CHAPTER 23
Parasitic Infestations of Surgical Importance in Children

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Introduction

The term "parasitic infestation" is used to refer to those infections caused by protozoa, helminthes, and arthropods. They are a major cause of morbidity and mortality in infants and children. However, parasitic infestations have received relatively little attention compared with infections due to viral, bacterial, and fungal agents.

Parasitic infestations are a worldwide problem in children, the prevalence and variety of organisms being greatest in areas with a warm, moist climate and in communities where standards of hygiene are low. But parasitic diseases are now occurring more frequently in developed countries due to immigration and increased foreign travel. This cosmopolitan distribution, in addition to the complications that often attend these infestations, make this subject an important surgical problem.

Of the parasitic diseases, those of surgical interest in children are:

1. Protozoan infections
   - Amoebiasis
2. Helminthic infections
   - *Ascaris lumbricoides* – Intestinal nematode
   - Dracontiasis (Dracunculiasis) – Tissue nematode
   - Malayan and Bancroftian filariae – Tissue nematode
   - *Schistosomiasis* (Blood fluke) – Trematode
3. Arthropodal infections
   - Myiasis – Tissue-invading arthropods
   - Chigoe (jigger) – *Tunga penetrans*
4. Hydatid disease

Amoebiasis

Amoebiasis is a human intestinal infectious disease caused by the protozoan parasite *Entamoeba histolytica*. It is a ubiquitous parasitic infection affecting approximately 10% of the world's population and is the third most common cause of death from parasitic infections, the first two being malaria and schistosomiasis.

Demographics

Amoebic infections were previously reported as uncommon among children by the World Health Organization (WHO), which described *shigella* species as the most common and most important cause of dysentery in this age group. However, recent studies from Africa have shown that this condition is endemic, both in its invasive and noninvasive (carrier) states; it may affect any age group and has no gender preference in children. The reasons thought to be responsible for the endemism include poverty, malnutrition, and poor sanitation, among others. Amoebiasis is not uncommon even in some Western countries, however, as a result of immigration and increased foreign travel.

Aetiology/Pathophysiology

Amoebiasis is caused by the pathogenic *Entamoeba histolytica*, commonly transmitted via the faeco-oral route when water or food contaminated by faeces are consumed. Humans are the only reservoir and there are no intermediate hosts.

Upon ingestion of contaminated food or water, the cysts travel to the small intestine, where trophozoites are released (encystation). In 90% of patients, the trophozoites re-encyst and produce asymptomatic infection, which usually spontaneously resolves within 12 months. In the remaining 10% of patients who are infected, the parasite causes symptomatic amoebiasis.

E. histolytica causes its primary lesion in the colon, where the caecum and rectosigmoid are areas of predilection. The incubation period varies from 2 days to 4 months. Invasive disease begins with the adherence of *E histolytica* to colonic mucins, epithelial cells, and leukocytes mediated by a galactose-inhibitable adherence lectin. Following adherence, trophozoites invade the colonic epithelium to produce the ulcerative lesions typical of intestinal amoebiasis. The trophozoites of *E. histolytica* lyse the target cells by using lectin to bind to the target cells' membranes and the parasite's ionophore-like protein to induce a leak of ions (i.e., Na^+, K^+, Ca^{2+}) from the target cells' cytoplasm. Numerous haemolysins, encoded by plasmid (ribosomal) DNA (rDNA) and cytotoxic to the intestinal mucosal cells, have been described in *E. histolytica*. An extracellular cysteine kinase causes proteolytic destruction of tissue, producing flask-shaped ulcers. Phorbol esters and protein kinase C activators augment the cytolytic activity of the parasite.

Liver abscesses due to amoebiasis are 10 times more frequent in adults than in children. Amoebic liver abscess, however, is equally common in both sexes among prepubertal children, probably, in agreement with the equal distribution of intestinal disease in both sexes in children. Spread of amoebiasis to the liver occurs via the portal blood, after the pathogenic organisms have evaded the complement-mediated lysis in the bloodstream. Trophozoites ascend the portal veins to produce liver abscesses filled with acellular proteinaceous debris (so-called anchovy paste). The trophozoites of *E. histolytica* lyse the hepatocytes and the neutrophils. This explains the paucity of inflammatory cells within the liver abscesses. The neutrophil toxins may contribute to hepatocyte necrosis.

Clinical Presentation

The clinical presentation of amoebiasis is variable. It ranges from asymptomatic cyst passage to amoebic colitis, amoebic dysentery, amoeboma, and extraintestinal disease. *E. histolytica* infection is asymptomatic in about 90% of cases; invasive disease occurs in the remaining 10%. Severe disease is more common in children, especially if malnourished. Extraintestinal disease usually involves only the liver, but rare extraintestinal manifestations include amoebic brain abscess, pleuropulmonary disease, ulcerative skin, and genitourinary lesions.

Amoebic Colitis

Amoebic colitis affects all age groups, but its incidence is strikingly high in children 1–5 years of age. The clinical features depend upon the transmural as well as the longitudinal extent of the disease. The onset may be insidious, with nonspecific dysenteric symptoms, and is often confused with gastroenteritis or herbal intoxication. Severe amoebic colitis in infants and young children tends to be rapidly progressive with frequent extraintestinal involvement and high mortality rates. Rectal loss of blood and mucus is a frequent but not constant finding and may raise suspicion of intussusception or typhoid. The association between progressive disease and clinically overt malnutrition is striking, and the relationship may be provocative. The passage of large volumes of malodourous stools with slough from the mucosa in a child with preexisting malnutrition suggests amoebic colitis. Occasionally, amoebic dysentery is associated with sudden onset of fever, chills, and severe diarrhoea, which may result in dehydration and electrolyte disturbances.

Progressive disease in children is manifested by increasing abdominal distention with discomfort, tenderness, and toxaemia. Classical signs of peritonitis may develop very late, if at all, due to omental wrap.

Amoebic Liver Abscess

Amoebic liver abscess, a serious manifestation of disseminated infection, is uncommon in children, although some cases have been reported. Although diffuse liver enlargement has been associated with intestinal amoebiasis, liver abscess occurs in <1% of infected individuals and may appear in patients with no clear history of intestinal disease. This contrasts with the high incidence of cases of amoebic liver abscess (61%) seen in the surgical ward in Natal, South Africa, which occurred in association with active amoebic colitis.

