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Prevalence and Risk Factors of Dry Eye Disease in Diabetic Patients Attending a Tertiary Care Teaching Hospital

Dr. Snehal Dhanraj Raut
Assistant Professor, Department of Ophthalmology, Saraswathi Institute of Medical Sciences, Hapur

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Corresponding Author:

Dr. Snehal Dhanraj Raut
Assistant Professor, Department of Ophthalmology, Saraswathi Institute of Medical Sciences, Hapur.

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ABSTRACT

Background: Background: Diabetes mellitus (DM) is associated with multiple ocular complications, including dry eye disease (DED), which significantly impacts quality of life. Data on the prevalence of DED in diabetic populations in our region is limited. **Aim:** To determine the prevalence and associated risk factors of DED in diabetic patients attending a tertiary care teaching hospital. **Methods:** This cross-sectional study included 120 diabetic patients. All participants underwent a detailed ocular evaluation including the Ocular Surface Disease Index (OSDI) questionnaire, tear film break-up time (TBUT), Schirmer's test I, and corneal fluorescein staining. DED was diagnosed based on established criteria. **Results:** The prevalence of DED among diabetic patients was found to be 58.3% (70 out of 120 patients). A significant association was observed between the presence of DED and increasing age (p=0.002), longer duration of diabetes (p=0.001), poorer glycemic control (HbA1c >7%, p=0.005), and the presence of diabetic retinopathy (p=0.008). The most common symptoms reported were grittiness (72.8%) and fluctuating vision (65.7%). **Conclusion:** Dry eye disease is highly prevalent among the diabetic population in this study. Regular screening for DED should be integrated into the routine ophthalmological assessment of diabetic patients, especially those with longer disease duration and poor glycemic control.

Keywords: Dry Eye Disease, Diabetes Mellitus, Tear Film, Ocular Surface, Prevalence, Tertiary Care.

INTRODUCTION:

Diabetes mellitus (DM) represents a formidable and escalating global health challenge, with its prevalence reaching epidemic proportions and imposing a substantial burden on healthcare systems worldwide.[1] Characterized by chronic hyperglycemia resulting from defects in insulin secretion, action, or both, diabetes exerts its systemic deleterious effects through persistent microvascular and macrovascular complications, which are primary drivers of significant morbidity, disability, and mortality.[2] The ocular system is exquisitely vulnerable to these metabolic disturbances, with diabetic retinopathy (DR) being the most extensively studied and a leading cause of preventable blindness among the working-age population.[3] Consequently, ophthalmic surveillance for diabetic patients has historically been dominated by a posterior segment paradigm, emphasizing regular and meticulous fundoscopic screening for the detection and management of retinopathy.

However, a growing body of compelling evidence underscores that the impact of diabetes on the eye is not anatomically confined to the retina.[4] The ocular surface—a complex, integrated, and dynamic functional unit comprising the cornea, conjunctiva, tear film, and their associated neural networks, glands, and immune components—is increasingly recognized as a critical and vulnerable target of diabetic pathophysiology.[5] Dry eye disease (DED), a multifactorial disorder of the ocular surface characterized by a loss of tear film homeostasis, has emerged as a significant, yet frequently underdiagnosed and undertreated, ocular comorbidity in this population.[6] DED manifests as a symptomatic spectrum of ocular discomfort, visual disturbance, and tear film instability, often with concurrent ocular surface inflammation and damage.[7] It represents not merely a nuisance but a condition that can profoundly degrade quality of life, impair functional vision for critical tasks like reading and driving, and potentially complicate the outcomes of other necessary ocular interventions, such as cataract surgery.[8]

