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REVIEW

Hepatic Hydrothorax: An Updated Review on a Challenging Disease

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Abstract
Hepatic hydrothorax is a challenging complication of cirrhosis related to portal hypertension with an incidence of 5–11% and occurs most commonly in patients with decompensated disease. Diagnosis is made through thoracentesis after excluding other causes of transudative effusions. It presents with dyspnea on exertion and it is most commonly right sided. Pathophysiology is mainly related to the direct passage of fluid from the peritoneal cavity through diaphragmatic defects. In this updated literature review, we summarize the diagnosis, clinical presentation, epidemiology and pathophysiology of hepatic hydrothorax, then we discuss a common complication of hepatic hydrothorax, spontaneous bacterial pleuritis, and how to diagnose and treat this condition. Finally, we elaborate all treatment options including chest tube drainage, pleurodesis, surgical intervention, Transjugular Intrahepatic Portosystemic Shunt and the most recent evidence on indwelling pleural catheters, discussing the available data and concluding with management recommendations.

Keywords Hepatic hydrothorax · Cirrhosis · Pleural effusion · Thoracentesis

Introduction

Hepatic hydrothorax (HH) is one of the pulmonary complications of cirrhosis along with hepatopulmonary syndrome and portopulmonary hypertension. It shares common pathophysiological pathways with ascites secondary to portal hypertension; however, it is less well defined and more challenging to treat. While ascites is common in cirrhotic patients reaching 50% within 10 years of diagnosis [1], the incidence of HH is reported to be between 5 and 11% [2, 3]. In this article, we will review the epidemiology, pathophysiology and efficacy of different treatment options of HH.

Definition and Epidemiology

Hepatic hydrothorax is defined as the accumulation of more than 500 ml, an arbitrarily chosen number, of transudative pleural effusion in a patient with portal hypertension after excluding pulmonary, cardiac, renal and other etiologies [4]. As said, its incidence is reported to be between 5 and 11% [2, 3] in patients with liver cirrhosis. It is noteworthy that the majority of cases occurs in patients with decompensated disease, with more than 90% in class B or C Child Pugh classification [2, 5]. While most of the cases are associated with ascites detectable at least by clinical imaging [2, 6], ascites might not be detected clinically in up to 42% of patients [6]. HH without any evidence of ascites is also reported [6–11].. HH is right-sided in 59–80% of cases, left sided in 12–17%, and bilateral in 8–24% of cases [2, 5, 6].

Diagnosis and Clinical Presentation

The clinical presentation of HH is quite variable. While some patients remain asymptomatic, the majority present with progressively increasing dyspnea on exertion and decreasing exercise tolerance that may be associated with cough and/or chest pain. Acute tension HH with rapid accumulation leading to hemodynamic collapse is extremely rare

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[12–14]. In one case, it was triggered by abdominal manual compression [13] and had a fatal outcome in another one [12].

Pleural fluid analysis usually reveals transudative effusion by Light's criteria [6]. Using the same criteria and as cirrhotic patients with ascites or HH are commonly on diuretic therapy, some cases are classified as exudative. The above still share the common pathophysiological mechanism as transudative effusions and they do not warrant investigation for infection or malignancy. Those mislabeled cirrhosis-related pseudoexudative effusions can be reclassified as transudative using an Albumin ratio < 0.6 (77%) or an Albumin gradient > 1.2 g/dl (62%) [15].

While hepatic hydothorax can be suspected based on imaging and presentation in the right clinical setting, it is still necessary to confirm the diagnosis by performing a thoracentesis to rule out the presence of other etiologies such as infections (spontaneous bacterial pleuritis, [16, 17] tuberculosis [17]), malignancies (lymphoma [18], adenocarcinoma [17]) and chylothorax [19–22]. In fact, thoracentesis yielded another diagnosis in 30% of cirrhotic patients with pleural effusion in prospective studies. In one prospective study, only 35% of cases of left-sided effusions were due to uncomplicated HH. Other etiologies included tuberculosis, empyema, adenocarcinoma and spontaneous bacterial pleuritis [17]. Liver cirrhosis is a common cause of chylothorax representing up to 20% of cases reviewed by Romero et al. [22] and is the most common cause of transudative chylothorax [19, 20]. Performing thoracentesis appears to be safe in cirrhotic patients and pneumothorax occured in 7 out of 76 therapeutic (9%) procedures and in 2 cases out of 200 (1%) diagnostic procedures [17]. It is, therefore, recommended to confirm the diagnosis of HH by performing a thoracocentesis.

