Case Report
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Unmasking a Hidden Culprit: Neurocysticercosis, an Overlooked Cause of Acquired Epilepsy

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Neurocysticercosis (NCC) is a common parasitic brain infestation caused by the ingestion of Taenia solium eggs, predominantly in developing countries. In this report, we presented the case of a 44-year-old woman who exhibited stroke symptoms and had a decade-long history of recurrent headaches and epilepsy. At presentation, a non-contrast computed tomography scan of the brain was performed and revealed hypodense oval lesions and calcified cysts in both cerebral hemispheres, strongly indicative of NCC. The patient responded positively to treatment with dexamethasone, albendazole, and carbamazepine. This case study underscores the importance of neuroimaging in investigating patients with neurological conditions like epilepsy, especially in developing countries. Early diagnosis and effective treatment are crucial in preventing and controlling NCC, reducing its impact on public health. **(2024;14:42-46)**

Key words: Epilepsy, Neurocysticercosis, Parasitic diseases, *Taenia solium*

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Introduction

Neurocysticercosis (NCC) is a neglected tropical parasitic infestation of the central nervous system (CNS) caused by the encysted larvae of the pork tapeworm, *Taenia solium* (*T. solium*).[1] It is the most common parasitic infestation of the brain worldwide and is prevalent in areas with poor sanitation and limited access to clean water.[2] NCC poses a significant public health concern worldwide, particularly in developing countries.[2] The distribution of NCC varies greatly, with its occurrence being relatively rare in developed nations but more prevalent in regions of developing countries, including Africa, Southeast Asia, and Latin America.[3] Despite its profound impact on individuals and communities, NCC remains underreported and often misdiagnosed.

The global prevalence of NCC is not well documented, but the World Health Organization (WHO) has estimated that the total number of people affected by NCC, including both symptomatic and asymptomatic cases, ranges from 2.56 to 8.30 million.[2] NCC significantly contributes to preventable cases of epilepsy worldwide.[4] In countries where the parasite is endemic, NCC is estimated to account for about 30% of all epilepsy cases.[2] Certain developing nations have reported high coexistence rates, reaching as high as 70% of NCC and epilepsy cases.[5] The exact mechanism behind NCC-induced seizures remains unclear, but it is believed to be linked to the inflammatory response that occurs during cyst degeneration.[6]

With the advancement of modern neuroimaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) scans, the detection of NCC has become more accessible and accurate. However, optimal management of this condition requires a multidisciplinary approach involving neurologists, radiologists, infectious disease specialists, and neurosurgeons. The clinical presentation of NCC varies depending on factors such as the number, size, and location of cysts and the host immune response.[7] The integration of advanced diagnostic tests, antiparasitic drugs, anti-inflammatory treatments, and minimally invasive surgical interventions has significantly improved the prognosis of patients with NCC.[7]

This case report describes a Nigerian female from a rural community who exhibited stroke symptoms and had a 10-year history of recurring seizures and headaches. After neuroimaging, the patient was diagnosed with NCC. This case underscores the importance of neuroimaging and considers NCC a potential differential diagnosis in patients presenting with neurological symptoms in endemic regions. Early diagnosis and prompt treatment can significantly improve patients' outcomes.

Case Report

A 44-year-old female presented to the neurology clinic with a 3-day history of left limb weakness and difficulty walking. She did not manifest additional symptoms, such as alterations in consciousness, fever, vomiting, neck stiffness, or neck pain. Ten years ago, she was diagnosed with epilepsy, specifically classified as right focal unaware with bilateral tonic-clonic seizures; however, she did not undergo any neuroimaging investigation. Throughout this period, she had been experiencing recurrent headaches. She was prescribed carbamazepine tablets, 200 mg twice daily, to be taken orally for her epilepsy. However, she was not regular with her medication regimen, which led to seizures occurring a few days per month.

Two months prior to her presentation, she had memory impairment and slurred speech, which resolved after a few days. Her memory issues were evident in her difficulty recalling family members' names and recent events. She had no history of sphincteric abnormalities and had not smoked, consumed alcohol, or used recreational drugs. She was not previously diagnosed with hypertension or diabetes mellitus. As a trader and consumer of pork, she lived her entire life in an area with poor sanitary conditions and a lack of potable water, where free roaming of pigs was common.

