Postpartum Thyroiditis – Mechanisms, Diagnosis, and Treatment

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Abstract

Introduction:

Postpartum thyroiditis is a common form of thyroid illness that usually happens in the first year after giving birth. It appears as short-term high or low thyroid activity. The time right after having a baby is seen as a period with more risk for getting autoimmune illnesses. These issues may get better during pregnancy but often become worse or show up for the first time after childbirth due to reactivation of Th1-dominant immune answers. Postpartum thyroiditis is the most usual autoimmune illness, affecting about half of women who have thyroid peroxidase antibodies found in early pregnancy.

Purpose:

This review aims to advance knowledge and improve the treatment of postpartum thyroiditis by summarizing current guidelines and presenting practical solutions for clinical practice.

Material and Methods:

In this article, we provide a comprehensive overview of postpartum thyroiditis. We analyze current research and present the main characteristics of the disease, including its symptoms, causes, diagnostic techniques, treatment plans, and future prospects.

Discussion:

Postpartum thyroiditis remains a serious medical problem due to its ambiguous and variable course, including typical symptoms of hyperthyroidism and of hypothyroidism. These vague symptoms lead to delays in diagnosis, which can lead to permanent organ damage and a serious decline in quality of life, as well as long-term complications for the mother. Even with treatment, which can help slow the disease and control its progression, the duration of the disease and the difficulty in selecting and thoroughly examining women during pregnancy and the postpartum period, which would allow for earlier diagnosis. In turn, accurate and early diagnostics combined with hormonal diagnostics of women before planned pregnancy are methods that reduce the risk of long-term problems and improve the prognosis.

Keywords:

‘Postpartum thyroiditis’, ‘hypothyroidism’, ‘hyperthyroidism’, ‘autoimmune markers’.

Introduction:

Postpartum thyroiditis (PPT) is a frequently encountered thyroidal disorder that typically presents within 1 year following delivery. It refers to the transient appearance of hyperthyroidism, hypothyroidism, or hyperthyroidism progressing into hypothyroidism. [1]

Postpartum (PP) period is regarded as a period of vulnerability to develop or relapse some autoimmune diseases (ADs), especially cytokine-mediated excessive T helper 1 (Th1) cell-mediated AD (e.g., rheumatic arthritis (RA), multiple sclerosis (MS), postpartum thyroiditis (PPT). These skin conditions commonly resolve during pregnancy, but then recur, or present for the first time, after delivery. This is seen to be because of the return of Th1-based immunity, which is lessened during pregnancy. [1,2]

The most common postpartum autoimmune illness (AD) is postpartum thyroiditis (PPT), a thyroid issue that happens in the first year after birth or loss of pregnancy. PPT takes place in the early time after giving birth in about half of women who have autoantibodies to the enzyme thyroid peroxidase during the first part of pregnancy. Thyroid peroxidase is an enzyme important for making thyroid hormone, but many women with this autoantibody stay normal. PPT often has three parts: going from normal function to a short time of high activity (22%), then a longer time of low activity (48%) and back to normal again. [2]

Purpose:

This write-up hopes to help people understand postpartum thyroiditis, looking at signs, how common it is, genetic reasons, body functions and the body's immune system issues, plus ways to find and treat it. The main aim is to raise knowledge about this illness, help with spotting it early on, and enhance treatment choices for everyone. It also wants to steer the use of proven practices into everyday health care while dealing with problems like taking medicine regularly and handling long-term issues often linked with PPT; in the end hoping for better results for patients.

