LYMPHATIC FILARIASIS

*WUCHERERIA BANCROFTI*

History

Filariasis has been known from antiquity. Elephantiasis had been described in India

by Sushrutha (circa 600 BC) and in Persia by Rhazes and Avicenna. The term ‘Malabar

.leg’ was applied to the condition by Clarke in 1709 in Cochin

Microfilaria was first observed by Demarquay (1863) in the hydrocoele fluid of

a patient from Havana, Cuba. The genus is named after Wucherer, a Brazilian physician

who reported microfilariae in chylous urine in 1868. Microfilaria was first

demonstrated in human blood in Calcutta by Lewis (1872), who called it Filaria

sanguinis hominis. The female adult worm was described by Bancroft (1876) in

Brisbane, Australia and the male worm by Bourne (1888). Manson (1878) in China

identified the Culex mosquito as the vector. This was the first discovery of insect

transmission of a human disease. Manson (1879) also demonstrated the nocturnal

.periodicity of microfilariae in peripheral blood

Distribution

W. bancroftiis distributed widely in the tropics and subtropics of Asia, Africa and

South America (Fig. 18.2). Over 900 million persons live in areas endemic for lymphatic

filariasis and are therefore at risk of infection. In 1999, over 90 million persons were

estimated to be infected, with or without clinical manifestations—over 81 million

.with Wuchereriaand over 8 million with Brugia

The largest number of cases of filariasis occur in India, where over 300 million

people live in endemic zones. It is estimated that at least 6 million attacks of acute

filarial disease occur every year in India and that over 15 million persons have chronic

filarial disease. The endemic areas are mainly along the sea coast and along the



banks of the large rivers, though infection occurs virtually in all states, except in

.the North West

FIGURE 18.2: Geographical distribution of Wuchereria bancrofti

Morphology and Life Cycle

The adults are whitish, translucent, thread-like worms with smooth cuticle and tapering

ends. The female is larger (70-100 × 0.25 mm) than the male (25-40 × 0.1 mm). Males

and females remain coiled together usually in the abdominal and inguinal lymphatics

and in the testicular tissues. The adult worms live for many years, probably 10 to

.years or more ١٥

The worm is ovoviviparous. The embryo (microfilaria) is released encased in its

.elongated egg-shell, which persists as a sheath. The microfilaria has a colourless

translucent body with a blunt headand pointed tail. It measures 250 to 300 µm in

length and 6 to 10 µm in thickness. It is actively motile and can move forwards

and backwards within the sheath, which is much longer than the embryo (Fig.
.(18.3

When stained with Leishman or other Romanowsky stains, structural details can

be made out. Along the central axis of the microfilaria can be seen a column of

granules, which are called somatic cellsor nuclei. The granules are absent at certain

specific locations—a feature which helps in the identification of the species. The specific

.locations are the following

a. At the head end is a clear space devoid of granules, called the cephalic space. In

Microfilaria bancrofti, the cephalic space is as long as it is broad while in M. ,malayi

it is longer than its breadth. With vital stains a styletcan be demonstrated projecting

.from the cephalic space

b. In the anterior half of the microfilaria, is an oblique area devoid of granules called

.the nerve ring

c. Approximately midway along the length of the microfilaria is the anterior V-spot

.which represents the rudimentary excretory system



.FIGURE 18.3: Morphology of Microfilaria bancrofti

;Sheath; 2. Stylet; 3. Cephalic space; 4. Nuclei .١

;Nerve ring; 6. Anterior V-spot; 7. Posterior V-spot .٥

Tail .٨

.d. The posterior V-spot(Tail-spot) represents the cloaca or anal pore

.e. The genital cells (G-cells) situated anterior to the anal pore

f. The internal (central) body of Manson extending from the anterior V-spot to

.G-cell 1, representing the rudimentary ailmentary system

g. The tail tip, devoid of nuclei in Mf.bancrofti, bears two distinct nuclei in Mf. malayi

The microfilariae circulate in the blood stream. In India, China and many other

Asian countries. They show a nocturnal periodicity in peripheral circulation, being

