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COMPARATIVE ASSESSMENT OF TEAR FILM DYSFUNCTION AND CORNEAL NERVE SENSITIVITY IN PATIENTS WITH AND WITHOUT DIABETES MELLITUS

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ABSTRACT

Background: Diabetes mellitus can affect the ocular surface and corneal nerves, leading to tear film instability, dry eye changes, reduced corneal sensitivity, and delayed epithelial healing.

Objective: To compare tear film dysfunction and corneal nerve sensitivity in patients with and without diabetes mellitus.

Methods: This comparative cross-sectional study was conducted at Opthalmogology department, Mughal Eye Hospital Lahore from November 2024 to August 2025, included 35 participants, divided into a diabetic group (n = 18) and a non-diabetic control group (n = 17). Demographic and clinical data were recorded. Tear film dysfunction was assessed using tear breakup time, Schirmer test, and fluorescein staining.

Results: Diabetic patients showed significantly reduced tear breakup time compared with non-diabetic participants (6.1 ± 2.0 vs 10.2 ± 2.7 seconds; $p < 0.001$). Schirmer test values were also significantly lower in the diabetic group (8.4 ± 3.1 vs 14.0 ± 4.2 mm/5 min; $p < 0.001$), while fluorescein staining scores were higher (1.44 ± 0.92 vs 0.47 ± 0.62; $p = 0.001$). Tear film dysfunction was more frequent among diabetic patients than controls (77.8% vs 29.4%; $p = 0.004$).

Conclusion: Patients with diabetes mellitus had significantly greater tear film dysfunction and reduced corneal nerve sensitivity compared with non-diabetic individuals.

KEYWORDS: Diabetes mellitus; tear film dysfunction; dry eye disease; corneal sensitivity; diabetic keratopathy; Schirmer test

INTRODUCTION

The Diabetes mellitus is a chronic metabolic disorder in which there is a chronic hyperglycemia due to the deficiency of insulin production, insulin action, or both [1]. Hyperglycemia over the long term causes structural and functional damage to various organ systems, especially the blood vessels and nerves, which is known to result in complications like retinopathy, nephropathy, neuropathy and cardiovascular disease [2]. Diabetic retinopathy is often the focus in clinical practice; however, diabetes can also impact the anterior segment of the eye such as the tear film, corneal epithelium and corneal nerves. These modifications can lead to eye discomfort, decrease in tear production, slower corneal healing, decreased corneal sensation, and a lack of protection on the eye surface [3]. An important ocular surface defect is tearing film dysfunction which can occur more often in persons with diabetes mellitus. The tear film is vital for proper lubrication of the eye surface, optical clarity, and protection from environmental stressors, as well as healthy eye surface and epithelium [4]. Dry eye disease is a multifactorial disorder of the ocular surface characterized by a loss of tear film homeostasis, tear film instability, hyperosmolarity, inflammatory, damage and neurosensory abnormalities of the ocular surface [5]. Hence, tear film dysfunction is not just a minor inconvenience, but it can have an impact on visual function, reading, tolerance to digital devices and quality of life. Diabetic patients can have tear film dysfunction for several reasons. Chronic hyperglycemia can lead to a change in lacrimal gland function, decreased reflex tearing, a decreased density of goblet cells, disruption of mucin production and development of inflammation of the ocular surface [6]. Diabetes can also cause meibomian gland dysfunction, which leads to an unstable lipid layer and loss of tears. Further, autonomic neuropathy can affect tear production, and corneal nerve injury can diminish the blink reflex and sensory feedback that is necessary for tear production. The net effect of these interactions can be tearing film instability, decreased tear breakup time, increased staining of the ocular surface, and symptoms of dryness, burning, foreign body sensation, irritation, and vision changes [7].

Another clinically important parameter in diabetic eye disease is corneal nerve sensitivity. Corneal sensory nerves are critical for the maintenance of epithelial integrity, tear secretion, blink reflex, wound healing and maintenance of ocular surface homeostasis, and are one of the most highly innervated tissues in the body [8]. Peripheral neuropathy in diabetes can affect the corneal nerves, leading to decreased corneal sensitivity, decreased nerve fiber density, delayed healing of the epithelium, recurrent epithelial defects and increased risk of neurotrophic keratopathy [9]. Injury to the cornea's nerves and loss of corneal sensory function may occur due to hyperglycemia-related oxidative stress, advanced glycation end-products, inflammation, and microvascular injury. It is particularly relevant that the two systems are closely related; the relationship between tear film dysfunction and corneal nerve sensitivity is particularly important [10]. Normal corneal sensation activates reflex tearing and reflex blinking, which promotes tear film stability. Although symptoms may not be significant, when corneal sensitivity is reduced, there may be severe damage to the ocular surface [11]. This makes the clinical issue in diabetes: some patients may not report that their eyes are very dry or irritated, even though there are objective signs of OCD. Research has indicated that diabetic patients, including those with neuropathy, may exhibit a decrease in corneal sensitivity and dysfunction of the tear film, without significant symptoms [12].

