



HYPOTHYROIDISM AS MANIFESTATION OF POSTPARTUM THYROIDITIS

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Article History :

Received date : 2026/02/08
Revised date : 2026/03/17
Accepted date : 2026/04/21
Published date : 2026/05/30



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E-ISSN :

ISSN 3048-1368



P-ISSN

ISSN 3048-1376



ABSTRACT

Introduction:

Postpartum thyroiditis (PPT) is a destructive autoimmune inflammatory disorder of the thyroid gland that occurs within one year after delivery in women without a prior history of thyroid disease. The condition is caused by an autoimmune process that induces thyroid follicular damage and thyroglobulin proteolysis. Clinically, PPT typically presents with a triphasic pattern, beginning with a transient thyrotoxic phase, followed by hypothyroidism, and ultimately progressing toward recovery to a euthyroid state.

Case Illustration: A 37-year-old woman presented with a gradually enlarging anterior neck swelling without associated pain. The patient had no previous history of thyroid disease or autoimmune disorders. Five months before clinical assessment, she had delivered her third child. Thyroid function tests revealed hypothyroidism, while thyroid ultrasonography demonstrated findings consistent with thyroiditis. The patient was subsequently

treated with levothyroxine and scheduled for monthly follow-up evaluations. Following treatment, thyroid hormone levels returned to within normal limits, and the neck swelling gradually decreased.

Discussion:

The hypothyroid phase of postpartum thyroiditis (PPT) may occur due to depletion of thyroglobulin stores and cessation of the proteolytic process, resulting in decreased serum T3 and T4 levels. Patients with symptomatic hypothyroidism, as well as asymptomatic individuals with TSH levels >10 mU/L, are recommended to receive levothyroxine therapy at a dose of 50–100 mcg daily accompanied by periodic thyroid function monitoring. Most patients recover completely and return to a euthyroid state within one year. However, persistent hypothyroidism may develop in up to 50% of patients despite initial clinical improvement.

Conclusion: The diagnosis of postpartum thyroiditis remains challenging because its clinical manifestations are often subtle and nonspecific. Therefore, a multidisciplinary approach is essential to ensure continuity of thyroid disorder management from pregnancy through the postpartum period.

Keyword: Hypothyroidism, postpartum, thyroiditis

INTRODUCTION

Postpartum thyroiditis (PPT) is a destructive autoimmune inflammatory disorder of the thyroid gland that occurs within one year after delivery in women without a prior history of thyroid disease. The condition may also develop following miscarriage or in women previously treated with levothyroxine for underlying Hashimoto's thyroiditis^{3,6,7,10}. The reported prevalence of PPT ranges from 1–21%, with approximately one in 20 pregnant women experiencing thyroid dysfunction during the postpartum period^{1,4,8}. The risk of developing PPT is known to be higher in individuals with other autoimmune disorders, such as type 1 diabetes mellitus, as well as in patients with a personal or family history of thyroid disease^{3,7}. Clinically, PPT generally follows a triphasic course characterized by an initial transient thyrotoxic phase, followed by hypothyroidism, and eventually a return to a euthyroid state. Although most cases resolve spontaneously within 12 months, some patients may progress to permanent hypothyroidism^{3,6}.

CASE ILLUSTRATION

A 37-year-old woman presented with a complaint of anterior neck enlargement that had gradually developed over the previous week. The patient first noticed the swelling while looking in the mirror. The enlargement was not accompanied by pain or erythema and did not cause dysphagia or dyspnea. The patient denied symptoms suggestive of hyperthyroidism, including palpitations, tremor, weight loss, increased appetite, excessive sweating, and heat intolerance. She also denied symptoms associated with hypothyroidism, such as fatigue, excessive hair loss, impaired concentration, sleep disturbances, depression, mood changes, cold intolerance, and weight gain.

The patient denied any previous history of similar complaints or recent infections. There was no history of chronic disease, thyroid disorders, or autoimmune diseases, including type 1 diabetes mellitus and systemic lupus erythematosus (SLE). She also denied any history of neck surgery or long-term hormonal medication use. The patient had delivered her third child via

uncomplicated cesarean section five months prior to the current evaluation. No family history of similar complaints, autoimmune disease, or malignancy was reported.

