



Medfools Parasites Chart for USMLE I

Parasitology Notes for USMLE I

“Heey.... fecal oral transmission???”

PROTOZOA – INTESTINAL and UROGENITAL

<i>Entamoeba Histolytica</i> (bloody diarrhea)					
Diseases	Characteristics	Habitat/Trans	Pathogenesis	Diagnosis	Treatment
Amebic dysentery- bloody, mucus diarrhea, liver and pulmonary abscess. Mostly asymptomatic.	Trophozoite has single nucleus and ingested RBC , cysts have 4 small nuclei. Prevalent among male homosexuals.	No animal reservoir. Fecal-oral transmission.	Excyst in ileum, invade colonic epithelium, cause necrosis (teardrop ulcer), spread (liver, lung)	ID trophozoites and cysts in stool. Serology positive in invasive amebiasis Absence of PMNs.	Metronidazole, iodoquinol. Cysts removed by filtration, killed by boiling, not chlorination.
<i>Giardia lamblia</i> (Nonbloody diarrhea)					
Giardiasis- nonbloody, foul smelling diarrhea, nausea, anorexia, flatulence, abdominal cramps for wks/months. Outbreaks at day care centers, mental hospitals.	Pear shaped trophozoite , 2 nuclei, 4 pairs flagella, suction disk. Thick walled oval cyst w/ 4 nuclei 5% US stools have cysts, 50% asymptomatic carriers.	Fecal contamination of food/water. Homosexual transmission.	Excyst in duodenum, attaches-NO invasion, inflammation, malabsorption of protein/fat.	Trophozoites/cysts in stool, string test , serology.	Metronidazole Cysts removed by filtration, killed by boiling, iodine, not chlorination.
<i>Cryptosporidium</i> (Nonbloody diarrhea)					
Cryptosporidiosis in immunocompromised- watery nonbloody diarrhea, fluid loss, malnutrition		Fecal-oral transmission of oocysts from human/animal sources.	Complex cycle occurs in epithelial cells of jejunum, NO invasion	ID kinyoun acid-fast (red) oocytes in stool smear.	No effective therapy. Try azithromycin
<i>Trichomonas vaginalis</i> (NO cyst form)					
Trichomoniasis- vaginal itching w/ watery foul smelling, green vaginal discharge. Men asymptomatic.	No cyst. Pear shaped trophozoites, 4 anterior flagella, undulating membrane (jerky movement) 25-50% sexually active women infected	Sexual contact.	Infects vagina, prostate. Predisposing factor is loss of vaginal acidity		Both partners: metronidazole

PROTOZOA – BLOOD and TISSUE

Plasmodium

Diseases	Characteristics	Habitat/Trans	Pathogenesis	Diagnosis	Treatment
<p>Malaria- fever, chills, HA, myalgias, arthralgias two wks post bite. Fever spikes accompanied by nausea, vomiting, abdominal pain, drenching sweats. Splenomegaly is common. Anemia due to lysis of RBCs, splenic sequestration of damaged RBCs.</p> <p><i>P. falciparum</i> causes most severe malaria-can infect RBCs at all stages and causes adherence of RBCs to cerebral vascular endothelium via knob proteins (“cerebral malaria”).</p> <p><i>P. ovale</i>, <i>P. vivax</i> cause benign malaria.</p> <p><i>P. malariae</i> produces fevers each 72 hours, others every 48 hrs. <i>P. falciparum</i> can cause almost constant fever.</p>	<p>Endemic to 91 countries, 300-500 million cases, mostly sub-Saharan Africa.</p>	<p>Female <i>Anopheles</i> mosquito</p>	<p><u>Asexual Schizogony in humans</u> <u>Exoerythrocytic phase:</u> Mosquito injects sporozoites which attack hepatocytes, sporozoites replicate, differentiate into merozoites which infect RBCs. <u>Erythrocytic phase:</u> Merozoites in RBCs, differentiate into ring shaped trophozoites, develop into schizonts filled with merozoites. Merozoites lyse RBCs at regular intervals and further infect RBCs. <u>Sexual Sporogony in mosquitoes</u> Some blood merozoites develop into male/female gametocytes in RBCs. Female mosquito eats these RBCs, form one female macrogamete or 8 spermlike microgamete in gut. Diploid zygote differentiates into motile ookinete which burrows through gut wall. Oocyst w/ haploid sporozoites form on stomach wall, sporozoites released and migrate to mosquito salivary glands. Female mosquito injects sporozoites into next human victim (sucker.)</p>	<p>Thick and thin Giemsa stained smears. Usually ring shaped trophozoites are ID.</p>	<p>Acute malaria w/ chloroquine which kills merozoites, sporozoites in blood. Mefloquine used in <i>P. falciparum</i> resistant to chloroquine. Primaquine to get hepatic stages of <i>P. ovale</i> and <i>P. vivax</i>.</p> <p>Prophylax w/ chloroquine/ mefloquine.</p> <p>Prevent w/ pyrimethamine impregnated bednets. (kills mosquito cycle)</p>

