Sri Lanka Journal of Medicine **SLJM**

Narrative Review

Citation: Risly NMM, Jayasinghe IK, Athauda N et al. 2025. Hepatic Hydrothorax in Decompensated Cirrhosis: A Case Based Narrative Review of Management Challenges and Strategies, Sri Lanka Journal of Medicine pp. 46-53.
DOI: https://doi.org/10.4038/sljm.v34i4.608

Hepatic Hydrothorax in Decompensated Cirrhosis: A Case Based Narrative Review of Management Challenges and Strategies

NMM Risly [1], IK Jayasinghe[1], N Athauda[1], JF Sahana[2]

[1]National Hospital Kandy, Sri Lanka
[2]Teaching Hospital, Peradeniya, Sri Lanka

Correspondence:
Risly NMM
E mail: risly526@gmail.com
https://orcid.org/0009-0000-4743-5034

ABSTRACT

In Sri Lanka, liver transplantation is rarely performed for decompensated cirrhosis. Hepatic hydrothorax (HH) is a rare yet significant complication of decompensated cirrhosis, affecting approximately 5–10% of patients with portal hypertension. It results from ascitic fluid shifts through diaphragmatic defects, resulting in pleural effusions, mainly on the right side. HH is often refractory to standard diuretic therapy and poses considerable management challenges due to the lack of local consensus guidelines. This narrative review discusses the clinical complexities encountered in two patients with HH and highlights key management considerations, including the effective diuresis and mitigation of other associated complications along with meticulous fluid management strategies; yet ultimately emphasizing liver transplantation as the definitive treatment along with other possible therapeutic strategies.

Keywords: Hepatic hydrothorax, TIPS, liver transplantation

INTRODUCTION

In Sri Lanka, which is a resource poor country, the liver transplantation is rarely performed for decompensated cirrhosis. Due to the higher number of admissions with decompensated cirrhosis to the medical wards, it's justifiable to have consensus guideline-based approach for management of complications. Among many of the complications, the Hepatic hydrothorax (HH) is a relatively rarely encountered, yet a vital complication of decompensated cirrhosis, occurring in approximately 5–10% of patients with advanced disease complicated by portal hypertension. It is characterized by the fluid shift and accumulation of pleural fluid (transudative), mainly seen in the right hemithorax, due to defects on the diaphragm that allow the shift of ascitic fluid into the pleural space [1,2,3]. Unlike pleural effusions from cardio-renal etiologies, HH is often refractory to conventional therapy with diuretics and poses a significant therapeutic challenge in the absence of local consensus guidelines. Fluid overload in cirrhotic patients, often manifesting as ascites and edema, is primarily managed with diuretics, particularly spironolactone and furosemide. Nevertheless, in patients with hepatic encephalopathy (HE) (which is again a common co-existing and expected complication in cirrhosis), diuretic therapy

Received: 2025-02-01 **Accepted revised version: 2025-10-23** **Published: 2026-01-13**

presents unique challenges, as electrolyte disturbances, intravascular volume depletion, and impaired ammonia metabolism; can exacerbate neurocognitive dysfunction [4,5]. We hope to discuss on the management related issues in view of the latest available literature based on two complicated cases.

CASE PRESENTATION

Case 1

A 60-year-old man with a five-year history of alcoholic cirrhosis (Child-Pugh C) and porto-hypertensive gastropathy presented with worsening shortness of breath over two days, accompanied by right-sided pleuritic chest pain, altered sleep patterns, intermittent confusion, and progressive abdominal distension. Despite these symptoms, he did not have fever, melena, or hematemesis at presentation. He had also not opened bowels for two days but exhibited no other features like cold intolerance, long standing constipation, weight gain and apathy suggestive of hypothyroidism. Additionally, there were no complaints of frothy urine, reduced urine output, or periorbital edema. His past medical history was unremarkable for diabetes mellitus, chronic kidney disease, or other significant comorbidities.

