International Journal of Ophthalmology & Visual Science
2026, Vol. 11, No. 1, pp. 1–8
https://doi.org/10.11648/j.ijovs.20261101.11

Research Article

Assessment of Meibomian Gland Function and Tear Film Stability in Type 2 Diabetic Patients

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Abstract

Background: Type 2 Diabetes Mellitus (T2DM) is frequently associated with ocular surface disorders, including dry eye disease (DED) and meibomian gland dysfunction (MGD). Chronic hyperglycemia, systemic inflammation, and autonomic neuropathy may disrupt meibomian gland morphology and tear film composition, contributing to both evaporative and aqueous-deficient dry eye. Understanding these alterations is essential for early detection and intervention in diabetic eye care. *Aim:* To evaluate meibomian gland function and tear film stability in patients with T2DM and compare these parameters with age- and sex-matched healthy individuals. *Methods:* This cross-sectional study was conducted at Vision Eye Hospital, Dhaka, Bangladesh, from June 2024 to May 2025. A total of 260 participants were enrolled, comprising 130 T2DM patients (diagnosed per ADA/WHO criteria) and 130 healthy controls. Ethical approval was obtained from the Institutional Review Board, and informed consent was taken from all participants. Data collection included structured ocular history, the Ocular Surface Disease Index (OSDI) questionnaire, and clinical tests such as tear break-up time (TBUT), non-invasive TBUT (NIBUT), Schirmer I test, tear meniscus height (TMH), and non-contact meibography. Statistical analysis was performed using SPSS version 26.0; independent t-tests and chi-square tests were applied, with significance set at $p < 0.05$. *Results:* T2DM patients demonstrated significantly worse ocular surface parameters compared to controls. OSDI scores were higher in diabetics (34.6 ± 12.1 vs. 15.3 ± 7.8; $p < 0.001$), indicating more severe symptoms. Tear film stability was reduced, with lower TBUT (6.1 ± 2.2 s) and NIBUT (5.8 ± 2.4 s) compared to controls (10.3 ± 2.5 s and 11.2 ± 2.7 s; both $p < 0.001$). Aqueous tear production was impaired, reflected by lower Schirmer I values (9.4 ± 3.8 mm vs. 15.6 ± 4.2 mm; $p < 0.001$) and reduced TMH (0.18 ± 0.05 mm vs. 0.25 ± 0.06 mm; $p < 0.001$). Meiboscores were significantly higher in the diabetic group (3.7 ± 1.2 vs. 1.8 ± 0.9; $p < 0.001$), indicating greater gland dropout. HbA1c and duration of diabetes correlated significantly with meiboscore and tear film parameters ($r = –0.41$ and +0.43; $p < 0.01$). *Conclusion:* T2DM is significantly associated with both meibomian gland dysfunction and tear film abnormalities. Early ocular surface assessment in diabetic patients is recommended to detect subclinical changes and prevent visual discomfort and quality-of-life impairment.

Keywords

Type 2 Diabetes Mellitus, Meibomian Gland Dysfunction, Tear Film Stability, Dry Eye Disease, Ocular Surface, Schirmer Test

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Received: 15 December 2025; **Accepted:** 26 December 2025; **Published:** 20 January 2026

1. Introduction

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder that affects over 500 million people worldwide, with rising prevalence in low- and middle-income countries, particularly in South Asia [1]. While much attention has been given to retinal complications such as diabetic retinopathy, increasing evidence indicates that diabetes also adversely affects the ocular surface, contributing to disorders such as dry eye disease (DED) and meibomian gland dysfunction (MGD) [2, 3].

DED is a multifactorial disease of the ocular surface characterized by loss of tear film homeostasis, tear film instability, and ocular surface inflammation, leading to symptoms such as dryness, burning, and visual disturbance [4]. The tear film comprises lipid, aqueous, and mucin layers, with the lipid layer produced by the meibomian glands playing a vital role in reducing tear evaporation and maintaining film stability [5]. MGD, defined as a chronic, diffuse abnormality of the meibomian glands, often results in altered meibum quality or quantity, terminal duct obstruction, gland atrophy, and tear film disruption, making it the leading cause of evaporative dry eye [6].

