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CASE STUDY

Chylothorax complicating a thoracic aortic aneurysm

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Chylothorax complicating a thoracic aortic aneurysm. V. Gil-Suay, E. Martínez-Moragón, A. de Diego, J. Sanchis. ©ERS Journals Ltd 1997.

ABSTRACT: Chylothorax is an uncommon condition, usually associated with advanced malignant disease or trauma to the thoracic duct. Idiopathic chylothorax and other diverse causes have been anecdotally reported.

We describe a rare case of chylothorax, developing as a result of damage to the thoracic duct by a thoracic aortic aneurysm. Tetracycline pleurodesis was successful in controlling the chylous effusion.

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In the medical literature, it is recognized that an aortic aneurysm may compress or erode the thoracic duct, thus allowing chyle to enter the pleural space [1–4]. However, in a MEDLINE search from 1966 to 1996, no case reports of chylothorax caused by aneurysm were found. We describe the case of a patient in whom chylothorax was the presenting manifestation of thoracic aortic aneurysm. Conservative management followed by drainage and tetracycline pleurodesis led to a favourable outcome of chylous effusion.

Case report

An 81 year old woman was admitted to the hospital with a 7 day history of increasing dyspnoea, dry cough and abdominal pain. She had no haemoptysis, fever or weight loss. The patient had never smoked and denied previous diagnosis of pulmonary disease. There was a past medical history of essential hypertension, with double aortic valve disease and poor left ventricular function. The patient was maintained on digoxin and diltiazem. Two months before admission, a chest radiograph showed only enlarged cardiac silhouette and chronic diffuse interstitial oedema.

The patient was cyanotic and breathless at rest. The cardiac frequency was 78 beats·min^{-1} and the respiratory frequency was 32 breaths·min^{-1}. Physical examination revealed jugular venous distention and hepatomegaly. Peripheral pulses were intact. No lymphadenopathy or peripheral oedema was found. Diastolic murmur of aortic regurgitation was heard. There was dullness at the base of the right lung, and breath sounds were diminished over the lower half of the right lung.

Routine blood cell counts, coagulation and serum biochemistry studies were normal. Arterial blood gas data showed a pH of 7.42, an arterial carbon dioxide tension (P_{a,CO_2}) of 5.8 kPa (44 mmHg), and an arterial oxygen tension (P_{a,O_2}) of 7.5 kPa (56 mmHg). Chest radiography revealed: a widened aortic arch, with widening and tortuosity of the descending thoracic aorta; cardiomegaly, with evidence of enlarged mediastinum; and a right pleural effusion (fig. 1).

A thoracocentesis with pleural biopsy was performed. Pleural fluid was milky in appearance, with a pH of 7.38, total protein level of 4.3 g·L^{-1}, cholesterol concentration of 1,100 mg·mL^{-1} (2.84 mmol·L^{-1}), and triglyceride concentration of 3.18 g·L^{-1} (3.59 mmol·L^{-1}), serum triglyceride 0.79 mmol·L^{-1}. Cholesterol/triglyceride ratio was 0.34. A white blood cell count revealed 4×10^9 cells·L^{-1} (73% lymphocytes, 20% eosinophils and 7% neutrophils). Bacterial, fungal, and acid-fast bacillus smears and cultures were negative, as was cytology, which demonstrated only lymphocytes and mesothelial cells. A pleural biopsy specimen revealed nonspecific pleural inflammation. A tuberculin skin test purified protein derivative (PPD)-RT 23, 2 UI) was negative.

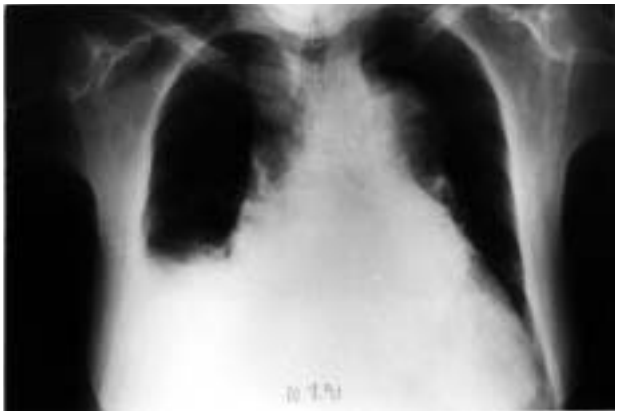
Thoracostomy tube drainage and substitution of dietary fat with medium-chain triglycerides were initially prescribed. During the following 7 days, a total of 1,500 mL of chyle was drained from the pleural space, and symptoms improved.

Computed tomographic (CT) scan of the chest, abdomen and pelvis at this time showed an ascending aortic aneurysm (maximum diameter 7 cm) and dissection of the descending aorta (maximum length 8 cm), with no evidence of tumour or lymphadenopathy (fig. 2). Because of the advanced age of the patient and her poor heart function, surgical correction was refused. Three weeks later, she was discharged from the hospital in a good condition.

After 3 months, the patient was readmitted because of pleuritic right-sided chest pain and increasing dyspnoea. A chest radiograph confirmed the recurrence of right-sided pleural effusion. Drainage and tetracycline pleurodesis were performed, and thereafter no chyle reaccumulated.

