Parasitic Colitis

Elizabeth M. Hechenbleikner, MD¹ Jennifer A. McQuade, MD, FACS, FASCRS²

¹ Department of Surgery, MedStar Georgetown University Hospital, Washington, District of Columbia

² Department of Colorectal Surgery, Virginia Hospital Center, Arlington, Virginia

Address for correspondence Jennifer A. McQuade, MD, FACS, FASCRS, Colorectal Surgery, Virginia Hospital Center, 1625 N. George Mason Drive, Suite 454, Arlington, VA 22205 (e-mail: jmcquademd@gmail.com).

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Abstract

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Over one billion people worldwide harbor intestinal parasites. Parasitic intestinal infections have a predilection for developing countries due to overcrowding and poor sanitation but are also found in developed nations, such as the United States, particularly in immigrants or in the setting of sporadic outbreaks. Although the majority of people are asymptotically colonized with parasites, the clinical presentation can range from mild abdominal discomfort or diarrhea to serious complications, such as perforation or bleeding. Protozoa and helminths (worms) are the two major classes of intestinal parasites. Protozoal intestinal infections include cryptosporidiosis, cystoisosporiasis, cyclosporiasis, balantidiasis, giardiasis, amebiasis, and Chagas disease, while helminth infections include ascariasis, trichuriasis, strongyloidiasis, enterobiasis, and schistosomiasis. Intestinal parasites are predominantly small intestine pathogens but the large intestine is also frequently involved. This article highlights important aspects of parasitic infections of the colon including epidemiology, transmission, symptoms, and diagnostic methods as well as appropriate medical and surgical treatment.

Parasitic enteric infections are a serious health burden in developing countries,^{1,2} particularly among infants and children, and are common in Africa, Asia, and Latin American countries.³ In the United States, parasitic intestinal infections are generally uncommon without specific risk factors, such as traveling to endemic areas or having a compromised immune system.⁴ Protozoa and helminths are the two major classes of parasites that infect the human gastrointestinal (GI) tract causing a range of clinical outcomes from asymptomatic colonization to life-threatening illness. Protozoa are microscopic, unicellular organisms that invade cells and have different patterns of motility. In contrast, helminths are multicellular, worm-like organisms that reside inside the GI tract.⁵ Intestinal parasites are predominantly small intestine pathogens but the large intestine is also frequently involved.

Depending on the specific pathogen and the host's overall health, parasitic intestinal infections can develop acutely with severe abdominal pain and fever mimicking appendicitis or in a chronic fashion with weight loss and diarrhea presenting like inflammatory bowel disease (IBD).⁶ In general, protozoa

cause diarrheal illnesses, while helminths cause abdominal pain and can lead to intestinal obstruction or prolapse in individuals with a large burden of worms. In summary, parasitic intestinal infections are common worldwide causing tremendous morbidity. Most infections are self-limiting and respond to proper medical treatment while surgical intervention is reserved for complications of infection. This article highlights important aspects of parasitic infections of the colon including epidemiology, transmission, clinical presentation, and diagnostic methods as well as appropriate medical and surgical treatment.

Protozoal Infections

Protozoan enteric infections are caused by a diverse group of parasites including the coccidia, ciliates, flagellates, and amoebae, all of which result in diarrheal illness. In contrast, *Trypanosoma cruzi* infection (i.e., Chagas disease) causes constipation due to chronic colonic dilation and hypoperistalsis.⁷ The protozoan life cycle has two major stages: the cyst stage and trophozoite stage. Cysts are excreted in feces, have

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robust protective walls, and are responsible for the transmission of the parasite. The trophozoite stage generally occurs directly inside cells or hollow organs and is responsible for the intestinal pathology and clinical manifestations.

