







Insights on thyroid function in menopause: a review

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ABSTRACT

This review explores the complex relationship between thyroid function and menopause, focusing on physiological interactions, clinical implications, and diagnostic challenges associated with thyroid dysfunction in postmenopausal women. Drawing on recent studies and current understanding, it examines how menopausal hormonal changes influence thyroid health, increasing the risk of conditions such as hypothyroidism, hyperthyroidism, and subclinical thyroid disorders. Overlapping symptoms, including mood changes, sleep disturbances, weight fluctuations, and reduced libido, complicate diagnosis, as they can be attributed to both menopause and thyroid dysfunction. Hormonal shifts can also impact bone mineral density, cardiovascular health, and metabolism, while prolonged estrogen exposure and surgical menopause are linked to an elevated risk of thyroid cancer. Diagnostic complexity is heightened by age-related changes, comorbidities, and potential interferences in thyroid function tests. Routine thyroid assessment is recommended for women with surgical menopause or other risk factors. Personalized management, including menopausal hormone therapy (MHT), can address overlapping symptoms and mitigate skeletal and cardiovascular risks. A patient-centred approach is crucial, with treatment tailored to individual health profiles. Further research is needed to clarify the interactions between menopause and thyroid function to improve clinical care.

KEYWORDS

thyroid function, menopause, hyperthyroidism, hypothyroidism, surgical menopause

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1. INTRODUCTION

Menopause is a natural physiological transition that women undergo at a specific stage in their lives. Concurrently, subclinical hypothyroidism—predominantly resulting from autoimmune thyroiditis—is a prevalent endocrine disorder among women of reproductive age, with an estimated prevalence of 6–10% [1]. This condition frequently coexists with or emerges during the menopausal transition. Perimenopause is characterized by a variety of symptoms, including menstrual irregularities, night sweats, hot flashes, mood disturbances, muscle and joint pain, anxiety or depression, and insomnia.

Additionally, women may experience decreased libido and symptoms related to vulvovaginal atrophy, such as vaginal dryness and dyspareunia [2].

Similarly, thyroid disorders present with a range of symptoms that vary in severity and can adversely affect women's quality of life (QoL) at any life stage, including during perimenopause [3]. Due to the overlap of symptoms, such as increased sweating, mood swings, anxiety or depression, decreased libido, and menstrual irregularities, differentiating between menopausal and thyroid-related conditions can be challenging. Both hypothyroidism and hyperthyroidism influence circulating sex hormone levels and disrupt autonomic system regulation, which can consequently impair sexual function leading to issues such as diminished libido, reduced sexual desire and arousal, and dyspareunia [4]. Moreover, suboptimal thyroid function has been suggested to influence the age at which menopause occurs. However, a Mendelian randomization study utilizing a two-sample approach and data from genome-wide association studies (GWAS) found no significant association between genetically predicted thyroid function and ovulatory function [4].

It is essential to summarize the most recent research on this subject because of the intricacy of hormone interactions during menopause and the high incidence of thyroid dysfunction in menopausal women. Healthcare professionals will be better equipped to diagnose and treat thyroid-related diseases in menopausal women if they have a thorough understanding of how menopausal changes impact thyroid physiology and the clinical signs of thyroid disorders. By giving a thorough overview of thyroid function alterations during menopause and emphasizing diagnostic difficulties, clinical implications, and therapeutic techniques, this study seeks to close the information gap.

2. PHYSIOLOGY AND FUNDAMENTAL MECHANISMS UNDERLYING THYROID FUNCTION

The thyroid diverticulum originates at the end of the fourth week of embryonic development as a solid, proliferative mass of endoderm located at the foramen cecum, which subsequently forms part of the tongue [5]. This endodermal mass migrates caudally through the developing neck along the thyroglossal duct towards its eventual anatomical position just inferior to the cricoid cartilage [6]. Under normal embryogenesis, the thyroglossal duct undergoes regression and typically obliterates by the end of the fifth week. The only remnant of this developmental pathway is the foramen cecum at

the base of the mature tongue. By this stage, the isolated thyroid gland differentiates into two distinct lobes connected by an isthmus of thyroid tissue. It continues its downward migration and attains its definitive location by the end of the seventh week of gestation [7]. During this process, cells from the ultimobranchial bodies integrate into the developing thyroid tissue, giving rise to the parafollicular cells, or C cells, which are responsible for the secretion of calcitonin. Additionally, the connective tissue framework of the thyroid gland originates from neural crest cells that invade the glandular structure during its development [8].

