

Review Article

Beyond glycemic control: ocular effects of GLP-1 receptor agonists across medical and surgical ophthalmology

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ABSTRACT

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) have become a cornerstone in the management of type 2 diabetes mellitus and obesity because of their proven metabolic and cardiovascular benefits. As their clinical use continues to expand, increasing attention has been directed toward their potential ophthalmic effects. This narrative review summarizes the current evidence regarding the biological mechanisms, therapeutic potential, and ocular safety profile of GLP-1RAs across medical and surgical ophthalmology. Experimental studies demonstrate that GLP-1 receptors are expressed in multiple ocular tissues and that receptor activation exerts anti-inflammatory, antioxidant, vasculoprotective, and neuroprotective effects through modulation of oxidative stress, mitochondrial homeostasis, and retinal cell survival. Clinically, evidence from cardiovascular outcome trials initially raised concern regarding diabetic retinopathy progression, particularly following the SUSTAIN-6 trial. However, subsequent randomized trials, observational studies, and recent systematic reviews have not demonstrated a consistent class-wide increase in diabetic retinopathy risk. Current evidence suggests that the reported retinal complications are more likely associated with advanced baseline retinal disease and rapid glycemic improvement than with direct retinal toxicity. Emerging data also indicate potential applications in glaucoma, retinal neuroprotection, age-related macular degeneration, and ocular surface disease, although these findings remain largely supported by preclinical or observational evidence. A possible association between semaglutide and nonarteritic anterior ischemic optic neuropathy has recently been reported, but available evidence is insufficient to establish causality. Overall, GLP-1RAs appear to maintain a favorable ocular safety profile while offering promising therapeutic opportunities beyond metabolic disease. Future prospective ophthalmology-specific studies are needed to clarify their long-term ocular effects, identify patients at increased risk of retinal complications, and determine whether their experimental neuroprotective properties translate into clinically meaningful benefits.

Keywords: Glucagon-like peptide-1 receptor agonists, Diabetic retinopathy, Neuroprotection, Glaucoma, Ophthalmology

INTRODUCTION

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) have transformed the management of type 2 diabetes mellitus and obesity by improving glycemic control, promoting clinically meaningful weight loss, and reducing major cardiovascular events in high-risk populations. Large cardiovascular outcome trials, including SUSTAIN-6, PIONEER-6, and LEADER, established the systemic efficacy and cardiovascular safety of semaglutide and liraglutide.^{1,2,3} However, these trials also brought increasing attention to their potential ocular effects.

In SUSTAIN-6, semaglutide was associated with a significantly higher incidence of diabetic retinopathy complications compared with placebo, particularly among patients with pre-existing retinopathy.¹ By contrast, PIONEER-6 did not identify a clear imbalance in retinopathy-related adverse events, while LEADER showed a small but non-significant increase in retinopathy events with liraglutide.^{2,3} These apparently conflicting findings raised concerns regarding whether GLP-1RAs exert a direct adverse effect on the retina or whether the observed risk is primarily related to rapid improvement in glycemic control.

Beyond safety concerns, increasing experimental evidence suggests that GLP-1 receptor activation may exert direct protective effects within ocular tissues. GLP-1 receptors have been described in retinal ganglion cells, retinal pigment epithelial cells, and vascular endothelial cells, supporting potential anti-inflammatory, antioxidant, vascular-stabilizing, and neuroprotective actions.⁴ In preclinical models, GLP-1 analogues have reduced hyperglycemia-induced retinal ganglion cell injury and modulated mitochondrial quality-control pathways, including PINK1/Parkin-mediated mitophagy.⁵ These findings have generated interest in the possible role of GLP-1RAs in diabetic retinopathy, glaucoma, age-related macular degeneration, and ocular surface disease.

