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Anterior ischemic optic neuropathy in patients treated with semaglutide: report of four cases with a possible association

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Abstract

Background Semaglutide, a glucagon-like peptide-1 receptor agonist, is increasingly used worldwide for its cardiometabolic benefits. However, it has recently been associated with nonarteritic anterior ischemic optic neuropathy (NAION). This report presents four clinical cases and explores a possible association.

Case presentation Four male patients were diagnosed with NAION during semaglutide treatment, with treatment durations of less than one year in three cases. All presented with significant optic disc edema and intraretinal fluid on optical coherence tomography, along with crowded optic disc and small Bruch's membrane opening diameters (< 1.4 mm in three cases). One patient exhibited optic disc drusen. Visual field defects corresponded with ganglion cell layer atrophy on optical coherence tomography. Systemic risk factors varied; two patients had only obesity.

Conclusions Our observation suggests that individuals with small Bruch's membrane opening diameter may be at risk of developing NAION during semaglutide treatment.

Keywords Semaglutide, nonarteritic anterior ischemic optic neuropathy, case-series.

Background

Nonarteritic anterior ischemic optic neuropathy (NAION) is a leading cause of sudden vision loss, primarily affecting middle-aged and elderly individuals. Its hallmark features are an acute unilateral painless onset of visual impairment, relative afferent pupillary defect (RAPD) and hyperemic optic disc swelling. Common risk factors include structural predispositions, such as a crowded optic disc and optic disc drusen (ODD), and systemic conditions like sleep apnea syndrome, hypertension, hyperlipidemia, nocturnal hypotension, diabetes mellitus, and hypercoagulability [1, 2].

Semaglutide, a glucagon-like peptide-1 receptor agonist (GLP-1 RA), has become an integral component of weight management and type 2 diabetes treatment due to its potent efficacy in glycemic control and cardiovascular risk reduction. Despite its widespread use, studies have suggested a possible association between semaglutide and NAION [3–5]. This potential risk raises questions about the ocular safety of GLP-1 RAs, in particularly the need for identifying predisposed individuals.

This case series presents four instances of NAION in patients receiving semaglutide therapy.

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Table 1 Case characteristics

	Case 1	Case 2	Case 3	Case 4
Age	47	63	60	55
Sex	Male	Male	Male	Male
Blood pressure	140/90	158/98	142/86	
BMI, kg/m^2	28.7	25.9	44.1	27.5
Smoking	no	no	no	no
HbA1c, mmol/mol	31	39	54	31
Diabetes	None	None	DM2	None
Cholesterol, mmol/L	5.1	4.8	2.1	3.3
Semaglutide dose, mg per week	2.4	1.0	1.0	1.7
Best corrected visual acuity				
OD	20/20*	20/20	20/32*	20/32*
OS	20/20	20/40*	20/20	20/16
RAPD	moderate	moderate	moderate	moderate
IOP, mmHg				
OD	12	15	11	
OS	13	18	13	
Perimetric MD, dB				
OD	-13.8	-2.0	-18.8	-23.5
OS	-3.0	-15.3	-0.4	-3.9

*Refers to the eye with nonarteritic anterior ischemic optic neuropathy

BMI Body mass index, RAPD Relative afferent pupillary defect. IOP Intraocular pressure. MD Mean deviation

Table 2 Optical coherence tomography findings

	Case 1	case 2	Case 3	Case 4
ODD	**No**	**No**	**Yes**	**No**
BMO, µm				
OD	1355*	1350	1492*	1329*
OS	1445	1383*	1507	1295
GCL Volume, mm^3	4 weeks:	3 weeks:	10 weeks:	3 weeks:
OD	0.92*	1.11	0.76*	0.98*
OS	1.05	0.85*	1.31	1.08
IRF/SRF presence*				
Peripapillary	Yes	Yes	Yes	Yes
Within ETDRS grid	Yes, within grid II	Yes, within grid II	Yes	Yes

The time in weeks indicates the duration between the patient-reported onset of visual symptoms and the GCL measurements

*Refers to the eye with nonarteritic anterior ischemic optic neuropathy. ODD optic disc drusen, BMO Bruch's membrane opening, GCL Ganglion cell layer, IRF/SRF intraretinal fluid / subretinal fluid, ETDRS Early treatment diabetic retinopathy study

Case presentation

All patients in this case-series were diagnosed with NAION, at the Department of Ophthalmology, Rigshospitalet, Denmark in the period from May to December of 2024.

