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CASE PRESENTATION

Hashimoto's thyroiditis and obstetric complications: report of a complex clinical case

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ABSTRACT

Introduction: hashimoto's hypothyroidism is an autoimmune disease that represents the most common cause of hypothyroidism in regions with sufficient iodine, and its prevalence is significant among women of reproductive age.
Objective: to describe the therapeutic management of a patient with Hashimoto's thyroiditis during pregnancy.
Case Report: a 36-year-old woman, with a clinical history of Hashimoto's hypothyroidism under regular treatment, presented with elevated antibody titers at 14 weeks of gestation and sought medical consultation due to decompensation. A difficult-to-control hypothyroidism was identified, secondary to active Hashimoto's thyroiditis and insufficient therapeutic adherence during the preconception stage. This combination favored an unfavorable endocrine and immunological environment, resulting in hypertensive disorders of pregnancy, as well as preterm delivery and intrauterine growth restriction.
Conclusions: hashimoto's thyroiditis is a common cause of hypothyroidism during pregnancy, and its proper management is crucial to avoid both maternal and fetal complications. Monitoring thyroid function and antibody levels is essential to adjust treatment and prevent adverse outcomes. Unfortunately, this did not occur in our case, optimal TSH levels were never achieved, and adverse results were experienced.

Keywords: Hashimoto's Thyroiditis; Hypothyroidism; Preeclampsia; Preterm Delivery; Intrauterine Growth Restriction.

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INTRODUCTION

Hashimoto's thyroiditis during gestation constitutes a prevalent form of autoimmune hypothyroidism, characterized by the presence of antithyroid autoantibodies, especially those directed against thyroid peroxidase (TPOAb). These antibodies can alter thyroid function by inducing inflammation and progressive destruction of glandular tissue. In contexts with adequate iodine intake, this entity represents a frequent cause of clinical and subclinical hypothyroidism.[1,2,3]

Clinical hypothyroidism is defined by elevated concentrations of thyroid-stimulating hormone (TSH) together with decreased levels of free thyroxine (T4), whereas subclinical hypothyroidism is characterized by elevated TSH with T4 within the normal range. During pregnancy, reference values for TSH vary according to trimester, with the recommended upper limit being 2.5 mU/L in the first trimester and 3.0 mU/L in subsequent trimesters, according to the guidelines of the American Thyroid Association (ATA).[4,5,6]

Hashimoto's thyroiditis is one of the main causes of thyroid dysfunction in pregnant women, with a significantly higher prevalence in women of reproductive age. During gestation, up to 18 % of women may present positivity for anti-TPO antibodies, which is associated with alterations in thyroid function and an increased risk of adverse outcomes such as spontaneous abortion and preterm birth.[7,8,9,10]

Various risk factors modulate the clinical expression of Hashimoto's thyroiditis during pregnancy. The presence of TPOAb and TGAb has been linked to obstetric complications such as gestational hypertension, gestational diabetes mellitus, intrauterine growth restriction, and the need for neonatal intensive care. Furthermore, TPOAb positivity may interfere with the thyroid response to the physiological stimulus of human chorionic gonadotropin (hCG), favoring the development of preterm birth.[11,12,13]

Other factors that may contribute to thyroid dysfunction in pregnancy include advanced maternal age, elevated body mass index, multiple gestation, and the use of assisted reproductive techniques such as in vitro fertilization. These elements have been associated with a higher prevalence of thyroid alterations and with adverse perinatal outcomes.[5] This raised the question of highlighting the importance of interdisciplinary management, for which the present investigation was developed, which aimed to describe the therapeutic management of a patient with Hashimoto's thyroiditis during pregnancy.

CASE PRESENTATION

A 36-year-old female patient, Ecuadorian, mestiza, married, nurse by profession. She presents a personal history of Hashimoto's hypothyroidism diagnosed 8 years ago with irregular treatment with levothyroxine before pregnancy (previous dose 125 µg/day); preeclampsia in the previous pregnancy (4 years ago), preterm birth at 32 weeks, neonatal death due to neonatal complications; pregestational class I obesity (BMI 30 kg/m²); mild chronic anemia, arterial hypertension controlled with methyldopa. Among family history, it is noted that her mother had type 2 diabetes mellitus and hypothyroidism, her father had arterial hypertension, and a sister had Hashimoto's thyroiditis.

