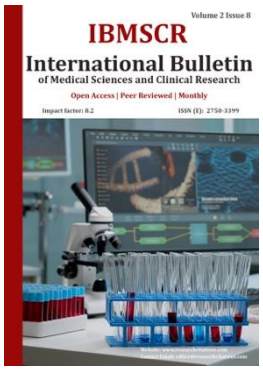

EARLY DETECTION AND MANAGEMENT OF MENSTRUAL AND OVARIAN DYSFUNCTION ASSOCIATED WITH THYROID DISORDERS IN REPRODUCTIVE-AGED WOMEN

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Abstract. Thyroid disorders are among the most prevalent endocrine conditions affecting women of reproductive age and are increasingly recognized as significant contributors to menstrual and ovarian dysfunction. Despite this, thyroid-related reproductive abnormalities remain underdiagnosed in gynecologic practice. Objective: This narrative review aims to synthesize current evidence on the pathophysiological mechanisms, clinical manifestations, early detection strategies, and management of menstrual and ovarian dysfunction associated with thyroid disorders in reproductive-aged women. Methods: A comprehensive narrative review was conducted using Scopus-indexed literature, including observational studies, clinical reviews, and international guidelines. Relevant studies examining the association between thyroid dysfunction and menstrual abnormalities, ovulatory disorders, ovarian reserve, and fertility outcomes were critically analyzed. Results: Thyroid dysfunction, particularly subclinical hypothyroidism, is highly prevalent among women presenting with abnormal uterine bleeding, with reported rates ranging from 20% to 44%. Menstrual irregularities may precede overt thyroid disease, highlighting their role as early clinical indicators. Thyroid dysfunction disrupts the hypothalamic–pituitary–ovarian axis, contributing to anovulation, altered ovarian reserve, and infertility. Additionally, thyroid autoimmunity is associated with premature ovarian insufficiency and adverse reproductive outcomes. Early screening using serum thyroid-stimulating hormone, particularly in women with menstrual disorders, infertility, or polycystic ovary syndrome, facilitates timely diagnosis. Restoration of euthyroidism through appropriate medical management improves menstrual regularity, ovulation, and fertility outcomes. Conclusion: Early detection through routine screening in high-risk populations and timely management is essential to improve reproductive health outcomes. Integration of thyroid evaluation into standard gynecologic assessment should be strongly considered.

Keywords: thyroid dysfunction, menstrual disorders, abnormal uterine bleeding, ovarian dysfunction, ovulatory disorders, female infertility, polycystic ovary syndrome, thyroid autoimmunity.

Introduction. Thyroid disorders are among the most common endocrine conditions affecting women of reproductive age and have substantial implications for menstrual function, ovulation, fertility, and pregnancy outcomes [1,2]. The reproductive consequences of thyroid dysfunction extend beyond overt disease; subclinical hypothyroidism, thyroid autoimmunity, and thyroid dysfunction associated with gynecologic comorbidities such as polycystic ovary syndrome (PCOS) may also influence the menstrual cycle and ovarian physiology [3-5]. Because thyroid disease is frequently underrecognized in gynecologic practice, reproductive symptoms may be treated symptomatically while the endocrine cause remains undiagnosed.

The relationship between thyroid status and female reproductive health is biologically plausible and clinically well established. Thyroid hormones influence the hypothalamic-pituitary-ovarian axis, sex hormone metabolism, prolactin secretion, ovarian follicular development, luteal function, and endometrial homeostasis [1,2,6]. Consequently, both hypothyroidism and hyperthyroidism may present with menstrual abnormalities, anovulation, subfertility, miscarriage, and adverse obstetric outcomes [1,2,7]. Importantly, menstrual disturbances may precede overt thyroid disease, making early detection especially relevant in reproductive-aged women presenting with abnormal uterine bleeding (AUB), oligomenorrhea, amenorrhea, or infertility [8].