On initial assessment, he appeared tachypneic, with an oxygen saturation of 90% on room air. A moderate right-sided pleural effusion was noted, but he remained hemodynamically stable, apart from tachycardia with a heart rate of 110 beats per minute. His Glasgow Coma Scale (GCS) was 14/15, and his abdomen was markedly distended. Basic investigations ruled out sepsis, and the pleural effusion was classified as transudative. A respiratory consultation was obtained, and given his poor prognosis (MELD score of 40), a conservative approach was adopted. The patient underwent frequent thoracentesis and ascitic drainage to alleviate symptoms.

Given that he was already in Grade 2 hepatic encephalopathy, diuresis was initiated cautiously, with meticulous electrolyte monitoring to prevent further neurological deterioration. His management was coordinated by a multidisciplinary team, ensuring careful titration of therapy. With continuation of two weeks of inward patient management in which, his ascites and pleural effusion gradually resolved, and his condition stabilized. He was subsequently discharged home in a stable state, with ongoing outpatient follow-up planned. Patient got readmitted in 1 week with recurrent HH, maximal diuresis done and discharged following 1 week on inward management. Later after another week later, patient again got readmitted with the same complaints and managed by gastro enterology team in-ward for 1 month with inotrope dependency (due to the complication of hepatorenal syndrome) and finally succumbed.

Case 2

A 56-year-old male with chronic liver disease (Child-Pugh C) presented with multiple episodes of hematemesis and progressive confusion over three days. He also reported worsening abdominal distension and increasing shortness of breath.

On examination, he was tachypneic, tachycardic, and hypoxic (SpO_2 90% on room air). His Glasgow Coma Scale (GCS) was 13/15, and a right-sided pleural effusion was noted. Given his history and clinical presentation, he was managed as a case of decompensated chronic liver disease with hepatic hydrothorax.

Initial management focused on careful salt restriction and diuresis, bowel clearance with a Kleen enema, blood transfusion, and empirical antibiotics, which led to an improvement in his hepatic encephalopathy. However, he remained dyspneic and oxygen-dependent, prompting the decision to insert a chest tube drain. The chest drain remained in place for 10 days, consistently draining more than 250 mL/day, making removal challenging. Given the persistent drainage, pleurodesis with doxycycline was performed via ultrasound-guided instillation after the intercostal tube was removed. Over the next four days, his respiratory status improved, and he was subsequently discharged home after a 20-day hospital stay. His MELD score at discharge was 36.

One month later, he was readmitted with abdominal pain and recurrent right-sided pleural effusion. However, as there were no signs of hepatic encephalopathy, he was managed with

maximum salt restriction and diuresis, leading to symptomatic improvement.

Both cases involved patients with decompensated chronic liver disease (Child-Pugh C) presenting with hepatic hydrothorax, ascites, and worsening encephalopathy, posing significant management challenges. A key difficulty was balancing effective diuresis while minimizing the risk of worsening hepatic encephalopathy, particularly given their high MELD scores (36 and 40, respectively). Careful salt restriction and diuretic therapy were crucial, with a preference for spironolactone over aggressive loop diuretics, as excessive fluid removal risked precipitating encephalopathy and electrolyte disturbances. Despite initial conservative management, including frequent thoracentesis and ascitic drainage, one patient required chest tube insertion and later pleurodesis due to persistent hydrothorax, while the other improved gradually with cautious diuresis. Both patients faced recurrent effusions and hospital readmissions, reflecting the progressive nature of end-stage liver disease and the limited options available short of liver transplantation.

Key Management Challenges that we encountered

1. Balancing Diuresis and Hepatic Encephalopathy: Both patients had high MELD scores (36 and 40), increasing the risk of diuretic-induced encephalopathy. Spironolactone was preferred over aggressive loop diuretics to maintain electrolyte balance. Careful salt restriction was crucial to avoid excessive sodium loss.

2. Recurrent Effusions and Readmissions: Despite initial stabilization, both patients experienced recurrent hydrothorax, requiring readmission. In the absence of definitive treatment of liver transplantation; readmission is unavoidable.

3. Fluid Management Approaches: One patient was managed with frequent thoracentesis and ascitic drainage, avoiding invasive interventions. The other required chest tube insertion and later pleurodesis, indicating more aggressive intervention for persistent hydrothorax.

