

**Lecture: Introduction to Medical Helminthology. Pathogenic species of Phylum *Platyhelminthes***

1. Introduction to Medical Helminthology
2. General characteristic of Phylum *Platyhelminthes*
3. Class Flukes (*Trematoda*)
4. Class Tapeworms (*Cestoidea*)

**Medical Helminthology** studies biological features and geographic distribution of parasitic worms (helminthes), the course of helminthic invasions, diagnosis, prophylaxis and control of helminthic diseases.

Helminths are invertebrates that develop through egg, larval (juvenile), and adult stages. The definitive classification is based on the external and internal morphology of egg, larval, and adult stages. The main phyla which contain vermiform parasites are **Platyhelminthes** (flatworms) and **Nemathelminthes** (roundworms). Flatworms include tapeworms (Cestodes) and flukes (Trematodes).

Parasitic diseases may affect all the world population. Helminthic parasites of humans are cosmopolitans, widely distributed in the world with most species generally being more prevalent in tropical climates. A table below lists some important helminthes and their general geographical distributions.

***Distribution and prevalence of some parasitic helminths***

Species	Distribution	No. infected
<u>Flatworms (flukes)</u> Blood flukes: <i>Schistosoma mansoni</i> <i>S. haematobium</i> <i>S. japonicum</i>	Caribbean, South America, Africa Middle East, Africa, Asia Asia, Far East	250 000 000
<i>Paragonimus westermani</i> (lung fluke)	Asia	22 000 000
<u>Flatworms (tapeworms)</u> <i>Diphyllobothrium latum</i> (broad fish tapeworm) <i>Taenia solium</i> (pork tapeworm)	North America, South America, Europe, Central Africa, foci in Asia North America, South America, Europe	(?) (?)
<u>Roundworms</u> <i>Ascaris lumbricoides</i> (maw worm) <i>Enterobius vermicularis</i> (pinworm)	World-wide World-wide	800 000 000 500 000 000
<i>Wuchereria bancrofti</i>  <i>Brugia malayi</i>	South America, Africa, Asia, Pacific Islands  Asia	  250 000 000

The most characteristic feature of parasitic worms is that they may reside in humans for years, in many instances for decades, actively producing eggs or larvae. Almost all species can not complete their life cycles in human host and require one or two obligatory intermediate hosts or specific conditions of environment. A common name of a disease caused by helminths is helminthosis.

Depending on the features of their life cycle, the helminthic parasites can be classified into three groups:

- **biohelminths** which develop with alternation of hosts. As a rule, in this case the factors of pathogen transmission include animate entities (such as crustaceans, insects) of food products of animal origin.

Examples of biohelminthes are liver fluke *Fasciola hepatica*, beef tapeworm *Taeniarrhynchus saginata*, round worm *Trichinella spiralis*.

- **geohelminths** which develop without alternation of hosts and part of their life cycle passes in soil. In geohelminthiasis, factors of transmission are objects of inanimate nature, such as water or soil, but also fruit and vegetables raised in soil and contaminated with invasive eggs. Examples are maw worm *Ascaris lumbricoides*, dog heart worms *Toxocara spp.*
- **contact helminths**, in which all stages of parasite development pass in human organism. Direct contact with patient and host may occur by physical contact (through handgrip) or indirectly by contaminated objects (through pencils, toys etc). Examples are dwarf tapeworm *Himenolepis nana*, pinworm *Enterobius vermicularis*.

Helminthic parasites can enter into human body by different modes. In **active penetration** of helminths into human body, larvae penetrate host skin in soil contact (i.e., acquired by walking barefoot on contaminated soil). In **passive penetration**, parasitic invasion is acquired by ingestion of poorly cooked meat, which contains encysted larvae, or egg-contaminated water or food supplies. The particular parasites in certain regions of world are transmitted to human by insect vector, e.g., filarial worms by mosquitoes of genera *Culex*, *Aedes*, *Anopheles* (**vector transmission**).

