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Peritoneal Dialysis in Hepatorenal Syndrome: A Viable Therapeutic Approach

Ramona Nicotera[1]* and Giovanni Mazzitello[2]

[1]*Chief Physician of the Nephrology and Dialysis Department, Italy*

[2]*Director of Nephrology and Dialysis, Italy*

***Corresponding author:** Ramona Nicotera, Soverato H, ASP CZ, Via Carlo De Cardona, 88068, Italy

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ABSTRACT

Ultrafiltration represents an alternative strategy to diuretic therapy in edematous syndromes, particularly in cases of diuretic resistance and/or the need to limit dosage due to worsening renal function or electrolyte imbalances. The two main techniques enabling this treatment are hemodialysis (HD) and peritoneal dialysis (PD). Extracorporeal ultrafiltration is indicated in emergency situations requiring rapid and effective fluid removal. However, this approach is associated with a higher risk of bleeding and anemia, accelerated decline of glomerular filtration rate, and increased costs compared to peritoneal dialysis. PD appears to offer advantages in hepatorenal syndrome. Ascites, the primary complication of cirrhosis, is defined as the pathological accumulation of fluid in the peritoneal cavity driven by portal hypertension. It results from a series of biochemical and vascular disturbances leading to abnormal fluid retention. Conventional therapeutic measures, including salt restriction and the use of loop diuretics combined with spironolactone, fail in approximately 10% of cases due to renal function deterioration and development of diuretic resistance, necessitating alternative therapies. PD may provide clinical benefits in this context.

Keywords: Peritoneal Dialysis; Hepatorenal Syndrome; Ultrafiltration; Diuretic Resistance; Peritonitis

Abbreviations: HRS: Hepatorenal Syndrome; ESRD: End-Stage Renal Disease; NO: Nitric Oxide; EGF: Endothelial Growth Factor; GFR: Glomerular Filtration Rate; PDGF: Platelet-Derived Growth Factor; RAAS: Renin-Angiotensin-Aldosterone System; SNS: Sympathetic Nervous System; ADH: Antidiuretic Hormone; CKD: Chronic Kidney Disease; TIPS: Transjugular Intrahepatic Portosystemic Shunt; PD: Peritoneal Dialysis; HRS: Hepatorenal Syndrome; RRT: Renal Replacement Therapy; HD: Hemodialysis; ESKD: End-Stage Kidney Disease; ACLF: Acute-On-Chronic Liver Failure; SBP: Spontaneous Bacterial Peritonitis; EPS: Encapsulating Peritoneal Sclerosis

Hepatorenal Syndrome: Definition, Pathophysiology, and Clinical Implications

The first definition of Hepatorenal Syndrome (HRS) dates back to 1996, with a subsequent revision published in 2007 [1-3]. The diagnostic criteria imply the presence of extensive pathophysiological correlations between hepatic and renal dysfunction. Two types of HRS have been described: Type 1 and Type 2. Type 1 HRS is characterized by an acute onset and a rapidly progressive decline in renal function. It often arises following gastrointestinal bleeding, large-volume paracentesis, acute alcoholic hepatitis, or spontaneous bacterial peritonitis. Its clinical course is frequently complicated by heart failure, acute cerebrovascular events, and adrenal insufficiency. Type 2 HRS, in contrast, presents a more chronic evolution and is defined by a slow and progressive decline in renal function, potentially leading to end-stage renal disease (ESRD). The exact pathophysiological mechanisms underlying the development of HRS remain incompletely understood; however, it is now well established that splanchnic vasodilation represents the primum movens in its pathogenesis. In the early stages of cirrhosis, hepatic inflammation promotes collagen deposition within hepatic sinusoids, increasing intrahepatic vascular resistance and worsening portal hypertension. As a consequence, there is a massive upregulation of nitric oxide (NO) and other vasodilatory mediators released by endothelial cells.

