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Lymphatic Filariasis: Disease Outbreaks in Military Deployments from World War II

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Lymphatic filariasis (LF) is the second most common parasitic disease worldwide, after malaria. It should always be considered in the differential diagnosis for military personnel returning from disease-endemic areas. Numerous outbreaks of LF have been reported in military deployments from World War II. In contrast to the presentation of LF in indigenous populations, which often involves such uncommon complications as elephantiasis and hydrocele, the clinical presentation of LF in military personnel can vary widely and is often vague and nondescript. Common symptoms are pain and swelling of the genitalia, closely followed by lymphangitis of the arms and legs. All three species produce similar disease.

Introduction

Lymphatic filariasis (LF) is the second most common vector-borne disease, after malaria. LF is caused by three species of nematode parasites, which can be spread by a wide range of mosquito species. The World Health Organization estimated the global burden of infection to be 120 million cases, with 1 billion people being at risk of infection.[1] LF is also the second most common cause of long-term disability.[1] It has a widespread geographic distribution, mainly in the tropical regions of the world (Fig. 1). *Wuchereria bancrofti* is the most common species and accounts for ~90% of cases. *Brugia malayi* is confined to East and Southeast Asia, and *Brugia timori* is found only in Timor and nearby islands. Given its widespread distribution and the increase in military deployments in filariasis-endemic areas, it should be expected that expatriate cases would occur and that steps should be taken to prevent transmission of LF among military personnel. After a brief review of the life cycle of LF, this article examines published accounts of LF affecting more recent military deployments since World War II.

Life Cycle of LF

The life cycle of LF is given in Figure 2. The adult worms live in the lymphatic vessels, where the females produce specialized motile embryos (microfilariae) that circulate in the peripheral blood. In most parts of the world, the concentration of microfilariae peaks at night (nocturnal periodicity), coinciding with the peak biting time of the vector mosquito. In the central Pacific region, where the vector is a daytime biter, the microfilariae peak occurs at midday (diurnal periodicity). It should be noted that, after the ingestion of microfilariae, there is maturation in the mosquito but, unlike in malaria, there is no multiplication. Also unlike in malaria, the infective stage (in LF, the larvae 3 or "L3" stage) is not inoculated directly into the new host. The L3 larvae are deposited onto the skin surface and migrate down through the puncture made by the proboscis of the mosquito.[2] Because of the lack of multiplication in the mosquito and the method of entry of the infective stage, it has been suggested that the transmission of filarial parasites is not as efficient as that of malaria and that many bites and/or a long period of exposure is required before infection occurs. This assumption is difficult to prove and, as shown below, may be erroneous.

Incidence of LF in Military Deployments

Many thousands of cases of filariasis occurred among the U.S. forces stationed in filariasis-endemic areas during World War II, especially in the Pacific theater, and filariasis was the leading cause of nontrauma medical evacuation in the South Pacific.[3] The situation is well attested to in the literature, with ~60 articles published during or soon after World War II.[4] It is estimated that ~38,300 naval personnel alone were exposed to filariasis and, of those, 10,421 were diagnosed as having the disease.

In one unit stationed in a highly disease-endemic area in Samoa, 70% of the exposed troops became infected.[5] One-fifth of the troops stationed on a particular island were admitted to the hospital with symptoms of filariasis, and there were 127 cases of filariasis at a field hospital in Samoa during a 4-month period. On Tonga-Tapu Island in the central Pacific region, 532 men were diagnosed as having filariasis in a single year. A total of 2595 patients with filariasis were admitted to the U.S. Marine Corps hospital at Klamath Falls, Oregon, during a 17-month period.[6] Troops were also infected elsewhere in the Pacific region, including Samoa, Wallis Island, New Caledonia, Bougainville, Cook Islands, Woodlark Island (Milne Bay Province, Papua, New Guinea), Ellice Island, Vanuatu, and the Solomon Islands.

According to Swartzwelder,[3] filariasis caused a significant loss of manpower and money and seriously compromised the fighting ability of U.S. Army units in the South Pacific. Swartzwelder[3] gave the following illustration. The 134th Field Artillery Battalion and the 404th Combat Engineer Company were stationed on Tongatapu Island in the central Pacific region from May 1942 to May 1943 and then on Woodlark Island (Milne Bay Province, Papua, New Guinea) from July 1942 to January 1944. Because of the large number of personnel infected with filariasis, the units were withdrawn to Sydney, Australia, where the prevalence of filariasis (based on clinical findings) among the evacuees was found to be 65% in the 134th Battalion and 55% in the 404th Company. The commanding general of the 6th U.S.

