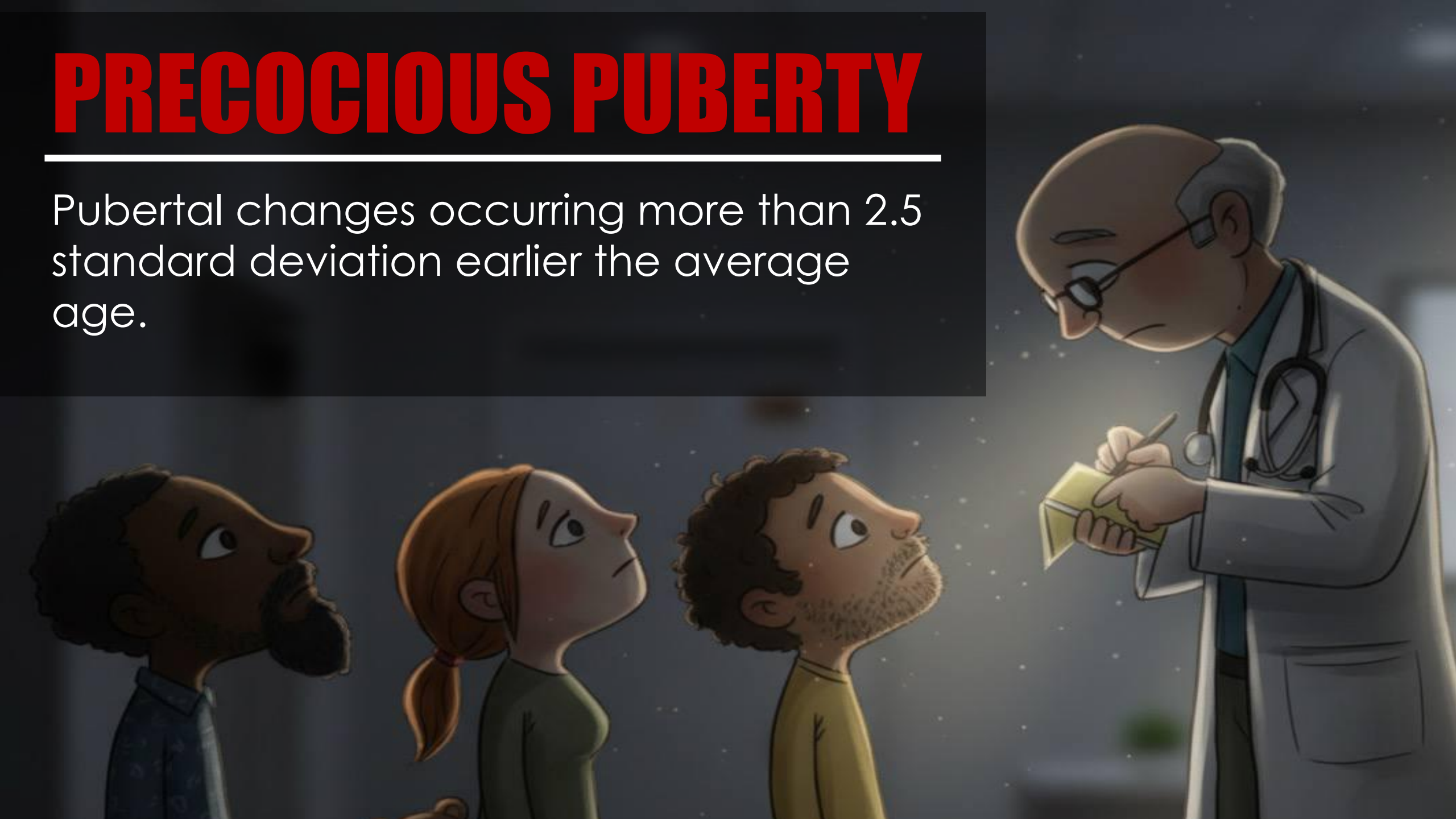
PRECOCIOUS PUBERTY

MUFSILA PARVEEN
VISUDHA V R

PRECOCIOUS PUBERTY

Pubertal changes occurring more than 2.5 standard deviation earlier the average age.



PRECOCIOUS PUBERTY

Pubertal changes before 8 years or menarche before 9 years can be considered precocious.

WHY IS IT IMPORTANT?

- Main parental concern: early menarche.

WHY IS IT IMPORTANT?

Physician's concern:

- Rule out underlying pathology.
- Assess growth acceleration and psychological impact.
- Most cases (especially in girls) are idiopathic.

CLASSIFICATION

Central (True) / Gonadotrophin-Dependent Precocious Puberty (CPP)

Peripheral / Pseudoprecocious / Gonadotrophin-Independent Precocious Puberty (PPP)

Incomplete / Partial Precocious Puberty

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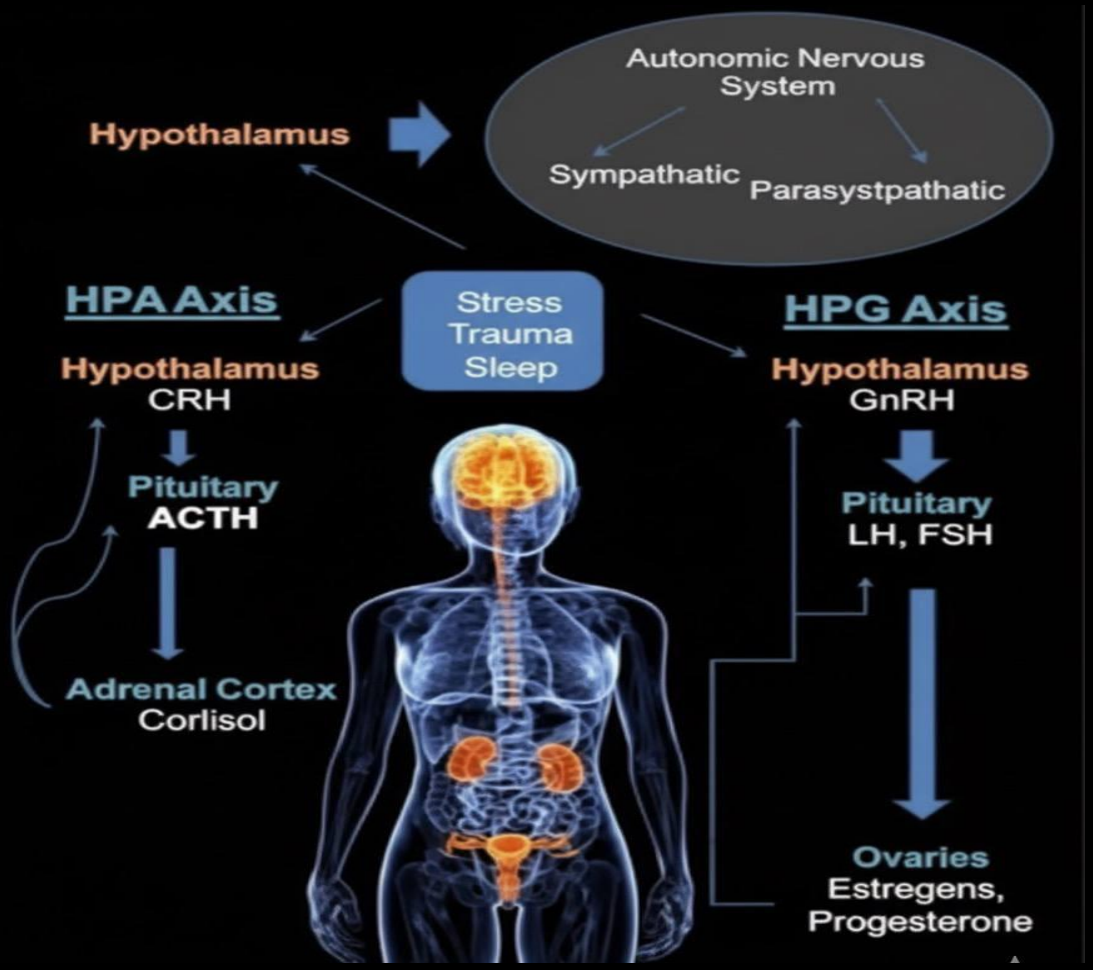
CENTRAL/ GONADOTROPHIN DEPENDENT

