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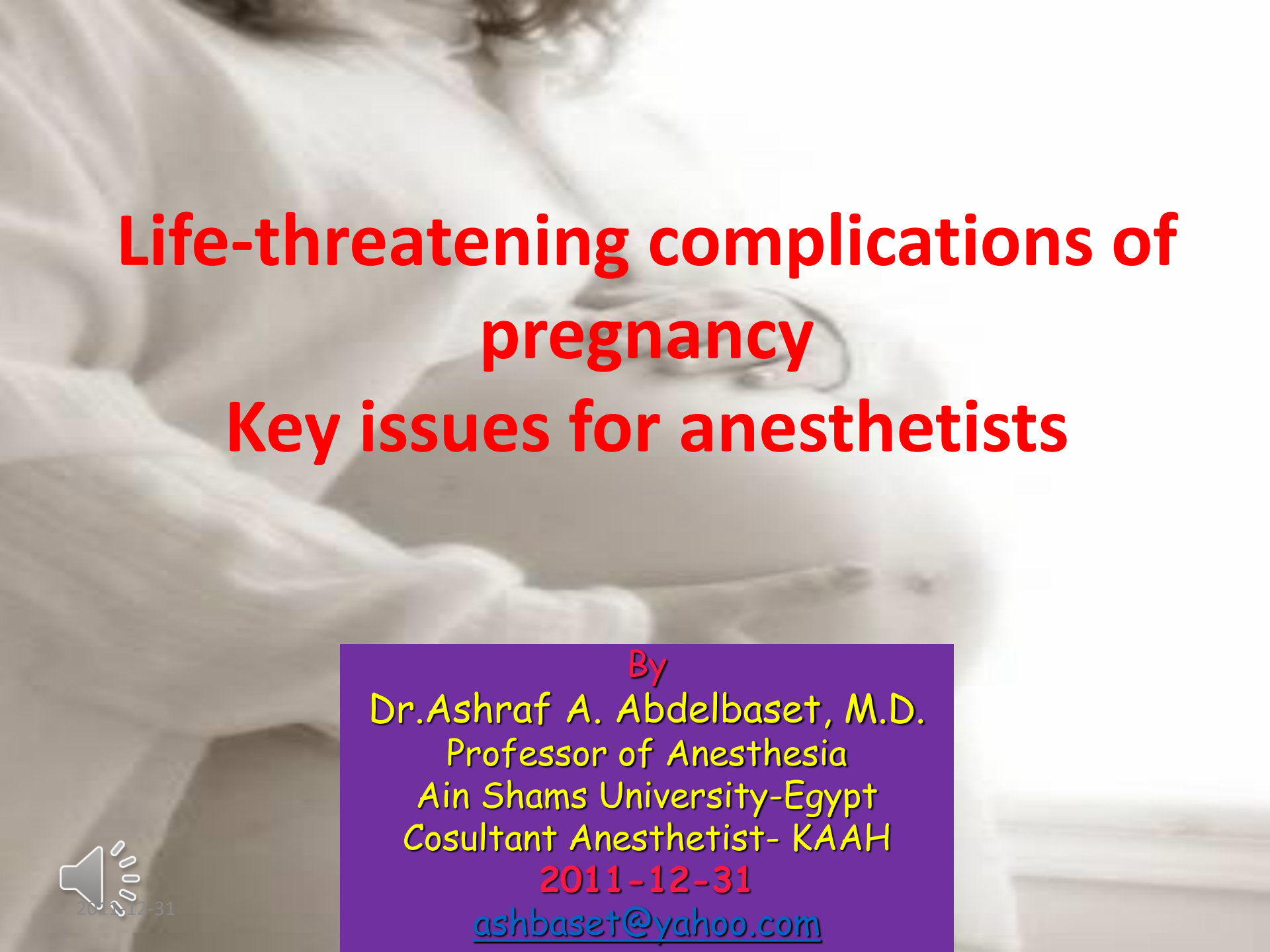