Seven months later, the patient returned to the hospital because of acute pulmonary oedema, and died 24 h after admission. Autopsy was not authorized.

a)



b)

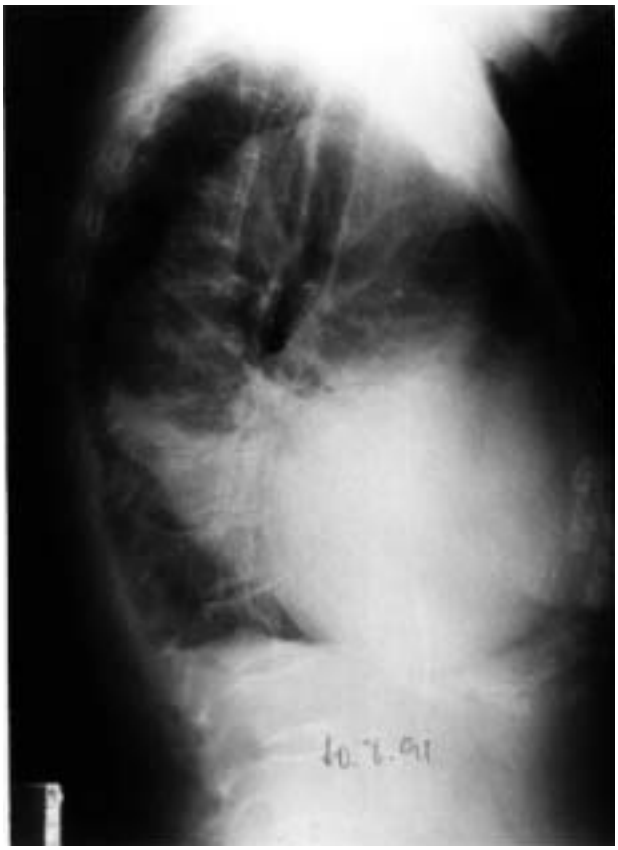


Fig. 1. – a) Posteroanterior; and b) lateral chest radiograph, demonstrating a thoracic aortic aneurysm and right pleural effusion.

Discussion

The diagnosis of chylothorax is suggested by the finding of milky, turbid, and odourless pleural fluid. However, similar characteristics are present in empyema and pseudochylothorax. Clearance of the fluid with centrifugation argues against a chylous effusion, and may represent an empyema. A cloudy supernatant is suggestive, but should be distinguished from pseudochylothorax. Pseudochylous effusion is seen in patients with long-standing pleural effusion, such as tuberculosis and rheumatoid arthritis. This pleural fluid presents high cholesterol level (>6.5 mmol·L^{-1}) and low-to-moderate triglyceride concentration [1].

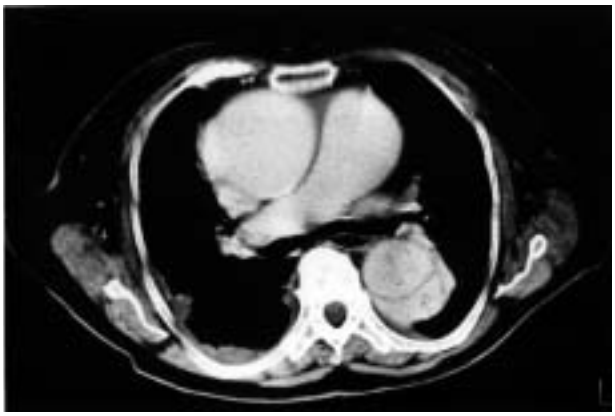


Fig. 2. – Computed tomographic scan, showing a right chylothorax (after pleural drainage) in association with an ascending aortic aneurysm and dissection of the descending aorta.

Chylous effusions are exudative, lymphocyte-predominant, and sterile. A pleural fluid triglyceride level greater than 1.1 g·L^{-1} (1.24 mmol·L^{-1}) and a cholesterol/triglyceride ratio of <1 suggest chylous effusion, whilst a triglyceride level of less than 0.5 g·L^{-1} (0.56 mmol·L^{-1}) rules it out, unless the patient has been without enteral nutrition. The demonstration of chylomicrons by lipoprotein electrophoresis establishes the definitive diagnosis of chylothorax, especially in cases with triglyceride levels of 0.56–1.24 mmol·L^{-1} [1, 4].

Chylothorax usually develops as a result of extrinsic compression, obliteration, rupture, or leaks in the thoracic duct. In the present case, the absence of malignancy or trauma and the coexistence of the aortic aneurysm and chylothorax established the latter cause.

The location of chylothorax depends on the side of the injury and the integrity of the mediastinal pleura. With an impaired mediastinal pleura, chylothorax occurs immediately after violation of the thoracic duct. Injury below the level of the fifth thoracic vertebra usually produces a right-sided chylothorax, whereas injury above that level induces a left-sided chylothorax. If the mediastinal pleura is intact, several days may elapse before chyle fills the mediastinum and ruptures into the pleural space, usually on the right side at the base of the pulmonary ligament [4]. The right-sided location in the present patient was explained by the latter mechanism.

The management of chylous effusions comprises maintenance of adequate nutrition, reduction of lymph flow into the thoracic duct, and maintenance of full expansion of the affected lung. Multiple thoracentesis or continuous tube drainage decompress the pleural space [3]. In an attempt to minimize chyle formation, initial treatment consists of bed rest and either total parenteral nutrition or a high protein diet, with dietary fat replaced by medium-chain triglycerides [5]. When these conservative measures fail, some authors advocate thoracotomy with ductal ligation [6, 7].

Pleurodesis has been used in chylothorax of various causes with favourable outcome [8, 9]. There are insufficient data to recommend one treatment modality as more effective, and, certainly, the aetiology and severity of the chylous effusion influence its treatment and outcome.

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