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ORIGINAL ARTICLE

To study the ocular surface changes in patients of diabetes mellitus

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ABSTRACT:

Aim: To study and evaluate the ocular surface changes in patients with diabetes mellitus and identify the correlation between glycemic control, disease duration, and ocular surface parameters. **Materials and Methods:** This prospective, observational study included 100 diabetic patients, aged 30–80 years, recruited from a tertiary care hospital. Patients with a history of ocular trauma, recent ocular surgery, or active ocular infections were excluded. Clinical history, including diabetes duration, glycemic control (HbA1c levels), and dry eye symptoms (Ocular Surface Disease Index - OSDI questionnaire), was recorded. Ophthalmic examinations included Tear Film Break-Up Time (TBUT), Schirmer's Test, Corneal Fluorescein Staining, and Conjunctival Impression Cytology. Data were analyzed to identify associations between diabetes-related factors and ocular surface changes. A p-value of <0.05 was considered statistically significant. **Results:** The mean age was 55.8 ± 10.2 years, with a male predominance (58% males, 42% females). The mean HbA1c was 8.1 ± 1.5%, reflecting poor glycemic control. 60% of patients reported moderate-to-severe dry eye symptoms based on OSDI scores. 68% had abnormal TBUT, while 55% had abnormal Schirmer's test results, indicating impaired tear production and stability. 40% of patients demonstrated moderate-to-severe corneal epithelial damage through fluorescein staining. Conjunctival impression cytology revealed 45% reduced goblet cell density and 25% squamous metaplasia, indicating chronic ocular surface inflammation. **Conclusion:** Ocular surface changes, including tear film instability, corneal damage, and conjunctival epithelial abnormalities, are common in diabetic patients and are significantly associated with poor glycemic control and disease duration. Early detection and comprehensive management are essential to prevent long-term complications and improve ocular health outcomes in diabetic patients.

Keywords: Diabetes Mellitus, Ocular Surface Disease, Tear Film Instability, Corneal Staining, Impression Cytology.

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INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. It is a global health concern with increasing prevalence, affecting millions worldwide. While diabetes is widely recognized for its complications involving major organs such as the heart, kidneys, and nervous system, its impact on ocular health is often overlooked. Among the various ocular complications of diabetes, including diabetic retinopathy, cataract, and glaucoma, ocular surface disorders remain under-recognized and underdiagnosed despite their significant impact on the patient's quality of life.[1]The ocular surface is a highly specialized and integrated system composed of the cornea, conjunctiva, tear film, eyelids, and associated glands, including the lacrimal and meibomian glands. This system works in harmony to maintain the transparency of the cornea, support clear vision, and protect the eye from environmental insults and infections. A stable tear film, produced and regulated by the ocular surface, is essential for preserving corneal integrity and ensuring optimal visual function. In diabetes, chronic hyperglycemia leads to multiple physiological and structural changes in these components, resulting in a range of ocular surface disorders collectively referred to as diabetic ocular surface disease.[2]Hyperglycemia-induced damage is multifactorial, involving neuropathy, microvascular dysfunction, and chronic inflammation. Diabetic neuropathy affects the corneal nerves, impairing corneal sensitivity and reducing reflex tear production. This decreased sensitivity leads to inadequate blinking and incomplete eyelid closure, further compromising tear distribution across the ocular surface. Additionally, autonomic neuropathy affects the lacrimal and meibomian glands, resulting in decreased aqueous tear production and lipid layer dysfunction, both of which are essential for tear film stability. These changes create an unstable tear film, predisposing diabetic patients to dry eye disease, corneal epithelial damage, and delayed wound healing.[3] Moreover, diabetes alters the composition of the tear film, with studies reporting reduced tear production, lower tear film break-up time, and increased osmolarity. The imbalance in tear film homeostasis creates a hyperosmolar environment on the ocular surface, triggering inflammatory pathways and leading to cellular damage. The conjunctival epithelium, which plays a critical role in tear film stability through goblet cell production, is also adversely affected in diabetes. Goblet cell density decreases significantly in diabetic patients, resulting in reduced mucin production and further tear film instability.Corneal complications are another hallmark of diabetic ocular surface disease. The cornea, being

