Parasitic Infections

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Abstract

Parasitic infections of the gastrointestinal tract are a cause of morbidity to millions of individuals worldwide. These parasites are endemic in underdeveloped countries with poor sanitation allowing for spread through contaminated water supplies. While much is known about these parasites, the cutaneous manifestations caused by infection are infrequently reported in the literature. The deposition of the parasites into the perianal region often leads to significant skin irritation. Cutaneous findings vary from a mild pruritus ani to a macular rash to even severe perianal ulceration. This article discusses the perianal cutaneous findings caused by the parasitic illnesses, amebiasis, schistosomiasis, *Enterobius vermicularis*, strongyloidiasis, and cutaneous larva migrans, as well as their respective management.

Keywords

- schistosomiasis
- pinworm
- cutaneous
- parasite
- colitis
- dysentery
- helminth
- nematode
- trophozoite
- cercaria
- filariform

Parasitic infections of the gastrointestinal (GI) tract are a serious health burden in developing countries affecting millions of individuals each year. Protozoa and helminthes are the two major classifications of parasitic infections. Protozoa are microscopic single-celled organisms that invade cells and have different patterns of motility. Helminthes are multicellular worm-like organisms that reside inside the GI tract. In general, protozoa cause diarrheal illnesses, whereas helminthes cause abdominal pain or obstruction due to the burden of worms. Most parasitic infections are self-limited or easily treatable with proper medical therapy in the healthy individuals.

Cutaneous Amebiasis

Amebiasis is a protozoan infection caused by the parasite *Entamoeba histolytica* with or without clinical manifestations.[1] The large intestine of the human body is the only known natural host of *E. histolytica*.[2] Amoebic cysts are passed between individuals through fecal–oral transmission of contaminated food and water. Approximately, 480 million people are infected with amebiasis and result in the death of between 40,000 and 110,000 people a year.[3] True figures are difficult to calculate, as most cases are asymptomatic. Additionally, historical reports do not distinguish infection between *E. histolytica* from the nonpathogenic but morphologically identical forms *Entamoeba dispar* and *Entamoeba moshkovskii*.

Prevalence rates of amebiasis are highest in developing countries in sub-Saharan and tropical Africa, Indian subcontinent, and central and South America. Poor sanitation allows for transmittal of the cysts through contaminated water. Up to 40% of diarrhea may be caused by *E. histolytica* in these countries. Risk factors in developed countries include communal living, oral and anal sex, a compromised immune system, and migration or travel from endemic areas.[4] It is believed that 80 to 90% of infections have an asymptomatic course as carriers. In endemic areas, individuals may have several episodes of reinfection followed by clearance of the parasite. There is no evidence of induction of a long-lasting immunity to these pathogens.

The amoebic life cycle is straightforward. The infective form is the cyst, which is ingested from contaminated food or water. Amoebic cysts are hardy and can survive for several weeks under appropriate humidity conditions or in water. Cysts pass through the stomach unharmed after ingestion. In

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the small intestine, encystation occurs leading to eight trophozoites, which are the motile form of the parasite. Trophozoites live within the colonic lumen feeding on starches and mucosal secretions. They interact metabolically with the host's gut bacteria.[5] The trophozoites can invade the colonic wall leading to an acute diarrheal illness as well as disseminate within the human body. Encysts continue the cycle via feces. Trophozoites can be passed via the stool but cannot survive outside the human host.

Clinical manifestations of amebiasis range from asymptomatic infection to amoebic colitis and dysentery as well as invasive amebiasis characterized by liver abscess. Amoebic ingestion leads to an acute illness in only 10 to 20% of individuals do. Amoebic colitis is characterized by abdominal pain and the presence of diarrhea, sometimes with mucous and blood—a clear dysenteric syndrome. The disease is usually self-limited lasting up to 4 or 5 weeks. Fever and other systemic manifestations may be absent. A chronic nondysenteric form can develop in individuals of all ages manifested by alternating episodes of pain and diarrhea with constipation. These symptoms can mimic an irritable bowel syndrome. In children, pregnant women, and immune-compromised hosts, the disease can take a more aggressive form. Complications include a systemic colitis, colonic ameboma, fulminant colitis, or toxic megacolon.

Cutaneous amebiasis (CA) is characterized by damage to the skin and subcutaneous tissues caused by amoebic trophozoites most commonly in the perianal and perineal regions. While usually a rare manifestation of disease, CA can be frequent in endemic countries. *Hospital General de Mexico* estimated that 1 of every 300 dermatologic cases was due to CA between 1960 and 1980.[6] It can be the only expression of the disease. More commonly, it is associated with the other GI symptoms.

