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CASE REPORT

Amniotic Fluid Embolism Complicated with Hepatic Rupture: A Case Report

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Background: Amniotic fluid embolism (AFE) is a rare but highly fatal complication during delivery. It occurs when amniotic fluid suddenly enters the maternal circulation, causing acute multi-organ failure. Prompt recognition and management are crucial for improving survival rates.

Case Presentation: We reported a 37-year-old woman at 39 weeks gestation who developed AFE during labor induction. The patient presented with sudden cyanosis, dyspnea, and restlessness, followed by cardiac arrest during emergency cesarean section. Postoperative refractory postpartum hemorrhage (RPPH) necessitated multiple interventions, including hysterectomy and vascular embolization. Subsequent laparotomy revealed a subcapsular hematoma with linear tear in the left hepatic lobe, which was successfully repaired. The patient required massive transfusion and intensive care but eventually stabilized.

Conclusion: This case involved a life-threatening AFE complicated by cardiac arrest, RPPH, and traumatic liver rupture. The patient was successfully treated through multidisciplinary collaboration. It highlights three key aspects of critical maternal care: rapid recognition, damage control, and systemic resuscitation.

Keywords: amniotic fluid embolism, hepatic rupture, disseminated intravascular coagulation, post-partum hemorrhage, subcapsular hematoma

Introduction

Amniotic fluid embolism (AFE) represents one of the most catastrophic complications in obstetrics.[1] According to the Society for Maternal-Fetal Medicine guidelines, AFE diagnosis remains clinical, requiring sudden cardiorespiratory collapse with coagulopathy in laboring/postpartum women, while excluding other causes.[2] The condition occurs when amniotic fluid components enter the maternal circulation, triggering a cascade of pathophysiological events including pulmonary vasospasm, right heart failure, and systemic inflammatory response.[3,4] Emerging evidence suggests that systemic inflammation and coagulopathy may contribute to subcapsular hematoma formation. The liver, being a highly vascular organ, is particularly susceptible to ischemic injury during AFE, potentially leading to parenchymal damage, subcapsular hematoma, or even rupture.[5,6] The management of AFE requires a multidisciplinary approach, including prompt delivery of the fetus, correction of coagulopathy and supportive care.[7] Long-term outcomes depend on the extent of organ damage and the timeliness of interventions.[8]

This case report described a rare presentation of AFE complicated by hepatic rupture, highlighting the diagnostic challenges and therapeutic strategies in managing this life-threatening condition. This case provided valuable insights into the multidisciplinary collaboration required for managing such complex conditions.

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Case Presentation

A 37-year-old woman, gravida 5 para 1, at 39 weeks of gestation was admitted with minor vaginal bloody discharge. Her obstetric history included one full-term vaginal delivery and three induced abortions. Post-admission ultrasound examination suggested macrosomia (abdominal circumference 36.8 cm), and the patient opted for trial of vaginal delivery.

Labor induction with dinoprostone was initiated. Approximately 30 minutes after active labor commenced, the patient developed sudden cyanosis, dyspnea, and unresponsiveness. Vital signs showed blood pressure of 138/79 mmHg and oxygen saturation of 92%. Fetal bradycardia (80 bpm) was noted, prompting suspicion of AFE. Emergency cesarean section was performed within 20 minutes of symptom onset with bilateral uterine artery ligation and Hayman suture. A male neonate weighing 3560g was delivered with Apgar scores of 3 at 1 minute. The neonate was resuscitated and transferred to the neonatal unit. Cardiac arrest occurred intraoperatively and blood pressure could not be measured. Cardiopulmonary resuscitation (CPR) and electrical defibrillation were performed. During the cesarean section, the patient experienced a total blood loss of 8100 mL, accompanied by massive transfusion therapy with blood products.

Within 24 hours after cesarean section, due to refractory postpartum hemorrhage (RPPH), the patient underwent total abdominal hysterectomy, laparotomy, and vascular interventional procedures. Intraoperative exploration revealed diffuse oozing from multiple sites, and bedside ultrasound demonstrated expanding intra-abdominal fluid collections, consistent with ongoing hemorrhage. The intensive care unit (ICU), hepatobiliary surgery, interventional radiology and obstetrics teams suspected hepatic subcapsular hematoma rupture or hepatic laceration. Given the dual blood supply to the liver (hepatic artery and inferior vena cava), two options were considered: vascular embolization or repeat laparotomy. Due to the patient's poor tolerance for multiple surgeries, multidisciplinary discussion led to the decision to perform thoracic and abdominal angiography with left internal thoracic artery embolization and inferior vena cava angiography.

