131 - 236 Protozoal Intestinal Infections and Trichomoniasis

236 Protozoal Intestinal Infections and Trichomoniasis

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Protozoal Intestinal

Infections and

Trichomoniasis PROTOZOAL INFECTIONS ■ ■GIARDIASIS Giardia duodenalis (also known as G. lamblia or G. intestinalis) is a cosmopolitan protozoal parasite that inhabits the small intestines of humans and other mammals. Giardiasis is one of the most com mon parasitic diseases in both developed and developing countries worldwide, causing both endemic and epidemic intestinal disease and diarrhea. In resource-constrained areas with limited access to adequate sanitation and safer water, prevalence rates of giardiasis may be 20–40%, especially among young children. Life Cycle and Epidemiology (Fig. 236-1) Infection follows the ingestion of environmentally hardy cysts, which excyst in the small PART 5 Infectious Diseases Excystation follows exposure to stomach acid and intestinal proteases, releasing trophozoite forms that multiply by binary fission and reside in the upper small bowel adherent to enterocytes. Causes: Asymptomatic infection, acute diarrhea, or chronic diarrhea and malabsorption. Small bowel may demonstrate villous blunting, crypt hypertrophy, and mucosal inflammation. Encystation occurs under conditions of bile salt concentration changes and alkaline pH. Smooth-walled cysts can contain two trophozoites. Cysts are ingested (10–25 cysts) in contaminated water or food or by direct fecal-oral transmission (as in day-care centers). Cysts can survive in the environment (up to several weeks in cold water). They may also infect nonhuman mammalian species. Cysts and trophozoites are passed in the stool into the environment. FIGURE 236-1 Life cycle of Giardia. (Reproduced with permission from RL Guerrant et al [eds]: Tropical Infectious Diseases: Principles, Pathogens and Practice, 2nd ed. Elsevier, 2006.)

FIGURE 236-2 Flagellated, binucleate Giardia trophozoites. intestine, releasing flagellated trophozoites (Fig. 236-2) that multiply by binary fission. Giardia remains a pathogen of the proximal small bowel and does not disseminate hematogenously. Trophozoites remain free in the lumen or attach to the mucosal epithelium by means of a ventral sucking disk. As a trophozoite encounters altered conditions, it forms a morphologically distinct cyst, which is the stage of the para site usually found in the feces. Trophozoites may be present and even predominate in loose or watery stools, but it is the resistant cyst that survives outside the body and is responsible for transmission. Cysts do not tolerate heating or desiccation, but they do remain viable for months in cold fresh water. The number of cysts excreted varies widely but can approach 107 per gram of stool. Ingestion of as few as 10 cysts is sufficient to cause infection in humans. Because cysts are infectious when excreted, person-to-person transmission occurs where fecal hygiene is poor. Giardiasis is especially prevalent in day-care centers; person-to-person spread also takes place in other institutional settings with poor fecal hygiene and during anal– oral contact. If food is contaminated with Giardia cysts after cooking or preparation, foodborne transmission can occur. Waterborne transmis sion accounts for episodic infections (e.g., in campers and travelers) and for major epidemics in metropolitan areas. Surface water, ranging from mountain streams to large municipal reservoirs, can become contaminated with fecally derived Giardia cysts. The efficacy of water as a means of transmission is enhanced by the small infectious inocu lum of Giardia, the prolonged survival of cysts in cold water, and the resistance of cysts to killing by routine chlorination methods that are adequate for controlling bacteria. Viable cysts can be eradicated from water by either boiling or filtration. In the United States, Giardia (like Cryptosporidium; see below) can cause waterborne epidemics of gastroenteritis. Giardia is common in developing countries, and infections may be acquired by travelers. There are several recognized genotypes or assemblages of G. duode nalis. Human infections are due to assemblages A and B, whereas other assemblages are more common in other animals, including cats and dogs. Like beavers from reservoirs implicated in epidemics, dogs and cats have been found to be infected with assemblages A and B; this finding suggests both that these animals may have been infected from human sources and that they might be sources of further human infections. Giardiasis, like cryptosporidiosis, creates a significant economic burden because of the costs incurred in the installation of water filtration systems required to prevent waterborne epidemics, in the management of epidemics that involve large communities, and in the evaluation and treatment of endemic infections. Pathophysiology The reasons that some, but not all, infected patients develop clinical manifestations and the mechanisms by which Giardia causes alterations in small-bowel function are largely unknown. Although trophozoites adhere to the epithelium, they are not invasive but may elicit apoptosis of enterocytes, epithelial bar rier dysfunction, and epithelial cell malabsorption and secretion.

