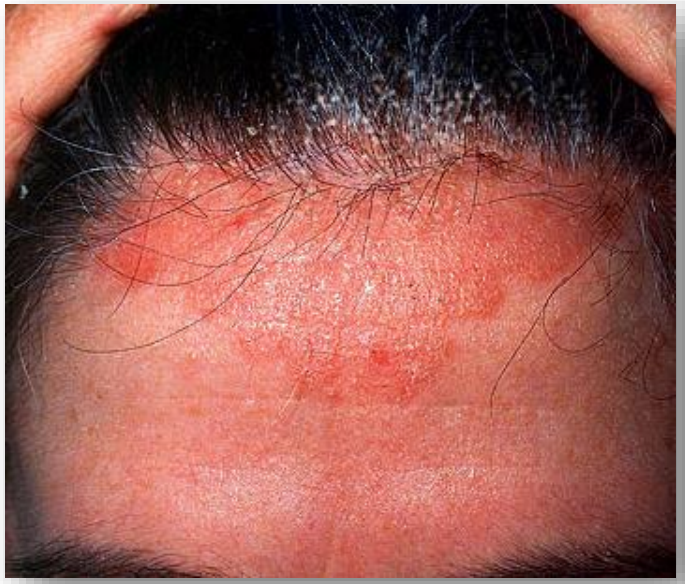
Prurigo nodularis

Pt presented single lesion with severe itching

- **What is your diagnosis ?**
  - Lichen simplex chronicus (neurodermatitis)
- **What other sites can be affected ?**
  - Forearms, Palms, back and neck
- **Treatment:**
  - Potent topical steroids

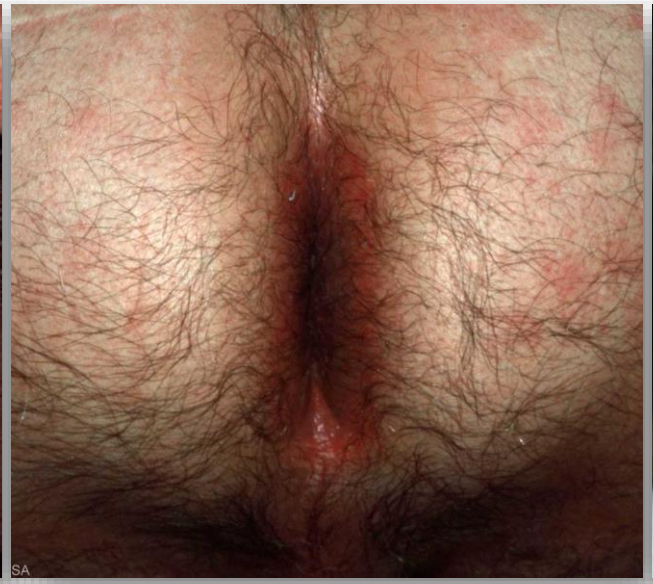


Localized pruritus



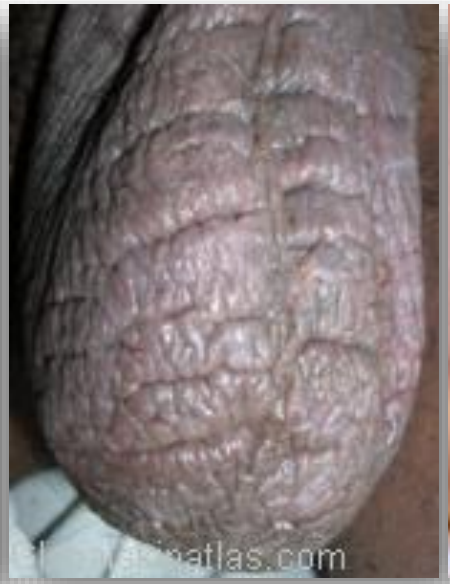
Seborrheic dermatitis

Result in scalp pruritus



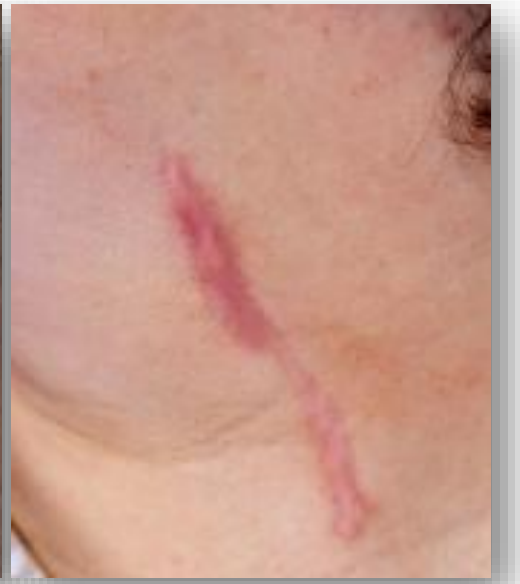
Pruritus ani

Erythematous lesions on the anal skin due to itching



Pruritus scrota

Thickening of the scrotal skin due to itching



Scar pruritus

associated with normal wound healing, nerve regeneration.

Pruritus & systemic disease

1. **Renal pruritus**
   - Mechanism still unknown
   - **Site**: Can be localized or generalized
   - **Treatment**: Gabapentin 200-300mg after hemodialysis session
   - **Definitive treatment**: Renal transplantation
2. **Cholestatic pruritus**
   - **Cause**: Any liver disease can cause pruritus.
   - **Timing**: Worse at night.
   - **Site**: Worse on hands and feet and body regions constricted by clothing.
   - **Treatment**: treating and removing the primary cause, ex. remove gallbladder stones.

Pruritus & systemic disease

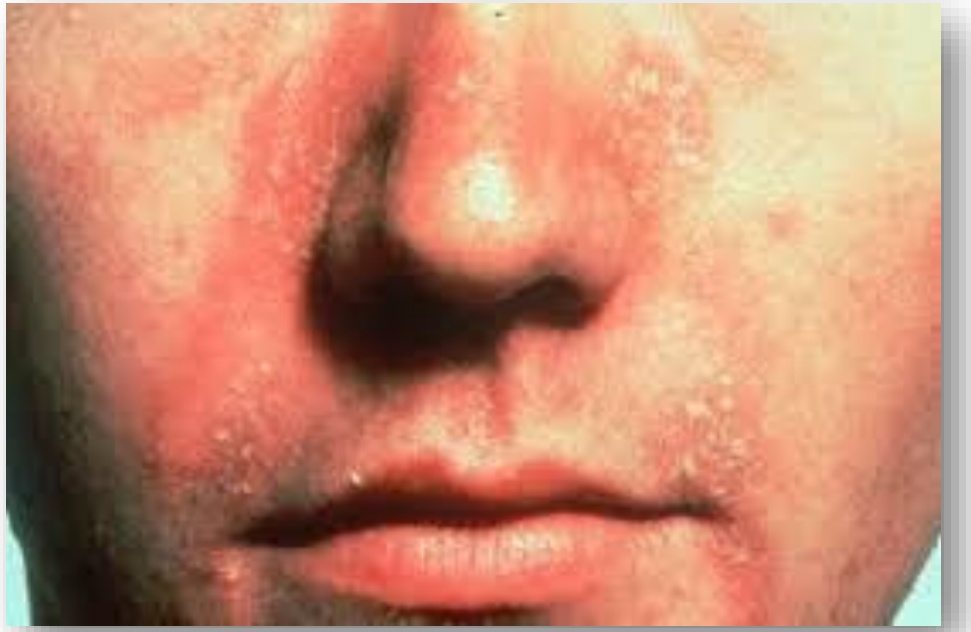
3. **Hematologic pruritus**
   - **Causes**: Iron deficiency, Polycythemia rubra vera
   - **Treatment**: Aspirin 300 mg t.i.d, phototherapy
4. **Pruritus and malignancy**
   - Any malignancy can induce pruritus as a paraneoplastic phenomenon
   - **Hodgkin disease**: strong association
   - **Non-Hodgkin's lymphoma**: less common (2%)
   - **Leukemia**: Chronic Lymphocytic Leukemia

Pruritus & pregnancy

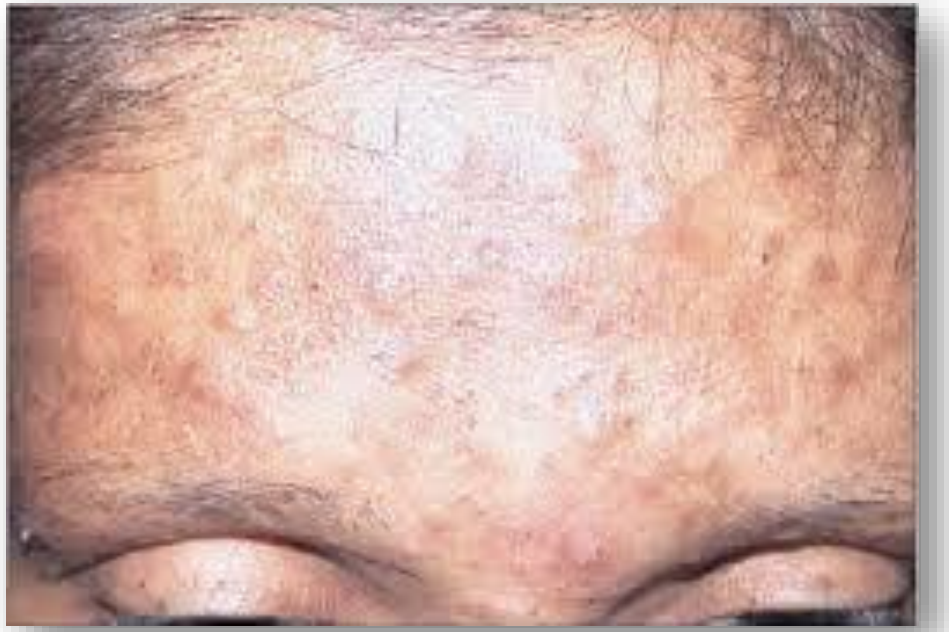
- **Dermatosis of pregnancy**: pemphigoid gestationes, pruritic urticarial plaques and papules of pregnancy, Prurigo of pregnancy, cholestasis of pregnancy.
- Cholestasis of pregnancy:
  - Generalized pruritus with or without jaundice
  - Absence of primary skin lesions
  - Biochemical abnormalities consistent with cholestasis
  - Disappearance of signs and symptoms after delivery
  - Recurrence during subsequent pregnancies
  - Increased serum bile acids (cholic acid, deoxycholic acid, chenodeoxycholic acid)
  - Prothrombin time should be monitored because Vit.K deficiency can occur due to impaired absorption
  - **Treatment**: cholestyramine, phototherapy, urodeoxycholic acid 15 mg\kg\day.

Pruritus in HIV infection and AIDS

- Severe pruritus is common.
- AIDS patients may develop several pruritic conditions like, severe seborrheic dermatitis, eosinophilic folliculitis.



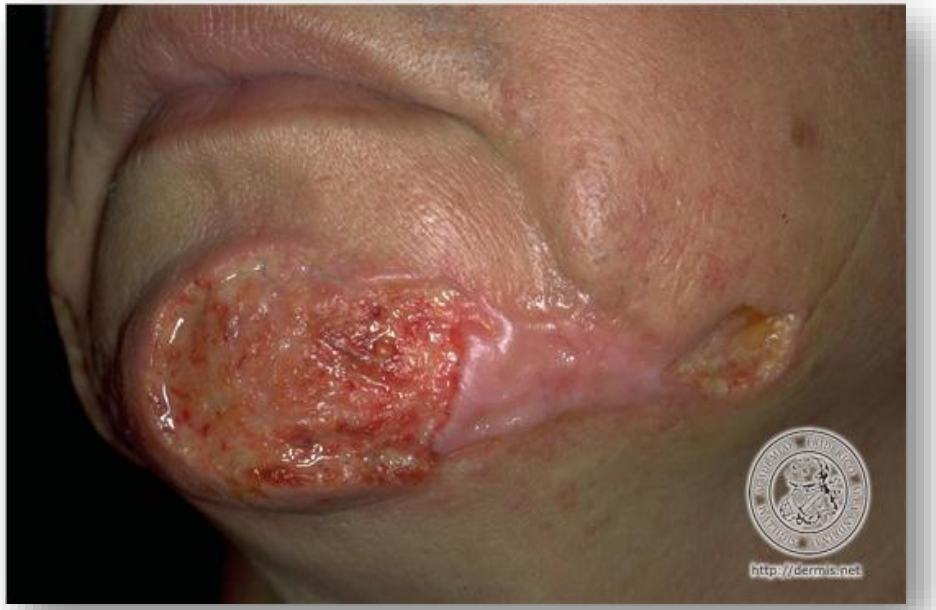
Erythema on the nasolabial folds and face.
(Severe seborrheic dermatitis).



Very itchy inflammatory infiltrate which is seen under the microscope occurring in the face, upper chest and upper back. (Eosinophilic folliculitis).

Psychogenic pruritus

- **Excoriation disorder**: recurrent skin picking resulting in lesions and significant distress or impairment in daily functioning
  - Involves repeated attempts to decrease or stop picking
  - Usually begins in adolescence
  - The majority of affected individuals are female and often have comorbid obsessive-compulsive disorder, trichotillomania, or major depressive disorder.



Consultation with psychiatrist is recommended

Investigations if systemic cause is suspected

1. CBC
2. KFT
3. LFT
4. Urine analysis
5. ESR
6. Chest X-Ray
7. Hepatitis profile
8. Fasting Blood Sugar (F.B.S)

Skin manifestations of internal diseases

Scope

1. Skin and internal malignancy
2. Skin and endocrine disease
3. Skin and CT diseases
4. Skin and sarcoidosis
5. Skin and renal disease
6. Skin and liver disease
7. Skin changes in malabsorption and malnutrition
8. Xanthomas
9. Skin and pregnancy
10. Skin and Bacterial endocarditis
11. Skin and Behcet’s disease
12. Skin and IBD

Skin and internal malignancy

Paraneoplastic Dermatosis

- Disorders associated with malignancy in most or all cases.
- Disorders strongly associated with malignancy.
- Dermatosis that may be associated with cancer.
- Familial cancer syndromes and skin.

- Mention 5 skin manifestation with paraneoplastic
  - Any 5 manifestation of the 4 categories

Mention 2 manifestations

سنوات (2)

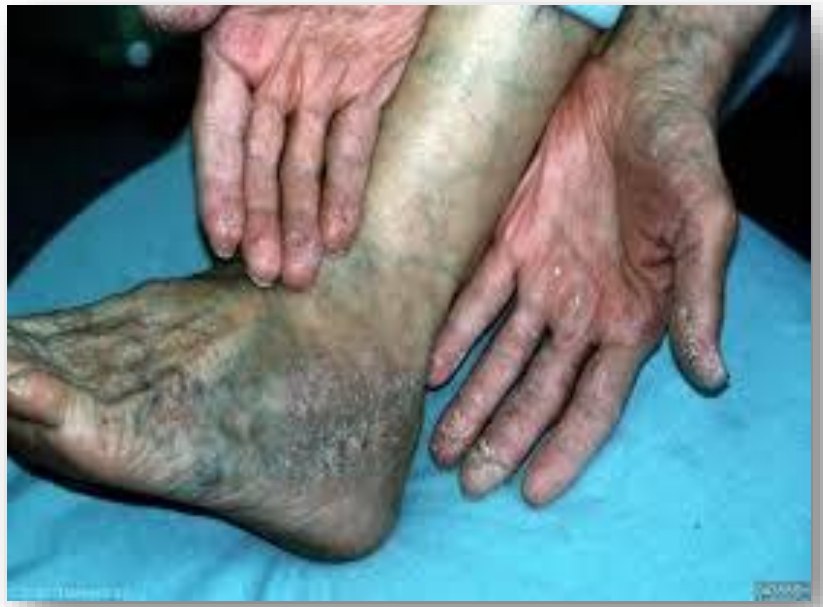
شرح

Disorders almost always associated with malignancy

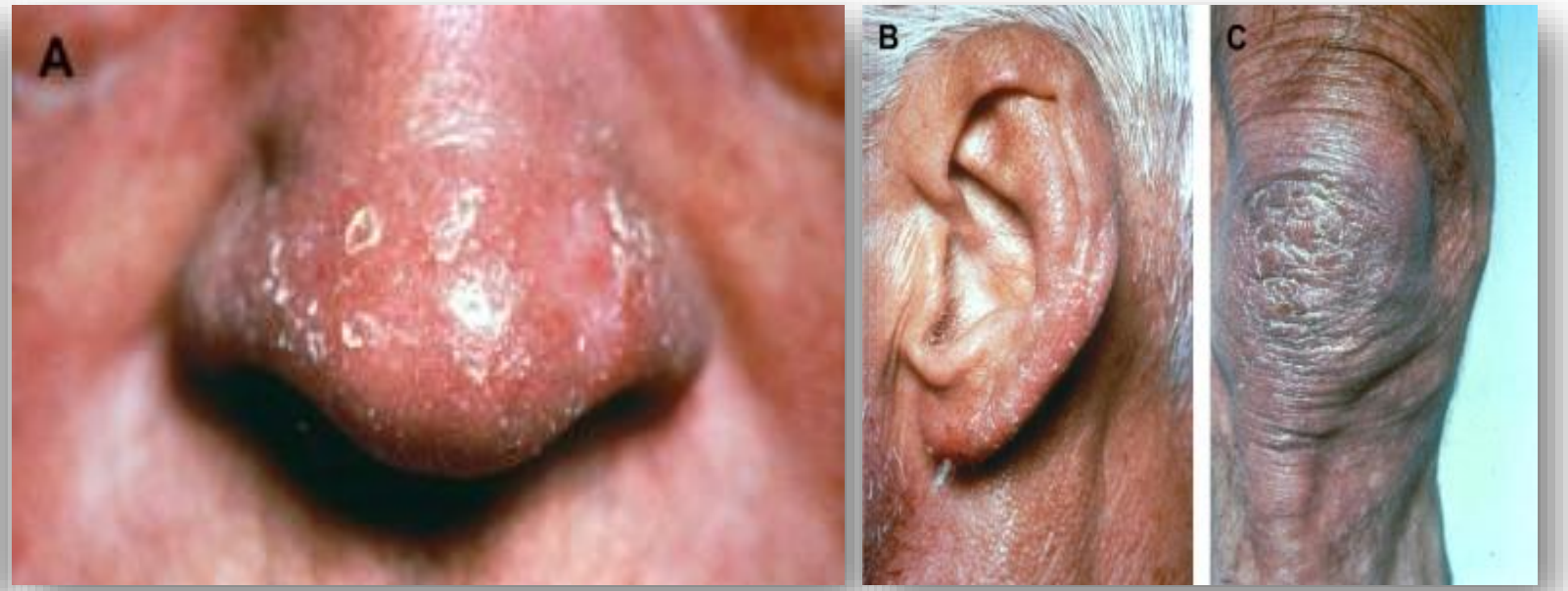
1. **Bazex syndrome (Acrokeratosis paraneoplastica):** Acral lesion involving nose and helices (larynx, pharynx, esophagus ca)
2. **Glucagonoma syndrome:** Necrolytic migratory erythema, associated with pancreatic tumor
3. **Paget`s disease of the breast:** associated with ductal breast cancer
4. **Carcinoid syndrome:** Flushing and erythema of head and neck
5. **Erythema gyratum repens:** Appearance of grains of Wood
6. **Ectopic ACTH syndrome:** Hyperpigmentation and features of Cushing's syndrome
7. **Paraneoplastic pemphigus:** associated with lymphoma, Leukemia
8. **Acquired hypertrichosis lanuginosa**
9. **Tripe palms**: thickened palms associated with GI malignancy

Bazex syndrome

- Paraneoplastic acrokeratosis, or Bazex syndrome is a cutaneous condition characterized by psoriasiform changes of hands, feet, ears, and nose, with involvement of the nails and periungual tissues being characteristic and indistinguishable from psoriatic nails.
- The condition is associated with carcinomas of the upper aerodigestive tract.



