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Case Report

Hepatic hydrothorax after blunt chest trauma

Shang-Chiung Wang [a], Chih-I. Chen [a], Chen-Chi Liu [a], Chien-Ying Wang [a], Isaac Chun-Jen Chen [a], Mu-Shun Huang [a,b,*]

[a] *Division of Trauma, Department of Emergency Medicine, Taipei-Veterans General Hospital, Taipei, Taiwan, ROC*
[b] *Department of Emergency Medicine, National Yang-Ming University School of Medicine, Taipei, Taiwan, ROC*

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Abstract

We report a successful treatment result in a rare case of hepatitis C virus-related cirrhosis, who had sustained hydrothorax after blunt thoracoabdominal trauma. This was a female patient with liver cirrhosis, Child–Turcotte–Pugh class A, without ascites before injury. She sustained blunt thoracoabdominal trauma with a left clavicle fracture dislocation and right rib fractures. There was no hemopneumothorax at initial presentation. However, dyspnea and right pleural effusion developed gradually. We inserted a chest tube to relieve the patient's symptoms, and the daily drainage amount remained consistent. Hepatic hydrothorax was confirmed by the intraperitoneal injection of radioisotope 99mTc-sulfur colloid that demonstrated one-way transdiaphragmatic flow of fluid from the peritoneal cavity to pleural cavities. Finally, the hydrothorax was treated successfully by minocycline-induced pleural symphysis. To the best of our knowledge, this is the first case of hepatic hydrothorax developed after thoracoabdominal trauma.

Keywords: chest; hemothorax; hydrothorax; injury; radiography

1. Introduction

Blunt thoracic trauma, a common trauma, comprises about 75% of all thoracic injuries,[1] and minor ones make up a great part. Common complications of minor blunt chest trauma include muscle contusion, rib fractures, hemothorax, and pneumothorax. Critical management of hemopneumothorax is guided by chest X-ray, otherwise by pain control and respiratory care. When the chest X-ray of a patient who has sustained blunt chest trauma shows opacity in the pleural cavity, hemothorax is the diagnosis in most cases. Tube drainage is indicated for moderate to huge amount of hemothorax. In some cases, however, hydrothorax is diagnosed after the drainage has been performed. Of these, hepatic hydrothorax is very rare and develops only in patients with liver cirrhosis. Here, we present a successful treatment result in a rare case of hepatic hydrothorax after blunt thoracoabdominal injury.

2. Case report

A 75-year-old female sustained a blunt chest trauma following a road traffic accident and was sent to a community hospital for first aid. She had had hypertension for many years that was controlled with amlodipine. She also had hepatitis C-related liver cirrhosis, Child–Turcotte–Pugh class A. On arrival at the initial community hospital, her vital signs were stable and consciousness was clear; physical examination at that time revealed multiple superficial bruises on face, back, and limbs. There was no evidence of active bleeding, expansile or pulsatile hematoma, or subcutaneous emphysema. A chest X-ray revealed left clavicle fracture but no hemopneumothorax.

The patient was admitted to the hospital, fasted, and received supportive care in the form of intravenous fluid and analgesics. Three days after the injury, she underwent open

* Corresponding author. Dr. Mu-Shun Huang, Division of Trauma, Department of Emergency Medicine, Taipei Veterans General Hospital, 201, Section 2, Shih-Pai Road, Taipei 112, Taiwan, ROC.
E-mail address: mshuang@vghtpe.gov.tw (M.-S. Huang).

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reduction and internal fixation for the clavicle fracture. However, after the orthopedic operation, she developed progressive exertional dyspnea and right-side pleural effusion. Thoracentesis yielded a hemorrhagic fluid. Pleural fluid biochemistry showed an exudative pleurisy with a pH of 8.0. In the microscopic examination of the pleural fluid, there were high red blood cell (RBC) count of 41,535/mm^3, white blood cell (WBC) count of 107/mm^3, and few lymphocytes. Pleural fluid smears and cultures were negative for bacteria and acid-fast bacilli. The patient experienced mild relief from dyspnea after thoracentesis. The patient was considered to have traumatic hemothorax.

