General surgery department of SGMU
Lecturer –ass. Khilgiyaev R.H.

Echinococcosis

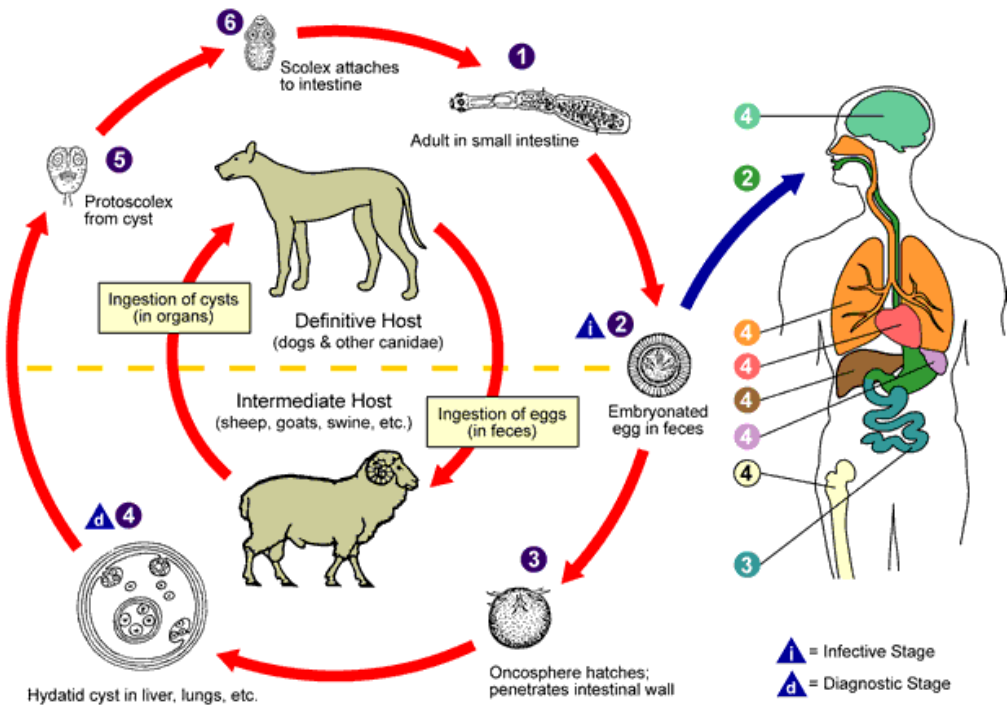
Cycle of ***Echinococcus***

- In the normal life cycle of ***Echinococcus*** species, adult tapeworms (3-6 mm long) inhabit the small intestine of carnivorous definitive hosts, such as dogs, coyotes, or wolves, and echinococcal cyst stages occur in herbivorous intermediate hosts, such as sheep, cattle, and goats.

- A number of other suitable intermediate hosts, such as camels, pigs, and horses, are involved in the life cycle in many parts of the world.

- In the typical dog-sheep cycle, tapeworm eggs are passed in the feces of an infected dog and may subsequently be ingested by grazing sheep.

- They hatch into embryos in the intestine, penetrate the intestinal lining, and are then picked up and carried by blood throughout the body to major filtering organs (mainly liver and/or lungs).



Epidemiology

- After the developing embryos localize in a specific organ or site, they transform and develop into larval echinococcal cysts in which numerous tiny tapeworm heads (called protoscolices) are produced via asexual reproduction.
- These protoscolices are infective to dogs that may ingest viscera containing echinococcal cysts (with protoscolices inside), mainly because of the habit in endemic countries of feeding dogs viscera of home-slaughtered sheep or other livestock.
- Protoscolices attach to the dog's intestinal lining and, in approximately 40-50 days, grow and develop into mature adult tapeworms, once again capable of producing infective eggs to be passed to the outside environment with the dog's feces.
- Because humans play the same role of intermediate hosts in the tapeworm life cycle as sheep, humans also become infected by ingesting tapeworm eggs passed from an infected carnivore.
- This occurs most frequently when individuals handle or contact infected dogs or other infected carnivores or inadvertently ingest food or drink contaminated with fecal material containing tapeworm eggs

