REVIEW ARTICLE

Thyroiditis with a Focus on Hashimoto's Thyroiditis and Postpartum Thyroiditis: A Rapid Review for Busy Practitioners

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Abstract

Thyroiditis comprises a diverse group of inflammatory thyroid disorders, presenting with a variety of clinical manifestations that range from transient dysfunction to permanent hypothyroidism. This rapid review, "Thyroiditis with a Focus on Hashimoto's Thyroiditis, and Postpartum Thyroiditis," provides a concise and up-to-date summary of the etiology, pathophysiology, clinical manifestations, diagnostic strategies, and treatment approaches for various forms of thyroiditis, with a specific focus on Hashimoto's (HT) and postpartum thyroiditis (PPT). HT is the most common autoimmune disease affecting the endocrine system, yet its presentation, diagnosis, and management can be complex for practitioners. The paper also discusses the management of subclinical and overt hypothyroidism. PPT stands out due to its unique temporal association with childbirth and miscarriage and its triphasic course, characterized by an initial hyperthyroid phase followed by hypothyroidism and euthyroid phase, respectively. We synthesize current evidence to offer practical guidance for busy clinicians, emphasizing key diagnostic markers, treatment considerations for both hyperthyroid and hypothyroid phases, and the importance of long-term follow-up to identify persistent hypothyroidism, a sequela of PPT. By highlighting the distinct features of PPT within the broader context of thyroiditis, this review facilitates timely recognition and optimal care for affected individuals. Furthermore, we briefly touch upon other forms of thyroiditis, such as subacute, drug-induced, infectious/ suppurative, and Riedel/fibrous thyroiditis.

Keywords: *Thyroiditis, Hashimoto's thyroiditis, postpartum thyroiditis, thyroid dysfunction, hyperthyroidism, hypothyroidism, thyroid-stimulating hormone, thyroid receptor antibodies, thyroid peroxidase antibodies.*

INTRODUCTION

Thyroiditis is the inflammation of the thyroid gland that comprises various clinical disorders [1].

This rapid review discusses thyroiditis, in particular, Hashimoto's Thyroiditis (HT) and Postpartum Thyroiditis (PPT) among the adult population. In addition, other types of thyroiditis are examined briefly. Furthermore, this article is intended to assist busy primary care practitioners in acquiring updated knowledge. Internal medicine physicians may benefit from this article.

HT, initially described by Dr Hakaru Hashimoto in 1912, is characterized by the immune system mistakenly attacking the thyroid gland, resulting in persistent inflammation and ongoing degradation of thyroid tissue. Over time, this damage reduces the thyroid's ability to produce hormones, ultimately resulting in a state of hypothyroidism.

Globally, HT prevalence rates differ. In accordance with systematic reviews and meta-analyses, the overall prevalence is 7.5%, although it is higher in low and middle-income groups (11.4%) [2].

PPT describes a thyroid disorder that arises during the first year following childbirth or miscarriage in women who do not have toxic nodules or TSH receptor antibodies present [3]. Thyroid autoimmunity affects 5-20% of women of childbearing age and is more prevalent in women than in men [4]. Though it is not preventable, morbidity/mortality can be reduced by awareness and early diagnosis.

METHODOLOGY

The objective of this rapid review is to summarize the existing evidence on the various types of thyroiditis, in particular HT and PPT. To achieve this objective, an internal protocol was developed to define the research theme, establish inclusion and exclusion criteria, and outline the search strategy. This synthesis approach was selected to ensure transparency and minimize bias. A structured literature search was conducted across PubMed, MEDLINE, the National Library of Medicine, the American Thyroid Association, European Thyroid Association, the British Medical Journal, and US Family Physician databases, covering publications from August 2003 to April 2025. The eligible studies included combinations of terms such as thyroiditis, Hashimoto's thyroiditis, postpartum thyroiditis, autoimmune thyroiditis, and levothyroxine. Case reports, case series, and non-peer-reviewed materials were excluded. Of the 145 products identified, 40 were retained after duplicates were verified and removed. Data were collected and synthesized thematically to provide an overview of the epidemiology, pathophysiology, diagnosis, and treatment of thyroiditis subtypes, with emphasis on the consistency and clarity of the available evidence.

