IV. PARASITES

A. Protozoa

1. GI/GU
   a. *Cryptosporidium*
      1) Appearance: cysts are small, **stain pink in stool specimens**
      2) Lab assays: stool O&P
      3) Virulence factors: none significant
      4) Epidemiology: ubiquitous but **typically causes disease in AIDS patients**
      5) Clinical Diseases: watery diarrhea, causes severe malabsorption in AIDS patients
      6) Treatment: supportive and immune reconstitution—nitazoxanide may have a role
      7) Resistance: none
      8) Prophylaxis: boil or filter water, chlorination does not work
   b. *Entamoeba histolytica*
      1) Appearance: two phases of life-cycle: cyst has four nuclei, trophozoite has one nucleus and is not flagellated and often contains ingested red blood cells (see Figure 4.1)
      2) Lab assays: stool O&P should reveal cyst or trophozoite, anti-amoeba antibody titers are diagnostically useful
      3) Virulence factors: none significant
      4) Epidemiology: fecal-oral transmission
      5) Clinical Diseases: amoebic dysentery and amoebic liver abscess
      6) Treatment: metronidazole followed by iodoquinol or paromomycin—the latter are necessary to kill encysted organisms in the bowel lumen that are not killed by metronidazole
      7) Resistance: none
      8) Prophylaxis: boil or filter water, chlorination has no effect, careful hand-washing and separation of human wastes from crop fields (don't fertilize crops with human feces)
   c. *Giardia lamblia*
      1) Appearance: two phases of life cycle: cyst has four nuclei and has a thicker wall than *Entamoeba*, **trophozoite is oval with two nuclei and has four pairs of flagella** (see Figure 4.2)
      2) Lab assays: stool O&P, string test = patient swallows a string down into the duodenum while the physician holds onto the far end and then pulls the string back up out of the mouth, revealing the trophozoites stuck onto the string
      3) Virulence factors: none significant
      4) Epidemiology: fecal-oral transmission, often via streams in the wilderness as many animals carry *Giardia* as well, **classic**

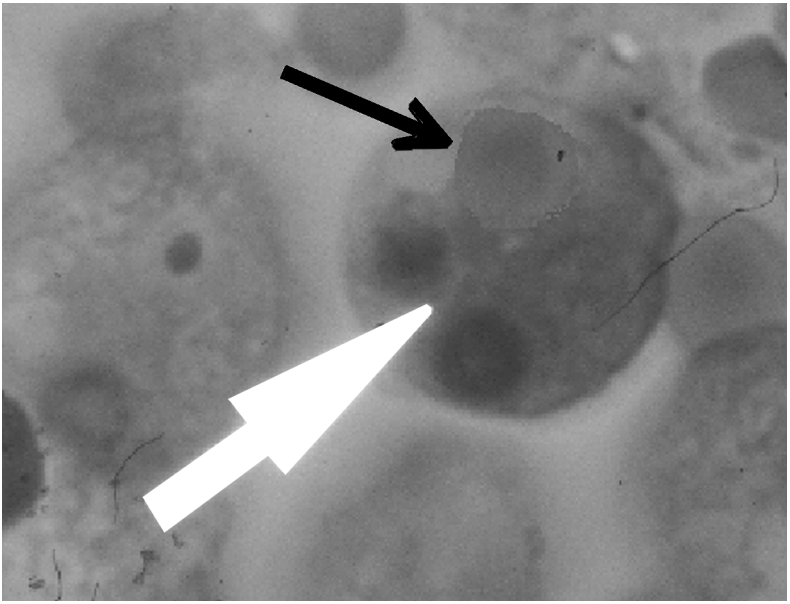

FIGURE 4.1 **Classic *Entamoeba* Cyst**

Classic Entamoeba *cyst (light arrow) with an ingested red blood cell (dark arrow).*

Boards scenario is a hiker who drinks from a stream and gets diarrhea, disease is common in patients with IgA deficiency

5) Clinical Diseases: chronic diarrhea, no tissue invasive disease
6) Treatment: metronidazole
7) Resistance: none
8) Prophylaxis: boiling or filtering water, chlorination doesn't work

d. *Trichomonas*

1) Appearance: there is no cyst stage, **pear-shaped trophozoite, with four flagella, highly motile on wet mount**
2) Lab assays: wet mount
3) Virulence factors: none significant
4) Epidemiology: sexually transmitted, resides in the vagina or, rarely in male GU tract, can be spread by asymptomatic carriers
5) Clinical Diseases: vaginitis with **green, frothy discharge**, can cause urethritis in men, **watch out for Boards questions where both the male and female sex partners have GU symptoms, or questions in which the female is diagnosed with *Trichomonas* and treated appropriately but comes back**