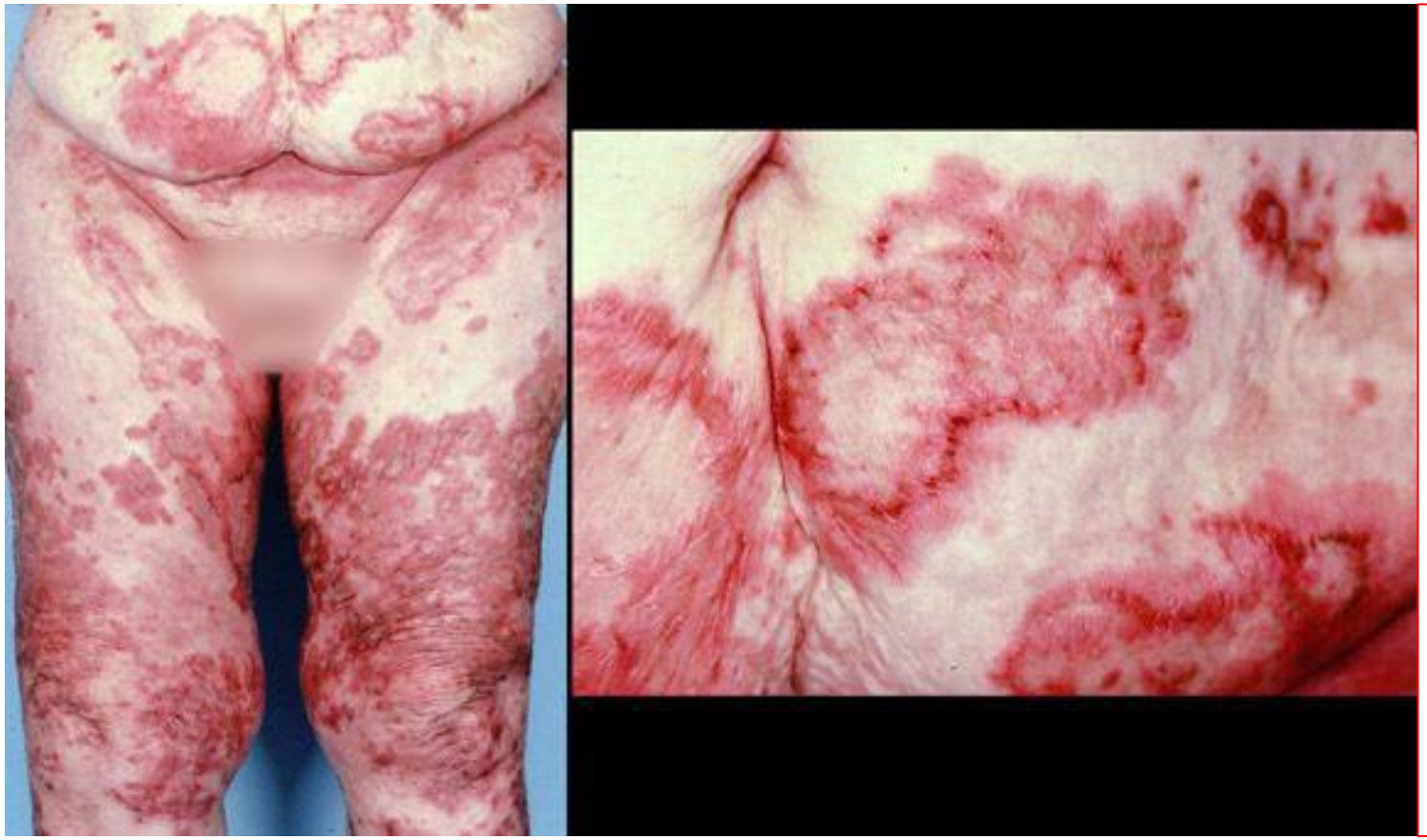
Acral erythema and scaling (tips of fingers and feet)



Erythema and scaling on the tip of the nose , ear helices and the knee.

Glucagonoma syndrome

❖Necrolytic migratory erythema, must take a skin biopsy



What is your diagnosis?

A. Dermatomyositis

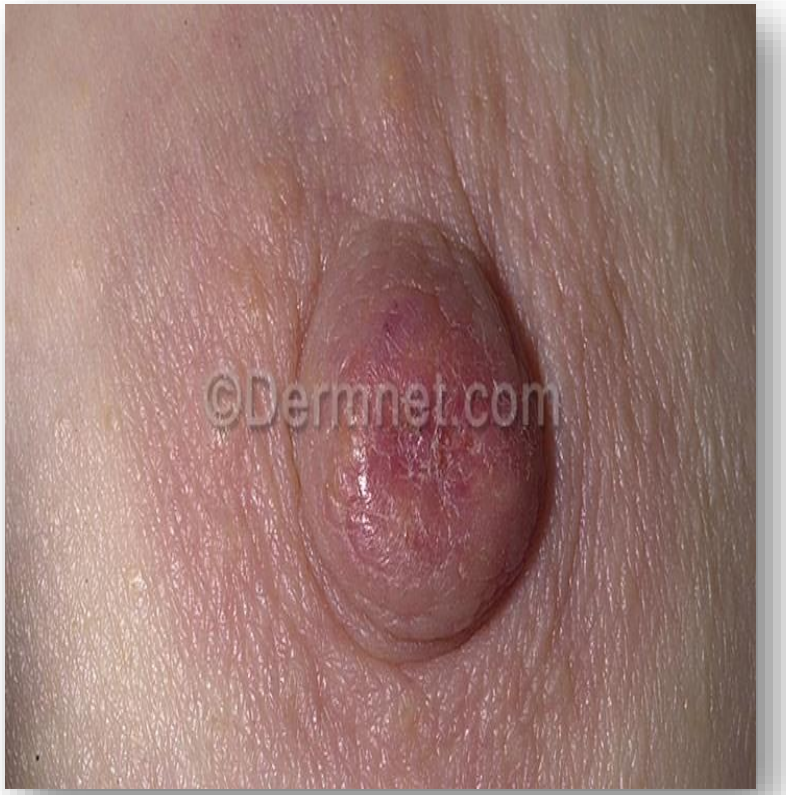
B. Pancreatic tumor (Glucagonoma syndrome)

C. Bazex syndrome

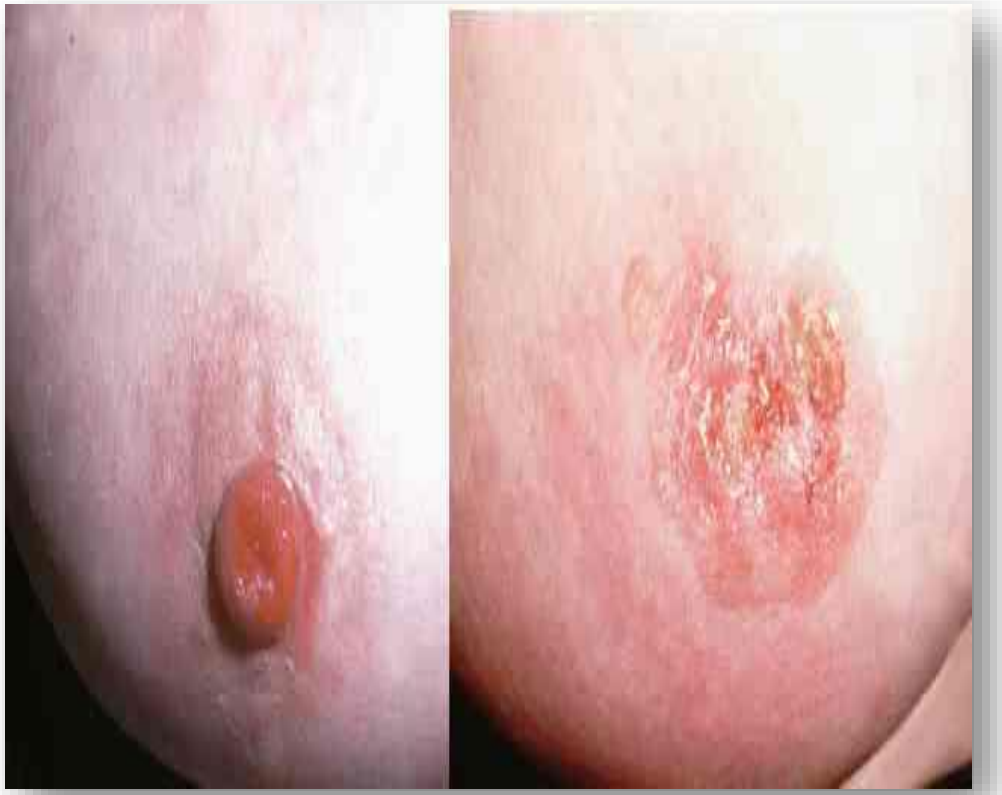
D. Xanthoma

سنوات (1)

Paget`s disease of the breast

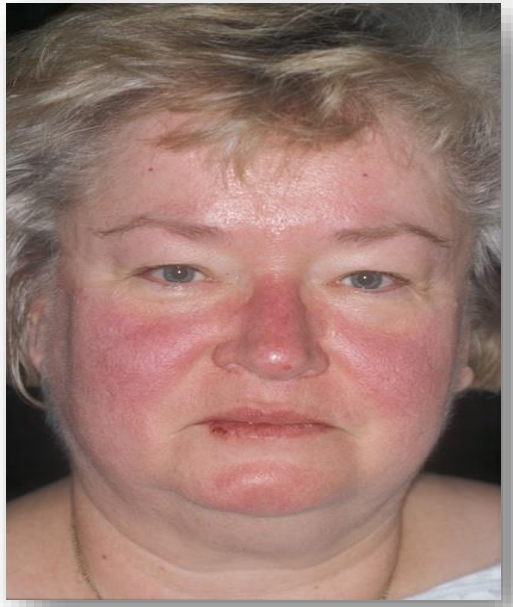


Early manifestation of Paget's disease: Erythema and scaling, skin biopsy will confirm the diagnosis



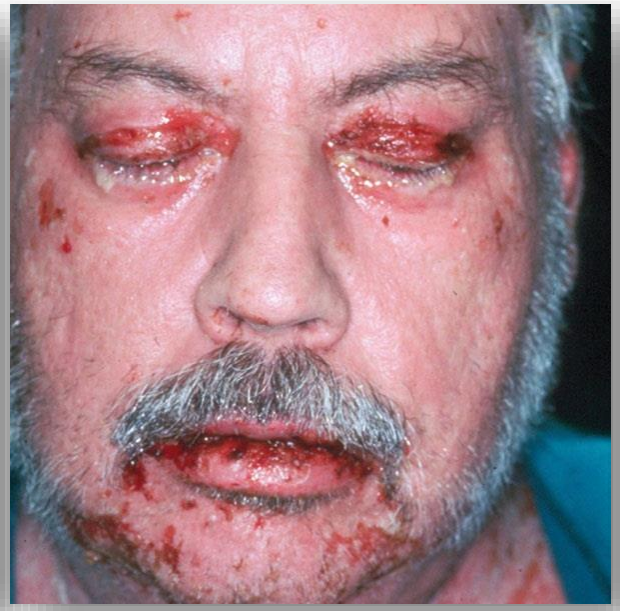
Erythema, scaling and destruction of the nipple, must take a skin biopsy.

Disorders almost always associated with malignancy

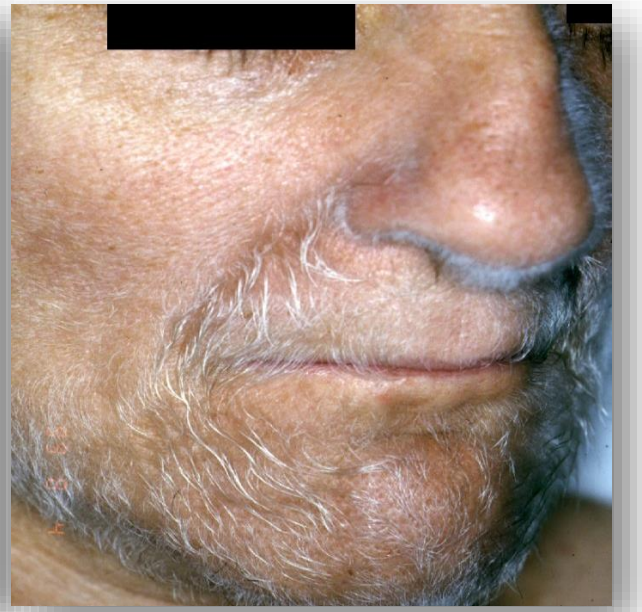


Flushing of the face (Erythema)

Cushingoid features



Paraneoplastic pemphigus



Long, white Lanugo hair (Acquired hypertrichosis lanuginosa)

Paraneoplastic pemphigus

- Severe erosions not responding to treatment
- This disease is characterized by severe involvement of the mucous membranes and has a characteristic immunofluorescence, usually associated with hematological malignancy.

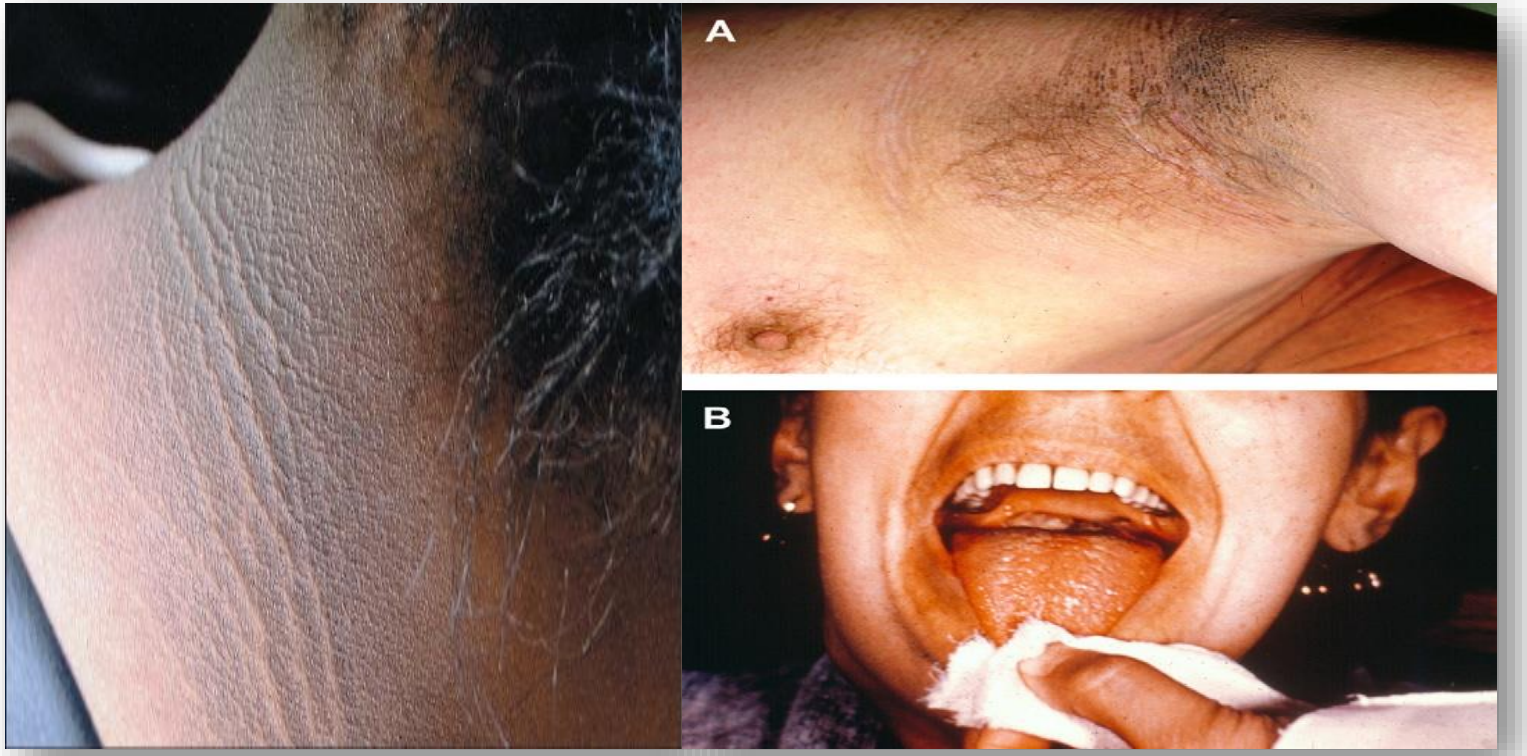
Disorders strongly associated with malignancy

Around 30% of cases

1. **Acanthosis nigricans**: associated with cancers of GIT and GUT
2. **Dermatomyositis in adults**: associated with Ovarian, lung, colorectal carcinoma
3. **Anti-epilegrin cicatricial pemphigoid**, one third of patients develop cancer
4. **Extramammary Paget's disease**: subtype of bullous pemphigoid (scaly plaque on anogenital area, associated with caner of GIT and GUT)
5. **Neutrophilic dermatosis**, **Sweet's syndrome:** 20% develop leukemia especially acute myelogenous leukemia.

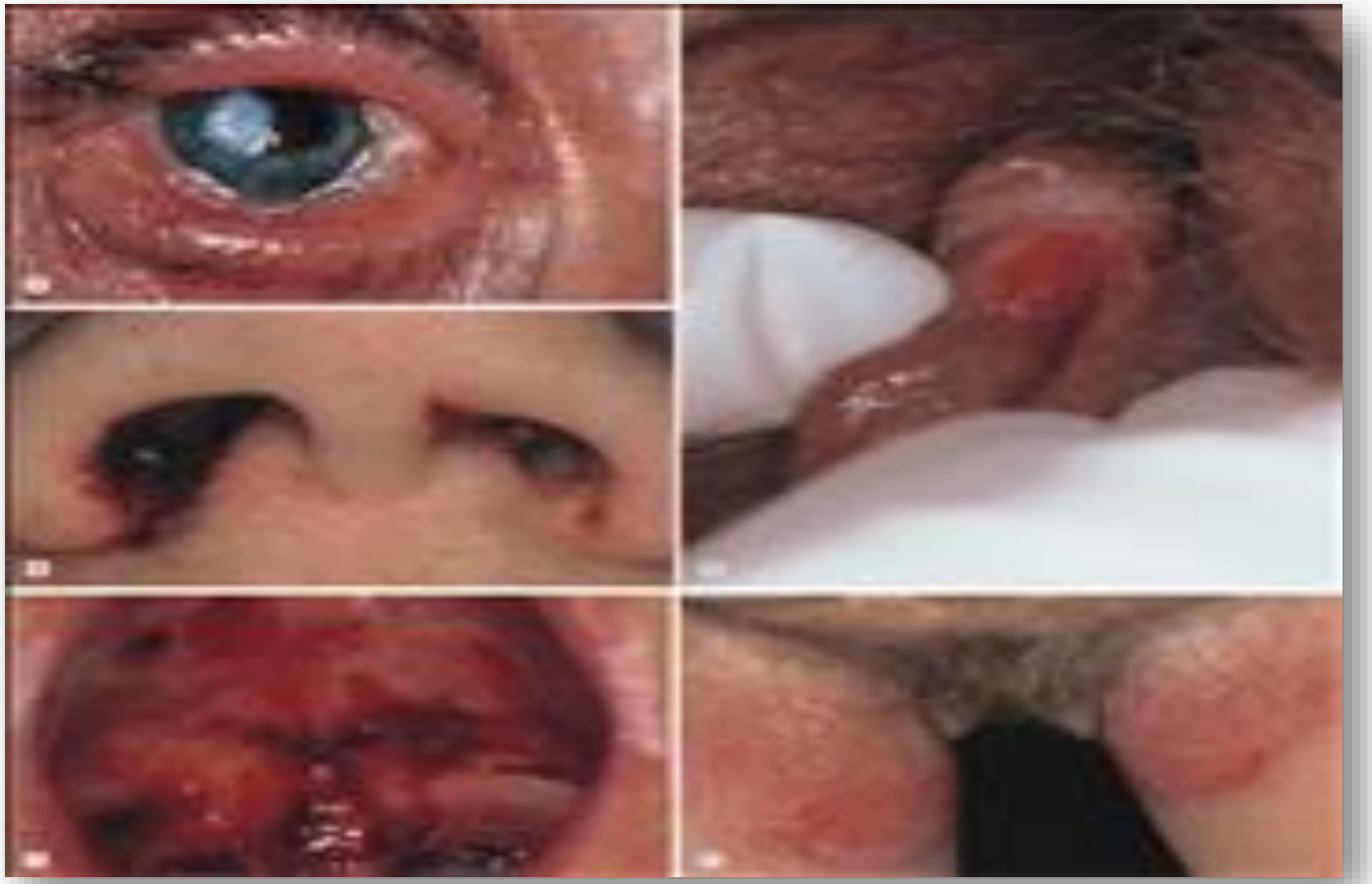
Malignant Acanthosis nigricans

- Hyperpigmentation and velvety thickening of the skin in any flexural area
- **The malignant type** is widespread and involving the mucous membrane
- **The benign type** (associated with DM , Obesity) isn't widely spread and does not involve the mucous membranes.

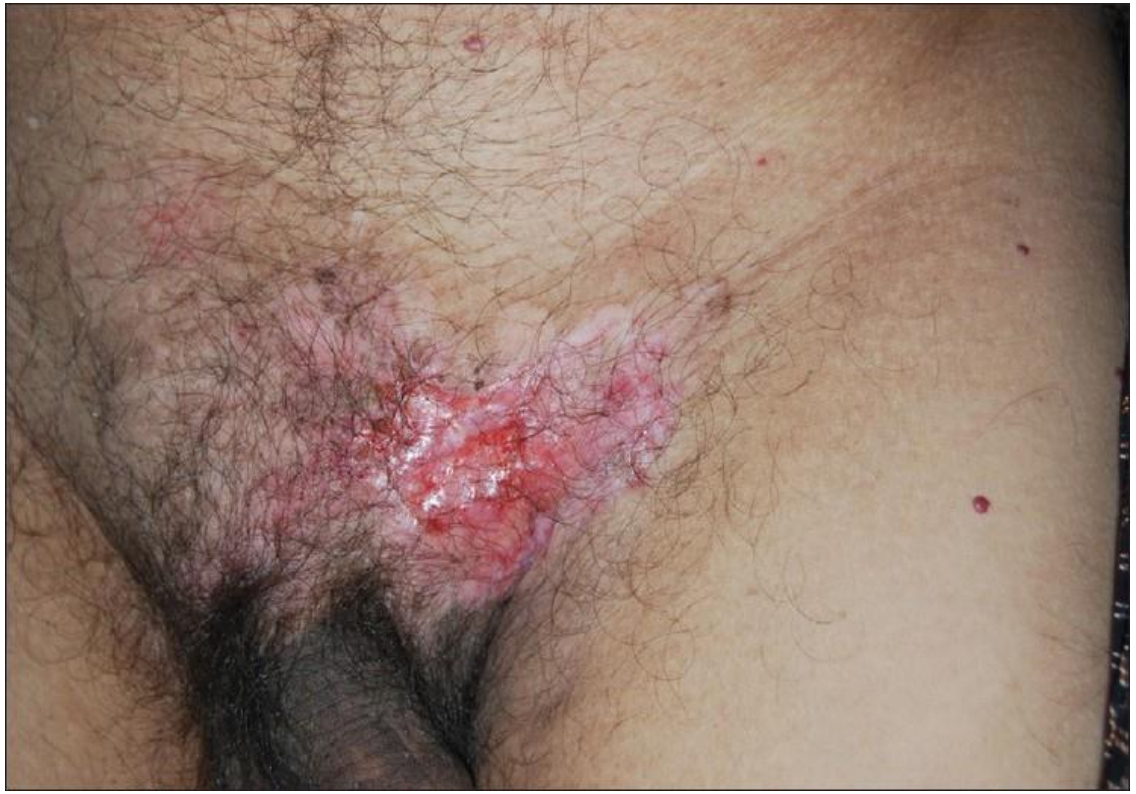


Anti-epilegrin cicatricial pemphigoid

- Autoimmune Subepithelial blistering disorder of the skin and mucous membranes leading to scarring.

