PITUITARY GLAND

The adult pituitary gland is a small (0.6gram,1.3x 1x0.6cm), : tan, bean-shaped gland that lies at the base of the brain within the sella turcica;

المايسترو هو الغدة النخامية

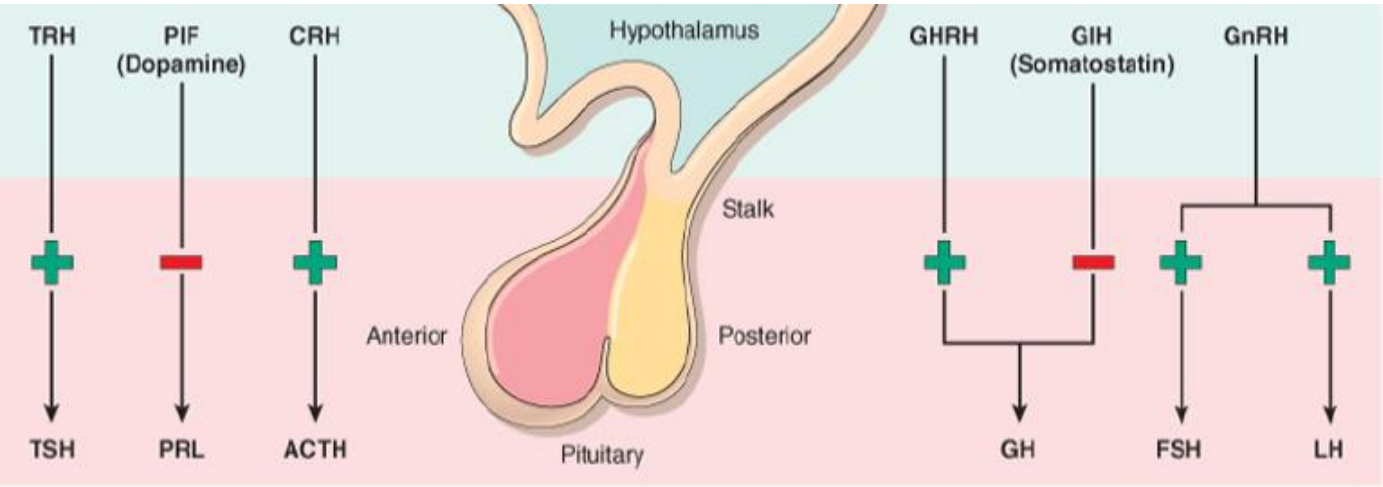
Tan قهوائية فاتحة

bean-shaped فولة

composed of 2 distinct lobes:

1- anterior lobe (adenohypophysis)	2- posterior lobe (neurohypophysis)
occupying 80% of the gland	occupying 20%
composed of epithelial cells derived embryologically from the developing oral cavity, containing basophilic, eosinophilic, or poorly staining ("chromophobic") cytoplasm (depending on the type of trophic hormone within their cytoplasm.) عنا هرمونات متعددة تتكون وشو الهرمون يلي بتكون جوا كل خلية هو يلي بعطيك لونها	

*Under the control of various stimulatory (+) & inhibitory (-) hypothalamic releasing factors, → the J Normal adenohypophysis (anterior pituitary) releases SIX hormones



#Symptoms of pituitary disease can be divided into

1)Hyperpituitarism	2) Hypopituitarism
A disorder arises from excessive secretion of trophic hormones,	A disorder caused by deficiency of trophic hormones
most often results from **<u>functioning</u>** anterior pituitary adenoma, but may also be caused by other pituitary & extra-pituitary lesions	results: from a variety of destructive processes, **the most common is **<u>nonfunctional</u>** pituitary adenomas, which may encroach upon & destroy adjacent native(original) anterior pituitary parenchyma. Encroachتضغط وتدمر **Other causes includes surgery(remove the gland) , radiation, ischemic (chihan syndrome) injury & inflammatory reactions (sarcoidosis)

