AMNIOTIC FLUID EMBOLISM: OBSTETRICAL ANESTHESIA

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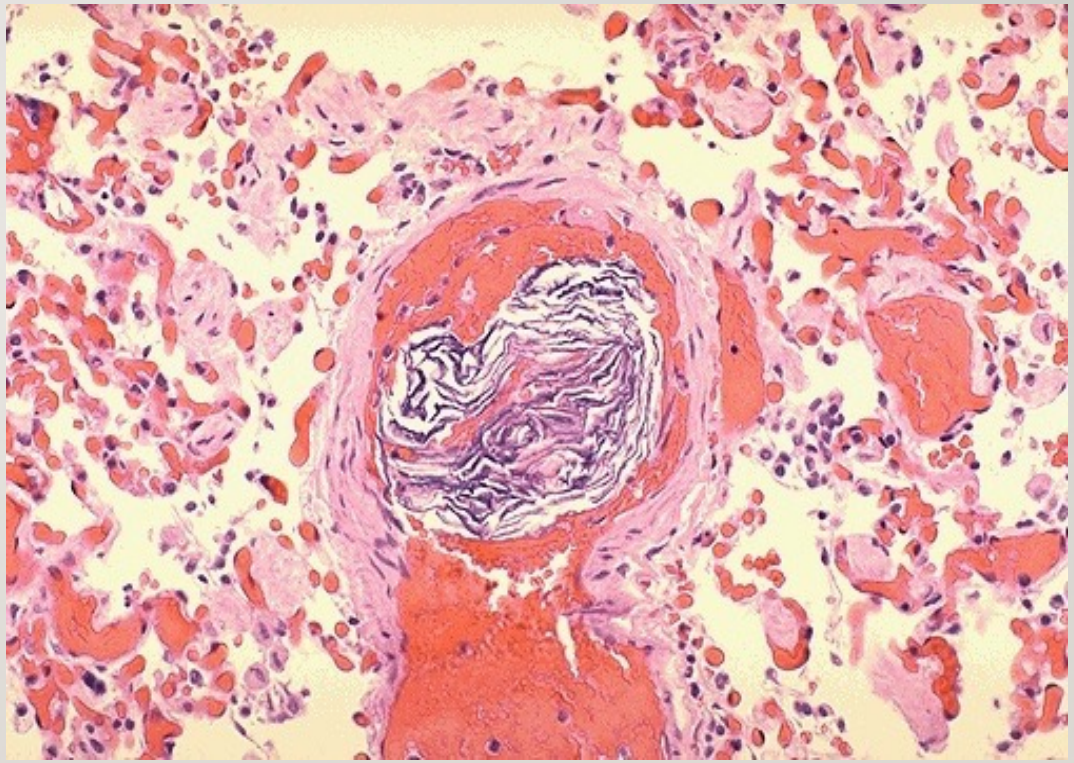
WHAT IS AN AMNIOTIC FLUID EMBOLISM?

- A rare catastrophic condition that occurs when amniotic fluid or fetal antigen/matter enters maternal circulation
- Characterized by rapid on set 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
- New, Novel treatment protocol: Barbara Leighton MD: University of Washington in Missouri 2011-2014
- Increasing Anesthesia case reports since 2011: Improved Patient Outcomes



