Hepatic hydrothorax

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INTRODUCTION

Hepatic hydrothorax refers to the presence of a pleural effusion (usually >500 mL) in a patient with cirrhosis who does not have other reasons to have a pleural effusion (eg, cardiac, pulmonary, or pleural disease) [1-3]. Hepatic hydrothorax occurs in approximately 5 to 15 percent of patients with cirrhosis. Patients who develop hepatic hydrothorax are more likely to have ascites, hepatic encephalopathy, acute kidney injury (AKI), and increased risk of mortality. In a retrospective analysis of 495 patients with cirrhosis and pleural effusion, 16 percent had hepatic hydrothorax [4]. While patients with ascites can often tolerate up to 5 to 10 L of fluid with only mild symptoms, those with a pleural effusion can have severe symptoms (such as shortness of breath, cough, and hypoxemia) with as little as 500 mL of fluid.

This topic will review the clinical manifestations, diagnosis, and management of hepatic hydrothorax. Other complications of cirrhosis, including ascites and variceal hemorrhage, are discussed elsewhere. (See "Cirrhosis in adults: Overview of complications, general management, and prognosis" and "Ascites in adults with cirrhosis: Initial therapy" and "Ascites in adults with cirrhosis: Diuretic-resistant ascites" and "Primary prevention of bleeding from esophageal varices in patients with cirrhosis" and "Prevention of recurrent bleeding from esophageal varices in patients with cirrhosis".)

PATHOGENESIS

Although the exact mechanisms involved in the development of hepatic hydrothorax are incompletely understood, it probably results from the passage of ascites from the peritoneal cavity into the pleural cavity through small diaphragmatic defects. These defects are typically less than 1 cm (and may be microscopic) and are generally located in the tendinous portion of the diaphragm [5-10]. Hepatic hydrothorax becomes apparent when the absorptive capacity of the pleural space is exceeded. The pathologic mechanisms behind the formation of ascites are discussed in detail elsewhere. (See "Pathogenesis of ascites in patients with cirrhosis".)

The diaphragmatic defects are more often found in the right hemidiaphragm, likely due in part to the fact that the left hemidiaphragm is thicker and more muscular. Hepatic hydrothorax develops on the right side in approximately 73 to 85 percent of patients, on the left side in approximately 13 to 17 percent, and bilaterally in approximately 8 to 24 percent [3,4,11,12].

The negative intrathoracic pressure generated during inspiration promotes the passage of fluid from the abdominal cavity to the pleural space. The combination of increased intraabdominal pressure and a negative intrathoracic pressure facilitates movement of fluid from the peritoneal cavity into the pleural space through defects in the diaphragm [13]. This could explain why some patients with hepatic hydrothorax do not have apparent ascites [14-16]. This theory is supported by studies using 99mTc-human albumin or 99mTc-sulphur colloid, which demonstrate unidirectional passage of these markers from the abdominal cavity to the pleural cavity [14,17-21].

Similar to spontaneous bacterial peritonitis (SBP) in patients with ascites, patients with hepatic hydrothorax may develop spontaneous bacterial empyema (SBEM). It is thought to occur due to seeding of the pleural effusion with bacteria that spread directly from the abdominal cavity in a patient with SBP or from bacteremia. The etiologic agents in most cases are *Escherichia coli*, *Streptococcus* species, *Enterococcus*, *Klebsiella*, or *Pseudomonas* [22]. Approximately one-half of episodes are associated with SBP [23].

CLINICAL MANIFESTATIONS

Common clinical manifestations in patients with hepatic hydrothorax include dyspnea, a nonproductive cough, pleuritic chest pain, and fatigue due to hypoxemia [1,2,4,11,24,25]. Patients often have signs of ascites (eg, abdominal distension) as well as other findings associated with cirrhosis (eg, palmar erythema) [3]. Rarely, a pleural effusion may be an incidental finding in an asymptomatic patient. (See "Cirrhosis in adults: Etiologies, clinical manifestations, and diagnosis", section on 'Clinical manifestations'.)

Less common presentations include an acute tension hydrothorax with associated severe dyspnea and hypotension [26] or spontaneous bacterial empyema (SBEM). SBEM has been described in 13 to 16 percent of patients with hepatic hydrothorax [23,27]. Infection should be suspected in a patient with hepatic hydrothorax who develops fever, new or worsening pleuritic chest pain, encephalopathy, or an unexplained worsening of renal function. (See 'Thoracentesis and fluid testing' below.)

DIAGNOSIS

The diagnosis of hepatic hydrothorax includes documentation of a pleural effusion and exclusion of alternative causes for the effusion. A pleural effusion can usually be demonstrated on a frontal chest radiograph, although lateral views may be required if the effusion is small. Pleural effusions in patients with hepatic hydrothorax are typically found in the right hemithorax, though they may be left-sided or bilateral. A thoracentesis as well as additional imaging (eg, a chest computed tomographic scan and echocardiogram) should be performed to confirm the diagnosis and to exclude infection or an alternate cause of a pleural effusion. Up to 20 percent of patients will have evidence of infection or a cause of pleural effusion other than hepatic hydrothorax [28]. (See "Pleural fluid analysis in adults with a pleural effusion" and 'Pathogenesis' above.)

Thoracentesis and fluid testing — Patients with suspected hepatic hydrothorax should undergo thoracentesis with testing of the pleural fluid. (See "Ultrasound-guided thoracentesis".)

Diagnostic tests that should be performed on the pleural fluid include (table 1) [1,11,29-31]:

- Cell count and differential
- Gram stain
- Culture
- Protein, albumin, lactate dehydrogenase (LDH), and bilirubin concentrations
- Pleural fluid pH

In addition, serum albumin, LDH, and bilirubin concentrations should be determined.

When obtaining cultures, the fluid should be inoculated directly into a blood culture bottle at the bedside. In one study, the sensitivity for detecting spontaneous bacterial empyema (SBEM) using this method was 77 percent, compared with 33 percent for inoculations done once the fluid was received by the microbiology laboratory [23,30].