The Coccidia: *Cryptosporidium*, *Cystoisospora*, and *Cyclospora*

Cryptosporidium, *Cystoisospora*, and *Cyclospora* cause the diarrheal illnesses cryptosporidiosis, cystoisosporiasis, and cyclosporiasis, respectively. *Cryptosporidium* species are found worldwide from Sub-Saharan Africa to the United Kingdom and Australia. *Cystoisospora* is common in the tropics and subtropics and *Cyclospora* is endemic in Haiti, Peru, China, and Nepal.⁸ All three diseases result from ingesting infectious oocysts which excyst to sporozoites, invade epithelial cells, and reproduce making more oocysts that are shed in feces.⁹ In contrast to *Cystoisospora* and *Cyclospora*, *Cryptosporidium* generates oocysts that stay inside the GI tract reinfesting epithelial cells (i.e., autoinfection) along with mature, infectious oocysts that are excreted with feces. Contaminated drinking or recreational water from infected humans or cattle is the main source of disease spread for *Cryptosporidium*. Oral-anal sexual practices also increase the risk of *Cryptosporidium* infection. In contrast, humans are the only hosts and reservoirs for *Cystoisospora* and *Cyclospora*, which are generally spread via contaminated water or food. Children and acquired immunodeficiency syndrome (AIDS) patients in developing countries are particularly vulnerable to intestinal coccidial infections, while sporadic waterborne outbreaks generally happen in developed countries.

Characteristically, patients present with profuse non-bloody, watery diarrhea along with nonspecific symptoms, such as abdominal pain, nausea, and/or sweats. The acuity and severity of diarrheal disease depends on the immunologic health of the patient. Immunocompetent patients usually present with acute, self-limiting symptoms whereas immunocompromised patients (e.g., AIDS patients) may have protracted and severe diarrheal disease resulting in malnutrition and weight loss. In addition, AIDS patients frequently develop extraintestinal problems including gallbladder and/or biliary tract infection.¹⁰

Coccidian protozoa usually parasitize small intestine epithelial cells but have also been found in the appendix, colon, and rectum, particularly in the setting of cryptosporidiosis. Characteristic histopathologic changes include an intense inflammatory response as well as blunting and atrophy of villi. Nonspecific mucosal abnormalities may be noted at the time of colonoscopy. Notably, cystoisosporiasis causes peripheral eosinophilia which is not seen with other coccidian parasites. Diagnosis can be confirmed by examining stool specimens for the presence of oocysts which may require concentration procedures (e.g., Sheather sugar flotation) and/or staining (e.g., modified Ziehl-Neelsen staining).^{11,12} Polymerase chain reaction (PCR) diagnostic techniques are also available and are more sensitive. If fecal analyses fail, intestinal biopsies can be obtained for diagnosis.

Intestinal coccidial infections are treated with medical intervention. Treatment is initially aimed at properly rehydrating patients and addressing any nutritional needs. Nitazoxanide is used to treat cryptosporidiosis in immunocompetent adults and pediatric patients¹³ as well as AIDS patients with low CD4 counts (i.e., < 100 cells/ μ L).¹⁴ Patients with AIDS having very low CD4 counts (i.e., < 50 cells/ μ L) are less likely to respond to therapy and can progress to fulminant infection with high mortality.¹⁵ Trimethoprim/sulfamethoxazole (TMP-SMX) is the treatment of choice for cystoisosporiasis and cyclosporiasis. It is imperative to optimize highly active antiretroviral therapy (HAART) in human immunodeficiency virus (HIV) patients, particularly for cryptosporidiosis for which HAART is the cornerstone of therapy.