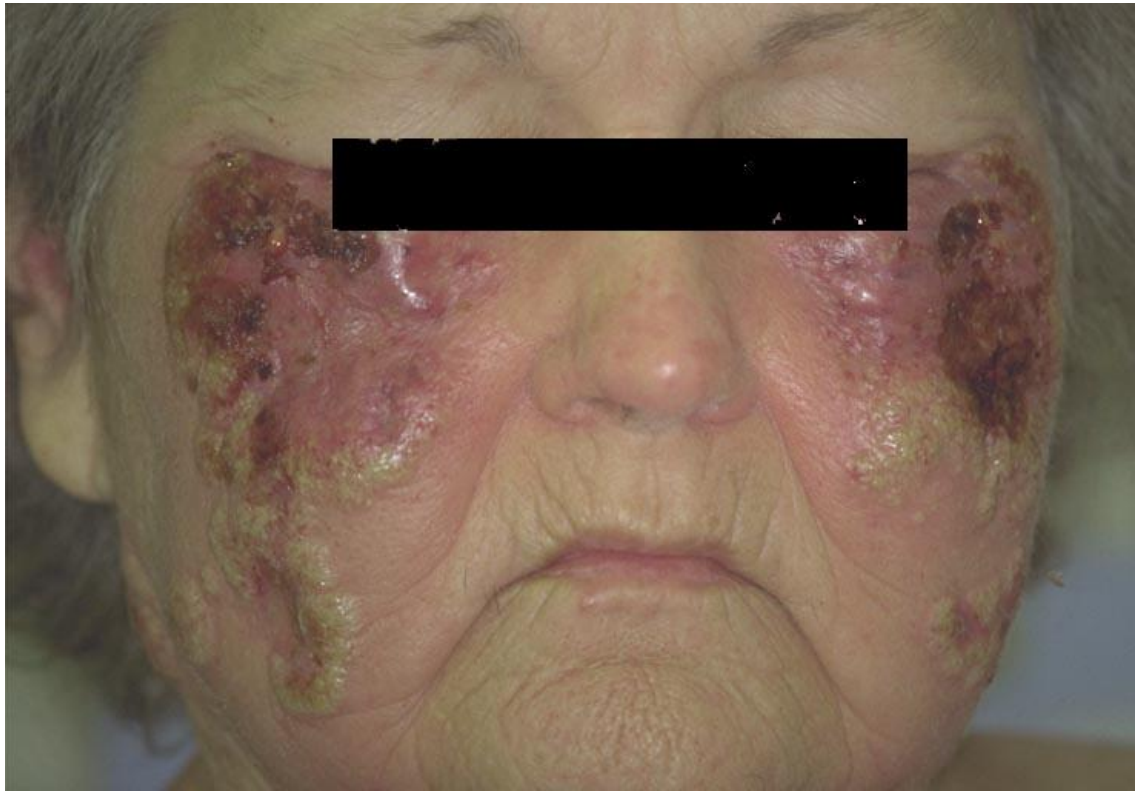


Disorders strongly associated with malignancy



Extramammary Paget`s disease

Eczematous lesions in the groins or peri-anal area not responding to steroids, antifungals, diagnosed by skin biopsy.



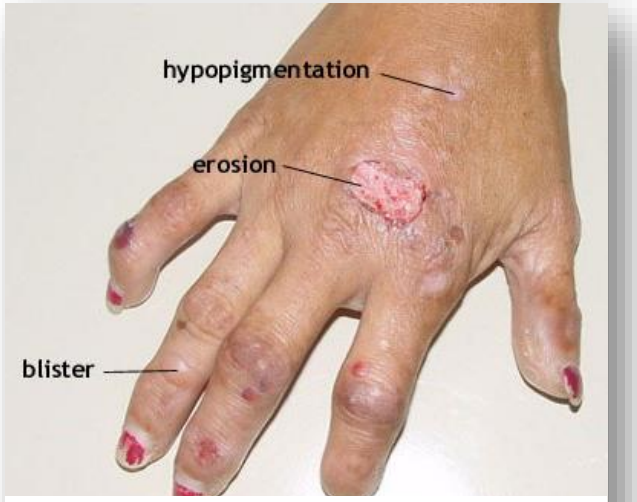
Sweet`s syndrome

Acute Edematous, tender lesions, investigations will show Leukocytosis, diagnosed by skin biopsy.

Dermatosis that may be associated with cancer

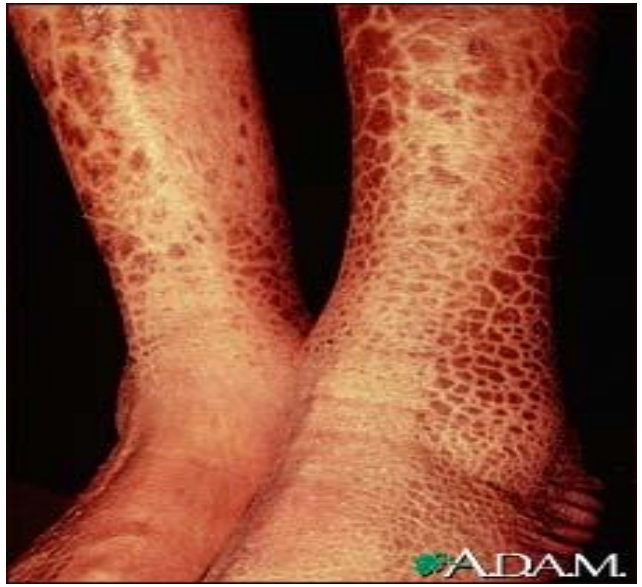
1. **Acquired ichthyosis**: may be associated with lymphoma
2. **Exfoliative erythroderma**: may be associated with lymphoma
3. **Necrobiotic xanthogranuloma**: may be associated with paraproteinemia
4. **Acquired Porphyria Cutanea Tarda**: may be associated with hepatic cancer

   - Patients present with Photosensitivity which is manifested by Erosions, Blisters and Hypopigmentation in the sun exposed areas, diagnosed by skin biopsy.



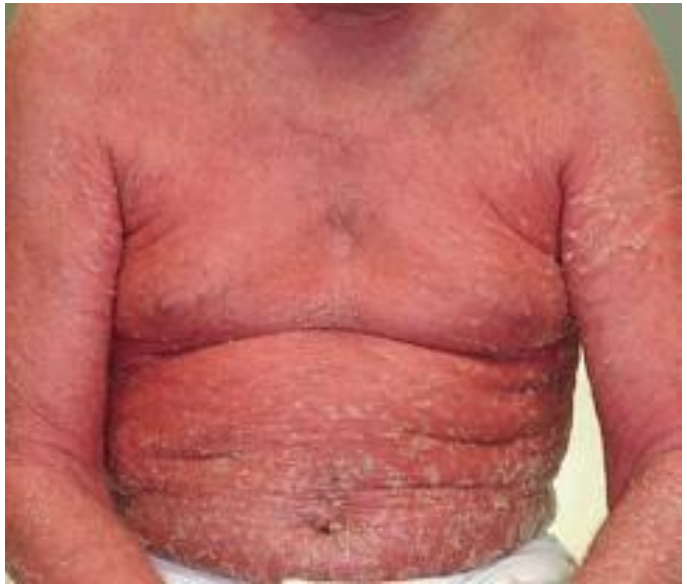
Porphyria cutanea tarda

Dermatosis that may be associated with cancer



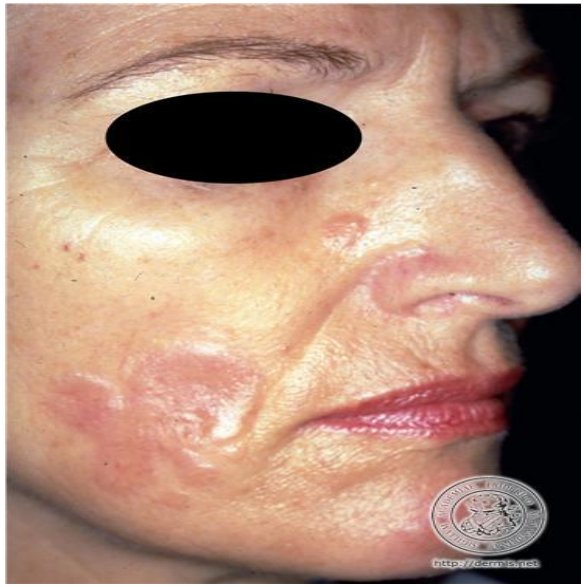
Acquired ichthyosis

Dry, rough skin with prominent scaling.



Exfoliative erythroderma

Widespread erythema and scaling.



Necrobiotic xanthogranuloma

Yellowish plaques on the face, could be Lymphoma, Sarcoidosis, etc.

So, diagnosis is by skin biopsy.

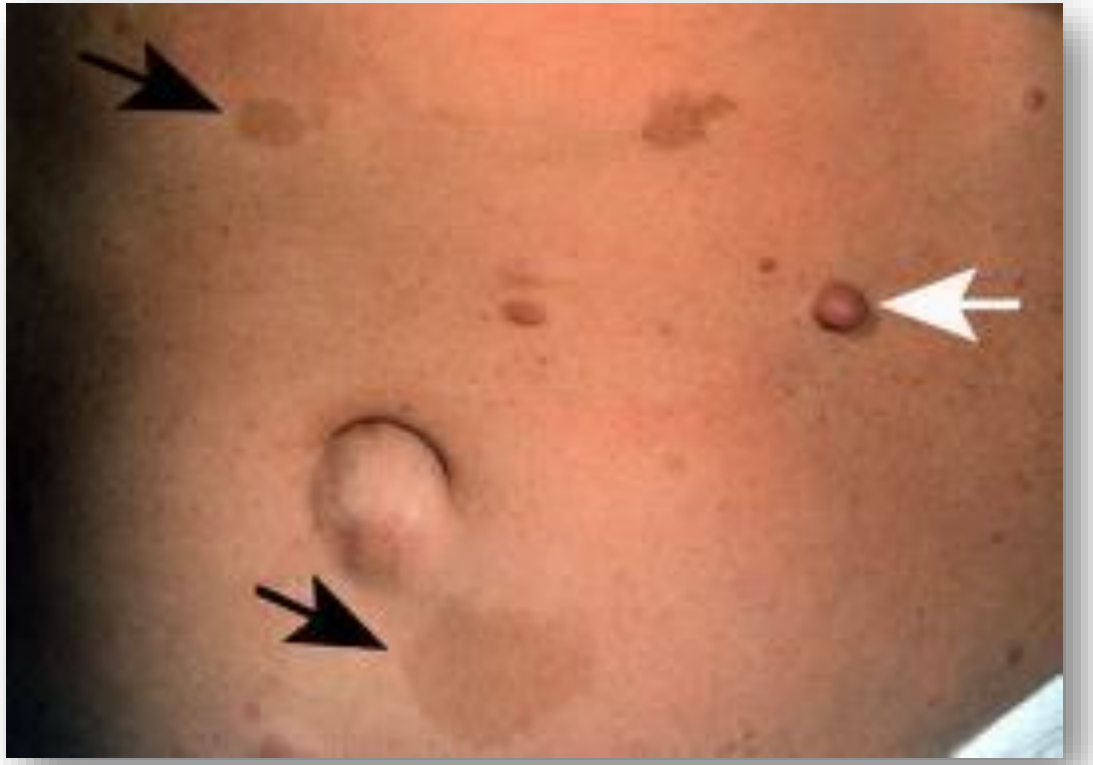
Familial cancer syndromes and skin

These syndromes has skin manifestations and have possible associated malignancies:

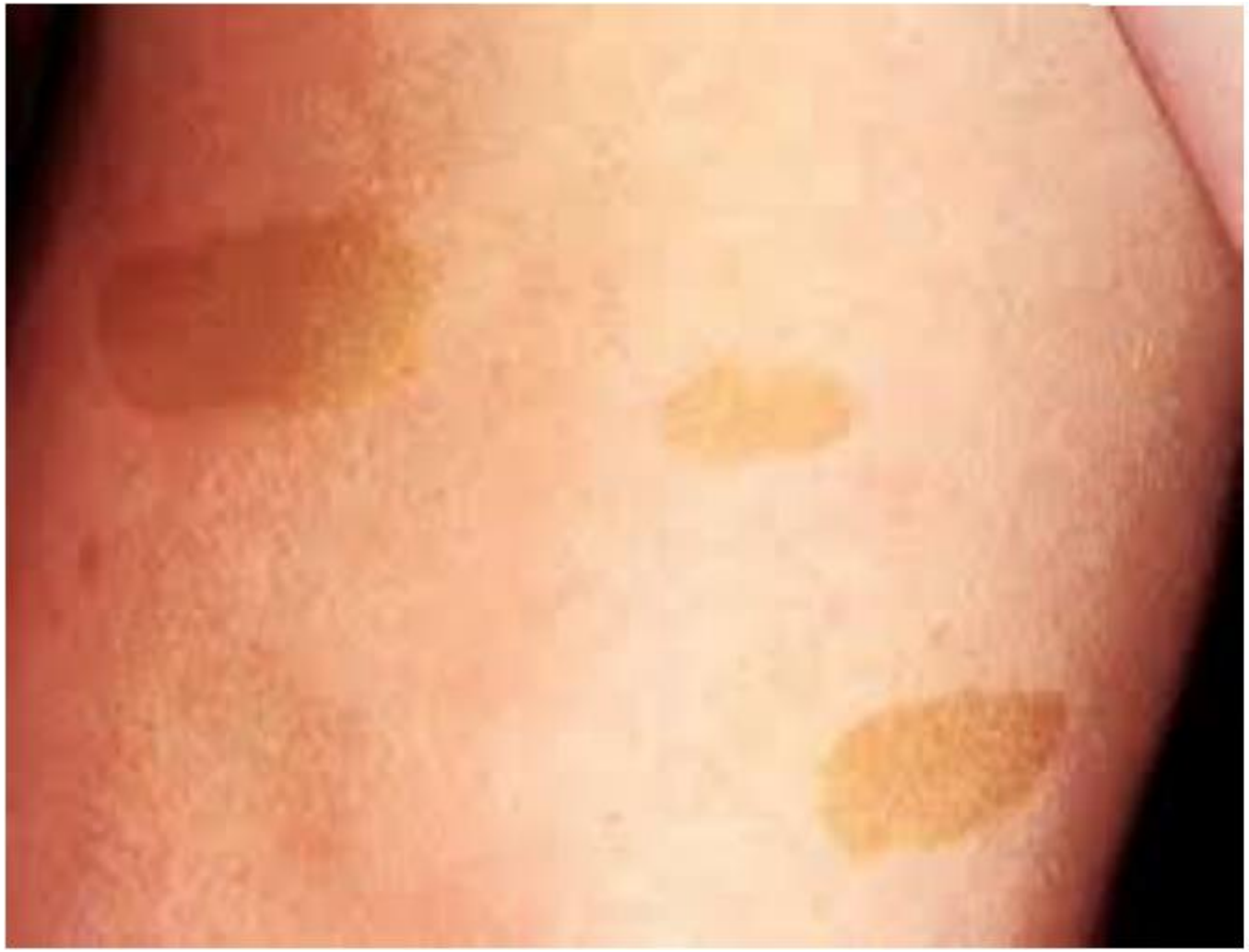
1. **Cowden`s disease**: Thyroid, breast, GIT carcinoma
2. **Muir-Torre syndrome**: Sebaceous tumors, GI carcinoma
3. **Gardner`s syndrome**: GIT carcinoma
4. **Ataxia telangiectasia**: Lymphoma, Leukemia
5. **Neurofibromatosis**: Kidney, Brain tumors

Neurofibromatosis

- Neurofibroma = White arrow
- Cafe’ au lait spots = Black arrows
- Diagnosed by skin manifestations



Neurofibromatosis



Café au lait macules and patch
If more than 6 and bigger than 1.5 cm (0.5 in children) then it is neurofibromatosis

Skin and endocrine disease

Diabetes mellitus, Thyroid diseases, Adrenal diseases

Mention 5 manifestations

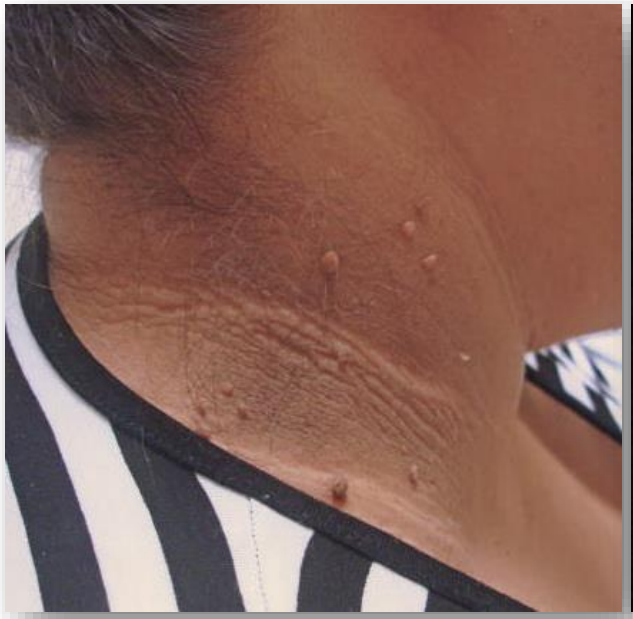
سنوات (1)

شرح

Skin manifestations of Diabetes Mellitus

1. Diabetic dermopathy (the most common skin manifestation of DM)
2. Acanthosis nigricans
3. Acral dry gangrene
4. Diabetic bullae
5. Diabetic cheiroarthrpathy
6. Disseminated granuloma annulare
7. Eruptive xanthoma
8. Necrobiosis lipoidica Diabeticorum
9. Neuropathic ulcer
10. Rubiosis: chronic flush of neck, face and upper extremities
11. Sclerodema adultorum of buschke
12. Hemochromatosis, bronzing of the skin due to melanin
13. Perforating skin disorder

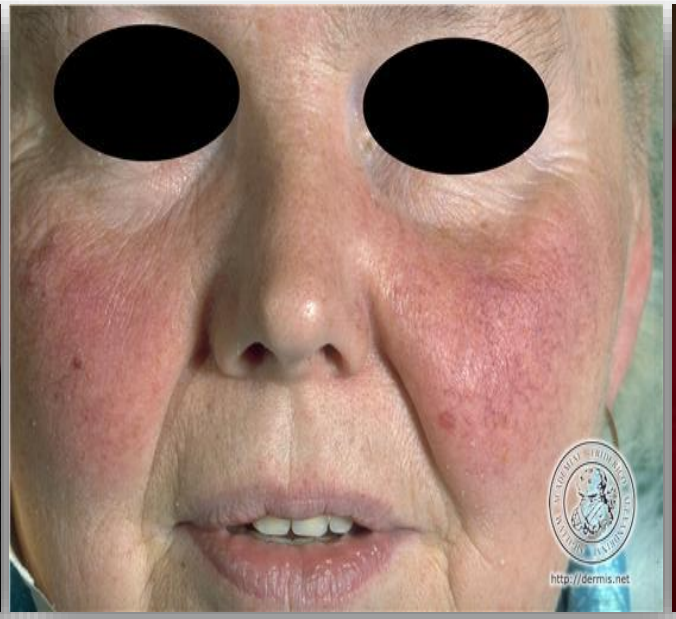
Skin manifestations of Diabetes Mellitus



Acanthosis nigricans

Diabetic bullae

Large, Few (1-2) blisters on the acral site



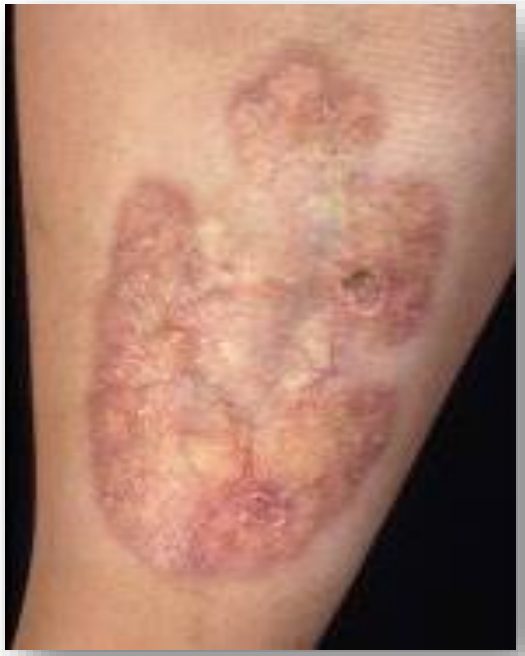
Rubeosis facie Diabeticorum

Facial flushing, could present on the neck and upper extremities.

