Case Report

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Survival after an amniotic fluid embolism following various treatments conducts. a case report

Abstract

Background: Amniotic fluid embolism (AFE) is a rare complication of pregnancy whose physiopathology is not completely known and still not frequently remembered by intensive care physicians and obstetricians. Being extremely serious with sudden onset with hypotension or cardiorespiratory collapse, respiratory difficulty and coagulopathy. The objective of the case report is to describe a serious case that, with precise procedures, was discharged from hospital on the 13th day after 11 days in an ICU.

Case report: Female patient, 34 years old, pregnancy III, birth by cesarean II, at 39 weeks of gestational age, scheduled for elective cesarean section. Normal evolution during pregnancy. Following the delivery of her baby, the mother suffered a cardiocirculatory and respiratory collapse. Following a protracted treatment, transesophageal echocardiography demonstrated evidence of acute pulmonary hypertension, with an empty left ventricle and an over-distended right ventricle. Six hours after the first symptoms with a new condition, she was taken to the ICU where she recovered after resuscitation maneuvers. On the 7th day of mechanical ventilation, the patient was extubated without complications. On the 10th day of hospitalization, a new chest CT scan was performed showing the evolution of the pulmonary condition.

Conclusion: AFE is a rare but serious condition with high mortality and morbidity rates. Sudden cardiovascular collapse is induced by hypoxemia and hypotension Early detection, diagnosis, and treatment of AFE are essential to avoiding fatal result. The management of AFE involves a multidisciplinary team.

Keywords: Spinal anesthesia, Cesarean Section, Amniotic Fluid, Embolism, Pregnancy, Acute Respiratory Distress Syndrome, Electrical Impedance Tomography, Titration of Positive End-Expiratory Pressure

Key points

• Amniotic fluid embolism is a rare but potentially fatal complication of pregnancy associated with high morbidity and mortality.

• The pathophysiology of AEF probably involves a response like systemic inflammatory response syndrome, whereby fetal materials enter the maternal bloodstream, resulting in pulmonary vasoconstriction and acute pulmonary hypertension with right heart failure.

• AFE is catastrophic emergency with sudden onset and rapid progression.

• The management of AFE involves a multidisciplinary team and complementary exams that are important for diagnosis and treatment, leading factors to successful maternal and fetal outcome.

• High index of suspicion of AFE should be considered in any intrapartum or postpartum collapse where the obvious cause of collapse is not identified.

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Humberto de Sousa Cândido, MD,[1] Luiz Eduardo Imbelloni, MD, PhD,[2] Angelo Antônio Gomes de Carvalho, MD,[3] Dilene Morais Barbosa Gisch, MD,[4] Rogério Silva Monteiro, MD,[5] Stela Nunes Menezes, MD,[6] Tolomeu A. A. Casali, MD, PhD,[7] Robson de Brito Oliveira, MD[8]

[1]Anesthesiologist of the Hospital Cristo Redentor, GO and Hospital Albert Einstein, Brazil
[2]Anesthesiologist and Researcher of Various Hospital, Rio de Janeiro, Brazil
[3]ICU Intensivist of the Hospital Albert Einstein, GO and Hospital Albert Einstein, Brazil
[4]Gynecologist, Obstetrician and Colposcopist of the Hospital Cristo Redentor, Brazil
[5]Cardiologist of the Hospital Cristo Redentor, Brazil
[6]General clinic of the Hospital Cristo Redentor, Brazil
[7]Adjunct Professor of Anesthesiology at the UFG Faculty of Medicine, Responsible for the CET-SBA of the Hospital CRER, Brazil
[8]General surgeon of the Hospital Cristo Redentor, Brazil

Correspondence: Dr. Luiz Eduardo Imbelloni, Anesthesiologist and Researcher of Various Hospital, Rio de Janeiro, RJ, Brazil, Tel + 55.11.99429-3637, Email dr.luiz.imbelloni@gmail.com

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Introduction

Amniotic fluid embolism (AFE) occurs during pregnancy, with low incidence, but is extremely serious with sudden onset and presents with severe hypotension or cardiorespiratory arrest, respiratory distress and coagulopathy, first described by Matthew Baillie in the nineteenth century.[1] In Brazil the first report is from 1926.[2]

A review of several obstetric guidelines failed to find the incidence of AFE in Brazil. AFE is a rare obstetric emergency, with an estimated occurrence 1.9 cases per 100,000 maternities (UK) to 6.1 per 100 000 maternities (Australia).[3] Maternal mortality rates in developed countries range from 61% in women with the most severe form of AFE and from 13% to 44% in other series in which some patients did not have all the features of the classic fulminant AFE syndrome.[4] Survival depends on early recognition and prompt treatment.