The inclusion criteria were a diagnosis of NAION in the context of semaglutide treatment. All patients had undergone a comprehensive neuroophthalmological examination, and the diagnosis was confirmed by a neuro-ophthalmology expert. At the time of the initial examination, optical coherence tomography (OCT) scans were performed using the DRI-OCT Triton (TopCon Healthcare, Tokyo, Japan). Follow-up scans were conducted with the Heidelberg Spectralis (Heidelberg Engineering, Heidelberg, Germany) spectral-domain OCT. The extent of peripapillary fluid was quantified based on the Early Treatment Diabetic Retinopathy Study, ETDRS grid [6]. The Bruch's membrane opening (BMO) diameter was measured as an average, derived from six radial scans spaced equally and captured using the Heidelberg Spectralis OCT [7]. Blood samples, clinical data, and patient characteristics were collected as part of their diagnostic evaluation.

Patient characteristics of the four cases are listed in Table 1, whereas OCT findings are summarized in Table 2.

Case 1

A 47-year-old man presented with progressively worsening blurred vision in OD over three days. He had no

previous ocular history except for hypermetropia (+ 5.00 D OU). His medical history was significant for obesity, which led to treatment with semaglutide (Wegovy) over the past 11 months, resulting in an 18 kg weight loss. For the preceding six months, he had received weekly injections of 2.4 mg. Upon examination, his visual acuity (VA) was 20/20 in both eyes, and he demonstrated a moderate RAPD in OD. Fundoscopy revealed optic disc edema with subretinal fluid in OD on OCT (Fig. 1). Intraocular pressure (IOP) was measured at 12 mmHg in OD and 13 mmHg in OS. Ishihara color vision testing was normal, and systemic workup revealed no symptoms or laboratory evidence, i.e., C-reactive protein, thrombocytes and sedimentation rate, suggestive of giant cell arteritis

