

Original Article

Dry Eye Disease And Corneal Nerve Sensitivity: A Comparative Analysis Between Diabetics And Non-Diabetics

Tooba Ilyas¹, Asfandiyar Asghar², Rana Intisar-ul-Haq³, Tehmina Nazir⁴, Naila Obaid⁵

Abstract

Objective: To compare the prevalence of dry eye disease (DED) and corneal nerve sensitivity (CNS) in diabetic and non-diabetic individuals.

Methodology: A Comparative cross-sectional study was conducted in the Ophthalmology Department, Fauji Foundation Hospital, Rawalpindi, Pakistan, from January - June 2025. A total of 300 adults aged 40–70 years were enrolled, including 150 patients with diabetes and 150 demographically comparable individuals without diabetes. DED symptoms were assessed using the SPEED questionnaire, and objective assessments included corneal nerve sensitivity, Schirmer's test and TBUT. Data were analysed using the Mann–Whitney U, Chi-square, and Fisher's exact tests, with Firth penalised logistic regression used to estimate adjusted odds ratios (aOR) with 95% confidence intervals (CI).

Results: Diabetic participants had higher HbA1c [7.61(0.94)% vs. 5.37(0.26)%; $P<0.001$], higher SPEED scores [18.77(2.84) vs. 11.87(1.32); $P<0.001$], lower Schirmer's values [6.97(2.26) mm vs. 17.50(0.94) mm; $P<0.001$], and shorter TBUT [7.21(2.09) s vs. 11.85(1.09) s; $P<0.001$]. DED and reduced corneal nerve sensitivity were each present in 83.3% of diabetic participants but not in any of the controls ($P<0.001$). Within the diabetic cohort, longer duration of diabetes independently predicted DED (aOR 1.55 per year; $p=0.002$).

Conclusion: Diabetes mellitus is strongly associated with DED and corneal nerve dysfunction. Reduced corneal nerve sensitivity may serve as an early marker of diabetic complications, thus supporting routine ocular surface and corneal sensitivity assessments for timely detection and intervention.

Keywords: Diabetes mellitus; dry eye disease; corneal nerve sensitivity; Schirmer tear test; tear film break-up time; HbA1c

Introduction

Dry eye disease (DED) is a prevalent, multifactorial ocular condition characterised by reduced tear production or tear film instability.¹ The tear film protects the ocular surface from environmental insults and provides a smooth refractive surface that is essential for clear vision.² Disruption of tear film homeostasis can cause blurred vision, dryness, irritation, redness, foreign body sensation, and light sensitivity.² Globally, DED affects an estimated 5–34% of individuals, emphasising its substantial burden on public health systems.¹

Diabetes mellitus (DM) is defined by persistent hyperglycaemia and gradually affects multiple organ systems, including the eyes. Diabetic eye disease comprises conditions such as DED, cataract, glaucoma, diabetic retinopathy, and macular oedema, all of which can compromise vision and quality of life.³ Evidence supports a strong association between DM and DED, with both conditions being closely linked to corneal nerve sensitivity, which is an essential component of ocular surface health.

In DM, systemic metabolic disturbances impair the tear film composition and reduce tear production, leading to classic DED symptoms such as dryness, irritation, and blurred vision.^{4,5} Diabetes also causes structural changes in the corneal nerves, reducing neurotrophic support and corneal sensitivity. Decreased corneal nerve sensitivity impairs ocular surface reflexes, increasing the risk of dryness, delayed healing, and epithelial

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damage.⁶⁻⁸ Importantly, corneal nerve dysfunction may also serve as an early biomarker of diabetes, allowing the identification of high-risk individuals before the development of clinical disease.

Studies have reported a DED prevalence of 45.75% in diabetics compared with 9.5% in non-diabetics, while reduced corneal nerve sensitivity was observed in 47% of diabetics and 3% of non-diabetics.¹ Despite this global evidence, comprehensive local data from Pakistan on the burden of DED and corneal nerve dysfunction in patients with diabetes remain limited, highlighting a gap in regional clinical knowledge.

