
بسم الله الرحمن الرحيم
In the name of ALLAH, the Most Gracious, the Most Merciful

Development of Adrenal Glands

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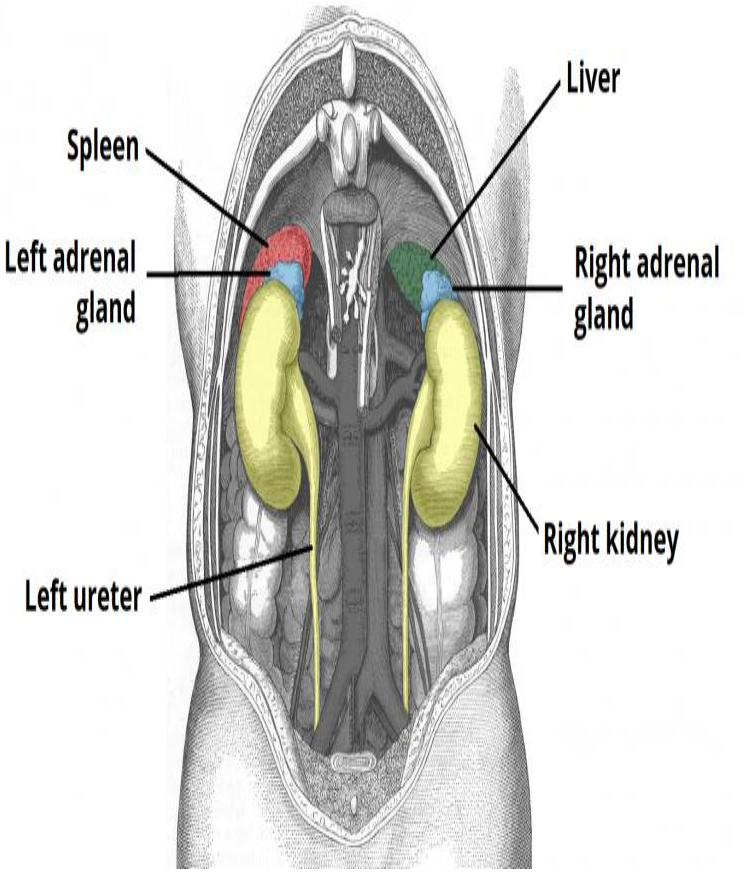
Learning Objectives :

- Introduction
- Anatomy
- Development of gland
- Anomalies

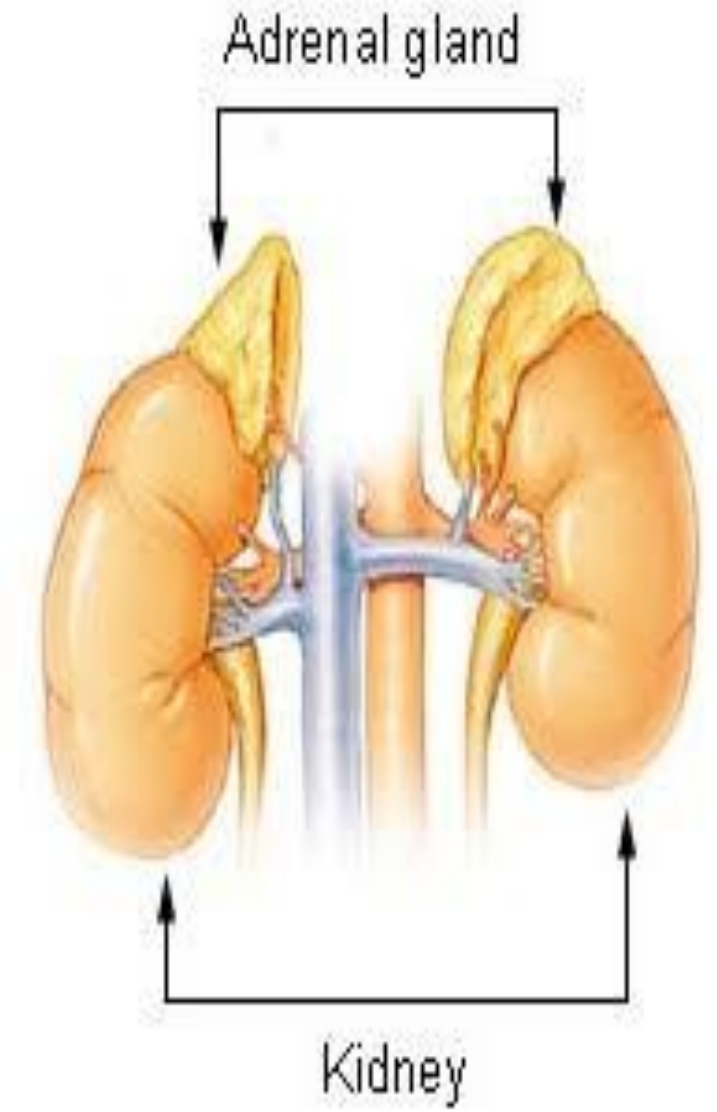
Anatomy

- Weight - 4 gm.
- The adrenal (or suprarenal) glands are paired endocrine glands situated over the medial aspect of the upper poles of each kidney
- They are retroperitoneal, with parietal peritoneum covering their anterior surface only



- The right gland is pyramidal in shape, contrasting with the semi-lunar shape of the left gland.
- Each Gland consists of 2 parts

A. Cortex
B. Medulla



Development Of Adrenal Gland :

- The cortex and medulla of adrenal glands have different origins .
- The **Cortex** develops from **mesenchyme (**coelomic epithelium)**.**
- The **Medulla** develops from **Neural crest cells**

