

REVIEW ARTICLE

Pre- and post-transplant renal complications in liver cirrhosis: Pathophysiology, risk factors, and management

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Renal dysfunction is a major determinant of morbidity, mortality, transplant candidacy, and post-transplant outcomes in cirrhosis. Acute kidney injury (AKI) develops frequently during hospitalization and is driven by circulatory dysfunction, portal hypertension, infection, gastrointestinal bleeding, diuretic exposure, nephrotoxins, and hepatorenal syndrome (HRS). Chronic kidney disease is increasingly recognized in this population and often reflects recurrent AKI, viral or alcohol-related glomerular disease, metabolic dysfunction-associated steatotic liver disease, diabetes, hypertension, or persistent renal hypoperfusion. Diagnosis remains difficult because serum creatinine underestimates renal impairment in cirrhosis. Urinalysis, renal ultrasonography, medication review, volume assessment, and selected biomarkers such as urinary neutrophil gelatinase-associated lipocalin, cystatin C, and tissue inhibitor of metalloproteinases-2/insulin-like growth factor-binding protein 7 may improve phenotyping, although biomarker-guided algorithms require further validation. Management before transplantation requires early discontinuation of nephrotoxins, careful volume resuscitation, albumin when indicated, timely treatment of infection, and vasoconstrictor therapy for HRS-AKI, with renal replacement therapy considered when standard indications arise. After liver transplantation, renal injury remains multifactorial and is influenced by pre-existing kidney disease, perioperative hemodynamic instability, ischemia-reperfusion injury, sepsis, and calcineurin inhibitor nephrotoxicity. Kidney-sparing immunosuppression, cardiovascular risk control, and structured follow-up are central to preserving renal function. Decisions regarding liver-alone transplantation, kidney-after-liver transplantation, or simultaneous liver-kidney transplantation should integrate the duration and reversibility of renal dysfunction, dialysis exposure, estimated glomerular filtration rate, and Organ Procurement and Transplantation Network Safety Net criteria. Accurate renal phenotyping across the pre- and post-transplant continuum is essential for improving survival and guiding transplant strategy.

Keywords: Liver cirrhosis; Liver transplantation; Acute kidney injury; Chronic kidney disease; Calcineurin inhibitors

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1. Introduction

Renal dysfunction is a frequent and clinically consequential complication of cirrhosis. Acute kidney injury (AKI) occurs in approximately 20–50% of hospitalized patients with cirrhosis and is a major contributor to morbidity, resource use, and mortality.^{1,2,3} It is also associated with higher rates of hospitalization, readmission, and both short- and long-term death.⁴

Patients with cirrhosis are predisposed to AKI because portal hypertension produces complex hemodynamic, inflammatory, and neurohormonal disturbances. These changes reduce effective arterial blood volume, impair renal perfusion, and increase susceptibility to renal functional decline. Bacterial infection, gastrointestinal bleeding, nephrotoxic medications, and large-volume paracentesis can further destabilize the circulation and precipitate AKI.⁵

Chronic kidney disease (CKD) is also increasingly recognized among patients with cirrhosis. Reported CKD rates have risen from approximately 1% in 2005 to 46.8% in 2019, underscoring a growing clinical and public health burden.^{6,7} The coexistence of CKD worsens outcomes by increasing the risk of superimposed AKI, dialysis requirement, bacterial infection, short- and long-term mortality, and the likelihood of needing liver transplantation.^{7,8}

Renal impairment also influences outcomes after liver transplantation. Pre-existing renal dysfunction is associated with postoperative complications and reduced survival after transplantation.¹ Early recognition, accurate phenotyping, and targeted management of renal dysfunction are therefore essential to optimize both pre-transplant stabilization and post-transplant recovery.

1.1. Scope and significance of the study

Renal complications in cirrhosis arise from overlapping hemodynamic, inflammatory, metabolic, infectious, and drug-related mechanisms. This review synthesizes the pathophysiology, diagnosis, prevention, and management of AKI and CKD before liver transplantation. It extends the discussion to post-transplant renal injury, including calcineurin inhibitor (CNI) nephrotoxicity, kidney-sparing immunosuppression, and decision-making for simultaneous liver-kidney transplantation (SLKT) versus liver-alone transplantation. The goal is to support earlier identification of reversible renal injury and more precise transplant planning.