The etiopathogenesis of DED in the context of diabetes is complex, multifactorial, and synergistic, reflecting the systemic nature of the disease. Chronic hyperglycemia serves as the foundational insult, driving multiple parallel pathways that

converge on ocular surface dysfunction.[9] Firstly, diabetic peripheral neuropathy can damage the sensory (trigeminal) and autonomic nerves that form the afferent and efferent arms of the lacrimal functional unit.[10] This neurotrophic keratopathy impairs the neural reflex arc essential for basal and reflex tear secretion from the lacrimal gland, leading to aqueous tear deficiency.[11] Secondly, diabetes is strongly and independently associated with meibomian gland dysfunction (MGD).[12] Hyperglycemia may alter the composition and melting point of meibum, leading to glandular obstruction, atrophy, and a deficient lipid layer of the tear film.[13] This results in excessive tear evaporation, defining the evaporative subtype of DED, which studies suggest may be the predominant form in diabetic cohorts.[14] Thirdly, a persistent state of low-grade systemic inflammation and oxidative stress, hallmarks of diabetes, creates a pro-inflammatory microenvironment on the ocular surface.[15] Elevated levels of inflammatory cytokines can disrupt the healthy ocular surface epithelium, promote goblet cell loss, and further destabilize the tear film.[16]Additionally, the frequent use of medications common in diabetic patients (e.g., antihypertensives, antidepressants) and the potential for delayed corneal epithelial healing compound the risk and severity of DED.[17]

While international studies report a widely variable prevalence of DED in diabetic populations, ranging from 15% to over 54%, this disparity highlights the critical influence of differing diagnostic criteria, study population characteristics (e.g., community-based vs. hospital-based), and geographic, ethnic, and environmental factors.[18] Data specifically from patients attending tertiary care teaching hospitals—institutions that typically serve as referral centers managing individuals with more advanced disease, longer duration, poorer systemic control, and a higher burden of microvascular complications—remain particularly sparse in our regional and national context.[19] This patient subset likely represents a group at the highest risk for ocular surface disease, making their investigation a clinical priority.

Understanding the precise magnitude and pattern of this problem in our specialized clinical setting is therefore crucial. It moves beyond academic interest to direct clinical relevance. A clear epidemiological picture is the first step in advocating for a paradigm shift in diabetic eye care—from a primarily retina-centric model to a more holistic approach that encompasses the entire ocular system.[20]

Therefore, this study aims to systematically investigate the prevalence and clinical characteristics of dry eye disease among a consecutively enrolled cohort of diabetic patients attending the Ophthalmology Outpatient Department of a tertiary care teaching hospital. By employing a standardized diagnostic approach that combines validated symptom assessment (OSDI questionnaire) with key objective clinical tests (TBUT, Schirmer's test, corneal staining), this research seeks to accurately delineate the burden of DED. Furthermore, it aims to analyze its association with key diabetic parameters such as disease duration, degree of glycemic control (HbA1c), treatment modalities, and the presence and severity of diabetic retinopathy. The findings are anticipated to provide robust local evidence to underscore the necessity of integrating comprehensive ocular surface evaluation into the standard diabetic eye care protocol. This integration is essential for enabling timely diagnosis, targeted management, and ultimately, the preservation of both visual function and ocular comfort in this vulnerable population.

MATERIALS & METHODS:

A hospital-based, cross-sectional observational study was conducted over a period of six months. The study was carried out in the outpatient department of the Ophthalmology at Saraswathi Institute of Medical Sciences, Hapur tertiary care teaching hospital. The target population consisted of all known diabetic patients (Type 1 or Type 2) aged 18 years and above, seeking routine or referral-based ophthalmic care at the hospital during the study period.

Inclusion Criteria:

- Diagnosed patients with diabetes mellitus (Type 1 or Type 2) for at least one year.
- Age ≥ 18 years.
- Willing to provide informed consent.

Exclusion Criteria:

- History of ocular surgery or trauma within the preceding six months.
- Active ocular infection or inflammation (e.g., conjunctivitis, uveitis).
- Diagnosis of connective tissue diseases known to cause dry eye (e.g., rheumatoid arthritis, Sjögren's syndrome).
- Regular use of topical ocular medications (other than artificial tears), such as glaucoma drops.
- Contact lens wearers.
- Patients with facial nerve palsy or eyelid abnormalities affecting blink.

Sample Size Calculation

A sample size of 120 was calculated using the single population proportion formula, based on an assumed prevalence (p) of dry eye disease in diabetics of 50% (to achieve maximum variability, as local data was limited), a 95% confidence level (Z=1.96), and a margin of error (d) of 9%.
The formula used was: $n = (Z^2 * p * (1-p)) / d^2$. This yielded a minimum sample of 119, which was rounded to 120.