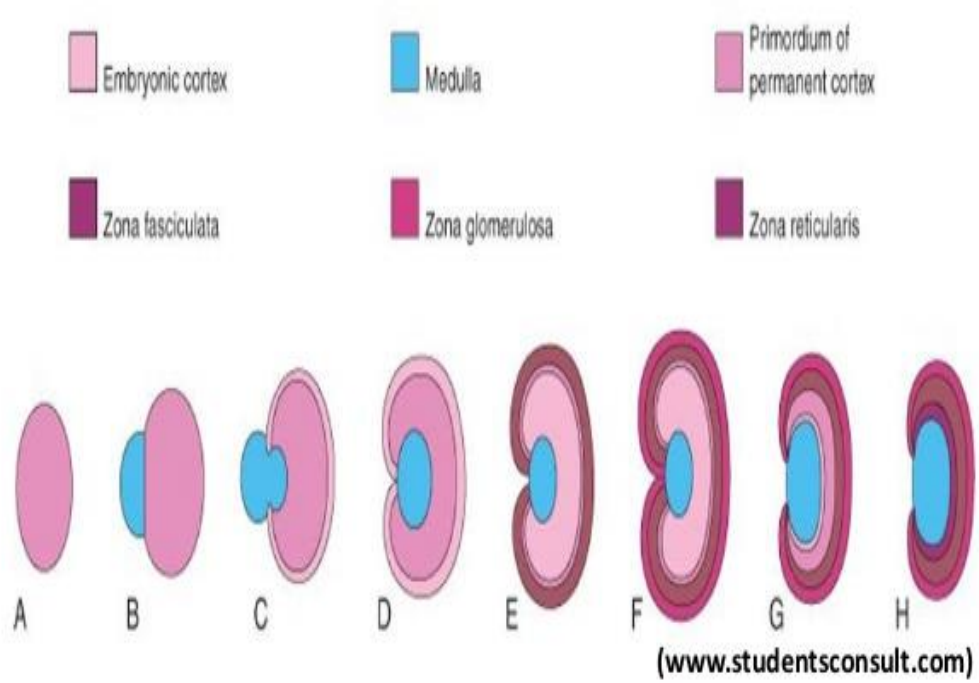
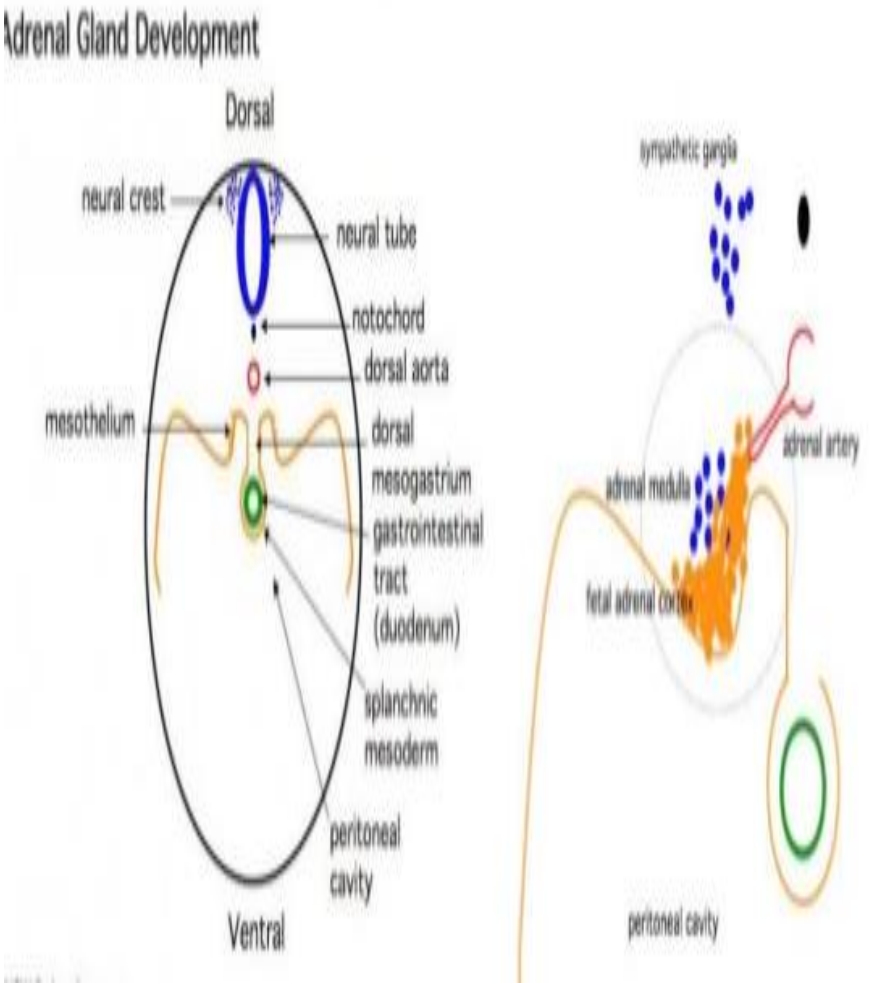