the most innervated structure of the human body, is highly susceptible to damage from diabetic neuropathy. Chronic hyperglycemia disrupts the corneal epithelium's ability to heal after injury, increasing the risk of persistent corneal epithelial defects, recurrent corneal erosions, and infectious keratitis. Furthermore, diabetic keratopathy, a condition characterized by corneal epithelial abnormalities, stromal edema, and endothelial dysfunction, is frequently observed in diabetic patients.[4] In addition to structural changes, diabetic patients often experience significant symptomatic discomfort, including dryness, foreign body sensation, burning, redness, and blurred vision. These symptoms can vary in severity and are often correlated with the duration of diabetes and the level of glycemic control. Poorly controlled diabetes has been shown to exacerbate ocular surface damage, underscoring the importance of systemic disease management in preventing ocular complications.Impression cytology studies have provided further insight into cellular changes on the ocular surface in diabetes. These studies have demonstrated squamous metaplasia and reduced goblet cell density, both of which are indicative of chronic ocular surface inflammation and dysfunction. These cellular changes, combined with tear film instability and corneal nerve damage, create a vicious cycle of inflammation and impaired healing on the ocular surface.[5] Early diagnosis and management of ocular surface disease in diabetic patients are essential to prevent long-term complications and improve their quality of life. However, ocular surface changes in diabetes are often underdiagnosed due to their subtle presentation and the focus on more severe diabetic complications such as retinopathy. Routine ocular surface evaluation, including tear film analysis, corneal fluorescein staining, and impression cytology, can aid in the early detection of ocular surface abnormalities in diabetic patients.[6] The management of diabetic ocular surface disease requires a multidisciplinary approach involving tight glycemic control, artificial tear substitutes, anti-inflammatory therapies, and patient education regarding ocular hygiene and environmental modifications. Emerging treatments, such as nerve growth factors and advanced tear substitutes, hold promise for improving ocular surface outcomes in diabetic patients.[7] The pathophysiology involves a complex interplay of neuropathy, tear film dysfunction, corneal epithelial damage, and conjunctival changes. Given its impact on the quality of life and potential for vision-threatening complications, ocular surface changes in diabetes warrant increased attention from both endocrinologists and ophthalmologists. Early detection, preventive strategies, and targeted therapies are crucial in mitigating the burden of ocular surface disease in diabetic patients.

MATERIALS AND METHODS

This prospective, observational study was conducted to evaluate ocular surface changes in patients with diabetes mellitus. A total of 100 diabetic patients, aged between 30 and 80 years, were recruited from the ophthalmology outpatient department of a tertiary care hospital over a period of six months. Inclusion criteria included patients with a confirmed diagnosis of diabetes mellitus (Type 1 or Type 2) for at least one year, irrespective of glycemic control status. Exclusion criteria included patients with a history of ocular trauma, ocular surgery in the past six months, active ocular infections, autoimmune disorders affecting the ocular surface, or the use of topical medications affecting tear production. After obtaining informed consent, a detailed history was recorded, including duration of diabetes, glycemic control (assessed through HbA1c levels), systemic medications, and symptoms of dry eye disease using the Ocular Surface Disease Index (OSDI) questionnaire.

All patients underwent a comprehensive ophthalmic examination, including visual acuity assessment, slit-lamp biomicroscopy, and fundus examination. Ocular surface evaluation included tear film break-up time (TBUT), Schirmer's test, corneal fluorescein staining, and conjunctival impression cytology. TBUT was performed using fluorescein dye to measure the time taken for the first dry spot to appear on the tear film after blinking. Schirmer's test without anesthesia was conducted to measure baseline tear production. Corneal fluorescein staining was graded according to a standardized scale to assess epithelial damage. Impression cytology samples were collected from the superior bulbar conjunctiva to evaluate goblet cell density and epithelial morphology. The results were analyzed to determine the correlation between diabetes duration, glycemic control, and ocular surface changes. Statistical analysis was performed using appropriate software, and a p-value of less than 0.05 was considered statistically significant. The study adhered to the ethical guidelines of the Declaration of Helsinki, and institutional ethical committee approval was obtained prior to commencement.

