

# Isolated hepatic hydrothorax: an unusual complication in liver cirrhosis

Peter S,<sup>1</sup> Dhanoa J,<sup>1</sup> Peter P<sup>2</sup>

<sup>1</sup>Department of Internal Medicine, Christian Medical College & Hospital, Ludhiana, Punjab, India

<sup>2</sup>Department of Radiodiagnosis, Christian Medical College & Hospital, Ludhiana, Punjab, India

## ABSTRACT

Ascites and hydrothorax are common complications of liver cirrhosis, however, unilateral isolated hepatic hydrothorax without associated ascites is an unusual and challenging complication of liver cirrhosis. Though rare, this condition often poses both diagnostic and therapeutic dilemma, especially in patients without other symptoms or risk factors for cirrhosis. It usually leads to prolonged hospitalization and may cause early mortality. Unilateral isolated hepatic hydrothorax may also be the only pointer to underlying cirrhosis in the absence of ascites. Liver transplantation remains the only definitive treatment for this condition.

**Keywords:** Liver cirrhosis, hepatic hydrothorax, liver transplantation.

## INTRODUCTION

Hepatic hydrothorax is a manifestation of decompensated chronic liver disease, similar to ascites, hepatic encephalopathy and variceal hemorrhage. It is defined as pleural effusion more than 500 ml in the background of cirrhosis without underlying cardiopulmonary disease, and is usually seen on the right side.<sup>[1]</sup> Hepatic hydrothorax is an uncommon complication of end stage liver disease, occurring in about 5 to 10 % of cirrhotics.<sup>[1,2]</sup> Occurrence of pleural effusion in the absence of ascites or in association with minimal ascites is usually associated with conditions like congestive cardiac failure, nephrotic syndrome, pulmonary adenocarcinoma, infections like tuberculosis or parapneumonic effusion.<sup>[3]</sup> The isolated respiratory symptoms may confuse the physician leading to extensive diagnostic evaluation, as liver cirrhosis is not commonly considered a cause of unilateral pleural effusion.

We discuss a lady with this unusual complication who presented with recurrent, isolated, massive right sided pleural effusion as a complication of non-alcoholic fatty liver disease with cirrhosis, and with no evidence of ascites. Although such presentations are rare,<sup>[4-6]</sup> this case report highlights the importance of considering an uncommon differential of liver cirrhosis as the underlying cause of hydrothorax, even in the absence of ascites.

## Case report

A 57 year old lady presented to our outpatient department (OPD) with complaints of progressively increasing dyspnoea for 2 weeks along with orthopnoea and pedal oedema, with no associated fever, angina or wheezing. She had been a diabetic and hypertensive for the last 20 years, well controlled on medications. She did not have history of any cardiac disease, jaundice, altered sensorium, oliguria, or bleeding from any site. There was no history of blood transfusions, drug addictions or high risk behaviour.

On examination, vitals were stable. General physical examination revealed mild pallor and bilateral pedal oedema. There was reduced intensity of breath sounds on the right side. Abdominal examination showed moderate

### \*Corresponding address:

Dr. Soumia Peter  
Assistant Professor, Department of Internal Medicine  
Christian Medical College & Hospital, Ludhiana, Punjab, India  
Phone: 8054900362  
Email: soumiaanna@rediffmail.com

DOI: 10.5530/ogh.2013.1.15

splenomegaly with absence of palpable liver, ascites or dilated veins. Rest of the systemic examination were unremarkable. A clinical diagnosis of decompensated liver cirrhosis with complications of portal hypertension and right sided pleural effusion was made.

Laboratory investigations showed haemoglobin of 8.2 gm/dl, with coagulopathy (PT of 18 sec with control of 12, PTTK of 30 sec with control of 22), and hypoalbuminemia (2.5 g/dl). The total leucocyte count, random blood sugar level and liver enzymes were normal. Chest roentgenogram showed massive right pleural effusion [Figure 1]. Ultrasonogram showed coarse echotextured nodular liver, suggestive of cirrhosis, with splenomegaly and dilated portal vein, along with significant right sided pleural effusion with no evidence of free fluid in the abdomen. The renal function tests and iron studies were normal. Her viral markers, ceruloplasmin levels, autoimmune panel and work up for primary biliary cirrhosis were negative. Ophthalmic examination was negative for Keyser-Fleischer ring. Liver biopsy showed cirrhosis with moderate activity and marked steatosis. The modified Hepatic Activity Index (HAI) grade calculated was 9/18 and modified HAI stage was 5/6. On the basis of this evidence, she was diagnosed to have non-alcoholic fatty liver disease with liver cirrhosis.

She had dyslipidemia with low high density lipoprotein (25 mg/dl) and elevated total cholesterol (300 mg/dl) and low density lipoprotein (220 mg/dl). Echocardiogram showed left ventricular hypertrophy with normal ejection fraction. An upper Gastrointestinal endoscopy showed 3 large varices which were banded.

