Review Article

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Neurocysticercosis and movement disorders: A literature review

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Abstract:

Neurocysticercosis (NCC) is a specific form of cysticercosis that affects the central nervous system. It is caused by the tapeworm *Taenia solium*, which is often found in pigs. NCC is considered one of the "great simulator/mimickers" of other diseases. In this context, movement disorders (MDs) can occur in a small percentage of individuals with NCC. This review aims to evaluate the clinicoepidemiological profile, pathological mechanisms, and historical features of NCC-associated MD. Relevant reports in six databases were identified and assessed by two reviewers without language restriction. A total of 71 reports containing 148 individuals who developed an MD related to NCC were identified. NCC-associated MD included parkinsonism ($n = 47$), ataxia ($n = 32$), chorea ($n = 18$), dystonia ($n = 13$), tremor ($n = 8$), myokymia ($n = 6$), myoclonus ($n = 4$), ballism ($n = 1$), tics ($n = 1$), and others ($n = 18$). The mean and median ages were 36.58 (standard deviation: 20.51) and 35 years (age range: 1–88 years), respectively. There was a slight predominance of female sex (52.17%). On follow-up, 58.90% of the individuals had a full recovery; two deaths were reported. We believe that the majority of cases reported were only diagnosed because patients had classical clinical manifestations generally investigated by neuroimaging, resulting in incidental findings suggestive of NCC, which were later supported by laboratory examinations. Therefore, the association between NCC and MD is probably underreported. Clinicians should be wary of this association, mainly in endemic areas for cysticercosis.

Keywords:

Chorea, cysticercosis, dystonia, literature review, movement disorder, myoclonus, neurocysticercosis, parkinsonism, *Taenia solium*

Introduction

Neurocysticercosis (NCC) is a specific form of cysticercosis that affects the central nervous system (CNS). It is caused by the tapeworm *Taenia solium* (*T. solium*), which is often found in pigs. The association of cysticercosis and CNS infection was first extensively described in the early 20th century by English authors. This finding can be explained by the fact that many of the patients reported had a history of travel to work in India, Egypt, and Gibraltar, places known for a high incidence of cysticercosis. When these travelers returned to the UK, the British Army medics reported their possibly imported cases. In the 1930s, a large number of cases were reported by physicians of the Royal Army Medical Corps working at the Queen Alexandra Military Hospital at Millbank by the Thames River.[1]

This cestode infection is classified by the World Health Organization (WHO) as a "neglected tropical disease," which represents a diverse group of communicable diseases prevailing in tropical areas in about 150 countries, affecting >1 billion people. Common endemic areas include Asia, Eastern Europe, and South America [Figure 1]. The WHO proposed in the early 2000s that effective control can be achieved when selected public health approaches are combined and applied locally.[2]

Pigs are commonly the intermediate host for *T. solium*. Humans are the definitive host,

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but can serve as intermediate as well. NCC occurs due to the accidental ingestion of eggs of the pork tapeworm by humans, resulting in the development of the larval form of *T. solium* (cysticercus) in the brain [Figure 2].[3,4] It is worth mentioning that NCC is only acquired from the fecal-oral route (ingestion of eggs), not via the ingestion of cysticerci in undercooked pork, which is associated with taeniasis.[3]

When the invasion of the CNS occurs, the cysticerci usually develop in five main stages [Figure 3].[5-7] The existence of the noncystic stage is not unanimously accepted, but we included it because findings of its development are observable through neuroimaging even before the vesicular phase.[6] The other four are also known as Escobar's pathological stages, which depict the natural evolution of NCC, and are identified by pathological and radiological specific features.[5] These stages are the vesicular, colloidal (colloidal–vesicular), granular (granular–nodular), and calcific (nodular) stages.[7]

Cysticercosis has an unremarkable presentation because the presence of cysts in most tissues is generally asymptomatic. Hence, the prevalence of NCC is likely underdiagnosed. The clinical manifestations can range from asymptomatic brain lesions to mimicking any neurological disorder, depending or not on the location of the cysticerci implantation.[4] It is noteworthy that, together with neurosyphilis and AIDS, NCC is considered one of the "great simulator/mimickers" of other diseases.[8] The most common presentation is seizures.[4] In endemic areas, NCC represents the etiology of an important percentage of adult-onset epilepsy. Some studies have found that NCC accounts for approximately one of every three epilepsy cases in developing countries.[9] In this context, movement disorders (MDs) usually occur in about 3% of individuals with NCC.[10] The mechanism related to NCC-associated MDs may be a direct toxic effect of the cysticerci, edema, hypoxia affecting the nerve axons, and secondary to meningeal inflammation. In this way, the present literature review aims to evaluate the clinicoepidemiological profile, pathological mechanisms, and historical features of NCC-associated MDs.

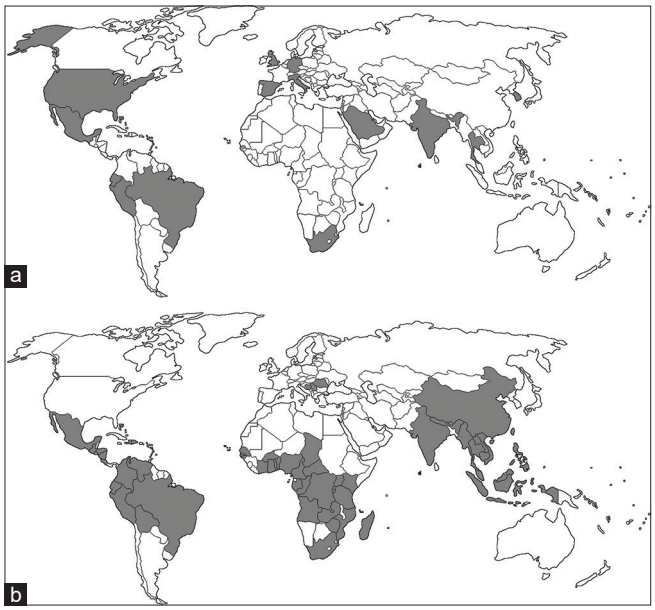


Figure 1: Comparative maps of authors' country × endemicity. (a) Country of the individuals who developed a movement disorder secondary to NCC. (b) Endemicity of *Taenia solium*, 2018. Data from the WHO, Neglected diseases (https://apps.who.int/neglected_diseases/ntddata/taenia/taenia. html, accessed on 08/19/2020)

Methods

Definitions

NCC was defined by the revised diagnostic criteria and degrees of diagnostic certainty for NCC by Del Brutto *et al*. [Table 1].[11] The majority of the studies featured neuroimaging findings. Cases without radiographic features were confirmed with histological demonstration of the parasite in biopsy material from neurosurgical interventions, identification of specific anticysticercal antibodies in cerebrospinal fluid (CSF), or cysticercosis outside the CNS associated with neurological impairment. The clinical characteristics and definitions of the MDs such as dystonia, restless leg syndrome, akathisia, dyskinesia, tremor, parkinsonism (PKN), tic, chorea, ballism, and myoclonus were obtained from the reference article by Jankovic and Tolosa.[12] In the cases where the non-English literature was beyond the authors' proficiency (English, Portuguese, Spanish, and German), and the English abstract did not provide enough data, such as articles in Korean, Thai, and Japanese, the Google Translate service was used.[13]

Search strategy

We searched six databases in an attempt to locate all reports about MDs associated with NCC that were published

Table 1: Revised diagnostic criteria for neurocysticercosis, only the neuroimaging part (Del Brutto *et al*., 2017)

