Current challenges in neurocysticercosis: recent data and where we are heading

Christina M. Coyle[a], *Javier A. Bustos*[b,*] *and Hector H. Garcia*[c,d,*]

Purpose of review
Neurocysticercosis (NCC) is still a significant contributor to neurological disease in vast regions of the world, and increasingly diagnosed in nonendemic countries because of travel and immigration from endemic settings. There is a need for clinicians in endemic and nonendemic regions to understand the complexities of its diagnosis and management.

Recent findings
Recent information on the performance and use of available imaging and immunodiagnostic tools as well as antiparasitic and anti-inflammatory therapeutic regimes were assessed.

Summary
Imaging and serology data should be assessed in the context of the specific type of NCC to improve diagnostic precision. In terms of therapeutic approaches, more controlled data is required on the efficacy and safety of combined antiparasitic therapy, and antiseizure and anti-inflammatory regimes should be optimized to minimize perilesional damage and reduce the risk of epilepsy.

Keywords
albendazole, cysticercosis, epilepsy, intracranial hypertension, neurocysticercosis, praziquantel, *Taenia solium*

INTRODUCTION

Neurocysticercosis (NCC) is the most common helminthic cause of neurological disease in endemic regions, contributing significantly to the burden of epilepsy and other neurological morbidity [1–4]. The distribution of this disease includes Latin America, sub-Saharan Africa, and large parts of Asia including the Indian subcontinent, but with increased migration of individuals from endemic to nonendemic regions cases are seen with some frequency in the United States, United Kingdom, and Europe [5▪▪,6–8]. There is a need for clinicians in endemic and nonendemic regions to understand the complexities of its diagnosis and management.

NCC is a complex disease with a pleomorphic clinical presentation driven by the location and stage of cysts, burden of disease, and host response [9]. It can vary from completely asymptomatic infection to severe disease and death and in endemic regions, it is regarded as the 'great imitator'. Neuroimaging is the cornerstone of diagnosis and, when clearcut, it establishes the diagnosis of NCC. But this is frequently not the case, particularly in nonendemic regions where there is a lack of familiarity with this disease leading to misdiagnosis and delays in treatment [10▪▪,11]. This diagnostic challenge is followed by a series of management challenges, as NCC is an extremely complex disease in which macroscopic parasitic lesions cause focal inflammation and damage in the brain parenchyma, or occupy extraparenchymal spaces.

Generally, parenchymal brain cysticercosis with active inflammation presents with seizures as the major manifestation, which respond well to antiepileptic drugs [12]. Extraparenchymal NCC is not characterized by seizures, but rather by mass effects, hydrocephalus and intracranial hypertension. Extraparenchymal NCC has a worse prognosis with very high mortality rates in those who do not receive optimum management, which is not available in

[a]Department of Infectious Diseases, Albert Einstein College of Medicine, Bronx, New York, USA, [b]Center for Global Health, School of Health Sciences, Universidad Peruana de Ciencias Aplicadas (UPC), [c]Center for Global Health, Universidad Peruana Cayetano heredia, (UPCH) and [d]Cysticercosis Unit, Instituto Nacional de Ciencias Neurológicas, Lima, Perú

Correspondence to Hector H. Garcia, MD PhD, Cysticercosis Unit, Instituto Nacional de Ciencias Neurologicas, Lima, Peru.
E-mail: hgarcia@jhsph.edu

*for the Cysticercosis Working Group in Peru.

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KEY POINTS

- Imaging and serology data should be assessed in the context of the specific type of NCC of each patient.
- Further data on combined albendazole with praziquantel antiparasitic therapy should serve to confirm its safety and efficacy.
- Sound use of antiseizure medication and optimizing anti-inflammatory therapy should be considered in individuals with parenchymal NCC.
- Comorbidities should be included in the assessment of patients with NCC.

most endemic areas. Although outcomes are improved in this form of disease with appropriate antiparasitic and anti-inflammatory treatment with close follow-up of neuroimaging, serum antigen, and clinical response to help complete parasite resolution, which can be months. In recent years, our understanding of the pathophysiology of NCC has improved considerably and new concepts are now established on its diagnosis and management [13].