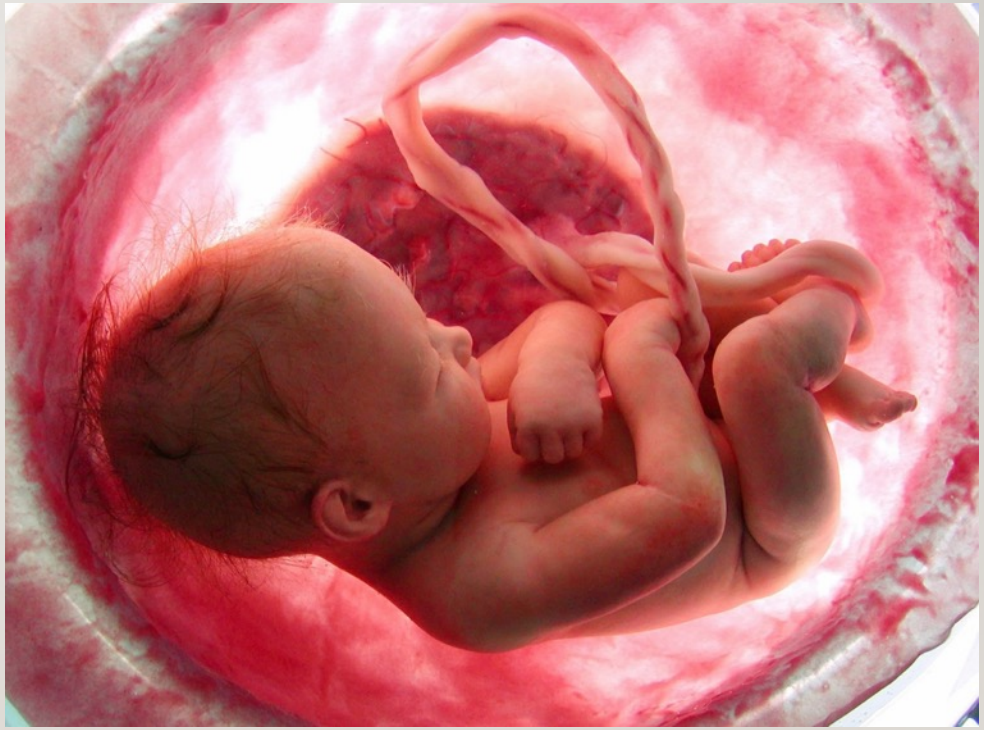
AMNIOTIC FLUID EMBOLISM

- 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

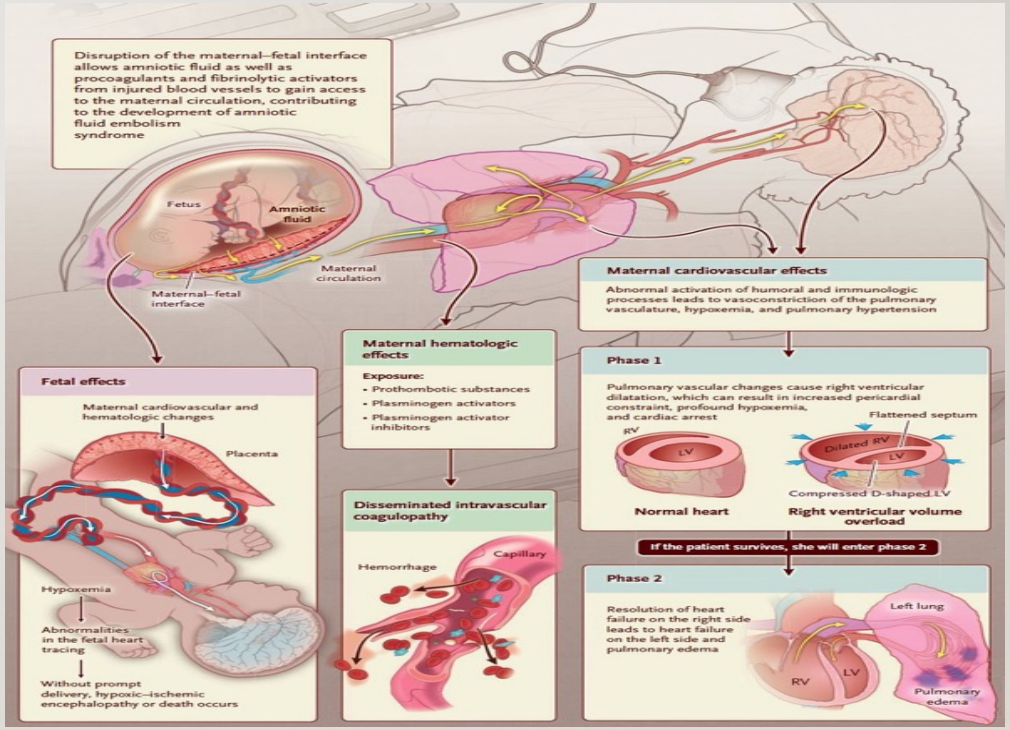
AFE DIAGNOSIS

- 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

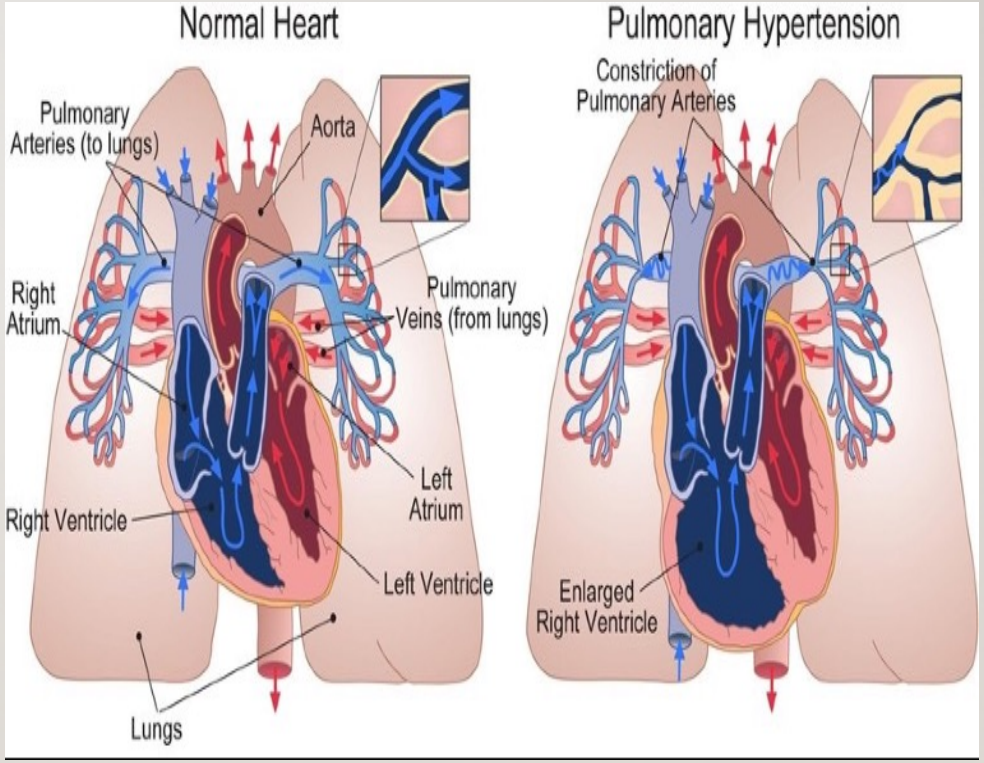
- Pathogenesis unclear however believed to entry of amniotic fluid that contains fetal cells and other antigenic materials pass into Maternal Circulation via a breach in the Maternal/Fetal Interface

AMNIOTIC FLUID EMBOLISM: PATHOGENESIS

- 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

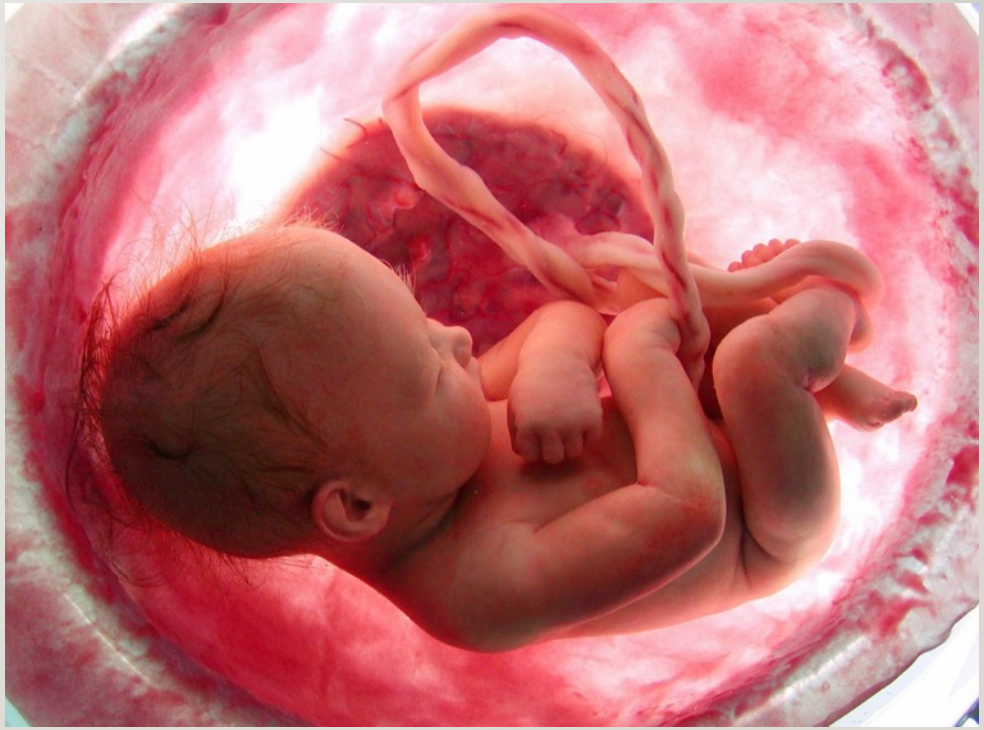
- LV failure then causes hypoxia of Left Ventricle Pump/Muscle leads to further maternal inflammatory mediators, plus a direct depressant effect of the amniotic fluid on the myocardium if it reaches cardiac circulation