Objective

To compare tear film dysfunction and corneal nerve sensitivity in patients with and without diabetes mellitus.

METHODOLOGY

This comparative cross-sectional study was conducted at Opthalmogology department of Mughal Eye Hospital Lahore from November 2024 to August 2025. Thirty-five patients were included and randomly allocated to two groups for this study. The patients with diabetes mellitus were categorized into Group A and the non-diabetic patients were categorized into Group B as controls. A total of 35 patients were studied. Among them, 18 patients were included in the diabetic group, and 17 patients were included in the non-diabetic control group. Consecutive sampling technique was applied; it is a non-probability sampling method. All patients who met the inclusion criteria were included until they reached the desired sample size. The study included patients older than 18 years of age and of both sexes. The patients included in the diabetic group had a diagnosed diabetes mellitus and age matched individuals without diabetes were included in control group. Patients were eligible to be included and only those who agreed to do so and who could give informed consent were included. Patients who had previous eye surgery, contact lens use, an active eye infection, allergic conjunctivitis, corneal ulcers, corneal opacity, other autoimmune diseases or thyroid eye disease, glaucoma, topical eye medications, history of eye trauma or any other systemic disease that might affect tear film or corneal sensitivity were excluded. Patients unable to cooperate in eye examination were also excluded.

Data Collection

Demographic and clinical information were collected in a structured proforma, following informed consent. Data involved age, gender, diabetes type, length of diabetes, glycemic control status (where applicable), ocular symptoms, dry eye symptoms history, medication, and pertinent systemic and/or ocular history. Comprehensive ophthalmic examination was conducted on all participants, which involved visual acuity, slit-lamp examination, tear film assessment and corneal sensitivity test. Evaluation of tear film dysfunction was made by measures of tear breakup time, Schirmer test and ocular surface staining. The tear breakup time was timed following instillation of fluorescein dye, and the time interval from a complete blink to the first appearance of a dry spot on the cornea was timed in seconds. A short tear break up time was thought to be a sign of tear film instability. Standard Schirmer strips were placed in the lower conjunctival fornix for five minutes and the wetting level measured in millimeters (mm). Ocular surface epithelial damage was measured using fluorescein staining.The corneal nerves sensitivity was tested using a corneal esthesiometer, if available. Gently tested central cornea and the patient's response was recorded. Decreased corneal sensitivity was deemed as indicative of compromised corneal nerve sensitivity. In the absence of esthesiometry, corneal sensitivity was evaluated clinically, using a sterile cotton wisp, and the response graded as normal, reduced or absent. The main outcome measures were tear film dysfunction and the sensitivity of the corneal nerves. Tear film dysfunction was evaluated by measuring tear breakup time, Schirmer test, and fluorescein staining. The sensitivity of the cornea nerves was evaluated by corneal esthesiometry or clinical corneal sensitivity testing. The comparisons were made between diabetic and non-diabetic participants.

Data Analysis

Data were analyzed and entered SPSS (version 26.0). Means and standard deviations were used to present quantitative variables like age, duration of diabetes, tear break up time, Schirmer test value and corneal sensitivity score. Qualitative variables like gender, tear film dysfunction and decreased corneal sensation were described as frequency and percentage. Normally distributed quantitative variables were compared between the two groups (diabetic versus non-diabetic) using independent-samples t test, and the Mann–Whitney U test was used for non-normally distributed quantitative variables. Chi-square test or Fisher’s exact test was used for categorical variables. The p value of < 0.05 was used as the criterion for statistical significance.

RESULTS

Table 1 shows the baseline characteristics of the diabetic and non-diabetic groups. The diabetic group included 18 participants, while the non-diabetic group included 17 participants. The mean age was slightly higher in the diabetic group (54.6 ± 8.7 years) compared with the non-diabetic group (52.1 ± 9.3 years). Gender distribution was almost similar in both groups, with males comprising 55.6% of the diabetic group and 52.9% of the non-diabetic group, while females comprised 44.4% and 47.1%, respectively. The mean duration of diabetes among diabetic participants was 8.3 ± 4.6 years.

