Parasitic Infections of the Gastrointestinal Tract

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Introduction
Parasites that infect the gastrointestinal tract can be divided into the protozoa and the helminths. The helminths consist of nematodes (roundworms) and platyhelminths (flatworms); platyhelminths are either cestodes (tapeworms) or trematodes (flukes). The importance of establishing a good working relationship with a laboratory that specializes in parasitology cannot be overemphasized. Many commercial laboratories do "batch" work on purged stools or perform serologic tests with unreliable antibodies, leading to spurious results. Stool kits that contain preservatives can eliminate missed diagnoses caused by the examination of specimens that are not fresh, not preserved, and therefore lost. We refer readers to two excellent articles [1,2] that review the treatment of parasitic diseases, including the diseases covered here.

Protozoa
Protozoa are unicellular organisms that cause significant disease in both healthy and immunocompromised hosts. Ingestion of just a few protozoa may result in significant symptoms and debility. Patients with AIDS or other immunodeficiency syndromes are at particular risk for overwhelming or chronic infections with some of these pathogens.

Amebiasis
Entamoeba histolytica infection causes colitis or hepatic abscess in up to 100 million people worldwide per year. Up to 10% of the world's population has E. histolytica infection, and 100,000 people each year die of it. This infection is prevalent in areas of crowding, in persons of lower socioeconomic status, and in places with poor sanitation. The prevalence of amebiasis in the United States is about 4%; male homosexual patients and institutionalized persons have the highest risk for it. The severity of disease worsens with underlying cancer, steroid use, malnutrition, pregnancy, and young age [3].

The life cycle of E. histolytica involves two stages: cyst (infective) and trophozoite (invasive). Ingested cysts are resistant to gastric acid and pass into the small bowel, where they excyst as trophozoites. These pass into the colon, where they feed on bacteria and cellular debris. Trophozoites expelled in the stool degenerate rapidly, but trophozoites may encyst in the colon, and these cysts may remain viable for weeks to months after being passed in the stool.

A specific adherence protein allows the trophozoite to bind to colonic mucus, epithelial cells, and neutrophils. Trophozoites also produce various proteolytic enzymes and hemolysins, causing cytolysis of cells and surrounding tissue [4]. Cell-mediated immunity may play a role in preventing or limiting the severity of recurrence after treatment. Invasion into the bowel wall may lead to hematogenous spread to the liver, lungs, and brain.

Symptomatic disease occurs in only 10% of infected patients [3]. Clinical manifestations of intestinal disease range from asymptomatic carriage to acute or chronic colitis, perianal ulceration, ameboma, dysentery, fulminating colitis, toxic megacolon, and perforation. Extraintestinal amebiasis most often involves the liver. Whereas at least 75% of patients with liver abscess have stool cultures positive for ameba, up to 50% have no clinical history of diarrhea before presentation [5]. The clinical presentation of hepatic amebiasis includes fever, weight loss, right upper quadrant pain, right shoulder or scapular pain, and pleuritic pain. Extension into the lung produces cough, dyspnea, and pleuritic chest pain. Laboratory tests show leukocytosis with a leukocyte count of more than 10,000 mm$^3$, mild anemia, an elevated alkaline phosphatase level, and a high sedimentation rate.

The characteristic endoscopic appearance of amebiasis includes discrete, punctate, hemorrhagic ulcers and normal intervening mucosa; trophozoites are seen on histologic samples from an ulcer's edge (Fig. 1). Most cases can be
Richelle of the peritoneum or pericardium. Liver abscesses may be needed to rule out pyogenic abscess. The cyst fluid is sterile and shows trophozoites. Amebic liver abscesses appear on ultrasonography or computed tomography as inhomogeneous, single or multiple cystic masses with well-defined borders. Amebomas are usually found in the rectosigmoid or cecum and are caused by a chronic granulomatous reaction with trophozoites within the mass; lesions also may be annular and indistinguishable from colon cancer on barium enema. The inflamed cecum may have a conical appearance, but the ileum is never involved.

