Case Report

Recurrent pulmonary embolism - Is filarial lymphedema a risk factor for deep vein thrombosis?

Yang Liang Boo, Hong Tak Lim, Pek Woon Chin, Suat Yee Lim, Fan Kee Hoo

Hospital Ehche' Besar Hajjah Kalsom, Jalan Kota Tinggi and Universiti Putra Malaysia, Serdang, Selangor, Malaysia

Lymphatic filariasis is caused by nematodes that inhabit the lymphatic system and subcutaneous tissues. The presence of filarial lymphedema or elephantiasis leads to disfigurement and disability. However, its relationship with deep vein thrombosis and pulmonary embolism was not well-documented. Here, we described a case of recurrent pulmonary embolism associated with left leg elephantiasis. (Rawal Med J 201;41:259-261).

Keywords: Filariasis, pulmonary embolism, deep vein thrombosis

Lymphatic filariasis is caused by nematodes that inhabit the lymphatic system and subcutaneous tissues. *Wuchereria bancrofti*, *Brugia malayi*, and *Brugia timori* are three main species that lead to lymphatic filariasis.[1] 90% of the infections are caused by *Wuchereria bancrofti* while most of the remainder by *Brugia malayi.*[1] World Health Organization (WHO) had estimated a total of 120 million people in tropical and subtropical areas of the world were infected with lymphatic filariasis and almost 15 million (mostly women) had elephantiasis of the leg. Approximately 66% of those at risk of infection lived in the WHO South-East Asia Region and 33% in the African Region.[2] The presence of filarial lymphedema or elephantiasis leads to disfigurement and disability.[3] However, its relationship with deep vein thrombosis (DVT) and pulmonary embolism was not well-documented. Hajdu et al previously reported a case of massive pulmonary embolism in association with bilateral elephantiasis and attributed the possible underlying mechanism as a result of reduced mobility and venous stasis.[4] Here, we describe a case of recurrent pulmonary embolism associated with left leg elephantiasis.

CASE PRESENTATION

A 36 year-old lady presented with sudden onset of breathlessness associated with cough, wheezing, and chest tightness. She had left leg elephantiasis at the age of 22 and was treated accordingly. She had a history of hospital admission one year ago for acute pulmonary embolism and was started on oral anticoagulant therapy (OAC). Unfortunately, she failed to follow-up. Otherwise, there was no recent surgical procedure, trauma, and travel history. She was single and denied taking any medication that was associated with increased risk of DVT. Clinically, she was obese (BMI >40kg/m^2) with left leg elephantiasis. She was tachycardic and tachypneic requiring oxygen. Blood pressure was 120/60 mmHg on admission. There was presence of left leg swelling with minimal edema and skin changes (Fig. 1).

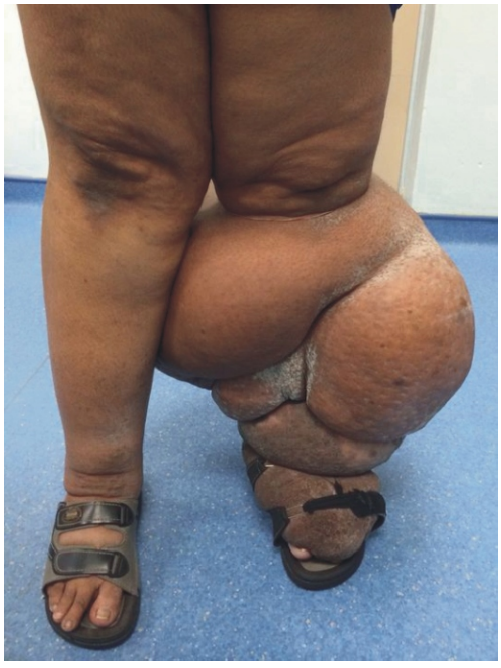
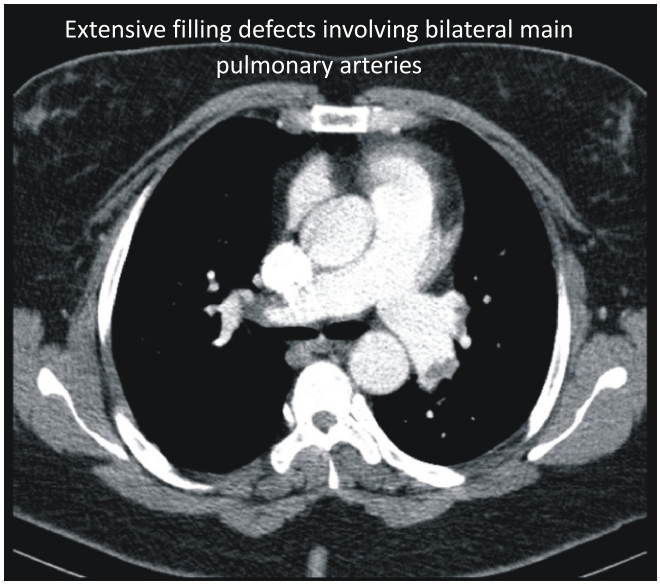