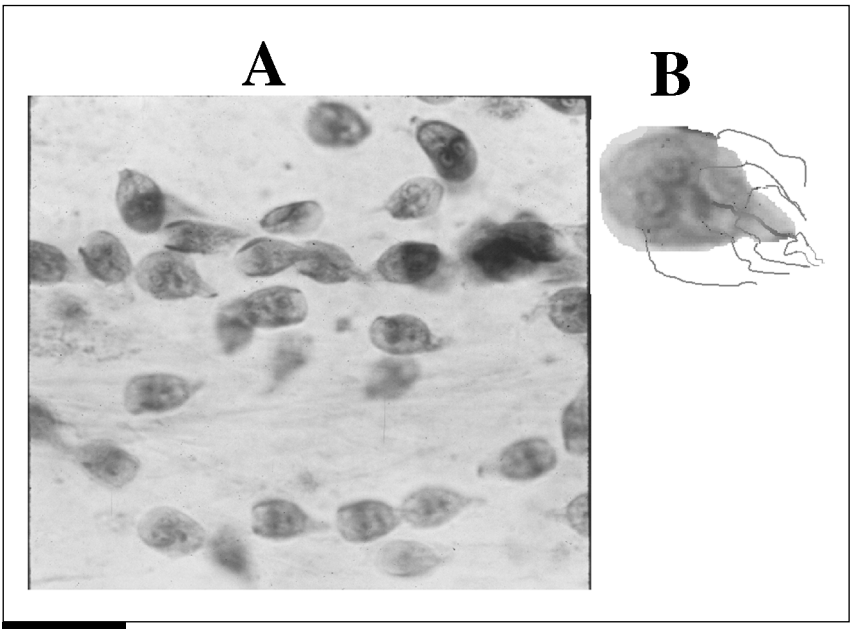

FIGURE 4.2 ***Giardia*** **Trophozoites**

A) Typical appearance of Giardia *trophozoites from an intestinal biopsy. B) Close-up of an individual trophozoite, revealing the presence of two nuclei and four pairs of flagella.*

shortly with a new infection—both sex partners must be treated simultaneously to eradicate the disease

6) Treatment: metronidazole, treat both sex partners
7) Resistance: none
8) Prophylaxis: condoms

2. Invasive protozoa
   a. *Babesia*
      1) Appearance: intracellular ring forms within red cells similar to malaria
      2) Lab assays: blood smear, serology
      3) Virulence factors: none significant
      4) Epidemiology: transmitted via tick bite, **typically occurs in the New England area, specifically along the coast**
      5) Clinical Diseases: high fevers, shaking chills, myalgias, abdominal pain, nausea/vomiting, followed by **severe hemolytic anemia particularly dangerous in splenectomized patients**
      6) Treatment: clindamycin plus quinine
      7) Resistance: none
      8) Prophylaxis: avoid tick bites

TABLE 4.1 Summary of GI/GU Protozoa

ORGANISM	APPEARANCE	DISEASE	TREATMENT
Cryptosporidium	Cyst stain pink in stool, nuclei not visible	Watery diarrhea, typically in AIDS	Immune reconstitution
Entamoeba	Thin-walled cyst with 4 nuclei, **trophozoite ingests red blood cells and is not flagellated**	Bloody diarrhea, invasive hepatic abscess	Metronidazole and iodoquinol
Giardia	Thick-walled cyst with 4 nuclei, **trophozoite is oval and has 4 pairs of flagella**	Non-bloody diarrhea, often in hikers	Metronidazole
Trichomonas	No cyst, **trophozoite is oval with 4 single flagella**	Vaginitis	Metronidazole (treat both sex partners)

b. *Leishmania spp.*
   1) Appearance: amastigotes are small intracellular organisms seen within macrophages
   2) Lab assays: blood smear or tissue biopsy reveal organisms within macrophages, serologies often positive
   3) Virulence factors: none significant
   4) Epidemiology: transmitted via sandfly bite, visceral disease seen in Asia and Africa, cutaneous disease seen in Central and South America as well as Asia and Africa, mucocutaneous disease seen only in Central and South America
   5) Clinical Diseases:
      a) **Kala azar = visceral leishmaniasis: caused by *L. donovani***, organism is concentrated in the reticuloendothelial system (liver, spleen, lymph nodes, bone marrow), causing **massive hepatosplenomegaly, pancytopenia**, hemorrhage, and susceptibility to secondary infections, patients also get hyperpigmented skin
      b) **Cutaneous leishmaniasis: caused by *L. tropica*** (Old World disease in Asia and Africa) and ***L. mexicana*** (New World disease in Central/South America), disease starts with **erythematous papule at site of sandfly bite**, can either heal spontaneously or progress to large, granulomatous ulcerations which often is secondarily infected by bacteria
      c) **Mucocutaneous leishmaniasis: caused by *L. braziliensis*, also starts as papule at site of sandfly bite, but can**

disseminate to multiple mucosal spots, creating granulomatous erosions of nose and mouth

6) Treatment: sodium stibogluconate, ketoconazole, liposomal amphotericin B, miltefosine
7) Resistance: unusual
8) Prophylaxis: long-sleeve shirts, long pants, insect repellents, pesticides to kill sandflies

c. *Plasmodium* (malaria): *falciparum*, *vivax*, *ovale*, *malariae*

1) Appearance: schizonts appear like "**signet rings**" in red blood cells, while the gametocyte of *P. falciparum* appears like a large crescent attached to a thin ring (see Figure 4.3)
2) Lab assays: thin and thick smear of whole blood to detect trophozoites
3) Virulence factors: none significant
4) Epidemiology: **transmitted by bite of *Anopheles* mosquito, infects several hundred million people worldwide**, particularly affects Africa and all Mediterranean countries, **causes about 1 million deaths per year**, life cycle is complicated:

