

# Preoperative Preparation And General Anesthesia Administration With Sevoflurane In A Patient Who Develops Thyrotoxicosis And Cardiogenic Dysfunction Due To A Hydatidiform Mole

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## Abstract

### INTRODUCTION

A hydatidiform mole is a gestational trophoblastic disease that originates from the placenta. It is a rare but life threatening disorder. Human Chorionic Gonadotrophin (hCG) present in the human placenta is identical with the  $\alpha$ -subunit of the Thyroid-Stimulating Hormone (TSH) but has a weak thyroid-stimulating activity during normal pregnancy.

Molar thyrotropin existing in the serum of the patient with hydatidiform moles differs from hCG by its larger molecular size and the duration of action. These substances are responsible for thyrotoxicosis observed in gestational trophoblastic patients.

### CASE REPORT

A 27 year-old woman with 17 weeks twin-pregnancy was admitted to our hospital complaining of sweating, dyspnea, hypertension, vaginal bleeding, and leg swelling. Hydatidiform mole, thyrotoxicosis and preeclampsia were diagnosed and an anesthesia consultation was requested because the mole had to be removed urgently. The patient was anxious and vital signs were as follows:

Blood pressure 180/110 mmHg, heart rate 116 beats/minute, respiration rate 24/ minute, body temperature 36,6°C.

In the physical examination conjunctival hyperemia, moderate pretibial edema, and bilateral pulmonary sibilant rales were detected. The thyroid gland was diffusely palpable. Laboratory findings were as follows:

Haemoglobin: 8,7 g/dL, White Blood Cells: 7300, Platelets: 149.000 /mm<sup>3</sup>

Serum total protein: 4,3g/dl, Albumine: 2,5g/dl, ALT: 366u/L, LDH: 656u/L, Urinary protein: 800mg/24h, FreeT3: 10,79 pq/ml, FreeT4: 3,33 ng/ml, TSH: 0.00ulU, HCG: 460.000mIU/ml, Arterial blood gases: pH: 7,31, PaO<sub>2</sub>: 81,9 mmHg, PaCO<sub>2</sub>: 40,1mmHg, HCO<sub>3</sub>: 20,2 mmol/L, O<sub>2</sub>SAT: %97,6

The electrocardiogram was consistent with sinus tachycardia. The chest radiogram demonstrated basilar stasis and there was diffuse hyperplasia in the thyroid sonography. The patient was admitted to the intensive care unit and propylthiouracil 100 mg/ 6 hours, propranolol HCL 20 mg/ 8 hours, captopril 25mg/ 12 hours, and amlodipine 10mg/24 hours were administered. 2 units whole blood and 2 units erythrocyte suspension was given for preoperative anemia. 24 hours later the patient's general well being partly improved and her repeated thyroid functions were as follows:

FreeT3: 5,19 pq/ml, FreeT4: 2,21 ng/ml, TSH: 0.00ulU,

Arterial blood gases: pH: 7,465 nEqL, PaO<sub>2</sub>: 70,8 mmHg, PaCO<sub>2</sub>: 27,6mmHg, HCO<sub>3</sub>: 21,8mmol/L, O<sub>2</sub>SAT: %98,5

After stopping oral medication she was admitted to the operation room with a sodium nitroprusside infusion to control her blood pressure fluctuations. Anesthesia was induced by midazolam, propofol, fentanyl and maintained with 2.5 % sevoflurane + nitrous oxide 50 % in oxygen. Vecuronium bromide was used for neuromuscular blockade.

During the intraoperative period, 20 mg furosemid, 40 mg methylprednisolone, 1 unit whole blood, and 1 unit erythrocyte suspension was given. 500 cc colloid and 2000 cc crystalloid fluid were administered for hydration. After evacuating the mole and the coexisting viable fetus by vacuum aspiration and mechanical extraction, 10 IU oxytocin as well as 0.2 mg methylergobasine were administered. The patient recovered without any problem and was admitted to the intensive care unit with spontaneous respiration under oxygen support.

Oral intake resumption of propylthiouracil 100mg/6 hours and propranolol HCL 20mg/8 hours was prescribed again. On the first postoperative, the pathologic hemodynamic activity partially regressed while the thyroid function tests were detected as follows:

FreeT3: 2.11 pg/ml, FreeT4: 1.11ng/ml, TSH: 0.00uIU.

By the third postoperative day, vital signs improved and propylthiouracil dose could be decreased to 50 mg/6 hours. On the fourth postoperative day, the patient was transferred to the normal clinic and on the fourteenth day, medications were stopped gradually and hCG levels were followed periodically.

## DISCUSSION

The treatment of an hydatidiform mole is surgical evacuation. Serious morbidity and even mortality can be seen if no precautionary measures are taken during the evacuation of the molar pregnancy. Thyrotoxicosis, anemia and dehydration due to bleeding aggravate cardiac dysfunction and respiratory insufficiency. The patients must be admitted to an intensive care unit preoperatively and blood count, electrolytes, blood gases, thyroid, hepatic and renal functions,  $\beta$  hCG and chest radiogram should be carefully evaluated. As there is no time to make the patient pharmacologically euthyroid, intravenous administration of iodine and  $\beta$ -adrenergic receptor blockers for emergency treatment of hyperthyroidism may be advisable. Perioperative management of thyrotoxicosis focuses on the control of sympathetic activity so that cardiovascular side effects are not manifested. After adequate supportive treatment the molar pregnancy must be evacuated.

Excessive fluid infusion must be avoided to prevent pulmonary edema and diuretics should be given intermittently. Mechanical ventilation support must be provided if necessary. Blood replacement may be required to

treat bleeding. Uterine relaxation may increase blood loss and inhaled anesthetics with known tocolytic qualities such as halotane, enflurane and isoflurane should therefore only be used in low concentrations. Nitrous oxide, opioids and muscle relaxant agents may be preferred. In our case, we used 2.5 % Sevoflurane + Nitrous oxide 50 % in oxygen combination and we did not observe any side effects. Solak and Akturk preferred spinal anesthesia in stable patients because of its preferable effects on the pulmonary system, and nontocolytic pharmacologic properties.

During the evacuation of the mole extreme uterine contractions may lead to trophoblastic embolization. Twiggs et al. describes a patient who had two episodes of subclinical pulmonary embolization after evacuation of the mole. Therefore, if bleeding and uterine contractions cannot be controlled properly oxytocin and ergometrine should be administered during and after the evacuation. We must be aware of the possibility of a thyroid storm during evacuation as reported by Kim et al.. Yao suggested that thyrotoxicosis thyroid storm was mostly seen in the postoperatively period.

Because of cardiopulmonary complications in the postoperative period intensive care management is essential. Besides pulmonary embolization and disseminated intravascular coagulopathy development must be considered.

## CORRESPONDENCE TO

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