High frequency of spinal involvement in patients with basal subarachnoid neurocysticercosis

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ABSTRACT

Objective: To determine the frequency of spinal neurocysticercosis (NCC) in patients with basal subarachnoid NCC compared with that in individuals with viable limited intraparenchymal NCC (≤ 20 live cysts in the brain).

Methods: We performed a prospective observational case-control study of patients with NCC involving the basal cisterns or patients with only limited intraparenchymal NCC. All patients underwent MRI examinations of the brain and the entire spinal cord to assess spinal involvement.

Results: Twenty-seven patients with limited intraparenchymal NCC, and 28 patients with basal subarachnoid NCC were included in the study. Spinal involvement was found in 17 patients with basal subarachnoid NCC and in only one patient with limited intraparenchymal NCC (odds ratio 40.18, 95% confidence interval 4.74–340.31; $p < 0.0001$). All patients had extramedullary (intradural) spinal NCC, and the lumbosacral region was the most frequently involved (89%). Patients with extensive spinal NCC more frequently had ventriculoperitoneal shunt placement (7 of 7 vs 3 of 11; $p = 0.004$) and tended to have a longer duration of neurologic symptoms than those with regional involvement (72 months vs 24 months; $p = 0.062$).

Conclusions: The spinal subarachnoid space is commonly involved in patients with basal subarachnoid NCC, compared with those with only intraparenchymal brain cysts. Spinal cord involvement probably explains serious late complications including chronic meningitis and gait disorders that were described before the introduction of antiparasitic therapy. MRI of the spine should be performed in basal subarachnoid disease to document spinal involvement, prevent complications, and monitor for recurrent disease. *Neurology*® 2012;78:1394–1400

GLOSSARY

AED = antiepileptic drug; **BS** = basal subarachnoid neurocysticercosis; **FLAIR** = fluid-attenuated inversion recovery; **IP-NCC** = intraparenchymal neurocysticercosis; **NCC** = neurocysticercosis; **OR** = odds ratio.

Neurocysticercosis (NCC), a parasitic infection of the brain, is caused by the larval stage (cysticercus) of the pork tapeworm *Taenia solium*.¹ Subarachnoid NCC, a form of extraparenchymal involvement, includes cysts in the spaces over the cerebral convexities, small cysts in fissures, or larger cysts in the Sylvian fissures not infrequently leading to mass effects.^{2,3} Involvement of the basal cisterns is a very morbid form because the parasite is commonly aberrant (racemose cyst) consisting of a proliferating membranous vesicular structure that invades the spaces of the brain. In addition, basal subarachnoid NCC (BS-NCC) is severe because it results in mass effects and incites an intense inflammatory response causing arachnoiditis resulting in hydrocephalus, nerve entrapments, vasculitis, and strokes.^{4–8}

MRI has increased the detection of a variety of spinal lesions.⁹ Spinal involvement may appear as ill-defined masses, well-defined cysts, or masses of multilocular cysts with low inten-

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Supplemental Data

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sity on T1-weighted sequences, high signal intensity on T2-weighted sequences, and low intensity on fluid-attenuated inversion recovery (FLAIR) sequences, or diffuse arachnoiditis.^{10–13} Spinal NCC may be intramedullary or extramedullary (intradural or extradural); both intramedullary and extradural forms are rare, and most disease involves the subarachnoid spaces, which may result from direct CSF dissemination.^{14–19}

The reported frequency of spinal involvement vary from 0.25% to 5.8% but is usually not looked for in the absence of overt disease.^{14,20–23} Here, we report the results of a prospective case control study using MRI to assess the frequency of the simultaneous spinal involvement in patients with BS-NCC or limited intraparenchymal NCC (IP-NCC).

METHODS Patients. From May 2010 to December 2010, male or female individuals aged between 16 and 65 years attending the Cysticercosis Unit of the Instituto Nacional de Ciencias Neurológicas in Lima, Peru, with a diagnosis of BS-NCC or IP-NCC only as demonstrated by MRI or CT scan and confirmed by serology on Western blot, were invited to join the study. BS-NCC was defined as lesions of any size compromising the basal cisterns, seen as hypodense lesions on CT scan or seen on MRI as lesions with low intensity similar to that of CSF on T1-weighted sequences and high intensity on T2-weighted sequences or hypointense signal on FLAIR sequences. Individuals with NCC involving the interhemispheric brain space, the Sylvian fissure, or lateral ventricles were enrolled only if the basal cisterns were affected. Spinal involvement consisted of subarachnoid space-occupying cystic lesions in the subarachnoid space as noted for BS-NCC, changes due to inflammation or scarring such as clumping of nerves on enhancement if performed, and possible intramedullary, intradural-extramedullary, or extradural involvement similar to the descriptions for spinal NCC infection. Patients with IP-NCC had one or more intraparenchymal viable cysts, which were defined as circumscribed, rounded hypodense vesicles visible on CT scan or low-intensity signal lesions on T1-weighted sequences, high intensity on T2-weighted images and low intensity on FLAIR sequences. Patients with parenchymal cysts surrounded by irregular perilesional edema (or contrast enhancement) were included only if the presence of fluid was confirmed by MRI as defined above. Patients with IP-NCC and additional small well-defined intraventricular cysts in a lateral ventricle were also included in the IP-NCC group.