Life-threatening complications of pregnancy
Key issues for anesthetists

By
Dr.Ashraf A. Abdelbaset, M.D.
Professor of Anesthesia
Ain Shams University-Egypt
Cosultant Anesthetist- KAAH
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ashbaset@yahoo.com

Life-threatening complications of pregnancy
Key issues for anesthetists

1: 30 000 Pregnant Women will require Cardiopulmonary Resuscitation (CPR)

- Morris S, Stacey M. Resuscitation in pregnancy. Br Med J 2003

Anatomical and Physiological Effects of Pregnancy on Cardiopulmonary Resuscitation

Cardiovascular	**Aortocaval compression when supine** **Effective chest compressions difficult due to lateral tilt, rib flaring, breast size**
Respiratory	**Decreased functional residual capacity** **Increased oxygen demand** **Decreased chest wall compliance**
Gastro-intestinal	**Increased intra-abdominal pressure** **Increased risk of aspiration**
Other	**Paddle placement compromised by breast enlargement** **Increased incidence of liver, spleen and rib trauma during CPR due to altered chest shape and increased intra-abdominal pressure**

Causes of cardiovascular collapse in the obstetric patients

Hypovolaemia	• Obstetric haemorrhage • Non-obstetric haemorrhage
Hypoxia	Convulsions (eclamptic or other aetiology • Other neurological events • Total spinal anaesthesia •
Embolic	Pulmonary thromboembolism • Amniotic fluid embolism • Air embolism •
Toxic	Magnesium sulphate overdose • Local anaesthetic toxicity • Overdose of antihypertensive agents •
Other	Tension pneumothorax • Cardiac tamponade • Myocardial infarction • Sudden adult death syndrome •

Life threatening causes related to pregnancy

- Severe pre-eclampsia & eclampsia.
- Severe obstetric hemorrhage.
- Thrombo-embolic disorders.
- Amniotic fluid embolism.
- Uterine atony.

Hypertensive disorders of pregnancy

- Etiology: Heterogeneous group of diseases of incompletely understood etiology.
- Incidence: 6% of all pregnancies and 15-20% of maternal mortality in developed countaries.
- Include pregnancy-induced hypertension, preeclampsia, eclampsia, and the syndrome of Haemolysis, Elevated Liver enzymes and Low Platelets (HELLP).

Co-morbidities associated with severe pre-eclampsia and eclampsia

- Placental abruption
- D.I.C/HELLP
- Pulmonary oedema
- Acute renal failure
- Aspiration pneumonia
- Stroke
- Cardiopulmonary arrest

Monitoring

- **Central invasive monitoring is rarely needed to guide therapy.**
- **In pre-eclampsia there is poor correlation between central venous and pulmonary artery wedge pressures.**
- **However, Central venous monitoring might be helpful when arterial pressure interpretation is further complicated by sepsis or hemorrhage.**

Monitoring

- **Central venous catheterization should ideally be undertaken via a peripheral site In the presence of coagulopathy.**
- **Jugular venous cannulation should only be undertaken with ultrasound guidance.**
- **Intra-arterial monitoring can be very helpful for assessing the acute response to antihypertensive therapy and for repeated blood sampling.**

Treatment of hypertensive disorders in pregnancy

- **Early diagnosis.**
- **The aim of treatment should be to keep arterial pressure below 170/110mm Hg and above 130/90mm Hg in order that maternal cerebral, renal, and uteroplacental circulations are not compromised.**
- **The mainstays of hypertension management are intravenous (i.v.) boluses of hydralazine (2.5–5 mg) or labetalol (10–20 mg) every 10–20 min. Nifedipine (5 mg) is effective by the sublingual route.**
- **Start Mg sulphate.**

Eclampsia

- **The term eclampsia describes convulsions (when there is no other aetiology) in usually the 2nd and 3rd trimesters of pregnancy, or postpartum, in women with symptoms or signs of pre-eclampsia.**

- **Eclampsia complicates about 1 in 2000 deliveries in Europe and developed countries.**

- **Eclamptic seizures can be the first manifestation of a hypertensive disorder of pregnancy, in the absence of hypertension and proteinuria.**

Warning manifestations of impending eclampsia

- Persistent occipital or frontal headache.
- Blurred vision, photophobia.
- Epigastric and/or right upper quadrant pain.
- Altered mental status…… Seizures
- Can be antepartum, intrapartum or postpartum.

