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CASE REPORTS / CASE SERIES

The role of echocardiography in amniotic fluid embolism: a case series and review of the literature

Le rôle de l'échocardiographie dans l'embolie de liquide amniotique : une série de cas et une revue de la littérature

Camille Simard, MD · Stephen Yang, MD, MSc · Maral Koolian, MD, MSc · Roberta Shear, MD · Lawrence Rudski, MD · Jed Lipes, MD

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Abstract

Purpose *Amniotic fluid embolism (AFE) is a rare, but often fatal condition characterized by sudden hemodynamic instability and coagulopathy occurring during labour or in the early postpartum period. As the mechanisms leading to shock and the cardiovascular effects of AFE are incompletely understood, the purpose of this case series is to describe how AFE presents on echocardiography and review limited reports in the literature.*

Clinical features *We describe three cases of AFE at the Jewish General Hospital, a tertiary care centre in Montreal, Canada. All cases met the Clark diagnostic criteria, which comprise 1) sudden cardiorespiratory arrest or both hypotension and respiratory compromise, 2) disseminated intravascular coagulation, 3) clinical onset during labour or within 30 min of delivery of the placenta, and 4) absence of fever. Two patients had a cardiac arrest and the third developed significant hypotension and hypoxia. In all patients, point-of-care echocardiography at the time of shock revealed signs of right ventricular failure including a D-shaped septum, acute pulmonary hypertension, and right ventricular systolic dysfunction.*

Conclusion *This case series and literature review of AFE emphasizes the importance of echocardiography in elucidating the etiology of maternal shock. The presence of right ventricular failure may be considered an important criterion to diagnose AFE.*

Résumé

Objectif *L'embolie de liquide amniotique (ELA) est une complication rare mais souvent fatale caractérisée par une*

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C. Simard, MD
Department of Medicine, McGill University, Montreal, QC, Canada

S. Yang, MD, MSc
Division of Critical Care, Department of Medicine, Jewish General Hospital, McGill University, 3755 ch. de la Côte-Sainte-Catherine Rd, Suite H-364.1, Montreal, QC H3T 1E2, Canada

Department of Anesthesia, Jewish General Hospital, McGill University, Montreal, QC, Canada

M. Koolian, MD, MSc
Division of General Internal Medicine, Jewish General Hospital, McGill University, Montreal, QC, Canada

R. Shear, MD
Department of Obstetrics and Gynecology, Jewish General Hospital, McGill University, Montreal, QC, Canada

L. Rudski, MD
Azrieli Heart Center, Jewish General Hospital, Montreal, QC, Canada

Department of Medicine, Jewish General Hospital, McGill University, Montreal, QC, Canada

J. Lipes, MD (✉)
Division of Critical Care, Department of Medicine, Jewish General Hospital, McGill University, 3755 ch. de la Côte-Sainte-Catherine Rd, Suite H-364.1, Montreal, QC H3T 1E2, Canada
e-mail: jed.lipes@mcgill.ca

Division of General Internal Medicine, Jewish General Hospital, McGill University, Montreal, QC, Canada

instabilité hémodynamique et une coagulopathie soudaines survenant pendant le travail obstétrical ou au début de la période postpartum. Étant donné que les mécanismes menant au choc et les effets cardiovasculaires de l'ELA ne sont que partiellement compris, le but de cette série de cas était de décrire comment l'ELA apparaît à l'échocardiographie et de passer en revue les rares comptes rendus dans la littérature.

Caractéristiques cliniques *Nous décrivons trois cas d'ELA survenus à l'Hôpital général juif, un centre tertiaire de soins à Montréal, au Canada. Tous les cas remplissaient les critères diagnostiques de Clark, qui comportent 1) un arrêt cardiorespiratoire soudain ou une hypotension accompagnée d'une détresse respiratoire, 2) une coagulation intravasculaire disséminée, 3) une apparition clinique pendant le travail obstétrical ou dans un délai de 30 minutes suivant la délivrance du placenta, et 4) l'absence de fièvre. Deux patientes ont subi un arrêt cardiaque et le tiers des patientes ont manifesté une hypotension et une hypoxie significatives. Chez toutes les patientes, l'échocardiographie au chevet au moment du choc a révélé des signes d'insuffisance ventriculaire droite, y compris un septum en forme de D, une hypertension pulmonaire aiguë et une dysfonction systolique ventriculaire droite.*

