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Neuroimaging findings from a case of neurocysticercosis. Case report

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ABSTRACT

Introduction: Human cysticercosis occurs when humans ingest *Taenia solium* eggs from the fecal-oral route. The infection in the central nervous system is called neurocysticercosis and is considered the principal cause of late-onset epilepsy in endemic countries in Latin America, Asia, and Africa.

Case report: 71-year-old female in a state of neglect with a medical history of untreated rheumatoid arthritis. She presented her first seizure episode. Cerebral magnetic resonance imaging with contrast reported parenchymal and subarachnoid neurocysticercosis in different stages.

Discussion: The high incidence of neurocysticercosis in endemic countries is associated with poverty conditions such as less hygienic conditions and permanent contact with domestic animals. The four stages of classification of neurocysticercosis is useful for pathologists and radiologists to identify the life cycle of the parasite within the body and the specific imaging findings of each phase. After diagnosis, treatment includes the use of niclosamide or praziquantel.

Conclusions: neurocysticercosis is a public health problem in endemic countries that requires further attention. Clinical manifestations are variable and neuroimaging findings are essential to making a correct diagnosis.

Keywords
Neurocysticercosis, Seizures, magnetic resonance imaging, treatment

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INTRODUCTION

Human cysticercosis occurs when humans ingest Taenia solium eggs from the fecal-oral route, harbouring the parasite in the intestine. Later, the eggs evolve into oncospheres, which are carried to other tissues, including the central nervous system (CNS) and meninges, where they evolve into larval forms, or cysticerci. (1) The infection in the CNS is called neurocysticercosis (NCC) (1) and is considered the principal cause of late-onset epilepsy in endemic countries in Latin America, Asia, and Africa. (2,3)

Clinical manifestations depend on the number of lesions, size, stage, and location; some of the most common include seizures in up to 70–90% of patients with NCC, hydrocephalus, which can cause headaches, vertigo, loss of consciousness, and an increase in intracranial hypertension due to CSF obstruction. (2) Less often, symptoms include cognitive and psychiatric impairment. (4) Neuroimaging is the

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cornerstone of diagnosis; in addition, a CSF test would be useful to detect even antibodies/antigen to Taenia solium. (2) Magnetic resonance imaging (MRI) is especially useful to identify four of the four types classified by Escobar of NCC: the vesicular, colloidal vesicular, granular nodular, and calcified nodular stages, with the non-cystic stage detectable only by laboratory tests. (5, 6) We present the case of a 71-year-old female with findings compatible with NCC.

CASE REPORT

A 71-year-old female in a state of neglect with a medical history of untreated rheumatoid arthritis presented to the emergency room with a tonic-clonic seizure with sphincter relaxation of 20 minutes duration. She was treated in a first-level hospital with diazepam and phenytoin with modulation of the crisis and was subsequently transferred to a hospital of greater complexity.

On presentation, she was awake, disoriented in time, with elevated blood pressure (BP: 160/95 mmHg), and all other vital signs were normal (heart rate: 75 bpm, breath rate: 18 bpm, temperature: 36.9 °C, oxygen saturation: 98%). Laboratories reported a complete blood count with anemia (hemoglobin 11.6 g/dL, NR: 13.2 to 16.6 g/dL, hematocrit: 36%, NR: 38.3% to 48.6%), normal leukocytes (7.700 mm3, NR: 4.000–10.000 mm3), platelets in lower limit (156.000 mm3, NR: 150.000–400.000 mm3), antibody to human immunodeficiency virus negative, and syphilis antibodies non-reactive.

