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Case Report

Thyrotoxic Crisis in a Pregnant Woman at 16 Weeks of Gestation in the Gyneco-Obstetric Intensive Care Unit: A Case Report

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Abstract: Thyrotoxic crisis during pregnancy is a rare but life-threatening condition, most commonly associated with Graves' disease. This case report details a 25-year-old primigravida at 16 weeks of gestation who presented with severe thyrotoxicosis. Symptoms included tachycardia, hyperthermia, and fatigue. Laboratory findings confirmed suppressed TSH and elevated free T4 levels. Immediate treatment with beta-blockers and antithyroid drugs stabilized the patient. Pregnancy alters thyroid physiology, increasing the risk of hyperthyroidism complications such as miscarriage, preterm birth, and preeclampsia. Treatment involves antithyroid medications, with PTU preferred in the first trimester due to MMI's teratogenic risks. Close monitoring of thyroid hormone levels and fetal well-being is critical. Despite the severity of the initial presentation, the patient's pregnancy progressed without complications, highlighting the importance of early and aggressive management.

Keywords: Thyrotoxic crisis, Pregnancy, Hyperthyroidism, Graves' disease, Thyroid storm, Beta-blockers, Antithyroid drugs (ATDs), PTU (propylthiouracil), Maternal-fetal health, Neonatal hyperthyroidism, Thyroid hormone monitoring.

1. INTRODUCTION

Thyrotoxic crisis during pregnancy is a rare but potentially fatal medical emergency associated with hyperthyroid function. It occurs mainly in the context of Graves' disease, affecting approximately 0.1% to 0.4% of pregnancies [1]. This condition can lead to severe complications, including miscarriages, preterm births, and preeclampsia. Rigorous management of this pathology is crucial to protect maternal and fetal health. Close clinical and biological monitoring is necessary to prevent the need for thyroidectomy or worsening of symptoms [2].

2. CLINICAL OBSERVATION

We report the case of a 25-year-old primigravida patient with no significant medical history, hospitalized in an emergency setting at 16 weeks of gestation for a thyrotoxic crisis during an ongoing pregnancy. The patient presented with intense fatigue, palpitations, and recent weight loss. Additional symptoms included episodes of insomnia, agitation, and tremors.

Clinical examination revealed a heart rate of 135 beats per minute, blood pressure at 140/85 mmHg, hyperthermia at 38°C, and excessive sweating. Cardiac auscultation was unremarkable except for the tachycardia, and thyroid examination did not show a notable goiter. Respiratory findings were normal, with the patient being eupneic and showing oxygen saturation of 98% on room air. No abdominal or lower limb abnormalities were detected, and bowel sounds were normal.

Emergency laboratory tests showed a suppressed TSH level (<0.01 mIU/L), significantly elevated free thyroid hormones (free T4 at 36 pmol/L), moderate hyperglycemia, and mild hyponatremia at 133 mmol/L. Inflammatory markers were positive, with CRP at 9.2 mg/L, indicating an inflammatory response. Abdominal ultrasound confirmed the absence of obstetric complications and a normal pregnancy progression.

The patient was immediately placed on beta-blockers and synthetic antithyroid medications, with close hemodynamic monitoring.

3. DISCUSSION

Thyrotoxicosis during pregnancy is a rare but potentially severe complication with significant implications for both mother and fetus. The reported case

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involves a 25-year-old patient at 16 weeks of gestation who developed a thyrotoxic crisis. This condition represents a clinical challenge, as pregnancy alters thyroid physiology and may exacerbate underlying hyperthyroidism or trigger new decompensation.

Uncontrolled thyrotoxicosis during pregnancy is associated with increased risks of miscarriage, intrauterine growth restriction, preterm birth, preeclampsia, and gestational hypertension [3]. Additionally, pregnant women with thyrotoxicosis face a heightened risk of congestive heart failure and thyroid storm, both of which are potentially life-threatening for the mother [4].

In this case, the patient presented with marked tachycardia (135 bpm) and hyperthermia, which are typical signs of thyrotoxic crisis. Prompt recognition of these signs is crucial, as delays in management can lead to severe consequences for both mother and fetus [2, 5].

Pregnancy induces significant changes in thyroid physiology. Plasma volume and thyroxine-binding globulin (TBG) concentrations increase substantially, resulting in elevated total T3 and T4 levels [5]. In patients with pre-existing hyperthyroidism, these changes may worsen the condition, necessitating close monitoring. Furthermore, TSH levels are often suppressed due to the effects of human chorionic gonadotropin (hCG), complicating the diagnosis of thyrotoxicosis during pregnancy [6].