Exclusion criteria included a history of therapy with anticyclicercal drugs within the previous year (except for treated patients who demonstrated continued viability of cysts), isolated intraventricular NCC, more than 20 intraparenchymal brain cysts, a history of other disease of the spine or medulla (tuberculosis, brucellosis, tumors, or transverse myelitis), spine surgery, or unwillingness or inability to undergo a MRI scan including patients with metal objects in the brain, eye, or body, an increased blood creatinine level, a history of hypersensitivity to gadolinium, or pregnancy and inability or unwillingness of the

subject or legal guardian/representative to give written informed consent.

Patients were referred by their attending neurologists, and information regarding the duration of illness, surgical history, presence of hydrocephalus, treatments (antiparasitic, corticosteroid, and antiepileptic drug [AED] use) was collected. The study did not alter in any way the patient's clinical management except by providing additional information on potential involvement of the spine. The attending physician was solely responsible for decisions regarding the care of the patient.

Standard protocol approvals, registrations, and patient consents. The study and written consent forms were reviewed and approved by the institutional review boards of the Universidad Peruana Cayetano Heredia, the Instituto Nacional de Ciencias Neurológicas, and the National Institute of Allergy and Infectious Diseases, NIH. Informed written consent was obtained from all patients.

Procedures. MRI of the brain and the whole spinal cord was performed, using a 1.5-T MRI scanner (Magnetom Avanto; Siemens AG, Germany). Based on the literature and because of logistic and economic constraints, we prioritized the study to the cervical region and limited the full spine examination to a single T2-weighted sequence as described below. Initially a 3-dimensional FLAIR sequence in axial slices was obtained from the brain and axial images of the brain and the cervical spine with 2-mm thickness were acquired by 3-dimensional T2-weighted sampling perfection with application-optimized contrasts by using different flip angle evolutions sequences. Subsequently, weighted sagittal images of the posterior fossa, cervical, thoracic, and lumbar spinal segments were obtained by 2-dimensional turbo spin-echo sequences with 1.5-mm thickness also at T2. Finally, high-resolution weighted axial images were obtained by 3-dimensional volumetric interpolated breath-hold examination sequences at T1 after IV contrast medium administration (gadolinium), from the vertex to C7, and were reconstructed in 2-mm-thick contiguous transverse images. MRI scans were read by 2 independent neuroradiologists masked to the study, and the interpretations were reviewed by the study investigators. Disagreements were resolved by consensus.

Sample size. The sample size was calculated, taking into account a prevalence of 20% of BS-NCC in imaging at the Institute. Because of the prevalence of 50% IP-NCC, 1 of every 2 patients was enrolled by simple random sampling, until the total number of patients was reached. The sample size of this study was limited to 60 patients for each type of NCC (total of 120 patients). However, during the course of the study, the large differences of involvement between the groups allowed for less than the full number permitted.

Statistical analysis. Categorical variables were analyzed by a χ^2 test or Fisher exact test when appropriate. A χ^2 test was also used to evaluate the proportions of spinal involvement according to BS-NCC or IP-NCC. Continuous variables that were normally distributed were analyzed by Student *t* test, whereas non-normal variables were analyzed by the Mann-Whitney *U* test. All tests were 2-tailed. Statistical significance was defined as $p \leq 0.05$. All statistical analyses were performed using SPSS version 16.0 (SPSS, Chicago, IL).

