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The Pathophysiology of Sepsis

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Sepsis

Sepsis is still a leading cause of hospital admissions and often leads to death if not identified early and treated properly (Pilcher, Cooper & Bellomo, 2015). In the US there are approximately 200,000 deaths annually due to sepsis (Puskarich, Illich, & Jones, 2014).

It is important for advance practice nurses to recognize risk factors for sepsis. By understanding the pathophysiology of sepsis nurse practitioners will be able to recognize early signs and symptoms of this inflammatory disease process and implement early medical management. Early medical treatment in septic patients is crucial to increase patient survival rates.

The Importance Understanding Sepsis

There is a high mortality rate linked with the diagnosis of sepsis and septic shock, which ranges from 30% to 40% (Wawrzeniak, Loss, Martins Moraes, De La Vega, & Victorino, 2015). By understanding the pathophysiology of systemic inflammatory response syndrome (SIRS), nurse practitioners will be able to recognize early warning signs and provide prompt treatment. Prompt treatment is the key to improving patient outcomes and decreasing patient mortality rates.

Patients who are lucky to survive sepsis have a greater risk for future infections and usually acquire chronic health issues. Survivors may have a decreased quality of life, neurocognitive dysfunction, impaired kidney functioning or cardiovascular disease (Cawcutt & Peters, 2014). It is crucial that healthcare providers understand the clinical features, pathophysiological process, and potential complications of sepsis in order to initiate appropriate treatment for these patients.

Since survivors will most likely have chronic health conditions, many family nurse practitioners will be providing follow up care for these patients.

Pathophysiological Process

Disease Process

The systemic response to severe infections leads to a lowered ability to fight infections, which can result in sepsis or multiorgan failure (Lord, Midwinter, Chen, Belli, Brohi, Kovacs, & ... Lilford, 2014).

Several of the microcirculatory alterations occurring in septic patients including altered glycocalyx, endothelial dysfunction, impaired cellular communication, and alterations in adhesion and production of red blood cells, white blood cells, and platelets (De Backer, Cortes, Donadello, & Vincent, 2014).

Severe injury or infection is associated with the systemic inflammatory response syndrome (SIRS) (Lord, et al., 2014). The normal immune response contains a series of feedback loops intended to restore homeostasis in response to an infection or injury. Raised levels of anti-inflammatory cytokines, persistent inflammation, and immunosuppression can lead to sepsis and possibly multiorgan dysfunction if the body is unable to return to homeostasis (Lord, et al., 2014).

Signs & Symptoms

Sepsis can be difficult to diagnose because the signs and symptoms of sepsis can be subtle and often mimic other disorders (Lopez-Bushneil, Demaray & Jaco, 2014).

To diagnose SIRS a patient must have 2 of the following 4 criteria:

- Hypothermia or hyperthermia.
- Leukocytosis or leukopenia.
- Tachycardia
- Tachypnea

Information retrieved from (Sagy, Al-Qaqaa, & Kim, 2013).

Other possible manifestations of sepsis include:

- Hyperglycemia in patients who do not have diabetes.
- Increased procalcitonin.
- Increased C-reactive protein.
- Altered mental status

Information retrieved from (Cawcutt & Peters, 2014).

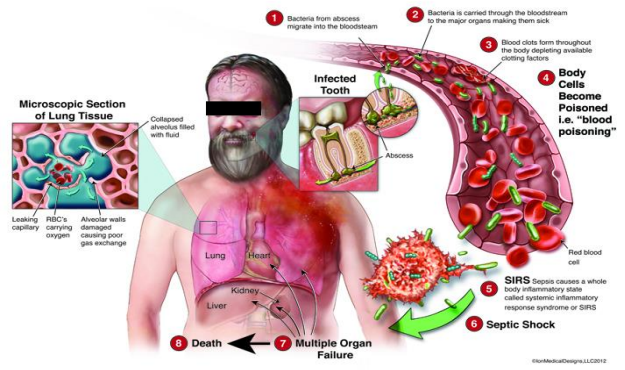
Please refer to ***Table 1***. for a complete list of definitions, signs and symptoms of SIRS, Sepsis, Severe Sepsis & Septic Shock.

Table 1. Definitions of Systemic Inflammatory Response Syndrome (SIRS), Sepsis, Sever Sepsis, and Septic Shock

SIRS	**Meets 2 of the following 4 criteria:** • Heart rate > 90 beats/min • Temperature >104 degrees F or < 96.8 degrees F • White blood cell count >12,000 or < 4,000 or > 10% immature band forms • Respiratory rate > 30 breaths/min or arterial CO2 < 32mm Hg
Sepsis	• SIRS as a result of a documented or suspected infection
Sever Sepsis	**Sepsis Plus evidence of organ dysfunction** • Systolic blood pressure <90mmHg or >40mmHg drop from baseline • Urine output <0.5mL/kg per hour for 2 hours despite fluid resuscitation • Increase in creatinine > 0.5mg/dL • Elevated bilirubin • Arterial hypoxemia • Coagulation abnormalities • Paralytic ileus • Decreased capillary refill
Septic Shock	**Sepsis with hypotension despite fluid resuscitation or hyperlactemia** • Lactate > or equal to 4mmol/L • Vasopressin support needed

The above information was retrieved from (Cawcutt & Peters, 2014).

