249 Taenia solium (Cysticercosis)

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Definition

Cysticercosis is a parasitic infection of human tissues with larvae of the pork tapeworm, *Taenia solium*.

Etiology

Definitive host: human tapeworm infection

The *T. solium* tapeworm infects the human small bowel. It typically lives for a few months or years (rarely up to 20 years), growing usually to 2–4 m in length (rarely up to 8 m). Interestingly, infection with more than one *T. solium* tapeworm at a time is very rare. Each tapeworm intermittently produces thousands of eggs when the most distal tapeworm segments (proglottids) are released. These gravid tapeworm segments may be passed intact or they may rupture allowing parasite eggs to disperse in the feces. The eggs are highly infectious and remain viable within the environment for many months.

Intermediate host: porcine cysticercosis

Known since antiquity, porcine cysticercosis was described by Aristotle. Pigs become infected when they ingest material contaminated with human feces containing microscopic tapeworm eggs. Within the pig bowel the eggs hatch, releasing activated oncospheres that then penetrate the bowel wall and are carried to the host tissues, probably by the bloodstream. Within 3 months the oncospheres develop into fluid-filled cysticerci, which are usually ovoid in shape and 5–10 mm in length. Except in the rare racemose form of the disease, each cysticercus initially contains a 2 mm long, living larval tapeworm scolex.

When humans ingest undercooked, cysticercotic (measly) pork, cysticerci are digested within the bowel, releasing tapeworm larvae that attach to the wall of the jejunum and grow into adult tapeworms, completing the parasitic life cycle. The relationship between cysticerci and the adult intestinal tapeworm was proven in 1853 by feeding condemned convicts with raw measly pork and, after execution, demonstrating the presence of young tapeworms within the human bowel.

Human cysticercosis

Human cysticercosis is acquired through the fecal–oral route. When humans ingest tapeworm eggs they may develop cysticerci within their tissues in the same way as pigs. Cysticercosis is therefore contracted from ingesting material contaminated by human feces containing tapeworm eggs, not from eating infected pork containing cysticerci. Strict vegetarians and people who have never eaten pork may develop cysticercosis, although they are not at risk of developing intestinal tapeworm infection. Humans are an incidental intermediate host and represent a 'dead-end' for the parasite, unless human tissues are eaten raw by cannibals, who might then develop an intestinal tapeworm.

Humans harboring an intestinal tapeworm can infect themselves (anus–hand–mouth) or others directly with cysticercosis through unhygienic food preparation. However, most patients with cysticercosis do not harbor an adult tapeworm within their bowel and most people infected with a tapeworm are believed not to develop symptomatic cysticercosis. Indeed, it is not known what proportion of tapeworm carriers will develop asymptomatic or symptomatic cysticercosis over time.

Geographic distribution

Cysticercosis is endemic on all continents except Australasia and is common in nonMuslim developing countries including much of South and Central America, Asia and Africa. Prevalence varies greatly depending upon the prevalence and type of animal husbandry, hygiene and dietary practices. Since cysticercosis is a disease of poverty that has quite nonspecific clinical features, this disease is generally least recognized in the areas where it is most common. For example, the prevalence of neurocysticercosis has not been defined in many parts of sub-Saharan Africa. In general, cysticercosis is likely to exist in most less-developed counties where pigs roam freely and are eaten by humans.

Prevalence

Neurocysticercosis is often said to be the most common parasitic infection of the brain and the most common single cause of adult-onset epilepsy worldwide. However, AIDS-associated cerebral toxoplasmosis is increasing in incidence and the proportion of epilepsy attributable to cysticercosis has not been defined in most endemic areas. Autopsy studies have revealed cysticercosis in 0.1–3.5% of people in Mexico, although up to 80% of these infections were apparently asymptomatic and the infection was unrelated to the cause of death. In Peru, 12% of patients with epilepsy are seropositive compared with about 1% of the general population.

