

# Cestodes (Tapeworms)

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## Taeniasis

### Summary

- *Taenia saginata* : infection only via beef with larvae, resulting in an adult intestinal worm
- Infection with *Taenia solium* larvae present in pork results in an adult intestinal worm: vague abdominal symptoms or asymptomatic
- Feco-oral infection via human feces containing *Taenia solium* eggs results in cysticercosis: epilepsy, subcutaneous nodules, nodules located in muscles, etc.
- *Taenia asiatica* : resembles *Taenia saginata*, but is transmitted via pigs. No cysticercosis in humans.

### Life cycle



*Taenia saginata*. The adult cestode can grow to several meters in length. Copyright ITM

Eating insufficiently cooked infected beef (*Taenia saginata*) or pork (*T. solium*) leads to infection with adult tapeworms. Humans are the natural final host and the only carriers of these cestodes, and thus also the only distributors of their eggs. The adult worms live in the small intestine and are several meters long. The pre-patent period is approximately 3 months.

A third species of human *Taenia* has been described in Asia (*Taenia asiatica*). The clinical importance of this has still to be determined. At present insufficient is known about *T. asiatica*. The adult worm is morphologically very similar to *T. saginata*. The life cycle of this cestode is different, however. Unlike *T. saginata*, which causes infections in the skeletal muscles of cattle, *T. asiatica* affects the liver, omentum, serosa and lungs of pigs. At present, *Taenia asiatica* does not seem to cause neurocysticercosis in humans, but more study is needed.

## Clinical aspects

Below, the symptoms present due to infection with an adult worm are described.

Most carriers of adult worms are asymptomatic. The length of an adult worm is usually  $\leq 5$  m for *T. saginata* (however, it may reach up to 25 m) and 2 to 7 m for *T. solium*. Some people present nausea, anorexia or epigastric pain. The loose segments of *T. saginata* (not of *T. solium*) may actively creep outside through the anus, and cause local discomfort. Each segment contains approximately 60,000 eggs. *Taenia* may have a role in malnutrition (5 to 7 cm of worm has to be produced every day, for which food is needed), but only if there are also other reasons for malnutrition. In only 15% of patients peripheral eosinophilia is present. Note that while many humans can carry *T. solium* adult worms without any apparent effect, these people are the only source of eggs. When ingested, these eggs can produce larvae both in the natural host and in humans. The larvae are the cause of cysticercosis in both pig and human. Human-to-human transmission can therefore take place so that cysticercosis can occur in people who do not eat pork or who have no pigs in their surroundings.

## Diagnosis of infection with an adult worm

Finding proglottids in the feces, or a history of motile proglottids crawling out of the anus is important.

Eggs are sometimes found in the stools. The eggs are sticky and easily get onto the peri-anal skin. They can be detected in the peri-anal region with a Scotch tape test. There is no morphological difference between the eggs of *T. saginata* and those of *T. solium*.

Differentiation can be made by the proglottids: a uterus with 10 branches or less in the dangerous *T. solium* and a highly branched uterus (12 or more) in the harmless *T. saginata*. *Taenia* antigens may be found in the feces. Only rarely can the tapeworm's head be discovered. The undamaged scolex of *T. solium* bears two rows of hooks. The scolex of *T. saginata* is hookless. However, dysmorphic tape worms are sometimes encountered.

## Treatment

- Niclosamide (Yomesan®) 4 tablets each of 500 mg will be taken together and chewed well. If the patient should vomit there is a theoretical risk that *T. solium* eggs will pass

back into the stomach, activate and subsequently give rise to cysticercosis.

- Praziquantel (Biltricide®), in a very low dose (5-10 mg/kg), is also very effective. Praziquantel in a higher dose can sometimes provoke complications - such as sudden neurological symptoms - should cysticerci be present in the brain. This complication seems however extremely rare in endemic areas where praziquantel mass treatment is used to control schistosomiasis.

For successful treatment, the scolex must be destroyed and eliminated; a residual scolex can result in regrowth of the entire tapeworm. Some experts recommend purgative treatment to be associated with antihelminthic drugs to have more probability to obtain the scolex in the stool, but this method is far from being universally accepted.

