Clinics of Surgery

Case Report ISSN: 2638-1451 | Volume 7

# Inhalational Sevoflurane May Contribute to Successful Resuscitation of Amniotic Fluid Embolism: A Case Report

**Chen S[1], Li X[1], Guo N[1], Li X[1], Teng B[2*] and Liu D[1*]**

[1]Department of Anesthesiology, Third Affiliated Hospital of Sun Yat-sen University, No.600 Tianhe Road, Guangzhou 510630, China

[2]Department of Obstetrics, Third Affiliated Hospital of Sun Yat-sen University, No.600 Tianhe Road, Guangzhou 510630, China

**[*]Corresponding author:**

Dezhao Liu and Benqi Teng,
Department of Anesthesiology, The Third Affiliated Hospital of Sun Yat-sen University, Department of Obstetrics, The Third Affiliated Hospital of Sun Yat-sen University No.600 Tianhe Road, Guangzhou 510630, China, Tel: +86-15360884591, +86-13711275716,
E-mail: sumsldz@126.com, tengbq@mail.sysu.edu.cn

Received: 11 Jan 2022
Accepted: 21 Jan 2022
Published: 28 Jan 2022
J Short Name: COS



**Citation:**

Liu D and Teng B, Inhalational Sevoflurane May Contribute to Successful Resuscitation of Amniotic Fluid Embolism: A Case Report. Clin Surg. 2022; 7(1): 1-5

**Keywords**:

Amniotic fluid embolism; Cesarean delivery; Inhalation induction; Obstetric emergency; Sevoflurane

**Abbreviations:**

AFE: amniotic fluid embolism; SEV: sevoflurane

## 1. Abstract

**1.1. Background:** Amniotic fluid embolism (AFE) is a rare but catastrophic obstetric emergency with a high mortality rate. Currently, the precise pathophysiology of AFE is unclear and there is still no specific therapy to treat it. Here, we reported a case of AFE in which inhalational sevoflurane (SEV) induction was used during cesarean section and discussed the potential role of inhalation induction in AFE anesthesia management.

**1.2. Case presentation:** A 34-year-old woman developed severe AFE during vaginal delivery. Owing to her peripheral circulatory collapse and failure to quick establishment of venous access, inhalational SEV induction was used during cesarean delivery. We found that SEV induction saved precious rescue time, and may relieve pulmonary vasospasm and pulmonary hypertension. The well outcome of this patient indicated that SEV inhalation induction might be considered as an alternative approach in early respiratory support and rescuing critically ill AFE patient.

**1.3. Conclusions:** To our knowledge, this is the first case reported of inhalational SEV induction in an AFE patient. It indicated that SEV inhalation induction might be considered as an alternative approach in early respiratory support and rescuing severe AFE patient with peripheral circulatory collapse or failure to quick establishment of venous access.

## 2. Introduction

AFE is a rare, unpredictable, and potentially catastrophic obstetric emergency that occurs when amniotic fluid or fetal material enters the maternal bloodstream and triggers a severe reaction [1]. The reported incidence of AFE is 0.8-1.8 per 100,000 deliveries with a mortality rate as high as 41% [2,3] due to the unclear pathophysiology and lack of specific therapy for the condition. Currently, there is no international consensus regarding diagnostic criteria for AFE, and it is still a diagnosis of exclusion dependent on clinical presentation [4]. AFE might develop suddenly during labor, delivery, or immediately postpartum, and proceed rapidly within a few minutes. The presentation of AFE is often catastrophic, and it can present as sudden hypoxemia, impaired consciousness, massive bleeding or coagulopathy, pulmonary edema or right ventricular failure, and cardiovascular collapse or even cardiac arrest [5,6].

A mother and fetus who survive an AFE may suffer severe neurological damage or brain death [7,8]. Early recognition and aggressive rescue strategies can enhance both maternal and perinatal outcomes. Here, we describe a case of suspected AFE in which inhalational SEV induction was used during a cesarean section and discuss the potential role of inhalation induction in AFE anesthesia management.