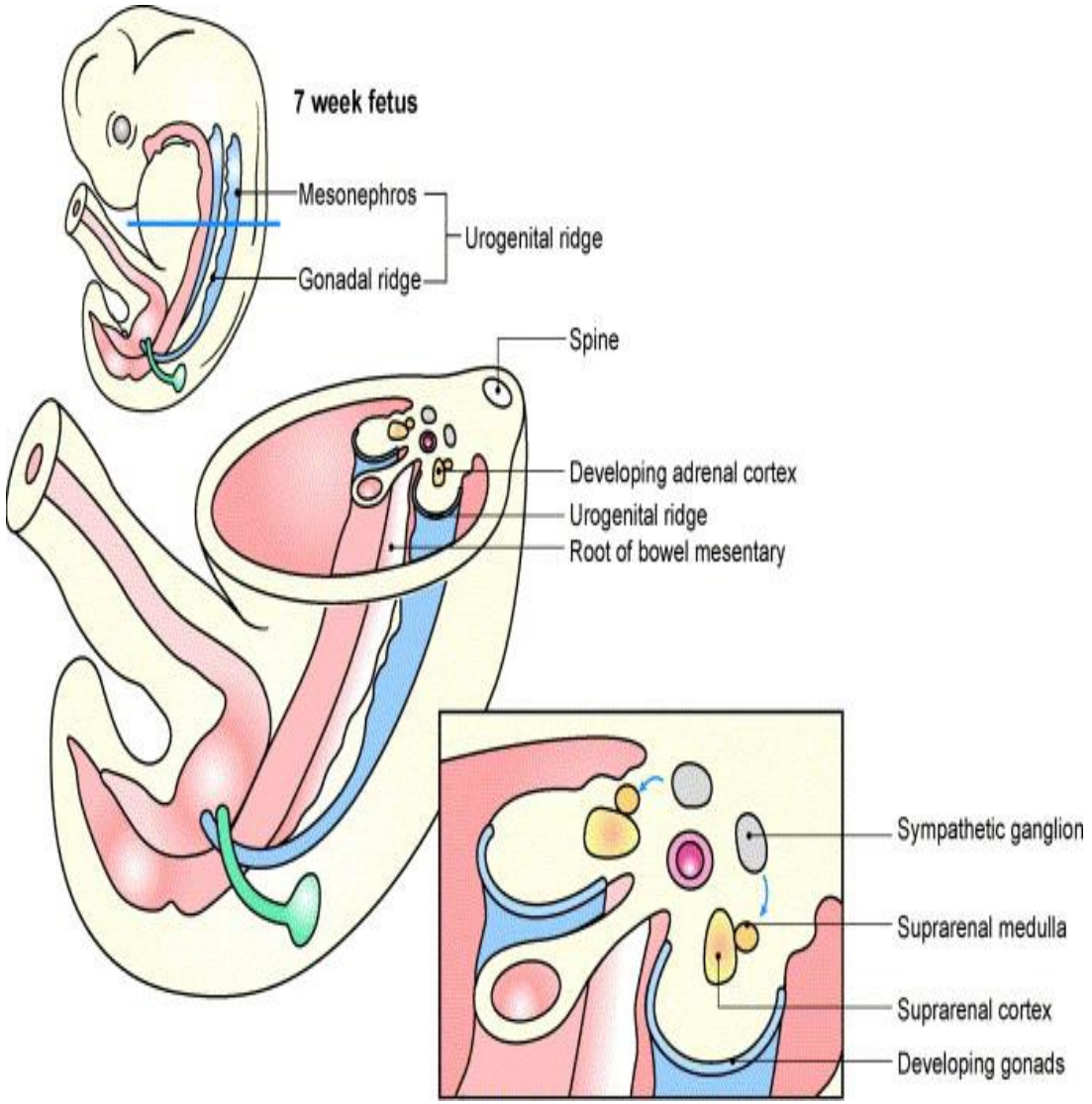
Fig. 15: Schematic drawings illustrating development of the suprarenal glands. A, At 6 weeks, showing the mesodermal primordium of the fetal cortex. B, At 7 weeks, showing the addition of neural crest cells. C, At 8 weeks, showing the fetal cortex and the early permanent cortex beginning to encapsulate the medulla. D and E, Later stages of encapsulation of the medulla by the cortex. F, Newborn infant showing the fetal cortex and two zones of the permanent cortex. G, At 1 year, the fetal cortex has almost disappeared. H, At 4 years, showing the adult pattern of cortical zones. Note that the fetal cortex has disappeared and that the gland is much smaller than it was at birth (F).

- During 6th week , the cortex begins as an aggregation of mesenchymal cells on each side of embryo between the root of dorsal mesentery and the developing gonad.

- The cells that form medulla are derived from an adjacent sympathetic ganglion , which is derived from neural crest cells



- Initially neural crest cells form a mass on medial side of embryonic cortex. As they are surrounded by cortex , the cells differentiate into secretory cells of suprarenal medulla .
- Later more mesenchymal cells arise from the mesothelium and enclose the cortex . These cells give rise to permanent cortex of suprarenal gland

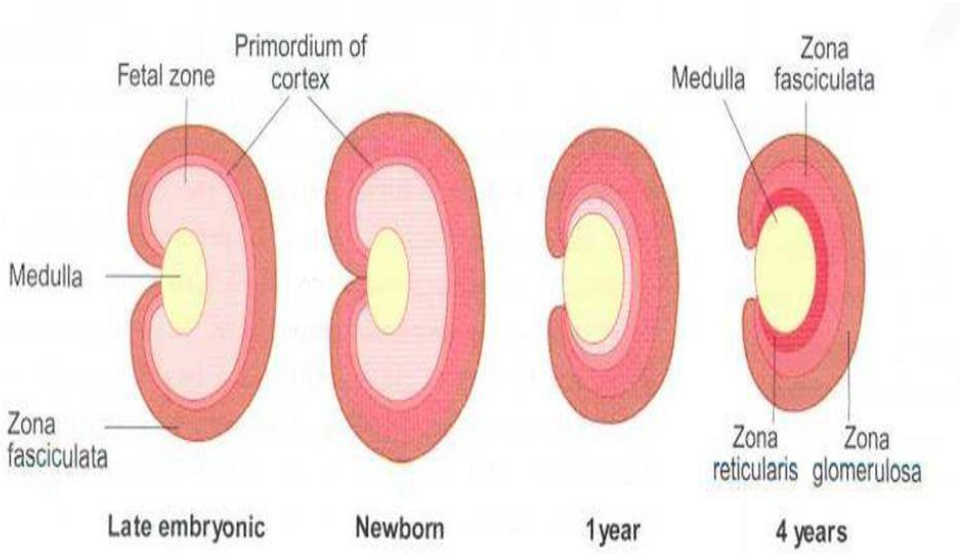


- Immunohistochemical studies identify a “ transitional zone’ that is located between the permanent cortex and the fetal cortex.

