Gestational Trophoblastic Disease with Hyperthyroidism and Severe Pre-eclampsia: Anesthetic Management

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ABSTRACT

Gestational trophoblastic disease originates from abnormal proliferation of molar tissue and most of them are not complicated. However, life threatening complications such as hyperthyroidism and severe pre-eclampsia can occur. We report the successful anesthetic management with general anesthesia technique in the molar pregnancy with pre-eclampsia and hyperthyroidism scheduled for trans-abdominal hysterectomy without any complications.

Keywords: Anesthesia, gestational trophoblastic, hyperthyroidism, severe pre-eclampsia

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INTRODUCTION

Gestational trophoblastic disease (GTD) is a spectrum of tumors which results from an abnormal proliferation of trophoblastic tissue in the developing placenta.[1] Hyperthyroidism is due to stimulation of thyroid gland by the high levels of human chorionic gonadotropin (hCG) which its alpha subunit is similar to the structure of thyroid stimulating hormone (TSH) or by a thyrotropin produced by trophoblasts.[2] GTD involves increased progesterone level and released vascular endothelial growth factor which can cause pregnancy induced hypertension in up to 27% of women.[3] We report the anesthetic management of a patient with GTD complicated with these two rare conditions.

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CASE REPORT

A 45-year-old woman, G5P3A1, presented with amenorrhea for 4 months and diagnosis of molar pregnancy was made from the urine pregnancy test and abdominal ultrasonography. She underwent dilation and curettage. Two weeks later she presented with severe nausea, vomiting and the uterus size was 16 weeks' pregnancy size. The serology showed beta-hCG 718,886 IU/l, TSH <0.05 uIU/mL (0.27-4.2), freeT4 3.3 ng/dl (0.93-1.7) and free T3 7.92 pg/ml (2.0-4.4), which are features consistent with retained molar pregnancy and hyperthyroidism. She received the second dilation and curettage together with oral methimazole and propranolol. A week later she developed hypertension and beta-hCG was 919,910 IU/l. The obstetrician started the chemotherapy (methotrexate and folinic acid) and antihypertensive drug (amlodipine). After the treatment, platelet count decreased to 58,000/ul.

Five days later, she developed epigastric pain and the blood pressure was 180/110 mmHg. The diagnosis was severe pre-eclampsia and treatment with MgSO4 was started. She was posted for transabdominal hysterectomy, but due to her low level of platelets, she was transferred to our hospital.

On physical examination, her body weight was 60 kg, height 145 cm, temperature 37.5°C with pulse rate of 85/min, blood pressure 155/97 mmHg, and respiratory rate 25/min. She had pitting edema on both legs without tremors or ophthalmopathy. Thyroid gland was not enlarged and no bruit was heard. She had tachypnea and fine crepitation on both lower lungs. Her heart sounds were normal. Abdominal examination showed positive shifting dullness, fluid thrill and the uterus was 18 weeks' pregnancy size.

Laboratory results were hemoglobin 10.8 g/dl, platelet count 52,000/ul, serum bicarbonate 18 mmol/L. Liver function tests showed SGOT 90, SGPT 58 and ALP 81 U/L. Total protein levels were 5.5 and albumin 2.6 g/dl. Thyroid function tests revealed TSH <0.005 uIU/mL, free T3 2.7 pg/ml, free T4 1.91 ng/ml and beta-hCG 728,683 IU/l. Coagulation tests were in normal limit. Chest radiography (Fig 1) showed interstitial infiltration in both lower lungs. Transvaginal ultrasonography (Fig 2) revealed a uterus size 18*10*12 cms with mixed echoic, multiple vesicle lesion, which are features consistent with hydatidiform mole.

At our hospital, she received propranolol tablet 10 mg three times a day and continued MgSO4 infusion. Her vital signs were controlled so she was scheduled for transabdominal hysterectomy on the following day.

General anesthesia was planned with invasive arterial pressure prior to induction, ECG, oxygen saturation, end tidal carbon dioxide, temperature, nerve stimulator and urine output monitoring. In the operating room her blood pressure was 170/90 mmHg, heart rate 80/min, respiratory rate 26/min and oxygen saturation of 96%. Rapid sequence induction was done with intravenous propofol 2 mg/kg, rocuronium 0.1 mg/kg and fentanyl 2 mcg/kg. The patient was intubated with a 7.0 number cuffed endotracheal tube. Anesthesia was maintained with O_2, air, sevoflurane 1.0-1.5% and morphine. Hemodynamic parameters and oxygen saturation were maintained throughout the operation and no manifestations of thyroid storm, hypertension, hypotension or arrthymia were noted. The patient received 600 mL of Ringer's lactate solution. Ascites fluid was 1,000 ml, blood loss 200 ml and urine output 180 ml. Arterial blood gas on FiO_2 0.6 showed pH 7.38, pCO_2 40 mmHg, pO_2 260 mmHg, bicarbonate 24 mmol/L, base excess 0.5 and SaO_2 99%. The operative time was 2.5 hours. The patient was extubated and diltiazem 2 mg was administered for

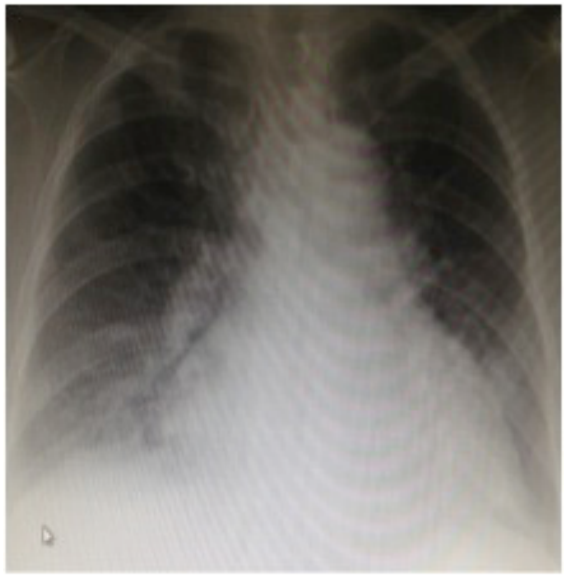

Fig 1. Chest radiography.