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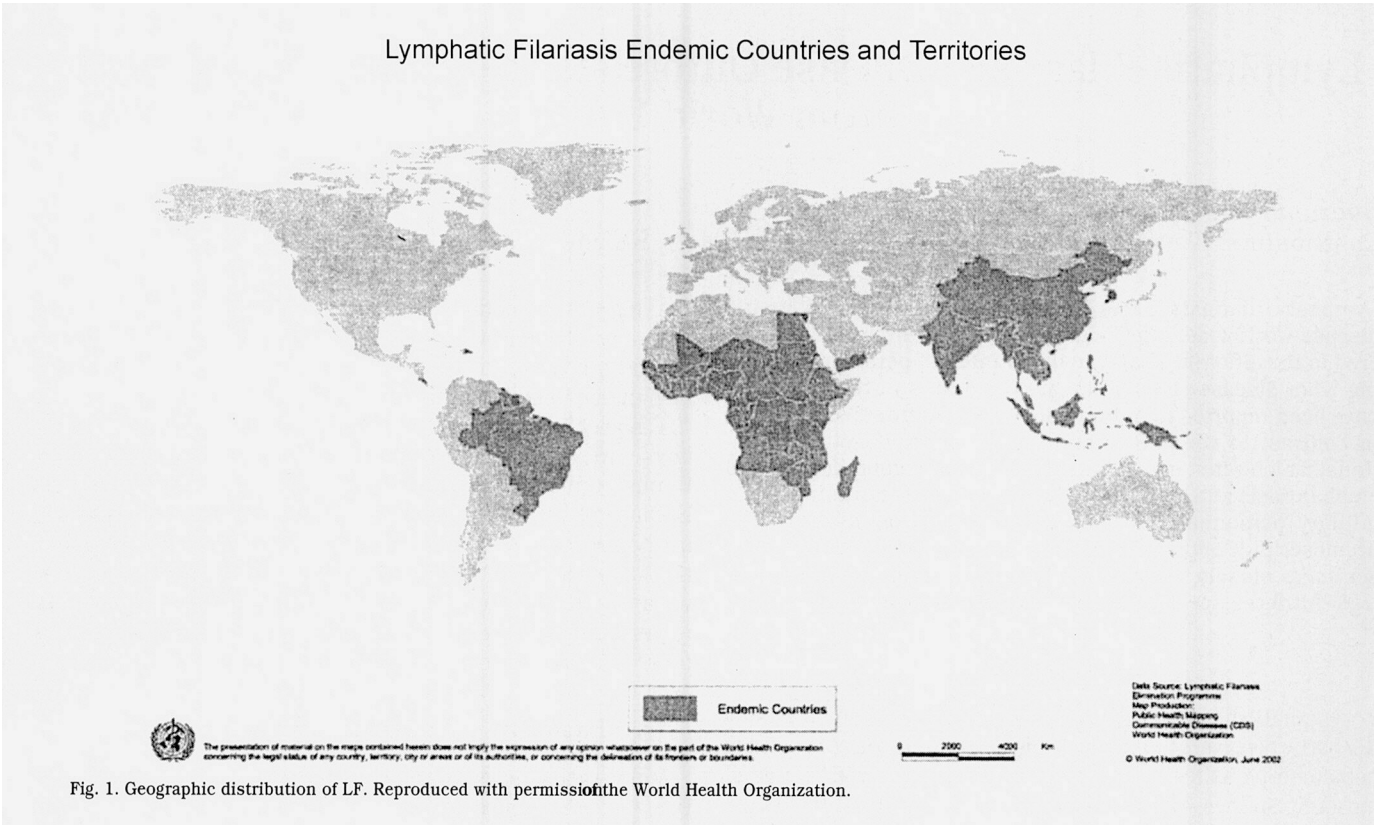

Fig. 1. Geographic distribution of LF. Reproduced with permission of the World Health Organization.

Army recommended that the entire unit be withdrawn from active service and return to the United States, because the fighting efficiency of the forces had been seriously impaired and their rehabilitation would extend for an indefinite period. There was also a high economic cost to the U.S. forces, estimated by Napier[7] at ~100 million dollars.

