Parasitic Colitides

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ABSTRACT

Parasitic infections are a major worldwide health problem, and they account for millions of infections and deaths each year. Most of the infections as well as the morbidity and mortality from these diseases occur in the developing world in rural regions. However, these diseases have become more common in Western countries and in big cities over the past 25 years. These changing disease patterns can be attributed to emigration from the third world to developed countries and migration of rural populations to the big cities in developing nations. These parasitic infections have protean manifestations and consequences. The medical problems range from chronic asymptomatic carrier to fulminant infections and even death. Several factors such as the host immune status, the infecting organism, and the availability of treatment all play key roles in the outcomes of parasitic colitides. The two major classes of parasites causing these infections are the helminthes (ascariasis, strongyloidiasis, enterobiasis, trichuriasis, and schistosomiasis) and the protozoa (*Isospora*, *Cryptosporidium*, *Cyclospora*, *Trypanosoma cruzi*, *Giardia lamblia*, and *Balantidium coli*). This article summarizes the salient features of each parasite with respect to epidemiology, transmission, pathogenesis, clinical features, diagnosis, and treatment. The vast majority of these infections have a self-limited clinical course or are easily treated with medical management, and surgery is rarely needed.

KEYWORDS: Protozoa, helminth, infectious colitis, Chagas disease, schistosomiasis, enterobiasis, trichuriasis, strongyloidiasis, ascariasis, giardiasis, *Cyclospora*, *Isospora*, *cryptosporidium*, *Balantidium coli*

Objectives: Upon completion of this article, the reader should be able to summarize the features and management of parasitic colitides.

Acute diarrheal illness is a major worldwide problem that is one of the five leading causes of mortality each year.¹ The major causes are generally viral and bacterial illnesses and less often protozoal infections. Furthermore, protozoal infections generally involve the small intestine instead of the colon. As a result, parasitic colitides are an uncommon cause of diarrhea. Nonetheless, parasitic infections of the colon are big problem in the developing world and are not uncommon in Western countries. They are caused by either protozoal or hel-

minthic organisms. The clinical manifestation of each of these infections is as unique as the geographic distribution is varied. The diagnosis is often elusive and, in fact, sending stool samples for ova and parasite analysis is generally not cost effective in most patients with diarrhea. These tests should be used sparingly and in the appropriate patients. For instance, immunocompromised patients are at increased risk for developing a parasitic infection with cryptosporidium, and children in day care centers and travelers to Russia and Nepal are at

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increased risk for becoming infected with *Giardia*. In these populations, it is reasonable to examine the feces for ova and parasites when the index of suspicion is high. As previously mentioned, the parasitic colitides have protean manifestations, and a high index of suspicion in an at-risk individual is necessary to make the diagnosis. Protozoal infections are more likely to result in diarrhea, whereas the helminthic infections generally result in abdominal pain or obstructive symptoms, or both. In any case, medical management is the preferred mode of treatment and surgical therapy is reserved for complications such as obstruction and perforation. This article reviews the salient points regarding the epidemiology and transmission, the pathogenesis and clinical features, and the diagnosis and treatment of the protozoal and helminthic colitides.

PROTOZOAN COLITIDES

Trypanosoma Cruzi (Chagas Disease)

EPIDEMIOLOGY AND TRANSMISSION

Chagas disease is endemic throughout Central America and South America. It can also be found in the southern United States. It is increasingly prevalent in North America as the number of immigrants from Latin America increases. Up to 18 million persons in Latin America are infected, with ~300,000 new cases developing each year and more than 50,000 deaths yearly. The majority of cases are found in endemic areas of Brazil.² The vector for Chagas disease is the reduviid bug, which is also known as the kissing bug (triatomine species). This vector tends to live in houses, especially in the cracks of walls and furniture. Generally, the bug takes a blood meal from a human and at the same time defecates at the site. An intense itching causes the host to scratch and thereby transfer the egg-laden feces to the bloodstream. The parasite also infects wild animals such as armadillos, raccoons, and opossums as well as domesticated animals such as dogs, cats, and rodents. Finally, the protozoa can be acquired by blood transfusion, maternal-fetal transmission, and organ transplantation.^{3,4}

