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Hepatic Hydrothorax: A Comprehensive Approach to Diagnosis and Management

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Keywords
Hepatic hydrothorax · Chronic liver disease · Cirrhosis · Portal hypertension

Abstract

Background: Hepatic hydrothorax (HH) is an uncommon but serious complication of advanced cirrhosis, characterized by transudative pleural effusion in the absence of cardiopulmonary or renal pathology. Despite its relatively low prevalence, HH carries significant morbidity and mortality, often presenting complex diagnostic and therapeutic challenges. ***Summary:*** The pathogenesis of HH is multifactorial, primarily involving the passage of ascitic fluid through diaphragmatic defects, exacerbated by altered hemodynamics, hypoalbuminemia, and neurohormonal dysregulation. Diagnosis relies on identifying transudative pleural effusion and excluding alternative etiologies. Management requires a stepwise multidisciplinary approach. Initial therapy includes sodium restriction, diuretics, and paracentesis to control ascites. Refractory cases may require serial thoracenteses, transjugular intrahepatic portosystemic shunt (TIPS), or surgical interventions in selected cases. Liver transplantation remains the definitive treatment leading to survival outcomes comparable to other transplant indications. ***Conclusion:*** HH develops in advanced cirrhosis and predicts a poor prognosis, with median survival often less than 1 year without transplantation. Optimal care requires early recognition, individualized treatment planning, and close multidisciplinary coordination between hepatology, pulmonology, interventional radiology, and surgical teams. Further research is needed to refine risk stratification and establish standardized management algorithms to improve patient outcomes.

Hidrotórax hepático: abordagem sistemática ao diagnóstico e tratamento

Palavras Chave
Hidrotórax hepático · Doença hepática Crónica · Cirrose · Hipertensão portal

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Resumo

Contexto: O hidrotórax hepático (HH) é uma complicação pouco frequente, mas grave, da cirrose avançada, caracterizada por um derrame pleural transudativo na ausência de patologia cardiopulmonar ou renal subjacente. Apesar da sua prevalência relativamente baixa, o HH associa-se a morbilidade e mortalidade significativas, apresentando frequentemente desafios diagnósticos e terapêuticos complexos. ***Sumário:*** A patogénese do HH é multifatorial, envolvendo principalmente a passagem de líquido ascítico através de defeitos diafragmáticos, sendo exacerbada por alterações hemodinâmicas, hipoalbuminemia e desregulação neuro-hormonal. O diagnóstico baseia-se na identificação de um derrame pleural transudativo e na exclusão de etiologias alternativas. O tratamento requer uma abordagem multidisciplinar sistematizada em etapas sucessivas. A terapêutica inicial inclui restrição de sódio, diuréticos e paracenteses para controlo da ascite. Nos casos refratários pode ser necessária a realização de toracocenteses seriadas, a colocação de um shunt portossistémico intra-hepático transjugular (TIPS) ou intervenções cirúrgicas em doentes selecionados. O transplante hepático permanece como tratamento definitivo, proporcionando sobrevidas comparáveis a outras indicações de transplante. ***Conclusão:*** O HH surge na cirrose avançada e associa-se a um prognóstico reservado, com sobrevida frequentemente inferior a um ano na ausência de transplante. A abordagem otimizada requer reconhecimento precoce, planeamento terapêutico individualizado e estreita coordenação multidisciplinar entre equipas de hepatologia, pneumologia, radiologia de intervenção e cirurgia. São necessários estudos adicionais para otimizar a estratificação do risco e estabelecer algoritmos de tratamento padronizados que melhorem o prognóstico clínico dos doentes.

Introduction

Hepatic hydrothorax (HH) refers to a transudative pleural effusion in patients with chronic liver disease and associated portal hypertension, in the absence of alternative causes such as cardiopulmonary or renal diseases [1, 2]. HH typically arises in the context of significant ascites, reflecting shared underlying pathophysiological mechanisms [3, 4]. In contrast with ascites, which may be well tolerated even at large volumes, relatively small volumes of pleural fluid – often as little as 500 mL – can lead to substantial symptoms and poor functional outcomes [5, 6]. Nevertheless, HH occasionally presents without clinically detectable ascites or in the setting of non-cirrhotic portal hypertension, which makes the diagnosis difficult and delays its management [7, 8].

