

Neurocysticercosis: Manifestations, Diagnosis and Treatment

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ABSTRACT

Neurocysticercosis, is the infection caused by the larval form of the tapeworm *Taenia solium*. It is considered as the most common parasitic disease of the central nervous system and the most common cause of acquired epilepsy. This has primarily been a disease that remains endemic in countries with poor economy, but because of increased migration neurocysticercosis is being diagnosed more frequently worldwide. During the past decades improved diagnostics, imaging, and treatment have led to more accurate diagnosis and improved prognosis for patients. This article is a review about the current data about neurocysticercosis, including recent diagnostics and treatment developments.

Keywords: Cysricercosis, epilepsy, tapeworm, CNS

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INTRODUCTION

Neurocysticercosis (NCC) is caused by the infection of brain by larval stage of the tapeworm *Taenia solium*^{1,2}. It is the most common parasitic disease of the nervous system in humans and the single most common cause of acquired epileptic seizures in the developing world, where prevalence rates of active epilepsy are twice those in developed countries³⁻⁶. The disease is endemic in most of the developing world, where all the conditions favoring the transmission of this parasitosis, including warm climate, severe poverty, and illiteracy are combined^{7,8}. The disease is also a health problem in urban centers of developing countries, where NCC is a major cause of admissions to neurological hospitals⁹. However, NCC has also been recognized in persons with no history of travel to endemic areas¹⁰.

Here we aim to review the most important aspects of this disease, with emphasis on recent diagnostic and therapeutic advances.

Life Cycle and Pathogenesis

Taenia solium is a two-host zoonotic cestode. The adult stage is a 2- to 4-m-long tapeworm that lives in the small intestine of humans. No other final hosts are known for *T. solium* tapeworms in nature. As in all cestodes, the gravid proglottids at the terminal end of the worm are full of eggs that are the source of infection with the larval stage, or cysticercosis. The natural intermediate host is the pig, harboring larval cysts anywhere in its body. Humans become infected with cysts by accidental ingestion of *T. solium* infective eggs by fecal-oral contamination^{11,12}. The complex life cycle of *Taenia solium* involves two hosts. In the normal cycle of transmission, the adult *T. solium* inhabits the small intestine of humans, where it is attached to the intestinal wall by its potent suckers and hooks. Gravid proglottids are detached from the distal end of the worm and are passed with the feces, liberating thousands of fertile eggs to the environment. In places with deficient disposal of human feces, pork is fed with

human feces containing *T. solium* eggs. The eggs of *T. solium* can remain viable for up to 2 months in water, soil or vegetation. Once the eggs are in the intestinal tract, the actions of bile and pancreatic enzymes dissolve their protective coatings. Liberated from their coats, they become embryos (oncospheres), penetrate the intestinal wall and enter the bloodstream. There they are carried to the tissues of the host, where they eventually localize as cysts within the brain, the skeletal muscles, and the eyes and become established as cystic larvae or cysticerci. Human cysticercosis should be considered as a disease mostly transmitted from person to person, and the role of infected pork is to perpetuate the infection¹³⁻¹⁷.

Clinical Presentation

From the moment a cysticercus invades the brain tissue, for example, it is exposed to a hostile environment. When the host's immune system recognizes the parasite as foreign, it usually mounts an appropriate inflammatory reaction to overcome the infection. Laboratory studies and information from other cestodes suggest that viable cysts actively modulate the host's immune system to evade destruction by it. As in many cases, however, such a vigorous response does not occur, and the host reaches a state of immunologic tolerance to the parasite, leaving it almost undisturbed for many years. No clear explanation exists for the individual differences observed in the severity of immunologic response against infection of the central nervous system by cysticerci¹⁸. The clinical manifestations of NCC largely depend on the number, type, size, localization, and stage of development of cysticerci, as well as on the host immune response against the parasite. There are no pathognomonic features or a typical NCC syndrome¹⁹⁻²². NCC can cause many neurologic symptoms. Late-onset epilepsy is the most common clinical symptom; NCC is the leading cause of epilepsy worldwide²³. Seizures are more frequently observed in patients with parenchymal NCC than in those with subarachnoid or ventricular disease²⁴. Epileptogenesis in patients with calcified NCC has been a subject of debate²⁵. While calcifications have been considered inert lesions, recent data suggest that calcified cysticerci are not clinically inactive nor pathologically inert lesions, as they may cause recurrent seizures when parasitic antigens trapped in the calcium matrix are exposed to the host immune system due to a process of calcification remodeling²⁶. Patients with NCC may also present with headache associated with intracranial hypertension (another common presentation in disease-endemic countries), focal signs or dementia.