The lady had prolonged hospitalisation for almost two months during which she underwent multiple therapeutic

pleurocentesis for the rapidly filling right sided pleural effusion. Pleural fluid was transudative and was negative for mycobacterial Polymerase chain reaction (PCR) and for malignant cytology. She was also initiated on diuretics and salt restricted diet. Despite these measures, her respiratory status deteriorated and so she was considered for Transjugular intrahepatic porto-systemic shunt (TIPS) followed by liver transplantation. However, before she could be taken up for the procedure, she went into cardio respiratory arrest and could not be revived.

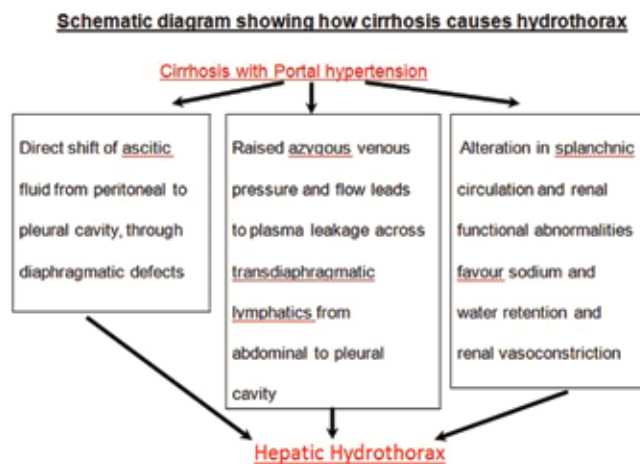
## DISCUSSION

Although pleural effusion with coexisting ascites is a common feature in cirrhotics, isolated pleural effusion can occur in a small proportion of patients, causing diagnostic as well as therapeutic dilemma. Hepatic hydrothorax can lead to prolonged hospitalization and mortality.<sup>[7]</sup>

Among the various mechanisms proposed for the pathophysiology of hepatic hydrothorax, the most likely one is the direct shift of ascitic fluid from the peritoneal to the pleural cavity, through diaphragmatic defects, due to the close anatomical relationship of bare areas of the liver with the diaphragm.<sup>[7]</sup> The hydrothorax is usually right sided (85%), with isolated left sided presentation in 13% and bilateral hydrothorax seen in only 2%.<sup>[8]</sup> The increase in azygous venous pressure and flow may lead to the leakage of plasma fluid traversing from the abdominal cavity to pleural cavity across transdiaphragmatic lymphatics. The additional factors for causing pleural effusion is the alteration in splanchnic circulation and the renal functional abnormalities favouring sodium and water retention and renal vasoconstriction.<sup>[9-10]</sup> [ Figure 2]



**Figure 1.** Chest roentgenogram shows massive right sided pleural effusion causing “white out” of the right hemithorax. The left hemithorax appears clear. There is mild mediastinal shift towards the left side.



**Figure 2.** Schematic diagram showing how cirrhosis causes hydrothorax.

Hepatic hydrothorax should be suspected in any advanced cirrhotic presenting with progressively worsening dyspnoea and cough with unilateral pleural effusion, usually on the right side, and minimal (less than 50 ml of ascitic fluid) or absent ascites. Alternatively, it may also be the only pointer to underlying cirrhosis in the absence of ascites. The patient may have complications of tension hydrothorax manifested by respiratory failure and hypotension or spontaneous infection of the pleural fluid, called spontaneous bacterial empyema.<sup>[11]</sup> A diagnostic thoracentesis should be performed in all patients with suspected hepatic hydrothorax after confirming pleural effusion on a chest roentgenogram. Pleural effusions associated with cirrhosis and portal hypertension are transudative.<sup>[12-13]</sup> Other conditions causing exudative pleural effusion like pyogenic infections, malignancy, tuberculosis, chylothorax and pancreatitis can be ruled out.

Computed tomography of the chest should be done to rule out any mediastinal, pulmonary or pleural lesion.<sup>[5]</sup> Abdominal sonogram helps to confirm the cirrhosis with accompanying pleural effusion and absent or minimal ascites, as well as allowing for measurement of portal and hepatic vein diameters and exclusion of pelvic masses. Injection of Technetium-99m sulphur colloid into the peritoneal cavity may demonstrate the flow of tracer into the pleural cavity, despite the absence of significant ascites.<sup>[14]</sup> A liver biopsy may be performed to confirm cirrhosis and to find out the cause of cirrhosis. Pericarditis or right heart failure can be ruled out by echocardiography.<sup>[5]</sup>

The patient should be started on diuretics (Frusemide [40 mg/day] and Spironolactone [100 mg/day]). These doses may be increased upto 160 mg/day and 400 mg/day respectively depending upon the clinical response. Sodium is restricted to 70 to 90 millimoles per day.<sup>[10,15]</sup>

Persistence of pleural effusion in spite of the above measures is called refractory hydrothorax. The definitive management for this condition is liver transplantation but many patients are not stable enough to undergo this procedure. As a bridge to it, patients may be subjected to repeated therapeutic thoracentesis, transjugular intrahepatic portosystemic shunt (TIPS) or video assisted thoracoscopy for closure of diaphragmatic defects (VATS) with or without pleurodesis.<sup>[2,16]</sup>