Other tests may be useful if certain diagnoses are suspected (see "Pleural fluid analysis in adults with a pleural effusion"):

- Triglyceride level (chylothorax)
- Polymerase chain reaction for *Mycobacterium* (tuberculosis)
- Amylase concentration (pancreatitis)
- Cytology (malignancy)

Pleural effusions deriving from portal hypertension are transudative in nature and therefore similar to the ascitic fluid, with a low protein concentration (<2.5 g/dL) [1] and with a serum-to-pleural fluid albumin gradient >1.1 g/dL [4].

However, there may be some differences in fluid composition between the pleural fluid and the ascitic fluid because the mechanisms of fluid absorption from the pleural space are different than those in the peritoneal cavity [1,24,29]. For instance, total protein and albumin may be slightly higher in hepatic hydrothorax compared with levels in the ascitic fluid [9].

As an example of pleural fluid findings in patients with hepatic hydrothorax, a retrospective study of 41 patients with hepatic hydrothorax found that 33 patients had solitary hydrothorax, and of these, 31 (94 percent) were transudative [30]. Sixteen (48 percent) had a total protein level <1.5 g/dL in the pleural fluid, none had a serum albumin value <1.5 g/dL, and microbiologic cultures were negative in 31 patients. The median pleural fluid pH was 7.49, total protein was 1.5 g/dL, and lactate dehydrogenase (LDH) was 65 international unit/L. The median pleural fluid:serum protein ratio and median pleural fluid LDH:upper limit of normal LDH ratio were 0.25 and 0.27, respectively.

In uncomplicated hepatic hydrothorax, the polymorphonuclear cell (PMN) count is low (<250 cells/mm^3), whereas in the setting of SBEM the PMN count is elevated [1,23].

The diagnostic criteria for SBEM are:

- Positive pleural fluid culture and a PMN cell count >250 cells/mm^3
- Negative pleural fluid culture and a PMN cell count >500 cells/mm^3
- No evidence of pneumonia on a chest imaging study

Data suggest that prompt testing of pleural fluid was associated with better outcomes. In an observational study including 54 patients with SBEM, early thoracentesis (ie, <24 hours from presentation) was associated with lower rates of mortality and intensive care unit (ICU) admission compared with delayed thoracentesis (7 versus 41 percent and 26 versus 56 percent, respectively) [32].

Imaging studies — Hepatic hydrothorax can typically be detected on a frontal chest radiograph. However, we generally recommend that patients also undergo a computed tomographic scan of the chest to exclude mediastinal, pulmonary, or pleural lesions. An abdominal ultrasound with Doppler study should be performed to examine the liver, rule out liver masses, ascertain the patency of the portal and hepatic veins, and detect ascites. Finally, echocardiography should be performed to rule out a cardiac cause for the pleural effusion. (See "Imaging of pleural effusions in adults" and "Diagnostic evaluation of the hemodynamically stable adult with a pleural effusion", section on 'Consider additional imaging'.)

In cases where the diagnosis is uncertain, an intraperitoneal injection of 99mTc-sulphur colloid or 99mTc-human serum albumin can be helpful. The radioisotopes migrate from the peritoneal cavity into the pleural space, therefore establishing a communication between both spaces and confirming the diagnosis of hepatic hydrothorax [14,17,33] (image 1). The optimal time to do an isotope study is shortly after therapeutic thoracentesis, when fluid is reaccumulating in the pleural cavity. When the chest is maximally filled with fluid, there may be minimal transfer of the isotope across the diaphragm, resulting in a false-negative study. Failure of the marker to show up in the pleural space (when the study has been performed shortly after thoracentesis) indicates an alternate cause of the pleural effusion.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of pleural effusions is broad and includes cardiopulmonary disease, chylothorax, tuberculosis, malignant effusions, and hemothorax (table 2 and table 3). Differentiating among the causes should take into account the patient's history, fluid analysis from a thoracentesis, and imaging test results. (See "Pleural fluid analysis in adults with a pleural effusion".)

Factors that suggest hepatic hydrothorax as the diagnosis include a history of cirrhosis (required), a transudative effusion, a right-sided effusion, and the simultaneous presence of ascites. However, even in the appropriate clinical setting, alternative diagnoses need to be excluded prior to making a diagnosis of hepatic hydrothorax. (See 'Diagnosis' above.)

MANAGEMENT

The management of patients with hepatic hydrothorax is typically multidisciplinary and includes input from hepatology, pulmonology, interventional radiology, and surgery. Treatment of hepatic hydrothorax is similar to the treatment of ascites [31]. Patients who are actively

consuming alcohol should be encouraged to abstain completely. Even if alcohol is not the only cause of their liver disease (eg, in patients with cirrhosis due to chronic hepatitis C), the alcohol-related component of their liver disease may improve dramatically with abstinence. Management then includes dietary sodium restriction and diuretics (algorithm 1). In addition, patients with confirmed hepatic hydrothorax should be referred for liver transplantation if they are otherwise suitable candidates. (See "Ascites in adults with cirrhosis: Initial therapy", section on 'Alcohol abstinence' and "Liver transplantation in adults: Patient selection and pretransplantation evaluation".)

Patients who are severely symptomatic should undergo a therapeutic thoracentesis followed by management with a sodium-restricted diet and diuretics (furosemide 40 mg and spironolactone 100 mg daily to start). Patients who are mildly to moderately symptomatic can be treated initially with sodium restriction and diuretics. Management options for patients who are refractory to sodium restriction and diuretics include serial thoracenteses, transjugular intrahepatic portosystemic shunt (TIPS) placement, pleurodesis, thoracoscopic surgery to repair diaphragmatic defects, and liver transplantation.

Chest tubes should not be placed for the treatment of hepatic hydrothorax (though they may be needed for patients with spontaneous bacterial empyema (SBEM) and frank pus, or in patients undergoing pleurodesis). Placement of chest tubes in patients with hepatic hydrothorax can result in massive protein and electrolyte depletion, infection, renal failure, and bleeding [34-36]. A review of cases from a tertiary care center over a 10-year period indicated that 16 out of 17 patients developed serious complications following chest tube placement [37]. These complications included acute kidney injury (AKI), pneumothorax, and empyema. Furthermore, once inserted, it may be impossible to remove the tube because of the continuous reaccumulation of fluid [36]. Other complications include subcutaneous emphysema; lung, spleen, liver, and stomach lacerations; hemothorax resulting from intercostal artery laceration; unilateral pulmonary edema from rapid removal of fluid; and placement of the tube into the abdominal cavity.

