International Journal of Science and Research (IJSR)
ISSN: 2319-7064
ResearchGate Impact Factor (2018): 0.28 | SJIF (2019): 7.583

Hepatic Hydrothorax: A Concise Review of Literature

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Abstract: ***Hepatic hydrothorax (HH) is defined as an excessive transudative pleural effusion associated with portal hypertension without cardiac, pulmonary and pleural disease, with volume of more than 500 ml. The underlying mechanisms of HH are similar to those that cause fluid accumulation and ascites in portal hypertension. Several theories have been proposed as the mechanism of developing HH, including displacement of peritoneal fluid into the pleural cavity through diaphragmatic defects, hypoalbuminemia resulting in decreased colloid osmotic pressure, and lymphatic leakage from the thoracic duct. The management of HH cases is considered difficult, so that in most cases, the management of HH follows the ascites protocol in portal hypertension. There are several principles of therapy in cases of HH, which are reducing the production of ascitic fluid, preventing the movement of ascites through diaphragm, and drainage of the pleural cavity.***

Keywords: Hepatic Hydrothorax, Pleural Effusion, Ascites, Portal Hypertension

1. Introduction

Pleural effusion is a syndrome marked by accumulation of fluid in pleural cavity. Usually, the amount of pleural fluid is approximately 10 ml [1]. In the condition of pleural effusion, pleural fluid volume is >10 ml with the type of transudative or exudative pleural fluid. Until now, Light's criteria were used to differentiate the types of pleural fluid, and it is still the gold standard in considering differential diagnosis. Heart failure and cirrhosis of the liver are the most common causes of transudative pleural effusions. Pneumonia, tuberculosis, and malignancy are the most common causes of exudative pleural effusions [2].

Hepatic hydrothorax (HH) is defined as an excessive transudative pleural effusion associated with portal hypertension without cardiac, pulmonary and pleural disease [3]. The volume of pleural fluid obtained is usually more than 500 ml. In 79.5% -85% of cases there is a congestion on the right and left sides of around 13% -17.5%; whereas, only 2% -3% of patients have fluid on both sides of the pleural cavity [1], [3].

HH is not a common complication of end-stage liver disease and is rarely observed, depending on the diagnostic method, can be found in nearly 5%-10% of patients, who constitute 2%-3% of all causes of pleural effusion [1], [2], [4]. However, once HH has occurred, it can cause respiratory failure which worsens the clinical course of decompensated liver cirrhosis [1].

2. Pathophysiology

The underlying mechanisms of HH are similar to those that cause fluid accumulation and ascites in portal hypertension. Portal hypertension and splanchic vasodilation play an important role in ascites formation [5]. Concomitant portal hypertension and splachnic vasodilatation, together with activation of various neurohormonal signaling pathways, cause renal dysfunction and decrease excretion of sodium, water, and glomerular filtration rate [6]. Unidirectional flow of ascitic fluid into the pleural cavity is due to the pressure gradient created by negative intra-thoracic pressure during respiration and positive intra-abdominal pressure exacerbated by ascites.

Several theories have been proposed as to the mechanism of developing HH in patients with cirrhosis of the liver. These theories include displacement of peritoneal fluid into the pleural cavity through diaphragmatic defects, hypoalbuminemia resulting in decreased colloid osmotic pressure, and lymphatic leakage from the thoracic duct [3].

Table 1: Light's Criteria

Criteria	Exudate	Transudate
Ratio of Effusion protein/serum protein	>0.5	<0.5
Absolute level of Lactate dehydrogenase (LDH) in pleural fluid	>200 IU/L	<200 IU/L
Ratio of Effusion LDH/Serum LDH	>0/6	<0.6

Until now, the most widely accepted mechanism for causing HH is fluid displacement from the peritoneal to the pleural cavity via a diaphragmatic defect. According to Huang et al., thoracoscopic diaphragmatic defects can be divided into four types of morphology, which are: Type 1 - no obvious defects; Type 2 - blisters located on the diaphragm; Type 3 - rupture (fenestration) defect in the diaphragm; Type 4 - there are several gaps in the diaphragm [7].