## 3. Case Presentation

A 34-year-old woman (BMI of 29.2), gravida 3 (0-0-2-0), was admitted to the obstetrical department at $41^{+1}$ weeks gestation. She had a spontaneous abortion 3 years prior and an artificial abortion 1 year earlier owing to fetal abnormality. Fortunately, she had an

uneventful course of this pregnancy and no other previous medical history. The patient met the criteria for vaginal delivery after obstetricians' assessment. A double balloon catheter and oxytocin (20 units/day, intravenously) were used to induce labor as the baby was overdue. Within 25 h of admission, the patient experienced an artificial rupture of the membranes, the amniotic fluid was clear, and the cervix was dilated to 2 cm. Intravenous infusion of oxytocin was continued. After 1.5 h of rupturing the membranes, the patient suddenly complained of severe abdominal pain, shortness of breath, and dysphoria. Within seconds, she proceeded to exhibit unconsciousness, generalized spasm, and cyanosis. Physical examination identified a stiff abdomen and tetanic contraction of uterus. Meanwhile, her blood pressure dropped from 123/78 to 90/52 mmHg, heart rate increased from 76 to 112 beats/min, and oxygen saturation dropped to 85%. The fetal heart rate declined from a baseline of 142 to 80 beats/min. The patient's airway was immediately supported with oxygen by facemask, and spasm activity ceased within 60 s with 5 g intravenous magnesium sulfate. Based on the rapidly progressing clinical symptoms, AFE was highly suspected based on Clark's criteria [9,10]. The patient was immediately transferred to the operating room for emergency cesarean section within 8 min.

The patient underwent cesarean delivery under general anesthesia with inhalational SEV induction owing to peripheral circulatory collapse and failure to quick establishment of venous access. In order to avoid aspiration, our team placed a gastric tube within 1min before induction and compressed the esophagus with the Sellick method. SEV was initially administered at a concentration of 8% (6 L/min 100% oxygen) and gradually reduced to 2-3% when the minimum alveolar concentration reached 1.3. After 2 min, anesthesia induction was completed, and the operation started. At the same time, a new peripheral intravenous catheter was inserted and 80 mg propofol, 70 mg rocuronium, and 40 μg remifentanil were rapidly infused. An endotracheal tube was placed under visual laryngoscopy. Auscultation of bilateral lungs was grossly normal, and the peak airway pressure was 20-22 cm $H_2O$ with a volume-controlled-mode (tidal volume of 500 mL). A radial arterial line and a central venous line were placed with ultrasound guided following intubation.

A 2800-g, 47-cm infant was delivered 3 min after the operation began. The 1- and 5-min Apgar scores were 3 and 6 points, respectively. The infant was resuscitated with tracheal intubation due to severe asphyxia. The 10-min Apgar score was 8 points, and the infant was subsequently transferred to the neonatal intensive care unit (ICU). Immediately after delivery, the maternal heart rate increased to 125 beats/min, oxygen saturation decreased to 93%, and blood pressure reduced to 65/33 mmHg. A norepinephrine infusion starting at 1 μg/kg/h was titrated to maintain blood pressures of approximately 95-100/55-60 mmHg. SEV (1.5-2%), remifentanil (2-3 μg/kg/h), and cisatracurium (8 mg/h) were added to deepen the anesthetic effect as the hemodynamics stabilized. Uterine atony developed within minutes after the expulsion of the placenta, and ongoing bleeding and unclotted blood flowed from the surgical site and uterine cavity. Multiple doses of intramuscular carboprost tromethamine and intravenous oxytocin (40 units) were used to promote postpartum uterine contractions. Initial laboratory test results were extremely abnormal with a PT of 27.6s, APTT of 72.3s, fibrinogen of 0.6g/L, hemoglobin of 100g/L, and PLT $45\times10^9$/L (Figure 1). The hemoglobin/fibrinogen (H/F) ratio in this patient was 166.67 (100/0.62) one hour after symptom emergence (Figure 1B). Patients with a high H/F ratio of more than 100 may develop not only postpartum hemorrhage with coagulopathy, but also cardiopulmonary failure [10]. The possibility of AFE, which was previously considered, was now strongly suspected. Rapid transfusion of packed red cells, fresh frozen plasma, and large amounts of colloid and crystalline solutions were initiated. Meanwhile, the patient was evaluated by the obstetricians for B-Lynch suture or even hysterotomy if the bleeding could not be controlled. The estimated surgical blood loss was 2200 mL. She received a total of 6000 mL rehydration, including 10 units of packed erythrocytes, 2000 mL of fresh frozen plasma, 1 unit of platelets, and 20 units of cryoprecipitate. In addition, 80 mg methylprednisolone and 1 g tranexamic acid were intravenously infused during the surgery. Her arterial blood gases (ABS) showed moderate metabolic acidosis (PH 7.22, BE -8.1) and hypoxemia ($PaO_2$ 128 mmHg) in the early stage after onset. Arterial pressure, central venous pressure, and urinary output (about 6 mL/kg/h) were satisfactory during the operation. At 4 h of continued resuscitation, the blood loss was minimal due to the application of gauze packing in combination with an intrauterine balloon tamponade. Her postoperative bedside cardiac/abdominal ultrasound, serum myocardial enzyme, ABS and head/abdomen CT scans were all normal, while her chest CT showed scattered bilateral segmental pulmonary arteries and subsegmental pulmonary arteries embolism (Figure 2A). The patient was transferred to the ICU after CT scans. Over the next 24 h, she was weaned from ventilator support and extubated, her coagulopathy gradually resolved (Figure 1), and her cardiovascular status stabilized. Five days after surgery, a second chest CT scan indicated that the original pulmonary lesions achieved full recovery without vascular intervention therapy (Figure 2B). The patient was discharged home 14 days after surgery with a healthy newborn. After discharge, the patient was followed up at 42 days with no apparent neurologic deficits. All personal information was anonymized, and we obtained the patient's informed consent for publication of her clinical data.

