Case Report

Rapid recovery of severe altitudinal visual field defects in a case of acute optic neuritis without significant optic nerve head edema

Abstract

Background: Optic neuritis (ON) is an acute inflammatory demyelinating optic neuropathy that typically presents with subacute, painful vision loss, and an afferent pupillary defect. Although most cases occur in younger adults and are associated with multiple sclerosis (MS), ON can present atypically—by age, field pattern, or severity—and overlap with ischemic and inflammatory mimickers in patient's ≥50 years old. Altitudinal visual field defects are classically linked to non-arteritic anterior ischemic optic neuropathy (NAION), yet they may occur in ON and can misdirect early management. Rapid, high-dose corticosteroid therapy accelerates visual recovery in typical ON, while urgent exclusion of giant cell arteritis (GCA) and other non-demyelinating etiologies remains essential in older adults.

Case report: A 52-year-old Caucasian male presented with sudden blurry and jumping vision in the upper half of his left eye. Visual acuity was mildly impaired OS, but his visual field (VF) showed deep superior altitudinal defects. MRI orbits and brain with and without contrast showed an increased T2 signal and enhancement of the intra-orbital segment of the left optic nerve, compatible with optic neuritis. Several periventricular and subcortical white matter FLAIR/T2 hyperintensity. Cerebral spinal fluid chemistry confirmed the presence of oligoclonal band (IgG) with a high synthesis rate. The patient was referred to neurology for further management and put on 80 mg/d prednisone with taper schedule. Altitudinal VF defects were resolved almost completely after a month. Further workup by neurologist confirmed MS diagnosis and he is being managed by neurologist and rheumatologist.

Conclusion: This case highlights an atypical presentation of ON in a mid-life patient with a deep, but transient, superior altitudinal defects. After extensive neurological and laboratory work ups, MS was the definitive diagnosis. Sudden vision loss and optic neuropathy require a interprofessional team including internist, optometrist, radiologist, neurologist and rheumatologist for timely and effective management.

Keywords: optic neuritis, multiple sclerosis, altitudinal visual field defect, MRI, demyelinating disorder

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Abbreviations: VF, visual field; GCA, giant cell arteritis; ON, Optic neuritis; NAION, non-arteritic anterior ischemic optic neuropathy; MS, multiple sclerosis; CSF, cerebrospinal fluid; MRI, magnetic resonance imaging; VAMC, VA medical center; UCC, urgent care clinic; MOGAD, myelin oligodendrocyte glycoprotein antibody–associated disease; RNFL, retinal nerve fiber layer.

Introduction

Optic neuritis (ON) is an acute inflammatory demyelinating disorder of the optic nerve classically presenting with subacute, painful vision loss and dyschromatopsia. Although ON has diverse infectious and idiopathic causes, the most common non-infectious etiology worldwide is multiple sclerosis (MS); ON also occurs in neuromyelitis optica spectrum disorder (NMOSD) and myelin oligodendrocyte glycoprotein antibody–associated disease (MOGAD), where clinical course and visual prognosis differ substantially from typical MS-associated ON.[1,2] The relative frequency of ON subtypes varies by geography, reflecting regional differences in MS and NMOSD prevalence, and underscores the need for early etiologic classification to guide therapy.[2]

From an epidemiologic standpoint, ON is a common first demyelinating event and a sentinel presentation for MS. Long-term follow-up of ON cohorts demonstrates that approximately half of patients develop MS within 15 years after an initial ON episode, highlighting the condition's prognostic significance for central nervous system demyelination.[2,3] Typical ON affects young adults with a female predominance, often unilateral with spontaneous visual recovery, whereas atypical ON forms show distinct patterns: AQP4-IgG–positive NMOSD tends to be more severe, may involve the chiasm, and has poorer visual outcomes with higher relapse risk; MOGAD-ON frequently presents in younger patients (including children), with marked optic disc edema and variable recovery, and can be monophasic or relapsing.[2,4,5]

