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Case Report

Unveiling the Uncommon: Cirrhosis-Induced Pleural Effusion in the Absence of Ascites

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ABSTRACT

Hepatic hydrothorax is a well-recognized complication of advanced liver disease, typically occurring in patients with ascites. However, the presentation of hepatic hydrothorax in the absence of significant ascitic fluid is very uncommon and illustrates a unique diagnostic dilemma. This case report presents a 58-year-old female with a significant history of liver cirrhosis secondary to chronic Hepatitis B infection, who developed a massive right-sided pleural effusion lacking any concomitant ascites. The clinical presentation, diagnostic workup, and management of this case are discussed, highlighting the pathophysiological mechanisms underlying this unusual presentation. The case highlights the need for clinicians to maintain a high index of suspicion for hepatic hydrothorax in cirrhotic patients who exhibit respiratory symptoms, even in the absence of ascites, to ensure timely and appropriate management.

Keywords: Hepatic hydrothorax; Cirrhosis; Pleural effusion; Ascites

Introduction

Hepatic hydrothorax is defined as the presence of a pleural effusion, typically right-sided, in patients with portal hypertension and liver cirrhosis, occurring in the absence of primary cardiac, pulmonary, or pleural disease. It is reported in approximately 5-11% of patients with cirrhosis, most commonly in association with ascites[1]. The pathophysiology of hepatic hydrothorax is primarily attributed to the transfer of ascitic fluid through diaphragmatic defects into the pleural cavity, facilitated by negative intrathoracic pressure and increased abdominal pressure due to portal hypertension[2]. However, hepatic hydrothorax can rarely occur without significant ascitic fluid, complicating the diagnostic process[3].

In this report, we detail the case of a 58-year-old female with advanced liver cirrhosis secondary to chronic Hepatitis B infection, presenting with a large right-sided pleural effusion and near-total lung collapse without accompanying ascites. This uncommon presentation necessitates a comprehensive understanding of the alternative pathophysiological mechanisms contributing to pleural effusion in cirrhosis, such as increased

hydrostatic pressure in the pleural capillaries and decreased oncotic pressure from hypoalbuminemia[4]. Additionally, the case underscores the usefulness of utilizing diagnostic tools such as Light's criteria to differentiate the nature of the pleural effusion and guide subsequent management strategies.

Managing hydrothorax, especially when ascites is not present, requires an approach that includes thoracentesis for symptom relief, diuretics for addressing fluid overload, and potentially considering liver transplantation for patients with advanced liver disease[5]. This case underscores the importance of identification and awareness of this manifestation of hepatic hydrothorax to improve patient outcomes.

Case Presentation

A 58-year-old female with a known history of liver cirrhosis secondary to chronic Hepatitis B infection presented to the emergency department with acute onset dyspnea, right-sided pleuritic chest pain, and intermittent abdominal discomfort. Her medical history is significant for esophageal varices, hypertension, and hepatic portal vein thrombosis. She is status post inferior vena cava (IVC) filter placement in July 2023. The patient reported a recent travel to the Dominican Republic, where she experienced an episode of hematemesis necessitating hospitalization and multiple blood transfusions due to significant blood loss. Despite prior treatment, she has never been on antiviral therapy for Hepatitis B and has not previously consulted with a hepatologist.

On physical examination, the patient was found to be tachypneic (RR 34), icteric, and mildly hypoxic. Notably, breath sounds were markedly decreased on the right side of the thorax. The abdomen was soft, non-tender, and non-distended, with no evidence of ascites on examination. The patient's skin was significantly jaundiced, with multiple excoriations noted.

Laboratory investigations demonstrated significant hepatic dysfunction with elevated bilirubin, increased liver enzymes, prolonged coagulopathy, and decreased albumin. Renal function was compromised with a substantially increased serum creatinine level. Electrolyte imbalances included hyponatremia and hypokalemia (**Table 1**).

Table 1: Laboratory Investigations on Admission.

Parameter	Result	Reference Range
Sodium (Na)	135 mmol/L	135-145 mmol/L
Chloride (Cl)	100 mmol/L	96-106 mmol/L
BUN	21 mg/dL	7-20 mg/dL
Potassium (K)	3.1 mmol/L	3.5-5.0 mmol/L
Bicarbonate (HCO3)	22 mmol/L	22-29 mmol/L
Creatinine (Cr)	1.9 mg/dL	0.6-1.2 mg/dL
ALT	125 U/L	7-56 U/L
AST	97 U/L	5-40 U/L
Total Bilirubin	14.8 mg/dL	0.1-1.2 mg/dL
Albumin	1.2 g/dL	3.5-5.0 g/dL
PT	22 sec	11-13.5 sec
INR	2.3	0.8-1.1

Imaging studies included a chest X-ray, which revealed a large right-sided pleural effusion with associated collapse of the right lung and leftward mediastinal shift. A computed tomography angiography (CTA) of the chest confirmed these findings and additionally demonstrated liver morphology consistent with cirrhosis, with no evidence of pulmonary embolism (**Figure 1**).

