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Hepatic Chylothorax: An Uncommon Pleural Effusion

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Abstract

An 83-year-old male with chronic obstructive pulmonary disease and liver cirrhosis presented with confusion and dyspnea. On chest X-ray, he had the right mid to lower lung zone white out. Ultrasound-guided thoracentesis drained 1.5 L of milky white pleural fluid which was transudative according to chemical analysis. Transudative chylothorax in liver cirrhosis without ascites is rare, but can happen. When the flow of ascitic chylous fluid into the pleural space equals the rate of ascites production, clinical absence of detectable ascites will occur. Hepatic chylothorax is important and should be kept in differentials when evaluating patients with liver cirrhosis.

Keywords

chylothorax, transudative, exudative, pleural effusion, liver cirrhosis, hepatic hydrothorax, ascites

Background

Hepatic hydrothorax occurs in 5% to 10% of cirrhotic cases whereas chylothorax is an extremely rare finding.[1,2] Chylothoraces are usually exudative but those associated with liver cirrhosis are transudative.[3] Liver cirrhosis accounts for only 1% of chylothorax and occur in association with chylous ascites.[4,5] We present an unusual case of transudative chylothorax due to liver cirrhosis without the presence of significant ascites. Understanding the biochemistry and physiology of chylothorax in liver cirrhosis can help in avoiding unnecessary diagnostic procedures and also aide in effective management.

Case Presentation

An 83-year-old male was brought to the emergency room (ER) from nursing home due to altered mental status. Patient's past medical history was significant for hepatic cirrhosis secondary to hepatitis C virus and chronic respiratory failure with baseline oxygen requirement of 2 L by nasal cannula. At baseline, patient had dementia, but nursing home personnel reported worsening confusion in past few days. Home medications included lactulose and rifaximin for liver cirrhosis. Patient was not able to provide much history due to confusion, but he denied chest pain, cough or chills. On physical examination, patient was not oriented to time or place, and there was dullness to percussion with absent breath sounds in the lower right side of chest. No signs of ascites were found on abdominal examination. Patient was afebrile with temperature 97.5 F. The respiratory rate was 16/min, blood pressure 116/74 mm Hg and pulse ox was 97% on 2 L of oxygen. Labs were significant for moderately elevated ammonia levels of 67 µmol/L (normal range: 11-32 µmol/L). Urinalysis was positive for urinary tract infection. Arterial blood gas (ABGs) showed respiratory acidosis with metabolic compensation (pH 7.35 cmH_2O, PCO_2 57 mm Hg, PO_2 84 mm Hg, HCO_3 31.5 mm Hg, PaO_2/FiO_2 300). Chest X-ray (Figure 1) showed white out in right mid and lower lung zones suggestive of pleural effusion or pneumonia. Bedside ultrasound showed large right pleural effusion and no ascitic fluid. The computed tomography (CT) head was negative for acute intracranial changes. The echocardiogram showed a normal ejection fraction.

Differential diagnosis included hepatic, metabolic, respiratory encephalopathy, and sepsis among other diagnoses. Patient's lactulose dose was increased and patient had bowel

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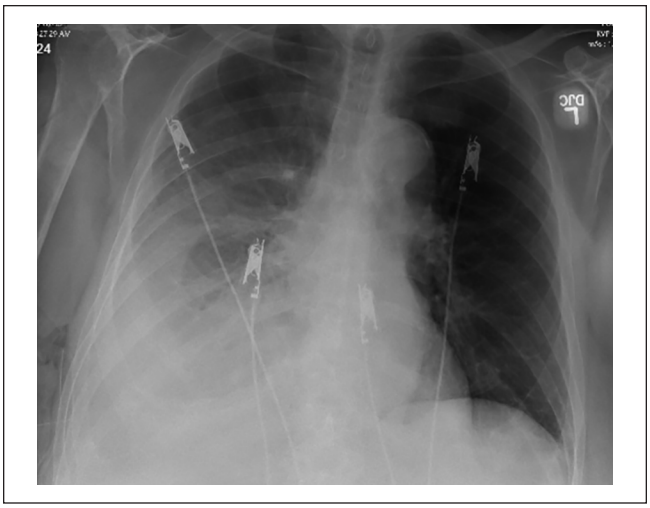

Figure 1. Chest X-ray showing moderate right pleural effusion.

movements with decline in ammonia levels. He was also started on antibiotics Ceftriaxone for treatment of urinary tract infection (UTI). He was started on intravenous diuretics and chest X-ray was repeated in 3 days, which did not show any change in pleural effusion. An ultrasound guided thoracentesis was done which drained 1.5 L milky white fluid from the right pleural space (Figure 2). Pleural fluid analysis showed protein concentration of 1.0g/dL, lactate dehydrogenase (LDH) of 45 μ/L, glucose of 97mg/dL and predominantly mononuclear white blood count (WBC) of 68.2%. Pleural fluid culture was negative for any growth. Pleural fluid cytology negative for malignant cells. The corresponding serum protein concentration was 4.6 g/dL and LDH was 204 μ/L with pleural: serum protein ratio of 0.21 and LDH ratio of 0.22 consistent with transudative pleural effusion according to Light's criteria. In addition, the pleural fluid triglyceride and cholesterols levels were 145 and 13 mg/dL, respectively, which was consistent with chylothorax. With these treatments, patient's mental status improved. He was also weaned off the supplemental oxygen and reported improvement in breathing.