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# Cysticercosis

## Summary

- Infection by *T. solium* eggs followed by development of cysticerci (= larvae) in the body.
- Symptoms depending on localization of the larvae.
- Neurocysticercosis with epilepsy is a common complication.

## General

At the end of the 19th century cysticercosis was still occurring frequently in Europe. At that time cysticercosis was found in 2% of the autopsies in Berlin. Nowadays the disease has virtually disappeared in the West. There are still occasional imported cases. Approximately 50 million people worldwide are estimated to have cysticercosis infection, although subclinical infection may underestimate this number. The disease occurs in regions where pigs are kept and eaten (thus not in Muslim regions). In many poor areas pigs are not kept in a pigsty, but run about in the open. This is encouraged in some areas, so that the animals

function as a kind of free waste-disposal system. These are generally also places where the sanitary facilities are inadequate. The animals can become infected from human faeces via coprophagy. Insufficient meat control is an important risk factor in endemic regions. Not cooking meat through is another risk factor. Cases of cysticercosis in non-endemic regions may sometimes occur via infection from the carriers of adult worms who have come from endemic regions. If these infected migrants are employed in a household they may cause infections in their new surroundings (e.g. Mexican women who go to work in households in the USA).

## Life cycle

When larval *Taenia solium* infect a human they develop into an adult tapeworm. In contrast, if the eggs of *Taenia solium* are swallowed (food or water infected with human feces) the larvae (oncospheres) which emerge from them penetrate the intestinal wall and spread throughout the whole body via the blood stream. Therefore note that cysticercosis is caused by infected human faeces and not directly by eating insufficiently cooked pork. People with cysticercosis do not necessarily have an adult tapeworm. Auto-infection in humans infected with an adult *Taenia solium*, is a possibility, however. In approximately 40% of people with cysticercosis an adult worm is found in the intestinal tract. The larvae migrate to various tissues and within 2 months convert into what are known as bladder worms (cysticerci). The typical bladder worm is a small ellipsoidal bag measuring 5-15 mm surrounded by a white translucent membrane. This bag contains clear fluid and a single round head, the protoscolex. When the cysticerci die off they are absorbed or encapsulated and calcify. Each egg produces 1 cysticercus. Larval multiplication does not occur. In the brain, cysticerci can become extremely large (many centimeters diameter) when they develop in the ventricles (racemose form, see below).