TABLE 236-1 Diagnosis of Intestinal Protozoal Infections PARASITE STOOL O+P FECAL ACID-FAST STAIN FECAL ANTIGEN IMMUNOASSAYS FECAL NAATS OTHER Giardia + + + DFA Cryptosporidium ± + + + DFA Cystoisospora ± + + Cyclospora ± + + Dientamoeba ± + + Balantidium + Microsporidia – + Special fecal stains, tissue biopsies Abbreviations: DFA, direct immunofluorescence assay; NAATs, nucleic acid amplification tests; O+P, conventional ova and parasites. Consequent lactose intolerance and, in a minority of infected adults and children, significant malabsorption are clinical signs of the loss of epithelial brush-border enzyme activities. In most infections, the morphology of the bowel is unaltered; however, in chronically infected, symptomatic patients, the histopathologic findings (including flattened villi) and the clinical

manifestations at times resemble those of tropical sprue and gluten-sensitive enteropathy. The pathogenesis of diarrhea in giardiasis is not known. The natural history of Giardia infection varies markedly. Infections may be asymptomatic, transient, recurrent, or chronic. G. duodenalis parasites vary genotypically, and such variations might contribute to different courses of infection. Parasite as well as host factors may be important in determining the course of infection and disease. Both cellular and humoral responses develop in human infections, but their precise roles in disease pathogenesis and/or control of infection are unknown. Because patients with hypogammaglobulinemia suffer from prolonged, severe infections that are poorly responsive to treatment, humoral immune responses appear to be important. The greater sus ceptibilities of the young than of the old and of newly exposed persons than of chronically exposed populations suggest that at least partial protective immunity may develop. Clinical Manifestations Disease manifestations of giardiasis range from asymptomatic carriage to fulminant diarrhea and malabsorption. Most infected persons are asymptomatic, but in epidemics, the propor tion of symptomatic cases may be higher. Symptoms may develop sud denly or gradually. In persons with acute giardiasis, symptoms develop after an incubation period that lasts at least 5–6 days and usually 1–3 weeks. Prominent early symptoms include diarrhea, abdominal pain, bloating, belching, flatus, nausea, and vomiting. Although diarrhea is common, upper intestinal manifestations such as nausea, vomiting, bloating, and abdominal pain may predominate. The duration of acute giardiasis is usually >1 week, although diarrhea often subsides. Individuals with chronic giardiasis may present with or without having experienced an antecedent acute symptomatic episode. Diarrhea is not necessarily prominent, but increased flatus, loose stools, sulfurous belching, and (in some instances) weight loss occur. Symptoms may be continual or epi sodic and may persist for years. Some persons who have relatively mild symptoms for long periods recognize the extent of their discomfort only in retrospect. Fever, the presence of blood and/or mucus in the stools, and other signs and symptoms of colitis are uncommon and suggest a different diagnosis or a concomitant illness. Symptoms tend to be inter mittent yet recurring and gradually debilitating, in contrast with the acute disabling symptoms associated with many enteric bacterial infec tions. Because of the less severe illness early on and the propensity for chronic infections, patients may seek medical advice late in the course of the illness; however, disease can be severe, resulting in malabsorption, weight loss, growth retardation in children, and dehydration. A number of extraintestinal manifestations have been described, such as urticaria, anterior uveitis, and arthritis; whether these are caused by giardiasis or concomitant processes is unclear. Giardiasis can be severe in patients with hypogammaglobulinemia and can complicate other preexisting intestinal diseases, such as that occurring in cystic fibrosis. In patients with AIDS, Giardia can cause enteric illness that is refractory to treatment.