Fig. 1. Left lower limb elephantiasis

Blood investigations showed positive D-dimer and arterial blood gas showed type 1 respiratory failure. Full blood count showed hemoglobin of 12.8g/dL with leucocytosis and neutrophils predominant. Eosinophil count was normal ($0.06x10^9$/L). Liver function test, renal profile, and coagulation profile were unremarkable. Her chest radiography (CXR) showed cardiomegaly. Computed tomography pulmonary angiography (CPTA) revealed filling defect in bilateral main pulmonary arteries consistent with bilateral pulmonary embolism (Fig. 2).

Fig. 2. CTPA showed extensive filling defects involving both main pulmonary arteries at the bifurcation (arrow).



Echocardiogram showed evidence of right sided heart failure with dilated right atrium and ventricle. She was diagnosed with recurrent pulmonary embolism with left leg elephantiasis. Ultrasound lower limb was technically difficult because of patient's habitus and lymphedema, thus was not carried out. DVT clinical probability scoring for this patient was calculated and rated as high pre-test probability for DVT.

As she was hemodynamically stable, anticoagulation with low molecular weight heparin (LMWH) was started. She was supported with oxygen therapy and monitored closely for any deterioration. After a week of hospitalization, she was discharged well with OAC.

DISCUSSION

Filarial lymphedema is a chronic inflammation of the lymphatic vessels leading to swelling of the limbs. The presence of severe lymphedema or elephantiasis is debilitating and causes marked disfigurement. Disease progression will lead to severe lymphatic stasis and immobility indirectly serves as a risk factor for developing DVT of the affected limb, which may lead to pulmonary embolism.[4] In our patient, we postulated that in the presence of lower limb elephantiasis, it led to immobility, venous stasis, and increases the risk of developing DVT and subsequent pulmonary embolism. Obesity had compounded her overall risk of DVT and thromboembolism. Unfortunately, ultrasound doppler left lower limb could not be carried out in this case. Thus, confirmation of DVT could not be ascertained. Other risk factors for DVT were not present in this patient. Wells score system is a sensitive tool in assessing the probability of DVT.[5] There are a total of 9 selected criteria on clinical findings and the outcome are divided into low, moderate, and high probability. This scoring system was utilized for our patient and she was categorized into high probability.

The mainstay of treatment for pulmonary embolism is anticoagulation. Thrombolysis is indicated in patients with hemodynamic instability.[6] In recurrent pulmonary embolism, life long anticoagulation is recommended. Our patient had multiple factors that increase the overall risk in developing thromboembolism and pulmonary embolism. Thus, it was important to counsel her on the importance of anticoagulation and improved her compliance to the therapy.

In summary, the presence of severe venous stasis, obesity, and reduced mobility are risk factors that increase the risk of DVT and pulmonary embolism, especially in patient with elephantiasis. In this case, we observed recurrent pulmonary embolism complicating filarial lymphedema. Future studies are important to confirm this observation, and thus, preventing morbidity and mortality in patients with elephantiasis.

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Conception and design: YL
Collection and assembly of data: HT, PW
Analysis and interpretation of the data: SY
Drafting of the article: YL
Critical revision of the article for important intellectual content: FK
Statistical expertise: HT
Final approval and guarantor of the article: YL, FK
Corresponding author email: Fan Kee Hoo: hoofan@gmail.com
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