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RECENT TRENDS IN TREATMENT AND MANAGEMENT OF FILARIASIS

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ABSTRACT

Filariasis is the name for a group of tropical diseases caused by various thread-like parasitic round worms (nematodes) and their larvae. The larvae transmit the disease to humans through a mosquito bite. Filariasis is characterized by fever, chills, headache, and skin lesions in the early stages and, if untreated, can progress to include gross enlargement of the limbs and genitalia in a condition called elephantiasis. While filariasis is rarely fatal, it is the second leading cause of permanent and long-term disability in the world. The World Health Organization (WHO) has named filariasis one of only six "potentially eradicable" infectious diseases and has embarked upon a 20-year campaign to eradicate the disease. These infections have a significant economic and psychosocial impact in endemic areas, disfiguring and/or incapacitating more than 40 million individuals. Studies from the Indian subcontinent have shown that infected patients lose significant time from work because of the disease costing the national treasury a minimum of $842 million per year. The treatment of filariasis consists of using medicines that kill the worms combined with the treatment to relieve the symptoms. Filariasis may be treated in early, mild cases with a three-week course of antifilarial drugs. This medication usually cures the infection, but may cause a reaction marked by fever, illness, and muscle or joint pains. Treatment for symptomatic relief includes bed rest, antibiotic use for secondary infections, elastic stockings and pressure bandages to reduce swelling and fluid accumulation, and suspensory bandaging for swollen testicles or breasts. Chronic infections are more difficult to treat effectively. Small accumulations of fluid may benefit from local injection of sclerosing (condensing) agents. Surgery may be required. Mass accumulations may be managed using shunt procedures combined with removal of excess fatty and fibrous tissue, drainage and physical therapy.

INTRODUCTION: In all cases, a mosquito first bites an infected individual then bites another uninfected individual, transferring some of the worm larvae to the new host. Once within the body, the larvae migrate to a particular part of the body and mature to adult worms. Filariasis is classified into three distinct types according to the part of the body that becomes infected: lymphatic filariasis affects the circulatory system that moves tissue fluid and immune cells (lymphatic system); subcutaneous filariasis infects the areas beneath the skin and whites of the eye; and serous cavity filariasis infects body cavities but does not cause disease. A larva matures into an adult worm within six months to one year and can live between four and six years. Each female worm can produce millions of larvae, and these larvae only appear in the bloodstream at night, when they may be transmitted, via an insect bite, to another host [1, 2].

A single bite is usually not enough to acquire an infection; therefore, short-term travelers are usually safe. A series of multiple bites over a period of time is required to establish an infection. As a result, those individuals who are regularly active outdoors at night and those who spend more time in remote jungle areas are at an increased risk of contracting the filariasis infection [6, 7]. The disease is rarely fatal, and with continued WHO medical intervention, even gross elephantiasis is now becoming rare. The best way to prevent filariasis isto prevent being repeatedly bitten by the mosquitoes that carry the disease by: limiting outdoor activities at night, particularly in rural or jungle areas; wearing long sleeves and pants and avoiding dark-colored clothing that attracts mosquitoes; avoiding perfumes and colognes; treating clothing ahead of time with permethrin (Duramon, Permanone); wearing DEET insect repellent, citronella or lemon eucalyptus to repel insects; if sleeping in an open area or in a room with poor screens, use a bed net to avoid being bitten while asleep; using air conditioning; and in highly infested areas, taking ivermectin preventatively. Scientists are working on a vaccine. Diethylcarbamazine, orivermectin are effective drugs in the treatment of most filarial infections. Other drugs include albendazole and mebendazole. Antihistamines or corticosteroids can decrease allergic reactions. In case of elephantiasis, one needs to take a yearly dose of medicine that kills the microscopic worms in the blood [4, 5].

History of Filariasis: Lymphatic Filariasis is thought to have affected humans since approximately 4000 years ago. Artifacts from ancient Egypt (2000 BC) and the Nok civilization in West Africa (500 BC) show possible elephantiasis symptoms. The first clear reference to the disease occurs in ancient Greek literature, where scholars differentiated the often similar symptoms of lymphatic filariasis from those of leprosy. The first documentation of symptoms occurred in the 16th century, when Jan Huyghen van Linschoten wrote about the disease during the exploration of Goa. Similar symptoms were reported by subsequent explorers in areas of Asia and Africa, though an understanding of the disease did not began to develop until centuries later.

