

Hepatic Hydrothorax in A Patient with Liver Cirrhosis

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ABSTRACT

Hepatic hydrothorax is a transudative pleural effusion which presents in 5-10% patients with liver cirrhosis. Although fairly uncommon, it is associated with higher morbidity and lower survival rate. The mechanism is yet to be understood fully, but the most widely accepted pathogenesis involves the presence of portal hypertension, diaphragmatic defects, and negative intrathoracic pressure, all of which lead to the formation of unidirectional passage of ascitic fluid from peritoneal cavity into pleural space. Due to its origin, the pleural effusion has similar characteristics to ascitic fluid. We herein report the case of a 60-year-old woman with advanced liver cirrhosis and right-sided moderate hepatic hydrothorax. Treatment given to the patient includes diuretics, sodium restriction, and repeated thoracentesis. Subsequent evaluation of the patient revealed improvement both clinically and radiologically.

Keywords: Hepatic hydrothorax, Liver cirrhosis, Transudate, Pleural effusion

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INTRODUCTION

Hepatic hydrothorax is an infrequent complication of liver cirrhosis, which presents as transudative pleural effusion not secondary to cardiopulmonary disease or other causes. Although the earlier cases of pleural effusion in liver cirrhosis was described by Laennec, but not until 1958 the terminology of hepatic hydrothorax was first introduced, stressing the role of liver cirrhosis as the cause of transudative pleural effusion (Morrow et al., 1958). The prevalence of hepatic hydrothorax is estimated to be 5-10% in liver cirrhosis (Lv et al., 2018). Although quite uncommon, the presence of hepatic hydrothorax is associated with morbidity and mortality. Patients with hepatic hydrothorax have poorer prognosis, with median survival time 8-12 months (Hou et al., 2016).

The exact mechanism of hepatic hydrothorax has not been well understood, but it is thought to occur due to direct passage of ascitic fluid from peritoneal to pleural space via diaphragmatic defects (Silva Cruz et al., 2019). These defects are usually small (<1 cm) and mostly occur on the right side. Histologically, right diaphragm is more tendinous while left diaphragm is more muscular. Diaphragmatic defects are known to develop on these tendinous part; this might explain the right-side predominance of hepatic hydrothorax (Lv et al., 2018). Other factors contributing to the development of hepatic hydrothorax are negative intrathoracic pressure, positive intraabdominal pressure, and malnutrition (which causes thinning of diaphragm), creating a unidirectional passage of ascitic fluid and accumulation in pleural space (Lv et al., 2018; Chaaban et al., 2019).

It must be noted that while in the majority of cases it is associated with the presence of ascites, it may also occur without the evidence of ascites (Chaaban et al., 2019).

We report a case of a 60-year-old patient with advanced liver cirrhosis who was admitted due to variceal bleeding but was found to have right-sided hepatic hydrothorax on following workup. The patient was successfully treated with multiple thoracentesis, diuretics, and low sodium diet and was subsequently discharged from hospital.

CASE REPORT

A 60-year-old woman was admitted due to fresh blood vomiting several hours before hospital admission. She had been hospitalized 4 times in the last year due to recurrent episodes of haematemesis and melena. She had previously been diagnosed as having chronic hepatitis B and liver cirrhosis for 2 years and had undergone upper gastrointestinal endoscopy twice, with the last procedure taken on 3 months before admission and was diagnosed with grade 2 esophageal varices. Medication regularly taken including propranolol, furosemide, and spironolactone. On history taking, she also complaint of having persistent dyspnea for 2 months especially on recumbent position and therefore she had to use 2 pillows to be able to sleep. The shortness of breath was sometimes accompanied by intermittent cough. No chest pain or palpitation were noted. There was no history of diabetes mellitus, hypertension, asthma, nor any heart, lung, or kidney disease.