No active bleeding was seen on hepatic angiography, but intra-abdominal bleeding persisted. Soon afterwards, repeat laparotomy was performed. Intraoperative findings included diffuse oozing from multiple sites including the hysterectomy pedicles, a subcapsular hematoma on the diaphragmatic surface of the left hepatic lobe (segment III) with a 7.0*2.0 cm linear tear near the visceral surface, both consistent with DIC-associated coagulopathy. The tear was sutured, and the left hepatic artery was ligated. Postoperative CT scan of the upper abdomen were shown (Figure 1). The postoperative CT evaluation revealed critical findings of hepatic trauma repair, including heterogeneous parenchymal density suggesting residual hemorrhage, irregular hepatic contour consistent with surgical intervention, and evidence of both active contrast extravasation indicating ongoing bleeding as well as subcapsular hematoma formation.

Throughout the procedure, the patient developed disseminated intravascular coagulation (DIC) with cumulative blood loss exceeding 26000 mL. She received massive transfusion of blood products (red blood cells, plasma, cryoprecipitate, platelets, fibrinogen and albumin). Postpartum hemoglobin (HB), platelet (PLT), and fibrinogen (FIB) levels 48 hours

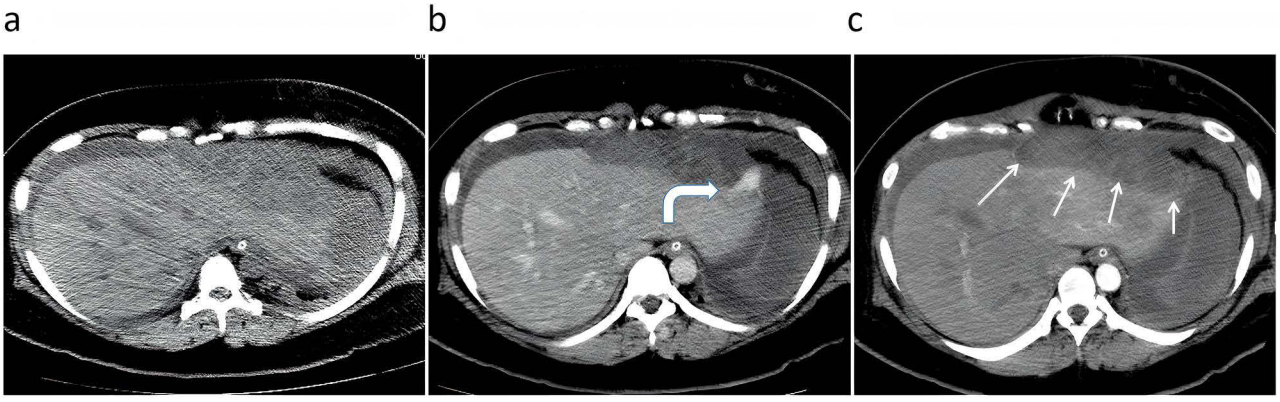

Figure 1 Postoperative CT scan of the upper abdomen after hepatic rupture repair. (**a**) Non-contrast CT image at the same level; (**b**) Enhanced CT image showing contrast extravasation (curved arrow); (**c**) Subcapsular hematoma (white arrow).
Abbreviation: CT, computed tomography.

