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Management of Amniotic Fluid Embolism in a Grand Multipara after Amniotomy: A Case Report

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Abstract

Background: The Amniotic Fluid Embolism (AFE) is a life-threatening obstetric complication with a low incidence that occurs approximately in 2 to 8 per 100,000 deliveries with a fatality rate between 11 to 26% in developed nations.

Objective: Remark the importance of prompt decision making in the management of a patient with AFE to yield favorable maternal and neonatal outcomes in the Hospital Angeles Lomas, in México.

Case report: This is a 34-year-old, gravida 7, para 6, abortus 1, woman admitted for labor management at gestational age of 37 + 5 weeks. Following amniotomy, she suddenly goes into a cardiorespiratory collapse, requiring cardiopulmonary resuscitation (CPR) and advanced airway management.

Conclusion: Since this is an infrequent condition that presents as a sudden and unforeseeable emergency, the rapid recognition and the instant and accurate decision making of the multidisciplinary team, impacts directly patient prognosis. Once the patient is stable, taking a close look at the clinical presentation, laboratory parameters and imaging tests lead to the exclusion diagnosis of AFE.

Keywords: Amniotic Fluid Embolism, cardiovascular collapse, amniotomy

Introduction

The amniotic fluid embolism (AFE) is a low incidence obstetric complication with a high fatality rate depending on the geographic region¹. In UK, the obstetric surveillance system stated the fatality rate was 19% during 2005-2014, though the other 9 systematic reviews revealed that the fatality rate was 24.8%². The global occurrence of AFE ranges from 1 in 8000 to 1 in 80 000 deliveries and global incidence remains unclear ^[1]. However, the incidence in the US, UK, Australia and Canada occurs in 1.7 to 7.7 per 100,000 deliveries, whereas de mortality rate in developing countries is about 1.8 to 5.9 per 100,000 deliveries, and in developed countries it is 0.5 to 1.7 per 100, 000 deliveries ^[1, 2]. In Germany, AFE was a leading cause of death during childbirth, and in Australia, it is the leading direct cause of maternal death ^[3]. The classic triad is hypoxia, hypotension, and coagulopathy, though the clinical presentation includes sudden cardiovascular collapse, dyspnea, cyanosis, pulmonary edema, seizures, altered mental status, and disseminated intravascular coagulation (DIC) ^[2]. A big percent ultimately developed coagulopathy that can lead to postpartum hemorrhage, considered one of the major complications ^[3-5]. However, the most common initial symptoms were a feeling of agitation or of impending doom, hypotension, dyspnea, and fetal compromise ^[3]. The initial approach of these patients is based on the clinical suspicion of a pulmonary embolism, however, after taking laboratory and imaging tests, the differential diagnosis of AFE can be established ^[6].

Case Report

This 34-year-old female was pregnant with 37 + 5 weeks, G7, P5, A1. It is important to mention that in her third pregnancy, during the delivery of the placenta, she experienced placental abruption causing obstetric hemorrhage that required management in the Intensive Care Unit, evolving satisfactorily. According to her medical history, she does not have any chronic-degenerative disorders, however, in her family history is mentioned that her sister suffered an unspecific thrombotic event.

Current condition: A pregnant woman with 37+5 weeks is admitted in the labor room during the latent phase of the first labor stage. At the examination, she is neurologically

intact, alert, conscious, normal integument coloration, normal cardiopulmonary system, abdomen with a normal gravid uterus. The cervix at 4 cm dilatation and 60% effacement with intact membranes. Meanwhile, the cardiotocography monitor reports the fetal heart rate (FHR) in normal range. According to these findings, the obstetrician in charge decides to start labor management.

First, initiation of labor analgesia with injection of local anesthetic through epidural catheter. After the administration of intravenous fluid loading with 300 ml of crystalloids, the epidural block is performed at lumbar L2-L3 interspace using a Touhy needle No 16 G and the catheter was inserted in cephalic orientation without any type of incident. It is administered by epidural catheter, ropivacaine 0.2%, 28 mg, with a total volume of 14 ml achieving 1/10 of the Numerical Rating Scale (NRS). Under non-invasive monitoring, the patient is hemodynamically stable with vital signs in normal range.

It has been 20 minutes after the epidural block, the obstetrician in charge makes a second assessment of the patient finding a cervix at 6 cm dilation and 80% effacement, fetal presentation at the second Hodge plane. To speed up the labor, the obstetrician performs an amniotomy, obtaining clear amniotic fluid with few normal mucus. Meanwhile, the fetal assessment shows FHR in normal range.

Five minutes after the amniotomy, the patient complains about moderate precordial pain and dyspnea, also at first sight it was notorious the acrocyanosis and distress face. According to the fetal monitoring, it shows abrupt decrease of the FHR, consequently the patient is taken to the delivery room as an emergency.

