AMNIOTIC FLUID EMBOLISM (AFE)

ASEEM GROVER
GROUP NO 163 (2)

AMNIOTIC FLUID EMBOLISM

- AFE is thought to occur when amniotic fluid , fetal cells, hair, or other debris enter the maternal circulation.
- Ricardo Meyer (1926); reported the presence of fetal cellular debris in the maternal circulation.
- Steiner and Luschbaugh (1941) described the autopsy findings of eight cases of AFE.
- Until 1950, only 17 cases had been reported.
- AFE was not listed as a distinct heading in causes of maternal mortality until 1957 when it was labeled as obstetric shock.
- Since then more than 400 cases have been documented, probably as a result of an increased awareness.

AMNIOTIC FLUID EMBOLISM

- Overall incidence ranges from 1 in 8,000 to 1 in 80,000 pregnancies.
- 10% of maternal deaths in USA &16% in U.K.
- The first well-documented case with ultimate survival was published in 1976
  (Resnik R, et al. Obstet Gynecol 1976;47:295-8).
- 75 % of survivors are expected to have long-term neurologic deficits.
- If the fetus is alive at the time of the event, nearly 70 % will survive the delivery but 50% of the survived neonates will incur neurologic damage.

AMNIOTIC FLUID EMBOLISM

- Time of event:

  -During labor.

  -During C/S.

  -After normal vaginal delivery.

  -During second trimester TOP.

- AFE syndrome has been reported to occur as late as 48 hours following delivery.

Risk factors of AFE

- Advanced maternal age
- Multiparity
- Meconium
- Cervical laceration
- Intrauterine foetal death
- Very strong frequent or uterine tetanic contractions
- Sudden foetal expulsion (short labour)
- Placenta accreta
- Polyhydramnios
- Uterine rupture
- Maternal history of allergy or atopy
- Chorioamnionitis
- Macrosomia
- Male fetal sex
- Oxytocin (controversial)

Nevertheless, these and other frequently cited risk factors are not consistently observed and at the present time
Experts agree that this condition is not preventable.

Experimental AFE

The cardiorespiratory effects of acute intravascular injection of amniotic fluid have been studied in pregnant ewes :

- **The initial response was hypotension.**
- **A 40 % decrease in mean arterial pressure was followed by a 100 % increase in mean pulmonary artery pressure.**
- **Little change occurred in the left atrial pressure or the pulmonary artery wedge pressure.**
- **A 40 percent fall in cardiac output was associated with the rapid rise in pulmonary artery pressure.**
- **These changes resulted in a two- to threefold increase in pulmonary vascular resistance and a two- to threefold decrease in systemic vascular resistance.**

Experimental AFE

- Intravascular injection of amniotic fluid in rhesus monkeys failed to produce cardiovascular changes similar to the syndrome observed in pregnant ewes or humans.

Pathophysiology

- Poorly understood.

- Cotton (1996), has proposed a biphasic model.

Phase 1:

Amniotic fluid and fetal cells enter the maternal circulation biochemical mediators pulmonary artery vasospasm pulmonary hypertension elevated right ventricular pressure hypoxia myocardial and pulmonary capillary damage, left heart failure acute respiratory distress syndrome

Phase 2:

