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Neurological sequelae in a patient with previous neurocysticercosis

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Introduction

Teniasis and cysticercosis are caused by the platelminth *Taenia solium*, a parasite that has in its cycle the pig and man. Teniasis occurs with the ingestion of raw or undercooked contaminated pork containing cysticercus. On the other hand, human cysticercosis occurs through ingestion of *T. solium* eggs through fresh vegetables, raw meat or contaminated water, in the body, the larvae migrate and acquire the cystic form, usually in the host's muscle tissue. Neurocysticercosis (NCC) develops when the invasion of the larvae occurs in the human central nervous system, found in the nervous tissue or in the intraventricular, subarachnoid, and spinal cord spaces, where there is circulation of CSF, leading to different clinical manifestations and prognosis.

Objective

To report the case of a patient with NCC with neurological complications at IIER.

Case description

Female, 37 years old, carrier of diabetes mellitus type 2 (DM2), migraine and structural epilepsy. She has a previous history of rheumatic fever in childhood and NCC since 2001, contracted when she lived in the interior of Bahia where she raised pigs and ate this meat, treated at IIER in May/2019. A headache associated with absence and vertigo crises began in October/2020 as consequences of NCC, reasons for which the patient was hospitalized and later referred to the neurology outpatient clinic, after CSF analysis without evident alterations. The patient was admitted to the IIER with a history of intermittent vertigo for 2 days and shooting headache in the right frontal region of the skull, in addition to the return of absence seizures until then extinct since the last hospitalization in October/2020, accompanied by the neurologist physician who increased valproic acid from 1.25 g to 1.5 g a day, also reported that this time, presents very significant worsening of vertigo. The patient presents multiple calcifications of marked hyposignal with predominance in the cortico-subcortical regions on magnetic resonance imaging (MRI) and also evidenced on computed tomography (CT).

Discussion

Due to the different locations of the cysticercus in the CNS, the number, size, age and vitality of the patient, the stage of evolution and their reactions on the host and immune response, different signs and symptoms occur in NCC. The parenchymal form usually presents with headache and seizures, with a good prognosis, as it tends to respond well to anticonvulsant therapy. In this case, the patient had vertigo, headache in the right frontal region, and absence seizures. Chronic calcified CCHN is formed due to an inflammatory response from the host, the calcified lesion is not completely inactive and may cause seizures. The patient had a predominance of hypodense nodules on CT characterizing calcified lesions bilaterally in the brain parenchyma, in which in most cases, may be the only evidence on neuroimaging. In addition to having calcifications, on MRI from previous admissions there was a single central encephalic lesion with a hyposignal halo on T2, with features compatible with the colloid nodular stage. Treatment is based on brain involvement and the specific type of disease. When there are one or more cystic or degenerative lesions antiparasitic and symptomatic treatment is given, when there are only calcified cysts, it is managed according to the symptoms.

Keywords: Neurocysticercosis, Cysticercus, Taenia solium.