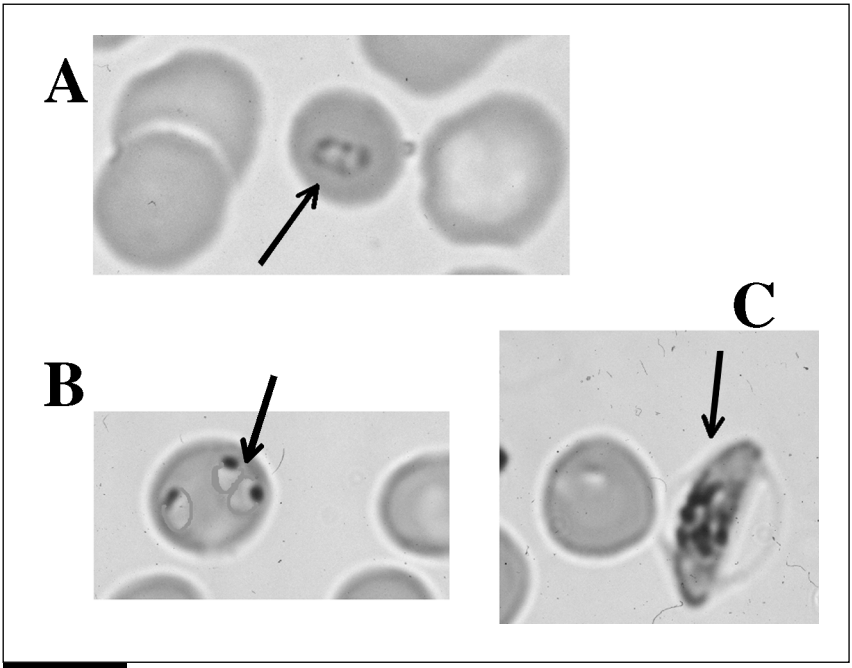

FIGURE 4.3 **(A and B) "Signet Ring" Schizonts of Malaria, (C) *P. falciparum***

A) and B) show typical "signet ring" schizonts of malaria in red blood cells. C) shows the classic "crescent" shaped gametocyte of P. falciparum *found in peripheral blood.*

sporozoites inoculated into blood from mosquito bite, seed the liver and transform into merozoites, leave the liver and infect red blood cells, transform into trophozoite which multiplies and then transforms back into merozoites and bursts the red cell, infects new red cells and then the cycle repeats—**note that *P. ovale* and *P. vivax* can lie dormant in the liver, allowing long-term relapse**

5) Clinical Diseases:
   a) General characteristics: all four species cause a cluster of symptoms including **paroxysmal fevers and shaking rigors, myalgias, diaphoresis, severe headache, arthralgias, and signs such as splenomegaly and hepatomegaly, red cell lysis causes anemia, initially the fever is continuous, but after several days the red cell bursts become synchronized in *P. vivax, ovale,* and *malariae* infections, note that *falciparum* may never become synchronized**
   b) **Tertian fever**: synchronous red cell bursts in ***P. vivax* and *ovale*** cause tertian fevers (q48 hrs = tertian because it occurs on the 3rd day, it is not q3 days), **relapse is common in tertian fever because *vivax* and *ovale* have a latent phase in the liver**
   c) **Quartan fever**: synchronous red cell bursts in ***P. malariae*** causes quartan fevers (**q72 hrs** because it occurs on the 4th day, it is not q4 days)
   d) ***P. falciparum* causes the most severe malaria, can cause continuous or irregular fevers without patterns, the organism burden in *P. falciparum* infections is much higher, and the red cells become sticky and can clog capillaries and cause DIC, leading to strokes and renal failure, CNS disease in a malaria patient is almost always due to *P. falciparum*, and carries a very high mortality**
6) Treatment:
   a) Tertian fever: **if *P. vivax* or *ovale* proven or suspected, treat with chloroquine (for merozoites in blood) plus primaquine (for latent organisms in liver)**—beware pts with G6PDH deficiency, in whom quinine-derivatives cause dangerous hemolytic anemia
   b) *P. falciparum* and Quartan fever: if *P. falciparum* or *malariae* proven or suspected, treat with chloroquine alone (primaquine not needed since there is no latent liver phase)—beware pts with G6PDH deficiency
7) Resistance: an increasingly severe problem, resistance endemic in South America, Africa, SE Asia, and parts of Middle East with few proven alternatives to quinine-derivatives, quinine plus doxycycline may cover some resistant organisms, other regimens are so unusual they are not likely testable on the USMLE

TABLE 4.2 Summary of *Plasmodium spp.*

ORGANISM	FEVER TIMING	CNS Dz	LATENT?	TREATMENT*
Falciparum	Irregular or q48 hr	Yes	No	CQ
Vivaxlovale	q48 hr	No	Yes	CQ + PQ
Malariae	q72 hr	No	No	CQ

*Assumes not resistant.
CQ, chloroquine; PQ, primaquine.

8) Prophylaxis:
   a) Mosquito netting and DEET insect repellent are keys to avoid inoculation—it is much more effective to prevent bites than to treat infection due to increasing drug resistance
   b) Chemoprophylaxis depends on if travel is to area with resistant organism
      i) Non-resistant area (Central America north of the Panama Canal, some parts of the Middle East and some parts of the Caribbean): chloroquine for several weeks before the trip, during the trip, and for several weeks after the trip
      ii) Resistant area: mefloquine, atovaquone-proguanil, or doxycycline before, during the trip, and after the trip

d. *Toxoplasma gondii*
   1) Appearance: biopsy of infected tissue reveals crescent-shaped organisms
   2) Lab assays: IgM serologies to detect acute infection
   3) Virulence factors: none significant
   4) Epidemiology: **transmitted via the feces of kittens (older cats less likely)**, or via ingestion of poorly cooked meat containing cysts, can also be transmitted vertically if the mother is newly infected during pregnancy
   5) Clinical Diseases:
      a) Immunocompetent people either get asymptomatically exposed or develop **mild heterophile negative mononucleosis**
      b) AIDS patients: **clinical disease primarily seen in HIV patients**, immunosuppression allows reactivation of the organism, classically causing severe **encephalitis with multiple ring-enhancing lesions in the brain**
      c) Congenital: causes multi-organ disease and can lead to spontaneous abortion or severe congenital retardation
   6) Treatment: pyrimethamine plus sulfadiazine plus folinic acid (folinic acid is used to prevent folate deficiency caused by the drugs—**note that folate cannot be used because folate is**

upstream of the drug-induced block in the biosynthetic pathway, folinic acid is used because it is downstream of the block)

