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appear to be particularly susceptible to PCP. The glucocorticoid exposure threshold that warrants chemoprophylaxis is ill-defined, but such preventive therapy should be strongly considered for any patient who is receiving more than the equivalent of 20 mg of pred nisone daily for 30 days or who is receiving glucocorticoids in con junction with other immunosuppressive agents. Clinical experience also suggests that chemoprophylaxis is useful for patients receiving certain immunosuppressive agents (e.g., tumor necrosis factor inhibi tors, antithymocyte globulin, rituximab, and alemtuzumab). The duration of such chemoprophylaxis is empirically estimated based on prior clinical experience and immunologic factors that would plausi bly relate to immunity, such as CD4+ T-cell counts, recognizing that such estimates are not precise. TMP-SMX is the most effective prophylactic drug; few patients experience a PCP breakthrough when they are reliably taking a recom mended TMP-SMX chemoprophylactic regimen. Several TMP-SMX regimens have been used successfully. Regimens of one single-strength or double-strength tablet daily are the regimens with which there is the most experience, but one double-strength tablet two or three times weekly also has been recommended for various PLWH and non-HIVinfected populations of patients. For patients who cannot tolerate TMP-SMX (usually because of hypersensitivity or bone marrow suppression), alternative drugs include daily dapsone, weekly dapsone-pyrimethamine, atovaquone, and monthly aerosol pentamidine. Patients who develop hypersensi tivity to TMP-SMX can sometimes tolerate the drug if a gradual doseescalation protocol is used. Atovaquone is effective and well tolerated; however, this drug is available only as an oral preparation, and gastro intestinal absorption is unpredictable in patients with abnormal gas trointestinal motility or function. Aerosolized pentamidine is effective, but it is not as effective as TMP-SMX and may not provide protection in areas of the lung that are not well-ventilated. Dapsone cross-reacts with sulfonamides in a substantial fraction of patients and is rarely use ful in patients with a history of life-threatening reactions to TMP-SMX. ■ ■FURTHER READING Buchacz K et al: Incidence of AIDS-defining opportunistic infections in a multicohort analysis of HIV-infected persons in the United States and Canada, 2000–2010. J Infect Dis 214:862, 2016. Del Corpo O et al: Diagnostic accuracy of serum (1-3)-β-D-glucan for Pneumocystis jirovecii pneumonia: A systematic review and metaanalysis. Clin Microbiol Infect 26:1137, 2020. Lécuyer R et al: Characteristics and prognosis factors of Pneumocystis jirovecii pneumonia according to underlying disease. Chest 165:1319, 2024. Le Gal S et al: Pneumocystis infection outbreaks in organ trans plantation units in France: A nation-wide survey. Clin Infect Dis 70:2216, 2020. Ma L et al: Genome analysis of three Pneumocystis species reveals adaptation mechanisms to life exclusively in mammalian hosts. Nat

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Section 17 Protozoal and Helminthic Infections: General Considerations Sharon L. Reed, Charles E. Davis