CA often presents in the pediatric population. Infants suffering from amoebic dysentery expel contaminated feces into their diapers. The repeated soilage contained in diapers allows for constant contact of the irritated perianal skin with the invasive trophozoites. This sequence results in characteristic ulcers seen in the diaper area of the buttocks, perineum, and inguinal folds (►**Figs. 1** and **2**).[7] CA presents as a well-defined, indurated enlarging plaque with overlying ulcers and sinuses discharging pus.[8]

The majority of CA is from sexual transmission in adults. It affects men more than women in a ratio of 1.9:1.[9] It is an emerging pathogen in men who have sex with men (MSM). Those infected with amoeba can spread the parasite through anoreceptive intercourse. A recent report describes five men with invasive amebiasis in Australia. All were MSM and four were HIV positive.[10] These patients presented with colitis and liver abscess. Trophozoites form ulcers in the perianal region or on the penis. Left untreated these ulcers progress causing severe tissue destruction, which may require surgical intervention (►**Fig. 3**).[11]

Diagnosis is established by demonstration of the cysts or trophozoites within the stool or colonic mucosa of the patients. Historically, a microscopic examination of a direct smear of the feces by wet mount or fixed and stained slide was performed. Three stool cultures are often needed for diagnosis. Unfortunately, microscopic examination cannot distinguish between *E. histolytica* and the nonpathogenic *E. dispar* or *E. moshkovskii*. Stool culture technique followed by isoenzyme analysis has been considered the gold standard

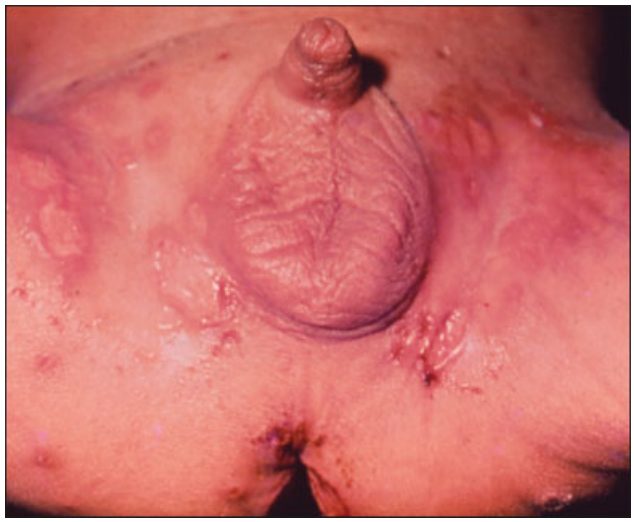

Fig. 1 Male child with multiple amoebic ulcers in the diaper distribution. (Reproduced with permission of Magaña et al. *JAMA Dermatology*.)[7]

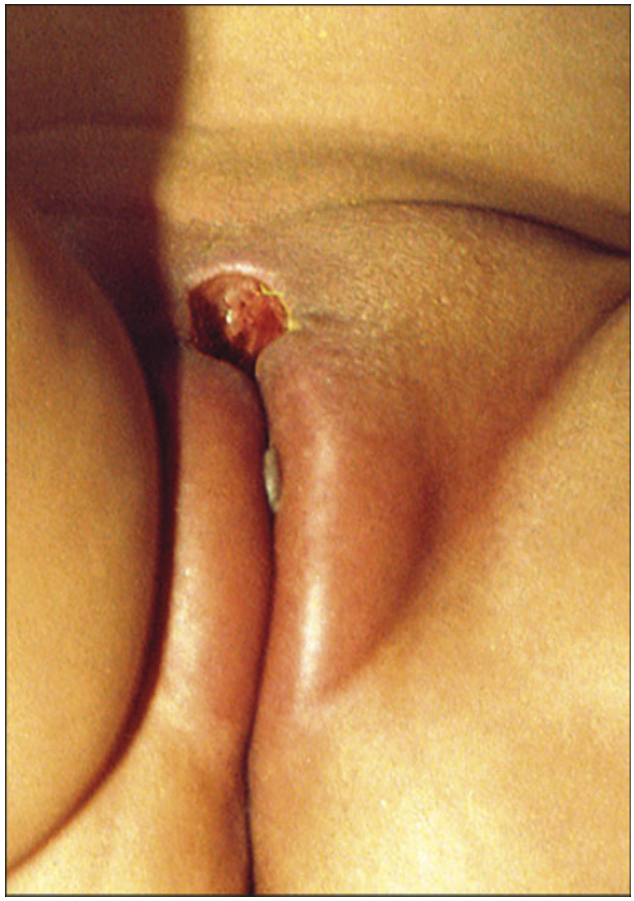

Fig. 2 A deep cutaneous amoebic ulcer in the genital region of an infant female with amoebic dysentery. (Reproduced with permission of Magaña et al. *JAMA Dermatology*.)[7]