Procedure for Data Collection

Data collection was performed in a single visit through a structured, sequential protocol:

1. **Informed Consent & Interview:** Eligible patients were identified, the study was explained, and written informed consent was obtained. A pre-tested proforma was used to record demographic details, diabetic history, medication history, and relevant systemic history.
2. **Symptom Assessment:** The validated Ocular Surface Disease Index (OSDI) questionnaire was administered to quantify dry eye symptoms over the preceding week.
3. **Ocular Examination:** The following tests were performed in sequence to avoid influencing results:
    - **Visual Acuity:** Measurement of best-corrected visual acuity using a Snellen chart.
    - **Tear Film Break-Up Time (TBUT):** After instilling one drop of fluorescein sodium, the patient was asked to blink naturally and then hold the eyes open. The time interval between the last complete blink and the first appearance of a dry spot in the corneal tear film was measured three times using a cobalt blue filter, and the average was recorded in seconds.
    - **Schirmer's Test I:** A standardized sterile Schirmer's strip was placed at the junction of the middle and lateral third of the lower eyelid, without topical anesthesia. The patient was asked to close their eyes gently. The length of wetting (in millimeters) was recorded after five minutes.
    - **Corneal Staining:** Fluorescein staining was assessed using the Oxford Grading Scheme (0-5) under cobalt blue light to evaluate corneal epithelial damage.
    - **Slit-Lamp Biomicroscopy:** A detailed anterior segment examination was performed to assess for meibomian gland dysfunction, blepharitis, and other abnormalities.
    - **Fundus Examination:** Following pupillary dilation with tropicamide 0.8%, a detailed fundus examination was performed using a +90D lens at the slit-lamp to diagnose and grade diabetic retinopathy.

Data Analysis

All data from the patient proformas were entered into a Microsoft Excel spreadsheet. Data cleaning was performed to check for errors, inconsistencies, and missing values. To ensure confidentiality, patient identifiers were removed, and a unique study identification number was assigned to each record. The anonymized dataset was then exported to IBM SPSS Statistics software (Version 25.0) for statistical analysis.

RESULTS:

A total of 120 diabetic patients were enrolled and completed the study protocol. The findings are presented in the following tables.

Table 1: Demographic and Clinical Characteristics of the Study Population (N=120)

Characteristic	**Mean ± SD / Frequency (%)**
Age (years)	56.4 ± 9.8
Gender	
- Male	57 (47.5%)
- Female	63 (52.5%)
Type of Diabetes	
- Type 2	120 (100%)
- Type 1	0 (0%)
Duration of Diabetes (years)	8.7 ± 5.2
Glycemic Control (HbA1c, %)	8.1 ± 1.6
Categories of HbA1c	
- ≤7.0% (Good Control)	38 (31.7%)
- >7.0% (Poor Control)	82 (68.3%)
Diabetic Retinopathy (DR)	

Characteristic	Mean ± SD / Frequency (%)
- Absent	78 (65.0%)
- Present (Any Stage)	42 (35.0%)
- Non-Proliferative DR (NPDR)	36 (30.0%)
- Proliferative DR (PDR)	6 (5.0%)

A total of 120 diabetic patients were enrolled and completed the study protocol. The demographic and clinical profile of the participants is summarized in **Table 1**. The mean age of the cohort was 56.4 ± 9.8 years, with a near-equal gender distribution (47.5% male, 52.5% female). All participants had Type 2 diabetes, with a mean disease duration of 8.7 ± 5.2 years. The average HbA1c was 8.1 ± 1.6%, with the majority of patients (68.3%) having poor glycemic control (HbA1c >7.0%). Diabetic retinopathy was present in 42 patients (35.0%), predominantly in its non-proliferative stage (30.0%).

Table 2: Prevalence and Subtypes of Dry Eye Disease (DED)

DED Status	Frequency (n)	Percentage (%)
Overall DED (Diagnosed)	70	58.3
- DED Absent	50	41.7
Subtype of DED (n=70)		
- Evaporative DED (Primary MGD)	45	64.3
- Aqueous-Deficient DED	25	35.7

The primary outcome, the prevalence of dry eye disease (DED), is presented in **Table 2**. Using the composite diagnostic criteria, 70 out of 120 patients were diagnosed with DED, yielding a prevalence of **58.3%**. Among these patients with DED, the evaporative subtype, primarily driven by meibomian gland dysfunction, was more common (64.3%, n=45) than the aqueous-deficient subtype (35.7%, n=25).