1.2. Narrative review approach and search strategy

This article is a narrative review rather than a systematic review or meta-analysis. A targeted literature search

was conducted in PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar for English-language guidelines, consensus statements, prospective cohorts, systematic reviews, and major reviews published from January 2000 to May 2026. Search terms included “cirrhosis,” “liver transplantation,” “acute kidney injury,” “chronic kidney disease,” “hepatorenal syndrome,” “ATN,” “biomarkers,” “uNGAL,” “cystatin C,” “TIMP-2/IGFBP7,” “calcineurin inhibitor nephrotoxicity,” “simultaneous liver-kidney transplantation,” “SGLT2 inhibitors,” and “MASLD.” Priority was given to international guidance documents, transplant-focused evidence, and studies with clinically relevant diagnostic or management implications. Because the objective was to synthesize pathophysiology and clinical management for a broad readership, formal PRISMA screening, risk-of-bias scoring, and quantitative pooling were not performed.

2. Definition and diagnosis of renal impairment in pre-transplant patients

In 2015, the International Club of Ascites (ICA) adopted serum creatinine (SCr)-based AKI criteria for patients with cirrhosis, incorporating the 2012 Kidney Disease: Improving Global Outcomes framework with cirrhosis-specific application. AKI is defined as an increase in SCr of ≥ 0.3 mg/dL ($26.5 \mu\text{mol/L}$) within 48 hours, or an increase of $\geq 50\%$ from baseline that is known or presumed to have occurred within the preceding seven days. For the 2015 ICA-AKI definition and staging system, urine output is intentionally excluded from the formal diagnostic criteria because it is difficult to interpret in cirrhosis owing to ascites, diuretic exposure, and altered effective arterial blood volume. Nevertheless, oliguria remains clinically important for bedside risk assessment and is incorporated into newer Acute Disease Quality Initiative/ICA hepatorenal syndrome (HRS)-AKI criteria.⁹⁻¹²

Acute kidney injury is categorized into stages according to the magnitude of SCr elevation, with higher stages consistently associated with worse outcomes. A baseline SCr is needed for proper diagnosis of AKI. This should ideally be a stable measurement from within the past three months. Any documented measurement from the past 12 months can be used if this is not available. If previous values are unavailable, the initial SCr measurement on admission to hospital is recommended as the reference baseline.^{9,13,14} However, persistent structural and/or functional kidney damage (e.g., proteinuria, hematuria, imaging abnormalities, or glomerular filtration rate [GFR] < 60 mL/min) lasting longer than three months is known as CKD. The condition is categorized as acute kidney disease (AKD) if these results last more than a week and less than three months.¹⁵

2.1. Pathophysiology of renal impairment in patients with cirrhosis

Cirrhosis promotes portal hypertension through fixed structural resistance from hepatic fibrosis and dynamic intrahepatic vasoconstriction mediated by activated myofibroblasts and sinusoidal dysfunction. Portal hypertension and intestinal barrier disruption increase bacterial translocation and systemic inflammation, accompanied by elevated levels of mediators such as nitric oxide, interleukin-6, and tumor necrosis factor- α . These processes drive splanchnic vasodilation and reduce effective arterial blood volume.¹⁶⁻¹⁸

Splanchnic pooling decreases renal perfusion pressure. Initially, sympathetic activation and increased cardiac output help preserve kidney blood flow, but these compensatory responses fail as cirrhosis decompensates. Subsequent activation of the renin-angiotensin-aldosterone system promotes sodium and water retention, leading to ascites and edema. Cirrhotic cardiomyopathy further limits the cardiovascular response to stress, so bleeding, infection, aggressive diuresis, or nephrotoxin exposure can rapidly precipitate AKI.^{1,17-19}

Chronic kidney disease in cirrhosis frequently follows recurrent or incompletely resolved AKI. Although the mechanisms linking AKI to CKD remain incompletely defined, persistent tubular injury, microvascular dysfunction, inflammation, and maladaptive repair are likely contributors. In one cohort, approximately 25% of patients with advanced cirrhosis who developed AKI and survived beyond three months developed CKD, compared with 1% of those without AKI.⁸

Viral hepatitis, alcohol-related liver disease, and immune-complex glomerular injury may directly contribute to structural kidney disease, including membranoproliferative glomerulonephritis, membranous nephropathy, and IgA nephropathy.²⁰ The growing burden of metabolic dysfunction-associated steatotic liver disease, particularly when combined with diabetes, hypertension, and dyslipidemia, further increases CKD risk.²¹⁻²³

2.2. Causes and diagnosis of acute kidney injury in cirrhosis (Pre-Transplant Setting)

Acute kidney injury in cirrhosis is commonly classified as pre-renal, intrinsic renal, or post-renal, although mixed phenotypes are frequent in advanced disease.

