

# Neurocysticercosis: Revisited

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## ABSTRACT

Neurocysticercosis is the most common helminthic central nervous system infection worldwide. Its common clinical manifestations are seizures, headaches and focal neurological deficit. The diagnostic criteria has recently been proposed, based upon clinical manifestations, neuroimaging, histology and serology. Recent advances in neuroimaging and serology facilitate more accurate diagnosis. Many clinical trials have confirmed the safety and efficacy of albendazole in decreasing the burden of parasites and reducing the number of seizures. Corticosteroid can also have some role in certain cases. Overall, seizures can be controlled with one drug, resulting in good prognosis. (*J Infect Dis Antimicrob Agents* 2005;22:27-38.)

## INTRODUCTION

Cysticercosis is caused by the encysted larval stage of the pork tapeworm, *Taenia solium*. Neurocysticercosis (NCC) is well known as the most common helminthic central nervous system (CNS) infection in developing countries, and is the most common cause of symptomatic epilepsy worldwide. Recently, it has become more common in industrialized countries, due to ease of international travel and the high migration rate of people from endemic countries in Latin America, Asia and Africa.<sup>1</sup>

To summarize the tapeworm life cycle, there are three phases including egg, larva, and adult.<sup>2</sup> Eating the egg permits development of the larva in the soft tissues of the intermediate host. Eating such larva-infested tissues allows the adult to form in the intestinal tract of the definitive host, where the egg is then produced and

discharged, allowing the cycle to repeat. Humans are the only known definitive host where the larval form matures into the adult in the small intestine. This human intestinal infection by the adult cestode is called taeniasis. Every few days, several gravid proglottids are released from the distal end of the worm, producing thousands of eggs, which are shed in the stool. The pig is the usual intermediate host (humans can also serve as this type of host) when *T. solium* eggs are ingested rather than the larvae, leading to cysticercosis. Eggs lose their proteinaceous coat in the gastrointestinal tract, pass through the intestinal wall, and lodge in human tissues, predominantly muscle, other soft tissues and the CNS. Individual larvae or cysticerci, when implanted in the brain or its coverings, often produce the symptoms of the CNS infection known as

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NCC. By ingesting *T. solium* eggs, therefore, humans and pigs can serve as intermediate hosts for the larval form of the disease called cysticercosis, of which NCC is a subtype. Ingestion by fecal-oral contamination is the mechanism through which humans acquire NCC. Eggs may be transferred by either direct contact or by ingestion of contaminated food.

### Pathogenesis and pathology

Once situated in tissue, the cysticerci evolve continuously through four important stages. First, there may be diffuse edema as the parasites migrate to the CNS, especially if the number of organisms is high. This stage is mild or inapparent in most patients. Second, a thin-walled cyst containing the fluid and a live larva develops over several months. The parasite escapes the host immune surveillance by secreting a serine proteinase inhibitor, called teniastatin, which inhibits the complement activation, lymphocytic migration and cytokine formation. Third, an inflammatory reaction surrounds and damages the cyst filling with the caseous material while the larva degenerates and dies. This stage is associated with a release of cyst contents and antigens into the cerebrospinal fluid (CSF) and serum. Finally, the cyst itself degenerates, and then is replaced by the fibrotic tissue and becomes mineralized. This results in an inactive calcified nodule.

The presence of the cyst is not always associated with the clinical symptoms. It is only when the cysticerci undergo degeneration that the inflammatory response starts, and the symptoms like seizures occurs. Therefore, the symptoms in NCC may be delayed for several years or the infection may remain subclinical. It appears that the host inflammatory response to the parasite is an important factor that initiates symptoms.

### Clinical manifestations of NCC

NCC is associated with a wide variety of clinical manifestations. These are determined by several important factors including the burden of organisms, the

location of encystment, the stage of cysticerci and the host response to the infection. The location is a critical factor in the symptom development with extraparenchymal cysticercosis (subarachnoid, ventricular and cisternal) often producing more serious disease. There are six main clinical syndromes.<sup>3</sup>

#### 1. Asymptomatic NCC.

It is often seen in endemic area. The cysticerci are in the second stage of development, and escape the host immune response.