.seen in large numbers in peripheral blood only at night, between 10 pm and 4 am

This correlates with the night biting habit of the vector mosquito. Periodicity may

also be related to the sleeping habits of the hosts. It has been reported that if the

sleeping habits of the hosts are reversed, over a period, the microfilariae change

their periodicity from nocturnal to diurnal. Nocturnal periodic microfilariae are

believed to spend the day time mainly in the capillaries of the lung and kidneys

or in the heart and great vessels. In the Pacific islands and some parts of the Malaysian

archipelago, the microfilariae are non-periodic or diurnal subperiodic, in that they

occur in peripheral circulation at all times, with a slight peak during the late afternoon

.or evening. This is related to the day biting habits of the local vector mosquitoes

Some authors separate the subperiodic Pacific type of W. bancroftias a distinct ) species

.(designated W. pacifica, but this is not widely accepted

.Humans are the definitive host. No animal host or reservoir is known for W

bancrofti. The intermediate host is the female mosquito, different species acting as

vectors in different geographic areas. The major vector in India and most other parts

.(of Asia is Culex quinquefasciatus (C.fatigans

Microfilariae do not multiply or undergo any further development in the human

body. If they are not taken up by a female vector mosquito, they die. Their lifespan

is believed to be about 2 to 3 months. It is estimated that a microfilarial density

of at least 15 per drop of blood is necessary for infecting mosquitoes. Densities of

.microfilariae or more per ml of blood may be seen in some carriers ٢٠،٠٠٠

When a vector mosquito feeds on a carrier, the microfilariae are taken in with

the blood meal and reach the stomach of the mosquito. Within 2 to 6 hours, they



cast off their sheaths (exsheathing), penetrate the stomach wall and within 4 to 17

.hours migrate to the thoracic muscles where they undergo further development

During the next 2 days, they metamorphose into the first-stage larva which is a

sausage-shaped form with a spiky tail, measuring 125-250 × 10-15 µm. Within a ,week

,it moults once or twice, increases in size and becomes the second-stage larvae

measuring 225-325 × 15-30 µm. In another week, it develops its internal structures

and becomes the elongated third-stage filariform larva, measuring 1500-2000 × 15-25 µm. It is actively motile. This is the infective larva. It enters the proboscis sheath

of the mosquito, awaiting opportunity for infecting humans on whom the mosquito

.feeds

There is no multiplication of the microfilaria in the mosquito and one microfilaria

develops into one infective larva only. The time taken from the entry of the microfilaria

into the mosquito till the development of the infective third-stage larva located in

its proboscis sheath, constitutes the extrinsic incuation period. Its duration varies with

environmental factors such as temperature and humidity as well as with the vector

.species. Under optimal conditions, its duration is 10 to 20 days

When a mosquito with infective larvae in its proboscis feeds on a person, the

.larvae get deposited, usually in pairs, on the skin near the puncture site

The larvae enter through the puncture wound or penetrate the skin by .themselves

The infective dose for man is not known, but many larvae fail to penetrate the skin

by themselves and many more are destroyed in the tissues by immunological and

other defence mechanisms. A very large number of infected mosquito bites are

required to ensure transmission to man, perhaps as many as 15,000 infective bites

.per person

After penetrating the skin, the third-stage larvae enter the lymphatic vessels and

are carried usually to abdominal or inguinal lymph nodes, where they develop into

adult forms. There is no multiplication at this stage and only one adult develops

from one larva male or female. They become sexually mature in about 6 months

and mate. The gravid female worm releases large numbers of microfilariae, as many

as 50.000 per day. They pass through the thoracic duct and pulmonary capillaries

.(to the peripheral circulation (Fig. 18.4

.The period from the entry of the infective third-stage larvae into the human host

till the first appearance of microfilariae in circulation is called the biological incubation

period or the prepatent period. This is usually about 8 to 12 months. The period

from the entry of the infective larvae, till the development of the earliest clinical

manifestation is called the clinical incubation period. This is very variable, but is

.usually 8 to 16 months, though it may often be very much longer

Pathogenesis

The outcome of filarial infection varies in different persons. In endemic areas, infection

may be entirely asymptomatic in most persons. Carriers may have very high

microfilarial density in peripheral blood (20,000 per ml or more) without any ill

effects. Such persons appear to tolerate microfilariae, the immune response being