Table 1. Baseline Demographic and Clinical Characteristics of Participants

Variable	Diabetic Group (n = 18)	Non-Diabetic Group (n = 17)
Age, years	54.6 ± 8.7	52.1 ± 9.3
Male gender	10 (55.6%)	9 (52.9%)
Female gender	8 (44.4%)	8 (47.1%)
Duration of diabetes, years	8.3 ± 4.6	—
Fasting blood glucose, mg/dL	166.4 ± 38.2	94.3 ± 12.1
HbA1c, %	8.1 ± 1.4	5.4 ± 0.4

Tear breakup time was significantly lower in the diabetic group (6.1 ± 2.0 seconds) compared with the non-diabetic group (10.2 ± 2.7 seconds), with a mean difference of −4.1 seconds, showing greater tear film instability among diabetic patients ($t = -5.12$, $p < 0.001$). Schirmer test values were also significantly reduced in diabetic participants (8.4 ± 3.1 mm/5 min) compared with non-diabetic participants (14.0 ± 4.2 mm/5 min), with a mean difference of −5.6 mm/5 min, indicating reduced tear secretion in the diabetic group ($t = -4.50$, $p < 0.001$).

Table 2. Comparison of Tear Film Parameters Between Diabetic and Non-Diabetic Participants

Tear Film Parameter	Diabetic Group (n = 18)	Non-Diabetic Group (n = 17)	Mean Difference	Test Statistic	p-value
Tear breakup time, seconds	6.1 ± 2.0	10.2 ± 2.7	−4.1	$t = -5.12$	<0.001
Schirmer test, mm/5 min	8.4 ± 3.1	14.0 ± 4.2	−5.6	$t = -4.50$	<0.001
Fluorescein staining score	1.44 ± 0.92	0.47 ± 0.62	+0.97	$U = 55.0$	0.001

Reduced tear breakup time of less than 10 seconds was observed in 15 diabetic participants (83.3%) compared with 7 non-diabetic participants (41.2%), and this difference was statistically significant ($p = 0.010$). Reduced Schirmer test values of ≤10 mm/5 min were found in 13 diabetic patients (72.2%) compared with 5 non-diabetic participants (29.4%), showing significantly reduced tear production among diabetics ($p = 0.011$).

Table 3. Frequency of Tear Film Dysfunction According to Diagnostic Parameters

Tear Film Finding	Diabetic Group (n = 18)	Non-Diabetic Group (n = 17)	p-value
Reduced tear breakup time, <10 seconds	15 (83.3%)	7 (41.2%)	0.010
Normal tear breakup time, ≥10 seconds	3 (16.7%)	10 (58.8%)	
Reduced Schirmer test, ≤10 mm/5 min	13 (72.2%)	5 (29.4%)	0.011
Normal Schirmer test, >10 mm/5 min	5 (27.8%)	12 (70.6%)	
Positive fluorescein staining	12 (66.7%)	4 (23.5%)	0.010
No fluorescein staining	6 (33.3%)	13 (76.5%)	
Overall tear film dysfunction present	14 (77.8%)	5 (29.4%)	0.004
Overall tear film dysfunction absent	4 (22.2%)	12 (70.6%)	

The mean corneal sensitivity was 4.1 ± 0.9 cm in the diabetic group compared with 5.5 ± 0.5 cm in the non-diabetic group, showing significantly impaired corneal sensory function in diabetic patients ($t = -5.72$, $p < 0.001$). Reduced corneal sensitivity was observed in 11 diabetic participants (61.1%) compared with only 2 non-diabetic participants (11.8%), while normal corneal sensitivity was present in 7 diabetic participants (38.9%) and 15 non-diabetic participants (88.2%).

Table 4. Comparison of Corneal Nerve Sensitivity Between Diabetic and Non-Diabetic Participants

Corneal Sensitivity Parameter	Diabetic Group (n = 18)	Non-Diabetic Group (n = 17)	Test Statistic	p-value
Corneal sensitivity, cm	4.1 ± 0.9	5.5 ± 0.5	$t = -5.72$	<0.001
Reduced corneal sensitivity	11 (61.1%)	2 (11.8%)	$\chi^2 = 8.99$	0.003

Normal corneal sensitivity	7 (38.9%)	15 (88.2%)	$\chi^2 = 8.99$	0.003

Tear film dysfunction was significantly more severe among diabetic participants (p = 0.006). In the diabetic group, only 4 participants (22.2%) had no dysfunction, while 5 (27.8%) had mild dysfunction, 6 (33.3%) had moderate dysfunction, and 3 (16.7%) had severe dysfunction. In contrast, most non-diabetic participants had no tear film dysfunction, with 12 participants (70.6%) showing no dysfunction, 3 (17.6%) having mild dysfunction, 2 (11.8%) having moderate dysfunction, and none having severe dysfunction.