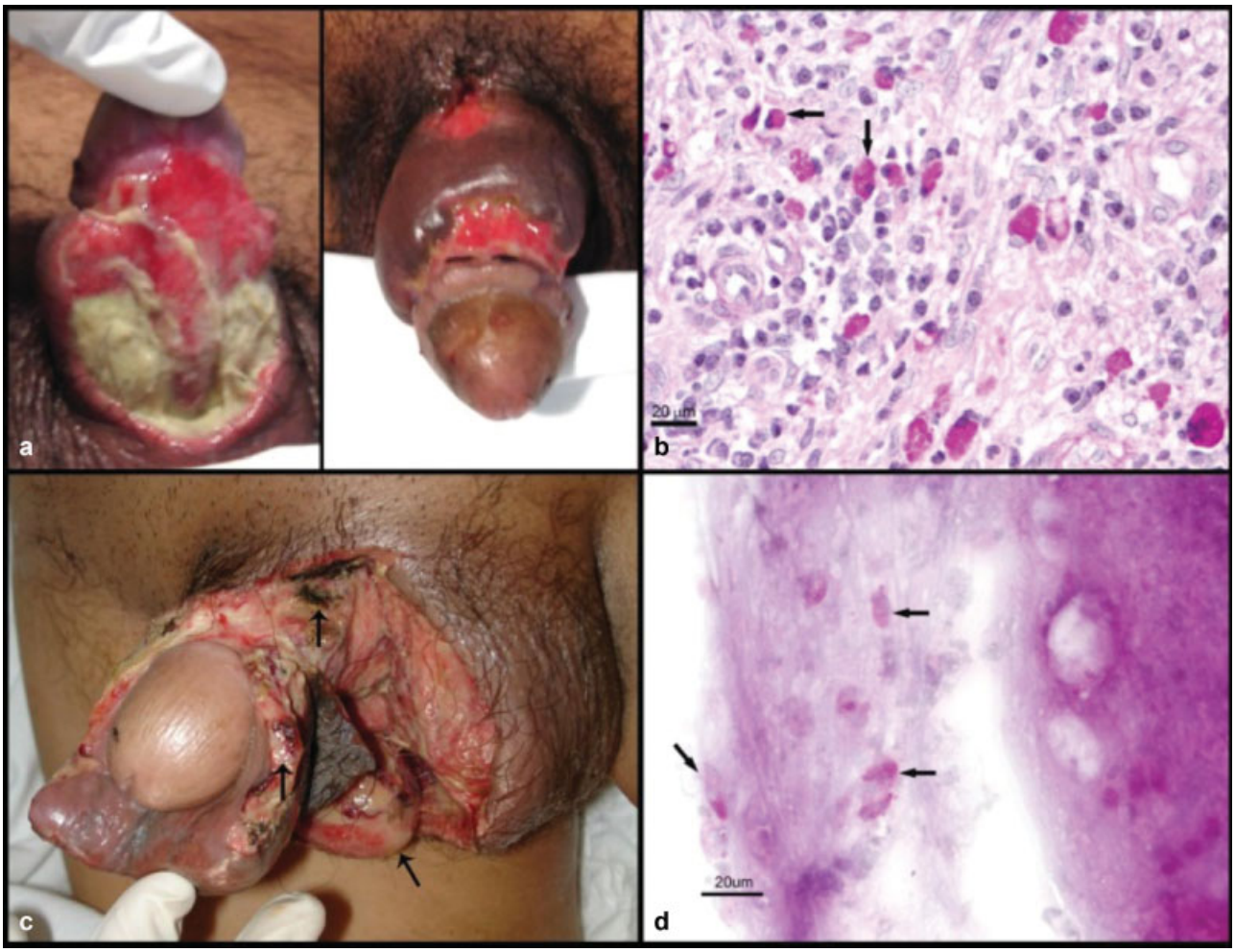

Fig. 3 (a, b) Highly tissue-destructive examples of penile cutaneous amebiasis. (c, d) Low- and high-power views of amoebic trophozoites indicated with arrows. (Reproduced with permission of Moran et al.)[11]

for many years and can distinguish between *E. histolytica* and *E. dispar*.[12] Cultures can be obtained from stool samples, colonic biopsies, or liver abscess aspirates. The test takes 1 to 4 weeks to complete because it is not feasible as a routine procedure. Enzyme linked immunosorbent assay (ELISA) tests have been developed that detect antigens in fresh and preserved stool specimens. ELISA is rapidly performed and standardized for diagnosis of amoebic infection. Its accuracy is equal to stool culture techniques. Polymerase chain reaction of stool DNA is highly sensitive and specific—but with limited by access at most institutions.

CA is readily diagnosed by smears or biopsies of the infected ulcers. It is best to take the sample from the edges of the ulcer instead of the necrotic center. Trophozoites of *E. histolytica* are round or oval unicellular basophilic structures measuring 20 to 50 µm. A halo seen around the organisms is believed to be tissue retraction due to dehydration. An inflammatory infiltrate surrounds the ulcer with neutrophils, lymphocytes, and eosinophils. Erythrophagocytosis by amoebae is constant finding, demonstrating the pathogenicity of these organisms.

