Acute Kidney Injury in Cirrhosis – Full clinical guideline

Reference no.: CG-T/2023/118

AKI is common in unwell hospitalised cirrhotics and is often multifactorial. Hepatorenal syndrome accounts for < 25% of AKI in cirrhotic patients admitted to hospital. Creatinine and eGFR should not be relied on as indicators of AKI as they can underestimate the occurrence of AKI in cirrhotic patients. **The key is to recognise a change from baseline.** It is not unusual for a cirrhotic patient to have a baseline creatinine of approximately 30 and so for a Creatinine of 70 with eGFR >60 to represent a significant deterioration in renal function. For practical purposes, management should focus on searching for and correcting any precipitant and offering general supportive measures. In cirrhosis the majority of cases are caused by a reduction in the effective circulating volume which needs to be restored. **Prompt recognition and treatment will avoid a rapid progression to established renal failure,**

AKI in cirrhosis is defined by modified KDIGO (Kidney Disease Improving Global Outcomes) criteria. Due to Na+ retention patients with cirrhosis may be oliguric despite maintaining GFR, while urine output may be increased due to diuretics. Urine output is, therefore, an unreliable indicator of AKI in cirrhosis.

AKI Stage 1: Increase in serum creatinine ≥ 26 µmol/L within 48h or increase of ≥ 1.5-2 x reference serum creatinine taken within 3/12
AKI Stage 2: Increase of ≥ 2-3 x reference serum creatinine taken within 3/12
AKI Stage 3: Increase of > 3 x reference serum creatinine taken within 3/12 or creatinine ≥ 354 µmol/l or initiation of renal replacement therapy

Hepatorenal syndrome (a diagnosis of exclusion): AKI (as defined above) in a patient with **cirrhosis and ascites** who has had no response to 48hrs of diuretic withdrawal and plasma volume expansion (see below) **and** absence of shock and no current nephrotoxic drugs **and** normal renal ultrasound **and** absence of proteinuria (> 500mg/day) or haematuria (> 50 RBC per high power field)

Determining the cause:

- Are there causes of hypovolaemia ? - review diuretics and lactulose
- Are there other potential drug causes? - NSAIDS, ACEI, Aminoglycosides etc
- Assess volume status - if the MAP < 65mmHg then autoregulation mechanisms are overwhelmed and renal blood flow begins to decline in proportion to renal perfusion press
- Screen for sepsis – urine, blood, ascitic fluid, CXR
- Urinalysis – blood or protein ?
- Arterial blood gas to assess acid base disturbance / Lactate
- USS Renal tract to exclude obstruction
- Renal biopsy rarely performed due to bleeding risk (complicates up to 1/3 of percutaneous renal Bxs in patients with cirrhosis; TJRenal Bx n=55 – 8 internal bleeding and 4 perinephric haematomas)

AKI stage 1 – Management

- Stop nephrotoxic drugs – NSAIDs, ACEI, gentamicin
- Reduce or stop diuretics, lactulose, antihypertensives (including Carvedilol)
- Volume expand with 1.5L 0.9% saline (in 500ml boluses) or 1g/kg albumin (maximum 100g/day) - aim for MAP > 80mmHg
- Rapid reversal of AKI over 48hrs suggests a volume responsive pre-renal AKI
- Responders - monitor UEs every 2/7 in hospital and every 2-4/52 during first 6/12 post discharge

AKI stage 2/3 – Management

- STOP diuretics, STOP Carvedilol

- Volume expand with 1.5L 0.9% saline (in 500ml boluses) or 1g/kg albumin (maximum 100g/day) - aim for MAP > 80mmHg
- If no response to volume expansion then commence vasoconstrictor (no difference between NA and Terlipressin in metanalysis of 4 RCTs – n =154) ∴ NA appropriate in ICU setting.
- Terlipressin - 1mg 6hrly increasing to a maximum of 2mg 4hrly - increase dose on day 3 if there has not been a 25% reduction in creatinine (EASL 2010). Continue for up to 14 days. Give in combination with 20% human albumin solution - 40 g (2 x 100ml bottles) daily following the initial 1g/kg for 2 days.
- Midodrine (alpha 1 adrenergic agonist)/ Octeotride combination a potential alternative to Terlipressin*

Non-responders

- Take advice from Renal team if renal function continues to deteriorate
- Renal replacement therapy should be considered in those that fulfil criteria for renal support (resistant hyperkalaemia, pulmonary oedema, pH < 7.25 and uraemic pericarditis) and where there is a reasonable prospect that liver synthetic function will recover or of future transplantation. Patients with a reversible factor such as sepsis or acute alcoholic hepatitis would be more likely to recover liver function
- Consider internal jugular CVP insertion under US guidance (consider whether you need to correct and INR > 1.5 or Platelets < 80) if patient in HDU/ICU setting. Aim CVP 8-12mmHg and administer 250ml boluses of crystalloid. The dynamic response of CVP to filling is more useful than absolute values especially in presence of tense ascites, which can falsely elevate JVP.
- In the presence of tense ascites perform limited 5L paracentesis (to minimise haemodynamic derangement) to relieve abdominal compartment syndrome and improve renal venous return.
- If hypotension/ reduced MAP - Echocardiogram to exclude cardiomyopathy.

Further reading

1. EASL clinical practice guidelines on management of ascites, spontaneous bacterial peritonitis and hepatorenal syndrome. Journal of Hepatology 2010; 53: 397-417
2. NICE clinical guideline 148. Acute Kidney injury. December 2019
3. AASLD practice guideline: Diagnosis, evaluation and management of ascites, SBP and HRS updated 2021
4. Baveno VII – Renewing consensus in portal hypertension 2022

Reference no.:

Documentation Controls (these go at the end of the document but before any appendices)

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	3	2022	Liver Management Group	Previous guideline expired
Intended Recipients: All clinicians managing patients with liver disease				
Training and Dissemination: Forms part of liver handbook which is disseminated to all clinicians rotating through Hepatology				
Development of Guideline: **Job Title: Dr A Austin, Dr N Taylor and Dr A Lawson (Consultant Hepatologist)**				
Consultation with: Liver management group				
Linked Documents: State the name(s) of any other relevant documents				
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