

WHAT IS AFE?

Amniotic fluid embolism (AFE) is characterized by acute and rapid collapse of mother and/or baby as a result of an anaphylactic-like reaction to the entrance of amniotic fluid or fetal debris entering the maternal circulatory system; a normally benign result of delivery. Although rare, it is often fatal and remains a leading cause of maternal mortality and morbidity in the United States.

INCIDENCE

Clinical series based on population or administrative databases that do not include individual chart review by individuals with expertise in critical care obstetrics are likely to overestimate the incidence and underestimate the mortality of this condition. The reported incidence of AFE ranges from 1.9 to 6.1 per 100,000 deliveries. Based on careful review of current literature by experts, it is more likely to be 2.5 per 100,000 or 1 in 40,000. The mortality rate is estimated to range from 20-60% depending on the severity of symptoms.

RISK FACTORS

Data regarding the presence of risk factors for amniotic fluid embolism are inconsistent and contradictory; at present, no putative risk factor has been identified that would justify modification of standard obstetric practice to reduce the risk of this condition.

PATHOPHYSIOLOGY

The pathophysiology appears to involve an abnormal maternal response to fetal tissue exposure associated with breaches of the maternal-fetal physiologic barrier during parturition. This response and its subsequent injury appear to involve activation of pro-inflammatory mediators similar to that seen with the classic systemic inflammatory response syndrome.

CLINICAL PRESENTATION

Amniotic fluid embolism most often occurs during labor, delivery or shortly afterward and can cause a severe, rapid decline in the mother's health. In classic cases of AFE, women will exhibit a triad of symptoms including: hypoxia, hypotension and coagulopathy resulting in sudden cardiovascular collapse or cardiac arrest. In atypical cases, one or more of these signs may be absent.

Initial symptoms may include increased anxiety, agitation, impending sense of doom, confusion, nausea, or shortness of breath that are accompanied by abnormal vital signs, loss of consciousness, seizure, and/or cardiopulmonary arrest. Fetal hypoxia and FHR abnormalities often precede maternal cardiopulmonary manifestations. Coagulopathy is also a major component of AFE, although some patients may expire before their clotting status can be assessed.

DIAGNOSIS

Diagnosis of AFE is a clinical one. Identification of elements of the classic triad of hypotension, hypoxia, and coagulopathy as well as the careful exclusion of other conditions is essential. Presence of fetal squames in the maternal circulation is no longer diagnostically definitive.

KEY POINTS:

- **SUSPECT AFE WITH CLASSIC TRIAD:** hypoxia, hypotension, and coagulopathy
- **TREATMENT IS AGGRESSIVE & SUPPORTIVE**
- **AFE IS A DIAGNOSIS OF EXCLUSION & OFTEN OVER DIAGNOSED**

MANAGEMENT

Treatment is aggressive and supportive. If AFE is suspected the first step is to provide high-quality cardiopulmonary resuscitation. Left lateral uterine displacement or delivery during resuscitation efforts may increase cardiac preload and improve the effectiveness of CPR by relieving inferior vena cava pressure caused by the gravid uterus. Intubation will likely be needed for ongoing respiratory support. Even prior to clinical signs of hemorrhage, activating a massive transfusion protocol is recommended.

RECURRENCE

Multiple case studies have reported no recurrence however, due to the rarity of the condition, limited data, and common diagnostic confusion, there are no definitive conclusions regarding risk of recurrence.

CURRENT RESEARCH

Clinicians are encouraged to assist in the enrollment of cases into the The Amniotic Fluid Embolism Patient Registry™, a research database at Baylor College of Medicine in collaboration with the AFE Foundation. Immediate cases (<7 days) may be eligible for specimen research. More information can be found at www.afesupport.org.

REFERENCES & FURTHER READING

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