PATHOGENESIS AND CLINICAL FEATURES

The vast majority (70% to 90%) of infected individuals are asymptomatic carriers.⁵ Chagas disease generally involves the heart, nervous system, and digestive tract, with the esophagus and colon being most often affected. This disease has both an acute and a chronic phase with the acute phase lasting 4 to 8 weeks. The acute phase usually involves the heart, lymph nodes, spleen, and nervous system. A generalized enlargement secondary to inflammation of these organs is characteristic. The damage is secondary to direct tissue invasion as well as

the immune response to the organism. Generally, there are no intestinal symptoms early in the disease. The chronic phase develops over the next 40 to 50 years. This results in chronic inflammatory changes and fibrosis. Damage to the esophagus and the colon manifests itself during the chronic phase of the infection with only 6% of those infected ever developing colonic complications. The inflammation and fibrosis of the chronic phase lead to a thickened colonic wall with dilation of the distal colon. These colonic manifestations result in a decreased ability of the anal sphincter to relax as well as decreased colonic motility. The main symptom is constipation, which can become so severe that some patients even develop stasis ulcers, toxic megacolon, and/or volvulus.⁶

DIAGNOSIS AND TREATMENT

Generally, the colonic manifestations are treated symptomatically. The diagnosis is made by serology and radiography; colonoscopy is rarely useful because biopsy of the bowel wall rarely yields the parasites that have invaded the colonic wall. Initial treatment focuses on symptomatic relief, which starts with a diet high in fiber and fluids. Laxatives, enemas, and even manual disimpaction may be needed. The main medical treatments are benznidazole and nifurtimox. If an acute complication occurs, resection of the involved bowel is indicated. Surgery for chronic complications is also indicated in Chagas disease. Various operations have been described including the Duhamel procedure for disease that involves the distal rectum and extended left hemicolectomy with low coloproctostomy for disease that involves upper rectum and sigmoid colon but spares the distal rectum.⁷

Giardia lamblia

EPIDEMIOLOGY AND TRANSMISSION

Giardia is one of the two most common parasites in the United States. It is found worldwide, especially in places where there are poor sanitary conditions and a lack of water treatment facilities. A good example of this is St. Petersburg, Russia.⁸ Travelers to endemic areas, children in day care centers, and people who practice high-risk behavior (men who have sex with men) are at particularly increased risk. Despite good facilities in the United States, several million people are infected each year.⁹ Some of the natural reservoirs are dogs, cattle, and sheep, but their role in human infection is unclear.

Transmission of the parasite is generally by infection with the cysts, which can survive in a moist environment for several days. Person-to-person transmission is the commonest route, especially in institutions where hygiene is poor.^{10,11} Cysts can be killed by cooking and boiling water.¹²

PATHOGENESIS AND CLINICAL FEATURES

The *Giardia* parasite mainly infects the small intestine but is also found in the colon. The main symptoms are foul-smelling, watery diarrhea; steatorrhea; bloating; crampy abdominal pain; and nausea with emesis.¹³ Generally in parasitic illnesses, the diarrhea is secondary to organism invasion into the bowel wall and the subsequent inflammatory response. Because *Giardia* is a noninvasive organism, it is not clear why diarrhea is such a prominent symptom. The incubation period once a person is infected is as long as 2 weeks; however, 60% of those infected remain asymptomatic.¹⁴ Patients who are symptomatic often have prolonged illnesses of up to 4 weeks, and from 15% to 50% of these individuals can develop chronic giardiasis. Chronic infection is manifested by fatigue, malaise, weight loss, malabsorption, and loose stools but not diarrhea.¹⁵

DIAGNOSIS AND TREATMENT

The diagnosis is generally made by microscopic examination of stool samples. The feces generally do not have fecal leukocytes, and the peripheral blood does not exhibit eosinophilia. Microscopy of the feces is very successful at identifying the organisms. If one sample is examined, the diagnosis can be made in 50% to 70% of cases and with three samples in 90%. Recently, blood tests have been developed against *Giardia* antigens. An enzyme-linked immunosorbent assay (ELISA) is very sensitive at detecting infection more than 90% of the time.^{16,17} Controversy exists about which individuals infected with *Giardia* need to be treated. Those with symptoms should be treated with metronidazole (Flagyl) 250 mg by mouth three times per day for 5 days. Asymptomatic individuals should probably be treated only if they are immunosuppressed. An alternative to Flagyl is albendazole 400 mg daily for 5 days. This regimen is 97% effective.¹⁸

Cryptosporidium

EPIDEMIOLOGY AND TRANSMISSION

Cryptosporidium has a worldwide distribution but is far more common in developing countries than in industrialized nations. In fact, it is responsible for 1% to 3% of diarrheal illnesses in industrialized countries and 10% in developing countries.¹⁹ The main mode of transmission is the fecal-oral route from an infected individual or contaminated water supply. Transmission of the infection from sexual partners and household members and among children in day care centers is common.²⁰

