

Amniotic fluid embolism: diagnosis and management

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The practice of medicine continues to evolve, and individual circumstances will vary. This publication reflects information available at the time of its submission for publication and is neither designed nor intended to establish an exclusive standard of perinatal care. This publication is not expected to reflect the opinions of all members of the Society for Maternal-Fetal Medicine.

OBJECTIVE: We sought to provide evidence-based guidelines regarding the diagnosis and management of amniotic fluid embolism.

STUDY DESIGN: A systematic literature review was performed using MEDLINE, PubMed, EMBASE, and the Cochrane Library. The search was restricted to English-language articles published from 1966 through March 2015. Priority was given to articles reporting original research, in particular randomized controlled trials, although review articles and commentaries were consulted. Abstracts of research presented at symposia and scientific conferences were not considered adequate for inclusion. Evidence reports and published guidelines were also reviewed, and additional studies were located by reviewing bibliographies of identified articles. Grading of Recommendations Assessment, Development, and Evaluation (GRADE) methodology was used for defining the strength of recommendations and rating quality of the evidence. Consistent with US Preventive Task Force guidelines, references were evaluated for quality based on the highest level of evidence.

RESULTS AND RECOMMENDATIONS: We recommend the following: (1) we recommend consideration of amniotic fluid embolism in the differential diagnosis of sudden cardiorespiratory collapse in the laboring or recently delivered woman (GRADE 1C); (2) we do not recommend the use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of amniotic fluid embolism; at the present time, amniotic fluid embolism remains a clinical diagnosis (GRADE 1C); (3) we recommend the provision of immediate high-quality cardiopulmonary resuscitation with standard basic cardiac life support and advanced cardiac life support protocols in patients who develop cardiac arrest associated with amniotic fluid embolism (GRADE 1C); (4) we recommend that a multidisciplinary team including anesthesia, respiratory therapy, critical care, and maternal-fetal medicine should be involved in the ongoing care of women with AFE (Best Practice); (5) following cardiac arrest with amniotic fluid embolism, we recommend immediate delivery in the presence of a fetus ≥ 23 weeks of gestation (GRADE 2C); (6) we recommend the provision of adequate oxygenation and ventilation and, when indicated by hemodynamic status, the use of vasopressors and inotropic agents in the initial management of amniotic fluid embolism. Excessive fluid administration should be avoided (GRADE 1C); and (7) because coagulopathy may follow cardiovascular collapse with amniotic fluid embolism, we recommend the early assessment of clotting status and early aggressive management of clinical bleeding with standard massive transfusion protocols (GRADE 1C).

Key words: amniotic fluid embolism, cardiorespiratory arrest, pregnancy

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A mniotic fluid embolism is a rare but potentially lethal condition. Because of a lack of international consensus regarding diagnostic criteria, estimates of both incidence and mortality rates associated with amniotic fluid embolism vary widely.¹⁻⁴ These issues have recently been reviewed in detail and are not the focus of this manuscript.^{2,5}

Rather we emphasize that despite its low incidence in the general population of pregnant women, both maternal and perinatal morbidity and mortality are significant with amniotic fluid embolism, even in cases ideally managed. Because of the rarity of this condition, most physicians and institutions have limited experience with the management of amniotic fluid embolism.

The purpose of this document is to provide clinicians with information that may improve the ability to make a timely diagnosis and establish appropriate supportive treatment to patients suffering from amniotic fluid embolism to improve maternal and perinatal outcomes.

What is amniotic fluid embolism and what are its clinical features?

