J Cases Obstet Gynecol, 2017;4(4):84-86

Case Report

# Near Miss Postpartum Woman with an Amniotic Fluid Embolism, a Rarity Requiring Multidisciplinary Approach

Natalia Hanson[1]*, Christopher Heron[1], Emil R. Szabo[2]

[1]Department of Family Medicine, Penn State Health Milton S. Hershey Medical Center, Mount Nittany Medical Center,1850 East Park Ave, Suite 207, State College, PA, 16803, United States
[2]Department of Obstetrics- Gynecology, Solo Practitioner, 251 Easterly Parkway, State College, PA, 16801, United States

## Abstract

An amniotic fluid embolism (AFE) is a condition with one of the highest pregnancy- associated mortality rates, accounting for 5-15% of pregnancy-related deaths worldwide. This article describes a case of a 36- year- old G4P1 at 38 weeks of gestation who underwent a planned Cesarean section for a placenta previa which was complicated by cardiac arrest and disseminated intravascular coagulopathy secondary to a suspected AFE. The patient was treated with a multidisciplinary approach, rapid response to alterations in vital stability and factor VII. The unpredictable nature of AFE and the gravity of its sequelae warrant high suspicion in those at risk for AFE, as early recognition and prompt treatment can improve morbidity and mortality.

## Key Words:

Amniotic fluid embolism, disseminated intravascular coagulation, vaginal bleeding, factor VII

## Introduction

An amniotic fluid embolism (AFE) is a rare condition with one of the highest mortality rates associated with pregnancy, accounting for approximately 5-15% of pregnancy related deaths worldwide [1,2,3]. These mortalities often result from AFE-induced cardiac arrest and disseminated intravascular coagulopathy (DIC). We examine a case of AFE in the setting of primary hospitalization in order to review the evaluation and management of the condition, its complications, and to reiterate the importance of prompt recognition of AFE.

**Article History:**
Received: 11/07/2016
Accepted: 21/07/2017

***Correspondence:** Natalia Hanson
**Address:** Penn State Health Milton S. Hershey Medical Center, Mount Nittany Medical Center,1850 East Park Ave, Suite 207, State College, PA, 16803, United States
**Tel:** 8144241230
**Fax:** 8142352482
**e-mail:** nhanson@hmc.psu.edu

## Case Presentation

A 36-year-old G4P1 patient at 38-weeks of gestation presented for a scheduled cesarean section for complete placenta previa. She has one daughter born via spontaneous vaginal delivery and a history of two spontaneous abortions. Her prenatal screening was unremarkable except for non-immunity to rubella; she was group B strep negative, O+ blood type, and Coombs negative. She smoked less than five cigarettes per day during the pregnancy and reported taking Ambien for sleep. Her pregnancy course was unremarkable except for painless spotting which started during the second trimester secondary to a complete placenta previa confirmed by ultrasound. A Cesarean section was scheduled, and given concerns for postpartum complications, the procedure was moved to a larger operating room. Prior to the operation, blood values were as follows: Hgb: 11.7, Hct: 35.3, PTT: 27.7, INR: 0.9 The child was born with an APGAR of 8/9 at 0804. At 0805 the patient developed seizure-like activity and became unresponsive. She was intubated, received Atro-

pine 0.8 mg and compressions were initiated. Return of spontaneous circulation occurred at 0812 and a central venous catheter was placed. There was an increase of bleeding noted from the vagina at 0900 refractory to fundal massage, Cytotec per rectum, Hebamate 250 mg IM, and Methergine 0.2 mg IM were administered. An infusion of normal saline with Pitocin was started. Laboratory values drawn at 0930 were concerning for DIC: Hgb: 8.2, Hct: 24.6, PLT: 79, INR: 2.0, PTT: 66.9, DDimer: >500, Fibrin degrade products : >40. Given the profound blood loss and anemia, the patient was transfused with six units of packed red blood cells, four units fresh frozen plasma, and two units cryoprecipitate. A Bakrey balloon was also placed. A transthoracic echocardiogram revealed increased pulmonary artery pressure >35, indicative of moderate to severe pulmonary hypertension with decreased right ventricular function. At this point, the patient required a higher level of care than available, and arrangements were made for transfer to tertiary care center. Prior to helicopter transfer, the patient was given a Factor VII infusion. Studies performed at the tertiary center reflected that the most likely cause of the symptoms were an AFE resulting in DIC and cardiac arrest. She was discharged home after a week stay in the hospital and her lab values were as follows: INR: 1.02, Hgb: 10.9, Plt:223.

