Published in final edited form as:

Curr Opin Infect Dis. 2011 October ; 24(5): 423–427. doi:10.1097/QCO.0b013e32834a1b20.

Cysticercosis of the Central Nervous System - How Should It Be Managed?

Hector H. Garcia, MD, PhD, Armando E. Gonzalez, DVM, PhD, and Robert H. Gilman, MD, DTMH for The Cysticercosis Working Group in Peru

Abstract

Purpose of review—*Taenia solium* neurocysticercosis has been long recognized as an important cause of neurological morbidity in most of the world. Unwarranted generalization of diagnostic and treatment recommendations made it difficult to assess individual prognosis and responses for each type of NCC. Understanding of the main clinical presentations (dependent on number, location, size and stage of parasites, as well as on the immune response of the host) allows a better view of treatment options and expected outcomes.

Recent findings—Current treatment options are still limited and involve symptomatic agents, antiparasitic agents or surgery. The importance of adequate symptomatic management, the potential for improved antiparasitic treatment regimes, in particular combination therapy, and the increasingly important role of minimally invasive neurosurgery are also revised in this manuscript.

Summary—Treatment decisions in NCC should be individualized in relation to the type of NCC. Initial measures should focus on symptomatic management to later consider antiparasitic therapy when appropriate. Appropriate patient categorization, new antiparasitic regimes and minimally invasive surgery are improving the prognosis of patients with NCC.

Keywords

Taeniasis; cysticercosis; *Taenia solium*; treatment; albendazole; praziquantel

INTRODUCTION

Taenia solium, the pork tapeworm, is endemic in most of the world, including most developing countries where pigs are raised. Infection of the human nervous system by *T. solium* larvae (neurocysticercosis) is a very common event in endemic regions, where symptomatic NCC accounts for approximately one third of seizure disorders.[1-5] and contributes to other neurological disease.[6]

Treatment of NCC is not a simple task. The multiple possible combinations of numbers of lesions, location (involving its relationship with surrounding structures, key in neurological pathology), size, involutive stage, and associated inflammation, open a wide array of clinical pictures.[7] Treatment, however, faces a surprising scarcity of therapeutic tools: only two antiparasitic drugs are of use, anti-inflammatory treatment is mostly restricted to steroids,

Corresponding author Hector H. Garcia, MD, PhD. Professor, Department of Microbiology and Director, Center for Global Health - Tumbes, Universidad Peruana Cayetano Heredia, Lima, Peru; Head, Cysticercosis Unit, Instituto Nacional de Ciencias Neurológicas, Lima, Peru. H. Delgado 430, SMP, Lima 31, Peru. Telephone +511 3287360, hgarcia@jhsph.edu.

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and only recently minimally invasive surgery added to open surgery and shunt placement as surgical options. We will review here the main concepts on disease pathogenesis and treatment, and critically revise existing therapeutic options.

THE INFECTION AND THE DISEASE

As per many helminths, the life cycle of *T. solium* involves a definitive host, a carnivore, who ingests the larvae located in the flesh of the intermediary host. These larvae develop as adult tapeworms (usually one in the case of *T. solium*) in the intestines of the definitive host and produce infective eggs which are disseminated with the stools into the environment, to be ingested by a suitable intermediary host (a herbivore). After ingestion, the embryos in the eggs are liberated, cross the intestinal wall, reach the circulatory system, and are dispersed to the tissues where they develop in cystic larvae or cysticerci. The cycle closes when the larvae-infected flesh of the intermediate host is eaten by a carnivore predator.[8]

In the usual cycle of *T. solium*, man act as definitive host, harboring the tapeworm, and pigs act as intermediate hosts. Unfortunately, man can also become infected with cysticercosis by ingesting tapeworm eggs via fecal oral contamination. Resulting cysts may locate in almost any organ or tissue. In most locations they will not be noticed, but when cysts locate in the nervous system or the eye, symptoms may be not only noticeable but can produce severe and incapacitant disease, some times lethal.[9]

Human NCC infection in endemic regions seems to be a very common event. Population serosurveys find up to 20 to 25% of the entire population with specific antibodies to *T. solium*, and CT scan surveys in healthy population find between 10 and 20% of them with brain calcifications likely representing resolved cysts.[1,3,4] Each of these provide only a partial assessment of infection burden in a population, since many infected individuals resolve the infection and become seronegative, and of course people with brain cysts are only a sub-set of all infected individuals.