Relatively little has been published recently about the prevalence of cysticercosis in the former Eastern-block countries and sub-Saharan Africa, and the marked differences between the clinical features and epidemiology of cysticercosis between Latin America and Asia make assumptions about cysticercosis based upon epilepsy prevalence quite uncertain.

Epidemiology

In industrialized countries, meat inspection, sewage disposal, tethering of pigs and cooking of pork all reduce propagation of the parasite life cycle. However, immigration from developing countries and increasing travel to endemic regions cause sporadic cases of cysticercosis. For example, cysticercosis accounts for up to 2% of neurologic/neurosurgical admissions in Southern California and immigrant cooks with tapeworm infection caused an outbreak of cysticercosis in orthodox Jews in New York.

Pathophysiology

Living cysticerci actively evade and suppress immune recognition, especially within the so-called 'immunologically privileged sites' such as the brain and the eyes. Histopathologic examination of living cysticerci excised from human tissues usually reveals minimal.

predominantly cellular, host inflammation. Cysticerci are frequently found without any sign of cellular inflammation, especially in striated muscle. In other tissues and in cysticerci damaged by therapy there may be a significant inflammatory infiltrate but cysticerci still remain viable for a variable period as demonstrated by their ability to evaginate. It is uncommon for living cysticerci to induce symptoms and when this does occur it is usually by causing obstruction to the flow of CSF or pressure on adjacent tissues.

In infected humans, symptoms most commonly result not from the initial presence of living cysticerci but rather from the subsequent death of one or more cysticerci, which is usually associated with increased inflammation. This explains the usual delay between the formation of cysticerci, which occurs within 3 months of infection, and the development of symptoms several years later. This point is well illustrated by a classical epidemiologic study of the natural history of cysticercosis in which English soldiers returning from India and remaining in Great Britain (a nonendemic area) developed seizures caused by cysticercosis after a median of 7 years rather than immediately after infection.

Inflammation around dying cysticerci may be asymptomatic or it may lead to acute symptoms that can prove fatal. Lymphocytes and plasma cells usually outnumber giant multinuclear cells and foamy macrophages. Eosinophils are conspicuous. Adjacent necrosis and perivascular infiltration of mononuclear lymphoid cells may be followed by formation of a granuloma, fibrous scar or calcification.
Several classification systems define neurocysticercosis as active if living or degenerating cysticerci are present, and inactive if there are calcifications or fibrosis. However, cysticerci at different sites may be viable or at various stages of degeneration and in some studies most patients had both types of lesion.

In the brain there are two forms of cysticerci. In the more common form, cysticerci located in the brain parenchyma and/or floating freely in the ventricles are ovoid, 5–10 mm structures, each of which contains a scolex. A second, uncommon form called racemose ('bunches of grapes') cysticerci describes large lobulated vesicular structures that lack a scolex and are usually found in the basal cisternal spaces. This form of cysticerci is not usually present in pediatric cases and may be either a degenerative form or a response to a different anatomic location. In general, parenchymal cysticercosis follows a benign course whereas racemose cysticercosis often proves fatal.

Clinical features

The clinical features of cysticercosis are very variable, depending upon the inflammatory response around cysticerci, their number, size and location. Although cysticerci can infect any tissue, the great majority of clinically relevant lesions affect the brain and spinal cord (termed neurocysticercosis), the eye, muscles or skin.

Neurocysticercosis

The most common symptom of cysticercosis is epilepsy. Focal seizures with secondary generalization are most frequent, but all types of epilepsy can occur. Single or multiple cysticerci are usually present within the brain parenchyma and may be surrounded by a focal encephalitis and edema. Raised intracranial pressure may cause headache and vomiting. Occasionally, cysticerci may also cause dementia or psychiatric illness, often in association with hydrocephalus.

Cysticerci degenerating in or near the meninges can cause chronic meningitis. When the base of the brain is affected, the resultant basal arachnoiditis is often particularly severe and may cause cranial nerve palsies and potentially fatal raised intracranial pressure.