In 1866, Timothy Lewis, building on the work of Jean-Nicolas Demarquay and Otto Henry Wucherer, made the connection between microfilariae and elephantiasis, establishing the course of research that would ultimately explain the disease. In 1876, Joseph Bancroft discovered the adult form of the worm. In 1877, the life cycle involving an arthropod vector was theorized by Patrick Manson, who proceeded to demonstrate the presence of the worms in mosquitoes. Manson incorrectly hypothesized

that the disease was transmitted through skin contact with water in which the mosquitoes had laid eggs. In 1900, George Carmichael Low determined the actual transmission method by discovering the presence of the worm in the proboscis of the mosquito vector [2, 3].

Pathophysiology of Filariasis: The filarial life cycle, like that of all nematodes, consists of 5 developmental or larval stages in a vertebral host and an arthropod intermediate host and vector. Adult female worms produce thousands of first-stage larvae or microfilariae that are ingested by a feeding insect vector. Some microfilariae have a unique daily circadian periodicity in the peripheral circulation. The arthropod vectors, mosquitoes and flies, also have a circadian rhythm in which they obtain blood meals.

The highest concentration of microfilariae usually occurs when the local vector is feeding most actively. Microfilariae then undergo two developmental changes in the insect. Third-stage larvae then are inoculated back into the vertebral host during the act of feeding for the final two stages of development. These larvae travel through the dermis and enter regional lymphatic vessels. During the next 9 months, these develop into mature worms (20-100 mm in length). An average parasite can survive for about 5 years. The prepatent period is defined as the interval between a vector bite and the appearance of microfilaria in blood, with an estimated duration of about 12 months.

The following factors affect the Pathogenesis of Filariasis:

- The quantity of accumulating adult worm antigen in the lymphatics
- The duration and level of exposure to infective insect bites
- The number of secondary bacterial and fungal infections
- The degree of host immune response

Filarial infection generates significant inflammatory immune responses that participate in the development of symptomatic lymphatic obstruction. Increased levels of immunoglobulin E (IgE) and immunoglobulin G4 (IgG4) secondary to antigenic (from dead worms) stimulation of Th2-type immune response have been demonstrated. Studies have shown that there is a familial tendency to lymphatic obstruction, providing support for the hypothesis that host genes influence lymphedema susceptibility. Prenatal exposure seems to be an important determinant in conferring greater immune tolerance to parasite antigen.

Thus, individuals from endemic areas are often asymptomatic until late in disease when they have high worm burden, whereas non-immune expatriates tend to have brisk immune responses and more severe early clinical symptoms, even in light infections. Recent studies have shown that lymphatic filarial parasites contain rickettsialike *Wolbachia* endosymbiotic bacteria. This association has been recognized as contributing to the inflammatory reaction seen in filariasis. Symptoms of filariasis are species-dependent and body-site–dependent and can be acute or chronic in nature. Up to 70% of infected individuals remain asymptomatic. Symptoms usually do not manifest until adolescence or adulthood, when worm burden is usually the highest. Several variations have been observed.

Lymphatic Filariasis:

- The symptoms of lymphatic filariasis predominantly result from the presence of adult worms residing in the lymphatics.
- The clinical course is broadly divided into asymptomatic microfilaremia, acute phases of adenolymphangitis (ADL), and chronic irreversible lymphedema.

Three acute syndromes have been described in filariasis, as follows:

- Acute ADL: This refers to the sudden onset of febrile painful lymphadenopathy. Pathologically, the lymph node is characterized by a retrograde lymphangitis, distinguishing it from bacterial lymphadenitis. Symptoms usually abate within one week, but recurrences are possible.
- Filarial fever: This is characterized by fever without the associated adenitis.

Tropical Pulmonary Eosinophilia:

- TPE is a form of occult filariasis.
- Presenting symptoms include a paroxysmal dry cough, wheezing, dyspnea, anorexia, malaise, and weight loss.
- Symptoms of TPE are usually due to the inflammatory response to the infection. Characteristically, peripheral blood eosinophilia and abnormal findings on chest radiography are observed. TPE is usually related to *W bancrofti* or *B malayi* infection.

