CASE REPORT

An unusual case of optic and chiasmal neuritis caused by hypersensitivity to mycobacterium: A case report

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Abstract

We describe Magnetic Resonance Imaging (MRI) findings in a 49-years-old female who presented with subacute visual loss. MRI findings were suggestive of optic neuritis and chiasmal neuritis. She had necrotic mediastinal lymphadenopathy which was diagnosed as tuberculosis. Optic neuritis and chiasmal neuritis in this case were most probably due to hypersensitivity to mycobacterium. To the best of authors' knowledge, these unusual MRI findings of optic and chiasmal neuritis caused by hypersensitivity to mycobacterium have not been previously reported.

Keywords: Optic Neuritis, Chiasmal Neuritis, Tuberculosis, Hypersensitivity to Mycobacterium, Magnetic Resonance Imaging

Introduction

Optic neuropathy is a broad term that refers to damage inflicted on the optic nerve due to various congenital or acquired pathologies. The term optic neuritis is used when inflammation is the pathophysiologic basis of optic neuropathy [1]. In this case report, a 49-year-old female was found to have optic neuritis, chiasmal neuritis and necrotic mediastinal lymphadenopathy. Mediastinal lymphadenopathy was proved to be tuberculous lymphadenitis. Since there was no evidence of direct involvement of optic nerves and chiasm by mycobacterium, the optic neuritis and chiasmitis was assumed to be due to hypersensitivity to mycobacterium.

Case Report

A 49-year-old female presented with gradual loss of vision in both eyes (right more than left), bilateral peri-orbital pain and low-grade fever since 15 days. She also complained of intermittent non-focal headache and breathlessness. Breathlessness was Modified Medical Research Council (MMRC) Grade I. General examination was unremarkable. On ophthalmic examination, visual acuity was 6/36 in right eye and 6/18 in left eye. Relative afferent pupillary defect was present on right side. Near visual acuity was N18 in right eye and N8 in left eye. Color vision in the bilateral eyes was intact. Contrast enhanced Magnetic Resonance Imaging (MRI) of brain and orbits revealed thickening of intracranial portions of bilateral optic nerves and optic chiasm (Figure 1). No post contrast enhancement was noted in optic nerves and optic chiasm (Figure 2). No obvious meningeal enhancement was noted. Brain was unremarkable.

Frontal chest radiograph revealed bilateral hilar prominence. Positron Emission Tomography – Computed Tomography (PET-CT)study revealed necrotic mediastinal (prevascular, pretracheal, bilateral hilar, subcarinal, paraesophageal) lymph nodes with 18F-Fluorodeoxyglucose (FDG) avidity (Figure 3).

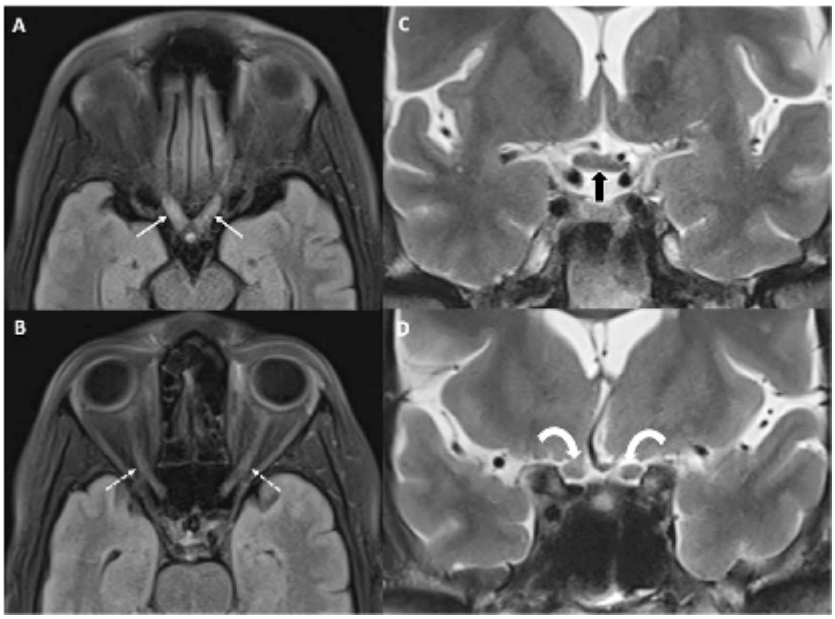

Figure 1: MRI brain FLAIR axial images (Figures 1A and 1B) showing thickened hyperintense intracranial portions of bilateral optic nerves (arrows) and while intra-canalicular and intra-orbital portions are not thickened (dotted arrows). Coronal T2-weighted images (Figures 1C and 1D) revealed thickened optic chiasm (black arrow) and intracranial portions of bilateral optic nerves (curved arrows).