Intracranial hypertension is most commonly caused by hydrocephalus related to granular ependymitis, arachnoiditis or ventricular cysts. Other causes include giant cysts or cysticercotic encephalitis. The course of disease may remain subacute or chronic for many years, then present with focal signs of a cerebrovascular event secondary to an acute inflammatory response to the parasites. Cysticerci can also invade the spinal cord, the eyes and the subcutaneous and muscular tissues of the body. Muscular pseudo hypertrophy is seen more frequently in patients from Asia who has cysticercosis; it accounts for 0.6% of cases in China, but is extremely rare in America¹⁸.

The natural history of parenchymal cysticercosis has been studied by pathological examination²⁷⁻²⁹, and imaging studies using CT,^{30,31} and MRI³². Viable cysts are 10 to 20 mm in diameter, thin-walled sacks filled with clear cyst fluid.

On imaging studies, the wall is not visible and the fluid is isodense with the cerebrospinal fluid. There is little or no evidence of perilesional inflammation, and they do not enhance with contrast media on neuroimaging. As the parasite loses the ability to control the host immune response, an inflammatory process begins. Initially, the cysts show slight pericyclic contrast enhancement. Later they become markedly inflamed and edematous and appear as ring-like or nodular areas of enhancement after the injection of contrast. This phase has been called "granulomatous cysticercosis," "cysticerci in encephalitic phase," or "enhancing lesions." Finally, the cyst is processed by the cellular response, and its remnants either are not detectable by imaging or become calcified lesions. "Giant" cysts, measuring more than 50 mm in diameter, are occasionally found, located primarily in the Sylvian fissure. Cysticercotic encephalitis is a rare form of the disease in which patients have numerous inflamed cysticerci, leading to diffuse, severe cerebral edema. Extraparenchymal NCC includes cysticerci in the ventricles and basal cisterns (racemose cysticercosis). Since the cyst membrane is thin and the fluid is isodense with the cerebrospinal fluid, uninflamed extraparenchymal cysticerci are usually not visible on CT and may only reveal subtle, indirect findings on MRI. Scans may reveal hydrocephalus without noticeable cysts, ependymitis, distorted basal cisterns, or basal meningitis.

DIAGNOSIS

The diagnosis of NCC is suspected in patients living in endemic areas presenting with a compatible clinical picture and lesions suggestive of NCC on CT scans.

Neuroimaging is the mainstay of diagnosis of NCC. Table 1 shows proposed objective diagnostic criteria³³.

These criteria are complex and need validation in population or hospital-based studies. The major drawback of these criteria is that they do not differentiate between NCC and tuberculoma, which is of utmost importance in endemic countries, such as India³⁴⁻³⁶. Moreover, access to enzyme-linked immunoelectrotransfer blot assay (EITB) is limited in India and consequently the usefulness of these criteria has been questioned^{37,38}.

Imaging

Apart from histologic examination the diagnosis of NCC is based on results of neuroimaging studies. Computed tomography is the most commonly used neuroimaging test for the disease and maintains relatively good diagnostic sensitivity when used in disease-endemic areas^{39,40}. Viable cysts are non-enhancing, either in leptomeninges or parenchyma. After several years, the organism dies and the cyst starts to degenerate and enhance. The cyst wall thickness due to inflammation forms a fibrous capsule and the cyst fluid become denser. Eventual cyst shrinkage can take months to years with replacement by a small calcification. Calcifications in the brain parenchyma are the most common finding in computed tomography studies and, in many cases, the only radiologic evidence of the disease. For that reason, computed tomography remains the best screening tool for assessing patients with suspected NCC⁴¹⁻⁴³.

Preliminary experience with proton magnetic resonance spectroscopy has shown promise in differentiating tuberculoma from NCC⁴⁴.

Small lesions, especially those situated close to bone or within ventricles, may be missed on computed tomography scans. A magnetic resonance imaging scan is therefore often added for increased diagnostic sensitivity and accuracy. Magnetic resonance imaging is also the modality of choice when evaluating patients

with intra-ventricular cysticercosis and when assessing brainstem cysts and small cysts located over the convexity of the cerebral hemispheres. The main shortcoming of magnetic resonance imaging is its failure to detect small calcifications. The appearance of cysticerci in brain parenchyma on neuroimaging scans depends on their stage of development. Vesicular cysts appear as rounded lesions with signal properties similar to those of cerebrospinal fluid in both T1- and T2-weighted images. The scolex may be seen within the cyst as a high-intensity nodule, giving the lesion a pathognomonic “hole-with-dot” appearance.