7) Resistance: none, but alternative therapies are required in sulfa-allergic patients
8) Prophylaxis: AIDS patients and pregnant women should avoid kittens and should not clean kitty litter, cook meats thoroughly, **AIDS patients with CD4 count <200 per μL should be on trimethoprim-sulfamethoxazole prophylaxis**

e. *Trypanosoma*

1) Appearance: large, crescentic trypomastigotes seen in blood, smaller, circular amastigotes seen in tissues
2) Lab assays: biopsy and serologies
3) Virulence factors: **antigenic shift**—the organism can shift its surface antigens as antibody responses develop, keeping it one step ahead of the immune response
4) Epidemiology: ***T. cruzi* transmitted via reduviid bug**, endemic to Central and South America, which bites the patient and then passes organism through the broken skin by defecating in the bite wound, ***T. gambiense* and *T. rhodesiense* transmitted by the tsetse fly, endemic to Africa**
5) Clinical Diseases:
   a) Chagas disease: caused by *T. cruzi*, **reduviid bug often bites the face near the eyes, so look for Romana's sign, a swollen/puffy cheek near the eye**, acutely the organism causes lymphoreticular disease with fever, lymphadenopathy, and hepatosplenomegaly, chronic persistence of the organism leads to amastigote invasion of the heart and colon, **causing heart block and myocarditis/dilated cardiomyopathy as well as megacolon, and achalasia**
   b) African Sleeping Sickness: caused by *T. gambiense* and *T. rhodesiense*, presents with an ulcer at the site of the fly bite, **can lead to either an acute, severe encephalitis with rapid decline in CNS function leading to coma, or a chronic course over several years**
6) Treatment:
   a) Chagas disease: if caught during acute infection, nifurtimox effective, there is no antimicrobial therapy for chronic disease; however, **pacemakers are crucial if heart block develops**
   b) African Sleeping Sickness**: suramin or melarsoprol may be effective prior to onset of CNS disease** but they don't cross the blood brain barrier well, so useless once CNS disease sets in
7) Resistance: unusual
8) Prophylaxis: insect netting, insect repellent

TABLE 4.3 Summary of Invasive Protozoa

	TRANSMISSION	EPIDEMIOLOGY	TISSUE TROPISM
Babesia	Tick bite	• Coastal New England	Red blood cells
Leishmania	Sandfly bite	• Mucocutaneous dz in Central/South America • Cutaneous dz in Central/South America & Asia/Africa • Visceral dz in Asia/Africa	Macrophages
Plasmodium	*Anopheles* mosquito	Africa, Middle East, Caribbean, Central/ South America, SE Asia	RBCs (liver for *P. vivax/ovale*)
Toxoplasma	Cat feces or cysts in undercooked meat	Ubiquitous	Brain
Trypanosoma	• Reduviid bug (*cruzi*) • Tsetse fly (other *spp*)	• Central/South America (*cruzi*) • Africa (other *spp*)	Heart (*cruzi*), CNS (other *spp*)

B. Metazoa (Multicellular Animals)

1. Tapeworms (cestodes)
   a. *Diphyllobothrium latum*
      1) Appearance: **longest tapeworm**, up to 30 feet
      2) Lab assays: stool O&P
      3) Virulence factors: none significant
      4) Epidemiology: **acquired by consuming raw, freshwater fish**
      5) Clinical Diseases: weight loss, diarrhea, **vitamin B12 deficiency**
      6) Treatment: praziquantel/niclosamide
      7) Resistance: none
      8) Prophylaxis: cook fish
   b. *Echinococcus*
      1) Appearance: small tapeworm
      2) Lab assays: stool O&P
      3) Virulence factors: none significant
      4) Epidemiology: fecal oral transmission from **dog feces, with sheep an important intermediate host—thus shepherds are commonly patients**
      5) Clinical Diseases: hydatid cysts in any organ of the body, if cysts rupture can cause fatal anaphylaxis

6) Treatment: careful surgical excystation with or without albendazole
7) Resistance: none
8) Prophylaxis: good hygiene

c. *Hymenolepis nana*
1) Appearance: small tapeworm (up to 5 cm long)
2) Lab assays: stool O&P
3) Virulence factors: none significant
4) Epidemiology: fecal oral transmission, with humans as major host
5) Clinical Diseases: typically asymptomatic
6) Treatment: praziquantel/niclosamide
7) Resistance: none
8) Prophylaxis: good hygiene

d. *Taenia saginata* (beef tapeworm)
1) Appearance: tapeworm can be several meters long
2) Lab assays: stool O&P
3) Virulence factors: none significant
4) Epidemiology: **ingestion of undercooked beef**, transmitted to cattle via fecal-oral route
5) Clinical Diseases: typically asymptomatic, although some patients might suffer discomfort (and embarrassment!) due to the **occasional protrusion of the tapeworm tail from the anus**
6) Treatment: praziquantel/niclosamide
7) Resistance: none
8) Prophylaxis: good hygiene

e. *Taenia solium* (pork tapeworm)
1) Appearance: tapeworm can be several meters long
2) Lab assays: stool O&P
3) Virulence factors: none significant
4) Epidemiology: **can be transmitted either by ingestion of larva in undercooked pork causing tapeworm infection of gut, or by ingestion of eggs in food due to fecal contamination**, eggs mature into larva in gut and burrow into tissues, causing cysticercosis
5) Clinical Diseases:
a) Tapeworm infection: like *T. saginata*, often asymptomatic
b) Cysticercosis: space-occupying lesions occur in tissues, often in brain, and death of the larva induces inflammatory response which can cause seizures and chemical meningitis—**new onset seizures in an immigrant from Latin America is neurocysticercosis until proven otherwise**
6) Treatment: seizure medications, steroids for edema, with or without albendazole or praziquantel

TABLE 4.4 Summary of Tapeworms (cestodes)

