

Lymphatic Filariasis

Filarial worms are tissue dwelling nematode. The larval stages are inoculated by biting mosquitoes or flies. The larval developing into adult worm (2-5cm long) which after mating, produce millions of microfilariae [120-320 μ m long] that migrate in blood or skin.

- Life cycle is completed when the vector takes up microfilariae while feeding on humans.
- Disease is due to the host's immune response to the worm (both adult and microfilariae) particularly dying worms, and the pattern of severity vary with the site and stages of each species. The worms are long lived, the microfilariae survive 2-3 yrs and adult worms 10-15 yrs. The infection does not subside and host is individuals are constantly exposed to re-infection.

• Adult worms of *Wuchereria bancrofti* and *Brahmari* mainly severely affect lymphatic system. *Wuchereria bancrofti* microfilariae of these two species rarely pathogenic to blood but affects pulmonary capillaries moderately.

• Infection with filarial worms *Wuchereria bancrofti* found in Malaya is associated with clinical outcomes ranging from sub-clinical infection to elephantiasis and hydrocoele. Elephantiasis can arise from infection with *Wuchereria bancrofti* or *Anopheles* transmitted by night biting culicid or *Anopheles* mosquito.

In adult worm 4-10 cm in length, live in lymphatics, and female produce microfilariae which circulate in body fluids in peripheral blood usually at night.

Pathology:

Toxin release by adult worm cause lymphangioec-tasia, this is dilatation of lymphatic vessels leads to lymphatic dysfunction and chronic clinical manifestation of lymphatic filariasis, lymphoedema and hydrocoele.

- Death of the adult worm results in acute filarial lymphangitis. The filariae are symbiotic infected with rickettsia like bacteria [wolbachia] and release of lipopolysaccharides from these bacteria contributes to inflammation.
- Lymphatic obstruction persist after death of all adult worms.
- Secondary bacterial infections cause tissue destruction.

Clinical Features:

- Acute filarial lymphangitis presents with pain, fever, tenderness and erythema along the course of inflamed lymphatic vessels.
- Inflammation of the spermatic cord, epididymis and testes is common.

- It lasts for few days but may recure several times.
- A year later temporary oedema becomes more persistent and regional lymph node enlarge in response.
- Progressive enlargement, coarsening, corrugation, fissuring and bacterial infection of skin and subcutaneous tissues is gradually extending to cause elephantiasis.

Investigation / Diagnosis

- In the earlier stages of lymphangitis, the diagnosis is made on clinical ground, supported by eosinophilia and sometimes by the filarial serology. Filarial infection cause highest eosinophile count.
- Microfilariae can be found in peripheral blood at night, and either are seen moving in a wet blood cell or are detected by microfilaria of a sample of blood.
- Indirect fluorescence and ELISA detect antibody in over 95% of active cases and 50% of established elephantiasis.
- Highly sensitive and specific immunochromatographic card test for detection of filarial infection are now available.

Treatment

- The main aim of the individual is aimed at ceasing and halting disease progression.
- Diethylcarbamazine [DEC] kills microfilariae and adult worms.
- A single dose of either ivermectin or albendazole in combination with diethylcarbamazine also eliminates microfilariae and adult worms (but ivermectin does not prevent further infection).

Prevention

- Vector control
- Treatment of mosquito population in endemic areas with annual single dose DEC also alongside with vector control with ivermectin and albendazole from reduces the parasite transmission.

Japanese encephalitis

This flavivirus is an important cause of encephalitis in Japan, Russia, India etc. Pigs and aquatic birds are the virus reservoirs and transmission is by mosquitoes. Exposure to rice paddies is a recognised risk factor.

Clinical features:

The incubation period is 4-21 days. Most infections are probably subclinical in childhood and only around 1% of infections leads to encephalitis.

Encephalitis

There is an initial systematic illness with fever, malaise and anorexia, followed by photophobia, vomiting, headache and changes in brain-stem functions.

- Neurological features from encephalitis include meningitis, seizure, cranial nerve palsies, flaccid paraparesis and extrapyramidal features.
- Approximately half of survivors are left with neurological sequelae.

Investigation, Treatment and Prevention:

Treatment is supportive and often hypotonaemic. CSF (cerebrospinal fluid) reveals lymphocytosis and elevated protein. serological testing may be helpful and there is CSF antigen test.

- Treatment is supportive, anticipating and treating complications.
- Vaccination is available for endemic areas during the monsoon period. Effective prophylaxis in some endemic countries includes this vaccination in their childhood schedules.

Amoebiasis

It is caused by Entamoeba histolytica which is spread b/w humans by its cysts. E. histolytica also causes amoebic dysentery or liver abscess.

Pathology:

Cysts of E. histolytica are ingested in water or uncooked foods contaminated by human faeces. Infection may also be acquired through anal-oral sexual practices. In the colon, vegetative trophozoites form emerge from the cysts. The parasite may invade the mucous membrane of the large bowel producing lesions. These are flask-shaped ulcers varying greatly in size and surrounded by healthy mucosa. Shallow amoebic ulcers may cause severe haemorrhage but rarely perforate the bowel wall.

Amoebic trophozoites can emerge from the vegetative cyst from the bowel and be carried to the liver in a portal venule. They can rapidly destroy the liver parenchyma, causing an abscess. The liquid contents at first have a characteristic pinkish colour which may later change to chocolate brown.

Cutaneous amoebiasis, though rare, causes pyoderma, genital ulcers, peri-oral or peri-abdominal ulcers, wound ulceration and ulcers on the fingers and toes.

Clinical features: - Sporadic liver abscesses
Intestinal amoebiasis - amoebic dysentery
 Intestinal amoebic infections are asymptomatic in the

incubation period of amoebiasis ranges from 2 weeks to many yrs, followed by a chronic course with abdominal pain and so on more unformed stool a day. Offensive diarrhoea alternating with constipation, and blood or mucus in the stool are common. There may be abdominal pain.

Amoebic liver abscess -

This abscess is usually found in right hepatic lobe. There may not be associated diarrhoea. Early symptoms may be local discomfort only and malaise; later, a swinging temperature and sweating may develop, usually without marked systemic symptoms or associated cardio-vascular signs. An enlarged, tender liver, cough and pain in the right shoulder are characteristic, but symptoms may remain vague and signs minimal. Test -

Investigation - Stool and any sputum should be examined at once under microscope for motile trophozoites containing red blood cells. Sigmoidoscopy may reveal typical flask-shaped ulcers, which should be scraped and examined immediately for E. histolytica.

In endemic areas $1/3^{rd}$ of the population are symptom less possessors of amoebic dysentery. Serum antibodies are detected by immunofluorescence in over 95% of patients with hepatic amoebiasis and intestinal amoebiasis but only about 60% of dysenteric amoebiasis.

Treatment:

Intestinal and early hepatic amoebiasis responds quickly to oral metronidazole or other long acting nitroimidazoles like tinidazole or ornidazole. Nitrooxazanide is an alternative drug.

Prevention:

we should not eat fresh uncooked vegetables or drink uncleaned water.
(man's notes)

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