Introduction to

Parasitic Infections The word parasite comes originally from the Greek parasitos (para, alongside of; and sitos, food), meaning someone who eats at another's table or lives at another's expense. Although the same is true of many bacteria and viruses, the designation parasite is reserved, by conven tion, for helminths and protozoa. These organisms are larger and more complex than bacteria, with a eukaryotic cell structure similar to that of human host cells. Historically, this similarity has made it difficult to find effective antiparasitic agents that do not cause unacceptable toxicity to human cells. Fortunately, intensive research and modern techniques have now provided suitable agents for safe and effective treatment of most parasitic infections. See Chap. S12 for details on diagnostic procedures and Chap. 229 for details on treatment. Internal parasites of human beings are divided into two types: hel minths (worms) and protozoa. Helminths are multicellular organisms that can often be seen with the naked eye (Chap. 237). There are two phyla: Platyhelminthes (flat worms) and Nemathelminthes (round worms). Both phyla include some genera that mature in the gastroin testinal tract and others that migrate through the tissue after ingestion or skin penetration. Tables S12-1 and S12-2 present the helminthic genera, their definitive and intermediate hosts, their geographic distri butions, and the parasitic stages in the human body. CHAPTER 228 Introduction to Parasitic Infections The key to understanding which helminths use humans as defini tive hosts is to remember that helminth ova develop into larvae, and larval stages develop into adults. Humans serve as the definitive host when they ingest helminth larvae, which develop into adults in the intestine and usually cause mild disease, often without any symptoms. (The exception is ingestion of the late-stage larvae of the somatic or tissue flukes, as shown in Table S12-1.) In contrast, if humans ingest helminth ova and serve as the intermediate host, the ova develop into larvae, which penetrate the intestine, migrate through the tissue, and invade organs where they mature into adults. Intermediate hosts with parasitic invasion of organs may experience severe disease. Protozoa are microscopic single-celled organisms. Among the many differences between helminths and protozoans, the most important is the ability of protozoa (like bacteria) to multiply within the human body and cause overwhelming infections. A major mechanism pro moting unrestrained growth is evasion of the host immune response either by antigenic variation (Trypanosoma brucei) or by survival inside host cells (e.g., Plasmodium, Babesia, Cryptosporidium, Leishmania, and Toxoplasma). In contrast, almost all helminths require stages in other hosts to complete their life cycles and multiply. As a result, except for Strongyloides

and Capillaria, which can complete their life cycle in humans, increases in the burden of infection with helminths require repeated exogenous reinfections. Thus, permanent residents of endemic countries, who are exposed repeatedly, may have heavy severe infections, while most travelers with one or two exposures are unlikely to experience the full spectrum of chronic helminthic infections. In contrast to helminthic infections, naïve patients with their first protozoal infection usually are the most severely affected because partial immunity often limits the number of parasites during recurrent infections. Protozoan replication to large numbers in the host also pro motes the development of drug-resistant forms, especially in malaria (Chap. 229). Because protozoa belong to many different phyla, it is easier to understand the pathogenesis and management of protozoal infections when they are classified by the site of infection (intestinal protozoans, free-living amebae, and blood and tissue protozoans)

(Table S12-3). Immunocompromised hosts are at risk of disseminated infection with several protozoa, including Leishmania, Toxoplasma, Cryptosporidium, and Trypanosoma cruzi, which are AIDS-defining illnesses. Among the helminths, Strongyloides can disseminate in immunocompromised individuals.

HELMINTHIC INFECTIONS The Platyhelminthes (flatworms) are categorized as tapeworms (ces todes) and flukes (trematodes). Tapeworms are composed of a head or scolex bearing the holdfast organs and segments, which become gravid as they mature. Some tapeworms can reach lengths of many yards; the longest tapeworms develop in the intestine, where they rarely cause serious disease. In contrast, flukes are small leaf-shaped organisms whose size is not a measure of disease severity. ■ ■FLATWORMS Cestodes Tapeworms cause either intestinal or somatic infection, depending on the species. Intestinal infections occur when the human host ingests larvae in the tissue of the intermediate host, whereas somatic infections occur when humans accidentally ingest ova excreted from the wild or domesticated definitive animal host. INTESTINAL TAPEWORMS As shown in Table S12-1, humans acquire most intestinal tapeworms by eating the insufficiently cooked flesh of the intermediate host. Thus, Taenia saginata is commonly called the beef tapeworm, Taenia solium the pork tapeworm, and Diphylloboth rium latum the fish tapeworm. Hymenolepis nana is capable of com pleting its life cycle in the human intestine and is acquired by ingestion of infected grain beetles or of ova from infected humans or mice. None of these parasites causes significant damage, and infection is usually asymptomatic. There are two occasional exceptions. When people ingest T. solium ova from their own intestine or from another infected individual, it can cause somatic infection. D. latum avidly absorbs vitamin B12 in the intestine and can cause pernicious anemia in 1–2% of infected Scandinavians with a genetic predisposition. PART 5 Infectious Diseases SOMATIC TAPEWORMS There are three major causes of somatic tape worm infections. Two species of Echinococcus cause echinococcosis.

