Volume 4 | Issue 4 | 2024

ISSN | 2771-019X

Journal of
Clinical & Medical Images
Case Reports

Open Access | Case Report

# Neurocysticercosis - Diagnostic mystery

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## Abstract

Neurocysticercosis (NCC), a parasitic infection of the central nervous system caused by the larval stage of Taenia solium, presents a diagnostic conundrum due to its rare and often nonspecific clinical manifestations. This paper aims to unravel the diagnostic mystery surrounding NCC, shedding light on its epidemiology, pathophysiology, clinical presentation, and the challenges encountered in its diagnosis. Despite being considered a rare disease, NCC is the leading cause of acquired epilepsy worldwide, underscoring its clinical significance. The complexity of NCC diagnosis lies in its diverse clinical presentations, which can range from headaches, dizziness and seizures to more severe neurological cognitive deficits, often leading to misdiagnosis. Furthermore, the limitations of current diagnostic methods, including serological tests and neuroimaging, contribute to the diagnostic dilemma. This paper emphasizes the need for improved diagnostic criteria and novel diagnostic tools to enable early and accurate detection of NCC. By enhancing our understanding of NCC, we can pave the way for better management strategies, ultimately improving patient outcomes in this under-recognized disease.

**Received: Jul 15, 2024**

**Accepted: Aug 16, 2024**

**Published Online: Aug 23, 2024**



**Cite this article:** Hurła M, Pikor D, Kościelecka K, Drelichowska A, Dorszewska J, et al. Neurocysticercosis - Diagnostic mystery. J Clin Med Images Case Rep. 2024; 4(4): 1721.

**Keywords:** Neurocysticercosis; Central nervous system; Diagnostic challenges; Acquired epilepsy; Misdiagnosis; Rare disease.

## Introduction

Neurocysticercosis (NCC) is a parasitic infection of the central nervous system caused by the larval stage of Taenia solium [1]. This disease is considered the most frequent parasitic infection of the human brain [2] and is a leading cause of acquired epilepsy worldwide [3,4]. Despite its significant impact on global health, NCC remains a neglected tropical disease, predominantly affecting low- and middle-income countries [5]. Interestingly, NCC is not confined to these regions. It has been increasingly reported in high-income countries. Recent observations in our patients seem to highlight the rising prevalence of NCC in Poland. The rise of NCC in such regions can be attributed to increased global travel and immigration [6-10]. However, the true prevalence of NCC in Poland, as well as in other parts of Europe, remains unknown due to the lack of comprehensive epidemiological data. Moreover, there is a certain lack of acknowledgment regarding NCC, not only in Poland but also in other high-income European countries. This lack of recognition leads to NCC being currently treated as one of the other endemic diseases instead of being associated with its true origin. This misclassification can obscure the understanding of NCC's true epidemiology and hinder efforts to address the disease appropriately. Furthermore, the stigma associated with parasitic infections in general may contribute to underreporting and a

lack of focused public health initiatives. In taeniasis, a human disease, the life cycle of T. solium typically involves humans as definitive hosts and pigs as intermediate hosts. Adult T. solium residing in the human small intestine release eggs or egg-containing proglottids through stool into the environment [11]. Pigs become infected by ingesting human stool or water/vegetation contaminated with T. solium eggs. These eggs hatch, releasing larval oncospheres in the pig intestine [12]. Oncospheres penetrate the intestinal wall, enter the bloodstream, and develop into cysticerci. Humans become infected by ingesting raw or undercooked infected meat [13]. In cysticercosis, humans are infected by inadvertently ingesting food containing T. solium eggs. In the human intestine, eggs release larval oncospheres, which penetrate the intestinal wall, enter the bloodstream, and develop into cysticerci in various organs [14]. The complexity of NCC diagnosis lies in its diverse clinical presentations, which can range from headaches and seizures to more severe neurological deficits, often leading to misdiagnosis [15]. Furthermore, the limitations of current diagnostic methods, including serological tests and neuroimaging, contribute to the diagnostic dilemma [16]. This underscores the need for improved diagnostic criteria and novel diagnostic tools to enable early and accurate detection of NCC. The underdiagnosis and misdiagnosis of NCC have significant implications for its epidemiology. The current statistics, especially in Europe, may significantly underestimate the true prevalence of NCC. This underestimation could hinder the development of effective public health strategies and interventions to control and prevent NCC. Without accurate data, it is challenging to allocate resources effectively and design targeted interventions. The global health community must recognize the importance of addressing NCC with the same rigor applied to other neglected tropical diseases. Efforts to increase awareness and improve diagnostic capabilities are critical. Investment in public health education campaigns and training for healthcare providers in non-endemic regions could facilitate earlier identification and treatment of NCC, reducing the disease burden and improving patient outcomes. This paper aims to unravel the diagnostic mystery surrounding NCC, shedding light on its epidemiology, pathophysiology, clinical presentation, and the challenges encountered in its diagnosis. By enhancing our understanding of NCC, we can pave the way for better management strategies, ultimately improving patient outcomes in this under-recognized disease. Improved awareness and education among healthcare professionals about the diverse presentations of NCC can lead to earlier detection and treatment. Additionally, advancing research to develop more sensitive and specific diagnostic tools will play a critical role in mitigating the burden of NCC. Through these efforts, we can hope to reduce the morbidity and mortality associated with this parasitic infection, particularly in regions where it has been historically overlooked. Enhancing collaboration between endemic and non-endemic regions can foster the exchange of knowledge and strategies, promoting a more comprehensive approach to managing and controlling NCC globally.

