Early Recognition and Intervention is Key in Amniotic Fluid Embolism (AFE) – Comparison of Two AFE Cases

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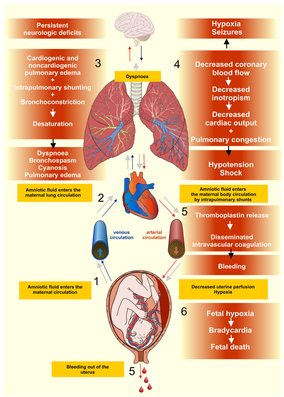
INTRODUCTION

Amniotic-fluid embolism (AFE) is a rare but highly feared complication by both obstetricians and anesthesiologists due to its high mortality[1]. It has a reported frequency of 1.9 to 6.1 per 100,000 births and a mortality rate as high as 60%. It is diagnosed by clinical diagnosis and it is a diagnosis of exclusion. There are both maternal and fetal risk factors, but the most common ones include extremes of maternal age or advanced maternal age greater than 35, medical induction of labor, cesarean delivery, and placenta previa/accreta. It is described as a syndrome of severe anaphylactoid reaction to pregnancy with an abrupt change in mental status, cardiovascular collapse, and disseminated intravascular coagulopathy (DIC) occurring around the time of delivery. The pathophysiology of AFE or some people call it anaphylactoid syndrome of pregnancy continues to be researched and debated[2]. One aspect that is consistent is that it begins with the introduction of amniotic fluid into the mother's circulation. The mode of entry of amniotic fluid into the mother is not fully understood, but a hypothesis suggests that it may be due to pressure gradient. Then the pathophysiology of AFE can be divided into two phases: Phase 1 lasts about 30 minutes and it consists of sudden pulmonary vasoconstriction resulting in pulmonary hypertension and right-sided heart failure;

Phase 2 consists of subsequent left-sided heart failure, endothelial activation and subsequent leakage and bleeding[3]. In recent years, there have been some case reports presenting successful management of AFE using extracorporeal membrane oxygenation (ECMO). We report two case reports of AFE at the same institution, one where ECMO was quickly instituted and the other where it was not.



Gei G., Hankins GDV.: *Amniotic fluid embolism: an update.* Contemp Ob/Gyn. January 2000:45:53–66
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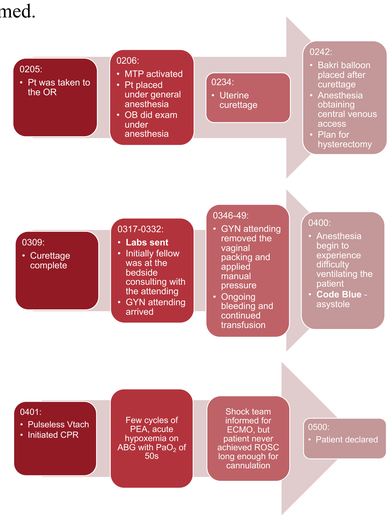
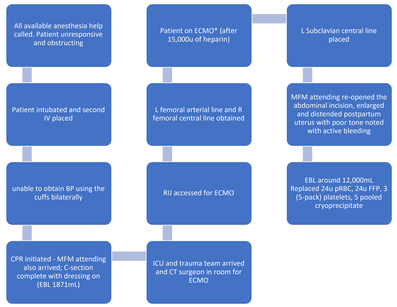
CASE 1

A 15-year-old, gravida 1, para 1-0-0-1 with no medical history other than asthma. She underwent full term vaginal delivery at the outside hospital, which was complicated by postpartum hemorrhage requiring utter-tonics. A Bakari balloon was placed and patient was aggressively transfused and stabilized at the outside hospital. At the outside hospital, patient had fibrinogen level of 60s, INR of 1.4, and estimated blood loss of about 3 liters. On arrival, patient was noted to be in advanced DIC with fibrinogen of 0. The patient quickly went into pulseless electrical activity (PEA) with cardiovascular collapse. Cardiopulmonary recitation (CPR) was quickly initiated. During CPR, the patient continued to have distending abdomen possibly from intraabdominal bleeding. ECMO team was called, but patient never had return of spontaneous circulation (ROSC) or palpable pulse long enough to initiate ECMO. Patient was pronounced deceased after an hour of resuscitation efforts. On autopsy, AFE was confirmed.