Incubation Period

Filariasis is often thought of as a slowly developing disease that requires a long period of exposure for infection. A notable feature of World War II cases was the short exposure time to clinical manifestation of disease, in some cases only 1 month.[4] One of the most notable features of these cases was that microfilaremia was detected for only approximately 20 patients.[4,5] Despite the failure to demonstrate microfilariae in most cases, there was little doubt about the diagnosis. The clinical findings were typical of acute filariasis and, most telling of all, adult worms were recovered in ~30% of cases.[4] There are at least two possible explanations for the lack of circulating microfilariae. In expatriate cases, the adult worms induce a marked inflammatory reaction, with a dense collection of inflammatory cells. It is possible that the microfilariae get trapped within the lesion and are unable to make their way into the circulation. Another, probably more plausible, explanation is that the immune response to the parasite among previously unexposed expatriates is more vigorous than that seen among indigenous residents, for whom blunting of the immune response (or "tolerization") starts in the womb,[8] and the microfilariae are rapidly destroyed. When the rare cases of microfilaremia did occur, the microfilariae showed the diurnal periodicity pattern that was usually seen in the native population.[9] Almost all cases occurring among the U.S. troops the cases could be termed "acute filariasis" and would fall into the modern category of filarial adenolymphangitis, and it was observed by Huntington et al.[10] that the signs and symptoms were identical to those of the syndrome called "mumu," present in Samoan indigenous populations. The attacks commonly lasted 2 to 5 days, with occasional incidences of 2 weeks or, very rarely, 1 month.[11] Recurrent attacks were common, occurring at intervals of a few weeks to several months. Hard exercise, fatigue, and a hot climate were thought to be precipitating factors.[4]

Presentation of LF

The presenting diagnostic criteria used by Hodge et al.[12] are provided in Table I. The most common presenting signs and symptoms in the U.S. Armed Forces cases were pain and swelling of the genitalia, closely followed by lymphangitis of the arms and legs. Lesions of the genitalia (in order of frequency) were funiculitis, epididymitis, scrotal edema and inflammation, lymph scrotum, orchitis, hydrocele, and varicocele. Lymphangitis occurred in 51% to 80% of cases. The most common site was the arm, followed by the leg, groin, buttock, and abdomen. An important feature was the tendency for the inflammation to spread in a retrograde direction. Some authors reported skin temperature increases in the affected area but others did not. It should be noted that generalized fever was uncommon. Lymphedema was occasionally observed but was not common. The prevalence of lymphadenitis varied widely from group to group and ranged from 7% to 85%. When lymphadenitis was present, the most commonly affected nodes were the epitrochlear and the inguinal, but other nodes were often involved. Evidence of hypersensitivity was common, with many patients

