Anesthesia for Patients with Concomitant Sepsis and Cardiac Dysfunction

Abed Abubaih, MD, Charles Weissman, MD*

KEYWORDS

• Sepsis • Septic shock • Cardiac dysfunction • Physiologic monitoring
• Sepsis-induced cardiac dysfunction • Coronary artery disease • Vasopressors
• Inotropic agents

KEY POINTS

- Cardiac dysfunction in patients with sepsis may be caused by the sepsis itself or preexisting cardiac disease.
- Anesthetic management in patients with severe sepsis or septic shock with concomitant cardiac disease involves optimizing hemodynamic and metabolic functions.
- Among the aims of managing these patients is preventing further organ damage, such as using protective ventilation to prevent or reduce lung injury.
- Increased mortality is caused by the inability of patients with septic shock with concomitant cardiac disease to adequately compensate for the sepsis-induced cardiovascular changes.

INTRODUCTION

Patients suffering from sepsis and septic shock often undergo surgery to treat the source of their sepsis or a complication thereof. Anesthetizing such patients is challenging especially if there is concurrent cardiac dysfunction. Sepsis is associated with cardiac dysfunction under a variety of circumstances (**Box 1**). Therefore, anesthesiologists faced with a patient with sepsis with concurrent cardiac dysfunction must be cognizant of the patient's cardiac status and the cause of the cardiac problem so that they can appropriately tailor their anesthetic management and physiologic monitoring.

Cardiac Dysfunction Attributable to Sepsis

Although sepsis is usually associated with a hyperdynamic state (elevated cardiac output in face of reduced systemic vascular resistance), there is often underlying

Department of Anesthesiology and Critical Care Medicine, Hadassah – Hebrew University Medical Center, Hebrew University – Hadassah School of Medicine, Jerusalem, Israel
* Corresponding author. Hadassah-Hebrew University Medical Center, Kiryat Hadassah, POB 12000, Jerusalem 91120, Israel.
E-mail address: charles@hadassah.org.il

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Box 1
Sepsis and concomitant cardiac dysfunction

1. Cardiac dysfunction secondary to the sepsis itself (sepsis-induced cardiac dysfunction).
2. Preexisting cardiac dysfunction (eg, chronic heart failure, ischemic heart disease) in the patient with sepsis.
3. Combined acute cardiac disease and sepsis-induced cardiac dysfunction (eg, bacterial endocarditis and septic shock).
4. The stress of sepsis and septic shock (eg, hypotension, tachycardia) causing cardiac dysfunction (eg, myocardial ischemia and infarction).

disordered myocardial function (**Box 2**). Compensatory mechanisms, such as tachycardia and left ventricular dilation, attempt to compensate for this myocardial dysfunction. When these compensatory mechanisms fail and/or cardiac filling decreases substantially, septic shock ensues. In some patients, a hypodynamic (low cardiac output) state is found, with 25% of adult patients having relatively low cardiac outputs even after fluid resuscitation.[1] Merx and Weber[2] found that the survival rate decreased in patients with sepsis with cardiac dysfunction (septic cardiomyopathy).

Preexisting Cardiac Dysfunction in the Patient with Sepsis

Not only can sepsis itself cause cardiovascular dysfunction, but preexisting cardiac conditions can be exacerbated by sepsis and its treatment. A significant cardiac history, defined as prior myocardial infarction, abnormal treadmill report, nuclear medicine study, or coronary angiogram, or history of congestive heart failure or arrhythmia requiring treatment, was associated with increased mortality in severe sepsis and septic shock.[3]

Box 2
Cardiac dysfunction associated with sepsis

1. Reduced global left ventricular systolic function.
2. Decreased global longitudinal peak strain.
3. Depressed left ventricular ejection fraction (LVEF) (20%–60% of patients with septic shock).
4. Left ventricular dilatation.
5. Left ventricular diastolic dysfunction.[40]
6. Right ventricular systolic dysfunction.[41]

- Patients with sepsis with either systolic or diastolic dysfunction or a combination of both have higher mortality than those diagnosed with sepsis but without diastolic or systolic dysfunction. However, left ventricular systolic dysfunction associated with sepsis when defined as low LVEF is neither a sensitive nor a specific predictor of mortality.[42]
- Sepsis-associated cardiac dysfunction is reversible in survivors.
- Attenuation of the adrenergic response at the cardiomyocyte level likely explains the reduced response to exogenously administered catecholamines.
- Catecholamine-mediate myocardial injury may possibly occur in addition to septic (inflammatory)-mediated myocardial depression.