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Lymphatic Filariasis

Filarial worms are tissue dwelling nematodes. The larval stages are inoculated by biting mosquitoes or flies. The larvae developing into adult worm (2-50cm long) which after mating, produce millions of microfilariae [170 - 320 μm 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 its pattern and severity vary with the site and species of each. The worms are long lived, the microfilariae survive 2-3 yrs and adult worms 10-15 yrs. The infections are chronic and persist in individuals constantly exposed to re-infection.
- Adult worm of Wuchereria bancrofti and Brugia malayi severely affect lymphatics & vessels and microfilariae of these two exist rarely pathogenic to blood but affects pulmonary capillaries moderately.

Infection with filarial worms Wuchereria bancrofti, Brugia malayi is associated with clinical outcomes ranging from asymptomatic infection to lymphatic hydrocele and elephantiasis.

W. bancrofti is transmitted by night biting culicine or Anopheline mosquito in most areas.

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Adult worm 4-10 cm in length, live in lymphatics, and female produce microfilariae which circulate in large nos in peripheral blood usually at night.

Pathology :

Toxin release by adult worm cause lymphangiectasia, this dilatation of lymphatic vesseles leads to lymphatic disfunction and chronic clinical manifestation of lymphatic filariasis, lymphoedema and hydrocele.

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

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 recurs several times in a year.
- Temporary oedema becomes more persistent and regional lymph nodes enlarge.
- Progressive enlargement, coarsening, corrugation, fissuring and bacterial infection of skin and subcutaneous tissue result in gradually advancing irreversible "elephantiasis".

Investigation / Diagnosis

- In the earlier stages of lymphangitis, the diagnosis is made on clinical ground; supported by eosinophilia and sometimes by true filarial serology. Filarial infection cause highest eosinophile count of
- Microfilariae can be found in peripheral blood at night, and either are seen moving in a wet blood cell or are detected by microfil= teration of a sample of lysed blood.
- Indirect fluorescence and ELISA detect antibody in over 95% of active cases and 70% of establish elephantiasis
- Highly sensitive and specific immunochromatographic card test for detection of filarial infection are now available.

Treatment

- Clean the lymph with antiseptic
- Give oral antibiotic for at least
- Treatment of the individual is aimed at reversing and Halting disease progression.
- Diethyl carbamazine [DEC] kills microfilariae and adult worms.
- A single dose of either ivermectin or albendazole in combination with DEC also eliminates microfilaria for a year.

Prevention

- Vector control
- Treatment of whole population in endemic areas with annual single dose DEC either alone or in combination with ivermectin or albendazole can reduce or eliminate infection.

Japanese encephalitis

This flavivirus is an impt 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 4-24 days. most infections are probably subclinical in childhood and only around 1% of infections leads to encephalitis

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

- Neurological features other than encephalitis include meningitis, seizures, cranial nerve palsies, flaccid or spastic paralysis and extrapyramidal features
- Approximately, half of survivors are left with neurological sequelae

Investigation, Treatment and Prevention :

- There is neutrophilia and often hyponatraemia. 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 for travellers to endemic areas during the monsoon period is effective prophylaxis. some endemic countries include 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 dysentry 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 forms emerge from the cysts. The parasite may invade the mucus membrane of the large bowel producing lesions. These are flask-shaped ulcers, varying greatly in size and surrounded by healthy mucosa. Amoebic ulcers may cause severe haemorrhage but barely 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 multiply rapidly and 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 progressive genital, peri-anal or peri-abdominal surgical wound ulceration.

Clinical features :-

Intestinal amoebiasis - amoebic dysentery

Most amoebic infections are asymptomatic. The

incubation period of amoebiasis ranges from 2 weeks to many yrs, followed by a chronic course with abdominal pain and 2 or 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:-

The 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.

6. Investigations:-

Stools and any exudate 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 1/3rd of the population are symptom less passers of amoebic cysts.

Serum antibodies are detected by immuno-flourescence in over 95% of patients with hepatic amoebiasis and intestinal amoebiasis but in only about 60% of dysenteric amoebiasis.

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Treatment :

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

Precaution :

We should not eat fresh uncooked vegetables or drink uncleaned water.

(mam's notes)