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Case Report

Amniotic fluid embolism during cesarean section: a rare and catastrophic obstetric: a case report

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ABSTRACT

Amniotic fluid embolism (AFE) is a rare but catastrophic obstetric emergency characterized by sudden cardiovascular collapse, respiratory failure, and disseminated intravascular coagulation. It most commonly occurs during labour, cesarean section, or the immediate postpartum period and remains an important cause of maternal mortality worldwide. We report the case of a 28-year-old primigravida who developed sudden respiratory distress, hypoxemia, profound hypotension, and coagulopathy immediately following placental delivery during an emergency cesarean section under spinal anesthesia. The patient rapidly deteriorated and required endotracheal intubation, mechanical ventilation, aggressive fluid resuscitation, vasopressor support, and massive transfusion of blood products. Intraoperative chest radiography demonstrated diffuse bilateral pulmonary infiltrates consistent with acute pulmonary edema, supporting the clinical diagnosis of amniotic fluid embolism. Following multidisciplinary management in the intensive care unit, including ventilatory support and correction of disseminated intravascular coagulation, the patient recovered progressively, was successfully extubated after 48 hours, and was discharged without neurological sequelae. This case highlights the importance of early recognition, prompt multidisciplinary intervention, and aggressive supportive management in improving maternal outcomes in amniotic fluid embolism.

Keywords: Amniotic fluid embolism, Cesarean section, Obstetric anesthesia, Maternal collapse, Disseminated intravascular coagulation

INTRODUCTION

Amniotic fluid embolism (AFE) is a rare but catastrophic obstetric emergency characterized by the sudden onset of hypoxemia, cardiovascular collapse, and disseminated intravascular coagulation during labour, caesarean section, or the immediate postpartum period. Although the reported incidence is low, ranging from approximately 1.9 to 7.7 cases per 100,000 pregnancies, AFE remains one of the leading causes of maternal mortality and severe maternal morbidity worldwide.[1,2] Early recognition and prompt multidisciplinary intervention are essential to improve maternal and fetal outcomes.

The exact pathophysiology of AFE remains incompletely understood. Current evidence suggests that the syndrome results from an abnormal maternal inflammatory and immunological response triggered by fetal antigens entering the maternal circulation, rather than simple mechanical obstruction by amniotic fluid components. This response leads to pulmonary vasoconstriction, right ventricular dysfunction, hypoxemia, and activation of the coagulation cascade, often resulting in disseminated intravascular coagulation (DIC).[2,3]

Because no specific laboratory test confirms the diagnosis, AFE remains a diagnosis of exclusion based on its characteristic clinical presentation. Immediate airway management, aggressive hemodynamic resuscitation,

correction of coagulopathy with blood component therapy, and coordinated multidisciplinary care are the cornerstones of management and have significantly improved maternal survival in recent years.[3-5]

We report a rare case of amniotic fluid embolism occurring during emergency caesarean section under spinal anesthesia, highlighting its clinical presentation, anesthetic management, multidisciplinary treatment, and favorable maternal outcome following early recognition and prompt intervention.

CASE REPORT

Patient information

A 28-year-old primigravida at 38 weeks of gestation was admitted to the labour ward with prolonged labour and failure of labour progression. The patient had received regular antenatal care and her pregnancy had been uncomplicated.

There was no history of hypertension, diabetes mellitus, cardiovascular disease, asthma, or coagulation disorders. She had no previous surgical history and no known drug allergies.

Because labour failed to progress despite adequate uterine contractions, the obstetric team decided to proceed with an emergency lower segment caesarean section.

Clinical findings

Upon arrival in the operating room, standard monitoring was instituted including electrocardiography, pulse oximetry, and non-invasive blood pressure monitoring.

Initial vital signs were as follows blood pressure was 118/76 mmHg, heart rate was 86 beats per minute, respiratory rate was 16 breaths per minute, and oxygen saturation was 99% on room air.

Physical examination revealed no abnormal findings. Airway assessment demonstrated Mallampati grade II with adequate mouth opening and normal neck mobility.

Diagnostic assessment

Routine antenatal laboratory investigations were within normal limits.

Hemoglobin was 11.6 g/dl, platelet count was 210,000/mm³, and coagulation parameters was within normal range.

