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CASE REPORT

Sequential Optic Neuropathy in Idiopathic Intracranial Hypertension: A Diagnostic Challenge of Possible Superimposed Nonarteritic Anterior Ischemic Optic Neuropathy

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ABSTRACT

Idiopathic intracranial hypertension (IIH) and non-arteritic anterior ischemic optic neuropathy (NAION) are distinct optic nerve disorders that may present diagnostic overlap in patients with optic disc edema and shared vascular or structural risk factors. We report a 40-year-old woman with obesity and systemic vascular comorbidities who initially presented with painless vision loss in the right eye consistent with NAION. Four months later, she developed new left optic disc edema, accompanied by headaches and pulsatile tinnitus. Neuroimaging demonstrated partial empty sella, posterior globe flattening, and bilateral transverse sinus stenosis, and lumbar puncture revealed elevated opening pressure with normal cerebrospinal fluid composition, supporting a diagnosis of IIH. Following treatment with carbonic anhydrase inhibitors, papilledema partially improved; however, a new inferior arcuate visual field defect with worsening disc edema subsequently developed in the left eye, raising concern for possible superimposed NAION. During follow-up, disc edema gradually resolved while the visual field defect persisted. This case underscores the diagnostic complexity of distinguishing possible superimposed NAION from progressive papilledema-related visual loss and highlights the importance of considering ischemic optic neuropathy when new or asymmetric visual field defects develop in patients with IIH.

Keywords: idiopathic intracranial hypertension, nonarteritic anterior ischemic optic neuropathy, optic disc edema, papilledema, sequential optic neuropathy

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INTRODUCTION

Idiopathic intracranial hypertension (IIH) predominantly affects young obese women and is characterized by elevated intracranial pressure (ICP) without an identifiable structural lesion,[1,2] with normal neuroimaging and cerebrospinal fluid composition, and is commonly associated with papilledema.

Nonarteritic anterior ischemic optic neuropathy (NAION), the most common acute optic neuropathy in individuals older than 50 years with vascular risk factors,[3] can also occur in younger patients with predisposing vascular risk factors or a small crowded optic disc ("disc-at-risk"). Although IIH and NAION are pathophysiologically distinct, papilledema-related optic nerve head crowding has been proposed as a potential predisposing factor for NAION in susceptible individuals.[4,5]

Reports describing NAION in the setting of IIH remain limited,[4-9] and the temporal relationship between these entities is not well defined. Most published reports describe a single ischemic event occurring in association with IIH, with limited longitudinal follow-up documenting the evolution of both conditions. Here, we present a patient with obesity and vascular risk factors who experienced sequential presumed NAION before and after the diagnosis of IIH, highlighting the diagnostic challenge of distinguishing superimposed NAION from progressive papilledema during the clinical course of IIH.

CASE REPORT

A 40-year-old woman was referred to the neuro-ophthalmology clinic one month after presenting to the emergency department with painless vision loss in the right eye (OD) upon awakening, where she was clinically diagnosed with presumed NAION. Her medical history was notable for hypertension, type II diabetes mellitus, hyperlipidemia, migraines, Hashimoto thyroiditis, and polycystic ovarian syndrome. Body mass index was 59.9 kg/m^2. Medications included lisinopril, metformin, glimepiride, semaglutide, atorvastatin, pregabalin, levothyroxine, and duloxetine.

At her initial neuro-ophthalmology evaluation, best-corrected visual acuity (BCVA) was 20/50 OD and 20/25 in the left eye (OS), with a right relative afferent pupillary defect (RAPD). Fundus examination demonstrated optic disc edema OD and a small, crowded optic disc ("disc-at-risk") OS. Automated perimetry showed an inferior altitudinal defect with superior arcuate involvement OD and a normal visual field OS. Magnetic resonance imaging (MRI) of the brain and orbits, performed during the initial emergency department evaluation, demonstrated no optic nerve enhancement or compressive lesion, and laboratory investigations for infectious and inflammatory optic neuropathies were negative. Aspirin 81 mg daily had been initiated, and lisinopril dosing had been adjusted for improved blood pressure control.

