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CASE REPORT

# Non-Arteritic Anterior Ischemic Optic Neuropathy in an Otherwise Healthy Young Adult Patient Treated with Liraglutide and Semaglutide for Weight Loss: A Cautionary Tale

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**Purpose:** To report a case of non-arteritic ischemic optic neuropathy (NAION) in an otherwise healthy patient treated with Glucagon-Like Peptide-1 (GLP-1) receptor agonists (RAs) liraglutide and semaglutide.

**Observations:** A 47-year-old Caucasian female with a Body Mass Index (BMI) of 27.92, and no known history of diabetes, hypertension, or ischemic heart disease, developed a progressive decline in visual acuity in the right eye one month after initiating liraglutide therapy for weight loss. Upon symptom presentation, ophthalmic examination performed elsewhere revealed a best-corrected visual acuity (BCVA) of 20/40, optic nerve head (ONH) swelling, and inferior hemifield scotoma on Humphrey Visual Field testing. Oral corticosteroids were prescribed and discontinued because of poor glycemic control. Liraglutide therapy was continued for further three months and then switched to semaglutide owing to poor clinical results. Eight months later, the patient discontinued weight loss therapy because of progressive visual deterioration and presented to our clinic for a second opinion. Upon examination, BCVA was limited to 20/400, ONH edema was evident and confirmed on optical coherence tomography (OCT), and further worsening of the visual field defects was detected. Given the absence of anatomical and/or systemic risk factors, NAION secondary to GLP-1 RAs was diagnosed.

**Conclusion and Importance:** In our patient, liraglutide likely served as the initial trigger for the NAION, with semaglutide acting as an additional contributing factor in the progression of the disease. This case adds to the complex puzzle regarding the association between GLP-1 RAs therapy and NAION. Given the increasing use of these drugs for both obesity and diabetes and the close temporal correlations between GLP-1 RAs use and NAION, healthcare providers should be aware of the possible risk of serious ocular complications. Upon onset of visual symptoms, early ophthalmologic diagnosis and treatment interruption are essential to prevent or limit severe visual morbidities.

**Keywords:** non-arteritic anterior ischemic optic neuropathy, liraglutide, semaglutide, glucagon-like peptide 1 receptor agonist, ocular side effects, weight loss

## Introduction

Non-arteritic ischemic optic neuropathy (NAION), a common cause of optic nerve swelling and damage, is characterized by disrupted blood flow to the optic nerve head (ONH), ultimately resulting in a permanent vision loss.[1] Established risk factors include systemic conditions such as hypertension, diabetes mellitus, and hyperlipidemia, as well as anatomical susceptibility often due to a small, crowded optic nerve head predisposing to ischemia. However, the role of pharmacologic agents in precipitating or exacerbating NAION remains an area of ongoing investigation with the potential effects of certain drugs remain still controversial.[1] Although recent studies have reported a connection between NAION and the use of Semaglutide, a Glucagon-Like Peptide-1 (GLP-1) receptor agonist approved by the US Food and Drug Administration (FDA) in December 2017 for type 2 diabetes (T2D) and in December 2022 for obesity, further investigation is needed to fully understand

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the clinical implications of this association.[2,3] Herein, we describe a case of progressive visual loss in an otherwise healthy young adult patient lacking traditional vascular risk factors or predisposing optic disc anatomy who developed NAION after the sequential use of two GLP-1 receptor agonists (RAs) for weight loss.