The increased shear stress on the sinusoidal walls contributes to the development of collateral vessels through the reopening of previously closed arterial channels and neoangiogenesis. This neovascularization is mediated by elevated production of growth factors such as endothelial growth factor (EGF) and platelet-derived growth factor (PDGF). A reduction in glomerular filtration rate (GFR) is closely associated with a decrease in effective plasma volume due to marked splanchnic vasodilation. This leads to activation of the renin–angiotensin–aldosterone system (RAAS) and the sympathetic nervous system (SNS), alongside increased secretion of antidiuretic hormone (ADH). The net effect is afferent arteriolar vasoconstriction, further reducing GFR. Patients with HRS typically exhibit low urinary sodium excretion due to increased tubular reabsorption, despite reduced GFR. Consequently, diuretic therapy with furosemide and spironolactone is often ineffective, given the reduced sodium delivery to the loop of Henle and distal tubules. Additionally, small amounts of water are reabsorbed in the distal nephron in response to elevated ADH levels, contributing to oligo-anuria. The translocation of gut-derived bacteria into the portal circulation is also implicated in HRS pathogenesis, triggering immune activation and a massive release of pro-inflammatory cytokines such as TNF-α, IL-6, and IL-1α. These cytokines contribute to systemic inflammation involving the lungs, heart, and kidneys [4-6].

From a clinical standpoint, hepatic failure-regardless of its duration-induces severe protein–energy malnutrition, hypoalbuminemia, hyperammonemia, and electrolyte disturbances. These patients also exhibit poor response to diuretics, chronic vasodilation, increased susceptibility to infections, and coagulopathy. The metabolic complications of renal failure further worsen the prognosis. In patients with HRS, dialysis is associated with significantly higher risks compared to non-cirrhotic patients, and there are no conclusive data regarding long-term outcomes [6-9]. Hemodialysis is often associated with serious complications. Severe comorbidities frequently result in poor vascular access options for arteriovenous fistula creation. The requirement for heparin administration during dialysis sessions may exacerbate underlying coagulopathy. The greatest risks arise from acute fluid shifts in patients with already reduced effective circulating volume and third-space fluid accumulation, leading to further hemodynamic instability. As a result, quality of life in these patients is markedly compromised, with substantial economic burden [10,11].

Diuretic Resistance: Mechanisms and Clinical Implications in Renal and Hepatic Disease

Diuretic resistance is defined as the failure to achieve adequate relief of volume overload, edema, or congestion despite the use of maximally titrated doses of loop diuretics (e.g., furosemide ≥80 mg once or multiple times daily in patients with reduced glomerular filtration rate or heart failure). This condition presents a significant therapeutic challenge in managing syndromes such as congestive heart failure, liver cirrhosis, and nephrotic syndrome [1-2].

The pathophysiology of diuretic resistance is multifactorial and involves several mechanisms:

1. **Neurohormonal activation:** Increased activity of the renin-angiotensin-aldosterone system (RAAS) and the sympathetic nervous system promotes sodium and water retention, blunting the natriuretic effects of diuretics.

2. **Tubular adaptation:** Chronic diuretic therapy can induce compensatory hypertrophy and enhanced sodium reabsorption in distal nephron segments, reducing drug efficacy over time.

3. **Reduced renal perfusion:** In advanced heart failure or liver cirrhosis, renal hypoperfusion may limit delivery of the drug to its site of action in the nephron.

4. **Pharmacokinetic alterations:** Reduced bioavailability, altered clearance, and the short half-life of loop diuretics may all contribute to suboptimal responses. The short duration of action in particular allows for post-diuretic sodium retention during interdose intervals

5. **Excessive sodium intake and electrolyte imbalances:** High daily sodium intake exceeding the diuretic-induced natriuresis, as well as associated electrolyte disturbances such as hyponatremia, hypokalemic hypochloremic metabolic alkalosis, and reflex neurohormonal activation, can further aggravate resistance [1-3,12,13].

Diuretic resistance may present in two forms:

- Acute resistance, or the "braking phenomenon," is characterized by a transient reduction in response occurring shortly after treatment initiation.

- Chronic resistance refers to a sustained inadequate response despite dose escalation or combination therapy [1-3].

