Management of Sepsis in Patients with Pulmonary Arterial Hypertension in the Intensive Care Unit

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KEYWORDS

• Pulmonary arterial hypertension • Sepsis • Right ventricular failure • Cardiac output

KEY POINTS

- Sepsis is an inflammatory process that results in increased capillary membrane permeability and vasodilation.
- Hemodynamic changes that occur in sepsis include a decrease in systemic vascular resistance (SVR) that must be compensated by an increase in cardiac output (CO).
- In pulmonary arterial hypertension (PAH), there is an increase in right ventricular end diastolic pressure and a decrease in CO.
- Management of septic shock in PAH is complex.
- Goals of therapy include maintaining tissue perfusion and eradicating the cause.

Sepsis begins with an inflammatory process that results in increased capillary membrane permeability and vasodilation. Proinflammatory and anti-inflammatory mechanisms are activated and contribute to a cascade of endothelial injury, global tissue hypoxia, and formation of microthrombi. Patients with sepsis experience a decrease in intravascular volume and a fall in SVR that must be compensated for by an increase in CO. In patients diagnosed with PAH, the ability to suddenly increase CO to meet increased metabolic demands may be severely limited. Patients with increased pulmonary artery pressures in the setting of sepsis may experience rapid deterioration of right ventricular function, hemodynamic instability, and death.[1–4] As a result, the management of septic shock in PAH patients in the intensive care unit can be extremely challenging.

The pulmonary vasculature contains arteries and arterioles that branch in the lungs to create a dense capillary bed to advance blood flow. The pulmonary capillary bed is

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a high-volume, low-pressure, low-resistance system and is highly vascularized as blood is delivered to and taken away by arterial and venous circulations, respectively. Despite generating essentially the same CO, the vascular bed through which the right and left ventricles pump is very different. The pulmonary vascular resistance (PVR), through which the right ventricle must pump against, is remarkably low (less than 150 dynes/s/cm^5/m^2) and the SVR through which the left ventricle must pump against is considerably higher (800–1200 dynes/s/cm^5/m^2). In healthy individuals, the arterioles are able to dilate in times of increased metabolic demand. During exercise, the CO may increase by 4-fold above baseline with a minimal increase in pulmonary artery pressure and reduction in PVR.[3,5]

In PAH, there is an increase in pulmonary artery resistance and right ventricular failure as a result of smooth cell proliferation in the intima causing a thickening of the arteriole walls with progressive vascular narrowing.[6,7] This leads to elevated PVR and increased right ventricular end-diastolic pressure. As right ventricular end-diastolic pressure rises and exceeds left ventricular end-diastolic pressure, the interventricular septum shifts toward the left ventricle, decreasing left ventricular compliance, resulting in decreased left ventricular CO. The ability of filling pressures of the right ventricle to affect filling pressures of the left ventricle via the interventricular septum is referred to as ventricular interdependence. Ventricular interdependence represents a challenge to fluid management in PAH patients who are in septic shock.[3]

MANAGEMENT OF SEPSIS IN PULMONARY ARTERIAL HYPERTENSION

Patients with PAH presenting with septic shock have inadequate perfusion of tissues and lack adequate oxygen delivery to organs resulting in lactic acidosis, multiorgan failure and possibly death. Septic shock must be treated immediately to minimize the risk for multiple organ failure and death. The need for aggressive management of infectious complications in PAH patients with septic shock is warranted because sepsis at any time in an intensive care unit is a strong predictor of death.[8]

Management of septic shock in PAH is complex and requires expertise. Goals of therapy include maintaining adequate tissue perfusion by increasing intravascular volume and increasing CO while eradicating the cause. In patients with PAH, goal-directed therapy focuses on augmenting right ventricular function while decreasing PVR. Strategies to maximize right ventricular function include optimizing right ventricular preload, improving right ventricular contractility, and reducing right ventricular afterload, which is achieved by lessening PVR. In PAH, an acute reduction in PVR is problematic due to the relatively fixed nature of the pulmonary vascular disease. Treatment strategies are directed at optimizing right ventricular function and limiting increases in PVR in response to metabolic derangements caused by sepsis or vasopressors administered during resuscitation.[3,7,9]

RIGHT VENTRICULAR PRELOAD

Treatment of septic shock in PAH patients should be initiated as quickly as possible with proper fluid management. Once an organism invades a host, an inflammatory response is initiated to restore homeostasis. Cytokines are released and damage endothelial cells that line blood vessels resulting in profound vasodilation and increased capillary permeability. A decrease in intravascular volume and venous vascular tone lowers central venous pressure (CVP).[1,3,10]

To maintain CO in acute right ventricular decompensation in septic shock, establishing adequate right-sided filling pressure is essential. Therefore, early goal-directed therapy includes initiation of volume resuscitation if CVP is low. Fluid