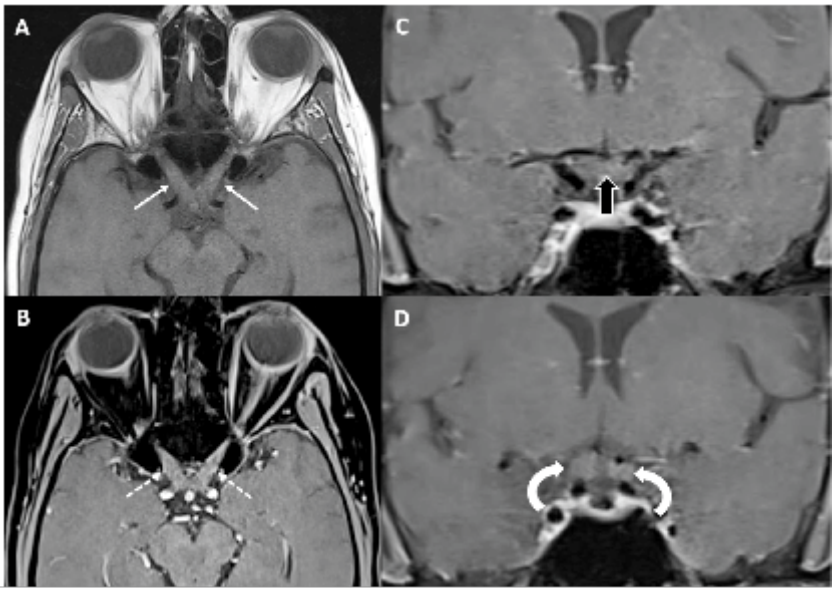

Figure 2: Non-contrast MRI brain T1-weighted axial image (Figure 2A) showing thickened intracranial portions of bilateral optic nerves (arrows). Post-contrast axial (Figure 2B) and coronal (Figures 2C and 2D) T1-weighted images showing no enhancement of involved portions of optic nerves (dotted arrows and curved arrows) and optic chiasm (black arrow).

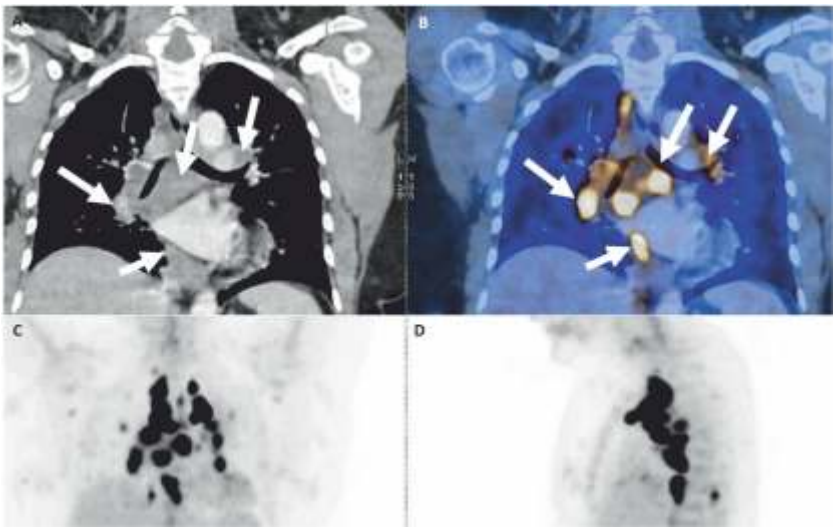

Figure 3: Coronal reformatted post-contrast CT chest image (Figure 3A) showing necrotic mediastinal lymphadenopathy (arrows). Corresponding FDG-PET-CT chest fusion image (Figure 3B) showing FDG uptake in mediastinal lymph nodes. FDG-PET images (Figures 3C and 3D) showing FDG uptake in mediastinal lymph nodes.

