Section: Ophthalmology

Original Research Article

TEAR FILM DYSFUNCTION AND MEIBOMIAN GLAND ABNORMALITIES IN PATIENTS WITH PTERYGIUM: A COMPARATIVE STUDY

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Received : *17/07/2025*
Received in revised form : *05/09/2025*
Accepted : *29/09/2025*

Keywords:
Pterygium, Schirmer test, TBUT, Meiboscore, Meibomian gland dysfunction.

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DOI: 10.47009/jamp.2025.7.5.152

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2025; 7 (5); 796-799

ABSTRACT

Background: Aim: To evaluate tear film abnormalities and Meibomian gland dysfunction in patients with pterygium compared to healthy individuals. **Materials and Methods:** A prospective, comparative study was conducted on 40 patients with primary pterygium and age-matched healthy volunteers. Tear film stability and quantity were assessed using tear breakup time (TBUT <10 seconds) and Schirmer's test (<15 mm), while Meibomian gland morphology was graded using meiboscore. **Results:** The mean Schirmer's test value in the pterygium group was 15.6 ± 7.76 mm, indicating a tendency toward mild dry eye, while mean TBUT was 11.3 ± 5.39 seconds, significantly lower than in controls. Meibomian gland dropout, reflected by higher meiboscore, was also significantly associated with pterygium. **Conclusion:** Pterygium is associated with significant ocular surface alterations, including reduced tear film stability, borderline aqueous deficiency, and Meibomian gland dysfunction. These changes contribute to ocular discomfort and emphasize the need for early detection and management of ocular surface disease in pterygium patients.

INTRODUCTION

Pterygium is a non-neoplastic elastotic degeneration originated in the bulbar conjunctiva that can extend to the corneal surface. It can cause symptoms of ocular discomfort, corneal irregularities, aesthetic issues thus compromising visual acuity and patients quality of life.[1-3] The correct pathogenesis of the injury is not yet completely understood. Age, hereditary factors, sunlight, chronic inflammation, microtrauma, and dry climate are some possible contributing factors.[4,5] The word Pterygium originated from the Greek word which means a wing like structure and was first described by Hippocrates. Its development is unrelated to antecedent injury or inflammation and mostly pterygia are seen nasally.[6]
The tear film consists of three layers: the outer lipid layer, the middle aqueous layer, and the innermost mucin layer. In dry eye disease, dysfunction may arise from reduced aqueous secretion or increased evaporation due to lipid layer instability. Some studies have pointed varying changes in tear film and ocular surface are related to pterygium, but consistent correlations are not found.[7-10] Although numerous theories have been listed in the pathogenesis of the pterygium (e.g. exposure to ultraviolet radiation, viral infection, oxidative stress, genetic problems, inflammatory mediators, extracellular matrix modulators) the mechanism responsible for development of pterygium remains controversial.[11]
If an association exists, the underlying mechanisms and whether one condition could exacerbate the progression of the other are still uncertain. Current literature provides limited evidence on which specific components of the pre-corneal tear film are most affected in pterygium. Therefore, at present, the role of tear film deficiency and instability in relation to pterygium is still poorly understood. The study was conducted with the aim to evaluate how ocular surface parameters correlate with pterygium, its clinical presentation and its impact on ocular surface structures.

MATERIALS AND METHODS

This was an observational study conducted on all patients undergoing surgery for pterygium from January 2024 to December 2024 at a tertiary eye care center in Rajasthan and comparison with healthy volunteers. The study was conducted after obtaining permission from Institutional Ethics Committee and Research Review Board of the Hospital.

Study group was patients with unilateral pterygium (n = 40) and control group included age-matched healthy volunteers (n = 40). Inclusion Criteria was Patients of age >18 years with unilateral pterygium and willing to be part of the study.

Detailed clinical history was taken for every patient including demographic data, personal and family history of pterygium, ocular and systemic comorbidities, ocular or systemic medications, visual acuity as well as a full ophthalmic exam.

Patients excluded from the study were those having systemic diseases which were associated with dry eye, patients on systemic medications or antiglaucoma drugs, patients routinely using contact lens and those having other adnexal disease. Patients having anterior or posterior segment disease which may alter the tear film stability and integrity, who underwent ocular surgery recently and those with recurrent or bilateral pterygium were also excluded.

