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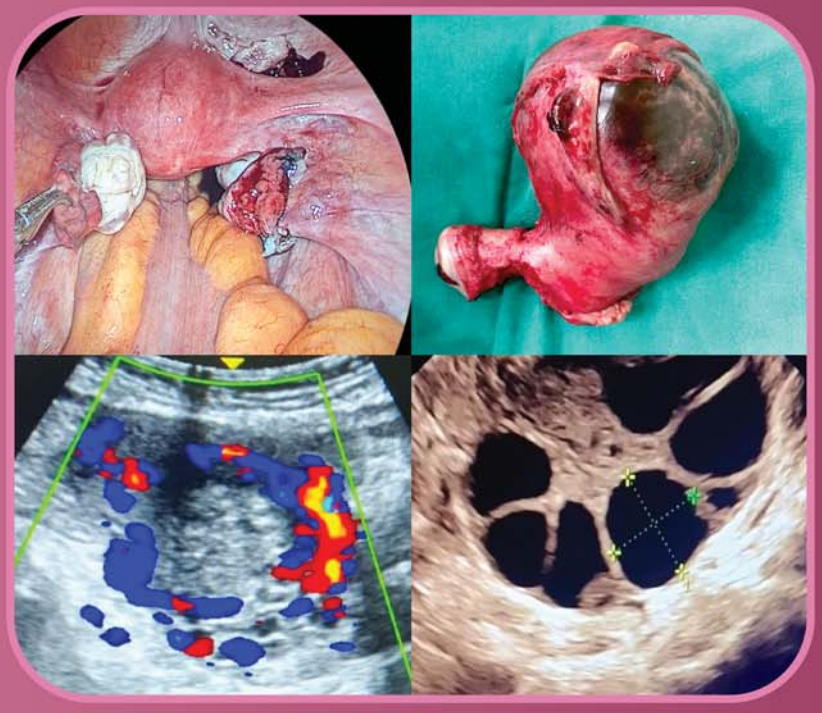

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CHAPTER 34

Polycystic Ovary Syndrome: Diagnosis, Pathophysiology, and Management

Neelansha Varshney, Anu Chaudhary

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is a prevalent endocrinological disorder affecting 10–13% of reproductive-aged women globally, with significant implications for fertility, metabolic health, and quality of life.[1] The European Society of Human Reproduction and Embryology (ESHRE) 2023 guidelines endorse the Rotterdam 2003 criteria for PCOS diagnosis, emphasizing a nuanced approach to adolescents and incorporating advancements in ultrasound and metabolic management.[2]

DIAGNOSIS OF PCOS

Rotterdam Criteria (ESHRE/ASRM 2003, Endorsed by ESHRE 2023)

PCOS diagnosis in adults requires **two of three** criteria, with exclusion of other etiologies (e.g., congenital adrenal hyperplasia, thyroid dysfunction):[2]

- **Oligo-/Anovulation:**
  - Oligoamenorrhea: Cycles >35 days or <8 menses/yr.
  - WHO classification of ovulation disorders **(Table 1)**:
    - Group I: Hypothalamic–pituitary failure (hypogonadotrophic hypogonadism).
    - Group II: Hypothalamopituitaryovarian dysfunction (predominantly PCOS).
    - Group III: Ovarian failure.[3]
- **Clinical/Biochemical Hyperandrogenism:**
  - **Clinical:** Modified Ferriman-Gallwey (mFG) score ≥4–8 for hirsutism **(Table 2)**.[4]
  - **Biochemical:** Elevated total/free testosterone or free androgen index (FAI = Total Testosterone/SHBG × 100).
- **Polycystic Ovarian Morphology (PCOM):**
  - Ovary volume ≥10 cc and/or ≥20 follicles (2–9 mm) per ovary on transvaginal ultrasound (TVS) with ≥8 MHz transducer[5] as shown in **Figures 1A and B**.

TABLE 1: WHO classification of ovulation disorders.[3]

Group	*Description*	*Example*
I	Hypothalamic–pituitary failure	Hypogonadotrophic hypogonadism
II	Hypothalam opituitary ovarian dysfunction	PCOS
III	Ovarian failure	Premature ovarian insufficiency

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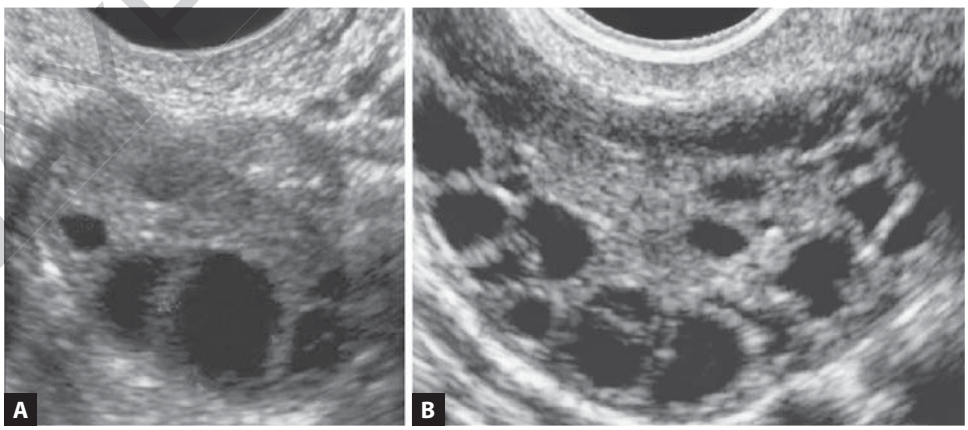
TABLE 2: Modified Ferriman-Gallwey scoring system for hirsutism (9 areas, score 0–4 per area, total ≥4-8 indicates hirsutism).[4]

