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Amniotic Fluid Embolism (AFE) Survivor: Case Report

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ABSTRACT

Amniotic fluid embolism (AFE) is a rare but life-threatening condition that occurs during delivery or in the immediate postpartum period. It is characterized by the entrance of amniotic fluid, fetal cells, hair, or other debris into the maternal pulmonary circulation, causing cardiovascular collapse and disseminated intravascular coagulation. Analyzing and reviewing how AFE cases present and are managed can help clinicians rapidly make a diagnosis and start aggressive treatment to improve patient outcomes. A high index of suspicion is necessary given that AFE contributes to a significant portion of the maternal morbidity and mortality rates in the United States.

Keywords: Amniotic fluid embolism, Maternal morbidity, Intrapartum complications, Disseminated intravascular coagulation, Cardiopulmonary arrest

INTRODUCTION

Amniotic fluid embolism (AFE) is a catastrophic obstetric complication that occurs during delivery or in the immediate postpartum period. It is characterized by the entrance of amniotic fluid, fetal cells, hair, or other debris into the maternal pulmonary circulation, causing cardiovascular collapse and disseminated intravascular coagulation [1]. Amniotic fluid is composed mainly of water (99%). The other 1% is formed of nutrients such as glucose, amino acids, lipids, phospholipids, urinary waste products like urea, and shed cells from the fetal body skin and urinary bladder [2].

Even though AFE has been recognized to be rare with an incidence of 2 to 8 cases per 100,000 deliveries, it still accounts for 7.5 to 10% of the maternal mortality rate in the United States [3].

Although the exact pathophysiology of AFE remains unknown, there are two main theories. The first is that fetal material can physically obstruct the maternal micro-vessels in multiple organs like the lung or kidney. The second theory is that the liquids released from the amniotic fluid in the peripartum period can cause an anaphylactoid reaction that can lead to pulmonary vasospasm and activation of platelets, white blood cells, and complement components [4]. Recent studies conducted reveal that the occurrence of AFE is a consequence of the maternal immune response to the fetal material causing complement activation or anaphylactoid reactions rather than an embolism itself [5].

The following four criteria must be present to make a diagnosis of AFE: Acute hypotension or cardiac arrest, acute hypoxia, coagulopathy or severe hemorrhage in the absence of other explanations, all of which must occur during labor, cesarean delivery, dilation & evacuation or within 30 min postpartum with no other explanation of findings [6].

The management of AFE once diagnosed is largely supportive using a multi-disciplinary approach. The patient's vital signs and oxygenation should be continuously monitored in the critical care unit until stabilized. Rehabilitation in the form of physiotherapy and psychotherapy is important to reduce the mother's morbidity [7].

Due to the unpredictable nature of the disease along with the high maternal mortality rate, clinicians must maintain a high degree of suspicion when dealing with women with signs of AFE. This case report aims to showcase how a case of AFE can present so clinicians can make a rapid diagnosis and start immediate treatment to improve the prognosis.

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CASE PRESENTATION

The patient was a 35-year-old pregnant woman with an obstetric score of gravida 15, para 4, abortion 11, and live 4. She was admitted to the labor and delivery unit for monitoring of contraction pain at 39 weeks of gestation, with cervical dilation of 3 cm, and contractions occurring every 15 min. The patient's vitals were normal and the fetal heart rate was 150 bpm. On examination 3 h later, it was found that she was in active labor with a cervix dilated to 6 cm and contractions occurring every 2 min with adequate strength. The fetal heart rate was 140 beats per minute. The patient complained of breathlessness and chest pain in addition to contraction pain. While her vitals were being assessed, she suddenly became unresponsive. Her blood pressure dropped to 80/40 mmHg, her pulse was 110 bpm, respiratory rate was 28/min, and oxygen saturation was 91%. The fetal heart rate showed recurrent bradycardia down to 100 bpm. She was immediately incubated and oxygen was administered in the labor room after which she was rushed to the operating room for an emergency cesarean delivery. The baby was born with an APGAR score of 3 at 1 min, 5 at 5 min, and weighed 9 lbs. While removing the placenta, a moderate degree of abruption was noted. While suturing the uterus closed, the patient had a cardiopulmonary arrest. Cardiopulmonary resuscitation (CPR) was rapidly administered and the patient's pulse returned. The surgery continued. The uterus was noted to be boggy, after which oxytocin and carboprost were administered. The uterus contracted well and there were no signs of uterine bleeding, but the patient started oozing blood from the IV lines. A JP drain was placed, but no bleeding was noted. After the surgery, the patient was placed on mechanical ventilation in the ICU. Her vitals stabilized, and a chest X-ray revealed mild pulmonary vascular congestion with no evidence of pneumothorax or pleural effusion. Laboratory studies revealed hemoglobin of 7 g/dl and evidence of disseminated intravascular coagulation like a platelet count of 70,000, and prolonged PT and PTT. Two units of packed red blood cells were cross-matched and administered. On examination, the patient was not responding adequately to questions, could not recognize her family, and displayed weakness in both lower limbs. MRI and EEG showed no acute intracranial abnormality. She was discharged to the telemetry wards after extubating. On examination later, she demonstrated motor aphasia, bilateral leg weakness, and depression.

