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Case Study

Amniotic Fluid Embolism Following Cesarean Section - ICU Management

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ABSTRACT

Amniotic fluid embolism (AFE) is exceedingly rare, incompletely understood and most catastrophic complications of pregnancy, the exact pathophysiology of which is still unknown. Passage of amniotic fluid into the maternal circulation was first reported by Meyer in 1926. Symptoms include dyspnea, hypotension, seizures, disseminated intravascular coagulation (DIC), poor fetal prognosis. We report a case of a 31 year old primigravida who underwent a Cesarean Section because of fetal distress following prostin induction of labour at 40 weeks and 3 days of her pregnancy. During the surgery her hemodynamic and respiratory conditions were stable. Six hours post-operatively, she developed dyspnea, which further deteriorated and worsened rapidly. This report highlights the importance of monitoring a patient of post cesarean section and diagnostic facilities needed to suspect amniotic fluid embolism.

Key words: Pregnancy, cesarean section, amniotic fluid embolism, intensive care.

1. INTRODUCTION

The Amniotic fluid embolism (AFE) is exceedingly rare, incompletely understood and most catastrophic complications of pregnancy, the exact pathophysiology of which is still unknown. Passage of amniotic fluid into the maternal circulation was first reported by Meyer in 1926.[1] However, the syndrome was first described by Steiner and Lushbaugh in 1941.[2] Symptoms include dyspnea, hypotension, seizures,

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disseminated intravascular coagulation (DIC), poor fetal prognosis. It is estimated to be the fifth most common cause of maternal mortality in the world. Also there has been discrepancy with respect to the incidence and mortality of amniotic fluid embolism. One likely explanation for this inconsistency is the lack of sensitive and specific diagnostic studies to definitively identify cases of amniotic fluid embolism, leading to both over and underreporting.[3]

2. CASE PRESENTATION

A 31years old, weight 74kg and height 172cm primigravida underwent a Cesarean Section because of foetal distress following prostin induction of labour at 40 weeks and 3 days of her pregnancy. Her pregnancy course was uneventful. During the surgery her hemodynamic and respiratory conditions were stable. Six hours post-operatively, she developed dyspnea, which further deteriorated and worsened rapidly. SpO_2 level decreased by 82% on oxygen at 12litres per minute via polymask. She was soon shifted to Intensive Care Unit with suspection of amniotic fluid embolism. HR-124 per minute, BP- 108/70mmHg. ECG - sinus tachycardia. Arterial blood gas analysis showed the following values: pH 7.16; PaO_2 126 mmHg; $PaCO_2$ 58 mmHg; HCO_3^- 18.0 mmol/L. CXR-PA View - Diffuse infiltration was observed throughout the lungs. (Figure 1).

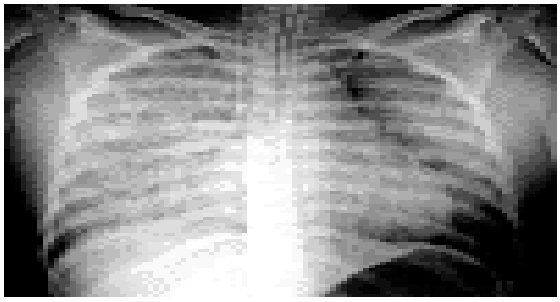

Fig 1: CXR-PAV

Chest CT- showed diffuse homogeneous ground-glass opacity in the bilateral peripheral fields (Figure 2).

2D-ECHO was normal with EF=60%. Patient was intubated and put on mechanical ventilator on CMV mode (RR/TV/PEEP/FiO_2-20/400/05/60%) with sedation and paralyzation. Brain natriuretic peptide (BNP) was high (2,032 pg/ml). Repeated 2D-ECHO showed fall in EF=40%. Subsequently, she developed

Fig 2: CT Chest

respiratory and renal failure, requiring mechanical ventilation and haemodialysis.

Within the span of six days patient's overall condition improved a lot, ventilatory support was minimized and she was extubated successfully. In ICU patient was shown sign and symptoms of depression may be because of the stress she undergone so referred to psychiatrist and treated successfully via oral medication and councelling. Patient was discharged from the intensive care unit on the ninth day.

3. DISCUSSION

Amniotic fluid embolism (AFE) is exceedingly rare, incompletely understood and most catastrophic complications of pregnancy, the exact pathophysiology of which is still unknown. Passage of amniotic fluid into the maternal circulation was first reported by Meyer in 1926. In 1941, the syndrome was first described by Steiner and Lushbaugh, they reported the presence of mucin, amorphous eosinophilic material and squamous cells in women with AFE, consistent with the presence of amniotic fluid. They hypothesized that amniotic fluid was forced into the maternal circulation during contractions leading to the embolic event. Clark *et al.* recognized that the clinical course and hemodynamic changes of AFE were similar to patients with anaphylactic shock and proposed that AFE was more of an immunologic than embolic phenomena. [5] Amniotic fluid contains many vasoactive and procoagulant substances, such as platelet activating

factor, cytokines, bradykinin, thromboxane, leukotrienes and arachidonic acid, and entrance of even minute amounts of these substances into the maternal circulation could cause the syndrome.[9] This would explain why fetal cells were not always found in women who suffered AFE. Symptoms include dyspnea, hypotension, seizures, disseminated intravascular coagulation (DIC), poor fetal prognosis. Noncardiogenic pulmonary edema causing severe hypoxemia is usually an early symptom.[4] The true incidence of AFE is unknown but is estimated to occur between 1 in 8,000 and 1 in 80,000 deliveries,[5-7] with reported mortality rates in older reports as high as 60% even with aggressive and immediate treatment.[7] AFE, as a clinical syndrome, was first characterized by the presence of amniotic fluid debris in the maternal pulmonary circulation, and the amniotic fluid was thought to cause an embolic phenomenon.[8,9]

Our patient underwent caesarean section because of foetal distress following prostin induction of labour developed severe dyspnea and hypoxemia six hours post operatively, her chest CT showed diffuse homogeneous ground-glass opacity in the bilateral peripheral fields, suggestive of an early stage of acute respiratory distress syndrome, 2D ECHO showed 60 % of ejection fraction, BNP was 2,032 pg/ml. Repeated 2D-ECHO showed fall in EF=40%. She made a full recovery.

The likely diagnosis was amniotic fluid embolism (AFE), a rare complication of pregnancy with a variable presentation, ranging from cardiac arrest and death through to mild degrees of organ system dysfunction with or without coagulopathy. Amniotic fluid embolism is a clinical diagnosis primarily based on a constellation of clinical sequelae rather than on isolated signs and symptoms. It is important to exclude other causes of sudden cardiorespiratory failure, including pulmonary thromboembolism, air embolism, anesthetic complications, aspiration of gastric contents, anaphylaxis, sepsis, myocardial infarction, and cardiomyopathy.

Firstly, anesthetic complication or massive aspiration was unlikely because the intraoperative course was smooth and her consciousness was well maintained even when she had developed hypoxemia. Secondly, the CT and ultrasonic cardiogram findings did not correlate with pulmonary hypertension or right ventricular failure. Thirdly, blood, sputum, urine and wound culture examination proved negative for infection. Finally, the absence of ischemic changes in 12-lead electrocardiogram and her rapid recovery of left ventricular function did not correlate with myocardial infarction or cardiomyopathy.

4. CONCLUSION

In conclusion, when respiratory distress occurs in patients during the perinatal period, it is important to bear in mind the possibility of amniotic fluid embolism and to proceed with appropriate intensive care. This report highlights the importance of monitoring a patient of post cesarean section and diagnostic facilities needed to suspect amniotic fluid embolism.

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