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Recurrent Optic Neuritis - An Atypical Case Report

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Abstract: ***Introduction: Optic neuritis is an acute Inflammation of the optic nerve[1]. It is characterized by temporary vision loss, periorbital pain, defective colour vision. The various causes of optic neuritis can be infections, ischemic, infiltrative or compressive, demyelinating disorders[1]. It typically develops in young adult between the age group of 20-40 years. Some of the cases can be recurrent, unilateral or bilateral presentation[2]. Case Presentation: A 28 year old male presented to our hospital with complaints of defective vision in the left eye for 3 days. He had a previous history of similar complaints in the right eye 5 years back in 2017 for which MRI Brain was done and revealed no significant findings and has been treated with corticosteroids. The initial examination revealed 6/60 vision in the left eye, with pupil showing grade 1 relative afferent pupillary defect (RAPD) and defective colour vision. Further investigations were done and MRI brain showed features characteristic of demyelinating disorder – multiple sclerosis. Conclusion: This case is reported for its atypical presentation in the right eye 5yrs back which was diagnosed as retrobulbar neuritis with normal MRI findings following which recurrence occurred in the contralateral eye, left eye recently with MRI findings suggestive of demyelinating disorder, which was then diagnosed to be a case of multiple sclerosis[3].***

Keywords: Recurrent optic neuritis, Multiple sclerosis, Demyelination, Corticosteroids, Case Report

1. Introduction

Optic neuritis is a demyelinating inflammatory disorder of the optic nerve. An isolated optic neuritis occurs as a unilateral, subacute, and painful loss of vision[4]. It can also have bilateral or recurrent presentation[5]. Diagnosis of a recurrent cases may be challenging. The term bilateral (BON) optic neuritis is defined if both eyes were involved simultaneously within 3 week and Recurrent optic neuritis if a new unilateral attack occurs after an interval of more than 4 weeks. Acute idiopathic optic neuritis is the most common cause of optic neuropathy in young adults[10]. It is an isolated inflammatory optic neuropathy secondary to demyelination and is a clinically isolated syndrome suggestive of multiple sclerosis (MS).MS is one of the major causes of recurrent unilateral ON.

Clinical Findings

A 28year old male presented to our OPD with complaints of defective vision in the left eye for the past 3 days. He had history of defective vision in the right eye since 2017 and was diagnosed as right eye retro bulbar neuritis in an outside hospital. MRI findings taken at that time was normal and was given 3 doses of IV methyl prednisolone under ONTT (optic neuritis treatment trial)[6]. He had normal bowel and bladder habits, normal sleep pattern and no history of substance abuse. There was no significant family history. Facial symmetry was normal. Both eyes were orthophoric. Extraocular movements were full. Visual acuity in right eye was 6/6 and Anterior segment was normal. On examining, right eye fundus was normal. Left eye, visual acuity was 6/60 with pupil showing grade 1 RAPD. Rest of the anterior segment was normal. In left eye, Fundus Media clear, Disc 0.3:1 CDR, margins defined, mild hyperemia, vessels normal, macula FR+. All routine investigations were done. RBS:81mg/dl, CREATININE:0.7mg/dl, HBA1C:5.27%, CBC: NORMAL, TFT: NORMAL. Lumbar puncture with CSF analysis was done. CSF fluid for AFB: negative. CSF fluid for glucose: 67mg/dl (normal: 45-80mg/dl). CSF fluid for protein: 91mg/dl (normal: 20-40mg/dl). MRI BRAIN WITH ORBITS (25/04/2022) Multiple punctate and discrete T2/FLAIR hyper intensities in the B/L thalamocapsular region, right temporal lobe, left mid brain, anterior pons, right middle cerebellar peduncles, left cerebellum, periventricular and subcortical white matter region and collossa septal interface. Few faint patches of hyper intensities seen in the visualized upper cervical spinal cord. Few lesions in periventricular white matter show diffusion restriction. Few T1 black holes in the periventricular region. Left optic nerve appears thickened and faint hyperintensity. Findings together suggestive of multiple sclerosis. VEP TEST- Within normal limits. Anti-nuclear antibodies: negative. He was treated with Intravenous methyl prednisolone for 3 days with reduced salt and water intake. Oral WYSOLONE 60mgwas started for 5days and then tapered over and stopped. visual acuity after 1 week both eyes: 6/6

2. Discussion

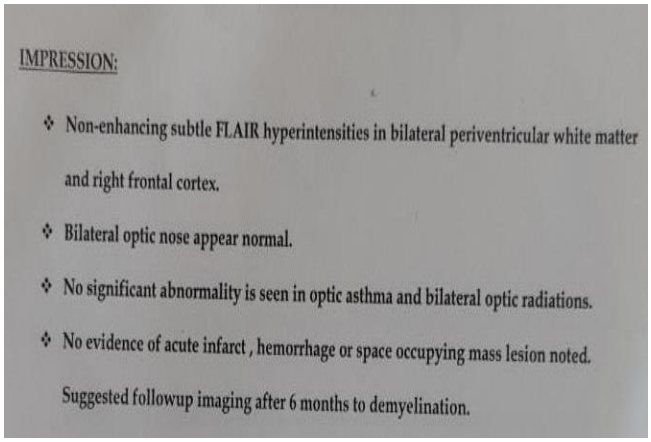
Optic neuritis (ON) is an acute inflammatory demyelinating disorder of the optic nerve, multiple sclerosis being the most common demyelination disorder. The typical characteristics of ON include unilateral, subacute, and painful visual loss without systemic or other neurological symptoms and ON is mostly seen in young females. Relapses and recurrence are one of the long-term features of ON but initial presentation is usually monophasic. Atypical features of ON being painless, complete loss of vision, gross colour vision defect and optic atrophy at the time of presentation. The etiology of ON may include demyelination, infections, immunological, traumatic, toxin exposures. Idiopathic ON where the cause of ON is not known still is the largest etiological factor. Second most common cause of ON is demyelination disorders namely Multiple sclerosis [MS], neuromyelitis optica[NMO], clinical isolated syndrome[CIS].