The Ciliates and Flagellates: *Balantidium coli* and *Giardia lamblia*

The ciliate, *Balantidium coli*, and flagellate, *Giardia lamblia*, are responsible for the protozoan illnesses known as balantidiasis and giardiasis, respectively. *B. coli* infection is frequently asymptomatic and is more limited in its geographic distribution compared with *G. lamblia*. Balantidiasis is relatively uncommon in humans and largely confined to the tropics and subtropics. Unlike the intestinal coccidial infections, balantidiasis is not typically seen in immunocompromised patients.¹⁶

Pigs are the major reservoir for *B. coli* and infection is common in areas with poor sanitary conditions where public water supplies are contaminated with pig feces. Once humans ingest infective cysts, excystation occurs and trophozoites colonize the large intestine and may invade the colonic wall. Trophozoites then undergo encystation and mature cysts are excreted in feces. Human-to-human spread of *B. coli* infection is possible, albeit much less common. Balantidiasis may present in a similar fashion to amebiasis (*E. histolytica* infection) with acute abdominal pain, bloody diarrhea, and colon ulceration.¹⁷ Surgical intervention is reserved for serious complications, such as bleeding and perforation.¹⁸ Microscopically, trophozoites can be easily identified in wet mount preparations of freshly collected diarrhea while cysts are found in normal, formed stool samples. Colon biopsies can also be used to identify the parasite and determine the amount of inflammation and damage to the intestinal wall.¹⁶ In adults, medical treatment with tetracycline or metronidazole typically resolves balantidiasis.¹⁴

In contrast to balantidiasis, giardiasis is one of the most common protozoan diarrheal illnesses in humans and is frequently found in other animal species.¹⁰ The presence of HIV infection does not appear to increase the risk of infection. In the United States, *G. lamblia* is the most prevalent protozoan parasite.¹⁹ Giardiasis is transmitted mainly by ingesting contaminated water but can be passed between humans or, less commonly, contracted from infected animals. After ingestion of infectious cysts, trophozoites multiply in the small or large intestine either staying in the lumen or attaching to the epithelium without mucosal invasion. Similar to cryptosporidium, cysts are immediately infectious after being

excreted in feces; hence, giardiasis is easily passed between household members or in day care centers. In developing countries, infection can become rampant in areas of crowding and poor sanitation.

Clinically, *G. lamblia* infection can result in asymptomatic colonization, acute diarrhea, chronic diarrhea with malabsorption and weight loss, or nonspecific GI complaints (e.g., dyspepsia).^{20,21} Acute diarrhea is usually self-limiting and frequently accompanied by cramping, bloating, and steatorrhea. In the chronic setting, giardiasis can negatively affect growth and cognitive development in children and has been included in the World Health Organization's Neglected Diseases Initiative.²² Microscopic examination of stool samples is commonly used to make the diagnosis. Similar to balantidiasis, cysts are almost always found in formed stool while trophozoites are excreted in diarrhetic fecal samples. Enzyme-linked immunosorbent assays (ELISA) to detect serum antibodies against *G. lamblia* are also available and are very sensitive. If stool and serum studies fail to make the diagnosis, duodenal aspirates or biopsies may be obtained. Treatment is generally the same regardless of the patient's immunologic status and is predicated on whether symptoms are present.⁶ The preferred antiparasitic agents are tinidazole or metronidazole.¹⁴ In developing countries, reinfection rates are high thus treatment of asymptotically colonized patients may not be worth the cost.

The Amoebae: *Entamoeba histolytica*

Entamoeba histolytica is an invasive colon pathogen found globally but concentrated in Central and South America, Africa, and India.²³ *E. histolytica* moves and feeds with pseudopodia or "false feet" and is transmitted via the fecal-oral route; humans are the predominant reservoir and host. The life cycle is similar to that of *B. coli*; while extraintestinal spread is rare in balantidiasis, *E. histolytica* invades the mucosa causing ulceration, spreads in the bloodstream, and commonly infects other organs. *E. histolytica* infection has three clinical scenarios: (1) asymptomatic colonization, (2) intestinal amebiasis, or (3) extraintestinal amebiasis. In extraintestinal amebiasis, the most common infection is a liver abscess, but brain and splenic abscesses may develop. Intestinal amebiasis commonly presents with abdominal pain, tenesmus, fever, and bloody diarrhea with mucus. Intestinal symptoms that develop abruptly in children and immunocompromised patients are often misdiagnosed as appendicitis.⁶ Despite the rarity of amebic acute appendicitis, it is a possibility and predominantly occurs in regions where the risk of amebiasis is high.²⁴ Serious complications include hemorrhage from ulceration, peritonitis due to perforation,²⁵ toxic megacolon,²⁶ and fulminant colitis which frequently require urgent surgical exploration and intestinal resection. In the setting of fulminant colitis, the colon can be extremely friable and difficult to manipulate due to extensive necrosis.²⁷

