



**GYNECOLOGICAL SYNDROMES AND REPRODUCTIVE HEALTH: DIAGNOSTIC
APPROACHES TO MENSTRUAL DISORDERS, PELVIC PAIN, AND INFERTILITY**

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Abstract

Background. Menstrual disorders, pelvic pain, and infertility are among the most clinically important presentations encountered in gynecology and reproductive medicine. Although commonly approached as separate problems, they frequently coexist and may arise from shared structural, endocrine, inflammatory, or functional mechanisms. Endometriosis, adenomyosis, polycystic ovary syndrome (PCOS), pelvic inflammatory disease (PID), uterine structural disease, and disorders of the hypothalamic-pituitary-ovarian axis can produce overlapping combinations of abnormal bleeding, pain, ovulatory dysfunction, and impaired fertility. Objective. To critically analyze contemporary diagnostic approaches to menstrual disorders, pelvic pain, and infertility and to develop an integrated framework for evaluating women with overlapping reproductive-health presentations. Methods. A structured narrative review of current clinical guidelines, systematic reviews, meta-analyses, authoritative reviews, and relevant primary studies was undertaken. PubMed/MEDLINE and publicly accessible Cochrane Library records were searched, together with official guidance from the World Health Organization (WHO), International Federation of Gynecology and Obstetrics (FIGO), European Society of Human Reproduction and Embryology (ESHRE), American Society for Reproductive Medicine (ASRM), American College of Obstetricians and Gynecologists (ACOG), National Institute for Health and Care Excellence (NICE), and other professional organizations. Recent evidence was prioritized while retaining clinically important landmark classifications. Direct authenticated searches of Scopus and Web of Science were not available and are not claimed. Results. Contemporary diagnostic practice favors syndrome-directed but integrated evaluation. Pregnancy and urgent pathology must first be excluded. Abnormal uterine bleeding should be classified using FIGO terminology and PALM-COEIN etiologic categories. Amenorrhea requires systematic assessment of pregnancy status and hypothalamic, pituitary, ovarian, thyroid, and anatomical causes. Chronic pelvic pain requires consideration of gynecological and non-gynecological pain generators. Endometriosis is increasingly evaluated with clinical assessment and high-quality imaging rather than routine diagnostic laparoscopy. Infertility assessment should evaluate ovulation, ovarian reserve when clinically relevant, uterine and tubal factors, and the male partner in parallel. Conclusion. Menstrual dysfunction, pelvic pain, and infertility are diagnostically interconnected. A stepwise, individualized approach based on clinical phenotype, reproductive goals, targeted laboratory testing,



appropriate imaging, and selective invasive assessment is preferable to indiscriminate testing.

Keywords: abnormal uterine bleeding; amenorrhea; dysmenorrhea; chronic pelvic pain; endometriosis; polycystic ovary syndrome; infertility; ovarian reserve; reproductive health; diagnostic algorithm.

Introduction

Menstrual abnormalities, pelvic pain, and infertility represent overlapping clinical domains rather than completely independent gynecological syndromes. Their diagnostic importance extends beyond symptom control because each may signal an underlying disorder with consequences for reproductive potential, sexual health, quality of life, psychological well-being, anemia, endocrine health, pregnancy planning, or long-term gynecological morbidity. Infertility itself is recognized by the World Health Organization (WHO) as a disease of the male or female reproductive system, defined by failure to achieve pregnancy after 12 months or more of regular unprotected sexual intercourse [1,2]. WHO estimates indicate that approximately one in six people of reproductive age experience infertility during their lifetime [2,3].

The apparent simplicity of individual symptoms can be misleading. Heavy or irregular bleeding may reflect uterine structural pathology, ovulatory dysfunction, systemic disease, medication exposure, or endometrial abnormalities. Amenorrhea may originate from pregnancy, functional hypothalamic suppression, hyperprolactinemia, thyroid disease, PCOS, primary ovarian insufficiency (POI), or anatomical abnormalities. Dysmenorrhea may be primary, but persistent, progressive, or treatment-resistant pain raises the possibility of endometriosis, adenomyosis, uterine pathology, or congenital reproductive-tract anomalies [4-12].

Pelvic pain introduces further diagnostic complexity because symptom location does not reliably identify the affected organ. Gynecological causes coexist with gastrointestinal, urinary, musculoskeletal, neurological, and pelvic-floor disorders, particularly in chronic pelvic pain (CPP). Modern pain models also recognize that chronic symptoms can be maintained by peripheral pathology, pelvic-floor dysfunction, altered nociceptive processing, and central sensitization simultaneously [19-21].

The clinical overlap is particularly evident in endometriosis. A patient may initially report severe dysmenorrhea, later develop non-cyclic CPP or deep dyspareunia, and



subsequently present with infertility. Similarly, PCOS may manifest as oligomenorrhea or amenorrhea before fertility is attempted. Adenomyosis can present with heavy menstrual bleeding, dysmenorrhea, chronic pelvic discomfort, and reproductive impairment. PID may begin with acute or subacute pain but later become clinically relevant because of tubal damage and infertility [11-24].

Diagnostic strategies based exclusively on the dominant symptom therefore risk fragmentation. A woman evaluated only for menstrual irregularity may have fertility-relevant anovulation; a patient treated repeatedly for dysmenorrhea may have endometriosis; an infertility investigation focused solely on ovarian reserve can overlook tubal, uterine, endometriotic, or male factors. A unified diagnostic model is therefore clinically useful because it connects symptom patterns to underlying pathophysiology while reducing both delayed diagnosis and unnecessary testing.

The aim of this review is to critically analyze contemporary diagnostic approaches to menstrual disorders, pelvic pain, and infertility and to identify clinically useful strategies for integrated assessment of women with reproductive-health disorders.