#### 2. Parenchymal NCC.

This form occurs when cysticerci develop within the brain, predominantly at the gray-white junction. Seizures are the most common presenting features. Headaches, altered mental status and focal neurologic deficits are also reported relatively frequently with parenchymal NCC.<sup>4</sup>

#### 3. Subarachnoid NCC (cysticercotic arachnoiditis).

Patients usually present with the symptoms and signs of meningitis and increased intracranial pressure (ICP). Headaches, papilledema, optic atrophy, vomiting, coma, dementia and cranial nerve deficits may occur. It may cause the vasculitis. Subarachnoid cysts can grow to enormous sizes, and produce the symptoms relating to the mass effect within the CNS, especially those located at the basilar cisterns.

#### 4. Intraventricular NCC.

This form is frequently found in conjunction with subarachnoid NCC. Intraventricular cysts often cause the CSF obstruction, hydrocephalus and increased ICP.

#### 5. Spinal NCC.

This is rare, but is the most severe form of NCC. It may cause the spinal compression, resulting in paresis, incontinence, sensory deficit, nerve root pain or cauda equine syndrome.

#### 6. Ocular cysticercosis.

The most common location is the subretinal, in proximity to the macula. Other locations may occur such as the anterior chamber, lens and vitreous body.

Although the specific clinical syndromes can be described as above, it is important to remember that many patients may have the mixed forms of NCC, and can have various combinations of signs and symptoms.

A differential diagnosis includes tuberculosis, echinococcosis, paragonimiasis, sparganosis, cryptococcosis and cystic astrocytoma for parenchymal NCC, and echinococcosis, coenurosis, CNS tumors, epidermoids, and arachnoid/colloid cysts for extra-axial NCC.

### New diagnostic criteria

**Table 1. Revised diagnostic criteria for neurocysticercosis.<sup>6</sup>**

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#### Categories of criteria

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##### Absolute

1. Histologic demonstration of the parasite from biopsy of a brain or spinal cord lesion
2. Cystic lesions showing the scolex on CT or MRI
3. Direct visualization of subretinal parasites by funduscopic examination

##### Major

1. Lesions highly suggestive of neurocysticercosis on neuroimaging studies<sup>1</sup>
2. Positive serum EITB<sup>2</sup> for the detection of anticysticercal antibodies
3. Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel
4. Spontaneous resolution of small single enhancing lesions<sup>3</sup>

##### Minor

1. Lesions compatible with neurocysticercosis on neuroimaging studies<sup>4</sup>
2. Clinical manifestations suggestive of neurocysticercosis
3. Positive CSF ELISA for detection of anticysticercal antibodies or cysticercal antigens
4. Cysticercosis outside the CNS<sup>5</sup>

##### Epidemiologic

1. Evidence of a household contact with *T. solium* infection
  2. Individuals coming from or living in an area where cysticercosis is endemic
  3. History of frequent travel to disease endemic areas
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<sup>1</sup> CT or MRI showing cystic lesions without scolex, enhancing lesions, or typical parenchymal brain calcifications.

<sup>2</sup> Enzyme-linked immunoelectrotransfer blot assay using purified extracts of *T. solium* antigens, as developed by the Centers for Disease Control and Prevention of the United States.

<sup>3</sup> Solitary ring-enhancing lesions measuring less than 20 mm in diameter in patients presenting with seizures, a normal neurologic examination, and no evidence of an active systemic disease.

<sup>4</sup> CT or MRI showing hydrocephalus or abnormal enhancement of the leptomeninges, and myelograms showing multiple filling defects in the column of contrast medium. Seizures, focal neurologic signs, intracranial hypertension, and dementia.

<sup>5</sup> Histologically confirmed subcutaneous or muscular cysticercosis, plain X-ray films showing "cigar-shaped" soft-tissue calcifications, or direct visualization of cysticerci in the anterior chamber of the eye.

