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# A Strategy of Bundle Transfusion for Amniotic Fluid Embolism-Induced Disseminated Intravascular Coagulation: Report of Two Cases

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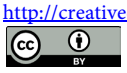



## Abstract

**Aim:** Amniotic fluid embolism (AFE) is a potentially lethal obstetric emergency with a high maternal mortality rate. Our report details two successful cases of patients who developed non-overt disseminated intravascular coagulation (DIC) following cesarean section, likely induced by AFE, and were managed using a bundle transfusion strategy prior to experiencing massive postpartum hemorrhage. **Methods:** An aggressive bundle transfusion strategy was initiated upon early laboratory evidence of coagulopathy (notably hypofibrinogenemia and prolonged clotting time) in suspected amniotic fluid embolism and administered as a rapid, near-simultaneous combination of tranexamic acid, fibrinogen concentrate, prothrombin complex concentrate, cryoprecipitate, and blood components. **Results:** The bundle transfusion strategy effectively corrected coagulation dysfunction before the condition progressed to DIC and postpartum hemorrhage. **Discussion:** This case report provides a reference for transfusion strategies in non-overt DIC induced by AFE, but the risks associated with bundle transfusion warrant further evaluation in future studies. **Conclusion:** The successful treatment of both cases is due to early detection of AFE and aggressive management of coagulopathy before clinical DIC presented. **Synopsis:** We report two successful treatment cases due to early detection of AFE and aggressive management of coagulopathy before clinical DIC presented.

## Subject Areas

Women's Health

## Keywords

Amniotic Fluid Embolism, Coagulopathy, Disseminated Intravascular

Coagulation, Bundle Transfusion

## 1. Introduction

Amniotic fluid embolism (AFE) is a potentially lethal obstetric emergency characterized by sudden cardiovascular collapse, respiratory failure, and disseminated intravascular coagulation (DIC) [1]. This condition arises when amniotic fluid or fetal debris enters the maternal circulation, eliciting an anaphylactoid and obstructive reaction. Although rare, AFE accounts for 5% to 15% of maternal deaths in developed countries [2]. The pooled estimated incidence of AFE is approximately 1 in 15,200 deliveries in North America and 1 in 53,800 deliveries in Europe [2]. Key risk factors for AFE identified in large population-based cohort studies include cesarean section, placenta previa, eclampsia, abruptio placentae, and forceps delivery [3] [4]. DIC, a severe complication often associated with AFE, is characterized by coagulopathy and decreased fibrinogen levels, necessitating urgent intervention [5]. This report details two successful cases of patients who developed DIC following cesarean section, likely induced by AFE, and were managed using a bundle transfusion strategy prior to massive postpartum hemorrhage.

## 2. Case Report

### 2.1. Case 1

A 38-year-old $G_1P_0$ woman was admitted at $38^{+5}$ weeks of gestation for an elective cesarean section. Upon admission, the patient exhibited stable vital signs and no labor pains, with the fetus presenting in a cephalic position and in a normal state. On the day of surgery at 08:00 AM, she underwent a lower uterine segment cesarean section. Five minutes after the birth of a vigorous 3700 g infant, the patient experienced a choking cough, followed by an acute drop in oxygen saturation to 86%, accompanied by transient loss of consciousness. Her blood pressure fell transiently to 71/41 mmHg, raising suspicion for AFE. Other potential causes of acute cardiopulmonary instability, including pulmonary thromboembolism, anaphylaxis, air embolism, and anesthetic-related complications, were considered; however, the abrupt onset during delivery combined with early coagulopathy and hypofibrinogenemia supported a diagnosis of amniotic fluid embolism. She was administered intravenous dexamethasone (20 mg push and 40 mg drip), along with norepinephrine infusion and endotracheal intubation to stabilize her blood pressure and oxygen levels. Although the estimated blood loss during the cesarean section was 400 ml, which is within the normal range, bilateral ligation of the uterine arteries was performed, and uterotonics were administered due to signs of a slightly atonic uterus. The patient was subsequently transferred to the intensive care unit (ICU) for further monitoring and management, with blood tests for complete blood count and coagulation function initiated.