Pre-renal AKI is common and may follow variceal hemorrhage, vomiting, diarrhea, overdiuresis, or other causes of intravascular volume depletion. HRS-AKI is a distinct functional phenotype characterized by intense renal vasoconstriction in the setting of systemic and

splanchnic vasodilation.²⁴

Intrinsic renal causes include acute tubular necrosis (ATN), usually from ischemia, sepsis, or nephrotoxin exposure, and glomerular disease related to viral hepatitis, alcohol-associated liver disease, or immune-complex deposition. Tubulointerstitial nephritis may also occur after infections or drug exposure. Post-renal AKI is uncommon in cirrhosis and generally reflects urinary tract obstruction from malignancy, benign prostatic hyperplasia, or other obstructive processes.^{24,25} In a retrospective study of 423 patients with cirrhosis and AKI, pre-renal azotemia and ATN accounted for more than 80% of cases, whereas post-renal etiologies represented fewer than 0.5%.²⁵ A prospective study of liver transplant candidates found pre-renal injury to be the most frequent cause, whereas post-renal causes were not identified.²⁶

2.3. Hepatorenal syndrome

Hepatorenal syndrome is a functional renal impairment phenotype that occurs in advanced cirrhosis with ascites. Updated consensus criteria define HRS-AKI using the following elements:

- (i) Diagnosis of cirrhosis with ascites.
- (ii) Increase in serum creatinine (SCr) ≥ 0.3 mg/dL (26.5 μ mol/L) within 48 hours, or $\geq 50\%$ from baseline within seven days, and/or urine output < 0.5 mL/kg/h for > 6 hours.
- (iii) No clinical improvement in urine output or SCr after 24 hours of adequate volume expansion when volume expansion is clinically indicated.
- (iv) No clear alternative explanation for renal impairment, including structural kidney disease.
- (v) Albumin administration is recommended during diagnostic evaluation but is no longer an absolute diagnostic requirement for HRS-AKI.¹²

Hepatorenal syndrome terminology is time-based. HRS-AKI refers to hepatorenal syndrome meeting AKI criteria; HRS-AKD refers to hepatorenal syndrome with acute kidney disease persisting for > 7 days but < 3 months; and HRS-CKD refers to hepatorenal syndrome with CKD lasting ≥ 3 months. This distinction separates the acuity of renal dysfunction from the underlying cirrhosis-associated HRS physiology and avoids conflating HRS-AKI with AKD.¹²

2.4. Diagnosis and management of renal dysfunction in patients with cirrhosis

Evaluation of AKI in cirrhosis should begin with a structured assessment of intravascular volume, hemodynamic stability, infection, bleeding, ascites burden, and recent medication exposure. Hypovolemia

from diarrhea, vomiting, gastrointestinal bleeding, or excessive diuresis requires prompt but individualized fluid resuscitation. Renal ultrasonography should be used to exclude obstruction and to identify structural features suggestive of CKD, although post-renal causes are rare in this population.²⁶

Urinalysis is required to identify proteinuria, hematuria, or active sediment suggestive of intrinsic renal disease. Biomarkers such as urinary neutrophil gelatinase-associated lipocalin (uNGAL) may help distinguish ATN from pre-renal azotemia or HRS-AKI, but their routine use in cirrhosis remains investigational and should be interpreted in clinical context.²⁷

Management should be phenotype directed. Nephrotoxic medications should be stopped promptly.²⁸ Fluid therapy should be guided by clinical volume assessment, oliguria, and dynamic responsiveness testing such as passive leg raise or small fluid challenge. Blood products are appropriate for hemorrhage, balanced crystalloids for volume depletion, and albumin for spontaneous bacterial peritonitis or suspected HRS-AKI.²⁷⁻²⁹

Albumin offers oncotic and potential immunomodulatory effects, but randomized trials have not consistently shown superiority over crystalloids for mortality or renal recovery.³⁰ In tense ascites, large-volume paracentesis with albumin replacement of 6–8 g per liter removed beyond 5 L can reduce intra-abdominal pressure and support renal perfusion.³¹

For HRS-AKI, terlipressin or norepinephrine with albumin is recommended when not contraindicated. Meta-analyses suggest comparable efficacy for HRS reversal, although terlipressin may provide additional benefit in selected patients with acute-on-chronic liver failure.³²⁻³⁵ Treatment should target a mean arterial pressure increase of at least 15 mmHg, as this response is associated with improved SCr and possible HRS reversal.³⁶ Patients require close monitoring for respiratory failure, volume overload, and ischemic events.^{37, 38}

Because transplant-free survival remains poor even after HRS-AKI reversal, early liver transplant referral is essential. Vasoconstrictor therapy should be considered a bridge to definitive transplant evaluation or recovery when feasible.³⁹

