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Silent Swells: Unraveling Hepatic Hydrothorax without Ascites in a case of Chronic Liver Disease

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ABSTRACT

Hepatic hydrothorax, commonly associated with chronic liver disease (CLD), typically presents alongside ascites due to portal hypertension. However, the occurrence of hepatic hydrothorax without ascites is an unusual and diagnostically challenging manifestation. This case report describes a 58-year-old male with a history of alcohol use disorder who presented with dyspnoea and pleural effusion in the absence of ascites. Diagnostic imaging and laboratory findings confirmed hepatic hydrothorax without any abdominal fluid accumulation, complicating the typical clinical picture of decompensated liver disease. Management included therapeutic thoracentesis and diuretic therapy, with successful resolution of symptoms. This case underscores the importance of considering hepatic hydrothorax in patients with CLD, even in the absence of ascites, and highlights the need for a comprehensive diagnostic and therapeutic approach to improve patient outcomes.

Keywords: Hepatic Hydrothorax, Chronic Liver Disease, Portal hypertension, Ascites

INTRODUCTION

Hepatic hydrothorax is a common complication associated with chronic liver disease (CLD), generally manifesting alongside ascites as a result of portal hypertension and fluid dynamics within the body [1]. This condition typically involves the translocation of fluid into the pleural space, leading to respiratory symptoms and requiring appropriate

management strategies [2]. However, the occurrence of hepatic hydrothorax without the presence of ascites presents a perplexing clinical scenario that can challenge standard diagnostic and therapeutic approaches [3,4]. In such cases, the absence of ascites may obscure the underlying hepatic pathology and complicate the clinician's ability to recognise the underlying etiology of pleural effusion. This case report highlights an exceptional instance of hepatic hydrothorax in a patient with CLD devoid of ascites, aiming to enhance our understanding of this rare presentation and underscore the importance of comprehensive evaluation in patients with advanced liver disease. Through this exploration, we hope to contribute valuable insights into the complexities of hepatic hydrothorax and its management in clinical practice.

CASE PRESENTATION

Patient Information

A 58-year-old male presented to the emergency department with complaints of progressively worsening dyspnea, a nonproductive cough, and intermittent chest discomfort over the past month. He had no history of fever, night sweat, and weight loss. He has no history of palpitation, chest pain, facial puffiness, and leg swelling. He had no history of recent travel or any contact to patients with tuberculosis. He reported no significant abdominal swelling or any gastrointestinal symptoms. His urine output was normal. The patient had an earlier medical history of alcohol use disorder of 120ml of whiskey daily for almost 27 years. He has no past comorbidities.

Clinical Examination

Upon initial assessment, he was conscious, oriented, afebrile, with a pulse rate of 92 beats per minute, blood pressure of 132/72mmHg and oxygen saturation of 95% in ambient air. The notable findings included a mild tachypnoea (Respiratory rate of 20cycles/minute) and visible use of accessory muscles for breathing. He has no pallor, clubbing, edema, or any significant lymphadenopathy. A head-to-toe examination revealed no significant skin or nail lesions. Upon inspection, the right hemithorax displayed reduced expansion compared to the left, indicating possible fluid accumulation. Percussion revealed dullness over the right lung field, in contrast to normal resonance on the left. Auscultation revealed diminished breath sounds on the right mammary, axillary, infra-axillary, and infra scapular areas, with no adventitious sounds noted. Tactile fremitus was significantly reduced when assessed on the right. The patient presented with mild central cyanosis, and upon palpation, no tenderness was noted in the right thorax. There was mild splenomegaly obscuring the Traube’s space. All other systemic examinations were normal. Overall, these findings were highly suggestive of a right pleural effusion.

Diagnostic Assessment

The initial chest radiograph showed right plaque right pleural effusion **(Figure 1)** which warranted a further detailed work up. A 12-Lead-electrocardiogram was normal sinus rhythm, and the 2-Dimensional echocardiography was within normal.

