Am. J. Trop. Med. Hyg., 97(3), 2017, pp. 653–657
doi:10.4269/ajtmh.17-0069

Review Article

Reliability of Diagnostic Criteria for Neurocysticercosis for Patients with Ventricular Cystic Lesions or Granulomas: A systematic review

Javier A. Bustos,[1,2]† Hector H. García,[1,2]† and Oscar H. Del Brutto[3]*†

[1]*Department of Microbiology, Center for Global Health – Tumbes, Instituto Nacional de Ciencias Neurológicas, Lima, Peru;* [2]*Cysticercosis Unit, Instituto Nacional de Ciencias Neurológicas, Universidad Peruana Cayetano Heredia, Lima, Peru;* [3]*School of Medicine, Universidad Espíritu Santo – Ecuador, Guayaquil, Ecuador*

Abstract. Intraventricular neurocysticercosis (NCC) is a severe form of NCC requiring prompt diagnosis and treatment. We aimed to assess the reliability of the most recent version of diagnostic criteria for this form of NCC. Two systematic literature reviews were performed; one included case reports of patients with intraventricular cysticercosis and the other included case reports of patients with intraventricular cystic lesions or granulomas caused by infections other than NCC. All assessed cases were categorized according to the last revision of the long-standing Del Brutto's set of diagnostic criteria to determine its sensitivity, specificity, and predictive value for this form of NCC. The search disclosed 128 patients with intraventricular NCC and 41 with other infections. The set of diagnostic criteria classified as definitive NCC 93 cases with intraventricular NCC (sensitivity 72.7%, 95% CI, 63.9–79.9%), as well as four cases with other infections (specificity 90.2%, 95% CI, 75.9–96.8%). The positive and negative predictive values of the criteria were 0.96 (95% CI, 0.89–0.99) and 0.51 (95% CI, 0.39–0.63), respectively. The revised Del Brutto's set of diagnostic criteria for NCC is acceptably sensitive and highly specific for diagnosing patients with the ventricular form of the disease.

INTRODUCTION

Diagnostic criteria for neurocysticercosis (NCC) aim to standardize the diagnostic approach to this common helminthic infection of the nervous system and reduce the risk of misdiagnosis that occurs when epidemiological data, clinical manifestations, and results from complementary tests are independently used to confirm or discard the disease. The first set of diagnostic criteria for cysticercosis, published in 1996, considered the diagnosis of both systemic cysticercosis and NCC.[1] Then, a second version, confined to the diagnosis of NCC, was reported in 2001.[2] The latter used four categories of diagnostic criteria (absolute, major, minor, and epidemiologic) stratified according to their diagnostic strength, and two degrees of diagnostic certainty (definitive and probable) based on the possibility that the disease is present in a given patient. These criteria have proved useful for the diagnosis of NCC in both hospital and field settings and have been widely used in endemic as well as in nonendemic areas, being—according to Scholar Google—the most frequently cited reference in NCC, with more than 760 quotations up to June 2017.

More recently, our group presented a revised version of diagnostic criteria, updated to provide simpler operational definitions and to incorporate modern advances that have improved the diagnosis of NCC.[3] This revised set was structured in absolute, neuroimaging, and clinical/exposure criteria. As in the 2001 set, proper interpretation of these criteria allows two degrees of diagnostic certainty, definitive and probable.

Some forms of presentation of NCC are more severe than others and are, consequently, associated with worse prognosis. One of them, intraventricular NCC, deserves special attention as it is frequently associated with intracranial hypertension and may even cause sudden death.[4–6] Therefore, efforts should be directed to recognize this condition as efficiently as possible. We conducted a systematic review of the literature of all published cases of intraventricular NCC to assess the sensitivity of the revised Del Brutto's set of diagnostic criteria to diagnose this form of the disease. For specificity assessment, we reviewed all reported cases of primary intraventricular cystic lesions or granulomas related to infections other than NCC.

METHODS

Search strategy. Two systematic literature reviews were performed following the recommendations of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines,[7] one for sensitivity and the other for specificity assessment. For the former, we reviewed individual case reports of patients with intraventricular cysticercosis and for the latter, we reviewed individual case reports of patients with intraventricular cystic lesions or granulomas caused by infections other than NCC. The following paragraphs describe the search strategies in detail.

