Shock in Obstetrics

Definition of Shock:

Inadequate perfusion (blood flow) leading to inadequate oxygen delivery to tissues.

What is needed to maintain perfusion?

- Pump ---- Heart
- Pipes ----- Blood Vessels
- Fluid ----- Blood

Types of shock:

Hypovolemic Shock
Cardiogenic Shock
Neurogenic and Anaphylactic Shock
Septic Shock

Three broad mechanisms of shock are recognized:

- **Hypovolemic** — fall in intravascular volume
- **Cardiogenic** — fall in cardiac output
- **Distributive, most often due to sepsis** — fall in systemic vascular resistance

In Obstetric cases shock is most commonly due to either hemorrhage or sepsis.

Causes:

- 1-Concealed haemorrhage: any bleeding as in episiotomy, vaginal hematoma, rupture uterus without obvious external bleeding leads to obstetric shock.
- 2-Uterine inversion.
- 3-Amniotic fluid embolism
- 4-Septic shock

Obstetric shock:

- It does not differ from surgical shock; it results from depression of many functions.
- Inadequate perfusion, oxygen depletion, accumulation of metabolites.
- Hypotension without significant external bleeding may develop in an obstetric patient

Causes of Obstetric shock:

- **Bleeding in early pregnancy.**
- **Antepartum hemorrhage.**
- **Postpartum hemorrhage.**
- **Fluid loss e.g Hyperemesis gravidarum, Diarrhea, keto-acidosis.**
- **Plasma loss e.g Severe burns.**
- **Supine hypotension syndrome.**
- **Splanchnic shock: sudden drop in intrauterine pressure e.g Hydramnios.**

The most common form of hypovolemic shock in obstetrics is hemorrhagic shock due to massive obstetric hemorrhage

Stages of Shock:

-Compensated
-Uncompensated
-Irreversible

-Compensated Shock:

Defense mechanisms are successful in maintaining perfusion
Presentation:
Tachycardia
Decreased skin perfusion
Altered mental status

-Uncompensated Shock

Defense mechanisms begin to fail
Presentation
Hypotension
Marked increase in heart rate
Rapid, thready pulse
Agitation, restlessness, confusion

-Irreversible Shock

Complete failure of compensatory mechanisms.
Marked loss of tissue perfusion causes cell damage and death even in presence of resuscitation.

Shock:

Signs and Symptoms:

- Hypotension
- Rapid weak pulse
- Pallor
- Sweating
- Cold extremities
- Oliguria or anuria
- confusion

Initial Treatment in Shock:

- Secure, maintain airway
- Apply high concentration oxygen
- Assist ventilations as needed
- Place patient in the Trendelenburg position
- Control obvious bleeding
- Prevent loss of body heat

- Restoration of Circulation
  Volume – Fluid Choices

Insert at least two large bore IV cannulas

- Crystalloids for initial resuscitation
- Rapidly infuse 5% dextrose in lactated Ringer's solution while blood products are obtained.
- Colloids/ to replace blood loss.

Complications of Hypovolemic shock:

- **1) Acute renal failure.**
- **2) Pituitary necrosis (Sheehan‟s syndrome).**
- **3) Disseminated intravascular coagulation**

Neurogenic Shock:

Abnormal vessel tone---Due to trauma and tissue damage as in painful conditions.

Causes of neurogenic shock:

- Disturbed ectopic pregnancy.
- Concealed accidental hemorrhage.
- Manual removal of placenta without anaesthesia.
- Difficult forceps or breech extraction.
- Rapid evacuation of uterus…. Polyhydramninos.
- Uterine inversion.

SEPTIC SHOCK:

- Septic shock refers to a constellation of infection-mediated clinical findings, with impaired vascular integrity, resulting in inadequate tissue oxygenation and circulatory failure.
- Cellular hypoxia, organ dysfunction, and death ensue if the course of this process is left unaltered.
- Sepsis remains an important cause of maternal mortality in obstetrics, along with thromboembolism, hemorrhage, and hypertension.
- It results from body's response to bacteria in bloodstream.
- Vessels dilate, become "leaky".

PREGNANCY AND SEPTIC SHOCK — Pregnancy is traditionally considered an immune compromised state.

- Incidence — The incidence of bacteremia is approximately 8 to 10 percent in obstetric patients with clinical evidence of local infection. These patients rarely progress to more significant complications, such as septic shock.

Etiology —

- Post-cesarean delivery endometritis
- Endometritis following vaginal delivery
- Urinary tract infections
- Septic abortion
- Intra amniotic infection
- Necrotizing fasciitis
- Toxic shock syndrome

Predisposing factors:

- Prolonged premature rupture of membranes,
- Cerclage in the presence of ruptured membranes,
- Retained products of conception,
- Pregnancy with a retained intrauterine contraceptive device,
- Instrumentation of the genitourinary tract.
- **Microbiology** — The principal etiologic agents of septic shock are **endotoxin producing aerobic Gram-negative bacilli, Gram-positive bacteria** and **mixed** or **fungal infections**. Anaerobic organisms (eg, Bacteroides species, Fusobacterium, Peptostreptococci, Clostridium) are usually involved in mixed infections.
- Prolonged hospitalization and use of broad spectrum antibiotics increase the risk of infection with resistant gram negative organisms and Pseudomonas sp.
- Anaerobes are part of the normal genitourinary and gastrointestinal flora, but may become pathogens when the normal mechanisms limiting their growth are altered.
- Antibiotics, decreased local vascular supply, foreign body material, and tissue trauma all favor anaerobic infection

CLINICAL MANIFESTATIONS — The severity of the clinical presentation of sepsis is determined by the vigor of the host inflammatory response, rather than the virulence of the inciting infection.