Whether NCC is or not a major contributor to seizures and other neurological morbidity has been questioned by some authors on the basis of the many asymptomatic infected individuals described above. However, evidence supports a major impact in the prevalence of seizure disorders. First, individuals with brain cysticercosis are a sizable subset of patients with seizures in neurological centers in endemic countries. Even in population studies, approximately 30% of cases of seizures seem to be attributable to NCC.[1,2] Furthermore, treatment of viable brain cysts with antiparasitic agents results in local inflammation around the lesion and may reproduce or exacerbate the same symptomatology (hence the use of steroids), providing additional evidence of the relationship between symptoms and lesions. [10]

Another subject of discussion is the “transient” nature of symptoms in NCC. Patients with only viable brain cysts seem to have few symptoms, which increase at the time of the degeneration of at least one of the cysts, and then becomes less symptomatic after the parasite resolves. However, many individuals persist with viable cysts for decades, and calcified lesions may also be symptomatic decades after the parasite died. This may not however be the case in individuals with a single degenerating brain nodule as seen in India, where seizure relapses are way less common.[11]

So, while there are many asymptomatic infected individuals with NCC in population settings, the proportion which become symptomatic is large enough to contribute to the burden of neurological disease in endemic regions, aimed by the chronicity of the disease. [10]

TYPES OF NCC AND THEIR PARTICULARITIES

In order to understand the general treatment directions and particular approaches in NCC, one needs first to be considered that NCC may result in many different clinical presentations, depending on number, location, and stage of the lesions. This had been mentioned in the old British literature,[12,13] but became particularly clear after the introduction of CT. As early as 1986, Estanhol [14] and others differentiated between “benign” and “malignant” NCC on the basis of its intra- or extraparenchymal location, which in general lines reflects very well a marked difference in evolution and prognosis. Most recent advances in imaging and syndromic characterization led us to better understand the spectrum of clinical presentations.

Intraparenchymal Brain Cysticercosis

Parenchymal disease is mostly characterized by seizures. Invasive embryos reach the brain through terminal arteries and grow in the grey matter. By growing towards the inside, they become surrounded by brain parenchyma and grow as rounded, cystic, fluid filled vesicle structures until its usual size of one to two centimeters. Cysts in the brain cortex may actually grow towards the surface and subarachnoid space of the convexity of the brain, but they still behave as intraparenchymal lesions. Intraparenchymal parasites follow an involutionary course: after surviving for years as viable cysts, they are detected and attacked by the host's immune system, lose its survival equilibrium and die. Their contents become gradually cloudy and colloidal, the cysts shrink, and finally resolve to leave a residual calcified scar.[15]

A common presentation of intraparenchymal NCC, particularly frequent in the Indian subcontinent (where most cases of NCC present like this) is a single, degenerating intraparenchymal brain cyst. It presents at younger ages and carries a very benign prognosis. [16,17]

Other less frequent presentations are *cysticercotic encephalitis* where multiple (hundreds) small intraparenchymal cysts are inflamed causing severe intracranial hypertension,[18] and *massive, non-encephalitic cysticercosis*, where again hundreds of intraparenchymal cysts survive with minimal evidences of inflammation, mostly causing occasional seizures. [19]

Extraparenchymal Neurocysticercosis

Parasitic larvae located in the subarachnoid space or in the basal cisterns follow a progressive course with growth and invasion of neighboring spaces. In this process they loose its form and become membranous extensions; this infiltration commonly results in obstructive hydrocephalus and intracranial hypertension, compromising the life of the patients.[20] Cysts in the ventricles can directly block the flow of the cerebrospinal fluid also resulting in hydrocephalus. Parasites located in the Sylvian fissure tend to grow disproportionately and may reach several centimeters, behaving as a benign, slow growing tumoral mass.[21]

As a gross summary, intraparenchymal NCC usually follow a bening course and is characterized by seizures; while extraparenchymal NCC is progressive, associated with intracranial hypertension, and some times lethal. The later is also very difficult to treat requiring mutiple courses of antiparasitic therapy (see below).