Sensitivity assessment. Using the combined key words "*cysticercosis*" or "*neurocysticercosis*" and "*ventricular*" or "*intraventricular*" or "*ventricle*", we conducted a search of the English literature (January 1990 to December 2016) of patients with this form of the disease, by the use of the electronic databases of MEDLINE (http://www.pubmed.gov), EMBASE (http://www.embase.com), Global Index Medicus, regional indices (http://www.globalhealthlibrary.net), Scopus (https://www.scopus.com), and the Web of Science (http://apps.webofknowledge.com). No additional limits or filters were applied to the search; instead, abstracts of all articles as well as clinical notes without an abstract were read to identify potentially eligible reports. Thereafter, a manual search that included the authors' files as well as the list of references of already selected articles was undertaken to find papers not included in these electronic databases or not detected during

* Address correspondence to Oscar H. Del Brutto, Air Center 3542, PO Box 522970, Miami, FL 33152-2970. E-mail: oscardelbrutto@hotmail.com
† These authors contributed equally to this work.

the initial search. Selected articles were those reporting original data on individual patients with *Taenia solium* cysticercosis located in the ventricular cavities with the inclusion of a published figure of a CT or MRI supporting this assertion. Articles presenting index case(s) as part of a series of patients with ventricular NCC were also considered eligible.

Potentially included articles were independently reviewed by two of the authors (J.A.B. and O.H.D.), and disagreements in inclusion criteria or data abstraction were resolved by consensus with another author (H.H.G.). Abstracted data of selected articles included citizenship as well as the migratory or travel status of patients, demographic profile, clinical manifestations, results from immunological tests and CSF analysis (if available), specific location of parasites within the ventricular cavities, visualization of the scolex within the cyst, associated neuroimaging findings (hydrocephalus and coexistence of other forms of NCC such as parenchymal brain cysts or calcifications and subarachnoid cysts or spinal cysts), and response to cysticidal drugs (when used before surgery). Histopathological confirmation was recorded in those who underwent surgical resection of lesions. Abstracted data were used to categorize included patients into definitive or probable cases of NCC according to the revised set of diagnostic criteria.[3]

Specificity assessment. To assess the specificity of the revised set of diagnostic criteria for NCC, we also searched the previously mentioned electronic databases, focusing on papers in the English language (January 1990 to December 2016) that included individual information of patients with acquired infections of the CNS (other than NCC) potentially associated with a primary infection of the ventricular system in the form of cystic or granulomatous lesions.[8] Combined key words included "*cerebrum*" or "*cerebral*" or "*brain*", "*ventricular*" or "*intraventricular*" or "*ventricle*", and "*abscess*" or "*pyogenic abscess*" or "*tuberculomas*" or "*tuberculosis*" or "*mycobacterium*" or "*mycotic granuloma*" or "*cryptococcoma*" or "*toruloma*" or "*cryptococcosis*" or "*coccidioidomycosis*" or "*paracoccidioidomycosis*" or "*blastomycosis*" or "*blastomyces*" or "*histoplasmosis*" or "*histoplasma*" or "*aspergillosis*" or "*aspergillus*" or "*candida*" or "*candidiasis*" or "*mucormycosis*" or "*pseudallescheria*" or "*parasitic*" or "*hydatid*" or "*echinococcus*" or "*echinococcosis*" or "*sparganum*" or "*sparganosis*" or "*coenurus*" or "*coenurosis*" or "*toxoplasmosis*" or "*toxoplasma*" or "*schistosoma*" or "*schistosomiasis*". A manual search of relevant literature, similar to that used for sensitivity assessment, was also undertaken to detect additional papers not indexed in these electronic databases.

Selected articles were those that reported original data on individual patients with primary ventricular infections of the CNS with the inclusion of a published figure of a CT or MRI supporting this assertion, confirmed by histopathological examination or a clear response to specific anti-infective agents. Infants (≤ 1 year of age), patients with focalized parenchymal brain abscess ruptured to the ventricular cavities, and those with secondary infections of already implanted ventricular shunts were not included. Patients with evidence of systemic disease caused by the same microorganism responsible for the ventricular lesion were excluded. Likewise, patients known to be infected by HIV were not considered, as AIDS often presents with infections related to several microorganisms at different times, and neuroimaging findings might be equivocal.