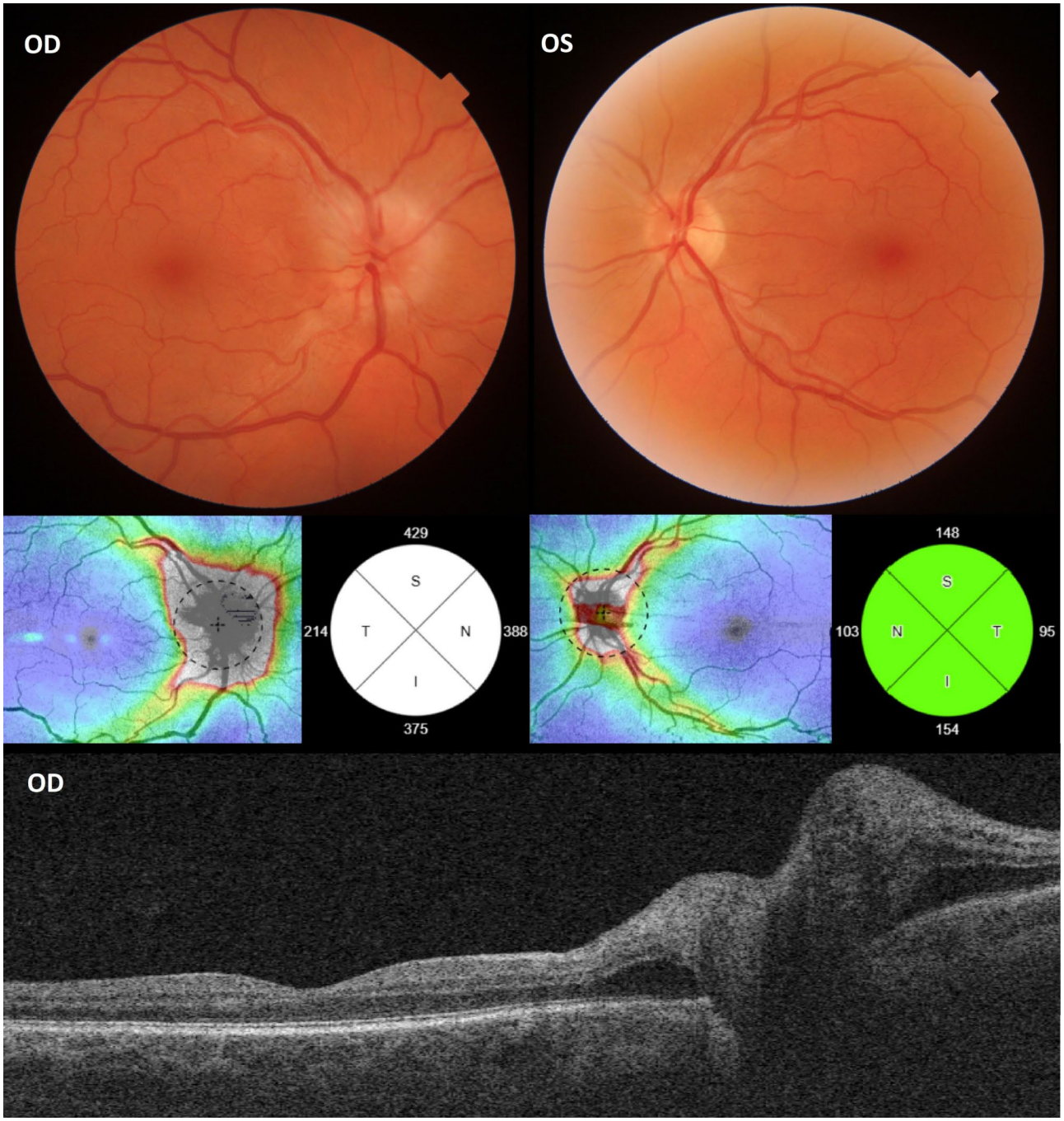

Fig. 1 Clinical manifestation of optic disc edema in Case 1: Upper panel shows the initial fundus photographs of both eyes, OD with optic disc edema. Middle panel shows the optical coherence tomography (OCT)-derived retinal thickness map while the circle diagram illustrates the four segments of the peripapillary retinal nerve fiber layer thickness upon initial examination. Lower panel shows the cross-sectional OCT scan of macula and optic nerve head of OD