Once in the delivery room, the patient loses conscious and alertness besides there are not carotid nor femoral pulse, hence the anesthesiologist in charge starts cardiopulmonary resuscitation (CPR). After the end of the first CPR cycle, the pulse is checked without return of spontaneous circulation meanwhile asystole is identified on the monitor, so 1 mg of IV adrenaline is administered and CPR continues. At the end of the second CPR cycle, there is pulse with return of spontaneous circulation, however, the patient goes into a supraventricular tachycardia with heart rate (HR) of 220 beats per minute.

Then, advanced airway management is performed successfully, and they call a code (MATER) in the Hospital. The multidisciplinary team starts management of supraventricular tachycardia by doing vagal maneuvers that fail to resolve it, due to the patient's hemodynamic stability, they perform the first electrical cardioversion at 50 Joules unsuccessfully, and the second electrical cardioversion is at 100 Joules reaching a sinus HR of 135 beats per minute.

Beyond patient's supportive care, a female baby is born by vaginal delivery using the Vacuum extraction delivery technique. The newborn requires two cycles of positive pressure ventilation, giving her an initial APGAR score of 6 points and 8 points at 10 minutes, she is taken to Special Care Nursery.

After the vaginal delivery, venous central catheter and a urinary catheter are placed, then vasopressors and sedation infusion start (midazolam, propofol and neuromuscular blocking agents). While the patient is being taken to the Intensive Care Unit (ICU), some pink frothy secretions appear in the orotracheal tube, raising suspicion of acute pulmonary edema. Additionally, unexplained hematuria is

observed through the urinary catheter, raising concern for disseminated intravascular coagulation (DIC). The following days she continued with supportive care in ICU.

The presumptive diagnosis was pulmonary thromboembolism (PTE) versus AFE. The first report of laboratory test was hematocrit 29.4% and hemoglobin 9.62 g/dl, leucocytes 37,000/mm³, platelets 170,000/mm³, D-Dimer 10,000 ng/ml, fibrinogen 0, prothrombin time 13.1 seconds, partial thromboplastin time 30.4 second, INR 1.08 second. It is important to mention that the D-Dimer levels maintained in those ranges for at least three days. Additionally, the pulmonary angiography showed no blood clots in lung vessels. According to the clinical presentation, laboratory tests and imaging studies, PTE was ruled out, allowing for the diagnosis of AFE to be established by exclusion.

The patient is treated by a multidisciplinary team including ICU, cardiology and hematology, they provided supportive care and blood components transfusions until she evolved favorably. Also, the female newborn had favorable evolution and both patients were discharged from the hospital after five days of clinical improvement.

Discussion

AFE is a life-threatening obstetric emergency, even though the incidence varies according to the population studied, in all cases is very low ^[1]. But the fatality rate is high, ranging from 11% to 26% ^[1]. This condition usually presents as an unexpected cardiopulmonary collapse and subsequent coagulopathy, the prognosis of the mother and neonate depends mainly on a prompt, aggressive rescue response by the multidisciplinary team ^[7, 8].

Some of the reported risk factors for the development of AFE include multiparity, advanced maternal age, male fetus, trauma, medical induction of labor, caesarean section, operative vaginal delivery, placental abruption, placenta previa, preeclampsia, and cervical laceration or uterine rupture ^[2, 3]. Also, it has stated that the third trimester of pregnancy, slow progress in labor or lack of it, following amniorrhexis, amniotomy, amniocentesis or amnioinfusion, and conditions like chorioamnionitis, are associated with an increased risk of AFE ^[9-11]. However, risk factors with strong evidence proposed for AFE are induction of labor by any means, assisted delivery, cesarean section, renal disease, placenta previa, placental abruption, polyhydramnios, eclampsia, amnioinfusion, and dilation and curettage ^[2, 3]. In another study, placental accreta spectrum was the risk factor with greatest association with this condition (10.01;95% CI, 7.03-14.24) ^[3]. Moreover, it is mentioned a 25-fold higher risk of AFE in women with cerebrovascular disorder, and 70-fold greater risk in those with cardiac disease ^[3]. According to ethnicity, non-Hispanic Blacks have more than twice the risk of developing this disorder ^[3]. Further, the International AFE registry reveals that 66% of women with AFE reported atopy and/or latex or food allergies ^[3]. Besides, the analysis of the national registry in US exposes that 70% of cases of AFE occur during labor, 11% after vaginal delivery, and 19% during cesarean delivery ^[12]. Furthermore, one population-based case-cohort study reported it has been associated to spontaneous vaginal delivery increasing the risk 12 times than cesarean delivery (OR 12.3; IC 95% 3.3-39.2) ^[1, 13].