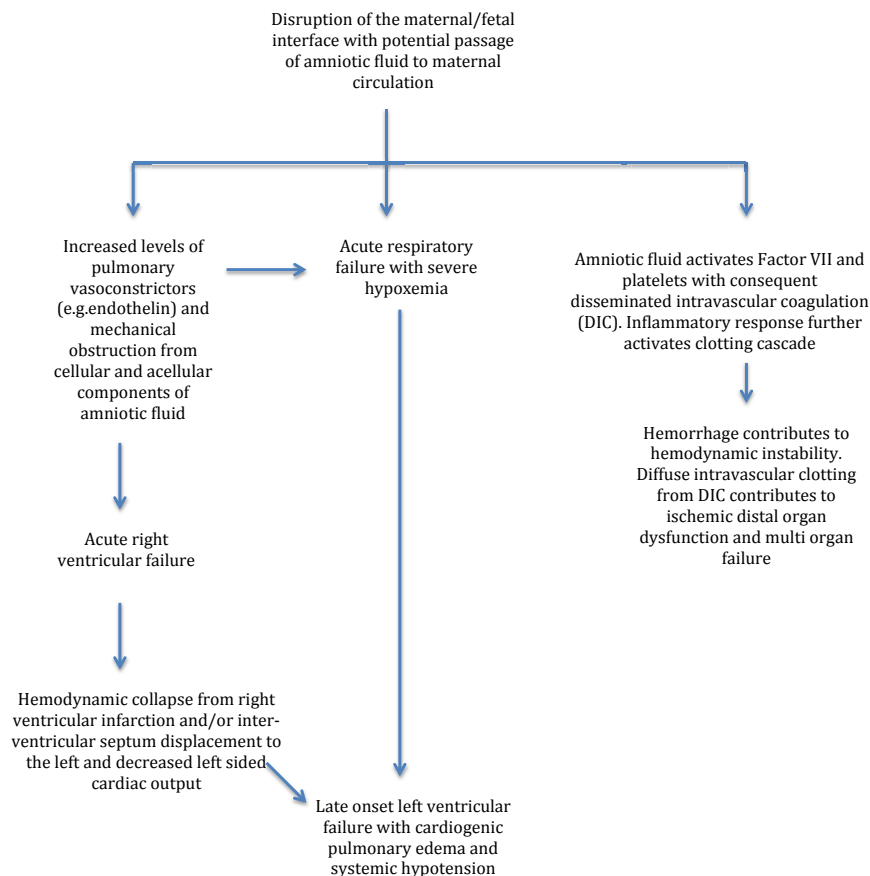
A detailed review of the pathophysiology of amniotic fluid embolism is beyond the scope of this document but may be found elsewhere and is summarized in Figures 1 and 2.^{1,2,5} It appears to involve a complex sequence of events triggered in certain women by entrance into the maternal circulation of

material from the fetal compartment, resulting in an abnormal activation of proinflammatory mediator systems similar to the systemic inflammatory response syndrome.

The typical presentation of amniotic fluid embolism includes a triad of sudden hypoxia and hypotension, followed in many cases by coagulopathy, all occurring in relation to labor and delivery. The diagnosis of amniotic fluid embolism is clinical, based on the presence of these elements and the exclusion of other likely causes. Amniotic fluid embolism should be considered in the differential diagnosis in any pregnant or immediately postpartum woman who suffers sudden cardiovascular collapse or cardiac arrest, seizures, severe respiratory difficulty, or hypoxia, particularly if such events are followed by a coagulopathy that cannot be otherwise explained.

The analysis of the national registry reveals that 70% of cases of amniotic fluid embolism occur during labor, 11% after a vaginal delivery, and 19% during a cesarean delivery.¹ These figures suggest that the mode of delivery may alter the timing of amniotic fluid embolism but not its

