CASE REPORT

ACUTE PULMONARY EDEMA AFTER EVACUATION OF MOLAR PREGNANCY

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ABSTRACT

Cardiopulmonary dysfunction has been observed after the removal of benign hydatidiform mole which can lead to substantial morbidity and mortality. We report a case of 30 year old woman who came to casualty with a complete vesicular mole. Evacuation of vesicular mole was done under spinal anesthesia as an emergency procedure. Immediately after evacuation she developed acute massive pulmonary edema with extensive crepitations over both lung fields. She was successfully managed with extensive perioperative management in the form of continuous monitoring, broad spectrum antibiotics, oxygen inhalation, diuretics and she did well postoperatively. She was discharged on 9th post operative day in satisfactory and stable condition.

Key Words: Cardiopulmonary distress; Hydatidiform mole; Adult respiratory distress syndrome; Molar pregnancy.

INTRODUCTION

Molar pregnancy occurs in 1 in 1945 pregnancies worldwide1 and more commonly in Asian countries2. The incidence in India is one in 400 pregnancies3. Of the molar pregnancies 80% are uncomplicated and follow an unremarkable course. However, 20% are associated with severe perioperative complications that may lead to morbidity and mortality in otherwise healthy women. Anemia, hyperthyroidism and acute cardiopulmonary distress are significant complications of complete molar pregnancy4, 5. Acute cardiopulmonary distress and adult respiratory distress syndrome (ARDS) have been reported after evacuation of mole in 27% of cases6 and some fatalities have been described5, 6. The aim of this case report is to highlight critical nature of one of the complications associated with a molar pregnancy and how effectively it was managed with medical and anesthetic interventions.

CASE REPORT

Patient was a 30 years old Gravida2, Para1, Abortion0, Live1 with history of 6 months of amenorrhea. She came to medical casualty of our hospital with a complaint of vaginal bleeding and severe abdominal pain. She belonged to a rural background, with poor socio-economic status and had no antenatal visits. Her past history was unremarkable. At the time of admission on examination she was conscious, oriented, afebrile, pale and had mild pedal edema. Her pulse rate was 124 per minute, blood pressure was 140/80 mm of Hg, respiratory rate was 16 per minute. ECG on the monitor was normal. Chest auscultation revealed normal heart sounds and no added sounds in the lung fields. On examination per abdomen uterus was of 32 weeks size with no fetal parts felt. On examination per vaginal bleeding was present with passage of big clots and grape like structure. Two IV canulae of 18 G were passed and samples for blood grouping and cross matching, complete blood count, renal function test, liver function test, bleeding time, clotting time, serum beta-hcg were sent to the laboratory. Her sonography revealed complete molar pregnancy with huge bulky uterus with its superior border extending up to supraumbilical region measuring 32.5*20.7* 18.4 cms with whole of uterus is filled and replaced by multiple cystic areas of varying sized with interspaced thick hypechoic septigiving snoestorm appearance with no fetal part seen. On investigation her beta hcg level was 3,20,040 miu/ml, hemoglobin was 07 gm/dl, blood group was B Rhesus positive, and total count, differential count, renal function test, liver function test, coagulation profile were within normal limits. High risk consent was obtained from the patient and the attendants for emergency evacuation of the mole under anesthesia. Two units of Red cell concentrate, 1000 ml of Ringer’s lactate and 500 ml of hydroxyethyl starch 6% were infused peroperatively. After pre-medication induction was done with spinal anesthesia under strict aseptic and antiseptic precautions, in sitting position in L3-L4 space giving injection lignocaine with adrenaline 1.5 ml. Infusion of 20 units of oxytocin in 500 ml of Ringer Lactate solution was started during the evacuation procedure. Evacuation was completed in 15 minutes. Intraoperative blood loss was limited to 1000 ml. Immediately after evacuation she became drowsy and developed tachypnoe with her pulse rate 150/min, blood pressure was 140/80 mm of Hg and SpO2 was 74%. On examination bilateral extensive crepitations were present. Resuscitation with 100%
oxygen by bains circuit, injection deriphylline, dexona and frusemide were done. As per the advice of physician patient was shifted in critical care unit for continuous monitoring. She was placed in propped up position. Oxygen inhalation(8-10 liters/minute), injection frusemide(40mg intravenous every 15 minutes up to total of 1000 mg in 5 hours) were given and her oxygen saturation returned to normal. She was given broad spectrum antibiotics, injection frusemide intravenously for 5 days. The condition was diagnosed as noncardiogenic pulmonary edema. Results of investigations revealed a hemoglobin concentration of 8.9gm/dl; blood counts, coagulation profile, renal and liver function tests were within normal limits, D-dimer was 0.5 mg/l. Her 2D-echo and sonogram of abdomen-pelvis were normal. She had 4 units of Red cell concentrate, of which 2 units preoperatively and 2 units postoperatively. She was shifted to general ward on 6th postoperative day. Her beta-hcg level was 14,100 miu/ml after 1 week of evacuation. Her beta hcg level returned to normal limit within 9 weeks. She was discharged on 9th postoperative day in stable condition. She was followed up for a period of 6 months.

