Review Article

Diagnosis and Treatment of Neurocysticercosis

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Neurocysticercosis, the infection caused by the larval form of the tapeworm Taenia solium, is the most common parasitic disease of the central nervous system and the most common cause of acquired epilepsy worldwide. This has primarily been a disease that remains endemic in low-socioeconomic countries, but because of increased migration neurocysticercosis is being diagnosed more frequently in high-income countries. During the past three decades improved diagnostics, imaging, and treatment have led to more accurate diagnosis and improved prognosis for patients. This article reviews the current literature on neurocysticercosis, including newer diagnostics and treatment developments.

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1. Introduction

Neurocysticercosis (NCC) is a neurologic infection caused by the larval stage of the tapeworm Taenia solium. In the developing world, NCC, infection of the central nervous system (CNS) with the T. solium larvae, is the most common cause of acquired epilepsy [1–3]. Because of globalization, many clinicians in industrialized countries who are unfamiliar with NCC are now faced with managing this disease. Humans are the definitive hosts for this parasite, and swine are the intermediate hosts. The adult tapeworm develops in human hosts after they ingest live cysticercus in undercook pork. NCC develops when humans accidentally ingest eggs. This occurs when feces of human carriers contaminates food, although the most important risk factor for the acquisition of cysticercosis is the proximity of a tapeworm carrier [4, 5]. Adult tapeworms shed proglottids, and each proglottid contains approximately 1000 to 2000 eggs. Once the hexacanth embryo reaches the parenchyma it forms cysticerci which undergo four stages of involution [6].

The first is the vesicular stage characterized by a cyst with a translucent vesicular wall, transparent fluid, and a viable invaginated scolex. During this stage there is little host inflammatory reaction. The cyst then develops a thick vesicular wall, the fluid becomes turbid, and the scolex degenerates during the next stage, which is termed the colloidal stage. An intense inflammatory host response is seen and is reflected in the pathology which reveals varying degrees of acute and chronic inflammation [1, 6]. Radiographic examination reveals cystic lesions with edema and enhancement and seizures are common [7]. The cyst continues to degenerate as it moves into the granular stage which is characterized by a thick vesicular wall, degenerated scolex, gliosis, and little inflammatory host response. Ultimately the parasite transforms into coarse calcified nodules; the calcific stage [1, 2, 6].

2. Clinical Manifestations of Neurocysticercosis (NCC)

The clinical manifestations of NCC range from asymptomatic to life threatening. Within the CNS it can affect the parenchyma, subarachnoid space, or intraventricular system. Ocular and spinal disease occurs, but is less common. Therefore, the clinical manifestations are pleomorphic and dependant on the location, number, and stage of the cysts at presentation. NCC is the leading cause of adult-onset
epilepsy in areas of the world where it is endemic, particularly in Latin America, Asia, and Africa [1]. Seizures are commonly generalized tonic-clonic or simple partial. Epilepsy occurs more frequent in patients with parenchymal disease, although it can occur in patients with cysts in the cortical sulci [1]. Seizures due to cysticercosis usually occur when the dying cyst incites an inflammatory reaction, but has been reported in the cystic stage. For many patients epilepsy may be the sole presentation of the disease with 50%–70% of patients experiencing recurrent seizures [8, 9].

There are multiple ways that cysticercosis can cause seizures. As noted, seizures can occur early in the disease in the setting of intense inflammation associated with viable or degenerating cysts. They can also occur secondary to vasculitis and infarction which occurs in the setting of subarachnoid disease [10]. Lastly, increasing evidence implicates calcified NCC in the development and maintenance of seizures and epilepsy [11]. Patients who present with seizures in regions where infection with T. solium is endemic commonly have calcified brain lesions observed on computerized axial tomography (CT) scan that are typical for NCC. In population-based studies calcified lesions on CT are much more common than viable cysts, and they are more prevalent in patients with epilepsy than they are in asymptomatic patients [11–13]. Strong evidence supports the role of calcified lesions in seizures; there is a high prevalence of cerebral calcifications in patients with seizures in the absence of other etiologies, and there is a positive correlation between endemic populations with increased proportions of calcification and seizure activity. In addition, individuals with calcified granulomas have increased risk of ongoing seizure [14–17].

There has been increasing evidence that perilesional edema, which occurs episodically, is associated with seizures [14, 15, 18–20]. Perilesional edema appears as a bright signal using magnetic resonance imaging (MRI) FLAIR or T2 imaging (Figure 1). It is almost always accompanied by enhancement around the calcified focus [11]. Previously calcified NCC has been classified as the inactive form of the disease, suggesting that it is less important than other forms of NCC [21]. Recently a growing literature is finding that perilesional edema related to calcifications seems to be a relatively frequent phenomenon, with reports ranging from 23%–35% in literature [8, 14, 22]. The natural history or pathophysiology of perilesional edema is not yet known, but it appears that it recurs, and repeated episodes tend to be associated with the same lesions in a patient. In a recent prospective nested case-control study, 110 patients with seizures or headaches and calcified lesions in an endemic region were followed for recurrent seizures. Of those with recurrent seizures, perilesional edema was noted on MRI in 50% as opposed to 9% of asymptomatic matched controls [23]. This study suggests that perilesional edema is a common and potentially preventable cause of seizure in endemic regions.

Although, seizures are the most common clinical manifestation of parenchymal NCC, focal neurologic signs have been reported and are usually related to the number, size, and location of the parasites in individuals with parenchymal disease. Intracranial hypertension can occur in patients with parenchymal NCC and is termed cysticercotic encephalitis [1, 24, 25]. This manifestation has been best described in children and young woman and is a result of the acute inflammatory response to massive cysticercal infection resulting in brain edema. Patients present with a syndrome characterized by clouding of consciousness, seizures, decreased visual acuity, headache, vomiting, and papilledema which can be subacute or acute in onset [1, 22]. These patients are treated with mannitol and corticosteroids in an attempt to control the inflammation and intracranial hypertension. Patients may even require decompressive temporal craniotomy. Those individuals with this form of NCC would not be candidates for antiparasitic agents, since treatment could exacerbate the inflammation and edema. Other causes of intracranial hypertension in patients with parenchymal NCC include the development of a large cyst that displaces midline structures or obstructs the flow of cerebrospinal fluid (CSF) in the cerebral aqueduct.