The process of selection of these papers was similar to that described for sensitivity assessment (two authors independently reviewed all papers, and discrepancies were resolved by consensus with all authors). Abstracted data focused on epidemiological and demographic data together with clinical manifestations, neuroimaging findings, and results of immune diagnostic tests and CSF analysis (if available). The revised set of diagnostic criteria for NCC was applied to all selected cases to determine how many of them could be erroneously included as a definitive case of NCC.

RESULTS

Selected articles (and patients). Figures 1 and 2 summarize the selection process. For sensitivity assessment, database search initially identified 453 papers, of which 105 described individual patients with intraventricular NCC. Manual search allowed the identification of 13 additional reports. These 118 papers (listed in Supplemental Appendix 1) reported a total of 138 patients, 10 of which did not include a figure showing neuroimaging findings and were thus excluded. For specificity assessment, database search identified an initial list of 980 papers, 44 of which described individual patients aged > 1 year, with focal intraventricular infections other than NCC, and no evidence of HIV infection. Manual search allowed the identification of six additional reports. These 50 papers (listed in Supplemental Appendix 2) reported a total of 52 cases, 11 of which were excluded; four because a figure showing neuroimaging findings was not published, and seven because they had evidence of systemic disease manifestations before the onset of neurological manifestations corresponding to the ventricular lesion or during the work-up at the time of admission.

Characteristics of patients with intraventricular NCC. Age was available in 125 of the 128 cases (mean age: 32.3 ± 14.4 years) and gender in 124 (49% women). Sixty-five patients (51%) were resident of countries where the disease is endemic (mostly India, Mexico, China, and Brazil), 47 (37%) were immigrants (or visitors) from endemic to nonendemic areas (mostly Latin American countries to the United States), and three were travelers from nonendemic to endemic countries. Epidemiological information was not available in the 13 remaining cases (12 were reports originated from the United States and one from Spain).

Headache (acute, subacute, or chronic) was the most common form of presentation of the disease (110 cases, 86%). This was associated with vomiting, decreased level of consciousness, altered mental status, or blurred vision in 81% of cases (only a few patients had associated focal neurological deficits). Results from fundoscopic examination were specified in only 40 cases, 31 of whom had papilledema. Only 15 patients had seizures, which were more frequently noticed in those with associated parenchymal brain cysts or calcifications.

Cysts were most often located within the IV ventricle (49 cases), followed by the III (36 cases), and lateral (34 cases) ventricles. An uncommon location was the cavum septum pellucidum (four cases). Five additional patients had cysts in more than one ventricular cavity. Sixteen patients (13%) had the so-called "ventricular migration sign," that is, visualization of the cyst within a different ventricular cavity on sequential neuroimaging studies. An eccentric dot within the cyst,

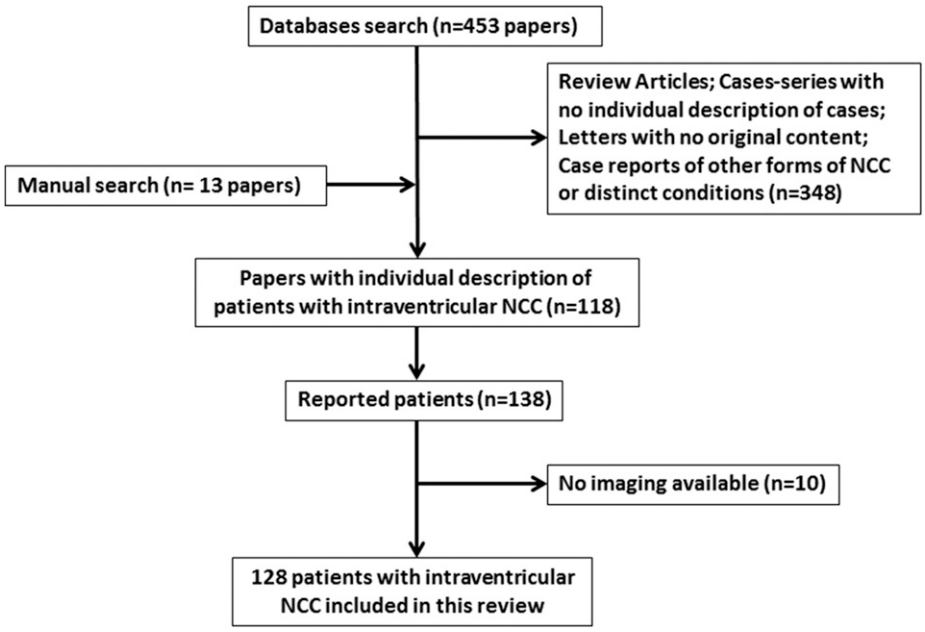

FIGURE 1. Flow diagram for identification of patients with intraventricular neurocysticercosis.