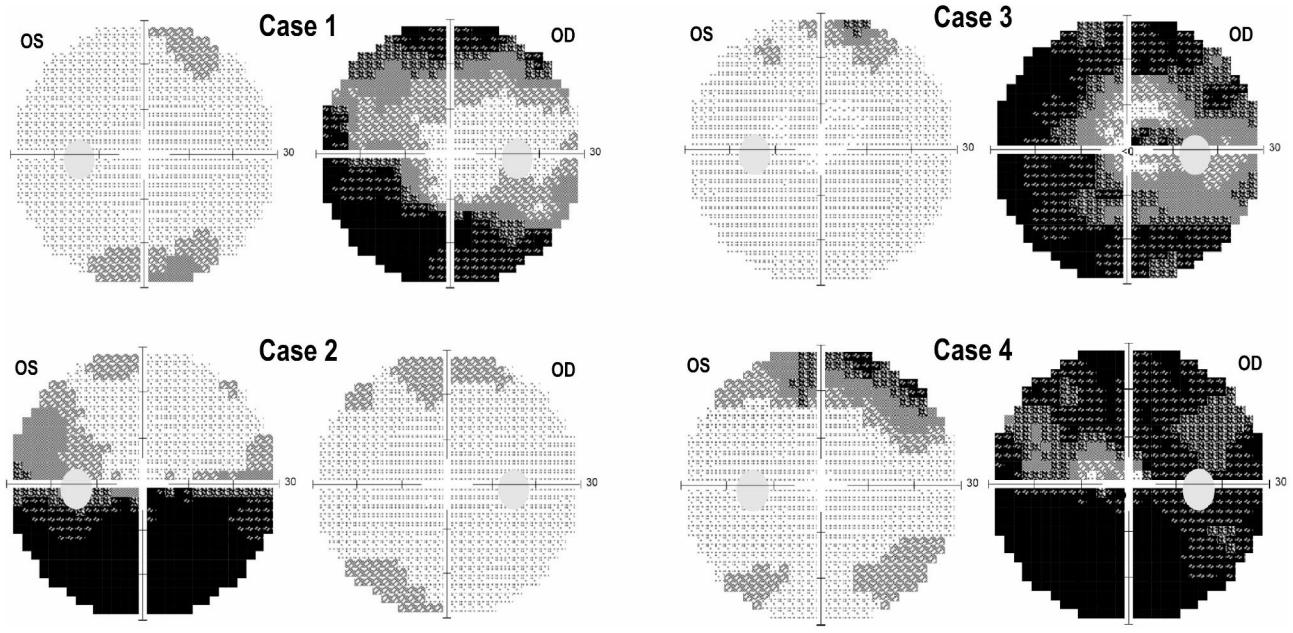

Fig. 2 30-degree standard automated perimetry (grayscale) showing characteristic visual field defects. Case 1: severe lower altitudinal defect in OD, mean deviation (MD) of -13.8 dB. Case 2: Lower altitudinal visual field defect in OS, MD of -15.3 dB. Case 3: Concentric visual field defect with central sparring in OD, MD of -18.8 dB. Case 4: Concentric visual field defect with small central sparring in OD, MD of -23.5 dB

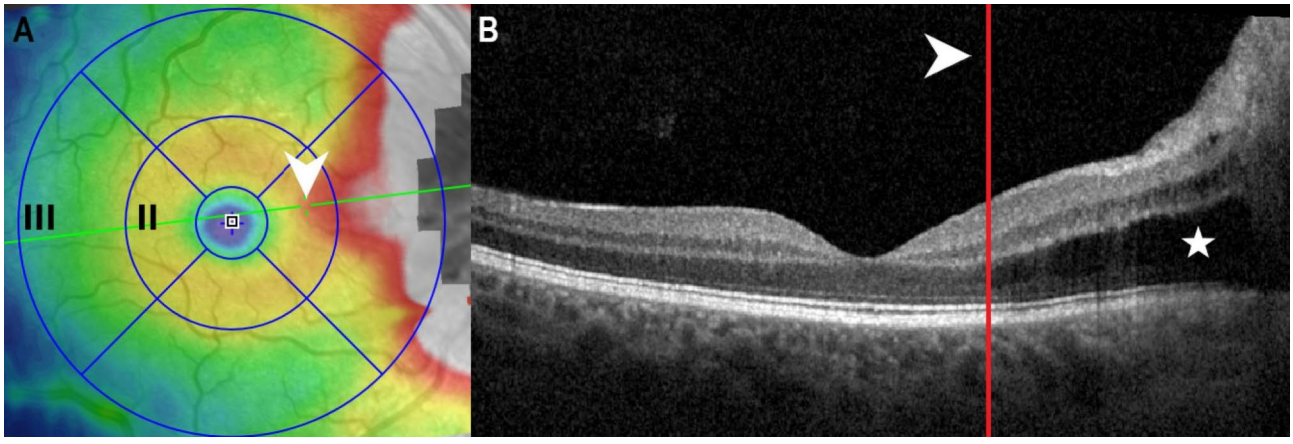

Fig. 3 Intraretinal fluid extension on OCT in Case 1. **(A)** Retinal thickness map with ETDRS grid overlay showing the margin of the intraretinal fluid extending into ring II (Arrowheads). **(B)** OCT B-scan showing the peripapillary intraretinal fluid (Star)

(GCA). OS appeared normal, except for a crowded optic disc without cupping.

At a two-weeks follow-up, the patient had experienced gradual worsening of blurriness in OD. Standard automated perimetry (SAP) revealed an inferior altitudinal visual field (VF) defect with a mean deviation (MD) of -13.8 dB OD (Fig. 2). OCT showed progression of the optic disc edema compared to the initial visit, with intra- and subretinal fluid extending into the macula, within 1.5 mm of the fovea corresponding to ETDRS grid II (Fig. 3). Ophthalmoscopy revealed few small flame-shaped hemorrhages in OD. However, VA remained 20/20 OU.

