Neurocysticercosis Presenting with Seizures: Diagnostic Value of the Dot Sign in Non-Endemic Areas

Neurocisticercose e Crises Epiléticas: O Valor Diagnóstico do Dot Sign em Áreas Não Endémicas

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Palavras-chave: Convulsões/etiologia; Neurocisticercose/complicações; Neurocisticercose/diagnóstico; Neurocisticercose/diagnóstico por imagem

A 26-year-old woman presented with new-onset tonic-clonic seizures and left-sided monoparesis. The patient was originally from Cape Verde and had been living in Portugal for six months, with access to drinking water and adequate hygiene conditions, no relevant past medical history, and no known similar cases among close contacts.

Brain computed tomography (CT) revealed a poorly defined right frontal cortico-subcortical cystic lesion measuring approximately 12 x 7 mm on the axial plane, with a millimetric central hyperdense focus and surrounding hypodensity.

Magnetic resonance imaging (MRI) (Fig. 1) revealed a solitary cystic lesion exhibiting a central 'dot sign' on T2 FLAIR, accompanied by peripheral enhancement and surrounding vasogenic edema – findings consistent with parenchymal neurocysticercosis (NCC) in the colloidal stage.[1]

Cerebrospinal fluid analysis showed mild pleocytosis with polymorphonuclear cell predominance; glucose and protein levels were normal. Microbiological tests and *Taenia solium* serology were negative. The MRI excluded intracranial extraparenchymal involvement, skeletal radiographs showed no evidence of muscular cysticercosis and cardiac involvement was excluded by cardiac MRI.

Combined albendazole-praziquantel therapy was initiated on admission and praziquantel was discontinued on day seven after exclusion of disseminated disease.[2] Dexamethasone and levetiracetam were also administered, with complete clinical stability and no seizure recurrence. Follow-up at three months demonstrated evolution to a calcified nodule, supporting the diagnosis.

Neurocysticercosis, caused by the larval stage of *Taenia solium*, is a leading cause of acquired epilepsy in endemic regions.[3] Most parenchymal cases are asymptomatic, with clinical manifestations largely determined by the stage of cyst evolution – vesicular, colloidal, granular nodular, or calcified.[4-7]

Among symptomatic patients, seizures are the most frequent presentation, occurring in 70% - 90% of cases.[8] The colloidal stage is especially symptomatic, as increased cyst-wall permeability elicits a strong inflammatory response, resulting in perilesional oedema, peak immune activation, and symptoms such as seizures and headache.[3-7]

Neuroimaging is central to diagnosis, with MRI optimal for detecting viable cysts and visualizing the scolex, which is the viable larval structure within the cyst and constitutes

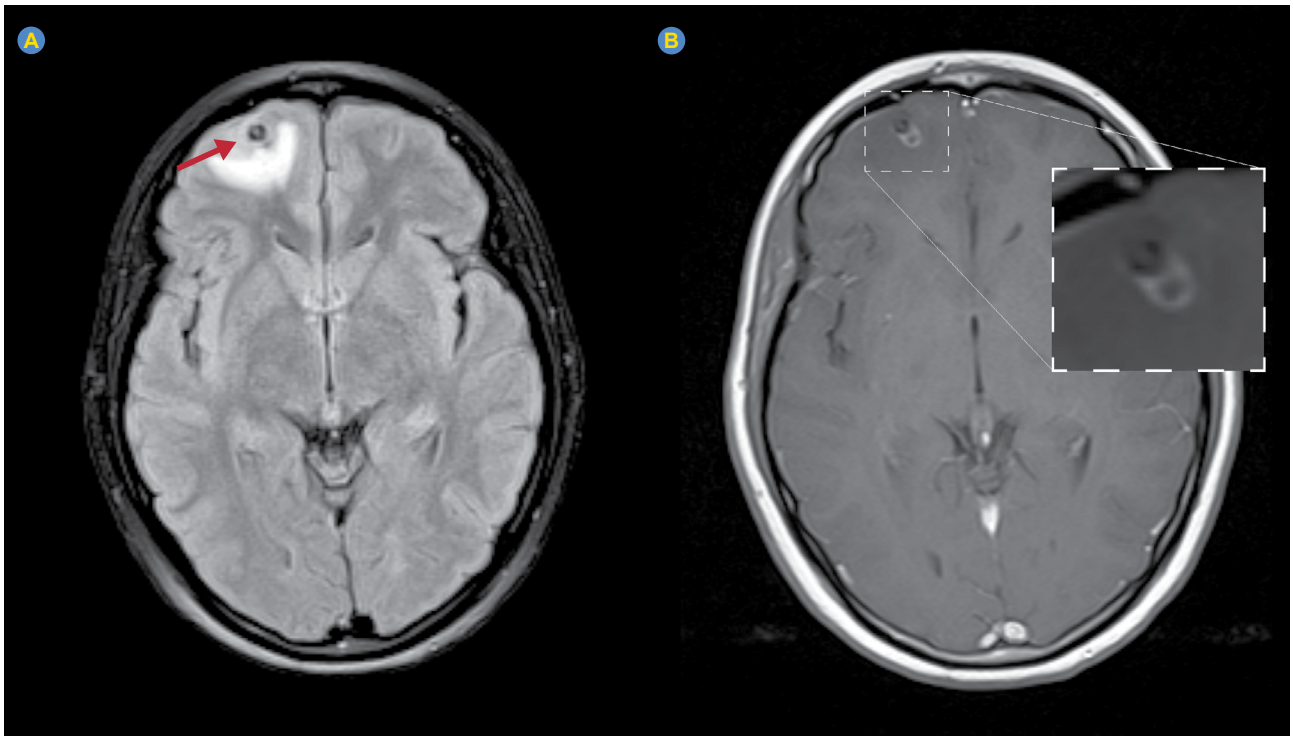

Figure 1 – (A) Cerebral MRI demonstrates a cystic lesion in the right frontal cortico-subcortical region, with a central 'dot sign' on T2-FLAIR (red arrow) representing the scolex and eliciting surrounding vasogenic oedema. No hemorrhage was evident on T1-weighted or T2* sequences (not shown). Post-contrast T1-weighted images (B) clearly depict peripheral wall enhancement of the cyst.
MRI: magnetic resonance imaging; FLAIR: fluid-attenuated inversion recovery

the hallmark imaging feature of neurocysticercosis. Its signal and density characteristics produce the classic 'hole-with-dot' appearance – best appreciated on MRI – widely regarded as pathognomonic of viable parenchymal cysticerci.[5,9]

On the other hand, CT is more sensitive for identifying calcifications, particularly in later disease stages.[5-8]

While NCC is endemic in low- and middle-income countries,[2] its prevalence is increasing in developed regions due to migration from endemic areas.[10] The unequivocal identification of the scolex on MRI enabled a confident diagnosis.

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AUTHOR CONTRIBUTIONS

All authors contributed equally to this manuscript and approved the final version to be published.

PROTECTION OF HUMANS AND ANIMALS

The authors declare that the procedures were followed according to the regulations established by the Clinical Research and Ethics Committee and to the Helsinki Declaration of the World Medical Association updated in October 2024.

DATA CONFIDENTIALITY

The authors declare having followed the protocols in use at their working center regarding patients' data publication.

PATIENT CONSENT

Obtained.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

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