

Association of Erythropoietin Gene rs1617640 Polymorphism with Diabetic Retinopathy in North Indian Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Background: Diabetic retinopathy (DR) represents a critical microvascular consequence of Type 2 diabetes mellitus (T2DM) and remains a primary etiology for avoidable vision loss. This investigation explored how the rs1617640 variant within the erythropoietin (EPO) gene correlates with DR susceptibility among a North Indian cohort.

Methods: Within a hospital-centered, case-control framework, we recruited 160 individuals diagnosed with T2DM. This population comprised 80 cohorts presenting with DR along with an equivalent control group of 80 diabetic subjects free of retinal complications. Participants underwent comprehensive clinical, ophthalmic, metabolic, and molecular evaluations, with genotyping achieved via PCR-RFLP methodologies.

Results: Individuals presenting with DR exhibited markedly elevated HbA1c ($10.17 \pm 1.98\%$ vs. $8.14 \pm 1.78\%$; $p < 0.001$) alongside higher blood pressure readings, BMI metrics, and circulating glucose levels relative to the control arm. The G allele and corresponding GG homozygous genotype demonstrated significantly higher frequencies in the DR cohort (22.5% vs. 12.5%, OR = 2.64, $p = 0.029$; and 43.8% vs. 30.6%, OR = 1.78, $p = 0.017$, respectively). Furthermore, sight-threatening manifestations of DR clustered preferentially among those carrying the GG genotype.

Conclusion: The rs1617640 polymorphism of the EPO gene is strongly tied to DR presentation. Nonetheless, multivariate modelling established that diminished glycemic control (reflected by heightened HbA1c) stands as the primary autonomous risk factor for advanced DR, confirming that sustained hyperglycemia exerts a more profound impact than isolated genetic predisposition.

KEYWORDS: Diabetic retinopathy, Type 2 diabetes mellitus, Erythropoietin, rs1617640 polymorphism, HbA1c, Gene.

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INTRODUCTION

Diabetes mellitus (DM) contributes to a significant proportion of the global disease burden due to its insidious nature and chronic hyperglycemia resulting from a complex interplay of factors. Of the microvascular complications, diabetic retinopathy (DR) is a leading cause of global preventable blindness in the working adult population.¹ Current classification of DR is either the non-proliferative (NPDR) type or the proliferative (PDR) type. Extreme stages of the latter type encompass what is considered vision-threatening diabetic retinopathy (VTDR) including special cases of severe NPDR, PDR.^{2,3}

Multi-factorial nature of chronic hyperglycemia gives rise to a myriad of complex DR pathologies and includes oxidative stress, adverse inflammatory responses, and pathological angiogenesis among others.⁴⁻⁶ Newer theories based on clinical observation suggest that DR may not be simply a micro-angiopathic condition but more a neuro-degenerative condition affecting the vascular system as well.⁷ This has led to newer focus on neuro-protection and the vascular pathway as areas of potential research interest. Erythropoietin (EPO) is a member of this potential research

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interest.^{8,9} It is a glycoprotein that, among other effects, has been shown to protect and maintain the integrity of the retinal vasculature in the condition of diabetic retinopathy in experimental animal studies.¹⁰⁻¹² This has led to elevated concerns of both neuroprotection and pathological angiogenesis and the EPO in the retinopathy of diabetes.¹³⁻¹⁴

Human EPO gene consists of several SNPs, such as rs1617640, rs507392, and rs551238, and is located at 7q22.1 on chromosome 7. These SNPs may be associated with EPO expression and with probable susceptibility to diabetic microvascular complications.¹⁵ Several studies showed that the EPO gene SNPs and the diabetic retinopathy

polymorphisms were associations that produced varied results and were inconsistent for different ethnic groups.^{16–20} Many of those studies discussed the rs1617640 G allele and its association as a risk factor for DR, while some studies argued that the T allele was the risk factor, clearly suggesting the ethno-genetic diversity.^{16–20}

India is known to have one of the largest diabetic population segments in the world. However, studies of associations of EPO genetic polymorphisms and diabetic retinopathy in the Indian population, particularly North Indian population, are sparse. For this reason, this study aims to evaluate potential associations of erythropoietin gene polymorphism and diabetic retinopathy in T2DM in Northern part of India as well as measure the potential relatives of diabetic retinopathy as well as erythropoietin gene polymorphism with the severity of the disease.

