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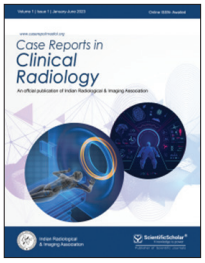

Case Report

Atypical demyelinating lesion of corpus callosum mimicking malignant mass lesion

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ABSTRACT

Tumefactive demyelination is a demyelinating disease that exhibits clinical and radiologic features similar to those of brain tumors. These lesions present a significant diagnostic challenge. The utilization of magnetic resonance imaging (MRI) imaging can contribute to the preoperative diagnosis of these lesions. In this case report, we highlight the importance of MRI in the diagnosis of tumefactive demyelination.

Keywords: Demyelination, Tumefactive lesion, Magnetic resonance imaging, Brain tumors, Diagnosis

INTRODUCTION

Demyelinating diseases can pose a challenge for clinicians in differentiating them from cerebral neoplasms, as both can present as mass lesions with similar clinical and radiological features, especially in the absence of temporal dissemination.[1] Tumefactive demyelinating lesions, characterized by solitary large lesions (>2 cm) with mass-like characteristics, typically display incomplete ring enhancement on post-gadolinium magnetic resonance imaging (MRI).[2] Common clinical symptoms include headache, cognitive changes, mental confusion, aphasia, and apraxia. Distinguishing large demyelinating lesions from brain abscesses or brain tumors can be challenging, often requiring a biopsy for a definitive diagnosis.[3] Tumefactive demyelination typically presents with acute symptoms and follows a monophasic course, with most cases experiencing complete remission on treatment with corticosteroids.[4]

CASE PRESENTATION

A 34-year-old male with k/c/o of diabetes and hypertension presented with subacute onset right-side visual disturbance (optic neuritis) for 1 year, left-side internuclear ophthalmoplegia, left-side facial palsy, numbness, and right-sided pyramidal sign for 1.5 years. He was on steroid therapy. Neuromyeitisoptica and Mylin oligodendrocyte glycoprotein antibody panels were found to be negative. Vasculitic panel of antibodies, such as antinuclear antibody, antineutrophil cytoplasmic antibody, anti-double stranded-deoxyribonucleic acid, and beta-2-glycoprotein, factor 5 leiden mutation, and methylenetetrahydrofolate reductase all were negative. Clinically, diagnosis was made up of demyelinating disease. 3T MRI brain and spine acquisition with axial reformats reveals fluid-attenuated inversion recovery (FLAIR) hyperintensity in the right optic nerve [arrow in Figure 1a], along with multiple rounded lesions in the bilateral medial temporal lobe,