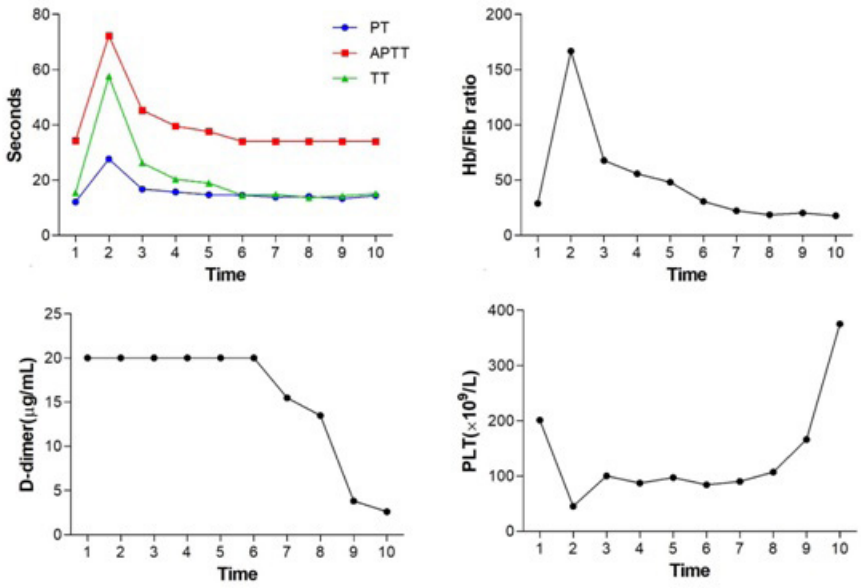


**Figure 1:** Changes in main indicators of coagulation function. (A) Prothrombin time (PT), activated partial thromboplastin time (APTT), and thrombin time (TT) during symptom onset. (B) Hemoglobin/fibrinogen (H/F) ratio in this patient. (C) D-dimer in this patient. (D) Platelet (PLT) count in this patient. The patient started to receive blood transfusion 1 h after symptom onset. "Time 1" means "2 days before surgery"; "Time 2" means "1 h after symptom onset"; "Time 3" means "5 h after symptom onset"; "Time 4" means "8 h after symptom onset"; "Time 5" means "12 h after symptom onset"; "Time 6" means "19 h after symptom onset"; "Time 7" h "2 days after surgery"; "Time 8" means "3 days after surgery"; "Time 9" means "4 days after surgery" and "Time 10" means "7 days after surgery".

