Decompensated Cirrhosis and Fluid Resuscitation

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KEYWORDS

• Cirrhosis • Albumin • Normal saline • Hepatorenal syndrome • Antidiuretic hormone • Hyponatremia

KEY POINTS

- Understanding of the unique physiology of end-stage liver disease is imperative to resuscitation of the patient with cirrhosis.
- The effects of albumin resuscitation in the patients with cirrhosis are more than mere volume expansion.
- Decompensated cirrhotics are total body volume expanded but intravascularly volume deplete.

INTRODUCTION

Physician encounters with patients with cirrhosis have become prevalent, with 1 in 10 Americans having some form of liver disease. Despite advances in the treatment of hepatitis C, the incidence of liver disease has not decreased and according to the National Institute of Health 10% of children in the United States have nonalcoholic fatty liver disease.[1] Cirrhosis carries a significant increase in mortality with the Centers for Disease Control and Prevention citing it as the fourth leading cause of death of Americans between the ages of 45 and 54 and the twelfth leading cause overall.[2] In-hospital mortality is reportedly 44% to 74% in some studies with yearly costs approaching $13 billion.[3] Given the prevalence of liver disease it is likely that all surgeons independent of specialty will encounter a patient with cirrhosis with nearly 10% of patients with cirrhosis undergoing surgery in their last 2 years of life. The understanding of resuscitation of the decompensated patient with cirrhosis is vital to decreasing morbidity and mortality. This article enhances the understanding of the unique physiology of the patient with decompensated cirrhosis to guide their needs in fluid resuscitation in critical illness.

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PHYSIOLOGY OF LIVER DISEASE

Before discussing specifics of fluid resuscitation in patients with liver disease it is imperative to understand the unique physiology of the patient with cirrhosis (Fig. 1). Portal hypertension in the setting of cirrhosis leads to splanchnic and arteriolar vasodilation. The exact mechanism of this is not exactly understood but nitric oxide is thought to play an important role. This dilation leads to a significant decrease in systemic vascular resistance, decreasing the effective arterial blood volume and blood pressure, which leads to a cascading chain of events. In response to the decrease of effective circulating blood volume the sympathetic nervous system and the renin-angiotensin system (RAAS) increase to try to compensate along with excretion of endogenous vasopressin. The activation of the RAAS leads to an increase in release of antidiuretic hormone leading to sodium and water retention with a disproportionate amount of free water retention increasing plasma volume, which can result in significant hypervolemic hyponatremia. The increase in sympathetic nervous system leads to an increase in heart rate and overall increase in cardiac output, which increases splanchnic blood flow.

DETERMINATION OF VOLUME STATUS

Determination of volume status in the patient with cirrhosis is important but often difficult to determine given that up to 50% of extracellular fluid may be in the extravascular space manifesting is ascites and edema.[4] Patients who seem total volume expanded may often be intravascularly volume depleted putting them at risk for hepatorenal syndrome (HRS). Overresuscitation of the postoperative patient with liver disease can result in ascites and hyponatremia, which is difficult to treat.[5] In a study aimed to evaluate the effect of plasma expansion with albumin in patients with cirrhosis with renal failure, global end-diastolic blood volume index but not central venous pressure served as an indicator of cardiac preload. When examining predictors of fluid responsiveness, central venous pressure, global end-diastolic blood volume index, stroke volume index, and cardiac index were significantly lower than in nonresponders, where a systemic vascular resistance index was significantly higher.[6]

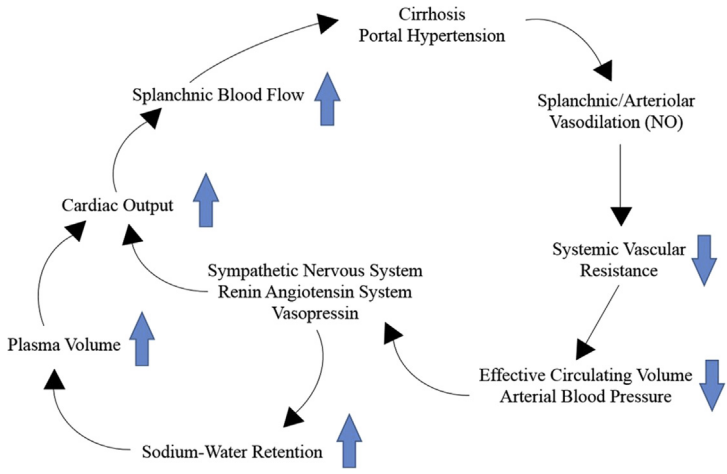

Fig. 1. Flowchart of physiologic events.