Rationale

The ocular implications of GLP-1RAs remain clinically relevant but incompletely defined. Recent systematic reviews and meta-analyses have not demonstrated a significant overall association between GLP-1RA use and the development or progression of diabetic retinopathy.^{6,7} This suggests that the early worsening observed in selected trials may be influenced by baseline retinopathy severity, the magnitude and speed of glycated hemoglobin reduction, and other patient-specific factors rather than by a uniform toxic effect of the drug class.

Nevertheless, real-world data continue to identify potentially vulnerable subgroups. A recent cohort study of patients treated with tirzepatide reported increased odds of incident proliferative diabetic retinopathy among individuals with pre-existing non-proliferative disease or

maculopathy, despite a lower risk of new-onset retinopathy among patients without baseline disease.⁸ In addition, emerging concern regarding nonarteritic anterior ischemic optic neuropathy has broadened the discussion beyond diabetic retinopathy. A recent meta-analysis found no overall increase in ocular disorders or diabetic retinopathy with semaglutide but identified a possible association with nonarteritic anterior ischemic optic neuropathy, although the available evidence was insufficient to establish causality.⁹

At the same time, preclinical studies and observational clinical data suggest possible benefits in retinal neuroprotection, glaucoma prevention, inflammation control, and ocular surface homeostasis.^{4,5,10,11} The coexistence of potential therapeutic effects and unresolved safety signals creates a complex clinical landscape that requires integrated interpretation. This is particularly relevant for ophthalmologists, who increasingly encounter patients receiving GLP-1RAs for diabetes, obesity, and cardiovascular risk reduction.

Objective

This narrative review aims to summarize the current evidence regarding the ocular effects of GLP-1 receptor agonists, with emphasis on their biological mechanisms, potential therapeutic applications, and emerging safety concerns. It also seeks to provide a clinically oriented overview of their implications across medical and surgical ophthalmology, including diabetic retinopathy, glaucoma, retinal neuroprotection, ocular surface disease, nonarteritic anterior ischemic optic neuropathy, and perioperative considerations.

METHODS

Literature search

A narrative literature review was conducted to identify studies evaluating the ocular effects of glucagon-like peptide-1 receptor agonists (GLP-1RAs). The literature search was performed using PubMed/MEDLINE, Scopus, and Google Scholar. Search terms included combinations of "GLP-1 receptor agonist," "semaglutide," "liraglutide," "tirzepatide," "diabetic retinopathy," "glaucoma," "retinal neuroprotection," "ocular surface disease," "nonarteritic anterior ischemic optic neuropathy," and "ophthalmology." Additional relevant articles were identified through manual screening of reference lists from selected publications. Priority was given to studies published between 2016 and 2026, reflecting the period of greatest clinical adoption of GLP-1RAs and the emergence of evidence regarding their ophthalmic implications.

Selection criteria

Original clinical studies, randomized controlled trials, observational studies, systematic reviews, meta-analyses,

and relevant experimental investigations evaluating the ocular effects of GLP-1RAs were considered for inclusion. Preference was given to studies addressing diabetic retinopathy, glaucoma, retinal neuroprotection, ocular surface disease, optic neuropathy, and other clinically relevant ophthalmic outcomes. Narrative reviews, editorials, conference abstracts, duplicate publications, and studies lacking direct relevance to ophthalmology or GLP-1 receptor agonists were excluded. The selected literature was synthesized narratively to provide a clinically oriented overview of the current evidence

Current evidence on GLP-1 receptor agonists in ophthalmology

Biological basis of GLP-1 receptor signaling in ocular tissues

Growing evidence suggests that the biological effects of glucagon-like peptide-1 receptor agonists (GLP-1RAs) extend well beyond glucose regulation. Experimental studies have demonstrated the expression of GLP-1 receptors in multiple ocular structures, including retinal ganglion cells, retinal pigment epithelial cells, vascular endothelial cells, Müller glia, and other neural retinal components, providing a biological rationale for their potential therapeutic role in ophthalmology.^{8,12} Activation of these receptors appears to regulate several intracellular pathways involved in oxidative stress, inflammation, apoptosis, mitochondrial homeostasis, and vascular integrity.

