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HbA1c AND THE RISK OF DIABETIC RETINOPATHY PROGRESSION: CURRENT EVIDENCE AND CLINICAL IMPLICATIONS

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ABSTRACT

Background: Diabetic retinopathy (DR) is one of the most common microvascular complications of diabetes mellitus and remains a leading cause of preventable vision loss worldwide. Glycated hemoglobin (HbA1c), reflecting long-term glycemic control, is widely recognized as an important biomarker for assessing the risk of diabetic complications.

Objective: This narrative review aimed to summarize current evidence regarding the relationship between HbA1c levels and the development and progression of diabetic retinopathy, with particular emphasis on its clinical significance in risk assessment and disease prevention.

Methods: A review of landmark randomized controlled trials, epidemiological studies, systematic reviews, meta-analyses, and current clinical guidelines was conducted. Evidence from the Diabetes Control and Complications Trial (DCCT), the United Kingdom Prospective Diabetes Study (UKPDS), the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR), ACCORD, ADVANCE, and recent reviews was synthesized to evaluate the role of HbA1c in diabetic retinopathy.

Results: The available evidence consistently demonstrates that elevated HbA1c levels are strongly associated with an increased risk of developing and progressing diabetic retinopathy in both type 1 and type 2 diabetes mellitus. Sustained improvement in glycemic control significantly reduces the risk of retinal complications, although no universally accepted HbA1c threshold predicting disease onset has been established. HbA1c should therefore be interpreted as a continuous biomarker of retinal risk alongside other clinical risk factors.

Conclusions: HbA1c remains one of the most valuable modifiable predictors of diabetic retinopathy and an essential component of individualized diabetes management. Optimizing long-term glycemic control, combined with regular ophthalmologic assessment and comprehensive risk factor management, remains the cornerstone of preventing diabetic retinopathy progression and preserving visual function.

KEYWORDS

Glycated Hemoglobin (HbA1c); Diabetic Retinopathy; Diabetes Mellitus; Glycemic Control; Microvascular Complications; Retinal Disease

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Introduction

Diabetes mellitus is a chronic metabolic disorder characterized by elevated blood glucose levels resulting from either absolute insulin deficiency (type 1 diabetes) or a combination of insulin resistance and progressive pancreatic β -cell dysfunction (type 2 diabetes).

Long-standing, poorly controlled diabetes may lead to the development of microvascular complications, including diabetic retinopathy, which is caused by progressive damage to the retinal microcirculation. Diabetic retinopathy may manifest clinically with visual disturbances, scotomas, and in advanced stages can lead to irreversible vision loss and blindness. Glycated hemoglobin (HbA1c) is a key biomarker of long-term glycemic control and reflects average blood glucose levels over the preceding 2–3 months. Its clinical importance lies in its ability to assess chronic glycemic exposure, enabling earlier identification of patients at increased risk of diabetic complications and guiding timely therapeutic interventions.

Several studies have demonstrated a strong association between elevated HbA1c levels and the risk of developing and progressing diabetic retinopathy. However, the exact relationship between specific HbA1c values and the risk stratification of retinal complications remains a subject of ongoing research. This review summarizes current evidence regarding the association between HbA1c and diabetic retinopathy progression, with particular emphasis on its clinical utility as a continuous biomarker of retinal risk.

Methodology

This study was conducted as a narrative literature review aimed at summarizing current evidence on the association between glycated hemoglobin (HbA1c) and the development and progression of diabetic retinopathy. Relevant scientific publications were identified through a review of peer-reviewed articles, landmark randomized controlled trials, epidemiological studies, systematic reviews, meta-analyses, and current clinical practice guidelines addressing glycemic control and diabetic retinopathy.

The literature selection focused on studies evaluating the role of HbA1c as a predictor of diabetic retinopathy in patients with type 1 and type 2 diabetes mellitus. Particular attention was given to high-quality evidence, including the Diabetes Control and Complications Trial (DCCT), the United Kingdom Prospective Diabetes Study (UKPDS), the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR), the ACCORD Eye Study, the ADVANCE trial, and the American Diabetes Association Standards of Care.