PRECOCIOUS PUBERTY

CPP

- Due to premature activation of hypothalamic–pituitary–gonadal (HPG) axis.
- Isosexual sexual characters
- More common in girls



FEATURES

- Sexual characteristics appear in normal order (thelarche → pubarche → menarche).
- Cyclical vaginal bleeding
- Breast enlargement & pubic hair

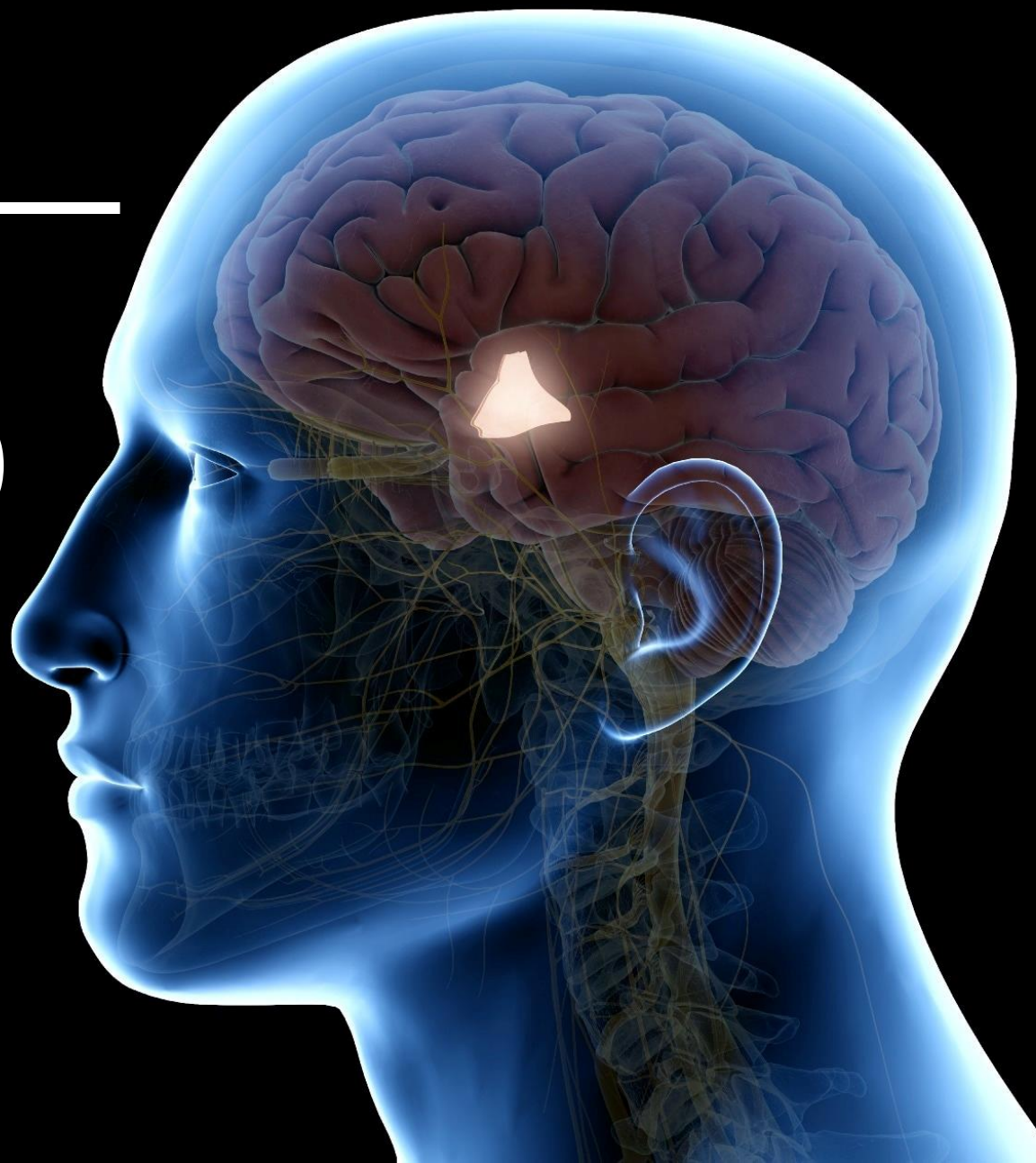
CAUSES

- Idiopathic: ~90% of cases.
- Genetic mutations: Kisspeptin gene or its receptor.