Materials and Methods

This article was designed as an evidence-based narrative review rather than a systematic review or original clinical study. No new patient cohort, experimental dataset, or statistical analysis was generated. A structured literature search was undertaken in September 2026. PubMed/MEDLINE and publicly accessible Cochrane Library records were searched alongside official guideline repositories and professional-society publications. Priority organizations included WHO, FIGO, ESHRE, ASRM, ACOG, NICE, the Endocrine Society, and the Centers for Disease Control and Prevention (CDC).

Search concepts included combinations of: menstrual disorders, abnormal uterine bleeding, PALM-COEIN, amenorrhea, oligomenorrhea, ovulatory dysfunction, dysmenorrhea, chronic pelvic pain, endometriosis, adenomyosis, polycystic ovary syndrome, pelvic inflammatory disease, female infertility, infertility diagnosis, ovarian reserve, anti-Müllerian hormone, tubal patency, hysterosalpingography, hysterosalpingo-contrast sonography, and reproductive health.

Publications were prioritized if they were recent clinical practice guidelines, consensus statements, systematic reviews, meta-analyses, or clinically relevant high-



quality primary investigations. Literature from approximately 2020-2026 was preferentially selected, while earlier publications were retained when they represented major classification systems or still-current guidance. Peer-reviewed human research directly addressing diagnostic evaluation was favored. Exclusion criteria included non-scientific sources, duplicate publications, superseded recommendations when more current guidance was available, poorly relevant studies, and material with insufficient bibliographic verification.

Direct authenticated searches of Scopus and Web of Science were not available in the present environment; therefore, these databases are not represented as having been independently searched. No formal PRISMA screening flow, meta-analysis, quantitative evidence synthesis, or study-level risk-of-bias assessment was performed. Accordingly, the manuscript should be interpreted as a structured narrative synthesis rather than a systematic review.

[Menstrual Disorders: Classification and Diagnostic Evaluation](#)

[Abnormal Uterine Bleeding](#)

Abnormal uterine bleeding (AUB) describes uterine bleeding that differs from expected menstruation in frequency, regularity, duration, or volume. In reproductive-aged, non-pregnant patients, FIGO terminology provides a standardized framework for describing the bleeding pattern and classifying possible causes [4].

The FIGO PALM-COEIN system separates structural from predominantly non-structural etiologies. The PALM group comprises Polyp, Adenomyosis, Leiomyoma, and Malignancy/hyperplasia. The COEIN group comprises Coagulopathy, Ovulatory dysfunction, Endometrial causes, Iatrogenic causes, and conditions Not otherwise classified [4]. The classification is etiologic rather than mutually exclusive; more than one abnormality can coexist in an individual patient.

Diagnostic assessment begins with characterization of the bleeding phenotype. History should establish timing, regularity, duration, perceived volume, intermenstrual or postcoital bleeding, medication exposure, contraceptive use, pregnancy possibility, symptoms of anemia, bleeding tendency, endocrine symptoms, and personal risk factors for endometrial pathology. Pregnancy testing is



fundamental in reproductive-aged patients because pregnancy-related bleeding is not classified through PALM-COEIN.

A complete blood count is appropriate when bleeding is clinically significant. Iron status, particularly ferritin, should be considered in heavy or prolonged bleeding or when iron deficiency is suspected; clinically important uterine bleeding may cause iron deficiency before or in association with overt anemia [6]. Routine broad coagulation testing is not justified for every patient, but targeted evaluation is appropriate when the history suggests an inherited or acquired bleeding disorder.

Endocrine testing should be driven by phenotype rather than routinely ordered as a broad panel. TSH, prolactin, androgen assessment, or other endocrine studies may be appropriate when ovulatory dysfunction, thyroid disease, hyperprolactinemia, PCOS, or another endocrine disorder is suspected.

Imaging strategy depends on the clinical question. Transvaginal ultrasonography is generally the first-line method for assessing the uterus and adnexa and can identify leiomyomas, features suggestive of adenomyosis, endometrial abnormalities, and adnexal disease. When a focal intracavitary abnormality such as an endometrial polyp or submucosal leiomyoma is suspected, hysteroscopy or saline-infusion sonography may provide superior cavity assessment. NICE recommends selecting ultrasound or hysteroscopy according to history and examination and recommends outpatient hysteroscopy when submucosal fibroids, polyps, or endometrial pathology are suspected [7].

Endometrial sampling should not be triggered solely by the presence of bleeding. The threshold depends on age, bleeding pattern, persistence, response to treatment, and risk of endometrial hyperplasia or malignancy. Risk factors such as chronic anovulation, obesity, PCOS, persistent intermenstrual or irregular bleeding, tamoxifen exposure, or failure of appropriate therapy may lower the threshold for histological evaluation. In postmenopausal bleeding, the diagnostic paradigm differs substantially because exclusion of endometrial malignancy becomes a primary concern.

Adolescent AUB requires particular attention to anovulatory cycles, pregnancy where relevant, and inherited bleeding disorders. In reproductive age, pregnancy-related causes, PALM-COEIN conditions, endocrine disease, and contraceptive or

medication effects must be distinguished. Perimenopausal patients have increasing prevalence of structural lesions and endometrial pathology, making age- and risk-adjusted evaluation essential.

Amenorrhea

Amenorrhea should be approached anatomically and endocrinologically rather than as a single diagnosis. Current ASRM guidance recommends evaluation of primary amenorrhea when menstruation has not occurred by age 15 despite otherwise normal secondary sexual development, with earlier assessment when pubertal development is abnormal or when an anatomical disorder is suspected [9].

The first diagnostic step in secondary amenorrhea is exclusion of pregnancy. Subsequent evaluation should integrate the previous menstrual pattern, timing of symptom onset, weight change, caloric intake, exercise load, psychosocial stress, medication exposure, galactorrhea, headache or visual symptoms, vasomotor symptoms, hirsutism, acne, virilization, chronic systemic disease, and reproductive history.

