Acute Kidney Injury in Cirrhosis

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KEYWORDS

• Acute on chronic liver failure (ACLF) • Acute kidney injury • Cirrhosis
• Hepatorenal syndrome • Acute dialysis quality initiative • Acute kidney injury network

KEY POINTS

- Evaluation of renal function and identification of the cause of acute kidney injury (AKI) in cirrhotic patients remains a challenge.
- Serum creatinine (sCr) remains the most commonly used clinical index of kidney function; however, it is influenced by a variety of factors.
- Given the limitations of calculating glomerular filtration rate (GFR) with current laboratory techniques, neutrophil gelatinase-associated lipocalin (NGAL), cystatin C, and other novel biomarkers potentially may in the future assist in differentiating hepatorenal syndrome (HRS)-AKI from structural kidney damage.
- Vasoconstrictor therapy (noradrenalin and terlipressin) is the primary therapy in HRS-AKI, although without a demonstrated mortality benefit.
- Predicting renal recovery post–liver transplant (LT) based on current criteria (time on renal replacement therapy [RRT], biomarkers) remains controversial.

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INTRODUCTION

AKI is a frequent complication of end-stage liver disease, especially in those with acute-on-chronic liver failure (ACLF), occurring in up to 50% of hospitalized patients with cirrhosis.[1–5] The high incidence of AKI is due to the combination of an impaired effective arterial blood volume secondary to arterial vasodilation, with increased intrarenal vasoconstriction and impaired renal autoregulation that predisposes to renal dysfunction, and several precipitating factors related to cirrhosis, typically bacterial infections and gastrointestinal bleeding.[6–8] There is no specific blood or urine biomarker that can reliably identify the cause of AKI in cirrhotic patients. Traditional diagnostic criteria focused particular attention on HRS and its physiology of renal vasoconstriction and splanchnic vasodilatation[9] with criteria based on elevation in sCr level greater than 50% over baseline with value greater than1.5 mg/dL (133 μmol/L). Initial studies suggested that irreversibility of HRS had a deleterious impact on mortality.[10] However, subsequent studies have questioned these criteria as being narrow and required a broader look at AKI in cirrhosis.[11,12]

EPIDEMIOLOGY

Classification of renal dysfunction in cirrhotic patients can be based on acuity of presentation (acute, chronic, or acute on chronic); however, most cirrhotic patients (~70%) have AKI without structural changes.[13] Causes of AKI include hypovolemia/prerenal azotemia, intrinsic renal/parenchymal disorders (acute tubular necrosis [ATN], interstitial nephritis, glomerular disease), obstructive nephropathy, and HRS.[14]

HRS is a severe complication of advanced cirrhosis. Its clinical manifestations are related to changes in renal, hepatic, and the systemic circulation. HRS is a consequence of intense renal vasoconstriction leading to a reduction in renal perfusion and glomerular filtration. The ability of the kidney to excrete sodium and free water is also severely impaired without histologic changes accounting for this renal impairment. Traditionally, HRS has been described in 2 different clinical patterns, according to intensity and onset of renal failure.[15] Type 1 HRS increasingly represents the severe end of the spectrum of renal failure in cirrhosis. It is characterized by rapidly progressive renal failure with oliguria. It is defined as a doubling of the sCr level to greater than 2.5 mg/dL or a 50% reduction in 24-hour creatinine clearance to a level less than 20 mL/min in less than 2 weeks. Type II HRS progresses slowly and represents a less severe deterioration in renal function that may remain stable for extended periods. The dominant clinical feature of a patient with type II HRS is refractory ascites. This condition is the result of intense sodium retention, reduced glomerular filtration, and marked stimulation of the renin-angiotensin system. The Acute Dialysis Quality Initiative (ADQI) and the International Club of Ascites (ICA) have proposed a revision of these traditional criteria to define HRS and to remove sCr cutoff values to define HRS.[11,12,14,16]

PATHOPHYSIOLOGY OF HEPATORENAL SYNDROME

HRS is derived primarily from circulatory failure. According to the peripheral vasodilatation model, in cirrhosis the decrease in splanchnic and systemic arterial vascular resistance is likely related to increased expression of endothelial nitric oxide synthase and the concentration of nitric oxide and its metabolites in the splanchnic as well as systemic circulation.[17] In contrast, the production of nitric oxide in the intrahepatic circulation is reduced, exacerbating portal hypertension. The resultant decreased mean arterial pressure (MAP) and low total systemic vascular resistance is offset initially in

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