D.PHARM • SECOND YEAR

Pharmacotherapeutics

CHAPTER 2.13

Women’s Health



ENGLISH CHAPTER NOTES

DIPLOMA IN PHARMACY

POLYCYSTIC OVARY SYNDROME (PCOS)

DEFINITION

A multifactorial endocrine disorder in reproductive-aged women, involving hyperandrogenism, ovulatory dysfunction and/or polycystic ovarian morphology.
A major and common cause of anovulatory infertility.

MECHANISMS

- Familial tendency; genetic and environmental factors interact.
- Obesity can worsen insulin resistance and symptoms, but is not required for PCOS.

Insulin resistance → hyperinsulinemia → increased androgen production and reduced SHBG.
SHBG = sex hormone-binding globulin.

- Ovarian and adrenal androgen excess contributes to hirsutism and acne.
- Follicular arrest prevents normal dominant-follicle development and ovulation.
- LH:FSH may be raised; a ratio **>2:1** is not a diagnostic rule.

ADULT DIAGNOSIS

Rotterdam criteria: **2 of 3**, after excluding other causes:

1. Oligo/anovulation.
2. Clinical or biochemical hyperandrogenism.
3. Polycystic ovarian morphology.

ULTRASOUND UPDATE

- With suitable modern imaging: **≥20** follicles, sized **2–9 mm**, in at least **1** ovary.
- Ovarian volume **≥10 mL** can be used when technology is older or follicle counts are unreliable.
- The older **≥12**-follicle threshold is not the current modern-imaging criterion.
- Ultrasound morphology is not recommended for diagnosing PCOS in adolescents.

POLYCYSTIC OVARY SYNDROME (PCOS)

CLINICAL FEATURES

- Oligomenorrhea: infrequent periods.
- Amenorrhea: absent periods.
- Infertility due to anovulation.
- Hirsutism: excess terminal hair on the face, chest or abdomen.
- Acne and oily skin.
- Central obesity may be present.
- Acanthosis nigricans: dark, thickened skin suggesting insulin resistance.
- Depression, anxiety and body-image distress.

LONG-TERM RISKS

- Type 2 diabetes mellitus.
- Endometrial hyperplasia and cancer.
- Dyslipidemia and increased cardiovascular risk.
- NAFLD: non-alcoholic fatty liver disease.

Risks need assessment and follow-up; not every patient develops these complications.

LIFESTYLE AND SUPPORT

- Healthy lifestyle is foundational for everyone, even without weight loss.
- If overweight, achievable weight reduction helps metabolic health.
- A 5–10% weight loss may improve ovulation; it is not guaranteed.
- Balanced, sustainable diet; low-glycemic-index foods may help.
- Regular aerobic and resistance exercise.
- Psychological support for mood and body image.
- Discuss reproductive goals, fertility care and preservation when appropriate.

POLYCYSTIC OVARY SYNDROME (PCOS)

CYCLES AND ANDROGEN SYMPTOMS

Combined Oral Contraceptives (COCs) regulate cycles and reduce hirsutism or acne.

Select by symptoms, contraindications and preferences; drospirenone is an example, not universally the best choice.

ANTI-ANDROGENS

- **Spironolactone** may help persistent hirsutism and acne.
- **Finasteride** is a specialist alternative.
- **Flutamide** is generally avoided because of serious liver toxicity.

Effective contraception is essential when pregnancy is possible because anti-androgens can harm fetal development.

FERTILITY TREATMENT

Letrozole: aromatase inhibitor; first-line ovulation induction for anovulatory infertility when no other infertility factor is present.

Clomiphene citrate: an alternative ovulation-induction medicine.

Ovulation induction requires specialist assessment and monitoring.

INSULIN RESISTANCE

Metformin improves insulin sensitivity and metabolic outcomes; may help cycles and ovulation.

It is not the most effective ovulation-induction treatment by itself.

ACNE

Topical retinoids, appropriate antibiotics or hormonal treatment may be used.

Avoid topical retinoids during pregnancy or when planning pregnancy.

INDIVIDUALIZE

Pregnancy goals, metabolic risks and the dominant symptoms guide treatment.

COCs prevent pregnancy; they are not infertility treatment.

DYSMENORRHEA

DEFINITION: Painful menstruation; symptoms may be severe enough to interfere with daily activities.

PRIMARY DYSMENORRHEA

- Painful menses without identifiable pelvic disease.
- Usually begins **6–12 months** after menarche, as ovulatory cycles become established.

MECHANISM

Excess endometrial prostaglandin **F2α (PGF2α)**
↓
strong uterine contractions and reduced blood flow
↓
ischemia and pain.

FEATURES

- Crampy lower-abdominal pain at or just before menstruation.
- Pain may radiate to the lower back or thighs.
- Nausea, vomiting, diarrhea, fatigue and headache.
- No identifiable pelvic abnormality on evaluation.

SECONDARY DYSMENORRHEA

- Menstrual pain due to pelvic pathology.

CAUSES

- Endometriosis.
- Adenomyosis.
- Fibroids.
- Pelvic inflammatory disease (PID).
- Copper IUDs may worsen cramps; not all IUDs do.

FEATURES

- Pain may start earlier and persist after menstruation.
- Dyspareunia; pain during intercourse.
- Abnormal bleeding or infertility may occur.
- Progressive, atypical or persistent pain needs pelvic evaluation.

Treat the underlying cause, not only the pain.

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