Chest tube insertion for management of hepatic hydrothorax has also been associated with an increased risk of mortality [38]. In a study of 1981 patients with hepatic hydrothorax, patients who had chest tube insertion had higher risk of mortality compared with patients who underwent thoracentesis (odds ratio [OR] 2.1, 95% CI 1.4-3.1) [38].

Sodium restriction — Education about the importance of dietary sodium restriction is a central component of the management of hepatic hydrothorax. Patients should be advised to consume no more than 88 mEq (2000 mg) of sodium per day (figure 1 and table 4). This level of sodium restriction can be achieved in an outpatient setting without the purchase of special

foods and is the most practical and successful approach to sodium restriction [1,24]. Patients should also be instructed to avoid nonsteroidal antiinflammatory drugs, which can cause sodium retention and renal failure. (See "Ascites in adults with cirrhosis: Initial therapy", section on 'Dietary sodium restriction'.)

Diuretics — Sodium restriction alone will be effective only in the small subset of patients whose urinary sodium excretion is more than 78 mEq/day (88 mEq intake minus 10 mEq of nonurinary losses). Most patients will also require therapy with diuretics. Patients should be started on furosemide 40 mg daily and spironolactone 100 mg daily. If there is no response, the doses of the diuretics may be increased in a stepwise fashion every three to five days by doubling the doses (maintaining a ratio of 40 mg:100 mg). The maximum doses are furosemide 160 mg daily and spironolactone 400 mg daily. (See "Ascites in adults with cirrhosis: Initial therapy", section on 'Diuretic therapy'.)

Refractory hydrothorax — Patients who have persistent hydrothorax despite a sodium-restricted diet and diuretic therapy or who develop diuretic-related complications are considered to have refractory hydrothorax. Small series suggest that 20 to 25 percent of patients have refractory hydrothorax [39,40].

It is important to exclude noncompliance with dietary sodium restriction and/or diuretics prior to diagnosing refractory hydrothorax. In addition to the history, measurement of 24-hour urinary sodium excretion can be helpful for determining adherence to management recommendations. (See "Ascites in adults with cirrhosis: Initial therapy", section on 'Diuretic resistance'.)

Management options for patients who are considered to be truly refractory include repeated thoracenteses, TIPS placement, pleurodesis, surgical repair of defects in the diaphragm, and liver transplantation. Our approach is to start with repeated thoracenteses. However, for patients who require thoracentesis every two to three weeks, alternative treatments should be considered because these patients are at increased risk of having adverse events from repeated thoracentesis. For such patients, we typically suggest TIPS placement if their Child-Pugh score is <13 (calculator 1 and calculator 2), they are younger than 70 years of age, and they do not have hepatic encephalopathy. For patients who are not good candidates for TIPS placement, we will consider pleurodesis or thoracoscopic repair of the diaphragmatic defect.

Data suggest that refractory hepatic hydrothorax is associated with increased mortality risk. In a retrospective study including 47 patients with refractory hepatic hydrothorax who were compared with 47 patients with refractory ascites who were matched for age, sex, and model

for end-stage liver disease (MELD)-Na score, refractory hepatic hydrothorax was associated with higher mortality rates at one year (51 versus 19 percent) [41].

Thoracentesis — Therapeutic thoracentesis is the most effective way to reduce large effusions (greater than 1.5 L) [1,11,29]. After thoracentesis, diuretics should be continued to prevent reaccumulation of fluid. As a general rule, no more than 2 L of fluid should be removed because of the risk of pulmonary edema and hypotension [42]. However, this "2 L rule" was developed in patients with causes other than cirrhosis for their effusions; patients with cirrhosis may be able to tolerate the removal of larger volumes of fluid. The amount of fluid present prior to thoracentesis can be estimated by chest radiograph or computed tomographic scan. (See "Large volume (therapeutic) thoracentesis: Procedure and complications" and "Imaging of pleural effusions in adults".)

Complications of thoracentesis include pain at the puncture site, pneumothorax, hemothorax, vasovagal episodes, hemoptysis, air embolism, laceration of the liver or spleen, empyema, hemoptysis, and subcutaneous emphysema [43]. The probability of complications increases in patients who require frequent therapeutic thoracenteses (eg, every two to three weeks). The alternative approaches outlined below should be considered for these patients. While performing frequent thoracentesis may relive symptoms, it is not generally used as a long-term strategy because the benefit of such an approach is uncertain [44].

Coagulopathy is not considered a contraindication to therapeutic thoracentesis. One study demonstrated no increased risk of bleeding in patients with prothrombin times up to twice the midpoint of the normal range, or platelet counts of more than 50,000/microL; the authors concluded that prophylactic transfusions are not necessary [45]. Patients with a serum creatinine more than 6 mg/dL had greater blood loss compared with those with lower values.

Transjugular intrahepatic portosystemic shunt — Several groups have reported a beneficial effect of TIPS placement in patients with hepatic hydrothorax. Unfortunately, few of the studies describe how the refractoriness of the hydrothorax to medical therapy was determined. Despite this shortcoming, most series have demonstrated response rates in the range of 70 to 80 percent [46-52]. However, TIPS placement is associated with serious complications including technical complications (eg, cardiac arrhythmias, traversal of the liver capsule), portosystemic encephalopathy, and TIPS stenosis. Because of the potential risks of TIPS placement, we generally reserve it for patients with a Child-Pugh score ≤13 (calculator 1 and calculator 2) who do not have contraindications to TIPS. The pre-TIPS evaluation is discussed separately. (See "Overview of transjugular intrahepatic portosystemic shunts (TIPS)".)