## Discussion

An AFE typically ensues when amniotic fluid/ fetal cells enter the maternal blood stream [3]. It usually requires cells to enter the circulation in order for an embolism to occur. The fetal material then accumulates in vessels and the resultant pulmonary vasoconstriction leads to pulmonary hypertension [1,2,3]. There is, consequentially, an increase in right ventricular pressure and subsequently right congestive heart failure. This can lead to cardiopulmonary collapse as reflected by sudden hypoxia, increased work of breathing, respiratory distress and finally cardiac arrest [3,4]. The cells may also invade the uterine tissue locally [3]. This causes an anaphylactoid-like reaction and can ultimately result in DIC or uncontrollable postpartum hemorrhage secondary to an atonic uterus. Typically, evidence of an amniotic embolism and subsequent complications arise within thirty minutes of initial insult [1]. A diagnosis of amniotic embolism is a diagnosis of exclusion, as there is no uniform clinical diagnostic criteria for an AFE [2,4]. The proposed descriptive symptoms include sudden onset of restlessness, tachypnea, new audible wheeze, altered level of consciousness, and fetal compromise such as fetal bradycardia [3,5]. Because of the rapid onset and severity of symptoms, a high level of suspicion for AFE should be maintained in order to preempt and compensate for its sequelae. Commonly cited risk factors include age greater than 35, placenta previa, cesarean section, multiple pregnancies and induction of labor [1,3,4,6]. There has been some research with regards to diagnostic markers used to assess severity of AFE. Since approximately 50% of women with an AFE will develop DIC [1], diagnostic markers to identify DIC- CBC, fibrinogen, fibrinogen- fibrin split products, PTT and INR- are recommended [6]. While marked decreases of C3/C4 levels have been demonstrated to have 100% specificity and 88% sensitivity for an AFE [8], use is limited due to availability and turn- around time. C1 esterase inhibitor, a more recently identified marker, inhibits c1 esterase, factor XIIa and Kallikrein [3]. Low levels of C1 esterase inhibitor, it is theorized, can be found prior to the onset of major symptoms of an AFE, but further research is needed. Treatment of AFE should begin with basic lifesaving care, including, when indicated, intubation, volume replacement, early use of pressers, and correction of coagulopathies [2,5]. It is important to assess fibrin levels promptly as DIC can progress quickly. It was found that the fibrinogen levels would decline to <100 mg/dL (nm 200-400 mg/dL) within two hours of physical signs of DIC. [3]. Delayed transfusion results in an increase in mortality rate and early transfusion with fresh frozen plasma can be indispensable to help control the DIC [3]. There have been some instances where recombinant factor VIIa has been used to successfully manage DIC in patients with AFE [1]. These women were noted to have a decrease in tissue factor concentration, increased uterine atony, uterine rupture or abnormal placenta [9]. However, recombinant factor VII is not well established, and should only be considered with women who are refractive to traditional treatment. In our case, there were no symptoms suggesting an AFE prior to the cardiac arrest. However, high suspicion was warranted in this case given her maternal age, placement of the placenta, and method of delivery. Completing the caesarean section in the larger operating room allowed for a larger, multidisciplinary team to help with resuscitation and provide more efficient care. This case reflects the importance of considering all potential com-

plications and their associated risk factors, because even with high suspicion, there may not be any warning signs. AFE is dangerous but a manageable rarity in delivering mothers. High suspicion and prompt recognition of AFE can improve morbidity and mortality. It is important to consider AFE with sudden changes such as restlessness, new onset wheezing, respiratory changes, uncontrolled postpartum bleeding, and hemodynamic changes in the fetus. There is no definite tool that can be used to predict the onset of AFE, thus the importance of high clinical suspicion. There are developing diagnostic tool that may be able to help evaluate patients for suspected AFE. This ongoing development of evidence based medicine and collection of data and research will further improve the obstetric care of these women as well as a strong and supportive multidisciplinary team.