Diagnosis integrates clinical features, magnetic resonance imaging (MRI), and laboratory biomarkers. Typical ON is characterized by unilateral painful vision loss with reduced color vision and an afferent pupillary defect; MRI often reveals short-segment optic nerve enhancement, and cerebrospinal fluid (CSF) oligoclonal bands and brain MRI lesions refine MS risk assessment, aided by the 2017 McDonald criteria that enable earlier MS diagnosis in appropriate contexts.[2,6] In contrast, atypical phenotypes provide diagnostic clues: NMOSD-ON commonly shows long posterior optic nerve/chiasmal involvement and requires confirmation with AQP4-IgG serology per international consensus criteria; MOGAD-ON often exhibits long anterior nerve enhancement with perineural involvement and optic disc swelling, with diagnosis supported by MOG-IgG testing and dedicated clinical criteria.[2,4,5] Given the therapeutic divergence across

MS-ON, NMOSD-ON, and MOGAD-ON, early differentiation—sometimes on clinical grounds before serology and full imaging are available—is critical to optimize acute management and long-term outcomes.[2]

This is a case report presenting an atypical case of optic neuritis without significant optic nerve head swelling. Further, a short overview of the management of ON and its associated MS diagnosis.

Case report

A 52-year-old Caucasian male presented with sudden blurry and jumping vision in the upper half of his left eye two days ago. He thought he had a migraine episode, took his medication and went to sleep all day the previous day. The visual changes persisted so he went to urgent care clinic (UCC) at a VA medical center (VAMC) and was subsequently referred to the eye clinic. He did not have any previous symptoms of flashes or floaters, or any eye pain. There were not any neurological deficits such as impaired speech, facial weakness, arm or leg weakness. He denied nausea or vomiting. He had no scalp tenderness or jaw claudication, and no new shoulder pain. His last eye exam at a local eye clinic two years ago was unremarkable and he used over-the-counter readers as needed. His past medical history included allergic rhinitis, anemia, chronic back pain, depression, hypertension, and migraines. His medication list consisted of amlodipine, famotidine, gabapentin, hydrocordone-acetaminophen, sumatriptan and zolpidem.

Best-corrected visual acuity was 20/20- OD, 20/25- OS but jumping vision and cannot see the top half. Extraocular muscle motility was full without restrictions, but with moderate pain on eye movement OS; confrontation fields were full OD, and restricted superiorly OS; and pupils were equal, round, reactive to light with mild afferent pupillary defect OS. Anterior segment findings were unremarkable, and intraocular pressures were 14 mmHg in both eyes via Goldmann applanation tonometry. Amler grid testing confirmed hazy top half of the grid OS.

Dilated fundus examination revealed relatively normal fundus with cup-to-disc (C/D) ratio of 0.45 OD, 0.40 OS. The fundus photo showed relatively normal fundus, despite sudden loss of vision superiorly OS (Figure 1). Ocular coherent tomography (OCT) macular cube scans showed relatively normal retinal layers through the macula OD, OS (Figure 2A). Ganglion cell analysis showed significantly thinner inferior temporal sectors OS (Figure 2B). Figure 3 showed significant thinning of retinal nerve fiber layer (RNFL) inferiorly OD, OS. Visual field testing showed a cluster of defects superior temporally and inferior nasally OD, and a severe altitudinal defect superiorly OS (Figure 4 A, B).

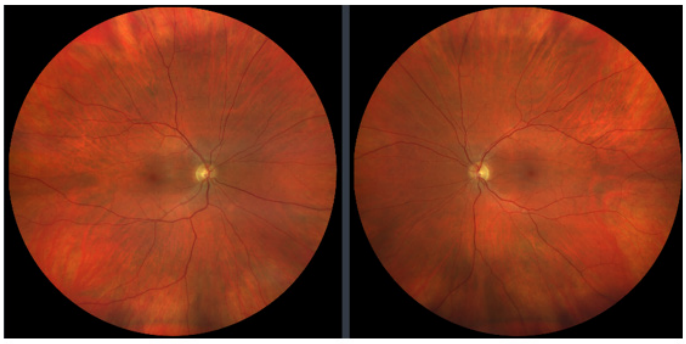

Figure 1 Relatively normal fundus photos, despite sudden loss of superior vision OS.