*** biochemical mediators DIC Hemorrhagic phase characterized by massive hemorrhage and uterine atony.***

Pathophysiology

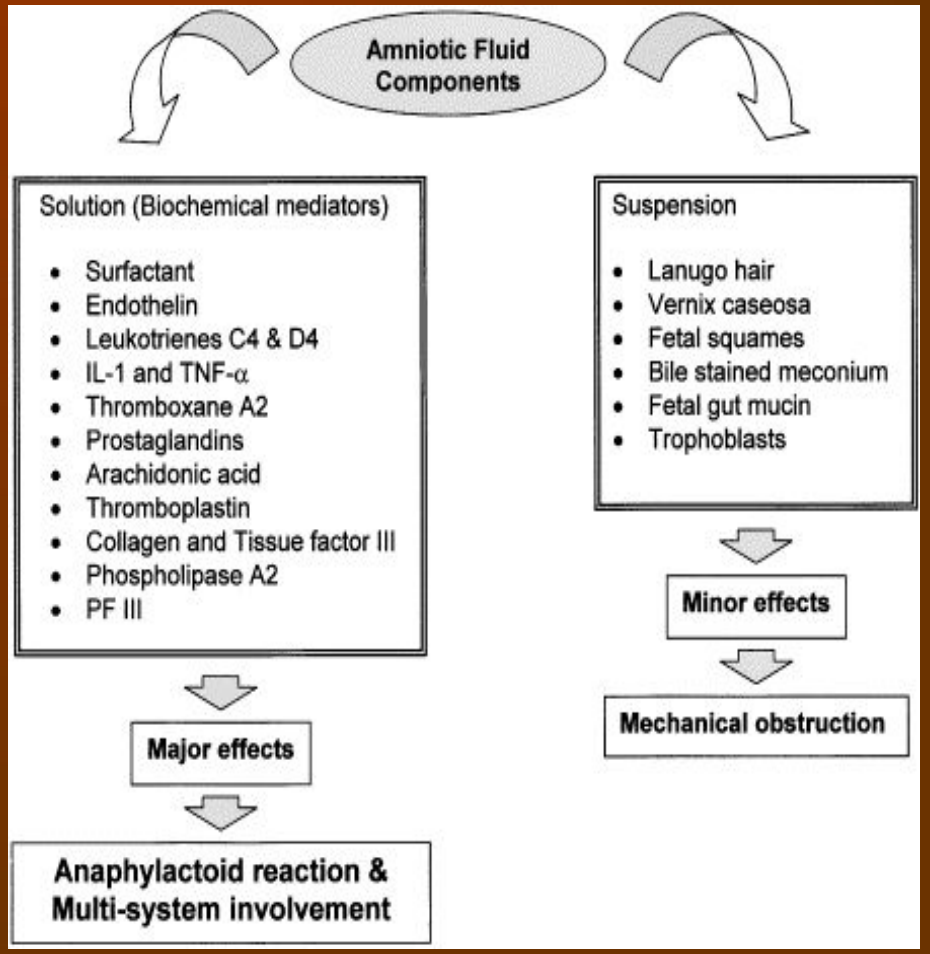
- The similar homodynamic derangements seen with AFE syndrome , anaphylactic, and septic shock have led investigators to postulate a substance in amniotic fluid resulting in the release of primary and secondary endogenous mediators (i.e. arachidonic acid metabolites) which might also be responsible for the associated coagulopathy in AFE.
- The prevention of fatal homodynamic collapse in experimental AFE with inhibitors of leukotriene synthesis would support an anaphylactic mechanism for AFE.

Pathophysiology

- Measurement of tryptase (a degranulation product of mast cells released with histamine during anaphylactic reactions) levels to further investigate the anaphylactic nature of AFE.
- The syndrome does not appear to be dependent on the amount of fluid or particulate matter that enters the vasculature.

Pathophysiology

- **To emphasize that the clinical findings are secondary to biochemical mediators rather than pulmonary embolic phenomenon; Clark et al have suggested renaming this clinical syndrome the *"anaphylactoid syndrome of pregnancy"***



Clinical presentation

The classic clinical presentation of the syndrome has been described by five signs that often occur in the following sequence:

(1) Respiratory distress
(2) Cyanosis
(3) Cardiovascular collapse ***cardiogenic shock***
(4) Hemorrhage
(5) Coma.

Clinical presentation

- A sudden drop in O_2 saturation can be the initial indication of AFE during c/s.
- More than 1/2 of patients die within the first hour.
- Of the survivors 50 % will develop DIC which may manifest as persistent bleeding from incision or venipuncture sites.

The coagulopathy typically occurs 0.5 to 4 hours after phase 1.

Clinical presentation

- 10-15% of patients will develop grand mal seizures.
- CXR may be normal or show effusions, enlarged heart, or pulmonary edema.
- ECG may show a right strain pattern with ST-T changes and tachycardia.

Diagnosis

- In 1941, Steiner and Luschbaugh described histopathologic findings in the pulmonary vasculature in 8 multiparous women dying of sudden shock during labor.
- Findings included mucin, amorphous eosinophilic material , and in some cases squamous cells.
- The presence of squamous cells in the pulmonary vasculature once considered pathognomonic for AFE is neither sensitive nor specific (only 73% of patients dying from AFE had this finding).
- The monoclonal antibody TKAH-2 may eventually prove more useful in the rapid diagnosis of AFE.

Laboratory investigations in suspected AFE

Non specific

- complete blood count
- coagulation parameters including FDP, fibrinogen
- arterial blood gases
- chest x-ray
- electrocardiogram
- V/Q scan
- echocardiogram

Specific

- cervical histology
- serum tryptase
- serum sialyl Tn antigen
- zinc coproporphyrin
- PMV analysis (if PA catheter *in situ*)

Differential diagnosis

Obviously depends upon presentation

- Anaphylaxis (Collapse)
- Pulmonary embolus (Collapse)
- Aspiration (Hypoxaemia)
- Pre-eclampsia or eclampsia (Fits, Coagulopathy)
- Haemorrhage (APH ; PPH)
- Septic shock
- Drug toxicity ($MgSO_4$, total spinal, LA toxicity)
- Aortic dissection

Management of AFE

GOALS OF MANAGEMENT:

- **Restoration of cardiovascular and pulmonary equilibrium**
  - **-Maintain systolic blood pressure >90 mm Hg.**
  - **-Urine output > 25 ml/hr**
  - **-Arterial pO_2 > 60 mm Hg.**
- **Re-establishing uterine tone**
- **Correct coagulation abnormalities**

Management of AFE

- As intubation and CPR may be required it is necessary to have easy access to the patient, experienced help, and a resuscitation tray with intubation equipment, DC shock, and emergency medications.
- IMMEDIATE MEASURES :

  -Set up IV Infusion, O_2 administration.