MATERIAL AND METHODS

Participants and Study Design

From 2023 to 2026, this case and control study was conducted on patients in the Department of Ophthalmology, ELMC&H, Lucknow and in the Department of Personalized and Molecular Medicine, Era University. One hundred sixty patients with T2DM, 80 of whom had DR and the other 80 had no retinopathy (who were the control group) were recruited into the study. The study was approved by the Institutional Ethics Committee (IEC), ELMC&H (Ref. No. ELMC&H/R-Cell/2024/3266; IEC Registration No. ECR/717/Inst./U.P./2015/RR-21). Using power of 90% based on rates of the rs1617640 reported in literature the sample size was calculated.²⁰ Inclusion criteria consisted of a diagnosis of T2DM and the ability to provide written informed consent. Exclusion criteria involved hazy ocular media preventing a fundus examination as well as end-stage renal illness.

Clinical and Biochemical Evaluation

Patient evaluation commenced with a thorough documentation of diabetic history, covering baseline clinical characteristics such as disease duration, ancestral diabetic lineage, prior treatments, and active ocular symptoms. Anthropometric metrics including blood pressure. Following this initial screening, comprehensive ophthalmic profiling was conducted using best-corrected visual acuity scales. Progression of DR was cataloged via the Early Treatment Diabetic Retinopathy Study guidelines, specifically monitoring for indicators of vision-threatening diabetic retinopathy (VTDR)—categorized here by advanced non-proliferative or proliferative changes, alongside clinically significant macular edema. Optical coherence tomography (OCT) and fundus imaging supplemented these examinations based on individual clinical indications. Simultaneously, metabolic status was mapped through sterile venous blood collections. These samples were processed using standard laboratory

techniques to quantify fasting and postprandial glucose concentrations, along with glycated hemoglobin (HbA1c) percentages for all study participants.

DNA Extraction and Genotyping

For the genetic study, a volume of 2 mL peripheral venous blood was collected in EDTA vials and preserved at -80 degrees centigrade for future analysis. Extraction of Genomic DNA was from blood sample using procedure with magnesium chloride (MgCl₂). The quality and purity of the DNA were assessed using spectrophotometry. The integrity and concentration of the DNA were assessed by 2% agarose gel electrophoresis which was stained with ethidium bromide.

The erythropoietin (EPO) gene rs1617640 polymorphism was amplified by means of the polymerase chain reaction (PCR) process. In this case, peculiar EPO gene polymorphic region primers were used. The amplification was performed on a thermal cycler. Genotype determination was performed by RFLP method. The restriction enzyme used was MnlI. The distinct genotypic patterns were studied using the agarose gel electrophoresis method.

RESULT OF OUR STUDY

T2DM were enrolled in the study, 80 patients with DR (Group A) and 80 diabetic patients without DR (Group B). Comparative analysis of demographic, metabolic, ocular, and genetic parameters was performed between the two groups.

Table 1: The mean age was (54.11 ± 9.65 vs. 54.91 ± 13.52 years; $p = 0.667$). However, anthropometric analysis demonstrated significantly higher mean body weight (73.61 ± 10.03 vs 65.60 ± 11.28 kg; $p < 0.001$) and BMI (26.68 ± 2.86 vs 24.66 ± 2.17 kg/m²; $p < 0.001$) in Group A compared to Group B.