The most lethal AFE syndrome consists of the triad of hypoxia resulting from acute lung injury and transient pulmonary vasospasm, hypotension or cardiac arrest, and a consumptive coagulopathy, all occurring during labor or termination of pregnancy or shortly after delivery, and in the absence of reasonable alternative explanations.[5] We report a case of sudden AFE in a patient undergoing a third cesarean section, with severe arterial hypotension, dyspnea, severe pulmonary hypertension, severe hypoxemia, cardiocirculatory and respiratory collapse, immediately responsive to maneuvers with administration of vasopressors and ventilation under a mask, evolving 6 hours later with supraventricular tachycardia and need for tracheal intubation, remaining in the ICU for 10 days, having been discharged from hospital on the 13th day of AFE.

Case report

Female patient, 34 years old, pregnancy III, birth by cesarean II, abortion zero, at 39 weeks of gestational age, ASA II, scheduled for elective cesarean section, for August 26, 2024. Taking a daily multivitamin, and with comorbidities such as grade I obesity, history of allergic rhinitis and dust allergy. Denies allergy to medications, smoking, alcoholism or use of illicit drugs, and no complications in previous two cesarean sections. During the pregnancy period there are no changes.

During the pre-anesthetic visit, the airway examination showed Mallampati III. The patient fasted for eight hours before surgery on solid foods and three hours on clear liquids and was taken to the surgical center. Monitoring with non-invasive blood pressure (NIBP=130x70 mmHg), pulse oximetry (96%), cardioscopy (83 bpm), respiratory rate (16 irpm) and axillary temperature (36.5ºC) was installed.

Venipuncture was performed with a 20G catheter in the left upper limb and antibiotic prophylaxis was administered with 2 g of cefazolin and 100 ml of 0.9% saline solution, associated with 40 mg of tenoxicam, 8 mg of ondansetron and 10 mg of dexamethasone.

The patient was placed in a sitting position, antisepsis was performed with alcoholic chlorhexidine, and local anesthesia was administered with 3 ml of 2% lidocaine without vasoconstrictor using a hypodermic needle (13x0.45 mm) in the interspace between L3-L4, and subarachnoid puncture was performed with a 27G Quincke needle on the first attempt. After the release of clear cerebrospinal fluid (CSF), 0.5% hyperbaric bupivacaine 12.5 mg and morphine 80 µg were injected intrathecally in the same syringe.

Immediately after the subarachnoid injection, the patient was placed in the supine position and a sterile urinary catheter was inserted by the nursing team. After a few minutes, her BP dropped to 80x60 mmHg, with improvement after intravenous administration of 0.5 mg metaraminol and infusion of 500 ml of warmed Ringer's lactate, keeping her BP stable at around 110 x70 mmHg. After 15 minutes, the cephalic dispersion by the needle test was at T6.

After the newborn was removed, 100 ml of 0.9% saline solution and 5 IU of oxytocin were infused intravenously, when a sudden onset of persistent hypotension, dyspnea, respiratory distress, and hypoxemia occurred, with a rapid drop in saturation to 60% and a drop in BP to 50x20 mmHg. Ventilatory assistance was initiated using a face mask with a reservoir at 12 liters/min and a new dose of metaraminol 0.5 mg and ephedrine 5 mg intravenously was administered, with little response to the use of vasopressors. Pulmonary auscultation was performed with the presence of bilateral crackles up to the apex, when the diagnostic hypothesis of acute bilateral pulmonary edema was raised, and non-invasive ventilation was initiated for 5 minutes in an anesthesia machine, with a positive end-expiratory pressure (PEEP) of 8, tidal volume of 400 mL and respiratory rate of 16 irpm with good response, evolving with improvement in the respiratory discomfort, increase in saturation to 94% and increase in blood pressure to 100 x 60 mmHg, being kept by non invasise ventilation (NIV) on a mask at 8 L/min.