In patients with chronic kidney disease (CKD), higher doses of furosemide are often required due to several additional factors. These include reduced renal perfusion, an expanded volume of distribution caused by hypoalbuminemia and impaired protein binding, decreased secretion of the drug into the proximal tubule due to competition for organic anion transporters (notably by urate and other retained solutes), and a reduced filtered sodium load resulting from impaired glomerular filtration [2,3,12,13]. In patients with advanced liver disease, particularly those with cirrhosis and ascites, diuretic resistance is a common and complex clinical issue. It results from a combination of hemodynamic, hormonal, and pharmacokinetic alterations unique to the cirrhotic state. Splanchnic vasodilation leads to effective hypovolemia, which triggers intense activation of RAAS, sympathetic nervous system, and antidiuretic hormone (ADH) release, all of which favor sodium and water retention. This neurohormonal activation is exacerbated by hypoalbuminemia, which reduces oncotic pressure and alters drug distribution and binding. Furthermore, reduced renal per-

fusion and glomerular filtration contribute to impaired drug delivery and sodium filtration.

Hypokalemia and metabolic alkalosis, often worsened by aldosterone excess and diuretic therapy, may further limit the response. In this context, diuretic resistance is typically defined by the failure to mobilize ascitic fluid despite maximum tolerated doses of spironolactone and furosemide, and it often marks a transition to more advanced stages of cirrhosis, where options such as large-volume paracentesis, albumin infusion, or transjugular intrahepatic portosystemic shunt (TIPS) may be required [7-9].

Peritoneal Replacement Therapy in Hepatorenal Syndrome

Peritoneal dialysis (PD) removes excess water and sodium primarily through osmotic ultrafiltration. Dialysis solutions containing glucose are commonly used; glucose acts as an osmotic agent, drawing water from the peritoneal capillaries into the dialysate. Sodium is removed via both convective and diffusive mechanisms. However, after a typical 4-hour dwell time, the osmotic gradient generated by glucose diminishes due to its absorption into the systemic circulation. For this reason, icodextrin-a glucose polymer-is increasingly employed as an alternative osmotic agent, providing sustained and effective ultrafiltration during long dwell periods. Peritoneal dialysis (PD) may represent a beneficial renal replacement therapy (RRT) in patients with hepatorenal syndrome (HRS) [11,14-18]. In cirrhotic patients, in addition to the removal of conventional uremic toxins, there is also a need to eliminate liver-derived toxins, many of which are protein-bound, particularly to albumin. Among extracorporeal therapies, hemofiltration-as compared to conventional hemodialysis (HD)-allows for enhanced removal of endotoxins and middle molecules, improving cardiovascular stability and reducing the risk of hypotension. Peritoneal dialysis, on the other hand, enables a gradual and continuous removal of solutes and fluids, thus better preserving residual renal function when present and potentially facilitating renal recovery when reversibility exists. Ascitic fluid contains reabsorbable toxins that may return to the plasma.

Frequent drainage of ascites and lavage of the peritoneal cavity using PD may reduce the concentration gradient of such toxins, thereby limiting reabsorption and enhancing their clearance [15-20]. Peritoneal clearance is generally satisfactory, also due to a commonly observed high or high-average peritoneal membrane permeability, though the underlying reasons for this characteristic remain unclear. In non-cirrhotic patients, ultrafiltration may be reduced due to increased glucose absorption and a decreased osmotic gradient between dialysate and plasma, resulting in impaired fluid removal. In contrast, cirrhotic patients often maintain effective ultrafiltration despite high peritoneal permeability. This paradox may be explained by increased intracapillary pressure, which drives fluid into the peritoneal cavity via a mechanism akin to ascites formation. Additionally, hypoalbuminemia facilitates transudation even when dialysate osmolality is reduced. Enhanced peritoneal permeability-driven by elevated intracapillary pressures-may support the clearance of otherwise poorly dialyzable toxins, particularly those with limiting size, charge, or conformational properties. In most patients, peritoneal drainage leads to progressive and continuous removal of ascitic fluid, which may induce sustained interstitial and intracellular fluid refilling into the vascular compartment, thereby improving fluid and electrolyte balance. Furthermore, PD enables individualized dialysis prescription.