At four months, she reported mild worsening of blurred vision OD and longstanding non-migrainous headaches occasionally associated with pulsatile tinnitus. BCVA was 20/60 OD and 20/25 OS with persistent RAPD OD, and color vision remained normal bilaterally. The right optic nerve appeared diffusely pale, while the left demonstrated new Frisen grade 2 disc edema. Optical coherence tomography (OCT) showed global retinal nerve fiber layer thickness of 45 µm OD and 160 µm OS **(Figure 1A)**. Ganglion cell layer (GCL) analysis demonstrated superior thinning OD with preserved architecture OS (GCL volume 0.74 mm^3 OD and 0.96 mm^3 OS) **(Figure 1B)**. Automated perimetry showed a stable defect OD and a newly enlarged blind spot OS **(Figure 1C)**.

Repeat MRI brain and orbits with venography demonstrated partially empty sella, distension of bilateral optic nerve sheaths, flattening of the posterior globe, and bilateral transverse sinus stenosis **(Figure 2)**. Lumbar puncture demonstrated an opening pressure of 31 cm H_2O with normal cerebrospinal fluid contents, consistent with IIH. Acetazolamide 500 mg twice daily was initiated but transitioned to methazolamide 50 mg twice daily because of intolerance to side effects.

One month after initiation of methazolamide, symptoms were stable and left optic disc edema had decreased clinically to Frisen grade 1. Retinal nerve fiber layer (RNFL) thickness measured 124 µm OS **(Figure 3)**. Three months after starting methazolamide, she reported a new inferior visual field defect OS described as a "black spot," without headache, transient visual obscurations, or pulsatile tinnitus. BCVA was 20/50 OD and 20/25 OS with persistent RAPD OD, and color vision remained normal bilaterally. The right optic nerve remained diffusely pale, while the left demonstrated Frisen grade 3 disc edema, more prominent superiorly. RNFL thickness measured 43 µm OD and 260 µm OS **(Figure 4A)**. Automated perimetry showed a stable defect OD and a new inferior arcuate defect OS **(Figure 4B)**.

Because the new visual field defect and worsening optic disc edema raised concern for either suboptimally controlled IIH or superimposed NAION, methazolamide was increased to 100 mg twice daily while close follow-up was arranged. A hypercoagulable evaluation was ordered

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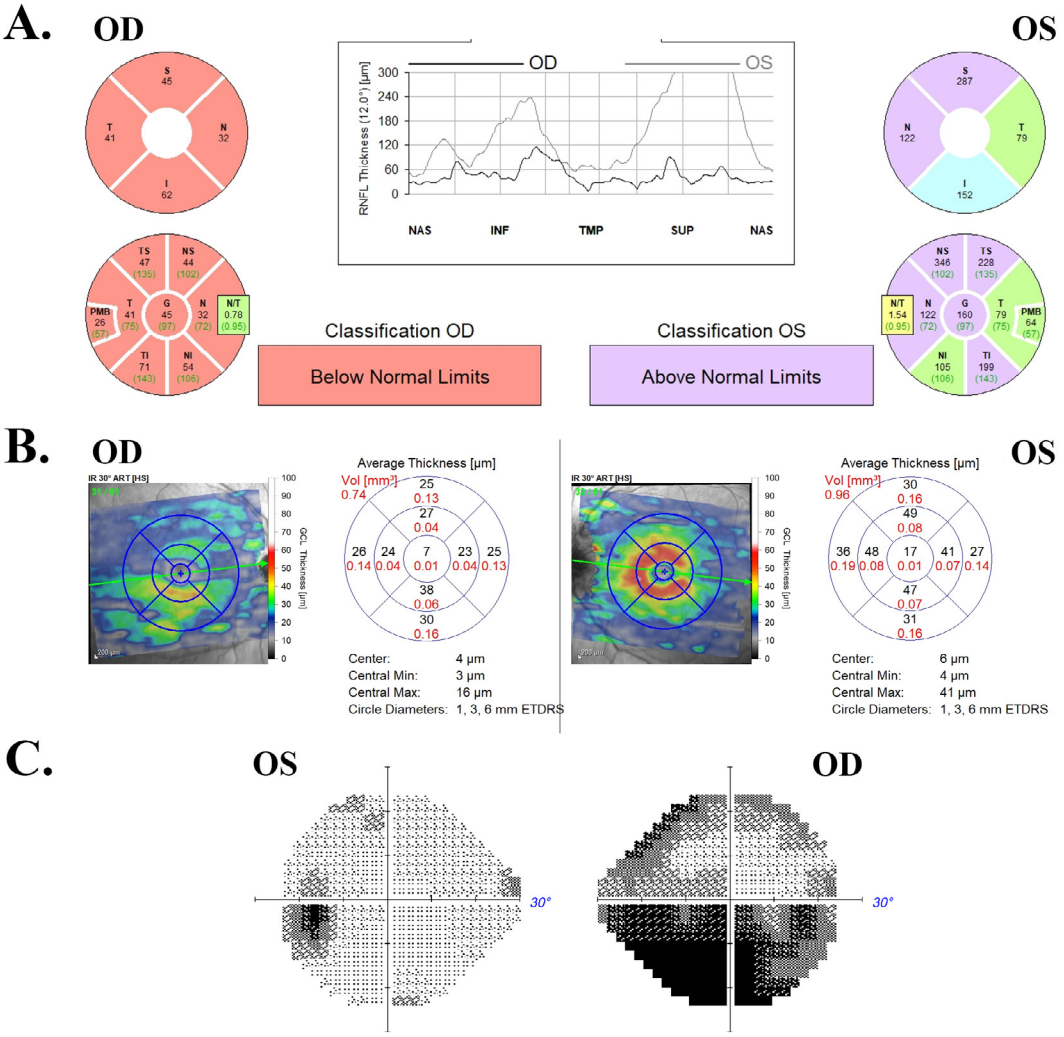