CAUSES

CNS lesions:

- Hypothalamic hamartoma (most common organic cause)

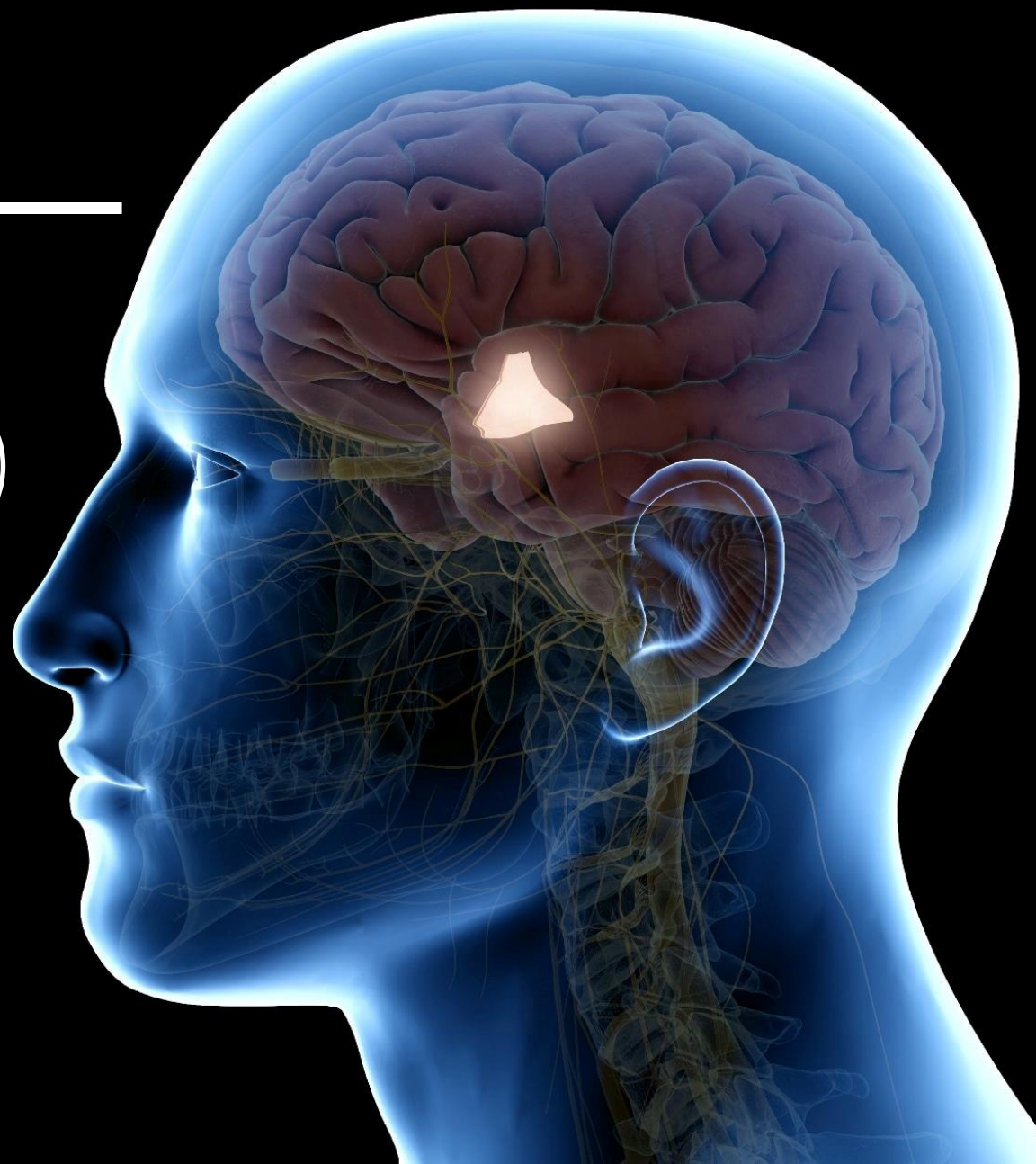


CAUSES

CNS lesions:

- Hypothalamic hamartoma (most common organic cause)

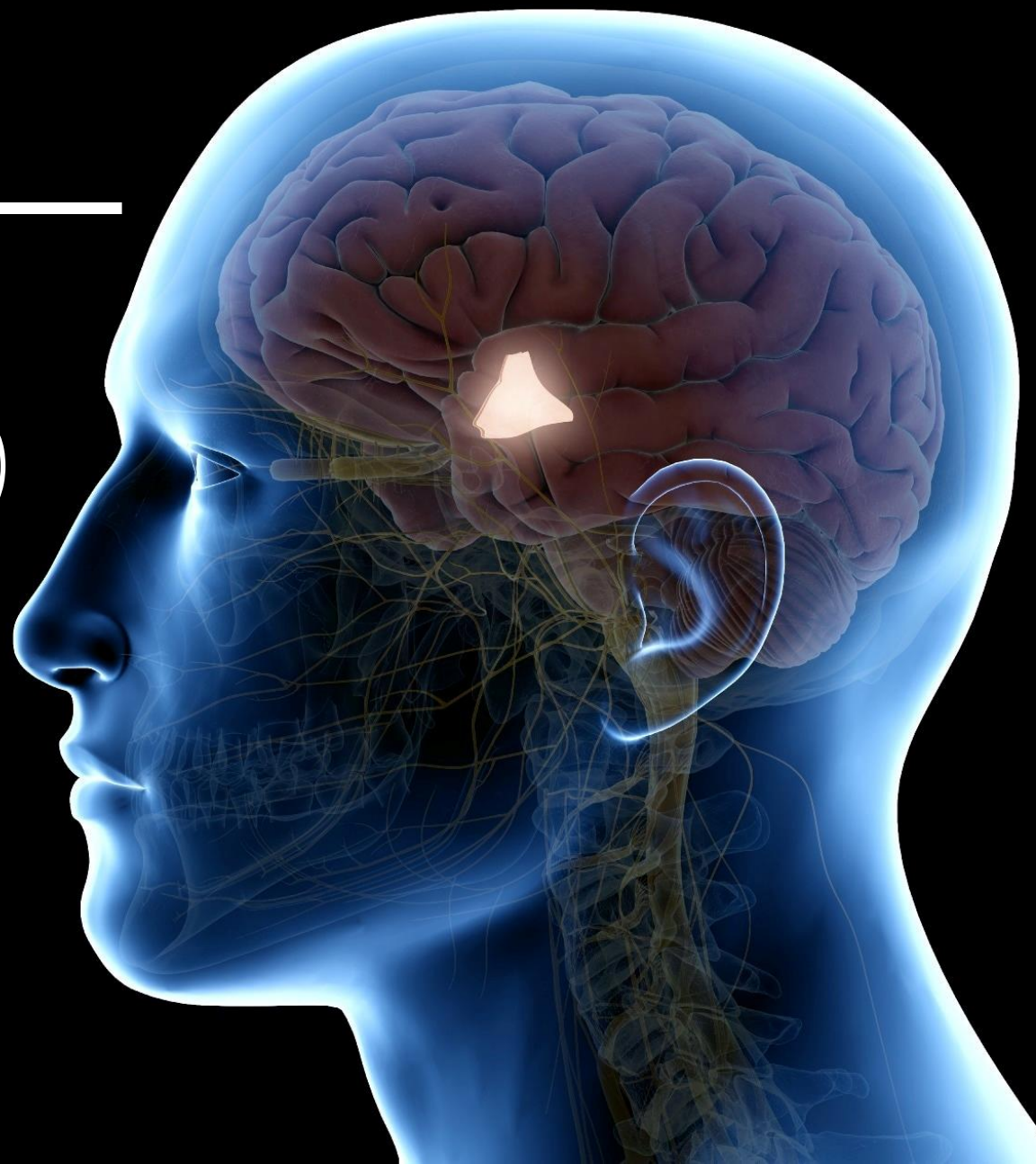
Hamartoma is a non progressive hypothalamic developmental malformation, which constitutes an ectopic site for production of GnRH that influences the Gonadotrophin secretion.