Neuroimaging criteria	Description
Major	Cystic lesions without a discernible scolex
	Enhancing lesions
	Multilobulated cystic lesions in the subarachnoid space
	Typical parenchymal brain calcifications
Confirmative	Resolution of cystic lesions after cysticidal drug therapy
	Spontaneous resolution of single, small enhancing lesions
	Migration of ventricular cysts documented on sequential neuroimages
Minor	Obstructive hydrocephalus
	Abnormal enhancement of basal leptomeninges

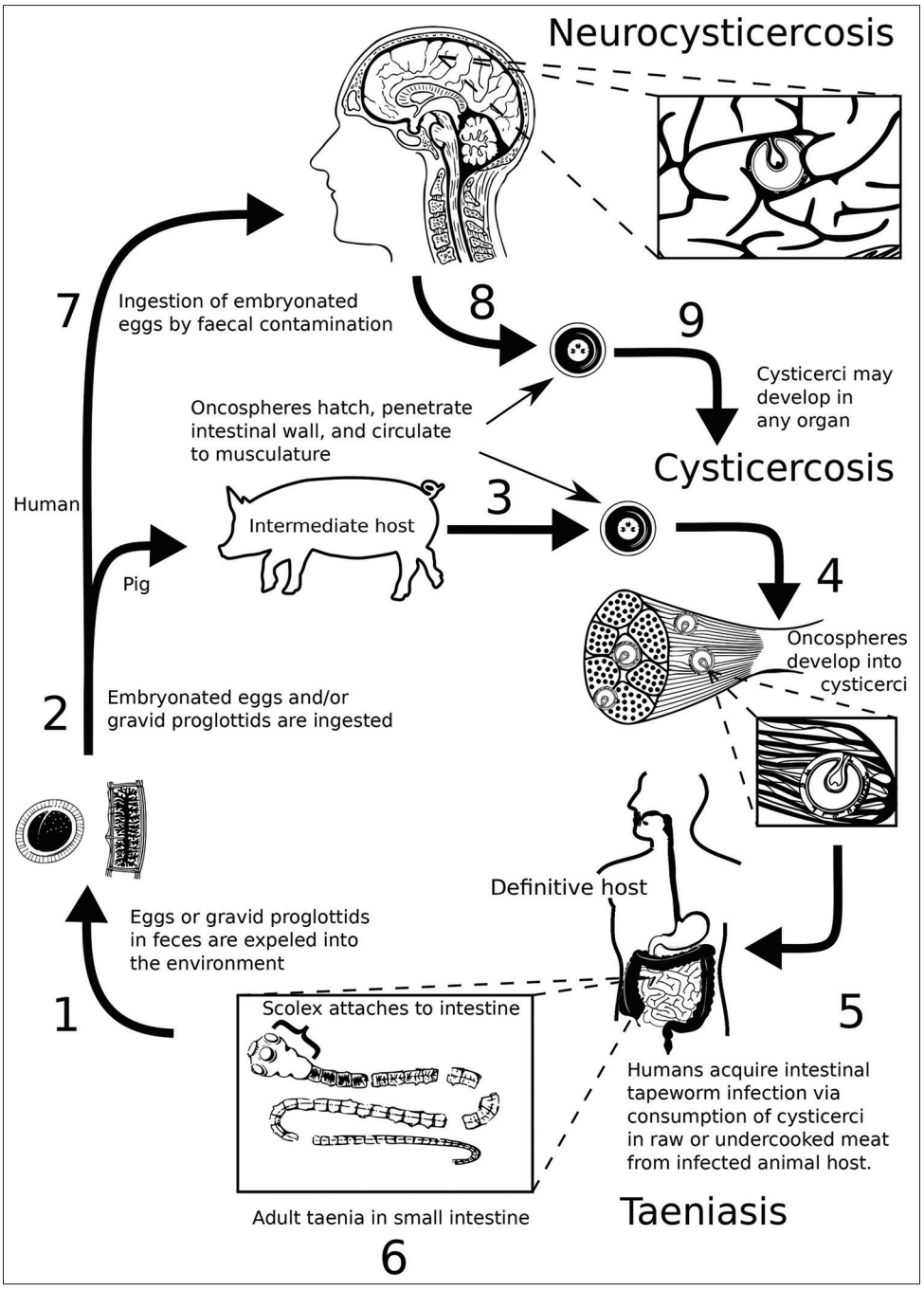


Figure 2: Cysticercosis lifecycle

until 2020 in electronic form. Excerpta Medica (Embase), Google Scholar, Latin American and Caribbean Health Sciences Literature (Lilacs), Medline, Scientific Electronic Library Online (Scielo), and ScienceDirect were searched. The search terms used were "chorea, tremor, parkinsonism, myoclonus, ataxia, dystonia, myokymia, ballism, tic, dyskinesia, stuttering, restless legs syndrome, akathisia, restlessness, hyperkinetic, hypokinetic, bradykinesia, movement disorder."[14] These terms were combined with "cysticercosis, neurocysticercosis" [Other 1 – Supplementary Material].

Inclusion and exclusion criteria

Case reports, case series, original articles, letters to the editor, bulletins, and poster presentations published up to June 2020 were included in this review with no language restriction. The two authors independently screened the titles and abstracts of all papers found in the initial search. Disagreements between the authors were discussed to establish consensus. Cases where the cause of MD was already known and the motor symptoms were not worsened or were not related to NCC were excluded. Cases that were not accessible by electronic

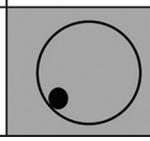
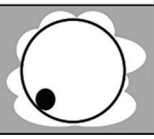
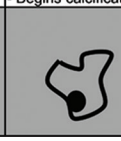
Stage	1. Non-Cystic	2.Vesicular	3.Colloidal-vesicular	4.Granular-nodular	5.Calcific
Description	Tissue invasion by the cysticercus	Viable parasite with intact membrane.	Parasite begins to degenerate. Cyst fluid becomes turbid. As the membrane becomes leaky edema surrounds the cyst. Host develops immune response. This is the most symptomatic stage.	Parasite degenerates. Host immune response decreases. Edema decreases as the cyst retract further.	Dead parasite. End-stage quiescent calcified cyst remnant; no edema.
Clinical manifestations	Usually asymptomatic	Seizure, intracranial hypertension, hydrocephalus, meningitis	Seizure		
Pathological mechanism	Inflammatory reaction	Compression and inflammatory reaction	Inflammatory reaction	Perilesional gliosis	Perilesional gliosis
Immunological response	Neutrophilic	Th 1 to Th 2	Th 1		Mixed Th 1 to Th 2
Radiological appearance (MRI features)	Local focus of edema. There might be nodular contrast enhancement. Usually no imaging studies are performed. Usually not correlated with imaging.	- "cyst with dot sign" represents the parasitic cyst with, usually eccentric, scolex. - Cyst (CSF-like signal): T1 hypointense, T2 hyperintense. - Scolex: isointense to parenchyma on T1 and T2, hypointense onT2 sequences, hyperintense on FLAIR - Usually no surrounding vasogenic edema	- Vasogenic edema surrounding the cyst. - Ring-like contrast enhancement - Formation of fluid-fluid level - Cyst: formation of capsule, hypointense on T2 sequences. Increased signal in the cyst fluid - scolex is seen early in the colloidal phase but gradually shrinks down and is harder to identify	- Residual cyst is smaller in size, thickening of the capsule. Isointense to the parenchyma on T1-/iso-hypointense on T2-weighted sequences - Calcified scolex (target appearance) - Minimal vasogenic edema might persist - Nodular or micronodular contrast-enhancement	- Calcified nodule without contrast-enhancement - Hypointense nodule on T2 sequences
Resume	Edema?	- Cyst + scolex. - Non-enhancement	- Ring enhancement. - Edema	- Decreased enhancement and edema - Begins calcification	- Obvious calcification
Imaging	-				

Figure 3: Stages of *Taenia solium* cysts in neurocysticercosis. Noncystic, vesicular, colloidal (colloidal-vesicular), granular (granular-nodular), and calcific (nodular)

means, including after a formal request E-mailed to the authors, were also excluded.