In this article, patient has presented with history of left eye defective vision with pain and no diplopia in the present episode and similar history in right eye in 2017 following initial monophasic presentation and complete recovery of

vision from the first episode. At present the symptoms are in other eye(left eye) and was more intense than the previous episode. On further examination, there was RAPD, full extra ocular movements, mild colour vision defect and fundus showing mild hyperemia. Other investigations like CSF analysis – helps in ruling out infective etiology, MRI – helps in establishing demyelination etiology (in our case, shows typical MS features), VEP – features like increased latency and reduced amplitudes helps in deciding on treatment, prognostication and risk of conversion to MS . Other specific tests like NMO and Aquaporin 4 tests helps in further specifying the type of demyelination. MRI imaging not only helps in identifying the demyelination etiology but also helps in differentiating the type of demyelination between MS ON and NMO ON, where in NMO ON shows minimum of 3 longitudinal demyelination patches and MS ON shows more focal loss and seen in more anterior part of optic nerve. VEP features like abnormal waves helps in identifying the high risk cases for conversion to MS in setting of first episode of ON with normal MRI imaging. Further assay for Ig G index helps as predictor for MS conversion. OCT is a non invasive and easily available investigation to assess RNFL thickness and to identify the old episodes of ON. B scan ultrasonography helps in documentation of ON like thickening of ON. After initial treatment according to ONTT trial, patient had visual acuity improvement and symptomatic relief. Patient was further kept under periodic reviews and neurological assessments for further treatment planning.

3. Conclusion

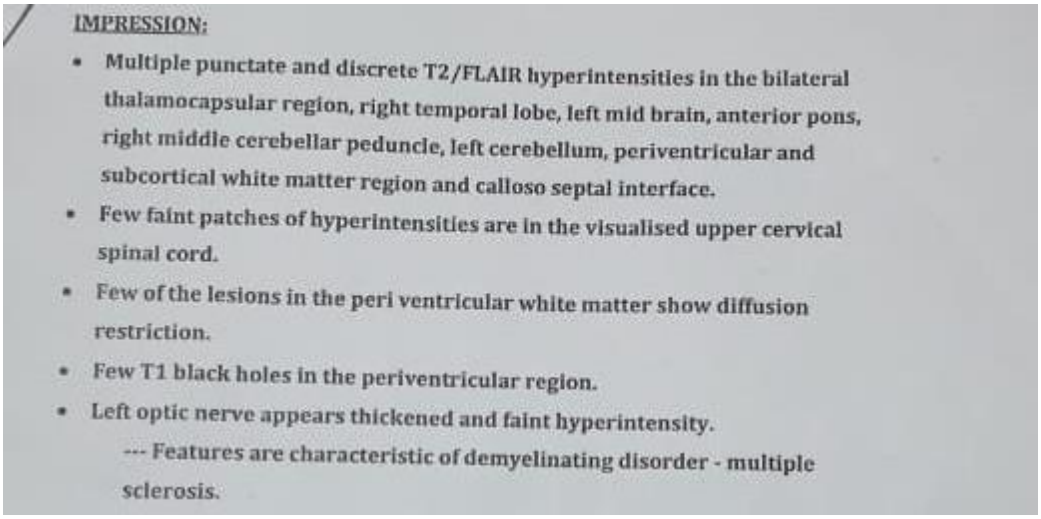
This case is reported for its atypical presentation with initial findings of optic neuritis in right eye and normal MRI findings and recurrence of optic neuritis occurring within 5 years in the contralateral eye i.e., left eye and MRI findings suggestive of demyelination [6]. Even though the most common initial clinical presentation of optic neuritis is without a previous demyelinating event, during the course of the disease demyelination can occur at any time frame. Once acute management of optic neuritis is done as per the standard ONTT trial, the rate of conversion to multiple sclerosis with demyelination events should be monitored in these cases constantly at regular intervals irrespective of clinical symptoms[7][8]. Serial MRI screenings helps to predict the risk of developing MS. Management strategy for these type of presentations in young adults should include acute and long term therapies and follow up should be included to watch for recurrent attacks and prevent further damage of the optic nerve head. Multidisciplinary approach should be employed involving ophthalmologist, neurologist and rheumatologists for a holistic approach in monitoring these cases[9,10].


IMPRESSION:

- Non-enhancing subtle FLAIR hyperintensities in bilateral periventricular white matter and right frontal cortex.
- Bilateral optic nose appear normal.
- No significant abnormality is seen in optic asthma and bilateral optic radiations.
- No evidence of acute infarct , hemorrhage or space occupying mass lesion noted.

Suggested followup imaging after 6 months to demyelination.

Figure 1: MRI Brain Report (2017)


IMPRESSION:

- Multiple punctate and discrete T2/FLAIR hyperintensities in the bilateral thalamocapsular region, right temporal lobe, left mid brain, anterior pons, right middle cerebellar peduncle, left cerebellum, periventricular and subcortical white matter region and calloso septal interface.
- Few faint patches of hyperintensities are in the visualised upper cervical spinal cord.
- Few of the lesions in the peri ventricular white matter show diffusion restriction.
- Few T1 black holes in the periventricular region.
- Left optic nerve appears thickened and faint hyperintensity.

--- Features are characteristic of demyelinating disorder - multiple sclerosis.

Figure 2: MRI Brain Report (2021)

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