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

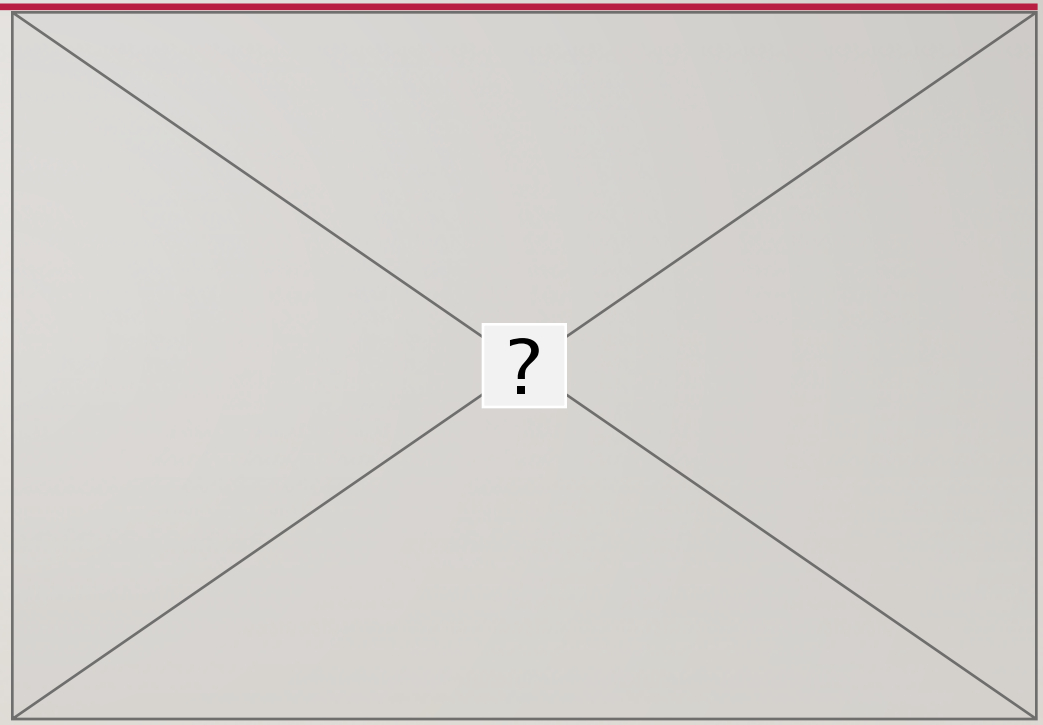
AMNIOTIC FLUID EMBOLISM: PHYSIOLOGY

- 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



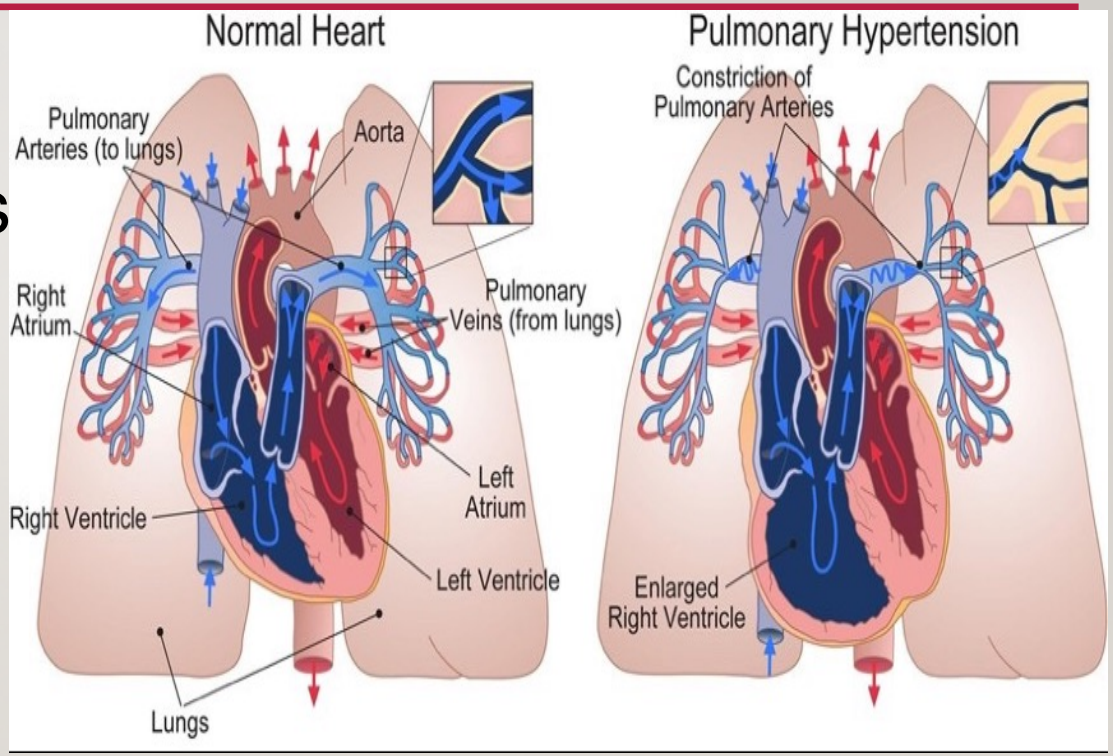
AMNIOTIC FLUID EMBOLISM: SIGN & SYMPTOMS

- Most (about 90%) of patients experiencing AFE present with a abrupt, catastrophic and rapidly progressive clinical presentation
- Classically: hypotension, hypoxia with non cardiogenic pulmonary edema plus hemorrhage due to DIC.



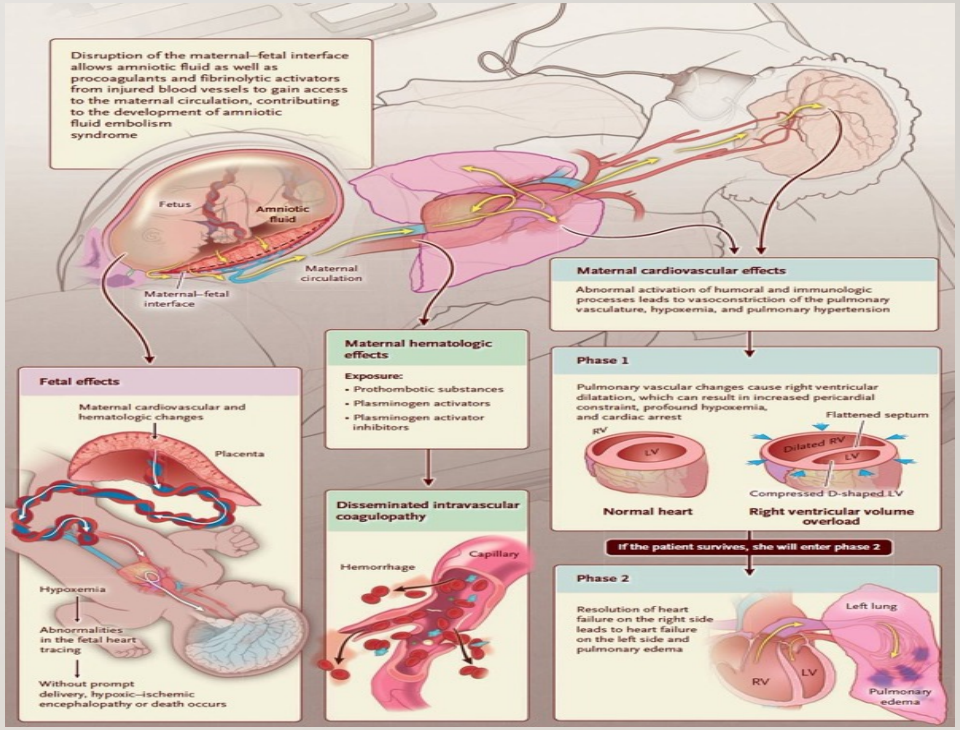
AFE: CASCADE OF CATASTROPHE

- Acute pulmonary hypertension (HTN) results in severe ventilation/ perfusion mismatch



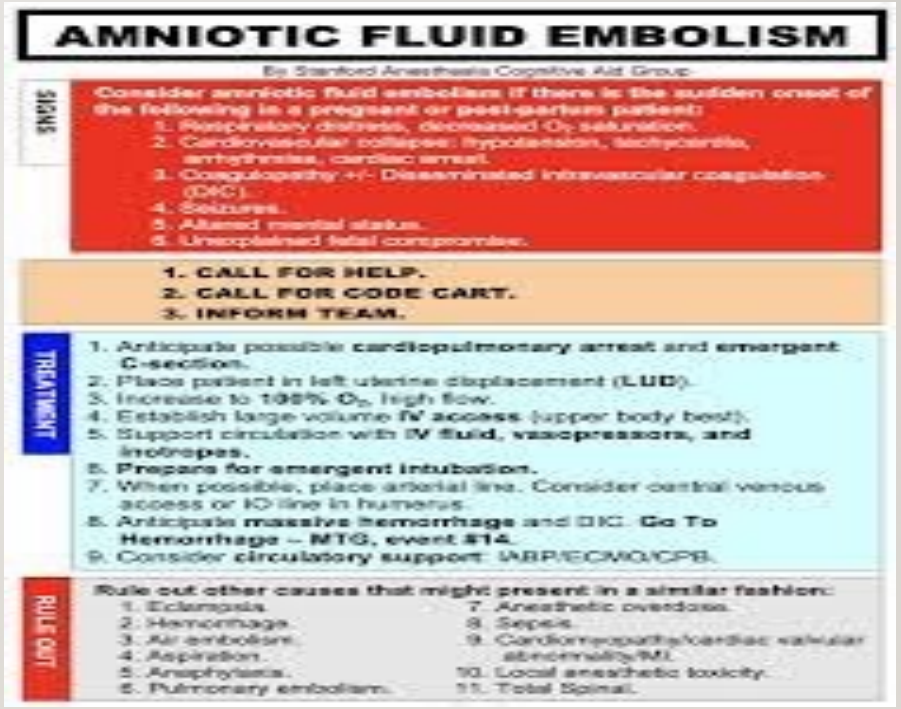
AMNIOTIC FLUID EMBOLISM: SIGNS & SYMPTOMS

