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Sudden Altitudinal Visual Field Loss in a Healthy Patient Following Dose Escalation of Tirzepatide (Mounjaro): A Case Report

BACKGROUND

Tirzepatide, marketed as Mounjaro/Zepbound, is increasingly used for weight management and glycaemic control due to its dual GLP-1/GIP receptor agonist activity and favourable metabolic profile. Although ocular adverse events are considered uncommon, they remain poorly characterised, and concerns regarding safety persist in the literature. Emerging reports of sudden visual disturbance, particularly following dose escalation, raise the question of whether rapid metabolic or haemodynamic changes may contribute to optic nerve ischaemia in susceptible individuals. Here, we present a case of acute visual field loss in a patient without conventional vascular risk factors, highlighting the need for increased clinical awareness of potential ocular adverse events associated with tirzepatide.

OBJECTIVE

To describe an atypical case of sudden altitudinal visual field loss in a healthy, non-diabetic patient following a recent dose escalation of Tirzepatide, and to consider the possible association between GLP-1/GIP agonist therapy and non-arteritic ischaemic neuropathy (NAION) in individuals without conventional risk factors.

RELATED LITERATURE

- Katz BJ, et al. Ophthalmic Complications Associated With the Antidiabetic Drugs Semaglutide and Tirzepatide. JAMA Ophthalmology. 2025. PMID: 39883468.
- Wang L, et al. Semaglutide or Tirzepatide and Optic Nerve and Visual Pathway Disorders in Type 2 Diabetes. JAMA Network Open. 2025; PMID: 40788646.
- Lakhani M, et al. Association of Glucagon-Like Peptide-1 Receptor Agonists With Optic Nerve and Retinal Adverse Events: A Population-Based Observational Study Across 180 Countries. American Journal of Ophthalmology. 2025. PMID: 40383360.

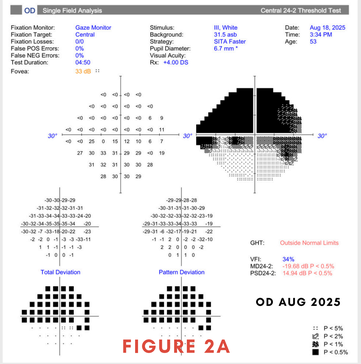
CASE DESCRIPTION

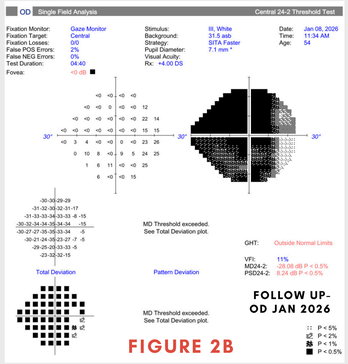
A 54-year-old woman presented with sudden-onset painless superior visual field loss in the right eye (Figure 2A). On a background history of migraine, prior ENT and orthopaedic procedures, an allergic disorder, and a mild right eye chemical injury. She had no history of diabetes, hypertension, hyperlipidaemia, hypermetropia, or sleep apnoea. At presentation, visual acuity was 6/4.8 bilaterally with a right relative afferent pupillary defect. The anterior segments were quiet in both eyes, with clear corneas, deep and quiet anterior chambers, normal iris architecture, no signs of intraocular inflammation, and mild bilateral cortical lens opacities. Intraocular pressures were within normal limits.

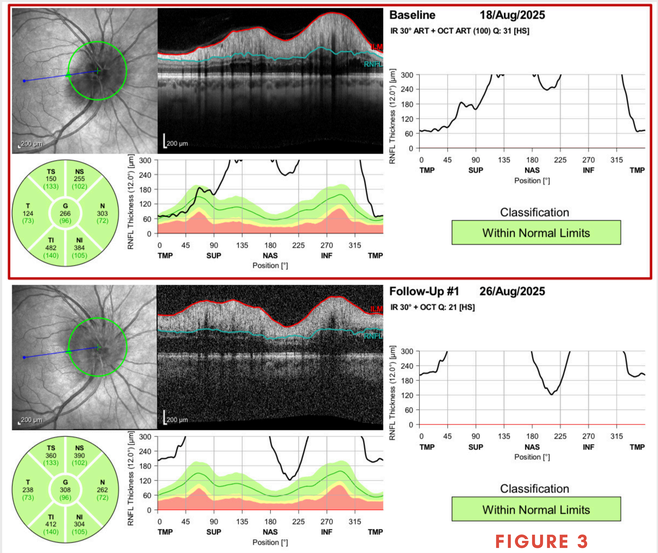
Dilated fundus examination of the right eye showed a swollen, hyperaemic optic disc with blurred margins consistent with optic disc oedema, without peripapillary or retinal haemorrhages, cotton wool spots, retinal emboli, cherry-red spot, venous engorgement, or macular abnormality (Figure 1A). The left optic disc was normal with a cup-to-disc ratio of 0.3 and healthy neuroretinal rim. Fundus fluorescein angiography demonstrated delayed capillary filling of the right optic nerve head without evidence of retinal vascular occlusion. OCT carried out demonstrated retinal nerve fibre layer thickening and optic disc swelling (Figure 3). CT head was normal, nil acute.

Inflammatory markers were not significantly raised (ESR 14 mm/hr, CRP 7.6 mg/L), and there were no clinical features of giant cell arteritis. Full blood count showed neutrophilia and lymphocytosis. She was started on oral prednisolone 40 mg daily without improvement, with subsequent deterioration of right visual acuity to 6/30 (left 6/4.8). Her medications included propranolol, amitriptyline, candesartan, atogepant, fremanezumab, and Botox, and she had been taking Tirzepatide for 7 months for weight loss, with recent dose escalation preceding symptom onset. There were no established vascular risk factors for non-arteritic anterior ischaemic optic neuropathy. The clinical picture was consistent with NAION, agreed by multiple clinicians, despite the absence of classical risk factors.

RESULTS/FINDINGS







DISCUSSION

Emerging literature suggests a possible association between GLP-1 receptor agonists, including tirzepatide, and acute optic nerve events. Case reports (Katz et al.) describe sudden visual loss and optic disc swelling following initiation or dose escalation, while larger observational data (Wang et al.) indicate a small increased risk of NAION and related optic neuropathies, mainly in diabetic populations. Pharmacovigilance evidence, including the large population-based study by Lakhani et al. (2025), also demonstrates disproportionate reporting of optic nerve and retinal adverse events, supporting a potential safety signal without confirming causality. Overall, these findings raise concern for a possible class effect, although mechanisms remain unclear and may involve non-glycaemic factors such as rapid weight loss or vascular changes, warranting further investigation and setting the context for the present case.

CONCLUSION

This case describes NAION in a non-diabetic patient following tirzepatide dose escalation, with a temporal relationship and absence of typical vascular risk factors making it a notable observation, although causality cannot be confirmed. It underscores the need to consider medication-related causes, including GLP-1/GIP agonists, in patients with sudden unexplained visual field loss and optic disc swelling. Clinicians should remain aware of potential under-recognised acute optic nerve ischaemic events, with prompt investigation, multidisciplinary input, and continued pharmacovigilance to better define risk and causality.