SHOCK IN OBSTETRICS

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“shock during pregnancy is one of the most difficult problems faced by the obstetricians and necessitates initiation of management even before full identification of its cause for better survival”

DEFINITION

- It is a clinical condition arising out of an inability of the circulatory system to provide adequate tissue perfusion causing cellular hypoxia and organ damage
- It is a systemic disorder affecting multiple organ systems
- Perfusion may either be decreased throughout the body or distributed poorly
- Incidence- it accounts for 0-3%

Types and causes

Hemorrhagic shock

- Hemorrhagic shock due to hypovolemia is the most common cause of shock in obstetrics
- </= 1000ml-compensated
- 1000-1500ml-mild
- 1500-2000ml-moderate
- >2000ml-severe

Non-hemorrhagic shock

- Septic shock due to infections
- Hypertensive disorders
- Anaesthesia
- Cardiogenic
- Neurogenic
- embolism

Hypovolemic or hemorrhagic shock

- Causes
- Commonest of all is atonic PPH

- Early pregnancy
  - Abortion
  - Ectopic pregnancy
  - Gestational trophoblastic disease
- Antepartum hemorrhage
  - Placenta previa
  - Abruptio placenta
  - Rupture uterus
- Post partum hemorrhage
  - Traumatic PPH
  - Atonic PPH

Clinical picture

- Pallor
- Rapid and thready pulse
- Low blood pressure
- Cold clammy extremities
- Air hunger
- Diminution of vision
- Oliguria
- Anuria

Phases of hemorrhagic shock

Phase of compensation

- Blood loss less than 15%
- Postural hypotension is noted
- Sympathetic stimulation is the initial response leading to peripheral vasoconstriction to maintain blood supply to vital organs
- ↓ venous return causes ↓CO due to constriction of pre and post capillary sphincters

Clinical picture

- Pallor
- Tachycardia
- Normal blood pressure
- Tachypnea
- Sweating
- Hyperventilation
- At this phase, transfusion resuscitation and control of hemorrhage are usually effective in restoring the normal circulation and perfusion