Several mechanisms have been proposed to explain how T2DM contributes to MGD, including chronic hyperglycemia-induced oxidative stress, subclinical inflammation, and microvascular changes that impair gland function and structure [7, 8]. Recent clinical and imaging studies have revealed that patients with T2DM often exhibit significant morphological and functional alterations in the meibomian glands. Yang et al. reported a higher degree of meibomian gland dropout and lower tear production in T2DM patients with DED compared to both non-diabetic DED patients and healthy individuals [9]. Similarly, Yu et al., using confocal microscopy, demonstrated reduced acinar density, increased acinar size, and significantly shortened non-invasive tear breakup time (NIBUT) in diabetic patients, indicating meibomian gland compromise at both structural and cellular levels [10]. Hao et al. further confirmed that long-standing diabetes correlates with greater meibomian gland loss and lipid layer thinning [11].

Despite these findings, gaps remain in understanding how diabetes influences ocular surface parameters in different populations. Few studies have comprehensively assessed both tear film stability and meibomian gland morphology in South Asian cohorts, where the burden of diabetes is increasing rapidly. Therefore, this study aims to evaluate the function and morphology of meibomian glands, along with tear film parameters, in patients with T2DM in Bangladesh, and to compare the findings with age- and sex-matched healthy controls. Clarifying these associations may facilitate earlier diagnosis of subclinical DED, improve clinical management, and guide preventive strategies in diabetic eye care.

2. Materials and Methods

2.1. Study Design and Setting

This observational, cross-sectional study was conducted at Vision Eye Hospital, Dhaka, Bangladesh, over a 12-month period from June 2024 to May 2025.

2.2. Study Population

A total of 260 participants were enrolled and divided into two equal groups:

1) Cases: 130 patients diagnosed with Type 2 Diabetes Mellitus (T2DM), based on criteria established by the American Diabetes Association (ADA) or World Health Organization (WHO).
2) Controls: 130 age- and sex-matched healthy individuals with no history of diabetes or systemic illness.

2.3. Selection Criteria

Inclusion Criteria:

1) Age between 40 and 70 years
2) For cases: Confirmed diagnosis of T2DM for at least one year
3) For controls: Normal fasting blood glucose and HbA1c levels, no systemic or ocular disease
4) Ability and willingness to provide informed consent

Exclusion Criteria:

1) History of contact lens wear within the past six months
2) Ocular trauma or prior ocular surgery
3) Active ocular infection or inflammation (e.g., blepharitis, conjunctivitis)
4) Autoimmune disease (e.g., Sjögren's syndrome)
5) Use of systemic or topical medications affecting tear production or ocular surface
6) Any other condition known to alter tear film or meibomian gland function

2.4. Ethical Considerations

The study received approval from the Institutional Review Board of Vision Eye Hospital, Dhaka. All procedures were conducted in accordance with the Declaration of Helsinki, and informed written consent was obtained from each participant.

2.5. Study Variables

Independent Variable:

Presence or absence of Type 2 Diabetes Mellitus.

Dependent Variables:

1) Subjective symptoms: Ocular Surface Disease Index (OSDI) score
2) Tear film stability: Tear film break-up time (TBUT),

Non-invasive TBUT (NIBUT)
3) Tear secretion: Schirmer I test (mm)
4) Tear volume: Tear meniscus height (TMH)
5) Meibomian gland morphology: Meiboscore (upper and lower lids, total 0–6)
6) Duration of diabetes (for case group)
7) HbA1c levels (glycemic control, for correlation analysis)

Confounding Variables:

Age, sex, environmental exposure, screen time, systemic medications (excluded via criteria).

2.6. Data Collection Procedures

Participants underwent a standardized ocular examination by a trained ophthalmologist. OSDI questionnaires were completed to evaluate dry eye symptoms. TBUT was measured using slit-lamp biomicroscopy with fluorescein dye, while NIBUT and TMH were assessed with the Keratograph 5M (Oculus, Germany). The Schirmer I test (without anesthesia) evaluated aqueous tear production. Meibomian gland structure was examined via non-contact infrared meibography, and gland loss was graded using a standardized meiboscore (0–3 per lid).