CAUSES

CNS lesions:

- Hypothalamic hamartoma (most common organic cause)
- Trauma
- Tuberculous/other meningitis
- Tumour



PERIPHERAL/ GONADOTROPHIN INDEPENDENT

PRECOCIOUS PUBERTY

PPP

- Due to estrogen or androgen secretion without HPG axis activation.
- Independent of GnRH and gonadotropins.
- Negative feedback suppresses hypothalamic GnRH and pituitary LH & FSH

FEATURES

- Breast development
- Growth of sexual hairs
- Unlikely to produce menarche
- Advanced bone age

TYPES of PPP

Depending upon the hormone being secreted

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Depending upon the hormone being secreted

HETEROSEXUAL

ISOSEXUAL

TYPES of PPP

Depending upon the hormone being secreted

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ISOSEXUAL

CAUSES

- CAH
- Androgen secreting tumours
- Medicinal intake of androgens

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Depending upon the hormone being secreted

HETEROSEXUAL

ISOSEXUAL

CAUSES

- CAH
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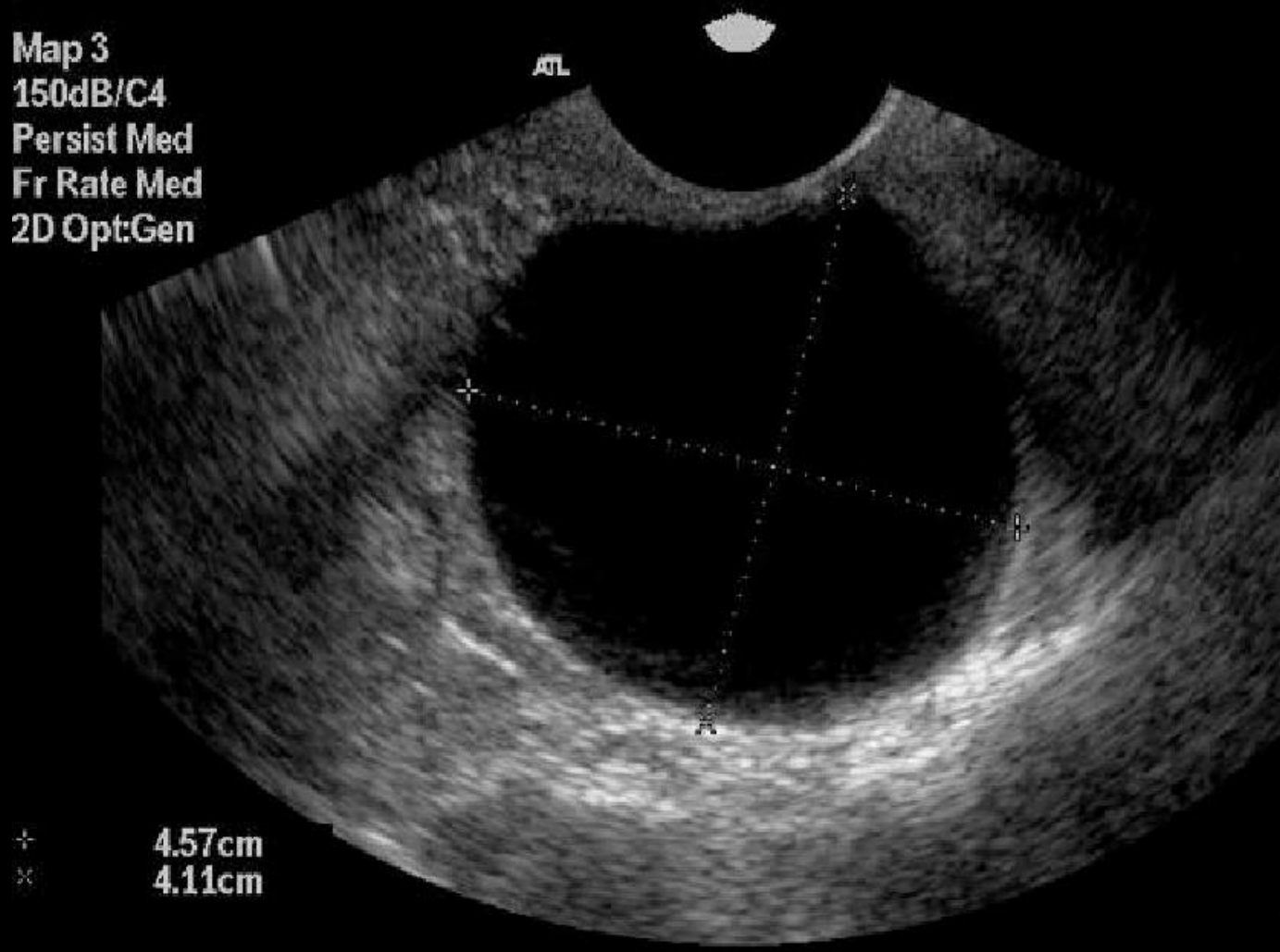
- McCune Albright syndrome
- Granulosa cell tumour

