Case Report

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Hepatic Hydrothorax without Apparent Ascites and Dyspnea - A Case Report

Internal Medicine Section

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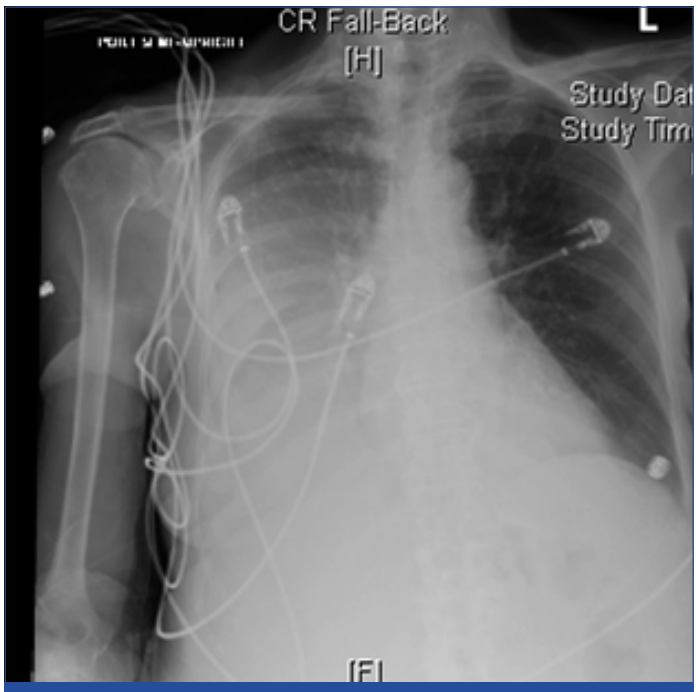
ABSTRACT

A 78-year-old female with a past medical history of alcoholic cirrhosis was hospitalised with recurrent lower gastrointestinal bleeding due to rectal ulcers. The ulcers were successfully treated with cautery and placement of clips. However, a recurrent large right-sided pleural effusion without apparent ascites and dyspnea were found incidentally during the hospitalisation. The initial fluid analysis was exudate based on Light's criteria with high protein. The fluid analysis was repeated five days later, after rapid reaccumulation which revealed transudates. Other causes of pleural effusion like heart failure, renal failure or primary pulmonary diseases were excluded. Hepatic hydrothorax was considered and the patient was started with the treatment of Furosemide and Spironolactone. The atypical presentation of hepatic hydrothorax may disguise the diagnosis and delay the treatment. Therefore, for a patient with recurrent, unexplained unilateral pleural effusions, even with atypical fluid characterisation and in the absence of ascites, hepatic hydrothorax should still remain on the top differential with underlying cirrhosis to ensure optimal treatment.

Keywords: Cirrhosis, Light criteria, Liver, Pleural effusion

CASE REPORT

A 78-year-old Caucasian female, with a past medical history of alcoholic cirrhosis, admitted for recurrent rectal bleeding secondary to rectal ulcers and was successfully treated with cautery and placement of clips. However, a recurrent massive right-sided pleural effusion without apparent ascites or dyspnea was found incidentally during the hospitalisation [Table/Fig-1]. Liver ultrasound findings were consistent with cirrhosis. Chest and abdominal CT were performed as a follow-up, which confirmed a large right-sided pleural effusion. There were no additional abnormal findings identified on CT, no acute infiltration, pulmonary oedema, pleural thickening or malignant disease, nor ascites. Oxygen saturation was 98% on room air for the patient. She had no leukocytosis or elevated hepatic transaminases [Table/Fig-2]. Renal function and electrolytes were



[Table/Fig-1]: Large right pleural effusion. The cardiomediastinal silhouette is within normal limits. Boney structure is unremarkable.