2.5. Prevention of acute kidney injury in cirrhosis

Prevention is central because AKI in cirrhosis is associated with high morbidity and mortality. Strategies include maintaining euvoolemia, avoiding excessive diuresis or laxative use during hypovolemia, minimizing exposure

to nephrotoxins, and promptly treating infection. Contrast-induced nephropathy remains a concern, but N-acetylcysteine and bicarbonate have not consistently prevented AKI in this setting.¹²

Albumin infusion after large-volume paracentesis is recommended at 6–8 g per liter of ascites removed beyond 5 L to reduce post-paracentesis circulatory dysfunction.¹² Red blood cell transfusion is generally recommended when hemoglobin falls below 7 g/dL to maintain oxygen delivery while avoiding unnecessary volume overload.¹²

In selected patients, transjugular intrahepatic portosystemic shunt (TIPS) may improve portal hemodynamics and renal perfusion, although candidacy must be assessed in light of the risks of encephalopathy, cardiac reserve, and liver function.¹² For spontaneous bacterial peritonitis, albumin combined with antibiotics reduces renal complications and mortality. Albumin dose and duration should be individualized using daily volume and hemodynamic assessment.¹²

For septic shock due to infections other than spontaneous bacterial peritonitis, a mean arterial pressure target above 65 mmHg is recommended. Balanced crystalloids such as lactated Ringer's solution or PlasmaLyte are appropriate for initial resuscitation, while albumin should be used cautiously to avoid pulmonary edema and volume overload.¹²

3. Renal replacement therapy

Renal replacement therapy (RRT) should be considered when renal failure progresses despite optimized medical management (Table 1). Indications are similar to those in patients without cirrhosis and include refractory acidosis, severe electrolyte disturbance, fluid overload, and uremic complications.⁹ Continuous RRT is preferred in hemodynamically unstable patients because it limits intradialytic hypotension and permits gradual correction of metabolic derangements.⁴⁰

Anticoagulation during continuous RRT is challenging in cirrhosis. Although regional citrate anticoagulation is favored in many critically ill patients because it reduces bleeding risk, impaired citrate metabolism in liver failure may increase the risk of hypocalcemia and acid-base disturbances; therefore, monitoring and individualized selection are required.^{39,41,42}

Peritoneal dialysis is rarely used in cirrhosis but may offer hemodynamic stability and ascites control in selected patients. Its limitations include hypoalbuminemia and increased peritonitis risk.^{43,44} Prognosis remains poor in patients with cirrhosis requiring RRT, with six-month mortality often ranging from 60% to 80%.⁴⁵

Early involvement of palliative care is recommended to support symptom control and goals-of-care discussions.^{46,47} A time-limited trial of RRT may be appropriate as a bridge to decision-making, transplant candidacy, or family preparation, depending on reversibility and liver transplantation candidacy.

In patients with refractory ascites and portal hypertension, TIPS has been associated with improved glomerular filtration rate in observational studies. Although randomized evidence is limited, selected patients with HRS-AKI or resistant portal hypertensive complications may benefit from TIPS as a bridge to transplantation or as a strategy to reduce recurrent AKI risk.⁴⁸⁻⁵⁰

4. Spectrum, mechanisms, and diagnostic challenges of renal disease in end-stage liver disease

Renal dysfunction in end-stage liver disease spans reversible functional injury, such as HRS, structural injury

such as ATN, and progression to CKD. HRS reflects intense renal vasoconstriction driven by splanchnic vasodilation and systemic circulatory dysfunction, whereas ATN results from ischemia, sepsis, or exposure to nephrotoxins. CKD increasingly reflects cumulative injury from recurrent AKI, diabetes, hypertension, metabolic disease, and sustained neurohormonal activation. Portal hypertension amplifies renal vulnerability through systemic inflammation, endothelial dysfunction, and impaired renal autoregulation.⁵²

Differentiating HRS-AKI, pre-renal azotemia, ATN, and established CKD remains difficult. SCr underestimates renal impairment in cirrhosis because of sarcopenia, reduced creatine production, and altered hepatic metabolism. uNGAL has shown diagnostic value (Table 2) for ATN in cirrhosis; in a previous study, an uNGAL cutoff of 220 ng/mL yielded 89% sensitivity and 78% specificity, with an area under the receiver operating characteristic curve of 0.854 for ATN-AKI.^{51,53} Cystatin C provides a creatinine-independent estimate of GFR, while tissue

