CASE REPORT

Hepatic Hydrothorax in the Background of Minimal or No Ascites: A Case Report

Ketan Agarwal[1], Vishnu Shankar Ojha[2], Ratnadeep Biswas[3], Divendu Bhushan[4]

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Abstract

Aim and background: Chronic liver disease (CLD) is a progressive deterioration of liver function that usually remains asymptomatic for long periods of time. An uncommon complication of long-standing portal hypertension with end-stage liver disease is hepatic hydrothorax.

Case description: Here, we present an unusual case of an asymptomatic CLD patient presenting with sudden onset dyspnea. The chest X-ray showed pleural effusion in the right middle and lower zones. Biochemical analysis of the pleural fluid confirmed the transudative nature of the fluid. Minimal ascites were noted, which could not be tapped. A diagnosis of hepatic hydrothorax was made, and the patient showed marked improvement following the initiation of therapy with diuretics and β-blockers.

Conclusion: Negative intrathoracic pressure in the pleural cavity during inspiration, coupled with the presence of small fenestrations in the diaphragm and the transdiaphragmatic lymphatics, favors the flow of fluid across these fenestrations and into the pleural cavity leading to hepatic hydrothorax.

Clinical significance: The absence of ascites should not be used as a criterion to preclude a diagnosis of hepatic hydrothorax. Timely interventions, including both medical and surgical modalities, should be initiated for its management, but the patient may ultimately require liver transplantation.

Keywords: Ascites, Case report, Exudates and transudates, Hepatic hydrothorax, Pleural effusion, Portal hypertension.

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Introduction

Chronic liver disease (CLD) is a progressive deterioration of liver function that usually remains asymptomatic for long periods of time. They typically start with gradual, nonspecific symptoms such as weight loss, fatigue, or anorexia, but sometimes an acute complication can occur. One such complication can be decompensated liver disease with portal hypertension, where there is transudative accumulation of fluid, commonly in the form of ascites.[1] Furthermore, an uncommon complication of long-standing portal hypertension with end-stage liver disease is hepatic hydrothorax.[2] Transudative pleural effusions, typically greater than 500 mL, in individuals with portal hypertension without any other underlying renal or cardiopulmonary etiology are referred to as hepatic hydrothorax.[3] In >90% of cases, hepatic hydrothorax is associated with ascites.[4] Here we present an unusual clinical picture of a long-standing asymptomatic CLD patient presenting with sudden-onset dyspnea later diagnosed as hepatic hydrothorax without any evidence of ascites.

Case Description

History and Examination

A 56-year-old man presented to our emergency department with shortness of breath and heaviness in the right side of his chest for 4 days. He came with an intercostal drainage (ICD) tube inserted *in situ* 1 day back from another medical center. The bag contained serous fluid. On examination, his higher mental functions were normal; he was tachypneic with an oxygen saturation of 97% and nasal prongs at 2 L/minute. Pallor, icterus, clubbing, cyanosis, lymphadenopathy, and edema were absent. Examination of the respiratory system revealed reduced expansion and air entry to the right side of the chest. The examination of the other systems was unremarkable.

[1–4]Department of General Medicine, All India Institute of Medical Sciences, Patna, Bihar, India

Corresponding Author: Vishnu Shankar Ojha, Department of General Medicine, All India Institute of Medical Sciences, Patna, Bihar, India, Phone: +91 9931176590, e-mail: vsojha12@gmail.com

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Investigations

The examination findings were corroborated by the chest X-ray, which showed the presence of pleural effusion in the right middle and lower zones (Fig. 1). Direct microscopy of the pleural fluid showed 150 cells/mL^3 with a neutrophilic predominance of 80%. The culture of the pleural fluid grew *Pseudomonas aeruginosa*. On biochemical analysis, glucose levels in the pleural fluid were 77.0 mg/dL, the pleural protein/serum protein ratio was <0.5, and the pleural lactate dehydrogenase (LDH)/serum LDH ratio was <0.6, suggesting the transudative nature of the fluid according to the modified light's criteria.[5] The biochemical analysis results have been summarized in Table 1.

Ultrasonography (USG) of the abdomen showed a normal-sized liver with coarse echotexture a dilated portal vein (14 mm) and splenomegaly (13.8 cm), which were suggestive of CLD with portal hypertension (Fig. 2). Minimal ascites was noted, which could not

be tapped. Grade III esophageal varices were visualized on upper gastrointestinal endoscopy, and endoscopic variceal ligation was performed.