	TRANSMISSION	CLINICAL Dz
Diphyllobothrium	Raw fish	Weight loss, diarrhea, B12 deficiency
Echinococcus	Dog feces or ingestion of infected sheep meat	Causes anaphylaxis when cysts rupture often after trauma/ traffic accident
Hymenolepis	Human feces	Asymptomatic
Taenia saginata	Undercooked beef	Typically asymptomatic, worm may protrude from anus
Taenia solium	Undercooked pork or human feces	Asymptomatic tapeworm infection, or ingestion of eggs in feces causes cysticercosis, which presents with seizures and chemical meningitis from ruptured cysts in the brain

7) Resistance: none
8) Prophylaxis: good hygiene

2. Flukes (trematodes)
   a. *Clonorchis sinensis* (**Asian liver fluke**)
      1) Appearance: not remarkable
      2) Lab assays: stool O&P
      3) Virulence factors: none significant
      4) Epidemiology: **transmitted by ingestion of raw freshwater fish, endemic to Asia**
      5) Clinical Diseases: may be asymptomatic, however, the flukes lodge in the liver and can cause **hepatitis and biliary obstruction** ultimately leading to cirrhosis or hepatocellular carcinoma
      6) Treatment: praziquantel/albendazole
      7) Resistance: none
      8) Prophylaxis: cook fish
   b. *Paragonimus westermani* (**Asian lung fluke**)
      1) Appearance: not remarkable
      2) Lab assays: stool O&P
      3) Virulence factors: none significant
      4) Epidemiology: transmitted by **ingestion of raw crab meat, endemic to Asia**, organism penetrates intestinal wall, migrates through the diaphragm to the lung and can thus be transmitted either by feces or sputum

5) Clinical Diseases: chronic cough, hemoptysis and dyspnea
6) Treatment: praziquantel
7) Resistance: none
8) Prophylaxis: cook crab meat

c. *Schistosoma japonicum, mansoni,* and *haematobium*
1) Appearance: ova are ovoid with sharp protuberance called a spine at one end, *S. haematobium* ova have a big spine at the very terminus of the egg, *S. mansoni* ovum have big spine off to the side (about 2 o'clock if the terminus is 12 noon), while *S. japonicum* have a less prominent spine (see Figure 4.4)
2) Lab assays: stool O&P
3) Virulence factors: none significant
4) Epidemiology:
a) Infection occurs by direct **penetration of human skin by free-swimming larva, life-cycle requires freshwater environment with snails** (which are intermediate hosts)
b) *S. mansoni* occurs in tropical areas around the world, including Africa, the Middle East, and South America, but **does not occur in Asia**
c) *S. japonicum* **occurs in Asia**
d) *S. haematobium* occurs in Africa and the Middle East

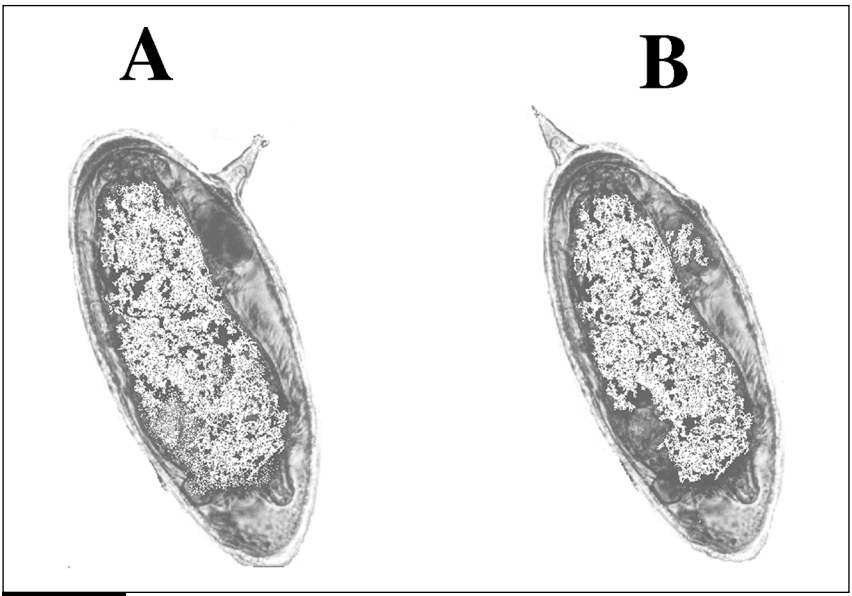

FIGURE 4.4 **(A) *Schistosoma mansoni*, (B) *Schistosoma haematobium***

A) The classic appearance of Schistosoma mansoni*, with a terminal spine at approximately 2 o'clock. B) In contrast, the spine of* Schistosoma haematobium *is at the very terminus (12 o'clock).*

TABLE 4.5 Summary of Flukes (trematodes)

	TRANSMISSION	LOCATION	CLINICAL Dz
Clonorchis	Raw freshwater fish	Asia	Hepatitis, biliary obstruction
Paragonimus	Raw crab meat	Asia	Chronic cough, hemoptysis
Schistosoma haematobium	Skin penetration by freshwater larvae	Africa, Middle East	Hematuria, transitional bladder CA
Schistosoma japonicum	Skin penetration by freshwater larvae	Asia	Granulomatous hepatitis, cirrhosis, portal hypertension
Schistosoma mansoni	Skin penetration by freshwater larvae	Africa, Middle East, South America	Granulomatous hepatitis, cirrhosis, portal hypertension

5) Clinical Diseases:
   a) *S. japonicum* & *mansoni*: acute infection **causes pruritis and erythematous eruption at the site of skin penetration**, after several weeks fevers and lymphadenopathy begin, tissue penetration causes **marked eosinophilia, adult schistosomes reside in the liver venules and their eggs cause granulomatous reaction in the liver, leading to portal hypertension and hepatosplenomegaly, can induce cirrhosis** leading to all the usual sequelae
   b) *S. hematobium*: **adult worms reside in the bladder venous plexus**, and their eggs cause granulomas and fibrosis in the bladder, leading to **hematuria, can also induce transitional cell carcinoma of the bladder**
6) Treatment: praziquantel
7) Resistance: starting to be seen for *S. mansoni*
8) Prophylaxis: avoid swimming in waters with host snails

