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REVIEW

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Ascites in patients with end-stage renal disease: Challenges and solutions from diagnosis to management

Akash Roy[1] | **Anand V. Kulkarni**[2]

[1]Department of Hepatology, Institute of Gastrosciences and Liver Transplantation, Apollo Multispeciality Hospitals, Kolkata, India

[2]Department of Hepatology, AIG Hospitals, Hyderabad, India

Correspondence
Anand V. Kulkarni, Department of Hepatology and Liver Transplantation, AIG Hospitals, Hyderabad 500032, India.
Emails: anandvk90@gmail.com; dranand.v@aighospitals.com

Abstract

Metabolic diseases have exponentially increased in recent years, which has led to an increased prevalence of metabolic dysfunction–associated steatotic liver disease and concomitant kidney diseases. Ascites are a common presentation of cirrhosis, and renal impairment in cirrhosis is well described. However, patients with end-stage renal disease (ESRD) may also present with ascites even in the absence of cirrhosis. The literature on the management of patients with ESRD with ascites with or without concomitant cirrhosis is limited. Massive ascites in this population are often refractory to medical therapy and are associated with dismal prognosis. Pathophysiologically, increased hepatic vein hydrostatic pressure, fluid retention, increased peritoneal membrane permeability, and impaired peritoneal lymphatic drainage are proposed mechanisms for ascites in ESRD without cirrhosis. Identifying underlying cirrhosis and portal hypertension (PH) has therapeutic implications in such patients. However, diagnostic tools such as serum ascites albumin gradient and noninvasive tests to identify cirrhosis have limited utility in ESRD. Hemodialysis and continuous ambulatory peritoneal dialysis are effective but can be associated with hemodynamic compromise and peritonitis, especially in those with PH. TIPS for ascites has a limited role in the presence of ESRD due to the increased risk of HE. Kidney transplant is the treatment of choice in ESRD with ascites without PH. Simultaneous liver–kidney transplant remains the definitive treatment in the presence of PH, but is less commonly feasible, and kidney transplant alone in the presence of PH can be associated with the risk of decompensations. This review discusses the approach and management of ascites in chronic kidney disease and ESRD specifically.

Keywords: ascites, chronic kidney disease, portal hypertension

Abbreviations: AKI, acute kidney injury; CKD, chronic kidney disease; ESRD, end-stage renal disease; KT, kidney transplant; SAAG, serum ascites albumin gradient; SLKT, simultaneous liver–kidney transplant.

INTRODUCTION

Ascites refers to the pathological accumulation of fluid in the peritoneal cavity. The causes of ascites vary depending on the context where it is studied. Malignant ascites are more common in oncology settings, while infectious causes like tuberculosis predominate in infectious disease units. However, overall, cirrhosis is the most common cause of ascites, accounting for 80%–85% of the cases.[1] Patients with cirrhosis and ascites frequently are complicated by renal dysfunction, spanning a wide spectrum of acute kidney injury (AKI) and acute kidney disease to chronic kidney disease (CKD).[2,3] The common causes of renal dysfunction include diuretics, hypovolemia, hepatorenal syndrome, acute tubular necrosis due to sepsis, hyperbilirubinemia, and rarely post-renal obstruction. The approach and management of these patients with cirrhosis/chronic liver disease (CLD) and ascites who develop renal injury is well known and has clear recommendations.[3] However, it is not uncommon for patients with end-stage renal disease (ESRD) to present with ascites, and such patients are frequently referred to gastroenterologists/hepatologists when planning for kidney transplant (KT).[4] Furthermore, with the rising burden of lifestyle-related diseases such as metabolic dysfunction–associated steatotic liver disease, hypertension, and diabetes mellitus, the incidence of kidney and liver diseases would exponentially increase in the near future, meriting simultaneous liver–kidney transplants (SLKTs), which is less feasible in clinical practice.[5] The magnitude of the increasing burden is evidenced by the fact that almost 45% of non-electively hospitalized patients with cirrhosis fit the criteria of CKD, with those having CKD being older and having metabolic dysfunction–associated steatotic liver disease the dominant etiology in contrast to the previous reports of 13%–17% prevalence of CKD.[5,6] Further, the number of SLKTs performed in recent years has increased by 5–8 times.[7] Additionally, after an episode of AKI, it is estimated that almost 1 in 4 cirrhosis patients progress to CKD in 3 months.[8] On the other hand, the prevalence of cirrhosis in patients with CKD, specifically those on dialysis, is around 5%, as reported in a systematic review of over 7000 studies.[9]

Thus, although it is assuring to know that the burden of viral hepatitis B and C, which is known to have unique kidney manifestations (membranous and membranoproliferative glomerulonephritis), is reducing, due to vaccination strategies, universal precautions, and effective antivirals, the burden of kidney disease especially with the growing prevalence of metabolic dysfunction–associated steatotic liver disease is on the rise.[10,11] Table 1 shows the common causes of simultaneous liver and kidney dysfunction in clinical practice. Understanding the pathophysiology and appropriate management of such patients with renal disorders and ascites may help guide management and improve the outcomes. There remain key gray zones and knowledge gaps in the understanding, diagnosing, and managing ascites in patients with renal disease.[12] In this review, we discuss the pathophysiology, differential diagnosis, diagnostic approach, and management options and focus on the unique challenges of evaluating patients with renal disorders and ascites.