Site	*Grade 1*	*Grade 2*	*Grade 3*	*Grade 4*
Upper lip	Few hairs at the outer margin	Small moustache at outer margin	Moustache extending halfway from outer margin	Moustache extending to mid-line
Chin	Few scattered hairs	Scattered hairs with small concentrations	Complete cover, light	Complete cover, heavy
Chest	Circumareolar hairs	Circumareolar hairs with mid-line hair	Fusion of circumareolar hairs with mid-line hair giving three-quarter cover	Complete cover
Upper back	Few scattered hairs	More than a few scattered hairs but still scattered	Complete cover, light	Complete cover, heavy
Lower back	Sacral tuft of hair	Sacral tuft of hair with some lateral extension	Three-quarter cover	Complete cover
Upper abdomen	Few mid-line hairs	Rather more but still mid-line	Half cover	Complete cover
Lower abdomen	Few mid-line hairs	Mid-line streak of hair	Mid-line band of hair	An inverted V-shaped growth
Upper arm and thigh	Sparse hair growth affecting not more than a quarter of limb surface	More than a quarter coverage but still incomplete	Complete cover, light	Complete cover, heavy
Forearm and legs	–	–	–	Complete cover, heavy

Note: Forearm and hand, lower leg, and feet are **not included** in the "hormonal" score and only single value is added even when hirsutism involves these extremities bilaterally. Minimum score is **0** and maximum score is **36**. A score of **<8** = No hirsutism, **8–16** = Mild hirsutism,**17–25** = Moderate hirsutism, **>25** = Severe hirsutism. Values may vary across different ethnic groups.



Figs. 1A and B: Difference between (A) normal ovary and (B) polycystic ovary.[60]

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Adolescent PCOS Diagnosis (ESHRE 2023)

Adult criteria may overdiagnose PCOS in adolescents due to pubertal overlap (e.g., irregular cycles, multifollicular ovaries). Diagnosis requires:[6]

- **Irregular Menstrual Cycles** (based on years post-menarche):
  - *90 days for any cycle (>1 year post-menarche).*
  - Cycles <21 or >45 days (1–3 years post-menarche).
  - Cycles <21 or >35 days (>3 years post-menarche).
  - Primary amenorrhea by age 15 or >3 years post-thelarche.
  - <1 year post-menarche: Normal pubertal transition, no diagnosis.
- **Hyperandrogenism:** Hirsutism (mFG ≥4–8), severe acne, or biochemical hyperandrogenemia (high-quality assays). Hirsutism is a stronger marker than acne, which may be transient.[7]
- **Ultrasound:** Not recommended <8 years post-menarche due to a high incidence of multifollicular ovaries.[8]
- **Anti-Müllerian Hormone (AMH):** Not used for diagnosis due to lack of standardized cutoffs.[9]
- **Consensus:** Diagnosis deferred until 2 years post-menarche; all Rotterdam criteria (anovulation, hyperandrogenism, PCOM) required for confirmation.[10]

Historical Diagnostic Criteria

The Rotterdam criteria remain the gold standard, superseding earlier frameworks **(Table 3)**.[11]

TABLE 3: Evolution of PCOS diagnostic criteria.[11]

Criteria	*Year*	*Requirements*
NIH	1990	Hyperandrogenism + anovulation
AE-PCOS	2006	Hyperandrogenism + one of anovulation/PCOM
Rotterdam	2003	Two of: Hyperandrogenism, anovulation, PCOM
NIH 2012	2012	Rotterdam + phenotype identification

Phenotypes and BMI in PCOS

PCOS phenotypes reflect clinical heterogeneity, influencing management **(Tables 4 and 5)**:[12]

- **Type A:** Anovulation + Hyperandrogenism + PCOM (classic PCOS).
- **Type B:** Anovulation + Hyperandrogenism.
- **Type C:** Hyperandrogenism + PCOM.
- **Type D:** Anovulation + PCOM (nonhyperandrogenic).

Mnemonic: Type **A** for **A**ll (all three are present)

1. Type **B** for **B** mode ultrasound (USG criteria is absent)
2. Type **C** for **C**ycles (cycles are normal)
3. Type **D** for an**D**rogenic (androgenic feature is not there)

TABLE 4: PCOS phenotypes.[12]

Phenotype	*Features*	*Prevalence*
A	Anovulation + Hyperandrogenism + PCOM	60–70%
B	Anovulation + Hyperandrogenism	15–20%
C	Hyperandrogenism + PCOM	10–15%
D	Anovulation + PCOM	5–10%

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TABLE 5: Different PCOS phenotypes.

Feature	*Phenotype A*	*Phenotype B*	*Phenotype C*	*Phenotype D*
Hyperandrogenism and hirsutism	✓	✓	✓	✗
Ovulatory dysfunction	✓	✓	✗	✓
Polycystic ovarian morphology	✓	✗	✓	✓

- **BMI:**
  - **Lean PCOS:** Elevated luteinizing hormone (LH), normal insulin sensitivity.
  - **Obese PCOS:** Insulin resistance, hyperandrogenemia; insulin amplifies LH effects.[13]

Pathophysiology of PCOS (Fig. 2)

PCOS arises from complex interactions of hyperinsulinemia, hyperandrogenism, and genetic predisposition, perpetuating a cycle of anovulation and metabolic dysfunction.[14]