MAIN LINES AND GOALS OF TREATMENT

Symptomatic NCC cases are usually individuals with seizures, headaches, intracranial hypertension, or other neurological symptoms. Management involves adequate symptomatic measures and, when appropriate, anti-parasitic treatment or surgery. Anti-parasitic therapy in NCC is never an urgent action and it seems clearly appropriate to wait until the patient is stable and without symptoms to initiate antiparasitic treatment. Appropriate symptomatic management should be instituted first. Symptomatic therapy is key and involves analgesics, antiepileptic drugs, anti-inflammatories (mostly steroids), and management of intracranial hypertension when present.[10]

In patients with seizures secondary to NCC, antiepileptic drug (AED) therapy should be administered as for any other cause of epilepsy. Seizures usually respond very well to first-line AED monotherapy with either phenytoin, carbamazepine or valproate.[22] Increased intracranial pressure arises from perilesional edema (in which case it usually responds well to oral corticosteroids, which may be required long term), or obstructive hydrocephalus. In the later, the insertion of a ventriculoperitoneal shunt may be required. Shunt obstruction is a common event because of the parasite cellular debris, and the increased protein levels in CSF which are usual in basal subarachnoid NCC.[23] Concomitant use of corticosteroids has been reported to decrease the frequency of shunt obstruction.[24]

Antiparasitic agents, either albendazole (15 mg/kg/d p.o. for 8 to 15 days) or praziquantel (50 to 75 mg/kg/d p.o. for 15 days) are effective in killing live cysticerci. Albendazole is currently the drug of choice because of slightly greater efficacy, better availability and lower cost.[25,26] A short course of three doses of 75-100 mg/kg of praziquantel in the same day (every two hours) was reported in Mexico in 1996,[27] but its efficacy appear to be lower in patients with multiple cysts.

Anti-parasitic treatment of patients with viable or degenerating cysts seems associated with better seizure evolution.[25,28] On the other hand, the use of antiparasitic treatment in NCC was for long time a matter of intense controversy. Between the second and the fifth days of anti-parasitic therapy, patients usually have an exacerbation of neurologic symptoms, attributed to local inflammation due to the death of the larvae. Apparently, the destruction of live parasite cells exposes antigens and triggers an intense local inflammatory reaction which exacerbates symptoms and can be lethal because of intracranial hypertension. For this reason, anti-parasitic treatment should be given under hospital conditions, simultaneously with corticosteroids to control edema and intracranial hypertension.[10] Coadministration of corticosteroids reduces blood levels of praziquantel;[29] however this is not thought to be clinically relevant at usual doses of administration.

The partial efficacy of existing antiparasitic regimes (only 60-70% of cysts die, and only 30-40% of patients are free of viable brain cysts after an initial course of treatment) [25,26,28] forces the search for better regimes. Increased doses of ABZ, up to 30 mg/kg/d, have been tried with success by some groups,[30,31] but the lack of a thorough safety assessment in the published literature conveys caution with this approach, since ABZ is associated with some frequency with increased liver enzymes, usually transient, and decreased blood cell counts. More recently, the combined use of ABZ and PZQ for NCC has been reported and seems a very promising alternative.[32-34] A large trial comparing ABZ and combined ABZ+PZQ is currently under way in Peru.

The use of steroids along anti-parasitic treatment is also poorly studied. Published regimes vary enormously in terms of the drug of choice, doses, and length of treatment. Most authors use a moderate amount of dexamethasone (usually 0.1 mg/kg/d) from the day before onset of antiparasitic treatment until the end of antiparasitic treatment.