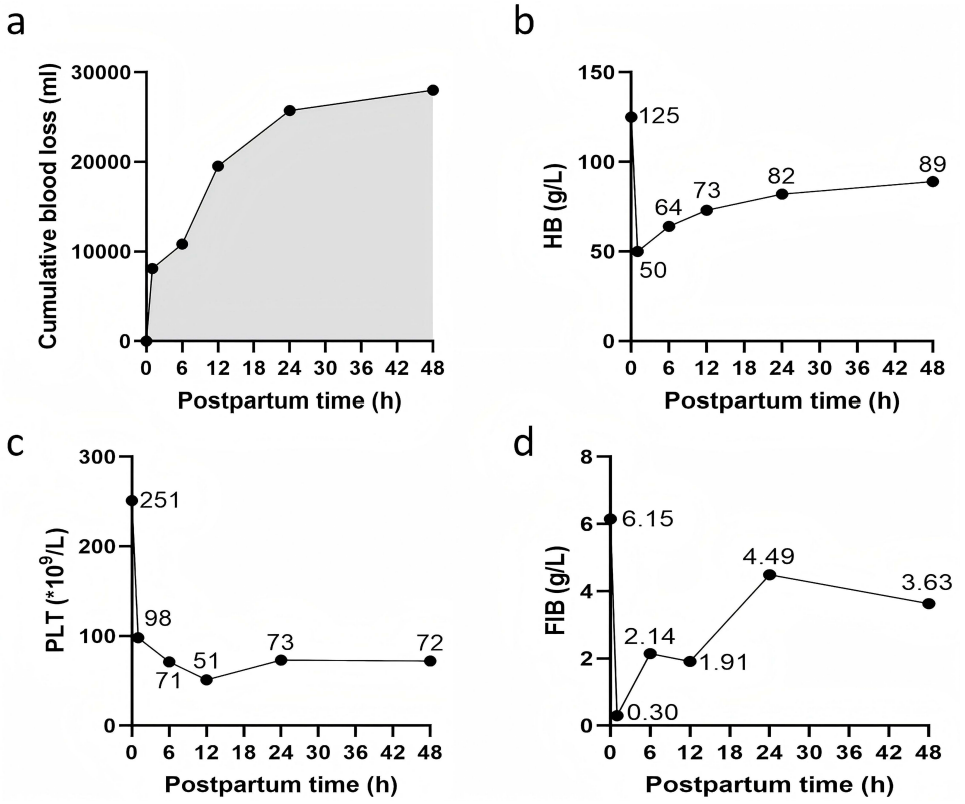

Figure 2 Dynamic changes in key clinical indicators within 48 hours postpartum (line graphs). (**a**) Cumulative blood loss; (**b**) Hemoglobin levels; (**c**) Platelet counts; (**d**) Fibrinogen levels. Data are derived from serial measurements of the index case.

after delivery were shown (Figure 2). The HB level reached a minimum of 50 g/L, the PLT count reached a minimum of $51*10^9$/L, and the FIB level reached a minimum of 0.3 g/L.

The patient was transferred to the ICU for sedation, analgesia, anti-infection, anti-allergy, anticoagulation, blood pressure control (130–150/80-100mmHg), diuresis, intracranial pressure reduction, corticosteroid pulse therapy, gastric protection, and liver protection. Serial CT imaging during recovery demonstrated gradual resolution of the subcapsular hematoma and stable surgical repair sites, with no evidence of rebleeding or new parenchymal injuries. Finally, bleeding was controlled and vital signs stabilized. She was transferred to a local hospital for further rehabilitation at 42 days postpartum. The clinical trajectory from labor induction to 48-hour postpartum were shown (Figure 3), encompassing symptoms, critical interventions, and laboratory parameters, with subsequent delineation of AFE-associated liver rupture pathogenesis.

Discussion

AFE is a rare but fatal obstetric complication. It usually occurred during labor, especially after membrane rupture, delivery, or induction. Its exact pathophysiology remains unclear, but it is believed to involve amniotic fluid, fetal cells, or placental components entering the maternal circulation and triggering an immune response.[9]

AFE diagnosis relies on clinical symptoms, particularly acute circulatory collapse during or after delivery. There are no special lab or imaging tests for AFE. But tests like blood gas analysis, electrocardiography, and coagulation studies can help check its severity.[10] This case developed hypoxia (SpO2 92%), hypotension (initial blood pressure 138/79 mmHg but rapidly deteriorating), and coagulopathy after induction, consistent with the classic triad of AFE.

The biphasic pathological model of AFE was evident in this case: an initial anaphylactoid reaction causing pulmonary artery spasm and right heart failure (manifested as sudden dyspnea and cyanosis), followed by systemic inflammatory response syndrome and DIC triggered by amniotic fluid procoagulants.[11,12] Post-CPR hypertension (130–150/80-