Diagnosis can be made via microscopic examination of stool to identify trophozoites or cysts but this is mainly done in places where updated techniques are not available. Currently, serologic assays (e.g., indirect fluorescent antibody

test) and the detection of *E. histolytica* antigens in stool are the preferred diagnostic methods. Conventional and real-time PCR tests are available but are more expensive.²⁸ In addition, colonoscopy may aid in the diagnosis; biopsies of ulcerated colon tissue may demonstrate trophozoites (→ Fig. 1).⁶ Notably, deep, flask-shaped ulcers are common in amebiasis and can be mistaken for ulcerative colitis (UC) during colonoscopy.²⁷ In the setting of presumed UC, patients may mistakenly receive steroid treatment which can lead to hyperinfection and have fatal consequences.²⁹ For intestinal amebiasis, treatment with metronidazole or tinidazole should be followed by a luminal agent, such as iodoquinol or paromomycin.¹⁴ Liver abscesses usually respond to metronidazole and do not require drainage.²⁷

Chagas Disease: *Trypanosoma cruzi*

Trypanosoma cruzi is the parasite that causes Chagas disease, infecting humans and other mammals. The disease is also known as American Trypanosomiasis because it is endemic in parts of Latin America, particularly Brazil, Argentina, and Mexico, where millions of people are infected. In the United States, over 300,000 people are likely to suffer from Chagas disease.³⁰ Transmission is predominantly vector borne through infected triatomine bugs which are almost exclusively found in Latin America. During a bite, the triatomine vector defecates and infective metacyclic trypomastigotes enter the host's bloodstream. Other less common routes of transmission include congenital, blood borne, and oral; solid organ donors can also transmit *T. cruzi* to recipients.³⁰

Upon entry into the host, trypomastigotes invade a variety of cell types becoming amastigotes where they multiply, become trypomastigotes again, and spread throughout the body. Chagas disease has both acute and chronic presentations. In the acute setting, localized redness and swelling are commonly noted at the triatomine bite wound. As the parasite spreads systemically, patients may develop generalized lymphadenopathy and complain of fevers and fatigue. Acute Chagas disease is diagnosed via microscopic visualization of parasites in blood smears and typically resolves within

Fig. 1 *Entamoeba histolytica* trophozoites in the intestinal tissue stained with hematoxylin and eosin.

8 weeks.³¹ Upon resolution of the acute phase, patients may transition into an asymptomatic disease state for years before developing end-organ dysfunction. Chronic Chagas disease predominantly causes cardiac and GI complications. In chronic disease, serodiagnostic assays are used to identify IgG antibodies to *T. cruzi*.⁷ Treatment is recommended for asymptomatic adults with chronic Chagas disease, particularly women of child-bearing age. The most common medications used to treat Chagas disease are benznidazole and nifurtimox.¹⁴

GI complications are heavily focused in South American countries.³² Megaesophagus is the most common GI problem but megacolon also develops.⁷ The distal aspects of the colon and rectum appear to be the most affected, which can be demonstrated on barium enema or other radiographic studies.³² The pathogenesis involves thickening of the intestinal wall, degeneration of neurons, progressive dilation, ineffective peristalsis, constipation, and ultimately megacolon (→Fig. 2). The abnormally dilated colon can also present as a volvulus. Patients with constipation should initially be treated with increased dietary fiber and water intake, laxatives, and enemas. Apart from surgical emergencies (e.g., volvulus), elective resection is offered to patients with chronic, debilitating symptoms who fail conservative treatment. Surgical options include rectosigmoidectomy with ileal loop interposition³³ or the Duhamel–Haddad operation which involves a sigmoidectomy followed by sewing the descending colon to the posterior aspect of the rectal stump.³⁴

Fig. 2 Representative drawing of a patient with abnormally dilated loops of large intestine due to chronic Chagas disease.