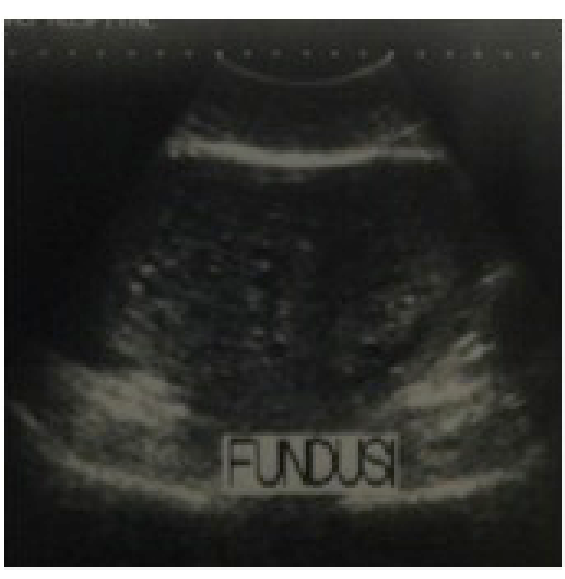

Fig 2. Transvaginal ultrasonography.

TABLE 1. Pre and postoperative laboratory value.

Lab values	Normal range	Pre-operative	Post-operative
Platelets (/ul)	-	52,000	274,000
Free T3 (pg/ml)	2.0-4.4	2.7	-
Free T4 (ng/ml)	0.93-1.7	1.91	-
TSH (uIU/mL)	0.27-4.2	<0.005	-
beta-hCG (IU/l) for 17-24 weeks pregnancy	4,060 - 165,400	728,683	5,458

controlling her blood pressure. Her hemodynamic paremeters were stable in the recovery room. Then she was transferred to a general ward. MgSO4 was stopped, and intermittent intravenous morphine was given for pain control.

On 1st post-operative day her blood pressure was 140/90 mmHg without any antihypertensive drug. She had bleeding at her gum and no other abnormal bleeding. Platelet decreased to 26,000/ul, hemoglobin was 10 g/dl and beta-hCG 150,653 IU/l. She did not received any blood or platelet transfusion. Platelets increased to 274,000/ul on the 5th post-operative day. She was discharged on the 8th post-operative day and beta-hCG was 5,458 IU/l. The pathological finding was invasive mole. Nine weeks after discharge, her blood pressure was in normal limits and beta-hCG decreased to 979 IU/l.

DISCUSSION

Specific complications of molar pregnancy include hyperemesisgavidarum, gestatinonal hypertension and pre-eclampsia, anemia, and thyrotoxicosis. GTD complicated with hyperthyroidism and severe pre-eclampsia is rare and there are only few case reports of these associations.[4] Hyperthyroid state can range from asymptomatic increase of thyroid hormones to thyroid storm. About 7% and 2% of women with molar pregnancy develop biochemical and clinical hyperthyroidism, respectively.[5] The goal of preoperative management is optimization of the hyperthyroid state. Propylthiouracil, methimazole, Lugol's iodine and beta adrenergic blockers are used for blocking thyroid hormone synthesis. Despite the maximal medical control of hyperthyroidism and treatment with chemotherapy which acts directly against the tumor, the hyperthyroid state is only partially controlled in some patients. It sometimes needs to be optimized over a short period in the emergency setting. There are case reports using of plasmapheresis as an alternative treatment to obtain rapid normalization of serum thyroid hormone levels before surgery.[6] The hyperthyroid state resolves rapidly after surgical evacuation of trophoblastic tissue.[7] This patient had no symptoms of hyperthyroidism. She was treated with methimazole and propranolol before surgery and did not continue any medication after surgery.

Pre-eclampsia which happens in the 1st or early 2nd trimester is suspected with a sign of a hydatidiform mole, because it rarely occurs in the early stage of a normal pregnancy[8].This patient had clinical signs and symptoms of severe pre-eclampsia such as pulmonary edema, ascites and thrombocytopenia which can lead to eclampsia. MgSO4 was administered to decrease peripheral vascular resistance and risk of seizure. It has some analgesic properties which are enhanced by volatile anesthetics and also potentiates the effects of non-depolarizing neuromuscular blocker. For this patient, general anesthesia was planned because of abnormal platelet count and rapid sequence induction was performed to prevent aspiration due to abdominal distention from ascites and large uterus size. Neuromuscular junction activity was monitored by using a nerve stimulator. Volume status was meticulously adjusted because this patient had pulmonary edema and low serum albumin level. There was no abnormal bleeding from her surgical field, so she did not receive platelet transfusion. Intraoperative hemodynamic disturbance especially during intubation and extubation was

controlled to prevent cardiovascular and cerebral complications. Post-operative close monitoring of thyroid storm and severe pre-eclampsia was needed. After removal of her trophoblastic tissue, the patient had stable hemodynamic parameters and hCG level tapered down.

In conclusion, anesthesiologists should be aware of perioperative complications that can occur with gestational trophoblastic disease such as hyperthyroidism and pre-eclampsia. Preoperative investigation, optimization of a patient's conditions, which require knowledge, vigilance and a multidisciplinary team approach are needed for the prevention of morbidity and mortality and better outcome of a patient.

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