Onchocerciasis:

- This also is known as hanging groins, leopard skin, river blindness, or sowda.
- Symptoms result from the presence of microfilariae in the skin and include pruritus, subcutaneous lumps, lymphadenitis, and blindness.
- Patients with onchocerciasis may report impaired visual acuity due to corneal fibrosis.

Loiasis:

- The symptoms of *L loa* infection are usually confined to subcutaneous swellings on the extremities, localized pain, pruritus, and urticaria.
- Microfilaremia tends to be asymptomatic.
- Occasionally, the worm is observed migrating through the subconjunctiva or other tissues [9, 10, 11].

CAUSES OF FILARIASIS:

Lymphatic Filariasis:

- Mosquitoes of the genera *Aedes, Anopheles, Culex,* or *Mansonia* are the intermediate hosts and vectors of all species that cause lymphatic filariasis.
- Acute lymphatic filariasis is related to larval molting and adult maturation to fifth-stage larvae. Adult worms are found in lymph nodes and lymphatic vessels distal to the nodes. Females measure 80-100 mm in length and males are 40 mm.

- The most commonly affected nodes are in the femoral and epitrochlear regions.
- Abscess formation may occur at the nodes or anywhere along the distal vessel.
- Infection with *B timori* appears to result in more abscesses than infection with *B malayi* or *W bancrofti.*
- Cellular invasion, with plasma cells, eosinophils, and macrophages, together with hyperplasia of the lymphatic endothelium, occurs with repeated inflammatory episodes. The consequence is lymphatic damage and chronic leakage of protein-rich lymph in the tissues, thickening and verrucous changes of the skin, and chronic streptococcal and fungal infections, which all contribute to the appearance of elephantiasis.
- *B malayi* elephantiasis is more likely to affect the upper and lower limbs, with genital pathology and chyluria being rare [13, 14].

Occult Filariasis:

- Occult filariasis denotes filarial infection in which microfilariae are not observed in the blood but may be found in other body fluids and/or tissues.
- The occult syndromes are TPE, *D immitis* or *D repens* infection, filarial arthritis, filarial breast abscess, and filarial-associated immune-complex glomerulonephritis.
- TPE most likely results from a hyperresponsiveness to *W bancrofti* or *B malayi* antigen.
- Human infection with *D immitis* may result in pulmonary lesions of immature *Dirofilaria* worms in the lung periphery. If *D immitis* larvae lodge in branches of the pulmonary arteries, they can cause pulmonary infarcts [12, 13].

Onchocerciasis:

- Microfilariae from the skin are ingested by the *Simulium* species of blackflies.
- Chronic onchocerciasis cases are hyperresponsive to parasite antigen, have increased eosinophilia, and result in the presence of high levels of serum IgE.
- Patterns of onchocercal eye disease also are associated with parasite strain differences at the DNA level.

Loiasis:

- Mango flies or deerflies of *Chrysops* transmit loiasis.
- Response to *L loa* infection appears to differ between residents and nonresidents in endemic areas. Nonresidents with infection appear to be more prone to symptoms than residents, despite lower levels of microfilaremia. Eosinophil, serum IgE, and antibody levels are also higher in nonresidents.

Filariasis refers to parasitic infection caused by filarial parasites. The clinical manifestation (table 1 &2, figure 1-3) and treatment of filariasis depends on the type of filarial parasitic worm (nematode) involved [15, 17].

TABLE 1: THE FOLLOWING TABLE LISTS THE PARASITE AND THE FILARIAL DISEASE CAUSED

FILARIAL PARASITE	FILARIAL DISEASE
Onchocerca volvulus	Onchocerciasis
Wuchereria bancrofti	Bancroftian filariasis (lymphatic filariasis)
Brugia malayi and *Brugia timori*	Malayan filariasis (lymphatic filariasis)
Loa loa	Loiasis
Mansonella species	Mansonelliasis
Dirofilaria species	Dirofilariasis

TABLE2: ALL FILARIAL INFECTIONS CAUSE SOME TYPE OF SKIN PROBLEMS IN ADDITION TO SYSTEMIC MANIFESTATIONS

DISEASE	PARASITE	VECTOR
Onchocerciasis	*O volvulus*	Blackflies: *Simulium* species
Bancroftian filariasis	*W bancrofti*	Mosquitoes: *Anopheles, Aedes, Culex,* and *Mansonia* species
Malayan filariasis	*B malayi* and *B timori*	Mosquitoes: *Anopheles, Aedes, Culex,* and *Mansonia* species
Loiasis	*L loa*	Red flies: *Chrysops* species
Mansonelliasis	*M streptocerca*	Midges: *Culicoides* species
Dirofilariasis	*Dirofilaria* species	Mosquitoes: *Culex* species