Toxoplasma gondii

<p>Toxoplasmosis- usually asymptomatic, can cause hererophil antibody-negative infectious mononucleosis. Life threatening encephalitis in immunocompromised b/c reactivation of dormant cysts. Transplacental transmission results in stillbirth or fetal infection (encephalitis, retinitis, microcephaly, mental retardation)</p>	<p>5-50% seropositivity rate in US</p>	<p>Cat host. Humans eat cysts in meat or cat feces</p>	<p>Cysts rupture and invade gut mucosa, ingested by macrophages, differentiate into tachyzoites (rapidly multiplying trophozoites), disseminate to brain, muscle.</p>	<p>Serology or crescent shaped trophozoites</p>	<p>Sulfadiazine and pyrimethamine</p>
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Trypanosoma cruzi (Chagas disease)

<p>Chagas disease- <u>Acute:</u> edematous nodule (Chagoma) at bite site (<u>periorbital</u>, perioral), fever lymphadenopathy, H/Smegaly, resolves in 2 months <u>Indeterminate-</u> low levels of parasitemia, serological evidence of infection <u>Chronic- myocarditis</u> (arrythmia, dilated cardiomyopathy, CHF), megacolon, megaesophagus. Death usually by arrythmia.</p>	<p>Rural Central and South America, Southern US</p>	<p>Humans, animal reservoirs. Reduviid (kissing) bug as vector</p>	<p>Reduviid bug ingests trypomastigotes from animal’s blood, trypomastigotes multiply in gut, excreted at bite, invade skin, disseminate in blood, amastigotes proliferate inside macrophages and myocardium, amastigotes differentiated into blood borne trypomastigotes which are taken up by reduviid bug at next meal.</p>	<p>Thick and thin blood smears for trypomastigotes. Serology Muscle biopsy revealing amastigotes. Xenodiagnosis</p>	<p>Nifurtimox kills trypomastigotes, NO therapy for chronic form.</p>
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<i>Trypanosoma gambiense/rhodesiense</i> (African sleeping sickness)					
<p>Trypanosomal chancre (many organisms) at bite site.</p> <p>African sleeping sickness- Cyclical fever, lymphadenopathy, demyelinating encephalitis, HA, insomnia, slurred speech, ataxia, mood changes, somnolence, coma.</p>	<p>Trypomastigotes, no amastigotes.</p> <p>Sub-Saharan Africa</p>	<p>Tsetse fly vector. “Sleep-Sleep fly”</p> <p>Human and animal reservoirs.</p>	<p>Skin to blood/LN to CNS</p> <p>Antigenic variation of VSGs –variable surface glycoproteins</p>	<p>ID trypomastigotes in blood smear, LN or CSF.</p>	<p>Suramin in pre encephalitis stage. Melarsoprol for CNS involvement.</p>
<i>Leishmania donovani</i> (Kala-azar)					
<p>Kala-azar (visceral leishmaniasis)- RES involvement. Chronic low grade fever, anorexia, weight loss, skin hyperpigmentation. Bone marrow involvement results in anemia, leukopenia, thrombocytopenia and secondary infections, coagulopathies. Massive splenomegaly. Disease lasts months to years.</p>	<p>Mediterranean/ Middle East, Saharan Africa, India</p>	<p>Sandfly vector. Dog, fox, rodent reservoirs</p>	<p>Female sandfly ingests macrophages containing amastigotes, amastigotes differentiate into promastigotes in sandfly gut, multiply, migrate to pharynx, transmitted to humans w/ bite, in human macrophages promastigotes differentiate back into amastigotes.</p>	<p>ID amastigotes in spleen, LN, BM biopsy.</p> <p>Leshmanin DTH skin test negative, poor cellular response.</p>	<p>Sodium stibogluconate</p>
<i>Leishmania tropica, mexicana, braziliensis</i> (Cutaneous, mucocutaneous)					
<p>Ulcers confined to skin/mucous membranes, often superinfected by bacteria. Multiple satellite nodules coalesce and ulcerate. In diffuse cutaneous leishmaniasis, lesions grow/spread all over skin. Disfiguring granulomatous lesions destroy nasal cartilage (like lepromatous leprosy)</p> <p><i>L. tropica</i> – Cutaneous – Old world <i>L. mexicana</i> – Cutaneous – Americas <i>L. braziliensis</i> – Mucocutaneous -- Central/South America</p>		<p>Sandfly vector. Forest rodent reservoirs</p>	<p>Basically like <i>L. donovani</i></p>	<p>ID amastigotes in skin lesions</p>	<p>Sodium stibogluconate</p>
<i>Pneumocystis carinii</i> (PCP)					
<p>Pneumonia- acute fever, nonproductive cough, dyspnea, tachypnea. CXR shows diffuse, bilateral infiltrates or may be normal. Untreated cases fatal.</p>	<p>Classified as a fungus</p>	<p>Inhalation of cysts. NOT person-person</p>	<p>Cysts establish life long latent infection in lungs. In immunocompromised, cysts reactivate and induce exudative inflammation which compromises gas exchange in the alveoli.</p>	<p>Silver stained induced sputum specimen, bronchoalveolar lavage fluid, bronchial tissue biopsy reveal <i>pneumocystis</i>.</p>	<p>TMP-SMZ Pentamidine</p> <p>Prophylax when CD4 < 200</p>