CASE 2

A 43-year old, gravid 3, para 0-0-2-0, 38 weeks gestation with history of gestational hypertension, gestational diabetes, right fundal fibroid, obesity (BMI 43), and anemia presented for induction of labor for continued gestational hypertension. During labor, fetus did not tolerate the labor and had several late decelerations. Therefore, a decision was made to deliver via cesarean section (c-section). Patient was initially planned for non-scheduled, non-urgent, c-section, but patient was progressing and had eaten something in the evening, so the c-section was delayed until the next morning. Patient was taken to the operating room and proceeded with combined spinal-epidural as usual protocol at our institution. The skin incision to delivery time was 14 minutes. The baby's Apgars scores was 9 and the mother was doing well. During fascial closure, the patient noted to have a change in mental status. Patient briefly responded to verbal commands, but soon lost consciousness. There was no intra-abdominal or vaginal bleeding noted on the surgical field. Patient's hemoglobin was checked with Hemacue, which was 8.0 and glucose level was 200. Following events ensued:



The patient was taken to our cardiothoracic ICU on VA-ECMO. She was decannulated 2 days later, and the patient was discharged 16 days later with full neurological functions.

DISCUSSION

AFE is a difficult clinical diagnosis. Recent literature portrays a syndrome of severe anaphylactoid reaction to pregnancy with an abrupt change in mental status, CV collapse and DIC occurring around the time of delivery[2]. DIC is present in about 83% of the patients with AFE with variable time of onset[3]. The exact pathophysiology is still poorly understood, but the current theory states that the tissue factor present in amniotic fluid activates the extrinsic pathway. This leads to binding of factor VII and activation of factor X resulting in a consumptive coagulopathy[3]. The key factors of management of AFE are early recognition and prompt resuscitation. It is important to ask for help early, immediately start high-quality cardiopulmonary resuscitation (CPR), intubate and secure the airway, obtain adequate access, and start any necessary vasopressors or inotropes, and aggressive management of bleeding including activation of massive transfusion protocol. It is also important to immediately deliver a fetus at more than 23 weeks of gestational age and start support for the right heart.[4] The utilization of ECMO on AFE patients has been reported in recent literatures. However, there are not enough data and there are concerns for increased bleeding risks in AFE patients[5] . From these cases, we learned AFE should be considered early in patients with CV collapse and/or DIC at the time of delivery. Also, institution of ECMO should be considered promptly despite the risk of bleeding[4]. However, it is not always easily established depending on the institutions protocol and availability of the trained personnel. In more recent literatures, possible use of A-OK therapy has been discussed[6]. The A-OK therapy includes a cocktail of atropine (1 mg), ondansetron (8 mg), and ketorlac (30 mg). The idea is to inhibit pathways that are involved in the pathogenesis of AFE.[6] Atropine and ondansetron may block serotonin and vagal stimulation improving CV function, and ketrolac inhibits the coagulopathy by inhibiting thromboxane.[6] The evidence is limited on this therapy, but these medications should be considered if there is a suspected diagnosis of AFE.

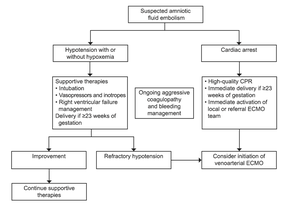


Fig. 1. Management of severe amniotic fluid embolism. CPR, cardiopulmonary resuscitation; ECMO, extracorporeal membrane oxygenation.
Viau-Lapointe. Extracorporeal Therapies for AFE. Obstet Gynecol 2019.

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