OBSTETRIC SHOCK AND ITS MANAGEMENT

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ABSTRACT
Shock is a state of circulatory impairment characterized by defective tissue perfusion resulting in abnormal cellular function and metabolism & inadequate tissue perfusion to meet tissue demands.[1] Parturition is a natural process. In majority of the cases it happens without any complications. In obstetrics the standard rule is non interference unless called for. Interfering with a normal labor unnecessarily is a starting point for many problems. However a normal labor can turn into abnormality at any point of time suddenly with no warning. Prompt recognition and management can improve maternal and fetal outcome in obstetric shock.

KEYWORDS: Shock, Parturition, Obstetric shock.

INTRODUCTION
Though any type of shock can occur during pregnancy but obstetric shock mainly refers to a shock which specifically occurs due to obstetrical condition of the patient. Among various types of shock -hypovolemic, septic and neurogenic shock are related to obstetrics. Hypovolemic shock mainly occurs due to obstetrical haemorrhage which is a potential cause for maternal deaths. Hence these three types of shock and their management are explained here.
Obstetric shock is one such problem which needs to be treated aggressively if patient’s life is to be saved. An effort is made herein to delineate all the conditions in obstetrics where shock is a possibility and treatment thereof.

**GENERAL FEATURES OF SHOCK**

- Low Blood Pressure.
  - Systolic BP is usually below 90 mmHg.
- Respiration is rapid and shallow.
- Rapid weak pulse- Tachycardia (>100/min).
- Restlessness, confusion, unresponsiveness, irritability
- Pale, cold, clammy or sweaty skin.
- Drowsiness, coma.
- Oliguria.

**PREDISPOSING FACTORS**

- Severe anemia and ill health.
- Toxaemia of pregnancy.
- Haemorrhage related to pregnancy.
- Prolonged labour, starvation, dehydration, acidosis.
- Retained placenta for a long time.
- Prolonged rupture of membranes.

1. **HYPOVOLEMIC SHOCK**

- Haemorrhagic shock- Associated with postpartum or postabortal haemorrhage, ectopic pregnancy, placenta praevia, abruptio placentae, rupture of uterus, Hydatidiform mole, with difficult forceps and obstetric surgery.
- Fluid loss shock- associated with vomiting, Hyperemesis gravidarum, diarrhoea, diuresis or too rapid removal of amniotic fluid, Prolonged labor (dehydration, starvation, acidosis).
- Supine hypotensive syndrome- occurs due to compression of IVC by gravid uterus. Shock occurs in 10 % patients in supine position.
- Shock associated with DIC- intrauterine dead fetus syndrome and amniotic fluid embolism, pre eclampsia.
OBSTETRIC HEMORRHAGE CAUSES

<table>
<thead>
<tr>
<th>Abnormal Placentation</th>
<th>Injuries to the Birth Canal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placenta previa</td>
<td>Episiotomy and lacerations</td>
</tr>
<tr>
<td>Placental abruption</td>
<td>Forceps or vacuum delivery</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>Cesarean delivery or hysterectomy</td>
</tr>
<tr>
<td>Hydatidiform mole</td>
<td>Uterine rupture</td>
</tr>
<tr>
<td>Previous postpartum hemorrhage</td>
<td>Coagulation Defects</td>
</tr>
<tr>
<td>Preeclampsia/eclampsia</td>
<td>Placental abruption</td>
</tr>
<tr>
<td>Uterine Atony</td>
<td>Severe preeclampsia</td>
</tr>
<tr>
<td>Obstetrical hemorrhages are responsible for 13.4% of all maternal deaths</td>
<td>Anticoagulant treatment</td>
</tr>
<tr>
<td></td>
<td>Congenital coagulopathies</td>
</tr>
<tr>
<td></td>
<td>Prolonged retention of dead fetus</td>
</tr>
</tbody>
</table>

Classification of haemorrhagic shock based on total blood volume.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>No change</td>
<td>Tachycardia</td>
<td>Moderate Tachycardia</td>
<td>Marked tachycardia</td>
</tr>
<tr>
<td>Respiration</td>
<td>Normal</td>
<td>Tachypnoea</td>
<td>Moderate Tachypnoea</td>
<td>Marked Tachypnoea</td>
</tr>
<tr>
<td>MAP</td>
<td>Normal</td>
<td>Mildly dec</td>
<td>&lt;60 mm of Hg</td>
<td>Decreased</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>Normal</td>
<td>Mildly reduced</td>
<td>Reduced</td>
<td>Markedly Reduced</td>
</tr>
<tr>
<td>SVR</td>
<td>Normal</td>
<td>Increased</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>UOP (ml/hr)</td>
<td>&gt;30</td>
<td>20-30</td>
<td>5-15</td>
<td>Anuric</td>
</tr>
<tr>
<td>Mental status</td>
<td>Normal</td>
<td>Anxious</td>
<td>Confused</td>
<td>Confused</td>
</tr>
</tbody>
</table>