Phase of decompensation

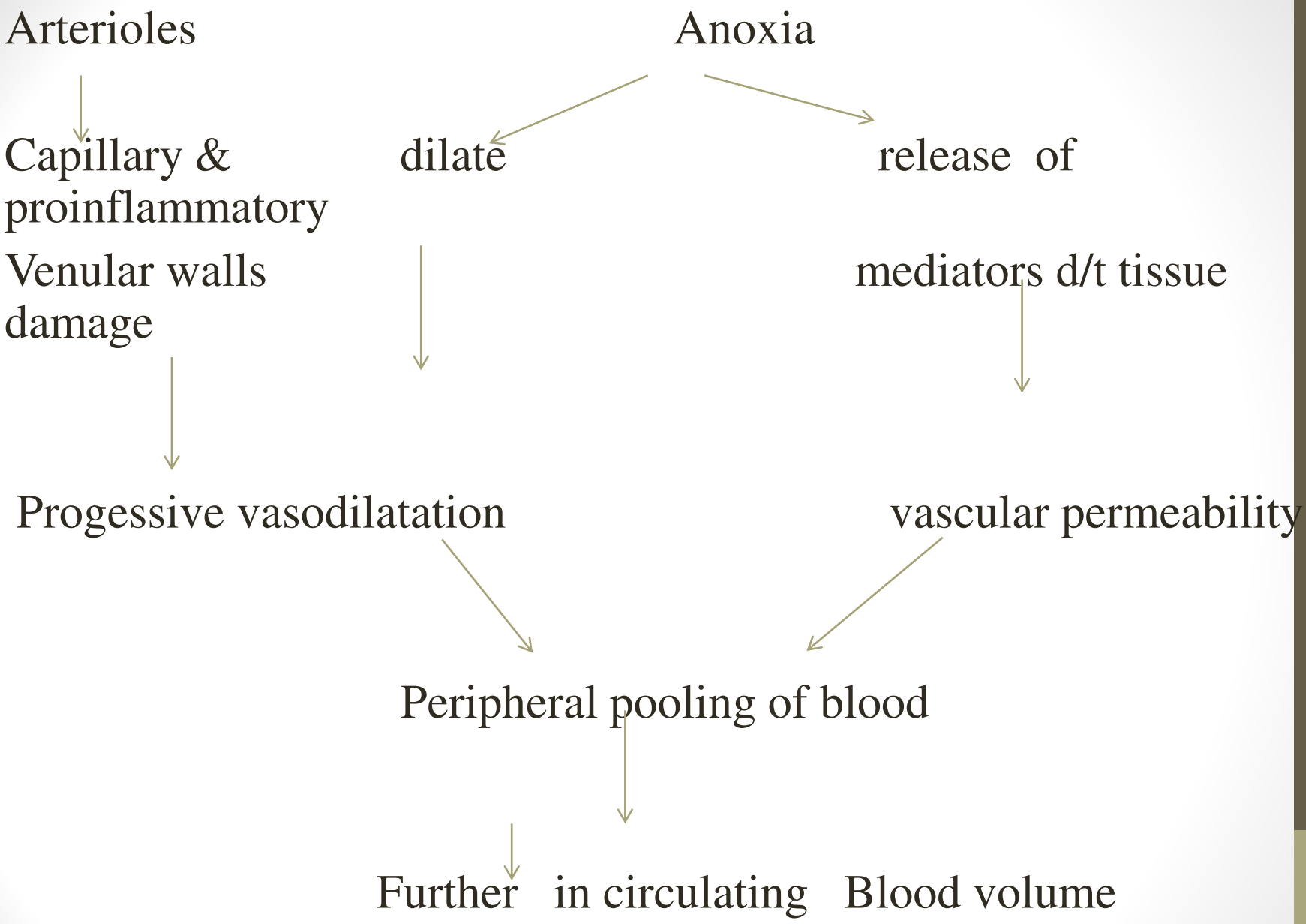
- Blood loss is 20-35%
- Blood loss exceeds 1000ml in normal patient or less if other adverse factors are operating like severe anemia.
- There is relaxation of precapillary sphincters and damage to the microcirculatory bed due to thromboxane A2 and leukotriens.