FIGURE 18.4: Life cycle of W. bancrofti. 1. Adult male and female

.in lymph node; 2. Microfilaria in peripheral capillaries at night. 3

.Microfilaria ingested by mosquito reaches its stomach where it; 4

Sheds its sheath, penetrates gut wall and enters thoracic muscles

where it develops into; 5. Short first-stage larva; 6. Second-stage

larva and; 7. Infective third-stage larva which lies in the proboscis

sheath. When the mosquito bites a person, it is deposited on the

skin; 8. Penetrates, reaches lymphatics and develops into adult

inhibited by antigen-specific suppressor cells or other suppressor factors. On the

other hand, in persons coming to endemic areas from places where filariasis is ,absent

infection may cause early clinical manifestations such as lymphangitis and lymphadenitis. They mount an immune response against the infection, so that microfilariae

.may not be demonstrable in them

The infective larvae that enter the human body through mosquito bite migrate

in the lymphatics and moult, during which they release their body proteins, secretions

and other products. In some persons these may cause irritation, directly or due to

.hypersensitivity or other immunological inflammation

Immune reactions are more common when the worms become adults. The typical

manifestations of filariasis are caused by the adult worms blocking lymph nodes

and vessels, either mechanically or more commonly due to allergic inflammatory

reactions to worm antigens and secretions. The affected lymph nodes and vessels

are infiltrated with macrophages, eosinophils, lymphocytes and plasma cells, and

show endothelial hyperplasia. The vessel walls get thickened and the lumen narrowed

or occluded, leading to lymph stasis and dilatation of lymph vessels. The worms

inside lymph nodes and vessels may cause granuloma formation, with subsequent

scarring and even calcifiation. Inflammatory changes damage the valves in lymph

vessels, further aggravating lymph stasis. Increased permeability of lymph vessel

walls leads to leakage of protein-rich lymph into the tissues. This produces the typical

hard pitting or brawny oedema of filariasis. Fibroblasts invade the oedematous

tissues, laying down fibrous tissue, producing the non-pitting gross oedema of

.elephantiasis. Recurrent secondary bacterial infections cause further damage



Animal models have been developed, such as experimental filarial infection in cats with Brugia pahangior Br. malayi.These have helped in understanding the pathogenesis of the disease, but in cats and other animals, filarial infection does not cause elephantiasis. Elephantiasis is a feature unique to human filariasis, apparently caused by human erect posture and consequent hydrodynamic factors affecting lymph .flow

In some persons, immune reactions to filarial antigens may produce clinical conditions unrelated to the lymphatic lesions described above. In these, microfilariae .are not demonstrable in blood. These are known as occult filariasis

Clinical Manifestations

Filariasis leads to a wide spectrum of clinical manifestations, ranging from carrier state with no evident disease to chronic incapacitating illness. Filariasis does not .kill, but may cause great suffering, disfiguration and disability

The earliest manifestations are seen during the stage of 'invasion', when the ,infective larvae enter the body and undergo development. In some persons hypersensitivity to the antigens of the larvae causes constitutional symptoms such as malaise, headache, nausea, vomiting and low grade fever. Recurrent attacks of pruritus and urticaria may occur. Some develop 'fugitive swellings'—raised, ,painless

tender, diffuse, red areas on the skin, commonly seen on the limbs. These disappear

.spontaneously after a few days, but may reappear at the same or different sites

The characteristic manifestations of filariasis are due to obstruction of lymph vessels

and nodes. The essential features are lymphadenopathy, lymphangitis, lymphangiovarix, lymphorrhagia or chylorrhagia, hydrocoele, lymphoedema and .elephantiasis

.Depending on the sites affected, the clinical presentations vary

Lymphadenitis

Repeated episodes of acute lymphadenitis with fever occur very frequently. The

inguinal nodes are most often affected, and axillary nodes less commonly. The swollen

nodes may be painful and tender

Lymphangitis

The acutely inflamed lymph vessels may be seen as red streaks underneath the .skin

Lymphatics of the testes and spermatic cord are frequently involved, with epididymoorchitis and funiculitis. Acute lymphangitis is usually caused by allergic or inflammatory reaction to filarial infection, but may often be associated with streptococcal

.infection also

Filarial Fever

High fever of sudden onset, often with rigor, lasting for two or three days is the

typical picture. This occurs repeatedly at intervals of weeks or months. It is

accompanied by lymphangitis and lymphadenitis, with resultant lymphoedema. When

.FIGURE 18.5: Microfilaria from human blood

Thick drop, haematoxylin staining

Magn. x 700

the lymph nodes affected are intra-abdominal and hence not noticeable, the diagnosis

.may be difficult

Lymphangiovarix

Dilatation of lymph vessels commonly occur in the inguinal, scrotal, testicular and