Conclusion *Cette série de cas et revue de littérature de l'ELA souligne l'importance de l'échocardiographie pour élucider l'étiologie du choc maternel. La présence d'une insuffisance ventriculaire droite peut être considérée un critère important pour diagnostiquer une ELA.*

Keywords Amniotic fluid embolism · Right ventricular failure · Maternal cardiac arrest · Echocardiography · POCUS

Introduction

Amniotic fluid embolism (AFE) is a rare obstetrical emergency characterized by acute hypotension, hypoxia, and coagulopathy. The syndrome occurs during labour or soon after delivery and is a leading cause of maternal cardiac arrest.[1] The true incidence of AFE is confounded by several factors including the use of different diagnostic criteria, a clinical presentation that mimics more common obstetrical complications, and the absence of a gold standard test.[2] In a Canadian population-based cohort study, the incidence of AFE was 1 per 40,000 deliveries, with a reported mortality of 27%.[3] Conflicting evidence exists with regards to identifiable risk factors for AFE. Reported risk factors include situations that are likely to lead to exchange of maternal and fetal fluid, including Cesarean delivery (CD), instrumented vaginal delivery, and cervical trauma.[2]

Although incompletely understood, AFE is thought to arise from the patient's reaction to amniotic fluid components.[2] Both occlusion of vessels with amniotic fluid material and immune or anaphylactoid reactions have been postulated in precipitating the clinical syndrome.[2] Macroscopic occlusion of maternal pulmonary vessels with fetal debris leading to hypoxia and cardiovascular collapse, however, is no longer a favoured primary pathophysiologic process as fetal material can be found in women during labour without AFE and injection of amniotic fluid is often benign in animal models.[4]

No single diagnostic test or biomarker has been identified to reliably diagnose AFE in the clinical setting, leaving accurate and prompt diagnosis an important challenge.[5] Moreover, because of the fulminant course and the rapidity with which this condition evolves, the study of this entity has been challenging. Several diagnostic criteria have been proposed; the Clark criteria (Table 1) are commonly used and are thought to be the most specific.[6,7]

More recently, point-of-care echocardiography has emerged as a critical tool in the diagnosis and management of patients in shock and cardiac arrest.[8] We report three cases of AFE highlighting the importance of point-of-care echocardiography in the workup of maternal shock, as well as a review of the literature. These cases occurred at the Jewish General Hospital (JGH), a tertiary care centre in Montreal, Quebec and a teaching site for McGill University. The JGH provides specialized obstetric care, with over 4,000 births annually, and houses a level III neonatal intensive care unit (NICU). Written informed consent was obtained from all patients.

Case 1

A 27-yr-old woman, gravida 7, para 4, abortus 2 (G7P4A2), was admitted at 34 weeks' gestation for anti-d alloimmunization and suspected fetal anemia. She was known for a history of a sinus venosus atrial septal defect with resultant pulmonary hypertension (101 mm Hg) and had undergone surgical closure in 2016. Postoperatively, she had been followed regularly with normalization of her pulmonary pressure and right ventricular (RV) function. During her current pregnancy, she was followed at the Centre for Pulmonary Vascular Disease at the JGH and underwent serial echocardiography examinations. Four days prior to delivery, the patient had a transthoracic echocardiography with a systolic pulmonary artery pressure of 35 mm Hg. She had a normal pre-delivery RV dimension (39 mm in apical four-chamber view) with preserved function (via visual assessment, RV fractional

Table 1 Clark diagnostic criteria for amniotic fluid embolism

1. Sudden onset of cardiorespiratory arrest, or both hypotension (systolic blood pressure < 90 mm Hg) and respiratory compromise (dyspnea, cyanosis, or peripheral capillary oxygen saturation [SpO_2] < 90%)
2. Documentation of overt disseminated intravascular coagulation[9] following appearance of these initial signs or symptoms using the scoring system of Scientific and Standardization Committee on DIC of the ISTH modified for pregnancy. Coagulopathy must be detected prior to loss of sufficient blood to itself account for dilutional or shock-related consumptive coagulopathy
3. Clinical onset during labour or within 30 min of delivery of placenta
4. No fever (≥ 38.0 °C) during labour

DIC disseminated intravascular coagulation, *ISTH* International Society on Thrombosis and Haemostasis