- 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 bradyarrhythmia and / or asystole

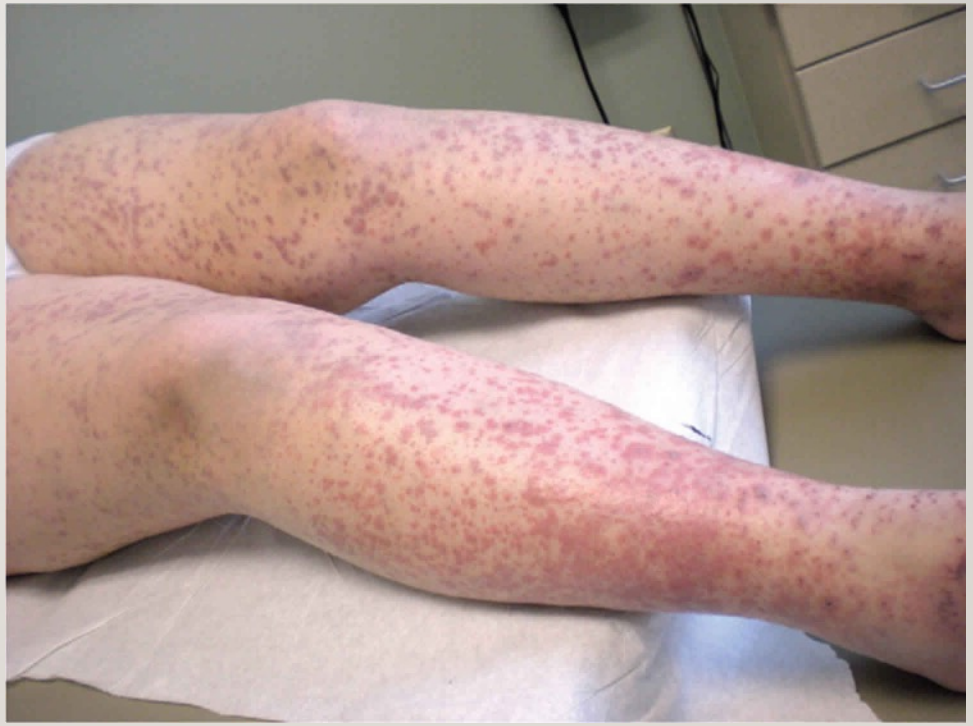


AMNIOTIC FLUID EMBOLISM: SIGNS & SYMPTOMS

- 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

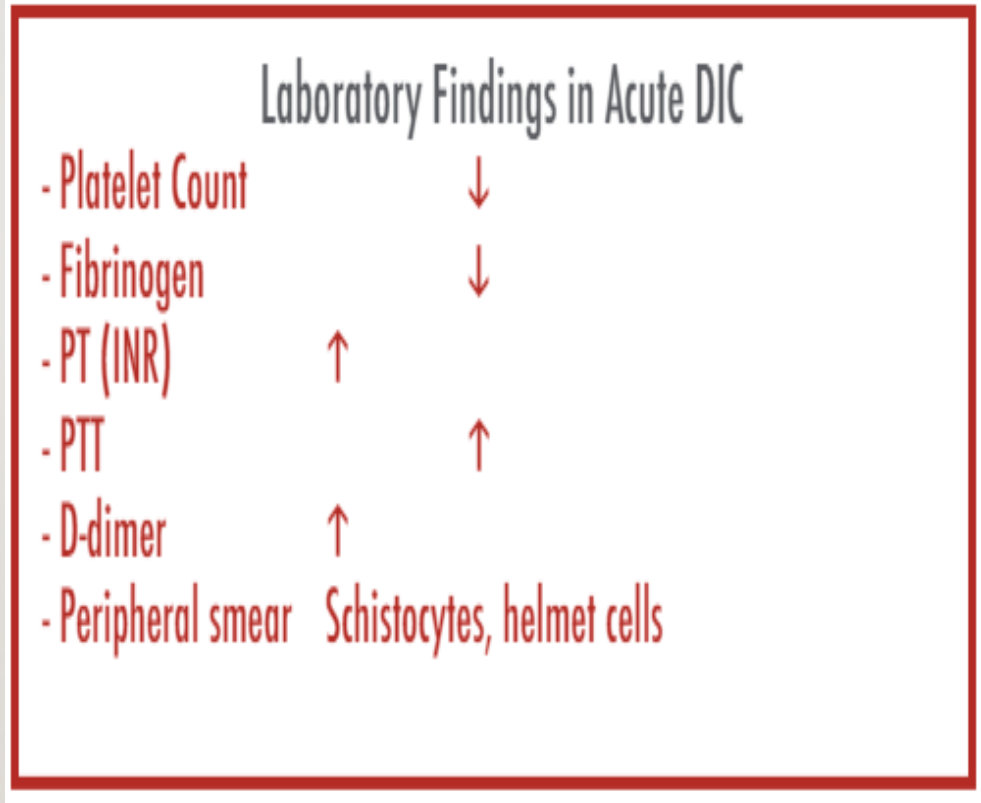
AMNIOTIC FLUID EMBOLISM: DIC COMMON

- 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.