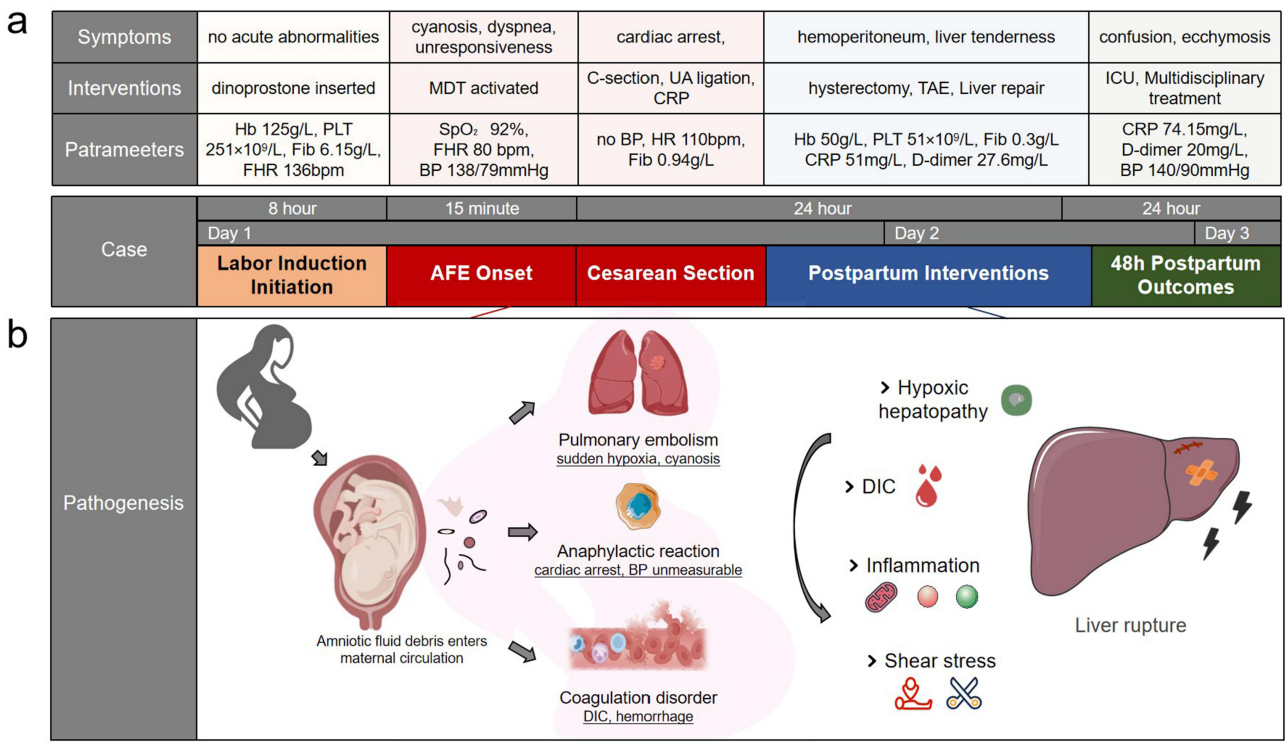

Figure 3 Clinical timeline and pathogenic mechanisms of AFE complicated by hepatic rupture. (**a**) Integrated timeline documenting symptom onset, therapeutic interventions, vital signs and parameters. Time intervals reflect critical phases from labor induction to 48-hour postpartum monitoring. (**b**) Proposed pathophysiological sequence of AFE leading to hepatic rupture through combined effects of systemic hypoxia, coagulopathy, inflammation, and mechanical stress on the compromised liver architecture.

Abbreviation: AFE, amniotic fluid embolism.

100 mmHg) may reflect cerebral hypoxia-induced sympathetic overactivation or vasopressor effects. This paradoxical hypertension was rare in AFE and warranted caution for cerebral edema and posterior reversible encephalopathy syndrome. Recent advances in AFE risk stratification highlight the importance of recognizing high-risk scenarios, particularly when placental abnormalities or coagulopathic predisposition coexist. While universal risk assessment remains challenging due to AFE's rarity, heightened vigilance is warranted in cases with abrupt hemodynamic collapse and unexplained DIC, as these may signal impending multi-organ involvement.[13]

The mechanism of hepatic rupture secondary to AFE warranted in depth exploration. Key differential diagnoses such as HELLP syndrome and uterine rupture were systematically excluded. HELLP syndrome was ruled out based on the absence of hemolysis and elevated liver enzymes (ALT/AST levels remained stable pre-DIC). Uterinary rupture was deemed unlikely given the lack of fetal distress prior to AFE onset, no evidence of uterine dehiscence during surgical exploration, and the temporal sequence of symptoms (classic AFE triad preceding hepatic rupture). These distinctions reinforce the diagnosis of AFE-related hepatic injury. In this case, we hypothesized that spontaneous rupture was highly likely. AFE-induced hypoxia and shock damaged the liver through ischemic injury from poor perfusion, resulting in sinusoidal endothelial swelling and hepatocyte necrosis.[11] This compromised both the liver's metabolic function and structural stability, reducing its mechanical resilience. Concurrently, the systemic inflammatory response triggered by AFE flooded the body with inflammatory mediators and cytokines. These substances increased vascular permeability,[14] facilitating the formation of subcapsular hematomas. Expanding hematomas increased intracapsular pressure, while concurrent DIC worsened the condition. DIC disrupted the normal coagulation–anticoagulation balance, leading to abnormal bleeding tendencies.[15] During the patient's course, with factors such as multiple surgeries and fluctuations in intra-abdominal pressure, the subcapsular hematoma in the liver was under continuous stress. The left hepatic lobe, especially at the junction of the diaphragmatic and visceral surfaces where the hematoma and tear were found in

