Isolated Spinal Cord Neurocysticercosis

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ABSTRACT

The incidence of neurocysticercosis is increasing in the US. The diagnosis is primarily made based on imaging findings, with clinical presentation and epidemiological exposure also playing a role. The differential diagnosis for neurocysticercosis (NCC) is extensive, and being able to differentiate between these conditions on imaging is crucial to making a proper diagnosis. Herein we present a case of a 37-year-old female who presented with lower extremity weakness and was found to have isolated spinal NCC. In this article, we will discuss the symptoms and imaging findings of neurocysticercosis to help guide diagnosis and management.

CASE REPORT

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A 37-year-old Hispanic female with no past medical history presented to an outside hospital emergency department complaining of lower back and hip pain after falling on the sidewalk on the day before. She was found to have tenderness to palpation at the level of L4-L5 and of the right paraspinal muscles. At that time her sensation and strength were intact bilaterally and she was ambulatory with steady gait. Radiographs of the lumbar spine and right hip were negative for acute abnormality, and she was sent home with a diagnosis of muscle strain. She subsequently began to have weakness in her lower extremities, which continued to progress, and she returned to the emergency department one week after her initial presentation complaining of abdominal tenderness, low back pain, and lower extremity weakness. She noted that any movement of her lower extremities caused significant lower back pain, making it difficult for her to walk. On physical examination she was found to have tenderness of the paraspinal muscles and decreased range of motion secondary to pain. Her gait was antalgic and strength was intact in the lower extremities. She had no saddle anesthesia. She underwent CT of the thoracolumbar spine and contrast-enhanced MRI of the thoracolumbar spine, which demonstrated an intradural, intramedullary peripherally enhancing expansile cystic lesion occupying the majority of the spinal canal at the T8 level (Figures 1-4). Subsequent MRI of the brain was negative for additional lesions. Laboratory values were within normal limits.

Upon admission, neurology and neurosurgery were consulted. Initial neurological exam showed 3+ patellar and Achilles reflexes with clonus and decreased strength in the hips, knees, and with plantar flexion and dorsiflexion. Extensive laboratory testing was done, including lumbar puncture and serological evaluation, all of which was unremarkable. She was started on steroids and ciprofloxacin for an incidentally diagnosed urinary tract infection.

The patient underwent a T7-T9 laminectomy with intradural biopsy and subtotal resection of the intramedullary thoracic spinal lesion and expansion duraplasty the week after her second presentation to the emergency department, though

no intraoperative images were obtained. Pathological examination showed this lesion to be consistent with cysticercosis, and infectious disease was consulted for further management (Figure 5). She subsequently began a 14-day course of albendazole, and steroids were continued. She has since completed her course of medication. At the latest follow-up, her lower extremity strength and sensation has continued to improve with physical therapy, though she endorses heaviness in her legs and back and uses a walker for ambulation at all times. She also endorses migraines 1-2 times per week since completing her steroid therapy.

DISCUSSION

Etiology & Demographics:

Neurocysticercosis (NCC) is an uncommon disease in the United States caused by the larvae of the pork tapeworm *Taenia solium*. Estimates of its incidence range from 0.2-0.6 cases per 100,000 up to 1.5-1.8 in Hispanics, which is attributed to higher immigration status among the Hispanic population [1]. The incidence of NCC in the US has increased, and there are more cases of NCC diagnosed in the US every year than in all other countries combined [2]. This rise has been driven by an influx of immigrants to non-endemic areas and contributed to local spread of the disease [3]. Brain parenchymal disease is most commonly reported, though there has been a recent increase in the incidence of extraparenchymal disease. The majority of cases present with parenchymal NCC (91%), with the remainder having ventricular cysts (6%), subarachnoid cysts, and spinal cysts (0.2%), and these patients often have disease in more than one of these locations [4]. Though intraparenchymal lesions are most common, it is important to consider NCC in isolated extraparenchymal lesions as well.

The most common cause is *Taenia* solium, though there are eight species of cestodes (aka tapeworms) that can infect humans [5]. Pigs are usually the intermediate host leading to human infection, and it is the ingestion of the larvae from the intermediate host that permits larval invasion. These larvae develop into the adult form and persist in the intestinal tract, where they continue to produce eggs which can further transmit the disease. NCC develops via a fecal-oral route when these eggs are ingested. They hatch and the embryos invade directly through the intestinal mucosa to enter the blood stream. These can reach the brain, as well as other parts of the body, where they will trigger an inflammatory response followed by calcification [6].