At one month, the optic disc edema had decreased significantly, and retinal fluid was no longer observed. MD on SAP had improved slightly to -12.7 dB. Macular OCT revealed atrophy of the ganglion cell layer (GCL) corresponding to the VF defect. Enhanced depth imaging OCT (EDI-OCT) of the optic nerve head showed no evidence of ODD. The BMO measured 1355 µm in OD and 1445 µm in OS.

Case 2

A 63-year-old man presented with a five-day history of a progressing dark shadow in the lower VF on OS. His ocular history was significant for focal laser

photocoagulation performed 3 years earlier for a retinal tear in OD and bilateral refractive lens exchange for presbyopia conducted 3 weeks prior. His medical history included arterial hypertension, hyperlipidemia and obesity. To address his obesity, he had been treated with semaglutide (Wegovy) for five months, currently receiving weekly injections of 1.7 mg. On initial examination VA was 20/20 in OD and 20/40 in OS. A moderate RAPD was observed in OS. Fundoscopic examination revealed pronounced optic disc edema in OS, with OCT showing intra- and subretinal fluid extending into the nasal macula reaching fovea centralis. IOP was 15 mmHg in OD and 18 mmHg in OS. He expressed no symptoms supporting GCA and inflammation biomarkers were within normal limits. Funduscopic examination of OD was unremarkable apart from a crowded optic disc and the presence of sequelae from prior focal laser photocoagulation.

On follow-up two weeks later, the patient reported slight improvement in vision, with VA increasing to 20/32 in OS. SAP showed normal VF in OD, while OS exhibited a lower altitudinal defect with MD of -15.3 dB (Fig. 2). The optic disc edema in OS had decreased significantly, leaving residual inferior segmental edema. OCT of the macula revealed hard exudates corresponding to previously present retinal edema, which had resolved. Thinning of the GCL was evident and correlated with the observed VF loss. EDI-OCT of the optic nerve head revealed discrete hyperreflective bands OU, but no ODD. Measurements of the BMO were 1350 µm in OD and 1383 µm in OS.

Case 3

A man aged 60 years presented with a one-day history of intense visual blurring in the VF of OD. His ocular history included only routine diabetic retinopathy screening with all previous evaluations being unremarkable. His medical history included diabetes type 2 over the past 18 years, arterial hypertension, hyperlipidemia, obesity, sleep apnea treated with continuous positive airway pressure, and prior myocardial infarction. Additionally, he occasionally used Tadalafil and had quit smoking following his diabetes diagnosis. He had been treated with semaglutide (Ozempic) at a weekly dose of 1.0 mg for the past five years, with no dose escalation due to side effects. His HbA1c was recorded at 54 mmol/mol.

On examination VA was 20/32 in OD and 20/20 in OS. A moderate RAPD was noted in OD. Funduscopy revealed pronounced optic disc edema in OD, accompanied by peripapillary intraretinal fluid and buried ODD detected on OCT. The intraretinal fluid extended slightly into the macula with the fluid margin 2.4 mm from fovea centralis corresponding to within the ETDRS grid (scan including ETDRS grid was not obtained). IOP of 12 mmHg in OD and 17 mmHg in OS were measured. He expressed no symptoms supporting GCA and inflammatory biomarkers were within normal limits. Examination of OS was unremarkable, aside for crowded optic disc with no cupping and buried ODD detected on OCT.

At a four-week follow-up, the patient's vision deteriorated further to VA 20/40 in OD. SAP of OS was normal, while OD showed concentric VF defect with central sparing and MD of -18.8 dB (Fig. 2). The optic disc edema had partially resolved, remaining mainly in the nasal, superior, and inferior segments. Furthermore, there was no longer intraretinal fluid on OCT, but we observed diffuse thinning of the GCL in OD. The BMO measured 1492 µm in OD and 1507 µm in OS. A cerebral magnetic resonance imaging scan performed due to his comorbidities showed no abnormalities.

