

Is this Septicemic Shock? What do I do?



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Reaching The Unreached FOGSI 2010 INITIATIVE

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- 2nd Vice President elect of FOGSI (2011)
- Ex-Chairman, Rural Obstetrics Committee of FOGSI (2004-2008)
- Executive committee member-ISOPARB
- Managing committee member-IAGE
- Steering committee member-Asia Safe Abortion Partnership
- Past President, Solapur OBGY Society (2001)
- Prof. & HOD, Dept. of OBGY, Gandhi Natha H. Medical College
- Visited many countries like USA, UK, Thailand, Srilanka, Nepal, Pakistan, Bangla Desh, Singapore, Malaysia, South Africa, China , Portugal & all over India to deliver lectures on various topics in OBGY
- Authored a book “Hypertensive Disorders in Pregnancy” and contributed more than 10 chapters in various books
- Active Rotarian

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Significance...

- Obstetric sepsis accounts for 7% MMR
- It is one of the leading cause of maternal morbidity
- Morbidity is 10-15 times that of MMR
- Sepsis is among the leading causes of preventable maternal death not only in developing but also in developed countries
- It is usually reported as the fourth leading cause of maternal death after **Haemorrhage, Eclampsia & Unsafe Abortion**



Predisposing Factors

- Chorioamnionitis
- Postpartum endometritis
- Urinary tract infections
- Pylonephritis
- Septic abortion
- Postoperative necrotizing fasciitis
- Toxic shock syndrome



Physiological Adaptations to Pregnancy leading to more Morbidity

- Elevation of diaphragm
- Delayed gastric emptying
- Ureteral dilatation
- Increased susceptibility to endotoxin
- Immunocompromised state



Mortality from Septic Shock

- Younger age group
- Increased pelvic vascularity
- Transient nature of bacteremia
- Type of organisms involved
- Primary site pelvis is more amenable to surgical & medical intervention
- Lack of associated medical diseases

Incidence of death from sepsis in obstetric patient is 0-3% as compared to 10-81% in non-obstetric patients



Septic Shock...

- It is constellation of clinical findings marked by alteration of ability of the host to maintain vascular integrity and homeostasis, resulting in inadequate tissue oxygenation and circulatory failure, which results from the Systemic Inflammatory Response to an infectious insult
- In its course, cellular hypoxia, multiple organ dysfunction (MODS) and death ensue



Systemic Inflammatory Response Syndrome (SIRS)

Characterized by 2 or more of the followings

- Body temperature $< 36^{\circ}\text{C}$ or $> 38^{\circ}\text{C}$
- Pulse > 90
- RR > 20 or $\text{PaCO}_2 < 32$ mm of Hg
- WBC $< 4000 \mu\text{l}$ or $> 12,000 \mu\text{l}$ or more than 10% immature forms in differential count