Psychiatric manifestations of NCC, such as depression and psychosis, have been described [26, 27]. A recent study found that patients admitted to a chronic inpatient psychiatric unit were more likely to have a positive serology for T. solium then healthy controls in the community. Of these inpatients, those with mental retardation were found to carry an increase risk of cysticercosis compared with patients with other psychiatric disorders. These patients were not carrying adult Taenia spp. in their stool and did not have CNS imaging, but the high prevalence of a positive cysticercosis serology in the inpatient psychiatric group suggests that there is a large proportion of cysticercosis in this group of patients [28]. Further studies are needed to explore the relationship between NCC and psychiatric disease.

Subarachnoid NCC is a common finding at autopsy, but when cysticerci find their way to the Sylvian fissure or the basilar cisterns the result can be devastating for the patient. The cysticercus larva (after embedding itself in the parenchyma) undergoes four stages of evolution: vesicular, vesicular colloid, granular nodular, and nodular calcified
visual acuity and visual field defects [31, 32]. Acute aseptic meningitis associated with subarachnoid disease has been described [1]. Cysts in the third and fourth ventricles can result in an inflammatory reaction throughout the ventricular system leading to granular ependymitis. When this occurs the cyst capsule can become fixed to the ventricular wall with strong adhesions and fibrosis [41]. Increased intracranial pressure due to hydrocephalus can occur if ependymitis occurs at the level of the cerebral aqueduct. These patients tend to have a more chronic course than those with cysts in the fourth ventricle [44].

Spinal cord involvement in NCC is rare, accounting for 1%–5% of all cases [1, 45]. Spinal cord involvement can be intramedullary or extramedullary with the latter being more common. Intramedullary cysts are most common in the thoracic spine and patients usually present with gradual onset of myelopathy similar to the presentation of intramedullary tumors [46–52]. Extramedullary cysts or leptomeningeal NCC is usually an extension of subarachnoid disease which has migrated from the basilar cisterns. Cysts may be single or may form clumps of multiple cysts extending along the entire spinal canal [46, 47]. The resulting clinical picture is characterized by a combination of radicular pain and motor deficits of subacute onset and progressive course [1].

Intraventricular cysticerci may be located in the anterior chamber, the lens, the vitreous and the subretinal space, but the latter is the most common location. Cysts in the subretinal space can cause progressive decrease in visual acuity. Vitreous cysts can produce worsening vision with the perception of something moving within the eye. Cysts in the anterior chamber may induce a severe iridocyclitis, while retro-ocular intraorbital cysticerci may cause decreased visual acuity due to pressure on the optic nerve [1, 53, 54].

### 3. Radiological Manifestations

Neuroimaging of parenchymal NCC depends on the stage of the development of the parasites. In the vesicular stage the cysticerci appear as cystic lesions within the brain parenchyma [7]. CT and MRI reveal that the cyst wall is thin and well demarcated from the parenchyma. The cysts lack perilesional edema and do not enhance after administration of contrast medium. There may be a bright nodule in their interior giving the lesion a “hole with dot” appearance that represents the scolex (Figure 2(a)) [55]. As the cysts begin to degenerate they appear as ill-defined lesions surrounded by edema which enhance after contrast medium administration. This is the colloidal stage of the cyst and represents the so-called “acute encephalitic phase” of NCC which likely represents an intense host reaction to the parasite (Figure 2(b)). MRI reveals a thick and hypointense presentation of fourth ventricle cyst is the Bruns’ syndrome which is characterized by episodic headache, papilledema, neck stiffness, sudden positional vertigo induced by rotary movements of the head, nausea and vomiting, drop attacks and loss of consciousness with rapid recovery and long asymptomatic periods [1]. Cysts in the third and fourth are a well-described cause of sudden death due acute obstructive hydrocephalus [41–43].

A degenerating cyst in the ventricles can result in an inflammatory reaction throughout the ventricular system leading to granular ependymitis. When this occurs the cyst capsule can become fixed to the ventricular wall with strong adhesions and fibrosis [41]. Increased intracranial pressure due to hydrocephalus can occur if ependymitis occurs at the level of the cerebral aqueduct. These patients tend to have a more chronic course than those with cysts in the fourth ventricle [44].
Ventricular cysts appear on CT images as cystic lesions. They are initially isodense with the CSF and are therefore not well visualized. However, their presence can be inferred from distortions of the ventricular system causing asymmetric or obstructive hydrocephalus [57]. In contrast, most ventricular cysts are well visualized by MRI because their signal properties differ from those of the CSF, particularly using FLAIR techniques [7]. They may also move within the ventricular cavities in response to movements of the patients’ head (ventricular migration sign), a phenomenon that is best observed with MRI than with CT [58]. Occasionally, this finding facilitates the diagnosis of ventricular cysticercosis.

In patients with spinal NCC, CT may reveal symmetrical enlargement of the cord (intramedullary cysts) or pseudoreticular formations within the spinal canal (leptomeningeal cysts). MRI reveals intramedullary cysticerci to be ring-enhancing lesions that may have an eccentric hyperintense nodule representing the scolex. Myelography still has a role in the diagnosis of patients with spinal leptomeningeal cysticercosis because it shows multiple filling defects in the column of contrast material corresponding to each cyst [59]. Leptomeningeal cysts may be mobile (changing their position according to the movements of the patient) [7, 59, 60].

4. Serology

Only tests based on detection of antibodies specific for *T. solium* antigens are reliable for clinical diagnosis and epidemiologic studies. To date, these are limited to those based on the use of purified glycoprotein antigens derived from *T. solium* cysticerci. The current assay of choice is the electroimmunotransfer blot (ETIB) using partially purified antigenic extracts [61, 62]. This assay has a specificity approaching 100% and a sensitivity of 94%–98% for patients with two or more cystic or enhancing lesions. A major limitation of these tests are frequent false negative results in patients with single intracranial cysticerci, in which fewer than 50% test positive. Sensitivity of specific antibody assays is also relatively low in patients with only calcified cysticerci [63].

