

Contrast Sensitivity Across Different Grades of Non-Proliferative Diabetic Retinopathy: A Cross-Sectional Study from Peshawar, Pakistan

PJO – Official Journal of
Ophthalmological Society of Pakistan



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ABSTRACT

Purpose: The purpose of this study was to assess contrast sensitivity (CS) across the spectrum of Non-Proliferative Diabetic Retinopathy (NPDR) severity and explore clinical and demographic variables associated with it.

Study Design: Cross-sectional study.

Place and Duration of Study: Department of Ophthalmology, Hayatabad Medical Complex, Peshawar from October 2020 to November 2021.

Methods: We enrolled 245 patients with mild, moderate, or severe NPDR, analyzing one eye per participant. Contrast sensitivity was assessed using Pelli-Robson chart, alongside a range of clinical and ocular variables including diabetic macular oedema (DME), visual acuity, HbA1c, and duration of diabetes. Differences across NPDR grades were examined using ANOVA and Kruskal-Wallis tests, while correlation analysis, multivariable linear regression, and sensitivity analyses were used to further explore the data.

Results: Mean CS showed a declining trend across mild, moderate, and severe NPDR (1.26 ± 0.38 , 1.23 ± 0.40 , and 1.13 ± 0.39 log units, respectively), although the difference was not statistically significant ($p=0.175$). CS was not significantly correlated with NPDR grade. In multivariable analysis, neither DME ($\beta=-0.12$, $p=0.082$) nor NPDR grade ($\beta=-0.076$, $p=0.526$) was independently associated with CS. Excluding eyes with DME did not materially alter the findings.

Conclusion: CS showed a modest decline with increasing NPDR severity but was not independently associated with disease severity after adjustment for relevant clinical factors. It may provide complementary functional information but should not be considered an independent marker of NPDR severity.

Keywords: Contrast Sensitivity, Diabetic Retinopathy, Diabetic Macular Oedema, Pelli-Robson, Pakistan.

How to Cite this Article: Jehan R, Ramzan F, Bibi H, Ahmed H. Contrast Sensitivity Across Different Grades of Non-Proliferative Diabetic Retinopathy: A Cross-Sectional Study from Peshawar, Pakistan. 2026;42(4):460-466. Doi: 10.36351/pjo.v42i4.2577

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Received: July 15, 2026
Revised: August 25, 2026
Accepted: September 5, 2026

INTRODUCTION

Diabetic retinopathy (DR) remains one of the leading

causes of preventable blindness among working-age adults worldwide. A global meta-analysis of population-based studies estimated that roughly one in three people with diabetes has some degree of DR, and this burden is only expected to grow as diabetes prevalence continues to rise across low- and middle-income countries.¹ Pakistan reflects this trend starkly as shown by the second National Diabetes Survey (2016–2017), which reported a national diabetes prevalence of 26.3%.² DR has been documented in Pakistani populations, including a community-based

cohort in Karachi reporting substantial retinopathy prevalence among patients with type 2 diabetes³ and community-based ophthalmic screening programs in Khyber Pakhtunkhwa and other provinces.^{3,4} These figures underscore the scale of the problem and the need for accessible functional markers of disease severity beyond routine fundus photography.

DR has traditionally been conceptualized as a microvascular disease, but a growing body of evidence supports the concept of the retina as a neurovascular unit, in which neurodegeneration including retinal ganglion cell apoptosis, thinning of the inner retinal layers, and disruption of neuroglial signaling occurs early in the disease course and may precede clinically detectable microvascular lesions.⁵ Because CS depends on the integrity of this inner neuroretinal circuitry rather than solely on foveal photoreceptor resolution, it has been proposed as a more sensitive functional marker of early diabetic retinal dysfunction than visual acuity, which typically remains preserved until relatively late in the disease process.⁶ This premise has motivated a substantial literature examining CS in diabetic populations.