Case 4

A 55-year-old man presented to a community-based eye clinic with a five-day history of progressive blurring in the lower VF of OD. He was referred to our department with a suspected diagnosis of NAION. His ocular history was significant for one uncomplicated episode of anterior uveitis in OD. His medical history included only obesity, which was the reason for initiating semaglutide (Wegovy) therapy eight months earlier. He had received injections of 1.7 mg weekly for the past six months. He presented no symptoms suggestive of GCA. Two weeks later, his vision had worsened slightly. VA was 20/32 in OD and 20/16 in OS, with moderate RAPD in OD. Funduscopy revealed optic disc edema with the presence of small splinter hemorrhages. OCT identified peripapillary intraretinal fluid and a crowded optic disc without evidence of ODD. The intraretinal fluid extended just slightly across the margin of the ETDRS grid. Examination of OS was normal, however with a crowded optic disc with no cupping. SAP showed inferior altitudinal defect with severe superior constriction and central sparing, MD of -23.5 dB OD (Fig. 2). OCT revealed peripapillary intraretinal fluid and crowded optic disc but no ODD. Furthermore, early thinning of GCL was evident on OCT. OS appeared normal on examination, although a crowded optic disc with no cupping was noted. BMO measurements were 1329 µm in OD and 1295 µm in OS.

Discussion

In the current case series, we report four instances of NAION occurring in patients treated with semaglutide. All four cases share similar features, including a 'disc at risk' in the fellow eye, characteristic VF abnormalities, and intraretinal fluid. Eventually, the optic disc edema resolved, leaving residual structural damage i.e., GCL atrophy, consistent with the typical presentation of NAION. Anatomical predisposition was evident in all four cases including small BMOs and

crowded discs without cupping. Notably, cases 1, 2, and 4 exhibited BMO diameters below 1.4 mm, markedly smaller than normative values reported by Zhang et al. (1.62 ± 0.28 mm horizontal, 1.74 ± 0.27 mm vertical) and Dai et al. (1.62 ± 0.25 mm horizontal, 1.85 ± 0.26 mm vertical) [8, 9]. We propose that small BMO size is not the sole determinant of susceptibility, as the BMO in the non-affected eyes of cases 2 and 4 was slightly smaller than in the affected eyes. However, whether this small difference holds clinical significance remains uncertain. EDI-OCT showed only ODD in case 3 and only case 1 expressed hypermetropia.

Systemic risk factors varied significantly among the four cases. Case 1 and 4 had only one identified systemic risk factor, namely obesity. Notably, these two cases also had the smallest BMO. In contrast, case 3 presented with multiple systemic risk factors commonly associated with NAION while having the largest BMO among the four cases. Also, the one receiving the lowest dose of semaglutide at the time of NAION onset was case 3. With the caveat that this case series includes only four cases, it appears that narrower optic nerve heads may pose a greater risk for semaglutide-associated NAION, potentially independent of systemic risk factors. Although most studies suggest a slight male predominance in the risk of developing NAION, it is interesting that all four cases in our series were male.

GLP-1 receptors are widely expressed in the human retina and GLP-1 RA have demonstrated multiple beneficial effects, particularly in preclinical studies. These effects include antioxidative and anti-inflammatory properties, restoration of retinal capillary patency under ischemia-reperfusion conditions, promotion of reparative angiogenesis, and inhibition of pathological retinal neovascularization [10]. Despite these benefits, intensive glycemic control in patients with diabetes can worsen diabetic retinopathy, a phenomenon known as 'early worsening', which typically occurs within the first year of intensive treatment [11]. Several studies have reported early worsening following semaglutide treatment, although the underlying mechanism remains unclear [11–14]. The most significant factor associated with this effect appears to be the magnitude of HbA1c reduction [11]. In this context, only case 3 had diabetes and had been treated with semaglutide (Ozempic) for the past five years, whereas cases 1, 2, and 4 had received less than one year of semaglutide therapy. Additionally, case 3 had evident ODD and multiple other risk factors. Thus, case 3 might more accurately be classified as ODD-AION, whereas the other three cases could be categorized as semaglutide associated AION. The mechanism by which GLP-1 RA may contribute to NAION remains unclear but may involve vascular dysregulation or compromised perfusion of the optic nerve head in predisposed individuals. Since, all four cases exhibited a small BMO, the potential phenomenon of 'early worsening' may compound its effects in the presence of a small BMO, increasing retinal nerve fiber layer thickness and potentially amplifying susceptibility to ischemic events. This may explain the pronounced edema and intraretinal fluid observed in all four cases. It is important to emphasize that the role of 'early worsening' in individuals without diabetes remains poorly understood. Also, GLP-1 is known to induce nitric oxide mediated vasodilation, and rodent studies have demonstrated that GLP-1 infusion acutely increases microvascular perfusion [15]. However, whether it effects the vascular regulation of the optic nerve is unknown. Further studies are needed to confirm or reject this hypothesis and to better understand the relationship between semaglutide and NAION.