The role of surgery was for long time limited to shunt placement and open craniotomies to excise cysts from the ventricles, Sylvian fissure, or more rarely, large cysts in other locations, with the risks and drawbacks of open cranial surgery.[35] The advent of minimally invasive neurosurgery (neuroendoscopy) has greatly changed this view and now the recommended management of intraventricular cysts is neuroendoscopic extraction, and fenestration of the anterior wall of the IIIrd ventricle during the same procedure may save the need for a shunt, although no controlled data is yet available for this claim.[36] Its use for other NCC locations as those in basal cistern lesions has been reported but more extensive experience seems required before assessing risks and benefits.

ANIMAL MODELS FOR TREATMENT

Naturally infected pigs can be purchased with some facility in endemic countries and provide a suitable treatment model.[37] Infected pigs are identified by peasants by palpation of the lower surface of the animal's tongue, where cysts frequently locate. This simple screening method detects heavily infected animals and thus the chance of these having brain cysts is higher.[38] Drawbacks of this model include inter-animal variability in cyst numbers, and lack of certainty on whether degenerating cysts are due to treatment or whether they were already involuting before therapy.

Experimental infections in pigs can be done orally,[39] or by local injection of infective oncospheres (intramuscular oncosphere assay, IMOA).[40] The oral model has a much more variable cyst yield, and the IMOA assay does not follow the natural intestinal route.

Comparative treatment trials can be performed in naturally or experimentally infected pigs by adequate group allocation, enough sample size to detect efficacy differences, and appropriate logistic procedures including separating groups to avoid ingestion of the other drugs after excretion in stools. Cyst degeneration is not immediate but results from the attack of the pig cellular immune system[41] and thus a period of several weeks after treatment should be allowed to evaluate the effects of the tested therapies.

PREVENTION AND CONTROL

Beyond the standard measures to avoid fecal oral contamination and appropriate pig raising, active control interventions have been tried with variable results, mostly using mass human chemotherapy. A large scale elimination program in Peru found no transmission in 101 of 104 intervened villages after one year with a combined scheme (CWGP 2011, unpublished data) using repeated rounds of mass human chemotherapy with niclosamide followed by identification of taeniasis cases with coproantigen detection, and mass porcine chemotherapy with oxfendazole, plus vaccination of pigs with TSOL18, a highly effective vaccine produced at the University of Melbourne in Australia.[39,42] From here onwards, the control tools used should be made affordable and less costly, and tested in different geographical and cultural scenarios towards the goal of widespread elimination and potential eradication.

CONCLUSION

Despite its importance as a cause of neurological disease in most of the world, treatment of NCC faces a notable scarcity of tools and controlled efficacy data. Antiparasitic treatment seems of benefit in most cases but its efficacy is sub-optimal. Combining albendazole and praziquantel may improve cysticidal efficacy without compromising safety. In the surgical side, neuroendoscopy appears as the most important advance in NCC management.

Acknowledgments

HG is now a Wellcome Trust Senior International Research Fellow.