The World Health Organization guidelines recommend treatment for *E. histolytica* infection regardless of symptoms.[1] If *E. dispar* is the only pathogen, treatment is not needed and search for other causes should continue. Treatment choices should be determined by the clinical scenario.[13] Asymptomatic infections (cysts) are treated with luminal amebicides such as paromomycin, diloxanide furoate, iodoquinol, quinfamide, and tetracycline.[14] These drugs prevent mucosal invasion and halt ongoing spread of the cysts by eradicating the luminal parasites. Some recommend follow-up stool samples to confirm eradication of the cysts.[15] Symptomatic infections require treatment with tissue amebicides—metronidazole or tinidazole. A 5-day course of metronidazole to treat systemic infection followed by a 10-day course of paromomycin is appropriate. Recent reports have shown effectiveness with nitazoxanide for treatment of invasive *E. histolytica*. A 3-day course has been shown to treat the systemic illness and eliminates the colonization within the colon.[16] Treatment has the added benefit of eliminating other helminthes.

Schistosomiasis Anal Dermatitis

Schistosomiasis, or bilharzia, is a systemic illness caused by parasitic trematode worms called schistosomes. A majority of human infections are caused by *Schistosoma mansoni*,

S. haematobium, and *S. japonicum*. Less common are *S. mekongi* and *S. intercalatum* as these species are geographically limited. The disease is most commonly found in Africa, Asia, and South America. It is endemic in Egypt. Approximately 200 million individuals are infected across these areas. Approximately 800 million people live in areas where the disease is common in more than 70 countries.[17] The disease is especially common among children in developing countries as they are more likely to play in contaminated water. In tropical countries, schistosomiasis is second only to malaria among parasitic diseases with the greatest economic impact. An estimated 4,400 to 200,000 people die from it each year.

The life cycle of the parasite begins by contamination of freshwater with human feces or urine containing the parasite eggs. Once a schistosome egg is immersed in water, a short-lived, nonfeeding, free-living stage known as the miracidium emerges. The miracidium uses cilia to follow chemical and physical cues thought to increase its chances of finding the first intermediate host in its life cycle—a freshwater snail. After infecting a snail, it develops into a mother sporocyst. It in turn undergoes asexual reproduction, yielding large numbers of daughter sporocysts. They asexually produce another short-lived, free-living stage—the cercaria. Cercariae use a tail-like appendage allowing them to swim and penetrate human skin. During this process, they lose their tail and become schistosomulae. These migrate into the circulatory system where they settle and mature into the adult worm. Once mature, the worms mate and the female produce eggs. The worms migrate to the intestine (*S. japonicum* and *S. mansoni*) or the bladder (*S. haematobium*) allowing eggs to be released by either the feces or urine thereby contaminating the water.

Schistosomiasis has been historically associated as a disease of the tropics. The vector is avian in North America and Europe. Waterfowl acts as the primary host in the life cycle of the parasites in contrast to the contamination by human urine and fecal discharge in other parts of the world. These species of schistosomes do not invade the human skin or colonize a human host. They remain on the skin causing a vigorous allergic reaction and inflammation. Initially, the reaction causes mildly itchy spots on the skin. Within hours, these spots become raised papules which are intensely pruritic. Each papule corresponds to the penetration site of a single parasite.[18]

Cercarial dermatitis is the short-term immune reaction occurring in the skin of humans that have been infected by the water-borne schistosomatidae. It commonly occurs within hours of infection and generally does not last more than a week. It is common in freshwater, brackish, and marine habitats worldwide. It represents one form of disease-specific perianal dermatitis. There are no permanent effects to people from this condition. Orally administered hydroxyzine, an antihistamine, is sometimes prescribed to treat swimmer's itch and similar dermal allergic reactions. Bathing in oatmeal, baking soda, or Epsom salts can also provide relief of symptoms.

Cercarial pruritus probably has been around as long as humans. The condition was known to exist as early as in the 1800s. It was not until 1928 that a biologist found that the dermatitis was caused by the larval stage of a group of flatworm parasites in the family schistosomatidae. The genera most commonly associated with swimmer's itch in humans are *Trichobilharzia* and *Gigantobilharzia*. It can also be caused by schistosome parasites of nonavian vertebrates, such as *Schistosomatium douthitti*, which infects snails and rodents. Other taxa reported to cause the reaction include *Bilharziella polonica* and *Schistosoma bovis*. Swimmer's itch can occur in marine habitats, especially along the coasts.

Some laboratory evidence indicates snails shed cercariae most intensely in the morning and on sunny days. Exposure to water in these conditions may therefore increase risk. Duration of swimming is positively correlated with increased risk of infection in Europe and North America. Shallow inshore water may harbor higher densities of cercariae than open offshore water. Children may become infected more frequently and more intensely than adults. This observation probably reflects their tendency to swim for longer periods inshore, where cercariae also concentrate. Stimuli for cercarial penetration into host skin include unsaturated fatty acids including linoleic and linolenic acids. These substances occur naturally in human skin and are found in sun lotions as well as creams based on plant oils.