Interestingly, all four cases in our series presented with unilateral NAION rather than bilateral involvement. This supports a multifactorial model in which semaglutide may amplify ischemic susceptibility in predisposed individuals, such as those with small BMO, ODD or otherwise crowded optic nerve head, rather than acting as a direct neurotoxic agent. In the vast majority of cases, the overall benefits of GLP-1 RAs, including glycemic control, weight loss, and cardiovascular protection, will outweigh this potential risk. Identifying individuals at higher risk may help optimize patient selection and mitigate adverse outcomes.

Initial imaging was performed with DRI-OCT Triton, while follow-up assessment was conducted using Heidelberg Spectralis, introducing potential variability in BMO diameter and RNFL thickness measurements. SS-OCT (Triton) generally reports numerically greater values regarding these parameters than SD-OCT (Spectralis) due to deeper penetration, longer wavelength, and differing segmentation algorithms [16–18]. Given these inconsistencies, direct interchangeability between the two modalities is not recommended, and standardized imaging protocols or regression-based conversion equations should be applied to ensure measurement comparability in longitudinal studies [16]. However, both modalities effectively detect optic nerve head structures such as BMO, ODD and lamina cribrosa [19].

This case series provides valuable insights into a rare but important side effect of semaglutide, supported by detailed clinical documentation and imaging. However, with only four cases and single-center data, this limits the generalizability of our findings and precludes any definitive conclusions regarding causality. Potential confounding factors, such as systemic risk factors and prior ocular surgery may have influenced NAION development, making it difficult to establish a direct causal relationship with semaglutide use. As noted, the technological differences between SS-OCT and SD-OCT, may have introduced

minor variability in measurements. Hence, future studies should implement standardized imaging protocols or establish conversion factors to enhance longitudinal comparability.

Conclusions

In conclusion, this case series of four patients who developed NAION in the setting of semaglutide therapy highlights that all cases in the affected eye exhibited a crowded optic nerve head with small BMO. Interestingly, a smaller BMO diameter appears to correspond to fewer systemic risk factors. Whether individuals with smaller BMO are at higher risk of developing NAION, regardless of systemic risk factors, should be explored in larger patient cohorts.

Abbreviations

NAION	Nonarteritic anterior ischemic optic neuropathy
RAPD	Relative afferent pupillary defect
ODD	Optic disc drusen
GLP-1 RA	Glucagon-like peptide-1 receptor agonist
OCT	Optical coherence tomography
ETDRS	Early Treatment Diabetic Retinopathy Study
BMO	Bruch's membrane opening
VA	Visual acuity
IOP	Intraocular pressure
GCA	Giant cell arteritis
SAP	Standard automated perimetry
MD	Mean deviation
GCL	Ganglion cell layer
EDI-OCT	Enhanced depth imaging optical coherence tomography
VF	Visual field

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Author contributions

HA and SH contributed to the data collection, interpretation and writing of the manuscript. HA prepared the figures. SH was the major contributor in the conception of the work and critical review. Both authors approved the final manuscript.

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Data availability

The datasets utilized and/or analyzed in this study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

Ethics committee approval was not required as the study utilized data obtained through standard patient care practices. The authors declare that they adhered to the CARE guidelines/methodology.

Consent for publication

Informed written consent for publication was obtained from the patients.

Competing interests

The authors declare no competing interests.

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