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Semaglutide and the Eye: Indian Expert Commentary.

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Abstract: There has been a significant change in the management algorithms and clinical use of semaglutide due to its multiple benefits includingglycemic reduction, weight loss, cardiovascular effects, renal protection, hepatic benefits amongst others. The role of semaglutide in popularising the class of GLP-1 receptor agonists cannot be denied. Despite the clear proven benefits, there has been some concernregarding potential eye-related adverse effects – particularly diabetic retinopathy and non-arteritic anterior ischemic optic neuropathy (NAION) with GLP-1 based therapies including semaglutide. This comprehensive review by a panel of expert diabetologists and ophthalmologists is an attempt to evaluate the available clinical data and arrive at a conclusion regarding the causal association of these adverse effects with the use of semaglutide (limited to the innovator brand of semaglutide in the doses of 0.25 mg, 0.5 mg and 1.0 mg for the use in people with type 2 diabetes mellitus) and provide guidance to every physician regarding its use.

Keywords: GLP-1, semaglutide, Ozempic, diabetic retinopathy, NAION

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INTRODUCTION

Glucagon-like peptide-1 receptor agonists (GLP-1RAs), particularly semaglutide, provide substantial metabolic and organ-protective benefits. Semaglutide provides powerful glycemic control, produces clinically meaningful weight loss, lowers major adverse cardiovascular events by ~20–26%, reduces kidney disease outcomes by ~24%, improves functionality in peripheral arterial disease, and has also been associated with improved liver outcomes, including higher odds of MASLD/MASH improvement. Overall, the benefit–risk profile of semaglutide remains strongly favourable, with well-established benefits outweighing rare and generally manageable adverse effects—most commonly gastrointestinal events such as nausea, vomiting, diarrhoea, and constipation. However, there have been emerging reports of ocular safety signals with GLP-based therapies (GLP-1 receptor agonists and GIP-GLP dual agonist), which are discussed in detail in this comprehensive review.[1,2]

Rationale for intensive glycaemic control in diabetes mellitus and concept of "early worsening of retinopathy"

Diabetes mellitus is a leading cause of preventable blindness worldwide, largely due to diabetic retinopathy (DR) and diabetic macular oedema. DR affects approximately one-third of people with diabetes globally (~95 million), and prevalence increases steeply with longer duration of T2D with ~18% when diabetes duration is <10 years, rising to ~51–52% after ≥10 years.[3–5]In real-world analyses of retinopathy complications, higher-risk patients tended to have longer diabetes duration, higher baseline HbA1c (9.4% vs 8.7%), greater insulin use (65% vs 58%), and a high prevalence of pre-existing DR (83.5%), underscoring the importance of baseline retinal status and overall glycaemic burden.[6]

The Diabetes Control and Complications Trial (DCCT) in type 1 diabetes and the UK Prospective Diabetes Study (UKPDS) in type 2 diabetes established beyond doubt that chronic hyperglycaemia is a key modifiable driver for development of microvascular complications. In DCCT, intensive insulin therapy achieving an HbA1c of around 7.0% (vs ~9.0% in the conventional group) reduced the risk of onset of DR by ~76% and progression of established DR by ~50% and also reduced microalbuminuria and clinical nephropathy.[7] Long-term follow-up in the EDIC study showed a "metabolic memory" or legacy effect, with persistent protection from microvascular and later cardiovascular events despite subsequent convergence of HbA1c between groups.[8]

In newly diagnosed type 2 diabetes, UKPDS intensive therapy (sulfonylurea/insulin, and metformin in overweight patients) reduced HbA1c by ~0.9% and resulted in a ~25% reduction in microvascular endpoints, driven predominantly by need for retinal photocoagulation.[9] Long-term follow-up demonstrated sustained reductions in microvascular disease and, over time, lower rates of myocardial infarction and all-cause mortality.[10] These pivotal trials underpin contemporary guideline recommendations to achieve and maintain near-target HbA1c early in the course of diabetes to minimise ocular and renal complications.[11,12]

Early worsening of retinopathy with rapid glycaemic improvement: lessons from DCCT and UKPDS

An important but unclear phenomenon in DCCT and other trials was "early worsening" of DR in a subset of patients starting intensive glycaemic control, particularly those with:

- pre-existing moderate–severe DR
- high baseline HbA1c
- rapid, large falls in HbA1c over a short period (e.g. ≥1–2% within months).[7]

A similar pattern has been described in UKPDS and subsequent studies: rapid glucose normalisation can temporarily destabilise the microvasculature. However, this early worsening did not negate the long-term benefit of intensive control. Over years of follow-up, the intensive arms in DCCT/EDIC and UKPDS had substantially less progression of retinopathy, nephropathy and neuropathy than conventional arms. Thus, early worsening is best understood as a short-term, predictable response to rapid metabolic change in already damaged microvasculature, rather than a harmful effect of intensive therapy per se. This historical experience is critical context when interpreting retinopathy signals with potent glucose-lowering agents.