Electrocardiography showed normal sinus rhythm. Chest radiography performed during antenatal evaluation revealed no pulmonary abnormalities.

Based on these findings, the patient was considered suitable for regional anesthesia.

Anaesthetic management and intraoperative course

Spinal anesthesia was administered at the L3-L4 interspace with 0.5% hyperbaric bupivacaine (10-12 mg) using a sterile technique. Sensory blockade was assessed using a cold swab and confirmed to reach the T6 dermatome before the surgical procedure was commenced.

The initial intraoperative course was uneventful. A healthy neonate was delivered with Apgar scores of 8 and 9 at one and five minutes, respectively.

Shortly after placental delivery, the patient suddenly complained of severe dyspnea and restlessness. Within a few minutes, oxygen saturation decreased to approximately 85% despite oxygen supplementation, and the blood pressure dropped to 70/40 mmHg. Simultaneously, tachycardia developed with a heart rate exceeding 120 beats per minute.

The patient became progressively agitated and subsequently developed altered consciousness, suggesting significant hypoxia and compromised cerebral perfusion.

Immediate resuscitative measures were initiated. Oxygen was administered via face mask and rapid intravenous fluid infusion was started. Intravenous ephedrine was administered in repeated boluses in an attempt to restore blood pressure.

Despite these measures, the patient's respiratory status deteriorated further. Endotracheal intubation was therefore performed following induction with propofol and succinylcholine, and mechanical ventilation was initiated.

During surgery, excessive bleeding from the operative field was observed. Laboratory investigations subsequently revealed abnormalities in coagulation parameters consistent with disseminated intravascular coagulation.

Following delivery of the fetus, a chest radiograph was obtained intraoperatively to evaluate the cause of the acute respiratory deterioration. The radiograph demonstrated diffuse bilateral pulmonary infiltrates suggestive of acute pulmonary edema (Figure 1). These findings supported the clinical suspicion of cardiopulmonary compromise associated with amniotic fluid embolism.

The patient received transfusion of packed red blood cells, fresh frozen plasma, and platelet concentrates to correct the coagulopathy. Vasopressor support with norepinephrine infusion was initiated to maintain hemodynamic stability. After initial stabilization, the patient was transferred to the intensive care unit for further management and monitoring.

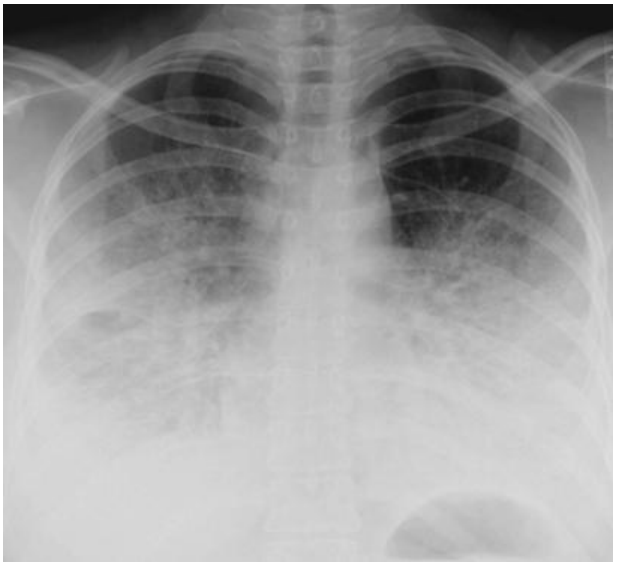

Figure 1: Portable chest radiograph obtained following the onset of cardiopulmonary collapse during cesarean section, demonstrating diffuse bilateral pulmonary infiltrates consistent with acute pulmonary edema in a patient with suspected amniotic fluid embolism.

Follow-up and outcome

In the intensive care unit, the patient remained on mechanical ventilation with continued hemodynamic monitoring. Serial laboratory investigations showed gradual improvement in coagulation parameters following blood component therapy.

Over the next 48 hours, the patient demonstrated progressive clinical improvement. Vasopressor support was gradually tapered and discontinued, and respiratory function improved sufficiently to allow successful extubation.

Neurological examination revealed no deficits. The patient was subsequently transferred to the obstetric ward after stabilization.

She was discharged home on postoperative day seven in stable condition along with her newborn.