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TYPES OF THYROIDITIS

1. Chronic autoimmune thyroiditis (Hashimoto's thyroiditis)
2. Postpartum thyroiditis
3. Subacute thyroiditis
4. Silent thyroiditis
5. Drug-induced thyroiditis
6. Infectious/suppurative
7. Riedel/fibrous thyroiditis

Chronic Autoimmune Thyroiditis (Hashimoto's Thyroiditis)

Women are 7-10 times more likely to develop HT than men. It is a common autoimmune disease that results from genetic susceptibility, X chromosome inactivation patterns influenced by environmental factors and microbiome composition, and an imbalance in self-tolerance mechanisms [5]. A lumpy, firm, symmetrical, and painless goiter is often the early sign of HT, characterized by varying degrees of lymphocytic infiltration and fibrous changes in the thyroid gland [1]. HT presents with hypothyroidism; occasionally, thyrotoxicosis is caused by the fluctuating effects of stimulating and inhibiting thyroid autoantibodies [3]. Thyroid peroxidase antibody (TPO) and Thyroglobulin antibody levels are inversely associated with the patient's overall health [6]. Approximately 10% of people with chronic autoimmune hypothyroidism have atrophic thyroid glands instead of goiter. This may be the ultimate phase of thyroid dysfunction in HT [7].

Typical symptoms include arthralgia, cognitive impairment, constipation, depression, difficulty concentrating, cold intolerance, dry skin, edema, exhaustion, hair changes (such as dryness, thinning, or loss), weakness, lethargy, infertility, impaired memory, menorrhagia, myalgia, changes in voice, and weight. Common signs include goiter, hoarseness of voice, coarse facies, macroglossia, thin eyebrows, delayed relaxation of deep tendon reflexes, edema, bradycardia, periorbital edema, diastolic hypertension, pleural effusion, pericardial effusion, and hypothermia. Moreover, high C-reactive protein, hyponatremia, hyperprolactinemia, elevated creatine kinase, elevated low-density lipoprotein (LDL) and triglycerides (TG), proteinuria, and low-voltage electrocardiography (ECG) are among the laboratory abnormalities noted [8].

High levels of serum TPO antibodies are found in 90% of individuals diagnosed with Hashimoto's thyroiditis. Individuals with overt hypothyroidism, characterized by raised thyroid-stimulating hormones (TSH) and lowered free T4 levels, should receive treatment with levothyroxine, aiming for a TSH level between 1 and 3 m IU/L [9].

Seronegative HT occurs in 5-10% of patients. Therefore, diagnosing seronegative HT is a bit complicated [5].

Compared to their seropositive counterparts, patients with seronegative HT may exhibit a milder form of hypothyroidism at the time of diagnosis [2].

To determine if the patient has hypothyroidism or hyperthyroidism, TSH should be measured. To confirm autoimmune thyroid illness, thyroid microsomal antibodies (also known as thyroid peroxidase antibodies) and thyroid receptor antibodies can be measured. The C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are significantly increased in bacterial infectious thyroiditis [10]. The normal values of thyroid function tests (TFT) are shown in Table **1**.

Table 1: Reference ranges of TFT for healthy adults [13].

Test	From	To	Units
TSH	0.4	4.0	mU/L (milliunits per litre)
FT4	9.0	25.0	pmol/L (picomoles per litre)
FT3	3.5	7.8	pmol/L (picomoles per litre)

All reference ranges are assay-specific and laboratory-dependent.

Circadian Rhythm

In the early morning, TSH levels peak and then decline, reaching a trough in the early afternoon and evening. Generally, levels are lower in summer and higher in winter. It is best to take a TSH blood sample early in the morning and repeat it at the same time for follow-up visits to assess the response to treatment.

The TSH target ranges from 4 to 6 mIU/L for individuals aged 70 to 80 years. The French Endocrine Society recommends using the patient's age, divided by 10, as the upper limit of normal for TSH (in mIU/L) when screening and following elderly individuals for hypothyroidism [11]. The trimester-specific reference ranges for thyroid-stimulating hormone (TSH) are shown in Table **2**.

Table 2: Trimester-specific reference ranges for thyroid-stimulating hormone [14].

Test	Non pregnant	First Trimester	Second Trimester	Third Trimester
Thyroid-stimulating hormone (mIU per L)	0.3 to 4.3	0.1 to 2.5	0.2 to 3.0	0.3 to 3.0

Taking biotin (vitamin B7) can interfere with thyroid function test results; therefore, it's better to stop the supplement for 3-5 days before thyroid levels are tested for accurate results [12].

