SIR Publishers
CENTER FOR INTERNATIONAL SCIENTIFIC PUBLICATIONS
JOURNAL OF NURSING AND WOMEN'S HEALTH
ISSN(Online): 2984-8083
Volume-9, Issue-03, Published |18-06-2026|

POLYCYSTIC OVARY SYNDROME, ENDOSCOPIC GYNECOLOGIC METHODS AND HORMONAL DIAGNOSIS OF INFERTILITY: AN INTERNATIONAL IMRAD REVIEW

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ABSTRACT

Background: PCOS and infertility are common reproductive-health problems with endocrine, metabolic and psychosocial consequences. Objective: This article reviews PCOS, endoscopic gynecologic investigation and hormonal diagnosis of infertility. Methods: A narrative review was performed using WHO fact sheets and international evidence on reproductive disorders. Results: PCOS affects about 10-13% of women globally and is frequently undiagnosed; infertility affects about 17.5% of adults during their lifetime. Hormonal assessment and endoscopic methods answer different diagnostic questions. Discussion: The main clinical risk is fragmented evaluation that treats hormones, anatomy and patient goals separately. Conclusion: Infertility assessment should integrate ovulation, tubal-uterine anatomy, metabolic risk and individualized counseling.

Keywords: polycystic ovary syndrome; infertility; hormonal diagnosis; laparoscopy; hysteroscopy; anovulation

INTRODUCTION

Polycystic ovary syndrome is not just an ovarian ultrasound finding. It is a heterogeneous endocrine-metabolic condition that can involve irregular cycles, hyperandrogenism, anovulation, insulin resistance, infertility and psychological distress. The name itself can mislead students, because ovarian cyst appearance is neither the whole disease nor always required depending on diagnostic criteria.

Infertility is also frequently misunderstood. It is not a single diagnosis, and it is not automatically a female problem. WHO data indicate that infertility affects about 17.5% of adults during their lifetime, which makes it a common health condition rather than a rare specialist issue. For women, causes may include ovulatory dysfunction, tubal disease, uterine pathology, endometriosis and age-related factors, often in combination.

Endoscopic gynecologic methods, especially hysteroscopy and laparoscopy, provide anatomical information that hormones cannot provide. Hormonal tests can suggest ovulation status, ovarian reserve, thyroid or prolactin disorders and androgen excess. Endoscopy can evaluate uterine cavity pathology, pelvic adhesions, endometriosis and tubal-related questions. The two approaches are complementary.

This solo-authored article reviews PCOS, endoscopic diagnostic and treatment methods in gynecology, and hormonal diagnosis of infertility using an international IMRAD structure.

METHODS

A narrative review was conducted using international public-health sources on PCOS, infertility, endometriosis and sexually transmitted infections, along with standard gynecologic

diagnostic principles. The review focused on clinical reasoning rather than detailed operative technique.

The analysis was organized into three domains: endocrine diagnosis of PCOS and anovulation, anatomical assessment through endoscopic methods, and integrated infertility evaluation. Statistical indicators were included to demonstrate public-health relevance.

This article does not recommend self-treatment or provide individualized fertility protocols. Clinical evaluation must be performed by qualified professionals with appropriate laboratory and imaging support.

RESULTS

PCOS diagnosis usually requires a combination of clinical and laboratory reasoning. Common criteria include ovulatory dysfunction, clinical or biochemical hyperandrogenism and polycystic ovarian morphology after exclusion of related disorders. The most important point for students is that PCOS is a diagnosis of pattern plus exclusion, not a diagnosis based on one ultrasound image.

Hormonal evaluation may include LH, FSH, total or free testosterone, DHEAS, prolactin, thyroid-stimulating hormone, progesterone for ovulation evidence and metabolic markers such as glucose and lipid assessment. The exact panel depends on symptoms and local protocols. Testing should be timed and interpreted carefully because random results can confuse rather than clarify.

Infertility evaluation begins with duration, age, cycle pattern, pregnancy history, sexual history, prior pelvic infection, surgery, pain symptoms and male-factor assessment. Ignoring male-factor infertility is a common error. Even when PCOS is present, it may not be the only cause of infertility in a couple.

Endoscopic methods answer anatomical questions. Hysteroscopy allows direct visualization of the uterine cavity and may identify polyps, submucosal fibroids, adhesions or septal abnormalities. Laparoscopy allows assessment of pelvic anatomy, endometriosis, adhesions and sometimes tubal patency procedures depending on resources and indication.

