Human lymphatic filariasis is not a disease, but a syndrome!

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There cannot be any ‘filarial disease’ by definition, exclusively due to lymphatic filarial infection in humans. Clinical manifestations produced as a consequence of lymphatic dysfunction with filarial origin can only be termed ‘filarial syndrome’.

The International Task Force for Disease Eradication has identified lymphatic filariasis as one of the six diseases considered eradicable or potentially eradicable\(^1\). In addition, the World Health Assembly adopted a resolution supporting the global elimination of lymphatic filariasis, and a 20-yr elimination programme is now under way\(^2\). The strategy for the elimination of filariasis aims to control both transmission through community-wide (mass) chemotherapy programmes and the disease itself via individual patient management\(^3\).

Lymphatic filariasis caused by *Wuchereria bancrofti* (bancroftian filariasis), *Brugia malayi* (brugian filariasis) and *Brugia timori* (Timorian filariasis) is an important public health problem. All infections produce essentially similar clinical presentations in man, related mainly to the pathology of the lymphatic system\(^4\).

Adult filarial worms are thread-like measuring 4.0–6.5 cm in length and 0.1–2.8 mm in width (Figure 1a). They are coiled together in lymphatic vessels and glands. Eggs lie in the upper uterus. The young ones are called microfilariae (Mf; Figure 1b), and they measure 280 × 7 \(\mu\)m. Mf begin to appear in the peripheral blood 6–12 months after patent infection. They undergo three stages of development in the insect host (Culicine, Anopheline or Aedine mosquitoes) to form infective larvae, which are transmitted to the human host by the female mosquito while feeding. The infective form makes its way to the nearest lymph glands where it becomes adult in three months to one year.

When the worms are challenged either physically (exacerbation/manual strain) or therapeutically, they release immunologically provocative substances which cause sustained mechanical trauma (inflammation) of the lymphatic vessels\(^5\). Damage to the lymphatic vessels (lymphangitis) is mediated by an immune response to the substances released by the adult worms. Successful bouts of lymphangitis inevitably lead to severe fibrosis of the lymphatic vessels and formation of lymphatic hypertension and lymphoedema. Longstanding (3–7 years) lymphatic hypertension generally resulted in elephantiasis (Figure 2).

The causative agent for the original ‘lymphatic dysfunction’ could be the filarial worm. However, infection is not synonymous with disease. Indeed, in endemic areas of filariasis, there is a wide spectrum of host response to the infection. In the case of filarial infection, there are many people without any disease symptoms though they harbour parasites for years. The immune hyper-responsive persons (to parasites/parasite products) alone suffer from lymphatic dysfunction. Elephantiasis and hydrocoele are some of the later consequences of lymphatic dysfunction. They do not appear suddenly in the early stage of filariasis. Appearance of these symptoms is independent of the administration of anti-filarial drug. However, there are ample evidences at present to show that pyogenic microorganisms are responsible for acute/chronic clinical symptoms/manifestations\(^7\).

Generally, lymphoedema, elephantiasis, hydrocoele and other related manifestations are some of the consequences of lymphatic pathology, but are not always necessarily the results of filariasis. Any obstructive lesion involving a major lymphatic vessel may cause lymphostasis distal to the obstruction, which will lead to elephantiasis. Such obstructions may follow infections, acute or chronic (such as tuberculosis), tumours, surgery or irradiations\(^6\).

In the case of tropical pulmonary eosinophilia (TPE), there is a vigorous immune response directed against the microfilariae with consequent pathology. Immunological hypersensitive reactions to microfilarial antigens are probably the cause of the TPE syndrome. However, its clinical profile is not unique, and other helminths sometimes induce similar presentations\(^7\).

The role of secondary infection (mainly bacterial, e.g. streptococcal) in the production of lymphoedema has long been suspected\(^1\). Similarly, non-filarial elephantiasis has been reported in many African coun-

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**Figure 1.** *Wuchereria bancrofti.* **a.** Adult (thread-like worm); **b.** Microfilaria.

**Figure 2.** Elephantiasis of lower limb.
tries and India, mainly due to the irritant effect of mineral particles, ‘silicon’ (as aluminium silicates) absorbed through the skin of the feet while walking without shoes in certain areas of clay soils of volcanic origin. Yet another manifestation, the hydrocoele generally known to occur as a result of filarial infection in endemic areas, could also be due to non-filarial etiology.

Treatment of asymptomatic filarial parasite carriers and clinical management of patients are two different entities and need to be dealt with separately. The anti-filarial drugs (Diethylcarbamazine, Ivermectin, Albendazole, etc.) are given to individuals with microfilariae, or to the community in endemic areas mainly to eliminate the parasites and to interrupt transmission. Early diagnosis and treatment of infection carriers will prevent most of them from producing clinical expressions.

The treatment should be based on symptoms/sufferings of the individual when the manifestations are at a late stage. During this stage, live worms are less likely to be present as evidenced (with the available diagnostic tools) by many investigators, and hence the anti-filarial drugs are of little use. In other words, the reversal of lymphoedema is possible only at the early stage. Once the oedema has become persistent and turns to ‘elephantiasis’ (or ‘hydrocoele’), reversal is difficult with anti-filarial drugs. For morbidity management, simple foot hygiene and physiotherapy are advocated along with chemotherapeutic measures during early stages, and surgery at late stages, occasionally. However, this is independent of filarial infection and hence does not warrant any anti-filarial drugs.

In many endemic areas, a large proportion of the population shows varying degrees of microfilaraemia without any evidence of filarial disease and some people never develop a patent infection or show any signs of the disease. In other words, the filarial ‘infection’ carriers are not the ‘diseased’ individuals, nor does the infection in individuals definitely lead to ‘disease’ stage (with/without anti-filarial drug). This distinction is important from the case management point of view. Clinical manifestations commonly seen in the filarial endemic areas are also found in the non-endemic areas. Therefore, clinical manifestations due to filarial infection in humans may be termed more appropriately as ‘filarial syndrome’.


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