E. granulosus is acquired by accidental ingestion of ova from dogs infected when fed the infected tissues of sheep or other animals by sheepherders or hunters. E. multilocularis is transmitted primarily in sub-Arctic areas when humans ingest ova from foxes, dogs, or cats that have been infected through consumption of the tissues of infected rodents. Both species cause hydatid cysts when the eggs hatch into lar vae, penetrate the intestine, and migrate into the liver or lung. Ingested T. solium ova cause somatic disease (cysticercosis) when the larvae penetrate the intestine, migrate into tissue, and form cysts (cysterci), usually in the muscles or central nervous system (CNS). Trematodes Flukes also cause both intestinal and somatic infections (Chap. 241 and

Table S12-1). Most fluke infections are localized to Asia, Africa, Southeast Asia, or the Pacific islands. Infection with intestinal flukes is usually asymptomatic, although heavy infections sometimes cause abdominal discomfort and mucous diarrhea. Liver flukes and lung flukes cause somatic infections when humans ingest a larval form from an intermediate host. Adults develop in the intestine, migrate into adjacent tissues, and cause disease. The major liver flukes (Clonorchis sinensis, Opisthorchis spp., and Fasciola hepatica) are causes of recurrent bacterial cholangitis (due to obstruction) or portal hypertension and cir rhosis. Only F. hepatica can be acquired worldwide; it is especially com mon in sheep-raising areas, where the animals ingest water plants (e.g., watercress). The lung flukes (Paragonimus spp.) occur globally except in Europe; most lesions occur as pulmonary cysts, although occasional lesions develop in the CNS or the abdominal cavity. The blood flukes cause schistosomiasis, one of the most common and serious parasitic infections (Chap. 241 and Table S12-1). The major species are Schistosoma mansoni, S. haematobium, and S. japonicum. All are transmitted to humans when free-swimming larvae exit an infected snail in freshwater and penetrate the skin. Swimmer’s itch sometimes follows skin penetration but is usually of short duration.

The larvae then wander in the skin until they find a blood vessel and migrate to the target organ. S. mansoni and S. japonicum migrate to the mesentery vessels and eventually make their way to the liver, while S. haematobium targets the veins around the ureter and blad der. Extensive egg deposition by S. mansoni and S. japonicum and the immune reactions to the ova cause granuloma formation and, with many repeated exposures, portal vein obstruction and cirrhosis. The same process in the ureters and bladders during infection with S. hae matobium eventually interferes with urine flow and leads to repeated urinary tract infections and kidney damage. ■ ■ROUNDWORMS Nematodes Roundworms are nonsegmented bisexual organisms. The species that infect humans include intestinal and tissue groups. Humans may also acquire certain nonhuman mammalian round worms that either can be limited to the skin or can migrate to tissues and cause serious disease (the larva migrans syndromes). INTESTINAL ROUNDWORMS The major intestinal roundworms are Ascaris lumbricoides, Necator americanus (New World hookworms), Ancylostoma duodenale (Old World hookworms), Trichuris trichiura (whipworms), Enterobius vermicularis (pinworms), and Strongyloides stercoralis. Taken together, infections caused by intestinal roundworms are the most common infections in the world. Ascaris, hookworms, and Trichuris infect about 900 million individuals, and at least 100–370 million have strongyloidiasis. These infections are most common in resource-poor developing countries, especially where people defecate outside and/or human feces is used as fertilizer (“night soil”). Infection is transmitted either by ingestion of ova (A. lumbricoides, T. trichiura, and E. vermicularis) or by active penetration of the skin by larvae (hookworms and S. stercoralis) (Table S12-2). Intestinal roundworms cause serious health problems in residents of endemic regions with poor sanitation, but travelers are at low risk of developing significant disease from most of these parasites. Intestinal blockage and malnutrition from heavy Ascaris infections and anemia from heavy hookworm infections are now restricted to areas of heavy endemicity. Except in the case of Strongyloides and Capillaria, which can reproduce in the body, multiple exposures over time are neces sary for the development of severe disease. Strongyloides infection persists over decades and can disseminate when the immune system is compromised. Although Capillaria remains localized to the intestine, infections can become so heavy that protein-losing enteropathy and malnutrition cause serious disease. The life cycles of Ascaris and the hookworms involve migration through the heart and lungs before development into adults in the intestine. In particular, Ascaris