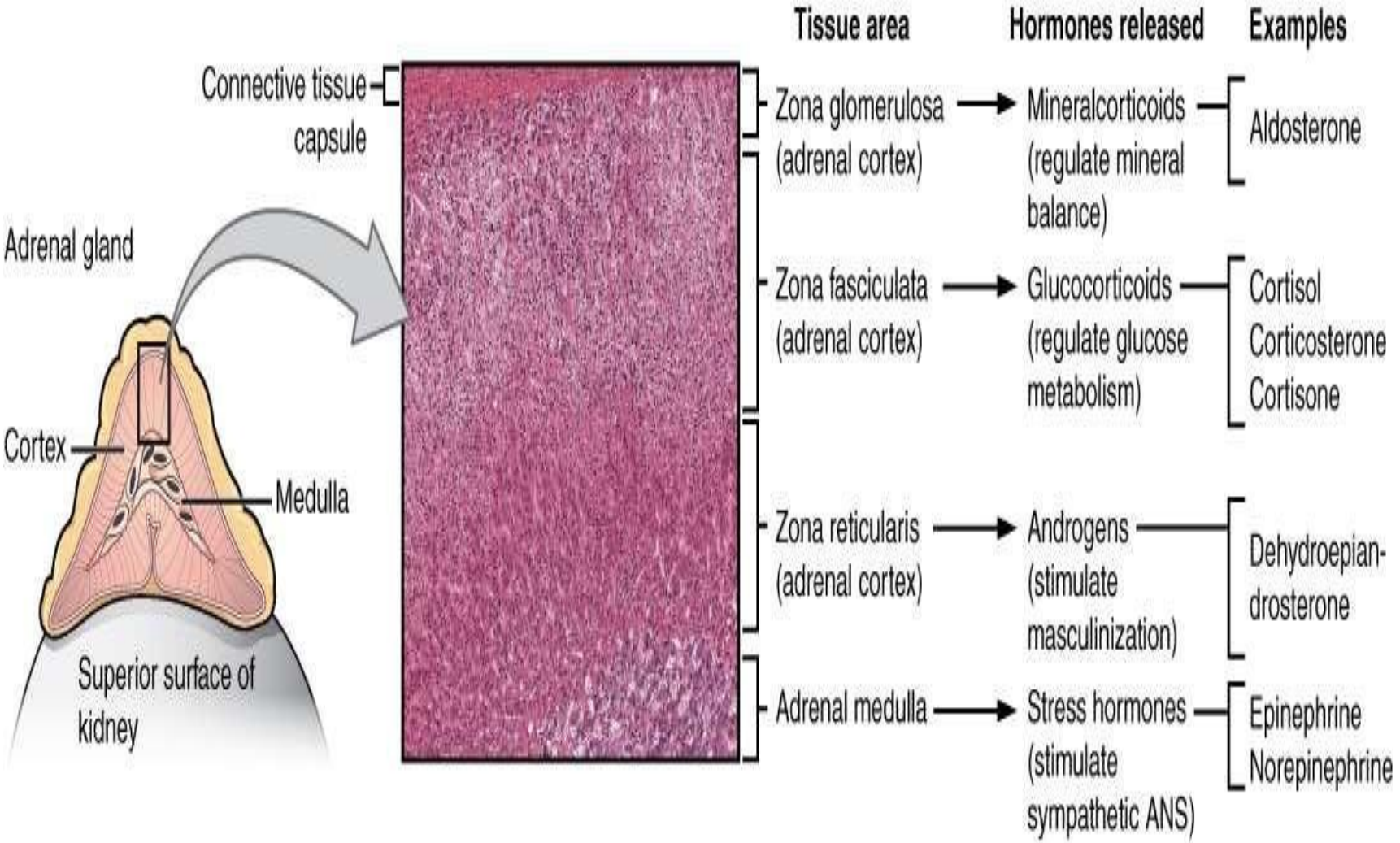
- The *zona Glomerulosa* and *zona fasiculata* are present at birth , but *zona reticularis* is not recognizable until end of 3rd year

- Relative to body weight fetal suprarenal glands are 10 - 20 times larger than in adults and are large compared with kidneys

Schematic diagram showing the changes in the adrenal gland during development.



FUNCTION


Tissue area
Hormones released
Examples
Connective tissue capsule
Adrenal gland
Cortex
Medulla
Superior surface of kidney
Zona glomerulosa (adrenal cortex)
Mineralcorticoids (regulate mineral balance)
Aldosterone
Zona fasciculata (adrenal cortex)
Glucocorticoids (regulate glucose metabolism)
Cortisol
Corticosterone
Cortisone
Zona reticularis (adrenal cortex)
Androgens (stimulate masculinization)
Dehydroepian-drosterone
Adrenal medulla
Stress hormones (stimulate sympathetic ANS)
Epinephrine
Norepinephrine

Clinical Correlations

1. Congenital Adrenal Hyperplasia and Adrenogenital Syndrome :

- **An abnormal increase in cells of suprarenal cortex results in excessive production during fetal period.**
- **In Females it causes masculinization of the external genitalia**
- The adrenogenital syndrome associated with CAH manifest itself in various forms that can b correlated with enzymatic deficiencies of cortisol biosynthesis.

