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Case Report

From hypothyroidism to Graves' disease during pregnancy: a case of thyrotoxicosis following long-term hypothyroidism

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ABSTRACT

Graves' disease is an autoimmune thyroid disorder characterized by hyperthyroidism, diffuse goiter, and orbitopathy, and it rarely occurs during pregnancy. A 23-year-old woman presented to the emergency department at 20 weeks of her second pregnancy with complaints of palpitations, sweating, and fatigue. She had previously been diagnosed with hypothyroidism by her family physician and had been on levothyroxine therapy. She had also been followed with hypothyroidism during her first pregnancy. Evaluation revealed findings consistent with thyrotoxicosis and positive thyroid autoantibodies, leading to a diagnosis of Graves' disease. Propylthiouracil (PTU) therapy was initiated, and thyroid function was closely monitored throughout pregnancy. Both maternal and fetal outcomes were uneventful, and delivery occurred without complications. The development of Graves' disease during pregnancy in a patient previously diagnosed with hypothyroidism is exceedingly rare. This case highlights the clinical importance of the transition from hypothyroidism to Graves' hyperthyroidism during pregnancy.

Keywords: Graves' disease, pregnancy, hyperthyroidism

INTRODUCTION

Graves' disease is the most common cause of hyperthyroidism in iodine-sufficient regions of developed countries.[1] It results from excessive stimulation of thyroid-stimulating hormone (TSH) receptors by TSH receptor antibodies (TRAb), leading to thyroid hypertrophy, increased hormone synthesis, and consequently thyrotoxicosis, diffuse goiter, and sometimes ophthalmopathy.[2]

Pregnancy represents a physiological state in which the Th1/Th2 balance of the immune system is altered, potentially influencing the course of autoimmune thyroid disorders. Suppression of the autoimmune response during pregnancy and its reactivation in the postpartum period may contribute to the onset of Graves' disease.[3]

The transition from hypothyroidism to hyperthyroidism is extremely uncommon, even among patients with autoimmune thyroid disorders. In the largest 10-year follow-up study of patients with TBAb-induced hypothyroidism, Takasu and Matsushita[4] reported that only 5.9% (2 of 34) of patients developed thyroid-stimulating antibodies (TSAb) and became hyperthyroid, highlighting the rarity of such autoimmune switching.

The mainstay of Graves' disease management involves controlling symptoms and normalizing thyroid hormone levels. Although treatment during pregnancy is generally similar to that in non-pregnant individuals, certain adjustments are required. The goal is to maintain mild maternal hyperthyroidism to prevent fetal hypothyroidism, as the fetal thyroid is more sensitive to antithyroid drugs.[5] Overtreatment with thionamides may lead to fetal goiter and primary hypothyroidism, whereas undertreatment increases the risk of transient central hypothyroidism.[6]

This case represents a rare instance of Graves' hyperthyroidism developing during pregnancy in a patient previously diagnosed with hypothyroidism, emphasizing the importance of vigilant thyroid monitoring in pregnant women.

CASE

A 23-year-old woman presented to the emergency department at 20 weeks' gestation of her second pregnancy with complaints of severe palpitations. Six years earlier, during a routine evaluation at a family medicine clinic, she had been diagnosed with primary hypothyroidism based on an isolated elevation of thyroid-stimulating hormone (TSH) with normal free thyroxine (FT4) levels; thyroid autoantibodies [anti-thyroid peroxidase (anti-TPO), anti-thyroglobulin (anti-Tg), and thyroid-stimulating hormone receptor antibody (TRAb)] were not assessed at that time. Levothyroxine therapy was initiated, and she achieved a clinically euthyroid state on follow-up.

Her obstetric history included two spontaneous pregnancies conceived without assisted reproductive techniques. During her first pregnancy, thyroid autoantibodies (anti-TPO, anti-Tg, TRAb) were assessed for the first time and found to be negative. She remained euthyroid and clinically stable on

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levothyroxine throughout that pregnancy. The early course of her second pregnancy was uneventful, with normal fetal growth and routine obstetric monitoring.