PATHOPHYSIOLOGY

The pathophysiology of ascites in cirrhosis is primarily driven by sinusoidal portal hypertension (PH) with

TABLE 1 Common causes of simultaneous kidney and liver dysfunction

Cause	Clinical/laboratory diagnosis
Liver dominant	
Cirrhosis due to any etiology with HRS–CKD	ICA-ADQI criteria
Alcohol-associated liver disease with IgA nephropathy	H/o alcohol use Hematuria
Chronic HBV and HCV associated with glomerulonephritis	History of HBV and HCV infection Biopsy evidence of glomerulonephritis
MASLD with diabetic/hypertensive kidney disease	History of obesity, metabolic diseases
Wilson disease with renal tubular acidosis, Fanconi syndrome, nephrolithiasis (due to hypercalciuria), penicillamine-related proteinuria	Urine examination
Kidney dominant	
ADPKD and PCLD	Imaging
Amyloidosis	Hepatomegaly Kidney biopsy, transjugular liver biopsy, abdominal fat pad examination.
Systemic lupus erythematosus and liver involvement due to SLD, AIH, PBC, and NRH	Autoimmune serology History of steroid for SLD/azathioprine intake for NRH

Abbreviations: ADPKD, autosomal dominant polycystic kidney disease; ADQI, Acute Dialysis Quality Initiative; AIH, autoimmune hepatitis; HRS–CKD, hepatorenal syndrome–chronic kidney disease; ICA, International Club of Ascites; MASLD, metabolic dysfunction–associated steatotic liver disease; NRH, nodular regenerative hyperplasia; PBC, primary biliary cholangitis; PCLD, polycystic liver disease; SLD, steatotic liver disease.

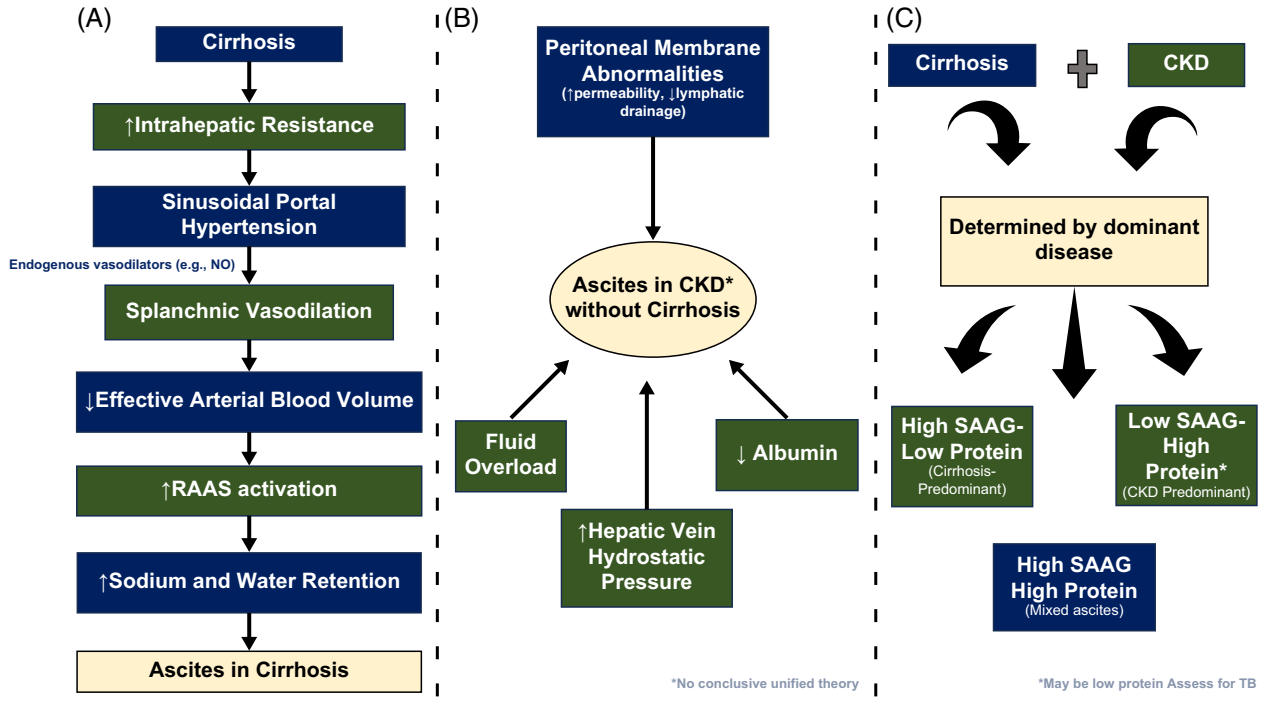

FIGURE 1 Pathophysiological processes involved in ascite formation are: (A) cirrhosis, (B) chronic kidney disease, and (C) concomitant pathologies. Abbreviations: CKD, chronic kidney disease; NO, nitric oxide; RAAS, renin-angiotensin-aldosterone system; SAAG, serum ascites albumin gradient.

downstream effects of splanchnic vasodilatation, decreased effective arterial blood volume, and intense renal sodium retention.[13] Factors like hypoalbuminemia, decreased oncotic pressure, and cirrhotic cardiomyopathy play additive roles. On the contrary, the mechanisms and pathophysiology of ascites in ESRD are diverse and poorly understood. Whereas specific subgroups like nephrotic syndrome ascites can be attributed to severe hypoalbuminemia for patients on dialysis, multiple theories have been proposed, including increased hepatic vein hydrostatic pressure, fluid retention, increased peritoneal membrane permeability, and impaired peritoneal lymphatic drainage.[14] Such varied postulated mechanisms may overlap with other potential causes, like cardiac failure, which is predominantly driven by volume overload and increased hydrostatic pressure.[15] A third situation arises when both ESRD and cirrhosis exist together where multiple drivers may be present, with usually one as dominant, and also get complicated by unrelated causes like peritoneal infections, including tuberculosis in high endemic regions.[16] Figure 1 gives a brief overview of the potential pathophysiological processes involved.

