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INCIDENCE, CLINICAL PROFILE OF DIABETIC RETINOPATHY & ITS CORRELATION WITH GLYCEMIC CONTROL IN A TERTIARY CARE CENTER OF RAIGARH (CHHATTISGARH)

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ABSTRACT

Background: Diabetic retinopathy (DR) is a primary cause of avoidable blindness in the industrial regions of Chhattisgarh.

Aim: To analyze the 3-year incidence, demographic trends, and clinical profile of DR in the Raigarh area.

Methods: A hospital-based longitudinal observational study was conducted on 423 diabetic patients screened from 2023 to 2025. DR was graded using the International Clinical Diabetic Retinopathy (ICDR) scale.

Results: The overall prevalence of DR was 21.28%. High-risk glycemic levels (PPBS > 200 mg/dl and HbA1c > 8.5%) showed a strong correlation with advanced DR stages.

Conclusion: Systematic screening and lifestyle modification are essential to mitigate the burden of diabetic blindness in the district.

Keywords: Diabetic Retinopathy, Glycemic Control, Hba1c, Raigarh, Post-Prandial Blood Sugar.

INTRODUCTION

Diabetic retinopathy (DR) is a common microvascular complication of diabetes mellitus and a leading cause of preventable blindness, particularly in the working-age population. It results from chronic hyperglycemia-induced retinal microvascular damage, leading to ischemia, vascular leakage, and vision-threatening complications such as macular edema and proliferative diabetic retinopathy. The global burden of diabetes is increasing rapidly, and consequently, the incidence of DR is expected to rise significantly in the coming years due to aging populations, obesity, and sedentary lifestyles [3]. In India and other developing countries, the prevalence of DR among diabetic patients ranges from approximately 17% to 30%, highlighting its major public health importance [4,5].

The risk of DR increases with longer duration of diabetes and poor glycemic control, making metabolic regulation a key modifiable factor [6]. Glycated hemoglobin (HbA1c) is the most reliable marker of long-term glycemic control and has been strongly associated with both the presence and severity of DR. Numerous studies have demonstrated a clear dose-response relationship, where higher HbA1c levels correlate with increased risk and progression of retinopathy [1,7]. Patients with poorly controlled diabetes (HbA1c >8%) are significantly more likely to develop moderate to severe DR compared to those with better glycemic control [2,9]. Landmark evidence from major clinical trials such as the DCCT has established that tight glycemic control significantly reduces both the onset and progression of DR. The pathogenesis of DR is primarily driven by chronic hyperglycemia-induced mechanisms including oxidative stress, accumulation of advanced glycation end-products, endothelial dysfunction, and breakdown of the blood-retinal barrier, ultimately leading to capillary occlusion and retinal ischemia [8]. In addition to glycemia, systemic factors such as hypertension,



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dyslipidemia, anemia, and renal disease further worsen retinal damage and accelerate disease progression [9]. Recent evidence also suggests that glycemic variability, not just sustained hyperglycemia, contributes independently to microvascular complications, including DR [10]. In India, delayed diagnosis of diabetes and inadequate screening practices contribute to a higher proportion of patients presenting with established DR at diagnosis [6]. Lack of awareness regarding diabetic eye disease further delays intervention, increasing the risk of progression to advanced stages. DR remains a significant and growing public health challenge, with strong evidence supporting the role of strict glycemic control in reducing its incidence and severity [3–10].

MATERIALS AND METHODS

Study Design: It was a hospital-based, longitudinal observational study.

Place of Study: Department of Ophthalmology Late Shri Lakhiram Agrawal Memorial Government Medical College Raigarh (Chhattisgarh)

Period of Study: January 2023 to December 2025

Study Population: The study population comprised all diagnosed cases of diabetes mellitus (Type 1 and Type 2) attending the tertiary care center of Raigarh, Chhattisgarh during the study period 2023–2025 who met the inclusion criteria. It included adult patients (≥ 18 years) of both genders who underwent detailed ophthalmic evaluation for diabetic retinopathy, along with availability of glycemic control parameters such as fasting blood sugar (FBS), postprandial blood sugar (PPBS), and HbA1c. The population was assessed for clinical profile and categorized according to the severity of diabetic retinopathy to study its incidence and correlation with glycemic control.