Figure 1 Structural and functional evaluation at four-month follow-up

(A) Peripapillary OCT demonstrates diffuse RNFL thinning OD (global average 45 µm) and RNFL thickening OS (160 µm), consistent with optic disc edema OS. (B) GCL analysis shows superior thinning OD (GCL volume 0.74 mm^3) with preserved architecture OS (0.96 mm^3). (C) Automated perimetry reveals an inferior altitudinal defect with superior arcuate involvement OD and an enlarged blind spot OS. (OD – right eye, OS – left eye)

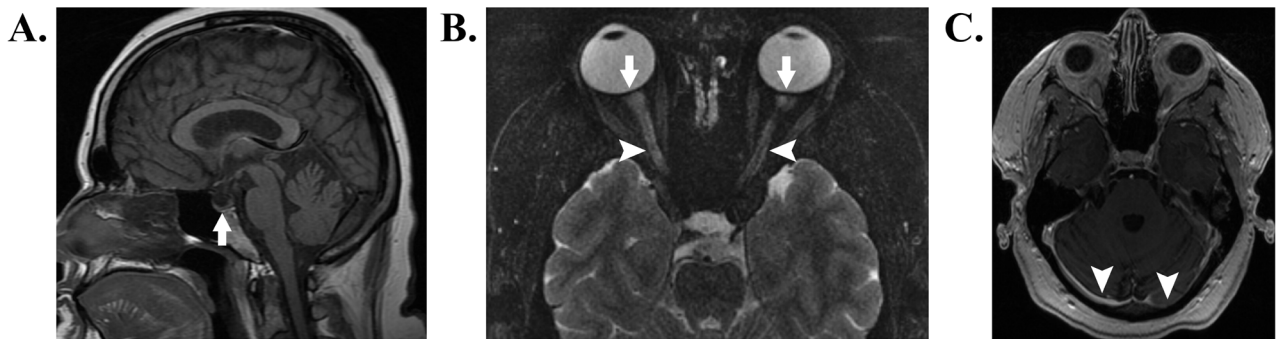


Figure 2 MRI findings supporting the diagnosis of IIH

(A) Sagittal pre-contrast T1-weighted brain image demonstrating a partially empty sella (arrow). (B) Axial fat-saturated T2-weighted orbital image demonstrating distension of the bilateral perioptic subarachnoid spaces (arrowheads) and flattening of the posterior globes (arrows). (C) Axial post-contrast T1-weighted brain image at the level of the transverse sinuses (arrowheads). MRI with MR venography demonstrated bilateral transverse sinus stenosis.