ETIOLOGY OF ASCITES IN PATIENTS WITH RENAL DISEASE

While the intuitive approach to looking for the presence of liver disease in such patients is common, the etiology of ascites tends to be varied and mandates a systematic approach. Figure 2 shows the multiple aetiologies that can lead to ascites in patients with renal disease and clues to diagnostic evaluation. Unfortunately, studies on patients with renal disorders as a whole and the aetiologies of ascites are limited and are restricted to patients with ESRD who are on maintenance hemodialysis. A recent study assessed the spectrum of ascites in adult patients with CKD.[17] Among 362 consecutive patients with stage III or above CKD, 47 (12.9%) had ascites, of whom 3 had preexisting cirrhosis or HCC. On evaluating those without a preexisting cause of ascites, 57.5% had high serum ascites albumin gradient (SAAG) ascites, of whom only 14% had cirrhosis or cardiac as a definite cause, whereas in 43%, no cause could be identified. Further, 42.5% of patients had low SAAG ascites, of whom a majority (67%) had multiple causes. Therefore, the workup of patients with CKD and ascites may yield surprising results, and in up to one-fourth of patients, no cause can be identified.[17]

APPROACH TO PATIENTS WITH RENAL DISEASE AND ASCITES

The first step in evaluating ascites, irrespective of whether there is associated renal disease, is to analyze the ascitic fluid total and differential cell count, protein content, SAAG, and, in appropriate scenarios,

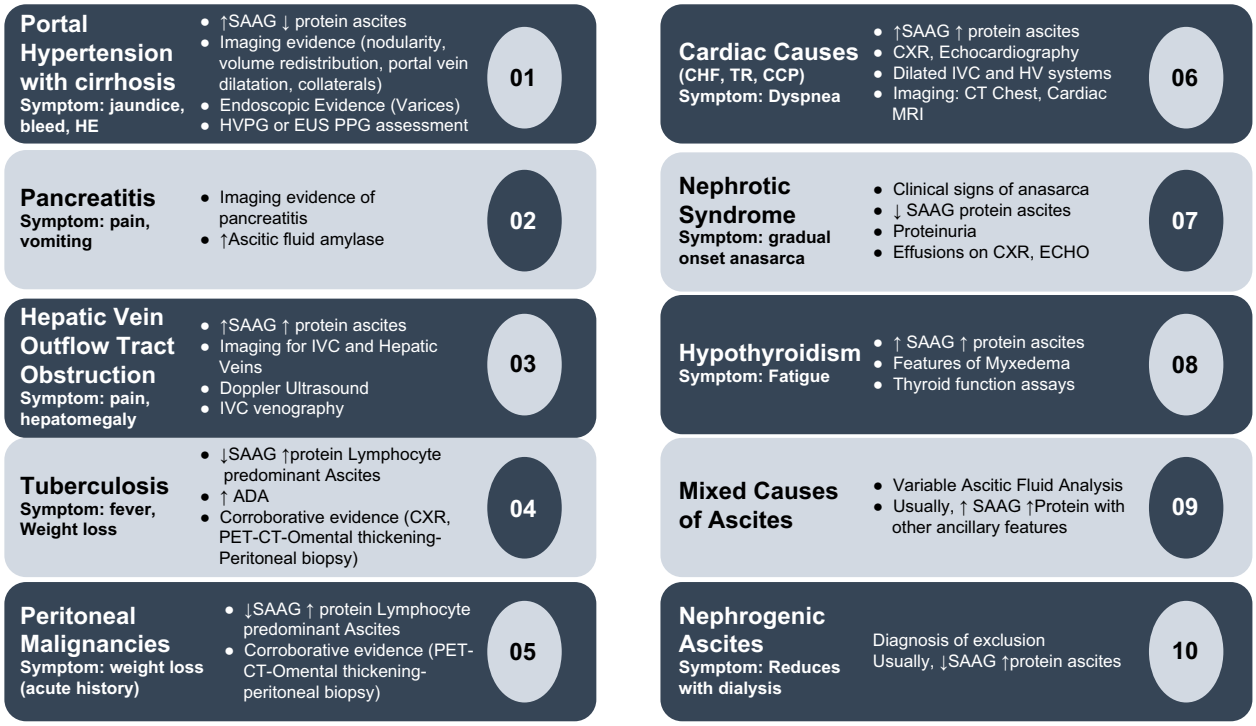

FIGURE 2 Common causes of ascites in patients with renal disease and indicators on evaluation. Abbreviations: ADA, adenosine deaminase; CXR, chest x ray; ECHO, echocardiocardiography; EUS PPG, endoscopic ultrasonography portal pressure gradient; HC, hepatic vein; HVPG, hepatic venous pressure gradient; IVC, inferior vena cava; PET-CT, positron emission tomography computed tomography; SAAG, serum ascites albumin gradient.

adenosine deaminase levels, malignant cytology, amylase levels, and triglyceride levels.[18] One of the most valuable tests in this regard is the SAAG, which, when having a value of $\geq$1.1 g/dL, predicts PH as a cause of ascites with 97% accuracy.[1] However, it should be mentioned that in the landmark study, patients were mostly recruited from hepatology inpatient and outpatient services, thereby increasing the pretest probability of having portal hypertensive ascites.[19] While these remain sacrosanct teachings, some recent studies have demonstrated different results. In a recent Mexican study, the diagnostic accuracy of SAAG was only 57%, with a sensitivity and specificity of 66% and 86%, respectively.[20] Similarly, in unselected medical cohorts, SAAG $\geq$1.1 g/L had a sensitivity of 85.5% and specificity of 60.6% for diagnosing PH as a cause of ascites (diagnostic accuracy = 78.5%).[21] Another study showed SAAG having a sensitivity of 90% and specificity of 77.5% in differentiating cirrhotic from non-cirrhotic ascites at a cut-off value of $\geq$1.1 gm/dL.[22] Table 2 summarizes some of the recent studies showing the variability in diagnosing portal hypertensive ascites with SAAG.[20–24] Additionally, there is some literature also to suggest that in the presence of severe hypoproteinemia and severe hypergammaglobulinemia, the interpretation of SAAG is inaccurate.[25,26] Therefore, although SAAG remains an "ideal test" in identifying the cause of ascites, cautious interpretation is required in patients with hypoproteinemia.

