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Post-traumatic acute disseminated encephalomyelitis

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A 23-year-old black African male fell and bumped his head from a tackle while playing soccer. He subsequently became blind from optic neuritis. An MRI of the brain showed white-matter changes suggestive of acute disseminated encephalomyelitis (ADEM). MR spectroscopy of the brain showed a demyelination pattern. This case report brings to the fore unsettled questions about ADEM, among them being whether it can occur as a post-traumatic event.

Introduction

The term acute disseminated encephalomyelitis (ADEM) was first used in 1950 to describe an immune-mediated encephalomyelitis from infection, immunizations, and allergies (1). ADEM has a striking resemblance to multiple sclerosis in that it is also a white-matter (demyelinating) condition. Unlike multiple sclerosis, however, it tends to be monophasic, even though recurrences have been described. These recurrent forms blur the distinction between ADEM and multiple sclerosis (2). ADEM is a potentially serious condition that can cause encephalopathy and even demise of a subject. Its association with various infections is well described (3, 4). ADEM has also been described post vaccination (5). Poser linked central nervous system trauma to formation and even enlargement of multiple sclerosis plaques (6). ADEM, on the other hand, has not been described post head trauma. Howeer, Irani once mentioned a claim by "some investigators" that ADEM could occur "at the heels of trauma." He went on to state that there wasn't much more to be said since this is not a well understood phenomenon (7). A literature search for a link between head trauma and ADEM came to naught. Nakamura et al described a 36-year-old man who presented with "multiple sclerosis like" white-matter changes post trauma, with optic neuritis and high cerebrospinal fluid (CSF) protein (8). The diagnosis of ADEM has some difficulties, since there are no specific biochemical markers for the condition.

Case report

A 23-year-old black African male patient fell and bumped his head from a tackle during a soccer game. He denied losing consciousness and did not recall being disoriented subsequently. No further details about the incident were available. Three days later, he suddenly became completely blind, with an unsteadiness of gait. He had dilated pupils bilaterally, with bilateral swelling of the discs. The features were suggestive of optic neuritis, a finding made in agreement with our ophthalmologists. The rest of the neurological examination revealed no abnormalities except for increased deep-tendon reflexes in the lower limbs. However, he had normal tone with flexor plantar responses and normal sensation. He had a cautious gait as a result of the visual impairment. An MRI exam (Fig. 1) showed large lesions in the white matter with no evidence of blood (as could have arisen from trauma).

MR spectroscopy showed a markedly high choline with a reduced N-acetylaspartate (NAA)-to-creatine (Cr) ratio. There was also a lactate peak, shown as an inverted doublet (Fig. 2).

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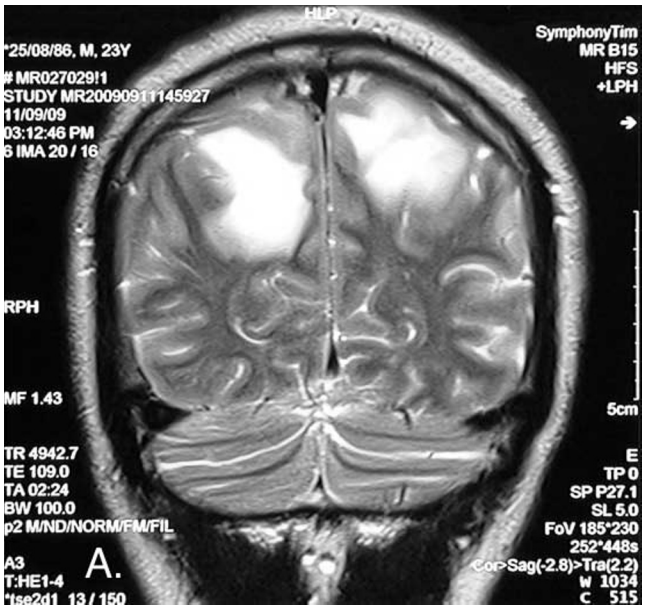

Figure 1A. 23-year-old male with post-traumatic acute disseminated encephalomyelitis. Coronal T2-weighted image shows parasagittal high-signal intensity lesions with vasogenic edema in white matter of both hemispheres.

