

# Hepatic Hydrothorax in the Patient with Non-Ascitic Liver Cirrhosis: Case Report

## Asitsiz Karaciğer Sirozlu Hastada Hepatik Hidrotoraks

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Geliş Tarihi/Received: 08.04.2010  
Kabul Tarihi/Accepted: 04.05.2011

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**ABSTRACT** Hepatic hydrothorax is the fluid accumulation of at least 500 mL in the pleural cavity of a liver cirrhosis patient without the presence of lung or heart disease. Hepatic hydrothorax is a non-frequent complication of portal hypertension, but occurs in 5-12% of all cases of liver cirrhosis patients, and is generally encountered in ascitic cases. Hepatic hydrothorax development is rare in non-ascitic liver cirrhosis in patients who use diuretics. We report a case of a 48-year-old female with non-ascitic liver cirrhosis that to the emergency department complaining of dyspnea, and whom later developed hepatic hydrothorax.

**Key Words:** Liver cirrhosis; pleural effusion; pleurodesis

**ÖZET** Hepatik hidrotoraks, akciğer veya kalp hastalığı bulunmaksızın karaciğer sirozu olan bir hastada plevral boşlukta en az 500 mL sıvı toplanması olarak tanımlanmaktadır. Portal hipertansiyonun sık görülmeyen bir komplikasyonu olan hepatic hidrotoraks, olguların %5-12'sinde ortaya çıkmaktadır. Hepatik hidrotoraks genellikle asitli olgularda karşımıza çıkar. Diüretik kullanan asitsiz karaciğer sirozunda hepatic hidrotoraks gelişimi nadir olarak görülür. Biz, nefes darlığı şikâyeti ile acil servisimize başvuran asitsiz karaciğer sirozlu 48 yaşında hepatic hidrotoraks gelişmiş kadın olguyu sunmak istedik.

**Anahtar Kelimeler:** Karaciğer sirozu; plevral effüzyon; plörodezis

**Türkiye Klinikleri J Gastroenterohepatol 2011;18(2):84-7**

**H**epatic hydrothorax is the fluid accumulation of at least 500 mL in the pleural cavity of a liver cirrhosis patient without the presence of lung or heart disease. Hepatic hydrothorax is a non-frequent complication of portal hypertension, but occurs in 5-12% of all cases of liver cirrhosis patients, and is generally encountered in ascitic cases. However, there are studies in which hepatic hydrothorax development is reported without ascites. In this case report, we discuss the diuretic-using patient who developed hepatic hydrothorax from non-ascitic liver cirrhosis. We also discuss the literature about the issue.

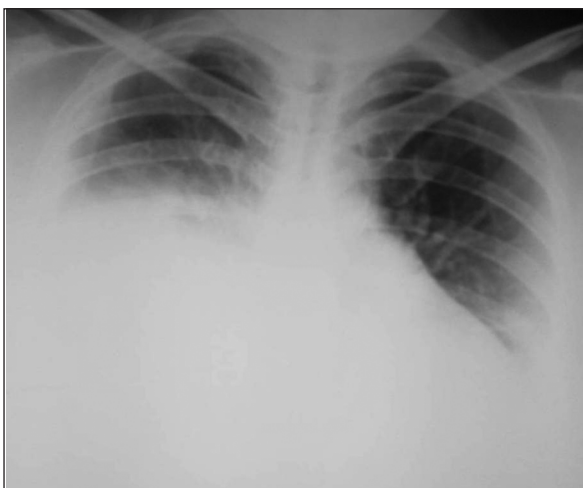
### CASE REPORT

In the patient's history, she used oral anti-diabetics for 12 years for diabetes mellitus, and used insulin for 5 years. The patient had an operation 10 years ago for a multi-nodular goiter; however, she did not use any medica-

tion for this condition. The patient's diagnosis for liver cirrhosis occurred 2.5 years ago. Liver cirrhosis was diagnosed with histopathologic examination of liver biopsy due to abdominal ascites. The patient has not any alcohol her history. Hepatitis markers were negative. The patient was prescribed diuretics for this condition (Furosemide 1 x 40 mg). A coronary angiography was performed 1 year ago with normal results.

The 48-year-old female patient presented at the emergency department complaining of dyspnea, but not of cough, fever, sputum, or palpitations. The patient was tachypneic on physical examination. Oscultation found decreased breaking in the right hemithorax. Posteroanterior chest X-ray (Figure 1) and computerized thorax tomography (Figure 2) revealed right pleural effusion. A complete abdominal ultrasonography revealed no ascites in the abdomen. The findings were compatible with chronic liver disease. Ejection fraction was 60% in the transthoracic echocardiography. We did not detect cardiological pathology, a possible explanation for the pleural effusion.

