

OBSTETRICAL SHOCK

Prof. M.C.Bansal
MBBS,MS,MICOG,FICOG
Professor OBGY
Ex-Principal & Controller
Jhalawar Medical College &
Hospital
Mahatma Gandhi Medical
College, Jaipur.

SHOCK

- ▣ Shock is a critical condition and a life threatening medical emergency.
- ▣ Shock results from acute , generalised , inadequate perfusion of tissues; below that needed to deliver the oxygen and nutrients for normal function.
- ▣ Prompt recognition and management can improve maternal and fetal outcome in obstetrical shock.

AETIOLOGY OF SHOCK

Major causes of shock include –

- ▣ 1. Hypovoluemic → Hemorrhage(occult /overt) , hyperemesis, diarrhoea, diabetic acidosis, peritonitis, burns.
- ▣ 2. Septic→sepsis, endotoxaemia.
- ▣ 3. Cardiogenic→cardiomyopathies , obstructive structural , obstructive non structural , dysrrhythmias, regurgitant lesions.
- ▣ 4. Distributive→Neurogenic- spinal injury, regional anesthesia,
- ▣ 5. Anaphylaxis.

PATHOPHYSIOLOGY

Untreated shock progresses through three stages.

- ▣ Stage1 Compensated --Fall in BP and cardiac output is compensated by adjustment of homeostatic mechanism, if cause removed -iv fluid therapy → it is reversible.
- ▣ Stage2 Decompensate--Maximal compensatory mechanism are acting but tissue perfusion is reduced. Vital organ(cerebral , renal, myocardial) function reduced.
- ▣ Stage3 Irreversible--Vital organ perfusion badly impaired. Acute tubular necrosis , severe acidosis, decreased myocardial perfusion and contractility → the profound decrease in perfusion leads to cellular death & Organ failure.

DIAGNOSIS

A high index of suspicion and physical signs of inadequate perfusion and oxygenation are the basis of initiating prompt treatment.

Initial management does not rely on knowledge of the underlying cause.

There are no laboratory tests for shock.

Basic investigations should be sent
e.g. Hb, BT, CT, PCV. Blood for grouping and cross matching, FB Sugar, routine urine analysis.

INITIAL MANAGEMENT

- ▣ Shocked pt requires teamwork--Senior anaesthetist , obstetrician , physician and hematologist are to be summoned immediately.
- ▣ Obstetrical units should have established protocols for dealing with shock.
- ▣ Practice “FIRE DRILL”.
- ▣ MOET,ALSO training courses for individuals and team.
- ▣ Active management of shock should start as soon as it is suspected or expected aiming for prompt restoration of tissue perfusion and oxygenation.

INITIAL RX

Resuscitation follows---ABC

- ▣ A → Airway--Patent airway is assured and high pressure oxygen (15 l/ min) using mask/ intra tracheal intubation and anaesthesia machine.
- ▣ B → Breathing--Ventilation checked and supported if needed .
- ▣ C → Circulation--
 - 1 Insert two wide bore cannulas
 - 2 Restore blood volume and reverse hypotension with crystalloids/colloids.
 - 3 Initial request for 4-6 units of blood should be sent. O Rh negative blood may be transfused

RX - CIRCULATION

- ▣ Monitor the response to therapy - Pulse , BP , SPO2 / pulse oxymetry, urine output & its pH .
- ▣ Position of patient - Head down and left lateral tilt to avoid aortocaval compression which may further worsen the hypotension.
- ▣ Vasoactive drugs (inotropes and vasopressors) are considered if the cause of shock is thought to be due to myocardial depression or profound vasodilatation.
- ▣ These drugs have no part in hypovolumic shock.

HEMODYNAMIC CONSIDERATION IN PREGNANCY

- ▣ Pregnancy produces a hyperdynamic , hypervolaemic , maternal circulation.
- ▣ This serves the purpose of saving mother against haemorrhage to some extent.
- ▣ Cardiac output increases by 50% , blood volume by 45% reaching a peak at 32-34 wks.
- ▣ 30% loss of fluid may be tolerated without any tachycardia.
- ▣ Aortocaval compression aggravates the instability seen in haemorrhage.
- ▣ In antenatal period , uteroplacental hypoperfusion may occur before maternal signs are evident . Adversely affects on fetal well-being , can be detected →FHR abnormalities on cardiotocograph.