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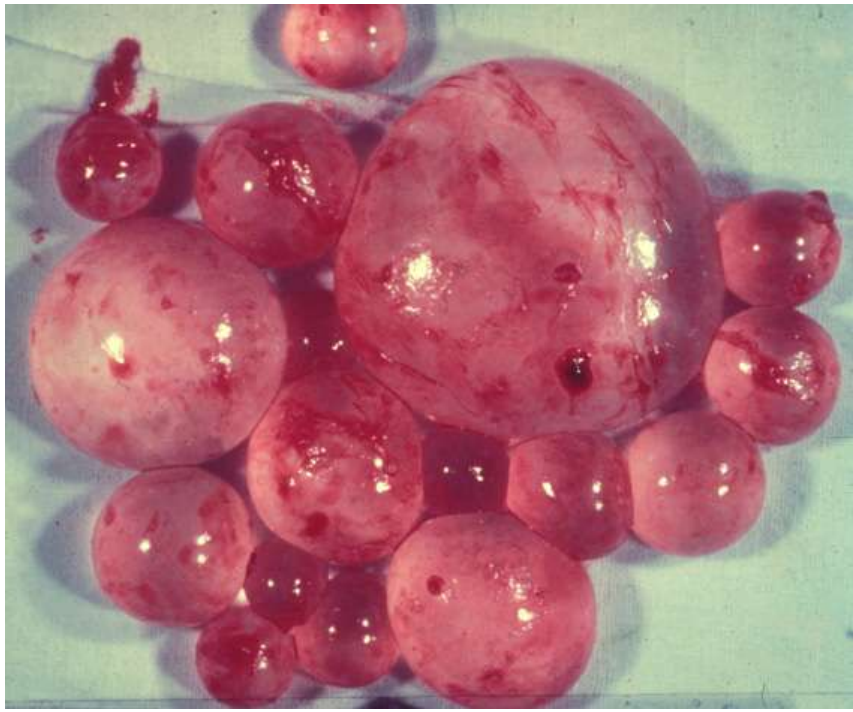
Primary and secondary echinococcosis

• In primary echinococcosis, metacestodes develop from oncospheres after peroral infection with eggs.

• In secondary echinococcosis, larval tissue proliferates after being spread from the primary site of the metacestode.

- This can occur by spontaneous trauma such as induced rupture or during medical interventions.
- In each anatomic site, cysts are surrounded by the periparasitic host tissue (pericyst), which encompasses the endocyst of larval origin.
- Inside the laminated layer, or hyaline membrane, the cyst is covered by a multipotential germinal layer, giving rise to the production of brood capsules and protoscolices.



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The clinical features of echinococcosis

- The spectrum of symptoms depends on the following:
- Involved organs
- Size of cysts and their sites within the affected organ or organs
- Interaction between the expanding cysts and adjacent organ structures, particularly bile ducts and the vascular system of the liver
- Complications caused by rupture of cysts
- Bacterial infection of cysts and spread of protoscolices and larval material into bile ducts or blood vessels
- Immunologic reactions such as asthma, anaphylaxis, or membranous nephropathy secondary to release of antigenic material
- After a variable incubation period, infections may become symptomatic if cysts are growing and exerting pressure on adjacent tissue and inducing other pathologic findings.

symptoms

- Common symptoms are upper abdominal discomfort and pain, poor appetite, and a self-diagnosed mass in the abdomen.
- Physical findings are hepatomegaly, a palpable mass if on the surface of the liver or other organs, and abdominal distention, jaundice, liver abscess.
- If cysts in the lung rupture into the bronchi, intense cough may develop, followed by vomiting of hydatid material and cystic membranes, chest pain, pneumothorax

laboratory tests

- Generally, routine laboratory tests do not show specific results.
- Indirect hemagglutination test and enzyme-linked immunosorbent assay are the most widely used methods for detection of anti-*Echinococcus* antibodies (immunoglobulin G).
- Depending on the test system used and other parameters, approximately 10% of patients with hepatic cysts and 40% with pulmonary cysts do not produce detectable serum IgG antibodies and exhibit false-negative results.
- Children aged 3-15 years may produce minimal serologic reactions.

