

## Microbiology Key Point Review

### Helminths I

#### TREMATODES

- Subclass Digenea and called “FLUKES.”
- Unsegmented worms.
- All eggs exit from the host into an aquatic environment.
- Life cycle: Snails are first intermediate host. Snails give off infectious larvae/cercariae that encyst or penetrate humans via skin to complete life cycle. An amphibian may be the second intermediate host and flukes must be ingested for life cycle to be completed.

Disease: Fascioliasis and Fasciolopsiasis

Etiologic Agents:

- Fasciola HEPATICA = LIVER fluke.
- Fasciolopsis buski = INTESTINAL fluke.

Source:

- Eat raw aquatic vegetation (i.e. will have the word “Water” as in water chestnut). The encysted metacercariae are on the aquatic vegetation.

Clinical Manifestations:

- Fasciola HEPATICA = Liver fluke:
  - Acute fascioliasis causes a febrile fasciolitic eosinophilic syndrome → Triad of fever, high eosinophilia, and painful liver enlargement.
  - Chronic fascioliasis occurs when adult worms lodge in bile ducts → Obstructive biliary or inflammatory nature.
- Fasciolopsis buski = INTESTINAL fluke:
  - Adult worms attach to mucosa of duodenum or jejunum → Ulceration and inflammatory changes → Severe pain and hunger before meals and intermittent diarrhea.

Pathology:

- Fasciola HEPATICA = Liver fluke:
  - Young fluke forms burrows in liver during migration = Acute phase.
  - Adult fluke lodges in biliary channels → Hyperplasia of ductal epithelium and periportal fibrosis = Chronic phase.

Laboratory Diagnosis:

- Fasciola HEPATICA = Liver fluke:
  - Definitive diagnosis = Eggs in feces (but...keep in mind, will be symptomatic for 6-8 weeks before eggs are in feces).
  - Suggestive diagnosis = Triad of symptoms (i.e. fever, high eosinophilia, painful liver enlargement). + History of eating aquatic plant + alternation of liver laboratory values.
- Fasciolopsis buski = INTESTINAL fluke:
  - Definitive diagnosis = Eggs in feces (keep in mind...a large number of eggs will appear in feces prior to patient becoming symptomatic).

Epidemiology:

- Fasciola HEPATICA = Liver fluke:
  - Areas where herbivorous animals are raised and watercress is eaten raw.
- Fasciolopsis buski = INTESTINAL fluke:
  - Areas where water caltrops and other water plants are eaten.

Treatment:

- Fasciola HEPATICA = Liver fluke:
  - Bithionol and Triclabendazole.
- Fasciolopsis buski = INTESTINAL fluke:
  - Praziquantel.

Disease: Paragonimiasis.

Etiologic agent: Paragonimus westermani.

Source: Metacercariae from crustaceans. Clues: Raw, undercooked, wine soaked crabs or crayfish.

Clinical manifestations:

- Acute symptoms: Early migration of worms → **Diarrhea, abdominal pain, fever, chills**, chest pain, cough.
- Chronic symptoms: Cough, chest pain, **eosinophilia, hemoptysis**.
- CNS + Brain may also be affected.

Pathology:

- **Fibrous cyst** surrounds worms and cyst is filled with parasite eggs, necrotic material, **Charcot-Leyden crystals**.
- Lung tissue and Brain: Granulomas.
- Abdominal wall, liver, genitals, skin: Abscesses.

Laboratory diagnosis:

- Eggs in sputum, stools, pulmonary exudates.
- Skin antigen test will be positive.
- ELISA is most sensitive and specific.

Epidemiology:

- Worldwide.
- Greatest in Asian populations b/c eat raw/partially cooked crabs and crayfish.

Treatment:

- Praziquantel, Bithionol, Niclofolan.
- Recheck patients in 6 months.

Diseases: Schistosomiasis, Bilharziasis.

Etiologic agents: BLOOD flukes!!!

Source: Schistosome cercariae live in fresh water → Penetrate exposed skin → Excyst in stomach → Invade venous system and mate and lay eggs → Potential to spread to lung, heart, other organs. Note: Infected SNAILS shed cercariae.

