

THE ADRENAL GLANDS

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Adrenal glands* suprarenal glands*
are small, triangular-shaped glands
located on top of both kidneys.

Retroperitoneal

Blood supply

superior adrenal art- inferior phrenic art.

The middle adrenal art- abdominal aorta.

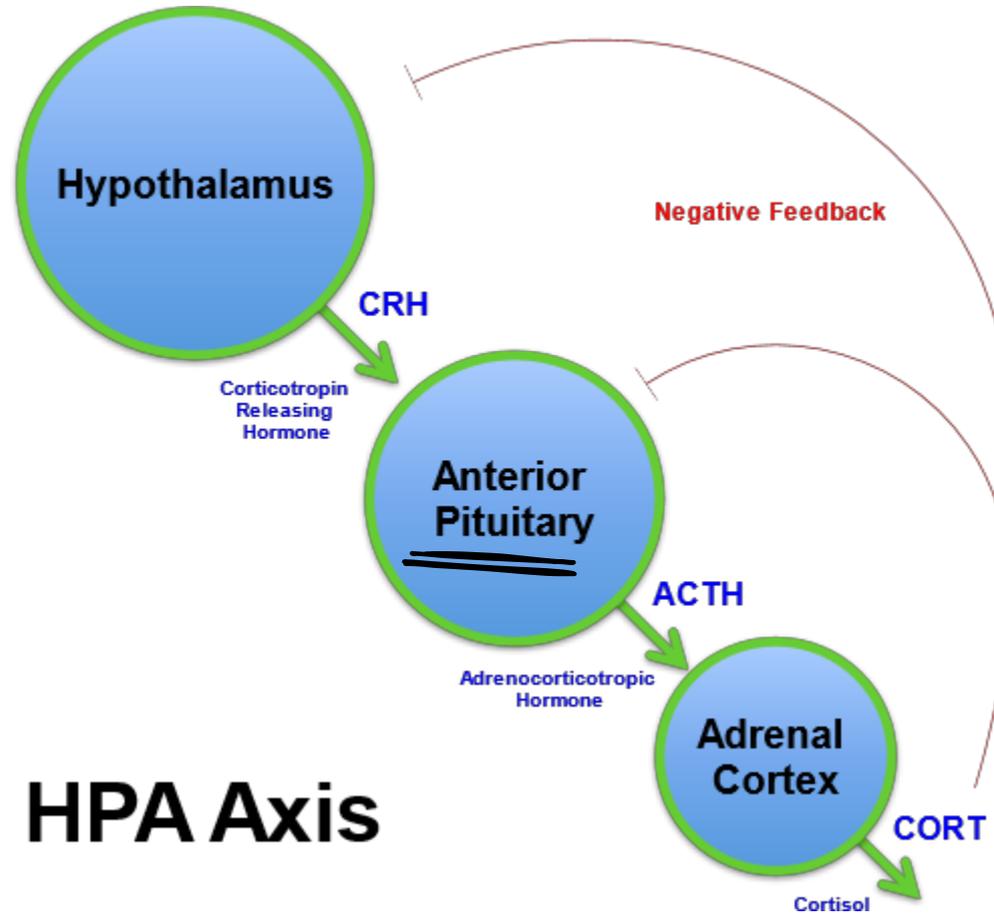
The inferior adrenal art- renal artery bilaterally

Venous drainage renal vein

THE ADRENAL GLANDS

- Adrenal cortex:
- Zona glomerulosa... Mineralocorticoids, Aldosterone
- Zona fasciculata.....Glucocorticoids cortisol
- Zona reticularis.....Sex Hormones
- Adrenal medulla : Adrenaline
 - Noradrenaline
 - Dopamine

Hypothalamic – pituitary – adrenal axis



Adrenal incidentaloma (non-functional)

Non- functional adrenal mass

incidentaloma

- * More than 1 cm
- * Asymptomatic
- * Found incidentally
- * Diagnosed by radiological imaging / ct

Incidental Adrenal Mass (>1 cm) diagnosed on CT/MRI
 Hormonal evaluation in all patients

1. Dexamethasone (1 mg) suppression test
2. Plasma or 24-h urine metanephrines
3. If hypertensive, include plasma aldosterone:renin ratio