## Case Report

A 47-year-old Caucasian female with a Body Mass Index (BMI) of 27.92 and no known personal history of diabetes, hypertension, or ischemic heart disease presented to our institution with a 1-year history of progressive and severe visual acuity decline in the right eye (RE). The patient reported that ocular symptoms started one month after initiating therapy with liraglutide, a GLP-1 RA, for weight loss. The initial treatment involved daily administration of liraglutide, with the dosage gradually increased every 10 days from 0.6 mg/day to 2.4 mg/day. After the onset of visual acuity decline, the patient underwent ophthalmological evaluation. The Snellen best-corrected visual acuity (BCVA) was 20/40 in the RE, and fundus examination revealed ONH swelling. All the other ocular findings were unremarkable. Humphrey Visual Field (HVF) testing revealed a scotoma in the inferior hemifield, particularly in the inferotemporal quadrant, with partial involvement of the central field (Figure 1A). The patient was prescribed oral corticosteroids (25 mg/day prednisone), which was discontinued after one month due to poor glycemic control (blood glucose 135 mg/dL). Due to the inadequate clinical response to weight loss therapy, the treatment was switched after four months from liraglutide to semaglutide, another GLP-1 RA. The therapy began with 0.25 mg once weekly for one month, then increased to 0.50 mg weekly for additional 3 months. After an additional 8 months, given the progressive decline in visual acuity, the patient discontinued weight loss therapy and presented to our clinic for a second opinion. Upon examination, BCVA was limited to 20/400 in the RE. Fundus examination revealed ONH edema with prominent vascular tortuosity in the RE (Figure 2A) and normal findings in the left eye (LE) (Figure 2B). Spectral-domain optical coherence tomography (SD-OCT) (Heidelberg Engineering, Heidelberg, Germany) confirmed severe ONH swelling, with thickening of the peripapillary retinal nerve fiber layer (RNFL) and marked thinning of the macular ganglion cell complex (GCC) in the RE (Figure 2C and E). The SD-OCT parameters were within the normal limits in the LE (Figure 2D and F). The Bruch's membrane opening (BMO) diameter, determined as the mean of two linear scans passing vertically and horizontally through the ONH captured using SD-OCT, was 1749 µm in the RE and 1660 µm in the LE. HVF testing showed the progression of the altitudinal defect, with a deep scotoma in the inferotemporal quadrant, diffuse involvement of the inferonasal quadrant, and extension into

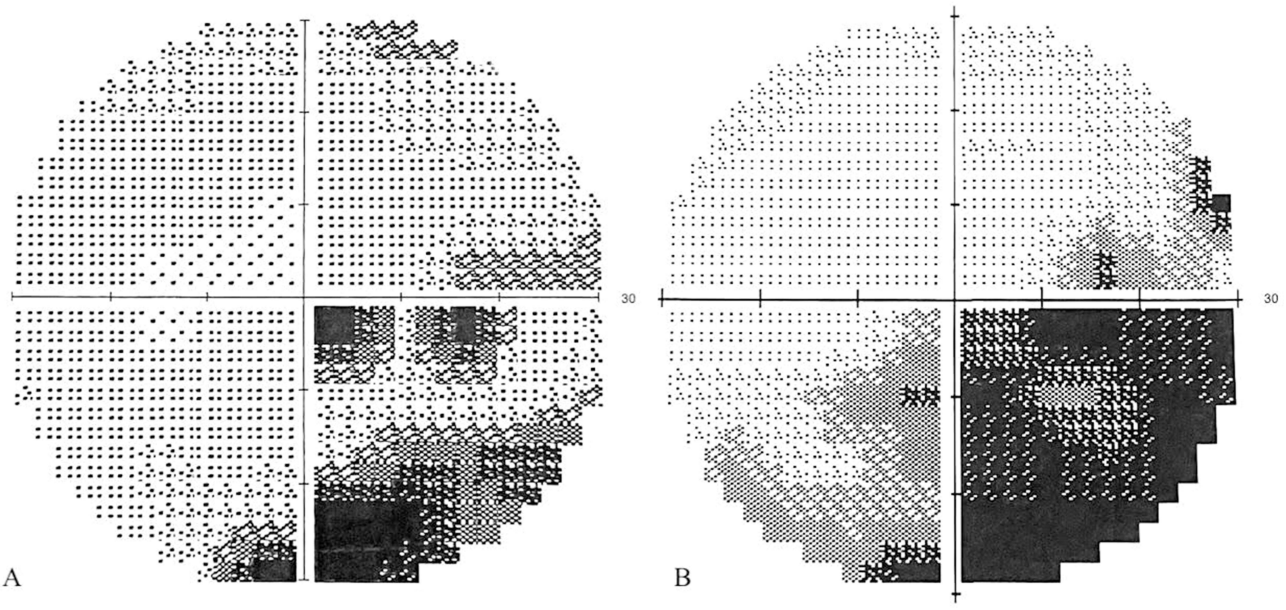


**Figure 1** 30-degree standard automated perimetry (grayscale) showing characteristic visual field defects in right eye (RE). One month after the beginning of liraglutide therapy: deep defect in the inferior hemifield, particularly in the inferotemporal quadrant, with partial involvement of the central field was detected, mean deviation (MD) of −6.87 dB (**A**). Eight months after the beginning of systemic therapy: lower altitudinal defect, with a deep scotoma in the inferotemporal quadrant, diffuse involvement of the inferonasal quadrant and extension into the central field, MD of −12.11 dB (**B**).