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The patient in question, at 14 weeks of gestation, consulted for severe fatigue since the beginning of pregnancy, somnolence, dyspnea, edema at the level of lower limbs, cold intolerance, constipation, anxiety with depressive episodes, occasional palpitations, constipation, dry skin, and decreased concentration.

On physical examination, she was alert, anxious, with myxedematous facies, anthropometry (weight 78 kg, height 158 cm, BMI 31,3 kg/m²), blood pressure 140/90 mmHg, heart rate 58/minute, dry skin, cold to touch; thin, brittle hair; diffuse thyroid enlargement with grade II goiter, without nodules, firm consistency, no bruits; edema of lower limbs and slightly decreased osteotendinous reflexes; and fetus with heart rate of 160 beats on average.

Laboratory tests at week 6 (Table 1) report values: TSH: 7,5 µU/mL; free T4: 0,3 ng/dL; anti-TPO antibodies > 600 IU/mL; hemoglobin: 10,8 g/dL with low mean corpuscular volume and decreased mean corpuscular hemoglobin; hypoferritinemia, lipid profile with total cholesterol 300 mg/dL; uric acid 5 mg/dL; normal hepatic and renal tests. Imaging studies report serial obstetric ultrasounds with fetal growth at the 10th percentile at week 20, and Doppler of uterine arteries with flow redistribution. Thyroid ultrasound shows a diffusely hypoechoic gland, without nodules and with heterogeneous consistency compatible with chronic thyroiditis.

Table 1. Laboratory Results.

Complementary Test	Results	Reference Ranges
TSH (mIU/L)	7,5	0,27 – 0,42
Free T4 (ng/dL)	0,3	1,2 – 2,2
Anti-TPO antibodies (IU/mL)	> 600	5 – 34
Hemoglobin (g/dL)	108	12.0 – 14.0
Ferritin (ng/dL)	20	10 – 120
Total cholesterol (mg/dL)	300	Adequate: < 200 mg/dL Borderline high: 200-239 mg/dL High: ≥ 240 mg/dL
LDL cholesterol	170	Optimal: < 100 mg/dL Near optimal: 100-129 mg/dL Borderline high: 130-159 mg/dL High: 160-189 mg/dL Very high: ≥ 190 mg/dL
HDL cholesterol (mmol/L)	42	Protective: ≥ 60 mg/dL Normal: 40-59 mg/dL Risk factor: <40 mg/dL

Given this situation, difficult-to-control Hashimoto's hypothyroidism was considered due to high active autoimmunity (elevated anti-TPO) and the risk of hypertensive disorders of pregnancy and fetal problems such as intrauterine growth restriction. Given this clinical picture, her therapeutic management was progressively readjusted; the levothyroxine dose was increased to 200 µg/day. Methyldopa was maintained as an antihypertensive. Nutritional supplements were added: folic acid (5 mg/day) and iron (60 mg/day). At 20 weeks, acetylsalicylic acid was initiated due to preeclampsia risk. Table 2 shows the chronology of the clinical evolution presented by the patient.

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Table 2. Clinical Evolution and Summary of Complications.

Gestational Age	Events
Week 6	First prenatal visit. Fatigue, mild palpitations. Elevated TSH (7.5 µU/mL).
Week 8	Initial adjustment of levothyroxine (from 125 to 175 mcg/day). Difficult control.
Week 12	Persistence of symptoms, TSH: 6.9 µU/mL. Referral to endocrinology.
Week 14	Normal ultrasound.
Week 20	TSH: 5.8 µU/mL. Fetus with restricted growth (10th percentile). Initiation of acetylsalicylic acid. Dose increase to 200 mcg/day.
Week 28	Reduction of fetal movements. Doppler: altered cerebroplacental index. Hospitalization for surveillance.
Week 30	Worsening of hypertension (160/100 mmHg). TSH: 4.5 µU/mL. Induction of fetal lung maturation. Confirmation of chronic hypertension with superimposed preeclampsia.
Week 32	Emergency cesarean section due to acute fetal distress. Preterm newborn at 32 weeks (1500 g), Apgar 7/8. Admission to Neonatal Intensive Care Unit.

The patient presented difficult-to-control hypothyroidism, secondary to active Hashimoto's thyroiditis and insufficient therapeutic adherence in the preconception stage. This combination favored an unfavorable endocrine and immunological environment, which resulted in hypertensive disorders of pregnancy, specifically chronic hypertension with superimposed preeclampsia, in addition to preterm birth and intrauterine growth restriction (IUGR). These adverse outcomes reinforce the need for strict endocrinological follow-up and therapeutic optimization prior to conception, especially in women with autoimmune thyroid diseases.

