

CASE REPORT

Total intravenous anesthesia for evacuation of a hydatidiform mole and termination of pregnancy in a patient with thyrotoxicosis

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SUMMARY. Clinical hyperthyroidism is found in approximately 5% of women with a hydatidiform mole, as human chorionic gonadotropin secreted by molar tissue is structurally similar to thyroid-stimulating hormone. A hydatidiform mole occasionally presents with a co-existing viable fetus. Surgical evacuation may be indicated for significant hemorrhage or preeclampsia. Perioperative management in the presence of hyperthyroidism may be complicated by a thyroid storm. We report a case of total intravenous anesthesia with propofol and remifentanyl, combined with an esmolol infusion, to control sympathetic hyperactivity during surgery.

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INTRODUCTION

Gestational trophoblastic disease, of which hydatidiform mole is the most common presentation, is an uncommon complication of pregnancy, occurring in 0.5 to 2.5 per 1000 pregnancies.¹ In approximately 5% of cases of hydatidiform mole, clinical hyperthyroidism is present² and occasionally severe thyrotoxicosis may develop.^{3,4} This results from secretion of human chorionic gonadotropin (hCG), the α subunit of which is structurally similar to thyroid stimulating hormone (TSH).

The presence of both fetus and molar tissue is rare. The probability of achieving a viable baby is approximately 40%, although continuation of pregnancy carries the risk of hemorrhage, preeclampsia and pulmonary emboli. Consequently women must be adequately counseled regarding these risks and, when appropriate, the pregnancy may be terminated.^{5–7} In severe thyrotoxicosis, where maternal health is threatened, termination of pregnancy may be advocated in addition to medical treatment to reduce the risk of a thyroid storm.^{4,8} When surgery is required total intravenous anesthesia (TIVA) may be of benefit although its use in this situation has not previously been reported. We describe the anesthetic

management of a case of surgical evacuation of molar tissue and termination of pregnancy complicated by hyperthyroidism, using TIVA with propofol, remifentanyl and esmolol.

CASE REPORT

A 25-year-old nulliparous woman was admitted to the emergency department at 12 weeks' gestation with vaginal bleeding associated with severe nausea and vomiting. She reported a one-month history of heat intolerance, sweating, agitation, palpitations and weight loss. She weighed 50 kg and was 160 cm tall (body mass index 19.5 kg/m²). There was no significant medical history. On examination, she was pale and agitated and looked very tired. Her heart rate was 145 beats/min and her blood pressure 150/85 mmHg. No abnormality was detected on palpation of her thyroid gland.

Her hemoglobin was 8.2 g/dL, a fall from a previous value of 12.8 g/dL taken two weeks previously. The serum β -hCG concentration was 650 000 mU/mL (normal non-pregnant range 0–5.3). Thyroid function tests showed a free iodothyronine (FT₃) 15.92 pg/mL (normal range, 1.8–4.6 pg/mL), free thyroxine (FT₄) 5.71 ng/dL (normal range, 0.9–1.7 ng/dL), and thyroid stimulating hormone (TSH) 0.01 μ U/mL (normal range, 0.27–4.2 μ U/mL). Her biochemical profile was otherwise normal. A hydatidiform mole with a co-existing live fetus was diagnosed by ultrasound. Computerized

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tomography scanning revealed minimal trophoblastic invasion of the myometrium. Thyroid scintigraphy was normal.

A multi-disciplinary approach to management of the case was adopted with the involvement of obstetricians, endocrinologists and anesthesiologists. Her thyrotoxic state was considered to be related to the hydatidiform mole and because of severe bleeding and associated anemia, termination of pregnancy and evacuation of the molar tissue were advised. The risks of continuing the pregnancy were discussed with the woman and her written consent for the procedure was given.

