



## Postpartum thyroiditis , how to diagnose and when to treat it ?

Sara kasem abdelaal<sup>1</sup>, Ahmed Abdelrahem Ahmed Taha <sup>2</sup>,  
marwa zanaty elsayed<sup>1</sup>

<sup>1-</sup> Sohag University Hospital, Department of internal medicine, Sohag, Egypt

<sup>2-</sup> Aswan University Hospital, Department of Obstetrics and Gynecology, aswan, Egypt.

### Abstract:

A damaging autoimmune condition known as postpartum thyroiditis (PPT) affects women without even a background of thyroid illness before becoming pregnant and develops during the first 12 months of giving birth.

PPT is an autoimmune condition linked to thyroid peroxidase antibodies (TPO).

postpartum thyroiditis may occur after abortion. As a result of pregnancy's immunosuppressive state, the level of anti - TPO antibodies naturally declines during pregnancy. An 80% likelihood of PPT exists for women who continue to be anti - TPO antibody positive during the third trimester of pregnancy.

Temporary or persistent thyroid illness may result from PPT. The following three phases of PPT have been proposed:1)Temporary hyperthyroidism 2) Temporary hypothyroidism , and 3) Temporary hyperthyroidism followed by hypothyroidism and eventually recovery, which is a traditional type of PPT.

Endocrine Society clinical recommendations advise screening risky women for postpartum thyroiditis like those with a positive anti - TPO antibody test, a history of PPT, or type 1 DM. Serum TSH levels for risky women should be checked at 3 and 6 months after giving birth

**Keywords:** Postpartum thyroiditis, anti-thyroid peroxidase antibody, levothyroxine.

**DOI :1 0.21608/SMJ.2023.197357.1375**

### Introduction:

A damaging autoimmune condition known as postpartum thyroiditis (PPT) affects women without even a background of thyroid illness before becoming pregnant and develops during the first 12 months of giving birth.

Temporary or persistent thyroid illness may result from PPT. The following three phases of PPT have been proposed: 1) Temporary hyperthyroidism (thirty - two % of cases), 2) Temporary hypothyroidism (forty - three % of cases), and 3) Temporary hyperthyroidism

followed by hypothyroidism and eventually recovery, which is a traditional type of PPT (twenty - five % of cases).

PPT is an autoimmune condition linked to thyroid peroxidase antibodies (TPO).

When anti -TPO antibodies are positive early in pregnancy, pregnant women have a 30% to 52% chance of having PPT<sup>(1)</sup>

postpartum thyroiditis may occur after abortion.<sup>(2)</sup> As a result of the pregn-

ancy's immunosuppressive state, the level of anti - TPO antibodies naturally declines during pregnancy. An 80% likelihood of PPT exists for women who continue to be anti - TPO antibody positive during the third trimester of pregnancy.<sup>(3)</sup>

Endocrine Society clinical recommendations advise screening risky women for postpartum thyroiditis like those with a positive anti - TPO antibody test, a history of PPT, or type 1 DM. Serum TSH levels for risky women should be checked at 3 and 6 months after giving birth<sup>(1)</sup>

### **Etiology:-**

It is an autoimmune condition and is characterized by antibodies against thyroid peroxidase (TPO). destructive thyroiditis with lymphocytic infiltration is another name for postpartum thyroiditis. Postpartum thyroiditis has Hashimoto thyroiditis' histological characteristics, and both conditions are linked to the HLA-D and HLA-B haplotypes. The significance of hereditary risk factors is demonstrated by these studies.<sup>(4)</sup>

### **Epidemiology**

Postpartum thyroiditis (PPT) is prevalent in 8% of pregnancies and occurs between 1.1% and 16.7% of them. It has been observed that rates vary across the globe. The rates are impacted by some variables, including the length of postpartum follow-up and the iodine level of the populations under study. DM (type 1) and favorable family history are two factors that increase the incidence of PPT, which are reported to be present in 19.1% and 20.0% of high-risk individuals, respectively. For patients who have previously experienced PPT, the reported recurrent rate of PPT is 42.4%.<sup>(5)</sup>