FIGURE 1
Proposed pathophysiology of amniotic fluid embolism

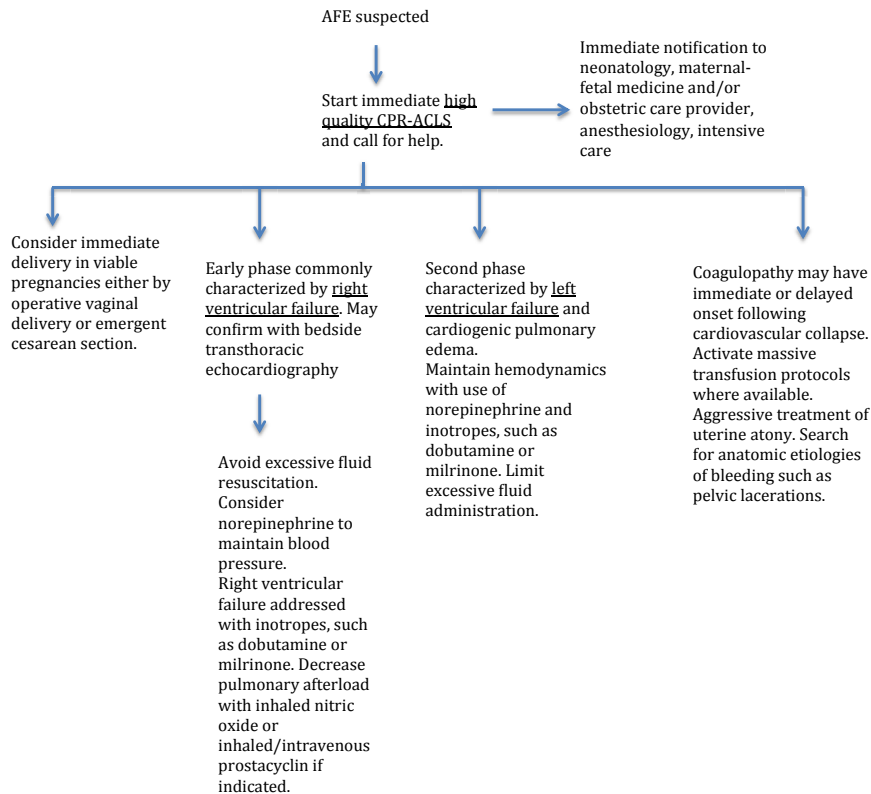


DIC, disseminated intravascular coagulation.

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FIGURE 2

Immediate supportive treatment in suspected cases of amniotic fluid embolism



CPR, cardiopulmonary resuscitation; ACLS, advanced cardiac life support; AFE, amniotic fluid embolism.

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occurrence. In rare instances, amniotic fluid embolism may occur during the first or second trimesters of pregnancy, at the time of pregnancy termination, or amniocentesis.⁶

The clinical presentation of amniotic fluid embolism is, in its classic form, dramatic. A period of anxiety, change in mental status, agitation, and a sensation of doom may precede the event.⁷ Patients may progress rapidly to cardiac arrest, with pulseless electrical activity, asystole, ventricular fibrillation, or pulseless ventricular tachycardia. In cases occurring prior to delivery, electronic fetal monitoring will demonstrate decelerations, loss of variability, and terminal bradycardia as oxygenated blood is shunted away from the uterus, and catecholamine-induced uterine hypertonus causes a further decline in uterine perfusion.^{1,2}

Disseminated intravascular coagulation is present in up to 83% of cases.¹ The coagulopathy of amniotic fluid embolism may occur in conjunction with the cardiopulmonary manifestations, be manifest only after initial cardiopulmonary resuscitation has been completed, or in very rare cases may be the only finding in women without cardiorespiratory compromise.⁸⁻¹⁰

Disseminated intravascular coagulation is commonly manifested by hemorrhagic complications including bleeding from venipunctures or surgical sites, hematuria, gastrointestinal hemorrhage, and vaginal bleeding. As with any condition involving diminished uterine perfusion, coexistence with uterine atony is not uncommon. However, bleeding from incompletely controlled atony followed by hypovolemic shock and either a consumptive or dilutional coagulopathy cannot be attributed to amniotic fluid embolism, nor does amniotic fluid embolism occur as a mild coagulopathy followed hours later by sudden cardiovascular collapse in the absence of interval hemorrhage and hypovolemia.

Reported risk factors for amniotic fluid embolism include situations in which the exchange of fluids between the maternal and fetal compartments is more likely, such as operative delivery (cesarean or vaginal), placenta previa, placenta accreta, and abruption. An association between induction of labor and amniotic fluid embolism is inconsistently reported. Abnormalities of uterine tone (hypo- or hypertonus) described commonly in cases of amniotic fluid embolism may be the consequence of uterine

hypoperfusion secondary to profound maternal shock and hypoxia with massive catecholamine release, rather than the cause.¹

Other putative risk factors include cervical lacerations, uterine rupture, eclampsia, polyhydramnios, and multiple gestations; as outlined in the previous text, a tendency to overdiagnose amniotic fluid embolism in cases actually involving other causes of primary hemorrhage may contribute to these reports. Sociodemographic risk factors such as maternal age and race/ethnicity are also reported in some series.¹¹⁻¹⁶ However, given the rare and unpredictable nature of amniotic fluid embolism, there are no risk factors sufficiently established to justify any alteration in standard obstetric care.

