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THE EFFECTS OF GLP-1 RECEPTOR AGONISTS ON OCULAR DISEASES: A NARRATIVE REVIEW

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ABSTRACT

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are incretin-based agents widely used in the management of type 2 diabetes mellitus and obesity. Beyond their metabolic effects, accumulating evidence indicates that these drugs may significantly influence ocular tissues, potentially exerting neuroprotective and anti-inflammatory actions, while also raising concerns regarding retinal and optic nerve complications.

Aim: The aim of this narrative review was to summarize the current evidence on the effects of GLP-1RAs on ocular diseases, with particular emphasis on diabetic retinopathy, glaucoma, age-related macular degeneration, cataract, non-arteritic anterior ischemic optic neuropathy, and other visual disturbances.

Methods: A structured literature review was conducted using PubMed and Google Scholar. Cohort studies, systematic and narrative reviews, case reports, pharmacovigilance analyses, and preclinical investigations published up to May 2026 were analyzed.

Results: Experimental data suggest anti-inflammatory, antioxidant, vascular, and neuroprotective effects of GLP-1RAs on ocular tissues. Clinical studies report possible benefits in glaucoma and age-related macular degeneration, whereas findings in diabetic retinopathy remain inconsistent. Reported adverse events include transient worsening of diabetic retinopathy, blurred vision, cataract, and possible semaglutide-associated non-arteritic anterior ischemic optic neuropathy. These outcomes may be influenced by rapid HbA1c reduction, weight loss, baseline ocular disease, and vascular risk factors.

Conclusions: GLP-1 receptor agonists may exert both protective and adverse ocular effects. Further prospective studies are required to determine their long-term ophthalmic safety and clinical significance.

KEYWORDS

GLP-1 Receptor Agonists; Semaglutide; Tirzepatide; Diabetic Retinopathy; Glaucoma; Non-Arteritic Anterior Ischemic Optic Neuropathy; Age-Related Macular Degeneration; Ocular Complications

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1. Introduction

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are incretin-based drugs widely used in the treatment of type 2 diabetes mellitus and obesity. Initially introduced for glycemic control, these agents have gained increasing attention because of their additional effects, including body weight reduction and cardiovascular and renal benefits [Bethel et al., 2021; Kapoor et al., 2023]. Tirzepatide, a dual glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 receptor agonist, further expanded the therapeutic role of incretin-based treatment due to its strong metabolic effects and significant impact on weight reduction [Jastreboff et al., 2022].

GLP-1 receptors are expressed in multiple tissues throughout the body, including the cardiovascular system, kidneys, gastrointestinal tract, central nervous system, and ocular tissues. Their presence has been demonstrated in the retina, retinal pigment epithelium, retinal ganglion cells, and the optic nerve [Gong & Orge, 2026; Puddu & Maggi, 2022]. This widespread receptor distribution suggests that GLP-1RAs may influence several biological pathways involved in ocular diseases.

Many ophthalmic disorders are associated with oxidative stress, inflammation, vascular dysfunction, and neurodegeneration. These mechanisms contribute to the pathogenesis of diabetic retinopathy, glaucoma, age-related macular degeneration (AMD), and optic nerve disorders [Eleftheriadou et al., 2024; Thomas et al., 2021]. Experimental studies suggest that GLP-1RAs may exert anti-inflammatory, antioxidant, and neuroprotective effects within retinal tissues [Puddu & Maggi, 2022; Bain et al., 2019]. In addition, improved glycemic control and reduction of cardiovascular risk factors may indirectly contribute to ocular protection.

At the same time, concerns regarding the ophthalmic safety of GLP-1RAs have emerged. Rapid improvement in glycemic control has been associated with transient worsening of diabetic retinopathy, similarly to observations made during intensive insulin therapy [Bethel et al., 2021; Eleftheriadou et al., 2024]. Furthermore, observational studies and case reports have raised concerns about possible associations between GLP-1RAs and visual disturbances, cataract, and non-arteritic anterior ischemic optic neuropathy (NAION) [Katz et al., 2025; Hsu et al., 2025]. However, current evidence remains inconsistent, and many reported complications may be influenced by preexisting metabolic and ocular disorders rather than direct drug toxicity [Chen et al., 2026].

The aim of this review was to summarize current evidence regarding the effects of GLP-1 receptor agonists on ocular diseases, with particular emphasis on diabetic retinopathy, glaucoma, age-related macular degeneration, cataract, optic nerve disorders, and other visual complications.

2. Methodology

A structured literature review was conducted using electronic databases, including PubMed and Google Scholar. Publications relevant to the effects of glucagon-like peptide-1 receptor agonists (GLP-1RAs) on ocular diseases were identified. The literature search included studies published up to May 2026.

The following keywords were used: “GLP-1 receptor agonist,” “retinopathy,” “glaucoma,” “age-related macular degeneration,” “non-arteritic anterior ischemic optic neuropathy,” “visual disturbances,” and “ocular disease.”

Various types of publications were included, such as case reports, retrospective cohort studies, systematic and narrative reviews, and preclinical studies conducted on in vitro and ex vivo models. Studies were selected based on their relevance to ocular outcomes associated with GLP-1 receptor agonists, including both potential beneficial and adverse ophthalmic effects.

