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Non-Arteritic Anterior Ischemic Optic Neuropathy In A Young Male: A Case Report

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Abstract
Non-arteritic anterior ischemic optic neuropathy (NAION) is the most common acute optic neuropathy in older adults and is rarely encountered in young individuals. We report a case of a young male presenting with painless diminution of vision in the right eye associated with relative afferent pupillary defect (RAPD), optic disc edema, and characteristic visual field defects. Fundus examination revealed right optic disc edema and left superior sectoral disc pallor. Visual field analysis demonstrated generalized depression in the right eye and inferior altitudinal scotoma in the left eye. Follow-up after 3 months showed complete resolution of disc edema with persistent visual field defects and residual optic disc pallor, supporting the diagnosis of NAION. This case highlights the occurrence of NAION in a young patient and emphasizes the importance of differentiating NAION from inflammatory, hereditary, toxic, and compressive optic neuropathies.

Keywords: *NAION, optic disc edema, ischemic optic neuropathy, RAPD, altitudinal scotoma*

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I. Introduction

Non-arteritic anterior ischemic optic neuropathy (NAION) is the most common acute optic neuropathy occurring in middle-aged and elderly individuals and results from ischemia of the optic nerve head supplied by the short posterior ciliary arteries.[1,2] It typically presents with sudden painless diminution of vision associated with optic disc edema and characteristic visual field defects, most commonly altitudinal defects.[3]

The condition is commonly associated with systemic vascular risk factors such as diabetes mellitus, hypertension, dyslipidemia, nocturnal hypotension, and obstructive sleep apnea.[4,5] NAION occurring in young adults is uncommon and often creates a diagnostic dilemma due to overlapping features with optic neuritis, hereditary optic neuropathies, toxic-nutritional optic neuropathy, and compressive lesions.[6]

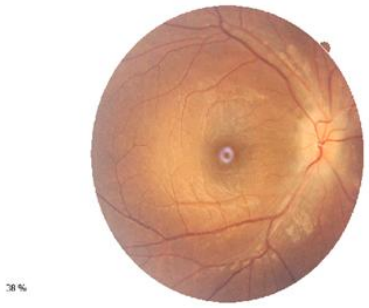
We report a case of a young male presenting with unilateral optic disc edema, relative afferent pupillary defect, and sequential bilateral visual field defects, eventually diagnosed as non-arteritic anterior ischemic optic neuropathy following clinical evaluation and follow-up.

II. Case Presentation

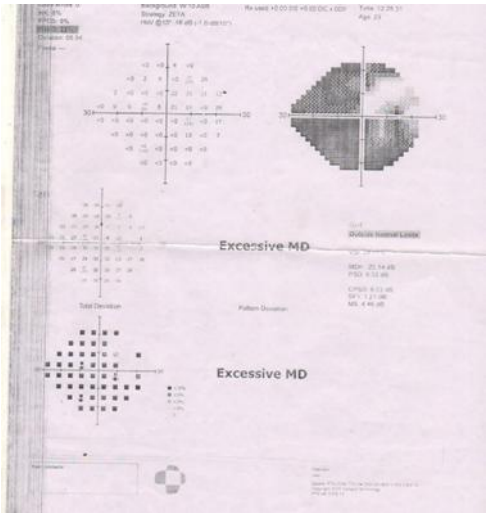
A young male 30 years old patient presented with complaints of sudden painless diminution of vision in the right eye . There was no history of ocular pain, redness, trauma, diplopia, transient visual obscurations, or headache. There was no significant history of diabetes mellitus, hypertension, or other systemic illness.He was also chronic alcoholic.

On ophthalmic examination, best corrected visual acuity (BCVA) was 6/12 in the right eye and 6/9 in the left eye.

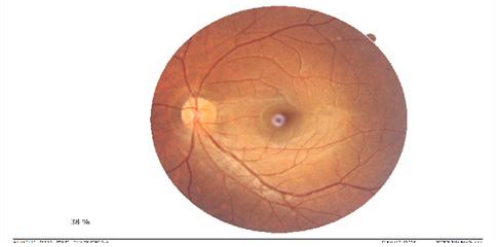
Pupillary examination revealed a relative afferent pupillary defect (RAPD) in the right eye. Extraocular movements were full and painless. Anterior segment examination was unremarkable in both eyes.



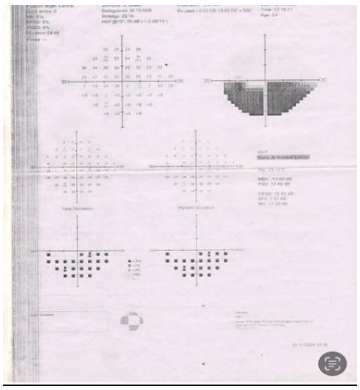
Right eye fundus pic and fields at present



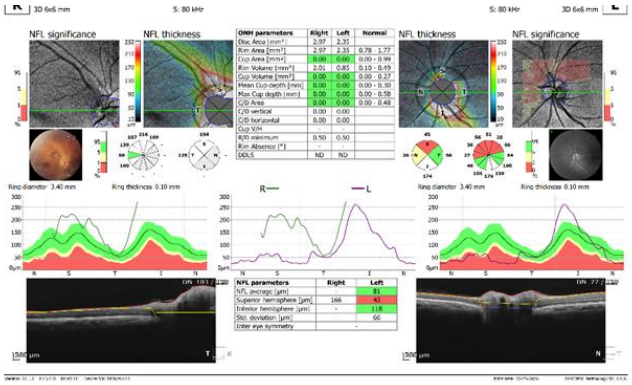
Left eye fundus pic, oct and fields at present



OCT -DISC of both eyes at present



Fundus examination showed hyperemic optic disc edema in the right eye, while the left eye showed mild superior sectoral optic disc pallor.