Data extraction

A total of 1,986 reports were identified from the search, of which 1,915 were excluded as they did not meet the inclusion and exclusion criteria [Figure 4]. When provided, we extracted MD type, authors, department, year of publication, country of occurrence, number of patients affected, age, sex, CSF analysis, neuroimaging features, patient's status at follow-up, and important findings of clinical history and management. The data were extracted by two independent authors, double-checked to ensure matching, and organized by whether or not the MD was caused by NCC.

Statistical analysis

Categorical variables were represented as proportions; continuous variables were represented as mean, standard deviations (SDs), median, and range.

General data

A total of 71 articles containing 148 individuals presenting MDs related to NCC were reported [Tables 2 and 3].[10,15-84] Between 1903 and 2020, there were 81 patients from South America, 36 from Asia, 12 from Europe, 12 from North America, and 7 from Africa. The MDs encountered were PKN, ataxia, chorea, dystonia, tremor, myokymia, myoclonus, ballism, tics, and other not clearly defined. The mean and median ages were 36.58 (SD: 20.51) and 35 years (age range: 1–88 years), respectively. There was a slight female predominance (52.17%). On follow-up, more than half of the individuals (58.90%) had a full recovery. In addition, two deaths were reported due to the severity of other diseases co-occurring with NCC.[22,73]

Interestingly, individuals with PKN had a higher frequency of complications. Moreover, those patients with a poor outcome had other associated clinical manifestations such as seizure, intracranial hypertension (ICH), cognitive impairment, and a focal neurologic deficit.

Alarcón *et al*. in 2017 studied 23 individuals who developed MDs secondary to NCC.[10] They presented interesting conclusions, which changed many aspects of the current understanding of this infectious disease and abnormal movements. First, PKN was the most common MD in NCC, and was not related to the specific localization of the lesions. Second, individuals with chorea, dystonia, and tremor had a better prognosis than those who developed PKN. Third, chorea and dystonia were associated with specific lesions in the basal ganglia. It is worth mentioning that these characteristics were also

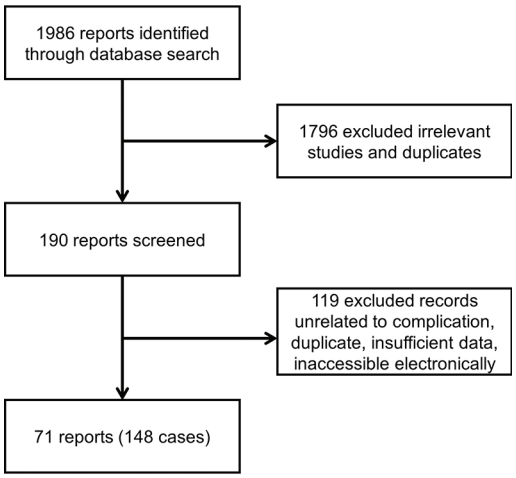


Figure 4: Flowchart of the search process

found in our study and we will further discuss them in the following sections.

CHOREA – The first report

Pereira was probably the first to report a MD associated with tapeworm infection, published in 1903 on the "Clinical Notes" on "The Lancet."[16] He described the case of a child who developed generalized chorea with a gastrointestinal infection, possibly taeniasis, without any heart involvement or a history of pharyngitis. After his publication, two other authors, Hodge in 1903 and Galbraith in 1904, reported similar cases.[15,17] However, both patients had heart murmurs, which, in association with the chorea, could be the clinical manifestation of rheumatic fever caused by an autoimmune reaction to Group A β-hemolytic streptococci. Moreover, many drugs with unknown efficacy were attempted. Consequently, it is possible that these authors induced MDs in their patients, due to the use of substances that today are known to be extremely neurotoxic. Interestingly, Galbraith also wrote in 1903 that there was a distinct increase of chorea, which he correlated with the increased incidence of poliomyelitis at that time.[17]

Chorea, dystonia, and myoclonus affected a younger population when compared to the general data. Furthermore, chorea had the second-worst prognosis on follow-up because only about half of the patients had a full recovery. One death was reported by Bickerstaff in 1952.[22] He reported a patient with racemose NCC in which surgical excision of the lesion was tried, but she had extensive lesions in the arachnoid. The patient died of respiratory infections during hospitalization. Racemose NCC is characterized by the infection of the basal subarachnoid region. It is a relatively rare form of NCC with an appearance resembling "a collection of transient membranes forming a cluster like a bunch of grapes."[85]

The cause of chorea in NCC could be explained by the mass effect, inflammation, hydrocephalus, or ischemia associated with the cysticerci. In this context, the majority of patients with neuroimaging showed at least one lesion localized in the basal ganglia region.[10] Another explanation for this involuntary movement could be an exacerbated immune response because many patients had alleviation of the symptoms when corticosteroids were started.[72,82] In addition, others had ICH, and a ventriculoperitoneal shunt (VPS) was performed to alleviate the symptoms.[77] Vasculitis of the right middle cerebral artery was observed in one individual.[50] The presence of stenosis may lead to a change in the blood flow, and, due to this variable flux, abnormal movements can occur. This concept is supported by Echebarria, who studied patients with suspected NCC and established a correlation among the cerebral blood flow velocity and CSF pressure with the diagnostic criteria for NCC-associated MDs.[62]

One interesting fact is that in east Asian countries, the coinfection of NCC and Japanese encephalitis is being reported.[78] When these infections occur concomitantly, it is difficult to give a clear diagnosis of the main cause of the abnormal movements. We believe that specific features such as cystic lesions found in the basal ganglia associated with chorea are highly suggestive of chorea caused by NCC, as shown by this review and by Alarcón *et al*.[10]

PARKINSONISM – Diffuse brain inflammatory reactions?

Meyer (1906) and Felici (1938) were the first to associate PKN and cysticercosis; both studies contributed with the demonstration of postmortem microscopical analysis of the parasite.[22] Assis and Tenuto reported positive serum and CSF for cysticercosis in a patient with the association of PKN and ICH.[21]

The co-occurrence of PKN and ICH should be highlighted because almost half of the individuals reported in the literature [Table 2] with NCC and PKN had ICH. These clinical manifestations can be explained by the cerebral cysticercus causing edema or direct obstruction of the flow of the CSF, raising the intracranial pressure, and leading to the resting tremor and bradykinesia.[86] Cysts causing hydrocephalus have already been described in the fourth ventricle, perimesencephalic, meningeal, and brainstem areas. Alarcón *et al*. (2017) stated that PKN is not related to the specific localization of the lesions. This can be explained by the previously described hypothesis, even though the majority of the individuals reported by Alarcón *et al*. (2017) did not present any signs of ICH.