Diabetic cheiroarthropathy

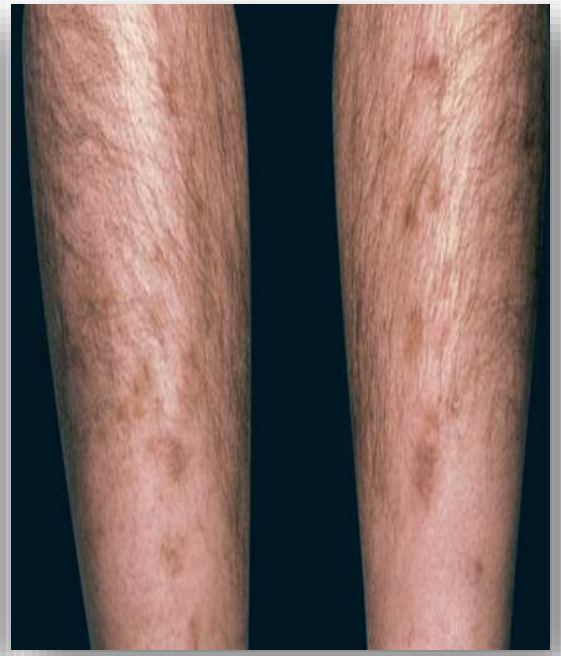
Inability to fully flex or extend the fingers

Skin manifestations of Diabetes Mellitus



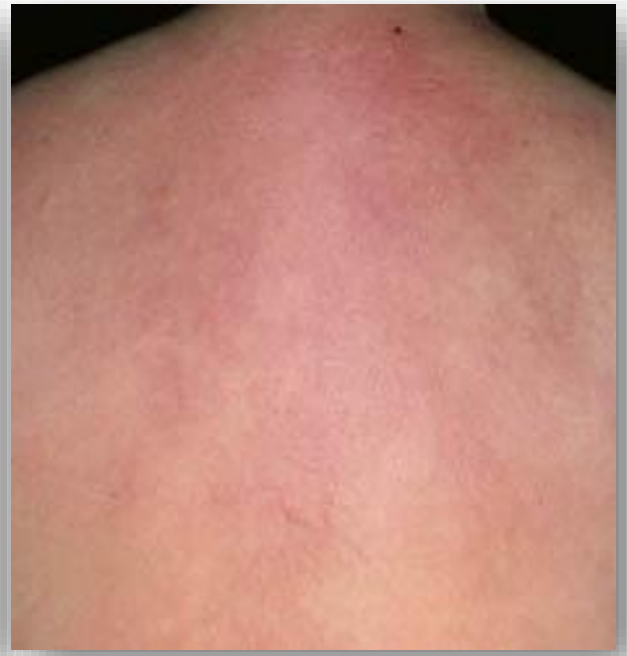
Necrobiosis Lipoidica Diabeticorum

Erythematous yellowish plaques with Telengectasia and atrophic center on the shins , could ulcerate



Diabetic dermopathy

Asymptomatic scar-like lesions, brown in color on the shins



Scleredema adultorum of Buschke

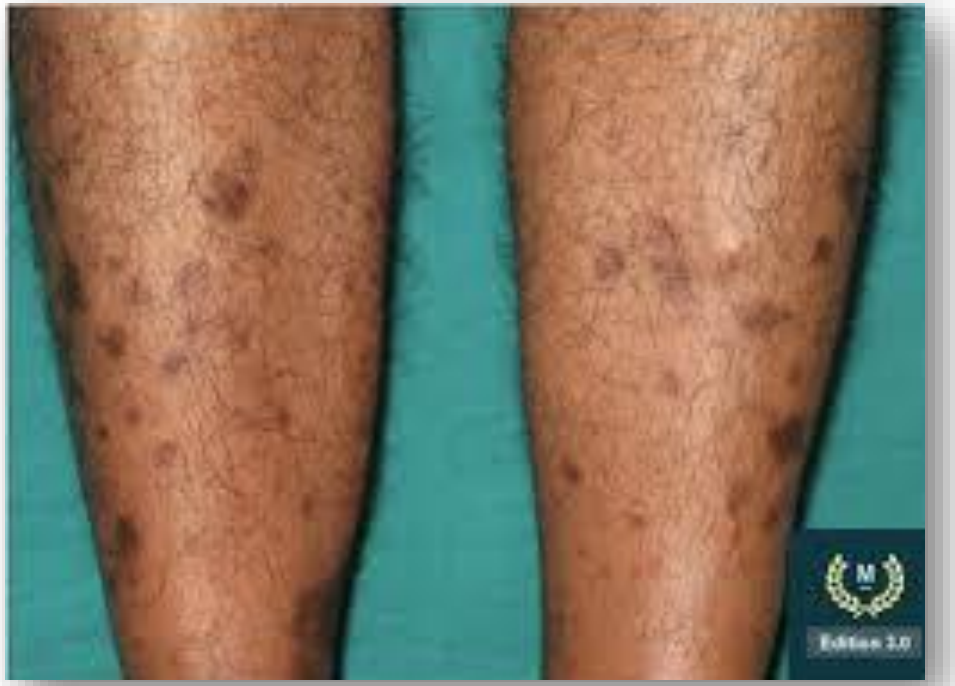
Erythema and thickening of the skin due to Mucin accumulation (Back, Chest and Face)

Granuloma annulare

Discolored plaques and papules in a ring pattern

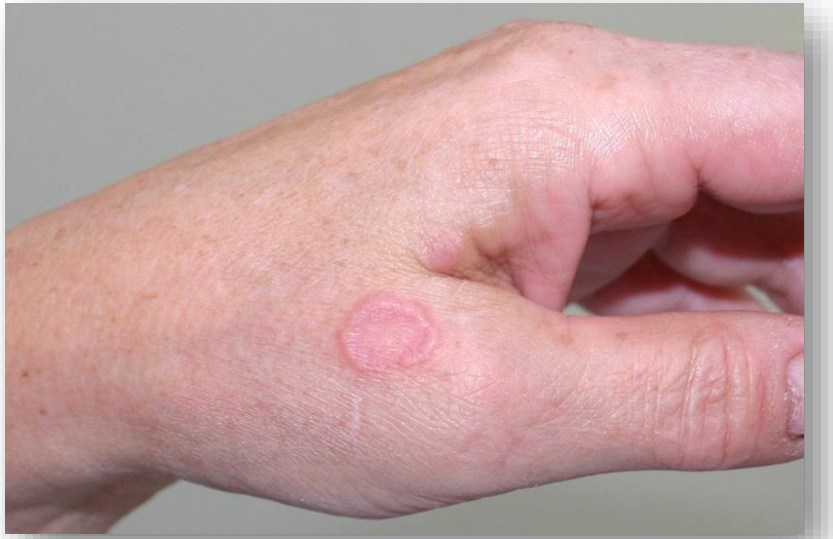
60-year-old male, long standing DM, diagnosis

- **Diagnosis**: Diabetic dermopathy
- **Characteristics**:
  - Small, brown, scar-like lesions seen on the shins in some people with diabetes.
  - The lesions are thought to be associated with diabetic microangiopathy



Granuloma annulare

- **Characteristics**:
  - Typically, lesions of granuloma annulare are groups of firm, skin-colored papules, often arranged in rings, and commonly occurring on the dorsa of the hands and feet
- **Course**: spontaneous resolution
- **Treatment**: if persistent, intralesional triamcinolone or cryotherapy
- **Note**: Have a weaky association with DM



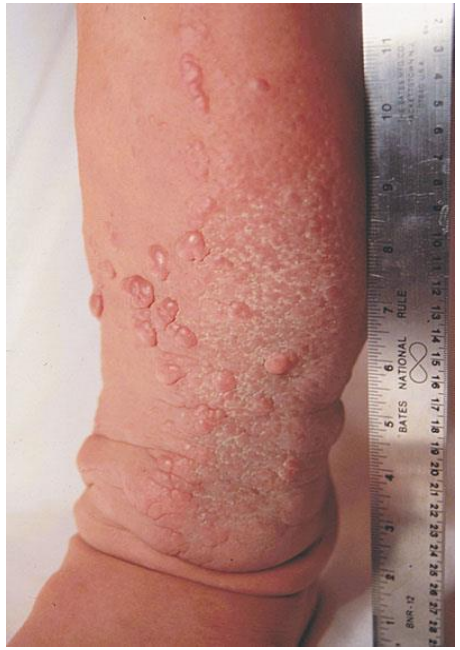
What is the diagnosis ?

- Ganuloma annulare

Skin manifestations of hyperthyroidism

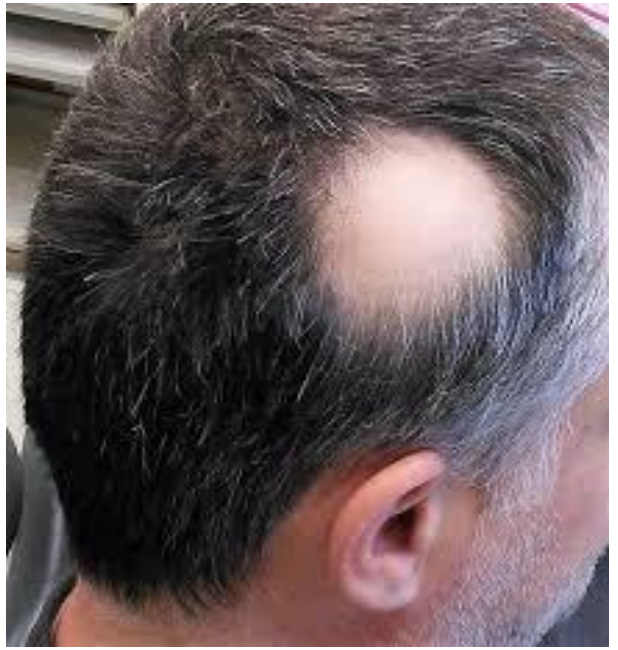
- **Cutaneous changes**: Fine, velvety, smooth warm and moist (increased sweating), hyperpigmentation, pruritus.
- **Cutaneous disease**: pretibial myxedema , thyroid Acropachy (Clubbing), urticaria, dermographism and vitiligo.
- **Hair changes**: Fine, thin, mild diffuse alopecia.
- **Hair disease**: Alopecia areata.
- **Nail Changes**: Onycholysis, koilonychia, clubbing.
- **Investigations**: TSH, T3, T4, Anti-Thyroperoxidase, anti-Thyroglobulin antibodies

Skin manifestations of hyperthyroidism

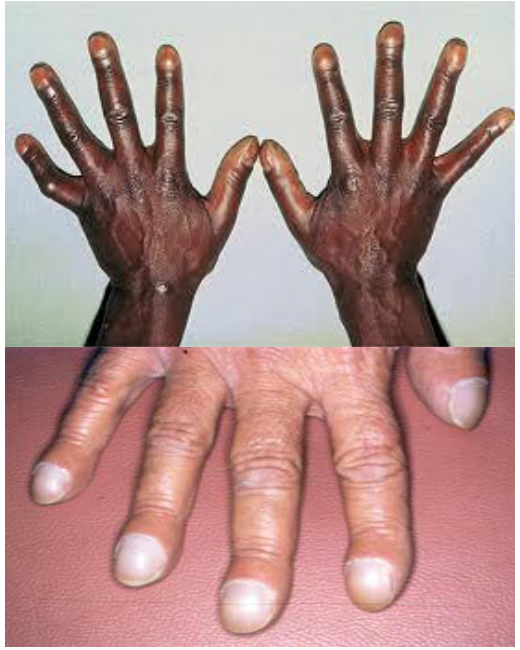


Pretibial myxedema

Onycholysis



Alopecia areata



Clubbing

Pretibial myxedema

- Thickened skin with verrucous lesions due to Mucin deposition

Mention 5 manifestations

سنوات (1)

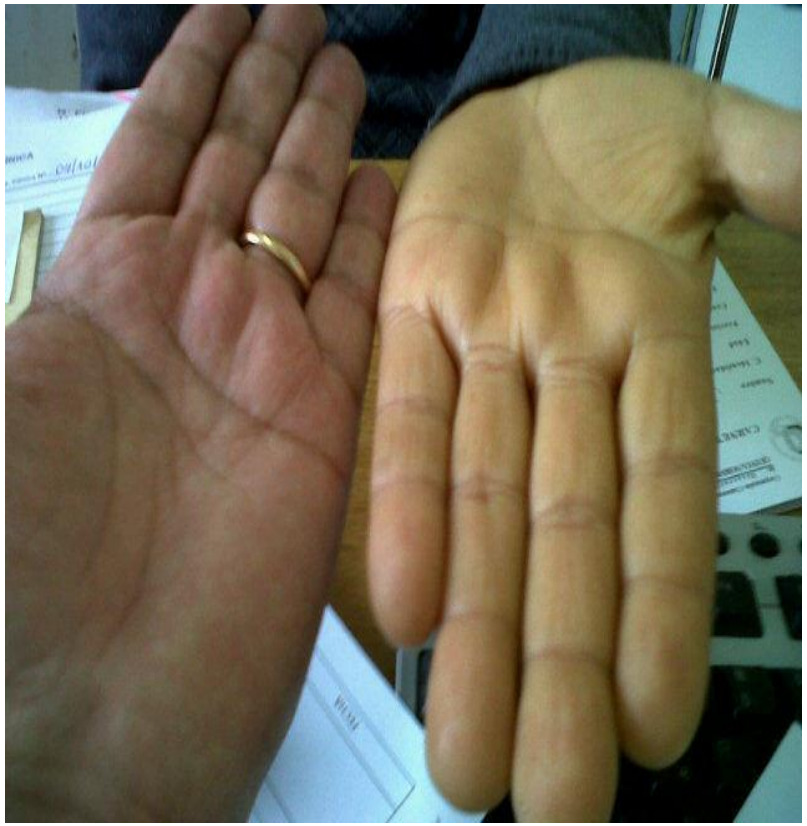
شرح

Skin manifestations of hypothyroidism

- **Cutaneous changes**: Dry rough, coarse skin, cold and pale boggy and edematous skin (Myxedema), yellow discoloration (carotinemia), Easy bruising (capillary fragility)
- **Cutaneous disease**: Ichthyosis, palmoplantar keratoderma, eruptive and tuberous xanthoma
- **Hair changes**: Dull, coarse brittle hair, slowly growing, alopecia of lateral eyebrows
- **Nail changes**: Thin brittle, striated nails, slow growth, Onycholysis
- **Investigations**: TSH, T3, T4, Anti-Thyroperoxidase, anti-Thyroglobulin antibodies

Skin manifestations of hypothyroidism

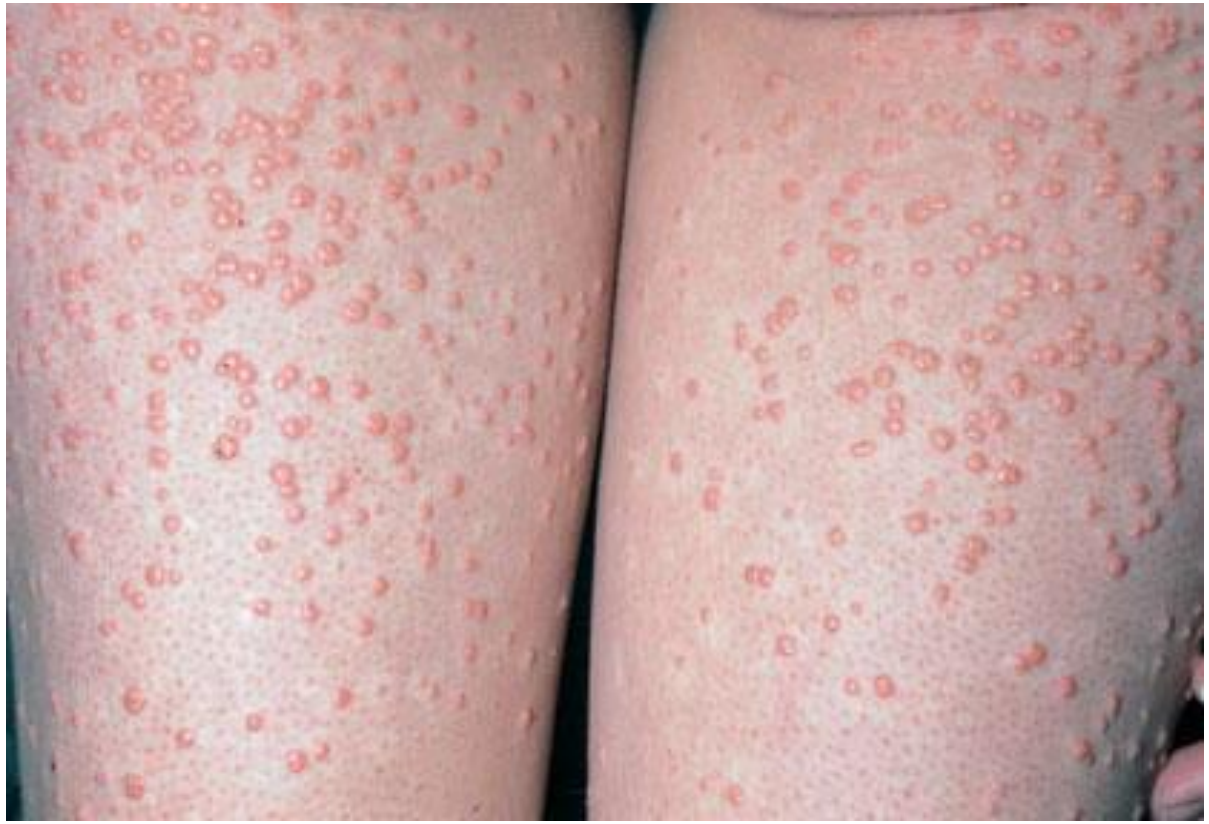
Facial features of hypothyroidism



Carotinemia

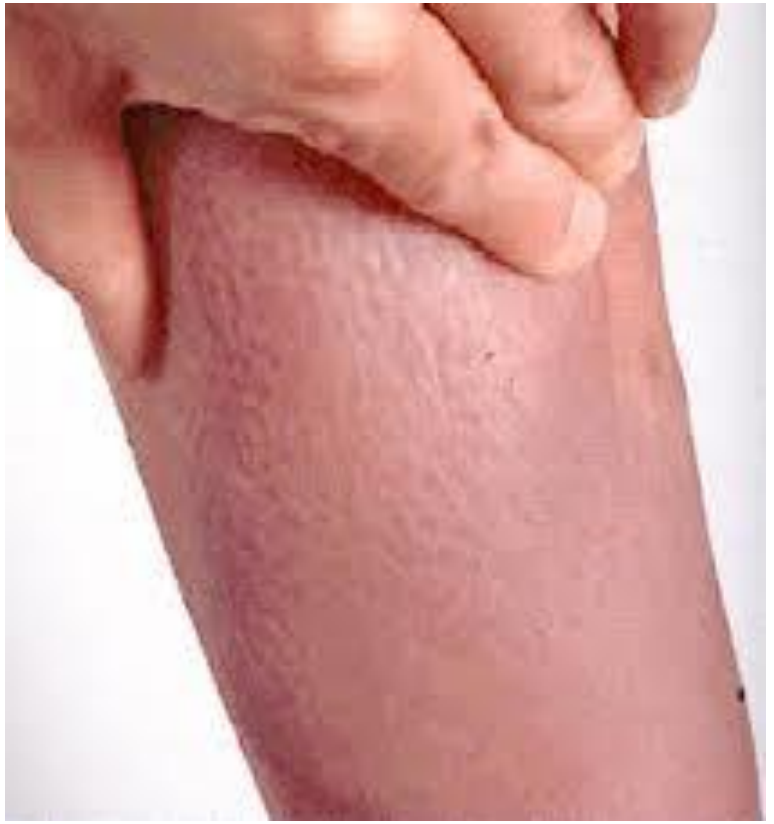
Yellow discoloration in the thick skin (Soles and palms)

Skin manifestations of hypothyroidism



Eruptive xanthoma

Yellowish papules



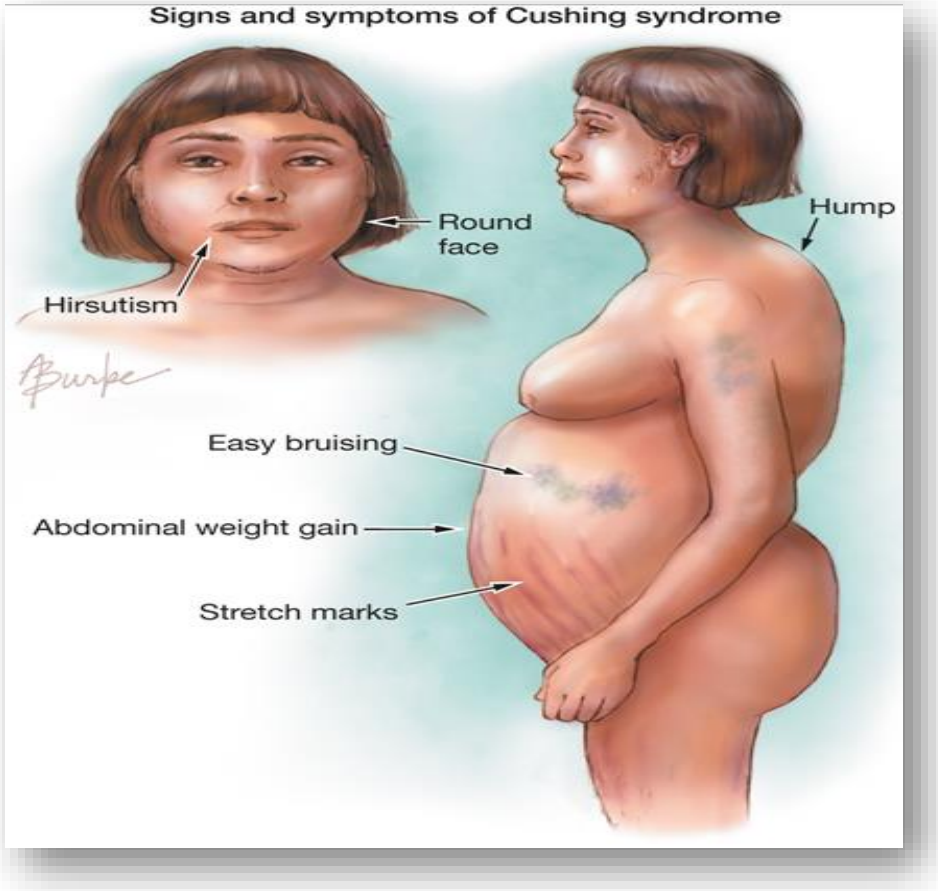
Myxedema

Thick skin due to Mucin deposition

Skin manifestations of Cushing's syndrome

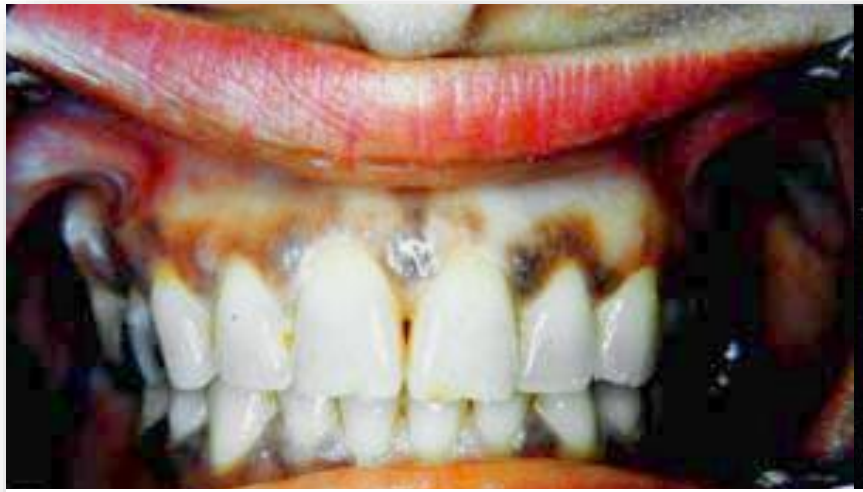
1. Moon face.
2. Buffalo hump.
3. Pelvic girdle fat deposition, reduced fat on arms and legs.
4. Striae, purpura after minor trauma.
5. **Skin infections**: TV (Tinea versicolor), dermatophytosis, candidiasis.
6. Acne, Hirsutism.