CESTODES

Hermaphroditic flatworms

<i>Taenia solium (Pork tapeworm)</i>					
Diseases	Characteristics	Habitat/Trans	Pathogenesis	Diagnosis	Treatment
Cysticercosis - HA, vomiting, seizures, uveitis, retinitis Taeniasis - usually asymptomatic	Solex w/ 4 suckers, circle of hooks		Gravid proglottids ingested by pigs (intermediate hosts), develop into larvae which burrow holes in blood vessels, go to skeletal muscle. Humans eat raw pork containing cysticerci (encysted larvae), mature in gut. If humans eat eggs, larvae spread to eyes/brain where they encyst to form cysticerci. Space filled/calcified lesions.	ID gravid proglottids w/ 5-10 uterine branches in stool. Cysts by Xray or CT. Larvae may be floating in vitreous.	Niclosamide Praziquantel Treat asymptomatics to prevent autoinoculation, cysticercosis
<i>Taenia saginata (Beef tapeworm)</i>					
Taeniasis- asymptomatic No cysticercosis in humans	Solex w/ 4 suckers, no hooklets		Same life cycle, except cattle host.	Gravid proglottids w/ 15-25 uterine branches. (remember COWS are bigger than PIGS, more branches)	Niclosamide Praziquantel
<i>Diphyllobothrium latum (Fish tapeworm)</i>					
Mostly asymptomatic or Megaloblastic anemia (B12 deficiency) due to preferred uptake of B12 by worm.	Solex w/sucking grooves, no hooks. Gravid uterus in rosette form. Eggs oval w/ operculum. (diagnostic)	Scandinavia and Japan.	Eggs in fresh water, ingested into crustaceans , differentiate into larvae for fish . Humans infected by eating undercooked fish.		Niclosamide Praziquantel
<i>Echinococcus granulosus (Dog tapeworm)</i>					
Unilocular hydatid cyst disease - usually asymptomatic , may cause hepatic dysfunction. Cyst contents cause anaphylaxis	Scolex and 3 proglottids (small)	Dogs definitive hosts, sheep intermediate, humans dead end.	Worms in dog intestine dump eggs, ingested by sheep or humans. Oncospheres form and spread to organs (liver), form hydatid cysts . Dogs eat slaughtered sheep, cycle complete		Niclosamide Praziquantel Stop feeding the dogs sheep bits!
<i>Hymenolepis nana (Dwarf tapeworm)</i>					
Usually asymptomatic	Most common tapeworm in US, esp. SE USA	No intermediate host	Eggs directly infectious for humans. Many worms found , unlike others which exist singly	Eggs in stool	Niclosamide Praziquantel

TREMATODES					Flukes																								
<i>Schistosoma</i> (snails)																													
Diseases	Characteristics	Habitat/Trans	Pathogenesis	Diagnosis	Treatment																								
<p>Schistosomiasis- itching at site of penetration (“swimmer’s itch”), fever, chills, diarrhea, lymphadenopathy, eosinophilia. Chronic infection leads to GI hemorrhage, H/Smegaly, death by ruptured esophageal varicities.</p> <p>S. haematobium infection can cause bladder cancer.</p>	<p>Schistosomes are separate sexes that live attached to each other. Females reside in male grooves. 