A study that reviewed the course after TIPS placement in 28 patients with hepatic hydrothorax revealed that the 30-, 90-, and 150-day mortality rates were 14, 25, and 53 percent, respectively [53]. One-year survival without liver transplantation was 41 percent. In 68 percent of patients there was complete resolution of symptoms, and in 57 percent of patients there was disappearance of effusions on radiograph. TIPS placement was successful in 15 of 20 patients with a Child-Pugh score ≤10 and in only one of eight patients with a Child-Pugh score >10. In a meta-analysis of six studies that included a total of 198 patients with a mean duration of follow-up of 10 months, complete response to TIPS was seen in 56 percent (95% CI 45-67 percent) and a partial response in 18 percent (95% CI 11-24 percent) [54]. The incidence of hepatic encephalopathy was 12 percent (95% CI 6.3-17 percent), and the 45-day mortality was 18 percent (95% CI 11-24 percent). The mortality rate in refractory hepatic hydrothorax is comparable with those seen in patients with refractory ascites. The most important predictors of poor outcomes after TIPS in this population are older age, severe underlying liver disease, and/or associated renal dysfunction. In an observational study including 51 patients with hepatic hydrothorax who underwent TIPS, the mean portal pressure gradient was reduced from 23 mmHg to 7 mmHg, while at 6 months, 10 patients (20 percent) had complete resolution of hepatic hydrothorax [55]. Serious complications reported within 30 days after TIPS placement included portosystemic encephalopathy (eight patients [16 percent]), ischemic hepatitis (four patients [8 percent]), and mortality (9 patients [18 percent]).

Risk factors for mortality after TIPS placement for hepatic hydrothorax include a Child-Pugh score >10, a pre-TIPS MELD score >15 to 17, and an elevated pre-TIPS creatinine [52,53]. In addition, a lack of response in the hydrothorax after TIPS placement is associated with an increased mortality rate [52]. These findings have suggested that TIPS should be considered early given the high morbidity and mortality in this population.

Pleurodesis — Chemical pleurodesis is commonly used to treat patients with recurrent, symptomatic pleural effusions due to malignancy. However, little information is available to guide the clinician in the management of recurrent, symptomatic, nonmalignant pleural effusions, even though both chemical pleurodesis and talc poudrage have been used in this setting. (See "Chemical pleurodesis for the prevention of recurrent pleural effusion".)

Clinical experience suggests that hepatic hydrothorax is the most difficult form of nonmalignant pleural effusion to treat with chemical pleurodesis. Because of the rapid migration of fluid from the abdomen into the pleural space, it is often difficult to keep the two pleural surfaces apposed long enough for the inflammatory process to result in pleural symphysis [22]. Thus, it is rarely performed and is typically reserved for patients for whom no other options exist.

In addition to the challenge of achieving pleural symphysis, multiple adverse events (eg, fever, renal failure, pneumothorax, hepatic encephalopathy) have been reported in case series of patients who have undergone pleurodesis for hepatic hydrothorax [56-59]. In a meta-analysis of 13 studies including 180 patients who underwent pleurodesis by chemical and/or mechanical means (ie, conventional thoracoscopy or video-assisted thoracic surgery), the pooled rate of complete response was 72 percent (95% CI 65-79 percent) [59]. Complications related to chemical pleurodesis were reported in six studies including 63 patients, and the pooled incidence of adverse events was 82 percent (95% CI 66-94 percent). (See "Medical thoracoscopy (pleuroscopy): Equipment, procedure, and complications".)

Thoracoscopic repair — Thorascopic repair of the diaphragmatic defects seen in patients with hepatic hydrothorax may be possible. A diaphragmatic repair involving a pleural flap and surgical mesh reinforcement has been described, but experience is limited [60]. A retrospective analysis of 63 patients with refractory hepatic hydrothorax who underwent thoracoscopic mesh onlay reinforcement to repair diaphragmatic defects found that after a median 20.5 months, only four patients experienced recurrence [61]. Surgical repair of diaphragmatic defects prevents unidirectional shifting of fluid into the thoracic cavity from the abdomen. However, surgical repair has been associated with high mortality in patients with decompensated cirrhosis; thus, this approach may be an option for patients who are not candidates for TIPS but have low MELD scores. The procedure can be performed with video-assisted thoracic surgery with or without mesh, while the type of diaphragmatic defect may influence the repair strategy [61].

Liver transplantation — Patients with hepatic hydrothorax typically have end-stage liver disease, and liver transplantation is the definitive treatment for hepatic hydrothorax. Patients with hepatic hydrothorax who do not have contraindications to liver transplantation should be referred for a transplantation evaluation. The outcomes in patients with hepatic hydrothorax who undergo liver transplantation are similar to those in patients who undergo liver transplantation for other reasons [62]. (See "Liver transplantation in adults: Patient selection and pretransplantation evaluation".)

Other treatment options — Medications that cause splanchnic vasoconstriction can improve the underlying hemodynamic derangements that lead to extracellular fluid accumulation in cirrhosis, can cause increased natriuresis, and theoretically can improve the action of diuretics in order to maximize fluid mobilization.

A case report described successful treatment of a patient with a refractory hepatic hydrothorax despite TIPS placement and pleurodesis who was treated with intravenous octreotide (at a dose of 25 mcg/hour on the first day, 50 mcg/hour on the second day, and then 100 mcg/hour for

five more days) [63]. Another report described successful treatment of hepatic hydrothorax in a patient with concomitant hepatorenal syndrome after the administration of terlipressin (1 mg intravenous bolus injection every six hours along with intravenous albumin infusion of 40 g daily for five days) [64]. It is unclear whether these approaches will lead to a sustained benefit.

Chest tube placement has been avoided due to the risk of complications and poor outcomes [34-36]. However, indwelling pleural catheters (IPCs) have been studied as a possible treatment option for patients with refractory hepatic hydrothorax. In a retrospective study of 62 patients with hepatic hydrothorax who underwent IPC placement, adverse events (eg, empyema) occurred in 22 patients (36 percent), while 10 patients (16 percent) underwent successful liver transplantation following IPC placement [65]. Additional studies are needed to examine the outcomes and risks associated with IPC for patients with refractory hepatic hydrothorax.

Treatment of infection — SBEM has been described in 13 to 16 percent of patients with hepatic hydrothorax. The etiologic agents in most cases are *E. coli*, *Streptococcus* species, *Enterococcus*, *Klebsiella*, or *Pseudomonas* [22]. If infection is suspected, therapy with an intravenous antibiotic should be started after the pleural fluid has been sampled for cell count and differential, Gram stain, and culture [23]. (See 'Thoracentesis and fluid testing' above.)