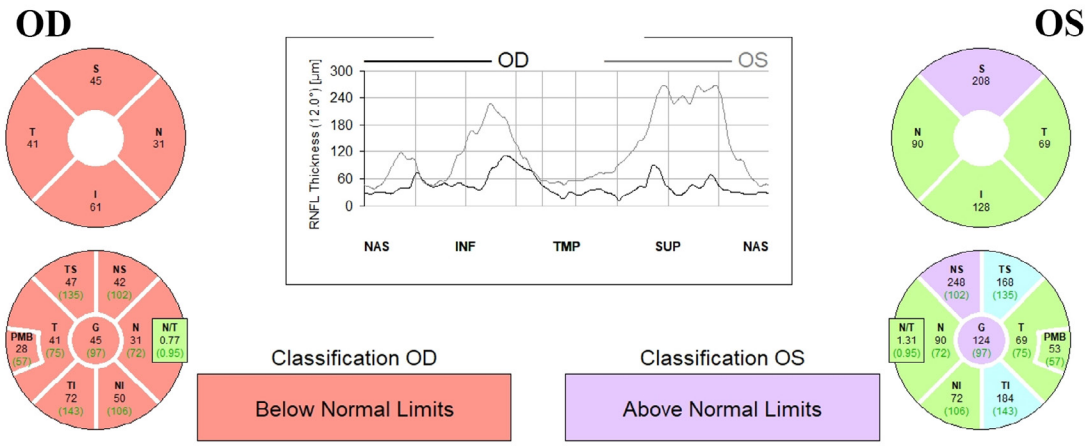


Figure 3 Peripapillary OCT one month after methazolamide
OCT demonstrates stable diffuse RNFL thinning OD (global average 45 µm) and decreased RNFL thickness OS (124 µm), consistent with improving disc edema OS. (OD – right eye, OS – left eye)