CAUSES

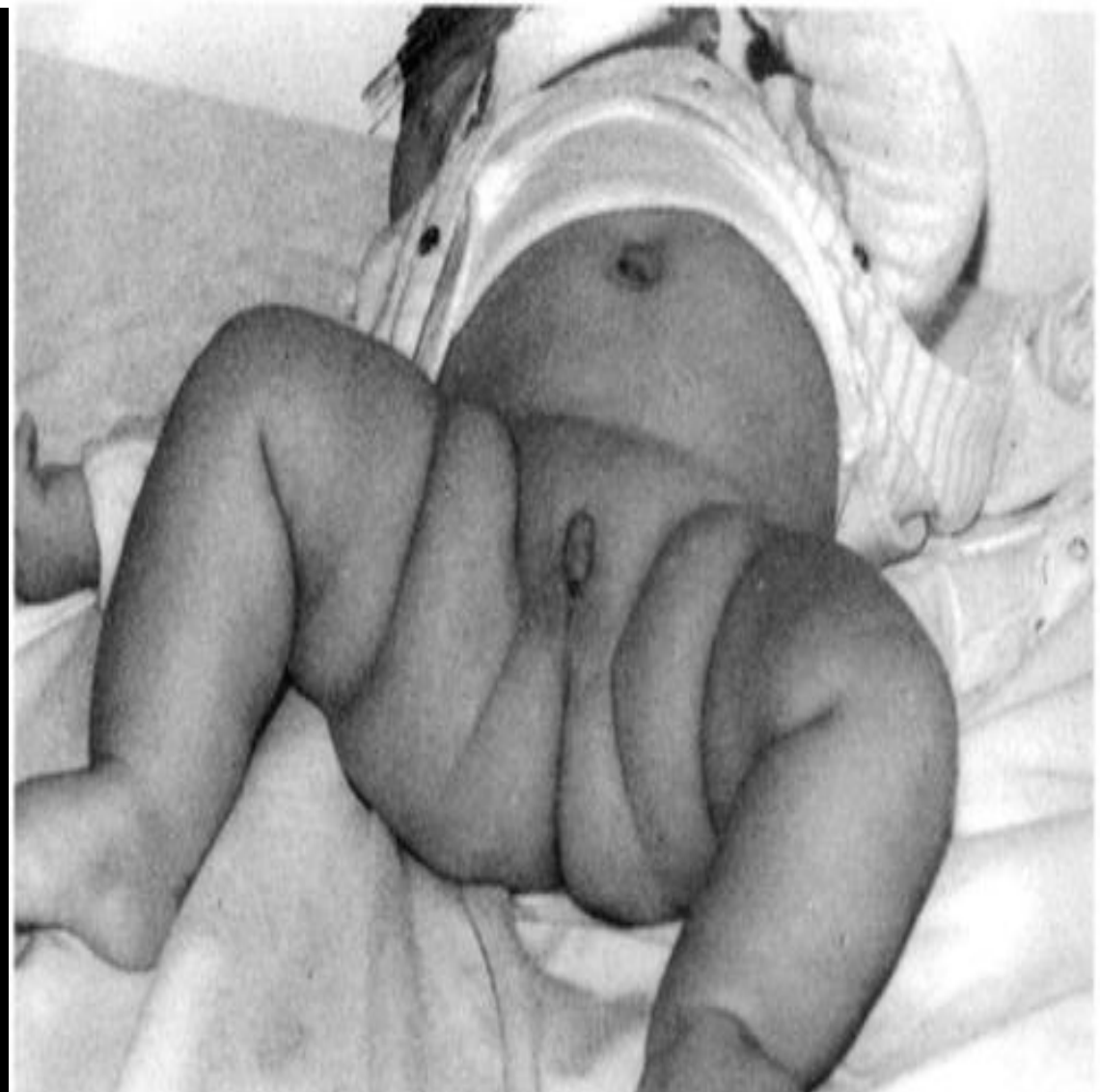
FUNCTIONAL OVARIAN CYST

- Most common cause
- Presentation – transient breast development & vaginal bleeding
- Ultrasound- unilateral/bilateral clear cyst > 15mm in diameter
- Self limited – do not need treatment
- Early manifestation of McCune Albright syndrome – need followup



CONGENITAL ADRENAL HYPERPLASIA

Congenital Adrenal Hyperplasia (CAH) is a group of genetic disorders that cause a deficiency in an enzyme needed to produce cortisol and aldosterone in the adrenal glands.