ELISA: enzyme-linked immunosorbent assay, CNS: central nervous system, CT: computed tomography, MRI: magnetic resonance imaging

In 1996, the first diagnostic criteria for cysticercosis was proposed, based on the objective evaluation of clinical, radiological, immunological and epidemiological data.<sup>5</sup> However, the criteria is very complex, and the specificity has been questioned. Therefore, during the consensus meeting in Peru, the working group has revised and simplified the criteria, shown in Tables 1 and 2.<sup>6</sup> This criteria will standardize the diagnosis and may aid in future clinical studies.

### Diagnostic tools

In approximately 50 percent of cases, a CSF analysis shows pleocytosis (often lymphocytic, but

**Table 2. Revised degrees of certainty for the diagnosis of neurocysticercosis.<sup>6</sup>****Diagnostic Certainty****Definitive**

1. Presence of one absolute criterion
2. Presence of two major plus one minor and one epidemiologic criterion

**Probable**

1. Presence of one major plus two minor criteria
2. Presence of one major plus one minor and one epidemiologic criterion
3. Presence of three minor plus one epidemiologic criterion

The presence of two different lesions highly suggestive of neurocysticercosis on neuroimaging studies should be considered as two major diagnostic criteria. However, positive results in two separate types of antibody detection tests should be interpreted only on the basis of the test falling in the highest category of diagnostic criteria.

occasionally eosinophilic profile), decreased glucose, increased protein and elevated opening pressure. A lumbar puncture can help exclude other infectious or malignant diagnosis.

The development of improved immunodiagnostic tools has contributed to our knowledge on the importance of taeniasis/cysticercosis by enabling seroepidemiological surveys and community-based studies to be carried out. As serologic testing is becoming an important diagnostic criteria, there are several methods of testing to be used in various settings. Enzyme-linked immunosorbent assay (ELISA) is a simple and rapid test for the detection of cysticercus antibodies in the serum. For example, the antigen used in one study is a complete homogenate of cysticercus cellulosa cysts obtained from infected pigs and dotted onto a nitrocellulose membrane. This simple dot-ELISA test showed a high sensitivity of 56 percent and specificity of 92 percent.<sup>6</sup> Dekumyoy and colleagues evaluated the indirect ELISA method in Thai population, and found a 90 percent sensitivity and 86 percent specificity. There was a cross-reactivity with echinococcosis and gnathostomiasis using this ELISA.<sup>8</sup>

Enzyme-linked immunoelectrotransfer blot assay (EITB), using purified extracts of *T. solium* antigens,

was developed by the Centers for Disease Control and Prevention of the United States to detect the specific antibodies. It may be more convenient to confirm the diagnosis in suspected cases. The specificity and sensitivity are very high for pediatric patients with more than two lesions. But the sensitivity is only moderate in those with one lesion. It was found that almost all CSF samples are positive for anti-*T. solium* IgG using ELISA or EITB method. The use of these techniques could improve the immunodiagnosis of the vesicular stage of NCC, and allow better evaluation of NCC cases both pre- and post-treatment.<sup>9</sup> Monoclonal-antibody-HP10-antigen-trapping ELISA, which has been used successfully to detect viable *T. solium* cysticercosis, was used to study the CSF of NCC patients in Mexico. The sensitivity was higher in cases of inflammatory disease, compared with non-inflammatory disease, and in cases of multiple-cyst cysticercosis, compared with single-cyst cysticercosis.<sup>10</sup>

However, because there is no standardized immunodiagnostic test, the potential use of immunodiagnostic tools to identify cases of NCC in man without neuroimaging is subject to debate. The correlation between a positive serology and the neurological symptoms and/or the lesions indicative for NCC on the neuro-imaging techniques is poor-to-fair in most studies. This may