200 million cases worldwide</p>		<p>Free swimming cercariae penetrate human skin, differentiate into larvae, enter venous circulation, mature into adult form. Females lay eggs spread to gut or bladder, excreted in feces or urine. Eggs hatch in fresh water, penetrate snails, differentiate to free swimming cercariae.</p> <p>Pathogenesis mediated by host granuloma response to antigenic eggs in organs. Schistosomes coast themselves in host antigens, immune evasion.</p>	<p>Characteristic eggs in feces, urine.</p>	<p>Prizaquantel</p>																								
<table border="0"> <tr> <td><u>Trematode</u></td> <td><u>Affected veins</u></td> <td><u>Eggs</u></td> <td><u>Endemic areas</u></td> <td></td> <td></td> </tr> <tr> <td><i>S. mansoni</i></td> <td>Large intestine</td> <td>Large lateral spine</td> <td>Africa, Caribbean</td> <td></td> <td></td> </tr> <tr> <td><i>S. japonicum</i></td> <td>Small intestine</td> <td>Small lateral spine</td> <td>Orient</td> <td></td> <td></td> </tr> <tr> <td><i>S. haematobium</i></td> <td>Bladder</td> <td>Large Terminal spine</td> <td>Africa, Mid East</td> <td></td> <td></td> </tr> </table> <p>(think of bladder as terminal organ of GU system)</p>	<u>Trematode</u>	<u>Affected veins</u>	<u>Eggs</u>	<u>Endemic areas</u>			<i>S. mansoni</i>	Large intestine	Large lateral spine	Africa, Caribbean			<i>S. japonicum</i>	Small intestine	Small lateral spine	Orient			<i>S. haematobium</i>	Bladder	Large Terminal spine	Africa, Mid East							
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<i>Chlonorchis sinensis</i> (snails, fish)																													
<p>Oriental liver fluke- mostly asymptomatic, or upper abdominal pain, anorexia, hepatomegaly, eosinophilia with high worm burden</p>		<p>Korea, China, Japan</p>	<p>Humans infected by eating undercooked fish containing encysted larvae. Larvae excyst in duodenum, immature flukes enter biliary ducts. Host inflammatory response causes hyperplasia/fibrosis of biliary tree. Adults pass eggs in feces. Eggs eaten by fresh water snails (1st intermediate host), hatch in snail gut into free swimming cercariae. Cercariae encyst under scales of fish (2nd intermediate host) and cycle repeats</p>	<p>Typical operculated eggs in stool</p>	<p>Prizaquantel</p>																								
<i>Paragonimus westermani</i> (snails, crabs)																													
<p>Lung fluke- chronic cough w/ bloody sputum (resembles TB), Dyspnea, pleuritic chest pain, recurrent secondary bacterial pneumonias.</p>		<p>Orient, India</p>	<p>Humans eat undercooked crabs, containing encysted larvae which excyst in small intestine. Larvae penetrate intestinal wall and through diaphragm into lung. Adults make eggs which are coughed up, swallowed, excreted into fresh water. Eggs develop into miracidia and enter fresh water snails (1st host) in which they develop into free-swimming cercariae which enter crabs (2nd host) and cycle repeats.</p>	<p>ID operculated eggs in sputum or feces</p>	<p>Prizaquantel</p>																								