Intraventricular or basal cysticercosis can cause obstructive hydrocephalus. This is usually progressive but may rarely be intermittent and positional because of the varying location of a free-floating cysticercus. Cysticerci degenerating in any part of the brain may cause vasculitis and cerebral infarcts, most commonly when the base of the brain is infected.

The rare encephalitic form of cysticercosis occurs most commonly in children and young females and consists of massive infection of the brain. Lesions are of similar size, in the same phase of development. Although this form of the disease may be associated with a prominent immune reaction causing severe brain edema, the principal cause is likely to be unusually heavy infection that might result from ingestion of an entire tapeworm segment containing thousands of viable eggs. These patients have clinical symptoms of intracranial hypertension, seizures that are difficult to control and a reduced level of consciousness.

Spinal cysticercosis is uncommon and often associated with severe symptoms due to increased pressure either directly from the cysticercus or secondarily from the inflammatory reaction. Cervical spinal cysticercosis is usually associated with racemose cysticercosis of the posterior fossa.

Ophthalmic cysticercosis

Cysticerci infecting the eye are most commonly retinal or subretinal, but may float freely in the vitreous or aqueous humors. Light during ophthalmoscopy has been reported to induce movement and even evagination of a living parasite. Inflammation around degenerating cysticerci usually threatens vision by causing chorioretinitis, retinal detachment or vasculitis.

Muscular and subcutaneous cysticercosis

Some patients with cysticercosis have characteristic subcutaneous cysticerci palpable as pea-like nodules. These are usually asymptomatic, although transient local pain and tenderness may occur as cysticerci degenerate. The frequency of subcutaneous nodules varies considerably by geographic area with less than 5% of patients in South America and over 20% of patients in Asia having subcutaneous nodules.

Subcutaneous nodules are also common is cysticercosis patients in parts of Africa. These lesions are easy to biopsy and thus can provide histopathologic proof of disease. However, a clinical diagnosis may be made in typical cases without a biopsy.

Multiple parasites may infect the muscles and rare cases of massive muscular pseudohypertrophy have been reported. The enormous numbers of cysticerci present in the muscle are associated with an increase in the size of the muscle although the muscle is extremely weak. These patients may also have multiple cysticerci in the brain, pleura, heart and tongue, and the prognosis is poor, as in the encephalitic type of infection.

Laboratory and radiologic diagnosis

Neurocysticercosis should be considered in any patient who develops neurologic symptoms, especially if associated with adult-onset seizures. It should be actively excluded in endemic countries or in patients with a history of travel to such an area.

Patients should be asked whether they have passed tapeworm segments, even decades previously, and stool specimens should be examined for tapeworm eggs since some cases of cysticercosis result from autoinfection from an intestinal tapeworm. Microscopy is quite an insensitive test for diagnosing intestinal *T. solium* tapeworm infections; however, an enzyme-linked immunosorbent assay (ELISA) for the detection of tapeworm antigen in stool has recently been developed and tested with promising results, but this is not generally available. *T. solium* eggs in the feces cannot be macroscopically differentiated from other

tapeworm eggs, but only *T. solium* eggs cause human cysticercosis. Treatment with an antihelminthic agent causes the worm to be passed in the feces, allowing definitive identification of the tapeworm species.

Biopsy

A search should be made for subcutaneous cysticerci, excision biopsy of which confirms the diagnosis of cysticercosis. The gross biopsy specimen is a white, fluid-filled, semi-opaque bladder, typically 5–10 mm in diameter but rarely up to 70 mm. This bladder contains a solid, 2 mm long larval tapeworm scolex. In live specimens, independent movement of the scolex may be seen after excision. Microscopic examination reveals four suckers and a characteristic double row of hooks on the scolex. These are the apparatus with which an ingested scolex attaches to the human intestinal wall.

Radiology

Plain X-rays of the thighs or other muscles may reveal multiple calcifications along the fascial plains formed by degenerated cysticerci and plain X-rays of the skull may reveal intracranial calcifications.

