AFE AMNIOTIC FLUID EMBOLISM FOUNDATION
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AMNIOTIC FLUID EMBOLISM
CLINICAL FACT SHEET

WHAT IS AFE?

Amniotic Fluid Embolism (also referred to as anaphy lactoid syndrome of pregnancy) remains one of the most devastating conditions in obstetric practice with an incidence of approximately 1 in 40,000 deliv eries and a reported mortality rate ranging from 20% to 60%. It is a leading cause of maternal mor tality and morbidity in the United States and abroad.

KEY POINTS:

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

INCIDENCE AND RISK FACTORS

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 both overestimate the incidence and underestimate the mortality of this condition by the inclusion of women who did not have amniotic fluid embolism. 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 are essential. Presence of fetal squames in the maternal circulation is no longer diagnostically definitive.

MANAGEMENT

Treatment is aggressive and supportive. A multidisciplinary approach is ideal. If AFE is suspected the first step is to provide high-quality cardiopulmonary resuscitation and expeditious delivery if not yet delivered. 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.

CURRENT RESEARCH

The Amniotic Fluid Embolism Patient Registry ™ is an ongoing prospective and retrospective research database through Baylor College of Medicine and the AFE Foundation. Immediate cases (<7 days) may be eligible for specimen research. More information can be found at www.afesupport.org

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