Table 2: Compared to controls, patients with diabetic retinopathy had poorer metabolic and blood pressure profiles. Group A's (patients with retinopathy) mean fasting blood sugar levels were significantly higher (214.26 ± 51.95 vs 174.40 ± 41.60 mg/dL; $p < 0.001$). Group A's (retinopathy patients) postprandial blood sugar levels were also significantly higher (345.71 ± 72.58 vs 277.86 ± 67.08 mg/dL; $p < 0.001$). HbA1c levels for retinopathy patients were also significantly higher ($10.17 \pm 1.98\%$ vs $8.14 \pm 1.78\%$; $p < 0.001$). DR Patients have higher systolic and diastolic blood pressure (149.95 ± 11.30 vs 132.75 ± 11.38 mmHg and 93.73 ± 8.70 vs 82.43 ± 8.78 mmHg, $p < 0.001$ for both, respectively).

Table 3: Poor glycemic control (HbA1c $\geq 7\%$) was significantly more prevalent in Group A compared to Group B [78 (97.5%) vs 50 (62.5%); $p < 0.001$]. In contrast, good glycemic control (HbA1c $< 7\%$) was observed predominantly among controls [30 (37.5%) vs 2 (2.5%)].

Table 4: Mean presenting visual acuity was significantly poorer in Group A (0.83 ± 0.48 vs 0.54 ± 0.24 logMAR; $p < 0.001$), and mean BCVA was also significantly reduced (0.71 ± 0.53 vs 0.18 ± 0.23 logMAR; $p < 0.001$). Intraocular pressure was comparable between the groups ($p = 0.496$). According to WHO visual impairment classification, normal or mild visual impairment was observed in 50.63% eyes in Group A associated to 95.63% eyes in Group B. Blindness observed in 25 eyes (15.60%) compared to only 1 eye (0.63%) in Group B.

Table 5A: Among eyes with diabetic retinopathy, mild NPDR was the most common stage, observed in 53 eyes (33.12%), followed by moderate NPDR in 44 eyes (27.50%). Severe NPDR and proliferative diabetic retinopathy were observed in 25 eyes (15.62%) and 27 eyes (16.88%), respectively, while advanced diabetic eye disease was present in 11 eyes (6.88%).

Table 5B: Non-sight-threatening diabetic retinopathy (Non-STDR) accounted for 97 eyes (60.62%), whereas sight-threatening diabetic retinopathy (STDR) was present in 63 eyes (39.38%).

Table 5C: Diabetic maculopathy was observed in 44 eyes (27.5%), while it was absent in 116 eyes (72.5%).

Table 6: Analysis of EPO rs1617640 polymorphism demonstrated that the TT genotype was more common among controls [41 (51.2%)] compared to cases [28 (35.0%)]. However, the GG genotype was significantly more frequent among patients with diabetic retinopathy [18 (22.5%) vs 10 (12.5%); OR = 2.64, 95% CI: 1.10–6.32; $p = 0.029$]. Allelic analysis revealed significantly higher frequency of the G allele in Group A compared to Group B, G allele shows and increased susceptibility to diabetic retinopathy.

DISCUSSION

This study investigated the effect of the rs1617640 polymorphism in the EPO gene on DR and highlighted important clinical, metabolic, ocular and genomic distinctions of T2DM with and without DR. There remained no significant distinctions in demographic. In agreement with the literature on the Pakistani, Ethiopian, Cameroonian and Nepalese populations, the results suggest that sex may not be an independent risk factor.^{22–26} The mean age of participants in both groups was approximately 54 years, and diabetic retinopathy is known to affect primarily middle aged and elderly individuals of almost all the existing ethnic populations.^{22,24–28}

Patients with DR had suggestively higher BMI associated to the control group, indicating that retinopathy patients are mostly overweight. Similar associations between obesity and diabetic retinopathy were reported in the Ethiopian population, where being overweight and obesity were shown to considerably increase the risk of diabetic retinopathy.^{23,26} With respect to the remaining metabolic parameters, FBS, PPS, also HbA1c levels shows significantly higher in the DR group and were in agreement with the indicated studies from Nepal, Cameroon and Egypt where poor glycemic control was shown to be a significant factor in the advancement of DR.^{24,29,30} The majority of retinopathy patients (97.5%) had poor glycemic control ($\text{HbA1c} \geq 7\%$), indicating the significance of chronic hyperglycemia in the development of retinal microvascular damage. Retinopathy patients also had significantly higher blood pressure, and it was indicated in the cited studies that hypertension and poor glycemic control were significant contributing factors to diabetic retinopathy.²⁴

Table 1: Demographic as well as Anthropometric Characteristics of Study Participants (n = 160).