The patient presented with supraventricular tachycardia with an increase in HR to 145 bpm, which improved after an infusion of 150 mg of amiodarone diluted in 100 mL of 5% glucose solution in 10 minutes and was transferred to the red room where a chest X-ray was performed on the bed and a transthoracic Doppler echocardiogram. Radiographic changes were compatible with bilateral pulmonary congestion and an area of atelectasis on the left. Doppler echocardiography showed mildly reduced right ventricular (RV) systolic function, moderately increased left ventricular (LV) systolic function due to diffuse hypokinesia, left ventricular ejection fraction (LVEF) according to the Simpson method of 46%, LV diastolic function with altered relaxation, mitral, tricuspid, aortic and pulmonary insufficiency, asynchronous movement of the interventricular septum without the presence of shunts and signs of mild pulmonary arterial hypertension (Table 1-3).

Table 1 Assessment of Structural Parameters through transthoracic echodopplercardiogram performed with GE echocardiograph model

Structural Parameters	Evaluation	Reference
Aorta (Root Diameter)	27 mm	21-34 mm
Left Atrium	27 mm	27-40 mm
Right Ventricular Diameter	32 mm	20-40 mm
LV End-Diastolic Diameter	59 mm	42-56 mm
LV End-Systolic Diameter	46 mm	21-35 mm
Diastolic Septal Thickness	6 mm	6-11 mm
LVPW Diastolic Thickness	6 mm	6-11 mm
LA Volume	20 ml/m2	<34 ml/m2

Vivid T8 with multifrequency sectoral transducer

Table 2 Assessment of Ventricular Relations and Functions through transthoracic echodopplercardiogram performed with GE echocardiograph model Vivid T8 with multifrequency sectoral transducer

Ventricular Relations and Functions	Evaluation	Reference
Left Atrium / Aorta Ratio	1	1.0±0.5
Ejection Fraction (Teicholz)	44%	50-75%
Left Ventricular Mass	127.5 g	67-162 g
Mass Index	74.4 g/m2	43-95 g/m2
Percentage of Cavity Shortening	22%	≥ 32%
Septum / LVPP Ratio	1	< 1.3
End Diastolic Volume	173 ml	73-158 ml
Systolic Volume	76 ml	54-99 ml
Volume / Mass Ratio	1.36 ml/g	> 0.42 ml/g
End Systolic Volume	97 ml	18-57 ml

Table 3 Transthoracic Echodopplercardiogram Conclusions

1. Slightly reduced right ventricular systolic function.
2. Moderate enlargement of the left ventricle.
3. Slightly reduced left ventricular systolic function due to diffuse hypokinesia.
4. LVEF by the Simpson method of 46%.
5. Left ventricular diastolic function with altered relaxation.
6. Mitral, tricuspid, aortic and pulmonary insufficiency.
7. Asynchronous movement of the interventricular septum without the presence of shunts.
8. Signs of mild pulmonary arterial hypertension.

Laboratory tests were performed (Table 4). After the results of the complementary tests, the following diagnostic hypotheses were made pulmonary thromboembolism, peripartum cardiomyopathy, amniotic embolism and anaphylactic shock. Ultrasound-guided central access was performed in the right internal jugular vein and intravenous dobutamine 0.3 µg/kg/min was started, with improvement in hemodynamic and respiratory status. The patient was maintained on intermittent NIV and furosemide 80 mg IV was administered. She showed significant clinical improvement, but 6 hours later she presented a new worsening with intense dyspnea, respiratory distress, and hypoxemia. Orotracheal intubation was indicated after intravenous

induction with fentanyl 100 µg, etomidate 16 mg, and rocuronium 90 mg in rapid sequence under direct laryngoscopy (Comark-Lehane 2b), and pink fluid was seen draining from the airways.

Table 4 Laboratory tests

Exams	Results
Creatinine	0.7 mg/dl
Urea	24 mg/dl
Sodium	136 mmol/l
Chloride	106 mmol/l
Potassium	4 mmol/l
Pyruvic transaminase	15 U/l
Oxaloacetic transaminase	28 U/l
Direct bilirubin	0.15 mg/l
Indirect bilirubin	0.71 mg/l
D-dimer	3.2 µg/ml
Troponin	0.55 ng/ml
CP-MB	158 U/l

Connected to mechanical ventilation in volume-controlled ventilation mode with VT of 400 ml, RR of 22 bpm, PEEP of 10 and peak pressure of 35. Maintenance sedation was initiated with fentanyl 50 µg/h, midazolam 8 mg/h, rocuronium 24 mg/h and dobutamine 3 µg/kg/min and norepinephrine 0.07 µg/kg/min to maintain clinical stability. Patient was referred to the ICU of the Albert Einstein Hospital in Goiânia by ambulance with medical support. She was admitted to the unit at 10 hours after the cesarean section and several tests were performed, including CT angiography, and the diagnosis of amniotic fluid embolism with progression to acute respiratory distress syndrome (ARDS) was defined (Figure 1).

