



AN INCIDENTAL FINDING OF GRANULAR NODULAR NEUROCYSTICERCOSIS ON BRAIN CT-SCAN: A CASE REPORT

Christian Hasudungan Nainggolan ^{1*}, Muhammad Irvanie Rama Harahap ¹, Yulia
Rachmawati ¹, Terawan Agus Putranto ¹

¹ Department of Radiology, Gatot Soebroto General Army Hospital, DKI Jakarta,
10410, Indonesia

*Corresponding Author :

E-mail: chyll_s@yahoo.com, muhammad.irvanie.harahap@upnvj.ac.id

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ABSTRACT

Introduction

Neurocysticercosis (NCC), the commonest central nervous system (CNS) parasitic infection of *Taenia solium* larva, stands as a significant yet preventable cause of severe neurological morbidity globally. Although computed tomography (CT) and MRI (magnetic resonance imaging) are reliable tool for its diagnosis, prevention and early diagnosis is essential to avoid long-term sequelae

Case Presentation

This report details the case of a 49-year-old woman with intermittent headache, several seizure episodes, and neck stiffness. A non-contrast and contrast-enhanced brain computed tomography (CT) scan incidentally revealed multiple, round, hypodense lesions

strategically located at the grey-white matter junctions within the frontotemporoparietal lobes and the cerebellum. These lesions, measuring between 3-7 mm, demonstrated characteristic post-contrast rim enhancement and were surrounded by perilesional edema, findings highly consistent with the granular nodular stage of NCC. The patient was then treated with antiepileptics, corticosteroids, and anthelmintic drugs.

Conclusions

This case highlights the role of neuroimaging in the incidental diagnosis of NCC and its diverse radiological spectrum, particularly in the granular nodular stage, which can pose diagnostic challenges if not carefully considered.

Keywords

Computed tomography scan, incidental finding, neurocysticercosis, taenia solium

INTRODUCTION

Introduction

Neurocysticercosis (NCC), caused by infestation of the metacestode larval stage of the pork tapeworm *Taenia solium*, is one of the most common parasitic infections of the human central nervous system (CNS) and a leading cause of acquired epilepsy worldwide [1, 2]. Its global burden is very substantial, particularly in developing regions such as Latin America, sub-Saharan Africa, parts of Asia, and Eastern Europe, where inadequate sanitation is widespread and free-roaming pig farming supports the parasite's life cycle. Although it is a hallmark of poverty, it is also becoming more prevalent in developed countries as a result of increasing migration [1].

Intestinal taeniasis is usually contracted due to consuming undercooked pork infected with *T. solium* cysticerci. The adult tapeworm then resides in the human intestine, producing gravid proglottids that release eggs into the environment through faeces. Meanwhile, neurocysticercosis occurs when humans, acting as accidental intermediate hosts, ingest these *T. solium* eggs via contaminated food or water (fecal-oral route) [2]. Once ingested, the eggs hatch in the intestine, releasing oncospheres that penetrate the intestinal wall, enter the bloodstream, and disseminate to various tissues, including the brain. Within the CNS, these oncospheres develop into cysticerci, fluid-filled structures containing an invaginated scolex. [2]

The clinical manifestations of NCC are very diverse, ranging from entirely asymptomatic infections (often discovered incidentally on neuroimaging performed for other symptoms, as what happened in this case) to severe, life-threatening neurological complications. This variability is primarily determined by the number, size, location, and the stage of viability and degeneration of the cysticerci. Moreover, it is also affected the inflammatory immune response of the host [3,4]. Epileptic seizure is its most frequent clinical presentation, affecting an estimated over half of symptomatic individuals and usually the main lead to the diagnosis of NCC. Other common manifestations include chronic headaches, focal neurological deficits, signs of increased intracranial pressure (such as nausea, vomiting, and papilledema), and even cognitive impairment [4].