.abdominal sites

Lymphorrhagia

Rupture of lymph varices leads to the release of lymph or chyle. The clinical picture

,depends on the sites involved and include lymph scrotum, lymphocoele, chyluria

.chylous diarrhoea, chylous ascites and chylothorax

Hydrocoele

This is a very common manifestation of filariasis. Accumulation of fluid occurs due

to obstruction of lymph vessels of the spermatic cord and also by exudation from

,the inflamed testes and epididymis. The fluid is usually clear and straw coloured



but may sometimes be cloudy, milky or haemorrhagic. The hydrocoele may be

unilateral or bilateral and is generally small in size in the early stage, but may

occasionally assume enormous proportions in association with elephantiasis of the

.scrotum. The largest reported hydrocoele weighed over 100 kilograms

Lymphoedema

This follows successive attacks of lymphangitis and usually starts as swelling around

,the ankle, spreading to the back of the foot and leg. It may also affect the arms

breast, scrotum, vulva or any other part of the body. Initially the oedema is pitting

.in nature, but in course of time becomes hard and nonpitting

Elephantiasis

.This is a delayed sequel to repeated lymphangitis, obstruction and lymphoedema

Lymph exudate accumulating in the region stimulates connective tissue hypertrophy

and hyperplasia. The part gets grossly enlarged and misshapen. The skin surface

becomes coarse, with warty excrescences. Cracks and fissures develop with secondary

bacterial infection. Elephantiasis is seen most commonly in the leg, but may also

involve other parts of the body including the arm, breast, scrotum, penis and .vulva

Occult Filariasis

This term is applied to clinical conditions not directly due to lymphatic ,involvement

but to hypersensitivity reactions to filarial antigens. Here microfilariae are not seen

in blood but may be present at the affected sites. The condition may be caused by

.Wuchereria, Brugia or by some animal filaria also

,The best studied syndrome of occult filariasis is Tropical Pulmonary Eosinophilia

which presents with low grade fever,loss of weight, anorexia and pulmonary

symptoms such as dry nocturnal cough, dyspnoea and asthmatic wheezing. Blood

.eosinophil count is above 3000 per cmm and may even go up to 50,000 or more

IgG levels are elevated. Chest radiography shows mottled shadows resembling miliary

tuberculosis. Young adults are more commonly affected. There is considerable

.geographical difference in its incidence, which is probably genetically conditioned

Microfilariae are not usually detectable in blood, but lung biopsies have shown

microfilariae in some cases. It has been suggested that in these cases, there is a failure

in the suppression of immune response to microfilarial antigens, so that microfilariae

.are filtered out and destroyed in the lungs, with allergic inflammatory reaction

Serological tests with filarial antigens are usually strongly positive. Nonspecific

antibody production occurs and biological false-positive reactions are often seen in

.serological tests for syphilis. Prompt response to DEC confirms the diagnosis

,Occult filariasis has also been reported to cause arthritis, glomerulonephritis

thrombophlebitis, tenosynovitis and dermatoses. Endomyocardial fibrosis has been

.claimed to be associated with filariasis, but the relationship has not been proven



Diagnosis

The diagnosis of filariasis depends on the clinical features, history of exposure

.in endemic areas and on laboratory findings

:The laboratory tests that can be used for diagnosis include the following

a. Demonstration of microfilaria in peripheral blood. Microfilaria may also be

.detected in other specimens such as chylous urine or hydrocoele fluid

.Sometimes it can be seen in biopsy specimens

.b. Demonstration of the adult worm in biopsy specimens

.c. Skin tests with filarial antigens

.d. Demonstration of antibody to filarial antigens by serological tests

.e. Demonstration of filarial antigens in blood by serological tests

.f. Indirect evidence such as eosinophilia

Demonstration of microfilaria in the peripheral blood is the diagnostic test most commonly employed. It is also the method used for carrier surveys. It has also the advantage that the species of the infecting filaria can be identified from the

morphology of the microfilaria seen. In India and other areas where the prevalent filarial species is nocturnal periodic, 'night blood' samples are collected between pm and 4 am. Microfilaria can be demonstrated in unstained as well as stained ١٠

.preparations

Unstained Film

From a finger prick, two or three drops of blood are collected on a clean glass ,slide

a cover slip applied and sealed with vaseline. Examination under the low power microscope will show the actively motile microfilariae lashing the blood cells .around

The examination may be conveniently made the next morning as microfilariae retain