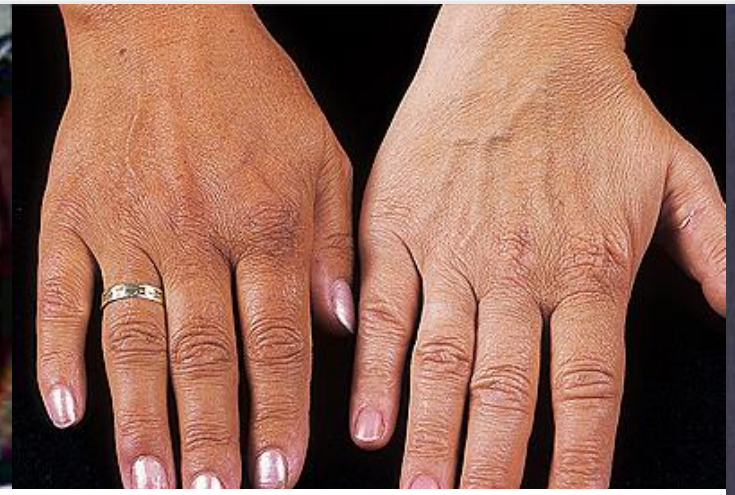
❖**Investigations**: ACTH, Dexamethasone suppression test.



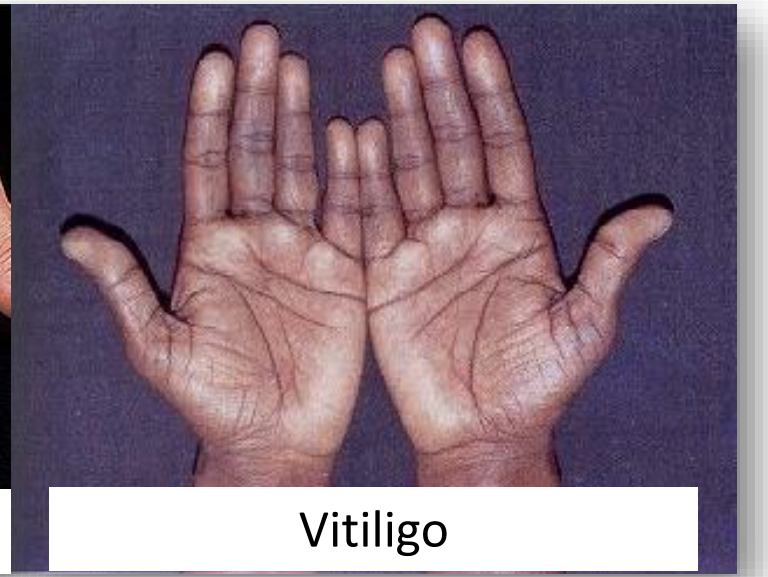
Skin manifestations of Addison's disease

- **Hyperpigmentation**: Diffuse, more on sun exposed areas, palmar creases, mucous membranes, nails , axillae, nipples and perineum.
- Vitiligo.
- **Investigations**: ACTH.



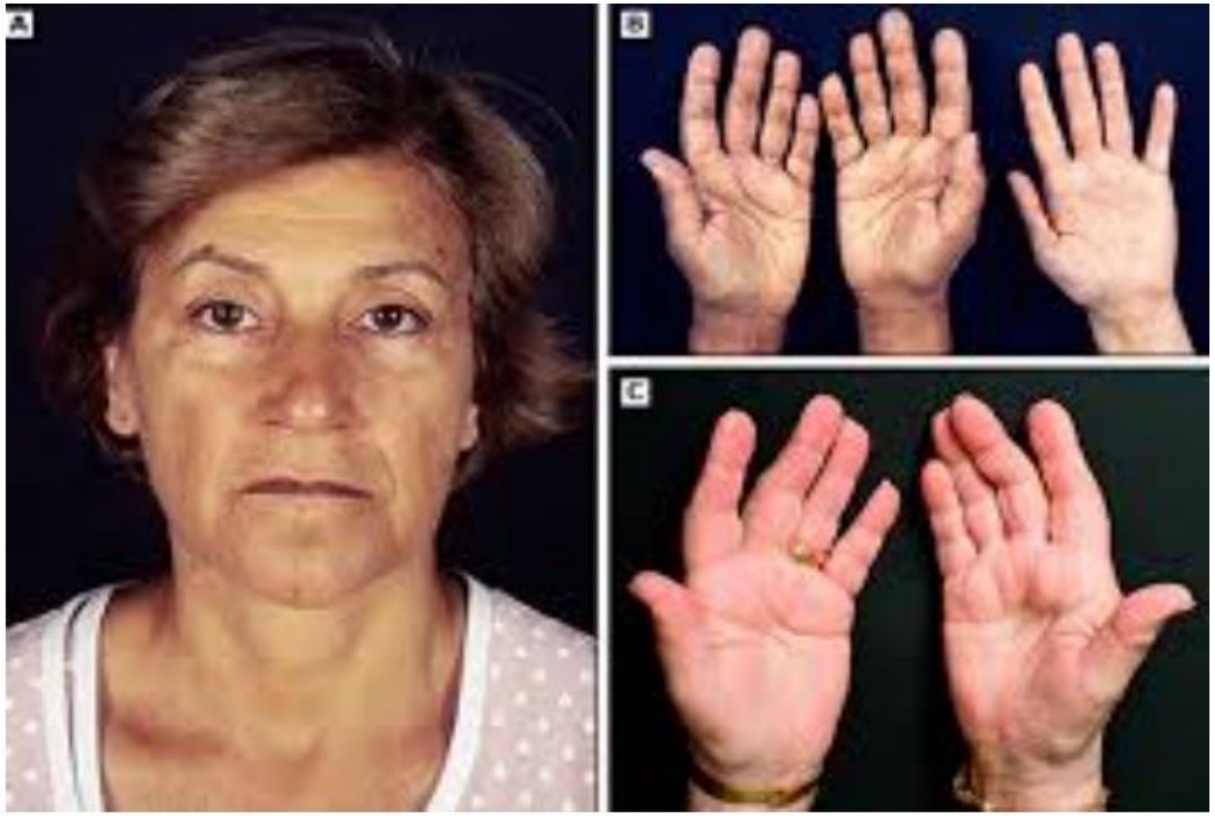


Hyperpigmentation



Vitiligo

Skin manifestations of Addison’s disease



Addison’s diseases

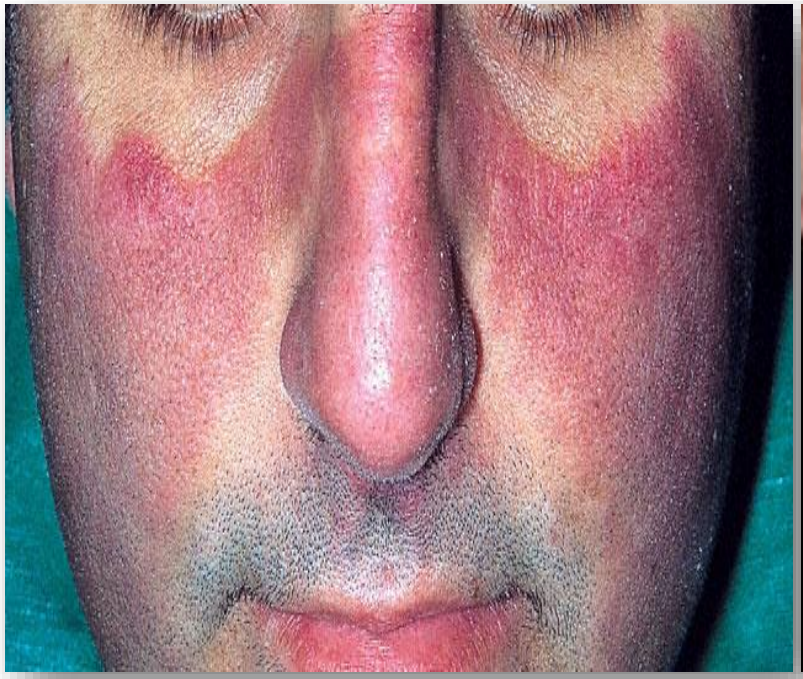
Skin manifestations of connective tissue diseases

SLE, Dermatomyositis, RA, Scleroderma

Skin manifestations of SLE

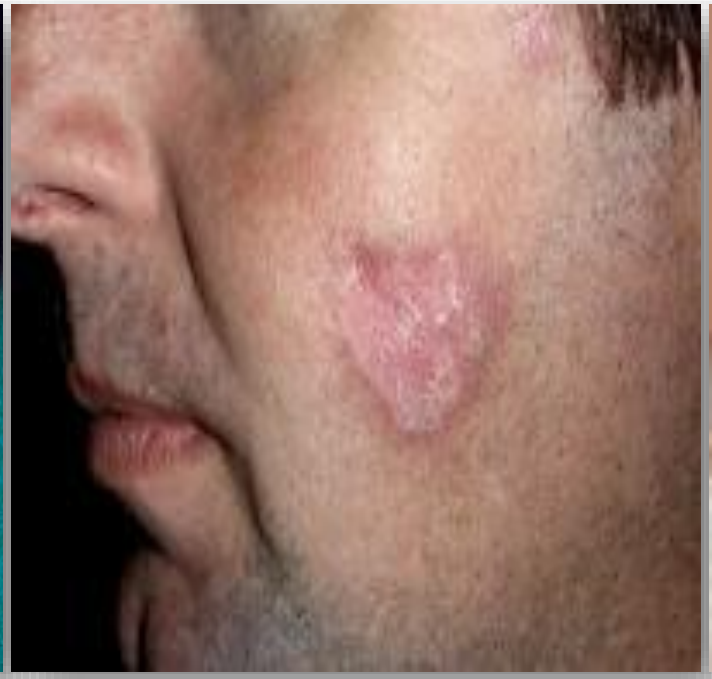
1. Malar Erythema.
2. Photosensitivity.
3. Discoid skin lesions.
4. Livedo reticularis, ulcers.
5. Urticaria, urticarial vasculitis
6. Periungual telangiectasia and erythema.

Skin manifestations of SLE



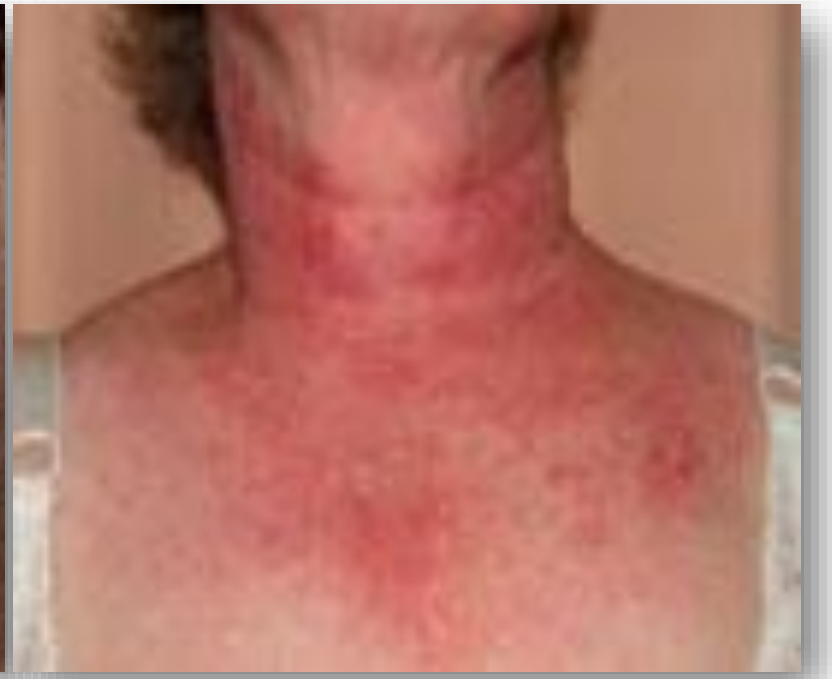
Malar rash

Erythematous rash affecting the cheeks and the bridge of the nose



Discoid lupus

Discoid scarring lesion



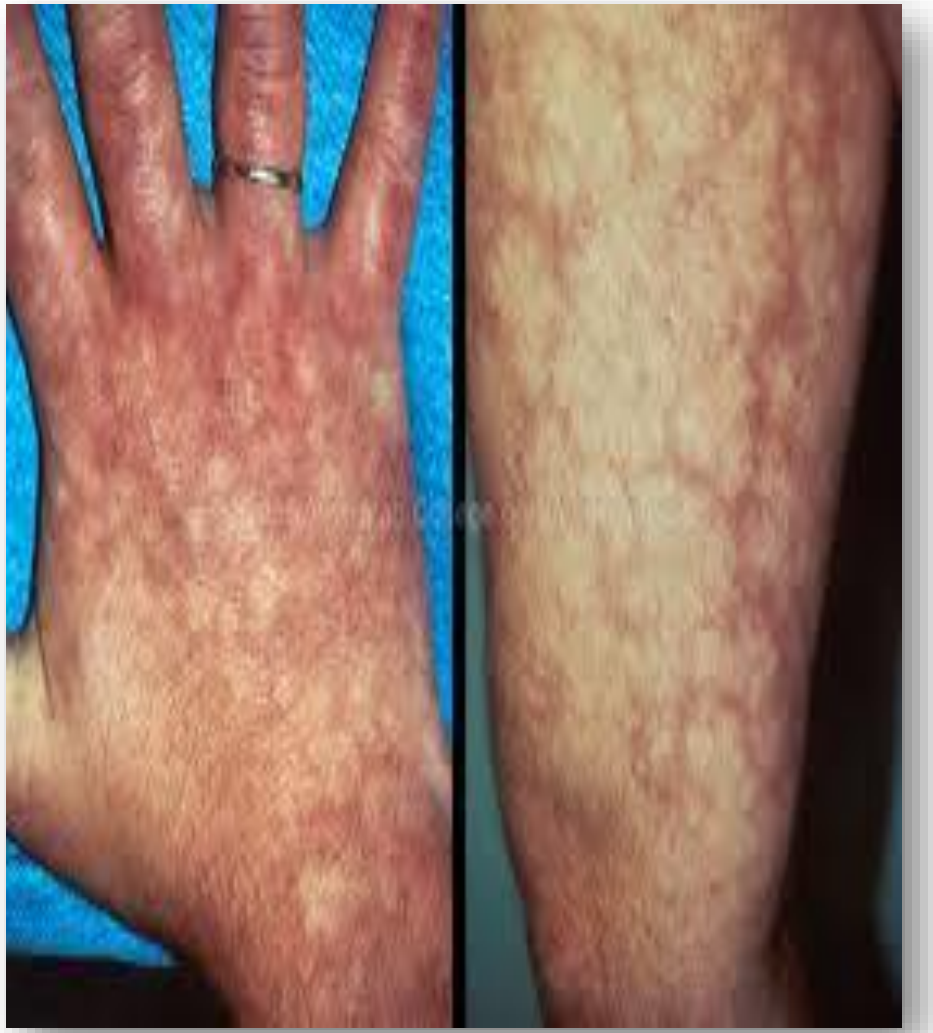
Photosensitive rash

Eczematous lesions with erythema and itching on the sun exposed areas

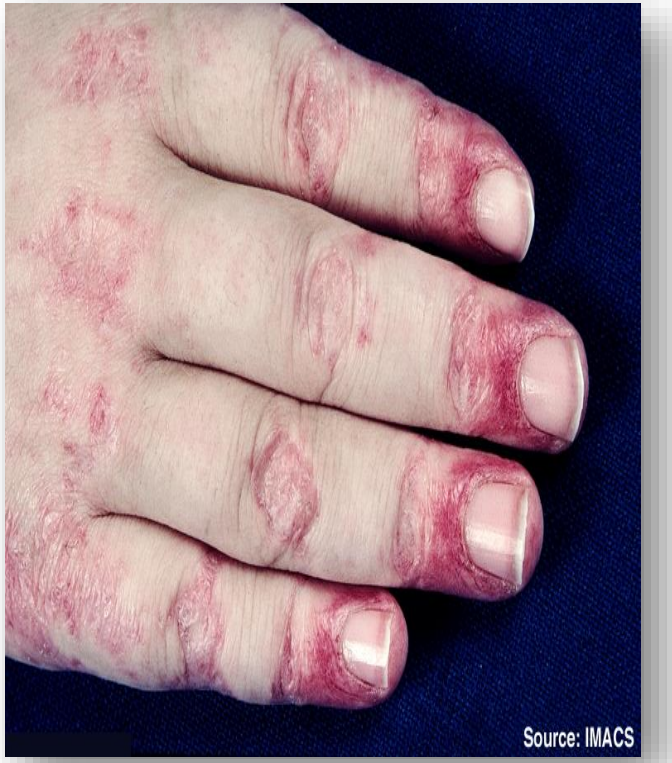
Skin manifestations of SLE

Livedo reticularis

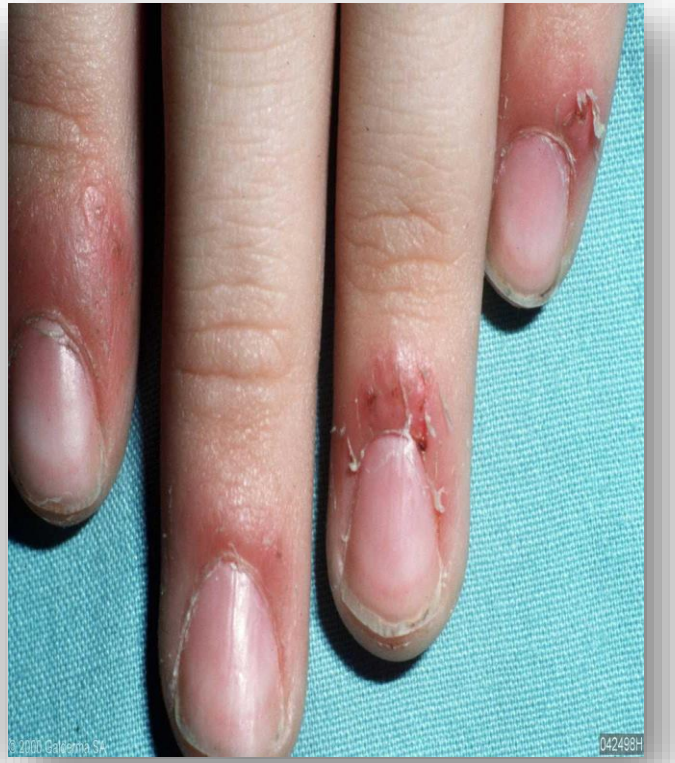
- Lace-like pattern of erythema due to thrombo-embolic event leading to vascular compromise , occurs on CT diseases and hypercoagulative states.



