Systemic Manifestations of Parasitic Diseases

N. R. Humaney

ABSTRACT

Parasitic infestations are a major health problem world wide, and they account for millions of infestations and deaths each year. Most of the infestations and the morbidity and mortality from these diseases occur in the developing world in rural areas. These parasitic infestations have protean manifestations and consequences. The medical problems range from chronic asymptomatic carrier stage to fulminant disease and even death. Other factors such as host immune status, the infecting organisms and the availability of treatment all play a key role in the outcome of parasitic colitides.[1]

Parasites are the creatures that invade a host, attach themselves externally and internally (to tissue and organs) and rob the host of nutrients. Some worms eventually weaken and cause disease in their gracious host. Helminths infestations interrupt the quality of life and some times cause serious disease.

INTRODUCTION

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Helminths (from Greek Helminths meaning worm) are worm like parasites. These are multicellular and have bilaterally symmetrical, elongated, flat or round bodies. Based on their shape helminths are classified into two broad groups: 1) Cylindrical worm (Nematodes) 2) Flat worm (Platyhelminths). The flat worms are again classified into 2 categories 1) Leaf like (Trematode) or Fluke like and 2) Tape like (Cestodes). Systemic manifestations depend on organ involvement by the worm Infestations.[2]

Clinically important parasitic infestations and their systemic manifestations are discussed below.

CESTODES[3] : Cestode species that most commonly cause human infections are as follows.

Address for correspondence

Associate Professor,
Department of Medicine,
NKPSIMS and LMH, Nagpur

1) Diphyllobothrium Latum:- Infection is acquired by ingestion of parasitic cysts in tissue of smoked or uncooked fresh water fish

Clinical features: Patients with D. Latum infection have few or no symptoms; non-specific complaints like weakness, dizziness, craving for salt, diarrhoea and abdominal discomfort may be present. Some patient may experience vomiting, severe abdominal pain and weight loss. In cases of multiple infections, biliary or intestinal obstruction may occur. 1 to 2% of patients with D. Latum infection develop significant vit B_{12} deficiency, resulting in megaloblastic anaemia or neurologic disease. Folate deficiency may also occur. Vitamin B_{12} deficiency may occur due to extensive vitamin uptake by worm and/or by worm-induced interference with gastrointestinal uptake by host despite normal gastric acidity and intrinsic factor production. Vitamin B_{12} deficiency is most common among older patients with low dietary intake of vitamins and multiple tapeworms in the proximal jejunum. In the debilitated host, nervous system complications can be quite extensive and can range from peripheral neuropathy to syndrome of severe combined degeneration.[3]

2) Taenia Saginata (Beef tapeworm): Infection is acquired by consuming cyticerci in the muscle tissues of infected cattle.

Clinical features: T. Saginata infection may cause non-specific complaints of weakness and mild abdominal discomfort in up to one third of patients. Because its proglottides are motile, they may cause acute abdominal symptoms by migrating into and obstructing the appendix or pancreatic and biliary ducts. A psychologically distressing feature of infection (and often the first symptom reported by the patients) occurs when motile proglottides migrate out of the anus onto skin or clothing or when they are observed moving in the faeces.

3) Taenia Solium (Pork worm):

Causes human infection in two different forms. Individuals who consume undercooked pork containing cyst develop intestinal tapeworms. Individuals who consume eggs may develop cysts within tissues of the body (cysticercosis). Intestinal tapeworms produce no or minimal symptoms which are limited to mild, non-specific abdominal complaints.

In cysticercosis, clinical manifestations depend on the location and number of infecting cysts. Neurocysticercosis may be divided into discrete syndromes.

1. Acute invasive stage:- Patients may experience fever, headache, and myalgias associated with peripheral eosinophilia. Heavy infection results in cysticercal encephalitis.

2. Parenchymal CNS cysticercosis (50% cases): - May be associated with seizures (focal/generalised) intellectual impairment and personality changes.

3. Subarachnoid cysticercosis (30% cases): - May be associated with obstruction to CSF flow, may present with raised intracranial tension, sensorial changes and emotional disturbances.

4.Ventricular disease:- Intraventricular cysticercosis occurs in about 15% of cases[4] and can cause diagnostic difficulties. They give rise to episodes of obstructive hydrocephalus, which may spontaneously remit and recur as a result of the ball-valve effect of cysticerci intermittently occluding the ventricular outlet foramina. Untreated, however, most cases will progress to sustained hydrocephalus.

5. Spinal disease:- A variety of spinal cord syndromes has been reported in association with cysts in and around the cord and the cauda equina. The most common presentation is of progressive paraplegia developing over a period of weeks.

6. Disappearing lesions:- There have been reports, mainly from India, of single small enhancing lesions on computed tomography (CT), seen commonly in patients presenting for the first time with epilepsy but disappearing within a few months of follow-up. Many causes have been suggested for these lesions, including tuberculosis, but a series of excision biopsies have shown that cysticerci are most often responsible.

7. Ocular cysticercosis:- The parasite appears to have a tropism for the eye, although estimates of the incidence of ocular disease vary widely. Shanchez Fontan has described a series of 70 cases of ocular disease from Mexico 4. The great majority of cysts were sub retinal or in the vitreous humour, but they could occur at any site. The initial presentation is most often as a scotoma but, if the inflammatory reaction is marked, vision may be lost.