### **GENERAL CHARACTERISTIC OF PHYLUM PLATYHELMINTHES**

**Platyhelminthes** (Greek *Platy* - flat + *helminthes* - worms) are dorso-ventrally flattened, **triploblastic** and **bilaterally symmetrical** eumetazoans with an "organ grade" of body organization. There being no body cavity (**acoelomate**), internal organs lie embedded in a loose mesodermal tissue called **parenchyma**. Absence of body cavity and vascular system indicates that parenchyma must act as an important transport medium, besides its supporting or skeletal function. Flatworms are characterized by having an internally closed (**protonephridial**) excretory system. Most species are *monoecious* and have **hermaphroditic reproductive system**. These animals can reproduce *asexually* and as well as *sexually*. Life cycles are complicated with one or more larval stages and intermediate hosts.

This phylum includes parasites such as the tapeworms and flukes, as well as free-living (i.e., non-parasitic) organisms such as the planarians.

Most of about 10,000 species of flatworms, so far known, are parasitic with important structural and functional adaptations to suit their parasitic mode of life.

### **CLASS FLUKES (TREMATODES)**

Features of digeneatic Trematodes:

- Adult flukes are unsegmented worms flattened dorso-ventrally, and leaf-shaped (adult stage is called *marita*).
- They have 2 suckers. The first one is the prominent *oral sucker*, around the mouth, this has two functions, **a)** to hold an animal to its host and **b)** to assist in feeding. The second sucker is found a little way further down the animal's body and it has only a single function, that of attachment.
- Body is covered by thick, resistant cytoplasmic layer called **tegument**, which helps protect against digestive enzymes in those species that inhabit the gut of larger animals. The tegument is outer syncytial layer and consists of an organic layer of proteins and carbohydrates called the **glycocalyx**. Beneath the basement membrane of tegument is the **musculature** of body wall. It includes an outer layer of circular, a middle layer of longitudinal and inner layer of oblique or diagonal muscle fibers.
- The alimentary canal is incomplete. The anus is absent. The digestive system has only one opening that functions as both mouth and anus.
- The excretory system is well-developed and is called *protonephredia*. Protonephredia comprises a network of tubules within the animal's body tissues. Excretion and osmoregulation are controlled by "*flame cells*" located in *protonephredia* (these are absent in some forms). These flame cells possess long cilia which carry out a beating function. When the cilia beats, it gives the flame cells an appearance of a flickering candle and this is where the cells get their name. Excess water and body wastes enter the flame cells, are pushed into the tubules by the movement of cilia and thrown out of the body from the pore on the surface.

- The sexes of parasites are not separated (*monocious*). Flukes are *hermaphroditic* (having both male and female organs) except blood flukes *Schistosomes*, which are *dieocious* (having separated sexes).
- The flukes are oviparous, they lay operculated eggs. An exception is *Schistosome* eggs, which are not operculated.
- The life-cycles of all parasitic species include the snails as the first intermediate hosts.

Depending on the habitat in the infected host, flukes can be classified as blood flukes, liver flukes, lung flukes and intestinal flukes. Flukes causing most human infections are *Schistosoma* species (blood flukes), *Paragonimus westermani* (lung fluke), and *Clonorchis sinensis* (liver fluke). Other less important flukes are *Fasciola hepatica* and *Opisthorchis felinus* that both are liver flukes and *Metagonimus yokogawai* that is intestinal fluke.

### **FASCIOLA HEPATICA**

*Fasciola hepatica*, also known as **sheep liver fluke**, is a large liver fluke. It causes *fascioliasis* which is a disease of world-wide distribution.

**The life cycle** of *F.hepatica* is with a **definitive mammalian** (sheep or some cattle) host and an **intermediate snail** host. It is further complicated due to occurrence of a series of different larval stages.

Humans become infected via the consumption of water-cress, water chestnuts, or other aquatic plants that are contaminated with parasitic larva **adolescariae** (metacercariae). The larvae excyst in the duodenum, penetrate the intestinal wall and, through the body cavity, reach bile ducts of liver. There they develop and mature into adult worms that can persist 10-15 month in liver before they proceed to the bile ducts to lay their eggs. Each adult, once securely lodged in the liver, can produce up to 25,000 unembryonated eggs per a day. Further development takes place in fresh water. If landed in water, the egg become embryonated and develops a *miracidium* larva. The miracidium invades an aquatic snail *Lymnaea*. Inside the snail, miracidium divides asexually through a single generation of *sporocyst* and two generations of *rediae*, finally to develop into *cercaria*, a larva that is capable of swimming with its large tail.