Numerous small abscesses may coalesce to form large abscesses, which expand towards the surface and may rupture, giving rise to amoebic peritonitis. Amoebic liver abscess may occur months to years after exposure, so a high index of suspicion is very important. In children, fever is the hallmark of amoebic liver abscess and is frequently associated with abdominal pain, distention, and enlargement and tenderness of the liver. Changes at the base of the right lung, such as elevation of the diaphragm and atelectasis or effusion, may also occur.

Investigations

Stool examination

Light microscopy examination of a fresh stool smear for trophozoites that contain ingested red blood cells (RBCs) is rather insensitive. It is positive in 10% of patients, showing the presence of haematogenous amoebae. It cannot distinguish other species of *Entamoeba* from *E. histolytica*. Fulminant amoebic colitis or its complications may exist with a negative stool parasitology if treatment has started prior to referral. Stools for examination must be fresh when examined or be preserved in polyvinyl alcohol for later microscopy. Material from rectal scrapings has also proved most helpful. An enzyme immunoassay kit to specifically detect *E. histolytica* in fresh stool specimens is now commercially available in specialised centres.

Serologic studies

Serum antibodies against amoebae are present in 70–90% of individuals with symptomatic intestinal *E. histolytica* infection. Antiamoebic antibodies are present in as many as 99% of individuals with liver abscess who have been symptomatic for longer than a week. However, serologic tests do not distinguish new from past infection because the seropositivity persists for years after an acute infection. Several methods, such as indirect haemoagglutination antibody (IHA), enzyme-linked immunosorbent assay (EIA), and immunodiffusion (ID) tests are now commercially available in specialised centres.

Imaging studies

- *Chest radiography* may reveal an elevated right hemidiaphragm and a right-sided pleural effusion in patients with amoebic liver abscess.
- *Ultrasonography* is preferred for the evaluation of amoebic liver abscess due to its low cost, rapidity, and lack of adverse effects. A single lesion is usually seen in the posterosuperior aspect of the right lobe of the liver. Multiple abscesses may occur in some patients.
- *Computed tomography* (CT) and *magnetic resonance imaging* (MRI) may be done in selected cases.

Other tests

- Leucocytosis without oesinophilia is observed is 80% of cases.
- Mild anaemia may be noted.
- Liver function tests reveal elevated alkaline phosphatase levels (in 80% of patients), elevated transaminase levels, mild elevation of serum bilirubin level, and reduced albumin levels.
- The erythrocyte sedimentation rate is elevated.

Medical Treatment

Asymptomatic infections are not treated in endemic areas. However, in nonendemic areas asymptomatic infection should be treated because of its potential to progress to invasive disease. Luminal agents that are minimally absorbed by the gastrointestinal (GI) tract (e.g., paromomycin) are best suited for such therapy.

Metronidazole is the mainstay of therapy for invasive amoebiasis. Tinidazole is being used for intestinal or extraintestinal amoebiasis. Nitroimidazole therapy leads to clinical response in approximately 90% of patients with mild to moderate colitis. Chloroquine has also been used for patients with hepatic amoebiasis. Intraluminal parasites are not affected by nitroimidazole therapy. Therefore, nitroimidazole therapy should be followed by treatment with a luminal agent such as paromomycin to prevent a relapse.

Broad-spectrum antibiotics may be added to treat bacterial superinfection in a case of fulminant amoebic colitis and suspected perforation. Bacterial coinfection with amoebic liver abscess has occasionally been observed (both before and as a complication of drainage), and adding antibiotics to the treatment regime is reasonable in the absence of a prompt response to nitroimidazole therapy.

Surgical Treatment

Surgical intervention is required for acute abdomen due to perforated amoebic colitis, massive GI bleeding, or toxic megacolon. Toxic megacolon is rare, however. Surgical attempts to correct amoebic bowel perforation or peritonitis should be avoided, although some patients may benefit from peritoneal lavage.

Unlike pyogenic liver abscess, amoebic liver abscess generally responds to medical therapy alone, and drainage is seldom necessary. When necessary, imaging guided percutaneous treatment (needle aspiration or catheter drainage) has replaced surgical intervention as the procedure of choice for reducing the size of an abscess. The indications for drainage of amoebic liver abscess include the presence of left-lobe abscess (>10 cm in diameter), and impending rupture and abscess that does not respond to medical therapy within 3 to 5 days.

Ascariasis

Ascariasis is the parasitic infestation by the largest intestinal nematode of man, which is found worldwide. It is now a significant public health problem in many parts of the world. The organism maintains an ideal host-parasite relationship without any observable harm in the vast majority of individuals, but heavy parasitisation of the intestinal tract by *Ascaris lumbricoides* may be associated with nutritional disturbances and, more important, intestinal obstruction or perforation.

Demographics

Ascariasis is a common problem in the tropics and subtropics, where the moist humid climates of alternating dry season and rainy season permit all-year embryonation of the ova of *Ascaris lumbricoides*. This is further aggravated by the poor environmental standards, improper disposal of sewage, and low socioeconomic conditions prevailing in most cities in Africa.

Although it occurs at all ages, ascariasis is most common in children 2 to 10 years of age; the prevalence decreases after the age of 15 years. The incidence is higher in males than females, probably because they are more exposed to outdoor activities. Infants may be infested soon after birth, the mother transmitting the ova with her dirty fingers. In developing counties with poor sanitary conditions, more than 70% of children are infested, and globally more than 1.5 billion people are infested with *Ascaris lumbricoides*.

Aetiology/Pathophysiology

Ascariasis is caused by *Ascaris lumbricoides*, a large lumen-dwelling nematode contracted by the consumption of its eggs. Transmission occurs mainly via ingestion of water or food contaminated with these eggs from human faeces and occasionally via inhalation of polluted dust. Children playing in contaminated soil may acquire the parasite from their dirty hands**.** Transplacental migration of larvae has also occasionally been reported.

The eggs reach the small intestine, where the larvae are liberated. The larvae penetrate the small intestinal wall and migrate through the lymphatics and bloodstream to the liver, and then to the lungs, where they enter the alveoli. There they pause for at least 2–3 weeks and molt, giving rise to allergic bronchopneumonia in previously infected and sensitised individuals. Later, they wander up the bronchi and trachea, giving rise to bronchitis with bronchospasm and urticaria and occasionally larvae in the sputum. Most larvae are swallowed and grow to adulthood in the small intestine. Adult worms do not multiply in the human host, so the number of adult worms per infested person relates to the degree of continued exposure to infectious eggs over time.