Clinical & Imaging Findings:

NCC can present with a variety of symptoms including seizures (66%), hydrocephalus (16%) and headaches (15%), in addition to altered vision, focal neurological deficits, and meningitis [1,8]. CT and MRI of the brain and spinal cord are commonly used diagnostic tools to identify NCC lesions. CT imaging has a high sensitivity and specificity in most forms of NCC and is superior to MRI in identifying calcified granulomas [5]. MRI offers the advantage of higher contrast resolution, which can help to identify lesions that are not obvious on CT. This is particularly helpful in the evaluation of ventricular lesions. There are five disease stages – non-cystic, vesicular, colloidal vesicular, granular nodular, calcified nodular – and the imaging findings depend on the stage of the disease. The first stage, the non-cystic stage, is often radiologically occult. The remaining four stages can be differentiated based on imaging findings. There is progressive rim-enhancement and surrounding edema as the cyst matures until it reaches the calcified nodular stage, which has no rim-enhancement or edema [5]. Ventricular cysts are often diagnosed based on the presence of ventricular obstruction, mass effect, the presence of a cyst rim, or CSF flow void adjacent to the cyst [7]. A central scolex (larval head) is another important imaging finding and can be seen in almost 50% of cases [7].

The primary imaging finding of NCC is rim-enhancing cystic lesions with surrounding edema, which can also be seen with many other conditions. Because of this extensive differential diagnosis, it is important to be able to properly identify NCC and differentiate it from these other similar appearing conditions. Several conditions can present with similar symptoms and show single or multiple rim-enhancing nodules, including tuberculoma, pyogenic brain abscess, mycotic granuloma, and primary or metastatic brain tumor [9]. Cystic lesions can be seen with other parasitic infections including echinococcosis and coenurosis. Parenchymal calcifications can be seen with metabolic disorders, vascular malformations, intracranial neoplasms, and congenital anomalies [10].

Diagnostic criteria for NCC can be broken down to the absolute, neuroimaging, and clinical/exposure criteria. The absolute criteria include direct visualization of subretinal cysticercus, histological confirmation from a surgical sample, or demonstration of a scolex within a cystic lesion on imaging. The neuroimaging criteria are summarized below (Table 1). Clinical/exposure criteria are related to antigen testing, cysticercosis seen outside the CNS, close contact with an infected individual, and symptoms. Definitive diagnosis requires one absolute criterion with two major neuroimaging criteria plus at least one clinical/exposure criteria, one major and one confirmative neuroimaging criteria plus at least one clinical/exposure criteria, or one major neuroimaging criterion plus two clinical/exposure criteria [11].

Treatment & Prognosis:

Treatment depends on several factors, including the form and type of disease, the location and number of cysts, and patient symptoms [12]. Treatment is based on medications that are used to treat the symptoms, such as antiepileptic drugs, cysticidal agents, and corticosteroids. Other immunosuppressive or anti-inflammatory agents can also be used to help control the potentially harmful host inflammatory response. Surgical intervention can also be considered, though it is rarely used due to early diagnosis and the prevalence of pharmacologic therapy [13]. Overall, the prognosis is good if the patient receives the appropriate treatments.

Differential Diagnoses:

As stated in the differential diagnoses table below, there are a few other considerations in the differential diagnosis of NCC. Metastasis, tuberculoma, and tuberculous and other

pyogenic abscesses are the are the primary conditions that should be ruled out when considering NCC.

Metastasis

Metastasis can present as hypodense lesions on CT, similar to NCC. MRI must be used to differentiate these diseases. Both are hyperintense on T2-weighted imaging, though metastases present as homogeneously enhancing T1 hypointense lesions. Conversely, NCC is hyperintense on T1-weighted imaging and enhances peripherally. Metastases are also intensely FDG avid on PET, while NCC does not demonstrate FDG avidity.