Computed tomography (CT) has revolutionized the antemortem diagnosis of neurocysticercosis, but availability is severely limited in the regions where cysticercosis is most common. Living cysticerci are visible as 5–10 mm hypodense lesions that do not enhance with intravenous contrast. High resolution scans reveal the small, hyperdense scolex within each living cysticercus. Degenerating cysticerci, which are more likely to be associated with symptoms, are surrounded by edema with ring or nodular enhancement after the administration of intravenous contrast. End-stage calcified cysticerci are seen as hyperdense lesions.

Magnetic resonance imaging (MRI) provides more detailed images of living and degenerating cysticerci. MRI frequently demonstrates cysticerci that may not be seen on CT, especially those located in the posterior fossa. However, MRI often will not demonstrate calcifications as clearly as CT.

Serology and CSF examination

Several different types of serologic test have been used to diagnose cysticercosis. An enzyme-linked immunoelectrotransfer blot (EITB) has been evaluated in several endemic and nonendemic countries and yields greater than 98% sensitivity and specificity except for 50–80% sensitivity for single ring-enhancing lesions. This test utilizes a group of lentil–lectin purified glycoprotein antigens in an immunoblot format to detect infection-specific antibodies in serum or CSF samples. One of its advantages is its equal sensitivity in serum or spinal fluid, so a lumbar puncture is not required for accurate serologic diagnosis.

Despite the reliability of this assay, results must be interpreted with caution in patients from endemic regions where approximately 1% of the healthy urban population and 10% of rural villagers in pig-raising areas may be seropositive. However, patients with clinical symptoms tend to have more specific bands than those who are asymptomatic and patients with six or more specific EITB bands have more intracranial lesions than those with fewer bands. The cysticercosis EITB has largely superseded the less sensitive ELISA assays.

CSF examination may be normal and, even when abnormal, there are no characteristic features. There may be moderate CSF pleocytosis and elevated protein concentration. Eosinophils are present in the CSF of up to 25% of cysticercosis patients, but these may be mistaken for lymphocytes unless appropriate stains are used.

Differential diagnosis

The heterogeneous clinical features of cysticercosis make diagnosis difficult but the combination of radioimaging and reliable serology greatly facilitate antemortem diagnosis. A single ring-enhancing intracranial lesion may present a difficult differential diagnosis since 20–50% of patients with these types of lesion may have a negative EITB serologic test. Other possible causes of this lesion are tuberculosis, a neoplasm or brain abscess caused by bacteria, fungi or *Toxoplasma gondii*.

The differential diagnosis of cystic images other than cysticercosis are subarachnoid or porencephalic cysts, hydatid cysts and cystic astrocytomas. Intracranial calcifications may also be caused by tuberculosis, toxoplasmosis, tuberous sclerosis and cytomegalovirus. Cysticerci outside the CNS are generally easier to identify but subcutaneous cysticerci may be clinically mistaken for multiple subcutaneous lipomas.

Management

The treatment of cysticercosis is controversial and is best considered with reference to the natural history of the condition, which is therefore discussed here, before specifics of cysticercosis therapy.

Natural history

The natural history of cysticercosis is poorly defined because most infections are asymptomatic. In some rural pig-raising, less-developed country communities, over 10% of the population have antibodies to *T. solium* but a great majority are asymptomatic. Due to the lack of longitudinal community studies, the risk of symptomatic disease developing in asymptomatic individuals who have intracranial cysticerci is not known. Also, it is not clear how often symptomatic patients spontaneously recover. However, it is clear from the research discussed in the pathophysiology section above that initial cerebral infection with living cysticerci causes little inflammation and is usually asymptomatic. Usually, it is the degeneration of one or more cysticerci some years later that is associated with symptomatic inflammation.

