Critical care management of molar pregnancy in a peripheral set-up

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INTRODUCTION

Abnormalities of chorionic villi with trophoblastic proliferation and villous stromal oedema are the characteristics of molar pregnancy. Moles usually occupy the uterine cavity, but ovary and oviduct can be an ectopic site.¹ Molar pregnancy can be complete or partial depending on the presence or absence of embryonic elements, respectively. The patient may present with vaginal bleeding, increased uterine size, hyperemesis gravidarum, pre-eclampsia, hyperthyroidism, or theca lutein ovarian cysts. Trophoblastic embolisation with respiratory distress (RD) can occur rarely in a patient with complete mole. We present a case of molar pregnancy with cardiopulmonary complication post-evacuation.

CASE REPORT

A 22-year-old primigravida was reported to the outpatient department of obstetric and gynaecology at 13 weeks' period of gestation with history of mild vaginal bleeding. She had no other complaints to offer. General and systemic examination was within normal limits except that mild pallor was present. The uterus was smooth and of 16 weeks' size. On per speculum examination there was no bleeding and os was closed.

The patient had Hb of 9 g% and the serum beta-hCG level was 114,000 mIU/mL. All other haematological and biochemical parameters were within normal limits. Radiographs of chest (PA view) was normal (Figure 1). Ultrasonography revealed uterus filled with heterogeneous mass with multiple cystic components, small amount of fluid, and "snow storm appearance".

She underwent suction and evacuation under general anaesthesia. Injection pitocin drip was continued post-evacuation

for four hours. Anaesthesia technique used was general anaesthesia with oral endotracheal intubation with intermittent positive pressure ventilation (IPPV). Total anaesthesia time was 30 minutes. A dose of 1,000 mL of Ringer lactate was given intra-operatively. The patient had unexplained tachycardia towards the end of the procedure (HR: 160/minute, regular, sinus rhythm) which was managed with injection esmolol 100 mg i.v. in divided doses. The patient had an uneventful and complete recovery. In post-anaesthesia care unit, the patient developed tachypnoea (respiratory rate of 40/minute) and oxygen saturation of 94–95% (room air) on pulse oximetry. The patient was shifted to intensive care unit (ICU) for observation and was advised for radiograph of chest (PA view) and ECG.

In ICU, the patient complained of dry cough. On examination, the patient did not appear in distress. Her limbs were warm, she was afebrile, had tachycardia (pulse: 120/minute, regular, bounding), tachypnoea (respiratory rate: 46/minute), hypotension (BP: 80 mmHg systolic) and hypoxia (oxygen saturation was 70% in room air). Further, TLC was found to be 14,200/mm³ (which increased to 20,800/mm³ on the third postoperative day). Chest auscultation revealed crackles in left infrascapular, interscapular, and axillary regions. In view of the above clinical findings along with chest radiograph (PA view) showing bilateral pulmonary infiltrates (left>right) (Figure 2) and ECG showing sinus tachycardia, T wave inversion in leads I, aVL, V₁ to V₄, ST depression in V₁–V₃, a provisional diagnosis of: (i) systemic inflammatory response syndrome (SIRS) with shock;

Figure 1 Pre-operative chest radiograph (PA view).

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Figure 2 Postoperative chest radiograph on the same day evening.

Figure 3 Postoperative chest radiograph on day one on ventilator.

and (ii) acute pulmonary insufficiency/acute RD syndrome with differential diagnosis of: (a) pulmonary embolism of trophoblastic cells, and (b) sepsis was made.

The patient was placed on mechanical ventilator (Hamilton Galileo, Hamilton Medical AG, Switzerland) after endotracheal intubation. The initial mode used was synchronised intermittent mandatory ventilation (SIMV) with pressure support (PS), FiO_2 of 1.0, tidal volume: 400 mL, rate: 14/minute, peak end expiratory pressure (PEEP): 8 cm H_2O , and PS above PEEP: 10 cm H_2O . Right internal jugular vein was cannulated with double lumen central venous catheter. Initial CVP was 17 cm H_2O . Dopamine infusion was started at the rate of 5 $\mu\text{g}/\text{Kg}/\text{minute}$. With this, initially the BP improved to 100/80 mmHg. She was managed with antibiotics, injection low molecular weight heparin subcutaneously (in view of suspected embolism of trophoblastic cells), injection furosemide after stabilisation of BP, and supportive care.²

Since the patient was not maintaining oxygen saturation despite high PEEP and FiO_2 of 1.0, ventilator mode was changed to airway pressure release ventilation (APRV) with inverse ratio ventilation (IRV).³ With this the oxygen saturation improved to 98% over the next two days and FiO_2 was reduced accordingly (Figure 3).