Functional Mass
 (Hormonal evaluation abnormal/positive)

Endocrinology Consultation

- Confirmation testing of autonomous secretion of cortisol, catecholamines, aldosterone, other
- Medical and preoperative management

Consider surgery

Positive autonomous hormonal secretion
 Growth >1 cm
 Size of mass ≥ 4 cm

Nonfunctional Mass
 (Hormonal evaluation normal/negative)

Size of adrenal mass <4 cm

Size of adrenal mass ≥4 cm

Benign Imaging Features

- Homogenous
- Low density
- Smooth margins
- Unenhanced CT ≤10-HU attenuation

Suspicious Imaging Features

- Heterogeneous
- Necrosis
- Irregular margins
- Unenhanced CT >10-HU attenuation

Take Biopsy

CT with contrast

↑ fact
 ≥50% contrast washout at 10 min

<50% contrast washout at 10 min

Consider Conservative Management

- Repeat imaging 6–12 mo
- Repeat hormonal evaluation annually for 4 yr

Consider Surgery

- If history of malignancy: PET-CT, biopsy

Functional Adrenal Abnormalities

- *Benign or malignant tumors or hyperplasia*
- **Cortex** : *Cortical tumors* :
 - Cortisone secreting tumors-**Cushing's Syndrome**
 - Aldosterone secreting tumors-**Conn's Syndrome**
 - Sex hormone secreting tumors-**Virilisation** or **Feminization**.
 - ↳ male physical characteristics (such as muscle bulk, body hair, and deep voice) in a female

Diffuse Hyperplasia

- *Primary or a consequence of stimulation by trophic hormones leading to hypercortisism, Conn's disease or Adrenogenital syndrome*

Stimulation

1. Brain
2. Disease outside Brain Release Hormone
3. Adrenoc

Medulla

- Tumors secreting adrenaline/nor-adrenaline
(Phaeochromocytoma)

Cushing's Syndrome

=Primary adrenal disease:

- Adenoma
- Carcinoma
- Primary adrenal hyperplasia “ ACTH independent

=Secondary adrenal disease:

- Primary pituitary micro-adenoma
- Non pituitary source “ Ectopic ACTH syndrome “

Cushing's Syndrome

- ***Definition:***

Excess circulating cortisol that occurs as a result of endogenous steroid hyper secretion, due to:

ACTH dependent or

ACTH_ independent disease

Or exogenous steroid medication.

ACTH-Dependent *Secondary*

1. Pituitary microadenoma. *M/C* - Cushing disease -

2. Ectopic ACTH secretion:

Small cell carcinoma. Lung.

Fore gut carcinoid. Endocrine tumor - ACTH.

Ectopic CRH Syndrome:

✧ *Medullary thyroid tumor.*

✧ *Pancreatic neuro-endocrine tumors*

ACTH Independent Primary

- Adrenocortical Adenoma
- Bilateral nodular hyperplasia
- Adrenal carcinoma.

Cushing's Syndrome

- Physiological and bodily changes caused by excess of circulating cortisol:
- Commonest cause is **iatrogenic**: *administration of steroids for the treatment of other diseases*