PATHOPHYSIOLOGY OF SYMPTOMATIC NEUROCYSTICERCOSIS

After infection, parenchymal brain NCC establishes in the human brain and then after a variable asymptomatic period is detected by the host immune system and destroyed, which elicits local inflammation and ultimately leads to scarring [14]. It is now well established that these will frequently become epilepsy foci [15]. Radiological signs of residual gliosis and calcification are associated with worse evolution of the underlying seizure disorder [16].

In both porcine models of NCC and PET scan studies in humans, lesions of NCC showed evidence of perilesional inflammation [15,17]. Inflammation is thought to lead to epileptogenesis either by causing gliosis and/or by causing areas of blood–brain barrier dysfunction. Data from animal models is now available that demonstrates chronic inflammation and damage around parenchymal brain cysts begins in the early stages of the infection [15,17,18▪]. Although the inflammation ensures cyst resolution, it also seems to play a role in damage and scarring which may drive many of the chronic symptoms, such as seizures [19▪▪,20–23].

On the other hand, subarachnoid NCC (SANCC) has a long latent period of 10–20 years [24]. This serious form of NCC is different from other forms as it is characterized by aberrant proliferating parasite derived from *Taenia solium* cysts infiltrating the arachnoid spaces. This can lead to chronic arachnoiditis, mass effects, and hydrocephalus. The inflammation can cause vasculitis and lead to strokes [25,26]. The response rate of subarachnoid NCC to a first course of antiparasitic treatment is low, therefore, many experts have successfully treated patients with albendazole (ABZ) and praziquantel with steroids for a prolonged course. It is now clear that the proliferative membranes of subarachnoid lesions involves parasite germinative cells, which seems key to modulate inflammation and edema to reduce risks associated with intracranial hypertension [27]. Genomic characterization of this cell subpopulation should allow the development of specific diagnostic tests and therapeutic approaches, which would change clinical outcomes.

CHALLENGES IN THE DIAGNOSIS OF NEUROCYSTICERCOSIS

The primary diagnostic approach for NCC is neuroimaging and it may be quite characteristic, particularly in the case of multiple viable cysts with well defined scolices or extensive subarachnoid disease [28,29]. In general, MRI is much more sensitive than computed tomography scan (CT) for viable NCC. Balanced steady-state gradient-echo sequences [Fast Imaging Employing Steadystate Acquisition (FIESTA), Balanced Fast Field Echo (BFFE), True Fast Imaging with Steady state Precession (TrueFISP), FIESTA-C, and Constructive Interference Steady State (CISS), depending on the magnetic resonance company] discriminate structures in liquid cavities, which makes these sequences very helpful in defining the parasitic lesions in the subarachnoid space [30–32]. On the other hand, MRI is not well suited to detect calcifications, although susceptibility-weighted sequences may help delineate larger calcified lesions. Calcifications, particularly small ones, are much better detected on noncontrast CT scans. The subtleties of unusual imaging presentations of NCC can frequently escape the untrained eye leading to delayed diagnosis [33]. Some challenging scenarios are nonspecifically appearing inflamed, enhancing parenchymal lesions; atypical parenchymal cysts (i.e. not well defined, some may have contents); enhancing calcifications with or without edema; small subarachnoid lesions; and chronic basal inflammation (Fig. 1). In these cases, new MRI protocols may help but, will not necessarily lead to a confirmed diagnosis. The supportive role of confirmatory serology in these cases becomes crucial.

Immunodiagnosis in NCC involves the detection of specific antibodies and antigens. Antibody detection is more sensitive but, cannot be used to