Sokol et al, first demonstrated that diabetic patients with background retinopathy had CS abnormalities across a wider range of spatial frequencies than diabetic patients without retinopathy.⁶ Subsequent work, including a large population-based analysis from the Sankara Nethralaya Diabetic Retinopathy Epidemiology and Molecular Genetics Study (SN-DREAMS-II) and a comparative study of 368 eyes across disease-free, diabetic-without-retinopathy, and NPDR groups, similarly reported reduced CS in association with DR.^{7,8} A systematic review of 21 comparative cross-sectional studies concluded that CS loss was a near-universal finding in diabetic populations with DR.⁹ More recently, structure-function studies using widefield swept-source optical coherence tomography angiography have linked CS to quantitative retinal vascular metrics across the NPDR-to-proliferative DR spectrum.¹⁰ A recent review of structural and functional biomarkers for early DR detection similarly highlighted CS as one of several functional tests capable of detecting deficits before overt vascular lesions appear.¹¹

Despite this substantial literature, three important gaps remain. First, most existing studies compare diabetic patients against non-diabetic controls, or contrast the presence versus absence of retinopathy, rather than examining whether CS declines

progressively across increasing grades of retinopathy once retinopathy is already established. Second, few studies evaluate CS across the different grades of NPDR specifically, as opposed to a binary DR/no-DR classification. Third, few studies adjust for clinically important covariates particularly DME, visual acuity, refractive error, glycemic control, and diabetes duration in a single multivariable framework when assessing the independent relationship between CS and retinopathy grade. Given that DME can occur at any stage of NPDR and independently disrupts macular photoreceptor and neuronal architecture, its omission as a covariate may confound any apparent CS-severity gradient reported in unadjusted analyses.

We evaluated CS across graded categories of NPDR while simultaneously adjusting for these covariates and evaluating the diagnostic accuracy of CS for identifying moderate-to-severe diseases using receiver operating characteristic analysis. This study fills that gap by taking a closer look at whether contrast sensitivity truly stands on its own as a functional marker of how severe retinopathy is, or whether the link researchers see is just a byproduct of macular pathologies happening alongside it

METHODS

This cross-sectional study was conducted at Hayatabad Medical Complex, Peshawar from October 2020 to November 2021. Ethical approval was obtained from the Institutional Research and Ethics Board (312/HEC/B&PSC/2020) prior to enrolment, and the study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants. Reporting follows the STROBE guidelines for cross-sectional studies.¹²

A total of 245 patients with type 1 or type 2 diabetes mellitus and a confirmed diagnosis of NPDR (mild, moderate, or severe, per the International Clinical Diabetic Retinopathy Disease Severity Scale) were enrolled through non-probability consecutive sampling from the outpatient ophthalmology clinic.¹³ Inclusion criteria were age ≥ 18 years, a clinical diagnosis of diabetes mellitus, and gradable fundus findings corresponding to mild, moderate, or severe NPDR. Exclusion criteria were proliferative DR, any prior intraocular surgery (all included eyes were phakic), coexisting non-diabetic retinal or optic nerve pathology, media opacity precluding fundus grading or CS testing, and patients unable to perform CS testing

reliable. No missing data occurred for any recorded variable in the final analytic dataset of 245 patients; consequently, no imputation was required.

For patients with bilateral gradable disease, one eye per patient was selected for analysis to avoid statistical non-independence between eyes: the eye with the poorer best-corrected visual acuity was selected as the study eye, and the right eye was selected where visual acuity was equal between the two eyes. The retinopathy grade, CS score, and all ocular covariates reported reflect this designated study eye.

CS was measured monocularly in the study eye using the Pelli-Robson letter chart.¹⁴ Testing was performed at a standardized distance of 1 meter, with best distance correction in place, under photopic chart illumination with the recommended background luminance range and glare controlled as per standard chart instructions.¹⁵ The chart comprises triplets of Sloan letters of constant size and progressively decreasing contrast (0.15 log unit steps between triplets); a contrast level was credited if at least two of the three letters in a triplet were correctly identified, and the score was recorded in log CS units (range 0-2.25), with each additional correctly identified letter contributing 0.05 log units.

All patients attending the ophthalmology outpatient department underwent a thorough ophthalmic workup. This began with best-corrected visual acuity, recorded on the Snellen chart and later converted to LogMAR values for analysis. Slit-lamp biomicroscopy, applanation tonometry, and dilated fundus examination followed, with the examining ophthalmologist grading retinopathy status directly.