FIG. 1: FEMALE AEDES POLYNESIENSIS CHOOSE TO HUMANS, PIGS, DOGS, HORSES, BIRDS OR OTHER ANIMALS AS HOSTS

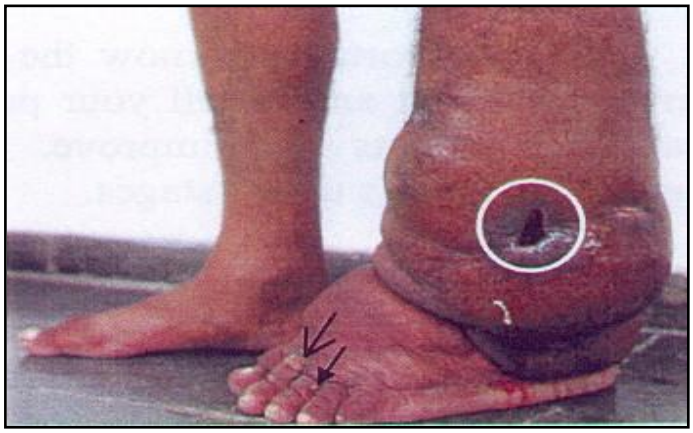

FIG. 2: THE LEG OF A PATIENT SUFFERING FROM LYMPHATIC FILARIASIS

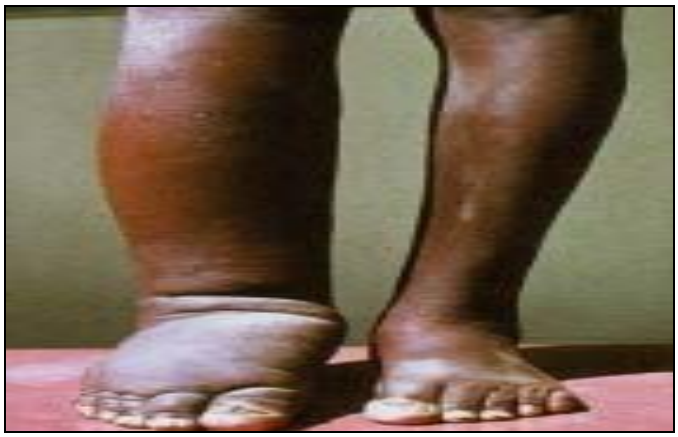

FIG. 3: LYMPHATIC FILARIASIS CAN PROGRESS TO ELEPHANTIASIS, A SWELLING AND THICKENING OF BODY TISSUES

SYMPTOMS: Adult worms live in the lymph vessels and nodes, while the younger forms are found primarily in the blood. The symptoms are seen four to twelve months after infection, and usually begin with swelling and inflammation in the genitals or extremities. Other symptoms include fever, pain and swelling of lymph glands, headache, and inflammation of the lymph drainage areas, swelling of the scrotum, skin rashes and blindness. Progression of the disease often causes enlargement of the legs resulting in a condition called elephantiasis or lymphatic filariasis. This enlargement occurs due to the

presence of lymphoedema or presence of fluid in the tissue spaces that may begin to accumulate in the first 24 hours. The skin becomes thick and rough and the increase in the size and weight of the affected parts lead to disability [18, 19].

PREVENTION: Prevention includes giving medicine that kills the microscopic worms, to the entire community in the areas where the infection is prevalent. Avoiding mosquito bites is another form of prevention. These mosquitoes usually bite between the hours of dusk and dawn. One can follow these steps, if living in an infected area:

- Sleep under a mosquito net.
- Use mosquito repellents on the exposed skin.

Take a yearly dose of medicine that kills the worms in the blood [20, 21].