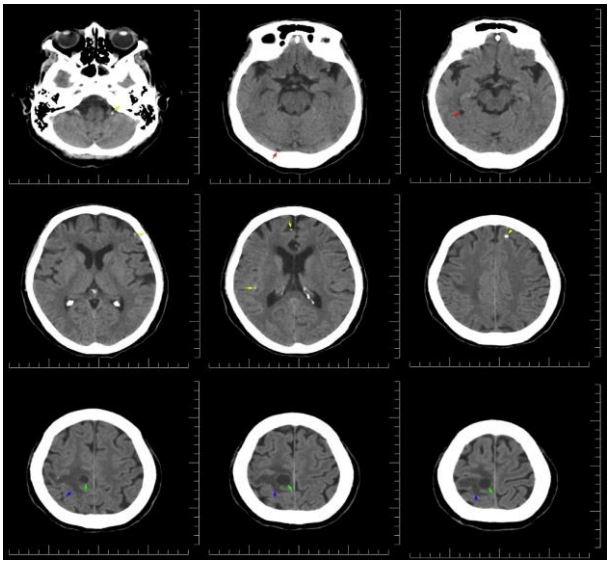

Figure 1. Cranial CT scan showing nonspecific punctate left occipital, bilateral frontal, and right parietal calcifications (yellow arrows), hypodense right temporal intra-axial lesion (red arrows). Right parietal intra-axial lesion rounded, hypodense with liquid density (6.5 HU), hyperdense wall measuring 13x15 mm (green arrows), and vasogenic edema (blue arrows).

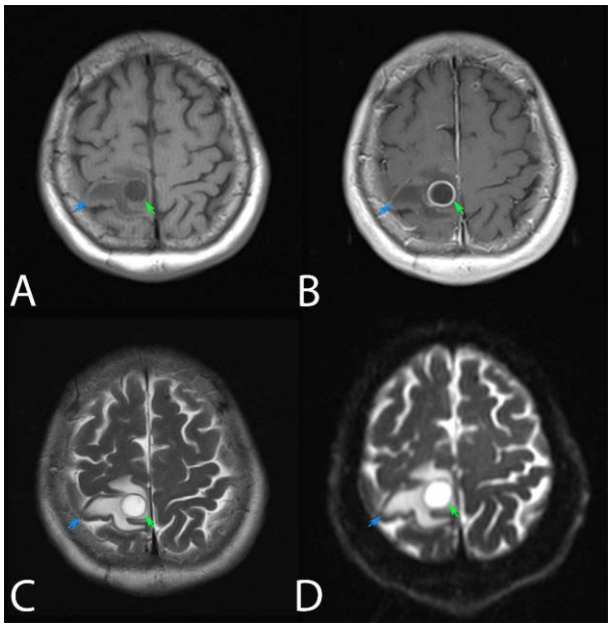

Figure 2. Brain MRI with contrast showing focal cortico-subcortical lesion in the right parietal region (green arrows) (approximately 20x16 mm) with discretely heterogeneous internal signal, predominantly hypointense in T1 (**A**), hyperintense in T2 (**C**), and diffusion-weighted (**D**), identifying iso-intense eccentric focus in all sequences and capsular-pseudocapsular margin less than 2 mm thick. Capsular-septal and eccentric focus enhancement, especially in postcontrast sequences (B), associated perilesional vasogenic edema conditioning discrete local mass effect with effacement of adjacent cortical sulci (blue arrows).

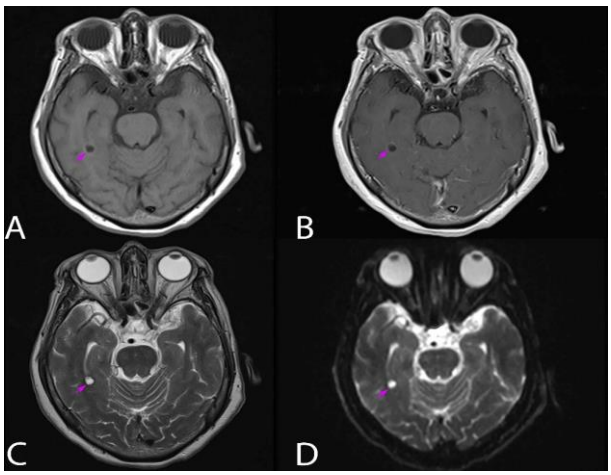

Figure 3. Brain MRI with contrast showing focal cortico-subcortical lesion in the right temporo-basal region (purple

arrow) with hypointense signal in T1 (**A**), hyperintense in T2 (**C**), and diffusion-weighted (**D**) (approximately 7 mm in diameter) with small isointense eccentric focus in all sequences and a thin capsular-pseudocapsular margin less than 2 mm thick. Fine capsular/pseudocapsular and eccentric focus enhancement, especially in postcontrast sequences (B), without evidence of adjacent vasogenic edema.