According to the medical history of our patient, her risk factors were multiparity (six vaginal deliveries), advanced

maternal age, placental abruption in her third pregnancy, performance of amniotomy, third trimester of pregnancy and management of labor.

The pathophysiology of this condition has two main theories, the mechanical theory states that a clot is produced from amniotic fluid components like meconium, sebum, lanugo and cells, that jeopardize respiratory mechanics; the second one, immune mediated theory explains that abnormal maternal response at fetus tissue leads to a clinical presentation similar to a systemic inflammatory response syndrome due to inflammatory mediators and complement activation [14, 15]. Fetal antigens enter maternal circulation, triggering a response similar to systemic inflammatory response syndrome with activation of the coagulation cascade leading to DIC and inflammatory mediated suppression of myocardial function [3]. About 90% of the cases debut by sudden cardiorespiratory collapse [9].

Progression usually occurs in two phases: phase 1; pulmonary artery vasospasm with pulmonary hypertension and elevated right ventricular pressure cause hypoxia [3]. Hypoxia causes myocardial capillary damage and pulmonary damage, left heart failure, and acute respiratory distress syndrome [3]. Women who survive these events may enter phase 2 that consists of a hemorrhagic phase characterized by massive hemorrhage and DIC; however, fatal consumptive coagulopathy may be initial presentation [3]. DIC is present in up to 83% of cases and it is commonly manifested by bleeding from venipunctures or surgical sites, hematuria, gastrointestinal and vaginal hemorrhage [12]. Also, pulmonary edema is a common occurrence in survivors. Pink frothy secretion indicating pulmonary edema and hematuria were presented by our patient while she was being taken to ICU [3].

The clinical diagnostic criteria of AFE comprised of six clinical symptoms (hypotension, cardiac arrest, acute hypoxia, cyanosis, dyspnea, coagulopathy) and timing

(onset during labor, cesarean section, dilation and evacuation, within 30 min postpartum) [2]. However, all the following four criteria must be present for a diagnosis of AFE:

1. Sudden onset of cardiorespiratory arrest or both hypotension (SBP <90mmHg) and respiratory compromise (dyspnea, SpO₂ <90%) [3].
2. Documentation of overt DIC following the events in item 1. Coagulopathy must be detected prior to the loss of enough blood to itself be the cause of a dilutional or consumptive coagulopathy [3].
3. Clinical onset during labor or with 30 minutes of the delivery of the placenta [3].
4. No fever (<38.0°C) during labor [3].

Also, our patient met the four criteria for the diagnosis of AFE, she presented during labor a sudden cardiorespiratory arrest, dyspnea and cyanosis with no fever and after the CPR she started with hematuria, decrease in platelets count and fibrinogen.

Prompt resuscitation should start as early as possible before diagnosis with AFE for better prognosis [2]. The rescue management is basically the cornerstone, continuing with aggressive supportive care [1]. Immediate and precise multidisciplinary team response determines patient survival, in this case report, it is mentioned the effective and successful response made by the hospital multidisciplinary team that led to the mother and daughter wellness and survival. According to a metanalysis compiling studies on AFE from 1966 to 2015, published by the Society for Maternal-Fetal Medicine, recommends a multidisciplinary approach and emphasizes that a diagnosis of AFE is a diagnosis of exclusion [12]. Additionally, it highlights Recommendations about the management of AFE in table 1 [12].

Table 1: Summary of Recommendations

Number	Recommendations	Grade
1	We recommend consideration of AFE in the differential diagnosis of sudden cardiorespiratory collapse in the laboring or recently delivered woman.	1C Strong Recommendation Weak-quality evidence
2	We do not recommend the use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of AFE; at the present time, AFE remains a clinical diagnosis.	1C Strong Recommendation Weak-quality evidence
3	We recommend the provision of immediate high-quality cardiopulmonary resuscitation with standard BCLS and ACLS protocols in patient who develop cardiac arrest associated with AFE.	1C Strong Recommendation Weak-quality evidence
4	We recommend that a multidisciplinary team including anesthesia, respiratory therapy, critical care, and maternal-fetal medicine should be involved in ongoing care of woman with AFE.	Best practice
5	Following cardiac arrest with AFE, we recommend immediate delivery in the presence of a fetus >23 weeks of gestation.	2C Weak Recommendation Weak-quality evidence
6	We recommend the provision of adequate oxygenation and ventilation and, when indicated by hemodynamic status, the use of vasopressors and inotropic agents in the initial management of AFE. Excessive fluid administration should be avoided.	1C Strong Recommendation Weak-quality evidence
7	Because coagulopathy may follow cardiovascular collapse with AFE, we recommend early assessment of clotting status and early aggressive management of clinical bleeding with standard massive transfusion protocols.	1C Strong Recommendation Weak-quality evidence

ACLS, Advanced Cardiac Life Support; AFE, Amniotic Fluid Embolism; BCLS: Basic Cardiac Life Support; GRADE, Grading of Recommendations Assessment, Development and Evaluation.