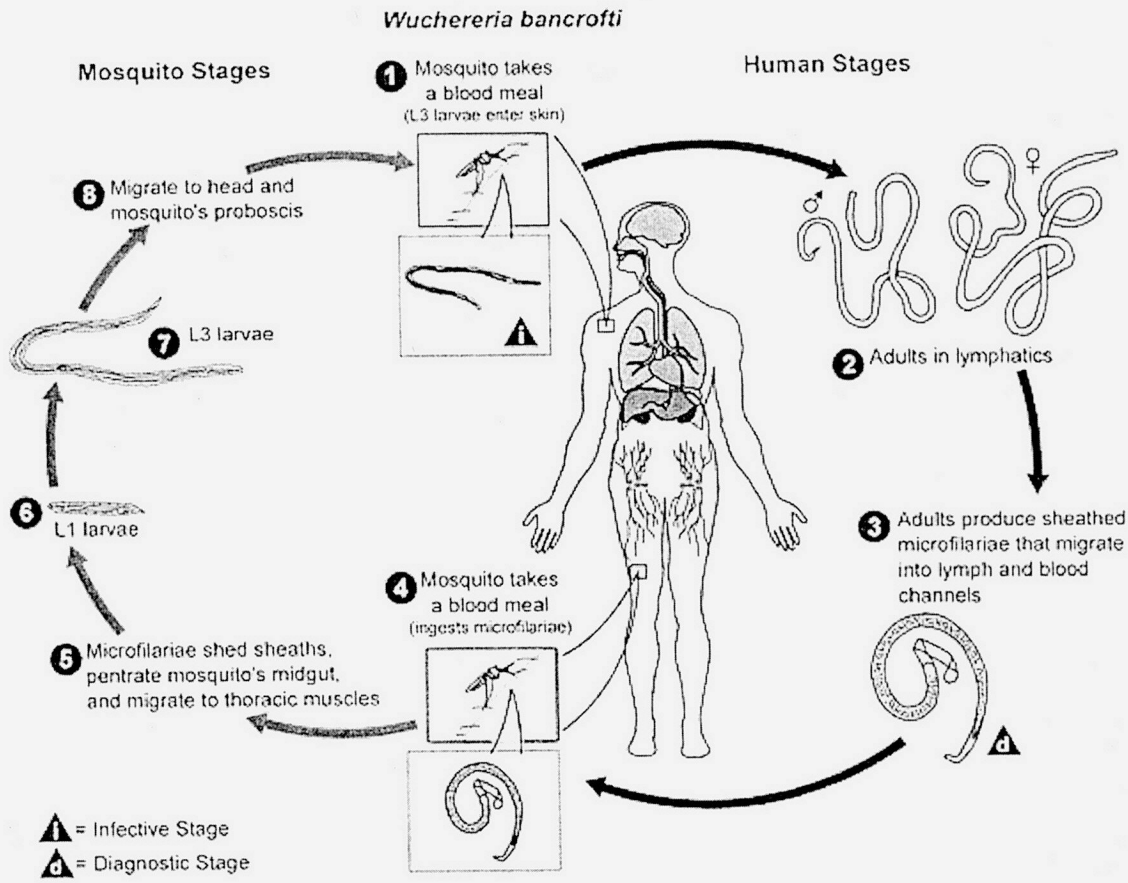

Fig. 2. Life cycle of LF. Reproduced with permission of the Centers for Disease Control and Prevention, National Center for Infectious Diseases.

suffering urticaria and transient swellings. Some of the latter resembled the "Calabar swellings" or "fugitive swelling" usually associated with *Loa loa* infections. A small number of patients developed a macular erythematous rash. Hypersensitivity-type manifestations were seen only while the patients were a disease-endemic area, which raises the possibility that they were caused by a reaction to mosquito-injected L3 larvae. One study reported a high incidence of conjunctivitis and some cases of photophobia. The most common laboratory finding was eosinophilia; in a few cases, there was a very high eosinophil count associated with a chronic cough, a hallmark of tropical eosinophilia syndrome.[13-15]

Psychological disturbances were commonly described.[3,6,11,16-22] Typical manifestations were depression, irritability, concentration difficulties, nervousness, anxiety, sleep disorders, and fear. It is possible that filariasis per se does produce some mood swings and depression,[17] but many of these symptoms might have been precipitated by the patients observing Polynesian and Melanesian nationals with disfiguring diseases such as severe hydrocele and elephantiasis. Affected men would rather carry their scrotums in a wheelbarrow than risk loss of sexual function with treatment. As it turned out, however, these fears were unfounded. No cases of chronic pathological conditions occurred. Although Behm and Hayman[11] reported on 33 patients who believed their sexual potency had been impaired by filariasis and Smith[21] reported that many of his patients reported a loss of libido, there was no evidence of long-term effects on sexual function and the prevalence of impotence and sterility was no higher among filariasis patients than

TABLE I

DIAGNOSTIC CRITERIA FOR ACUTE LF, ACCORDING TO HODGE ET AL.[12]

Involvement of the genitalia
Episodes frequently follow periods of manual labor.
Testicular pain is the most common complaint.
The pain may radiate up the spermatic cord or may appear first in the lower abdomen and radiate down the spermatic cord to the testicle.
The spermatic cord and epididymis are thickened and indurated but the vas deferens is not involved.
Acute hydrocele is often present.
Pain in the mesial aspect of the thigh on the affected side may be present.
Scrotal edema is often present.
Involvement of the superficial lymphatic vessels of the extremities
Pain in the extremity radiating distally is the usual complaint.
Pain may be noticed first in the proximal lymph nodes.
The affected extremity becomes edematous.
A definite "red streak" characteristic of acute lymphangitis appears proximally. The affected lymphatic vessel is palpable, and usually only a single vessel is involved.
There is increased local heat.
The lesion extends centrifugally.
Resolution begins proximally and extends centrifugally.
The lymph nodes draining the affected area are enlarged and tender.
Severe constitutional manifestations are usually absent.

in the general population.[4] Coggeshall[6] reported that 107 of 504 marriages among Marines with a diagnosis of filariasis resulted in pregnancy within 6 months.

Behm and Hayman[11] and Wartman[4] reported that in most cases, even without treatment, attacks became progressively milder when patients were removed from the disease-endemic area. Most men were free of symptoms after they had been out of the disease-endemic area for 20 months but occasional cases persisted for up to 3 years. In contrast, Trent,[23,24] who reevaluated 25 men who had been diagnosed as having filariasis during World War II, found that many of them still had evidence of the disease 15 years after leaving the disease-endemic area. Intermittent genital symptoms (pain, swelling, and tenderness precipitated or aggravated by standing or physical exercise) were present in 32% of cases. Forty percent of patients had abnormal physical findings, such as thickening of the spermatic cord, the presence of a tender nodule near the testis, induration of the testis, varicocele, or thickening of the scrotal skin. Recurrent lymphangitis still occurred in 12% of cases, with attacks occurring 1 to 3 times per year and lasting for 1 to 3 days. Symptoms consisted of sudden onset of fever, headache, malaise, swelling of affected lymphatic vessels, and swelling of regional lymph nodes. In 68% of cases, there were episodes of swelling of the extremities and enlargement of the regional lymph nodes draining the area. These episodes tended to occur most frequently in the summer months, and the patients complained that the skin of the affected extremities became "puffy, stiff, and tight."