The adult worms give rise to mechanical problems due to their size and the smaller diameter of the lumen of the bowel of children. Also, due to their large number and mass, they lead to a severe nutritional drain in these patients. A temperature elevation to 39°C, certain drugs, such as antihelminthic, and some unknown influences cause the worms to congregate, sometimes resulting in intestinal obstruction (Figure 23.1) and migration out of the gut into the bile duct, oesophagus, mouth, pancreatic duct, or appendix, and occasionally the liver. Adult worms may perforate the gut, leading to peritonitis. Sometimes, the presence and activity of large numbers of worms alone may be associated with vomiting, fever, and abdominal pain. By far, small intestinal obstruction (whether simple occlusive, intussusception, or volvulus) accounts for many of the serious pathologic effects attributed to this worm.

Clinical Presentation

The presentation of ascariasis may be straightforward. Early symptoms may be related to the larval migration in the lung. In established cases, the child may be malnourished. Worms may have been vomited out or passed rectally. The difficulty, however, is in clinching the diagnosis of intestinal obstruction as a result of ascaris worms. There is, therefore, need for a high index of suspicion in all cases of intestinal obstruction in children. A history of a recent purgative will be important, since these have been known to precipitate obstructions.

Among other presentations, pyrexia of moderate degree may be observed; colicky central abdominal pain may be the chief complaint; vomiting may be frequent, either due to the activity of the worms or as a result of actual obstruction; the abdomen may be generally tender; and in half the cases, an abdominal mass that is ill-defined, mobile, and sometimes multiple and commonly situated in the umbilical region may be palpable.

Figure 23.1: Exceptional ascaris burden causing acute intestinal obstruction.

Eosinophilia is present in the early phases of infestation, but due to the mixture of parasitic infestations present at the same time, it is not diagnostic.

Investigations

Erect plain abdominal x-ray

Radiographs are useful in heavily infested children where the worms appear radiolucent. A mass of worms may contrast against the gas in the bowel, typically producing a "whirlpool" effect. The radiographs also show features of intestinal obstruction, such as abdominal distention, dilated bowel loops, and multiple air fluid levels and free gas under the diaphragm in cases with intestinal perforation.

Ultrasonography

Ultrasonography may be helpful, with the round worm appearing sonographically as a thick echogenic strip with a central anechoic tube or multiple long, linear, parallel echogenic strips without acoustic shadowing. Curling movements of the worms may be observed on prolonged scanning.

Stool examination for ova

This is not helpful where infestation rates are high.

Treatment

Children with uncomplicated ascariasis are managed as paediatric outpatients and rarely referred to the surgeon. However, following intestinal obstruction due to ascariasis, the various options in management are as follows:

Conservative approach

Various authors have recorded a high success rate with a conservative approach. They observed that, unlike other mechanical causes of intestinal obstruction, most cases of acute intestinal obstruction due to ascariasis can be managed conservatively. This approach is, however, most suitable for mild cases with partial obstruction; it entails decompression of the bowel, intravenous fluid replacement, antispasmodics, and anthelmintic administered after the attack has subsided.

Surgical approach

Complete obstruction should be relieved surgically after resuscitation of the patient by any of the following methods:

- *Milking:* The bolus of worms is broken up and massaged into the larger diameter caecum and ascending colon.
- *Enterostomy:* The antimesenteric border of the bowel is opened, through which the worms are carefully extracted and the resulting opening repaired transversely in two layers.

- *Resection:* The affected bowel segment is surgically removed with the contained worms and an end-to-end anastomosis is performed. This is indicated in those cases for which the mass of worms is very tightly packed, causing partial necrosis of the gut, and stretching the gut wall may threaten its viability.

Prevention

- Mass treatments with single-dose mebendazole or albendazole for all preschool and school-age children every three to four months have been used in some communities.
- Encourage proper and safe methods of sewage disposal.
- Protect food from dirt and soil.
- Thoroughly washing raw food materials is good practice.
- Encourage proper hand-washing habits and other sanitary measures.

Dracontiasis

Dracontiasis is a disease caused by the adult female *Dracunculus medinensis*, the oldest human parasite. It is more commonly known as guinea worm disease (GWD), after the Europeans who first saw the disease on the Guinea Coast of West Africa in the 17th century.

Currently, transmission occurs in only 10 countries of the world, all in sub-Saharan Africa. Other countries are either certified free of transmission or are presently in the precertification period. The goal is for dracontiasis to be the first parasitic disease to be eradicated and the first disease in history eradicated through behaviour change, without use of vaccines or cure.

Demographics

Dracontiasis is essentially a disease affecting rural communities. Whilst the occurrence of cases is possible in urban environments, such cases usually have been "imported" and were contracted elsewhere. At present, the disease is endemic mainly in West Africa, the site of 9 of the 10 countries where transmission occurs: Benin, Burkina Faso, Côte d'Ivoire, Ghana, Niger, Nigeria, Mali, Mauritania, and Togo. The tenth country is Ethiopia in East Africa.

Dracontiasis rarely occurs in children younger than the age of 3 years because the babies are generally breast-fed, and the long period of incubation delays the first emergence of the worm to one year after weaning. The incidence of the disease increases significantly after 5 years and is maximal between 15 and 45 years of age. The incidence is high in active adults who, because of their farming activities, drink larger quantities of water and use water from unsafe sources such as nontreated ponds far from the village and close to the farming field.

The disease usually shows no significant gender difference in prevalence of infection. However, in some communities, such as in northern Nigeria, the rate of infection appears to be higher in males. This is observed in populations in which women do not participate in farming activities, and thus are less exposed to drinking water from unsafe sources.

Aetiology/Pathophysiology

GWD is acquired by the ingestion of the water flea, cyclops, in drinking water. It is the only disease transmitted exclusively through drinking water. The guinea worm requires a host (man) and an intermediate host (cyclops) for its full development. The larvae mature in the cyclops found in standing dirty water (e.g., puddles, ponds, and dams). When such water is consumed, the infected cyclops is digested in the gastric hydrochloric acid, thus liberating the larvae. The larvae, male and female, burrow through the intestine to enter the circulation. The males, only 2–3 cm long, die after fertilising the females.

