

Sepsis and Septic Shock

Barbara Holmes Gobel, RN, MS, AOCN®, and Glen J. Peterson, RN, OCN®, ACNP

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Sepsis and septic shock are both potentially life-threatening oncologic emergencies. Septic shock is one stage in a clinical continuum of infection and systemic inflammatory mechanisms progressing from sepsis to severe sepsis and finally to septic shock. These stages make up a syndrome referred to as *systemic inflammatory response syndrome* (SIRS) (Muckart & Bhagwanjee, 1997). Septic shock is manifested by hemodynamic instability and alterations in cellular metabolism caused by sepsis. Septic shock is characterized by fever, chills, tachycardia, tachypnea, mental status changes, and hypotension/hypoperfusion that persist despite aggressive fluid challenge. This disorder occurs when the body fails to initiate an adequate immune response to bacterial, viral, or fungal infection. Management of this syndrome requires prompt assessment and treatment. Early detection and intervention related to sepsis and septic shock increase patients' chances of a positive outcome. Patients with cancer who are granulocytopenic are at risk for the development of sepsis and septic shock, particularly patients with hematologic malignancies. Nursing management of sepsis and septic shock requires knowledge of the risk factors for development of this syndrome, measures to prevent infection and sepsis, and knowledge of the many various medications that are used to treat sepsis and septic shock. Early hemodynamic assessment of patients suspected of sepsis is critical for definitive recognition and treatment to provide maximal benefit in terms of a positive outcome.

Pathophysiology

Infection

Infection occurs when an organism or microbe enters the bloodstream and is able to colonize and reproduce within a host. Disease results if the infectious organism, such as gram-negative bacteria, causes injury, pathologic changes, or organ dysfunction in the host (see Figure 6-1). Risk factors include granulocytopenia, malignancy-related immunosuppression, age, loss of skin or mucosal injury, long intensive care stays, central vascular access devices, indwelling devices, diabetes, and organ-related diseases.

Sepsis

In the presence of infection, the body mounts an inflammatory response. If a localized inflammatory response is insufficient to manage the infection, sepsis occurs. Sepsis is defined as a systemic response to infection. Substances such as endotoxins (released from gram-negative bacteria) and exotoxins (released from gram-positive bacteria) are released from the cell walls of dead bacteria after they have been phagocytized by neutrophils (Gobel, 2005). These toxins activate the coagulation cascade and the complement systems (Peterson, 1998). The activation

of the complement system causes a release of vasoactive mediators such as kinins, histamine, interleukins, and tumor necrosis factor- α (TNF- α), all of which lead to vasodilation and potentially to capillary leak syndrome. These vasoactive mediators further activate the coagulation cascade, which leads to the aggregation of platelets and formation of fibrin, resulting in the coagulopathies seen in sepsis. Initially, increases in inflammatory mediators may characterize sepsis, but as sepsis persists, a shift toward an anti-inflammatory immunosuppressive state occurs (Oberholzer, Oberholzer, & Moldawer, 2001). Patients with sepsis who are immunosuppressed have a loss of delayed hypersensitivity, an inability to clear infection, and a predisposition to nosocomial infections (Oberholzer et al.).

A high cardiac output with widespread vasodilation and normal or slightly elevated blood pressure usually characterize the early stage of sepsis. Vasodilation increases if sepsis continues without adequate treatment, and a capillary leak may develop, resulting in significant edema. Without adequate treatment, the blood pressure falls, cardiac function becomes further depressed, and severe sepsis occurs. In severe sepsis, decreased blood flow occurs because of the continuation of fibrin formation and aggregation

Barbara Holmes Gobel, RN, MS, AOCN®, is an oncology nurse specialist at Northwestern Memorial Hospital and adjunct faculty at Rush University Medical Center in Chicago, IL; and Glen J. Peterson, RN, OCN®, ACNP, is a nurse practitioner in Hematological Malignancies and Blood and Marrow Transplant at the University of Colorado Hospital and Health Sciences Center in Aurora. No financial relationships relevant to the content of this article have been disclosed by the editorial staff.

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