Although HH is an uncommon complication of decompensated cirrhosis, it is associated with significant morbimortality and often poses considerable diagnostic and therapeutic challenges. Clinical management is complex and relies on a comprehensive and multidisciplinary approach. The current review aimed to explore the clinical features, diagnostic challenges, and updated therapeutic strategies of HH, highlighting the need for a multidisciplinary approach to optimize patient management and prognosis.

Epidemiology

HH accounts for only 2% of all causes of pleural effusion [6]. Among patients with liver cirrhosis, its prevalence is estimated in 5–16%, mostly when ascites and more advanced or decompensated cirrhosis are present [8–10]. In fact, only 10% of patients with HH do not have concomitant ascites [6]. HH is more frequent in the right hemithorax (73–85%), although it may also present as a left or bilateral pleural effusion (13–17% and 2–10%, respectively) [10, 11].

Pathogenesis

The pathophysiology of HH is complex and remains only partially understood, with several synergistic mechanisms being proposed. Ultimately, HH becomes clinically evident when the natural absorptive capacity of the pleural space is exceeded by a higher rate of fluid accumulation [1].

The main underlying mechanism is thought to be the direct passage of fluid from the peritoneal cavity into the pleural space through small diaphragmatic defects. This unidirectional flow concept has been demonstrated in several studies using radiolabeled compounds [12–14]. It is driven by the negative intrathoracic pressure that generates a permanent gradient between the peritoneal and pleural spaces, which is further exacerbated by the increased intra-abdominal pressure from ascites [1, 2].

Four distinct types of diaphragmatic defects in patients with HH are proposed: type 1, no visible defect; type 2, diaphragmatic blebs; type 3, diaphragmatic discontinuities or fenestrations; and type 4, multiple diaphragmatic gaps [15].

Notably, the defect type did not correlate with pleural effusion volume [15]. In the general population, diaphragmatic defects can occur in up to 20%, being typically small or even microscopic [16]. In cirrhotic patients, elevated intra-abdominal pressure caused by ascites accumulation combined with sarcopenia-related weakening of the diaphragmatic musculature may promote the enlargement of these defects [16–18]. They are more frequent in the right hemi-diaphragm, where a less strong muscular structure creates anatomical points of weakness, helping to explain the right-sided predominance of HH [17, 19–21]. Additional contributing factors that promote pleural fluid accumulation include reduced plasma oncotic pressure from hypoalbuminaemia, azygos vein hypertension associated with portosystemic shunting, and secondary hyperaldosteronism causing decreased sodium and water urinary excretion [20, 22].

Clinical Manifestations

The clinical presentation is generally indistinguishable from pleural effusions of other etiologies. However, as patients often present ascites and other stigmata of portal hypertension and liver cirrhosis (e.g., palmar erythema, spider angioma, abdominal collaterals, gynecomastia), HH is rarely the first manifestation of the underlying disease [2].

Common symptoms include dyspnea, nonproductive cough, pleuritic chest pain, and fatigue [10, 17, 23]. The magnitude of symptoms depends on the volume and rate of pleural fluid accumulation. Large or rapidly developing effusions can lead to acute respiratory failure or hemodynamic instability [24, 25]. Rarely, HH may be an incidental finding on chest radiograph in an asymptomatic patient, as even relatively modest fluid accumulation usually causes symptoms [1, 2].

Spontaneous bacterial empyema (SBE) complicates HH in approximately 13–16% of cases, and frequently, it is concomitant with spontaneous bacterial peritonitis (SBP) [23, 26]. The development of fever or signs of hepatic decompensation such as encephalopathy, jaundice, renal failure, hyponatremia, and/or variceal hemorrhage in the absence of another identifiable cause should raise suspicion for SBE [26].