DIAGNOSIS:

History: The history will include travel in tropical or subtropical areas and significant history of insect bites. The incubation period will have been at least eight months for travelers and up to two to three years for native persons in a country where filariasis is prevalent. Symptoms of acute disease include episodes of fever with pain and/or swelling of lymph nodes (lymphadenitis) and lymphatic vessels (lymphangitis). These episodes will occur at irregular intervals and last several days. With disease progression, inflammation of the epididymis or testicles as well as involvement of the abdominal, pelvic, and peritoneal lymph vessels may occur intermittently. Lymph node enlargement may persist. Allergy-like symptoms of hives and rashes are likely to be found in travelers who are infected repeatedly. Individuals with chronic disease may show symptoms of obstruction and interference with normal lymphatic flow. Symptoms may include a rapid increase in the size (mass) of the extremities, genitals, or breasts [22, 23].

Physical Examination: It may show characteristic painful/swollen lymph nodes (lymphadenitis), which is most severe at the affected node and decreases in intensity with distance from the node (retrograde). In later stages, involvement of the peritoneal lymphatics will also occur in a retrograde fashion. Abdominal palpation (examination by pressing on the abdomen) may reveal a swollen spleen or liver. In chronic cases, swelling of the scrotum and enlarged lymphatic vessels are characteristic physical symptoms. The infected area, commonly a limb or the scrotum, becomes enormously enlarged, and the skin becomes thick, coarse, and cracked (fissured). Hard masses may accumulate in the breasts, legs, hands or testicles. A milky, white substance (chyle) may appear in the urine as a result of lymphatic vessel rupture into the urinary tract.

Tests: The diagnosis of filariasis is confirmed by microscopic examination of blood or lymph fluid for the presence of microfilariae. Blood tests will show immature worms in the blood after six to twelve months of infection. It may take two to three years for worms to develop in the blood of indigenous persons. Worms may also be present in fluids drawn from swollen areas. Each species of filarial nematode will show characteristic structure and form (morphology) under microscopic examination. Microscopic (histologic) examination of the skin in areas of mass accumulation will show hardening and loss of elasticity. Lymph nodes may become fibrotic and secondary bacterial infections may be

detected. In blood studies (serology), chronic infections may show high filarial antibody titer and IgE levels. Lung infection will show high eosinophil counts (eosinophilia). In occult disease, worms are not present in the blood. Adult worms may be detected in tissues using ultrasonography. X-rays may show scattered, small nodular lesions on the lungs. There may be increased evidence of vascular damage on the chest films [24].

Risk of Filariasis: Over 120 million have already been affected by it, over 40 million of them are seriously incapacitated and disfigured by the disease.One-third of the people infected with the disease live in India, one third are in Africa and most of the remainder are in South Asia, the Pacific and the Americas.In communities where the condition is endemic, 10-50% of men and up to 10% of women can be affected.Though the infection is generally acquired early in childhood, the disease may take years to manifest itself [23, 25].

Medication of Filariasis: Filariasis can be effectively treated by oral administration of drugs like, Diethyl carbamazine (DEC) 6 mg/ kg /day once a day or in divided doses for 2-3 weeks. This drug kills the microfilariae very effectively while the effect on the adult worms is slow. The drug is started in small doses and gradually increased over a course of few days. Side effects like swelling of the lymph nodes, abscess, ulceration, fever, headache, body ache and allergic reactions can occur due to the killing of the microfilariae and the adult worms. Several courses of treatment may be required. Supportive management includes, bed rest, elastic bandages for reducing the edema and antibiotic drugs to combat the secondary infection. Lvermectin has also been used in single or two divided doses (starting dose of 20mic per kg followed by 400 mic per kg). For chronic lymphatic obstruction elevation of affected limb, elastic stockings can be tried. Injecting sclerosants can treat small hydrocoeles. Surgical management may also be necessary. For elephantiasis, surgical procedures like removal of the excess of subcutaneous tissue or lymphovenous shunt may be necessary. Early cases have a good prognosis if properly treated. Cases identified in the late stages have a poor prognosis [27, 29]. Treatment depends on the infecting parasite.

Onchocerciasis: Ivermectin is the antihelminthic drug of choice

Nodulectomy: Surgical removal of the palpable nodules in combination with ivermectin therapy may be useful

Lymphatic Filariasis: Diethylcarbamazine (DEC) is the drug registered for use in this disease, however it is associated with many side effects Ivermectin and DEC appears to be effective combination as they act synergistically. Tetracycline antibiotics are used to kill *Wolbachia* bacteria. Severely damaged extremities may undergo surgical decompression of the lymphatic system.