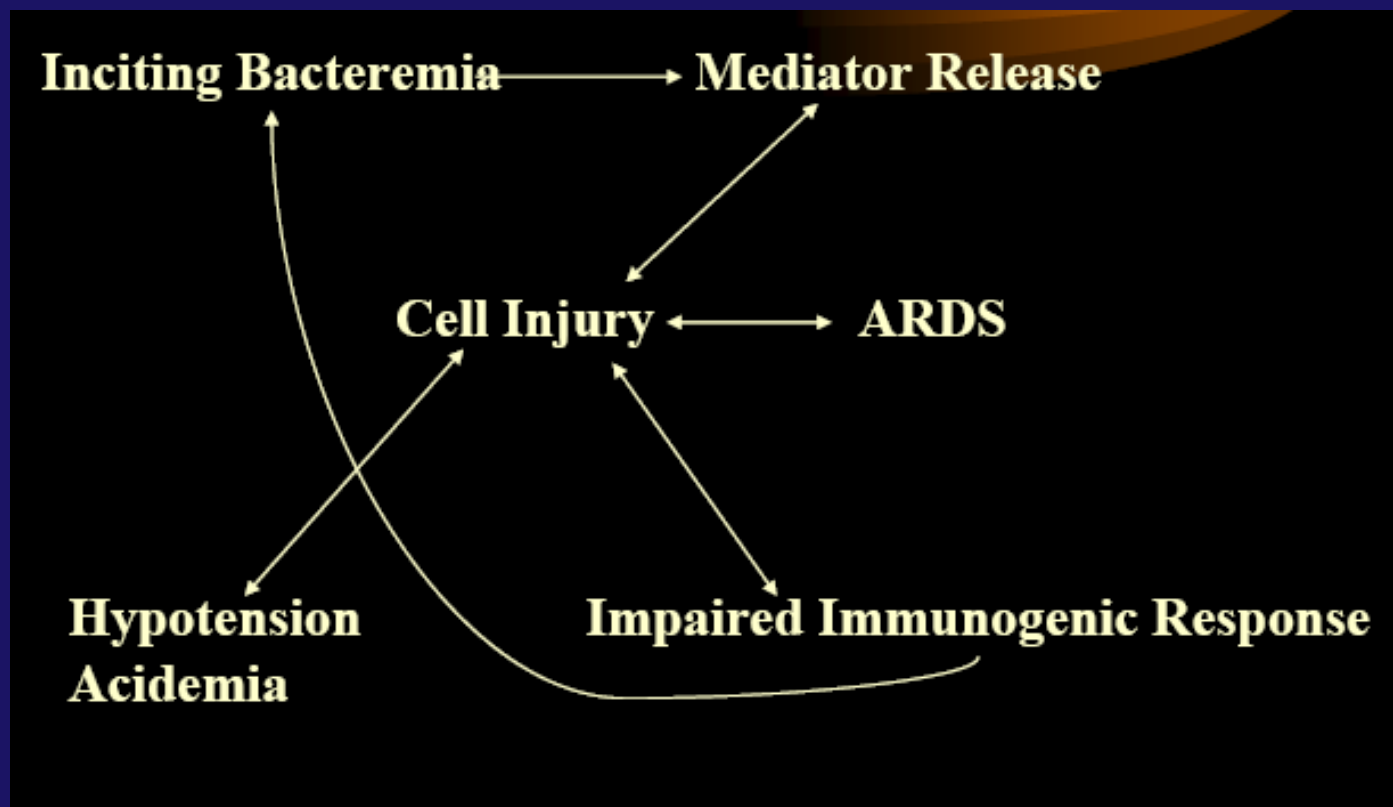
When SIRS is associated with organ dysfunction, hypoperfusion or hypotension it is called Severe Sepsis & If hypoperfusion persists despite adequate fluid resuscitation, it is Septic Shock



Systemic Inflammatory Response Syndrome (SIRS)

- Multiple organ dysfunction syndrome (MODS) is terminal phase of this spectrum represented by the progressive physiological deterioration of interdependent organ systems such as homeostasis cannot be maintained without active intervention
- Commonly affected organs are pulmonary system leading to Acute respiratory distress syndrome (ARDS) & kidneys leading to Acute renal failure (ARF)

Septic Shock - Cascade



Presenting Features of Septic Shock...

- Early (Warm) Shock: Hyperdynamic circulation & decreased systemic vascular resistance
- Late (Cold) Shock: Abnormal perfusion & oxygenation secondary to peripheral vasoconstriction & myocardial dysfunction
- Secondary (Irreversible) Shock: Terminal condition with MODS



Presenting Features of Septic Shock...

- Early (Warm) Shock: Altered mental status, warm skin & flushing, tachypnea, tachycardia, hypotension
- Late (Cold) Shock: Cool clammy skin, oliguria, cyanosis, ARDS
- Secondary (Irreversible) Shock: Obtundation, anuria, hypoglycemia, DIC, Myocardial failure

Laboratory Features of Septic Shock...

- Early (Warm) Shock:
WBC count depressed first followed by marked leucocytosis,
Transient increase in BSL due to gluconeogenesis secondary to catecholamine release followed by hypoglycemia secondary to hepatic dysfunction
Early evidence of DIC: Decreased platelet count, decreased fibrinogen, elevated FDP, elevated Thrombin time,
Arterial blood gases may show transient respiratory alkalosis



Laboratory Features of Septic Shock...

- Late features:
 - Profound metabolic acidosis
 - Electrolyte disturbances
 - DIC
- Cardiac changes:
 - Initially tachycardia, increased Cardiac output and Cardiac index
 - Both ventricles dilate & Ejection fraction decrease
 - Apart from alteration in coronary flow or myocardial oxygenation, circulating Myocardial Depressant Factor is responsible for cardiac dysfunction

Treatment of Septic Shock

Therapeutic Goals

- Oxygen Delivery
- Organ perfusion
- Neutralizing effects of inflammatory mediators



Treatment of Septic Shock

Interventions towards these goals

- Improvement in functional circulating intravascular volume
- Establishment & maintenance of an adequate airway to facilitate management of respiratory failure
- Assurance of adequate tissue perfusion & oxygenation
- Initiation of diagnostic evaluations to determine the septic focus
- Institution of empiric antimicrobial therapy



Volume Expansion

- Needed because of profound vasodilatation, increased capillary permeability & extravasation of fluid into extravascular space
- BP, Heart rate, urine output & hematocrit are used to assess the adequacy, but CVP monitoring or if available PA catheter would be optimum
- Common endpoint should be PCWP 14-16 mm Hg and care taken to avoid pulmonary edema
- Crystalloids or colloids ???