Diagnosis

HH should be considered in every patient with known or suspected underlying liver disease who presents with pleural effusion, particularly when right-sided and concomitant ascites is evident [1, 4, 7]. The diagnosis implies documentation of pleural effusion, usually with chest radiography or computed tomography (shown in Fig. 1), exclusion of alternative causes (e.g., renal, cardiac, mediastinal, pleural, and pulmonary pathology), and pleural fluid analysis compatible with HH (Table 1). The minimum diagnostic workup to confirm HH and rule out other causes includes thoracocentesis with laboratory fluid analysis, contrast-enhanced chest computed tomography, and transthoracic Doppler echocardiography [19]. Pleural fluid should be sent for cell count and differential, Gram stain, bacterial culture, fluid pH, total protein, albumin, lactate dehydrogenase (LDH), and bilirubin concentrations. Routine serum laboratory evaluation should comprise creatine and urea, as well as LDH, total protein, albumin, glucose, and total bilirubin. N-terminal prohormone of brain natriuretic peptide (NT-proBNP) can also be a useful biomarker to differentiate hepatic hydrothorax from cardiac pleural effusions [27]. Depending on the clinical context and level of suspicion, additional serum or pleural fluid investigation may also be warranted to exclude alternative conditions within the broad differential diagnosis of pleural effusions [28, 29].

By definition, an uncomplicated HH is a transudative pleural effusion, which must be confirmed using Light's criteria [28]. Other typical pleural effusion features of uncomplicated HH may further support the diagnosis (Table 1). As in the case of ascitic fluid in patients with cirrhosis, HH has a typical low protein concentration (<2.5 g/dL) and a serum-to-pleural fluid albumin gradient ≥1.1 g/dL. However, differences can be found in fluid composition between pleural and ascitic fluids within the same patient, likely reflecting distinct absorptive mechanisms of both pleural and peritoneal spaces [6, 20, 30]. Notably, diuretic therapy can lead to a misclassification of a significant proportion of effusions as exudates as it can increase the concentration of protein, LDH, and lipids in pleural fluid [31].

In the setting of SBE, HH can present as either a transudate or an exudate [26]. Diagnostic criteria of SBE are pleural fluid polymorphonuclear (PMN) count ≥500 cells/μL or PMN ≥250 cells/μL with a positive culture, in the absence of pneumonia (Table 1). Other pleural fluid characteristics that suggest SBE are lower glucose concentration and pH [26, 32].

When diagnostic uncertainty persists, non-invasive imaging techniques such as abdominal/thoracoabdominal Doppler ultrasound, peritoneal scintigraphy, or magnetic resonance imaging may be performed to demonstrate

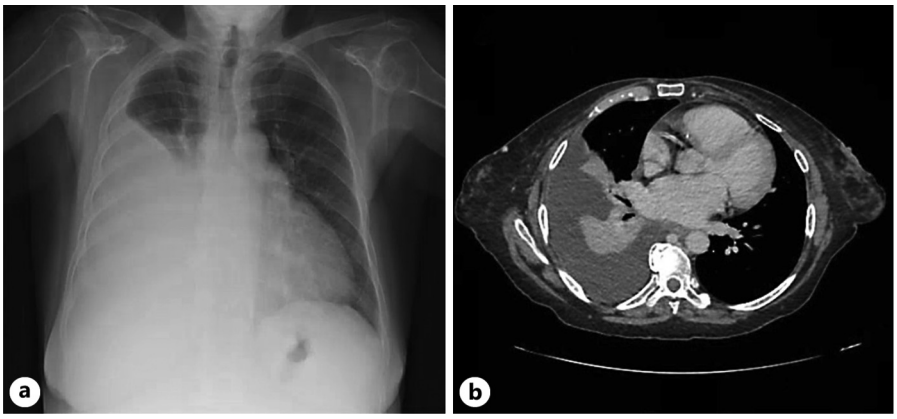

Fig. 1. Right-sided hepatic hydrothorax evident on chest radiography (**a**) and computed tomography (**b**).

Table 1. Pleural fluid characteristics of hepatic hydrothorax

Criteria	Values
Light's criteria for transudate[a]	
Pleural fluid total protein/serum total protein ratio	≤0.5
Pleural fluid LDH/serum LDH ratio	≤0.6
Pleural fluid LDH level	≤2/3 of the upper limit of normal serum LDH
Other typical pleural fluid characteristics of uncomplicated HH	
Pleural fluid polymorphonuclear cell count	<250/mm^3
Serum-to-pleural fluid albumin gradient	≥1.1
Pleural fluid total protein level	<2.5g/dL
Pleural fluid bilirubin/serum bilirubin ratio	<0.6
Pleural fluid pH	>7.4
Pleural fluid glucose level	Similar to serum glucose level
Diagnostic criteria for SBE[b]	
Pleural fluid polymorphonuclear cell count and pleural fluid culture	>500 cells/mm^3 + negative fluid culture or >250 cells/mm^3 + positive fluid culture

[a]All criteria must be fulfilled in order to classify the pleural effusion as a transudate.
[b]In the context of SBE, pleural fluid analysis can demonstrate both transudative and exudative characteristics.

diaphragmatic defects and fluid migration [12, 33–35]. Nevertheless, these investigations are less widely available, being rarely required in routine clinical practice.