Besides ICH, other explanations based on the cysts' location appear to be insufficient, as it has been observed

Table 2: Literature review of neurocysticercosis-associated movement disorder

Reference	Country/Year	*n* cases	Age/sex	Neuroimaging	Follow-up	Important CH and CM
			Chorea			
Pereira	The UK/1903	1	6/female	NA	CR	CH: Chorea. There was no suspicion of rheumatic fever. CM: Filix was used and in 1 month, the patient had a CR
Hodge	The UK/1903	1	9/female	NA	NA	CH: Chorea. CM: Filix was used
Galbraith	South Africa/1904	1	10/male	NA	CR	CH: Chorea induced by a tapeworm. Probably by Taenia solium. CM: the prescription of some herbals reduced the symptoms. Presence of hurt murmur. Maybe it is a case of Group B streptococcal infection. The author believed that the cause was related to the gastrointestinal infection
Brotto	Brazil/1947	1	37/female	Cranial X-ray	No	CH: Chorea. The individual presented with epilepsy. CM: The symptoms alleviated with sulfacetamide (Albucid)
Bickerstaff *et al*.	The UK/1952	1	50/female	Ventriculography	Death	CH: Chorea CM: Cyst excision
Bhigjee *et al*.	South Africa/1987	1	15/female	Multiple cysticerci, with some in the head of the right caudate nucleus and striatum	No	CH: Hemichorea+headache. CM: Haloperidol. The symptoms were alleviated
Joubert and Jenni	South Africa/1990	1	NA	NA	NA	CH: Chorea. From 88 patients with NCC, 1 developed chorea (1.1%). CM: PZQ
Bouldin and Pinter	The USA/2006	1	11/male	T2-weighted hyperintensity and Gd-enhancement in the M1 segment of the right middle cerebral artery. Arterial stenosis	CR	CH: Hemichorea. CM: Prednisone and aspirin. The symptoms recovered. The antiparasitic medication was not started to avoid increasing neurological damages
Cosentino *et al*.	Peru/2006	1	22/female	Visible hyperdense dot corresponding to the parasite scolex, cysts in multiple locations	CR	CH: Hemichorea, which was episodic, disappearing during sleep, and usually increasing with stress. CM: Dexamethasone and ALB. The symptoms recovered
Verma *et al*.	India/2006	1	12/female	A single ring-enhancing lesion with perifocal edema in the left thalamus	NA	CH: Hemichorea. CM: ALB, steroids, and haloperidol. The symptoms were alleviated
Balaji and Meikandan	India/2011	1	NA	NA	NA	Clinical and radiological findings of 58 South Indian children diagnosed with NCC
Dewan *et al*.	India/2011	1	10/female	Ring-enhancing lesion involving the right paramedian midbrain with mild perilesional edema	NA	CH: Generalized chorea. CM: Steroids, haloperidol, and ALB. She developed ICH, and mannitol was started. The symptoms improved
Venkatarathnamma *et al*.	India/2013	1	25/male	Multiple hypodense lesions with central hyperdensity in the parenchyma	NA	CH: Possible orofacial chorea. Phenytoin may be a confounding variable. CM: ALB, steroids, and sodium valproate. The symptoms improved
Costa *et al*	Brazil/2016	1	12/male	Multiple cysts (meninges, suprasellar, and prepontine areas) of cysticerci (racemose form); hydrocephalus	CR	CM: VPS was performed. Dexamethasone, ALB, and phenobarbital. The symptoms recovered

Contd...

Table 2: Contd...

Reference	Country/Year	*n* cases	Age/sex	Neuroimaging	Follow-up	Important CH and CM
Yoganathan *et al*.	India/2016	1	11/male	Hyperintensity involving bilateral basal ganglia, thalami, substantia nigra, and hippocampi. There was a cysticercus in the right posterior temporal lobe with ring enhancement and perilesional edema	NA	CH: Orofacial DKN; possible chorea. Coinfection of Japanese encephalitis and NCC
Alarcón *et al*.	Ecuador/2017	1	21/female	Left thalamic cyst; right calcifications on putamen	CR	CH: Hemichorea. CM: ALB. The symptoms recovered
Gupta *et al*	India/2019	1	60/female	A peripheral ring-enhancing lesion with an eccentric nodule in the left frontal parasagittal region	No	CH: Hemichorea. CM: Haloperidol. The symptoms improved
Kumar *et al*	India/2020	1	77/male	Multiple cysts in the cortex, subcortex, and basal ganglia, with surrounding edema and calcified scars	No	CH: Hemichorea. CM: ALB and prednisolone. The symptoms improved
				Tremor		
Dixon and Smithers	The UK/1935	1	24/NA	NA	NA	CH: Localized tremor. It was a possible focal motor onset seizure with impaired consciousness
Scott *et al*.	India/2005	1	1/male	Ring-enhancing lesions in cortical, subcortical regions and also in the basal ganglia	CR	CH: Tremor involving the tongue and left upper and lower limbs. CM: ALB and corticosteroids. The symptoms recovered
Alarcón *et al*.	Ecuador/2017	5	67/male	Right frontal and right lenticular cysts and left caudate infarcts	CR	CH: Tremor involving the left upper limb. CM: ALB. The symptoms recovered.
			63/female	Left parietal cyst	CR	CH: Tremor involving the right upper and lower limbs. CM: ALB. The symptoms recovered
			50/female	Bilateral frontal, parietal, and temporal cysts, hydrocephalus	CR	CH: Bilateral tremor. CM: ALB. The symptoms recovered
			35/male	Cyst in right sylvian cistern and striatal cysts	CR	CH: Tremor involving the right upper limb. CM: ALB. The symptoms recovered
			43/female	Left temporal cyst and right lenticular and thalamic calcifications	CR	CH: Tremor involving the right upper limb. CM: ALB. The symptoms recovered
Campos *et al*	Ecuador/2018	1	21/female	Intraventricular cyst with scolex in the right lateral ventricle, and another cyst in the fourth ventricle	NA	CH: Cerebellar outflow tremor+ophthalmoparesis. Video recording. CM: steroids and ALB. The symptoms improved
				Parkinsonism		
Meyer	Germany/1906	NA	NA	NA	NA	Racemose cysticercosis presenting as PKN quoted by Bickerstaff in 1952
Felici	Italy/1938	NA	NA	NA	NA	Racemose cysticercosis presenting as PKN quoted by Bickerstaff in 1952
Brotto	Brazil/1947	1	9/male	Cranial X-ray	NA	CH: PKN+ICH
Assis and Tenuto	Brazil/1948	1	38/female	Ventriculography	CR	CH: PKN+ICH. Serum and CSF were positive to cysticercosis. CM: Surgical management of a giant cyst
Ronge *et al*	Germany/1978	1	NA	NA	NA	CH: PKN+temporal lobe epilepsy+psychiatric symptoms. There were parasites in the Sylvian fossa and bilateral necrosis of the pallidum

Contd...

Table 2: Contd...