Recent work has expanded this field in several directions. First, multiple observational studies from gynecology settings have shown a meaningful prevalence of thyroid dysfunction among women presenting with menstrual disorders or AUB, with subclinical hypothyroidism often emerging as the most frequent abnormality [9-13]. Second, growing evidence links thyroid dysfunction and thyroid autoimmunity with ovarian dysfunction, including ovulatory disorders, altered ovarian reserve, PCOS, and premature ovarian insufficiency [4,5,14-17]. Third, contemporary reproductive medicine guidelines emphasize thyroid screening and optimization before assisted reproduction, especially in women with thyroid autoimmunity or elevated TSH [18,19].

Given these developments, a high-quality narrative synthesis is needed to integrate pathophysiological mechanisms with clinical recognition and management. This review discusses the early detection and management of menstrual and ovarian dysfunction associated with thyroid disorders in reproductive-aged women, with emphasis on clinical suspicion, diagnostic evaluation, and evidence-informed management.

Thyroid-Reproductive Axis: Pathophysiological Basis. The interaction between thyroid function and female reproduction occurs through both central and peripheral mechanisms. Thyroid hormones modulate hypothalamic and pituitary signaling, influence gonadotropin secretion, and interact with prolactin and sex steroid metabolism [1,2,6]. In hypothyroidism, increased thyrotropin-releasing hormone may stimulate prolactin secretion, resulting in hyperprolactinemia, impaired gonadotropin-releasing hormone pulsatility, and anovulation [2,6]. At the same time, hypothyroidism alters estrogen metabolism, decreases sex hormone-binding globulin, and may disturb luteal function and endometrial maturation [1,2].

At the ovarian level, thyroid hormone receptors are expressed in reproductive tissues, supporting a direct role for thyroid hormones in folliculogenesis, steroidogenesis, and corpus luteum physiology [20,21]. Experimental and clinical data indicate that ovarian responsiveness may be altered in thyroid dysfunction, which helps explain menstrual abnormalities, poor ovulatory quality, and impaired fertility [20,21]. Brown and colleagues further emphasized that thyroid function affects multiple sites of the female hypothalamic-pituitary-gonadal axis, linking thyroid disruption to menstrual irregularity, infertility, premature ovarian insufficiency, and PCOS [21].

Autoimmunity adds another layer of complexity. Thyroid autoimmunity is more frequent in women and has been associated with infertility, miscarriage, ovarian dysfunction, and reproductive endocrine disorders even in the absence of overt thyroid dysfunction [5,16,19,22]. This suggests that the reproductive burden of thyroid-related disease cannot be

understood solely through serum TSH and free hormone levels; immune-mediated mechanisms may also be relevant.

Menstrual Dysfunction Associated with Thyroid Disorders. Menstrual abnormalities are one of the most common reproductive presentations of thyroid disease. Classical descriptions associate hypothyroidism with menorrhagia and hyperthyroidism with oligomenorrhea, hypomenorrhea, or amenorrhea, but real-world clinical patterns are often mixed [1,2,8]. Across studies, reported manifestations include menorrhagia, oligomenorrhea, amenorrhea, polymenorrhea, metrorrhagia, and irregular cycles [8-13,23].

A key clinical observation is that reproductive symptoms may be an early sign of thyroid dysfunction. In the study by Joshi et al., menstrual irregularities and lactation failure preceded overt thyroid dysfunction or goiter in a notable proportion of women, supporting the idea that gynecologic symptoms may represent an early warning signal rather than merely a downstream complication [8]. This finding remains clinically important because it argues for proactive endocrine evaluation in women presenting with menstrual complaints.

More recent gynecologic studies reinforce this association. Ajmani et al. reported thyroid dysfunction in 44% of women with menstrual disorders, with subclinical hypothyroidism being the most prevalent abnormality [11]. Verma et al. found thyroid dysfunction in 33% of women with AUB, again with subclinical hypothyroidism predominating [9]. Mahale et al. reported thyroid dysfunction in 25% of women with AUB and observed that menorrhagia was the most common menstrual abnormality in hypothyroid women [10]. Similarly, Sridevi et al. found an overall thyroid dysfunction prevalence of 28.8% among women with AUB, with subclinical hypothyroidism as the most common disorder and menorrhagia as the dominant bleeding pattern [12]. Sahu and Rath also noted that 20% of women presenting with menstrual disorders had thyroid abnormalities, and menorrhagia was frequent in hypothyroid and subclinical hypothyroid groups [13].