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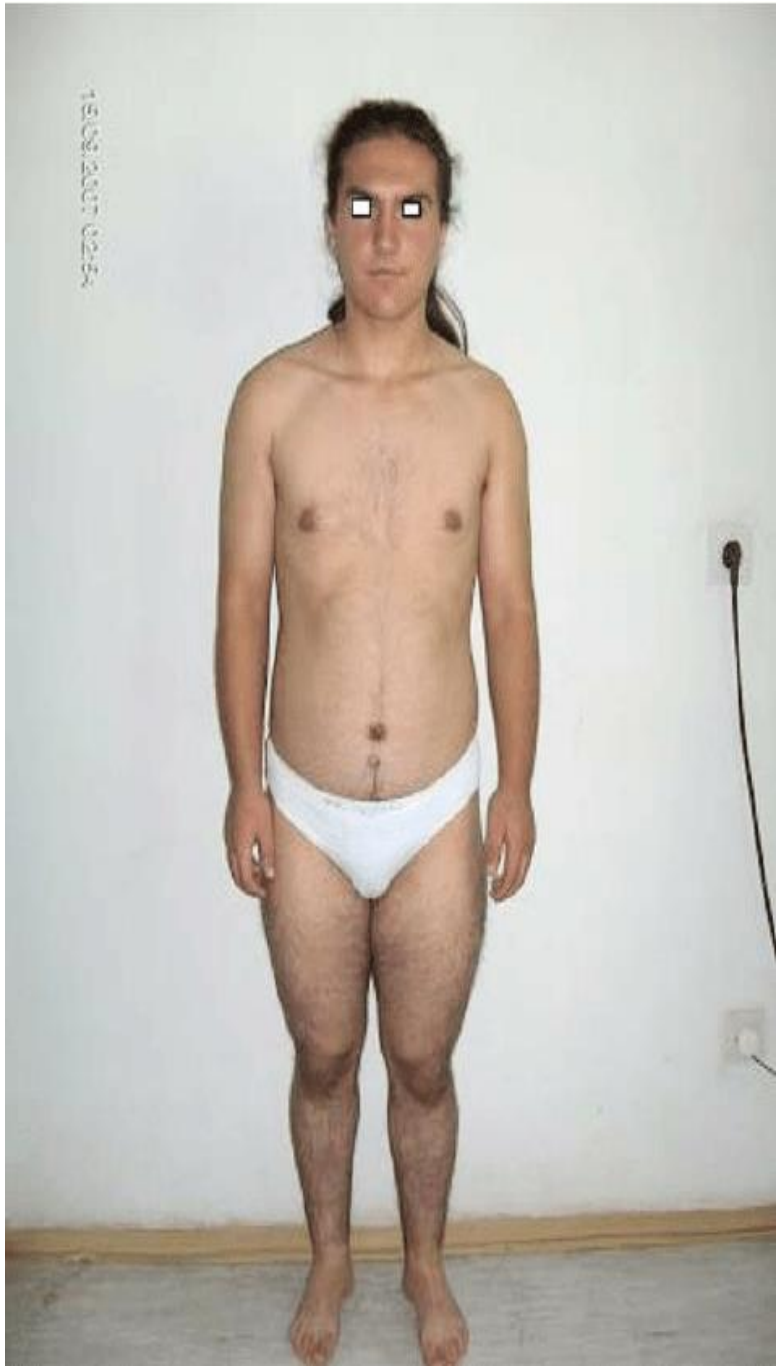


2. Adrenal cortical tissue in ectopic sites

- Deep to the capsule of the kidney
- Fused to liver or/ kidney

Adrenal Hypofunction

- **Causes**
  - Glucocorticoid treatment
  - Autoimmune adrenalitis
  - Tuberculosis
  - Adrenalectomy
  - Secondary tumor deposits
  - Amyloidosis
  - Haemochromatosis
  - Histoplasmosis, tuberculosis, CMV, AIDS
  - adrenal haemorrhage

Common

Adrenal Hypofunction

- **Other causes**
  - ACTH blocking antibodies
  - Mutation in ACTH receptor gene
  - Adrenal hypoplasia congenita
  - Familial adrenal insufficiency

Addison disease

- Autoimmune
- Isolated or associated with other autoimmune disease
- Presents with tiredness, weight loss, skin pigmentation
- Aldestrone & cortisol low, high ACTH, high renin
- Low sodium , high potasium
- ACTH stimulation test
- Adrenal antibodies
- Treatment : cortisol + aldestrone

Adrenal Hypofunction

Addison's disease Primary hypoaldosteronism

Addison's disease:

- pathogenesis

Progressive destruction of entire adrenal cortex ,

- This is usually **autoimmune** based.

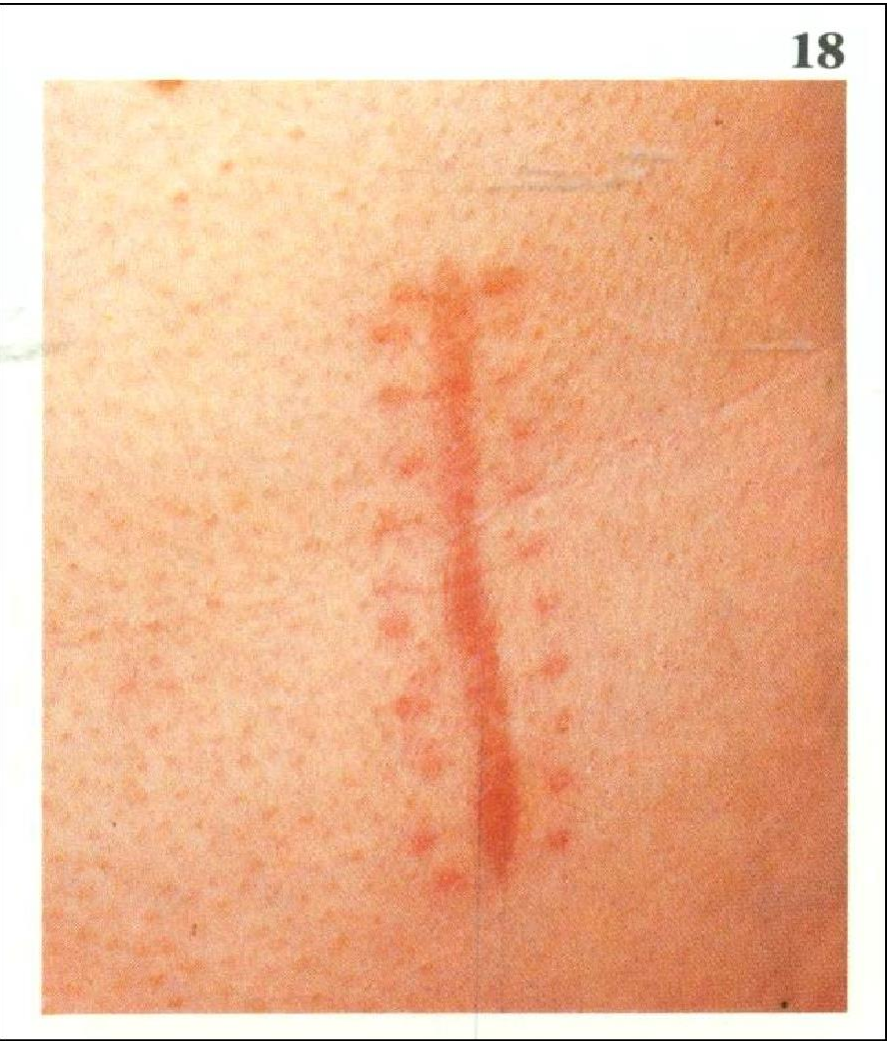
Most likely the result of **cytotoxic T lymphocytes**, although 50% of patients have circulating adrenal **antibodies**.