4. Electrolyte and Volume Management: The risk of hypokalemia-induced worsening encephalopathy, dilutional hyponatremia and intravascular volume depletion due to splanchnic vasodilation resulting in hypotension which is associated with hepatorenal syndrome was mitigated by careful diuretic titration, intravenous human albumin and vasopressin. Close electrolyte monitoring helped avoiding complications related to excessive fluid removal.

5. Absence of consensus management for hepatic hydrothorax: There are multiple modalities in management of hepatic hydrothorax yet there is no consensus on the best modality even among the respiratory teams).

DISCUSSION

In our narrative review, we hope to the discuss literature-based pathophysiology, available treatment modalities and approaches to manage similar complicated scenarios. The primary mechanism underlying HH is the direct ascitic fluid shift via diaphragmatic microdefects, facilitated by negative intrathoracic pressure. This results in large-volume pleural effusions that can lead to respiratory distress [6]. HH is frequently a clinical diagnosis, supported by imaging and diagnostic thoracentesis, which reveals a transudative effusion (a low-protein, low-cell count, high-serum albumin gradient (SAAG)) [7]. During the initial presentation, HH can be misdiagnosed due to the masquerading other conditions, necessitating differentiation from cardiogenic effusions, parapneumonic effusions, and malignancy-related pleural fluid collection [8,9,10,11].

Management Strategies

The cornerstone of management of HH includes sodium restriction and diuretic therapy, typically using spironolactone with or without furosemide to control ascites and reduce accumulation fluid in the thoracic cavity [12,13,14]. However, aggressive diuresis can precipitate hepatic encephalopathy and renal dysfunction, necessitating careful titration. In patients with refractory hydrothorax,

large-volume thoracentesis being a temporary measure, having the risk of precipitating spontaneous bacterial empyema (SBE) [11]. For refractory cases, transjugular intrahepatic portosystemic shunt (TIPS) which is again a hard treatment to obtain freely in the country; has shown efficacy in reducing portal hypertension and effusion recurrence (A simplified approach is shown in Figure 1 adapted from Reference 1) [1,12,13]. Nevertheless, TIPS is contraindicated in patients with advanced hepatic encephalopathy or right heart dysfunction as in our cases. When HH remains resistant to conservative measures, pleurodesis using talc or doxycycline, or surgical repair (laparoscopic and open surgical) of diaphragmatic defects, may be considered, even though variable success rates are reported [12,13,14].