Treatment

Management of HH remains challenging as no evidence-based standardized approach has been established in the literature. The complexity of cirrhotic patients coupled with the non-negligible potential for severe complications with each treatment option pose additional difficulties to decision-making. A comprehensive strategy guided by a multidisciplinary team integrating hepatologists, pulmonologists, interventional radiologists, thoracic surgeons, and general surgeons is essential to optimize clinical outcomes [1, 23]. Therapeutic interventions are categorized as medical, endovascular, or surgical and should be employed in a stepwise manner. Liver transplantation remains the

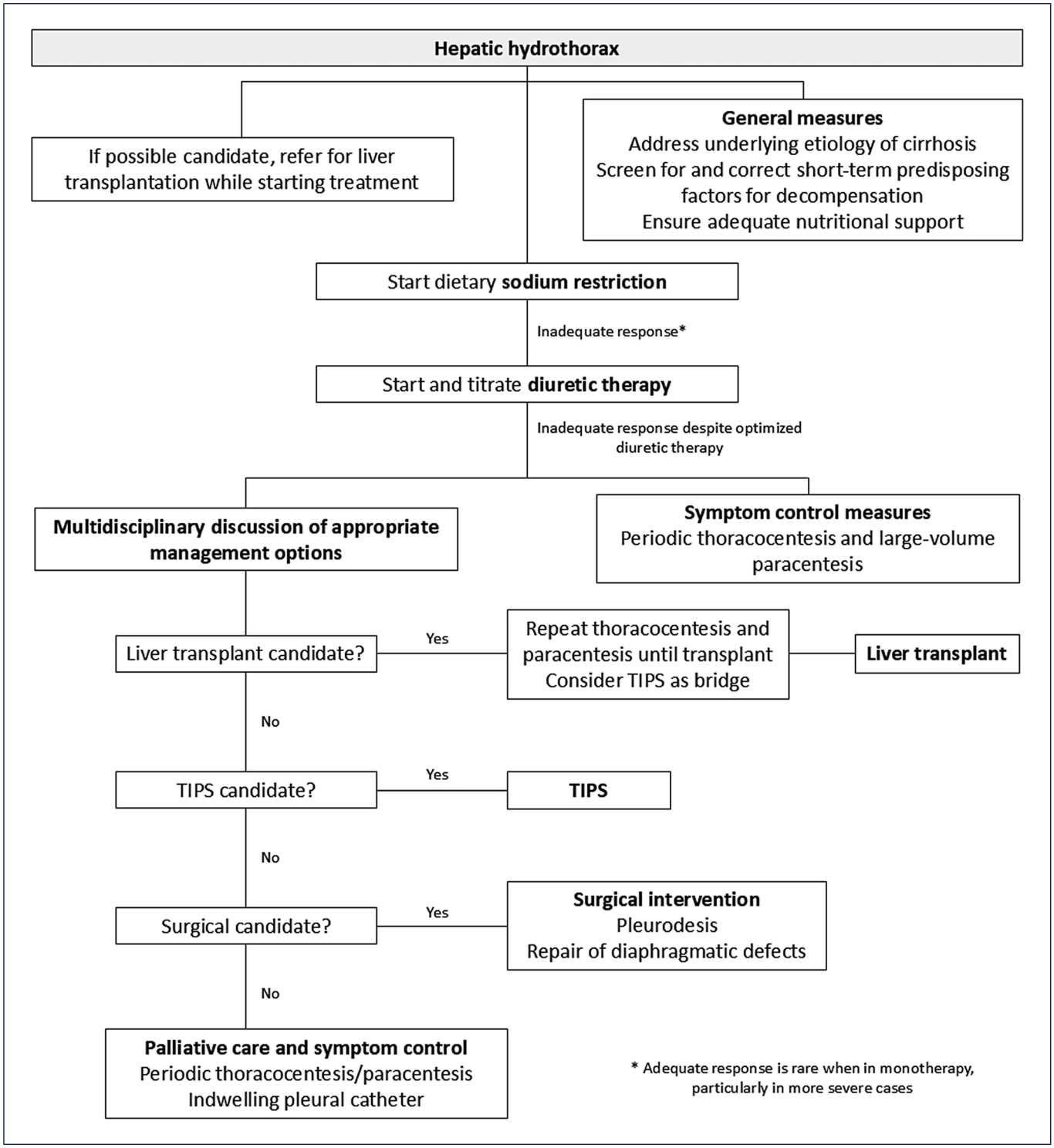

Fig. 2. Treatment algorithm of hepatic hydrothorax.

definitive curative treatment of HH and must be considered at the time of diagnosis in potentially candidate patients [17]. A proposed treatment algorithm is shown in Figure 2.

Medical Treatment

General Measures

While cirrhosis has traditionally been considered to be irreversible, more recent evidence shows that removal of the etiology of the liver disease may lead to at least partial regression of both structural and functional liver changes [36]. Emerging data support the possibility of "recompensation," a still-evolving and non-consensual concept currently defined only by expert consensus [37]. Therefore, underlying causes of cirrhosis should be addressed (e.g., initiating antiviral therapy in patients with viral hepatitis-related cirrhosis or ensuring complete alcohol abstinence in alcohol-related liver disease) as it can improve fibrosis, leading to increased liver function, slower disease progression, and reduced risk of liver-related complications and mortality [38–40]. Other short-term predisposing factors for decompensation should be proactively assessed and corrected whenever present, including treatment of infection, correction of electrolyte disorders, and management of constipation [37].

In addition to removal of the underlying etiology and risk factors for decompensation of cirrhosis, initial management primarily focuses on preventing and controlling ascites, thereby reducing fluid accumulation in the pleural cavity. Sodium excretion is impaired in most cirrhotic patients as a consequence of neurohormonal changes caused by portal hypertension and systemic circulatory dysfunction, and this is particularly more pronounced in decompensated or advanced cirrhosis [41, 42]. As a result, dietary sodium restriction is a cornerstone of HH management, aiming to limit hydrosaline retention. A moderate restriction of 80–120 mEq of sodium