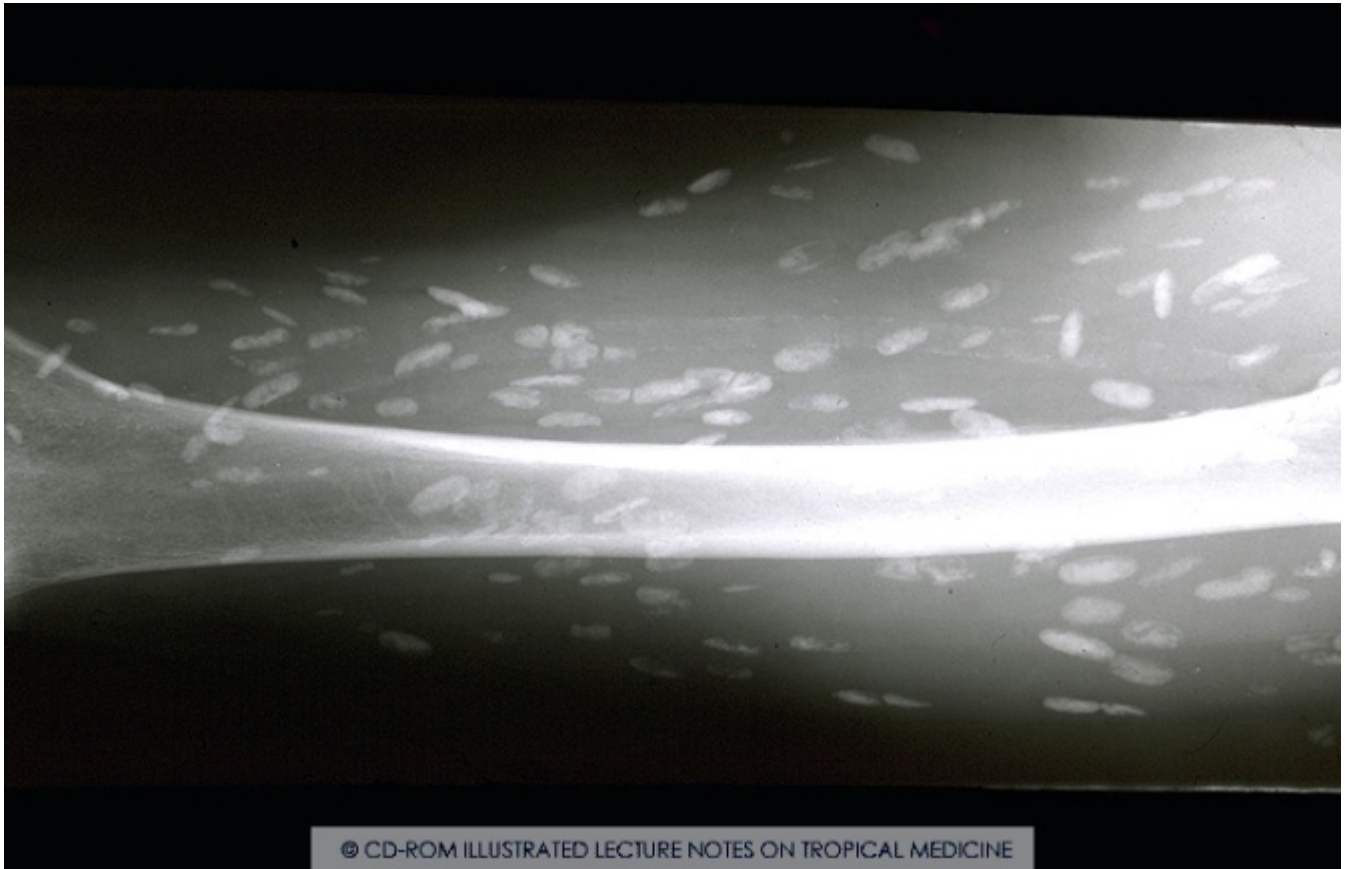
Cysticerci which are present in pork, evaginate normally in the human intestine to then grow to full adult worms. Evagination is also possible, (but rare) in the human eye and intraventricular evagination may occur in the brain. These are sites where no inflammatory capsule is formed around the parasite. Evagination does not occur in the muscles or in the cerebral parenchyma.

## Clinical aspects

Symptoms vary greatly, depending on the location of the cysticerci and the immune response of the individual. Small cysts may be found in the muscles, subcutis, eyes and in other tissues, where they are usually asymptomatic but may cause discomfort when inflamed. They appear as small nodules (5-10 mm). Sometimes they are much larger (e.g. 30 mm or even larger). In neurocysticercosis (NCC) there are parenchymal, subarachnoidal, intraventricular, spinal and ocular forms. Racemose cysticercosis is an aberrant development form of the parasite similar to a bunch of grapes. Live cysticerci in the central nervous system often cause remarkably few symptoms. When the parasites degenerate there may be focal encephalitis and oedema. Sometimes they may trigger a severe inflammatory response within a few days, which can be fatal.

In the brain they often cause late-onset epilepsy (common in Mexico and South America). Adult-onset seizures in endemic regions are therefore highly suggestive of neurocysticercosis. This can manifest itself as focal seizures (Jacksonian epilepsy). This is often followed by postictal confusion or transient paralysis (Todd's paralysis) and/or speech  $\pm$  vision problems. Todd's paralysis usually disappears within 48 hours. Intraventricular cysts may cause obstructive hydrocephalus. According to a recent meta-analysis, the main syndromes caused by NCC by order of frequency are epilepsy (80%), focal deficits (15%), intracranial hypertension (10%) and cognitive declines (5%). Mixed locations (parenchymal + meningeal) are common, but spinal localization is rare. Chronic meningitis, paralysis of cranial nerves, spinal cord lesions and mass effects may occur.

Psychiatric symptoms, encephalitis-like, are possible in case of severe infection. All these neurological problems may be acute or delayed forms. Focal calcifications are detectable 8 months to 10 years after infection. Larvae cannot be regarded as dead, unless the lesion is **completely** calcified.



Radiograph of an arm, showing elongated oval calcifications, typical of cysticercosis (calcified cysts in muscle).



*Taenia solium*, calcified cysticerci in the muscles of the legs. Such lesions are typically oval and elongated when localised in a muscle.