The collected evidence was analyzed qualitatively and synthesized to provide an overview of the current understanding of the relationship between HbA1c and diabetic retinopathy. Emphasis was placed on the clinical significance of long-term glycemic control, the pathophysiological mechanisms linking chronic hyperglycemia with retinal damage, and the practical implications of HbA1c for risk assessment, prevention, and disease management.

HbA1c as a marker

Glycated hemoglobin (HbA1c) is a biomarker that reflects average blood glucose levels over the preceding 2–3 months and is widely used as a standard tool for long-term glycemic monitoring in both type 1 and type 2 diabetes mellitus [10]. It is currently considered a fundamental parameter in the assessment of glycemic control in diabetic patients [20].

Recent studies have demonstrated a strong association between persistently elevated HbA1c levels and both the incidence and severity of diabetic complications [1, 2]. Moreover, evidence suggests that the relationship between HbA1c and microvascular complications is continuous and approximately linear, with no clearly defined safe threshold below which the risk is eliminated [3]

Pathophysiology of diabetic retinopathy

Diabetic retinopathy is a progressive microvascular complication of diabetes mellitus associated with structural and functional alterations in the retinal vasculature. Chronic hyperglycemia plays a key role in activating multiple biochemical pathways that contribute to vascular damage [6,8].

Persistently elevated blood glucose levels promote the formation of advanced glycation end-products (AGEs), which accumulate within the vessel walls and alter their structural integrity and function [11]. At the same time, hyperglycemia induces oxidative stress and activates inflammatory pathways, leading to endothelial dysfunction and increased vascular permeability [12].

These processes contribute to pericyte loss, thickening of the capillary basement membrane, and disruption of the blood–retinal barrier. As the disease progresses, capillary occlusion and retinal ischemia develop, resulting in tissue hypoxia.

In response to hypoxic conditions, there is upregulation of vascular endothelial growth factor (VEGF), which promotes pathological neovascularization and progression to proliferative diabetic retinopathy [13,14]. If left untreated, these changes may ultimately lead to irreversible vision loss.

HbA1c and risk of diabetic retinopathy progression

Glycated hemoglobin (HbA1c) is the standard biomarker for assessing long-term glycemic control and one of the most important predictors of the development and progression of diabetic retinopathy (DR). The landmark Diabetes Control and Complications Trial (DCCT) demonstrated that intensive glycemic control significantly reduced both the incidence and progression of diabetic retinopathy in patients with type 1 diabetes compared with conventional therapy, establishing HbA1c as a major determinant of microvascular outcomes [1]. Similarly, the United Kingdom Prospective Diabetes Study (UKPDS) showed that intensive blood glucose control significantly reduced the risk of microvascular complications, including diabetic retinopathy, in patients with newly diagnosed type 2 diabetes [2]. Furthermore, the epidemiological analysis of the UKPDS cohort demonstrated a continuous relationship between HbA1c levels and the risk of microvascular complications, showing that each 1% reduction in HbA1c was associated with an approximately 35–37% decrease in the risk of microvascular disease [3].

The Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR) further identified poor glycemic control as one of the strongest predictors of both the onset and progression of diabetic retinopathy, independent of diabetes duration [4,5]. These findings have been consistently supported by subsequent epidemiological studies and systematic reviews, which demonstrated that elevated HbA1c levels are associated with a higher prevalence and greater severity of diabetic retinopathy, as well as an increased risk of progression to vision-threatening complications, including proliferative diabetic retinopathy and diabetic macular edema [6–9,15,19]. In addition, a meta-analysis by Jin et al. confirmed that higher HbA1c levels are independently associated with a significantly increased risk of diabetic retinopathy in patients with type 2 diabetes mellitus [16].