Trematodes

Schistosomiasis (Bilharziasis):[4]

Clinical features: Divided into acute and chronic stages.

a. Schistosome dermatitis (swimmers itch)

b. Acute schistosomiasis: fever, chills, liver and spleen enlargement and malaise, diarrhoea, wt loss, cough, dyspnoea, chest pain, pericarditis are important findings. High values of circulating immune complexes are found. Acute disease is more frequently observed in individuals living outside the endemic area.

c. Chronic schistosomiasis: Tissue injury is mediated by egg induced granulomas and subsequent appearance of fibrosis. The symptoms and signs depend upon the intensity of infection and the species of infecting

schistosome. S. haematobium infection manifest with painless hematuria, bladder neck obstruction, and recurrent urinary tract infection. S.mansoni infection manifests with abdominal pain, malaise, hepato-splenomegally (because of fibrosis), portal hypertension (splenomegaly). Hepatomegaly is fibrotic rather than cirrhotic. S.japonicum manifestations resemble those of S. mansoni.

Flukes [5]

1. Liver flukes:

Opisthorchiasis: Infected individual may be asymptomatic or symptomatic with right upper quadrant discomfort, dyspnoea, change in bowel habits, cholecystitis, gallstones or obstructive jaundice.

Clonorchiasis: Specificity of symptoms such as anorexia, epigatric pain, diarrhoea

Chronic symptoms like cholangitis, gallstones, cholangiocarcinoma.

2. Intestinal flukes:

Fasciolopsiasis

Echinostomiasis

Heterophyasis

Fasciolopsiasis: The giant intestinal flukes attach to the mucosa of small intestine (duodenum, jejunum). Most individuals are asymptomatic but heavy infection causes intestinal obstruction and protein losing enteropathy. Abdominal pain and diarrhoea causing oedema and anasarca due to hypoalbuminemia may occur.

Echinostomiasis: They present with diarrhoea and abdominal pain.

3. Lung flukes [paragonimiasis]: Features of Acute Paragonimiasis include diarrhoea, abdominal pain, fever, malaise associated with cough, dyspnoea, night sweats. Lung parenchyma demonstrates haemorrhages; established pulmonary stress results in mild chronic cough with production of mucoid, rusty brown sputum. Haemoptysis that may be severe and life threatening occurs rarely.

Extra pulmonary Paragonimiasis commonly affects tissues in brain, abdominal organs and skin.

Acute cerebral Paragonimiasis causes fever, headache, visual disturbances paralysis, generalised or focal disturbances.

Chronic cerebral paragonimiasis causes space-occupying lesion causing epilepsy or paralysis.

Abdominal and cutaneous Paragonimiasis results in space occupying lesion, abscess or migratory swellings.

Nematodes (Round worms) [6]

Intestinal Nematodes:

Ancylostomiasis (Hook Worm disease):

Dermatitis, usually on the feet (ground itch), may be experienced at the time of infection. The passage of the larvae through the lungs in a heavy infection causes a paroxysmal cough with blood stained sputum, associated with patchy pulmonary consolidation. When the worms have reached the small intestine, vomiting and epigastria pain resembling peptic ulcer disease may occur. Sometimes frequent loose stool are passed. Iron deficiency anaemia, protein loosing enteropathy and hypoproteinaemia may develop in the undernourished. High output cardiac failure may result from the chronic iron deficiency anaemia. The mental and physical development of children may be retarded. A well nourished child with light infection may be asymptomatic.

Strongyloidiasis[7]

Clinical features:

Penetration of skin by infective larvae produces itchy rash.

Presence of worm in gut causes abdominal pain, diarrhoea, steatorrhoea, and weight loss.

Allergic phenomena like urticarial plaques and papules, wheezing and arthralgia may occur.

Autoinfection produces transient itchy linear urticarial weal across abdomen and buttocks (larva currens)

Systemic (super) infection produces diarrhoea, pneumonia, meningoenchephylitis and death.

Ascaris Lumbricoides (Round worm):

Intestinal ascariasis causes symptoms ranging

from occasional vague abdominal pain to malnutrition. The large size of adult worm and its tendency to aggregate and migrate can result in sever obstructive complications. In endemic areas it causes up to 35% of all intestinal obstruction, most commonly in the terminal ileum. Obstruction can be further complicated by intussusceptions, volvulus, haemorrhagic infarction and perforation. Other features include bile, pancreatic duct or appendicular obstruction due to adult worms.

Enterobius vermicularis (Threadworm):

(Anal itching) The gravid female worm lays ova around anus, causing intense itching, especially in night. The ova often carried to mouth on fingers and so reinfection takes place. In females the genitalia may be involved. The adult worm may be seen on buttocks or in stool.

Trichuris Trichiura (Whipworm):[7]

Infection is contracted by the ingestion of earth or food contaminated with ova, which have become infective after lying for 3 weeks or more in moist soil. Whipworm inhabits the caecum, lower ileum, appendix, colon and anal canal. There are usually no symptoms, but intense infection in children may cause persistent diarrhoea or rectal prolapse, and stunting of growth.

Thus systemic manifestations of parasitic diseases can be variable and need to be looked for.

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