REFERENCES

1. Montano SM, Villaran MV, Ylquimiche L, Figueroa JJ, Rodriguez S, Bautista CT, Gonzalez AE, Tsang VC, Gilman RH, Garcia HH. Neurocysticercosis: association between seizures, serology, and brain CT in rural Peru. *Neurology*. 2005; 65:229–233. [PubMed: 16043791]
- * 2. Goel D, Dhanai JS, Agarwal A, Mehlotra V, Saxena V. Neurocysticercosis and its impact on crude prevalence rate of epilepsy in an Indian community. *Neurol India*. 2010; 59:37–40. [PubMed: 21339656] [Large door to door epilepsy study showing the proportion of NCC-associated cases.]
- * 3. Medina MT, Aguilar-Estrada RL, Alvarez A, Duron RM, Martinez L, Dubon S, Estrada AL, Zuniga C, Cartagena D, Thompson A, et al. Reduction in rate of epilepsy from neurocysticercosis by community interventions: The Salama, Honduras Study. *Epilepsia*. 2010 [Secular decrease in seizure cases, particularly in NCC-associated seizures, following a community intervention.]
4. Del Brutto OH, Santibanez R, Idrovo L, Rodriguez S, Diaz-Calderon E, Navas C, Gilman RH, Cuesta F, Mosquera A, Gonzalez AE, et al. Epilepsy and neurocysticercosis in Atahualpa: a door-to-door survey in rural coastal Ecuador. *Epilepsia*. 2005; 46:583–587. [PubMed: 15816956]
5. Ndimubanzi PC, Carabin H, Budke CM, Nguyen H, Qian YJ, Rainwater E, Dickey M, Reynolds S, Stoner JA. A systematic review of the frequency of neurocysticercosis with a focus on people with epilepsy. *PLoS Negl Trop Dis*. 2010; 4:e870. [PubMed: 21072231]
- * 6. Ciampi de Andrade D, Rodrigues CL, Abraham R, Castro LH, Livramento JA, Machado LR, Leite CC, Caramelli P. Cognitive impairment and dementia in neurocysticercosis: a cross-sectional controlled study. *Neurology*. 2010; 74:1288–1295. [PubMed: 20404310] [Controlled study demonstrating a high frequency of cognitive impairment in NCC]
- ** 7. Mahanty S, Garcia HH. Cysticercosis and neurocysticercosis as pathogens affecting the nervous system. *Prog Neurobiol*. 2010; 91:172–184. [PubMed: 20035822] [Comprehensive review on NCC pathogenesis and manifestations]
8. Flisser A. Taeniasis and cysticercosis due to *Taenia solium*. *Prog Clin Parasitol*. 1994; 4:77–116. [PubMed: 7948938]
9. Garcia HH, Del Brutto OH. Neurocysticercosis: updated concepts about an old disease. *Lancet Neurol*. 2005; 4:653–661. [PubMed: 16168934]
10. Nash TE, Singh G, White AC, Rajshekhar V, Loeb JA, Proano JV, Takayanagui OM, Gonzalez AE, Butman JA, DeGiorgio C, et al. Treatment of neurocysticercosis: current status and future research needs. *Neurology*. 2006; 67:1120–1127. [PubMed: 17030744]
11. Garcia HH, Gonzalez AE, Rodriguez S, Tsang VC, Pretell EJ, Gonzales I, Gilman RH. Neurocysticercosis: unraveling the nature of the single cysticercal granuloma. *Neurology*. 2010; 75:654–658. [PubMed: 20713953]
12. McArthur WP. Cysticercosis as seen in the British army with special reference to the production of epilepsy. *Trans R Soc Trop Med Hyg*. 1934; 27:343–363.
13. Dixon, HB.; Lipscomb, FM. Cysticercosis: an Analysis and Follow-up of 450 cases. Office HMsS., editor. Vol. vol 299. Medical Research Council; London: 1961.
14. Estanol B, Corona T, Abad P. A prognostic classification of cerebral cysticercosis: therapeutic implications. *J Neurol Neurosurg Psychiatry*. 1986; 49:1131–1134. [PubMed: 3783174]
15. Escobar, A. The pathology of neurocysticercosis. In: Palacios, E.; Rodriguez-Carbajal, J.; Taveras, JM., editors. Cysticercosis of the central nervous system. Charles C. Thomas; 1983. p. 27–54.
16. Rajshekhar V. Etiology and management of single small CT lesions in patients with seizures: understanding a controversy. *Acta Neurol Scand*. 1991; 84:465–470. [PubMed: 1792850]
17. Rajshekhar, V.; Chandy, MJ. Solitary cysticercus granuloma: the disappearing lesion. Orient Longman; Chennai: 2000.
18. Rangel R, Torres B, Del Bruto O, Sotelo J. Cysticercotic encephalitis: a severe form in young females. *Am J Trop Med Hyg*. 1987; 36:387–392. [PubMed: 3826497]

