

Amenorrhea, Hirsutism & polycystic ovary syndrome

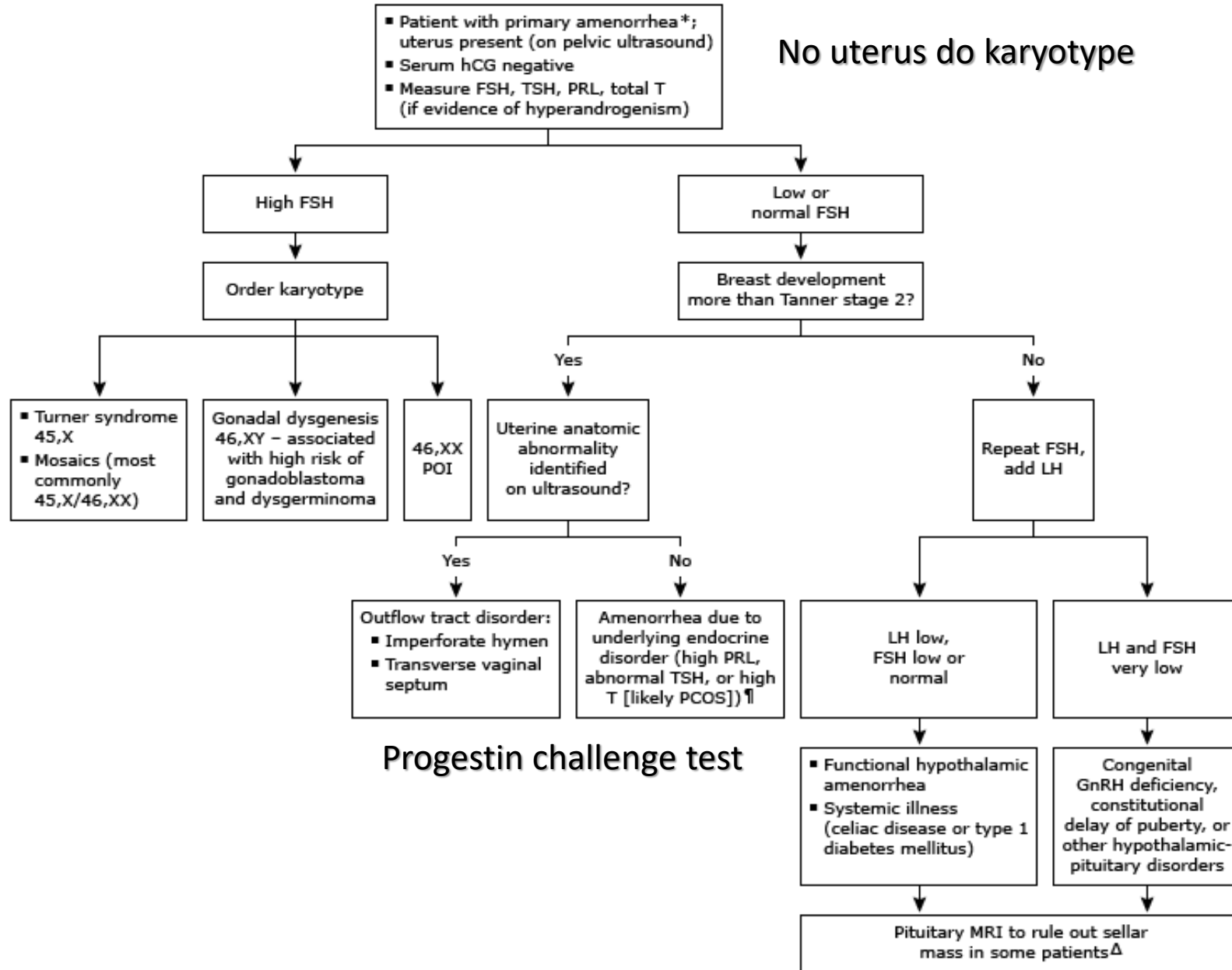
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Primary Amenorrhea

- Primary amenorrhea is defined as the absence of menses at age 15 years in the presence of normal growth and secondary sexual characteristics.
- However, at age 13 years, if no menses have occurred and there is a complete absence of secondary sexual characteristics such as breast development, evaluation for primary amenorrhea should also begin.
- This usually occurs as a manifestation of delayed puberty but may also be a consequence of anatomical defects of the female reproductive system.

