Hepatic hydrothorax without ascites.

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ABSTRACT

Hepatic hydrothorax (HH) in the absence of ascites is a rare complication of liver cirrhosis. A 58-year-old woman presented to our department with dyspnea because of massive right pleural effusion requiring chest tube drainage. Investigations confirmed chronic hepatitis C, She had no signs of hepatic failure or ascites but there was evidence of portal hypertension. After pleural drainage and standard treatment for ascites with restriction of salt intake and diuretic administration, the pleural effusion improved with minimal recurrence.

KEYWORDS: Hepatic hydrothorax, Cirrhosis

CASE REPORT

INTRODUCTION

Hepatic hydrothorax (HH) is defined as significant pleural effusion, usually greater than 500 ml, in a patient with cirrhosis of the liver with no primary cardiac or pulmonary disease.[1] Its development is not associated with any particular cause of cirrhosis.[2]

Pleural effusion occurs in approximately 5% of patients with cirrhosis of the liver, depending on the criteria applied.[3] In this case report we discuss a patient presenting with right massive hydrothorax without ascites as a manifestation of liver cirrhosis. There was radiological evidence of portal hypertension.

CASE REPORT

A 58-year-old woman was admitted to our Ahmadi General Hospital, KOC, with a two week history of dyspnea, orthopnea and cough. She had a history of Caesarean section at the age of 22 in India. She had no history of blood transfusion. On physical examination, she looked well except for mild tachypnea. We noted decrease breath sound and dullness on percussion involving the lower right hemithorax. The liver and spleen were not palpable and there was no ascites. Palmar erythema and vascular spider were absent. Cardiac examination was normal. Laboratory data on admission are shown in Table I. Platelet count was low (70,000/µl) with hypoalbuminemia (27 gm/L) and prolonged prothrombin time. Hepatitis B surface antigen was not detected. Hepatitis C virus (HCV) antibody and HCV RNA were detected, indicating active HCV infection. Urinalysis revealed no proteinuria.

Chest X-ray showed massive right pleural effusion. A chest tube was inserted and 2700 ml of fluid was drained. Subsequently the daily volume of drained fluid was 2000 ml, and this remained unchanged for three days. Pleural fluid was transudative. Computed tomography (CT) of the chest revealed no evidence of tumor, lymphadenopathy or pleural-based lesion. Abdominal CT and ultrasonography revealed no ascites or focal hepatic lesion. There was portosystemic shunt shown on CT (Recanalized paraumbilical vein) [Figure 1]. Endoscope examination revealed grade 1 esophageal varices.

Patient was started on Albumin infusion, diuretics (Spironolactone, Frusemide) and low salt diet. The following days, her pleural fluid drainage decreased from 500 ml to 100ml per day. The chest tube was removed without any complication. She was maintained on diuretic therapy in addition to low salt diet. Her follow up chest X-ray after a week and two month later, showed no recurrence of effusion.

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Table I. Laboratory Data on Admission

Test	Result
White Cell Count (per mm3)	7,500
Red cell count	4.44
Hemoglobin (g/L)	13.4
Hematocrit (%)	38
Platelet count (per mm3)	70
Prothrombin Time (sec)	16
INR	1.4
Partial-thromboplastin time (sec)	27
Urea nitrogen (mmol/l)	5.4
Creatinine (umol/l)	78
Total Bilirubin (ummol/L)	30
Total Protein (gm/L)	65
Albumin (gm/L)	27
Alkaline phosphatase (U/L)	280
Aspartate aminotransaminase (U/L)	77
Alanine aminotransaminase) (U/L)	70
Anti-HCV (COI)	50
HCV genotype	1 b
HCV RNA (KIU/ml)	36

