

Non-Arteritic Ischaemic Optic Neuropathy Associated with Semaglutide Use

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Abstract

Presentation

A 50-year-old obese male with non-insulin dependent diabetes mellitus (NIDDM) taking Semaglutide, a glucagon-like peptide-1 receptor agonist (GLP-1RA) presented with sudden, painless vision-loss from his right eye.

Diagnosis

Right eye visual acuity was 6/24. There was Frisen grade IV optic nerve head (ONH) oedema. This was confirmed at imaging with ocular coherence tomography (OCT). The patient was diagnosed with non-arteritic anterior ischaemic optic neuropathy.

Treatment

Semaglutide was ceased.

Discussion/Conclusion

GLP-1RAs such as Semaglutide are being increasingly recognised as a potential risk factor for NAION. A case such as this has not yet been reported in Ireland.

Introduction

Non-arteritic ischaemic optic neuropathy (NAION) is an optic neuropathy that can cause permanent vision loss¹. Differential diagnoses include 'arteritic' anterior ischaemic optic neuropathy secondary to giant cell or temporal arteritis and optic neuritis with papillitis. Semaglutide has been approved in Europe since 2018 for the treatment of diabetes and obesity. A relationship between GLP-1RAs and NAION has been acknowledged in the literature, with a reported incidence of 1:10,000².

Case Report

A 50-year-old male presented to an ophthalmology emergency department with a nine-day history of sudden, painless vision-loss from his right eye.

His past medical history was significant for NIDDM, obesity with body mass index 32.1kg/m², hyperlipidaemia and obstructive sleep apnoea. He was a smoker. He had been taking Semaglutide 1mg weekly for two years.

On examination, visual acuity was 6/24 and 6/5 in his right and left eyes respectively. There was a right relative afferent pupillary defect (RAPD) and Frisen grade IV ONH swelling with pallor, nerve fibre layer haemorrhages and consequent small vessel obscuration. The left ONH was a small, crowded 'disc-at-risk'. Ocular coherence tomography (OCT) demonstrated right ONH oedema with retinal nerve fibre layer (RNFL) 386um. An inferonasal visual field defect was apparent at Goldmann visual field testing. (*Figure Legend - Figure 1*)

Haemoglobin A1c (HbA1c) was elevated at 44mmol (normal range <42mmol) while C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were both within normal range at <5mg/L and <15mm/hour respectively. No abnormality was identified at computed tomography (CT) of brain and orbits.

The patient ceased Semaglutide and was advised to attend his general practitioner (GP) for consideration of alternative medications for glycaemic control in addition to input regarding optimisation of his blood pressure control and serum cholesterol reduction.

Six weeks later, right eye visual acuity had improved to 6/18. Right ONH swelling had resolved with RNFL 105um at OCT. His visual field defect had reduced in size at Goldmann visual field testing. (*Figure Legend - Figure 2*)

Discussion

The pathophysiology of NAION is poorly understood. It is postulated to be due to a lack of perfusion to the short ciliary arteries supplying the ONH. A compartment-like syndrome develops that compresses the axons of the ONH and results in apoptosis of retinal ganglion cells³. How GLP-1RAs might contribute to an increased risk of NAION is unclear. One theory is that GLP-1 receptors at the ONH are stimulated and activate the sympathetic nervous system which then alters ONH perfusion, potentially increasing the risk of NAION⁴.

There have been multiple studies supporting an association between GLP-1RAs and NAION. One study found an increased incidence of NAION in patients with NIDDM taking Semaglutide (8.9%) compared to controls (1.8%)⁴. Another found Semaglutide doubled the five-year-risk

of NAION in patients with NIDDM⁵. These findings have been supported elsewhere in the literature^{6, 7}.

Management of GLP1-RA-related NAION is that for NAION 'in general' and involves addressing cardiovascular risk factors to optimise vascular supply to the ONH to reduce the risk of a sequential event in the fellow eye (15-20% over 5-years)⁸. This often involves the control of blood pressure with anti-hypertensive agents, addressing hyperlipidaemia with statins and fibrates and supporting smoking cessation.

This patient had multiple risk factors for NAION – male gender, NIDDM, hypertension, hyperlipidaemia, obstructive sleep apnoea, and smoking. The European Medicines Agency recommends ceasing Semaglutide immediately in patients who develop NAION³, while the American Academy of Ophthalmology recommends an individualised approach when considering cessation, advocating consideration of the individuals' overall health, risk factors, and alternative therapeutic options⁹.

Visual outcomes following NAION, including that related to Semaglutide, vary from visual recovery to bilateral, sequential NAION¹⁰.