DISCUSSION

AFE remains one of the most dramatic and unpredictable obstetric emergencies encountered in perioperative obstetric care. Although uncommon, the condition continues to be associated with significant maternal morbidity and mortality due to its abrupt onset and rapid clinical deterioration. In contemporary obstetric practice, improvements in critical care and early resuscitation have reduced mortality rates compared with earlier decades; however, the syndrome still represents a major cause of maternal collapse during labour and operative delivery.[1]

The pathophysiology of AFE has evolved considerably from the original concept of simple mechanical obstruction of pulmonary vessels by amniotic fluid debris. Current evidence supports the hypothesis that AFE represents a systemic inflammatory response syndrome triggered by fetal antigens entering the maternal circulation, leading to an anaphylactoid reaction.[2] This process results in the release of vasoactive and inflammatory mediators, including endothelin, thromboxane, leukotrienes, and complement activation products, which collectively contribute to pulmonary vasoconstriction, acute right ventricular dysfunction, and severe hypoxemia.

The initial cardiopulmonary collapse observed in AFE is believed to be primarily related to acute pulmonary hypertension and right ventricular failure, which subsequently impair left ventricular filling and reduce cardiac output. This hemodynamic pattern has been described in the ‘biphasic model’ proposed by Clark and further supported by echocardiographic findings in several clinical reports.[3] In this model, the early phase is characterized by pulmonary vasospasm and right ventricular overload, while the later phase involves left ventricular dysfunction and systemic hypotension.

In our patient, the sudden onset of dyspnea, hypoxemia, and hypotension shortly after placental delivery strongly suggested acute cardiopulmonary compromise. The chest radiograph (Figure 1) demonstrated bilateral pulmonary infiltrates compatible with pulmonary edema , a finding that has been reported in many cases of AFE. Pulmonary edema in this context is thought to result from increased pulmonary capillary permeability combined with myocardial dysfunction and ventilation–perfusion mismatch.

Another defining feature of AFE is the development of disseminated intravascular coagulation. Approximately half of patients with AFE develop significant coagulopathy during the clinical course.[4] The mechanism underlying this phenomenon is believed to involve the presence of tissue factor and other procoagulant substances within amniotic fluid. When these components enter the maternal circulation, they activate the extrinsic coagulation pathway, resulting in widespread thrombin generation and consumption of clotting factors.

The differential diagnosis of sudden maternal collapse during cesarean delivery is broad and includes pulmonary thromboembolism, anaphylaxis, myocardial infarction, peripartum cardiomyopathy, and severe obstetric hemorrhage.

In practice, the diagnosis of AFE is usually made clinically after exclusion of these conditions. Proposed diagnostic criteria emphasize the presence of sudden cardiovascular collapse, respiratory compromise, and coagulopathy occurring during labour or shortly after delivery in the absence of another identifiable cause.[5]

Management of AFE is largely supportive and requires rapid coordination among anesthesiologists, obstetricians, and critical care specialists. Immediate priorities include airway stabilization, adequate oxygenation, and aggressive hemodynamic support. Vasopressor and inotropic agents may be required to maintain adequate perfusion. Simultaneously, correction of coagulopathy with blood component therapy—including packed red blood cells, fresh frozen plasma, platelets, and cryoprecipitate—is essential to control hemorrhage.

Recent advances in obstetric critical care have introduced additional therapeutic strategies for severe cases of AFE. These include extracorporeal membrane oxygenation (ECMO), mechanical circulatory support, and advanced hemodynamic monitoring. Several recent case reports and observational studies have demonstrated favorable outcomes with early ECMO initiation in patients with refractory cardiopulmonary failure associated with AFE.[6]

Early recognition and prompt resuscitation remain the most important factors influencing survival. The anesthesiologist plays a central role in this process because intraoperative monitoring allows rapid detection of hemodynamic instability during cesarean delivery.

In the present case, early recognition of maternal deterioration and immediate initiation of airway management, vasopressor support, and blood component therapy likely contributed to the favorable outcome. This case underscores the importance of maintaining a high index of suspicion for AFE when sudden cardiopulmonary collapse occurs during operative delivery.

CONCLUSION

AFE is a sudden and life-threatening obstetric emergency that demands immediate recognition and intervention. This case underscores the importance of vigilant intraoperative monitoring, rapid resuscitation, and effective multidisciplinary coordination in improving maternal outcomes.

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