3. Results

3.1. Mechanisms of action of GLP-1 receptor agonists in ocular tissues

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) may influence ocular tissues through both direct and indirect mechanisms. GLP-1 receptors have been identified in several structures of the eye, including retinal ganglion cells, retinal pigment epithelium, Müller cells, and the optic nerve [Gong & Orge, 2026; Puddu & Maggi, 2022; Xu et al., 2025]. The presence of these receptors suggests that GLP-1RAs may affect pathways involved in retinal injury, neurodegeneration, inflammation, and vascular dysfunction [Gong & Orge, 2026; Puddu & Maggi, 2022]. One of the most commonly described effects of GLP-1RAs is the reduction of oxidative stress. Chronic hyperglycemia and mitochondrial dysfunction contribute to excessive production of reactive oxygen species (ROS), leading to retinal cellular injury and endothelial dysfunction. Several experimental studies have demonstrated that GLP-1RAs may reduce oxidative stress by activating intracellular pathways involved in cell survival and antioxidant activity, including AMPK and PI3K/Akt signaling [Puddu & Maggi, 2022]. Reduction of oxidative stress has been associated with decreased apoptosis of retinal cells and stabilization of the blood–retinal barrier [Anastasiou et al., 2025].

Another important mechanism involves anti-inflammatory activity. Chronic inflammation plays a major role in the pathogenesis of diabetic retinopathy, glaucoma, and age-related macular degeneration. Studies have shown that GLP-1RAs may decrease the expression of pro-inflammatory cytokines, including tumor necrosis factor alpha (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6) [Bain et al., 2019; Shao et al., 2026]. Inhibition of inflammatory pathways, including NF-κB and the NLRP3 inflammasome, has also been observed in experimental retinal models [Shao et al., 2026]. Some authors have additionally reported reduced retinal neuroinflammation and lower microglial activation following GLP-1RA administration [Puddu & Maggi, 2022; Anastasiou et al., 2025; Shao et al., 2026].

Potential vascular effects of GLP-1 receptor agonists have also been described. Endothelial dysfunction and impaired retinal perfusion contribute to retinal ischemia and progression of ocular diseases. Experimental studies suggest that GLP-1RAs may improve endothelial function, decrease vascular permeability, and stabilize retinal microcirculation [Gong & Orge, 2026]. Some studies additionally demonstrated reduced expression of vascular endothelial growth factor (VEGF) and attenuation of pathological angiogenesis [Shao et al., 2026].

Increasing attention has also been directed toward the neuroprotective properties of GLP-1RAs. Retinal ganglion cell loss represents one of the major pathological mechanisms in glaucoma and optic nerve disorders. Experimental data indicate that activation of GLP-1 receptors may reduce neuronal apoptosis, improve

mitochondrial function, and attenuate excitotoxicity within retinal tissues [Puddu & Maggi, 2022; Silverii et al., 2025]. Some reports have also indicated that semaglutide and other GLP-1 receptor agonists may promote retinal cellular regeneration and improve neuronal survival in experimental injury models [Anastasiou et al., 2025].

Apart from direct ocular mechanisms, GLP-1RAs may indirectly influence ocular health through systemic metabolic improvement. Better glycemic control, reduction in insulin resistance, body weight reduction, and favorable cardiovascular effects may contribute to decreased microvascular injury and improved retinal perfusion [Bethel et al., 2021; Sadeghi et al., 2025]. However, rapid improvement in glycemic control may also transiently worsen diabetic retinopathy, similarly to the phenomenon observed during intensive insulin therapy [Kapoor et al., 2023; Eleftheriadou et al., 2024].

Although many studies suggest potentially protective effects of GLP-1RAs within ocular tissues, the currently available evidence remains limited. Most data originate from experimental and observational studies, while clinical evidence is still relatively scarce. Furthermore, the majority of available studies concern semaglutide and liraglutide, whereas substantially fewer data are available for newer agents such as tirzepatide. Therefore, further studies are necessary to better understand the mechanisms underlying the ocular effects of GLP-1RAs and their possible clinical implications.

3.2. Diabetic retinopathy

Diabetic retinopathy (DR) is one of the most common microvascular complications of diabetes mellitus and remains a major cause of vision loss worldwide. Its pathogenesis is complex and involves chronic hyperglycemia, oxidative stress, endothelial dysfunction, retinal ischemia, inflammation, blood–retinal barrier breakdown, and pathological angiogenesis [Puddu & Maggi, 2022; Shao et al., 2026; Zheng et al., 2023].

Because GLP-1 receptor agonists exert anti-inflammatory, antioxidant, and neuroprotective effects, their possible role in diabetic retinopathy has been widely investigated. Experimental studies have demonstrated that activation of retinal GLP-1 receptors may reduce oxidative stress, decrease inflammatory cytokine expression, inhibit apoptosis, and stabilize the blood–retinal barrier [Puddu & Maggi, 2022; Shao et al., 2026]. Some studies have additionally shown reduced VEGF expression and attenuation of retinal neuroinflammation following GLP-1RA treatment [Machida et al., 2025; Xie et al., 2025].

