amniotic fluid embolism [created by Paul Young 02/10/07]

general

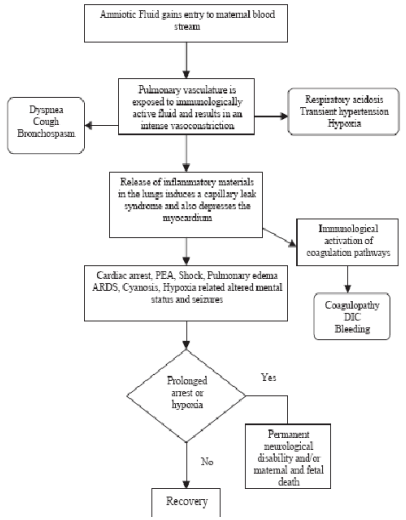
- Amniotic fluid embolism is a rare syndrome that can have debilitating and lethal consequences.
- First was reported in 1926 by Meyer who described fetal cellular debris in the maternal circulation that was associated with symptoms of hypotension and dyspnea.

epidemiology

-Reported incidence ranges from as high as 1 case in 8000 deliveries to as low as 1 case in 80,000 deliveries.
- The reported mortality rate was as high as 86% in some earlier reports; later studies demonstrated a mortality rate of approximately 26%.
- Only 15% of survivors are neurologically intact

pathogenesis

- It has been a common belief that the spillage of amniotic fluid in the blood stream results in embolization of this fluid to the pulmonary vasculature, causing acute cor pulmonale. It was believed that the microembolization of fetal products to the pulmonary vasculature caused sudden hypoxia and cardiovascular collapse. Evidence in the literature, however, suggests that it is not as simplistic.
- The pathophysiology of this syndrome is believed to be humoral. The clinical picture of cardiovascular collapse, left ventricular failure, coagulopathy, and disseminated intravascular coagulation (DIC) is believed to share common pathways with sepsis and anaphylaxis, hence the suggested term anaphylactoid syndrome of pregnancy.
- It is postulated that exposure to fetal products incites an immunologic response in the mother that unleashes a cascade of mechanisms, leading to profound shock.
- Hemodynamic data corroborate the premise of a humoral cause rather than an embolic cause. In amniotic fluid embolism, the pulmonary artery occlusion pressure is elevated, the left ventricular stroke work index is low, the mean pulmonary arterial pressure is increased only moderately, and the change in central venous pressure is variable.



clinical manifestations

- The first response is an intense vasoconstriction of the pulmonary vasculature, causing severe hypoxia, respiratory acidosis, and right heart failure. Little concrete evidence supports this initial event, as pulmonary artery catheters may not be inserted in this early phase. Animal studies have demonstrated this model of intense vasoconstriction and hypoxia.
- The second phase is believed to consist of left heart failure and hemodynamic collapse with pulmonary edema.
- The third phase in the continuum of events is neurologic dysfunction, including seizures and coma. There may be overlapping coagulopathy and DIC.

Signs and symptoms of amniotic fluid embolism and their reported frequency*

Signs and symptoms	Percentage
Hypotension	100
Fetal distress	100
Pulmonary edema or ARDS	93
Cardiopulmonary arrest	87
Cyanosis	83
Coagulopathy	83
Dyspnea	49
Seizure	48
Atony	23
Bronchospasm	15
Transient hypertension	11
Cough	7
Headache	7
Chest pain	2

- The clinical picture can be dramatic, with sudden acute dyspnea quickly progressing to asystole or pulseless electrical activity. Patients who are diagnosed early enough to be resuscitated may develop life-threatening hematologic abnormalities.
  - No laboratory assays or bedside investigations can confirm the diagnosis. The hematologic picture may show DIC, or the chest radiograph may show a diagnosis of acute respiratory distress syndrome, but these conditions occur usually late in the course of the illness and are not specific
  - Various investigators have proposed the use of laboratory assays, such as zinc-coproporphyrin I and monoclonal TKH-2 antibodies, but these reports are anecdotal. There is no established role of these assays in confirming the diagnosis.

treatment

- The life-threatening evolution of an amniotic fluid embolism requires prompt resuscitation, with airway and hemodynamic support in the critical care setting
- Less severe scenarios may present with hypotension and acute respiratory failure. In such cases, supplemental oxygenation should be given, and early intubation should be performed with high oxygen concentrations and positive end-expiratory pressure, if needed, to treat refractory hypoxemia.
- The principles of treating hemodynamic collapse are similar to those of treating sepsis. Crystalloids and vasopressors should be used to maintain an adequate mean arterial pressure to ensure organ perfusion.
- Use of a pulmonary artery catheter may be beneficial in fluid management, because the cardiovascular dynamics change from right-sided to left-sided ventricular failure as the syndrome evolves.
- Hematologic manifestations are best treated by serial and frequent monitoring of laboratory parameters, such as blood counts, fibrinogen, and coagulation & correction of coagulopathy
- Other interventions, like steroids, antithrombin III infusion, and leukotrine inhibitors, have been tried, but the data to support their routine use are lacking.