Table: Symptoms of neurocysticercosis

Headache	(23-98%)
Meningism	(29-33%)
Papilloedema	(48-84%)
Convulsions	(37-92%)
Abnormal mental state	(74-80%)
Focal deficits (motor and/or sensory)	(3-36%)
Ataxia	(5-24%)
Myelopathy	(<1%)
Cerebral nerve defect	(1-36%)
Visual disturbances	(5-34%)

## Diagnosis

Diagnosis is made by means of excision of skin nodules or by using serological techniques, including antigen detection. Antibody and antigen detection may be carried out on cerebrospinal fluid as well as serum, but are often negative if only one or two lesions are present. Lesions can be demonstrated by radiology, such as radiography targeting the soft tissues (shoulder and thigh muscles) and X-ray of the skull. In muscles, the calcified cysts tend to be elongated ovals.

MRI scanning of the brain is clearly superior to CT from a diagnostic point of view and scolices can sometimes be visualized through MRI. Living cysticerci are seen on a CT scan as hypodense lesions which are not enhanced by IV contrast. Degenerating cysticerci are

sometimes isodense or hyperdense with an edematous ring-shaped zone which can be enhanced by IV contrast. They may disappear within 3 months. Sometimes the diagnosis can only be made by stereotactic brain biopsy.

The parasite is often surrounded by an inflammatory infiltrate with plasma cells. Immunoglobulins may accumulate in the cytoplasm of reactive plasma cells, and form prominent eosinophilic inclusions (Russell bodies). They are however not specific for cysticercosis.

Serology is available and EITB (enzyme-linked immunoelectrotransfer blot assay) uses affinity-purified glycoprotein antigens fractionated by electrophoresis which performs better than ELISA. Antigenic tests have been also developed, reflecting the presence of viable cysts (if their numbers is sufficient however), in contrast to antibody detection tests reflecting only (past) exposure. Field-adapted lateral flow antigen-based diagnostics are being evaluated.

The most important **differential diagnoses of cerebral ring-shaped lesions:**

- Cerebral toxoplasmosis
- Hydatid cyst
- Ectopic worms (Paragonimus, Schistosoma)
- Amoebiasis
- Tuberculosis and cryptococcosis
- Bacterial abscesses, septic emboli, nocardiosis
- Glioma, lymphoma, metastasis
- Haemangioma

## Treatment

Therapy of neurocysticercosis is extremely complex and depends on the cysticerci stages (viable, degenerating, calcified) and localizations (intra- or extra-parenchymal). It remains a rather controversial area. Expert advice and a multidisciplinary approach are often required. The most important is to treat the symptoms first, mainly the seizures with anti-epileptic

drugs (up to 2 years after the last seizure, like for other epilepsy). Medical treatment, when indicated, is based on administration of praziquantel (50 mg/kg/day) or albendazole (15 mg/kg/day) for 2 weeks, but in case of multiple cysticerci (>3), association of both drugs was found superior in seizure reduction.

In adequately selected patients (see below), antihelminthic therapy may reduce the risk of generalized seizures by 67% as well as of the number of seizures, and lead to complete resolution of the lesions at CT/MRI in a substantial proportion of patients. But in real life, the outcome is rarely this favourable. It is important to note that when the bladder worms die off they cause a local tissue reaction. Neurological symptoms may therefore exacerbate (generally on the 2nd to 4th day of treatment) and can be very difficult to manage where there are no neurosurgical facilities or neuro-imaging. This effect can be mitigated by starting dexamethasone 1 day before the other drugs, maintained up to one month (a dose of 8 mg/day seems optimal), before it's slowly tapered down. Albendazole does not interfere with carbamazepine (Tegretol®) or phenytoin (Diphantoine®). Corticosteroids reduce the blood level of praziquantel and increase albendazole levels, but this is probably of no clinical importance.

Recently the benefit of medical therapy has been questioned. Probably many patients with infections recover spontaneously. When treating neurocysticercosis, it is important to know beforehand whether there are intra-ocular lesions. Degeneration of a cysticercus in the retina, together with accompanying inflammation, may lead to acute blindness. Surgical removal via vitrectomy should be considered, but such a procedure is not without risk.