CAUSES OF HEMORRHAGIC SHOCK

- ▣ Antenatal → Ruptured ectopic pregnancy, Incomplete abortion, MTP, Uterine perforation during evacuation , APH, Uterine rupture, Abdominal wall hematoma, Non obstetrical intra abdominal bleeding.
 - ▣ Intra natal → uterine rupture.
 - ▣ Post natal → PPH(primary, secondary) Atonic , Traumatic, Retained tissue , Thrombosis, Acute uterine inversion .
- Nonhaemorrhagic hypovolaemic shock , Burns
Hyperemesis gravidarum , Ac. Diarrhoea

MANAGEMENT

- ▣ The diagnosis of underlying cause and definitive treatment is initiated once resuscitation is under way.
- ▣ Surgical/ obstetrical--- ectopic pregnancy, abortion, uterine perforation ,APH, uterine rupture. PPH, inversion of uterus.

DEVELOPMENTS IN MANAGEMENT OF SHOCK

➤ A. CELL SALVAGE

- ❑ Auto transfusion with salvaged red cells avoids the hazards of homologous transfusion. Blood is removed from operative site through heparinised suction tubing and a filter collecting reservoir and processed by washing and centrifugation to remove contaminating debris.
- ❑ The resulting RBC have a haematocrit of 55-80 % and can be returned to patient quickly.
- ❑ The risk of amniotic fluid is obviously a concern. Use of separate suction for amniotic fluid and leukocyte depletion filter has been found in removing fetal component from the salvaged blood.

MANAGEMENT

- ❖ Disadvantages of salvaged cell transfusion-
- ▣ 1 Units have capital and maintenance cost.
- ▣ 2 Staff require training and regular CME/workshops to update itself.
- ▣ 3 Technique is of no use in PPH as faecal and urine contamination with blood.

MANAGEMENT

- B.RECOMBINANT ACTIVATED FACTOR VII
 - ▣ rFVIIa promotes clot formation through its action at many stages in clotting cascade. It forms a complex with tissue factor a key initiator in homeostasis, leading to production of small amount of thrombin and activating factor V ,VII and platelet aggregation at the site of injury. Hence aids in conversion of fibrinogen in to fibrin and formation of clot.
 - ▣ C.PELVIC ARTERIAL EMBOLISATION

CARDIOGENIC SHOCK

- ▣ The failure of heart to provide adequate output leads to tissue under perfusion.
- ▣ Back pressure on lungs leads to Pulmonary edema.
- ▣ Pregnancy puts progressive strain on cardiac function as pregnancy progresses , the peak being between 32-34 wks.
- ▣ Pre existing cardiac disease further increases the risk.
- ▣ Cardiac related death are 2nd most common causes of death in pregnancy and commoner than the direct leading cause , thromboembolism.

RX → CARDIODGENIC SHOCK

- ▣ Early diagnosis of cardiac lesion.
- ▣ Surgical correction of operable cardiac lesion , before pregnancy is planned.
- ▣ Medical control of decompensated cardiac lesion by cardiac correction before pregnancy is planned.
- ▣ Avoiding Pregnancy/MTP at 6-8 wks if cardiac condition is not under control.
- ▣ Management of pregnancy in such patients by the expert team of cardiologist and obstetrician .
- ▣ Initial Rx of shock is similar , further Rx depends on cardiac lesion→By the team present in cardiac ICU

ANAPHYLACTIC SHOCK

Definition - A serious allergic reaction that is rapid in onset and may result in death.

Aetiology - Pharmacological agents ,insect stings, foods , latex may trigger
ANAPHYLAXIS

ANAPHYLACTIC SHOCK

Pathophysiology - An exaggerated immunological response to antigen to which an individual has been previously sensitized. It is a type 1 hypersensitivity (IgE mediated) response causing breakdown and degradation of mast cells and basophils releasing mediators (Histamine , Serotonin, Bradikynin , Thromboxane , tryptase and leukotrienes) into plasma . These substances cause increased mucous membranes secretions , increased capillary permeability and leakage , marked vasodilatation and bronchospasm.