Zheng et al. (2023) demonstrated that higher GLP1R expression was associated with a lower risk of background diabetic retinopathy (odds ratio [OR] = 0.83; 95% confidence interval [CI]: 0.71–0.97) and severe non-proliferative diabetic retinopathy (OR = 0.72; 95% CI: 0.53–0.98). However, no significant correlation was found between GLP-1 and the risk of proliferative diabetic retinopathy (OR = 0.98; 95% CI: 0.91–1.05) or overall diabetic retinopathy (OR = 0.97; 95% CI: 0.92–1.03).

Despite these potentially protective mechanisms, clinical studies have produced inconsistent findings. One of the main concerns is transient worsening of diabetic retinopathy after rapid improvement in glycemic control, similarly to the phenomenon previously observed during intensive insulin therapy [Bethel et al., 2021; Kapoor et al., 2023].

Meta-analyses of cardiovascular outcome trials have generally not demonstrated an overall increase in retinopathy risk among patients treated with GLP-1 receptor agonists. However, greater HbA1c reduction was associated with higher risk of retinopathy progression, suggesting that rapid glycemic improvement may play a more important role than direct retinal toxicity [Bethel et al., 2021].

Lu (2026) proposed the so-called “angiogenesis–inflammation hypothesis,” according to which worsening of diabetic retinopathy following semaglutide therapy results not only from rapid glycemic improvement but also from transient enhancement of angiogenic and inflammatory processes within the retina. This may occur through increased expression of VEGF, angiopoietin-2 (Ang-2), and pro-inflammatory cytokines such as TNF-α and IL-6.

Current evidence suggests that the use of GLP-1 receptor agonists may accelerate the progression of diabetic retinopathy and diabetic macular edema [Kurek, 2023]. Particular attention has been focused on semaglutide, which has been associated with an increased incidence of retinopathy complications (HR ≈ 1.76 vs. placebo), including vitreous hemorrhage, worsening of vision, and the need for photocoagulation therapy [Marso et al., 2016].

Several observational studies have suggested possible beneficial retinal effects of GLP-1 receptor agonists. In patients with preexisting diabetic retinopathy, GLP-1RA therapy was associated with a lower risk of blindness (HR = 0.77; 95% CI: 0.73–0.82) [Eleftheriadou et al., 2024]. Experimental studies have also

demonstrated improved retinal perfusion, reduced oxidative stress, and decreased retinal ganglion cell apoptosis after GLP-1RA administration [Xu et al., 2025; Anastasiou et al., 2025].

On the other hand, pharmacovigilance analyses and case reports have described worsening diabetic retinopathy and other retinal complications during GLP-1RA therapy [Murray et al., 2026; Cool et al., 2024]. Many authors have emphasized that progression of DR appears to be mainly associated with rapid HbA1c reduction, poor baseline metabolic control, long duration of diabetes, and preexisting retinal disease [Bethel et al., 2021; Kapoor et al., 2023].

Overall, currently available evidence suggests that GLP-1 receptor agonists may exert both beneficial and potentially adverse effects in diabetic retinopathy. Experimental studies generally support their anti-inflammatory and neuroprotective properties, whereas clinical findings remain inconsistent. Further prospective studies are required to better determine the relationship between GLP-1RA therapy and diabetic retinopathy progression.

3.3. Glaucoma

The pathophysiology of glaucoma is primarily characterized by progressive neurodegeneration of retinal ganglion cells (RGCs), frequently accompanied by elevated intraocular pressure (IOP). Both mechanisms may potentially be modulated by glucagon-like peptide-1 (GLP-1) receptor agonists [Ekici & Moghimi, 2023].

Activation of GLP-1 receptors expressed in retinal tissues has been associated with several neuroprotective effects, including attenuation of RGC apoptosis, reduction of oxidative stress, and suppression of inflammatory pathways [Amaral et al., 2025]. Beyond direct retinal neuroprotection, GLP-1 receptor agonists may improve retinal microcirculation by enhancing endothelial function and tissue perfusion, which may be particularly relevant in the pathogenesis of normal-tension glaucoma (NTG). Furthermore, the systemic metabolic effects of these agents—including improved glycemic control, weight reduction, and cardiovascular protection—may indirectly contribute to retinal and optic nerve preservation [Xie et al., 2025; Luo et al., 2026].

Accumulating observational evidence indicates a reduced incidence of glaucoma among patients treated with GLP-1 receptor agonists, although most currently available data concern semaglutide and liraglutide, potentially limiting extrapolation to the entire drug class [Amaral et al., 2025].

Amaral et al. (2025), in a recent meta-analysis, demonstrated a significantly lower incidence of glaucoma among GLP-1 receptor agonist users compared with controls (HR = 0.71; 95% CI: 0.60–0.85; $I^2 = 29\%$), with moderate heterogeneity and a moderate risk of bias related mainly to confounding factors and intervention classification. Likewise, another observational study reported new glaucoma diagnoses in only 0.51% of patients treated with GLP-1 receptor agonists compared with 1.33% in the control group (HR = 0.56; $p = 0.01$), further supporting a potential neuroprotective role of GLP-1 signaling in ocular tissues [Sterling et al., 2023].

Further evidence was provided by Muayad et al. (2025), who demonstrated that patients receiving GLP-1 receptor agonists had a significantly lower relative risk of developing primary open-angle glaucoma (POAG) compared with metformin users after 1 year (RR = 0.59; 95% CI: 0.39–0.88), 2 years (RR = 0.50; 95% CI: 0.32–0.78), and 3 years of follow-up (RR = 0.51; 95% CI: 0.34–0.75). In addition, the requirement for first-line glaucoma interventions, including topical hypotensive therapy or laser trabeculoplasty, was reduced in the GLP-1RA group.