Various strategies targeting the mollusk and avian hosts of schistosomes have been used by lakeside residents in recreational areas of North America to deal with outbreaks of swimmer's itch. Some work suggests that administering praziquantel to hatchling waterfowl can reduce local swimmer's itch rates in humans. Work on schistosomiasis showed that water-resistant topical applications of the common insect repellent DEET prevented schistosomes from penetrating the skin of mice.[3]

The systemic symptoms of schistosomiasis are mediated by immune reactions to the deposition of eggs in host tissues. Acute infection, also called Katayama's syndrome is characterized by fatigue, malaise, fever, cough, myalgia, diarrhea, hematuria, and abdominal pain. These symptoms usually begin within 1 to 2 months of infection. This syndrome is seen more often in travelers to endemic areas, and less so to local residents likely because of immune priming to the fetus in utero born to mothers infected by schistosomes. The chronic manifestations of schistosomal infection vary over time as the cercariae, and later adult worms and their eggs migrate through the body. *Schistosoma haematobium* worms reside within the perivesical venous plexuses migrating to the bladder to lay eggs. Eggs that are not excreted remain inside the bladder where their antigens induce a granulomatous inflammatory reaction. Classic symptoms include hematuria, increased urinary frequency, dysuria, and lower abdominal pain. *Schistosoma mansoni*, *S. japonicum*, *S mekongi*, and *S. intercalatum* reside in the vascular system of the GI tract. After maturing in the hepatic sinusoids, the female worms travel against the blood stream to the higher oxygen content around the distal colon and rectum.[19] Egg deposition again leads to edema and congestion of the colon wall, granuloma formation leading to polyps, lower GI bleeding, and crampy abdominal pain.

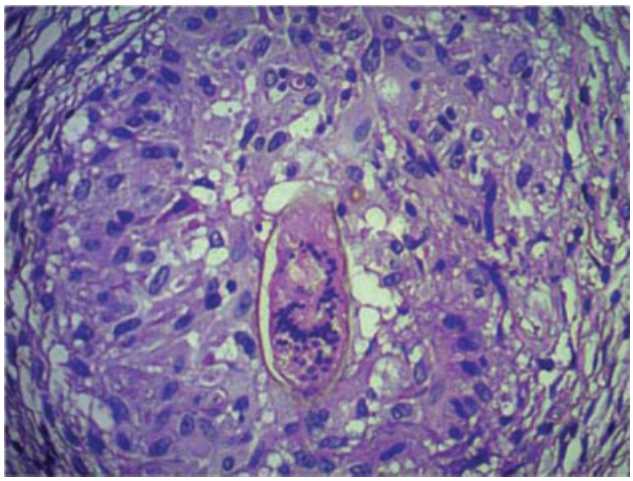

Fig. 4 Clustered dermal granulomas secondary to chronic *S. haematobium* infection in microscopic evidence. (Reproduced with permission of Lupi et al.)[20]

Bilharziasis cutanea tarda represents a form of late skin involvement. It occurs in persons with chronic, visceral disease. The chronic phase is characterized by clusters of granulomas located in the dermis[20] (►**Figs. 4** and **5**). These are most commonly formed by *S. haematobium*. They occur several months after initial infection and may be the only sign leading to diagnosis of an underlying cercarial infection in a patient without any other symptoms.

Lesions are most commonly found in the perianal region—caused by a granulomatous reaction to deposited eggs within the dermis. Findings are a small skin colored or pigmented 2 to 4 mm papules that form clusters causing pruritus.[21]

Diagnosis of schistosomiasis begins with a deliberate history and physical examination. Recent travel to endemic areas is relevant to additional testing. Characteristic rash may be seen from cercarial infection. Blood counts will show eosinophilia and anemia. Serologies signify previous infection but cannot confirm acute infections.

Microscopic identification of eggs in stool or urine is the most practical method for diagnosis of schistosomal infection. Stool examination should be performed when infection with *S. mansoni* or *S. japonicum* is suspected. Urine examination should be performed if *S. haematobium* is suspected. Eggs can be present in the stool when infected with all *Schistosomiasis* species. The extent at which the eggs are shed will determine the ability to make the diagnosis. Several stool specimens may be necessary. The examination can be performed on a simple smear with 1 to 2 mg of fecal material. Tissue biopsy (rectal biopsy for all species and biopsy of the bladder for *S. haematobium*) may demonstrate eggs when stool or urine examinations are negative.

Antibody detection can be useful to indicate schistosome infection in people who have traveled to areas where schistosomiasis is common and in whom eggs cannot be demonstrated in fecal or urine specimens. Test sensitivity and specificity vary widely among the many tests reported for the serologic diagnosis of schistosomiasis. Results are dependent on both the type of antigen preparations used (crude, purified, adult worm, egg, and cercarial) and the test procedure.

Praziquantel and oxamniquine are two drugs available for the treatment. Schistosomiasis is treatable by a single oral dose of the drug praziquantel annually. There is ongoing research into developing a schistosomiasis vaccine that will prevent the parasite from completing its life cycle in humans. As of September 2014, Eurogentec Biologics developed a vaccine called "Bilhvax" against *S. haematobium* infection in partnership with INSERM and researchers from the Pasteur Institute.[22]

Schistosomiasis caused by parasitic flat worms produces a cutaneous dermatitis of feet, anus, and other parts, after swimming in polluted streams. Historically, schistosomiasis has been associated with the vector freshwater snails prevalent in the tropics. Infection with *S. mansoni*, *S. haematobium*, and *S. japonicum* causes systemic infestation and an itchy skin rash. The vector is avian in North America. The schistosome eggs are immersed in the water where the cercariae penetrate the skin of the swimmer and cause pruritus ani. The diagnosis is confirmed by identification of eggs in stool. Antibody detection may be performed. Schistosomiasis can be detected as an incidental finding in anorectal surgery (►**Fig. 6**). The disease is preventable. The best drug for eradication is a single dose of oral praziquantel.

