Heart failure caused by thyrotoxicosis in pregnancy - case report

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Summary

Purpose of Investigation: To emphasize the importance of untreated thyrotoxicosis in pregnancy. When left untreated, severe maternal, fetal and even neonatal adverse outcomes such as preeclampsia, premature labor, low birthweight infants and increased perinatal mortality are prone to complicate the pregnancy.

Presentation: A case of thyrotoxicosis untreated during pregnancy is reported.

Conclusion: Many authors have concluded that there is no need for routine assessment of the thyroid hormones and TSH levels in pregnancy. Nonetheless laboratory assessment for thyrotoxicosis should be done in cases with suspicious symptoms and signs. All thyrotoxic women should also be under treatment during pregnancy. Early diagnosis and/or control of hyperthyroidism would decrease the incidence of complications during pregnancy.

Key words: Thyrotoxicosis; Pregnancy.

Introduction

Management of thyrotoxicosis in pregnancy is still a difficult challenge for obstetricians and endocrinologists. The incidence is estimated as 0.1-0.2% in pregnancy [1]. Classically it has been stated that Graves' disease is the most common cause of hyperthyroxinemia in pregnancy. Three forms of clinical presentation can be observed: 1) active disease under treatment; 2) remission state; or 3) yet undiagnosed. The last one has the greatest risk for obstetric complications. It is well known that untreated hyperthyroidism may cause severe adverse consequences, and maternal-fetal outcomes are directly related to the adequate control of hormone levels [2]. Untreated hyperthyroidism is associated with increased risk of preeclampsia, premature labor, low birthweight infants, and perinatal mortality [3]. Pregnancy itself also poses an additional risk to the condition. While cardiac high-output state of hyperthyroidism can be mostly compensated in non-pregnant cases, approximately 10% of untreated women experience cardiac failure during pregnancy [4]. Pregnancy complications such as severe preeclampsia, eclampsia, sepsis, hemorrhage and anemia can further increase the risk of cardiac decompensation [5]. In this report, we present a case of thyrotoxicosis complicated with heart failure during pregnancy and emphasize the importance of diagnosis and treatment before or in early phases of gestation.

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Case Report

A 22-year-old woman, gravida 1, para 0, was referred to our clinic at 31 weeks of pregnancy with suspected preeclampsia. She had an unremarkable medical history. She complained of respiratory distress and abdominal pain. Her blood pressure was 160/90 mmHg and body temperature was 38.5°C. She had tachycardia (152 beats/min) and tachypnea (45 per min). Significant exophthalmia and edema in the lower extremities were detected. Her thyroid gland was diffusely enlarged and her skin was sweaty. She also had muscle fasciculation, exaggerated deep-tendon reflexes and proximal muscle weakness. The findings were suggesting thyrotoxicosis.

Obstetric ultrasound examination showed a 31-week fetus. The fetus was anatomically normal and appropriately grown for the gestational age. Amniotic fluid volume was normal and, minimal sub-chorionic clot areas were detected inside the placenta. She had three uterine contractions in ten minutes and in cervical assessment there was 2 cm of dilation and 20% effacement. Fetal heart rate tracing showed significant fetal tachycardia (170/bpm) with normal variability.

In biochemical assessment, the results of complete blood count, liver and renal function tests were normal. Urine analysis showed moderate (2+) proteinuria. The serum TSH level was low (0.028 uIU/ml), and free T4 (> 6 ng/dl, normal reference 0.8-2.2 ng/dl) and total T3 3.36 ng/ml (normal reference 1.4-2.4 ng/ml) levels were high. Anti TPO (20.2 IU/ml) and anti TG (41.2 IU/ml) were negative, but thyroid stimulating immunoglobulin (TSI) was not determined.

With the diagnosis of preeclampsia, magnesium sulphate prophylaxis was initiated. After consultations with endocrinologists the diagnosis of thyrotoxicosis was also established, and propylthiouracil (200 mg/daily), propanolol (160 mg/daily) and prednisolone (48 mg/daily) medications were initiated for the treatment. Special care was taken for the urine output follow-up and fluid balance. After 13 hours, the respiratory complaints of

the patient worsened. The patient had more severe dyspnea with orthopnea and had bilateral rales in the basal segments of the both lungs. Chest radiography showed cardiomegaly with bilateral infiltrates at the bases of the lungs. The findings were compatible with the diagnosis of pulmonary edema and heart failure according to Framingham criteria. She was transferred to the intensive care unit with the diagnosis of thyroid storm.