3)Earliest changes are:**Local mass effects**	**4) invasive** pituitary Adenoma	
دايما بس نفكر بورم بشكل عام كيف بدنا نعرف الاعراض والاثار تبعته ؟ By pressing on adjacent structures When it spreads to blood and lymphatics هاي النقطة معناها انه بس تكبر الغدة النخامية شو رح تعمل	that extend **beyond the sella turcica into the base of the brain** **produce **seizures (epilepsy)** or **obstructive hydrocephalus**; **involvement of cranial nerves** can result in **cranial nerve palsy** ((F8.3))	**Pituitary A, as an **expanding intracranial mass,** may **produce signs & symptoms (S&S) of ↑ intracranial pressure (ICP) (headache + nausea + vomiting)** **Occasionally, ► **sudden severe acute hemorrhage into an A** is associated with clinical evidence of **rapid ↑ ICP & depression of consciousness, a** neurosurgical emergency termed **<u>pituitary apoplexy</u>**, because it may cause sudden death.
#Radiographic abnormalities of the sella turcica (by brain X ray of the skull), including : **sellar expansion ** bony erosion ** disruption of the diaphragma sellae the diaphragmaالخيمة يلي بتغطي الغدة النخامية #Expanding pituitary lesions often compress decussating fibers in the optic chiasm, causing visual field abnormalities, classically in the form of bilateral defect in the lateral (temporal) visual fields, the so- called bitemporal hemianopsia هاد بنشوفه لما نسوي تخطيط للبصر بجهاز خاص فنشوف انه عنا defect of vision in both eyes of temporal area		

HYPERPITUITARISM & PITUITARY ADENOMAS (A)

 # causes :

*The **most common** cause*	Other, *less common* causes include:
***adenoma** arising in the anterior lobe.*	(1) *hyperplasia & carcinomas* of the anterior pituitary, *مجرد زيادة بعدد tumor هاي مو الخلايا* (2) secretion of hormones by some extra-pituitary tumors ex : small lung ca and this is called paraneoplastic syndrome (3) certain hypothalamic disorders.

#types :

*****Pituitary A are classified on the basis of hormone (s) produced by the neoplastic cells *(which are detected by* immunohistochemical *stains performed on tissue sections) into one of following thr*ee types** (Table 20-1)**:**

H&E staining نقسمها حسب انو اكتر هرمون بنتج .....ما بنقدر نحدد النوع ب

لازم نسويلها صبغات لكل انواع الهرمونات فقط بعد استخدام immunohistochemical *stains*

(1) **Functional**	(2) **Silent**	(3) **Hormone negative (Null)**
(i.e., associated with clinical manifestations of hormone excess) الفعال تزيد الهرمونات بالجسم بحيث تعطيك علامات سريرية	**(i.e., associated with immunohistochemical and/or EM demonstration of hormone production at the tissue level only, without clinical manifestations of hormone excess)** **الصامت ما بتعطي علامات سريرية بس بتقدر تشوف الزيادة immunohistochemical and/or** EM	pituitary A (based on **absence** of immunohistochemical reactivity & EM demonstration of lineage-specific differentiation)**.** **هاي اثناء الحياة ما بتعطي علامات ومو مأدية الى زيادة هرمون معين** لما تفحصها ب section بتلاقي adenoma بتسوي immunohistochemical reactivity & EM ما بنلاقي اي دليل على hormone production **Hormone negative** يعني ما بتطلعلك هرمون من الاصل
►**Both functional & silent pituitary A** are usually composed of a **single cell type & produce a single predominant hormone**, but there are **exceptions;** Some pituitary A can **secrete two hormones** (growth hormone & prolactin being the most common combination); عادة ال Adenoma بتكون زيادةتكاثر بنوع واحد من خلايا تسوي نوع واحد من الهرمونات وفي حالات نادرة خلية واحدة ولكنها تنتج هرمونين بنفس الوقت ►Rarely, pituitary adenomas are **plurihormonal.** **Plurihormonal تنتج اكتر من هرمونين ولكنها حالات نادرة جدا**		