The diagnosis of NCC relies on a combination of clinical evaluation, epidemiological history (living or travel history to endemic area), neuroimaging findings (CT and/or MRI), and, in rare cases, serological tests (as in enzyme-linked immunoelectrotransfer blot [EITB] or enzyme-linked immunosorbent assay [ELISA]) [5]. Neuroimaging is currently the paramount examination, not only for diagnosis but also for staging the disease and guiding therapeutic decisions. Examination using CT scan is particularly sensitive for detecting calcified lesions, which represent the end-stage of the degeneration, and often more accessible in resource-limited, endemic settings. Magnetic resonance imaging, on the other hand, offers superior soft tissue resolution and more sensitivity for identifying viable cysts (vesicular stage), cysts with surrounding edema (colloidal and granular nodular stages), and complications such as hydrocephalus or arachnoiditis [2,4].

The incidental discovery of NCC, as illustrated in this case report, highlights the importance of considering parasitic infection in the differential diagnosis of intracranial lesions, even in patients with non-specific or mild neurological symptoms, especially those with a potential history of exposure to endemic areas.

CASE REPORT

A 49-year-old woman presented to the outpatient clinic with a chief complaint of intermittent headaches over the last three months. The headaches were described as dull and diffuse, with visual analog score of 5-6/10, with no specific aggravating or relieving factors. She reported two generalized tonic-clonic seizure episodes during this period, each lasting about 2-3 minutes, followed by post-ictal confusion phase. There was no reported aura prior to the seizures, incontinence, or tongue biting. The patient also complained of mild neck stiffness, especially in the morning. She denied any history of fever, recent weight loss, visual disturbances, or motoric weakness. There was no significant past medical history, and she was not on any regular medications. Her family history was unremarkable, with no known history of tuberculosis, malignancy, or seizure disorders in first-degree relatives. She resided in a suburban area of

Indonesia, a known endemic region for *Taenia solium* infection, but had no specific recollection of consuming undercooked pork or direct contact with pigs.

On general physical examination, the patient was fully alert. Her vital signs were within normal limits. A thorough neurological examination revealed no focal motor or sensory deficits. Her cranial nerve functions were intact. Meningeal signs were equivocal on initial examination. Fundoscopy was normal, with no evidence of papilledema. Her gait and coordination were normal. Routine laboratory investigations were performed, including a complete blood count, erythrocyte sedimentation rate, liver function tests, renal function tests, fasting blood glucose, and a chest X-ray. All these parameters were within normal limits. Given her presentation with headaches and seizures, a computed tomography scan, with and without contrast, of the brain was performed.

The non-contrast CT-cscan images of the brain revealed multiple, well-defined, round, hypodense lesions scattered throughout the cerebral parenchyma, predominantly at the grey-white matter junctions in the bilateral frontotemporoparietal lobes. Similar lesions were also noted in the cerebellar hemispheres with varying size. Several of these lesions exhibited central hyperdensity suggestive of calcification. Following intravenous administration of iodinated contrast media, the scan demonstrated discrete, smooth, ring-like peripheral enhancement of many of these hypodense lesions. Some of the enhancing lesions were surrounded by a halo of hypodensity representing edema. There was no significant midline shift or evidence of obstructive hydrocephalus. The overall imaging appearance was highly suggestive of the granular nodular stage of neurocysticercosis (Figure 1).