DISCUSSION

Molar pregnancies develop as a result of abnormal fertilization and are categorized as complete or partial. In a complete molar pregnancy the placenta becomes edematous secondary to grossly enlarged hydropic degeneration of the chorionic villi and the fetus fails to develop. Cord and amniotic membranes are absent. Patients of molar pregnancy have high serum levels of Human Chorionic Gonadotropin (HCG) and a larger than expected uterus size for the gestational age. The obstetric management requires therapeutic termination of the pregnancy involving complete evacuation of the uterine contents. The critical nature of the associated complications requires advanced perioperative anesthetic management. A large number of the complications is associated with molar pregnancy, which includes acute cardiopulmonary distress, hyperthyroidism with thyroid storm, severe anemia, hemorrhage, trophoblastic embolism, DIC and pregnancy induced hypertension etc. Acute pulmonary edema was seen in the present case which was managed aggressively and patient did well postoperatively. Acute cardiopulmonary distress has been observed after evacuation of molar pregnancy in 27% of the cases and more so in patients with uterine size of 16 weeks or greater. Symptoms usually develop within 4-12 hours after evacuation of the uterus and are marked with cough, tachycardia, tachypnoea, hypoxemia, diffuse rales and bilateral pulmonary infiltrates on a chest radiograph. Variable amounts of trophoblastic cells enter the venous circulation and this embolisation is responsible for the cardiopulmonary crisis in more than 50% of cases. Symptoms may vary in severity, with some requiring mechanical ventilation, vasopressor support and in the most severe cases, massive trophoblastic embolism may lead to death. Hyperthyroidism in these patients is thought to occur as a manifestation of excessive levels of circulating HCG or from a thyrotropin like substance released from the mole. The resultant thyroid storm may lead to high output cardiac failure. Elevated levels of these might have been contributory in the development of pulmonary edema. Anemia in these patients is secondary to chronic occult bleeding from multiple hemorrhagic areas throughout the placenta and massive blood loss during surgery, as was seen in our case. Transfusion related acute lung injury, although rare, is another possible cause of cardiopulmonary distress, which manifests within 6 hours of transfusion and is marked by noncardiogenic edema, but this possibility is ruled out in the present case as ARDS developed in this patient prior to institution of blood transfusion. During surgery only 1000 ml of Ringer’s lactate and 500 ml of hydroxyethyl starch was given. This excludes the contributory role of fluid overload in the development of pulmonary edema. It may be stressed that the communication between the anesthesiologist and the obstetrician should begin soon after the diagnosis of molar pregnancy has been established to provide adequate time for a comprehensive preoperative workup and baseline laboratory tests including coagulation studies, electrolytes, CBC with platelets, thyroid function tests and baseline chest radiograph. ABG analysis is recommended to demonstrate signs and symptoms of pulmonary edema preoperatively. Because of the potential blood loss, adequate intravenous access and immediate availability of blood products should be established before induction. A review of literature about the anesthetic techniques for evacuation of hydatidiform mole suggests general anesthesia with endotracheal intubation as the technique of choice because of the potential for development of acute intraoperative hemorrhage and possible need of ventilatory support if acute cardiopulmonary distress develops. Use of an oxytocin infusion before or during uterine evacuation is suggested as a mechanism for avoiding trophoblastic embolism and to control hemorrhage. On the contrary some authors counter this and are of the view that if used before evacuation, it may increase the risk of acute pulmonary insufficiency.

CONCLUSION

We conclude that evacuation of molar pregnancy can be associated with acute cardiopulmonary distress in the form pulmonary edema (ARDS) and required to be managed aggressively with blood replacement, diuretics and ventilatory support. Thus a detailed preanesthesia workup, preoperative optimization, careful anesthetic management and liberal arrangement of blood transfusion products are necessary for any case of molar pregnancy to maximize the outcome.

REFERENCES


Volume 4 | Issue 1 | Jan – Mar 2014

Page 102