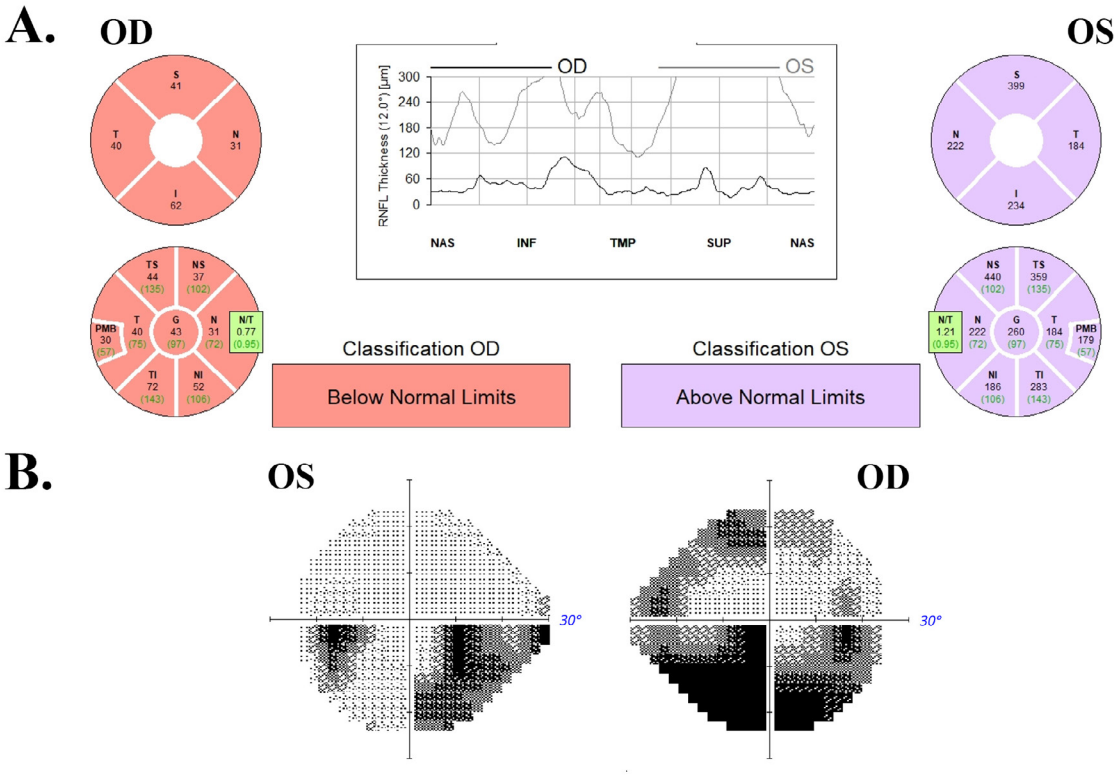


Figure 4 Structural and functional progression three months after methazolamide
(A) Peripapillary OCT demonstrates stable diffuse RNFL thinning OD (global average 43 µm) and marked RNFL thickening OS (260 µm), consistent with worsening disc edema OS. (B) Automated perimetry shows a stable defect OD and a new inferior arcuate defect OS. (OD – right eye, OS – left eye)

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and was negative, and a sleep study was requested but not completed. During subsequent visits, she remained free of symptoms attributable to elevated ICP. The visual field defect OS persisted without further decline. Gradual resolution of disc edema OS was observed, and methazolamide was later tapered to 50 mg twice daily.

At the final visit, seven months after the new event, BCVA was 20/60 OD and 20/25 OS with persistent RAPD OD. Both optic nerves appeared pale, more pronounced OD, without residual edema OS. RNFL thickness measured 42 µm OD and 74 µm OS **(Figure 5A)**. GCL analysis demonstrated superior thinning in both eyes (GCL volume 0.63 mm³ OD and 0.68 mm³ OS) **(Figure 5B)**. Automated perimetry remained stable, showing an inferior altitudinal defect with superior arcuate involvement OD and an inferior arcuate defect OS **(Figure 5C)**.