Drug therapy

Drug therapy with either albendazole (15 mg/kg orally for 8–15 days) or praziquantel (50–75 mg/kg/day orally for 2 weeks) is effective in killing live cysticerci. Albendazole is currently the drug of choice because of slightly greater efficacy and lower cost. A short course of three doses of 75 mg/kg of praziquantel in the same day has been reported to be efficacious. Between the second and the fifth day of anticysticercal therapy, there is usually an exacerbation of neurologic symptoms attributed to the death of larva(e) and associated local inflammation. For this reason, albendazole or praziquantel is generally given simultaneously with corticosteroids to control edema and intracranial hypertension (a proven adult treatment regime is 400 mg of albendazole given orally every 12 hours and 2 mg of dexamethasone given orally every 8 hours for 10 days). Most clinicians treat neurocysticercosis patients in hospital to ensure that these adverse effects can be detected and dealt with swiftly. Although co-administration of corticosteroids reduces the blood levels of praziquantel and may increase the blood levels of albendazole, this is not thought to be clinically relevant.

The weight of clinical opinion is that treatment with albendazole or praziquantel is useful for symptomatic cysticercosis. Most reports of albendazole or praziquantel efficacy have been retrospective or unblinded. However, prospective placebo-controlled trials have confirmed clinical benefits from this therapy. The great majority of clinicians prefer to administer one of these drugs to patients with symptomatic neurocysticercosis caused by viable cysticerci. Patients with seizures associated with only degenerated or calcified cysticerci are unlikely to benefit from therapy with albendazole or praziquantel. In all patients with seizures caused by neurocysticercosis, supportive care with anticonvulsant drugs is also extremely important.

Asymptomatic neurocysticercosis

In most cases, viable cysticerci living within the human brain are asymptomatic but imaging studies performed for other reasons may occasionally reveal them. If the cysticerci are not causing symptoms and are not associated with radiologic evidence of inflammation then the parasites may be assumed to be alive and successfully evading immune-mediated inflammation. In such cases, cestocidal treatment usually kills the parasites and allow the prophylactic (or prompt) administration of corticosteroids if treatment causes symptoms. However, cysticerci infecting the brain often resolve without symptoms so an alternative choice is not to treat and advise the patient to seek medical help if neurologic symptoms develop. Research has yet to define the optimal course of action in these uncommon cases.

Symptomatic neurocysticercosis

Anticonvulsant drugs should be administered to neurocysticercosis patients who have a significant frequency of seizures, as for any other cause of epilepsy. Raised intracranial pressure, with or without associated arachnoiditis, usually responds to short-term oral corticosteroids. Corticosteroids may be required chronically in rare cases of persistent intracranial inflammation. Insertion of a ventriculoperitoneal shunt for hydrocephalus is beneficial in the short term but shunt blockage is common when the CSF protein concentration is elevated. Concomitant use of corticosteroids in patients with ventriculoperitoneal shunts has been reported to improve prognosis.

A viable cysticercus producing mass effect should be treated with anticysticercal drugs. In the case of patients with epilepsy and viable cysticerci, anti-helminthic therapy with albendazole has been proven to reduce seizure frequency and is therefore recommended. However, if epilepsy or other symptoms are caused by one or more cysticerci that are seen on neuroimaging studies to be degenerating, then cestocidal therapy is unlikely to be of any benefit. In such cases, an expectant policy has been utilized in parts of India with no intervention over a 6–12 week period unless there is clinical deterioration. Repeat CT scan after this period usually shows reduction in size or disappearance of a degenerating cysticercus. In some cases, differentiating a tuberculoma from a cysticercal granuloma may be difficult, often necessitating empirical therapy for tuberculosis.

Ophthalmic cysticercosis

The inflammation associated with a degenerating intraocular cysticercus is sufficiently severe to require both local and systemic corticosteroids but there may still be permanent loss of vision in the affected eye. There are reports of successful treatment with cestocidal drugs, cryotherapy and photocoagulation, but ocular cysticercosis is usually treated surgically. Excision of a living cysticercus before the onset of significant intraocular inflammation has a good prognosis.