per day (which corresponds to 4.6–7 g of salt) is generally recommended [6, 42], although several studies support even stricter sodium limits [2, 11]. To achieve this restriction, patients should receive clear and practical support, including avoiding the addition of salt during cooking or while eating and limiting specific high-sodium foods. Education on reading food labels and identifying sources of sodium is also essential as adherence to dietary recommendations is often challenging without structured guidance [43]. Optimizing overall nutritional support is also crucial since malnutrition has a major impact on both complications of liver disease and overall outcomes [44]. Sarcopenia, a major component of malnutrition in cirrhotic patients, has also been suggested to contribute directly to the pathogenesis of hepatic hydrothorax by weakening the diaphragm through thinning and separation of its tissues [17, 18]. Therefore, referral to an experienced dietitian is strongly recommended as it can substantially improve low-sodium diet adherence and improve overall nutritional status.

Pharmacologic Treatment

Although dietary sodium restriction plays a central role in HH management, it has a relatively low success when used in monotherapy, particularly in more severe cases [1, 17]. Most patients require diuretics as part of the initial medical therapy to control ascites. Spironolactone is typically the first-line agent, started at 100 mg per day. Furosemide is frequently added at an initial dose of 40 mg/day if spironolactone monotherapy is insufficient or if hyperkalemia develops [23, 41, 42]. Close monitoring of body weight, blood pressure, orthostatic intolerance, serum creatinine, urea, and electrolytes is needed to assess therapeutic response and identify possible adverse effects. Diuretic-induced renal impairment, electrolyte disturbances, and hemodynamic instability are generally reversible upon drug discontinuation. The target weight loss with diuretic therapy should be at least 2 kg per week, with daily limits of up to 0.5 kg in patients without peripheral edema and 1 kg in those with edema [42]. Dose adjustments should be performed weekly in a stepwise approach, maintaining the spironolactone-to-furosemide ratio of 100:40 mg/day. Monitoring urinary electrolytes can help guide titration: a urine sodium-to-potassium ratio <1 suggests inadequate diuretic therapy and should prompt dose escalation when feasible [41, 45]. Titration is continued until ascites is adequately controlled, the highest tolerated doses are reached, or the maximum doses of 400 mg/day for spironolactone and 160 mg/day for furosemide are achieved.