Diagnosis (Table 236-1) Giardiasis is diagnosed by detection of parasite antigens in the feces, by identification of cysts in the feces or of trophozoites in the feces or small intestines, or by nucleic acid amplifi cation tests (NAATs). Cysts are oval and measure 8–12 μm × 7–10 μm. Mature cysts characteristically contain four nuclei. Trophozoites are pear-shaped, dorsally convex, flattened parasites with two nuclei and four pairs of flagella (Fig. 236-2). The diagnosis is sometimes difficult to establish. Direct examination of fresh or properly preserved stools as well as concentration methods can be used. Because cyst excretion is variable and may be undetectable at times, repeated examination of stool, sampling of duodenal fluid, and biopsy of the small intestine may be required to detect the parasite. Tests for parasitic antigens in stool are often more sensitive than or comparable to microscopic examina tion, are easier to perform, have a more rapid turn-around

time, are often cost-comparable, and are often the first-line test in many institu tions. NAATs are also highly sensitive. CHAPTER 236 TREATMENT Giardiasis Protozoal Intestinal Infections and Trichomoniasis

Cure rates with metronidazole (250–500 mg thrice daily for 5 days) are usually >90%. Tinidazole (2 g once by mouth) may be more effective than metronidazole. Nitazoxanide (500 mg twice daily for 3 days) is an alternative agent for treatment of giardiasis. Paro momycin, an oral aminoglycoside that is not well absorbed, can be given to symptomatic pregnant patients, although information is limited on how effectively this agent eradicates infection. Alben dazole and mebendazole also can be used as alternative agents, although their efficacy is lower than that of single-dose tinidazole. Quinacrine, secnidazole, ornidazole, and furazolidone are other alternative agents that have demonstrated some efficacy. Almost all patients respond to therapy and are cured, although some with chronic giardiasis experience delayed resolution of symptoms after eradication of Giardia. For many of the latter patients, residual symptoms probably reflect delayed regeneration of intestinal brush-border enzymes. Continued infection should be documented by stool examinations before treatment is repeated. Patients who remain infected after repeated treatments should be evaluated for reinfection through family members, close personal contacts, and environmental sources as well as for hypogamma globulinemia. In cases refractory to multiple treatment courses, prolonged therapy with metronidazole (750 mg thrice daily for 21 days) or therapy with varied combinations of multiple agents has been successful. Prevention Giardiasis can be prevented by consumption of uncon taminated food and water and by personal hygiene during the pro vision of care for infected children. Boiling or filtering potentially contaminated water prevents infection. ■ ■CRYPTOSPORIDIOSIS The coccidian parasite Cryptosporidium causes diarrheal disease that is self-limited in immunocompetent human hosts but can be severe in persons with AIDS or other forms of immunodeficiency. Two species

of Cryptosporidium, C. hominis and C. parvum, cause most human infections.

Life Cycle and Epidemiology Cryptosporidium species are widely distributed in the world. In resource-constrained areas, cryptosporidiosis is the second most common cause of moderate to severe diarrhea during the first two years of life (following rotavirus). Cryptosporidiosis is acquired by the consumption of oocysts (50% infectious dose: ~10–100 C. hominis/parvum oocysts in nonimmune individuals), which excyst to liberate sporozoites that in turn enter and infect intestinal epithelial cells. The parasite’s further develop ment involves both asexual and sexual cycles, which produce forms capable of infecting other epithelial cells and of generating oocysts that are passed in the feces. Cryptosporidium species infect a num ber of animals, and C. parvum can spread from infected animals to humans. Since oocysts are immediately infectious when passed in feces, person-to-person transmission takes place in day-care centers and among household contacts and medical providers. Waterborne transmission (especially that of C. hominis) accounts for infections in travelers and for common-source epidemics. Oocysts are quite hardy and resist killing by routine chlorination. Both drinking water and recreational water (e.g., pools, waterslides) have been increasingly recognized as sources of infection. Pathophysiology Although intestinal epithelial cells harbor cryp tosporidia in an intracellular vacuole, the means by which secretory diarrhea is elicited remain uncertain. No characteristic pathologic changes are found by biopsy. The distribution of infection can be spotty within the principal site of infection, the small bowel. Cryptosporidia are found in the pharynx, stomach, and large bowel of some patients and at times in the respiratory tract. Especially in patients with AIDS, involvement of the biliary tract can cause papillary stenosis,