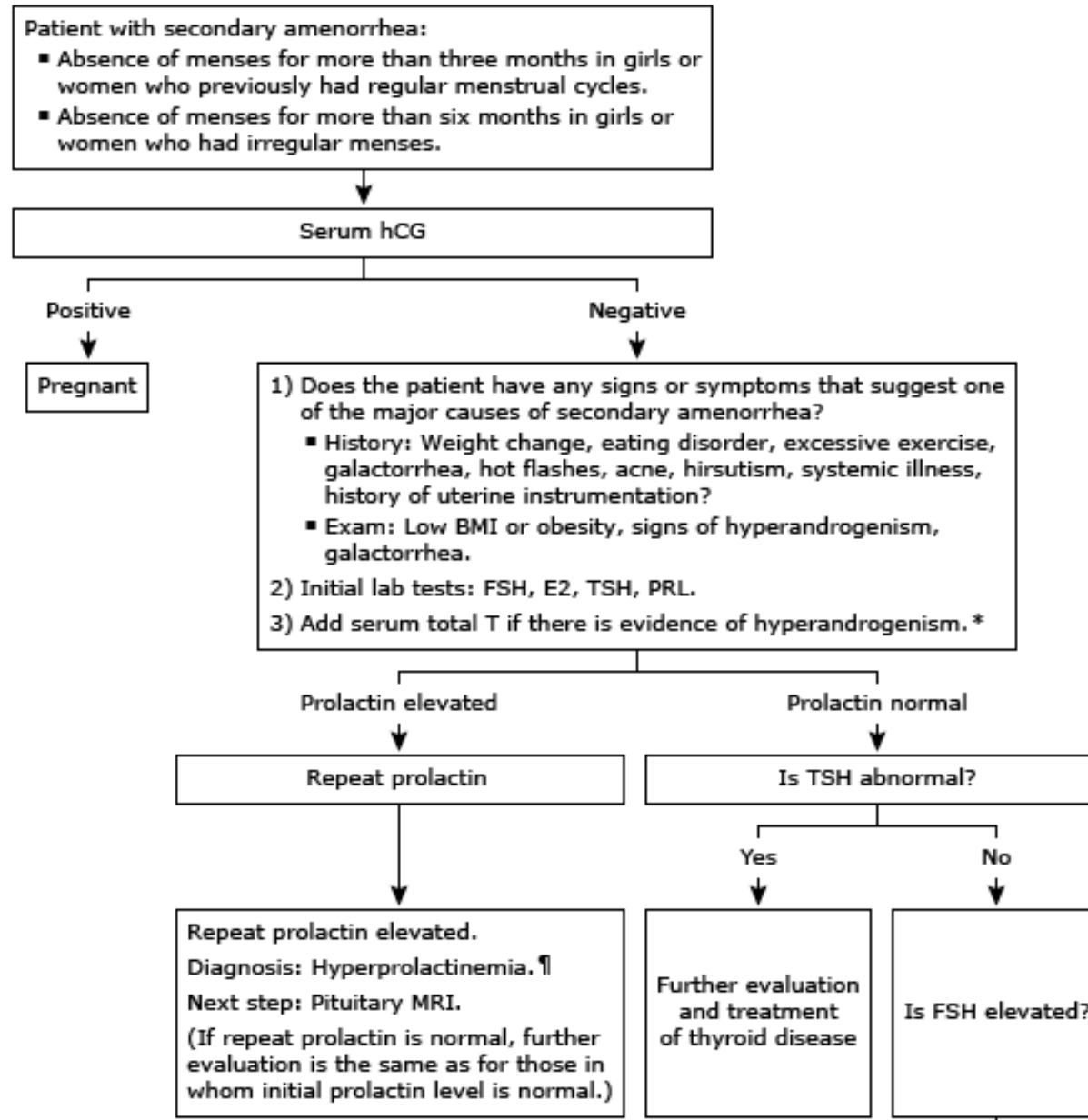
Evaluation and causes of primary amenorrhea