Tuberculoma

Similar to metastases, tuberculomas also present as hypodense lesions on CT. The main distinctions between tuberculoma and NCC are that tuberculomas are isointense on T1-weighted imaging, isointense to hypointense on T2-weighted imaging, and intensely FDG avid on PET. NCC lesions are hyperintense on T1-weighted imaging and are not FDG avid on PET.

Tuberculous and other pyogenic abscesses

Abscesses and NCC have considerable overlap on imaging. The main differences are that abscesses show diffusion restriction on DWI and an FDG avid rim on PET. NCC lesions do not demonstrate diffusion restriction and show no FDG avidity on PET.

TEACHING POINT

Neurocysticercosis (NCC) should be suspected in patients with rim-enhancing intramedullary lesions on CT or MRI, especially in those from endemic areas or who have had a close exposure. Though rare, isolated spinal cord lesions can be seen, and the lack of brain lesions should not exclude NCC from the differential.

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FIGURES

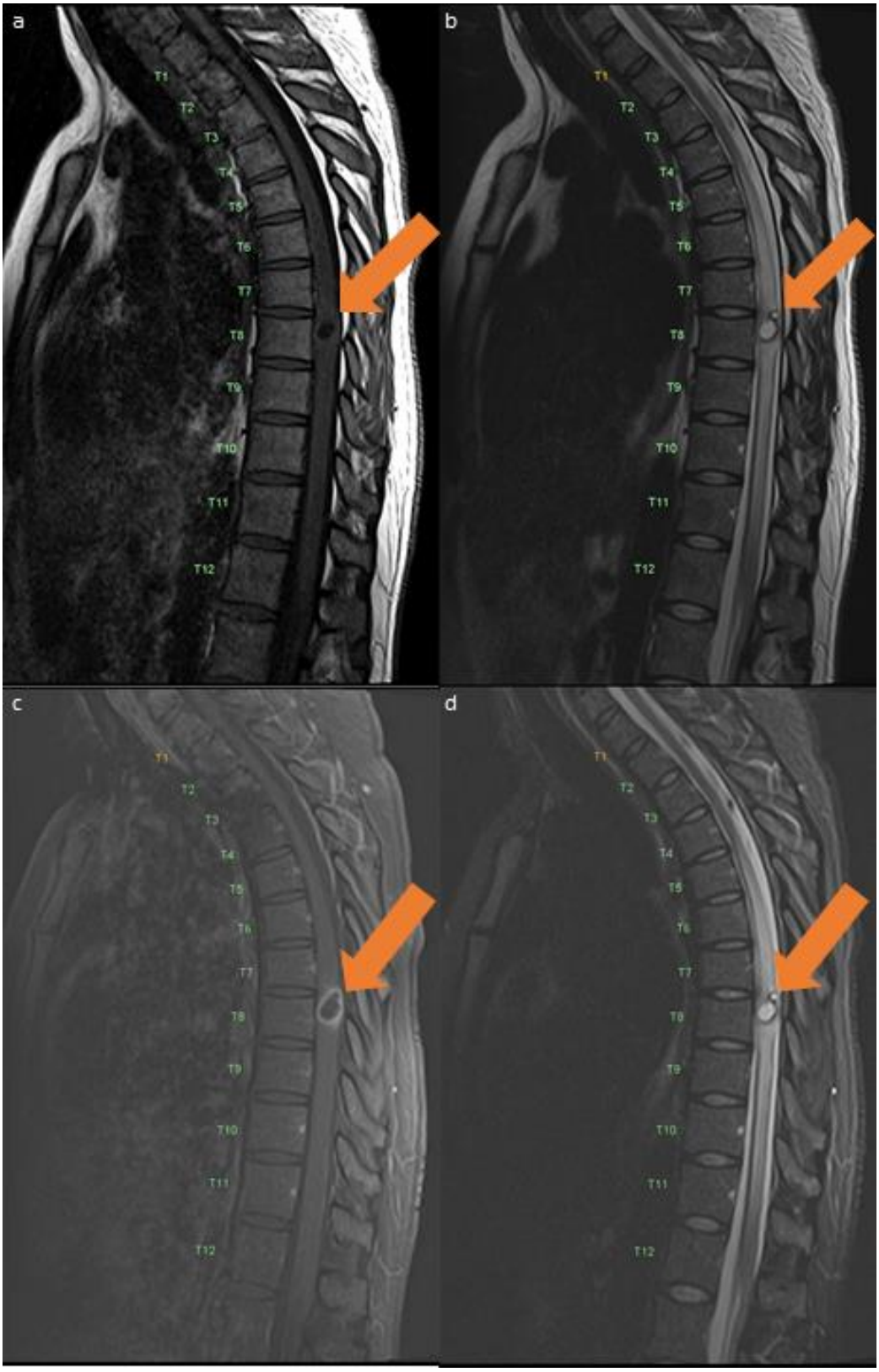

Figure 1: 37-year-old Hispanic female diagnosed with Neurocysticercosis (NCC).

Findings: Sagittal pre- and post-contrast 3T MRI Images of the thoracic spine are shown above, with the arrows indicating an intradural, intramedullary spinal lesion which was determined to be NCC. These images show a T1w hypointense, T2w/STIR hyperintense, peripherally enhancing intradural, intramedullary lesion at the level of T8.

Technique: a) T1-weighted b) T2-weighted c) T1-weighted Post-Contrast d) STIR sagittal MRI of the thoracic spine.

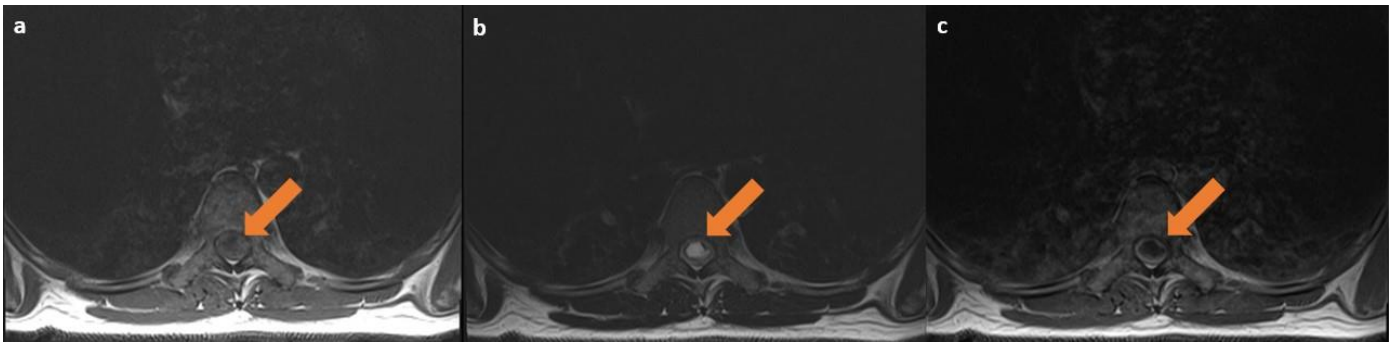

Figure 2: 37-year-old Hispanic female diagnosed with Neurocysticercosis (NCC).

Findings: Axial pre- and post-contrast 3T MRI Images of the thoracic spine are shown above, with the arrows indicating an intradural, intramedullary spinal lesion which was determined to be NCC. These images show a T1w hypointense, T2w hyperintense, peripherally enhancing intradural, intramedullary lesion at the level of T8.