PATHOGENESIS AND CLINICAL FEATURES

Cryptosporidium causes damage to the host cells by both direct invasion and an intense local immune response at the site of entry into the intestinal cells. The parasite

directly penetrates the enterocyte of both the small and large intestine and eventually alters the cell membrane, thereby damaging the epithelium. Inflammatory mediators released by the intestinal epithelium in response to the parasitic invasion contribute to the damage. The result is a secretory diarrhea that often leads to malabsorption.²¹ Infections with this parasite are more common in patients with human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS). In patients with HIV and in the immunocompetent host, the clinical picture ranges from asymptomatic carrier to life-threatening diarrheal illness. The clinical presentation is one of high-volume watery diarrhea with blood, abdominal pain, nausea, and emesis. Malnutrition and weight loss are common features in the chronic infection.^{22,23}

DIAGNOSIS AND TREATMENT

The infection is diagnosed by identification of the oocysts in the stool or a tissue biopsy. If samples are sent for ova and parasite analysis, make sure three separate samples on three consecutive days are sent. Then a methanol-fixed fecal smear will increase the sensitivity. Another technique that improves the yield is to use special staining to look for acid-fast organisms. Finally, ELISA tests have been developed and are very sensitive and specific.²⁴ Treatment of cryptosporidial infection is difficult and often unsuccessful. Treatment recommendations depend on the host immune status. In immunocompetent patients, no treatment is needed unless the patient develops chronic or prolonged symptoms that fail to resolve spontaneously. In children, nitazoxanide is used, and in the immunocompromised host highly active antiretroviral therapy (HAART) in combination with paromomycin, spiramycin, and clarithromycin has been recommended.²⁵ The prognosis for patients with an intact immune system is very good as the disease is generally self-limited. In HIV-infected individuals, at least 50% of patients develop chronic disease, and without the successful initiation or maintenance of HAART, the mean survival with chronic disease is 25 weeks and with fulminant disease 5 weeks.²⁶

Isospora

EPIDEMIOLOGY AND TRANSMISSION

Isospora infection is most common in third world countries and among travelers to those countries. In the United States, *Isospora* is relatively uncommon, but when diagnosed here it is generally found in Hispanics, immigrants, and homosexual males with HIV rather than intravenous drug abuse. Spread occurs through direct human-to-human contact with food and water contaminated with the infective oocysts.²⁷⁻³⁰

PATHOGENESIS AND CLINICAL FEATURES

Clinical infection with *Isospora* results in fever, malaise, abdominal pain, diarrhea, vomiting, and dehydration. Both the small bowel and large bowel can be affected. Symptoms are usually self-limited in immunocompetent hosts, whereas *Isospora* often becomes a chronic diarrheal disease in the immunocompromised, causing protracted watery diarrhea, malnutrition, and weight loss.³¹

DIAGNOSIS AND TREATMENT

Isospora infection is difficult to detect by standard techniques used to identify ova and parasites in stool samples, and special acid-fast stains or immunofluorescence staining must be employed. Another useful diagnostic tool is endoscopy and tissue biopsy as the organisms can be seen in mucosal biopsy samples. Once it is diagnosed, the treatment is always medical and the most common antibiotic used is Bactrim DS, one tablet by mouth four times daily for 10 days and then one tablet twice daily for 3 weeks. High relapse rates have been reported among immunocompromised patients, and suppression with Bactrim DS three times per week is recommended. Alternatively, sulfadoxine and pyrimethamine have proved effective.³²

Cyclospora

EPIDEMIOLOGY AND TRANSMISSION

Much like *Isospora*, *Cyclospora* is not common in the United States, but when diagnosed it is almost always found in immunocompromised individuals and in travelers to endemic regions in Europe, Latin America, and Asia. It is transmitted by consuming contaminated fruit, vegetables, and water.^{33–35} Direct person-to-person transmission is not common as the infective oocysts, which are found in human feces, take several days to become infective.