When the parasite begins to degenerate, the lesion becomes heterogeneous, and its appearance varies depending on the degree of degeneration. Cysticercotic encephalitis involves multiple cysts in the brain parenchyma associated with severe, diffuse inflammation.

Cysticercosis may present with a single intraparenchymal brain lesion with nodular or ring enhancement detected with the use of contrast in neuroimaging studies. The differential diagnosis for this type of lesion includes brain tumors, hydatidosis, multiple sclerosis and tuberculomas. *T. solium* larvae occasionally are deposited in ventricular spaces or in the sub arachnoid space. There, free from surrounding solid tissues, the cyst walls, which develop are often more delicate, and also can grow larger and into more complex shapes. This behavior is described by the term “racemose” cysticercosis⁴⁵⁻⁴⁹.

Pathologic Examination

Detection of the parasite in a biopsy of a brain or spinal cord lesion is one of the proposed absolute criteria for the diagnosis of NCC⁵⁰⁻⁵². Grossly, the lesion shows a fibrous capsule, easily demarcate from adjacent brain tissue, when sectioned is cystic; the cyst contents may be grumous and nondescript, but occasionally have recognizable organism-like shapes. Histologically, cysticerci manifest 4 distinct stages in addition to

Table 1. Diagnostic criteria for definitive or probable diagnosis of Neurocysticercosis*

Level of criteria	Findings
Absolute Major	Pathologic demonstration of the parasite; CT scans or MRI showing a cystic lesion with scolex; direct fundoscopic visualization of parasite. Major Lesions highly suggestive of neurocysticercosis on neuroimaging; positive serum enzyme-linked immunoblot assay for cysticercal antibodies; resolution of cysts after antiparasitic therapy; spontaneous resolution of a small solitary enhancing lesion
Minor	Lesions compatible with neurocysticercosis on neuroimaging; clinical manifestations suggestive of neurocysticercosis; positive cerebrospinal fluid enzymelinked immunosorbent assay for anticysticercal antibodies or cysticercal antigens; cysticercosis outside of the central nervous system
Epidemiologic	Household contact with <i>Taenia solium</i> infection; immigrants from or living in an endemic area; history of frequent travel to disease-endemic areas

* Diagnosis: definitive – one absolute criterion or two major + one minor + one epidemiologic criterion; probable – one major + two minor criteria, or one major + one minor + one epidemiologic criterion, or three minor + three epidemiologic criteria.

the racemose form; vesicular, colloidal, granular and calcified⁵³. The vesicular stage consists of vesicles with viable organisms. Each viable organism is composed of a larva containing an invaginated scolex (head) and is surrounded by translucent fluid that is lined by a thin membranous wall. The reactive inflammatory response and associated changes, which are more likely to correlate with clinical symptoms, are usually not elicited with viable cysticerci but instead are found with degenerating cysts. Depending on the inflammatory process, cysticerci have been documented to survive in the brain for up to several years⁵⁴. The inflammatory response induced by degenerating cysticerci consists predominantly of lymphocytes and plasma cells, with variable edema, gliosis, fibrosis, and necrosis²⁴. The degeneration process begins with the colloidal stage. The vesicle fluid becomes more turbid and the larvae become hyalinized. The inflammatory infiltrate becomes more intense, extending to surrounding structures, and includes lymphocytes, some neutrophils and eosinophils, and the formation of multi-nucleated histiocytic giant cells⁵⁵. The reactive inflammatory process can cause arteritis, arachnoiditis, and ependymitis. The granular-nodular stage involves progressive degenerative changes with increased larval decay, vesicle involution, and thickening of the vesicle wall. The larvae in this stage begin to mineralize with calcium. The degenerative process ends with the nodular-calcified stage. In this stage, cysticerci are replaced by collagen and calcify. Although this stage is associated with decreased inflammation, foreign body giant cells and gliosis may persist. The racemose form of NCC most likely represents cysticercal decay with cyst enlargement and hydropic changes. Racemose cysticerci usually do not contain scolices and are considered nonviable encysted organisms. The degenerated vesicle wall is characteristically convoluted with external bulbous projections and evokes an inflammatory response. The associated reactive process elicited by the racemose form can also cause meningitis.

Other Diagnostic Techniques

Demonstration that antibodies to species-specific antigens of *T. solium* can be detected by EITB assay stimulated investigators to develop highly purified antigens of cysticercus to be used in a reliable immune diagnostic test for cysticercosis⁵⁶. The main weakness of this test is that it may be false-negative in up to 50% of patients with a single cerebral cyst or in those with calcifications alone. Another weakness is that the test may be positive in patients who had been exposed to the adult

parasite without developing cysticercosis. Detection of circulating parasitic antigens using monoclonal antibodies has a poor sensitivity as a screening tool for the diagnosis of NCC; however, antigen detection may be of value to monitor the response to cysticidal therapy⁵⁷. The frequency of positive stool exams for *T. solium* eggs among patients with NCC has varied from one series to another and seems to be related to the severity of infection⁵⁸. Specific coproantigen detection by ELISA and PCR will improve the screening for *T. solium* carriers among healthy individuals from endemic areas⁵⁹.

