He complained of progressive breathlessness on exertion for a period of one month. He gave no history of cough, expectoration, fever, wheeze or hemoptysis. He did not give any history of trauma.

He was working as a casual laborer in the agriculture sector. He consumed alcohol on at least five days in a week and smoked at least two packets of beedis in a week.

Nine months back he was admitted to a hospital for treatment of acute abdominal pain. A diagnosis of acute on chronic calcified pancreatitis was made. The most probable cause for the chronic pancreatitis was due to excessive alcohol intake by the patient. A pancreatoduodenostomy was performed on him. He remained asymptomatic for a period of three months. A follow-up ultrasound done after three months showed multiple calcification of the head of the pancreas with bulky tail, and minimal ascites and one old calcified granuloma in the right lobe of the liver. He was put on a low-fat diet, small frequent feeds and received pancreatic enzyme supplements from the time he was operated upon.

On examination he was thin built and emaciated and was systemically well. There were no palpable lymphadenopathies. He had bilateral pitting pedal edema. On chest examination there was diminished movement of the right lower hemithorax when compared to the left. Percussion of the right hemithorax yielded a dull note in the right infraaxillary and infrascapular areas which persisted on deep inspiration. On auscultation diminished breath sounds in the right basal regions were apparent.

**INTRODUCTION**

Malignancy and trauma are the leading causes of chylothorax. It is more frequent after cardiothoracic and, head and neck surgeries in which the left subclavian artery is mobilized. Penetrating trauma and sudden extension of spine are other causes. Of all cases of malignancy-associated chylothorax 75% cases are due to lymphoma. The other minor causes are sarcoidosis, thrombosis of the superior vena cava, congenital chylothorax, hypothyroidism, etc., which constitute a small number of cases. In India infectious diseases like tuberculosis[1] and filariasis[2] have to be considered as differential diagnoses. We report a case of chylothorax of probable tuberculous origin. Repeated thoracocentesis and antituberculous therapy were adequate treatment.

**CASE REPORT**

A 42-year-old male presented with pain in the epigastrium, radiating to the back and feeling of fullness of the abdomen for a duration of six months. He gave history of loss of appetite and weight loss of 5 kg over a period of six months.

He complained of progressive breathlessness on exertion for a period of one month. He gave no history of cough, expectoration, fever, wheeze or hemoptysis. He did not give any history of trauma.

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On palpation his abdomen was distended, epigastric tenderness was present and bowel sounds were heard. Examinations of other systems were unremarkable.

His hemogram revealed low hemoglobin of 9.2 mg/dl and elevated erythrocyte sedimentation rate of 32 mm in half hour. Renal and liver function tests were within normal limits. Sputum smear examination for acid-fast bacilli was negative in the two samples obtained. The result of the Mantoux test done with 5 TU and read after 48 h was 0 mm. Peripheral smear examination for microfilaria was negative. Chest skiagram revealed apparent elevation of right hemidiaphragm with lateral peaking, a heterogeneous opacity in the right lower zone and loss of lung volume on the right hemithorax. A lateral decubitus view revealed tracking of fluid.

Ultrasonogram of chest was done and pleurocentesis was performed at the site marked for pleural fluid aspiration. The pleural fluid aspirated was milky white in color and odorless [Figure 1 and Table 1].

With pleural fluid triglyceride >110 mg/dl and pleural fluid to serum cholesterol ratio less than 1 a diagnosis of chylothorax was made. Pleural fluid cytology was negative for malignant cells. Ziehl-Neelsen staining of pleural fluid smear was 3 plus positive for AFBs [Figure 2]. Computed tomography (CT) scan of the chest showed right-sided hydrothorax with partial collapse of the right lower lobe and fluid in minor fissure [Figure 3]. CT scan of abdomen was suggestive of acute on chronic pancreatitis with ascitis. Ascitic fluid was so minimal that ascitic fluid aspiration could not be done. It did not reveal any evidence of mass lesion or enlarged lymph nodes.

The patient was started on antituberculous treatment with a four-drug regimen with isoniazid, pyrazinamide, rifampicin and ethambutol. He did not give consent for intercostal drainage or any surgical or interventional procedure, hence pleural aspiration was done in a staggered manner in three sittings with 500 ml being aspirated in each sitting. As octreotide was not available in our institution he was put on a low-fat diet, intravenous fluids and monitored. The patient's general condition improved and he was discharged from the hospital after three weeks.

**DISCUSSION**

Chyle is a milky, opalescent fluid that contains chylomicrons, triglycerides and lymphocytes, it is bacteriostatic and non-irritating and has little propensity to form fibrothorax. It is absorbed from the intestines and transported via the cisterna chyli and thoracic duct to the venous system; 1.5-2.5 L of chyle normally empties into the venous system daily. So in chylothorax it reaccumulates rapidly.