Sample Size: 423 patients.

Inclusion Criteria:

- Patients diagnosed with Type 1 or Type 2 Diabetes Mellitus.
- Patients aged ≥ 18 years.
- Patients attending the ophthalmology or medicine outpatient/inpatient department of the tertiary care center.
- Patients willing to participate and provide informed consent.
- Patients undergoing retinal examination for evaluation of diabetic retinopathy.

Exclusion criteria:

- Patients with gestational diabetes mellitus.

- Patients with retinal diseases other than diabetic retinopathy (e.g., hypertensive retinopathy, retinal vein occlusion, age-related macular degeneration).
- Patients with a history of ocular trauma or previous retinal surgery.
- Patients with media opacities preventing proper fundus examination (dense cataract, corneal opacity, vitreous hemorrhage).
- Patients with severe systemic illness who were unable to undergo ophthalmic evaluation.

Study Variable: The study variables include demographic, clinical, ophthalmic, and biochemical parameters of diabetic patients attending the tertiary care center of Raigarh (Chhattisgarh) during 2023–2025. The independent variables comprise glycemic control indicators such as fasting blood sugar (FBS), 2-hour postprandial blood sugar (PPBS), and HbA1c levels, along with demographic factors like age, gender, and duration of diabetes. The dependent variable is the severity of diabetic retinopathy (No DR, Non-Proliferative DR, and Proliferative DR). The study analysed the distribution of clinical profile and DR severity and assessed the association between FBS, PPBS, and HbA1c (%) with different stages of diabetic retinopathy.

Statistical Analysis: For statistical analysis, data were initially entered into a Microsoft Excel spreadsheet and then analyzed using SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism (version 5). Numerical variables were summarized using means and standard deviations, while Data were entered into Excel and analyzed using SPSS and GraphPad Prism. Numerical variables were summarized using means and standard deviations, while categorical variables were described with counts and percentages. Two-sample t-tests were used to compare independent groups, while paired t-tests accounted for correlations in paired data. Chi-square tests (including Fisher's exact test for small sample sizes) were used for categorical data comparisons. P-values ≤ 0.05 were considered statistically significant.

RESULT

A total of 423 patients were analyzed over the 3-year study period. The 40–60 years age group represented the largest screened cohort at 56.6%. Notably, screenings for the younger age group (<40 years) rose from 8 in 2023 to 19 in 2025.

Table 1: Distribution of Clinical Profile and DR Severity (2023–2025)

Age In Years	2023 (N=132)	2024 (N=109)	2025 (N=182)	Total (N=423)
Age < 40 Years	8	6	19	33
Age 40–60 Years	80	77	135	292

Age > 60 Years	44	26	28	98
Male Gender	80	98	123	301
No DR	105	87	141	333
Mild NPDR	9	7	14	30
Moderate NPDR	13	11	16	40
Severe NPDR	1	2	6	9
PDR	4	2	5	11

Table 2: Association between Fasting Blood Sugar (FBS) and DR Severity

Fasting Blood Sugar	No Dr	Mild Npdr	Moderate Npdr	Severe Npdr	Pdr	Total
< 100 Mg/Dl	27	2	3	1	0	33 (7.8%)
100 - 125 Mg/Dl	79	7	10	1	1	98 (23.16%)
> 125 Mg/Dl	227	21	27	7	10	292 (69.03%)
Total	333 (78.72%)	30 (7.09%)	40 (9.45%)	9 (2.12%)	11 (2.6%)	423 (100%)

Table 3: Association between 2-Hour Post Prandial Sugar (PPBS) and DR Severity

2 Hour Post Prandial	No Dr	Mild Npdr	Moderate Npdr	Severe Npdr	Pdr	Total
< 140 Mg/Dl	13	1	1	0	0	15 (3.54%)
140 - 200 Mg/Dl	60	5	7	2	0	74 (17.49%)
> 200 Mg/Dl	260	24	32	7	11	334 (78.95%)
Total	333 (78.72%)	30 (7.09%)	40 (9.45%)	9 (2.12%)	11 (2.6%)	423 (100%)