***Table 20-1. Classification of Pituitary Adenomas (A)

اعتمادا على الهرمون يلي بتطلعه

<table>
<tr><td>Hermone producing</td><td><table>
<tr><td>Cell</td><td>Tumor</td></tr>
<tr><td>*Prolactin cell</td><td>(lactotroph) A</td></tr>
<tr><td>*GH cell</td><td>(somatotroph) A</td></tr>
<tr><td>*TSH cell</td><td>(thyrotroph) A</td></tr>
<tr><td>*ACTH cell</td><td>(corticotroph) A</td></tr>
<tr><td>*Gonadotroph cell A</td><td>= silent gonadotroph A includes most so-called null cell A</td></tr>
</table></td></tr>
<tr><td>**Mixed (plurihormonal) A</td><td>= GH + prolactin A, the most common mixed adenomas</td></tr>
<tr><td>**Hormone-negative A</td><td>*come to clinical attention at a → later stage
*are more likely to be macroadenomas
*they may cause hypopituitarism as
they destroy adjacent anterior pituitary parenchyma
هي ما بتسوي هرمونات بس بتكبر وتدمرلك بقية الغدة تذكر انه :
The most common cause of hypopituitarism is non functioning A
The most common cause of hyper is functioning A</td></tr>
</table>

Notes

Most pituitary **A** (97%) occur as ***isolated*** neoplasms,

but in about 3% of cases, they are associated with ***multiple endocrine neoplasia type 1*** **(MEN-1).**

Pituitary **A** are either ***microadenomas:*** (if less than1 cm in Ø) or ***macroademomas***: (if they exceed 1 cm in Ø).

#Pathogenesis of pituitary adenomas

(1) One of the **common genetic alterations** (present in 40% of GH -secreting somatotroph cell A & in a minority of ACTH -secreting corticotroph cell A) is mutation of the ***GNAS1*** **gene**, which results in constitutive activation of a stimulatory G-protein, *A mutation in the* α *-subunit that interferes with its intrinsic GTPase activity therefore results in constitutive activation of Gs* α*, persistent generation of cAMP, & unchecked cellular proliferation.*

هدول بس للاسترشاد والمعرفة وغير مطالبين فيهم بالامتحان

(2) By definition, Pituitary A that arise in the context of familial MEN-1 syndrome harbor mutations in the ***MEN-1*** gene.

(3) Additional molecular abnormalities present in ***aggressive or advanced pituitary A*** include activating mutations of the ***RAS*** **oncogene**, overexpression of the ***C-MYC*** **oncogene**, & inactivation of the metastasis suppressor **gene *NM23*,** suggesting that these genetic events are linked to disease progression.

#Morphology

#grossly :

Small pituitary **A**	**Larger adenomas**
is **well-circumscribed** & **soft** lesion **(Why? Because there is very little stroma), confined** by the sella turcica واضحة المعالم من الخارج ومحاطة ب the sella turcica	*commonly with foci of hemorrhage and/or necrosis, ***extend** (I) superiorly through the sellar diaphragm into the suprasellar region, where they **compress the optic chiasm & adjacent structures (8.3 &F20-3**), (II) frequently **erode** the sella turcica & anterior clinoid processes, يمكن رؤية هذه التغيرات بالاشعة (III) extend locally into the cavernous & sphenoidal **sinuses**. ►**30% of** pituitary **A are {invasive adenomas}, they are grossly not encapsulated & infiltrate adjacent <u>bone, dura, & (uncommonly) brain.</u>** **هي ورم حميد بس تكون بهاي الحالة غازية بالعين المجردة ما الها capsule وتغزو يلي تحته خط**

#Histology

◙H,

***pituitary uniform, polygonal cells** composed of **A** arranged in sheets, cords, or papillae.

*Supporting connective tissue, or reticulin, is sparse which means very little (resulting in **softness** of **A**) .

*The nuclei may be **uniform or pleomorphic**& mitotic activity is usually scanty.