The female then matures in the connective tissue to measure 550–800 cm long by 1.7–2.00 mm in diameter at 10–14 months after infection, before it emerges from the subcutaneous tissue, mainly of the lower leg around the ankle (Figure 23.2). However, it can emerge at any part of the body, including the trunk, arms and hands, buttocks, thigh, knee joint, genital area, and, rarely, the neck. At this stage, the female body is mostly occupied by a distended uterus containing millions of larvae. A substance secreted by the gravid female causes a blister to form in the skin of the host around the anterior extremity of the female worm. When in contact with water, as occurs during swimming, washing, bathing, or wading, the blister ruptures; the worm protrudes and discharges hundreds of thousands of larvae into the water. The larvae are then ingested by the cyclops and mature in about 3 weeks, thus completing the life cycle of the parasite.

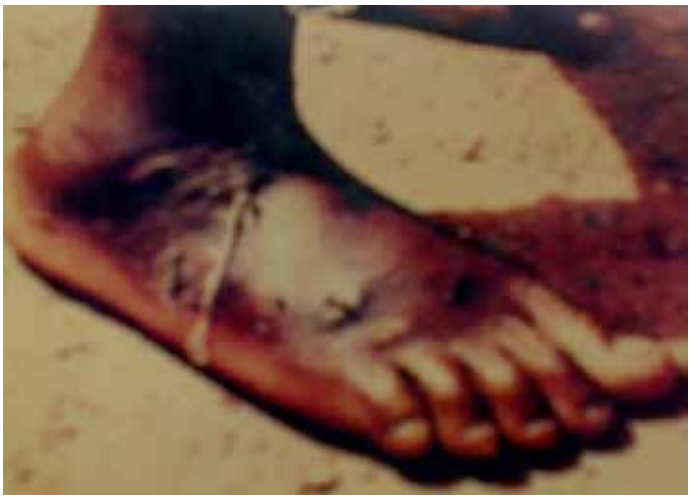

Figure 23.2: Dracontiasis in a child (note the worm emerging from the ankle).

Generally, two types of lesions are produced in man: vesicles, which ulcerate, and subcutaneous or deep abscesses around dead adult worms. Calcification of worms, which sometimes occurs in tissues, may induce local manifestations: pulmonary, cardiovascular, abdominal, urogenital, or gyn-obstetrical.

Secondary bacterial superinfection at the point of emergence of the worm is rather common when nursing is not available, and may lead to an aggravation of the condition and complications such as septicaemia and tetanus. Severe arthritis and ankylosis may be due to either the release of *D. medinensis* embryos inside the joint (aseptic arthritis) or the bacterial infection of the tunnel of the worm (septic arthritis). Such complications may lead to physical deformity and limitation of mobility.

Clinical Presentation

- GWD is rare in children <3 years old, but increases significantly after 5 years of age. Both sexes are equally affected.
- Symptoms arise when a live worm reaches the skin at the site of emergence.
- A cutaneous blister erupts with an intense burning sensation locally. A few hours before the development of the local lesion, the symptoms are exacerbated and may include erythema, urticarial rash, intense pruritus, nausea and vomiting, diarrhoea, dyspnoea, giddiness, and syncope.
- The lesion develops within a few hours in the form of a papule centred by a veside and surrounded by a local induration. On contact with water, the blister bursts and the anterior part of the worm emerges and discharges larvae and internal fluids. At this stage, the pain and the burning sensation are reduced and the other symptoms also tend to decrease.
- The worm can emerge at any place of the body, but most commonly at the lower part of the leg around the ankles.
- Calcified worms, which remain asymptomatic, have been discovered by chance on x-ray or during a surgical intervention.

- Secondary bacterial superinfection, septicaemia, tetanus, severe arthritis, and ankylosis may be additional clinical manifestations of GWD.

Treatment

The main treatment is extraction of the worm by cautious winding around a matchstick and gentle traction applied daily until it is removed. Wet compresses are applied to the ulcer daily until the discharge from the worm ceases.

Application of a topical antibiotic to the lesion prevents secondary bacterial infection and complications. The use of niridazole (Ambilhar®) (25 mg/kg in two divided doses given orally daily for 10 days), thiabendazole (50 mg/kg daily for three days), or metronidazole (10 mg/kg per dose at 8-hour doses daily for 10–20 days), can help to lessen the intense tissue reaction, make extraction easier, and relieve the pain.

The worm may be removed intact before it breaks through the skin. Preoperatively, an antihistamine is given to prevent untoward allergic reaction.

Prevention

Measures are directed to three different areas:

1. Providing a safe drinking water supply
   - Providing piped water or drilled boreholes equipped with hand pumps are appropriate, although they are expensive to maintain.
   - Improving the existing water system, such as protecting open wells or using concrete or stone masonry parapets, is a sustainable intervention. Small dams and ponds can be equipped with infiltration galleries to prevent people from wading into the water and therefore preventing infestation of the water sources by the parasite larvae.
2. Filtering drinking water
   - When safe drinking water is not available, transmission can be interrupted by using filters made from fine mesh (100 microns).
   - Ordinary cloth filters can be used at the household level, with the water boiled and aerated to restore taste.
   - A monofilament nylon cloth filter is more robust and has the ability to remove the vector of the disease from drinking water.
3. Chemically treating pond water
   - The application of temephos (Abate®) to surface water sources, mainly ponds, is an effective measure to prevent transmission by killing the vector. Treatment of the drinking water sources should be conducted monthly throughout the transmission season.

Schistosomiasis

Schistosomiasis is a group of diseases caused by trematodes (blood flukes) of the genus *Schistosoma*, the important species being *S. haematobium*, *S. mansoni*, and *S. japonicum*. It is also named bilharziasis in honour of Theodor Bilharz, a young German pathologist who discovered the aetiological agent for *S. haematobium* in Egypt in 1851. After malaria, schistosomiasis is the second most prevalent and most important parasitic disease in the world, with profound economic and public health consequences.

Demographics

Schistosomiasis remains a global health problem in the 21st century with an estimated 200 million people in 74 countries infected, of whom 85% are living in sub-Saharan Africa; the remainder live in South and Central America, the Caribbean, and the Far and Middle East. Travelers to endemic areas (particularly Africa) are at high risk of infection, and with increasing immigration globally, the chances of importing this disease to nonendemic areas are greatly increased.

The occurrence of species of schistosomiasis are highly variable from one country to another. *S. mansoni* is the most widespread, with *S. haematobium* concentrated in Africa and the Middle East, and *S. japonicum* primarily found in Asia. On the whole, school-aged children are more often and more heavily infected than adults because of their play habits and hygiene. Also, both the prevalence and intensity of infection have been found to be higher among males than females in many surveys. Like other parasitic diseases, poverty, ignorance, poor living conditions, inadequate sanitation, inadequate or total lack of public health facilities, and lack of safe water supplies, as well as deplorable personal and environmental hygiene characteristic of many developing Third World countries, are identified as important factors contributing to the increasing transmission of schistosomiasis.