Physical examination should assess body habitus, secondary sexual characteristics, signs of androgen excess, thyroid abnormalities, galactorrhea where relevant, and features suggesting congenital or acquired reproductive-tract abnormalities. Initial laboratory assessment commonly includes TSH and prolactin, together with FSH and estradiol when ovarian or central dysfunction is being considered [9]. LH may assist interpretation in selected contexts but should not be treated as an obligatory screening test for every patient. Androgen testing is appropriate in the presence of clinical hyperandrogenism. Pelvic ultrasonography provides information on uterine anatomy, endometrial development, ovarian morphology, and potential congenital abnormalities.

Functional hypothalamic amenorrhea (FHA) is a diagnosis of exclusion and commonly occurs in association with low energy availability, weight loss, excessive exercise, physiological or psychological stress, or combinations of these factors [10]. It should not be diagnosed simply because a patient is lean or physically active; pregnancy, thyroid dysfunction, hyperprolactinemia, PCOS, POI, and structural disease must first be considered.



POI should be suspected when amenorrhea or marked menstrual irregularity occurs in a younger woman together with biochemical evidence of ovarian insufficiency. Contemporary POI guidance emphasizes appropriate biochemical confirmation and etiologic evaluation rather than assuming irreversible ovarian failure from a single abnormal hormone measurement [12]. Hyperprolactinemia requires confirmation and assessment of medication, physiological, endocrine, and pituitary causes. Thyroid dysfunction may disrupt menstrual cyclicity through multiple endocrine pathways. Anatomical causes should be considered particularly in primary amenorrhea or when normal endocrine function coexists with absent or abnormal menstrual outflow.

Oligomenorrhea and Ovulatory Dysfunction

Oligomenorrhea is clinically important because menstrual irregularity is often a practical marker of abnormal ovulatory function. FIGO's contemporary ovulatory-disorder framework groups disorders according to hypothalamic, pituitary, ovarian, and PCOS-related mechanisms, emphasizing that anovulation is an etiologically heterogeneous state [8].

PCOS is one of the major causes of chronic ovulatory dysfunction. In adults, the 2023 International Evidence-based PCOS Guideline retains a diagnostic framework based on two of three features after exclusion of alternative disorders: clinical or biochemical hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology [11]. In appropriately selected adults, anti-Müllerian hormone (AMH) may be used as an alternative means of defining polycystic ovarian morphology within the diagnostic algorithm, but it is not a standalone diagnostic test for PCOS. Diagnostic criteria in adolescents require additional caution because normal pubertal physiology may mimic elements of adult PCOS.

The reproductive implication of persistent anovulation is not limited to infertility. Chronic unopposed estrogen exposure in some anovulatory states may increase risk of endometrial hyperplasia. Conversely, hypothalamic hypogonadism may produce low estrogen exposure with implications for bone health. Identifying the underlying mechanism therefore matters more than simply documenting irregular menstruation.

Dysmenorrhea

Primary dysmenorrhea refers to painful menstruation in the absence of identifiable pelvic pathology, whereas secondary dysmenorrhea results from an underlying disorder. The distinction is principally clinical.

Features that increase suspicion of secondary disease include new or progressive pain, onset after years of painless menstruation, pain extending substantially beyond menstruation, deep dyspareunia, infertility, abnormal bleeding, pelvic mass, abnormal examination, congenital anomaly, poor response to appropriate first-line therapy, or associated bowel and urinary symptoms.

Endometriosis is a central consideration, particularly when dysmenorrhea coexists with chronic pelvic pain, deep dyspareunia, cyclical bowel or bladder symptoms, or infertility. Adenomyosis, leiomyomas, PID, and congenital reproductive-tract abnormalities should also be considered according to age and clinical context [13,14,23,24].

Pelvic Pain: Clinical Evaluation and Differential Diagnosis

Pelvic pain should first be classified by temporal profile and clinical urgency. Acute pelvic pain may reflect ectopic pregnancy, ovarian torsion, hemorrhagic ovarian pathology, PID, appendicitis, urinary disease, or other urgent conditions. Cyclical pain raises suspicion of hormonally responsive gynecological disease, particularly endometriosis or adenomyosis. CPP requires a broader framework because multiple pain generators commonly coexist [19-21].

History

A clinically useful pain history includes onset, duration, location, severity, radiation, cyclicity, precipitating and relieving factors, and relationship to menstruation, sexual intercourse, urination, defecation, physical activity, posture, or food intake.

Pregnancy possibility, previous pelvic infection, surgery, endometriosis, obstetric trauma, fertility history, contraception, and medication use should be assessed.

The clinician should also ask about dyschezia, urinary urgency or bladder pain, altered bowel habit, dyspareunia, vulvovaginal symptoms, low-back or hip pain, and symptoms suggesting neuropathic pain. Psychosocial factors should be evaluated as potential contributors to symptom burden and pain processing without implying that pain is psychogenic.

Physical Examination

The examination should be hypothesis-driven. General and abdominal assessment may identify hemodynamic compromise, peritoneal irritation, abdominal masses, scars, hernias, or musculoskeletal tenderness. Pelvic examination may assess discharge, cervical motion tenderness, uterine size or mobility, adnexal masses, focal tenderness, nodularity, and pelvic-floor muscle tenderness when clinically appropriate.

A normal examination does not exclude endometriosis or other clinically significant disorders. Conversely, tenderness does not establish a unique etiology.

Major Gynecological Causes

Endometriosis, adenomyosis, PID, ovarian cysts, ovarian torsion, ectopic pregnancy, leiomyomas, and postoperative or inflammatory adhesions are important gynecological considerations. Acute pain with pregnancy or possible pregnancy requires urgent evaluation for ectopic pregnancy. Sudden severe unilateral pain with nausea or vomiting warrants consideration of ovarian torsion. Fever, cervical motion or adnexal tenderness, abnormal discharge, and relevant sexual-risk history may support PID, although no single clinical finding provides complete diagnostic certainty [22].

