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Lauren Tomczak

Otterbein University, tomczak1@otterbein.edu

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The Pathophysiology of Sepsis

Lauren Tomczak, BSN, RN, CCRN, SRNA

Otterbein University, Westerville, Ohio

Introduction

- Sepsis is a life-threatening organ dysfunction caused by simultaneous systemic inflammation and coagulation in response to microbial infection (Schub, 2018).
- Sepsis is a disease that has been known and studied for over 2000 years yet there is still so much of it that 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, Trasy, Molnar, & Fazakas, 2015).
- Sepsis is associated with high morbidity and mortality and accounted for \$23.7 billion in healthcare expenditures in 2013 (McCance & Huether, 2019).

Pathophysiology

- Bacteremia:
 - Gram-negative organism releases endotoxin
 - Gram-positive organism releases exotoxin
- Release of proinflammatory cytokines
- Activation of:
 - Complement system
 - Coagulation system
 - Kinin System
 - Neutrophil, endothelial, and monocyte-macrophage activity
- Release of anti-inflammatory cytokines
- Endothelial cell dysfunction:
 - Capillary leak
 - Microvascular thrombus
 - Cell adhesion
 - Tissue hypoxia
 - Impaired vascular tone
 - Free radical damage
- Multiple organ dysfunction:
 - Altered mental status
 - PaO₂/FiO₂ <300; tachypnea
 - Urine <0.5mL/kg/hr
 - Hypotension with tachycardia
 - Thrombocytopenia; ↑ D-dimer
 - Metabolic acidosis; ↑ lactate
 - Poor capillary refill
- Death

(McCance & Huether, 2019)

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 immune mediators that produce the clinical signs and symptoms of sepsis (Ward & Levy, 2017).
- When the host response induced against a local infection fails to contain it locally, it progresses to sepsis, septic shock, and death (Bhan, Dipankar, Chakraborty, & Sarangi, 2016).
- The mortality rate is 20% of hospitalized patients with sepsis and 60-80% of patients with septic shock (Schub, 2018).
- Restoration of hemodynamics in septic patients does not always prevent or repair organ dysfunction (Ward & Levy, 2017).

Signs and Symptoms

- Fever >38.3°C (100.4°F)
- Hypothermia <36°C (96.8°F)
- Tachycardia: HR >90 bpm
- Tachypnea: RR >30 bpm
- Progressive deterioration of mental status
- Altered mental status
- Significant edema of positive fluid balance (>20mL/kg over 24 hr)
- Hyperglycemia (BG >140 mg/dL in the absence of diabetes)
- Leukocytosis: WBC >12,000/mm³
- Leukopenia: WBC <4000/mm³
- Normal WBC with >10% bands
- CRP >2 SD above normal value
- PCT >2 SD above normal value
- Arterial hypotension (SBP <90 mmHg; MAP <70 mmHg; SBP decrease >40 mmHg)
- 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
- Coagulation abnormalities (INR >1.5 or PTT >60 seconds)
- Illeus
- Thrombocytopenia (platelet count <100,000/mm³)
- Hyperbilirubinemia (plasma total bilirubin >4 mg/dL or 70 mmol/L)
- Hyperlactatemia (>1 mmol/L)
- Decreased capillary refill or mottling

(McCance & Huether, 2019)

Cellular Level Pathophysiology

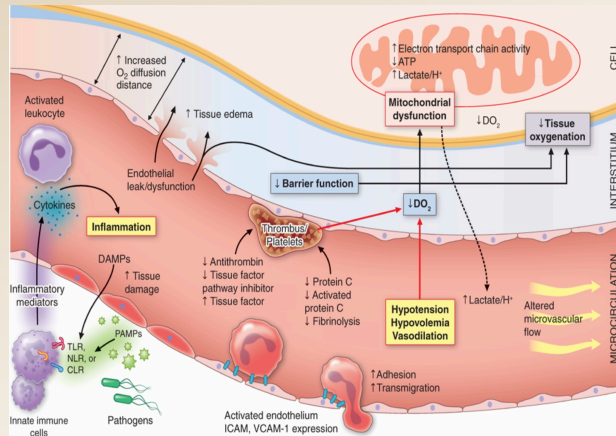


Figure 2. Select mechanisms implicated in the pathogenesis of sepsis-induced organ and cellular dysfunction (Seymour & Angus, 2018).