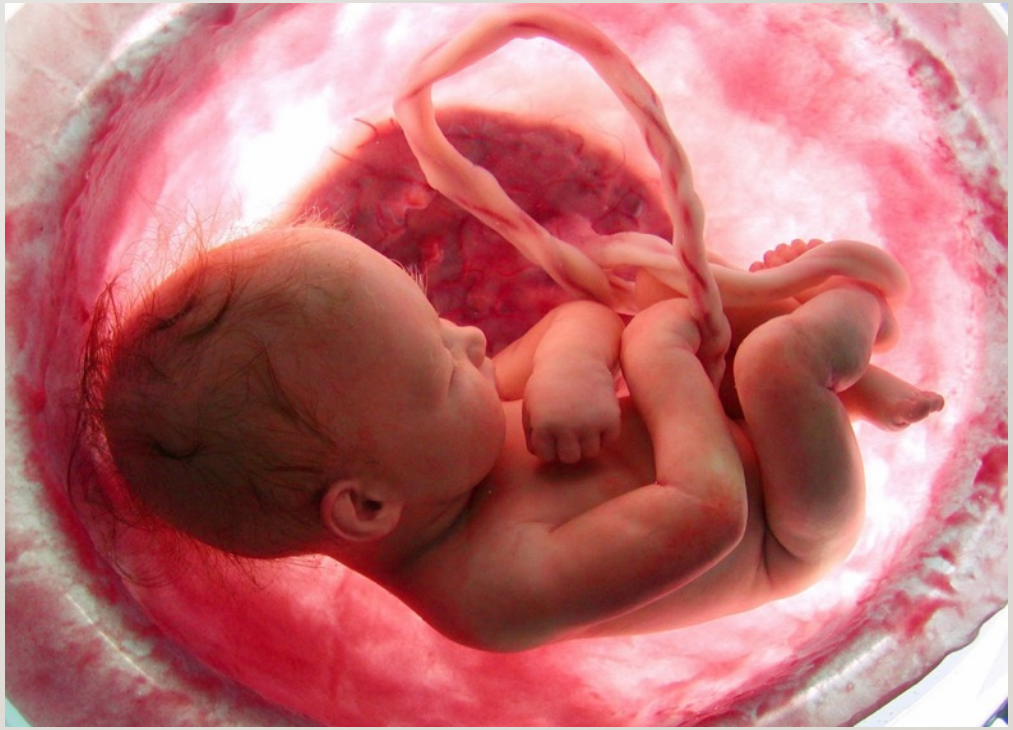
AMNIOTIC FLUID EMBOLISM: DIC

- 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



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 arrythmias a concern

AMNIOTIC FLUID EMBOLISM: EMERGENCY MANAGEMENT

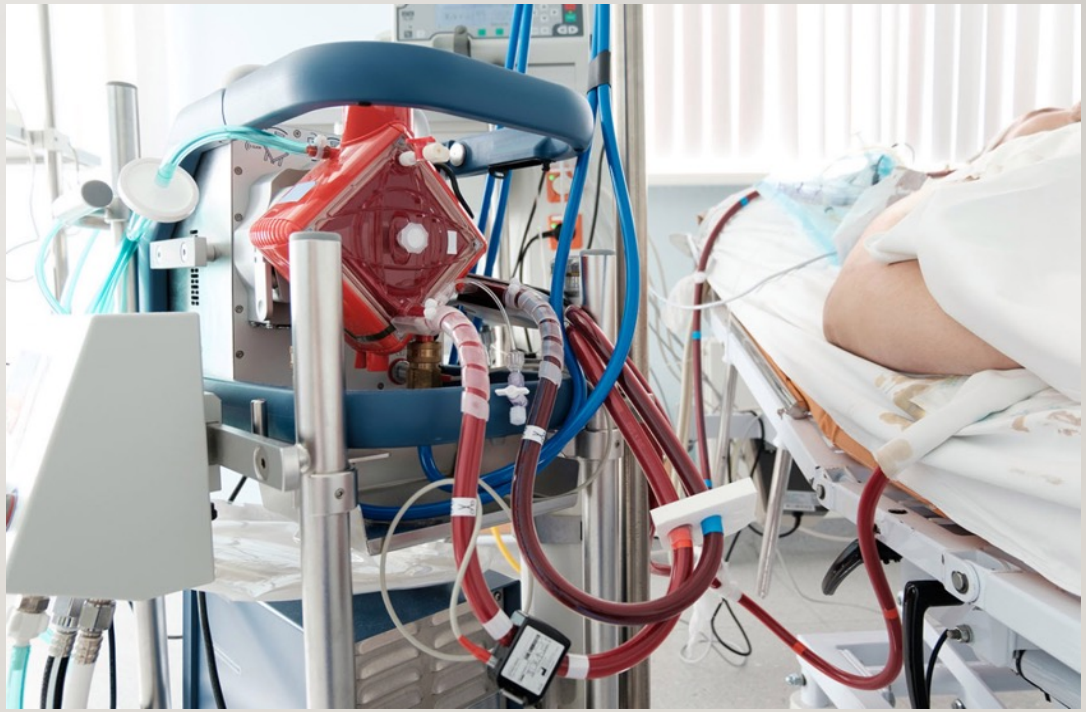
- Avoid Vasopressin if Fetus NOT delivered as it increase uterine contractions
- Avoid Dopamine due as patients with septic shock has poorer outcomes

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

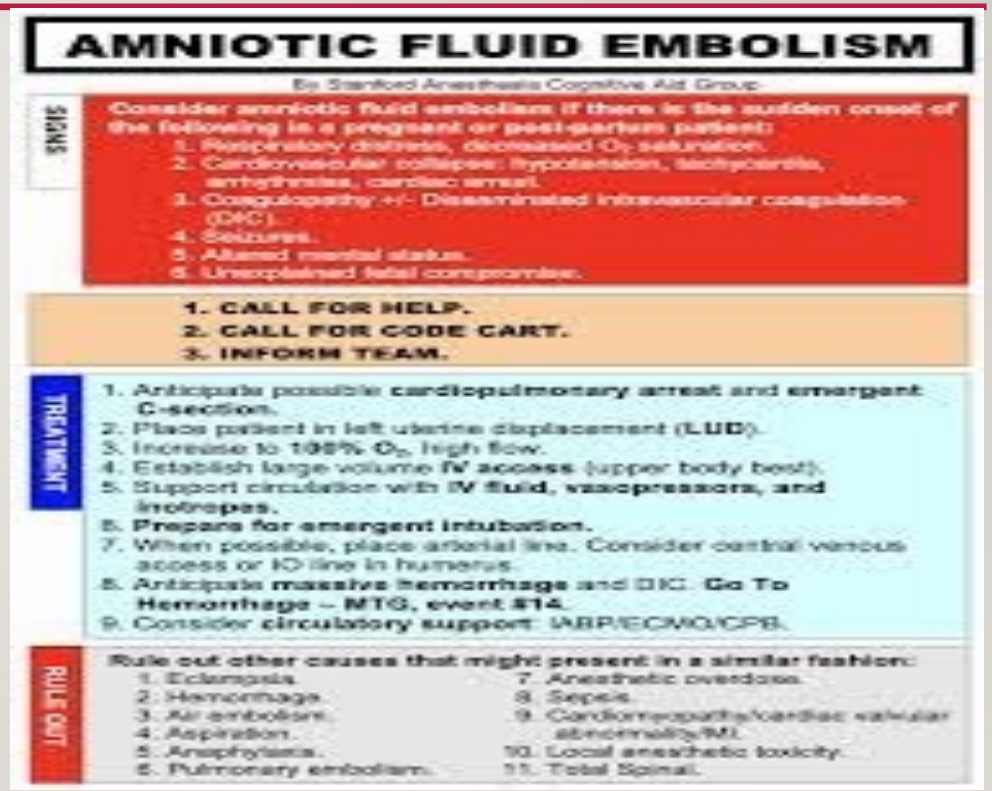
AMNIOTIC FLUID EMBOLISM: EMERGENCY INTERVENTIONS

- 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

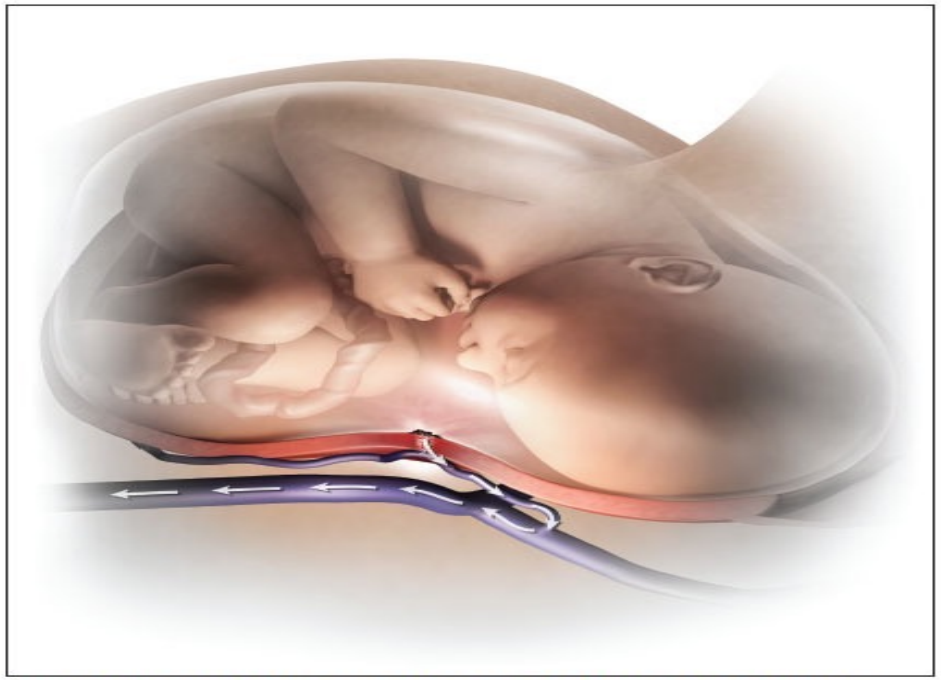
- AFE presenting before 22-23 weeks gestation, immediate delivery should be considered if as delivery of fetus at this early stage of resuscitation is believed to increase the chance of maternal survival



