Acute Pulmonary Edema After Large Molar Pregnancy Uterine Evacuation

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Abstract- Cardiopulmonary complications have been observed after the evacuation of benign hydatidiform mole which can lead to substantial morbidity and mortality. We report a 30-years-old woman who came to our outpatient department of obstetrics and gynecology at 9 weeks gestational age with sonography which represented molar pregnancy. Evacuation of the mole was done under spinal anesthesia as an elective procedure. The patient had a complaint of dyspnea, 6 hours after evacuation. CXR showed some reticular opacity in the field of both lungs with increased Broncho vascular marking. CT angiography showed pulmonary edema with subsegmental atelectasis in the base of both lungs without any pattern of pulmonary thromboembolism. Pulmonary edema was regressed by administering frusemide and conservative management after 8 days of ICU admission. She was discharged on the 10th postoperative day in satisfactory and stable condition.

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Keywords: Molar pregnancy; Gestational trophoblastic disease; Pulmonary edema; Trophoblastic embolism; Cardiopulmonary symptoms; Molar evacuation; Uterine suction

Introduction

Hydatidiform mole is a disease characterized by hydatidiform hydrophilic villi and atypical hyperplastic trophoblasts which seen in women of reproductive age. There are some complications associated with molar pregnancy and its evacuation. Rising b-HCG following the development of molar pregnancy leads to thyrotoxicosis, pulmonary edema and acute respiratory insufficiency. Life-threatening acute pulmonary edema and coronary insufficiency are the most serious complications of thyrotoxicosis (1). Cardiopulmonary symptoms are rare in women treated in the first trimester (2,3). After uterine evacuation for a complete hydatidiform mole in the second trimester, approximately 2 percent of patients used to develop cardiopulmonary symptoms, including chest pain, dyspnea, tachypnea, and tachycardia (2). Respiratory distress is usually attributed to trophoblastic embolization, but can also be due to cardiopulmonary complications of thyroid storm, toxemia, and massive fluid replacement. We described a patient with molar pregnancy who had cardiopulmonary complications after uterine evacuation.

Case Report

A 30-year-old woman, gravida 3 para 1 abortion 1, came to our outpatient department of obstetrics and gynecology at 9 weeks gestational age with sonography which represented molar pregnancy and lab data, which showed BHCG over 450,000 mIU/ml. she was scheduled for elective evacuation of pregnancy and other lab data such as CBC, Liver function tests, Renal function tests and TSH was requested. All of them were normal, Hb was 10.1 g/dl and TSH was 0.4 mIU/ml. Also, CXR was done and reported as normal.

On the day of surgery, she came to the emergency room with a complaint of mild vaginal bleeding. At the initial examination, the patient's general conditions were satisfactory, cardiac and respiratory auscultation was normal, pulse rate was 108 beats/min, respiratory rate was 16/min and body temperature was 37°. On speculum examination, there was a mild bleeding and internal os was closed. Uterine height was 20 weeks size. She was moved to the operation room for suction and evacuation of molar pregnancy. Before starting the anesthesia and surgery, 20 mg propranolol tablet was given orally to the patient because of tachycardia in the range of 100-110 beats/min. Then spinal anesthesia was done. Infusion of 20 units of oxytocin in 1 liter of ringer was started and

