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Case report

Reversible dementia in the elderly as a rare manifestation of neurocysticercosis

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ABSTRACT

Neurocysticercosis (NCC) is a leading cause of seizure and epilepsy worldwide. There are no path gnomonic features or a typical NCC syndrome. In this communication we describe an interesting case of multiple NCC in an elderly presenting only with dementia and responded well to steroid and cerebral decongestant. The case report highlights an uncommon presentation of NCC and recognizing this reversible cause will avoid unnecessary investigation and delay in treatment.

1. Introduction

Neurocysticercosis (NCC) is a common parasitic infection of the human central nervous system caused by pork tapeworm *Taenia solium*. NCC is a serious public health problem in several developing countries in Latin America, Asia and Africa. It is well known that NCC, the commonest parasitic brain disease,[1] causes epilepsy, focal neurological signs, intracranial hypertension, stroke and neuro-psychiatry symptoms.[2,3] We report a case of an elderly person presented with rapidly progressive dementia as the isolated manifestation of multiple NCC.

2. Case report

A 65-year-old right-handed male, postgraduate and clerk by profession, presented with occipital headache which was moderate to severe in intensity with occasional bouts of vomiting of 4 months' duration and a gradual progressive decline in cognitive functions of 3 months' duration. Initially he had problem in performing his professional work. He had great difficulty in calculation and made mistakes in his files, had difficulty in comprehension of paragraphs, forgot information, and misplaced objects and files. He was unable to supervise his children's studies and often lost his temper and responsed with irritability. There was marked slowness in doing daily activity and thought processes, lack of spontaneity, apathy, and lack of interest toward the environment and himself. Gradually he became unconcerned about his personal hygiene, forgot to eat meals, and got lost in new places. He was unable to recall his address, telephone numbers, or names of close relatives within a span of three months. His cognitive decline was severe enough to impair his personal and occupational performance. His symptoms were observed by his wife and reported to our hospital.

There was no history suggestive of coronary artery disease, cerebrovascular disease, head injury, joint pain, photosensitivity or rashes. A review of the central nervous system did not reveal any history of loss of consciousness, seizure like activity, abnormal involuntary body movement, gait ataxia, dysphagia, dysarthria, or any weakness. There was no history suggestive of diabetes and hypertension or any past or family history of any similar psychiatric or neurological illness.

The general physical and systemic examination was normal. Although he was conscious and alert, he resisted evaluation. Upon mental state examination, his Folstein's mini mental status examination (MMSE) score was 18 (orientation of place 4/5, calculation 2/5, registration 1/3, recall 0/3, language 6/9). Immediate, recent, and remote memory was impaired and on lobar function testing using Strub and Black scheme there was impairment in declarative memory (semantic and episodic), executive function (judgment, abstract thinking), naming, and visual spatial orientation. The speech was relevant and coherent. There were no focal neurological deficits and the cranial nerves were intact. The fundus examination was normal.

Laboratory investigations included complete blood count, general blood picture, erythrocyte sedimentation rate, routine chemistry, liver and renal function tests, enzyme-linked

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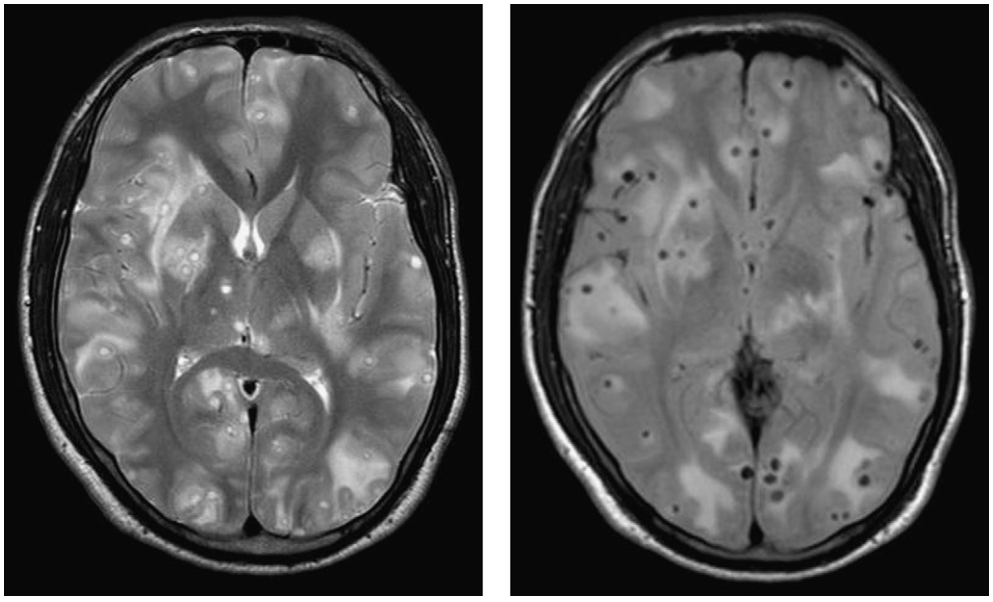