Additional medical options include splanchnic and peripheral vasoconstrictors. Agents such as octreotide, terlipressin, and midodrine have shown potential benefit in some studies as therapeutic approaches for HH [46–48]. However, current evidence remains limited, and these drugs are not routinely employed in clinical practice.

Paracentesis

In patients with HH and tense ascites, large-volume paracentesis (LVP) is recommended. By reducing intra-abdominal pressure and ascitic volume, LVP indirectly decreases the rate of pleural fluid accumulation, and it

has also been shown to significantly improve lung volumes, leading to symptomatic improvement [49, 50]. In this setting, the removal of as much fluid as possible will maximize clinical benefits and extend the interval to the next paracentesis [42]. Intravenous colloid replacement with 6–8 g of albumin for every liter of fluid removed is typically recommended for paracentesis of 5 L or more to prevent associated circulatory dysfunction [42, 51, 52].

An implantable, subcutaneous device – the Automated Low Flow Pump System (Alfapump® system) – has recently been developed to manage refractory ascites. It was shown to significantly reduce the median number of large-volume paracenteses per month in a multicenter trial with 60 patients [53]. The system continuously transfers ascitic fluid from the peritoneal cavity into the urinary bladder, from where it is excreted with urine. Although it is designed for refractory ascites, it may also have a role in selected patients with HH, particularly when ascites is the predominant driver of pleural fluid accumulation. By reducing ascitic burden, it reduces the transdiaphragmatic movement of fluid. However, it has a high rate of complications, including infection, renal impairment, and device-related issues such as catheter migration or blockage, which currently restrict its widespread use in clinical practice [54–56]. Further studies are needed to better define its efficacy, safety, and role in the management of hepatic hydrothorax.

Serial Thoracocentesis

Up to 30% of cirrhotic patients with HH can develop recurrent or persistent pleural effusions despite sodium restriction and adequate diuretic therapy [2]. Those patients are typically referred to as having a diuretic-resistant HH. In contrast, patients who develop adverse effects requiring discontinuation of diuretics are classified as having diuretic-intractable HH. In these refractory cases, serial thoracocentesis is considered the standard of care [57]. Repeated thoracentesis can induce pleural inflammation, triggering the local release of proinflammatory cytokines such as tumor necrosis factor alpha (TNF-α). This, in turn, may stimulate plasminogen activator inhibitor-1 (PAI-1) production, promoting fibrin deposition and potential pleurodesis [58]. This intervention provides immediate symptom relief and may, in some cases, contribute to control of the effusion. It is a low-cost, widely available, and relatively simple procedure, although with potentially significant risks, particularly in patients with HH, where complications may include pneumothorax, hemothorax, and pleural infection [59]. Unlike LVP, there is no evidence to routinely recommend albumin infusion following thoracocentesis as the fluid volumes typically removed are much smaller.

Chest Drainage

Conventional chest tubes should be avoided in HH as they are associated with high mortality rates [60]. This is largely due to rapid, large-volume loss of electrolyte and protein-rich pleural fluid, which can precipitate acute kidney injury, exacerbate hepatic encephalopathy, and lead to death. Additional concerns include the high risk of infection and hemothorax, as well as the persistent accumulation of pleural fluid, which often complicates chest drain removal [61–63]. Chest tube insertion followed by talc slurry pleurodesis is also frequently unsuccessful in this setting, owing to the rapid fluid reaccumulation [64].

Indwelling Pleural Catheter

Indwelling pleural catheters (IPCs) are widely used in the management of symptomatic malignant pleural effusions, but their role in non-malignant pleural effusions is less well established [64]. Spontaneous pleurodesis has been reported in approximately 50% of patients with non-malignant pleural effusions (only 12% of whom have HH), while specific rates in the HH population are around 31% [65, 66]. IPCs are an option for patients who poorly tolerate repeated thoracenteses, particularly those who are not candidates for liver transplantation and in whom symptom palliation is the primary objective [57]. They allow outpatient symptom control, with drainage schedules tailored to the patient needs, reducing hospital admissions and improving quality of life. Infection is one of the most frequent and concerning complications, especially in immunosuppressed cirrhotic patients. This is particularly critical in liver transplant candidates, where an infectious episode may delay or preclude a potentially life-saving procedure. Other potential drawbacks include nutritional and electrolyte imbalances associated with repeated fluid drainage [57, 64]. For these reasons, IPC placement in HH should generally be reserved for patients managed with a palliative intent, in whom liver transplantation is no longer an option and the primary goal is durable symptom relief.

