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Idiopathic Bilateral Chylothorax Presenting as a Left-Sided Neck Swelling

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Chylothorax is a relatively rare condition characterized by pleural fluid of a milky appearance and elevated concentration of triglycerides and chylomicrons. Bilateral chylothorax is even more unusual, with the majority of cases resulting from trauma or in association with a neoplasm. We describe a case of bilateral spontaneous chylothorax which presented as a sudden neck swelling in a 53-year-old woman after she sneezed. The patient remains well with a normal x-ray and CAT scan of the chest and abdomen at 18-month follow-up.

Case Report

A 53-year-old woman was admitted to Henry Ford Hospital for evaluation of dyspnea and left-sided pleuritic chest pain associated with a nonproductive cough. Twenty-four hours prior to admission, she sneezed while climbing into the bathtub and noted an abrupt painful swelling of her left cervical area extending to the left shoulder. Although the swelling resolved within 12 hours, she experienced shortness of breath upon awakening the next morning. The patient was evaluated in the Emergency Room where a chest x-ray demonstrated bilateral pleural effusions (Fig 1). There was no history of trauma, recent infection, fever, or weight loss. Her past medical history included hypertension for five years, which was being treated with 500 mg of methyldopa two times a day, and a four-year history of arthralgias of the back and ankles.

Physical examination was unremarkable except for dullness to percussion and decreased breath sounds over both lung bases. Laboratory studies included a hematocrit of 45.9%, hemoglobin of 15.3 g%, and a WBC count of 5,700/mm³ with a normal differential cell count. Laboratory abnormalities included an albumin of 3.9 g/dL, cholesterol of 293 mg/dL, and serum triglyceride of 114 mg/dL. Arterial blood gases on room air showed a pH of 7.42, a PaO₂ 69 with a saturation of 94%, a PaCO₂ of 43, and bicarbonate of 28. ANA was negative. Bilateral thoracenteses yielded a creamy-appearing fluid. Fluid analysis showed a protein of 4.3 g/dL; glucose 133 mg/dL; LDH 156 IU/L; and a WBC count of 500/mm³ with 9 polymorphonuclear leukocytes, 45 lymphocytes, 8 mononuclear cells, 20 macrophages, and 17 mesothelial cells. Cholesterol content was 131 mg/dL, and triglyceride was 2,360 mg/dL. Lipoprotein electrophoresis revealed alpha fraction of 6.9%, pre beta fraction 68%, beta fraction 18.3%, and chylomicrons 6.8%. Pleural fluid cytology and acid fast bacillus smears were negative. There was no growth of bacteria, fungi, or acid fast bacilli. Sudan IV dye staining of the fluid was 4+ for fat. The patient’s PPD skin test was negative with positive mumps and candida controls. A computed tomography (CT) scan of the chest and abdomen failed to demonstrate any pathology, except for bilateral effusion. The effusions had completely resolved within one month (Fig 2). Treatment consisted of bed rest and medium-grain triglyceride diet. At 18 months, the patient remained asymptomatic; a repeat CT scan of the chest and abdomen remained normal and chest x-ray was unremarkable (Fig 3).

Discussion

Most ingested fat remains unsplit and enters the bloodstream by the thoracic duct in the form of chylomicrons. Fat enters the intestinal lacteal vessels and is then transported to the cisterna chyli, which is a lymphatic structure located on the body of the second lumbar vertebra. The thoracic duct leaves the cisterna, passes through the esophageal hiatus of the diaphragm, ascends extrapleurally into the posterior mediastinum on the anterior surface of the vertebral column on the right, and lies between the azygos vein and descending aorta in close proximity to the esophagus and pericardium. At the level of the fourth to sixth thoracic vertebrae, the duct crosses to the left of the vertebral column and continues cephalad to the superior mediastinum between the aortic arch and subclavian artery along the left side of the esophagus. At about the level of the seventh cervical vertebra, it arches laterally and then downward and passes anteriorly to the subclavian artery, vertebral artery, and thyroid.

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Fig 2—Resolution of bilateral pleural effusions at one month.

cervical trunk and empties into the venous system near the angle of the internal jugular and the subclavian veins. Anatomic variations exist in all portions of the thoracic duct (1,2). About 1,500 to 2,400 mL of chyle empty into the venous system daily. Injury to the thoracic duct below the T5 level results in a right-sided chylothorax, while injury to the thoracic duct above the T5 level leads to left-sided chylous effusions (2). Since the thoracic duct usually crosses from the right to the left side of the thorax at the T4-T6 level, injury at this point might be expected to result in bilateral effusions.

The leading cause of chylothorax is tumor, which is in the lymphoma group about 75% of the time (3). Chylothorax may be the presenting symptom of lymphoma (3-5). The second leading cause of chylothorax is trauma, usually involving a cardiovascular procedure. In a review by Maloney and Spencer (6), the incidence of chylothorax following cardiovascular surgery was 0.5%, whereas Bower (1) reported an incidence of 0.24%. Penetrating wounds to the chest or neck as well as blunt trauma to the thoracic duct may lead to a chylothorax. The third category of chylothorax is idiopathic, including cases of congenital chylothorax. A fourth category of miscellaneous causes includes filariasis (7), lymph node enlargement (8), cirrhosis (9), heart failure (10), and pulmonary lymphangiomyomatosis (11,12). Meade (13) described five cases of spontaneous unilateral chylothorax, which he believed were due to hyperextension of the spine with rupture of an inherently weakened thoracic duct. In 1975, Reilly and Tsou (14) reported a case of bilateral chylothorax apparently resulting from stretching and yawning and assumed that a disruption of the thoracic duct was created between the fourth and sixth thoracic vertebrae by hyperextension of the spine. Similarly, Gullane and Marsh (15) described a case of bilateral spontaneous chylothorax presenting as an anterior neck mass after swimming and assumed the mechanism to be the same as Reilly and Tsou had reported. Our case of bilateral spontaneous chylothorax initially presented as a left-sided neck swelling which occurred in the absence of any demonstrable pathology, trauma, or structural abnormality. We presume that the sudden hyperextension of the spine when the patient sneezed while climbing into the bathtub may have resulted in injury to a weakened portion of the thoracic duct with leakage of chyle into both pleural spaces.

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References