Table 4: Association between HbA1c (%) and DR Severity

Hba1c (%)	No DR	Mild NPDR	Moderate NPDR	Severe NPDR	PDR	Total
< 6.4	3	1	1	0	0	5 (1.18%)
6.5 - 8.5	192	6	10	0	0	208 (49.17%)
8.5 - 10	67	18	16	7	9	117 (27.65%)
> 10	71	5	13	2	2	93 (21.98%)
Total	333 (78.72%)	30 (7.09%)	40 (9.45%)	9 (2.12%)	11 (2.6%)	423 (100%)

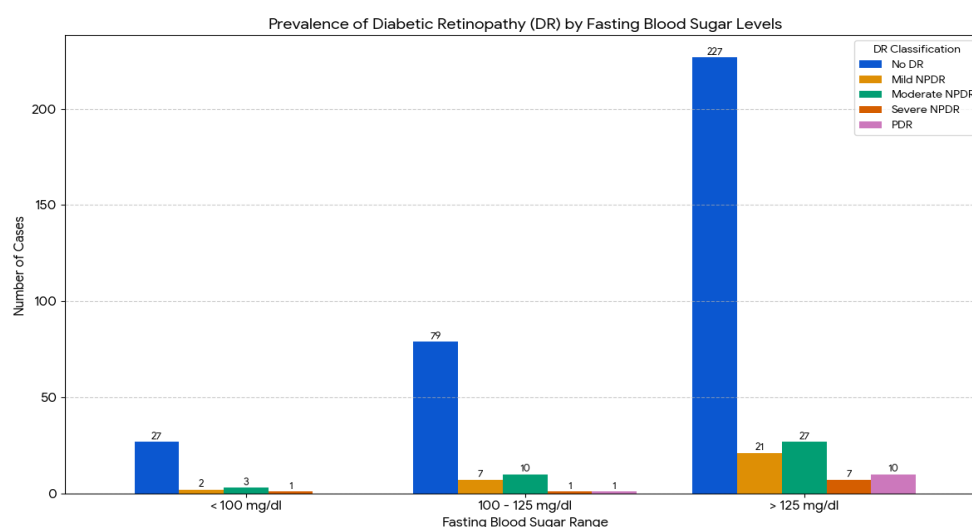


Figure 1: Association between Fasting Blood Sugar (FBS) and DR Severity

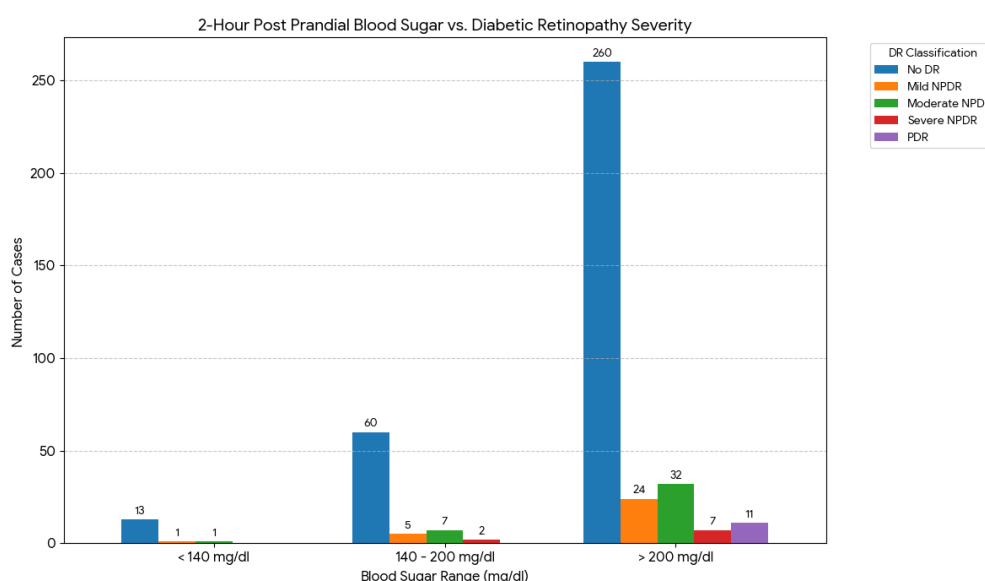


Figure 2: Association between 2-Hour Post Prandial Sugar (PPBS) and DR Severity

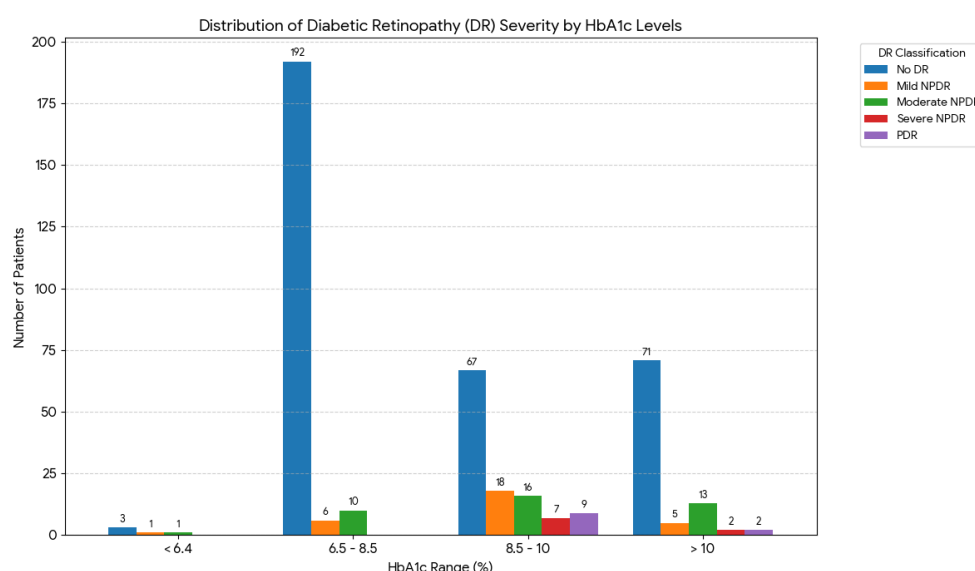


Figure 3: Association between HbA1c (%) and DR Severity

The study included a total distribution of patients across different demographic and clinical categories. In terms of age, the majority of patients belonged to the 40–60 years group with a total of 292 cases, followed by 98 cases in the >60 years group and 33 cases in the <40 years group. Male patients constituted a higher proportion with a total of 301 cases across data points. Regarding diabetic retinopathy status, the majority of patients had no diabetic retinopathy (n=333). Among those with diabetic retinopathy, mild NPDR was seen in 30 cases, moderate NPDR in 40 cases, severe NPDR in 9 cases, and proliferative diabetic retinopathy (PDR) in 11 cases, indicating that non-proliferative forms were more common than proliferative disease in the study population (Table 1). The distribution of

diabetic retinopathy according to fasting blood sugar levels showed that most patients had poor glycemic control. Out of 423 patients, the majority (292; 69.03%) had fasting blood sugar >125 mg/dl, among whom 227 had no diabetic retinopathy, while the remaining showed mild NPDR (21), moderate NPDR (27), severe NPDR (7), and PDR (10). Patients with fasting blood sugar 100–125 mg/dl accounted for 98 cases (23.16%), with most having no DR (79), followed by mild NPDR (7), moderate NPDR (10), severe NPDR (1), and PDR (1). Only 33 patients (7.8%) had fasting blood sugar <100 mg/dl, of which 27 had no DR and a very small proportion had mild (2), moderate (3), and severe NPDR (1), with no PDR cases. Overall, diabetic retinopathy severity increased with worsening