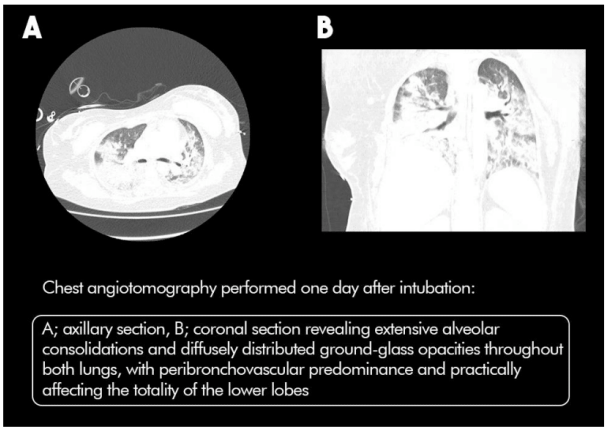

Figure 1 Chest angiotomography after intubation.

Due to the low ratio of partial pressure of O_2 to fraction of inspired O_2 of 80 (P/F), the patient was placed in the prone position and thoracic electrical impedance tomography was used to assist in alveolar recruitment maneuvers and PEEP titration (Figure 2,3). There was a good response to treatment with an increase in the P/F ratio to 280 after approximately 2 hours and above 400 after 16 hours in the prone position. The patient was changed to the supine position after 16 hours, returning to the supine position, with the P/F ratio remaining above 400.

On the 7th day of mechanical ventilation, the patient was extubated without complications. On the 10th day of hospitalization, a new chest CT scan was performed showing the evolution of the pulmonary condition (Figure 4). The patient remained in the ICU for another 2 days and another 2 days in the apartment. She was discharged from hospital on the 13th day of hospitalization, without neurological sequelae and with an indication for home respiratory physiotherapy and recorded what happened on all days of hospitalization (Figure 5).

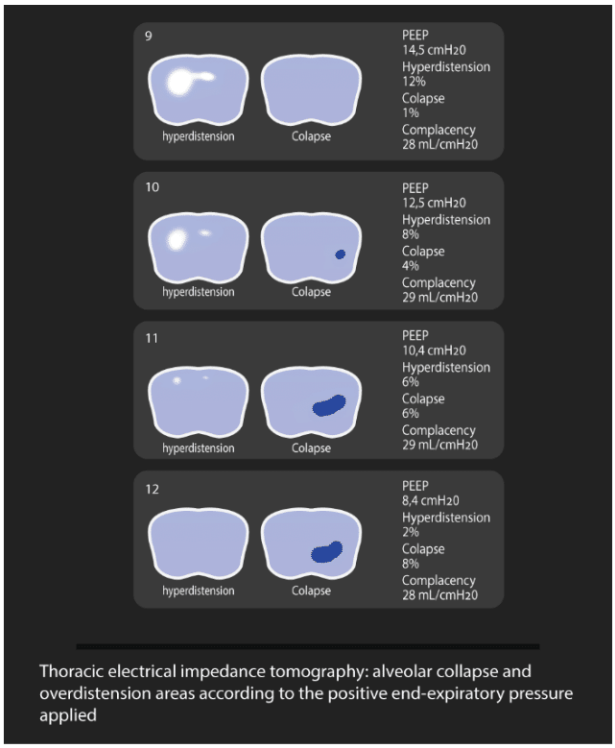

Figure 2 Thoracic electrical impedance tomography.