## Clinical presentation

### Our patients

Among the five patients the presentation of symptoms was diverse and non-specific (Table 1).

In the first patient, the predominant symptoms were headache, dizziness, and balance disorders. MRI of the head with contrast revealed 53 diffuse cystic structures in both cerebral hemispheres with varying diameters of 2-6 mm, enhancing after intravenous administration of contrast. Areas of oedema and distortion of the left lateral ventricle were present around the lesions.

The second patient primarily presented with balance disorders, headaches, speech difficulties of aphasia type, asymmetry of gaze - left nystagmus, left lower limb dysmetria, and cerebellar syndrome. MRI of the head with contrast visualised multiple, diffuse, calcified lesions in the white matter of both cerebral hemispheres and cerebellum, ranging in size from 2-3 to 7 mm. There were twenty foci in the right cerebral hemisphere and sixteen in the left. Eight pathological foci were detected in both cerebellar hemispheres, with no features of oedema, gliosis, or enhancement after administration of contrast medium. The ventricular system was not displaced, but both lateral ventricles were discreetly wider supra-ventricularly, with a lumen up to 15 mm. There was a generalised, moderate degree of subarachnoid space dilatation.

The third patient was found to have epilepsy, positional tremor, and headaches. MRI of the head with contrast revealed partially calcified pathological foci at the cortical/subcortical border in the right cerebral hemisphere and the parietal, temporal, and parietal-temporal-occipital borderlands. There were also pathological foci in the left cerebral hemisphere, with some showing ring-like enhancement after administration of contrast medium. Additionally, an irregularly shaped zone of swelling was seen around a lesion in the marginal parts of the left temporal lobe.

The fourth patient presented with aphasia-type speech disorder and right-sided hemiparesis. MRI of the head with contrast showed a lesion within the white matter of the left frontal lobe, with fluid content and no enhancement after contrast medium administration. Surrounding the lesion, there was a small area of oedema without pathological enhancement. Single punctate areas of increased signal were observed subcortically within the right frontal lobe and in the insula region.

The fifth patient suffered from spinning dizziness and sensory disturbances in the left lower limb. MRI of the head with contrast revealed high signal foci in the high areas of the right parietal lobe and the posteroanterior parts of the right occipital lobe. Multiple diffuse vascular ischaemic foci were also observed bilaterally in the white matter of the frontal and parietal lobes, with no pathological enhancement after contrast medium administration.

### General population

Neurocysticercosis poses a diagnostic challenge due to its highly diverse clinical presentation. Population studies conducted in endemic regions of the disease indicate asymptomatic courses in the majority of infected individuals [17,18]. In the remaining cases, NCC presents a wide spectrum of symptoms, often resembling other neurological disorders. The clinical manifestations of neurocysticercosis are influenced by factors such as the patient's immune response, as well as the quantity, size, and location of T. solium cysts [19]. The characterization of clinical manifestations of neurocysticercosis appears to primarily rely on the localization of lesions within the brain parenchyma or in the extraparenchymal space [20]. In parenchymal neurocysticercosis, numerous small cysts (up to 2 cm in diameter) are observed within the brain parenchyma. Seizures are most commonly described as a manifestation of this form of the disease [21]. Headaches and dizziness are also frequently reported