Table 1. Comparative overview of major renal dysfunction types in cirrhosis

Type of renal dysfunction	Key pathophysiology	Diagnostic features	Management approach	Prognosis
Pre-renal (volume depletion)	Reduced effective blood volume from hemorrhage, overdiuresis, or diarrhea; preserved renal parenchyma.	Rapid improvement with volume repletion; bland urinalysis.	Volume repletion with albumin or crystalloids; stop nephrotoxins.	Good if identified early. ^{1,5,9}
HRS-AKI	Severe renal vasoconstriction with systemic and splanchnic vasodilation; primarily functional injury.	No response to adequate volume correction; bland urine; low urine sodium may be present.	Albumin plus vasoconstrictor therapy when indicated; urgent transplant evaluation.	Poor unless reversed or transplanted. ^{9,12,32}
ATN	Ischemic or toxic tubular injury, commonly after sepsis, hypoperfusion, or nephrotoxin exposure.	Muddy brown casts; higher uNGAL; fractional excretion of sodium may be >2%.	Remove nephrotoxins; supportive care; RRT when standard indications arise.	Moderate to poor; high mortality in cirrhosis. ^{24,51}
CKD	Cumulative injury from recurrent AKI, diabetes, hepatitis, MASLD, and persistent renal hypoperfusion.	GFR <60 mL/min/1.73 m ² for >3 months, proteinuria, or imaging abnormalities.	Compensated proteinuric CKD: ACE inhibitor/ARB only with close BP, potassium, and SCr monitoring. Decompensated ascites, hypotension, or active AKI: avoid RAAS blockade. Prevent AKI and plan transplant strategy.	Worsens transplant candidacy and long-term outcomes. ^{7,20,21}
Post-renal (rare)	Mechanical obstruction, such as BPH or malignancy.	Hydronephrosis on ultrasound or elevated post-void residual.	Relieve obstruction.	Excellent if resolved early. ^{24,25}

Abbreviations: ACE: Angiotensin-converting enzyme; AKI: Acute kidney injury; ARB: Angiotensin receptor blocker; ATN: Acute tubular necrosis; BPH: Benign prostatic hyperplasia; BP: Blood pressure; CKD: Chronic kidney disease; GFR: Glomerular filtration rate; HRS-AKI: Hepatorenal syndrome–acute kidney injury; MASLD: Metabolic dysfunction–associated steatotic liver disease; RAAS: Renin–angiotensin–aldosterone system; RRT: Renal replacement therapy; SCr: Serum creatinine; uNGAL: Urinary neutrophil gelatinase-associated lipocalin.

Table 2. Comparative overview of selected renal biomarkers in cirrhosis and liver transplantation

Biomarker	Biological signal and main role	Representative cutoff/performance	Differential performance and clinical recommendation
uNGAL	Tubular injury marker; best-supported adjunct for identifying structural tubular injury.	ATN-AKI cutoff 220 ng/mL: sensitivity 89%, specificity 78%, AUROC 0.854; early AKI prediction cutoff 84.8 µg/gCr reported in decompensated cirrhosis. ^{53,55}	Highest in ATN, intermediate in HRS-AKI, lowest in pre-renal AKI. Use as an adjunct when HRS-AKI vs ATN is unclear; do not use as a stand-alone diagnostic test because infection and inflammation may elevate levels.
Cystatin C	Creatinine-independent functional marker of GFR; less affected by sarcopenia than SCr.	Cystatin C-based GFR <55 mL/min/1.73 m ² has been reported for early AKI recognition; admission cystatin C independently predicted AKI in decompensated cirrhosis. ⁵⁵	Useful for renal risk stratification and refining GFR estimates; limited specificity for ATN vs HRS-AKI. Consider with SCr trends, MELD context, and clinical trajectory.
TIMP-2/IGFBP7	Cell-cycle arrest marker reflecting tubular stress and risk of moderate-to-severe AKI.	FDA-cleared for risk assessment of stage 2–3 AKI in critically ill patients; cirrhosis-specific performance is inconsistent, with stronger rationale in perioperative liver transplantation. ^{54,55}	May support perioperative AKI prediction, but evidence is insufficient for routine etiologic phenotyping in cirrhosis. Use remains investigational outside validated institutional protocols.
Combined panel	Integrates biomarker data with urinalysis, microscopy, renal ultrasonography, hemodynamics, infection status, and medication exposure.	No universally validated cutoff or algorithm in cirrhosis.	Most clinically promising strategy for future studies. Recommended only as an adjunct to clinical judgment until prospective cirrhosis-specific algorithms are validated.