Retinopathy and semaglutide: Interpreting SUSTAIN-6

Once-weekly semaglutide is a GLP-1 receptor agonist that produces large, rapid reductions in HbA1c and clinically meaningful weight loss. [13–15] In the cardiovascular outcomes trial SUSTAIN-6, semaglutide (0.5 or 1.0 mg weekly) significantly reduced 3-point major adverse cardiovascular events (3-P MACE) by ~26% vs placebo in high-risk T2DM, on top of existing standard of care.[16] Though the trial was not designed to assess retinopathy, an increased rate of adjudicated DR complications was observed: events occurred in ~3.0% of semaglutide-treated vs ~1.8% of placebo-treated participants, with a hazard ratio of about 1.7 (95% CI 1.11–2.78; $P = 0.02$).[16]

Subsequent analyses and expert reviews have consistently shown that this signal was mainly observed in a specific subgroup: patients with pre-existing advanced DR at baseline, long-standing diabetes, very high baseline HbA1c, and large, rapid HbA1c reductions early after randomisation, frequently on background insulin therapy. In participants without baseline retinopathy, the absolute rate of DR events was low and similar between groups. Till date, no mechanistic data demonstrate direct retinal toxicity of semaglutide. Also,while immunohistochemistry localization studies show equivocal and heterogenous results about the presence of GLP-1 receptor expression in retinal tissue, it is unlikely that GLP-1-RAs has a direct toxic effect on the retina.[17]Professional societies and guideline groups therefore interpret the SUSTAIN-6 DR findings as another manifestation of the classic early-worsening phenomenon seen in DCCT and UKPDS, triggered here by semaglutide's potency and speed of HbA1c reduction. However, these are temporary changes as early worsening is typically seen in the first 6–12 months after rapid control and stabilises or improves thereafter, and the US-FDA recommends intensive glycemic control for better long-term outcomes.[11,12]As in DCCT/UKPDS, the expected long-term effect of improved glycaemic control is favourable for DR risk, provided patients are appropriately monitored and treated in collaboration with ophthalmology.

Though it lacks standardised DR grading, recent real-world data from the AAO IRIS Registry suggest that, in adults aged 51–75 years with T2D treated for 2 years with injectable semaglutide (n>48,000; 2013–2021), worsening of DR was uncommon in eyes with no or background DR (2.2%) and in early NPDR (3.5%), while almost 60% of eyes with severe NPDR or PDR actually improved over follow-up, indicating that overall retinal outcomes may be favourable when glycaemia is optimised and patients are appropriately monitored.[18]

NAION: Definition, pathophysiology and baseline risk

Non-arteritic anterior ischaemic optic neuropathy (NAION) is an acute ischaemic optic neuropathy characterised by sudden, painless monocular vision loss, optic disc swelling and corresponding visual-field defects. It is believed to result from hypoperfusion of the arteries supplying the optic nerve head. It is very rare, with an incidence of about 2–10 cases per 100 000 person-years though diagnosis is often clinical and under-coded, which may lead to both under- and mis-reporting in pharmacovigilance datasets.

Established risk factors for NAION include:

- age >50 years;
- vascular risk factors (hypertension, type 2 diabetes, dyslipidaemia, smoking);
- obstructive sleep apnoea and nocturnal hypotension;
- small, crowded optic discs ("disc at risk");
- a history of NAION in the fellow eye.

A variety of systemic conditions and medications have been temporally associated with NAION, but causality is difficult to prove because underlying vascular risk is often high. Thus, patients with diabesity (combination of diabetes and obesity), often with hypertension and sleep apnoea are at intrinsically higher risk of NAION regardless of specific glucose-lowering drug choice. This background risk is critical when interpreting any apparent clustering of NAION cases in GLP-1RA-treated populations.[19]

Semaglutide and NAION: What does the evidence show?