Detection of circulating parasite antigen reflects the presence of live parasites establishes the presence of ongoing viable infection and may permit quantitative verification of successful treatment [64–66]. Garcia and others have used Ag-ELISA based on the use of a monoclonal antibody (HP10) that reacts with a repetitive carbohydrate epitope found in excretory/secretory and surface antigens of living cysticerci [66, 67]. This assay had a sensitivity of 86% when tested on (CSF) samples from a series of 50 Peruvian patients with NCC [68]. The specificity of the assay is about 96% and it has been used to follow patients after treatment. Parasite antigen levels fell significantly by 3 months after treatment in patients with “cured” parenchymal disease after albendazole therapy [66]. This study found that the sensitivity is low in intraparenchymal NCC, especially in patients with only a few intraparenchymal cysts [66]. In
a study examining patients with hydrocephalus and NCC the assay was positive in 14 of 29 patients, but negative in patients with calcifications [69]. A drop in antigen levels (serum and CSF) after treatment in subarachnoid disease has been reported in a small number of patients [70]. The management of subarachnoid disease is particularly complicated and the appropriate endpoint for treatment has not been established. Further studies employing this assay to follow patients with subarachnoid disease are needed. Recently a monoclonal antibody-based ELISA to detect T. solium antigens in urine has been described. The overall sensitivity of urine antigen detection for viable parasites was 92%, which decreased to 62.5% in patients with a single cyst. Most individuals with only calcified cysticercosis were urine antigen negative. This assay could be useful in diagnosis of NCC and evaluating the efficacy of treatment.

5. Treatment

5.1. Parenchymal Disease. Praziquantel and albendazole are antiparasitic agents that are effective against T. solium cysticerci killing between 60% and 85% of parenchymal brain cysticerci [71]. Most trials show greater cyst reduction with albendazole administration. However, most of these studies have been uncontrolled, observational imaging studies. The majority of studies evaluated praziquantel at a dosage of 50 mg/kg/d for 2 weeks, although studies describing a single day regimen have also been described [8, 72–83]. Higher doses have been used, but there is limited experience in literature [71, 72]. A dose of 15 mg/kg of albendazole for four weeks was initially employed, but later reduced to 15 days and then to one week [74, 76, 80, 81, 83–88]. Between the second and fifth day of treatment with an antiparasitic agent there may be an exacerbation of neurologic symptoms which has been attributed to inflammation secondary to killing of the cysticerci. Because of this inflammation steroids are generally administered in conjunction with albendazole or praziquantel to control the resulting edema [71]. It should be noted that steroids decrease the plasma level of praziquantel, but not albendazole [89].

Randomized studies evaluating the clinical benefit of treatment have yielded conflicting data with some studies indicating a benefit and others failing to show a difference [90–94]. There has been much controversy whether cysticidal drugs modify the natural course of neurocysticercosis. In 2004 a randomized, placebo-controlled trial of treatment of adults with 20 or less viable parenchymal cysts and a history of seizures using albendazole demonstrated a reduction in seizures and enhanced resolution of cysts after treatment [95]. Although a landmark study, the treatment was not completely effective. The number of patients who became free of seizures was similar in the two groups, but the reduction in the number of the seizures among patients who received the treatment was significant in patients with generalized seizures, not in the group with partial seizures. Further studies are needed to determine whether longer or repeated courses of therapy will result in a decrease in seizures overall and leave patients with fewer remaining cysticerci. A recent meta-analysis confirmed that treatment of parenchymal NCC is clinically beneficial [96]. These authors concluded drug therapy results in better resolution of colloidal and vesicular cysticerci, lower risk for recurrence of seizures in patients with colloidal cysticerci, and a reduction in the rate of generalized seizures in patients with vesicular cysticerci. However, there were not sufficient data to determine conclusively the superiority of either albendazole or praziquantel as first-line treatment of NCC in this meta-analysis [96]. Despite the numerous studies, an optimal therapeutic regimen for neurocysticercosis has not been established. The evidence favors albendazole over praziquantel, but longer courses and repeated courses might be needed for patients with multiple cysts. Future trials should look to define the optimal therapeutic regimen. A recent prospective, randomized placebo, controlled trial examined combination therapy with albendazole and praziquantel versus albendazole alone in 110 children with seizures and single enhancing lesions. There were no differences in recurrent seizures and resolution of the lesions. Larger studies are warranted with combination therapy in both parenchymal disease and extraparenchymal forms of neurocysticercosis [97].

Single enhancing lesions have a good prognosis. Studies examining this group of patients have shown variable clinical results, probably due to the heterogeneity of morphology of single enhancing lesions. The most rigorous double-blinded randomized treatment trial showed an initial increase in seizure occurrence, but in a follow-up evaluation at two years there was a significant benefit of treatment [87, 88]. The previously mentioned meta-analysis found that enhancing lesions benefited from treatment with antiparasitics [96]. Solid nodular cysts that are degenerating have shown resolution with antiparasitic treatment. Calcified cysts need not be treated with antiparasitic agents [4, 71].

Anticonvulsants are should be used to control seizures. Serum levels of phenytoin and carbamazepine may be lowered when given concomitantly with praziquantel [98].

There is no proven effective treatment for perilesional edema associated with calcified lesions. Steroids can control symptoms, but there are no data that treatment with steroids will prevent recurrent edema [4, 14]. Methotrexate has been used in patients with recurrent perilesional edema to control the host inflammatory response as a steroid sparing agent in patients requiring long-term steroids [99, 100]. Patients with cysticercotic encephalitis should not be treated with cysticidal drugs because this may exacerbate the intracranial hypertension. Treatment should be aimed at relieving edema with corticosteroids (up to 32 mg per day of dexamethasone) and mannitol at doses of 2 mg/kg per day [2].