Treatment

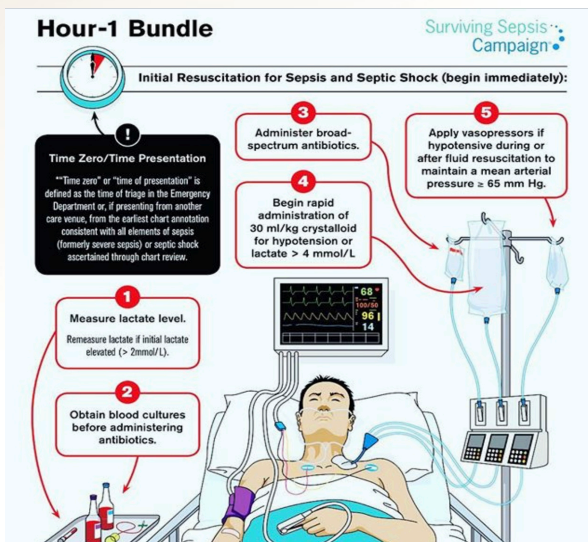


Figure 3. The goal of the Surviving Sepsis Campaign (SSC) is to begin resuscitation and management immediately (Schorr, 2018).

Implications for Nursing Care

- Patients in septic shock have improved outcomes if antibiotics are administered within the first hour of diagnosis (Nagalingam, 2018)
- Nurses are instrumental to improving outcomes for patients with sepsis of septic shock (Schorr, 2018)
- Nursing responsibilities include: collecting blood and urine specimens; assessing the patient for hemodynamic instability; assessing for and managing pain; administering IV fluid resuscitation, antibiotics, corticosteroids, and vasopressor medications; monitoring intake, output, vital signs, laboratory and diagnostic results (Schub, 2018)

Adjunct Therapies

- Immunomodulation strategies
- Extracorporeal removal of cytokines via the addition of devices to hemofiltration or ECMO circuits
- Low-dose glucocorticosteroids to mitigate the inflammatory process
- Intravenous immunoglobulins to neutralize endotoxins
- Thiamine supplementation to support ATP generation via the Krebs cycle
- Vitamin C, vitamin E, selenium, and zinc for mitochondrial protection, improved microcirculatory flow, and restoration of endothelial integrity
- Coenzyme Q10, melatonin, glutamine, and L-carnitine to assist in metabolic resuscitation and reduce oxidative stress
- Gut microbiome mapping, manipulation, and restoration
- Fecal transplantation to restore gut health and function
- Decatecholaminization with cardio-selective beta-1 adrenergic blockers and alpha-2 adrenoreceptor agonists
- Heparin as an immunomodulator and antithrombotic agent

(Chausse, Malekele, & Paruk, 2018)

Conclusions

- In-depth and comprehensive knowledge on the immune cell activities and their correlation with severity of sepsis will help clinicians and scientists design effective immunomodulatory therapeutics for sepsis (Bhan et al., 2016).
- Delays in treatment directly influence mortality in patients with sepsis (Nagalingam, 2018).
- Sepsis is one of the most common causes of death among ICU patients worldwide (Schub, 2018).
- Prompt recognition of sepsis symptoms and treatment initiation are essential to managing septic patients (Schorr, 2018).
- A guiding principle is that septic patients are complex and need detailed initial assessment and ongoing reevaluation of their response to treatment (Schorr, 2018).
- Norepinephrine is considered the vasopressor of choice in septic shock (McCance & Huether, 2019).

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Case Presentation

Prevent sepsis and improve early recognition.



Figure 1. Think sepsis. Act fast. Time matters (Ward & Levy, 2017).