Loiasis: DEC is the drug of choice Ivermectin is contraindicated as it can cause retinal haemorrhage and severe or fatal encephalopathy. Live adult worms can be extracted as they traverse the eye.

Mansonelliasis: Combination treatment with DEC and mebendazole appears to be the ideal treatment for *M perstans* infections, Ivermectin appears to be effective treatment against *M streptocerca* infections.

Dirofilariasis: Surgical removal of the lesion or extraction of the worm is the only method of treatment for human infections.

Lymphatic Filariasis:

Diagnosis of Lymphatic Filariasis: The recent developments in the diagnosis of lymphatic filariasis are given below, which have heralded changes in the management strategies.

Membrane filtration method for microfilaria detection: Venous blood drawn at night and filtered through millepore membrane filters, enables easy detection of microfilaria and to quantify the load of infection. They are usually seen in the early stages of the disease before clinical manifestations develop. Once lyphoedema develops microfilaria are generally absent in the peripheral blood. The Quantitative Blood Count (QBC) methods also can be used to identify the microfilaria and to study their morphology in the blood drawn at night. Though this can be performed quickly, it is no more sensitive than examination of the conventional blood smear.

Ultrasonography: Recently, ultrasonography using a 7.5 or 10 MHz probe has helped to locate and visualize the movements of living adult filarial worms of W. bancrofti in the scrotal lymphatics of asymptomatic males with microfilaraemia. The constant thrashing movement of the adult worms in their 'nests' in the scrotal lymphatics is described as the 'filaria dance sign. The lymphatic vessels lodging the parasite are dilated and this dilation is not seen to revert to normal even after the worms are killed by administration of diethylcarbamazine. Ultrasound has been used to study the effect of drugs on the adult worms and to retrieve them surgically from the dilatedscrotallymphatics. Ultrasonography is not useful in patients with filarial lymphoedema because living adult worms are generally not present at this stage of the disease. Similarly ultrasonography has not helped in locating the adult worms of B. malayi in the scrotal lymphatics since they do not involve the genitalia [27, 29.]

Lymphoscintigraphy: The structure and function of the lymphatics of the involved limb can be assessed by lymphoscintigraphy. After injecting radio-labeled albumin or dextran in the web space of the toes, the structural changes are imaged using a gamma camera. Lymphatic dilatation, dermal back flow and obstruction can be directly demonstrated in the oedematous limbs by this method. Lymphoscintigraphy has shown that even in the early, clinically asymptomatic stage of the disease, there are lymphatic abnormalities in the affected limbs of people harboring microfilaria.

Immunochromatographic Test (ICT): Highly sensitive and specific filarial antigen detection assays, both as card test and in ELISA based format are now available for the diagnosis of W. bancrofti infection. The card test has the advantage that it can be performed on blood sample drawn by finger prick at any time of the day. This test is positive in early stages of the disease when the adult worms are alive and becomes negative once they are dead. At present no such test is available for B. malayi filariasis, where the detection of IgG4 antibodies is helpful [25, 28].

DNA probes using Polymerase Chain Reaction (PCR): These tests are of high specificity and sensitivity, which are available to detect parasite DNA in humans as well as vectors in both bancroftian and brugian filariasis. Though this method is quick and easy to perform, the

disadvantage is that it requires sophisticated equipment and is available only in very few centers [29, 32].

Treatment and Prevention of Lymphatic Filariasis: DEC (6 mg/kg) is the drug registered for use in lymphatic filariasis. Efficacious treatment is the administration of high-dose DEC. Unfortunately, DEC administered in this fashion causes adverse effects, which remained a disincentive to its use in many locales. A low dosage of DEC can be administered to all residents of an endemic area except infants, pregnant women, elderly persons, and persons with debilitating disorders. Sometimes, low-dose DEC is combined with albendazole. Ivermectin (400 mcg/kg/d) is an equally potent microfilaricide, and the combination of DEC and ivermectin provides significant synergism. Tetracycline antibiotics kill Wolbachia endosymbionts and have a macrofilaricidal effect in lymphatic filariasis.