5.2. Extraparenchymal NCC. There are no controlled trials on the management of subarachnoid disease. In a series of patients treated with only CSF diversion, 50% died at a median follow-up of 8 years and 11 months [101]. Cysticidal drugs with steroids and shunting for hydrocephalus have been used with success in subarachnoid disease [46, 102, 103]. The host inflammatory reaction around the cysts may
result in occlusion of leptomeningeal vessels resulting in stroke or hydrocephalus [9, 101]. Therefore, steroids must be used in conjunction with therapy [2, 4, 55]. Most experts consider subarachnoid NCC an indication for treatment with antiparasitic agents [71]. There is no consensus on the dose of antiparasitic agent or length of treatment for this form of NCC. A study of 33 patients with giant cysterceroci in the Sylvian fissure treated with albendazole (15 mg/kg/d for 4 weeks) found only one single death from aplastic anemia at 59 months, with patients requiring several courses of therapy [102]. Therefore, a single course in patients with subarachnoid disease patients is probably inadequate and long-term therapy (months) might be required to treat some patients. Similarly, the optimal dose and duration of steroids has not been determined. Methotrexate has been used as a steroid sparing agent in subarachnoid disease in patients requiring long-term steroids and experiencing intolerable side effects [99].

Therapy for ventricular disease needs to be individualized. Anthelmintic treatment of the fourth, third, and lateral ventricle has been reported [41, 104-107]. If hydrocephalus is present, surgery has been the mainstay in medical therapy [106]. Surgery has been the mainstay in this form of NCC [39, 108]. There is a growing literature supporting flexible neuroendoscopy to remove approachable subarachnoid cysts and cysts lodged in the lateral, third, and fourth ventricles [109–112]. Cysts that enhance on MRI may not be suitable for endoscopic removal.

It is important to recognize that the management of NCC is complicated and involves antiinflammatory agents, antiparasitic drugs, and in some cases surgery. It should be managed by physicians knowledgeable in this field.

References

[1] O. H. Del Brutto, J. Sotelo, and G. Roman, Neurocysticercosis: A Clinical Handbook, Swets & Zeitlinger, Lisse, The Netherlands, 1998.
[2] O. H. Del Brutto, "Neurocysticercosis," Seminars in Neurology, vol. 25, no. 3, pp. 243–251, 2005.
[3] H. H. Garcia, A. E. Gonzalez, C. A. W. Evans, and R. H. Gilman, "Taenia solium cysticercosis," The Lancet, vol. 362, no. 9383, pp. 547–556, 2003.
[4] T. E. Nash, G. Singh, A. C. White, et al., "Treatment of neurocysticercosis: current status and future research needs," Neurology, vol. 67, no. 7, pp. 1120–1127, 2006.
[5] A. G. Lescano, H. H. Garcia, R. H. Gilman, et al., "Taenia solium cysticercosis: a clinical and molecular approach," PLoS Neglected Tropical Diseases, vol. 3, article e371, 2009.
[6] A. Escobar, "The pathology of neurocysticercosis," in Cysticercosis of the Central Nervous System, E. Palacios, J. Rodriguez-Carbajal, and J. M. Taveras, Eds., pp. 27–54, Charles C. Thomas, Springfield, Ill, USA, 1983.
[7] H. H. Garcia and O. H. Del Brutto, "Imaging findings in neurocysticercosis," Acta Tropica, vol. 87, no. 1, pp. 71–78, 2003.
[8] O. H. Del Brutto, R. Santibanez, C. A. Noboa, R. Aguirre, E. Diaz, and T. A. Alarcon, "Epilepsy due to neurocysticercosis: analysis of 203 patients," Neurology, vol. 42, no. 2, pp. 389–392, 1992.
[9] G. F. McCormick, C. S. Zee, and J. Heiden, "Cysticercosis cerebri. Review of 127 cases," Archives of Neurology, vol. 39, no. 9, pp. 534–539, 1982.
[10] O. H. Del Brutto, "Cysticercosis and cerebrovascular disease: a review," Journal of Neurology, Neurosurgery and Psychiatry, vol. 55, no. 4, pp. 252–254, 1992.
[11] T. E. Nash, O. H. Del Brutto, J. A. Butman, et al., "Calcific neurocysticercosis and epileptogenesis," Neurology, vol. 62, no. 11, pp. 1934–1938, 2004.
[12] A. Fleury, T. Gomez, I. Alvarez, et al., "High prevalence of calcified silent neurocysticercosis in a rural village of Mexico," Neuroepidemiology, vol. 22, no. 2, pp. 139–145, 2003.
[13] J. Garcia-Noval, E. Moreno, F. de Mata, et al., "An epidemiological study of epilepsy and epileptic seizures in two rural Guatemalan communities," Annals of Tropical Medicine and Parasitology, vol. 95, no. 2, pp. 167–175, 2001.
[14] T. E. Nash, J. Pretell, and H. H. Garcia, "Calcified cysterceroci provoke perilesional edema and seizures," Clinical Infectious Diseases, vol. 33, no. 10, pp. 1649–1653, 2001.
[15] S. A. Antoniuk, I. Bruck, L. H. Coutinho Dos Santos, et al., "Seizures associated with calcifications and edema in neurocysticercosis," Pediatric Neurology, vol. 25, no. 4, pp. 309–311, 2001.
[16] A. Thussu, A. Arora, S. Prabhakar, V. Lal, and I. M. S. Sawhney, "Acute symptomatic seizures due to single CT lesions: how long to treat with antiepileptic drugs?" Neurology India, vol. 50, no. 2, pp. 141–144, 2002.
[17] S. Rajadhyaksha, K. N. Shah, S. Kanhere, N. Naik, and R. Mehta, "Does treatment change the outcome of seizures and computerized tomographic lesions in intracranial granulomas?" Journal of Tropical Pediatrics, vol. 45, no. 3, pp. 161–165, 1999.
[18] T. E. Nash and N. J. Patronas, "Edema associated with calcified lesions in neurocysticercosis," Neurology, vol. 53, no. 4, pp. 777–781, 1999.
[19] T. N. Sheth, L. Pilon, J. Keystone, and W. Kucharczyk, "Persistent MR contrast enhancement of calcified neurocysticercosis lesions," American Journal of Neuroradiology, vol. 19, no. 1, pp. 79–82, 1998.
[20] S. Y. Park, A. J. Barkovich, and P. S. Weintrub, "Clinical implications of calcified lesions of neurocysticercosis," Pediatric Infectious Disease Journal, vol. 19, no. 6, pp. 581–583, 2000.
[21] J. Sotelo, V. Guerrero, and F. Rubio, "Neurocysticercosis: a new classification based on active and inactive forms. A study of 753 cases," Archives of Internal Medicine, vol. 145, no. 3, pp. 442–445, 1985.
[22] R. K. Garg, "Neurocysticercosis," Postgraduate Medical Journal, vol. 74, no. 872, pp. 321–326, 1998.
[23] T. E. Nash, J. A. Butman, and N. J. Patronas, "Perilesional brain oedema and seizure activity in patients with calcified neurocysticercosis: a prospective cohort and nested case-control study," The Lancet Neurology, vol. 7, no. 12, pp. 1099–1105, 2008.
[24] R. Rangel, B. Torres, O. Del Bruto, and J. Sotelo, "Cystercotic encephalitis: a severe form in young females," American Journal of Tropical Medicine and Hygiene, vol. 36, no. 2, pp. 387–392, 1987.
[25] N. Ignacio Madrazo, B. Olhagaray, M. Becerra, M. A. Sandoval, and L. Raúl Soto, "Acute cysticercosis encephalitis: description of a histologically confirmed case," Neurosurgery, vol. 33, no. 5, pp. 983–985, 1993.
[26] O. V. Forlenza, A. H. G. Filho, J. P. S. Nobrega, et al., "Psychiatric manifestations of neurocysticercosis: a study of 38 patients from a neurology clinic in Brazil," Journal of
Neurology, Neurosurgery and Psychiatry, vol. 62, no. 6, pp. 612–616, 1997.

