Case Report

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Silent Thyroiditis Presented as Transient Hypothyroidism during Pregnancy: A Case Report

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ABSTRACT

Silent thyroiditis during pregnancy is an under-recognized clinical condition and a rare case, often manifesting as transient hypothyroidism. We report a 23-year-old woman at 8 weeks of gestation who presented with newly diagnosed hypothyroidism [TSH = 85 mIU/L (0.4–4.5), FT4 = 0.4 ng/dL (0.7–2.5), and anti-TPO = 78 IU/mL (up to 16)]. Thyroid function tests had been normal eight months prior. Levothyroxine (LT4) therapy was initiated, restoring euthyroidism. Treatment was discontinued postpartum, and the patient maintained normal thyroid function for over one year.
Our case is unique in that silent thyroiditis occurred unusually early in pregnancy and differed from the more common postpartum cases. Silent thyroiditis should be considered in pregnancy-related hypothyroidism, particularly when prior thyroid function was normal. Prompt diagnosis and treatment can prevent adverse maternal and fetal outcomes.

Introduction

Silent thyroiditis, often referred to as postpartum thyroiditis, is an autoimmune condition that can manifest in women within the first year after childbirth. Its prevalence ranges from 5% to 16.7%, with a notable incidence of 3.9% to 8% [1]. Pregnancy induces a state of immune tolerance, which can be disrupted postpartum, leading to thyroiditis [2]. Women with thyroid autoimmunity face increased risks of miscarriage and pregnancy complications [3]. Subclinical hypothyroidism related to postpartum thyroiditis may impair neuropsychological development in offspring [3]. While many cases are transient, a significant percentage can progress to permanent hypothyroidism, requiring long-term monitoring [1].

Hypothyroidism during pregnancy can lead to various adverse outcomes for both the mother and the fetus. Maternal hypothyroidism is associated with increased risks of preterm birth, hypertensive disorders, and postpartum complications. For the fetus, risks include low birth weight, respiratory distress, and developmental issues [4,5]. In iodine-deficient regions, autoimmune thyroiditis, such as Hashimoto's thyroiditis, is recognized as the most common cause

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of hypothyroidism during pregnancy. Other less common causes of hypothyroidism, which may only be identified during pregnancy, include ablative therapies of the thyroid gland, such as radioiodine treatment or thyroidectomies, drug-induced thyroid gland destruction, and various forms of thyroiditis [6, 7].

Silent thyroiditis, also known as immune-mediated destructive thyroiditis, typically progresses through three phases: transient thyrotoxicosis, transient hypothyroidism, and, finally, euthyroidism. This condition, commonly referred to as postpartum thyroiditis, generally affects women during the postpartum period, with a prevalence of approximately 5% [8]. While this condition is frequent in the postpartum period, silent thyroiditis occurring early in pregnancy is rare; thus, only a few cases have been reported in the literature to date in this context [9].

The literature highlights the importance of screening for thyroid dysfunction during early pregnancy to prevent adverse outcomes [10]. Considering that pregnancy is a state of increased thyroid hormone requirements, it is plausible that hypothyroidism could develop during pregnancy in patients with borderline low thyroid function. Here, we report a case of silent thyroiditis that presented as isolated hypothyroidism during early pregnancy.

Case presentation

A 23-year-old woman in the 8th week of pregnancy was referred to our center for further evaluation of abnormal thyroid function laboratory values. The medical history revealed slight fatigability over the last two months. There was no history of previous hyper- or hypothyroidism, thyroid surgery, recent viral infections, or neck pain. Additionally, the results of thyroid function tests conducted eight months prior to her current pregnancy were within normal limits, with a TSH of 2.3 mIU/L and an anti-TPO of 4 IU/mL.

The general physical examination was unremarkable, except for mild periorbital edema. Examination of the thyroid gland revealed a firm but slightly enlarged thyroid (weighing approximately 25 g) without tenderness or palpable nodules. Her laboratory results were significant, showing TSH = 85 mIU/L (reference: 0.4–4.5), FT4 = 0.4 ng/dL (reference: 0.7–2.5), and anti-TPO = 78 IU/mL (reference: up to 16). The diagnosis of hypothyroidism was confirmed through repeated measurements to exclude potential laboratory errors.

Considering high TSH in the presence of low T4, Levothyroxine (LT4) at a dosage of 115 µg/day was initiated. Euthyroidism was restored (TSH = 0.5 mIU/L, Total Thyroxine [TT4] = 12.3 µg/dL, and T3 resin uptake [T3RU] = 27.9%) after four weeks of LT4 treatment.