  -Airway control endotracheal intubation

  maximal ventilation and oxygenation.
- LABS : CBC,ABG,PT,PTT,fibrinogen,FDP.

Management of AFE

- Treat hypotension, increase the circulating volume and cardiac output with crystalloids.
- After correction of hypotension, restrict fluid therapy to maintenance levels since ARDS follows in up to 40% to 70% of cases.
- Steroids may be indicated (recommended but no evidence as to their value)
- Dopamine infusion if patient remains hypotensive (myocardial support).
- Other investigators have used vasopressor therapy such as ephedrine or levarterenol with success (reduced systemic vascular resistance)

Management of AFE In the ICU

- To assess the effectiveness of treatment and resuscitation, it is prudent to continuously monitor ECG, pO_2, CO_2, and urine output.
- There is support in literature for early placement of arterial, central venous, and pulmonary artery catheters to provide critical information and guide specific therapy.

Management of AFE In the ICU

- Central venous pressure monitoring is important to diagnose right ventricular overload and guide fluid infusion and vasopressor therapy. Blood can also be sampled from the right heart for diagnostic purposes.
- Pulmonary artery and capillary wedge pressures and echocardiography are useful to guide therapy and evaluate left ventricular function and compliance.
- An arterial line is useful for repeated blood sampling and blood gases to evaluate the efficacy of resuscitation.

Management of AFE Coagulopathy

- DIC results in the depletion of fibrinogen, platelets, and coagulation factors, especially factors V, VIII, and XIII. The fibrinolytic system is activated as well.
- Most patients will have hypofibrinogenemia, abnormal PT and aPTT and low Platelet counts
- Treat coagulopathy with FFP for a prolonged aPTT, cryoprecipitate for a fibrinogen level less than 100 mg/dL, and transfuse platelets for platelet counts less than 20,000/mm^3

Restoration of uterine tone

- Uterine atony is best treated with massage, uterine packing, and oxytocin or prostaglandin analogues.
- Improvement in cardiac output and uterine perfusion helps restore uterine tone.
- Extreme care should be exercised when using prostaglandin analogues in hypoxic patients, as bronchospasm may worsen the situation.

Sympathomimetic Vasopressor agent

Dopamine

- Dopamine increases myocardial contractility and systolic BP with little increase in diastolic BP. Also dilates the renal vasculature, increasing renal blood flow and GFR.
- DOSE: 2-5 mcg/kg/min IV; titrate to BP and cardiac output.
- Contraindications: ventricular fibrillation, hypovolemia, pheochromocytoma.
- Precautions: Monitor urine flow, cardiac output, pulmonary wedge pressure, and BP during infusion; prior to infusion, correct hypovolemia with either whole blood or plasma, as indicated; monitoring central venous pressure or left ventricular filling pressure may be helpful

Maternal Mortality in AFE

- Maternal death usually occurs in one of three ways: (1) sudden cardiac arrest, (2) hemorrhage due to coagulopathy, or (3) initial survival with death due to acute respiratory distress syndrome (ARDS) and multiple organ failure
- For women diagnosed as having AFE, mortality rates ranging from 26% to as high as 86% have been reported.
- The variance in these numbers is explained by dissimilar case definitions and possibly improvements in intensive care management of affected patients.

Further issues in the Management

- **Transfer:**

  Transfer to a level 3 hospital may be required once the patient is stable.
- **Deterrence/Prevention:**

  Amniotic fluid embolism is an unpredictable event.
- Risk of **recurrence** is unknown. The recommendation for elective cesarean delivery during future pregnancies in an attempt to avoid labor is controversial.
- **Perimortem cesarean delivery:**

  After 5 minutes of unsuccessful CPR in arrested mothers, abdominal delivery is recommended.

Medical/Legal Pitfalls

- **Failure to respond emergently is a pitfall. AFE is a clinical diagnosis. Steps must be taken to stabilize the patient as soon as symptoms manifest.**
- **Failure to perform perimortem cesarean delivery in a timely fashion is a pitfall.**
- **Failure to consider the diagnosis during legal abortion is a pitfall. A review of the literature indicates that most case reports of AFE have occurred during late second-trimester abortions.**

SUMMARY

- AFE is a sudden and unexpected rare but life threatining complication of pregnancy.
- It has a complex pathogenesis and serious implications for both mother and infant
- Associated with high rates of mortality and morbidity.
- Diagnosis of exclusion.
- Suspect AFE when confronted with any pregnant patient who has sudden onset of respiratory distress, cardiac collapse, seizures, unexplained fetal distress, and abnormal bleeding
- Obstetricians should be alert to the symptoms of

THANK YOU