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The Pathophysiology of Sepsis
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Introduction
- Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection (Schröter, 2018).
- Sepsis is a disease that has been known and studied for over 2000 years yet there is still so much of it which is not understood (Ward & Levy, 2017).

Sepsis means a very heterogeneous population, which varies in etiology and severity; therefore, universally applicable diagnostic criteria and treatment algorithms are difficult to be defined (Laszlo, Trayn, Molnar, & Fabian, 2015).

Sepsis is associated with high morbidity and mortality and accounted for $1.3 Billion in healthcare expenditures in 2013 (McCance & Huether, 2019).

Pathophysiology
- Bacterial
  - Gram-negative organism release endotoxin
  - Gram-positive organism release endotoxin
- Release of proinflammatory cytokines
  - Activation of: Complement system, Coagulation system, Renin System, Thromboxane, endothelial, and monocyte/macrophage activity
  - Release of anti-inflammatory cytokines
- Endothelial cell dysfunction: Capillary leak, Microvascular thrombosis, Cell adhering, Tissue hypoxia, Impaired vascular tone
- Free radical damage
- Multiple organ dysfunction: Altered mental status, Protein/FO: < 30% tachyphagia, Urine >0.5mL/kg/hr
- Hypotension with tachycardia
- Thrombocytopenia: <100,000/m L
- Metabolic acidosis; ↑ lactate
- Hyperglycemia
- Tachycardia: HR > 90 bpm

Signs and Symptoms
- Fever >38.3°C (100.4°F)
- Hypotension >60°C (98.8°F)
- Tachypnea >30 breaths
- Progressive deterioration of mental status
- Altered mental status
- Significant oedema of positive fluid balances (>20L/kg/24 hr)
- Hypothermia (36°C ± 0.5°C) in the absence of diabetes
- Leukocytosis: WBC >12,000/mm³
- Leukopenia: WBC < 6000/mm³
- Normal WBC with >15% bands
- CRP >2.0 mg/dl above normal value
- PCT >2.0 mg/dl above normal value
- Urine protein: CRP >90 mg/dl
- MAP >70 mmHg
- SBP decrease >40 mg/dl

Cellular Significance of Pathophysiology
- Host-derived molecules and foreign products of infection converge on molecular mechanisms that cause unbalanced activation of innate immunity. Foreign and endogenous molecules interact with pathogen recognition receptors expressed on or in the immune system. Activation of pathogen recognition receptors culminates in the release of immunomediators that produce the clinical signs and symptoms of sepsis (Ward & Levy, 2017).

Figure 2. Select mechanisms implicated in the pathogenesis of sepsis involving organ and cellular dysfunction (Seymour & Angus, 2018).

Signs of Sepsis
- Svo2 >70%
- Cardiac index <3.5L/min
- Arterial hypoxemia (PaO2/FiO2 < 300 mmHg)
- Acute oliguria (urine output < 0.5 mL/kg/hr for at least 2 hr)
- Creatinine increase > 0.5 mg/dL
- Compensated shock (HR > 15 or PTT > 60 seconds)
- Base
- Thrombocyteplatelet count < 100,000/mm³
- Hyperbilirubinemia
- Intrahepatic cholestasis
- Hyperlactatemia (> 1 mmol/L)
- Decreased capillary refill or swelling

McCance & Huether, 2019)

Cellular Level Pathophysiology

Figure 3. The goal of the Surviving Sepsis Campaign (SSC) is to begin resuscitation and management immediately (Schröter, 2018).

Treatment

Implications for Nursing Care
- Patients in septic shock may have improved outcomes if antibiotics are administered within the first hour of diagnosis (Schröter, 2018).
- Nurses are instrumental to improving outcomes for patients with sepsis of septic shock (Schröter, 2018).
- Delays in treatment directly influence mortality in patients with sepsis (Schröter, 2018).
- Sepsis is one of the most common causes of death among ICU patients worldwide (Schröter, 2018).
- Prompt recognition of sepsis symptoms and treatment initiation is essential to managing septic shock (Schröter, 2018).

Adjunct Therapies
- Immunomodulation strategies
- Extracorporeal removal of cytokines via the addition of devices to hemofiltration or ECMO circuits
- Low-dose glucocorticoids to mitigate the inflammatory process
- Intravenous immunoglobulins to modulate the immune system
- Thiamine supplementation to support ATP generation via the Kreb's cycle
- Vitamin C, vitamin E, selenium, and zinc to modulate the immune system
- Improved microcirculatory flow, and restoration of endothelial integrity
- Conzyme Q10, melatonin, glutathione, and L-arginine to assist in metabolic resuscitation and reduce oxidative stress
- Gastrointestinal mapping, manipulation, and restoration
- Fecal transplantation to restore gut health and function
- Decannulation with cardiac-selective beta-1 adrenergic blockers and alpha 2 adrenergic agonists
- Heparin as an immunomodulator and antithrombotic agent

References (cont.)