AMNIOTIC FLUID EMBOLISM

A TRUE OBSTETRICAL EMERGENCY

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The purpose of this education activity is to enhance the knowledge of the Birthing Center Registered Nurse in the area of early recognition of amniotic fluid embolism by addressing the hallmark signs and symptoms in an effort to improve patient outcomes as evidenced by decreased mortality rate.
OBJECTIVES

Upon completion of this activity, attendees will be able to:

1. Identify the hallmark signs and symptoms of amniotic fluid embolism (AFE)
2. Understand the disease process of AFE
3. List the causes of AFE
4. Recall the incidence, occurrence, morbidity, and mortality rates related to AFE
5. Describe the nursing responsibilities in the care of a patient diagnosed with AFE
6. Explain the treatment involved in treating a patient with AFE
AMNIOTIC FLUID EMBOLISM (AFE)

- Is a rare obstetric emergency in which it is postulated that amniotic fluid, fetal cells, hair, or other debris enter the maternal circulation, causing cardiorespiratory collapse.
- In 1941, Steiner and Luschbaugh described AFE for the first time after they found fetal debris in the pulmonary circulation of women who died during labor.
Current data from the National Amniotic Fluid Embolus Registry suggest that the process is more similar to anaphylaxis than to embolism, and the term **ANAPHYLACTOID SYNDROME OF PREGNANCY** has been suggested because fetal tissue or amniotic fluid components are not universally found in women who present with signs and symptoms attributable to AFE.[1]
The pathophysiology of AFE is poorly understood. Based on the original description, it was theorized that amniotic fluid and fetal cells enter the maternal circulation, possibly triggering an anaphylactic reaction to fetal antigens.

However, fetal material is not always found in the maternal circulation in patients with AFE, and material of fetal origin is often found in women who do not develop AFE.
In any case usually during labor or other procedure, amniotic fluid and debris, or some as yet unidentified substance, enters the maternal circulation; this may trigger a massive anaphylactic reaction, activation of the complement cascade, or both.
Progression usually occurs in 2 phases.

In phase I, pulmonary artery vasospasm with pulmonary hypertension and elevated right ventricular pressure cause hypoxia. Hypoxia causes myocardial capillary damage and pulmonary capillary damage, left heart failure, and acute respiratory distress syndrome.

Women who survive these events may enter phase II. This is a hemorrhagic phase characterized by massive hemorrhage with uterine atony and DIC; however, fatal consumptive coagulopathy may be the initial presentation.
In the United States of AFE is estimated at 1 case per 8,000-30,000 pregnancies. The true incidence is unknown because of inaccurate diagnoses and inconsistent reporting of nonfatal cases.
Maternal mortality approaches 80%. However, it was 61% in the US national registry, which listed 46 cases.

AFE is the cause of 5-10% of maternal mortality in the United States.

Of patients with AFE, 50% die within the first hour of onset of symptoms.

Of survivors of the initial cardiorespiratory phase, 50% develop a coagulopathy.
Survival is uncommon, although the prognosis is improved with early recognition and prompt resuscitation.

Most women who survive have permanent neurologic impairment. Neonatal survival was 79% in the US registry and 78% in the UK registry.

The intact infant survival rate is 70%. Neurologic status of the infant is directly related to the time elapsed between maternal arrest and delivery.
Amniotic fluid embolism (AFE) usually occurs during labor but has occurred during abortion, after abdominal trauma, and during amnioinfusion.

A woman in the late stages of labor becomes acutely dyspneic with hypotension.

She may experience seizures quickly followed by cardiac arrest.

Massive DIC-associated hemorrhage follows and then death.