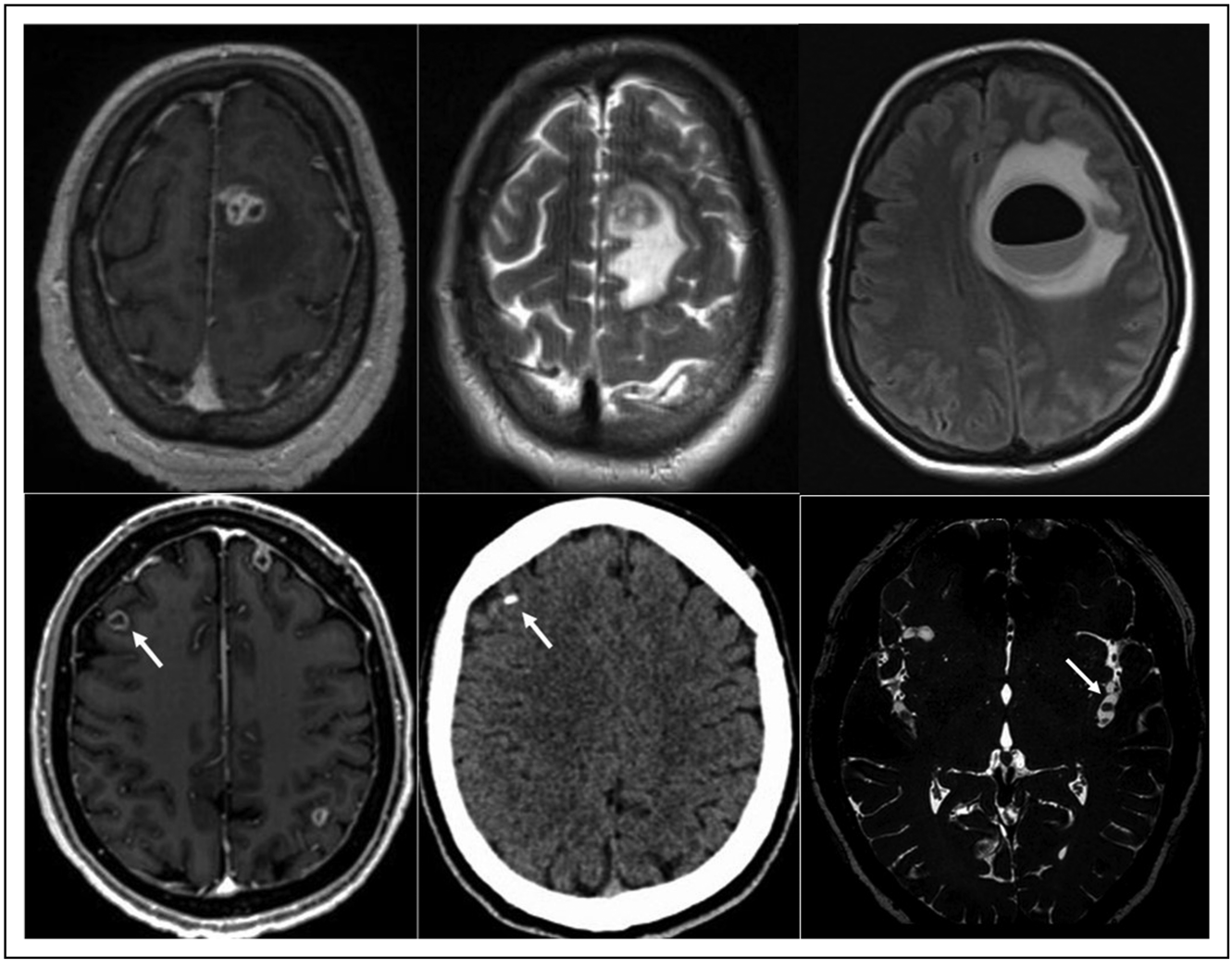

FIGURE 1. Atypical neuroimaging in neurocysticercosis. (a) Nonspecifically appearing inflamed, enhancing parenchymal lesions (above left and center); (b) atypical parenchymal cyst showing a density level (above right); (c) enhancing calcifications without edema (bottom left and center, arrow), and (d) small, likely early, subarachnoid lesions (bottom right, arrow).

monitor response to treatment. On the other hand, antigen detection confirms the presence of living parasite and can be used for therapeutic decisions [18▪,34]. The assay of choice for antibody detection is the immunoelectro transfer blot assay using lentil-lectin purified parasite glycoprotein antigens (LLGP-EITB), which is highly sensitive and specific, but the sensitivity drops in the setting of a single parenchymal lesion or calcified parenchymal disease. Serum is more sensitive than cerebrospinal fluid (CSF). On the other hand, serum ELISAs using crude antigens should be avoided as a diagnostic tool, especially in the setting where the imaging is not clear cut, as there is significant cross reaction with other parasitic infections. Antigen detection requires monoclonal antibody-based ELISA assays [35,36▪▪,37] PCR assays in serum, CSF and urine have been reported, but more information is yet needed to assess the added value of molecular testing [38▪,39–41].