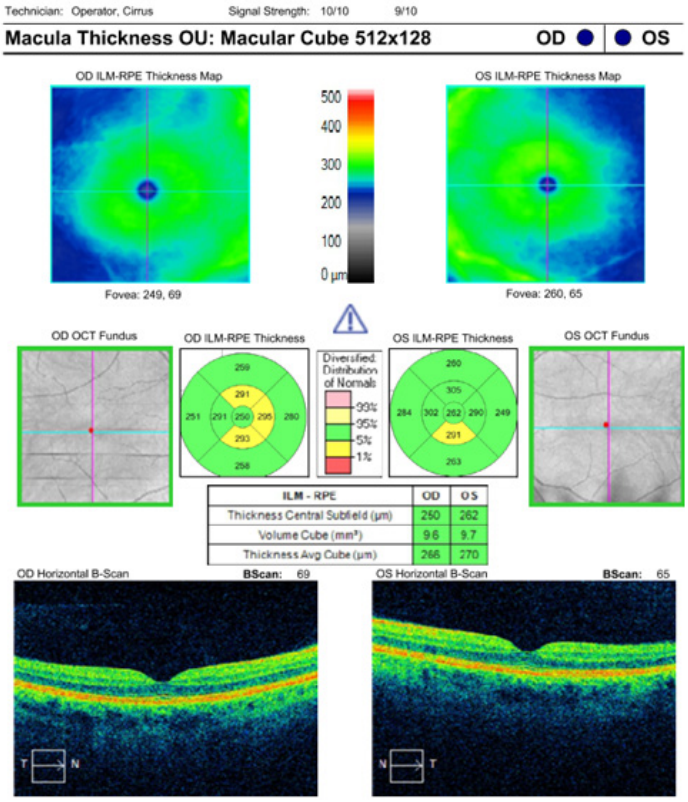

Figure 2A Relatively normal macular thickness OU

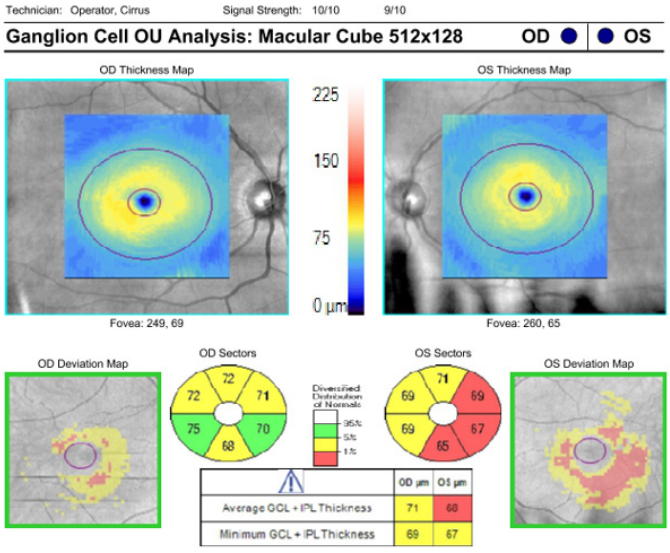

Figure 2B Ganglion cell analysis showed significantly thinner inferior temporal sectors OS.

A week later, MRI orbits and brain with and without contrast showed an increased T2 signal and enhancement of the intraorbital segment of the left optic nerve, compatible with optic neuritis (Figure 5A). Several periventricular and subcortical white matter FLAIR/T2 hyperintensity. One lesion in the right supraventricular white matter could represent a demyelinating lesion (Figure 5B).