Most patients die within an hour of onset.
Currently no definitive diagnostic test exists. The United States and United Kingdom AFE registries recommend the following 4 criteria, all of which must be present to make the diagnosis of AFE[1, 9, 10]:

1. Acute hypotension or cardiac arrest
2. Acute hypoxia
3. Coagulopathy or severe hemorrhage in the absence of other explanations
4. All of these occurring during labor, cesarean delivery, dilation and evacuation, or within 30 minutes postpartum with no other explanation of findings
In case reports, patients are described as developing acute shortness of breath, sometimes with a cough, followed by severe hypotension. The following signs and symptoms are indicative of possible AFE:

- **Hypotension**: Blood pressure may drop significantly with loss of diastolic measurement.
- **Dyspnea**: Labored breathing and tachypnea may occur.
- **Seizure**: Tonic clonic seizures are seen in 50% of patients.
- **Cough**: This is usually a manifestation of dyspnea.
- **Cyanosis**: As hypoxia/hypoxemia progresses, circumoral and peripheral cyanosis.
- **Fetal bradycardia**: In response to the hypoxic insult, fetal heart rate may drop to less than 110 beats per minute (bpm). If this drop lasts for 10 minutes or more, it is a bradycardia. A rate of 60 bpm or less over 3-5 minutes may indicate a terminal bradycardia.
SIGNS AND SYMPTOMS

- Pulmonary edema: This is usually identified on chest radiograph.
- Cardiac arrest
- Uterine atony: Uterine atony usually results in excessive bleeding after delivery. Failure of the uterus to become firm with bimanual massage is diagnostic.
- Coagulopathy or severe hemorrhage in absence of other explanation (DIC occurs in 83% of patients.) [10]
- Altered mental status/confusion/agitation
AFE is considered an unpredictable and unpreventable event with an unknown cause. In the national registry, 41% of patients had a history of allergies.

Reported risk factors for development of AFE include multiparity, advanced maternal age, male fetus, and trauma.

In a retrospective review of a 12-year period encompassing 180 cases of AFE, of which 24 were fatal, medical induction of labor increased the risk of AFE.

In the same study, AFE was positively associated with multiparity, cesarean section or operative vaginal delivery, abruption, placenta previa, and cervical laceration or uterine rupture.
ANESTHETIC CONSIDERATIONS FOR AMNIOTIC FLUID EMBOLISM

COMMUNICATION IS KEY!
Communication is Key!

- What is the patient’s history?
- Physical exam
  - Vitals - BP, HR, RR, Sp02
  - Appearance, color, mental status
  - Estimated Blood loss
  - Labs
  - IV access
- What has been done?
## Estimated Blood Loss

<table>
<thead>
<tr>
<th>Percent Blood Loss</th>
<th>Physical Exam</th>
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<tbody>
<tr>
<td>&lt; 20%</td>
<td>No changes</td>
</tr>
<tr>
<td>20-25%</td>
<td>Tachycardia</td>
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<tr>
<td></td>
<td>Narrow pulse pressure</td>
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<tr>
<td></td>
<td>Orthostatic hypotension</td>
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<tr>
<td>30-35%</td>
<td>Hypotension</td>
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<tr>
<td></td>
<td>Oliguria</td>
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<tr>
<td></td>
<td>Tachypnea</td>
</tr>
<tr>
<td>&gt;40%</td>
<td>Shock</td>
</tr>
<tr>
<td></td>
<td>Altered mental status</td>
</tr>
<tr>
<td></td>
<td>Anuria</td>
</tr>
</tbody>
</table>

When to get Anesthesiology involved?

- ASAP
- How can we help?
  - Airway management
  - IV sedation
  - Analgesia
  - Supportive measures, IV access, Monitoring
What type of Anesthesia is appropriate?