These studies, while mostly cross-sectional and hospital-based, consistently point in the same direction: thyroid dysfunction is common enough in women with menstrual complaints to justify routine clinical attention. Importantly, the predominance of subclinical hypothyroidism across several datasets suggests that reliance on overt hypothyroid symptoms alone may miss a sizeable proportion of affected patients [9-13].

The mechanisms underlying menstrual dysfunction differ somewhat between hypothyroidism and hyperthyroidism. In hypothyroidism, altered estrogen metabolism, reduced coagulation factor synthesis, anovulation, and endometrial changes may contribute to heavy bleeding [1,2]. Ajmani et al. observed proliferative endometrium more often in hypothyroid women, whereas hyperthyroid women more often had atrophic endometrium, highlighting a biologically coherent tissue-level difference [11]. In hyperthyroidism, increased sex hormone-binding globulin, enhanced peripheral estrogen metabolism, and disturbed gonadotropin dynamics contribute more commonly to infrequent or scanty menstruation [1,2].

From a practical perspective, the literature supports thyroid function testing in women with unexplained AUB, especially when structural causes are absent or symptoms are accompanied by ovulatory disturbance, infertility, galactorrhea, or signs suggestive of endocrine disease [9-13].

Ovarian Dysfunction, Ovulation, and Ovarian Reserve. The effects of thyroid disorders on ovarian function extend beyond menstrual cycle patterning. Ovulatory dysfunction is a

major pathway through which thyroid disease contributes to subfertility. Hypothyroidism may impair ovulation through hyperprolactinemia, gonadotropin dysregulation, luteal inadequacy, and reduced progesterone production [2,6,24]. Hyperthyroidism may also impair cycle regularity and fecundity, although hypothyroidism is more consistently associated with anovulation in contemporary gynecologic literature [1,2].

Gul et al. recently showed that women with ovulatory disorders had significantly higher mean TSH levels than controls, and subclinical hypothyroidism was substantially more common in the ovulatory disorder group [25]. This finding supports the view that even mild thyroid dysfunction may be clinically relevant in women with ovulatory impairment.

Interest has also grown in the relationship between thyroid function and ovarian reserve. Kabodmehri et al. found that increasing TSH was associated with greater odds of reduced AMH, particularly in women older than 35 years, suggesting that thyroid status may influence ovarian reserve or at least track with diminished ovarian function in infertility populations [15]. Weghofer et al. similarly explored whether thyroid function or thyroid autoimmunity affects functional ovarian reserve, reinforcing the clinical relevance of this question in reproductive medicine [16]. While causality remains incompletely resolved, these studies justify including thyroid status in the broader endocrine assessment of women with low ovarian reserve or unexplained subfertility.

Premature ovarian insufficiency also appears to intersect with thyroid autoimmunity. Ashrafi et al. found significantly higher anti-thyroid antibody positivity in women with premature ovarian failure, especially familial cases, suggesting an autoimmune contribution in at least a subset of patients [17]. These findings are consistent with the broader observation that autoimmune endocrine disorders tend to cluster and that ovarian dysfunction may occur in the context of systemic or organ-specific autoimmunity [5,17,22].

PCOS, Endometriosis, and Thyroid Disease. PCOS is the gynecologic condition most frequently discussed alongside thyroid dysfunction. Several studies in your reference set reported substantial thyroid abnormalities among women with PCOS [3,4,14,26]. Sharma et al. found hyperthyroidism, hypothyroidism, and subclinical hypothyroidism among women with polycystic ovarian disease, along with high rates of menstrual irregularity and androgenic symptoms [3]. Ganvir et al. reported a very high overall prevalence of thyroid dysfunction in PCOS, with both overt and subclinical hypothyroidism represented [26]. Altuntaş and Güneş also identified increased thyroid autoimmunity, thyroid volume, and nodularity in euthyroid women with PCOS, especially in the classic phenotype [14].