- **Genetic Variation in Gonadotropin Secretion:**
  - Increased LH pulse frequency/amplitude, low-normal follicle-stimulating hormone (FSH), LH:FSH ratio 2:1 or 3:1.
  - No progesterone feedback (due to anovulation) sustains high LH.
  - Reduced FSH from:
    - Rapid gonadotropin-releasing hormone (GnRH) pulses.
    - Elevated estrone (E1) from peripheral aromatization of androstenedione.
    - Inhibin B from small antral follicles.[15]
- **Insulin Secretion and Action:**
  - Insulin sensitivity reduced by 35–40%; insulin resistance in 65–80% of PCOS women.
  - Impaired glucose tolerance (IGT) in 35%; type 2 diabetes mellitus (T2DM) in 7–10%.
- **Insulin promotes:**
  - Ovarian androgen synthesis.
  - Reduced sex hormone-binding globulin (SHBG), increasing free androgens.
  - Anovulation via synergistic LH effects.[16]
- **Androgen Synthesis and Action:**
  - Hyperandrogenism (from ovaries, adrenals, or peripheral tissues) disrupts GnRH feedback, increasing LH and intraovarian androgens.
  - Continuous LH stimulation and stromal hyperplasia cause follicular atresia, forming PCOM.
  - *Cycle:* Hyperandrogenism → anovulation → stromal hypertrophy → hyperandrogenism.[17]

Clinical Features[18]

- **Anovulation:** Oligomenorrhea due to inhibited FSH-induced LH receptor formation, preventing dominant follicle development.
- **Hyperandrogenism:** Hirsutism, acne, androgenic alopecia.
- **Infertility:** Anovulation is the primary cause.

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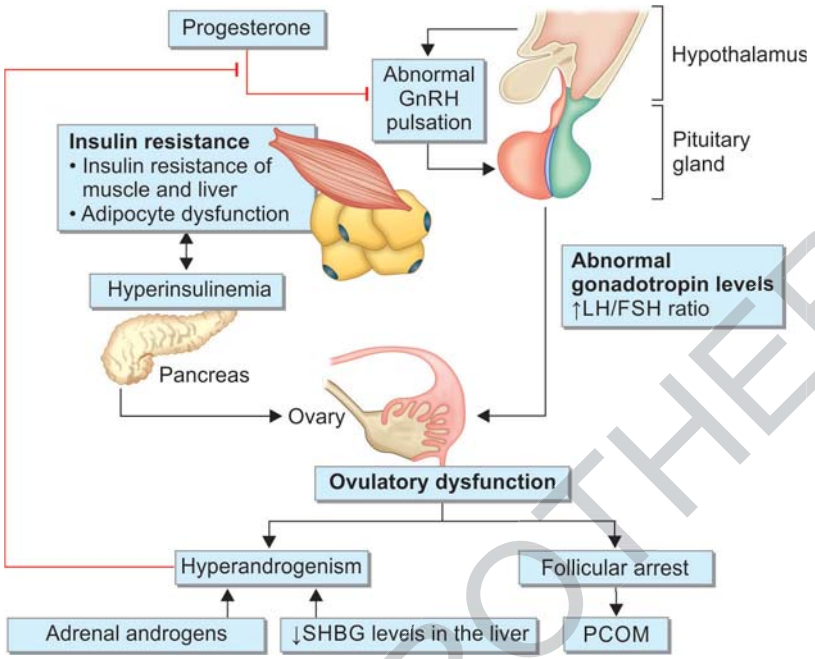

Fig. 2: Pathophysiology of PCOS (cycle of hyperandrogenism, insulin resistance, and anovulation).[14]

Q1. Assessment and Treatment of Infertility in PCOS. *(UP 2011, 2019, 5 + 5 Marks)*

Assessment of Infertility in PCOS

Infertility in PCOS primarily stems from anovulation, but other female, male, or combined factors must be excluded. A comprehensive evaluation includes history, examination, and investigations.[19]

- **History:**
  - **Couple:**
    - Duration of marriage, contraception use, fertile window knowledge.
    - Sexual history: Frequency, dyspareunia, erectile dysfunction, premature/retrograde ejaculation.
    - Past medical/surgical history (e.g., diabetes, hypertension, tuberculosis).
    - Occupation, substance abuse (smoking, alcohol).
  - **Female-Specific (PCOS):**
    - Menstrual history: Age at menarche, cycle length, flow, dysmenorrhea.
    - Onset/duration of androgenic features (hirsutism, acne, alopecia).
    - Hoarseness of voice, weight gain, sleep disturbances, galactorrhea, headache.
    - Obstetric history, prior infertility treatments.[20]
- **Examination:**
  - **General:**
    - Height, weight, BMI (obese: ≥30 kg/m^2).
    - Waist circumference (>80 cm indicates visceral fat), waist-hip ratio (>0.8).
    - Blood pressure, thyroid palpation, pallor.

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- Acne, hirsutism (mFG score ≥4–8), androgenic alopecia, acanthosis nigricans [shown in **Fig. 3**].
- Signs of other disorders (e.g., Cushing's: purple striae, buffalo hump).

- **Pelvic:**
  - Tanner staging, clitoral enlargement, uterine size, adnexal masses.[21]



Fig. 3: Acanthosis nigricans.

- **Investigations (Table 6):**
  - **Blood:** Complete blood count, fasting blood sugar/oral glucose tolerance test (OGTT), thyroid profile, prolactin, testosterone, FAI.
  - **Semen Analysis:** Mandatory for male partner.
  - **Ultrasound:** Day 2 TVS for uterus, ovaries, endometrial thickness, antral follicular count (AFC). Follicular monitoring (day 9–18) for ovulation.
  - **Tubal Patency:** Hysterosalpingography (HSG) on day 8–9 if tubal pathology suspected (e.g., history of pelvic inflammatory disease).[22]
  - **Preconception Risk:** Assess blood pressure, OGTT, lipid profile for IGT, T2DM, dyslipidemia, hypertension.[23]

TABLE 6: Investigations for infertility in PCOS.[22]

Test	*Purpose*	*Timing*
OGTT	Detect IGT/T2DM	Preconception
Thyroid/Prolactin	Rule out endocrine causes	Day 2–3
TVS	Assess PCOM, ovulation	Day 2, 9–18
Semen analysis	Male factor	Anytime
HSG	Tubal patency	Day 8–9