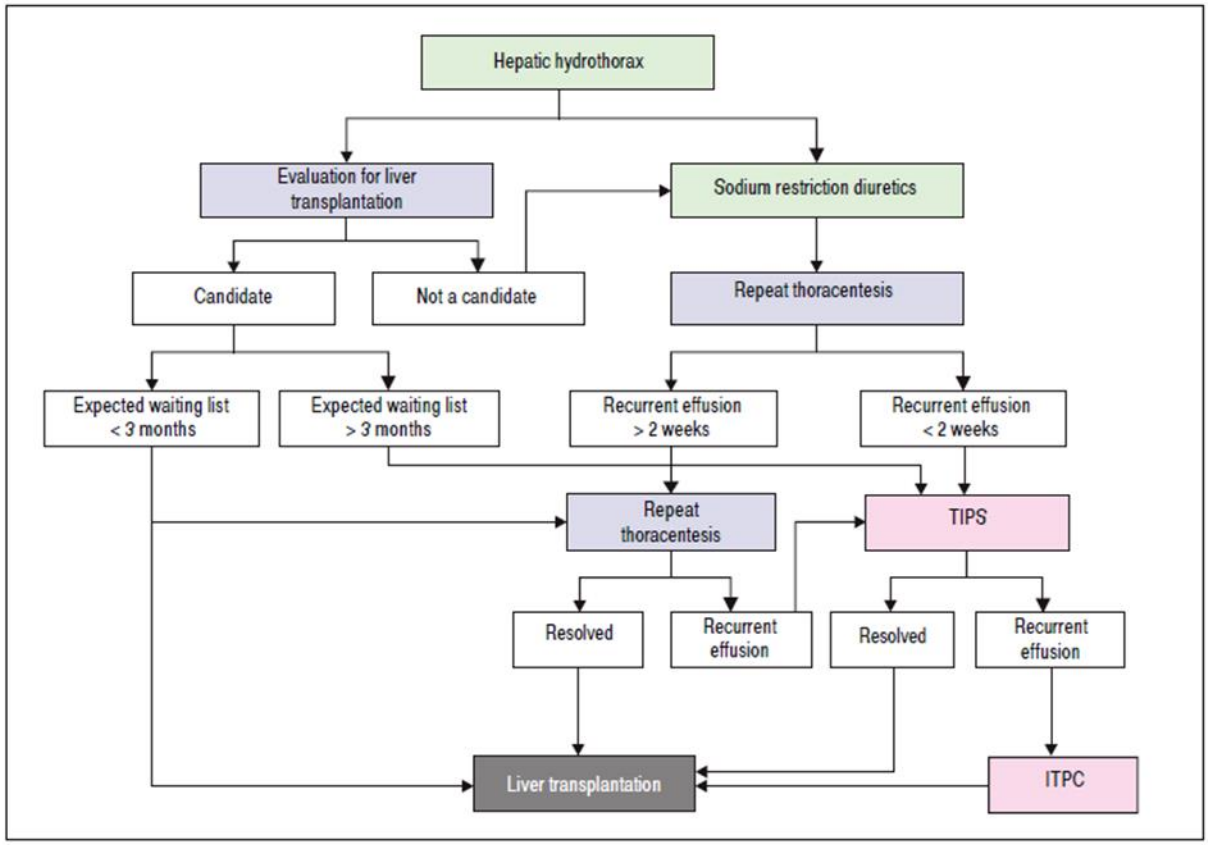

Figure 1: Management approach of Hepatic Hydrothorax, Adapted from Reference 1 ITPC: Indwelling tunneled pleural catheter. TIPS: Trans jugular Intrahepatic Portosystemic Shunt

Diuretics in Cirrhotic Ascites: Balancing Volume Control and Hepatic Encephalopathy (HE) Risk

Spironolactone, a potassium-sparing aldosterone antagonist, is the first-line diuretic in cirrhosis-related ascites due to secondary hyperaldosteronism [15]. Furosemide, a loop diuretic, is often added targeting a better diuresis and to prevent hyperkalemia that could be precipitated by spironolactone effect. However, aggressive fluid loss by therapeutic diuresis may worsen intravascular volume depletion, precipitating HE by reducing hepatic perfusion and worsening azotemia and risking hepatorenal syndrome [16,17]. Presence of hepatic encephalopathy per se can worsen the prognosis of the patient as well. In such a scenario, we recommend meticulous input output monitoring with ultrasound guided (central venous pressure guided if in an intensive care setting) fluid repletion.

Diuretic-Induced Hepatic Encephalopathy

Diuretic-induced hypokalemia plays a crucial role in HE exacerbation. Hypokalemia promotes ammonia production via renal ammoniagenesis and shifts ammonia into the central nervous

system, worsening neurotoxicity [18]. Additionally, overly aggressive excessive diuresis may cause metabolic alkalosis (contraction alkalosis), which facilitates the conversion of ammonium (NH_4^+) to ammonia (NH_3), enhancing ammonia's ability to cross the blood-brain barrier [18]. To mitigate these risks, potassium levels should be closely monitored, and potassium supplementation or the preferential use of spironolactone over furosemide should be considered [17,18]. In refractory cases of ascites, Hepatorenal Syndrome and HH, the infusion of albumin seems to have a place and shown to reverse diuretic induced HE, likely due to improved circulatory, hemodynamic stability due to the improved intravascular volume and impedance to intravascular volume contraction by its well-known osmotic effects [16]. Despite of the tailored management in each case; there were recurrence and one patient succumbed as well, indicating the progressive and refractory nature of the illness and it's difficult to recommend the best strategies depending on these two cases. We acknowledge that this is a limitation to the study; yet we emphasize that strategies tailed to resource poor setting like Sri Lanka should be adopted for reducing the morbidity related to the illness.