Two hours post-surgery (11:00 AM), the patient exhibited increasing vaginal bleeding, with an additional loss of 360 ml without clots. Laboratory tests indicated a mild decrease in hemoglobin from 116 g/L prior to surgery to 101 g/L, prolonged clotting time (international normalized ratio (INR) 1.21), and a significant reduction in fibrinogen level from 4.93 g/L before surgery to 1.34 g/L. D-dimer, indicative of fibrinolytic system activation, increased to 31.20 mg/L, respectively. Rapid fibrinogen decline of <1.5g/L, prolonged INR of >1.2 and increasing vaginal bleeding were considered indicative of early coagulopathy in suspected AFE and served as the trigger for simultaneous bundle transfusion prior to massive hemorrhage. A multidisciplinary team comprising obstetricians, anesthesiologists, hematologists, and ICU staff initiated aggressive supportive care, including a transfusion strategy comprising 1 g of tranexamic acid, 4 g of fibrinogen, 800 IU of human prothrombin complex, 10 units of cryoprecipitate, 800 ml of frozen plasma, and 8 units of packed red blood cells. In addition to uterotonics, digital subtraction angiography followed by uterine artery embolization was employed to manage the postpartum hemorrhage. Although these therapies have no function to treat coagulopathy, they help reduce the speed of bleeding loss, which gives us time to correct coagulation dysfunction, thereby halting the development of DIC.

By 17:00 PM, the fibrinogen level had increased to 2.23 g/L, and the D-dimer level decreased to 9.97 mg/L, with clotting time returning to the normal range (INR 1.02), indicating a successful rescue from coagulopathy. **Figure 1** summarizes the time course and progression of Case 1. The pregnant woman stayed in the ICU for one day, and extubation was conducted after her consciousness and blood pressure recovered to normal. She was discharged 5 days after birth, without later bleeding or thrombotic events.

## 2.2. Case 2

A 36-year-old $G_2P_1$ woman was admitted at $40^{+2}$ weeks of gestation, complaining of irregular uterine contractions without labor progress. She was admitted for induction of labor by COOK cervical dilatation balloon the previous night (18:00 PM), followed by artificial rupture of membranes (7:20 AM) and oxytocin intravenous infusion (9:20 AM) on the following day. At 18:00 PM the contraction interval was 3 minutes, and the fetal heart rate monitoring showed frequent early deceleration; the oxytocin intravenous infusion was stopped. The contraction remained strong, and the fetal head began to decrease. At 20:10 PM, the woman suddenly presented with trembling and a transient increase in heart rate of 170 bpm, but rapidly returned to 110 bpm, and the blood pressure was in the normal range. Considering the unstable state of the woman, intravenous dexamethasone 10mg push was given in case of AFE, and emergent cesarean section was arranged after informed consent. The surgery (21:00-21:50 PM) was smooth, and the blood loss was 300 mL, which was in the normal range. Since the coagulation test before surgery (20:50 PM) showed an increased level of D-dimer to 21.06 mg/L, it was

retested at 23:00 PM. At 1:07 AM, we were informed of the dramatic decrease in fibrinogen to 0.96 g/L and prolonged clotting time (INR 1.39). Vaginal bleeding without clot was presented, leading to a total of 545 mL blood loss after surgery. The marked hypofibrinogenemia state of <1g/L, prolonged INR of 1.2, and non-clotting bleeding were considered indicative of coagulopathy and served as the trigger for immediate bundle transfusion. A strategy of simultaneous transfusion, including 1 g of tranexamic acid, 4 g of fibrinogen, 800 IU of human prothrombin complex, 6 U of cryoprecipitate, 400 ml of fresh frozen plasma, and 2 units of packed red blood cells, was conducted to give aggressive support to the patient. Differential diagnoses such as uterine atony–related hemorrhage, placental abruption, sepsis, and thromboembolic events were evaluated, but were considered less likely given the preceding transient cardiopulmonary instability and disproportionate coagulation abnormalities. Uterine massage, oxytocin drip, and prostaglandins were also used to strengthen uterine contractions. At 4:30 AM, the fibrinogen increased to 2.15 g/L, and the clotting time returned to the normal range (INR 1.11), which indicated a relief of AFE. **Figure 1** presented the time course and progression of Case 2. The woman was discharged 4 days after cesarean section, without other complications including later bleeding or thrombotic events.