Treatment of Spontaneous Bacterial Empyema

In the setting of SBE, prompt initiation of intravenous antibiotic therapy is indicated. In most cases, the causative microorganisms are *Eschericia coli*, *Klebsiella*, *Streptococcus*, and *Enterococcus* species [26]. Similar to SBP, it is commonly performed with a third-generation

cephalosporin (e.g., Ceftriaxone 2 g daily) for 7–10 days. However, the selected antibiotic regimen should take into consideration the severity of infection, risk factors for multidrug-resistant microorganisms, and local resistance patterns [67–69]. Chest tube drainage should generally be avoided, although it may be needed in case of severe infection or frank pus is present. Albumin infusion (typically 1.5 g/kg on the first day and 1 g/kg on the third day), repeat thoracocentesis to assess treatment response, and secondary prophylaxis to prevent recurrence (typically with norfloxacin or ciprofloxacin) have less robust evidence in SBE compared to SBP but may be considered based on extrapolated SBP data [70].

Endovascular Treatment

The creation of a transjugular intrahepatic portosystemic shunt (TIPS) has emerged in recent years as a reasonable treatment option for the management of portal hypertension-related complications, including refractory/recurrent hydrothorax [1, 2]. This is well depicted in the recent standards of practise document of the Cardiovascular and Interventional Radiology Society of Europe (CIRSE) [71] and also recognized by the American Association for the Study of Liver Diseases (AASLD) [72].

TIPS is performed by catheterizing one of the hepatic veins via right jugular vein access, followed by puncture of the portal vein system, dilation of the tract, and deployment of a stent with variable length and adjustable diameter [71]. The rationale is to create a low-resistance communication between portal venous flow and systemic circulation, thereby reducing portal and intra-abdominal pressure, with a consequent decrease in transdiaphragmatic movement of fluid into the pleural space, resulting in symptomatic relief for most patients [1]. It should be considered in patients awaiting, or not suitable for, liver transplantation in whom optimal primary medical therapy and symptom control measures (paracentesis and thoracocentesis) were not enough [71, 72].

Some case reports and case series have documented favorable outcomes regarding refractory/recurrent hydrothorax [73–78], reporting long-term symptomatic relief in approximately 57%–79% of patients [75, 76], with complete resolution rates ranging from 53% to 59% [75, 78] and partial improvement in an additional 11%–28% of cases [77, 78]. The need for repeated thoracentesis is also markedly reduced, with most patients no longer requiring further pleural drainage procedures [73, 74, 76]. Improvement in Child-Pugh score is observed in a substantial portion of patients after TIPS [72, 74, 75], as well as an increase in post-TIPS serum albumin levels and urinary sodium excretion [76].

Appropriate patient selection is critical for treatment success. Contraindications include congestive heart failure, severe pulmonary hypertension (mean pulmonary artery pressure >45 mm Hg), severe uncorrected coagulopathy, and chronic hepatic encephalopathy [71]. In patients with a high Child-Pugh score (>12) or Model for End-Stage Liver Disease (MELD) score (>15–18), other treatment options should be carefully considered [71, 74, 78].

Complications most frequently observed are hepatic encephalopathy, early liver failure, heart failure secondary to an increase in preload, shunt infection, and dysfunction (including thrombosis and stenosis) [71]. In summary, TIPS is the primary interventional radiology procedure for refractory hydrothorax, with consistent evidence supporting its efficacy, although careful candidate selection is imperative to avoid the risk of procedure-related and long-term complications.