Life Cycle of the Parasite

Of the different *Schistosoma* species that can infect humans, *S. haematobium*, *S. mansoni*, and *S. japonicum* are the most important because they cause the vast majority of infections.

Man is the definitive host of these parasites; *S. japonicum*, however, can live in other animals such as dogs, cats, cows, pigs, and rats. The intermediate host is the snail—bulinus for *S. haematobium*, biomphalaria and australorbis for *S. mansoni*, and oncomelania for *S. japonicum*.

For transmission to occur, there must be humans (or in the case of *S. japonicum*, animals) and snails living in close proximity and moving through the same aqueous environments. Additionally, infected humans must excrete their faeces or urine into or nearby the snail-infested water. When these conditions necessary to maintain the multistage life cycle are met, humans become infected when they come into contact with the cercariae during swimming, bathing, washing, or wading in infested water, or ingesting water from snail-infested sources. The cercariae penetrate the skin or mucous membrane to enter the body. They travel via the bloodstream, lung, and liver, and finally lodge within 30 days in the venules of the portal system, where they mature into adult worms. The adult males then move against the flow of blood, carrying the females in their gynaecophoric canal to the vesicular veins (in case of *S. haematobium*) or the mesenteric veins (in the case of *S. mansoni* and *S. japonicum*) in order to produce eggs.

Fertilised eggs or ova are released by the female parasites within the vasculature, then they cross the endothelium and basement membrane of the vein by means of a lytic substance they secrete, and enter the basement membrane and epithelium of the bladder or intestines, depending on the species involved. As a result, many eggs enter the lumen and are released from the body in urine or in the stool, but many are held up in the wall and die after 3 weeks; it is these dead ova that provoke the various pathological reactions. Those that are released from the body perish in 8 hours unless they come into contact with fresh water. The next phase of the flukes' life cycle takes place when humans urinate or defaecate into or near fresh water.

The eggs liberate their larvae or miracidia, which must enter the liver of the appropriate snail within 48 hours or die. In the snail, the miracidium forms a sporocyst that divides several times, forming daughter sporocysts containing cercariae. The sporocyst matures in 9 weeks and ruptures, releasing many cercariae excreted by the snails into the water. The tailed cercariae swim in the water until they come into contact with a human and the cycle is restarted. They die within 48 to 72 hours if no such contact is made. The life cycle takes 12–14 weeks.

Pathology

The pathological changes depend on the intensity and frequency of infection and the duration of exposure. The earliest reaction is papular dermatitis at the sites of entry of the cercariae, followed by pulmonary inflammatory reaction as the cercariae pass through the lungs. These changes may not be clinically apparent, especially in people normally resident in the endemic areas.

In the established infection, the basic pathological reaction is provoked by dead ova and consists of the formation of foreign body granulomata and fibrosis. The granuloma is made up of an ovum surrounded by epithelioid cells, plasma cells, lymphocytes, eosinophils, giant cells, and fibroblasts.

In *S. haematobium* infection, the lesions are most marked in the bladder and lower part of the ureters. The bladder often contains focal polypoid mucosal lesions or plaques of large masses of eggs. Eggs of *S. haematobium* in the bladder and ureteral walls appear to have a tendency to calcify, giving a "sand" appearance to these focal lesions and making them visible radiologically. Late effects are fibrous contraction of the bladder.

Ureteral polyps, strictures, and obstruction may lead to pyelonephritis and hydronephrosis. Cystitis with squamous metaplasia and ulceration leading to haematuria are common findings throughout the course of *S. haematobium* infection. Carcinomas of the bladder, of which half are squamous cell and almost half transitional with a few adenocarcinomas, are late complications.

The changes are more marked in *S. japonicum* infection compared to *S. mansoni* because a lot more ova are produced. The mucosa of the large bowel is hyperaemic and studded with pseudotubercles and shallow ulcers. Sessile or pedunculated polyps from coalescence of granulomata and epithelial hyperplasia are often present. Fibrosis leads to rigidity and consequent narrowing of the bowel.

The liver is also affected, especially in *S. japonicum*. It can be small, enlarged, or normal in size and nodular. The initial granulomata result in fibrosis around the terminals of the portal vein with ova embedded in them. These changes lead to portal hypertension with splenomegaly, ascites, and oesophageal varices.

Clinical Manifestation

Symptoms start after age 5 years but are marked in the second decade of life, and without treatment are severe in the third decade. Soon after penetration of the cercariae, there is a pricking sensation followed by papular dermatitis at the sites of penetration. This lasts for 2–3 days. About 4 weeks later, as a result of allergy to the developing flukes, the patient experiences intermittent fever, malaise, urticaria, and cough. These symptoms last about 2–8 weeks. Like the initial symptoms, these are mild, transient, or absent in infection by *S. haematobium*, especially in patients resident in endemic areas. In *S. mansoni* and *S. japonicum* infections, an allergic reaction to schistosomules in the liver results in pyrexia; malaise; painful, tender, and enlarged liver; splenomegaly; and at times jaundice and urticaria. From 6 to 24 months after the initial infection, symptoms develop due to the excretion of ova and the reaction to dead ova, the severity depending on the intensity of infection and the number of eggs produced.

Intermittent terminal haematuria after strenuous exercise is the main symptom of urinary schistosomiasis (*S. haematobium* infection). It occurs in about 50% of patients and is initially caused by damage of the mucosa by escaping ova and later by granulomatous ulcers or bilharzioma. Other symptoms are frequency of urination and burning sensation.

Investigations

The cornerstone of diagnosis of schistosomiasis is the detection of schistosome eggs in urine or faeces observed in saline. The characteristics of the respective ova aid their identification and diagnosis.