19. Garcia HH, Del Brutto OH. Heavy nonencephalitic cerebral cysticercosis in tapeworm carriers. The Cysticercosis Working Group in Peru. *Neurology*. 1999; 53:1582–1584. [PubMed: 10534273]
- * 20. Fleury A, Carrillo-Mezo R, Flisser A, Sciutto E, Corona T. Subarachnoid basal neurocysticercosis: a focus on the most severe form of the disease. *Expert Rev Anti Infect Ther*. 2010; 9:123–133. [PubMed: 21171883] [Illustrative description of a common and severe form of NCC]
21. Proano JV, Madrazo I, Avelar F, Lopez-Felix B, Diaz G, Grijalva I. Medical treatment for neurocysticercosis characterized by giant subarachnoid cysts. *N Engl J Med*. 2001; 345:879–885. [PubMed: 11565520]
22. Del Brutto OH, Santibanez R, Noboa CA, Aguirre R, Diaz E, Alarcon TA. Epilepsy due to neurocysticercosis: analysis of 203 patients. *Neurology*. 1992; 42:389–392. [PubMed: 1736171]
23. Garcia HH, Gonzalez AE, Gilman RH, Bernal T, Rodriguez S, Pretell EJ, Azcurra O, Parkhouse RM, Tsang VC, Harrison LJ. Circulating parasite antigen in patients with hydrocephalus secondary to neurocysticercosis. *Am J Trop Med Hyg*. 2002; 66:427–430. [PubMed: 12164300]
24. Roman, RA Suastegui; Soto-Hernandez, JL.; Sotelo, J. Effects of prednisone on ventriculoperitoneal shunt function in hydrocephalus secondary to cysticercosis: a preliminary study. *J Neurosurg*. 1996; 84:629–633. [PubMed: 8613855]
25. Garcia HH, Pretell EJ, Gilman RH, Martinez SM, Moulton LH, Del Brutto OH, Herrera G, Evans CA, Gonzalez AE. A trial of antiparasitic treatment to reduce the rate of seizures due to cerebral cysticercosis. *N Engl J Med*. 2004; 350:249–258. [PubMed: 14724304]
26. Sotelo J, del Brutto OH, Penagos P, Escobedo F, Torres B, Rodriguez-Carbajal J, Rubio-Donnadieu F. Comparison of therapeutic regimen of anticysticercal drugs for parenchymal brain cysticercosis. *J Neurol*. 1990; 237:69–72. [PubMed: 2192018]
27. Corona T, Lugo R, Medina R, Sotelo J. Single-day praziquantel therapy for neurocysticercosis. *N Engl J Med*. 1996; 334:125. [PubMed: 8531958]
28. Del Brutto OH, Roos KL, Coffey CS, Garcia HH. Meta-analysis: Cysticidal drugs for neurocysticercosis: albendazole and praziquantel. *Ann Intern Med*. 2006; 145:43–51. [PubMed: 16818928]
29. Vazquez ML, Jung H, Sotelo J. Plasma levels of praziquantel decrease when dexamethasone is given simultaneously. *Neurology*. 1987; 37:1561–1562. [PubMed: 3627459]
30. Agapejev S, Meira DA, Barraviera B, Machado JM, Marques PC, Mendes RP, Kamegasawa A, Ueda AK. Neurocysticercosis: treatment with albendazole and dextrochloropheniramine. *Trans R Soc Trop Med Hyg*. 1989; 83:377–383. [PubMed: 2617585]
31. Gongora-Rivera F, Soto-Hernandez JL, Gonzalez Esquivel D, Cook HJ, Marquez-Caraveo C, Hernandez Davila R, Santos-Zambrano J. Albendazole trial at 15 or 30 mg/kg/day for subarachnoid and intraventricular cysticercosis. *Neurology*. 2006; 66:436–438. [PubMed: 16382035]
32. Guo DM, Xie SP, Jia JP. Therapeutic efficacy of praziquantel, albendazole and a combination of the two drugs in cysticercosis. *Zhongguo Ji Sheng Chong Xue Yu Ji Sheng Chong Bing Za Zhi*. 2003; 21:187–188. [PubMed: 14628357]
- ** 33. Kaur S, Singhi P, Singhi S, Khandelwal N. Combination therapy with albendazole and praziquantel versus albendazole alone in children with seizures and single lesion neurocysticercosis: a randomized, placebo-controlled double blind trial. *Pediatr Infect Dis J*. 2009; 28:403–406. [PubMed: 19325515] [Initial controlled study of combined albendazole plus praziquantel therapy for NCC]
34. Garcia HH, Lescano AG, Lanchote V, Pretell EJ, Gonzales I, Bustos J, Takayanagui OM, Bonato P, Horton J, Saavedra H, et al. Pharmacokinetics of Combined Treatment with Praziquantel and Albendazole in Neurocysticercosis. *Br J Clin Pharmacol*. 2011
- ** 35. Rajshekhar V. Surgical management of neurocysticercosis. *Int J Surg*. 2010; 8:100–104. [PubMed: 20045747] [Updated review on current surgical approaches to NCC management.]
36. Torres-Corzo JG, Tapia-Perez JH, Vecchia RR, Chalita-Williams JC, Sanchez-Aguilar M, Sanchez-Rodriguez JJ. Endoscopic management of hydrocephalus due to neurocysticercosis. *Clin Neurol Neurosurg*. 2010; 112:11–16. [PubMed: 19767141]