Table 3: Association of Dry Eye Disease with Patient Characteristics

Characteristic	DED Present (n=70)	DED Absent (n=50)	p-value
Mean Age (years), Mean ± SD	59.1 ± 8.5	52.4 ± 10.1	**0.002***
Gender, n (%)			0.42
- Male	35 (50.0%)	22 (44.0%)	
- Female	35 (50.0%)	28 (56.0%)	
Diabetes Duration >5 yrs, n (%)	55 (78.6%)	22 (44.0%)	**<0.001***
HbA1c >7.0%, n (%)	55 (78.6%)	27 (54.0%)	**0.005***
Diabetic Retinopathy Present, n (%)	33 (47.1%)	9 (18.0%)	**0.008***

The analysis of associations between DED and key patient characteristics is detailed in **Table 3**. Patients with DED were significantly older than those without DED (59.1 vs. 52.4 years, p=0.002). A strong association was found with diabetes duration, as 78.6% of DED patients had diabetes for more than five years, compared to only 44.0% in the non-DED group (p<0.001). Poor glycemic control (HbA1c >7.0%) was significantly more prevalent in the DED group (78.6% vs. 54.0%,

p=0.005). Furthermore, the presence of diabetic retinopathy was strongly associated with DED, occurring in 47.1% of DED patients versus 18.0% of non-DED patients (p=0.008). No significant association was found between DED and gender (p=0.42).

Table 4: Ocular Surface Parameters in Patients with and without DED

Ocular Parameter	DED Patients (n=70), Mean ± SD / n (%)	Non-DED Patients (n=50), Mean ± SD / n (%)	p-value
OSDI Score	32.5 ± 11.8	8.2 ± 3.1	**<0.001***
Tear Film Break-Up Time (TBUT, sec)	6.8 ± 2.1	13.5 ± 3.2	**<0.001***
TBUT ≤10 seconds, n (%)	70 (100%)	12 (24.0%)	**<0.001***
Schirmer's Test I (mm/5 min)	8.4 ± 4.7	16.8 ± 5.5	**<0.001***
Schirmer's ≤10 mm/5 min, n (%)	48 (68.6%)	7 (14.0%)	**<0.001***
Positive Corneal Staining (≥ Grade 1), n (%)	37 (52.9%)	3 (6.0%)	**<0.001***

Table 4 compares the objective and subjective ocular surface parameters between the two groups. As expected, DED patients had a significantly higher mean OSDI symptom score (32.5 ± 11.8) compared to non-DED patients (8.2 ± 3.1, p<0.001). All objective tear film tests showed markedly abnormal results in the DED group. The mean tear film break-up time (TBUT) was severely reduced in DED patients (6.8 seconds) compared to controls (13.5 seconds, p<0.001), with 100% of DED patients having an abnormal TBUT (≤10 sec). Similarly, Schirmer's test values were significantly lower in the DED group (8.4 mm/5 min vs. 16.8 mm/5 min, p<0.001), with 68.6% showing aqueous deficiency (≤10 mm). Positive corneal fluorescein staining (≥ Grade 1) was observed in 52.9% of DED patients but in only 6.0% of controls (p<0.001).

Table 5: Frequency of Dry Eye Symptoms among DED Patients (n=70)

Symptom (from OSDI)	Frequency (n)	Percentage (%)
Grittiness / Foreign Body Sensation	51	72.8
Fluctuating / Blurry Vision	46	65.7
Ocular Redness	36	51.4
Light Sensitivity (Photophobia)	32	45.7
Burning or Stinging Sensation	29	41.4

Table 5 outlines the frequency of specific dry eye symptoms reported by the 70 patients diagnosed with DED. The most commonly reported symptoms were grittiness or a foreign body sensation (72.8%), followed by fluctuating or blurry vision (65.7%). Ocular redness was reported by 51.4% of patients, while light sensitivity and a burning or stinging sensation were reported by 45.7% and 41.4% of patients, respectively.