Her examination revealed a temperature of 37.0°C, a heart rate of 78 beats/minute, a blood pressure of 120/76 mmHg, a respiratory rate of 20 breaths/minute, and an oxygen saturation of 97% in room air. Neurological examination revealed left hemiparesis with muscle strength of 4/5 in her left arm and leg, left facial nerve palsy, and a Glasgow coma score of 15. Fundoscopy did not reveal any remarkable retinal changes, and other systemic examinations were mostly normal. These initial clinical findings suggested a likely ischemic right hemispheric stroke.

A non-contrast CT scan revealed multiple oval-shaped, hypodense lesions in both cerebral hemispheres, with the largest measuring 2.04×1.35 cm (right frontal lobe), 2.03×1.46 cm (left frontal lobe), and 1.77×1.60 cm (right parietal lobe), along with multiple small calcific densities in both hemispheres (Fig. 1). No evidence of infarction was observed. Neuroimaging findings were strongly indicative of NCC.

Her laboratory investigations were essentially normal, aside from an elevated erythrocyte sedimentation rate (Table 1). Serological tests for parasitic infections were not done due to the unavailability of test equipment in our facility.

The patient was treated with oral dexamethasone (4 mg twice daily) for 10 days, oral albendazole (400 mg twice daily) for 30 days, oral carbamazepine CR (200 mg twice daily), and oral folic acid (5 mg daily). The patient underwent physical therapy for limb weakness. At her 3-month follow-up visit, she showed remarkable improvement, with no seizures, headache or memory issues. She remains under follow-up care, and there has been a delay in scheduling another neuroimaging procedure because of her financial constraints.

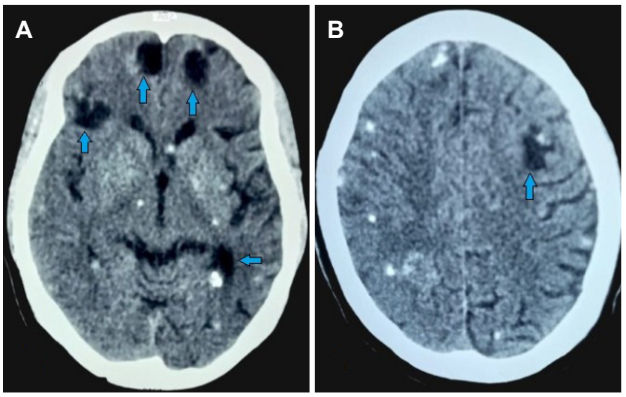

Figure 1. Axial section of a non-contrast brain CT scan showing: (A) multiple oval-shaped, hypodense lesions in both cerebral hemispheres, with the largest in the right frontal, right parietal, and left frontal lobes (blue arrows). Multiple small calcific densities are also present in both hemispheres. (B) Multiple small-sized calcific densities in both cerebral hemispheres and an oval-shaped, hypodense lesion (blue arrow). CT, computed tomography.

Discussion

NCC, a zoonotic disease, is a major concern for the WHO and is often overlooked worldwide as a medical diagnosis.[8] It is the primary parasitic brain infestation and a leading cause of preventable epilepsy in many low- and middle-income countries.[3,9] In developed countries, sporadic instances of NCC have been recorded, stemming from factors like travel, immigration, and person-to-person transmission.[10]

The life cycle of *T. solium* involves two hosts: humans and pigs. Human infestations can occur when tapeworm eggs are ingested through contaminated food or water.[2] Once ingested, the eggs hatch in the intestines, and the larvae migrate to various tissues, including the brain, muscles, and eyes, where they form cysticerci.[3,9] Invasion of the CNS by larvae results in NCC. Taeniasis, on the other hand, occurs from ingestion of raw or uncooked pork, resulting in adult tapeworms residing in the intestine with resultant symptoms.[9] The time between the initial infestation and the onset of symptoms can vary from several months to years.[2]

Table 1. Patient's laboratory test results

Investigation	Patient's value	Reference range
Hemoglobin (g/dL)	12.3	11-15.5
Hematocrit (%)	36.9	36.0-48.0
White blood cell count	8.7×10^9/L	$4\text{-}11\times10^9$/L
Neutrophils (%)	60.5	40.0-70.0
Lymphocytes (%)	38.6	20.0-40.0
Eosinophils (%)	0.8	1.0-4.0
Basophils (%)	0.1	0.5-1.0
Platelet count	309×10^9/L	$150\text{-}450\times10^9$/L
Erythrocyte sedimentation rate (mm/hour)	55	0-20
Random plasma glucose (mg/dL)	85	70-140
Urinalysis		
pH	6	4-8
Specific gravity	1.025	1.005-1.030
Serum creatinine (mg/dL)	1.1	0.7-1.4
Stool microscopy	No ova or cyst of parasite seen	
Aspartate aminotransaminase (IU/L)	12	5-36
Alanine aminotransaminase (IU/L)	18	7-55
HIV	Non-reactive	
Hepatitis B surface antigen	Non-reactive	
Anti-HCV	Non-reactive	
Mantoux test (mm)	0	<4

HIV, human immunodeficiency virus; HCV, hepatitis C virus.