Differentials

An evaluation of the cause of CLD was done. There was no history of alcohol intake or diabetes mellitus. The hepatitis B surface antigen and the hepatitis C core antigen were negative. The iron profile was not suggestive of hemochromatosis. Around 24-hour urinary copper and ceruloplasmin levels were within normal limits. Antinuclear antibody (ANA) screening with the human epithelial-2 (HEp-2) cell line (ANA-HEp-2) was positive. Antibody markers of autoimmune liver disease (antimitochondrial M2, antiliver kidney microsome type 1, anti-liver cytosol 1, anti-SP100, anti-glycoprotein-210, and antibodies to soluble liver antigen) were negative. The echocardiography and kidney function tests were unremarkable as well.

Treatment and Follow-up

A diagnosis of hepatic hydrothorax was established, the ICD was removed, and the patient was started on diuretics and β-blockers. There was a marked symptomatic improvement following the initiation of therapy, and a chest X-ray on serial follow-up showed significant improvement in effusion.

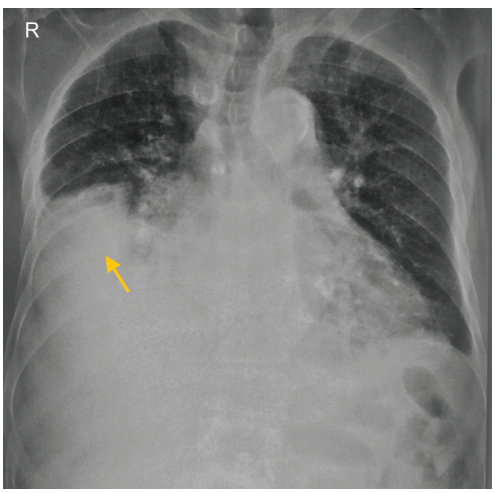


Fig. 1: Chest radiograph showing right-sided pleural effusion in posteroanterior view. The arrow points to the effusion present in the right middle and lower zones

Discussion

Third spacing refers to the shifting of body fluid to the interstitial space, which makes it unavailable for circulation.[6] The underlying etiologies behind such shifts are broadly classified as either transudative or exudative. Exudative fluid is often due to inflammatory conditions like pneumonia, tuberculosis, cancer, etc., which lead to the leaking of fluid and leukocytes into the interstitium. Whereas transudative effusions are primarily caused by imbalances between the starling forces.[7] Many conditions like heart failure, chronic kidney disease, etc., are known to cause transudative effusions, and one such cause is CLD.[8]

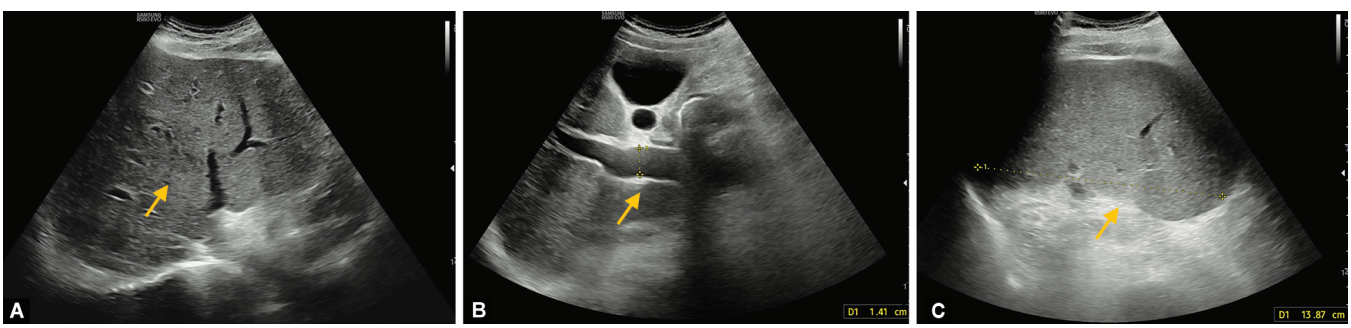
The CLD is defined as a progressive decline of liver functions lasting for <6 months. It involves continuous damage to the liver parenchyma that eventually leads to fibrosis and cirrhosis. It affects all categories of liver functions, like the biosynthesis of plasma proteins and clotting factors, detoxification, metabolic functions, bile production, etc.[1] Thus, CLD can present with numerous complications, such as edema, ascites, hydrothorax, and many more.

Hepatic hydrothorax is defined as a collection of 500 mL or more of transudative pleural fluid in patients with portal hypertension without any other cardiac etiologies.[3,9] In our patient, the biochemical analysis confirmed the transudative nature of the fluid, which, along with the deranged liver function tests and USG findings consistent with portal hypertension, confirmed the diagnosis of hepatic hydrothorax.