RESULTS Patient population. Of 65 patients who consented to participate, 6 were lost to follow-up before having the MRI examination performed. All of

Table 1 Characteristics of study patients

	Limited intraparenchymal brain NCC (n = 27)	Basal subarachnoid NCC (n = 28)
Age, y, mean $\pm$ SD	31.26 $\pm$ 10.47	45.39 $\pm$ 12.42
Age, y, range	17–59	25–65
Male sex, n (%)	14 (52)	10 (36)
Duration of illness, mo, median (range)	24 (1–384)	36 (6–480)
Strong antibody response (7 bands on Western blot), n (%)	16 (59)	28 (100)
Symptoms and signs, n (%)		
Moderate to severe headache	6 (19)	25 (89)
Nausea and vomiting	13 (48)	24 (86)
Acute confusional state	0 (0)	4 (14)
Epilepsy	24 (86)	17 (61)
Other	3 (11)	7 (25)
Previous clinical history, n (%)		
Hydrocephalus	0 (0)	26 (93)
Ventriculoperitoneal shunt surgery	0 (0)	14 (50)
Antiparasitic treatment	10 (37)	11 (39)
Corticosteroid treatment	10 (37)	18 (64)
AEDs	21 (78)	16 (57)
Surgical removal of cysts	1 (4)	2 (7)

Abbreviations: AED = antiepileptic drug; NCC = neurocysticercosis.

them had an interview, clinical evaluation, and blood tests but we were unable to recontact them to arrange the scan appointment, even after repeated phone calls. In 2 cases, relatives informed the study team that the patients had traveled back to their native cities (no further information was obtained from the other 4 lost patients). Another 4 patients were excluded: 2 had isolated IV ventricle cysts without subarachnoid NCC, and 2 did not show viable cysts on MRI. The remaining 55 patients formed the study population of this protocol, including patients with IP-NCC (n = 27) and patients with BS-NCC (n = 28).

There were 24 male (43.6%) and 31 female patients. The mean ($\pm$ SD) age for all patients was 38 $\pm$ 13.4 years (range 17–65 years). Baseline char-

acteristics of patients in the IP-NCC and the BS-NCC groups are summarized in table 1. Patients with IP-NCC were younger and had a shorter duration of illness than the patients with BS-NCC ($p = 0.17$). The median number of viable cysts for all patients with IP-NCC was 3 (range 1–20). All patients with BS-NCC had strongly reactive serology (7 antibody bands on Western blot) vs only 16 in the IP-NCC group ($p < 0.001$, χ^2 test). The other 11 patients in the IP-NCC group had either responses to 5 antibody bands (n = 4), 4 bands (n = 4), or 3 bands (n = 3).

A history of moderate to severe headache was more prevalent in patients with BS-NCC than in patients with IP-NCC (odds ratio [OR] 29.17, 95% confidence interval [CI] 6.49–131.04; $p < 0.0001$), as was a history of nausea and vomiting (OR 6.46, 95% CI 1.76–23.71; $p = 0.003$). All but 2 patients with BS-NCC and none of those in the IP-NCC group presented with a history of hydrocephalus (OR 14, 95% CI 3.68 to 53.22; $p < 0.0001$). Conversely, a history of seizures was more frequent in the IP-NCC group (OR 5.18, 95% CI 1.25–21.41; $p = 0.016$). Other symptoms and signs (in general, more prevalent in the BS-NCC group) were paresthesias, decreased vision, decreased memory, paraparesis, hemiparesis, and motor ataxia. Three patients had surgical removal of cysts before entry into the study, 1 in the IP-NCC group and 2 in the BS-NCC group. Two patients underwent an excision of a giant intraparenchymal cyst by craniotomy, whereas the other patient underwent neuroendoscopic excision of cysts located in the prepontine and magna cisterns. Fourteen patients, all in the BS-NCC group, had prior placement of ventriculoperitoneal shunts because of hypertensive hydrocephalus.

There were no differences regarding previous antiparasitic treatment (albendazole in all cases) between the IP-NCC and BS-NCC group ($p = 0.86$). Of the patients, 37 (67%) were taking AEDs: 21 (78%) in the IP-NCC group and 16 (57%) in the BS-NCC group. The most commonly used AEDs were phenytoin (n = 22) and carbamazepine (n = 12). A history of corticosteroid treatment was more frequent in the BS-NCC group than in IP-NCC group (OR 29.17, 95% CI 6.49–131.04; $p = 0.043$). The presence of associated cysts in lateral ventricles was no different between both groups of patients ($p = 0.84$).