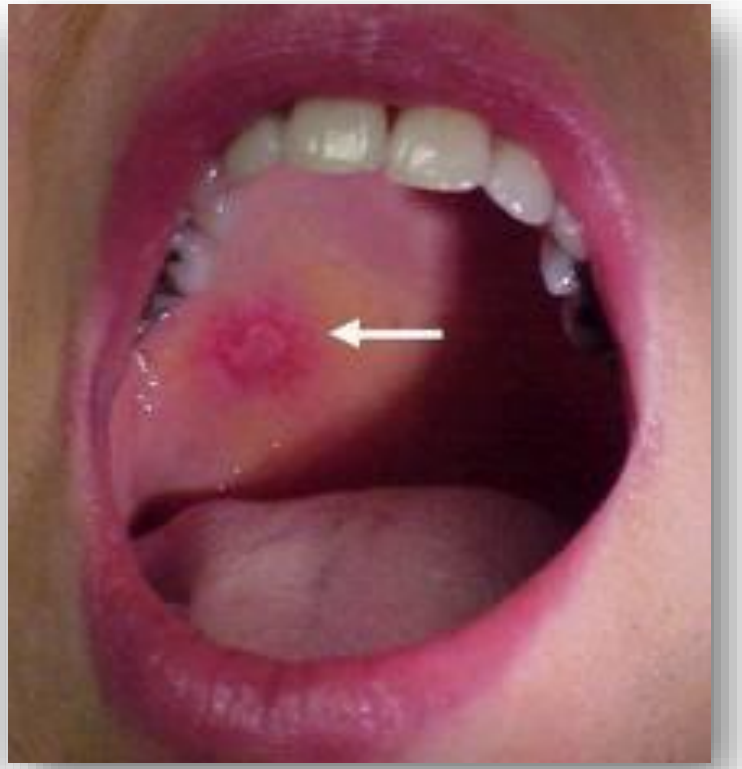
Skin manifestations of SLE



Periungual erythema and Telengectasia

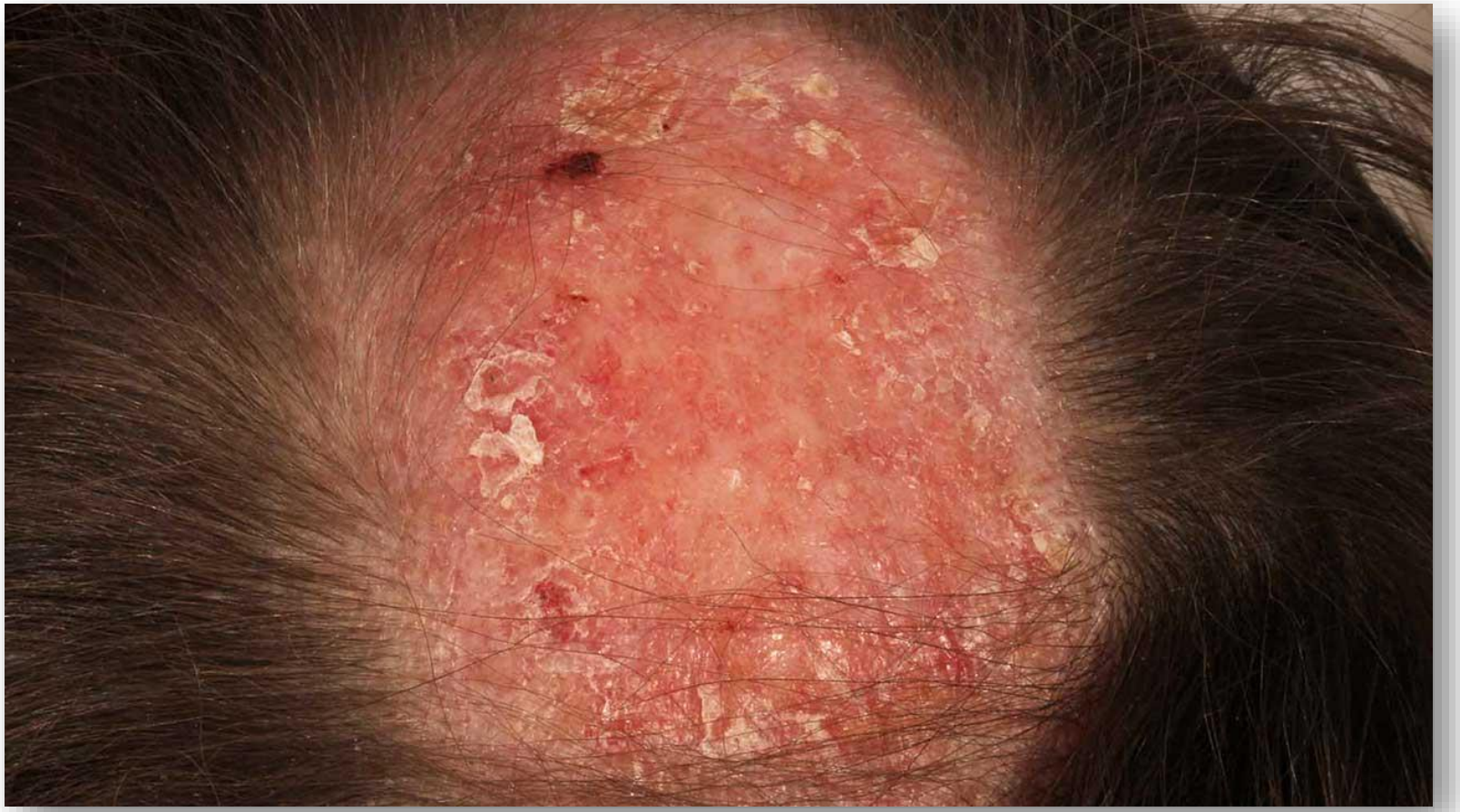


Periungual erythema and necrotic areas



Oral ulcer

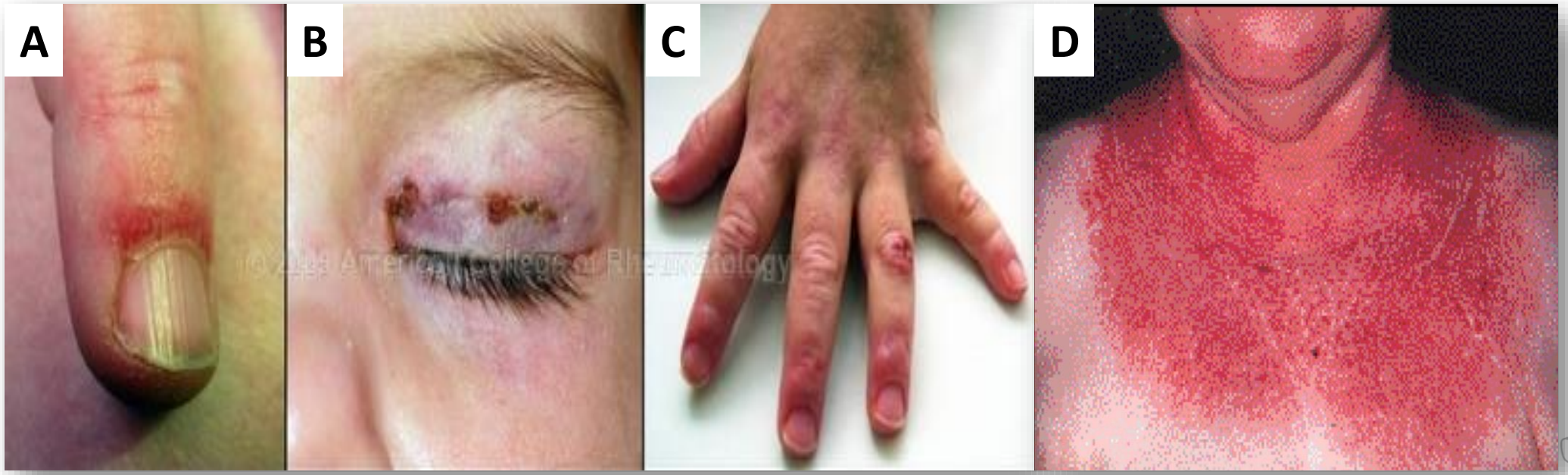
What is the diagnosis



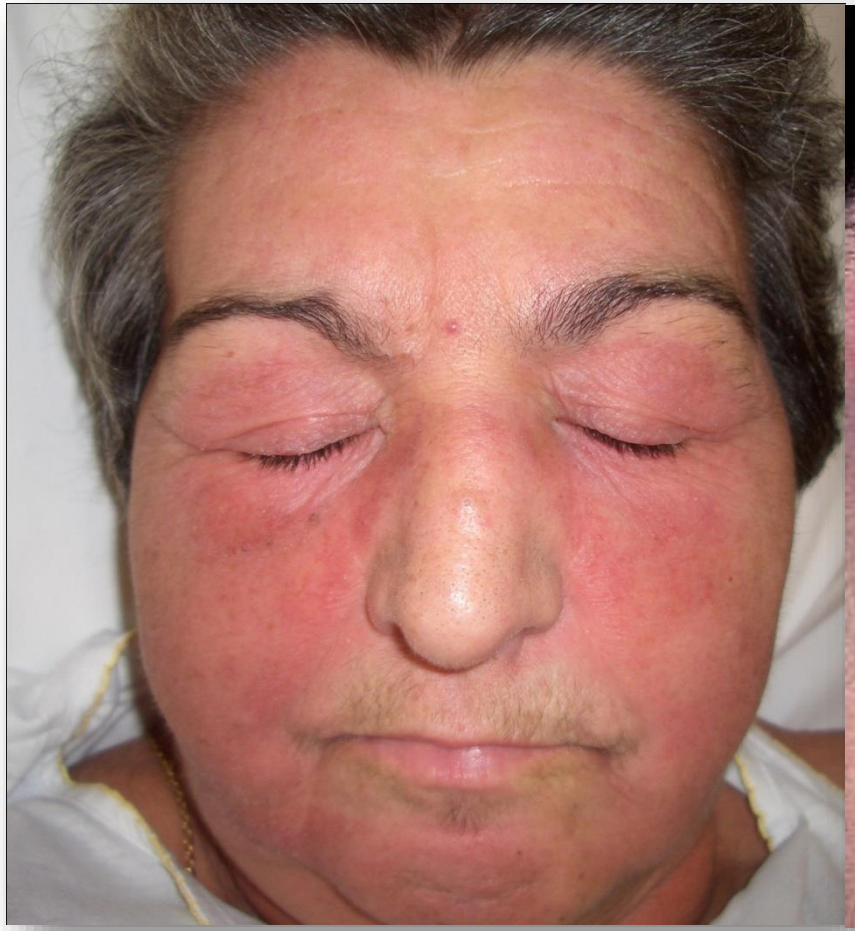
Lupus alopecia

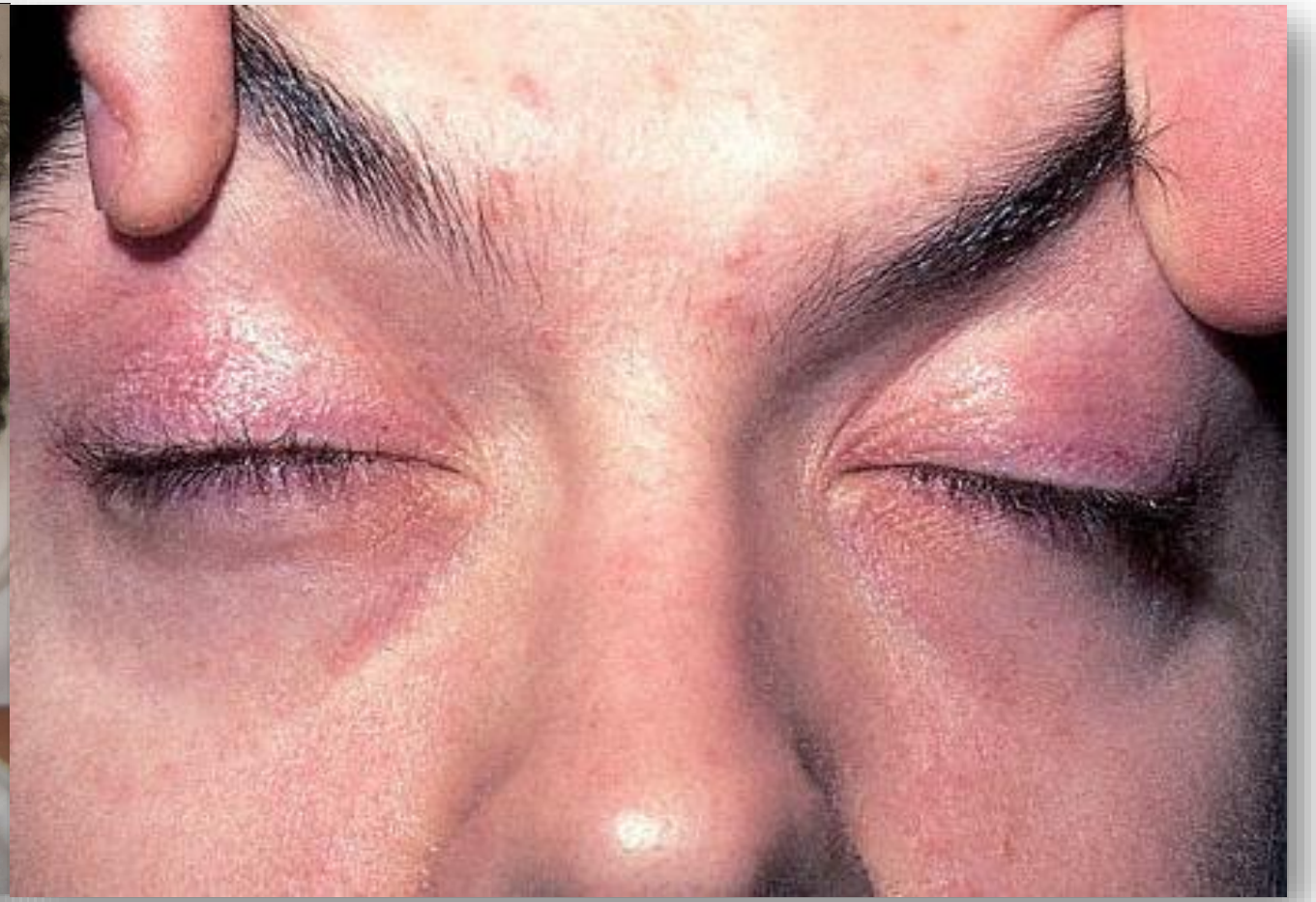
Skin manifestations of Dermatomyositis

A. **Periungual telangiectasia and erythema**
B. **Heliotrope erythema**
C. **Gottron's papules**
D. **Photo distributed Poikiloderma** (Triad of: Atrophy + Hyperpigmentation + Telangiectasia +/- Hypopigmentation)



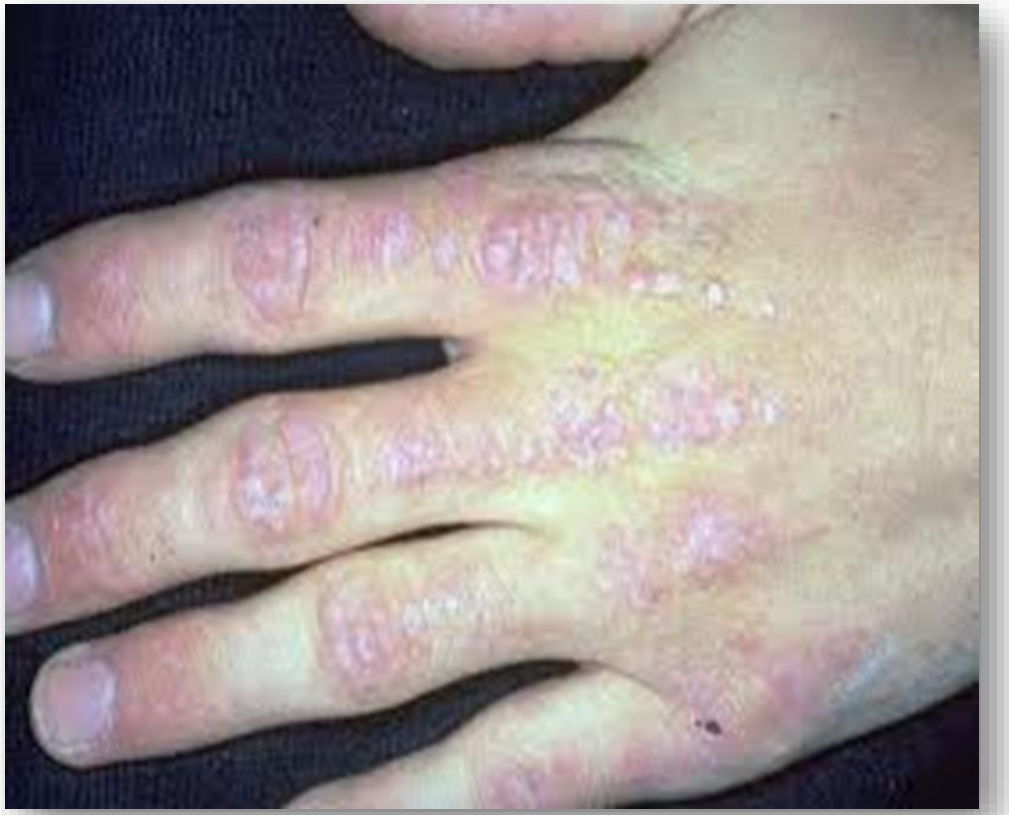
Heliotrope erythema





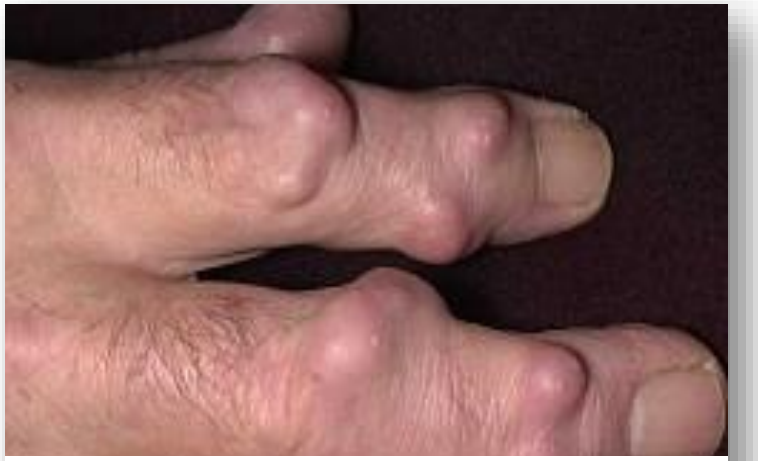
This sign is seen in which disease

- **What is the name of this ?**
  - Gottron's papules
- **This sign is seen in which disease ?**
  - Dermatomyositis

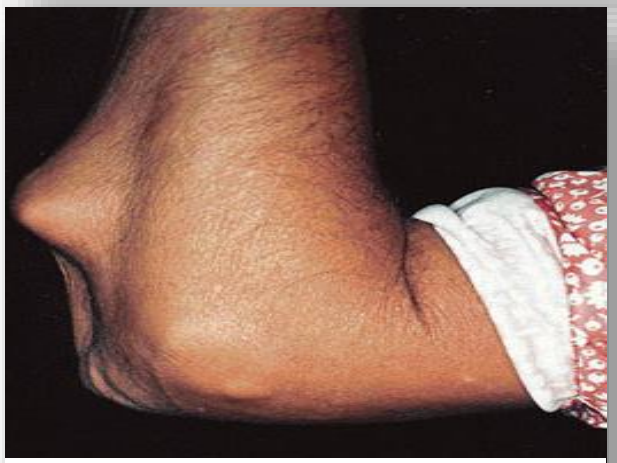


Skin manifestations of Rheumatoid arthritis

1. Rheumatoid nodules
2. Vasculitis
3. Erythema elevatum diutinum
4. Sweet`s Syndrome
5. Purpuric papules on distal digits
6. Periungual telangiectasia and erythema
7. Pyoderma gangrenosum



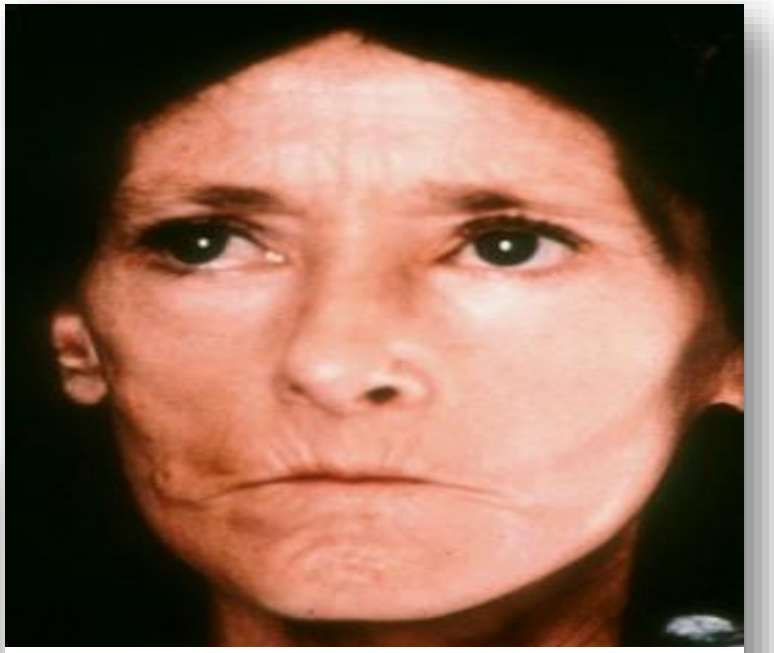
Rheumatoid nodules



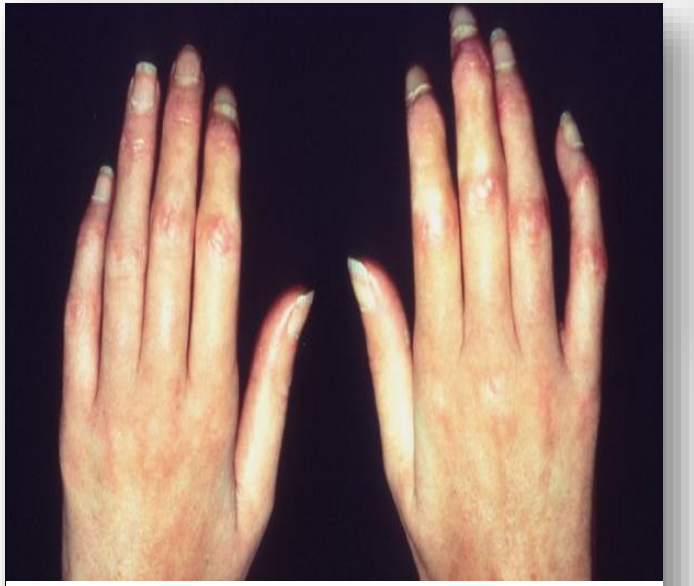
Rheumatoid nodules

Skin manifestations of Scleroderma

1. Skin sclerosis, Tight bound skin.
2. Peaked nose.
3. Perioral furrows.
4. Periungual telangiectasia and erythema.
5. Sclerodactyly.