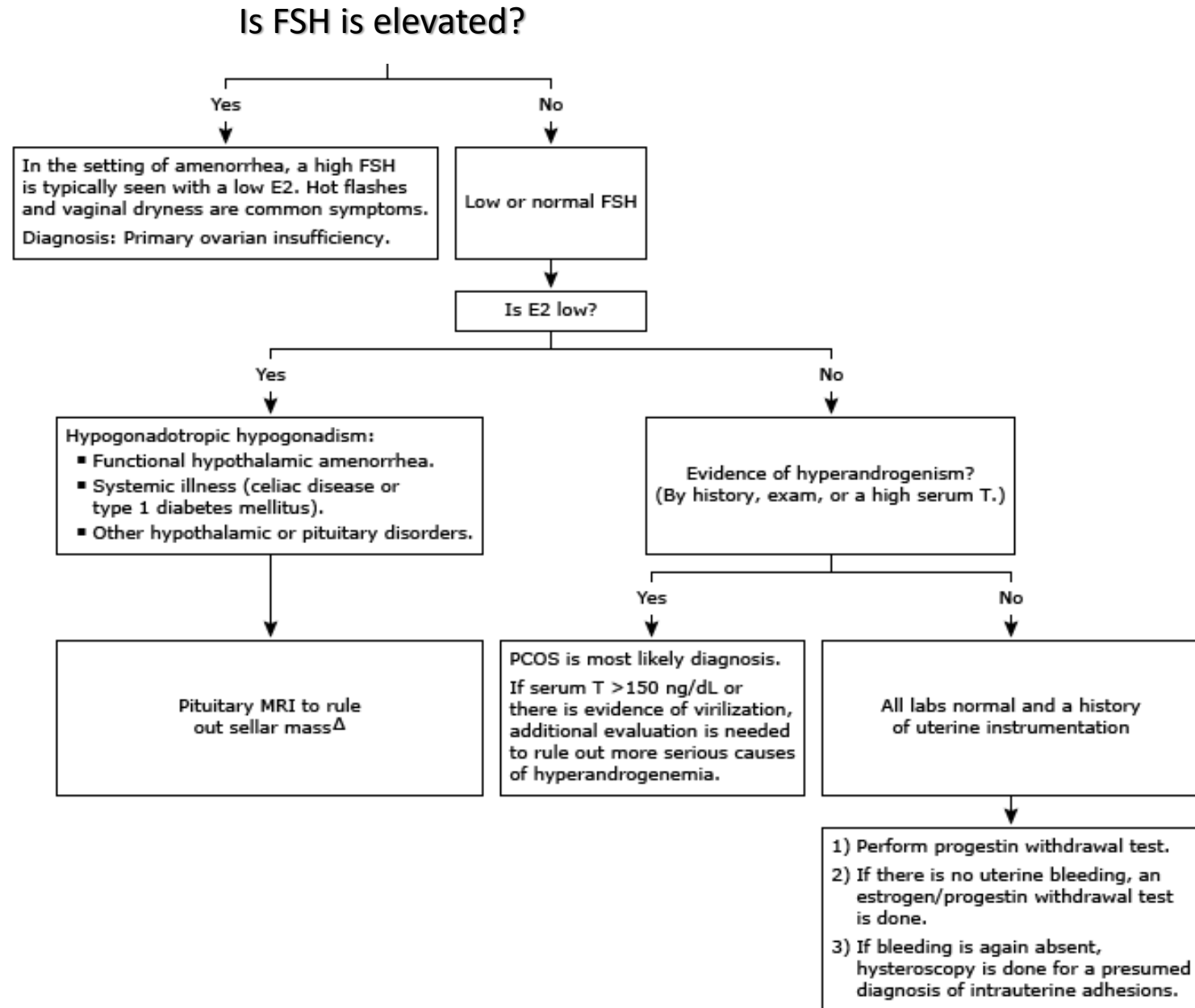
Secondary Amenorrhea

- Absence of menses for more than three months in girls or women who previously had regular menstrual cycles or six months in girls or women who had irregular menses.
- Missing a single menstrual period may not be important to assess, but amenorrhea lasting three months or more and oligomenorrhea (fewer than nine menstrual cycles per year or cycle length greater than 35 days) require investigation.
- The etiologic and diagnostic considerations for oligomenorrhea are the same as for secondary amenorrhea.

Evaluation and causes of secondary amenorrhea



Evaluation and causes of secondary amenorrhea (cont.)