This study aimed to assess and compare DED between diabetic (case) and non-diabetic (control) individuals in the Pakistani population using the Standard Patient Evaluation of Eye Dryness (SPEED) questionnaire and objective measures, including corneal nerve sensitivity, Schirmer's test, and tear breakup time (TBUT). The findings may support early detection, tailored management, and improved ocular health in patients with diabetes while addressing the gap in local clinical practice.

Methodology

This comparative cross-sectional study was conducted at the Ophthalmology Department of Fauji Foundation Hospital, Rawalpindi, between January and June 2025. The study was approved by the Institutional Ethical Review Board (Reference No. [825/RC/FFH/RWP]) and conducted in accordance with ethical standards and principles, with approval obtained before the commencement of the research.

The sample size was calculated using the WHO Sample Size Calculator with a significance level of 5%, power of 80%, P1 = 47.75%, and P2 = 9.75%, resulting in a total of 300 participants, including 150 diabetic and 150 non-diabetic participants. Eligible participants were recruited using a non-probability consecutive sampling technique.

The diabetic group included patients with diabetes mellitus, irrespective of glycaemic control or disease duration, whereas the non-diabetic group comprised participants without a history of diabetes. Both males and females aged 40–70 years were included. Participants were excluded if they had ocular surface disease, recent ocular surgery, contact lens use, severe systemic or neurological disorders, medications affecting tear production, or corneal nerve function, or significant environmental exposures.

After obtaining written informed consent, the participants were enrolled according to the predefined eligibility criteria. The privacy, confidentiality, and autonomy of the participants were maintained throughout the study. Demographic data and diabetes-related history were recorded using a structured pro forma. Dry eye symptoms were assessed using the Standard Patient Evaluation of Eye Dryness (SPEED) questionnaire.^{8,27} Objective assessments were performed starting with corneal nerve sensitivity using a cotton-tipped applicator, followed by evaluation of tear production using Schirmer's test (Schirmer's test strip without topical anaesthetic) and tear film stability using tear breakup time (TBUT). All assessments were performed by a single investigator using standardised procedures to ensure consistency and to minimise measurement bias. All the participants completed the study assessments, and there were no missing data. DED severity was categorised as mild (SPEED 6–10, Schirmer 10–15 mm, and TBUT 6–10 s), moderate (SPEED 11–15, Schirmer 5–9 mm, and TBUT 4–5 s), and severe (SPEED >15, Schirmer <5 mm, and TBUT <4 s).

Data analysis was performed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA; RRID: SCR_002865), with confidence intervals and penalised regression analyses performed using Python 3 (SciPy and Statsmodels). The predefined analytical plan was established before data analysis, including DED and reduced corneal nerve sensitivity as primary outcomes, adjusted for age, gender and diabetes duration. Age and HbA1c levels were analysed as continuous variables, and no data-driven variable selection was performed. Quantitative variables were assessed for normality using the Shapiro–Wilk test and Q–Q plots and presented as mean with standard deviation and 95% confidence intervals. Non-normally distributed variables were analyzed using the Mann–Whitney U-test. Categorical variables were reported as frequencies and percentages and analysed using the Chi-square and Fisher's exact tests. Firth's penalised logistic regression was used for DED due to complete separation from the absence of DED in the control groups. A two-sided P-value ≤ 0.05 was considered to be statistically significant.

Results

The study included 300 participants aged 40–70 years, of whom 150 were diabetic and 150 were non-diabetic. The two groups were comparable in age, with a mean age of 57.62 (5.85) years in the diabetic group and 57.31 (5.82) years in the non-diabetic group, with a mean difference of 0.31 years [95% CI (–0.80 to 1.52)]; ($P = 0.83$). Females comprised 71.67% ($n = 215$) of the study population, resulting in a male-to-female ratio of 1:2.5. Among the participants with diabetes, 39 (26.0%) were males, and 111 (74.0%) were females, whereas the non-diabetic group included 46 (30.7%) males and 104 (69.3%) females. Diabetic participants had significantly higher glycated