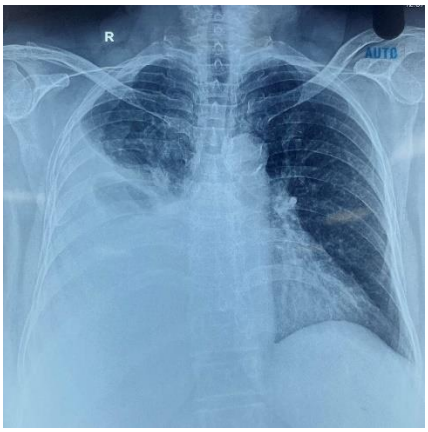

Figure 1: The chest radiograph revealed an increased density in the right hemithorax, blunted at the costophrenic angle, elevated right hemidiaphragm and left mediastinal shift. The lung fields are largely obscured, indicating limited visibility of the underlying lung structures. Overall, the findings suggest a right pleural effusion

A complete hemogram showed normocytic and normochromic anemia with hemoglobin of 11.2g% (normal range 13-17g%), Total count of 4.2 $x10^3$µL (normal range: 4 $x10^3$µL – 10 $x10^3$µL) and Platelet count of 163 $x10^3$µL (150 $x10^3$µL- 410 $x10^3$µL). Biochemical testing showed no gross abnormalities, except for a mild hepatitis pattern liver dysfunction characterised by an albumin level of 3.0 g/dL (normal range: 3.5-5.0 g/dL), aspartate aminotransferase (AST) at 62 IU/L (normal range: 10-40 IU/L), and γ-glutamyl transpeptidase (GGT) at 129 IU/L (normal range: 9-48 IU/L). Additionally, the bilirubin level was measured at 22 µmol/L (normal range: 5-20 µmol/L), with an international normalized ratio (INR) of 1. These findings suggest liver dysfunction and warrant further evaluation to determine the underlying cause.

Following a diagnostic thoracentesis, a straw-coloured pleural fluid sample was collected for evaluation. The laboratory analysis revealed a total protein concentration of 3.0 g/dL (normal range: <3.0 g/dL), placing it at the upper threshold for transudative effusions, while the serum total protein was recorded at 6.5 g/dL (normal range: 6.0-8.0 g/dL). The lactate dehydrogenase (LDH) level in the pleural fluid measured 120 IU/L (normal range: <200 IU/L), which is within normal limits, compared to a serum LDH level of 180 IU/L (normal range: 100-200 IU/L). The cell counts of the pleural fluid showed slightly elevated white blood cells with a predominance of lymphocytes, and cultures did not show any signs of infection. Additionally, biochemical tests revealed low glucose levels (normal range: 60-100 mg/dL) alongside elevated pleural fluid protein, pointing to a potentially atypical transudative effusion.

A basic liver screen, which included tests for hepatitis A, B, and C viruses, as well as autoimmune markers such as antinuclear antibody, anti-smooth muscle antibody, anti-mitochondrial antibody, and anti-liver–kidney microsomal antibody, along with assessment of immunoglobulin levels, yielded unremarkable results. Tumour markers like alphafeto protein, CA-19-9 and CEA were within normal limit. Urinalysis showed no proteinuria, and the urinary albumin and creatinine ration was within normal limit.

The liver ultrasound revealed characteristics consistent with cirrhosis. A follow-up contrast-enhanced CT scan of the chest, abdomen, and pelvis confirmed the presence of a simple pleural effusion but there were no signs of ascites **(Figure 2 and Figure 3).** An upper Gastrointestinal Endoscopy was done and revealed grade I esophageal varies without any hemorrhagic signs.

