



Sepsis-associated In-hospital Cardiac Arrest



Sepsis-associated cardiac arrest is a relatively common occurrence with especially poor outcomes. A review paper, published in *Journal of Critical Care*, discusses the epidemiology of sepsis-associated in-hospital cardiac arrest in adults and children, the relevant physiology responsible for its pathogenesis and poor outcomes, and potential therapeutic interventions based on this pathophysiology.

Over 200,000 patients suffer an in-hospital cardiac arrest (IHCA) each year in the United States, with only one-fourth surviving to hospital discharge. The precise mechanisms by which septic shock leads to cardiac arrest have not been explicitly characterised. However, the cardinal manifestations of septic shock, including vasodilation, hypovolemia, and myocardial dysfunction, along with concurrent hypoxemia, acidosis, and metabolic derangements likely form the basis of the complex pathophysiologic pathways that both contribute to cardiac arrest and impede successful return of spontaneous circulation (ROSC) in these patients, according to researchers.

Prompt recognition and treatment of septic shock are the first steps in preventing IHCA in these patients. Early goal-directed therapy aimed at optimising intravascular volume status and correcting metabolic derangements, in addition to timely administration of antimicrobials and other source control measures can reduce mortality in septic shock.

"Recognition of sepsis-associated cardiac dysfunction, likely a major precipitator of cardiac arrest, is important. This can be accomplished through clinical examination, measurement of serum levels of natriuretic peptides, and echocardiography, and should trigger the use of inotropic support in addition to vasopressors," the authors note.

High-quality cardiopulmonary resuscitation (CPR) with emphasis on appropriate chest compression rate, depth, and recoil; vasopressor administration; and timely defibrillation has been shown to increase the likelihood of ROSC, survival to hospital discharge, and satisfactory neurologic recovery following IHCA. Given the higher rate of adverse outcomes from IHCA in the setting of septic shock, the authors say further work is necessary to identify targeted therapeutic interventions to enhance CPR and improve outcomes specifically in sepsis-associated IHCA.

The higher rates of post-ROSC mortality with sepsis-associated IHCA reflect the overall severity of disease and degree of multiple organ dysfunction in these patients, which is not simply corrected with ROSC.

Derangements in vascular tone and intravascular volume status persist and likely worsen after cardiac arrest, the authors explain. While aggressive fluid resuscitation may have preceded or accompanied resuscitation from cardiac arrest, relative intravascular hypovolemia can persist and further volume administration may be indicated.

"Experimental conditions that model sepsis-associated IHCA need to be developed to test novel CPR algorithms and post-resuscitative therapies. Most importantly, sepsis-associated cardiac arrest provides a prime example for the need to tailor therapies to the physiologic response of the patient. Understanding and targeting sepsis-related pathophysiology before, during, and after cardiac arrest has great potential to improve patient outcomes," the study concludes.

Source: Journal of Critical Care

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