Vasoactive Drugs

- Needed as fluid resuscitation many times is not adequate to restore cardiovascular performance
- Commonly used drug is dopamine hydrochloride
- It has dose dependent effects like low doses increase mesenteric and renal flow while increasing doses predominant effect is on myocardial contractibility
- Other inotropic agents are Dobutamine, norepinephrine or epinephrine specially in dopamine resistant shock but it causes increased myometrial work & O₂ requirement

Oxygenation

- Mitochondrial and cellular dysfunction causes decreased ability to extract the oxygen that is delivered & reduced peripheral tissue utilization of oxygen due to microvascular shunting
- Additionally hypophosphatemia, alkalosis and multiple transfusions cause shift of oxyhemoglobin dissociation curve to left
- So excessive oxygenation and watch on lactic acid concentration is required

Antimicrobial Therapy

- Aggressive investigation into underlying etiology of sepsis & empiric antimicrobial therapy started
- Microbiological evaluation of specimens from blood, urine, sputum and wound
- Empiric therapy should cover aerobic and anaerobic gram-positive and gram-negative bacteria like parenteral Ampicillin 2 g/6 hrs, Aminoglycoside 500 mg/ 8 hrs with watch on renal function & Metronidazole 500 mg/ 8 hrs

Managing ARDS

- Diagnosis is on the basis of progressive hypoxemia, normal PCWP, diffuse infiltrate on X-ray chest & decreased pulmonary compliance
- Cornerstone of treatment involves intubation and ventilatory support to maintain adequate gas exchange at nontoxic levels of inspired oxygen
- Positive end-expiratory pressure is often necessary to achieve this goal

Coagulation System

- Best treatment of DIC is treating underlying cause, exception is when platelets fall below 10,000 per ml when platelet transfusion is required
- Use of heparin is monitored properly and all sources of heparin like subcutaneous injections for DVT, flushing indwelling catheters & total parenteral nutrition solutions

Renal Function

- Renal function is best monitored with indwelling catheter and serial creatinine and BUN levels
- Additionally management of electrolyte imbalances, correction of metabolic acidosis, stabilization of coagulation defects, prophylaxis for DVT with subcutaneous heparin or sequential compression boots is required

Gastrointestinal Tract & Nutrition

- GI tract could be reservoir of infection and source of considerable mortality, so three interrelated areas provision of adequate nutrition, prevention of effect of translocation of bacteria from gut to systemic circulation & stress ulcer prophylaxis need attention
- These are necessary to maintain splanchnic circulation and integrity of GI mucosa
- Specific nutrients like glutamine, arginine, & omega-3 fatty acids may have significant immunomodulatory functions



Controversial Treatment Modalities “Steroids”

- Benefits of stabilization of lysosomal membranes, inhibition of complement-induced inflammatory changes, and inflammatory mediators
- As many studies could not find benefit, their use should be reserved for those patients with documented adrenal insufficiency or in fibroproliferative phase of ARDS to prevent evolving fibrosis

Controversial Treatment Modalities

- PG Synthetase inhibitor: As there is increased production and decreased degradation of PG F₂ in severe sepsis
- Antipopolysachharide immunoglobulin
- Antiendotoxin and anticytokine therapies
- Narcotic anatgonists like Naloxane



Surgical Therapy

- Evacuation of uterus in septic abortion under umbrella of antibiotics
- Expediate delivery in cases of chorioamnionitis in a viable fetus
- Hysterectomy if microabscess formation identified within myometrial tissues
- In Septic pelvic thrombophlebitis if heparin and antibiotics are unsuccessful

Remember...

- During management maternal interests should take precedence even in face of the potential deleterious effects shock or drugs on fetus
- If fetal compartment is a source of infection, therapy includes initiating delivery
- Joint consultation with experts in infectious diseases or critical care medicine

Signs of Critical Illness

Physical

- Signs of sympathetic activation: tachycardia, hypertension, pallor, clamminess and peripheral shutdown
- Signs of systemic inflammation: fever or hypothermia, tachycardia and increased respiratory rate
- Signs of organ hypoperfusion: cold peripheries, hypoxemia, confusion, hypotension and oliguria

Biochemical

- Metabolic acidosis
- High or low white cell count
- Low platelet count
- Raised urea and creatinine concentrations
- Raised C reactive protein concentration





Thank you

"God could not be everywhere and therefore he made mothers"

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