fasting blood sugar levels, indicating a clear association between poor glycemic control and progression of diabetic retinopathy (Table 2). The distribution of diabetic retinopathy according to 2-hour postprandial blood sugar levels showed a strong association between higher glucose levels and severity of retinopathy. Out of 423 patients, the majority (334; 78.95%) had postprandial blood sugar >200 mg/dl, among whom 260 had no diabetic retinopathy, while mild NPDR was seen in 24, moderate NPDR in 32, severe NPDR in 7, and PDR in 11 cases. Patients with postprandial blood sugar 140–200 mg/dl accounted for 74 cases (17.49%), with 60 having no DR, 5 mild NPDR, 7 moderate NPDR, and 2 severe NPDR, while no PDR cases were observed in this group. Only 15 patients (3.54%) had postprandial blood sugar <140 mg/dl, of which 13 had no DR, 1 had mild NPDR, and 1 had moderate NPDR, with no severe NPDR or PDR cases. Overall, increasing postprandial blood sugar levels were associated with higher severity of diabetic retinopathy, demonstrating a clear correlation between poor glycemic control and disease progression (Table 3). The distribution of diabetic retinopathy according to HbA1c levels demonstrated a strong correlation between poor glycemic control and severity of retinopathy. Out of 423 patients, the majority (208; 49.17%) had HbA1c levels between 6.5–8.5%, among whom 192 had no diabetic retinopathy, while mild NPDR was seen in 6 cases and moderate NPDR in 10 cases. Patients with HbA1c 8.5–10% accounted for 117 cases (27.65%), with 67 having no DR, 18 mild NPDR, 16 moderate NPDR, 7 severe NPDR, and 9 PDR cases, showing increased severity with worsening glycemic control. Similarly, 93 patients (21.98%) had HbA1c >10%, among whom 71 had no DR, while moderate NPDR (13), mild NPDR (5), severe NPDR (2), and PDR (2) were also observed. Only 5 patients (1.18%) had HbA1c <6.4%, with minimal retinopathy changes. Overall, increasing HbA1c levels were associated with higher grades of diabetic retinopathy, indicating a clear dose–response relationship between poor long-term glycemic control and progression of retinopathy (Table 4).

DISCUSSION

The present study demonstrates a strong and consistent association between glycemic control and the severity of diabetic retinopathy (DR). The majority of patients belonged to the 40–60 years age group, accounting for 292 cases, followed by 98 cases in the >60 years group and 33 cases in the <40 years group. Male patients constituted a higher proportion with a total of 301 cases, reflecting similar demographic patterns reported in previous epidemiological studies Duh EJ et al. [11]. A higher male predominance has also been attributed to

increased healthcare utilization patterns and occupational risk exposure Sabanayagam C et al. [12].

In our study population, the majority of patients had no diabetic retinopathy (333 cases; 78.72%), while non-proliferative diabetic retinopathy (NPDR) constituted the most common form among affected individuals, including mild NPDR in 30 cases (7.09%), moderate NPDR in 40 cases (9.45%), severe NPDR in 9 cases (2.12%), and proliferative diabetic retinopathy (PDR) in 11 cases (2.6%), indicating that non-proliferative forms were more prevalent than proliferative disease. This pattern is consistent with global data showing NPDR as the earliest and most frequently observed stage of DR Cheung N et al. [13]. The relatively lower proportion of PDR suggests early detection in a tertiary care setting Ting DS et al. [14].

The distribution of diabetic retinopathy according to fasting blood sugar levels showed that most patients had poor glycemic control. Out of 423 patients, the majority (292; 69.03%) had fasting blood sugar >125 mg/dl, among whom 227 had no diabetic retinopathy (53.65%), while mild NPDR was seen in 21 (4.96%), moderate NPDR in 27 (6.38%), severe NPDR in 7 (1.65%), and PDR in 10 (2.36%). Patients with fasting blood sugar 100–125 mg/dl accounted for 98 cases (23.16%), with 79 (18.67%) having no DR, followed by mild NPDR (7; 1.65%), moderate NPDR (10; 2.36%), severe NPDR (1; 0.23%), and PDR (1; 0.23%). Only 33 patients (7.8%) had fasting blood sugar <100 mg/dl, of which 27 (6.38%) had no DR and a very small proportion had mild (2; 0.47%), moderate (3; 0.71%), and severe NPDR (1; 0.23%), with no PDR cases. Overall, diabetic retinopathy severity increased with worsening fasting blood sugar levels, indicating a clear association between poor glycemic control and progression of diabetic retinopathy Yau JW et al. [15].