Long-term cohort data published by Allan et al. (2025) also indicated a protective association between GLP-1 receptor agonist therapy and glaucoma risk. In the subgroup receiving GLP-1RAs, the hazard ratio for POAG was 0.72 (95% CI: 0.61–0.85) over a 3-year follow-up and 0.79 (95% CI: 0.69–0.91) over 5 years.

Preclinical investigations further reinforce these clinical observations. In an experimental mouse model of glaucoma, Lawrence et al. (2023) induced ocular hypertension and subsequently treated animals with systemic or topical GLP-1 receptor agonists. Systemic liraglutide administration resulted in near-complete preservation of retinal ganglion cells, whereas topical administration increased RGC survival by 24% with liraglutide ($p = 0.0048$) and by 29% with lixisenatide ($p = 0.0096$). Treatment was additionally associated with a marked reduction in pro-inflammatory cytokine expression (IL-1α, TNF-α, C1q; $p < 0.0001$). Importantly, topical liraglutide also produced a modest but statistically significant reduction in intraocular pressure compared with untreated glaucomatous eyes ($p < 0.05$), suggesting both neuroprotective and pressure-modulating properties.

Sterling et al. (2021) expanded these findings in a cohort study evaluating the association between GLP-1 receptor agonist use and glaucoma development. The authors demonstrated that GLP-1 receptor agonist

therapy was associated with a significantly reduced incidence of glaucoma (HR = 0.54; 95% CI: 0.35–0.85; p = 0.007), corresponding to an approximate 46% relative risk reduction. This observation further supports the hypothesis that GLP-1 receptor agonists may exert clinically relevant neuroprotective effects within the retina and optic nerve.

Taken together, the currently available evidence suggests that GLP-1 receptor agonists may represent a promising therapeutic strategy for glaucoma prevention and potentially for slowing disease progression. However, most available data originate from observational or preclinical studies. Large prospective randomized clinical trials are still required to establish causality and determine the clinical applicability of these findings.

3.4. Age-related macular degeneration (AMD)

Age-related macular degeneration (AMD) is a progressive disorder affecting the macula and represents one of the leading causes of irreversible blindness among elderly individuals. AMD occurs in two major forms: dry AMD, characterized by degeneration of the retinal pigment epithelium and photoreceptors, and wet AMD, associated with abnormal choroidal neovascularization. The pathogenesis of AMD includes inflammatory processes, metabolic disorders, oxidative stress, mitochondrial dysfunction, extracellular matrix remodeling, alterations in retinal pigment epithelium, lipid dysregulation, and angiogenesis [Gong & Orge, 2026; Thomas et al., 2021; Gheorghe et al., 2015; Jiang et al., 2025].

Due to the influence of GLP-1 receptor agonists on these pathways, their potential disease-modifying role in AMD has been considered. Experimental studies have demonstrated anti-inflammatory and antiangiogenic effects of GLP-1 receptor agonists, mediated through inhibition of the NLRP3 inflammasome and suppression of IL-1β, IL-6, and TNF expression [Machida et al., 2025].

Several studies have investigated the impact of glucagon-like peptide-1 receptor agonists on AMD. An in vitro study evaluated the effects of liraglutide on retinal pigment epithelium cells damaged by free fatty acids. Liraglutide was shown to reduce oxidative stress and lipid droplet accumulation, with its mechanism associated with the activation of AMP-activated protein kinase (AMPK). Furthermore, the drug influenced exosome secretion and intercellular signaling [Tsou et al., 2025].

In a retrospective cohort study, Allan et al. (2025) reported that the use of GLP-1 analogues was associated with a statistically significant reduction in the risk of developing AMD. For dry AMD, a lower risk was observed compared with metformin (HR = 0.68; 95% CI: 0.56–0.84), insulin (HR = 0.72; 95% CI: 0.58–0.89), and statins (HR = 0.70; 95% CI: 0.57–0.87). This effect emerged after approximately 3 years of therapy and persisted in the 5-year analysis compared with metformin (HR = 0.69; 95% CI: 0.52–0.91), insulin (HR = 0.66; 95% CI: 0.50–0.87), and statins (HR = 0.67; 95% CI: 0.51–0.88). A reduced risk of exudative AMD was also demonstrated (HR = 0.70; 95% CI: 0.58–0.84).

Another cohort study showed that GLP-1 receptor agonists were associated with a reduced risk of developing AMD in individuals without diabetes compared with other medications used for obesity treatment. A statistically significant reduction was observed for dry AMD (HR = 0.47; 95% CI: 0.28–0.78) and for any form of AMD (HR = 0.61; 95% CI: 0.43–0.85), whereas no significant difference was found for exudative AMD (HR = 0.63; 95% CI: 0.30–1.32) [Myers et al., 2026].

In a cohort study by Ahuja et al. (2025), involving overweight or obese individuals without diabetes, the use of GLP-1 analogues was associated with a marked reduction in the risk of developing non-exudative AMD compared with other weight-loss medications. The reduced risk was observed after 5 years (RR = 0.16; 95% CI: 0.10–0.28), 7 years (RR = 0.13; 95% CI: 0.08–0.22), and 10 years (RR = 0.09; 95% CI: 0.05–0.16). However, no significant effect on progression to exudative AMD was identified.