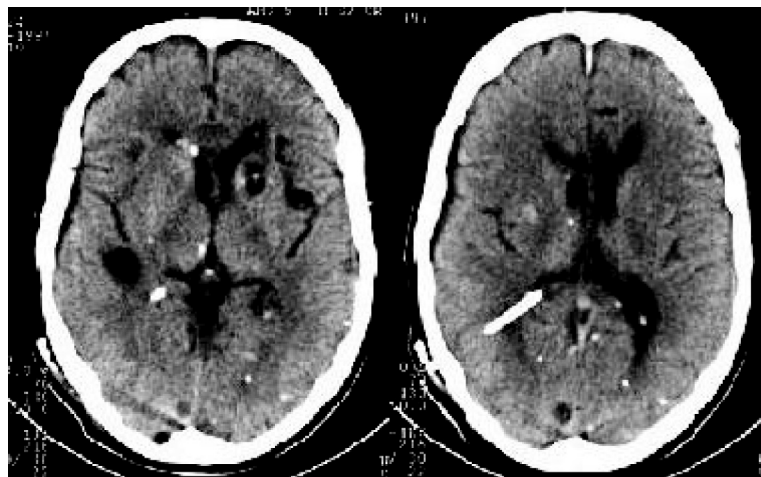
be explained by the unpredictable clinical outcome of the infection and the variable immunological response of the host to the infection.<sup>11</sup> Another major problem is that in many developing countries, the neuroimaging methods are inaccessible and/or too expensive for the rural population at risk. Under these conditions, serologic testing may be the only available diagnostic tool.

### Neuroimaging

Computed tomography (CT) will show the cyst and granuloma stages of NCC. These cysts can be solitary or multiple and usually are 5-20 mm in diameter. Over half of children who are affected with NCC have a solitary lesion. The lesions locate most often in the

cortex or at the gray-white junction. Approximately one half of the lesions have a punctate high density within the ring (scolex). CT is superior to magnetic resonance imaging (MRI) study in detecting calcification, which can be useful in differentiating the punctate cyst of NCC in the granuloma wall from other causes of granulomas. However, calcification is observed less frequently in children than in adults. CT can also detect the edema around the cyst, which is associated with the death of the organism (Figure 1).

MRI is the best imaging test overall for the diagnosis due to its high sensitivity and image resolution. MRI is useful to detect lesions of the spinal cord, posterior fossa, brainstem, subarachnoid and ventricles. Gadolinium contrast may help in increasing diagnos-



**Figure 1.** CT demonstrated multiple lesions with variable characteristics. Neurocysticercosis was confirmed by CSF study.

These images were representative of different stages co-existing: calcification with no edema, calcification with a cystic cavity and active inflammatory lesion with edema, and no calcification. A scolex was visible in the lesion near the head of the left caudate.

This patient had a severe hydrocephalus due to obstruction of

the cerebral aqueduct by a cysticercus (not shown), and a VP shunt was placed in the right lateral ventricle.

(From <http://www.medstudents.com.br/image/neuro/cystic/>

tic yield. It will also show the larval death, visible as enhancement of the cyst wall, which indicates that the cyst has changed into a granuloma. In addition, MRI (as well as CT) can show vasogenic edema around the cyst which is indicative of the inflammatory response to the organism death. MRI can be used as a follow-up imaging to document an improvement based on both a decrease in the granuloma diameter and a resolution of vasogenic edema (Figure 2).

Neuroimaging is very helpful in the diagnostic process. While MRI is more sensitive and specific than CT, but it is offset by the high cost and unavailability in certain hospitals. MRI can also assist in differentiating various forms of unusual manifestations of NCC.<sup>12</sup> Chawla and colleagues studied the correlation of the MRI findings and histopathology in swine NCC. A T2-weighted MRI demonstrated all viable cysts identified by histopathology. With MRI, non-enhancement of some early degenerated cysts along with the absence of edema is likely to underestimate the stage of NCC evolution, and these cysts may be misdiagnosed as the viable cysts.<sup>13</sup>