Material and methods:

This study is a systematic review of the existing literature on the diagnosis and treatment of postpartum thyroiditis. A comprehensive search of PubMed, Embase, and Scopus databases was conducted to identify peer-reviewed articles, clinical trials, and reviews published between 1990 and 2025. Keywords were used to identify studies addressing the clinical manifestations of postpartum thyroiditis, including title and abstract searches for "postpartum thyroiditis,"

"hypothyroidism," "hyperthyroidism," "autoimmune markers," and "thyroiditis." We included studies that provided information on diagnostic criteria, long-term complications, treatment, and patient outcomes, sought publication in languages other than English, or did not provide original data. Data extraction focused on procedural procedures, treatment efficacy, and long-term outcomes, particularly regarding the risk of postpartum thyroiditis in subsequent pregnancies. Results were then collated to highlight emerging trends and improve understanding of postpartum thyroiditis management.

Etiology:

PPT is an autoimmune disease that develops in the first year of delivery, when the immune system rebounds after pregnancy-induced immunosuppression. The association of PPT with antithyroid antibodies, prominent T-cell defects, and a histologic picture resembling that of thyroiditis strongly indicates an immunologic pathophysiology for PPT. Further evidence in favor of an autoimmune cause is the presence of a higher frequency of HLA-DR-3, 4, 5 antigens in women who are affected by postpartum thyroiditis. Women who show thyroid peroxidase or thyroglobulin in the first trimester of pregnancy have a 33–50% likelihood of experiencing PPT. Conversely, women with negative thyroid antibody titers have a 0–5% likelihood of experiencing thyroid dysfunction post-delivery. Additionally, a higher thyroid antibody titer increases the chances of developing PPT. Studies indicate a notable decrease in thyroid antibody titers as pregnancy advances – around 25% of women who show positive antibody titers in the first trimester have undetectable levels by the third trimester. [3]

PTT has been observed more often in women possessing the HLA-DR3, DR4, or DR5 phenotypes. These results are comparable to those seen in women with Hashimoto's thyroiditis. PTT has also been noted in individuals with Graves' disease and primary autoimmune hypothyroidism. Typically, PPT happens in women who have positive antithyroid antibodies (TPOAb) during early pregnancy. TPOAb levels decrease during pregnancy, but increase notably and swiftly in the postpartum phase, similar to all immunoglobulin G antibodies. TPOAb antibodies frequently attach to complement, leading to initial cell damage. The involvement of complement in the progression of PPT is reinforced by the finding that TPOAb-positive women who experience PPT exhibit increased complement activation compared to TPOAb-positive women without PPT. Lymphocytic infiltration in the thyroid is the primary pathological characteristic of PPT. Nevertheless, no change in the T to B lymphocyte ratio is seen in peripheral blood. Biopsies within the thyroid of women with PPT have shown a higher

CD4+/CD8+ ratio and heightened T lymphocyte activation. T cell clones responsive to thyroglobulin (Tg), TPO, and the TSH receptor probably grow in the thyroid during the initial stage of PPT. [4,5]

Nonetheless, the function of complement-binding antibodies targeting thyroid peroxidase (TPO), IgG subclasses of TPO, and NK cells remains ambiguous, with inconsistent data found across these domains. All these potential mechanisms that can lead to the onset of PPT are temporary, as in most women, the condition does not become chronic and thyroid function normalizes again. Antibody titers after childbirth usually surpass those seen in the first trimester, with a return to baseline levels occurring roughly one year following delivery. [3,4]

Epidemiology:

The occurrence of after-birth thyroiditis is different in various groups, from 1. 1% to 16. 7%. The issue is famous for its high return rate—about 70% of ladies who had after-birth thyroiditis before getting it again in later births. Also, women with good thyroid peroxidase antibodies who did not have after-birth thyroiditis in their first birth face roughly a 25% chance of getting the illness in another pregnancy. Unlike, women who do not have thyroid antibodies and do not have a past of PPT in their first pregnancy usually do not get the issue in later pregnancies. [3,6]

Many things have been found to play a part in causing PPT, like having thyroid autoantibodies (mostly TPOAb, but also thyroglobulin antibodies), a past case of postpartum thyroiditis, type 1 diabetes mellitus (because it's autoimmune), and a family history of thyroid issues. On the other hand, things like smoking, factors related to childbirth, mother’s iodine level and baby’s sex do not seem to affect the chance of getting PPT. [7]