ANAPHYLACTIC SHOCK

Symptoms and signs -

1. Cutaneous -- (80%) flushing , pruritis , urticaria , rhinitis , conjunctival erythema, lacrymation
2. Cardiovascular -- cardiovascular collapse , hypotension, vasodilatation, pale , cold clammy skin , nausea , vomiting.
3. Respiratory – airway oedema , stridor , wheezing , dyspnoea , cough , chest/throat tightness , hypoxia – confusion , increased airway resistance.

ANAPHYLACTIC SHOCK

Symptoms and signs -

4. Gastrointestinal -

nausea , vomiting , abdominal pain .

5. C N S -

Hypotension causes collapse with/without unconsciousness , dizziness , incontinence , confusion and throbbing headache .

MANAGEMENT OF ANAPHYLACTIC SHOCK

- ▣ 1. Basic shock management → ABC
- ▣ 2. Circulatory management
- ▣ 3. Primary (Special aspect)
 - Stop administration of suspected substance and call for help.
 - Subcutaneous 1ml injection of diluted Adrenaline (1:1000)
 - Early intra tracheal intubation-airway edema will make it problematic later.
 - Supine/trendelenberg position with raised legs increases venous return.
 - Start vasopressor drugs and monitor BP.
 - Rapid infusion for plasma volume expansion , with crystalloids

MANAGEMENT OF ANAPHYLACTIC SHOCK

▣ 4. Secondary

- Atropine may be given if significant bradycardia.
- If bronchospasm – nebulise /I V Amino/Derriphyllin or Beta 2 agonist such as Salbutamol , Inhaled Ipravent may be particularly useful for treatment of bronchospasm in patients on B-blockers.
- Antihistamines - IV Chlorpheniramine.
- Corticosteroids - Effcorlin in I V drip . Dexamthesone.

Referral to critical care unit.

INVESTIGATIONS IN ANAPHYLACTIC SHOCK

- ▣ Immediate - Elevated serum Tryptase , indicates Mast cell degradation . 3 samples of blood are taken at 1st,2nd,3rd hr following suspected reaction.
- ▣ Late - The aim is to identify causative agent. Refer to immunologist/allergist for investigation.

AMNIOTIC FLUID EMBOLISM

- ▣ Amniotic fluid embolism is a rare , devastating condition .
- ▣ It is responsible for (8%) of the direct maternal deaths .
- ▣ It's incidence is 1 in 80,000 - 120,000 .
- ▣ It is characterized by an abrupt cardiovascular collapse and coagulopathy during labor or in the immediate post partum period.

AMNIOTIC FLUID EMBOLISM

PATHO - PHYSIOLOGY

- ▣ Exact mechanism of AFE not clear.
- ▣ The process is more similar to anaphylactic shock.
- ▣ Amniotic fluid found in the pulmonary circulation produces intense pulmonary vasospasm and pulmonary hypertension.
- ▣ When ventilation perfusion mismatch occurs , profound hypoxia ensues.
- ▣ Hypoxia may account for 50% maternal deaths in 1st hr of its onset.
- ▣ Following initial phase there is a phase of hemodynamic compromise caused by left ventricular failure . Right heart parameters return to normal . This mechanism is yet not clear (animal model studies).

AMNIOTIC FLUID EMBOLISM

CLINICAL FEATURES

- ❑ Delivering woman develops acute dyspnoea , hypotension ,seizures.
- ❑ Tachycardia , tachypnoea .
- ❑ cough - blood tinged frothy sputum .
- ❑ Cyanosis - circum oral and peripheral .
- ❑ Fetal bradycardia as a result of hypoxic insult.
- ❑ Uterine atony - PPH . Dark colored blood which does not clot → DIC .
- ❑ Pulmonary oedema – typical X- Ray changes present.
- ❑ Cardiac arrest.