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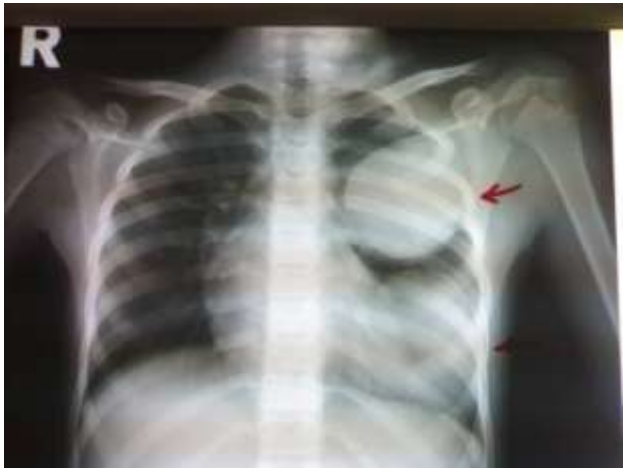
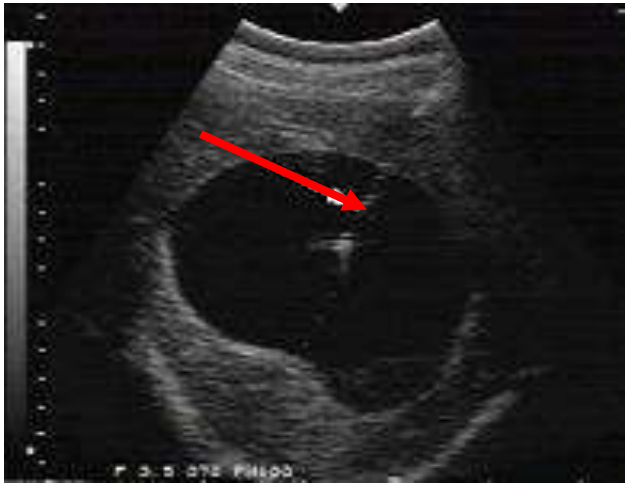
Radiographic, Ultrasonography

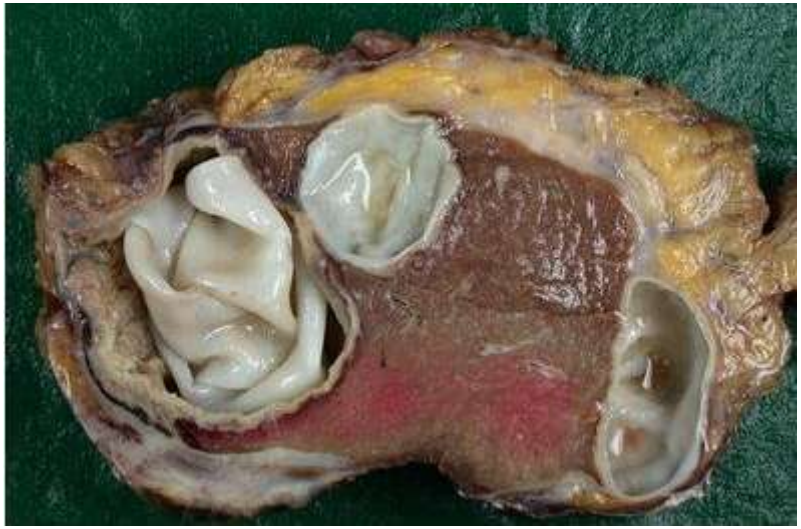
- Radiographic examination is useful for cysts in the lungs, bone, and muscle and for detecting calcified cysts.
- Ultrasonography is the procedure of choice
- CT scanning has the advantage of inspecting any organ (lungs cannot be explored with ultrasonography), detecting smaller cysts when located outside the liver, locating cysts precisely, and sometimes differentiating parasitic from nonparasitic cysts.
- Endoscopic retrograde cholangiopancreatography may be indicated in patients with cholestatic jaundice.





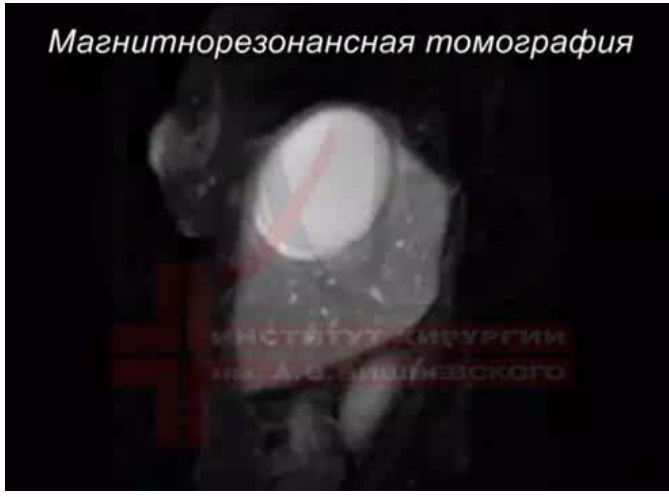
Treatment

- Two benzimidazolic drugs, mebendazole and albendazole, are the only anthelmintics effective against cystic echinococcosis



Surgery

- Surgery was the only treatment available before the introduction of anthelmintic drugs. It is considered the first choice of treatment for echinococcosis
- Usually, radical surgery (total pericystectomy) is indicated for liver cysts. More radical interventions have higher intraoperative risks but less numerous relapses. With the inclusion of chemotherapy prior to or after surgery, less-aggressive surgery may be possible.
- Surgery for pulmonary cysts includes extrusion of cysts, pericystectomy, and lobectomy.
- Peripheral and unilobar echinococcal cysts, regardless of how complicated they are, can also be treated with laparoscopic surgery using partial cystopericystectomy and drainage.