How should you manage a patient with sudden cardiac arrest in whom amniotic fluid embolism is suspected?

Amniotic fluid embolism should be considered in the differential diagnosis of sudden cardiorespiratory compromise in any pregnant or recently postpartum patient (GRADE 1C). Initial resuscitation of cardiac arrest does not require a specific diagnosis of amniotic fluid embolism because initial maternal treatment (with basic cardiac life support and advanced cardiac life support protocols) is similar, regardless of the exact etiology.

We do not recommend the use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of amniotic fluid embolism; at the present time, amniotic fluid embolism remains a clinical diagnosis (GRADE 1C).

We recommend the provision of immediate high-quality cardiopulmonary resuscitation with standard basic cardiac life support and advanced cardiac life support protocols in patients who develop cardiac arrest associated with amniotic fluid embolism (GRADE 1C). We recommend that a multidisciplinary team including anesthesia, respiratory therapy, critical care, and maternal-fetal medicine should be involved in the ongoing care of women with AFE (Best Practice). The most critical immediate action is to start chest compressions before rescue breathing is administered.¹⁷

Chest compressions should be performed similarly to nonpregnant individuals. The hands of the provider should be placed in the lower half of the sternum.¹⁷ Chest compressions should be performed hard and fast, achieving a depth of at least 2 inches and allowing complete chest recoil between compressions. Patients who are undelivered should be tilted to the left lateral decubitus position or, preferably, have the uterus displaced laterally by an assistant to prevent aortocaval compression by the gravid uterus.¹⁷

The use of vasopressors, antiarrhythmic agents, and defibrillating doses is not different from those utilized in nonpregnant individuals. Although concerns that electric arcing may occur if fetal monitors are in place at the time of cardioversion or defibrillation are largely theoretical, it is reasonable to remove such monitors while cardiopulmonary

resuscitation is in progress. However, the presence of such monitors should not delay defibrillation when indicated.¹⁷ The components of high-quality cardiopulmonary resuscitation are summarized in [Table 1](#).

If the patient is undelivered at the time of cardiac arrest, expeditious delivery is indicated if the fetus has reached an age of potential viability (≥ 23 weeks). Not only may this be life saving for the fetus but in theory may assist in maternal resuscitation by removing venacaval compression. An operative vaginal delivery (forceps or vacuum assisted) should be performed in laboring patients in whom obstetrical conditions support such an intervention. If a vaginal delivery is not an option, emergency cesarean delivery is generally indicated.

Classically the indication for a perimortem cesarean delivery has been a failure to obtain spontaneous circulation after 4 minutes of cardiopulmonary resuscitation to reduce the profound fetal hypoxia occurring during maternal cardiac arrest.¹⁷ This time frame is ideal but is rarely achievable when cardiac arrest is unexpected.

We recommend that preparations for emergent perimortem cesarean delivery be initiated simultaneously with the initiation of cardiopulmonary resuscitation, and if the cardiac arrest is still ongoing as the instruments become available, proceed with cesarean delivery. The dismal prognosis of adult cardiac arrest not amenable to, or unresponsive to, immediate direct current countershock suggests that maternal prognosis will not be significantly compromised by such an operation.

Some authors recommend moving this threshold to 20 weeks to improve maternal perfusion, but no evidence exists that such previsible cesarean delivery improves the outcome in cases of amniotic fluid embolism-related maternal cardiac arrest.¹⁷

Following cardiac arrest with amniotic fluid embolism, we recommend immediate delivery in the presence of a fetus ≥ 23 weeks of gestation (GRADE 2C). In cases of maternal hemodynamic instability that does not involve one of the lethal dysrhythmias, cases must be individualized based on the

TABLE 1
Components of high-quality cardiopulmonary resuscitation in pregnancy

Components

- Rapid chest compressions (100 \times minute)
- Perform hard compressions, achieving a depth of at least 2 inches
- Assure adequate chest recoil between compressions
- Minimize interruptions of chest compressions
- Avoid prolonged pulse checks (no more than 5–10 seconds)
- Resume chest compressions immediately after defibrillating
- Switch provider of compressions every 2 minutes to avoid fatigue
- Lateral displacement of uterus during resuscitation

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fetal age and degree of compromise, maternal condition, and the availability of anesthetic support. No data exist to guide delivery decisions under these circumstances.