On all patients the tests performed were Schirmer test, Tear Break Up time (TBUT),

Schirmer I test was performed with a (no. 41 Whitmann filter paper). The filter paper was folded 5 mm from one end and inserted at the junction of the middle and outer third of the lower lid, taking care not to touch the cornea or lashes. The patient was asked to keep the eyes gently closed. After 5 minutes the filter paper was removed and the amount of wetting from the fold measured. <15 mm of wetting after 5 minutes was considered to be abnormal.

Tear Break Up time (TBUT) - For this test Fluorescein 2% or an impregnated fluorescein strip moistened with non-preserved saline was instilled into the lower fornix. The patient was asked to blink several times. The tear film was examined at the slit lamp with a broad beam using the cobalt blue filter. After an interval, black spots or lines appear in the fluorescein-stained film indicating the formation of dry areas. The Breakup Time is the interval between the last blink and the appearance of the first randomly distributed dry spot. Patient with TBUT < 10 second were selected.

Meibomian glands were estimated by autorefractor keratometry machine by everting upper and lower lids and then meibomian glands were estimated and meiboscore was calculated. The classification scale, adapted from Arita et al,[12] used the following score for each eyelid: 0 (no loss of glands); 1 (loss involving less than one third of the total meibomian gland area); 2 (loss between one third and two thirds of the total area of the meibomian gland); and 3 (loss more than two-thirds of the total meibomian gland area).

Pterygium patients were classified according their graduation: grade 1 to 4 according to fibrovascular tissue extension towards the cornea (grade 1 when the lesion reaches the limbus, grade 2 when it covers the cornea at about 2 mm, grade 3 when it reaches the pupil margin and grade 4 when it exceeds the pupil).

The data was compiled in MS Excel. Descriptive statistics was used to present the data. SPSS (version 26) was used to analyse the data. Results on continuous measurements were presented as Mean+SD (Min -Max) and results on categorical measurements were presented as numbers (%). Significance was assessed at 5% level of significance.

RESULTS

The study included 80 patients distributed in 2 groups. Group I comprises of 40 patients having pterygium of mainly grade 2 or grade 3. Group II were control healthy volunteers, 40 in number, visiting eye OPD not having pterygium.

The patients included in the study group were maximum in the age group 31-40 years with a mean age of 44.8 ± 5.06 year [Table 1]. In comparison the mean age of control group was 43.4 ± 4.37 years. There was no difference among the two group in reference to age with a p-value > 0.05.

Table 1: Age specific distribution of patient

Age in years	Study group	Control Group
18-30	5	6
31-40	13	12
41-50	11	13
51-60	11	9
Total	40	40

The mean value of Schirmer test in study group was found to be 15.6±7.76mm whereas in control group was 21.3±5.48mm [Table 2]. Schirmer value in study group pterygium patient was statistically significant with p-value of 0.012.

Table 2: Schirmer test – distribution of patients in study and control group

Schirmer (mm)	Study group	Control Group
5-10	18 (45 %)	5 (12.5 %)
11-20	15 (37.5 %)	12 (30 %)
>20	7 (17.5 %)	23 (57.5 %)
Total	40	40

ISSN (O): 2687-5365; ISSN (P): 2753-6556

On performing TBUT test, mean value in study group was found to be 11.3 ± 5.39 seconds and in control group was 15.4 ± 4.33 seconds. The result was statistically significant with p value 0.011.

Table 3: TBUT Test –frequency distribution the two groups

TBUT (in seconds)	Study group	Control group
<5	6 (15 %)	1 (2.5 %)
5-10	18 (45 %)	8 (20 %)
11-15	11 (27.5 %)	15 (37.5%)
16-20	5 (12.5 %)	16 (40 %)
Total	40	40

Meiboscore in pterygium patient was statistically significantly higher with a mean value of 2.45 ± 1.04 in pterygium patients and 1.07 ± 0.96 in control group [Table 4].

Table 4: Meiboscore-Frequency distribution in study and control group

Meiboscore	Study Group	Control Group
0	8 (20 %)	14 (35 %)
1	11 (27.5 %)	12 (30 %)
2	13 (32.5 %)	9 (22.5 %)
3	8 (20 %)	5 (12.5 %)
Total	40	40

DISCUSSION

The present study demonstrated significant alterations in tear film stability, aqueous secretion, and Meibomian gland morphology among patients with pterygium when compared to healthy controls. Specifically, patients with pterygium exhibited lower Schirmer values, reduced TBUT, and higher meiboscores, suggesting that pterygium not only disturbs the ocular surface mechanically but also contributes to the pathophysiology of dry eye disease and Meibomian gland dysfunction (MGD).

