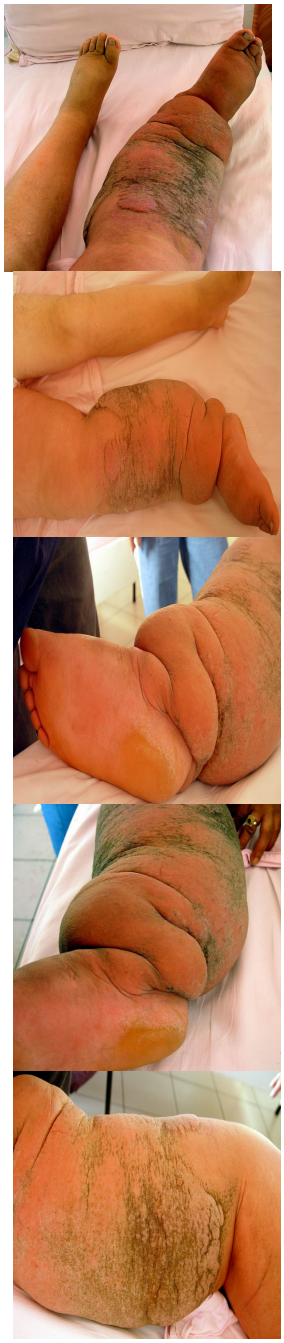

FILARIASIS

Synonyms and Diseases.

Malayan filariasis (Brugia malayi), Timor filariasis (B. timori), Bancroft filariasis, filariasis bancrofti, and wuchereriasis (Wuchereria bancrofti). Some of these parasites can cause elephantiasis and tropical pulmonary eosinophilia.

Epidemiology.

Lymphatic filariases are widely distributed in tropical and sub-tropical areas, with the largest number of infections occurring in India, South Asia, East Asia and Pacific Islands, and sub-Saharan Africa. eFig. 207-2.1 in the on-line edition shows the geographic distribution of W. bancrofti, B. malayi, and B. timori in the Southeast Asian and Western Pacific regions. W. bancrofti occurs in the northern part of South America (Guyana, Surinam, and some coastal regions of Brazil). In the United States infections are seen primarily in immigrants and persons with prolonged visits to endemic areas.

Etiology and Pathogenesis.

Lymphatic filariasis is caused by three filarial nematodes: B. malayi, B. timori, and W. bancrofti, parasites transmitted by infective mosquitoes.

Summary of Filarial Parasites

SPECIES	**GEOGRAPHIC DISTRIBUTION**	**MANIFESTATIONS**
Brugia malayi	Southeast Asia	Lymphangitis, lymphadenitis, lymphedema, hydrocele, elephantiasis, tropical pulmonary eosinophilia

B. timori

Indonesia

Same as above

Loa loa

Africa

Angioedema, pruritic migratory swellings; migration of worm across eye

Mansonella ozzardi

Central and South America

Pruritus

M. perstans

Africa, South America

Pruritus, angioedema

M. streptocerca

Africa

Pruritus, papular rash, lichenification

Onchocerca volvulus

Africa, Central and South America

Pruritic papules, nodules, lichenification; keratitis, retinitis, blindness

Wuchereria bancrofti

Tropics worldwide

Lymphangitis, lymphadenitis, lymphedema, hydroceles, chyluria, elephantiasis, tropical pulmonary eosin

[a] Dracunculiasis and dirofilariasis, which are also filarial infections, are covered in

The incubation period is 5 to 18 months, although B. malayi microfilaremia may occur as early as 2 to 3 months after exposure. W. bancrofti microfilariae first appear in peripheral blood 8 to 12 months after exposure, but symptoms can begin as early as 1 to 3 months after exposure. Adult worms live an average of 10 to 15 years; the microfilariae, probably 6 to 12 months. Symptoms and sequelae can persist after the death of all parasites.

Clinical Findings.

Lymphatic filariasis was generally thought to occur mainly as a seriously handicapping disease of adults, being sporadically observed in children. However, new highly sensitive diagnostic tests have revealed that it is first acquired in childhood, often with as many as one-third of children infected before the age of 5 years in endemic areas. Initial lymphatic damage generally remains sub-clinical for years or gives rise only to recurrent lymphangitis or non-specific presentations of adenitis/adenopathy. It takes years for the characteristic clinical features of chronic filariasis to appear. In endemic areas, the prevalence of clinical manifestations increases after age 20. The clinical course ranges from asymptomatic to severely disabling manifestations (less than 1 percent of those infected). Manifestations may be acute, chronic, or recurrent.