The association between elevated HbA1c and retinal damage is supported by well-established pathophysiological mechanisms. Chronic hyperglycemia promotes oxidative stress, the accumulation of advanced glycation end products (AGEs), activation of protein kinase C, increased flux through the polyol pathway, and persistent inflammatory responses. These interconnected processes lead to endothelial dysfunction, pericyte loss, capillary occlusion, disruption of the blood–retinal barrier, and progressive retinal ischemia, ultimately resulting in neurovascular damage of the retina [11–14].

More recent randomized clinical trials have reinforced the importance of intensive glycemic control in preventing diabetic retinopathy progression. The ACCORD Eye Study demonstrated that intensive glucose lowering significantly reduced the 4-year progression of diabetic retinopathy compared with standard therapy [17]. Likewise, the ADVANCE trial reported an overall reduction in microvascular complications, although the effect on diabetic retinopathy alone was less pronounced [18]. Importantly, rapid reductions in HbA1c may occasionally result in transient early worsening of diabetic retinopathy, particularly in patients with advanced pre-existing retinal disease. Therefore, gradual optimization of glycemic control, together with regular ophthalmologic examinations, is recommended to minimize this risk while preserving the long-term benefits of intensive treatment [14,17]. Current American Diabetes Association (ADA) guidelines recommend individualized HbA1c targets, generally below 7% for most non-pregnant adults, as an effective strategy for reducing the risk of diabetic retinopathy progression while minimizing the risk of hypoglycemia [20].

Table 1. Landmark clinical studies evaluating the association between HbA1c and diabetic retinopathy.

Study	Study Design	Population	Main findings
DCCT (1993)	RCT	Type 1 DM	Intensive glycemic control significantly reduced the incidence and progression of diabetic retinopathy.
UKPDS (1998)	RCT	Newly diagnosed Type 2 DM	Intensive glucose control reduced microvascular complications, including diabetic retinopathy.
UKPDS 35 (2000)	Prospective cohort	Type 2 DM	Each 1% reduction in HbA1c was associated with approximately 35–37% lower risk of microvascular complications.
WESDR	Epidemiological study	Type 1 and Type 2 DM	Poor glycemic control independently predicted diabetic retinopathy development and progression.
Yau et al.	Meta-analysis	Global population	Elevated HbA1c was consistently associated with increased prevalence of diabetic retinopathy.
Jin et al.	Meta-analysis	Type 2 DM	Higher HbA1c independently increased diabetic retinopathy risk.
ACCORD Eye	RCT	Type 2 DM	Intensive glucose lowering reduced diabetic retinopathy progression.
ADVANCE	RCT	Type 2 DM	Intensive glycemic control reduced overall microvascular complications.

HbA1c Levels and the Risk of Diabetic Retinopathy: Clinical Interpretation

From a clinical perspective, the relationship between HbA1c and diabetic retinopathy (DR) should be interpreted as a continuous risk spectrum rather than a dichotomous exposure–outcome relationship. Although numerous epidemiological studies have demonstrated that higher HbA1c levels are associated with an increased risk of developing DR, this association does not define a single universal cutoff value. Instead, HbA1c should be regarded as a dynamic biomarker reflecting cumulative glycemic exposure over time, which influences both the initiation and progression of retinal microvascular damage [3,6–9,15,19].

In clinical practice, this continuous relationship has important implications for risk assessment. Patients with persistently elevated HbA1c levels remain at progressively higher risk of developing advanced stages of diabetic retinopathy, including proliferative diabetic retinopathy and diabetic macular edema, which represent the leading causes of vision loss among individuals with diabetes [6–9,15,19]. At the same time, the absence of a clearly defined HbA1c threshold suggests that even modest improvements in glycemic control may result in clinically meaningful reductions in long-term ocular risk.