The risk of acquiring HT is higher in people with a previous history of autoimmune diseases, in comparison to people without the same [15].

An increased prevalence of autoimmune thyroiditis (AIT) is associated with excessive iodine consumption. Immune dysfunction may be linked to low selenium levels; a lower selenium intake is thought to be a risk factor for the development of autoimmune thyroid diseases (AITD) [16].

The occurrence of another autoimmune disorder is 14.3% among those with HT (P=.005). The most prevalent comorbid autoimmune disorder is rheumatoid arthritis, reported in 4.24% of HT patients [17]. The risks for other autoimmune disorders associated with HT are markedly elevated (>10 for pernicious anemia, systemic lupus erythematosus, celiac disease, Addison's disease, and vitiligo) [18].

Treatment

Drug Treatment

The objective of treatment for hypothyroidism is to achieve a euthyroid condition, reduce hypothyroid symptoms, and avoid overtreatment [19].

The thyroid gland in patients with preserved degree of endogenous function approximately produces 85-100 mcg of T4 per day and 5-6.5 mcg of T3 per 24 hours. The rest of 26.5 mcg/day of T3 is produced by the peripheral conversion of T4 to T3 by type 1 and type 2 deiodinases [16].

Levothyroxine should be given once daily, 30 to 60 minutes before meals. In nonpregnant patients, TSH should be checked every 6-8 weeks if within the normal range, and then every 6-12 months. If no change in their clinical condition is observed, referral to an endocrinologist is required [5].

Adults under 60 years with primary hypothyroidism should begin taking 1.5 to 1.8 mcg of levothyroxine per kilogram bodyweight, whereas 60 years of age or older adults or patients with ischemic heart disease are advised to start with 12.5 to 50 mcg per day and increase it every 2 to 3 weeks to slowly bring TSH down to the target value [11]. For pregnant women, a previously steady dosage is increased to 9 weekly doses, or the dosage is increased by 30-50% at the start of pregnancy, and referral to an endocrinologist is advised [5, 20].

Screening for Vitamin D if clinically indicated, followed by supplementation, may help patients with HT. The evidence shows that TPO antibody and Thyroglobulin antibody levels are significantly reduced following 6 months of vitamin D intake [21].

Counselling

The patients are informed that thyroid disease usually responds to treatment, so compliance with drug treatment is the mainstay of treatment. Guidance is given to patients to take levothyroxine on an empty stomach. Timely follow-up and monitoring of TSH levels will reduce risks of under- and over-treatment [22].

Dietary Modifications

The evidence suggests that anti-inflammatory nutrients, such as vitamin D, antioxidants, monounsaturated and polyunsaturated fatty acids, magnesium, and zinc, are essential for reducing thyroid inflammation [21]. Thyroid hormone synthesis and metabolism are enhanced by iodine and selenium. Due to the high prevalence of anemia and cardiovascular disease in HT patients, adequate intake of iron, folic acid, and vitamin B12 is necessary. Recent studies suggest that those with food intolerances and celiac disease should avoid lactose and gluten. Restricting pro-inflammatory foods such as sweets, refined carbohydrates, and saturated fats is the foundation of a well-balanced diet. [16, 21, 23] A proper diet with iron, folic acid, and vitamin B12 is particularly required because this group of individuals with HT has a high prevalence of anemia and cardiovascular disease [16]. Also, regardless of celiac disease, patients with HT benefit from a gluten-restricted diet as far as the progression and the possible disease complications are concerned [2].

The World Health Organization (WHO) recommends 250 micrograms of iodine per day for pregnant and lactating women. The WHO has declared that a daily iodine intake of more than 500 micrograms/day may be excessive during pregnancy. The WHO criteria are supported by recent population data [16, 20, 24]. To achieve the appropriate iodine levels, seafood (for instance, sardines, shrimp, seaweed, scallops, cod, salmon, and tuna), animal products (for example, eggs, yogurt, and cow's milk), and fruits (such as strawberries and cranberries) are recommended as the primary sources of iodine in the diet [23].

Rock salt lacks iodine; however, it is mistakenly believed to have health benefits, such as lowering blood pressure and relieving colds and coughs. These health benefits are unproven and may raise blood pressure by increasing daily salt intake, due to the misconception that salt lowers blood pressure. Minerals such as iron, potassium, zinc, and calcium have been detected in rock salt; however, they are present only in trace amounts and don't significantly contribute to its health benefits [25].