Treatment of Infertility in PCOS

Management addresses anovulation, metabolic risks, and long-term complications, guided by ESHRE 2023, RCOG, and NICE NG197 (2023).[2,24]

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- **Lifestyle Modification** (First-Line, Nonpharmacological):
  - **Diet:** Energy deficit of 500–750 kcal/day (1,200–1,500 kcal/day), low sugar/fat.
  - **Weight Loss:** 5–10% loss improves ovulation (30–40%), reduces visceral fat (30% with 10% loss). BMI <30 kg/m^2 targeted.[25]
  - **Exercise:** 150 min/week moderate or 75 min/week vigorous activity, plus muscle-strengthening twice weekly. 60–90 min/day for weight loss.[26]
  - **Folic Acid:** 400 µg/day (5 mg/day if obese) preconception.[27]
- **Ovulation Induction** (First-Line, Pharmacological) **(Tables 6 and 7)**:
  - **Letrozole** (Preferred):
    - Aromatase inhibitor; 2.5–7.5 mg/day, day 3–7 of cycle.
    - Mechanism: Reduces estrogen, increases FSH/LH, promotes follicular growth.
    - Advantages: Higher ovulation (70–85%), pregnancy (20–30%), and live birth rates versus clomiphene; no antiestrogenic effects on endometrium/cervix.
    - Side Effects: Headache, hot flashes, fatigue.[28]
  - **Clomiphene Citrate (CC):**
    - Selective estrogen receptor modulator; 50–150 mg/day, day 3–7.
    - *Mechanism:* Blocks estrogen receptors, increases FSH/LH.
    - *Advantages:* Ovulation in 70–85%, pregnancy in 60–70%.
    - *Disadvantages:* Multiple pregnancy risk (7–10%), ovarian hyperstimulation syndrome (OHSS), antiestrogenic effects.
    - *Side Effects:* Hot flashes, mood swings, visual disturbances.[29]
  - **Monitoring:** TVS follicular monitoring (day 9–18); hCG trigger (5,000–10,000 IU) when follicle ≥17 mm.[30]

TABLE 7: Comparison of Letrozole versus Clomiphene citrate.[28,29]

Parameter	*Letrozole*	*Clomiphene citrate*
Class	Aromatase inhibitor	Selective estrogen receptor modulator
Dose	2.5–7.5 mg/day	50–150 mg/day
Ovulation rate	70–85%	70–85%
Pregnancy rate	20–30%	15–20%
Multiple pregnancy	Low (1–2%)	High (7–10%)
Side effects	Headache, fatigue	Hot flashes, visual disturbances

- **Second-Line Pharmacological Treatment:**
  - **Gonadotrophins:**
    - Low-dose FSH (37.5–75 IU/day) step-up protocol for monofollicular development.
    - *Success rate:* 65–70% pregnancy in 6 months.
    - *Risks:* OHSS (5–10%), multiple pregnancy (20–30%). Cancel cycle if ≥2 follicles >14 mm.
    - *Indication:* CC resistance.[31]
  - **Metformin:**
    - 500–2,500 mg/day (TDS or 850 mg BD).

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- Improves insulin sensitivity, ovulation (30–40%), and pregnancy rates in CC-resistant cases.
- Less effective than letrozole/CC; no follicular monitoring needed.
- NICE NG197: CC, metformin, or combination for WHO Group II anovulation.[24]

- **Laparoscopic Ovarian Drilling (LOD):** Shown in **Figure 4**
  - Second-line for CC-resistant anovulation.
  - *Technique:* 4 punctures/ovary, 40W, 4 mm deep, 4 seconds each.
  - *Benefits:* Ovulation (73%), pregnancy (72%) within 2 years; reduced LH/androgens, lower OHSS/multiple pregnancy risk.
  - *Risks:* Adhesions, reduced ovarian reserve.[32]

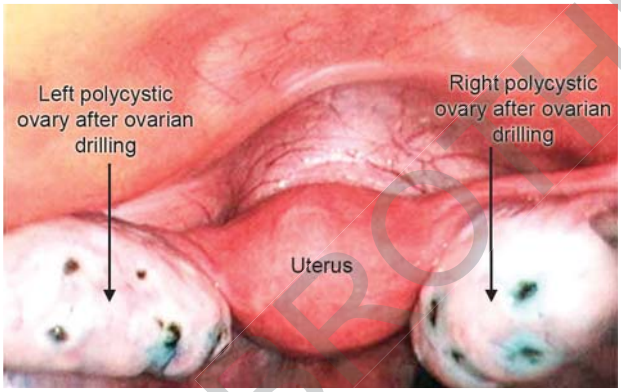

Fig. 4: Completion of laparoscopic ovarian drilling.

- **In Vitro Fertilization (IVF):**
  - *Indication:* Failure of ovulation induction (6 cycles if <25 years, 12 cycles if ≥25 years), or additional factors (e.g., tubal/male infertility).
  - *Protocol:* GnRH antagonist with GnRH agonist trigger preferred (lower OHSS risk vs. hCG trigger). Frozen embryo transfer improves pregnancy rates.
  - *Outcomes:* No increased risk of miscarriage, preterm birth, or fetal growth restriction versus non-PCOS.[33]
  - **Adjunct Metformin:** Improves ovulation, reduces early miscarriage, but limited evidence for routine use. Concerns for childhood obesity with in utero exposure.[34]
- **Other Therapies:**
  - **Antiobesity Drugs:** Orlistat, liraglutide discontinued preconception.
  - **Bariatric Surgery:** For BMI ≥35 kg/m^2 or 30–34.9 kg/m^2 with comorbidities. Delay pregnancy 1 year post surgery to avoid prematurity, small for gestational age (SGA).[35]
- **Risks of Ovulation Induction:**
  - **Multiple Pregnancy:** Increased perinatal mortality, maternal risks (e.g., preeclampsia).
  - **OHSS:** PCOS patients at high risk (5–10%). Symptoms: Nausea, ascites, pleural effusion. Management: Fluid balance, colloids, heparin for thromboembolism prevention.[36]