PATHOGENESIS AND CLINICAL FEATURES

Clinically, patients develop diarrhea, flu-like symptoms, flatulence, fatigue, and malaise. Generally, infections are self-limited, but immunodeficient patients can develop chronic symptoms.³⁶

DIAGNOSIS AND TREATMENT

Diagnosis is achieved by detecting oocysts in the stool. Once again, these organisms can be elusive and acid-fast stains and fluorescence microscopy are often required.³⁷ Treatment is always medical, and Bactrim DS one tablet by mouth twice daily for 7 days is prescribed for normal hosts; immunocompromised patients are treated similarly but then require three times weekly maintenance therapy with Bactrim DS as the agent of choice.³⁸

Balantidium coli

EPIDEMIOLOGY AND TRANSMISSION

Balantidium coli is found in the tropical and subtropical regions of Papua New Guinea, the Philippines, Latin America, and Iran. The parasite is carried by monkeys and pigs. Communities that live in close association with swine have an increased incidence of infection as the porcine reservoirs infect the human food and water supplies.³⁹

The organism is spread to humans through ingestion of cysts in contaminated water and food or from person to person. Trophozoites are very resistant to temperature and can survive outside the host for several days at low temperatures as long as they are in a moist environment.

PATHOGENESIS AND CLINICAL FEATURES

Symptoms are caused by the trophozoites invading the mucosal lining of the ileum and colon. This produces mucosal ulceration and inflammation.

As with all parasitic infections, there is a spectrum of disease from asymptomatic carrier to acute fulminant colitis. The main symptoms include diarrhea, hematochezia, abdominal pain, and weight loss in the acute form of the disease. If untreated, this can progress to toxic colitis with fever, peritonitis, perforation, and even death.

DIAGNOSIS AND TREATMENT

The diagnosis is made by isolating the trophozoites in the stool. Unlike many other parasites, these are rather large and can be seen with a hand lens or microscope and even by the naked eye on occasion. The organisms can also be seen on biopsy specimens taken during endoscopy. The biopsy specimens from ulcers found in the rectum and colon often reveal the organisms.^{40–42} In most cases, treatment is medical with tetracycline 500 mg four times per day for 10 days. Alternative treatment medications are bacitracin, ampicillin, and flagyl.⁴³ Surgery is reserved for fulminant toxic colitis with perforation or failed medical management.

HELMINTHIC COLITIDES

Schistosomiasis

EPIDEMIOLOGY AND TRANSMISSION

Schistosoma is a parasitic blood fluke that affects 200 million persons worldwide, resulting in 200,000 deaths each year.⁴⁴ Many different types of *Schistosoma* exist; three major types infect humans:

1. *Schistosoma haematobium* is found in Africa, the Middle East, and India. Infection with this species results in renal and bladder sequelae.

2. *Schistosoma mansoni* is found in sub-Saharan Africa, the Caribbean, the Middle East, and South America. Infection with this species results in intestinal and hepatic complications.
3. *Schistosoma japonicum* is found in Thailand, Indonesia, China, and the Philippines. Infection with *S. japonicum* causes intestinal and hepatic sequelae.

Infection with any of these organisms can result in schistosomiasis in people indigenous to endemic areas as well as travelers who only have a brief exposure to fresh water contaminated with the parasite.⁴⁵ The parasite is transmitted to humans who come into contact with fresh water that is contaminated with the infected larva.

PATHOGENESIS AND CLINICAL FEATURES

The parasites penetrate the skin, migrate into the blood stream, and make their way to the pulmonary capillaries. From here they make their way into the arterial circulation and finally settle in the portal and mesenteric venous circulation. They mature into adult parasites in the liver over the next 1 to 4 weeks. The parasites do not reproduce within the human host. The females produce eggs that are shed in the urine and feces of the host. The eggs hatch in fresh water and miracidium (immature larva) then finds a snail host within which to develop to adulthood. As a result, humans cannot contract the infection directly from human feces and urine.⁴⁶ Most infected individuals remain asymptomatic for the duration of their life. Of those who develop symptoms, travelers to endemic areas tend to have an acute response secondary to an immune reaction, whereas chronic complications from the disease are found in persons who live in endemic areas. This pattern of disease expression is probably due to the chronicity of infection and reinfection seen in endemic regions.⁴⁷ Symptoms from intestinal schistosomiasis develop secondary to eggs penetrating the bowel wall and causing ulceration and scarring.⁴⁸ Polyps arise secondary to granulomatous inflammation around eggs in the bowel wall and then the bowel ulcerates and strictures.⁴⁹

The main symptoms that develop from the scarring and structuring are pain, bloating, and bloody diarrhea.