AMNIOTIC FLUID EMBOLISM MANAGEMENT

- Initial management → ABC
- Circulatory management →
 - ▣ 1. Treat hypotension with vasopressors crystalloids and Colloids I V transfusions .
 - ▣ 2. Women who survive the initial phase require ICU admission and prompt management of DIC and left heart failure.
 - ▣ 3. Coagulopathy is treated with fresh frozen plasma, cryoprecipitate and platelets as directed by coagulation studies .
 - ▣ 4. Activated recombinant factor VIIa has also being used.
 - ▣ 5. Plenty of fresh heparinized blood .
 - ▣ 6. Surgery - Perform emergency caesarean surgery in arrested mother who are un responsive ?

DISTRIBUTIVE SHOCK

- ▣ There is no loss in intra vascular volume or cardiac function.
- ▣ The primary defect is a massive vasodilatation leading to relative hypovolaemia , reduced perfusion pressure.
- ▣ Poor blood flow to tissue → tissue anoxia
→ clinical features of shock .
- ▣ ABC of initial management.

NEUROGENIC SHOCK

SPINAL INJURIES

- ▣ Spinal cord injury may produce hypotension and shock as a result of sympathetic nervous system dysfunction . Loss of sympathetic tone causes wide spread vasodilatation.
- ▣ Initial management requires ABC , fluid resuscitation and vasopressor drugs to counteract vasodilatation .
- ▣ Atropine may be necessary in high lesions as bradycardia may occur due to unopposed vagal activity.

NEUROGENIC SHOCK

ANAESTHESIA

1. Shock may occur during any type of anaesthesia or analgesia for labour or delivery.
2. Shock caused by general anaesthesia is usually due to adverse drug reaction (anaphylactic type).
3. High spinal block ---it occurs when over dose of local anaesthetic drug is administered into epidural or subarachnoid spaces .

Factors include —

- i . Drug dose is reduced in pregnancy.
- ii . High spinal block may follow excessive spread of drug
- iii . Accidental intrathecal injection of LA intended for epidural space. Unrecognised dural puncture, migration of epidural catheter in to intrathecal space.
- iv . Hypotension may be aggravated by incorrect positioning , absence of lateral tilt -- aortocaval compression.

NEUROGENIC SHOCK FEATURES

CLINICAL

- ▣ All regional anesthesia techniques produce → sympathetic and motor blockade.
This only becomes problem when it is high and extensive
- 1. Hypotension – preceded by nausea or not feeling well.
- 2. Bradycardia – unopposed vagal tone due to blockage of cardio acceleratory fibers(T1-T4)
- 3. Difficulty in breathing due to paralysis of intercostal muscles and diaphragm.
- 4. Upper limb neurological signs (C5-T1) tingling of fingers and weakness.

NEUROGENIC SHOCK

MANAGEMENT OF HIGH BLOCK

- ▣ Basic shock management → ABC
- ▣ Support of cardiovascular system by Vasopressors , Inotropes.
- ▣ Intra tracheal intubation and ventilation support with ventilator.
- ▣ Sedatives can be used to reduce the awareness once initial resuscitation is achieved.

NEUROGENIC SHOCK LOCAL ANAESTHETIC TOXICITY

- ▣ It is related to high plasma concentration due to high dose given –I V route , rapid absorption
- ▣ It may occur during subcutaneous infiltration or epidural top up.
- ▣ Intravenous injection of LA while giving regional blocks pudendal , paracervical /episiotomy and caudal .
- ▣ Increased and generous blood supply in pregnancy aids rapid absorption.

NEUROGENIC SHOCK CLINICAL FEATURES LOCAL ANAESTHETIC TOXICITY

- ▣ CNS - light headedness , tinnitus , dizziness , circumoral numbness metallic taste , anxiety , confusion , feeling of impending doom , generalized tonic-clonic seizures leading to loss of consciousness and coma , respiratory depression
- ▣ CVS – tachycardia , hypotension , dysrrhythmia and refractory cardiorespiratory arrest.
- ▣ Bupivacaine exhibit signs of toxicity in obstetrical cases.

