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Case Presentation

Presentation and Rehabilitation in a Patient With Toxoplasmosis Encephalitis: A Case Study and Review

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Abstract

Toxoplasma gondii is an opportunistic infection that often presents in the setting of acquired immunodeficiency syndrome. The infection can cause severe and potentially fatal encephalitis because of the reactivation of latent infections in the setting of immune suppression. Diagnosing toxoplasmosis encephalitis (TE) in immunocompromised patients often is difficult because the signs and symptoms can be nonspecific, but making a diagnosis of TE is even more challenging in a patient who is not known to have human immunodeficiency virus/acquired immunodeficiency syndrome and shows no other signs of being immunocompromised. Early diagnosis and treatment can result in rapid radiologic and clinical improvement; however, no studies exist that evaluate the utility of functional rehabilitation for patients diagnosed with TE. Although previous studies report a good prognosis for patients who receive antibiotic treatment, they do not discuss the extent to which functional abilities lost during the infection are returned after their treatment. We discuss a case of stroke-like presentation of cerebral TE in a patient whose human immunodeficiency virus status was previously unknown and report the functional improvements that were made during acute inpatient rehabilitation.

Introduction

Toxoplasma gondii is an intracellular parasite that emerged as a major opportunistic infection in the setting of acquired immunodeficiency syndrome (AIDS), presenting as a severe and potentially fatal encephalitis because of the reactivation of latent infections in the setting of immune suppression [1]. Toxoplasmosis encephalitis (TE) is the most common opportunistic infection of the central nervous system in patients with AIDS ($CD4^+$ count <200 cells/mm^3), and it is also the most common cause of focal neurologic deficits in HIV-positive patients [2,3]. Previous estimates have suggested that TE will ultimately develop in 30%-40% of patients with AIDS in the absence of prophylaxis [4].

Cerebral toxoplasmosis in patients with HIV/AIDS may present with headache, seizures, and focal neurologic deficits, and early diagnosis and treatment can result in rapid radiologic and clinical improvement [3]. To date, however, no studies exist that evaluate the feasibility of functional rehabilitation for patients diagnosed with TE. Here, we discuss a case of stroke-like presentation of cerebral TE in a patient whose HIV status was unknown previously and report the functional improvements that were made during acute inpatient rehabilitation.

Case Description

A 30-year-old, right-handed man with no significant medical history was in his usual state of health when he began to develop right-sided numbness and blurred peripheral vision in the right eye. After approximately 1 month of persisting symptoms, the patient presented to an outside hospital and was found to have several lesions in the brain on head computed tomography (CT) scan with a high suspicion for cerebrovascular accident. He was placed on anticoagulation during his first day in the hospital. Magnetic resonance imaging (MRI) of the brain was obtained and showed 2 enhancing lesions, one in the left frontal lobe measuring $1.8 \times 1.5 \times 1.6$ cm with surrounding edema and one in the left thalamus measuring $1.6 \times 2.1 \times 1.4$ cm. The radiology report determined the lesions were most consistent with metastatic disease; however, a CT scan of the chest, abdomen, and pelvis with contrast from the outside hospital was unremarkable.

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Because stroke was ruled out, the decision was made to proceed with a brain biopsy, given the concern for malignancy and the lack of a primary tumor source identified on full-body imaging; however, the biopsy was delayed because of the administration of anticoagulation, and the patient was instead started on dexamethasone and discharged to home with a plan for close follow-up. At home, the patient's symptoms progressed to more dense deficits and an increased area of numbness reported, so he presented to our institution for a second opinion.