Cases of PPT have also been mentioned after a miscarriage, but the exact rate in this situation is not clear. It is thought that even short pregnancies might cause enough immune changes to start painless thyroiditis after birth. [3,6]

The least time of being pregnant needed to start these immune changes, as well as how bad the symptoms felt by women with PPT, are key areas for future study. It is known that women who have thyroid autoantibodies are at higher risk of getting postpartum thyroid problems, which hints that the real number of PPT in this group may be lower than thought. Since many of these women want more pregnancies later, early testing of thyroid health is suggested to improve mother’s health before fetal thyroid growth begins. [6]

Symptoms:

The clinical progression of PPT differs but is similar to subacute thyroiditis without experiencing thyroid pain. A typical triphasic pattern with temporary thyrotoxicosis, then a hypothyroid stage, and finally a return to euthyroidism is observed in about 20% of patients. Thyrotoxicosis, resulting from the secretion of preformed thyroid hormone by the thyroid gland, usually persists for 2–3 months, and the ensuing hypothyroidism typically resolves in 6–8 months. Nonetheless, around 20% of women face temporary hypothyroidism, while the other 23% encounter solely temporary hyperthyroidism. While 80% of women facing the hypothyroid stage of PPT will return to a euthyroid state, about 20–50% of individuals may ultimately experience lasting hypothyroidism after 3–12 years of monitoring. The occurrence of asymptomatic hyperthyroidism is 33%. Hyperthyroidism that is not treated typically resolves on its own in 2–3 months. [2,7]

Symptoms of PPT encompass the usual indicators of both hyperthyroidism and hypothyroidism. Identifying symptoms as signs of a pathological process relies on the severity of thyroid dysfunction and whether the patient and/or physician unknowingly link the symptoms to the stress of caring for a newborn.The hormonal disturbances in the hyperthyroid phase of postpartum thyroiditis (PPT) are generally less intense than those in the hypothyroid stage. The hyperthyroid stage, in fact, is often identified only after the diagnosis of hypothyroidism has been made. Patient history, carefully reviewed, might have indicated hyperthyroidism signs that were transient during the first two to six months following the delivery. More frequently mentioned by patients in this hyperthyroid phase than in postpartum women with normal thyroid function are the following symptoms: tiredness, rapid heart rate, irritability, heat intolerance, and nervousness. Contrarily, during the hypothyroid phase, the affected women have trouble concentrating, lack of focus, lower energy levels, memory problems, dry skin, thinning hair, increased cold sensitivity, and various aches. The symptoms occur more frequently than in postpartum women with normal thyroid function. [2,3]

Some studies evaluated the rates of chronic hypothyroidism following the first year postpartum and found a range of 4% to 54%. The majority of studies suggested that 20–40% of women develop chronic hypothyroidism in the 3–12 year range following the onset of postpartum hypothyroidism. Risk factors for progression to chronic hypothyroidism included higher initial levels of thyroid antibodies as well as TSH in the early stages of hypothyroidism. These women were older, multiparous, and had more moderate to severe ultrasound hypoechogenicity. [7,19]

Diagnosis:

When checking for thyroid issues in pregnant folks, using TSH levels as the first test is a smart move, since it's the best indicator of thyroid problems. Also, checking the free T4 levels in the blood can help tell the difference between obvious thyroid issues and the less noticeable ones. Overt hypothyroidism is when your TSH levels are high and your free T4 levels are low. In subclinical hypothyroidism, your TSH levels are up but your free T4 levels are still okay. Similarly, women with overt hyperthyroidism have low serum TSH concentrations with an elevated free T4 concentration, and women with subclinical hyperthyroidism have low serum TSH concentrations with a normal free T4 concentration. Getting a precise read on the free T4 levels in pregnant ladies is kinda tricky. When a pregnant person has high estrogen, it doubles up the amount of TBG in their blood. When figuring out what's going on with thyrotoxicosis, you've got to consider Graves' disease, and it can show up after having a baby too. Checking for those specific thyroid antibodies can help tell apart postpartum thyroid issues from Graves' disease. Usually, if you find THR antibodies, it's a sign of Graves' disease. [3,7]