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SHORT QUESTIONS

Q1. Long-Term Complications and Management of PCOS. *(DNB 2023, Paper 3, 5 Marks)*

Long-Term Complications

PCOS increases risks of metabolic, reproductive, and psychological complications due to hyperandrogenism and insulin resistance **(Table 8)**.[37]

TABLE 8: Long-term complications of PCOS.[37]

Category	*Complications*	*Prevalence*
Anovulation	Infertility, endometrial cancer	Infertility: 70–80%; Cancer: 2–6-fold risk
Hyperandrogenism	Acne, hirsutism, androgenic alopecia, psychological distress	Hirsutism: 60–70%; Acne: 15–30%
Insulin Resistance	IGT, T2DM, gestational diabetes, metabolic syndrome	IGT: 35%; T2DM: 7–10%
Metabolic	Hypertension, dyslipidemia, cardiovascular disease	Hypertension: 20–30%
Sleep	Obstructive sleep apnea (OSA)	5–10%
Psychological	Depression, anxiety, eating disorders	Depression: 30–40%
Pregnancy	Miscarriage, gestational hypertension, preeclampsia, preterm delivery, SGA	Miscarriage: 20–40%

- **Anovulation**:
  - **Endometrial Cancer:** Unopposed estrogen increases risk 2–6-fold. Risk factors: Prolonged amenorrhea, abnormal uterine bleeding (AUB), obesity.[38]
- **Insulin Resistance:**
  - IGT (35%), T2DM (7–10%), gestational diabetes (20–30%).
  - Metabolic syndrome: Hypertension, dyslipidemia, central obesity.[39]
- **Cardiovascular:**
  - Increased risk of hypertension (20–30%), dyslipidemia, and stroke.[40]
- **Obstructive Sleep Apnea (OSA):**
  - 5–10% prevalence; symptoms: Snoring, daytime fatigue. Exacerbated by androgens/insulin resistance.[41]
- **Psychological:**
  - Depression (30–40%), anxiety, body image issues due to hirsutism, infertility, obesity.[42]
- **Pregnancy:**
  - Miscarriage (20–40%), gestational hypertension, preeclampsia, preterm delivery, SGA.[43]

Management

- **Endometrial Protection:**
  - Induce ≥4 menses/year with progestins (e.g., medroxyprogesterone 10 mg for 12 days) or combined oral contraceptives (COCs).

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  - Levonorgestrel intrauterine device (LNG-IUD) for endometrial protection.
  - TVS/endometrial biopsy for AUB or endometrial thickness >7 mm.[38]
- **Metabolic Screening:**
  - **OGTT:** At diagnosis, every 1–3 years (annually if BMI ≥25 kg/m^2, family history of T2DM, GDM, or age >40 years).
  - **Lipid Profile/BP:** At diagnosis, annually if abnormal or high risk.[24]
  - **Metformin:** 500–2,500 mg/day for IGT/T2DM, improves insulin sensitivity.[44]
- **OSA Screening:** Berlin Questionnaire for snoring, fatigue; refer for polysomnography if positive.[41]
- **Psychological Support:**
  - Cognitive behavioral therapy (CBT) first-line.
  - Lifestyle and cosmetic interventions (e.g., laser hair removal) improve quality of life.
  - Antidepressants/antianxiety medications for moderate-to-severe cases.[42]
- **Pregnancy Monitoring:**
  - Early OGTT, BP monitoring at antenatal visits to detect gestational diabetes/hypertension.[43]

Q2. Clinical Features, Evaluation, and Management of Hyperandrogenism/Hirsutism

(DNB 2023, Paper 3, 5 Marks)



Fig. 5: Features of hirsutism.

Clinical Features of Hyperandrogenism

- **Hirsutism:** Excessive terminal hair in male-pattern areas (60–70% of PCOS women) as shown in **Figure 5**.
- **Acne:** Inflammatory lesions, often severe (15–30%).
- **Androgenic Alopecia:** Male-pattern hair loss (5–10%).
- **Anovulation/Oligomenorrhea:** Due to ovarian androgen-induced follicular atresia (70–80%).
- **Virilization (Rare):** Deepening voice, clitoromegaly, temporal balding; suggests tumors or congenital adrenal hyperplasia (CAH) (45).

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Causes of Hirsutism

- PCOS (70–80%).
- Nonclassical CAH (raised 17α-hydroxyprogesterone).
- Androgen-secreting tumors (ovary/adrenal).
- Cushing's syndrome, acromegaly, severe insulin resistance.
- *Drug-induced:* Testosterone, danazol, phenytoin, minoxidil (46).

Clinical Evaluation

- **History:** Onset, progression, AUB, weight gain, virilization symptoms, drug use.
- **Examination:**
  - mFG score (9 areas, score 0–4; ≥4–8 indicates hirsutism).
  - *Acne:* Count active lesions/scars (no validated scale).
  - *Alopecia:* Ludwig/Olsen scales **(Fig. 6)**.
  - Signs of CAH (e.g., hyperpigmentation), Cushing's (striae), or tumors (rapid virilization).[47]
- **Biochemical:**
  - Total/free testosterone, FAI; androstenedione, DHEAS if normal testosterone.
  - 17α-hydroxyprogesterone for CAH (>200 ng/dL or ACTH stimulation test).
  - Stop COCs 3 months prior for accurate androgen assessment.[48]
- **Imaging:** TVS for PCOM; adrenal/ovarian imaging if tumor suspected.[49]