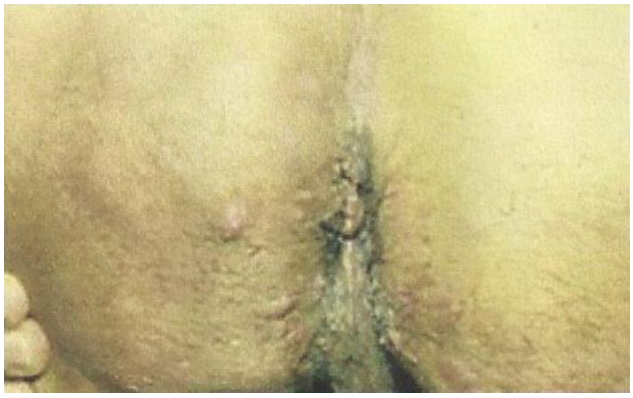

Fig. 5 Physical appearance of bilharziasis cutanea tarda where visible skin nodules reflect clustered granulomas within the dermis. (Reproduced with permission of Lupi et al.)[20]

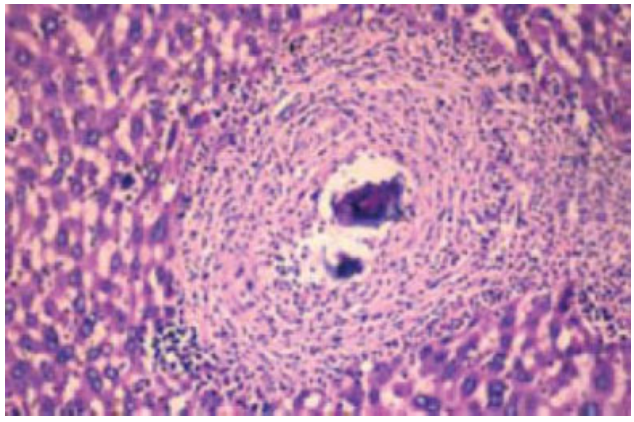

Fig. 6 Histopathology of phagocytosis of *S. mansoni* found incidentally during hemorrhoidectomy. (This image is provided courtesy of X. Delgadillo.)

Pinworm

The species *Enterobius vermicularis* is known as pinworm, threadworm, or seatworm in various parts of the world. It is a nematode (round worm)—a common helminth parasite in the human intestinal tract. *Enterobius vermicularis* is transmitted via the fecal–oral route. Humans are hosts only to *E. vermicularis* (formerly *Oxyurias vermicularis*).

The life cycle begins with eggs being ingested. The eggs hatch in the duodenum. The emerging pinworm larvae grow rapidly and migrate through the small intestine toward the colon. They molt twice and become adult during this migration. Females survive for 5 to 13 weeks, and males ~7 weeks. The male and female pinworms mate in the ileum. Males die after mating and are passed out with stool. The gravid female pinworms settle in the ileum, cecum, appendix, and ascending colon, where they attach themselves to the mucosa and ingest colonic contents.

Almost the entire body of a gravid female becomes filled with eggs. The estimations of the number of eggs in a gravid female pinworm range from ~11,000 to 16,000. The egg-laying process begins about 5 weeks after initial ingestion of pinworm eggs by the human host. The gravid female pinworms migrate through the colon toward the rectum at a rate of 12 to 14 cm/h. They emerge from the anus where the female pinworms deposit eggs. Deposition occurs by contracting and expelling the eggs, dying and then disintegrating, or bodily rupture due to the host scratching the worm. The female becomes opaque and dies after depositing her eggs. The reason the female emerges from the anus is to obtain the oxygen necessary for the maturation of the eggs.

The pinworm has a worldwide distribution. It is the most common helminth infection in the United States, Western Europe, and Oceania. A study by the Centers of Disease Control and Prevention reported an overall incidence rate of 11.4% among children in the United States. Pinworms are particularly common in children, with prevalence rates in this age group having been reported as high as 61% in India, 50% in England, 39% in Thailand, 37% in Sweden, and 29% in Denmark. Finger sucking has been shown to increase both incidence and relapse rates. Nail biting has been similarly associated. It spreads from host to host through contamination. Pinworms are common among people living in close contact. It tends to affect all members within a household. The prevalence of pinworms is not associated with gender, nor with any particular social class, race, or culture. Pinworms are an exception to the tenet that intestinal parasites are uncommon in affluent communities.