sclerosing cholangitis, or cholecystitis. PART 5 Infectious Diseases Clinical Manifestations Asymptomatic infections can occur in both immunocompetent and immunocompromised hosts. In immu nocompetent persons, symptoms develop after an incubation period of ~1 week and consist principally of watery nonbloody diarrhea, sometimes in conjunction with abdominal pain, nausea, anorexia, fever, and/or weight loss. In these hosts, the illness usually subsides after 1–2 weeks. In contrast, in immunocompromised hosts (especially those with AIDS and CD4+ T-cell counts <100/μL), diarrhea can be chronic, persistent, and remarkably profuse, causing clinically signifi cant fluid and electrolyte depletion. Stool volumes may range from 1 to 25 L/d. Weight loss, wasting, and abdominal pain may be severe. Bili ary tract involvement can manifest as mid-epigastric or right-upperquadrant pain. Diagnosis (Table 236-1) Evaluation starts with fecal examination for small oocysts, which are smaller (4–5 μm in diameter) than the fecal stages of most other parasites. Because conventional stool exami nation for ova and parasites (O+P) does not detect Cryptosporidium, specific testing must be requested. Detection is enhanced by evaluation of stools (obtained on multiple days) by several techniques, including modified acid-fast and direct immunofluorescent stains and enzyme immunoassays. NAATs also are useful. Cryptosporidia can also be identified by light and electron microscopy at the apical surfaces of intestinal epithelium from biopsy specimens of the small bowel and, less frequently, the large bowel. TREATMENT Cryptosporidiosis Nitazoxanide, approved by the U.S. Food and Drug Administration (FDA) for the treatment of cryptosporidiosis, is available in tablet form for adults (500 mg twice daily for 3 days) and as an elixir for children. This agent has not been effective for the treatment of immunosuppressed patients or HIV-infected patients, in whom improved immune status due to antiretroviral therapy can lead to amelioration of cryptosporidiosis. Otherwise, treatment includes

supportive care with replacement of fluids and electrolytes and administration of antidiarrheal agents. Biliary tract obstruction may require papillotomy or T-tube placement. Prevention requires minimizing exposure to infectious oocysts in human or animal feces. Use of submicron water filters may minimize acquisition of infection from drinking water. ■ ■CYSTOISOSPORIASIS The coccidian parasite Cystoisospora belli (formerly referred to as Isospora belli) causes human intestinal disease. Infection is acquired by the consumption of oocysts, after which the parasite invades intestinal epithelial cells and undergoes both sexual and asexual cycles of devel opment. Oocysts excreted in stool are not immediately infectious but must undergo further maturation. Although C. belli infects many animals, little is known about the epidemiology or prevalence of this parasite in humans. It is most com mon in tropical and subtropical countries. Acute infections can begin abruptly with fever, abdominal pain, and watery nonbloody diarrhea and can last for weeks or months. In patients who have AIDS or are immunocompromised for other reasons, infections often are not selflimited but rather resemble cryptosporidiosis, with chronic, profuse watery diarrhea. Eosinophilia, which is not found in other enteric protozoan infections, may be detectable in immunocompetent hosts. The diagnosis (Table 236-1) is usually made by detection of the large (~25 μm) oocysts in stool by modified acid-fast staining. Oocyst excre tion may be low-level and intermittent; if repeated stool examinations are unrevealing, sampling of duodenal contents by aspiration or smallbowel biopsy (often with electron microscopic examination) may be necessary. NAATs are effective newer diagnostic tools. TREATMENT Cystoisosporiasis Trimethoprim-sulfamethoxazole (TMP-SMX, 160/800 mg two times daily for 7–10 days; and, for HIV-infected patients, then continuing three times daily for 3–4 weeks) is effective. For patients intolerant of sulfonamides, pyrimethamine (50–75 mg/d) or cipro floxacin 500 mg two times a day can be used.