The clinical differences between parenchymal and extraparenchymal NCC are known for more than a century. However, the knowledge of the relationships between type of disease, antibody response, and circulating levels of parasite antigens has not reflected in the development of pragmatic diagnostic and treatment approaches [42]. Departing from neuroimaging, we should have a stage-specific approach to diagnosis, including immunodiagnostic information [43,44]. Discrepancies between diagnostic techniques occur in some types of disease because of the intrinsic nature of each tool (i.e. the increased sensitivity of CT for calcified lesions), but in general, these follow predictable patterns, as sketched in Table 1.

Table 1. Diagnostic positivity of neuroimaging and immunodiagnostic tests by type of neurocysticercosis

	Imaging		Serology	
Parenchymal	**CT**	**MRI**	**Antigen**	**Antibody**
Viable cysts (1 or 2)	+[a]	++	−/+	+
Multiple viable cysts	+++	+++	++	++
Degenerating cyst, single	+[a]	++	−	−/+
Calcification	+++	−/+[b]	−	−/+
Extraparenchymal				
Intraventricular	−/+	++	+++	+++
Subarachnoid	−/+	++	+++	+++

CT, computed tomography.
[a]May miss small cysts.
[b]Best seen on MRI susceptibility-weighted images.

So, whenever imaging suggests only resolved or nonviable NCC and serology does not seem compatible (enzyme-linked immunoelectrotransfer blot (EITB) is strongly positive and/or or Ag-ELISA is clearly positive), it would be advisable to further review existing image data or add new imaging examinations, and also vice versa, when neuroimages suggest subarachnoid disease or multiple viable cystic disease and serology is negative, it should raise doubts on the cause. A major challenge to establishing the diagnosis on the patient level or estimating the burden of disease in endemic regions is the neuroimaging examinations driving the diagnosis of NCC, CT, and particularly MRI, are poorly available or too expensive. Access to EITB and monoclonal antibody-based antigen detection ELISA commercial assays is also limited in most of the world, compounding this dilemma.

CHALLENGES IN THE MANAGEMENT OF NEUROCYSTICERCOSIS

Parenchymal neurocysticercosis

A significant drawback in the management of parenchymal NCC is incomplete resolution of viable cysts following ABZ therapy, but the combined use of ABZ with praziquantel (PZQ) has greatly improved the efficacy of cysticidal regimens in the treatment of three or more viable cysts in the parenchyma. This synergy is not seen in individuals with one to two cysts treated with dual therapy, suggesting that the additive effect seen in greater cyst burden somehow involves boostering the immune response of the host resulting in improved resolution of cysts [45▪,46,47].

Another management conundrum is the persistence of active seizure disorders after all cysts have resolved, likely a composite result of infection burden, cyst characteristics, initial inflammatory response, and response to immunomodulatory therapy. After antiparasitic treatment of parenchymal disease, the cyst eventually collapses and is replaced by fibrotic tissue as it transforms into a granuloma that results in a calcified granuloma in 38% of cases [20,48,49]. On CT scan, they appear as small hyperdense lesions with or without enhancement. Perilesional edema may also be present around calcifications and is usually associated with symptoms, especially seizures. Improving approaches to better manage perilesional [50,51] inflammation to reduce the likelihood of chronic brain damage and scarring are required. The current approach involves steroids, which are frequently associated with significant side effects. Future efforts should target inflammation or calcification processes itself to reduce the likelihood of leaving residual calcified scars. Appropriate antiseizure regimes (including close monitorization of adherence), and reasoned approaches to antiseizure medication (ASM) withdrawal (not before two seizure-free years) are mandatory [46,52,53]. Guidelines on how to withdraw ASM in NCC are not based on evidence [54].

