MATERNAL911

Amniotic Fluid Embolism

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Maternal 911 Education Systems, LLC
475 West Center St.
Ithaca, MI 48847
www.maternal911.com

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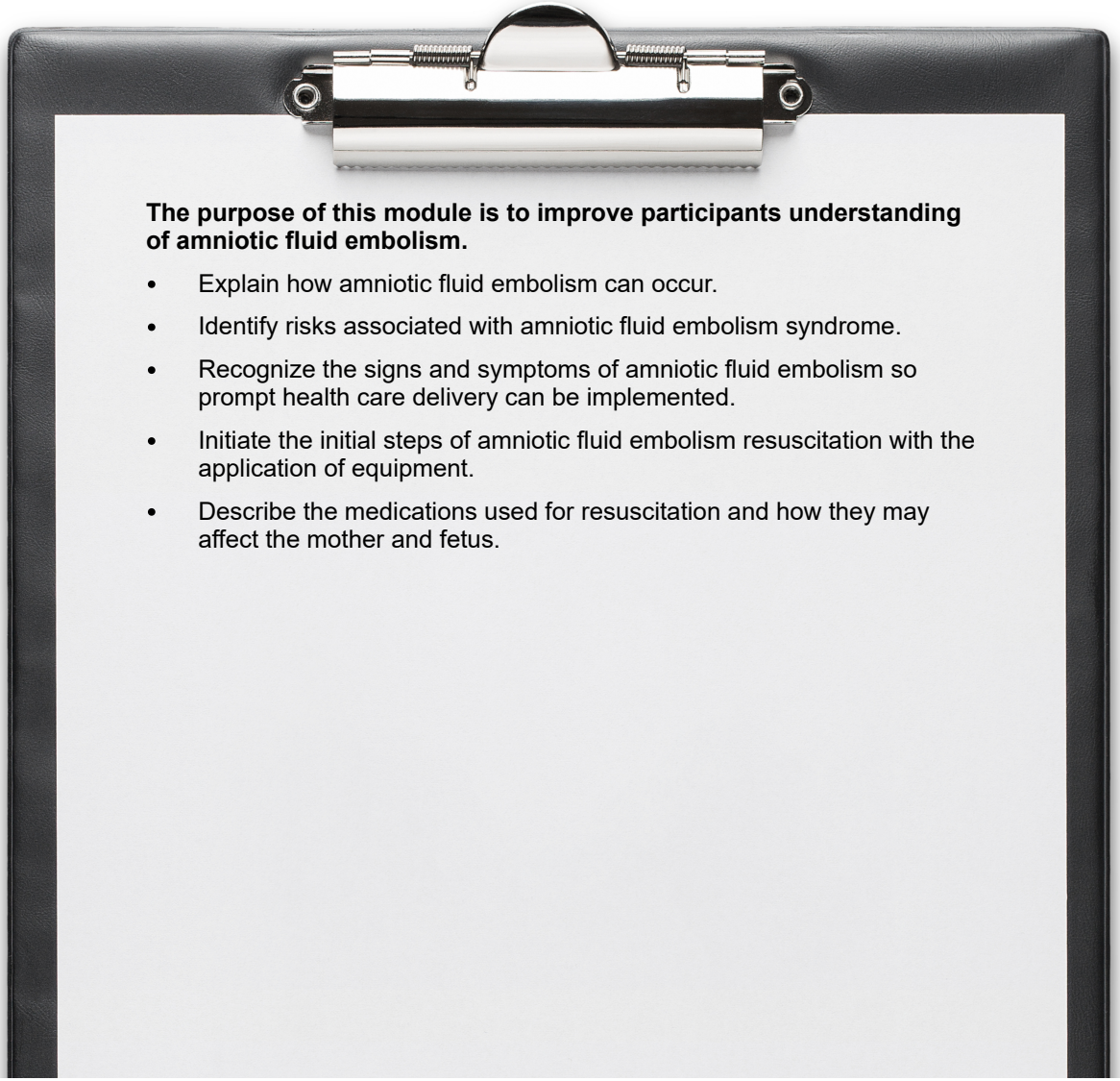
Course Description:

Amniotic Fluid Embolism (AFE) is a high acuity, low occurring process that when recognized may be life-saving. The course will help by giving understanding of the disease and its management. AFE is a detrimental disease process that is life threatening for the women it effects. The AFE module will provide knowledge for this low occurring process.