The thyroid gland is a key regulator of metabolism, growth, and development through the synthesis and release of thyroid hormones, primarily thyroxine (T4) and triiodothyronine (T3). These hormones are produced by the follicular cells of the thyroid gland via iodination and coupling of tyrosine residues in thyroglobulin. The hypothalamic-pituitary-thyroid (HPT) axis tightly controls their production: the hypothalamus secretes thyrotropin-releasing hormone (TRH), which stimulates the anterior pituitary gland to release thyroid-stimulating hormone (TSH). TSH then acts on the thyroid gland to promote the synthesis and release of T4 and, to a lesser extent, T3. Although T4 is the major thyroid hormone produced, it is biologically less active and undergoes peripheral conversion to the more potent T3 via deiodinase enzymes [9].

Thyroid hormones exert their physiological effects primarily through binding to nuclear thyroid hormone receptors (THR), which interact with thyroid hormone response elements (TREs) within the promoter regions of target genes, thereby modulating gene transcription. This genomic action impacts numerous physiological processes, including basal metabolic rate, thermogenesis, lipid and carbohydrate metabolism, and cardiovascular function. Additionally, thyroid hormones can induce non-genomic effects by interacting with mitochondrial proteins and plasma membrane receptors, rapidly influencing cellular metabolism [10].

One of the most significant roles of thyroid hormones is their ability to enhance cellular function and metabolism across almost all nucleated cells. They increase basal metabolic rate, heat production, and oxygen consumption by activating mitochondrial uncoupling proteins, thereby enhancing thermogenesis. This rise in energy expenditure necessitates increased blood flow, ventilation, and mechanisms for heat dissipation, such as sweating. Through positive chronotropic and inotropic effects, thyroid hormones increase resting heart rate, cardiac output, and stroke volume, while simultaneously dilating blood vessels in the heart, muscles, and skin, leading to reduced peripheral vascular re-

sistance. They also elevate blood volume via the activation of the renin-angiotensin-aldosterone system [11].

T3 specifically boosts ventilation and respiratory rate and facilitates oxygen delivery by promoting haemoglobin and erythropoietin synthesis. It also aids in the absorption of essential nutrients such as folate and cobalamin, crucial for red blood cell production [12,13]. Furthermore, T3 plays a vital role in bone metabolism, promoting adult bone remodelling and linear bone growth, which is essential during prenatal development [14]. Its influence extends to the central nervous system, where it enhances wakefulness, alertness, and reflexes, and to the autonomic nervous system, promoting sympathetic activation. Moreover, thyroid hormones impact pituitary function, renal clearance, and reproductive health, further illustrating their wide-ranging physiological influence [15].

Feedback regulation within the HPT axis is essential for maintaining thyroid hormone homeostasis. Elevated levels of T4 and T3 inhibit TRH and TSH production through a negative feedback loop at both the hypothalamic and pituitary levels. Conversely, low thyroid hormone levels stimulate increased TRH and TSH secretion. Disruption of this finely tuned axis can lead to thyroid dysfunction, manifesting as hypothyroidism or hyperthyroidism, both of which may be exacerbated by hormonal changes during menopause. The decline in estrogen levels during menopause can influence thyroid function by altering the synthesis of thyroid-binding globulin (TBG) and modulating peripheral conversion of T4 to T3. These interactions complicate the clinical assessment of thyroid function in postmenopausal women, highlighting the importance of understanding the underlying mechanisms of thyroid regulation [16].

Therefore, thyroid hormones are indispensable for maintaining physiological balance and metabolic equilibrium. Their profound effects on cardiovascular, respiratory, metabolic, and neurological systems underline their importance in sustaining overall health and responding to physiological stressors.