Recent observational and pharmacovigilance reports have raised concerns about a possible association between semaglutide and NAION, based mainly on small, retrospective cohorts in which NAION appeared more frequently among semaglutide-treated patients than among some comparators. These analyses are limited by small event numbers, confounding by indication, incomplete adjustment for vascular risk and the absence of randomisation. In a retrospective analysis from a neuro-ophthalmology referral cohort, the 3-year cumulative incidence of NAION was higher in semaglutide users than comparators (e.g., 8.9% vs 1.8% in T2D; 6.7% vs 0.8% in overweight/obesity). However, these rates likely overestimate real-world risk because the population is biased for older, high-risk patients and ophthalmology specialist ascertainment (diabetes/ hypertension / obstructive sleep apnoea, "crowded" discs), and the design demonstrates association and not causation.[20]

Another study pooling mainly observational/pharmacovigilance data reported an increased NAION signal with semaglutide (pooled HR ~2.62) with time-dependency (more evident after ≥2 years exposure). Still, the evidence is vulnerable to confounding by indication and residual vascular-risk confounding (semaglutide users often have longer diabetes duration, higher HbA1c/BMI and comorbidity), and the signal appears concentrated in older males and those with longer diabetes duration, higher HbA1c, pre-existing diabetic retinopathy, and obesity.[21] Even histopathologic data in NAION were limited (n ≈ 3–6 across reports) and frequently obtained from atypical or late-stage cases, lacking correlation with acute clinical findings.[22] Recently EMA's Pharmacovigilance Risk Assessment Committee (PRAC) reviewed all available data on NAION with semaglutide, including data from non-clinical studies, clinical trials, post-marketing surveillance and the medical literature. PRAC concluded that NAION is a very rare side effect of semaglutide, potentially affecting up to 1 in 10,000 [2] Unlike EMA, the US-FDA has not recommended addition of NAION as an adverse drug reaction of semaglutide in the label. Semaglutide has been studied in clinical programs involving over 60,000 patients and post-marketing use in >49 million patient years of exposure. The innovator company has reviewed data sources related to its semaglutide brands for NAION and the cumulative data does not suggest a causal relationship between semaglutide and NAION. The follow-on semaglutide brands are new to market and hence do not have such analyses performed as of today. In contrast, Silverii et al. conducted a meta-analysis of 69 randomized controlled trials, including 144,226 patient-years in GLP-1 RA arms and 132,922 patient-years in control arms. Across these trials there were 8 cases of ischaemic optic neuropathy in patients receiving a GLP-1 RA versus 4 cases in comparators; 6 events occurred on semaglutide and 1 each on liraglutide and dulaglutide. The pooled fixed-effects estimate showed no statistically significant excess risk (OR 1.53, 95% CI 0.53–4.44; p = 0.43). The approximate incidence was 5.6 vs 3.0 cases per 100,000 patient-years in GLP-1RA and control arms, respectively, implying an absolute risk increase of 2.6/100,000 patient-years and an estimated number needed to harm ≈ 38,460 patient-years, consistent with background epidemiology and insufficient to confirm or exclude a small drug-related effect.[19]

A pooled analysis of all completed sponsor-run phase 2–4 placebo-controlled RCTs of semaglutide and liraglutide, NAION was not increased versus placebo. Across >93,000 participants (GLP-1RA arms 61,729; placebo 31,776) and >203,000 participant-years, 3 confirmed NAION events (all in semaglutide trials; none with liraglutide) occurred in GLP-1RA arms versus 5 events in placebo arms, giving similar incidence rates (~8 vs ~7 per 100,000 participant-years). Reported cases typically had background vascular risk factors (e.g., age >50 years, obesity, smoking, dyslipidaemia, hypertension), and there was no consistent time-to-onset pattern.[23] In a large retrospective cohort study using a de-identified global EMR database (N: T2DM-only 37,314; obesity-only 129,690; T2DM+obesity 130,216), semaglutide exposure was not associated with an increased risk of NAION in matched populations with T2D only (HR 1.51 at 3 years; 95% CI 0.71–3.25), obesity only (HR 0.72 at 3 years; 95% CI 0.24–2.16), or T2D with obesity (HR 1.19 at 3 years; 95% CI 0.78–1.82), with similarly non-significant results at 1 and 2 years across groups; overall, these findings suggest that avoiding semaglutide solely due to NAION concern may not be warranted given its established cardiometabolic benefits. [24] Real-world evidence from TriNetX is largely reassuring. In a US propensity-matched analysis (≈107,662–130,000 per arm), semaglutide was not associated with increased NAION, with 5-year NAION RR 0.70 (≈30% lower relative risk) and very low absolute incidence (0.065% at 5 years).[25]