Discussion

Chylothorax is a milky white pleural effusion rich in chylomicrons and triglycerides.[5] The high triglycerides concentration, usually greater than 110 mg/dL, gives its chylous appearance, but at times it can be serous or bloody, in which case diagnosis can be confirmed with protein electrophoresis for presence of chylomicrons.[6] Usually, chylothoraces are exudative (according to Light's criteria) and caused by trauma to the thoracic duct for example during thoracic surgeries, or blockage of lymphatics from tumors or mediastinal lymphadenopathy causing the leakage of chyle.[7,8] In a retrospective review of 876 consecutive thoracenteses by Agrawal et al,[9] only 22 met the criteria for chylothorax with triglycerides

Figure 2. Pleural fluid picture—milky white fluid.

$>$ 110 mg/dL and/or presence of chylomicrons. Out of these 22 chylothorax, 14 were exudative and 7 were transudative effusions. The transudative chylothoraces are found to be associated with liver cirrhosis, nephrotic syndrome, superior vena caval thrombosis and congestive heart failure.[3,10,11] Literature reviews by Diaz-Guzman et al[12] and Agrawal et al[9] have identified liver cirrhosis as the most common causes of transudative chylothorax.

The pathophysiological mechanisms for transudative chylothorax in the setting of liver cirrhosis leads back to 1962 when Dumont and Mulholland suggested that portal hypertension resulting from liver cirrhosis causes increased pressure in thoracic duct causing dilation and rupture of lymphatics and extravasation of chyle in peritoneal cavity causing chylous ascites.[13-15] The chylous ascitic fluid then translocates into the thoracic cavity through small congenital defects in the diaphragm which was suggested in 1998 by Romero et al.[16] This can be demonstrated by scintigraphy after injecting radio-opaque dye in peritoneal cavity and demonstrating its activity in thoracic cavity after 90 minutes. In addition, technique such as lymphoscintigraphy can locate the leaking lymphatics and aid in surgical interventions.[17]

Our patient did not have significant ascites, but had chylothorax from liver cirrhosis, which is a rare finding. This is because the negative intra-thoracic pressure and positive intraperitoneal pressure during inspiration phase of respiratory cycle causes the fluid to readily diffuse from peritoneal cavity into thoracic cavity through the diaphragm. When the flow of ascitic fluid into the pleural space equals the rate of production, we see clinical absence of detectable ascites.

Chylothoraces are associated with significant morbidity and mortality from malnutrition, immunosuppression, and respiratory compromise.[18,19] Treatment of chylothorax due to liver cirrhosis includes diuretics, transjugular intrahepatic portosystemic shunt (TIPS) and liver transplant.[19-21] Our patient was not a candidate for surgery due to advanced age and comorbidities. He was also not a candidate for TIPS due to hepatic encephalopathy. Conservative management of chylothorax due to liver cirrhosis includes intermittent thoracentesis for symptomatic relief and reduction in flow of chyle by diet modification with medium chain triglycerides. Medium chain triglycerides are readily absorbed in portal circulation and transported to the liver for oxidation as compared with long-chain fatty acids which are carried via chylomicrons in lymphatics.[22] Reducing the chyle flow allows the thoracic duct to heal. If diet modification is not effective, then total parenteral nutrition is used to further reduce the chyle flow.[23-26] Somatostatin analog, octreotide, reduces the gastrointestinal blood flow and indirectly reduces the lymphatic flow.[27]

Our patient was prescribed diuretics and counseled about diet modification. Indwelling pleural catheters are contraindicated in chylothorax due to risk of malnutrition and immunosuppression due to loss of fat soluble vitamins, protein and immunoglobulins.[28] Patients requiring repeated thoracentesis may require more aggressive intervention such as pleurodesis, pleurectomy, and pleural peritoneal shunting.[29]

Conclusion

1. Transudative chylothorax is a rare cause of hepatic hydrothorax and should be kept in differentials as treatment options are different and tailored according to patient needs.
2. Liver cirrhosis accounts for only 1% of chylothorax.
3. Chylothoraces are usually exudative except in conditions like congestive heart failure, liver cirrhosis and nephrotic syndrome.
4. Patients can develop pleural effusion without ascites in liver cirrhosis. When the flow of ascitic fluid into the pleural space equals the rate of production, we see clinical absence of detectable ascites.

Author's Note

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Declaration of Conflicting Interests

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Ethics Approval

Our institution does not require ethical approval for reporting individual cases or case reports.

Informed Consent

Informed consent for patient information to be published in this article was not obtained because

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