Parameter	Group A (n=80)	Group B (n=80)	Statistical Value	p-value
Male, n (%)	45 (56.25)	40 (50.00)	—	—
Female, n (%)	35 (43.75)	40 (50.00)	—	—
Age (years)	54.11 ± 9.65	54.91 ± 13.52	$t = -0.43$	0.667
Weight (kg)	73.61 ± 10.03	65.60 ± 11.28	$t = 4.75$	<0.001
BMI (kg/m^2)	26.68 ± 2.86	24.66 ± 2.17	$t = 5.04$	<0.001
Height (m)	1.69 ± 0.10	1.65 ± 0.13	$t = 2.17$	0.031

Table 2: Assessment of Metabolic and Clinical Parameters among different Groups (n = 160).

Parameter	Group A (Mean $\pm$ SD)	Group B (Mean $\pm$ SD)	t-value	p-value
Fasting Blood Sugar (mg/dL) (FBS)	214.26 ± 51.95	174.40 ± 41.60	5.36	<0.001
Postprandial Blood Sugar (mg/dL) (PPS)	345.71 ± 72.58	277.86 ± 67.08	6.14	<0.001
HbA1c (%)	10.17 ± 1.98	8.14 ± 1.78	6.85	<0.001
Systolic BP (mmHg)	149.95 ± 11.30	132.75 ± 11.38	9.59	<0.001
Diastolic BP (mmHg)	93.73 ± 8.70	82.43 ± 8.78	8.18	<0.001

Table 3: Distribution According to HbA1c Categories and Duration of Diabetes (n = 160).

Parameter	Group A n (%)	Group B n (%)	Statistical Value	p-value
HbA1c <7%	2 (2.5)	30 (37.5)	$\chi^2 = 28.48$	<0.001
HbA1c $\geq$ 7%	78 (97.5)	50 (62.5)	-	-
Duration <10 years	66 (82.5)	62 (77.5)	$\chi^2 = 0.625$	0.429
Duration >10 years	14 (17.5)	18 (22.5)	-	-

Table 4: Ocular Characteristics and Visual Impairment Profile (n = 320 eyes).

Parameter	Group A	Group B	Statistical Value	p-value
Presenting VA (logMAR)	0.83 $\pm$ 0.48	0.54 $\pm$ 0.24	t = 6.68	<0.001
BCVA (logMAR)	0.71 $\pm$ 0.53	0.18 $\pm$ 0.23	t = 11.82	<0.001
IOP (mmHg)	18.88 $\pm$ 2.85	19.08 $\pm$ 2.56	t = -0.68	0.496
Normal/Mild visual impairment	81 (50.63%)	153 (95.63%)	—	—
Moderate visual impairment	49 (30.63%)	6 (3.78%)	—	—
Severe visual impairment	5 (3.1%)	0 (0%)	—	—
Blindness	25 (15.60%)	1 (0.63%)	—	—

Table 5A: ETDRS Classification and Severity of Diabetic Retinopathy in Group A (n = 160 eyes).

Parameter	Number of Eyes	Percentage (%)
Mild NPDR	53	33.12
Moderate NPDR	44	27.50
Severe NPDR	25	15.62
Proliferative DR	27	16.88
Advanced Diabetic Eye Disease	11	6.88

Table 5B: Distribution of STDR and Non-STDR in Group A (n = 160 eyes).

Parameter	Number of Eyes	Percentage (%)
Non-STDR	97	60.62
STDR	63	39.38

Table 5C: Distribution of Diabetic Maculopathy in Group A (n = 160 eyes).

