

AMNIOTIC FLUID EMBOLISM SYNDROME

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AMNIOTIC FLUID EMBOLISM SYNDROME

- Unpreventable, unpredictable and often-fatal complication of pregnancy
- Catastrophic condition
- Rare
(1.9-6.1 in every 100,000 deliveries)

AMNIOTIC FLUID EMBOLISM

- Rapid collapse of mother and/or baby as a result of an allergic-like reaction to amniotic fluid entering the maternal circulatory system
- Amniotic fluid is known to enter the maternal circulation through the endocervical veins, the placental insertion site, or a site of uterine trauma

AMNIOTIC FLUID EMBOLISM

TWO PHASES

- **Phase 1**: rapid respiratory failure and cardiac arrest
- **Phase 2**: hemorrhagic phase
 - Disseminated intravascular coagulopathy (DIC)
 - The coagulopathy associated with AFE is likely mediated by factor VII activation (extrinsic pathway) that is induced by tissue factor

AMNIOTIC FLUID EMBOLISM

- Diagnosis of exclusion
- It was once believed to be diagnosed only through autopsy based on the presence of fetal material found in the vasculature of the lungs. This is no longer a valid diagnostic criteria.

AMNIOTIC FLUID SYNDROME

- Precipitous or tumultuous labor
- Advanced maternal age
- Multiple gestation
- Male fetus
- Medical induction of labor
- Cesarean or instrumental deliveries
- Placenta previa and abruption or other abnormalities
- Grand multiparity (≥ 5 live or stillbirths)
- Cervical lacerations/trauma
- Fetal distress
- Eclampsia
- Medical induction of labor
- Uterine rupture
- Ethnic minority status

EARLY SIGNS AND SYMPTOMS

- A sudden sense of doom, agitation, anxiety, or change in mental status
- Hypotension
- Fetal Distress
- Nausea or Vomiting
- Shortness of Breath/dyspnea
- Skin Discoloration/cyanosis
- Frothing from the mouth
- Fetal heart rate abnormalities
- Loss of consciousness
- Seizure like activity
- Uterine atony post delivery

CLINICAL PRESENTATION

- 1) Respiratory distress, restlessness, and cyanosis
- 2) Hypotension due to cardiogenic shock/arrest (reported up to 85% of patients with AFES die from cardiogenic shock or its complications; half of these within the first hour.)
 - Cardiac dysrhythmias
 - Profound DIC
 - Bleeding from the uterus, incisions, or IV sites is common
- 3) Seizure (50%)
- 4) Coma
- 5) Death (60-90%)

TREATMENT



- There are no specific treatments for AFES
 - Correct hypoxemia and hypotension
 - Be prepared to administer CPR with lateral displacement of the uterus and modified ACLS for maternal physiology
 - Oxygenation
 - Early intubation and mechanical ventilation with O₂ at 100%
 - Increase the fraction of inspired oxygen, increase PEEP, and prolonging or inversing the inspiratory to expiratory ratio
 - Hemodynamic support
 - Intravenous fluids should be avoided in excess because fluid overload will result in further right ventricle dilation.
 - Blood products
 - Vasopressors and Inotropic medications

TREATMENT

- Central venous catheter or 2 large bore IV's
- Arterial line for accurate measurement of blood pressure
- Transesophageal echocardiography (right heart failure)
- Tranexamic Acid
- Expedited delivery of the fetus



A-OK TREATMENT FOR AFE



- Atropine is a competitive, reversible muscarinic antagonist that when bound causes parasympathectomy.
 - Muscarinic receptors are found throughout the body in multiple subtypes.
 - When bound to acetylcholine, muscle contraction occurs resulting in vagal mediated pulmonary artery spasm and pulmonary hypertension.
- Beneficial effects of Atropine for treatment of AFE:
 - Decreases vasoconstriction in pulmonary vasculature
 - Can help with bradycardia and heart blocks commonly observed in phase I of AFE.

A-OK TREATMENT FOR AFE



- O- Ondansetron (Zofran) is a 5-HT₃ receptor antagonist
 - 5-HT₃ receptors are a specific subtype of serotonin, which is a physiologic neurotransmitter with multiple functions.
 - 5-HT₃ receptors are in abundance in the vagal efferent terminals in the heart and lungs, not only acetylcholine/MAchR mediated function.
 - Approximately 50% of efferents are serotonergic in heart and lungs.
- Beneficial effects of Ondansetron for treatment of AFE:
 - Contributes to vagotomy via 5-HT₃ antagonism, which can prevent cardiovascular collapse.

A-OK TREATMENT FOR AFE



- K- Ketorolac (Toradol) is an intravenous non-steroidal anti-inflammatory, which is known to inhibit the production of prostaglandins, specifically thromboxane A₂.
 - Thromboxane A₂ functions:
 - Role in the activation and aggregation of platelets, which is a component of primary hemostasis
 - Central to the amplification of the hemostatic mechanism
 - Overproduction of thromboxane A₂ occurs during even a normal pregnancy
 - An abundance of thromboxane A₂ can lead to the cascade of inappropriate clotting seen with DIC
- Beneficial effects of Ketorolac for treatment of AFE:
 - Inhibits formation of clots and the extension of clots in situ
 - Decreases the cascade of inappropriate clotting

OUTCOMES

- Overall mortality (60-90%)
- One of the leading causes of maternal mortality
- Survivors have potential
 - neurologic injury
 - short term memory
 - hypoxic-anoxic brain injury.
- Neonatal outcomes are poor
 - Improve with early delivery

CURRENT RESEARCH

- AFE is still considered very rare. The exact mechanism of what causes AFE is unknown, and it remains unpreventable.
- AFE Foundation in collaboration with Baylor College of Medicine in Houston, Tx launched the first ever international patient registry which will be used as a basis for comprehensive clinical research on AFE.

Amniotic Fluid Embolism

Simulation Toolkit



REFERENCES

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