Peaked nose and perioral furrows due to skin sclerosis

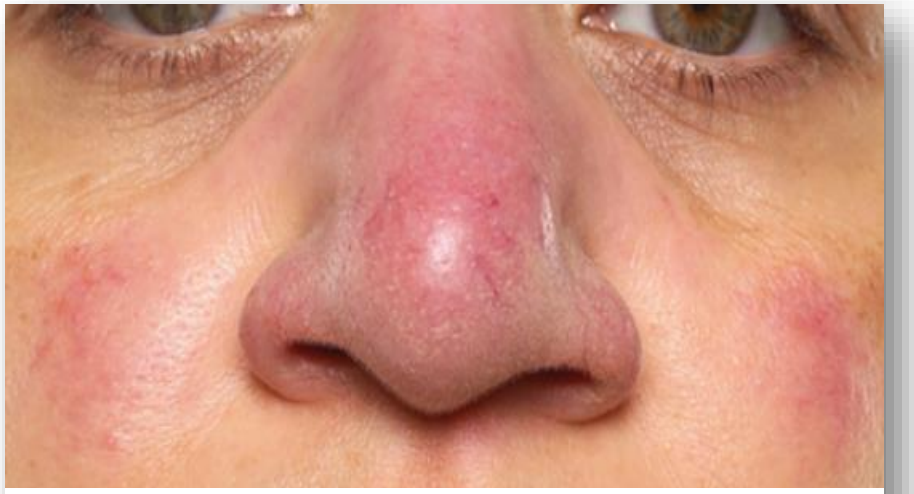


Sclerodactyly

Miscellaneous

Skin and Sarcoidosis

1. **Lupus pernio**: the skin of the nose and ears is involved in the granulomatous process, and becomes swollen and purplish in color
2. **Erythema nodosum**: this takes the form of tender, erythematous nodules on the legs
3. **Scar sarcoid**: sarcoid granulomas localize in old scar tissue, making the scars prominent
4. **Papules, nodules and plaques**: these often have a purplish/brown color



Lupus pernio

سنوات (1)

In what disease is Lupus Perino seen in ?

sarcoidosis

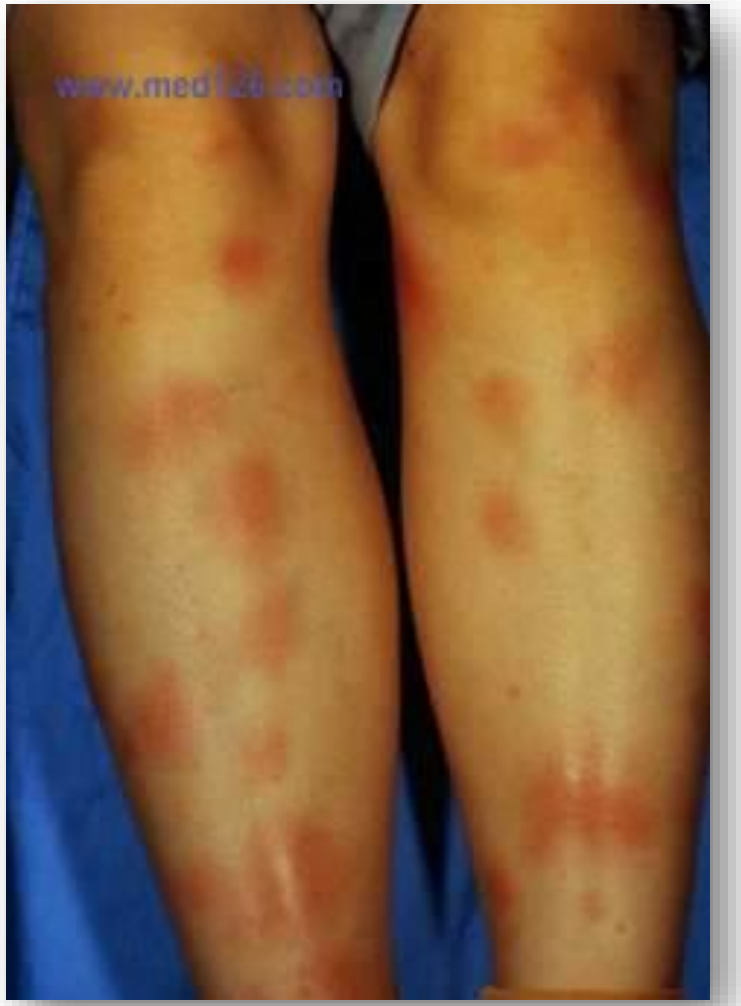
Erythema nodosum

❖**Characterized by**:

- Development of multiple, tender, erythematous nodules, usually on the shins but occasionally also on the forearms

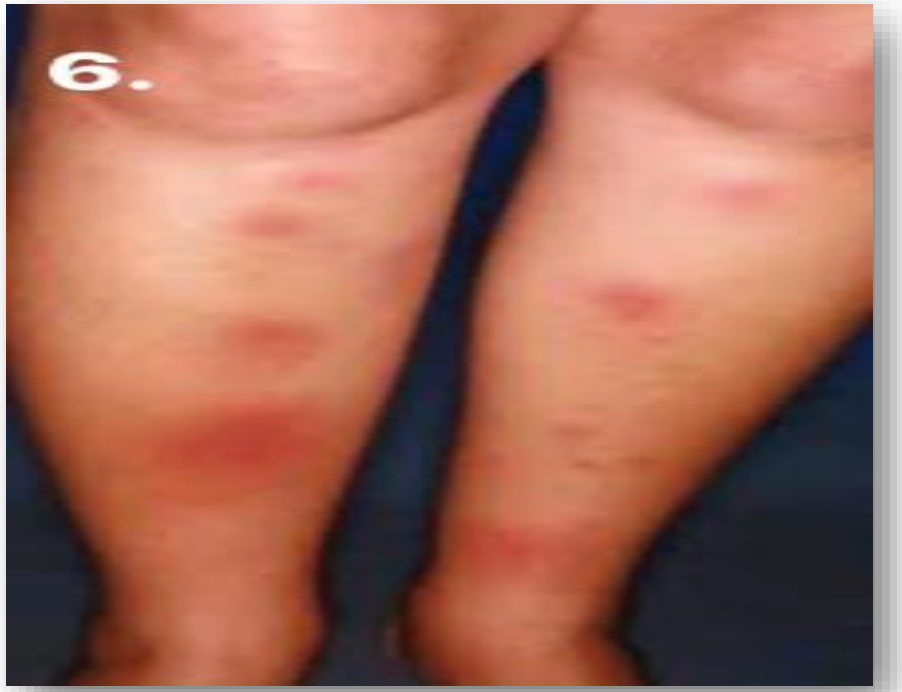
❖**Causes**:

1. Streptococcal infection
2. Primary tuberculosis (TB)
3. Drugs
4. Sarcoidosis
5. Inflammatory bowel disease
6. Connective tissue diseases
7. Malignancy (Lymphoma)



Tender lesion with sudden onset, the diagnosis is

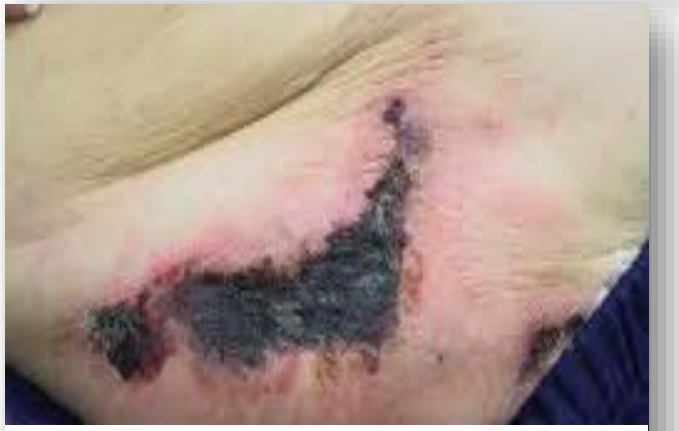
A. Cellulitis
B. Deep vein thrombosis (DVT)
C. Erythema multiforme
D. Erythema nodosum
E. Granuloma annulare



Skin and Kidney disease

1. Pruritus and dry skin
2. Pigmentations, yellowish shallow, pale skin
3. Half and half nails (White/red, Lindsay's nails)
4. perforating disorder, folliculitis.
5. Pseudoporphyria
6. Calciphylaxis

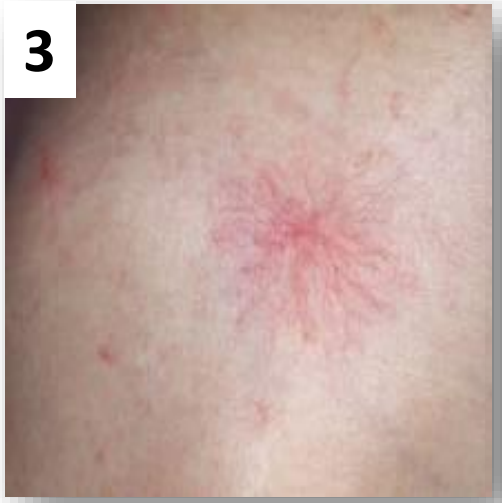
Lindsay's nails
Half and half nails

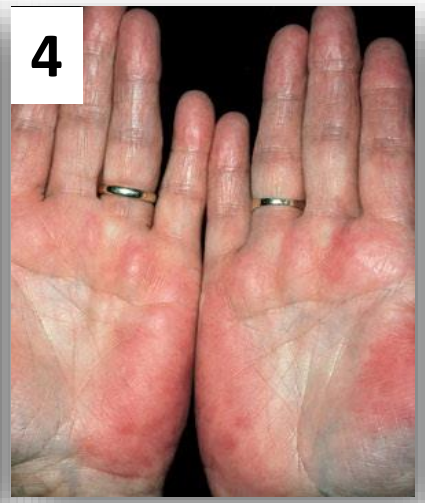


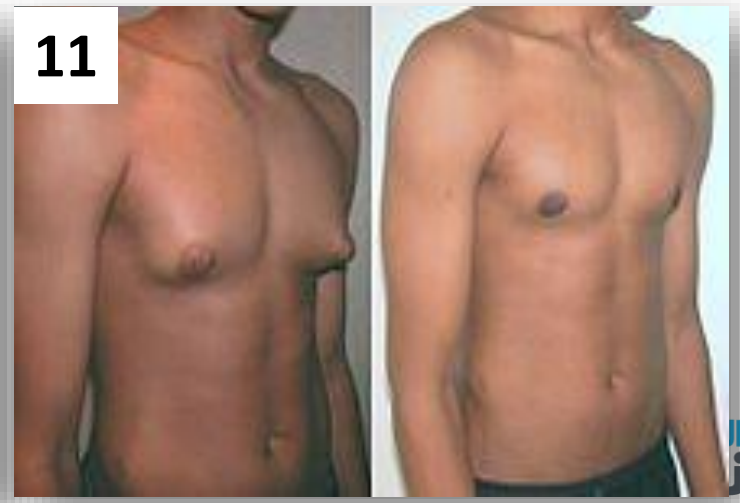
Calciphylaxis

Skin and Liver disease

1. Pruritus due to obstructive jaundice.
2. Hyperpigmentation (due to bile and melanin).
3. Multiple spider nevi
4. Palmar erythema
5. White nails (terry`s nails): due to Hypoalbuminemia
6. Lichen planus
7. Polyarteritis nodosa
8. Porphyria cutanea tarda
9. Xanthoma, primary biliary cirrhosis
10. Hair loss and generalized aesteatotic eczema
11. Gynecomastia (in cirrhosis)

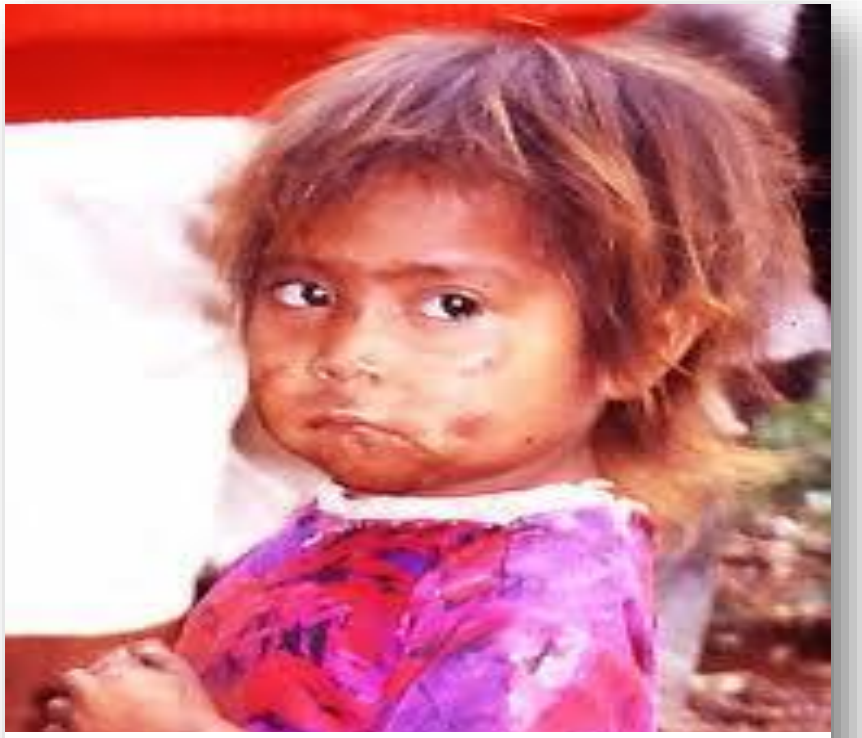






Skin changes in malabsorption and malnutrition

- Itching, dryness, pigmentations.
- Brittle nails and hair.
- **Kwashiorkor**: dry red brown hair.
- **Iron deficiency**: pallor, itching, diffuse hair loss, koilonychia.
- **Vit. A deficiency**: dry skin, follicular hyperkeratosis, xerophthalmia.
- **Vit. C deficiency**: Scurvy.



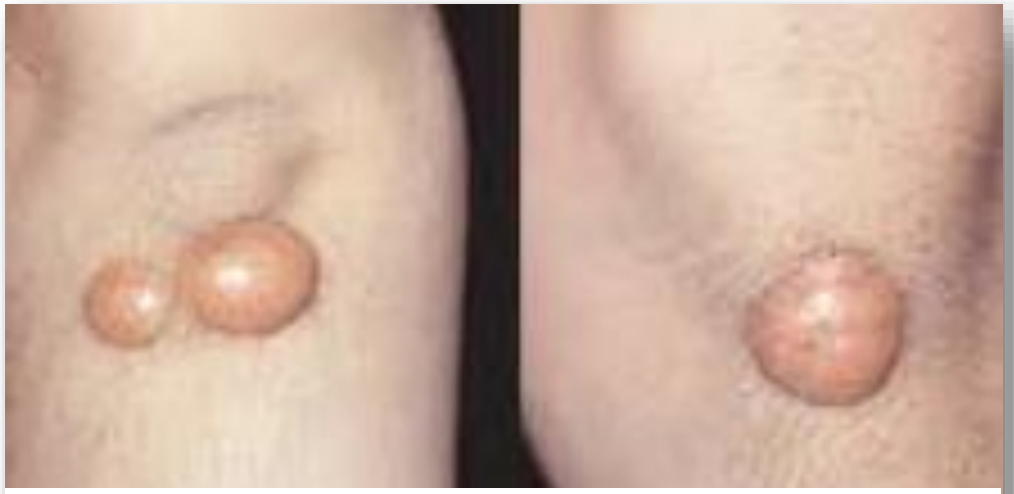
Kwashiorkor disease

Brittle dry and brawny pigmented hair due to protein deficiency

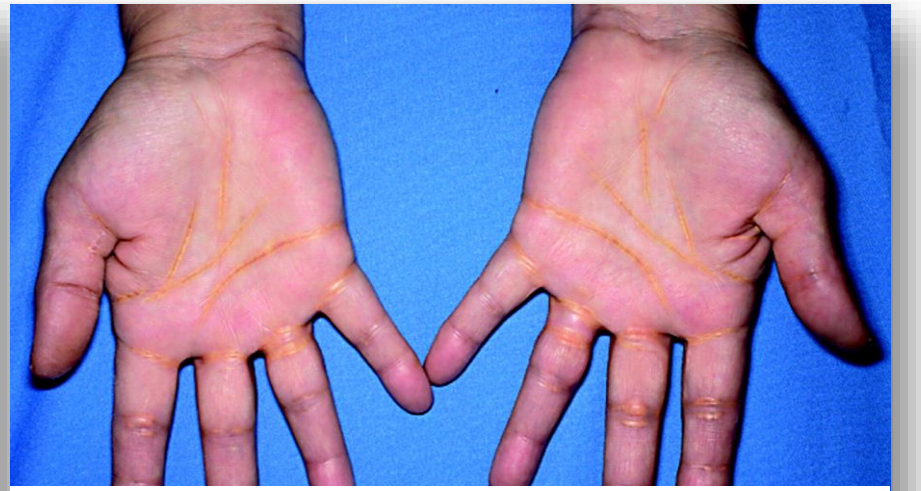
Xanthomas

- Yellowish lesions diagnosed clinically or by biopsy
- **Types**:
  - Nodular (Tuberous).
  - Linear (Usually on the palm creases).
  - Eruptive papules.
- **Primary hyperlipidemia**:
  - Genetic, six groups.
- **Secondary hyperlipidemia**:
  - Diabetes mellitus.
  - Cirrhosis.
  - Nephrotic syndrome.
  - Hypothyroidism.