Today, experts recommend antihelminthic drugs only in case of viable (symptomatic) cysts without inflammation and degenerating cysts with inflammation (to accelerate involution). Granulomatous cysts and those already calcified, for which symptomatic treatment and steroids are sufficient, do not benefit from antihelminics. More and more a segregation is made between intra-parenchymal NCC, where medical therapy may be carefully proposed, except when there are too many lesions (**contra-indicated** because of the high risk of diffuse cerebral oedema), and extra-parenchymal (intraventricular, meningeal, spinal, ocular,...) NCC where neurosurgery is almost always required (due to risk of intracerebral obstruction and hydrocephalus) in combination with longer antihelminthic treatment at higher dose (extraparenchymal cysticerci are usually less susceptible to medication).

Sometimes a ventriculoperitoneal shunt must be inserted in obstructive hydrocephalus. Shunt blockage is common if the cerebrospinal fluid contains large amounts of protein. Appropriate treatment of NCC is nearly unfeasible without adequate diagnostic/surgical facilities, and impossible without an initial careful neuro-imaging assessment. In remote, low-resource settings, it is often preferable to just treat the complications (epilepsy) with a symptomatic antiepileptic therapy, rather than using an etiologic treatment without possibility of monitoring or neurosurgical intervention.

## Prevention

Since humans are the only reservoir for adult *T. solium* the disease can be controlled by improved sanitation and hygiene, in particular by controlling pollution with human faeces. Human carriers should be treated. To reduce the number of carriers of *Taenia solium*, proper statutory meat inspection should be carried out. Furthermore, meat should be heated to above 56°C or stored for at least 10 days at -10°C (requiring a freezer). Eating raw or insufficiently cooked pork should be discouraged. If there is a patient with cysticercosis, it is best to investigate whether the patient, close family members, domestic staff and friends are carriers of adult *T. solium* and constitute a possible source. A faecal examination and an antigen-capture ELISA test are used for screening. If positive, a CT scan of the brain is carried out (detection of cysts in the brain).

Pigs can be treated with a single administration of oxfendazole, a benzimidazole. Vaccination of pigs is under study, and appears to be efficacious under controlled environments. There should be a strong recommendation that pigs not be allowed to run free. They are coprophagic. If humans do not compost their faeces, but use them directly as pig fodder or on the fields, the animals will become infected.

Composting kills the eggs. Washing hands with soap after using the toilet should be encouraged. Parasitic infections in which faeces play a part, are a taboo subject in some communities. A control program needs to take account of this.

There is no vaccine for human protection against *T. solium*. However, vaccines are available to prevent *T. saginata* infection in cattle and *T. ovis* in sheep.

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# Cystic echinococcosis

## Summary

- Infection from eggs in dog feces.
- Larvae form large cysts with internal daughter cysts.
- Cysts in the liver and lungs, rarely in other organs.
- Often asymptomatic, sometimes symptoms due to pressure upon surrounding organs.
- Risk of rupture with anaphylaxis or dissemination.

## General

There are several tape worms in the genus *Echinococcus*: *E. granulosus*, *E. multilocularis*, *E. vogeli*, *E. oligarthus*, *E. shiquicus*. The most important and frequent one is *E. granulosus*, causing cystic echinococcosis or hydatid disease. *Echinococcus granulosus* is a small tapeworm (a few mm long) which infects dogs and other canines. Its distribution is world-wide. In some regions the problem is very important such as North Kenya around Lake Turkana and Kyrgyzstan and the surrounding central Asiatic republics. Various animals (sheep, goats, cattle, pigs) may become infected with the eggs in dog faeces. In the animal's intestine the larva (called "oncosphere") emerges from the egg. It penetrates the intestinal wall and is carried by the venous blood towards the portal vein. After development of the parasite, hydatid cysts are formed in internal organs. The cycle is completed when a dog has the opportunity to eat offal containing hydatid cysts. In the dog's intestine adult *E. granulosus* then develop, after which egg laying can begin. Each hydatid cyst leads to multiple adult worms.