Our recommendations based on practical Considerations in Cirrhotic Patients with HE with HH as per the literature are listed down and shown in a visual algorithm (Figure 2) for better understanding

1. Start with Spironolactone Alone: Begin with 100 mg daily, titrating up to 400 mg/day as needed, while monitoring for hyperkalemia [15,17].

2. Add Furosemide Cautiously: Introduce furosemide (20–40 mg/day) only if weight loss is inadequate or hyperkalemia develops (3) [15]. The ratio of spironolactone to furosemide should be maintained at 100:40 mg to balance potassium levels [5].

3. Monitor Electrolytes and Renal Function: Check serum potassium, sodium, creatinine, and urea regularly to prevent hypokalemia and worsening HE [18]. May add potassium supplementation if necessary and consider IV albumin in large volume diuresis. In the meantime; maintenance of strict input output monitoring along with ultrasound guided fluid repletion in a high dependency unit setting (Central venous pressure guided fluid replacement is preferred if in an intensive care setting)

4. Adjust and modify therapy in worsening of HE: Reduce or hold diuretics if HE progresses, opting for albumin infusion in cases of significant intravascular depletion [16].

5. Consider Alternative Strategies: In diuretic-refractory ascites, options like paracentesis with albumin infusion or transjugular intrahepatic portosystemic shunt (TIPS) should be explored [4,17].

Prognosis and Future Directions

Hepatic hydrothorax signifies advanced liver disease and carries a poor prognosis, particularly in patients with high MELD scores.

The definitive treatment remains liver transplantation, which addresses the underlying portal hypertension and fluid imbalance [19]. Future research should focus on improving diagnostic accuracy, optimizing diuretic protocols, and refining TIPS selection criteria to enhance patient outcomes.

Conclusion

Management of hepatic hydrothorax in a decompensated CLCD needs multidisciplinary approach and careful considerations of case-by-case analysis. Diuretics are essential in managing fluid overload in cirrhosis, but their use in patients with hepatic encephalopathy requires careful balancing to avoid precipitating electrolyte imbalances, intravascular depletion, and worsening HE and hepatorenal syndrome. Close monitoring and adjustments based on clinical and biochemical parameters are critical to optimizing patient outcomes.