Approximate Time to Complete: 40 minutes

Click here to download a print version of this course.



The purpose of this module is to improve participants understanding of amniotic fluid embolism.

- Explain how amniotic fluid embolism can occur.
- Identify risks associated with amniotic fluid embolism syndrome.
- Recognize the signs and symptoms of amniotic fluid embolism so prompt health care delivery can be implemented.
- Initiate the initial steps of amniotic fluid embolism resuscitation with the application of equipment.
- Describe the medications used for resuscitation and how they may affect the mother and fetus.

Amniotic fluid embolism syndrome (AFES)

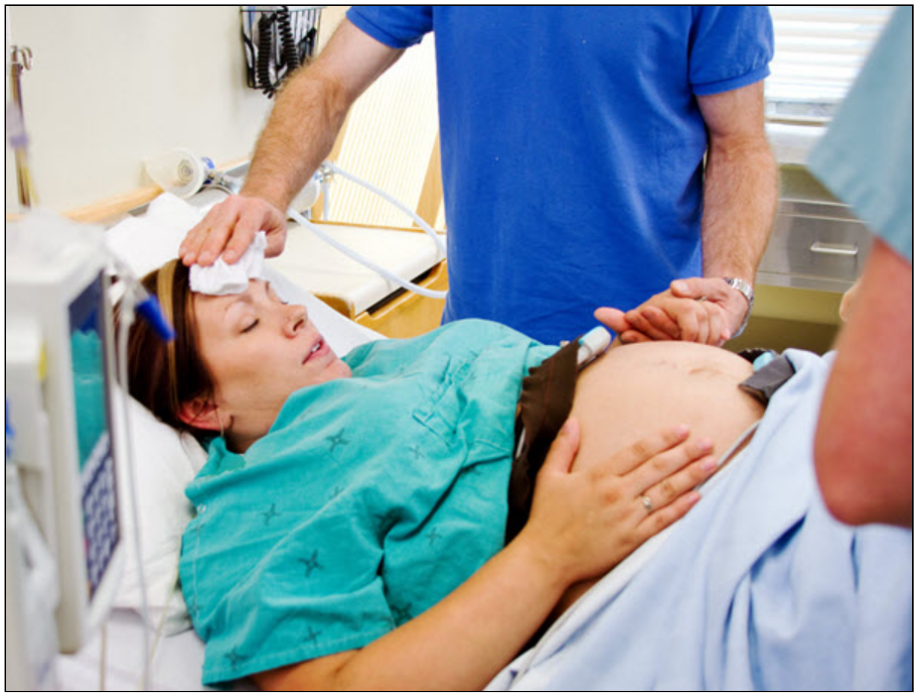
- *Also called anaphylactiod syndrome of pregnancy.*
- *A catastrophic condition that occurs during pregnancy or shortly after delivery [1-3].*

- First reported in 1926
- Not widely recognized until 1941.
- In 1941 autopsy's in a series of eight women dying from sudden shock during labor reported squamous cells and mucin of fetal origin in the maternal pulmonary vasculature [4,5].
- These same fetal squamous cells have been described in the vascular beds of maternal renals, liver, spleen, pancreas and brain [6].

- AFES is different from venous thromboembolism and is reviewed in a separate module.
- The incidence rate is between 1 - 12 cases per 100,000 deliveries in most studies [7-12].