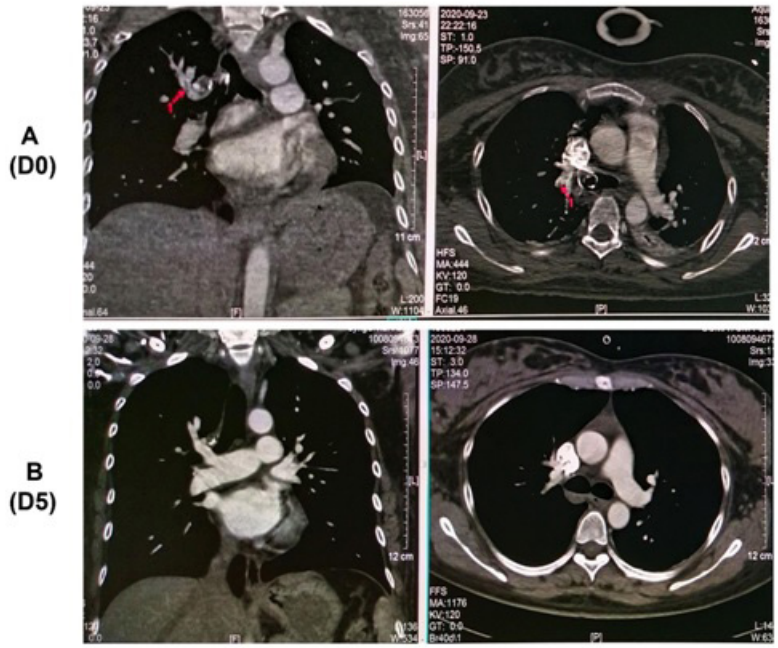


**Figure 2:** Pre- and post-contrast chest computed tomography (CT) scans showing pulmonary lesions. (A) Immediate postoperative (D0) CT scans showing scattered segmental pulmonary artery embolism (red arrow). (B) CT scans on postoperative day 5 (D5) indicating that the pulmonary lesions had fully resolved.

## 4. Discussion

According to the Japanese criteria for scientific reporting of AFE[10], as well as this patient's demographic features and clinical evolution, we can likely diagnose this case as AFE. Firstly, this patient had several potential risk factors associated with AFE, including advanced maternal age (34-years-old), late pregnancy stage ($41^{+1}$ weeks), third pregnancy with two previous abnormal pregnancies, and induction of labor with oxytocin. Secondly, she showed clinical signs associated with AFE, including sudden shortness of breath, severe abdominal pain, and fetal distress during induction of labor, which quickly proceeded to hypoxia, unconsciousness, hypotension, and consumptive coagulopathy, indicated by a high hemoglobin/fibrinogen ratio (100/0.6=166.67). This finding was consistent with a case-control study, which showed that a high H/F ratio was a promising clinical marker for the earlier assessment of AFE-related consumptive coagulopathy [10]. Thirdly, the differential diagnoses were excluded from hemorrhage, pulmonary thromboembolism, anaphylaxis, eclampsia, and neurologic diseases.

The pathophysiological mechanism of AFE has not been fully characterized yet. Traditionally, AFE was assumed to occur only during a breach in the maternal-fetal barrier. However, the usual absence of pulmonary vessel mechanical obstruction, a great variability in the clinical course, and the lack of suitable animal models that fully reproduce the disease suggest that physical obstruction of the maternal pulmonary vessel is not the main mechanism of AFE. Funk et al. demonstrated that mild to moderate pulmonary artery pressure is thought to be caused by intrinsic pulmonary artery spasms [11]. Meanwhile, the current available evidence suggests that the hemodynamic response to AFE is biphasic, with initial increases in pulmonary arterial pressure and right ventricular failure followed by left ventricular failure [12]. Currently, the clinical treatment of AFE mainly focuses on symptomatic and supportive care, including control of hemorrhage, correction of coagulopathy, cardiovascular support, and preventing subsequent organ failure [13]. Rapid remission of pulmonary vasoconstriction and maintenance of circulatory homeostasis are the most important aspects of AFE salvage and therapy.

In our case, the patient exhibited chest tightness, dyspnea, and hypoxemia before surgery, we suspected the development of pulmonary vasospasm or pulmonary hypertension. We performed fast and successful inhalational induction in this patient due to circulation instability and failure to establishment of venous access. Surprisingly, we found that inhalational SEV induction significantly relieved her pulmonary vasospasm and pulmonary hypertension. SEV acts as a potent bronchodilator via reduction in parasympathetic nervous tone and inhibition of the voltage-dependent calcium and potassium channels in the bronchial smooth muscle [14]. In a rat model of pulmonary arterial hypertension, SEV inhalation improved right ventricular function by upregulating NO release [15]. In our case, we did not institute pulmonary artery catheterization for proper hemodynamic assessment and management due to the emergency condition, so it was difficult to observe the change in pulmonary arterial pressure. The patient was extubated on postoperative day 1; and pulmonary embolism disappeared without vascular intervention therapy 5 days after surgery. Therefore, the well outcome of this patient indicated that SEV induction may play an important role in the AFE cesarean section. SEV induction not only saved precious time, but also might relieve pulmonary vasospasm and pulmonary hypertension. The potential mechanism of early SEV inhalation induction to improve the pulmonary function and overall prognosis of AFE patients still needs further research. This case indicated that, except for intravenous induction, SEV inhalation induction might be considered as an alternative approach in rescuing AFE patient with peripheral circulatory collapse or failure to quick establishment of venous access.