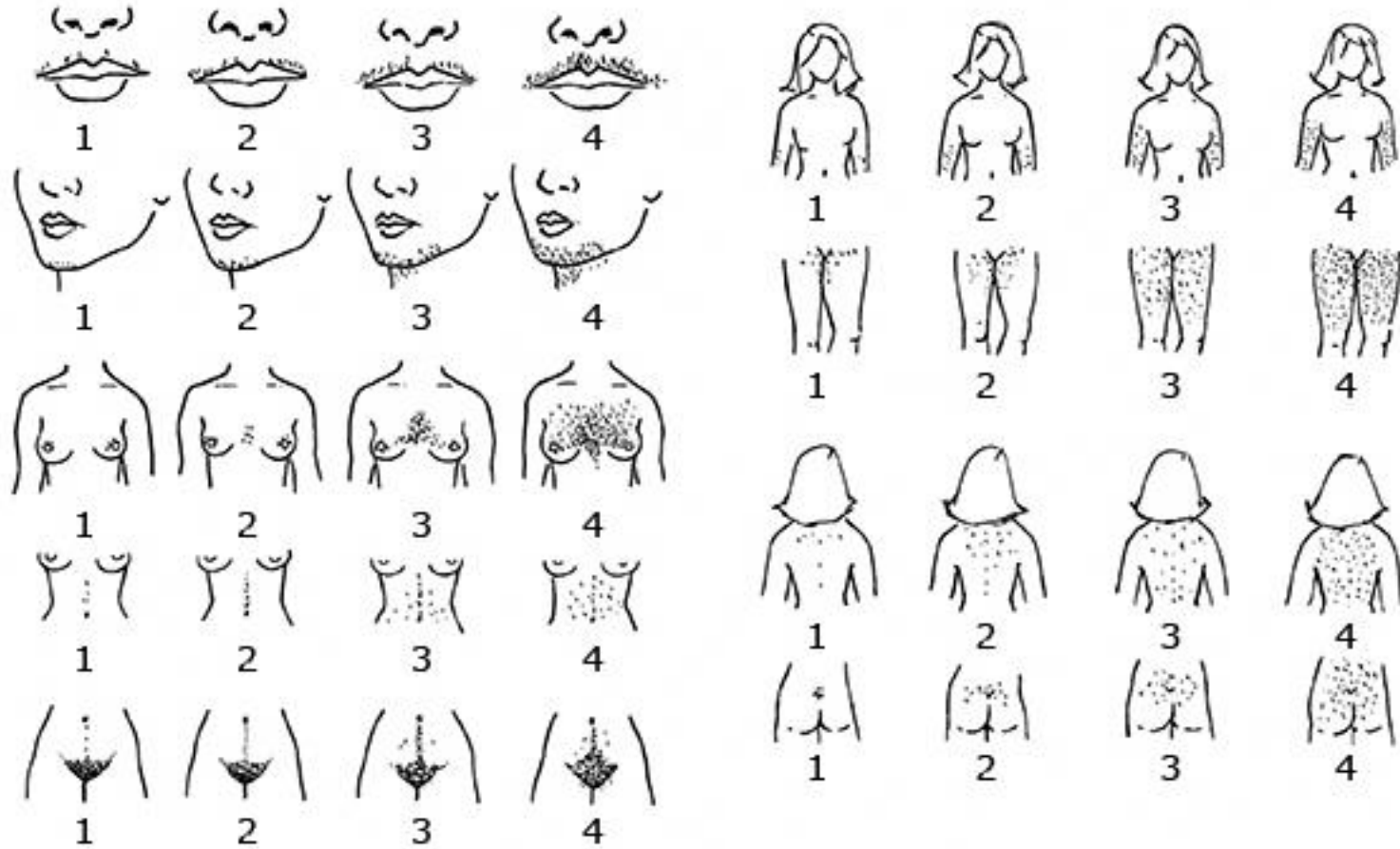
Management

- Directed toward the underlying cause.
- In oestrogen-deficient women, replacement therapy may be necessary to treat symptoms and/or to prevent osteoporosis.
 - Conjugated estrogen (day 1 to 21)+
 - progestogen (day 14 to 21) of the normal menstrual cycle.
- If oestrogenic side-effects (fluid retention, weight gain, hypertension and thrombosis) are a concern, then lower-dose oral or transdermal HRT may be more appropriate.