A defect in thoracic wall maybe an entryway of air causing pneumothorax. But, even with diaphragmatic defects, pneumothorax rarely occurs after a laparoscopic procedure. This is because the pressure gradient between the peritoneal and pleural cavities changes in patients with ascites. The pressure in abdominal cavity increase due to ascites, alongside with the change in diaphragm, which is thinner due to malnutrition in cirrhosis patients, the diaphragmatic defect become larger. In this larger defect, the peritoneal herniation can occur into the pleural space. This herniation is known as a pleuroperitoneal bleb. This herniation is possible to rupture, forming a channel through which fluid can pass. In

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this patient, nearly all of the ascitic fluid rapidly crosses the diaphragm into the pleural cavity. When ascitic fluid formation exceeds lymphatic absorption and moves to the pleural cavity there will be a buildup of fluid that causes Hepatic Hydrothorax [3].

3. Diagnosis

3.1 Clinical Manifestation

The clinical manifestation is usually dominated by signs and symptoms of cirrhosis and portal hypertension, which are ascites, spider naevi, asterixis, hepatosplenomegaly, caput medusa, and hepatic encephalopathy. In 79.5%-85% of cases there is a congestion on the right and left sides of around 13% -17.5%; whereas, only 2% -3% of patients have fluid on both sides of the pleural cavity [1], [3].

Patients may be asymptomatic where pleural effusion is an accidental finding on chest imaging performed for other reasons or the patient may have symptoms such as shortness of breath (34-35%), cough (20-22%), nausea (11%), pleuritic chest pain (8%) hypoxemia or respiratory failure associated with large pleural effusions. This clinical mainfestation depends on various factors such as pleural fluid volume, rate of pleural fluid accumulation and the presence of associated cardiopulmonary disease [8], [9].

3.2 Further Evaluation

Hepatic Hydrothorax is confirmed in patients with portal hypertension and ascites who present with pleural effusions after exclusion of primary lung, cardiac or pleural disease. Effusion can be seen on chest x-rays or in other imaging studies such as chest or abdominal ultrasound and CT [10].

It is necessary to know the characteristics of pleural fluid in cases of HH, including polymorphonuclear cell count <250 cells / mm3, total protein <2.5 g/dl with a ratio of total pleural fluid protein/total serum protein <0.5, pleural fluid LDH ratio / Serum LDH <0.6, serum pleural albumin gradient (SPAG)> 1.1 g/dl, glucose values are similar to serum glucose values and pH 7.4-7.55 [1], [3], [4].

When we first suspected HH, the initial evaluation of the effusion should be pleural fluid analysis. This is to identify the type of the fluid and rule out other causes of the effusion such as infection, inflammation or malignancy[3]. In a study by Xiol et al., from 60 cirrhotic patients with pleural effusions, 42 patients (70%) were found to have pleural fluid analysis result corresponding with hydrothorax. The rest of the patients (30%) had a diagnosis other than HH such as spontaneous bacterial empyema in 9 patients (15%), tuberculosis in 2 patients, adenocarcinoma in 2 patients and parapneumonic empyema in 2 patients; 3 is an undiagnosed exudate [11].

Other tests that we need to consider in evaluating pleural fluid include pH, triglycerides, adenosine deaminase and PCR for tuberculosis, amylase and cytology. These tests need to be run to rule out empyema, chylothorax, tuberculosis, pancreatitis and malignancy, in patients suspected with other diagnoses. A chest CT scan, brain natriuretic peptide (BNP), and an echocardiogram to evaluate heart function are often indicated to evaluate other causes of pleural effusion [4], [12]. Scintigraphic studies can be done in case of unclear hydrothorax. Using intraperitoneal instillation radiolabeled particle, diagnosis of HH can be confirmed when there is communication between peritoneal and thoracic cavities [12].

4. Management

The management of HH cases is considered difficult, so that in most cases, the management of HH follows the ascites protocol in portal hypertension [13], [14]. There are several principles of therapy in cases of HH, especially refractory HH, which are reducing the production of ascitic fluid, preventing the movement of ascites through diaphragm, and drain the pleural cavity[3].

1) Reducing Ascitic Fluid Production [3]

a) Medication

The main principle of drug therapy is to reduce sodium levels by reducing intake and increasing excretion with diuretics [1], [3]. The recommended sodium level is <2,000 mg / day. The initial dose of Spironolactone is 50-200 mg/day or Amiloride 5-10 mg/day, and the Spironolactone dose is titrated 100 mg/week with a maximum dose limit of 400 mg/day.