3. Intestinal Roundworms (nematodes)
   a. *Ascaris lumbricoides*
      1) Appearance: large roundworms, can be a foot long, and can form mass-like conglomerations in the intestines (see Figure 4.5)
      2) Lab assays: stool O&P
      3) Virulence factors: none significant
      4) Epidemiology: fecal-oral transmission of eggs, after ingestion, eggs hatch in the intestine, the larva penetrate the intestinal wall and enter the bloodstream, then the larva escape into the lungs, penetrate the alveoli and are coughed up the trachea

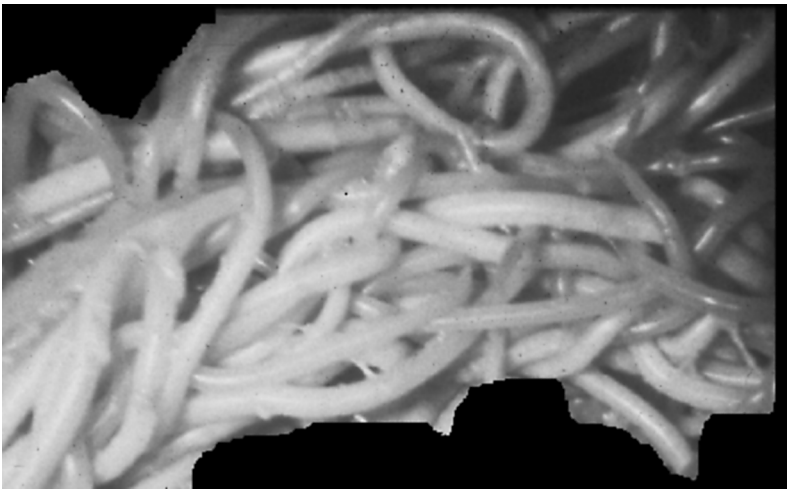

FIGURE 4.5 ***Ascaris* Worms**

Appearance of a mass of Ascaris *worms.*

so that they are swallowed back down into the intestine where they mature into adults and pass more eggs—**this is the most common parasitic infection in the world, with up to 1 billion people infected**

5) Clinical Diseases: although infection can be asymptomatic, **eosinophilic pneumonia** occurs during larval migration into the alveoli, malnutrition can occur due to adult worms scavenging nutrients in the gut, and **bowel obstruction** can occur due to luminal occlusion by large numbers of the large adult worms
6) Treatment: albendazole or mebendazole, pyrantel pamoate as 2nd line
7) Resistance: none
8) Prophylaxis: good hygiene

b. *Enterobius vermicularis* (pinworm)
1) Appearance: small white worms, 1 cm or less
2) Lab assays: scotch tape test = place scotch tape over anus and examine on a slide, the tape picks up the eggs which are visible under the microscope, worms can be directly visualized by stool O&P (but eggs are not present in stool)
3) Virulence factors: none significant
4) Epidemiology: **most common helminthic infection in the U.S.**, transmission is fecal-oral, **at night the female worm migrates out the anus and lays eggs in the perianal area**
5) Clinical Diseases: **perianal pruritus**

6) Treatment: albendazole or mebendazole, pyrantel pamoate as 2nd line
7) Resistance: none
8) Prophylaxis: good hygiene

c. Hookworm (*Ancylostoma duodenale* and *Necator americanus*)
1) Appearance: long, thin worms, *Necator* has cutting plates to grab hold of the intestinal wall while *Ancylostoma* uses teeth
2) Lab assays: stool O&P
3) Virulence factors: none significant
4) Epidemiology: ***Ancylostoma duodenale* is found in most of the underdeveloped world (Old World hookworm), while *Necator americanus* (New World hookworm) is found in the southeastern U.S., infection occurs via direct penetration of skin in contact with moist soil containing larvae**, like *Ascaris* the larvae migrate via blood to the lungs where they are coughed up and swallowed, allowing mature adults to develop in the intestines and to pass eggs in the stool
5) Clinical Diseases: a pruritic, **erythematous dermatitis occurs at the site in the skin where the larvae penetrate, and eosinophilic pneumonia occurs** during transmigration of the larvae through the alveoli, some time after infection iron deficient anemia develops (**hookworm is the #1 cause of iron deficiency anemia in the world**), with all the usual anemia symptoms (e.g., weakness, fatigue, etc.), eosinophilia is typical
6) Treatment: albendazole or mebendazole, pyrantel pamoate as 2nd line
7) Resistance: none
8) Prophylaxis: good sanitation, avoid direct skin contact with contaminated soil (e.g., don't walk barefoot through the soil)

d. *Strongyloides stercoralis*
1) Appearance: small round worms (see Figure 4.6)
2) Lab assays: stool O&P
3) Virulence factors: none significant
4) Epidemiology: endemic throughout the tropical world, and also in the southeastern U.S., direct contact of skin with **soil contaminated by feces allows larval penetration into the host**, the larva migrates via blood to the lung where they are coughed up and swallowed, allowing adult maturation in the intestines, the adult worms lay eggs which either are defecated out to start the next cycle, or mature into larva within the infected host, allowing another round of infection within the same host in a process called autoinfection, **autoinfection can lead to overwhelming infection in immunocompromised patients**
5) Clinical Diseases: **Cutaneous Larva Migrans = severe local contact dermatitis occurs at the site of skin penetration**, pts can be asymptomatic, but some develop diarrhea, eosinophilic