Epidemiologic factors that may lead to a diagnosis of NCC include evidence of household contact with *T. solium*, immigration from an area where the disease is endemic and a history of repeated travel to disease-endemic regions. An accurate and complete patient history is crucial for NCC identification. Patients should be questioned about recent travel. Immigrants are in high risk for bringing this condition with them from less-developed countries. However, with the increase in world travel, persons who would previously not have been exposed to parasitic illnesses such as NCC are now susceptible⁵². The use of epidemiologic information together with clinical suspicion and laboratory tests is essential in making the final diagnosis and starting treatment. However, the combination of epidemiologic, clinical and laboratory information may lead to an accurate diagnosis and avoid an invasive diagnostic procedure.

NCC should be included in the differential diagnosis of neurologic infections in HIV patients in endemic populations. Consideration of the patient's immune status should alert the clinician to potential atypical presentations. Despite the wide endemicity of cysticercosis and HIV infection, less than 30 cases of NCC have been reported in HIV infected patients⁵¹.

TREATMENT

A single therapeutic approach is not expected to be useful in every patient with NCC. Characterization of the disease in terms of viability of cysts, degree of the host's immune response to the parasite, and location and number of lesions is important for rational therapy⁶⁰. Therapy usually include a combination of symptomatic and cysticidal drugs. Surgery has also a role in the management of some patients⁶¹. Also, utilization of new neurosurgical techniques, such as radioguided surgery, could dramatically improve extent of lesion resection⁶².

Pharmaceutical treatments for symptoms include analgesics, corticosteroids, and antiepileptic medications.

Antiparasitic medications are generally effective but there have been rare treatment-related deaths (1%-4%) in cases with hydrocephalus, intracranial hypertension, and massive parasitic loads^{60,63}.

The introduction and subsequent widespread use of two potent cysticidal drugs (praziquantel and albendazole) have drastically changed the prognosis of most patients with NCC. The initial regimen of praziquantel at doses of 50 mg/kg/day (given every 8 hours) for 15 days was arbitrarily chosen. It was then suggested that if cysticerci are exposed to high concentrations of the drug maintained for up to 6 hours by giving 3 individual doses of 25 to 30 mg/kg at two-hour intervals, this might be sufficient to destroy the parasites. While preliminary results with this new regimen were encouraging, it seems that the single-day course of praziquantel works better for patients with a single parenchymal brain cyst, and that the 15-day trial should be used for those with more than one cyst^{64,65}. Albendazole, the other cysticidal drug, was initially administered at doses of 15 mg/kg/day during one month. Further studies showed that the length of therapy could be shortened to one week without lessening the efficacy of the drug, and even to three days if the patient has a single brain cyst. Albendazole has been superior to praziquantel in trials comparing the efficacy of these drugs. Another advantage of albendazole is that it also destroys subarachnoid and ventricular cysts. In some of these cases, particularly in patients with large subarachnoid cysts, higher doses (up to 30 mg/kg/day) or more prolonged, or even repeated, courses of albendazole may be needed^{66,67}.

Placement of a ventriculoperitoneal shunt is indicated in cases of intracranial hypertension before the use of antiparasitic drugs, and surgical removal may be appropriate for large or intraventricular cysts. Surgery eliminates the inflammatory nidus, potentially obviating the need for a complication prone shunt. Treatment of intraventricular cysticercosis is a contentious issue. Medical management is less favored because of poor cysticidal CSF penetration, delay in response and, lysis of the cyst leading to ependymitis with resultant acute complications and chronic sequelae⁶⁸⁻⁷⁰. Open surgery was the only available modality previously but now neuroendoscopy has become the treatment of choice⁷¹.

Probably the best advice on the treatment of NCC would be not to generalize but to approach and assess each case individually. The selection of a treatment option must include in the consideration of risks and benefits the economic situation of the patient. In areas where NCC is endemic, where most cases are seen, follow-up

neuroimaging examinations may not be performed for economic reasons, and patients may not have access to modern surgical techniques or may be managed in centers where intensive care is not available. The inclusion of long-term outcomes, including the proportion of residual calcifications and the appearance of hydrocephalus, is mandatory to provide a fair evaluation of risk-benefit analysis. The standard of care will change, but still the best option has to be defined.

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