However, due to persistent dyspnea, she was transferred to our hospital on the 13th day after injury. On admission, the patient seemed well except for moderate dyspnea; we noted dullness to percussion above the right lung. Chest radiography confirmed a large right pleural effusion (Fig. 1); chest tube thoracostomy was done to drain fluid and 2700 mL of fluid was drained. However, the drainage fluid appeared clear transudate and, under microscopic examination, showed RBC count of 480/mm^3, WBC count of 170/mm^3, pleural fluid lactate dehydrogenase (LDH) level of 99 U/L, and serum LDH level of 407 U/L. Subsequently, the daily volume of drained fluid was 1000 mL, and this volume remained unchanged. Culture and cytology of pleural effusion were all negative. Abdominal ultrasonography and computed tomography (CT) was performed for evaluation of liver cirrhosis, and no evidence of ascites was found. As the drainage amount persisted to be huge every day, radioisotope scintigraphy with Tc-99m phytate was arranged, and hepatic hydrothorax was finally confirmed (Fig. 2). A combination of diuretic therapy and free-water restriction and chemical pleurodesis with minocycline was given through the chest tube. Three days later, the drainage volume had decreased to 100 mL a day, and the drainage tube was then removed. X-ray film of the chest showed no more residual pleural effusion, and the patient was discharged in stable condition. The patient remained asymptomatic, and the chest roentgenogram did not show pleural fluid re-accumulation at 1-month follow-up.

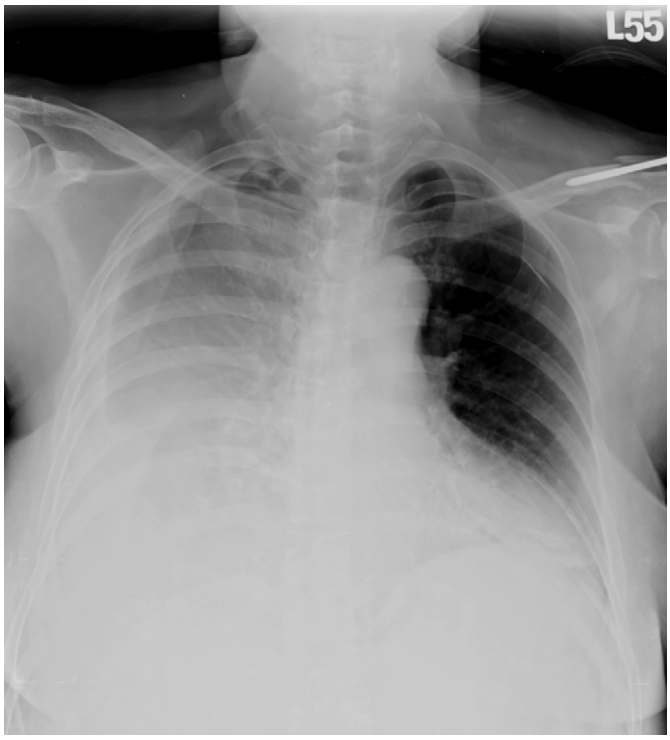

Fig. 1. Chest radiography performed on the 13th day after injury showing right massive hydrothorax.

3. Discussion

Hepatic hydrothorax is a relatively uncommon complication of portal hypertension with an estimated prevalence of 5–12% in patients with cirrhotic liver.[2] Pleural effusion usually occurs on the right side (65–87% of reported cases) and is combined with ascites.

First described by Emerson in 1955, the proposed pathogeneses of hepatic hydrothorax is diaphragmatic fenestration.[3] Microscopic examination of these diaphragmatic defects shows discontinuities in the collagen bundles of the tendinous portion of the diaphragm.[4] As a result of ascites or straining, increases in the intra-abdominal pressure may lead to small herniations of the peritoneum through these defects into the pleural cavity. These herniations, also called pleuroperitoneal blebs, may rupture and allow free communication between the peritoneal and the pleural cavity. This theory is supported by the fact that radiolabeled substance injected intra-abdominally in patients with hepatic hydrothorax moves into the pleural cavity.[5] Autopsy studies also suggest that pleuroperitoneal blebs occur less frequently in the left hemidiaphragm, as it seems to be thicker and more muscular.[6] Hence, blunt chest trauma may produce sudden and abrupt increases in the pleuroperitoneal pressure gradient, followed by the rupture of pleuroperitoneal blebs. This theory can explain the