Similar real-world evidence is seen from the U.S. Military Health System. In a large retrospective cohort of 1,212,775 beneficiaries, semaglutide use in patients with type 2 diabetes was associated with a significantly lower risk of NAION compared with non-GLP-1 receptor agonist therapies (adjusted OR 0.36, 95% CI 0.25–0.51), while no significant association was observed among individuals treated for overweight or obesity. NAION events were rare overall, supporting the absence of an excess NAION risk with semaglutide.[26] In a systematic review and meta-analysis, the absolute risk of NAION with semaglutide was found to be low. This meta-analysis included 8 RCTs and 8 observational studies, covering more than 16,42,452 patients with either T2D or Obesity.While the risk was slightly higher in the observational studies, RCT data showed no difference vs non-users. Furthermore, it was not specified, in many clinical trials whether ischemic neuropathy was arteritic or non arteritic, which is a challenging differential diagnosis and reporting for many ophthalmologists, as the two diseases share the same ICD-9 coding. This might have led to over-reporting in many cases. Now, NAION (Non-Arteritic Anterior Ischemic Optic Neuropathy) uses ICD-10 codes, replacing older ICD-9 codes with much greater detail on laterality (left/right), specific causes (like diabetes, hypertension), and severity, reflecting modern medicine and improving data for research and outcomes, thanks to ICD-10.[27,28]

Professional and regulatory bodies have responded cautiously. The American Academy of Ophthalmology (AAO) and NANOS emphasise that current evidence is inconclusive, that the absolute risk of NAION with semaglutide appears very low, and that patients should not abruptly discontinue therapy without consulting their clinicians.[29,30] In June 2025, the World Health Organization reviewed semaglutide and NAION signals and concluded that available data do not establish a causal relationship, but recommended ongoing pharmacovigilance and clear communication of potential visual warning signs to patients [31] Further, WHO has included GLP-1 RAs including semaglutide in the Essential Medicines List, reflecting its overall cardiometabolic benefits outweighing the uncertain and rare potential risk of NAION.[32] Importantly, the innovatorsemaglutide has been evaluated in more than 65,000 patients across clinical trials and has accumulated over 49 million patient-years of post-marketing exposure. Taken together, the weight of evidence currently does not support a proven causal relationship between innovator semaglutide and NAION. Rather, the event has been classified as being very rare and likely to occur sporadically in predisposed individuals with underlying vascular or ophthalmic risk factors, consistent with the background rate in diabetes populations. At present, data favour continued use of semaglutide when clinically indicated, with heightened awareness and prompt evaluation of new visual symptoms rather than pre-emptive avoidance.

Clinical implications: How to use semaglutide safely in patients at ocular risk

In the context of diabesity, semaglutide offers significant and powerful HbA1c reductions (~1.8% mean in overall population, and ~2.8% mean in population with high baseline HbA1c), substantial weight loss and proven cardiovascular and renal benefits.[13–16,33]The DCGI/CDSCO-approved label for Ozempic in India recommends early use in adults with type 2 diabetes, as adjunct to diet and exercise. The approval for other brands of semaglutide 1.0 mg is slightly different – in people with metformin intolerance or hypersensitivity. The most frequently encountered adverse events would be those related to the gastrointestinal tract (transient, mild to moderate nausea, vomiting, constipation or diarrhoea). With respect to the eye-related complications, a practical, risk-balanced approach is required (depicted in Figure 1).