Ascariasis

- Ascariasis is a human infection caused by a round-worm Ascaris lumbrioides and characterized by early pulmonary and later intestinal symptoms.
- Being the only reservoir humans are infected by eating food (e.g. vegetables) contaminated with mature ova.

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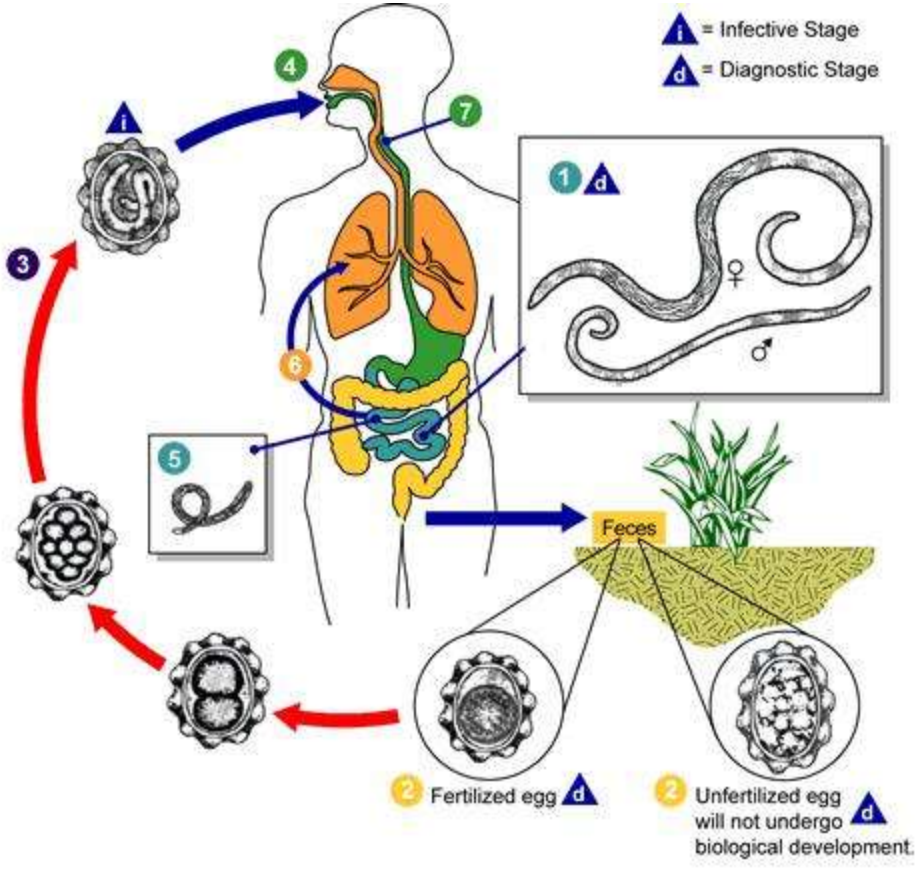
Pathogenesis

- These hatch in the duodenum and the larvae migrate through the wall of the small intestine and are carried by the lymphatic and bloodstream to the lungs.

- Here they pass into alveoli, ascend the respiratory tract and are swallowed.
- They mature in the jejunum, where they remain as adult worms.
- Disease may be caused by both the larval migration through the lung and the presence of the adult worm in the intestine.



diagnosis

- Infection with the adult worm is usually diagnosed by finding eggs in the stool.
- Occasionally, adult worms are passed in the stool or vomited.
- Larvae are occasionally found in the sputum during the pulmonary phase.

intestinal obstruction

- A tangled mass of worms may cause intestinal obstruction (mostly in children or young adults) with typical colicky pains, “acute abdomen” syndrome, vomiting with intestinal contents and specific X-ray findings; a heavy infestation will compete for nourishment and contribute to malabsorption.