During a cranial computed tomography (CT) scan, a round and hypodense lesion in the right parietal lobe of 13x15 mm in diameter was reported with vasogenic edema and nonspecific punctate calcifications on the left frontal and right parietal areas (Figure 1). Cerebral magnetic resonance image (MRI) with contrast reported parenchymal and subarachnoid neurocysticercosis in different stadiums: right parietal intra-axial lesion in the vesicular colloid stage and perilesional vasogenic edema (Figure 2); lesions in the vesicular stage in the right temporal intra-axial lobe (Figure 3) and extra-axial frontal bilateral lobe, temporary right and pericallosal; intra-axial lesions in the nodular-calcified stage left frontal, right parietal, and right temporal lobes.

The patient started treatment with albendazole 600 mg PO diary for 10 days, praziquantel 2.000 mg PO every day per 10 days, dexamethasone 4 mg IV every 8 hours per two days, dalteparin 2.500 U SC every day, and antiseizure medications with levetiracetam 500 mg PO every 12 hours. Because of the national shortage of praziquantel, the patient could not continue treatment; furthermore, on day nine, she was evaluated by an infectologist, who recommended continued management with albendazole and dexamethasone for 30 days due to subarachnoid compromise. The patient completed the new recommended therapeutic regimen and was discharged to continue follow-up by a specialist.

Discussion

The high incidence of NCC in endemic countries (predominantly low- to middle-income countries) is associated with poverty conditions such as less hygienic conditions and permanent contact with domestic animals. The rise of cases associated with poor scientific production leads to the abiding of infection. (7,8) Our case is not far from this reality, since the patient lives in conditions of poverty and neglect in an endemic country.

Even before Escobar described the four stages of NCC, Carpio in 1994 proposed a classification of NCC according to the viability and location of the parasite. (8, 9) This classification is also important to identify the parasite activity, being stage active when the parasite is alive, transitional if the parasite is in the degenerative phase, and inactive if the parasite has been eradicated. (9) This classification can be useful for clinicians, just as the Escobar classification is useful for pathologists and radiologists to identify the life cycle of the parasite within the body and the specific imaging findings of each phase. At the vesicular stage, in computed tomography (CT) and MRI, the cyst fluid has similar characteristics to CSF; the vesicular wall measures up to 4 mm and the lesion up to 20 mm. Notice that the scolex is eccentrically within the lesion; it can be iso or hyperintense in both T1 and T2 weighted sequences and hyperdense on CT.

In addition, at this stage, there is no calcification or perilesional oedema. (5, 6) In the next stage, the colloidal vesicular, the cyst fluid becomes turbid due to an inflammatory response at the brain parenchyma after the larval degeneration from the scolex, is hyperdense on CT in comparison with the CSF, and the cyst wall is irregular and thicker. (9) The patient presented multiple small lesions at the vesicular stage in subcortical and deep supratentorial bihemispheric white matter, thalami, lenticular nuclei, internal capsules, midbrain, pons, and in both cerebellar hemispheres, and one colloidal vesicular lesion at the right parietal lobe with surrounding edema.

The cyst gets smaller in the granular stage, turning into a granulomatous nodular lesion. There is often gliosis around the lesion, and there is some edema but not as much as in the previous stage. Finally, the lesion is about a quarter of its original size in the nodular calcified stage, which is a nonactive stage of the disease. There is no oedema around the lesion. (9) In this scenario, CT imaging is better suited to identify this lesion than the MRI because it can clearly depict the calcified nodule; however, in the MRI, it is possible to identify mild contrast enhancement surrounding the calcification, and seizure activity could be present even knowing that this is the non-active stage of disease. (6) In our case, the patient presented lesions compatible with granular and calcified stages at the left parietal and frontal lobes, easily identifiable on CT.

After confirming the diagnosis, treatment includes the use of niclosamide or praziquantel, the

latter being more effective. (11) Unfortunately, our patient could not receive Praziquantel treatment due to a supply shortage and was treated with a less effective alternative regimen while waiting to resupply. (11)

Conclusion

NCC is a public health problem in endemic countries that requires further attention. Clinical manifestations are variable; however, the disease may be suspected in cases of late-onset epilepsy. Knowledge of different parasite stages and neuroimaging findings is essential to making a correct diagnosis.

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