To our knowledge, this is the first description of Semaglutide-related NAION in Ireland. It is important to have a high suspicion of the same in patients taking GLP-1RAs presenting with acute visual disturbance, to allow for early detection, consideration of alternative glycaemic control and modification, where possible, of associated risk factors⁷.

Declaration of Conflicts of Interest:

None declared.

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Figure Legend

Figure 1. (a) Fundal image of right pale optic nerve head with Frisen grade IV oedema and nerve fibre layer haemorrhages with consequent small vessel obscuration. (b) OCT of the right optic nerve head confirmed the presence of oedema. Retinal nerve fibre layer (RNFL) thickness was 386um. (c) Right eye Goldmann visual field with marked restriction of the inferonasal field.

Figure 2. (a) Fundal image of right pale optic nerve head, six weeks following initial presentation with Frisen grade II optic nerve head swelling and diminishing nerve fibre layer haemorrhages. (b) Right eye Goldmann visual field with reduced restriction of the inferonasal field.

Figures

Figure 1.

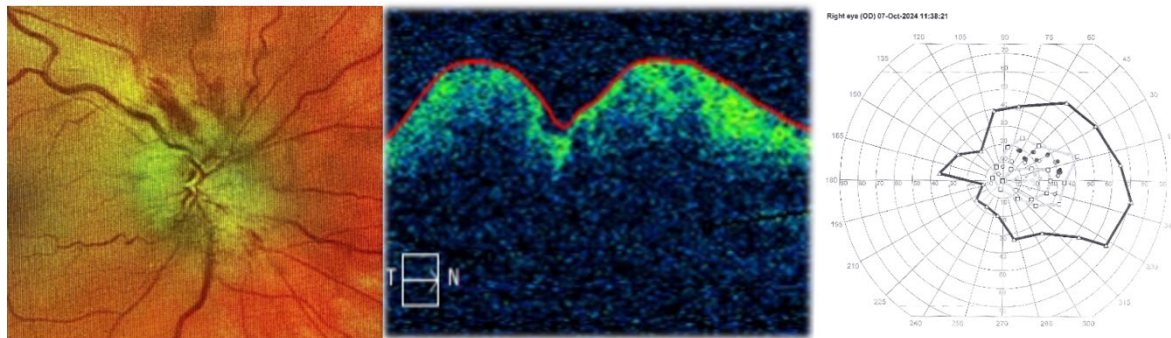


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Figure 2.

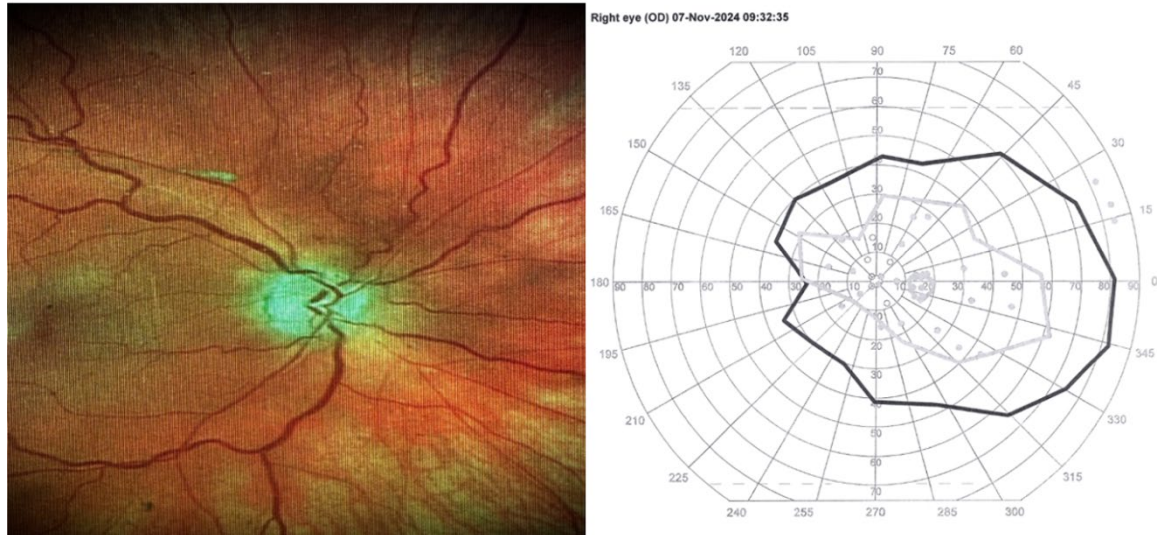


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