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 is likely caused by amniotic fluid entering the uterine veins. The amniotic fluid triggers an inflammatory and anaphylactoid response that causes cardiopulmonary dysfunction.

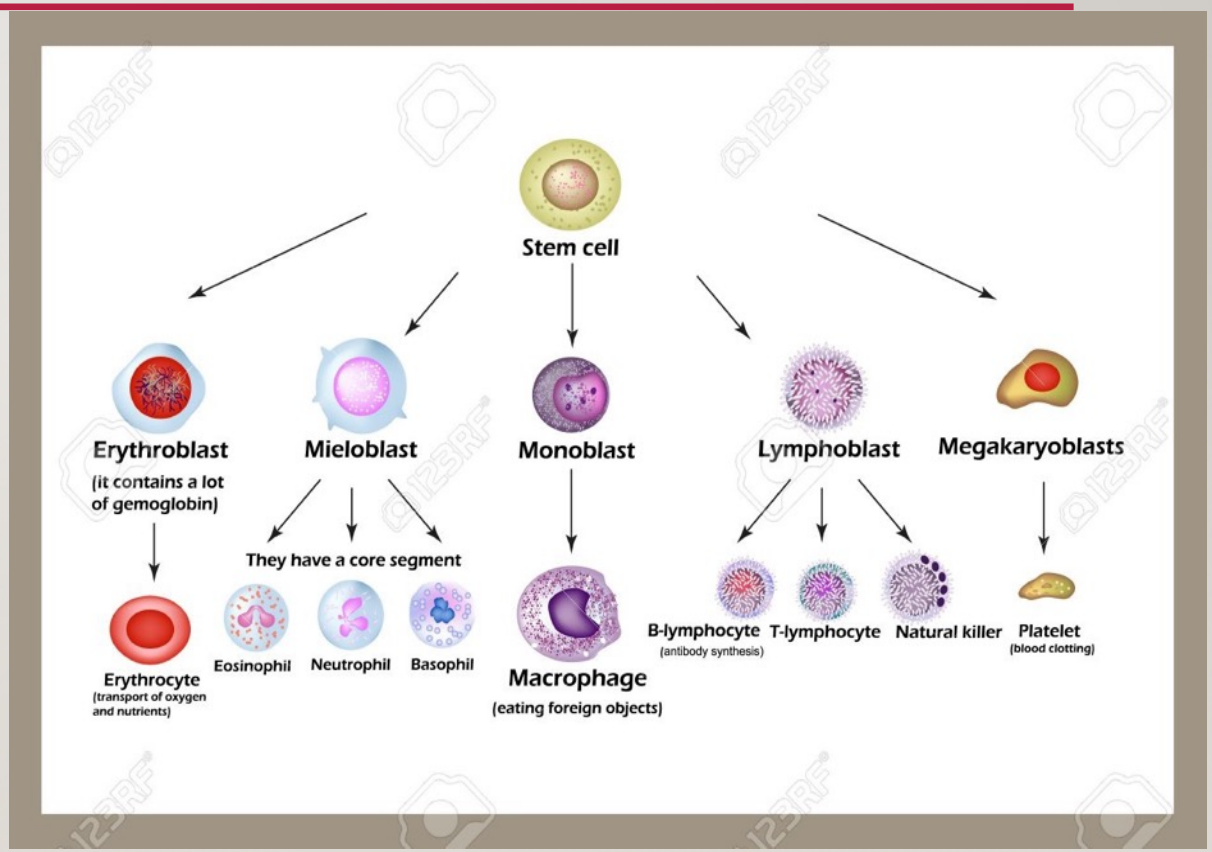
AMNIOTIC FLUID EMBOLISM
A NOVEL NEW TREATMENT

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

A-OK medication regimen [3]
Atropine 1 mg (vagolytic)
Ondansetron 8 mg (5-HT3 antagonist)
Ketorolac 30 mg (cyclooxygenase inhibitor)

AFE: CLOSER LOOK AT PHYSIOLOGY

- “Killer Platelet” platelet activation & degranulation
- Thromboxane & Serotonin Release (severe pulmonary hypertension)
- Vagal reflex bradycardia & vasodilation (inflammatory mediators)



AFE: WHY THE AOK PROTOCOL??

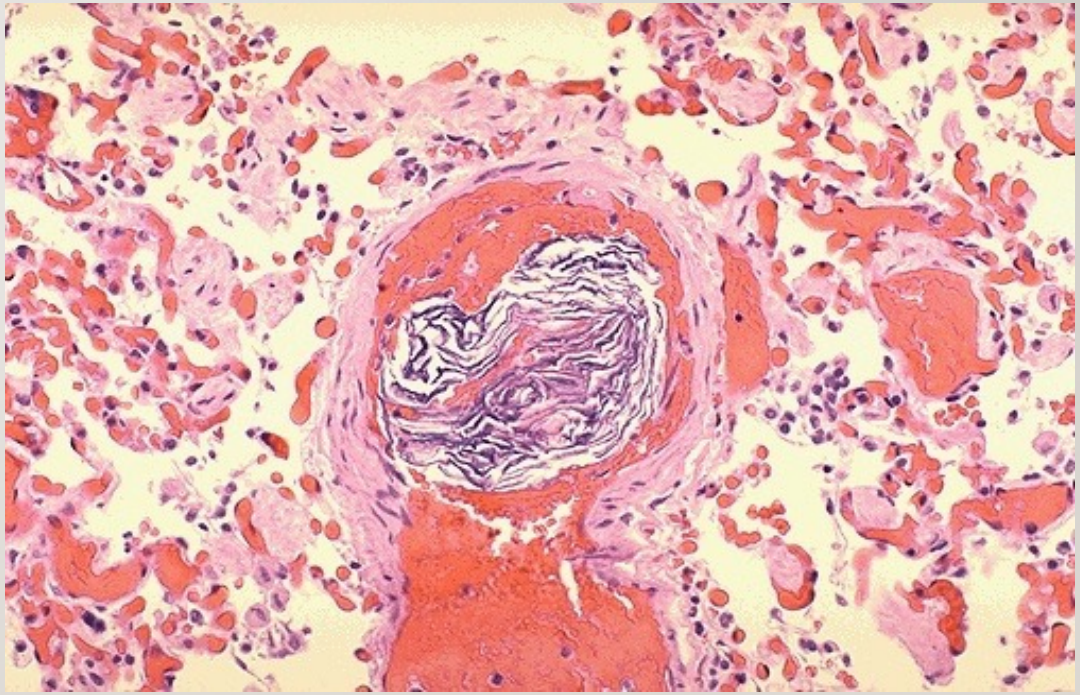
- Atropine produces VAGOLYSIS (stops the bradycardia and promotes forward flow of cardiac cycle)
- Ondansetron blocks Serotonin receptors (combats the pulmonary hypertension)
- Ketorolac fights Killer Platelets by blocking thromboxane release
- Off labels use for AOK however numerous case reports now 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