**Acknowledgement**
None

**Declaration of Interest**
None

## References

1. Shen F, Wang L, Yang W, Chen Y. From appearance to essence: 10 years review of atypical amniotic fluid embolism. Arch Gynecol Obstet 2015; 293(2):329-34.
2. Rath W, Hofer S, Sinicina I. Amniotic Fluid Embolism: an interdisciplinary challenge. Deutches Arzteblatt International 2014; 111 (8): 126-32.
3. Kanayama N, Tamura N. Amniotic fluid embolism: Pathophysiology and new strategies for management. Journal of Obstetrics and Gynecology Research 2014; 40 (6): 1507-17.
4. Fitzpatrick KE, Tuffnell D, Kurinczuk JJ, Knight M. Incidence, risk factors, management and outcomes of ambiotic- fluid embolism: a population- based cohort and nested case- control study. British Journal of Obstetrics and Gynecology 2015; 123 (1): 100-9.
5. Kulshrestha A, Mathur M. Amniotic fluid embolism: A diagnostic dilemma. Anesthesia Essays and Researches 2011; 5 (2): 227-230.
6. Cunningham FG, Nelson D. Disseminated Intravascular Coagulation Syndromes in Obstetrics. Obstetrics and Gynecology: Clinical Expert Series 2015; 126 (5): 999- 1011.
7. Busardo F, Frati P, Zaami S, Fineschi V. Amniotic Fluid Embolism Pathophysiology Suggest the New Diagnostic Armamentarium: B- Tryptase and Complement Fraction C3-C4 are the Indespensable Working Tools 2015; 16: 6557-70.
8. Benson MD, Kobayashi H, Silver RK, Oi H, Greenberger PA, Terao T. Immunologic studies in presumed amniotic fluid ambolism. Obstet Gynecol 2001; 97: 510-14.
9. Phillips LE, McLintock C, Pollock W, Gatt S, Popham P, Jankelowitz G, et al. Recombinant activated factor VII in obstetric hemorrhagic experiences from the Australian and New Zealand Haemostasis Registry. Anesth Analg; 109 (6): 1908-1915.
10. Yoneyama K, Sekiguchi A, Matsushima T, Kawase R, Nakai A, Asakura H, et al. Clinical characteristics of amniotic fluid embolism: An experience of 29 years. The Journal of Obstetrics and Gynecology Research 2014; 40 (7): 1862-70.
11. Nakagami H, Kajihara T, Kamei Y, Ishihara O, Kayano H, Sasaki A, et al. Amniotic components in the uterine vasculature and their role in amniotic fluid embolism. The Journal of Obstetrics and Gynecology Research 2015; 41 (6): 870-75.
12. Liao WC, Jaw FS. A noninvasive evaluation analysis of amniotic fluid embolism and disseminated intravascular coagulopathy. The Journal of Maternal-Fetal and Neonatal Medicine 2011; 24 (11): 1411-15.
13. Guillaume A, Sananes N, Akladios C, Boudier E, Diemunsch P, Averous G, et al. Amniotic fluid embolism: 10 year retrospective study in a level III maternity hospital. European Journal of Obstetrics & Gynecology and Reproductive Biology 2013; 169: 189-192.
14. Eskandari N, Feldman N, Greenspoon J. Factor VII Deficiency in Pregnancy Treated with Recombinant Factor VIIa. The American College of Obstetricians and Gynecologists 2002; 99 (5): 935-37.