.their viability and motility for a day or two at room temperature

Stained Film

A 'thick and thin' blood smear is prepared on a clean glass slide and dried, The

thick part of the smear is dehaemoglobinised by applying distilled water. The smear

is fixed in methanol and stained with Giemsa, Leishman or polychrome methylene

blue stains. Microfilariae may be seen under the low power microscope in the thick

.film. Their morphology can be studied in the thin film

(By using a micropipette for taking a known quantity of blood (20 to 60 cu mm

for preparing the smear and counting the number of microfilariae in the entire stained

.smear, microfilaria counts can be obtained

Concentration Techniques

When the microfilaria density is low, examination of large volumes of blood, 1 ml

or more, gives more positive results. Concentration techniques are used for this

purpose. In the sedimentationmethods, blood is obtained by venepuncture, the red



cells lysed and the microfllariae concentrated by centrifugation. In the filtrationmethods

used at present larger volumes of blood, up to 5 ml can be filtered through millipore

or nucleopore membranes. The membranes may be examined as such or after ,staining

for microfilariae. The filter membrane technique is much more sensitive so that blood

can be collected even during day time for screening. The disadvantages of the

.technique are the cost and the need for venepuncture

DEC Provocation Test

A small dose of diethyl carbamazine (2 mg per kg body weight) induces microfilariae

to appear in peripheral blood even during day time. For surveys, blood samples can be collected 20 to 50 minutes after the administration of one 100 mg tablet of .DEC to adults

Microfilaria may be demonstrated in centrifuged deposits of lymph, chylous urine or other appropriate specimens. Adult filarial worms can be seen in sections of .biopsied lymph nodes, but this is not employed in routine diagnosis

Intradermal injection of filarial antigens (extracts of microfilariae, adult worms and third-stage larvae of Br.malayior of the dog filaria Dirofilaria immitisinduces an immediate hypersensitivity reaction. But the diagnostic value of the skin test is .very limited due to the high rate of false-positive and negative reactions

Several serological tests, including complement fixation, indirect haemagglutination, indirect fluorescent antibody, immunodiffusion and immunoenzyme tests

have been described. But the tests available now are not sufficiently sensitive or specific to be used either for individual diagnosis or surveys. Highly sensitive techniques are now being tried for detection of filarial antigens in blood. These hold

.promise

Prevention and Control

The two major measures in prevention and control of filariasis are eradication of the vector mosquito and detection and treatment of carriers. The recommended ,teatment is diethyl carbamazine (DEC) 6 mg per kg body weight daily for 12 days the drug being given for 2 weeks, 6 days in a week. The treatment may have to

be repeated in endemic areas, every 2 years or so. Mass chemotherpay has been tried, but it may pose difficulties in large endemic areas such as India. As DEC is non-toxic, it can be safely administered in combination with food items such as .common salt

Treatment

DEC is the drug of choice. It is actively microfilaricidal, and in large enough doses may be fatal to adult worms also. Allergic reactions may occur due to the release of antigens from the large numbers of microfilariae which die on administration .of the drug

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BRUGIA MALAYI

The genus Brugia was named after Brug, who in 1927 described a new type of microfilaria in the blood of natives in Sumatra. The adult worm of B.malayiwas described by Rao and Maplestone in India (1940). Besides B. malayi, the genus includes

B. timori, which parasitises humans in Timor, Indonesia and a number of animal .species, such as B. pahangiand B. pateiinfecting dogs and cats

The geographical distribution of B.malayiis much more restricted than that of W. bancrofti. It occurs in India and Far East, Indonesia, Philippines, Malaysia, ,Thailand

Vietnam, China, South Korea and Japan. In India, Kerala is the largest endemic ,area

.particularly the districts of Quilon, Alleppey, Kottayam, Ernakulam and Trichur

Endemic pockets occur in Assam, Orissa, Madhya Pradesh and West Bengal. B.malayi

.and W. bancroftimay be present together in the same endemic area, as in Kerala

In such places, B. malayitends to be predominantly rural and W. bancroftiurban in

.(distribution (Fig. 18.6

FIGURE 18.6: Geographical distribution of

Brugia malayi

The adult worms of B.malayiare generally similar to those of W.bancrofti, though

,smaller in size. The microfilariae are, however, different in a number of respects

