PATHOLOGY LAB





A-pituitary gland

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NORMAL ANTERIOR PITUITARY GLAND

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

Nórmal anterior pituitary gland

Normal pos.pituitary 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).



The patient dye due to large mass of pituitary lesion that compressed of adjecent ventricle and adjecent normal brain. Any The lesion either benign or malignant it called mass effect

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Mass effect of pituitary adenoma

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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 1cm. or more, specially in nonfunctioning tumor
- Hemorrhage & necrosis seen in large tumors (pituitary apoplexy).

Microscopic picture:

- Uniform cells, <u>one cell type (monomorphism)</u>
- 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, ↑BP, HF.....etc

NORMAL PITUITARY GLAND

Reticulin stain highlight the septa between cells

Pituitary adenoma

Monotomas cells Absent reticulin stain

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,hypertension ,diabetic,orthoarthritis

ACROMEGALY V.S DWARFISM



Increase GH due to pituitary adenoma secreting GH cause acromegaly in adult and gigantism in children



Decrease GH due to hypopituitarism or due to SHEEHAN`S SYNDROME or enzyme defect or abnormal development of pituitary gland

GROSS SECTIONS OF PITUITARY ADNOMA



B-Thyroid gland



Follicles lined by flat epithlium

Normal thyroid gland

Hyperthyroidism symptoms



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.

 $\begin{array}{l} \mbox{MYXEDEMA} in adults \rightarrow \mbox{Apathy, slow mental processes, cold} \\ intolerence, accumulation of mucopolysaccharides in \\ subcutaneous tissue, deepening of the voice and constipation. \end{array}$

Lab.tests : ↑ TSH in primary hypothyroidism, unaffected in others↓ T4 in both.

SEVERE HYPOTHYROIDISM (CRETINS)

از اكانت الام معها Hypothroidism الطفل بينولد معو Cretinism in infancy early

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.
- Microscopically :
 - Dense infiltration by lymphocytes & plasma cells
 - Formation of lymphoid follicles, with germinal centers
 - Presence of HURTHLE CELLS
 - Fibrosis if present does not extend outside

Hashimoto`s thyroiditis Normal thyroid tissue with adjecent lymphoid aggregate

一般にお客様など、人に考慮した

Numerous cells could be progress to B cell lymphoma or papillary thyroid carcinoma

SUBACUTE GRANULOMATOUS THYROIDITIS :

Middle aged , more in females. Viral etiology ?

Painfull

- Self-limited (6-8w)
- Acute onset of pain in the neck , fever, ↑ ESR, ↑ WBC
- Transient thyrotoxicosis.
- Morphology :
 - 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
- Histology

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.

Autoimmune disease associated with fibrosis in other organs like pancreas ,salivary glands

Reidle's Thyroiditis

Extensive fibrosis

GRAVE'S DISEASE : MORPHOLOGY :

- Smooth enlargement of gland with diffuse hyperplasia & hypertrophy
- Lining epithelium of acini : Tall & hyperplastic ± papillae
- Colloid :

Minimal thin colloid with scalloped edge

GRAVE`S DISEASE Colloid and follicular cells have tall cell changhes

A LOW LOOP

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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 \uparrow 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 → 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.

THYROID TRANS

Multinodular goitor Enlargment of thyroid gland duo to hyperplasia and hypertrophy of thyroid Commonin endemic areas duo to iodine deficiency And in other areas most common cause is unknown ©WebPathology

Multinodular goitor Normal appearing follicles which is variable in size And cholestrolic cleft In middle And between them fibrosis not well define capsule

1-FOLLICULAR ADENOMA

- Usually single.
- Well defined capsule
- Commonest is follicular + 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).

Follicular adenoma

Capsule and inside it normal appearing of the follicles

Adenoma with intact thick capsule

ices Skavia

With follicles

1- PAPILLARY CARCINOMA :

- \checkmark Most common malignant tumor of thyroid gland (70-80 %). Capsular and vascular
- \checkmark Affect female more than male .
- ✓ <u>Cold</u> on Scan by radioactive Iodine.
- is follicular carcinoma ✓ <u>Gross</u> :small nodules without sharp margins (irregular margins), some of them appear encapsulated.

invasion the diagnosis

- \checkmark Solitary or multifocal, solid or cystic, \pm calcification.
- \checkmark <u>M/E</u>: 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 <u>lymphatic</u> (ipsilateral L.N.), sometimes from occult tumor .Hematogenous spread late ✓ Prognosis is <u>GOOD</u> (10 years survival more than 90 %).

edition Papillary thyroid carcinoma Grossly white lesion

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FNA of Papillary CA (nuclear changes)

Papillary CA

63

Psammoma body in Papillary CA

2- FOLLICULAR CARCINOMA :

- \checkmark Account for 15 % of thyroid malignancy .
- ✓ Usually <u>cold</u> 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).
- \checkmark Not all showing histological vascular invasion show metastasis.
- \checkmark *Metastasize* usually by blood \rightarrow Lungs, Bone, Liver ...etc.
- ✓ <u>*Treatment*</u> by surgery + Radioactive Iodine + Thyroxin.
- \checkmark <u>*Prognosis*</u> is not as good as papillary except in very well
 - differentiated forms.
- \checkmark <u>*M/E*</u>: Tumors composed of cuboidal cells forming follicles filled with colloid or solid nest , strands of less differentiated cells.
- <u>Clinically</u>: present as slowelly enlarged painless nodule (cold nodule)

Follicular Carcinoma

Follicular carcinoma

B

Capsular invasion)

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3- MEDULLARY CARCINOMA:

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

Could be sporadic or familial arising from C cell lead to section of calcitonin that will be deposted as amyiloid

Medullary CA with amyloid

Congo red for amyloid

Medullary carinoma Composed of polygonal or spindle cell along will amyloid This amyloid is acellular eosinophilic can be highlighted by congo red stain

4- ANAPLASTIC CARCINOMA :

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

Parathyroid gland

C-Parathyroid gland

Normal parathyroid gland

Figure 1

Adenoma in parathyroid gland Composed of rest chief and oxyphilic cells

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.

Hypocalcemia

Know the difference between 1°, 2°, 3° hyperparathyroidism

Primary

PTH ⇒ palcium (normal renal function)
 -80% parathyroid adenoma, 15% parathyroid hyperplasia, carcinoma is rare 5%

normal Ca

Secondary

Poor renal function $\implies \int calcium, fo_4 \implies fPTH$

Tertiary

Hyperplastic parathyroids from chronic stimulation continue post renal transplant

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