Clinical manifestations:

- Penetration of skin: Itching.
- Acute phase: Fever, blood eosinophilia.
- S. haematobium: HEMATURIA and DYSURIA.
- S. somiasis: Portal hypertension.

Pathology:

- Granulomas and fibrosis form around eggs: Liver, intestine, bladder, other tissues.
- Fibrosis around portal veins → Portal HTN.
- Fibrosis around ureter or ureterovesical junction → Obstructive uropathy.

Laboratory diagnosis:

- Eggs in stool, urine, tissue.

Epidemiology:

- Humans are infected during exposure to infected snails in water.

Treatment:

- *S. mansoni* = Oxamniquine.
- *S. haematobium* = Metrifonate.
- PRAZIQUANTEL is effective against ALL schistosome species.

## CESTODES

- Called “TAPEWORMS.”
- Live in digestive tract of hosts.
- Almost all rely on intermediate hosts for transmission.
- Humans are usually the host of the adult tapeworm while other mammals are intermediate hosts.

Diseases:

- Taeniasis: BEEF tapeworm and PORK tapeworm.
- Cysticercosis: BEEF, PORK, HUMAN, human neurocysticercosis, cerebral cysticercosis.

Etiologic agents:

- Adult is called TAPEWORM.
- Larval is called CYSTICERCUS.
- *Taenia solium* = Human → Pig lifecycle. Human → Human lifecycle.
- *Taenia saginata* = Human → Cattle lifecycle.

Source:

- Taeniasis: Human is DEFINITIVE host of adult tapeworm. Humans eat raw/undercooked PORK or BEEF containing cysticerci.
- Cysticercosis: Cattle, pigs, humans are INTERMEDIATE hosts. They ingest eggs in feces → Cysticercosis is the larval stage in these intermediate hosts.

Clinical Manifestations:

- Taeniasis: Asymptomatic.
- Human cysticercosis: May present similar to a brain tumor (i.e. seizures, hydrocephalus, meningitis, arachnoiditis, HA, N and V, visual disturbances, confusion, dizziness, ataxia)...SUM: NEUROLOGICAL SYMPTOMS.

Pathology:

- Taeniasis: Tapeworms cause inflammatory response in intestines.
- Cysticercosis:
  - Live = MINIMAL SYMPTOMS.
  - Dying/Dead = INFLAMMATORY RESPONSE = Eosinophils and lymphocytes and calcification.

Diagnosis:

- Taeniasis: Egg, proglottid, or scolex in stool (use proglottid for species differentiation).
- Human cysticercosis: CT to dx neurocysticercosis.

Treatment:

- Taeniasis: Niclosamide or praziquantel.
- Human cysticercosis: Supportive treatment, surgery, chemotherapy with praziquantel.

Disease: Diphyllbothriasis (aka. "FISH TAPEWORM).

Source: Fish are second intermediate or paratenic hosts of Diphyllbothrium species.

Acquire by eating raw or undercooked flesh, hard roe, or liver.

Clinical manifestations:

- Most cases are asymptomatic or subclinical.
- Symptoms: Fatigue, weakness, diarrhea, numbness of extremities, hunger sensation, salt craving.
- Clue to infection with *D. latum* = PERNICIOUS ANEMIA.

Pathology:

- Mechanical irritation of intestinal mucosa and competition for nutrients. → Malabsorption.
- *D. latum* prevents V B12 from combining with intrinsic factor → V B12 absorption is blocked → B 12 Deficiency.

Laboratory diagnosis:

- Eggs or proglottids in feces.

Epidemiology:

- Clue: Consumption of raw fish or fish products.

Treatment:

- Niclosamide, praziquantel, bithionol.

Disease: Echinococcosis = Hydatid Disease.

Etiologic agents:

- *E. granulosus* = Cystic hydatid disease.
- *E. multilocularis* = Alveolar hydatid disease.
- *E. vogeli* = Polycystic hydatid disease.

Source:

- Ingestion of eggs directly from dogs or in foods and fomites contaminated with feces of infected definitive hosts.

Clinical manifestations:

- Slow growing cysts → Symptoms vary according to size, number, and location of cysts.

Epidemiology:

- *E. granulosus* occurs in dog-sheep cycles.
- *E. multilocularis* occurs in fox-rodent cycles.
- *E. vogeli* occurs in dog-rodent cycles.

