Taenia (including cysticercosis) and hydatid disease

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Taeniosis

Taenia spp. are cestodes of the order Cyclophyllidea and comprise a scolex with four suckers, a small neck, and a large body formed by a chain of proglottids. The scolex attaches the parasite to the small intestinal mucosa. Proglottids near the scolex are immature, those in the middle of the chain are mature and contain male and female sexual organs (hermaphroditic), and those in the distal end of the chain are gravid and contain thousands of eggs.

There are two major differences between *T. solium* and *T. saginata*.

- The scolex of *T. solium* is armed with a double crown of hooks (Figure 1), whereas that of *T. saginata* is unarmed.
- The uterus of gravid *T. solium* proglottids has 7–13 lateral branches, whereas that of *T. saginata* has 15–20.

There is no difference in the structure of the eggs of *T. solium* and *T. saginata*; those of both species are 30–40 µm in diameter, are spherical, and contain a six-hooked embryo (oncosphere).

Epidemiology: cestodes of the genus *Taenia* are common parasites of humans; conservative estimates are 3 million individuals infected with *T. solium* and 45 million with *T. saginata* worldwide. *T. saginata* has a worldwide distribution, whereas *T. solium* infection is prevalent in developing countries (except the Muslim countries of Asia and Africa) and in industrialized nations with high rates of immigration from endemic areas.

Life cycle: *Taenia* spp. require two hosts to complete their life cycle. Humans are definitive hosts for both *T. solium* and *T. saginata* and carry the adult worm attached to the small intestine, from which eggs are detached and excreted with faeces, contaminating water and vegetation. Eggs are ingested by intermediate hosts (pigs for *T. solium*, cattle for *T. saginata*) and carried to the host tissues, where the larval form (cysticercus) develops. The cycle is completed when the larvae are ingested by definitive hosts (in undercooked pork or beef), within which they develop into adult parasites. Humans may also act as intermediate hosts of *T. solium*, developing a severe disease called cysticercosis (see below).

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Clinical features and diagnosis: taeniosis is a benign condition producing loss of appetite, nausea, abdominal discomfort, constipation or diarrhoea. Mild eosinophilia and elevated serum IgE levels are common but nonspecific findings. *T. saginata* and *T. solium* infections cannot be differentiated by the appearance of the eggs in faeces. However, enzyme-linked immunosorbent assay (ELISA) using *Taenia*-specific antibodies for copro-antigen detection and DNA hybridization techniques for eggs have improved the diagnosis of taeniosis, enabling improved screening for *T. solium* carriers in endemic areas.

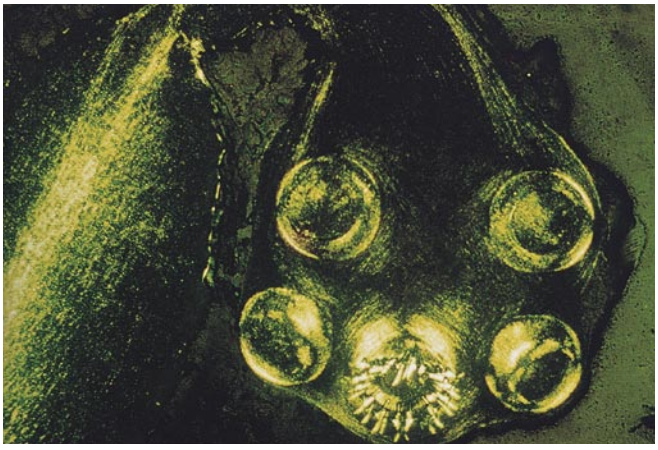
Management: both *T. solium* and *T. saginata* are highly sensitive to praziquantel, 5–20 mg/kg single dose. Niclosamide, 2 g in two divided doses 1 hour apart, is an alternative. Praziquantel and niclosamide kill the worm, but do not inactivate the eggs. Therefore, proper disposal of faeces after treatment is important to avoid the accumulation of a massive load of eggs in the environment.

Cysticercosis

Epidemiology: cysticercosis occurs when humans become intermediate hosts of *T. solium* after ingesting eggs in contaminated water or food, or by the faeco-oral route in individuals harbouring the adult parasite in their intestine. The disease is linked to poverty and ignorance of the mode of transmission. It is common in developing countries, where deficient disposal of human faeces, low levels of education, slaughtering of swine without veterinary control and the presence of free-roaming pigs around households (Figure 2) favour transmission of the parasite.

Pathology: cysticerci are vesicles containing an invaginated scolex that is structurally similar to the adult scolex. The parasites are found in the eye, skeletal muscles and CNS. Cysts in the CNS may lodge in the brain parenchyma, subarachnoid space, ventricular system or spinal cord. Parenchymal brain cysts usually measure 1 cm in diameter; subarachnoid and ventricular cysts may grow to 5 cm or more.

