The Journal of
OBSTETRICS AND GYNECOLOGY of India

J Obstet Gynecol India Vol. 59, No. 4 : July/August 2009 pg 356-357

Case Report

Hydatidiform mole with hyperthyroidism – perioperative challenges

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Key words : hydatidiform mole, hyperthyroidism, anaesthesia

Introduction

Hydatidiform pregnancy occurs in 1:1000 pregnancies worldwide. Incidence is higher in Asian countries. Prevalence of hyperthyroidism during complete molar pregnancy is as high as 7%[1]. Thyroid hyperfunction in molar pregnancy is attributed to excess of human chorionic gonadotropin, which has a weak intrinsic thyroid stimulating activity and molar thyrotropin, which differs from hCG by being larger in molecular size and longer in duration of action[2]. Clinical hyperthyroidism due to trophoblastic disease is cured by evacuation of molar tissue. The anesthesia management in these women is very challenging and serious morbidity and even mortality can result if adequate precautionary measures are not taken prior to evacuation.

Case report

A 26 year old, 40 kg female (gravida 3, para 0) presented to the antenatal clinic with a history of 2 months amenorrhea, excessive vomiting, fever, and vaginal bleeding. She had undergone check curettage with histopathology indicative of vesicular mole twice in the past two years. During the course of her investigations then, thyroid stimulating hormone (TSH) and prolactin levels had been checked and were normal. However, despite medical advice regarding regular follow up, she had failed to do so. Her present complaints were a history of weight loss and generalized weakness.

On examination, she was thin built with an agitated look. Her pulse rate was 140/minute and blood pressure 110/70 mm Hg. A thyroid swelling was noted on examination. There was no evidence of airway obstruction, ophthalmopathy, dermopathy or tremors. Heart sounds were normal. There was no murmur on auscultation. Rest of the systemic examination was unremarkable. A transvaginal sonograph revealed complete vesicular mole.

The rest of her investigations were as follows: Hemoglobin 10.2 g/dL βhCG: 1289474 microunits/mL. TSH <0.01 microIU/mL, T3 700ng/dL, and T4> 30/µg/dL. Liver function tests, renal function tests and chest x-ray were normal. An endocrinologist's opinion was sought. She was started on tablet propylthiouracil 100mg three times a day, tablet, propranolol 20mg three times

Paper received on 26/04/2006 ; accepted on 01/10/2006

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days and tablet dexamethasone 0.5mg, three times a day. Lugol's iodine 8 drops three times a day was given with strict monitoring of pulse rate and blood pressure. After one week, her pulse rate stabilized at 84/minute. On the day before surgery, Lugol's iodine was stopped and tablet diazepam 10 mg was administered at bedtime.

It was decided to administer spinal anesthesia. Consent was obtained after explaining risks due to thyrotoxicosis. Subarachnoid block with 2.5 cc of 0.5% bupivacaine was given and a sensory level of T10 achieved. Suction evacuation was performed. She maintained stable hemodynamics throughout the procedure. Monitoring included pulse oximetry, ECG and noninvasive blood pressure measurement. Postoperative course was uneventful. Histopathology confirmed complete vesicular mole. Propylthiouracil was stopped on the 1st postoperative day. Dexamethasone was tapered and stopped in the next 4 days. The postoperative investigations on day 3 were as follows: B βhCG 4100 micro IU mL. T3 0.9 ng/dL, T4 14.1 µg/dL. She was discharged on the 7th day with advice to follow up regularly with βhCG levels and to use barrier contraception until the βhCG levels returned to normal.

Discussion

Trophoblastic hyperthyroidism poses a multitude of challenges to the anesthesiologist. High output cardiac failure secondary to thyrotoxicosis, thyroid storm, hypertension, embolisation of pulmonary arteries by trophoblastic materials, hypovolemia, disseminated intravascular coagulation, and pulmonary edema secondary to severe anemia can occur in the perioperative period.

Use of both general [3] and spinal [4] anesthesia has been reported for evacuation of the mole. General anesthesia may be the preferred technic in hypotensive bleeding patients scheduled for emergency evacuation. As there is no time to make the patient euthyroid, intravenous administration of iodine and β blockers for emergency management of hyperthyroidism is advisable. Uterine relaxation caused by inhaled anesthetics may however increase blood loss. In stable patients, spinal anesthesia is preferable due to its nontocolytic properties and safety in hyperthyroid patients. Intravenous fluids and blood must be administered judiciously as these patients have a propensity to develop pulmonary edema.

Thyrotoxic crisis or thyroid storm is an extreme accentuation of thyrotoxicosis. Thyroid storm occurs in 2% to 4% of pregnant women with hyperthyroidism [5]. Most cases of thyroid storm during pregnancy occur in patients who have received either incomplete or no treatment of the preexisting hyperthyroidism. Marked tachycardia, arrhythmias, pulmonary edema and congestive cardiac failure may occur [6]. If unrecognized the condition is invariably fatal. Combined use of propylthiouracil, iodide and dexamethasone restores serum T3 concentration to within normal range within 24 to 48 hours. In the absence of cardiac insufficiency, β blockers to ameliorate symptoms should be started preoperatively.

Women with a history of molar pregnancy are at a risk of further molar pregnancies. Much has been reported in the literature about the incidence, recurrence and persistence of hydatidiform mole. Thyroid function tests should be mandatory in all women with hydatidiform mole and these women should be stabilized with β blockers and antithyroid medication prior to induction of anesthesia for their surgical evacuation. Vigilant monitoring and intensive care should be extended into the postoperative period because there is a likelihood of occurrence of cardiopulmonary complications, thyroid storm, and disseminated intravascular coagulation.

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