Acute Sensorimotor Deficit as a Lone Manifestation of Neurocysticercosis: An Important Differential Diagnosis in Emergency Departments of Developed Countries

Sir,

Neurocysticercosis (NCC) is a leading infection of the central nervous system in endemic regions and is increasingly seen in the globalized areas with frequent population shifts. Therefore, familiarity with its presentation and pathologic findings are important across clinical settings. NCC presents significant health-care burden, particularly Asia, Africa, Eastern Europe, and Latin America.[1,2] In terms of clinical manifestations, 79% of patients report seizures and 38% headaches,[3] while other signs/symptoms are nonspecific and depend on the number, stage, and size of lesions as well as the host immune response.[4] Whereas, humans are the definitive hosts who contract a gastrointestinal disease (i.e., teniasis) and humans can also become intermediate hosts (i.e., fecal–oral egg transmission in food/water contaminated by human feces) resulting in cysticercosis, a disseminated soft-tissue infection of muscle, eye, and/or brain.

A 42-year-old male presented to the emergency department of a large US urban city with acute left-sided weakness (leg worse than arm, which was worse than face) in the early morning, with difficulty ambulating. Examination revealed decreased sensation on the left side. Vitals were stable. Given that the patient was at baseline health the prior night, a "code stroke" was activated. The patient emigrated from Honduras one decade ago, has no history of headaches or seizures, and worked in industrialized food processing. Initial laboratory tests were unremarkable. Head computed tomography (CT) revealed a 1.8 cm × 1.6 cm × 2.1 cm lesion in the right superior frontal gyrus with edema [Figure 1a], postcontrast ring enhancement, and calcifications near the right posterior insula and right superior parietal lobe. Diffusion-weighted imaging of the brain with apparent diffusion coefficient [Figure 1b] and trace image comparisons [Figure 1c] demonstrated a likely scolex, a marginated cyst that is pathognomonic hallmark of NCC.

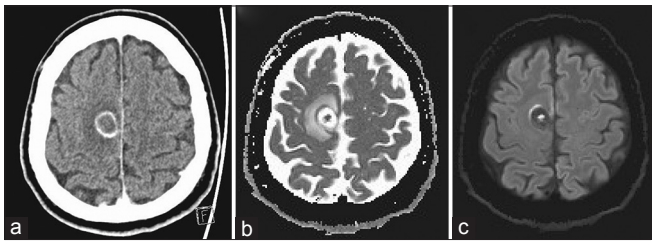

Figure 1: Computed tomography (a) axial view of right hemispheric lesion with surrounding edema; diffusion-weight imaging and apparent diffusion coefficient (b); and trace (c)

Treatment was initiated with intravenous dexamethasone, followed by high-dose oral albendazole 400 mg twice daily. The vasogenic edema surrounding the scolex most likely explained the acute onset of neurologic deficits. Serum studies resulted positive for anticysticercal antibodies. Medical treatment of 2 weeks of high-dose albendazole and 16 days of prednisone taper was completed, and notably, the patient returned to his baseline neurological status.

Particularly, in the US geographic Southwest, a continued surge in immigrants and international travel has been associated with rising reports of NCC. For patients who do not present with the classic vignette, clinical vigilance is necessary. The four NCC pathological stages are as follows: vesicular (small cyst with clear fluid with scolex with no surrounding edema), colloidal vesicular (wall thickening with surrounding brain edema as inflammation response initiates), granular nodular (cyst degeneration with pericystic gliosis and mild edema), and nodular calcified (shrunken lesion as a calcified nodule).[5] The 2002 Consensus Guidelines list the following absolute diagnostic criteria: histology of parasite from biopsy, cystic lesion of scolex on CT or magnetic resonance imaging, or subretinal parasites by fundoscopy. Albendazole and praziquantel are both effective anthelmintics. To reduce the risk of edema and intracranial hypertension, corticosteroids are initiated 1 day before the start of anthelmintics, followed by a slow taper once the anthelmintic is completed.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms for publication before preparation of the manuscript. In the form, the patient has given consent for images and clinical information to be reported in the journal. The patient understands that their name and initials will not be published and due efforts to be made to conceal their identity, but anonymity cannot be guaranteed. This report contains no identifiable patient data.

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Conflicts of interest

There are no conflicts of interest.

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