[27] H. B. F. Dixon and F. M. Lipscomb, “Cysticercosis: an analysis and follow up of 450 cases,” Special Report Series 299, Medical Research Council, Her Majesty’s Stationery, London, UK, 1961.

[28] N. W. Meza, N. E. Rossi, T. N. Galeazzi, et al., “Cysticercosis in chronic psychiatric inpatients from a Venezuelan community,” American Journal of Tropical Medicine and Hygiene, vol. 73, no. 3, pp. 504–509, 2005.

[29] E. E. Bickerstaff, P. C. Cloake, B. Hughes, and W. T. Smith, “The racemose form of cerebral cysticercosis,” Brain, vol. 75, pp. 1–18, 1952.

[30] R. D. Lobato, E. Lamas, J. M. Portillo, et al., “Hydrocephalus in cerebral cysticercosis. Pathogenic and therapeutic considerations,” Journal of Neurosurgery, vol. 55, no. 5, pp. 786–793, 1981.

[31] J. R. Keane, “Cysticercosis: unusual neuro-ophtalmologic signs,” Journal of Clinical Neuro-Ophthalmology, vol. 13, no. 3, pp. 194–199, 1993.

[32] J. R. Keane, “Neuro-ophtalmologic signs and symptoms of cysticercosis,” Archives of Ophthalmology, vol. 100, no. 9, pp. 1445–1448, 1982.

[33] F. Barinagarrementeria and O. H. Del Brutto, “Lacunar syndrome due to neurocysticercosis,” Archives of Neurology, vol. 46, no. 4, pp. 415–417, 1989.

[34] J. L. Soto-Hernandez, S. G.-L. Andrade, L. A. Rojas-Echeverri, F. Texeira, and V. Romero, “Subarachnoid hemorrhage secondary to a ruptured inflammatory aneurysm: a possible manifestation of neurocysticercosis: case report,” Neurosurgery, vol. 38, no. 1, pp. 197–200, 1996.

[35] F. Barinagarrementeria and C. Cantu, “Frequency of cerebral arteritis in subarachnoid cysticercosis: an angiographic study,” Stroke, vol. 29, no. 1, pp. 123–125, 1998.

[36] O. H. Del Brutto and J. Sotelo, “Neurocysticercosis: an update,” Reviews of Infectious Diseases, vol. 10, no. 6, pp. 1073–1087, 1988.

[37] R. Jankowski, R. D. Zimmerman, and N. E. Leeds, “Cysticercosis presenting as a mass lesion at foramen of Monro,” Journal of Computer Assisted Tomography, vol. 3, no. 5, pp. 694–696, 1979.

[38] J. S. King and Y. Hosobuchi, “Cysticercus cyst of the lateral ventricle,” Surgical Neurology, vol. 7, no. 3, pp. 125–129, 1977.

[39] M. L. J. Apuzzo, W. R. Dobkin, C. S. Zee, J. C. Chan, S. L. Giannotta, and M. H. Weiss, “Surgical considerations in treatment of intraventricular cysticercosis. An analysis of 45 cases,” Journal of Neurosurgery, vol. 60, no. 2, pp. 400–407, 1984.

[40] W. T. Couldwell, C.-S. Zee, and M. L. J. Apuzzo, “Definition of the role of contemporary surgical management in cisternal and parenchymatous cysticercosis cerebri,” Neurosurgery, vol. 28, no. 2, pp. 231–237, 1991.

[41] A. C. Cuetter and R. J. Andrews, “Intraventricular neurocysticercosis: 18 consecutive patients and review of the literature,” Neurosurgical Focus, vol. 12, article e5, 2002.

[42] C. S. Zee, H. D. Segall, M. L. Apuzzo, J. Ahmadi, and W. R. Dobkin, “Intraventricular cysticercal cysts: further neuroradiologic observations and neurosurgical implications,” American Journal of Neuroradiology, vol. 5, no. 6, pp. 727–730, 1984.

[43] J. R. Keane, “Death from cysticercosis. Seven patients with unrecognized obstructive hydrocephalus,” Western Journal of Medicine, vol. 140, no. 5, pp. 787–789, 1984.