Among the proposed mechanisms, suppression of pro-inflammatory cytokines, attenuation of oxidative stress, preservation of the blood-retinal barrier, and modulation of microglial activation have received the greatest attention. These effects may reduce retinal neurovascular injury and slow the progression of chronic retinal diseases characterized by persistent inflammation and metabolic dysfunction.^{8,13,14} In addition, GLP-1R activation has been associated with improved mitochondrial function, reduced reactive oxygen species production, and enhanced cellular survival through activation of anti-apoptotic signaling pathways.

Neuroprotection has emerged as one of the most promising areas of GLP-1RA research. Zhou et al. demonstrated that GLP-1 analogs significantly reduced retinal ganglion cell injury in diabetic experimental models by regulating PINK1/Parkin-mediated mitophagy, thereby preserving mitochondrial integrity and reducing neuronal apoptosis.¹³ These findings support the hypothesis that GLP-1RAs may protect retinal neurons independently of their systemic metabolic effects. Similarly, recent mechanistic reviews have proposed additional pathways involving ERK1/2 signaling, calcium homeostasis, GABAergic modulation, and inhibition of inflammatory cell recruitment, suggesting that GLP-

1RAs act through multiple complementary mechanisms rather than a single molecular target.¹⁴

Despite these encouraging findings, most mechanistic evidence originates from animal models and in vitro studies. Differences in receptor distribution, ocular pharmacokinetics, and disease progression between experimental models and humans limit direct clinical translation. Consequently, although the biological rationale for ocular protection is increasingly compelling, confirmation through prospective human studies remains necessary before these mechanisms can be translated into routine ophthalmic practice.^{8,12,14} Figure 1 summarizes the proposed biological mechanisms and clinical implications of GLP-1 receptor agonists in ophthalmology.

Current clinical evidence

Clinical evidence regarding the ophthalmic effects of GLP-1 receptor agonists has expanded considerably during the past decade. However, available studies demonstrate a heterogeneous pattern in which potential therapeutic benefits coexist with concerns regarding retinal safety, particularly in patients with diabetic retinopathy.

Interest in this field originated following the publication of SUSTAIN-6, in which semaglutide significantly reduced major cardiovascular events but was unexpectedly associated with an increased incidence of diabetic retinopathy complications compared with placebo (HR 1.76, 95% CI 1.11–2.78).¹ Importantly, most affected patients had pre-existing diabetic retinopathy, and retinal complications appeared early after treatment initiation, suggesting that rapid metabolic improvement rather than direct retinal toxicity could explain the observed findings.

Subsequent cardiovascular outcome trials produced less concerning results. In PIONEER-6, oral semaglutide did not demonstrate a clinically meaningful imbalance in retinopathy-related adverse events, with most reported cases corresponding to mild non-proliferative disease identified during routine ophthalmic examinations.² Likewise, the LEADER trial reported a numerically higher incidence of retinopathy among liraglutide-treated patients, although this difference did not reach statistical significance.³ Collectively, these studies indicate that the retinal signal observed in SUSTAIN-6 may not represent a uniform class effect.

More recent evidence has further clarified this apparent discrepancy. Two contemporary systematic reviews and meta-analyses independently concluded that GLP-1 receptor agonists are not associated with a significant overall increase in diabetic retinopathy progression.^{7,14} Yang et al reported a pooled relative risk of 1.07 (95% CI 0.90–1.28), whereas Alwafi et al found a pooled relative risk of 1.00 (95% CI 0.71–1.43), supporting an overall

neutral retinal safety profile despite isolated signals observed in individual studies.^{7,14}