complications of ascariasis

- The complications of ascariasis may require surgery.
- The surgical approach in intestinal obstruction requires that the tangled mass of the worms should be descended to the large intestine during laparotomy with subsequent medical therapy; enterotomy and removal of the helminth may occasionally be indicated.

appendicitis

- Ascaris limbricoides - associated appendicitis lacks specific signs.
- The adult worms may inadvertently be found in a patient operated on for acute appendicitis, in which obstruction of the appendix can be evident.

obstructive jaundice

- Other complications include blockage of the bile or pancreatic duct, which may only rarely be found.
- The maturation of the worms in the intestine can lead to penetration through Vater's ampulla into the common bile duct and the biliary tree.
- This, in turn, results in obstructive jaundice, suppurative cholangitis, acute cholecystitis, hepatic abscess.

clinical picture

- The clinical picture of biliary ascariasis includes the signs of
  - obstructive jaundice,
  - suppurative cholangitis,
  - hepatic abscess
  - acute onset and rapid progression of sepsis characterized by severe toxaemia.

establish the definitive diagnosis

- Endoscopic retrograde pancreatocholangiographia,
- ultrasound scanning,
- CT

Surgical treatment

- The surgical methods include cholecyst- or choledochotomy, parasite extraction with external bile duct drainage.

pancreatitis

- Pancreatic involvement in ascariasis occurs if the worms gain entrance to the main pancreatic duct, which results in chronic indurative or acute pancreatitis.
- The only option is surgery - pancreaticotomy with removal of the worms and postoperative specific antiparasitic drag therapy.

gastrointestinal tract perforation

- Ascaris limbricoides - associated gastrointestinal tract (oesophagus, stomach, intestine) perforation results from ulcerative and necrotic changes of the mucosal membranes, which, in turn, may produce the clinical signs of peritonitis.

Postsurgical complications

- Postsurgical complications of ascariasis are due to active migration of the worms following the operation.
- They may also be vomited with occasional subsequent mechanical asphyxia caused by their penetration into respiratory tract.

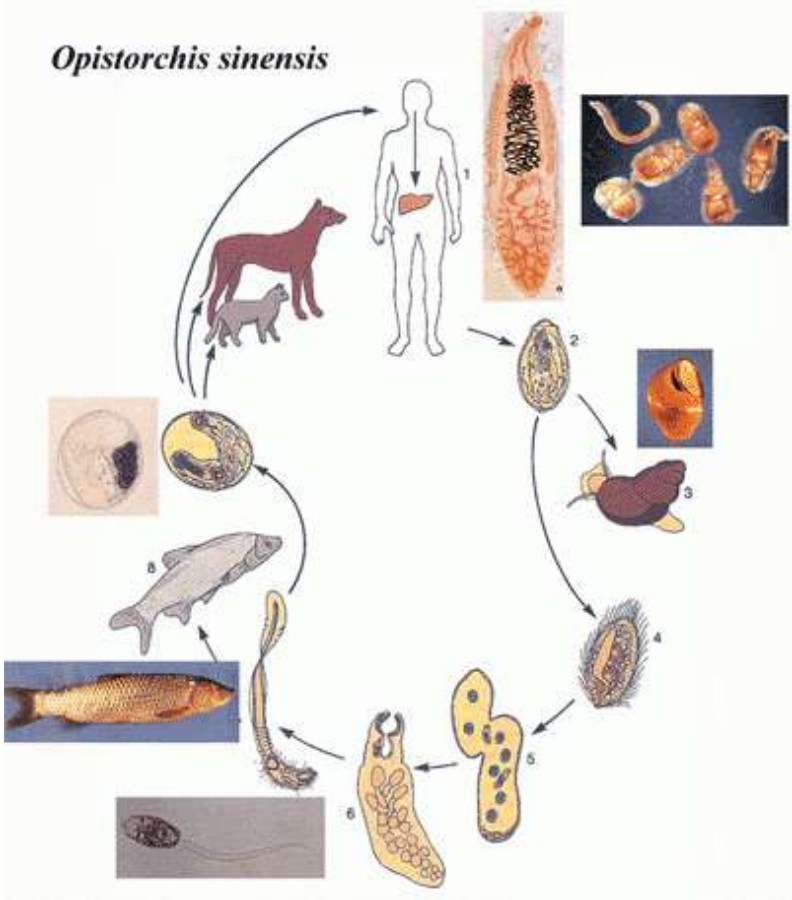
peritonitis

- Surgeries that involve incision ol hollow organs may be complicated by discharging of the worms into the free abdominal cavity through sutures with subsequent suppurative peritonitis.
- Severity of the postsurglcal complications of as-cariasis requires that each elective surgery should be preceded by identification of the ova in faeces.
- Once the diagnosis of ascaris has been confirmed, preoperative drug therapy is mandatory.