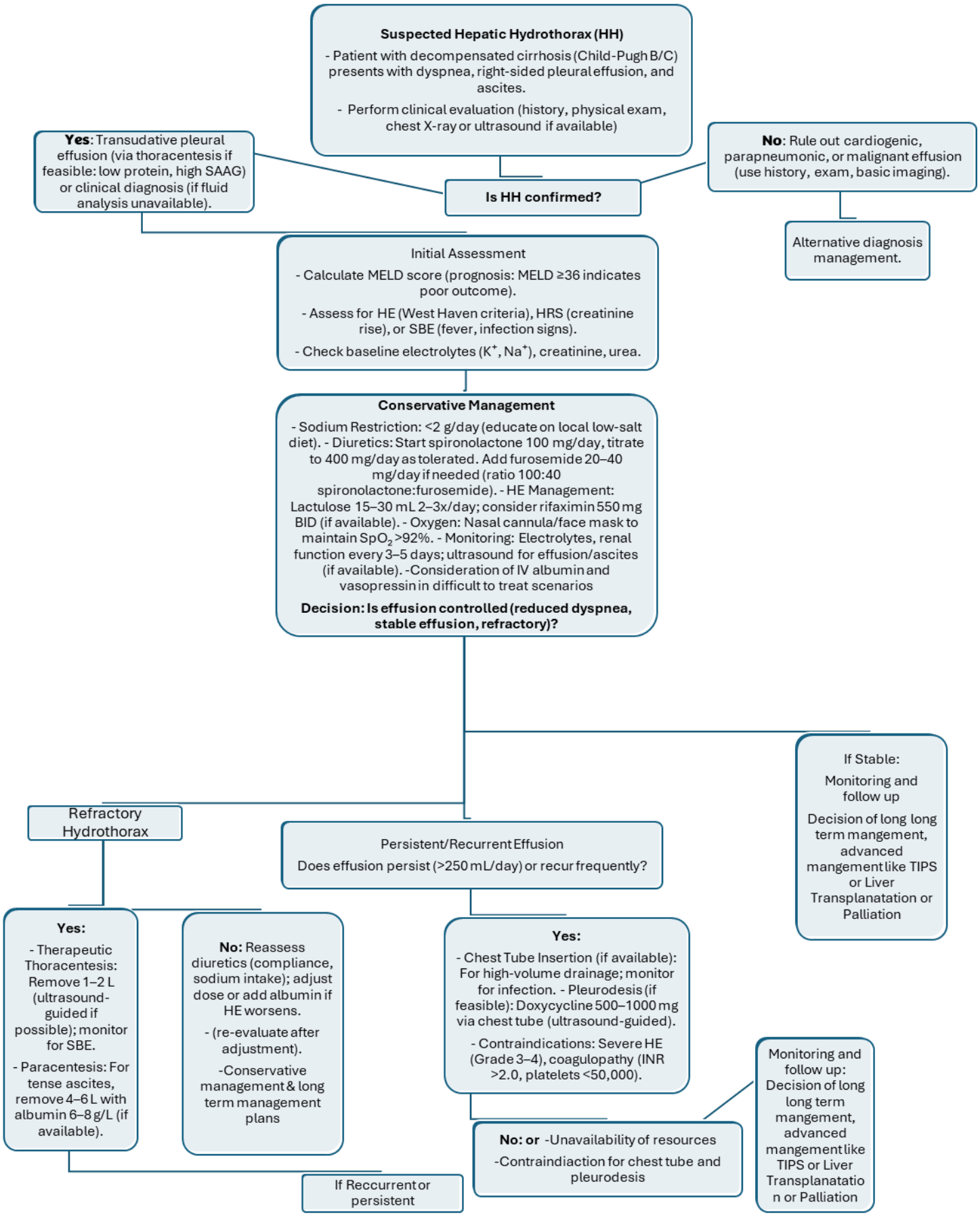

Figure 2: Suggested algorithm for management of hepatic hydrothorax

Abbreviations

HH: Hepatic Hydrothorax

HE: Hepatic Encephalopathy

TIPS: Transjugular Intrahepatic Portosystemic Shunt

MELD: Model for End-Stage Liver Disease

SBE: Spontaneous Bacterial Empyema

ITPC: Indwelling Tunnelled Pleural Catheter

Author declaration

Acknowledgement
None

Authors' contributions:
NMMR: Conceptualization, case management, manuscript drafting; NA: Case management, critical review and final approval; JFS: Manuscript drafting; IKJ: Critical review and final approval.

Conflicts of interest:
The authors declare that there is no financial or non-financial conflict of interest.

Funding statement:
Self-funded.

Ethics statement:
Not applicable. No identifiable patient data were used in this narrative review.

Statement on data availability:
All data used in this narrative review are derived from publicly available literature sources, which have been cited accordingly. No new patient data were generated or analyzed.