Treatment:

- Surgical removal of cysts.

## Helminths II

### NEMATODES

- Nonsegmented round worms.
- Separate sexes.
- Route of entry into a host varies.
- Complete gut (unlike trematodes and cestodes).

Disease: Enterobiasis.

Etiologic agent: *Enterobius vermicularis* (PINWORM).

Source: Humans.

Clinical Manifestations:

- Anal itching, eczematous dermatitis around anus, insomnia, enuresis, GI upset, GU infections.

Pathology:

- Humans ingest eggs → Larvae develop in small intestine → Migrate to large intestine → Develop in colon → At night, female migrates to perianal regions, deposits eggs and then migrates back inside.
- The above helps to understand the pathology:
  - Mild inflammation/ulceration of colon mucosa.
  - Intense irritation of perianal skin.
  - Granuomas at ectopic sites.

Laboratory diagnosis:

- See eggs.
- See female pinworm (threadlike in perianal area).
- Scotch tape test is used to diagnosis infection.

Treatment:

- Pyrantel pamoate or mebendazole.

Control:

- Personal hygiene because people self inoculate.

Disease: Ascariasis (ROUNDWORM).

Etiologic agent: *Ascaris lumbricoides*.

Source: Human: Eggs pass out in feces → Develop in soil → Humans ingest food contaminated with eggs.

Clinical Manifestations:

- Lung: Cough, SOB (dyspnea), wheezing, rales.
- GI: Symptoms of obstruction.

Pathology:

- Humans ingest eggs → Larvae hatch in small intestine → Blood stream → Migrate to liver and undergo larvae development → Migrate to lung and larvae coughed up and swallowed → In the small intestine develop into adults → Eggs passed out through feces.

- The above helps to understand the pathology:
  - Larvae migration: Eosinophilic infiltration and granuloma formation around pathos of migrating larvae. Migration also results in obstruction (e.g. bile ducts, small intestine).
  - Adults: Minimal change in intestinal mucosa.

Laboratory diagnosis:

- Egg or adult worms.

Epidemiology:

- Highest in children.

Treatment:

- Mebendazole, pyrantel pamoate, piperazine citrate.

Disease: Trichuriasis (WHIPWORM).

Etiologic agent: *Trichuris trichura*.

Source: Human. Ingest eggs in feces contaminated soil → Larvae hatch in small intestine → Migrate and molt 4x and go to colon → Adults live and mate in colon → Eggs passed out in feces.

Clinical manifestations:

- Mostly asymptomatic.
- Since colonize GI tract...if symptomatic symptoms include abdominal pain, bloody diarrhea, and weight loss.
- Appendicitis if worms fill lumen.
- Rectal prolapse in children secondary to irritation and straining during defecation.
- Anemia and eosinophilia in severe infections because worms suck blood from the intestine.

Pathology:

- Worm sucks blood in the intestinal mucosa.

Treatment:

- Mebendazole.

Disease: Hookworm (Ancylostomiasis, Necatoriasis).

Etiologic agents: *Ancylostoma duodenale*, *Necator americanus*.

Source: Humans, cats. Eggs passed into fecal material → **Eggs develop in the environment** → Larvae get on plants and molt → Humans come in contact with plants → **Larvae penetrate unbroken skin** → Larvae migrate in the blood stream to heart and lung → Migrate up trachea and are swallowed → Adults mature and mate in small intestine → Eggs passed in feces.

Clinical Manifestations:

- **Hypochromic microcytic anemia of iron deficiency because worms “suck blood” and get iron from the blood.**
- Acute: Maculopapular rash and pruritis at site of penetration. Cough, rales, eosinophilia because of worms in lung. Epigastric pain, N and V, and diarrhea because of worms in GI tract.
- Know that there may be a chronic infection.

Pathology:

- Lung: Hemorrhages.

- GI: Worms attach to mucosa → Cytolysis of mucosa → **“Suck blood.”**

Treatment:

- Mebendazole, pyranil pamoate, bephenium hydroxynaphthoate, tetrachlorethylene.

Disease: Strongyloidiasis.

Etiologic agent: *Strongyloides stercoralis*, *Strongyloides fulleborni*.