this case, was more vulnerable due to the high shear stress in this area. All these factors combined likely caused the subcapsular hematoma to rupture spontaneously. This spontaneous rupture hypothesis is consistent with the overall pathophysiological process of AFE and the patient's clinical manifestations, further emphasizing the complexity and severity of AFE-related hepatic damage.

AFE management primarily involves supportive care and emergency surgical intervention.[3,7,9] This case highlighted the complexity of AFE management and the importance of multidisciplinary collaboration. First, prompt delivery is crucial to prevent further entry of amniotic fluid into the maternal circulation. Fetal bradycardia (80 bpm) indicated fetal distress, forming a vicious cycle with maternal circulatory collapse and necessitating emergency cesarean section. Second, immediate CPR and defibrillation are critical in AFE-induced cardiac arrest.[16] Third, AFE often causes severe postpartum hemorrhage, which is difficult to control with conventional methods. This case employed a damage control surgery approach: initial uterine artery ligation and Hayman suture, followed by total hysterectomy, and finally vascular embolization and hepatic repair. This stepwise approach aligns with the FIGO guidelines for postpartum hemorrhage.[17] Additionally, AFE often accompanies DIC, posing significant treatment challenges. Massive blood product transfusion and supportive care helped correct coagulopathy and stabilize the patient.[18] Finally, hepatic rupture, a common complication of AFE multi-organ damage, was effectively managed through timely imaging and surgical intervention. As an important metabolic and detoxifying organ of the human body, the repair and functional recovery of the liver after rupture were key links in the treatment of this case. After repairing the liver rupture, closely monitoring liver function indicators and appropriate liver-protecting treatment should be given to promote the regeneration of hepatocytes.

Multidisciplinary collaboration was vital for success. Specialists combined expertise to create tailored treatment plans. Obstetricians, familiar with childbirth - related situations, quickly decided to perform a cesarean section to terminate the pregnancy, reducing the risk of further entry of amniotic fluid into the maternal circulation. ICU doctors, proficient in maintaining the stability of vital signs and supporting multiple organ functions, played a key role when the patient experienced critical conditions such as cardiac arrest and shock. Interventional radiologists attempted to control bleeding points through precise angiography and embolization techniques. Hepatobiliary surgeons were responsible for the repair surgery of the liver rupture. This model of close cooperation among multiple team reflects the advantages of modern medicine in dealing with complex cases and provides an example for improving the success rate of treating similar cases.

In conclusion, this case highlighted the life-threatening nature of AFE and its potential for multi-organ involvement. The successful outcome was achieved through timely recognition, multidisciplinary collaboration, and systematic implementation of damage control strategies. It underscored the importance of preparedness and coordinated team response in managing complex obstetric emergencies.

Abbreviations

AFE, amniotic fluid embolism; CPR, cardiopulmonary resuscitation; RPPH, refractory postpartum hemorrhage; ICU, intensive care unit; DIC, disseminated intravascular coagulation.

Data Sharing Statement

Data is provided within the manuscript.

Ethics Approval and Informed Consent

The study was approved by the Ethics Committee of Fujian Maternity and Child Health Hospital, Fuzhou, China. Informed consent was obtained from the patient for publication of clinical details. Institutional approval was not required to release case details. This study complies with the Declaration of Helsinki.

Consent for Publication

Written informed consent for publication of the clinical details were obtained from the patient.

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Disclosure

The authors have declared no competing interests in this work.