DISCUSSION:

This cross-sectional study of 120 diabetic patients at a tertiary care teaching hospital reveals a significant burden of ocular surface disease, with a 58.3% prevalence of clinically diagnosed dry eye disease (DED). This finding situates our cohort at the higher end of the global prevalence spectrum reported in the literature and underscores DED as a major, though frequently overlooked, diabetic complication in patients with advanced or long-standing disease. The high prevalence can be attributed to our study setting—a referral hospital managing complex cases—and our use of a composite diagnostic

criterion combining symptoms and objective signs, which enhances detection sensitivity compared to studies relying on a single test.[21]

The observed prevalence aligns closely with several studies in similar clinical environments but diverges from community-based reports. For instance, a 2016 study by Zhang et al. in a Chinese tertiary hospital reported a DED prevalence of 54.2% among type 2 diabetics, utilizing similar diagnostic benchmarks of symptom questionnaires and tear film instability.[22] Our finding of 58.3% corroborates this high burden in specialized care settings. Conversely, a large community-based study by Manaviat et al. (2008) found a lower prevalence of 32.7%, highlighting how selection bias toward patients with more severe systemic disease in hospital-based studies captures a population at inherently higher risk.[23] The predominance of the evaporative subtype (64.3%) in our cohort is a critical insight, consistent with the pathophysiological understanding that meibomian gland dysfunction (MGD) is a central driver of diabetic dry eye. This mirrors the findings of a 2019 study by Alshamrani et al., which identified MGD as the leading cause of DED in diabetics, implicating altered meibum composition and gland obstruction due to hyperglycemia and dyslipidemia.[24]

Our analysis identified robust, statistically significant associations between DED and key markers of diabetes severity, reinforcing the systemic nature of ocular surface damage. The strong correlation with longer diabetes duration (>5 years) and poorer glycemic control (HbA1c >7.0%) is pathophysiologically coherent.[25] Chronic hyperglycemia is the cornerstone of diabetic microangiopathy and neuropathy.[26] Over time, it can impair the innervation and vascular supply to the lacrimal and meibomian glands, disrupt corneal epithelial integrity, and perpetuate a pro-inflammatory ocular surface environment.[27] The significant association with diabetic retinopathy ($p=0.008$) is particularly compelling. It suggests that DED and retinopathy may not be parallel but potentially interrelated complications, sharing common underlying pathways of microvascular endothelial dysfunction, chronic inflammation, and neuronal damage.[28] This association implies that the diagnosis of retinopathy on fundoscopy should serve as a potent clinical cue to meticulously examine the anterior segment.[29]

The clinical profile of our DED patients revealed a high symptomatic burden, with grittiness and fluctuating vision being most common. This visual fluctuation, often reported by patients, is a direct consequence of an unstable tear film and is a significant contributor to reduced quality of life and functional disability.[30] The objective signs were pronounced, with severely reduced TBUT being almost universal among DED patients, further emphasizing the evaporative pathology.[31] The presence of positive corneal staining in over half of the DED patients indicates a level of epithelial disease that extends beyond discomfort to potential structural compromise, increasing the risk of infection and complicating other ocular procedures.[32]

Limitations and Strengths: This study is limited by its single-center, cross-sectional design, which prevents the establishment of causality. The absence of a matched non-diabetic control group limits direct comparative analysis of risk attribution. However, the study's strengths lie in its rigorous, standardized diagnostic protocol for DED, the consecutive enrollment of patients, and the detailed correlation with systemic diabetic parameters.

CONCLUSION:

This study provides compelling evidence that dry eye disease is a highly prevalent and clinically significant comorbidity in diabetic patients attending a tertiary care center, particularly those with longer disease duration, suboptimal glycemic control, and existing retinopathy. The findings advocate for a fundamental shift in the standard diabetic eye exam from a retina-centric model to a comprehensive ocular health assessment. We recommend that dry eye evaluation—including a simple symptom screen (e.g., OSDI) and TBUT measurement—be integrated into the routine ophthalmological follow-up of all diabetic patients. Early detection and management of DED are essential not only to alleviate symptoms and improve visual quality but also to preserve ocular surface integrity and optimize outcomes for future ocular interventions, ultimately enhancing the overall well-being of the growing diabetic population.

Declaration:

Conflicts of interests: The authors declare no conflicts of interest.

Author contribution: All authors have contributed in the manuscript.

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