Helminth Infections

Helminths are macroscopic parasites with complex life cycles infecting billions of people worldwide.⁹ Nematodes and schistosomes are responsible for the majority of human helminth infections. A subset of nematodes is called soil-transmitted helminths (STHs), including *Ascaris lumbricoides*, *Trichuris trichiura*, and *Strongyloides stercoralis*. In contrast, *Enterobius vermicularis* is an intestinal nematode that does not require a soil phase for transmission. Schistosomes are parasitic flatworms commonly referred to as flukes. STHs are found in the tropical climates of Africa, Asia, and Latin America and schistosomes are common in the Middle East, South America, Africa, and Asia.³⁵ *E. vermicularis* is found in both tropical and temperate climates around the world.

Soil-Transmitted Helminths: *Ascaris lumbricoides*

Ascaris lumbricoides causes the intestinal illness known as ascariasis. Over 800 million people are estimated to be infected with the *Ascaris* roundworm.³⁶ In developing countries, ascariasis is very common resulting in impaired growth, cognitive development, and decreased school attendance particularly among preschool-aged children and adolescents.³⁷ In the United States, ascariasis is rare but tends to occur in the rural Southeast³⁸ and in immigrants³⁹ from the developing world. Patients with HIV do not appear to be at increased risk for infection.⁴⁰

Infection occurs when embryonated *Ascaris* eggs are ingested from contaminated food and/or water. Characteristically, eggs are shed in the feces of infected individuals into the soil where they mature over a few weeks to become infectious. Upon ingestion, eggs hatch into larvae in the small intestine where they penetrate the intestinal wall to enter the bloodstream and travel to the lungs. In the lungs, the larvae ascend the respiratory tract and are swallowed back into the GI tract. The majority of adult worms are found in the jejunum and ileum and can grow to over 30 cm. Female worms produce thousands of eggs each day which are shed in feces. Diagnosis is made via microscopic visualization of eggs in fecal samples.

Most people who are infected with *Ascaris* worms have no symptoms. In patients with ascariasis, symptoms range from minimal GI complaints to severe abdominal pathology.⁴¹ Surgical complications of ascariasis are frequent in children. Patients with a large worm burden are more likely to have intestinal symptoms and may present with obstruction, perforation, intussusception, and/or bowel necrosis requiring emergent surgical intervention.⁴² *Ascaris* worms can also migrate into the appendix causing acute appendicitis.³⁹ In adult women, biliary and pancreatic complications are common. Adult worms can obstruct the extrahepatic biliary tree resulting in complications, such as cholangitis, pancreatitis, and hepatic abscess.³⁶ Abdominal computed tomography, plain films, and ultrasound are useful diagnostic modalities for establishing intestinal and/or biliary complications of ascariasis.^{43,44} Effective anthelmintic agents include albendazole and mebendazole.

Soil-Transmitted Helminths: *Trichuris trichiura*

Whipworm or *Trichuris trichiura* infection affects an estimated 600 to 800 million people globally.³⁶ Similar to *Ascaris*, *T. trichiura* eggs take several weeks to embryonate in soil and cause infection upon ingestion from contaminated food or water. In contrast to ascariasis, trichuriasis is a disease of the large intestine. Once the *Trichuris* eggs are ingested, larval maturation preferentially happens in the cecum and there is no migration to extraintestinal sites. Mature whipworms attach themselves to the epithelial surface causing inflammation and colitis which, in serious infections, can affect the entire colon and rectum (►Fig. 3).⁴⁵