Fig. 1. (A) T2 and (B) FLAIR magnetic resonance images of brain, revealing multiple ring lesions with prominent hyopintensity in centre surrounded by hyperintensity at the periphery with eccentric scolex scattered throughout gray and white matter.

immunosorbent assay (ELISA) for human immunodeficiency virus, and collagen vascular profile, all of which were within the normal limits. Thyroid function tests and vitamin B12 levels were within normal range. Electroencephalogram was normal. Magnetic resonance imaging revealed multiple cysts involving all of the cerebral cortex and some of the cysts had prominent scolex. There was adjoining hyperintensity on T2 and FLAIR images in a few of the lesions (Fig. 1). The ELISA to IgG antibody against *Taenia solium* glycoprotein in serum was positive. The patient was diagnosed as a probably case of NCC based on the revised diagnostic criteria of NCC by Del Bruto et al.[4] We excluded the other causes like tuberculomas, fungal granuloma, toxoplasmosis and tumors.

The patient was offered a steroid (prednisolone) 1 mg/kg body weight for 28 days followed by acetazolamide 250 mg twice daily for 2 months. There was improvement in cognitive functions after 2 weeks, the headache subsided and MMSE score improved to 24(calculation 4/5, registration 3/3, recall 2/3). He was able to do his official work independently after 6 months.

3. Discussion

NCC is a pleomorphic disease whose manifestation varies with the number, size, and topography of the lesion and the intensity of immune response of host to parasite. In this report we describe a patient presenting with dementia as the only manifestation of multiple NCC in the absence of any focal neurological deficit or seizure or features of raised intracranial hypertension.

Although the neuropsychiatric dimension of NCC was reported earlier, there are reports of NCC presented with psychiatric and intellectual impairment,[5] and with dementia as an isolated manifestation.[6] Patients with NCC often display cognitive impairment. Forlenza et al[7] reviewed 38 cases of NCC and reported psychiatric illness and cognitive decline in 65.8% and 87.5% of cases, respectively. Depression was the most frequent psychiatric diagnosis (52.6%) and 14.2% of the patients were psychotic. Mild to moderate cognitive deficits were more common than frank dementia. Interestingly, the number, type of brain lesions, use of corticosteroids, or epilepsy did not correlate significantly with the severity of psychiatric symptoms. Andrade et al[8] in a cross-sectional controlled study reported dementia in 5 (12.5%) out of 40 patients with NCC and the assessment of neuropsychological function of NCC cases showed significant impairment in executive function, memory, constructive praxis, and verbal fluency compared with healthy controls and epilepsy controls.

Dementia syndrome observed in patients with NCC could be a combined effect, resulting from multiple parasitic and vascular lesions, disrupting frontal-parietal-temporal networks related to intellectual functioning in patients with vulnerable brains (because of repeated epileptic seizures, low educational levels, and advanced age). The results of this study suggest that dementia occurs frequently in patients with untreated NCC, and it is reversible in most.[9] Another potential mechanism postulated for cognitive decline is an inflammatory reaction accompanying the death of the parasite with edema and dysfunction of perilesional tissue.

Guidelines for treatment of NCC must be individualized in terms of number and location of lesions, as well as based on the viability of the parasites within the nervous system. The Cochrane Database review on drugs for treating NCC concludes that there is insufficient evidence to assess whether cysticidal therapy in NCC is associated with beneficial effects.[10]

Three major arguments against the use of cysticidal therapy in NCC have been raised: (1) there are immediate risks because of neurologic symptoms due to the acute inflammation that results from the death of the cysts; (2) the long-term prognosis of the underlying seizure disorder may worsen because of increased scarring due to the acute inflammation; and (3) treatment may be unnecessary since most cysts recess spontaneously within a short period. Corticosteroids are frequently used to decrease neurological symptoms due to the death of the parasite and are the primary management for chronic cysticercosis arachnoiditis or encephalitis. Mannitol, at doses of 2 g/kg/day, is also used for acute intracranial hypertension secondary to NCC.[11] We treated our patient with steroid and osmotic diuretics with dramatic improvement in symptoms.

We conclude that our case report is a rare nonepileptic manifestation of a very common endemic problem, NCC. It is important that the clinician should be familiar with this rare presentation as the dementia is reversible in most cases when diagnosed at an early

stage. Recognizing this reversible cause will avoid unnecessary investigation and delays in treatment.

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