**The Pathophysiology and Treatment of Sepsis**

**Introduction**

Sepsis, according to consensus definition is the response that host has on a microbiological event that is induced by the presence of virus, fungi or bacteria in the bloodstream (Singer, Deutschman, & Seymour, 2016). Severe sepsis is defined as sepsis that is associated with organ dysfunction in forms of coagulation, abnormalities or dysfunctional mental status. Septic shock is sepsis that is accompanied by hypotension which could require that a vasopressor agent is administered. Septic shock is associated with tissue perfusion signs in the form of oliguria (Singer et al. 2016). Sepsis infection is either caused by the presence of bacteria, fungi or protozoa in the bloodstream. This paper will examine the pathophysiology of sepsis, its clinical manifestation, and medical management.

**Pathophysiology**

Sepsis has a pathogenesis that involves interaction between the immune system of the host and the microorganisms that infects the host. In this interaction bacteria as well as their by-products leads to the reaction of host body in response that involves several cells such as platelets, mast cells, leukocyte and endothelial cells in a process known as immune response process. This process also leads to coagulation, anti and pro-inflammatory, apoptosis and adhesion and complement activation (Mossie, 2013). This cells and processes are balanced so that they optimally protect the host from the invasion of pathogens as well as facilitate healing processes. These group of cells balances Pro-inflammatory and anti-inflammatory. However, when there is no balance between the mediation processes, sepsis is produced.

The imbalance causes the excess inflammatory response to invading pathogens to extend to the non-target area resulting in general functional alteration. This further trigger the release of other mediators and vasodilators which is known as inflammatory response to infection that leads to further general functional alteration. Following this, monocytes and macrophages secrete inflammatory cytokines damaging vascular endothelium. This triggers thrombin release and fibrin clot formation that coagulates blood. These processes lead to organ dysfunction. To begin with, sepsis disrupts nitric oxide regulation and thus leads to widespread vasodilation. This together with reduced contraction ability of myocardial as well as capillary leakage, causes cardiac dysfunction (Mossie, 2013).

Sepsis is also associated with intravascular coagulation, extended clotting time and low platelets count which results in hematological dysfunction. Another organ failure is liver dysfunction. This is caused as a result of insufficient hepatic blood flow compared to oxygen demand. Also, the endothelial dysfunction that is caused by sepsis is associated with alveolar collapse, rising work of breathing and hypoxemia. Moreover, due to the fact that sepsis causes systemic vasodilation as well as coagulation factors and neutrophil secretions, renal hypoperfusion could result (Marik, 2014).

Lastly, sepsis is associated with metabolic dysfunction as a result of an excessive reaction to normal factors such as starvation and stress. Factors such as impaired oxygen use could lead to a metabolic response that will cause changes at the cell level. This impairs glucose utilization, gastrointestinal dysfunction and impaired lactate concentration (Singer et al., 2016).

**Clinical Manifestation**

Organ failure first presents Sepsis's clinical manifestation. When immune cells secret various groups of mediators to respond to pathogens such as endotoxin, the free endotoxin formed from degradation of the cell wall of Gram-negative bacteria combines with macrophage receptors and triggers a cascade of reactions. Patients with severe sepsis will show biphasic hemodynamic response as well as immunological response. The early phase mainly caused by pro-inflammatory response will be exhibited through, tissue perfusion, increase heart rate and low vascular resistance (Marik, 2014).

The second phase known as the hypo-responsive phase is exhibited clinically through hypotension, low cardiac output, small pulse and low contraction and relaxation of cardiac. It also in advance stages may result in nosocomial infection and even death (Marik, 2014).

**Medical Management**

There is growing evidence that effective management of sepsis begins with early recognition and early goal-directed antibiotic therapy. Also, resuscitation could be important in the management of sepsis. The adequacy, liquid type, and volume are important considerations that could help in achieving high success with resuscitation. Another management is the use of vasopressors such as colloids or crystalloid during the management and resuscitation in sepsis shock. However, this should be given with care as colloids could be limiting to the capacitance of large venous vessels (Marik, 2014).

The other method that could be used in the management of sepsis where the infected parts are surgically drained. Mechanical ventilation could also be initiated especially through the supply of low tidal volume to the lungs. This protective lung ventilation is efficient management of sepsis with improved health outcomes (Marik, 2014).

**Conclusion**

This paper examined the pathophysiology of sepsis. Sepsis is a very complex condition caused by process of chain reactions between host response to invading pathogens and the resultant excessive response that triggers cascades of reaction that causes organ dysfunction. The clinical manifestations of sepsis were also discussed. The medical management of sepsis through early recognition and therapy was also highlighted. Other treatment intervention for sepsis included use of vasopressors, mechanical and protective lung ventilation.

**References**

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