Along with SAAG, the other test that aids in an initial stratification is the ascitic fluid total protein content. The common causes of high SAAG–high protein ascites are early stages of hepatic vein outflow tract obstruction, cardiac failure, constrictive pericarditis, severe hypothyroidism, and mixed ascites. Contrarily, a low SAAG–high protein ascites indicates peritoneal causes (peritoneal malignancy, tuberculosis). Ascites in nephrotic syndrome are common in the pediatric population and deserve special mention.[27] Ascites are usually low SAAG and low protein but may be high SAAG when complicated with liver disease or cardiac failure, which is not uncommon. Cirrhosis usually presents high SAAG low protein ascites, whereas protein may increase with mixed ascites. Low SAAG ascites, although uncommon in cirrhosis, have been reported in a small study of 76 patients. A definite etiology could be identified in only 38% of patients. Most commonly, these were caused by primary bacterial peritonitis (38%), followed by peritoneal carcinomatosis or malignant ascites (28%) and nephrotic syndrome (17%).[28] Overall, the use of SAAG and the protein content of ascitic fluid need to be cautiously interpreted in patients with suspected renal disease and ascites. A "high SAAG" ascites cannot be considered a definitive indicator of portal hypertensive ascites in patients with ESRD, especially when the patients have hypoalbuminemia due to protein loss.

TABLE 2 Diagnostic performance of SAAG in differentiating ascites in recent studies

Authors	Year, country	No. of patients	Setting and design	Sn	Sp	AUC	Comments
Carventez Pérez et al[20]	2020, Mexico	87	Gastroenterology Prospective	66	86	0.57	Ascitic fluid albumin performed better than SAAG
Subhani et al[21]	2022, UK	162	Secondary Care Retrospective	85.5	65	0.78	—
Prabhu et al[22]	2019, India	60	General Medicine Prospective	90	77.5	0.75	The study also assessed lipid gradients and found them to be no superior to SAAG
Du et al[23]	2018, China	629	Multicentric Prospective	80	97	0.89	SAAG <1.1 and ascitic fluid cholesterol >45 increase specificity to 99% for non-portal hypertensive ascites
Zhu et al[24]	2019, China	704	Multicentric Prospective One training and three validation cohorts	80	96	0.89	Proposed serum ascites total protein gradient, which had a sensitivity of 88%, specificity of 92%, and diagnostic accuracy of 90%

Abbreviations: AUC, area under receiver operating curve; SAAG, serum ascites albumin gradient; Sn, sensitivity; Sp, specificity.

ASCITES IN PATIENTS WITH ESRD WITH OR WITHOUT DIALYSIS

Although ascites in patients with ESRD with or without dialysis have been an area of interest since the 1970s, the overall literature is limited. While other causes of ascites, as mentioned before, remain relevant in ESRD, a specific entity of nephrogenic ascites is noted in patients with ESRD on dialysis. Nephrogenic ascites are described as a clinical condition of refractory ascites in patients with ESRD on renal replacement therapy.[29] Controversies exist as sections from nephrology have questioned the existence of this entity.[12,29] Multiple terms have been used to describe the condition (dialysis ascites, hemodialysis-associated ascites, and idiopathic ascites), but nephrogenic ascites is the preferred one as the onset of ascites may occur earlier in the course of renal failure and well before the initiation of dialysis.[4]

The pathogenesis remains poorly understood, with multiple theories suggesting hepatic venous pressure, oncotic pressure changes, and peritoneal membrane dysfunction.[4,30] One of the earliest studies with 60 patients postulated increased fluid retention as a possible cause, but this has been questioned as dialysis patients frequently display signs of fluid overload, even without demonstrable ascites. Increased peritoneal permeability has also been postulated, with peritoneal biopsies suggestive of chronic inflammation; however, the evidence supporting the hypothesis remains limited and inconclusive.[31]

Some common themes emerge in such patients with the presence of moderate to massive ascites, hypertension, absent or minimal lower extremity edema, cachexia, and a history of dialysis-associated hypotension.[14] Diagnostic approaches remain as outlined prior, with initial evaluation focussing on ascitic fluid analysis. Tasneem et al[32] evaluated 90 patients (age >16 years) on maintenance hemodialysis and reported nephrogenic ascites to be the most common cause of ascites. The authors analyzed SAAG in individual cases and reported ascites to be low SAAG–high protein in 60% (frequently associated with a hemorrhagic appearance) and high SAAG–high protein in 33.3%, which was associated with cardiac ejection fraction $<40\%$ and PH. High SAAG–low protein ascites were noted only in 6.7% and were more commonly associated with PH. Inevitably, the onset of such states heralds a poor outcome and is associated with an extremely poor prognosis with poor responsiveness to diuretics, paracentesis, and albumin infusions. Table 3 provides a summary of key studies with patients with ESRD with or without dialysis and ascites.[14,17,30–35]

SPECIAL CIRCUMSTANCES: EXCLUSION OF PORTAL HYPERTENSION IN POTENTIAL RENAL TRANSPLANT RECIPIENTS

Prospective renal transplant candidates mandate exclusion of CLD and specifically PH in the course of transplant workup. The presence of ascites in them needs evaluation and is often challenging. Background clinical history about metabolic risk factors, alcohol consumption, time of onset and progression of ascites, and physical examination to rule out stigmata of CLD and peripheral signs of vena cava or hepatic vein outflow tract obstruction form essential steps in the initial evaluation. Frequently, patients on dialysis have chronic viral hepatitis B or C and need viral load evaluation even in the absence of antibodies to identify occult infections.