Doxycycline at 100 mg/d for 6-8 weeks has demonstrated efficacy against lymphatic filariasis. For control, the World Health Organization has long recommended a single, yearly oral dose of ivermectin (400 mcg/kg) with DEC (6 mg/kg), or, under specific conditions, either of these drugs alone or daily use of DEC-fortified salt. DEC is also effective in killing some, but not all, adult worms. Eberhard et al reported that ivermectin has the same ability as DEC to decrease the level of circulating W bancrofti antigen Og4C3; ivermectin also suppresses microfilaremia for prolonged periods, but it does so without killing the adult worm. Curiously, a single dose of DEC has essentially the same long-term effectiveness in decreasing microfilaremia and in the apparent killing of adult worms as the 1- to 3-week courses previously recommended. This has enormous implications for control programs, but studies should be performed to decide if the standard 6- to 12-day regimen for most infections and the 34-week regimen for tropical eosinophilic syndrome should be altered for individual patients. Using DEC alone can cause problems. DEC sometimes can precipitate acute inflammatory reactions, especially in persons with Malayan filariasis, which merely accelerates the natural history of the disease. The efficacy of albendazole is now under investigation. SmithKline Beecham is donating albendazole at no cost to organizations and governments wishing to assess the drug's ability to control filariasis. Aplysinopsin, an extract from marine sponges that can be produced synthetically, has significant adulticidal activity against filariae. CDRI compound 92/138, a synthetic analogue of aplysinopsin, also destroys developing larval forms (L3 and L4 stages) of filarial nematodes.

The aggressive treatment of chronic lymphoedema and elephantiasis can lead to a surprising reversal of symptoms. The treatment consists of providing long-term, low-dose DEC (to eradicate persistent or new filarial infections) with diligent attention to the local area of the lymphedematous extremity. The most important local treatments are those measures that prevent superficial bacterial and fungal infection. Additionally, patients should use limb elevation, special massage techniques, and elastic stockings to protect the affected extremity. Patients with severely damaged extremities may benefit remarkably from surgical decompression of the lymphatic system through endovenous shunt surgery followed by excision of redundant tissue. Surgical correction or repeated drainage is the treatment for hydroceles. Surgical correction sometimes is used to correct chyluria. Interestingly, the diagnostic lymphangiography itself often

appears to terminate the leak of chyle into the urine, probably because of its sclerosing effects on the lymphatic vessels that have ruptured into the renal pelvis. Patients with tropical eosinophilia respond dramatically to DEC. Symptoms of untreated patients may resolve spontaneously, but the eosinophilia usually persists, and these patients frequently have recurrences or relapses. Bronchoalveolar lavage reveals that even patients treated with DEC often have persistent mild chronic alveolitis that can cause a mild, chronic, interstitial lung disease. Steroid therapy can be used with anthelminthic therapy to lessen minor allergic reactions. Mebendazole with levamisole is a promising alternative therapy for tropical pulmonary eosinophilia patients who are allergic to DEC. Importantly, in places where onchocerciasis and/or loiasis are likely to be co-endemic (e.g., sub-Saharan Africa, Latin American countries, Yemen), DEC should not be used for treatment of lymphatic filariasis because of the risk of reactions caused by the presence of one or both of the 2 parasites [29, 32].

CONCLUSION: Filariasis is an infection caused by a parasitic worm and is transmitted by insect-bites. It is more prevalent in the tropical areas of Africa, Asia, Central and South America. In India, it is common in eastern Uttar Pradesh and Bihar. Lymphatic filariasis affects more than 120 million people worldwide – over 40 million of these are seriously incapacitated and disfigured by the disease. This disease spreads from person to person by mosquito bites. When a mosquito bites an infected person, microscopic worms circulating in his blood enter and infect the mosquito. These worms then pass to the other person when this infected mosquito bites him. The worms transferred from the mosquito, move through the skin, and travel to lymph vessels, where they grow into adults. An adult worm lives for about seven years. The adult worms mate and release millions of microscopic worms into the blood. There are eight different types of this worm, out of which three are responsible for causing the disease: Wucheria bancrofti and Brugia malayi cause lymphatic filariasis, and Onchocera volvulus causes onchocerciasis (river blindness). The medicationis started at low doses to prevent reactions caused by large numbers of dying parasites. These medications can cause severe side effects in up to 70% of patients. These side effects can be controlled with antihistamines and anti-inflammatory drugs (corticosteroids). Rarely, treatment with diethylcarbamazinemay leads to a fatal inflammation of the brain (encephalitis).

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