CLINICAL FEATURES

Compensatory phase
- mild vasoconstriction
- normal BP
- Tachycardia
- Diaphoresis
- Warm extremities
- restless and anxious
- Can be easily managed

Reversible phase
- hypotension
- Patient becomes pale
- tachycardia
- cold periphery with sweating due to intense vasoconstriction
- Patient is conscious
- normal UOP

Irreversible phase
- hypotension continues
- Cold - clammy extremities
- ashen grey colored skin
- Low volume pulse
- Oliguria
- mental confusion
- Metabolic acidosis, coagulopathy, thrombocytopenia
EFFECT ON FETUS

• The effect of shock on pregnancy depends on the gestational age of the fetus, the type and severity of the hemorrhage, and the extent of disruption of normal uterine and fetal physiology.
• The survival of fetus depends on adequate uterine perfusion and delivery of oxygen.
• The uterine circulation has no auto regulation i.e. uterine blood flow is related directly to maternal systemic blood pressure, at least until the mother approaches hypovolemic shock.
• Maternal catecholamine causes uteroplacental vasoconstriction and compromised fetal circulation.
• At that point, peripheral vasoconstriction will further compromise uterine perfusion. Once obvious shock develops in the mother, the chances of saving the fetus are about 20%.

2. SEPTIC SHOCK

• Also called as endotoxic shock.
• Associated typically with septic abortion, chorio-amnionitis, pyelonephritis and Puerperal sepsis rarely postpartum endometritis.
• Although significant causes of death in obstetric patients, the incidence of death in obstetric patients with septic shock is lower than that in non obstetric patients with septic shock (3% in obstetric patients vs 10-80% in nonobstetric patients).
• 20% are Gram neagative (strepto/staphylococcus), anearobes and clostridium species. Others are pseudomonas aeruginosa, klebsiella, proteus, bacteroids and aerobactor aerogens

CLINICAL FEATURES

<table>
<thead>
<tr>
<th>Initial phase</th>
<th>Late phase</th>
<th>Irreversible phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Alert</td>
<td>• Intense sphincter constriction at end of capillary bed</td>
<td>• Cold and clammy</td>
</tr>
<tr>
<td>• flushing of face</td>
<td>• pale with profuse sweating</td>
<td>• Ashen grey cyanotic appearance.</td>
</tr>
<tr>
<td>• warm skin</td>
<td>• cold clammy extremities</td>
<td>• Hypotension (severe sepsis)</td>
</tr>
<tr>
<td>• Temp &gt; 38 or &lt; 36 C</td>
<td>• UOP is markedly reduced</td>
<td>organ hypo perfusion and dysfuntion</td>
</tr>
<tr>
<td>• Bounding pulse</td>
<td>• Cold shock.</td>
<td>lactic acidosis oliguria.</td>
</tr>
<tr>
<td>• HR &gt; 90</td>
<td></td>
<td>renal failure, ARDS, cardiac</td>
</tr>
<tr>
<td>• RR &gt; 20</td>
<td></td>
<td>failure MODS</td>
</tr>
<tr>
<td>• WBC &gt; 12,000 or &lt; 4,000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Warm shock.</td>
<td></td>
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</tbody>
</table>
EFFECT ON FETUS

- Fetal compromise under these circumstances is directly attributable to maternal cardiovascular decompensation and the attendant decrease in uteroplacental blood flow.
- Regarding the effect of endotoxin on the fetus, data in experimental animals are less clear.

3. NEUROGENIC SHOCK

- Chemical injury- associated with aspiration of gastrointestinal contents during general anesthesia specially in caesarean section.
- Drug induced- associated with spinal anesthesia.
- Initially normovolemic and becomes hypovolemic in later stage due to pooling and stagnation of blood in microcirculatory unit.
- Does not show expected response with fluid replacement.
- More/less same Basic pathology to hemorrhagic shock except-
  - Compensatory phase is very transient.
  - Reversible phase - pallor is absent and face may be flushed.
  - Temperature remains normal or subnormal.