CONGENITAL ADRENAL HYPERPLASIA

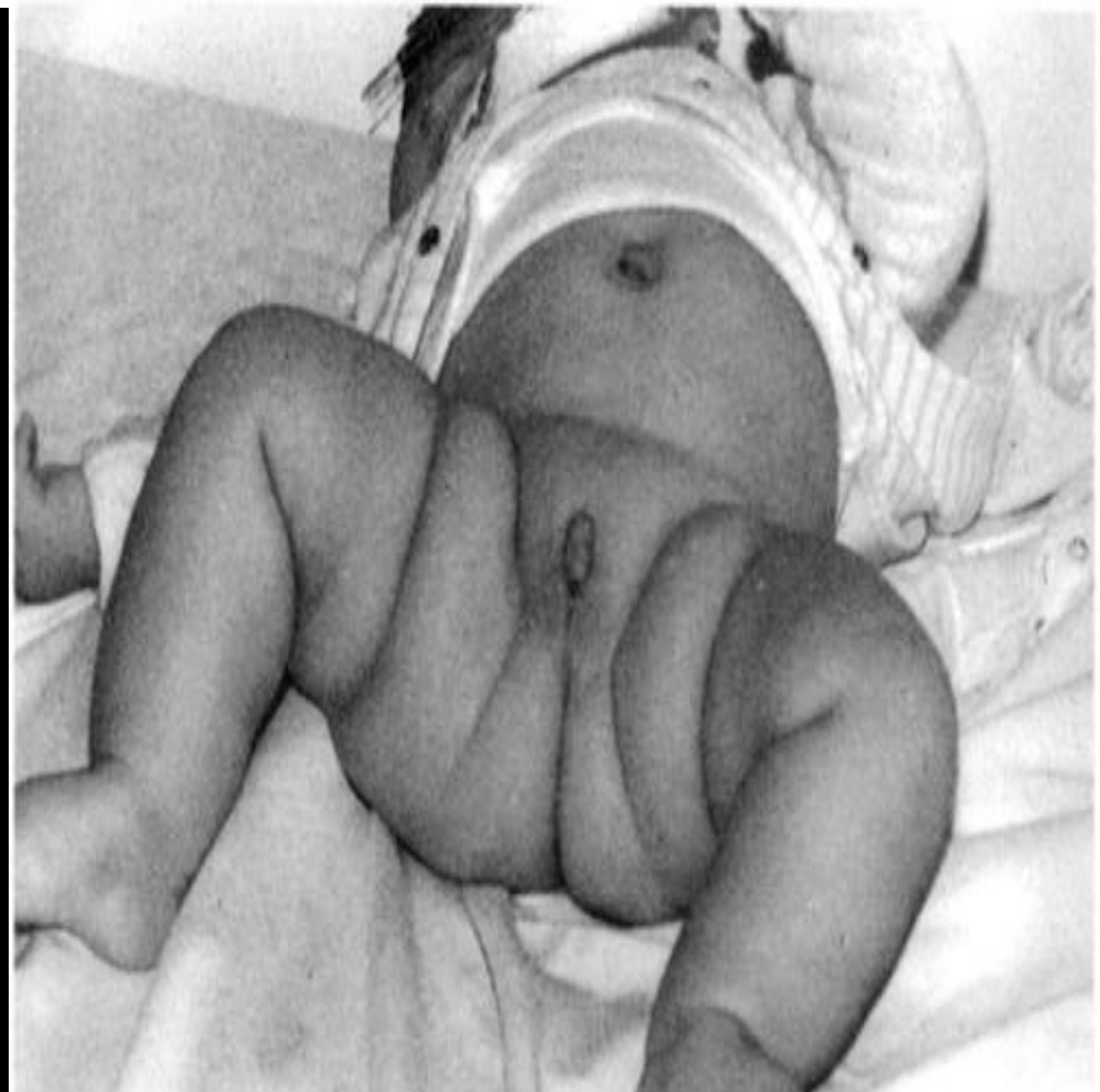
Block in cortisol synthesis

↓

Increased ACTH

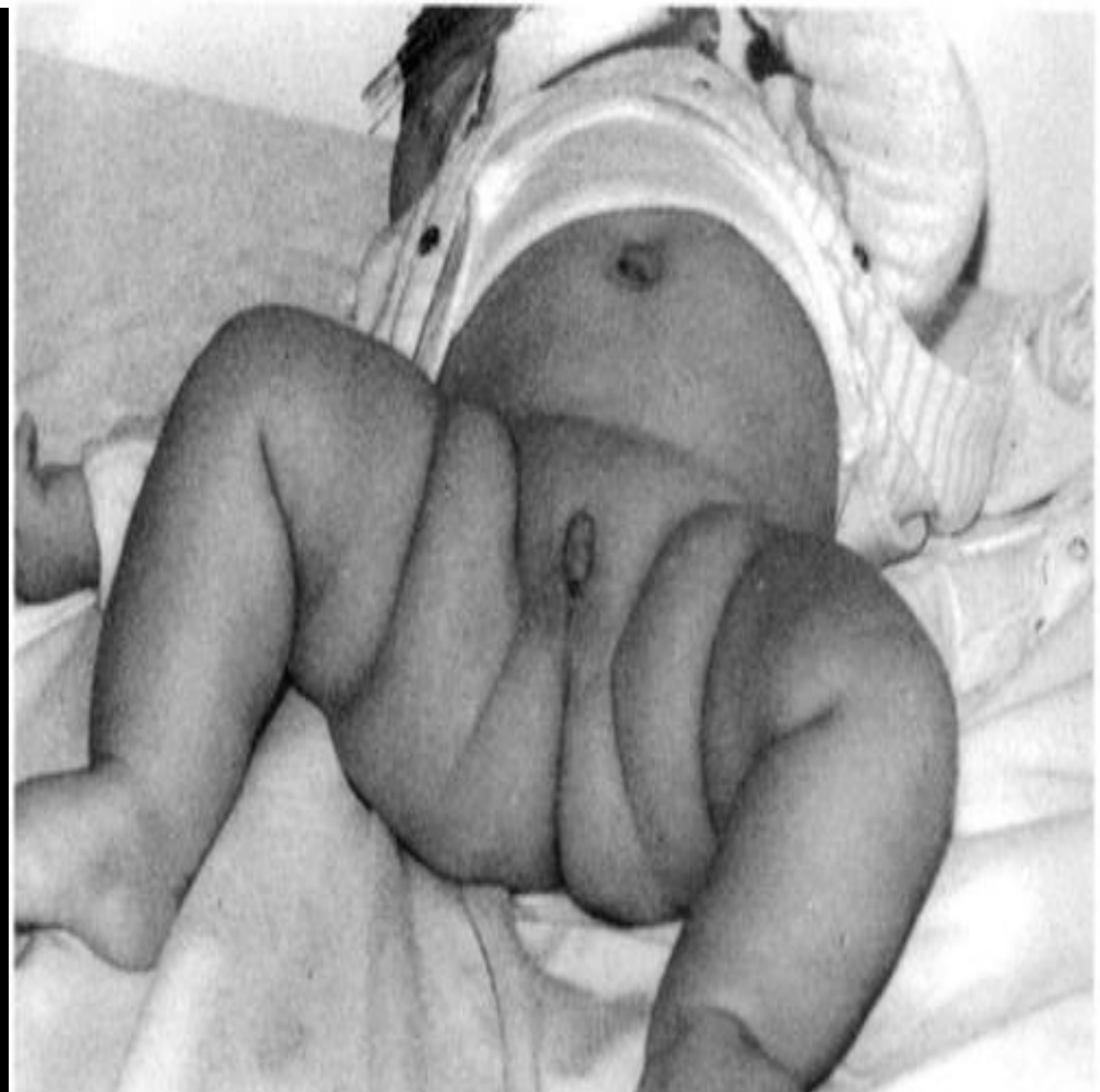
↓

Adrenal hyperplasia &
excess androgen production



CONGENITAL ADRENAL HYPERPLASIA

- Classic CAH: atypical genitalia at birth.
- Milder forms: present later with pubic hair in prepubertal girl.
- Treatment: glucocorticoids to stop excess androgen production.