Figure 1. Image shows how a tooth infection can progress to a systemic infection and even death if left untreated.



The above information was retrieved from www.nutekmedicalcentremumbai.com

Underlying Pathophysiology

- The inflammation response is the body's response to noxious stimuli.
- Neutrophils mediate cellular changes.
- Vasodilation and inflammation occurs.
- The invading pathogenic microorganism stimulates the release of cytokines.
- Cytokines trigger an exaggerated inflammatory response.
- Fluid leaks from the circulation into the interstitial tissue.
- A decrease in intravascular volume results in hypotension.
- Inadequate tissue perfusion leads to cellular hypoxia and lactic acidosis.
- Nitric oxide is produced by the endothelium during hypoxia.
- An exaggerated release of tumor necrosis factor and excessive levels of nitric oxide contribute to systemic hypotension.

Above information retrieved from (Dunkley, & McLeod, 2015)

The complement system normally has three primary responsibilities:

1. Recruitment of inflammatory cells to the site of infection.
2. Covering the bacterial membrane of the pathogen.
3. Destroying the pathogen.

- During sever infection excessive activation of the complement system can lead to inappropriate inflammatory response.
- This can cause hypotension, disseminated intravascular coagulation, or multiple organ dysfunction syndrome.
- The inflammatory response disrupts normal coagulation and can lead to fibrin and platelet plugs.
- Depressed neurological functioning is also often present due to inflammation affecting neurons, glial cells, and endothelial cells which induce breakdown of the blood brain barrier.

Above information retrieved from (Dunkley, & McLeod, 2015)

Altered Compensatory Mechanisms

- Tachypnea is a respiratory compensatory response to lactic acidosis.
- The brainstem is stimulated to expel carbon dioxide in order to try and compensate for metabolic acidosis.
- Reduction in cardiac output and stimulation of baroreceptors in the carotid sinus and aortic arch stimulate the sympathetic nervous response.
- Adrenaline is released from the adrenal glands, which stimulates alpha 1 receptors and leads to vasoconstriction.
- Stimulation of the beta 1 receptors in the in the myocardium, causes an increase in heart rate and contractility.
- The renin-angiotensin-aldosterone system is a compensatory mechanism, which causes retention of sodium and water and leads to vasoconstriction.
- In septic patients these normal compensatory mechanisms go into overdrive, are altered, and have difficulty restoring homeostasis.

Information retrieved from (Dunkley, & McLeod, 2015)

Significance of Pathophysiology

Understanding the pathophysiology of sepsis has led to the development of sepsis protocols, which have proven to reduce mortality and length of hospital stay when appropriate treatment is initiated early (Lopez-Bushneil, Demaray, & Jaco, 2014).

By carefully studying the pathophysiology of sepsis, experts have determined that by the time organ dysfunction is identified, patient prognosis is poor (Lee, 2015). Urgent interventions should be taken for patient's exhibiting signs and symptoms of systemic inflammatory response syndrome to prevent the progression of this disease process.

By understanding the pathophysiology behind sepsis, advanced nurse practitioners will be able to treat symptoms, prevent patient discomfort and potential harm.

Implication for Nursing

Investigating the pathophysiological process of sepsis has allowed clinicians to formulate suggested treatment bundles for improving the outcome for septic patients. Patients who are diagnosed early and treated aggressively will have a greater chance of survival.

Sepsis protocols have been created which provide guidelines for appropriate treatment and management of these patients. The biggest focus of treatment is early fluid resuscitation, appropriate use of vasopressors, and early antibiotic administration (Cawcutt & Peters, 2014). One of the most important steps is to target the source of the infection so appropriate therapy can be initiated (Cawcutt & Peters, 2014).

Nurse practitioners should order blood, wound, urine or sputum cultures depending upon the suspected source of infection. The source of infection should be removed when possible or controlled aggressively in order to prevent mortality.

Conclusion

Advance practice nurses should understand the sepsis continuum. If a infection is not properly identified and controlled, it can progress to sepsis, which can lead to septic shock, multiple organ dysfunction syndrome, multiple organ failure and ultimately death (Dunkley, & McLeod, 2015).

Understanding the pathophysiology of sepsis and recognizing early warning signs of SIRS will allow clinicians to make appropriate treatment interventions. Many patients either ignore or are unable to recognize early warning signs and symptoms of an infection therefore, healthcare providers must be advocates for these patients.

It is important for advanced practice nurses to obtain a detailed patient history and physical. If an infection is suspected, starting appropriate treatment is pertinent. Quick diagnosis and appropriate management can lead to improved outcomes and decreased patient mortality in septic patients.

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