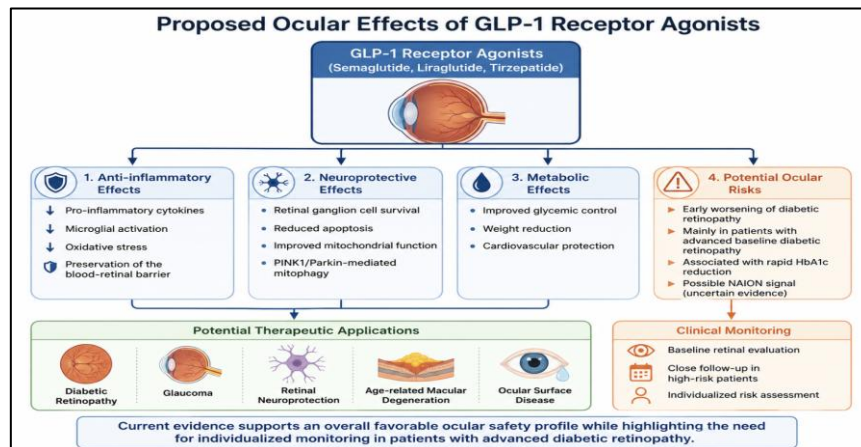


Figure 1: Proposed mechanisms and clinical implications of GLP-1 receptor agonists in ophthalmology.

Proposed mechanisms underlying the ophthalmic effects of glucagon-like peptide-1 receptor agonists (GLP-1RAs). GLP-1 receptor activation exerts anti-inflammatory, antioxidant, vasculoprotective, and neuroprotective effects through modulation of oxidative stress, microglial activation, mitochondrial homeostasis, and retinal ganglion cell survival. These mechanisms may contribute to potential therapeutic applications in diabetic retinopathy, glaucoma, retinal neuroprotection, age-related macular degeneration, and ocular surface disease. Conversely, current clinical evidence suggests that patients with advanced pre-existing diabetic retinopathy may experience transient early worsening following rapid glycemic improvement, highlighting the importance of individualized ophthalmic monitoring during treatment initiation. NAION = nonarteritic anterior ischemic optic neuropathy.

The concept of early worsening of diabetic retinopathy has become the most accepted explanation for these apparently conflicting findings. Bain et al. emphasized that rapid reductions in glycated hemoglobin have historically been associated with transient worsening of diabetic retinopathy following intensive insulin therapy, bariatric surgery, and pregnancy, suggesting that the phenomenon predates GLP-1 receptor agonists themselves.⁵ Accordingly, rapid metabolic improvement, rather than direct pharmacologic retinal toxicity, is currently considered the most plausible explanation for retinal deterioration observed in selected high-risk patients.

Real-world studies nevertheless suggest that baseline retinal status remains clinically important. Buckley et al. recently reported that tirzepatide therapy was associated with increased odds of incident proliferative diabetic retinopathy among patients with pre-existing retinal disease, whereas individuals without baseline diabetic retinopathy experienced a lower incidence of new retinal disease.⁴ These observations reinforce the importance of individualized ophthalmic monitoring during treatment initiation rather than supporting universal concern regarding retinal toxicity.

Beyond diabetic retinopathy, accumulating observational evidence suggests possible protective effects in glaucoma and retinal neurodegeneration. Experimental neuroprotection, together with epidemiological studies reporting lower glaucoma incidence among GLP-1RA users, has generated considerable interest in their potential disease-modifying role.^{12,14} Emerging evidence

also suggests possible benefits for age-related macular degeneration, dry eye disease, and ocular surface inflammation; however, these findings remain preliminary and require confirmation in prospective clinical trials.^{12,14}

Ocular safety, limitations and future directions

Although current evidence is generally reassuring, several important safety considerations remain unresolved. The strongest clinical concern continues to be early worsening of diabetic retinopathy among patients with advanced pre-existing retinal disease undergoing rapid glycemic improvement. Careful ophthalmic evaluation before initiating treatment and close follow-up during the first months of therapy may therefore be appropriate in selected high-risk individuals.^{1,4,5}

Another emerging topic is nonarteritic anterior ischemic optic neuropathy (NAION). A recent systematic review and meta-analysis involving more than 73,000 participants found no increase in overall ocular adverse events or diabetic retinopathy with semaglutide but identified a possible association with NAION. Nevertheless, the number of reported cases was extremely small, and the available evidence remains insufficient to establish a causal relationship.¹¹ Consequently, clinicians should interpret this signal cautiously while awaiting additional prospective studies.