haemoglobin (HbA1c) levels than non-diabetics [7.61 (0.94) vs. 5.37 (0.26); mean difference 2.24%, 95% CI (2.08–2.39); $P < 0.001$]. Subjective assessment using the SPEED questionnaire showed greater ocular discomfort in diabetics [18.77 (2.84) vs. 11.87 (1.32); mean difference 6.90, 95% CI (6.40–7.40); $P < 0.001$]. Objective evaluation demonstrated lower Schirmer's values [6.97 (2.26) mm vs. 17.50 (0.94) mm; mean difference –10.53 mm, 95% CI (–10.93 to –10.14); $P < 0.001$] and shorter TBUT [7.21 (2.09) s vs. 11.85 (1.09) s; mean difference –4.64 s, 95% CI (–5.02 to –4.26); $P < 0.001$] in patients with diabetes, reflecting reduced tear production and tear film instability. These findings confirmed significant impairment in both subjective symptoms and objective tear parameters, as shown in Table 1 and Figure 1, which illustrates the higher SPEED scores and lower TBUT values among diabetic participants.

Table 1: Glycemic control and tear film parameters

Parameter	Diabetics Mean (SD)	Non-diabetics Mean (SD)	Mean Difference (95% CI)	P value
HbA1c (%)	7.61 (0.94)	5.37 (0.26)	2.24 (2.08 to 2.39)	<0.001
SPEED score	18.77 (2.84)	11.87 (1.32)	6.90 (6.40 to 7.40)	<0.001
Schirmer's test (mm)	6.97 (2.26)	17.50 (0.94)	-10.53 (-10.93 to -10.14)	<0.001
TBUT (seconds)	7.21 (2.09)	11.85 (1.09)	-4.64 (-5.02 to -4.26)	<0.001

Distribution of SPEED scores and tear break-up time by diabetic status

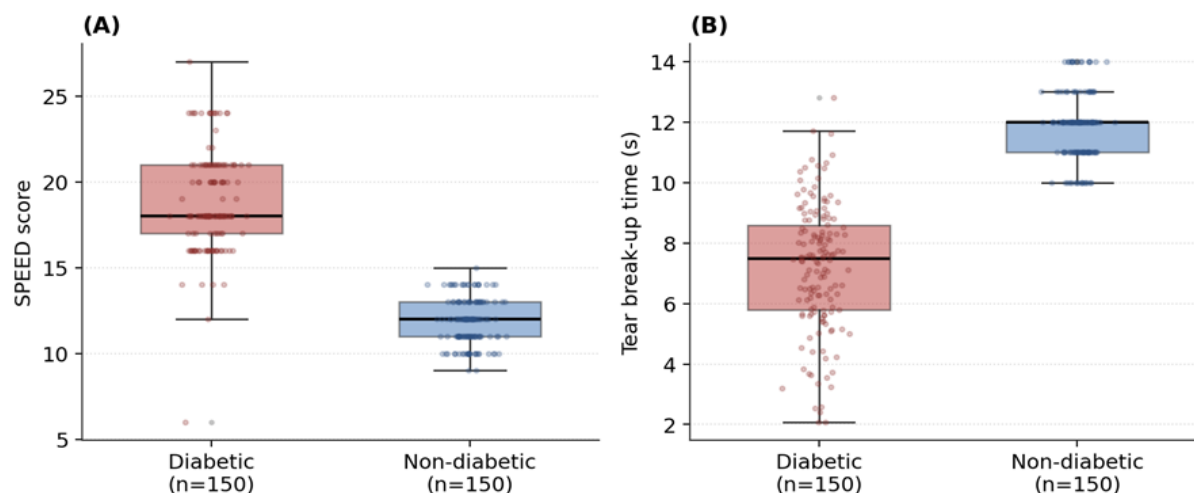


Figure 1: Comparison of SPEED scores (A) and TBUT values (B) between diabetic and non-diabetic participants.