Mf.malayiis smaller in size; shows kinks and secondary curves; its cephalic space

is longer; carries double stylets at the anterior end; the nuclear column appears blurred

in Giemsa-stained films; and the tail tip carries two distinct nuclei, one terminal

.(and the other subterminal (Table 18.2, Fig. 18.7

BRUGIA TIMORI

Br. timori is limited to Timor and some other islands of eastern Indonesia. The vector

is Anopheles barbirostrisa night feeder. No animal reservoir is known. The microfilaria

.is larger than Mf. malayi. The sheath of Mf. timorifails to take Giemsa stain



Table 18.2: Distinguishing features of Mf. bancrofti and Mf. malayi

| Features | Mf. bancrofti | Mf. malayi |
|---|---|---|
| Length | 250 to 300 µm | 175 to 230 µm |
| Appearance | Graceful, sweeping curves | Kinky, with secondary curves |
| Cephalic space | Length and breadth equal | Almost twice as long as broad |
| Stylet at enterior end | Single | Double |

Excretory pore Not prominent Prominent

Nuclear column Discrete nuclei Blurred

,Tail tip Pointed: free of nuclei Two distinct nuclei, one at tip

the other subterminal

Sheath Faintly stained Well-stained

The lesions produced by B.timoriare milder than those of bancroftian or malayan

filariasis. A characteristic lesion is the development of draining abscesses caused

by worms in lymph nodes and vessels along the saphenous vein, leading to .scarring

FIGURE 18.7: Differentiating features between Mf. bancrofti

and Mf. malayi. 1. Cephalic space as long as broad and

carries one stylet in bancrofti. It is longer than broad and

,carries two stylets in malayi. 2. Body nuclei round, distinct

well-separated in bancrofti. They are angular, blurred and

.squeezed together in malayi. 3. No nuclei in tail tip in bancrofti

Two widely spaced nuclei at tail tip in malayi. 4. Body curves

,large, regular, smooth in bancrofti. Several small, irregular

. angular kinks in malayi

SUBCUTANEOUS FILARIASIS

LOA LOA

Loa loa known also as the 'African eye worm' or the worm causing loiasis, 'fugitive

swellings' or 'calabar swellings', was first detected in the eye of a patient in West

Indies in 1770. But at present, it is limited to its primary endemic areas in the forests

.of West and Central Africa, where about 10 million people are affected

The adult worm measures about 30 to 70 mm in length and 0.3 to 0.5 mm in

thickness. In infected persons, they live in the subcutaneous tissues, through which

they wander. The microfilariae are sheathed. They appear in peripheral circulation

only during the day (diurnal periodic). The vectors are day biting flies of the genus

.Chrysops, in which the microfilariae develop into the infective third-stge larvae

Infection is transmitted through the bite of infected Chyrysops. Natural infection

.is seen in some African monkeys

.The pathogenesis of loiasis depends on the migratory habit of the adult worm

Their wanderings through subcutaneous tissues set up temporary foci of inflammation, which appear as swellings, of up to 3 cm in size. These are the .calabar swellings

They are called fugitive swellings, because they disappear in a few days, only to reappear

elsewhere. Ocular manifestations occur when the worm reaches the subconjunctival

tissues during its wanderings. The ocular lesions include granulomata in the bulbar

.conjunctiva, painless oedema of the eyelids and proptosis

Diagnosis rests on the appearance of fugitive swelling in persons exposed to

infection in endemic area. The adult worm can be demonstrated by removal from

the skin or conjunctiva. Microfilariae may be shown in peripheral blood collected

.during the day. High eosinophil count is common

Treatment is by surgical removal of the adult worms when they come to accessible

sites. DEC is active against the worm, but has to be used with caution as severe

adverse reactions may develop following the sudden death of large numbers of

microfilariae. Simultaneous administration of corticosteroids minimises such .reactions

FIGURE 18.8: Onchocerca volvulus

ONCHOCERCA VOLVULUS

History and Distribution

Onchocerca volvulus, the ‘convoluted filaria’, or the ‘blinding filaria’ producing

onchocerciasis or ‘river blindness’was first described by Leuckart in 1893. It affects

about 40 million people, mainly in tropical Africa, but also in Central and South

America. A small focus of infection exists in Yemen and south Arabia. Onchocerciasis

.is the second major cause of blindness in the world



Morphology and Life Cycle

The adult worms are seen in nodules in subcutaneous connective tissues of infected

persons. The worms are whitish, opalescent, with transverse striations on the .cuticle