The literature contains innumerable case reports in which various novel therapeutic modalities have been used in women with presumptive amniotic fluid embolism, and the patient did not die.⁷ Unfortunately, evidence of a causal, as opposed to an anecdotal, connection between most of these and survival from amniotic fluid embolism is lacking. We focus here only on the better-supported ancillary treatment options.

The use of venoarterial extracorporeal membrane oxygenation has been described in cases of amniotic fluid embolism refractory to conventional resuscitation maneuvers.⁷ However, the use of anticoagulation during extracorporeal membrane oxygenation may worsen bleeding in the profoundly coagulopathic patient with active hemorrhage. Because of these concerns, as well as lack of adequate evidence of benefit, extracorporeal membrane oxygenation is controversial and not routinely recommended in the management of amniotic fluid embolism.

After successful resuscitation, postcardiac arrest management is of paramount importance.¹⁸ Hemodynamic instability is common, and patients may require fluids, vasopressors, and inotropes. The goal is to maintain a mean arterial blood pressure of 65 mm Hg.¹⁸ Fever may worsen ischemia-reperfusion injury to the brain and should be aggressively treated. Hyperoxia will also worsen ischemia-reperfusion injury, and administration of 100% oxygen after the survival of cardiac arrest should be avoided. The inspired fraction of oxygen should be weaned to maintain a pulse oxymetry value of 94–98%.¹⁸ Serum glucose should be maintained between 140 mg/dL and 180 mg/dL with the use of intravenous insulin infusions if needed.¹⁹

Mild therapeutic hypothermia, defined as cooling the patient to a temperature between 32°C and 34°C for 12–24 hours, has been recommended by the American Heart Association to increase the rate of a favorable neurological outcome and reduce mortality following cardiac arrest.¹⁸ A recent study, however, found no differences in outcomes between targeted temperatures of 33°C vs 36°C among survivors of cardiac arrest who were treated with mild therapeutic hypothermia.²⁰ Current guidelines recommend targeted temperature management of cardiac arrest victims aiming at temperatures between 32° and 36°C.¹⁸

The data on therapeutic hypothermia during pregnancy is limited to case reports.^{21,22} Most survivors of amniotic fluid embolism will have been delivered during the course of successful resuscitation. The main concern limiting the use of therapeutic hypothermia in these patients is a concern that this may increase the risk of hemorrhage. In patients not demonstrating significant disseminated intravascular coagulation and bleeding, therapeutic hypothermia should

be considered. Targeting a temperature of 36°C (as opposed to lower temperatures with a concomitant increased risk of hemorrhage) is an option. Such decisions must be made in conjunction with the available medical critical care team.

What are the considerations for hemodynamic support in a patient with amniotic fluid embolism?

Immediate survivors of amniotic fluid embolism require multidisciplinary management, including maternal-fetal medicine subspecialists and intensive care specialists. The management of suspected amniotic fluid embolism is supportive and focuses on rapid maternal hemodynamic stabilization.

The initial phase of amniotic fluid embolism consists mainly of right ventricular failure. If available, transthoracic and/or transesophageal echocardiography may provide valuable information. Immediately after presentation, the echocardiography will commonly reveal a severely dilated hypokinetic right ventricle (acute cor pulmonale) with deviation of the interventricular septum into the left ventricle. Hypoxia, acidosis, and hypercapnia should be avoided because they increase pulmonary vascular resistance and lead to further right heart failure.¹³ Right ventricular output may be improved by using inotropes such as dobutamine and milrinone. These agents also will lead to pulmonary vasodilation.