In this case, laboratory results showed suppressed TSH and significantly elevated free T4 levels, confirming severe thyrotoxicosis. This aligns with the literature, which emphasizes the need for close monitoring of pregnant women with hyperthyroidism, particularly during the first trimester when risks are highest [7].

The treatment of thyrotoxicosis during pregnancy primarily involves synthetic antithyroid drugs (ATDs), such as propylthiouracil (PTU) or methimazole (MMI). However, it is essential to limit doses to avoid iatrogenic hypothyroidism, which can negatively impact the fetus [8]. In this case, the patient was treated with ATDs under strict control of free T4 levels. Beta-blockers, such as propranolol, are also recommended to manage cardiovascular symptoms associated with hyperthyroidism [9].

Studies show that PTU is preferred over MMI during the first trimester due to MMI's teratogenic risk. However, after the first trimester, MMI may be reintroduced due to the hepatic risk associated with PTU [8]. The patient received appropriate treatment with regular monitoring of thyroid hormones and inflammatory markers.

In addition to pharmacological management, regular monitoring is essential to prevent complications related to thyrotoxicosis during pregnancy. Persistent tachycardia observed in our patient is a sign of adrenergic overstimulation, common in this context. This condition can quickly progress to heart failure or thyroid storm if not adequately managed. Recent studies highlight the importance of closely monitoring cardiac function in pregnant women with hyperthyroidism [5].

Although effective, antithyroid medications require frequent adjustments due to hormonal fluctuations during pregnancy. Optimal control of thyroid hormone levels not only reduces the risk of maternal hyperthyroidism but also minimizes fetal complications. For example, uncontrolled hyperthyroidism exposes the fetus to risks of neonatal hyperthyroidism, particularly if maternal TRAb levels remain high in the third trimester [6].

Neonatal hyperthyroidism is a rare but severe complication caused by elevated maternal TSH receptor antibody (TRAb) levels crossing the placenta and stimulating the fetal thyroid gland. Recent studies indicate that measuring TRAb levels in pregnant women with Graves' disease is a key indicator for assessing the risk of fetal and neonatal thyrotoxicosis. If TRAb levels are significantly elevated, fetal ultrasound monitoring should be implemented to evaluate signs of fetal hyperthyroidism, such as tachycardia or intrauterine growth restriction [10].

In our case, while TRAb levels were not measured, follow-up ultrasounds showed no fetal abnormalities. This underscores the importance of proactive management and monitoring of biological markers to prevent potential complications.

Management of thyrotoxicosis during pregnancy extends beyond the acute phase. Long-term risks for both mother and fetus must also be considered. Recent studies suggest that women who experience hyperthyroidism during pregnancy have an increased risk of postpartum relapse, particularly if Graves' disease is the underlying cause [5]. These patients require rigorous endocrinological follow-up, particularly in the months following delivery, to detect early signs of disease reactivation.

Thyroidectomy may be considered for patients intolerant to antithyroid drugs or with long-term contraindications, although this option is typically reserved for refractory cases or relapse after medical treatment [6]. In this case, the patient did not require surgical intervention, as medical management achieved rapid and effective control of thyrotoxicosis.

Fetal development can be severely compromised by poorly controlled hyperthyroidism. Impaired fetal growth, low birth weight, and long-term

neurodevelopmental disorders have been reported in children born to mothers with uncontrolled thyrotoxicosis [11]. However, strict control of thyroid hormone levels during pregnancy can minimize these risks.

In our case, the patient was stabilized quickly after initiating treatment, and follow-up ultrasounds revealed no fetal abnormalities. This outcome highlights the importance of early and aggressive management of thyrotoxicosis to prevent neonatal complications. Proper control of free T4 and TRAb levels during the second and third trimesters is essential to assess the risk of neonatal hyperthyroidism, although no abnormalities were observed in this particular case.

4. CONCLUSION

Thyrotoxic crisis in pregnancy, although rare, represents a severe endocrinological emergency requiring rigorous multidisciplinary management. This case illustrates the importance of rapid diagnosis and appropriate management to prevent maternal and fetal complications. Through close monitoring and antithyroid therapy tailored to pregnancy, the patient was stabilized without surgical intervention, and the pregnancy progressed favorably without neonatal complications.

Despite the rarity of this condition, it is imperative to consider it in any pregnant patient presenting with hyperthyroid symptoms. Early recognition of clinical signs, combined with prompt and individualized medical management, significantly improves prognoses for both mother and fetus.

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