Visual field examination revealed generalized depression of fields in the right eye and inferior altitudinal scotoma in the left eye.

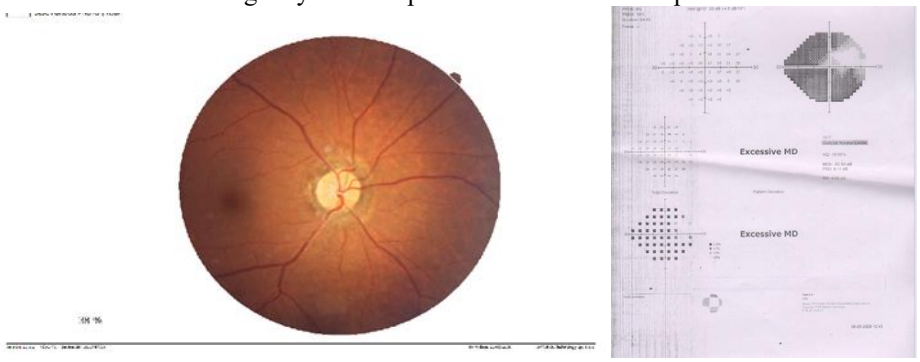
Color vision was mildly reduced in the right eye.

Routine systemic investigations including complete blood count, blood sugar profile, lipid profile, ESR, CRP, serum Vitamin B12 levels and mitochondrial DNA mutation analysis were advised to exclude inflammatory and nutritional causes. Neuroimaging was performed to rule out compressive and demyelinating lesions and was not suggestive of alternative pathology.

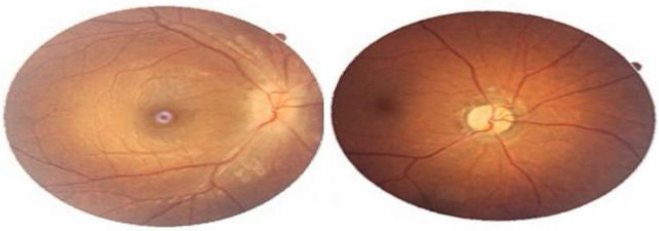
The patient was initially treated with intravenous methylprednisolone (IVMP) 1 g diluted in 500 mL of 5% dextrose solution once daily for three consecutive days. Following intravenous therapy, oral prednisolone (Wysolone) 60 mg daily was started for 10 days and gradually tapered thereafter. Topical nepafenac 0.1% eye drops (Micronec) were also prescribed.

The patient was followed up after three months. At follow-up, visual acuity remained stable at 6/12 in the right eye and 6/9 in the left eye. Visual field defects persisted without progression. Fundus examination showed complete resolution of right optic disc edema with development of mild optic disc pallor. Left eye findings remained stable with superior sectoral pallor and field defects remained same.

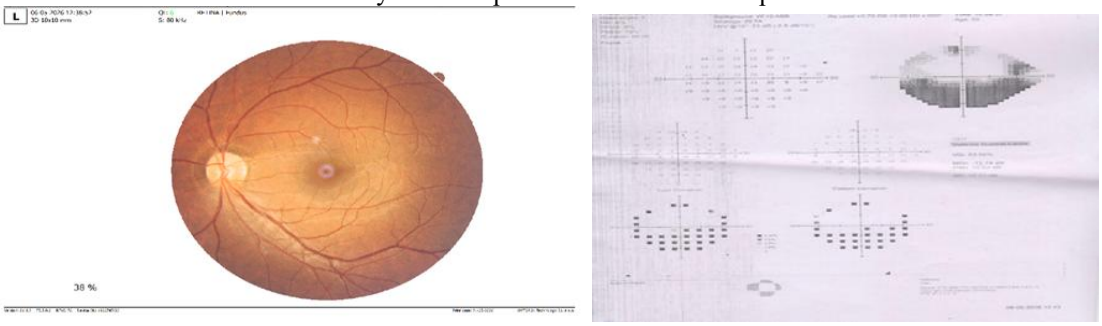
Right eye fundus pic and fields at follow up



Comparison of fundus photo of right eye after 3 months



Left eye Fundus pic and fields at follow up



Based on the clinical presentation, visual field defects, evolution of optic disc changes, and exclusion of other causes of optic neuropathy, a diagnosis of non-arteritic anterior ischemic optic neuropathy (NAION) was made.

III. Discussion

NAION is typically a disorder of older adults and is infrequently seen in younger patients. Diagnosis in young individuals requires careful exclusion of optic neuritis, compressive optic neuropathy, Leber hereditary optic neuropathy (LHON), and toxic-nutritional optic neuropathy.[6]

The characteristic features supporting NAION in our patient included painless diminution of vision, RAPD, optic disc edema followed by disc pallor, and persistent visual field defects. The presence of altitudinal visual field defect strongly favored ischemic optic neuropathy.

Optic neuritis was considered less likely because of absence of pain on eye movements, lack of significant visual recovery, and persistence of visual field defects over follow-up. Toxic-nutritional optic neuropathy generally presents with bilateral symmetrical central or centrocecal scotomas rather than altitudinal defects. LHON usually presents with bilateral severe central visual loss and temporal disc changes in young males.

Despite systemic corticosteroid therapy, visual acuity and field defects remained largely unchanged, although optic disc edema resolved during follow-up.

Resolution of optic disc edema with subsequent optic atrophy over time further supported the diagnosis of NAION.

IV. Conclusion

NAION should be considered in the differential diagnosis of young patients presenting with painless visual loss, RAPD, optic disc edema, and altitudinal field defects even in the absence of classical vascular risk factors. Careful follow-up and exclusion of other causes of optic neuropathy are essential for accurate diagnosis.

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