Adrenal glands:

Anatomy

Is located above (or attached to) the upper pole of the kidney. pyramidal in structure and weighs ~ 4 g . Consists of the adrenal cortex and adrenal medulla. A retroperitoneal structure in its own sub-compartment in Gerota's fascia.

The adrenal gland:

1- Medulla: epinephrine and norepinephrine.
2- Cortex (from inner to outer):
   a- Reticularis ? androgens (sex hormones).
   b- Fasciculata ? cortisol
   c- Glomerulosa ? aldosterone

Blood supply of the adrenal gland :

Arterial sources of adrenal blood supply ? inferior phrenic artery , aorta and renal artery .

Venous ? right : post IVC

left : renal vein

it is consisted of the cortex(mesoderm, regulated by anterior pituitary) and medulla(neuroectoderm , regulated by sympathetic N).

comparison between right and left suprarenals :

physiology:

medulla (short-term stress response) secretes epinephrine and nor-epinephrine:

1- Increase heart rate
2- Increases blood pressure
3- Conversion of glycogen to glucose and release it to the blood
4- Bronchioles dilation
5- Increase metabolic rate
6- Change blood flow pattern so(increase alertness , decrease digestive system activity and reduce urine out put).

Cortex (chronic / long -term stress response)
Mineralcorticoids/glomerulosa (aldosterone):
1-sodium and water retention by kidneys
2-increased blood volume and blood pressure

Glucocorticoids / fasciculata :
1- increased blood sugar
2- immune system suppression
3- proteins and fats conversion to glucose and breaking down for energy.

*pics

Disorders of the adrenal glands

a- disorders due to abnormal anatomy (mass effect). Adrenal glands are retroperitoneal and they are in a big space so mostly the presentation is not due to mass effect

b- disorders due to abnormal physiology (hypo or hyper secretion of hormones).

Neoplasms arising from cortex and mets:

- adenoma
- carcinoma
- myelolipoma
- metastatic carcinoma

**cortical adenoma

- unpredictable endocrine function(silent – no hyperfunction- , hypercortisolism , hyperaldosteronism).
- Solitary nodule
- Much smaller than cortical carcinoma <50 gm.
- The color: yellow (majorly bcz of lipid laden cells) black (minority / excess lipofuscin pigment).
- CT characteristics : sharp margins , smooth homogenous , lipid rich , wash out > 50% @ 15 mins & <10 Hu on noncontrast images.

** secreting tumors (functional):

1- Hyperaldosteronism; primary hyperaldosteronism (conn's syndrome)

Quick revision aldosterone function: Aldosterone hormone increase sodium reabsorption so increase sodium amount in your blood stream and also it increases the amount of potassium released in the urine so increases blood volume.

Primary hyperaldosteronism is an adrenal problem independent on juxtaglomerular apparatus(low renin , high aldosterone). Secondary hyperaldosteronism is a normal response of low renal artery pressure .

conn's syndrome :

1- solitary adrenal adenoma(90%).
2- Bilateral adrenal hyperplasia(20%).
3- Adrenal carcinoma (rare).
4- Unilateral adrenal hyperplasia(very rare).

Clinical features of Conn's syndrome

Often asymptomatic if symptomatic:
(Frontal headache, Muscle weakness to flaccid paralysis
decreased muscle strength (because of low potassium level), Polyuria and Polydipsia
and Hypertension).

Investigation of Conn's syndrome

A. Serum Electrolytes

1. Serum Potassium decreased
2. Serum Sodium increased (Mild)
3. Metabolic Alkalosis

B. Increased serum level of aldosterone

C. Greatly decreased serum level of renin

D. Sodium loading test(by giving 2 L saline if aldosterone decreased then it is not conns)

Localization

Ultrasound , CT scan(the best) ,MRI and angiographic blood sampling(right or left).

Treatment of conn's disease

A. Adrenal Adenoma

Surgical excision (unilateral adrenalectomy)

B. Adrenal Hyperplasia

Spironolactone (Aldactone)

2- Hypercortisolism ;cushing's syndrome :

a- Endogenous :

1- ACTH dependent 85%

- Cushing's disease(pituitary)
- Ectopic ACTH syndrome (small cell lung carcinoma)
- Ectopic CRH syndrome (bronchial carcinoid tumours)

2- ACTH independent 15%

- Adrenal adenoma
- Adrenal carcinoma
- Less common : micronodular hyperplasia , macronodular hyperplasia .

b- Exogenous : glucocorticoid treatment

**pseudo-cushing's syndrome : Major depressive disorder, alcoholism, obesity.

Investigation of Cushing's syndrome
Screening tests

1- Urinary free cortisol; The measurement of 24 hour excretion of cortisol in urine integrates the variations in plasma free cortisol concentrations seen during the entire day.

2- Dexamethasone suppressive tests ; In normal subjects, administration of a supraphysiological dose of glucocorticoid results in suppression of ACTH and hence of cortisol secretion (cortisol <50 nmol/l). This is the basis of dexamethasone suppression tests, of which there are several types.

Treatment of Cushing's syndrome
Transsphenoidal surgery(pituitary tumor removal process) , Pituitary radio-therapy, Bilateral or unilateral adrenalectomy,Medical therapy(mitotane +chemo).

3- Hyperandrogenism (precocious puberty)
4- Pheochromocytoma
Rule's of 10%:
- Bilateral (90% unilateral)
- Familial (non-sporadic)
- Pediatric

• Malignant
• Normotensive mnemonics;
• Extra-adrenal
• Multiple
• *Childhood presentation breaks the rules- 25% bilateral, multiple, extra-adrenal

pheo manifestations :

triad headache , tachycardia , diaphoresis(excessive sweating).(هدت)
Other symptoms ; pallor , flushing , palpitations , abd/chest pain, weakness , N/V , psychosis.

Small tumors are more likely symptomatic .

The Diagnosis of pheo :

- Plasma free metanephrines(epinephrine metabolite) : most sensitive test
- 24 hour urinary catecholamines (2x normal is diagnostic)
- VMA
- Clonidine suppression test >50% reduction of catecholamines no pheo .

Pre-op Management

1- Early alpha blockad
2- Do NOT use b-blocker before alpha not to decrease CO while vascular resistance is still high.
3- control hypertension- phenoxybenzamine

4- IV hydration
5- Prevent cardiac arrhythmias

** give adrenaline after gland removal till adaptation

Adrenal cortical carcinoma :

Mean age 50 years
Larger: typically > 100 gm
#Gross clues for malignancy (some apparent by imaging):Large size, hemorrhage, necrosis, invasion of adrenal vein.
#Absolute criteria for malignancy: invasion & metastasis
#5 year survival: 20-30%;most tumor-related deaths occur within first two years after diagnosis.

The adrenal "Incidentaloma":

What is it? *"an adrenal mass, generally more than 1 cm. diameter, that is discovered accidentally during a radiologic examination performed for indications other than an evaluation for adrenal disease."*
Nature of incidental found adrenal mass*:* Nonfunctioning adenoma 82%, Functioning: Cushing's 5%, Pheo 5% , Aldosteronoma 1%,Malignancy: Metastasis 3%, ACC 4%

Incidental adrenal mass management:
Indications of surgery

1- Symptomatic hormonally active surgical removal
2- The mass is > 5 cm surgical removal

Observe if the mass < 3cm :

- Old mass : CT at 6 months then annual for 4 years .
- New mass : at 3 months then 1 year and then no further workup.

Refer to surgical approach slides I have not written any thing about these.