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Chylothorax complicating pneumonectomy

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Chylothorax is an uncommon clinical entity caused by obstruction or rupture of the thoracic duct. Malignant disease, mostly lymphoma or metastatic carcinoma, accounts for half and surgical trauma for a quarter of all cases.[1] Occasionally, chylothorax is caused by thrombosis of the great veins, benign cysts, or lymphangiomas of the thoracic duct, tuberculosis, mediastinitis, paravertebral abscess, filariasis, or amyloidosis.

We present an unusual case of postpneumonectomy chylothorax with serious haemodynamic effects necessitating reoperation.

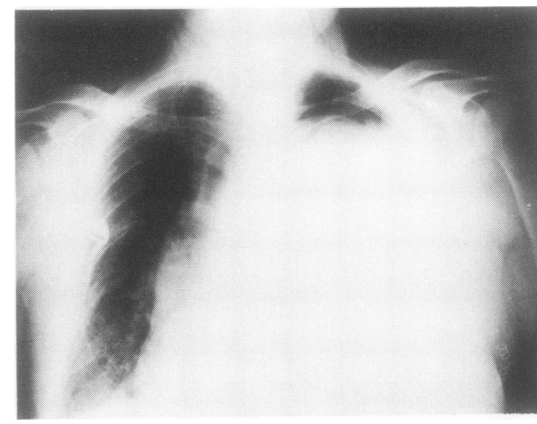
Case report

In February 1984 a 60 year old man was admitted to our hospital because of thoracic pain, weight loss, and progressive atelectasis of the left lung. On physical examination the breath sounds were absent over the left lung; there was no lymphadenopathy or hepatomegaly. The serum levels of alanine aminotransferase and γ-glutamyl transpeptidase were raised. The chest radiograph showed complete atelectasis of the left lung, deviation of the mediastinum to the left, and elevation of the left diaphragmatic dome. Fibreoptic bronchoscopy revealed obstruction of the left main bronchus by tumour, biopsy of which showed moderately differentiated squamous cell carcinoma. Computed tomography showed no enlargement of mediastinal lymph nodes. An ultrasound study of the upper abdomen revealed nothing abnormal. Because of the raised liver enzyme levels laparoscopy with liver biopsy was performed, with negative results.

At left thoracomy the mediastinum appeared to be free of tumour and a radical pneumonectomy was carried out. Biopsy specimens of hilar and subcarinal lymph nodes were all negative. The immediate postoperative course was normal.

On the fourth day after surgery the left hemithorax was almost completely filled up, with deviation of the mediastinum to the right (fig). Despite the evacuation of 600 ml of fluid from the thoracic cavity the patient developed severe dyspneoa, tachycardia, hypotension, and appreciable pulsus paradoxus. Ten hours later another 1600 ml of pink coloured chest fluid was withdrawn, with immediate relief of symptoms. A pulmonary artery catheter was inserted, which showed over the next 24 hours a progressive increase of the pulmonary capillary wedge pressure from 0 to 22 mm Hg and of the mean pulmonary artery pressure from 10 to 29 mm Hg. A bedside ultrasound study of the heart showed severe compression of the heart cavities, the total transverse diameter being reduced to 8 cm. After another evacuation of pinkish fluid (2400 ml) the pulsus paradoxus disappeared and the heart was restored to the midline position. The results of biochemical analysis of the fluid are shown in the table. The high triglyceride and low cholesterol content, as well as the lymphocytic differential formula of the smear, were pathognomonic for a chylous effusion. Cultures and cytological examination gave negative results.

On the 10th postoperative day the chest was reopened. Up to that time about 10 l of pleural fluid had been evacuated. Two hours before the operation butter and milk were administered through a gastric tube. Milky fluid could be seen oozing out posteriorly adjacent to the aorta at the origin of the intercostal arteries, where a pleural flap had been removed to cover the bronchial stump. An obvious chylous fistula was not seen. The thoracic duct was searched for, but could not be found. All dissected tissue between the aorta and azygos vein was sutured or clipped until no more oozing was evident. After this operation the left hemithorax filled up at the normal rate and three days later the patient left the intensive care unit.