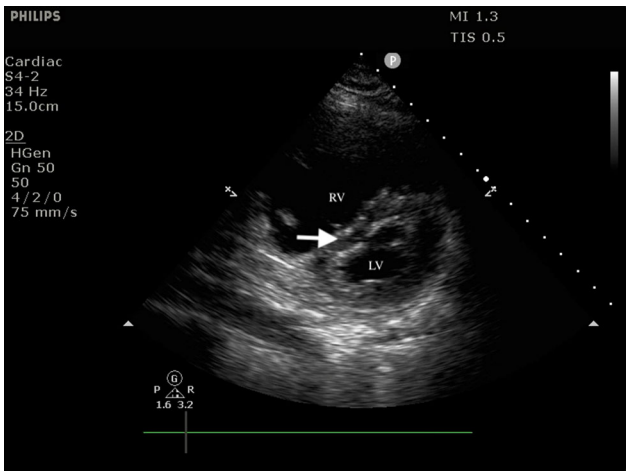

Fig. 1 Transthoracic parasternal short-axis view showing a dilated right ventricle (RV) with a D-shaped septum (white arrow), and a small underfilled left ventricle (LV)

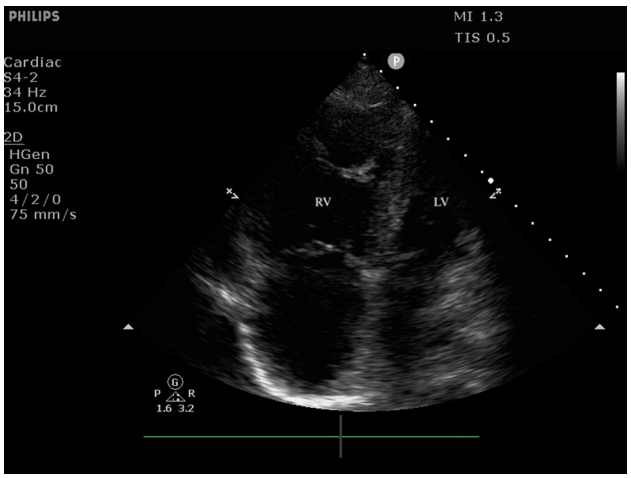

Fig. 2 Transthoracic apical four-chamber view showing a severely dilated right ventricle (RV)

area change [FAC] of 40% myocardial performance index 0.2, and tissue Doppler imaging RV S' of 10 $cm{\cdot}sec^{-1}$). After therapeutic options for fetal anemia were discussed, the patient underwent an elective CD for breech presentation at 34 weeks' gestation. Spinal anesthesia was performed using bupivacaine 0.75% 9.75 mg with fentanyl 12.5 µg and preservative-free morphine 150 µg. Blood pressure was maintained after spinal anesthesia without need for vasopressors. A 2.27 kg male baby was delivered with APGAR scores of 7 and 9 at one and five minutes, respectively. Immediately post-delivery of the baby and prior to delivery of the placenta, the patient had an asystolic cardiac arrest and required cardiopulmonary resuscitation (CPR) and urgent intubation. There was return of spontaneous circulation after three minutes of CPR and 1 mg of intravenous epinephrine. A point-of-care echocardiogram performed by an intensivist showed a severely dilated right ventricle with an akinetic free wall, RV FAC of 17%, a D-shaped septum as well as moderate tricuspid regurgitation (Figs 1 and 2 and Electronic Supplementary Material eVideo 1, eVideo 2). Femoral access was obtained for extracorporeal membrane oxygenation (ECMO); however, the patient maintained hemodynamic stability with vasopressor support and did not require initiation of ECMO. Her hemodynamic status stabilized with infusions of intravenous norepinephrine at 10 $µg{\cdot}min^{-1}$ and milrinone at 0.375 $µg{\cdot}kg^{-1}{\cdot}min^{-1}$. She underwent a computed tomography-pulmonary angiogram (CTPA) which showed no pulmonary embolism (PE) or proximal deep vein thrombosis but dilated right cardiac chambers and pulmonary arteries suggestive of RV strain. She was subsequently admitted to the intensive care unit (ICU). Investigations revealed an international normalized ratio (INR) of 1.0, a platelet count of 77 x $10^9{\cdot}L^{-1}$, a fibrinogen level of 2.5 $g{\cdot}L^{-1}$, and a normal prothrombin time (PT) of 10.9 sec. The patient's International Society on Thrombosis and Haemostasis (ISTH) disseminated intravascular coagulation (DIC) score was 27 (a cut-off of > 26 suggestive of DIC).[9] She was successfully extubated after 24 hr and had a favourable clinical evolution. She was discharged home six days post cardiac arrest from AFE and has been clinically well on follow-up visits.