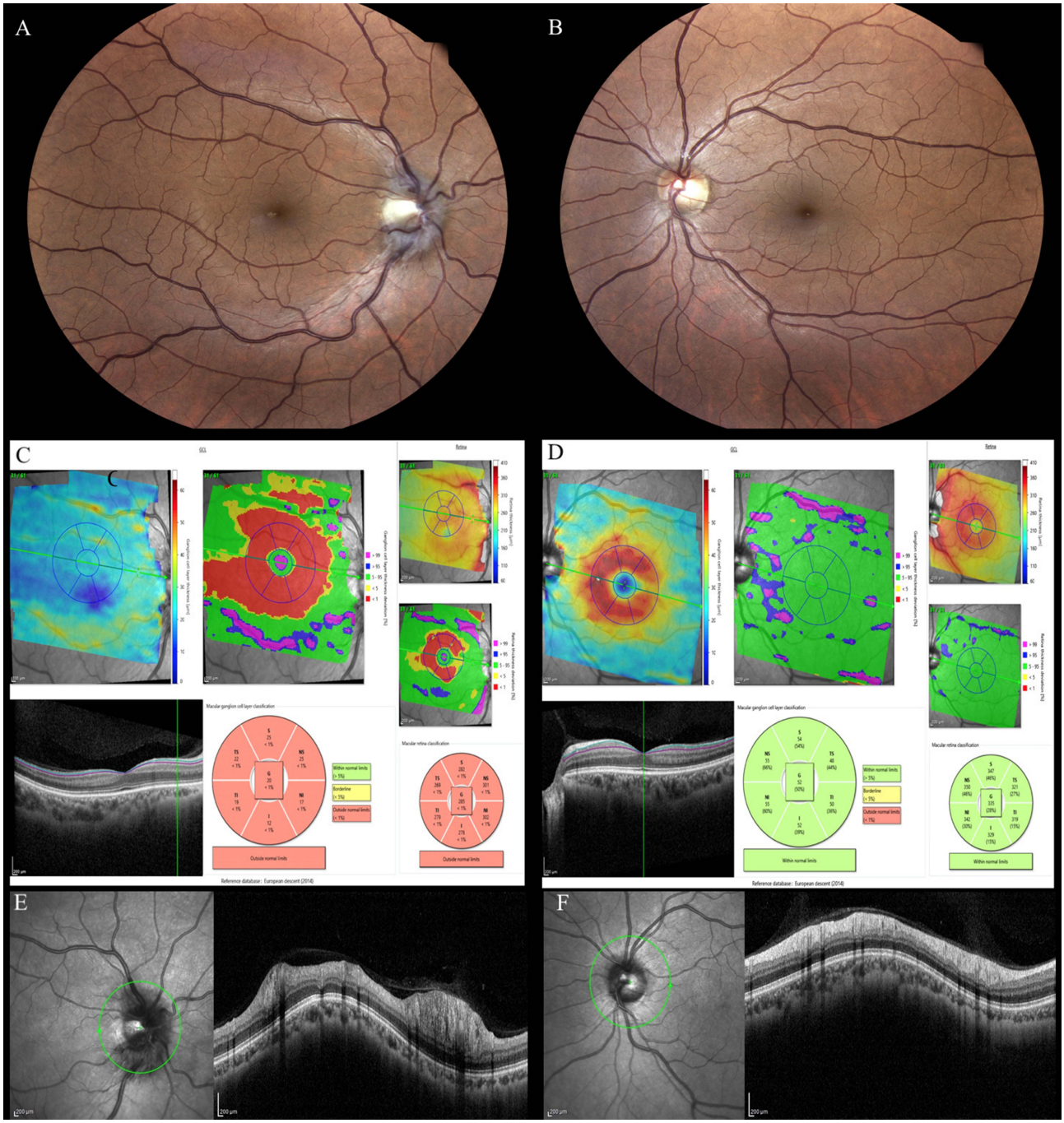


**Figure 2** Fundus photographs of both eyes, right eye (RE) with optic disc edema and vascular tortuosity (**A** and **B**). Optical coherence tomography (OCT)-derived ganglion cell layers map with ETDRS grid overlay showed marked thinning of the macular ganglion cell complex (GCC) in the right eye (**C**). In optic nerve head scans (**E** and **F**), the green circle evidences the circumpapillary retinal nerve fiber layer (RNFL) scan path, used to measure RNFL thickness around the optic disc. OCT B-scan displayed optic nerve head swelling with thickening of the peripapillary RNFL in the affected eye (**E**). The GCC and the RNFL of the left eye (LE) were within limits (**D** and **F**).

the central field (Figure 1B). Given the lack of other anatomical and/or systemic risk factors, a diagnosis of NAION associated with the use of GLP-1 RAs was made.

## Discussion

GLP-1 RAs are FDA-approved drugs that are widely used to treat T2D and obesity. These drugs exert their effects through multiple mechanisms, including enhancement of β-cell function, delayed gastric emptying, and inhibition of glucagon secretion.


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Despite their widespread use and good safety profile, recent studies have documented a possible association between GLP-1 RAs and NAION.[2–6] Hathaway and colleagues were the first to report an increased risk of NAION in individuals treated with weekly semaglutide. In a retrospective analysis of 710 T2D patients and 979 overweight or obese patients, the cumulative incidence of NAION was 8.9% over 36 months among 194 semaglutide users with T2D, with a hazard ratio (HR) of 4.28. Similarly, among 361 overweight or obese semaglutide users, the cumulative incidence over 36 months was 6.7%, with a HR of 7.64.[2] In agreement with these results, a large cohort study of 424152 Danish individuals identified an elevated risk of developing NAION in T2D patients treated with semaglutide. In detail, semaglutide users was associated with a higher incidence rate of NAION (0.228 vs 0.093 per 1000 person-years) than non-users and with a significantly elevated risk of upcoming NAION, with a reported HR of 2.19.