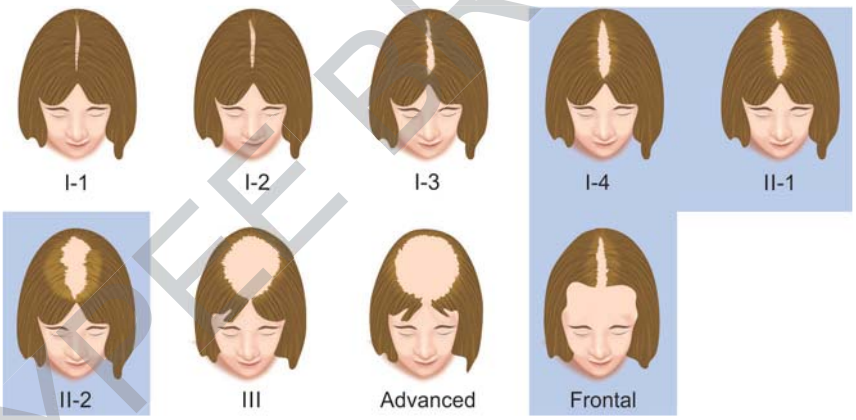

Fig. 6: *Placeholder:* Ludwig's classification of female hair loss.[47]

Management of Hirsutism

- **Lifestyle:** 5–10% weight loss reduces hirsutism by 50% in obese patients.[25]
- **Cosmetic:**
  - Photoepilation (laser): 30% hair reduction.
  - Electrolysis: Effective but painful, 18–24 months.
  - Waxing, shaving, depilatory creams.[50]
- **Pharmacological:**
  - **COCs** (First-Line): Reduce LH, increase SHBG, decrease free testosterone. Effect in 9–12 months (50% hirsutism reduction).

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- Preferred: 20–30 µg ethinyl estradiol with desogestrel, norgestimate, or drospirenone (lower DVT risk).
- Avoid 35 µg ethinyl estradiol + cyproterone acetate as first-line due to DVT risk (51) as shown in **Figure 7**.

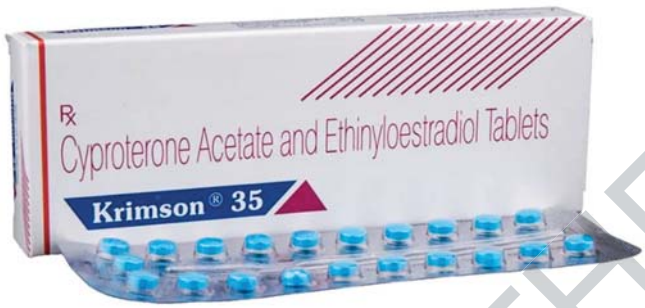

Fig. 7: OCP having ethinyl estradiol and cyproterone acetate.

- **Antiandrogens** (After 6 Months of COCs if Suboptimal):
  - Spironolactone (25–100 mg/day): Low side effects, requires contraception.
  - Cyproterone acetate, finasteride: Effective but higher risks (e.g., hepatotoxicity with flutamide).
  - Eflornithine cream: Topical, reduces hair growth, may cause rash.[52]
- **Metformin:** Reduces androgens by 20%, useful in obese/nonobese patients.[44]

Q3. Recent Guidelines in Management of PCOS.

(DNB 2023, Paper 3; UP 2015, Paper 4, 5 Marks)

ESHRE 2023, RCOG, and NICE NG197 Guidelines

Management of PCOS is multidisciplinary, addressing anovulation, hyperandrogenism, metabolic risks, and psychological effects.[2,24]

- **Lifestyle Management:**
  - **Diet:** Energy deficit (500–750 kcal/day) for weight loss (BMI <30 kg/m^2).
  - **Exercise:**
    - *Prevention:* 150–300 min/week moderate or 75–150 min/week vigorous activity, plus muscle training twice weekly.
    - *Weight loss:* 250 min/week moderate or 150 min/week vigorous activity.[26]
  - **Behavioral Support:** SMART goals (Specific, Measurable, Achievable, Realistic, Timely).[25]
- **Pharmacotherapy (Table 9):**
  - **COCs** (First-Line for Hirsutism/Irregular Cycles):
    - 20–30 µg ethinyl estradiol with third-/fourth-generation progestins (e.g., drospirenone).
    - Cyproterone acetate for severe hirsutism/acne (not first-line).
    - Progestin-only pills or LNG-IUD for endometrial protection.[51]

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TABLE 9: Pharmacotherapy for PCOS.[2,24]

Drug	*Indication*	*Dose*	*Effect*
COCs	Hirsutism, irregular cycles	20–30 µg ethinyl estradiol	Reduces androgens, cycle control
Metformin	IGT, T2DM, BMI ≥25 kg/m^2	500–2,500 mg/day	Improves insulin sensitivity
Spironolactone	Hirsutism (second-line)	25–100 mg/day	Reduces androgens
Myo-Inositol	Metabolic (experimental)	4,000 mg/day	Limited benefit

- **Metformin:**
  - 500–2,500 mg/day for IGT, T2DM, or BMI ≥25 kg/m^2.
  - Improves insulin resistance, androgens; less effective for hirsutism versus COCs.[44]
- **Antiobesity Drugs:** Liraglutide, orlistat, semaglutide (discontinue preconception).[35]
- **Antiandrogens:** Spironolactone, finasteride after 6 months of COCs; contraception mandatory.[52]
- **Inositol:** Myo-inositol (4,000 mg/day) experimental for metabolic benefits; metformin preferred for hirsutism/obesity.[53]