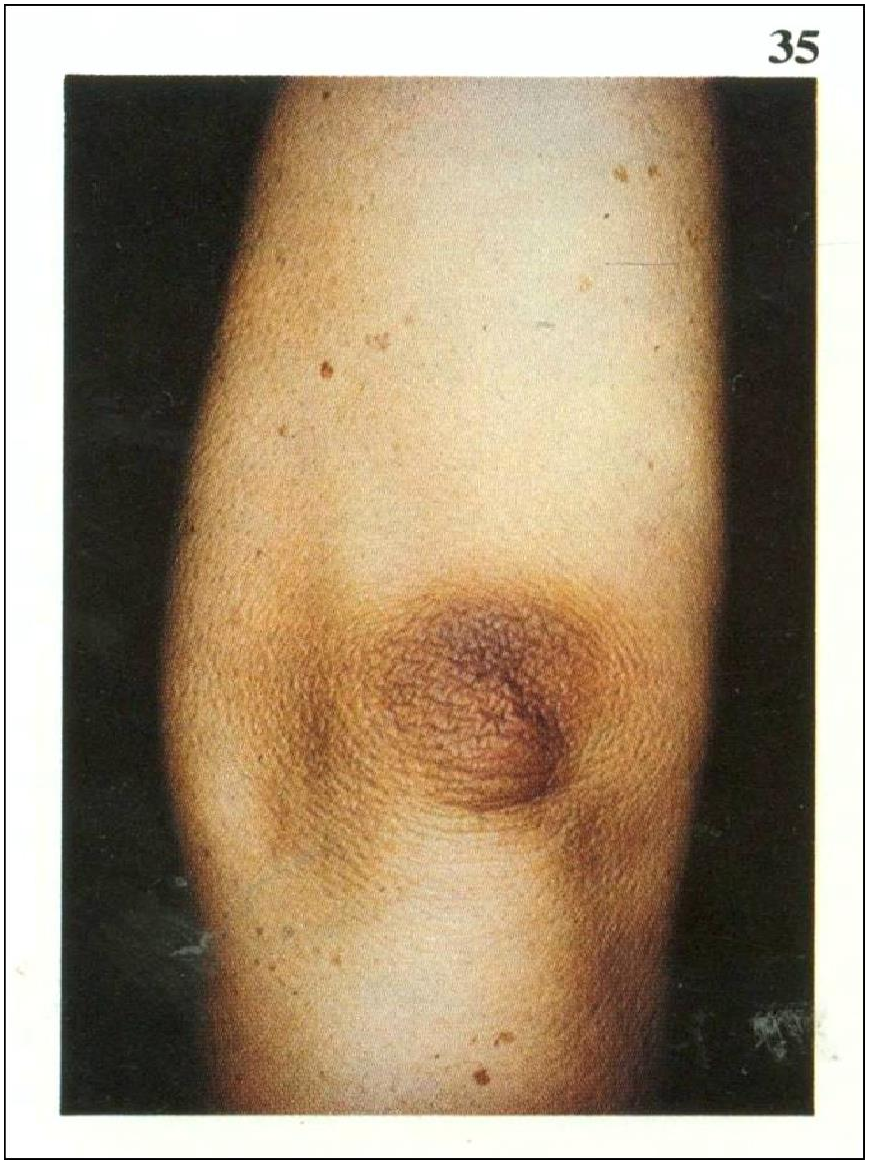
Addison's disease: Clinical features

Common	Less common
Tiredness, generalized weakness, lethargy	Hypoglycemia
Anorexia, nausea, vomiting	Depression
Hyponatremia Hyperkalemia ,Hypercalcemia	Convulsions
Dizziness and postural hypotension	
Pigmentation	
Loss of body hair (woman)	