3. THYROID HORMONE SYNTHESIS AND METABOLISM

3.1. The essential role of iodine

Thyroid hormones, primarily T4 and T3, are synthesized in the thyroid gland through a series of tightly regulated biochemical processes that rely on an adequate supply of exogenous iodine. The

synthesis begins with the active uptake of iodine from the bloodstream into the thyroid follicular cells via the sodium-iodide symporter (NIS). Once inside the follicular cells, iodine is oxidized by the enzyme thyroid peroxidase (TPO) and subsequently incorporated into the tyrosine residues of thyroglobulin to form monoiodotyrosine (MIT) and diiodotyrosine (DIT). The coupling of two DIT molecules produces T4, while the combination of one MIT and one DIT forms T3. This iodinated thyroglobulin is stored within the colloid of the thyroid follicles. Upon stimulation by TSH, thyroglobulin is endocytosed, and proteolytic cleavage releases free T4 and T3 into the circulation. Peripheral metabolism of T4 to the more biologically active T3 occurs predominantly in the liver and kidneys through deiodination by deiodinase enzymes. An adequate exogenous iodine supply is essential for maintaining thyroid hormone synthesis and preventing disorders such as iodine deficiency goiter and hypothyroidism. Iodine deficiency can disrupt hormone production, leading to reduced T4 and T3 levels, compensatory TSH elevation, and thyroid hyperplasia. Thus, dietary iodine intake remains critical for sustaining normal thyroid function and metabolic homeostasis [17].

3.2. Regulation of thyroid hormone synthesis

The regulation process begins with the hypothalamus releasing TRH, which stimulates the anterior pituitary to secrete TSH. TSH acts on the thyroid gland, promoting the synthesis and release of T4 and T3. Upregulation of thyroid hormones occurs in response to external stimuli, such as cold exposure or increased metabolic demands. This triggers the hypothalamus to increase TRH secretion, leading to elevated TSH levels and subsequent stimulation of T4 and T3 production, thereby enhancing metabolic rate and thermogenesis [18]. Conversely, downregulation of thyroid hormones is achieved through negative feedback mechanisms, where elevated circulating levels of T4 and T3 inhibit TRH and TSH secretion at the hypothalamic and pituitary levels, respectively. This feedback loop helps prevent excessive hormone production and maintains serum levels within a physiological range [19]. Additionally, thyroid hormone autoregulation at the glandular level minimizes TSH response variability, maintaining consistent hormonal output despite metabolic fluctuations. This autoregulatory mechanism involves adaptive changes in TSH sensitivity, which stabilize thyroid function even under metabolic stress [20].

Peripheral conversion of T4 to the more bio-

logically active T3 predominantly occurs in the liver and kidneys via deiodinase enzymes, which adjust the T3 pool based on metabolic requirements [19]. Importantly, the thyroid gland exhibits autoregulatory capacity that allows it to adjust hormone synthesis independently of TSH, particularly in response to variations in iodine availability. For example, iodine deficiency can reduce T4 production, prompting increased TSH stimulation, while excess iodine can transiently inhibit thyroid hormone synthesis through the Wolff-Chaikoff effect [18]. These multi-level regulatory mechanisms enable the thyroid to respond effectively to varying environmental and physiological conditions, ensuring metabolic equilibrium and adaptation to stressors.

4. BONE REMODELING AND CALCIUM METABOLISM: THE ROLE OF THYROID HORMONES

The bone remodeling cycle is a continuous process of bone formation and resorption, essential for maintaining skeletal integrity and calcium homeostasis. This cycle consists of two main phases: osteoclastic resorption and osteoblastic formation. Osteoclasts, which are multinucleated cells of hematopoietic origin, break down bone matrix by secreting hydrochloric acid and proteolytic enzymes, releasing calcium and phosphate into the bloodstream [21]. Following resorption, osteoblasts, originating from mesenchymal stem cells, synthesize new bones by depositing collagen and facilitating mineralization with calcium and phosphate. This coordinated process is tightly regulated by systemic hormones and local signaling factors, including parathyroid hormone (PTH), vitamin D, and thyroid hormones [22].