**Table 1:** Symptoms and MRI findings.

| Patient ID | Symptoms | MRI Findings |
|---|---|---|
| 1 | Headache, dizziness, balance disorders | 53 diffuse cystic structures in both cerebral hemispheres (2-6 mm), Enhancing after intravenous administration of contrast, Areas of oedema, Distortion of the left lateral ventricle |
| 2 | Balance disorders, headaches, aphasia, left nystagmus, left lower limb dysmetria, cerebellar syndrome | Multiple, diffuse, calcified lesions in the white matter of both cerebral hemispheres and cerebellum (2-3 to 7 mm), 20 foci in the right cerebral hemisphere, 16 foci in the left cerebral hemisphere, 8 foci in both cerebellar hemispheres, No oedema, gliosis, or enhancement after contrast medium administration, Discreetly wider lateral ventricles supra-ventricularly (up to 15 mm), Generalised, moderate subarachnoid space dilatation |
| 3 | Epilepsy, positional tremor, headaches | Partially calcified pathological foci at the cortical/subcortical border in the right cerebral hemisphere and parietal, temporal, parietal-temporal-occipital borderlands, Pathological foci in the left cerebral hemisphere with some showing ring-like enhancement after contrast medium administration, Irregularly shaped zone of swelling around a lesion in the marginal parts of the left temporal lobe |
| 4 | Aphasia, right-sided hemiparesis | Lesion within the white matter of the left frontal lobe with fluid content and no enhancement after contrast medium administration, Small area of oedema surrounding the lesion without pathological enhancement, Single punctate areas of increased signal subcortically within the right frontal lobe and in the insula region |
| 5 | Spinning dizziness, sensory disturbances in the left lower limb | High signal foci in the high areas of the right parietal lobe and the posteroanterior parts of the right occipital lobe, Multiple diffuse vascular ischaemic foci bilaterally in the white matter of the frontal and parietal lobes, No pathological enhancement after contrast medium administration |

symptoms. In cases of larger cysts, focal symptoms such as muscle weakness, aphasia, or visual disturbances may occur [22]. Cognitive dysfunction [23] and psychiatric symptoms [24] are also part of the clinical presentation of parenchymal NCC. The presence of cysticerci in different regions of the brain parenchyma can lead to a variety of neurological deficits, depending on the specific areas affected by the cysts. These may include memory impairment, difficulties in executive function, and alterations in behavior. Furthermore, the inflammatory response to the cysts can exacerbate symptoms, leading to periods of exacerbation and remission, further complicating the clinical picture. Extraparenchymal neurocysticercosis primarily affects the brain ventricles, subarachnoid space, and meninges [25]. Cysts within the ventricles can obstruct cerebrospinal fluid outflow, leading to hydrocephalus and increased intracranial pressure. Larger cysts, especially in the lateral ventricular horns, can cause mass effect by displacing adjacent brain structures. When T. solium cysts are located in the subarachnoid space, they tend to form large aggregates, spreading into surrounding tissues and triggering an intense inflammatory response from the immune system. Subarachnoid NCC is characterized by increased intracranial pressure and arteritis [26], potentially leading to intracerebral hemorrhage or ischemic stroke. The chronic inflammation associated with this form of the disease can lead to fibrotic changes and persistent alterations in cerebrospinal fluid dynamics. Meningeal neurocysticercosis involves widespread inflammation of the meninges, leading to adhesions and the accumulation of cerebrospinal fluid, resulting in hydrocephalus [27]. Additionally, this form of the disease includes vasculitis and entrapment of cranial nerves within the inflammatory exudate, resulting in focal neurological symptoms. These symptoms can include cranial nerve palsies, sensory deficits, and motor weakness, depending on the nerves involved. It is important to note that neurocysticercosis does not have a single pathognomonic symptom, and it can manifest with nearly all neurological symptoms. The possibility of numerous cysts scattered throughout various areas of the central nervous system significantly complicates the characterization of a specific clinical picture for NCC. This diversity in clinical presentation necessitates a high index of suspicion and the use of comprehensive diagnostic modalities, including neuroimaging and serological tests, to accurately identify and assess the extent of the disease. The multifaceted nature of NCC underscores the need for individualized treatment approaches tailored to the specific manifestations and locations of the cysts in each patient.