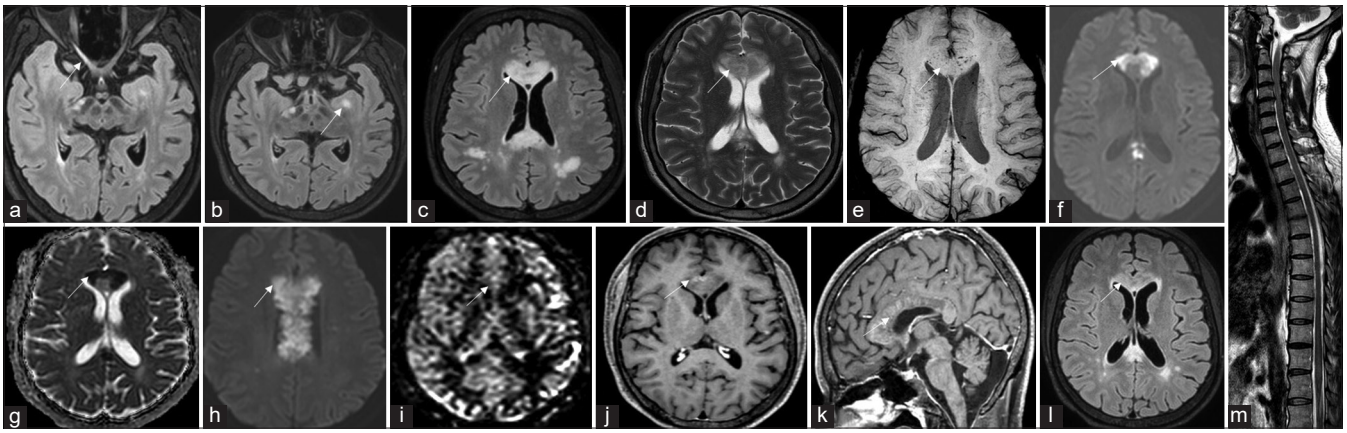

Figure 1: 34-year-old male presented with the right optic neuritis and pyramidal signs as described above, magnetic resonance imaging (MRI) acquisition with axial reformats reveals fluid-attenuated inversion recovery (FLAIR) hyperintensity in the right optic nerve (arrow in a), multiple rounded lesions in the bilateral medial temporal lobe and bilateral parietal lobe (arrow in b). Diffuse, expansile T2-FLAIR hyperintensity (arrow in c and d) is noted involving genu, body, and splenium of corpus callosum. The lesion shows foci of microhemorrhages as blooming on susceptibility weighted imaging (arrow in e), along with central facilitated diffusion (red arrow in f and g) and peripheral restriction in the corpus callosum lesion (white arrow in f-h). Base image of arterial spin labelling shows no raised perfusion (arrow in i). Patchy peripheral enhancement is seen on post-contrast images (arrow in j and k). Follow-up MRI Scan after 1 month reveals significant resolution in the corpus callosum lesion. Whole spine MRI screening T2 acquisition in sagittal reformat (arrow in l) (m) reveals no abnormal signal hyperintensity in the cord.

right internal capsule and corticospinal tract at midbrain, and bilateral parietal lobe [arrow in Figure 1b]. Diffuse, expansile T2-FLAIR hyperintensity [arrow in Figure 1c and d] is noted involving genu, body, and splenium of corpus callosum. The lesion shows foci of microhemmorhages as blooming on susceptibility-weighted imaging (SWI) [arrow in Figure 1e], along with central facilitated diffusion [red arrow in Figure 1f and g] and peripheral restriction in the corpus callosum lesion [white arrow in Figure 1f-h]. Base image of arterial spin labelling shows no raised perfusion [arrow in Figure 1i]. Patchy peripheral enhancement is seen on post-contrast images [arrow in Figure 1j and k]. Follow-up MRI scan after 1 months reveals significant but partial resolution in the Figure 1l corpus callosum lesion. Whole spine MRI screening T2 acquisition in sagittal reformat [Figure 1m] revealed no abnormal signal hyperintensity in the cord.

DISCUSSION

Tumefactive demyelination often presents with radiological features that resemble primary brain neoplasms, frequently necessitating brain biopsy for accurate diagnosis. These large lesions exhibit mild mass effects and surrounding edema. They are commonly associated with various conditions such as multiple sclerosis, neuromyelitis optica spectrum disorder, Baló concentric sclerosis, myelinoclastic diffuse sclerosis (Schilder disease), acute disseminated encephalomyelitis, acute hemorrhagic leukoencephalitis, and autoimmune-mediated encephalitis.[5] In our case, clinical features such as optic neuritis and on-off symptoms support a demyelinating etiology. Involvement of the right corticospinal tract at the right midbrain (cerebral peduncle) and in the right internal capsule (not shown) explaining the left upper motor neuron facial palsy. The imaging findings revealed a large lesion with restricted diffusion, crossing the midline and involving the corpus callosum, and mimicking lymphoma, metachromatic leukodystrophy, and glioma. However, certain imaging features, such as incomplete open ring enhancement, mild effect, mild or absent perilesional edema, and peripheral incomplete diffusion restriction in the corpus callosum lesion, along with the presence of optic neuritis and other lesions, provide clues suggesting a demyelinating disease.[6] Therefore, it is essential to differentiate callosal lesions from other tumor mimics based on typical imaging characteristics and clinical history. [Table 1] is describing the corpus callosum lesions which show the diffusion restriction. Their differential features are described in [Table 1]. The interval image also shows the regression of lesion support over-diagnosis of tumefactive demyelination.

Table 1: The lesions which involve the corpus callosum and show diffusion restrictions.

Diffusion restricting lesions of corpus callosum	
Tumefactive demyelinating lesion	Peripherally restricting, peripherally enhancing lesion
Lymphoma	Homogenously enhancing and homogenously restricting lesion
Glioblastoma	Irregular diffusion restriction
Cytotoxic lesion of corpus callosum	Transient, focal diffusion restricting lesion

CONCLUSION

Tumefactive demyelination can mimic an intracranial mass lesion, leading to potential misdiagnosis. This case report serves as an illustration of how radiological characteristics can assist in accurate diagnosis, thus preventing unnecessary brain biopsy or aggressive treatments that could be harmful to the patient.

TEACHING POINTS

Demyelinating lesions have a peripheral open ring type of (toward gray matter) diffusion restriction and contrast enhancement. Tumefactive demyelination lesions have normal perfusion and minimal edema as compared with mass lesions which have high perfusion, mass effect, and significant edema.

DIFFERENTIAL DIAGNOSIS

Tumefactive demyelination, high-grade gioma, lymphoma, and metachromatic leuckodystrophy.

MCQs

1. Which is the best imaging sequence to differential tumefactive demyelination and tumor
    a. DSC perfusion
    b. DWI
    c. T1 FS+C
    d. SWI

Answer Key: a

2. Lesion crossing midline
    a. Lymphoma
    b. Glioma
    c. Tumefactive demyelination
    d. All

Answer Key: d

3. Which is best option to confirm demyelination etiology from tumor
    a. Clinical history of optic neuritis
    b. SWI
    c. DWI
    d. Mass effect

Answer Key: a

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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