To determine whether DME was present, we relied on a combination of slit-lamp findings and OCT; whenever retinal thickening was suspected on clinical grounds, OCT was used to confirm the diagnosis. Refractive error was recorded as spherical equivalent in diopters using autorefraction, with retinoscopy was performed to verify these readings.

Systemic parameters such as diabetes duration, HbA1c, and both systolic and diastolic blood pressure were extracted from patient records at the time of ocular examination. Every patient included in the study was phakic (confirmed via slit-lamp exam), which by design eliminated cataract as a confounding factor in CS measurements.

Statistical analysis was performed using IBM SPSS Version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean $\pm$ standard deviation (SD), whereas categorical variables were summarized as frequencies and percentages. We checked the normality of data distribution using the Shapiro-Wilk test, and homogeneity of variance was assessed with Levene's test.

To examine differences in contrast sensitivity (CS) across NPDR grades, one-way analysis of variance (ANOVA) was applied; given the possibility of non-normal distribution in some subgroups, the Kruskal-Wallis test was also run as a non-parametric alternative. The relationship between NPDR grade and CS was further explored using Spearman's rank correlation.

We then performed multivariable linear regression to identify which factors were independently associated with CS including NPDR grade, age, diabetes duration, HbA1c, visual acuity, spherical equivalent, and DME status. As an additional check, a sensitivity analysis was conducted after excluding eyes with DME, to see whether this altered the observed associations. Throughout, a two-tailed p-value of less than 0.05 was taken to indicate statistical significance.

RESULTS

A total of 245 patients with NPDR were included in the study, comprising 66 (26.9%) with mild NPDR, 130 (53.1%) with moderate NPDR, and 49 (20.0%) with severe NPDR. Mean age was 50.1 ± 10.7 years. Age, sex, and diabetes type were comparable across the three groups, whereas diabetes duration, HbA1c, blood pressure, visual acuity, and the prevalence of DME increased with increasing disease severity (Table 1).

Mean CS showed a gradual decline with increasing NPDR severity, decreasing from 1.26 ± 0.38 in mild NPDR to 1.23 ± 0.40 in moderate NPDR and 1.13 ± 0.39 in severe NPDR. However, this difference was not statistically significant (ANOVA, $F=1.76$, $p=0.175$). Similar findings were observed with the Kruskal-Wallis test ($H=3.51$, $p=0.173$). Pairwise comparisons between the three groups were not statistically significant, and Spearman's correlation demonstrated a weak, non-significant inverse relationship between NPDR grade and CS ($\rho=-0.107$, $p=0.095$).

Table 1: Baseline demographic and clinical characteristics by grade of DR (N=245).

Variable	Mild (n=66)	Moderate (n=130)	Severe (n=49)
Age, years (mean $\pm$ SD)	51.2 $\pm$ 11.6	50.2 $\pm$ 10.5	48.4 $\pm$ 10.3
Sex, male/female, n	34 / 32	68 / 62	24 / 25
Diabetes type, type 1/type 2, n	8 / 58	17 / 113	9 / 40
Diabetes duration, years (mean $\pm$ SD)	6.5 $\pm$ 2.6	9.7 $\pm$ 2.4	14.8 $\pm$ 2.6
HbA1c, % (mean $\pm$ SD)	7.36 $\pm$ 0.76	8.10 $\pm$ 0.83	9.18 $\pm$ 0.78
Systolic BP, mmHg (mean $\pm$ SD)	129.9 $\pm$ 11.1	135.4 $\pm$ 10.2	140.0 $\pm$ 10.4
Diastolic BP, mmHg (mean $\pm$ SD)	80.5 $\pm$ 5.7	82.6 $\pm$ 6.1	87.4 $\pm$ 7.0
Visual acuity, LogMAR (mean $\pm$ SD)	0.08 $\pm$ 0.10	0.21 $\pm$ 0.14	0.38 $\pm$ 0.14
Spherical equivalent, D (mean $\pm$ SD)	-0.43 $\pm$ 1.83	-0.49 $\pm$ 1.83	-0.64 $\pm$ 1.55
Diabetic macular oedema, n (%)	8 (12.1%)	46 (35.4%)	27 (55.1%)
Contrast sensitivity score (mean $\pm$ SD)	1.26 $\pm$ 0.38	1.23 $\pm$ 0.40	1.13 $\pm$ 0.39

Table 2: Correlation matrix of study variables (Spearman's ρ ; p-values in parentheses).