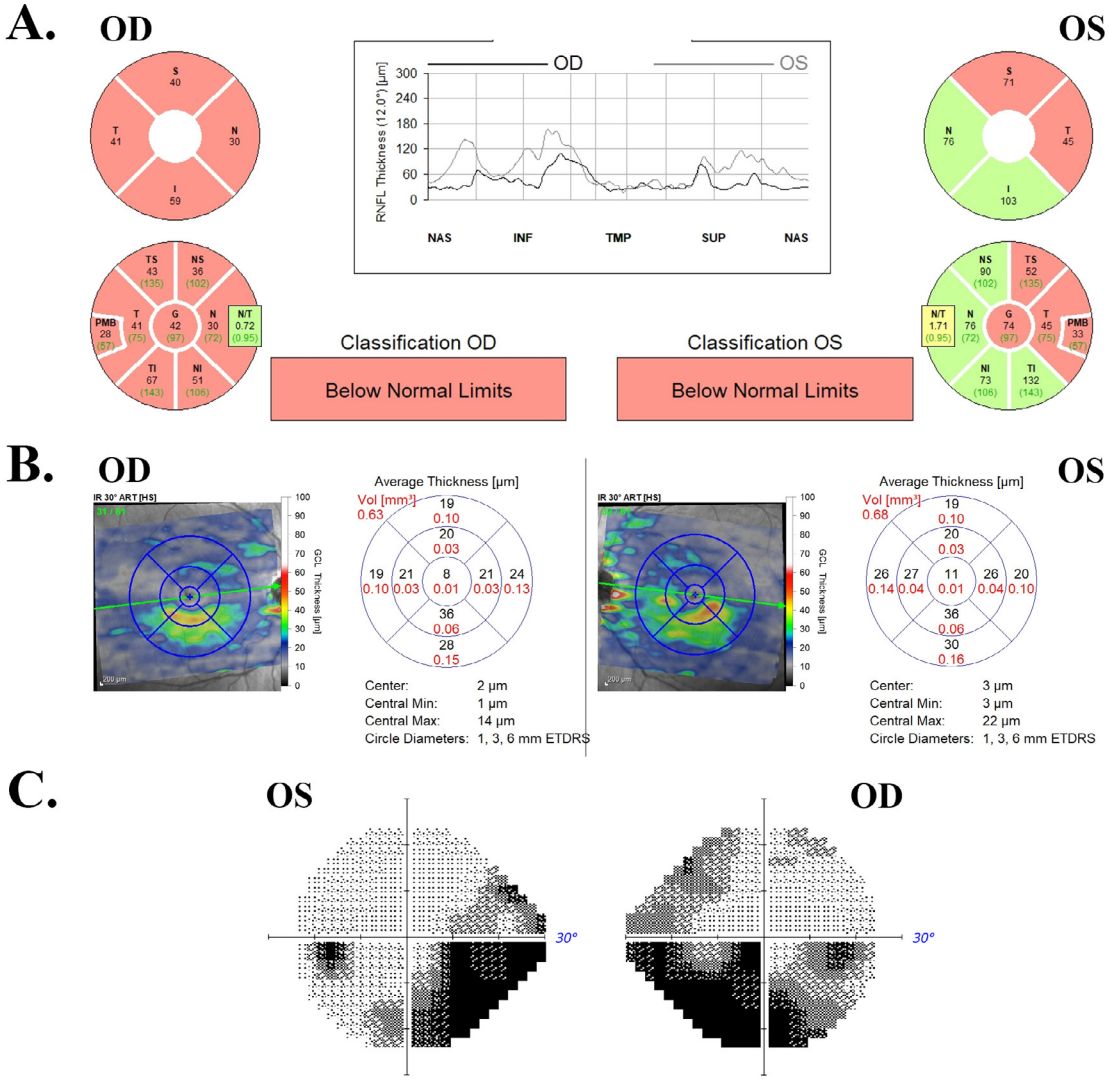


Figure 5 Structural and functional findings at final follow-up

(A) Peripapillary OCT demonstrates diffuse RNFL thinning OU (global average 42 µm OD, 74 µm OS), with resolution of prior disc edema OS. (B) GCL analysis shows superior thinning OU (GCL volume 0.63 mm³ OD, 0.68 mm³ OS). (C) Automated perimetry reveals a persistent inferior altitudinal defect with superior arcuate involvement OD and an inferior arcuate defect OS.

DISCUSSION

We report a young obese woman with vascular risk factors who developed NAION OD, followed by newly diagnosed IIH with papilledema in the OS, and subsequently an episode of worsening optic disc edema OS that raised concern for possible superimposed NAION.

IIH commonly produces visual disturbance through transient visual obscurations and enlarged blind spots in the early stages.[10,11] Permanent visual loss may occur in untreated or severe papilledema and often begins with nasal field loss, progressing to arcuate scotomas, peripheral constriction, and eventually central vision loss.[10,11] Visual decline is typically gradual and relatively symmetric unless the disease becomes fulminant or macular involvement occurs.[10,12]

In contrast, visual loss in NAION is classically acute, unilateral, and painless, often noticed upon awakening.[3,13] Optic disc swelling may be segmental or diffuse, and the typical visual field defect is inferior altitudinal loss. A small, crowded optic disc represents the primary anatomical risk factor.[3,13] Fellow-eye involvement occurs in approximately 15–25% of patients within five years,[14] particularly in those with persistent systemic vascular risk factors.

In our patient, papilledema initially improved following methazolamide therapy, accompanied by reduction in optic disc edema and resolution of symptoms suggestive of elevated ICP. At a subsequent follow-up visit, however, she developed a new inferior arcuate visual field defect with worsening optic disc edema OS. Although arcuate visual field defects may occur in papilledema, the development of a new inferior arcuate defect despite initial improvement in papilledema raised concern for possible superimposed NAION. Nevertheless, progression of papilledema due to suboptimally controlled IIH could not be excluded. Because both possibilities remained in the differential diagnosis, methazolamide dosage was increased and close follow-up was arranged, together with evaluation for hypercoagulable states and obstructive sleep apnea given their potential contribution to bilateral NAION.[14,15]

During follow-up, she remained free of symptoms of elevated ICP, while the optic disc edema gradually resolved and the visual field defect remained stable without further deterioration. Methazolamide was subsequently tapered. In retrospect, this clinical course favored superimposed NAION over progressive papilledema. However, because the initial management appropriately addressed both diagnostic possibilities and confirmatory ancillary testing such as fluorescein angiography and diffusion-weighted MRI was not performed, the diagnosis cannot be established with complete certainty. This underscores how difficult it can be to separate a superimposed ischemic event from ongoing papilledema in patients with IIH, particularly when both entities may coexist during the same clinical course.