Haemogram	Levels	Normal range
WBC	6.9	(4.5-11.0 thousands/mm^3)
Neutrophils %	78 H	(50.0-75.0 %)
Lymphocytes %	11.6 L	(17.0-42.0 %)
Monocytes %	7.6	(4.0-11.0 %)
Eosinophils %	1.7	(0.4-6.0 %)
Basophils %	1.1	(0.0-2.0 %)
Absolute Neutrophil Count	5.4	(1.5-8 thousands/mm3)
RBC	2.56	(3.80-5.20 million cells/uL)
Hgb	7.6 L	(12.0-15.0 g/dL)
Hct	22.3 L	(35.0-49.0 %)
MCV	86.8	(80.0-100.0 fL)
MCH	29.7	(26.5-34.0 pg)
MCHC	34.3	(32.0-36.0 %)
RDW	17.1	(<17.0 %)
Platelet Count	80 L	(150 - 450 thousands/mm^3)
MPV	8.8	(6.6-10.2 fL)
Chemistry		
Sodium	141	(136-145 mmol/L)
Potassium	4.2	(3.5-5.1 mmol/L)
Chloride	114 H	(98-107 mmol/L)
Carbon Dioxide	19 L	(21-32 meq/L)
Total Bilirubin	1.8 H	(0.2-1.0 mg/dL)
AKP	46	(45-117 units/L)
AST	14 L	(15-37 units/L)
ALT	12	(13-56 units/L)
Total Protein	4	(6.4-8.2 g/dL)
Albumin	1.8	(3.4-5.0 g/dL)
Globin	2.2	(2.8-4.4 g/dL)
A/G Ratio	0.8	(1.3-2.8)
BUN	20 H	(7-18 mg/dL)
Creatinine	1.13	(0.60-1.30 mg/dL)

Glucose	129 H	(74-106 mg/dL)
Calcium	7.5 L	(8.5-10.1 mg/dL)
Phosphorus	3.7	(2.5-4.9 mg/dL)
Magnesium	1.8	(1.8-2.4 mg/dL)
NT-Pro-BNP	228	(0-900 pg/mL)
GFR	46	90-120 mL/min/1.73 m^2
BUN/CREA	17.7	(9.3-24.4 ratio)

[Table/Fig-2]: Complete blood count and blood chemistry.
Abbreviations: WBC: White Blood Cell Count; RBC: Red Blood Cell Count; Hbg: Haemoglobin; Hct: Hematocrit; MCV: Mean Corpuscular Volume; MCH: Mean Corpuscular Haemoglobin; MCHC: Mean Corpuscular Haemoglobin Concentration; RDW: Red Cell Distribution Width; MPV: Mean Platelet Volume; BUN: Blood Urea Nitrogen; GFR: Glomerular Filtration Rate;

Coagulation		
PT	12.4	(9-12.5 SEC)
INR	1.2	
APTT	25	(21-35 SEC)

[Table/Fig-3]: Coagulation test.
Abbreviations: PT: Prothrombin Time, INR: International Normalized Ratio, APTT: Activated Prothrombin Time

	1st fluid analysis	2nd fluid analysis	Transudate	Exudate
Color	Yellow, clear	Yellow, clear	Clear	Cloudy
PH	7.41	7.36	7.40 to 7.55	<7.40
WBC	106	126	< 1000	> 1000
SEG Neutrophils	2	2		
Lymphocytes	26	26		
Eosinophils	-	4		
Basophils	-	1		
Monocyte/ Macrophage	72	67		
Glucose	-	101mg/dL	> 60 mg/dL	< 60 mg/dL
Protein mg/ dL	4.9	2.2	< 3 g/dL	> 3 g/dL
Pleural protein / serum protein	0.66	0.42	< 0.5	> 0.5
SAAG	-	1.4	> 1.2 g/dL	< 1.2 g/dL
Pleural LDH / serum LDH	0.54	0.42	< 0.6	> 0.6
Cholesterol	-	< 50	<50	>50
Pathology	No malignant cells	No malignant cells		

[Table/Fig-4]: Fluid analysis.
Abbreviations: LDH: Lactate dehydrogenase;
SAAG: serum-ascites albumin gradient

also in the normal range [Table/Fig-2]. Hepatitis B and hepatitis C tests were negative. Coagulation tests were normal [Table/Fig-3]. The Echocardiogram showed normal heart function with Left Ventricular Ejaculation Fraction (LVEF) of 60% with no evidence of pericardial effusion. NT-proBNP was in the normal range.