Pleomorphic متعدد الاشكال

Scanty قليل جدا

*The cytoplasm of the cells may be acidophilic, basophilic, or chromophobic like the origin , depending on the type & amount of secretory product within the cell.

صورة 3-20 لو شرحنا يلي ماتوا بسبب زيادة IC pressure رح نلاقي انه adenoma سبب نادر جدا

ال A لو كانت functioning وزادت هرمون معين رح تعطي clinical manifestations والمريض يسعى للعلاج ويعالج

ولكن لوكانت non functioning تضل تكبرتكبر الى ان تسوي late clinical manifestations

هون عبرت الى base of brain وصعدتواستحوذت على تقريبا ثلث او اكتر من brain tissue

هون هي اصلا حجمها الاصلي نص غرام هي صارت ٦٠ ل ٧٠ اضعاف حجمها الاصلي وهذا هودليل انها non functioning

نوتس على صورة 4-20 :

ب ant pituitary gland النورمال تتكون من تلت انواع من الخلايا acido baso chromophobe اما هون كل الخلايا وحدة monomorphism وهاد معناته انه في neoplastic proliferation تكملة الشرح النقطة يلي تحت

*This cellular monomorphism & the absence of a significant reticulin network **(no supporting stroma so it is very soft) distinguish pituitary adenomas** from non-neoplastic, **normal** anterior pituitary parenchyma (F20-4).

*The functional status of the **A cannot** be reliably predicted from its histologic appearance(by H & E stain) so we have to do immunohistochemistry staining for all hormones produced by ant pituitary .

#Clinically :

*diagnosed pituitary **A** accounts for about **10% of intracranial neoplasms**.

*They are discovered incidentally in as many as **25% of routine autopsies after death**.

*More recent CT & MRI studies (during life) suggest that approximately **20% of "normal" adult pituitary glands harbor an incidental** lesion measuring **3 mm** or more in Ø, usually **a silent adenoma**.

normal يظهر عليهم طبيعين جدا

*Pituitary A are usually found in adults, with a peak incidence from the 30s to the 50s.

Prolactinomas

#Def.

*Prolactinomas are the **most common type of hyper functioning pituitary A**.

*They range from small, to large expansile tumors associated with considerable mass effect.

effects of local mass ارجعوا للجدول تبعون

*Prolactin is demonstrable within the cytoplasm of the neoplastic cells by immunohistochemical techniques

IHC يعني بتقدر تشوف انه فيA بس ما بتقدرتعرف نوعها بزبط الا لما تستخدم
واذا كانت + بس ل prolactin وقتها بنحكي انها Prolactinomas

#clinical manifestations :

*__*Hyperprolactinemia*__ causes

1- galactorrhea : milk will be secreted from breast

2- amenorrhea loss of menstrual cycle

3- loss of libido (sexual desire) , & infertility,

*manifestations more obvious in premenopausal females than in males or postmenopausal females; therefore, prolactinomas are usually **diagnosed at an earlier stage in females of reproductive age than in other individuals**.

*In contrast, hormonal manifestations may be quite

subtle (mild) in men & older women, in whom the tumors may

reach considerable size before coming to clinical attention.

لانه ما في علامات ف رح يجو بمرحلة متأخرة من المرض

*In addition to prolactin-secreting pituitary **A**, **hyperprolactinemia** may be **caused** by pregnancy, high-dose estrogen therapy, renal failure, hypothyroidism, hypothalamic lesions, & dopamine-inhibiting drugs (e.g., **reserpine**).