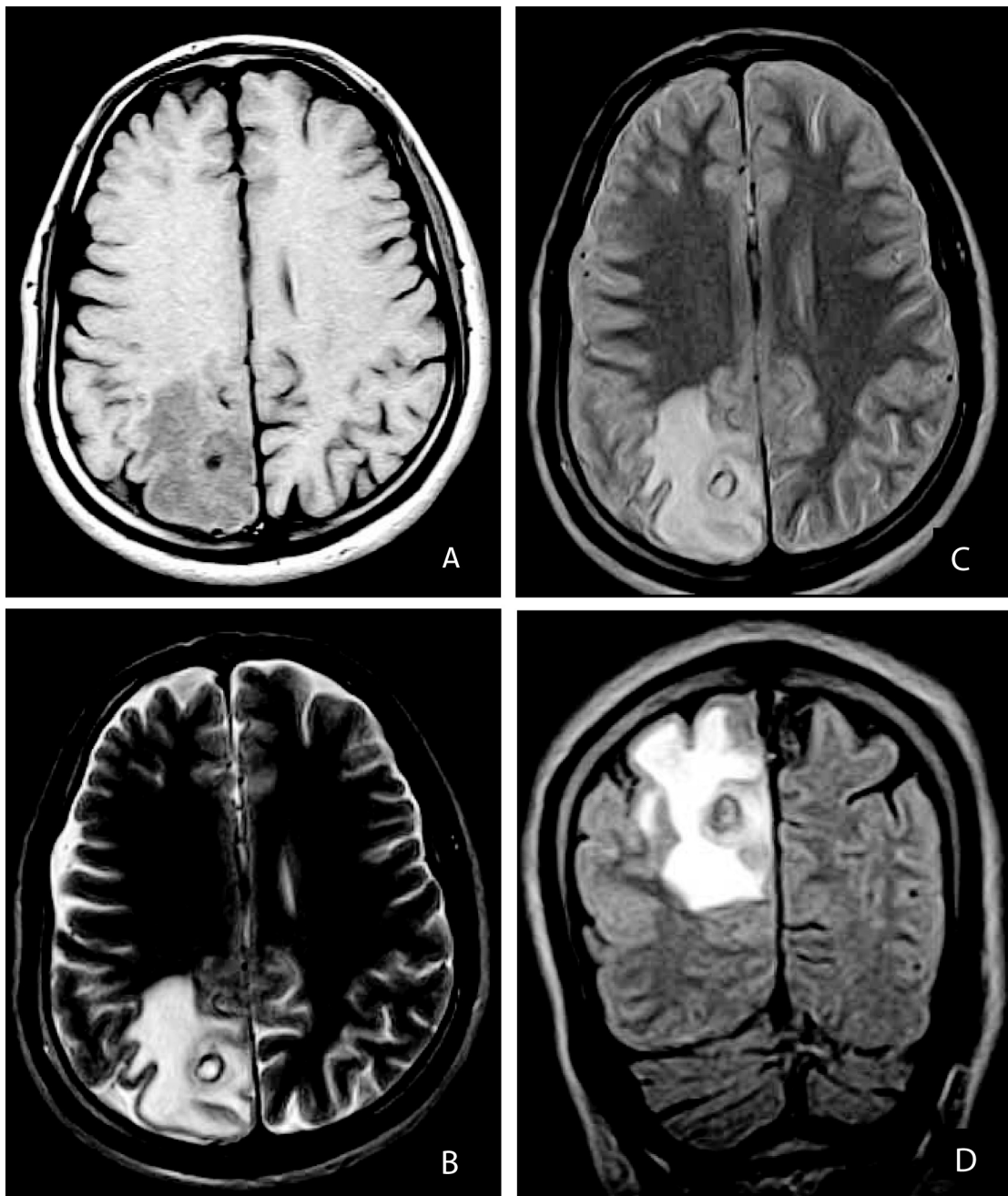
Plain radiography of the soft tissues may show classical cigar-shaped calcifications, but they rarely are helpful due to their low yield. Skull radiography can also be performed, even though they rarely are helpful with the advent of CT or MRI. A separation of the cranial sutures can be occasionally observed in children with increased ICP.

### Seizures and neurocysticercosis

At present, there is overwhelming evidence supporting NCC as a cause of seizures and epilepsy. Although seizures are the main clinical manifestations of parenchymal NCC, recent studies emphasize that such seizures are the result of the host inflammatory response, even in patients who only have calcifications with no viable cysticerci.<sup>14,15</sup> The most common form of the disease, chronic calcific NCC, is the end result