One-third of individuals with pinworm infection are totally asymptomatic. The main symptoms are pruritus ani and perineal pruritus. The itching occurs mainly during the night and is caused by the female pinworms migrating to lay eggs around the anus. Both the migrating females and the clumps of eggs are irritating, but the mechanisms causing the intense pruritus have not been explained. The intensity of the itching varies. It can be described as tickling, crawling sensations, or even acute pain. The itching leads to continuous scratching of the area around the anus. Localized perianal trauma can result in other complications such as secondary bacterial infections, including bacterial dermatitis and folliculitis. General symptoms are insomnia and restlessness. A considerable proportion of children suffer from loss of appetite, weight loss, irritability, emotional instability, and enuresis (inability to control urination).[23]

Pinworms cannot damage the skin and they do not normally migrate through tissues. However, they may move onto the vulva and into the vagina in females. Migration ensues to the external orifice of the uterus, and onward to the uterine cavity, fallopian tubes, ovaries, and peritoneal cavity. This process produces vulvovaginitis associated with vaginal discharge and pruritus vulvae. The pinworms can also enter the urethra, and presumably, they carry intestinal bacteria with them. One report indicated that 36% of young girls with a urinary tract infection also had pinworms.

Diagnosis depends on finding the eggs or the adult pinworms. Individual eggs are invisible to the naked eye. They can be seen using a low-power microscope. On the contrary, the light-yellowish thread-like adult pinworms are clearly visually detectable—usually during the night when they move near the anus, or on toilet paper. Transparent adhesive tape applied on the anal area will pick up deposited eggs. The diagnosis can be made by examining the tape with a microscope. Females do not lay eggs every day and the number of eggs varies. Therefore, this test is most successful if done every morning for several days.

Pinworms do not lay eggs in the feces, but sometimes eggs are deposited in the intestine. Routine examination of fecal material gives a positive diagnosis in only 5 to 15% of infected subjects and of little practical diagnostic use. In a heavy infection, female pinworms may adhere to stools that pass out through the anus. They may thus be detected on the surface of the stool. Adult pinworms are occasionally seen during endoscopy.[24]

Pinworm infection cannot be totally prevented under most circumstances. This difficulty is due to the prevalence of the parasite and the ease of transmission through soiled night clothes, airborne eggs, contaminated furniture, toys, and other objects. Infection may occur in the highest strata of society, where hygiene and nutritional status are typically high. Counseling is sometimes needed for upset parents who had found their children are infected and who may not realize how prevalent the infection is.

Preventative action focuses on personal hygiene and the cleanliness of the living quarters. The main measures are keeping fingernails short, washing, and scrubbing hands and fingers carefully—especially after defecation and before meals. Ideally, bed covers, sleeping garments, and hand towels should be changed daily. Simple laundering of clothes and linen disinfects them. Children should wear gloves while asleep. Bedroom floors should be kept clean. Household detergents have little effect on the viability of pinworm eggs. Cleaning the bathroom with a damp cloth moistened with an antibacterial agent or bleach will merely spread the still-viable eggs. Similarly, shaking clothes and bed linen will detach and spread the eggs.

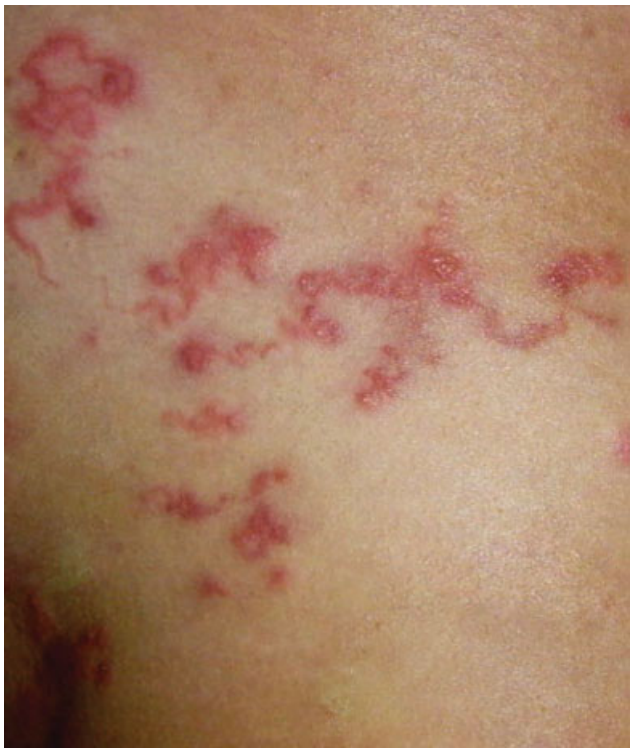

Fig. 7 A classic presentations of cutaneous larva migrans with serpiginous vermiform patterns over the right upper gluteal and lumbar–sacral regions. (This image is provided courtesy of F. Pereira.)

Medication is the primary treatment for pinworm infection. However, reinfection is frequent regardless of the medication used. Total elimination of the parasite in a household may require repeated doses of medication for up to a year or more. The first retreatment is recommended in 2 weeks because the drugs kill the adult pinworms, but not the eggs. If one household member spreads the eggs to another, it will be a matter of 2 or 3 weeks before those eggs become adult worms and thus amenable to treatment. Asymptomatic infections, often in small children, can serve as reservoirs of infection. The entire household should be treated regardless of whether or not symptoms are present.