Other specific interventions aimed at decreasing the pulmonary vascular resistance include sildenafil, inhaled or intravenous prostacyclin, and inhaled nitric oxide. Hypotension in this phase should be mainly treated with vasopressors such as norepinephrine or vasopressin.²³ The commonly used doses of these agents are provided in [Table 2](#).

We recommend the provision of adequate oxygenation and ventilation and, when indicated by hemodynamic status, the use of vasopressors and inotropic agents in the initial management of amniotic fluid embolism. Excessive fluid administration should be avoided (GRADE 1C).

After an initial phase of right ventricular failure, left ventricular failure predominates. Excess fluid administration should be especially avoided in the setting of a massively dilated right ventricle because this will increase the overdistention of the ventricle and increase the risk of a right-sided myocardial infarction. Increased distention of the right ventricle will also displace the interventricular septum to the left, further compromising the cardiac output because of left ventricular obliteration.

Minutes to hours following the initial presentation, the right ventricular function usually improves, and left ventricular failure with cardiogenic pulmonary edema becomes the prominent finding.⁷ In patients who are not intubated, noninvasive mechanical ventilation or endotracheal intubation should be considered. Left-sided heart failure should be treated by optimizing cardiac preload, the use of

TABLE 2
Recommended doses for agents commonly used in cases of acute right ventricular failure

Agent	Dose
Sildenafil	20 mg tid PO or through nasogastric/orogastric tube
Dobutamine	2.5–5.0 $\mu\text{g}/\text{kg}$ per minute. Higher doses may compromise right ventricular filling time caused by tachycardia.
Milrinone	0.25–0.75 $\mu\text{g}/\text{kg}$ per minute. Most common side effect is systemic hypotension.
Inhaled nitric oxide	5–40 ppm. Follow methemoglobin levels every 6 h, and avoid abrupt discontinuation.
Inhaled prostacyclin	10–50 ng/kg per minute
Intravenous prostacyclin	Start at 1–2 ng/kg per minute through a central line and titrate to desired effect. Side effects include systemic hypotension, nausea, vomiting, headache, jaw pain, and diarrhea.
Norepinephrine	0.05–3.3 $\mu\text{g}/\text{kg}$ per minute

PO, per os; tid, twice a day.

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vasopressors in cases of hypotension to maintain coronary perfusion pressure, and inotropes (dobutamine or milrinone) to increase left ventricular contractility. Severe pulmonary congestion not responsive to diuretic therapy may require fluid removal through dialysis.

Later in the clinical course, some patients with persistent severe inflammation and prolonged care in the intensive care unit may develop nosocomial infections and distributive shock with noncardiogenic pulmonary edema from severe sepsis.²⁴ No evidence exists to justify the routine use of steroids in cases of amniotic fluid embolism. The overall management of amniotic fluid embolism is summarized in Figure 2.

How is the coagulopathy associated with amniotic fluid embolism managed?

Disseminated intravascular coagulation is present in most cases of amniotic fluid embolism. The onset is variable; disseminated intravascular coagulation may be manifest either immediately following cardiovascular collapse or in the later phases of the syndrome.

Severe hemorrhage may require simultaneous medical and surgical approaches. Medical management classically includes the administration of blood products to maintain a platelet count above 50,000/mm³ and normal (or close to normal) activated partial thromboplastin time and international normalized ratio.

In the setting of massive hemorrhage, blood product administration should not be delayed while awaiting the

results of laboratory tests. Instead, early aggressive resuscitation with packed red blood cells, fresh-frozen plasma, and platelets at a ratio of 1:1:1 (hemostatic resuscitation) results in improved outcomes.²⁵

Because coagulopathy may follow cardiovascular collapse with amniotic fluid embolism, we recommend the early assessment of clotting status and early aggressive management of clinical bleeding with standard massive transfusion protocols (GRADE 1C).