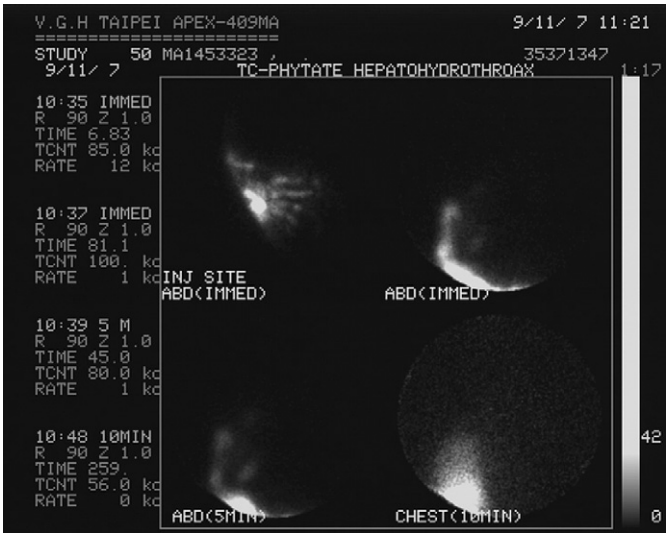

Fig. 2. Tc-99m phytate hepatohydrothorax scintigraphy revealing that the isotope injected in the abdomen migrated to the chest (arrowhead) 10 minutes later.

development of right hepatic hydrothorax after blunt thoracoabdominal trauma in this patient, and the diaphragmatic defect caused by blunt trauma may be few and small because of the effectiveness of chemical pleurodesis.

Hepatic hydrothorax should be suspected in patients with unilateral pleural effusion and established liver cirrhosis. Although majority of these cirrhotic patients have ascites, hepatic hydrothorax may occur in the absence of ascites.[7] Clinically, the most commonly experienced symptoms in patients with hepatic hydrothorax are dyspnea, chest pain, and nonproductive cough. The severity of these symptoms and signs depends on the rate of fluid accumulation and the total volume of pleural fluid. A diagnostic thoracocentesis should be performed and primary cardiopulmonary causes should be excluded in all patients with suspected hepatic hydrothorax. In trauma patients, pleural effusion analysis is particularly important when the clinical course and characteristics are unusual. Further investigations including chest CT, pleural biopsy, and thoracoscopy might be needed. If the diagnosis of hepatic hydrothorax is in doubt, especially if ascites are absent or pleural effusion is left sided, radioisotope scintigraphy with intraperitoneal injection of 99mTc-sulfur colloid is helpful. Migration of radioisotope into the pleural cavity within hours confirms the communication between the pleural and peritoneal spaces. This study has sensitivity of 71% and specificity of 100%.[8]

Therapeutic management for hepatic hydrothorax includes sodium-restricted diet, diuretics, transjugular intrahepatic portsystemic shunts, and surgical interventions.[7] Tube thoracostomy with chemical pleurodesis, repair of diaphragmatic defects, and peritoneovenous shunts are surgical approaches. Considering the mild severity of liver cirrhosis, absence of obvious ascites, and the recent event of blunt trauma, pleurodesis with minocycline was adopted in this patient.

To the best of our knowledge, we believe that this is the first reported case in which a minocycline pleurodesis procedure used successfully for symphysis resulted in control of blunt thoracoabdominal trauma and of hepatic hydrothorax. The indication for consideration of chemical pleurodesis should be expanded to include the potential treatment of diaphragmatic defects and of massive hydrothorax after blunt thoracoabdominal trauma.

In conclusion, traumatic hepatic hydrothorax is a rare complication in cirrhotic patients after blunt thoracoabdominal trauma. Minocycline pleurodesis is a safe and convenient procedure. Treatment of this complication is similar to that in nontrauma patients.

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