Posteroanterior chest radiograph showing filled up left thoracic cavity and deviation of the mediastinum to the right.

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Results of the biochemical analysis of the pleural fluid and of corresponding serum values

	Pleural fluid	*Serum (blood)*
Density	1024	
Total protein (g/100 ml)	1·8	4·7
Albumin/globulin (g/100 ml)	68/32	47/53
Lactate dehydrogenase (U/l)	176	343
Amylase (SE/100 ml)	298	139
Glucose (mg/100 ml)	111	138
Erythrocytes ($\times 10^{12}$/l)	0·04	
Leucocytes ($\times 10^{9}$/l)	1·1	(17·7)
Formula (%)	0/1/0/0/99/0	(1/81/0/0/12/6)
Triglycerides (mg/100 ml)	387	129
Cholesterol (mg/100 ml)	46	105
Turbidity	++	0

Conversion: traditional to SI units—Glucose: 1 mg/100 ml = 0·0555 mmol/l; triglycerides: 1 mg/100 ml = 0·0113 mmol/l; cholesterol: 1 mg/100 ml = 0·0259 mmol/100 ml.

Discussion

Chylothorax rarely complicates thoracic surgery, occurring mostly after cardiovascular and oesophageal procedures. Its incidence in cardiovascular operations is 0·2–0·5%.[2] It is even more exceptional in pleuropulmonary surgery and this is the first case we have encountered in 15 years, during which about 700 thoracic operations, mainly pneumonectomies and lobectomies, have been performed.

The chylothorax presented as an unusually fast filling up of the empty thoracic cavity, eventually leading to cardiovascular collapse due to cardiac tamponade. The filling pressure of the left ventricle rose to 22 mm Hg to overcome the transmural pressure difference. This is readily explained as the thoracic duct pressure at the height of flow is 10–28 cm H_2O, while the flow rate may be up to 110 ml per hour, which amounts to 2500 ml of chyle in 24 hours.[3]

The macroscopic aspect of the pleural fluid in postoperative chylothorax may be misleading because it is blood stained and has a low fat content owing to fasting. The diagnosis is confirmed by demonstrating that the fluid has a much higher concentration of triglycerides and a lower concentration of cholesterol than the serum.[4] In chyle most leucocytes are lymphocytes and pancreatic exocrine enzymes are usually present at a higher titre than in serum. Fat globules can be stained with Sudan III.

The "normal" anatomy of the thoracic duct is present in only half of the population and two or more main ducts can be found at some stage in 50% of people.[3] The main duct usually crosses the midline at the level of the fifth thoracic vertebra, so that section or perforation below this level produces a right sided and above this level a left sided chylothorax. The main duct enters the left subclavian vein near its junction with the internal jugular vein. Many other minor lymphaticovenous anastomoses exist, however, between the thoracic duct system and the azygos, intercostal, and lumbar veins. This explains why the main duct can be safely ligated at any point.[3]

Most authors advise conservative treatment of chylothorax for one or two weeks with repeated thoracocenteses or tube drainage. Meanwhile adequate nutrition has to be maintained by intravenous hyperalimentation. If the fistula does not close spontaneously surgery is indicated, even in non-traumic chylothorax and malignant disease. Supradiaphragmatic ligation of the duct by a mass ligature techique encircling all tissue between the azygos vein and the aorta is an effective method, avoiding difficult identification of the fistula site or dissection of the duct.[5]

Our case is rather exceptional because of the dramatic cardiovascular collapse due to cardiac compression by the accumulated chyle. Surgical intervention was necessary and the preoperative diagnosis was based on the characteristic results of the analysis of the chest fluid.

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