Patients with postpartum thyroiditis usually have high levels of TPO and thyroglobulin antibodies, and sometimes even mildly positive THR antibodies too. After giving birth, the thyroid starts to release hormones, and the balance between T4 and T3 is usually more skewed than in Graves' disease. When you check someone with Graves' disease, you'd expect to see stuff like bulging eyes and a weird sound from their neck (that's the thyroid murmur), but those aren't there in postpartum thyroiditis. Usually, when they check for thyroid issues, they find that postpartum thyroiditis shows less activity and Graves' disease shows more widespread activity. This test isn't recommended for women who are pregnant or nursing. [3,19,20]

Elevated thyroid hypoechogenicity is a frequent observation in women experiencing thyroid hyperplasia. In a forward-looking study, women with positive thyroid antibodies who subsequently experienced thyroid hyperplasia exhibited a greater occurrence of thyroid hypoechogenicity compared to women with either positive or negative thyroid antibodies who did not develop thyroid hyperplasia. Hypoechogenicity frequently occurred before hormonal indications of thyroid hyperplasia. Sadly, the predictive accuracy of thyroid ultrasonography is restricted, as a considerable number of women with marked thyroid hypoechogenicity remain euthyroid during the postpartum phase. [3,10,19]

Even though testing every single new mom isn't the best use of money compared to just checking the ones who seem at risk or have symptoms, it's still a good idea and it helps a lot more. Women at risk for whom TSH testing for the diagnosis of postpartum thyroiditis (PPT)

should be considered include high-risk populations between 6 and 12 weeks postpartum, women with postpartum depression, milk production problems, and multiple symptoms suggestive of hyperthyroidism or hypothyroidism, particularly within 3-6 months postpartum. [11]

Treatment:

Most women with hyperthyroidism during the postpartum thyroiditis phase do not require treatment since hyperthyroidism is usually mild and rarely lasts more than a few months. In symptomatic women, short-term beta-blocker therapy is responsive, and the dose is adjusted according to the severity of symptoms. Administration of beta-blockers (e g. propranolol, 10 to 20 mg q8h) in a dose that controls symptoms is recommended for symptomatic patients in the thyrotoxic phase. Postpartum thyroiditis is a destructive process, and antithyroid drugs are indicated in most cases. The decision to treat women with hypothyroidism detected at the stage of postpartum thyroiditis is complex and requires the assessment of multiple parameters. Women with TSH >10 mU/L or symptomatic women with TSH >4 but <10 mU/L should be treated with levothyroxine. Asymptomatic women with TSH 4-10 mU/L planning to conceive in the near future also require treatment, as recent studies have shown increased miscarriage rates in subclinical hypothyroidism, and lower future IQ scores in children born to women with mild thyroid hormone abnormalities. The duration of levothyroxine treatment is controversial. Two possibilities are: attempting to discontinue treatment around one year postpartum after the appearance of PPT, or continuing treatment until delivery and initiating a trial for discontinuing levothyroxine one year after the birth of the last baby. However, taking into account the high incidence of PPT and the negative impact of even minor changes in thyroid equilibrium on the future pregnancy, we consider it appropriate to continue treatment until delivery. [3,4,8,12,17] The recommendation is to decrease the levothyroxine dose approximately 12 months postpartum in case the patient is not pregnant, trying to conceive, or breastfeeding, with periodic monitoring of thyroid function. In women who develop PPT, 25–30% of cases will progress to permanent hypothyroidism. Therefore, it is not all women who need L-T4 replacement doses for treatment. However, the key lies in identifying those who will develop chronic hypothyroidism. It is reported that permanent hypothyroidism is more common in women with pregnancy having high titers of antimicrosomal antibodies (AMAb), and in women who have suffered hypothyroidism alone, especially in its extreme form. The annual progression rate from PPT to permanent hypothyroidism over the five years is 3. 6%.