* In addition, **any mass in the suprasellar compartment** may disturb the normal inhibitory influence of hypothalamus on prolactin secretion, resulting in hyperprolactinemia, a condition known as the ***stalk effect,…***

*therefore, it should be kept in mind that ***mild*** elevations of serum prolactin (<200 μ g/L) in an individual with a pituitary adenoma do not necessarily indicate a prolactin-secreting neoplasm

لوكان ارتفاع بسيط بالبرولاكتين فهو مو شرط يكون prolactinomas ولكن لو كان عالي جدا فهي case of prolactinoma

هاي الجملة يلي فوق عادها مرتين وحكا ضروري نعرفها

Growth Hormone-Producing Adenomas

*GH-producing **(somatotroph cell**) **A** including those that produce a mixture of GH & other hormones (e.g., prolactin), are the **second most common type of functional pituitary A.**

*Because the clinical manifestations of excessive GH may be subtle **(mild),** somatotroph cell **A** may be **quite large** by the time

they come to clinical attention.

◙ H, GH-producing **A** composed of densely or, sparsely, granulated cells, & immunohistochemical stains demonstrate GH within the cytoplasm of the neoplastic cells.

*Small amounts of immunoreactive prolactin are often present as well.

*Persistent hypersecretion of GH stimulates the hepatic secretion of insulin-like GF I (somatomedin C), which causes many of the clinical manifestations.

If a GH-secreting **A occurs **before the epiphyses (growth plate in long bones) close**	*If elevated levels of GH persist, or present **after closure of the epiphyses**
as is the case in prepubertal children, excessive GH result in ***gigantism***, characterized by a generalized ↑ in body size, with disproportionately long arms & legs. ***لو GH زادت قبل اكتمال نمو العظام العظام رح تنمو اكثر من لازم ....*** ***وبذلك تؤدي الى Gigantism*** العملقة	, individuals develop ***acromegaly,*** in which **growth** is most conspicuous in soft tissues + skin+ viscera+ bones of the face, hands,& feet. Enlargement of the jaw (especially in lower jaw) results in its protrusion **(prognathism),** with broadening of the lower face &separation of the teeth. The hands & feet are enlarged, with broad, sausage-like fingers.

*In practice, most cases of gigantism are also accompanied by evidence of acromegaly.

*GH excess is associated with → abnormal glucose

tolerance + **DM + hypertension + CHF + generalized**

muscle weakness + arthritis + osteoporosis.

*Prolactin is demonstrable in a number of GHproducing

A & in some cases may be released in sufficient quantities to produce S & S of **hyperprolactinemia.**

في بعض الاحيان بالاضافة لزيادة GH بتصير تفرز prolactin فبتعطيك علامات تبعت ال**hyperprolactinemia**

Corticotroph Cell Adenomas (ACTH-Cell A)

*Are mostly **microadenomas** at the time of diagnosis **(Why? because it produces sufficient amount of ACTH which result in clinical manifestations)** although some may be quite large (in case it does not lead to sufficient amount of ACTH) .

*These **A** stain positively with PAS stains, as a result of the accumulation of glycosylated ACTH protein, & the secretory granules can be detected by immunohistochemical methods**.**

طيب هلا كيف بدنا نعرف النوع عن طريق الصبغة يلي حكيناها بس لازم اول شي نجيب العينة كيف ؟

You pass through the nose through the base of the skull ….. reach the pituitary adenoma and remove it

This is called transnasal which used for many years

There are other methods like MRI and X ray

▼ **ACTH-cell A** may be **silent (without increase in ACTH)** or may cause ***hypercortisolism*** **(also known as *Cushing syndrome*)** because of the stimulatory effect of ACTH on the adrenal cortex. Such process is designated ***Cushing disease*** **(**because it is the pattern of hypercortisolism originally described by **Dr. Harvey Cushing)**.