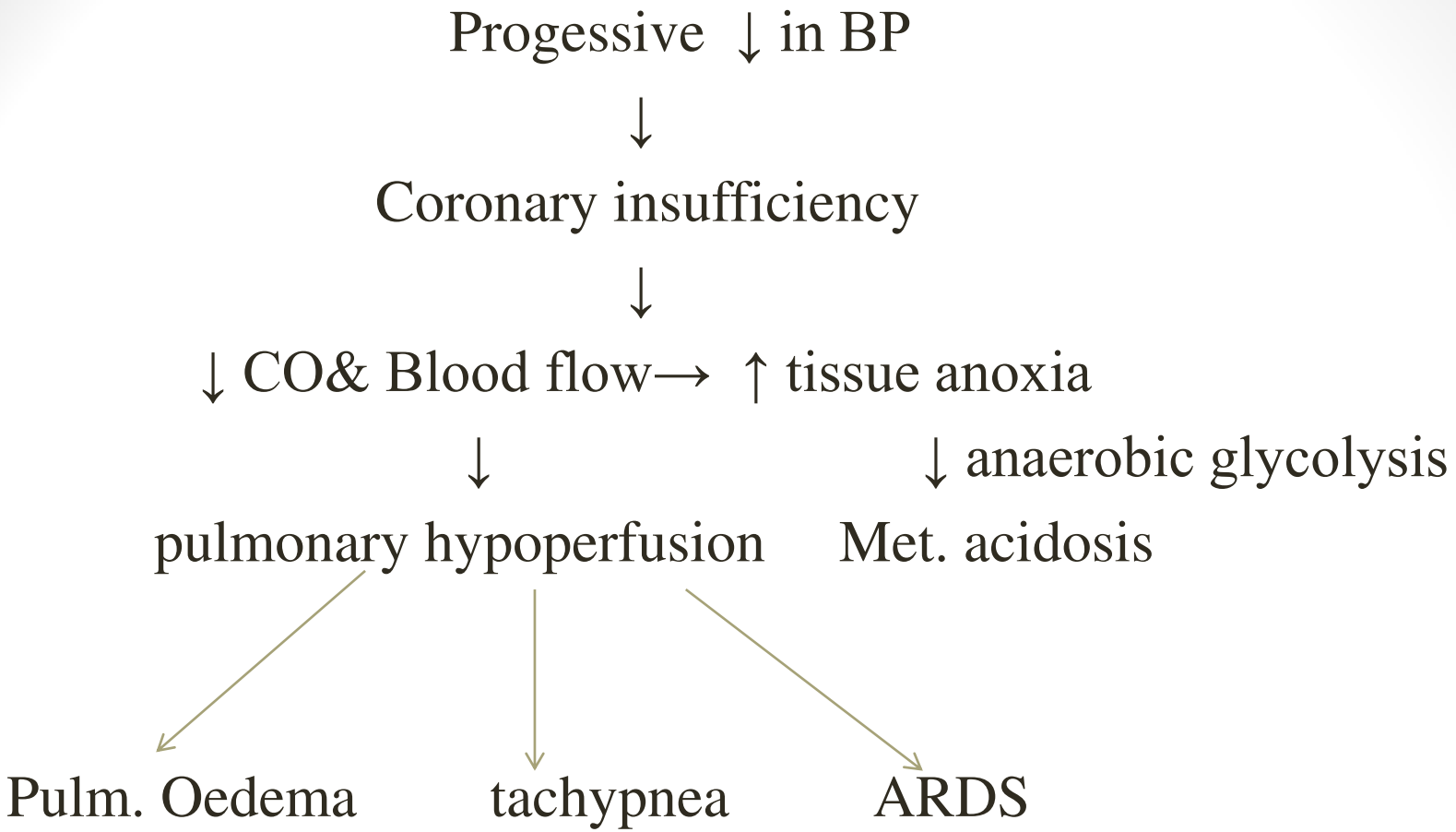
Clinical picture

- classic clinical picture of shock
- Cold and clammy skin
- Tachycardia
- Tachypnea
- Low pulse pressure
- Low systolic pressure
- Adequate treatment at this phase improves
- If untreated it can become irreversible by passing onto the phase of cellular damage

Irreversible decompensated shock

- Blood loss is more than 40%
- Profound hypotension with only the carotid pulse being palpable
- Oliguria and anuria are noted


Arterioles
Anoxia
Capillary & proinflammatory
dilate
release of
Venular walls damage
mediators d/t tissue
Progessive vasodilatation
vascular permeability
Peripheral pooling of blood
Further
in circulating
Blood volume


Progessive ↓ in BP
↓
Coronary insufficiency
↓
↓ CO& Blood flow→ ↑ tissue anoxia
↓ ↓ anaerobic glycolysis
pulmonary hypoperfusion Met. acidosis
Pulm. Oedema
tachypnea
ARDS

Hypercoagulability of blood

- Tissue damage→activates coagulation cascade

  ↓

  slowing of blood stream &

  vascular thrombosis

  ↓

  hypercoagulability of blood

- Clinically at this stage the patient has features of coma, worsened heart function and progressive renal failure due to acute tubular necrosis

Management of hemorrhagic or hypovolemic shock

"speed is vital and rapid restoration of the circulating blood volume is the key to successful outcome"

- Establishment of airway and oxygen therapy at a rate of 6-8 L per minute to maintain O2 saturation of > 92% and PaO2 of 80-100 mm Hg
- ET intubation and mechanical ventilation are required for severe hypoxia, severe tachypnea and coma to maintain oxygen supply
- Establishment of two wide bore IV lines
- Elevation of legs to facilitate venous return
- Keep the woman warm

- Volume replacement should initially be done by crystalloid solutions
- Plasma expanders, like, hemaccel may also improve the microcirculation
- They will remain in circulation for 24hrs to 48hrs
- Plasma protein fraction or fresh frozen plasma and later, when available, whole blood preferably packed cells should be transfused quickly
- Insert a foleys catheter to measure urine output
- Positioning of the patient in the trendelenburg tilt may aid the venous return

