Role of the peritoneopleural pressure gradient in the genesis of hepatic hydrothorax. An isotopic study

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Summary  Hepatic hydrothorax is defined as the development of significant pleural effusion in a patient with cirrhosis without primary pulmonary or cardiac disease. This complication is seen in 4—10% of patients with cirrhosis. The pleural effusion is a result of a direct passage of ascitic fluid into the pleural cavity through a defect in the diaphragm. We report two patients with posthepatitis cirrhosis presenting with a significant pleural effusion. The peritoneopleural communication was demonstrated by peritoneal scintigraphy. The role of the peritoneopleural pressure gradient is discussed.

Résumé  L’hydrothorax hépatique est défini par le développement d’un épanchement pleural chez un cirrhotique en l’absence de pathologie pulmonaire ou cardiaque. Cette complication se rencontre chez 4—10% des cirrhotiques. L’épanchement pleural résulte d’un passage direct du liquide d’ascite dans la cavité pleurale à travers des brèches diaphragmatiques. Dans ce travail, nous rapportons les cas de deux patients atteints de cirrhose posthépatitique présentant un épanchement pleural abondant. La communication peritonéopleurale est montrée par la scintigraphie péritonéale. Le rôle du gradient de pression péritonéopleural est discuté.

Introduction

Hepatic hydrothorax is defined as the presence of marked pleural effusion in a patient with cirrhosis without primary
pulmonary or cardiac disease. Pleural effusion occurs in approximately 4–10% of patients with cirrhosis and it is frequently on the right side [1–3].

A number of different mechanisms have been suggested to explain the development of pleural effusion in patients with cirrhosis. The most likely explanation is a direct passage of the ascitic fluid into the pleural space through congenital or acquired defects in the diaphragm [1,2].

The purpose of this report is to demonstrate the development of pleural effusion by the isotopic method in two patients with cirrhosis. The role of the peritoneopleural pressure gradient is discussed.

Case reports

Case 1

A 46-year-old man with a history of postviral hepatitis B cirrhosis (child’s disease class C) was admitted to hospital for abdominal distension and progressive dyspnea. On admission the patient was apyretic and had tachypnea. Physical examination showed significant ascites, dullness to percussion and absence of breath sounds over the right hemithorax. The admitting chest X-ray showed a large right pleural effusion. Analysis of the ascitic liquid showed a protein level of 10 g/L and a cell count of 220 per milliliter. Thoracentesis yielded a large amount of transudative liquid that was similar to the ascitic fluid, suggesting an abdominal origin. After pleural evacuation the respiratory symptoms diminished but effusion rapidly recurred. To confirm thoracoabdominal communication, peritoneal scintigraphy was performed. After an intraperitoneal injection of 185 MBq (5 mCi) of Tc-99m macroaggregated serum albumin, rapid passage of radioactivity into the right pleural space was observed confirming the abdominal origin of the pleural fluid and suggesting a large diaphragmatic defect (Fig. 1). The initial speed of the radioactive flow was 460 counts per minute. A second dynamic acquisition was performed immediately after a pleural evacuation of 1.5 L of fluid. A significant increase in the passage of radioactivity into the pleural cavity was observed with a speed reaching 1080 counts per minute (Fig. 2).

Abdominal ultrasound showed an atrophic liver and a large defect in the right hemidiaphragm. Medical treatment associating a low salt diet and spironolactone led to progressive regression of the pleural effusion and ascites. No recurrence of the pleural effusion was observed 12 months later.

Case 2

A 42-year-old woman with a two-year diagnosis of postviral hepatitis B cirrhosis (child’s disease class B) was admitted for progressive dyspnea and abdominal distension. At physical examination the patient was subicteric with shortness of breath and an enlarged abdomen. Breath sounds were absent throughout the entire right hemithorax. A chest X-ray revealed massive right-sided pleural effusion of the whole pleural cavity (Fig. 3). A diagnostic pleural tap produced a clear noninfected yellow fluid with low protein content. Peritoneal scintigraphy performed after injection of 185 MBq (5 mCi) of sulfur colloid particles confirmed the direct communication between the peritoneal and pleural cavities with an immediate migration of radiotracer into the right pleural space (Fig. 4). Injection of sulfur colloid particles into the pleural space three days later showed no abdominal passage of radioactivity even on the image performed at the twenty-fourth hour (Fig. 5). This confirmed the one-way...
movement of ascitic liquid from the abdomen to the pleural cavity.

The patient was treated with sodium restriction, diuretics (furosemide 40 mg/day and spironolactone 100 mg/day) and repeated thoracocentesis. The patient’s condition progressively improved and pleural effusion disappeared 10 days later. During two years follow-up, pleural effusion recurred eight times as a result of poor compliance to medical treatment.