TABLE 3 Studies on ascites in patients with CKD with/without dialysis

Authors	Year, country	Number of patients/ type of study	Demographic characteristics	Cause of ascites	Comments
Wang et al[31]	1974, USA	60/ Prospective	CKD on dialysis 8 (13.3%)	Not specified Attributed to fluid retention and increased capillary permeability due to uremia	-
Singh et al[33]	1974, USA	68/Prospective	CKD with dialysis for at least 2 mo 8 (11.6%)	3 had congestive cardiac failure All had ascitic fluid protein ≥ 2 g/dL	Liver biopsy was done in 6, of whom 3 had features of chronic hemosiderosis. Peritoneal biopsy done in 1 having necrotizing candidiasis Ascites was frequently intractable with poor responses to paracentesis, dialysis, or albumin infusions
Arsimendi et al[34]	1976, USA	197/Prospective	CKD on dialysis 6 (3.0%) had ascites	Congestive heart failure, n = 2 IVC/Budd–Chiari syndrome, n = 2 Chronic peritonitis, n = 1 Nephrotic syndrome, n = 1	Ascitic fluid transudative in all except 1 (with peritonitis). There was no consistent association either with the etiology of renal disease or treatment of uremia. Occurrence of refractory ascites heralded poor prognosis
Gotloib and Servadio[30]	1976, Israel	23/Prospective	CKD on dialysis 6 (26%) had ascites	One patient had features of cardiac cirrhosis on biopsy, although there were no clinical features of heart failure. Cause attributed to hypoproteinemia and volume overload	All with ascites had hepatomegaly attributed to a volume overload state with a reduction in liver size after volume reduction
Glück and Nolph[14]	1987, USA	138[a]/Ambispective	138 cases with CKD on dialysis and ascites	Etiology was established in 20 out of 138 cases Cirrhosis or CLD, n = 7 Pancreatitis, n = 4 Congestive cardiac failure, n = 3 Tuberculosis, n = 3 IVC obstruction/BCS, n = 2 Hypothyroidism, n = 1 Constrictive pericarditis, n = 1	Use of different therapeutic modalities (ultrafiltration, local steroid, ascitic fluid re-infusion, peritoneal dialysis) achieves symptom control in about 50% of cases. Renal transplantation was the most effective
Tasneem et al[32]	2016, Pakistan	90/Prospective	CKD on dialysis Males 55% Median age 33 years	Nephrogenic 77.8% Cardiac failure 16.7% Hypothyroidism 6.67% Cirrhosis 4.4% Abdominal TB 2.2% Peritoneal carcinomatosis 1.1%	Severe ascites in $>50\%$ of cases. Predictors of severity were hypoalbuminemia <2.8 g/dL and ejection fraction $<40\%$
Sai Spandana et al[17]	2024, India	362/Prospective	CKD stage III or above 72.5% males Age 46.9 ± 13.3 47 (12.9%) had ascites	High SAAG–high protein, n = 12 High SAAG–low protein, n = 11 Low SAAG–high protein = 14 Low SAAG–low protein = 3	Etiology was established in 72.5%, while the rest were of indeterminate etiology Nephrogenic ascites were seen in 27.4% Very poor survival
Muliawan and Nur[35]	2024, Indonesia	47/Prospective	CKD on dialysis 53.2% females 10 (21.3%) had ascites	Associated heart failure in 11 (52%) Rest not elaborated	Increased risk of ascites with hypoalbuminemia (LR 22.08) and malnutrition (LR 51.14) based on SGA

[a]Includes a survey and collection of reported cases.

Abbreviations: BCS, Budd–Chiari syndrome; CLD, chronic liver disease; CKD, chronic kidney disease; LR, likelihood ratio; SAAG, serum ascites albumin gradient; SGA, subjective global assessment; TB, tuberculosis.

Patients with ESRD have low liver enzyme levels due to low enzyme activity due to uremia and the hemodilution effect of ESRD.[36] Therefore noninvasive tests of liver fibrosis like fibrosis-4 are inaccurate in CKD.[37] While anemia and leukopenia due to chronic inflammation, bone marrow suppression, erythropoietin deficiency, and dialysis are common in ESRD, a low platelet count may herald the presence of PH.[38] Liver elastography is challenging in patients with ESRD on dialysis, especially with ascites. Further, liver stiffness measurement by transient elastography decreases significantly after dialysis in patients, hence mandating post-dialysis measurements for accurate assessment of liver fibrosis.[39] However, transient elastography assessments are unreliable and not possible in the presence of ascites, and other modalities like shear wave elastography and magnetic resonance elastography may be useful in such settings, although the latter gets limited in the presence of iron deposition, which is common in ESRD.[40,41] Furthermore, emerging literature suggests that liver stiffness is falsely elevated in dialysis and returns to lower values after KT, thereby questioning the role of elastography in the pretransplant evaluation of KT recipients.[42] Liver biopsy remains confirmatory for assessing the degree of fibrosis, but caution needs to be exerted given the risks of bleeding due to hemostatic dysfunction in ESRD.[43]

Imaging characteristics for the presence of surface nodularity, volume redistribution of the liver, dilated splenoportal axis, splenomegaly, and collaterals provide clues to the presence of underlying cirrhosis and PH. Endoscopy to rule out varices is usually the next step, with the presence of varices confirming clinically significant portal hypertension. If cirrhosis is suspected based on clinical findings, additional evaluation is needed to determine if clinically significant portal hypertension is present with the use of hepatic venous pressure gradient. Patients with clinical evidence of decompensated cirrhosis (esophageal varices or other clinical findings, such as ascites or HE, among others) or significant PH (hepatic venous pressure gradient >10 mm Hg) should be evaluated for SLKT.[44] In contrast, patients with compensated cirrhosis without PH or those without evidence of cirrhosis can proceed to KT alone.[45] Figure 3 provides an overview of the steps in evaluating CLD in prospective renal transplant recipients and the fallacies of the available tests. A population that, however, remains a gray zone is those with non-cirrhotic PH requiring KT, who need to be stratified as per risks of surgery in the presence of PH as these are not candidates for liver transplantation unless complicated by liver failure and parenchymal extinction.[46]