Physical examination revealed a moderately ill general condition with good nutritional status. The patient weighed 50 kg and measured 155 cm in height, with a body mass index of 20.8 kg/m². She was fully conscious (*compos mentis*) with a blood pressure of 110/70 mmHg, regular pulse rate of 90 beats/minute with adequate volume, respiratory rate of 20 breaths/minute, axillary temperature of 37°C, oxygen saturation of 99%, and a visual analog scale (VAS) score of 0/10. Ophthalmologic examination demonstrated non-anemic conjunctivae, non-icteric sclerae, and absence of exophthalmos. Jugular venous pressure (JVP) was within normal limits, and no cervical or axillary lymphadenopathy was detected. Examination of the anterior neck revealed mild diffuse enlargement. Palpation demonstrated grade IB thyroid enlargement with a soft-elastic consistency, smooth surface, absence of nodules, no tenderness, and no associated lymphadenopathy. Thoracic and abdominal examinations were within normal limits. Examination of the extremities revealed warm acral temperature without edema or tremor.

Laboratory investigations demonstrated a TSHs level of 82.99 uIU/mL and an FT4 level of 6.51 pmol/L. Thyroid ultrasonography revealed enlargement of both thyroid lobes accompanied by hypervascularization suggestive of thyroiditis. Based on the clinical history, physical examination, and supporting investigations, the patient was diagnosed with hypothyroid-phase postpartum thyroiditis. The patient was subsequently treated with levothyroxine 100 mcg once daily and scheduled for routine monthly follow-up. Following treatment, thyroid hormone levels returned to within normal limits, and the anterior neck enlargement gradually regressed.

DISCUSSION

Postpartum thyroiditis (PPT) is an inflammatory disorder of the thyroid gland that occurs following childbirth and may result in either thyrotoxicosis or hypothyroidism⁹. The exact etiology of PPT remains unclear; however, it is widely considered an autoimmune condition with a pathophysiological mechanism similar to that of Hashimoto's thyroiditis^{5,9}. The autoimmune

process leads to thyroid follicular destruction and thyroglobulin proteolysis. During pregnancy, the immune system undergoes physiological suppression and subsequently becomes reactivated after delivery. This immune reactivation induces damage to thyroid follicular cells, resulting in the uncontrolled release of preformed thyroid hormones into the circulation. Excessive release of thyroxine (T4) and triiodothyronine (T3) produces a transient hyperthyroid phase that persists until thyroglobulin stores are depleted. During this phase, synthesis of new thyroid hormones is suppressed, and serum TSH levels decrease due to elevated circulating T3 and T4 levels. Thyroid hormone synthesis gradually resumes once the inflammatory process subsides^{3,6}. Furthermore, postpartum thyroiditis has been associated with several human leukocyte antigen (HLA) haplotypes, particularly HLA-DR3, HLA-DR4, and HLA-DR5 within class II regions, as well as HLA-B8 and HLA-Bw35 within class I regions. These genetic markers are associated with an increased risk of developing permanent hypothyroidism following the postpartum period^{3,12}.

Clinically, PPT may occur in two phases, although not all patients experience both phases. Approximately one-third of patients experience both thyrotoxic and hypothyroid phases, whereas another one-third experience only either the thyrotoxic or hypothyroid phase alone^{4,7,8,9}. The hyperthyroid phase generally occurs within the first to fourth month postpartum and may be asymptomatic or accompanied by symptoms such as palpitations, anxiety, insomnia, heat intolerance, weight loss, and fatigue. In contrast, the hypothyroid phase typically develops between the fourth and eighth month postpartum and is characterized by fatigue, cold intolerance, weight gain, and depression^{3,7,9}. Most patients return to a euthyroid state within 12 months; however, approximately 20–50% of cases may progress to permanent hypothyroidism. In addition, PPT may adversely affect cardiovascular, neurological, and metabolic function^{3,6}.