NEMATODES - INTESTINAL

Roundworms

<i>Enterobius vermicularis</i> (Pinworm)				
Diseases	Characteristics	Pathogenesis	Diagnosis	Treatment
Perianal itching	Life cycle confined to humans	Humans infected by ingesting eggs. Eggs hatch in small intestine, larvae migrate to colon. Male/female mating, female migrates to anus at night to release eggs . Larvae carried to mouth by fingers which have scratched itchy skin, cycle repeats	Recover eggs by famous Scotch tape method Eggs NOT in stool	Mebendazole kills adults.
<i>Trichuris trichiura</i> (Whipworm)				
Mostly asymptomatic, maybe diarrhea	Worldwide, Southern US	Humans eat eggs in soil contaminated with human feces . Adult worms burrow into intestinal mucosa, do not cause anemia , unlike hookworms	Eggs in stool	Mebendazole
<i>Ascaris lumbricoides</i>				
Ascariasis - mostly asymptomatic, but ascaris pneumonia can result from damage of larvae migration through lung, inducing inflammation with eosinophilic exudate	Common in tropics. Largest nematodes, 25cm.	Humans eat eggs in soil contaminated with human feces . Eggs hatch in small intestine, larvae migrate through gut mucosa into bloodstream to lungs, coughed up and swallowed. Mature in small intestine, persist in lumen, do not attach, live off ingested food . Thousands of eggs laid daily, passed in feces, form embryos in warm soil. Human ingestion completes cycle.	ID corrugated eggs in Stool	Mebendazole Pyrantel pamoate
<i>Ancylostoma/Necator</i> (Hookworm)				
Microcytic anemia - weakness pallor, pneumonia with eosinophilia		Cycle like <i>Ascaris</i> , except <i>larvae</i> in soil penetrate skin. Adults use cutting plates to attach to intestine. Major damage is by loss of blood .	Eggs in stool.	Mebendazole Pyrantel pamoate
<i>Strongyloides stercoralis</i>				
Strongyloidiasis - mostly asymptomatic, pneumonitis can occur , and gut mucosal damage. Eosinophilia can be striking.	Tropics, SE Asia, Southern US	Two life cycles. One in humans, one in soil. Infectious larvae penetrate skin, migrate to lungs, alveoli, trachea where they are swallowed. In small intestine, larvae become adults, enter mucosa and produce eggs. Eggs hatch, larvae passed in stool. In soil , larvae differentiate into male/females, mate, produce infectious larvae .	LARVAE, not eggs in stool	Thiabendazole
<i>Trichinella spiralis</i>				
Trichinosis - Gastroenteritis , followed 1-2 wks later with fever, muscle pain, periorbital edema, eosinophilia .	Worldwide, esp. E. Europe, W. Africa	Pigs reservoir in US . Infection by eating raw pork (or Bear!) containing larvae encysted in muscle. Adult forms arise and release larvae into blood with dissemination to organs. Larvae only develop in striated muscle .	Muscle biopsy .	No Treatment for trichinosis. Thiabendazole for early infections kills adult worms.

