

CHYLOTHORAX DUE TO FILARIASIS — A CASE REPORT

K L Goh
H W Tan
T G Loh
S Yap

**Departments of Medicine and Surgery
Faculty of Medicine
University of Malaya
Kuala Lumpur
Malaysia**

K L Goh, MBBS, MRCP (UK)
Lecturer in Surgery

H W Tan, MBBS, MRCP (UK)
Lecturer in Medicine

T G Loh, MBBS, MRCP (UK)
Professor of Medicine

S Yap, MBBS, FRCS (Edin, Glas)
Lecturer in Medicine

SYNOPSIS

A case of chylothorax due to filariasis is reported. The causes and management of chylothorax in general are discussed and the pathogenesis of chylothorax due to filariasis is described.

INTRODUCTION

Chylothorax, the accumulation of chyle in the pleural cavity is an unusual problem. It is believed to be first described by Bartolet (1633) (1) and the first case of traumatic chylothorax reported by Quincke (1875) (1). Since then many cases of chylothorax due to various causes have been reported.

However, case reports of chylothorax due to filariasis (2) have been very few. We report such a case below.

CASE REPORT

M.H. is a 34 year old Malay man who complained of progressive breathlessness for one month. There was no accompanying complaints of cough, fever nor loss of weight.

He had been well previously except for insidious development of swelling and induration of both lower limbs over the past twenty years; which was clinically diagnosed as elephantiasis following a filarial infection.

On admission, he was mildly dyspnoeic but otherwise generally well. Examination of the respiratory system revealed a massive right pleural effusion. There were no other chest signs; the cardio-

A chest x-ray done is as shown in Fig. 2. Thoracentesis revealed a milky (Fig. 3), non-putrid pleural fluid. Sudan III (Fig. 4) staining demonstrated numerous fat globules and chemical analysis showed a cholesterol level of 2.1 mmol/L and a triglyceride level of 23.0 mmol/L; confirming that it was chyle.

No bacteria could be cultured from the fluid and stains for acid-fast bacilli were negative. No malignant cells were found on cytological examination. Other laboratory tests including the full blood count and ESR were normal and blood film examination for

microfilaria on several occasions were negative.

A chest tube was inserted to drain the fluid and a low-fat diet prescribed. However, chyle continued to drain in significant amounts and food intake was stopped and patient hyperalimmented. Pleural drainage of chyle slowly decreased over the subsequent three weeks and the chest tube removed after surgical talc was instilled for pleurodesis.

A repeat chest x-ray (Fig. 5) showed a fully expanded right lung with no underlying lung nor mediastinal shadows.

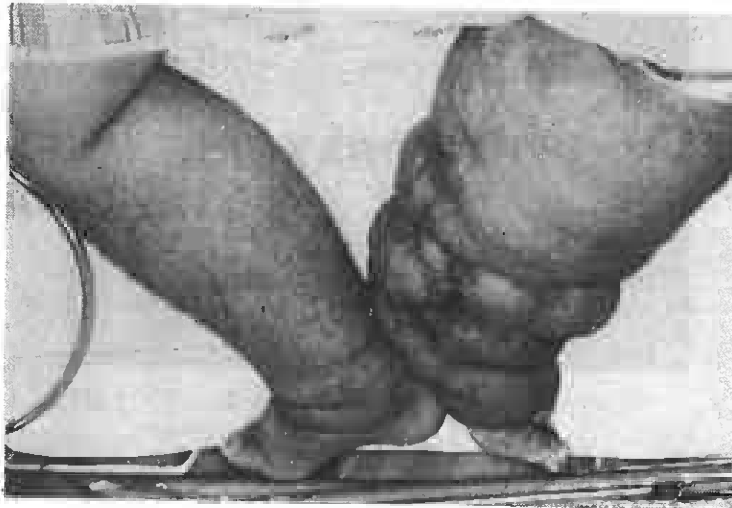


Figure 1 Photograph of patient's leg. Elephantiasis of both lower limbs.



Figure 2 Chest x-ray of patient of admission.