of the host inflammatory response to the larval *T. solium* cysticercus. Therefore, small punctate and single or multiple calcifications are common in *T. solium* endemic populations, and most of them are calcified cysticercal granulomas. These lesions are not clinically inactive because they are also a cause of seizures and focal symptoms in this population. Perilesional edema is present at times around implicated calcified foci.<sup>15</sup> Solitary cysticercus granuloma is also a cause of seizure<sup>16</sup>, and can become an epileptic focus. It is not known whether the presence of calcium in a lesion is solely a visual marker of past or present pathology, or if it plays a direct or indirect role in the induction of seizures. Direct calcium toxicity has also been suggested. Calcium may form an insoluble matrix that could release incorporated antigens at certain times. Direct injury to brain tissues associated with single calcified or non-calcified cysticercal granuloma is another possible reason for continued or recurrent seizure activity.<sup>15</sup>

Prognosis for seizure recurrence in patients with newly diagnosed NCC is dependent on active brain lesions. Seizure recurrence is high after the first acute symptomatic seizure, related to persistence of active brain lesions. Recurrence risk is low in those in whom the NCC lesion clears, comparable to seizure risk following other brain insults leading to a static encephalopathy. Patients with NCC should receive antiepileptic medication (AED) until the acute lesion clears on CT.<sup>17</sup> Overall recurrence risk was 40 percent at one year. Therefore, seizures in the context of edema and a degenerative lesion should be considered acute symptoms, even if they occur many months after presentation. It is appropriate to monitor cyst activity with CT and continue AED until resolution of the acute lesion. After this time, AED may be discontinued. Seizure recurrence among those with cyst resolution was about 22 percent which is in line with other structural brain abnormalities and acute symptomatic seizures.<sup>17</sup>



**Figure 2.** (A) The T1-weighted MRI revealed a well circumscribed lesion that was isointense to CSF. It was hyperintense to CSF with a peripheral rim that was isointense to white matter on the Proton, T2-weighted (B and D) and FLAIR (C) MRIs. An eccentric speck that was isointense to white matter was seen within this lesion on the FLAIR images and would represent the scolex. Perilesional edema was noted. This lesion represented a cysticercus in the colloidal vesicular stage.

(From <http://www.mribhatia.com/braintf7/index.html>. Accessed on March 30, 2005)

Nearly 85 percent of the patients with a solitary cerebral cysticercus granuloma have a good seizure outcome following resolution of the lesion and early withdrawal of AEDs. However, recurrence of seizures can be expected in about 15 percent of patients. Patients with more than two seizures, those with breakthrough seizures, and those whose follow-up CT scan shows a calcific residue of the granuloma have a higher risk of recurrence, and therefore need to be appropriately cautioned after withdrawal of AEDs. AED therapy might also have to be continued for longer periods in patients with these risk factors.<sup>18</sup>

The clinical manifestation of NCC in children is slightly different from adults. Most of them presented with partial seizures. Single enhancing lesions are commonly seen in neuroimaging studies. Corticosteroids are often indicated in those with cerebral edema. However, prognosis is quite good and seizures are mostly controlled with one AED. Seizure recurrence is low except in those with multiple lesions.<sup>19</sup>

### **Treatment and recent evidence from clinical trials**

Usually, NCC is treated with antiparasitic drugs along with symptomatic therapy. Patients with inactive parenchymal NCC with evidence of calcified lesions or degenerating parasites on neuroimaging do not require antiparasitic treatment. As seizures are common symptoms in these patients, chronic anticonvulsant therapy is required. Patients with inactive disease and hydrocephalus due to prior subarachnoid or ventricular infection also do not require antiparasitic treatment, but ventriculoperitoneal shunt may be required. Shunt failure is uncommon in this group.<sup>20</sup>

Previous meta-analysis showed that there is insufficient evidence to determine whether cysticidal therapy is of any clinical benefit to patients with NCC. But it does not exclude the possibility that more patients remain seizure-free when treated with cysticidal drugs.<sup>21</sup>

Therefore, a large, double-blinded randomized placebo-controlled trial was conducted to compare 800 mg of albendazole and 6 mg of dexamethasone per day for 10 days with two placebos, to treat patients with viable parenchymal cysts. Antiparasitic therapy was able to decrease the burden of parasites. The treatment group had the same number of partial seizures as the placebo group but the number of seizures with generalization was reduced in the treatment group. The treatment was safe and effective.<sup>22</sup>

Therefore, patients with active parenchymal disease should be treated with albendazole, a benzimidazole antihelminthic agent, or praziquantel. Albendazole is preferred because it is cheaper, has better penetration into subarachnoid cysts and is unlikely to have pharmacological interference with corticosteroids and other AEDs. The dose of albendazole is 15 mg/kg/day divided in two oral doses for 8 to 28 days along with dexamethasone.