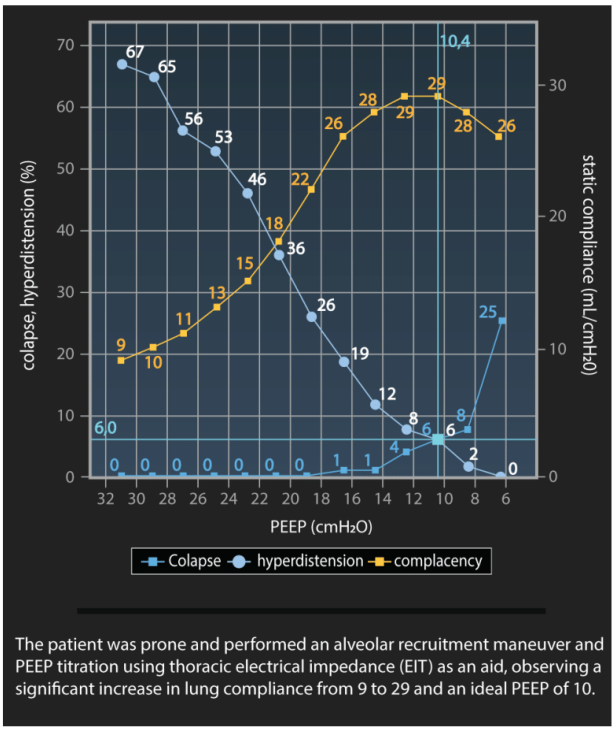

Figure 3 Patient in prone position for alveolar recruitment.

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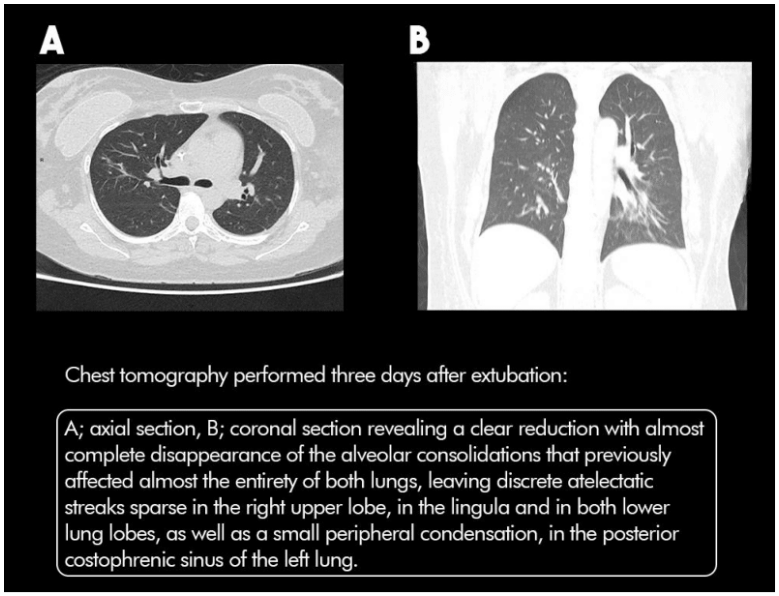

Figure 4 Chest angiotomography after extubation.

Day	Timeline	Events	Team, Drugs and Doses	Exams
1	08:30	Admission Maternity	Reception maternity	Normal Pre-Operative
	10:30	Operation Room	Obstetrics	
		Monitoring, venoclysis,	Nursing	
	10:40	drugs	Anesthesiology	
	10:50	Spinal Anesthesia	Bupivacaine	
		Start Cesarean	Hyperbaric=12.5mg	
	10:59		Morphine=80µg	
	11:10	Bradycardia,	Metaraminol=0.5mg	
	11:15	Hypotension		
		Newborn male	Atropine=0.50mg,	
		Severe hypotension	Ephedrine=10mg	
	11:25	Non-P wave	Amiodarone	Acute Pulmonary Edema
	11:40	tachyarrhythmia,	O2 Mask 6L	ECG: Infra D2, D3, AVF
		Dyspnea	Bilateral Crackles,	Chest X-ray,
		Lung auscultation	Furosemide=80mg	Transesophageal
	11:55	Red Room	Intensivist, Monitoring, NIV	Echocardiography (Table
	12:10			I, II, III)
	12:20			Improves Hypotension
	12:25	Hypokinesia, APE	Dobutamine=2.5µg/kg/min	
	13:00	Orotracheal Intubation.	Tube 7.5	Ultrasound
		IPV		
		IJVP	Fentanyl, Midazolam	
		Sedation	Intensivist, Monitoring, IPV	
		Intensive Care Unit		
2	10:00	ICU: On Endotracheal Tube	Division Intensive Care Unit	Table IV
3	10:00	ICU: On Endotracheal Tube	Division Intensive Care Unit	Evolution PEEP (Figure 1,2,3,4)
7	10:00	Extubation	Division Intensive Care Unit	
10	10:00	To Maternity Ward	Maternity Ward	
13	10:00	Discharge	Your Residence	Figure 5

Figure 5 Timeline, events, and management of the case.