Thyroid hormones are essential regulators of bone remodeling. T3 directly stimulates osteoblastic activity, promoting bone formation, while also indirectly enhancing osteoclastic resorption by increasing the expression of receptor activator of nuclear factor kappa-B ligand (RANKL) on osteoblasts [18]. Hyperthyroidism, characterized by excessive thyroid hormone levels, accelerates bone turnover, leading to increased resorption and decreased bone mineral density (BMD), heightening the risk of osteoporosis and fractures [19]. Conversely, hypothyroidism reduces bone turnover, resulting in diminished osteoclastic and osteoblastic activity, potentially leading to low bone remodeling and secondary osteoporosis [20]. Moreover, calcium metabolism is intimately linked with thyroid dysfunction. Hyperthyroidism increases serum calcium levels due to elevated

bone resorption, while hypothyroidism may reduce calcium release from bones. Moreover, dysregulation of PTH, which controls serum calcium levels, can further disrupt calcium homeostasis in thyroid disorders [18]. Vitamin D status also influences calcium absorption and bone mineralization, and deficiencies can compound the adverse skeletal effects of thyroid hormone imbalance. Maintaining euthyroid status and optimal calcium and vitamin D levels is therefore crucial for preserving bone health and metabolic equilibrium [19].

5. THYROID HORMONES AND THEIR IMPACT ON THE CENTRAL NERVOUS SYSTEM

Thyroid hormones play a crucial role in the central nervous system (CNS), affecting various physiological processes such as mood regulation, cognitive function, and emotional stability. The two main thyroid hormones, T3 and T4, regulate the expression of genes essential for neuronal development, neurotransmitter synthesis, and synaptic plasticity. These hormones also influence the levels of essential neurotransmitters like serotonin, dopamine, and norepinephrine, all of which are linked to mood disorders. In cases of hypothyroidism, where T3 and T4 levels are low, individuals often experience symptoms such as depression, lethargy, and cognitive impairment. This occurs due to reduced neurotransmitter activity and altered neuron communication [18]. On the other hand, hyperthyroidism, characterized by excessive thyroid hormone levels, is commonly associated with symptoms of anxiety, irritability, and sleep disturbances. These effects are thought to arise from an overactive sympathetic nervous system and increased turnover of neurotransmitters [20].

Additionally, thyroid hormones influence neurogenesis and neuroplasticity, critical memory and learning processes. Disruptions in thyroid hormone levels can lead to issues in these processes, resulting in cognitive deficits and mood instability. Research has shown that thyroid disorders significantly affect the severity of psychosomatic symptoms in patients, with both hypothyroidism and hyperthyroidism worsening the frequency and intensity of anxiety, irritability, and depression [23]. Given the extensive impact of thyroid hormones on the CNS, it is crucial to consider thyroid dysfunction when diagnosing mood disorders and cognitive impairments, especially in individuals who present with unexplained neuropsychiatric symptoms.

6. PHYSIOLOGY AND PATHOPHYSIOLOGY OF THYROID GLAND IN MENOPAUSE

6.1. Effects of menopause in bone remodeling and calcium metabolism

During menopause, the decline in estrogen levels significantly impacts both bone remodelling and thyroid function, leading to an imbalance between osteoclastic resorption and osteoblastic formation. Estrogen normally inhibits osteoclast activity by downregulating receptor activator of RANKL and upregulating osteoprotegerin (OPG), which acts as a decoy receptor for RANKL. Reduced estrogen during menopause leads to increased RANKL expression and decreased OPG, resulting in heightened osteoclastic activity and accelerated bone resorption [11]. Consequently, postmenopausal women are at a higher risk of osteoporosis and fractures due to rapid bone loss. At the same time, the physiology of the thyroid gland undergoes significant changes during menopause due to hormonal fluctuations. As estrogen and progesterone levels decline, thyroid hormone metabolism and function may be affected. Estrogen influences the production of TBG, and lower estrogen levels reduce TBG, potentially leading to altered levels of T3 and T4 [18]. This change in thyroid hormone dynamics can impact bone health, as thyroid dysfunction during menopause may further exacerbate bone loss.