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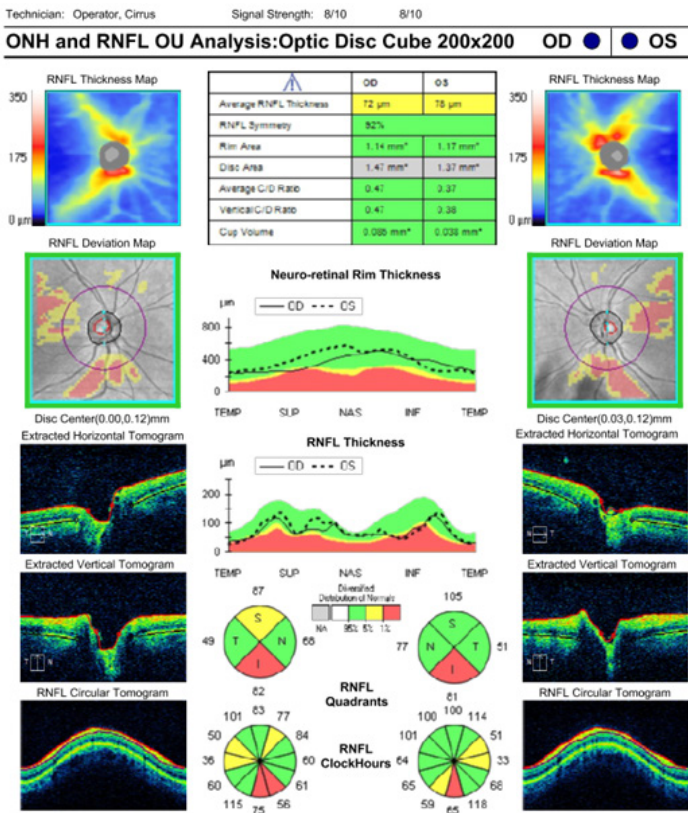

Figure 3 Significant thinning of retinal nerve fiber layer (RNFL) inferiorly OD, OS.

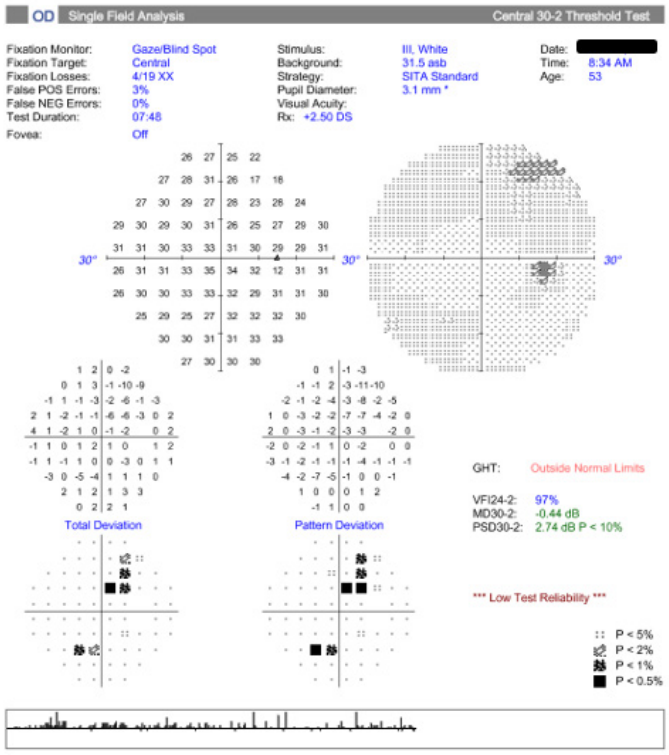

Figure 4A Visual field testing showed clusters of defects superior temporally and inferior nasally OD.

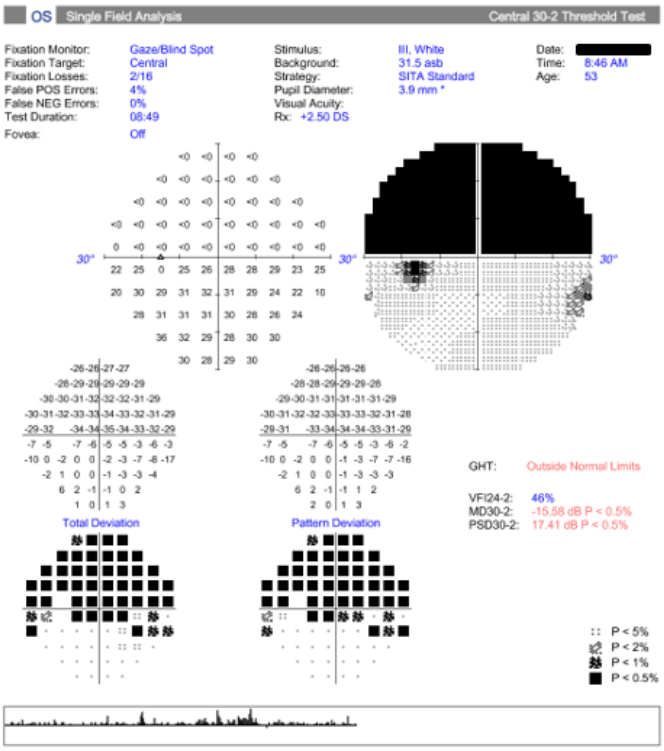

Figure 4B Visual field testing showed severe altitudinal defects superiorly OS.