Physiotherapy, speech therapy, and psychotherapy were started and the patient showed steady improvement. After a few days, she could take a few small steps with assistance, talk appropriately to her family, swallow, and her mental condition stabilized. She was discharged to a rehabilitation center for further therapy and was treated with Donepezil, Methylphenidate, and Trazodone. After 4 weeks, she recovered with only mild residual memory lag. The baby was discharged in good health along with the mother.

DISCUSSION

Amniotic fluid embolism (AFE) occurs either during delivery or the immediate postpartum period [1]. Our patient experienced it during delivery.

AFE requires a rapid diagnosis and treatment to minimize the disease's burden in the postpartum period. Identifying risk factors can aid clinicians in keeping a high index of suspicion on patients. AFE is found to be more common in older women (mean age: 32 years), multiparous women, fetal macrosomia, and rapid labor associated with the use of uterine stimulants [8]. This patient is aged 35 years, is a multipara, and her baby weighed 9 lbs., which is considered macrosomia.

Signs and symptoms that are indicative of a possible AFE include cough, altered mental status and rapid decline in pulse oximetry, hypotension, cyanosis, fetal bradycardia, encephalopathy, uterine atony, acute pulmonary hypertension, and coagulopathy/severe hemorrhage. In addition, acute dyspnea, sudden agitation, chills, shivering, sweating, coughing, and anxiety are common premonitory symptoms [9]. The patient under discussion displayed loss of consciousness, a rapid drop in oxygen saturation to 91%, blood pressure of 80/40 mmHg, fetal heart rate of 100 bpm, breathlessness, and evidence of DIC.

As demonstrated in the patient, the management of AFE includes a multidisciplinary approach with anesthesia, critical care, and maternal-fetal medicine. A review done in 2016 recommends that the diagnosis of AFE is made clinically due to its varying laboratory findings. Immediate high-quality CPR with advanced life support protocols should be followed, following which immediate delivery is indicated. The patient should be monitored for adequate oxygenation and hemodynamic status should be maintained with vasopressors/inotropes if necessary. Finally, coagulation studies should be done early and managed aggressively with standard massive transfusion protocols [10]. Our patient's baby was delivered through cesarean section after noting fetal distress and maternal hemodynamic instability, as a vaginal delivery was not imminent. She had a cardiopulmonary arrest during the surgery, which was managed with immediate CPR. After stabilization, her further management continued in the critical care unit.

CONCLUSION

AFE is a rare, acute, life-threatening birth complication that can catastrophically impact both the baby and the mother. It remains to be a leading cause of maternal morbidity and mortality in the United States because of its unforeseen nature. The patient discussed above presented with the typical signs and symptoms of AFE like loss of consciousness, hemodynamic instability and drop in oxygen saturation, fetal bradycardia, and clinical features of disseminated intravascular coagulation. She was rapidly managed with immediate stabilization and delivery. With

supportive management and regular rehabilitation, the patient and her baby made a full recovery.

Given the unexpected nature of the disease, identifying risk factors of amniotic fluid embolism is essential to maintain a high index of suspicion when necessary. AFE should be included in the differential diagnosis of all women with acute cardiopulmonary arrest while in labor or in the immediate postpartum period. Analyzing and reviewing how AFE cases present can help clinicians rapidly make a diagnosis and start aggressive treatment to improve patient outcomes. In addition, further research must be done to confirm the pathophysiology of AFE in order to suggest a method of early detection and prevention.

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