Follow-up laboratory data obtained eight weeks after initiating LT4 therapy revealed a TSH of 0.4 mIU/L, a TT4 of 11.9 µg/dL, and a T3RU of 29.8% (Figure 1). Long-term follow-up of the patient after one year with normal thyroid function strongly supports the diagnosis of silent thyroiditis (Table 1).

It should be noted that the patient missed further medical follow-ups during pregnancy and presented four weeks after delivery with a TSH of 0.02 mIU/L, TT4 of 9.1 µg/dL, and a T3RU of 33.9%, despite taking 800 µg/week of LT4. The LT4 dosage was reduced to 350 µg/week; however, TSH remained suppressed at 0.01 mIU/L after eight weeks.

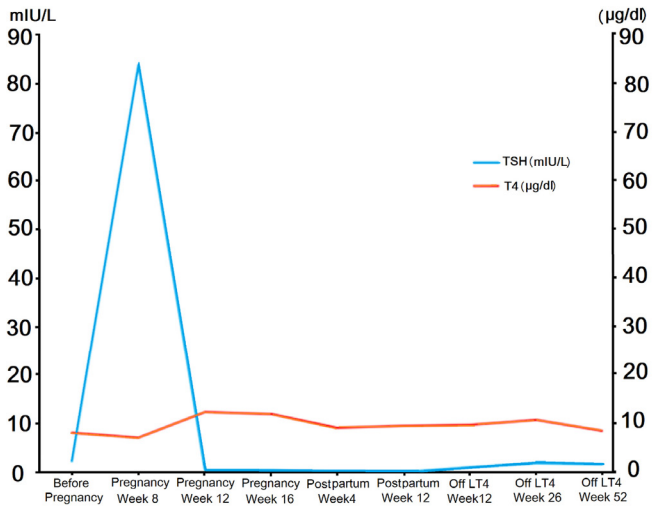

Fig. 1. The evolution of serum TSH and T4.

Table 1. Thyroid hormone changes during pregnancy and postpartum

Date	TSH (m IU/L)	TT4 (µg/dL)	Treatment
8th week of pregnancy	85	0.4 (FT4/ng/dL)	115µg/d LT4
12th week of pregnancy	0.5	12.3	115µg/d LT4
16th week of pregnancy	0.4	11.9	115µg/d LT4
4 weeks postpartum	0.02	9.1	decreased to 50 µg/d LT4
8 weeks postpartum	0.01	9.6	discontinued
12 weeks postpartum	1.05	9.8	-
26 weeks postpartum	1.85	10.8	-
52 weeks postpartum	1.59	8.5	-

LT4 was subsequently discontinued, and TSH levels at 12, 26, and 52 weeks were recorded as 1.05, 1.85, and 1.59, respectively (Figure 1). Transient hypothyroidism due to silent thyroiditis was diagnosed, and the patient was advised to undergo annual screening for persistent hypothyroidism. Furthermore, no complications were observed in either the mother or the child during pregnancy or up to approximately one year postpartum.

Written informed consent was obtained from the patient after she was provided with the necessary information regarding the aims of this report.

Discussion

Our case highlights a rare form of silent thyroiditis that manifests as transient hypothyroidism at the beginning of pregnancy, differing from the more frequently reported postpartum forms. Comparative literature reviews reveal that silent thyroiditis during pregnancy is underrepresented, potentially due to its frequent misdiagnosis as gestational hypothyroidism or early Hashimoto's thyroiditis. Kamijo et al. reported transient subclinical hypothyroidism in 0.19% of pregnant women with no proven etiology, underscoring the importance of distinguishing silent thyroiditis from other thyroid dysfunctions during pregnancy [11].

Additionally, Shields et al. documented recovery of thyroid function after delivery in 75.4% of cases of subclinical hypothyroidism, particularly in anti-TPO-negative individuals [12]. In contrast, our patient exhibited persistent anti-TPO positivity and a severe hypothyroid phase, suggesting autoimmune involvement despite no prior history of thyroid dysfunction. This aligns with the findings of Stagnaro-Green et al., who reported a high rate of persistent hypothyroidism in cases of postpartum thyroiditis in southern Italy [13].

The transient nature of our case, confirmed by the resolution of thyroid dysfunction postpartum, supports the diagnosis. Silent thyroiditis typically follows a triphasic course but can present solely as thyrotoxicosis or hypothyroidism in up to 50% of cases. Transient hypothyroidism is frequently observed after the thyrotoxic phase of destructive thyroiditis. Less commonly, it may occur following the radioiodine treatment of Graves' disease or the early phase of Hashimoto's thyroiditis [14].