INVESTIGATIONS

- Full blood count.
- Urea and electrolytes.
- Creatinine, glucose.
- ABG, lactate.
- Coagulation profile.
- Urine R/M.
- Cultures- blood, urine, sputum.

MANAGEMENT

Goals of Treatment

- ABCDE.
- Airway.
• Control work of Breathing.
• Optimize Circulation.
• Assure adequate oxygen Delivery.
• Achieve End points of resuscitation.

**MONITORING**

• **Skin temperature**, visible peripheral veins to assess tissue perfusion.
• **Urine output (UOP)**.
• **Heart rate and ECG along with fetal monitoring**.
• **Respiration rate and SpO2**.
• **Arterial blood pressure**.
• **Mental status, pupil size and reaction**.
• **CVP** in critically ill patient to assess circulating volume and myometrial contractile state.
• **Pulse oxymeter and blood gas analysis**.
• **Measurement of left arterial pressure** by Swan-Ganz Catheter in selected cases.

**MANAGEMENT OF HYPOVOLEMIC SHOCK**[^4]

❖ **Infusion and Transfusion**

Blood transfusion as early as possible.
Crystalloids- NS
Colloids – polygelatin solutions
Dextran
Human albumin solutions.

❖ **Oxygen administration**

Initial phase by face mask at 6-8L/min rate.
Later phases- ventilation by endotracheal intubation.
Oxygen delivery to maintain SpO2 >92%, PaO2 80-100 mm Hg
PaCO2 30-35 mm Hg
pH > 7.35.

❖ **Pharmacologic agents**

Vasopressor drugs- minimal use.
Vasoactive drugs, inotropes and corticosteroids to be used.
Control of hemorrhage- specific surgical and medical treatment according to cause of hemorrhage should be done along with general management of shock.

MANAGEMENT OF SEPTIC SHOCK[5]

- Examination of postpartum endometrium along with blood, urine, sputum to be done if suspected.
- In case of amnionitis - amniocentesis which, in the absence of definitive evidence of an alternative source, must be performed.
- Cervical or high vaginal swab for smear and C/S.
- Ultrasonography pelvis abdomen.
- For women with pyelonephritis, a prompt search for obstruction caused by calculi or by a perinephric or intrarenal phlegmon or abscess. Renal sonography or “one-shot” pyelography may be used to diagnose obstruction and calculi. CT may be helpful to identify a phlegmon or abscess.
- The uterus to be evacuated in case of septic abortion.
- In patients with chorioamnionitis, after initial resuscitation of the mother, a plan must be made and initiated as to the route of delivery. In addition, efforts to intervene on behalf of the fetus by operative delivery of an unstable mother may end in catastrophic results for both the fetal and the maternal patients.
- The obvious exception to this axiom is when the source of sepsis is the fetal compartment in which case delivery becomes a critical part of therapy.
- With obstruction, ureteral catheterization, percutaneous nephrostomy, or flank exploration may be lifesaving.
- Hysterectomy to be done in case of puerperal sepsis if needed.
- Women following cesarean delivery suspicious of having peritonitis should be carefully evaluated for uterine incisional necrosis or bowel perforation. CT abdomenopelvis is helpful in this condition. Then prompt surgical exploration and hysterectomy are usually necessary in incisional necrosis.

MANAGEMENT OF NEUROGENIC SHOCK

- Fluid replacement- amount of fluid infused depends on CVP, UOP and its specific gravity
- Vasoactive drugs and corticosteroids- limited use of vasoactive drugs, phenylephrine can be used. Corticosteroids is used in selected cases
 Correction of acidosis and ventilation

 Elimination and correction of source of neurogenic stimulus - like inversion of uterus - replacement of the uterus either manually, surgically or by hydrostatic pressure.

CONCLUSION

• It is important that caretakers of critically ill pregnant women be cognizant of physiological changes of pregnancy and apply them for guidance of therapeutic interventions.

• Survival and outcomes improve with early perfusion, adequate oxygenation, and identification with appropriate treatment of the cause of shock. Effective resuscitation and management of the mother is the key to optimising fetal survival.

• During pregnancy, many difficult management issues are amplified by concerns about the fetus. Still, optimal therapy for the mother remains the 1st priority.

REFERENCES