Dry eye disease was observed in 125 (83.33%; 95% CI 76.6–88.4) diabetic participants, whereas none of the age-matched non-diabetic participants were affected [0.0%; 95% CI 0.0–2.5; Fisher's exact $P < 0.001$]. Similarly, reduced corneal nerve sensitivity was present in 125 (83.33%) diabetics compared with 0 (0.0%) non-diabetics ($P < 0.001$). Among diabetic participants aged 40–55 years, DED and reduced corneal nerve sensitivity were present in 37 (72.55%), compared with 88 (88.89%) in those aged 56–70 years, demonstrating a higher prevalence with advancing age ($P = 0.011$). Gender-based analysis showed a higher prevalence in males than in females (36 [92.3%] vs 89 [80.2%]), although the difference was not statistically significant ($P = 0.080$) (Table 2).

Further analysis highlighted the effects of glycaemic control on ocular and systemic complications. Among patients with HbA1c $\leq 7.0\%$ ($n = 45$), reduced corneal nerve sensitivity was present in 20 (44.4%), diabetic retinopathy in 12 (26.7%), and peripheral neuropathy in 10 (22.2%). In contrast, those with HbA1c levels $> 7.0\%$ ($n = 105$) had universal corneal nerve sensitivity loss, with diabetic retinopathy and peripheral neuropathy in 74.3% and 68.6% of patients, respectively ($P < 0.001$).

The prevalence differences were 55.6% (95% CI 41.6–69.0) for reduced corneal sensitivity, 47.6% (95% CI 31.4–61.3) for diabetic retinopathy, and 46.4% (95% CI 30.2–60.0) for peripheral neuropathy, demonstrating a strong association between poor glycaemic control, ocular surface dysfunction, and diabetic complications. (Table 3)

Table 2: Prevalence of dry eye disease and reduced corneal nerve sensitivity with age- and gender-based stratification among diabetic participants

Variable	Diabetics (n = 150) n/N(%)	Non-diabetics (n = 150) n/N(%)	Mean Difference (95% CI)	P value
Dry eye disease	125/150 (83.3)	0/150 (0.0)	83.3 (76.1 to 88.4)	<0.001
Reduced corneal nerve sensitivity	125/150 (83.3)	0/150 (0.0)	83.3 (76.1 to 88.4)	<0.001
Age (Diabetics)	40–55 yrs: 37/51 (72.6) 56–70 yrs: 88/99 (88.9)	—		0.011
Gender (Diabetics)	Male: 36/39 (92.3) Female: 89/111 (80.2)	—		0.080

Table 3: Association of HbA1c with corneal nerve sensitivity, diabetic retinopathy and peripheral neuropathy in diabetic patients (n = 150)

HbA1c level	n/N (%)	Reduced corneal nerve sensitivity	Normal corneal nerve sensitivity	Diabetic retinopathy present	Diabetic retinopathy absent	Peripheral neuropathy present	Peripheral neuropathy absent	P value
≤7.0%	45 (30.0)	20/45 (44.44)	25/45 (55.56)	12/45 (26.67)	33/45 (73.33)	10/45 (22.22)	35/45 (77.78)	<0.001
>7.0%	105 (70.0)	105/105 (100.0)	0/105 (0.0)	78/105 (74.29)	27/105 (25.71)	72/105 (68.57)	33/105 (31.43)	<0.001
Difference (95 % CI)		55.6 (41.6 to 69.0)		47.6 (31.4 to 61.3)		46.4 (30.2 to 60.0)		

Multivariable Firth penalised logistic regression showed a strong association between diabetes and DED after adjusting for age and gender (aOR 1713.2, [95% CI 107.4–27339.8]; $P < 0.001$). The wide CI reflects complete separation due to the absence of DED among the controls and requires cautious interpretation. Within the diabetic cohort, longer diabetes duration independently predicted DED (aOR 1.55 per year, 95% CI 1.17–2.05; $P = 0.002$), whereas age and gender were not significant predictors. Higher HbA1c levels were also associated with DED (aOR 533.0 per 1% increase, 95% CI 28.0–10150.0; $P < 0.001$), with cautious interpretation of age effects after HbA1c adjustment due to possible collinearity between age and diabetes-related factors. (Table 4)