On the first postoperative day onwards, the patient developed hypotension again which was not responding to dopamine infusion at the rate of >10 $\mu\text{g}/\text{Kg}/\text{minute}$. Consequently, noradrenaline infusion was added at the rate of 0.1 $\mu\text{g}/\text{Kg}/\text{minute}$ and slowly increased to 0.5 $\mu\text{g}/\text{Kg}/\text{minute}$. When despite CVP of 8 cm H_2O and maximum infusion rates of dopamine and noradrenaline, mean arterial pressure was <65 mmHg, vasopressin infusion was added at 0.04 units/minute.

Blood pressure started stabilising from the fourth postoperative day and all vasopressor and inotropic support was tapered and ventilatory support was weaned off on the sixth postoperative day. All drugs were stopped on the 10th postoperative day. The patient was discharged from the hospital on the 12th postoperative day. Serum beta-hCG gradually returned back to normal by 13 weeks of post-evacuation. She was prescribed with low dose oral contraceptive pills for contraception during follow-up.

DISCUSSION

Peak levels of beta-hCG reach 100,000 mIU/mL between the 60th and 80th day after the last menses. Beginning at about 10–12 weeks' gestation, maternal plasma levels of beta-hCG begin to decline, and a nadir reached by about 20 weeks and maintained at this level for the rest of the pregnancy.

Suction and evacuation is the preferred method of evacuation of a molar pregnancy, regardless of uterine size, for patients who desire to preserve fertility. At the time of molar evacuation trophoblastic cells in variable amounts escape from the uterus into the venous outflow.⁴ Generally, RD distress has been known to develop in two percent of patients with a complete mole in the past, but currently it occurs rarely.⁵ Embolisation with a large amount of trophoblastic cells is probably uncommon, sudden death from trophoblastic embolism in pregnancy have been described.⁶ Usually, RD is diagnosed in a patient with excessive uterine size and markedly elevated beta-hCG levels. The uterine size of our patient was larger than the period of gestation and beta-hCG level was 114,000 mIU/mL.

Patients may have chest pain, dyspnoea, tachypnoea, tachycardia and may have severe RD during and after molar evacuation. Chest radiograph may show bilateral pulmonary infiltrates. Usually, RD resolves within 72 hours with cardiopulmonary support, some patients may require mechanical ventilation. Differential diagnosis for respiratory insufficiency includes complications of thyroid storm, pre-eclampsia and massive fluid replacement.

Primarily, SIRS is defined as two or more of the following variables:

1. Fever (temperature >38°C) or hypothermia (temperature <36°C).
2. Tachycardia (heart rate >90/minute).
3. Tachypnoea (respiratory rate >24/minute).
4. Leucocytosis (>12,000/ mm^3) or leucopenia (<4,000/ mm^3) or >10% bands.

Sepsis is defined as SIRS with proven or suspected microbial aetiology. Refractory shock is a shock lasting for more than a hour and does not respond to fluid/pressor administration.⁷

Inflammatory response is an extremely complicated process triggered by a physical, chemical or infectious insult to the host. The hallmark of shock in such cases is decrease in peripheral vascular resistance despite increased levels of catecholamines. Before the vasodilatory phase there may be a period during which oxygen delivery to tissues is compromised by myocardial depression, hypovolaemia, and other factors. Ventilation-perfusion mismatch produces fall in arterial PO₂ early in the course. Increasing alveolar capillary permeability results in increased pulmonary water content, which decreases pulmonary compliance and interferes with oxygen exchange. Progressive pulmonary infiltrates and arterial hypoxemia (PaO₂/FiO₂<200) with pulmonary capillary wedge pressure <18 mmHg indicate the development of ARDS.⁷

CONFLICTS OF INTEREST

None identified.

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