Our findings of reduced Schirmer test values are consistent with earlier reports indicating a strong association between pterygium and reduced tear secretion. Kotecha et al. (2021) observed a significant correlation between pterygium and dry eye using Schirmer's test and tear film breakup time, with pterygium patients more likely to fall into the mild to moderate dry eye spectrum.[13] Similarly, Roka et al. (2013) in their study from Nepal noted significantly reduced Schirmer values in pterygium patients compared to controls, suggesting that tear hyposecretion may contribute to ocular surface instability.[14] In our study, nearly half of the pterygium patients demonstrated Schirmer's values <10 mm, reinforcing the likelihood of a concomitant aqueous-deficient dry eye component in these individuals.

Tear film stability, as measured by TBUT, was also significantly reduced in the study group. This aligns with findings from both Indian and international studies that consistently show lower TBUT values in pterygium patients.[13,14] The reduced TBUT indicates instability of the tear film lipid layer, which is critical for preventing premature evaporation. Fen et al. (2017) demonstrated that patients with pterygium had not only shorter TBUT but also distinct alterations in Meibomian gland morphology when compared with normal subjects.[15] This highlights the interrelationship between tear film instability and Meibomian gland function in pterygium.

Indeed, one of the striking results of the present study was the significantly higher meiboscores among pterygium patients. Meibomian glands play a central role in ocular surface health, and dysfunction in these glands is now recognized as one of the leading causes of evaporative dry eye.[16] Fen et al. (2017) reported similar results, observing increased gland dropout in pterygium patients compared to healthy controls.[15] The mechanical elevation of the conjunctiva by the fibrovascular tissue in pterygium may distort the eyelid-globe interface, affecting the normal expression of meibum and leading to secondary gland atrophy. Additionally, chronic inflammation in pterygium, mediated by cytokines and matrix metalloproteinases, may also extend to the eyelid margin and adversely affect glandular function.[17]

The pathogenesis of pterygium itself has been attributed to a combination of ultraviolet (UV) radiation exposure, genetic predisposition, and chronic ocular surface inflammation.[17,18] Mackenzie et al. (1992) confirmed UV exposure as the most important environmental risk factor for the development of pterygium.[18] This chronic UV-related insult also affects the tear film and goblet cells, further destabilizing the ocular surface.

The observed alterations in tear film and Meibomian glands have important clinical implications. Reduced tear volume and stability in pterygium patients contribute not only to ocular discomfort but may also influence surgical outcomes. Kilic and Gurler (2006) demonstrated that pterygium excision followed by limbal conjunctival autografting improved tear film parameters postoperatively, suggesting that restoration of the ocular surface anatomy can partially reverse tear dysfunction.[19] Nevertheless, persistent Meibomian gland dropout, as observed in our study, may limit full recovery of tear film stability and could predispose to recurrent symptoms.

Our findings also align with the international workshop definition of Meibomian gland dysfunction as "a chronic, diffuse abnormality of the Meibomian glands, commonly characterized by terminal duct obstruction and/or qualitative/quantitative changes in glandular secretion".[20] In pterygium patients, such dysfunction may represent a secondary process initiated by chronic ocular surface inflammation and mechanical factors, further amplifying tear instability.

Taken together, these results emphasize that pterygium is not merely a localized conjunctival lesion but a disease with widespread ocular surface implications. The interplay between aqueous tear deficiency, evaporative dry eye from Meibomian gland dysfunction, and ocular surface inflammation creates a vicious cycle, aggravating symptoms and impacting quality of life. Therefore, evaluation of tear film parameters and Meibomian gland status should be an integral part of the clinical assessment of pterygium patients. Preoperative optimization of the ocular surface, including lubrication and management of MGD, may improve patient comfort and surgical outcomes.

CONCLUSION

This study demonstrates that pterygium is associated with significant alterations in ocular surface physiology, including reduced tear secretion, decreased tear film stability, and increased Meibomian gland dropout. These changes highlight the multifactorial nature of ocular surface disease in pterygium, where both aqueous-deficient and evaporative dry eye components coexist. The findings reinforce the need for comprehensive evaluation of tear function and Meibomian gland status in all pterygium patients. Early identification and management of ocular surface abnormalities may not only improve patient comfort but also optimize surgical outcomes and reduce the risk of recurrence.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556