### Current diagnostic methods

Diagnosis of neurocysticercosis can be challenging. It is essential to prioritize patient interviews and clinical symptom assessment. Histological confirmation is often not feasible, with neuroimaging methods, corroborated by serology, being paramount. To determine cyst localization, morphology, and stage, CT and MRI are optimal. However, defining the stage of involution can be challenging due to ongoing parasite degeneration. In imaging, live vesicular cysts manifest as small, rounded lesions devoid of contrast enhancement, often with minimal pericystic edema. Confirmation of parasite etiology is feasible when viable cysts containing visible scolices or tapeworm scolex (reminiscent of a hole-with-dot appearance) are identified. Conversely, identifying degenerative cysts may obviate the need for biopsies. These cysts, characterized by poorly defined borders, surrounded by edema, exhibit marked ring or nodular contrast enhancement [28]. The debate persists regarding the superior diagnostic modality, CT, or MRI. Diffusion-weighted MRI images are preferred for showcasing scolex in colloidal cysticerci, while CT excels at presenting calcified cysticerci, identifiable as non-enhancing hypersensitive nodules lacking peripheral edema. Conventional MRI may lack sensitivity, necessitating susceptibility-weighted image protocols [29,30]. Cysts may vary in size and location, from small, subarachnoid cysts in cortical sulci to larger ones in Sylvian fissures or the basal CSF. Subarachnoid neurocysticercosis may coincide with hydrocephalus, arachnoiditis, or mass effects, with approximately 60% extending to the spinal region [31]. Both CT and MRI adequately reveal arachnoiditis, evidenced by visible abnormal leptomeningeal enhancement at the brain's base [29,30,32,33]. Enhanced visibility of intraventricular and cisternal cysts is achievable with MRI protocols such as FLAIR (fluid attenuated inversion recovery), FIESTA (fast imaging employing steady state acquisition

sequence), CISS (constructive interference in steady state) or BFFE (balanced fast field echo), surpassing CT. In CT, their poor visibility due to isodensity with CSF may only allow differentiation of enlarged ventricles or hydrocephalus [30,34,35]. For imaging spinal cord and spinal subarachnoid space cysticercosis, MRI is superior, though vigilance is necessary to differentiate cysts from spinal tumors, which could prolong diagnosis [35-37]. Among the arsenal of immunological methodologies, the Enzyme-Linked Immunoelectrotransfer Blot (EITB) stands out as one of the most extensively validated. This assay is adept at detecting antibodies targeting T. solium, facilitated by lentil lectin purified glycoprotein antigens (LLGP). Positive EITB results are observed in patients harboring at least two viable cysts within the nervous system or those afflicted with subarachnoid disease, boasting a sensitivity of approximately 98% [38]. Nonetheless, in cohorts presenting with a singular intracranial cysticercus or calcified cysticerci, sensitivity markedly diminishes, fluctuating between 50-60% [39,40]. Furthermore, studies have elucidated a marginally reduced sensitivity in Cerebrospinal Fluid (CSF) compared to serum for antibody detection via EITB (90% vs. 100%) [39]. It is imperative to note that the presence of antibodies signifies a broader cysticercus infection spectrum, encompassing not only neurocysticercosis but also muscular or subcutaneous cysticercosis. In instances where access to EITB is restricted, ELISA serves as a viable alternative. The sensitivity of ELISA is estimated at 89%, with a specificity of 93% [41]. While CSF is preferred over serum for ELISA, its sensitivity for detecting subarachnoid neurocysticercosis parallels that of EITB. Analogous to EITB, ELISA exhibits enhanced reliability in patients harboring multiple viable cysts or those with cysts yet to undergo calcification [40]. Additionally, post-successful treatment or surgical intervention, there is a precipitous decline in serum antigen levels [42,43]. Consequently, ELISA warrants consideration as a therapeutic monitoring tool, particularly for gauging the response to antiparasitic interventions in individuals afflicted with subarachnoid neurocysticercosis [40,42,43]. The ancillary tests hold diminished significance in the diagnosis of neurocysticercosis. Certain patients exhibit eosinophilia in blood analyses, albeit at a modest level—typically peaking at around 10%. Concurrently, Cerebrospinal Fluid (CSF) manifests alterations. Predominantly, discussions revolve around mononuclear pleocytosis, with counts not surpassing 300 cells per mL, alongside diminished protein concentrations ranging between 50 mg/dL and 300 mg/dL. A subset of patients may present reduced glucose levels in CSF, indicative of a dismal prognosis. Nevertheless, overarching abnormalities in CSF predominantly afflict patients with active inflammation, multiple lesions, or the presence of parasites within the ventricular or subarachnoid compartments [44].