The principles of management of eclampsia

- **The basic principles of emergency management i.e. Airway, Breathing, Circulation.**
- **Patent airway must be maintained and attempts made to prevent aspiration of gastric contents.**
- **Oxygen at high FIO2 should be administered via face mask.**
- **The patient should be nursed in the lateral decubitus position to minimize the risk of aspiration and to avoid aortocaval compression.**

Magnesium and the anaesthetist

- **Mg** is the agent of choice for primary and secondary prevention of eclamptic seizures.
- **Mg** reduces cerebral and systemic vasospasm.
- **Mg** is administered as an initial i.v. bolus dose of 5 g over no less than 10 min, followed by a maintenance infusion of 1 g /h

Magnesium and the anaesthetist

- Patients receiving Mg infusions must be regularly assessed for Mg toxicity:
- Nausea, vomiting are early symptoms.
- flushing an early sign.
- Arterial blood pressure, pulse, respiratory rate.
- Patellar reflexes should be assessed hourly.
- Regular laboratory: measurement of serum Mg concentration (therapeutic range 2–3 mmol/l) is useful but not mandatory.

Management of magnesium toxicity

- **Stop Mg infusion.**
- **100% oxygen should be administered via a facemask.**
- **Cardiovascular and ventilatory support given as required.**
- **10-20 ml of 10% calcium chloride or gluconate should be administered i.v. over 5–10 min.**
- **Doses of non-depolarizing muscle relaxants must be reduced and monitoring of neuromuscular function during general anesthesia is mandatory.**

Anesthesia
in severe pre-eclampsia and eclampsia

Regional techniques confer advantages over GA :

- **Avoidance of the pressor response to tracheal intubation and failed intubation secondary to laryngeal oedema.**

- **Facilitates continued monitoring of neurological status.**

- **Epidural and combined spinal–epidural anesthesia facilitate postoperative analgesia by infusion.**

- **Hypertension of pre-eclampsia is not sympathetically mediated So phenylephrine and ephedrine can be used in spinal induced hypotension in standard doses without causing an exaggerated hypertensive response.**

Anesthesia
in severe pre-eclampsia and eclampsia

General anaesthesia is preferable in the presence of :

- **coagulopathy.**
- **S/S of impending eclampsia (eclamptic seizure is not necessarily a contraindication to regional anesthesia).**

Regional anaesthesia may be appropriate after:

- **Single seizure if consciousness has been fully regained.**
- **Platelet count is greater than 100 000mm3.**
- **Serum Mg concentration is within the therapeutic range.**

Anesthesia
in severe pre-eclampsia and eclampsia

General anaesthesia requires special considerations :

- **Risk of *difficult intubation* due to laryngeal edema.**

- ***Pressor response* to intubation will be exaggerated and must be obtunded to reduce the risk of intra cerebral hemorrhage e.g. i.v. alfentanil (10 ugkg1) and pediatrician should be aware.**

- ***Direct arterial pressure monitoring* should be considered prior to induction of general anesthesia if time permits.**

- **Before *extubation*, consider specific therapy (e.g. labetalol in 10–20 mg increments) to oppose a dangerous pressor response.**

Anesthesia
in severe pre-eclampsia and eclampsia

- If a swollen larynx was evident at laryngoscopy, or intubation was traumatic, post-extubation stridor is a possibility and a period of postoperative ventilation may be considered.

- Adequate analgesia is important to reduce hypertension in the postoperative period.

- (NSAIDs) should be avoided for at least the first 24 h postpartum as renal function may be impaired and coagulation indices deranged.

Thromboembolic disease

- The leading direct cause of maternal death despite thrombo-prophylaxis.
- Early detection and treatment of thromboembolic disease is vital to reduce maternal mortality and morbidity.
- Attempts should be made to prevent the development of thromboembolic disease by identification of patients at risk and institution of appropriate prophylaxis.