Recent reports suggest that KT is safe in patients with compensated cirrhosis with excellent outcomes except for increased rates of hepatic decompensation, which can be medically managed.[47] Undergoing KT in the presence of PH is challenging and requires assessment for prognosis and prediction of post-operative decompensation. Risk stratification has been

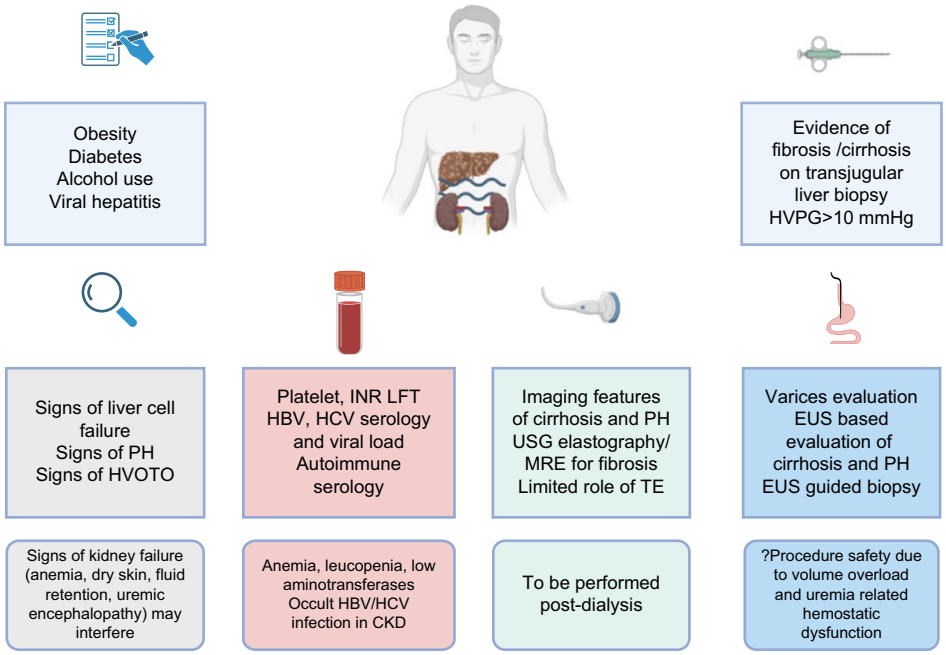

FIGURE 3 Evaluation of liver disease and PH in potential kidney transplant candidates. Created with biorender.com with permission. Abbreviations: CKD, chronic kidney disease; EUS, endoscopic ultrasonography; HVOTO, hepatic venous outflow tract obstruction; INR, international normalized ratio; LFT, liver function test; MRE, magnetic resonance elastography; PH, portal hypertension; TE, transient elastography; USG, ultrasonography.

based upon multiple scores, such as Child–Turcotte–Pugh, the MELD, the Mayo Surgical Risk Score, and, recently, the VOCAL-Penn surgical risk score.[48,49] Importantly, MELD and Mayo risk scores will have limited application in ESRD patients due to intrinsically raised creatinine, and non-portal hypertensive ESRD-associated ascites would introduce a fallacy in the Child–Turcotte–Pugh-class A score. However, their application and validation, specifically those undergoing KT, are lacking. Overall, the literature on outcomes of patients with compensated cirrhosis undergoing KT is also limited. Parsikia et al[50] reported no differences in renal graft outcomes in 18 patients with compensated cirrhosis (all Child–Turcotte–Pugh-class A), and only one reported death due to liver disease decompensation. A recent Indian study of 19 patients with ESRD undergoing KT with documented PH (varices, collaterals on imaging, or hepatic venous pressure gradient >5 mm Hg) also showed fair outcomes, with only one patient developing postoperative hepatic decompensation.[51] Further granular data on this subgroup's risk assessment and outcomes is needed. In Table 4, we summarize available data on KT alone and its outcomes in those with cirrhosis with or without PH, although most did not include individuals with ascites.[47,50–53]

TREATMENT OPTIONS FOR PATIENTS WITH RENAL DISEASE AND ASCITES

Options for management in patients with renal disease and ascites are limited if no other contributing etiology is identified. In the absence of liver cirrhosis, multiple options like ultrafiltration, local steroid, ascitic fluid re-infusion, and peritoneal dialysis (PD) have been used but achieve symptom control in $<50\%$ of cases.[14] Diuretics are used in volume-overloaded states but can

TABLE 4 Recent studies on kidney transplant alone in patients with compensated cirrhosis

Authors/year	Number of patients	Characteristics	Characterization of portal hypertension (PH)	Outcomes
Parsikia et al, 2015[50]	N = 18	88.8% males Mean age 56.9 ± 2.4 All were CTP-A Most commonly, HCV cirrhosis	Diagnosis of cirrhosis based on biopsy in 15 patients 3 had only radiological evidence Details of PH not provided Portal pressure measurement was done for 5 of 18 patients (median, 3 mm Hg, range 0–8)	Overall decompensation was not mentioned One patient died due to hepatic decompensation with preserved renal graft
Nho et al, 2015[52]	N = 12	92% males Age 48.5 ± 7.6 All HBV cirrhosis 8 were CTP-A and 4 CTP-B One patient had ascites None had HE	Not mentioned	4 patients developed HCC (median of 35 mo) Other decompensations not clarified
Dodge et al, 2023[53]	N = 34	79.4% of males are at a median age of 54 (IQR 44–61). Etiology: CHC (35.3%), followed by MASLD (17.6%)	Clinical signs of PH were present in 15 (44.1%) patients, 29.4% with nonbleeding esophageal varices or collaterals seen on imaging, and 5 patients with splenomegaly without varices. No patients had ascites or HE before KTA. HVPG was assessed in 10 patients (median 7 [range 3–14, IQR 6–8])	Hepatic decompensation developed in 8 (23.5%), of whom 4 died and 2 received LT
Nathani et al, 2024[47]	N = 32	Most commonly, chronic hepatitis C		Higher rate of decompensation Higher rate of infections
Gadde et al, 2024[51]	N = 19	All males 45.5 ± 11.3 Most common cause MASLD (36.8%)	All had evidence of PH confirmed by either endoscopy showing varices (8/19), HVPG >5 (12/19), or portosystemic collaterals on imaging (8/19). 7/19 had platelet count $<100{,}000$. Splenomegaly was present in 15 patients. 10 had ascites.	Two (10.5) patients had hepatic decompensation post-transplantation