Pathophysiology

The most supported mechanism of HH formation is the direct passage of fluid from the peritoneal to the pleural cavity through diaphragmatic defects. The presence of these defects was confirmed by various modalities including direct visualization during surgery [23], color doppler ultrasonography [24], MRI [25, 26], SPECT-CT [27], scintigraphy [26–28], and dye infusion in the peritoneal cavity [29–32]. These defects were classified as blebs, fenestrations, and large defects. They seem more frequent on the right hemidiaphragm as it is less muscular and more tendinous than the left one, and has more frequent embryologic defects explaining the predominant right-sided laterality The increased abdominal pressure from ascites and thinning of the diaphragm from malnutrition increases the risk of bleb rupture and fluid passage to the pleural compartment facilitated by the negative intrathoracic pressure [4, 33]. The increased abdominal pressure from ascites and thinning of the diaphragm from malnutrition increases the risk of bleb rupture and fluid passage to the pleural compartment facilitated by the negative intrathoracic pressure [4]. Azygous vein hypertension, lymphatic duct leakage and hypoalbuminemia might also play a role in the accumulation of fluid [4]. Furthermore, patients with HH have a high prevalence of diastolic dysfunction and left atrial enlargement, when evaluated by echocardiography, which might also be a contributing factor to the pleural fluid accumulation [6].

Spontaneous Bacterial Pleuritis

Spontaneous bacterial pleuritis, previously designated as spontaneous bacterial empyema [34], is an underdiagnosed complication of HH with high morbidity and mortality [3, 35, 36]. It is the spontaneous infection of the pleural fluid without associated pneumonia or secondary causes as postoperative empyema and infections related to chest tube insertion. It is postulated to be caused by spontaneous bacteremia reaching the pleural fluid or the passage of infected ascitic fluid from spontaneous bacterial peritonitis (SBP) to the pleural space.

The incidence of spontaneous bacterial pleuritis ranges between 16 and 54% of patients with HH [2, 3, 16, 35], as reported in mutilple prospective studies. 14–43% of these cases occur without associated SBP [3, 16], and thus one should still perform a thoracocentesis to investigate possible pleuritis in HH in the absence of SBP when pleural and abdominal fluids were sampled systematically in cirrhotic patients with HH and ascites, 31 out of 48 patients with SBP were found to also have spontaneous bacterial pleuritis [16]. Infected fluid can flow from the peritoneal to the pleural cavity but not in the other direction due to the negative intrapleural pressure; this might explain why isolated spontaneous bacterial pleuritis occurred more frequently than spontaneous bacterial peritonitis.

The clinical presentation of spontaneous bacterial pleuritis includes dyspnea, fever, abdominal pain, encephalopathy and worsening kidney function [3]. Pleural fluid analysis reveals a transudative character [6]. In a study by Gurung et al. [6], it was reported that the median LDH level was 80 IU/L, glucose levels were above 80 mg/dl and pH levels were above 7.35, strikingly different from the profile of pleural fluid studies in empyema characterized namely a low glucose, a pH less than 7.25, and the LDH above 600 IU/l [37]. When compared to sterile HH, two studies [3, 36] showed higher pleural fluid LDH in patients with spontaneous bacterial pleuritis, though they remained transudative and no cut-off value of LDH could be used for diagnosis. The main distinguishing feature when compared to sterile

HH is the pleural fluid Absolute Neutrophil Count (ANC) [6]. Hence, the diagnostic criteria are ANC > 250 cells/mm^3 with positive pleural fluid cultures, or ANC > 500 cells/mm^3 in case of negative cultures after excluding parapneumonic effusion [3, 4]. Direct inoculation of pleural fluid in blood culture bottles increased the microbiological yield [3, 36]. The germs recovered are mostly Enterobacteriaceae (mainly *Ecoli* and *Klebsiella* species) like SBP [3, 16] with ESBL, MRSA and Pseudomonas organisms reported in hospital acquired cases [35]; hence the antibiotic regimen is similar to SBP. Noteworthy is that the use of albumin to prevent renal failure was not evaluated in patients with infected HH.