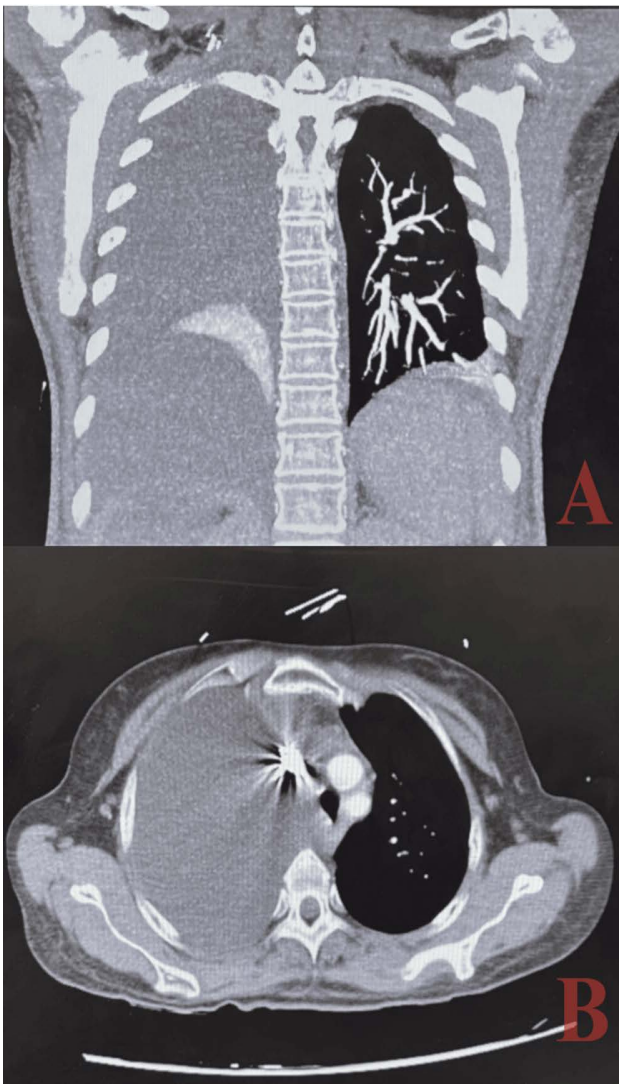

Figure 1: CT Angiography Imaging in Coronal and Axial Planes Demonstrating Right-Sided Pleural Effusion and Lung Collapse: The coronal (A) and axial (B) CT angiography images show a large right-sided pleural effusion leading to nearly complete collapse of the right lung.

The differential diagnosis for the pleural effusion included transudative causes such as hepatic hydrothorax, particularly given the absence of ascites, as well as other etiologies like congestive heart failure and pulmonary embolism.

Thoracentesis was performed, yielding approximately 1.5 liters of straw-colored fluid. Analysis of the pleural fluid was consistent with a transudative effusion based on Light's criteria. Light's criteria are used to classify pleural effusions into transudates or exudates, with transudative effusions typically having a pleural fluid protein to serum protein ratio <0.5, a pleural fluid LDH to serum LDH ratio <0.6, and a pleural fluid LDH level <2/3 the upper limit of normal serum LDH[6]. Based on these criteria, the ratios provided indicate that the pleural fluid is consistent with a transudate (**Table 2**).

A post-thoracentesis chest X-ray showed re-expansion of the right lung with stable bibasilar opacities and minimal blunting of the right costophrenic angle. Notably, there was a small right apical pneumothorax (**Figure 2**).

Table 2: Analysis of Pleural Fluid According to Light's Criteria.

Parameter	Pleural Fluid	Serum	Ratio	Reference Range (Transudate Criteria)
Protein (g/dL)	1.0	6.0	0.17	Pleural fluid protein/serum protein < 0.5
LDH (U/L)	150	600	0.25	Pleural fluid LDH/serum LDH < 0.6 or Pleural fluid LDH < 2/3 the upper limit of normal serum LDH (600 U/L)