NEUROGENIC SHOCK

MANAGEMENT OF LA TOXICITY

- ▣ Basic shock management → ABC
- ▣ Special aspects →
 1. Circulation - Advanced life support with external cardiac massage and defibrillation . Arrhythmias may be resistant to conventional therapy.
 2. Maintain BP – Vasopressors and inotropic drugs
 3. Seizure management – diazepam 5-10 mg I V slowly.
 4. Lipid rescue recent work on animals now seems to be important tool of successful therapy (lipid rescue TM website).
 5. LSCS to salvage baby.
 6. Use of sedatives - to reduce the risk of awareness.

SEPTIC SHOCK

- ▣ It remains a significant cause of maternal death. Mortality Rate due to it , is 3% in obstetric patients.

SEPTIC SHOCK

➤ Nomenclatures -

- 1 Systemic inflammatory response syndrome → (SIRS) is recognized by presence of one or two of the following :-
 - i) temp <36 , or >38 degree centigrade.
 - ii) HR >90 per minute.
 - iii) blood gas $\text{PaCO}_2 < 4.3\text{KPa}$ (32mmHg).
 - iv) WBC $>12000/\text{mm}^3$ or with immature neutrophils.
- 2 Sepsis → SIRS with clinical evidence of infection.

SEPTIC SHOCK

➤ Nomenclature -

- 3 Septic shock → Sepsis with hypotension despite adequate fluid resuscitation.

To diagnose it:-

- (i) Evidence of infection.
- (ii) +ve blood culture
- (iii) refractory hypotension , patient requiring vasopressors / inotropic drugs.

- 4 Sepsis with multi organ failure (MODS) → Hypotension , hypoxia , oliguria metabolic acidosis , thrombocytopenia , DIC , depressed level of consciousness

SEPTIC SHOCK PATHOPHYSIOLOGY OF SEPSIS

- ▣ 1. Causative micro organism - E.coli, Streptococcus type A&B, Klebsiella species, staphylococcus aureus , these bacteria induce an exaggerated inflammatory response.
- ▣ 2. Cellwall of these bacteria secrete -lipid A moiety of lipopolysacharide(Gram-ve)while Lypoteicholic acid and super antigen Cytotoxins leading to massive production of cytokinins.
- ▣ 3.Inflammatory cytokinins - activate tissue factor – Peripheral triggering of coagulation - thrombin production - cleaving of fibrinogen in to fibrin

SEPTIC SHOCK PATHOPHYSIOLOGY OF SEPSIS

4. Cytokinins – disturb body modulators of coagulation /inflammation -- protien C & S , Anti thrombin III and tissue factor inhibitor – thus worsen Coagulopathy by decreasing fibrinolysis.
5. Imbalance between Inflammation , Coagulation & Fibrinolysis → Massive wide spread intravascular micro thrombi formation.
6. Massive production of cytokinins , Protiens C & S Interleukins → decreased peripheral resistance → vasodilatation → hypotension → hypovolaemia → decreased Pco2 → decreased tissue perfusion → increased cell wall permeability → transfer of fluid intravascular & intracellular to extracellular compartment → tissue edema → generalized tissue anoxia .

SEPTIC SHOCK PATHOPHYSIOLOGY OF SEPSIS

7. Decreased myocardial , renal , cerebral pulmonary and liver perfusion occurs.
 8. Various cytokinins , nitric oxide , B receptor down regulation , prostacyclins, endothelin -- massive vasodilatation micro thrombi , decreased oxygenation , anoxia - lipid acidosis .
 9. Decreased placental perfusion -- fetal anoxia -- fetal death in utero.
 10. Pulmonary edema → ARDs
 11. Decreased renal perfusion → acute tubular necrosis → oliguria → renal failure .
 12. Cerebral dysfunction → decreased level of consciousness → coma.
 13. DIC → MODS → Death
- .

PREDISPOSING FACTORS IN OBSTETRICS TO SEPTIC SHOCK

- ▣ Post LSCS Endometritis(15-85%)
- ▣ PROM
- ▣ Infected RPOC(1-2%)
- ▣ Post vaginal delivery endometritis (1-4%)
- ▣ Chorioamnionitis
- ▣ Water birth delivery - due to faecal contamination.
- ▣ Pyelonephritis , pneumonia , appendicitis.
- ▣ Toxic shock syndrome <1%
- ▣ Septic abortion →RPOC , Uterine perforation → peritonitis.
- ▣ Pregnancy with retained IUCD.
- ▣ Cx cerclage in PROM cases.
- ▣ Intra amniotic infection.

