Cirrhosis of the liver, an exceptional cause of chylothorax: two cases

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Chylothorax, a condition featuring an infrequent form of pleural effusion, is generally caused by tumours or traumatism. Only about 1% of chylothorax cases are caused by cirrhosis of the liver. Two such cases are described in these case reports.

Introduction

Chylothorax is a relatively uncommon condition (1) where triglyceride-rich fluid containing chylomicrons accumulates in the pleural cavity. Light (2) has classified chylothorax cases into four major groups: chylothorax secondary to tumours; those caused by traumatism; idiopathic cases (including congenital chylothorax); and miscellaneous cases. This last group, which accounts for about 8% of all chylothorax patients, includes cases caused by cirrhosis of the liver, which make up approximately 1% of the total (1). These case reports describe two cases of right chylothorax caused by hepatic cirrhosis that were seen recently in the authors' clinic.

Case Reports

CASE NO. 1

Patient 1 was a 52-year-old male who drank approximately 180 g of ethanol day⁻¹, who was periodically admitted to hospital with hydropic decompensation, and a right pleural effusion with a serous appearance and the biochemical characteristics of a transudate (3). Hepatic cirrhosis had been diagnosed 2 yr before the present occasion, on which he came to the Emergency Service with periumbilical pain and a swollen abdomen after dyspnoea of 4 days duration. Physical examination showed normal blood pressure and no fever, jaundiced skin and mucosas with stigmata of chronic hepatopathy, signs of right pleural effusion, increased abdominal perimeter, a reducible umbilical hernia and surge ascites. Blood analysis results were as follows: haemoglobin 9.7 g dl⁻¹, haematocrit 30%, leucocytes 4800 mm⁻³, differential leucocyte counts normal, platelet count 130 000 mm⁻³, prothrombin activity 53%, partial thromboplastin time 33.5 s, triglycerides 34 mg dl⁻¹, total protein 5.5 g dl⁻¹, total bilirubin 5.1 mg dl⁻¹ (direct bilirubin 1.7 mg dl⁻¹), Na 126 meq 1⁻¹, other biochemical parameters normal. Chest X-rays showed massive right pleural effusion, and abdominal ultrasonography showed hepatosplenomegaly with a diffuse increase in hepatic echogenicity (but no focal lesions) and ascites. The ascites contained chylomicrons, had a milky appearance, and showed the following biochemical characteristics: total protein 1.1 g dl⁻¹, LDH 13 IU, cholesterol 10 mg dl⁻¹, triglycerides 200 mg dl⁻¹, amylase 12 U/L, ADA 4 U/L; culture of ascites gave negative results. Characteristics of the pleural fluid are listed in Table 1.

CASE NO. 2

A 40-year-old male who drank approximately 220 g of ethanol day⁻¹ was admitted on account of loss of consciousness and generalized tonic-clonic convulsions following massive ingestion of alcohol. Hepatic cirrhosis had been diagnosed 1 yr previously. Physical examination showed normal blood pressure, pulse and temperature, chronic hepatopathic stigmata, signs of right pleural effusion, 3 cm of hepatomegaly below the costal arch and surge ascites. Blood analysis results were as follows: haemoglobin 11.5 g dl⁻¹, haematocrit 34%, leucocytes 7800 mm⁻³, differential leucocyte counts normal, prothrombin activity 65%, partial thromboplastin time 35 s, other biochemical parameters normal. Chest X-rays showed massive right pleural effusion, and abdominal ultrasonography showed ascites.
signs of portal hypertension and homogeneous hepatosplenomegaly without focal lesions. No compression of the thoracic duct was observed in CT scans. The ascites contained chylomicrons, had a milky appearance, and showed the following biochemical characteristics: total protein 2.9 g dl⁻¹, LDH 137 IU, cholesterol 57 mg dl⁻¹, triglycerides 275 mg dl⁻¹, ADA 15 U/L; culture of ascites gave negative results. Characteristics of the pleural fluid are listed in Table 1.

**Discussion**

In their review of 191 cases described in eight series published between 1964 and 1986, Valentine et al. (1) noted that the commonest cause of chylothorax (45.5%) was neoplastic obstruction of the thoracic duct, 80% of these cases being due to lymphomas. The second most common cause (28%) was traumatic lesion of the thoracic duct (due in 90.5% of these cases to surgical procedures). The third largest group of cases (18%) comprised idiopathic and congenital chylothorax. The remaining 8% of cases were secondary to a variety of diseases including pulmonary lymphangioleiomyomatosis, tuberous sclerosis, and cirrhosis of the liver. Hepatic cirrhosis only accounted for about 1% of cases; indeed, the relative rarity of chylothorax secondary to hepatic cirrhosis is responsible for the description of individual cases in the literature (4). Chylothorax is diagnosed, as in the present two cases, on the basis of chylomicrons and a triglyceride concentration >110 mg dl⁻¹ in the pleural fluid (5). However, 30-40% of patients with chylous ascites have chylothorax (6).

A physiopathological mechanism by which hepatic cirrhosis might cause chylothorax was suggested by Dumont and Mulholland (7), who observed that hepatic cirrhosis patients with ascites exhibited increased hepatic capillary pressure associated with a proportional increase in lymph flow in the liver and thoracic duct. Since it seems likely that, in these patients, increased pressure in hepatic lymph vessels and the thoracic duct had led to extravasation of chyle and hence to the formation of chylous ascites (8), it is also possible that chylous fluid descending towards the peritoneal cavity might enter the pleural cavity via microscopic anatomical defects in the diaphragm, in the same way as pleural transudates secondary to ascitic hepatic cirrhosis (5).

To sum up, chylothorax is a condition featuring an infrequent type of pleural effusion that is usually caused by tumours (mainly lymphomas) or trauma-tism. Hepatic cirrhosis is an exceptional cause, but when chylothorax is diagnosed it should be borne in mind because patients with chylothorax originated by hepatic cirrhosis run a risk of malnutrition — and hence of deterioration — due to the accumulation of large quantities of protein, fat and electrolytes in the chylous pleural fluid.

**References**