Thromboembolic disease

Risk factors:

Pregnancy results in a hypercoaguable state that is enhanced by additional risk factors including :

- Obesity
- Qperative delivery
- pre-eclampsia
- Excessive blood loss
- Immobility.

Thromboembolic disease

Presentation:

•Often non-specific but include dyspnea, collapse, chest pain, hemoptysis, tachycardia, cough, apprehension, faintness and raised jugular venous pressure.

•There may also be symptoms and signs of deep vein thrombosis.

•The onset of symptoms can be rapid and dramatic—most deaths from pulmonary embolism occur within 6 h of the onset of symptoms..

Thromboembolic disease

Acute management:

- The treatment of acute thromboembolic disease is dependent upon the severity of the clinical situation and the local availability of resources and expertise.
- *Anticoagulation* provides the mainstay of management of acute thromboembolic events.
- *Early supportive therapy* should be instituted, including high fractional inspired oxygen, fluids, inotropes and CPR if required.
- I.V. unfractionated heparin by infusion remains the preferred treatment in massive pulmonary embolism but low molecular weight heparin regimens have gained acceptance.

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
- 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 fetal death
- Strong frequent uterine contractions
- Sudden fetal expulsion (short labour)
- Placenta accreta
- Polyhydramnios
- Uterine rupture
- Maternal history of allergy
- 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.

Pathophysiology of AFE

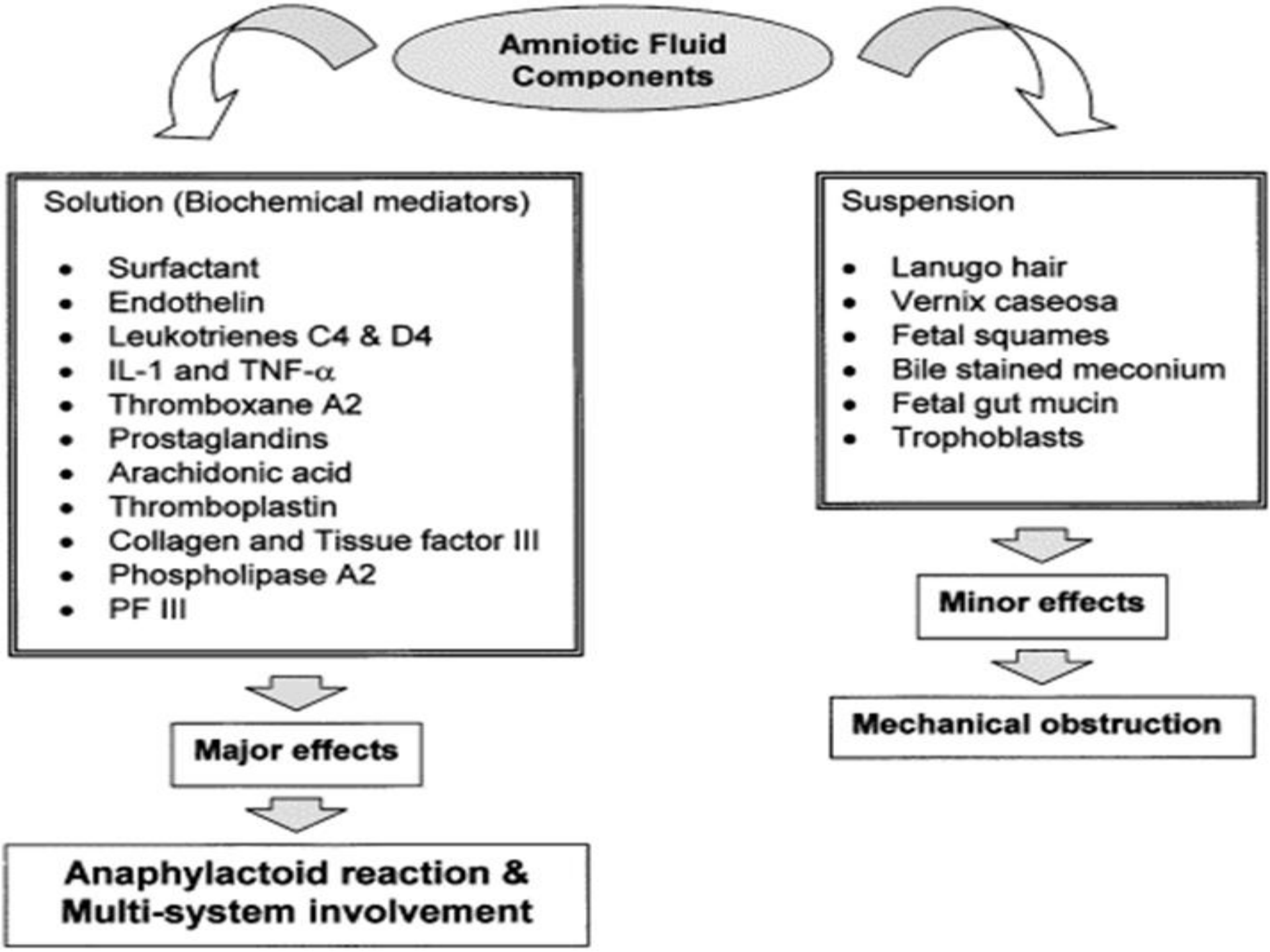
- Poorly understood.
- Cotton (1996), has proposed a biphasic model.