Cercariae leave the snails and swim until they find aquatic vegetation or other grass to which they can adhere. There they form cysts called **adolescariae** (metacercariae), which is invasive stage of liver fluke to human. When human and sheep eat these plants, they become infected and life cycle is repeated.

**Pathogenicity and symptoms.** The adult worms cause *inflammation, tissue destruction, and obstruction of the biliary fluid*. Clinical manifestations include *headaches, rashes, muscle pain, jaundice, abdominal pain, loss of appetite, anemia, nausea, and vomiting*.

A single mammal host may harbour as many as 200 flukes. Extensive damage of host's liver, causing "**liver rot**" is the common consequence of such heavy infection.

**Laboratory diagnosis** Adult *F. hepatica* is identified from eggs in a stool sample by microscopy.

**Prophylaxis** (preventive measures): liver flukes are killed in the cooking process; humans can avoid fascioliasis by not consuming raw plants containing adolescariae, eating raw shellfish or raw liver. Control measures for *F.hepatica* ideally should also involve removal of flukes in affected livestock, reduction of the intermediate host snail population, and prevention of livestock access to snail-infested pasture.

### **CLONORCHIS SINENSIS**

The *C.sinensis*, also known as **Chinese** or **Oriental liver fluke**, is a widespread parasite of human, dogs and cats (that are definitive hosts) in the southeast of Asia. Disease *clonorchiasis* is widespread in China, Hong Kong, Japan, Korea and Taiwan, currently infecting an estimated 30,000,000 humans. Clonorchiasis also has been reported in non-endemic areas. Related flukes parasitizing European cats (*Opisthorchis felinus*) and dogs (*O. viverrini*) infect humans in the endemic areas.

The adult flukes usually have spindle-shaped body, 10-25 mm by 3-5 mm. Eggs are flask-shaped, measured 29×16 mcm.

In **life cycle**, the 1<sup>st</sup> intermediate host is fresh water snail, the 2<sup>nd</sup> intermediate hosts are many species of freshwater fish, particularly cyprinids (carp) or crayfish.

Human is infected by eating raw or improperly cooked fish which carries *metacercaria* larvae – invasive stage. When the *metacercaria* is digested by definitive host, the larval worm migrates up the bile ducts where it matures into an adult. In human liver, the mature worms produce eggs. The eggs pass in the feces and find their way to fresh water. The egg hatches to produce a *miracidium* larva. The miracidium after a series of developments (*sporocyst* → *redia* → *cercaria*) produces free-swimming *cercaria*. The latter penetrates under the scales of fish, encysts in fish muscle, and forms *metacercaria* infectious to human. *C. sinensis* has a life span of 10-30 years.

The worm causes *irritation of the bile ducts* which become dilated and deviated. Most pathologic manifestations result from *inflammation and obstruction of bile ducts*.

**Symptoms** of clonorchiasis include *nausea, vomiting, abdominal pain, loss of appetite, swollen liver, fatigue, headaches, rashes, muscle pain, and fever*. Heavier infections produce *anemia, liver enlargement, slight jaundice, edema, ascites and diarrhea*. In long-standing infections, such complications as *cholangitis, cholelithiasis, pancreatitis, and cholangiocarcinoma* can develop, which can be fatal if treatment is left too late.

**Laboratory diagnosis** is based on symptoms and the finding the characteristic eggs in the feces or biliary drainage.

**Prophylaxis:** The effective methods are intensive health education of discourage the culinary habits of eating raw or undercooked fish (fish should be cooked well before consumption), improved sanitation (sewage must be treated before disposal), and mass treatment of infected population to reduce transmission.

### **PARAGONIMUS WESTERMANI**

***Paragonimus westermani* or lung fluke** is a thick, fleshy, brown, egg-shaped worm, ~14×7 mm, that infects the lungs of humans after eating an infected raw or undercooked crab or crayfish. The lung fluke is widespread in the Far East and South East Asia.