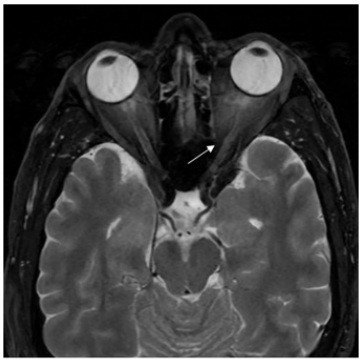

Figure 5A MRI Orbit showing asymmetric increased T2 signal and enhancement of the intraorbital segment of the left optic nerve (white arrow).

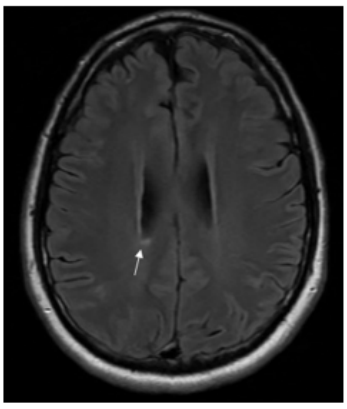

Figure 5B One lesion in the RIGHT supraventricular white matter could represent a demyelinating lesion (white arrow).

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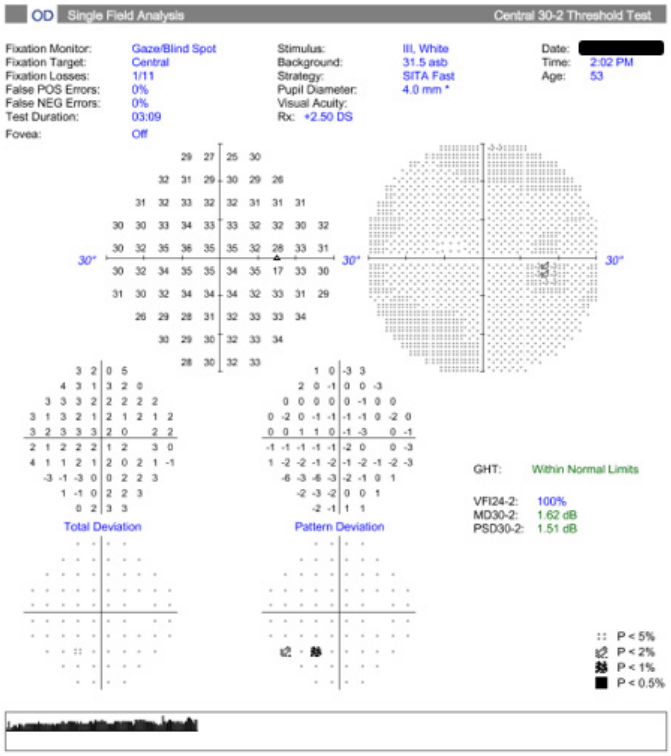

Figure 6A Visual field testing showed clusters of defects superior temporally and inferior nasally OD.

