

CASE REPORT

Post-evacuation cardiopulmonary distress in a case of molar pregnancy

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ABSTRACT

Molar pregnancy is a gestational trophoblastic disease wherein the swollen chorionic villi grow in a way that resembles a cluster of grapes. Patients with molar pregnancies very often need anesthesia for suction evacuation. Though molar pregnancy is usually uncomplicated, few cases may be associated with perioperative complications. Various anesthetic techniques have been used for molar evacuation. We present here a case of molar pregnancy with hyperthyroidism, in which general anesthesia (GA) was administered to the patient with laryngeal mask airway. Six hours after evacuation, she went into acute cardiopulmonary distress. She was fortunate enough to be picked up early and was managed successfully.

Key words: General anesthesia; Laryngeal mask airway; Molar pregnancy, evacuation; Acute respiratory distress syndrome; ARDS; Cardiopulmonary distress

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INTRODUCTION

Hydatidiform mole is a gestational trophoblastic disease that originates from the placenta. It is a rare but life threatening disease.¹ Molar pregnancy is common in Oriental countries, Africa, Central and Latin America. The incidence in India is one in 400.² Prevalence of hyperthyroidism during complete molar pregnancy is as high as 7%.³ Complete evacuation of the uterus needs to be done as soon as the diagnosis is confirmed.⁴ 20% of cases of hydatidiform mole are associated with critical perioperative complications, e.g. cardiopulmonary distress.² This can lead to substantial morbidity and mortality.⁴ We report here a case of hydatidiform mole with hyperthyroidism that developed acute cardiopulmonary distress after suction evacuation under GA in our institute.

CASE REPORT

A 22 years old multigravida was admitted with a history of 12 weeks of amenorrhea and vaginal bleeding. Her uterine size on abdominal examination was larger than expected. Her investigations showed a hemoglobin of 9.8 g/dl, beta-HCG >750,000 IU/L and thyroid functions

markedly deranged (T3 = 18,000 ng/ml, T4=73 ng/ml, TSH <0.01 mU/L). Her ultrasonographic reports showed signs of a complete molar pregnancy. She was nil by mouth since 12 hours. She had a heart rate of 100 beats per min, blood pressure of 120/80 mmHg, a normal airway, was afebrile and was taken up for emergency suction evacuation due to increased vaginal bleeding.

On the operating table her heart rate was 150 beats/min and blood pressure was 180/100 mmHg. She was tachypneic with a respiratory rate of 30 breaths/min and a normal body temperature. On auscultation, her lung fields were clear. Oxygen saturation (SpO₂) on room air was 98%.

She was intravenously premedicated with inj. ondansetron 4 mg, midazolam 1 mg, fentanyl 100 µg and inj. metoprolol 2 mg. Anesthesia was induced with injection propofol 2.5 mg/kg. A classic laryngeal mask airway (LMA) No. 3 was inserted. Anesthesia was maintained with oxygen and nitrous oxide (3:5) and propofol infusion (50 µg/kg/hour) on spontaneous ventilation. Inj. oxytocin 40 units infusion at 30 drops/minute was started at the beginning of the suction evacuation. Intraoperatively, heart rate, blood pressure, SpO₂

and end tidal CO₂ were monitored. . The procedure lasted for approximately 20 min. Blood loss was approximately 500 ml. Whole blood 350 ml was transfused over the next hour. 500 ml of ringers lactate was infused intraoperatively. At the end of surgery, the LMA was removed . At the time of shifting to the postoperative ward she had a heart rate of 140 bpm and BP 160/100 mmHg. She was afebrile with clear lung fields.

Six hours after surgery she gradually became breathless and tachypneic. Her SpO₂ fell below 90%. On auscultation, bilateral crepitations were present over the lung fields, more marked at the bases. Patient was shifted to the intensive care unit. Oxygen was administered via face mask. The electrocardiogram showed a sinus tachycardia at 150 beats/min. Two dimensional echocardiography showed mild pulmonary artery hypertension with a left ventricular ejection fraction of 56%. Other parameters were normal. Chest x-ray revealed bilateral fluffy infiltrates. Pulmonologist opinion was sought. Pulmonary edema with features of acute respiratory distress syndrome was suspected. Central venous line was inserted and central venous pressure was measured to be 10 cmH₂O. The patient was given inj. furosemide 40 mg 12 hourly and tab metoprolol 25 mg 12 hourly in view of her tachycardia. Intravenous antibiotics and nebulization were also started.

The patient was comfortable the next day. Her SpO₂ improved, tachypnea settled down, heart rate and blood pressure came down to 100 beats/min and 120/70 mmHg respectively. The chest was clear. Thyroid function and beta HCG levels were repeated after a week and both values had decreased. She was discharged on the seventh postoperative day.

DISCUSSION

Trophoblastic hyperthyroidism has a widely divergent clinical picture from no symptoms to a thyroid storm.⁵ Nevertheless, our patient had severe biochemical hyperthyroidism with few clinical manifestations like tachycardia and a rise in blood pressure.

27% of cases of trophoblastic hyperthyroidism can develop severe perioperative complications like acute pulmonary distress.⁵ The proven etiology in more than half of the cases of acute cardiopulmonary distress observed after molar evacuation is trophoblastic embolisation.⁶ The other causes could be transfusion related acute lung injury (TRALI), inadvertent fluid overload occurring during anesthesia or drug induced pulmonary edema.^{2,6,7} Although the diagnosis of

TRALI is essentially clinical by exclusion of other likely possibilities, it can be confirmed by testing anti-leukocyte antibodies in donor blood.⁸ This test was not available to us and she developed features after 6 hrs of transfusion. Drug induced non-cardiogenic pulmonary edema may be due to pulmonary venoconstriction, capillary leak syndrome, intravascular fluid overload and reduced oncotic pressure.⁷ However, such drugs were not administered to our patient .The cause of pulmonary distress in our case could thus have been trophoblastic embolisation or pulmonary edema secondary to thyrotoxicosis.

Several cases of acute pulmonary edema have been observed post operatively within 24 hrs after evacuation.^{2,4} Many authors have reported successful management of these complications.^{1,3} However, some authors have also reported unsuccessful management and subsequent mortality.^{2,4} Our case presented with pulmonary edema within 24 hours of evacuation of the molar pregnancy and the management led to a successful outcome. It would have been better if the patient was optimized preoperatively with antithyroid drugs, as mentioned by several authors.^{2,3,5}

The anesthetic management of molar pregnancy needs to be standardized.⁹ Sedation, monitored anesthesia care, spinal anesthesia and GA have been used for evacuation of molar pregnancy.^{1-4,6} Total intravenous anesthesia (TIVA), GA with sevoflurane maintenance and GA with endotracheal intubation and non-depolarising muscle relaxant have all been described.^{1,2} Some anesthesia providers prefer to use LMA for administering GA for extra-abdominal procedures upto the first 12-14 weeks of pregnancy, provided the patient is empty stomach.¹⁰ In a review of the anesthetic management of 181 clinical cases of molar pregnancy done in three major academic hospitals of South Africa, GA was administered to 92.3% cases out of which 64.6% had supraglottic airways placed and the associated complications were substantial.⁹ We used the technique of GA with spontaneous ventilation, LMA and propofol for our case.

CONCLUSION

Postoperative cardiopulmonary complications can occur in trophoblastic hyperthyroidism and the anesthesiologists need to be careful while anesthetising these cases. Perioperative optimization with antithyroid drugs and a planned anesthetic management can lead to favorable postoperative outcomes.

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