Cardiogenic Shock in the Septic Patient

Early Identification and Evidence-Based Management

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KEYWORDS

• Cardiogenic shock • Sepsis • Myocardial dysfunction • Sepsis and heart failure

KEY POINTS

- Sepsis is an inflammatory process that results in vasodilation and increased capillary membrane permeability.
- In sepsis, hemodynamic changes occur, including a decrease in systemic vascular resistance and cardiac output.
- Myocardial dysfunction in sepsis includes impaired contractility.
- Rapid diagnosis, prompt supportive treatment, and revascularization of coronary arteries are priorities in cardiogenic shock.

Sepsis and septic shock are medical emergencies that are responsible for 1 in 4 deaths worldwide.[1] Seven in 10 adult patients with sepsis have chronic diseases such as diabetes, cardiovascular disease, heart disease, and cancer.[2] Such chronic diseases contribute to the patient's risk for sepsis and complicate the patient's treatment and recovery. It is reasonable to deduce that the presence of chronic cardiac disease increases the patient's risk for shock and the mortality risk when septic. Therefore, early identification and initiating the most current best practices are crucial to the patient experiencing optimal outcomes.

BACKGROUND

With the evolution of sepsis research, updated definitions for sepsis and septic shock are available. Sepsis is "a life-threatening organ dysfunction caused by a dysregulated

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host response to infection."[1] Septic shock is "a subset of sepsis with circulatory and cellular/metabolic dysfunction associated with a higher risk of mortality."[1] Both definitions articulate the severity of illness for a patient with either diagnosis. Patients with sepsis and preexisting cardiac disease, such as heart failure, are more vulnerable to cardiac and hemodynamic decompensation than patients with different comorbidities.[3]

The pathogenesis of sepsis involves myocardial depression early in the disease state, contributing to organ dysfunction and hemodynamic compromise.[4] An ultimate consequence of sepsis-induced myocardial depression and hemodynamic instability in the patient with chronic heart failure is cardiogenic shock. Cardiogenic shock is a low cardiac output (CO) state resulting in life-threatening end-organ hypoperfusion and hypoxia in the setting of adequate vascular volume.[5–7] The septic patient progressing to cardiogenic shock is likely to have higher morbidity and mortality rates.[7] Nurses must be able to identify and adequately manage patients at risk for sepsis-induced cardiogenic shock.

LITERATURE REVIEW

A literature review was performed to isolate evidence-based practices for identifying and managing patients experiencing sepsis-induced cardiogenic shock. The Cumulative Index to Nursing and Allied Health Literature and PubMed databases were searched using the key words: cardiogenic shock, sepsis, myocardial dysfunction, and sepsis and heart failure. The search was refined to filter related research and literature over the past 10 years. Thirty-five articles were found and their abstracts reviewed. Twenty manuscripts were specific to the topic. Of the 20 manuscripts included in the review, 14 were review of the literature, 3 were guidelines, 2 were consensus statements, and 1 was a randomized controlled trial.

Pathophysiology of Sepsis

Sepsis-induced myocardial dysfunction occurs secondary to myocardial injury from the dysregulated systemic release of inflammatory cytokines in response to the infectious toxin and mitochondrial dysfunction secondary to tissue ischemia. These pathophysiologic mechanisms can lead to the development of cardiogenic shock.[8] Cardiogenic shock is a physiologic response to a disease state such as sepsis. The severity of cardiogenic shock in the septic patient directly correlates to the degree of preexisting myocardial dysfunction and sepsis-induced myocardial injury.[9]

Sepsis-Induced Myocardial Dysfunction

Sepsis-induced myocardial dysfunction occurs immediately in response to endotoxins released by the causative microorganisms. Endotoxins injure endothelial cells, subsequently triggering the release of inflammatory immunocompetent chemicals such as cytokines, tumor necrosis factor, and interleukins. The dysregulated release of such inflammatory chemicals results in myocardial depression, vasodilation, increased capillary permeability, and enhanced nitric oxide production. Nitric oxide decreases myofibril response to calcium, causing mitochondrial dysfunction, the downregulation of beta-adrenergic receptors, and further systemic vasodilation. Consequently, mechanisms to maintain adequate CO and tissue perfusion are compromised secondary to myocardial contractile depression, absolute intravascular volume loss to the interstitial space, and relative intravascular volume loss from systemic vasodilation. Diminished oxygen delivery and increased oxygen demand result in an imbalance of oxygen needs for cellular metabolism, resulting in a state of