Technique: a) T1-weighted b) T2-weighted c) T1-weighted Post-Contrast

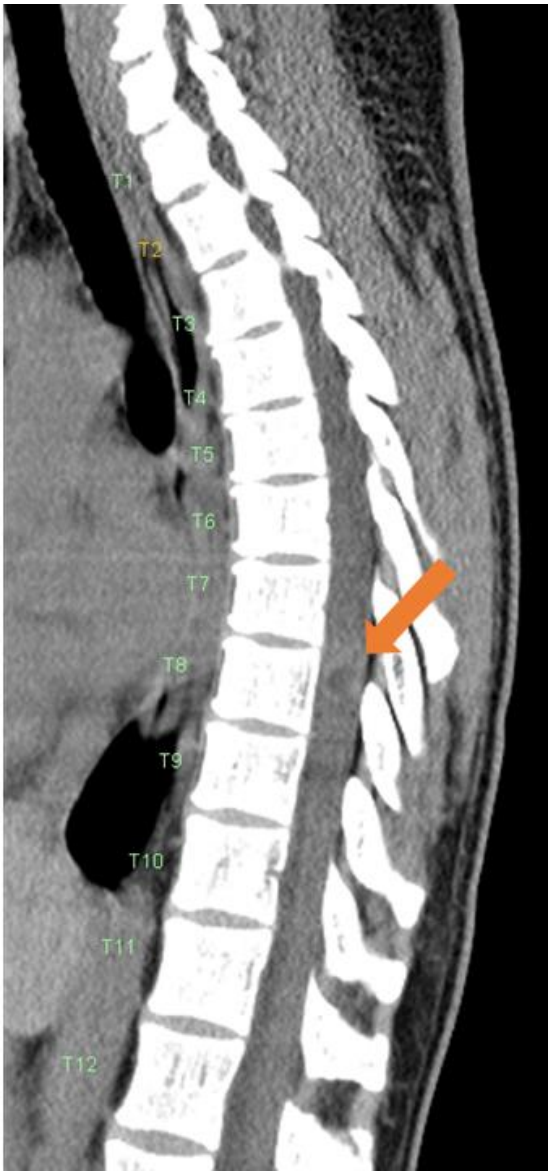

Figure 3 (left): 37-year-old Hispanic female diagnosed with Neurocysticercosis (NCC).
Findings: Sagittal CT image of the thoracic spine is shown above, with the arrow indicating an intradural, intramedullary spinal lesion which was determined to be NCC. These images show a peripherally high attenuating intramedullary spinal lesion with central low attenuation at the level of T8.

Technique: Sagittal CT image of the thoracic spine without IV contrast.

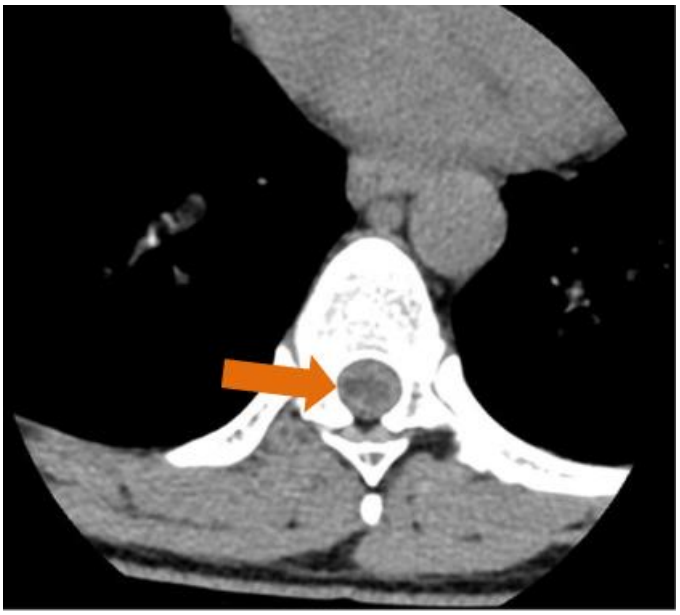

Figure 4 (left): 37-year-old Hispanic female diagnosed with Neurocysticercosis (NCC).

Findings: Axial CT image of the thoracic spine is shown above, with the arrow indicating an intradural, intramedullary spinal lesion which was determined to be NCC. These images show a peripherally high attenuating intramedullary spinal lesion with central low attenuation at the level of T8.

Technique: Axial CT image of the thoracic spine at the level of T8 without IV contrast.