After a short period of time, spontaneous decelerations and variability loss were detected during fetal heart monitorization, and the fetus was delivered immediately by cesarean section. A 1,600 g female neonate was delivered with 6/9 APGAR scores. Umbilical artery blood gas values were as follows: pH 7.109; PO_2 23 mmHg; PCO_2 73.2 mmHg; HCO_3 19.8 nmol/l and base excess -3.8 nmol/l. The patient stayed in the intensive care unit for one day and her laboratory and clinical signs returned to normal after ten days.

On the third postnatal day, the newborn had tachycardia, jitteriness, and diarrhea. On examination a small goiter was detected. Laboratory assessment of the newborn showed a raised FT4 concentration of 56 pmol/l and a reduced serum TSH level of < 0.02 mUI/ml. The neonate was successfully treated with oxygen, propanolol (1-2 mg/kg/d) and propylthiouracil therapy (10 mg/kg/d) and stayed in the neonatal intensive care unit for 20 days. She was discharged on the 31st day.

Discussion

The diagnosis of thyrotoxicosis in pregnancy can be difficult and confusing. The hyperkinetic state of pregnancy can mimic thyrotoxicosis. Furthermore, some physiologic changes seen in pregnancy can ameliorate symptoms of hyperthyroidism. Thus pregnancy can mask the signs and symptoms of hyperthyroidism. Common findings of hyperthyroidism are the presence of goiter, ophthalmopathy, proximal muscle weakness, tachycardia with a pulse rate above 100 beats/min, and weight loss or inability to gain weight in spite of a good appetite. Occasionally the patient may be seen for the first time with congestive heart failure secondary to long-term hyperthyroidism. Despite doubling or tripling of the cardiac output and a supernormal contractile function due to hyperthyroidism, the increase in both preload and blood volume causes an elevation in ventricular filling pressures and may lead to mild pulmonary and peripheral congestion. This "high-output heart failure" usually occurs in young individuals with severe and long-standing hyperthyroidism in the absence of any underlying heart disease and responds well to treatment. Once the thyroid tests normalize, cardiac findings such as systolic and diastolic murmurs disappear, as well as the radiologic enlargement of the cardiac silhouette. Therefore it is very important to distinguish symptoms and signs of thyrotoxicosis from pregnancy itself. In the first trimester, the occurrence of hyperemesis gravidarum accompanied by weight loss should raise the suspicion of thyrotoxicosis, and assessment of FT3, FT4 and TSH may be required. In our case, during the early antenatal visits, symptoms of severe hyperemesis were noticed and the disease was treated appropriately but no further investigation for thyrotoxicosis was made by the physicians. Hyperthyroidism was not diagnosed and the patient was not given medication until hospitalization in the 31st week of pregnancy. Another problem is that some women with thyrotoxicosis discontinue their medication in pregnancy with the fear of potential teratogen effects of the antithyroid drugs. In practice, many antithyroid drugs are nonteratogenic but patients must be informed about, and convinced of this reality.

Hyperthyroidism increases the risk of preterm delivery, preeclampsia and perinatal mortality in pregnancy [5, 7]. Nonpregnant women can usually tolerate the effects of hyperthyroidism. However alteration in the peripheral arterial system, hypervolemia and high cardiac output seen in pregnancy could facilitate heart failure. It is reported that heart failure can occur in 10% of pregnant women with uncontrolled hyperthyroidism [8-10]. Coexistent obstetric complications such as sepsis, hemorrhage and severe preeclampsia-eclampsia are common precipitating factors for cardiac decompensation. Our patient presented with the findings of preeclampsia and regular uterine contractions on admission. She also had the symptoms of cardiac failure, and the severity of heart failure had progressed with the additive effect of preeclampsia despite a regular balance between fluid intake and diuresis. The patient needed intensive care due to pulmonary edema with severe metabolic acidosis and hypoxia. The fetus subsequently developed fetal distress and was delivered immediately. It is likely that the fetal condition was affected by maternal pulmonary edema and severe heart failure.

Klein *et al.* concluded that there is no need for routine assessment of the thyroid hormones and TSH levels in pregnancy [11]. Nonetheless, in cases with suspicious symptoms and signs we recommend that a laboratory assessment for thyrotoxicosis should be done. Early diagnosis and control of the hyperthyroidism would decrease the incidence of complications. Special care must be taken for thyrotoxicosis during pregnancy. Severe complications of thyrotoxicosis in pregnant women, such as heart failure, can be prevented by early initiation of treatment, and by prevention and appropriate management of detoriating pregnancy complications, such as preeclampsia, anemia and infections.

Conclusion

Many authors have concluded that there is no need for routine assessment of the thyroid hormones and TSH levels in pregnancy. Nonetheless laboratory assessment for thyrotoxicosis should be done in cases with suspicious symptoms and signs. All thyrotoxic women should also be under treatment during pregnancy. Early diagnosis and/or control of the hyperthyroidism would decrease the incidence of complications in pregnancy.

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