Case Report

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Optic Neuritis with Unilateral Superior Altitudinal Visual Field Defect in Multiple Sclerosis: A Case Report

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ABSTRACT

We describe an exceptional instance of multiple sclerosis manifesting in a unilateral, superior visual field deficit accompanied by painful eye movements. A 16-year-old girl with no prior health issues presented with symptoms of blurred vision, ocular pain in eye movement, and a throbbing headache in her right eye. She experienced right-sided numbness for two weeks, which later affected her left limbs. Effective management of multiple sclerosis necessitates familiarity with its uncommon presentations due to the significance of early diagnosis and treatment. Unilateral superior altitudinal visual field defect, orbital pain upon eye movement, and left hemi-paresthesia may be an uncommon presenting symptom in MS patients. After completing treatment with methylprednisolone, visual acuity can improve completely.

Keywords: Altitudinal, Multiple sclerosis, Visual field

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Introduction

Multiple sclerosis (MS) is one of the most prevalent chronic demyelinating conditions affecting the central nervous system (CNS), leading to an array of symptoms, including spinal cord syndromes, brainstem syndromes, and optic neuritis (ON). A usual visual manifestation of MS is optic neuritis, typically occurring in one eye and often accompanied by symptoms such as aching pain during eye movement, blurred vision, dim vision, or impaired color perception. The central scotoma is the most frequently observed presentation associated with this condition **(1, 2)**. An altitudinal visual field defect (AVD) refers to a visual field impairment affecting the superior or inferior segment of the visual field while preserving the horizontal midline. AVDs are typically linked to ischemic optic neuropathy (ION) and occlusion of the hemibranch arteries or veins. While less frequently, altitudinal visual field defects can arise from advanced glaucoma or, in rare cases, compressive neuropathy due to tumors or aneurysms. Typical cases of ON associated with MS present as painful, subacute vision loss, in contrast to ION, which usually results in painless monocular vision loss with AVD, primarily observed in older individuals with vascular risk factors.

This case report contains the presentation of a patient with unilateral superior altitudinal visual field defect, a discussion documented in only a limited number of studies.

Case Presentation

A previously healthy 16-year-old girl was referred to our neurology department due to complaints of blurry vision and ocular pain when moving her right eye, along with a throbbing headache that had persisted for two days. She reported difficulty seeing the upper part of a computer or television screen and experienced reading issues when the visual deficit crossed the horizontal plane. To see objects in her blind superior

field, she had to strike her head against an object positioned above her.

One month earlier, she had experienced right-sided hemi-paresthesia that lasted for two weeks, which then reappeared in her left limbs. There were no reports of visual disturbances such as flashes or floaters. She denied any recent head or eye trauma, insect bites, symptoms indicative of increased intracranial pressure, ocular surgery, fever, or any other constitutional symptoms. Her family medical history and past ocular history were both unremarkable.

During the finger count (FC) examination, her visual acuity was observed to be 20/200 in the right eye and 20/20 in the left eye. A grade II relative afferent pupillary defect (RAPD) was detected in her right eye. The Ishihara test could not be performed due to the poor vision in her right eye but indicated normal color vision in the left eye. Intraocular pressure measurements were within normal limits. Ophthalmoscopic examination showed no signs of swelling or hyperemia of the optic disc, nor were there any indications of retinitis or vitritis in the posterior pole. While ocular movements were fully intact, they were accompanied by noticeable ocular pain during the upward movement of the right eye. The remainder of her neurological examination was unremarkable.

A routine laboratory assessment revealed normal results for the full blood count, erythrocyte sedimentation rate, renal function, C-reactive protein, folate, and vitamin B12 levels. Additionally, her serum tested negative for anti-aquaporin-4 antibodies (anti AQP4). Screening tests for collagen vascular diseases, autoimmune conditions, and infectious and venereal diseases, including human immunodeficiency virus (HIV) and hepatitis B and C, were also non-reactive.

The visual evoked potential (VEP) showed an absent waveform in the right optic pathway and a P100 wave latency of 103.5 ms in the left eye. Due to a unilateral visual field defect, we initially conducted a contrast-enhanced computed tomography (CECT) scan of the brain and orbit to rule out any space-occupying lesions but found no significant abnormalities. Humphrey's automated perimetry confirmed a superior altitudinal visual field defect in the right eye (Figure 1), while the left eye exhibited a normal field plot.