The association between NCC and hippocampal sclerosis (HAS) is being reported with increased frequency [55–57]. Although both are common conditions in endemic regions and may be coincidental, there is increasing evidence to suggest that NCC is involved in the pathogenesis of HAS. In a population-based, case–control study in Ecuador, HAS was more common in those individuals with calcified NCC than controls and when stratified by age, the association was stronger in older age groups [58]. In a cross-sectional study in Peruvian clinical patients and rural Ecuadorian village, individuals with calcified NCC were three times more likely to have HAC

on MRI than those with cystic NCC or healthy individuals. These studies suggests that it takes years for HAS to develop, but the pathophysiological mechanisms are not clear. Two possible explanations include repetitive subclinical seizure activity due to the calcified lesion or recurrent inflammation provoked by 'leaking' of antigens previously trapped within the calcifications [59].

A commonly neglected aspect in the management of patients with NCC is the frequent presence of comorbidities or nonepileptic manifestations (depression, altered cognition). These should be systematically assessed and monitored [60–62].

Extraparenchymal neurocysticercosis

Patients with extraparenchymal NCC frequently present for care more than a decade after infection, with extensive parasitic infiltration of the basal and interhemispheric spaces. Mass effects, hydrocephalus, and intracranial hypertension mark the clinical expression [63,64]. The efficacy of a first course of antiparasitic drugs is much lower than in parenchymal disease. The combination of ABZ with PZQ has been used in several case series in several centers, but controlled data is missing. A double-blind trial comparing ABZ with ABZ+PZQ has been performed in Peru and should issue results soon.

Unlike parenchymal NCC, higher doses and longer anti-inflammatory control are needed in extraparenchymal NCC to control inflammatory response to avoid severe intracranial hypertension that could lead to herniation and death of the patient [65▪,66,67,68▪]. Anti-inflammatory control reduces the likelihood of stroke due to chronic arachnoiditis, which may occur while tapering steroids. This leads to very prolonged courses of steroids and many times patents become steroid-dependent. Methotrexate and etanercept raise as alternative anti-inflammatory/immunosuppressive agents to limit steroid side effects.

The presence of *T. solium* antigen in the CSF and serum indicates viable cysticercosis infection, and antigen levels are usually very high in subarachnoid NCC [69]. Treatment decisions in SANCC are guided by complimentary measures, which include serial imaging in conjunction with monitoring serum antigen levels. A fall in cestode antigen to undetectable levels in the CSF predicts cure. More recently, serial measurements of CSF *T. solium* DNA by qPCR in patients treated for SANCC that become undetectable have been shown to predict inactive disease status or cure [70]. Prolonged and intensive therapy is needed, requiring considerable resources and expenses, including medication and testing, invasive lumbar punctures, side-effects of corticosteroids. Combined use of ABZ with PZQ has been reported in case reports or cases series, with good results, although controlled data is not available. Results from a large randomized trial in Peru comparing ABZ alone to combined ABZ with PZQ in SANCC should be available in the following months. Debulking surgery prior to treatment has been reported, which may limit prolonged and intensive therapy, which is expensive and can lead to corticosteroid side-effects [71▪].

CHALLENGES IN FOLLOW-UP

So far, seroconversion to negative in antigen or PCR assays seems to mark the cure of the disease, although there is a need for long-term follow-up of large series confirm its predictive value. Antibody may persist for many years after parasite resolution, particularly in patients with a strong antibody response at baseline. Clinicians should be aware of late seizure relapses. Data from published series shows that seizure relapses in parenchymal NCC may occur many years after initial seizure remission. Also, considering the very long prepatent period of subarachnoid NCC; if neurological symptoms reappear years after treatment of parenchymal NCC, patients should be screened to rule out extraparenchymal disease. There is scarce data on long-term sequelae of cured subarachnoid NCC, particularly on late cognitive decline or the risk of secondary stroke.

CONCLUSION

Appropriate use of imaging and serology data in the context of the specific type of NCC should improve diagnostic precision. Further data should become available on the improved efficacy of combined antiparasitic therapy in SANCC as well as confirming its safety. In parenchymal NCC, early initiation of ASM and careful monitoring of its effect should be combined with optimizing anti-inflammatory therapy in order to minimize perilesional damage and reduce the risk of epilepsy. More attention should be paid to the assessment of comorbidities and other disease manifestations such as cognition deficits and depression.

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Conflicts of interest

There are no conflicts of interest.

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