occasionally causes eosinophilic pneu monia (Loeffler’s syndrome) during heavy infections. Pinworms are the most common causes of intestinal roundworm infection persisting in the United States and other developed countries. The anal and peri neal itching caused by pinworm migration out of the anus and subse quent egg deposition is well known to families throughout the world. TISSUE ROUNDWORMS The major diseases caused by tissue round worms are filariasis, angiostrongyliasis, gnathostomiasis, and trichinel losis. By far, the most important globally is filariasis; the thread-like filarial worms infect an estimated 80 million individuals in tropical and subtropical areas of the world. Four filarial species cause three distinct diseases: lymphatic filariasis (Wuchereria bancrofti and Brugia malayi), river blindness (Onchocercus volvulus), and loiasis (Loa loa, the African eye worm). Humans, the major reservoir, acquire these infections from bites of infected arthropods (Table S12-2). The larvae develop into adults, which remain static in tissue: the lymphatics for lymphatic filariasis and subcutaneous tissue for O. volvulus and L. loa. After adults mate, next-stage larvae are produced, and their migration causes additional damage. Repeated bouts of migrating larvae and blocking of the lymphatics by adults are necessary to establish the syndrome of lymphatic filaria sis; thus, it is unusual for the short-term traveler (<3 months’ residence in an endemic region) to develop significant disease. In river blindness, the larvae produced by adult O. volvulus migrate through the skin and

eye, causing skin damage and eventual blindness. Loiasis is a milder disease restricted to central and western Africa. Although both the adults and the larvae of L. loa migrate through the skin and eye, many infected individuals are asymptomatic, and the infection is often diag nosed only when an adult worm migrates across the subconjunctival tissue and is visible to the patient and the physician. Red lumps in the skin from heavy cutaneous migration are called Calabar swellings. The other four major roundworm tissue infections are acquired by ingestion of larvae in undercooked food. The sources for trichinellosis are swine and other large mammals; for gnathostomiasis, freshwater fish and chicken; for ancylostomiasis, snails, fish, prawns, and crabs; and for Guinea worm, infected water fleas. Guinea worm infection (dracunculiasis, caused by Dracunculus medinensis) has been almost eradicated. Trichinella spiralis larvae penetrate the intestine and migrate widely, with a preference for skeletal tissue; the release of eosinophils and IgE causes muscle soreness and may cause palpebral swelling and other manifestations of generalized allergic reactions. Angiostrongylus cantonensis is the most common parasitic cause of eosinophilic men ingitis. Ingested larvae penetrate the intestine and migrate to the brain and meninges, where they quickly die and attract massive numbers of eosinophils. Although complications can occur, most individuals recover spontaneously. Gnathostoma spinigerum larvae also penetrate the intestine and migrate, showing a preference for the skin, eyes, and meninges. Mechanical damage from the migration and inflammation produced by the resultant immune reaction can cause boil-like lesions on the skin, painful eye damage, and eosinophilic meningitis. Although eosinophilic meningitis caused by G. spinigerum is less common than that caused by A. cantonensis, it is often more severe and can result in paralysis or brain hemorrhage. PROTOZOAL INFECTIONS ■ ■INTESTINAL PROTOZOA Entamoeba histolytica is the one intestinal protozoan that causes inva sive disease. This disease consists of dysentery or bloody diarrhea that must be differentiated from that due to bacteria such as Salmonella, Campylobacter, and Shigella. Although amebiasis usually has a slower onset with lower fever than these bacterial infections, E. histolytica can disseminate from the bloodstream to cause distant abscesses, particu larly of the liver. The diagnosis cannot be made by identification of the characteristic cyst or trophozoites (Chap. 230), as they are identical to those of the noninvasive E. dispar, which is more common globally. Cryptosporidium and Giardia are the