انتبه هاي ال A بتطلع كميات كبيرة من ACTH في ال pituitary يلي بروح يحفز both adrenals gland باليمنى واليسرى يخليهم ينتجوا كميات كبيرة من الكورتيزون فالحالة تنتنهي بشو ؟***hypercortisolism والمرض بسموا***

Cushing disease

ولكن عنا حالات كتيرة اخرى بصير فيها hypercortisolism ولكن اما نتيجة اخذ الكورتيزون بالفم او بسبب hyperplasia or tumor of adrenal وهدول كلهم اسمهم

hypercortisolism (also known as Cushing syndrome) انتبه انه مش syndrome disease

▼ ***Nelson syndrome***:

*characterized by the development of large, clinically **aggressive corticotroph A** in the pituitary after surgical **removal of the adrenal glands** for treatment of adrenal Cushing syndrome

هاااي مهمة جداو افتهموها

القصة انه مريض عنده A or hyperplasia بال adrenal فشلناها اليمنى واليسرى ونتيجة لذلك ب pituitary شو رح يصير **aggressive corticotroph A** عشان راح ال inhibitory effects

*because of a loss of the inhibitory effect of adrenal corticosteroids on a preexisting corticotroph microadenoma.

*Nelson syndrome, there is no hypercortisolism (because the adrenals are already removed) & instead, patients present with local mass effects of the pituitary tumor. In addition, there may also be **hyper-pigmentation**

Other Anterior Pituitary Neoplasms

Gonadotroph *(luteinizing hormone **[LH]**-producing & follicle stimulating hormone **[FSH]**-producing)* **A** can be difficult to recognize, because they secrete hormones **inefficiently,** variably & the secretory products usually **do not cause a recognizable clinical syndrome.**

كمية الهرمونات يلي بنتجها هدول الورمين

***[LH]-producing [FSH]-producing&* تكون قليلة وعادة لا تؤدي الى عوارض سريرية عشان هيك بتجيك الحالة وبتكون كبيرة**

*Gonadotroph **A** are most frequently found in middle-aged men & women when the tumors are large enough to cause **mass effects (Why?),**

*with neurologic symptoms, like impaired vision, diplopia, headaches, or pituitary apoplexy (hemorrhage) which lead to sudden death as we said in last lecture .

المريض ما بيجي بالعلامات تبعون زيادة الهرمونات هدول انما بيجي بعلامات local mass effect

*The neoplastic cells usually demonstrate immunoreactivity for the common gonadotropin α -subunit & the specific β -FSH & β -LH subunits; **FSH is usually the predominant secreted H**.

ال effects تبعون الهرمونات التفاصيل تبعتها نوخدها بالفسيو

الجملة يلي تحتهاخط عادها مرتين

*The availability of reliable immunoassays for the gonadotropin β -subunit & the recognition of gonadotroph specific transcription factors have led to the reclassification of many previously described hormone-negative **A** *("null cell **A** ")* as silent gonadotroph **A**.

بما انه قدرنا نقحصها ب immunoassay وقدرنا نفحصها بالتحليل المختبري معناته هاي silent وخرجت من خانة null or negative لانها بالحقيقة بتسوي كميات قليلة منهم

Thyrotroph A

*(thyroid- hormone [TSH] producing) ***A* are rare,** accounting for **about 1% of all pituitary A & are a very rare cause of hyperthyroidism.**

Nonfunctioning pituitary A comprise both:

هي نوعين

(1) clinically **silent** counterparts of the functioning **A** described above (e.g., a *silent gonadotroph adenoma*) & the

(2) Rare **true *hormone-negative*** **(null cell) A.**

هاي ما بتسوي ولا اشي ولا حتى كمية قليلة لا كلينيكال ولا ترى ب immunoassay

® While **75%** of all pituitary tumors are **functioning**……

, and typically **25% are nonfunctioning A.** the remaining 25% resent with either:

(A) **mass effects,** and/or

(B) they may destroys the residual anterior pituitary to produce **hypopituitarism.**

Pituitary carcinomas

****are exceedingly rare.***

*In addition to local extension beyond the sella turcica, these tumors virtually always have distant metastases

HYPOPITUITARISM (anterior pituitary)

يعني قلة انتاج الهرمونات من الغدة النخامية

#def. :

▼ Hypo function of the **anterior** pituitary occurs with **loss or absence of 75% or more** of the anterior pituitary parenchyma (for any reason).

