AMNIOTIC FLUID EMBOLISM: OBSTETRICAL ANESTHESIA

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WHAT IS AN AMNIOTIC FLUID EMBOLISM?

- A rare catastrophic condition that occurs when amniotic fluid or fetal antigen/matter enters maternal circulation
- Characterized by rapid onset of cardiovascular collapse with a cascade of symptoms
- Life threatening and can represent an anaphylactoid syndrome
AMNIOTIC FLUID EMBOLISM: AFE

- Quick recognition and prompt intervention essential for optimizing maternal outcomes
- Traditional treatment modalities
- Increasing Anesthesia case reports since 2011: Improved Patient Outcomes
AMNIOTIC FLUID EMBOLISM

- Diagnosis: AFE is clinical diagnosis based upon the presence of characteristic clinical findings and exclusion of other potential causes of these findings
- AFE Diagnosis should be suspected in pregnant or recently post partum women
- Rapid Diagnosis & Treatment

- AFE SIGNS:
  - Cardiovascular Collapse
  - Severe Respiratory Distress
  - Hypoxia
  - Seizures
  - Coagulopathy: DIC
AFE DIAGNOSIS

- AFE can arise during labor for vaginal delivery, during cesarean section or in the post partum period.
- AFE Diagnosis can be done retrospectively and investigative data including autopsy information.
- Atypical cases can represent one fourth of all total AFE cases, thus may only have one of the symptoms stated.
AMNIOTIC FLUID EMBOLISM

- Histopathology of squamous cells, trophoblastic cells and mucin from a Pulmonary catheter are NOT diagnostic of AFE.
- AFE Diagnostic Criteria can include: Hypotension Systolic BP less than 90
- Platelets under 100K
- Prolonged Prothrombin Time > 25% increase
- Fibrinogen Time prolonged > 200mg/L
AMNIOTIC FLUID EMBOLISM

- AFE diagnosis details:
  - Absence of a Fever During Labor
  - Clinical Onset during labor or within 30 minutes after labor
  - Scoring System for AFE Diagnosis details presence of:
    Hypotension Systolic less than 90mmHg, Low platelets (less than 100K, notable when under 50K), DIC (fibrinogen > 200mg/L)
AMNIOTIC FLUID EMBOLISM

- Incidence of AFE: ranges from 2 to 6 per 100,000 births globally however misdiagnosis and under reporting issues impact data
- Possible Over versus Under reporting internationally
AMNIOTIC FLUID EMBOLISM: PATHOGENESIS

• Pathogenesis unclear however believed to entry of amniotic fluid that contains fetal cells and other antigenic materials pass into Maternal Circulation via a breach in the Maternal/Fetal Interface
• Breach of Amniotic Fluid/Antigenic Materials into maternal circulation results in a ABNORMAL ACTIVATION of humoral and immunological processes that result in a cascade of physiological events
AFE: CASCADE OF CATASTROPHIC PHYSIOLOGY

• Release of Vasoactive and Procoagulant substances similar to SIRS
• Pulmonary Pressures elevate and Right Ventricular (RV) pressure increases, next RV failure occurs leading to Left ventricle (LV) failure
AMNIOTIC FLUID EMBOLISM: CASCADE OF CATASTROPHE

- LV failure then causes hypoxia of Left Ventricle Pump/Muscle leads to further maternal inflammatory mediators, plus a direct depressant effect of the amniotic fluid on the myocardium if it reaches cardiac circulation.
AMNIOTIC FLUID EMBOLISM: PULMONARY AND CARDIAC FAILURE

• Acute pulmonary HTN leading to Cardiac Failure then can result in Pulmonary Edema, Hypoxic Respiratory Failure

• Damage to Endothelial Alveolar Membrane and capillary leak syndrome demonstrated by high protein concentrations in edema and amniotic fluid debris in SPUTUM and alveolar spaces
AMNIOTIC FLUID EMBOLISM: PHYSIOLOGY

- Activation of factor 7 & platelets plus release of inflammatory mediators then triggers the coagulation cascade resulting in Disseminated intravascular coagulation (DIC)
- DIC results in ischemic distal organ failure or multi-organ failure
- Hemorrhage from DIC contributes to further hemodynamic instability
Most (about 90%) of patients experiencing AFE present with an abrupt, catastrophic and rapidly progressive clinical presentation.

Classically: hypotension, hypoxia with non-cardiogenic pulmonary edema plus hemorrhage due to DIC.
AFE: CASCADE OF CATASTROPHE

- Acute pulmonary hypertension (HTN) results in severe ventilation/perfusion mismatch.
AMNIOTIC FLUID EMBOLISM: SIGNS & SYMPTOMS

- Aura: about 1/3rd of patients describe a sense of sudden doom, chills, nausea, vomiting, agitation, anxiety, or a change in mental status immediately preceding the AFE event.
AMNIOTIC FLUID EMBOLISM: SIGNS & SYMPTOMS

- Sudden Cardiorespiratory failure or arrest: dyspnea, hypoxia, tachypnea, cyanosis, crackles, possibly wheezing
- May see pulseless Ventricular tachycardia or fibrillation bradycardia and / or asystole
AMNIOTIC FLUID EMBOLISM: SIGNS & SYMPTOMS

- If the patient survives the cardiorespiratory arrest, non cardiogenic pulmonary edema often develops as the sided LV dysfunction resolves
- Fluid Boluses often worsens pulmonary edema during resuscitation phase
AMNIOTIC FLUID EMBOLISM: DIC COMMON

- DIC occurs in approximately 80% of AFE patients typically after a short period of cardiorespiratory compromise however can occur before without cardiopulmonary compromise as well.
AMNIOTIC FLUID EMBOLISM: DIC