- *X-ray of the pelvis:* There may be calcification of the bladder wall and the lower end of the ureters in advanced cases.
- *Abdomino-pelvic ultrasound scan:* Ultrasound assessment of the changes in the urinary system are also promising. Ultrasound is still useful for early identification of periportal fibrosis and for assessment of hepatosplenomegaly.
- *Cystoscopy*: When examination of the urine and faeces is negative or there are marked bladder symptoms, cystoscopy is performed. Tubercles, sandy patches, granulomatous ulcers, or bilharzioma may be evident; biopsy of the lesion provides histological confirmation.
- *Rectal biopsy*: Ova are seen in snips of rectal mucosa.
- *Excretion urography*: Origraphy may demonstrate hydronephrosis, hydroureter, or multiple filling defects in the bladder due to bilharzioma.
- *Serological test*: Several serological tests may be done. Some of these may be used for screening of large populations.
- *Additional tests*: Tests such as sigmoidoscopy, liver biopsy, and barium swallow/oesophagogastroscopy may be indicated in intestinal schistosomiasis.

Strategies for Control

The four main foci for control of schistosomiasis are: (1) large-scale population-based chemotherapy, (2) vaccines, (3) molluscicides, and (4) environmental interventions. Various combinations of these strategies have resulted in remarkable progress toward reducing schistosomiasis. Sub-Saharan Africa, however, has had very little schistosomiasis control activity in the recent past. WHO is now rolling out initiatives to address this deficit. Current WHO initiatives target school-age children, with a goal of treating 75% of children at risk of schistosomiasis-related morbidity by 2010.

Medical Treatment

- *Praziquantel* is effective against all three species of schistomiasis. It may be given as a single oral dose of 50 mg/kg or 20 mg/kg three times at 4-hour intervals. Side effects are transient nausea, epigastric pain, pruritus or skin eruptions, headache, and dizziness. The cure rate is 80%.
- *Niridazole (Ambilhar)* is the drug of first choice for all the species of schistomiasis. The dose is 25 mg/kg (maximum 1.5 g) orally in two divided doses for 7–10 days. Side effects include confusion, depression, mania, epilepsy, slurred speech, and dark brown urine.
- *Metrifonate* is effective against *S. haematobrium* with a cure rate of 50–90%. It is given orally in a dose of 10 mg/kg repeated fortnightly for three doses.
- *Oxamniquine (Mansil®, Vansil®)* is effective against *S. mansoni* as a single oral dose of 30 mg/kg, but a second dose may be repeated in a few weeks. Fever, headache, and dizziness may occur.

Surgical Treatment

Surgical treatment may be necessary in later life. It is indicated for fibrous contracture or carcinoma of the bladder, stenosis of the ureter, hydronephrosis, portal hypertension and stenosis of the bowel.

Myiasis

Myiasis is a condition in which fly larvae (maggots) invade living tissue. They can be cutaneous, arterial, intestinal, or urinary in normal tissue or in preexisting wounds. Their importance lies in the fact that they are easily misdiagnosed, can cause mechanical damage, and cutaneous myiasis may require surgical removal of burrowed larvae.

Demographics

Myiasis is a parasitic infestation caused by the larvae of several fly species, such as *Cordylobia anthropophagi* (Tumbu fly), which is endemic in Tropical Africa and is known to have been widely distributed in the West African subregion for more than 130 years. Other fly species that produce larvae that cause myiasis are *Cordylobia rhodaini* (Lund fly), found in the rainforest areas of Tropical Africa, and *Dermatobia homonis* (human botfly), which is endemic in Central and South America.

Aetiology/Pathophysiology

Myiasis could result from a breach in healthy skin by the fly larvae themselves or through abrasions and wounds in which the respective flies deposit their eggs or larvae. The larvae can burrow through healthy tissue by using their cuticular spines aided by proteolytic enzymes.

The larvae arise from eggs deposited by the fly species on soil polluted with animal excrement, or clothing saturated with perspiration, or soiled diapers. After hatching, the larvae can stay alive for 7–20 days while attached to contaminated articles and clothing or the soil. They are activated by the warm body of the host and penetrate the skin and

develop in the subcutaneous tissue in 12 days. Dogs and small rodents are a particularly important reservoir for the parasite; humans are infected accidentally. Children, because of their daily habits and liking for pets, are especially prone to developing the disease. Under normal circumstances, the fly larvae that have penetrated the skin remain in the subcutaneous tissue below the skin orifice until they reach maturity without migrating to deeper structures. However, there have been two reports of fatal cases of cerebral infestation caused by migration of the larvae through the open fontanelles in children.

Ultimately, the larvae emerge from the swellings, which may be situated on the forearm, scrotum, and other parts of the body (Figure 23.3) and fall to the ground to pupate in 36 hours.

Clinical Presentation

The clinical presentation is usually simple and includes swelling of the part of the body involved, pain, and itching. The history may suggest recent handling of infested pests from which similar larvae may have been extracted. The child may be unkempt.

A boil-like lesion may be seen with a tiny opening at the top from which the motile tip of the larva may be observed. Application of water on the lesion activates the indwelling larva, making the diagnosis obvious.

Investigation

Diagnosis is clinical. However, the extracted larva should be submitted for parasitological identification (Figure 23.4).

Treatment

The larvae may spontaneously exit from the lesion, which then subsides unless secondarily infected. Alternatively, obstructing the cutaneous orifice by pouring water, oil, or liquid paraffin suffocates the larva, which wriggles out and can be squeezed out gently with the gloved hands. Topical antibiotic creams may be applied to prevent secondary infection. Rarely, larvae that have burrowed through the subcutaneous or deeper tissues may require surgical removal.

Hydatid Disease

Hydatid disease is a common problem in the developing countries. The cause of the disease in human beings is the hydatid cyst, which is a larval form of *Echinococci*. There are three species of *Echinococci* that may cause hydatid disease: *E. granularis*, *E. multilocularis*, and *E. olgiettas*. The clinical presentation and management of various forms of hydatid disease are similar with only minor differences. Hydatid cyst is the most common cestodes infection in the world, with cosmopolitan distribution. It is especially prevalent in sheep- and cattle-raising areas where canines such as dogs, wolves, jackals, and foxes are present.

Pathophysiology

The adult echinococcus is a small tapeworm, 3–6 mm long. It has a life-span of about 5 months. Canines (dogs, wolves, jackals, and foxes) are the definitive hosts. It resides in the upper small intestine of the host and lays eggs that are passed in the faeces of the canine and infect soil, water, and the bodies of the dogs and other animals. Cattle, sheep, and other animals get infected by ingesting these eggs and are intermediate hosts. Humans get infected by ingesting raw vegetables and water, and by close association with dogs. After ingestion, either by human beings or an intermediate host, the embryo is liberated in the small intestine, which then penetrates the intestinal mucosa to reach the portal circulation. It may settle in the liver and form a hydatid cyst. The embryo may pass the portal circulation and enter into the general circulation, where it can form a hydatid cyst in virtually any organ and tissue of the body, such as the lung, brain, and bones.