The benzimidazole compounds, albendazole and mebendazole, are the most effective. They work by inhibiting the microtubule function in the pinworm adults causing glycogen depletion. The parasite is eradicated by starvation effectively. A single 100 mg dose of mebendazole with one repetition after a week is considered the safest and is usually effective with cure rate of 96%. Mebendazole has no serious side effects, although abdominal pain and diarrhea have been reported. Pyrantel pamoate (also called pyrantel embonate) kills adult pinworms through neuromuscular blockade. It is considered as effective as the benzimidazole compounds and is used as a second-line medication. Other medications include pyrvinium pamoate (also called pyrvinium embonate), which works by inhibiting oxygen uptake of the adult pinworms. The authors recommend piperazine which causes flaccid paralysis in the adult pinworms.

Strongyloidiasis

Strongyloides stercoralis presents with GI and/or cutaneous symptoms. Notably, it is more commonly asymptomatic in more than 60% of cases. Marked eosinophilia may be the only recognizable abnormality. Filariform larvae are passed in the stool then attaching to the perianal skin.

Table 1 Clinical characteristics and treatment of perianal parasitic infections

	Cutaneous amebiasis	Cercarial dermatitis	Pinworm	Larva currens	Cutaneous larva migrans
Pathogen(s)	*E. Histolytica*	Schistosomatidae	*Enterobius vermicularis*	*Strongyloides stercoralis*	*Ancylostoma braziliense*
Transmission	Fecal oral 2 deg contaminated food or water[a]	Swimming (bathing) in contaminate water	Fecal oral and close contacts	Direct contact faces (larvae) to skin	Direct contact with soil or sand (dog hookworm)
Perianal semiology	Plaques with overlying ulcers, sinuses and discharge	Clustered papules and pruritus	Pruritus ani	Larva currens rash: erythematous papules and serpiginous tract	Serpiginous intensely pruritic epidermal eruptions
Diagnosis	Stool O&P cultures PCR ulcer biopsies	Microscopic examination for eggs (stool or tissue)	Microscopic examination (clear tape)	Multiple stool examination ($\leq$7)	History and physical examination
Potential sequelae	Dysentery ameboma, liver	Katayama syndrome lower GI bleeding	2 deg bacterial infection vulvovaginitis	Hyperinfection syndrome (CNS pulmonary, renal, and GI involvement)	Self-limited
Primary treatment	Nitazoxanide	Praziquantel	Mebendazole or albendazole	Ivermectin	Albendazole of ivermectin

Abbreviations: CNS, central nervous system; GI, gastrointestinal; PCR, polymerase chain reaction.
[a]Transmission in developed countries is more commonly associated with communal living, oral and anal sex, and/or a compromised immune system.

Migration through the skin at the anus is called exoautoinvasion. Infectious larvae demonstrate rapid intradermal migration producing the rash of *larva currens* consists of itchy erythematous papules and serpiginous tracts overlying the perianal region. The rash can extend rapidly by 10 cm/h. It may also involve the buttocks and upper thighs. Larva currens is the pathognomonic finding indicating strongyloidiasis. The gold standard for confirmation of the diagnosis of *Strongyloides* is serial stool examination. Traditionally, stool examinations are insensitive. Up to seven stool examinations may be required to reach a sensitivity of 100%. Ivermectin in a single dose (200 µg/kg orally for 1–2 days) is first-line therapy. Albendazole is an alternative (200 µg/kg orally for 1–2 days).

Strongyloidiasis has a broad symptomatic spectrum. Acute and chronic infections are most often subclinical. It is important to recognize that severe and hyperinfection syndrome can occur with a mortality approaching 90%. This form of the disease may have devastating complications of the central nervous system, pulmonary, and GI organ systems secondary to broad dissemination of larvae migrating throughout the body's organs.[25]

Cutaneous Larva Migrans

The hookworm *Ancylostoma braziliense* infects the intestines of dogs, cats, and other animals. Other colloquialisms for cutaneous larva migrans (CML) include creeping eruption, sandworms, ground itch, and plumber's itch depending on the geographic or occupational setting. Hookworm eggs are shed in feces contaminating the ground beneath. They develop into an infectious larval form over 1 to 2 weeks. These filariform larvae burrow through the skin of humans that comes in contact with the contaminated soil or sand. The parasite lacks a specific collagenase necessary to penetrate through the basement membrane of the dermis in humans. The infection is thereby limited to the epidermis creating vermiform burrows in serpiginous patterns characteristic sufficiently for a physical diagnosis[26] (►**Fig. 7**).

Infection produces an erythematous and intensely pruritic eruption. Scratching intensifies pain and permits secondary bacterial infections. Generally, CML is self-limited healing between weeks and months. Oral albendazole or ivermectin are effective treatments. Topical thiabendazole is also an option[27] (►**Table 1**).

Conflict of Interest
None.

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