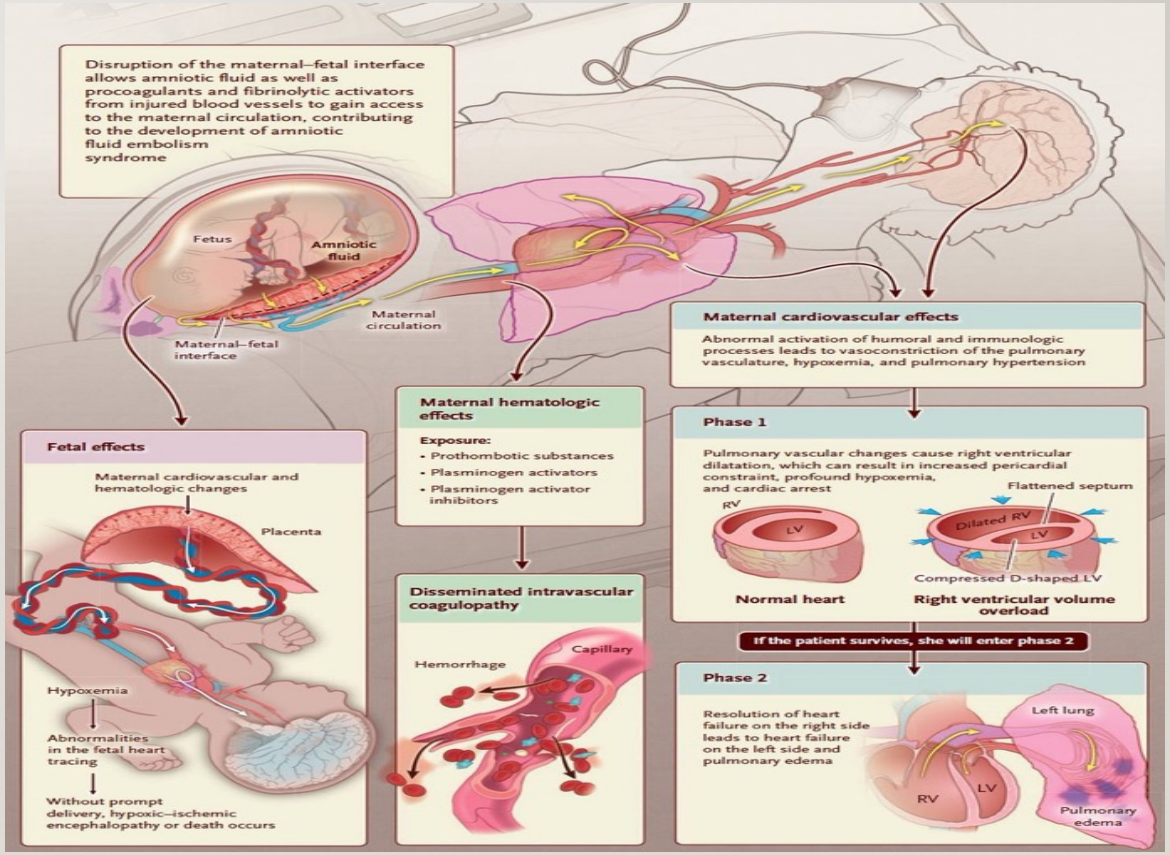
AFE DIFFERENTIAL DIAGNOSIS

- 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)