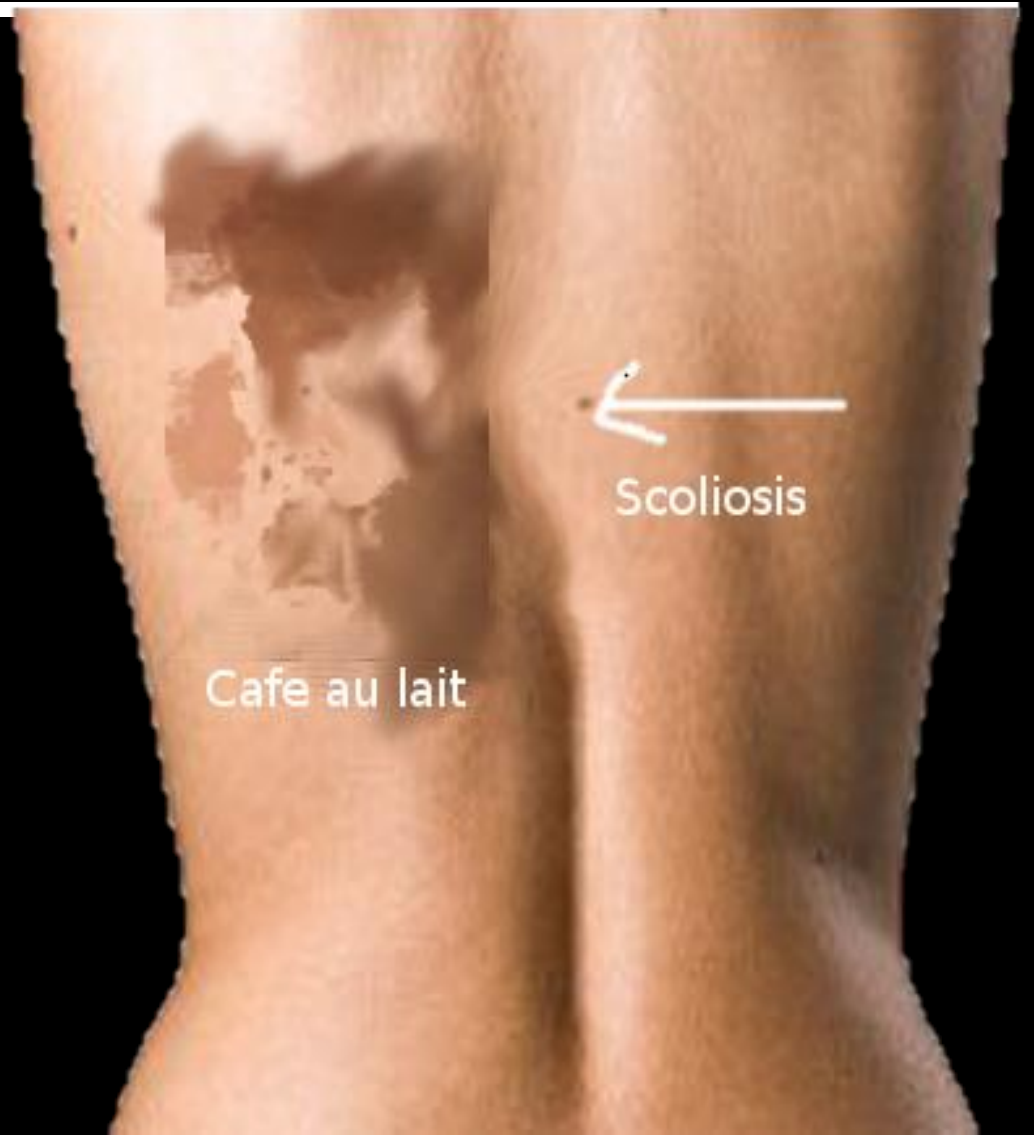


McCune Albright Syndrome

TRIAD

1.Polyostotic fibrous dysplasia
2. Café-au-lait spots
3. Precocious puberty

Cause: autonomous estrogen production (ovarian cysts).



NEOPLASMS

- Ovarian and adrenal neoplasms — though rare — can occur in prepubertal girls with pubertal changes.
- Granulosa cell tumours of the ovary can present with:
  - Breast enlargement
  - Vaginal bleeding

NEOPLASMS

- Leydig cell tumours and gonadoblastomas can cause virilising puberty (appearance of male characteristics).
- Clitoromegaly and pubic hair development in a small girl may indicate a sinister diagnosis like adrenocortical carcinoma.

EXOGENOUS ESTROGEN EXPOSURE

- Accidental use of oral contraceptive pills
- Estrogen creams
- Essential oils
- Make-up products with hormones.

Important to take history carefully.

INCOMPLETE /PARTIAL

PRECOCIOUS PUBERTY

INCOMPLETE PRECOCIOUS PUBERTY

- Only one sign of puberty appears early (before 8 years) without full activation of the HPG axis.
- Does not progress; may regress.
- Due to temporary hormone surge.

INCOMPLETE PRECOCIOUS PUBERTY

PREMATURE THELARCHE

- Breast development alone, before 8 years.
- No other signs of puberty.
- Often benign and self-limiting.

PREMATURE ADRENARCHE

- Pubic/axillary hair growth
- Due to increased DHEAS secretion.

PREMATURE MENARCHE

- Isolated vaginal bleeding (no other pubertal signs).
- Usually benign (must rule out trauma or sexual abuse).

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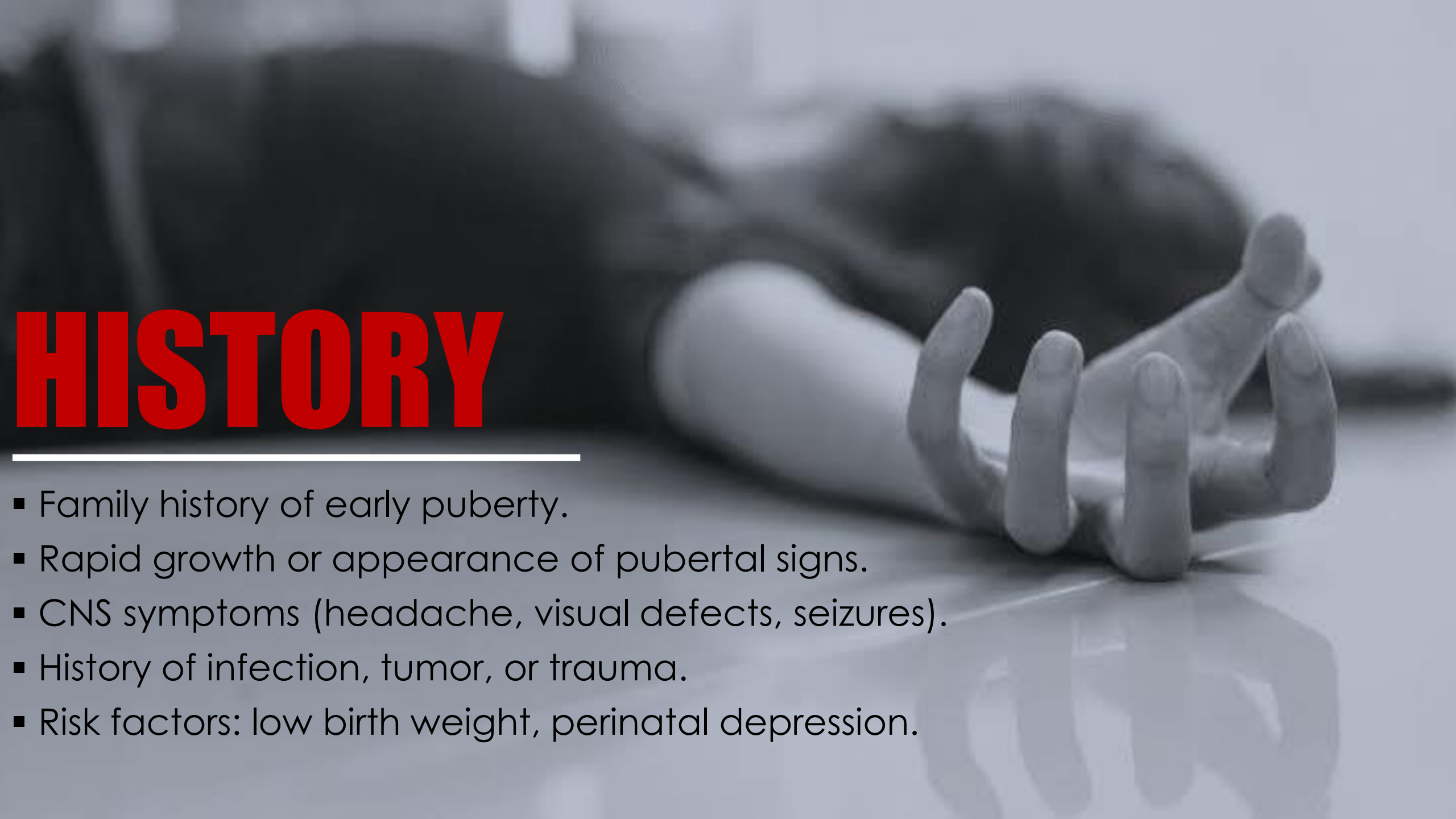
DIAGNOSIS



HISTORY

- Family history of early puberty.
- Rapid growth or appearance of pubertal signs.
- CNS symptoms (headache, visual defects, seizures).
- History of infection, tumor, or trauma.
- Risk factors: low birth weight, perinatal depression.