most common water-borne pro tozoal infections. Cryptosporidium can cause major outbreaks because it is highly infectious and resistant to high levels of chlorine (Chap. 236). Without immune reconstitution, immunosuppressed patients, particu larly those with AIDS, can develop severe, even fatal watery diarrhea. Infections caused by the remaining intestinal protozoans—Giardia, Isos pora, Cyclospora, and microsporidia (Chap. 236)—have a much more indolent course, with intermittent diarrhea. Microsporidia, unique intracellular protozoa that form infectious spores, may cause limited gastrointestinal infection in immunocompetent hosts, but patients with AIDS can develop chronic diarrhea and wasting or disseminated infec tion to the biliary or respiratory tract. ■ ■FREE-LIVING AMOEBAS The free-living amoebas Acanthamoeba and Naegleria are found world wide in freshwater and brackish water (Chap. 230 and Table S12-3). Organisms of these two genera cause very different syndromes. In immunocompromised individuals, Acanthamoeba may cause invasive infection, with brain masses and skin lesions. However, all humans are susceptible to Acanthamoeba keratitis after trauma to the eye and expo sure to contaminated water. In contrast, naeglerial meningitis, acquired in warm lakes or hot springs, causes sudden pyogenic and usually fatal meningitis. Balamuthia, reported only from the Americas, causes indo lent meningoencephalitis, with both cerebrospinal fluid pleocytosis and a space-occupying lesion, in immunocompetent patients. Despite the availability of miltefosine, which is active in vitro against Naegleria, infection of the CNS is almost universally fatal.

■ ■BLOOD AND TISSUE PROTOZOANS

Plasmodium and Babesia Malaria, caused by six species of Plasmo dium, carries higher mortality rates than any other parasitic infection (Chap. 231). All species are transmitted in tropical and subtropical areas by female Anopheles mosquitoes. Plasmodium falciparum is most common in sub-Saharan Africa, where it causes more than 80% of malaria infections and 90% of malarial deaths. Infection with

P. falciparum may be particularly severe because the organism can invade any erythrocyte, reaches very high parasite loads, damages organs by adhering to vascular epithelium, and is the most likely Plasmodium species to be resistant to antimalarial drugs. Plasmodium vivax, the dominant cause of malaria outside sub-Saharan Africa, reaches lower levels of parasitemia and exhibits less drug resistance because it invades only reticulocytes with Duffy antigen. Many Africans, especially in the western part of the continent, lack the Duffy blood group; consequently, Plasmodium ovale, another cause of milder malaria, can compete successfully with P. vivax. Both P. vivax and P. ovale pro duce persistent liver forms, which must be treated with primaquine (Chap. 229). Because malaria can cause a variety of symptoms ranging from acute fever to coma, this diagnosis must be considered in any traveler or immigrant from a malarial area. Recently, locally acquired P. vivax malaria has been detected in Florida and Texas and P. falci parum in Maryland; thus, malaria should be included in the differential diagnoses of unexplained fever even in patients who have never left the United States. Babesia also infects erythrocytes and may cause a nonspecific febrile illness or, in asplenic patients, severe infection. This parasite is carried by ixodid ticks and is geographically limited to the northeastern and midwestern United States, with only sporadic cases in Europe and other temperate areas. CHAPTER 228 Trypanosomes The three species of trypanosomes all have flagel lated bloodstream forms, but they cause very different diseases. T. cruzi, the cause of Chagas disease, is transmitted in South and Central America in the feces of blood-sucking reduviid bugs (Chap. 234). After initial parasitemia, patients are often