Relapses can occur in persons with AIDS and necessitate maintenance therapy with TMP-SMX (160/800 mg three times per week). ■ ■CYCLOSPORIASIS Cyclospora cayetanensis, a cause of diarrheal illness, is globally distrib uted: illness due to C. cayetanensis has been reported in the United States, Asia, Africa, Latin America, and Europe. The epidemiology of this parasite has not yet been fully defined, but waterborne transmission and foodborne transmission (e.g., by basil, sweet peas, and imported raspberries) have been recognized. The full spectrum of illness attribut able to Cyclospora has not been delineated. Some infected patients may be without symptoms, but many have diarrhea, flulike symptoms, and flatulence and belching. The illness can be self-limited, can wax and wane, or, in many cases, can involve prolonged diarrhea, anorexia, and upper gastrointestinal symptoms, with sustained fatigue and weight loss in some instances. Diarrheal illness may persist for >1 month. Cyclospora can cause enteric illness in patients infected with HIV. The parasite is detectable in epithelial cells of small-bowel biopsy samples and elicits secretory diarrhea by unknown means. The absence of fecal blood and leukocytes indicates that disease due to Cyclospora is not caused by destruction of the small-bowel mucosa. The diagno sis (Table 236-1) can be made by detection of spherical 8- to 10-µm oocysts in the stool, although routine stool O+P examinations are not sufficient. Specific fecal examinations must be requested to detect the oocysts, which are variably acid-fast and are fluorescent when viewed with ultraviolet light microscopy. NAATs are sensitive. Cyclospo riasis should be considered in the differential diagnosis of prolonged diarrhea, with or without a history of travel by the patient to other countries.

TREATMENT Cyclosporiasis Cyclosporiasis is treated with TMP-SMX (160/800 mg twice daily for 7–10 days). HIV-infected patients may require longer treatment courses, may experience relapses after treatment, and thus may require longer-term suppressive maintenance therapy. Nitazoxanide and ciprofloxacin may be alternative agents in patients unable to receive TMP-SMX. ■ ■MICROSPORIDIOSIS Microsporidia are obligate intracellular spore-forming protozoa that infect many animals and cause disease in humans, especially as opportunistic pathogens in AIDS. Microsporidia are members of a distinct phylum, Microspora, which contains hundreds of genera and thousands of species. The various microsporidia are differentiated by their developmental life cycles, ultrastructural features, and molecular Microsporidia Enterocytozoon bieneusi, Encephalitozoon spp., etc. Intracellular multiplication via merogony and sporogony E. bieneusi in epithelial cell Polar tubule pierces host epithelial cell, injects sporoplasm While E. bieneusi is primarily in the gastrointestinal tract, other species may invade the lung or eye or disseminate to cause: Presumed ingestion or respiratory acquisition of spores Person-to-person, zoonotic, waterborne, or food-borne transmission? Diagnostic spores present in stool, urine, respiratory fluids, cerebrospinal fluid, or various tissue specimens FIGURE 236-3 Life cycle of microsporidia. (Reproduced with permission from RL Guerrant et al [eds]: Tropical Infectious Diseases: Principles, Pathogens and Practice, 2nd ed. Elsevier 2006.)

taxonomy based on ribosomal RNA. The complex life cycles of the organisms result in the production of infectious spores (Fig. 236-3). Currently, at least 17 species of microsporidia are recognized as causes of human disease, most commonly of the genera Enterocytozoon and Encephalitozoon. Although some microsporidia are probably prevalent causes of self-limited or asymptomatic infections in immunocompe tent patients, little is known about how microsporidiosis is acquired.