[44] A. Salazar, J. Sotelo, H. Martinez, and E. Escobedo, “Differential diagnosis between ventriculitis and fourth ventricle cyst in neurocysticercosis,” Journal of Neurosurgery, vol. 59, no. 4, pp. 660–663, 1983.

[45] F. U. Ahmad and B. S. Sharma, “Treatment of intramedullary spinal cysticercosis: report of 2 cases and review of literature,” Surgical Neurology, vol. 67, no. 1, pp. 74–77, 2007.

[46] J. C. Bandres, A. C. White Jr., T. Samo, E. C. Murphy, and R. L. Harris, “Extraparenchymal neurocysticercosis: report of five cases and review of management,” Clinical Infectious Diseases, vol. 15, no. 5, pp. 799–811, 1992.

[47] J. L. De Souza Queiroz, A. Pellegrini Filho Jr., D. Callegaro, and L. Lopez De Faria, “Intramedullary cysticercosis. Case report, literature review and comments on pathogenesis,” Journal of the Neurological Sciences, vol. 26, no. 1, pp. 61–70, 1975.

[48] R. Garza Mercado, “Intramedullary cysticercosis,” Surgical Neurology, vol. 5, no. 6, pp. 331–332, 1976.

[49] M. Natarajan, K. R. Ramasubramanian, and A. K. Muthu, “Intramedullary cysticercosis of spinal cord,” Surgical Neurology, vol. 6, no. 3, pp. 157–158, 1976.

[50] E. D. Rocca and B. Neira, “Cysticercosis of the spine,” Revista de Neuro-Psiquiatria, vol. 42, no. 2, pp. 96–103, 1979.

[51] F. Cabieses, M. Vallenas, and R. Landa, “Cysticercosis of the spinal cord,” Journal of Neurosurgery, vol. 16, pp. 337–341, 1959.

[52] I. Akiyuchi, T. Fujiwara, H. Matsuyama, H. Muranaka, and M. Kameyama, “Intramedullary spinal cysticercosis,” Neurology, vol. 29, no. 11, pp. 1531–1534, 1979.

[53] N. A. Sabrosa and M. Zajdenweber, “Nematode infections of the eye: toxocariasis, onchocerciosis, diffuse subcutaneous neuroretinitis, and cysticercosis,” Ophthalmology Clinics of North America, vol. 15, no. 3, pp. 351–356, 2002.

[54] N. A. Sabrosa and E. C. de Souza, “Nematode infections of the eye: toxocariasis and diffuse subcutaneous neuroretinitis,” Current Opinion in Ophthalmology, vol. 12, no. 6, pp. 450–454, 2001.

[55] H. H. Garcia, O. H. Del Brutto, T. E. Nash, A. C. White Jr., V. C. W. Tsang, and R. H. Gilman, “New concepts in the diagnosis and management of neurocysticercosis (Taenia solium),” American Journal of Tropical Medicine and Hygiene, vol. 72, no. 1, pp. 3–9, 2005.

[56] H. R. Martinez, R. Rangel-Guerra, G. Elizondo, et al., “MR imaging in neurocysticercosis: a study of 36 cases,” American Journal of Neuroradiology, vol. 10, no. 5, pp. 1011–1019, 1989.

[57] I. Madrazo, J. A. Garcia-Renteria, M. Sandoval, and F. J. Lopez Vega, “Intraventricular cysticercosis,” Neurosurgery, vol. 12, no. 2, pp. 148–152, 1983.

[58] R. A. Rangel-Guerra, J. Herrera, G. Elizondo, and J. Gonzalez-Morantes, “Neurocysticercosis,” Archives of Neurology, vol. 5, p. 492, 1988.

[59] K. S. Kim and P. E. Weinberg, “Spinal cysticercosis,” Surgical Neurology, vol. 24, no. 1, pp. 80–82, 1985.

[60] G. Santin and J. Vargas, “Roentgen study of cysticercosis of central nervous system,” Radiology, vol. 86, no. 3, pp. 520–528, 1966.

[61] P. M. Schantz, V. C. Tsang, and S. E. Maddison, “Serodiagnosis of neurocysticercosis,” Reviews of Infectious Diseases, vol. 10, no. 6, pp. 1231–1233, 1988.

[62] V. C. W. Tsang, J. A. Brand, and A. E. Boyer, “An enzyme-linked immunoelectrotransfer blot assay and glycoprotein antigens for diagnosing human cysticercosis (Taenia solium),” Journal of Infectious Diseases, vol. 159, no. 1, pp. 50–59, 1989.
[63] M. Wilson, R. T. Bryan, J. A. Fried, et al., “Clinical evaluation of the cysticercosis enzyme-linked immunoelectrotransfer blot in patients with neurocysticercosis,” Journal of Infectious Diseases, vol. 164, no. 5, pp. 1007–1009, 1991.

[64] D. Correa, M. A. Sandoval, L. J. S. Harrison, et al., “Human neurocysticercosis: comparison of enzyme immunoassay capture technique based on monoclonal and polyclonal antibodies for the detection of parasite products in cerebrospinal fluid,” Transactions of the Royal Society of Tropical Medicine and Hygiene, vol. 83, no. 6, pp. 814–816, 1989.

[65] Nguekam, A. P. Zoli, P. Ongolo-zogo, P. Dorny, J. Brandt, and Se Geerts, “Follow-up of neurocysticercosis patients after treatment using an antigen detection ELISA,” Parasite, vol. 10, no. 1, pp. 65–68, 2003.

[66] H. H. Garcia, R. M. E. Parkhouse, R. H. Gilman, et al., “Serum antigen detection in the diagnosis, treatment, and follow-up of neurocysticercosis patients,” Transactions of the Royal Society of Tropical Medicine and Hygiene, vol. 94, no. 6, pp. 673–676, 2000.

[67] L. J. S. Harrison, G. W. P. Joshua, S. H. Wright, and R. M. E. Parkhouse, “Specific detection of circulating surface/secreted glycoproteins of viable cysterci in Taenia saginata cysticercosis,” Parasite Immunology, vol. 11, no. 4, pp. 351–370, 1989.