The majority of individuals infected with *T. trichiura* are asymptomatic or have minimal symptoms. Some patients with trichuriasis present with symptoms similar to IBD, such as bloody diarrhea, anemia, and abdominal pain. Serious complications, such as rectal prolapse, are more likely to happen with heavy worm infections and may require surgical intervention.⁴⁶ Microscopic visualization of *Trichuris* eggs from fecal samples confirms the diagnosis but may require concentration procedures. If noninvasive tests fail, endoscopy allows for direct visualization of *Trichuris* worms, assessment of the severity of colitis and/or proctitis, and removal of worms.⁴⁷ A short course of an anthelmintic agent, such as albendazole and mebendazole, is usually sufficient to eradicate infection.¹⁴

Soil-Transmitted Helminths: *Strongyloides stercoralis*

Strongyloides stercoralis is estimated to infect up to 100 million people worldwide.⁴⁸ Studies reporting the prevalence of strongyloidiasis in the United States are outdated but historically infection has been endemic in subregions of Appalachia.⁴⁹ In contrast to other STHs, *S. stercoralis* infection

results from infective larvae penetrating the skin, which usually happens in the feet while walking barefoot on the soil. Infective larvae enter the bloodstream traveling to the lungs where they mature, ascend the respiratory tree, and are swallowed. Female larvae typically burrow into the intestinal wall in the duodenum and jejunum and release eggs. The eggs hatch into noninfective larvae which are shed in feces contaminating the external surroundings. In the soil, noninfective larvae become infective either via direct development or through the release of eggs that hatch and mature into infective larvae.⁴⁸

Strongyloidiasis can affect various areas of the body including the GI tract, kidneys, alveoli, meningeal space, skin, and joints. In the GI tract, noninfective *S. stercoralis* larvae can become infective, invade the colonic mucosa, and cause an inflammatory pattern that mimics IBD, particularly UC.⁵⁰ *S. stercoralis* colitis may demonstrate aphthoid ulceration on colonoscopy, absence of haustra on radiographic studies, and/or peripheral eosinophilia.⁵¹ Disseminated strongyloidiasis is a rare form of *S. stercoralis* hyperinfection which spreads to multiple organs and is almost always fatal. In addition, the risk of strongyloidiasis appears to be increased in HIV/AIDS patients as well as patients with human T-cell lymphotropic virus type I (HTLV-I).

The diagnosis is typically made by visualization of larvae on wet-mount preparation of stool, which may require concentration procedures and/or multiple samples. *S. stercoralis* colitis is confirmed by identifying larvae in biopsy samples.⁵¹ The preferred drug for treatment is ivermectin but albendazole is a suitable alternative.

The Common Pinworm: *Enterobius vermicularis*

Enterobiasis or pinworm infection is caused by *Enterobius vermicularis* and is estimated to infect at least one billion people globally. Enterobiasis is the most common intestinal

Fig. 3 Adult *Trichuris trichiura* worms in hemorrhagic colon mucosa.

helminth infection in the United States.⁵² Humans are believed to be the only host for *E. vermicularis*. Children suffer the burden of most pinworm infections with an estimated 4 to 28% of this age group affected worldwide.⁵³ Enterobiasis is passed easily among people living in close quarters particularly institutionalized patients and household members who co-sleep or share sleeping space.

E. vermicularis eggs are ingested and hatch into larvae in the small intestine. The larvae migrate to the large intestine residing largely in the cecum and appendix. Adult and female larvae mature and mate inside the GI tract. The gravid adult females travel to the perianal region at night laying eggs which can cause intense itching. The eggs become infective within 6 hours and get deposited on clothes, sheets, and under fingernails when the host scratches the perianal region. Finger sucking and nail biting can result in autoinfection.⁵⁴ Although pruritus ani is the most common symptom, acute appendicitis is also frequently associated with enterobiasis in the pediatric population due to adult worms migrating into the appendix (►Fig. 4).⁵⁵ Diagnosis is different from other intestinal nematodes because *E. vermicularis* eggs are uncommonly shed with feces. The Scotch tape test, either done at night or early in the morning, is the best way to make the diagnosis. Briefly, scotch tape is pressed against the perianal region and then placed on a glass slide to visualize eggs under a microscope.⁵⁶ Medical treatment with albendazole or mebendazole is usually highly effective. Of note, following appendectomy for parasitic appendicitis, patients should undergo medical treatment to ensure pinworm infection is completely cleared.