Case 2

A 24-yr-old woman, G4P3 at 37 weeks' gestation, presented for decreased fetal movement and was found to have intrauterine fetal demise with six tight nuchal cords. The patient had no significant past medical or obstetrical history. She was admitted for induction of labour. Following rupture of membranes, the patient became minimally responsive and profoundly hypotensive with a systolic blood pressure of 60 mm Hg. A code blue was called, and she received fluid resuscitation and intravenous norepinephrine, which improved her hemodynamic parameters. She required supplemental oxygen via a 50% face mask for an oxygen saturation of 94% but no mechanical ventilation was needed. A limited point-of-care echocardiogram performed by the intensivist showed a D-shaped septum and RV systolic dysfunction visually suggestive of RV failure. She was admitted to the ICU and underwent a vacuum-assisted vaginal delivery. Upon delivery, the patient developed severe postpartum hemorrhage. Her INR was 1.2, platelet count was 93 x $10^9 \cdot L^{-1}$, fibrinogen level was 1.9 $g \cdot L^{-1}$, and PT was 14.2 sec. Her ISTH DIC score for pregnancy was 39, consistent with DIC. A massive transfusion protocol was activated, and she was transported to the operating room for repair of cervical and vaginal lacerations. A Bakri balloon and vaginal packing were placed. The patient received a total of ten units of packed red blood cells, ten units of fresh frozen plasma, 25 units of platelets, and 40 units of cryoprecipitate, as well as intravenous calcium chloride and tranexamic acid. The estimated blood loss was 6 L. Once hemostasis was achieved, she improved hemodynamically and returned to the ICU where she had a favourable clinical evolution. A CTPA was performed on day 1 of admission to the ICU and showed no evidence of PE, but findings suggestive of acute respiratory distress syndrome. She was discharged home in stable condition seven days later with a diagnosis of AFE.

Case 3

A 38-yr-old woman, G4P1 at 40+3 weeks' gestation, was admitted with spontaneous rupture of membranes. She had no significant past medical history. Her obstetrical history was remarkable for a past uncomplicated term delivery and two spontaneous first trimester miscarriages. During labour, the patient suddenly felt nauseated and unwell. She showed no signs of infection or bleeding suggestive of hypovolemia. She was found to be hypotensive with a systolic blood pressure of 80 mm Hg and soon after, sustained a cardiorespiratory arrest. Cardiopulmonary resuscitation was initiated, a bedside perimortem CD was performed and the ECMO team was activated. A 4.18 kg female baby was delivered with APGAR scores of 0, 4, and 6 at one, five, and ten minutes, respectively. She was transferred to the NICU and had a favourable clinical evolution with no short-term sequelae. After two minutes of CPR and delivery of the baby, the patient had return of spontaneous circulation and did not require ECMO cannulation. An infusion of norepinephrine was initiated, and the patient was transferred to the ICU. A transesophageal echocardiogram was performed by a cardiologist approximately one hour after her arrest, which showed a hyperdynamic left ventricle (LV) with a normal-sized right ventricle with mildly reduced systolic function. A small inferior vena cava with collapse suggested hypovolemia and it was noted that she developed massive postpartum hemorrhage. During the initial volume resuscitation, ongoing imaging showed progressive RV dilatation and dysfunction, worsened tricuspid regurgitation, and pulmonary hypertension (Figs 3 and 4). A massive transfusion protocol was initiated, and she received five units of packed red blood cells, five units of fresh frozen plasma, five units of cryoprecipitate, and five units of platelets. A Bakri balloon was inserted for uterine atony. Laboratory investigations confirmed the presence of DIC with an INR of 1.5, platelet count of 96 x $10^9 \cdot L^{-1}$, fibrinogen level of 0.7 $g \cdot L^{-1}$, PT of 16.7 sec, and ISTH DIC score of 52. Once stabilized, the patient underwent a CTPA, which was negative for PE. Her clinical status continued to improve, and she was discharged home in stable condition six days post arrest.