The complication of spontaneous bacterial pleuritis has a high mortality rate, reported between 20 and 38% in various studies [3, 35, 36]. Initial ICU admission, higher MELD-Na score, and initial antibiotic failure are associated with poor outcomes [35]. Recurrence is also high and can reach 40% of treated and discharged patients [36].

Management

There is no consensus whether diagnostic thoracentesis is indicated in all hospitalized patients with hepatic hydothorax. As mentioned above, the clinician should have a high index of suspicion for fluid infection. Hence, fluid sampling should be entertained when clinical or laboratory data suggest infection (fever, leukocytosis,.etc.) or when facing unexplained renal failure or worsening hepatic encephalopathy. Management of HH starts with sodium restriction to less than 2 g per day. When sodium restriction fails, spironolactone and furosemide are added in a ratio of 100: 40 mg/day, with the dose titrated upward as needed up to a maximum of 400: 160 mg/day. When the above fail to control symptoms or when renal complications arise from diuretic therapy, then therapeutic thoracentesis is indicated. In spite of the mentioned therapeutics, and in 27% of cases [2] the management becomes more challenging as the need for repeated therapeutic thoracentesis and further interventions arises. It is unclear whether abdominal paracentesis in patients with concomitant ascites prevents reaccumulation of HH.

Although thoracentesis and pleurodesis are generally safe, the risk of complications increases with repeated thoracentesis. A recent case control study, comparing repeat thoracentesis in 82 patients with HH (274 thoracenteses) to 100 patients with other etiologies (control group; 188 thoracenteses), revealed a higher risk of major complications [8% in HH vs. 0% in the control group ($p = 0.016$, 95% CI 1.5–14.6)] [38]. This is likely explained by a higher risk of hemothorax (four cases) due to coagulopathy and reexpansion pulmonary edema (four cases) due to larger fluid removal. The cumulative risk of complications increased with sequential thoracenteses and a prior complication was the strongest predictor of a subsequent one (OR = 17.1, $p = 0.0013$).

Chest tube drainage of HH is discouraged due to the associated high morbidity and mortality [39–41]. A complication rate of up to 90% is reported in multiple case series including acute kidney injury, electrolyte imbalance, and malnutrition due to persistent drainage, empyema and pneumothorax. On the other hand, the use of pigtail tube for drainage showed a low rate of complications in one case series, with a mean number of days used of 10. The complications were limited to pain at the site of insertion and catheter blockage [14]. To the best of our knowledge, there is no direct comparison of pigtail insertion, in a head to head study, with chest tube drainage.

Indwelling pleural catheters (IPC) are tunneled catheters inserted subcutaneously in the ambulatory setting. They are used in the treatment of malignant pleural effusions and allow intermittent drainage at home. They are also increasingly being used for nonmalignant effusions as in patients with congestive heart failure, renal failure and other benign etiologies [42, 43]. There are case reports of successful management of HH with IPC in the literature showing [44–46] spontaneous pleurodesis. Table 1 summarizes the published case series on IPC in HH. In one pilot study from Washington University [47], 24 cirrhotic patients with reccurent HH who were deemed suitable for further interventions had an

Table 1 Published case series on the use of Indwelling Pleural Cathethers for HH

Study	Design	No of patients	Pleurodesis (%)	Complications	Year
Chen et al. [47]	Prospective single Center	24	33	16.7% (Infection)	2016
Kniese et al. [48]	Retrospective single center	62	14	36% (Infection [16%], pneumothorax, cathether malfunction)	2018
Shojaee et al. [49]	Retrospective multicenter	79	28	29% (Infection [10%] renal failure, electrolyte imblance)	2018

indwelling tunneled catheter placed by the interventional pulmonary service. Spontaneous pleurodesis occurred in 8 of 24 patients (33.3%) with a mean time to pleurodesis of 131.8 days (range 14–287 days). Pleural fluid infection was identified in 4 of 24 patients (16.7%), 3 of whom had to have their catheter removed.