The diagnosis of postpartum thyroiditis is established based on clinical manifestations and thyroid function testing. Physical examination should include evaluation of vital signs to identify tachycardia, bradycardia, or hypertension; assessment of body weight and body mass index (BMI) to detect weight changes; observation of general clinical features such as diaphoresis, tremor, exophthalmos, and dry skin; as well as inspection and palpation of the thyroid gland to evaluate enlargement or nodules^{8,10}. The thyroid gland in PPT is generally non-tender. Physical examination

is also important for assessing the severity of hyperthyroidism. In more severe cases, arrhythmias such as atrial fibrillation, signs of right-sided heart failure, elevated body temperature, and altered mental status may be observed³.

Current guidelines from the American Thyroid Association (ATA) and the Endocrine Society do not recommend routine thyroid function screening in asymptomatic postpartum women. Nevertheless, serum TSH measurement at 6–12 weeks postpartum may be considered in high-risk populations, including patients with a previous history of PPT, Graves' disease, Hashimoto's thyroiditis receiving thyroid hormone replacement therapy, positive thyroid peroxidase antibodies (TPOAb), type 1 diabetes mellitus, non-thyroid autoimmune diseases, chronic hepatitis C infection, postpartum depression, or lactation difficulties^{5,8,10,11}. Measurement of thyroid antibodies, including TPOAb and thyroid-Stimulating Hormone Receptor Antibody (TRAb) or Thyroid-Stimulating Immunoglobulin (TSI), together with thyroid ultrasonography, may assist in differentiating PPT from Graves' disease⁸. In selected cases, radioactive iodine uptake and thyroid scintigraphy may also be utilized. Low radioactive iodine uptake is typically observed during the thyrotoxic phase of PPT, whereas increased uptake is characteristic of Graves' disease. However, these imaging modalities are contraindicated in pregnant and breastfeeding women. Therefore, TRAb measurement alone is generally sufficient to distinguish between the two conditions^{6,7}.

Levothyroxine therapy at a dose of 50–100 mcg daily is recommended in patients with TSH levels >10 mIU/L, regardless of symptom presence. In asymptomatic patients with TSH levels between 4–10 mIU/L, the Endocrine Society recommends repeat TSH evaluation within 4–8 weeks prior to initiating therapy³. Levothyroxine therapy should also be considered in patients with significant hypothyroid symptoms, breastfeeding women, or those planning another pregnancy in the near future. In addition, patients should receive counseling regarding contraceptive use during the monitoring period. After 6–12 months of therapy, levothyroxine dosage may be gradually tapered or discontinued to assess recovery of thyroid function, with serum TSH monitoring every 6–8 weeks. If treatment is deferred, thyroid function should still be periodically reassessed every 4–8 weeks until the patient achieves a euthyroid state^{3,6,8}. All women with PPT, including those with overt or subclinical hypothyroidism, whether treated or untreated with levothyroxine, require long-

term follow-up because of the persistent risk of permanent hypothyroidism². In patients who have returned to a euthyroid state, periodic TSH monitoring is recommended until 12 months postpartum and annually thereafter. These recommendations are based on the high lifetime risk of permanent hypothyroidism and recurrence rates of up to 70% in subsequent pregnancies⁸.

Antithyroid medications are not recommended during the thyrotoxic phase of postpartum thyroiditis because the condition results from the release of preformed thyroid hormones rather than increased thyroid hormone synthesis. Once the thyrotoxic phase subsides, serum TSH levels should be reevaluated within 4–8 weeks, or earlier if new symptoms arise, to detect the potential development of hypothyroidism, which commonly occurs between the fourth and eighth month postpartum. Patients with symptomatic thyrotoxicosis may receive beta-blocker therapy to alleviate symptoms such as palpitations, tremor, and shakiness. Medication dosage should then be gradually reduced as symptoms improve because the thyrotoxic phase of PPT is transient. Propranolol and metoprolol may be safely administered in breastfeeding women due to minimal excretion into breast milk^{1,3,8,9}.

CONCLUSION

The diagnosis of postpartum thyroiditis remains challenging because its clinical manifestations are often subtle and nonspecific. Therefore, a multidisciplinary approach is essential to ensure continuity of thyroid disorder management from pregnancy through the postpartum period.

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