متى بنقدر نشخص هاي الحالة اذا تدمر ٧٥ بالميةمن ant pituitary تدمر لاي سبب من الاسباب

#causes :

<table>
<tr><td>►Congenital</td><td colspan="2">► Acquired abnormalities:</td></tr>
<tr><td rowspan="2">(exceedingly rare)</td><td>1- intrinsic to the pituitary</td><td>2- ® less frequently, Hypothalamic tumors & other disorders</td></tr>
<tr><td>Main causes of anterior pituitary hypofunction are
(I) The most common is nonfunctioning pituitary adenomas ,destroying the residual gland
يعني A ما تشتغل تكبر تكبر وتدمرلك بقية الغدة وتؤدي الى hypo
(II) Ablation of the pituitary by surgery or radiation.
Ablation محي وتدمير كامل
III) Necrosis of the anterior pituitary, of which
(a) the most common, clinically significant cause, is postpartum ischemic necrosis of the anterior pituitary Sheehan syndrome
** details below
necrosis may also occurs in
(b) DIC = disseminated intravascular coagulation
نتيجة وجود عدد هائل من thrombi تدمرلك
brain ,pituitary .... بالجسم من many organs
(c) sickle cell anemia,
(d) ↑ ICP by compressing pituitary gland
(e) traumatic head injury may result in pituitary injury
(f) shock of any origin(hypovolemic ,cardiogenic , septic shock).
عادهم مرتين
(IV) less common causes include inflammatory lesions such as TB, or sarcoidosis, trauma, & metastatic neoplasms involving the pituitary.
(V) Rarely, mutations affecting the pituitary transcription factor Pit-1 can cause multihormonal deficiency.</td><td>that interfere with the delivery of pituitary hormone-releasing factors from the hypothalamus to the anterior pituitary.

®<u>Hypopituitarism accompanied</u> by evidence of <u>posterior pituitary dysfunction</u> in the form of <u>diabetes insipidus</u> <u>is almost always of hypothalamic origin</u></td></tr>
</table>

►*Sheehan syndrome*

* **During pregnancy** the anterior pituitary **enlarges** considerably, largely because of an ↑ in the size & number of prolactin-secreting cells.

بصير زيادة في عدد وحجم الخلايا يلي بتنتج بروﻻكتين تحضيرا لعملية انتاج الحليب بالثدي والرضاعة

*However, this physiologic enlargement of the gland is **not accompanied by an ↑ in blood supply** from the low-pressure portal venous system.

هذا ال enlargement لا يصاحبه زيادة بكمية الدم يلي بتيجي

ما هي النتيجة ؟ يلي تحت

*The enlarged gland is thus vulnerable to ischemic injury, especially in women who develop significant **hemorrhage** & hypotension during the peripartum period.

بتصير عرضة ل ischemia لانها كبيرة والدم يلي بيجها قليل

يعني اذا المرأة فقدت دم بالمدة يلي حوالين الولادة سواء قبل الولادة او بعد وغالبا بكون بعد

فاذا صار نزيف كمية الدم اصلا يا دوب مكفية فبتقل اكتر وبصير ل pituitary نقصان اكتر واحتمال ischemia وهاد بنسمي Sheehan syndrome ولكن هاد الحكي ب ant pituitary طب post ؟؟ ما رح تتأثر ليش ؟

* The **posterior pituitary**, is much less susceptible to ischemic injury in this setting, because it **receives its blood directly** from arterial branches, & therefore, it is usually **not affected**.

*In **ischemic necrosis,** *the residual gland is shrunken & scarred + infarction* معناته صار احتشاء

it will not recover …..it will be removed by macrophages and replace by fibrous tissue (scarred)

#The clinical manifestations

*depend on the specific hormone(s) that are lacking:

