Polycystic ovarian Syndrome

It hormonal disorder
- Common among women of Reproductive age
- They have infrequent or prolonged Menstrual periods.
- Excess of male hormones i.e androgens

Here the ovaries develop small collection of follicles ~~[illegible]~~ follicles & fail to release eggs

This disorder characterized by oligomenorrhea / Amenorrhea with symptoms of Hyperandrogenism like acne, hirsutism (Extra male like hair growth)

Etiology: 1. Excess insulin: ~~[illegible]~~ when cell becomes Insulin resistant
blood sugar rises
↓
Pancreas produce more Insulin
↓
Produces more androgens & interfere ⊂ ovulation

2. Heredity: Certain genes might be linked to PCOS.

3. Excess of androgens produced abnormally ⇐ high levels resulting Hirsutism and acne.

(2)

Signs & symptoms : 1. Irregular periods.
Excess of facial & bodyhair
2. i.e → Hirsutism
3. Polycystic ovaries :
4. Obesity

Pathogenesis :

PCOS have More frequent release of LH.
Frequency of GnRH be accelerated in PCOS
Bcoz of that More generation and Releasing
LH over FSH
Conc of LH ↑ Es relative to FSH
So ovaries synthesizing testosterone
Higher levels of free testosterone ↑ bioavailability
of hormone & further inhibits the hepatic
synthesis of - SHBG
Sex hormone binding Globulin

↑ Se in free Total testosterone levels
↓ Results
androgenization → features of Hirsutism
acne, anovulation

where there will be absence of dominant follicle
Prevents Development of Non dominant follicles.
resulting in formation of Multiple ovarian cysts.

Hirsutism - Excessive hair growth — Male pattern
Unwanted → Face Chest, Abd & back.

Due to ↑ androgens.
few disorders like Cong adrenal Hyperplasia
ovarian tumors
Adrenal tumors.

Blood tests → Determine the cause
Medical treatment varies depending upon the cause of hirsutism.

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Hypogonadism → Male disorder

Condition in which body does not produce enough testosterone

Testosterone plays a key role in Masculine growth & development during puberty.

Decreased production of Testosterone

Either from pituitary – Not producing stimulating Hormone

or

from Testes to produce adequate or failure to produce.

→ Primary : Hypogonadism

Known as primary testicular failure originated from a problem in Testes

→ Secondary → Problem in Hypothalamus or pituitary

Hypothalamus.

↓

GnRH : Signals pituitary to release FSH & LH

↓

Signals testes to produce testosterone

Etiology :

Primary : Autoimmune disorders like
Addison's Disease
Hypoparathyroidism
Severe Infections
Liver & Kidney disease
Radiation Exposure
Surgery on ovaries or Testes

Secondary :
Genetic disorders like Kallmann syndrome (Abnormal Hypothalamic development)
Infection like HIV & AIDS
Inflammatory Diseases like TB, Sarcoidosis
Pituitary disorder.
Use of Steroids (Long term usage)

Signs & Symptoms :
- Characterized by Serum testosterone < 300 ng/dl
- & atleast one sign or symptom

Sign : Absence or Regression of secondary sex characteristics

- Anemia
- Muscle wasting
- Reduced bone mass / bone mineral density
- Oligospermia
- Abdominal adiposity.

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Symptoms : Sexual dysfunction
Reduced energy / stamina
Depressed mood
↑ed irritability
Osteoporosis

In prepubertal males → sparse body hair
delayed Epiphyseal closure

Pathophysiology = Hypothalamus
↓ Releases
GnRH in pulsatile fashion.
↓ stimulates
Pituitary gland
↓ produces
FSH & LH
↓ triggers the activity of
Leydig cells in testes
so cholesterol is converted to Testosterone
↓
when Testosterone are sufficient slows pituitary
to release LH via −ve feed back
thereby slowing of Testerone production

Any change in testis, Hypothalamus or pituitary result to hypogonadism.