CASE REPORT

Transudative chylous pleural effusion: Case report

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KEYWORDS
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Summary
Transudative chylous pleural effusion is a very rare entity. Hereby we present a 46 year old man a case of chronic renal failure and nephrotic syndrome with chylous ascites, lower extremity edema and chylous transudative pleural effusion with slight response to ultrafiltration.
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Introduction
Chylothorax is an infrequent cause of pleural effusion that is most commonly caused by obstruction or disruption of the thoracic duct. While there are many recognized etiologies of chylothorax, malignancies and trauma related to surgical procedures account for most cases. Chylous effusions are typically exudates rich in triglycerides and chylomicrons. Nevertheless, in a small minority of patients, the chylothorax may be transudative in nature.1 In the sparse available literature regarding transudative chylothorax, the reported etiologies are few and include cirrhosis,2 amyloidosis,1 nephrotic syndrome,3 superior vena caval thrombosis,4 and congestive heart failure.5

Case report
A 48 yr old man a case of chronic renal failure was referred to department of pulmonology for management of dyspnea. The patient had been diagnosed as a case of nephrotic syndrome with ascites and lower extremity edema from 5 months ago. Patient denied cough, fever, anorexia or weight loss. Laboratory tests showed the following: Hb = 9.6 g/dL, MCV = 82 μm³, WBC = 5400/mm³, ESR = 74 mm/h, BUN = 18 mg/dL, Cr = 3.3 mg/dL, Na = 135 meq/L, K = 3.7 meq/L, P = 3.9 mg/dL, LDH = 186 U/L, AST = 11 U/L, ALT = 11 U/L, Alk-Pho = 204 U/L, Lipase = 88 U/L, Amylase = 73 U/L, Protein = 4.5 g/dL, Alb = 1.8 g/dL, Total Bilirubin = 0.3 mg/dL, Direct Bilirubin = 0.1 mg/dL, Chol = 238 mg/dL, HDL = 32, LDL = 189, TG = 84 mg/dL, ANA < 0.5, Anti Mitochondrial Antibody(IF) = Negative, Urinalysis = +4 protein, +3Glucose, 24 h urine for protein and creatinine was 10 gm and 1.2 gm respectively. CXR showed moderate right sided pleural effusion (Fig. 1).Echocardiography was normal.
A diagnostic thoracentesis had revealed milky-white fluid and hence chylothorax was suspected (Fig. 2). Milky fluid was sterile on culture and negative for malignant cells on two occasions with Protein = 1.1 g/dL, LDH = 99 U/L, Sugar = 120 mg/dL, WBC = 75 (segment = 65, Lymph = 10), TG = 132 mg/dL, Chol = 19 mg/dL, ADA = 7.5 IU/L; the effusion was transudative according to light criteria, thereby establishing transudative chylous effusion. His peritoneal fluid analysis showed LDH = 86 U/L, Chol = 25 mg/dL, protein = 0.7 g/dL, glucose = 121 mg/dL, TG = 126 mg/dL. The patient was dyspneic and in order to reduce his respiratory embarrassment, hemodialysis with ultrafiltration was recommended with slight beneficiary effect.

Discussion

"Chylothorax" is the occurrence of chylous in the pleural space, and is due to damage or blockage of the thoracic duct. The diagnosis is made by analysis of the pleural fluid, which contains high levels of triglycerides, and is confirmed by the finding of chylomicrons. Triglyceride levels greater than 110 mg/dL are highly suggestive of a chylous effusion. Cholesterol values should be measured simultaneously, since high triglyceride levels can occur in pseudochylothorax, but the cholesterol level is always very high (>200 mg/dL).

The causes of chylothorax may be divided into four major categories: tumor, trauma, idiopathic, and miscellaneous. Although chyle is reported to have protein concentrations in the transudative range, chylous effusions are typically exudative by standard criteria.

Transudative chylothorax is a very rare entity. Diaz-Guzman et al. in a literature review had reported only 13 patients with transudative chylothorax. The etiologies were cirrhosis, heart failure, systemic amyloidosis, superior vena cava obstruction and nephrotic syndrome.

The conventional notion that chylothorax develops when the thoracic duct is obstructed fails to explain events in transudative chylothorax. Translocation of chylous ascitic fluid across the diaphragm is the likely cause of chylothorax in the nephrotic syndrome and in cirrhosis. In our case the same laboratory characteristics of ascites and pleural fluid supports the above mentioned hypothesis.

Conflict of interest statement

The authors have no conflict of interest to declare.

References