The posterior end is curved, hence the name Onchocerca, which means ‘curved .’tail

The male measures about 30 mm in length and 0.15 mm in thickness, and the female

cm by 0.4 mm. The microfilariae are unsheathed and non-periodic. They ٥٠ measure

about 300 by 0.8 μm. The microfilaria are found typically in the skin and subcutaneous

lymphatics in the vicinity of parent worms. They may also be found in the conjunctiva

.(and rarely in peripheral blood (Fig. 18.8

Humans are the only definitive host. Day-biting female black flies of the genus

Simuliumare the intermediate hosts. They are 'pool feeders' and suck in blood and

tissue fluids. Microfilariae from the skin and lymphatics are ingested and develop

within the vector, becoming the infective third-stage larvae, which migrate to its

mouth parts. The extrinsic incubation period is about 6 days. Infection is transmitted

when an infected Simulium bites a person. The prepatent period in man is 3 to 15

months. The adult worm lives in the human host for about 15 years and the

.microfilariae for about 1 year

The vector Simulium species breed in 'fast-flowing rivers, and, therefore, the

disease is most common along the course of rivers. Hence, the name 'river .'blindness

Pathogenicity

Pathogenesis depends on the host's allergic and inflammatory reactions to the adult

worm and microfilariae. The infective larvae deposited in the skin by the bite of

the vector develop at the site to adult worms. Adult worms are seen singly, in pairs

or in tangled masses in subcutaneous tissues. They may occur in the subcutaneous

nodules or free in the tissues. The subcutaneous nodule or onchocercomais a

circumscribed, firm, non-tender tumour formed as a result of fibroblastic reaction around the worms. Nodules vary in size from a few mm to about 10 cm. They tend ,to occur over anatomical sites where the bones are superficial, such as the scalp scapulae, ribs, elbows, iliac crest, sacrum and knees. The nodules are painless and .cause no trouble except for their unsightly appearance

Microfilariae cause lesions in the skin and eyes. The skin lesion is a dermatitis with pruritus, pigmentation, atrophy and fibrosis. Ocular manifestations range from photophobia to gradual blurring of vision, progressing to total blindness. Ocular ,lesions include punctate or sclerosing keratitis, iridocyclitis, secondary glaucoma .choroidoretinitis and optic atrophy

Diagnosis

The microfilariae may be demonstrated by slicing off a sliver of skin, which is placed .on a slide in water or saline. The specimen is best collected around midday

Microfilariae may also be shown in aspirated material from subcutaneous .nodules

In patients with ocular manifestations, microfilariae may be found in conjunctival .biopsies



Prevention

In 1974, WHO launched a control programme in West Africa using aerial larvicide for vector control and treatment of patients with ivermectin. This is believed to .have prevented blindness in millions of children

Treatment

Enucleation of nodules may reduce the worm burden, but cannot eliminate the

infection. DEC and suramin have been used. DEC destroys microfilariae, but usually

causes an intense reaction (Mazzotti reaction) consisting of pruritus, rash, lymphadenopathy, fever, hypotension and occasionally eye damage. Ivermectin is the

.drug of choice

MANSONELLA STREPTOCERCA

Also known as Acanthocheilonema, Dipetalonemaor Tetrapetalonema streptocerca, this worm

is seen only in West Africa. The adult worms live in the dermis, just under the skin

surface. The unsheathed microfiliariae are found in the skin. Culicoides species are

the vectors. Chimpanzees may act as reservoir hosts. Infection may cause dermatitis

with pruritus and hypopigmented macules. Diagnosis is made by demonstration of

.the microfilariae in skin clippings. DEC is effective in treatment

SEROUS CAVITY FILARIASIS

MANSONELLA OZZARDI

Mansonella ozzardiis a New World filaria seen only in Central and South America

and the West Indies. The adult worms are found in the peritoneal and pleural cavities

.of humans. The non-periodic unsheathed microfilariae are found in the blood

Culicoides species are the vectors. The infection is found mainly in isolated populations

of Amerindians. Infection does not cause any illness. Diagnosis is made by

.demonstrating microfilariae in blood. No treatment is available

MANSONELLA PERSTANS

Also known as Acanthocheilonema, Dipetalonema orTetrapetalonema perstans, this worm

is extensively distributed in tropical Africa and coastal South America. The adult

worms live in the body cavities of humans, mainly in peritoneum, less often in pleura