2.7. Statistical Analysis

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize demographic and clinical variables. Independent sample t-tests and chi-square tests were applied to compare continuous and categorical data, respectively, between the diabetic and control groups. Pearson correlation was used to evaluate associations between HbA1c, duration of diabetes, and ocular surface parameters. A p-value <0.05 was considered statistically significant.

3. Results

Table 1. *Demographic and clinical characteristics of the study participant (n=260).*

Characteristic	T2DM Group (n = 130)	Control Group (n = 130)	p-value
Age (years)	58.2 ± 7.6	57.5 ± 7.3	0.45
Sex			
Male	68(52.3%)	70(53.9%)	0.80
Female	62(47.8%)	60(46.2%)	
Duration of diabetes (yrs)	8.4 ± 4.1	—	—
HbA1c (%)	8.2 ± 1.3	—	—

p-value obtained by Unpaired t-test and Chi-square test, p<0.05 was considered as a level of *significant

Table 1 shows the demographic and clinical profiles of the study participants. The mean age of the T2DM group was 58.2 ± 7.6 years, which was comparable to the control group (57.5 ± 7.3 years), with no statistically significant difference (p = 0.45). The distribution of sex was also similar between groups, with males representing 52.3% of the T2DM group and 53.9% of the control group (p = 0.80), confirming successful age- and sex-matching. The mean duration of diabetes in the T2DM group was 8.4 ± 4.1 years, and the average HbA1c level was 8.2 ± 1.3%, indicating suboptimal glycemic control.

Table 2. *Comparison of Ocular Surface Disease Index (OSDI) Scores between T2DM and Control Groups (n=260).*

Group	T2DM Group (n = 130)	Control Group (n = 130)	p-value
Mean OSDI Score	34.6± 12.1	15.3± 7.8	< 0.001 *

p-value obtained by Unpaired t-test, p<0.05 was considered as a level of *significant

Table 2 presents the comparison of OSDI scores between the two groups. The T2DM group demonstrated significantly higher mean OSDI scores (34.6 ± 12.1) compared to the control group (15.3±7.8), with a p-value of <0.001.

Table 3. *Comparison of tear film stability: TBUT and NIBUT between two groups (n=260).*

Parameter	T2DM Group (n = 130)	Control Group (n = 130)	p-value
TBUT (seconds)	6.1 ± 2.2	10.3 ± 2.5	< 0.001 *
NIBUT (seconds)	5.8 ± 2.4	11.2 ± 2.7	< 0.001 *

p-value obtained by Unpaired t-test, p<0.05 was considered as a level of *significant

Table 3 shows significant differences in tear film stability between diabetic and non-diabetic participants. The mean TBUT in the T2DM group was 6.1 ± 2.2 seconds, markedly shorter than the control group (10.3 ± 2.5 seconds), with a highly significant p-value (<0.001). Similarly, NIBUT was significantly reduced in diabetic patients (5.8 ± 2.4 seconds) compared to controls (11.2 ± 2.7 seconds).

Table 4. *Comparison of tear secretion and tear volume in T2DM and Control Groups (n=260).*

Parameter	T2DM Group (n = 130)	Control Group (n = 130)	p-value
Schirmer I test (mm)	9.4 ± 3.8	15.6 ± 4.2	< 0.001 *
TMH (mm)	0.18 ± 0.05	0.25 ± 0.06	< 0.001 *

p-value obtained by Unpaired t-test, p<0.05 was considered as a level of *significant

Table 4 compares tear secretion and volume between groups. The T2DM group had significantly reduced Schirmer I test values (9.4 ± 3.8 mm) compared to controls (15.6 ± 4.2 mm), indicating impaired aqueous tear production (p < 0.001). Similarly, tear meniscus height (TMH) was significantly lower in diabetic patients (0.18 ± 0.05 mm) than in healthy individuals (0.25 ± 0.06 mm).