### **Clinical presentation**

The majority of PPT patients have no symptoms. When in a thyrotoxic state, PPT patients exhibit only mild symptoms, such as irritability, palpitations, weariness, and heat sensitivity. while in the hypothyroid phase, most patients are symptomatic, such as constipation, dry skin, weariness, poor focus, sensitivity to cold, and paresthesia. According to a study, those with PPT and positive anti -TPO antibodies experienced higher symptoms than people with negative TPO antibodies.<sup>(6)</sup>

### **Diagnosis**

Thyroid function tests(level of TSH and free T4), and clinical manifestation are both required for the diagnosis of PPT. Patients with PPT share comparable biochemical findings with those with painless thyroiditis, including elevated or above-normal serum free-T4 and T3 levels and decreased serum TSH levels throughout the hyperthyroid phase, which may be subclinical or overt. Because of the extended inhibition of TSH that had happened in the hyperthyroid phase, patients who experience a hyperthyroid phase followed by a hypothyroid state may experience low serum T4 levels for days to weeks until serum TSH levels climb above the standard parameters. In 60 to 85% of PPT patients, blood levels of the anti-TPO antibody are elevated, and they are at their maximum during or shortly after hypothyroidism. C-reactive protein and/or erythrocyte sedimentation rate may have slightly increased in some of the patients .<sup>(7)</sup> The possibility of postpartum thyroid dysfunction, which can cause a variety of symptoms during the postpartum period, must be recognized by clinicians who treat pregnant women following delivery

### **Treatment**

According to a prospective study that looked at six hundred and five asymptomatic pregnant and postpartum women, 40% of hypothyroid cases and none of those with thyrotoxicosis required treatment. If medical intervention is needed, it can frequently be lowered over a year. Up to 20% of instances with PPT will require long-term care, according to several studies. The thyrotoxic state's transient nature informs management of it. As PPT is a degenerative form of thyroiditis that does not involve a rise in thyroid hormone synthesis, anti-thyroid therapies (methimazole and propylthiouracil) have not proven successful in treating this thyrotoxic state. Generally, symptoms are minimal. A low dose of propranolol may be beneficial in a small number of individuals with clinically severe symptoms. The serum TSH level should be tested after around four to eight weeks (or in case of development of new symptoms) after the thyrotoxic state of postpartum thyroiditis has subsided to screen for the hypothyroid state. Treatment should be started in people who experience severe symptoms, are lactating, or desire to become pregnant. If the patient has minor signs of hypothyroid postpartum thyroiditis and wants to become pregnant again, levothyroxine (LT4) treatment should be considered. Thyroid function must be assessed every four to eight weeks until the patient becomes euthyroid if treatment is postponed. Women must also receive advice on using contraception. It has not been determined how long the patient should keep taking LT4. According to the recommendations, women who are pregnant or plan to become pregnant should maintain an euthyroid state from twelve months post-partum, LT4 doses can be decreased to determine if the hypothyroid condition caused by PPT is permanent or temporary. Every 6 to 8 weeks, the serum TSH

level must be checked, and the dose should be gradually decreased.<sup>(6)</sup>

## References

1. Muller, A.F., H.A. Drexhage, and A.J.E.r. Berghout, *Postpartum thyroiditis and autoimmune thyroiditis in women of childbearing age: recent insights and consequences for antenatal and postnatal care.* 2001. **22**(5): p. 605-630.
2. De Groot, L., et al., *Management of thyroid dysfunction during pregnancy and postpartum: an Endocrine Society clinical practice guideline.* 2012. **97**(8): p. 2543-2565.
3. Prummel, M.F., et al., *Thyroid peroxidase autoantibodies in euthyroid subjects.* 2005. **19**(1): p. 1-15.
4. LiVolsi, V.A.J.A.j.o.c.p., *Postpartum thyroiditis: The pathology slowly unravels.* 1993. **100**(3): p. 193-195.
5. Nicholson, W.K., et al., *Prevalence of postpartum thyroid dysfunction: a quantitative review.* 2006. **16**(6): p. 573-582.
6. AlexanderErik, K., et al., *2017 Guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum.* 2017.
7. Lazarus, J., et al., *The clinical spectrum of postpartum thyroid disease.* 1996. **89**(6): p. 429-436.