Methimazole (10 mg 8-hourly) and propranolol (20 mg 6-hourly) were started to treat the thyrotoxicosis and achieve hemodynamic stability before surgery. However, as this was not effective, plasmapheresis was performed on two consecutive days, each treatment lasting 4 h in an effort to reduce the serum levels of FT₃ and FT₄. This was partially successful (Table 1) although FT₃ and FT₄ values did not return to normal. As vaginal bleeding continued, the risks of further delay whilst trying to normalize thyroid function were considered greater than those of proceeding with surgery. Consequently on the fifth day after admission, surgery was performed. Intramuscular midazolam 5 mg was given 45 min before transfer to the operating room. On arrival, electrocardiography (ECG), pulse oximetry (SpO₂), non-invasive blood pressure and tympanic membrane temperature were monitored. At this stage there was a sinus tachycardia of 130-150 beats/min with a blood pressure of 145/85 mmHg and the patient was still extremely agitated. A further 3 mg of midazolam was administered i.v. with little effect. Regional anesthesia was therefore considered unsuitable and total intravenous anesthesia (TIVA) was planned. A 20-gauge catheter was inserted into the left radial artery and bispectral index (BIS) monitoring applied. Anesthesia was induced with thiopental 250 mg and fentanyl 100 µg; muscle relaxation was achieved with vecuronium 5 mg. To attenuate the hemodynamic response to intubation esmolol 15 mg was given. The trachea was intubated and the lungs ventilated with a mixture of 35-65% oxygen in air. Anesthesia was maintained using propofol 125-250 µg·kg⁻¹min⁻¹ and remifentanyl 0.1-0.25 µg·kg⁻¹min⁻¹. Infusions were adjusted to maintain a BIS value between 40 and 60. Tachy-

cardia was controlled with an esmolol infusion of 200-300 µg·kg⁻¹min⁻¹.

The uterus was evacuated, with removal of molar tissue and termination of pregnancy. The blood loss was approximately 2000 mL. During the operation, which lasted 45 min, crystalloid 2000 mL, colloid 750 mL and three units of blood were given. Oxytocin 20 units was infused over 2 h. At the end of surgery propofol and remifentanyl infusions were stopped. At this stage the pulse was 110 beats/min and blood pressure 120/65 mmHg. She was transferred to the postoperative care unit (PACU) and ECG, SpO₂ and invasive blood pressure monitoring were continued. Analgesia was provided by i.v. fentanyl via a patient controlled analgesia device (20-µg/h infusion, 15-µg bolus with a 30-min lock-out time). The esmolol infusion was decreased and stopped one hour later. At this time her pulse was 100-120 beats/min and blood pressure 110/55-130/70 mmHg. She was observed on the PACU for 12 h. There were no further complications and she was then transferred to the gynecology ward.

The diagnosis of molar pregnancy was confirmed by histological examination. After surgery, serum FT₃, FT₄ and β-hCG values gradually decreased. Serum FT₃ and FT₄ values returned to normal two weeks later and the patient was clinically euthyroid three weeks after surgery; β-hCG values returned to normal two months later.

DISCUSSION

Hydatidiform mole is the most common form of gestational trophoblastic disease, a spectrum of conditions that originate from the placenta and include complete and partial moles, invasive moles, choriocarcinoma and placental site trophoblastic tumors.⁹ The incidence of hydatidiform mole varies from 1 in 1200 to 1 in 2500,^{1,10,11} although it is more common in Asia and the Far East.¹² Twinning, the presence of a complete mole with a viable fetus and placenta occurs rarely. Guidelines published by the Royal College of Obstetricians and Gynaecologists state that in such circumstances the pregnancy should be allowed to continue as there is no increased risk of developing persistent

Table 1. Serum FT₃, FT₄, TSH and β-hCG values

	Normal range	On first admission	After plasmapheresis	5 days after surgery	10 days after surgery	3 weeks after surgery	2 months after surgery
FT ₃ (pg/mL)	1.8-4.6	15.92	8.95	2.11	1.54	1.72	1.75
FT ₄ (ng/dL)	0.9-1.7	5.71	4.12	0.75	0.56	0.90	0.88
TSH (µU/mL)	0.27-4.2	0.01	0.01	0.03	0.10	0.18	0.25
β-hCG (mU/mL)	0-5.3	650 000	370 000	4839	587	6.5	2.5

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