Table 5. *Comparison of Meibomian Gland Morphology (Meiboscore) between two groups (n=260).*

Eyelid	T2DM Group (n = 130)	Control Group (n = 130)	p-value
Upper lid score	1.8 ± 0.6	0.9 ± 0.4	< 0.001 *
Lower lid score	1.9 ± 0.7	0.9 ± 0.5	< 0.001 *
Total score (0–6)	3.7 ± 1.2	1.8 ± 0.9	< 0.001 *

p-value obtained by Unpaired t-test, p<0.05 was considered as a level of *significant

Table 5 outlines the meiboscore values, reflecting meibomian gland dropout severity. Diabetic patients exhibited significantly higher scores in both upper (1.8 ± 0.6) and lower eyelids (1.9 ± 0.7) than controls (0.9 ± 0.4 and 0.9 ± 0.5, respectively), with a total mean meiboscore of 3.7 ± 1.2 in T2DM versus 1.8 ± 0.9 in controls (p < 0.001).

Table 6. *Correlation analysis between diabetic parameters and ocular surface findings.*

Correlation	r-value	p-value
HbA1c vs. TBUT	–0.41	< 0.01*

Correlation	r-value	p-value
HbA1c vs. Meiboscore	+0.39	0.02*
Duration of diabetes vs. Meiboscore	+0.43	< 0.01*
OSDI vs. Schirmer test	–0.35	0.03*

p-value obtained by Pearson Correlation test, p<0.05 was considered as a level of *significant

Table 6 presents correlation coefficients between diabetic status indicators and ocular surface parameters. There was a moderate negative correlation between HbA1c levels and TBUT ($r = –0.41$, $p < 0.01$), and a positive correlation between HbA1c and total meiboscore ($r = 0.39$, $p = 0.02$), indicating that poorer glycemic control is associated with worse tear stability and greater meibomian gland loss. Additionally, duration of diabetes correlated positively with meiboscore ($r = 0.43$, $p < 0.01$), and OSDI scores showed a negative correlation with Schirmer values ($r = –0.35$, $p = 0.03$).

4. Discussion

This observational, cross-sectional study was conducted at Vision Eye Hospital in Dhaka, Bangladesh, over a 12-month period from June 2024 to May 2025. A total of 260 participants were enrolled and divided equally into two groups: 130 patients with Type 2 Diabetes Mellitus (T2DM), diagnosed according to the American Diabetes Association (ADA) or World Health Organization (WHO) criteria, and 130 age- and sex-matched healthy individuals without any history of diabetes or systemic illness. The primary aim of the study was to assess the functional status of the Meibomian glands and the stability of the tear film in T2DM patients and to compare these ocular surface parameters with those of healthy controls.

In this study, no significant differences were observed in age ($p = 0.45$) or sex distribution ($p = 0.80$) between the T2DM and control groups, allowing unbiased comparison of ocular surface parameters. The diabetic group had a mean disease duration of 8.4 ± 4.1 years and a mean HbA1c of 8.2 ± 1.3%, indicating poor glycemic control. These findings align with previous studies showing that long-standing T2DM and elevated HbA1c are associated with ocular surface changes, particularly meibomian gland dysfunction and tear film instability [12, 13]. Chronic hyperglycemia in diabetes contributes to microvascular damage and autonomic neuropathy, affecting the lacrimal functional unit, including the meibomian glands [5, 14]. Prior studies by El-Sawy et al. and Lin et al. have similarly reported significant gland dropout and tear film instability in association with poor metabolic control [4, 15]. In our cohort, elevated HbA1c and longer disease duration likely played a key role in the ocular surface abnormalities observed.

Patients with Type 2 Diabetes Mellitus (T2DM) reported significantly higher Ocular Surface Disease Index (OSDI) scores compared to healthy controls (34.6 ± 12.1 vs. 15.3 ± 7.8; $p < 0.001$), indicating more severe subjective symptoms of dry eye disease (DED). These findings are consistent with previous studies demonstrating increased ocular surface discomfort among individuals with diabetes, even in the absence of overt ocular pathology [13, 16, 17]. Elevated OSDI scores in diabetics are often linked to neuropathic alterations in the corneal nerves and inflammatory damage to the tear film and ocular surface, both of which are aggravated by chronic hyperglycemia [14]. Shamsheer and Arunachalam [2] reported that diabetic patients frequently experience symptoms such as ocular irritation, dryness, and foreign body sensation compared to non-diabetics, while Yang et al. [9] found significantly worse symptom profiles in T2DM patients with meibomian gland dysfunction. The OSDI questionnaire, widely used and validated, captures not only symptom severity but also the impact of dry eye on visual function domains often affected in long-standing diabetes [3, 10]. In our study, OSDI scores negatively correlated with Schirmer I test values ($r = –0.35$; $p = 0.03$), supporting the clinical relevance of subjective symptoms. These findings highlight the importance of integrating routine symptom screening in diabetic eye evaluations, even when slit-lamp findings are minimal.