RESULTS

Table 1: Demographic and Clinical Characteristics of Study Participants

The study included 100 diabetic patients, with a mean age of 55.8 ± 10.2 years, reflecting a middle-aged population commonly affected by diabetes-related ocular complications. Out of the total participants, 58% were male and 42% were female, showing a slight male predominance in the sample. The mean duration of diabetes was 8.5 ± 4.3 years, indicating a significant disease burden over time. Regarding diabetes type, the majority of participants (82%) had Type 2 diabetes, while only 18% had Type 1 diabetes. The mean HbA1c level was 8.1 ± 1.5%, suggesting

poor glycemic control across the study population. The mean OSDI (Ocular Surface Disease Index) score was 28.4 ± 6.2, indicating the presence of moderate dry eye symptoms on average. These demographic and clinical findings highlight that the study population predominantly comprised middle-aged patients with long-standing and poorly controlled Type 2 diabetes, factors that are strongly associated with ocular surface disorders.

Table 2: Distribution of Ocular Surface Disease Index (OSDI) Scores

The OSDI questionnaire assessed the severity of dry eye symptoms among participants. 15% of patients had a normal OSDI score (0–12), indicating no significant dry eye symptoms. 25% reported mild symptoms (13–22), while 35% exhibited moderate symptoms (23–32). Additionally, 25% of patients experienced severe dry eye symptoms (OSDI >32). These results suggest that a significant proportion (60%) of diabetic patients suffer from moderate-to-severe dry eye disease, highlighting the burden of ocular surface symptoms in this population. The OSDI results also underscore the importance of routine screening and management of dry eye symptoms in diabetic patients to prevent further ocular complications.

Table 3: Tear Film Break-Up Time (TBUT) and Schirmer's Test Results

The stability and production of the tear film were assessed using TBUT and Schirmer's test. The mean TBUT was 7.2 ± 2.1 seconds, which is below the normal threshold of 10 seconds, indicating tear film instability. Abnormal TBUT results were observed in 68% of patients, reflecting a high prevalence of tear film dysfunction. Similarly, the mean Schirmer's test result was 8.5 ± 3.4 mm, with abnormal results observed in 55% of patients, indicating reduced tear production. These findings suggest that both qualitative and quantitative tear film abnormalities are common in diabetic patients, contributing significantly to ocular surface changes and discomfort.

Table 4: Corneal Fluorescein Staining Grading

Corneal fluorescein staining was performed to assess epithelial damage on the ocular surface. 22% of patients showed no staining (Grade 0), suggesting a healthy corneal surface. However, 38% had mild staining (Grade 1), while 30% had moderate staining (Grade 2), and 10% had severe staining (Grade 3). These findings indicate that 40% of patients had moderate-to-severe corneal epithelial damage, reflecting advanced ocular surface pathology. Corneal damage in diabetic patients is likely associated with reduced tear stability, impaired epithelial healing, and chronic hyperglycemia, emphasizing the need for targeted interventions.

Table 5: Conjunctival Impression Cytology Results

Conjunctival impression cytology revealed significant changes in the conjunctival epithelium. 30% of patients had normal epithelial morphology, indicating no significant damage. However, 45% showed reduced goblet cell density, which is a key marker of dry eye disease and ocular surface dysfunction. Additionally, 25% demonstrated squamous metaplasia, indicating chronic inflammation and cellular changes in the conjunctiva. These cytological findings suggest that a large proportion (70%) of diabetic patients exhibit structural and functional abnormalities in the conjunctival epithelium, contributing to tear film instability and persistent dry eye symptoms.

Table 1: Demographic and Clinical Characteristics of Study Participants

Parameter	Mean ± SD	Number	Percentage (%)
Age (years)	55.8 ± 10.2	-	-
Gender (Male)	-	58	58%
Gender (Female)	-	42	42%
Duration of Diabetes (years)	8.5 ± 4.3	-	-
Type of Diabetes (Type 1)	-	18	18%
Type of Diabetes (Type 2)	-	82	82%
HbA1c (%)	8.1 ± 1.5	-	-
OSDI Score (Mean ± SD)	28.4 ± 6.2	-	-

Table 2: Distribution of Ocular Surface Disease Index (OSDI) Scores

OSDI Score Range	Severity Category	Number of Patients	Percentage (%)
0–12	Normal	15	15%
13–22	Mild	25	25%
23–32	Moderate	35	35%
>32	Severe	25	25%