MRI evaluation. Table 2 summarizes the findings of the spinal MRI examinations by study group. Spinal involvement was found in 17 patients with BS-NCC, whereas it was only present in 1 patient with IP-NCC (OR 40.18, 95% CI 4.74–340.31; $p < 0.0001$ on χ^2 test). All patients had intradural-extramedullary

Table 2 Frequency of spinal involvement on MRI in patients with extra or intraparenchymal brain neurocysticercosis (NCC)

Involved regions	Basal subarachnoid NCC (n = 28)	Limited intraparenchymal brain NCC (n = 27)
Cervical, n (%)	2 (7)	—
Thoracic, n (%)	—	—
Lumbosacral, n (%)	2 (7)	1 (4)
Cervical-thoracic, n (%)	—	—
Thoracic-lumbosacral, n (%)	1 (4)	—
Cervical and lumbosacral, n (%)	5 (18)	—
Whole, n (%)	7 (25)	—
Total, n (%)	17 (61)	1 (4)

T2-weighted MRI images showing unilocular and multilocular cysts located throughout the entire spinal subarachnoid space (A, arrows), cervical subarachnoid space (B, arrow), thoracic subarachnoid space (C, arrows) and lumbar-sacral subarachnoid space (D, arrows).

NCC involvement, and none had intramedullary or extradural disease. Spinal cysts were more frequently located in the lumbosacral and cervical regions, followed by the thoracic region. Isolated regional involvement was found in 3 patients in the lumbosacral region and in 2 patients in the cervical spine. More than one segment was compromised in 13 patients (the entire spine in 7 patients, the cervical and lumbosacral region in 5 patients, and the thoracic and lumbosacral region in 1 patient). The patient with IP-NCC and spinal (lumbosacral) involvement

had 16 brain cysts, and 8 years of disease characterized by seizures and mild to moderate headache.

Details of the spinal NCC lesions and images from each case are presented in table e-1 on the *Neurology*[®] Web site at www.neurology.org and in figures 1 and 2. NCC-related MRI abnormalities were found in 18 patients. These included well-defined unilocular cysts in 5 (all in the cervical region), multilocular cyst formations in 17 (involving the cervical space in 9, the thoracic space in 8, and the lumbosacral space in 16), and signs

(A, B) T2-weighted MRI images showing intradural-extramedullary NCC involving the cervical region due to direct extension of multilocular cysts located in the basal subarachnoid cisterns (arrows).

consistent with adhesive arachnoiditis in 15 (all lumbo-sacral space). Distortions or flattening of the spinal cord or nerve roots were seen in all 18 cases.

BS-NCC group analysis. Because BS-NCC was strongly associated with spinal NCC, we performed a subgroup analysis comparing patients with BS-NCC and spinal involvement with those with BS-NCC but without spinal localization. There were no differences in age, sex, duration of illness, history of ventriculoperitoneal shunt, history of antiparasitic drug treatment, and associated lateral ventricular NCC.

Spinal NCC group analysis. Given that a sizable subgroup of patients ($n = 7$) had involvement of the whole spinal canal, we also compared these with patients with only regional involvement ($n = 11$). Patients with extensive involvement had a higher frequency of history of ventriculoperitoneal shunt surgery ($p = 0.010$, Fisher exact test) and tended to have a longer median duration of illness than those

with regional involvement ($p = 0.062$) (table 3). No differences were found for mean age, sex, and history of previous antiparasitic drug treatment.

Post-MRI clinical evaluation. The vast majority of patients in this series (54 of 55) had no clinical complaints related to spinal subarachnoid disease on regular initial neurologic examination. Of the 18 patients with spinal NCC, 3 were lost to follow-up (did not return after having the MRI performed). Abnormal neurologic findings related to spinal disease were found in 3 of the other 15 (20%), including the patient with related symptoms and another 2 patients.

DISCUSSION The major finding of the study is the high frequency of spinal involvement in BS-NCC. This association suggests that spinal infection arises from basilar subarachnoid cysts. Published spinal NCC series only evaluated clinically suspected cases, and thus an estimate of the overall frequency of spinal involvement was not possible.^{16,24,25} There is a striking difference between the low prevalence of symptomatic spinal disease in the literature and the high prevalence of mostly asymptomatic infection seen in this study.

Although multiple case reports and series referred to the association of brain and spinal NCC, spinal cases were always a small minority in large clinical NCC series.^{20–23} This may be a result of clinical manifestations of spinal disease appearing late in the course of the infection, as suggested by a previous study in which the presence of hydrocephalus occurred sometimes years before signs and symptoms of spinal disease.²⁵ Alternatively or in addition to the above, our findings may simply reflect the fact that no one has assessed the association in a prospective

Table 3 Comparison of patients with spinal neurocysticercosis (NCC) with whole and regional involvement

	Whole involvement (n = 7)	Regional involvement (n = 11)	p Value
Age, y, mean $\pm$ SD	41.71 $\pm$ 13.03	46.36 $\pm$ 13.85	0.488
Age, y (range)	27–62	25–65	
Male sex, n (%)	2 (29)	7 (64)	0.335
Duration of illness in months (median, range)	72 (24–336)	24 (5–480)	0.062 ^a
Ventriculoperitoneal shunt, n (%)	7 (100)	3 (27)	0.004 ^b
Antiparasitic drug treatment, n (%)	1 (14)	4 (36)	0.596

^a $p = 0.039$ if the patient with limited intraparenchymal NCC is not considered.