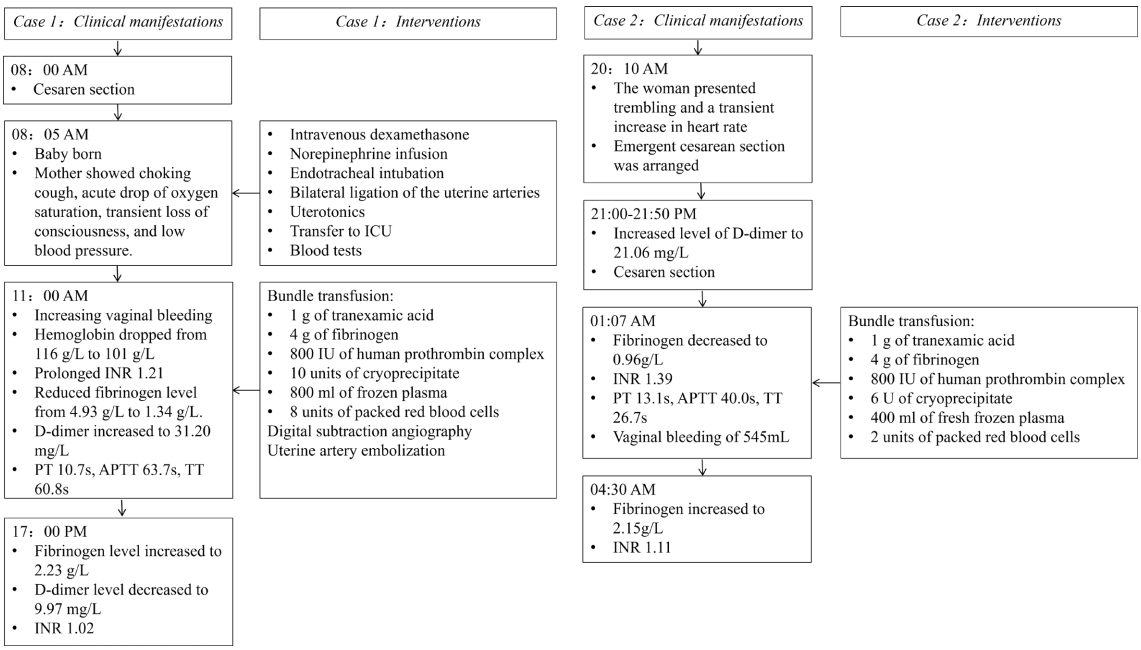


**Figure 1.** The time course and clinical progression of the two cases.

## 3. Discussion

In both cases, we observed a deterioration in laboratory indices prior to the onset of postpartum hemorrhage, indicative of non-overt DIC [6]. An aggressive bundle transfusion strategy, including tranexamic acid, fibrinogen, human prothrombin complex, cryoprecipitate, and component transfusion, was implemented and completed within a short time frame, appeared to support hemostasis and may have

contributed to preventing progression to DIC, thereby leading to severe hemorrhage, although causality cannot be established from these two cases. The Society for Maternal-Fetal Medicine recommends early aggressive management of clinical bleeding using standard massive transfusion protocols for AFE; however, recommendations regarding the management of non-overt DIC remain unclear [7] [8]. This case report provides a reference for transfusion strategies in non-overt DIC induced likely by AFE, but the risks associated with bundle transfusion warrant further evaluation in future studies.

Although there was a declining trend of maternal mortality rate due to AFE during the recent 20 years, the recent report of mortality rate is still as high as 17.7% [4] [9] [10]. Rapid awareness and early supportive treatment of DIC secondary to AFE are critical for successful rescue, since 40% of maternal deaths due to AFE might be prevented [11] [12]. The typical presentation of AFE includes a triad of sudden hypoxia and hypotension, followed in many cases by coagulopathy [8]. In fact, the clinical features of AFE are diverse. Clark *et al*. described the clinical features of 46 cases of AFE, among whom the most common presenting features were seizure or seizure-like activity (9/46, 30%), dyspnea (8/46, 27%), fetal bradycardia (5/46, 17%), and hypotension (4/46, 13%) [11]. Shivering-like tremor in the labor course with or without the use of epidural analgesia [13], fetal heart deceleration in the labor course, and epidural analgesia associated with a significant drop in blood pressure [14] were difficult for the obstetricians to distinguish. However, unexplained coagulopathy was strongly associated with AFE, with 83% of cases presenting with DIC [11]. The two cases of our report both presented prolonged clotting time and sharply decreased fibrinogen level, combined with typical or atypical cardiovascular dysfunction, and the treatment for AFE was initiated in a timely manner. Early detection and rescue of abnormal coagulation function is critical after the cardiovascular dysfunction is released.

## 4. Conclusion

In conclusion, the successful treatment of both cases is due to early detection of AFE and aggressive management of coagulopathy before clinical DIC presented. Early intervention with a comprehensive transfusion protocol, alongside standard obstetric measures, was feasible and clinically useful in these cases, though larger studies are needed to assess its efficacy. This report emphasizes the importance of early recognition and rapid treatment of abnormal coagulation markers in AFE, even before overt DIC develops. Early intervention played a crucial role in the patients' recovery. Further research is needed to refine management strategies for nonovert DIC in AFE cases and improve maternal outcomes.

## Authors' Contributions

Xiaotian Ni conceived and designed the case report. Xiaotian Ni and James Chang performed a detailed chart review and literature search. Xiaotian Ni and James Chang wrote the initial draft of the manuscript. Ming Liu provided substantial edits and

additions to the manuscript. All authors read and approved the final manuscript.

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## Patient Informed Consent

Patients described herein provided written informed consent to publish this case report.

## Availability of Data and Materials

The data were obtained from the patient's medical record and are not publicly available.

## Ethics Approval and Consent to Participate

This case report was deemed exempt by our Institutional Review Board.

## Conflicts of Interest

The authors declare no conflicts of interest.

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