consistent with the parasite's scolex, was noticed in 53 cases (41%). In five of these cases, the scolex was not visualized within the ventricular cyst but in associated parenchymal or subarachnoid cysts. Obstructive hydrocephalus (symmetrical or asymmetrical) was noticed in 116 patients (91%). Neuroimaging evidence of other forms of NCC were found in 51 cases (40%), being the most common subarachnoid cysts (N = 22), parenchymal brain calcifications (N = 16), and parenchymal brain cysts (N = 14). A few additional patients had associated spinal cysts (N = 4), ependymitis (N = 4), or intrasellar cysts (N = 2). Of note, several of these patients had more than one form of the disease. Two patients had a pituitary adenoma as an incidental finding (one of them also had a meningioma).

Results of cytochemical CSF analysis were reported in 31 cases, 21 of which demonstrated abnormalities consistent with an inflammatory process (increased lymphocytes and protein contents). Immunodiagnostic tests for recognition of anticysticercal antibodies were reported in only 41 patients (18 in CSF, 11 in serum, seven in both CSF and serum, and five not

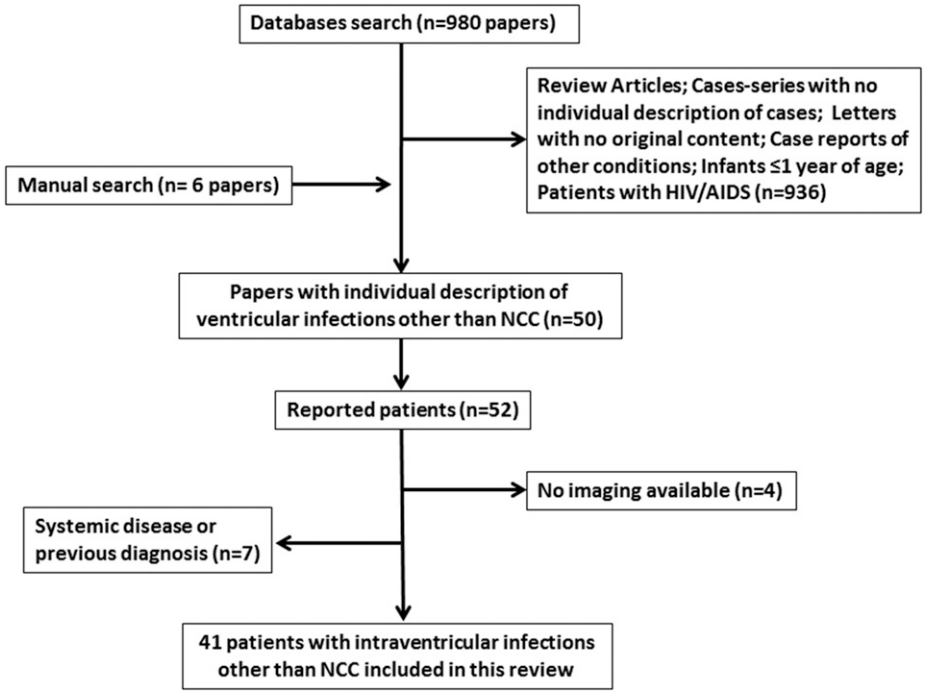

FIGURE 2. Flow diagram for identification of patients with infections of the ventricular system other than neurocysticercosis.

specified). ELISA was performed in 19 cases (positive in 15), enzyme-linked immunoelectro transfer blot (EITB, western blot) in eight (positive in six), and the complement fixation test in one (negative). In the remaining cases, there were no details on the type of immune tests performed, although they were reported as positive.

Characteristics of patients with other ventricular infections. The mean age of the 41 patients was 26.7 ± 18.3 years, 27 (66%) were women, and 22 of them (54%) were residents of, or migrated from, countries where cysticercosis is endemic. Headache (acute, subacute, or chronic) was the most common clinical manifestation (32 cases, 78%), and was often associated with vomiting, decreased level of consciousness, altered mental status, or blurred vision. Only six patients had associated focal neurological deficits and four had seizures. Results from fundoscopic examination were specified in only 16 cases, 14 of which had papilledema.