Rather than relying on a fixed cutoff value, current management strategies emphasize individualized glycemic targets. The American Diabetes Association recommends an HbA1c target of generally <7.0% for most non-pregnant adults while highlighting that treatment goals should be individualized according to factors such as patient age, duration of diabetes, comorbidities, life expectancy, and the risk of hypoglycemia [20]. This individualized approach aims to balance the reduction of microvascular complications with the avoidance of treatment-related adverse events.

Importantly, the clinical benefits of HbA1c reduction should also be interpreted within a temporal context. Although sustained improvement in glycemic control is associated with a lower risk of diabetic retinopathy development and progression, rapid reductions in HbA1c may occasionally lead to transient early worsening of retinopathy, particularly in patients with advanced pre-existing retinal disease [14,17]. Therefore, gradual optimization of glycemic control, accompanied by regular ophthalmologic monitoring, is recommended in patients at high risk of retinopathy progression.

Overall, the clinical interpretation of HbA1c focuses on risk stratification and long-term risk reduction rather than the identification of a single threshold for disease development. Maintaining HbA1c as close as safely possible to the individualized therapeutic target remains one of the most effective strategies for preventing the onset and progression of diabetic retinopathy, although glycemic management should always be integrated into a comprehensive approach that includes individualized patient care and regular ophthalmologic surveillance [1–3,20].

Clinical Implications

The available evidence indicates that HbA1c should be considered one of the key components of diabetic retinopathy (DR) risk assessment rather than solely a marker of glycemic control. The consistent association between elevated HbA1c levels and both the development and progression of DR highlights the importance of incorporating HbA1c into routine clinical decision-making for patients with both type 1 and type 2 diabetes mellitus [1–9,15,16,19].

One of the most important clinical implications of current evidence is that the risk of diabetic retinopathy increases progressively with worsening glycemic control rather than after exceeding a specific HbA1c threshold. Therefore, clinicians should interpret HbA1c as a continuous indicator of retinal risk rather than relying on a single cutoff value. Even modest improvements in glycemic control may translate into meaningful reductions in the long-term risk of microvascular complications [1–3]. This concept supports early therapeutic intervention aimed at optimizing glycemic control before irreversible retinal damage develops.

HbA1c should also be interpreted within a broader clinical context. Although it is one of the strongest modifiable predictors of diabetic retinopathy, retinal outcomes are influenced by multiple factors, including diabetes duration, patient age, blood pressure, lipid profile, and the presence of other microvascular complications [4,6–9,15,19]. Consequently, HbA1c should not be considered an isolated predictor but rather an integral component of a comprehensive risk assessment that also incorporates regular ophthalmologic examinations and individual patient characteristics.

Clinical trial evidence further emphasizes the importance of balancing the benefits and potential risks of intensive glucose-lowering therapy. Although sustained improvement in glycemic control reduces the long-term risk of diabetic retinopathy progression, rapid reductions in HbA1c may be associated with transient early worsening of retinal disease, particularly in patients with advanced pre-existing retinopathy [14,17]. Therefore,

patients undergoing substantial intensification of glucose-lowering therapy should receive appropriate ophthalmologic surveillance to facilitate early detection and management of retinal changes.

Overall, HbA1c remains one of the most valuable biomarkers for predicting the development and progression of diabetic retinopathy and for guiding preventive strategies. However, its greatest clinical value lies not in defining a single threshold for disease onset but in supporting continuous risk assessment, individualized treatment planning, and multidisciplinary management aimed at preserving visual function and reducing the long-term burden of diabetic eye disease [1–3,20].

Limitations

Despite the well-established association between HbA1c and diabetic retinopathy (DR), several limitations should be considered when interpreting the available evidence. First, although HbA1c is widely recognized as the standard biomarker of long-term glycemic control, it reflects average blood glucose concentrations over approximately 2–3 months and does not capture short-term glycemic fluctuations or day-to-day variability in glucose levels, which may also contribute to the development of diabetic complications [10,20].