Several limitations characterize the current body of literature. Most available clinical evidence consists of retrospective cohort studies or cardiovascular trials in

which ophthalmic outcomes were secondary endpoints. Definitions of diabetic retinopathy progression vary considerably among studies, follow-up duration is frequently limited, and few investigations incorporate standardized retinal imaging or visual function assessments. Furthermore, much of the mechanistic evidence derives from experimental models, limiting direct extrapolation to clinical practice.

Future research should prioritize prospective ophthalmology-focused randomized clinical trials incorporating standardized retinal endpoints, multimodal imaging, long-term follow-up, and evaluation of patient subgroups at greatest risk for retinal complications. Additional investigation is also required to determine whether the neuroprotective and anti-inflammatory properties observed experimentally can ultimately translate into disease-modifying therapies for glaucoma, diabetic retinopathy, and other chronic retinal disorders.

DISCUSSION

The ophthalmic effects of glucagon-like peptide-1 receptor agonists represent a clinically complex interaction between systemic metabolic improvement, baseline ocular vulnerability, and potentially direct actions within retinal and neural tissues. The available evidence does not support a simple interpretation of GLP-1RAs as either uniformly protective or inherently harmful to the eye. Instead, their ocular effects appear to depend on the clinical context, particularly the presence and severity of pre-existing diabetic retinopathy, the magnitude and velocity of glycemic improvement, the specific agent used, and the ophthalmic outcome under evaluation.

The diabetic retinopathy signal observed in SUSTAIN-6 remains the principal source of concern regarding GLP-1RA therapy. Semaglutide was associated with a significantly higher incidence of retinopathy complications than placebo, and this finding initially raised the possibility of direct retinal toxicity.¹ However, several features of the trial argue against a straightforward pharmacological toxic effect. Events occurred predominantly in patients with established retinopathy, poor baseline glycemic control, insulin use, and substantial reductions in glycated hemoglobin. Furthermore, the complications emerged relatively early after treatment initiation, a temporal pattern compatible with the previously recognized phenomenon of early worsening of diabetic retinopathy following rapid metabolic correction.⁵

The absence of a comparable statistically significant signal in PIONEER-6 and LEADER further weakens the hypothesis of a uniform class-wide retinal toxicity.^{2,3} Nevertheless, direct comparisons among these trials should be made cautiously. Differences in baseline ophthalmic risk, exclusion criteria, treatment exposure, glycemic trajectories, and ascertainment of retinal events

may partly explain their divergent findings. In particular, PIONEER-6 excluded patients with proliferative retinopathy or maculopathy requiring acute treatment, thereby selecting a population with lower immediate ocular risk than that included in SUSTAIN-6.² These methodological differences illustrate how baseline retinal phenotype may substantially influence the apparent safety profile of GLP-1RA therapy.

The findings of contemporary meta-analyses provide broader reassurance. Neither Yang et al. nor Alwafi et al. identified a significant overall association between GLP-1RA exposure and the development or progression of diabetic retinopathy.^{7,14} Their pooled estimates were close to neutrality, suggesting that the SUSTAIN-6 finding should not automatically be generalized to all GLP-1RAs or all patients with diabetes. At the same time, a neutral pooled effect does not exclude clinically important risk in selected subgroups. Meta-analytic estimates may obscure heterogeneous responses when patients with no baseline retinopathy are combined with those who have advanced non-proliferative or proliferative disease.