[68] H. H. Garcia, L. J. S. Harrison, R. M. E. Parkhouse, et al., “A specific antigen-detection ELISA for the diagnosis of human neurocysticercosis,” Transactions of the Royal Society of Tropical Medicine and Hygiene, vol. 92, no. 4, pp. 411–414, 1998.

[69] H. H. Garcia, A. E. Gonzalez, R. H. Gilman, et al., “Circulating parasite antigen in patients with hydrocephalus secondary to neurocysticercosis,” American Journal of Tropical Medicine and Hygiene, vol. 66, no. 4, pp. 427–430, 2002.

[70] A. Fleury, M. Hernández, M. Avila, et al., “Detection of HP10 antigen in serum for diagnosis and follow-up of subarachnoidal and intraventricular human neurocysticercosis,” Journal of Neurology, Neurosurgery and Psychiatry, vol. 78, no. 9, pp. 970–974, 2007.

[71] H. H. Garcia, C. A. W. Evans, T. E. Nash, et al., “Current consensus guidelines for treatment of neurocysticercosis,” Clinical Microbiology Reviews, vol. 15, no. 4, pp. 747–756, 2002.

[72] P. R. M. Bittencourt, C. M. Gracia, A. M. Gorz, S. Mazer, and T. V. Oliveira, “High-dose praziquantel for neurocysticercosis: efficacy and tolerability,” European Neurology, vol. 30, no. 4, pp. 229–234, 1990.

[73] D. Botero and S. Castano, “Treatment of cysticercosis with praziquantel in Colombia,” American Journal of Tropical Medicine and Hygiene, vol. 31, no. 4, pp. 811–821, 1982.

[74] M. Cruz, I. Cruz, and J. Horton, “Albendazole versus praziquantel in the treatment of cerebral cysticercosis: clinical evaluation,” Transactions of the Royal Society of Tropical Medicine and Hygiene, vol. 85, no. 2, pp. 244–247, 1991.

[75] L. D. deGhetaldi, R. M. Norman, and A. W. Douville Jr., “Cerebral cysticercosis treated biphasically with dexamethasone and praziquantel,” Annals of Internal Medicine, vol. 99, no. 2, pp. 179–181, 1983.

[76] S.-K. Kim, K.-C. Wang, S.-H. Paek, K.-S. Hong, and B.-K. Cho, “Outcomes of medical treatment of neurocysticercosis: a study of 65 cases in Cheju Island, Korea,” Surgical Neurology, vol. 52, no. 6, pp. 563–569, 1999.

[77] P. M. Lawner, “Medical management of neurocysticercosis with praziquantel,” Bulletin of Clinical Neurosciences, vol. 48, pp. 102–105, 1983.

[78] R. Leblanc, K. F. Knowles, D. Melanson, J. D. MacLean, G. Rouleau, and J. P. Farmer, “Neurocysticercosis: surgical and medical management with praziquantel,” Neurosurgery, vol. 18, no. 4, pp. 419–427, 1986.

[79] K. Markwalder, K. Hess, A. Valavanis, and F. Witassek, “Cerebral cysticercosis: treatment with praziquantel. Report of two cases,” American Journal of Tropical Medicine and Hygiene, vol. 33, no. 2, pp. 273–280, 1984.

[80] J. Soto, O. H. del Brutto, P. Penagos, et al., “Comparison of therapeutic regimen of anticysticercal drugs for parenchymal brain cysticercosis,” Journal of Neurology, vol. 237, no. 2, pp. 69–72, 1990.

[81] J. Soto, F. Escobedo, and P. Penagos, “Albendazole vs praziquantel for therapy for neurocysticercosis. A controlled trial,” Archives of Neurology, vol. 45, no. 5, pp. 532–534, 1988.

[82] A. Spina-Franca, J. P. S. Nobrega, J. A. Livramento, and L. R. Machado, “Administration of praziquantel in neurocysticercosis,” Tropenmedizin und Parasitologie, vol. 33, no. 1, pp. 1–4, 1982.

[83] O. M. Takayanagui and E. Jardim, “Therapy for neurocysticercosis: comparison between albendazole and praziquantel,” Archives of Neurology, vol. 49, no. 3, pp. 290–294, 1992.

[84] S. Agapiejev, D. A. Meira, B. Barraquiera, et al., “Neurocysticercosis: treatment with albendazole and dextrochloropheniramine. (preliminary report),” Revista do Instituto de Medicina Tropical de Sao Paulo, vol. 30, no. 5, pp. 387–389, 1988.

[85] F. Alarcon and J. C. Maldonado, “Short course of albendazole therapy for neurocysticercosis,” Clinical Neurology and Neurosurgery, vol. 108, no. 8, pp. 810–811, 2006.

[86] D. Botero, C. S. Uribe, J. L. Sanchez, et al., “Short course albendazole treatment for neurocysticercosis in Columbia,” Transactions of the Royal Society of Tropical Medicine and Hygiene, vol. 87, no. 5, pp. 576–577, 1993.

[87] H. H. Garcia, R. H. Gilman, J. Horton, et al., “Albendazole therapy for neurocysticercosis: a prospective double-blind trial comparing 7 versus 14 days of treatment,” Neurology, vol. 48, no. 5, pp. 1421–1427, 1997.

[88] P. C. Sanchetee, S. Venkataraman, R. M. Dhamija, and A. K. Roy, “Albendazole therapy for neurocysticercosis,” The Journal of the Association of Physicians of India, vol. 42, no. 2, pp. 116–117, 1994.

[89] M. L. Vazquez, H. Jung, and J. Soto, “Plasma levels of praziquantel decrease when dexamethasone is given simultaneously,” Neurology, vol. 37, no. 9, pp. 1561–1562, 1987.

[90] A. K. Baranwal, P. D. Singh, N. Khandelwal, and S. C. Singh, “Albendazole therapy in children with focal seizures and small single enhancing computerized tomographic lesions: a randomized, placebo-controlled, double blind trial,” Pediatric Infectious Disease Journal, vol. 17, no. 8, pp. 696–700, 1998.