On the other hand, in a cohort study involving 139,002 patients with diabetes—of whom 46,334 were treated with GLP-1 receptor agonists and 92,668 served as a control group—the risk of developing neovascular AMD (nAMD) was assessed over a 3-year follow-up period. The incidence of nAMD was higher in the GLP-1RA group compared with the control group (0.2% vs. 0.1%), corresponding to a more than twofold increased risk (adjusted HR = 2.21; 95% CI: 1.65–2.96) [Shor et al., 2025].

In a case reported by Takebayashi et al. (2026), a 58-year-old man treated for type 2 diabetes since the age of 33 years developed rapidly progressive nAMD following initiation of tirzepatide therapy. Prior to treatment, optical coherence tomography (OCT) showed no abnormalities in the macula. The first symptoms, including blurred and distorted central vision, appeared approximately one month after therapy initiation, while significant structural changes on imaging became evident after seven months, leading to a diagnosis of nAMD. Treatment was initiated, and tirzepatide was discontinued due to a possible association with disease onset. The

authors emphasized that a definitive causal relationship could not be established, although rapid improvement in glycemic control induced by incretin-based therapies may potentially contribute to the unmasking or progression of nAMD in susceptible individuals.

3.5. Non-arteritic anterior ischemic optic neuropathy (NAION)

Non-arteritic anterior ischemic optic neuropathy (NAION) is the most common cause of sudden vision loss related to the optic nerve. Its etiopathogenesis is complex and involves multiple risk factors, although the exact mechanisms have not yet been fully understood. Identified risk factors include older age, small optic nerve passage through the sclera, diabetes, hypertension, hypotension, hypercholesterolemia, obstructive sleep apnea, certain medications, vascular dysregulation, and genetic predisposition [Bernstein et al., 2011; Saleem et al., 2025].

In the context of glucagon-like peptide-1 receptor agonists (GLP-1RAs), several potential mechanisms that may link therapy with the development of NAION have been considered. These include rapid metabolic and hemodynamic changes associated with rapid improvement in glycemic control and significant weight loss, alterations in ocular microcirculation, and modulation of endothelial function. Additionally, the anti-inflammatory and neuroregulatory effects of GLP-1, along with the presence of GLP-1 receptors in the optic nerve and enhancement of sympathetic nervous system activity, may affect optic nerve perfusion in susceptible individuals. Consequently, increasing clinical and observational evidence has emerged in recent years investigating a possible association between GLP-1RA therapy and ischemic optic nerve events, including NAION [Cai et al., 2025; Hathaway et al., 2024; Hidalgo Ramos et al., 2025].

In a large multicenter study involving adults with type 2 diabetes receiving semaglutide, other GLP-1 receptor agonists, or non-GLP-1RA medications, semaglutide use was associated with a small increase in the incidence of NAION (14.5 per 100,000 person-years). The risk was not significantly different compared with most non-GLP-1RA therapies, although a higher risk was observed in certain analyses, and self-controlled case series indicated a modest but statistically significant increase (IRR = 1.32; 95% CI: 1.14–1.54) [Cai et al., 2025].

Another study of type 2 diabetes and overweight/obese patients showed that the use of semaglutide was associated with a higher incidence of NAION compared with non-GLP-1RA antidiabetic or weight-loss medications. In the type 2 diabetes cohort, 17 cases of NAION were reported among semaglutide users compared with 6 cases in the control group, with a cumulative 36-month incidence of 8.9% versus 1.8%, respectively (HR = 4.28; 95% CI: 1.62–11.29). In the overweight/obesity cohort, 20 versus 3 NAION cases were observed, corresponding to cumulative incidences of 6.7% and 0.8%, respectively (HR = 7.64; 95% CI: 2.21–26.36) [Hathaway et al., 2024].

A population-based study using national health registries from Denmark and Norway showed that the incidence of NAION was higher in semaglutide users compared with SGLT-2 inhibitor users (Denmark: 2.19 vs. 1.18 per 10,000 person-years; Norway: 2.90 vs. 0.92), with an increased adjusted risk (HR = 2.81; 95% CI: 1.67–4.75). However, the absolute risk remained low. In a supplementary self-controlled analysis, the symmetry ratios were 1.14 (95% CI: 0.55–2.36) in Denmark and 2.67 (95% CI: 0.91–8.99) in Norway [Simonsen et al., 2025].

On the other hand, Hsu et al. (2025) described no significant increase in NAION risk in the early follow-up period from 1 month to 1 year. However, at longer follow-up, semaglutide use was associated with an increased risk of NAION at 2 years (HR = 2.39; 95% CI: 1.37–4.18), 3 years (HR = 2.44; 95% CI: 1.44–4.12), and 4 years (HR = 2.05; 95% CI: 1.26–3.34). A higher risk was also observed in patients with concomitant hypertension and in those with a history of Ozempic (Novo Nordisk) exposure.