Administration of recombinant activated factor VII has been described in cases of amniotic fluid embolism.²⁶⁻²⁸ However, some authors believe that this treatment, in patients with disseminated intravascular coagulation and elevated levels of tissue factor (as occurs in amniotic fluid embolism), could lead to excessive diffuse thrombosis and multiorgan failure. The use of this agent may be considered as a last resort in cases in which hemorrhage cannot be stopped with massive blood component replacement and surgical interventions.²⁷

Both plasminogen activators and plasminogen activator inhibitors have been identified in amniotic fluid.²⁹ The presence of hyperfibrinolysis has been described in amniotic fluid embolism–related coagulopathy and should be considered in the management of amniotic fluid embolism. When available, bedside thromboelastography may aid in identifying bleeding patients who might benefit from the use of antifibrinolytics such as tranexamic acid or epsilon aminocaproic acid.³⁰

Uterine atony is common with amniotic fluid embolism and should be managed aggressively.³¹ The use of uterotonics such as oxytocin, ergot derivatives, and prostaglandins is appropriate when indicated. Refractory cases may require uterine tamponade with the use of packing or commercially available intrauterine balloons. Extreme cases may need bilateral uterine artery ligation, B-Lynch stitch, or hysterectomy. We caution, however, against making the diagnosis of amniotic fluid embolism based exclusively on hemorrhage from persistent atony with secondary coagulopathy; in our experience, this is a common diagnostic error.

In patients delivered vaginally, a thorough inspection of the cervix and vagina is warranted to rule out lacerations as the cause or as a contributing factor to profuse bleeding in a patient with disseminated intravascular coagulation. In patients with diffuse bleeding after or during a cesarean delivery that is not amenable to surgical control, consideration should be given to packing the pelvis and transfer to the intensive care unit for further medical therapy with delayed closure.

What other differential diagnoses should be considered when amniotic fluid embolism is suspected?

In the absence of the classic triad of hypotension, hypoxia, and subsequent coagulopathy, amniotic fluid embolism often remains a diagnosis of exclusion. The list

of conditions that may result in either acute cardiac or respiratory or hematological failure in pregnancy is relatively long and includes myocardial infarction, pulmonary embolism, air embolism, anesthetic complications, anaphylaxis, and eclampsia and in some cases sepsis. Providers caring for pregnant women with an acute clinical event and cardiorespiratory failure should narrow this list to clinically relevant diagnoses that require specific treatment strategies. Importantly, an exact diagnosis is not required to start treatment for presumed amniotic fluid embolism because immediate interventions, such as good-quality cardiorespiratory resuscitation, are supportive in nature.

Risk factors for myocardial infarction, such as advanced maternal age, diabetes, chronic hypertension, smoking, obesity, dyslipidemia, and a previous history of coronary artery disease may suggest this diagnosis. Cardiac troponins and a 12 lead electrocardiograph should be obtained as soon as possible. A bedside echocardiography may be useful in making a diagnosis of cardiogenic shock secondary to myocardial ischemia. Echocardiography will also aid in ruling out conditions such as a peripartum dilated cardiomyopathy.³²

Pulmonary embolism is a recognized complication of pregnancy. Computed tomography angiography or a ventilation perfusion scan may be useful in evaluating this potential diagnosis. In cases complicated by profuse bleeding, however, thromboembolism is unlikely.

A high spinal anesthesia can result in apnea but is unlikely to cause a dramatic drop in cardiac output or hemorrhagic manifestations. Inadvertent intravascular injection of local anesthetics may cause seizures and cardiovascular collapse.³³ Timing between injection and onset of symptoms may suggest this diagnosis or make it less likely. If local anesthetic toxicity is likely, consideration should be given to the use of intravenous lipids (20% Intralipid) in addition to other supportive measures.³⁴

Air embolism may also cause acute cardiorespiratory compromise. If venous air embolism is high on the list of potential diagnoses, normobaric 100% oxygen should be used. The patient should be turned to the left lateral decubitus to prevent air from migrating to the pulmonary artery. If a central line is in place, then aspiration of blood may be performed in an attempt to aspirate bubbles of air. If an arterial air embolism is suspected (eg, if neurological symptoms are present), hyperbaric oxygen therapy should be considered, if available.

Eclampsia is obviously a possibility in a patient in the latter half of pregnancy with new-onset seizures, although eclampsia is not commonly associated with cardiorespiratory arrest and acute profound coagulopathy. Transfusion reactions may cause acute pulmonary edema (transfusion-related acute lung injury) and coagulopathy when incompatible blood is administered. This is an uncommon event in modern practice.