References

1. Mazza GR, Youssefzadeh AC, Klar M, et al. Association of pregnancy characteristics and maternal mortality with amniotic fluid embolism. *JAMA Network Open*. 2022;5(11):e2242842. doi:10.1001/jamanetworkopen.2022.42842
2. Pacheco LD, Saade G, Hankins GD, Clark SL. Amniotic fluid embolism: diagnosis and management. *Am J Obstet Gynecol*. 2016;215(2):B16–24. doi:10.1016/j.ajog.2016.03.012
3. Combs CA, Montgomery DM, Toner LE, Dildy GA; Patient S, Quality Committee Sf M-FMEasso. Society for maternal-fetal medicine special statement: checklist for initial management of amniotic fluid embolism. *Am J Obstet Gynecol*. 2021;224(4):B29–B32. doi:10.1016/j.ajog.2021.01.001
4. Soecki G, Hlatchuk EC, Raymundo CT, et al. Heterotopic pregnancy and amniotic embolism: a case report. *BMC Pregnancy Childbirth*. 2024;24(1):752. doi:10.1186/s12884-024-06969-z
5. McBride SA, Small R, Hurd K, Claassens S. Preeclampsia and HELLP syndrome complicated by subcapsular liver hematoma and rupture. *CMAJ*. 2023;195(45):E1543–E1545. doi:10.1503/cmaj.230851
6. Chen W, Qi J, Shang Y, Ren L, Guo Y. Amniotic fluid embolism and spontaneous hepatic rupture during uncomplicated pregnancy: a case report and literature review. *J Matern Fetal Neonatal Med*. 2020;33(10):1759–1766. doi:10.1080/14767058.2018.1526915
7. Trieu NHK, Nguyen NN, Pham HM, Huynh DQ, Mai AT. Extracorporeal membrane oxygenation in amniotic fluid embolism: a systematic review of case reports. *ASAIO J*. 2025;71(2):143–148. doi:10.1097/MAT.0000000000002269
8. Qian J, Wolfson C, Kramer B, Creanga AA. Insights from preventability assessments across 42 state and city maternal mortality reviews in the United States. *Am J Obstet Gynecol*. 2024;232(4):394–e1. doi:10.1016/j.ajog.2024.08.030
9. Feng Y, Yang HX. Interpretation of Chinese expert consensus on diagnosis and management of amniotic fluid embolism. *Chin Med J*. 2020;133(14):1719–1721. doi:10.1097/CM9.0000000000000886
10. Wiseman D, Simard C, Yang SS, Koolian M, Abenhaim HA, Lipes J. Echocardiography findings in amniotic fluid embolism: a systematic review of the literature [Observations echocardiographiques lors d'une embolie de liquide amniotique: une revue systematique de la litterature]. *Can J Anaesth*. 2023;70(1):151–160. doi:10.1007/s12630-022-02343-9
11. Lao TT. Acute respiratory distress and amniotic fluid embolism in pregnancy. *Best Pract Res Clin Obstet Gynaecol*. 2022;85(Pt A):83–95. doi:10.1016/j.bpobgyn.2022.06.004
12. Chen KB, Chang SS, Tseng YL, et al. Amniotic fluid induces platelet-neutrophil aggregation and neutrophil activation. *Am J Obstet Gynecol*. 2013;208(4):318e1–7. doi:10.1016/j.ajog.2013.01.012
13. Cavoretto PI, Rovere-Querini P, Candiani M. Toward risk assessment for amniotic fluid embolisms. *JAMA Network Open*. 2022;5(11):e2242850. doi:10.1001/jamanetworkopen.2022.42850
14. Kobayashi H. Amniotic fluid embolism: anaphylactic reactions with idiosyncratic adverse response. *Obstet Gynecol Surv*. 2015;70(8):511–517. doi:10.1097/OGX.0000000000000197
15. Erez O, Mastrolia SA, Thachil J. Disseminated intravascular coagulation in pregnancy: insights in pathophysiology, diagnosis and management. *Am J Obstet Gynecol*. 2015;213(4):452–463. doi:10.1016/j.ajog.2015.03.054
16. He F, Li RR, Liu PS, Yang YL, Huang CJ, Chen DJ. Maternal cardiac arrest: a retrospective analysis. *BJOG*. 2021;128(7):1200–1205. doi:10.1111/1471-0528.16625
17. Group FPTW; Begum F, Beyeza J, Beyeza J, et al. FIGO and the international confederation of midwives endorse WHO guidelines on prevention and treatment of postpartum hemorrhage. *Int J Gynaecol Obstet*. 2022;158 Suppl 1(Suppl 1):6–10. doi:10.1002/ijgo.14199
18. Diguisto C, Baker E, Stanworth S, Collins PW, Collis RE, Knight M. Management and outcomes of women with low fibrinogen concentration during pregnancy or immediately postpartum: a UK national population-based cohort study. *Acta Obstet Gynecol Scand*. 2024;103(7):1339–1347. doi:10.1111/aogs.14828