In another cohort study including patients with type 2 diabetes and no prior ocular disease, users of semaglutide or tirzepatide were compared with patients receiving other antidiabetic therapies. Over a 2-year follow-up, NAION occurred in 0.04% of the GLP-1 receptor agonist group versus 0.02% in the comparison group, corresponding to a modestly increased risk (HR = 1.76; 95% CI: 1.01–3.07). A higher incidence was also observed for other optic nerve disorders in the semaglutide or tirzepatide group (0.12% vs. 0.07%; HR = 1.65; 95% CI: 1.18–2.31) [Wang et al., 2025].

In the Food and Drug Administration Adverse Event Reporting System (FAERS), GLP-1 analogues were associated with an increased frequency of reported ischemic optic neuropathy cases. Among patients with type 2 diabetes, semaglutide was associated with a higher reporting rate of optic ischemic neuropathy compared with metformin (ROR = 12.269; 95% CI: 0.915–1.967), while an increased reporting rate was also observed for tirzepatide (ROR = 4.619; 95% CI: 0.726–2.335) [Lu, 2026].

Although cohort studies provide important evidence regarding the association between GLP-1 receptor agonists and NAION, individual case reports have described rare optic nerve complications occurring in temporal association with treatment. Maher et al. (2025) reported the case of a 43-year-old woman with type 1 diabetes, obesity, and depression who developed inflammatory optic neuritis and scleritis one month after initiation of semaglutide therapy, which corresponds to the time required to reach steady-state drug levels. Systemic corticosteroid therapy and discontinuation of semaglutide resulted in rapid clinical improvement, with resolution of symptoms and recovery of baseline visual acuity within three months. Although a causal relationship between semaglutide use and the ocular event could not be definitively established, extensive diagnostic evaluation excluded alternative systemic etiologies. The favorable response to glucocorticoid therapy supported an inflammatory mechanism. The authors emphasized the importance of clinical vigilance for rare but potentially vision-threatening ocular complications in patients receiving GLP-1 receptor agonists.

Another case described a 34-year-old patient with long-standing diabetes who developed NAION after six months of semaglutide therapy. Following discontinuation of semaglutide and optimization of metabolic control, complete resolution of visual function and structural abnormalities was observed over a two-month follow-up. Although causality remains uncertain, the case suggests a potential association between GLP-1RA therapy and ischemic optic neuropathy, particularly in patients with underlying vascular risk factors [Bakheet & Chaudhary, 2026].

In a case reported by Lesin Gacina et al. (2025), a 55-year-old woman with type 2 diabetes, hypertension, and hyperlipidemia developed optic disc edema four months after initiation of semaglutide therapy. She was initially diagnosed with diabetic papillopathy, which progressed to non-arteritic anterior ischemic optic neuropathy, confirmed by visual field defects and fluorescein angiography. After discontinuation of semaglutide, corticosteroid treatment, and optimization of glycemic control, visual acuity improved, although optic nerve atrophy was observed during follow-up. While causality remains uncertain, the temporal association suggests a potential link between semaglutide use and ischemic optic neuropathy, particularly in patients with vascular risk factors.

A retrospective case series including 9 patients (mean age 57.4 ± 11.6 years) reported ophthalmic complications associated with semaglutide or tirzepatide use, with NAION observed in 7 cases, alongside isolated cases of papillitis and paracentral acute middle maculopathy. Among the 7 patients with NAION, 3 presented with typical features, including unilateral vision loss upon awakening, optic disc edema, and characteristic visual field defects, with supportive MRI findings in early imaging. The remaining cases demonstrated atypical presentations, such as bilateral optic disc swelling at onset, symptom recurrence shortly after semaglutide administration, and progressive vision loss over time. Although an association could not be established, the authors suggested that rapid improvement in glycemic control, rather than a direct drug effect, may contribute to these events [Katz et al., 2025].

On the other hand, a study involving patients with type 2 diabetes (approximately 120,000 treated with semaglutide) and individuals with overweight or obesity (approximately 58,000 treated with semaglutide) did not show a significant increase in the risk of NAION compared with patients receiving non-GLP-1RA therapies. In the type 2 diabetes cohort, the risk of NAION among semaglutide users was RR = 0.70 (95% CI: 0.523–0.937), with no increase observed over 5 years. Similarly, no increased risk was identified among patients receiving any GLP-1 receptor agonist (RR = 0.887; 95% CI: 0.735–1.071). The 5-year cumulative risk of NAION in patients with T2DM using semaglutide was 0.065%. In the overweight/obese cohort, no elevated risk was observed, and the cumulative 2-year risk of NAION was 0.038% [Abbass et al., 2025].

3.6. Cataract

Cataract is one of the leading causes of visual impairment worldwide. It is characterized by opacification of the normally transparent crystalline lens, resulting in progressive deterioration of visual acuity and, in advanced cases, blindness. The etiology of cataract is multifactorial and includes age-related (senile), congenital and developmental, iatrogenic, traumatic, and secondary forms associated with other ocular or systemic diseases, including inflammatory and metabolic conditions such as diabetes mellitus. Senile cataract develops due to cumulative metabolic and structural disturbances in the lens, including oxidative stress, glutathione depletion, calcium imbalance, and abnormalities in lens epithelial cell function. In contrast, diabetic cataract results from chronic hyperglycemia, leading to oxidative stress, non-enzymatic protein glycation, and osmotic imbalance, as well as metabolic disturbances that cause lens epithelial cell damage and apoptosis [Mishra et al., 2023; Gupta et al., 2014].