More recently, Kniese et al. Reported a 10-year experience of IPC insertion in patients with HH at a large liver transplant referral center [48]. Their retrospective review included 62 cirrhotic patients, most of whom had received salt restriction, diuretics and serial thoracentesis. The procedure was performed as a bridge to transplantation in 53% of cases and as a palliative procedure in 38%. In 22 cases, the IPC was removed: 9 due to pleurodesis, 6 following transplantation, and 7 due to complications (empyema, tube dislodgement, etc.), with a mean time to death of 180 days after IPC placement. Complications occurred in 22 patients (35%). Empyema was the most common complication and occurred in 10 patients, leading to death in 3. Other notable complications included pneumothorax in two patients, skin infection in one patient, and catheter malfunction in five patients. As malnutrition due to protein loss from frequent drainage is a concern in cirrhotic patients, it was noted that serum albumin and body mass index (BMI) decreased over follow-up, but it remains unknown whether this decrease is related to IPC or to the natural history of cirrhosis [48].

In another recently published multicenter series [49], Shojaee et al. reported 79 patients with cirrhosis and refractory HH who underwent IPC placement in eight institutions between 2010 and 2016, with palliation intent in 73% of the cases and as a bridge to transplantation in 27% of the cases. 10% had pleural space infection and 2 patients (2.5%) died from sepsis related to the IPC. Spontaneous pleurodesis was achieved in 28% of cases with a median time to removal of 55 days (10–370). Severe electrolyte abnormalities occurred in only one patient.

Both studies showed that IPC might be an effective treatment option in the management of patients with decompensated cirrhosis and HH as a bridge to transplantation or for palliation purposes with an acceptable risk of complications. The reported benefits included avoiding recurrent thoracentesis, a 30% rate of spontaneous pleurodesis, and the possibility of removal following transplantation. The main complication is infection of the pleural space or the skin, requiring close monitoring for infection symptoms and signs, and sometimes removal of the catheter.

Pleurodesis of HH has been attempted using different techniques. Medical pleurodesis through chest tube is reported in case reports and small case series, with a variable success rate [11, 14, 50–52]. 3 out of 8 patients had successful pleurodesis in the series reported by Assouad et al. and success in all 38 patients who underwent pleurodesis with no recurrence was reported by Sharaf-Eldin [52]. and success in all 38 patients who underwent pleurodesis with no recurrence was reported by Sharaf-Eldin et al. . While Assouad [14]. While Assouad et al. reserved medical pleurodesis for patients unable to tolerate general anesthesia for VATS, Sharaf-Eldin et al. included all patients, which might explain the difference in success rates. Moreover, Assouad et al. Used chest tubes with talc while Sharafeddin et al. Used pigtail catheters with povidine iodine. Other agents used and reported in the literature include talc, minocycline, OK-432, and povidone-iodine [18, 53]..

Surgical interventions to treat HH are multiple and include pleurovenous shunts with evidence of their utility limited to case reports with resolution of the effusion for up to 8 months in one case and 19 months of follow-up in another case [54, 55]. Thoracoscopy or VATS for pleurodesis with or without diaphragmatic defect repair [5, 29–32, 52, 56–66] has also been used to treat HH with a variable success rate reported in different case series. Thoracoscopy, before the era of VATS, was used for both pleurodesis and diaphragmatic mesh repair [5, 57, 59] with a success rate inferior to 50% in one case series from Brazil [65]. VATS had higher success rates ranging from 53 to 80% with more than one procedure sometimes required [52, 58, 62, 64]. Localizing and repairing the diaphragmatic defect increases the success rate [29–32, 60], and leads to improvement of mortality [67]. The procedure-related mortality is variable but reached 45% in one series of 11 patients [66]. While no procedure-related death was reported in other series, the 3-month mortality rate was also high, reaching up to 40% [65] which might be related to the decompensated cirrhosis in these patients. Complications related to the surgery included acute kidney injury [57], pleurocutanous fistula, and empyema [62]. Finally, in a meta-analysis of 13 studies including 180 patients, success with surgical pleurodesis was 73% [5]. The lack of randomized clinical trials and the retrospective and observational nature of most studies, in addition to the absence of clear selection criteria for surgery, makes drawing conclusions a difficult task.