Table 3: Tear Film Break-Up Time (TBUT) and Schirmer's Test Results

Parameter	Mean ± SD	Number Abnormal	Percentage Abnormal (%)
TBUT (seconds)	7.2 ± 2.1	68	68%
Schirmer's Test (mm)	8.5 ± 3.4	55	55%

Table 4: Corneal Fluorescein Staining Grading

Staining Grade	Description	Number of Patients	Percentage (%)
Grade 0	No staining	22	22%
Grade 1	Mild staining	38	38%
Grade 2	Moderate staining	30	30%
Grade 3	Severe staining	10	10%

Table 5: Conjunctival Impression Cytology Results

Cytology Finding	Number of Patients	Percentage (%)
Normal Morphology	30	30%
Reduced Goblet Cell Density	45	45%
Squamous Metaplasia	25	25%

DISCUSSION

The present study aimed to evaluate ocular surface changes in diabetic patients and identified significant abnormalities across various parameters, including demographic data, tear film stability, corneal integrity, and conjunctival morphology. In our study, the mean age of participants was 55.8 ± 10.2 years, with a slight male predominance (58% males, 42% females). The majority had Type 2 diabetes (82%), with poor glycemic control reflected by an HbA1c of 8.1 ± 1.5%. These findings are consistent with the study by Manaviat et al. (2010), where diabetic patients had a mean age of 56.4 ± 11.3 years, and Type 2 diabetes was predominant. They also reported poor glycemic control as a significant factor contributing to ocular surface disease. These demographic similarities suggest that age, type of diabetes, and glycemic control are important risk factors for ocular surface abnormalities in diabetic populations (Manaviat et al., 2010).[8] Our results revealed that 60% of patients experienced moderate-to-severe dry eye symptoms based on OSDI scores. This finding aligns with the study conducted by Seifart and Strempel (2009), where 57% of diabetic patients reported moderate-to-severe dry eye symptoms. Both studies emphasize the significant burden of symptomatic dry eye disease in diabetics and highlight the importance of routine screening using standardized questionnaires like OSDI to detect and manage ocular surface changes early (Seifart& Strempel, 2009).[9] The mean TBUT in our study was 7.2 ± 2.1 seconds, with 68% showing abnormal results. Similarly, the mean Schirmer's test value was 8.5 ± 3.4 mm, with 55% showing reduced tear production. These results are in line with the findings of Dogru et al. (2010), who reported significantly reduced TBUT and Schirmer's test values in diabetic patients compared to controls. The high prevalence of tear film abnormalities in both studies suggests that diabetes affects both the quality and quantity of tear production, likely due to autonomic neuropathy and meibomian gland dysfunction (Dogru et al., 2010).[10] In our study, 40% of patients exhibited moderate-to-severe corneal fluorescein staining, indicating significant corneal epithelial damage. This finding is supported by the results of Kumar et al. (2012), who reported a 38% prevalence of moderate-to-severe corneal staining among diabetic patients. Chronic hyperglycemia has been shown to impair corneal epithelial healing, contributing to persistent damage observed in both studies. These findings highlight the need for frequent corneal assessments in diabetic patients to prevent severe ocular complications (Kumar et al., 2012).[11] Conjunctival impression cytology in our study showed that 45% of patients had reduced goblet cell density, while 25% demonstrated squamous metaplasia, indicating chronic conjunctival damage and inflammation. A study by Yoon et al. (2011) similarly reported a high prevalence of goblet cell loss and squamous metaplasia in diabetic patients. Both studies suggest that prolonged hyperglycemia and ocular surface inflammation lead to significant structural changes in the conjunctiva, further contributing to dry eye disease and ocular discomfort (Yoon et al., 2011).[12]

CONCLUSION

In conclusion, ocular surface changes are a significant yet often overlooked complication of diabetes mellitus, resulting from a complex interplay of neuropathy, tear film dysfunction, corneal epithelial damage, and conjunctival alterations. These changes contribute to symptoms of dry eye, impaired vision, and increased susceptibility to corneal injuries and infections. Early detection through regular ocular surface evaluations and a multidisciplinary approach involving glycemic control, tear substitutes, and anti-inflammatory therapies are essential to prevent long-term complications and improve the quality of life for diabetic patients. Prioritizing ocular surface health in diabetes management is crucial for preserving visual function and overall ocular well-being.

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