In many cases-especially in the presence of residual diuresis or moderate ascites-1 to 2 daily exchanges, even with prolonged dwell times, may provide adequate solute clearance [14-20]. Notably, in high-permeability patients, PD effluent may contain albumin concentrations of 1–2 g/L after a few hours of dwell, suggesting that standard PD may already contribute to bilirubin clearance [20]. Malnutrition and hypoalbuminemia are frequently associated with both cirrhosis and dialysis. Splanchnic vasodilation and increased peritoneal permeability may contribute to significant losses of low- to medium-molecular-weight proteins (e.g., albumin, ferritin), as well as protein-bound or amino acid-derived toxins. Cirrhotic patients on PD exhibit higher early protein clearance than non-cirrhotic counterparts, although these losses tend to decline over time and do not typically result in significant reductions in plasma protein concentrations. In hepatic insufficiency, continuous glucose absorption through PD may be beneficial in the setting of impaired hepatic glycogen metabolism, thereby enhancing caloric intake. Current evidence does not support a detrimental effect of PD on nutritional status in cirrhotic patients. Caution is warranted regarding the use of amino acid-based PD solutions in cirrhotics. Commercial amino acid dialysates are often ineffective due to impaired hepatic protein metabolism and may contain excessive quantities of amino acids that are poorly metabolized, potentially leading to accumulation [17-22].

Both HD and PD maintain acceptable acid–base balance through acid removal and buffer provision. In HD, bicarbonate is the most common buffer. PD solutions typically contain either bicarbonate or lactate, which is metabolized by the liver into bicarbonate. In patients with hepatic failure, the continuous absorption and impaired metabolism of lactate could theoretically cause lactic acidosis and reach cardiotoxic levels. However, studies evaluating acid–base status in cirrhotic PD patients treated with lactate-buffered solutions have found values comparable to non-cirrhotic patients, and no reports of lactate-associated cardiac toxicity have been published [11]. Recent years have seen few studies on long-term outcomes in cirrhotic patients undergoing PD. Most publications are limited to case reports or small case series, often focused on patients with hepatorenal syndrome. The heterogeneity in etiology and clinical presentation among these patients makes group comparisons difficult. Furthermore, it is often challenging to distinguish between structural and functional renal failure, the latter of which may be reversible-a distinction that is

critical when considering isolated liver transplantation versus combined liver-kidney transplantation [17-22]. Chronic hypotension is common in cirrhosis and nearly universal during extracorporeal therapies. In HD, rapid fluid and osmotic shifts may induce intradialytic hypotension via activation of nitric oxide and kinin pathways.

In contrast, PD, through its continuous and gentle ultrafiltration, ensures more hemodynamic stability due to consistent vascular refilling. Interestingly, increased intra-abdominal pressure during PD may activate the renin–angiotensin–aldosterone system due to decreased renal perfusion. This mechanism may support the preservation or recovery of residual renal function [11]. Further support comes from a large cohort study conducted in Taiwan, which reported a significantly lower mortality in cirrhotic patients with end-stage kidney disease (ESKD) treated with PD compared to HD. This survival benefit persisted even after adjusting for key confounders such as Child–Pugh score, albumin levels, and comorbidities. The authors hypothesized that the more gradual fluid shifts and lower cardiovascular stress associated with PD may contribute to improved outcomes in these patients [23]. In the context of acute-on-chronic liver failure (ACLF) and severe AKI-a frequent scenario in type 1 HRS-a prospective Brazilian study investigated the safety and efficacy of high-volume PD. The study demonstrated that PD was able to achieve progressive ultrafiltration and correction of metabolic disturbances, despite the high severity of illness (mean MELD score >30). Although in-hospital mortality remained high (71.7%), PD was deemed technically feasible and safe in critically ill cirrhotic patients, offering an alternative in settings where extracorporeal techniques are not tolerated or available [24]. A recent systematic review and meta-analysis by Renaud, et al. [21].