DIAGNOSIS AND TREATMENT

The diagnosis is generally made by identifying the eggs using light microscopy and examining the urine and feces of the infected individual. Urine samples should be collected between 10 AM and 2 PM because this is the time of maximal egg excretion.⁵⁰ Eggs may also be found in tissue biopsies of the gastrointestinal or genitourinary tract.⁵¹ Finally, there is an ELISA that detects antischistosomal antibodies in infected individuals. As with most parasitic infections, the mainstay of

treatment is antibiotics. Praziquantel 40 to 60 mg/kg in divided doses depending on the infecting species exhibits 80% to 90% efficacy in markedly reducing infection and curing patients.⁵² Once again, surgery is limited to abdominal catastrophes secondary to perforation from obstruction or severe hemorrhage from intestinal ulceration.

Strongyloides stercoralis

EPIDEMIOLOGY AND TRANSMISSION

Strongyloides is found in most tropical and subtropical regions. In the United States this helminth is found in Appalachia and parts of the Southeast. It has become increasingly common in immigrants to the United States and military members who have been stationed in endemic areas. The parasite is transmitted by the fecal-oral route or in soil infected with feces. Generally, the parasite comes into direct contact with the hosts' skin. The larvae then penetrate the skin and migrate to the lungs through the blood stream. Larvae then go up the tracheobronchial tree, are swallowed, and eventually deposit themselves in the gastrointestinal tract.

PATHOGENESIS AND CLINICAL FEATURES

There is a broad range of clinical presentations. Clinical presentation ranges from an asymptomatic eosinophilia in the immunocompetent host to disseminated disease and sepsis in an immune compromised host. Generally, patients have mild cutaneous, gastrointestinal, and pulmonary symptoms that frequently wax and wane. The gastrointestinal symptoms are generally upper tract as the parasites usually deposit in the duodenum and jejunum. However, a high parasite burden can result in a severe enterocolitis that results in diarrhea, anorexia, nausea, and vomiting. Malabsorption is a common feature in severe infection.^{53,54}

DIAGNOSIS AND TREATMENT

Diagnosis requires a high index of suspicion in patients from endemic regions and in immunosuppressed hosts.^{55,56} The larvae are usually found in the stool on routine ova and parasite cultures. Because up to 25% of those infected have a negative stool examination, a special technique called the Baermann concentration technique has been developed. There is also serologic testing. Colonoscopy can also be utilized to make the diagnosis. Endoscopy findings show a loss of vascular pattern, edema, ulceration, and xanthomas. Treatment is with ivermectin at a dose of 200 µg/kg body weight as a single dose, with 77% efficacy versus 100% efficacy for two doses. The alternative is albendazole at 400 mg two times per day for 3 days.

Ascaris lumbricoides

EPIDEMIOLOGY AND TRANSMISSION

Ascaris is also known as the intestinal roundworm. Some estimates suggest that 25% of the world's population (1.4 billion people) is infected. The geographic distribution is ubiquitous because the eggs are sturdy and viable under a multitude of environmental conditions. In fact, eggs can live for up to 10 years in soil and are killed only by filtration and boiling of water.⁵⁷ Infected hosts are often asymptomatic and shed eggs for years.⁵⁸ There is no animal host. Infection rates are highest in the tropics, where there is year-round transmission, as opposed to drier climates, where transmission occurs mainly in the rainy moist months.⁵⁹ In the United States the prevalence in the population is 2%, but more than 30% of all cases occur in children younger than 5 years. Most of these cases are found in the rural Southeast.⁶⁰ Transmission is by ingestion of water, food, and raw fruits and vegetables contaminated with the eggs.

PATHOGENESIS AND CLINICAL FEATURES

The parasites usually inhabit the small intestine. They have a life span of 1 to 2 years. They are then ingested and the ova hatch in the small intestine, penetrate the bowel wall, and migrate to the lungs. The larvae mature within the lungs and then migrate up the tracheobronchial tree and are swallowed. They mature into adult worms.

Ascariasis affects all ages but is most common in young children aged 2 to 10. Infections tend to cluster in families. The immunocompromised do not seem to be at increased risk for infection with ascariasis.^{61,62} The majority of infections are asymptomatic. Clinically significant infections are usually secondary to a high worm load.⁶³ This helminth causes direct tissue damage as well as an immune response from the host that causes local damage. The main symptoms are abdominal discomfort, pain, anorexia, nausea, and emesis reminiscent of intestinal obstruction and malnutrition.⁶⁴ Intestinal obstruction by a mass of worms at the ileocecal valve has been reported as well. Additional complications of a high worm load are volvulus, intussusception, and perforation.⁶⁵