Early symptoms — Initial symptoms of sepsis may include malaise, nausea, vomiting, and, occasionally, profuse diarrhea.

- Bacteremia is typically manifested by:
- shaking chills,
- a sudden rise in temperature,
- tachycardia,
- and warm extremities.
- Reductions in cerebral blood flow may cause abrupt alterations in mental status.
- Tachypnea or dyspnea result from a direct effect of endotoxin on the respiratory center and may immediately precede the clinical development of acute respiratory distress syndrome (ARDS).

Early in the course of shock:

- **blood pressure may actually be normal due to peripheral vasoconstriction;**
- **perfusion is disproportionately diverted from the renal and splanchnic circulations to maintain central blood pressure.**
- **The diagnosis of septic shock is often overlooked before hypotension occurs, although the woman may appear critically ill.**

Late symptoms — Cold extremities, oliguria, and peripheral cyanosis are late manifestations in untreated and poorly responding cases.
marked reductions in cardiac output and systemic vascular resistance.
Overt evidence of prolonged cellular hypoxia and dysfunction include profound metabolic acidosis, electrolyte imbalances,
and disseminated intravascular coagulation (DIC).
If these symptoms are left unabated, rapid progression to irreversible shock is the rule.

DIAGNOSIS —
A careful physical examination and selected imaging studies

- The microbiological evaluation should include specimens from blood (at least two sets of blood cultures), urine, sputum, wound (in post-operative patients), and endometrium.
- Both ultrasound and computed tomography (CT) imaging are helpful
- **Laboratory findings —**
- The white blood cell count increases.
- A transient increase in blood glucose concentration due to catecholamine release and tissue underutilization is replaced by hypoglycemia when a reduction in gluconeogenesis subsequently occurs from hepatic dysfunction.
- Decreased platelet count, decreased fibrinogen concentration, elevated fibrin split products, and an elevated thrombin time.
- Respiratory alkalosis from tachypnea, but metabolic acidosis develops as the lactic acid concentration increases from tissue hypoxia.

Management:

- In pregnant women, priorities should first be directed toward maternal well-being, in spite of potential deleterious effects on the fetus. Improvements in the maternal status should have positive effects on the fetal condition since fetal compromise primarily results from maternal cardiovascular decompensation.
- Volume expansion —
- Vasoactive drug therapy —Dopamine is commonly used.
- Oxygenation —
- Antimicrobial therapy —

Empiric therapy in the septic patient should cover a wide variety of both aerobic and anaerobic Gram-negative and Gram-positive bacteria. A common antibiotic regimen is ampicillin (2 grams Q 4 hours), gentamicin (1.5 mg/kg Q 8 hours for patients with normal renal function), or

clindamycin (900 mg Q 8 hours) and metronidazole (15 mg/kg initially then 7.5 mg/kg Q 6 to 8 hours).

- Surgery —

COMPLICATIONS — Acute respiratory distress syndrome occurs as part of septic shock.
PROGNOSIS — Septic shock is a morbid event with high lethality

Cardiogenic Shock:

Pump failure/malfunction
(decreased contractility)

Causes of cardiogenic shock:

- Coronary spasm.
- Cardiomyopathy.
- Pulmonary embolism.
- Amniotic fluid embolism.
- Mendelson's syndrome.

Cardiogenic Shock
Symptoms:

- Tachycardia
- Tachypnea
- Respiratory distress
- Mental status change
- Cold extremities
- Poor perfusion
- Signs of dehydration

Amniotic fluid embolism:

- This condition occurs when amniotic fluid enters the maternal circulation.
- It causes cardio respiratory compromise as well as coagulation defect which is often severe.

Incidence and etiology:

- 1 in 30000 pregnancies.
- Associated with rupture membranes
  Rapid labor
  Vaginal delivery and c/s
- **Mechanism**: Access of amniotic fluid at higher pressure than usual into the maternal circulation through a defect somewhere near the placental site.

- **It is unpredictable and has catastrophic consequences: acute cardiopulmonary embarrassment and coagulation failure.**

Symptoms:

- -Sudden onset of severe chest discomfort.
- -Difficult breathing
- -Pallor
- -Cyanosis
- -Cardiovascular collapse

Signs:

- Venous congestion with raised JVP.
- Output failure with tachycardia, hypotension, and peripheral vasoconstriction.
- Haemorrhage, coagulation failure, petechial skin haemorrhage.
- Bleeding at puncture site, vaginal bleeding.
- Coagulopathic signs may be the presenting features without other symptoms.

Investigations:

- -No time for investigations
- -30 % will die in the first hr.
- -Suspicion------- when cardio respiratory collapse occurs during labor or soon after delivery.
- -Diagnosis only confirmed at postmortem, by finding pulmonary vasculature packed with amniotic debris and trophoblast or aspirating blood from the pulmonary artery and examine for trophoblastic tissue.
- -Coagulation profile requested.

Differential diagnosis:

- Thromboembolism.

Management:

- -Artificial ventilation
- -Cardio pulmonary resuscitation
- -Circulatory support
- -I.V dopamine, steroids may be useful
- -Correct acidosis
- -Treat coagulopathy
- -If the patient survive taken to the intensive care, anticoagulant, antifibrinolytics.
- -Fetus is unlikely to survive.
- -After stabilizing the maternal condition vaginal delivery is preferable.

Prognosis:

- Maternal mortality 90%
- Prevention by avoiding excessive uterine contraction with oxytocin