Intraventricular lesions were most often located within the lateral ventricles (36 cases, 88%), in the III ventricle in four (one patient had mixed lateral and III ventricle lesions), and in the IV ventricle in two. Obstructive hydrocephalus (symmetrical or asymmetrical) was noticed in 39 patients (95%), and four patients had associated parenchymal brain enhancing lesions. Cytochemical analysis of the CSF was reported in 27 cases, 24 of which demonstrated abnormalities consistent with an inflammatory process. Four patients had immunological diagnostic tests for recognition of anticysticercal antibodies and were negative in all cases.

The diagnosis was confirmed by surgical resection and subsequent histopathological examination in 32 patients and by resolution of lesions after administration of specific therapy in the remaining nine. The final diagnosis was tuberculoma in 12 cases, fungal granuloma in 12 (mostly cryptococcomas), cystic hydatid disease in nine, and pyogenic abscess in the remaining eight.

Application of diagnostic criteria. For patients with intraventricular NCC, the set allowed a definitive diagnosis of NCC in 93 cases (72.6%). Thirty additional patients were categorized as probable NCC; and in the five remaining patients, the information provided in the report did not permit the recognition of the case for inclusion within the probable NCC category (Supplemental Appendix 3). The set erroneously identified four patients with other infections (two with cryptococcosis, one with tuberculomas and the other with hydatid disease) as definitive NCC. In all these cases, misdiagnosis was related to the associated presence of parenchymal brain cystic or ring-enhancing lesions.

Reliability assessment of the revised diagnostic criteria. Reliability of the revised Del Brutto's set of criteria for a definitive diagnosis of intraventricular cysticercosis was assessed by calculating its sensitivity and specificity, as well as its positive and negative predictive value (using numbers of true and false positive NCC cases, and those of true and false negative patients with infections other than NCC). The set allowed a definitive diagnosis in 93/128 patients with intraventricular NCC, and in 4/41 of those with other infections. These numbers yielded the set a sensitivity of 72.7% (95% CI, 63.9–79.9%), a specificity of 90.2% (95% CI, 75.9–96.8%), a positive predictive value of 0.96 (95% CI, 0.89–0.99), and a negative predictive value of 0.51 (95% CI, 0.39–0.63).

DISCUSSION

NCC is defined as the infection of the CNS and its coverings by the larval stage of *Taenia solium,* the pork tapeworm. Parasites may locate anywhere in the CNS, including the brain or spinal cord parenchyma, the subarachnoid space (cranial or spinal), the ventricular system, the sellar region, the subdural space, the optic nerves, or the retina.[9] According to seminal pathological reports, cysticerci enter the cerebral ventricles through the choroid plexus of the lateral ventricles and then move downward within the cavities until their growing size preclude further migration, or when they get attached to the ependymal lining by the occurrence of a secondary inflammatory reaction (granular ependymitis).[10,11]

Clinical manifestations of intraventricular NCC are most often related to the obstruction of CSF transit by the effect of parasites occluding the foramina of Monro, the cerebral aqueduct, or the foramina of Luschka and Magendie. This causes a syndrome of intracranial hypertension of subacute onset, which can be associated—particularly in patients with simultaneous involvement of the brain parenchyma or the subarachnoid cisterns at the base of the brain—with focal neurological deficits or seizures.[12–14] A particular form of presentation of ventricular NCC is the so-called Bruns syndrome, characterized by headache, sudden positional vertigo, and loss of consciousness related to rotatory movements of the head.[15] Although many patients recover after these episodes, some may die as a result of acute hydrocephalus.[16]