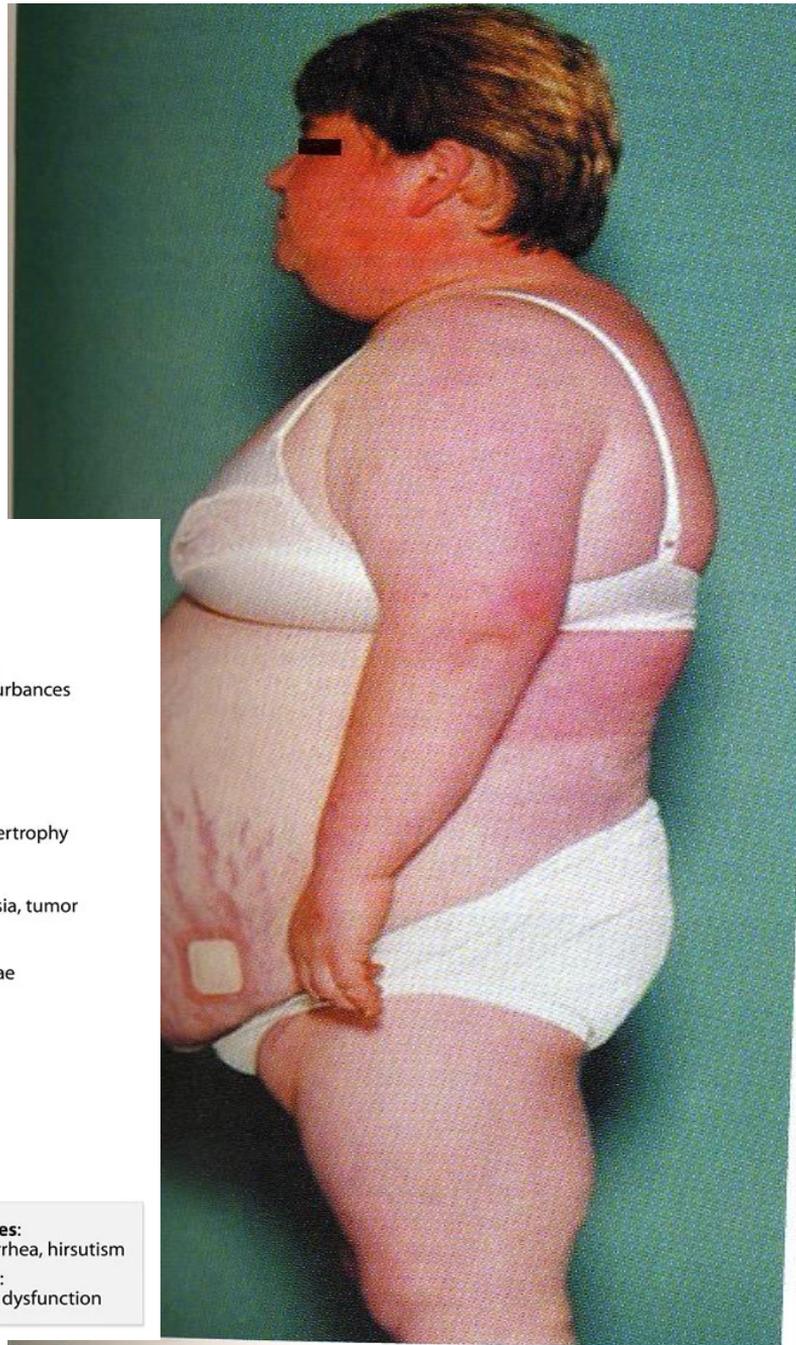
Action of glucocorticoids

- **Glucose metabolism**
 - **Peripheral glucose utilization**
 - **Lipid metabolism**
 - **Cells of immune system**
 - **Mediators of inflammation**
 - **Bone and minerals metabolism**
 - **Soft tissue and skeletal growth**
 - **Fluid and electrolytes homeostasis**
 - **C N System**
-) → 2M .

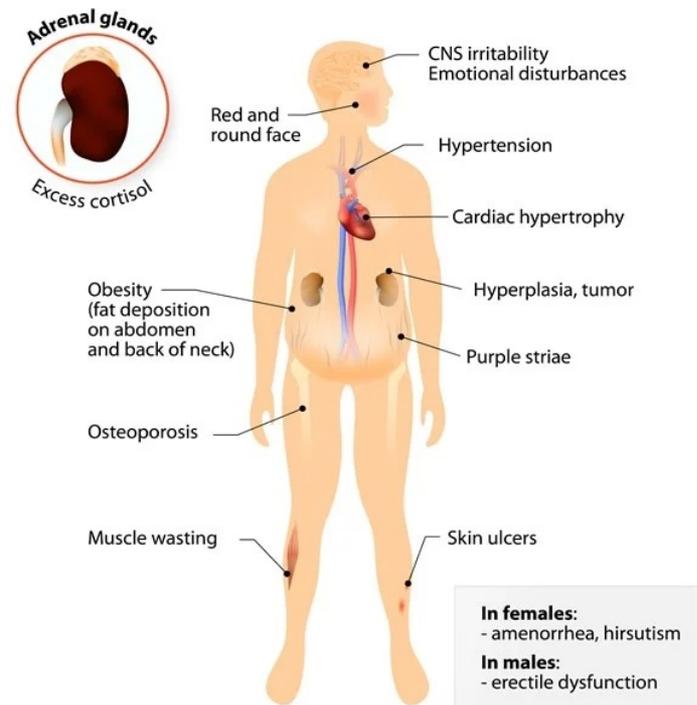
Clinical presentation

- Obesity
- Loss of connective tissue
- Hirsutism and Virilism
- Muscle weakness
- Osteoporosis
- Hypertension
- Glucose intolerance - DM
- Psychological changes