[4] Additionally, Ahmadi and Hamann recently described 4 cases of male patients who developed unilateral NAION while receiving semaglutide treatment. All patients exhibited anatomical predispositions, presenting with a "disc at risk" configuration characterized by a crowded optic disc without cupping and a small BMO (diameters below the normative value of 1.6–1.8 mm). These patients were treated with doses of semaglutide ranging from 1 mg to 2.4 mg once weekly.[5] Regarding liraglutide, only a recent study reported an association between its use and NAION development. In detail, of 2446482 patients with diabetes undergoing therapy, a 179% likelihood of being diagnosed with NAION was reported in patients receiving liraglutide treatment.[6] Conversely, two retrospective studies reported an absent or only slightly increased risk of NAION after semaglutide administration.[3,7] In an analysis involving approximately 300000 individuals, Chou and coauthors reported that semaglutide use was not associated with an increased risk of NAION compared to non–GLP-1 RA users. This finding remained consistent across various subgroups, including patients with differing baseline comorbidities (T2DM only, obesity only, or T2DM with obesity), as well as across different follow-up periods (1, 2, or 3 years).[7] Also, Cai and colleagues in a large multicenter study on 37.1 million patients with T2D described a modest increase in the risk of NAION among individuals with T2D associated with semaglutide use.[3]

Given the normal BMO diameter, the absence of other systemic predisposing conditions, such as hypertension, diabetes mellitus, and hypercoagulability, and the temporal relationship between the drugs intake and the ocular disease, a plausible association between GLP-1 RAs administration and NAION may be considered in our case. However, the exact pathogenic mechanism by which GLP-1 RAs elevate the risk of NAION remains unclear. Stimulation of GLP-1 receptors on the optic nerve or GLP-1 RA-induced activation of the sympathetic nervous system can alter ONH perfusion, thereby potentially increasing the risk of NAION.[2–4]

To our knowledge, this is the first comprehensive report of NAION occurring in a slightly overweight, healthy young adult after liraglutide use, confirming a potential link between GLP-1 RAs therapy and the development of complications at the ONH level.