The symptoms of NCC can vary significantly depending on the individual's immune system and the location of the parasites, whether in the parenchymal or extra-parenchymal regions of the brain.[7] In regions where the disease is widespread, it is often referred to as the "great imitator" because it can mimic various neurological conditions. Seizures and headaches are the most frequent symptoms when the parasites are located in the brain tissue.[7] The mechanism of epileptogenesis in NCC is still a subject of debate, but possible explanations include local inflammation and reactive gliotic scarring.[6] In endemic areas, late-onset seizures are highly suggestive of NCC, and approximately 80% of people with symptomatic NCC experience recurrent seizures.[11] Other possible manifestations of the disease include stroke, cognitive decline, neuropsychiatric disturbances, speech difficulties, extraocular palsy, movement disorders, and meningeal symptoms.[12] The patient in this report displayed symptoms such as stroke, seizures, prolonged headaches, and memory impairment at different intervals. Similar patterns of presentation have been previously documented in Africa.[13,14]

The diagnosis of NCC is challenging because its symptoms resemble other neurological conditions, and standard neuroimaging may struggle to detect cysticerci. However, recent advancements in imaging have improved the diagnosis of NCC. MRI is highly sensitive in detecting scolex and active noncalcified cysts, whereas CT scans excel in detecting calcified cysts.[7] Calcified lesions are the most common neuroimaging finding in NCC, and unfortunately, they are irreversible and remain a focus for epileptogenesis and neurological symptoms.[15] Serological tests can detect antibodies against *T. solium*, indicating current or past infection. Enzyme-linked immunosorbent assays and enzyme-linked immunoelectrotransfer blots are commonly used for serological testing.[9,12] Examining cerebrospinal fluid can also aid in diagnosis, with elevated antibody levels and increased leucocytes indicative of an active infection.[16] In this case, the history of epilepsy and recurrent headaches was suggestive of an underlying neurological condition. The delayed diagnosis was due to the lack of prior neuroimaging, which might have led to an earlier detection of the condition. However, the diagnosis was eventually confirmed with a CT scan during her current presentation.

The treatment of NCC requires a comprehensive approach that involves various medical, surgical, and community-based measures. To

eliminate parasitic cysts in the CNS, anthelmintic drugs, such as albendazole and praziquantel, are administered. Corticosteroids can also be used to reduce inflammation and manage symptoms. In severe cases, surgery may be required to remove the cysts.[9] The decision to use anthelminthic agents depends on the parasite's viability, which is determined through neuroimaging staging. For calcified lesions that continue to trigger seizures, lifelong treatment with antiepileptic drugs is necessary without the use of anthelminthic medications. Patients with viable lesions typically receive treatment with albendazole (15 mg/kg/day in two divided doses for 2 weeks) and/or praziquantel (50 mg/kg/day for 2 weeks).[7] Albendazole is preferred over praziquantel because of its superior penetration into the cerebrospinal fluid and fewer interactions with concurrent steroid and anticonvulsant use. Nonetheless, the impact of these interactions on the antiparasitic effects of praziquantel remains unclear.[17] Combining albendazole and praziquantel simultaneously for patients with multiple viable cysts in the brain parenchyma has been found to enhance the cysticidal effects without increasing drug-induced side effects.[18]

The use of steroids during the initiation of therapy mitigates the inflammatory response that occurs due to the death of the larvae, thus preventing the worsening of the neurological symptoms that usually occur between the second and fifth day of antiparasitic therapy.[17] This helps to control edema and intracranial hypertension that may occur during therapy when used with albendazole or praziquantel.[17] The optimal duration of treatment remains uncertain; however, studies have demonstrated the effectiveness of a 14-day therapy, which can be extended up to 30 days for extensive or extra-parenchymal disease.[9] In cases where hydrocephalus is present due to an intraventricular cyst, the recommended approach involves a ventricular shunt followed by surgical removal of the cyst and subsequent medical treatment.[19] The index patient showed improvement with a combination of dexamethasone, albendazole, and carbamazepine.