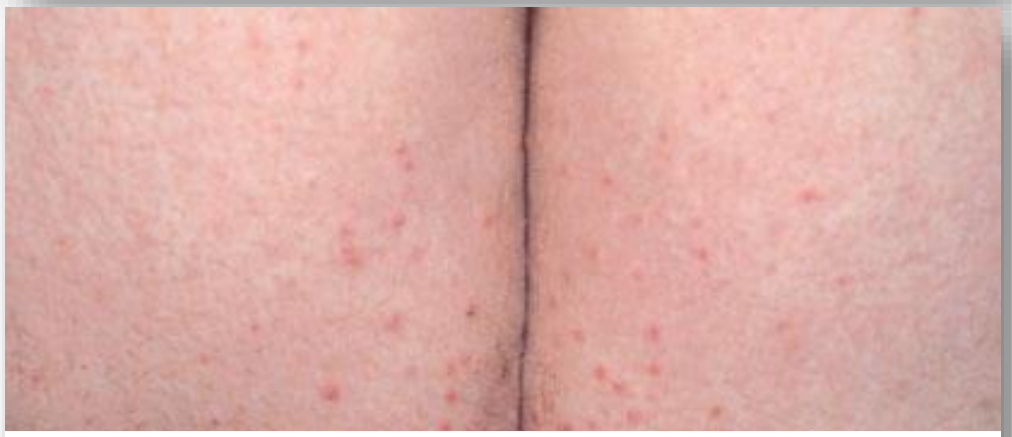
Xanthomas



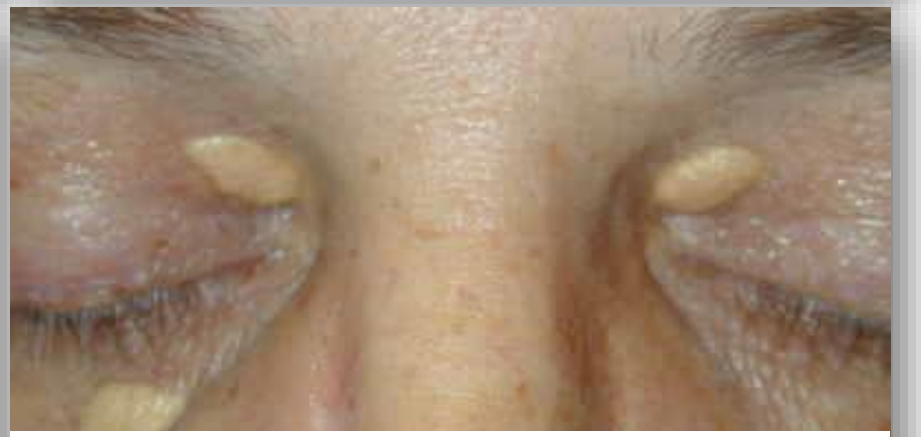
Nodular (Tuberous)



Linear (Palm creases)



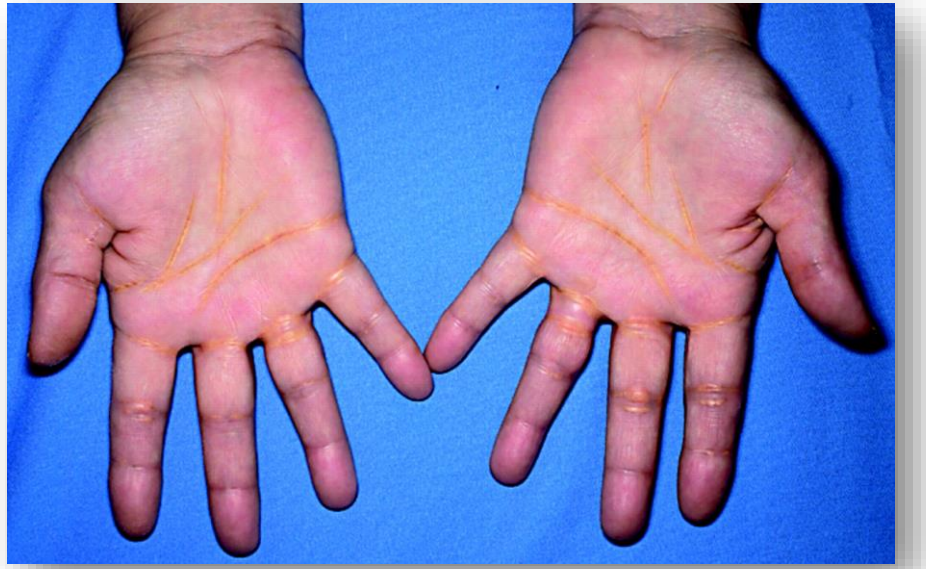
Eruptive papules



Xanthelasma

Xanthomas

- **What is the type of this xanthoma ?**
  - Linear
- **What is the best test to do for this patient ?**
  - Lipid profile
- **Mention other types of xanthomas**
  - Nodular (Tuberous)
  - Eruptive papules
  - Xanthelasma



Skin and pregnancy

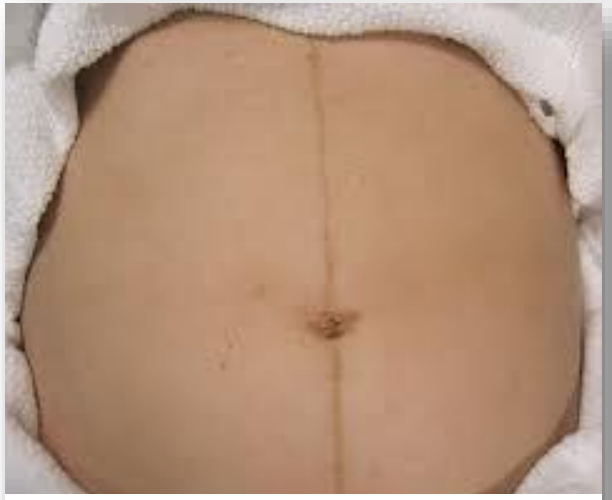
Due to hormonal changes: (Physiological)

1. Linea nigra.
2. Melasma.
3. Darkening of areola and nipples.
4. Palmar erythema.
5. Telangiectasia.
6. Striae.
7. Hair loss, especially after delivery (Telogen effluvium).
8. Skin tags
9. Pyogenic granuloma, mouth.

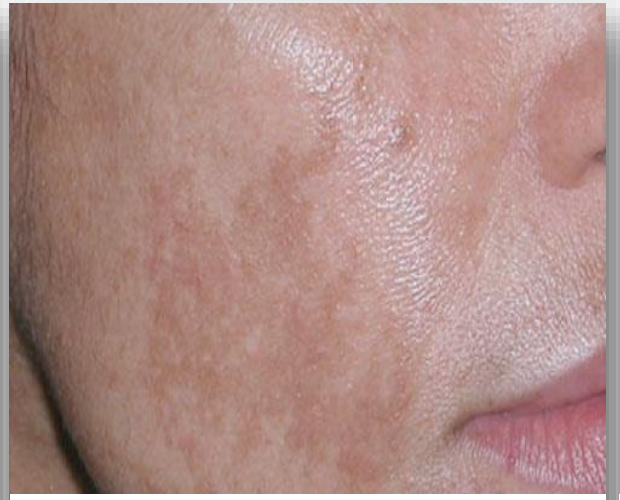
Dermatosis of pregnancy: (Pathological)

1. Pruritus of pregnancy.
2. Urticarial plaques and papules of pregnancy.
3. Impetigo herpetiformis (Generalized pustular psoriasis of pregnancy).
4. Pemphigoid gestations.
5. Prurigo of pregnancy.
6. Cholestasis of pregnancy.

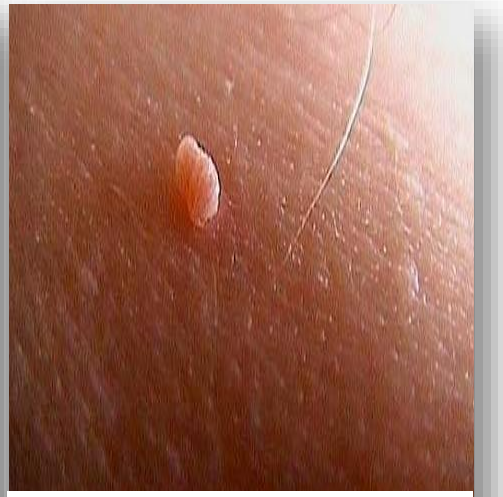
Skin and pregnancy



Linea nigra

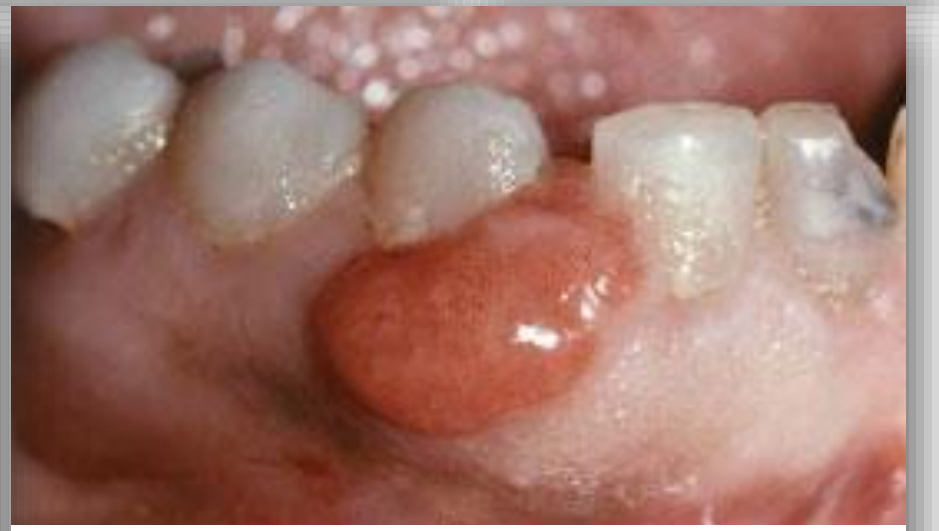


Melasma



Skin tags

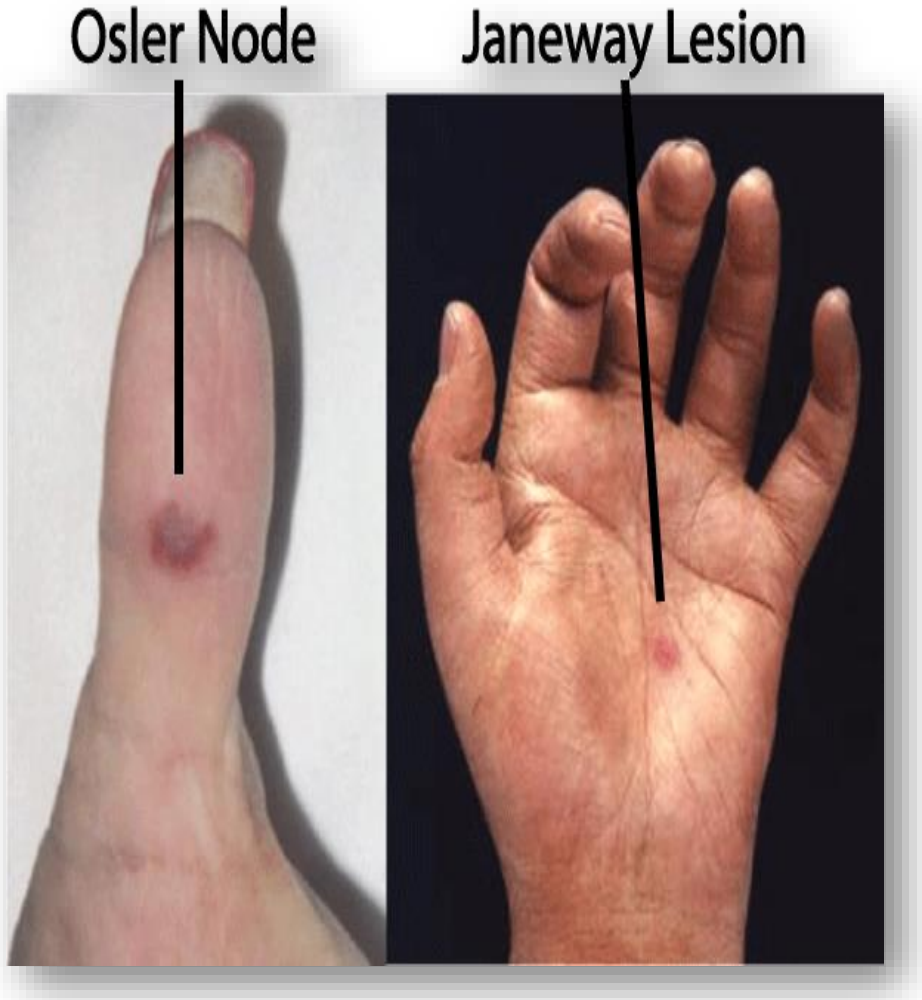
Striae



Pyogenic granuloma

Bacterial endocarditis

1. Purpura
2. Nail fold infarction
3. Splinter hemorrhage
4. Janeway lesions
5. Osler`s nodules



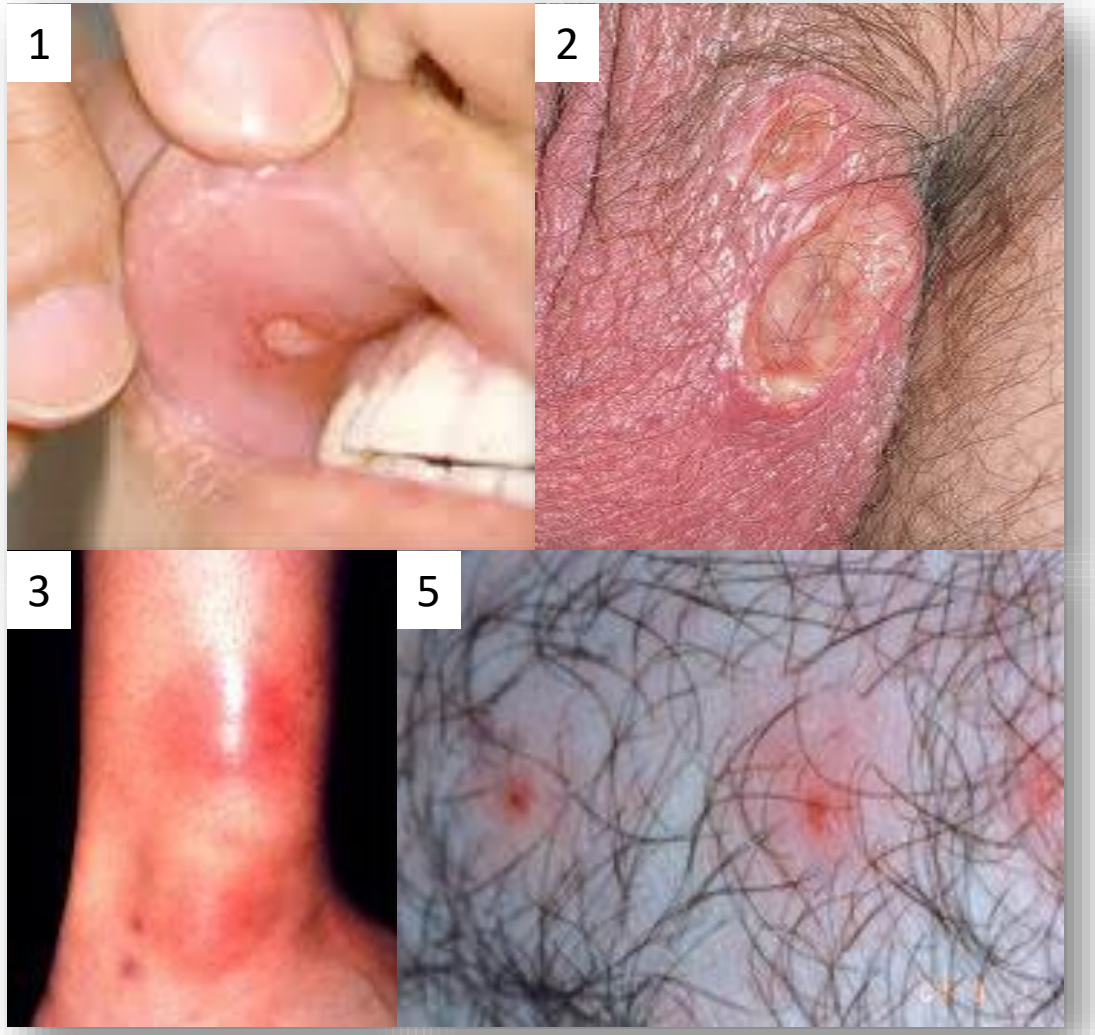
Mention 2 manifestations

سنوات (2)

شرح

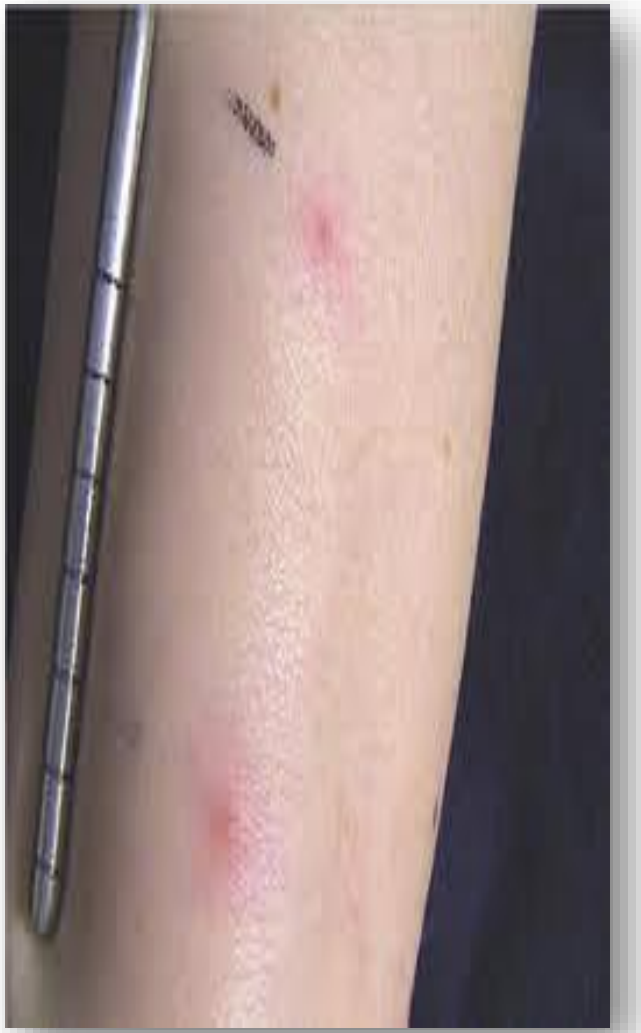
Skin manifestations of Bechet's disease

1. Painful Oral ulcers with yellowish base
2. Painful Genital ulcers with yellowish base
3. Erythema nodosum-like lesion
4. Erythema multiforme-like lesions
5. Acne form skin rash
6. Pathergy reaction



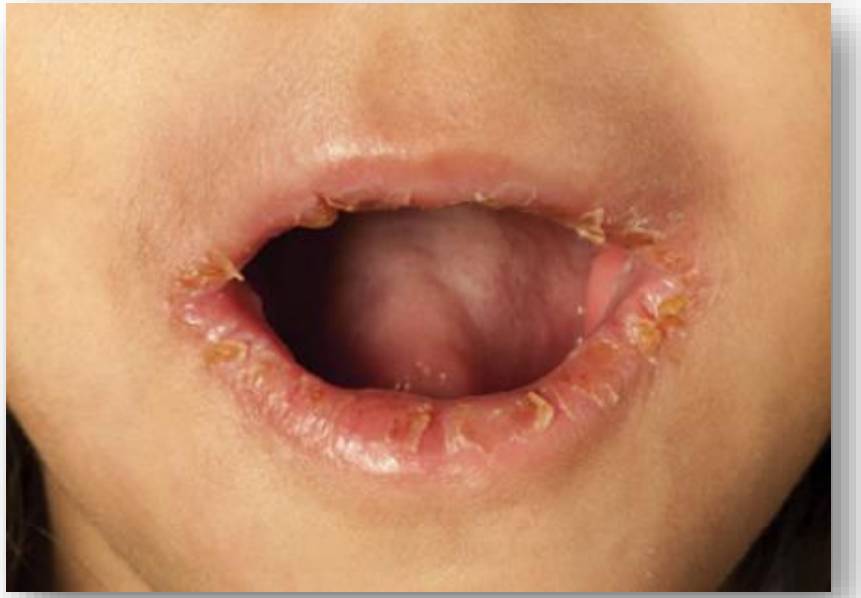
Pathergy reaction

- **Pathergy** is a skin condition in which a minor trauma such as a bump or bruise leads to the development of skin lesions or ulcers that may be resistant to healing
- Pathergy is seen with both Bechet's disease and pyoderma gangrenosum
- Doctors looking toward a diagnosis of Behçet's disease may attempt to induce a pathergy reaction with skin prick test (Development of a pustule at the site of a needle prick)



Skin and inflammatory bowel disease

1. Erythema nodosum (mentioned earlier)
2. Pyoderma gangrenosum (next slide)
3. Perianal and buccal mucosal lesions



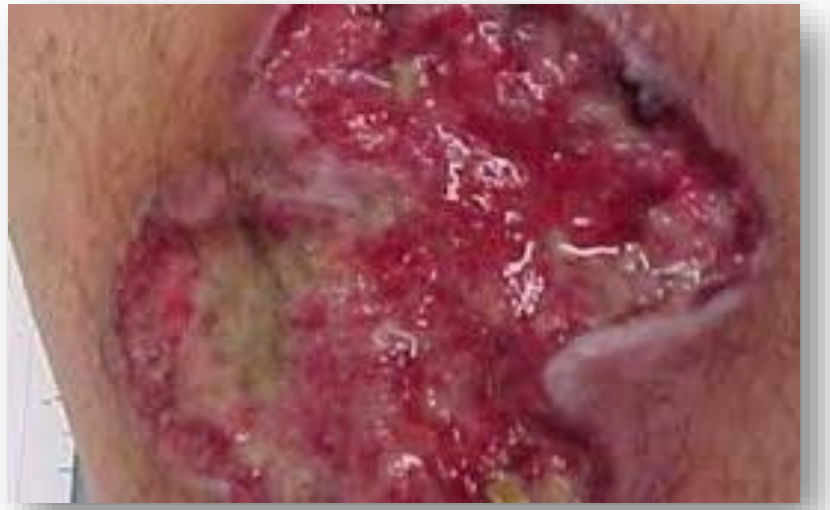
Swollen, fissured lips in a young girl. Inside the mouth there were mucosal tags. She was anemic with a history of diarrhea and weight loss. Investigations confirmed Crohn's disease.

شرح

Pyoderma gangrenosum

❖**Characterized by**:

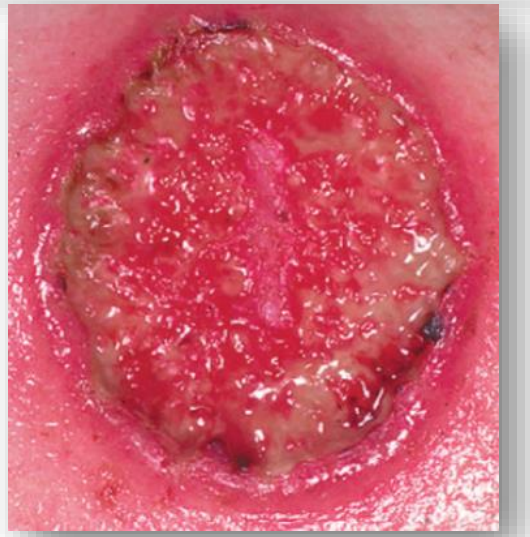
- The lesions may be single or multiple. They initially resemble boils, which subsequently break down to form necrotic ulcers with undermined purple edges



سنوات (3)

❖**Causes**:

1. Inflammatory bowel disease
2. Malignancy (myeloma & myeloid malignancies)
3. Connective tissue disorders (RA)



❖**Treatment of choice**: Systemic steroids


"That's all Folks!"