Several factors have been associated with AFES [7,12,13] they include:

- precipitous or tumultuous labor
- advanced maternal age
- cesarean and instrumental delivery
- placenta previa, placental abruption, placenta acreta, percreta, or increta
- grand multiparity (≥ 5 live births or stillbirths)
- cervical lacerations
- fetal distress
- eclampsia
- medical induction of labor
- uterine rupture
- polyhydramnios
- miscarriage or abortion
- amniocentesis

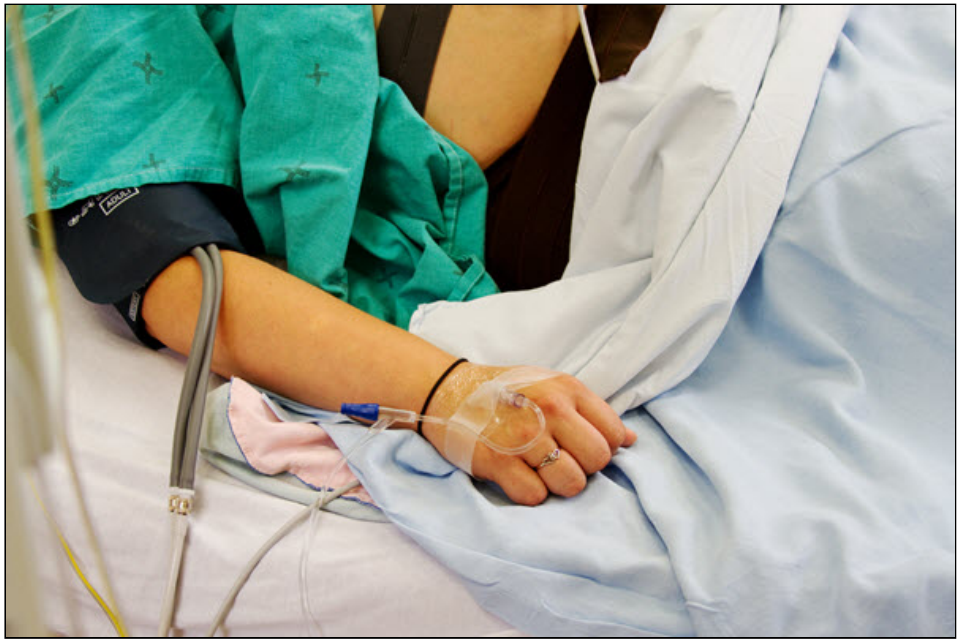


These factors are associated with the pathogenesis of AFES but not the direct cause.

AFES is best considered unpreventable and unpredictable.

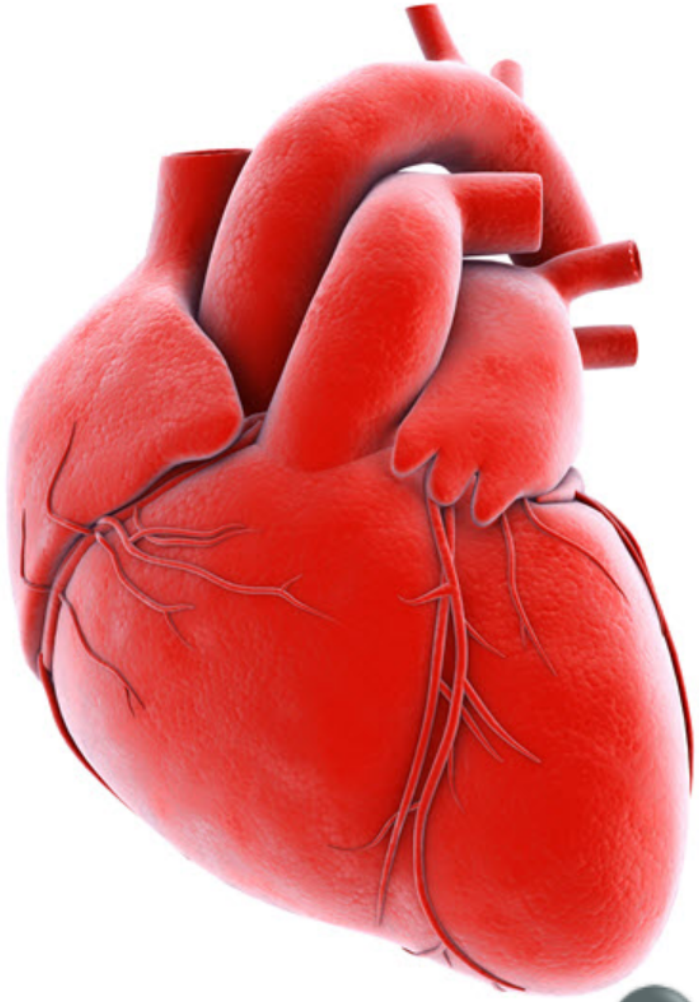
Stage or duration of labor has not been associated with risk for AFE.