Abbreviations: AKI: Acute kidney injury; ATN: Acute tubular necrosis; AUROC: Area under the receiver operating characteristic curve; FDA: Food and Drug Administration; GFR: Glomerular filtration rate; HRS-AKI: Hepatorenal syndrome–acute kidney injury; IGFBP7: Insulin-like growth factor-binding protein 7; MELD: Model for End-stage Liver Disease; SCr: Serum creatinine; TIMP-2: Tissue inhibitor of metalloproteinases-2; uNGAL: Urinary neutrophil gelatinase-associated lipocalin.

inhibitor of metalloproteinases-2/insulin-like growth factor-binding protein 7 may identify tubular stress and imminent AKI, especially in perioperative settings. These biomarkers remain context-dependent because infection, inflammation, and drug-related injury may confound interpretation.^{54,55}

Functional assessment may add value. A furosemide stress test, defined by urine output <200 mL within two hours, can identify patients at risk of AKI progression, and renal Doppler ultrasonography with a resistive index >0.79 may suggest intrinsic injury.⁵⁶ No single biomarker or functional test has replaced clinical judgment. Multimodal approaches that combine biomarkers, dynamic testing, imaging, and clinical trajectory data may improve transplant timing and SLKT decision-making, but prospective validation in cirrhotic cohorts remains necessary.^{57,58}

5. Post-transplant renal complications: Acute kidney injury and chronic kidney disease after liver transplantation

Acute kidney injury after liver transplantation is common and strongly influences morbidity, mortality, progression

to CKD, and quality of life. It is usually multifactorial, reflecting pre-transplant renal vulnerability, intraoperative hemodynamic events, ischemia-reperfusion injury, infection, and post-transplant nephrotoxin exposure. Early recognition of reversible causes and prevention of cumulative injury may improve both survival and long-term renal outcomes.^{59–61}

In the transplant population, primary and secondary mechanisms of AKI often overlap. Before transplantation, infection is a major precipitant of AKI in end-stage liver disease, followed by volume depletion, pre-renal azotemia, and HRS-AKI. Refractory ascites, large-volume paracentesis, and intensive diuretic exposure further increase vulnerability.⁵⁹

Pre-transplant characteristics shape postoperative renal risk. Prior AKI or CKD, obesity, hypertension, diabetes mellitus, viral hepatitis, alcohol-related liver disease, hepatocellular carcinoma, and high Model for End-stage Liver Disease (MELD) scores are associated with increased post-transplant renal dysfunction. Donor factors, including older donor age, marginal graft quality, and prolonged cold or warm ischemia times, may worsen ischemia-reperfusion injury and delay graft recovery.⁶¹

The intraoperative period is a critical window for renal injury. Hypotension and hemodynamic instability reduce renal perfusion, while post-reperfusion syndrome triggers abrupt cardiovascular changes and the release of inflammatory mediators. Substantial blood loss, portal hypertensive coagulopathy, and large volume transfusion expose renal tubules to additional ischemic and hemoglobin/iron-mediated stress. Intraoperative oliguria and glycemic variability may also signal or contribute to early renal injury.⁶¹

Postoperative factors determine whether AKI resolves or progresses to CKD. CNI nephrotoxicity is a leading cause of post-transplant renal dysfunction. Early toxicity results largely from afferent arteriolar vasoconstriction and reduced GFR, whereas concomitant aminoglycosides, antifungals, nonsteroidal anti-inflammatory drugs, iodinated contrast, infection, and sepsis amplify renal injury.⁶¹

Calcineurin inhibitors have improved patient and graft survival after transplantation, but nephrotoxicity remains clinically important. Early CNI nephrotoxicity, typically within the first year, is mediated by afferent arteriolar vasoconstriction and tubular cell injury and may improve after dose reduction or discontinuation. Chronic CNI nephrotoxicity, usually developing after one year, is often irreversible and characterized by arteriolar hyalinosis, glomerulosclerosis, tubular atrophy, and interstitial fibrosis. Tacrolimus and cyclosporine can also rarely precipitate thrombotic microangiopathy; in such cases, conversion to non-CNI immunosuppression should be considered.⁶⁰

Long-term CKD after transplantation is driven by chronic CNI exposure, metabolic complications, cardiovascular risk factors, and persistent urinary abnormalities. Hypertension, post-transplant diabetes mellitus, obesity, hyperlipidemia, and metabolic syndrome independently accelerate renal decline. Steroids and CNIs increase systemic vascular resistance and activate the renin-angiotensin-aldosterone system, while tacrolimus may impair pancreatic beta-cell function and increase the risk of diabetes, although it generally causes less hypertension than cyclosporine.⁶¹ Post-transplant metabolic dysfunction-associated steatotic liver disease and metabolic dysfunction-associated steatohepatitis add systemic inflammation and endothelial dysfunction, further contributing to kidney injury. Persistent proteinuria or active urinary sediment should prompt evaluation for diabetic nephropathy, thrombotic microangiopathy, or other intrinsic renal disease.⁶¹