The diagnostic criteria for SBEM are:

- Positive pleural fluid culture and a polymorphonuclear cell (PMN) cell count >250 cells/mm^3
- Negative pleural fluid culture and a PMN cell count >500 cells/mm^3
- No evidence of pneumonia on a chest imaging study

The antibiotics used to treat SBEM are the same as those used for the treatment of spontaneous bacterial peritonitis (SBP; typically, a third generation cephalosporin such as ceftriaxone 2 g every 24 hours for 7 to 10 days) [66]. Intravenous levofloxacin is an alternative for patients who are allergic to penicillin. Antibiotic therapy can be tailored once results from antibiotic sensitivity testing are available. In patients in whom there is slow clinical recovery, a repeat thoracentesis is useful to document that the patient is responding. (See "Spontaneous bacterial peritonitis in adults: Treatment and prophylaxis", section on 'Selecting empiric therapy' and "Epidemiology, clinical presentation, and diagnostic evaluation of parapneumonic effusion and empyema in adults".)

Mortality rates in patients with SBEM are as high as 20 percent, even with antimicrobial therapy [23,25,27]. Independent risk factors associated with poor outcomes in SBEM include a high MELD-Na score, initial intensive care unit admission, and initial antibiotic treatment failure [27]. (See "Model for End-stage Liver Disease (MELD)".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Cirrhosis".)

SUMMARY AND RECOMMENDATIONS

- **Background –** Hepatic hydrothorax refers to the presence of a pleural effusion (usually >500 mL) in a patient with cirrhosis who does not have other reasons to have a pleural effusion (eg, cardiac, pulmonary, or pleural disease). Hepatic hydrothorax occurs in approximately 5 to 15 percent of patients with cirrhosis. (See 'Introduction' above.)

- **Pathogenesis –** Although the exact mechanisms involved in the development of hepatic hydrothorax are incompletely understood, it probably results from the passage of ascites from the peritoneal cavity into the pleural cavity through small diaphragmatic defects. (See 'Pathogenesis' above.)

- **Clinical features –** Common clinical manifestations in patients with hepatic hydrothorax include dyspnea, a nonproductive cough, pleuritic chest pain, and fatigue due to hypoxemia. Patients often have signs of ascites (eg, abdominal distension) as well as other findings associated with cirrhosis (eg, palmar erythema). Rarely, a pleural effusion may be an incidental finding in an asymptomatic patient. (See 'Clinical manifestations' above.)

- **Diagnosis –** The diagnosis of hepatic hydrothorax includes documentation of a pleural effusion and exclusion of alternative causes for the effusion. A pleural effusion can usually be demonstrated on a frontal chest radiograph, although lateral views may be required if the effusion is small. Pleural effusions in patients with hepatic hydrothorax are typically found in the right hemithorax, though they may be left-sided or bilateral. A thoracentesis as well as additional imaging (eg, a chest computed tomographic scan and echocardiogram) should be performed to confirm the diagnosis and to exclude infection or an alternate cause of the effusion (table 2 and table 3). Up to 20 percent of patients will have evidence of infection or a cause of pleural effusion other than hepatic hydrothorax. (See 'Diagnosis' above.)

  - Diagnostic tests that should be performed on the pleural fluid include (table 1) (see 'Thoracentesis and fluid testing' above):

    - Cell count and differential

- Gram stain
- Culture (using blood culture bottles that are inoculated at the bedside)
- Protein, albumin, lactate dehydrogenase (LDH), and bilirubin concentrations
- Pleural fluid pH

In addition, serum albumin, LDH, and bilirubin concentrations should be determined.

- Other tests may be useful if certain diagnoses are suspected (see "Pleural fluid analysis in adults with a pleural effusion"):

  - Triglyceride level (chylothorax)
  - Polymerase chain reaction for mycobacterium (tuberculosis)
  - Amylase concentration (pancreatitis)
  - Cytology (malignancy)

- **Management –** Treatment of hepatic hydrothorax is similar to the treatment of ascites (algorithm 1) (see "Ascites in adults with cirrhosis: Initial therapy" and 'Management' above):

  - Patients who are actively consuming alcohol should be encouraged to abstain completely. Even if alcohol is not the only cause of their liver disease (eg, in patients with cirrhosis due to chronic hepatitis C), the alcohol-related component of their liver disease may improve dramatically with abstinence. (See "Ascites in adults with cirrhosis: Initial therapy", section on 'Alcohol abstinence'.)

  - We suggest that patients with hepatic hydrothorax are initially treated with a sodium-restricted diet and diuretics rather than a sodium-restricted diet alone (**Grade 2C**). Sodium restriction alone will be effective only in the small subset of patients whose urinary sodium excretion is more than 78 mEq/day (88 mEq intake minus 10 mEq of nonurinary losses). Patients should be started on furosemide 40 mg daily and spironolactone 100 mg daily. If there is no response, the doses of the diuretics may be increased in a stepwise fashion every three to five days by doubling the doses (maintaining a ratio of 40 mg:100 mg). The maximum doses are furosemide 160 mg daily and spironolactone 400 mg daily. (See "Ascites in adults with cirrhosis: Initial therapy", section on 'Dietary sodium restriction' and "Ascites in adults with cirrhosis: Initial therapy", section on 'Diuretic therapy'.)

    For patients who are severely symptomatic, a therapeutic thoracentesis can be performed along with the initiation of a sodium-restricted diet and diuretics to provide

more rapid fluid removal. (See "Large volume (therapeutic) thoracentesis: Procedure and complications".)

- Options for patients who are refractory to sodium restriction and diuretics include serial thoracenteses, transjugular intrahepatic portosystemic shunt (TIPS) placement, pleurodesis, thoracoscopic surgery for diaphragmatic repair, and liver transplantation. Our approach is to start with repeated thoracenteses. However, for patients who require thoracentesis every two to three weeks, alternative treatments should be considered because these patients are at increased risk of having adverse events from repeated thoracentesis. For such patients, we typically pursue TIPS placement if their Child-Pugh score is <13 (calculator 1 and calculator 2), they are younger than 70 years of age, and they do not have hepatic encephalopathy. Alternative treatments for patients who are not good candidates for TIPS placement include pleurodesis or thoracoscopic repair of the diaphragmatic defect. (See 'Refractory hydrothorax' above.)