Reference	Country/Year	*n* cases	Age/sex	Neuroimaging	Follow-up	Important CH and CM
Takayanagui and Jardim	Brazil/1983	2	NA	NA	NA	CH: From 238 individuals with NCC, 2 developed PKN (0.4%)
Cavalcanti	Brazil/1984	1	57/male	Cranial X-ray normal. Cranial CT scan with basal ganglia calcifications	NA	CH: Isolated PKN. CM: Levodopa and PZQ
Takayanagui and Jardim	Brazil/1990	1	NA	NA	NA	CH: PKN+ICH. The patient presented with seizures. From 151 individuals with NCC, 1 developed PKN (0.66%)
Tansanee	Thailand/1992	1	36/male	NA	NA	CH: PKN in a racemose cysticercosis
Keane	The USA/1995	4	32/female	NA	CR	CH: PKN+ICH. CM: VPS
Keane conclude that the occurrence of PKN in a previously shunted patient suggests obstruction and requires prompt evaluation of the VPS			22/female	NA	CR	CH: PKN+ICH. CM: VPS
			28/female	NA	No	CH: PKN+ICH. CM: VPS
			32/male	NA	No	CH: PKN+ICH. CM: VPS. Levodopa responsive
Verma *et al*.	The USA/1995	1	31/female	Multiple calcified and cystic lesions	No	CH: PKN+midbrain encephalitis. Video recording. CM: PZQ, dexamethasone, and phenytoin
Serrano-Dueñas and Placencia	Ecuador/1999	1	66/male	Obstructive hydrocephalus related to a fourth-ventricle cysticercus	No	CH: PKN+ICH. No levodopa response. CM: ALB and cyst removal
Mathew and Hassan	India/2001	1	NA	NA	CR	CH: reversible PKN
Sá *et al*.	Brazil/2004	2	32/female	Hyperintensity over the cerebral aqueduct and the 4th ventricle in keeping with ependymitis	No	CH: PKN. History of VPS. CM: levodopa was started, and the symptoms were alleviated. ALB was started and allowed later reduction of levodopa dosage
			30/male	Multiple calcifications and hydrocephalus. Morphologic abnormalities of the quadrigeminal and ambient cisterns	CR	CH: PKN+ICH. CM: A VPS was performed. Levodopa and ALB. The patient symptoms recovered
Patel *et al*.	India/2006	1	60/male	Multiple cysticerci in various stages (vesicular and granular). Few of them were cystic with a scolex	No	CH: PKN+DTN. CM: levodopa and trihexyphenidyl. The symptoms alleviated
Suwatcharangkoon *et al*.	Thailand/2006	1	NA/female	Multiple cystic lesions and dilatation of the lateral ventricles	NA	CH: PKN+ICH. CM: ALB was started, and a VPS was performed. The symptoms alleviated
Garcia Ruiz *et al*.	Spain/2008	1	29/female	Hydrocephalus and hyperintensity over the cerebral aqueduct in keeping with ependymitis	CR	CH: PKN+ICH+blepharospasm. CM: ALB and levodopa. The PKN symptoms recovered. Botulinum toxin was started, and DTN recovered
Cabo López *et al*.	Spain/2008	1	29/female	NA	NA	CH: PKN+ICH. CM: VPS was performed. Cysticidal drugs and levodopa
Prashantha *et al*.	India/2008	1	38/male	Cystic lesion with scolex and T1 ring-enhancing granulomas in the fourth ventricle	CR	CH: PKN+ICH. CM: VPS was performed. Levodopa was started. Anti-parasitic drugs were not started. The symptoms recovered
Munhoz *et al*.	Brazil/2009	2	43.1/2 male	NA	NA	Case series of eight individuals with possible infectious PKN

Contd...

Table 2: Contd...

Reference	Country/Year	*n* cases	Age/sex	Neuroimaging	Follow-up	Important CH and CM
Sharma *et al*.	India/2011	1	64/male	Multiple vesicular lesions in the cortex; dorsal midbrain and tegmentum of the midbrain	No	CH: progressive supranuclear palsy like+progressive cognitive decline. CM: dexamethasone and ALB. The symptoms did not improve
Lima *et al*.	Brazil/2012	1	38/female	Hydrocephalus because of an intraventricular cyst; edema in the midbrain periaqueductal region; ependymitis	CR	CH: PKN+ICH CM: VPS was performed. ALB was started and worsened the PKN symptoms. Methylprednisolone pulse therapy and levodopa. Surgical removal of the cyst. The symptoms recovered
Teive	Brazil/2012	1	38/female	Presence of supratentorial hydrocephalus with evidence of intraventricular cysts, as well as edema in the midbrain periaqueductal region	CR	CH: PKN+ICH. CM: Methylprednisolone and levodopa. The symptoms recovered
Verma *et al*	India/2013	2	50/female	Multiple cystic lesions with a mural nodule in bilateral basal ganglia	Death	CH: PKN+seizures. CM: ALB and steroids were started, which worsened the symptoms. The patient died due to ICH
			43/male	Multiple cysticerci lesions scattered within bilateral pallidum and putamen	CR	CH: PKN+seizures. CM: cysticidal drug and steroids. The symptoms resolved
Alarcón *et al*	Ecuador/2017	15	47/female	Temporal and right sylvian subarachnoid cysts, hydrocephalus	No	CH: PKN
			56/female	Right and left frontal cysts, bilateral sylvian and perimesencephalic cyst, hydrocephalus	No	CH: PKN+ICH
			60/female	Bilateral Sylvian, basal subarachnoid and perimesencephalic cysts, hydrocephalus	No	CH: PKN
			44/female	Left frontal cyst and calcification	CR	CH: PKN+seizure
			65/male	Bilateral frontal, parietal and occipital cysts, basal arachnoiditis, perimesencephalic and pontine subarachnoid cysts, hydrocephalus.	No	CH: PKN+ataxia+seizure
			48/female	Left frontoparietal cysts, hydrocephalus	CR	CH: PKN+seizure
			42/male	Perimesencephalic cysts, aqueductal ependymitis, and severe hydrocephalus	CR	CH: PKN+ataxia+seizure+ICH
			62/male	Bilateral subarachnoid sylvan cysts, perimesencephalic cysts, and cysts of the IV ventricle, hydrocephalus	CR	CH: PKN+ataxia+apraxia+ICH
			73/female	Tetraventricular hydrocephalus, hyperintensity of the aqueduct, subarachnoid cysts perimesencephalic	No	CH: PKN+ataxia
			47/male	Cyst IV ventricle, hydrocephalus, and ependymitis	CR	CH: PKN+ataxia+ICH
			70/female	Subarachnoid and parenchymal cysts, front right, in the ambient, pretectal and prepontine cistern, severe hydrocephalus	CR	CH: PKN+ICH

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Reference	Country/Year	*n* cases	Age/sex	Neuroimaging	Follow-up	Important CH and CM
			76/male	Hydrocephalus, frontal and parietal left cysts, arachnoiditis. Midbrain and striatal infarcts	No	CH: PKN+ICH+possible MKM. Presence of hemifacial spasm
			46/female	Hydrocephalus, suprasellar, prepontine and ambient cysts, anterior interhemispheric fissure and right valley Sylvian, frontal and IV ventricle cyst	No	CH: PKN+ataxia+ICH
			76/male	Severe hydrocephalus; left frontal and parietal cysts	No	CH: PKN+apraxia+ICH
			56/female	Triventricular severe hydrocephalus, Sylvian and IV ventricle cysts frontal and parietal cysts rights	No	CH: PKN+ICH
Oliveira *et al*.	Brazil/2020	1	59/female	Cystic lesions in the subcortical region; scolex associated with perilesional hyperintensity (vesicular stage); hydrocephalus; transependymal resorption	NA	CH: PKN+ICH. CM: VPS was performed. The symptoms improved. ALB and PZQ
				Myoclonus		
Wie	South Korea/1986	1	54/male	Multiple intraparenchymal punctate calcifications including the left dentate nucleus	NA	CH: Palatal (subcortical) MCL. CM: PZQ and trihexyphenidyl
Puri *et al*.	India/1991	1	11/female	Multiple, round, low densities with peripheral enhancement and signs of meningeal inflammation	CR	CH: Multifocal MCL. Reflex response positive. EEG positive. ELISA in serum and CSF was positive for NCC. CM: valproate and PZQ. The symptoms recovered
Keane	The USA/1993	1	29/male	Several large prepontine cysticerci cysts	No	CH: oculopalatal MCL (subcortical) and short-cycle periodic alternating nystagmus. CM: Exploration of his posterior fossa with the removal of cysts
Gokhale *et al*.	India/2015	1	8/male	A lesion with mixed-signal intensity in left high frontal gyrus with perilesional edema	CR	CH: multifocal MCL (subcortical). EEG was normal. CM: ALB and prednisolone. The symptoms recovered
				Ataxia		
Ronge *et al*	Germany/1978	1	NA	NA	NA	Vestibular ataxia
Takayanagui and Jardim	Brazil/1983	21	NA	NA	NA	Ataxia. From 238 individuals with NCC, 21 presented with ataxia (4.2%)
Barinagarementeria *et al*.	Mexico/1988	1	38/male	A hypodense suprasellar mass, an ill-defined small area of low density was seen just above the right cerebral peduncle	CR	CH: Ataxic hemiparesis. ELISA and complement fixation tests were positive for NCC. CM: Prednisone. The symptoms recovered
Joubert and Jenni	South Africa/1990	4	NA	NA	NA	Ataxia. From 88 patients, 4 individuals had ataxia (5%)
Singh *et al*.	India/1996	1	12/male	Multiple ring lesions in the posterior fossa with a central nidus characteristic of NCC	CR	CH: Truncal and limb ataxia. CM: ALB and prednisolone
Jha *et al*.	India/2006	1	10/male	Multiple cysts in the cerebellar hemisphere	No	CH: Truncal and limb ataxia. CM: Valproate, glycerin, and acetazolamide. The symptoms alleviated

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Table 2: Contd...