DISCUSSION

Various recent studies have demonstrated that elevated levels of anti-TPO antibodies constitute a factor of poor obstetric prognosis, being associated with a higher risk of preeclampsia, spontaneous abortion, preterm birth, and alterations in fetal development. The persistence of these antibodies reflects active autoimmunity, which may interfere with placental function and hormonal regulation during gestation. Therefore, their detection and monitoring should be part of the comprehensive evaluation of women with Hashimoto's thyroiditis of childbearing age.[7,14]

Furthermore, it has been documented that maintaining TSH below 2,5 mIU/L during pregnancy, together with early adjustments of levothyroxine, significantly improves perinatal outcomes in patients with Hashimoto's. This approach requires quarterly monitoring and effective health education that promotes therapeutic adherence and understanding of risk.[15]

The importance of controlling Hashimoto's hypothyroidism before pregnancy lies in the prevention of multiple maternal-fetal complications that may arise due to poor control of thyroid function. Euthyroxinemia prior to conception is fundamental to guarantee an optimal hormonal environment for embryonic and fetal development, especially during the first trimester, where the fetus depends exclusively on maternal thyroid hormones.[16]

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Poor control of hypothyroidism in women with Hashimoto's thyroiditis is associated with a higher risk of spontaneous abortions, intrauterine growth restriction (IUGR), preterm birth, preeclampsia, and premature placental abruption, as reported in our case. Furthermore, untreated or poorly controlled maternal hypothyroidism may negatively impact fetal neurological development, increasing the risk of cognitive deficits and delayed psychomotor development in childhood.[1,17,18]

Therefore, screening and adjustment of the levothyroxine dose before pregnancy is essential to achieve TSH values in the recommended range (< 2,5 µU/mL in women seeking to conceive). This not only improves fertility but also allows pregnancy to begin with adequate metabolic control, reducing the need for abrupt medication adjustments during pregnancy and ensuring a better prognosis for both mother and fetus.[19,20]

For all the aforementioned reasons, we summarize some updated data on the clinical management of Hashimoto's hypothyroidism. The treatment of Hashimoto's disease during pregnancy, delivery, and the postpartum period focuses on the use of levothyroxine (LT4) to correct hypothyroidism and maintain euthyroidism. Levothyroxine is the recommended treatment for maternal hypothyroidism, as it ensures an adequate supply of T4 to the fetus, crucial for fetal brain development.[21,22]

During pregnancy, it is common for women with preexisting hypothyroidism to need an increase in the levothyroxine dose to maintain adequate thyroid hormone levels. Close monitoring of TSH levels is recommended to avoid overtreatment or undertreatment.[4] The goal is to maintain the TSH level below 2,5-3,0 mIU/L, depending on the trimester of gestation.[23]

Regarding the treatment of subclinical hypothyroidism, although controversy exists, it is considered reasonable to treat with levothyroxine, especially if the TSH level is higher than 10 mIU/L or if there is presence of antithyroid antibodies. Evidence suggests that treatment may reduce the risk of preterm birth and spontaneous abortion.[24,25]

Treatment with levothyroxine (LT4) is fundamental to achieve these TSH levels. The need to adjust the LT4 dose during pregnancy is common, as thyroid hormone requirements increase significantly due to the physiological changes of pregnancy.[26] Regular monitoring of thyroid function, every 4 to 6 weeks, is essential to ensure that TSH levels remain within the optimal range.[22]

There is robust scientific evidence demonstrating that TSH levels above 2,5 mIU/L are associated with a higher risk of spontaneous abortion and other obstetric complications. Therefore, it is important that women with Hashimoto's hypothyroidism are adequately treated with LT4 to maintain euthyroidism and protect both maternal health and fetal development.[27,28]

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CONCLUSIONS

Hashimoto's thyroiditis represents a frequent cause of hypothyroidism during pregnancy, whose adequate management is essential to prevent maternal and fetal complications such as spontaneous abortion, preterm birth, preeclampsia, intrauterine growth restriction, and neurological alterations in the newborn. Constant monitoring of thyroid function and antibody levels, together with individualized adjustments of levothyroxine, allows optimization of TSH levels and improvement of perinatal outcomes; however, as evidenced in the clinical case presented, lack of adequate control may lead to adverse results. Therefore, the importance of multidisciplinary care and patient education is highlighted to guarantee therapeutic adherence and a healthy gestation.

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