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Molar Pregnancy Induced Hyperthyroidism: A Case Report

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Abstract **Case Report**

Hyperthyroidism is a rare complication of gestational trophoblastic disease (GTD). Here, We report the case of a 20-year-old patient who presented with signs of hyperthyroidism during a molar pregnancy. The diagnosis was confirmed through clinical examination, laboratory tests, and ultrasound findings. The serum thyroid hormones were elevated and returned to normal level after uterine evacuation of a molar pregnancy. Hyperthyroidism caused by hydatidiform mole is a rare form of pregnancy-induced hyperthyroidism and has a high mortality and life-threatening complication. Limited data are available regarding the optimal treatment, recognition and comprehensive management of the underlying etiology with a multidisciplinary team of endocrinologists, gynecologists and critical care physicians are crucial for a successful outcome.

Keywords: Hyperthyroidism, Gestational Trophoblastic Disease (GTD), Molar Pregnancy, Pregnancy-induced Hyperthyroidism, Uterine Evacuation.

INTRODUCTION

Gestational trophoblastic diseases (GTDs) are neoplastic disorders arising from the trophoblastic epithelium of the placenta, including hydatidiform moles (complete or partial), invasive mole, and choriocarcinoma. Thyrotoxicosis is a classic and rare complication of GTDs, in particular hydatidiform mole. In a normal pregnancy, β-human chorionic gonadotropin (hCG) exerts a mild thyrotropic effect, which can occasionally result in transient gestational hyperthyroidism. In GTD, markedly elevated hCG levels can further stimulate thyroid hormone production, potentially leading to thyrotoxicosis. Treatment is crucial to alleviate the patient's symptoms and prevent progression to thyroid storm, a life-threatening condition with a high mortality rate up to 30% even with appropriate management [1]. Here, We report the case of a 20-year-old patient who presented with signs of hyperthyroidism during pregnancy. This case highlights the importance of considering hyperthyroidism in patients with molar pregnancy and the need for appropriate management.

CASE PRESENTATION

A 20- year- old woman, G2P1, presented to her primary care provider with a 2 months history of vomiting, nausea and was unable to tolerate any food. symptoms of thyroid dysfunction are revealed: thremophobia apalpitation bleeding and weight loss. She had no significant past medical history, and her family history was unremarkable for any thyroid disorders.

Physical Examination

On examination, she was afebrile with blood pressure of 120/ 80 mm Hg and heart rate of 111 beats per minute. Head and neck examination demonstrated a non-tender and normal sized thyroid. There was no evidence of ophthalmopathy, acropachy, pretibial myxedema, or tremor. The remainder of the general examination was unremarkable.

Laboratory evaluation showed a normal complete blood count, hemoglobin 14,7 g/dL (range = 12-16), natraemia at 141 mmol/l, Hypokalemia at 2.7 mmol/l. Renal failure with urea at 0.49 g/l , creatinemia at 16.12 mg/l and GFR of 37.6 ml/min, an undetectable thyroid stimulating hormone (TSH) (<0,05 mui/ml), elevated free T4 (83,39 pmol/l; range = 12-22), elevated free T3 (22,41pmol/l; range=3,1-6,8). Transvaginal ultrasound of pelvis revealed a intrauterine pregnancy without a foetal heart beat, placenta with multiple tiny cystic lesions suggestive of a partial hydatidiform mole. Laboratory data showed β-hCG levels of 1,000,000 mIU/. Ultrasound of the thyroid gland showed generalized thyroid enlargement with uniform hypoechoic pattern, without a nodular appearance.

Treatment

Upon discovery of her suspected mole, biochemical hyperthyroidism and electrolyte disturbance (severe hypokalemia), the patient was transferred to intensive care unit for multidisciplinary specialist evaluation. Replacement therapy of hypokalemia was performed. The treating team urgently consulted the Endocrinology service,the patient was started on hydrocortisone 100 mg IV once and then 50 mg IV every 8 hours and propranolol 30 mg per day and patient was referred to the Obstetrics/Gynecology Department scheduled for a suction dilation and curettage to evacuate the mole and remove the ultimate source of the β-HCG. Laboratory tests after operative uterine evacuation showed declining β-HCG and free T4l (28,8 pmo/l).

