Hepatic Hydrothorax

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KEYWORDS

- Hepatic hydrothorax • Thoracentesis
- Transjugular intrahepatic portosystemic shunt • Cirrhosis
- Spontaneous bacterial empyema

KEY POINTS

- Hepatic hydrothorax is an uncommon complication of portal hypertension defined as a transudative pleural effusion in absence of cardiopulmonary pathology and is usually left-sided.
- Early diagnosis via pleural fluid sampling is essential to rule out other causes, which are found in up to 30% of suspected hepatic hydrothorax cases, and to diagnosis spontaneous bacterial empyema.
- Spontaneous bacterial empyema is under-recognized, present in up to 16% of patients with hepatic hydrothorax, and it is associated with a mortality rate of over 20%.
- In diuretic-refractory cases, thoracentesis is a main stay of treatment but is associated with complications. Both transjugular intrahepatic portosystemic shunt and other surgical procedures are considered bridging measures to liver transplantation in select patients, although management remains challenging and are frequently associated with poor outcomes.

INTRODUCTION: HEPATIC HYDROTHORAX

Hepatic hydrothorax (HH) is a relatively uncommon complication in patients with end-stage liver disease, and it is defined as a transudative pleural effusion usually greater than 500 mL in a patient with portal hypertension without any other underlying primary cardiopulmonary source.[1–3] Although approximately 50% of patients with end-stage liver disease will develop ascites, only 5% to 10% develop hepatic hydrothorax, which may result in dyspnea, hypoxia, and infection, and portends a poor prognosis. The most likely explanation for development is passage of fluid from the peritoneal space to the pleural space due to small diaphragmatic defects. Initial management consists of diuretics with dietary sodium restriction and thoracentesis, and a transjugular intrahepatic portosystemic shunt (TIPS) may ultimately be required. Despite its relative

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infrequency, afflicted patients can be quite symptomatic, develop morbid and fatal complications, pose management dilemmas, and should warrant evaluation for liver transplantation.

CLINICAL FEATURES

Epidemiology

The largest case series have described the incidence of hepatic hydrothorax to be approximately 5% to 10% of cirrhotic patients.[4–6] It is presumably more common in later stages of cirrhosis and has been estimated to be present in 4% to 6% of all patients with cirrhosis and in up to 10% of decompensated patients.[7] The estimation of the incidence is also affected by the sensitivity of detection as illustrated by a study of 862 cirrhotic patients requiring hospital admission that were evaluated with radiography, ultrasound, and computed tomography (CT), which found 15% had pleural effusions, although only 6.5% of patients had enough fluid to perform a thoracentesis.[8] Additionally, persistent massive ascites and HH have been reported in 2% of liver transplant recipients, all of whom had hepatitis C virus.[9]

Clinical Manifestations and Complications

Presentation

HH should always be suspected when a cirrhotic patient develops a pleural effusion. Most patients will first present with clinical signs and symptoms of cirrhosis and portal hypertension, although in other patients, pulmonary symptoms may dominate the clinical presentation. Although 5 to 8 L of ascites can accumulate in the abdominal cavity with only mild symptoms, relatively small amounts of fluid in the thoracic cavity (1–2 L) can cause severe symptoms such as shortness of breath, nonproductive cough, chest discomfort or tightness, and hypoxia. Because the development of HH is thought to be related to ascites, most patients will have concurrent ascites; however, it may not be detectable in 21% of patients.[1] Multiple clinical factors such as volume, rapidity of accumulation, and presence of associated pulmonary disease determine the severity of symptoms, ranging from a lack of symptoms to life-threatening respiratory failure.[1] In addition to dyspnea and hypoxia, patients with HH may develop further complications such as acute tension hydrothorax and infection, called spontaneous bacterial empyema.

A rarely reported presentation of HH is acute tension hydrothorax, which is associated with severe dyspnea and hypotension. It has been reported to occur acutely over the course of an hour and may be secondary to a sudden pleuroperitoneal bleb.[10]

Spontaneous bacterial empyema

Spontaneous bacterial empyema (SBEM) is an important and under-recognized distinct complication of HH and is defined as a spontaneous infection of a preexisting HH. The name is somewhat misleading, as there is usually no pus or abscess in the thoracic cavity, and the pathogenesis, course, and treatment are very different from empyema secondary to pneumonia. Although SBEM and spontaneous bacterial peritonitis (SBP) are closely related, SBEM is rarely described, and there are only a handful of dedicated studies describing the condition. Two studies from Taiwan found the overall incidence of SBEM to be 2% of all cirrhotic patients and 13% to 16% among cirrhotic patients with HH, which is similar to the prevalence of SBP in patients with ascites.[2,3] SBEM can easily be confused with a pleural empyema, as there is usually no evidence of pus or abscess formation in the thoracic cavity. The pathogenesis of SBEM remains unclear; one hypothesis postulates that pleural infection is caused by flow of infected ascites from the peritoneal to the pleural cavity via defects in the