Surgical Critical Care for the Patient with Sepsis and Multiple Organ Dysfunction

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KEYWORDS

• Multiple organ dysfunction syndrome • Sepsis • Septic shock • Surgical critical care • Severe sepsis • Sepsis treatment

KEY POINTS

- Sepsis and multiple organ dysfunction syndrome (MODS) are common problems among surgical intensive care unit patients, and a significant source of cost, morbidity, and mortality.
- The concept of sepsis must incorporate both the infectious insult and the host's response to that insult.
- Early recognition of the symptoms, physiologic disturbances, and laboratory findings of sepsis, and prompt and aggressive interventions, are critical to the success of therapy.
- Treatment of the patient with MODS should focus on control and treatment of an infectious source, along with supportive therapy to maintain organ homeostasis.
- Despite advances in treatment, sepsis and MODS carry a significant mortality rate; early discussion helps to establish expectations and goals of care.

INTRODUCTION

Sepsis and multiple organ dysfunction syndrome (MODS) are clinical entities commonly seen in the critically ill surgical patients. Advances in medical therapies have resulted in a more elderly population, and have allowed critically ill and injured patients to survive their initial illness, only to develop sepsis thereafter. There are an estimated 1.1 million cases of sepsis in the United States annually, costing more than $24.3 billion.[1]

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The concept of sepsis as a discrete clinical entity was first outlined by Bone and coworkers[2] in 1989, and can be defined as the invasion of microorganisms and/or their toxins into the patient's bloodstream, in concert with the patient's response to that infection characterized by systemic inflammation. Sepsis is characterized by hemostatic dysregulation and endothelial dysfunction, resulting in compromise of both the circulatory system and intracellular homeostasis. The resultant cellular hypoxia and programmed cell death (apoptosis) are responsible for organ dysfunction and death.

INCIDENCE AND RISK FACTORS

Sepsis is common in the intensive care unit (ICU), and 10% to 15% of patients in the ICU develop septic shock, with a mortality rates of 50% to 60%.[3] Sepsis remains the number one cause of death in noncardiac ICUs, and surgical patients account for one-third of sepsis cases in the United States.[1,4] In surgical patients, sepsis and septic shock are 10 times more common than perioperative myocardial infarction and pulmonary embolism, with septic shock carrying the highest mortality.[5]

Common sources of sepsis in surgical patients include surgical wound and organ space infections, pneumonia, urinary tract infections, and catheter-associated bloodstream infections. Among hospital-acquired infections, nosocomial pneumonias are most common at 43%, followed by urinary tract infections at 25%. Catheter-associated bloodstream infections increased with the increased use of central lines, invasive monitoring, and parenteral nutrition since the 1970s but have declined over the past decade in response to standardization of catheter insertion bundles. Surgical site infections also continue to decline owing to improvements in perioperative antibiotics and sterile technique but still account for 10% of nosocomial infections.[6] Polymicrobial infections are more common in patients with organ space infections.

Advanced age and male gender are known risk factors for the development of sepsis. Chronic health issues such as obesity, alcohol use, immune status, physical conditioning, and medical comorbidities can contribute to the genesis and progression of sepsis. Finally, we are just beginning to understand that genetic factors may be an influence in sepsis development and patient survival.[7–10]

PATHOGENIC STIMULUS

Fundamental to the diagnosis of sepsis is the presence of infection. Although activation of the systemic inflammatory response syndrome (SIRS) is the final common pathway leading to MODS, signal pathways vary depending on the infecting organism. In gram-negative sepsis, the initiation of the host immune response is mediated primarily by lipopolysaccharide from the bacterial cell wall. Lipopolysaccharide binds to CD14 and Toll-like receptor-4 receptors to induce activation of a transcription factor known as nuclear factor kappa-B.[11,12] Nuclear factor kappa-B then activates gene promoters, which results in the transcription and expression of genes for cytokines and other proinflammatory mediators.[13] Gram-positive bacteria lack lipopolysaccharide, and are characterized by cell wall components (peptidoglycans and others), as well as specific bacterial toxins. The main pattern of recognition in gram-positive bacteremia is via lipoteichoic acid, a cell wall component found in all gram-positive bacteria that, along with other components of gram positive bacteria, interacts with the Toll-like receptor-2 receptor.[14] The resulting release of tumor necrosis factor (TNF)-α, interleukin (IL)-6, and IL-10 seem to be through a similar pathway of signal transduction.[13] The initiation of the host response and release of proinflammatory