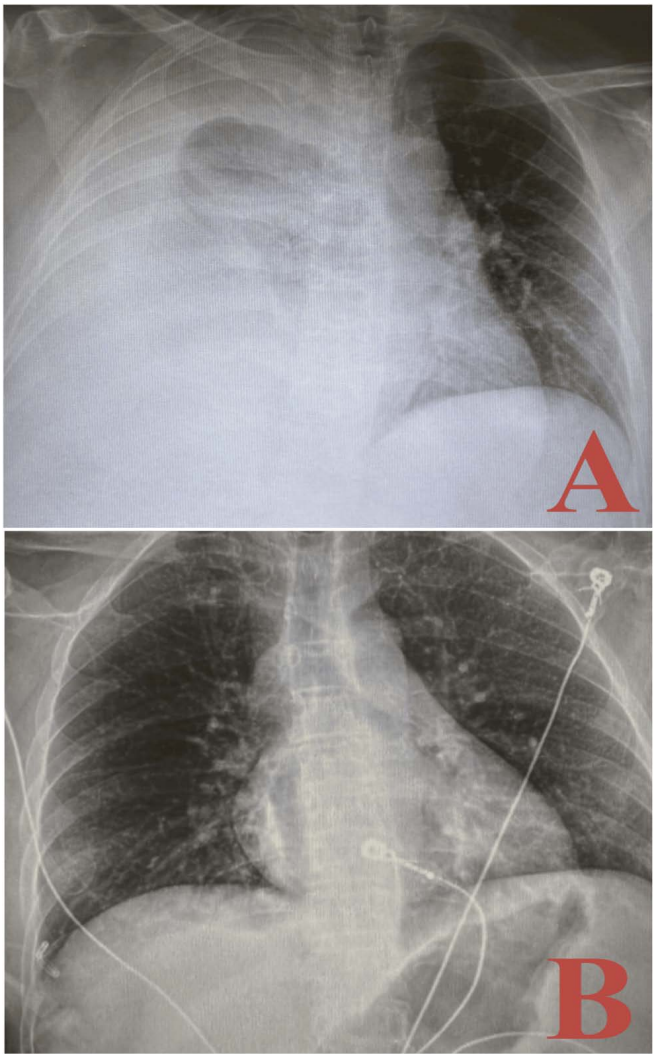

Figure 2: Chest X-Rays Pre- and Post-Thoracentesis Illustrating Resolution of Pleural Effusion: The pre-thoracentesis chest X-ray (A) shows a large right pleural effusion with associated near-complete collapse of the right lung and leftward mediastinal shift. The post-thoracentesis chest X-ray (B) demonstrates the re-expansion of the right lung, with resolution of the effusion and minimal residual blunting of the right costophrenic angle. A small right apical pneumothorax is also observed.

Discussion

This case of a large right-sided pleural effusion secondary to cirrhosis in the absence of significant ascites is particularly noteworthy due to its atypical presentation. Commonly, hepatic hydrothorax is associated with significant ascitic fluid; however, in this instance, the patient's presentation with a large pleural effusion without corresponding ascites necessitated a thorough diagnostic approach. The use of Light's criteria was instrumental in categorizing the effusion as transudative, consistent with hepatic hydrothorax rather than other etiologies such as exudative effusions, which might indicate malignancy or infection.

Pathophysiology and Alternative Mechanisms

The pathophysiology underlying hepatic hydrothorax in the absence of ascites remains complex and multifactorial. Several mechanisms potentially contribute to the formation of pleural effusion without significant ascitic fluid:

Increased Hydrostatic Pressure: Portal hypertension, a relatively common complication of cirrhosis, leads to elevated pressures within the splanchnic circulatory network. This elevated hydrostatic pressure can extend to the pleural capillaries, promoting the transudation of fluid into the pleural space[7]. Normally, this fluid accumulates in the peritoneal cavity as ascites, but in some cases, it may preferentially migrate into the pleural space through microscopic diaphragmatic defects or via direct transudation due to localized pressure gradients.

Decreased Oncotic Pressure: Hypoalbuminemia, frequently seen in patients with advanced liver disease, reduces plasma oncotic pressure, facilitating fluid movement from the intravascular compartment to the extravascular space. Albumin, a major plasma protein synthesized by the liver, plays a crucial role in maintaining colloid osmotic pressure. In cirrhosis, hypoalbuminemia results from impaired hepatic synthesis and increased capillary permeability, contributing to fluid accumulation in body cavities, including the pleural space[8].

Diaphragmatic Defects: Small, often microscopic, defects in the tendinous portion of the diaphragm can act as conduits for fluid passage from the peritoneal cavity to the pleural space. These defects may be congenital or acquired, possibly exacerbated by increased intra-abdominal pressure due to portal hypertension. The negative intrathoracic pressure generated during respiration further facilitates this fluid migration. This mechanism, although typically associated with the presence of ascites, can occur independently, leading to isolated pleural effusions. A similar presentation of a pleural effusion in the absence of abdominal ascites was noted in a case, in which, two patients who presented with right-sided pleural effusions and no abdominal ascites. Both patients had diaphragmatic defects: One was an old traumatic diaphragmatic tear and the other a pinpoint spontaneous perforation[9]. Another case describing a difficult diagnostic and therapeutic management of a massive pleural effusion on the right side in the absence of any relevant ascites mentions the likely direct movement of fluid from the peritoneal cavity into the pleural space through diaphragmatic defects[10].

