# A Case of Pseudochylothorax

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### **Abstract**

A 76-year-old man who had a history of both pulmonary and pleural tuberculosis, and developed pseudochylothorax is reported. It is suggested that if thoracic computed tomography is performed at the follow-up period of the patients with a significant history of pleural tuberculosis causing marked pleural

thickening, the case reports of pseudochylothoraces will increase and also its complications will be prevented and/or diagnosed earlier and treated more effectively.

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Key words: Pseudochylothorax, thoracic computed tomography, pleural tuberculosis

## Introduction

"Pseudochylothorax" or "chyliform effusion" or "cholesterol pleurisy" is a fluid which has a very high content of cholesterol and usually occurs due to long-standing of the fluid in a fibrotic pleura. Three most common causes of pseudochylothorax are tuberculous pleurisy, chronic rheumatoid pleurisy, and therapeutic pneumothorax (1,2).

# **Case Report**

A 76-year-old man was referred to our department because of tuberculous peritonitis and also an abnormal chest-x-ray. One month before admission, the patient complained of abdominal swelling and tenderness for a period over one month. His history was also significant for pleural tuberculosis which occurred 50 years ago and had been treated. Physical examination revealed ascites and he was hospitalised for evaluation of ascites by the department of general surgery. And then, laparoscopy was performed and many biopsy specimens were taken. The histopathologic examination of biopsy specimens revealed granulomatous inflammation with caseification necrosis, and thus were consistent with tuberculosis. The patient was referred to our department for treatment and follow-up.

On admission, the patient appeared well. Vital signs were within normal limits. Lung examination demonstrated strik-

Correspondence: Prof. Dr. M. Levent Erkan Ondokuz Mayıs Üniversitesi, Tıp Fakültesi, Göğüs Hastalıkları Bölümü 55139 Kurupelit, Samsun, Türkiye ing volume loss of left hemithorax with diminished breath sounds. Complete blood count and whole blood chemistry were all normal, except sedimentation rate which was 89 mm/hour. The chest-x-ray demonstrated marked volume loss and nearly total homogenous opacification of the left hemithorax, and also ipsilateral shifting of trachea and mediastinum (figure 1). Thoracic computed tomography (CT) scans revealed destruction of the left lung with grossly thickening and calcification of the ipsilateral pleural membranes enclosing a dense calcific effusion with compensatory signs of volume loss including tracheal and mediastinal shifting to the left, rib crowding and also contralateral lung herniation to the left (figure 2). When we compared the previous chest-x-rays and thoracic CT scans that were performed 10 years and 3 years ago respectively, with the new ones, we saw that there was no significant difference between them. We did thoracentesis which yielded a turbid, milky fluid without any obvious odor. Then, the fluid was centrifugated and it was detected that turbidity of the fluid persisted despite centrifugation. Lipid fraction analysis of pleural fluid was performed and the result was as follows; triglyceride: 32 mg/dl (N: <50 mg/dl), cholesterol: 421 mg/dl (N: <200 mg/dl). Direct smear of pleural fluid and bronchial lavage for acid fast bacilli and cultures for mycobacterium tuberculosis were both negative. Because of the diagnosis of peritoneal tuberculosis, antituberculous treatment consist-

Fig. 1. Postero-anterior chest x-ray demonstrate nearly total homogenous opacification and marked volume loss of the left hemithorax, and compensatory signs of it including dropping of the ipsilateral shoulder, rib crowding, ipsilateral mediastinal and tracheal shifting and contralateral lung herniation.

ing of isoniasid 300 mg/day, rifampicin 600 mg/day, pyrazinamide 2000 mg/day, and ethambutol 1500 mg/day were started.

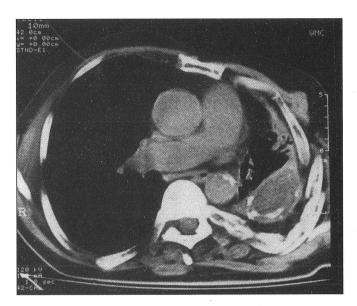


Fig. 2. Thoracic CT scans at the level of the right pulmonary artery, reveal destruction of the left lung with grossly thickening and calcification of the ipsilateral pleural membranes enclosing a dense calcific effusion with compensatory signs of volume loss.

## Discussion

Pseudochylothoraces are uncommon; only 172 cases had been reported in the literature until 1999 (3). In a series of 53 non-traumatic high lipid effusions, only 6 (11%) were chyliform pleral effusions (4).