## Conclusion

In conclusion, this case supports a potential association between GLP-1 receptor agonists and the development of NAION, with liraglutide likely contributing to disease onset and semaglutide potentially involved in its progression. While GLP-1 RAs are widely regarded as effective and generally safe treatments for T2D and obesity, their increasing use raises concerns about rare but serious ocular adverse events such as NAION. Clinicians should maintain a high index of suspicion, conduct thorough risk assessments, and provide appropriate patient counselling when initiating GLP-1 RA therapy. If visual symptoms arise during therapy, prompt ophthalmological investigation and treatment interruption are required. The conflicting evidence in the current literature highlights the urgent need for larger prospective studies to more accurately quantify the risk of NAION, as well as the development of clear, evidence-based monitoring guidelines. Additionally, a better understanding of the underlying pathogenic mechanisms, including potential effects on ONH perfusion or anatomical susceptibility, is essential to inform clinical decision-making and improve patient safety.

## Claims of Priority

After conducting a literature review on 23/03/25 utilizing PubMed, Google Scholar using the key words ("liraglutide"+"NAION" or "Optic neuritis"), we did not find any prior reports of NAION occurring in a slightly overweight healthy young adult after liraglutide use.

## Patient Consent

Written informed consent was provided by the patients to have the case details and any accompanying images published.

## Institutional Approval

Institutional review board (IRB) approval was not required for the publication of the case details.

## Informed Consent Statement

Informed consent was obtained from the patient.

## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work All authors attest that they meet the current ICMJE criteria for Authorship.

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## Disclosure

The authors declare no conflicts of interest in this work.

## References

1. Raizada K, Margolin E. Non-arteritic anterior ischemic optic neuropathy [Internet]. In: *StatPearls*. Treasure Island (FL):StatPearls Publishing;2025. http://www.ncbi.nlm.nih.gov/books/NBK559045/.
2. Hathaway JT, Shah MP, Hathaway DB, et al. Risk of nonarteritic anterior ischemic optic neuropathy in patients prescribed semaglutide. *JAMA Ophthalmol*. 2024;142(8):732–739. PMID: 38958939; PMCID: PMC11223051. doi:10.1001/jamaophthalmol.2024.2296
3. Cai CX, Hribar M, Baxter S, et al. Semaglutide and nonarteritic anterior ischemic optic neuropathy. *JAMA Ophthalmol*. 2025;143(4):304–314. PMID: 39976940; PMCID: PMC11843465. doi:10.1001/jamaophthalmol.2024.6555
4. Grauslund J, Taha AA, Molander LD, et al. Once-weekly semaglutide doubles the five-year risk of nonarteritic anterior ischemic optic neuropathy in a Danish cohort of 424,152 persons with type 2 diabetes. *Int J Retina Vitreous*. 2024;10(1):97. PMID: 39696569; PMCID: PMC11657653. doi:10.1186/s40942-024-00620-x
5. Ahmadi H, Hamann S. Anterior ischemic optic neuropathy in patients treated with semaglutide: report of four cases with a possible association. *BMC Ophthalmol*. 2025;25:132. doi:10.1186/s12886-025-03958-4
6. Bartelt K, Swaminathan S, Joyce B, Allen S, Deckert J. Liraglutide and insulin prescriptions associated with increased likelihood of rare vision loss; 2024 [cited March 15, 2025]; Available from: https://epicresearch.org/articles/liraglutide-and-insulin-prescriptions-associated-with-increased-likelihood-of-rare-vision-loss. Accessed August 5, 2025.
7. Chou CC, Pan SY, Sheen YJ, et al. Association between semaglutide and nonarteritic anterior ischemic optic neuropathy: a multinational population-based study. *Ophthalmology*. 2024;132(4):S0161–6420(24)00685–7. PMID: 39491755. doi:10.1016/j.ophtha.2024.10.030

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