A 89-year-old female presented with progressive dyspnea and abdominal distention for three days. She had a history of hepatitis C virus related liver cirrhosis, Child classification C. Her vital signs were temperature of 35.8°C, pulse rate of 125/min, respiratory rate of 26/min, and blood pressure of 142/80 mmHg. No cough, sputum, hemoptysis, chest pain, fever, night sweating or weight loss was complained. Physical examination showed there was a dull percussion and decreased breath sounds over the right lung field. The abdomen was soft with shifting dullness. No lymphadenopathy was palpable in the neck, axillary or inguinal area. The computed tomography and sonography of chest and abdomen showed liver cirrhosis, splenomegaly, pleural effusion and ascites (Fig. 1), but excluded the existence of lymphadenopathy or a solid tumor. Subsequent

![Fig. 1](image.png)
thoracentesis and paracentesis yielded the milky fluid (Figure 1C). Laboratory examinations showed total bilirubin of 6.2 mg/dL, total protein of 7.9 g/dL, albumin of 3.1 g/dL, triglyceride of 128 mg/dL, lactic dehydrogenase (LDH) of 428 U/L, and prothrombin time prolongation of 5.6 seconds. The level of LDH, total protein, and cholesterol were 186 U/L, 3.2 g/L, 36 mg/dL in pleural effusion and 128 U/L, 1.8 g/L, 28 mg/dL, respectively. However, the examination of the pleural effusion and ascites both revealed transudate with elevated triglyceride of 317 mg/dl and 292 mg/dl, respectively, but the results of cytologic testing, and tuberculosis culture of samples were all negative. Therefore, the diagnosis of chylothorax and chyloperitoneum was confirmed. Peritoneal scintigraphy detected radioactivity in the pleural cavity after intraperitoneal injection of radiotracer for 30 minutes. Despite palliative management with repeated drainage and a modified diet with medium-chain triglycerides, her condition rapidly deteriorated and expired eventually.

Chyloperitoneum, an uncommon event that occurs when milk-like lymphatic fluid accumulates in the peritoneum space, could be caused by abdominal surgery, blunt abdominal trauma, malignant neoplasms, spontaneous bacterial peritonitis, pelvic irradiation, peritoneal dialysis, and abdominal tuberculosis. Liver cirrhosis has been rarely reported as one of the possible etiologies of chyloperitoneum and chylothorax (1-4). In the present reported case, after exclusion of other etiologies, it is concluded that chylous ascites result from liver cirrhosis and move from the peritoneum into the pleural space across the defects of diaphragm.

The pathophysiology of chyloperitoneum secondary to liver cirrhosis remains unclear. One possible mechanism is that increasing hepatic capillary pressure and proportionately increasing lymph flow in the liver and the thoracic duct would occur in cirrhotic liver. Such an elevated pressure in hepatic lymph vessels and the thoracic duct might increase the risk of extravasation of chyle and cause the development of chylous ascites (5).

The most common etiologies of chylothorax are tumor and trauma, which cause obstruction or disturbance of the thoracic duct or one of its major divisions. In addition, several unusual causes, including filariasis, tuberculosis, pulmonary lymphangiomyomatosis, and intestinal lymphangiectasis were ever reported. However, our case demonstrated that chylothorax could be resulted from the leakage of chylous ascitic fluid from the peritoneal cavity into the pleural space through the microscopic anatomical defects in the diaphragm and a negative intrathoracic pressure would aid the unidirectional movement of chylous ascites from the peritoneum, across these defects of the diaphragm and into the pleural space (6). In a retrospective study by Maldonado et al, the presentations of chylothorax are protean, and different etiologies of chylothorax may have different appearance and biochemical characteristics (7). Transudative chylous effusion was uncommon, and most of them were caused by liver cirrhosis, such as our case.

Like our case, the prognosis of chylothorax secondary to liver cirrhosis have been poor and worse than other common causes of chylothorax due to malignancy or trauma (3,8). No procedure directed at controlling the pleural fluid, such as pleurodesis, thoracoscopic repair, and transjugular intrahepatic portosystemic shunt have been reported to be effective (9).

In conclusion, chylothorax and chyloperitoneum should be considered a possible complication in a patient with liver cirrhosis and the outcome is poor.
References