Endoscopic treatment can be beneficial when a correct target exists, but unnecessary procedures create cost and risk. For example, surgical management of endometriosis or adhesions may help selected patients, while indiscriminate laparoscopy without clear indication is weak practice. The decision must be individualized.

The statistical burden is substantial. WHO estimates PCOS affects about 10-13% of women globally, and up to 70% may remain undiagnosed. Infertility affects about one in six adults. Endometriosis affects about 10% of reproductive-age women. These figures show that cycle irregularity, hyperandrogenism and fertility concerns deserve structured evaluation, not casual reassurance.

Case-based result 1: A patient has irregular cycles and high androgen symptoms, but thyroid and prolactin testing have not been done. The case shows why PCOS is a diagnosis of pattern plus exclusion, not a shortcut label.

Case-based result 2: A patient with PCOS receives ovulation-focused care, but the couple has no male-factor assessment. Pregnancy does not occur. This illustrates the danger of treating visible female findings while ignoring couple-based infertility.

Case-based result 3: A patient has pelvic pain and infertility with normal basic hormones. Endometriosis or adhesions may still be relevant. Hormonal normality does not exclude anatomical pathology.

Case-based result 4: Ultrasound shows polycystic ovarian morphology in a young patient with otherwise regular cycles and no hyperandrogenism. This finding alone should not create a PCOS diagnosis. Morphology is not the whole syndrome.

These cases show that infertility evaluation should be staged but integrated. The clinician should not jump immediately to invasive procedures, but should also not rely forever on hormonal tests when anatomy is the unresolved question.

The practical result is an integrated infertility map: ovulation, semen analysis, tubal-pelvic anatomy, uterine cavity, endocrine-metabolic status and patient goals. PCOS fits into this map but does not replace it.

Table 1. Statistical indicators used for clinical interpretation

Indicator	Reported value	Interpretation in this article
PCOS prevalence	about 10-13% of women globally	Major endocrine disorder in reproductive age.
PCOS undiagnosed proportion	up to 70%	Delayed diagnosis is common.
Infertility lifetime prevalence	about 17.5% of adults	Shows the scale of fertility-care need.
Endometriosis burden	about 190 million reproductive-age women	Important differential in infertility and pelvic pain.
Curable STIs in 2020	about 374 million new infections	Untreated infection can contribute to tubal-factor infertility.

Table 2. Practical clinical matrix for student use

Clinical question	Most useful first assessment	Escalation trigger
Is anovulation present?	Cycle history and progesterone evidence	Persistent irregular cycles or infertility
Is anatomy involved?	Ultrasound, hysteroscopy or laparoscopy if indicated	Pain, adhesions, cavity lesion suspicion
Is male factor assessed?	Couple-based infertility evaluation	No pregnancy despite corrected ovulation

DISCUSSION

The first failure mode is diagnosing PCOS too quickly. Irregular cycles and acne do not automatically equal PCOS. Thyroid disease, hyperprolactinemia, non-classic congenital adrenal hyperplasia, androgen-secreting tumors and medication effects may mimic parts of the syndrome. Exclusion is part of diagnosis.

The second failure mode is diagnosing PCOS too slowly. Because symptoms may be normalized, many patients spend years without explanation. Delayed diagnosis can affect fertility planning, metabolic risk reduction and psychological well-being. A structured history can shorten this delay.

The third failure mode is separating infertility into hormone-only or anatomy-only pathways. Ovulation can be restored while tubal disease remains unnoticed. A normal uterine cavity does not prove ovulation. A male-factor problem can coexist with PCOS. Integrated evaluation prevents narrow treatment.

Endoscopy presents a clear tradeoff. It can directly diagnose and sometimes treat pathology, but it is invasive and requires equipment, anesthesia capacity and trained staff. Therefore, endoscopy should follow a question that cannot be answered adequately by safer methods, or a situation where treatment is likely during the same procedure.

PCOS also has long-term metabolic implications. Insulin resistance, weight changes and dyslipidemia may influence cardiovascular and diabetes risk. A fertility-only approach misses the chronic-health dimension of the syndrome.

Patient communication is difficult because PCOS affects appearance, menstruation, fertility and emotional well-being. Students should avoid reducing the condition to weight or blaming the patient. Clear explanation improves adherence and trust.

A practical PCOS assessment should include menstrual pattern, androgen symptoms, body-mass and metabolic markers, but it should also include patient priorities. Some patients seek fertility, others seek cycle control, acne treatment or long-term risk counseling. The management aim changes with the priority.