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evacuation by ultrasonography-guided suctioning and curettage was completed in 15 minutes. Vesicular tissue in great amount was evacuated and emptiness of the cavity was confirmed by sonography in the operation room. She was moved to the recovery unit and 500 cc Ringer additionally infused for her. Overall, 1.5 liter Ringer solution was infused during and after the surgery. At the recovery unit, her blood pressure was 145/95, PR was 110/min, RR was 17/min and O_2 sat in room air was 98%. She was moved to the ICU unit for controlling her vital signs. The patient had a complaint of dyspnea, 6hours after evacuation. At this time, her BP was 160/100 mmHg, PR was 118/min, RR was 28/min and O_2 sat in room air was 93%. The temperature was 37°. Heart and lungs auscultation was normal. On the examination, abdomen was soft without distention. The uterus was of 12 weeks size and vaginal bleeding was about spotting. According to the signs and symptoms and recent evacuation of molar pregnancy, our differential diagnosis of trophoblastic or thromboembolic pulmonary emboli, thyroid storm, preeclampsia, pulmonary edema and heart failure were raised. Consequently, radiography of chest, D-dimer, TSH, free T3 and T4, CBC, ABG, EKG and echocardiography were done. Unfortunately, we hadn't CT angiography in our hospital. So, CXR showed some reticular opacity in the field of both lungs with increased Broncho vascular markings (Figure 1). ABG showed PH=7.34, PCO_2=23.6, HCO3=13 and Beb=-10.8. Hb was 9.5 g/dl and WBC was 8800 (pmn 77%). ECG showed sinus tachycardia pattern. D-dimer was negative. Echocardiography revealed SPAP=26 mmHg and EF=55% and Normal valves function. With our cardiologist consults, we administered 20 mg labetalol stat and continue with 1 mg/min drip to control the patient's blood pressure and pulse rate. Additionally, UFH (unfractionated heparin) was started in therapeutic doses and 20 mg furosemide was injected. Pulse rate was 120/min, RR was 30/min and O2sat was 92% with an Oxygen mask. MgSo4 was started according to the probability of preeclampsia. After 3 hours and continuation of signs and symptoms other lab data were reported; free T3 and free T4 were in normal ranges and TSH was 0.25. At this time, we could move the patient to another hospital for lung CT angiography. CT angiography was done and it showed pulmonary edema with subsegmental atelectasis in the base of both lungs without any pattern of pulmonary thromboembolism (figure 2-3). According to the CT angiography and other Para clinical evaluations, the condition was diagnosed as no cardiogenic pulmonary edema in the field of trophoblastic emboli after the evacuation of molar pregnancy. Heparin and MgSo4 were discontinued. Blood pressure was controlled with labetalol and nifedipine. Pulmonary edema was regressed by administering furosemide and conservative management after 8 days of ICU admission. Her vital signs were completely normal. BHCG titer was 12520 mIU/ml and pathology reported incomplete molar pregnancy. Finally, she was discharged from hospital with weekly BHCG titer monitoring on the 10th day of surgery.