SMFM. Amniotic fluid embolism: diagnosis and management. Am J Obstet Gynecol 2016.

The diagnosis is one of the exclusion criteria, there is still

no pathognomic marker of AFE [16]. The presence of fetal squamous cells in pulmonary vasculature was once considered diagnostic, but it is now considered to be neither sensitive nor specific. Diagnosis is aided by non-specific and specific diagnostic tests which are summarized in table 2 [16].

Table 2: Diagnosis of AFE

Test	Findings
Non-specific	
Full blood count	Low Haemoglobin
Coagulation	Low platelets, increased PT and APTT, low fibrinogen
Arterial blood gas	Hypoxemia, raised PaCO ₂
Chest X-ray	Normal, cardiomegaly, pulmonary edema
ECG	Right or left ventricular dysfunction, low ejection fraction
More specific	
Pulmonary blood sample	Presence of squamous cells coated with neutrophils and presence of fetal debris
Sialyl Tn antigen	Raised
Zinc coproporphyrin	Raised
Serum tryptase levels	Normal or raised

PT, prothrombin time; APTT, activated partial thromboplastin time; V/Q, ventilation/perfusion.

Non-specific tests include a full blood count and coagulation screen to demonstrate low hemoglobin and abnormal coagulation, and arterial blood gases showing hypoxemia [16]. Chest X-ray in the early stages before ARDS develops may be normal, also ECG may demonstrate a right ventricular strain pattern in the early stage [16]. However, ultrasound is useful to demonstrate ventricular dysfunction [16].

Diagnostic tests include cytological analysis of central venous blood and broncho-alveolar fluid, Sialyl Tn antigen test, zinc coproporphyrin concentration, and serum tryptase concentrations [16]. The presence of squamous cells, especially if they are coated with neutrophils and found in significant large numbers along with fetal debris such as mucin or hair, is suggestive of AFE [16].

A study reveals changes in the activation of the complement pathway, complement levels in the AFE group were severely depressed [3]. C3 in AFE group was 44 compared with 117.2 in the postpartum control group, and C4 was 10.7 in the AFE group versus 29.4 in the postpartum group [3]. That suggests that complement activation may play an important role in the pathophysiology of AFE [3]. Also, in cases reported in the literature, there is an increase of serum tryptase levels, this may support the diagnosis of AFE [3]. Tryptase has been suggested to be a more convenient serum marker for diagnosis of AFE [1]. Since, mast cell degranulation is incited during anaphylactoid reaction; tryptase is also released during mast cell degranulation [1]. Serum tryptase will be detectable between 30 min and 2 h after the onset of anaphylaxis, and its peak is one to 2 h; its half-life is 2 h with stable concentration in serum [1]. Even the serum markers such as monoclonal antibody TKH-2 or zinc coproporphyrin are proven to be the sensitive methods for diagnosis of AFE, these markers are not immediately available for diagnosis of AFE [1].

Treatment of left heart failure is recommended with inotropic support [3]. For DIC, in the setting of massive hemorrhage, blood product administration should not be delayed while awaiting the results of laboratory tests [3]. Instead, early aggressive resuscitation with packed red blood cells, fresh-frozen plasma, and platelets at a ratio of 1:1:1 (hemostatic resuscitation) results in improved outcomes [3]. Consider activated factor VIIa for severe hemorrhage, as well as bilateral uterine artery embolization to control blood loss [3].

Mortality and fatality rates are high if classical triad symptoms of AFE are present, remarking that the first 85 minutes of the initial management are crucial for the patient

survival [2, 3]. Additionally, neonatal survival has been reported to be 79% in the US registry and 78% in UK registry. The intact infant survival rate is 70% [3]. Furthermore, the fetal mortality is about 5% [3]. It is important to mention that epigenetics studies show that mortality is lower in Hispanics compared to Caucasian and Afro-American population [17].

Conclusion

AFE is a low incidence condition; however, fatality rate is high. It is considered an unpredictable and unpreventable event that can be easily misdiagnosed, altering statistics. Providing clinicians with information that may improve the ability to make a timely diagnosis and establish appropriate supportive treatment is crucial to improve maternal and perinatal prognosis. The initial management centers in aggressive supportive care including cardiopulmonary resuscitation and advanced airway management. Once the danger has passed, treatment focuses in solving complications basically related to coagulopathy. Finally, AFE diagnosis relies on a combination of clinical signs, laboratory results and imaging tests to rule out other causes.

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Conflicts of Interest Disclosure

The authors declare that they have no financial conflicts of interest regarding the content of this report.

Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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