If with Spironolactone monotherapy there is no improvement in clinical conditions or there is hyperkalemia, then the use of Spironolanctone should be combined with Furosemide with initial dose of 40 mg/day titrated 40 mg/week with a maximum dose limit of 160 mg/day. The target weight loss for patients without peripheral edema is 0.5 kg/day and 1 kg/day with peripheral edema and ascites [1].

b) *Transjugular Intrahepatic Portosystemic Shunt* (TIPS)

The TIPS procedure is a surgical method of connecting the portal vein and hepatic vein for portal vein decompression to reduce ascites production and pleural effusion [3], [14]. The TIPS procedure is preferred if the patient is expected to have a liver transplant schedule in less than 3 months and can be used as palliative therapy in the patient who are not undergoing liver transplants [8].

c) Liver Transplantation

Liver transplantation is the definitive therapy in HH patients, but because it cannot be available all the time, and takes a long time, so patients are recommended for other therapies [1], [3].

2) Preventing the Movement of Ascites Through Diaphragm

a) *Paracentesis*
   This procedure is a simple procedure prior to thoracentesis to prevent fluid accumulation in the pleural cavity due to decreased intrathoracic pressure [3].
b) *Surgery for Diaphragm Disorders*

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This procedure is recommended to decrease the flow of fluid from the peritoneal to the pleural cavity. This procedure can be performed using Video-Assisted Thoracoscopic Surgery (VATS) or using certain substances, such as pleurodesis, or a combination of the three [3].

3) Drainage of the Pleural Cavity

a) Thoracentesis

This procedure is repeated 2-3 times a week, if the patient with HH experiences shortness of breath. However, thoracentesis procedure should be preceded by paracentesis to avoid recurrence of HH after the procedure [3].

b) Indwelling Tunneled Pleural Catheter (ITPC)

This procedure was initially done considering a chest tube may result in secondary infection, pneumothorax or hemothorax. It was used for palliative therapy in managing patient with effusion, and now it is a widely used procedure in symptomatic malignant effusion [12]

Table 2: Comparison between Therapeutic Modalities for Hepatic Hydrothorax

Treatment	Advantage	Disadvantages
Drug Therapy	• Cheap • Non-invasive	• Lower compliance rate • Increase risk of AKI and kidney failure • Not effective on refractory HH
Thoracentesis	• Reducing symptoms • Can be used as sample for pleural fluid analysis	• Needs to be repeated • Causing complication, such as Pneumothorax, Hemothorax, Lung Oedema
ITPC	• Controlled evacuation	• Causing complication, such as empyema, loculation, dislodgement, leakage and pneumothorax.
TIPS	• "*Bridge*" for liver transplant • The success rate is up to 70-80%	• Post-TIPS Hepatic Encephalopathy • Thrombosis and embolion "Shunt" • Lower life expectancy rate on MELD >15, CTP Class C, andcreatinine level>2 mg/dL pre-TIPS
Pleurodesis	• Diaphragm repair is possible to be done • The success rate can be increased with CPAP, somatostatin • Considered in patient who is contra-inticated with TIPS	• Needs to be repeated • Using general anaesthesia for VATS • Causing complications such as, Empyema, Sepsis, Septic Shock • Increase bleeding risk with mechanical Pleurodesis. • Not possible to be done in "*Trapped Lung*"
Surgery for Diaphragm Disorder	• Increasing the success rate of Pleurodesis	• Not always visualized • Invasive
Liver Transplantation	• The most effective therapy compared to other therapies	• Not always available

5. Summary

Hepatic Hydrothorax is a rarely observed complication of end stage liver disease. Patient with HH may be asymptomatic, but may also present with symptoms associated with pleural effusion. The diagnosis of HH should be made after finding pleural effusion and excluding other conditions such asprimary lung, cardiac or pleural disease.

The management of HH cases is considered difficult, so that in most cases, the management of HH follows the ascites protocol in portal hypertension. The definitive management of Hepatic Hydorthorax is liver transplantation. But since it is not readily available in all health center, other modalities have been proposed, which are reducing the production of ascitic fluid, preventing the movement of ascites through diaphragm, and drain the pleural cavity.

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