The pathogenesis of the third spacing of fluid in CLD is generally attributed to the decreased levels of serum albumin, which in turn lowers the plasma oncotic pressure. This mechanism is generally attributed to the cause of pleural effusion, jaundice, edema, or pericardial effusion. But unlike the cases reported by Kalambokis et al. and von Bierbrauer et al., who found concurrent ascites along with hepatic hydrothorax we observed an isolated case of hepatic

Table 1: Results of the biochemical analysis of serum and pleural fluid

Serial number	*Parameter*	*Result*	*Reference range*
1	Serum LDH (U/L)	186.98	230–460
2	Serum total protein (gm/dL)	5.50	6.4–8.3
3	Pleural LDH (U/L)	17.45	–
4	Pleural adenosine deaminase (U/L)	0.72	0–24
5	Pleural protein (gm/dL)	0.09	1–2
6	Pleural glucose (mg/dL)	77.0	–



Figs 2A to C: USG images of the abdomen. (A) The arrow in panel A points toward the coarse echotexture of the liver; (B) The arrow in panel B points toward a dilated portal vein, suggesting portal hypertension; (C) The arrow in panel C points toward splenomegaly

hydrothorax without the presence of ascites.[10,11] Similar cases of isolated hepatic hydrothorax have been reported previously by Sukcharoen et al., Doraiswamy et al., etc.[12,13]

The pathogenesis that has been hypothesized to explain this unusual finding is that the negative intrathoracic pressure in the pleural cavity during inspiration, coupled with the presence of small fenestrations in the diaphragm and the transdiaphragmatic lymphatics, favors the flow of fluid across these fenestrations and into the pleural cavity,[14] that is, when the negative intrathoracic pressure exceeds the abdominal pressure, hepatic hydrothorax ensues. Conversely, when the intra-abdominal pressure is greater than the transthoracic pressure, there is an accumulation of free fluid in the peritoneal cavity.[15] The flow of radiolabeled human albumin through these diaphragmatic defects has been demonstrated by Lv et al. and this is currently the most accepted theory explaining hepatic hydrothorax.[16] This mechanism is further supported by the finding that the hydrothorax was present on the right side in our case, as the right side of the diaphragm is thought to be less muscular (and thus naturally weaker) than the left.[17]

It was interesting to note that, despite the culture of the pleural fluid showing growth of *Pseudomonas aeruginosa*, the fluid was still transudative in nature. The presence of the ICD in the patient before arriving at our hospital could be the cause of this infection, or it's also likely that the *Pseudomonas* infection was acquired in the hospital itself.

Cirrhosis and portal hypertension have been implicated in hepatic hydrothorax, primarily due to alcoholic liver disease. But in our case, no apparent causes could be identified for the relatively rapid development of portal hypertension, as the previous liver function tests were unremarkable, there was no history of any drug or alcohol intake, and the viral and autoimmune markers for the liver disease were negative.

Multiple treatment modalities have been suggested for the management of hepatic hydrothorax. The initial management strategy is generally medical, which involves therapy with diuretics and a low-salt diet. Chest tubes, indwelling pleural catheters, pigtail catheters, video-assisted thoracoscopic surgery, and transjugular intrahepatic portosystemic shunts have all been employed for further management of hepatic hydrothorax, but liver transplantation remains the definitive treatment.[14] Our patient was treated with diuretics and β-blockers and showed marked improvement.

Conclusion

In conclusion, this case highlights a rare and clinically significant manifestation of CLD. The presented case underscores the importance of early detection and diagnosis, especially in patients with advanced liver disease who may not present with ascites. By bringing attention to this atypical presentation of hepatic hydrothorax, clinicians can broaden their differential diagnosis, and ensure the timely initiation of appropriate management. Such rare and atypical presentations warrant continued investigation to elucidate underlying mechanisms and refine therapeutic approaches.

Clinical Significance

In cases of unilateral transudative pleural effusions, hepatic hydrothorax is one of the important differentials. Negative intrathoracic pressures and small diaphragmatic defects are thought to cause the shift of fluid into the pleural cavity, and right-sided effusions are more common owing to the weaker right diaphragm. The absence of ascites should not be used as a criterion to preclude a diagnosis of hepatic hydrothorax. Timely interventions, including both medical and surgical modalities, should be initiated for its management, but the patient may ultimately require liver transplantation.

Orcid

Ketan Agarwal https://orcid.org/0000-0002-5889-6052
Vishnu Shankar *Ojha* https://orcid.org/0000-0002-9524-9073
Ratnadeep Biswas https://orcid.org/0000-0001-7136-4459
Divendu Bhushan https://orcid.org/0000-0002-8718-5890

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