Drug therapy

- Analgesics- 10-15 ml morphine intravenously if, there is pain, tissue damage or irritability
- Corticosteroids- hydrocortisone 1g or dexamethasone 20mg slowly (IV), It may reduce the perepheral resistance and potentiate cardiac response to improve tissue perfusion
- Sodium bicarbonate 100 mEq IV, if metabolic acidosis is demonstrated

- Vasopressors to increase the blood pressure so as to maintain renal perfusion

a) Dopamine 2.5-10 micrograms/kg/min by IV infusion is the drug of choice
b) Beta adrenergic stimulant isoprenaline 1mg in 500ml 5% glucose slowly IV infusion
c) Digitalization may be carried out if inspite of volume replacement the CVP remains high due to myocardial failure. This is unlikely unless as a terminal event
d) Vasodilators – phenoxybenzamine an alpha receptor blocker is given as 1mg/kg over 1 hr IV if it is felt that the vasoconstriction is persisting inspite of adequate volume replacement

- As this management is being done, simultaneously ultrasound examination is carried out to find the cause of blood loss and assess the fetus in antenatal patients and to rule out placental bits in postpartum patients
- In addition to medical management, surgical intervention depending upon the cause of hemorrhage like emergency cesarean for antepartum hemorrhage, laparotomy, internal iliac ligation or hysterectomy for severe postpartum hemorrhage may be required

Monitoring is done by-

- Assessment of central venous pressure
- Pulse rate
- Blood pressure
- Urine output
- Pulmonary capillary wedge pressure
- By clinical improvement in
  - pallor
  - Cyanosis
  - Air hunger
  - Sweating

Non haemorrhagic shock

Septic shock

- It refers to sepsis- related hypotension that persists in spite of adequate fluid replacement
- Incidence 1 in 8000
- Maternal mortality can reach 13% in severe sepsis and up to 30% in septic shock.
- Neonatal mortality can be as high as 40%

Predisposing factors

- Septicemia and septic shock usually occur following maternal infection at one of the following sites:

➢ pyelonephritis due to anatomic alterations in the renal tract in pregnancy

➢ Endometritis in the puerperium due to large area of denuded maternal tissues exposed to bacteria

➢ Septic abortion where inadequate evacuation of the products provides a nidus for bacterial proliferation

➢ Perforation of uterus with peritonitis

➢ Infection of surgical wounds, where a breach in the skin is liable for bacterial contamination

Clinical criteria for diagnosis of sepsis

- Infection and bacteremia may lead to sepsis, which is diagnosed by the presence of the following signs:

- Fever or hypothermia
- Tachycardia
- Tachypnea
- Leukocytosis or leukopenia
- Thrombocytopenia
- Hypoxemia
- Oliguria
- Increased serum creatinine

contd…….

- As the sepsis worsens, severe sepsis is diagnosed by the appearance of the following signs:
  - Hypotension
  - Worsening oliguria- urine output 30ml/hr for 2 hrs
  - Worsening renal failure- serum creatinine

> 2mg/dl

  - Acute lung injury
  - Serum bilirubin >2mg/dl
  - Platelets<1000000 mm3
  - Prothrombin time >1.5

Lysis of gram –ve bacteria

↓

Endotoxin (LPS) in circulation + LPS protien

↓

CD14 molecule on surface of monocyte/ macrophage

↓ elaborates proinflammatory Cytokines

TNF α & IL-1

↓

Promote vascular injury

Vasodilatation & hypotension

Activationof inflammatory responses

- C5a and C3a → microemboli and endothelial damage
- mast cells → histamine release and increased capillary permeability
- Coagulation system→enhances development of thrombi
- Kinin system→releases bradykinin, causes vasodilation and increased vascular permeability

- The net result of above mechanisms is vasodilatation and increased vascular permeability in septic shock
- Profound peripheral vasodilatation and pooling of blood causes hyperdynamic circulation in septic shock, incontrast to hypovolemic shock
- Increased vascular permeability causes development of inflammatory oedema
- DIC is prone to develop in septic shock due to endothelial cell injury by toxins
- Reduced blood flow produces hypotension, inadequate perfusion of cells and tissues