6. Treatment and prevention of acute kidney injury and chronic kidney disease after liver transplantation

The immediate post-transplant period is a vulnerable interval for kidney injury. Management should prioritize adequate renal perfusion, avoidance of intravascular volume depletion, judicious use of blood products, prevention of postoperative bleeding, and avoidance or minimization of nephrotoxic medications. Meticulous surgical hemostasis before abdominal closure remains important because postoperative bleeding can precipitate renal hypoperfusion and AKI.⁶⁰

Management of AKI after transplantation includes identifying reversible causes, optimizing hemodynamics, and treating HRS-AKI when it persists or recurs. Terlipressin with albumin may be used according to response-guided dosing, with discontinuation when no meaningful creatinine response occurs after an adequate treatment period. RRT is indicated for standard indications such as refractory hyperkalemia, acidosis, uremia, or uncontrolled fluid balance. Continuous modalities are preferred when hemodynamic instability or severe hyponatremia is present.⁶¹

Post-transplant CKD management should focus on modifiable risk factors. Blood pressure targets are <130/80 mmHg in patients with proteinuria and <140/90 mmHg without proteinuria. Dihydropyridine calcium-channel blockers are often preferred because they counter CNI-mediated vasoconstriction. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers should generally be avoided early after transplantation because of low renin activity and the risk of hyperkalemia, but they may be appropriate later in patients with diabetes, proteinuria, or established CKD. Glycemic control should target HbA1c <7%, with insulin favored during high-dose corticosteroid exposure. Sodium-glucose cotransporter 2 inhibitors may support weight and glycemic control after transplantation in selected recipients. Pravastatin and fluvastatin are commonly preferred among statins with CNIs because of their fewer cytochrome P450 interactions.⁶¹

Non-pharmacologic measures, including dietary sodium restriction (<2 g/day), daily physical activity (>30 minutes), and individualized protein intake, support blood pressure and proteinuria control while preserving nutritional status.⁶¹

6.1. Kidney-sparing immunosuppressive strategies

Kidney-sparing immunosuppressive strategies seek to reduce CNI-related nephrotoxicity without compromising

rejection prophylaxis. Their effectiveness varies depending on the timing after transplantation.⁶⁰

6.1.1. 0–1-month post-transplant

During the first month, delayed initiation of CNIs with antibody-based induction therapy, including antithymocyte globulin or CD25-targeted agents such as basiliximab or daclizumab, may reduce early CNI exposure. This strategy appears most useful in recipients with preserved pre-transplant kidney function, whereas benefit is less consistent in those with pre-existing renal impairment or early post-transplant AKI.⁶⁰

6.1.2. 1–12 months post-transplant

Between 1 and 12 months, CNI minimization or CNI-free strategies show clearer renal benefit. Reduced CNI exposure combined with mycophenolate mofetil can improve renal function without increasing rejection or mortality in selected patients. Mechanistic or mammalian target of rapamycin inhibitors, such as everolimus and sirolimus, may also allow CNI dose reduction; everolimus combined with reduced-dose CNIs has demonstrated sustained GFR benefits in several studies.⁶⁰

6.1.3. Beyond 12 months post-transplant

Beyond 12 months, evidence for conversion away from CNIs is less consistent. Late conversion to sirolimus alone has not reliably improved GFR and may increase the risk of rejection. In contrast, mycophenolate mofetil combined with low-dose CNI has improved GFR in some randomized trials and meta-analyses, although the risk of rejection remains higher when CNI exposure is substantially reduced or withdrawn. Decisions should therefore be individualized according to renal trajectory, rejection risk, graft function, and comorbidity profile.⁶⁰

6.2. Clinical impact on patient outcomes

Declining kidney function after liver transplantation increases morbidity, mortality, cardiovascular complications, CKD progression, and risk of end-stage renal disease.^{59, 61} Progression to dialysis-dependent kidney failure is associated with poorer graft survival and reduced overall survival, underscoring the importance of prevention, early recognition, and timely kidney transplant evaluation when recovery is unlikely.⁶⁰

6.3. Renal replacement therapy after liver transplantation

Renal replacement therapy remains an important supportive therapy for AKI in end-stage liver disease and after transplantation. Its use should be individualized based on AKI timing, underlying etiology, hemodynamic status,

transplant candidacy, and the probability of renal recovery. Although survival is poor among patients requiring RRT, it may provide essential support while candidacy, reversibility, or kidney-after-liver transplantation options are assessed.⁵⁹

6.4. Simultaneous liver-kidney transplantation versus liver-alone transplantation

Renal dysfunction is common in end-stage liver disease and may reflect hypovolemia, HRS-AKI, portal hypertension, intrinsic kidney disease, or progression to CKD. SLKT may improve survival in carefully selected candidates compared with liver transplantation alone, but inappropriate use may expose scarce kidney grafts to avoidable risk.⁶²