Abbreviations: CHC, chronic hepatitis C; CTP, Child–Turcotte–Pugh-class A (CTP-A), class B (CTP-B); KTA, kidney transplant alone; LT, liver transplantation; MASLD, metabolic dysfunction–associated steatotic liver disease.

lead to further compromise of renal function.[54] Spironolactone has risks of hyperkalemia which can further be aggravated in ESRD, hence loop diuretics may be preferred such as torsemide which has a predominant hepatic excretion.[55] Large-volume paracentesis in line with practices done for decompensated cirrhosis with refractory ascites is often resorted to for symptomatic relief.[56] Paracentesis-induced circulatory dysfunction is a common complication in those with refractory ascites, which is partially ameliorated by the use of albumin infusions.[57] However, the incidences of paracentesis induced circulatory dysfunction in those with ESRD and ascites without PH remain unknown, and the impact of preventive strategies also remains unexplored. However, the incidences of paracentesis induced circulatory dysfunction in those with ESRD and ascites without PH remain unknown, and the impact of preventive strategies such as modest volume paracentesis or albumin expansion also remains unexplored.[58] PD is a viable option in such patients as hemodialysis is often associated with hemodynamic compromise in an already compromised hemodynamic state. Additionally, it obviates the need for therapeutic paracentesis, although risks of peritonitis remain.[56] Ultimately, KT remains the definitive treatment for such patients.

The challenges are further exaggerated in patients with underlying cirrhosis and CKD. While SLKT or kidney after liver transplant is a recommended strategy, in resource-limited settings where deceased donation programs are limited, such patients are frequently left to medical management. Decompensated cirrhosis patients with glomerular filtration rate (based on MDRD-6 formula) ≤ 30 mL/min or requiring dialysis for ≥ 3 months or patients with sustained AKI with glomerular filtration rate ≤ 25 mL/min or requiring dialysis for ≥ 6 weeks should be considered for SLKT.[44] Overzealous diuretic use is fraught with complications of precipitating HE in patients with cirrhosis and needs cautious treading.[59] TIPSs commonly used in refractory ascites have mixed results in the presence of advanced CKD. In a recent large study with 248 patients with advanced CKD, the presence of advanced CKD was an independent predictor of post-TIPS mortality (higher mortality 13.70% vs. 8.60%, aOR = 1.56, $p = 0.03$).[60] Moreover, in those undergoing TIPS for recurrent ascites, impaired renal function or chronic renal dysfunction has been shown to increase the risk for HE after TIPS, possibly mediated by hyperammonemia as a result of disturbances in renal ammonia genesis, as well as impaired ammonia clearance via the kidneys compounded by heightened systemic inflammation in CKD.[61–63] Conversely, few reports suggest TIPS being associated with improved renal parameters in 1 week after TIPS (serum creatinine 1.37 $\pm$ 0.23 from 1.94 $\pm$ 0.54 mg/dL, $p < 0.001$], with the improvements being maintained across every CKD stage.[64] Hence, the use of TIPS in CKD needs to be based on careful patient selection on an individualized basis and in the absence of SLKT prospects.

PD remains an alternative option. In a recent meta-analysis with 2753 patients with cirrhosis undergoing

TABLE 5 Treatment modalities in individuals with ESRD and ascites

Treatment modality	Comments
Diuretics	• Frequently used for hypervolemic patients with ascites but needs cautious treading for risks of worsening renal function and complications • Torsemide/metolazone preferred due to higher efficacy Concerns about worsening of renal impairment, especially in those not on dialysis, leading to progression to dialysis[54]
Therapeutic paracentesis	• Based upon practices used for patients with cirrhosis and refractory ascites • Paracentesis-induced circulatory dysfunction is a concern, however, incidences in those with ESRD and ascites without portal hypertension are not known
TIPS	• Data primarily from refractory ascites and CKD Concerns about worse outcomes and more incidences of HE[60,61] • Conflicting reports also suggest improvement in renal functions post-TIPS[64] • Ideal candidacy still needs to be determined
Hemodialysis	• Most commonly used RRT modality in those with ESRD and ascites • Worse outcomes in ESRD with ascites in comparison to those without[17] • Challenges with hemodynamic impairments, especially with those having concomitant cirrhosis
CAPD	• Comparable outcomes in those with or without cirrhosis[56] • Maintains better hemodynamic stability • Increased odds of peritonitis[56]
KT alone	• Definitive option in ESRD • Associated with risk of decompensation in the presence of clinically significant portal hypertension • Limited data suggests fair outcomes in compensated cirrhosis.[51]
SLKT	• Recommended option in those with ESRD and cirrhosis-related clinically significant portal hypertension with or without ascites

Abbreviations: CAPD, continuous ambulatory peritoneal dialysis; CKD, chronic kidney disease; ESRD, end-stage renal disease; RRT, renal replacement therapy; SLKT, simultaneous liver–kidney transplant.