Winstead[25] reported on a patient who still complained of symptoms 30 years after contracting the disease during military service in Samoa. At the time of his examination (1972), there was no clinical evidence of active filariasis but the patient had a positive skin test and a weakly positive hemagglutination test. There is no evidence that any of those infected during World War II developed chronic hydrocele or elephantiasis. Although microfilaremia was very rare, occasional cases persisted for up to 15 years.[23,24,26]

There are several vexing questions to be answered with regard to the U.S. Armed Forces cases. Why did most of the cases occur in the central Pacific area? Most authors seem to believe that it was because the soldiers were in close contact with infected indigenous people in an area in which filariasis was highly endemic. Papua, New Guinea, and the Solomon Islands are also areas in which filariasis is highly endemic, and many U.S. troops were stationed there. Although some U.S. Armed Forces cases did occur in these areas, the numbers seem to be far lower than those for the Pacific area. Wartman[4] suggested that the large number of cases in the central Pacific area was attributable to the fact that the vector mosquito is a daytime feeder, the microfilariae have diurnal periodicity, and, because most of the troops' activities took place during the day, the troops were heavily exposed. This does not explain why fewer cases occurred in Papua, New Guinea, and the Solomon Islands. The vectors there are mainly nighttime feeders, but the troops, being in an active theater of war and occupying positions both night and day, would have been heavily exposed. Another interesting observation was the small number of filariasis cases among Australian troops, although they were stationed in highly disease-endemic areas such as Papua, New Guinea. Walker[27] stated that there were only 22 cases of filariasis among Australian troops; some of these men were from Queensland, where filariasis was endemic at the time, and might have acquired the infection before enlisting in the forces. There might simply have been a lack of familiarity with filariasis and thus the diagnosis was missed but this is unlikely, because the Australian forces medical staff members were aware of the risk of filariasis and had been extensively briefed on the U.S. experience in the Pacific region.[27]

Incidence of LF in More Recent Deployments

Filariasis has also been reported for troops serving in other theaters of war. Alhadeff[28] reported cases of acute filariasis among Mauritian troops stationed in the filariasis-endemic areas of Egypt. It difficult to say, however, whether these were locally acquired infections, because filariasis also occurs in Mauritius.[29] Filariasis also occurred among expatriate troops serving in Vietnam, where both *W. bancrofti* and *B. malayi* occur.[29-32] In the 1950s, a syndrome consisting of hypereosinophilia, bronchial asthma, and lymphadenopathy was observed for 151 French and North African troops serving in the Tonkin area of North Vietnam. Microfilariae of *B. malayi* were recovered from the lymph nodes of some patients, but there were no reports of circulating microfilariae. Treatment with arsenical agents or diethylcarbamazine was followed by rapid clinical improvement.[33] Brown and Armstrong[34] and Sullivan et al.[35,36] reported some cases of filariasis among U.S. service personnel. With the use of a soluble-antigen fluorescent antibody test, Colwell et al.[31] were able to show that 11% of U.S. service personnel who were stationed in filariasis-endemic regions of South Vietnam had antifilarial antibodies. In contrast, only 3% of those stationed in apparently non—disease-endemic areas were positive. Moreover, when the service personnel were characterized according to the degree of potential exposure, the prevalence of seropositivity among field troops was three times higher than that among base personnel. One serviceman was also found to have circulating microfilariae. Sullivan et al.[36] reported a case of clinical filariasis without microfilaremia that rapidly resolved after diethylcarbamazine therapy. Again, there appeared to be reported cases of filariasis among Australian military personnel. Most of Vietnam is malarious and, as far as can be ascertained, U.S. and Australian service personnel were receiving the same antimalarial prophylactic regimen.

Conclusions

LF should always be considered in the differential diagnosis for personnel returning from disease-endemic areas. Filariasis usually expresses itself in a very different manner among exposed military personnel, compared with indigenous populations, which often present images of uncommon complications such as elephantiasis and hydrocele. The clinical presentation of filariasis among military personnel can vary widely and can be very vague and nondescript. All three species cause similar disease.

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