However, it has been observed recently that the highest risk of permanent hypothyroidism is in women with high TPOAb, in the hypothyroid phase of PPT, and a hypoechoic thyroid. In these women, the risk of permanent hypothyroidism was 32%. Therefore, it seems necessary to have a long-term follow-up of women with these thyroid disorders who develop PPT. Women who have had postpartum thyroiditis should have their thyroid stimulating hormone (TSH) checked annually to assess for persistent hypothyroidism. Currently, there is no information regarding how often these women need to be monitored. In the normal population, the suggested screening for hypothyroidism is 5 yearly. [12,13,14,16]

Maternal hyperthyroidism may impact breastfeeding. For women with problematic breastfeeding, guidelines suggest the assessment of thyroid function and the institution of levothyroxine therapy when subclinical or overt hypothyroidism is identified. While controlled or randomized studies of comparative outcomes following treatment are lacking, case series indicate improved lactation in treated hypothyroid women. Lactation is advised for breastfeeding women with thyroid disease receiving thyroid medications like levothyroxine, propylthiouracil, methimazole, and carbimazole. They pass on only minimal amounts of such drugs into breast milk. The use of these drugs in mothers during lactation is safe, and thyroid function need not be tested in breastfed infants. [14,15,17,18]

Conclusion:

Within the context of postpartum thyroiditis (PPT), early identification is critical for effective diagnosis and clinical management. Comprehensive evaluation utilizing clinical assessment, thyrotropic hormone (TSH), free thyroxine (FT4), free triiodothyronine (FT3) levels, thyroid autoantibodies (TPOAb, TgAb), and occasionally thyroid ultrasound helps differentiate other thyroid diseases and inform management. Tailored monitoring, even if most cases of PPT are benign, helps mitigate the chances of developing chronic hypothyroidism, thus preserving maternal health.

PPT is an autoimmune thyroid disease that arises within the year after giving birth and is often biphasic or even triphasic, with temporary hyperthyroidism, then hypothyroidism, and a subsequent return to a euthyroid state. PPT’s immunology is the immune system’s response to the immunosuppressive state of pregnancy, most pertinent to women with thyroid autoantibodies or those with a history of autoimmune conditions in themselves or their family. While PPT is often accompanied by subtle, product-specific symptoms, systemic symptoms due to the immune response, including lethargy, mood alterations, and concentration

difficulties, can profoundly diminish a woman's postpartum recovery and their overall postpartum health.

However, early diagnosis, especially in women at high risk (e.g., TPOAb positive or type 1 diabetes patients), can lead to better clinical outcomes and thus, corroborates the idea of screening for postpartum thyroiditis in certain groups. The recurrence in the subsequent pregnancies is high, therefore long term follow up and education of the patient is very crucial. Ongoing studies in immunopathological mechanisms and predictive biomarkers are resulting in further enhancements in diagnostics and treatments. Like other autoimmune endocrine disorders, a multidisciplinary approach and tailored management plans remain essential for enhancing maternal outcomes and preventing chronic thyroid disease.