Another important limitation is the absence of a universally accepted HbA1c threshold that reliably predicts the onset or progression of diabetic retinopathy. Most epidemiological studies and randomized clinical trials demonstrate a continuous association between HbA1c and retinal risk rather than identifying a single cutoff value applicable to all patients [1–3,6–9,15,16,19]. Therefore, HbA1c should always be interpreted in the context of the individual patient and in combination with other established clinical risk factors.

Furthermore, diabetic retinopathy is a multifactorial disease. Although HbA1c is one of the strongest modifiable predictors of diabetic retinopathy, disease development and progression are also influenced by diabetes duration, blood pressure, lipid abnormalities, age, and other systemic factors [4,6–9,15,19]. Consequently, HbA1c alone cannot accurately predict the occurrence or progression of diabetic retinopathy and should be considered as one component of a comprehensive risk assessment.

It should also be acknowledged that the evidence summarized in this review originates from studies involving heterogeneous patient populations, including individuals with both type 1 and type 2 diabetes mellitus, varying durations of diabetes, different treatment strategies, and diverse definitions of diabetic retinopathy outcomes [1–9,15–19]. These methodological differences may limit the direct comparability of study findings and their generalizability to specific patient populations.

Although numerous clinical trials have demonstrated that sustained intensive glycemic control reduces the long-term risk of diabetic retinopathy progression, rapid reductions in HbA1c may occasionally result in transient early worsening of retinal disease in selected patients with advanced pre-existing retinopathy [14,17]. This observation highlights the importance of gradual optimization of glycemic control and individualized treatment strategies accompanied by regular ophthalmologic monitoring.

Overall, HbA1c remains an essential biomarker for assessing the risk of diabetic retinopathy. However, it should be interpreted as one component of a comprehensive clinical assessment rather than as an isolated predictor of retinal outcomes. Future research should focus on improving individualized risk stratification and optimizing strategies for preventing diabetic retinopathy progression across diverse patient populations.

Conclusions

The available evidence consistently demonstrates that HbA1c is one of the most important modifiable predictors of the development and progression of diabetic retinopathy (DR) in both type 1 and type 2 diabetes mellitus. Large randomized clinical trials, epidemiological studies, and systematic reviews have consistently shown that improved glycemic control is associated with a substantial reduction in the risk of retinal complications, whereas persistently elevated HbA1c levels significantly increase the likelihood of developing vision-threatening diabetic retinopathy [1–9,15–20].

Current evidence indicates that the relationship between HbA1c and diabetic retinopathy is continuous rather than defined by a single threshold value. The risk of retinal damage increases progressively with cumulative exposure to hyperglycemia, highlighting the importance of long-term glycemic control. Therefore, HbA1c should be considered as a key biomarker within a comprehensive risk assessment that also includes diabetes duration, blood pressure, lipid abnormalities, renal function, and individual patient characteristics [3,11–16,19,20].

From a clinical perspective, maintaining HbA1c within individualized therapeutic targets remains one of the primary goals of diabetes management and one of the most effective strategies for preventing the onset

and progression of diabetic retinopathy. However, intensive glycemic control should be implemented gradually in patients at high risk of retinal complications, particularly those with advanced diabetic retinopathy or poor baseline glycemic control, as rapid reductions in HbA1c may lead to transient early worsening of retinal disease. Consequently, glycemic optimization should always be accompanied by regular ophthalmologic examinations and comprehensive management of all modifiable cardiovascular and metabolic risk factors [14,17,20].

In conclusion, HbA1c is a robust and clinically valuable biomarker for assessing the risk of diabetic retinopathy and guiding long-term prevention and treatment strategies. Although no universally accepted HbA1c cutoff value for predicting the development or progression of diabetic retinopathy has been established, the consistent evidence supporting the benefits of sustained glycemic control reinforces the central role of HbA1c in preventing retinal complications and preserving visual function in patients with diabetes mellitus [1–3, 16, 20].

Ethics approval: Not required. This narrative review is based exclusively on previously published literature and does not involve direct recruitment of human participants, patient-level data collection, or animal experimentation.

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