Parameter	Number of Eyes	Percentage (%)
Maculopathy Present	44	27.5
Maculopathy Absent	116	72.5

Table 6: Genotype and Allele Frequency Distribution with Odds Ratio Analysis.

Variable	Category	Group A n (%)	Group B n (%)	OR (95% CI)	p-value
Genotype	TT (Reference)	28 (35.0)	41 (51.2)	1.00	—
	GT	34 (42.5)	29 (36.3)	1.71 (0.88–3.32)	0.11
	GG	18 (22.5)	10 (12.5)	2.64 (1.10–6.32)	0.029
Allele	T (Reference)	90 (56.2)	111 (69.4)	1.00	—
	G	70 (43.8)	49 (30.6)	1.78 (1.11–2.86)	0.017

Patients with diabetic retinopathy had the most severe visual impairment. Compared to the findings of other studies, the retinopathy group had the most cases of moderate to severe visual impairment and blindness.^{22,24,31} DR is a disease that affects the retina but not the pressure controlling system of the eye. According to the ETDRS classification, the majority of retinopathy cases were mild, moderate, or severe NPDR, and proliferative diabetic retinopathy was also present.

Similar findings have been reported in the Pakistani, Ethiopian, Nepalese, and Chinese populations, indicating that the early non-proliferative stage of diabetic retinopathy dominates the world.^{22,25,26,32} However, almost 40% of eyes in this study had sight threatening diabetic retinopathy (STDR). Diabetic maculopathy was also present in 27.5% of eyes and was reported to be one of the leading reasons for central visual impairment in T2DM patients.^{22,25,27,30}

The major outcome of this study was identifying the EPO rs1617640 as a genetic risk reason for developing DR. Compared to controls, retinopathy patients had a 2.64-fold risk of the EPO rs1617640 polymorphism in the GG genotype. These findings provide support for Mankoč Ramuš *et al.* study as well as Abhary *et al.*, who found EPO polymorphisms and danger for proliferative DR in the Slovenian and Australian populations, respectively.^{13,21} Erythropoietin is known to be a multifunctional cytokine that may have a role in retinal neuroprotection and ischemia, and may be involved in angiogenesis and remodeling.⁸⁻¹⁴ It is possible that polymorphisms of the EPO gene that affect gene expression may confer risk for developing diabetic retinopathy. These results, in combination with the Brazilian and Chinese studies, may suggest that the EPO polymorphism may have a protective role for retinopathy in some populations and is influenced by the environment.^{17,33}

The genotype distribution and severity of retinopathy were correlated. The GG genotype was most commonly identified in patients with sight-threatening DR, and the findings support the association of severe forms of proliferative retinopathy with EPO polymorphisms.^{13,21} The retinopathy to the combination of metabolic and genetic factors is complex. This variability is reported in other studies as well.^{13,26,29}

DR from Northern India, the EPO rs1617640 may act as a genetic susceptibility marker as there is a strong association of DR with the GG genotype and G allele. In the direction to understand the effects of EPO polymorphisms on the progression of diabetic retinal disease and to confirm our findings, we suggest that future studies focus on the functional aspects of these polymorphisms and be large, multicentric, and comprehensive in scope.

CONCLUSION

The current research study found that in North Indian Type 2 Diabetes patients, the EPO gene rs1617640 polymorphism was strongly linked to DR. The GG genotype, and G allele predicted a greater risk of developing DR and a greater severity of DR. In this study, multivariate analysis indicated that poor glycemic control (high HbA1c) had the greatest risk, and diabetic retinopathy was best termed a multifactorial disorder of metabolism with chronic hyperglycemia taking precedence over genetic susceptibility in the pathogenesis of the disorder. From these results, we could accept that genetic polymorphism of EPO contributes to the susceptibility to the disorder, but we could conclude that improving glycemic control is the best approach to delay, or preventing the progression of DR.

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