Hyperthyroidism, already associated with increased bone turnover, can amplify menopausal bone resorption, significantly lowering BMD and increasing fracture risk [19]. Conversely, hypothyroidism may initially appear protective due to reduced bone turnover; however, prolonged hypothyroidism can impair bone quality and mineralization, especially when accompanied by inadequate calcium or vitamin D intake [20].

Calcium metabolism during menopause is also adversely affected by hormonal changes. Decreased estrogen levels reduce intestinal calcium absorption and increase renal calcium loss, promoting secondary hyperparathyroidism. This compensatory rise in PTH further enhances osteoclastic bone resorption, exacerbating bone mass loss [22]. Additionally, vitamin D deficiency, prevalent among postmenopausal women, disrupts calcium homeostasis and bone mineralization. When thyroid dysfunction coexists, these metabolic disturbances are intensified, highlighting the importance of monitoring thyroid function and ensuring sufficient calcium and vitamin D intake in postmenopausal women to mitigate the risk of osteoporosis [18].

6.2. Effects of menopause on the central nervous system

During menopause, fluctuations in hormone levels can exacerbate psychiatric symptoms linked to thyroid dysfunction. These hormonal changes can influence mood regulation, cognitive function, and emotional stability, making postmenopausal women particularly vulnerable to both thyroid dysfunction and psychiatric disturbances [24]. In hypothyroidism, commonly observed in postmenopausal women, reduced levels of T3 and T4 can result in symptoms such as depression, lethargy, and cognitive impairment due to altered neurotransmitter activity [20]. In contrast, hyperthyroidism may lead to increased anxiety, irritability, and insomnia due to heightened sympathetic nervous system activity and increased neurotransmitter turnover. Studies have also shown that thyroid disorders during menopause can amplify psychosomatic symptoms such as fatigue, mood disturbances, and emotional instability, which are often observed in women undergoing hormonal fluctuations [23,24]. These psychiatric symptoms, alongside changes in thyroid hormone levels, underscore the complex interaction between hormonal changes during menopause and CNS function. Therefore, it is essential to consider thyroid dysfunction in the evaluation of mental health symptoms during menopause, particularly in women presenting with unexplained emotional or cognitive disturbances.

7. CLINICAL IMPLICATIONS

The occurrence of thyroid nodules tends to rise with age and is significantly more common in women than in men. In areas with sufficient iodine intake, the prevalence of thyroid nodules is about 5% in women and 1% in men [25]. Among those diagnosed with thyroid nodules, thyroid cancer is found in 7–15% of cases, with a higher incidence in women compared to men [26]. This gender difference is partially explained by hormonal fluctuations during the menopausal transition, which contribute to the observed differences in thyroid disease patterns between sexes [27]. Surgical menopause, which leads to a sudden decline in estrogen levels and severe menopausal symptoms, has been linked to an increased risk of thyroid cancer [28]. Interestingly, the mechanism underlying this association is not clearly understood, as natural menopause is correlated with a lower incidence of thyroid cancer [28,29]. Additionally, early onset of menarche and delayed natural menopause are linked to a heightened risk of thyroid cancer, indicating that extended exposure to estrogen may play a role in thyroid cancer development [30].

Nevertheless, a meta-analysis of 5,434 thyroid cancer patients, including 615 individuals who underwent hysterectomy, revealed that hysterectomy is associated with an increased likelihood of developing thyroid cancer later in life (standardized risk ratio [SRR] 1.43, 95% confidence interval [CI] 1.15–1.78) [31]. However, this finding should be interpreted cautiously due to considerable variability among the studies analysed ($I^2 = 47\%$), potentially due to differences in follow-up periods, as one study found a higher cancer risk mainly within the first two years post-surgery [32].