Opisthorchiasis

- Opisthorchiasis is an important hepatic and
- pancreatic fluke of humans.
- It is caused by Opisthorhis felineus.

•The other principal mammalian hosts of the disease are dogs, cats, foxes, pigs, while the first and second intermediate hosts are snails and fresh water fish (e. g. the carp and salmon), respectively.



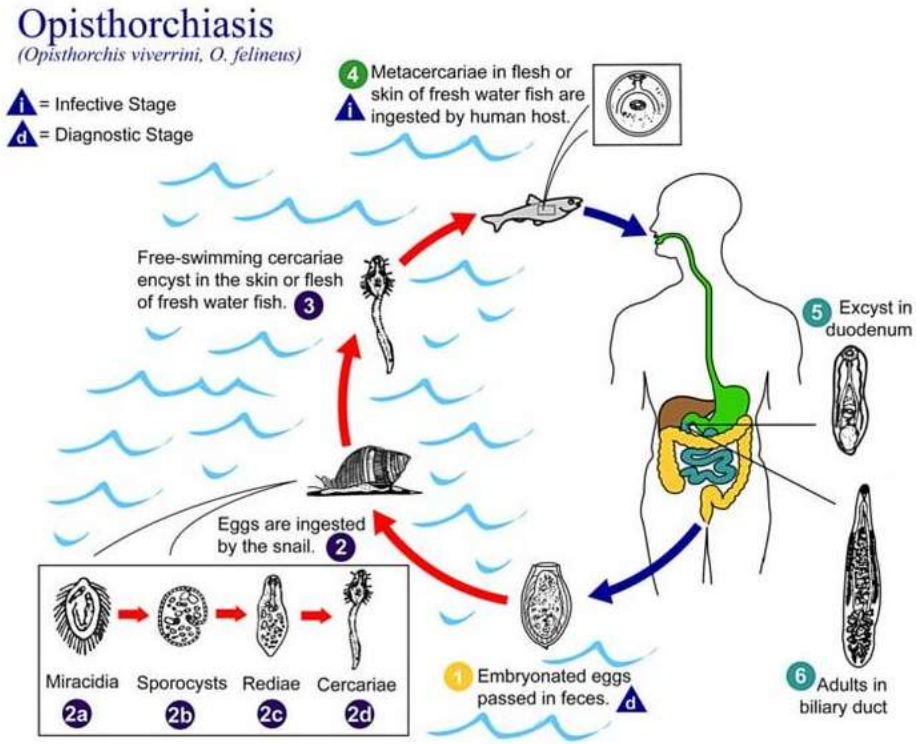
Pathogenesis

- The egg, on reaching fresh water, hatches into a free-swimming miracidium.
- After multiplication and further development within the snail, thousands of free-living cercariae are released and must enter the second intermediate host, where they encyst to form metacercariae.

• Infections follow ingestion of raw, dried, salted, or pickled fish containing the metacercariae.

• The larvae are released in the duodenum, enter the common bile duct, and migrate to the 2 order bile ducts (or, occasionally, the gall bladder and pancreatic ducts), where they mature in about 1 month into adult, flat flukes vaiying from a few mm to several cm in length.

• The mode of spread of the fluke is via ova in faeces or water.



complications

- The parasite may cause hepatic dystrophy and necrobiosis.
- The common surgical complications of opisthorchiasis are suppurative cholangitis with hepatic abscess.
- The perforation of the bile ducts results in peritonitis and hepatic abscess penetration into abdominal or thoracic cavities.
- Often, chronic opisthorchiasis may even cause hepatic carcinoma.
- Opisthorchiasis of the pancreas may be complicated by acute pancreatitis or pancreatic carcinoma.

signs

- The complications of opisthorchiasis lack characteristic signs.
- However, clinical and epidemiological findings often suggest the diagnosis, which can be confirmed only by identification of the eggs in faeces or duodenal contents.

treatment

- The treatment of the complicated opistorchiasis involves conventional methods applied in parasitic diseases coupled with pre- and postoperative medical therapy with hexachlorparaxicol.
- The surgery on the organs other than the liver or bile ducts or pancreas requires that specific therapeutic agents should be given postoperatively