STANDARD OPERATING PROCEDURE

Management of Acute Kidney Injury (AKI) in Cirrhosis

Special Region (1)

Union of Myanmar

Version: 1.0

Effective Date: 23rd April 2026

Review Date: 23rd April 2028

Approved by: Internal Medicine

1. PURPOSE

To provide a standardized, evidence-based approach for diagnosis and management of AKI in cirrhosis, including hepatorenal syndrome.

2. SCOPE

Applicable to:

- All adult patients with **chronic liver disease/cirrhosis**
- Inpatient and emergency settings

3. DEFINITIONS

AKI is defined as:

- Increase in serum creatinine (SCr) ≥ 0.3 mg/dL within 48 hours
  OR
- Increase ≥ 50% from baseline within 7 days

AKI STAGING

Stage 1	↑ SCr ≥0.3 mg/dL or 1.5–2× baseline
Stage 2	↑ SCr 2–3× baseline
Stage 3	↑ SCr >3× baseline OR ≥4.0 mg/dL OR dialysis

TYPES OF AKI

Type	Key Features
Pre-renal	Volume depletion, diuretics, GI loss
HRS-AKI	Functional renal failure, no structural damage
ATN	Ischemia/sepsis, tubular injury
Post-renal	Rare (obstruction)

Hepatorenal syndrome (According to the International Club of Ascites)

HRS-AKI is defined as:

- Cirrhosis with ascites
- AKI
- No response after 2 days of albumin (1 g/kg/day)
- No shock
- No nephrotoxic drugs
- No structural kidney disease

4. Pathophysiology

Portal hypertension in cirrhosis leads to marked splanchnic vasodilation, primarily mediated by increased nitric oxide and other vasodilatory substances. This results in a reduction in effective arterial blood volume despite a normal or increased total blood volume. In response, compensatory neurohormonal systems—including the renin–angiotensin–aldosterone system (RAAS) and the sympathetic nervous system (SNS)—are activated, leading to intense renal vasoconstriction. In advanced stages, this persistent renal hypoperfusion without intrinsic structural kidney damage culminates in the development of hepatorenal syndrome (HRS).

RISK FACTORS for AKI are:

- Spontaneous bacterial peritonitis (SBP)
- Large-volume paracentesis without albumin

- Over-diuresis
- GI bleeding
- Sepsis
- Nephrotoxic drugs (NSAIDs, aminoglycosides)

5. INITIAL MANAGEMENT

Stop diuretics, stop nephrotoxins, treat infections, assess volume status.

6. ALBUMIN CHALLENGE

- Albumin 1 g/kg/day (max 100 g) for 2 days

Response:

- Improvement → Pre-renal AKI
- No improvement → suspect HRS-AKI or ATN

7. HRS MANAGEMENT

- Standard: Terlipressin 1 mg IV every 4–6 hours + Albumin 20–40 g/day
- Alternative: Noradrenaline or Midodrine + Octreotide.

Noradrenaline infusion protocol
4 mg noradrenaline in 50 mL NS or D5W, Concentration: 80 mcg/mL
Dosage and titration = 0.05–0.1 mcg/kg/min IV infusion, Increase by 0.02–0.05 mcg/kg/min every 15–30 minutes • Usual Dose Range = 0.1–0.7 mcg/kg/min
Target • Increase MAP by ≥10–15 mmHg OR • Achieve MAP ≥65–70 mmHg
Duration • Continue for 5–14 days • Stop when: ◦ Serum creatinine improves to near baseline ◦ No response after adequate trial
Concomitant Therapy • Albumin mandatory ◦ Day 1: 1 g/kg (max 100 g) ◦ Then: 20–40 g/day

Note – for simple infusion rate calculation of noradrenaline, please refer to cardiogenic shock SOP.

8. DIALYSIS INDICATIONS

Hyperkalemia, acidosis, fluid overload, uremia.

9. MONITORING

Daily creatinine, urine output, electrolytes, MAP.

10. REFERENCES

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