CASE REPORT

Disseminated Cysticercosis

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CASE PRESENTATION

A 49-year-old male refugee from Uganda with a history significant only for hypothyroidism and hypertension presented to the neurology clinic with an eight-year history of seizures. He has not had a seizure for almost two years while on phenobarbital and phenytoin. He denied any history of parasitic infections or any kind of anti-parasitic treatment in the past. He did not have any headache, fever, nausea, or vomiting but reported intermittent episodes of dizziness. Findings on neurological and general physical examination were normal. Laboratory findings were notable for peripheral eosinophilia and thrombocytopenia present for a few months, which resolved in subsequent evaluations. Human immunodeficiency virus 1/2 Antigen/Antibody (HIV ½ Ag/Ab) screen was negative. T1-weighted magnetic resonance imaging (MRI) of his brain showed rim-enhancing cystic lesions in the left frontal and right occipital lobes, left parietal, left temporalis muscle, and left trapezius muscle. The parenchymal lesions were partially calcified. Cysticercosis serum antibody immunoglobulin G (IgG) was negative. Confirmatory immunoblot antibody testing offered by the Center for Disease Control and Prevention (CDC) was positive, supporting the diagnosis of disseminated cysticercosis.

DISCUSSION

Neurocysticercosis (NCC) is reported to be the most common parasitic disease affecting the central nervous system. It usually takes a median of 3.5 years of incubation period before the onset of symptoms, and it is caused by the larval stage of the tapeworm *Taenia solium*. It is the primary cause of acquired epilepsy worldwide.[1] NCC is spread by the consumption of *T. solium* egg sloughed in the stool of the human tapeworm carrier. It then hatches in the small intestine and disseminates via blood to the brain, striated muscles, and other tissues.

While NCC is predominantly a disease of the developing world, the incidence has increased in developed nations because of immigration from the endemic regions.[8] Patients with NCC usually present with seizures, and it is often the only clinical manifestation. Other common presentations include hydrocephalus, headaches, and symptoms of increased intracranial pressure (ICP).[2]