asymptomatic for years while the parasite multiplies intracellularly in muscle and ganglion cells. Although only a minority of patients go on to develop organ damage (megaesophagus and cardiomyopathy), all infected patients can spread the disease through transfusions, mother-to-child transmission, and organ transplants. Introduction to Parasitic Infections African trypanosomiasis is limited to sub-Saharan Africa, where it is transmitted by the bite of a tsetse fly. A history of a tsetse bite and the presence of a painful chancre are strong diagnostic clues (Chap. 234). Although the parasites causing this disease in western Africa (Trypano soma brucei gambiense) and eastern Africa (T. brucei rhodesiense) look identical, they are genetically and clinically distinct. T. b. gambiense causes low-level parasitemia with cyclical fevers over months or years before CNS invasion, whereas T. b. rhodesiense causes high-level para sitemia, invades the CNS early on, and can lead to death within weeks of onset. Leishmania Leishmaniasis is caused by more than 20 species of obligate intracellular protozoa transmitted by sandflies, which are present in almost 100 countries in tropical and temperate zones (Chap. 233). A wide spectrum of clinical symptoms result, ranging from self-healing, painless skin ulcers to mucocutaneous disease with destruction of the nose and palate to disseminated visceral leishmaniasis with hepatic and splenic involvement. The resulting disease depends on the infect ing strain and the host immune response. Visceral leishmaniasis can present as an acute febrile illness, with the later development of hepatosplenomegaly, and is an AIDS-defining illness in HIV-infected patients. More than 90% of cases of visceral leishmaniasis occur in India, Bangladesh, Ethiopia, Sudan, and Brazil. Toxoplasma Toxoplasma gondii is an obligate intracellular parasite that is found worldwide. Infection follows ingestion of oocysts in food or water contaminated by cat feces, ingestion of tissue cysts in under cooked meat, or transplacental transmission. After gastrointestinal invasion, tachyzoites can invade any nucleated cell and cause lifelong infection in most patients (Chap. 235). Clinical manifestations depend

on the host’s age and immune status at the time of infection. Congeni tal toxoplasmosis results from primary maternal infection; outcomes are most severe early in pregnancy and include visual, hearing, and cognitive impairments. Babies infected later in pregnancy may appear normal but can develop chorioretinitis decades later. Primary infection in immunocompetent hosts may be asymptomatic, may present as an infectious mononucleosis–like syndrome, or may manifest as chorio retinitis during outbreaks. During immunosuppression by AIDS or organ transplantation, reactivation of latent cerebral infection can be fatal unless diagnosed and treated early.

APPROACH TO THE PATIENT Parasitic Infection A thorough history and physical examination are the keys to diag nosis of any disease and particularly of parasitic infections. Because many of the more serious parasitic infections are uncommon in the United States, a travel history, particularly to developing nations, is a critical component. The longer the stay in an area endemic for significant parasitic infections, the greater the risk, even for healthy travelers. In addition, other factors increase the chance of acquiring these infections. Notably, immunocompromise greatly increases the likelihood of developing some of the more serious parasitic infec tions. Even healthy travelers with adventure itineraries, extensive travel to rural areas, or involvement in war zones or refugee camps are at increased risk. Immigrants from developing countries may seek care for symptoms or signs associated with parasitic infections. Information on the patient’s immunization history and adher ence to appropriate malarial chemoprophylaxis is critical. The recent approval of the first parasitic vaccines against P. falciparum is very exciting, but it will be targeted initially only for children in high prevalence areas. Typhoid fever is much less likely to be the cause of prolonged