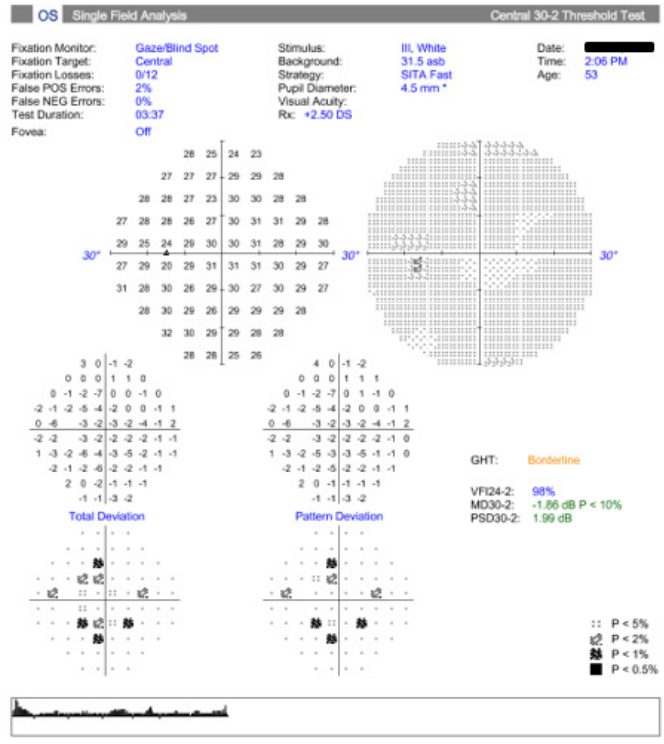

Figure 6B Visual field testing confirmed resolution of the superior altitudinal defects OS.

Two weeks later, blood work up confirmed anemia, and cerebral spinal fluid chemistry showed high CSF red blood cells 452 [normal high = 3], the presence of oligoclonal banc (IgG) with a high synthesis rate +5.9 [normal range = -9.9 to +3.3]. Patient was referred to neurology for further management and put on 80 mg/d prednisone for one week, then taper to 60 mg/d for two weeks, then 40 mg/d for 2 weeks, and 20 mg/d for 1 week.

Approximately a month after initial encounter and two weeks after high dose prednisone, his visual acuity was 20/20- OD, 20/20-OS. His vision superiorly OS had recovered significantly but still had some spotty areas as confirmed by visual field tests (Figure 6 A, B). The patient was scheduled for multiple sclerosis clinic at a local hospital in about a month.

Discussion

This case of a 54-year-old man with an acute, deep superior altitudinal visual field defect in the left eye and near-complete recovery after two weeks of high-dose oral prednisone is most consistent with inflammatory demyelinating optic neuritis (ON) rather than ischemic optic neuropathy. Rapid visual improvement after corticosteroids, pain with eye movements, and an afferent pupillary defect favor ON; by contrast, non-arteritic anterior ischemic optic neuropathy (NAION) typically presents in older adults with altitudinal defects but has slower, incomplete recovery and often a "disc at risk" (small crowded optic disc).[1,7] Altitudinal patterns can occur in ON, though they are classically associated with NAION; therefore, age >50 and an altitudinal scotoma appropriately broaden the initial differential to include arteritic AION due to giant cell arteritis (GCA), optic perineuritis, and compressive optic neuropathy.[1,7–9] The rapid steroid-responsive course here supports ON, but GCA must be excluded in any patient ≥50 years with acute optic neuropathy given the risk of bilateral irreversible vision loss.[8,10]

Diagnostic work-up should integrate structural, functional, and serologic testing. MRI of the brain and orbits with gadolinium and fat suppression helps distinguish typical MS-associated ON (short-segment intraorbital enhancement) from atypical ON patterns (e.g., long posterior nerve/chiasmal involvement in AQP4-IgG NMOSD; long anterior nerve with perineural enhancement and disc edema in MOGAD).[2,4,5] Optical coherence tomography (OCT) of the retinal nerve fiber layer (RNFL) and macular ganglion cell complex quantifies axonal injury and helps prognosticate recovery.[1,2] Given the patient's age and altitudinal defect, obtain ESR/CRP and platelet count promptly to screen for GCA, followed by temporal artery ultrasound/biopsy if inflammatory markers or clinical features (jaw claudication, scalp tenderness, polymyalgia) raise suspicion.[8,10] A demyelinating panel (AQP4-IgG and MOG-IgG cell-based assays) is indicated when atypical features (bilateral involvement, severe disc edema, recurrent episodes, poor recovery) are present or MRI suggests NMOSD/MOGAD.[4,5,11] For MS risk stratification, brain MRI using McDonald criteria and CSF oligoclonal bands remain pivotal in mid-life presentations of ON.[6]