**The life cycle** includes several species of snails of Genus *Melania* as the first intermediate host and fresh water crayfish or crabs as the second one. Definitive hosts are humans and many species of carnivorous mammals feeding on crabs and crayfish.

Lung flukes are localized the *parenchyma of lungs*. Adult worms produce the eggs, which are passed out in the sputum, are coughed up, swallowed, and passed in the feces. For the next development, the eggs have to reach fresh water where they hatch into *miracidium* larvae. The miracidium infects an aquatic snail and develops further through the stages of *sporocyst, redia, and cercaria*. The larvae emerge from the snails and go on to infect freshwater crabs and crayfish.

Humans become infected by eating raw crayfish or crabs that contain the *metacercariae*. In the duodenum, the cyst wall is dissolved and metacercaria is released. After penetrating the intestinal wall and wandering in the peritoneal cavity, the young flukes pass through the diaphragm to the lungs where they become established. Inside the lungs, the worm covers itself in granulation tissue, forming a capsule with fibrous walls around itself. These capsules can eventually ulcerate and then heal. When the cysts rupture and the eggs of the worm, which are contained inside, are then coughed up. At this point, the life cycle starts over.

**Symptoms:** Infected people may have a *chest pain, fever, chronic, deep, intermittent cough, bronchitis* and *coughing up blood*. The infection often resembles tuberculosis in many respects. In heavy infestations, *lesions may also be found in lungs, liver and brain*.

**Laboratory diagnosis.** Diagnosis is based on history and symptoms. The characteristic eggs are found in sputum. The location in the lungs is ascertained by radiography. Aberrant infections can be identified serologically.

**Prevention:** a) control and eradication of intermediate host, b) shellfish should be thoroughly cooked, in order to kill any larvae, c) never to eat raw freshwater crabs or crayfish. Travelers should be advised to avoid traditional meals containing undercooked freshwater crustaceans, d) health education, e) mass treatment of infected population in endemic areas.

## **BLOOD FLUKES (SCHISTOSOMA SPECIES) AND SCHISTOSOMIASIS**

**Schistosomiasis** (also known as *Bilharziasis*, or *Bilharzia*) is a term generally reserved for disease produced in humans by the 3 species of blood flukes *Schistosoma*: *Schistosoma mansoni* and *S.japonicum*, responsible for intestinal schistosomiasis, and *S haematobium* for urinary tract schistosomiasis. The schistosomiasis affects about 200 million people in 74 countries, and more than 650 million people who live in endemic areas are at risk. The disease is the second most prevalent tropical disease in the world after malaria.

The three species of *Schistosoma* have different geographic distribution.

*S. haematobium* is prevalent in Africa and the Middle East

*S. mansoni* is found in South America, the Caribbean, and Africa

*S. japonicum* is common in the Japan, Philippines, Indonesia, China (in 13 provinces)

Adult worms are 10-20 mm long. Unlike other trematodes, schistosomes have separate sexes (males and females) that are morphologically distinct. A male has an unusual lamelliform shape with marginal folds forming a *gynaecophoric canal* where a slender female worm resides.

*S.mansoni* and *S.japonicum* are found in mesenteric veins. *S. mansoni* is found more often in the veins that drain the large intestine, while *S. japonicum* resides more frequently in the veins that drain the small intestine. *S. haematobium* is in pelvic veins, occurring usually in the *venous plexus* of bladder, but can also be found in the rectal venules.

**Life cycle.** *Schistosoma* requires the use of **two hosts** to complete its life cycle: a definitive vertebrate host, where sexual reproduction occurs, and invertebrate intermediate host (aquatic snail), where asexual reproduction and development of larval stages occurs. Transmission between two hosts is achieved by free-living stages of the parasite: *cercaria* and *miracidium*.

Depending on the *Schistosoma* species, their eggs are shed either in the feces or urine of an infected human. If the eggs get up in water, larva *miracidium* hatches out, directly invades the snails (of the genera *Bulinus*, *Biomphalaria* or *Oncomelania*). *Cercariae* are released from infected snails and start waiting in water. They can survive about 48 hours in favourable conditions. When they sense that **human** skin is near, they quickly swim and attach with suckers. As they enter they transform into *schistosomulae* (another larval stage) and then get the bloodstream.