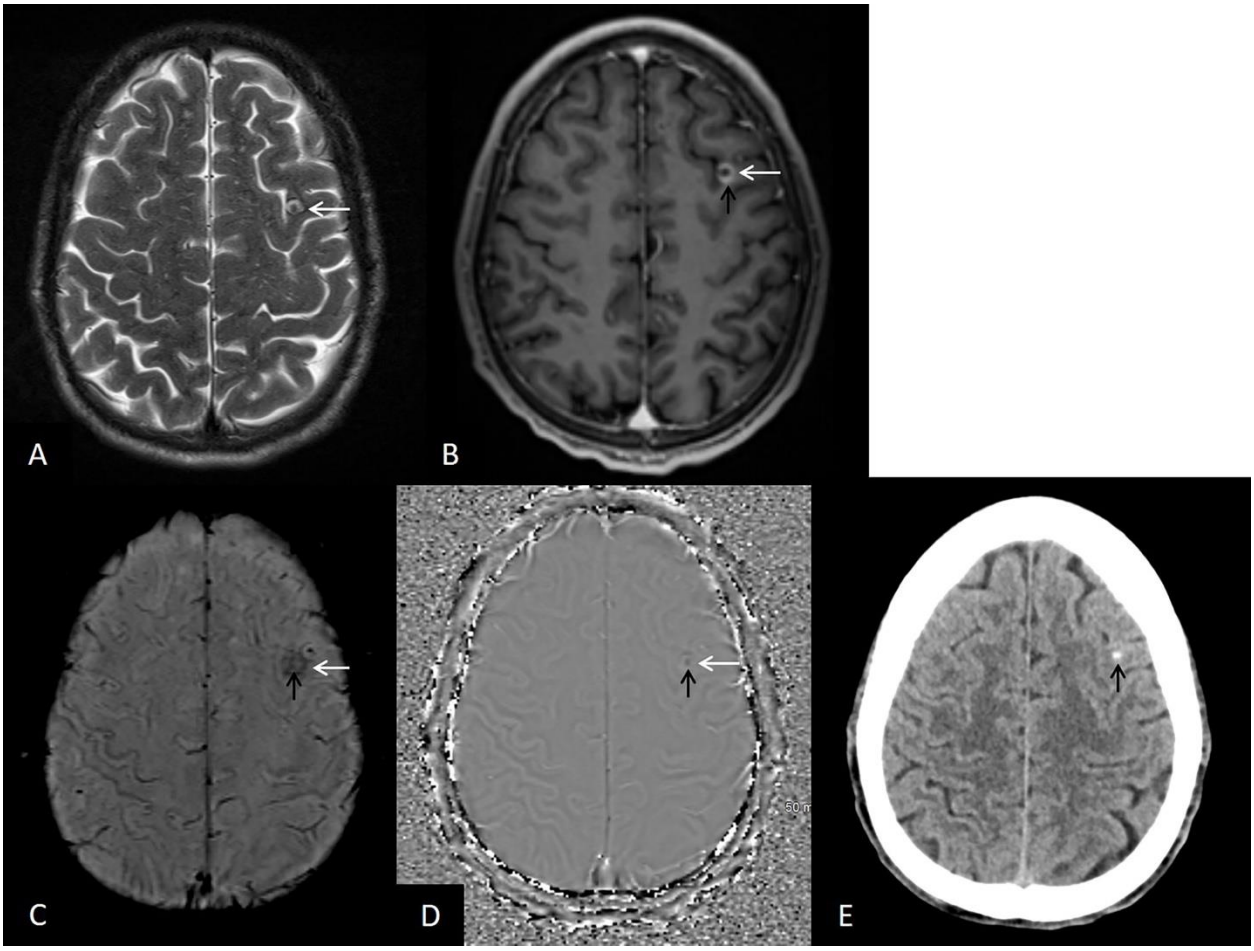

Figure. 1: Neurocystisercosis in a 49-year-old male. MRI shows left frontal NCC cystic lesion (white arrows) on axial T2 weighted image (A), with rim enhancement on axial contrast-enhanced 3D T1 MPRAGE (B) and central calcification (black arrows) on SWI (C), and phase image (D). Axial CT image (E) at the same level confirmed the central calcification.

The Infectious Disease Society of America (IDSA) guidelines recommend thorough history taking, physical examination, and neuroimaging studies in the diagnosis and management of NCC.[9] Exposure history should also be investigated carefully because of the latency period between the infection and onset of symptoms. The serologic testing method enzyme-linked immunotransfer blot assay is used as a test in patients with suspected NCC. Enzyme-linked immunosorbent assay (ELISA) are not recommended because of high rates of false-positivity and negativity.[3] Brain MRI and a non-contrast computed tomography (CT) scan are the best diagnostic measures in classifying patients with new diagnose of NCC.[4,5] Because most of the NCC cases present as parenchymal, subarachnoid, and spinal cysts, imaging studies provide more information than the immunodiagnostic test by incorporating the number, size, locations, and stages of the parasites.[6]

The management of the NCC depends on the stages of the disease. Four neuroimaging stages of the disease have been identified. These stages include vesicular, colloidal (colloidal vesicular), granular (granular nodular), and calcified (calcified nodular). On CT imaging, the vesicular stage is identified as 10 – 20 millimeters, a thin-walled cystic lesion with associated scolex

(small central isoattenuating focus). The vesicular stage has none to minimal surrounding edema or contrast enhancement. The colloidal stage is described by hyperattenuating cysts with pericystic enhancement and edema. The granular stage is similar to the colloidal stage but has significant edema and thicker-walled contrast-enhancing lesion. The calcified stage is characterized by the hyperdense lesion (calcified nodule) without edema and enhancement.[11]

The initial approach to managing NCC is to treat acute symptoms, including seizures. Following stabilization of seizure with anti-seizure drugs, antiparasitic and anti-inflammatory medicines (corticosteroids) should be administered.[9] Several randomized trials and meta-analysis studies suggested that the use of corticosteroids and a short course of albendazole (5mg/kg/day for 7days) as a treatment regimen results in rapid radiologic resolution with fewer seizures six months post-treatment.[6]

According to a randomized trial of 120 patients in Peru, the number of cysts determined the choice of antiparasitic in patients with viable parenchymal NCC and seizures.[9] Viable NCCs appear round and hypodense on a CT scan with none to minimal inflammation of the surrounding tissue.[10] For the patients with one to two viable cysts, albendazole monotherapy (15mg/kg per day up to 1200 mg per day, with a meal) is recommended. On the other hand, if the patient has more than two parenchymal cysticerci, treatment should include albendazole and praziquantel (50 mg/kg per day in three daily doses).[2] Corticosteroids should also be administered before and during antiparasitic therapy. The combination therapy is reportedly linked with a higher rate of radiographic resolution than just albendazole. IDSA suggested retreatment with antiparasitic therapy for cystic parenchymal lesions that persist for more than six months after completing the initial treatment and repeating the MRI every six months until the cystic component resolves.

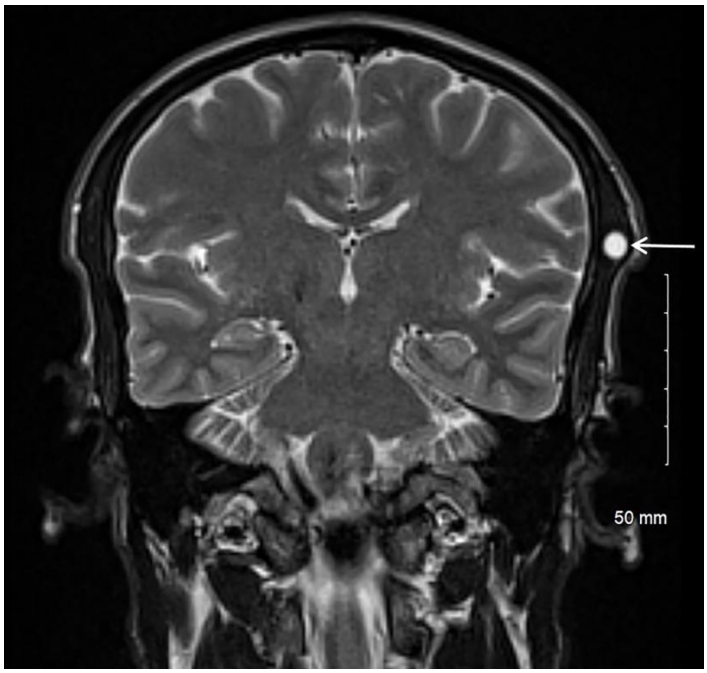

Figure 2: Neurocystisercosis in a 49-year-old male. Coronal T2 weighted image shows extra-cranial NCC cystic lesion (arrow) within the left temporalis muscle.

