## PATHOLOGY LAB



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## A-pituitary gland

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## NORMAL ANTERIOR PITUITARY GLAND

## Normal anterior pituitary gland

Normal Ant.pituitary composed of acidoophilic and pasophilic cells according to color and pale chromophobescells

Normal pos.pituitary<br>Composed of glial cells and nerve axons




The pink acidophils secrete growth hormone (GH) and prolactin (PRL)
The dark purple basophils secrete corticotrophin (ACTH), thyroid stimulating hormone (TSH), and gonadotrophins follicle stimulating hormone-luteinizing hormone (FSH and LH) .
The pale staining chromophobes have few cytoplasmic granules, but may have secretory activity.


The neurohypophysis shown here resembles neural tissue, with glial cells, nerve fibers, nerve endings, and intra-axonal neurosecretory granules.
The hormones vasopressin (antidiuretic hormone, or ADH) and oxytocin made in the hypothalamus (supraoptic and paraventricular nuclei) are transported into the intraaxonal neurosecretory granules where they are released.

## BEHAVIOUR OF PITUITARY ADENOMAS :

- Primary pituitary adenomas usually benign.
- Radiological changes in sella turcica .
- May or may not be functional(20\%). If functional (80\%), the clinical effects are secondary to the hormone produced.
- More than one hormone can be produced from the same cell ( monoclonal ).
- Local effects are due to pressure on optic chiasma (visual disturbance), or pressure on adjacent normal pituitary cells (reduce hormone production).



## Mass effect of pituitary adenoma

## CLINICAL FEATURES OF PITUITARY ADENOMA:

1- Symptoms of hormone production.
2- Visual field abnormalities (pressure on optic chiasma above sella tursica ).

3- Elevated intracranial pressure (blockage of CSF flow ): Headache, nausea, vomiting.

4- Hypopituitarism ( result from pressure on adjacent pituitary ): Diabetes insipidus .

5-Cranial nerve palsy ( invasion to brain ).

## MORPHOLOGY OF PITUITARY ADENOMAS :

- Well circumscribed, invasive in up to $30 \%$
- Size 1 cm . or more, specially in nonfunctioning tumor
- Hemorrhage \& necrosis seen in large tumors (pituitary apoplexy).
Microscopic picture:
- Uniform cells, one cell type (monomorphism)
- Absent reticulin network
- Rare or absent mitosis


Sella turcica with pituitary adenoma

## 1- PROLACTINOMA :

- 30\% of all adenomas, chromophobe or w. acidophilic
- Functional even if microadenoma, but amount of secretion is related to size
- Mild elevation of prolactin does NOT always indicate prolactin secreting adenoma!
- Other causes of $\uparrow$ prolactin include :
- estrogen therapy
- pregnancy
- certain drugs, e.g reserpine (dopamin inhibitor).
- hypothyroidism
- mass in suprasellar region ?


## 2- Growth hormone secreting adenoma :

## Structure :

Composed of granular ACIDOPHILIC cells and may be mixed with prolactin secretion.

Symptoms :
May be delayed so adenomas are usually large Produce GIGANTISM (children) or ACROMEGALLY (adults).
Diabetes, arthritis, large jaw \& hands, osteoporosis,个BP, HF.....etc

## NORMAL PITUITARY GLAND

Reticulin stain highlight the septa between cells


## Pituitary adenoma

Monotomas cells Absent reticulin stain

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## ACROMEGALY V.S DWARFISM



## GROSS SECTIONS OF PITUITARY ADNOMA


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## B-Thyroid gland



Follicles lined by flat
epithlium

Normal thyroid gland

## Hyperthyroidism symptoms



Hand tremors


Difficulty in sleeping


Hair loss


Elevated blood presure


Menstrual problems


Swollen eyes

Weight loss



Irregular heart rate


Nervousness


Feeling hungry

## HYPOTHYROIDISM IS COMMONER IN ENDEMIC AREAS OF IODINE DEFICIENCY

CRETINISM : hypothyroidism in infancy \& is related to the onset of deficiency .
If early in fetal life $\rightarrow$ Mental retardation, short stature, hernia, skeletal abnormalities, Protruding tongue.

MYXEDEMA in adults $\rightarrow$ Apathy, slow mental processes, cold intolerence, accumulation of mucopolysaccharides in subcutaneous tissue, deepening of the voice and constipation.