Causative organisms and toxins

Causative organisms	Toxins
• E.coli, klebsiella,pseudomonas cause pyelonephritis and endo metritis	Endotoxin
• Anaerobes and above mentioned bacteria causes pelvic infection and septic abortion	Endotoxin
• Group A hemolyticstreptococci staph aureus	Toxic shock syndrome like toxin
methicillin resistant staph auerius	TSST 1
Clostridium perfringens	Super antigen
Causes wound infection	exotoxin

Clinical features of septic shock

- Abrupt onset of fever, chills, and tachycardia

  It has the following phases

- Warm phase- phase of perepheral vasodilatation

  Tachycardia, Hypotension, Warm extremities, Low CVP due tomarked decrease in intravascular fluid volume

- Cold phase- phase of peripheral vasoconstriction

  Elevated CVP due to cardiac failure,Poor tissue perfusion and

  lactic acidosis,Tachypnea, Adult respiratory distress syndrome, Altered sensorium, DIC

- Multiorgan failure due to hypotension and DIC

  Acute renal failure, Altered LFT, Respiratory failure, Cardiac failure

Diagnosis of septic shock

- **History**
- Risk factors-
- Septic abortion
- Puerperal endometriosis
- Pyelonephritis
- Wound infection
- **Symptoms and signs**
- Fever, chills
- Hypotension
- Tachycardia, Tachypnea
- **Evidence of multiorgan failure**Oliguria/anuria, Respiratory distress, DIC, Cardiac failure, Altered sensorium

Management

- When sepsis is suspected, a three- pronged strategy is instituted
- All the steps are undertaken concurrently to
- Initiate emergency goal-directed treatment
- Identify the organism and antibiotic therapy
- Find the source of infection

Emergency goal directed treatment

- Achieve hemodynamic stability
- The women should be admitted to ICU
- IV access should be established through a central vein
- The bladder is catheterized and urine sent for culture
- Rapid infusion of IV NS 2-4 L to get the CVP upto 8-12 cm and maintaining a urine output of 30-50ml/hr is mandatory
- O2 administration through venturi mask to achieve a saturation of >95% and PaO2 of >65%. If this is unsuccessful, the patient may need to be intubated and ventilated
- Patients in whom hypotension persists even after rapid IV fluid administration, pressor agents like dopamine, norepinephrine, dobutamine may have to be cosidered

Identification of organisms and antibiotic therapy

Blood, urine, and pus are sent for culture

Pending culture reports, , antibiotics are started

- Initial therapy
  - Ampicillin or augmentin and gentamicin, Meropenam and aztreonam for gram- negative infections
- Additional therapy- surgical wound infections
  - Cloxacillin - suspected staphylococcal infection
  - Vancomycin/linezolid-suspected MRSA
  - High dose benzyl penicillin 20,00,000 units 24hrly –suspected group A beta hemolytic streptococci
  - Clindamycin- necrotizing fasciitis
  - High dose benzyl penicillin and clindamycin/ metronidazole- suspected clostridial myositis

Identification of site of infection

- Unless the site of infection is identified and the pus or infected tissue removed, it is not possible to eradicate the infection.
- Site of infection is identified by clinical examination, ultrasonography, computerized tomography, and/ magnetic resonance imaging when required
- If the uterus is found to be gangrenous, an emergency hysterectomy may be lifesaving.
- Pelvic abscess should be drained by colpotomy.
- Laparotomy is indicated when there is intraabdominal collection of pus

Neurogenic shock

- May be due to painful conditions like

- Acute inversion
- Rapid evacuation of uterine contents
- Vasovagal stimulation
- Spinal anaesthesia may cause serious hypotension due to blockade of the normal sympathetic vasomotor tone

Management

- General measures like fluid replacement
- Correction of acidosis
- Vasoactive drugs
- Corticosteroids
- Ventilation and elimination of the source of neurogenic stimulus

Acute inversion

- Acute inversion after delivery can be the cause of neurogenic shock
- Causes-MC mismanaged 3rd stage of labor

Diagnosis

- the classic triad of hemorrhage, shock and severe pelvic pain
- due to traction on the ligaments supporting the uterus causing para-sympathetic stimulation or due to hypovolaemia as a result of acute bloodloss
- A bimanual examination reveals cupped or the missing fundus.
- Vaginally, there is a fleshy reddish mass seen outside the vulva.