Routine laboratory tests showed the following: hemoglobin 9.19 g/dL, white blood cell 6.74 K/mm<sup>3</sup> albumin 3.5 g/dL, and LDH 218 IU/L. Tests of liver and renal function, and the thyroid-stimulating hormone level were normal. Within the pleural effusion, albumin was 2.1 g/dL, and LDH was 170 IU/L. Cytology of the pleural effusion and a comp-



**FIGURE 1:** Posteroanterior chest X-ray: Pleural effusion in right hemithorax.



**FIGURE 2:** Computerized thorax tomography: Pleural effusion in right hemithorax.

lete urine examination showed no abnormalities. There was no reproduction of pleural fluid in the urine culture. Since we did not detect any cardiac or pulmonary conditions causing the pleural effusion, we diagnosed the case as hepatic hydrothorax.

Pleural effusion was exudative according to Light criteria (0.6). However, we checked the serum-pleural effusion albumin gradient (1.4). It was possible that this was a false exudative result due to prolonged use of diuretics; however, the exudative was evaluated to be compatible with transudate. We drained the fluid by means of pleurocan catheter. We then used rifampicin for and terminated the pleurocan catheter.

## DISCUSSION

The development of pleural effusion without ascites with liver cirrhosis is a rare clinical situation. Hepatic Hydrothorax is an uncommon manifestation of portal hypertension occurring in approximately 5 to 12% of patients with cirrhosis of the liver.<sup>1,2</sup> The diagnosis of hepatic hydrothorax is usually suspected in a patient with advanced cirrhosis presenting with a unilateral pleural effusion, most commonly in the right side.<sup>3,4</sup> Patient usually present with symptoms related to shortness of bre-

ath, cough, hypoxemia and chest discomfort. Ascites is not always present.<sup>5-7</sup> In addition, pleural effusion is generally encountered on the right side in hepatic hydrothorax, where it was found in our case. Left pleural effusion (16%) and bilateral pleural effusion (16%) are rare.<sup>8,9</sup> Our patient had dyspnea and hypoxemia. There was no fever other signs of infection.

There are many proposed mechanisms for the occurrence of hepatic hydrothorax, including the following: direct passage of peritoneal fluid into the pleural space via diaphragmatic defects, azygous vein hypertension with leakage of plasma, passage of fluid from the peritoneal to the pleural cavities via transdiaphragmatic lymphatics, hypoalbuminemia and decreased colloid osmotic pressure, and thoracic duct lymphatic leakage.<sup>5,6</sup> But of these mechanisms, the direct passage of peritoneal fluid via diaphragmatic defects appears to explain most but not all cases of hepatic hydrothorax. Hepatic hydrothorax occurs when the accumulation of ascites in the pleural cavity surpasses the absorptive capacity of the pleura. The negative intrathoracic pressure favours the transfer of fluid across these defects and patients usually have minimal or mild ascite.<sup>10,11</sup> This mechanism has been corroborated with nuclear medicine studies using <sup>99m</sup>Tc-human albumin or <sup>99m</sup>Tc sulphur colloid and dye studies that show a unidirectional passage of these markers from the abdominal to the pleural cavity in the first 24 h administration.<sup>3,6</sup> But we did not use this nuclear medicine technique. The patients pleural effusion cytological and microbiological examination of the biochemical analysis of the malignancy, tuberculosis and other infection wasn't detected. 2.5 years before liver biopsy of patients were diagnosed with cirrhosis liver. It was hepatic hydrothorax evaluated because of the etiology of pleural effusion not any cardiac and liver problem.

Upon the occurrence of false exudative results arising from the increase in protein content in the fluid, we discussed the use of serum-pleural effusion/ascite gradients method in patients with pleural effusion and ascites who use diuretics. It was evident that the albumin level difference between

the serum-pleural effusion and/or ascite fluids are superior to the Light criteria for the differentiation of exudate and transudate.<sup>12,13</sup> Furthermore, we found the effusion to be exudative according to the Light criteria; however, later it became evident that the effusion qualified as transudate according to the serum-pleural effusion albumin gradient method due to prolonged use of diuretics. This condition prevented further invasive diagnostic procedures for the patient.

Talc, tetracycline, bleomycin are common used agents against to recurrent pleural effusions. But non of them is ideal. Features of ideal agents are cheap, effective, easy found and minimal side effects. Because of it explorations go on to find alternative agent. Kinakrin and iodopovidone were showed as alternative agents. But these are not in routinely practice.<sup>14</sup> Talc, rifampicin and autolog blood were compared in a study which aimed pleurodesis effectivity of these agents. Pleurodesis effect of rifampin was similar with talc. Yılmaz et al used rifampicin, talc, and autolog blood in 56 patients with hydrocell. They affirmed talc as the most effective agent in pleurodesis.<sup>15</sup> We got successful response from our patient who had poor renal functions pleurodesis with rifampin as alternative agent.