On admission the patient was alert. Her vital signs were stable, with blood pressure of 100/60 mmHg, heart rate of 76 beats per minute, respiratory rate of 22 breaths per minute, and axillary temperature of 36.6°C. Her thorax examination revealed decreased breath sounds and dull percussion on right side. No rhonchi or wheezing was found. There was no distention of jugular veins. Her abdomen was moderately distended with evidence of ascites. Bilateral pitting edema on lower extremities were noted. No active bleeding was observed on admission. Chest radiography showed moderate right-sided pleural effusion without any infiltrates or visible mass. Her laboratory results were as follows: hemoglobin 7.1 g/dL, WBC 9,680/ μ L, platelet 145,000/ μ L, AST 43 U/L, ALT 18 U/L, total bilirubin 2.65 mg/dL, random blood glucose 98 mg/dL, serum albumin 2.05 g/dL, BUN 16 mg/dL, serum creatinine 0.70 mg/dL, and normal urinalysis. Sputum smear and culture results were negative. Abdominal ultrasound on previous admissions showed shrunken liver with increased coarse echogenicity, blunted edge, and irregular border, with free extraluminal fluid, suggesting liver cirrhosis and ascites. Transient elastography was also done on previous admission with score of 39.7 kPa (F4), interpreted as severe fibrosis (cirrhosis). Both electrocardiography and echocardiography findings were normal.

Diagnosis of liver cirrhosis CTP C (hepatitis B related) with complications of variceal bleeding, right-sided pleural effusion, anemia, and hypoalbuminemia were made. Treatment administered including 1 bag packed red cell transfusion aiming for a hemoglobin level of 8 g/dL, 40 g albumin infusion, octreotide 25 mcg/hour for 5 days, omeprazole 40 mg 12-hourly, cefotaxime 1 g 8-hourly, lamivudine 100 mg/day, lactulose syrup 10 g 8-hourly, and low sodium diet. After 5 days, octreotide infusion was stopped and switched to propranolol 20 mg 8-hourly.

Pleural fluid analysis revealed transudates with clear pale yellow color, pH 8, WBC 119 cells/mL, PMN 37 cells/mL, MN 82 cells/mL, albumin 0.7 g/dL, protein 0.8 g/dL, LDH 68 U/L, glucose 118 mg/dL, with serum albumin 2.6 g/dL, serum protein 5.6 g/dL, and serum LDH 171 U/L which were taken at the same time as the pleural fluid sampling. Ascites fluid analysis also showed transudates with similar characteristics, with pH 8, WBC 62 cells/mL, PMN 22 cells/mL, MN 40 cells/mL, albumin 0.5 g/dL, protein 0.7 g/dL, LDH 51 IU/mL, glucose 125 mg/dL. Serum-pleural albumin gradient obtained from the calculations was 1.9 g/dL.

Based on the above results and after excluding other causes of transudative effusion, the patient was assessed as having hepatic hydrothorax. Furosemide 40 mg/day and spironolactone 100 mg/day were added to treatment. Thoracentesis were done 3 times with interval of 2-3 days, with a total of 1.5 L fluid being evacuated. The patient responded well to treatment and remain stable. After twelve

days of hospital care, there were both clinical and radiological improvements and the patient was subsequently discharged.

DISCUSSION

Hepatic hydrothorax is defined as the accumulation of transudative pleural effusion (typically more than 500 mL) in liver cirrhosis with portal hypertension which is not caused by cardiac, pulmonary, renal, or any other etiologies (Chaaban et al., 2019). It develops mostly in decompensated cirrhosis, with more than 90% occurs in CTP class B and C (Chaaban et al., 2019). As in our case, the patient presented with recurrent variceal bleeding and was diagnosed with decompensated cirrhosis with CTP score of 12 (CTP class C). While pleural effusion due to cardiac origin is usually bilateral, hepatic hydrothorax occurs mainly (80-85%) on the right side, although it may also occur on the left side in 13-17% and bilateral in 2-3% of cases (Krok & Cárdenas, 2012; Lv et al., 2018). This characteristic may help to differentiate between hepatic hydrothorax and pleural effusion due to cardiac disease. In our patient, pleural effusion was found to be solely on the right side. Common clinical manifestations include dyspnea and hypoxia related to the volume of pleural effusion. Most patients present with progressive dyspnea and may be accompanied by cough or pleuritic chest pain, while some may remain asymptomatic (Chaaban et al., 2019). Our patient also presented with progressive dyspnea and intermittent cough which developed within 2 months.