Prevention

- Proper training of all skilled birth attendants in the correct management of third stage of labor is essential to prevent this complication
- Early diagnosis, and immediate reposition without removal of placenta if attached are important to reduce the associated morbidity and mortality

Management

- Immediate resuscitation must be started simultaneously with the efforts to reduce inversion
- Intravenous fluids must be infused with a wide bore cannula pending the arrival of cross-matched blood for transfusion.
- Ergometrine or oxytocin should not be given, as these will only aggravate matters and make reduction or replacement of the uterus more difficult
- Inversion should be immediately replaced without attempting to remove the placenta from the inverted fundus, which can be delivered later

Amniotic fluid embolism

- It is a rare obstetric emergency in which amniotic fluid, fetal cells, hair or other debris enter the maternal circulation, causing cardiorespiratory collapse

Risk factors

- Older maternal age
- Induction of labor using prostaglandins instead of oxytocin
- Multiple pregnancy
- Polyhydramnios
- Placenta previa
- Placental abruption
- Operative deliveries
- Eclampsia
- Cervical lacerations and uterine rupture

Diagnosis

1. Sudden onset of cardiorespiratory arrest, or both hypotension i.e. systolic blood pressure <90 mmHg and respiratory compromise
2. DIC after appearance of the initial signs or symptoms. Coagulopathy must be detected before loss of sufficient blood to itself account for dilutional or shock-related consumptive coagulopathy
3. Clinical onset during labor or within 30mins of delivery of placenta
4. No fever during labor

Management

- Patient should be admitted to ICU
- Management is supportive as there is no specific therapy for AFE
- Immediate administration of O2 and IV fluids are most important measures to prevent further hypoxia and restore circulation
- If hypotension does not respond to fluids alone, vasopressors like dopamine or norepinephrine should be infused
- Deranged coagulation parameters should be treated by,cryoprecipitate or fibrinogen FFPs
- Plasmapheresis and haemofiltration have been shown to be successful in arresting the progress of the disease , probably by clearing the plasma of cytokines

Pulmonary thromboembolism

- It occurs due to thrombus blocking pulmonary artery
- The symptoms depend on size of the artery obstructed and there by the area of the lung which is not perfused
- Women are at higher risk of venous thromboembolic disease in pregnancy due to the physiological changes in pregnancy which promote venous stasis and hypercoagulability of blood
- The risk increases further during the postpartum period due to endothelial injury that occurs during delivery

Signs and symptoms

- Sudden onset dyspnoea
- Chest pain
- Features of collapse like tachycardia, cold clammy skin and syncope
- Chest pain may occur anywhere in the chest and may radiate to the shoulder, arm or jaw
- It is often associated with cough and haemoptysis

Diagnosis and treatment

- The diagnosis is easier in women with clinical suspicion of deep vein thrombosis like redness, swelling and tenderness over a vein in one of the legs
- Women with previous history of DVT should have a thrombophilia screen, preferably before pregnancy, because of the effect of pregnancy
- Low molecular weight heparin is as effective as unfractionated heparin for the acute treatment of pulmonary embolism
- Anti-coagulation must be continued for 3-6 months with either LMWH, or alternatively warfarin, which can be started once the acute phase is over
- Warfarin is not contraindicated during lactatiuon

Air embolism

- For an air embolus to enter the circulation, atleast part of the placental site must be exposed
- Risk factors
  - Trendelenburg position
  - Abruptio placentae
  - Placenta previa
  - Exteriorization of the uterus
  - Manual extraction of placenta
  - Severe preeclampsia
  - APH
  - Hypovolemia

Symptoms and diagnosis

- A lethal embolism may follow a bolus of

3-5ml of air

- Tachypnoea
- Chest pain
- Gasping
- The diagnosis may be facilitated by pre-cordial doppler monitoring, transoesophageal echocardiography