Table 4: Multivariable Firth penalized logistic regression for dry eye disease

Predictor	Adjusted OR (95% CI)	P-value
Model A – full sample (n = 300); outcome: DED		
Diabetes (vs no diabetes)	1713.2 (107.4 to 27339.8)	<0.001
Age (per year)	1.06 (0.98 to 1.14)	0.16
Female sex	0.44 (0.14 to 1.44)	0.18
Model B – diabetic cohort (n = 150); outcome: DED		
Age (per year)	0.97 (0.88 to 1.06)	0.46
Female sex	0.45 (0.13 to 1.57)	0.21
Diabetes duration (per year)	1.55 (1.17 to 2.05)	0.002
Model C – diabetic cohort, adding glycemic control		
HbA1c (per 1%)	533.0 (28.0 to 10150.0)	<0.001
Age (per year)	0.83 (0.72 to 0.97)	0.022
Diabetes duration (per year)	1.20 (0.93 to 1.53)	0.16
Female sex	0.37 (0.04 to 3.24)	0.37

Discussion

Dry eye disease (DED) is a multifactorial ocular surface disorder characterized by burning, stinging, itching, redness, foreign body sensation, and photophobia. Its pathogenesis involves tear film instability, hyperosmolarity, chronic inflammation, and neurosensory dysfunction. In diabetes mellitus, persistent hyperglycemia affects the lacrimal functional unit, meibomian glands, goblet cells, and corneal nerves, resulting in reduced tear secretion, impaired blink reflexes, autonomic dysfunction, and ocular surface damage.^{9,10}

In our study, 150 patients with diabetes were compared with age- and gender-matched non-diabetic controls to assess the prevalence of DED and corneal nerve sensitivity. Both groups had comparable baseline characteristics, with most participants in their fifth and sixth decades of life. Mangoli et al.¹ reported a similar mean age of 55.02 (12.21) years, reflecting the predominance of type 2 diabetes and a relatively higher average age.

In our study, redness was the most frequently reported symptom, followed by burning, stinging, foreign body sensation, pruritus, and photophobia. Bashorun et al.¹¹ reported grittiness in 58.3% of patients with diabetes and itching in 62.5% of non-diabetics. These differences may reflect variations in disease duration, environmental factors, symptom perception, and assessment methods.

In our study, DED was assessed using the SPEED questionnaire and objective tests (Schirmer's test and TBUT). DED and reduced corneal nerve sensitivity were observed in 83.33% of the diabetic participants compared with 0% of the controls. These findings are consistent with previous reports; Manchikanti et al.,¹⁵ observed moderate-to-severe DED symptoms in 76.2% of diabetics compared with predominantly normal-to-mild symptoms in 90.5% of non-diabetics, with significantly lower Schirmer and TBUT values in diabetics ($P < 0.01$). Similarly, Bashorun et al.¹¹ reported dry eye symptoms in 87.31% of diabetic participants, while Lamba et al.,¹⁶ demonstrated a higher prevalence of DED with reduced Schirmer's test and TBUT values.

In our study, the absence of DED and normal corneal nerve sensitivity among age- and gender-matched non-diabetic controls may reflect the use of stringent diagnostic criteria requiring both symptoms and objective tear film abnormalities, along with the exclusion of potential confounding factors. Corneal hypoesthesia is uncommon in healthy eyes and is typically associated with diabetic neuropathy and ocular injury. The comparable baseline characteristics between the groups strengthened the internal validity of our study and supported the association between diabetes and ocular surface dysfunction.

A well-established association between diabetes and DED has been supported by previous studies demonstrating reduced tear function and corneal nerve abnormalities. Dua et al.²² reported higher rates of neuropathy and ocular surface lesions, whereas Aljarousha et al.²⁵ and Liu et al.²³ observed reduced TBUT, Schirmer values, and corneal sensation, particularly in patients with poor glycaemic control or advanced diabetic complications. Similar diabetes-related corneal and ocular surface abnormalities, including postoperative changes, were reported by Ladea et al.,¹⁷ and Koestel et al.,²⁶ which is consistent with our findings.