Magnetic resonance imaging (MRI) of the brain and orbit revealed abnormal T2-weighted hyperintense signals in the pons, right temporal region, periventricular areas, and juxtacortical locations, with the presence of Dawson's finger sign, but no abnormal enhancement in the optic nerve or brain was noted.

Our case highlights a rare instance of subjective disturbance in the superior visual field due to altitudinal scotoma as an initial symptom of acute unilateral optic neuritis. This clinical presentation, along with the VEP and MRI findings, necessitated the exclusion of other potential diagnoses, leading to the decision to perform a lumbar puncture (LP). The opening intracranial pressure measured 10 cmH2O, and the cerebrospinal fluid (CSF) sample was clear. There was a negative result for anti-aquaporin-4 antibodies in the CSF, while CSF electrophoresis revealed three additional oligoclonal bands with an IgG index of 2.33, consistent with multiple sclerosis (MS).

This case underscores the significance of a comprehensive approach in the etiological differential diagnosis of optic neuritis and highlights the essential roles of neurological imaging and CSF analysis in confirming a diagnosis of multiple sclerosis. It illustrates the clinical complexities and presentations associated with optic neuritis, reinforcing the need for thorough investigations in patients who exhibit visual disturbances.

Another differential diagnosis often linked with ON in otherwise healthy young women is neuromyelitis optica spectrum disorder (NMOSD), which was excluded in this case due to the absence of anti-aquaporin-4 antibodies in both serum and CSF, as well as certain MRI findings. Non-arteritic anterior ischemic optic neuropathy (NAION) was also ruled out because of the presence of painful monocular visual loss, a unilateral superior altitudinal defect, the patient's age, and the lack of vascular risk factors such as diabetes, hypertension, or any significant surgical history.

This patient met the revised McDonald's criteria for diagnosing relapsing-remitting multiple sclerosis (RRMS): a history of a prior attack, the presence of MRI plaques indicating dissemination in space (DIS), and the detection of extra oligoclonal bands in the CSF, which confirms dissemination in time (DIT).

She was treated with intravenous methylprednisolone at a dose of 500 mg every 12 hours for five days and was subsequently discharged with an oral prednisolone regimen of 1 mg/kg/day, which was tapered gradually over two weeks. By the conclusion of the five-day methylprednisolone treatment, her visual acuity in the right eye had improved to 20/20.