Anaphylactic shock is a possibility, particularly in the setting of urticarial rash, and laryngospasm or bronchospasm immediately following the administration of medication known to cause anaphylaxis. Bronchospasm has been reported in about 15% of cases of amniotic fluid embolism. However, anaphylaxis is not usually accompanied by coagulopathy, and cardiac dysfunction is not commonly severe because hypotension associated with anaphylaxis is due primarily to vasodilation and increased vascular permeability. If anaphylaxis is suspected, treatment with epinephrine, steroids, and inhaled bronchodilators is indicated.

Bedside echocardiography demonstrating right ventricular dysfunction favors the diagnosis of amniotic fluid embolism over anaphylaxis and most of the other conditions that may mimic amniotic fluid embolism.

What is the prognosis and recurrence risk for patients who survive an episode of amniotic fluid embolism?

The recurrence rate of amniotic fluid embolism is difficult to define because of the rarity of the condition and high mortality rate. Multiple cases of uneventful subsequent pregnancies and no cases of recurrence have been reported.^{35,36} Patients should be cautioned, however, that the available sample size precludes definitive conclusions regarding recurrence risk.

Conclusions

Amniotic fluid embolism is a rare but often lethal condition. Maternal and perinatal mortalities appear to have decreased during the last decades likely because of improvements in the delivery of critical care, recognition of atypical or milder cases with no cardiorespiratory collapse, and the likely inclusion of patients with conditions other than amniotic fluid embolism, particularly in series based on administrative data.

The diagnosis remains clinical and is often one of exclusion because no single specific diagnostic test is currently available. Treatment is mainly supportive and involves the delivery of the fetus when indicated, respiratory support (usually in the form of endotracheal intubation and mechanical ventilation), and hemodynamic support with the judicious use of fluids, vasopressors, inotropes, and pulmonary vasodilators. Rapid initiation of treatment, aided by a high index of clinical suspicion, is essential.

The recurrence rate of amniotic fluid embolism is unknown but appears to be low. Much of the published literature regarding amniotic fluid embolism is of poor quality and likely includes a significant number of patients with other conditions. Uniform diagnostic criteria for amniotic fluid embolism cases reported in research publications are badly needed and may accelerate our understanding of this condition. ■

Summary of Recommendations

Number	Recommendations	GRADE
1	We recommend consideration of AFE in the differential diagnosis of sudden cardiorespiratory collapse in the laboring or recently delivered woman.	1C Strong recommendation Weak-quality evidence
2	We do not recommend the use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of AFE; at the present time, AFE remains a clinical diagnosis.	1C Strong recommendation Weak-quality evidence
3	We recommend the provision of immediate high-quality cardiopulmonary resuscitation with standard BCLS and ACLS protocols in patients who develop cardiac arrest associated with AFE.	1C Strong recommendation Weak-quality evidence
4	We recommend that a multidisciplinary team including anesthesia, respiratory therapy, critical care, and maternal-fetal medicine should be involved in ongoing care of women with AFE.	Best practice
5	Following cardiac arrest with AFE, we recommend immediate delivery in the presence of a fetus ≥ 23 weeks of gestation.	2C Weak recommendation Weak-quality evidence
6	We recommend the provision of adequate oxygenation and ventilation and, when indicated by hemodynamic status, the use of vasopressors and inotropic agents in the initial management of AFE. Excessive fluid administration should be avoided.	1 C Strong recommendation, Weak-quality evidence
7	Because coagulopathy may follow cardiovascular collapse with AFE, we recommend early assessment of clotting status and early aggressive management of clinical bleeding with standard massive transfusion protocols.	1C Strong recommendation, Weak-quality evidence

ACLS, advanced cardiac life support; AFE, amniotic fluid embolism; BCLS, basic cardiac life support; GRADE, Grading of Recommendations Assessment, Development, and Evaluation. SMFM. Amniotic fluid embolism: diagnosis and management. *Am J Obstet Gynecol* 2016.

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