INTRODUCTION
Acute kidney injury (AKI) is common in cirrhosis but functional renal failure defined as Hepatorenal syndrome (HRS) accounts for 20% of AKI. HRS is reversible syndrome in patients with cirrhosis and ascites can appear spontaneously or follow precipitating event. HRS can be defined as presence of cirrhosis with ascites with serum creatinine more than 1.5 mg/dl, no improvement of serum creatinine (=/> 1.5mg/dl) after at least 48 hours of diuretic withdrawal and volume expansion with albumin 1gm/kg body weight per day for two days, absence of hypovolemic shock or severe infection requiring vasoactive drugs to maintain the arterial pressure, no current or recent treatment with nephrotoxic drugs, proteinuria less than 500 gms/day and no micro hematuria. Evolving concepts of renal dysfunction in cirrhosis have been discussed by various international groups in the recent past. International ascites club defined HRS in 1996, 2007 and 2015 while kidney associations which defined renal failure in other instances also defined kidney failure in cirrhosis. KIDGO (kidney disease improving global outcome), RIFLE (risk, injury, failure, loss of function, end stage renal disease), AKIN (acute kidney injury network) and ADQI (acute dialysis quality initiative) are important amongst them.

DIAGNOSIS OF HRS (TABLE 1)
It is based on AKI stage 2 or 3 with already mentioned criteria remaining same. Diagnosis of chronic kidney disease in cirrhosis is based on criteria: GFR less than 60 ml/minute calculated using MDRD six formula. HRS type II is defined as a specific form of chronic kidney disease. As many of the cirrhotics have GFR less than 60 ml/minute with serum creatinine levels being normal. Acute on chronic kidney disease is defined as rise in serum creatinine 50% from the baseline or a rise of serum creatinine more than 0.3mg/dL in less than 48 hours in patients with cirrhosis with GFR less than 60 ml/minute for more than three months calculated by MDRD six formula.

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<tr>
<th>Parameter</th>
<th>Definition</th>
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<td>Base line Serum Creatinine</td>
<td>Stable SCr ≤3 months &lt;br&gt; If not available, a stable SCr closest to the current one &lt;br&gt; If no previous SCr at all, use admission SCr</td>
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<td>Definition of AKI</td>
<td>Increase in SCr &gt; 26.5 umol/l (0.3mg/dL) ≥48 hours, or &lt;br&gt; Increase 50% from baseline</td>
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<td>Staging</td>
<td>Stage 1: Increase SCr ≥ 26.4 umol/L (0.3mg/dL) or &lt;br&gt; Increase SCr ≥ 1.5 – 2.0 x from baseline &lt;br&gt; Stage 2: Increase SCr ≥ 2 – 3.0 x from baseline &lt;br&gt; Stage 3: SCr &gt; 3.0 x from baseline or &lt;br&gt; SCr ≥ 352 umol/L (4.0mg/dL) with an acute increase of ≥ 26.4 umol/L (0.3mg/dL) or &lt;br&gt; Initiation of renal replacement therapy</td>
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Table 1: Diagnosis of AKI in Cirrhosis

![Fig. 1: Pathophysiology of HRS](image-url)
improving survival of patients with cirrhosis. In addition to albumin, Terlipressin has shown to be effective in treatment of HRS. HRS reversal has been shown in as many as more than 30% patients. Bolus vs continuous infusion of Terlipressin has been shown to be more effective. Small dose of Terlipressin is required in these patients. Significantly less side effects are seen with 2mg Terlipressin over 24 hrs. The other option is Midodrine / Octreotide infusion has been shown to be effective, but its effectiveness is less than Terlipressin. The other option is norepinephrine which has been shown to be equally effective as Terlipressin. Meta-analysis shows equal efficacy of norepinephrine as compared to Terlipressin. Stage I AKI which reverses to normal has been shown to have 2% mortality while AKI if progresses to stage II has 29% mortality, Stage III has 50% mortality and with requirement to dialysis goes up to 55%. AKI stage II if non-progressive, has mortality of 7% while if it progress, mortality is 18% and with requirement of dialysis it goes beyond 50%. AKI stage III if does not progress, mortality is 21% while it progresses, mortality is more than 70%. Regression of stage of AKI is associated with improved survival. Continuous renal replacement therapy (CRRT) should be used with caution in patients with AKI. CRRT is preferred in the removal of inflammatory cytokines such as IL6 and TNF alpha. In type II HRS, TIPS (Transjugular intrahepatic portasystemic shunts) has been used anecdotally. Liver transplantation is definitive treatment for the AKI not responding to therapy. There is still a lot of debate as to when to perform simultaneous liver and kidney transplantation. The general consensus is to do simultaneous liver and kidney transplantation if AKI has been present for more than four weeks. For patients who receive a liver transplant alone, transient persistence of renal dysfunction post-transplant may require short term dialysis.

REFERENCES