Diagnosis of hepatic hydrothorax can be made based on the presence of liver cirrhosis with portal hypertension and exclusion of pulmonary, cardiac, or any other diseases which may cause pleural effusion. Pleural fluid analysis should be performed to help with diagnosis and exclude other possible causes (e.g., infection, malignancy). In hepatic hydrothorax, pleural fluid has transudative quality with characteristics similar to that of ascitic fluid. Light criteria is widely used to differentiate between transudates and exudates. According to this criteria, an effusion is considered to be exudate if one or more of the following conditions are met: pleural fluid/serum protein ratio >0.5; pleural fluid/serum LDH ratio >0.6; or pleural fluid LDH level is greater than two-thirds of the serum LDH upper normal limit (Light, 2011). Calculating serum to pleural fluid albumin gradient is also a useful method; due to its origin, hepatic hydrothorax usually has albumin gradient ≥ 1.1 , similar to serum-ascitic fluid albumin gradient in ascites secondary to portal hypertension (Runyon et al., 1992). In our patient, both pleural and ascitic fluid did not fulfill any of the Light criteria. Calculated serum-pleural fluid and serum-ascitic fluid albumin gradient were 1.9 g/dL and 2.1 g/dL, respectively. These findings suggested transudative quality, with similar fluid components between pleural and ascitic fluid. Negative sputum stain and culture, no other abnormalities on chest radiography, normal electro-

cardiography and echocardiography, normal renal function and urinalysis, combined with data from physical signs and symptoms excluded other possible causes of transudative pleural effusion. Thoracoscopy, scintigraphy, or other imaging methods can be considered to confirm the presence of diaphragmatic defects and/or the passage of ascitic fluid into pleural space when the diagnosis is uncertain (Lv et al., 2018). We did not perform thoracoscopy in our patient due to its invasive nature and weak general condition. Scintigraphic study was also not done due to resources limitations.

Currently there is no specific guideline for the management of hepatic hydrothorax. Initial therapy involves diuretics and sodium restriction (2-4 g/day). Diuretics most commonly used are spironolactone (100 mg/day initially, can be titrated up to 400 mg/day) which can be combined with furosemide (40 mg/day initially, up to 160 mg/day) (Cárdenas et al., 2020). Repeated thoracentesis might be necessary to relieve respiratory symptoms in patients with large volume of effusion. Large volume paracentesis with albumin infusion is recommended in patients with hepatic hydrothorax with tense ascites (Garbuzenko and Arefyev, 2017). In our patient, we gave medical treatments (diuretics and low sodium diet) combined with repeated thoracentesis. For diuretics we used combination between spironolactone 100 mg/day and furosemide 40 mg/day which were both maintained until the patient was discharged. We considered repeated thoracentesis needed to be done in our patient due to moderate volume of pleural effusion and the presence of respiratory symptoms. We did not perform large volume paracentesis because the ascites was not large and tense.

Chest tube insertion to drain effusion in hepatic hydrothorax can lead to volume depletion and electrolyte imbalance and therefore should be avoided (Silva Cruz et al., 2019). Other interventions which could be considered particularly in refractory cases include indwelling pleural catheter, pleurodesis, transjugular intrahepatic portosystemic shunt (TIPS), peritoneovenous and pleurovenous shunting, and thoracoscopy to repair diaphragmatic defects (Garbuzenko and Arefyev, 2017; Lv et al., 2018). These procedures are invasive and may not be suitable for every case. In our case, the patient's condition improved after treatment with diuretics and repeated thoracentesis, therefore we deemed these procedures unnecessary. Moreover, our patient was admitted due to recurrent variceal bleeding and weak general condition, which we considered to be high risk to undergo such invasive procedures. Until now, liver transplantation is the only definitive treatment for hepatic hydrothorax, since most patients have advanced stage of liver cirrhosis (Lv et al., 2018). However, liver transplantation is still rarely done in our country due to resources limitations.

CONCLUSION

In conclusion, we report a case of hepatic hydrothorax in a patient with advanced liver cirrhosis which was successfully

managed using combination of medical management and repeated thoracentesis. Hepatic hydrothorax is relatively uncommon but may pose serious complications and higher morbidity in patients with liver cirrhosis, which underlines its importance to diagnose carefully and exclude other possible causes. Pleural fluid analysis should be routinely performed in every patient. The main treatment consists of diuretics, sodium restriction, and repeated thoracentesis if necessary. Chest tube placement should be avoided while other invasive procedures might be considered on a case-by-case basis.

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