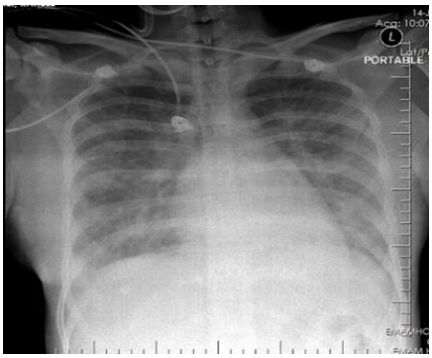

Figure 1. CXR

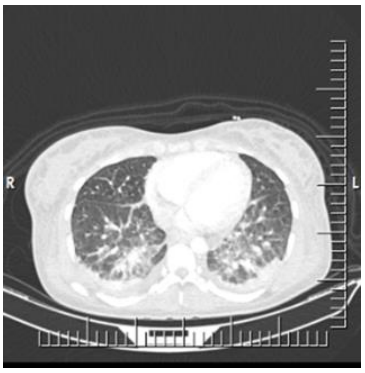

Figure 2. Lung CT angiography

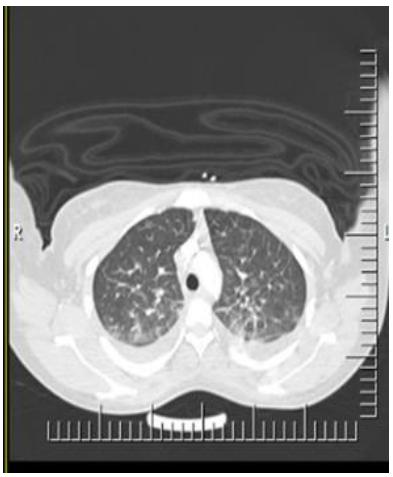

Figure 3. Lung CT angiography

Discussion

Hydatidiform mole is part of a group of diseases classified as gestational trophoblastic disease (GTD), which originates in the placenta and have the potential to locally invade the uterus and metastasize. The pathogenesis of GTD is unique because the maternal tumor arises from gestational rather than maternal tissue (4). 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 gestational age (5). The obstetric management requires therapeutic termination of the pregnancy involving the complete evacuation of the uterine contents (6). Suction curettage is the preferred technique for uterine evacuation, regardless of uterine size (7). During the suction evacuation, we do not use prostaglandins for cervical ripening. Starting at the time of anesthesia induction, we do administer an oxytocin infusion (10 units in 1 L Ringer lactate solution at 50 drops/min) to increase myometrial tone and facilitate contraction, and thus decrease blood loss (8) and avoid trophoblastic embolization (9).

A large number of complications are associated with a molar pregnancy, which includes acute cardiopulmonary distress, hyperthyroidism with thyroid storm, severe anemia, hemorrhage, trophoblastic embolization, DIC and pregnancy-induced hypertension (9-10).

In women treated in the first trimester, cardiopulmonary symptoms are rare (2-3). After uterine evacuation for the complete hydatidiform mole in the second trimester, some patients used to develop cardiopulmonary symptoms, including chest pain, dyspnea, tachypnea, and tachycardia. Auscultation of the chest usually reveals diffuse rales, and the chest radiograph often demonstrates bilateral pulmonary infiltrates (2). Respiratory distress is usually attributed to trophoblastic embolization, but can also be due to cardiopulmonary complications of thyroid storm, toxemia, and massive fluid replacement (11). Acute cardiopulmonary distress has been observed after the 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, tachypnea, hypoxemia, diffuse rales and bilateral pulmonary infiltrates on a chest radiograph (10-12). Variable amounts of trophoblastic cells enter the venous circulation and this embolization is responsible for the cardiopulmonary crisis in more than 50% of cases (13). Embolization with a large number of trophoblastic cells is probably uncommon, sudden death from a trophoblastic embolism in pregnancy have been described (14).

In our patient, the uterine size was about 20 weeks of gestation and the BHCG level was over 450,000 and symptoms occurred 6 hours after evacuation.

The signs and symptoms usually resolve within 72 hours after evacuation with cardiopulmonary support (11). 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 (9,13,15). 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 (15,16,17). Preeclampsia, associated with complete molar pregnancy resolves promptly after the molar evacuation and usually does not require medical management (2,3). The presence of pulmonary infiltrations can sometimes be misinterpreted as metastases, for which chemotherapy is inappropriately administered. In most cases, the infiltrates will resolve over 48 to 72 hours as the hCG level decreases. The presence of pulmonary nodule(s) after molar evacuation in the face of falling hCG levels does not require chemotherapy. Trophoblastic emboli usually resolve spontaneously as long as the HCG level ultimately normalizes (11).

In our patient, according to these probabilities, we checked free T3 and T4 and TSH level which was normal. During and after surgery, we gave 1.5 liters of ringer and this excluded the volume overload role on the development of pulmonary edema in our normal heart function patient. Our patient was improved by conservative management during the first 48-72 hours and her cardiopulmonary symptoms were firstly described with trophoblastic pulmonary emboli.

A review of literature about the anesthetic techniques for the evacuation of hydatidiform mole suggests general anesthesia with endotracheal intubation as the technique of choice because of its high potential for developing

acute intraoperative hemorrhage and the possible need for ventilation support if acute cardiopulmonary distress develops (9).

We concluded that the evacuation of molar pregnancy especially if the uterine size was over 16weeks of gestation and the BHCG level was markedly high, could be associated with cardiopulmonary complications. That may be fetal. So, the attention to vital signs and careful anesthetic management and preoperative and postoperative optimizations are necessary for any case of molar pregnancies. At the time that cardiopulmonary signs and symptoms are detected. First, we should rule out another important differential diagnosis. Then for trophoblastic embolization supportive care should be done.

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