The diagnosis of intestinal amebiasis is made by examination of three fresh or properly preserved stool specimens or by the discovery of trophozoites on biopsy specimens. Trophozoites may show characteristic hemophagocytosis, which is specific for E. histolytica. Serologic tests for amebiasis are highly sensitive and specific for E. histolytica, becoming positive 1 week after infection [3]. The cysts of Entamoeba dispar, a species that is morphologically identical to E. histolytica but nonpathogenic, may be confused with E. histolytica cysts on examination of the stool. However, patients with E. dispar do not have erythrocytes in their stool and do not have positive results on serologic testing. If a hepatic cyst is seen on ultrasonography or computed tomography, amebic liver abscess can be diagnosed by serologic and stool examination. Percutaneous aspiration of the cyst may be necessary for diagnosis of two sets of patients: 1) residents of areas where E. dispar is endemic, who are likely to have positive serologic findings; and 2) travelers in whom the results of serologic testing are not yet positive. The cyst fluid is sterile and shows trophozoites only if the edge of the cyst is sampled.

The treatment of intestinal and hepatic amebiasis is summarized in Table 1. Percutaneous drainage of amebic liver abscesses may be needed to rule out pyogenic abscess or to prevent rupture into the peritoneum or pericardium. Rupture into the peritoneum occurs in 5% of patients, usually patients with left lobe abscesses, and manifests as fever and a rigid, distended abdomen. Pericardial rupture is associated with fever, abdominal pain progressing to chest pain, congestive heart failure, and a pericardial friction rub.

Giardiasis
Giardia lamblia, or Giardia duodenalis, is a flagellated protozoan found worldwide [6]. Outbreaks of G. lamblia infection have been described in areas with infected water supplies, but hikers, campers, male homosexual persons, and children in day care centers have the greatest risk for this infection. Patients with common variable immunodeficiency and children with X-linked agammaglobulinemia are predisposed to recurrent infection. The ingestion of as few as 10 cysts may cause symptomatic infection. In the duodenum, cysts excyst and release two trophozoites, both of which attach to the mucosa with a ventral sucking disk. Invasion is unusual, although the organism has been found in the bile and gallbladder. Cysts, and occasionally trophozoites, are passed in the stool erratically. In the environment, only the cysts are viable and infectious.

Pathogenic mechanisms include direct irritation of the mucosa by trophozoite adherence and involve humoral and cellular host immune responses [7]. Loss of disaccharidas leads to lactose intolerance in 20% to 40% of cases; folate and vitamin B12 deficiency are unusual. A reversible pancreatic insufficiency causing malabsorption in the presence of intestinal giardiasis was recently described [8]. Symptoms can be mild and nonspecific (increased flatulence or belching) or more significant (colicky pain, cramps, bloating, fatigue, nausea, and diarrhea). Signs of malabsorption or vitamin deficiency may be present; growth retardation can occur in children. Laboratory findings are not specific for Giardia species infection and, except for involvement of the proximal intestine, radiographic findings may be indistinguishable from those seen with other malabsorption syndromes. Giardia species infection has no specific endoscopic appearance, although scalloping or loss of duodenal folds may be seen in severe cases.

Because shedding of cysts varies greatly among patients, up to three stool specimens should be examined. The diagnostic yield is increased by testing the stool for Giardia species antigen. If suspicion of giardiasis is high and these initial tests have negative results, upper endoscopy with biopsy and duodenal aspiration may be helpful. Histopathology may show trophozoites on the luminal surface of the villi, the architecture of which can range from normal to total villus atrophy (the latter is seen especially in patients with immunodeficiency syndromes) [9].

Nutritional deficiencies should be addressed immediately. Metronidazole, 250 mg three times daily for 5 days, usually eradicates infection in normal hosts, but this treatment may need to be continued for 2 to 6 months in patients with immune deficiencies. Sexual partners of infected persons, family members of infected persons, and...