- **Cosmetic Interventions:**
  - Laser/photoepilation, electrolysis for hirsutism; combined with COCs/metformin improves psychological outcomes.[50]
- **Bariatric Surgery:**
  - Indication: BMI ≥35 kg/m^2 or 30–34.9 kg/m^2 with comorbidities.
  - Improves ovulation, hirsutism, metabolic profile; delay pregnancy 1 year.[35]
- **Psychological Interventions:**
  - CBT for depression/anxiety; lifestyle/cosmetic measures enhance quality of life.[42]

Q4. Place of Ovarian Surgery for PCOS. *(DNB 2023, Paper 3, 5 Marks)*

Ovarian surgery is not first-line for PCOS but is a second-line option for clomiphene-resistant anovulatory infertility **(Table 10)**.[32]

- **Ovarian Wedge Resection:**
  - Historical technique; reduces androgens and restores ovulation (85% pregnancy rate initially reported).
  - Disadvantages: High risk of adhesions, premature ovarian failure, infertility.
  - Obsolete due to safer alternatives.[54]
- **Laparoscopic Ovarian Drilling (LOD):**
  - **Technique:** 4 punctures/ovary, 40W, 4 mm deep, 4 seconds each (Rule of 4).
  - **Benefits:**
    - Ovulation rate: 73%; pregnancy rate: 72% within 2 years.
    - Reduces LH, androgens, OHSS, multiple pregnancy risk.
    - Sustained hirsutism improvement (9 years).
  - **Complications:**
    - Adhesions, periadnexal cysts, reduced anti-Müllerian hormone (AMH), ovarian reserve loss.
    - Higher risks in obese patients, requires expertise.[55]

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- **Indications:** CC-resistant anovulation, no other infertility factors.

TABLE 10: Comparison of ovarian surgery techniques.[54,55]

Technique	*Ovulation rate*	*Pregnancy rate*	*Complications*
Wedge Resection	70–80%	50–85%	Adhesions, ovarian failure
LOD	73%	72%	Adhesions, reduced AMH

Q5. Role of Inositol in PCOS Management.

(DNB 2021, Paper 4, 3 Marks; DNB 2016, 3 + 3 + 4 Marks)

What are Inositols?

Inositols are polyalcohols acting as second messengers in insulin and FSH signaling. Two isomers are relevant: [53]

- **Myo-Inositol (MI):** Most abundant, enhances glucose uptake, FSH-mediated granulosa cell proliferation, and aromatase synthesis (reduces androgens) **(Table 11)**.
- **D-Chiro-Inositol (DCI):** Converted from MI under insulin stimulation; improves insulin sensitivity.
- **MI:DCI Ratio:** Normal women: 100:1; PCOS: 0.2:1, indicating impaired conversion.

TABLE 11: Clinical effects of Myo-Inositol in PCOS.[57]

Effect	*Improvement*	*Evidence level*
Insulin Resistance	30–40%	Moderate
Ovulation	50–60%	Limited
Androgens	20–30%	Limited
IVF Outcomes	Better oocyte quality	Emerging

Physiological Role

- **Insulin Signaling:** MI/DCI improve glucose uptake, reduce insulin resistance.
- **Ovarian Function:** MI enhances folliculogenesis, reduces androgen production.
- **Metabolic Health:** Lowers cardiovascular risk by improving lipid profile.[56]

Clinical Utility

- **Effects:**
  - Improves insulin resistance (30–40%), androgen levels (20–30%), ovulation frequency (50–60%).
  - Enhances egg/embryo quality, reduces FSH dose/days in IVF.
  - Regularizes cycles, accelerates weight loss.[57]
- **Dosage:** 4,000 mg/day (MI, or MI + DCI) in two divided doses.
- **Sources:** Fruits, nuts, beans, whole wheat.
- **Side Effects:** Safe up to 10 weeks; rare diarrhea, nausea.
- **ESHRE 2023:** Experimental therapy; limited evidence for ovulation, hirsutism, or weight loss. Metformin preferred for hirsutism/obesity.[2]
- **IVF:** Reduces stimulation dose, improves oocyte quality, but more studies needed.[58]

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Guidelines

- **RCOG Green-top Guideline No. 33 (2023):** Recommends lifestyle modification as first-line, letrozole over CC for ovulation induction, and annual metabolic screening (OGTT, lipids, BP).[59]
- **NICE NG197 (2023):** Advocates CC, metformin, or combination for WHO Group II anovulation; LOD for CC-resistant cases; and psychological support for quality of life.[24]
- **ESHRE 2023:** Endorses Rotterdam criteria, restricts ultrasound in adolescents, and considers inositol experimental.[2]

Key Points

- PCOS affects 10–13% of women, diagnosed by Rotterdam criteria (2/3: anovulation, hyperandrogenism, PCOM).
- Adolescent diagnosis requires irregular cycles and hyperandrogenism; ultrasound avoided <8 years post-menarche.
- Pathophysiology involves hyperinsulinemia, hyperandrogenism, and gonadotropin dysregulation.
- Infertility management: Lifestyle (5–10% weight loss), letrozole (first-line), gonadotrophins/LOD (second-line), IVF (third-line).
- Long-term complications: Endometrial cancer, T2DM, cardiovascular disease, psychological distress.
- Hirsutism management: COCs, anti-androgens (second-line), cosmetic therapies.
- Inositol: Experimental, improves metabolic parameters but limited for ovulation/hirsutism.
- RCOG/NICE/ESHRE emphasize lifestyle, metabolic screening, and patient-centered care.

Monitoring of Antiandrogen Therapy

Monitoring response to treatment can be done objectively using Ferriman-Gallwey system, scoring at baseline and, if possible, at each visit.

Treatment can be continued as long as the patient desires and should be discontinued if planning for pregnancy.

Monitoring testosterone levels after instituting pharmacologic therapy is generally unnecessary because hirsutism scores correlate poorly with serum androgen levels.