Several external groups[17–19] have proposed modified versions of the original 2001 Del Brutto's set of diagnostic criteria for NCC.[2] One of them focused on the diagnosis of NCC in populations from the Indian subcontinent.[17] Another proposal included antigen detection by ELISA in the absence of neuroimaging to confirm the diagnosis of NCC, an important misconception because immunological tests must be interpreted in the light of neuroimaging findings.[18] Yet, another set presented a specific category for the diagnosis of extraparenchymal NCC.[19] According to that category, a definitive diagnosis of extraparenchymal NCC can be done in patients presenting with subarachnoid or intraventricular cysts (not with an evident scolex) associated with at least two of the following: 1) hydrocephalus, 2) inflammatory CSF, 3) positive CSF immunological tests (ELISA or EITB), and 4) presence of single or multiple calcifications or parenchymal vesicular or degenerating cysts. When applying that modified set of diagnostic criteria to the patients reviewed here, it recognizes as definitive NCC 79 of 128 cases with ventricular NCC and 25 of 41 cases of other infections, yielding a sensitivity of 61.7% (95% CI, 52.7–70%) and a specificity of only 39% (95% CI, 24.6–55.5%). The poor specificity results from the fact that any patient with an intraventricular cyst or granulomatous lesion associated with hydrocephalus and an inflammatory CSF would be characterized as "definitive NCC." Both hydrocephalus and abnormalities in the cytochemical analysis of CSF are unspecific, and many patients with other infections requiring urgent medical therapy (i.e., brain abscess, tuberculomas, mycotic granulomas) might be misdiagnosed as NCC if that set is used.[20]

Major strengths of the present study include the systematic review of the literature of patients with intraventricular NCC as

well as that of patients with cystic or granular intraventricular lesions caused by infections other than NCC, and the high inter-rater agreement in the application of the revised Del Brutto's set of diagnostic criteria ($k = 0.981$, 95% CI: 0.94–1). However, the study has limitations. Publication bias may have affected the type of cases, over-representing those with more complex intraventricular lesions because of their rarity. Likewise, a series based on the international literature is likely incomplete because many cases have probably been published in local journals or not reported at all. Another limitation is the incompleteness of information provided in several case reports. Indeed, results of immune diagnostic tests were available in only 24% of NCC cases and, in some of them, there were no details on the type of test performed (ELISA or EITB). In addition, epidemiological data were incomplete as there was little mention on the tapeworm carrier status of the patient's household contacts (instead, some reports erroneously mentioned that the patient did not eat pork). Human cysticercosis is most often transmitted person-to-person, from *Taenia solium* carriers to healthy individuals, through nonhygienic handling of food or by direct contact with human feces.[21] Ingestion of raw or undercooked pork contaminated with cysticerci as the cause of human cysticercosis is a misbelief. The role of pigs is to maintain the infection cycle by causing human taeniasis.

In addition, evidence of systemic cysticercosis (muscle "cigar-shaped" calcification or subcutaneous nodules) was not mentioned at all, although many of the reported patients came from the Indian subcontinent and Southeast Asia, where systemic disease (including mass infestations) are not rare. Likewise, results from fundoscopic examination were described in only one-third of cases, and it is possible that a few subretinal cysts could have been missed. Indeed, the revised set of diagnostic criteria was not intended to be just a neuroimaging catalog but to integrate neuroimaging data together with clinical and exposure information. Therefore, it is highly recommended that authors publishing case reports on NCC make available all pertinent information for proper interpretation of findings in subsequent reviews. Incomplete information in regard to data of etiological, epidemiological, and clinical relevance preclude appropriate assessment and evaluation of the presumed diagnoses. Compulsory reporting of NCC cases to agencies such as the Pan American Health Organization or the World Health Organization would contribute to systematize data collection on the presentation of NCC and its subtypes.[22]

In summary, this systematic literature review provides evidence on the reliability of the revised Del Brutto's set of diagnostic criteria for diagnosing patients with intraventricular NCC, data that must be further supported by prospective studies.

Received January 27, 2017. Accepted for publication June 20, 2017.

Published online July 31, 2017.

Note: Supplemental appendices appear at www.ajtmh.org.

Financial support: J. A. B. is partially supported by FIC-NIH grant TW001140 and O. H. D. is supported by Universidad Espíritu Santo – Ecuador.

Authors' addresses: Javier A. Bustos, Universidad Peruana Cayetano Heredia, Center for Global Health, Lima, Peru, E-mail: javier.bustos@jhu.edu. Hector H. García, Department of Microbiology, School of Sciences, Universidad Peruana Cayetano Heredia, Centro de Salud Global Tumbes, Lima, Peru, E-mail: hgarcia1@jhu.edu. Oscar H. Del Brutto, Hospital-Clinica Kennedy, School of Medicine, Universidad Espiritu Santo – Ecuador, Guayaquil, Ecuador, E-mail: oscardelbrutto@hotmail.com.