- Chest tubes should **not** be placed for the treatment of hepatic hydrothorax. Placement of chest tubes in patients with hepatic hydrothorax can result in massive protein and electrolyte depletion, infection, renal failure, and bleeding. In addition, once inserted, it may be impossible to remove a chest tube because of the continuous reaccumulation of fluid.

- Patients with hepatic hydrothorax should be referred for liver transplantation if they are otherwise suitable candidates. (See "Liver transplantation in adults: Patient selection and pretransplantation evaluation".)

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Topic 1242 Version 22.0

GRAPHICS

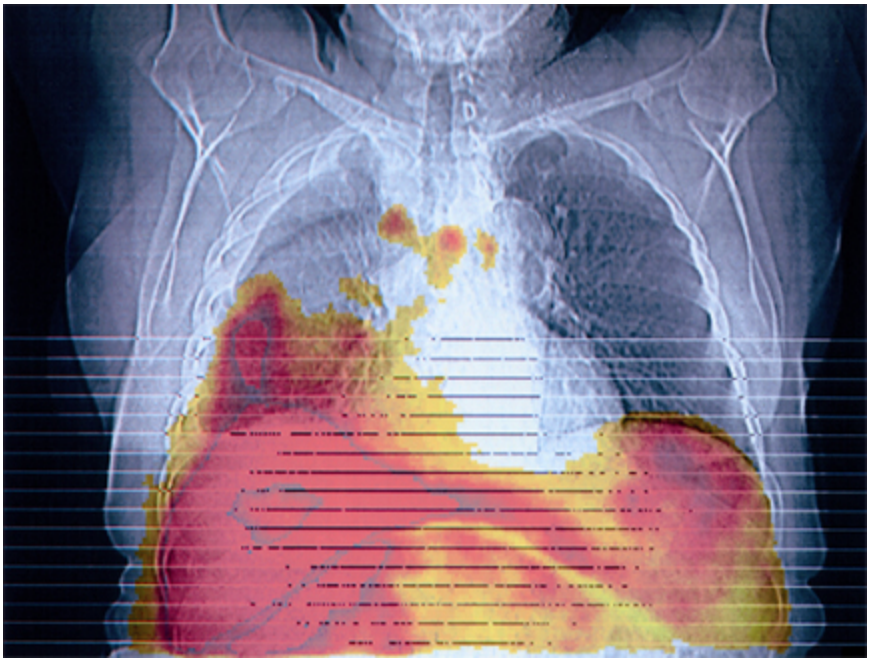
Characteristics of hepatic hydrothorax

Location
Right side (73 to 85%)
Left side (13 to 17%)
Bilateral (2 to 10%)
Fluid
Cell count <250 polymorphonuclear cells per mm^3
Protein <2.5 g/dL
Pleural fluid/serum total protein ratio <0.5
Pleural fluid/serum lacate dehydrogenase ratio <0.6
Serum-to-pleural fluid albumin gradient >1.1 g/dL
Pleural fluid/serum bilirubin ratio <0.6
pH >7.4
Glucose level similar to that of serum

Courtesy of Andres Cardenas, MD.

Graphic 52482 Version 4.0

Hepatic hydrothorax



Intraperitoneal injection of 500 MBq 99mTc-tagged nannocolloids, followed by scintigrams taken between 5 minutes and 24 hours post injection, demonstrated a transdiaphragmatic tracer flow into the right pleural cavity.

Reproduced with permission from: Truninger K, Frey LD. Hepatic hydrothorax without ascites. Schweiz Med Wochenschr 2000; 130:1706.

Graphic 64287 Version 3.0

Diagnoses established "definitively" by pleural fluid analysis

Disease	Diagnostic pleural fluid tests
Empyema	Observation (pus, putrid odor), positive culture
Malignancy	Positive cytology
Tuberculous pleurisy	Positive AFB stain, culture
Esophageal rupture	High salivary isoenzyme form of amylase, low pH (often as low as 6), ingested vegetable or meat fragments
Fungal-related effusions	Positive fungal stain, culture
Chylothorax	Triglycerides >110 mg/dL, chylomicrons by lipoprotein electrophoresis
Cholesterol effusion	Cholesterol >200 mg/dL with a cholesterol to triglyceride ratio >1, cholesterol crystals under polarizing light
Hemothorax	Ratio of pleural fluid to blood hematocrit >0.5
Urinothorax	Pleural fluid creatinine to serum ratio always >1 but diagnostic if >1.7
Peritoneal dialysis	Protein <0.5 mg/dL and pleural fluid to serum glucose ratio >1 in peritoneal dialysis patient
Extravascular migration or misplacement of a central venous catheter	Pleural fluid to serum glucose ratio >1, pleural fluid gross appearance mirrors infusate (eg, milky white if lipids infused)
Rheumatoid pleurisy	Cytologic evidence of elongated macrophages and distinctive multinucleated giant cells (tadpole cells) in a background of amorphous debris
Glycinothorax	Measurable glycine after bladder irrigation with glycine-containing solutions
Cerebrospinal fluid leakage into pleural space	Detection of beta-2 transferrin
Parasite-related effusions	Detection of parasites

Graphic 50027 Version 4.0

Observations of pleural fluid helpful in diagnosis

	Suggested diagnosis
Color of fluid	
Pale yellow (straw)	Transudate, some exudates
Red (bloody)	Malignancy, benign asbestos pleural effusion, postcardiac injury syndrome, or pulmonary infarction in absence of trauma
White (milky)	Chylothorax or cholesterol effusion
Brown	Long-standing bloody effusion; rupture of amebic liver abscess
Black[1-4]	Aspergillus niger, Rhizomes oryzae, metastatic melanoma, pancreaticopleural fistula, crack cocaine use, bronchogenic adenocarcinoma, esophageal perforation during treatment with activated charcoal, chronic hemothorax
Yellow-green	Rheumatoid pleurisy
Dark green	Biliothorax
Color of:	
Enteral tube feeding	Feeding tube has entered pleural space
Central venous catheter infusate	Extravascular catheter migration
Character of fluid	
Pus	Empyema
Viscous	Mesothelioma
Debris	Rheumatoid pleurisy
Turbid	Inflammatory exudate or lipid effusion
Anchovy paste	Amebic liver abscess
Odor of fluid	
Putrid	Anaerobic empyema
Ammonia	Urinothorax