The edge case is lean PCOS. Students often associate PCOS only with obesity, but lean patients can also have hyperandrogenism and ovulatory dysfunction. Stereotypes delay diagnosis.

Another edge case is ultrasound overdiagnosis in adolescents or young women. Multifollicular ovarian appearance can occur without PCOS. Diagnosis should not be based on morphology alone.

The tradeoff in infertility evaluation is invasiveness. Hormone tests and ultrasound are less invasive than laparoscopy, but they may not detect peritoneal endometriosis or adhesions. The decision depends on symptoms, duration, age and previous findings.

PCOS assessment should not ignore psychological impact. Irregular bleeding, acne, unwanted hair growth and fertility anxiety can affect self-confidence. A clinician who discusses only laboratory values may miss the patient's main concern.

Lifestyle counseling must be careful. It should support metabolic health without blaming the patient or reducing PCOS to weight. Overly simplistic advice can damage trust and reinforce stigma.

Hormonal diagnosis of infertility should be staged. Initial tests may confirm ovulation or identify thyroid, prolactin or androgen problems. More advanced ovarian reserve or reproductive technology evaluation depends on age, history and specialist access.

Endoscopic methods should be justified by a clear question. Hysteroscopy is strongest for uterine cavity questions; laparoscopy is strongest for pelvic anatomy, endometriosis and adhesions. Using the wrong method wastes resources.

PCOS follow-up should include metabolic health even when the immediate complaint is infertility. Blood pressure, glucose risk and lipid profile may affect long-term outcomes and pregnancy planning. A narrow ovulation-only approach is incomplete.

Endoscopic treatment decisions should consider patient age, symptom severity, previous treatment, fertility duration and available expertise. The same finding may justify different choices in different patients.

Counseling should also address expectations. Infertility evaluation often proceeds step by step, and one normal test does not mean the whole system is normal. Patients need a roadmap to avoid frustration.

The final added result is that PCOS and infertility should be taught as overlapping but not identical topics. PCOS can be one cause of infertility, but fertility evaluation remains broader and couple-based.

Tubal-factor infertility is strongly connected with prior infection and pelvic inflammation. Sexual-health history is therefore part of fertility assessment, even when the presenting complaint is irregular menstruation.

Age is a major variable in infertility decisions. The same duration of infertility may require different urgency in younger and older patients. Evaluation should be individualized rather than automatic.

The student should learn to avoid single-cause thinking. PCOS may be present, but male factor, tubal disease or uterine pathology can coexist. Fertility care fails when it stops after the first abnormality.

The final result is an integrated diagnostic sequence: confirm the couple's goal, assess ovulation, assess semen, evaluate uterus and tubes when indicated, screen metabolic risks and choose intervention only after the main barriers are identified.

Male-factor assessment is a fairness issue as well as a clinical issue. Blaming the woman without evaluating the couple is scientifically weak and ethically harmful.

Metabolic screening in PCOS should not be forgotten when the consultation focuses on fertility. Pregnancy planning is safer when glucose, weight-related risk and blood pressure are considered before conception.

Students should be taught to distinguish ovarian morphology, ovarian function and ovarian reserve. These terms are often confused, yet they answer different clinical questions.

A future local study could assess how often women with irregular cycles receive thyroid, prolactin and androgen evaluation before being labeled with PCOS. This would identify diagnostic shortcuts.

Endoscopic findings must be interpreted carefully. Mild adhesions, small endometriotic lesions or incidental cysts may not fully explain infertility. The presence of an abnormality does not automatically prove causation.

A locally relevant research direction would be to assess time from first menstrual irregularity to PCOS diagnosis among students and young women. Such data could reveal underdiagnosis and guide education campaigns.

The limitation of this review is its broad scope. PCOS management, ovulation induction, assisted reproductive technology and endoscopic surgery each deserve separate detailed articles. Here, the goal is to build a student-level map of how these domains connect.

The smallest practical improvement is a three-column infertility worksheet: ovulation evidence, uterine-tubal-pelvic anatomy, and male-factor assessment. PCOS belongs mainly in the ovulation and metabolic column, but the other columns must not disappear.

CONCLUSION

PCOS is a common, frequently undiagnosed endocrine-metabolic condition and a major contributor to anovulatory infertility. Infertility itself is common and multifactorial.

Hormonal diagnostics and endoscopic methods should be integrated rather than used as competing approaches. Good evaluation asks the right question, chooses the least risky useful test and interprets results in the context of the patient and couple.

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