Reference	Country/Year	*n* cases	Age/sex	Neuroimaging	Follow-up	Important CH and CM
Teive *et al*.	Brazil/2008	1	33/male	The intense inflammatory reaction at the cysts located in the basal cisterns and around the brainstem	NA	CH: Truncal ataxia in an individual with racemose form of NCC
Balaji and Meikandan	India/2011	1	NA	NA	NA	Clinical and radiological findings of 58 South Indian children diagnosed with NCC
Sharma *et al*	India/2015	1	17/female	Multiple ring-enhancing lesions involving bilateral cerebral hemisphere, cerebellum and brain stem with ring-enhancing lesion	NA	CH: Ataxia+lateral rectus muscle of left eye impairment
				Dystonia		
Jiménez-Jiménez *et al*.	Spain/1992	1	48/female	Multiple cysts including some in the left thalamus, and a lacunar infarction in the right internal capsule area	NA	CH: Bilateral blepharospasm
Sawhney *et al*.	India/1998	1	21/male	Hypodense cysts in the internal globus pallidus, a ring-enhancing lesion in left corona radiata, bifrontal diffuse white matter edema	CR	CH: Unilateral DTN of upper and lower limbs. Previous history of complex partial seizures. CM: Steroids, anticonvulsants
Serrano-Dueñas and Placencia	Ecuador/1999	2	60/male	Hydrocephalus; hypo/hyperdense cystic lesions in the cerebral cortex and subcortical white matter without involvement the basal ganglia	CR	CH: Cervical DTN (retrocollis) + ICH. CM: VPS was performed and ALB was started
			88/male	Normal-pressure hydrocephalus	No	CH: Cervical DTN (retrocollis) + ICH. CM: Biperiden was withdrawal due to side effects. Clonazepam was started, but the symptoms did not ameliorate
Frei and Truong	USA/2002	1	NA	Several lesions including in the basal ganglia	NA	CH: Scalp DTN. The diagnosis was done when an EEG was performed and muscle artifacts were observed
Jha *et al*	India/2006	1	6/male	Multiple lesions in basal ganglia	No	CH: Unilateral DTN of the lower limb. CM: Levodopa and trihexyphenidyl. The symptoms improved
Patel *et al*	India/2006	1	19/female	Single, ring-enhancing granuloma in the right thalamic region (colloidal stage)	No	CH: Unilateral DTN of hand. CM: ALB and corticosteroids. The symptoms did not improve
Hamed and Metaal	Saudi Arabia/2006	3	21/female	Multiple lesions in the frontal, temporal, and temporoparietal regions	NA	CH: Possible DTN. CM: PZQ, dexamethasone, and carbamazepine. The symptoms improved
			17/female	Multiloculated cystic lesion deep in the white matter of the right parietal lobe. There was a small satellite cyst seen at the cortical aspect of the lesion (colloidal stage)	CR	CH: Unilateral DTN of upper and lower limbs. CM: PZQ, steroids, tiapridal (benzamide), and carbimazole. The symptoms resolved
			19/female	Single left parietal deep white matter multiloculated cystic lesion (colloidal stage)	CR	CH: Unilateral DTN of the upper limb. CM: PZQ and tiapridal (benzamide). The symptoms resolved
Yoganathan *et al*.	India/2016	1	13/male	Asymmetric areas of hyperintensity involving the frontal, temporal, parietal lobes, caudate, and thalami;	NA	CH: Meige like syndrome+PKN. Coinfection of Japanese encephalitis and NCC

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Table 2: Contd...

Reference	Country/Year	*n* cases	Age/sex	Neuroimaging	Follow-up	Important CH and CM
				cysticerci granulomas in the right cingulate gyrus and right inferior frontal gyrus; ring enhancement		
Alarcón *et al*.	Ecuador/2017	2	23/female	Cyst in the left putamen	CR	CH: Unilateral DTN of the lower limb. CM: ALB. The symptoms recovered
			37/female	Bilateral putamen cysts	CR	CH: cervical DTN. CM: ALB. The symptoms recovered.
Myokymia						
Keane	The USA/1993	1	29/male	Several cerebral cysticerci cysts in addition to obstructive hydrocephalus	2x CR	CH: Facial MKM. EMG positive CM: Symptoms resolved following placement of a VPS. After 3 years, a problem with the shunt leads to the reappearance of the symptoms, which recovered after a new VPS
Beydoun	The USA/1994	1	34/male	Multiple cystic lesions with enhancing rims within the aqueduct and fourth ventricle	No	CH: Facial MKM. EMG positive. CM: Decadron, PZQ, and Dilantin. The symptoms alleviated
Gutierrez *et al*.	Mexico/1998	1	69/female	Subarachnoid cysticercus rostral to the pons	CR	CH: Possible facial MKM+ICH; described as hemifacial spasms. EMG was not performed. CM: VPS was done. Prednisone. The spasms disappeared 3 months after VPS
Bhatia *et al*.	India/2008	1	45/male	Ring enhancing lesions with surrounding edema on the right side of pons. Active degenerating (colloidal-vesicular) stage	CR	CH: Facial MKM. Video recording. CM: Clonazepam, prednisolone, and carbamazepine. The symptoms recovered
Razdan *et al*.	India/2009	1	20/male	Isointense ring lesion with an eccentric scolex with perilesional edema in the right posterior pons	CR	CH: Possible facial MKM; described as hemifacial spasms. CM: ALB and prednisolone. The symptoms recovered
Yang *et al*.	Perú/2020	1	38/male	Multiple cysts in the basal cisterns involving both cerebellopontine angle	CR	CH: Possible facial MKM, described as hemifacial spasm. Video recording. CM: VPS was performed. Gabapentin, dexamethasone, and ALB. Surgical decompression was performed, which recovered the symptoms
Ballism						
Karnik *et al*.	India/2011	1	11/female	Scolex in the left thalamus	No	CH: Hemiballismus. CM: ALB and prednisolone. The symptoms improved
Tics						
Anjana *et al*.	India/2020	1	29/female	Multiple focal discrete subcentimeter parenchymal ring-enhancing lesions in bilateral cerebral hemispheres with significant perilesional edema in bilateral frontal, right ganglion capsular, and left parietal regions	No	CH: Tics+psychosis. CM: Mannitol, steroids, ALB, and antipsychotics. The symptoms improved
Not clearly defined MDs						
Wallenburg	The UK/1928	NA	NA	Broughton-Alcock and others (1928) quote Wallenburg (no reference) as saying that automatic movements have been described		

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Table 2: Contd...