Humans are accidental hosts. If humans take water or food contaminated by dog faeces, they will develop one or more hydatid cysts. The cyst contains fluid and daughter cysts and is known as a hydatid cyst. On the inside of each cyst is a germinal membrane. From this membrane countless protoscolices (small heads) develop. There is thus multiplication at the larval stage. A capsule of connective tissue is formed around the cyst. This capsule consists

of the cyst wall together with the germinal membrane. The majority of cysts are found in the liver and lungs, but other locations are also possible (brain, bones, spleen, kidneys). These are often continuously growing cysts, which may produce pressure on surrounding organs, may rupture or die off and calcify. When the parasite has died and disintegrated the hooks which were situated at the former heads remain in the sandy fluid of the dead cyst, and these can be seen under a microscope. This is useful if there is doubt concerning the nature of a cystic lesion.

## Clinical aspects

Humans are generally infected faecal-orally during childhood. The cysts grow very slowly, about 1 to 2 cm per year. The carrier may remain asymptomatic for a long time and symptoms are unusual before the cyst has reached 10 cm in diameter, at least in the liver, its preferred localization. There may be mechanical consequences. Pressure on surrounding organs leads to various symptoms and complaints. Hepatic cysts may lead to an enlarged liver with local discomfort, obstructive icterus with or without cholangitis. If localization is in the central nervous system this produces symptoms of a brain tumour, epilepsy, compression of the spinal cord or brain stem and even eosinophilic meningitis if there is spillage. If situated in the skeleton there is often bone pain, sometimes with fractures. This has to be differentiated from ordinary bone cysts or tumours. Lung cysts are usually asymptomatic, but sometimes there is a cough and thoracic discomfort. Renal cysts are sometimes found by chance and may cause unilateral kidney destruction. Allergic reactions may also occur, such as urticarial rash, bronchospasm, anaphylactic shock after rupture of a cyst (which may be spontaneous, after trauma or during surgery). After rupture there may be dissemination of the protoscolices in the peritoneum or pleura. Mechanical aspiration of a cyst may sometimes lead to rupture with allergic shock and dissemination.

## Diagnosis

Plain X-ray of the abdomen (crescentic calcifications), X-ray of the lungs or CT scan. Ultrasound of the liver shows a round or oval hypodense zone with retro-acoustic intensification. The cyst can contain septa or daughter cysts. The wall may appear split (the endocyst separated from the pericyst) or it may be partially or completely calcified. Sometimes the cyst appears heterogeneous and produces a pseudo-tumorous image.

Sometimes the diagnosis is made during surgery. In case of doubt as to the nature of a cystic mass, the content of the lesions may be examined for the presence of hydatid sand or the presence of the typical small hooks which remain after the protoscolices degenerate. Serology may be negative in the case of well encapsulated liver cysts and lung cysts. Sometimes the serology is positive or the titer increases during treatment due to leakage of the cyst content and release of antigen which cause the immune response to increase.

## Ultrasound

Various types of cysts can be identified by ultrasound. The following signs are regarded as pathognomonic for cystic echinococcosis (CE):

- Unilocular, anechogenic round or oval lesions with a pronounced laminated membrane or with snow-like inclusions.
- Multivesicular cysts or cysts with multiple septa with a wheel-like appearance.
- Unilocular cysts with daughter cysts which may exhibit a honeycomb appearance.
- Cysts with floating laminated membranes (“water-lily sign”) which may also contain daughter cysts.

Ultrasound is also of utmost importance to stage the liver cysts according to the 2010 WHO classification (see Fig 2 below), between active (or early: CE1 and CE2), transitional (C3a and C3b) and inactive (or late: CE4 and CE5) lesions. This has immediate implication for the prognosis and treatment of cystic echinococcosis