Patients with Type 2 Diabetes Mellitus (T2DM) exhibited significantly reduced tear film stability, with both tear break-up time (TBUT: 6.1 ± 2.2 seconds vs. 10.3 ± 2.5 seconds) and non-invasive break-up time (NIBUT: 5.8 ± 2.4 seconds vs. 11.2 ± 2.7 seconds) markedly lower than in healthy controls ($p<0.001$). These findings are consistent with previous studies reporting tear film dysfunction in diabetic eyes, often attributed to altered meibum secretion, increased tear film osmolarity, and subclinical inflammation of the ocular surface epithelium [16-18]. Tear film stability is critical to maintaining ocular surface health, and its disruption is a hallmark of evaporative dry eye disease [3, 5]. In diabetic individuals, multiple factors including meibomian gland dysfunction (MGD), reduced blink rate, and diabetic neuropathy affecting the lacrimal functional unit may contribute to this instability [2, 8, 13]. Supporting our results, Hao et al. and Ekin et al. previously reported significantly decreased TBUT in T2DM patients [7, 11]. The significantly lower NIBUT observed in our study further highlights the detrimental effect of diabetes on the tear film's lipid layer. Importantly, reduced TBUT and NIBUT values are clinically relevant markers, as they have been linked to greater risks of corneal surface damage and visual disturbances in diabetic populations [15]. These findings emphasize the importance of regular tear film stability testing in diabetes care, even among patients who are asymptomatic, to enable early detection and timely intervention.

Patients with Type 2 Diabetes Mellitus (T2DM) demonstrated significantly reduced aqueous tear secretion and tear

volume compared to healthy controls. The mean Schirmer I test value was 9.4 ± 3.8 mm in the diabetic group versus 15.6 ± 4.2 mm in controls ($p < 0.001$), while tear meniscus height (TMH) was also markedly lower in diabetics (0.18 ± 0.05 mm vs. 0.25 ± 0.06 mm; $p < 0.001$). These findings indicate a clear presence of aqueous-deficient dry eye in T2DM, consistent with prior reports linking hyperglycemia to lacrimal gland dysfunction and reduced tear film volume [4, 19]. The decline in aqueous production is often attributed to autonomic neuropathy, microvascular damage, and systemic inflammation affecting lacrimal gland function [19]. Shamsheer and Arunachalam similarly noted that diabetic patients frequently exhibit Schirmer I values below 10 mm, signifying impaired basal tear secretion (2). Moreover, TMH an objective marker of tear reservoir status was significantly diminished in T2DM patients in studies by Hao et al. and Yu et al., further supporting tear volume reduction in this population [7, 10]. The concurrent reduction in TMH and Schirmer values in our cohort suggests that aqueous deficiency may manifest early in diabetes, potentially preceding more advanced ocular or systemic complications. When considered alongside tear film instability and meibomian gland dysfunction, these findings support a mixed-type dry eye mechanism in T2DM. Comprehensive tear film assessment, therefore, is essential in diabetic eye care, especially for those with prolonged disease duration or poor glycemic control.