At 21 weeks' gestation, however, she presented to the emergency department with severe palpitations, excessive sweating, and fatigue, though. Initial laboratory evaluation demonstrated suppressed TSH and markedly elevated FT4 and free T3 levels. Endocrinology consultation confirmed thyrotoxicosis secondary to Graves' disease. Thyroid autoantibody testing performed at presentation was positive, including elevated TRAb levels, indicating autoimmune stimulation. Considering the maternal and fetal risks associated with uncontrolled thyrotoxicosis during pregnancy, treatment with propylthiouracil (PTU) was initiated. The patient responded favorably, with gradual clinical improvement and normalization of thyroid hormone levels.

Thyroid ultrasonography revealed diffuse gland enlargement with increased vascularity, while ophthalmologic examination showed no evidence of Graves' orbitopathy. The remainder of the pregnancy was uneventful, and the patient delivered a healthy infant at term. Postpartum endocrinology follow-up demonstrated stable thyroid function.

DISCUSSION

This case demonstrates a rare occurrence of Graves' disease emerging during pregnancy in a patient with a prior diagnosis of hypothyroidism. Transition from hypothyroidism to Graves' hyperthyroidism has been described only in a limited number of reports, and to our knowledge, there are very few cases documented during pregnancy.[7,8] The underlying pathophysiology remains unclear, but it may involve immune modulation due to lymphocytic infiltration, fibrosis, and follicular damage of the thyroid gland. Although elevated anti-TPO antibody levels generally indicate progressive irreversible hypothyroidism, in some cases, follicular regeneration followed by the dominance of stimulating autoantibodies may result in hyperthyroidism.[7]

McDermott et al.[9] reported two female patients who, similar to our case, developed thyrotoxicosis after an initial diagnosis of primary hypothyroidism. Up to 2014, only 37 such cases of hypothyroidism converting to hyperthyroidism had been reported.[10] Autoimmune thyroid diseases are more common in women,[2] and the predominance of female patients in published cases supports the hypothesis of gender-related differences in immune response.[7,8,10]

Although the precise cause of hyperthyroidism following hypothyroidism is uncertain, it has been proposed that the shift between thyroid-stimulating and thyroid-blocking antibodies plays a central role.[11] During pregnancy, immune modulation characterized by a shift from Th1 to Th2 dominance leads to suppression of cell-mediated immunity and enhanced humoral activity. This shift may reduce autoimmune attacks during pregnancy but rebound postpartum, possibly triggering the transition from hypothyroidism to hyperthyroidism.[2] Physiological immune tolerance changes during pregnancy may enhance TRAb production, leading to abrupt thyrotoxicosis or Graves' disease. In support of this mechanism, Aranyosi et al.[12] reported a case diagnosed with Graves' disease at 20 weeks of gestation, complicated by orbitopathy.

Selection and dosage of antithyroid drugs during pregnancy are crucial for maternal and fetal safety. Methimazole has teratogenic potential; therefore, PTU is preferred during the first trimester.[13,14] In our patient, thyrotoxicosis detected in the second trimester was successfully controlled with PTU therapy.

The sudden development of Graves' hyperthyroidism during pregnancy in a patient with a history of hypothyroidism is a rare phenomenon. This transition may be influenced by shifts in the balance between thyroid-stimulating and blocking antibodies, as well as pregnancy-related alterations in immune response. This case emphasizes that clinicians should monitor not only TSH levels but also the patient's antibody profile and clinical symptoms. Regular assessment of thyroid function and autoantibodies during pregnancy facilitates the early detection of potential autoimmune transitions, thereby contributing to the protection of both maternal and fetal health.

CONCLUSION

This case illustrates that Graves' disease may develop during pregnancy in women with a prior history of hypothyroidism. Early diagnosis and appropriate antithyroid therapy are crucial for safeguarding maternal and fetal well-being. Monitoring thyroid function tests alongside autoimmune markers throughout pregnancy allows for the timely identification of potential autoimmune shifts.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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