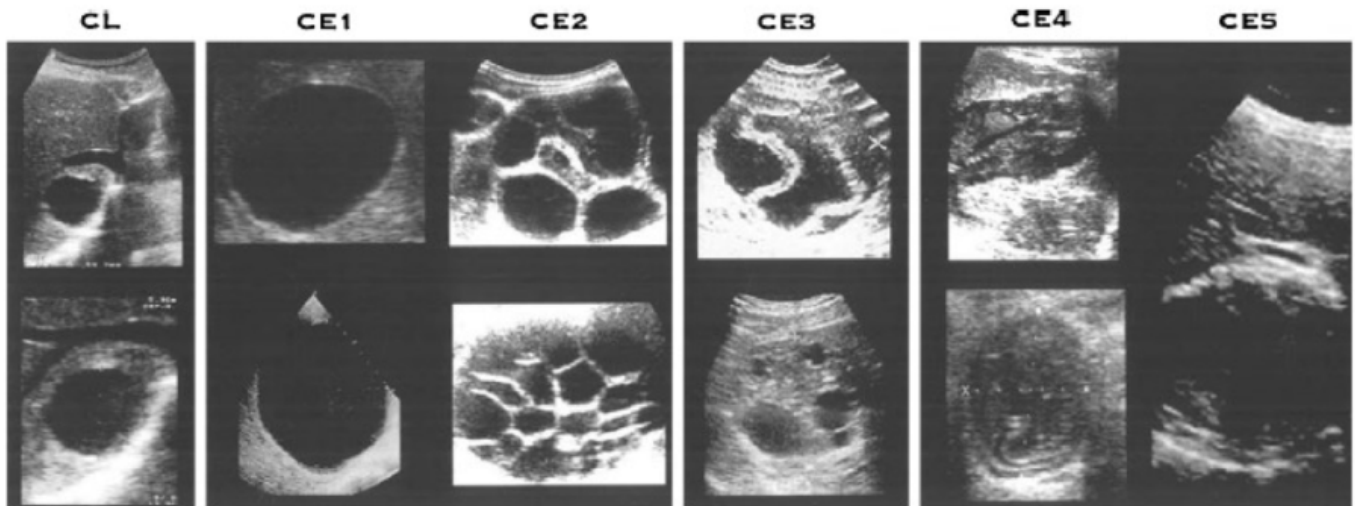


Fig. 2. WHO-IWGE standardized classification.

### WHO Classification of hepatic hydatid cysts

<b>CL</b>	Unilocular anechoic cystic lesion without any internal echoes and septations
<b>CE 1</b>	Uniformly anechoic cyst with fine echoes settled in it representing hydatid sand
<b>CE 2</b>	Cyst with multiple septations giving it multivesicular appearance or rosette appearance or honey comb appearance with unilocular mother cyst; this stage is the active stage of the cyst
<b>CE 3</b>	Unilocular cyst with daughter cysts with detached laminated membranes appearing as water lily sign; this is the transitional stage of the cyst
<b>CE 4</b>	Mixed hypo and hyperechoic contents with absent daughter cysts, these contents give an appearance of ball of wool sign indicating the degenerative nature of the cyst
<b>CE 5</b>	Arch-like thick partially or completely calcified wall; this stage of cyst is inactive and infertile

## Treatment

### Waiting

Many cysts remain stable, calcify or even involute spontaneously. Small, calcified cysts in the elderly can usually be left untreated. As a whole, a wait and see attitude is recommended for CE4 and CE5.

### Surgery

Pericystectomy or partial liver resection. Sometimes what is known as the “frozen-seal” method is applied. Using liquid nitrogen, a funnel is frozen onto the liver capsule to prevent accidental spillage.

The liver is opened and the cyst content evacuated. During the operation, lavage is carried out with a scolicidal agent. Surgery is the treatment of first choice for large cysts (> 10 cm), for CE2-CE3b lesions, if there is superinfection or communication with the biliary tract. For extrahepatic cysts, surgery is always the treatment of first choice. Albendazole is administered ideally prior to surgery (but optimal timing is unknown, up to 4 weeks), and praziquantel is given at the time of the operation. This is done in order to diminish the risk of disseminated infection in case of accidental rupture or spillage during operation. Post-operative complications are not unusual.

### Medication

Mebendazole is no longer used (only at high dose, in case of albendazole toxicity). Long-term therapy with albendazole (e.g. 800 mg daily for 6 to 9 months, blocks glucose uptake by the parasite) is usually used alone for CE1 and CE3a lesions < 5 cm and in combination with PAIR or surgery for bigger lesions or in CE2 and CE3b lesions. It is used in extended duration for inoperable and/or disseminated disease.

Previously this was given in cycles, but nowadays the medication is administered daily without interruption. The efficacy of medical therapy varies greatly (overall cure rate of 30%)

and clearly leaves much to be desired. Higher levels of albendazole sulphoxide (ricobendazole), the chief active metabolite, may be obtained by higher dosage, ingestion with a fatty meal, or by combination with praziquantel or cimetidine [cimetidine inhibits the breakdown of both albendazole and praziquantel].