Additionally, a large cohort study in Korea reported no significant difference in thyroid cancer incidence between women who underwent hysterectomy with or without oophorectomy [33]. This suggests that the increased risk may not solely result from decreased estrogen levels. It is possible that other common risk factors for thyroid cancer and indications for hysterectomy, as well as more frequent post-surgical screening, contribute to the observed relationship. Notably, hysterectomy has been associated with a heightened risk of thyroid cancer even when oophorectomy is not performed. Additionally, papillary thyroid cancer exhibits a poorer prognosis in postmenopausal women compared to premenopausal women [18], with older age itself being a negative prognostic factor in papillary thyroid cancer [34]. The level of estrogen receptor (ER) expression, particularly ER- α , may influence the aggressiveness of papillary thyroid cancer after menopause, as ER- α expression increases in the thyroid gland during menopause, potentially activating intracellular pathways implicated in thyroid cancer progression [35]. Despite the generally favourable prognosis of papillary thyroid cancer, postmenopausal status has been associated with a higher recurrence rate of differentiated papillary thyroid cancer [36]. This recurrence is attributed to decreased estrogen and elevated follicle-stimulating hormone (FSH) levels, mediated by epidermal growth factor receptor (EGFR) activation, which is more prominently expressed in papillary thyroid cancer cells in postmenopausal women compared to premenopausal women [37].

Thyroid hormones play a crucial role in regulating metabolic processes [38]. Metabolic dysregulation has been associated with both hypothyroidism and hyperthyroidism. Low concentrations of free thyroxine (fT4) have been linked to insulin resistance [39], with this association being more significant in postmenopausal women, indicating a potential synergistic effect with hormonal changes occurring during menopause [38]. On the other hand, hyperthyroidism negatively impacts cardiovascular function and is linked to a higher risk of developing heart failure and atrial fibrillation [40].

Sub-clinical hypothyroidism has been linked to elevated triglyceride (Tg) levels, an increased total cholesterol (TC)/high-density lipoprotein cholesterol (HDL-C) ratio, and a heightened risk of cardiovascular disease [41]. Additionally, thyroid-stimulating hormone (TSH) has shown a positive correlation with TC, low-density lipoprotein cholesterol (LDL-C), HDL-C, and Tg levels [42]. Weight gain is a common issue for women undergoing the menopausal transition as well as those with hypothyroidism [43]. The relationship between thyroid hormones, energy expenditure, and body weight is well-documented; suboptimal thyroid function is associated with decreased resting energy expenditure, elevated cholesterol levels, diminished gluconeogenesis and lipolysis, ultimately contributing to weight gain [44].

8. DIAGNOSIS, MANAGEMENT AND PRACTICAL RECOMMENDATIONS FOR INTERPRETING HORMONE LEVELS

Diagnosing thyroid dysfunction in postmenopausal women presents considerable challenges due to the substantial overlap of symptoms between menopause and thyroid disorders. Symptoms such as palpitations, insomnia, and weight gain are common to both conditions, complicating the differential diagnosis. Additionally, interpreting thyroid function tests in this population can be complex due to factors such as aging, comorbid conditions, and the effects of concurrent medications [45]. To address these diagnostic challenges, the American Society of Clinical Endocrinology recommends routine thyroid screening for older women, particularly those undergoing menopause [46].

The clinical significance of managing thyroid function in postmenopausal women is heightened by the increased risk of osteoporosis and fractures, which can be exacerbated by thyroid dysfunction. Hyperthyroidism, including subclinical forms, is associated with decreased bone mineral density (BMD) and a higher risk of vertebral fractures [47]. In elderly women, factors like reduced metabolic clearance and drug interactions can influence thyroxine requirements. Improper dosing of thyroxine can lead to severe complications such as atrial fibrillation, reduced bone density, and other tachyarrhythmias. Therefore, precise monitoring of thyroid function and individualized treatment plans are essential for protecting bone health in this demographic [48].

Healthcare providers must maintain clinical vigilance when assessing postmenopausal women, given the significant overlap of symptoms between

thyroid dysfunction and menopause. Routine screening is especially recommended for women with a history of surgical menopause or other identified risk factors [49]. It is also crucial to account for confounding variables when interpreting thyroid function tests, such as the potential interference caused by biotin supplementation [50]. An individualized treatment approach is key to managing both menopausal symptoms and thyroid dysfunction effectively. Menopausal hormone therapy (MHT) is a viable therapeutic option that can mitigate overlapping symptoms without increasing thyroid cancer risk. However, accurate dosing is essential to avoid cardiovascular or skeletal complications [51].