Previous reports have described NAION occurring in association with IIH across various clinical scenarios,[4-9] including simultaneous presentation, development during routine follow-up, and visual loss associated with untreated or refractory papilledema. Most reports, however, describe a single ischemic event with limited longitudinal structural and functional follow-up. In contrast, our patient experienced two presumed NAION episodes occurring before and after the diagnosis of IIH, allowing longitudinal assessment with serial OCT RNFL, GCL analysis, and automated perimetry. This longitudinal follow-up demonstrates how diagnostic confidence in distinguishing superimposed NAION from progressive papilledema evolved through serial clinical assessment rather than a single examination. Papilledema may further increase optic nerve head crowding in eyes with a preexisting disc-at-risk,[4,5] thereby creating a structural environment susceptible to ischemia. Our patient had multiple vascular risk factors, including hypertension, diabetes mellitus, hyperlipidemia, and obesity, that independently predispose to NAION. The second presumed NAION event in this case may therefore reflect a multifactorial process involving papilledema-related crowding superimposed on systemic vascular susceptibility.

Weight reduction is a key component of IIH management and may contribute to disease remission.[16] Our patient had been receiving semaglutide before her initial NAION event and had achieved approximately 10 kg of weight loss. Following the diagnosis of IIH, her papilledema responded well to methazolamide, and continued weight reduction remained an important treatment goal. After the second presumed NAION event, however, glucagon-like peptide-1 receptor agonists became a management consideration because of their possible association with NAION. Although current evidence remains inconclusive, with ongoing studies investigating a potential causal relationship, recent large retrospective studies have not demonstrated a clear association.[17,18] We therefore discussed this potential concern with the patient's internist and considered alternative glucose-lowering therapy if clinically appropriate.

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In diagnostically challenging cases, ancillary testing may assist in distinguishing NAION from papilledema-related visual decline. Fundus fluorescein angiography may show delayed disc filling in NAION,[9,19] whereas papilledema typically demonstrates bilateral disc leakage without perfusion delay. Diffusion-weighted imaging on MRI may demonstrate restricted diffusion at the optic nerve head in acute NAION,[4,6] although this finding is not universally present. In the present case, neither fluorescein angiography nor diffusion-weighted MRI was performed during the second episode of optic disc edema. These ancillary investigations might have improved diagnostic confidence in differentiating presumed superimposed NAION from progressive papilledema and therefore represent an important limitation of this report.

CONCLUSION

This case demonstrates how closely NAION and IIH can overlap clinically when they occur in close temporal association, particularly in young patients with vascular risk factors and obesity who develop sequential optic neuropathy. Both entities should be considered in patients with papilledema who develop new or asymmetric visual field loss. Recognition of this potential overlap is clinically important because distinguishing superimposed NAION from progression of papilledema may remain challenging and may influence diagnostic evaluation and management. Careful clinical assessment with appropriate ancillary testing remains essential to guide timely management and optimize visual outcomes.

Conflict of Interest

The authors report no conflicts of interest related to this work.

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During the preparation of this manuscript, the authors used ChatGPT (OpenAI) and Gemini (Google) for language editing, grammar improvement, and readability enhancement. The authors reviewed and edited the text and take full responsibility for the content, accuracy, and scientific integrity of the final manuscript.

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Formal analysis: -

Funding acquisition: -

Investigation: J.N., S.H.

Methodology: -

Project administration: -

Resources: S.H.

Software: -

Supervision: S.H.

Validation: -

Visualization: J.N.

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Writing – review & editing: J.N., S.H.

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