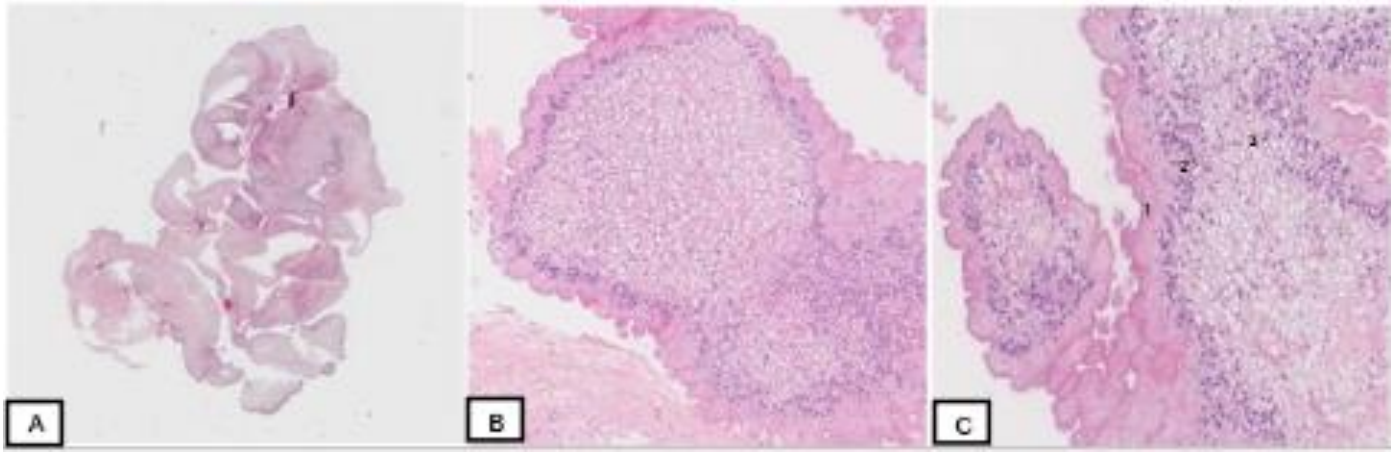

Figure 5: 37-year-old Hispanic female diagnosed with Neurocysticercosis (NCC).

Findings: Histological images from the intradural, intramedullary thoracic cyst resection. This was consistent with NCC and had a cystic wall containing three layers: (1) a wavy outer eosinophilic layer (cuticular layer), (2) a cellular layer (granular layer) composed of uniform small dark nuclei, and (3) a reticular inner layer composed of loosely textured stroma.

Technique: Hematoxylin and eosin (H&E) stain, a) magnification 20x b) magnification 100x c) magnification 200x

Major Criteria	Confirmatory Criteria	Minor Criteria
Cystic lesions without discernable scolex	Resolution of cystic lesions after therapy	Obstructive hydrocephalus
Enhancing lesions	Spontaneous resolution of single small, enhanced lesions	Abnormal enhancement of basal leptomeninges
Multilobulated cystic lesions in the subarachnoid space	Migration of ventricular cysts on sequential studies	
Typical parenchymal brain calcifications		

Table 1: Summary table of neuroimaging criteria for the diagnosis of neurocysticercosis[11].

Etiology	Fecal-oral transmission of the eggs of *Taenia solium*, which invade the digestive tract and can reach the brain, triggering an inflammatory response.
Incidence	0.2-0.6 cases per 100,000 people
Gender Ratio	1 male: 1 female[14].
Age Predilection	None.
Risk Factors	Being from an endemic area, close contacts from endemic area, poor personal hygiene, being unable to recognize cysticerci-containing meat, poor pig-raising practices, and a history of passing tapeworm proglottides[15].
Treatment	Anti-helminthics (e.g., Albendazole), corticosteroids, and anti-seizure medications
Prognosis	Affected by the number of lesions and extent of inflammation, but typically good[16].
Imaging Findings	Dependent on the stage of infection. The earliest lesions are often radiographically occult. Cystic lesions are T2w and STIR hyperintense and show peripheral enhancement on both CT and MRI. End-stage lesions are calcified and do not enhance[6].

Table 2: Summary table of neurocysticercosis.

Disease	CT	MRI - T1	MRI - T2	MRI- DWI	Enhancement	PET
NCC	Hypodense	Hyperintense	Hyperintense	No restriction	Peripheral	Not FDG Avid
Metastasis	Hypodense	Hypointense	Hyperintense	No restriction	Homogeneous	Intensely FDG avid
Tuberculoma	Hypodense	Isointense	Isointense or hypointense	No restriction unless centrally necrotic	Peripheral	Intensely FDG avid
Tuberculous and other Pyogenic Abscess	Hypodense	Hyperintense	Hyperintense	Restriction	Peripheral	FDG avid rim

Table 3: Differential diagnosis table for Neurocysticercosis.

ABBREVIATIONS

NCC = Neurocysticercosis

KEYWORDS

Neurocysticercosis; MRI; Spinal Cord; Brain; Parasite