Naik et al.,¹² and Kuo et al.,¹³ reported that longer diabetes duration, higher HbA1c levels, and poor glycaemic control were associated with tear film dysfunction and reduced Schirmer and TBUT values. Our findings corroborate these observations, with longer disease duration and poor glycaemic control associated with greater ocular surface abnormalities and reduced corneal sensitivity. Similarly, Mansuri et al.,¹⁴ and a systematic review by Chen et al.,²⁴ reported an increased risk of DED among patients with diabetes, particularly those with diabetic retinopathy, supporting our findings.

Poor glycaemic control was strongly associated with corneal nerve dysfunction, as all patients with HbA1c $>7.0\%$ had reduced corneal sensitivity compared with 44.44% of patients with well-controlled diabetes. Diabetic retinopathy (74.29%) and peripheral neuropathy (68.57%) were also more frequent in patients with poorly controlled DM, consistent with the findings of Zhou et al.¹⁹, Li et al.²⁰, and Ponirakis et al.²¹, supporting corneal sensitivity assessment as a potential early marker of diabetic microvascular and neuropathic complications.

Peripheral neuropathy is closely associated with reduced corneal sensitivity and ocular discomfort. In our study, patients with diabetes had significantly lower corneal sensitivity than controls, consistent with previous reports. Aljarousha et al.,²⁵ Dua et al.,²², Liu et al.,²³, and Qin et al.,¹⁸ demonstrated similar associations between reduced corneal sensitivity, poor glycaemic control, neuropathy, and ocular surface discomfort, supporting corneal sensitivity assessment as a potential early marker of diabetic neuropathic changes.

Multivariable analysis showed that diabetes duration, rather than age alone, independently predicted DED (aOR 1.55 per year), suggesting that the observed age-related effect reflected cumulative hyperglycaemic exposure. Corneal sensitivity assessment may serve as a simple, non-invasive marker of diabetic neuropathy risk and help identify patients at a higher risk of complications.^{7,8,14}

Clinically, our findings demonstrated a strong association between diabetes, poor glycaemic control, and ocular surface dysfunction, with a high prevalence of DED (83.33%), corneal hypoesthesia (83.33%), and impaired tear

film stability among diabetic participants. These results support the incorporation of ocular surface evaluation and corneal sensitivity assessment into routine diabetic eye care, enabling early detection and timely management, and complementing existing diabetic retinopathy screening strategies.¹⁵⁻¹⁹

In our study, the cotton wisp method provided a convenient assessment of corneal nerve sensitivity, but it lacked the quantitative precision of standardised esthesiometers, which are costly and not widely available. Although groups were matched for age and gender, residual confounding from diabetes duration, systemic comorbidities, and medication use may have influenced the findings. The single-centre design, non-probability consecutive sampling, and subjective symptom reporting may have introduced selection and response bias. The absence of DED among controls resulted in complete separation; although the Firth penalised regression provided adjusted estimates, the wide confidence intervals require cautious interpretation. Furthermore, the unavailability of advanced tools, such as in vivo confocal microscopy, restricts objective corneal nerve assessment. To address these limitations, standardised protocols were followed, and all assessments were performed by a single investigator to ensure consistency and minimise observer-related variability.

Conclusions

Our study highlights a strong association between diabetes mellitus, dry eye disease (DED), and reduced corneal sensitivity. Among the patients with diabetes, a longer disease duration was identified as an independent predictor of DED, suggesting the cumulative effect of chronic hyperglycaemia on ocular surface health. Corneal sensitivity assessment may provide a simple, non-invasive indicator of diabetic microvascular and neuropathic changes, including diabetic retinopathy and peripheral neuropathy. Early recognition of DED and corneal hypoesthesia, followed by appropriate management with lubricants, tear conservation strategies and regular monitoring, may help slow disease progression and reduce complications. Incorporating ocular surface evaluation and corneal sensitivity testing into routine diabetic eye care may facilitate the early detection of ocular and systemic complications, improve visual outcomes, and reinforce the role of comprehensive ophthalmic evaluation in diabetes management.

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Contributions:

TI - Conception, Design

AA - Acquisition, Analysis, Interpretation

TI - Drafting

AA RIUH TN NO - Critical Review

All authors approved the final version to be published & agreed to be accountable for all aspects of the work.

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