Microsporidiosis is most common among patients with AIDS, less common among patients with other types of immunocompromise, and rare among immunocompetent hosts. In patients with AIDS, intestinal infections with Enterocytozoon bieneusi and Encephalitozoon intestinalis are recognized to contribute to chronic diarrhea and wasting; these infections have been found in 10–40% of patients with chronic diar rhea. Both organisms have been found in the biliary tracts of patients with cholecystitis. E. intestinalis may also disseminate to cause fever, diarrhea, sinusitis, cholangitis, and bronchiolitis. In patients with AIDS, Encephalitozoon hellem has caused superficial keratoconjunc tivitis as well as sinusitis, respiratory tract disease, and disseminated Encephalitozoon intestinalis in epithelial cells, endothelial cells, or macrophages CHAPTER 236 Protozoal Intestinal Infections and Trichomoniasis
Spore-laden host epithelial cells sloughed into lumina of gastrointestinal, respiratory, or genitourinary tract Chronic diarrhea Cholangitis Sinusitis Bronchitis Nephritis Cystitis/prostatitis Keratoconjunctivitis Encephalitis Sloughed cells degenerate; spores shed in bodily fluids

infection. Myositis due to Pleistophora has been documented. Nosema, Vittaforma, and Microsporidium have caused stromal keratitis associ ated with trauma in immunocompetent patients.

Microsporidia are small gram-positive organisms with mature spores measuring 0.5–2 μm × 1–4 μm. Diagnosis of microsporidial infections in tissue often requires electron microscopy, although intra cellular spores can be visualized by light microscopy with hematoxylin and eosin, Giemsa, or tissue Gram’s stain. Light microscopy with a modified trichrome stain or fluorescent assays can be used on stool, tis sue, or urine. NAATs are useful for diagnosis and speciation. Definitive therapies for microsporidial infections remain to be established. For superficial keratoconjunctivitis due to E. hellem, E. cuniculi, E. intesti nalis, and E. bieneusi, topical therapy with fumagillin suspension has shown promise (Chap. 229). For enteric infections with E. intestinalis in HIV-infected patients, therapy with albendazole may be efficacious (Chap. 229). ■ ■OTHER INTESTINAL PROTOZOA Balantidiasis Balantidium coli is a large ciliated protozoal parasite that can produce a spectrum of large-intestinal disease analogous to amebiasis. The parasite is widely distributed in the world. Since it infects pigs, cases in humans are more common where pigs are raised. Infective cysts can be transmitted from person to person and through water, but many cases are due to the ingestion of cysts derived from porcine feces in association with slaughtering, with use of pig feces for fertilizer, or with contamination of water supplies by pig feces. Ingested cysts liberate trophozoites, which reside and replicate in the large bowel. Many patients remain asymptomatic, but some have persisting intermittent diarrhea, and a few develop more fulminant dysentery. In symptomatic individuals, the pathology in the bowel— both gross and microscopic—is similar to that seen in amebiasis, with varying degrees of mucosal invasion, focal necrosis, and ulceration. Balantidiasis, unlike amebiasis, only rarely spreads hematogenously to other organs. The diagnosis is made by detection of the parasite in stool or sampled colonic tissue. Tetracycline (500 mg four times daily for

10 days) is an effective therapeutic agent. Metronidazole is an alterna tive agent. PART 5 Infectious Diseases Blastocystosis Blastocystis hominis is an organism of uncertain pathogenicity. Some patients who pass B. hominis in their stools are asymptomatic, whereas others have diarrhea and associated intesti nal symptoms. Diligent evaluation reveals other potential bacterial, viral, or protozoal causes of diarrhea in some but not all patients with symptoms. Because the

pathogenicity of B. hominis is uncertain and because therapy for Blastocystis infection is neither specific nor uni formly effective, patients with prominent intestinal symptoms should be fully evaluated for other infectious causes of diarrhea. If diarrheal symptoms associated with Blastocystis are prominent, metronidazole (500–750 mg thrice daily for 5–10 days) or tinidazole (2 g once) can be tried. Alternative agents include paromomycin, TMP-SMX, and nitazoxanide. Dientamoebiasis Dientamoeba fragilis is unique among intestinal protozoa in that it has a trophozoite stage but not a cyst stage. How trophozoites survive to transmit infection is not known. When symp toms develop in patients with D. fragilis infection, they are generally mild and include intermittent diarrhea, abdominal pain, and anorexia. The diagnosis is made by the detection of trophozoites in stool; the lability of these forms accounts for the greater yield when fecal samples are preserved immediately after collection. NAATs are more sensitive than fecal microscopy. Paromomycin (25–35 mg/kg per day in three doses for 7 days) or metronidazole (500–750 mg three times daily for 10 days) is appropriate for treatment. Tetracycline, doxycycline, tinida zole, secnidazole, and ornidazole are alternative agents. Sarcocystosis Humans can be the definitive or dead-end inter mediate host for Sarcocystis spp. sarcocystoses are zoonotic infections. Humans can act as definitive hosts (the host in which a parasite com pletes its sexual life cycle) following consumption of undercooked beef