Non-Gynecological Differential Diagnosis

Chronic or recurrent pelvic pain may originate from the gastrointestinal tract, urinary system, musculoskeletal structures, pelvic floor, abdominal wall, peripheral nerves, or central pain mechanisms. Irritable bowel syndrome, inflammatory bowel disease, bladder pain syndrome, urinary infection or stones, pelvic-floor myalgia, abdominal-wall pain, hip pathology, neuropathic pain, and nociplastic pain states should be considered according to clinical presentation [20,21].

This wider differential is particularly important when symptoms persist despite apparently successful treatment of a gynecological lesion.

Endometriosis

Endometriosis deserves particular attention because it links menstrual pain, CPP, dyspareunia, and infertility. Contemporary guidance has shifted away from the historical assumption that laparoscopy is mandatory before a clinical diagnosis can be established. ESHRE recommends imaging as part of the diagnostic work-up and



recognizes that negative imaging does not exclude superficial peritoneal disease [13]. NICE's 2024 update recommends offering transvaginal ultrasonography to people with suspected endometriosis even when pelvic or abdominal examination is normal [14].

High-quality transvaginal ultrasound can identify ovarian endometriomas and many forms of deep endometriosis. Dedicated MRI is particularly useful when mapping deep disease, evaluating sites that are difficult to assess sonographically, or clarifying complex anatomy. Diagnostic performance depends strongly on protocol and operator expertise [15,16].

No blood, urine, menstrual, or other biomarker is sufficiently validated for routine standalone diagnosis of endometriosis. Contemporary biomarker research remains active, but systematic review evidence continues to show substantial heterogeneity and absence of a clinically validated universal marker [17].

Laparoscopy remains important when imaging is non-diagnostic but suspicion remains clinically significant, when empirical management is unsuccessful or inappropriate, or when surgery is required for another therapeutic reason. It should not automatically be used as the first diagnostic step in every symptomatic patient [13,14].

Female Infertility: Contemporary Diagnostic Approach

WHO defines infertility as a disease of the male or female reproductive system characterized by failure to achieve pregnancy after 12 months or more of regular unprotected sexual intercourse [1,2]. Diagnostic timing should nevertheless be individualized. ASRM recommends earlier evaluation when female age is higher or when a condition associated with impaired fertility is already known or suspected [25].

Infertility assessment should be systematic, expeditious, and minimally invasive where possible. It should not be structured as an exhaustive sequence in which one partner is evaluated completely before the other. Male and female factors may coexist, making parallel assessment more efficient [25,29,30].



Initial Clinical Assessment

Relevant history includes age, duration of attempted conception, menstrual cyclicity, prior pregnancies and their outcomes, previous infertility evaluation or treatment, sexual frequency and timing, contraception, pelvic infection, sexually transmitted infections, surgery, endometriosis, systemic disease, medications, chemotherapy or radiotherapy exposure, family reproductive history, and relevant lifestyle factors.

Age remains a major determinant of reproductive potential and influences the urgency with which evaluation should proceed. Menstrual regularity provides useful information regarding ovulation, although regular bleeding alone does not exclude all ovulatory abnormalities.

Ovulatory Assessment

A clearly regular menstrual pattern often provides useful evidence of ovulatory function. Additional biochemical confirmation is not necessarily required in every patient with predictable cycles. When ovulation is uncertain, appropriately timed serum progesterone may support evidence of recent ovulation, although interpretation requires correct timing relative to the individual's cycle rather than use of a universal calendar day.

Oligomenorrhea or amenorrhea requires etiologic assessment rather than repeated confirmation that ovulation is absent. PCOS, thyroid disease, hyperprolactinemia, functional hypothalamic dysfunction, and POI are among the principal considerations [9-12].

Ovarian Reserve

Ovarian reserve testing estimates aspects of remaining oocyte quantity or expected ovarian response; it does not directly measure spontaneous conception probability or oocyte quality. AMH and antral follicle count (AFC) are commonly used markers of ovarian reserve, while basal FSH has more limited sensitivity and greater cycle-to-cycle variability. Their principal value is in predicting response to ovarian stimulation and supporting treatment planning [26].

A low AMH should not be interpreted as proof that natural conception is impossible, while a high AMH does not guarantee natural fertility. Female age, diagnosis, tubal status, semen parameters, sexual factors, and other clinical variables remain



essential. ASRM specifically cautions against treating ovarian reserve tests as independent fertility tests in women without an infertility indication [26].

Tubal Patency

Tubal disease should be assessed when clinically relevant because tubal obstruction or damage can prevent sperm-oocyte interaction and may increase the risk of ectopic pregnancy. Hysterosalpingography (HSG) provides information about the uterine cavity and tubal patency but involves ionizing radiation and iodinated contrast. Hysterosalpingo-contrast sonography (HyCoSy) provides ultrasound-based assessment without ionizing radiation and can be integrated with pelvic sonography. Selection between methods depends on local expertise, availability, contraindications, and the broader diagnostic question [27,28].

Laparoscopy with chromopertubation is invasive and is not required as the routine first-line tubal test for every patient. It becomes more relevant when endometriosis, adhesions, or other pelvic pathology is strongly suspected or when operative assessment is otherwise justified [25,27].

Uterine Evaluation

Transvaginal ultrasonography is the principal first-line method for evaluation of uterine and adnexal anatomy. Saline infusion sonography can improve delineation of intracavitary lesions. Hysteroscopy permits direct visualization and potentially treatment of intrauterine pathology but should be used according to indication rather than as universal screening.