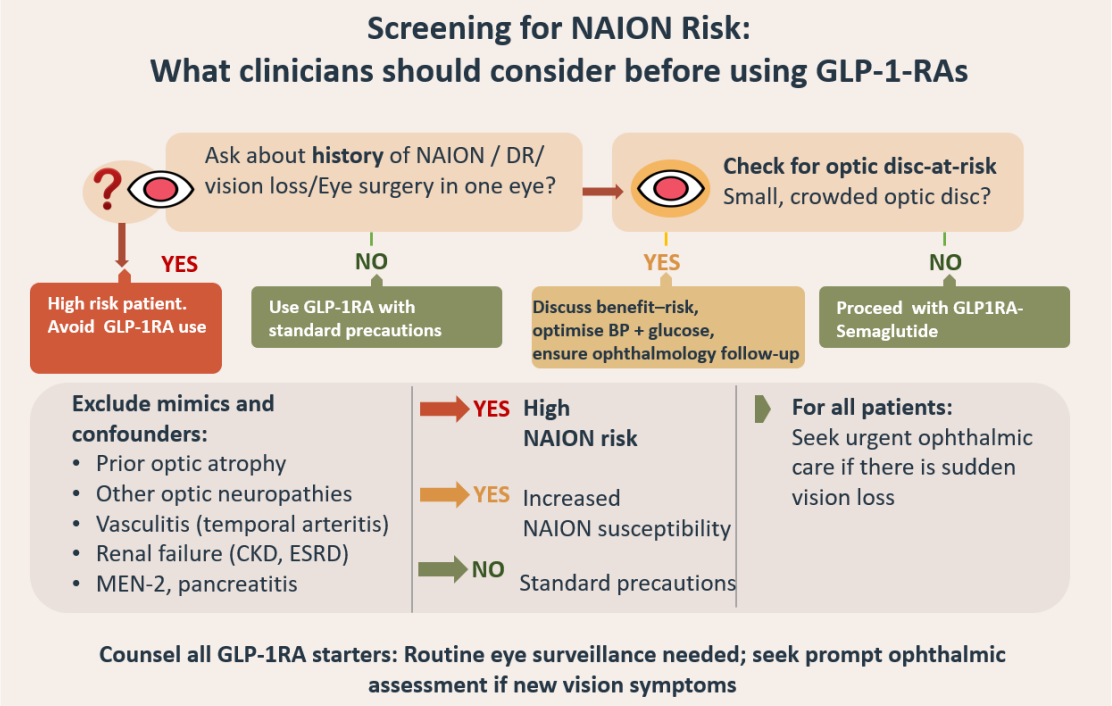


Figure 1: Screening for NAION risk before initiating GLP-1 receptor agonists

Summary and conclusion

Before initiation

- No additional precautions are necessary beyond the routine diabetic eye care advise. The recommendation is to have an annual examination for ocular complications (of diabetes) are recommended in people with diabetes or as advised by the treating ophthalmologist based on the retinopathy. Baseline retinal assessment and DR status in people with long-standing diabetes, high HbA1c or visual symptoms can be undertaken ideally with dilated fundus examination or retinal imaging.

During titration and follow-up

- Slow dose escalation (0.25 mg weekly for four weeks → 0.5 mg weekly for at least four weeks → 1.0 mg weekly as needed) to limit abrupt glycaemic shifts, paralleling lessons from DCCT/UKPDS early worsening.

- Educate patients about transient visual-symptom monitoring and the importance of regular eye review.The must also be educated to report any sudden painless vision loss or field defect immediately.

When to be cautious / consider alternatives?

- In patients with proliferative DR or recent high-risk ocular procedures where rapid glycaemic shifts may be undesirable
- In patients unable or unwilling to engage in eye monitoring despite high baseline DR risk.
- In patients with small crowded optic disk (disc at risk)
- In patients with sleep apnoea

In patients with a prior history of NAION or any form of optic nerve pathology, better to consider alternatives. Outside of these specific high-risk situations, there is no strong evidence-based reason to avoid semaglutide on ocular grounds. On the contrary, by improving glycaemic control and broader vascular health, semaglutide is likely to reduce long-term risk of DR progression and ocular vascular events in many patients with diabesity.In India, a cause of concern can be the lack of ophthalmology services capacity and the feasibility of the proposed monitoring strategy in routine practice.

CONCLUSION

Ocular safety concerns with semaglutide warrant a balanced interpretation of the available evidence. The retinopathy complications observed in SUSTAIN-6 were largely associated with rapid HbA1c reduction in patients with pre-existing diabetic retinopathy, consistent with the early-worsening phenomenon recognised in intensive glycaemic control trials. Although non-arteritic anterior ischaemic optic neuropathy (NAION) has been recognised as a very rare adverse effect, its absolute risk remains low, warranting clinical vigilance and continued surveillance. The ongoing FOCUS trial may provide further clarity regarding retinal safety. Given its established glycaemic, weight, cardiovascular and renal benefits, semaglutide retains a favourable benefit–risk profile in appropriately selected patients with type 2 diabetes and obesity, particularly when accompanied by appropriate ophthalmic monitoring and individualised risk assessment.

Declarations

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