**Symptoms and pathogenesis:** Symptoms vary with the species and the phase of infection.

In schistosomiasis, the pathogenic mechanism is determined by three factors:

1. **eggs** with sharp spines that traumatize tissues of organs and cause *ulcers, inflammatory processes, toxic influence, and allergization*. The eggs trapped in the tissues produce *granulomatous inflammatory reactions, fibrosis, and obstruction*.
2. **cercaria and schistosomulum (adolescent)**. When cercariae repeatedly penetrate the human skin, the *allergy* takes place. Initial invasion of the skin may cause *itching* and a *rash (swimmer's itch)*, *cercarial dermatitis* due to physical damage to the skin by proteases and other toxic substances secreted by the cercaria. The migration of the adolescents may induce localized *pneumonitis* and *urticaria*.
3. **adult blood flukes**. The mechanical and toxic effects of adults and their metabolites cause main symptoms of the disease. Intestinal symptoms include *abdominal pain* and *diarrhea* (which may be bloody). Urinary symptoms may include *frequent urination, painful urination* (dysuria), and *hematuria* (blood in the urine).

Acute febrile illness of schistosomiasis known as **Katayama's fever** usually occurs 4-8 weeks after the initial infection when the adult worms begin to produce eggs. Its clinical manifestations usually include *high fever, chills, myalgia, headache*, and a *general ill* appearance. An urticarial rash, which may include *giant urticaria*, and *diffuse lymphadenopathy* may be seen. *Cough, rales, and pulmonary infiltrates* may be noted, even in the absence of fever. Gastrointestinal symptoms of *anorexia, abdominal pain, and loose stools* sometimes are observed.

Heavy infection with many parasites may cause *fever, chills, lymph node enlargement*, and *hepatosplenomegaly* (liver and spleen enlargement).

**Laboratory diagnosis** is based on microscopy for finding of the eggs:

- urine sediment for *S. haematobium*
- concentrated stool samples for *S. mansoni, S. japonicum*

**Prophylaxis** of schistosomiasis is based on drug treatment, snail control, improved sanitation and health education.

## TAPEWORMS (CESTODES)

- Adult tapeworms are elongated, segmented, hermaphroditic flatworms that inhabit the small intestinal lumen of definitive host. Larval forms, which are cystic or solid, inhabit extraintestinal tissues.
- There is no alimentary canal; the worms absorb the digested food of its host over its whole surface (absorb food directly across tegument).
- A body of tapeworm has three regions: *scolex* (head), *neck*, and *strobila*. A scolex has *holdfasts organs* (spines, hooks, suckers), glands releasing sticky secretions, or a combination of these structures that a worm uses to attach itself to inner wall of intestine of a final host.
- A strobila is behind the neck. It consists of a row of segments called *proglottids*. The gravid proglottids at the end of worm break off and pass into environment with a host's feces

Clinically important cestodes pathogenic to human are *Tenia solium* (pork tapeworm), *Taeniarhynchus saginata* (beef tapeworm), *Diphyllobothrium latum* (broad fish tapeworm), *Hymenolepis nana* (dwarf tapeworm) and *Echinococcus granulosus* and *E. multilocularis*, which both cause hydatid disease. These cestodes have a worldwide distribution but incidence is higher in developing countries.

## TAENIA SOLIUM (PORK TAPEWORM) AND TAENIARHYNCHUS SAGINATA (BEEF TAPEWORM)

*T. saginata* can be up to 4-6 m long and 12 mm broad; it has a pear-shaped head (scolex) with four suckers but no hooks. It has a long flat body with several hundred proglottids. In mature proglottids, the gravid uterus contains 17-35 lateral branches.

*T. solium* is slightly shorter than *T. saginata*. It has a globular scolex with 4 suckers and a circular row of hooks (*rostellum*) that gives it a solar appearance. There is a neck and it has a long flat body (0.1 meter in length). The gravid proglottids of *T. solium* have uterus of 7-12 branches.

