Acute Pulmonary Edema Following Administration of Magnesium Sulfate in a Pregnant Patient

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ABSTRACT

Acute pulmonary edema affects 0.08% to 1.5% of women during pregnancy and during the postpartum period, and preeclampsia/eclampsia is a major obstetric cause of acute pulmonary edema. We present a case of a 23-year-old nulliparous woman who was referred to tertiary medical center for preterm labor and dyspnea (Mousavi Hospital). The patient complained of having suddenly developed respiratory distress and a decrease in O₂ saturation following the administration of magnesium sulfate. A chest radiograph taken at bedside showed widespread interstitial shadowing consistent with pulmonary edema. The patient was given prompt treatment, and she achieved full recovery. Pharmacological agents are one of the defendants used for lung edema during pregnancy. It is important to pay attention to lung edema due to tocolytic administration.

Keywords: Magnesium sulfate, Pulmonary edema, Tocolytic, Pregnancy

Introduction

The accumulation of fluid in the pulmonary interstitial spaces is the pathophysiology of pulmonary edema. Physiological changes can predispose pregnant women to this complication (1). Being an uncommon event, the risk of pulmonary edema is around 0.08% to 1.5% in women during pregnancy and in the postpartum period (2). Maternal and fetal morbidity and mortality increase in this situation (3). Various etiologies can result in pulmonary edema in pregnant patients, including preeclampsia, tocolytics, sepsis, pre-existing cardiac disease, and pregnancy-associated cardiac disease, fluid overload, and multiple gestations (2,4). The infusion of fluids in women who submitted to induced labor, caesarean sections, and prophylaxis with magnesium sulfate are known causes of pulmonary edema (5). Few cases of acute pulmonary edema resulting from the administration of magnesium sulfate have been reported to date. The diagnosis and treatment of this complication are vital. Here, we describe a case of acute pulmonary edema that was likely caused by the administration of magnesium in a pregnant woman.

Case Report

A nulliparous 23-year-old woman was referred to tertiary medical center for preterm labor and dyspnea (Mousavi Hospital). She had no past medical history. The patient complained of having suddenly developed respiratory distress and a decrease in O₂ saturation following administration of magnesium sulfate. She was transferred to our third level intensive care unit (ICU).

On arrival to ICU, her blood pressure was 120/75 along with a respiratory rate of 50 breath per minute and SpO₂ 96%; hence, 20 mg of furosemide was injected and the patient was intubated because of the respiratory distress. All necessary evaluations were performed for the patient including chest radiography (Figure 1), CT angiography (Figure 2), echocardiography, a complete workup for autoimmune disease, and sepsis. A chest radiograph taken at bedside revealed widespread interstitial shadowing consistent with pulmonary edema (Figure 1). There was no evidence of the thromboembolic event in the CT angiography report. On echocardiography, the cardiac function was normal, and there was no sign of valvular
heart disease or wall motion abnormality. Laboratory findings were as follows:
WBC = 14.4  Neut = 89.1%. Alp = 217 IU/L

Figure 1. Chest x-ray
Bedside chest radiographs shows evidence of both interstitial and alveolar edema. Alveolar edema manifests as nodular opacities.

Figure 2. CT Pulmonary Angiogram
Ground-glass opacity and centrilobular nodules are observed in the CT angiogram.
Anti ds DNA=0.2 IU/ML
Hb=11.4 g/dl  Lymph=6.4  Bil total=0.7 mg/dl
FANA= negative
HCT=34.7%  ALT=20 U/L
    Bil direct=0.5 mg/dl
Plt=167/microlier
AST=22 U/L
Procalcitonin <0.2 ng/mL

ABG, before intubation, showed moderate respiratory alkalosis (pH=7.48, Paco2=32, HCO3=22 O2 sat=89%)

The medical team concluded to perform a Cesarean section to improve respiratory conditions. After 24 hours of admission, Cesarean section was performed, and the mechanical ventilation of the patient was resumed. Airway resistance under mechanical ventilation was about 10 to 15 cm H2O and static compliance was about 33 cm H2O. We performed an open lung maneuver with airway pressure release ventilation mode (A.P.R.V) for 24 hours. Intravenous furosemide (bolus 40 mg over 2 min) was employed, and repeated after 30 min, and its infusion was continued for 24 hours. A chest X-ray was carried out the following day revealing a normal lung activity. We successfully extubated the patient after 48 hours of Cesarean section. The patient’s condition improved, and she was discharged one week after ICU admission.

Discussion

One of the most common causes of morbidity and mortality during pregnancy is acute pulmonary edema (6,7). In 2011, Cantwell reported that it is the fourth most common form of maternal morbidity (6). There is little information about the natural history of pulmonary edema among pregnant women (1). Fluid redistribution may be caused by sympathetic activation in the stress status which could lead to pulmonary edema (8). Due to the decreased plasma protein osmotic pressure, even small changes in hydrostatic pressure may cause pulmonary edema during pregnancy (9).

Various causes for pulmonary edema can be suggested for this case study, including aspiration, sepsis, cardiac disease, preeclampsia, and pharmacological drugs.

There was no evidence for fluid overload or valvar heart disease in echocardiography. CT Angiography did not detect any evidence for aspiration. According to primary hospital records and our evaluations, there was no evidence of fluid overload or other important causes for pulmonary edema in this patient and pulmonary edema has occurred after intubation, showed moderate aspiration.

Due to the decreased plasma protein osmotic pressure, even small changes in hydrostatic pressure may cause pulmonary edema during pregnancy.

Pharmacological agents are one of the defendants for lung edema during pregnancy. β-adrenergic tocolytic agents, corticosteroids, magnesium sulfate and illicit drugs including cocaine are the most common drugs that cause pulmonary edema during the pregnancy (9).

ARDS is rare (0.5%-5%) but is a feared complication of tocolysis with beta-2-mimetic agents and magnesium sulfate and the lesions of alveolar capillary membrane prominent (10). Magnesium sulfate is one of the most commonly used tocolytics for preterm labor (11). Samol et al. demonstrated that MgSO4 is associated with the development of pulmonary edema (12) and it is one of the morbid complications of this tocolytic. In the case of the present patient, her respiratory complications began at the time of the administration of magnesium sulfate.

The recommended treatment for pulmonary edema is airway management, oxygen administration, the application of open lung maneuver and administration of furosemide.

Fortunately, her symptoms were alleviated after 24 hours by application of open lung maneuver and diuretic administration.

Conclusion

Pharmacological agents are one of the defendants for lung edema during pregnancy. This complication occurred in the present woman with the administration of magnesium sulfate to control her preterm labor. When respiratory symptoms such as hypoxemia, tachypnea, and dyspnea occur after administration of magnesium sulfate, it is important to pay attention to lung edema due to tocolytic administration.

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Conflict of Interest

Authors declared no conflict of interests.

References


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