On examination, the patient was thin, alert, and oriented and in no acute distress. Vital signs were stable and within normal limits. There was evident psychomotor slowing and periods of inattention. There was noted right lower quadrantanopia with limitation of supraduction and nystagmus with right end-gaze. Cranial nerves were otherwise intact. The patient had decreased muscle bulk and significant atrophy in the upper and lower extremities. Left upper and lower extremity strength and sensation remained intact. Right upper extremity strength testing revealed 2+/5 shoulder abduction, 4/5 elbow flexion and extension, 2/5 wrist extension, and 2/5 finger abduction. Spasticity was evident and graded at a Modified Ashworth Score of 1+/4 in the right elbow flexors. Right lower extremity strength testing revealed 2/5 hip flexion, knee extension, and ankle dorsiflexion, with 4/5 ankle plantar flexion and extensor hallicus longus dorsiflexion. Sensation to light touch was absent in the right upper and right lower extremities. Reflexes were 2+ throughout, toes were upgoing with Babinski reflex testing bilaterally, and there was no clonus. There was notable difficulty with finger-to-nose testing using the right hand compared with the left.

A basic metabolic panel, complete blood count, and liver function tests on admission were unremarkable. A repeat MRI again showed numerous new enhancing lesions and progression of previously seen lesions. The largest lesions were located in the left thalamus, measuring 3.2 × 1.9 cm, the right thalamus, measuring 1.7 × 2.1 cm, and the left inferior frontal lobe, measuring 2.2 × 1.9 cm, all with surrounding vasogenic edema. Overall, the pattern was still thought to be most consistent with metastatic disease, but in the subsequent days after admission, blood work returned positive for HIV-1 RNA with a CD-4 count of 11 and a viral load of 555,973 copies/mL. An enzyme-linked fluorescent assay was positive for *Toxoplasma gondii*, and a subsequent brain biopsy confirmed TE.

The patient was immediately started on sulfadiazine, pyrimethamine, and leucovorin for the treatment of toxoplasmosis and ritonavir, darunavir, and emtricitabine and tenofovir disoproxil fumarate for the treatment of AIDS. He was also started on azithromycin for *Mycobacterium avium* complex prophylaxis. Given his stable condition, the patient was transferred to acute inpatient rehabilitation.

On initial rehabilitation assessment, the patient was alert and oriented to self and time but with only partial awareness to place and surroundings. He was able to follow 3-step commands but required repeated directions and cuing. He was easily distracted during tasks and had impaired immediate recall. In addition, the patient had a flat affect and showed delayed processing and decreased thought organization with word-finding deficits. Details on the patient's Functional Independence Measures for activities of daily living along with speech, occupational, and physical therapies are provided in Tables 1-3, respectively.

The patient's early rehabilitation course was complicated by a brief period of altered mental status. After his first complete day of therapies, the patient returned to his room and became diaphoretic, anxious, and confused. He was not answering questions appropriately and began repeating random words and phrases. He was given 0.1 mg of clonidine, which resolved his anxiety. The exact etiology of this event was not uncovered, and the patient was maintained on a twice-daily dose of 0.1 mg of clonidine for approximately 2 weeks before tapering off the medication. It is important to understand that if rehabilitation is going to be successful in improving function in patients with this disease, the neurologic sequelae that can prevent them from participating in therapy must be well managed.

The course of rehabilitation included a comprehensive program of physical therapy, occupational therapy, speech language pathology, rehabilitation nursing, recreational therapy, psychology, and medical management by a physiatrist. The patient was also followed closely by infectious disease specialists for coordinated care.

Before discharge, a repeat MRI showed stable multifocal rim enhancing lesions with a decrease in associated edema compatible with improvement posttreatment (Figures 1 and 2). No new enhancing lesions or enlarging lesions were seen. The absolute CD-4 count increased to 48, and the HIV viral load decreased to 616 copies/mL. Repeat physical examination revealed at least 4/5 muscle strength in the right upper and lower extremity except for hip flexion, which was 3/5.

Discussion

Toxoplasmosis is a rare disease caused by an intracellular protozoan parasite, but it commonly affects

Table 1
Speech therapy FIMs

Task	Status on Admission	Status at Discharge	FIM Change	FIM Efficiency
Comprehension: Auditory	5	6	1	0.022
Expression: Vocal	4	6	2	0.044
Problem Solving	3	5	2	0.044
Memory	3	6	3	0.067

FIM = Functional Independence Measure.