In the medical treatment of hepatic hydrothorax, several methods can be included: thorasynthesis, transjugular intrahepatic portosystemic shunt, videothoracoscopic diaphragmatic defect repair, and tube thoracostomy. However, tube thoracostomy is undesirable due to serious fluid-electrolyte loss.<sup>15</sup> Thorasynthesis is the first method of preference for the improvement of pulmonary symptoms in cases with hepatic hydrothorax. However, the risk of pneumothorax increases during drainage by thorasynthesis.<sup>16,17</sup> Furthermore, we believe that subsequent thorasyntheses also further increase the risk of empyema. Therefore, our preferred method for was the insertion of the pleurocan catheter.

## CONCLUSION

Our case provided evidence that where the pleural effusion is not associated with cardiac and pulmonary conditions in diuretic-using patients with li-

ver cirrhosis, the preferred course of action for the differentiation of exudative and transudative characteristics of pleural effusion is the use of the serum-pleural effusion gradient method, instead of the Light criteria. This course of action will also protect the patient from. Hepatic hydrothorax should be remembered in hepatic cirrhotic patient with pleural effusion and without cardiopulmonary

pathology. Differential diagnosis of effusion should be done with microbiologic, biochemical and cytopathologic tests. Pleurocan as a less invasive operation can used in treatment. We affirmed rifampin as pleurodthesis agent in these selected patients.

### **Acknowledgment**

*Thank you to Nova Tercüme who English translation of the article.*

## REFERENCES

1. Lazaridis KN, Frank JW, Krowka MJ, Kamath PS. Hepatic hydrothorax: pathogenesis, diagnosis, and management. *Am J Med* 1999; 107(3):262-7.
2. Garcia N Jr, Mihas AA. Hepatic hydrothorax: pathophysiology, diagnosis, and management. *J Clin Gastroenterol* 2004;38(1):52-8.
3. Cárdenas A, Arroyo V. Management of ascites and hepatic hydrothorax. *Best Pract Res Clin Gastroenterol* 2007;21(1):55-75.
4. Çolakoğlu ÖS. [Complications of cirrhosis]. *Türkiye Klinikleri J Int Med Sci* 2007;3(16):34-43.
5. Strauss RM, Boyer TD. Hepatic Hydrothorax. *Semin Liver Dis* 1997;17(3):227-32.
6. Cardenas A, Kelleher T, Chopra S. Review article: hepatic hydrothorax. *Aliment Pharmacol Ther* 2004;20(3):271-9.
7. John S, Paul MP, Murthy UK. An unusual presentation of cirrhotic pleural effusion in a patient with no ascites: a case report. *Cases J* 2009;2:6767.
8. Wojcikiewicz TG, Gupta S. Primary biliary cirrhosis presenting with ascites and a hepatic hydrothorax: a case report. *J Med Case Reports* 2009;3:7371.
9. Kinasevitz GT. Transudative Effusions. *Eur respir J* 1997;10(3):714-8.
10. Kirsch CM, Chui DW, Yenokida GG, Jensen WA, Bascom PB. Hepatic hydrothorax in the absence of ascites: report of two cases and review of the mechanism. *Am J Med Sci* 1991;302(2):103-6.
11. Menteş BB, Kayhan B, Görgül A, Unal S. Hepatic hydrothorax in the absence of ascites: report of two cases and review of the mechanism. *Dig Dis Sci* 1997;42(4):781-8.
12. Rocco VK, Ware AJ. Cirrhotic acites. *Ann Intern Med* 1986;105(4):573-85.
13. Rector WG Jr, Reynolds TB. Superiority of the serum-ascites albumin difference over the ascites total protein concentration in separation of "transudative" and "exudative" ascites. *Am J Med* 1984;77(1):83-5.
14. Dikensoy O, Light RW. Alternative widely available, inexpensive agents for pleurodesis. *Curr Opin Pulm Med* 2005;11(4):340-4.
15. Yılmaz U, Ekmekçiöğlü O, Tatlışen A, Demirci D. Does pleurodesis for pleural effusions give bright ideas about the agents for hydrocele sclerotherapy? *Int Urol Nephrol* 2000;32(1): 89-92.
16. Alberts WM, Salem AJ, Solomon DA, Boyce G. Hepatic hydrothorax. Cause and management. *Arch Intern Med* 1991;151(12):2383-8.
17. Milanez de Campos JR, Filho LO, de Campos Werebe E, Sette H Jr, Fernandez A, Filomeno LT, et al. Thoracoscopy and talc poudrage in the management of hepatic hydrothorax. *Chest* 2000;118(1):13-7.