Disclosure

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REFERENCES

1. Stagnaro-Green, A. (2002). Postpartum Thyroiditis. The Journal of Clinical Endocrinology & Metabolism, 87(9), 4042–4047
2. Groer, M., & Jevitt, C. (2014). Symptoms and Signs Associated with Postpartum Thyroiditis. Journal of Thyroid Research
3. Stagnaro-Green, A. (2004). Postpartum thyroiditis. Best Practice & Research Clinical Endocrinology & Metabolism, 18(2), 303–316
4. Roti, E., & degli Uberti, E. (2002). Post-partum thyroiditis – a clinical update. European Journal of Endocrinology, 146, 275–279
5. M. Kologlu, Fung, H., Darke, C., Richards, C. J., Hall, R., & McGreogr, A. M. (1990). Postpartum thyroid dysfunction and HLA status. European Journal of Clinical Investigation, 20(1), 56–60
6. Marqusee, E., A. Hill, J., & Mandel, S. J. (1997). Thyroiditis After Pregnancy Loss. The Journal of Clinical Endocrinology & Metabolism, 82(8), 2455–2457
7. Epp, R., Malcolm, J., Jolin-Dahel, K., Clermont, M., & Keely, E. (2021). Postpartum thyroiditis. BMJ, 372
8. Quintero, B. M., Yazbeck, C., & Sweeney, L. B. (2021). Thyroiditis: Evaluation and Treatment. *American Family Physician*, *104*(6), 609–617
9. Lee, S. Y., & Pearce, E. N. (2022). Assessment and treatment of thyroid disorders in pregnancy and the postpartum period. Nature Reviews Endocrinology, 18, 158–171
10. Adams, H., Jones, M., Othman, S., Lazarus, J., Richards, C., Parkes, A., Hall, R., & Phillips, D. (1992). The sonographic appearances in postpartum thyroiditis. Clinical Radiology, 45(5), 311–315
11. Allan, W., Haddow, J., Palomaki, G., Williams, J., Klein, R., Mitchell, M., Hermos, R., & Faix, J. (2000). Maternal thyroid deficiency and pregnancy complications: implications for population screening. Journal of Medical Screening, 7(3)
12. Peng, C. C., & Pearce, E. N. (2022). An update on thyroid disorders in the postpartum period. Journal of Endocrinological Investigation, 45, 1497–1506
13. Lazarus, J. H., Ammari, F., Richards, C. J., Harris, B., Oretti, R., & Parkes, A. B. (1997). Clinical aspects of recurrent postpartum thyroiditis. British Journal of General Practice, 47(418), 305–308

14. Premawardhana, L. D. K. E., Lazarus, J. H., Parkes, A. B., Ammari, F., John, R., Darke, C., & Adams, H. (2000). Postpartum Thyroiditis and Long-Term Thyroid Status: Prognostic Influence of Thyroid Peroxidase Antibodies and Ultrasound Echogenicity. The Journal of Clinical Endocrinology & Metabolism, 85(1), 71–75
15. Stagnaro-Green, A. (2012). Approach to the Patient with Postpartum Thyroiditis. The Journal of Clinical Endocrinology & Metabolism, 97(2), 334–342
16. Lazarus, J., Parkes, A., & Premawardhaana, L. (2002). Postpartum Thyroiditis. Taylor & Francis online, 35(3), 169–173
17. Samuels, M. H. (2012). Subacute, Silent, and Postpartum Thyroiditis. Medical Clinics, 96(2), 223–233
18. Moleti, M., Vita, R., Benvenga, S., Vermiglio, F., Di Mauro, M., & Alibrandi, A. (2020). Postpartum Thyroiditis in Women With Euthyroid and Hypothyroid Hashimoto's Thyroiditis Antedating Pregnancy. The Journal of Clinical Endocrinology & Metabolism, 105(7), 2421–2428
19. Di Bari, F., Granese, R., Le Donne, M., Vita, R., & Benvenga, S. (2017). Autoimmune Abnormalities of Postpartum Thyroid Diseases. Frontiers in Endocrinology, 8(166)
20. Sakaihara, M., Yamada, H., Kobashi, G., Fukushi, M., Fujimoto, S., Hirayama Kato, E., Ebina, Y., & Shimada, S. (2000). Postpartum thyroid dysfunction in women with normal thyroid function during pregnancy. Clinical Endocrinology, 53(4), 487–492