Discussion

Hydrothorax is an uncommon complication of cirrhosis occurring in four to 10% of patients with cirrhosis. Although ascites is usually present, pleural effusion can develop in the absence of ascites [4,5]. The exact mechanism of pleural effusion has been the subject of much debate and different mechanisms have been proposed to explain its development [1,2]. The most commonly accepted mechanism is that pleural effusion originates secondary to direct passage of ascitic fluid through congenital or acquired defects in the diaphragm into the pleural space. Such defects have been well documented by imaging techniques [6,7], thoracoscopy [8,9] or at autopsy [10,11].

The diagnosis of hepatic hydrothorax in a patient with advanced cirrhosis presenting with pleural effusion is usu-
ally obvious. In some cases, particularly when ascites is minimal or when effusion occurs in the left hemithorax, the diagnosis is more difficult and pleuropertitoneal communication must be demonstrated. Several methods are available and include intraperitoneal injection of blue dye [12], air [13,14] or radiolabeled particles [4,15,16] to evaluate pleural migration. The isotopic method, using Tc-99m macroaggregated serum albumin or Tc-99m sulfur colloid particles, is used most frequently because it is simple and safe. These radiolabeled particles, measuring between 3 and 100 μm, are not absorbed by the peritoneum so their intrapleural passage occurs only through an anatomical defect in the diaphragm. It has been demonstrated that both radiotracers are effective in demonstrating peritoneopleural communication, either with or without ascites [15-17]. Some authors recommend the intraperitoneal instillation of 300–500 mL of normal saline in cases with minimal ascites to favour the pleural passage of radioactive tracer [15]. The isotopic method can also provide an estimation of the size of the defect in the diaphragm by the speed that the radioactivity passes into the pleural cavity. Pleural activity that occurs a few minutes after peritoneal injection of tracer is usually associated with a significant defect [17,18]. This finding is clearly demonstrated in our first patient.

Several reports have demonstrated the one-way movement of fluid from the abdomen to the pleural space [19,20]. This unidirectional flow was clearly verified in our second patient who had no abdominal passage of radioactivity 24 h after intrapleural injection of sulfur colloid particles despite the large size of the suspected defect on peritoneal scintigraphy. This one-way movement of ascitic fluid is probably due to a permanent gradient pressure as a result of a negative intrathoracic pressure during the respiratory cycle and a positive intra-abdominal pressure [3]. This mechanism was strongly supported by Ikard et al. who found a resolution of pleural effusion during continuous positive artificial ventilation [21]. A siphon mechanism has also been suggested to explain the unidirectional movement of ascitic fluid but this seems less probable because it does not explain the unidirectional movement during a large defect and the reversible movement of liquid after positive artificial ventilation. As suggested by this report, significant pleural effusion may be a cause of false negative peritoneal scintigraphy results because the high pleural pressure can annul the peritoneopleural pressure gradient and prevent leakage of peritoneal activity into the pleural space. LeVeen et al. who demonstrated the pleural passage of peritoneal activity only after evacuating the pleural space supports this [23]. Therefore, the author recommends pleural evacuation in patients with abundant pleural effusion. Our report also illustrates the rapid reconstitution of the pleural effusion after thoracocentesis emphasizing that ascites should be completely controlled to obtain resolution of pleural effusion.

The treatment of hydrothorax is primarily medical, associating salt restriction, diuretics and pleural evacuation. With this treatment, pleural effusion is resolved in most patients [2]. When hydrothorax resists medical treatment, intrapleural injection of sclerosing agents, such as t alc or minocycline, can be attempted to obtain pleural symphysis [9,24]. These sclerosing agents are safe because they remain confined to the pleural space and do not damage the abdominal organs. Other more invasive methods may be used including peritoneovenous shunts, transjugular intrahepatic portosystemic shunt, and surgical or endoscopic repair of the defective diaphragm [14,25]. Liver transplantation, when possible, constitutes the only definitive treatment of hydrothorax and cirrhosis. Xi ol et al. reported a mean survival of 97 months with 70% survival at five years in 28 patients with hydrothorax treated with liver transplantation [26].

In conclusion, peritoneal scintigraphy is a simple and effective method to demonstrate pleuropertitoneal communication in patients with cirrhosis. Knowledge of the exact mechanism of the development of pleural effusion can help decide treatment. This paper and others have shown that the peritoneopleural pressure gradient forces the ascitic fluid into the pleural cavity across a defective diaphragm. Most therapeutic methods target the reduction of the peritoneopleural pressure gradient (diuretics, peritoneovenous shunts and transjugular intrahepatic portosystemic shunt) and closure of the defective diaphragm. Sclerosing agents can be safely used to obtain pleural symphysis thanks to the unidirectional movement of liquid from the abdomen to the pleural cavity.

References