The interprofessional management for acute ON includes coordinated care between internist, optometry, radiology, neurology, and rheumatology. In typical ON, high-dose corticosteroids accelerate recovery; historic ONTT data showed IV methylprednisolone (1 g/day × 3 days) hastens visual recovery, whereas low-dose oral prednisone increased relapse risk.[3] Contemporary evidence supports high-dose oral methylprednisolone (e.g., 500–1000 mg/day) as a reasonable alternative to IV therapy for MS relapses, with comparable outcomes and patient satisfaction—consistent with the favorable trajectory observed in this case.[12,13] If atypical ON is suspected (especially NMOSD), early plasma exchange (PLEX) added to IVMP improves visual outcomes, particularly when initiated within days

of onset; disease-specific long-term immunotherapy (eculizumab, inebilizumab, satralizumab, or ravulizumab for AQP4-IgG NMOSD) reduces relapse risk.[14–18] MOGAD-ON often responds briskly to steroids but may require a longer oral taper and relapse-prevention strategies (IVIG, rituximab, mycophenolate) if disease is relapsing or antibodies persist.[11,9–21] If GCA is suspected at any point, initiate immediate high-dose systemic glucocorticoids (often IV) and coordinate with rheumatology for confirmation and steroid-sparing therapy (e.g., tocilizumab) to prevent contralateral vision loss.[10]

Differential diagnosis in this case includes:

a) Demyelinating ON (MS-associated)—most common non-infectious cause of ON worldwide; typically unilateral, painful, with spontaneous recovery accelerated by steroids.[1–312,13]

b) AQP4-IgG NMOSD-ON—often severe, may be bilateral or chiasmal; poorer prognosis; early PLEX improves outcomes.[4,14–18]

c) MOGAD-ON—prominent disc edema, anterior/perineural enhancement; good steroid response but relapse-prone subset; consider extended taper and immunotherapy if relapsing.[5,11,19–21]

d) NAION/AAION (GCA)—altitudinal defect common; AAION is an emergency; NAION recovery limited; evaluate vascular risks and inflammatory markers.[7–10]

e) Optic perineuritis—painful vision loss with circumferential perineural enhancement; often steroid-responsive but requires prolonged taper and search for secondary causes (e.g., inflammatory, infectious).[9]

f) Compressive optic neuropathy—progressive deficits, optic disc pallor, or chiasmal signs; MRI critical.[1,2]

Clinical pearls for eye-care professionals

A. Age ≥50 + altitudinal defect = rule out GCA first. Check ESR/CRP/platelets urgently; do not delay corticosteroids if AAION is suspected.[8,10]

B. Steroid response does not equal ischemia. Rapid recovery after high-dose steroids strongly favors inflammatory ON over NAION.[3,12,13]

C. Let MRI pattern guide serology. Long posterior/chiasmal involvement → test AQP4-IgG; long anterior/perineural + disc edema → test MOG-IgG.[4,5,11]

D. Consider PLEX early for severe or steroid-refractory attacks when NMOSD is suspected; earlier initiation correlates with better visual outcomes.[14–16]

E. OCT adds value. OCT quantifies RNFL/GCC loss for prognosis.[1,2]

F. Tailor the steroid taper. Typical ON often needs a short taper; MOGAD/NMOSD may require longer tapers to reduce relapse risk.[5,11,14]

G. Coordinate care. Early primary care, radiology, neurology, and rheumatology input and clear pathways for MRI, serology, and infusion services improve time-to-treatment and outcomes.[2,14]

Conclusion

This case highlights an atypical presentation of ON in a mid-life patient with a deep superior altitudinal defect. Three practical points emerge:

a) Age ≥50 with acute optic neuropathy mandates immediate GCA screening and, if suspected, empiric steroids to protect the fellow eye;

b) Altitudinal defects do not exclude ON—rapid improvement with high-dose corticosteroids is more characteristic of inflammatory ON than NAION; and

c) Early, team-based pathways that couple expedited MRI with targeted serology (when indicated) optimize diagnosis and allow timely escalation (e.g., plasma exchange for NMOSD) when atypical features are present. Eye care professional may be the first to encounter this type of patient but interprofessional team of internist, radiologist, neurologist and rheumatologist are essential to timely and effective management in saving vision and life.

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Conflicts of interest

This case report is original, and the authors have no commercial interest in the subject of study. The authors do not have any financial conflicts of interest, and no funding agency is involved in this case report.

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