Muscular and subcutaneous cysticercosis

Asymptomatic subcutaneous or intramuscular cysticerci do not require treatment but neuroimaging studies should be performed to exclude the presence of coincidental brain infection. Cysticerci causing symptoms through local pressure may be excised or treated with cestocidal drugs. Transient, symptomatic inflammation around degenerating cysticerci may be ameliorated by corticosteroid or nonsteroidal anti-inflammatory drugs.

Immunologic therapy

Although immunotherapy with purified cysticercal antigens has been shown to cause the degeneration and impaired viability of cysticerci in naturally infected pigs, this research strategy has no place in the treatment of humans. Indeed, it is possible that reinfection or exposure to *T. solium* antigens in food may precipitate the degeneration of previously tolerated intracranial cysts. The important role of immunosuppressive corticosteroids is described above.

Complications

Related problems of cysticercosis include the socioeconomic impact of the disease.

Prognosis

Symptomatic cysticercosis usually has a benign prognosis but should be considered to be a serious disease as in a minority of cases it may be progressive and have fatal consequences. In general, more numerous intracranial cysticerci are associated with more severe disease that is less likely to respond to treatment. The location of cysticerci is also important: patients with fewer than 20 cysticerci with predominantly parenchymal location and the absence of hydrocephalus have a better prognosis than those who have multiple, basal or ventricular lesions, especially if associated with hydrocephalus.

The epilepsy associated with neurocysticercosis responds to anticonvulsant medications in a fashion similar to other forms of seizure disorder. The optimal duration of anticonvulsant medication has yet to be defined.

Prophylaxis

The transmission of cysticercosis depends on the presence of tapeworm carriers. Human infection with intestinal tapeworms is prevented by destruction, freezing or adequate cooking of cysticercotic (measly) pork. In contrast, human cysticercosis results from fecal–oral contamination, and basic hygiene and sanitation prevent this disease.

Community monitoring

Establishing the intensity of cysticercosis in the community and monitoring the effect of cysticercosis control interventions require the use of simple epidemiologic indicators. The age-stratified curve of the prevalence of human cysticercosis infection, as documented by serology, is useful but cannot demonstrate changes in infection patterns because antibodies to cysticercosis persist for many years, even after successful treatment.

Studies in Peru have shown that serologic monitoring of infection in pigs is a useful indicator because the prevalence of porcine infection correlates with the prevalence of human cysticercosis. Pigs are generally kept for less than 1 year, so changes in infection intensity may be detected rapidly. Also, the rate of infection that occurs in uninfected (sentinel) pigs reflects the intensity of *T. solium* tapeworm infection and fecal contamination in the community. These indicators of porcine infection can therefore be used to monitor the effect of control programs in communities.

Community interventions

The control of cysticercosis in endemic regions is complex. Control programs utilizing education or the administration of human antihelminthic drugs to eliminate intestinal tapeworms have generally had limited and unsustained effects. Mass simultaneous antihelminthic treatment of both pigs and humans is being tried in some communities.

However, endemic cysticercosis has disappeared from many developed countries as a result of improvements in sanitary conditions and changes in pig-rearing practices. In areas where these simple measures are not yet attainable, development of an effective vaccine against porcine cysticercosis would provide a potential tool for eradicating the disease.

Essential messages

- Neurocysticercosis should be considered in all cases of acquired epilepsy in endemic regions – generally but not exclusively nonMuslim, less-developed regions.
- Cysticercosis is acquired by ingesting material that has been contaminated by human feces containing microscopic tapeworm eggs. Cysticercosis therefore commonly occurs in vegetarians and people who do not eat pork.
- The key to treatment is symptomatic care with anticonvulsant medication. Antihelminthic therapy with albendazole or praziquantel has a role in selected patients and these should generally be administered with a short course of corticosteroids, which should also be given if there is evidence of intracranial inflammation. Surgical therapy is generally restricted to ventriculoperitoneal shunting in cases of hydrocephalus.

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