In a retrospective cohort study of overweight or obese patients without diabetes, GLP-1 receptor agonist use was associated with a significantly reduced risk of age-related cataract compared with other weight-loss drugs at 5 years (RR = 0.278; 95% CI: 0.246–0.314), 7 years (RR = 0.269; 95% CI: 0.241–0.299), and 10 years (RR = 0.198; 95% CI: 0.176–0.222). A similar effect was observed when compared with no pharmacological treatment, with consistent risk reduction across all time points. In contrast, the use of other weight-loss drugs was associated with an increased risk of cataract compared with untreated individuals [Jiang et al., 2025].

A retrospective analysis of Food and Drug Administration Adverse Event Reporting System (FAERS) reports assessed adverse events associated with the use of GLP-1 analogues. Among patients with type 2 diabetes, semaglutide was associated with an increased reporting rate of cataract compared with metformin (ROR = 31.879; 95% CI: 2.463–4.461), while liraglutide had an even higher reporting rate (ROR = 53.866; 95% CI: 2.945–5.028). Patients without diabetes also had an increased reporting rate of cataract with liraglutide (ROR = 9.628; 95% CI: 1.387–3.142) [Lu, 2026].

In a study by Virk and Allison (2026) evaluating ophthalmic adverse effects associated with GLP-1 receptor agonist therapy, cases of cataract were reported; however, the authors suggested that these events may have represented pre-existing conditions rather than treatment-related adverse effects. It was emphasized that patients with diabetes and multiple comorbidities already have an increased baseline risk of cataract development due to various pathophysiological mechanisms.

Another study involving 9,669 patients, with 84.4% of them having a diagnosis of diabetes, did not show that the use of GLP-1 analogues was associated with an increased risk of developing cataract [Luo et al., 2026].

Further studies are needed to clarify the relationship between GLP-1 receptor agonists and cataract risk. Given the multifactorial etiology of cataract and the interplay of multiple overlapping risk factors, future research should focus on long-term prospective studies accounting for metabolic influences and potential confounders.

3.7. Other visual disturbances

GLP-1 analogues, widely used in the management of diabetes and obesity, are increasingly investigated for their potential effects on the visual system, although current evidence remains inconclusive [Zhang & Finn, 2025]. The pathophysiology of visual disturbances encompasses both refractive alterations driven by changes in lens hydration and curvature, as well as structural damage to the retina and optic nerve associated with microangiopathy and impaired perfusion [Albanese et al., 2025]. Transient visual disturbances are primarily mediated by rapid changes in glycemic levels, which affect tissue osmolarity and result in reversible alterations in accommodation [Cool et al., 2024].

In contrast, more complex ocular pathologies are linked to chronic oxidative stress, inflammation, and endothelial dysfunction. GLP-1 receptor agonists may modulate these mechanisms both indirectly, through improved metabolic control, and directly, via effects on inflammatory pathways, vascular dynamics, and potential neuroprotective properties. The multifactorial nature of these interactions necessitates cautious interpretation of visual symptoms, with consideration of both direct pharmacological effects and secondary consequences of systemic metabolic improvement [Cool et al., 2024; Albanese et al., 2025].

One of the most frequently proposed explanations for transient blurred vision observed after initiation of GLP-1 receptor agonists is rapid glycemic correction. Sudden reductions in blood glucose may induce osmotic shifts within the crystalline lens, leading to temporary refractive instability and accommodation disturbances. These changes are generally reversible and tend to resolve following stabilization of glycemic levels. Similar phenomena have previously been described during intensive insulin therapy and are considered a consequence of abrupt metabolic normalization rather than direct retinal toxicity [Albanese et al., 2025; Choe & Hong, 2025].

At the same time, concerns regarding potential adverse ophthalmic effects of GLP-1 receptor agonists have emerged from pharmacovigilance analyses. Virk and Allison (2026), using data from the FDA Adverse Event Reporting System (FAERS) collected between 2005 and 2024, identified visual impairment, blurred vision, cataract, and blindness as the most commonly reported ocular adverse events associated with GLP-1 receptor agonists. The highest proportion of ocular adverse event reports was observed for exenatide (33.61%), whereas semaglutide and tirzepatide demonstrated progressively increasing annual reporting rates (2.23% and 0.79%, respectively; $p < 0.05$). Although spontaneous reporting databases cannot establish causality, these findings underscore the need for continued ophthalmologic surveillance in patients receiving GLP-1-based therapies.

Beyond transient visual disturbances, increasing attention has focused on the relationship between GLP-1 receptor agonists and diabetic retinopathy progression. In particular, rapid improvement in glycemic control has been hypothesized to transiently worsen retinal microvascular pathology, especially in patients with advanced pre-existing diabetic retinopathy. A retrospective cohort study involving adult patients with type 2 diabetes between 2015 and 2022 assessed the risk of diabetic retinopathy, ischemic optic neuropathy, and ocular complications among GLP-1 receptor agonist users compared with non-users. In a subgroup of 32,695 patients with pre-existing diabetic retinopathy, GLP-1 receptor agonist therapy was associated with a reduced risk of blindness (HR = 0.77; 95% CI: 0.73–0.82). These observations suggest a complex interaction between rapid metabolic improvement and pre-existing retinal vulnerability [Ramsey et al., 2025].