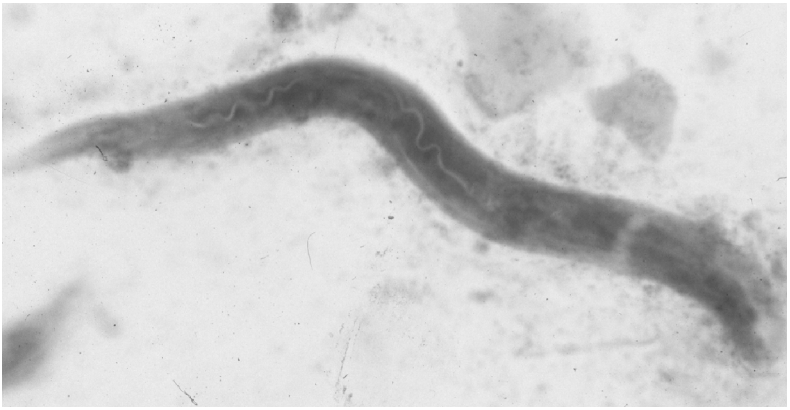

FIGURE 4.6 ***Strongyloides***

Appearance of Strongyloides *in the sputum of an infected patient.*

pneumonia, or sepsis from bacterial translocation across a damaged gut wall, **peripheral eosinophilia is prominent**

6) Treatment: ivermectin or albendazole
7) Resistance: none
8) Prophylaxis: good sanitation, avoid bare skin contact with contaminated soil (e.g., don't walk barefoot through the soil)

e. *Trichuris trichiura* (whipworm)
1) Appearance: long thin worm, with a thread-like tail extending twice the length of the worm
2) Lab assays: stool O&P
3) Virulence factors: none significant
4) Epidemiology: fecal-oral transmission of eggs, eggs mature into adults in intestine, which then lay more eggs, there is no tissue phase
5) Clinical Diseases: often asymptomatic, but may cause invasive diarrhea, does NOT typically cause iron-deficient anemia, **in patients with heavy worm burden rectal prolapse can occur**
6) Treatment: albendazole or mebendazole
7) Resistance: none
8) Prophylaxis: good sanitation and good hygiene

f. *Trichinella spiralis*
1) Appearance: larvae are oval with ring-like centers, found in tissue, particularly muscle
2) Lab assays: **muscle biopsy, note stool O&P not helpful, look for marked eosinophilia**

3) Virulence factors: none significant
4) Epidemiology: typically transmitted via **ingestion of undercooked pig, bear, or deer meat** containing larvae, larvae mature in the intestine, and lay eggs which penetrate into the bloodstream, seeding striated muscle throughout the body
5) Clinical Diseases: **Trichinosis**: an initial diarrhea is followed within 2 weeks by **severe myositis, headache with diffuse muscle aches, high fever, and extreme eosinophilia (up to 90% of peripheral white blood cells)**, can also cause CNS and cardiac damage
6) Treatment: albendazole or mebendazole with or without corticosteroids
7) Resistance: none
8) Prophylaxis: cook meats thoroughly

4. Tissue Roundworms (nematodes)
   a. *Dracunculus medinensis*
      1) Appearance: coiled worm up to a foot or more long can be seen burrowed beneath the skin, skin typically blisters and then ulcerates over the worm

TABLE 4.6 Summary of Intestinal Roundworms (nematodes)

	TRANSMISSION	CLINICAL Dz
Ascaris	Fecal oral	Eosinophilic pneumonia, bowel obstruction
Enterobius	Fecal oral	Perianal pruritius in children
Ancylostoma	Skin penetration by larvae in moist soil	Pruritic, erythematosus dermatitis at site of penetration, iron deficient anemia and eosinophilia, occurs in Europe and Asia
Necator	Skin penetration by larvae in moist soil	Pruritic, erythematosus dermatitis at site of penetration, iron deficient anemia and eosinophilia, occurs in the SE U.S.
Strongyloides	Skin penetration by larvae in moist soil	Local contact dermatitis at site of skin penetration, diarrhea, eosinophilic pneumonia, sepsis
Trichuris	Fecal oral	Invasive diarrhea, rectal prolapse
Trichinella	Undercooked pork or game (bear or deer)	Diarrhea, severe myositis, headache, extreme eosinophilia

2) Lab assays: none
3) Virulence factors: none significant
4) Epidemiology: transmission is via **ingestion of fresh-water crustaceans containing larvae**, larvae mature in the intestine and the adult migrates to skin, the disease is on the decline in Africa due to WHO efforts to eradicate
5) Clinical Diseases: pruritic and painful welts overlying the subcutaneous worm, with ulceration over the worm's head
6) Treatment: surgical withdrawal of the worm
7) Resistance: none significant
8) Prophylaxis: boil or filter suspect drinking water, avoid ingestion

b. *Loa loa*
1) Appearance: can be seen as small (centimeter) subconjunctival worm burrowing across the eye or skin
2) Lab assays: thick and thin smear to identify parasites in the blood
3) Virulence factors: none significant
4) Epidemiology: endemic to Africa, transmitted by the bite of the deer fly
5) Clinical Diseases: **dermatitis or conjunctivitis caused by hypersensitivity to migrating worm in skin or eye**
6) Treatment: diethylcarbamazine, surgical excision may be required for conjunctival infections
7) Resistance: none
8) Prophylaxis: eradication of deer fly vector

c. *Oncocerca volvulus*
1) Appearance: worm less than a millimeter long
2) Lab assays: skin biopsy revealing the parasite
3) Virulence factors: none significant
4) Epidemiology: endemic to Africa, **particularly riverbeds, transmitted by bite of blackfly**, the organisms do not travel through blood, instead migrate subcutaneously
5) Clinical Diseases: **causes "river blindness"** (that's right, the people go blind from worm migration into the eyes, hence the name)
6) Treatment: ivermectin plus corticosteroids for patients with eye infection
7) Resistance: none
8) Prophylaxis: eradicate blackfly vector, ivermectin can be taken prophylactically

d. *Toxocara canis*
1) Appearance: not significant
2) Lab assays: tissue biopsy
3) Virulence factors: none significant