NEMATODES - TISSUE

Roundworms

Wuchereria bancrofti (Filariasis)

Diseases	Characteristics	Pathogenesis	Diagnosis	Treatment
<p>Filariasis- adult worms cause obstruction of lymphatics, causing edema. Fever, lymphangitis, cellulitis develop.</p> <p>Elephantiasis- occur in patients repeatedly infected.</p>	Tropics, 200-300 million infected	<p>Female <i>Anopheles</i> mosquito deposits infective larvae on skin while biting. Larvae penetrate skin, enter LN, mature one year later into adults that produce microfilariae. These circulate in blood, esp. at night, and are ingested by mosquitoes in which they produce infective larvae. Microfilariae do NOT cause symptoms</p>	Thick blood smears taken at night show microfilariae	<p>Diethylcarbamazine effective vs. microfilariae.</p> <p>No tx vs. adults</p>

Oncocerca volvulus (River Blindness)

<p>Subcutaneous inflammation, pruritis, papules, nodules form in response to adult worm proteins. Microfilariae migrate through tissues, ultimately concentrating in the eyes. Leads to blindness.</p>	Millions infected in Africa, Central America	<p>Female blackfly (THINK BLACK=blindness) deposits infective larvae on skin while biting. Larvae enter wound, migrate to subcutaneous tissue, where they differentiate into adults in dermal nodules. Female produces microfilariae that are ingested when another blackfly bites. Microfilariae develop into infective larvae to complete the cycle.</p>	Microfilariae in tissue biopsy	<p>Ivermectin vs. microfilariae.</p> <p>Suramin vs. adults.</p>
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Loa loa

<p>Loiasis- hypersensitivity rxn results in localized subcutaneous edema. Adult worm may be seen crawling across conjunctiva, usually harmless.</p>	Only tropical central and western Africa	<p>Deer fly (mango fly) deposits infective larvae on skin. Larvae enter wound, wander around body, develop into adults. Females release microfilariae which enter blood during the day (compare to <i>Wuchereria</i>). Deer fly infests microfilariae which differentiate into infective larvae.</p>	Microfilariae in blood smear	Diethylcarbamazine
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Dracunculus medinensis (Guinea fire worm disease)

<p>Dracunculiasis- worm protrudes from skin ulcer, wind up on stick over days.</p>	<p>W.H.O. says just 17 countries have this disease as of 4/3/98. All in Africa. (109 countries <i>Dracunculus free!</i>)</p>	<p>Humans infected by tiny crustaceans (copepods) swallowed in drinking water. Larvae released in small intestine, migrate to body, develop into adults. Adult females ulcerate skin, release larvae which are eaten by copepods.</p>		<p>Wind worm up on a stick over days.</p>
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Toxocara canis (Visceral larva migrans)

<p>Visceral larva migrans- Blindness due to retinal involvement. Fever, hepatomegaly, eosinophilia are common.</p>	Dog is definitive host	<p>Dog sheds <i>T.canis</i> in eggs in soil. Humans eat eggs, hatch in small intestine, larvae migrate to organs (eyes, liver, brain), but are eventually encapsulated and die. Life cycle NOT completed in humans (dead end host.)</p>	<p>Larvae in tissue. Hypergammaglobulinemia, eosinophilia.</p>	Diethylcarbamazine
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*Note: if you're really interested about *Dracunculus*, visit the World Health Organization at <http://www.who.org/>