SEPTIC SHOCK - SYMPTOMS

- ▣ Abdominal pain.
- ▣ Vomiting.
- ▣ Diarrhea.
- ▣ Fever — later on hypothermia

SEPTIC SHOCK - SIGNS OF SEPSIS

- ▣ Tachycardia
- ▣ Tachypnoea
- ▣ Pallor
- ▣ Temperature $>38/ <36$ degree centigrade
- ▣ Hypertension --later Hypotension
- ▣ Cold peripheries , Clamminess
- ▣ Peripheral shut down
- ▣ Systemic inflammation
- ▣ Organ Hypoperfusion , Confusion , Oliguria , Bleeding diathesis , Altered mental state

SEPTIC SHOCK LABORATORY INVESTIGATIONS

- ▣ Abnormal TLC , DLC
- ▣ Low platelet , Coagulopathy – Low Fibrinogen
Fibrinogen degradation products , d-Dimer
, abnormal BT, CT, PT, Clot retraction
, ATPT, INR
- ▣ Raised blood urea , Serum creatinine
- ▣ Abnormal liver function tests

SEPTIC SHOCK MANAGEMENT

- ▣ General--It includes initial management of shock and circulatory management which requires rapid blood volume expansion to correct the absolute and relative hypovolaemia and maintain end organ perfusion.
- ▣ Improvement in maternal haemodynamic stability has direct effect on fetal viability.
- ▣ LSCS for fetal distress in unstable mother will drive last nail in her coffin.
- ▣ If fetal component is source of sepsis , then delivery becomes the essential part of active management.

SEPTIC SHOCK

SPECIAL

ASPECTS MANAGEMENT

- ▣ Quickly transfer to tertiary medical institution.
- ▣ Direct arterial and central venous monitoring.
- ▣ Take samples for culture - blood ,wound , higher swab from vagina and uterus , amniotic fluid , peritoneum , pouch of Douglas .
- ▣ Intra venous broad spectrum antibiotics against gram +ve & gram -ve and anaerobes.
- ▣ Removal of infective tissue P: evacuation of uterus , colpotomy , laparotomy and if required caesarean hysterectomy.
- ▣ Goal related therapy .

ADVANCES IN SEPSIS MANAGEMENT

- ▣ Early goal – directed therapy - modifying the initial Rx to achieve mean arterial pressure >65 mmHg , urine out put >0.5 ml/Kg/hr , CVP 8-12 mm Hg and normal mixed venous oxygen saturation . An effort to reduce end organ damage and tissue death . It improves outcome in septic patients.
- ▣ Insulin therapy - aggressive control of blood sugar has been demonstrated to improve outcome in septic patients.
- ▣ Activated protein C (APC) - Patient with sepsis has decreased APC levels. Its administration decreases mortality and reduces organ dysfunction.

ADVANCES IN SEPSIS MANAGEMENT

- ▣ Corticosteroid therapy ?-- In un selected septic paient it may worsen outcome because of secondary infection.
- ▣ In critically ill patient there may be relative adrenal insufficiency. In septic shock /the affected adrenals may not respond to increased demand of adrenocorticosteroids. Studies on Cortisone therapy in septic shock , have different results. Its beneficial effects in obstetrical sepsis is unknown.

Key Notes

- 1.Shock results from acute , generalized , inadequate perfusion of the tissue.
- 2.Substandard care is still common in its management → patients death.
- 3.Sepsis/ haemorrhage are common in obstetrics.
- 4.Signs of hypovolaemia develop very late because of physiological changes in pregnancy.
- 5.Teamwork is required for successful treatment.
- 6.Obstetrical units → Fire drills regularly.
- 7.Resuscitation to maintain tissue perfusion by ABC should be initiated as soon as shock is diagnosed.
- 8.Management of underlying cause is secondary task.
- 9.All therapy Is directed at optimising maternal condition and fetal wellbeing.