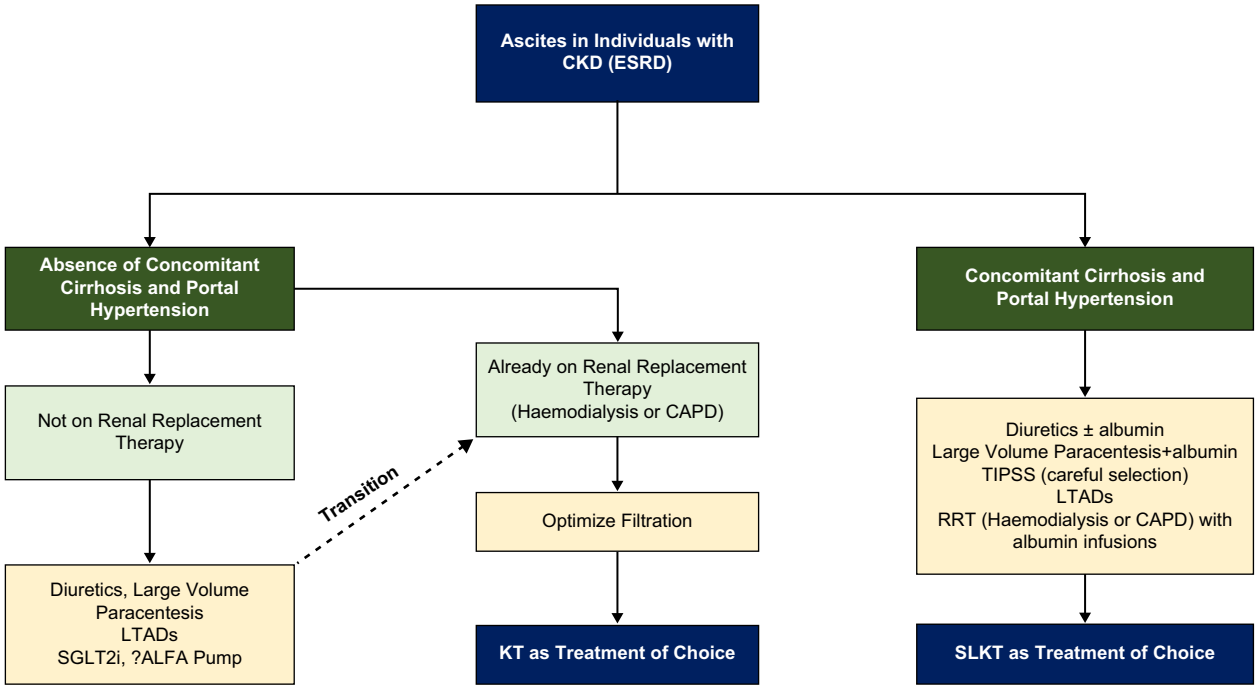

FIGURE 4 Algorithmic approach to the management of patients with end-stage renal disease with ascites. Abbreviations: ALFA, automated low-flow ascites pump; CAPD, continuous ambulatory peritoneal dialysis; LTAD, long-term abdominal drain; RRT, renal replacement therapy; SGLT2i, sodium-glucose co-transporter inhibitor; TIPS, transjugular, intrahepatic portosystemic shunts.

continuous ambulatory peritoneal dialysis, compared with those without cirrhosis, the odds of peritonitis was 1.10 (95% CI: 1.03–1.18), while mortality in cirrhosis patients on PD was comparable to non-cirrhosis controls, with a pooled OR of 1.05 (95% CI: 0.53–2.10).[65] Hence, continuous ambulatory peritoneal dialysis remains an option, albeit with a slightly increased risk of peritonitis. Encapsulating peritoneal sclerosis, a potential complication of PD associated with high mortality, also remains a concern.[66] Long-term indwelling catheters are helpful for palliative care in those not undergoing dialysis.[67] Automated low-flow ascites (ALFA) pumps are a novel device introduced for the management of refractory ascites.[68] However, their potential role in individuals with ESRD and ascites remains unexplored, and literature from patients with cirrhosis has raised concerns about incident events of AKI.[69] Furthermore, there remains an additional concern about the risk of infection with the use of such modalities.[70–72] Table 5 provides an overview of treatment options commonly utilized with patients with ESRD and ascites, and Figure 4 provides an algorithmic approach to the management of ascites with ESRD.

CONCLUSIONS AND FUTURE DIRECTIONS

Ascites in patients with renal disorders is an uncommon and challenging disorder. The approach to evaluation needs to be systematic with an attempt to rule out correctable causes. A significant proportion with ESRD have no identifiable cause, often designated as nephrogenic ascites, which has a grave prognosis, with KT being the only definitive option. Medical management in such patients is limited and often unsatisfactory. Future studies should evaluate predictors of ascites development in patients with ESRD, ideal noninvasive markers of cirrhosis and PH, and explore novel therapeutic strategies like automated low-flow ascites pumps, sodium-glucose co-transporter inhibitors, long-term albumin infusions during dialysis, and long-term home-based elastomeric vasoconstrictor therapy.[72–74] Consensus on diuretics of choice and the optimal trade-off between ascites control and worsening of renal functions needs well-designed studies with the involvement of nephrologists and hepatologists. In patients with cirrhosis, it is key to preserve renal functions. Key strategies should target optimization of hypertension and diabetes with diet and medications, control of obesity with lifestyle interventions including cessation of alcohol and smoking, and potential use of newer agents like semaglutide, and adoption of nephrotoxin stewardship like judicious use of proton pump inhibitors and avoidance of nephrotoxic agents like non-steroidal anti-inflammatory agents.[75–78] While limited promising data is emerging in KT alone in those with PH with preserved liver functions, the extrapolation of such strategies in those with ascites needs to be looked at with granularity. Complications with therapeutic paracentesis and preventive strategies for those with

ESRD with or without portal hypertension remain unexplored. Lastly, as we face the alarming complications of metabolic diseases, preventive measures (primordial) remain the only way to reduce the burden of ESRD and cirrhosis.

AUTHOR CONTRIBUTIONS

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CONFLICTS OF INTEREST

The authors have no conflicts to report.

ORCID

Akash Roy https://orcid.org/0000–0001–5126–1655
Anand V. Kulkarni https://orcid.org/0000–0002–1240–1675

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