Addison's disease: clinical features

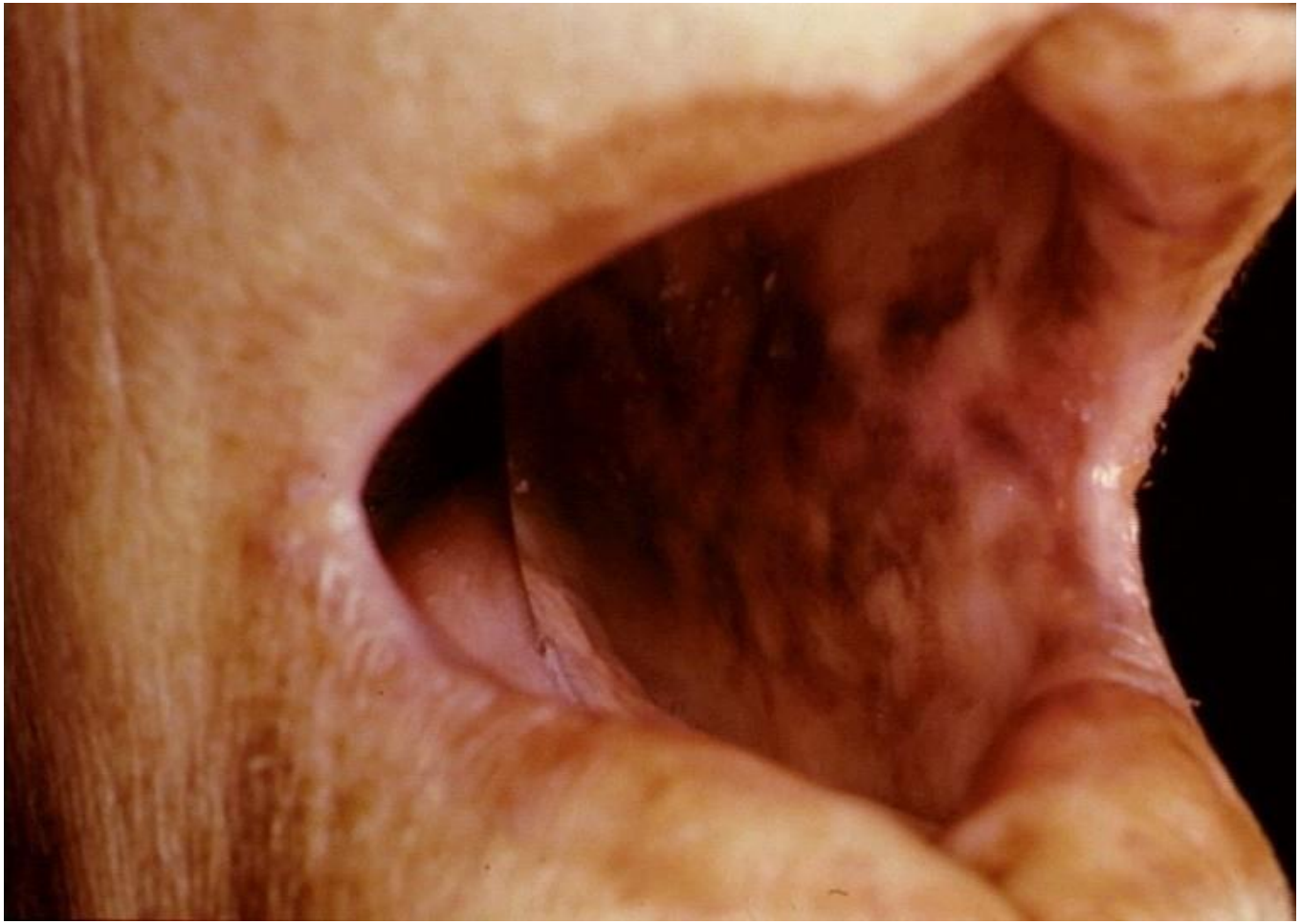
- hyperpigmentation





Addison's disease: clinical features

- Hyperpigmentation



ADRENAL CRISIS

- **Acute adrenal insufficiency**
- Medical emergency
- Acute in onset; can be fatal if not promptly recognized and treated
- **Clinical features** :
  - Severe hypovolaemia
  - Dehydration
  - Shock
  - Hypoglycaemia
  - possible mental confusion and loss of consciousness

ADRENAL CRISIS

- **Causes :**
  - Precipitated by stress
    - infection, trauma or surgery in patients with incipient adrenal failure/treated with glucocorticoids if dosage is not increase
  - Adrenal haemorrhage
    - due to cx of anticoagulant treatment
  - Meningococcal septicaemia

Disorders of adrenal cortex

ADRENAL HYPERFUNCTION

Adrenal Dysfunction

Increase function

- Cushing syndrome

  High Cortisol

- Hyperaldosteronism

  High aldestrone

- Pheochromocytoma

  High catecholamine

Hyperaldosteronism

- A medical condition where too much aldosterone is produced by the adrenal glands, which can lead to sodium retention and potassium loss.

- Types:
  - Primary hyperaldosteronism
  - Secondary hyperaldosteronism

Primary hyperaldosteronism (hyporeninemic hyperaldosteronism)

Conn’s syndrome

Primary aldosteronism

CONN'S SYNDROME

- Characterized by **autonomous** excessive production of **aldosterone** by **adrenal glands**

- Presents with HPT, hypokalaemic alkalosis and renal K+ wasting

Conn's Syndrome

- Causes:
  - Adrenal adenoma
  - Bilateral hypertrophy of zona glomerulosa cells
  - Adrenal carcinoma
    - Rare cause

Conn's syndrome

- **Clinical features:**
  - Hypertension : *aldosterone induced Na retention with increase in ECF volume*
  - Muscle weakness: *Due to decrease K+*
  - Muscle paralysis: *severe hypokalaemia*
  - Latent tetany and paraesthesiae
  - Polydipsia, polyuria and nocturia: *due to hypokalaemic nephropathy*

Disorders of adrenal cortex

ADRENAL HYPERFUNCTION

CUSHING'S SYNDROME

- Definition
- Clinical features
- Investigations
  - Screening for Cushing's syndrome
  - Elucidation of the cause of Cushing's syndrome

- Management

CUSHING'S SYNDROME

Adrenal cortex hyperfunction

- Any condition resulting from overproduction of **primarily glucocorticoid (cortisol)**

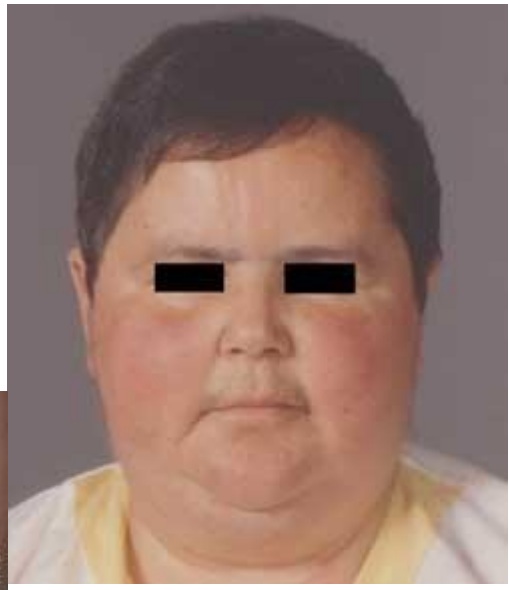
- Mineralocorticoid and androgen may also be excessive

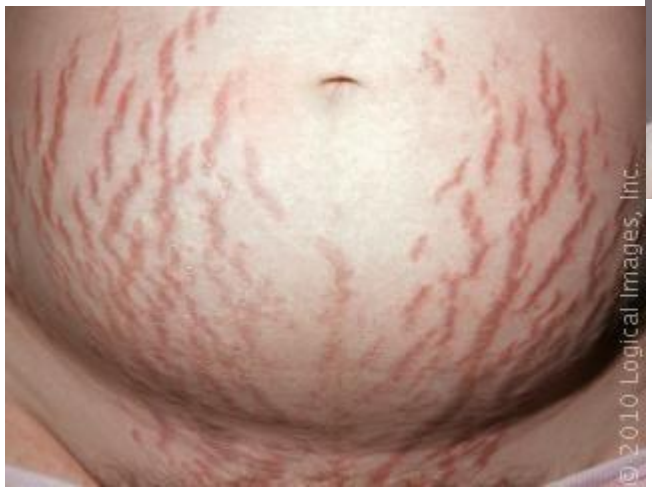
Etiology

- **Excess cortisol binding globulin**
  - Estrogen therapy : Osteoporosis, OCP
  - Pregnancy