**Table 2:** Diagnostic methods.

| Diagnostic Method | Description | Advantages | Limitations |
|---|---|---|---|
| Neuroimaging (MRI) | Provides detailed images of cyst characteristics | Shows live and degenerating cysts | Difficulty in determining stage of degeneration |
| Serological Tests (EITB) | Detects antibodies against T. solium antigens | High sensitivity and specificity | May miss infection in cases with few cysts or calcified lesions |
| CSF Analysis | Evaluates cerebrospinal fluid for abnormalities | Supports diagnosis | Not always feasible to collect CSF samples |

### Diagnostic challenges

Confirming neurocysticercosis can be difficult due to limitations in directly testing for the parasite. Doctors rely heavily on neuroimaging and serological tests for diagnosis. While advancements have been made, these tests aren't perfect. Clinical signs and neuroimaging findings can be vague, and serological tests may miss the infection, particularly in areas where the disease is common. Neuroimaging is a key diagnostic tool. CT and MRI scans provide valuable information on cyst characteristics, location, number of cysts, stage of development, and surrounding inflammation. However, precisely determining the stage can be challenging because the parasite degenerates over time. Live cysts appear as small, round lesions with minimal swelling around them and no dye enhancement, sometimes revealing the worm's head inside. In contrast, degenerating cysts have poorly defined borders, more swelling, and a distinct ring or clump-like enhancement, which can make diagnosis unclear [28-30]. Our patients' MRI scans matched what's reported in the literature. The presentation is highly varied and involves multiple symptoms, making interpretation challenging. Some changes are small, localized, and show no dye enhancement with minimal swelling. On the other hand, we observed calcified cysts and various stages of degeneration with ring-like enhancement in some patients. Diffusion-weighted imaging and ADC maps might be helpful in visualizing the scolex in these colloidal cysts, improving diagnostic accuracy and differentiating stages of degeneration. Calcified lesions are easily seen on CT scans as non-enhancing, dense nodules, but standard MRI sequences may need improvement for better detection. Additionally, MRI excels at imaging cysts in the spinal cord or surrounding the brain, providing a clearer view of the lesions [28,29,32,33]. Serological testing, particularly the Enzyme-Linked Immunoelectrotransfer Blot (EITB) assay, is crucial for confirming neurocysticercosis. EITB is highly sensitive in patients with numerous live parasites, but it may miss the infection in those with fewer cysts or calcified lesions. Other serological tests and antigen detection methods can provide additional diagnostic value, although their accuracy varies [38-40]. This is consistent with our findings, where some patients with calcified cysts showed lower antibody levels. Interestingly, serological tests in patients with presentations more likely indicative of live cysts produced inconsistent results, deviating from what's typically reported in the literature. Other non-specific findings associated with neurocysticercosis include increased white blood cells of a particular type (monocytosis) in the bloodstream, which we observed in our patients, and abnormalities in the Cerebrospinal Fluid (CSF), which we were unfortunately unable to collect. Beyond immunological and neuroimaging assessments, thorough differential diagnosis is essential. Accurately distinguishing neurocysticercosis from other diseases, like tuberculosis, is critical, especially in areas where both are prevalent [45]. Established diagnostic criteria exist to aid in this differentiation, considering clinical features, radiological findings, immunological data, and epidemiological information. Neuroimaging remains indispensable in the diagnostic process for neurocysticercosis. Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) scans are utilized extensively due to their ability to reveal intricate details of cystic structures within the central nervous system. CT scans are particularly effective in detecting calcified cysts, presenting as hyperdense nodules that do not enhance with contrast. MRI, however, is superior for evaluating the detailed anatomical context and characteristics of cysts in both the brain and spinal cord. MRI sequences, including T1-weighted,