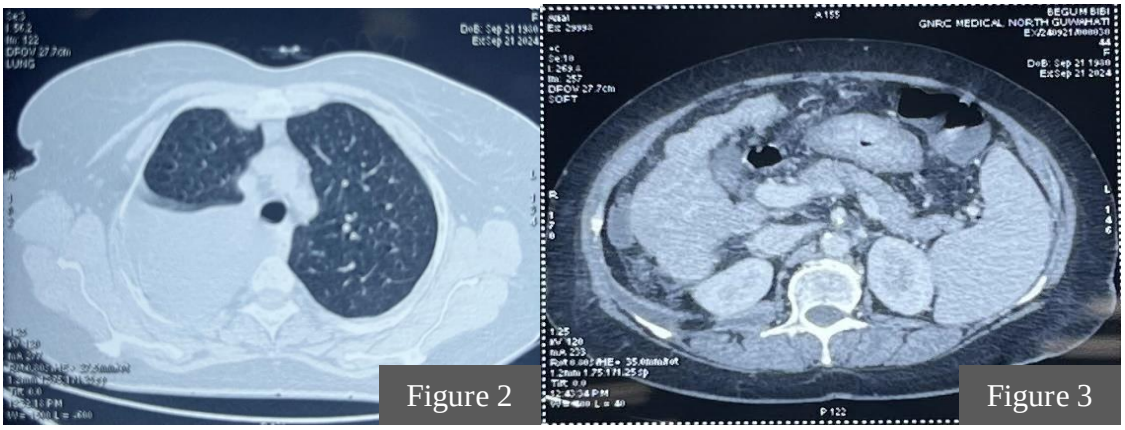

Figure 2 and 3: Chest CT shows large right sided pleural effusion with collapse of right lower lung without any significant parenchymal changes and CT abdomen shows shrunken liver with nodular margin, splenomegaly and dilated portal vein indicating chronic hepatic parenchymal disease with features of portal hypertension, however there is no ascites

Treatment Course

An Intercostal Drainage tube (ICD) was inserted under aseptic precaution. A total of 5 litres of straw yellow colour fluid was drained (Figure 4). Diuretics (Furosemide and spironolactone) and beta blocker was added and uptitrared to a daily dose of Furosemide 40mg, Spironolactone 200mg and Carvedilol 6.25mg. After 5 days of imitation of treatment the ICD output decreased below 50ml/day and gradually decreased on subsequent days. ICD was removed. He was discharged with the above medications after 2 days of strict monitoring.

Figure 4: Straw yellow coloured ICD output

Outcome and Follow-Up

After 4 weeks of initiation of treatment, he was completely asymptomatic. Repeat chest radiograph did not show any abnormality. It was noteworthy that, there was no physical or radiological evidence of ascites on the follow up visits after 8th and 12th week.

DISCUSSION

Hepatic hydrothorax is a clinical condition characterized by the accumulation of pleural fluid in CLD and is often a manifestation of advanced cirrhosis [5]. The management of hepatic hydrothorax involves a dual approach: addressing the underlying decompensated liver disease and managing the specific complications associated with hydrothorax itself [1,5].

The management of decompensated CLD is outlined in numerous international guidelines [6-8]. Key to this approach is the involvement of gastroenterologists or hepatologists who can provide tailored interventions based on specific disease characteristics. This may include antiviral therapy for viral hepatitis, management of alcohol use disorder, or pharmacological treatments targeting liver function improvement. Furthermore, assessment for liver transplantation is critical for eligible patients, as definitive management in end-stage liver disease may require transplantation to provide long-term survival [9].

Regular monitoring of liver function, ascitic fluid analysis, and screening for hepatocellular carcinoma are essential aspects of comprehensive care. Nutritional support is also critical, as patients with decompensated CLD often experience malnutrition [6]. Principles of a low-sodium, high-protein diet, along with dietary counseling, are important to improve overall patient outcomes [10].

Specific Management of Hydrothorax

The management of hepatic hydrothorax parallels that of ascites due to similar underlying mechanisms, specifically the influence of portal hypertension and fluid dynamics. The initial approach typically involves lifestyle modifications, including dietary sodium restriction (sodium intake of 70–90 mmol/day) and implementing a diuretic regimen [3,6].