Surgical Treatment

Surgical options include mechanical or chemical pleurodesis, repair of diaphragmatic defects (such as blebs, fenestrations, or oozing sites), and, in selected cases, prosthetic reinforcement of the diaphragm, typically performed via video-assisted thoracoscopic surgery (VATS) [79]. In one series, the overall success rate of pleurodesis – either alone or combined with diaphragmatic repair – was approximately 72%. Among patients who underwent both diaphragmatic repair and pleurodesis (n = 75), the reported success rate of pleurodesis ranged from 55.6% to 100% [80]. Another series reported a 94% success rate with diaphragmatic defect repair – with or without mesh reinforcement – without concomitant surgical pleurodesis. Patients underwent preoperative drainage of around 1,000 mL of pleural effusion via a pigtail catheter 2 days before surgery, reducing the risk of postoperative lung re-expansion edema [79]. However, complication rates are high – 82% [80] and 32% [79] in the two series, respectively. In the latter, 3-month mortality reached 25%, with 38% of deaths attributed to septic shock [79]. Although surgical approaches may prevent recurrence more effectively than less invasive measures, the physiological burden is considerable, and perioperative mortality remains substantial, reflecting the frailty of patients with advanced cirrhosis [79, 80]. For these reasons, surgical interventions are generally discouraged and should be considered only in highly selected patients

with refractory HH, following multidisciplinary discussion, and performed exclusively in specialized centers with expertise in managing advanced liver disease.

Liver Transplant

Liver transplantation represents the only definitive treatment for patients with refractory HH [10]. Patients potentially suitable for transplantation should be assessed earlier in the course of the disease [42]. Referral should be particularly considered in patients with diuretic-resistant and diuretic-intractable hepatic hydrothorax, a history of SBE, or a high MELD score (>15) [4]. Post-transplant outcomes for patients with HH are similar to those of other liver transplant patients [81, 82]. Liver transplantation provides a substantial survival advantage in cirrhotic patients with HH, especially for those with refractory disease. One series concluded that liver transplant in refractory HH patients conferred an additional survival benefit in this group when compared to patients with non-refractory HH or other cirrhosis complications [83].

Prognosis

Hepatic hydrothorax is generally regarded as a marker of advanced cirrhosis and poor hepatic reserve. Survival outcomes vary considerably depending on disease severity, transplant eligibility, and the presence of complications such as refractory HH or SBE [1, 2, 84]. Several studies have reported that the median survival of patients with HH ranges from 8 to 12 months without liver transplantation [10, 23]. Mortality is typically driven by progressive liver failure, renal dysfunction, malnutrition, and recurrent infections, as well as complications of therapeutic interventions [2, 23]. Patients with refractory HH have a particularly poor prognosis, with median survival reported as low as 4.7 months [83]. Following hospitalization for HH, 45-day survival is only 80%, and 12-month transplant-free survival may be as low as 41% [84].

The development of HH is associated with a significantly higher mortality when compared with cirrhotic patients without this complication, even after adjusting for MELD and Child-Pugh scores [85]. While various therapeutic approaches are available, none alter the natural history of disease except liver transplantation. Post-transplant survival in patients with HH is comparable to that of other cirrhotic populations, highlighting the importance of early referral to a transplant center [81–83]. For those who are not transplant candidates, prognosis remains poor despite advances in medical and interventional therapies, and management is largely palliative.

Conclusion

Hepatic hydrothorax is an uncommon but clinically significant complication of advanced liver disease, associated with considerable morbidity and mortality. Its pathogenesis reflects a complex interplay between portal hypertension, diaphragmatic defects, and systemic consequences of cirrhosis. Diagnosis should include careful exclusion of alternative causes of pleural effusion.

Management remains challenging and must be individualized, balancing efficacy with the substantial risks associated with each therapeutic option. Medical therapy aimed at controlling ascites, combined with procedures such as paracentesis or thoracocentesis, represents the mainstay of initial management. For refractory cases, endovascular and surgical strategies may be considered, but patients should be cautiously selected. Timely evaluation for transplant candidacy is critical as it remains the only long-term curative treatment option.

Given its complexity, the optimal management of hepatic hydrothorax requires a multidisciplinary approach. Future research should focus on refining risk stratification, standardizing therapeutic algorithms, and exploring novel strategies to improve outcomes. Early recognition, timely intervention, and multidisciplinary collaboration are essential to optimize care and survival in HH.

Statement of Ethics

Ethical review and approval were not required as the study is based exclusively on the published literature.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Ivo Mendes, Ana Siopa Inácio, and Martim Costa Urbano were responsible for data collection and created the first draft of the manuscript. Paulo Calvinho and Gonçalo Nunes critically revised the final manuscript.

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