Amniotic Fluid Embolism

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KEYWORDS

• Amniotic fluid embolism • Cardiorespiratory arrest • Pregnancy
• Disseminated intravascular coagulopathy • Maternal death

KEY POINTS

- Amniotic fluid embolism remains one of the most devastating conditions in obstetrics practice with reported mortality of 20% to 60%.
- The pathophysiology seems to involve an abnormal maternal response to fetal tissue exposure associated with breaches of the maternal–fetal physiologic barrier during parturition.
- This response seems to involve activation of proinflammatory mediator similar to systemic inflammatory response syndrome.
- 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, in some cases, pulmonary vasodilators. Rapid initiation of treatment, aided by a high index of clinical suspicion, is essential.

INTRODUCTION

Amniotic fluid embolism (AFE) is a catastrophic syndrome typically occurring during labor and delivery or immediately postpartum. Despite its recognition as a distinct entity for almost 100 years, the syndrome remains one of the most enigmatic and devastating conditions in obstetrics practice. Although rare, AFE has a high case fatality rate and remains a leading cause of maternal mortality in industrialized countries.[1–5] AFE is classically characterized by hypoxia, hypotension or hemodynamic collapse, and coagulopathy. Despite numerous attempts to develop animal models, AFE remains incompletely understood. Over the last 2 decades, more rigorous research efforts have greatly improved our understanding of this condition.

HISTORIC CONSIDERATIONS

The first case report of AFE was published in a 1926 Brazilian medical journal.[6] The condition was not widely recognized until 1941 when Steiner and Lushbaugh[7] described

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fetal mucin and squamous cells during postmortem examination of the pulmonary vasculature in women who had unexplained obstetric death. Despite widely disparate clinical presentation, these authors viewed all patients with such findings as having died of a unique clinical syndrome, regardless of clinical presentation. These authors concluded that the patients had died as a result of "pulmonary embolism by amniotic fluid," giving rise to the term *amniotic fluid embolism*.[7] In a follow-up report by Liban and Raz in 1961,[8] cellular debris was also observed in the kidney, liver, spleen, pancreas, and brain of several such patients, although the exact route of squamous cells from the venous to the arterial circulation was not discussed.

The pathognomonic nature of the pulmonary findings described by Steiner and Lushbaugh[7] went largely unchallenged for several decades. As a result, numerous case reports appeared in the medical literature describing an incredible variety of presumed clinical presentations of "amniotic fluid embolism" based solely on the finding of fetal cells or other debris in the pulmonary arteries at autopsy.[9,10] However, a critical review of the clinical details provided in the original description found that 7 of the 8 index patients seem to have died of conditions such as sepsis or hemorrhage from undiagnosed uterine rupture, and the cause was labeled as "amniotic fluid embolism" based solely on pulmonary histologic findings.[7,9,10]

In the 1980s, the pulmonary artery catheter was introduced into critical care obstetrics. As a result, more frequent examination of pulmonary artery histologic specimens during life became possible. Several reports in the 1980s documented identical pulmonary pathologic findings in pregnant women with a variety of conditions unrelated to AFE.[9–11] These findings cast doubt on the validity of cases reported between 1941 and 1985 in which the diagnosis of AFE was based on pathologic findings alone.

Several experimental animal models yielded conflicting results regarding the pathologic potential of intravascular amniotic fluid and the pathophysiologic underpinnings of AFE (**Table 1**).[9,10,12–14] These studies generally involved a description of pathophysiologic changes resulting from the injection of whole or filtered human amniotic fluid or meconium into the central circulation of various animal species.[12–14] Most studies assumed a simple, mechanical mechanism of injury that can be summarized as follows: amniotic fluid is somehow forced into the maternal circulation, which results in obstruction of pulmonary arterial blood flow as amniotic fluid cellular debris is filtered by the pulmonary capillaries. Such obstruction leads to hypoxia, right ventricular heart failure, and death. However, the only 2 such studies carried out in primates using autologous or homologous amniotic fluid showed no adverse physiologic effects at all despite the infusion in one series of a volume of amniotic fluid that would represent 80% of the entire uterine volume.[12,13] Perhaps the fairest evaluation of these studies would be to conclude that the injection of large amounts of amniotic fluid or fetal fecal material from one species into the central circulation of small mammals of a different species sometimes causes adverse physiologic effects; the relevance of this observation to the human syndrome of AFE is dubious at best. An objective evaluation of this body of evidence finds quite clearly that the entrance of homologous amniotic fluid into the central circulation of primates and humans is generally innocuous, even in large volumes.[12–14]

The modern era of AFE investigation was heralded in the 1980s with the publication of several studies made possible by the development of clinical techniques for pulmonary artery catheterization of critically ill women, basic science investigations of maternal–fetal physiology, and the establishment of the first systematic case registry of AFE.[2,11,14,15] These studies found several surprising results that led to reevaluation and rejection of earlier theories of pathogenesis.