Amniotic Fluid Embolism:
Pathophysiology and Diagnosis

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Disclosure

I am the Founder and Executive Director of the AFE Foundation. I also hold board positions with Scripps Health and the California Maternal Quality Care Collaborative.

I have no relevant financial relationships to disclose.
Outline

• History
• Clinical presentation
• Pathophysiology
• Diagnostic criteria
• Differential diagnoses
Learning Objectives

To become better acquainted with the clinical presentation and understand the pathophysiology of AFE to aid in early recognition and management of AFE; a leading cause of maternal death and suffering in all developed nations.
Historical References

• 1926 First recognized and reported in Brazil

• 1941 Steiner and Lushbaugh define AFE based on post mortem findings of fetal squames in vasculature

• 1948 “Let us be careful not to make it a waste basket for all cases of unexplained death in labor…” Eastman

• 1980’s S.L. Clark registry, increased diagnostics tools, review of cases begins disproves previous theory.

• 1995 Clark proposes new name: Anaphylactoid Syndrome of Pregnancy- despite its greater relevance the name is not widely adopted
General Understanding

• Classic triad:
  • Hypoxia
  • Hypotension or hemodynamic collapse
  • Coagulopathy

• Remains poorly understood:
  • Unpredictable
  • Rare
  • Acute
  • Lacks a gold standard diagnosis
  • Commonly over diagnosed
Theories of Cause

**Previous**

- Amniotic fluid forced into vasculature
- Cellular debris obstructs pulmonary arterial flow
- Hypoxia

**Current**

- Passage of amniotic fluid into maternal circulation
- Abnormal maternal response to fetal antigenic material
- Immune mediated response “Immunologic Storm”
Onset

- Before, during or shortly after labor (30 min)
- Vaginal or Cesarean Birth
- Amniocentesis
- Rupture of Membranes
- D&E
- Abortion
- Intrauterine Pressure Catheter
Clinical Presentation

- Shortness of breath
- Nausea
- Impending sense of doom
- Fetal bradycardia
- Profound Hypotension
- Cyanosis
- Seizure
- Cardiac Arrest
- DIC
- Organ Failure
- Death
Hemodynamic Changes

- Complex and variable
- Initially: pulmonary and systemic hypertension
- Subsequent: profound LV dysfunction
  - Contributing factors:
    - RV failure
    - Myocardial ischemia
    - Coronary artery vasospasm
    - Pulmonary injury/hypertension
Pathophysiologic Alterations

Exposure to amniotic fluid or foetal antigens

- Immunological release of mediators
- Mechanical obstruction
- Complement system activation
- Coagulation system activation

Cardiopulmonary effects
- Acute pulmonary hypertension
- Hypoxia
- Right ventricular failure

Left ventricular failure
- Neurological problems - coma, seizures, residual deficits
- Acute respiratory distress syndrome
- Other organ dysfunction (e.g. Acute kidney injury)

Consumption coagulopathy
- Cardiopulmonary collapse and death

Diagnostic Criteria

• Currently no specific test
• Based on clinical manifestation and exclusion of any other cause
• Onset during labor or within 30 minutes of delivery
• Must include: acute hypotension and/or cardiac arrest followed and DIC (unless unless patient does not survive within the hour to assess clotting status)
• Autopsy or imaging studies must exclude pulmonary thromboembolism, unless coagulopathy was part of the clinical presentation.
AFE Pretenders
Non Obstetric

- Acute myocardial infarction
- Anaphylaxis
- Anesthesia (toxicity/high spinal)
- Arrhythmia
- Aspiration

- Blood transfusion reaction
- Pulmonary edema
- Pulmonary embolism (air, fat, thrombi)
- Septic Shock
- Tension Pneumothorax
AFE Pretenders Obstetrical Causes

• Ecclampsia
• Placental abruption
• Uterine rupture
• Postpartum hemorrhage
• Peripartum cardiomyopathy
Conclusion

• Early recognition dramatically increases survivability
• Understanding current theories of cause is imperative
• Diagnosis must have thorough review
• Misdiagnosis has lasting impact