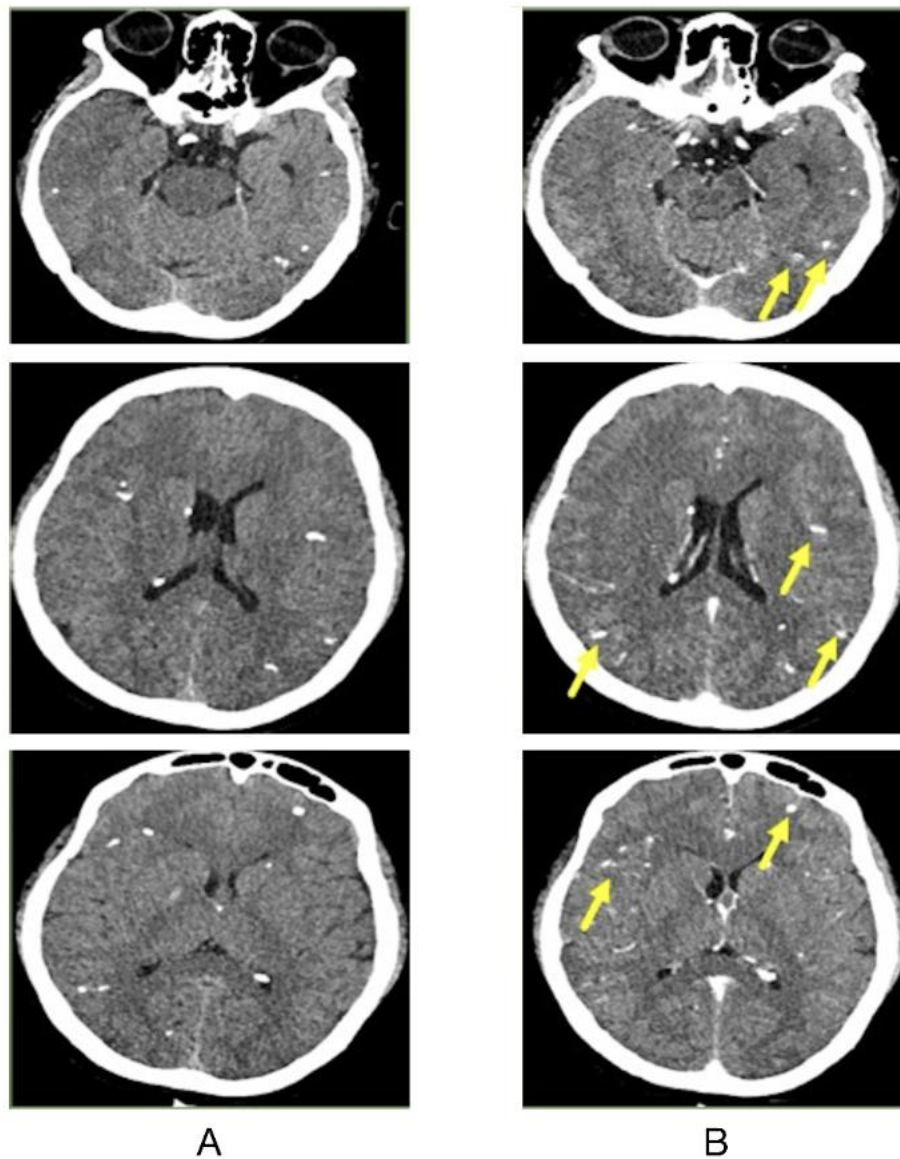


Figure 1. Axial Brain CT-scan (A) pre-contrast and (B) post-contrast showing multiple heterogeneous round hypodense lesion at the grey-white matter junction in the *frontotemporoparietal* lobe and cerebellum with post-contrast enhancement accompanied by surrounding edema consistent with granular nodular *Neurocysticercosis*

Based on the neuroimaging findings, a diagnosis of granular nodular neurocysticercosis was made. The patient was initiated on antiepileptic therapy) to prevent further seizure episodes. She was also started on oral corticosteroids to reduce the perilesional edema and inflammatory response. A decision was made to closely monitor her clinical response to steroids and antiepileptics before considering antiparasitic drugs, to avoid potential exacerbation of inflammation. The patient was followed up for four weeks following the therapy, with no recurrent episodes of seizures.

DISCUSSION

Neurocysticercosis (NCC) remains a significant public health challenge and a leading cause of acquired epilepsy in many parts of the world, especially in the areas with poor sanitation and water contamination [1,2,5]. The case presented in our study exemplifies the classic, although incidentally discovered, radiological features of the granular nodular stage of parenchymal NCC, highlighting the diagnostic utility of neuroimaging, especially in endemic regions. The initial symptoms of intermittent headaches and seizures, coupled with the CT findings of multiple small, ring-enhancing lesions with perilesional edema and calcifications at the grey-white matter junctions, are the main diagnostic characteristics of this disease [3,4]. The grey-white matter junction is a predilection site for hematogenous dissemination of the oncospheres, explaining the common location of the cysticerci in this region. [2,6]