There are marked variations in the severity of the inflammatory reaction against cysticerci. Some parasites escape detection by the host's immune system and remain viable, eliciting little or no inflammatory reaction. Other cysticerci evoke an intense inflam-



1 Scolex of *Taenia solium* showing the characteristic four suckers and double crown of hooks.

2 Free-roaming pigs with access to human faeces perpetuate the life cycle of *Taenia solium* in areas endemic for cysticercosis.

matory response, causing destruction of the parasites and severe diseases related to damage of the surrounding cerebral tissue.

There is increasing evidence suggesting that periodic remodelling of calcified cysticerci may be associated with the release of cysticercal antigens in the brain parenchyma that, in turn, elicit recurrent inflammatory changes and relapsing symptoms.

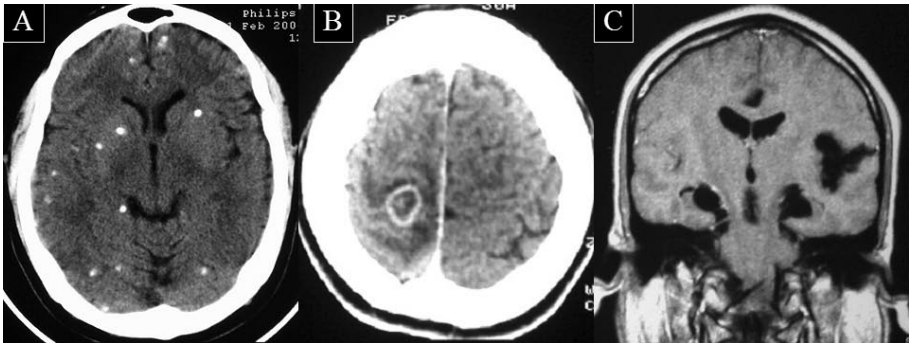
Clinical features: the signs and symptoms of neurocysticercosis depend on both the number and the location of lesions, and the severity of the host's immune reaction against the parasite.

- Epilepsy is the most common manifestation (70% of patients). Seizures are more common in those with parenchymal brain cysts than in other forms of the disease and usually occur in otherwise healthy, middle-aged individuals. The International League Against Epilepsy considers cysticercosis to be the most common explanation for the fact that active epilepsy is more common in developing countries than in industrialized nations.
- Various focal signs have been described in patients with neurocysticercosis, including pyramidal tract signs, sensory abnormalities and signs of brain stem dysfunction.
- Some patients present with increased intracranial pressure or dementia related to hydrocephalus or massive cysticercal infection of the brain parenchyma.
- Cysticercosis outside the CNS is usually asymptomatic, though subretinal cysticerci may cause sudden blindness, and massive infestation of skeletal muscles may produce a syndrome of muscular pseudohypertrophy.

Diagnosis of cysticercosis is a challenge; over-diagnosis and misdiagnosis have been the rule in almost every study that has attempted to determine the prevalence in a given population. Confusion also occurs with hospitalized patients undergoing sophisticated diagnostic tests, because of the lack of specificity of some neuro-imaging findings (Figure 3), and the high rate of false-positive and false-negative results that occur with the most commonly used immunological tests. Diagnostic criteria have been developed to evaluate objectively the clinical, radiological, immunological and epidemiological data, based on three degrees of diagnostic certainty (definitive, probable and possible, Figure 4). Proper use of these criteria should enable physicians to avoid most diagnostic pitfalls.

Management must be individualized according to the location of the parasites and the degree of disease activity.

- Patients with viable parenchymal brain or subarachnoid cysts must be treated with albendazole, 15 mg/kg/day for 8 days. A recent double-blind, placebo-controlled trial confirmed that this approach results in the disappearance of most cysts on neuroimaging and marked improvement of the clinical manifestations.[1] Praziquantel, 50 mg/kg/day for 15 days or 75–100 mg in three divided doses given on the same day 2 hours apart, is an alternative to albendazole. Patients with calcified lesions should receive only symptomatic treatment (e.g. anti-epileptic drugs for seizures). Patients with ventricular or intra-ocular cysts may also be treated with albendazole; however, the usual approach is surgical resection.
- Cysticidal drugs are contraindicated in those with cysticercotic encephalitis, because they may exacerbate the brain oedema associated with this condition. In these patients, dexamethasone, 16 mg/day, and mannitol, 100 ml 6-hourly, are advised for management of intracranial hypertension.
- In patients who have mixed forms of the disease, therapeutic priorities (e.g. shunt placement for hydrocephalus) must be considered before starting cysticidal drugs.
- Corticosteroids are indicated in patients with cysticercotic encephalitis or angiitis and in those with massive cysticercal infection of the brain parenchyma. Anti-epileptic drugs should be used in patients with seizures.



3 Neuro-imaging studies showing common but non-pathognomonic findings of neurocysticercosis, including parenchymal brain calcifications (left), a single, ring-enhancing lesion (centre) and subarachnoid cysts in the sylvian fissure and CSF cisterns at the base of the brain (right).