DISCUSSION

Gestational trophoblastic disease (GTD) is a heterogeneous group of rare tumours characterised by abnormal proliferation of trophoblastic tissue [2]. Most of these conditions are associated with the proliferation of trophoblast. The incidence of molar pregnancies in the United States and other developed countries is 1 per 1500 to 2000 (3). In Morocco, the incidence is 4.3/1000 pregnancies [4]. Transient gestational thyrotoxicosis occurs in 1 to 3% of pregnancies and is usually limited to the first half of gestation [5]. Patients usually present with second trimester vaginal bleeding and a uterus larger than expected for the gestational age [6]. As diagnosis is often made in the first trimester with elevated β-hCG and ultrasound examination, complications such as hyperemesis gravidarum, pre-eclampsia, and hyperthyroidism are less common [7]. Molar pregnancy–induced hyperthyroidism is a rare clinical condition caused by elevated levels of hCG produced by the molar tissue. This excessive hCG stimulation activates TSH receptors, leading to either subclinical elevations of thyroid hormones or, in more severe cases, overt thyrotoxicosis [8].

The maternal thyroid gland is stimulated by the rise in human chorionic gonadotropin (hCG) in the 1st trimester of pregnancy. In hyperthyroidism secondary to trophoblastic disease, the symptoms associated with Graves' disease (ophthalmic disease, pretibial myxedema, and acropachy) are missing because this type of thyrotoxicosis is usually of shorter duration [9]. In contrast, partial moles usually present with mildly elevated β-hCG levels compared to complete hydatid form.

The thyrotropic activity of β-hCG stems from the structural similarity with TSH. Glycoprotein hormones such as TSH, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and hCG share a common alpha subunit, while their distinct beta subunits determine their specific biological roles. Research has shown that normal hCG can bind to TSH receptors in the thyroid gland, exerting thyrotropic effects. Notably, one unit of hCG has a reactivity equivalent to 0.0013 mU of TSH [10]. Moreover the level of sialylation of β-hCG determines its bioreactivity on the TSH receptors and the β-hCG produced by hydatidiform moles has greater thyrotropic activity than normal β-hCG due to its decreased sialylation [11]. One unit of β-hCG produced by gestational trophoblastic disease (GTD) has thyrotropic activity ranging from 3.72 to 46.8 uU equivalents of TSH. It is estimated that for every 10,000 IU/L increase in hCG, there is a 0.1 μIU/mL decrease in TSH and a 0.1 ng/dL increase in free T4. The biological profile shows an increase in freeT4 andT3 with an undetectable level of TSH [12, 13].

Currently, there are no evidence-based guidelines for managing hyperthyroidism caused by gestational trophoblastic disease (GTD). The treatment of such conditions includes corticosteroids, management of peripheral effects of thyroid hormones (beta-blockers) [14]. Anti-thyroid drugs like methimazole or propylthiouracil are required in some patients depending on the degree of hyperthyroidism. Hospitalization in an intensive care unit and the use of plasmapheresis have been reported by Moskovitz *et al*., [6]. The definitive treatment for hyperthyroidism in the context of molar pregnancy is the evacuation of the molar tissue, which leads to the resolution of hyperthyroid symptoms [2]. Follow-up with serial, once hCG levels have returned to the reference range for a period of four weeks, they are monitored monthly for the next six months to check for signs of gestational trophoblastic neoplasia by tracking any increase in β-hCG levels [14, 15].

CONCLUSION

This case highlights the importance of recognizing the link between gestational trophoblastic disease and thyrotoxicosis in woman of childbearing age to allow for timely initiation of appropriate preoperative treatment. The biological profile shows an elevation in free T4 and T3 levels with undetectable TSH. Thyrotoxic symptoms are generally tolerable. However, in some cases, these symptoms can be severe require multidisciplinary approach. More research is needed to define the optimal treatment plan for these atypical presentation.

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