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 clinicopathological 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.
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].\cite{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.\cite{3}

When the invasion of the CNS occurs, the cysticerci usually develop in five main stages [Figure 3].\cite{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.\cite{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.\cite{5} These stages are the vesicular, colloidal (colloidal–vesicular), granular (granular–nodular), and calcific (nodular) stages.\cite{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.\cite{4} It is noteworthy that, together with neurosyphilis and AIDS, NCC is considered one of the “great simulator/mimickers” of other diseases.\cite{8} The most common presentation is seizures.\cite{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.\cite{9} In this context, movement disorders (MDs) usually occur in about 3% of individuals with NCC.\cite{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.

### Methods

#### Definitions

NCC was defined by the revised diagnostic criteria and degrees of diagnostic certainty for NCC by Del Brutto *et al.* [Table 1].\cite{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.\cite{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.\cite{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 |
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.” 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
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. 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. 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.

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. 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
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. Another explanation for this involuntary movement could be an exacerbated immune response because many patients had alleviation of the symptoms when corticosteroids were started. In addition, others had ICH, and a ventriculoperitoneal shunt (VPS) was performed to alleviate the symptoms. Vasculitis of the right middle cerebral artery was observed in one individual. 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.

One interesting fact is that in east Asian countries, the coinfection of NCC and Japanese encephalitis is being reported. 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.

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. Assis and Tenuto reported positive serum and CSF for cysticercosis in a patient with the association of PKN and ICH.

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. 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

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**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.” 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. 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.

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. 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.”

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**Figure 4:** Flowchart of the search process found in our study and we will further discuss them in the following sections.
Rissardo, et al.: NCC-associated movement disorder

| Reference          | Country/Year | n cases | Age/sex | Neuroimaging          | Follow-up | Important CH and CM                                                                 |
|--------------------|--------------|---------|---------|-----------------------|-----------|-------------------------------------------------------------------------------------|
| 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 herbas reduced the symptoms. Presence of hurt murm. 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 sufacetamide (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 cysterci, 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 perilocal 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. Phenytin 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 preoptine areas) of cysterci (racemose form); hydrocephalus | CR        | CM: VPS was performed. Dexamethasone, ALB, and phenobarbit. The symptoms recovered |

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...
| Reference                          | Country/Year | n cases | Age/sex | Neuroimaging | Follow-up | Important CH and CM                                                                 |
|-----------------------------------|--------------|---------|---------|--------------|-----------|------------------------------------------------------------------------------------|
| Takayanagi 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                                             |
| Takayanagi 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 cystercerosis                                               |
| 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                                                              |
| 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 cystercus | 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 |
| García 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. Cystidial 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...
| 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 preoptine cistern, severe hydrocephalus | CR        | CH: PKN+ICH                                                                  |
| Reference | Country/Year | n cases | Age/sex | Neuroimaging | Follow-up | Important CH and CM |
|-----------|-------------|---------|---------|--------------|-----------|---------------------|
| Rissardo, 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 |
| Weiner | 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 |
| 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                               |
| 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; hypodense 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             |

**Dystonia**

| Reference          | Country/Year    | n cases | Age/sex | Neuroimaging                                                                 | Follow-up   | Important CH and CM                                                                 |
|--------------------|-----------------|---------|---------|-----------------------------------------------------------------------------|-------------|------------------------------------------------------------------------------------|
|                     |                 |         |         |                                                                             |             |                                                                                     |

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Table 2: Contd...

| Reference        | Country/Year | n cases | Age/sex | Neuroimaging                                      | Follow-up | Important CH and CM                                                                 |
|------------------|--------------|---------|---------|--------------------------------------------------|-----------|--------------------------------------------------------------------------------------|
| 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                 |
|                  |              |         |         | Bilateral putamen cysts                           | CR        | CH: cervical DTN. CM: ALB. The symptoms recovered.                                    |
|                  |              | 37/female |         |                                                  |           |                                                                                      |
| 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 |           |                                                                                      |
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.

Takayanagi 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.[66] Prashantha et al. reported a case where

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only VPS and levodopa were attempted without any cysticidal drug, and the patient had a complete recovery. 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. On the other hand, Lima et al. reported a case where albendazole worsened PKN symptoms. 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.

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. 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.

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. 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. 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. 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.

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.

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. 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. 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. 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.
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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### 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 "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) 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 "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) 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 "parkinsons"[All Fields]) OR "parkinsonian disorders"[All Fields]) OR ((("neurocysticercosis"[MeSH Terms] OR "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) OR "cysticercosis"[All Fields]) OR "cysticercoses"[All Fields]))) | 33      |
| Myoclonus           | ((("myoclonus"[MeSH Terms] OR "myoclonus"[All Fields]) AND ((("neurocysticercosis"[MeSH Terms] OR "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) OR "cysticercosis"[All Fields]) OR "cysticercoses"[All Fields]))) | 3       |
| Ataxia              | ((("ataxia"[MeSH Terms] OR "ataxia"[All Fields]) OR "ataxies"[All Fields]) AND ((("neurocysticercosis"[MeSH Terms] OR "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) 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 "dystonias"[All Fields]) OR "dystonic disorders"[All Fields]) AND ((("neurocysticercosis"[MeSH Terms] OR "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) 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 "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) 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 "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) OR "cysticercosis"[All Fields]) OR "cysticercoses"[All Fields]))) | 22      |
| Tic                 | ((("tic"[Journal] OR "tic"[All Fields]) AND ((("neurocysticercosis"[MeSH Terms] OR "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) 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 "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) 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 "stammering"[All Fields]) OR "stuttering"[All Fields]) OR "stuttered"[All Fields]) OR "stutters"[All Fields]) OR "stutterings"[All Fields]) AND ((("neurocysticercosis"[MeSH Terms] OR "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) OR "cysticercosis"[All Fields]) OR "cysticercoses"[All Fields]))) | 1       |
| Restless legs       | ((("restless legs syndrome"[MeSH Terms] OR "restless legs"[All Fields] AND "legs"[All Fields]) AND "syndrome"[All Fields]) OR "restless legs syndrome"[All Fields]) OR ((("neurocysticercosis"[MeSH Terms] OR "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) 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 "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) 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 "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) OR "cysticercosis"[All Fields]) OR "cysticercoses"[All Fields]))) | 1       |
| Hyperkinesia        | ((("hyperkinesia"[All Fields] OR "hyperkinectic"[All Fields]) OR "hyperkinetics"[All Fields]) AND ((("neurocysticercosis"[MeSH Terms] OR "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) OR "cysticercosis"[All Fields]) OR "cysticercoses"[All Fields]))) | 0       |
| Hypokinesia         | ((("hypokinesia"[MeSH Terms] OR "hypokinesia"[All Fields]) OR "hypokinet"[All Fields]) AND ((("neurocysticercosis"[MeSH Terms] OR "neurocysticercoses"[All Fields]) OR "neurocysticercoses"[All Fields]) OR ((("cysticercosis"[MeSH Terms] OR "cysticercoses"[All Fields]) OR "cysticercosis"[All Fields]) OR "cysticercoses"[All Fields]))) | 0       |
| 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     |