with intact mitral valve prostheses is difficult since afterload reduction cannot redistribute regurgitant flow to the systemic circulation. In our patient, the success of medical management for 2 years suggests that the pseudoaneurysm acted initially as an “auxiliary atrium” which decompensated following afterload reduction, resulting in increased cardiac output. The recurrence of intractable heart failure after 2 years suggests that remodeling of the left ventricle may have contributed to cardiac decompensation.6

Our experience with this case strongly suggests that heart failure due to a large left ventricular pseudoaneurysm can be stabilized initially with vigorous medical treatment. However, because of the mechanical nature of the lesion, recurrent and refractory heart failure should be anticipated. In view of often prolonged delays for suitable cardiac donors, the patient with a large pseudoaneurysm, even if initially stable, may be considered for heart transplantation prior to decompensation.

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Peritoneal-Pleural Communications in Hepatic Hydrothorax Demonstrated by Thoracoscopy*

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To understand the mechanism of hepatic hydrothorax clearly, thoracoscopy was performed with a flexible bronchoscope. It revealed an intrathoracic influx of ascitic fluid via a bleb and two defects located in the tendinous portion of the right hemidiaphragm and confirmed the existence of transdiaphragmatic peritoneal-pleural communications. (CHEST 1996; 109:579-81)

Key words: fibrin glue; hepatic hydrothorax; OK-432; peritoneal-pleural communications; pleurodesis; thoracoscopy

Numerous investigations have been conducted on cases of hepatic hydrothorax complicated by cirrhosis. It has been suggested that the mechanism of the disease involves transdiaphragmatic influx of ascites into the intrathoracic space.

In a case of hepatic hydrothorax complicated by cirrhosis and hepatocellular carcinoma, thoracoscopy was performed to observe directly the transdiaphragmatic flow of ascitic fluid into the pleural cavity via a diaphragmatic bleb and defects. In this study, the usefulness of thoracoscopy in understanding the mechanism of hepatic hydrothorax is discussed.

Case Report

A 72-year-old woman, whose condition had been diagnosed as cirrhosis of the liver complicated by hepatocellular carcinoma after contracting the hepatitis C virus in 1986, was admitted to our hospital in February 1992 for coughing and dyspnea arising from massive right pleural effusion. She had been admitted to the hospital four times previously for the same symptoms. A physical examination at the time of admission revealed oedema, dyspneic conditions with vascular spider, palmar erythema, and icterus but no edema. Auscultation revealed no audible breathing in the right side of the chest. The abdomen was distended and tympanic with hepatospplenomegaly. Laboratory findings showed a normal peripheral blood cell count except for a platelet count of 51,000/mm³. Serum chemistry values revealed the following: aspartate aminotransferase, 84 U/L; alanine aminotransferase, 57 U/L; total protein value, 6.7 g/dL; albumin level, 3.1 g/dL; alkaline phosphatase, 149 U/L; total bilirubin value, 5.1 mg/dL; direct bilirubin value, 2.6 mg/dL; cholinesterase, 48 U/L; total cholesterol level, 83 mg/dL; and BUN value, 24 mg/dL. The value of α-fetoprotein was 1,545 ng/mL. A chest x-ray film showed marked pleural effusion in the right lung and a leftward shift of the mediastinum. Fluids obtained by thoracentesis and paracentesis, respectively, had similar characteristics: yellowish transudative fluid (total protein level, 0.7 and 0.8 g/dL); almost identical electrolyte levels; carcinoembryonic antigen, both 0.5 ng/mL; α-fetoprotein, 198 and 224 ng/mL; adenosine deaminase, 2.1 and 2.4 U/L; both cytologic study findings showed negative. Cultures for bacteria showed no recognizable organisms. To prove peritoneal-pleural communications, a pneumoperitoneum was performed to provide the pneumothorax on the right side with subdiaphragmatic free air (Fig 1). Furthermore, thoracoscopy with a flexible bronchoscope performed to aid in the detection of the communications revealed a bleb and two defects in the tendinous portion of the right hemidiaphragm (Fig 2). The bleb moved in response to changes in intrathoracic pressure, shrinking during inspiration and expanding during expiration. An intraperitoneal injection of indocyanine green dye showed an influx of greenish ascitic fluid via the defects during suction with a scope. The diaphragmatic bleb and defects were scattered with fibrin glue injected through the scope. Additional administration of diuretics and pleurodesis with OK-432 and minocycline on the 9th and 13th

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days of hospitalization resulted in the elimination of the ascites and pleural effusion, and the patient was subsequently discharged from hospital. However, 3 months later during readmission for hepatic encephalopathy, the patient died of hepatic failure caused by hepatocellular carcinoma.

**Discussion**

Since Morrow et al. described a patient with rapid accumulation of massive right pleural effusion following a diagnosis of cirrhosis of the liver as “hepatic hydrothorax,” various investigations of its cause have been presented. However, evidence remains limited in demonstrating intrathoracic transformation of air, radioisotopes, or other substances by intraperitoneal injection during the course of treatment and in detecting any diaphragmatic blebs or defects at autopsy.

Confirmation of peritoneal–pleural communications through the detection of a pneumoperitoneum-induced pneumothorax prompted us to perform thoracoscopy. A bleb and two defects in the right hemidiaphragm through which an intrathoracic influx of ascitic fluid was observed.

**Figure 1.** Chest x-ray films after pneumoperitoneum showed a right pneumothorax (left) and air (right) under both leaves of the diaphragm 1 h after diagnostic pneumoperitoneum.

**Figure 2.** Thoracoscopy revealed one bleb (arrow) and two defects (arrowhead) on the tendinous portion of the right hemidiaphragm through which an intrathoracic influx of ascitic fluid was observed.
iaphragn and intrathoracic influx of ascites wereverified. Lieberman et al\(^2\) reported a case wherein air bubbles following the pneumoperitoneum were identified by thoracoscopy. To our knowledge, however, there have been no reports documenting by thoracoscopy any intrathoracic influx of ascites during the course of treatment.

Our result yielded the following hypotheses on the pathogenesis of hepatic hydrothorax: First, accumulated ascites leads to the elevation of intraperitoneal pressure, causing formation of a bleb in the tendinous portion of the diaphragm. Second, the bleb ruptures and becomes a defect. Third, via the defect, aspiration of ascites to the intrathoracic space is induced by negative pressure. Finally, symptoms result from the accumulation of pleural effusion.

The treatment of hepatic hydrothorax requires a reduction of ascites by sodium restriction and diuretic agents. However, aggressive treatment of hydrothorax and ascites can lead easily to hepatic encephalopathy. In fact, three cases of hepatic hydrothorax encountered by the authors progressed to hepatic encephalopathy. Although peritoneovenous shunt (LeVeen shunt),\(^5\) surgical repair of the defects, or pleurodesis,\(^4\) or all three, have been performed as standard treatment procedures, these conventional techniques are limited in their range of applications. Drainage of the pleural fluid, although helpful for relieving symptoms, tends to deter closure of the diaphragmatic defects\(^3\) or aggravate existing conditions as a result of massive protein and electrolyte depletions.\(^6\) In the case of the present study, pleurodesis and diuretics administered after scattering fibrin glue were effective, causing hydrothorax and ascites to disappear completely. However, recurrent hepatic encephalopathy which developed after discharge from the hospital required readmission for further treatment. For the definitive treatment of hepatic hydrothorax associated with end-stage cirrhosis, liver transplantation should be considered.

We conclude that examination by thoracoscopy, not being a routine application in diagnosis, is useful in understanding the pathophysiologic mechanism of hepatic hydrothorax.

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