Many nutrients, including zinc, magnesium, curcumin, selenium, iron, B-complex vitamins, and vitamin D, have been found to help treat HT. In the treatment of HT, there is evidence that selenium (Se) supplements may be helpful as an adjuvant therapy to levothyroxine [23].

Subclinical Hypothyroidism

Subclinical hypothyroidism is a biochemical result of a normal FT4 level and an elevated TSH level. There may or may not be present hypothyroidism symptoms and signs. TSH frequently returns to normal on its own after 12 months or longer [1].

TSH levels above 10 mIU/L are associated with an increased risk of heart failure, ischemic heart disease, and fractures compared to those with a level between 2.0 and 2.5 mIU/L. In nonpregnant women with subclinical hypothyroidism, levothyroxine therapy should be considered if the TSH level is more than 10 mIU/L or if the TPO antibody level is elevated. Subclinical hypothyroidism patients who are pregnant or trying to

Table 3: Comparison of clinical guidelines for the management of subclinical hypothyroidism [26-28].

Guidelines	Consider Levothyroxine Treatment	Observe without Levothyroxine Treatment
American Thyroid Association (ATA) guidelines (2012) [26].	TSH >10 mIU/L, age <70 years	TSH <10 mIU/L, age >70 years
	TSH 4-10 mIU/L, age <65 years, plus symptoms	TSH 4-10 mIU/L, age >65 years
European Thyroid Association (ETA) guidelines (2013) [27].	TSH >10 mIU/L, age <70 years	TSH <10 mIU/L, minus symptoms, age <70 years
	TSH <10 mIU/L, age <70 years, plus symptoms	TSH <10 mIU/L, age >70 years
	TSH <10 mIU/L, age >70 years, plus symptoms or high cardiovascular (CV) risk	
National Institute for Health and Care Excellence (NICE) guidelines 2018 [28].	TSH >10 mIU/L, age <70 years	TSH >10 mIU/L, age >70 years
	TSH 4-10 mIU/L, age <65 years, plus symptoms	TSH 4-10 mIU/L, age >65 years

conceive should also be considered for drug treatment with levothyroxine [1]. The comparison of different clinical guidelines for the management of subclinical hypothyroidism is shown in Table **3**.

Postpartum Thyroiditis

PPT is a painless condition [29] and has an autoimmune etiology [5]. PPT presents as an isolated hyperthyroidism (thyrotoxicosis); hyperthyroidism succeeded by hypothyroidism; or hypothyroidism alone within one year of parturition. Postpartum thyroiditis is either a transient or persistent thyroid disorder that occurs within one year of childbirth or miscarriage [9].

According to global epidemiological research, the average prevalence of PPT is around 7% [30]. However, the prevalence varies from 1.1% to 11.4%. This difference is likely attributable to variations in ethnicities, geographic regions, and iodine intake. In the general population, postpartum thyroiditis has a lifetime incidence of 5.4% [18]. The disease has a significant probability of recurrence in future pregnancies [31].

Postpartum hypothyroidism is usually temporary; 70 to 80% of those with the condition ultimately no longer need medication because their thyroid starts making hormones at normal levels again [32]. For women diagnosed with PPT once, there is a high risk of up to 70% of developing the condition again in subsequent pregnancies [18]. It has also been reported that 15% to 50% of patients having a history of PPT will eventually develop permanent hypothyroidism [18]. An annual TSH test should be performed on individuals with a history of postpartum thyroiditis to evaluate permanent (persistent) hypothyroidism, and it should be continued for 5 to 10 years [33].

The PPT's hyperthyroid phase is brought on by the thyroid gland's autoimmune destruction, which releases thyroid hormone that has been stored; therefore, antithyroid drugs are usually ineffective. Peripheral beta antagonists are generally used to treat symptoms [29]. One potentially significant etiological factor for the cause of postpartum psychosis is autoimmune thyroid disease. Thus, it is necessary to screen thyroid peroxidase antibodies in patients who have postpartum psychosis [34]. Evidence also shows smoking plays a role in modifying immune tolerance to thyroid autoantigens and is considered a separate risk factor for PPT development (relative risk 3.1) [4].