NIV, Non Invasise Ventilation; APE, Acute Pulmonary Edema; IJVP, Internal Jugular Vein Puncture; IPV, Invasive Pulmonary Ventilation

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Discussion

The presentation of this case of AFE clearly demonstrates the severity of this pathology with imminent risk of death. The participation of a multidisciplinary team and the possibility of performing tests to confirm the diagnosis allowed an immediate response to clinical reactions, with hospital discharge on the 13th day after the event. In this case, the pregnant woman was in her third pregnancy and did not present any symptoms that were recorded and required treatment.

In Brazil, of the 1,048,576 deaths in 2023, only three had AFE as the underlying cause on their death certificate, however in 2024 there were already four reported cases of AFE.[6] If the incidence of AFE varies depending on the reporting methodology, certainly a lower incidence is always estimated.[3] In a retrospective study published in 2024, using the 2000-2019 Health Care Cost and Utilization Project, Nationwide Inpatient Sample (HCUP-NIS), AFE incidence rate remained stable (mean 4.9 cases/100,000 deliveries) and the case-fatality rate declined (mean 17.7 %.95 % CI 16.40–10.09).[7] In this case, the patient remained in the ICU for 10 days and was discharged from hospital fully recovered.

With the aim of determining the incidence of AFE with cesarean section and age from 1980 through 2005, there were 112,712,000 deliveries, of which 12,000 patients (11=100,000) had AFE, showed that the risk is higher with cesarean section and higher in women aged 30 years.[8] The patient was 34 years old and underwent a third cesarean section, and according to the study, this may have contributed to the development of the AFE. AFE remains unpredictable, with an origin that is still unclear. Several factors contribute to its development, including multiparity, male fetuses, early gestational age, cervical ripening, polyhydramnios, multiple gestation, gestational diabetes, operative delivery, manual extraction of the placenta, regional disparities, Asian and black races, asthma, use of illicit substances, and trauma.[9] The patient was over 30 years old, was multiparous, with two previous cesarean sections and the newborn was of the sex male, in the factors contributing to the onset of AFE.

AFE is characterized by a disruption in the placental-amniotic interface, leading to the entry of amniotic fluid and fetal elements, such as hair, meconium, skin cells, and intestinal mucin, into the maternal circulation. It is essential to note that the presence of squamous cells in the pulmonary circulation is no longer solely diagnostic for AFE, as clinical presentation plays a key role in the management of the syndrome.[10] Orotracheal intubation verified the presence of pink fluid was seen draining from the airways, chest radiography and CT confirmed the diagnosis.

The introduction of amniotic fluid and fetal elements triggers inflammatory mediators, including platelet-activating factor, tissue necrosis factor-alpha, interleukin 6, interleukin 1, phospholipase A2, endothelin, plasminogen activators, thromboplastins, and complement factors.[11] The patient did not present disseminated intravascular coagulation.

In a study with eighty-seven moderates to severe ARDS patients with the objective of comparing titration of PEEP with electrical impedance tomography (EIT) and with ventilator-embedded pressure–volume (PV) loop in moderate to severe ARDS, showed that PEEP titration guided with EIT, compared with PV curve, might be associated with improved driving pressure and survival rate.[12] In this case, the recruitment maneuver in the prone position and PEEP titration using EIT observed a significant increase in lung compliance from 9 to 29 with an ideal PEEP of 10.

Conclusion

AFE is a rare but serious condition with high mortality and morbidity rates and is the second leading cause of peripartum maternal death. It occurs when amniotic fluid enters the maternal bloodstream. Cardiovascular collapse often serves as the initial clinical indicator of AFE, having been the first sign of the complication. Sudden cardiovascular collapse is induced by hypoxemia and hypotension early detection, diagnosis, and treatment of AFE are essential to avoiding fatal outcome. The bedside use of thoracic electrical impedance tomography may be a clinical tool that is able to guide, at each breath, possible adjustments of regional ventilation, including the decision for alveolar recruitment maneuvers, in patients with acute respiratory distress syndrome.