- Depends on:
  - Timing – STAT?
  - Location - Patient’s room vs. OR
  - Volume status
  - Rate of blood loss
  - Airway/Mental status
  - Labs
Preparation for the OR

- Call for help!
- Administer an oral or intravenous antacid
- You **must** obtain at least 2 large bore IVs
- Continue resuscitation with colloid/crystalloids until blood products become available
- Type and crossmatch PRBCs, 2-4 units
  - Someone must call the blood bank
  - FFP and Cryoprecipitate take 30-45 minutes to thaw
  - Don’t forget rFactor VIIa and Kcentra
- Set up the OR
  - Y tubing, fluid warmer, aline/central line, bair hugger, drugs
- Don’t forget ECMO!!!!
Intraoperative Management

- Call for Help!
- RSI with cricoid pressure
- Hemodynamic monitoring (A-line, CVP, Foley)
- Keep the patient warm
- Send repeat labs
- Communication!!!!
- Decision to extubate depends on hemodynamic stability
- DO NOT REMOVE Epidural until coagulation profile has been normalized.
- Patient should be monitored in the ICU/IMU post operatively
AFE: NURSING RESPONSIBILITIES

QUICK RESPONSE IS THE KEY
Both Clark et al in the United States and Tuffnell2 in the United Kingdom have established national registries for suspected clinical occurrences of biphasic AFE.

They agree on **four hallmark signs of AFE**:

- acute hypotension or cardiac arrest
- acute hypoxia
- coagulopathy or severe clinical hemorrhage in the absence of other explanations
- all of these events must occur during labor, cesarean birth, or dilatation and evacuation within 30 minutes postpartum.

Clark et al have added **one additional qualifier**:

absence of any other significant confounding condition or potential explanation of the hallmark signs and symptoms.
In addition to the previously identified hallmark signs, the nurse should be alert for rapid decreases in blood pressure, sudden difficulty breathing, or any unexplained hemorrhage.
TREATMENT

- Prompt preparation for delivery increases the likelihood of survival for both mother and baby. Usually emergency Cesarean section.

- Prompt delivery of the fetus removes the obstruction the gravid uterus presents to maternal resuscitation.
Basic and advanced cardiac life support steps should already be in place.

Oxygenation is the key to avoiding irreversible neurologic injury for the patient.

Early tracheal intubation and mechanical ventilation are usually necessary to maintain normal oxygen saturation.

Monitoring of the patient with AFE includes continuous electrocardiographic (ECG) monitoring,
Pulse oximetry, and end-tidal carbon dioxide monitoring.
Clinicians should monitor the patient’s blood pressure continuously, if possible. These noninvasive monitors and ventilators are standard in ORs, but may not be immediately available in the obstetric department.
Quickly establish a large-bore peripheral IV catheter, a central venous pressure catheter, a pulmonary artery catheter, and a peripheral arterial line.

Gathering additional resources and manpower from the anesthesia and critical care staffs will help support the resuscitation team during this emergency.

If profound hemorrhage occurs, a transfusion of uncross-matched O-negative packed cells is recommended so transfusion is not delayed by waiting for type-specific and cross-matched blood.
Nursing personnel should see that coagulation studies for prothrombin time, partial thromboplastin time, D-dimer, fibrin split products, and platelets are sent immediately to the laboratory for analysis.

Coagulopathy and hemorrhage are common and often occur after the clinical diagnosis of AFE is made.

Disseminated intravascular coagulation (DIC) is found in 83% of patients with AFE.

Half of these patients may develop coagulopathy within four hours of the onset of the clinical symptoms.
RECOVERY

- In situations where the maternal patient responds favorably to these interventions and survives AFE, postpartum nursing activities will start directing her recovery; however, nurses should not wait to implement these actions.

- Performing and providing fundal massage can indicate the degree of uterine atony and the patient’s response to therapy.

- Administration of oxytocin, a medication familiar to obstetric nurses, will be required.

- Programming of IV pumps for oxytocin and the knowledge of its side effects of water intoxication from large infused volume may be unfamiliar to critical care or perioperative nurses.

- Keeping the obstetric nurse involved in the care of this patient after delivery, during resuscitation, and while providing supportive care allows the obstetric nurse to share his or her unique knowledge and expertise.