Albendazole cannot be used during pregnancy. The combination albendazole (10-15 mg/kg daily divided in two doses) with praziquantel (40 mg/kg once a week) is probably more effective than either drug alone.

## **PAIR**

Percutaneous treatment with the PAIR technique (puncture-aspiration-injection-reaspiration) can be used for CE1 and CE3a lesions. Daughter cyst should be ruled out, since their presence reduces the likelihood of successful treatment with PAIR. Experienced surgeons can perform a laparoscopic variant of this technique. In hospitals where the necessary equipment is available, after detection of a cyst an endoscopic retrograde cholangiography is carried out. This permits determination of whether there is any communication between the cyst and the biliary tract. Under ultrasound or CT guidance the cyst is punctured transhepatically with a fine needle. The cystic pressure can be measured. Vital cysts have a pressure of 8-75 cm water. Dead cysts have a low pressure (0-2 cm water). Subsequently 10-15 ml of cystic fluid is aspirated. Live protoscolices are actively motile upon microscopic examination.

Biochemical analysis of the fluid for the presence of bilirubin is carried out to exclude communication with the biliary tree. If there is sufficient evidence of active echinococcosis, the remaining cystic fluid is aspirated. Afterwards a protoscolicidal agent is injected (generally 95% ethanol or 15-20% hypertonic salt). As a guideline the amount injected should be 1/3 of the volume of the aspirated fluid.

After 10 to 30 minutes the cyst content is then aspirated again. The risk of rupture, dissemination or anaphylaxis is minimal if there is at least 1 cm (preferably 2 cm) between the liver capsule and the cyst wall.

If there is a cyst-to-biliary tract fistula, the PAIR technique cannot be used due to the risk of

sclerosing cholangitis. It is advisable to begin albendazole one week before and to continue administering this until 4 weeks after the procedure. PAIR cannot be used for extra-hepatic lesions. Those who have no experience with PAIR are advised to leave this to an expert as the complication rate is quite high.

## Prevention

De-worm dogs and prevent them from eating offal.

Keep dogs out of slaughterhouses.

The first results of a recombinant vaccine (EG95) administered to sheep and goats, are encouraging, and show protection of 83-100% for these animals.

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## Echinococcus multilocularis

*Echinococcus multilocularis* or fox tapeworm is closely related to *E. granulosus* (dog tapeworm). The parasite occurs in the northern hemisphere, often in regions with a cold climate such as Alaska, the Alps, Siberia, north-west China and central Turkey. The eggs of the parasite are cold-resistant.

Transmission by sleigh dogs is known. Treatment of these draught animals with praziquantel reduces the transmission to humans. In the wild there is a cycle between canines (including fox, wolf, etc.) and various rodents, including mice. Domestic dogs and cats may also become infected. Humans become infected accidentally by faecal-oral transmission, e.g. by eating contaminated berries, or drinking water contaminated with fox faeces. After infection with eggs the larvae develop, resulting in alveolar hydatidosis of the liver and other organs. The cysts may calcify, but usually continue to grow slowly and constantly and are similar to a malignant growth. Metastasis may occur. There may be growth through to the diaphragm and into the inferior vena cava. Treatment is difficult and involves liver surgery and/or long-term therapy with antihelmintics (even life-long in inoperable cases).

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## Hymenolepis nana

In 1921 Saeki demonstrated direct transmission (i.e. without intermediate host) of *H. nana* in humans, in contrast with *H. diminuta* for which human infection requires ingestion of infected insects. *H. nana* occurs in foci and has a cosmopolitan distribution.

The highest prevalence of this cestode is found in hot, dry regions. People become infected by swallowing an egg (faeco-oral transmission) or by accidentally swallowing an insect (flea, weevil) which acts as intermediate host. An intermediate host is not essential for infection. Humans are the only definitive host. The adult worm is found in the lumen of the small intestine. The adult parasite is smaller than *H. diminuta*: it only measures 2-4 cm (dwarf tapeworm). The strobila contains 100 to 200 proglottids.

The course of infection is almost always asymptomatic, but marked hypereosinophilia can be present. Malignant transformation of *H. nana* has been described in an HIV-infected patient in 2015, being a novel disease mechanism of a neoplasm in invertebrates invading human tissue. The treatment of choice is praziquantel.

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