Discussion

Amniotic fluid embolism is a leading cause of maternal mortality in developed countries, affecting one per 40,000 Canadian deliveries and approximately one in 10,000 deliveries in the United States.[2,3] The understanding of risk

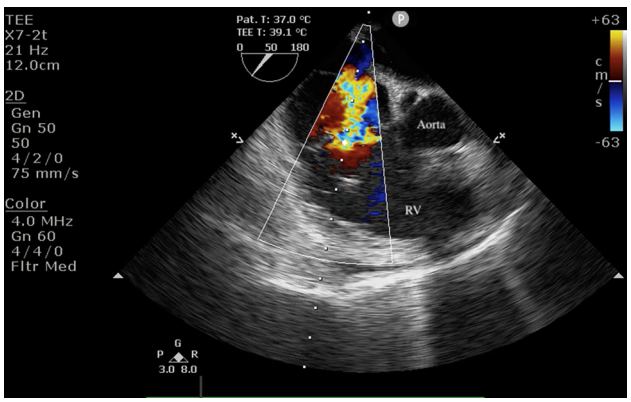

Fig. 3 Transesophageal right ventricular inflow-outflow view showing severe tricuspid regurgitation

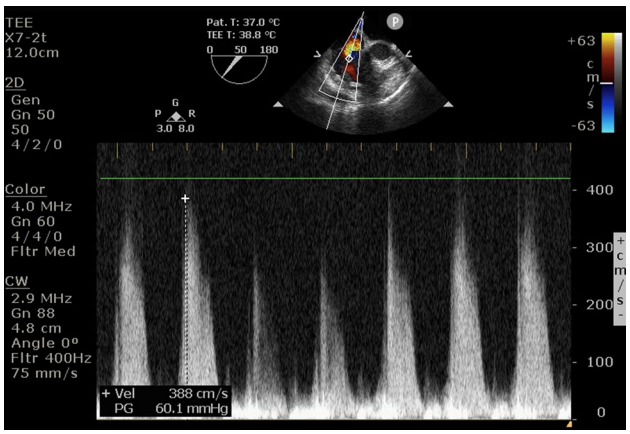

Fig. 4 Doppler flow showing severe pulmonary hypertension with a peak gradient of 60 mm Hg across the tricuspid valve

factors, diagnosis and therapies are limited by the lack of a uniform clinical definition and gold standard for diagnosis.[10] We present three cases of AFE, all of which met current diagnostic criteria as outlined by Clark *et al.*[10] In the management of all cases, an urgent bedside echocardiogram was obtained, which enabled a rapid diagnosis of RV failure. This allowed the exclusion of other etiologies of maternal cardiac arrest, including peripartum cardiomyopathy, hypovolemic shock, or anaphylaxis. Moreover, all patients had an urgent CTPA to exclude PE to ensure no targeted therapy (i.e., thrombolysis, anticoagulation, or embolectomy) needed to be initiated.

For patient number three, the initial echocardiogram occurred at the same time as her postpartum hemorrhage, and her apparent hypovolemia may have masked some of the changes usually associated with a failing right ventricle. Indeed, after the initial hypovolemia was corrected, her right ventricle became dilated with evidence of systolic dysfunction and pulmonary hypertension. Although excessive volume loading and post resuscitation cardiac arrest may have contributed to her RV dysfunction, she suffered a cardiac arrest with no other apparent cause and met all Clark criteria for AFE. Moreover, her echocardiography showed isolated RV dysfunction, which is uncommon following return of circulation post cardiac arrest.[11]

Although initially described as causing left ventricular failure, our case series highlights the importance of RV failure as an important pathophysiologic process in AFE leading to cardiopulmonary collapse. This series, together with previously reported echocardiographic case reports (Table 2), strongly indicates that AFE is associated with severe RV dysfunction leading to cardiopulmonary collapse.[12,13] We performed a literature search in Medline from inception up to February 2021 on the use of echocardiography in the diagnosis of AFE. Cases which included a description of echocardiography findings and met Clark criteria or had no alternative diagnosis other than AFE were included (Table 2).