TIPS, a non surgical option for refractory hydrothorax, decompresses the portal system by artificially creating a portocaval shunt. This reduces portal pressure, thus decreasing the amount of pleural effusion. TIPS has 20% to 90% response rate in treatment of refractory

hepatic hydrothorax. Its complications include hepatic encephalopathy, deterioration in liver function and shunt occlusion.<sup>[17-19]</sup>

Other surgical options available are pleurodesis or sealing the space between the parietal and visceral pleura with a sclerosing agent, and VATS, for the repair of diaphragmatic defects. VATS, in the treatment of refractory hepatic hydrothorax has 60% to 100% response. Complications of VATS include permanent leakage and pneumonia.<sup>[20]</sup>

## CONCLUSION

Refractory isolated hepatic hydrothorax without ascites is a rare and serious complication of cirrhosis causing diagnostic difficulty unless the treating physician is aware of this unusual entity. Unilateral hepatic hydrothorax may also serve as the only pointer to underlying cirrhosis in the absence of ascites. Liver transplantation remains the only definitive therapy.

## ACKNOWLEDGMENTS

Nil

## REFERENCES

1. Cárdenas A, Arroyo V. Management of ascites and hepatic hydrothorax. *Best Pract Res Clin Gastroenterol.* 2007; 21:55-75.
2. Sidappa PK, Kar P. Hepatic hydrothorax. *Trop Gastroenterol.* 2009; 30:135-41.
3. Xiol X, Castellote J, Cortes-Beut R, Delgado M, Guardiola J, Sese S. Usefulness and complications of thoracentesis in cirrhotic patients. *Am J Med.* 2001; 111:67-9.
4. Huang PM, Chang YL, Yang CY, Lee YC. The morphology of diaphragmatic defects in hepatic hydrothorax: thorascopic finding. *J Thorac Cardiovasc Surg.* 2005; 130:141-5.
5. Roussos A, Philippou N, Mantzaris GJ, Gourgouliannis KI. Hepatic hydrothorax: pathophysiology diagnosis and management. *J Gastroenterol Hepatol.* 2007; 22:1388-93.
6. Bitar Z, Fayed H, Al-Hamer EE, Hafiz H. Hepatic thorax without ascites. *Int Med J.* 2008; 7:55-8.
7. Martin Rossle, Alexander L Gerbes. TIPS for the treatment of refractory ascites, hepatorenal syndrome and hepatic hydrothorax : a critical update. *Gut.* 2010; 59:988-1000.
8. Lazaridis KN, Frank JW, Krowka MJ, Kamath PS. Hepatic Hydrothorax: Pathogenesis, Diagnosis and Management. *Am J Med.* 1999; 107:262-7.
9. Cardenas A, Arroyo V. Mechanisms of water and sodium retention in cirrhosis and the pathogenesis of ascites. *Best Pract Res Clin Endocrinol Metab.* 2003; 17:607-22.
10. Gines P, Cardenas A, Arroyo V, Rodes J. Management of cirrhosis and ascites. *N Engl J Med.* 2004; 350:1646-54.
11. Xiol X, Castellvi JM, Guardiola J, Sese E, Castellote J, Perello A et al. Spontaneous bacterial empyema in cirrhotic patients: a prospective study. *Hepatology.* 1996; 23:719-23.
12. Cardenas A, Kelleher T, Chopra S. Review article: hepatic hydrothorax. *Aliment Pharmacol Ther.* 2004; 20:271-9.
13. Gur C, Ilan Y, Shibolet O. Hepatic hydrothorax-pathophysiology, diagnosis and treatment-review of the literature. *Liver Int.* 2004; 24:281-4.
14. Bhattacharya A, Mittal BR, Biswas T, Dhiman RK, Singh B, Jindal SK et al. Radioisotope scintigraphy in the diagnosis of hepatic hydrothorax. *J Gastroenterol Hepatol.* 2001; 16:317-21.

15. Runyon B. Management of adult patients with ascites due to cirrhosis. *Hepatology*. 2004; 39:841–56.
16. Buchholz S, Kaplan V, Hauser M. Treatment of a right-sided pleural effusion in a patient with liver cirrhosis. *Chest*. 2000; 117:248–50.
17. Gordon FD, Anastopoulos HT, Crenshaw W, Gilchrist B, McEniff N, Falchuk KR. et al. The successful treatment of symptomatic refractory hepatic hydrothorax with transjugular intrahepatic portosystemic shunt. *Hepatology*. 1997; 25:1366.
18. Strauss RM, Martin LG, Kaufman SL, Boyer TD. Transjugularintrahepatic portal systemic shunt for the management of symptomatic cirrhotic hydrothorax. *Am J Gastroenterol*. 1994; 89:1520–22.
19. Chalasani N, Clark WS, Martin LG, Kamean J, Khan MA, Patel NH et al. Determinants of mortality in patients with advanced cirrhosis after transjugular intrahepatic portosystemic shunting. *Gastroenterology* 2000; 118:138.
20. Mouroux J, Perrin C, Venissac N, Blaire B, Richelme H. Management of pleural effusion of cirrhotic origin. *Chest*. 1996; 109:1093–6.