REFERENCES

1. Xiol X, Castellote J, Cortes-Beut R, Delgado M, Guardiola J, Sese E. Hepatic hydrothorax: clinical features, management, and outcomes in 77 patients and review of the literature. Medicine (Baltimore). 2004;83(1):1-13. Available from: https://www.elsevier.es/index.php?p=revista&pRevista=pdf-simple&pii=S166526811930105X&r=16
2. Badillo R, Rockey DC. Hepatic hydrothorax: an update and review of the literature. World J Gastroenterol. 2014;20(19):6535-40. Available from: https://pubmed.ncbi.nlm.nih.gov/24797168/
3. Garbuzenko DV, Arefyev NO. Hepatic hydrothorax: An update and review of the literature. World J Hepatol. 2017;9(31):1197-204. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC568020/
4. European Association for the Study of the Liver. EASL Clinical Practical Guidelines on the management of acute (fulminant) liver failure. J Hepatol. 2017;66(5):1047-1081. doi: 10.1016/j.jhep.2016.12.003. PMID: 28417882. https://pubmed.ncbi.nlm.nih.gov/28417882/
5. Starr SP, Raines D. Cirrhosis: diagnosis, management, and prevention. Am Fam Physician. 2011;84(12):1353-9. PMID: 22230269. Available from: https://pubmed.ncbi.nlm.nih.gov/22230269/
6. Singh A, Bajwa A, Shujaat A. Evidence-Based Review of the Management of Hepatic Hydrothorax. Respiration. 2013;86(2):155-73. Available from: https://pubmed.ncbi.nlm.nih.gov/23571767/
7. Porcel JM, Light RW. Diagnostic approach to pleural effusion in adults. Am Fam Physician. 2006;73(7):1211-20. Available from: https://pubmed.ncbi.nlm.nih.gov/16623208/
8. 8.Light RW. Pleural effusion. N Engl J Med. 2002;346(25):1971-7. Available from: https://www.nejm.org/doi/full/10.1056/NEJMcp010731
9. Porcel JM. Pearls and myths in pleural fluid analysis. Respirology. 2011;16(1):44-52. Available from: https://pubmed.ncbi.nlm.nih.gov/20573057/
10. Cárdenas A, Kelleher T, Chopra S. Review article: hepatic hydrothorax. Aliment Pharmacol Ther. 2004;20(3):271-9. Available from: https://pubmed.ncbi.nlm.nih.gov/15274663/
11. Castellote J, Xiol X, Gornals J, et al. Spontaneous bacterial empyema in cirrhotic patients: analysis of eleven cases. Hepatology. 1999;29(1):58-64. Available from: https://pubmed.ncbi.nlm.nih.gov/2179097/
12. Sanyal AJ, Genning C, Reddy KR, Wong F, Kowdley KV, Benner K, et al. The North American Study for the Treatment of Refractory Ascites. Gastroenterology. 2003;124(3):634-41. Available from: https://pubmed.ncbi.nlm.nih.gov/12612902/
13. Salerno F, Merli M, Cazzaniga M, Valeriano V, Rossi P, Lovaria A, et al. Randomized controlled study of TIPS versus paracentesis plus albumin in cirrhosis with severe ascites. Hepatology. 2004;40(3):629-35. Available from: https://pubmed.ncbi.nlm.nih.gov/15349901/
14. Luh SP, Chou MC. Diagnostic and therapeutic applications of videothoracoscopy: a review article. Int J Gerontol. 2007;1(2):53-60. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC136375/
15. Prakash R, Mullen KD. Mechanisms, diagnosis and management of hepatic encephalopathy. Nat Rev Gastroenterol Hepatol. 2010;7(9):515-25. doi: 10.1038/nrgastro.2010.116. PMID: 20703237. Available from: https://www.nature.com/artides/nrgastro.2010.116
16. Jalan R, Kapoor D. Reversal of diuretic-induced

hepatic encephalopathy with infusion of albumin but not colloid. Clin Sci (Lond). 2004;106(5):467-74. doi: 10.1042/CS20030357. PMID: 14678008. Available from: https://pubmed.ncbi.nlm.nih.gov/14678008/

17. Arkkila P, Nordin A. Treatment of ascites and its complications. Duodecim. 2016;132(18):1719-25. PMID: 29188952. Available from: https://pubmed.ncbi.nlm.nih.gov/29188952/
18. Gaduputi V, Chandrala C, Abbas N, Tariq H, Chilimuri S, Balar B. Prognostic significance of hypokalemia in hepatic encephalopathy. Hepatogastroenterology. 2014;61(133):1170-4. PMID: 25436277. Available from: https://pubmed.ncbi.nlm.nih.gov/25436277/
19. European Association for the Study of the Liver (EASL). EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. J Hepatol. 2018;69(2):406-60. Available from: https://pubmed.ncbi.nlm.nih.gov/29653741/