Patient further underwent endoscopic ultrasound guided biopsy from necrotic subcarinal lymph node. Histopathological report revealed multiple cores of fibro-collagenous tissue, extensive caseous necrosis with ill-defined aggregates of epithelioid histiocytes, occasional multinucleate giant cells and many lymphocytes forming granulomas – suggestive of necrotizing granulomatous lymphadenitis. Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) test of the biopsied lymph node was positive suggestive of tuberculous lymphadenitis.

Lumbar puncture revealed turbid Cerebrospinal Fluid (CSF). Cobweb/coagulum was absent. Raised leucocyte count (predominantly lymphocytes) and protein level were noted in CSF with normal glucose. No micro-organism was seen on gram stain. Acid fast bacilli were absent. CSF CBNAAT was negative. Culture of CSF showed no growth of micro-organism.

Serum Aquaporin-4 (AQP4) antibodies, Myelin Oligodendrocyte Glycoprotein (MOG), Anti-nuclear Antibody (ANA), anti-double stranded DNA (anti-dsDNA), Rhuematoid Factor (RF) tests were negative. Mitochondrial DNA sequencing for Leber's Hereditary Optic Neuropathy (LHON) was also negative.

Optic neuritis and chiasmal neuritis was presumed to be secondary to hypersensitivity reaction to mycobacterium in view of tuberculous necrotizing mediastinal lymphadenopathy, negative serological tests for other causes of optic neuritis and lack of evidence of CSF involvement by mycobacterium.

Patient was started on anti-tubercular therapy (Isoniazid, Rifampicin, and Pyrazinamide). Intravenous methylprednisolone 1 g daily was given for 5 days, followed by tapering dose. After a week, there was improvement in the vision of both the eyes (visual acuity: 6/12 in right eye and 6/9 in left eye). Complete resolution of ophthalmic symptoms was noted after 3 months.

Discussion

Optic neuritis is divided into typical and atypical types. Typical optic neuritis includes demyelination which is usually associated with multiple sclerosis whereas atypical optic neuritis includes infections, connective tissue diseases, vasculitis, sarcoidosis etc. [2]. Infective causes of optic neuritis include Lyme's disease, toxoplasmosis, human immunodeficiency virus, tuberculosis etc. Optic neuritis in Tuberculosis (TB) may be caused by direct infection by mycobacterium, contiguous spread from choroid, hematogenous spread or hypersensitivity to mycobacterium [3]. Tuberculous neuroretinitis and retrobulbaroptic neuritis are less common than papillitis [4].

Usual MRI features of optic neuritis in TB include retrobulbar optic nerve hyperintensity on T2-weighted images with nerve and/or sheath enhancement. Leptomeningeal enhancement, ependymitis and tuberculoma can be seen as additional features related to involvement of brain by tubercular infection [5-6]. Apart from optic neuritis, vision loss from tuberculosis may also result from tubercular meningitis with hydrocephalus, arachnoiditis of optic chiasm, or tuberculomas compressing either optic nerve or optic chiasm [7]. In our case, there was symmetrical thickening of only the intracranial portions of bilateral optic nerves and optic chiasm without post contrast enhancement and without meningeal enhancement. Preferential involvement of posterior portions of optic nerves can be seen in Neuromyelitis Optica Spectrum Disorder (NMOSD) [8] and Leber's Hereditary Optic Neuropathy (LHON) [9]. Enhancement of the optic nerves is noted in NMOSD while no enhancement is a feature of LHON [8-9]. Our case closely resembles the imaging pattern of LHON. Absent enhancement of optic nerves in optic neuritis is uncommon and has been reported [10]; however, hypersensitivity to mycobacterium as a cause is not yet reported in the literature. Optic neuritis and chiasmitis as the presenting feature in an individual having tuberculous lymphadenitis, as in our case, is very unusual and is scarcely reported in literature [11]. In our case significant improvement in patient's symptoms was observed after anti-tuberculous medication and corticosteroid.

Thickening of intracranial portions of bilateral optic nerves and optic chiasm without post-contrast enhancement is an unusual imaging feature of optic neuritis and chiasmitis secondary to hypersensitivity to mycobacterium. Also, optic neuritis as a presenting manifestation in TB, which is uncommon, in our case makes it unique.

Conclusion

To conclude, optic neuritis secondary to hypersensitivity to mycobacterium might be considered as one of the differential diagnosis of thickened optic nerves with absent postcontrast enhancement, especially in individuals from endemic areas.

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