Clinical Features

Atotal of 25% to 40% of patients with postpartum thyroiditis exhibit a triphasic pattern that is hyperthyroidism (weight loss, palpitations, increased hunger, nervousness, irritability, tremors of the hands, and sweating), followed by hypothyroidism (tiredness, weight gain, increased sensitivity to cold, constipation, dry skin, puffy face, hoarse voice, coarse hair and skin, irregular and heavier menses, loss of concentration, and depression) and then euthyroid phase. During the thyrotoxic phase, symptoms typically start two to six months after giving birth and are mild. This stage lasts for two to three months. The hypothyroid phase remains for three to twelve months and is usually symptomatic. While 40% of patients present with isolated hypothyroidism, they eventually become euthyroid around one year later [18].

Antibody testing is beneficial for distinguishing PPT from Graves' disease. The TRAb is a very sensitive and specific biomarker for Graves' disease; however, both Graves' disease and postpartum thyroiditis can cause high serum levels of TPO antibodies. Because of preferential T3 production, the T3 to T4 ratio is frequently greater (>20:1) in Graves' disease [35]—the clinical and laboratory features of Graves' disease and postpartum thyroiditis are shown in Table **4**.

Table 4: Clinical and laboratory features of Graves' disease and postpartum thyroiditis [30].

Characteristic	Graves' Disease	Postpartum Thyroiditis
Frequency	Common	Most common
Timing	4-7 months postpartum	2-6 months postpartum
Symptoms	Hyperthyroidism	Mild hyperthyroidism
Signs	Goiter, exophthalmos, hyperthyroidism	Goiter
Radioiodine uptake	High	Low
Triiodothyronine/ thyroxine ratio	High	Low
Thyroid peroxidase antibodies	High	High
Thyrotropin receptor antibodies	Present	Absent or slightly positive

Diagnosis

Patients with PPT frequently have elevated TPOAb and thyroglobulin antibodies; slightly positive TRAb have also been seen. Preformed thyroid hormones are released in postpartum thyroiditis, and the T4:T3 ratio is usually higher in this condition compared to Graves 'disease. PPT lacks clinical signs of Graves' disease, such as thyroid bruit and exophthalmos. Radioactive iodine (RAI-123) scans show reduced uptake in PPT, whereas Graves' disease shows enhanced, diffuse uptake. Pregnant or nursing women should not undergo this type of imaging modality [36]. If nursing mothers undergo a radioactive iodine uptake test, they must pump and discard milk for 2 days after RAI I 123 administration [33].

Individuals with a past medical history of thyroid disorders, type 1 diabetes mellitus (T1DM), gestational diabetes, chronic hepatitis C, autoimmune conditions, or a positive family history of thyroid issues are more likely to develop PPT. They should be tested for TSH levels between 6 and 12 weeks after giving birth [37].

Predictors of Permanent Hypothyroidism in Women with Postpartum Thyroiditis (PPT)

- Severity (TSH >20 mU/L) of hypothyroid phase of PPT
- Highly positive antithyroid peroxidase antibody (TPOAb) [38]

Treatment

The counselling of pregnant women about PPT is required, particularly those at high risk (highly positive thyroid peroxidase antibodies (TPOAb)). The approach focuses on awareness of symptoms, the condition's typical three-phase course, and the need for postpartum monitoring.

Treatment is usually not required if hyperthyroidism (thyrotoxicosis) symptoms are mild during the initial phase of PPT [14]. Patients experiencing symptoms during the thyrotoxic phase are treated with beta blockers (*e.g.*, propranolol, 10 to 20 mg every 8 hours), titrated to a dosage that relieves symptoms. Antithyroid drugs are not recommended because postpartum thyroiditis is a destructive process [29].

Levothyroxine is advised for the treatment of the hypothyroid phase, particularly if the patient is exhibiting symptoms, trying to conceive, or breastfeeding. The thyroid function test should be repeated every 4 to 8 weeks. If the patient is not pregnant, trying to conceive, or breastfeeding, it is advised to start tapering the levothyroxine dosage about 12 months after postpartum, and then periodically follow up with thyroid function tests [10]. Typically, thyroid function levels normalize within 12 to 18 months in most individuals with postpartum thyroiditis [39].

Women exhibiting symptoms during the hypothyroid phase of PPT should have levothyroxine administered. Levothyroxine treatment is not necessary for asymptomatic females with TSH levels less than 10 mU/L who do not intend to become pregnant within the following year. They should be reassessed in 4 to 6 weeks. Levothyroxine should be administered to all individuals with TSH >10 mU/L and to women with subclinical hypothyroidism who are trying to conceive [30].