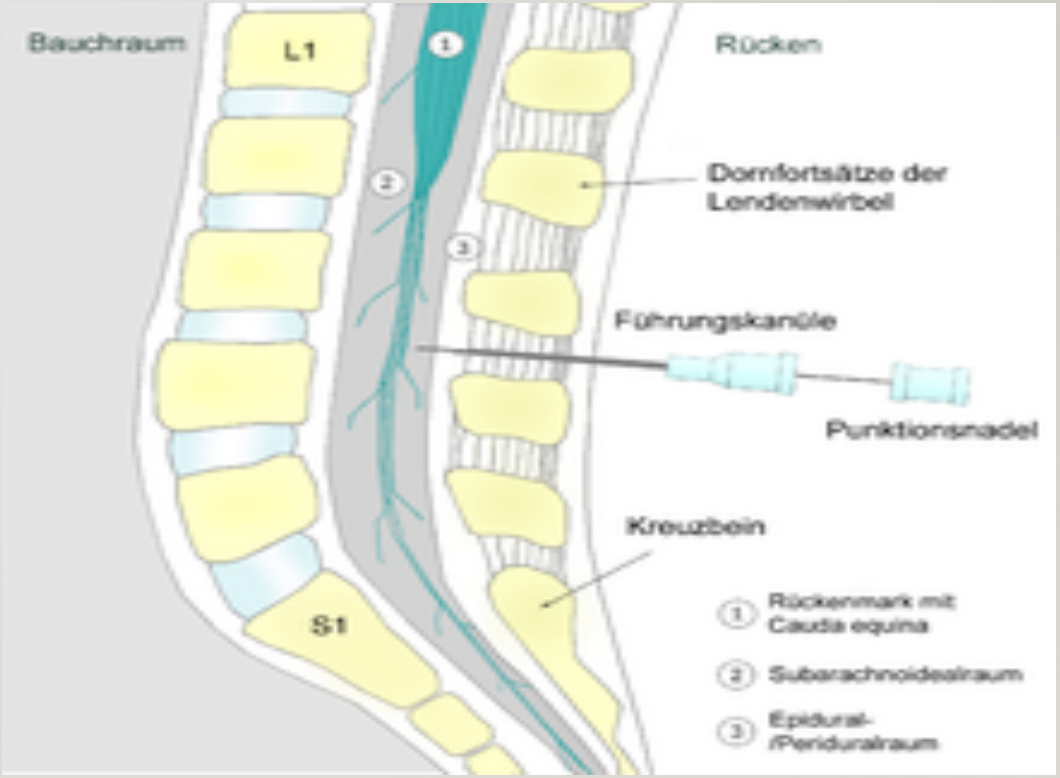
AFE: DIFFERENTIAL DIAGNOSIS

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



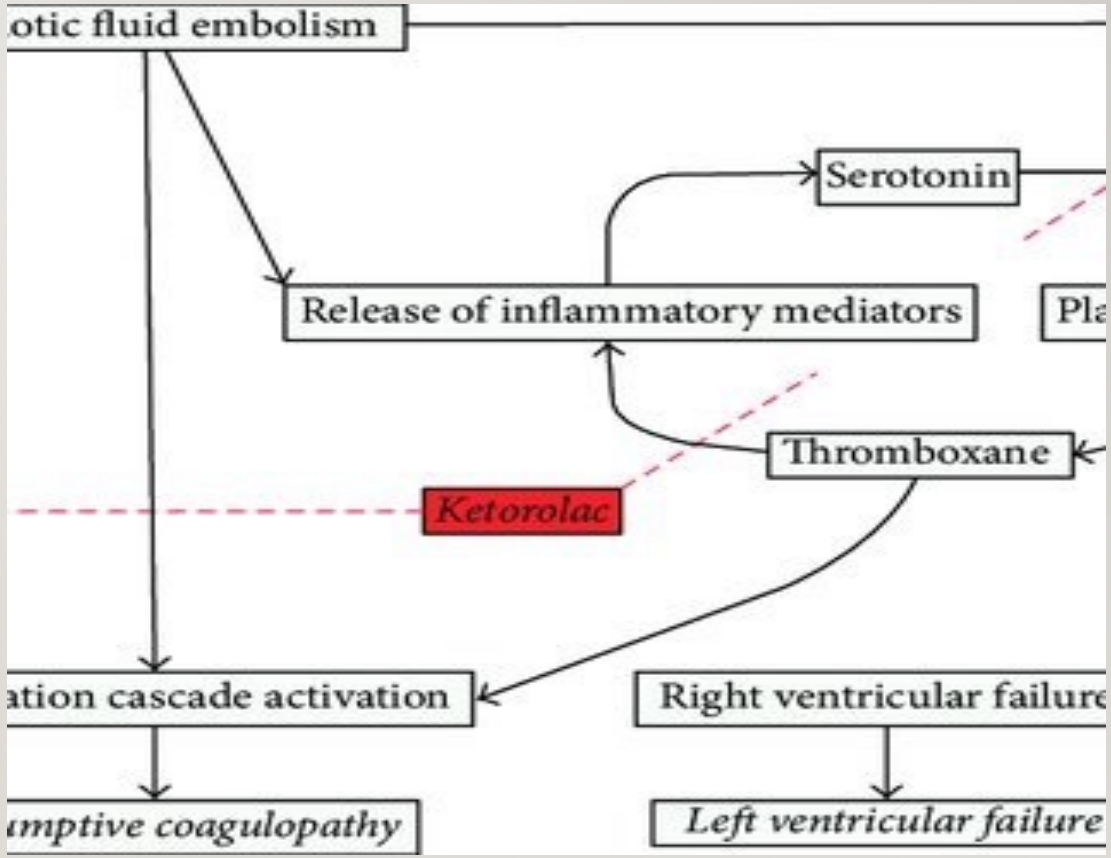
AFE: DIFFERENTIAL DIAGNOSIS

- Air embolism
- High spinal, local anesthesia toxicity
- Severe drug reactions
- Abrupt Maternal Hemorrhage



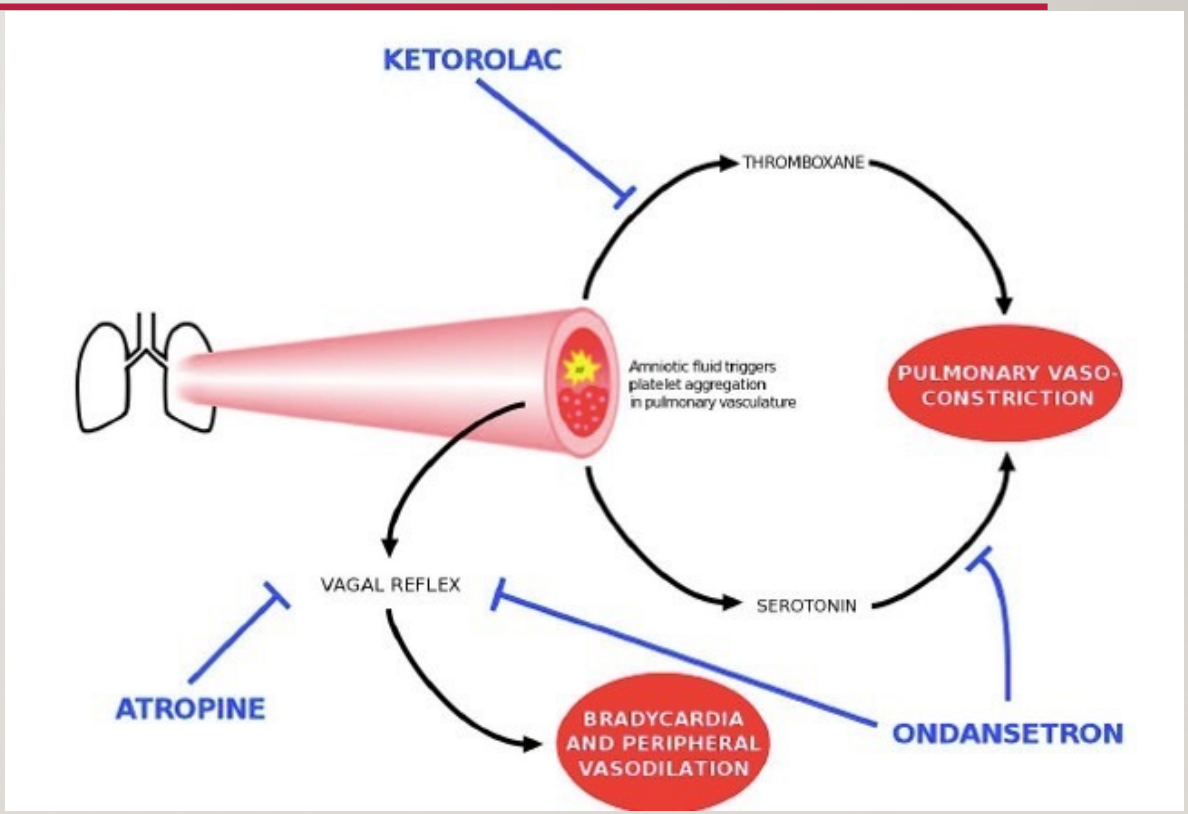
ANESTHESIA & AFE

- 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

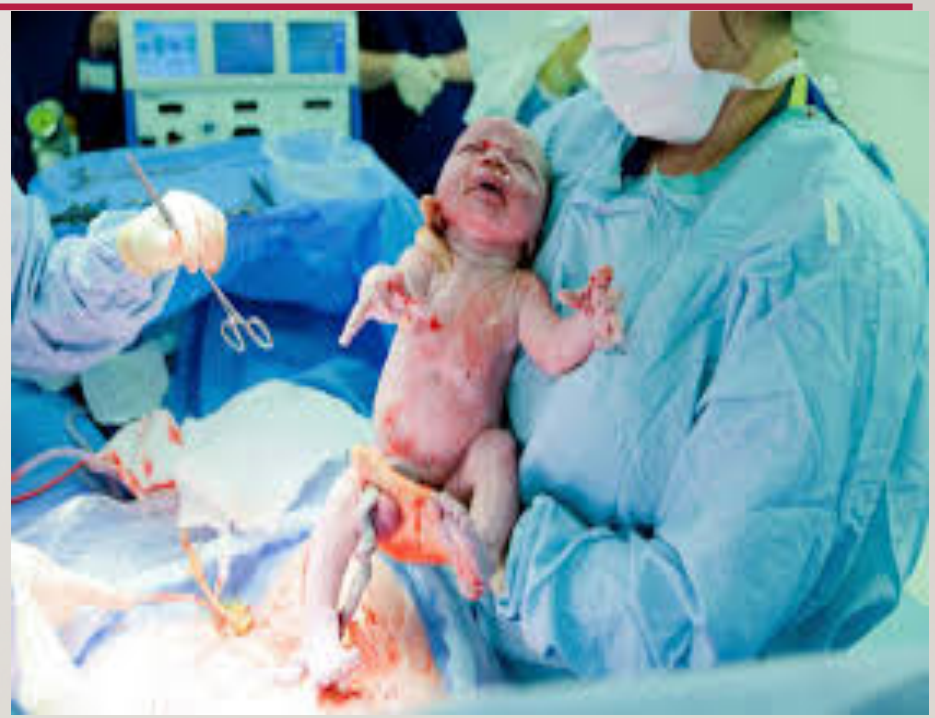
- Hypoxemia often profound resulting poor outcomes
- 50% of deaths occur in the first hour with those who survived seeing a about 80 percent neurological damage

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?