One small trial has assessed the efficacy of albendazole (15 mg/kg/day for one week) plus oral prednisone (1 mg/kg/day), compared with one-day course of praziquantel (100 mg/kg in three divided doses at 2-hour intervals) plus two 8-mg doses of intravenous dexamethasone for therapy of parenchymal NCC. Although the total number of cysts was significantly reduced from 64 to 7 in patients treated with albendazole and from 59 to 24 in those treated with praziquantel, the number of patients improving with albendazole was not significantly different from those treated with praziquantel.<sup>23</sup> Administration of dexamethasone a few hours after giving praziquantel allows the uptake of the drug by the cyst.

The adverse effects of antiparasitic drugs are a worsening of neurological status (headache, vomiting, dizziness, seizures, coma and increased ICP), and are believed to be due to host inflammatory response against dying parasites. Many cysts resolve spontane-

ously over time.

There is no consensus regarding treatment of active extraparenchymal NCC. Until recently, surgical removal of intraventricular NCC was done with or without ventriculoperitoneal shunt. Ventriculoperitoneal shunt in this group is complicated with frequent shunt failures. Neuroendoscopic removal of intraventricular NCC as an alternative method is less invasive. In patients with single ring-enhancing CT lesion presenting with seizures, treatment with AED alone is advocated as most of these resolve spontaneously. Treating them with anthelmintic agents does not improve the resolution of these lesions.<sup>20</sup>

Surprisingly, the clinical trial results are different in children. The frequency of healing of CT lesions in the albendazole and placebo groups are similar, as well as the seizure-free rate. Therefore, the treatment was not beneficial in NCC in children with ring-enhancing lesions in CT.<sup>24</sup> Another randomized trial studied the efficacy of corticosteroids, albendazole or both of them in children with focal seizures who had single small enhancing CT lesions. There was no significant difference in resolution of CT lesions in the three groups at three and six months of follow-up. Moreover, children in the corticosteroid group had significantly more seizure recurrence while on the AED.<sup>25</sup>

However, in children who had one or two ring-enhancing CT lesions, albendazole (15 mg/kg/day for 28 days) plus dexamethasone (0.15 mg/kg/day for 5 days) increased the complete or partial resolution of lesions and reduced the risk of subsequent recurrence of seizures.<sup>26</sup> Regarding the duration of treatment, one-week therapy was as effective as four weeks of albendazole treatment in the resolution of lesions and seizure control in children with NCC who had one-to-three lesions.<sup>27</sup> This discrepancy is likely due to the age of patients, number of lesions and treatment regimen.

A short course of oral prednisolone (1 mg/kg/day

for 10 days, followed by tapering over the next four days) has been studied in an open-label randomized trial in patients who already received AED. When compared with the AED alone, prednisolone plus an AED help in the rapid resolution of solitary cysticercus granuloma in patients with new-onset seizures. The resolution of lesions is associated with improved seizure-related prognosis.<sup>28</sup>

### Prevention

Improving sanitation, elimination of intestinal tapeworms, improving sewage disposal system, surveillance of pork farming, preventing pigs from entering human dwellings and consuming properly cooked clean vegetables and pork are some of the methods to prevent the occurrence of NCC.<sup>20</sup>

### CONCLUSION

NCC has diverse clinical manifestations; seizures are the most common. It is the major cause of epilepsy worldwide. Treatment should be individualized based on the location, number of cysticerci and host response. Antiparasitic therapy is recommended in patients with active or multiple lesions, but not in calcified lesion. In certain cases, a short course of corticosteroid may help to minimize the host reaction against dying parasites. The seizures from NCC are generally easy to control and have similar prognosis to other structural brain lesions.

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