Fibroids, endometrial polyps, congenital Müllerian anomalies, intrauterine adhesions, and adenomyosis may affect reproductive outcomes in selected patients. MRI is generally reserved for complex structural abnormalities, detailed mapping, or inconclusive ultrasound rather than routine infertility screening.

Endometriosis-Associated Infertility

Endometriosis-associated infertility is multifactorial. Mechanisms may include distorted tubo-ovarian anatomy, pelvic adhesions, inflammatory changes, endometriomas, and effects on ovarian reserve in selected patients.

Ovarian endometriomas create a particularly complex diagnostic-management interface because both the disease and ovarian surgery may influence ovarian

reserve. AMH or AFC may provide clinically relevant baseline information in fertility planning, but these markers cannot determine whether a specific endometrioma is the direct cause of infertility [13,26].

The decision to proceed to surgery, expectant management, or assisted reproduction is therefore not purely diagnostic and should incorporate age, pain, ovarian reserve, previous surgery, endometrioma characteristics, duration of infertility, tubal status, semen findings, and reproductive priorities.

Male Partner Assessment

Infertility is a couple-level clinical problem. Semen analysis should generally occur early and in parallel with female evaluation rather than after completion of extensive testing in the female partner [25,29]. WHO's sixth edition laboratory manual provides standardized methods for semen examination. An abnormal semen result requires appropriate clinical interpretation; conversely, a single apparently normal semen analysis does not establish fertility in isolation [29].

Integrated Diagnostic Approach to Menstrual Disorders, Pelvic Pain, and Infertility

An integrated approach begins by recognizing that the same disease can produce different syndromic presentations at different times. Endometriosis may first present as dysmenorrhea, later as non-cyclical pelvic pain or deep dyspareunia, and subsequently become relevant during infertility evaluation. A normal routine ultrasound does not fully exclude disease, particularly superficial endometriosis [13-17].

PCOS illustrates an endocrine-reproductive pathway. Oligomenorrhea or amenorrhea may represent chronic ovulatory dysfunction, while hyperandrogenism provides an additional diagnostic clue. Infertility becomes clinically apparent when pregnancy is attempted. An isolated ultrasound appearance resembling polycystic ovaries should not be mistaken for the syndrome itself [11].

Adenomyosis represents structural overlap. It may produce heavy menstrual bleeding, dysmenorrhea, chronic pelvic pain, uterine enlargement, and reproductive difficulty. Transvaginal ultrasound is a first-line diagnostic modality, with MRI useful when sonographic findings are uncertain or detailed mapping is required [23,24].



PID demonstrates an inflammatory pathway. Acute or subacute pelvic pain may be the presenting syndrome, but subsequent tubal scarring may become clinically apparent only during infertility evaluation. A remote history of infection therefore remains diagnostically relevant even when acute symptoms have resolved [22].

Leiomyomas may produce abnormal bleeding, bulk symptoms, pain, and reproductive impairment depending on size and location. Intracavitary or cavity-distorting lesions are generally more relevant to both bleeding and fertility than small asymptomatic non-cavity-distorting lesions.

The diagnostic objective is consequently not to order the largest number of tests but to identify the smallest rational set of investigations that can explain the patient's combined clinical phenotype. This requires iterative clinical reasoning: initial results should be interpreted against the pre-test probability of disease, and incidental findings should not automatically be assigned causality.

Diagnostic Algorithm

Step 1 — Exclude urgent conditions. Determine hemodynamic stability and assess for pregnancy. Urgent evaluation is required when ectopic pregnancy, ovarian torsion, severe acute hemorrhage, significant anemia with instability, tubo-ovarian abscess/severe PID, or another acute surgical condition is plausible.

Step 2 — Define the dominant and associated syndromes. Determine whether the presentation is primarily abnormal menstruation, amenorrhea/oligomenorrhea, pelvic pain, infertility, or a combination.

Step 3 — Perform focused history and examination. Characterize bleeding, pain phenotype, menstrual cyclicity, reproductive history, pregnancy possibility, endocrine symptoms, medication exposure, STI/PID history, previous surgery, infertility duration, and reproductive priorities.

Step 4 — Select basic investigations. Use pregnancy testing where relevant. Order CBC and iron studies when clinically indicated by bleeding. Use TSH, prolactin, FSH/estradiol, androgen studies, infection testing, or other laboratory studies according to phenotype rather than as indiscriminate panels.

Step 5 — Use first-line imaging appropriately. Transvaginal ultrasonography is the principal imaging method for most structural gynecological questions. Tailor the



examination when endometriosis, adenomyosis, uterine cavity pathology, or adnexal disease is suspected.

Step 6 — Perform syndrome-directed evaluation. Classify AUB using PALM-COEIN; investigate amenorrhea according to the hypothalamic-pituitary-ovarian-anatomical axis; assess CPP across organ systems; and evaluate infertility through ovulatory, uterine, tubal, and male factors.

Step 7 — Escalate selectively. Use hysteroscopy, saline infusion sonography, MRI, HSG/HyCoSy, endometrial sampling, or laparoscopy when the anticipated diagnostic benefit is sufficient to alter management.

Step 8 — Integrate findings. Avoid accepting the first abnormal result as necessarily causal. A fibroid, ovarian cyst, mild biochemical abnormality, or low AMH may coexist with another disorder that better explains the presentation.

Step 9 — Refer when complexity exceeds single-specialty evaluation. Reproductive endocrinology, endometriosis expertise, radiology, gastroenterology, urology, pelvic-floor physiotherapy, pain medicine, endocrinology, or other specialties may be appropriate.