Subacute Thyroiditis

Subacute (granulomatous) thyroiditis, commonly referred to as de Quervain thyroiditis, is believed to be an inflammatory response triggered by a viral infection [31]. Painful subacute thyroiditis is a self-limiting inflammatory condition [33]. Various viruses have been associated with the onset of subacute thyroiditis, such as mumps, coxsackie, influenza, and echoviruses [40]. SARS-CoV-2 has also been associated with subacute thyroiditis [38]. The neck pain can be bilateral or unilateral and may extend to the jaw. Patients also report dysphagia, along with increased sweating, tremors, and weight loss [1]. As many as 50 percent of patients experience symptoms of thyrotoxicosis, [35] which usually last for three to six weeks. After the thyrotoxic phase, about one-third of patients experience hypothyroidism, which may persist for as long as six months [18]. Most patients achieve a state of euthyroidism within a year of the disease's onset. Permanent hypothyroidism occurs in approximately 5% to 15% of patients [29]. Subacute thyroiditis is characterized by a markedly elevated erythrocyte sedimentation rate (ESR); therefore, C-reactive protein (CRP) is also elevated [1].

Silent Thyroiditis

It includes painless sporadic thyroiditis, silent sporadic thyroiditis, and subacute lymphocytic thyroiditis. Its etiology is autoimmune in nature, and it may occur as solitary hyperthyroidism or hyperthyroidism succeeded by hypothyroidism or isolated hypothyroidism [1].

Drug-Induced Thyroiditis

Many drugs, such as amiodarone, interferon-alfa, immune checkpoint inhibitors, interleukin-2, tyrosine kinase inhibitors, and lithium, have been linked to the onset of thyroiditis. The management approach starts with stopping the medication and treating the immediate symptoms [1].

Infectious/Suppurative

Commonly causing organisms are the Staphylococcus aureus and Streptococcus species. It leads to swelling, anterior neck pain, painful swallowing, fever with chills, tenderness, and lymphadenopathy. Management is done through hospitalization [18].

Riedel Thyroiditis (Fibrous Thyroiditis) Autoimmunity might play a role in disease development. Destructive thyroiditis features thick fibrosis that can spread into nearby tissues. Diagnosis is accomplished through thyroid biopsy [30].

ANOTHER WAY TO CLASSIFY THYROIDITIS: PAINFUL AND PAINLESS THYROIDITIS

The painful thyroiditis comprises subacute, infectious, radiation, trauma-induced thyroiditis, and rarely Hashimoto's thyroiditis. The painless group comprises postpartum thyroiditis, drug-induced thyroiditis, fibrous thyroiditis, and subacute lymphocytic thyroiditis [29].

CONCLUSION

This rapid review aims to compile updated clinical data about the two most prevalent types of autoimmune thyroiditis: Hashimoto's thyroiditis (HT) and postpartum thyroiditis (PPT). Existing guidelines confirm that the diagnosis of HT remains primarily dependent on the presence of TPO antibodies. High levels of serum TPO antibodies are found in 90% of individuals diagnosed with Hashimoto's thyroiditis, whereas seronegative HT can occur in 5-10% of patients. Therefore, the diagnosis of seronegative HT is complicated. The long-term management of HT has not dramatically shifted, but a growing consensus supports a personalized approach to levothyroxine initiation, particularly in symptomatic patients and women planning pregnancy.

For PPT, our findings highlight that it is often transient in nature, but also a significant proportion of women (approximately 15-50%) develop permanent hypothyroidism within a decade. Available clinical data confirms that a high baseline (TSH >20 mU/L) during the hypothyroid phase of PPT and high TPO antibodies titre remains the most robust predictor of this permanent defect. An annual TSH test should be performed on individuals with a history of postpartum thyroiditis to evaluate permanent hypothyroidism, and it should be continued for 5 to 10 years. This mandates that clinicians implement structured, long-term follow-up for all patients diagnosed with PPT, emphasizing patient education on symptom recurrence and the need for annual screening.

Further studies are needed to evaluate non-hormonal, adjunctive therapies for HT aimed at reducing the autoimmune burden and preserving thyroid function, rather than solely replacing the lost hormone. By addressing these areas, the clinical management of both Hashimoto's and Postpartum thyroiditis can evolve from reactive hormone replacement to proactive disease modification and prevention. Future research must focus on establishing validated risk scores for predicting permanent hypothyroidism in PPT beyond simple serum TSH and TPO antibodies status, perhaps incorporating genetic or immunological markers.

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None.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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