Renal Insufficiency in the Patient with Chronic Liver Disease

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KEYWORDS

• Hepatorenal syndrome • Chronic liver disease • Acute kidney injury • Hyponatremia

KEY POINTS

- Acute kidney injury has replaced the previous term acute renal failure and is defined as an abrupt loss of kidney function with retention of urea and electrolyte and fluid imbalances.
- The pathophysiology of renal disease in the setting of chronic liver disease and cirrhosis explains why those with cirrhosis are at risk for kidney injury.
- Hyponatremia is another complication of cirrhosis that may lead to renal injury and disease.

Patients with chronic liver disease frequently develop renal insufficiency, with an estimated prevalence of 20% to 25%.[1] It is essential to determine whether a chronic liver disease patient with renal insufficiency has a single etiology that is affecting liver and kidneys, a kidney disease whose cause is different than the liver disease, or kidney disease as the result of chronic liver disease. Kidney disease from liver disease may be acute or chronic, and may lead to dialysis or kidney transplantation with or without liver transplantation. Finally, kidney disease may affect candidacy for liver transplant and long-term outcomes after transplant.[2–4] Moreover, it is essential that this evaluation occur in a timely manner, as the overall survival of patients with cirrhosis who develop renal failure (from all causes) is poor, at 50% at 1 month and 20% at 6 months.[5] This article will discuss the revised nomenclature of acute and chronic kidney injury, the differential diagnosis of those with renal insufficiency with chronic liver disease, and specific therapies and strategies to reduce the risk of developing renal insufficiency.

DEFINITIONS OF ACUTE KIDNEY AND CHRONIC KIDNEY INJURY

Acute kidney injury (AKI) has replaced the previous term acute renal failure and is defined as an abrupt loss of kidney function with retention of urea and electrolyte

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and fluid imbalances. In 2004, a consensus group proposed a set of definitions (RIFLE criteria [Risk, Injury, Failure, Loss, ESRD]) that described 3 levels of renal dysfunction (risk, injury, and failure) along with 2 outcome measures (loss and end-stage renal disease).[6] However, the definition of AKI has been recently modified in the liver disease population. This is because serum creatinine is not a reliable marker in assessing kidney function in patients with liver disease due to the low production of creatinine and decreased muscle mass in those with cirrhosis.[7,8] Thus equations including the Cockcroft-Gault and Modification of Diet in Renal Disease, which include serum creatinine, typically overestimate glumerular filtration rate (GFR) in patients with cirrhosis.[9] In addition, creatinine clearance by urine collection may also overestimate GFR cirrhotic patients, and it is sometimes difficult to collect urine accurately in a cirrhotic patient, making this approach somewhat less practical.[10] The gold standard of assessing GFR in the general population relies on clearance of iohexol or iothalamate, although ascites limits the use of these tests in cirrhotic patients.[1]

For these reasons, the International Ascites Club and the Acute Dialysis Quality Initiative have advocated using the more recent Acute Kidney Injury Network (AKIN) definition of AKI for patients with cirrhosis.[11] The AKIN criteria are classified into 3 stages. Stage 1 is an increase in serum creatinine of at least 0.3 mg/dL or an increase of 150% to 200% from baseline; stage 2 is an increase in serum creatinine greater than 200% to 300%, and stage 3 is a serum creatinine greater than 300%, or at least 0.5 mg/dL in patients with baseline serum creatinine of at least 4 mg/dL or renal replacement therapy (RRT).[12,13] This classification has important prognostic value, as 1 study demonstrated a mortality rate of 2% in stage 1, 15% in stage 2, and 44% in stage 3, although patients with stage 1 AKI with a serum of no more than 1.5 mg/dL had a comparable mortality rate at 3 months as the same population without AKI.[14] A recent report noted that the AKIN definition predicted 30-day mortality, length of hospital stay, and organ failure with a tenfold higher risk of mortality in those with irreversible AKI.[15] Thus clinicians should be aware that even small changes in measured creatinine warrant evaluation and intervention.

THE PATHOPHYSIOLOGY OF THE RENAL CIRCULATION IN CHRONIC LIVER DISEASE

The pathophysiology of renal disease in the setting of chronic liver disease and cirrhosis explains why those with cirrhosis are at risk for kidney injury. The presence of portal hypertension disrupts the normal circulatory system by leading to increased production of vasodilators including nitrous oxide, carbon monoxide, and endogenous cannabinoids in the splanchnic system, and this vasodilatation leads to a reduction in systemic vascular resistance.[16–19] In those with early stage cirrhosis, portal hypertension is not as prominent, and the heart is able to increase cardiac output to compensate for the decrease in vascular resistance, maintaining an effective blood volume.[5,16] As cirrhosis progresses with increased portal hypertension, the decrease in vascular resistance becomes more predominant, and there is no longer cardiovascular compensation; a decrease in cardiac output may be observed.[20] This leads to renin–angiotensin system activation to raise arterial pressures.[18] Although this helps maintain arterial pressures, there are deleterious effects on the kidneys, including sodium and water retention, as well as increased renal vasoconstriction, leading to hypoperfusion of the kidneys. Finally, angiotensin 2-mediated efferent glomerular arteriole vasoconstriction also helps maintain renal perfusion, and all of these mechanisms may lead to or exacerbate edema and ascites.[16,21]

In those with cirrhosis, bacterial translocation (from intestine to extraintestinal sites) plays an important role in leading to kidney injury and disease.[22,23] Bacterial

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