The distribution of diabetic retinopathy according to 2-hour postprandial blood sugar levels showed a strong association between higher glucose levels and severity of retinopathy. Out of 423 patients, the majority (334; 78.95%) had postprandial blood sugar >200 mg/dl, among whom 260 (61.46%) had no diabetic retinopathy, while mild NPDR was seen in 24 (5.67%), moderate NPDR in 32 (7.57%), severe NPDR in 7 (1.65%), and PDR in 11 (2.6%) cases. Patients with postprandial blood sugar 140–200 mg/dl accounted for 74 cases (17.49%), with 60 (14.18%) having no DR, 5 (1.18%) mild NPDR, 7 (1.65%) moderate NPDR, and 2 (0.47%) severe NPDR, while no PDR cases were observed in this group. Only 15 patients (3.54%) had postprandial blood sugar <140 mg/dl, of which 13 (3.07%) had no DR, 1 (0.23%) had mild NPDR, and 1 (0.23%) had moderate NPDR, with no severe NPDR or PDR

cases. Overall, increasing postprandial blood sugar levels were associated with higher severity of diabetic retinopathy Monnier L et al. [17].

The distribution of diabetic retinopathy according to HbA1c levels demonstrated a strong correlation between poor glycemic control and severity of retinopathy. Out of 423 patients, the majority (208; 49.17%) had HbA1c levels between 6.5–8.5%, among whom 192 (45.39%) had no diabetic retinopathy, while mild NPDR was seen in 6 (1.42%) cases and moderate NPDR in 10 (2.36%). Patients with HbA1c 8.5–10% accounted for 117 cases (27.65%), with 67 (15.84%) having no DR, 18 (4.25%) mild NPDR, 16 (3.78%) moderate NPDR, 7 (1.65%) severe NPDR, and 9 (2.12%) PDR cases. Similarly, 93 patients (21.98%) had HbA1c >10%, among whom 71 (16.78%) had no DR, while moderate NPDR (13; 3.07%), mild NPDR (5; 1.18%), severe NPDR (2; 0.47%), and PDR (2; 0.47%) were also observed. Only 5 patients (1.18%) had HbA1c <6.4%, with minimal retinopathy changes. Overall, increasing HbA1c levels were associated with higher grades of diabetic retinopathy, indicating a clear dose–response relationship between poor long-term glycemic control and progression of retinopathy Brownlee M [19], Fong DS et al. [20].

CONCLUSION

The present study demonstrated that diabetic retinopathy is strongly associated with poor glycemic control and shows a clear dose–response relationship with fasting blood sugar, postprandial blood sugar, and HbA1c levels. Most patients belonged to the 40–60 years age group, and non-proliferative diabetic retinopathy was the most common clinical presentation. The severity of retinopathy increased significantly with worsening glycemic parameters, particularly in patients with elevated HbA1c levels. These findings highlight that HbA1c is the most reliable predictor of diabetic retinopathy progression. Early screening, strict glycemic control, and regular ophthalmic evaluation are essential to prevent vision-threatening complications in diabetic patients in the Raigarh region.