A tapeworm larval cyst (*cysticercus*) is ingested with poorly cooked infected meat; the larva escapes the cyst and passes to small intestine where it attaches to mucosa by the scolex suckers. The proglottids develop as the worm matures in 3 to 4 months. The adult may live in small intestine as long as 25 years if not identified and treated and tapeworms pass gravid proglottids with the feces. The eggs contained in the gravid proglottids are released after disruption of proglottids. *T. saginata* may produce up to 100,000 and *T. solium* may produce 50,000 eggs per a proglottid, respectively. The eggs extruded from the proglottid contaminate and persist on vegetation for several days and are consumed by cattle or pigs in which they hatch and form cysticerci.

**Symptoms and pathogenesis.** Disease caused by adult *Taenia solium* is called *taeniasis*, by adult *Taeniarhynchus saginata* – *taeniarhynchosis*.

The *Taenia* tapeworms attach to the intestinal walls but cause only *mild inflammation* at the site of attachment. As a result, most tapeworm carriers show no symptoms (*asymptomatic*) and usually become aware of the infection only after noticing tapeworm segments in their feces. The segments of beef tapeworm may spontaneously pass through the anus causing a noticeable sensation. Mild gastrointestinal symptoms, such as *nausea* or *abdominal pain*, can occur in infected individuals. In rare cases where the tapeworm segments migrate into the appendix, pancreas, or bile duct, there may be a sudden onset of severe abdominal discomfort.

### Cysticercosis

*T. solium* eggs can also infect humans and cause **cysticercosis** (larval cysts in lung, liver, eye and brain) resulting in blindness and neurological disorders. *Cysticercosis* is a potentially serious complication of taeniasis, *Taenia solium* infection, in which the larvae develop outside intestinal tract.

Human acquires cysticercosis by ingesting food, water, or feces containing eggs of *T. solium*. **Autoinfection** can also occur from contaminated hands or, less commonly, by regurgitating proglottids of an adult tapeworm from the gut into the stomach during a bout of nausea; this may occur spontaneously or in association with antihelminthic therapy.

The incidence of *cerebral cysticercosis* (also *neurocysticercosis*) can be as high 1 per 1000 population and may account for up to 20% of neurological case in some countries (e.g., Mexico); cysticercosis *ocular involvement* occurs in about 2.5% of patients and *muscular involvement* is as high as 10% (India).

### **Laboratory diagnosis:**

In taeniasis: recovery of proglottids or eggs from stool or perianal area.

Cysticercosis is confirmed by the presence of antibodies (immunology), ultrasonography, computed tomography scanning.

**Preventive control** can be very successful. It employs health education concerning raw pork and beef, meat inspection, sanitation and hygiene on pig and cattle farms, and proper sewage treatment and disposal. It is necessary to reveal and to treat sick humans.

### **DIPHYLLOBOTHRIUM LATUM, THE BROAD FISH TAPEWORM**

*Diphyllobothrium latum* causes *diphyllobothriasis*, also known as fish tapeworm disease or broad tapeworm disease. *D. latum* is the largest parasite of humans, reaching length up to 10 m and consisting of a chain of 3,000 to 4,000 segments, each up to 2 cm wide. An adult worm is characterized by a scolex with a pair of linear sucking grooves (*bothria*) instead of suckers and hooks, and by having a rosette-shaped uterus connected to the outside by a uterine pore through which the eggs are passed.

**The life cycle** of *D. latum* includes definitive host (fish-eating mammals and a human) and two intermediate hosts. The first intermediate host is a crustacean, *water flea (Cyclops)*, the second ones are many species of *freshwater fish*. Final larval stage of *D. latum* invasive for a human is **plerocercoid**.

Human and other animals are infected by eating raw or uncooked fish that contains *plerocercoid* larvae (15x2 mm) which attach to small intestinal wall and mature into adult worms in 3-5 weeks. Eggs discharged from gravid proglottids in the small intestine are passed in the feces. The egg hatches in fresh water to produce a ciliated *coracidium* larva, which needs to be ingested by a water flea *Cyclops*, where it develops into a *procercooid* larva. When infected *Cyclops* are ingested by freshwater fish, the *procercooid* larva penetrates the intestinal wall and develops into a *plerocercoid larva*, infectious to human.