typical



SYMPTOMS of Cushing's syndrome



Ectopic ACTH Secretion

- Rapid evolution of the Cushing;s
- Symptoms of the primary disease:
 - Small cell carcinoma of the lung
 - Carcinoid
 - Medullary Ca of Thyroid
 - Other primary carcinomas

Investigations:

①: Biochemical **diagnosis**

- Persistent increase in cortisol concentration. *Day - Night*
 - Cortisol suppression by dexamethasone ^{*Normally ↓ Cortisol*} → *Cushingy Not ↓*
 - Resistancy to insulin administration - DM -
 - 2 : Establishment of the **cause**
 - **Low ACTH = Adrenal disease**
 - **High ACTH = Extra- adrenal cause.**
- } → *-ve feedbacks .*

Anatomical details

- Pituitary: Skull X ray

CT

MRI

- Adrenals: US

CT

MRI

- Scintigraphy - cholesterol scan

- NP 59 scan

- Search for ectopic ACTH source

CT chest

Angiography

** Steroid scan * Nb-95*
most specific



Plan of Management

- Pituitary adenoma : Microadenectomy ✓
- ^{90% Bilateral} Hyperplasia : Bilateral adrenalectomy
- ^{Cancer - Unilateral} Solitary adenoma: Unilateral adrenalectomy

Perioperative Care

High level of cortisol - ما اوقفه فجأة -

Pre op high dose - continuous post op

then tapering

Adrenocortical Carcinoma

- Rare
- Any age 4-5th decades
- 60% : no important secretory function
- Benign or Malignant ? Pain
 - Weight loss
 - Weakness
 - Fever
- Functional tumors present depending on their type of secretion .

Treatment

- When possible Surgical resection
- Radiotherapy
- Chemotherapy

Aldosteronism

* Conn's Syndrome *

- **Primary due to :** tumor (Adenoma)
nodularity
hyperplasia

Secondary due to: Excess stimulation by Angiotensin

Commonest cause is :

“**Aldosterone producing Adenoma**”

Incidence: Females more than males

30—60 years of age

1% of patients investigated for hypertension

Pathophysiology

- Aldosterone :

Promotes sodium absorption

Promotes water retention

Increase potassium secretion

↑ Na ↓ K

Clinical features

Clinical suspicions should be raised when

Hypertension occur with hypokalemia.

- Moderate to severe hypertension
- Hypokalemia
- Muscle weakness
- Malaise
- polydipsia