DIAGNOSIS AND TREATMENT

The diagnosis is established by stool microscopy. Early in the infection, eggs do not appear in the stool. Occasionally a worm is found in the feces. Eosinophilia develops in many and is usually in the 5% to 15% range but can range as high as 50%.⁶⁶ X-ray diagnosis is made either by seeing a ball of worms on a plain film with a characteristic "whirlpool" effect or on a barium enema.⁶⁷ Treatment is medical with pyrantel pamoate 11 mg/kg with a maximum 1-g single dose. This regimen is 90% effective.⁶⁸ The mainstays, however, are mebendazole

and albendazole with a single-dose therapy that is more than 97% effective.⁶⁹ Surgical therapy is reserved for complications such as volvulus, intussusception, and perforation.

Trichuris trichiura

EPIDEMIOLOGY AND TRANSMISSION

This nematode, also known as the whipworm, infects ~25% of the world population.⁷⁰ The normal geographic distribution includes warm moist tropical climates. Transmission is secondary to poor hygiene and fecal-oral mechanisms.

PATHOGENESIS AND CLINICAL FEATURES

The eggs hatch into larvae in the intestine and then take 2 to 3 months to develop into worms. They live in the intestine embedded in the mucosa. The most common location is the cecum and ascending colon in light infections, but they can also be found in the descending colon and rectum in heavy infections.⁷¹ Infections are usually asymptomatic, but when they become symptomatic patients develop loose stools with blood and mucus. If the infection is severe, patients can develop secondary anemia, nocturnal stooling, dysentery, and even colitis. Rectal prolapse is also common.

DIAGNOSIS AND TREATMENT

The diagnosis is made by identifying the barrel-shaped eggs in the stool. Endoscopy can show worms embedded in bowel wall. Treatment is with mebendazole or albendazole. Surgery is reserved for rectal prolapse.

Enterobius vermicularis

EPIDEMIOLOGY AND TRANSMISSION

Enterobius vermicularis is commonly known as the pinworm. It is found worldwide, in both temperate and tropical climates. It is the most common helminth infection in the United States and Western Europe. It is most commonly found in schoolchildren 5 to 10 years old. Humans are the only natural host, and close, crowded conditions aid the transmission from person to person. The main route of spread is the fecal-oral mechanism.

PATHOGENESIS AND CLINICAL FEATURES

Adult worms live in the cecum and appendix. The female migrates onto the perianal skin at night to deposit her eggs. The larvae become infective during this 6-hour period. Most infections are asymptomatic, but when symptoms develop the most common symptom is pruritus ani.⁷² The symptoms that develop are generally secondary to the inflammatory changes and excoriation

from scratching at night.⁷³ Abdominal symptoms can develop and generally do so in patients with a high worm burden. The symptoms are then abdominal pain, nausea, and vomiting. Not infrequently, appendicitis and eosinophilic enterocolitis ensue in these patients.^{74,75}

DIAGNOSIS AND TREATMENT

The diagnosis is made by the scotch tape test. The bean-shaped eggs can be seen under the microscope. The highest diagnostic yield is obtained by performing the scotch tape test first thing in the morning or during the night. One swab is 50% diagnostic, whereas three swabs are 90% diagnostic. The eggs are not found in the stool, so standard ova and parasite studies are not useful. Medical treatment is with mebendazole 100 mg as a single dose. This results in a 95% cure rate. An alternative is albendazole 100 mg if younger than 2 years and 400 mg if older than 2 years. Treatment should be repeated at 2 weeks and then is 100% curative. Pyrantel pamoate 11 mg/kg is also 90% effective. Surgery is reserved for patients with appendicitis.

CONCLUSIONS

Parasitic infections of the gastrointestinal tract are a widespread worldwide problem with a multitude of clinical presentations and a varied spectrum of disease. Infections range from asymptomatic carrier to fulminant sepsis and death. Parasitic infections are difficult to diagnose, and making an accurate diagnosis requires a high index of suspicion. The protozoal colitides more frequently cause diarrheal illnesses and malnutrition. Surgical therapy is rarely indicated for protozoal infections with the exception of chronic constipation and obstruction in Chagas disease and toxic colitis in *Balantidium coli* infections. The helminthic colitides, on the other hand, generally result in obstructive symptoms, not diarrheal illness. The obstructive symptoms can be caused by stricture secondary to local inflammation or by a high worm burden resulting in appendicitis or even a bowel obstruction. Medical management of all protozoal infections is preferred, and surgical therapy is reserved for complications or failed medical management.

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