CUTANEOUS LESIONS

Early findings are lymphangitis with a characteristic retrograde progression, lymphadenitis, orchitis,

epididymitis, and, sometimes, fever. During the resolution of the acute phase of W. bancrofti filariasis, there may be extensive exfoliation of the skin of the affected limb. Lymphangitis is typically recurrent with 6 to 10 episodes per year, usually lasting 3 to 7 days each; the affected body part clinically appears normal between early episodes. Intermittent fevers and lymphangitis can recur for as long as 20 years, the lifetime for the adult worm, after an infected person leaves the endemic area.

After 10 to 15 years of infection, chronic disease is evident in sequelae of lymphatic obstruction including lymphedema, elephantiasis, hydrocele, and chyluria. The skin over the involved area is hypertrophic, verrucous, and fibrotic with redundant folds of skin . Fissures, ulceration, and gangrene may occur. Secondary bacterial infection is common. The anatomic locations most commonly afflicted with elephantiasis include the lower extremity, scrotum, and penis. Less commonly, the upper extremity, breast, and vulva are involved. Treatment does not reverse the late findings of scarring and lymphatic obstruction. W. bancrofti has been reported to cause a skin nodule on the breast.

Travelers to endemic areas have a hyper-responsive clinical presentation, with more intense inflammatory reactions to filarial parasites. The clinical findings may include lymphangitis, lymphadenitis, and groin pain from the associated lymphatic inflammation, urticaria, and a peripheral eosinophilia.

RELATED PHYSICAL FINDINGS.

Tropical pulmonary eosinophilia develops in some patients with filarial infections.

Laboratory Tests.

Patients with lymphatic filariasis have a peripheral eosinophilia, which may be high grade, and an elevated IgE. Persons with tropical eosinophilic syndrome have pulmonary infiltrates on chest x-ray films.

Special Tests.

Diagnosis is made by demonstrating microfilariae, preferably at night (midnight), in blood, urine, and other body fluids, and tissue. However, persons with active filarial infection may not be microfilaremic. The demonstration of the adult worm in lymphatics is possible, but biopsy of adenopathy is contraindicated. The usefulness of serologic tests has been limited by cross-reactions with other nematodes, but newer tests such as polymerase chain reaction (PCR) and antigen detection with better specificity are becoming available.

Differential Diagnosis.

The stage of the infection determines the differential diagnosis. Acute infection may resemble bacterial lymphangitis and other causes of nodular lymphangitis, such as sporotrichosis and leishmaniasis.

The differential diagnosis for the chronic stage includes the other causes of lymphedema and elephantiasis .

Treatment.

Treatment is suboptimal because drugs are only partially effective against adult worms in lymphatic ducts, the cause of the cutaneous manifestations. Of the four classic drugs with an antifilarial activity, diethylcarbamazine (DEC) and ivermectin are partially effective against adult worms, and albendazole is effective only with high dosages, which may induce severe adverse events. Recent studies document that a 6- to 8-week course of daily doxycycline can reduce the longevity or the reproductive capacity of these parasites by eliminating bacterial (Wolbachia) endosymbiots present in the lymphatic filariae. To be effective against microfilariae, treatment needs to combine doxycycline with either two doses of ivermectin, one at the end of the doxycycline course and the other 3 to 6 months later, or one single dose of DEC.

When treating filariasis with DEC, clinicians may use antihistamines or glucocorticoids to decrease the allergic reaction that may result from disintegration of microfilariae. Severe reactions may follow the administration of DEC to persons co-infected with filariasis and loiasis or onchocerciasis (a reason to prefer ivermectin instead of DEC in loiasis-endemic areas).

Management strategies for patients with lymphedema include elevation of the affected body part, compression stockings, skin care of the affected area, treatment of superficial fungal infections, as well as protection of the affected area from trauma and prompt antibiotic therapy for bacterial infections of the area. Surgical treatment includes debulking of the affected area, wide excision of chronic draining areas, and lymphatic shunts. Effectiveness varies, depending on the location of lymphedema.

Prevention.

The avoidance of mosquito bites in endemic areas is the primary means of prevention. Treatment of microfilaremic persons will reduce reservoir of parasites