Despite these concerns, accumulating experimental evidence indicates that GLP-1 receptor agonists may also exert protective effects within the retina and optic nerve. Activation of retinal GLP-1 receptors has been associated with reduced oxidative stress, suppression of inflammatory cytokines, improved endothelial function, and attenuation of neuronal apoptosis. Experimental studies additionally suggest potential neuroprotective effects on retinal ganglion cells and retinal microvasculature, raising the possibility that GLP-1 signaling may contribute to long-term preservation of visual function [Gong & Orge, 2026; Albanese et al., 2025].

Overall, currently available evidence suggests a complex and potentially bidirectional relationship between GLP-1 receptor agonists and visual disturbances. While transient refractive changes and worsening of pre-existing retinal disease have been reported, emerging data simultaneously indicate anti-inflammatory, vasculoprotective, and neuroprotective properties that may confer long-term ophthalmic benefits. Further prospective studies are required to determine whether observed ocular events result primarily from direct pharmacological effects, rapid metabolic correction, or progression of underlying diabetic microvascular disease.

4. Discussion

This review summarizes the current state of evidence on the ophthalmic effects of GLP-1 receptor agonists, drawing on preclinical research, observational cohort studies, pharmacovigilance analyses, meta-analyses, and individual case reports. Collectively, the available data depict a complex and bidirectional relationship between GLP-1RA therapy and ocular health. On one hand, mechanistic studies and several large observational analyses support potentially protective effects on the retina and optic nerve, particularly in the context of glaucoma and age-related macular degeneration. On the other hand, reports of transient diabetic retinopathy worsening, blurred vision, cataract, and possible semaglutide-associated NAION raise legitimate safety concerns that warrant continued surveillance.

Methodologically, the available literature is heterogeneous. The included publications differ substantially in study design, patient populations, follow-up duration, settings, and outcome assessment. Many cohort analyses rely on administrative or pharmacovigilance databases, which are susceptible to confounding by indication, surveillance bias, and incomplete capture of pre-existing ocular comorbidities. Patients with diabetes also undergo more frequent ophthalmic examinations, which may inflate detection of ocular pathology in exposed cohorts and bias risk estimates upward. Most current evidence concerns semaglutide and liraglutide, with substantially less data on tirzepatide and other newer agents, which limits class-wide extrapolation.

From a clinical standpoint, the apparent contradictions between protective and adverse findings may be reconciled by considering the dual influence of GLP-1RAs on ocular tissues: direct receptor-mediated effects (anti-inflammatory, antioxidant, neuroprotective, and vascular) on the one hand, and indirect metabolic effects driven by rapid HbA1c reduction and substantial weight loss on the other. Rapid glycemic correction, in particular, has long been recognized as a trigger for transient worsening of pre-existing diabetic retinopathy, and similar mechanisms may underlie some of the reported ischemic optic nerve events. Baseline ocular vulnerability, the rate of metabolic improvement, dose, treatment duration, and concomitant vascular risk factors appear to be important modifiers of ophthalmic outcomes.

This review is also subject to several limitations. The included publications were selected by the authors based on their relevance to the topic, which may have influenced the composition of the analyzed evidence. The cited studies differ substantially in their methodology, including variations in study design, patient populations, follow-up duration, study settings, and outcome assessment methods. The study populations were heterogeneous with respect to age, sex, comorbidities, and concomitant treatments, making direct comparison between studies challenging. Furthermore, some studies lacked appropriate control groups and were subject to potential confounding factors, which further limit the comparability of findings.

5. Conclusions

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are widely used in the treatment of type 2 diabetes mellitus and obesity, and recent studies suggest that their effects may extend beyond metabolic control to influence ocular tissues. Experimental and clinical data indicate that GLP-1RAs may reduce oxidative stress, inflammation, endothelial dysfunction, and neurodegeneration, mechanisms central to diabetic retinopathy, glaucoma, and age-related macular degeneration. At the same time, the available evidence remains inconsistent: some studies report potential protective effects on the retina and optic nerve, while others describe worsening of diabetic retinopathy, visual disturbances, and possible associations with non-arteritic anterior ischemic optic neuropathy.

In many cases, these complications may be attributable not only to the drugs themselves but also to rapid improvement in glycemic control, weight loss, preexisting ocular disease, and vascular risk factors. Discrepancies between published studies likely reflect variations in study design, patient characteristics, follow-up duration, baseline metabolic control, severity of ocular disease, treatment duration, drug dosage, HbA1c reduction, blood pressure control, and the rate of body weight loss. Additionally, patients with diabetes undergo ophthalmic examinations more frequently, which may increase detection of ocular disorders and influence observed outcomes. Because most currently available data originate from retrospective studies, pharmacovigilance analyses, and case reports, further prospective studies are needed to better evaluate the long-term ocular effects of GLP-1RAs and to determine whether reported ophthalmic complications are directly related to treatment or rather to metabolic changes occurring during therapy. Overall, GLP-1 receptor agonists appear to have potential beneficial effects on ocular tissues; however, careful ophthalmic monitoring should be considered, particularly in patients with preexisting retinal or optic nerve diseases and during the initial period of intensive metabolic improvement.

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