Management

- Unfortunately there is seldom time for effective treatment
- A useful immediate first aid procedure is to place the patient in the head down, lateral position in the hope of displacing the bolus of air towards the apex of the right ventricle
- Management includes aspiration of air, discontinuation of nitrous oxide, administration of 100% oxygen and flooding the surgical site with saline to avoid further air entry

Rupture uterus

- It is a condition that carries a very high mortality, if neglected through failure to diagnose it
- The diagnosis is not easy, particularly in incomplete rupture, but if shock persists inspite of adeqauate blood transfusion to replace bloodloss, this possibility should be excluded

DIC

- DEFINITION

Activation of coagulation in the microcirculation by entry of large amounts of tissue thromboplastin or widespread endothelial injury leading to activation of the intrinsic pathway of coagulation and consumption of coagulation factors with a resultant bleeding diathesis

Changes in normal pregnancy

- Pregnancy is considered to be a compensated hypercoagulable state due to the changes that occur in the coagulation pathways
- Platelet count marginally decreases, but platelet aggregation increases
- There is an increase in fibrinogen and factors 7,8,9,10
- Thrombin activation is enhanced
- The fibrinolytic pathway as represented by plasmin activity is partly suppressed

Causes of DIC in pregnancy

- Placental abruption- large amounts of thromboplastin at the sites of abruption
- Amniotic fluid embolism- fetal squames, fetal antigens, anaphylactic reaction
- Sepsis syndrome- endotoxins, exotoxins, SIRS,cytokine storm, endothelial injury
- Eclampsia and HELLP syndrome- endothelial injury
- IUFD- release of thromboplastin from placenta
- Acute fatty liver of pregnancy- endothelial injury, devcreased production of coagulation factors from liver

Clinical features

- Bleeding from venepuncture sites
- Ecchymoses
- Oozing/bleeding from incisions/lacerations/placental site

1. Episiotomy
2. Cesarean section incisions
3. Profuse vaginal bleeding

- Hypotension and shock
- Symptoms due to clotting in microvasculature

1. Tissue hypoxia and lactic acidosis
2. Decreased urine output
3. Metabolic acidosis
4. Acidotic breathing
5. Hypoxia and tachypnea, altered sensorium

Diagnosis

- A H/O predisposing obstetric events such as abruption, sepsis, or amniotic fluid embolism is usually present
- Clinical features of bleeding, ecchymoses, or end-organ failure are sufficient to make a clinical diagnosis
- Whole blood clotting time is markedly prolonged, a bedside clot retraction test can be performed by collecting a blood sample in a plain tube and observing the time taken for formation of clot, in established DIC, clot may not be formed for several hrs and even if a clot is formed, it is soft and friable and does not retract

cont…….

- All bleeding parameters such as bleeding time, clotting time, prothrombin time, partial thromboplastin time, and thrombin time are prolonged
- Perepheral smear shows thrombocytopenia and schistocytes
- Plasma fibrinogen is markedly decreased. High levels of fibrinogen and fibrin split products are present in the perepheral blood, these products inturn inhibit formation of fibrin and cause a vicious cycle

Management

- Control of hemorrhage, replacement of blood and blood products, and treatment of the underlying cause
- Packed cells are used for correction of anemia
- Platelet concentrates for treating thrombocytopenia
- Fresh frozen plasma or cryoprecipitate to replenish deficient factors
- Recombinant factor 7a can be used in uncontrollable bleeding, but its use may be associated with increased risk of stroke or pulmonary embolism
- Concurrently the underlying obstetric condition should be promptly managed.

Cardiogenic shock

- Circulatory collapse caused by failure of the heart to pump blood adequately
- Etiology

Failure of left ventricular ejection due to

Cardiac arrest

Myocardial infarction

Failure of ventricular filling

Cardiac tamponade

Pulmonary embolism

Any cause of obstetric shock can result in cardiac arrest

Cardiac arrest

- A variety of conditions, pregnancy related and non-pregnancy related can cause cardiac arrest
- Most frequent reasons for cardiac arrest in pregnancy and postpartum are obstetric hemorrhage (38.1%) followed byAFE(13.3%), acute coronary syndrome (10%) and venous thromboembolism in 4%
- Anaesthesia
- Bleeding
- Cardiovascular disorders
- Drugs
- Embolism
- Sepsis
- General causes like metabolic and electrolyte imbalance
- Hypertensive disorders including stroke