**Clinical symptoms** may be mild, depending on the number of worms. They include *abdominal discomfort, loss of weight, loss of appetite and some malnutrition*. *Tapeworm pernicious anaemia* and neurological problems associated with vitamin B<sub>12</sub> deficiency are seen in heavily infected individuals.

**Laboratory diagnosis** is based on finding many typical eggs and empty proglottids in feces.

**Prophylaxis:** To reveal and to treat sick humans. Not to eat insufficiently cooked fish. Prevention is achieved by freezing. Plerocercoids in fish are quickly killed by thorough cooking, freezing fish at -18°C or lower for 1 day, or thorough pickling. Not to feed domestic animals (cats, dogs) the raw fish.

### **HYMENOLEPIS NANA (DWARF TAPEWORM)**

*Hymenolepis nana* is causative agent of *hymenolepiasis*. Dwarf tapeworm is the most common cestode in human, mainly in children. In many tropical countries it is more common than either *T. saginata* or *T. solium*.

*Hymenolepis* adult worms are 20-40 mm long, have minute segments that are wider than long, a four-sucker scolex with a retractable spined anterior rostellum, and terminal gravid segments that break up and release their egg load after they are caught up in the fecal bolus.

**Life cycle.** *Hymenolepis* is a *contact helminth*. Human is only host, rodents are the reservoir. Invasion is commonly direct (handborn invasion), egg is invasive stage. The ingested eggs hatch in the duodenum, and the *oncosphere* embryos penetrate only into the villi. There, each *oncosphere* forms a *cysticercoid* larva that emerges into the gut lumen as a young worm.

**Clinical symptoms.** Light infections produce *vague abdominal disturbances* but heavier infections may cause *enteritis* and *malabsorption*. Manifestations include *anorexia, nausea, vomiting, abdominal pain, diarrhea*, and also *headache* may occur. External **autoreinfection** is possible because eggs are added to the mouth by contaminated fingers.

**Laboratory diagnosis** is based on finding eggs in the feces. Hygiene is the best **control**.

## **ECHINOCOCCUS GRANULOSUS (DOG TAPEWORM)**

*Echinococcus granulosus* is the causative agent of **cystic hydatid disease**, or *hydatidosis*, which is recognized as one of the major **zoonoses**. The worm is common in Asia, Australia, Eastern Africa, southern Spain, southern parts of South America and northern parts of North America, particularly in areas where sheep farming is prevalent.

This is the smallest of all tapeworms (3 to 9 mm long) with only 3-4 proglottids.

**Life cycle.** The adult worm lives in small intestine of domestic and wild carnivorous canids (dogs). Eggs, passed by infected animals, are ingested by grazing farm animals or humans, who have close contact with fecal-contaminated dog fur. Human is a dead end host. Within the sheep, cattle, or human intermediate host, the eggs hatch, the oncospheres penetrate small intestine, enter the circulation and form cysts in internal organs, mainly liver, lung, bones, and sometimes, brain.

A cyst is round and measures 1-7 cm in diameter, although it may grow to be 30 cm. The cyst consists of an outer anuclear hyaline cuticula and an inner nucleated germinal layer containing clear yellow fluid. This cyst crowds adjacent normal tissue and impairs the tissue's ability to function. If the cyst ruptures, the fluid within the cyst can cause **anaphylactic shock**.

**Symptoms.** Hydatids are slow-growing and infections may not be symptomatic for years. The symptoms, comparable to those of a slowly growing tumor, depend upon the location of the cyst and its size. Large abdominal cysts produce increasing discomfort. Liver cysts cause obstructive jaundice. Peribronchial cysts may produce pulmonary abscesses. Brain cysts produce intracranial pressure and epilepsy-like symptoms. Kidney cysts cause renal dysfunction.

Surgery is the only effective treatment for hydatidosis.

**Laboratory diagnosis** includes immunodiagnosis, ultrasound imaging (US), magnetic resonance (MR), and computed tomography (CT).

**Effective control** includes prevention of dog infection, veterinary control and regular deworming of utility dogs and shepherd's dogs, protection of foodstuffs from contamination with tapeworm eggs, prevention of contact of children with possibly infected dogs, and widespread education on the danger and method of spread of hydatid disease.