Table 5. Severity Distribution of Tear Film Dysfunction and Corneal Sensitivity

Severity Category	Diabetic Group (n = 18)	Non-Diabetic Group (n = 17)	p-value
Tear Film Dysfunction			0.006
No dysfunction	4 (22.2%)	12 (70.6%)	
Mild dysfunction	5 (27.8%)	3 (17.6%)	
Moderate dysfunction	6 (33.3%)	2 (11.8%)	
Severe dysfunction	3 (16.7%)	0 (0.0%)	
Corneal Sensitivity			0.004
Normal sensitivity	7 (38.9%)	15 (88.2%)	
Mildly reduced sensitivity	5 (27.8%)	2 (11.8%)	
Moderately reduced sensitivity	4 (22.2%)	0 (0.0%)	
Severely reduced sensitivity	2 (11.1%)	0 (0.0%)	

DISCUSSION

The current investigation aimed to make a comparison between patients with diabetes mellitus and non-diabetic control group regarding measures of tear film dysfunction and corneal nerve sensitivity. The results revealed that there were significant differences in tear film parameters between diabetic and non-diabetic cases such as tear breakup time, Schirmer test scores and fluorescein staining scores. Diabetic patients also had more tear film dysfunction than non-diabetic patients. Moreover, the sensitivity of the corneal nerves was significantly decreased in diabetic patients, suggesting that diabetic patients have decreased corneal sensory function. The results indicated that DM not only involves the posterior segment of the eye, but also the surface of the eye and corneal nerves. Diabetic patients have a shorter tear break-up time which suggests increased tear film instability. This discovery is clinically relevant as one of the major symptoms of dry eye disease is tear film instability. The TFOS DEWS II definition of dry eye disease includes disruption of tear film homeostasis, instability of tear film, inflammation, ocular surface damage, and neurosensory abnormalities. Thus, the shorter tear break-up time and higher fluorescein staining in diabetic patients is supportive of the presence of Ocular Surface Dysfunction in this group of patients [13].

In diabetics, the lower values of the Schirmer test indicate decreased aqueous tear production. This can happen because of lacrimal gland dysfunction secondary to diabetes, autonomic neuropathy and decreased reflex tearing. With chronic hyperglycemia, the lacrimal functional unit may suffer damage, tear secretion may change and inflammation of the ocular surface may occur [14]. Similarly, the previous literature reports that diabetes mellitus is often associated with a decrease in tear secretion and tear breakup time in diabetic patients when compared with nondiabetic patients. Results show that diabetic patients had a higher fluorescein staining score, reflecting higher damage to the ocular surface epithelium [15]. This could be due to: tear deficiency, instability of the tear film, decreased corneal sensation, delayed epithelial healing and inflammatory effects of diabetes. Several corneal diabetic complications may include superficial punctate keratopathy, prolonged epithelial defects, recurrent corneal erosions, and neurotrophic changes. These alterations render the diabetic cornea more susceptible to the damage of the corneal epithelium and impaired healing [16].

One of the important results from the present study was that diabetic patients had decreased corneal nerve sensitivity. The cornea is very richly innervated and normal corneal sensation is necessary for blinking, tears, maintenance of the cornea's epithelial surfaces, and wound healing. Peripheral neuropathy in diabetes mellitus can affect the cornea's nerves and lead to decreased corneal sensitivity and poor corneal surface protection [17]. Previous studies indicate that the corneal sensitivity and tear film function can be reduced early even in the absence of ocular symptoms in diabetic patients, especially those with diabetic neuropathy. The change in tear film dysfunction and the sensitivity of the cornea is particularly significant. A decrease in corneal sensitivity may lead to decreased reflex blinking and reflex tear production, also contributing to tear film instability [18]. Concurrently, the poor tear film quality may cause corneal irritation and epithelial changes. This forms a vicious cycle of diabetes-related nerve damage and tear film dysfunction [19,20]. In the present findings, it was observed that diabetic patients were found to have decreased tear film function as well as decreased corneal sensitivity supporting this. Patients with diabetes should be assessed regularly for tear break up time, Schirmer test, staining of the ocular surface and corneal sensitivity to determine if there is early involvement of the ocular surface prior to the onset of serious symptoms or complications. There were some limitations in this study. The number of participants was small (n = 35) and this could restrict the ability to generalise the findings. The cross-sectional design also did not allow for the assessment of causal relationships. Furthermore, corneal nerve sensitivity was

evaluated by clinical examination or esthesiometry but not using more sophisticated imaging technology such as in vivo confocal microscopy. Large-scale, multi-center studies with more follow-up periods, with thorough classification of the degree of diabetes and objective imaging of the corneal nerves would give increased evidenc.

CONCLUSION

The present study concluded that patients with diabetes mellitus had significantly greater tear film dysfunction and reduced corneal nerve sensitivity compared with non-diabetic individuals. Diabetic patients showed lower tear breakup time, reduced Schirmer test values, higher fluorescein staining scores, and a higher frequency of tear film dysfunction, indicating impaired tear stability, reduced tear secretion, and greater ocular surface damage. Corneal sensitivity was also markedly reduced in diabetic patients, suggesting diabetes-related corneal nerve involvement. Longer duration of diabetes and poor glycemic control were associated with worsening tear film parameters and reduced corneal sensitivity.

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