Diagnostic Red Flags

Urgent or expedited assessment should be considered with a positive pregnancy test accompanied by pelvic pain or bleeding; hemodynamic instability; syncope; sudden severe unilateral pelvic pain, particularly with nausea or vomiting; acute abdomen or peritoneal signs; severe or persistent bleeding causing clinically significant anemia; postmenopausal bleeding; fever or systemic toxicity with pelvic pain; a suspicious or rapidly enlarging adnexal or pelvic mass; unexplained constitutional symptoms when malignancy is plausible; or progressive neurological or urinary/bowel compromise suggesting complex deep pelvic disease.

Tables

Table 1. Differential Diagnosis of Menstrual Disorders, Pelvic Pain, and Infertility

Clinical presentation	Major possible causes	Key diagnostic clues	Initial investigations	Further evaluation
Heavy/irregular uterine bleeding	Polyp, adenomyosis, leiomyoma, ovulatory dysfunction, coagulopathy, endometrial disorder, malignancy/hyperplasia	Bleeding pattern, age, medications, anemia, endocrine symptoms	Pregnancy test, CBC; targeted ferritin/endocrine tests; TVUS	Hysteroscopy, endometrial sampling, SIS according to risk
Amenorrhea	Pregnancy, FHA, PCOS, hyperprolactinemia, thyroid disease, POI, anatomical disorder	Weight/energy change, hyperandrogenism, galactorrhea, vasomotor symptoms, pubertal history	Pregnancy test, TSH, prolactin, FSH/estradiol; TVUS	Androgen testing, pituitary assessment, genetic/anatomical evaluation when indicated
Oligomenorrhea + hyperandrogenism	PCOS, nonclassic adrenal hyperplasia, androgen-secreting disorders, other endocrine disease	Hirsutism, acne, biochemical androgen excess	Pregnancy exclusion, androgen profile, targeted endocrine testing	PCOS diagnostic assessment; further endocrine evaluation if atypical



Clinical presentation	Major possible causes	Key diagnostic clues	Initial investigations	Further evaluation
Cyclic pelvic pain/dysmenorrhea	Primary dysmenorrhea, endometriosis, adenomyosis, obstructive anomaly	Relation to menstruation, dyspareunia, infertility, bowel/bladder symptoms	History, examination, TVUS	Specialist ultrasound, MRI or laparoscopy selectively
Acute unilateral pelvic pain	Torsion, cyst rupture/hemorrhage, ectopic pregnancy	Sudden onset, nausea/vomiting, pregnancy possibility	Pregnancy test, CBC as indicated, urgent ultrasound	Emergency gynecological/surgical evaluation
Pelvic pain + fever/discharge	PID, tubo-ovarian abscess, urinary or gastrointestinal infection	Sexual history, cervical motion/adnexal tenderness, fever	Pregnancy test, STI testing, CBC/CRP selectively, ultrasound	Inpatient/imaging escalation according to severity
Infertility + regular cycles	Tubal disease, endometriosis, uterine pathology, male factor, unexplained infertility	Prior PID/surgery, pain, uterine symptoms, partner history	Semen analysis, TVUS, tubal assessment as appropriate	HSG/HyCoSy, hysteroscopy, laparoscopy selectively
Infertility + irregular cycles	PCOS, FHA, thyroid disease, hyperprolactinemia, POI	Oligo-/amenorrhea, weight changes, endocrine symptoms	Hormonal evaluation, TVUS, semen analysis	Ovarian reserve or specialized endocrine work-up where indicated



Table 2. Diagnostic Tools in the Evaluation of Gynecological and Reproductive Disorders

Diagnostic method	Main indication	Strengths	Limitations	Clinical interpretation
Pregnancy test	Amenorrhea, abnormal bleeding, acute pelvic pain	Rapid, essential first step	Timing may affect very early detection	Pregnancy-related pathology must be excluded before non-pregnancy algorithms
CBC	Significant AUB, suspected anemia/infection	Widely available	Does not establish etiology	Quantifies hematological consequence rather than cause
Ferritin	Heavy/prolonged bleeding, suspected iron deficiency	Detects depleted iron stores	Acute inflammation may complicate interpretation	Use according to bleeding burden and anemia risk
TSH	Amenorrhea, irregular cycles, endocrine suspicion	Identifies thyroid dysfunction	Not specific to reproductive disorder	Interpret with clinical and thyroid context



Diagnostic method	Main indication	Strengths	Limitations	Clinical interpretation
Prolactin	Amenorrhea/oligomenorrhea, galactorrhea	Detects hyperprolactinemia	Stress, medications and macroprolactin may complicate results	Persistent elevation requires etiologic assessment
FSH/estradiol	Amenorrhea, suspected ovarian or central dysfunction	Helps distinguish endocrine patterns	Single measurements can be misleading	Interpret together and within clinical context
Androgen assessment	Hyperandrogenism, suspected PCOS or alternative endocrine disease	Identifies biochemical androgen excess	Assay variability; not required in every patient	Marked or rapid abnormalities require alternative-cause evaluation
AMH	Ovarian reserve/treatment planning; selected adult PCOS algorithms	Cycle-stable marker of ovarian follicle pool	Poor standalone predictor of natural conception	Do not equate low AMH with sterility



Diagnostic method	Main indication	Strengths	Limitations	Clinical interpretation
Transvaginal ultrasound	AUB, pelvic pain, infertility	First-line uterine/adnexal imaging	Operator and protocol dependent	Normal imaging does not exclude superficial endometriosis
MRI	Complex endometriosis, adenomyosis, congenital/structural disease	Excellent soft-tissue mapping	Cost, access, expertise	Usually second-line/problem-solving modality
HSG	Tubal patency, cavity outline	Widely established, assesses both tubes	Radiation, contrast, discomfort; false proximal occlusion possible	Useful first-line tubal test in selected infertility work-ups
HyCoSy/HyFoSy	Tubal patency	No ionizing radiation; can combine with ultrasound	Operator/contrast availability dependent	Alternative to HSG where expertise exists
Hysteroscopy	Suspected intracavitary lesion	Direct visualization and possible treatment	Invasive relative to ultrasound	Best used when cavity pathology is clinically relevant