Echocardiographic findings in acute RV failure may show a dilated right ventricle, systolic dysfunction, and pulmonary hypertension.[14] Acute pulmonary hypertension from AFE can lead to RV pressure overload causing the interventricular septum to be displaced towards the LV, and is easily identified as a D-shaped septum on a parasternal short-axis view, as seen in two of our cases.[15] In addition to two-dimensional visual assessment of systolic function (eyeball technique), RV function can be quantified using several echo parameters including tricuspid annular systolic excursion, fractional area change, tissue Doppler-derived tricuspid lateral annular systolic velocity (S'), free wall longitudinal strain, and RV index of myocardial performance. Although no single parameter is considered best, a combination of both a visual and a quantifiable parameter is ideal.[16]

Although rare, echocardiography may also detect thrombus in the pulmonary artery or in the cardiac chambers as *clot in transit*.[17] Amniotic fluid embolism has been described with clot in transit, however distinguishing AFE from classic PE on echocardiography is not possible.[18] Five cases in the literature report clot in transit on echocardiography as probable cases of AFE; however, PE was not formally excluded with imaging, leaving uncertainty regarding the true diagnosis.[19–22] The presence of acute RV failure along with DIC during or soon after labour may suggest AFE, but PE has also been reported to occur during delivery.[23,24] The risk of venous thromboembolism is increased five-fold during pregnancy compared with age-matched nonpregnant women, and the relative risk in the postpartum period has been found to be as high as 60-fold.[25] Disseminated intravascular coagulation can also occur with massive PE or following cardiac arrest and its presence should not dissuade the clinician from eliminating PE as a possible diagnosis.[26–28]

Although left ventricular failure can occur, this is likely a late finding and has only been documented when echocardiography has been delayed.[29,30] The mechanism causing left ventricular failure may be related to progressive shock stemming from the initial AFE event. Dib *et al.* reported a left ventricular ejection fraction (LVEF) of 5–10% in a possible case of AFE performed 3.5 hr post hemodynamic decompensation.[29] Nevertheless, there were ST changes on the electrocardiogram, myocardial necrosis on laboratory testing, and normal coronaries, which may have indicated an alternative diagnosis such as Takotsubo syndrome (stress cardiomyopathy). Eiras Marino *et al.* reported biventricular involvement in a possible case of AFE with

Table 2 Summary of amniotic fluid embolism cases reporting echocardiography findings

Author	Year	Clark criteria met	Modality	Timing*	Right ventricle	Left ventricle	Pulmonary hypertension	Additional comments
Eiras Marino *et al.*[30]	2019	4/4	TEE	12 hours	NA	LVEF 8%	NA	Possible alternative diagnosis (cardiomyopathy)
Bernstein *et al.*[37]	2019	4/4	TEE	Peri-arrest	Dilated and hypokinetic RV with D-shaped septum	Normal	NA	_
Wise *et al.*[38]	2016	4/4	TTE	NA	Diffuse hypokinesis and McConell's sign	NA	Severe	RV failure possible from volume overload
Evans *et al.*[39]	2014	4/4	TEE	Peri-arrest	Dilated and hypokinetic RV with septal shift.	Hyperdynamic	NA	_
Ecker *et al.*[40]	2012	3/4	TEE	Peri-arrest	D-shape septum	Normal	NA	Absence of DIC
Vellayappan *et al.*[18]	2009	3/4	TEE	90 min	Dilated RV with moderate hypokinesis	Normal	None	Fetal material confirmed on embolus pathology
McDonnell *et al.*[41]	2007	4/4	TEE	4 hr	Dilated RV and severe dysfunction with septal shift	Normal	68 mm Hg	_
James *et al.*[42]	2004	4/4	TEE	30 min	Dilated RV and pulmonary arteries D-shaped septum	Normal	NA	Autopsy confirmed amniotic elements
Stanten *et al.*[43]	2003	4/4	TEE	10 min	Akinetic and dilated RV with septal shift	Hyperdynamic & underfilled	NA	No proximal PE seen on direct surgical exploration
Rufforny-Doudenko *et al.*[44]	2002	3/4	TEE	NA	Severe RV dilatation, hypokinesis and septal shift	NA	NA	Pathology of uterus showed fetal material in vasculature
Shechtman *et al.*[13]	1999	4/4	TEE	Peri-arrest	Severe RV dilatation, dysfunction, and septal shift	Normal	45 mm Hg	Autopsy confirmed amniotic elements
Dib *et al.*[29]	1996	4/4	TTE	3.5 hr	Moderate hypokinesis	LVEF 10%	NA	Possible alternative diagnosis (stress cardiomyopathy)