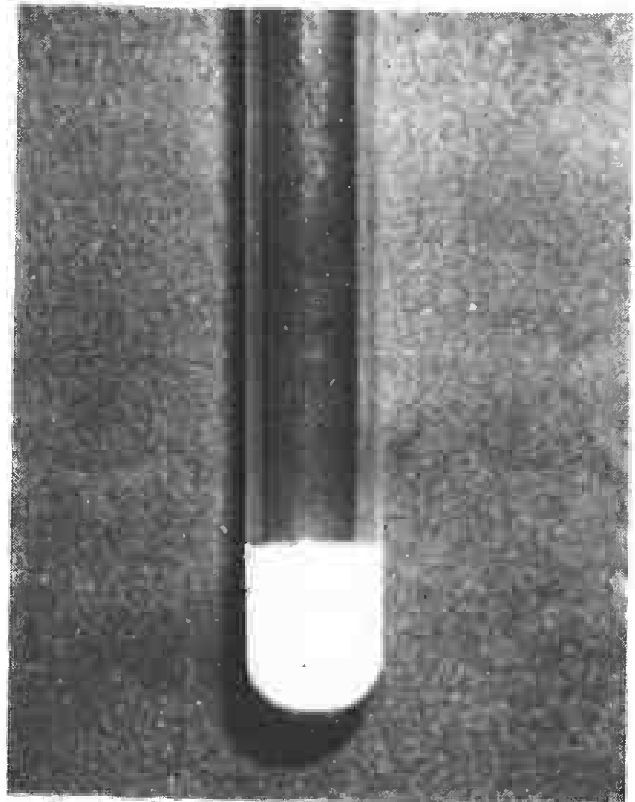


Figure 3 Chyle in a test-tube.

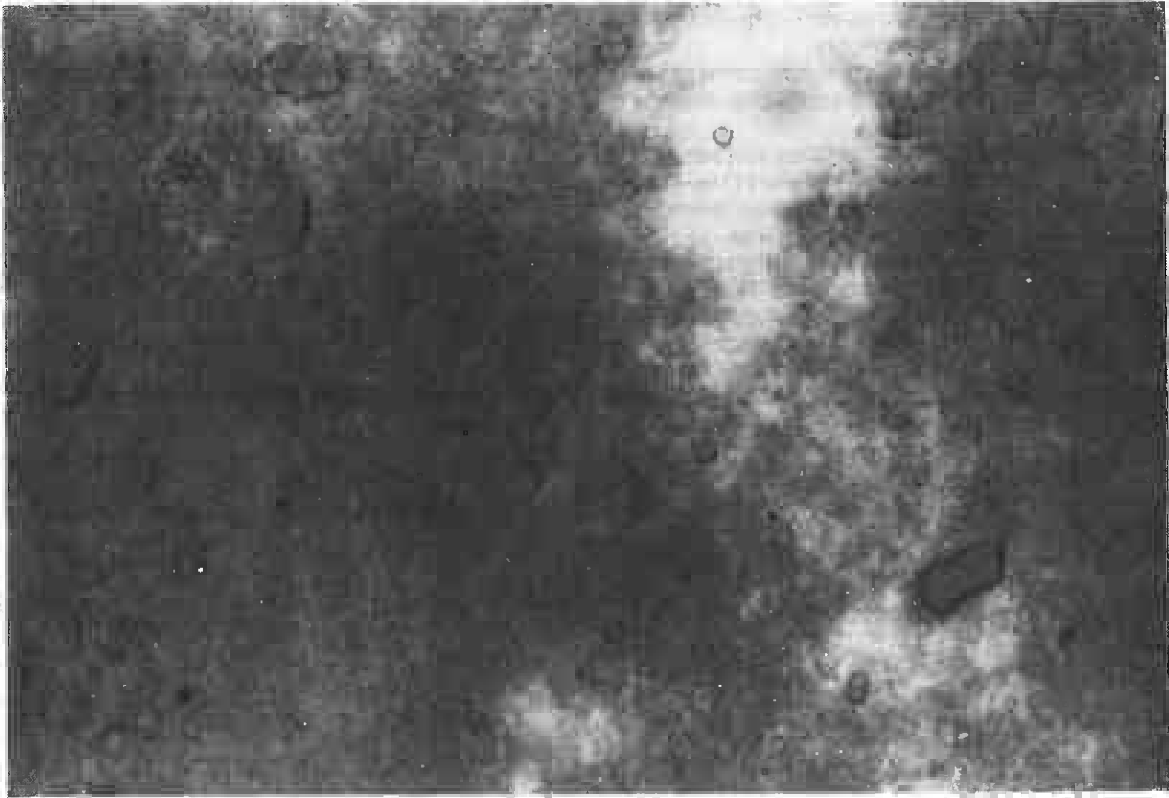


Figure 4 Microscopic picture. Fat-globules stained with Sudan III.