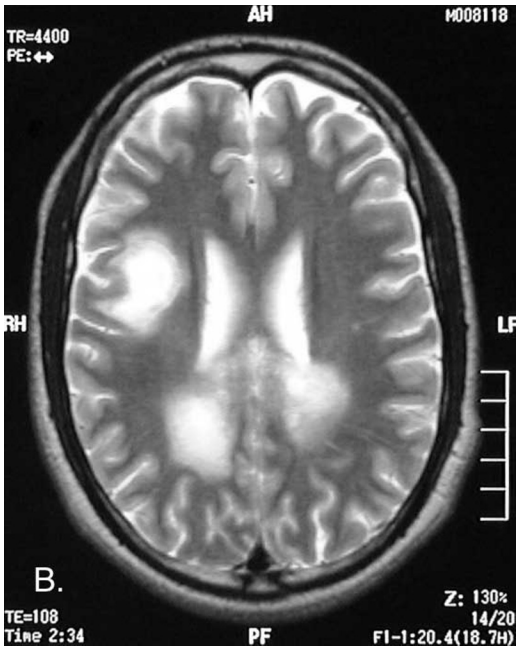

Figure 1B. 23-year-old male with post-traumatic acute disseminated encephalomyelitis. Axial T2-weighted image shows the high-signal parasagittal lesions with an additional lesion in the right parietal lobe.

At no time did the patient exhibit a pyrexia. A lumbar puncture was not done due to a concern for raised intracranial pressures. There was no evidence of venous sinus thrombosis, and the lesions were strictly confined to the white matter. He was put on high doses of methylprednisolone (500mg intravenous daily for 5 days) and then tapered with oral steroids over 12 days. The patient's vision improved to a point of seeing movement and the silhouette of objects and people. Later assessment showed a further improvement in which he was able to appreciate color. A full restoration of vision was never achieved, however. He developed some pallor of the disc, suggesting optic atrophy in spite of the steroids. He was found to be HIV-positive with a cluster of differentiation (CD) 4 count of 177 cells/mm3. Syphilis studies were nonreactive.

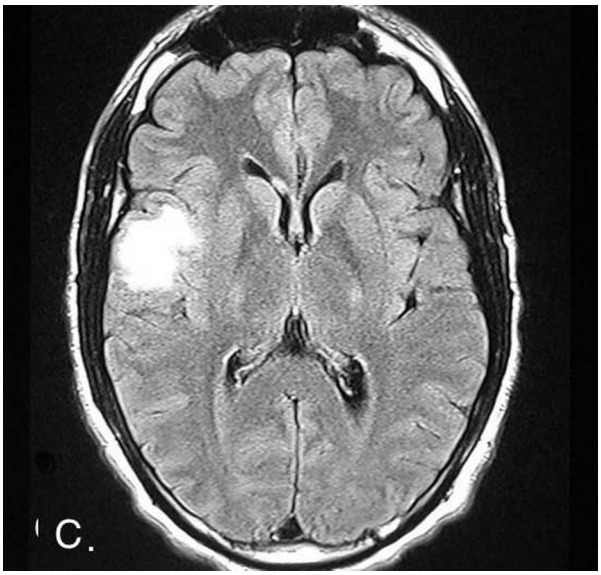

Figure 1C. 23-year-old male with post-traumatic acute disseminated encephalomyelitis. Axial FLAIR image showing the high-signal-intensity lesion in the right temporo-parietal region.

Discussion

The MRI showed white-matter changes of the same age that were reminiscent of ADEM. A choline rise is a feature of demyelination, albeit not specific (9). A lactate peak, even though a general feature of hypoxic brain conditions, has been described with ADEM (10). In multiple sclerosis, unlike with ADEM, optic-nerve involvement tends not to be simultaneous. Other pointers arguing against multiple sclerosis include the white-matter lesions of the same age, lesions > 2cm that are atypical for multiple sclerosis, occurring only with the rare tumefactive variant (11, 12). Multiple sclerosis is also very uncommon among black Africans, and the patient had no history of mixed lineage (13). The presence of Dawson's fingers (multiple-sclerosis lesions around the ventricle-based brain veins) is a sign attributed to multiple sclerosis rather than ADEM. This finding, however, was in only 21% of a study population with multiple sclerosis (14).