Hormone	clinical manifestations
► **GH** deficiency	in children can results in growth failure ***(pituitary dwarfism)*** تقزم بسبب الغدة النخامية You have to differentiate it from thyroid dwarfism In ***pituitary dwarfism*** : mental state and neurological function are normal …. All body parts reduce Whereas in thyroid dwarfism : In addition to growth failure there is mental abnormalities because of decrease thyroid hormone
► **Gonadotropin** or gonadotropin-releasing hormone (GnRH) deficiency	*in women leads to: amenorrhea & infertility *in men: ↓ libido, impotence, loss of pubic & axillary hair
► **TSH** deficiencies	result in failure to stimulate thyroid >>> symptoms of **hypothyroidism (secondary not primary)**
ACTH deficiencies	**No stimulation of adrenal cortex to produce cortisone >>> hypoadrenalism**
► **Prolactin** deficiency	results in failure of postpartum lactation خصوصا بحالات Sheehan syndrome الام ما رح يكون عندها حليب حتى ترضع الطفل لانه الخلايا يلي بتنتج بروﻻكتين تدمرت
►**MSH (Melanocyte stimulating hormone)** deficiency	Results in pallor from loss of stimulatory effects of MSH on melanocytes

POSTERIOR PITUITARY SYNDROMES

☼ **Anti Diuretic Hormone (ADH)** acts on the renal collecting tubules, to promote the resorption of free water.

ADH deficiency causes ***diabetes insipidus,*** with an inability of the kidney to resorb water from the urine, &

→ **excretion of large volumes of dilute urine** with an inappropriately **low** specific gravity(What is specific gravity)

specific gravity هو الوزن النوعي يعني ١ مل من الماء يساوي ١ غم

هون ال urine تساوي ١٠٢٠ – ١٠٤٠ بحالة **ADH** deficiency رح تصبح low لانه بصير مخفف

وهاي الشغلة بنقدر نستفيد منها بالتشخيص انه اول volume من urine الصبح بكون low specific gravity بالاضافة انه بنقدر نقيس levels of ADH

→ ↑ Serum Na & osmolality, resulting in **thirst & polydipsia**

لانه بس ينقص ال ADH رح يبطل قادر انه يمتص98% من المي و ينزل ب urine 2% من المي ويصفي لتر ل لتر ونص يوميا بس بهاي الحالة رح يصير يعبر مو ١و نص لتر لا رح يصير يعبر large quantities of urine لترين ثلاثة وهذا رح يؤدي الى زيادة تركيزالصوديوم بالدم وشعور شديد بالعطش وشرب كميات كبيرةمن المياه

Causes of diabetes insipidus are:

***head trauma, surgical procedures, tumors** & **inflammatory** disorders of the hypothalamus & pituitary**.**

*Some spontaneous **idiopathic cases** arise, in the absence of an underlying disorder.

#types :

Diabetes insipidus is either:

(I) Central diabetes insipidus results from **ADH deficiency,** or

هون بتعطي ADH وتتعالج الحالة

(II) *Nephrogenic* diabetes insipidus results from renal tubular *unresponsiveness* to circulating ADH.

بهاي الحالة ال ADH موجود بكميات كافية بس المشكلة انه الكليةلا تستجيب وبهاي الحالة لو اعطيته ADH ما رح يتحسن

#**The clinical manifestations** of both central & nephrogenic diseases are similar (So, **how** they can be differentiated?)

by administration of ADH (nasal spray) if the patient status improve >> central ….. if not >>> nephrogenic

*Patients who can drink water can generally **compensate** for urinary losses; but L bedridden patients, or patients with limited ability to obtain water, may develop N **life-threatening dehydration.**

S*yndrome of inappropriate ADH (SIADH)* secretion, ADH **excess** is caused by many extracranial & intracranial disorders.

►The most common causes of SIADH include:

the secretion of **ectopic ADH by malignant neoplasms** (particularly **small-cell carcinomas of the lung, SCCL**), non-neoplastic diseases of the lung, &

local injury to the hypothalamus and/or neurohypophysis.

SIADH causes → **resorption** of excessive amounts of free water, with → **resultant hyponatremia,** causing → **cerebral edema**, & **resultant neurologic dysfunction**.

Although total body water is ↑, blood volume remains normal &

peripheral edema does not develop