Case Report

Post-operative quadriplegia as the initial manifestation of tumefactive multiple sclerosis

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Abstract

Post-operative quadriplegia is a rarely encountered complication and not previously reported as the initial presentation of tumefactive multiple sclerosis. We present an unusual case of a patient with such manifestation and atypical lesions on brain magnetic resonance imaging. The patient was treated with methyl prednisolone pulse therapy and showed a dramatic response. Uncommon neurologic diseases can have very unusual presentations, which should be taken into consideration when encountered with such patients. Considering this fact will help physicians in better decision-making and proper treatment planning.

Keywords: Multiple sclerosis, postoperation, quadriplegia, tumefactive

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Introduction

Tumefactive multiple sclerosis (TMS) is an inflammatory demyelinating disease of the central nervous system, and has recently been described as a subtype of multiple sclerosis (MS) that is characterized by the appearance of solitary or multiple space-occupying lesions associated with ring enhancement and perifocal edema on magnetic resonance images (MRI).[1-3] This disease is an extremely rare variant of MS, occurring in 1/1000 cases of MS or 3 cases per million per year.[2] Postoperation quadriplegia as the initial manifestation of TMS has never been previously reported. These lesions pose considerable diagnostic uncertainty, in part due to the atypical neurologic symptoms that can be observed as a consequence of the size, location, and the potential associated mass effect and edema.

Case Report

A 34-year-old lady was transferred to the intensive care unit (ICU) due to developing quadriplegia and decreased level of consciousness in the postanesthesia care unit. She had received general anesthesia for an exploratory laparotomy procedure because of right upper quadrant abdominal pain 6 h before referral. The laparotomy had yielded to the sole finding of fatty liver with no evidence of cholecystitis or other surgical complications. During the operation, the patient was stable and the procedure was uneventful; however, in the recovery room it was noticed that the patient had developed quadriplegia, decreased level of consciousness, and aphasia.

The patient had a past history of diabetes mellitus and hypothyroidism and had been taking metformin, glibenclamide, and levothyroxine.

She arrived in ICU with a Glasgow Coma Scale of 8/15 and muscle power of 1/5 in all extremities. The deep tendon reflexes were 1/2 in upper extremities and 0/2 in lower limbs.

Due to neurological findings, brain MRI was performed which revealed a focus of high signal intensity in left cerebellar peduncle extending to the left side of medulla oblongata on T2 and fluid-attenuated inversion-recovery (FLAIR) images showing expansion and peripheral enhancement after contrast injection [Figure 1]. A hypersignal lesion in T2 and FLAIR images were also noted in the right temporal

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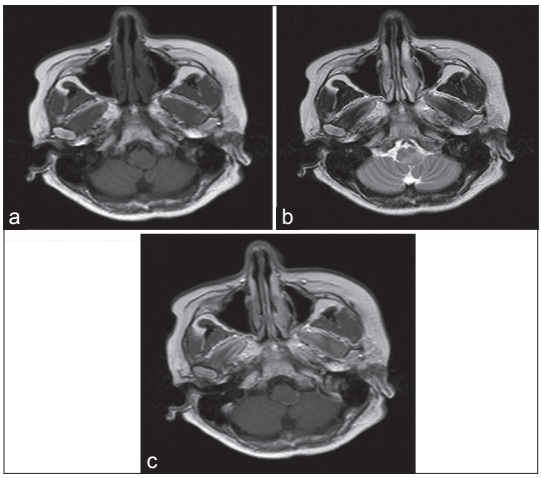

Figure 1: Brain magnetic resonance images (a-c) showing a focus of high signal intensity in left cerebellar peduncle extending to the left side of medulla oblongata on T2 and fluid-attenuated inversion-recovery images showing expansion and peripheral enhancement after contrast injection

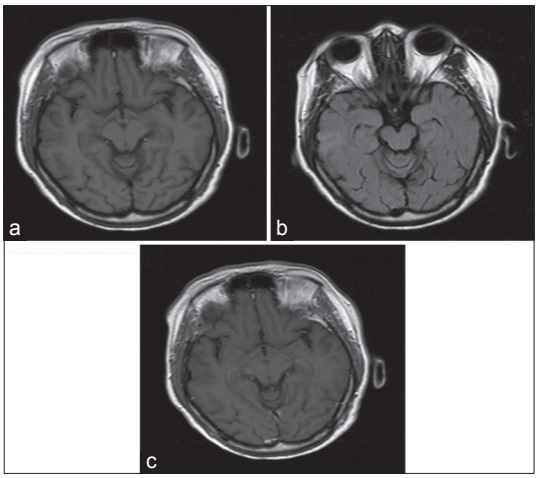

Figure 2: Images (a-c) revealing a lesion in right temporal lobe, involving both gray and white matter, which is hyposignal in T1, and hypersignal in T2, and fluid-attenuated inversion-recovery images

lobe, involving both gray and white matter, which was hyposignal in T1 [Figure 2].

Primary differential diagnosis included glioblastoma, rhombencephalitis, and TMS, so immediate complementary workups including blood samples, cerebrospinal fluid examinations, and rheumatologic serology tests were sent which were all within normal limits. Hence, based on all the findings, an impression of TMS was made, and the patient was started on methylprednisolone pulse therapy with the dosage of 500 mg intravenously every 12 h for 3 days. She showed dramatic response with improvement in muscle power, deep tendon reflexes, level of consciousness, and the ability to speak fluently after 4 days.

Discussion

Tumefactive multiple sclerosis is a very rare form of MS with the predominantly female distribution.[4] It can present with various symptoms, such as slowed mentation, memory lapses, limb weakness, confusion, gaze preference, visual changes, dizziness, and ataxia.[5-7] MS plaques on MRI typically appear as multiple, well-demarcated, homogenous, small ovoid lesions, lacking mass effect, and often oriented perpendicular to the long axis of the lateral ventricles.[8,9] In TMS, however, radiological findings totally differ including a solitary large lesion, size >2 cm, associated mass effect, perilesional edema, and/or the presence of ring enhancement.[10-13] The clinical and imaging characteristics of these demyelinating lesions may mimic primary and secondary brain tumors, brain abscess, tuberculoma, and other inflammatory disorders, e.g., sarcoidosis, primary Sjogren's syndrome, and thus confound the diagnosis.[14] The occurrence of tumor-like demyelination is reportedly rare being estimated at 1–2/1000 cases of MS.[9]

Limited numbers of previous case reports on TMS are available; however, this is the first reported patient with the initial presentation of postoperation quadriplegia and aphasia evoked after the general anesthesias a manifestation of TMS. Among the largest cohort of biopsy-proven TMS cases analyzed to date, common presenting symptoms in decreasing frequency included: Motor, cognitive, cerebellar, and brainstem dysfunction. In addition, atypical symptoms included headache, seizures, aphasia, cortical sensory loss or psychosis. The tumefactive episode represented the initial event in 61% of cases; however, 29% of patients had a prior history of relapsing neurologic symptoms, and 5% carried an established diagnosis of MS. Ring enhancement was the most frequently observed pattern of enhancement in this study.[2]

Acute onset of the disease, presence of ring enhancement, and dramatic response to pulse therapy helped establish the diagnosis in our patient.

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