Ethical approval

As this was a case report, ethical approval from the Institutional Review Committee was not sought. However, written informed consent was obtained from the patient.

Patient consent

Written consent for publication was obtained from the patient.

Disclosures

Name: Humberto de Souza Cândido, MD

Contribution: Anesthesiologist who performed spinal anesthesia during the third cesarean section and led the treatment until arrival at the ICU. And then monitored the treatment until hospital discharge.

Name: Luiz Eduardo Imbelloni, MD, PhD.

Contribution: The author has 50 years of experience in anesthesiology and worked as a consultant at the time of the AFE, and later wrote the Case Report, for possible publication in the Journal of Anesthesiology or Obstetrics and Gynecology.

Name: Angelo Antônio Gomes de Carvalho, MD

Contribution: Coordinator of the ICU at the Albert Einstein Hospital in Goiânia where the patient was hospitalized, and responsible for the conduct in the ICU together with the entire team that participated in the multidisciplinary care.

Name: Dilene Morais Barbosa Gisch, MD

Contribution: Gynecologist and obstetrician who performed the cesarean section and monitored the AFE treatment until hospital discharge. Subsequently, she monitored the patient until the end of the first month when she returned home.

Name: Rogério Silva Monteiro, MD

Contribution: Responsible for performing the bedside Doppler echocardiogram that helped in the conduct of starting dobutamine due to the patient having pulmonary edema associated with a low ejection fraction from ventricular dysfunction.

Name: Stela Nunes Menezes, MD

Contribution: Participation in the treatment of AFE during cesarean section until ICU transfer.

Name: Tolomeu A. A. Casali, MD, PhD

Contribution: This author was the preceptor during the HSC residency, and therefore, assisted during the AFE treatment procedure. He evaluated the Case Report before submitting it for publication.

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Name: Robson de Brito Oliveira, MD

Contribution: Cesarean section delivery assistant together with the obstetrician.

References

1. Baillie M. The Works of Matthew Baillie. 1825. Vol. I, pp. 229 and 230. Longman, Hurst, Rees, Orme, Brown and Gr–ecn, London.
2. Meyer JR. Embolia pulmonar amnio caseosa. *Bras Med.* 1926;2:301–303.
3. Knight M, Berg C, Brocklehurst P. Amniotic fluid embolism incidence, risk factors and outcomes: a review and recommendations. *BMC Pregnancy and Childbirth.* 2012;12:7.
4. Clark SL. Amniotic fluid embolism. *Obstet Gynecol.* 2014;123:337–348.
5. Conde–Agudelo A, Romero R. Amniotic fluid embolism: an evidence–based review. *Am J Obstet Gynecol.* 2009;201:445.e1–13.
6. Dados abertos do SIM (Sistema de Informações de Mortalidade) obtido do Departamento de Análise Epidemiológica e Vigilância em Saúde e Ambiente, da Coordenação Geral de Informações e Análises Epidemiológicas (CGIAE). Ministério da Saúde do Brasil, obtido pelo site: 2024.
7. Suissa N, Czuzoj–Shulman N, Abenhaim HA. Amniotic fluid embolism: 20–year incidence and case–fatality trends in the United States. *Eur J Obstet Gynecology and Reproductive Biology.* 2024;294:92–96.
8. Stein PD, Matta F, Yaekoub AY. Incidence of amniotic fluid embolism: Relation to cesarean section and to age. *Journal Women's Health.* 2009;18(3):327–329.
9. Mazza GR, Youssefzadeh AC, Klar M, et al. Association of pregnancy characteristics and maternal mortality with amniotic fluid embolism. *JAMA Netw Open.* 2022;5(11):e2242842.
10. Haftel A, Carlson K, Chowdhury YS. Amniotic fluid embolism. Last Update: January 10, 2024. Stat Pearls [Internet].
11. Griffin KM, Oxford–Horrey C, Bourjeily G. Obstetric disorders and critical illness. *Clin Chest Med.* 2022;43(3):471–488.
12. Hsu HJ, Chang HT, Zhao Z et al. Positive end–expiratory pressure titration with electrical impedance tomography and pressure–volume curve: a randomized trial in moderate to severe ARDS. *Physiol Meas.* 2021;42:014002.

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