Kirkegaard et al. reviewed the links between endometriosis, PCOS, and thyroid disease and concluded that the evidence points more strongly toward an association between hypothyroidism and PCOS, while the role of autoimmunity remains an area of uncertainty requiring better-designed studies [4]. This is important because menstrual and ovarian dysfunction in such patients may be multifactorial. Clinically, thyroid assessment should not replace standard evaluation for PCOS or endometriosis, but it should be integrated into it.

Infertility and Assisted Reproduction. Thyroid dysfunction is a well-recognized contributor to female infertility [2,18,19,24]. Poppe's review emphasized that female infertility in the setting of thyroid disease still raises "more questions than answers," but the importance of systematic thyroid assessment is clear [18]. Poppe and Velkeniers earlier highlighted the increased prevalence of thyroid autoimmunity in infertile women, particularly those with

endometriosis [27]. Redmond also underscored the broad reproductive burden of thyroid dysfunction, including menstrual disturbance, infertility, and pregnancy-related consequences [2].

A key contemporary issue is assisted reproductive technology (ART). The 2021 European Thyroid Association guideline recommends careful pre-ART thyroid evaluation and optimization, especially in women with hypothyroidism or thyroid autoimmunity [19]. Women treated with levothyroxine before ART should ideally achieve a TSH below 2.5 mIU/L, and subfertile women with hyperthyroidism should restore and maintain euthyroidism before treatment [19]. The guideline also notes that ovarian stimulation may exacerbate thyroid stress, particularly in women with thyroid autoimmunity [19].

Bucci et al. reviewed thyroid autoimmunity in female infertility and ART and concluded that although mechanistic and clinical uncertainties remain, thyroid autoimmunity warrants attention in reproductive medicine because of its associations with miscarriage risk and possibly ART outcomes [22]. Potiris et al. similarly described thyroid disease as an often underestimated cause of female infertility and emphasized that levothyroxine therapy in hypothyroid women may restore ovulatory function and improve reproductive outcomes [24].

Pregnancy Implications and the Need for Preconception Detection. Although the present review focuses on menstrual and ovarian dysfunction, pregnancy outcomes strengthen the case for early detection. Thyroid dysfunction before conception often persists into pregnancy or becomes clinically more important during gestation [1,7,19]. Antolič et al. reported increased infertility, menstrual irregularity, and several adverse pregnancy outcomes in women with thyroid dysfunction [28]. Mahadik et al. found that hypothyroidism in pregnancy was associated with anemia and adverse neonatal outcomes, including low birth weight and NICU admission [29].

These data matter because reproductive-aged women with menstrual irregularity or ovulatory dysfunction are often also planning pregnancy. Early thyroid detection in gynecology clinics may therefore improve not only cycle control and fertility but also downstream pregnancy outcomes [7,19,28,29].

Early Detection: Who Should Be Screened? The cumulative literature supports a low threshold for thyroid screening in reproductive-aged women with menstrual or ovarian dysfunction [8-13,15,19,25]. Based on the reviewed evidence, screening is particularly justified in women with: abnormal uterine bleeding or persistent menstrual irregularity; oligomenorrhea, amenorrhea, or suspected anovulation; infertility or recurrent pregnancy loss; PCOS, especially with obesity, insulin resistance, or treatment-resistant symptoms; low ovarian reserve or premature ovarian insufficiency; clinical suspicion of autoimmune disease; preconception planning or ART candidacy.

TSH is the most practical first-line test, but its interpretation should be clinical rather than purely numeric [2,12,19]. In women with reproductive dysfunction, TSH should usually be accompanied by free T4, and in selected cases by free T3, prolactin, anti-thyroid peroxidase antibodies, and anti-thyroglobulin antibodies [11,17,19,22]. Ultrasonography may be useful when nodular disease or autoimmune thyroiditis is suspected, particularly in PCOS populations where nodularity and autoimmunity may be more prevalent [14].

Management Principles. Management depends on the type and severity of thyroid dysfunction, reproductive goals, and the presence of coexisting gynecologic disease. In overt

hypothyroidism, levothyroxine is the standard treatment and often improves menstrual regularity, ovulatory function, and fertility [1,2,24]. Kumar and Kotur summarized that reproductive-aged women with hypothyroidism frequently present with menstrual irregularities, PCOS, infertility, and pregnancy loss, and that routine thyroid testing can facilitate early treatment and reduce unnecessary procedures [30].