EXAMINATION

- Measure height, growth velocity (cm/year).
- Assess Tanner stage for breast/pubic hair.
- Fundoscopy: papilledema → intracranial cause.
- Café-au-lait spots: suggest MAS.

INVESTIGATIONS

BONE AGE

HORMONAL TEST

IMAGING

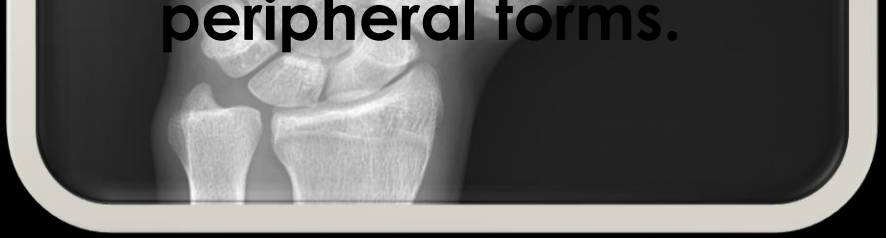
- Non dominant hand & wrist X-ray.
- Assesses advancement and tempo of puberty.
- Advanced in central & peripheral forms.

INVESTIGATIONS

BONE AGE

HORMONAL TEST

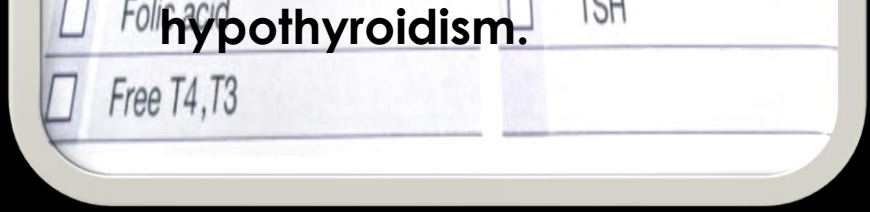
IMAGING

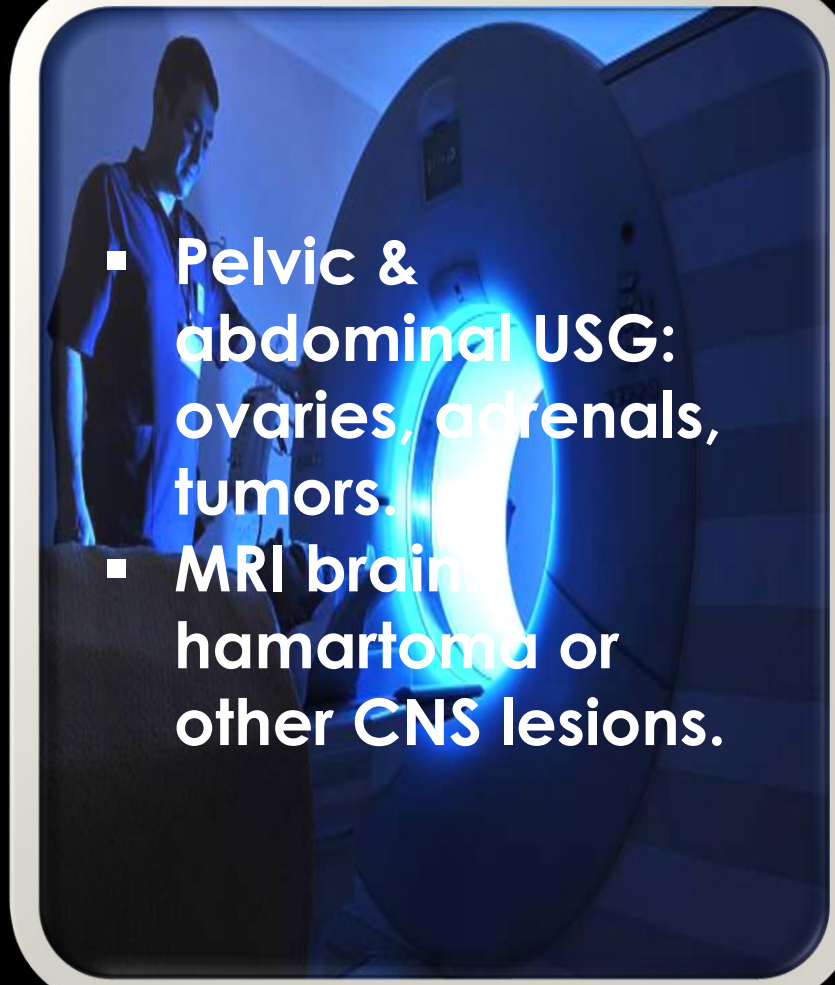


- FSH and LH
- LH <0.1 IU/L → prepubertal
- GnRH stimulation test:
- LH >3–5 IU/L → Central PP
- Estrogen/Testosterone – elevated in both types.
- DHEAS – high in adrenal cause.
- 17-OH Progesterone – ↑ in CAH.
- TSH – rule out hypothyroidism.

INVESTIGATIONS

- BONE AGE
- HORMONAL TEST
- IMAGING





- Pelvic & abdominal USG: ovaries, adrenals, tumors.
- MRI brain: hamartoma or other CNS lesions.


MANAGEMENT

CENTRAL PRECOCIOUS PUBERTY

- Goal: stop progression, arrest skeletal maturation, and manage psychological impact.
- These children should be guarded against sexual abuse.
- Treat underlying CNS lesions (surgery/radiation/chemotherapy if needed).
- GnRH agonists:
  Leuprolide (0.3 mg IM monthly)
  Triptorelin
- Continuous use ↓ LH/FSH secretion → ↓ sex steroids.
- Discontinue at expected normal puberty age.
- Outcome: ~75% achieve normal genetic height potential.

PERIPHERAL PRECOCIOUS PUBERTY

Treat underlying cause:

- Remove tumor if present.
- Stop exogenous hormones.
- Treat CAH with glucocorticoids.

Medications:

- Tamoxifen (anti-estrogen)
- Letrozole / Anastrozole (aromatase inhibitors).
- Pamidronate – for fibrous dysplasia in MAS
- Hypothyroidism-related sexual precocity → treat with thyroxine.

INCOMPLETE PRECOCIOUS PUBERTY

The variations of pubertal development such as premature thelarche, premature adrenarche and very rare benign self limiting conditions require only follow up to monitor for any progression.

REFERENCE:

SHEILA BALAKRISHNAN TEXBOOK OF GYNAECOLOGY 3rd EDITION