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Case Report

Histopathologically Compatible Neurocysticercosis Mimicking an Intracranial Tumor in a Child

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Abstract

Neurocysticercosis is the most common parasitic infection of the central nervous system and may mimic neoplastic, infectious, or granulomatous lesions, particularly in non-endemic settings. Diagnosis can be challenging when characteristic radiological findings are absent. An 11-year-old boy presented with persistent headache and dizziness. Brain magnetic resonance imaging (MRI) demonstrated a 6 × 8 mm right temporal lesion with ring-like peripheral enhancement and marked surrounding vasogenic edema. The lesion appeared hypointense on T1-weighted sequences and hyperintense on T2-weighted and FLAIR images. The differential diagnosis included neoplasm, bacterial abscess, granulomatous infection, and parasitic disease. Because the lesion could not be reliably characterized noninvasively and an intracranial tumor could not be excluded, neurosurgical excision was performed. Histopathological examination demonstrated parasitic structures morphologically compatible with cysticercosis, including PAS-positive tegumental components and sucker-like structures suggestive of a cestode larva. Following the pathological diagnosis, a detailed epidemiological history revealed chronic intermittent gastrointestinal complaints and a history of antiparasitic treatment among siblings. The patient received albendazole therapy, and follow-up neuroimaging demonstrated regression of both the lesion and surrounding edema. This case highlights the importance of considering histopathologically suspected neurocysticercosis in the differential diagnosis of solitary enhancing brain lesions in children, even in regions where the disease is considered uncommon. Histopathological evaluation may be required when neuroimaging findings are non-specific and malignancy cannot be excluded.

Introduction

Neurocysticercosis (NCC), caused by the larval stage of Taenia solium, is the most common parasitic infection of the central nervous system and remains a major cause of acquired epilepsy worldwide (1,2). Although the disease is highly prevalent in endemic regions of Latin America, sub-Saharan Africa, and parts of Asia, increasing global travel and migration have resulted in cases being reported in non-endemic countries as well (2-4).

Humans acquire cysticercosis through ingestion of T. solium eggs shed in the feces of a human tapeworm carrier. This route of transmission differs from intestinal taeniasis, which develops after ingestion of undercooked pork containing cysticerci (1,4,5). Consequently, neurocysticercosis may occur even in populations where pork consumption is uncommon, making clinical suspicion particularly challenging (6,7).

The clinical manifestations of NCC are highly variable and depend on the number, location, developmental stage, and host inflammatory response to the lesions (2,6,7). Solitary parenchymal lesions are particularly difficult to diagnose because they may radiologically mimic primary brain tumors, tuberculomas, pyogenic abscesses, fungal infections, and other granulomatous conditions (6-9). In such cases, characteristic neuroimaging findings may be absent, and definitive diagnosis may require histopathological evaluation (3).

We report an 11-year-old child presenting with a solitary enhancing temporal lesion initially suspected to represent an intracranial tumor. Histopathological examination following surgical excision demonstrated findings compatible with histopathologically suspected neurocysticercosis, highlighting the diagnostic challenges posed by this uncommon presentation in a non-endemic setting.

Case Report

An 11-year-old boy from Istanbul, Türkiye, was referred to our tertiary care center in 2021 with persistent headache and dizziness after a brain magnetic resonance imaging (MRI) performed at another hospital revealed an intracranial lesion. On admission, he was afebrile and in good general condition. Physical and neurological examinations were unremarkable, with no focal deficits, meningeal signs, or cranial nerve abnormalities. Routine laboratory tests, including complete blood count, C-reactive protein, and erythrocyte sedimentation rate, were within normal limits.

Contrast-enhanced MRI demonstrated a 6 × 8 mm lesion in the right temporal lobe showing ring-like peripheral enhancement with marked surrounding vasogenic edema (Fig. 1). The lesion appeared hypointense on T1-weighted sequences and hyperintense on T2-weighted and FLAIR images. Diffusion restriction was not a prominent feature. Because of the significant perilesional edema and absence of pathognomonic imaging findings, differentiation from neoplastic, inflammatory, infectious, and granulomatous lesions was not possible based solely on neuroimaging. Serological tests for human immunodeficiency virus, toxoplasmosis, echinococcosis, and brucellosis were also negative. Stool examinations for Entamoeba histolytica and Giardia antigens were negative.

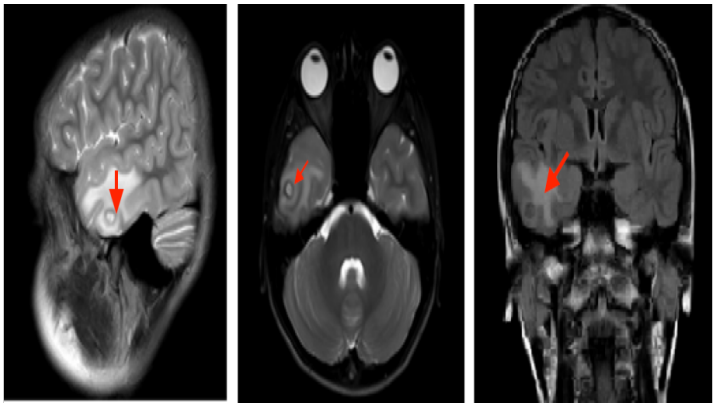

Fig. 1: Contrast-enhanced MRI showing a small right temporal ring-enhancing lesion with marked surrounding vasogenic edema