Prevention of NCC involves improving sanitation and hygiene practices to prevent contamination of food and water sources with tapeworm eggs. Public health measures such as proper disposal of human feces, adequate sewage systems, and education on personal hygiene are essential in reducing the incidence of this infestation.[2] Addressing *T. solium* and minimizing its impact requires public health interventions that involve the veterinary, human health, and environmental sectors. Prevention, early diagnosis, and prompt treatment are the key strategies for tackling this disease.

This case highlights the importance of timely neuroimaging in patients with neurological symptoms, especially those with a history of epilepsy and headaches. When evaluating patients with neurological deficits, especially in developing countries, it is essential to keep NCC in mind as a differential diagnosis, regardless of their existing neurological conditions. Early diagnosis and appropriate management are essential for preventing further complications and improving patients overall outcome. The fight against NCC requires a multidisciplinary approach, including public health measures and access to adequate healthcare, to reduce the burden of this devastating disease.

Conflict of Interest

The authors declare that they have no conflicts of interest.

References

1. Stelzle D, Abraham A, Kaminski M, et al. Clinical characteristics and management of neurocysticercosis patients: a retrospective assessment of case reports from Europe. *J Travel Med* 2023;30:taac102.
2. WHO. Taeniasis/cysticercosis [Internet]. Geneva: WHO, 2022 [cited 2023 Jun 13]. Available at : https://www.who.int/news-room/fact-sheets/detail/taeniasis-cysticercosis.
3. Owolabi LF, Adamu B, Jibo AM, Owolabi SD, Imam AI, Alhaji ID. Neurocysticercosis in people with epilepsy in Sub-Saharan Africa: a systematic review and meta-analysis of the prevalence and strength of association. *Seizure* 2020;76:1-11.
4. Ratcliffe C, Adan G, Marson A, et al. Neurocysticercosis-related seizures: imaging biomarkers. *Seizure* 2023;108:13-23.
5. de Oliveira Taveira M, Morita ME, Yasuda CL, et al. Neurocysticercotic calcifications and hippocampal sclerosis: a case-control study. *PLoS One* 2015;10:e0131180.
6. Steyn TJS, Awala AN, De Lange A, Raimondo JV. What causes seizures in neurocysticercosis? *Epilepsy Curr* 2022;23:105-12.
7. Garcia HH, Nash TE, Del Brutto OH. Clinical symptoms, diagnosis, and treatment of neurocysticercosis. *Lancet Neurol* 2014;13:1202-15.
8. Nash TE, Garcia HH. Diagnosis and treatment of neurocysticercosis. *Nat Rev Neurol* 2011;7:584-94.
9. Butala C, Brook TM, Majekodunmi AO, Welburn SC. Neurocysticercosis: current perspectives on diagnosis and management. *Front Vet Sci* 2021;8:615703.
10. Wallin MT, Kurtzke JF. Neurocysticercosis in the United States: review of an important emerging infection. *Neurology* 2004;63:1559-64.
11. Carabin H, Ndimubanzi PC, Budke CM, et al. Clinical manifestations associated with neurocysticercosis: a systematic review. *PLoS Negl Trop Dis* 2011;5:e1152.
12. Del Brutto OH. Human neurocysticercosis: an overview. *Pathogens* 2022;11:1212.
13. Mukuku O, Sánchez SS, Bugeme M, Garcia HH. Case report: three cases of neurocysticercosis in Central Africa. *Am J Trop Med Hyg* 2020;103:

1955-7.
14. Soyemi SS, Akinbo CO, Mgbehoma AI, Emiogun FE, Adegboyega OO, Obafunwa JO. Neurocysticercosis in a 14-year-old Nigerian: a case report and review of the literature. *Ann Trop Pathol* 2020;11:72-5.
15. Nash TE, Del Brutto OH, Butman JA, et al. Calcific neurocysticercosis and epileptogenesis. *Neurology* 2004;62:1934-8.
16. Rajshekhar V. Neurocysticercosis: diagnostic problems & current therapeutic strategies. *Indian J Med Res* 2016;144:319-26.
17. García HH, Evans CA, Nash TE, et al. Current consensus guidelines for treatment of neurocysticercosis. *Clin Microbiol Rev* 2002;15:747-56.
18. Garcia HH, Lescano AG, Gonzales I, et al. Cysticidal efficacy of combined treatment with praziquantel and albendazole for parenchymal brain cysticercosis. *Clin Infect Dis* 2016;62:1375-9.
19. Hamamoto Filho PT, Rodríguez-Rivas R, Fleury A. Neurocysticercosis: a review into treatment options, indications, and their efficacy. *Res Rep Trop Med* 2022;13:67-79.