Reference	Country/Year	*n* cases	Age/sex	Neuroimaging	Follow-up	Important CH and CM
Cosentino *et al*.	Peru/2002	NA	NA	To assess in 120 individuals with NCC, the frequency of basal ganglia location of lesions, and its clinical manifestations. Cysts or enhancing lesions in basal ganglia were found in 32 out of 120 cases (26.7%)		
Alarcon	Ecuador/2005	15	NA	Report of 15 individuals with NCC who developed an MD		
Echebarria	Spain/2009	NA	NA	Assessment of CBFV and cerebrospinal fluid pressure in individuals with NCC who developed MDs. There was a correlation between CBFV and diagnostic criteria derived from 2/3 criteria established in NCC diagnosis		
Sarangi *et al*	India/2013	1	NA	Report of a 10-year-old female who developed ataxia/DTN/apraxia of the hand. The case is not clear about the description of the neurological examination. CT: Isodense right posterior parietal lesion		
Singh *et al*	India/2015	1	NA	Report of a 14-year-old male who developed involuntary jerk movements. These movements could be MCL, tremor, and DTN. The neurological examination is not clearly described		

ALB: Albendazole, CH: Clinical history, CM: Clinical management, CR: Complete recovery, DKN: Dyskinesia, DTN: Dystonia, ICH: Intracranial hypertension, MCL: Myoclonus, MKM: Myokymia, NA: Not applicable/not available, NCC: Neurocysticercosis, PKN: Parkinsonism, PZQ: Praziquantel, VPS: Ventriculoperitoneal shunt, EEG: Electroencephalogram, ELISA: Enzyme-linked immunosorbent assay, IV: Intravenous, CBFV: Cerebral blood flow velocity, CSF: Cerebrospinal fluid, CT: Computed tomography, MD: Movement disorder

Table 3: Resume of neurocysticercosis-associated movement disorders

MD	PKN	Ataxia	Chorea	DTN	Tremor	MKM	MCL	Ballism	Tics	Others	General data
Cases (%)	47 (31.7)	32 (21.6)	18 (12.1)	13 (8.7)	8 (5.4)	6 (4.05)	4 (2.70)	1 (0.67)	1 (0.67)	18 (12.16)	148
Continent (%)											
Asian	8 (17.0)	4 (12.5)	7 (38.8)	7 (53.8)	1 (12.5)	2 (33.3)	3 (75)	1 (100)	1 (100)	2	36 (24.32)
European	5 (10.6)	1 (3.1)	3 (16.6)	1 (7.6)	1 (12.5)	0	0	0	0	1	12 (8.10)
North America	5 (10.6)	1 (3.1)	1 (5.5)	1 (7.6)	0	3 (50)	1 (25)	0	0	0	12 (8.10)
South America	29 (61.7)	22 (68.7)	4 (22.2)	4 (30.7)	6 (75)	1 (16.6)	0	0	0	15	81 (54.72)
Africa	0	4 (12.5)	3 (16.6)	0	0	0	0	0	0	0	7 (4.72)
Sex (%)											
Female	22 (46.8)	1 (3.12)	10 (55.5)	7 (53.8)	4 (50)	1 (16.6)	1 (25)	1 (100)	1 (100)	NA	48
Male	18 (38.2)	4 (12.5)	6 (33.3)	5 (38.4)	3 (37.5)	5 (83.3)	3 (75)	0	0		44
Unknown	7 (14.8)	27 (84.3)	2 (11.1)	1 (7.69)	1 (12.5)	0	0	0	0		56
Age (years)											
Range	9-76	10-38	6-77	6-88	1-67	20-69	8-54	11	29		1-88 (Md: 35)
Mean	46.54	22.00	24.25	31.00	38.00	39.16	25.50	11	29		36.58 (SD: 20.51)
Follow-up - % CR (number of reports)	48.48% (16/33) 1 death	66.66% (2/3)	54.54% (6/11) 1 death	66.66% (6/9)	100% (6/6)	83.33% (5/6)	66.66% (2/3)	0% (0/1)	0% (0/1)		58.90% (43/73)

In the "Others" subgroup are cases not specified about the movement disorder. CR: Complete recovery, DTN: Dystonia, MCL: Myoclonus, MD: Movement disorder, Md: Median, NA: Not available/not applicable, PKN: Parkinsonism, SD: Standard deviation, MCL: Myoclonus

that NCC can affect distant brain regions from the cysticercus location. Therefore, maybe the assumption of Alarcón *et al*. (2017) that patients with PKN had more inflammation could explain these findings; in addition, different CSF protein levels in individuals with and without MDs further support this hypothesis. Moreover, perhaps the abnormal movements only occurred after a misleading inflammatory reaction caused by the parasite death and cystic degeneration.

Takayanagui and Jardim studied only individuals with NCC, in 1983, from their 238 patients, of which 2 developed PKN (0.40%);[26] in 1990, among 151 patients, only 1 developed PKN (0.66%).[32] Thus, the incidence of PKN in patients with NCC is probably rare, affecting <1% of the individuals. From another standpoint, it is often possible to observe these clinical manifestations in developing countries, where *T. solium* is more prevalent.[4] For example, Alarcón *et al*. (2017) assessed 590 individuals with NCC, among these, 23 patients (3.89%) presented PKN and others MDs.

Some individuals with PKN presented other less prominent or late-onset clinical manifestations, such as seizure, psychiatric symptoms, lower limb dystonia, blepharospasm, cognitive decline, ataxia, apraxia, and myokymia. Sharma *et al*. reported the interesting case of an individual with progressive supranuclear palsy syndrome and early cognitive decline.[68]

One common management was the VPS in the ICH cases, which not surprisingly alleviated the parkinsonian symptoms, possibly by the aforementioned mechanisms.[86] Prashantha *et al*. reported a case where

only VPS and levodopa were attempted without any cysticidal drug, and the patient had a complete recovery.[60] In addition, some individuals presented levodopa-responsive PKN. In the case of Sá *et al*., the prescription of albendazole led to the alleviation of symptoms, and later reduction of the levodopa dose.[48] On the other hand, Lima *et al*. reported a case where albendazole worsened PKN symptoms.[69] Probably, the cysticidal drug would have caused an intense inflammatory reaction, which may be more commonly observed in forms involving intraventricular cysts and ependymitis/encephalitis.[87]

MYOCLONUS – Cysticidal drugs should be tried

Only four cases of myoclonus (MCL) and NCC were found in the literature, so there is scarce clinical information about this involuntary movement. The MCL types were palatal, oculopalatal, and multifocal. No specific cystic location in the neuroimaging was associated with MCL; also, each article found lesions in a different location. The cysts were found in the dentate nucleus, meninges, pons, and frontal gyrus.

The source of MCL was subcortical in 75% of individuals. This feature was interesting because when compared with drug-induced MDs that specifically affect determined neurotransmitters, a microscopical lesion would theoretically lead to abnormalities in electrodiagnostic studies.[88] However, only the study of Puri *et al*. revealed an electroencephalogram with the presence of generalized synchronous as well as the asynchronous, nonperiodic paroxysmal discharge of polyspike, spike, and sharp waves with well-modulated alpha background activity.[33]

In one individual, cysticidal drugs were not attempted. It is noteworthy that he was the only that did not have a complete recovery.

Dystonia – Maybe it is a vascular mechanism

Jiménez-Jiménez *et al*. reported the first case of NCC and dystonia (DTN); unfortunately, neuroimaging was only performed years after the first presentation, which may cast doubt on the possibility of the etiological diagnosis.[34] The DTN types reported in the literature were focal (blepharospasm, cervical, scalp, and writer's cramp), segmental (Meige syndrome), and hemidystonia.

More than 70% of the patients had at least one lesion localized in the basal ganglia region. Other areas affected were the thalamus, cortical and subcortical regions of the frontal lobe, parietal lobe, and temporal lobe.

