Pregnancy Complicated with Pulmonary Edema Due to Hyperthyroidism

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Hyperthyroidism is one of the most common causes of cardiac failure. Blood volume expands greatly during pregnancy, especially after the last part of the second trimester. Such expansion exacerbates the symptoms of heart failure and accelerates the development of pulmonary edema when abnormal thyroid function is not well controlled. Two cases of pregnancy complicated with congestive heart failure and pulmonary edema due to hyperthyroidism are reported here. Both patients did not receive treatment for hyperthyroidism during pregnancy, and both sought clinical help during the third trimester. The clinical problems were resolved by medical management before delivery. [*J Chin Med Assoc* 2005;68(7):336–338]

Key Words: heart failure, hyperthyroidism, pregnancy, pulmonary edema

Introduction

The following are causes of acute pulmonary edema in obstetric cases: cardiac failure, tocolytic therapy, iatrogenic fluid overload, severe pre-eclampsia, infection, and renal or hepatic failure.[1] Since hyperthyroidism is one of the most common medical diseases in the reproductive years,[2] it is not uncommonly encountered during pregnancy. Cardiac failure may develop when hyperthyroidism is not well controlled,[3] and can result in pulmonary edema when blood volume expands greatly with advanced gestational age. In this report, the cases of 2 pregnant women suffering from hyperthyroidism complicated with heart failure and pulmonary edema are presented.

Case Reports

Case 1

A 30-year-old, gravida 4, para 2, woman was transferred to our hospital because of orthopnea, dyspnea on exertion, hypertension, and generalized edema when she was 30 weeks' pregnant with twins. Despite normal maternal heart structure revealed by echocardiogram, the patient's cardiac function was assigned as New York Heart Association class IV. Although dilatation of both ventricles and mild pulmonary hypertension was found, left ventricular systolic function was preserved. The patient, who had a family history of hyperthyroidism, had experienced a similar episode more than 1 year before this pregnancy and had discontinued antithyroid medication in the early stages of pregnancy. On admission, the patient's serum levels of free thyroxine (free T_4) and thyrotropin (thyroid stimulating hormone, TSH) were 3.38 ng/dL (normal, 0.4–1.9 ng/dL) and 0.006 μIU/mL (normal, 0.4–4.0 μIU/mL), respectively. Chest X-ray showed cardiomegaly with pulmonary edema. The patient suffered from pre-eclampsia, with hypertension, proteinuria, and marked edema. High central venous pressure (28 cm H_2O) and pulmonary capillary wedge pressure (23 cm H_2O) were also found. The patient's condition was controlled after propylthiouracil, propranolol, and furosemide were administered, along with a total

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loss of 20 liters of body fluid. In addition, an intravenous injection of magnesium sulfate was given for tocolysis. Unfortunately, 1 fetus died on the third hospital day. The patient gave birth to the babies vaginally at the 38th week of gestation, with 1 healthy girl weighing 2,264 g; the other baby, weighing 1,510 g, had died from a twisted cord *in utero*.

Case 2

A 41-year-old, gravida 3, para 2, woman was admitted because of palpitations, tachycardia, and high blood pressure, on her first visit to our hospital during her 34th week of gestation. Although the patient, who had a family history of thyroid disease, had been diagnosed with hyperthyroidism 3 years earlier, she had refused medical treatment. On admission, blood tests revealed free T_4 4.80 ng/dL, and TSH 0.00 μIU/mL. Chest X-ray showed cardiomegaly and mild bilateral pleural effusion, suggesting pulmonary congestion or edema. Echocardiography revealed normal left-ventricle chamber size, with preserved left ventricular systolic function, and mild mitral valve prolapse, mitral regurgitation, pulmonary regurgitation, and pulmonary hypertension. The patient's condition stabilized within 10 hospital days after treatment with oral propylthiouracil 100 mg 4 times daily, and propranolol 10 mg twice daily, plus intravenous furosemide 40 mg twice daily. The patient gave birth vaginally at her 38th week of gestation to a healthy male baby weighing 3,000 g.

Discussion

The 2 most serious maternal complications of untreated hyperthyroidism are heart failure, being more common, and thyroid storm.[4] Heart failure is caused by the long-term myocardial effects of thyroxine,[2] and is aggravated in the latter half of pregnancy because of increased cardiac burden due to major blood volume expansion.[3] Such expansion is the principal reason why, as in our 2 cases, pregnant women with hyperthyroidism seek medical treatment during the third trimester.

Pulmonary hypertension, which is correlated with untreated or inadequately controlled hyperthyroidism,[5] can result in heart failure and pulmonary edema. Maternal mortality may occur peripartum, especially immediately after delivery, because of excessive venous return caused by the shrunken, gravid uterus. Unlike pregnancies complicated with organic heart diseases such as severe mitral or aortic stenosis, the prognosis of maternal heart failure combined with pulmonary edema due to hyperthyroidism is more favorable if hyperthyroid treatment is started as early as possible antepartum. However, it is critical to rule out the probability of organic heart disease. In this report, echocardiography performed in both patients revealed normal cardiac function and excluded the probability of cardiac abnormalities.

Adverse effects on pregnancy outcome, including spontaneous abortion, preterm delivery, stillbirth, low birth weight infants, and pre-eclampsia, are commonly seen in pregnancies complicated with untreated hyperthyroidism.[3,6–8] In case 1, 1 fetus died on the third hospital day because of a twisted cord *in utero*, rather than because of the effects of uncontrolled hyperthyroidism or the effects of the drug treatments used. Uncontrolled hyperthyroidism and multiple pregnancies were the probable causes of pre-eclampsia in this study. Fortunately, each patient's blood pressure was stable after the crises were over.

Medical therapy with thioamides, either propylthiouracil or methimazole, is safe and effective in the treatment of hyperthyroidism during pregnancy.[9] Good control can be obtained within a 2-month period after the patient receives adequate doses of thioamides;[9] maintenance of the least effective dose of these compounds is required until delivery and thereafter. Although beta-blockers can have potential adverse effects on the fetus, such as growth restriction, fetal bradycardia, neonatal respiratory depression, and neonatal hypoglycemia,[10–13] these agents are still required to provide symptomatic relief in pregnant mothers with thyroid toxicity at the initiation of thioamide therapy.[10] Despite neonatal hyperbilirubinemia as a possible adverse effect, furosemide is still mandatory for treating life-threatening pulmonary edema.[14] Beta-blockers and diuretics are not needed if the symptoms of heart failure subside.

In conclusion, the treatment of hyperthyroidism during pregnancy should be started as soon as possible after the diagnosis is confirmed. Beta-blockers and diuretics are not contraindicated during pregnancy complicated with life-threatening conditions. The treatment of heart failure induced by uncontrolled hyperthyroidism during pregnancy requires preferential management, unless delivery is imminent.

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