In this case, it was considered that, given completely normal thyroid function a few months before pregnancy, hypothyroidism—though severe—was most likely transient. This was confirmed by the return of normal thyroid function up to about one year after discontinuation of LT4 therapy. Typically, silent thyroiditis progresses through three phases: thyrotoxicosis, followed by hypothyroidism, and eventually euthyroidism. However, in up to half of patients, it may only present as isolated thyrotoxicosis or hypothyroidism. Moreover, permanent hypothyroidism may gradually develop in such patients several years later [13].

To the best of our knowledge, there is one reported case of silent thyroiditis during pregnancy that presented with thyrotoxicosis in the 4th week of gestational age and subsequently developed hypothyroidism four weeks later [9]. However, our case was first diagnosed with hypothyroidism during early pregnancy, suggesting a previously unnoticed thyrotoxic phase that occurred just before or in the initial days of pregnancy. It is noteworthy that the thyrotoxic phase of silent thyroiditis can mimic gestational thyrotoxicosis as well as other etiologies of thyrotoxicosis, particularly Graves' disease. This was exemplified in the reported case of silent thyroiditis during pregnancy by Sato et al., where the patient was initially managed for Graves' hyperthyroidism [9].

Silent thyroiditis, particularly in the context of pregnancy, is believed to arise from autoimmune mechanisms characterized by transient inflammation of the thyroid gland. Pregnancy-induced immunomodulation may initially suppress

autoimmune responses [15]; however, after childbirth, the immune response often triggers thyroid dysfunction. Early in the course of the disease, damage to the follicular cells causes the release of preformed thyroid hormones, resulting in transient thyrotoxicosis [15]. This phase is followed by hypothyroidism due to the depletion of thyroid hormone stores. Dysregulated immune responses, including T helper 1 (Th1) cell involvement, cytokine activation, and reduced regulatory T cell function, are central to the pathophysiology.

The disease may resolve spontaneously or progress to chronic hypothyroidism, underscoring the importance of monitoring and managing thyroid function in perinatal care [16, 17]. Genetic predisposition plays a significant role, particularly in women with a history of autoimmune diseases. Environmental factors, such as iodine intake and stress, may also influence the onset of silent thyroiditis [18].

The current guidelines of the American Thyroid Association recommend LT4 treatment when TSH levels exceed pregnancy-specific reference ranges (or TSH > 4.0 mIU/L) in patients with elevated TPO antibodies and advocate treatment when TSH is > 10 mIU/L in all patients. Treatment may also be considered for TSH levels in the range of 4–10 mIU/L in patients who are TPO Ab-negative. Gestational hypothyroidism is no longer defined by TSH > 2.5 mIU/L [15].

However, the case presented in this study exhibited minimal symptoms, including overt hypothyroidism associated with high anti-TPO antibody levels, which mimic typical Hashimoto's disease. Nonetheless, normal thyroid function, including anti-TPO antibody levels, a few months prior to pregnancy supports the diagnosis of the hypothyroid phase of silent thyroiditis. Treatment with LT4 may not significantly differ between the two conditions; however, in this case, a lower dose of LT4 was prescribed due to the substantial presence of viable follicular cells in silent thyroiditis.

Conclusion

Transient hypothyroidism can complicate pregnancy, necessitating early recognition and management to optimize maternal and fetal outcomes. Silent thyroiditis, although rare, should be considered in cases of hypothyroidism with prior normal thyroid function. Long-term follow-up is crucial for monitoring progression to permanent hypothyroidism. Regular thyroid function monitoring during pregnancy is essential to ensure optimal maternal and fetal health.

Thyroid disorders, particularly hypothyroidism, can significantly affect both the mother and child if left untreated. Given the prevalence of thyroid disorders among pregnant women, implementing strict monitoring protocols is imperative. By adhering to established thyroid guidelines, healthcare providers can detect and address thyroid dysfunction early, preventing potential complications such as premature birth, low birth weight, and fetal developmental delays.

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Ethical Considerations

Authors' contributions

M.A.K designed and directed the study. S.N. collected the data and drafted the manuscript. All authors discussed the results and contributed to the final manuscript.

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Conflict of Interests

The authors declare that they have no conflict of interest.

Ethical Statement

The local Ethics Committee affiliated with the Zahedan University of Medical Sciences has approved this study (Registration no.: IR.ZAUMS.REC.1402.406). A written consent form for publication was signed.

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