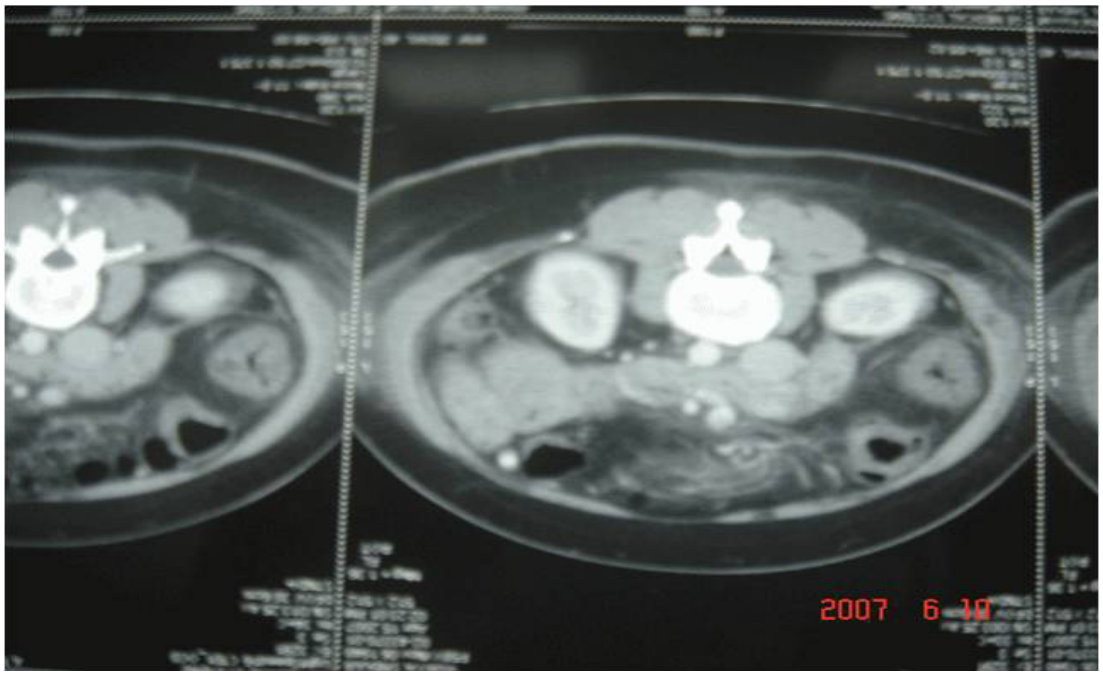

Figure 1. CT Abdomen showing Paraumbilical venous shunt

DISCUSSION

Hepatic Hydrothorax (HH) is a manifestation of decompensated chronic liver disease, similar to occurrence of ascites, hepatic encephalopathy and variceal hemorrhage. Although the exact mechanism involved in the development of HH has not been well-understood, several observations indicate that the most likely cause is passage of a large amount of the ascitic fluid from the peritoneal to the pleural cavity through diaphragmatic defects.[4,5,6] A defect in the diaphragm as a cause of HH in a patient with cirrhosis was first described in 1955 by Emerson.[4] HH occurs when the accumulation of ascitic fluid in the pleural cavity surpasses the absorption capacity of the pleura. Diaphragmatic defects represent small holes, usually less than < 1cm in the tendinous portion of the diaphragm.[7] The negative intrathoracic pressure favors the transfer of fluid across these defects. In most cases (85%) HH develops on the right side with 13% involving the

left side and 2% being bilateral.[8]

The most important aspect in the management of patients with cirrhosis and HH is evaluation for candidacy for liver transplantation.[7] Medical treatment consists of sodium restriction and diuretics. If medical treatment and therapeutic thoracentesis are needed repeatedly, three options are available for the treatment: pleurodesis through a chest tube drain, thoracoscopy with attempt of diaphragmatic repair followed by talc poudrage, and transjugular intrahepatic portosystemic shunting (TIPSS).[3]

Studies with 10 to 20 years of follow up suggest that cirrhosis occurs in up to 50 percent of chronically infected patients.[9] Complications of hepatitis C are mostly confined to patients who have developed cirrhosis. The development of cirrhosis is silent in the majority of cases.[9] Although these patients tend to be more symptomatic than those with chronic hepatitis alone, no clinical symptom, physical sign or lab test is particularly sensitive or specific for the diagnosis of cirrhosis. There are two important points in our case: firstly, massive pleural effusion without ascites is a rare presentation of hepatitis C with cirrhosis. This can lead to potential delay of definitive treatment and uncertainty as to its underlying cause. Large unilateral symptomatic pleural effusion usually tempts the insertion of chest tube. Chest tubes are sometimes used as treatment for HH, although patient with cirrhosis may have increased mortality with chest tube placement, with difficulty in removing the tube without correction of the underlying liver disease or portal hypertension.[10] Almost a third of all patients die with chest tube still in place.[10] Secondly, role of imaging in diagnosing liver cirrhosis. Diagnostic imaging does not have an important role in the diagnosis of liver cirrhosis since features of cirrhotic liver are present only in advanced disease.[11] Computed tomography (CT) and ultrasonography are, however, helpful in evaluating complications of cirrhosis, such as vascular patency and presence of neoplasm.[11] The CT abdomen in our patient showed paraumbilical vein shunt an important clue for diagnosing a patient presenting with massive pleural effusion for the first time. Paraumbilical vein shunt is an important prognostic factor that contributes, independently, to the prognosis of liver cirrhotic patients with portosystemic shunts.[12]

Regardless of the possible pathogenetic explanation the absence of ascites should not prevent the inclusion of cirrhosis in the differential diagnosis of an otherwise unexplained pleural effusion. This may avoid a delay in diagnosis and subsequent complication such as encephalopathy and unnecessary early chest therapeutic option such as chest tube drainage.

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