fever in an immunized individual. Similarly, PART 5 Infectious Diseases TABLE 228-1 Parasitic Infections, by Organ System and Signs/Symptomsa ORGAN SYSTEM, MAJOR SIGN(S)/SYMPTOM(S) PARASITE(S) GEOGRAPHIC DISTRIBUTION COMMENTS Skin Serpentine rash Hookworm Worldwide Can cause anemia in heavy infections Strongyloides Moist tropics and subtropics Disseminated infection in immunocompromise Toxocara (animal roundworm) Tropical and temperate zones Cutaneous or visceral larva migrans Itchy skin rash Onchocerca Mexico, Central/South America, Africa Painless ulcers Leishmania Tropics and subtropics Amastigotes detectable in biopsies; may cause destructive mucocutaneous infection; AIDS-defining infection Skin nodules Onchocerca Mexico, South America, Africa Large nodules of adult worms Loa loa (African eye worm) Western and central Africa Migratory nodules Gnathostoma Southeast Asia and China Migratory nodules with eosinophilia Painful nodules, especially involving feet Dracunculus (Guinea worm) Africa Nearly eradicated Central Nervous System Somnolence, seizures, coma Plasmodium falciparum Subtropics and tropics Cerebral malaria, especially in children Trypanosoma brucei rhodesiense Sub-Saharan eastern Africa Painful chancre from tsetse fly bite; death in weeks to months Space-occupying lesions, seizures Acanthamoeba Worldwide Immunocompromised individuals Balamuthia Americas Indolent meningoencephalitis with brain mass Toxoplasma Worldwide Reactivation disease in immunocompromise; ring-enhancing lesions; AIDS-defining infection Taenia solium Mexico, Central/South America, Africa Schistosoma japonicum Far East Aberrant eggs can form brain or spinal cord masses. Schistosoma mansoni Africa, Central/South America Aberrant eggs can form brain or spinal cord masses.

hepatitis A or B is unlikely to be the cause of jaundice and fever in fully immunized patients. In this era of increasing drug resistance, even adherence to appropriate malarial chemoprophylaxis does not guarantee that fever is not malarial. Nevertheless, most travel ers who acquire malaria have taken inadequate or no prophylaxis. Although these considerations do not prove that the symptoms are caused by parasites, they narrow the differential diagnosis. There are many other important aspects of the history, includ ing when symptoms began. Was the individual still in the endemic area at the time, or did the symptoms commence after return to the United States? If they started during travel, was any treatment received? Malaria must be the first consideration in a febrile patient returning from an endemic area. If the patient was well upon return from travel, the timing of symptom onset is a critical point. For example, if the chief manifestation is fever that began >10–14 days after departure from the endemic region, many tropical diseases can be ruled out, including dengue fever, chikungunya fever, and Zika virus infection. On the other hand, fever beginning several months or later after return makes malaria a likely diagnosis. Trav elers' diarrhea, the most common complaint of travelers, is usually caused by bacteria or viruses and resolves in a short time with or without treatment. Travelers' diarrhea that persists for weeks is much more likely to be parasitic in origin. Most patients who consult physicians after international travel either have troublesome symptoms or have been referred for symp toms or signs whose source was unclear to a referring caregiver. After a careful travel history including the individual's symptoms and the exact geographic zones visited, a thorough physical exami nation must be conducted. The symptoms, signs, and physical findings should help to establish possible diagnoses. Table 228-1 breaks down the symptoms of major parasitic infections by organ system and geographic distribution, with comments on clinical and epidemiologic associations. Larvae detectable in skin snips and nodules Cysticercosis; variable sized or calcified larval cysts on CT (Continued)