Diagnostic method	Main indication	Strengths	Limitations	Clinical interpretation
Endometrial biopsy	Risk of hyperplasia/malignancy	Histological diagnosis	Invasive; focal lesions may be missed by blind sampling	Use according to age, bleeding pattern and risk
Laparoscopy	Selected endometriosis/tubal/pelvic pathology	Direct pelvic evaluation and possible treatment	Surgical risks and costs	No longer mandatory as first test for all suspected endometriosis

Table 3. Major Disorders Linking Menstrual Dysfunction, Pelvic Pain, and Infertility

Disorder	Menstrual manifestations	Pain manifestations	Reproductive consequences	Key diagnostic methods
Endometriosis	Menstruation may be normal; associated bleeding symptoms possible	Dysmenorrhea, CPP, dyspareunia, dyschezia	Infertility/subfertility, distorted anatomy, endometrioma-associated issues	Clinical assessment, expert TVUS, MRI selectively, laparoscopy selectively
Adenomyosis	Heavy or prolonged bleeding	Dysmenorrhea, pelvic tenderness/CPP	Possible impaired reproductive outcomes	TVUS; MRI when necessary



Disorder	Menstrual manifestations	Pain manifestations	Reproductive consequences	Key diagnostic methods
PCOS	Oligomenorrhea, amenorrhea, irregular bleeding	Pain is not a defining feature	Chronic anovulation and infertility	Clinical/biochemical hyperandrogenism, ovulatory assessment, ultrasound or AMH in appropriate adults
Leiomyoma	Heavy bleeding, irregular bleeding depending on location	Pressure, dysmenorrhea, pelvic discomfort	Effects depend strongly on cavity distortion, size and location	TVUS, SIS/hysteroscopy for cavity lesions, MRI selectively
PID/tubal disease	Menstrual pattern may be unaffected; intermenstrual bleeding may occur	Acute/subacute pelvic pain, cervical motion/adnexal tenderness	Tubal factor infertility, ectopic pregnancy risk	Clinical assessment, STI testing, ultrasound; later HSG/HyCoSy for tubal patency
Müllerian anomalies	Amenorrhea, obstructed flow or normal menses depending on anomaly	Cyclic pain with obstruction	Infertility, miscarriage or obstetric consequences depending on anomaly	Ultrasound, 3D ultrasound and MRI selectively

Discussion

Major Diagnostic Challenges

The principal challenge across menstrual disorders, pelvic pain, and infertility is phenotypic overlap. Symptoms do not map reliably onto single diseases. Abnormal bleeding may arise from both structural and endocrine causes; pelvic pain can reflect multiple organ systems; and infertility frequently involves more than one partner or pathophysiological mechanism.

Diagnostic tests likewise have context-dependent value. A normal pelvic ultrasound does not eliminate endometriosis. A low AMH does not establish infertility. Polycystic ovarian morphology alone does not diagnose PCOS. Conversely, identification of a uterine fibroid does not prove that it is responsible for bleeding, pain, or infertility. The clinically relevant question is therefore not simply whether an abnormality exists, but whether the abnormality plausibly explains the patient's phenotype and whether identifying it changes management.

Diagnostic Delay

Endometriosis illustrates the consequences of fragmented symptom-based care. Contemporary systematic literature continues to document substantial diagnostic delay, although estimates vary widely according to health system, study design, and the definition used. A 2025 systematic review of publications since 2018 reported times to diagnosis ranging approximately from 0.3 to 12 years [18].

Several mechanisms may contribute: normalization of severe menstrual pain, empirical treatment without reassessment, fragmented care across specialties, limited access to specialist imaging, and the historical expectation that surgical confirmation is always necessary. The move toward structured symptom recognition, expert ultrasound, selective MRI, and earlier specialist referral has the potential to shorten diagnostic pathways without indiscriminately increasing surgery [13-16].

Limitations of Biomarkers and Imaging

Biomarkers are attractive because they promise minimally invasive diagnosis, yet biological heterogeneity, menstrual-cycle effects, phenotype differences, laboratory variation, and inadequate external validation have limited translation into routine endometriosis diagnosis [17].



Imaging has improved substantially but remains dependent on disease phenotype, equipment, protocol, and expertise. Dedicated imaging for deep endometriosis performs differently from a routine pelvic scan. Similarly, ultrasound and MRI for adenomyosis have broadly comparable diagnostic utility in appropriately performed examinations, supporting transvaginal ultrasonography as an efficient first-line test while preserving MRI for selected cases [24].

Overdiagnosis and Unnecessary Testing

More testing does not necessarily improve diagnostic accuracy. Indiscriminate laboratory panels increase the probability of incidental abnormalities. Imaging may identify lesions unrelated to symptoms. Repeated invasive procedures may expose patients to cost, anxiety, procedural complications, and treatment directed at findings of uncertain clinical relevance.

This problem is especially important in infertility. Ovarian reserve testing used outside an appropriate clinical context can generate misleading conclusions about reproductive potential. AMH should therefore be interpreted within age, diagnosis, and treatment context rather than being used as a binary test of whether a person is fertile or infertile [26]. The same principle applies to laparoscopy. The current role of surgery in suspected endometriosis is more selective than in historical algorithms because high-quality imaging and clinical diagnosis can often guide initial management [13,14].

Role of Individualized Assessment

Diagnostic strategy should be modified according to age, reproductive plans, symptom severity, duration, risk factors, previous therapy, comorbidities, and prior surgery. A patient with severe dysmenorrhea who is not currently attempting pregnancy has different immediate diagnostic priorities from a patient with the same pain phenotype who has two years of infertility. Similarly, management of irregular menstruation differs between an adolescent shortly after menarche, an adult with hyperandrogenism, an endurance athlete with low energy availability, and a patient with vasomotor symptoms suggesting POI.