Schistosomiasis: *Schistosoma*

Schistosomiasis is caused by a diverse group of *Schistosoma* species or trematodes and is estimated to infect 200 million people worldwide.⁵⁷ Schistosomiasis is a significant health

and economic burden in developing, impoverished nations where early childhood infection is common. Three predominant species infect humans, including *S. haematobium*, *S. mansoni*, and *S. japonicum*. *S. haematobium* and *S. mansoni* are endemic throughout parts of Africa, while *S. japonicum* is highly prevalent in China and the Philippines.

Humans are infected in freshwater when they come in contact with cercarial larvae from intermediate snail hosts. These free-swimming larvae penetrate the skin while the host is swimming or bathing which may result in an itchy rash. Once in the bloodstream, the larvae become schistosomulae, enter the mesenteric and portal circulation, mature to adult form, and migrate to various organs laying eggs. Ultimately, *S. haematobium* eggs are excreted in the urine while *S. mansoni* and *S. japonicum* eggs are shed with feces contaminating freshwater sources. The eggs hatch into miracidia larvae which then infect snails to restart the life cycle.⁵⁸

Acute and chronic sequelae happen as a result of the deposited eggs. Chronic sequelae are more likely to develop in endemic areas of infection. In the acute setting, the initial immune response results in nonspecific, generalized findings, such as peripheral eosinophilia, fever, and organomegaly (Katayama fever). In the chronic setting, inflammation and fibrosis cause varying degrees of organ injury.⁵⁸ *S. haematobium* infects the urogenital tract; eggs get deposited in the ureters and bladder which can cause dysuria, hematuria, bladder outlet obstruction, and renal failure. In contrast, *S. mansoni* and *S. japonicum* infect the liver and GI tract. In the liver, eggs localize to the inferior mesenteric and portal veins leading to portal hypertension, hepatic dysfunction, and hematemesis.^{59,60} In GI schistosomiasis, eggs are deposited in the intestinal submucosa causing abdominal pain and bloody diarrhea, and chronic colitis, ulceration, stricturing, and inflammatory polyps may also develop.⁶¹

The diagnosis of schistosomiasis can be made by visualizing eggs under a microscope from urine or fecal samples as

Fig. 4 Section from appendectomy specimen demonstrating intraluminal *Enterobius vermicularis* worms.

well as biopsies from the GI or urogenital tracts. ELISA techniques are also available for detecting antischistosomal antibodies in the blood but cannot differentiate between active or previous infection. The mainstay of medical therapy is praziquantel¹⁴ and is estimated to clear infection in 60 to 90% of the patients.⁵⁷ Surgery for GI schistosomiasis is reserved for complications, such as bleeding, perforation, and stricturing.

Conclusion

Parasitic intestinal infections are a serious global health burden, particularly in developing countries, and can have a detrimental effect on growth and cognitive development in children. GI parasites have a broad spectrum of clinical manifestations from asymptomatic colonization to overwhelming sepsis. Medical therapy is the mainstay of treatment while surgical intervention is reserved for complications of infection, such as perforation, bleeding, or fulminant colitis.

The World Health Organization has included protozoal (i.e., Chagas disease) and soil-transmitted helminth GI infections in their neglected tropical diseases initiative which is aimed at eliminating tropical infections with widespread deleterious effects. Mass drug administration of antiparasitic agents to endemic areas of infection has drastically improved but still has challenges; continued funding, epidemiological and drug research, and logistical support are required to sustain the delivery of proper medical care to those who most need it. Improved diagnostic techniques and vaccines offer additional opportunities in the global fight against pathogenic GI parasites.

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