Source: Soil contaminated with feces of infected humans, monkeys, or dogs.

Clinical manifestations:

- Asymptomatic.
- Abdominal pain from larvae and adults in small intestine.
- The disease may be lethal because of capacity for autoinoculation (i.e. larvae in the stomach develop and directly penetrate the intestinal wall). In immunocompromised hosts this may be lethal → Result in pulmonary hemorrhages, multiorgan failure, ARDS, sepsis, and death.

Pathology:

- Adults in small intestine → Inflammation.
- Migrating larvae → Inflammation, granulomas, or hemorrhages in an organ.

Diagnosis:

- Larvae in feces or other tissues/fluids.

Treatment:

- Thiabendazole.

NOTE: ALL OF THE ABOVE NEMATODES WERE INTESTINAL NEMATODES.

Disease: Trichinellosis.

Etiologic agent: *Trichinella spiralis*.

Source: Eat raw/undercooked swine and other meat containing *T. spiralis* larvae →

Larvae mature in small intestine → Larvae enter lymph system and go to heart, CNS, and skeletal muscle cells. Larvae mature and encyst in the muscle tissue.

Clinical Manifestations:

- Intestinal phase: Abdominal pain, diarrhea.
- Muscular phase: Fever, myalgia, malaise, periorbital edema.
- **Severity of symptoms depend on number of larvae ingested.**

Pathology:

- Enteritis, vasculitis, hemorrhage in eyes, brain, heart, and lungs, muscle fiber necrosis, eosinophilia.

Laboratory diagnosis:

- History of exposure and clinical symptoms.
- Definitive diagnosis by muscle biopsy.

Treatment:

- Mebendazole, thiabendazole, levamisole, or pyrantel.
- Corticosteroids for muscle inflammation.

Disease: Filariasis.

Etiologic agents: See lecture sheet.

Source: Human to human transmission via a mosquito bite (i.e. infected larvae get into humans) → Larvae enter and mature in the lymph system → Adults and microfilariae (offspring) travel to blood and are active in the peripheral blood at night.

Clinical Manifestations:

- Destruction of lymphatics in lower extremities → Elephantiasis.
- Involvement of lymph system: Lymphangitis, lymphadema, lymphadenopathy.
- Skin: Pruritus, dermatitis, subcutaneous nodules.
- Also think of it as in severity of infection:
  - Small infections: Enlarged lymph nodes.
  - Frequent infections: Acute febrile episodes, headaches, swollen lymph nodes.
  - Repeated infections: Fibrous tissue forms around dead filariae → Plugs up lymphatic system → Swelling of legs and genitals → Thick, scaly skin covers chronically swollen/edematous areas → Disfigurement is called elephantiasis because legs resemble elephant legs.

Diagnosis: Identification of microfilaria in peripheral blood at night (**NOCTURNAL PERIODICITY**).

Disease: Loa-Loa.

Etiologic agent: Loiasis.

Source: **Fly is intermediate host.** Fly feeds on human blood and injects larvae into human bloodstream → Adults live in subcutaneous tissues → Male and female adults mate and produce microfilaria → Microfilaria enter blood stream and are active in the day and quiet at night → Fly picks up microfilaria during a blood meal.

Clinical Manifestations: **Eye infection** → Corneal inflammation (keratitis).

Disease: Onchocerciasis.

Source: **Black fly.**

Etiologic agent: Onchocerca volvulus.

Clinical Manifestations: **River blindness.** Destruction of lymphatic tissue with severe infection.

Disease: Dracunculiasis.

Etiologic agent: Dracunculus medinensis.

Source: Drink water from freshwater ponds or wells containing infected copepods → Larvae released in small intestine → Larvae penetrate small intestine walls and migrate to abdominal wall to live in muscle tissue and subcutaneous tissues and develop into adults → Males and females mate and release larvae → When humans are standing in water, the warm water serves as a trigger and the larvae migrate out of the subcutaneous tissue into the surrounding water.

Clinical Manifestations:

- Blister with long, white worm emerging from skin.
- Emergence of worm causes: Arthritis, arthralgias, abscesses, cellulitis, and tetanus.

Treatment:

- Using a match stick, keep winding worm around stick until it is fully removed (will take a long time).