Individualization should not be confused with inconsistency. Standardized frameworks—PALM-COEIN for AUB, systematic amenorrhea assessment, current



PCOS criteria, and structured infertility evaluation—provide the foundation. Individual characteristics determine how those frameworks are applied.

Multidisciplinary Care

CPP is particularly suited to interdisciplinary assessment because gynecological pathology can coexist with bowel disorders, bladder pain, pelvic-floor myalgia, musculoskeletal pain, neuropathic mechanisms, or central sensitization [19-21]. Collaboration may involve gynecologists, reproductive-medicine specialists, endocrinologists, radiologists, gastroenterologists, urologists, pelvic-floor physiotherapists, pain specialists, and mental-health professionals. Multidisciplinary referral should be based on identified clinical needs rather than employed as a substitute for diagnostic reasoning.

Evidence Gaps

Several areas remain incompletely resolved. Endometriosis still lacks a validated non-invasive biomarker capable of reliably replacing clinical assessment and imaging. The optimal integration of AMH into adult PCOS diagnosis requires assay- and population-aware interpretation. The reproductive significance of adenomyosis varies across phenotypes and remains difficult to separate from coexisting disease. Chronic pelvic pain research remains complicated by heterogeneous definitions and overlapping pain mechanisms.

International guidelines also differ in healthcare context, resource availability, and procedural thresholds. These differences should be recognized rather than artificially converted into a single universal algorithm.

Practical Implications for Clinical Practice

1. Exclude pregnancy and urgent pathology before applying routine menstrual, pain, or infertility algorithms.
2. Use PALM-COEIN to organize the differential diagnosis of AUB, but remember that more than one cause may coexist.
3. In amenorrhea, begin with pregnancy exclusion and targeted assessment of thyroid, prolactin, ovarian, hypothalamic-pituitary, and anatomical causes rather than ordering unstructured hormone panels.



4. In suspected PCOS, diagnose the syndrome using accepted criteria and exclusion of mimicking disorders; do not diagnose PCOS from ovarian morphology or AMH alone.
5. Do not dismiss severe or progressive dysmenorrhea as normal menstruation when endometriosis or adenomyosis is clinically plausible.
6. In suspected endometriosis, use high-quality transvaginal ultrasonography early; reserve MRI and laparoscopy for appropriate clinical indications.
7. Evaluate CPP beyond gynecological organs and actively consider bowel, bladder, musculoskeletal, pelvic-floor, neuropathic, and nociplastic contributors.
8. Conduct infertility assessment in both partners in parallel. Female evaluation should address ovulation, uterine anatomy, tubal patency, and relevant ovarian factors while obtaining semen analysis early.
9. Interpret AMH and AFC primarily as ovarian-reserve and ovarian-response markers rather than direct measures of spontaneous fertility.
10. Escalate to hysteroscopy, endometrial sampling, advanced imaging, or laparoscopy only when the expected information is clinically meaningful and likely to alter management.

Limitations of the Review

This article is a narrative evidence synthesis rather than a systematic review. No PRISMA study-selection process, prospective protocol, quantitative meta-analysis, or formal risk-of-bias assessment was performed.

The evidence base is heterogeneous across menstrual, pain, and infertility disorders, and diagnostic recommendations vary between professional organizations and healthcare settings. Although PubMed/MEDLINE, publicly accessible Cochrane records, and authoritative guideline repositories were searched, direct authenticated searches of Scopus and Web of Science were not available. The review therefore cannot claim exhaustive retrieval of all potentially eligible literature indexed exclusively in those databases.



Recommendations in reproductive medicine continue to evolve. The manuscript reflects sources verified through September 2026 and should be checked against any guideline updates arising after that date before future publication.

Conclusion

Menstrual disorders, pelvic pain, and infertility are clinically interconnected manifestations of reproductive dysfunction. Evaluating them as isolated complaints can obscure diseases that cross traditional syndromic boundaries.

A structured diagnostic strategy should first exclude pregnancy and urgent pathology, then define the dominant clinical phenotype and associated symptoms. History and physical examination remain fundamental. Laboratory testing should be hypothesis-driven, while transvaginal ultrasonography is the principal first-line imaging method for many gynecological disorders.

Endometriosis, PCOS, adenomyosis, PID and tubal disease, leiomyomas, other uterine pathology, and endocrine disorders are particularly important because they can affect more than one of the three clinical domains. Modern diagnosis increasingly favors targeted non-invasive assessment before invasive procedures. Laparoscopy, hysteroscopy, endometrial biopsy, advanced imaging, and specialized endocrine or fertility investigations remain valuable when used for appropriate indications rather than as universal screening tools.

In infertility, evaluation of the female and male partners should proceed in parallel. Ovarian reserve markers must be interpreted according to their actual biological and clinical meaning and should not be used as standalone measures of natural fertility. An integrated, individualized diagnostic framework provides a more clinically coherent approach to reproductive-health disorders than isolated symptom-based investigation and may reduce both missed pathology and unnecessary testing.

Conflict of Interest

Conflict of interest: [To be confirmed by the author. If none, state: "The author declares no conflict of interest."]

Funding

Funding: [Specify funding source or "No external funding" after author confirmation].

Author Contributions

Author contributions: [To be confirmed by the author. For a single-author manuscript, list the roles actually performed in accordance with the target journal's authorship policy or CRediT taxonomy.]

Ethical Considerations

This manuscript is a review based exclusively on previously published literature and contains no new patient-level research. Ethical approval is generally not required for this type of work; the final statement should conform to the requirements of the selected journal and institution.

Data Availability

No new patient-level dataset was generated or analyzed for this narrative review. All evidence discussed derives from publicly available published literature and professional clinical guidance.

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