TABLE 228-1 Parasitic Infections, by Organ System and Signs/Symptomsa ORGAN SYSTEM, MAJOR SIGN(S)/SYMPTOM(S) PARASITE(S) GEOGRAPHIC DISTRIBUTION COMMENTS Pyogenic meningitis Naegleria Worldwide Motile trophozoites in fresh cerebrospinal fluid; pyogenic; rapid death Eosinophilic meningitis Angiostrongylus (rat lung worm) Southeast Asia, Pacific, Caribbean Gnathostoma Southeast Asia and China Migratory nodules Eyes Painful corneal ulcers Acanthamoeba Worldwide Freshwater and brackish water; corneal trauma; long-wear contact lenses Corneal opacification Onchocerca Mexico, Central/South America, Africa Congenital or adult visual loss Toxoplasma Worldwide Primary infection in pregnancy and normal hosts; reactivation infection in immunocompromised Retinal mass Toxocara Worldwide Ocular larva migrans Visible roundworm in eye Onchocerca Mexico, Central/South America, Africa L. loa Western and central Africa Worms may cross eye during migration. Pain, possible vision loss Gnathostoma Southeast Asia and China Migratory skin nodules, eosinophilia Lungs Pulmonary nodule/abscess Paragonimus Far East, Africa, Americas Ectopic migration to abdomen or central nervous system Cough, transient infiltrates, eosinophilia Migrating helminths Worldwide Loeffler’s syndrome from migrating Ascaris, hookworm, Strongyloides Heart Pulmonary edema P. falciparum (complication) Tropics and subtropics End-organ damage from severe malaria Cardiomegaly, arrhythmias Trypanosoma cruzi Mexico, Central/South America Late amastigote infection of myocardium; AIDS-defining infection Gastrointestinal Tract Hepatosplenomegaly Malaria (multiple episodes) Tropics and subtropics Splenomegaly with anemia and recurrent fever are hallmarks of malaria. S. mansoni Africa, Central/South America Portal obstruction with cirrhosis and late varices Leishmania donovani complex Tropics and subtropics Visceral leishmaniasis; AIDS-defining infection Hepatomegaly Entamoeba histolytica Tropics Acute with fever, right-upper-quadrant pain; or chronic with enlarged liver; hypoechoic abscess(es) on ultrasound or CT Echinococcus Sheep-raising areas Characteristic cysts of liver > lung Fasciola Sheep-raising areas Eosinophilia Cholangitis Clonorchis China, Southeast Asia Recurrent cholangitis and late cholangiocarcinoma Microsporidia Worldwide AIDS Cryptosporidium Worldwide AIDS-defining infection Bloody diarrhea E. histolytica Tropics Less fever than in diarrhea of bacterial etiology S. mansoni Africa, Central/South America Only in heavy, acute infection with fever and eosinophilia S. japonicum Far East Only in heavy, acute infection Watery diarrhea Cryptosporidium Worldwide Severe in immunocompromised patients Giardia Worldwide Foul-smelling stool with steatorrhea Isospora belli Worldwide Fever, abdominal pain, chronic diarrhea Microsporidia Worldwide Chronic diarrhea with AIDS Capillaria Southeast Asia, Egypt Malabsorption, wasting Passage of large roundworm (>6 cm) Ascaris Worldwide Patients may confuse the roundworm with an earthworm. Small roundworms visible around anus Pinworm Worldwide Anal itching; eggs rarely detected by ova and parasite (O&P) exam Trichuris Worldwide Rectal prolapse with heavy infection in children Passage of tapeworm segments T. solium or Taenia saginata Worldwide Usual reason for seeking medical care Diphyllobothrium latum Worldwide Pernicious anemia in genetically predisposed Scandinavians Genitourinary System Itchy discharge Trichomonas vaginalis Worldwide Common sexually transmitted disease of both sexes Hematuria Schistosoma haematobium Africa Hematuria with negative cultures, urinary tract infections, and late bladder cancer

(Continued) Most common cause globally of eosinophilic meningitis; spontaneous resolution Immune response to microfilaria in cornea Worms may cross eye during migration. CHAPTER 228 Introduction to Parasitic Infections (Continued)

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