[91] A. K. Baranwal, P. D. Singh, S. C. Singh, and N. Khandelwal, “Seizure recurrence in children with focal seizures and single small enhancing computed tomographic lesions: prognostic factors on long-term follow-up,” Journal of Child Neurology, vol. 16, no. 6, pp. 443–445, 2001.

[92] P. Singh, M. Ray, S. Singh, and N. Khandelwal, “Clinical spectrum of 500 children with neurocysticercosis and response to albendazole therapy,” Journal of Child Neurology, vol. 15, no. 4, pp. 207–213, 2000.

[93] M. V. Padma, M. Behari, N. K. Misra, and G. K. Ahuja, “Albendazole in single CT ring lesions in epilepsy,” Neurology, vol. 44, no. 7, pp. 1344–1346, 1994.
[94] A. Carpio, E. A. Kelvin, E. Bagiella, et al., “Effects of albendazole treatment on neurocysticercosis: a randomised controlled trial,” *Journal of Neurology, Neurosurgery and Psychiatry*, vol. 79, no. 9, pp. 1050–1055, 2008.

[95] V. Vazquez and J. Sotelo, “The course of seizures after treatment for cerebral cysticercosis,” *The New England Journal of Medicine*, vol. 327, no. 10, pp. 696–701, 1992.

[96] O. H. Del Brutto, K. L. Roos, C. S. Coffey, and H. H. Garcia, “Meta-analysis: cysticidal drugs for neurocysticercosis: albendazole and praziquantel,” *Annals of Internal Medicine*, vol. 145, no. 1, pp. 43–51, 2006.

[97] S. Kaur, P. Singhi, and N. Khandelwal, “Combination therapy with albendazole and praziquantel versus albendazole alone in children with seizures and single lesion neurocysticercosis: a randomized placebo-controlled double blind trial,” *Pediatric Infectious Disease Journal*, vol. 28, no. 5, pp. 403–406, 2009.

[98] P. R. M. Bittencourt, C. M. Gracia, R. Martins, A. G. Fernandes, H. W. Dickmann, and W. Jung, “Phenytoin and carbamazepine decrease oral bioavailability of praziquantel,” *Neurology*, vol. 42, no. 3, pp. 492–496, 1992.

[99] E. Mitre, K. R. Talaat, M. R. Sperling, and T. E. Nash, “Methotrexate as a corticosteroid-sparing agent in complicated neurocysticercosis,” *Clinical Infectious Diseases*, vol. 44, no. 4, pp. 549–553, 2007.

[100] P. B. Keiser and T. E. Nash, “Prolonged perilesional edema after treatment of parenchymal neurocysticercosis: methotrexate as a corticosteroid-sparing agent,” *Clinical Infectious Diseases*, vol. 36, no. 10, pp. e122–e126, 2003.

[101] J. Sotelo and C. Marin, “Hydrocephalus secondary to cysticercotic arachnoiditis. A long-term follow-up review of 92 cases,” *Journal of Neurosurgery*, vol. 66, no. 5, pp. 686–689, 1987.

[102] J. V. Proaño, I. Madrazo, F. Avelar, B. Lopez-Felix, G. Diaz, and I. Grijalva, “Medical treatment for neurocysticercosis characterized by giant subarachnoid cysts,” *The New England Journal of Medicine*, vol. 345, no. 12, pp. 879–885, 2001.

[103] O. H. Del Brutto, “Albendazole therapy for subarachnoid cysticerci: clinical and neuroimaging analysis of 17 patients,” *Journal of Neurology, Neurosurgery and Psychiatry*, vol. 62, no. 6, pp. 659–661, 1997.

[104] J. V. Proaño, I. Madrazo, L. Garcia, E. Garcia-Torres, and D. Correa, “Albendazole and praziquantel treatment in neurocysticercosis of the fourth ventricle,” *Journal of Neurosurgery*, vol. 87, no. 1, pp. 29–33, 1997.

[105] F. Góngora-Rivera, J. L. Soto-Hernández, D. González Esquivel, et al., “Albendazole trial at 15 or 30 mg/kg/day for subarachnoid and intraventricular cysticercosis,” *Neurology*, vol. 66, no. 3, pp. 436–438, 2006.

[106] A. C. Cuettler, J. García-Bobadilla, L. G. Guerra, F. M. Martínez, and B. Kaim, “Neurocysticercosis: focus on intraventricular disease,” *Clinical Infectious Diseases*, vol. 24, no. 2, pp. 157–164, 1997.

[107] C. S. Zee, H. D. Segall, S. Destian, J. Ahmadi, and M. L. J. Apuzzo, “MRI of intraventricular cysticercosis: surgical implications,” *Journal of Computer Assisted Tomography*, vol. 17, no. 6, pp. 932–939, 1993.

[108] B. O. Colli, N. Martelli, J. A. Assirati Jr., H. R. Machado, and S. de Vergueiro Jorjá, “Results of surgical treatment of neurocysticercosis in 69 cases,” *Journal of Neurosurgery*, vol. 65, no. 3, pp. 309–315, 1986.

[109] M. Bergsneider, L. T. Holly, J. H. Lee, W. A. King, and J. G. Frazee, “Endoscopic management of cysticercal cysts within the lateral and third ventricles,” *Journal of Neurosurgery*, vol. 92, no. 1, pp. 14–23, 2000.

[110] M. Bergsneider, “Endoscopic removal of cysticercal cysts within the fourth ventricle: technique and results,” *Neurosurgical Focus*, vol. 6, article e8, 1999.

[111] T. Gravori, T. Steineke, and M. Bergsneider, “Endoscopic removal of cisternal neurocysticercal cysts. Technical note,” *Neurosurgical Focus*, vol. 12, no. 6, article e7, 2002.

[112] B. Anandh, M. Mohanty, S. Sampath, S. S. Praharaj, and S. Kolluri, “Endoscopic approach to intraventricular cysticercal lesions,” *Minimally Invasive Neurosurgery*, vol. 44, no. 4, pp. 194–196, 2001.