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.


Amniotic Fluid Components
Solution (Biochemical mediators)
Surfactant
Endothelin
Leukotrienes C4 & D4
IL-1 and TNF-α
Thromboxane A2
Prostaglandins
Arachidonic acid
Thromboplastin
Collagen and Tissue factor III
Phospholipase A2
PF III
Major effects
Anaphylactoid reaction & Multi-system involvement
Suspension
Lanugo hair
Vernix caseosa
Fetal squames
Bile stained meconium
Fetal gut mucin
Trophoblasts
Minor effects
Mechanical obstruction

Clinical presentation of AFE

*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.**

Laboratory investigations in suspected AFE

Non specific

- CBC
- Coagulation parameters
- ABG
- Chest x-ray
- EKG
- V/Q scan
- Echocardiogram

Specific

- Cervical histology
- Serum tryptase
- Serum sialyl Tn antigen
- Zinc coproporphyrin

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

- CPR to:
  - 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

- ***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

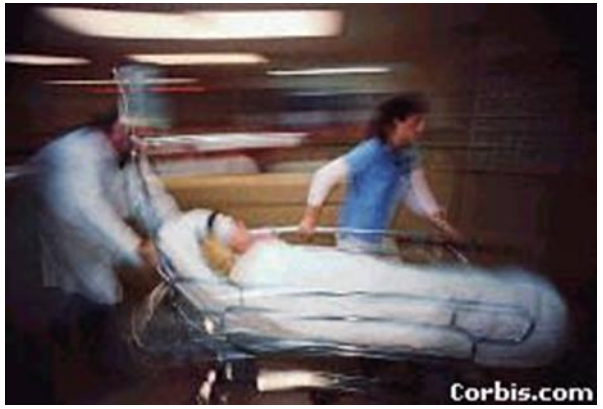
⇨maximal ventilation and oxygenation.

- ***LABS*** **: CBC,ABG, Coagulation profile.**

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).**
- ***vasopressor therapy*** **such as ephedrine or levarterenol**

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.
- *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

Maternal Mortality in AFE

- **Maternal death usually occurs in one of three ways:**

(1)Sudden cardiac arrest

(2)Hemorrhage due to coagulopathy

(3) Initial survival then death due to acute respiratory distress syndrome (ARDS) and multiple organ failure.

- **mortality rates ranging from 26% to 86% .**

Further issues in the Management

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

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 AFE and strive for prompt and aggressive treatment**

Massive obstetric hemorrhage

- **Massive hemorrhage remains a leading cause of maternal mortality and morbidity.**

- ***Severe obstetric hemorrhage is defined :***

as estimated blood loss greater than 1500 ml, peripartum fall in hemoglobin concentration of greater than 4 g dl or an acute transfusion of more than 4 units of blood.