The hydatid cyst is the larval form of *Echinococcus* and consists of an inner germinal layer and outer laminated membrane. Compressed host connective tissue forms the false capsule around the cyst. The germinal layer forms bulblike projections in the lumen called brood capsules. Inside the brood capsules, small invaginations occur that form the scolices, which are the future heads of the mature worms. These

Figure 23.3: Penile myiasis (arrow) with fly larva exiting lesion after it was squeezed out with gloved hands (courtesy of Dr UE Usang, Calabar, Nigeria).

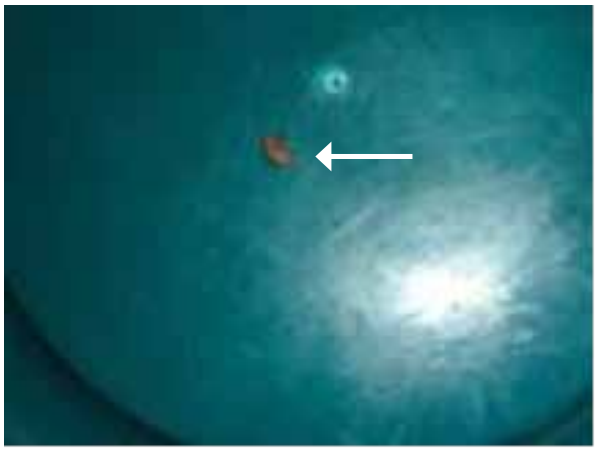

Figure 23.4: Fly larva of Cordylobia anthropophaga extracted from a 12-year-old child with penile myiasis (courtesy of Dr UE Usang, Calabar, Nigeria).

scolices have a sucker and hooklet and are infective. Multiloculated cysts are formed by infestation with *E. multilocularis*. After the death of the animal in the wild or the feeding of infected organs to the dogs by butchers, dogs and other canines ingest hydatid cyst-infested tissue. The scolices, using their hooks, settle in the proximal small intestine and grow into mature worms. Human beings are accidental hosts and do not complete the life cycle. In endemic areas, the parasite may cause infection in early childhood, but it takes a long time before the child may become symptomatic. Sometimes the disease may manifest 20–30 years after initial infection.

Epidemiology

Hydatid disease has a worldwide presence. It is seen in the countryside where cattle, sheep, dogs, and humans live in close association. In rural areas, especially in developing countries, animals are slaughtered in open areas and the infected tissue is fed to the street dogs, which may get infected and may also transmit the disease to humans. In adults, the liver and lungs are involved in 90% of the cases, and liver hydatid is seen in nearly 75% of the cases. In children, lung hydatid is more common.

Clinical Presentations

In humans, hydatid disease manifests as a hydatid cyst. Usually there is a single hydatid cyst, but there may be multiple cysts. The organs of predilection are the liver and lung, but it may involve virtually any tissues or organs of the body, such as bones, brain, spleen, heart, and peritoneum. The clinical presentation of a hydatid cyst depends on the organ involved and is either by its presence in the organ, its local pres-

sure effects, antigenic reaction, or rupture. The cyst may remain silent for a long time and regress without causing any symptoms. Patients usually have nonspecific symptoms such as cough, abdominal discomfort, low-grade fever, and malaise. In liver hydatid, the patient may present with abdominal discomfort, painful abdomen, palpable mass, and sometimes jaundice due to compression of the biliary ductal system. In the lungs, the hydatid cyst manifests as coughing, haemoptysis, and passage of white flakes in sputum, or incidental findings on a chest x-ray. Bone hydatid cysts present with bone pains and pathological fractures. Hydatid cysts in the brain usually present with features of a space-occupying lesion in the brain.

The rupture of a hydatid cyst may occur secondary to infections or trauma and can cause serious allergic reactions and even anaphylactic shock. The rupture of an untreated hydatid cyst can cause seedling and formation of multiple daughter cysts, especially in wide cavities such as the peritoneum and pleural cavities.

Diagnosis

In the endemic areas the diagnosis is often easy due to the prevalence of the disease. Patients with hapatomegaly should be properly investigated. Ultrasound evaluation is a useful tool in differentiating solid and cystic masses and can make a confident diagnosis in liver hydatid cysts. Simple liver cysts and abscesses are not uncommon in developing countries; the ultrasound evaluation can easy differentiate internal membrane and floating hydatid sand (scolices). In a ruptured hydatid cyst, the laminated membrane floats on the fluid surface and can be detected by ultrasound. A multilocular hydatid cyst can be diagnosed by its classic ultrasound appearance but may be confused with cystic tumours. In these cases a CT scan may be helpful in making a diagnosis. A lung hydatid cyst is suggested by the well-circumscribed homogenous opacity in the lung (Figure 23.5). A ruptured pulmonary hydatid cyst shows as a rounded cavity with air fluid level and floating laminated membrane and may give the appearance of the so-called "water lily" sign. Patients with hydatid cysts have moderate eosinophilia, and their immunoglobulin levels are elevated. The diagnosis may be confirmed by serological tests. Countercurrent immunoelectrophoresis (CIE) for scolex antigen and enzyme-linked immunosorbent assay (ELISA) can confirm the diagnosis in most cases. The Casoni test is performed by intradermal injection of crude sterile hydatid fluid, but it is not a reliable test.

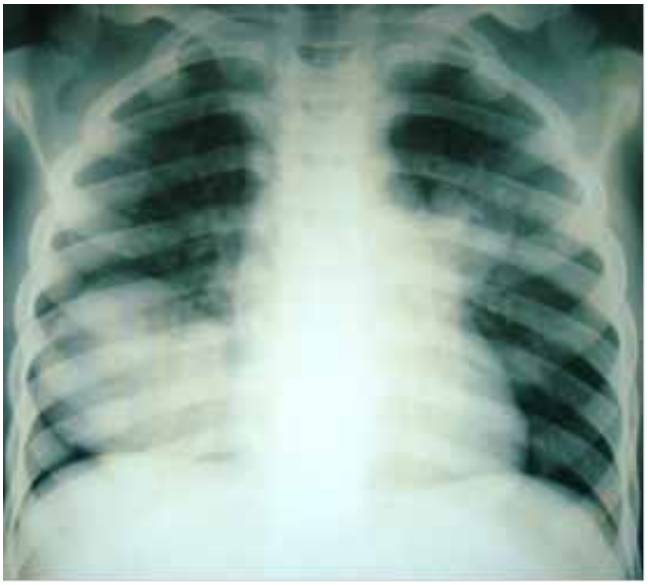

Figure 23.5: X-ray of hydatid disease of the lung.