Aldosterone antagonists (such as spironolactone) and loop diuretics (such as furosemide) can either be used alone or in combination. The rational approach involves starting with a single diuretic agent and gradually uptitrating the dose based on the patient's clinical response, similar to the management strategy for ascites [11]. In cases where hydrothorax becomes symptomatic, therapeutic thoracentesis is often performed to provide immediate relief from dyspnea and discomfort. This procedure allows for the removal of fluid and can provide symptomatic relief but may be temporary, as reaccumulation of fluid is common due to persistent portal hypertension and hepatic dysfunction [12]. For patients experiencing recurrent pleural effusion, more definitive interventions may be necessary. One potential option is formal chest drainage with pleurodesis. This procedure aims to obliterate the pleural space to prevent future reaccumulation of fluid. However, the effectiveness of pleurodesis in patients with hepatic hydrothorax can be limited, as rapid fluid reaccumulation can occur due to high portal pressures [13].

Research has suggested that adjunctive treatments, such as the use of tetracycline during pleurodesis in concert with continuous airway pressure, may enhance the effectiveness of the procedure by lowering the peritoneal gradient pressure [14]. However, the evidence surrounding this approach is sparse, with limited published data apart from anecdotal reports, necessitating further investigation into its validity. Another emerging strategy includes the deployment of indwelling pleural catheters, which allow for intermittent domiciliary drainage of pleural fluid based on symptoms [15]. This approach has gained traction, especially in the management of malignant pleural effusions, and could theoretically be beneficial for patients with hepatic hydrothorax, particularly those who are not candidates for invasive hepatic procedures [14,15]. Nonetheless, caution must be exercised, as the use of these catheters carries the risk of precipitating hepatorenal syndrome, an acute deterioration in kidney function in patients with advanced liver disease [16].

There are limited number of case studies on patient presenting with hepatic hydrothorax as an initial presentation of CLD, without ascites [17,18]. Hepatic hydrothorax typically occurs in patients with advanced liver disease, primarily due to complications arising from portal hypertension. However, it can manifest in the absence of overt ascites for several reasons:

1. Presence of Pathologic Fluid Dynamics: In some cases, changes in venous pressure and fluid dynamics can lead to pleural effusion without a significant accumulation of ascites. Conditions such as increased negative intrathoracic pressure can cause fluid to shift into the pleural space [19].
2. Transdiaphragmatic Fluid Movement: Fluid from the peritoneal cavity can migrate through small defects in the diaphragm (due to the high pressure in the abdomen) and accumulate in the pleural space. This can happen even if ascites is not pronounced [20].
3. Increased Hydrostatic Pressure: Portal hypertension can cause increased hydrostatic pressure in the systemic circulation, leading to fluid leakage across vascular membranes into the pleural cavity, even if ascites is not present [21].
4. Lymphatic Dysfunction: The lymphatic system may not effectively drain fluid in patients with liver disease, which can lead to fluid accumulation in softer tissues or cavitary spaces like the pleura, independent of ascitic fluid [22].

As a result of these factors, patients can develop hepatic hydrothorax without significant ascites, indicating a complex interplay between the liver's function and pleural fluid dynamics which requires further research.

CONCLUSION

In conclusion, this case report underscores that hepatic hydrothorax can occur independently of ascites, highlighting its potential as an early manifestation of CLD. This finding emphasises the importance of considering pleural effusions in patients with liver dysfunction, even in the absence of typical signs of ascites. Clinicians should remain vigilant in assessing and diagnosing hepatic hydrothorax, as prompt recognition can lead to timely management of

underlying liver conditions, ultimately improving patient outcomes. This case serves as a valuable reminder of the multifaceted presentations of hepatic complications, advocating for a comprehensive approach in clinical evaluation.

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