or pork that contain the protozoa Sarcocystis hominis or S. suihominis, respectively. In this setting, humans can develop intestinal symptoms including nausea, vomiting, and diarrhea that is often self-resolving within a few days. Human intestinal infection can result in transient shedding of infectious oocysts that are consumed by cattle or pigs in the environment, and in these hosts the protozoa encyst in muscle tissue, allowing completion of the life cycle when these tissues are con sumed by humans. Separately, humans can develop a transient myositis with peripheral eosinophilia following ingestion of oocysts of S. nesbitti shed by snakes. Intestinal sarcocystosis can be detected by stool exami nation, although oocysts are not usually detectable during the diarrheal phase. Intestinal sarcocystosis is self-limited and treated supportively. TRICHOMONIASIS Various species of trichomonads can be found in the mouth (in associ ation with periodontitis) and occasionally in the gastrointestinal tract. Trichomonas vaginalis—one of the most prevalent protozoal parasites in the United States—is a pathogen of the genitourinary tract and a major cause of symptomatic vaginitis (Chap. 141). Life Cycle and Epidemiology T. vaginalis is a pear-shaped, actively motile organism that measures about 10 × 7 μm, replicates by binary fission, and inhabits the lower genital tract of females and the urethra and prostate of males. There are 2–3 million infections each year in the United States. While the organism can survive for a few hours in moist environments and could be acquired by direct contact, person-to-person venereal transmission accounts for virtually all cases of trichomoniasis. Its prevalence is greatest among persons with mul tiple sexual partners and among those with other sexually transmitted diseases (Chap. 141). Clinical Manifestations Many men infected with T. vaginalis are asymptomatic, although some develop urethritis and a few have epididymitis or prostatitis. In contrast, infection in women, which has an incubation period of 5–28 days, is more often symptomatic and manifests with malodorous vaginal discharge (often yellow), vulvar erythema and itching, dysuria or urinary frequency (in 30–50% of patients), and dyspareunia. These manifestations, however, do not clearly distinguish trichomoniasis from other types of infectious vaginitis. Diagnosis Detection of motile trichomonads by microscopic exam ination of wet mounts of vaginal or prostatic secretions has been the conventional means of diagnosis. Although this approach provides an immediate diagnosis, its sensitivity for the detection of T. vaginalis is only ~50–60% in routine evaluations of vaginal secretions. Direct immunofluorescent antibody staining is more sensitive (70–90%) than

wet-mount examinations. T. vaginalis can be recovered from the ure thra of both males and females and is detectable in males after prostatic massage. NAATs are highly sensitive and specific for urine and for endocervical and vaginal swabs from women and often considered first-line diagnostics. TREATMENT Trichomoniasis Metronidazole (either a single 2-g dose or 500-mg doses twice daily for 7 days), tinidazole (a single 2-g dose) or secnidazole (a single 2-g dose) is effective. All sexual partners must be treated concur rently to prevent reinfection, especially from asymptomatic males. In males with persistent symptomatic urethritis after therapy for nongonococcal urethritis, metronidazole therapy should be consid ered for possible trichomoniasis. Alternatives to metronidazole for treatment during pregnancy are not readily available. Reinfection often accounts for apparent treatment failures, but strains of T. vagi nalis exhibiting high-level resistance to metronidazole have been encountered. Treatment of these resistant infections with higher oral doses, parenteral doses, or concurrent oral and vaginal doses of metronidazole or with tinidazole has been successful.

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