Evaluated the outcomes of PD in cirrhotic patients and found no significant difference in mortality when compared to HD. Notably, while peritonitis and hypotension were slightly more common in the PD group, the overall risk of PD technique failure or switch to HD was not significantly increased, suggesting that PD remains a feasible long-term modality even in the presence of cirrhosis [22]. Nevertheless, PD in cirrhotic patients is not free of complications. The presence of a peritoneal catheter in the context of impaired tissue repair may predispose to pressure sores or peritoneal bleeding. Ascitic fluid at the catheter insertion site may impair proper healing and increase the risk of leakage or incisional hernias. Moreover, PD may exacerbate the risk of hernias and hydrothorax [11]. Bajo et al. reported such complications, although these were not observed by other authors, possibly because effective PD reduces ascitic volume [19]. Gastrointestinal symptoms such as delayed gastric emptying are observed in both non-dialyzed cirrhotic patients and those on PD. While paracentesis does not improve gastric motility, drainage of the peritoneal cavity in PD may resolve this issue [19,22]. Infectious risk is also a concern. Viral transmission (HIV, HBV, HCV) via peritoneal effluent has been described, although viral loads are typically lower than in plasma. Use of twin-bag systems and sodium hypochlorite in drain bags has minimized transmission risks, with no reported infections among household contacts of PD patients [18-20].

However, cirrhotic patients' heightened susceptibility to infections may increase the risk of peritonitis or catheter tract infections. Cirrhotics are inherently at risk for spontaneous bacterial peritonitis (SBP), primarily caused by gram-negative bacteria translocated via lymphatic and hematogenous routes from the gut. This risk is compounded by diagnostic or therapeutic paracentesis procedures, which may introduce gram-positive organisms [74–79]. In larger cohorts, the incidence of gram-negative peritonitis was similar across cirrhotic and non-cirrhotic PD patients and matched controls [20]. This may reflect the protective effect of continuous peritoneal lavage in removing translocated bacteria, potentially outweighing the reduced local immune defenses [11]. Nonetheless, treating peritonitis in cirrhotics is more complex. Hepatically metabolized antibiotics may exhibit altered clearance, and their intraperitoneal absorption may be increased due to high membrane permeability, requiring dose adjustments for both systemic and intraperitoneal routes [15]. Encapsulating peritoneal sclerosis (EPS) has been reported in ~10% of cirrhotic patients on long-term PD-a higher prevalence than in non-cirrhotics, although the etiology remains unclear. A number of case reports and small series support that liver cirrhosis is a condition in which EPS can occur, sometimes in conjunction with other risk factors, sometimes in the absence of classic ones.

Watanabe Kusunoki et al. describe a patient with alcoholic liver cirrhosis and end-stage renal disease (ESRD) who developed EPS after 4 years of peritoneal dialysis, despite PD durations of >5 years being the more usual risk [24]. Another report escribes EPS in a patient after liver transplant (the patient had hepatitis C cirrhosis originally) [25]. EPS may also occur in cirrhotic patients not on PD, possibly related to recurrent peritonitis and associated peritoneal fibrosis. The exact pathophysiology remains unclear, but several factors have been implicated. Chronic liver disease and portal hypertension lead to alterations in the mesenteric microcirculation, which may predispose the peritoneum to fibrosis and encapsulation. Inflammation, triggered by recurrent episodes of SBP, may contribute to the formation of fibrous tissue in the peritoneal cavity, eventually leading to the characteristic thickening and encapsulation of the intestine. Additionally, the presence of ascitic fluid, particularly in patients with poor ascitic control or frequent paracentesis, may further exacerbate peritoneal injury and fibrosis. Another proposed mechanism involves the dysregulation of the immune response in cirrhotic patients, which may promote the excessive deposition of extracellular matrix proteins and the development of peritoneal sclerosis. ESP is associated with significant morbidity and mortality, and its diagnosis is often challenging, requiring a high index of suspicion, imaging studies, and histopathological examination [24-32].

Conclusions

Although peritoneal dialysis does not significantly improve overall mortality in cirrhotic patients-whose prognosis is often poor due to underlying hepatic disease-it may enhance quality of life. Taken together, these data suggest that PD, when carefully selected and managed, may represent a viable and often underappreciated option for renal support in patients with cirrhosis and HRS or AKI. Its benefits may be particularly pronounced in patients with refractory ascites, unstable hemodynamics, or contraindications to anticoagulation. However, further randomized trials and prospective studies are needed to establish standardized protocols and better identify which subgroups benefit most from this approach. Encapsulating sclerosing peritonitis is more prevalent in cirrhotic patients, but the underlying mechanisms require further investigation.

Conflict of Interest

The authors declare that they have no financial interests and no conflicts of interest.

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