Brain CT scanning plays a pivotal role in the diagnosis of NCC, particularly in developing countries where it is often more accessible and affordable than a MRI examination. Its high sensitivity for detecting calcifications makes it the modality of choice for both identifying the nodular calcified stage and for assessing the overall burden of the disease, which often correlates with its clinical characteristics [3,7]. The radiological appearance, characterized by multiple calcifications scattered throughout the parenchyma, is a classic, though not pathognomonic, CT sign of chronic NCC. However, in order to visualizing its vesicular stage and especially detecting extraparenchymal NCC (e.g., intraventricular or subarachnoid cysts), MRI is superior due to its excellent soft tissue contrast and multiplanar capabilities [4]. MRI sequences like FLAIR (Fluid-

Attenuated Inversion Recovery) are particularly sensitive for detecting perilesional edema, and T2-weighted images can clearly delineate the cyst wall and scolex. Diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) maps may also help differentiate NCC cysts from other cystic lesions. In this case, the CT scan obtained was sufficient for diagnosis.

The clinical presentation of NCC is highly variable. Seizures are indeed the most common symptom, occurring in up to 70-90% of patients with parenchymal NCC, and are often the initial manifestation [1-3]. The pathogenesis of seizures in NCC is multifactorial, involving direct mechanical irritation by the cyst, perilesional gliosis, and also the inflammatory response toward the parasites. Headaches are also frequent and may be caused by increased intracranial pressure from mass effect or edema. Focal neurological deficits may occur depending on the location of the lesions. Moreover, intraventricular NCC can cause obstructive hydrocephalus, leading to symptoms of increased intracranial pressure [2,4,6].

The management of NCC is complex and must be individualized. The mainstays of treatment include anti-inflammatory agents (corticosteroids), antiepileptic drugs (AEDs), and sometimes antiparasitic drugs (albendazole or praziquantel) to surgical intervention. Antiparasitic therapy aims to kill the viable cysts, but their use, particularly in the colloidal and granular nodular stages, can paradoxically worsen inflammation and edema as the parasites degenerate, potentially exacerbating neurological symptoms. Therefore, corticosteroids (e.g., dexamethasone) should also be administered concurrently with antiparasitic drugs to modulate this inflammatory response [1,7]. In cases of solitary cysts or minimal symptoms, a conservative approach with AEDs alone may be considered. AEDs are crucial for controlling seizures and are often continued for a variable period, sometimes long-term, especially if there is a significant burden of calcified lesions or recurrent seizures. In rare cases, surgical intervention may be necessary for large cysts causing mass effect, intraventricular cysts causing hydrocephalus (often requiring endoscopic removal or shunt placement), or cysts refractory to medical therapy [7].

This case highlights the need for increased awareness of NCC in endemic areas or individuals with relevant travel histories, even when clinical presentations are non-specific. The presence of intracranial calcifications, should always raise the suspicion of NCC. Distinguishing

NCC from other causes of ring-enhancing brain lesions, such as tuberculosis, brain abscesses, metastatic tumors, or toxoplasmosis (especially in immunocompromised individuals), is also crucial. The long-term prognosis of NCC varies. Many patients with parenchymal NCC respond well to medical therapy and experience resolution of symptoms and radiological findings. However, some patients, particularly those with calcified lesions, may develop chronic epilepsy requiring long-term AED therapy [18]. Recurrence of seizures can occur even years after successful antiparasitic treatment, often triggered by the persistence of calcified nodules. Therefore, regular follow-up, both clinical and radiological, is essential to monitor the treatment response and manage its complications.

CONCLUSION

This case report illustrates the incidental discovery of a NCC case, a significant global health issue and a leading cause of preventable epilepsy. Its clinical and radiological manifestations are diverse, thus a high index of suspicion for NCC is essential, even in patients with non-specific neurological symptoms, to ensure timely diagnosis and appropriate management, reducing the long-term neurological sequelae associated with this parasitic infection. Continued efforts towards prevention and early treatment are paramount to lessen the global burden of neurocysticercosis.

DECLARATIONS

Consent

Written informed consent for the publication of the data was obtained from the patients

Data availability

No data associated with this article

Underlying data

All data underlying the results are available as part of the article and no additional source data are required

Competing interests

None to be disclosed

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