The 2017 Organ Procurement & Transplantation Network policy defines SLKT eligibility using CKD, sustained AKI, or qualifying metabolic kidney disease. For CKD, candidates should have an estimated GFR ≤ 30 mL/min or be on regular dialysis. For sustained AKI, eligibility requires dialysis at least once weekly for six consecutive weeks, estimated GFR ≤ 25 mL/min for six consecutive weeks, or a combination of these criteria over the same duration.⁶²

The Safety Net policy prioritizes kidney transplantation for liver-alone recipients who develop dialysis dependence or estimated GFR ≤ 20 mL/min/1.73 m² between two months and one year after liver transplantation. Early evaluations have shown lower waitlist mortality for kidney-after-liver candidates, increased kidney-after-liver transplantation, and favorable post-transplant survival, although patients who die before qualifying or remain too critically ill may be underrepresented. Multidisciplinary assessment and vigilant follow-up are therefore essential.⁶⁰

After MELD-based allocation, end-stage renal disease after liver transplantation has increased as transplant volume and survival have improved. End-stage renal disease after liver transplantation carries substantial morbidity and mortality, and patients who remain dialysis dependent after liver transplantation have worse graft and patient survival than those receiving kidney transplantation. Timely kidney transplant evaluation is therefore important, particularly outside the Safety Net window.⁶²

Liver transplantation alone followed by kidney-after-liver transplantation has been used for patients who develop irreversible kidney failure, including CNI-related nephrotoxicity. Kidney-after-liver transplantation may reduce perioperative morbidity and expand access to deceased or living kidney donors. Registry analyses have shown trade-offs: some studies suggest shorter kidney graft half-life and lower rejection-free survival compared

with SLKT, whereas more recent data indicate that SLKT in candidates with very high MELD scores may increase early kidney allograft failure.⁶³

In high-risk recipients, particularly those with MELD scores >30, SLKT may be associated with inferior kidney graft survival and higher primary non-function because perioperative hypotension, vasopressor exposure, and systemic instability compromise early kidney graft function.⁶³

Delayed kidney transplantation after liver-alone transplantation, including temporary hypothermic machine perfusion until hemodynamic stabilization, has been explored. Although this approach increases cold ischemia time and the risk of delayed graft function and may reduce one-year estimated GFR, selected high-risk patients have shown improved one- and five-year survival compared with simultaneous kidney transplantation.⁶³

Overall, renal outcomes after liver transplantation alone depend on perioperative instability, MELD severity, reversibility of kidney injury, and timing of kidney transplantation. In selected high-risk candidates, delayed kidney-after-liver transplantation may provide better patient survival than SLKT, while the Safety Net policy supports timely kidney access for recipients who demonstrate persistent irreversible renal failure.^{62,63}

7. Limitations and real-world clinical applicability

This review provides a concise overview of the potential causes of kidney dysfunction in liver disease, both pre- and post-liver transplant, to help clinical care providers make informed decisions. Although the emerging biomarkers discussed in this article are effective tools, their applicability remains limited due to high costs and availability only in specialized centers, hindering practical use.

8. Conclusion

Renal dysfunction in cirrhosis is a central determinant of prognosis rather than a passive consequence of liver failure. Portal hypertension, systemic inflammation, circulatory dysfunction, infection, nephrotoxin exposure, and metabolic comorbidity interact to produce a continuum that includes pre-renal injury, HRS-AKI, ATN, and CKD. Diagnosis remains limited by the inaccuracy of SCr and by overlapping clinical phenotypes, while biomarker and functional testing approaches remain promising but insufficiently standardized. Management before transplantation requires rapid correction of reversible precipitants, careful volume strategy, early infection control, selective albumin and vasoconstrictor therapy,

and timely transplant referral. After liver transplantation, renal outcomes are shaped by pre-existing kidney disease, perioperative instability, ischemia-reperfusion injury, sepsis, and CNI nephrotoxicity. Kidney-sparing immunosuppression and rigorous cardiovascular and metabolic risk control may preserve renal function but must be balanced against the risk of rejection. Decisions regarding liver-alone transplantation, kidney-after-liver transplantation, and SLKT should be individualized based on renal trajectory, dialysis duration, reversibility, the Organ Procurement & Transplantation Network criteria, and multidisciplinary judgment. A continuum-based approach to renal phenotyping across the pre- and post-transplant period is therefore essential to improve survival and optimize transplant strategy.

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Conflict of interest

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Author contributions

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Further disclosure

We used Grammarly AI to polish the language. Content was not generated by AI. Authors take full responsibility for the content.

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