## 5. Declaration

### 5.1. Ethics approval and consent to participate

Written informed consent for publication of the clinical details and/or clinical images was obtained from the patient. A copy of the consent form is available for review by the Editor of this journal. An ethics board review was not required. And the case reporting adheres to CARE guidelines.

## 6. Authors' Contributions

Sufang Chen and Xiaoyun Li contributed equally to this article. Data collection: Sufang Chen, Na Guo and Xiang Li. Data analysis: all authors. Writing first manuscript: Sufang Chen, Benqi Teng and Xiaoyun Li. Modify the manuscript: Xiaoyun Li and Dezhao Liu.

## 7. Competing Interests

The authors declare no competing interests.

## 8. Acknowledgement

Not applicable.

## 9. Funding

None.

## References

1. Haftel A, Chowdhury YS. Amniotic Fluid Embolism. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2021.
2. Fitzpatrick KE, Akker TVD, Bloemenkamp KWM. Risk factors, management, and outcomes of amniotic fluid embolism: A multicountry, population-based cohort and nested case-control study. PLoS Med. 2019; 16(11): e1002962.
3. Fitzpatrick KE, Tuffnell D, Kurinczuk JJ. Incidence, risk factors, management and outcomes of amniotic-fluid embolism: a population-based cohort and nested case-control study. BJOG. 2016; 123(1): 100-109.
4. Ponzio-Klijanienko A, Vincent-Rohfritsch A, Girault A, et al. Eval-

uation of the 4 diagnosis criteria proposed by the SMFM and the AFE foundation for amniotic fluid embolism in a monocentric population. J Gynecol Obstet Hum Reprod. 2020; 49(9): 101821.

5. Sultan P, Seligman K, Carvalho B. Amniotic fluid embolism: update and review. Curr Opin Anaesthesiol. 2016; 29(3): 288-296.
6. Shen F, Wang L, Yang W. From appearance to essence: 10 years review of atypical amniotic fluid embolism. Arch Gynecol Obstet. 2016; 293(2): 329-334.
7. Spiliopoulos M, Puri I, Jain NJ. Amniotic fluid embolism-risk factors, maternal and neonatal outcomes. J Matern Fetal Neonatal Med. 2009; 22(5): 439-444.
8. Kramer MS, Rouleau J, Liu S. Amniotic fluid embolism: incidence, risk factors, and impact on perinatal outcome. BJOG. 2012; 119(7): 874-879.
9. Society for Maternal-Fetal Medicine (SMFM). Pacheco LD, Saade G, Hankins GD, Clark SL. Amniotic fluid embolism: diagnosis and management. Am J Obstet Gynecol. 2016; 215(2): B16-B24.
10. Oda T, Tamura N, Ide R. Consumptive Coagulopathy Involving Amniotic Fluid Embolism: The Importance of Earlier Assessments for Interventions in Critical Care. Crit Care Med. 2020; 48(12): e1251-e1259.
11. Funk M, Damron A, Bandi V. Pulmonary vascular obstruction by squamous cells is not involved in amniotic fluid embolism. Am J Obstet Gynecol. 2018; 218(4): 460-461.
12. Clark SL, Cotton DB, Gonik B. Central hemodynamic alterations in amniotic fluid embolism. Am J Obstet Gynecol. 1988 ;158(5): 1124-1126.
13. Pacheco LD, Clark SL, Klassen M, et al. Amniotic fluid embolism: principles of early clinical management. Am J Obstet Gynecol. 2020; 222(1): 48-52.
14. Regli A, von Ungern-Sternberg BS. Anesthesia and ventilation strategies in children with asthma: part I-preoperative assessment. Curr Opin Anaesthesiology. 2014; 27(3): 288-294.
15. Qin G, Luo H, Yin X. Effects of Sevoflurane on Hemodynamics and Inducible Nitric Oxide Synthase/Soluble Guanylate Cyclase Signaling Pathway in a Rat Model of Pulmonary Arterial Hypertension. Anesth Analg. 2017; 125(1): 184-189.