REFERENCES

1. Zhang B, Zhang B, Zhou Z, Guo Y, Wang D. The value of glycosylated hemoglobin in the diagnosis of diabetic retinopathy: a systematic review and Meta-analysis. *BMC Endocrine Disorders*. 2021 Apr 26;21(1):82.
2. Favas KM, Niveditha M, Yoosuf BT, Bhukya M, Gupta PC, Dutta P, Bansal D. Insights into the systemic risk factors associated with diabetic retinopathy in the Indian population: A comprehensive systematic review and meta-analysis. *Indian Journal of Ophthalmology*. 2025 Jan 1;73(Suppl 1):S24-30.
3. Nouri H, Abtahi SH, Mazloumi M, Samadikhadem S, Arevalo JF, Ahmadi H. Optical coherence tomography angiography in diabetic retinopathy: A major review. *Survey of ophthalmology*. 2024 Jul 1;69(4):558-74.
4. Saxena R, Singh D, Saklani R, Gupta SK. Clinical biomarkers and molecular basis for optimized treatment of diabetic retinopathy: current status and future prospects. *Eye and brain*. 2016 Feb 19:1-3.
5. Rajeshwari M, Patil K, Shruti B, Sagar H, Supriya C, Kallur R, Shivalee A. A cross sectional clinical study to evaluate the correlation between HbA1c levels and grades of diabetic retinopathy in diabetic patients at tertiary care hospital. *European Journal of Cardiovascular Medicine*. 2025 Dec 22;15:342-6.
6. Lee R, Wong TY, Sabanayagam C. Epidemiology of diabetic retinopathy, diabetic macular edema and related vision loss. *Eye and vision*. 2015 Sep 30;2(1):17.
7. Mondal R, Sengupta D, Dutta T, et al. A systematic review and meta-analysis of genetic variants and covariates associated with diabetic retinopathy in the Indian population. *Discov Med*. 2025.
8. Favas KT, et al. Systemic risk factors for diabetic retinopathy in India: meta-analysis. *Indian J Ophthalmol*. 2025.
9. Lakshminarayanan V, et al. Automated detection and diagnosis of diabetic retinopathy: a comprehensive survey. *arXiv preprint arXiv:2107.00115*. 2021.
10. Dey S, et al. Managing diabetic retinopathy with deep learning: a data-centric overview. *arXiv preprint arXiv:2604.02448*. 2026.
11. Duh EJ, Sun JK, Stitt AW. Diabetic retinopathy: current understanding, mechanisms, and treatment strategies. *JCI insight*. 2017 Jul 20;2(14):e93751.
12. Sabanayagam C, Banu R, Chee ML, Lee R, Wang YX, Tan G, Jonas JB, Lamoureux EL, Cheng CY, Klein BE, Mitchell P. Incidence and progression of diabetic retinopathy: a systematic review. *The lancet Diabetes & endocrinology*. 2019 Feb 1;7(2):140-9.
13. Cheung N, Wong IY, Wong TY. Ocular anti-VEGF therapy for diabetic retinopathy: overview of clinical efficacy and evolving applications. *Diabetes care*. 2014 Apr 1;37(4):900-5.
14. Ting DS, Cheung GC, Wong TY. Diabetic retinopathy: global prevalence, major risk

- factors, screening practices and public health challenges: a review. *Clinical & experimental ophthalmology*. 2016 May;44(4):260-77.
15. Yau JW, Rogers SL, Kawasaki R, Lamoureux EL, Kowalski JW, Bek T, Chen SJ, Dekker JM, Fletcher A, Grauslund J, Haffner S. Global prevalence and major risk factors of diabetic retinopathy. *Diabetes care*. 2012 Mar 1;35(3):556-64.
 16. Ceriello A. Postprandial hyperglycemia and diabetes complications: is it time to treat?. *Diabetes*. 2005 Jan 1;54(1):1-7.
 17. Monnier L, Colette C, Wojtusciszyn A, Dejager S, Renard E, Molinari N, Owens DR. Toward defining the threshold between low and high glucose variability in diabetes. *Diabetes care*. 2017 Jul 1;40(7):832-8.
 18. Klein BE, Myers CE, Howard KP, Klein R. Serum lipids and proliferative diabetic retinopathy and macular edema in persons with long-term type 1 diabetes mellitus: the Wisconsin epidemiologic study of diabetic retinopathy. *JAMA ophthalmology*. 2015 May 1;133(5):503-10.
 19. Brownlee M. Biochemistry and molecular cell biology of diabetic complications. *Nature*. 2001 Dec 13;414(6865):813-20.
 20. Fong DS, Aiello LP, Ferris III FL, Klein R. Diabetic retinopathy. *Diabetes care*. 2004 Oct 1;27(10).

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