Management

- It is same as in non pregnant woman
- Epinephrine is vasopressor of choice and should be administered by intravenous or intraosseous access above the diaphragm
- Prompt resuscitation of the mother provided on the spot can save both maternal and fetal life
- However resuscitation is difficult in pregnant patients due to the physiological changes of pregnancy

System	Changes in pregnancy	Effects on resuscitation
Cardiovascular	• Increased blood volume 40-50% • RBC volume- 20% • Cardiac output increases by 40% • Increased oxygen consumption by 20%	• Dilutional anaemia • Decreased O2 carrying capacity • Increased circulation demand
Respiratory	Increased oxygen consumption by 20%	Rapid decrease in PaO2 in hypoxia
GIT	• Delayed gastric emptying • Relaxed gastro-oesophageal sphincter	Aspiration of gastric contents
uteroplacental	Aortocaval compression	Decreased cardiac output, supine hypotension

Physiological changes in pregnancy and cardiopulomonary resuscitation

- The implication of these hemodynamic changes in a bleeding pregnant patient may lose up to 30-35% of her blood volume before manifesting signs like tachycardia and hypotension, although the fetal circulation suffers severely due to this loss
- An abnormal fetal heart pattern may be the first sign of signinificant maternal blood loss
- oxygen demand of the pregnant mother increases and the functional residual capacity of the lungs decreases due to elevation and splinting of the diaphragm by the enlarging uterus, intubation and ventilation may be difficult in obese women.
- Gastroesophageal sphincter is less competent due to the hormonal effects of the pregnancy which increase the risk of regurgitation of gastric contents and aspiration pneumonitis, H2 receptor antagonists can be given to reduce gastric acidity
- Patient should be placed in left lateral position to releive aortocaval compression.

Basic life support

- Must be instituted immediately while preparations are being made to gain an intravenous access to correct hypovelemia

❑ Airway and breathing

➢ Head tilt, chin lift manouver after ruling out head and neck injuries, jaw thrust manouver in case of head and neck trauma are recommended to open the airway

➢ Suction can be used to clear the airway

➢ 100% oxygen should be provided by a reservoir bag

➢ Carotid pressure should be maintained to prevent gastric contents regurgitation

- Circulation-
  - Chest compressions are given by applying rhythmic pressure over the sternum, which initiates circulation by increasing the intrathoracic pressure and directly stimulates the heart
  - Patient should be placed in supine position with the uterus displaced manually to left side if patient is >20 weeks POG in order to prevent aortocaval compression

Advanced life support

- Inhalation – with a cuffed endotracheal tube
- Vasopressor- epinephrine should be administered to patients with cardiac arrest in a dose of 1mg every 3-5 mins during CPR
- Defibrillation

Mendelson syndrome

- Gastric contents that are highly irritant may be inhaled during induction of anasthesia
- Chemical pnemonitis is more likely following aspiration of gastric contents
- Factors that increase risk-
  - Emergency surgical procedures
  - Inadequate depth of anaethesia
  - Obesity
  - Concomittant opioid administration
  - Impaired concsiousness
  - Lithotomy position
  - Difficult airway or intubation
  - Gastro intestinal reflux ,Hiatal hernia

Clinical features and prevention

- May appear between 2-5 hrs after anaesthesia
- Cyanosis
- Tachycardia
- Dyspnea
- Wheeze
- Crepitant rales
- Decreased arterial oxygen tension

Prevention

Solid foods should be avoided in labouring patients and those at additional risk factors including diabetes, morbid obesity and a difficult airway

Antacids decrease the risk of aspiration by increasing the pH of gastric contents

- Treatment
- Patient should be placed in trendelenberg position and oropharynx suctioned
- If signs of hypoxia present- patient should be intubated
- Bronchoscopy or pulmonary lavage should be done to remove particulate matter from the lungs
- Mechanical ventilation may be required depending on the patients condition


Thank
you