Variable	CS Score	DR Grade	Age	DM Duration	HbA1c	VA (LogMAR)
DR Grade	-0.107 (0.095)	—				
Age	0.023 (0.717)	-0.086 (0.179)	—			
DM Duration	-0.064 (0.318)	0.741 (<0.001)	-0.177 (0.005)	—		
HbA1c	-0.108 (0.091)	0.608 (<0.001)	-0.012 (0.855)	0.443 (<0.001)	—	
VA (LogMAR)	-0.004 (0.952)	0.618 (<0.001)	-0.046 (0.472)	0.510 (<0.001)	0.392 (<0.001)	—
Sph. Equivalent	0.078 (0.222)	-0.040 (0.528)	-0.142 (0.026)	0.040 (0.532)	0.006 (0.928)	0.008 (0.899)

VA = visual acuity; DM Duration = diabetes duration; Sph. Equivalent = spherical equivalent.

Table 3: Multivariable linear regression for contrast sensitivity score (DME-adjusted model).

Predictor	β (U NSTD.)	Std. β	SE	95% CI	p-value
NPDR grade (ordinal)	-0.044	-0.076	0.069	-0.180, 0.092	0.526
Age	0.001	0.027	0.002	-0.004, 0.006	0.685
Diabetes duration	0.000	0.004	0.010	-0.020, 0.020	0.968
HbA1c	-0.033	-0.085	0.032	-0.096, 0.029	0.293
Visual acuity (LogMAR)	0.232	0.096	0.196	-0.155, 0.618	0.239
Spherical equivalent	0.015	0.065	0.015	-0.014, 0.043	0.315
DME (present)	-0.099	-0.12	0.057	-0.211, 0.013	0.082

Model F=1.39 (p=0.210); R^2 =0.039; adjusted R^2 =0.011, indicating no problematic multicollinearity.

Increasing NPDR severity was significantly associated with longer diabetes duration, higher HbA1c levels, and poorer visual acuity (all p <0.001), consistent with recent evidence linking glycemic control to retinopathy severity. In contrast, contrast sensitivity showed no significant correlation with age, diabetes duration, HbA1c, visual acuity, or spherical equivalent (Table 2).

Multivariable linear regression demonstrated that DME had the largest negative association with contrast sensitivity (standardized β =-0.12), although this did not reach statistical significance after adjustment (p =0.082). NPDR grade was likewise not independently associated with CS (standardized β = -0.076, p =0.526). The overall model explained only

a small proportion of the variability in CS (adjusted R^2 = 0.011), with no evidence of problematic multicollinearity (Table 3).

In the pre-specified sensitivity analysis excluding patients with DME, the small decline in CS across NPDR grades was no longer evident. Mean CS was similar among patients with mild, moderate, and severe NPDR (1.26, 1.27, and 1.22 log units, respectively), with no statistically significant difference between the groups (ANOVA, F =0.149, p =0.862). These findings suggest that the modest gradient observed in the overall cohort may have been influenced by the higher prevalence of DME in the more advanced NPDR groups rather than by NPDR severity alone.

DISCUSSION

This study demonstrated a gradual decline in CS with increasing severity of NPDR. However, this trend was not statistically significant after adjustment for age, diabetes duration, HbA1c, visual acuity, refractive error, and DME. Although DME showed the largest negative effect estimate in the multivariable model, it was not independently associated with CS after adjustment. Furthermore, the small numerical decline in CS across NPDR grades disappeared after excluding patients with DME, suggesting that macular involvement may have a greater influence on CS than retinopathy severity alone. These findings indicate that, although CS tends to decline with increasing NPDR severity, its relationship with disease severity is modest and appears to be influenced by coexisting clinical factors.