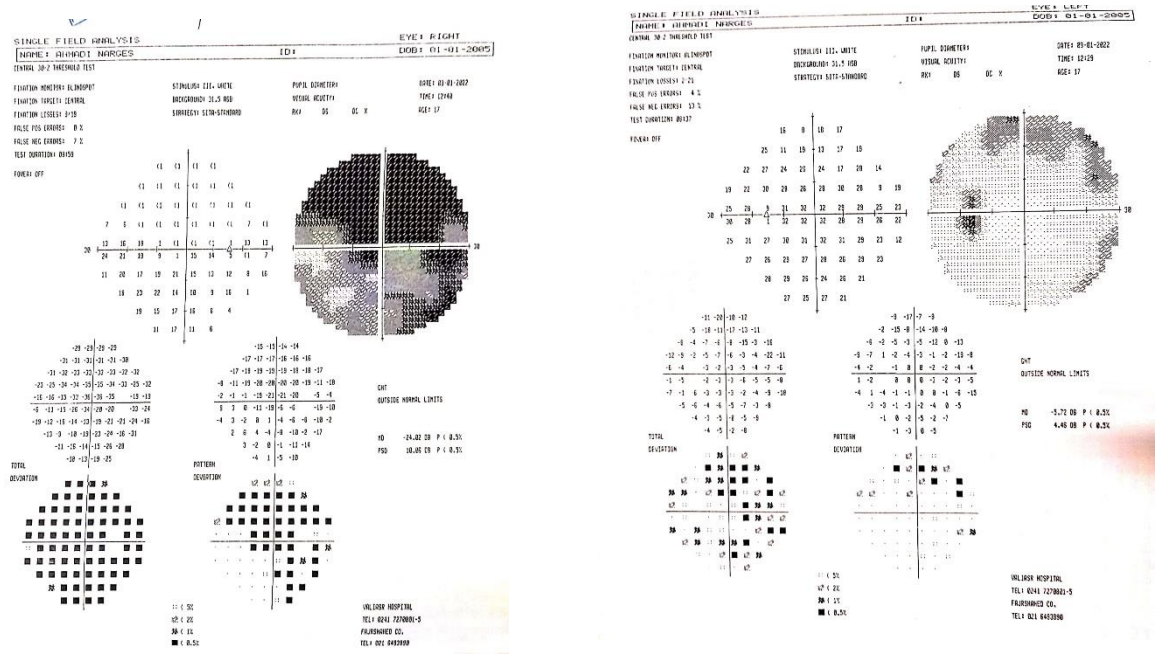

Figure 1. Humphrey perimetry of right eye (left) and left eye (right)

Discussion

In this report, we present a case of multiple sclerosis (MS) characterized by a unilateral superior altitudinal visual field defect.

Visual impairments, such as central scotoma, are common in MS and typically arise from retrobulbar optic neuritis or papillitis. However, demyelinating lesions can, less frequently, impact the retrochiasmal pathways. Research by Patterson et al. and Vidović et al. identified arcuate scotoma and varying degrees of visual field narrowing, accompanied by an enlarged blind spot and depression above, as the most prevalent defects observed **(3,4)**.

A similar case documented by Chin and Fathi also highlighted an atypical presentation of optic neuritis with a unilateral inferior low-altitude visual field defect **(5)**.

While the utility of visual field testing in diagnosing MS is somewhat limited, it is crucial to consider other differential diagnoses, such as space-occupying or life-threatening lesions, that can also cause altitudinal visual defects. Consequently, we pursued immediate brain imaging to eliminate these potential causes **(6)**.

While the pathophysiology of ON remains poorly understood, it is generally believed that inflammation and demyelination of the optic nerve play pivotal roles in its etiology **(7)**. The process is initiated by releasing cytokines and other inflammatory mediators, which facilitate the migration of activated peripheral T cells across the blood-brain barrier. This migration ultimately destroys myelin, neuronal cell death, and axonal degeneration **(8, 9)**. Altitudinal visual field defects may arise from direct inflammation of the optic nerve or secondary perfusion deficits caused by this inflammation. In certain cases, an altitudinal visual field defect can be attributed to the occlusion of the posterior ciliary artery, a vascular change that may be driven by direct vascular inflammation linked to anti-AQP4 antibodies **(2)**.

In the current patient diagnosed with MS, para-clinical assessments revealed abnormal T2-weighted hyper-intense signals in the pons, right temporal region, periventricular areas, and juxtacortical locations, along with the presence of the Dawson's fingers sign and abnormalities detected in CSF electrophoresis.

The primary objective of treatment is to diminish both the frequency and severity of attacks and to prevent axonal loss in cases of ON and MS **(9)**. After excluding systemic infections, intravenous methylprednisolone was administered **(10)**.

We present a case of an MS patient exhibiting unusual symptoms. She presented with blurred vision in her right eye, accompanied by a throbbing headache in the left temporal region, orbital pain during eye

movement, and left hemi-paresthesia. In her medical history, she reported experiencing right hemi-paresthesia approximately one month before her current symptoms. Clinical evaluation ultimately indicated a diagnosis of right retrobulbar neuritis, characterized by a superior altitudinal visual field defect.

Conclusion

In this report, we identified altitudinal visual field defects as a rare symptom of MS. This symptom can serve as a potential indicator for diagnosing MS, regardless of whether there is a prior history of optic neuritis. Our findings demonstrate that visual field defects can occur in altitudinal regions and central or peripheral areas, often progressing slowly and going unnoticed by MS patients. Our study emphasizes perimetry's importance in identifying overt and hidden optic nerve involvement in MS patients. It measures the extent of visual field loss, detects atypical cases of optic neuritis, and assists in providing patients with prognostic information.

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None.

Authors' Contribution

MM: Conceived, designed and did statistical analysis & editing of manuscript; data collection and Manuscript writing, review and final approval of manuscript.SHA: Conceived, did Statistical analysis & data collection and manuscript writing.MSA: Data collection and manuscript writing, final approval of manuscript

Conflict of Interest

The authors declare that they have no conflict of interest.

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