Because imaging did not reliably exclude a tumoral lesion and the surrounding edema was substantial, neurosurgical excision was performed. Histopathological examination revealed parasitic structures morphologically compatible with cysticercal larval tissue, including PAS-positive membranous tegumental components and sucker-like structures suggestive of a cestode larva. Ehrlich-Ziehl-Neelsen staining was negative for acid-fast bacilli. Serological testing for Taenia solium was not available at our institution. Following the histopathological findings compatible with cysticercosis, the epidemiological history was reviewed in greater detail. The patient had no history of foreign travel. The family reported intermittent abdominal pain and diarrhea since childhood, and several siblings had previously received antiparasitic treatment because of intestinal parasitic infection, suggesting possible household exposure to enteric parasites, although Taenia infection could not be confirmed. Stool examination for Taenia species was not available at the time of diagnosis, representing a limitation of the investigation. The patient received albendazole therapy (15 mg/kg/day in two divided doses) for 28 d. Follow-up MRI demonstrated marked regression of both the lesion and surrounding edema. The patient remained neurologically stable and asymptomatic during follow-up.

Discussion

This case illustrates the diagnostic difficulty of solitary parenchymal neurocysticercosis in a child presenting with nonspecific symptoms and imaging features suggestive of an intracranial mass. The lesion was initially considered within a broad differential diagnosis that included neoplastic, infectious, and granulomatous causes, and the histopathological findings strongly supported neurocysticercosis, making it the most likely diagnosis. This pattern has been described in colloidal vesicular neurocysticercosis, in which ring enhancement and disproportionate perilesional edema may strongly resemble a brain tumor or abscess (2,6,7).

According to the revised Del Brutto diagnostic criteria, histological demonstration of the parasite from biopsy material has been considered an absolute diagnostic criterion for neurocysticercosis (3). However, in our case, additional serological or molecular confirmation was unavailable. Therefore, although the histopathological findings together with the clinical and radiological features strongly support neurocysticercosis, the diagnosis should be interpreted with caution. Solitary ring-enhancing lesions in children represent a broad differential diagnosis including neurocysticercosis, tuberculoma, pyogenic abscess, fungal infections, demyelinating lesions, low-grade gliomas, and other primary central nervous system tumors (6,7,9). In non-endemic settings, neurocysticercosis may be overlooked because clinicians frequently prioritize neoplastic or inflammatory conditions. Consequently, tissue diagnosis may occasionally be required when characteristic imaging findings are absent.

Neurocysticercosis evolves through vesicular, colloidal vesicular, granular nodular, and calcified stages, each associated with distinct radiological appearances (2,6,7). The vesicular stage typically demonstrates a viable cyst with minimal inflammatory response, whereas the colloidal vesicular stage is characterized by ring enhancement and substantial surrounding edema. The prominent vasogenic edema and ring enhancement observed in our patient are most consistent with the colloidal vesicular stage, during which host inflammatory responses become more pronounced.

The present case occurred in Türkiye, where neurocysticercosis is considered uncommon and no nationwide surveillance data are available. Although sporadic cases have been reported, neurocysticercosis is not regarded as an endemic disease, and taeniasis is believed to occur at a relatively low frequency. Consequ-

ently, clinicians may have a low index of suspicion, particularly in children without a history of travel to endemic regions. However, humans acquire cysticercosis through ingestion of T. solium eggs shed by a human tapeworm carrier. This mechanism differs from intestinal taeniasis, which develops following consumption of undercooked pork containing cysticerci. Therefore, transmission may occur through contaminated food, water, or close household contact rather than direct pork consumption (1,4,5). In our patient, the history of chronic gastrointestinal complaints and antiparasitic treatment among siblings only gained significance after the pathological diagnosis and provided a plausible background for infection. Unfortunately, stool examination for Taenia species was not available, preventing further epidemiological investigation.

Current treatment recommendations for solitary parenchymal neurocysticercosis include albendazole-based therapy with or without adjunctive corticosteroids depending on lesion burden, location, and degree of inflammatory response (1,10). Surgical intervention is generally reserved for diagnostically uncertain lesions, lesions causing significant mass effect, or cases in which neoplasia cannot be confidently excluded. Although many patients can be managed medically once the diagnosis is established, surgery may still be unavoidable when a solitary enhancing lesion cannot be reliably distinguished from a tumor.

The patient improved after surgery and albendazole therapy. Nevertheless, radiological improvement following antiparasitic treatment should not be regarded as confirmatory evidence of neurocysticercosis because lesion regression may reflect reduced inflammation or the natural evolution of the lesion (10). In the present case, treatment response was considered supportive rather than diagnostic evidence.

Our case underlines the importance of considering neurocysticercosis in the differential diagnosis of enhancing brain lesions in children, even in apparently non-endemic settings. Increased awareness of this entity may facilitate earlier diagnosis and prevent unnecessary diagnostic delays.

Limitations

The absence of serological or molecular confirmation, lack of stool examination for Taenia species, and the lack of histopathological photomicrographs constitute important limitations of this report. Although histopathological findings strongly supported neurocysticercosis, these limitations should be considered when interpreting the diagnosis. Future reports of similar cases should incorporate complementary serological and molecular techniques, including PCR-based assays and sequencing where feasible, to further strengthen diagnostic confirmation.

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None.

Ethical considerations

Written informed consent for publication was obtained from the patient's parents or legal guardians. Ethics committee approval was not required for a single anonymized case report at our institution.

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Conflict of interest

The authors declare no conflict of interest.

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