Treatment

Management of patients with hydatid disease has changed significantly over the past few decades. Initially, surgery was considered the only option for hydatid cysts, but now medical management is useful in many cases. The drugs of choice are albendazole (12–15 mg/kg/day for 28 days) and praziquantel (50 mg/kg/day for 6-8 weeks). Mebendazole (200 mg/kg/day in 3 divided doses for 16 weeks) is also effective. Although primary surgery can eradicate hydatid cyst in most cases, there is always fear of daughter cyst formations. Furthermore, treatment with long-term albendazole can cure hydatid cyst in more than half the cases. The general consensus now is that patients with multiple hydatid cysts and having cysts less than 6 cm in diameter shall be treated medically, and any residual cysts may be tackled with surgery. In the case of a solitary cyst larger than 6 cm, surgery should be planned. Adjuvant medical therapy in these patients should be done to avoid recurrence of cysts and seedlings in adjacent tissue. PAIR (percutaneous aspiration, instillation [of hypertonic saline], and re-aspiration [after 15 minutes]) is another technique of treating hydatid cysts of the liver and lungs. These patients should also be treated with albendazole to avoid any recurrence. Recently, laparoscopic-assisted drainage of hydatid cysts has been performed with good results, but its advantage over PAIR and other procedures needs to be evaluated by long-term studies.

Evidence-Based Research

Table 23.1 presents an evidence-based study of intestinal parasitic infection among children in Karachi, Pakistan.

Table 23.1: Evidence-based research.

Title	**Prevalence and factors associated with intestinal parasitic infection among children in an urban slum in Karachi**
Authors	Mehraj V, Hatcher J, Akhtar S, Rafique G, Beg MA
Institution	Department of Pathology & Microbiology, Aga Khan University, Karachi, Pakistan. Awolowo University, Ile-Ife, Ilesa, Nigeria.
Reference	PLoS ONE 2008; 3(11):e3680.
Problem	Intestinal parasitic infections are endemic worldwide and have been described as constituting the greatest single worldwide cause of illness and disease. Poverty, illiteracy, poor hygiene, lack of access to potable water, and a hot and humid tropical climate are the factors associated with intestinal parasitic infections.
Intervention	The study aimed to estimate the prevalence and identify factors associated with intestinal parasitic infections among 1- to 5-year-old children residing in an urban slum of Karachi, Pakistan.
Comparison/ control (quality of evidence)	A cross-sectional survey was conducted from February to June 2006 in Ghosia Colony, Gulshan Town, Karachi, Pakistan. A simple random sample of 350 children aged 1–5 years was collected. The study used a structured pretested questionnaire, anthropometric tools, and stool tests to obtain epidemiological and disease data. Data were analyzed by using appropriate descriptive, univariate, and multivariable logistic regression methods. The mean age of participants was 2.8 years, and 53% were male. The proportions of wasted, stunted, and underweight children were 10.4%, 58.9%, and 32.7%, respectively. The prevalence of intestinal parasitic infections was estimated to be 52.8% (95% CI: 46.1; 59.4). Giardia lamblia was the most common parasite, followed by Ascaris lumbricoides, Blastocystis hominis, and Hymenolepis nana. About 43% of the children were infected with a single parasite, and 10% with multiple parasites. Age {Adjusted Odds Ratio (aOR) = 1.5; 95% CI: 1.1; 1.9}, living in rented households (aOR = 2.0; 95% CI: 1.0; 3.9) and a history of excessive crying (aOR = 1.9; 95% CI: 1.0; 3.4) were significantly associated with intestinal parasitic infections.
Outcome/ effect	Intestinal parasites are highly prevalent in this setting, and poverty was implicated as an important risk factor for infection. Effective poverty reduction programmes and promotion of deworming could reduce intestinal parasite carriage. There is a need for mass campaigns to create awareness about health and hygiene.

Historical significance/ comments	School-based health education for the control of soil-transmitted helminthiases in Kanchanaburi province, Thailand Anantaphruti MT, Waikagul J, Maipanich W, et al. T Ann Trop Med Parasitol 2008;102(6):521–528. Department of Helminthology, Faculty of Tropical Medicine, Mahidol University, 420/6 Ratchawithi Road, Bangkok 10400, Thailand Soil-transmitted helminthiases (STHs) are major parasitic diseases that cause health problems worldwide. School-based health education is one of several basic interventions currently recommended by the World Health Organization for the control of these infections. A 3-year programme of health education for the control of STHs has recently been completed in four primary schools in the Hauykayeng subdistrict of Thong Pha Phum district, in the Kanchanaburi province of Thailand. Overall, the percentage of the schoolchildren infected with STH increased between the start of year 1 of the intervention (16.6%) and the end of year 2 (23.8%), but showed signs of falling by the end of year 3 (19.4%). Although none of these year-on-year changes in overall prevalence was statistically significant, some significant trends were detected when the six school grades (i.e., age groups) were considered separately. The grade showing the highest prevalence of STH infection changed, from grade 6 (representing the oldest children investigated) at the start of year 1 (when grade 1 children were excluded from the survey) to grade 1 (representing the youngest children) at the ends of years 2 and 3. By the end of year 3, the children in grades 5 and 6 had significantly lower prevalences of infection than the grade 1 subjects. The prevalence of STH infection in the grade 1 children was significantly higher than that in any of the older grades at the end of year 2 and significantly higher than that in grades 3–6 at the end of year 3. These results indicate that health education had a greater impact on the children in the higher grades (who, presumably had better levels of understanding and practised better personal infection prevention) than on the younger children. Although school-based interventions can serve as a useful entry point for parasite control, more effort, including anthelminthic treatment, may be required among the youngest children. The activities need to be sustainable and supported by appropriate school health policies.

Key Summary Points

1. Parasitic infestations are a tropical disease with huge public health implications.
2. Parasitic infestations are associated with poor sanitation, low socioeconomic communities, and lack of primary health care.
3. The surgical relevance is the treatment of the complication of the disease (i.e., abscess formation, acute abdomen, and bowel obstruction).
4. A combination of medical and surgical treatment is pertinent; however, the focus for this disease should be on prevention.

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