All the patients that had a full recovery received cysticidal medication (albendazole or praziquantel). In 2016, Yoganathan *et al*. reported the case of an individual who developed Meige syndrome and PKN with a Japanese encephalitis and NCC coinfection.[78] This case reaffirms what was previously stated that the presence of specific features of NCC should always be searched in these patients, as we already exemplified in chorea cases.

Alarcón *et al*. (2017) proposed that DTN and chorea may be due to vascular mechanisms, which involve the perforating branches of the middle cerebral artery, leading to cerebral ischemia, blood–brain barrier damage, and increased levels of excitatory neurotransmitters, as already observed in altered glycemic states.[89] This hypothesis could partially explain the fact that the majority of the DTN and chorea cases had small cystic lesions, without edema or distortion of adjacent structures by pressure. Nevertheless, this does not explain why some individuals had unspecific localized lesions or active cysts localized in the basal ganglia.[45]

Myokymia (MKM)– Exceptionally highly reported

Myokymia (MKM) is an abnormal movement uncommonly reported with systemic diseases. However, from the group of systemic diseases, NCC seems to be the most commonly associated with MKM. In a review by Alarcón and Giménez-Roldán about MDs and systemic diseases, they only found reports of MKM related to NCC.[90]

Yang *et al*. described a case of hemifacial spasm secondary to arachnoiditis due to a cyst in the cerebellopontine angle, in which the cysticercus was removed and the facial nerve was liberated with complete recovery of the symptoms.[84] This case was probably the first to show neurosurgical intervention in MKM associated with NCC. The cases reported by Revuelta Gutierrez *et al*. and Razdan *et al*. of MKM caused by subarachnoid NCC were both treated by a course of corticosteroids.[41,64] The MKM had the second-best prognosis, where > 80% of the patients had a full recovery. However, it is worth mentioning that only six cases were found investigating the literature.

Keane in 1993 reported the case of a young adult male who presented with facial spasms accompanied by pretectal signs, and neuroimaging revealed obstructive hydrocephalus.[36] A VPS was performed with complete resolution of the symptoms. After about 3 years, the patient presented with similar symptoms, and the VPS was revised; after 1 month, he had a full recovery. Beydoun in 1994 reported a similar case presentation, but VPS was not performed and the patient did not have a full recovery.[37]

Conclusion

In sum, MDs associated with NCC already reported in the literature were PKN, ataxia, chorea, dystonia, tremor, myokymia, myoclonus, ballism, and tics. More than half of the individuals were from South American countries and about 25% from Asian countries. The most common MD reported was PKN. In addition, individuals who developed PKN had the worst prognosis of any other MD associated with NCC, except for ballism and tics, each with one case reported in the literature. Generally, individuals with chorea and dystonia have more specifically localized lesions. Due to the rarity of the clinical manifestations presented in this review, it is worth mentioning that case reports were included in the study. Because of this, an important limitation of the study is that it is not a systematic review. We believe that the majority of the cases presented in the literature were only reported because patients had classical clinical manifestations that prompted neuroimaging investigation, which was suggestive of NCC, and later supported by laboratory examinations. Therefore, the association between NCC and MD is probably underreported. Clinicians should be highly suspicious of this association, mainly in endemic areas for cysticercosis.

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Conflicts of interest
There are no conflicts of interest.

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Perú. Hemifacial spasm secondary to arachnoiditis due to neurocysticercosis: Clinical image. World Neurosurg 2020;143:180-2.
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Other 1: FreeText and MeSH search terms in the US National Library of Medicine

Category	Search terms	Results
Chorea	((“chorea”[MeSH Terms] OR “chorea”[All Fields]) OR “choreas”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	7
Tremor	(((((“tremor”[MeSH Terms] OR “tremor”[All Fields]) OR “tremors”[All Fields]) OR “tremoring”[All Fields]) OR “tremorous”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	10
Parkinsonism	((((((((((“parkinson disease”[MeSH Terms] OR (“parkinson”[All Fields] AND “disease”[All Fields])) OR “parkinson disease”[All Fields]) OR “parkinsons”[All Fields]) OR “parkinson”[All Fields]) OR “parkinson s”[All Fields]) OR “parkinsonian disorders”[MeSH Terms]) OR (“parkinsonian”[All Fields] AND “disorders”[All Fields])) OR “parkinsonian disorders”[All Fields]) OR “parkinsonism”[All Fields]) OR “parkinsonisms”[All Fields]) OR “parkinsons s”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	33
Myoclonus	(“myoclonus”[MeSH Terms] OR “myoclonus”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	3
Ataxia	((“ataxia”[MeSH Terms] OR “ataxia”[All Fields]) OR “ataxias”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	28
Dystonia	(((((“dystonia”[MeSH Terms] OR “dystonia”[All Fields]) OR “dystonias”[All Fields]) OR “dystonic disorders”[MeSH Terms]) OR (“dystonic”[All Fields] AND “disorders”[All Fields])) OR “dystonic disorders”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	10
Myokymia	((“myokymia”[MeSH Terms] OR “myokymia”[All Fields]) OR “myokymias”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	3
Ballism	((“dyskinesias”[MeSH Terms] OR “dyskinesias”[All Fields]) OR “ballism”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	22
Tic	(“tic”[Journal] OR “tic”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	1
Dyskinesia	(((“dyskinesiae”[All Fields] OR “dyskinesias”[MeSH Terms]) OR “dyskinesias”[All Fields]) OR “dyskinesia”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	23
Stuttering	((((((((((“stammerers”[All Fields] OR “stammers”[All Fields]) OR “stutterer”[All Fields]) OR “stutterer s”[All Fields]) OR “stutterers”[All Fields]) OR “stuttering”[MeSH Terms]) OR “stuttering”[All Fields]) OR “stammer”[All Fields]) OR “stammering”[All Fields]) OR “stutter”[All Fields]) OR “stuttered”[All Fields]) OR “stutters”[All Fields]) OR “stutterings”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	1
Restless legs syndrome	((“restless legs syndrome”[MeSH Terms] OR ((“restless”[All Fields] AND “legs”[All Fields]) AND “syndrome”[All Fields])) OR “restless legs syndrome”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	0
Akathisia	((((“akathisias”[All Fields] OR “psychomotor agitation”[MeSH Terms]) OR (“psychomotor”[All Fields] AND “agitation”[All Fields])) OR “psychomotor agitation”[All Fields]) OR “akathisia”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	1
Restlessness	((((“psychomotor agitation”[MeSH Terms] OR (“psychomotor”[All Fields] AND “agitation”[All Fields])) OR “psychomotor agitation”[All Fields]) OR “restlessness”[All Fields]) OR “restless”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	1
Hyperkinetic	(“hyperkinetic”[All Fields] OR “hyperkinetics”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	0
Hypokinetic	((“hypokinesia”[MeSH Terms] OR “hypokinesia”[All Fields]) OR “hypokinetic”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	0

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Other 1: Contd...

Category	Search terms	Results
Bradykinesia	((“hypokinesia”[MeSH Terms] OR “hypokinesia”[All Fields]) OR “bradykinesia”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	2
Movement disorder	((((“movement disorders”[MeSH Terms] OR (“movement”[All Fields] AND “disorders”[All Fields])) OR “movement disorders”[All Fields]) OR (“movement”[All Fields] AND “disorder”[All Fields])) OR “movement disorder”[All Fields]) AND (((“neurocysticercosis”[MeSH Terms] OR “neurocysticercosis”[All Fields]) OR “neurocysticercoses”[All Fields]) OR ((“cysticercosis”[MeSH Terms] OR “cysticercosis”[All Fields]) OR “cysticercoses”[All Fields]))	45
Total		**190**