Clinical features

- **Truncal obesity** with deposition of adipose tissue in characteristic site (moon face, buffalo hump)– exact mechanism unknown
- **Thinning of skin** – catabolic response
- **Purple striae** – catabolic response
- **Excessive bruising** – catabolic response





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- **Hirsutism** (esp adrenal carcinoma) - ↑ adrenal androgen
- **Menstrual irregularities** - ↑ adrenal androgen

- **Skin pigmentation** (ACTH ↑) – melanocyte stimulating activity

Cont..

- **Hypertension** – mineralocorticoid effect → sodium retention

– Potassium wasting → hypokalamic alkalosis

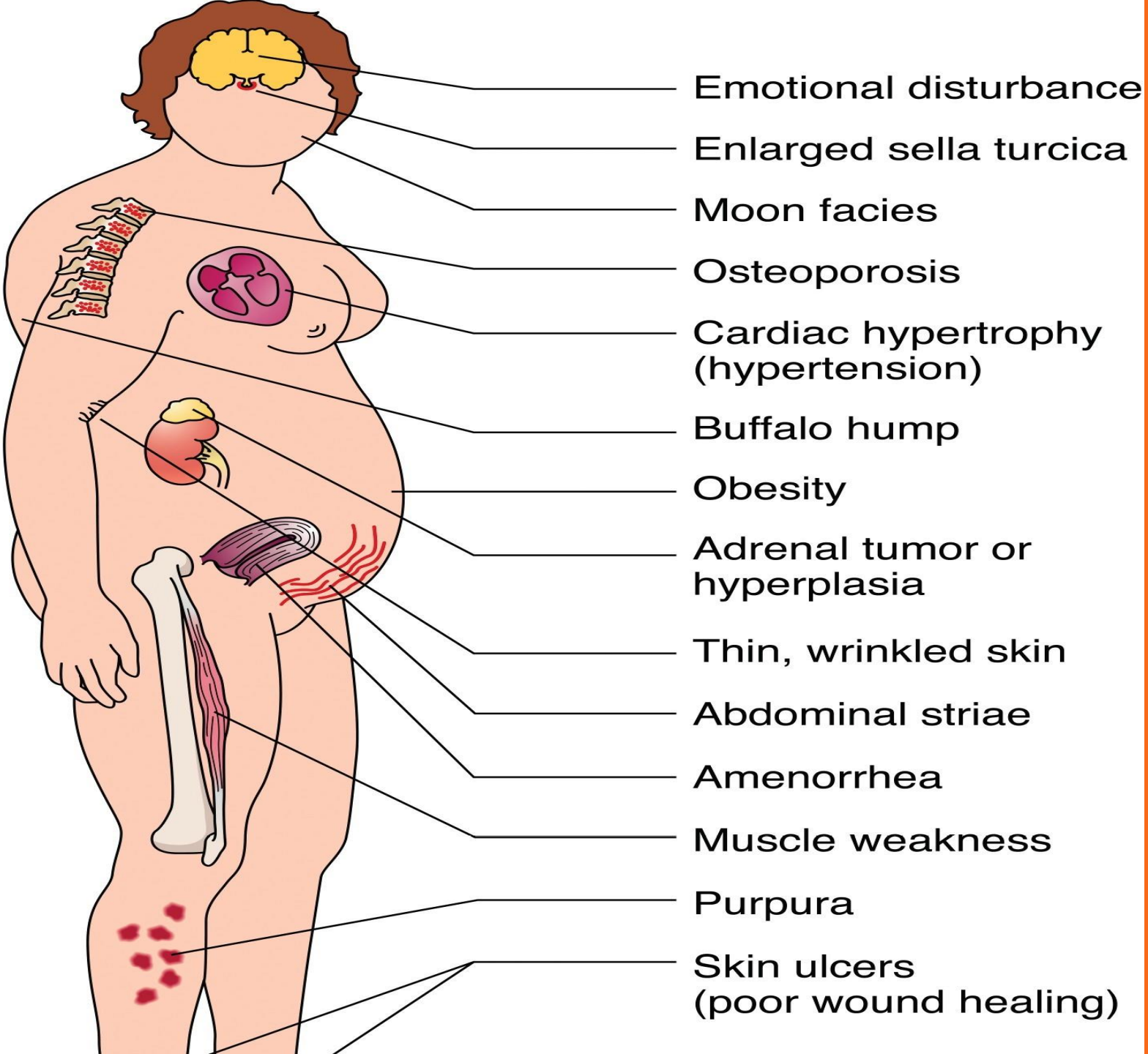
- **Glucose intolerance -** ↑ hepatic gluconeogenesis and insulin resistance

- **Muscle weakness and wasting** – catabolic response in peripheral supportive tissue

Cont..

- **Back pain** (osteoporosis and vertebral collapse) – inhibit bone formation

- **Psychiatric disturbances** – euphoria, mania, depression


Emotional disturbance
Enlarged sella turcica
Moon facies
Osteoporosis
Cardiac hypertrophy (hypertension)
Buffalo hump
Obesity
Adrenal tumor or hyperplasia
Thin, wrinkled skin
Abdominal striae
Amenorrhea
Muscle weakness
Purpura
Skin ulcers (poor wound healing)

Treatment

- Depend of Cushing's syndrome depends on the etiology:
  - Adrenal adenoma
  - Adrenal Carcinoma – resection
  - Cushing’s disease - transphenoidal hyposectomy
  - Drug (block cortisol synthesis) - metyrapone


THANK
YOU!