This distinction is supported by the real-world findings reported by Buckley et al. Patients without diabetic retinopathy at baseline appeared to have a reduced probability of developing new retinal disease during tirzepatide treatment, whereas those with pre-existing non-proliferative retinopathy or maculopathy had increased odds of incident proliferative disease.⁴ Although confounding cannot be excluded and tirzepatide is a dual GIP/GLP-1 receptor agonist rather than a selective GLP-1RA, these results reinforce the concept that baseline retinal status may be more relevant than drug exposure alone. The same treatment may therefore be associated with long-term microvascular benefit in patients with limited retinal damage while transiently destabilizing disease in those with advanced microvascular pathology and rapid glycemic improvement.

From a mechanistic perspective, the hypothesis of direct retinal toxicity is also difficult to reconcile with the growing experimental literature. GLP-1 receptor activation has been associated with anti-inflammatory, antioxidant, anti-apoptotic, and vascular-stabilizing effects within the retina.^{8,14} In experimental diabetic models, GLP-1 analogues protected retinal ganglion cells and improved mitochondrial quality control through PINK1/Parkin-mediated mitophagy.¹³ These biological effects suggest that GLP-1RAs could potentially attenuate chronic neurovascular injury rather than promote it. The apparent contradiction between early clinical worsening and experimental neuroprotection may therefore be temporal: rapid systemic metabolic shifts could transiently aggravate an unstable retinal microvasculature, whereas sustained receptor activation and improved metabolic control may provide longer-term neurovascular benefit.

This temporal framework may help reconcile the current evidence. Early worsening of diabetic retinopathy is not unique to GLP-1RAs and has previously been described after intensive insulin therapy, bariatric surgery, and pregnancy-related changes in glucose regulation.⁵ The underlying mechanisms remain incompletely understood but may involve changes in retinal blood flow, osmotic balance, vascular endothelial growth factor signaling, and autoregulatory responses following abrupt glucose reduction. Therefore, the rate of metabolic improvement may be a more plausible mediator of early retinal deterioration than a direct toxic action of GLP-1 receptor stimulation.

Beyond diabetic retinopathy, the possible ophthalmic benefits of GLP-1RAs remain promising but substantially less established. Preclinical evidence supports retinal ganglion cell preservation and suppression of neuroinflammation, providing a rationale for their potential use in glaucoma and other neurodegenerative ocular conditions.¹²⁻¹⁴ Observational studies reporting reduced glaucoma incidence among GLP-1RA users are biologically plausible, but they cannot determine whether the association results from direct neuroprotection, improved vascular and metabolic health, or differences in patient characteristics and healthcare utilization. Prospective studies using structural and functional glaucoma endpoints are therefore required before a disease-modifying role can be proposed.

Evidence regarding age-related macular degeneration and ocular surface disease is even more preliminary. Anti-inflammatory and vascular effects could theoretically influence choroidal neovascularization, retinal pigment epithelial dysfunction, tear-film homeostasis, and corneal epithelial integrity.^{12,14} However, current findings are inconsistent and predominantly derived from experimental studies or retrospective databases. These potential applications should therefore be regarded as hypothesis-generating rather than clinically established indications.

The possible association between semaglutide and nonarteritic anterior ischemic optic neuropathy introduces an additional layer of uncertainty. Natividade et al. found no increase in overall ocular disorders or diabetic retinopathy but reported a statistical association with NAION.¹¹ This signal requires careful interpretation because the event was extremely uncommon, confidence intervals were wide, and the accumulated evidence was insufficient to establish a definitive association. Moreover, patients receiving GLP-1RAs frequently have diabetes, obesity, hypertension, dyslipidemia, obstructive sleep apnea, and other established vascular risk factors for NAION. Residual confounding and detection bias may therefore contribute to the observed relationship. At present, the evidence supports clinical awareness rather than treatment avoidance or causal attribution.