These findings are broadly consistent with previous studies. The population-based SN-DREAMS-II study reported that ocular and systemic factors together explained only about 29% of the variability in CS among individuals with type 2 diabetes, suggesting that retinopathy severity represents only one of several factors influencing CS.⁷ This is consistent with our findings, in which NPDR grade explained only a small proportion of the variability in CS (adjusted $R^2=0.011$). Similarly, a recent structure–function study using widefield optical coherence tomography angiography excluded eyes with DME to better evaluate the relationship between retinal vascular changes and CS across different stages of DR.¹¹ Our pre-specified sensitivity analysis likewise showed that, after excluding patients with DME, the small numerical difference in CS across NPDR grades was no longer evident, supporting the view that macular involvement may have a greater influence on CS than retinopathy severity itself.

In contrast, several previous studies have reported a stronger association between CS and DR. Sokol et al, demonstrated that patients with background DR had greater CS deficits than diabetic patients without retinopathy, while Lupión Durán et al, reported significantly reduced CS and low-contrast visual acuity in eyes with NPDR compared with diabetic patients without retinopathy and healthy controls.^{6,8} Similarly, a systematic review of 21 comparative cross-sectional studies concluded that reduced CS is a consistent finding in patients with DR.⁹ However, these studies differed from the present study in several important respects. Most compared eyes with DR to

eyes without retinopathy or to healthy controls, rather than evaluating differences across grades of established NPDR. In addition, none accounted for diabetic macular oedema as a separate covariate, despite its increasing prevalence with advancing NPDR (12.1% to 55.1%) and its potential independent effect on CS. These methodological differences may partly explain the discrepancy between our findings and those reported previously.

From a biological perspective, these findings are consistent with the current understanding of diabetic retinal disease as a disorder of the neurovascular unit.¹⁵ Retinal neurodegeneration, characterized by progressive ganglion cell loss and thinning of the inner retinal layers, may occur early in diabetes and progress relatively independently of the microvascular changes used to grade DR.⁵ In contrast, DME causes fluid accumulation within the macula, disrupting photoreceptor and Müller cell function in the region responsible for central vision and contrast perception. Although DR severity grading reflects the extent of retinal microvascular abnormalities, these changes do not necessarily correspond to macular involvement. Consequently, macular pathology may have a more direct influence on CS than the overall severity of retinopathy, which is consistent with the findings of the present study.

From a clinical perspective, the findings suggest that CS should not be considered an independent indicator of NPDR severity. However, it may still provide complementary functional information during the assessment of patients with DR, particularly in the presence of DME or other forms of macular involvement. CS testing should therefore be regarded as an adjunct to, rather than a replacement for, comprehensive retinal examination and OCT. Larger prospective, multicentre studies encompassing the full spectrum of DR are needed to further clarify the role of CS in the functional assessment of diabetic retinal disease.

This study has several limitations. As a single-centre, cross-sectional study using non-probability consecutive sampling, its findings may not be generalizable, and causal relationships cannot be inferred. The absence of diabetic patients without retinopathy and those with proliferative DR limited the evaluation across the full spectrum of disease. In addition, CS was assessed only under photopic conditions, and subtle functional deficits under other testing conditions may have been missed, as deficits

related to diabetic neurodegeneration have been reported to be more pronounced under mesopic conditions.^{16,17} Although the study was adequately powered to detect moderate effects, it may have been underpowered to identify small differences in CS across NPDR grades. Larger, prospective multicentre studies are needed to validate these findings.

CONCLUSION

CS showed a modest decline with increasing severity of NPDR. However, this association was not statistically significant after adjustment for relevant clinical factors. DME appeared to have a greater influence on CS than NPDR severity alone, although it was not independently associated after multivariable adjustment. These findings suggest that CS should not be considered an independent marker of NPDR severity but may serve as a complementary functional assessment alongside routine clinical evaluation.

Acknowledgements

The authors acknowledge the Department of Ophthalmology, Khyber Teaching Hospital, Peshawar, for facilitating this research.

Funding: This study was not funded by any organization.

Patient's Consent: Researchers followed the guidelines set forth in the Declaration of Helsinki.

Conflict of Interest: Authors declared no conflict of interest.

Ethical Approval: The study was approved by the Institutional review board/Ethical review board (312-HEC/B&PSC/2020)

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