T2-weighted, and Fluid-Attenuated Inversion Recovery (FLAIR), provide comprehensive images that can differentiate between live and degenerating cysts, based on signal intensity and enhancement patterns. Diffusion-Weighted Imaging (DWI) and apparent diffusion coefficient (ADC) mapping further enhance diagnostic precision by delineating the scolex within colloidal cysts, a crucial feature for staging the infection [28,29,32,33]. Despite these advanced imaging techniques, the interpretation of results is complex. The polymorphic nature of neurocysticercosis presentations means that imaging findings must be correlated with clinical and epidemiological data. Live cysts are typically characterized by minimal perilesional edema and lack of contrast enhancement, while degenerating cysts exhibit increased edema, ring enhancement, and sometimes a more irregular appearance due to the host's inflammatory response. Our observations align with this, as patients presented with a spectrum of imaging findings, from non-enhancing nodules to extensively inflamed, ring-enhancing lesions [28-30]. Serological assays, particularly the EITB, serve as vital adjuncts to imaging. The EITB assay, known for its high sensitivity and specificity, detects antibodies against Taenia solium antigens, providing crucial evidence for neurocysticercosis. However, the assay's sensitivity diminishes in cases with low cyst burden or predominately calcified cysts, necessitating supplementary diagnostic approaches. Additional serological tests, including Enzyme-Linked Immunosorbent Assays (ELISA) and antigen detection methods, contribute further diagnostic insights, although variability in their accuracy persists. Our findings reflected these limitations, as serological results varied among patients with different cystic stages and burdens, underscoring the need for a multifaceted diagnostic strategy [38-40]. Monocytosis, indicative of a heightened immune response, and Cerebrospinal Fluid (CSF) abnormalities, such as pleocytosis or elevated protein levels, are supportive findings in neurocysticercosis. Unfortunately, CSF analysis was not feasible in our cohort, limiting our ability to correlate CSF parameters with other diagnostic indicators. The observed monocytosis in our patients aligns with the literature, suggesting an ongoing systemic inflammatory response to the parasitic infection. Differential diagnosis is paramount in endemic regions, where neurocysticercosis may mimic other neurological conditions, such as tuberculosis or malignancies. Established diagnostic criteria, incorporating clinical, radiological, immunological, and epidemiological data, are essential for accurate diagnosis and management. Comprehensive assessment using these criteria ensures that neurocysticercosis is distinguished from other conditions with similar presentations, facilitating appropriate therapeutic interventions [45].

## Conclusion and future prospect

Neurocysticercosis affects a significant number of individuals globally, particularly in developing nations, and represents a significant and often underdiagnosed issue in developed regions, resulting in limited literature available for the European Union populace. Confirmation of cases involving individuals presenting with a solitary cerebral lesion remains challenging, with antiparasitic therapy yielding suboptimal results and a scarcity of controlled data in various other domains, including approaches for modulating the inflammatory response to the degenerating parasite. In our study's described cases, manifestations such as headache, dizziness, and aphasia predominated, contrasting with the epileptic seizures documented in prior scientific literature. This discrepancy may be attributed to differing infection courses between developed and developing countries, environmental factors, or potential interspecies variations of the parasite across different geographical regions. T. solium exhibits immunosuppressive characteristics as a defensive mechanism against the host immune system, leading to the onset of intense epileptic seizures upon progressive lesion calcification and concurrent parasite demise, owing to an amplified inflammatory reaction. Given this, the administration of steroid medications and targeted antiparasitic therapy during hospital care is recommended to mitigate life-threatening epileptic seizures or focal symptoms, thereby enhancing survival prospects. Untreatable epileptic seizures, along with headaches and dizziness in patients, warrant consideration for a parasitic etiology, particularly in the presence of nonproliferative localized lesions in central nervous system tissue. Further investigation into this realm is imperative to address the growing issue of misdiagnosed patients, substantially enhancing their quality of life and health outcomes while facilitating accurate therapeutic interventions. The global burden of neurocysticercosis necessitates a deeper understanding and robust clinical approach to improve diagnosis and management strategies. In regions where the disease is endemic, the prevalence is markedly higher, underscoring the need for heightened awareness and advanced diagnostic methodologies. The limited literature for the European Union populace highlights a gap in comprehensive epidemiological data, suggesting an under-recognized prevalence and potential diagnostic oversight. This calls for an integrative approach combining public health surveillance, enhanced diagnostic tools, and interdisciplinary research to address the disease's hidden burden in developed countries. The challenge of confirming neurocysticercosis cases involving solitary cerebral lesions is compounded by the varied clinical presentations and the nonspecific nature of symptoms. Antiparasitic therapy, although a cornerstone of treatment, often yields suboptimal outcomes, emphasizing the need for individualized treatment regimens and the exploration of adjunct therapies. The scarcity of controlled data, particularly in modulating the inflammatory response during parasite degeneration, represents a critical gap in current clinical practice. Investigating immunomodulatory approaches, including the potential role of cytokine inhibitors or novel anti-inflammatory agents, could offer new therapeutic avenues and improve patient outcomes. In our study, the predominance of symptoms such as headache, dizziness, and aphasia contrasts sharply with the epileptic seizures more frequently reported in the literature. This variation may be reflective of differing pathogeneses, influenced by host immune responses, environmental factors, and possible genetic diversity of T. solium. The potential for interspecies variation of the parasite across geographical regions further complicates the clinical picture, suggesting a need for region-specific research to tailor diagnostic and therapeutic strategies effectively. The immunosuppressive properties of T. solium pose significant challenges in managing the disease, as the host's immune response to parasite degeneration can precipitate severe neurological complications. The resulting intense inflammatory reaction, particularly in cases of lesion calcification, underscores the critical role of anti-inflammatory therapies. The use of steroid medications in conjunction with targeted antiparasitic therapy aims to mitigate the inflammatory response, reduce seizure frequency, and alleviate focal neurological symptoms, thereby improving patient survival and quality of life. For patients presenting with untreatable epileptic seizures, headaches, and dizziness, especially in the context of nonproliferative localized CNS lesions, a parasitic etiology should be strongly considered. This necessitates a high index of suspicion and the use of advanced diagnostic imaging and serological testing to confirm the diagnosis. The