- The pathogenesis of AFES is thought to involve the amniotic fluid entering the maternal circulation through endocervical veins, the placental insertion site or a site of uterine trauma [14].
- Once the amniotic fluid reaches the maternal circulation it can precipitate cardiogenic shock, respiratory failure and most likely, an inflammatory and anaphylactoid response.



- Amniotic fluid has been shown to cause occlusion and vasospasm of the maternal pulmonary vasculature with animal studies, resulting in rapid development of pulmonary hypertension, acute cor pulmonale and systemic hypotension [15-17].
  - In women with AFES, invasive hemodynamic studies seem to contradict these animal studies [18,19].
  - When elevated pulmonary arterial pressure, elevated pulmonary capillary wedge pressure, decreased cardiac output and decreased cardiac index are combined it suggests the principal hemodynamic alteration in humans is left ventricular failure, rather than pulmonary hypertension and right ventricular failure.

- To reconcile the human and animal observations, a biphasic pattern of cardiogenic shock in AFES has been proposed [2,15,19].
- Initial acute pulmonary hypertension and right ventricular failure (usually lasting 15-30 minutes), according to the hypothesis, is followed by left ventricular
dysfunction [20,21].
- Studies utilizing transesophageal echocardiography to non-invasively measure the hemodynamic parameters early during AFES report vasospasm of the pulmonary vasculature, elevated pulmonary arterial pressure and right ventricular failure, thus supporting the bi-phasic hypothesis [20,21].
- The mechanism is unclear with the later phase left ventricular failure.



Mouse over the heart to see what the mechanism may involve.

- Among patients with AFES, hypoxemia is the most common manifestation of the resultant respiratory failure.
- Hypoventilation can also occur.
- The primary cause of hypoxemia involves severe ventilation/perfusion (V/Q) mismatching.

Factors contributing to the V/Q mismatch [2] :

- Acute pulmonary hypertension during the first phase of cardiogenic shock
- Cardiogenic pulmonary edema during the second phase

Other contributors may include [8,22] :

- Bronchospasm (about 15 percent of patients)
- Noncardiogenic pulmonary edema

- In 70% of patients who survive the first several hours, noncardiogenic pulmonary edema occurs [22].
- It generally develops as left ventricular dysfunction improves.
- Damage to the endothelial-alveolar membrane and capillary leak syndrome are evidence leading to high protein concentration in edematous fluid and the presence of amniotic debris in sputum and alveolar spaces.
- Widespread damage to the alveolar-capillary membrane causes non-cardiogenic edema to occur in AFES; it usually does not produce the clinical pattern typical of acute respiratory distress syndrome (ARDS).
- Women who survive the first few hours of AFES usually recover quite rapidly, whereas the course of ARDS tends to be protracted.

- Obstruction of the pulmonary vasculature seems unlikely to be the lone cause of AFES, since there is often a lag of many hours between the entry of amniotic fluid into the the maternal circulation and onset of symtoms and signs of AFES.
- Propositions have brought up how the lag may reflect evolution of AFES of an immunologic response or inflammatory reaction to the amniotic fluid.
- Support is evident by reports of decreased complement and increased inflammatory markers in some patients with AFES, including elevated serum tryptase levels and pulmonary mast cell activity [23-28].
- It is hypothesized that maternal circulation allows entry of fetal antigens via the amniotic fluid.
- The clinical manifestations and the severity may be related to the degree of immunologic stimulation or the balance of arachidonic acid metabolites such as leukotrienes in the amniotic fluid [29,30].