- A team approach to this lengthy—sometimes days-long—resuscitation effort will benefit the patient.
Unlimited family visitation in the PACU and CCU is instrumental in the maternal patient’s recovery, especially if the neonate is unable to visit or has died.

Despite neonatal outcome, facilitation of maternal-infant bonding should occur. This may mean having support persons in the PACU and working with the perinatal nursing staff members to safely transfer the neonate for visitation.

Pictures of the deceased neonate or family viewing and holding of the neonate's body has been shown to aid in grief resolution. Viewing should be encouraged throughout the patient’s hospitalization, and trips to the morgue to retrieve the neonate should be conducted with respect and modesty.
PATIENT CASE STUDY
JP is a 34 y.o. patient admitted on 10/23/14 at 1225 PM to OB Triage in early labor. She had been having regular contractions for 4 hours, no complaint of bleeding or ruptured membranes.

- Admission Vital Signs BP 120/60, HR 78, T-98.3
- Admission H&H was 12.7 and 38 and platelets were 252
- At 1407 the decision was made to admit JP for labor augmentation
JP is G6 P3 with 2 living children. She had 2 term babies born vaginally and 1 preterm stillbirth due to placental abruption. She also had 2 early miscarriages.

Her living children were 7lbs 5oz and 5lbs 10oz at delivery.

She is currently taking prenatal vitamins and iron.

She is allergic to Levoquin and Zithromycin.

She is a non-smoker and denies drug or alcohol use.

Surgical history: 2 D&C performed after spontaneous AB.
JP'S Prenatal labs are as follows:
O neg
HIV neg
RPR neg
Rubella Immune
Group B Strep neg
She received Rhogam at 28 weeks.
LABOR EVENTS

- 1407 JP was moved to a labor room. Anesthesia was notified of need for epidural.
- 1412 Epidural was placed and the procedure was uneventful
- 1430 Pitocin was started at 2mu/min
- 1500 Foley was placed and SVE was done, patient was now 8 cm
- JP had regular contractions, amniotic fluid was clear and fetal heart rate tracing was reactive.
- 1610 Patient was comfortable and had no complaints
1632-1642
- JP c/o SOB, chest pressure, and stated something is wrong.
- JP was pale and diaphoretic
- FHT's were bradycardic in the 60's
- O2 was applied, SVE 10cm, +2
- Dr. S. was called to come immediately
LABOR EVENTS (cont’d.)

VITAL SIGNS

- BP 90/40
- O2 sat 90
- HR 120
- 1642 Dr. S. arrived and baby boy was delivered with outlet forceps
  APGAR’s 7-9
JP continued to complain of SOB, chest pain and pressure, and her level of consciousness was decreased. At this time Dr. I., Anesthesiologist, was present at the bedside.

**VITAL SIGNS**
- 89/49
- 89/51
- 79/40
- HR 115-130 and thready
- O2 sats 89-93 %
- RR 18-22 shallow
Placenta was delivered without complications.

At 1700 large gush of bright blood (500ml) vaginally was noted.

Dr. S. was at the bedside and reassessed patient.

Over the next 30 minutes patient continued to have approximately 700ml blood loss.

2nd large bore IV was started and 2 units of PRBC's were ordered. JP continued to be hemodynamically unstable.

Methergine .2mg IM was administered, Misoprostol 1000 mcg was given rectally

Bedside curettage was performed and STAT H & H was drawn.
At 1815 the decision was made to take the patient to the OR for hysterectomy due to unabated vaginal bleeding.

After surgery patient was moved to ICU

JP received a total of 4 units of PRBC's, 2 units of FFP, and 1 unit of Cryoprecipitate in the first 36 post-partum hours.

Post op H&H was 9 and 28 and platelets were 97

On day 1 H&H was 7.1 and 12 and platelets were 102

24 hours later she was moved to the 5th floor

She was discharged home with her baby on day 4 post-partum fully recovered.
REFERENCES