## Lab.tests :

## SEVERE HYPOTHYROIDISM ( CRETINS)



از ا كانت الام معها
Hypothroidism
الطفل بينولا معو
Cretinism in infancy early


In adult the patient obese and have constipation ,hair loss

## HASHIMOTO'S THYROIDITIS : CHRONIC LYMPHOCYTIC THYROIDITIS

- Autoimmune disease characterized by progressive destruction of thyroid tissue
- Commonest type of thyroiditis
- Commonest cause of hypothyroidism in areas of sufficient iodine levels
- F:M = 10-20 :1, 45-65 yrs.


## MORPHOLOGY:

- Gland is a smooth pale goiter, minimally nodular, well demarcated.
- Dense infiltration by lymphocytes \& plasma cells
- Formation of lymphoid follicles, with germinal centers
- Presence of HURTHLE CELLS
- Fibrosis if present does not extend outside





## SUBACUTE GRANULOMATOUS THYROIDITIS:

- Middle aged, more in females. Viral etiology ?
- Self-limited (6-8w)
- Acute onset of pain in the neck , fever, $\uparrow$ ESR, $\uparrow$ WBC
- Transient thyrotoxicosis.
- Firm gland.
- Destruction of acini leads to mixed inflammatory infiltrate.
- Neutrophils, Macrophages \& Giant cells \& formation of granulomas



## SUBACUTE LYMPHOCYTIC THYROIDITIS : (SILENT)

- Middle aged females \& post partum patients
- Probably autoimmune with circulating AB
- May recur in subsequent pregnancies
- May progress to hypothyroidism
similar to Hashimoto's thyroiditis without Hurthle cell metaplasia


## - Reidel's Thyroiditis -

- Dense fibrosis without prominent inflammation involving the thyroid and contiguous neck structures.
- ass. with idiopathic fibrosis in other sites of the body.
- Circulating anti-thyroid antibodies, ? Autoimmune aetiology.



## GRAVE'S DISEASE : MORPHOLOGY :

- Smooth enlargement of gland with diffuse hyperplasia \& hypertrophy
- Lining epithelium of acini :

Tall \& hyperplastic $\pm$ papillae

- Colloid :

Minimal thin colloid with scalloped edge

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## Changes in Extrathyroid tissue :

- Generalized lymphoid hyperplasia

Ophthalmopathy : Edematous orbital muscles \&infiltration by lymphocytes followed by fibrosis

Thickening of skin \& subcutaneous tissue
Accumulation of glycosaminoglycans which are hydrophilic acid.

## DIFFUSE \& MULTINODULAR GOITRE

GOITER $=$ Enlargement of thyroid Most common cause is iodine deficiency $\rightarrow$ impaired hormone synthesis $\rightarrow$ 个TSH $\rightarrow$ hypertrophy \& hyperplasia of follicles $\rightarrow$ Goiter
Endemic : > 10\% of population have goiter
Sporadic : 1- Physiological demand
2- Dietary intake of excessive
calcium \& cabbages...etc
3 - Hereditary enzyme defects
4- most cases, the cause is not apperant.

## MORPHOLOGY:

- Initially diffuse $\rightarrow$ nodular with degenerative changes: colloid cysts, hemorrhage, fibrosis, calcification
- If large may extend retrosternally
- Pressure symptoms are a common complaint
- Picture is that of varying sized follicles, hemorrhage , fibrosis, cysts, calcification
- Patient is often EUTHYROID. but may be toxic or hypofunctioning.


Multinodular goitor Enlargment of thyroid gland duo to hyperplasia and hypertrophy of thyroid Commonin endemic areas duo to iodine deficiency And in other areas mostcommon cause is unknown
RT ThYROID TRANS INF


## 1-FOLLICULAR ADENOMA

- Usually single.
- Well defined capsule
- Commonest is follicular士 Hurthle cell change
- May be toxic
- Size 1-10cm. Variable colour


## MACROSCOPIC PICTURE

1- Uniform follicles, lined by cuboidal epithelial cells.
2- Focal nuclear pleomorphism, nucleoli ....
( Endocrine atypia )
3- Presence of a capsule with tumor compressing
surrounding normal thyroid outside .

* Integrity of capsule is important in the differentiation of adenoma from well differentiated follicular carcinoma.
Capsular \&/ or vascular invasion $\rightarrow$ Carcinoma
* Papillary changes : is more likely to prove malignant (no papillary adenoma ).