An ultrasound-guided diagnostic thoracentesis was performed. Pleural fluid profile was consistent with an exudate based on Light's criteria, with apleural protein of 4.9 mg/dL and pleural/serum protein ratio of 0.66. Gram stain and fluid culture were negative. Cytology did not reveal any malignant cells. A right-sided chest tube was placed with evacuation of about 1.35 L of fluid. However, the massive pleural effusion reaccumulated 5 days later. An ultrasound-guided chest tube was again placed based on prior diagnostics. The pleural effusion was drained, producing 4L. Repeated fluid analysis showed a protein level of 2.2 mg/dL, fluid /serum protein ratio of 0.42, pleural/serum LDH ratio of 0.53, cholesterol < 50, serum-ascites albumin gradient >1.2 g/dL [Table/Fig-4]. The repeat fluid was a transudate via Light's criteria. Hepatic hydrothorax was considered and she was started on treatment with sodium restriction along with Furosemide 20mg and Spironolactone 50mg. She was maintained on diuretics at discharge. Four months later her chest X-ray showed stable small to moderated effusion but not as large as before. Since she had been asymptomatic, she was advised for palliative thoracentesis if she has shortness of breath.

DISCUSSION

Hepatic hydrothorax occurs in approximately 5% to 10% of patients with cirrhosis, and the outcome is usually very poor [1]. A few cases of hepatic hydrothorax in the absence of ascites were reported before [2,3]. Here, we presented a rare case with an incidental finding of recurrent massive right-sided pleural effusion in the absence of ascites. The initial fluid analysis was exudate based on Light's criteria. These atypical presentations made the diagnosis and treatment even more challenging.

The diagnosis of hepatic hydrothorax is based on the presence of a pleural effusion (usually >500 mL) in a patient with cirrhosis by excluding other underlying primary cardiopulmonary causes [4]. The pleural effusion probably results from diaphragmatic defects, allowing the ascites to move from the peritoneal cavity into the pleural cavity [5]. However, the presence of ascites is not necessary for the diagnosis. This may be due to the negative intrathoracic pressure generated during inspiration [6]. An intraperitoneal injection of 99mTc-sulphur colloid or 99mTc-human serum albumin may be helpful if the diagnosis is unclear [7].

Diagnostic thoracentesis of the pleural fluid is required, which is classically found to be a transudate. Light's criteria are the most widely used criteria for fluid analysis. Although the sensitivity of Light's criteria for a diagnosis of exudates is as high as 98%, the specificity is only about 80% [8]. Furthermore, it was reported that about 18% of hepatic hydrothorax was misclassified as exudates based on the Light's criteria [9]. Thus, clinical judgment is required when evaluating patients with borderline test results. With respect to the patient we presented here, the first fluid analysis was an exudative process via Light's criteria with high protein level, although the other biochemistry markers including LDH ratio, glucose level, cell counts and pH indicated a transudate. More recent studies have examined other characteristics of pleural fluid that may help to determine whether the effusion is exudate or transudate, like Serum-Ascites Albumin Gradient (SAAG). The combined use of Light criteria and the albumin gradient may significantly increase the specificity [9,10].

Chest tube placement is a common treatment for large pleural effusion, however, it was reported that patients with hepatic hydrothorax who were treated with chest tube actually had higher mortality and longer length of stay compared to those who underwent thoracentesis [11,12]. Chest tube placement is associated with many severe complications in patients with hepatic hydrothorax, like acute kidney injury, pneumothorax, haemothorax, empyema, unilateral pulmonary oedema due to the rapid removal of fluid. Therefore, a chest tube is a relative contraindication for the treatment of hepatic hydrothorax and should be avoided whenever feasible.

The initial therapy for hydrothorax includes low sodium diet and diuretics. Furthermore, therapeutic thoracentesis can be attempted if diuretics are not effective. Transjugular Intrahepatic Portosystemic Shunt (TIPS) placement can be considered for refractory hepatic hydrothorax [13]. Eventually, all patients with confirmed hepatic hydrothorax should be considered for evaluation of liver transplantation.

CONCLUSION

We presented a rare case of hepatic hydrothorax in the absence of ascites. The absence of ascites may be due to the negative intrathoracic pressure generated during inspiration. This atypical

presentation may disguise the underlying diagnosis. Therefore, for patients with underlying cirrhosis presented with unexplained unilateral pleural effusions, hepatic hydrothorax should always remain on the top differential to provide optimal treatment. Currently, initial treatment of hepatic hydrothorax includes sodium restriction and diuretics to increase kidney excretion. Refractory pleural effusion may require serial thoracentesis. Indeed, chest tube for prolonged aspiration should be avoided due to the complications and poor outcome in these patients.

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