Other useful pharmacologic therapies can be advised based on symptoms related to hyperandrogenism like:

- Antibiotics, topical retinoids, or isotretinoin for acne
- Minoxidil topical solution to be applied on area of desired hair growth. Max: 2 mL/day.
- Eflornithine for hirsutism

  Topical eflornithine slows growth of unwanted facial hair in 4–8 weeks. Inhibits hair growth (potentially by inhibiting ornithine decarboxylase)
- Bicalutamide (≤50 mg daily)

  There is emerging data to support use with oral contraceptives in PCOS.[8]

Nonpharmacologic cosmetic therapies for hirsutism include shaving, depilating, hair bleaching, electrolysis, and laser hair removal.

Imeglimin used in diabetes is in trial phase.

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Drugs to Aid Weight Loss in PCOS[14]

Orlistat

It assists in weight loss by inhibiting intestinal fat absorption (inhibits gastric and pancreatic lipases). There are various studies in PCOS obese patients with good results.

Bupropion/Naltrexone

It is a new antiobesity medication which suppresses appetite. Its use and safety in PCOS needs further studies.

Glucagon-like peptide-1 (GLP-1) receptor agonists

This new group of medication promotes weight loss in PCOS. Liraglutide has been shown to be superior to placebo, as well as to metformin and orlistat in head-to head comparisons. The efficacy of Liraglutide in promoting weight loss in PCOS patients is proven.

Medical management may be needed for other associated problems in PCOS:[15]

- **Dyslipidemia:** Approximately 70% of patients with newly diagnosed disease have abnormal lipid levels, including increased total cholesterol level, high triglyceride levels, high LDL-C level, and decreased HDL-C level.
- **Hypertension:** Women with PCOS have a risk of developing hypertension which if untreated may accelerate atherosclerotic cardiovascular disease
- Nonalcoholic fatty liver disease is frequently seen in patients with polycystic ovary syndrome, likely as a result of insulin resistance as well as obesity
- Depression and anxiety
  Increased incidence and prevalence over lifetime

Special Populations

Adolescents

Diagnosis can be difficult in adolescents owing to overlap between normal pubertal development and characteristic features of polycystic ovary syndrome. **Suggested criteria include demonstration of chemical and/or biochemical hyperandrogenism and presence of persistent oligomenorrhea for at least 2 years after menarche.**[16]

Hormonal contraceptives: In early adolescence, using hormonal contraceptives is controversial. Ideal hormonal contraceptive regimen and appropriate duration of therapy for adolescents are uncertain. **The standard practice is to wait for 2 years after menarche to prevent reduction in peak bone mass.**[3]

Metformin therapy: Small, short-term studies have found that metformin restores menstrual regularity and improves hyperandrogenemia, insulin resistance, and glucose intolerance in obese and nonobese adolescents with PCOS.

Postmenopausal Women

After menopause, two of the key diagnostic criteria of PCOS (irregular menses and polycystic ovaries on transvaginal ultrasonography) are no longer applicable.

Most women who had polycystic ovary syndrome during their reproductive years continue to manifest both metabolic phenotype and unfavorable cardiovascular risk factors. Androgen levels are still higher than in postmenopausal women without history of PCOS.

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It is advisable to maintain vigilance for monitoring lipid levels, blood pressure, and glycemia in accordance with standards of care in this population of women.

CONCLUSION

PCOS is a heterogeneous endocrinopathy affecting adolescent girls. The exact etiology is unknown; however, hyperinsulinemia and hyperandrogenism form the main pathological basis of this syndrome. In adolescents, it may be difficult to differentiate irregular cycles of PCOS from physiologic anovulation. However, persistence of menstrual irregularity for >2 years post menarche is a strong predictor of long-term ovulatory dysfunction. COC pills are the first line of medical management for menstrual disturbances and acne, metformin for weight reduction and dysglycemia, and a combination of both in addition to other treatment modalities for treatment of hirsutism.

Clinical Case Scenario

Case: A 28-year-old woman presents with irregular menses (4–5 cycles/year), hirsutism, and BMI 32 kg/m^2.
USG: Polycystic ovaries.
Labs: Elevated testosterone, fasting insulin 20 μIU/mL.

Management

- **Diagnosis:** PCOS (Rotterdam: Oligo-anovulation, hyperandrogenism, polycystic ovaries).
- **Counseling:** Explain PCOS, lifestyle benefits, and fertility implications.
- **Treatment:**
  - *Lifestyle:* 5–10% weight loss, low-GI diet, 150 min/week exercise.
  - *Inositol:* MI 2 g + DCI 50 mg twice daily for 3 months.
  - *Metformin:* Start 500 mg daily, titrate to 1500 mg/day.
- **Follow-Up:** Reassess menses, insulin, lipids at 3 months.
- **Outcome: Menses regularize in 4 months; BMI drops to 30 kg/m^2; ovulation confirmed.**
  Exam Tip: Emphasize lifestyle as first-line, MI/DCI 40:1 ratio, and metformin's metabolic role.

Key Points

- **Prevalence in India:** 20–26%; obesity-driven, high IR burden.
- **Inositol:** MI/DCI (40:1) improves IR, ovulation, lipids; GI side effects at high doses.
- **Metformin:** Enhances insulin sensitivity, ovulation; less effective than clomiphene for fertility.
- **Thiazolidinediones:** Limited use due to safety concerns in pregnancy.
- **Guidelines:** Lifestyle first; inositol/metformin for IR; clomiphene for ovulation induction.

BOX 1: Important points to remember.

- *Rotterdam Criteria:* 2/3 (oligo-anovulation, hyperandrogenism, polycystic ovaries)
- *MI/DCI Ratio:* 40:1 optimal; excess DCI worsens androgens
- *Metformin:* Start 500 mg/day, titrate to 1,500–2,000 mg; monitor GFR annually
- *Lifestyle:* 5–10% weight loss restores ovulation in 60% of obese PCOS patients

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