The most common causes of pseudochylothoraces are pleural tuberculosis, chronic rheumatoid pleurisy, and therapeutic pneumothorax (1,2). The less common causes are traumatic which result in profuse bleeding into the pleural space, poorly treated empyema remnants, and any other disease states that have a potential for extensive fibrosis of the pleura, except asbestos-related pleural thickening (5).

The pathogenesis of pseudochylothorax is not known. Most patients with chyliform effusions have long-standing effusions with a thickened and calcified pleura. The diseased pleura may result in abnormally slow transport of cholesterol and also other lipids out of the pleural space and thus may lead to the accumulation of cholesterol (3).

The diagnosis of pseudochylothorax is not difficult. When thoracentesis yields a milky or turbid fluid, centrifugation of the fluid should be done. If turbidity per-

sists after centrifugation, it means that its high lipid content causes turbidity, in which only possibilities are chylothorax and pseudochylothorax, and to reach a final diagnosis clinical presentation and radiological appearance give us invaluable clues, but the definitive diagnosis is reached following the lipid fraction analysis of the pleural fluid (6). Clinically, the patients with chylothorax usually have an acute onset of chest pain and dyspnea, whereas the ones with pseudochylothorax are usually asymptomatic and have a chronic pleural effusion with a history of pleural tuberculosis or rheumatoid pleurisy. The case had a significant history of pleural tuberculosis and was asymptomatic until the development of ascites.

Radiologically, the pleural membranes are usually normal in chylothorax, but thickened and calcified in pseudochylothorax. Especially, thorax CT is invaluable and the technique of choice for revealing anatomy of the chest and for abnormalities of pseudochylothorax (5) In our case, thoracic CT scans revealed features of pseudochylothorax: First, pleural membranes were markedly thickened and calcified enclosing a dense calcific pleural fluid. Second, CT scans showed destruction of the left lung with volume loss of the ipsilateral hemithorax, and also the features of its compensation in a chronic fashion, are dropping of the ipsilateral shoulder, rib crowding, contralateral lung herniation, ipsilateral diaphragm elevation and ipsilateral shifting of mediastinum and heart. These all indicated that the process was a chronic one. Third, when we compared the CT scans performed 3 years and 10 years ago with the new ones, we noticed that there was no significant difference between them, indicating that the process was stable or at least, progressing slowly.

Clinical course is usually benign, but if the patient is symptomatic or if there has been a substantial increase in fluid size, thoracentesis should be performed to relieve dyspnea and to prevent its complications. Since reactivation of tuberculosis is always possible, cultures for tuberculosis should always be made on the fluid (7). The case was diagnosed as peritoneal tuberculosis, but the direct smear and culture of pleural fluid and also bronchial lavage were all negative for M. tuberculosis, and there was also no cavity formation or infiltration pattern in the lungs on the CT scans. Thus, we thought that the reactivation of the dormant bacilli of the peritoneum or any organ other than lungs probably caused peritoneal tuberculosis. The complications pseudochylothorax are (5); a) respiratory insufficiency, b) infections including reactivation of tuberculosis, nonspecific infections, and fungal infection, especially aspergillus (8), c) bronchopleural and/or pleurocutaneous fistulae.

We concluded that if the patients with a significant history of pleural tuberculosis causing marked pleural thickening are followed by serial thoracic CT scans, this is the best technique for evaluation of pseudochylothorax. In this way, the case reports of pseudochylothoraces will increase and also its complications will be prevented and/or diagnosed earlier and treated more effectively.

### References

- 1. Ferguson GC. Cholesterol pleural effusion in rheumatoid lung disease. Thorax 1966; 21:577-582.
- Hillerdal G. Chyliform (cholesterol) pleural effusion. Chest 1985; 88:426-428.
- Zamalloa GA, Irastorza RG, Aguayo FJ, et al. Pseudochylothorax: Report of 2 cases and review of the literature. Medicine (Baltimore) 1999; 78(3):200-7.
- Roy DH, Corr DT, Payne WS: The problem of chylothorax. Mayo Clin Proc 1967; 42:457-467.
- Hillerdal G. Chylothorax and pseudochylothorax. Eur Respir J 1997; 10:1157-1162.
- Staats BA, Ellefson RW, Budahn LL, et al. The lipoprotein profile of chylous and non-chylous pleural effusions. Mayo Clin Proc 1980; 55:700-704.
- 7. Debieuvre D, Gury JP, Jopard JM. Association pseudochylothoeax et tuberculose pleurale. Rev Pneumol Clin 1994; 50:175-177.
- 8. Hillerdal G. Pulmonary tuberculosis infection invading the pleura. Thorax 1981; 36:745-751.