Hirsutism

- Hirsutism is a clinical diagnosis defined by the presence of excess terminal hair growth (dark, coarse hairs) in androgen-dependent areas (eg, upper lip, chin, mid-sternum, upper and lower abdomen, upper and lower back, and buttocks) in which women typically have little or no hair.
- It is one of the most common presentations of endocrine disease. Typically affects 5-10 % of premenopausal females.
- Hypertrichosis is generalized excessive growth of vellus hair (the thin, non-pigmented hair that is typically found all over the body from childhood onwards).

Grading of hirsutism (Ferriman-Gallwey score)



Clinical evaluation

- Drugs (androgens, **phenytoin, penicillamine, diazoxide, minoxidil, and cyclosporine cause hypertrichosis**)
- Menstrual history
- BMI
- BP
- Virilization (clitoromegaly, deep voice, male-pattern balding, breast atrophy) or others like acne vulgaris.
- Cushingoid features
- Hirsutism of recent onset associated with virilization is suggestive of an androgen-secreting tumor, but this is rare.

Polycystic ovary syndrome PCOS

- PCOS is thought to be one of the most common endocrinopathies in women, affecting 10 percent of women in the reproductive age.
- PCOS is an important cause of both menstrual irregularity and androgen excess in women.
- Genetic factors probably play a role, since PCOS often affects several family members.

Clinical features of PCOS

Anovulatory cycles	Oligomenorrhea, amenorrhea (secondary or primary), and infertility
Hyperandrogenism	Clinical and biochemical
Polycystic ovaries	The typical polycystic appearance of the ovaries is seen on transvaginal ultrasound (TVUS). ≥20 follicles in each ovary and/or increased ovarian volume (>10 mL)
Metabolic/cardiovascular risk	Obesity/overweight up 85%, insulin resistance, IGT or T2DM, CVD risk, NAFLD, and sleep apnea
Mood	Depression, anxiety, eating disorders

PCOS diagnostic criteria

Two out of three of the following criteria are required to make the diagnosis

Oligo- and/or anovulation

Clinical and/or biochemical signs of hyperandrogenism

Polycystic ovaries (by ultrasound)

The diagnosis of PCOS is only **confirmed** when other conditions that mimic PCOS are excluded (eg, disorders that cause oligo/anovulation and/or hyperandrogenism, such as thyroid disease, NCCAH, hyperprolactinemia, and androgen-secreting tumors).

Causes, investigations, and treatment of hirsutism

Causes	Clinical features	Investigation finding	Treatment
Idiopathic	Often familial Mediterranean or Asian background	Normal	Cosmetic Anti-androgen
PCOS	Obesity Amenorrhea/oligomenorrhea Infertility	LH/FSH ratio > 2.5 Minor elevation in androgens (T, DHEA-S) Mild hyper PRL Pelvic/transvaginal US	Weight reduction Cosmetics Anti-androgens Metformin, glitazones, OCPs Gonadotrophins, clomifene
Non-classical CAH	Similar presentation to PCOS	Minor elevation in androgens (T, DHEA-S) high serum 17-OHP (>200 ng/ml, after ACTH > 1500 ng/ml)	Anti-androgens GCS
Exogenous androgens	Athletes Virilization	Low FSH, LH Urinary androgens	Stop androgens
Androgen-secreting ovarian or adrenal tumor	Rapid onset virilization	DHEA-S > 700 Mg/dl T > 150 ng/ml CT/MRI/pelvic US	Surgery
Cushing's syndrome	Typical features	Diagnostic tests	Surgery

Anti-androgens

Mechanism	Drug	Hazards
Androgen receptor antagonists	Cyproterone acetate Spironolactone Flutamide XXX	Hepatic dysfunction Feminization of male fetus Progesterone receptor agonist Dysfunctional uterine bleeding (DUB) Electrolyte's disturbances, DUB Hepatic injury
5 alpha reductase inhibitor	Finasteride	Less effective
Suppression of ovarian steroid production and elevation of sex hormone-binding globulin	Estrogens	Venous thromboembolism Hypertension Weight gain Dyslipidaemia Increased breast and endometrial carcinoma