37. Gonzales AE, Garcia HH, Gilman RH, Gavidia CM, Tsang VC, Bernal T, Falcon N, Romero M, Lopez-Urbina MT. Effective, single-dose treatment of porcine cysticercosis with oxfendazole. *Am J Trop Med Hyg.* 1996; 54:391–394. [PubMed: 8615453]
38. Gonzalez AE, Cama V, Gilman RH, Tsang VC, Pilcher JB, Chavera A, Castro M, Montenegro T, Verastegui M, Miranda E, et al. Prevalence and comparison of serologic assays, necropsy, and tongue examination for the diagnosis of porcine cysticercosis in Peru. *Am J Trop Med Hyg.* 1990; 43:194–199. [PubMed: 2389823]
39. Gonzalez AE, Gauci CG, Barber D, Gilman RH, Tsang VC, Garcia HH, Verastegui M, Lightowers MW. Vaccination of pigs to control human neurocysticercosis. *Am J Trop Med Hyg.* 2005; 72:837–839. [PubMed: 15964973]
40. Verastegui M, Gonzalez A, Gilman RH, Gavidia C, Falcon N, Bernal T, Garcia HH. Experimental infection model for *Taenia solium* cysticercosis in swine. Cysticercosis Working Group in Peru. *Vet Parasitol.* 2000; 94:33–44. [PubMed: 11078942]
41. Gonzalez AE, Falcon N, Gavidia C, Garcia HH, Tsang VC, Bernal T, Romero M, Gilman RH. Time-response curve of oxfendazole in the treatment of swine cysticercosis. *Am J Trop Med Hyg.* 1998; 59:832–836. [PubMed: 9840607]
42. Flisser A, Gauci CG, Zoli A, Martinez-Ocana J, Garza-Rodriguez A, Dominguez-Alpizar JL, Maravilla P, Rodriguez-Canul R, Avila G, Aguilar-Vega L, et al. Induction of protection against porcine cysticercosis by vaccination with recombinant oncosphere antigens. *Infect Immun.* 2004; 72:5292–5297. [PubMed: 15322025]

KEY POINTS

- Neurocysticercosis accounts for a substantial proportion of seizure disorders in endemic areas.
- Management of NCC is complex due to the variable nature of the lesions in terms of location, numbers, size, evolution, and immune response of the host
- Symptomatic management is the initial task and should not be neglected. Considerations on the use of antiparasitic drugs should be done after symptoms are appropriately managed.
- Improved anti-parasitic regimes with improved cysticidal efficacy are required. Combined albendazole and praziquantel appears to be a promising alternative.
- Minimally invasive neurosurgery (neuroendoscopy) has greatly improved the management of intraventricular NCC.