Transient Tunnel Vision as Initial Presentation of Anti-MOG Antibody Positive Optic Neuritis

To the Editor,

Myelin oligodendrocyte glycoprotein (MOG) is an essential component of the oligodendrocytes and is expressed in the myelin of the central nervous system (CNS). Optic neuritis (ON) and acute disseminated encephalomyelitis are inflammatory demyelinating disorders that been associated with anti-MOG antibodies.[1] We describe transient monocular tunnel vision, as a novel clinical presentation of anti-MOG antibody associated ON.

A 21-year-old nursing student presented with complaints of painful ocular movements since 2 weeks, followed by transient episodes of tunnel vision involving the left eye. She first noticed tunneling of her vision in the left eye during eye movement and noted that it was associated with peri-ocular pain. She noticed that her field of vision in the left eye had reduced to one third her normal and was able to see central objects such as faces and read text from her mobile phone, but was unable to see peripherally. The tunnel vision was paroxysmal, lasting about 20–30 minutes, resolving spontaneously but recurring every few hours. At this point, there were no symptoms in the right eye. Since 4 days after the onset of transient tunnel vision, she also developed blurring of vision, which was more prominent in the right eye. She also reported decreased brightness and impaired color vision in both eyes. On ocular examination her visual acuity in right eye was 6/18 and left eye was 6/9 and there was relative afferent pupil defect in the right eye. Fundus examination showed bilateral disc edema. The visual filed appeared normal on confrontation and there was loss of color vision in both eyes. Clinical localization was to the anterior visual pathway. Provisional diagnosis of an atypical optic neuritis was considered. MRI Brain with orbits showed thickening and enhancement of the optic nerves and nerve sheath (right >left) without any other white matter lesions in the brain [Figure 1]. Visual evoked potentials showed bilateral anterior optic pathway dysfunction. CSF analysis was normal (cells-3 glucose-53 mg/dl protien-32 mg/dl). Serum angiotensin converting enzyme (ACE) levels were in normal range. ESR, CRP, and antinuclear cytoplasmic antibodies was negative, connective tissue work up was negative. She was found to be negative for Aquaporin-4 antibodies but positive for serum myelin oligodendrocyte glycoprotein (MOG) antibody. She was given pulse therapy of Inj. Methylprednisolone pulse therapy 1 gm for 5 days, following which her visual symptoms improved dramatically

on the second d day and her visual acuity improved to 6/6 in both eyes and color and contrast perception improved to normal. She was started on a tapering schedule of steroids and azathioprine.

Our patient presented with an unusual symptom of recurrent episodes of monocular tunnel vision followed by bilateral optic neuritis. This is the first report of transient episodes of a peripheral scotoma as a harbinger of MOG antibody positive optic neuritis. Optic neuritis typically presents with periocular pain and a central scotoma and a peripheral scotoma in ON is uncommon. The central 5 to 10° of visual field which is subserved by the papillomacular bundle is centrally placed in the optic nerve and consists of small, closely packed fibers that are likely to have a high metabolic demand. Inflammatory diseases of optic nerve such as optic neuritis, toxins such as methanol, carbon disulphide, and nutritional deficiencies such as B12 deficiency typically manifest as a central scotoma and show degeneration of the "papillomacular bundle."[2] Tunnel vision is typically described in posterior visual loss involving the occipital cortex.[3] However, monocular peripheral scotomas can commonly be seen as a consequence of papilledema and rarely in optic perineuritis (OPN). Optic perineuritis is an inflammatory disorder affecting the optic nerve sheath characterized by periocular pain and optic nerve sheath enhancement on MRI. OPN is known to be a presentation of infections such as syphilis and inflammatory disorders such as sarcoidosis and in most cases, it remains idiopathic. Recently anti-MOG antibodies have been associated with idiopathic OPN and peripheral scotomas have been reported in patients with Anti-MOG associated OPN.[4] Also optic nerve sheath enhancement can be a prominent feature of MOG-IgG associated ON and is present in up to 50% of patients with MOG-IgG positive ON.[5] Thus concomitant presence of optic perineuritis in anti-MOG antibody associated optic neuritis is known.[5] We postulate that our patient's symptoms started as optic perineuritis initially and then went on to develop an optic neuritis. The initial symptom of tunnel vision was due to optic perineuritis and the subsequent symptoms that developed were due to bilateral optic neuritis. This hypothesis is supported by the enhancement of the optic nerve and the nerve sheath in the MRI of our patient. Transient mono-ocular visual loss has been described in vascular aetiologies such as amaurosis fugax and retinal migraine, and in transient visual obscuration of raised intracranial pressure.[6] We postulate that in our patient the optic perineuritis which predated the optic neuritis is responsible for the transient episodes of peripheral visual loss. The role anti-MOG antibodies in the pathogenesis of OPN are currently not known. Extension of the inflammation from the optic nerve to the nerve sheath and focal meningitis mediated by other unknown antigens are postulated mechanisms of OPN.[4] However, the mechanism for the transient nature of the symptoms is currently un-explained. We wish to highlight, that transient episodes of a peripheral scotoma can rarely be a harbinger of MOG antibody positive optic neuritis.

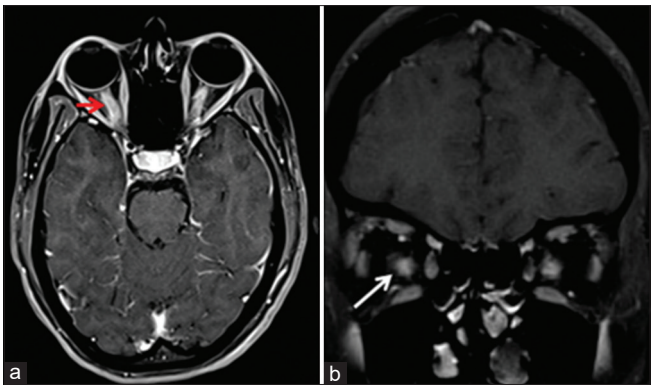

Figure 1: Shows post gadolinium T1 fat suppressed images of the patient with transient tunnel vision and optic neuritis. (a) Shows axial images of the brain and orbit with the red arrows pointing to the enhancement of the right optic nerve and nerve sheath. (b) Shows coronal images through the brain and orbit with the white arrow pointing to the enhancement of the right optic nerve

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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