Meibomian gland morphology was significantly altered in patients with Type 2 Diabetes Mellitus (T2DM), as reflected by higher total meiboscores compared to healthy controls (3.7 ± 1.2 vs. 1.8 ± 0.9; $p < 0.001$). Both upper and lower eyelids in the diabetic group exhibited greater gland dropout, supporting a strong association between T2DM and Meibomian Gland Dysfunction (MGD), particularly of the atrophic subtype that contributes to evaporative dry eye through inadequate lipid secretion. These results are consistent with earlier studies demonstrating more extensive gland loss in diabetics, attributed to chronic metabolic stress, microvascular compromise, and subclinical inflammation within the glandular tissue [7, 10, 14]. Yu et al. [10] reported significantly higher meiboscores and poorer meibum quality in diabetic patients compared to non-diabetic controls, a finding confirmed by more recent work from Hao et al. and Ekin et al. [7, 11]. In our study, total meiboscore correlated positively with both diabetes duration ($r = 0.43$) and HbA1c ($r = 0.39$), strengthening the role of long-term hyperglycemia in glandular degeneration. Importantly, structural changes on meibography may precede both symptoms and clinical signs, making it a valuable non-invasive tool for early detection [2]. Gland dropout compromises the lipid layer, increasing tear evaporation and destabilizing the tear film. When considered alongside aqueous tear deficiency, these findings indicate a mixed-mechanism dry eye phenotype in T2DM both common and frequently underdiagnosed. Routine meibographic evaluation in diabetic eye care may thus support earlier intervention and help preserve ocular surface integrity.

Correlation analysis revealed significant associations between systemic diabetic control and ocular surface parameters. HbA1c levels were negatively correlated with TBUT ($r = –0.41$; $p < 0.01$) and positively correlated with total meiboscore ($r = 0.39$; $p = 0.02$), indicating that poorer glycemic control is linked to reduced tear film stability and more extensive meibomian gland dropout. Similarly, diabetes duration showed a significant positive correlation with meiboscore ($r = 0.43$; $p < 0.01$), suggesting that prolonged metabolic stress contributes to progressive glandular degeneration. These findings are consistent with earlier reports by Yu et al. and Lin et al., who identified higher HbA1c and longer diabetes duration as predictors of advanced meibomian gland loss and abnormal tear film parameters [9, 15]. Additionally, a negative correlation between OSDI scores and Schirmer I values ($r = –0.35$; $p=0.03$) in our study indicates that symptom severity increases as aqueous tear production declines, further reflecting the multifactorial pathogenesis of dry eye in T2DM. Together, these associations emphasize the importance of early and routine ocular surface evaluation in diabetic patients particularly those with suboptimal glycemic control or longer disease duration. The use of simple, non-invasive tests and validated symptom questionnaires may serve as valuable tools for identifying early ocular involvement and preventing downstream complications.

5. Conclusion

This study confirms that Type 2 Diabetes Mellitus (T2DM) significantly affects ocular surface health by impairing meibomian gland structure, reducing tear film stability, and lowering aqueous tear production. Diabetic patients exhibited higher OSDI scores, shorter TBUT and NIBUT, lower Schirmer I test and tear meniscus height, and significantly higher meiboscores compared to healthy controls. These alterations were significantly associated with both poor glycemic control (elevated HbA1c) and longer duration of diabetes, indicating a progressive, multifactorial pathophysiology involving neural, inflammatory, and secretory dysfunction. The presence of both evaporative and aqueous-deficient dry eye features highlights the need for comprehensive ocular surface assessment in diabetic care. Early identification and management of these changes may prevent further deterioration, improve visual comfort, and enhance quality of life in this population.

6. Limitations

This study was limited by its cross-sectional design, preventing assessment of disease progression. It was conducted at a single center, which may affect generalizability. Additionally, other systemic factors such as diabetic neuropathy or medication effects were not evaluated.

7. Recommendations

Future longitudinal and multicenter studies are recommended to assess temporal changes and broader applicability. Routine screening for ocular surface dysfunction should be incorporated into diabetic eye care, with emphasis on early diagnosis and targeted management of dry eye disease in T2DM patients.

Abbreviations

T2DM	Type 2 Diabetes Mellitus
DED	Dry Eye Disease
MGD	Meibomian Gland Dysfunction
OSDI	Ocular Surface Disease Index
TBUT	Tear Break-Up Time
NIBUT	Non-Invasive Break-Up Time
TMH	Tear Meniscus Height
HbA1c	Glycated Hemoglobin A1c
IRB	Institutional Review Board
SPSS	Statistical Package for the Social Sciences
WHO	World Health Organization
ADA	American Diabetes Association

Conflicts of Interest

The authors declare no conflicts of interest.

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