REFERENCES

1. Del Brutto OH, Wadia NH, Dumas M, Cruz M, Tsang VC, Schantz PM, 1996. Proposal of diagnostic criteria for human cysticercosis and neurocysticercosis. *J Neurol Sci 142:* 1–6.
2. Del Brutto OH, et al., 2001. Proposed diagnostic criteria for neurocysticercosis. *Neurology 57:* 177–183.
3. Del Brutto OH, Nash TE, White AC Jr, Rajshekhar V, Wilkins PP, Singh G, Vazquez CM, Salgado P, Gilman RH, Garcia HH, 2017. Revised diagnostic criteria for neurocysticercosis. *J Neurol Sci 372:* 202–210.
4. Keane JR, 1984. Death from cysticercosis. Seven patients with unrecognized obstructive hydrocephalus. *West J Med 140:* 787–789.
5. Cuetter AC, Andrews RJ, 2002. Intraventricular neurocysticercosis: 18 consecutive patients and review of the literature. *Neurosurg Focus 12:* e5.
6. Hortobágyi T, Alhakim A, Biedrzycki O, Djurovic V, Rawal J, Al-Sarraj S, 2009. Cysticercosis of the fourth ventricle causing sudden death: a case report and review of the literature. *Pathol Oncol Res 15:* 143–146.
7. Moher D, Liberati A, Tetzlaff J, Altman DG, PRISMA Group, 2009. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *Ann Intern Med 151:* 264–269.
8. Magill AJ, Ryan ET, Hill DR, Solomon T, 2013. *Hunter's Tropical Medicine and Emerging Infectious Diseases,* 9th edition. China: Elsevier Inc.
9. Del Brutto OH, Garcia HH, 2014. *Cysticercosis of the Human Nervous System.* Berlin, Germany: Springfield-Verlag.
10. Escobar A, Nieto D, 1972. Parasitic diseases. Minckler J, ed. *Pathology of the Nervous System.* Vol. 3. New York, NY: McGraw-Hill Book Company.
11. Pittella JEH, 1997. Neurocysticercosis. *Brain Pathol 7:* 681–693.
12. Cuetter AC, Garcia-Bobadilla J, Guerra LG, Martinez FM, Kaim B, 1997. Neurocysticercosis: focus on intraventricular disease. *Clin Infect Dis 24:* 157–164.
13. Cuetter AC, Andrews RJ, 2002. Intraventricular neurocysticercosis: 18 consecutive patients and review of the literature. *Neurosurg Focus 12:* 5.
14. Kumar R, Kalra SK, Mahapatra AK, 2008. Intraventricular neurocysticercosis cyst(s) in Indian children. *Pediatr Neurosurg 44:* 118–123.
15. White AC Jr, 2015. Bruns syndrome is a common presentation of neurocysticercosis. *Am J Trop Med Hyg 93:* 672.
16. Jimenez Caballero PE, Mollejo Villanueva M, Marsal Alonso C, Alvarez Tejerina A, 2005. Síndrome de Bruns: descripción de un caso de neurocisticercosis con estudio anatomopatológico. *Neurologia 20:* 87–89.
17. Garg RK, 2004. Diagnostic criteria for neurocysticercosis: some modifications are needed for Indian patients. *Neurol India 52:* 171–177.
18. Gabriel S, Blocher J, Dorny P, Abatih EN, Schmutzhard E, Ombay M, Mathias B, Winkler AS, 2012. Added value of antigen ELISA in the diagnosis of neurocysticercosis in resource poor settings. *PLoS Negl Trop Dis 6:* e1851.
19. Carpio A, et al., 2016. New diagnostic criteria for neurocysticercosis: reliability and validity. *Ann Neurol 80:* 434–442.
20. Gilman RH, 2016. Diagnostic criteria for neurocysticercosis—a difficult update. *Nat Rev Neurol 12:* 560–561.
21. Lescano AG, Garcia HH, Gilman RH, Gavidia CM, Tsang VC, Rodrigues S, Moulton LH, Villaran MV, Montano SM, Gonzalez AE; Cysticercosis Working Group in Peru, 2009. *Taenia solium* cysticercosis hotspots surrounding tapeworm carriers: clustering on human seroprevalence but not on seizures. *PLoS Negl Trop Dis 3:* e371.
22. Román GC, et al., 2000. A proposal to declare neurocysticercosis an international reportable disease. *Bull World Health Organ 79:* 399–406.