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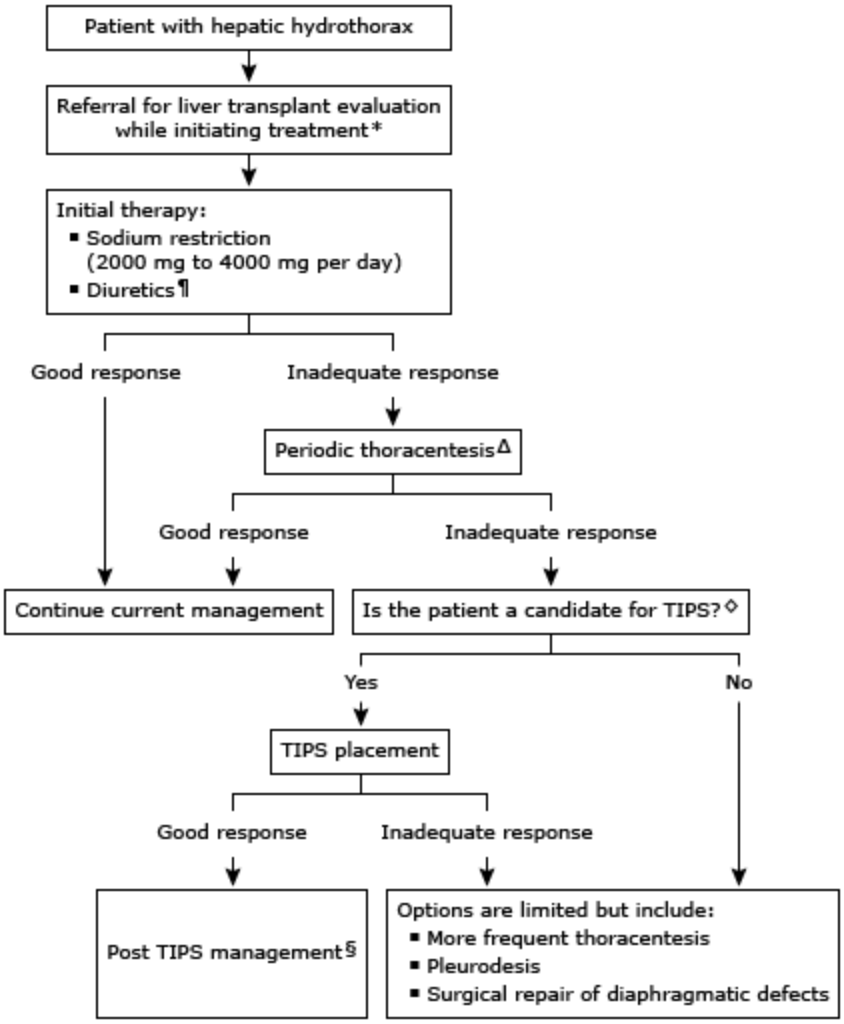
Graphic 74757 Version 2.0

Management of hepatic hydrothorax



This flowchart does not substitute for the clinical judgment of the treating specialist. Refer to UpToDate content on managing patients with hepatic hydrothorax and related topics.

TIPS: Transjugular intrahepatic portosystemic shunt.

* Patients with hepatic hydrothorax who do not have contraindications to liver transplantation should be referred for a transplantation evaluation.

¶ Initial diuretic doses are furosemide 40 mg daily and spironolactone 100 mg daily. If there is no response, diuretics may be increased in a stepwise fashion every three to five days by doubling the doses. Maximum doses are furosemide 160 mg daily and spironolactone 400 mg daily.

Δ Therapeutic thoracentesis can be performed periodically in conjunction with diuretic therapy.

◇ We generally reserve TIPS for patients with Child A or Child B cirrhosis, who are younger than 70 years of age, and who do not have hepatic encephalopathy. A cardiac evaluation should be performed in patients with signs, symptoms or a history of heart failure, tricuspid regurgitation, cardiomyopathy or pulmonary hypertension.

§ We monitor TIPS patency with Doppler ultrasound between three and six months after placement and then at six month intervals for the first two years.

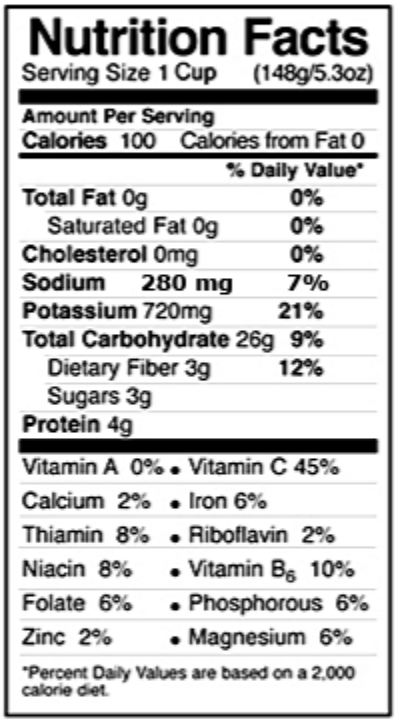
Graphic 80148 Version 5.0

Low-sodium diet

A low sodium diet is one that includes less than 2 grams (2000 milligrams) of sodium each day. The main source of sodium in the diet is table salt, which is often added when foods are processed, prepared, or just before eating. One teaspoon of salt contains about 2300 mg of sodium.

Sodium also occurs naturally in some foods. To determine the amount of sodium that a food contains, consult a reference book; one suggestion is Bobbie Mostyn's *Pocket Guide to Low Salt Foods*. Many websites also provide nutrient data (eg, www.nutrition.gov). Low sodium cookbooks are also available.