Lymphatic Obstruction: Impaired lymphatic drainage due to cirrhosis and portal hypertension can contribute to pleural effusion formation. The thoracic duct, responsible for draining lymph from the abdomen into the venous system, may be compromised by elevated pressures, leading to lymphatic overflow and subsequent transudation of fluid into the pleural cavity[11]. This lymphatic dysfunction is compounded by the systemic effects of cirrhosis, including hypoalbuminemia and altered vascular permeability.

Clinical Implications and Diagnostic Approach

The unusual presentation of hepatic hydrothorax without

ascites poses a diagnostic challenge. This case underscores the importance of maintaining a high index of suspicion in cirrhotic patients presenting with respiratory symptoms. Comprehensive diagnostic evaluation is essential and should include:

Imaging Studies: Chest radiography and computed tomography (CT) scans are pivotal in identifying pleural effusions and assessing the extent of lung involvement. In this case, imaging revealed a significant right-sided pleural effusion with near-total lung collapse and mediastinal shift, underscoring the severity of the presentation. CT angiography additionally confirmed liver morphology consistent with cirrhosis and excluded pulmonary embolism.

Pleural Fluid Analysis: Thoracentesis and subsequent analysis of pleural fluid are critical in differentiating transudative from exudative effusions. Light's criteria, based on pleural fluid protein and lactate dehydrogenase (LDH) levels relative to serum values, classify the effusion as transudative in this case. This classification is consistent with hepatic hydrothorax and helps exclude other etiologies such as malignancy, infection, or inflammatory conditions.

Laboratory Investigations: Evaluating liver function tests, coagulation profiles, and renal function is vital in understanding the overall impact of cirrhosis and guiding therapeutic interventions. In this case, significant hepatic dysfunction, including hyperbilirubinemia, hypoalbuminemia, elevated liver enzymes, and coagulopathy, was evident. Additionally, renal impairment and electrolyte imbalances were noted, reflecting the systemic effects of advanced liver disease.

Management Strategies

Managing hepatic hydrothorax, especially in the absence of ascites, requires a multifaceted approach:

Thoracentesis: This procedure serves as a critical intervention to alleviate symptoms and improve respiratory function[12]. In this patient, successful drainage of pleural fluid and subsequent lung re-expansion underscored the effectiveness of thoracentesis. Regular monitoring and repeat thoracentesis may be necessary to manage recurrent effusions. Complications such as pneumothorax, as observed in this case, highlight the need for careful technique and post-procedural monitoring.

Diuretics: Diuretics, including spironolactone and furosemide, can help manage fluid overload and reduce the recurrence of effusions. Their use must be balanced against the risk of renal impairment, which is often present in cirrhotic patients. Careful titration and monitoring of renal function and electrolytes are essential to optimize diuretic therapy. Additionally, sodium restriction may enhance the efficacy of diuretics in controlling fluid balance.

Transjugular Intrahepatic Portosystemic Shunt (TIPS): For patients with refractory hepatic hydrothorax, TIPS can reduce portal hypertension and decrease fluid transudation into the pleural space. This procedure involves creating a shunt between the portal and systemic venous circulation, thereby alleviating portal pressure. TIPS has shown efficacy in managing recurrent pleural effusions, although it carries risks such as hepatic encephalopathy and requires careful patient selection[13].

Liver Transplantation: In cases of advanced liver disease, liver transplantation remains the definitive treatment. This option should be considered in patients with severe hepatic dysfunction and refractory hydrothorax, as it addresses the underlying cause and improves long-term outcomes. Pre-transplant evaluation and optimization of the patient's clinical status are crucial to enhance post-transplant prognosis.

Conclusion

This case underscores the clinical significance of recognizing atypical presentations of pleural effusion in patients with cirrhosis. It emphasizes the necessity for thorough diagnostic evaluations, including advanced imaging and pleural fluid analysis using Light's criteria, to understand the complex interplay of factors contributing to pleural effusion in the absence of ascites. The successful resolution of the effusion through thoracentesis illustrates the effectiveness of appropriate intervention. However, it also calls for ongoing research into the pathophysiology of such presentations and potential preventive strategies to mitigate the risk of recurrence or complications. An enhanced understanding of these mechanisms could lead to improved patient outcomes and guide the development of targeted therapeutic approaches in cirrhosis-associated pleural effusions.

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