It is recommended to use anti-parasitic drugs in all patients with viable intraparenchymal neurocysticercosis (VPN). Antiparasitic therapy quickly treats the active cysts, diminishes the seizure risk, and reduces the chance of recurrent hydrocephalus. However, this could also worsen neurologic symptoms because of inflammation surrounding the degenerated cyst, especially if the patient has multiple parenchymal lesions leading to possible diffuse cerebral edema.[9] Thus, the use of corticosteroids mitigates these adverse effects. Since patients with possible infection need long-term treatment with corticosteroids, IDSA recommends screening for latent tuberculosis infection and screening or empirical treatment for *Strongyloides stercoralis.* [9]

For extra-parenchymal cyst, IDSA recommends identifying intraventricular and subarachnoid cysticerci using MRI with 3D volumetric sequencing in a patient with hydrocephalus and suspected NCC to eliminate the partial volume averaging effect.[9] For the optimal approach in managing intraventricular neurocysticercosis (IVN) in the 3rd ventricles, IDSA also advices removing the cysticerci by minimally invasive neuroendoscopy over other surgical or medical approaches.[9] Experts advise not to use anti-parasitic drugs preoperatively since it can disrupt parasitic integrity and inflammatory reaction, which would prevent successful cyst removal. However, for the 4th ventricular cysticerci, surgical intervention is preferred over medical therapy, if feasible.[9] Patients with NCC also need an ophthalmologic examination before initiation of anthelminthic therapy to exclude ocular cysticercosis.[9] If patients are suspected of acquiring NCC in an endemic area, screening the household members for a tapeworm carriage is crucial.

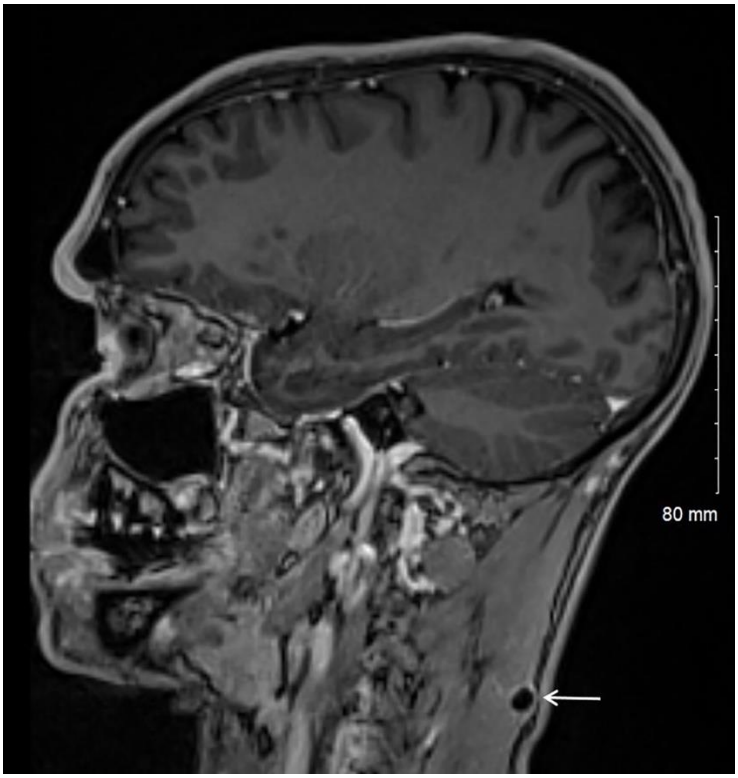

Figure 3: Neurocystisercosis in a 49-year-old male. Sagittal contrast enhanced T1 MPRAGE demonstrates rim enhancing NCC cystic lesion (arrow) within the left trapezius muscle.

PATIENT OUTCOME

Based on imaging and positive serologic testing results, the patient was diagnosed with VPN with extracranial involvement – disseminated cysticercosis. Ophthalmology examination did not reveal ocular involvement. Because of his travel history and anticipation of starting steroids, he was screened for strongyloidiasis, and it came back negative. He was diagnosed as having latent tuberculosis, for which he received isoniazid (INH) and vitamin B6 prior to starting him on neurocysticercosis therapy. He received dexamethasone, praziquantel, and albendazole for ten days. Plans for a repeat MRI in 6 months after completion of therapy will be made to document non-viable calcification of all lesions.

Notes

Potential conflicts of interest: The author reports no conflicts of interest in this work.

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