## ©dition

## Follicular adenoma

## Capsule and inside it normal appearing of the follicles


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## 1- PAPILLARY CARCINOMA :

$\checkmark$ Most common malignant tumor of thyroid gland (70-80 $\%$ ).
Affect female more than male . Cold on Scan by radioactive Iodine.

Capsular and vascular invasion the diagnosis is follicular carcinoma

Gross :small nodules without sharp margins (irregular margins), some of them appear encapsulated .
$\checkmark$ Solitary or multifocal, solid or cystic, $\pm$ calcification.
$\checkmark$ M/E: Composed of papillary architecture with fibrovascular core with cuboidal cells, less commonly may show follicles. The diagnosis is based on NUCLEAR FEATURES : Clear ( Glassy ), with grooves \& inclusions . Psammoma bodies are common.
Metastases mainly by lymphatic (ipsilateral L.N.), sometimes from occult tumor .Hematogenous spread late
$\checkmark$ Prognosis is GOOD ( 10 years survival more than $90 \%$ ).

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FNA of Papillary CA (nuclear changes)



Psammoma body in Papillary CA

## 2- FOLLICULAR CARCINOMA :

$\checkmark$ Account for $15 \%$ of thyroid malignancy . Usually cold but rarely functional ( hot ) Well circumscribed with prominent capsule or infiltrative, composed of follicles ,sometimes of Hurthle Cells.
In well differentiated encapsulated tumors , the diagnosis is based on CAPSULAR \& VASCULAR invasion (*adenoma).
Not all showing histological vascular invasion show metastasis. Metastasize usually by blood $\rightarrow$ Lungs, Bone, Liver ..etc. Treatment by surgery + Radioactive Iodine + Thyroxin.
Prognosis is not as good as papillary except in very well differentiated forms.
$\checkmark \underline{M / E}$ : Tumors composed of cuboidal cells forming follicles filled with colloid or solid nest, strands of less differentiated cells.
Clinically : present as slowelly enlarged painless nodule (cold nodule)




## 3- MEDULLARY CARCINOMA:

Account for $5 \%$ of thyroid carcinoma .
$\checkmark$ Arise from C cells(parafollicular cells) $\rightarrow$ CALCITONIN
$\checkmark 80 \%$ Sporadic, or $20 \%$ familial $\pm$ MEN Syndrome.
$\checkmark$ M/E :Composed of polygonal or spindle cells, usually with demonstrable AMYLOID in the stroma (altered calcitonin deposite).
$\checkmark$ Calcitonin demonstrated in tumor cells .
$\checkmark$ Level of calcitonin in serum may be useful for follow up
$\checkmark$ Calcitonin may be raised in family members, together with demonstrable RET mutation ( Marker for early diagnosis) . Metastases by blood stream .
$\checkmark$ Prognosis intermediate, worse in sporadic \& MEN syndromes.



## 4- ANAPLASTIC CARCINOMA :

$\checkmark$ Markedly infilltrative tumor, invading the neck, rapidly progressive PRESSURE SYMPTOMS.
$\checkmark$ Large cell anaplastic or small cell variant ( undifferentiated cells).
$\checkmark$ Radiosensitive tumor, no surgery.
$\checkmark$ Prognosis is extremely bad (die within 2 years of diagnosis), metastasis to distal site .
$\checkmark$ Morphology: Composed of pleomorphic giant cells, spindle cells or small cell anaplastic varients, which may be confused with lymphoma.
$\checkmark$ P53 mutation identified, consistent with tumor progression.



## C-Parathyroid gland



Normal parathyroid gland


Figure 1


Figure 2

Figure 3

Adenoma in parathyroid gland We not see adipose tissue
See it just in reminant of gland


## Chvostek's sign



An indication of tetany in which a unilateral spasm of the oris muscle is initiated by a slight tap over the facial nerve anterior to the external auditory canal.

Know the difference between $\mathbf{1}^{\circ}, \mathbf{2}^{\circ}, 3^{\circ}$ hyperparathyroidism

- Primary
§PTH $\Rightarrow$ §palcium (normal renal function) -80\% parathyroid adenoma, 15\% parathyroid hyperplasia, carcinoma is rare 5\%
- Secondary

Poor renal function $\Rightarrow \sqrt[\Omega]{ }$ calcium, $\widehat{\mathrm{P}_{4}} \Rightarrow \widehat{\mathrm{PTH}}$ normalca

- Tertiary

Hyperplastic parathyroids from chronic stimulation continue post renal transplant

Edited by : Batool Gharaibeh