Transjugular intrahepatic portosystemic shunts (TIPS) decrease the portal pressure by creating a vascular intrahepatic bypass via metallic stent deployment between the hepatic vein and the intrahepatic portion of the portal vein. They are used in the management of refractory ascites and in patients with refractory variceal bleeding. By diverting the portal blood flow into the hepatic vein, TIPS decreases portal pressure and the portosystemic pressure gradient thus preventing fluid accumulation in the peritoneal and pleural cavities. Patients who are not candidates for TIPS are those with hepatic encephalopathy, severe heart failure, and pulmonary hypertension, as well as those with a Pre TIPS Model For End-Stage Liver Disease MELD score more than 18 [68]. MELD score is a score used to stratify patients on liver transplantation waiting lists and can be used to predict

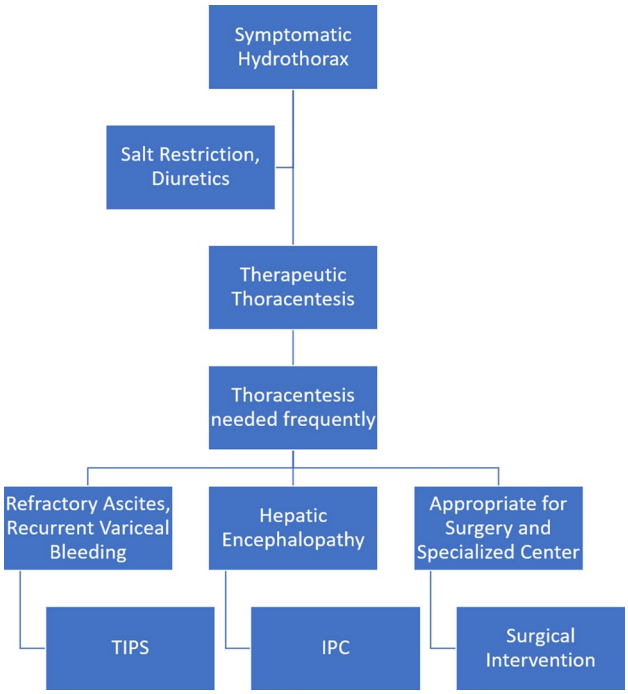

Fig. 1 Suggested algorithm for the management of Hepatic Hydrothorax. *TIPS* transjugular intrahepatic portosystemic shunt, *IPC* indwelling pleural cathether

3-month mortality in patients with cirrhosis. In HH, favorable response to TIPS was reported in multiple case reports and case series [69–74]. Success rates varied from 58 and 73% in the largest series [70–73]. Complications include hepatic encephalopathy [70], worsening liver function [72], liver ischemia [70], and shunt stenosis [70]. Pre-TIPS renal failure and high MELD scores were associated with worsening mortality [71] and clinical response was the strongest predictor of survival [71].

ManagementRecommendations

Figure 1 is a suggested stepwise algorithm for the management of HH. In the absence of definitive treatment and with the lack of high-quality evidence from randomized clinical trials, the management of refractory HH remains challenging and treatment needs to be individualized. For patients requiring frequent thoracentesis in the absence of associated refractory ascites, IPC is a viable option both as a bridge to transplantation and as a palliative procedure. IPC is also recommended for patients with contraindications for TIPS such as those with hepatic encephalopathy. TIPS should be offered to patients who also have concomitant refractory ascites or recurrent variceal bleeding, as it will also help in the management of these conditions. In centers of expertise, surgical intervention preferably via VATS for pleurodesis and diaphragmatic mesh repair, can be offered to patients with low surgical risk, less decompensated disease and good functional status.

Conclusion

HH is a complication of portal hypertension usually in the setting of decompensated cirrhosis related mainly to diaphragmatic defects and can frequently be complicated by spontaneous infection. Management starts with sodium restriction, but thoracentesis is often required for relief. More definitive management options for refractory cases includes TIPS, IPC, VATS with pleurodesis with variable success rates based on case series and retrospective cohorts. Randomized clinical trials in this setting are lacking and future research is needed.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflicts of interest.

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