integration of novel diagnostic technologies, such as high-resolution MRI and next-generation serological assays, could enhance the accuracy and timeliness of neurocysticercosis diagnosis. The imperative for further investigation into neurocysticercosis is clear, aiming to address the growing issue of misdiagnosed patients. Enhanced research efforts focused on understanding the pathophysiology, improving diagnostic accuracy, and developing targeted therapies will substantially benefit patients. Such advancements are crucial for enhancing health outcomes and quality of life for individuals affected by this parasitic disease, ultimately facilitating more precise and effective therapeutic interventions. Addressing neurocysticercosis requires a multifaceted approach that incorporates both clinical and public health perspectives. Epidemiological studies are essential to understand the true burden of the disease, especially in regions where it is underreported or misdiagnosed. Collaborations between health organizations, governments, and research institutions can foster the development of comprehensive surveillance systems, ensuring timely detection and appropriate management of cases. From a clinical standpoint, the development of standardized diagnostic protocols can significantly improve the accuracy of neurocysticercosis diagnosis. These protocols should integrate neuroimaging techniques, serological assays, and clinical criteria to provide a holistic assessment of the patient. Training healthcare professionals to recognize the diverse presentations of the disease is equally important, particularly in non-endemic regions where familiarity with neurocysticercosis might be limited. Therapeutically, the emphasis should be on personalized medicine, tailoring treatment regimens to the individual patient's condition. This includes not only addressing the parasitic infection but also managing the host's inflammatory response. The role of adjunct therapies, such as anti-inflammatory agents, should be explored through clinical trials to establish their efficacy and safety in combination with antiparasitic treatments. Public health initiatives are crucial in reducing the incidence of neurocysticercosis. These initiatives should focus on education and awareness campaigns to inform communities about the transmission of T. solium and the importance of proper sanitation and hygiene practices. In endemic areas, interventions such as mass drug administration programs and the improvement of livestock management practices can help reduce the prevalence of the disease. Furthermore, research into the genetic diversity of T. solium and its various strains can provide insights into the differing clinical manifestations observed in different regions. Understanding the genetic and molecular mechanisms underlying the parasite's immunosuppressive strategies can also lead to the development of novel therapeutic targets. In conclusion, neurocysticercosis presents a complex and multifaceted challenge that requires a concerted effort from the global health community. By advancing our understanding of the disease through research, improving diagnostic and therapeutic protocols, and implementing effective public health strategies, we can significantly reduce the burden of neurocysticercosis and improve the quality of life for affected individuals.

**Funding:** This research received no external funding.

**Conflicts of interest:** The authors declare no conflicts of interest.

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