- **Hemorrhage can be antepartum (APH) or postpartum (PPH), is frequently unexpected and can rapidly become catastrophic.**

Massive Obstetric Hemorrhage

- **The bleeding may be concealed (intraperitoneal) making accurate assessment of blood loss difficult.**

- **Disseminated intravascular coagulopathy (DIC) can complicate and contribute to massive obstetric hemorrhage.**

- ***Causes of DIC specific to obstetrics:***

Placental abruption, AFE, severe preeclampsia and prolonged intra-uterine fetal death..

Management of massive obstetric hemorrhage

- **There should be regular ‘fire drills’ to ensure staff familiarity with the protocols.**

- **Women at high risk of hemorrhage must be identified early, delivered in a center with appropriate facilities and have a multidisciplinary management plan.**

- **Primary management should be resuscitation with ongoing diagnosis and treatment of the cause.**

Management of Massive Obstetric Hemorrhage

- **Two large bore i.v. cannulae** should be sited and volume resuscitation commenced.

- **Initial volume replacement** should consist of 2 l of 0.9% saline or Hartmann's solution followed by colloid until blood is available.

- **Blood product replacement** should commence early. Group O negative RH blood must be available on all obstetric units and given until type-specific or fully cross matched blood becomes available.

Anesthetic Management of Massive Obstetric Hemorrhage

- **There is no place for regional anesthesia when hemorrhage has resulted in maternal cardiovascular instability.**
- **Physiological compensation for hemorrhage can mask the magnitude of the loss.**
- **Rapid sequence induction of general anesthesia is required and left lateral tilt should be used in cases of APH.**
- **Etomidate (0.3 mg kg1) or ketamine (1.5–2mg kg1) are preferable to thiopental if there is cardiovascular instability.**

Management of massive obstetric hemorrhage

- ***Invasive monitoring :***

arterial and central venous pressure should be considered but must not delay resuscitation.

- ***Coagulopathy :***

will dictate avoidance of jugular venous cannulation without ultrasound guidance.

Uterine atony

- **Syntocinon:** The first line treatment is oxytocin administered as a 5 unit i.v. bolus. It causes vasodilatation and hypotension especially in a cardiovascularly unstable patient.

- **E rgometrine** is the second-line agent. It causes uterine and vascular smooth muscle contraction and is administered as an intramuscular dose of 500 ug repeated after 2–4h. It can cause hypertension due to action at alpha and beta-adrenergic receptors and should be avoided in pre-eclampsia.

Uterine atony

- **Prostaglandin F 2** : third-line drug administered by deep intramuscular injection (250 mg repeated at an interval not less than 15min to maximum dose 2mg).

<u>However, it can cause:</u>

- 1.Intrapulmonary shunting and maternal hypoxaemia.
- 2.Nausea and vomiting.
- 3.Bronchoconstriction and should not be used in patients with asthma.

Uterine atony

Surgical intervention will depend on the clinical situation, and include:

- Bimanual compression.
- Balloon tamponade.
- B-Lynch suture.
- Selective embolization of uterine and hypogastric arteries by interventional radiologists.
- Ligation of uterine or hypogastric arteries
- Hysterectomy.

Blood management

- **Regular assessment** of hemoglobin concentration, platelet count and coagulation status, including fibrinogen concentration, to ensure appropriate administration of blood, platelets, fresh frozen plasma and cryoprecipitate.

- **Recombinant factor VIIa**, given as an initial i.v. bolus dose of 60 mgkg1, has been used successfully in refractory obstetric hemorrhage.

- **Red cell salvage** can help to avoid or reduce the requirement for donated red cell transfusions.

TAKE HOME MESSAGE

Acute life-threatening complications can arise during both pregnancy and the early postpartum period and result in maternal and fetal morbidity and mortality.

Substandard care and poor communication have been identified repeatedly as contributory factors to adverse outcomes from obstetric emergencies.

Prompt recognition of life-threatening conditions and early effective multidisciplinary management are essential to ensure optimal maternal and fetal outcome.

Dr. ashraf abdulbaset


Thank you for listening