4) Epidemiology: transmitted via **dog feces contaminating soil** and foodstuffs, eggs hatch into larvae in the intestine, the larvae then disseminate to multiple tissues
5) Clinical Diseases**: "Visceral Larva Migrans" = diffuse granulomatous reactions causing fevers, myalgias, headache, CNS disease and retinal disease**, with a **prominent eosinophilia**, typically presents in children playing in soil with dogs
6) Treatment: treat symptoms with corticosteroids and antihistamines, ivermectin, albendazole, or diethylcarbamazine for worms
7) Resistance: none
8) Prophylaxis: good sanitation and hygiene

e. *Wuchereria bancrofti* and *Brugia malayi*
1) Appearance: worm less than a millimeter long
2) Lab assays: thick blood smears with blood drawn at night
3) Virulence factors: none significant
4) Epidemiology: ***Wuchereria* is endemic to Africa while *Brugia* is endemic to Asia** (particularly Malaysia hence the species name *malayi*), **both transmitted by mosquito bites**, organisms mature in lymph nodes and then circulate in the blood, particularly at night
5) Clinical Diseases: **elephantiasis**, obstruction of lymphatics leads to severe lymphedema
6) Treatment: ivermectin effective against larvae but not adult worms
7) Resistance: none
8) Prophylaxis: prevention of mosquito bites

TABLE 4.7 Summary of Invasive Roundworms (nematodes)

	TRANSMISSION	CLINICAL Dz
Dracunculus	Freshwater crustaceans	Welts overlying the subcutaneous worm
Loa	Deerfly bite	Hypersensitivity to migrating worm
Oncocerca	Blackfly bite	Blindness
Toxocara	Dog feces	Visceral larva migrans = diffuse granulomas, retinitis, eosinophilia, headache, myalgias
Wuchereria and *Brugia*	Mosquito	Elephantiasis

TABLE 4.8 Overall Summary of Parasites

	KEY WORD/PHRASE	TREATMENT
	GI/GU Protozoa	
Cryptosporidium	• Diarrhea in AIDS patient	Immune reconstitution
Entamoeba	• Amoebic dysentery • Amoebic liver abscess	Metronidazole plus iodoquinol
Giardia	• Chronic non-bloody diarrhea in a hiker/camper	Metronidazole
Trichomonas	• Vaginitis with green, frothy discharge • Frequent recurrences because sexual partner needs treatment as well	Metronidazole
	Invasive Protozoa	
Leishmania	• Sandfly bite in Asia, Africa, Latin America • Cutaneous ulcer • Lymphadenopathy, hepatosplenomegaly	Sodium stibogluconate
Plasmodium	• Mosquito bite in Africa, Mediterranean, Latin America • Shaking rigors, severe headache, myalgias • Cyclical fevers	Chloroquine ± primaquine
Toxoplasma	• Exposure to kitten feces or poorly cooked meat • Typically in AIDS pts with ring-enhancing brain lesions	Pyrimethamine + sulfadiazine + folinic acid
Trypanosoma	• Reduviid bug bite in Latin America • Tsetse fly bite in Africa • Romana's sign = swelling near eye where bite occurred • Heart block in young person Megacolon	Nifurtimox or suramin acutely, nothing works for chronic dz

(*Continued*)

TABLE 4.8 ***Continued***

	KEY WORD/PHRASE	TREATMENT
	Tapeworms (cestodes)	
Diphyllobothrium	• Raw fish consumption • B_{12} deficiency	Albendazole
Echinococcus	• Shepherds, exposure to dogs and sheep • Large cysts seen in liver or lung	Albendazole and surgery
Hymenolepis	• Dog feces	Albendazole
Taenia saginata	• Undercooked beef • Tapeworm protrusion from anus	Albendazole
Taenia solium	• Undercooked pork or fecal-oral • Seizures and encephalitis in a Hispanic person	Albendazole ± seizure medicine
	Flukes (trematodes)	
Clonorchis	• Asian liver fluke • Raw freshwater fish	Albendazole
Paragonimus	• Asian lung fluke • Raw crab meat	Albendazole
Schistosoma	• Swimming in freshwater with snails nearby • Portal hypertension • Hematuria, bladder cancer	Albendazole
	Intestinal Roundworms (nematodes)	
Ascaris	• Eosinophilic pneumonia • Bowel obstruction • Iron deficiency	Albendazole
Enterobius	• Kid with itchy anus • Small white worms seen near anus • Eggs picked up from anus with scotch-tape	Albendazole

TABLE 4.8 ***Continued***

	KEY WORD/PHRASE	TREATMENT
Intestinal Roundworms (nematodes) *(Continued)*		
Ancylostoma	• Dermatitis at site of contact with moist soil • Iron deficiency anemia	Albendazole
Necator	• Dermatitis at site of contact with moist soil • Iron deficiency anemia • Southeastern U.S., poor areas	Albendazole
Strongyloides	• Local contact dermatitis (cutaneous larva migrans) at site of contact with moist soil • Eosinophilic pneumonia, auto infection	Ivermectin or albendazole
Trichuris	• Rectal prolapse	Albendazole
Trichinella	• Undercooked pig, bear, or deer meat • Severe myalgias with extreme eosinophilia	Albendazole
Invasive Roundworms (nematodes)		
Dracunculus	• Long worm seen coiled beneath soil	Worm extraction
Loa	• Small worm seen underneath conjunctiva	Diethylcarbamazine
Oncocerca	• Blackfly bite • River blindness in Africa	Ivermectin
Toxocara	• Visceral larva migrans • Retinal disease • Prominent eosinophilia	Steroids, antihistamines,? ivermectin
Wuchereria (Brugia)	• Elephantiasis	Ivermectin