Investigations

- Blood : Hypokalemia

Plasma aldosterone

- Urine : Increase urinary potassium

- Imaging : U S

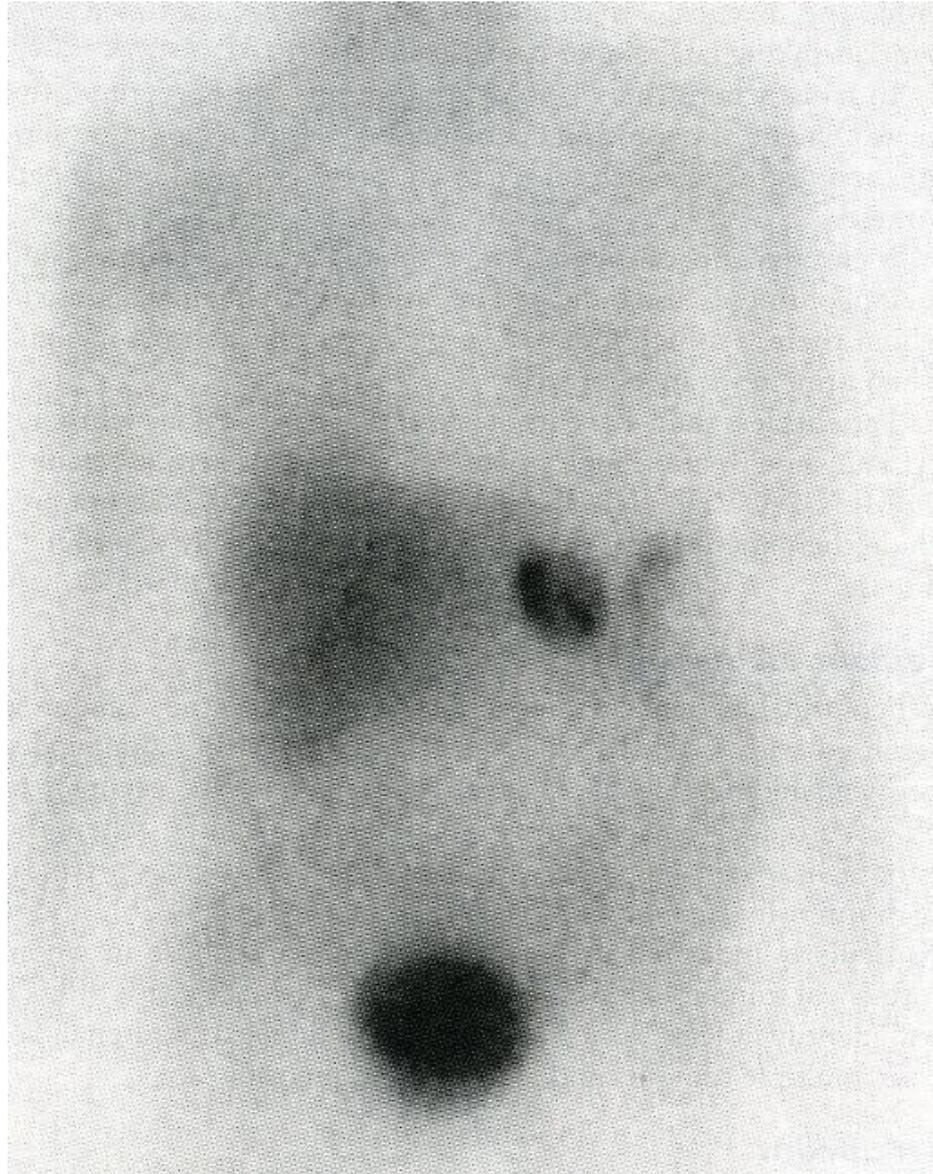
C T

M R I

Iodocholesrerol isotope

scan

Adrenal vein sampling - Hyperplasia



Treatment

- Spironolactone / Potassium-sparing diuretics
- ⦿ Adrenalectomy

Phaeochromocytoma

Phaeochromocytoma

Neuroblastoma

Paraganglioma

Ganglioneuroma

Are derived from the neural crest

tumor of 10%

Phaeochromocytoma

- 90% ---solitary – adrenal
- 5 –10% bilateral
- 10%---Exrta-adrenal
- 0.1% of patients investigated for hypertension
- Average size is 5 cm
- Discovered early because of catecholamines effects
- 10% are malignant
- Mostly secretes adrenaline

10% Bilateral

10% MTN

10% Extra Adrenal

10% malignant

Symptomatology

Young - MTN

- ⊙ Palpitation
- ⊙ Hypertension] M/C
- ⊙ Sweating and pallor
- ⊙ Anxiety
- ⊙ Chest pain & weakness 50%

Symptomatology

- Attacks often occur spontaneously but may be precipitated by vigorous exercise, Alcohol, tobacco and drugs : Anesthesia, phenothiazines & tricyclic antidepressants.

— Must rule out other neuroendocrine tumor $\frac{0}{0}$

Clinical associations

* Multiple endocrine neoplasia type 2 - β

Phaeo, medullary thyroid ca, hyperparathyroidism

* Neurofibromatosis .10% of patients with neurofibromatosis may develop phaeochromocytoma

HTN → High Risk to
Surgical Intervention.

Investigations

- A– 24 hours urinary vanyl mandilic acid (VMA) 60% sensitive.
- Urinary catecholamines . 90% sensitive
- Localization: C T scan
M R I
M I B G , isotope scan

Management:

= Adrenalectomy

- Preoperative management
- Operative management
- Post operative management