### Table 1: Pleural fluid analysis

|                | Pleural fluid | Serum  |
|----------------|---------------|--------|
| Cholesterol    | 34 mg/dl      | 84 mg/dl|
| Triglycerides  | 678 mg/dl     | 117 mg/dl|
| LDL cholesterol| 35 mg/dl      | 47 mg/dl|
| HDL cholesterol|              |        |
| LDH            | 56 units/liter|        |
| Amylase        | 25 units/liter|        |
| Protein        | 1.5 g/dl      | 4 g/dl  |
| Glucose        | 125 mg/dl     | 70 mg/dl|

LDL: Low density lipoprotein, HDL: High density lipoprotein, LDH: Lactate dehydrogenase
following drainage. The long intrathoracic course of the thoracic duct makes it prone for injury. The symptoms of chylothorax are almost exclusively related to the volume of fluid in the thoracic cavity. Fever and chest pain are virtually absent. Not all chylous fluids have a classic milky appearance. Indeed, almost half are either bloody or turbid in appearance. The primary cell in chyle is the small lymphocyte, and counts range from 400–6,800/mm³; hence prolonged drainage of a chylothorax can result in profound T-lymphocyte depletion.

Pleural fluid which is milky in appearance should be distinguished from pseudochylothorax and empyema. When pleural fluid is subjected to centrifugation, if the supernatant clears, the white color is due to large numbers of white blood cells, and it is probably an empyema. If the turbidity still persists, then on adding ethyl ether to pleural fluid, if turbidity clears, it is probably a pseudochylothorax caused by accumulation of cholesterol and lecithin globulin complexes in longstanding pleural effusions.

The presence of chylomicrons in the fluid as demonstrated by lipoprotein electrophoresis is generally considered the criterion standard in the diagnosis of chylothorax. Pleural effusions with a triglyceride level greater than 110 mg/dL are proved to be chylous. Conversely, a triglyceride level less than 50 mg/dL for a pleural effusion virtually excludes the diagnosis of chylothorax.

More than 50% of nontraumatic cases of chylothorax are related to tumor invading the thoracic lymph duct; lymphoma being responsible for 75% of the malignancy-associated occurrences of chylothorax. Trauma is the second leading cause of chylothorax, responsible for 25% of cases. Surgery is the most common cause of traumatic chylothorax, especially in operations that mobilize the left subclavian artery. Chylothorax may also be a result of left subclavian lines complicated by clot that obstructs the thoracic duct ostium. Penetrating trauma to the chest, such as gunshot or knife wounds, occasionally severs the thoracic duct, but non-penetrating trauma can also produce chylothorax. Falls, motor vehicle accidents, and compressive injuries to the trunk and abdomen are common causes of chylothorax. Everyday stresses such as coughing, sneezing, vomiting, and lifting heavy objects may produce a chylothorax. Approximately 25% of chylothoraxes have no identifiable cause; they are presumed to be secondary to minor trauma. Pulmonary lymphangiomyomatosis, which is a rare interstitial parenchymal disease, has been associated with chylothorax.

In this particular case the causes for chylothorax considered were trauma to the thoracic duct during abdominal surgery, malignancy, filariasis and tuberculosis. After laboratory and radiological investigations we found no evidence of malignancy or filariasis. A senior gastroenterologist opined that a thoracic duct trauma is unlikely to present as a chylothorax after a time lag of nine months. When AFB was demonstrated by Ziehl-Neelsen smear it was concluded that tuberculosis may be the most probable etiology. It is our hypothesis that the thoracic duct may be directly involved by *Mycobacterium tuberculosis*, though no conclusive proof could be provided for the same.

Conservative modalities for the management of chylothorax include treatment of cause, intercostal drainage, somatostatin, dietary changes with medium-chain triglycerides and parenteral hyper-alimentation. If the effusion does not resolve in two weeks or there is a reaccumulation of more than 1 L of fluid per day, a surgical modality like thoracic duct ligation or percutaneous embolization or pleuroperitoneal shunt is adopted. If patient is unfit for surgery, pleurodesis may be attempted.

In conclusion this reported case is a rare instance where AFB was demonstrated in a chylous pleural effusion in a patient without any evidence of tuberculous parenchymal lung involvement. It also shows that repeated thoracentesis or intercostal drainage is needed to alleviate the dyspnea caused by recurrent large-volume chylosal pleural effusions.

The patient was followed up after completion of six months of antituberculous treatment. Chest skiagram was repeated and it showed no evidence of pleural effusion. The patient was asymptomatic and had gained three kilograms of weight.

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