In subclinical hypothyroidism, treatment decisions are more individualized. The evidence is strongest for treatment when TSH is clearly elevated, thyroid autoantibodies are positive, infertility is present, or pregnancy/ART is planned [18,19,22]. In women with AUB but no reproductive plans, treatment still deserves consideration when symptoms, laboratory findings, and the broader clinical picture suggest thyroid-mediated dysfunction.

Hyperthyroidism requires etiologic diagnosis and restoration of euthyroidism with antithyroid therapy, radioiodine, or surgery as appropriate [1,7]. Reproductive counseling is important because uncontrolled hyperthyroidism can impair fertility and complicate pregnancy [1,7,19].

A multidisciplinary model is especially valuable for subfertility. Hussain et al. described improved fertility outcomes with coordinated management of thyroid-linked subfertility, reinforcing the importance of integrated gynecologic and endocrine care [31]. Although the evidence base for multidisciplinary models is still limited, the principle is sound: menstrual and ovarian dysfunction associated with thyroid disease should not be managed in isolation.

Emerging Considerations. Post-COVID reproductive endocrine disturbances have recently drawn attention. Petruk et al. described a phase-dependent thyroid pattern in women with menstrual dysfunction after COVID-19, with early thyrotoxic changes followed later by hypothyroid features and ovulatory dysfunction [32]. While this is still an emerging area, it suggests that thyroid assessment may be useful in women who develop new menstrual irregularity after significant viral illness.

Another area of interest is inflammatory signaling in pubertal menorrhagia associated with thyroid pathology, as explored by Tsysar et al. [33]. Although this is less directly relevant to adult reproductive-age practice, it supports the broader concept that thyroid-related menstrual dysfunction may involve endocrine-immune cross-talk rather than isolated hormonal imbalance.

Limitations of Current Evidence. Despite the consistency of associations, the literature has important limitations. Much of the available evidence is cross-sectional, hospital-based, and derived from gynecology clinics, which may overestimate prevalence. Definitions of thyroid dysfunction and menstrual outcomes are not always standardized. Subclinical disease remains particularly difficult to interpret, and causality cannot be assumed in every case. Likewise, the relationship between thyroid autoimmunity and ovarian reserve or ART outcomes is still not fully settled [15,16,19,22].

Nevertheless, the recurrent pattern across studies is clinically meaningful. Even if thyroid dysfunction is not the sole cause of menstrual or ovarian abnormalities, it is a sufficiently common and treatable contributor that it should not be overlooked.

Conclusion. Thyroid disorders are a significant and frequently underdiagnosed cause of menstrual and ovarian dysfunction in reproductive-aged women. The evidence supports meaningful associations between thyroid dysfunction and abnormal uterine bleeding, oligomenorrhea, amenorrhea, ovulatory disorders, infertility, altered ovarian reserve, PCOS,

and adverse reproductive outcomes [1-5,9-19,25]. Menstrual dysfunction may precede overt thyroid disease, which makes early detection especially important [8].

For high-quality gynecologic care, thyroid assessment should be incorporated into the routine evaluation of women with unexplained menstrual irregularity, ovulatory dysfunction, infertility, recurrent pregnancy loss, PCOS, or suspected premature ovarian insufficiency. TSH-based screening supported by targeted hormonal and autoimmune testing offers a practical pathway to early diagnosis. Timely treatment, especially restoration of euthyroidism in hypothyroid or hyperthyroid women, may improve menstrual function, ovulation, fertility, and pregnancy readiness [19,24,30].

In short, thyroid dysfunction should be considered not as a peripheral endocrine issue, but as a central differential diagnosis in reproductive-aged women with menstrual and ovarian dysfunction.

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Data Availability. The data supporting the findings of this study were obtained from previously published articles and publicly accessible scientific databases. Further details are available from the corresponding author upon reasonable request.

Ethics Declaration. Ethical approval was not required for this study because it is a narrative review based exclusively on previously published data.

Competing Interest. The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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