On food labels, the amount of sodium in food is listed on the nutrition label:



Nutrition Facts

Serving Size 1 Cup (148g/5.3oz)

Amount Per Serving	
Calories 100	Calories from Fat 0
	% Daily Value*
Total Fat 0g	**0%**
Saturated Fat 0g	**0%**
Cholesterol 0mg	**0%**
Sodium 280 mg	**7%**
Potassium 720mg	**21%**
Total Carbohydrate 26g	**9%**
Dietary Fiber 3g	**12%**
Sugars 3g	
Protein 4g	

Vitamin A 0%	• Vitamin C 45%
Calcium 2%	• Iron 6%
Thiamin 8%	• Riboflavin 2%
Niacin 8%	• Vitamin B_6 10%
Folate 6%	• Phosphorous 6%
Zinc 2%	• Magnesium 6%

*Percent Daily Values are based on a 2,000 calorie diet.

1 serving (1 cup) contains 280 mg of sodium

Decrease the amount of salt you eat gradually. Many people find that they do not miss salt after a few weeks of eating less.

Suggestions to decrease sodium include the following:

- Do not add salt to food while cooking or before eating. Teach family members to taste food before adding salt.
- Avoid eating at fast food restaurants. If this is not possible, choose restaurants that offer fruits or vegetables without sauces or dressings. Ask that no salt be used to prepare food, when possible.
- Do not use salt substitutes unless a healthcare provider approves. Herb and spice combinations that do not contain salt can be used to flavor foods.
- Water softeners remove calcium and add sodium to drinking water. Do not drink softened water. When purchasing bottled water, check the label to ensure that it does not contain sodium.
- Look at labels for over the counter medications. Avoid products that contain sodium carbonate and sodium bicarbonate. Sodium bicarbonate is baking soda.
- Look for food labels that say "no salt added", "low salt", or "low sodium". Item that are labeled low salt/sodium must have less than 140 mg of sodium per serving (check the serving size!)

Graphic 51602 Version 2.0

Low-sodium diet (continued)

Foods to choose	Foods to avoid
Breads:	
Whole-grain breads, English muffins, bagels	Biscuits, prepared mixes (pancake, muffin, cornbread)
Cereals:	
Cooked hot cereals (not instant), such as oatmeal, cream of wheat, rice, or farina; puffed wheat; puffed rice; shredded wheat	Instant hot cereals, many boxed cold cereals
Crackers and snack foods:	
All unsalted crackers and snack foods, unsalted peanut butter, unsalted nuts or seeds	Salted crackers and snack items (chips, pretzels, popcorn), regular peanut butter, prepared dips/spreads, salted nuts or seeds
Pasta, rice, and potatoes:	
Any type of pasta; potatoes; white or brown rice	Macaroni and cheese mix; rice, noodle, or spaghetti mixes; canned spaghetti; frozen lasagna; instant potatoes; seasoned potato mixes
Dried beans and peas:	
Any dried beans or peas without seasoning	Beans or peas prepared with ham, bacon, salt pork, or bacon grease; all canned beans
Meats and alternatives:	
Fresh or frozen beef, poultry, and fish; low-sodium canned tuna and salmon; eggs	Salted, smoked, canned, spiced, and cured meat, poultry, or fish; bacon; ham; sausage; lunch meats; hot dogs; breaded frozen meat, fish, or poultry; frozen dinners; pizza
Fruits and vegetables:	
Any fresh, frozen, or canned fruit, any fresh or frozen vegetables without sauce, canned vegetables without salt, low-salt tomato sauce/paste	Regular canned vegetables and vegetable juices, regular tomato sauce and tomato paste, olives, pickles, relishes, sauerkraut, frozen vegetables in butter or sauces, crystallized and glazed fruit, maraschino cherries, fruit dried with sodium sulfite
Dairy products:	
Milk, cream, sour cream, non-dairy creamer, yogurt, low-	Buttermilk, Dutch processed chocolate milk, processed cheese slices and spreads, processed cheese, cottage cheese, aged or natural cheese

sodium cottage cheese, low-sodium cheese	
Fats and oils:	
Plant oils (olive, canola, corn, peanut), unsalted butter or margarine	Prepared salad dressings, bacon, salt pork, fat back, salted butter or margarine
Soups:	
Salt-free soups and low-sodium bouillon cubes, unsalted broth, homemade soup without added salt	Regular canned or prepared soups, stews, broths, or bouillon; packaged and frozen soups
Desserts:	
Gelatin, sherbet, pudding, ice cream, salt-free baked goods, sugar, honey, jam, jelly, marmalade, syrup	Packaged baked goods
Beverages:	
Coffee, tea, soft drinks, fruit-flavored drinks, low-salt tomato juice, any fruit juice	Softened water; carbonated beverages with sodium or salt added; regular tomato juice (V-8); ask about alcoholic beverages
Condiments:	
Fresh and dried herbs; lemon juice; low-salt mustard, vinegar, Tabasco sauce; low- or no-salt ketchup; seasoning blends that do not contain salt	Table salt, lite salt, bouillon cubes, meat extract, taco seasoning, Worcestershire sauce, tartar sauce, ketchup, chili sauce, cooking sherry and wine, onion salt, mustard, garlic salt, soy sauce, tamari, meat flavoring or tenderizer, steak and barbecue sauce, seasoned salt, monosodium glutamate (MSG), Dutch processed cocoa

Graphic 63387 Version 1.0

Contributor Disclosures

Andrés Cárdenas, MD, PhD, MMSc, AGAF, FAASLD Grant/Research/Clinical Trial Support: Boston Scientific Corporation [Endoscopy in biliary complications after liver transplantation]. Consultant/Advisory Boards: Boston Scientific Corporation [Endoscopy in biliary complications after liver transplantation]. All of the relevant financial relationships listed have been mitigated. **T Barry Kelleher, MD, FRCPI** No relevant financial relationship(s) with ineligible companies to disclose. **Sanjiv Chopra, MD, MACP** No relevant financial relationship(s) with ineligible companies to disclose. **Bruce A Runyon, MD, FAASLD** No relevant financial relationship(s) with ineligible companies to disclose. **Kristen M Robson, MD, MBA, FACG** No relevant financial relationship(s) with ineligible companies to disclose.

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