• DIC signs in patients who have not yet delivered may include bleeding from invasive intervention sites: IV site, possibly urinary catheter, GI tract
• DIC in post delivery can manifest from vaginal area, uterus or incision sites for cesarean sections

<table>
<thead>
<tr>
<th>Laboratory Findings in Acute DIC</th>
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<tbody>
<tr>
<td>- Platelet Count ↓</td>
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<tr>
<td>- Fibrinogen ↓</td>
</tr>
<tr>
<td>- PT (INR) ↑</td>
</tr>
<tr>
<td>- PTT ↑</td>
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<tr>
<td>- D-dimer ↑</td>
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<tr>
<td>- Peripheral smear Schistocytes, helmet cells</td>
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AMNIOTIC FLUID EMBOLISM: EMERGENCY MANAGEMENT

- Multidisciplinary approach:
- Perform CPR as indicated High Quality CPR
- Control Hemorrhage & Reverse Coagulopathy: Tranexamic Acid (TXA) institute Massive Transfusion Protocol
AMNIOTIC FLUID EMBOLISM: EMERGENCY INTERVENTIONS

- During CPR if undelivered: remember Left Uterine Displacement, Monitor Fetal Heart Rate, Secure Airway as indicated to oxygenate.

- Vasoactive support using medications to ensure perfusion: Dobutamine, Epinephrine, Phenylephrine if arrhythmias a concern.
AMNIOTIC FLUID EMBOLISM: EMERGENCY MANAGEMENT

- Avoid Vasopressin if Fetus NOT delivered as it increase uterine contractions
- Avoid Dopamine due as patients with septic shock has poorer outcomes
AMNIOTIC FLUID EMBOLISM: EMERGENCY INTERVENTIONS

• Respiratory Support: administration of supplemental oxygen most often intubation & mechanical ventilation

• Protective Lung Strategies: lower tidal volumes, very cautious peep as tolerated
AMNIOTIC FLUID EMBOLISM: EMERGENCY INTERVENTIONS

- ECHMO should not be routinely used due to coagulation issues and increase risk of bleeding
- However if coagulopathy corrected, managed or goals are to promote oxygenation for fetal delivery with possible maternal demise impending
AMNIOTIC FLUID EMBOLISM: DELIVERY OF FETUS

- AFE presenting before 22-23 weeks gestation, immediate delivery should be considered if as delivery of fetus at this early stage of resuscitation is believed to increase the chance of maternal survival.
AMNIOTIC FLUID EMBOLISM: DELIVERY

- Maternal morbidity or death is a significant risk when cesarean section is performed in the presence of coagulopathy

- Immediate Massive Transfusion blood products must be immediately available with adequate access and resources to resuscitate adequately
AMNIOTIC FLUID EMBOLISM
A NOVEL NEW TREATMENT

- AOK Protocol
- Atropine 1 mg
- Ondansetron 8 mg
- Ketorolac 30 mg
- WHY??

A-OK medication regimen [3]

<table>
<thead>
<tr>
<th>Atropine 1 mg (vagolytic)</th>
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<tbody>
<tr>
<td>Ondansetron 8 mg (5-HT3 antagonist)</td>
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<tr>
<td>Ketorolac 30 mg (cyclooxygenase inhibitor)</td>
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</tbody>
</table>
• “Killer Platelet” platelet activation & degranulation
• Thromboxane & Serotonin Release (severe pulmonary hypertension)
• Vagal reflex bradycardia & vasodilation (inflammatory mediators)
AFE: WHY THE AOK PROTOCOL??

• Atropine produces VAGOLYSIS (stops the bradycardia and promotes forward flow of cardiac cycle)
• Ondansetron blocks Serotonin receptors (combats the pulmonary hypertension)

• Ketorolac fights Killer Platelets by blocking thromboxane release
• Off labels use for AOK however numerous case reports now in place in literature with quick return to perfusion and oxygenation
AMNIOTIC FLUID EMBOLISM: BARBARA LEIGHTON MD

- Washington University in Missouri
- Animal Studies using rabbits
- Case reports since 2011
- Presented in Society for Obstetrical and Antenatal Medicine 2012-2014
AFE DIFFERENTIAL DIAGNOSIS

• Several clinical conditions often may mimic AFE such as:
  • Hemorrhage secondary to uterine atony, lower genital track and/or uterine lacerations, retained placenta
  • Uterine abruption (most often associated with significant sudden onset of pain)
AFE: DIFFERENTIAL DIAGNOSIS

- Other disorders that may mimic AFE:
  - Thromboembolism
  - Myocardial infarction
  - Septic Shock
  - Anesthetic accident?
AFE: DIFFERENTIAL DIAGNOSIS

- Air embolism
- High spinal, local anesthesia toxicity
- Severe drug reactions
- Abrupt Maternal Hemorrhage
• CPR, controlled airway, adequate IV access or CVP
• Arterial Line
• PA catheter of limited value for immediate resuscitation period
AFE & PROGNOSIS

- AFE one of the leading causes for maternal mortality and morbidity
- Represents about 10% of all maternal deaths
- Mortality range 10-90%
AFE & PROGNOSIS

- Hypoxemia often profound resulting poor outcomes
- 50% of deaths occur in the first hour with those who survived seeing about 80 percent neurological damage
AMNIOTIC FLUID EMBOLISM

• Neonatal outcomes can be poor especially if there are delays in interventions unclear differential diagnosis
• More severe the presentation the worsened the maternal & fetal neurological outcomes
AFE KEY CONCEPTS

• Initial resuscitation and implementation of treatment during simultaneous differential diagnosis
• Delays to treatment worsen maternal and fetal outcomes
AFE QUESTIONS? AOK QUESTIONS?