- The symptoms and signs associated with AFES have a typical onset during labor and delivery, or immediately postpartum [2].
- In rare instances it has been reported following first or second trimester abortion, amniocentesis, or abdominal/uterine trauma and as late as 48 hours after cesarean delivery or post partum, [8.31-35].
- In review of 272 cases, most women present with rapid cardiorespiratory collapse [10].
- Preceding the onset of dyspnea and hypotension may be nonspecific symptoms such as chills, nausea, vomiting and agitation [8,10].
- Tonic-clonic seizure activity may also occur.
- A less severe presentation of AFES, partial amniotic fluid embolism syndrome, may present with only some of the major symptoms and signs [15,36,37].
- These patients with partial amniotic fluid embolism generally present with sudden onset of milder dyspnea and hypotension.
- In this situation, the clinical course tends to be abbreviated and the prognosis much better compared to those who have the full syndrome.

Click the terms in blue to learn more.

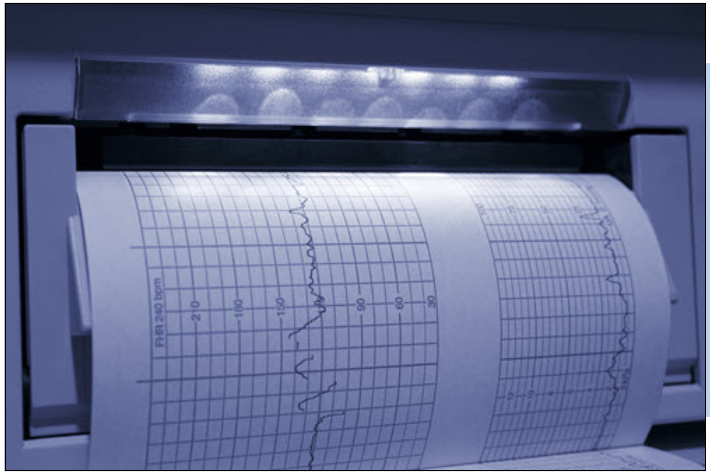
The major clinical findings are the abrupt and fulminant onset of:

- Hypotension due to cardiogenic shock
- Disseminated intravascular coagulation (DIC)
- Hypoxemia and respiratory failure
- Coma or seizures

AFES is diagnosed from clinical findings based on the constellation of clinical findings, rather than isolated signs and symptoms.

- AFES should be suspected whenever shock and/or respiratory compromise develops during labor or immediately postpartum.
- Other causes of sudden intrapartum or postpartum cardiorespiratory failure must be excluded.
- Sometimes the identification of amniotic fluid debris (squamous cells, trophoblastic cells, mucin, and lanugo) from blood samples drawn from the distal port of a pulmonary artery catheter.
- Such debris is common in maternal circulation without AFES, so finding the amniotic fluid debris alone should not be considered diagnostic [43] but should involve the constellation of signs and symptoms.

- There is no specific treatment for AFES.
- The therapeutic goal is to correct hypoxemia and hypotension so that ischemic consequences (i.e. hypoxic brain injury, acute kidney injury) are prevented in the mother and adequate oxygen delivery occurs to the fetus.



- Monitoring of maternal oxyhemoglobin saturation, heart rate and rhythm, and respiratory rate should be immediately initiated in all patients with suspected AFES.
- It is important to monitor the blood pressure non-invasively at frequent intervals until continuous blood pressure monitoring is established.
- Continuous monitoring of the fetal heart rate is desirable.

Catheters

- Following the initial assessment, plans should be made for both an arterial and a central venous catheter to be inserted.
- The initiation of therapies described below should not be delayed for catheter insertion as these procedure can be time consuming.
- The arterial catheter can be used to continuously monitor blood pressure.
- It also provides access to arterial blood for frequent measurement of arterial blood gases.