*Timing from maternal collapse to cardiac echocardiography

LVEF left ventricular ejection fraction, *NA* not available, *RV* right ventricle, *TTE* transthoracic echocardiography, *TEE* transesophageal echocardiography

an LVEF of 8%; however, their exam was performed 12 hr after a cardiac arrest.[30] The presence of acute left ventricular failure on echocardiography should prompt the clinician to consider other diagnoses including peripartum cardiomyopathy, pregnancy-associated myocardial infarction, or Takotsubo syndrome.[31,32]

In our cases, the patients were stabilized at the bedside with vasopressor and inotropic support with a plan for ECMO in case of ongoing shock. ECMO has been successfully utilized for maternal cardiac arrest, including AFE, and must be considered early in the resuscitation to minimize the duration of hypoperfusion.[33] Once stabilized on ECMO, the clinician may then proceed with more diagnostic tests and specific therapy if indicated.

Point-of-care ultrasound (POCUS), including echocardiography, is now the standard of care in critical care resuscitation and is widely available, even in resource-limited countries.[34] Echocardiography has been shown to decrease the time to diagnosis and alter management for patients with dyspnea, shock, or cardiac arrest.[35] Utilizing echocardiography may improve maternal outcomes by discriminating between various aetiologies of obstetrical shock and merits further study (Fig. 5). Moreover, as anesthesiologists and intensivists become increasingly skilled with POCUS in their routine practice, this should expand and facilitate its use in the acute care setting.[36]

In conclusion, we describe three cases of AFE with point-of-care echocardiography showing RV failure as the

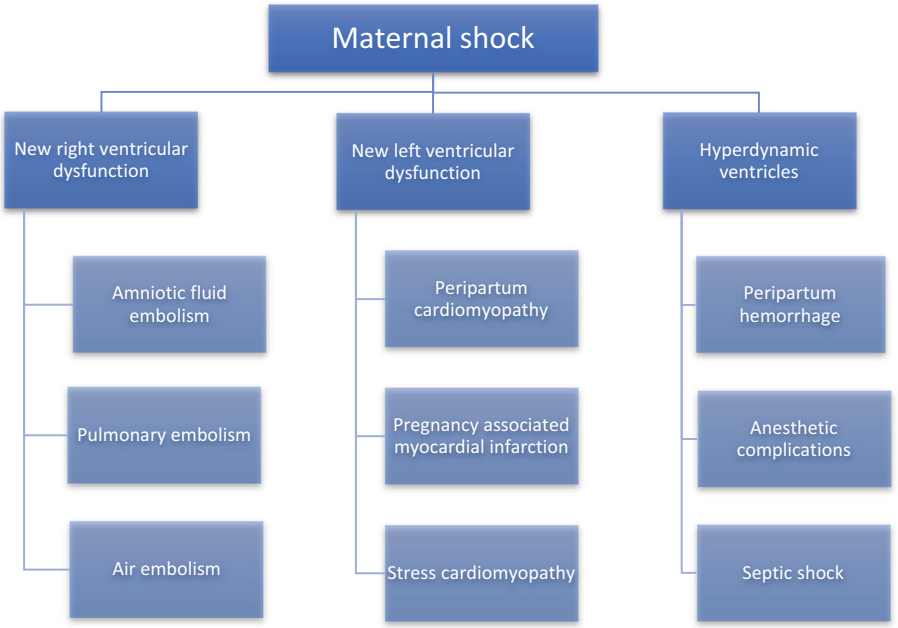

Fig. 5 Echocardiographic based differential diagnosis for maternal shock

primary etiology for cardiopulmonary collapse. A literature review of 12 case reports on AFE emphasizes the importance of applying POCUS echocardiography to identify the source of acute cardiopulmonary failure in obstetrical patients. The presence of RV failure and a negative scan for PE could be considered important criteria to diagnose AFE.

Author contributions *Camille Simard* and *Jed Lipes* contributed to all aspects of this manuscript, including study conception and design; acquisition, analysis, and interpretation of data; and drafting the article. *Stephen Yang* and *Maral Koolian* contributed to the conception and design of the study. *Roberta Shear* and *Lawrence Rudski* contributed to the acquisition, analysis, and interpretation of data.

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