^b $p = 0.010$ if the patient with limited intraparenchymal NCC is not considered.

controlled study before. The likely origin of spinal cysts from the basilar subarachnoid spaces would be predicted to result in generally greater, more advanced, and earlier manifest basilar subarachnoid disease compared with spinal infections, which would require additional time to progress and become clinically evident. Patients with hydrocephalus or other brain manifestations are now frequently treated so that spinal disease that would normally manifest later might be successfully prevented.

The clinical implication of this association is unclear because our study is a cross-sectional study aimed to assess the presence and degree of association; we did not include a specific additional clinical examination to look for subclinical disease to avoid biasing patient selection. In a follow-up study, we are now focusing on the clinical relevance of spinal involvement in BS-NCC. In the past, the prognosis of extraparenchymal NCC was very poor, but it has become better in recent years, most probably because of improvements in imaging, surgical management, and antiparasitic drug therapy.^{26,27} In the absence of spine neuroimaging, a subset of patients with complicated chronic, debilitating disease with pachymeningitis and gait disorders could have unsuspected symptomatic spinal NCC in addition to encephalic involvement.

Isamat de la Riva¹⁷ and Rocca¹⁸ in the late 1950s suggested a theory to explain how the cysts reach the lower spinal cord. They proposed that strategically located, small, or developing larvae present in the subarachnoid space settle to the basal cisterns by way of gravity and then further descend into the subarachnoid spaces of the lower parts of the spine including the lumbosacral space where they find adequate room to grow and develop. Many of the pathologic descriptions of cysticerci recovered from the spinal canal have a racemose morphology, which is the form that is mostly found in basilar subarachnoid disease.¹⁹ Because this form consists of membranous cells that proliferate, it is relative easy to envision seeding of the spine from the base of the brain and subsequent growth.

The pattern of cervical infection differs somewhat from that of the lumbosacral region consistent with at least 2 mechanisms resulting in infection of the spine. Cervical involvement consists mostly of unilocular or multilocular cystic forms, primarily due to direct extension of cysts located in the basal subarachnoid cisterns. Conversely, lumbosacral involvement has a more varied picture including multilocular cystic lesions causing spinal displacement or flattening, serpentine or septated forms, and clumping or displacement of the nerve roots most likely due to adhesive arachnoiditis. A limitation of

our study is that axial and postcontrast images were only obtained from the cervical region and not from the thoracic and lumbosacral spine so that the presence and degree of inferred inflammation could not be assessed in these regions. In preliminary studies at NIH we have seen frequent lumbosacral enhancement indicative of arachnoiditis, scarring, and displacement of the sacral nerves in the cauda equina.

BS-NCC constitutes the most severe type of NCC. Because there is no consensus on how to best manage it, increased understanding is required to treat BS-NCC effectively and with minimal side effects. Spinal NCC is frequently associated; 61% of the patients who had BS-NCC had intradural-extramedullary infection. The frequent presence of spinal involvement in patients with BS-NCC makes it important to assess or rule out its contribution to NCC-associated morbidity and to define its long-term evolution and response to therapy. Because all patients in this series also had viable brain cysticercosis, careful long-term follow-up evaluations are necessarily required and testing for viable parasites using serum or CSF antigen levels might be useful.²⁸ We are aware of patients who had continuing spinal disease/infection despite resolution of basilar cysts. The design of the study indicates that the results are generally applicable to patients with BS-NCC. We believe that MRI screening of the spinal canal of individuals with BS-NCC should be performed and if spinal NCC is found, appropriate treatment and follow-up should be instituted to prevent clinical disease.

AUTHOR CONTRIBUTIONS

Dr. Callacondo: in charge of seeing patients, gathering information, performing data analysis, writing most of the manuscript. Dr. Garcia: designed and organized study, verified data and imaging, over all in charge of performing the study, contributed to the writing of the manuscript. Dr. Gonzales: neurologist in charge. D. Escalante: performed MRI imaging and interpreted them. Dr. Nash: principal investigator of project, originated concept, designed study with Dr. Garcia, funded project, contributed writing, chief editor.

DISCLOSURE

The authors report no disclosures relevant to the manuscript. **Go to Neurology.org for full disclosures.**

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