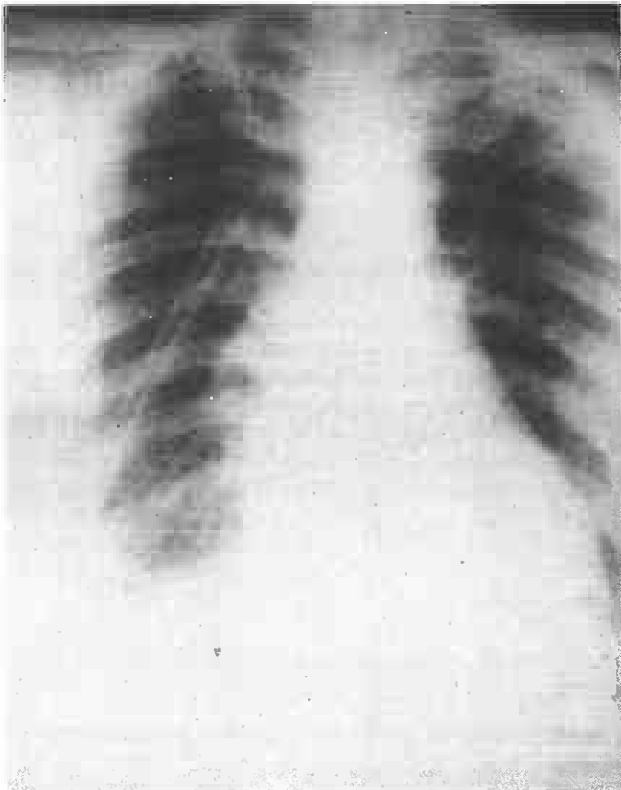


Figure 5 Chest x-ray — after drainage of the Chylothorax.

DISCUSSION

Chylothorax results from obstruction or disruption of the thoracic duct or intrathoracic lymphatic channels.

Surgical damage to the thoracic duct accounts for a quarter of all cases of chylothorax. (3) Intrathorax surgery not directly involving the thoracic duct have also been known to cause chylothorax. (3)

Penetrating injuries to the thoracic duct may be caused by gunshot or stab wounds, esophageal dilation and even translumbar aortography. Non-penetrating injuries may follow sudden hyperextension of the spine, fracture of vertebrae or ribs, blast and crush injuries. (3)

While these are important causes, reflux of chyle into the pleural cavity caused by obstruction of intrathoracic lymphatic channels by malignant disease accounts for more than half the cases. It has been reported in association with lymphoma, sarcoma, carcinoma, seminoma, teratoma and neuroblastoma. (3) Other causes of lymphatic obstruction are relatively rare and they include thrombosis of the superior vena cava, tuberculosis, mediastinitis, paravertebral abscesses and filariasis.

Filariasis as a cause of chylothorax is certainly uncommon and is perhaps a more common cause of chyluria. (2) In filariasis, (4) the living worms usually cause a diffuse endolymphangitis with lymphocytic and eosinophilic infiltration and fibroblastic proliferation. The more serious damage however occurs upon death of the parasite when a granulomatous reaction is provoked. The walls of lymph vessels swell from oedema and eosinophilic infiltration. Central foci of necrosis follows and finally this is replaced by fibrous tissue. When the number of worms is large, lymphatic obstruction may result when this occurs in the pelvic

and retroperitoneal lymphatic system, chyluria results and when in the thorax, chylothorax.

At this stage, microfilaria is seldom demonstrated in the peripheral blood film and antifilarial therapy of no benefit.

In our patient, the obvious features of past filarial infection, the absence of a history of chest trauma and the exclusion of an inthoracic malignancy leaves little doubt as to the aetiology of the chylothorax.

Management of chylothorax is a difficult problem while one may be able to locate the site of chylous effusion in some traumatic or post-surgical cases, this is not so when the cause is non-traumatic and pathology diffuse. Surgical repair is then not feasible.

Many of these cases respond to continuous pleural drainage with fatty-diet restriction. In others as with our patient, oral intake has to be stopped and the patient hyperalimeted instead. Persistent or recurrent chylothorax may be managed sometimes by pleurodesis after complete drainage of the chyle.

When all these measures fail, trans-thoracic or low thoracic duct ligation at the level of the diaphragm have been shown to be successful. (5)

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