These findings have several practical implications. Routine ophthalmic screening should continue to follow established diabetes care recommendations, but additional attention may be appropriate in patients with moderate-to-severe diabetic retinopathy, active macular disease, poor baseline glycemic control, or an anticipated rapid decline in glycated hemoglobin. In these individuals, baseline retinal assessment and closer follow-up after treatment initiation may allow early identification of clinically meaningful progression. Importantly, the available evidence does not justify withholding GLP-1RAs from most patients, given their substantial glycemic, cardiovascular, renal, and weight-related benefits. Clinical decisions should balance systemic benefit against individualized ocular risk rather than treating isolated safety signals as evidence of universal harm.

The present review also highlights important limitations in the literature. Most major trials were designed around cardiovascular rather than ophthalmic outcomes, retinal assessments were not consistently standardized, and event definitions varied considerably. Observational studies remain vulnerable to confounding by indication, surveillance intensity, comorbidity burden, and differences in diabetes severity. Additionally, much of the proposed therapeutic evidence comes from animal models whose receptor expression, ocular pharmacokinetics, and disease course may not accurately reproduce human pathology.

Future research should move beyond passive reporting of ocular adverse events and incorporate prospectively defined ophthalmic endpoints. Trials should stratify participants by baseline retinopathy severity, quantify the rate and magnitude of glycated hemoglobin reduction, and use standardized fundus photography, optical coherence tomography, optical coherence tomography angiography, and visual function measures. Studies should also distinguish early transient worsening from sustained structural progression. For glaucoma and retinal neurodegeneration, longitudinal investigations combining ganglion cell complex thickness, retinal nerve fiber layer measurements, visual fields, and neuroinflammatory biomarkers may clarify whether the experimental neuroprotective effects of GLP-1R activation translate into meaningful clinical benefit.

Overall, the current evidence favors a context-dependent interpretation. GLP-1RAs do not appear to produce a consistent increase in diabetic retinopathy across populations, but rapid metabolic improvement may precipitate early worsening in patients with advanced baseline disease. Simultaneously, their anti-inflammatory and neuroprotective properties raise the possibility of longer-term ocular benefit. Resolving this apparent paradox will require ophthalmology-specific prospective studies capable of separating drug-specific effects from the consequences of rapid systemic metabolic change.

CONCLUSION

Glucagon-like peptide-1 receptor agonists have expanded far beyond their original role as glucose-lowering agents and are increasingly recognized for their potential ophthalmic implications. Current evidence suggests that these agents do not confer a consistent class-wide increase in the risk of diabetic retinopathy progression. Rather, the retinal complications observed in selected studies appear to be largely influenced by baseline disease severity and the rapidity of glycemic improvement, emphasizing the importance of individualized patient assessment rather than generalized concern regarding retinal toxicity.

At the same time, accumulating experimental and emerging clinical evidence highlights the potential neuroprotective, anti-inflammatory, and vasculoprotective properties of GLP-1 receptor activation, supporting continued investigation into their role in diabetic retinopathy, glaucoma, and other retinal disorders. Although these findings are encouraging, their translation into routine ophthalmic practice remains limited by the predominance of preclinical studies and the scarcity of prospective trials specifically designed to evaluate ocular outcomes.

Overall, the available literature supports a balanced interpretation of GLP-1RA therapy. These agents offer substantial systemic metabolic and cardiovascular benefits while maintaining an overall favorable ocular safety profile in most patients. Nevertheless, individuals with advanced pre-existing diabetic retinopathy may benefit from closer ophthalmic monitoring during treatment initiation, particularly when rapid improvements in glycemic control are anticipated. Future ophthalmology-focused studies integrating standardized retinal imaging, functional visual outcomes, and long-term follow-up will be essential to clarify whether the promising biological effects of GLP-1 receptor activation can ultimately translate into disease-modifying therapies for retinal and neurodegenerative eye diseases.

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