A systematic approach to interpreting thyroid function tests is fundamental in managing thyroid disorders. The primary screening marker is TSH, as elevated levels typically indicate hypothyroidism and suppressed levels suggest hyperthyroidism. However, TSH alone may not suffice for diagnosis, especially in central hypothyroidism or non-thyroidal illness syndrome, where TSH levels may not reflect actual thyroid hormone activity [52]. Therefore, assessing Free T3 (FT3) and Free T4 (FT4) is vital, as these hormones represent the biologically active forms. FT3 is particularly valuable in detecting hyperthyroidism or in cases where TSH does not align with clinical symptoms, offering a more precise evaluation of thyroid function [53].

In addition to TSH and free hormone levels, measuring TBG is crucial, especially in patients with altered hormone binding, such as those on estrogen therapy or during pregnancy. Changes in TBG can influence total thyroid hormone levels without directly affecting the thyroid gland's functional status. Although TRH testing is less commonly employed, it can assist in identifying central thyroid dysfunction by evaluating hypothalamic and pituitary responses [54].

Practical recommendations:

- Screening and diagnosis: use TSH as the initial screening tool for thyroid dysfunction. If TSH levels are abnormal or discordant with symptoms, measure FT3 and FT4 for a more accurate assessment.
- Special considerations: account for factors affecting TBG levels, particularly in patients undergoing hormonal therapy or experiencing physiological changes like pregnancy.
- Monitoring and management: conduct routine thyroid screenings in postmenopausal women, especially those with surgical menopause or significant risk factors.
- Personalized treatment: opt for individualized

therapy, adjusting thyroxine dosages cautiously to avoid under- or over-treatment.

- Bone health: regularly evaluate BMD, particularly in patients with hyperthyroidism, to mitigate fracture risk.
- Comprehensive care: address lifestyle factors and nutritional status, including adequate calcium and vitamin D intake, to support bone health alongside thyroid management.
- By adopting a thorough and individualized approach to thyroid testing and management, clinicians can better address the unique challenges posed by thyroid dysfunction in postmenopausal women, improving diagnostic accuracy and patient outcomes.

9. CONCLUSION

Menopausal transition is closely related to thyroid function, which both influences and is influenced by the hormonal changes that occur during this period of life. Thyroid dysfunction and menopause have many symptoms, including mood swings, sleep issues, and metabolic changes, which make diagnosis difficult. In postmenopausal women who arrive with vague symptoms, this calls for increased clinical vigilance and a low threshold for hormonal assessment. The intricacy of detecting thyroid diseases after menopause is highlighted by the physiological relationships between thyroid function and estrogen reduction. Thyroid dysfunctions can be made worse by hormonal changes, which also affect cardiovascular health, metabolic control, and general quality of life. For prompt and efficient treatment, it is crucial to distinguish between menopausal symptoms and those associated with the thyroid. Clinicians should be aware that postmenopausal women have a higher risk of thyroid nodules and cancer, which is partly caused by hormonal changes and extended exposure to estrogen. To reduce the risk of osteoporosis and cardiovascular disorders associated with thyroid dysfunction, especially hyperthyroidism and subclinical hypothyroidism, regular monitoring and customized treatment plans are essential. Furthermore, thyroid problems' effects on quality of life and productivity at work highlight the necessity of thorough diagnostic and treatment methods. For postmenopausal women, routine thyroid monitoring is advised, particularly for those with risk factors including surgical menopause or pre-existing thyroid disorders. Menopausal hormone treatment and other personalized management options should be carefully weighed against the risks and benefits to address the overlapping symptoms. Clinicians should also be aware of any confounding variables in thyroid function testing, such as

changes with age and drug interactions. In general, enhancing the diagnosis and treatment of thyroid dysfunction in postmenopausal women requires a patient-centred, integrated strategy. To improve treatment methods for improving health outcomes in this population, future research should concentrate on clarifying the underlying mechanisms that relate thyroid function to menopausal hormone changes.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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