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#### Original Article

## Relationship of Environmental Exposure with Clinical Morphology of Primary Pterygium in Pakistani Adults

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#### Abstract

**Objective:** To determine the association of age, sex, daily sun exposure, smoking, and toxic chemical exposure with the clinical morphology of primary pterygium.

**Methodology:** This cross-sectional study was conducted in the Shifa College of Medicine Islamabad, Pakistan, from January 2025 to December 2025. Forty-one patients had primary unilateral pterygium. We recorded average daily sun exposure from the clinical history as hours per day. Smoking was recorded only as a categorical smoking-status variable. We recorded toxic chemical exposure only as present or absent. We examined the pterygium for visible inflammation, atrophic or progressive appearance, Stocker's line, Fuchs spots, corneal extension, and site.

**Results:** The mean age was  $52.15 \pm 14.36$  years and 24 patients (58.5%) were female. Mean daily sun exposure was  $5.54 \pm 1.00$  hours. Fifteen patients (36.6%) were smokers, and 27 (65.9%) reported toxic chemical exposure. Clinical inflammation occurred in 41.5%, progressive pterygium in 24.4%, Stocker's line in 36.6%, and Fuchs spots in 22.0%. We found no clear associations between the examined factors and clinical features. Continuous estimates were small and imprecise, while categorical comparisons included confidence intervals.

**Conclusion:** Among 41 surgically selected patients with unilateral primary pterygium, we found no clear associations between demographic or environmental factors and lesion morphology. Smaller associations cannot be excluded, and findings should not be generalized beyond this selected population.

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## Introduction

Pterygium is a fibrovascular growth of conjunctival tissue that crosses the limbus and extends onto the cornea. It is common in people who live or work in areas with strong sunlight. Recent reviews still identify ultraviolet exposure as one of the main environmental factors in pterygium, although inflammation, oxidative stress, abnormal cell growth and other pathways also take part in the disease [1]. A recent global analysis also showed that pterygium remains common worldwide and that its prevalence increases with age and varies between regions [2]. Environmental exposure may be more complex than sunlight alone. A large study from Shanghai reported that solar radiation and air pollutants, especially PM<sub>2.5</sub>, ozone and nitrogen dioxide, were related to pterygium visits. Occupational health research also continues to emphasize the effect of long-term solar exposure on the eyes of outdoor workers [3]. The clinical appearance of pterygium also varies from patient to patient. Some lesions are quiet and atrophic, while others are thick, vascular or progressive. Recent studies have linked pterygium morphology with dry-eye findings, vascular changes, systemic inflammatory markers, meibomian gland dysfunction and microscopic tissue changes [4]. This suggests that the visible appearance of a lesion may reflect several local and systemic processes rather than environmental exposure alone. In Pakistan, sunlight exposure is common in many occupations, but there is limited information on whether environmental exposure is related to the clinical appearance of pterygium after the disease has developed. Recent molecular and pathological work also shows that pterygium involves complex changes in genes, extracellular matrix, angiogenesis and inflammation [5,6,7]. In this study, I examined whether age, sex, daily sun exposure, smoking and toxic chemical exposure were related to inflammation, atrophic or progressive phenotype, Stocker's line and Fuchs spots in patients with primary pterygium [8].

## Materials and Methods

### Study design and participants

This cross-sectional study was conducted in the Shifa College of Medicine Islamabad, Pakistan, from January 2025 to December 2025. 41 patients with primary unilateral pterygium who were selected for surgical excision. Patients were assessed clinically before surgery. The parent research was approved by the Ethical Review Committee of the Shifa College of Medicine Islamabad, Pakistan and informed consent was obtained from the participants. Because inclusion required a

decision for surgical excision, I consider this cohort to represent a surgically selected, clinically significant group of pterygium cases rather than the full spectrum of patients with pterygium.

### Study period, setting and recruitment

We carried out clinical recruitment, surgical excision and specimen collection from January 2020 through October 2020. I included patients only when surgical excision of the pterygium had already been decided. After obtaining informed consent, I took a complete history and examined the eyes and pterygium clinically before surgery. The surgically excised pterygium specimen was then collected for the parent research. Using this approach, I recruited the 41 patients included in the present analysis.

### Clinical and exposure data

We obtained the exposure information from the clinical history. I recorded average daily sun exposure as the reported number of hours of sunlight exposure per day. I recorded smoking as a categorical smoking-status variable only. I recorded toxic chemical exposure as a binary history of exposure (present/absent). I examined the pterygium for visible inflammation, size, site, atrophic or progressive appearance, Stocker's line and Fuchs spots. Corneal extension was recorded as Type 1 when it was less than 2 mm, Type 2 when it extended up to 4 mm and Type 3 when it reached the pupillary margin or visual axis.

### Clinical classification criteria and observer assessment

Pterygium characteristics were assessed clinically before surgery, including the presence or absence of inflammation and classification as atrophic or progressive. Clinical inflammation refers to visible surface hyperemia/redness with increased vascularity; an atrophic phenotype refers to a thin, pale or translucent, relatively avascular lesion with the underlying episcleral vessels clearly visible; and a progressive phenotype refers to a thicker, fleshy and more vascular lesion with reduced or absent visibility of the underlying episcleral vessels [9,10]. Interobserver agreement statistic was reported.

### Statistical analysis

We analyzed the data at patient level. I summarized continuous variables as mean  $\pm$  standard deviation and categorical variables as frequency and percentage. Because I had only 41 patients and some outcome groups were small, particularly the 10 patients with progressive pterygium and the 9 with Fuchs spots, I kept the analysis to simple bivariate methods rather than fitting unstable multivariable models. I examined age and

daily sun exposure in relation to each clinical outcome using Spearman rank correlation and report *r* as the effect estimate with an approximate 95% confidence interval based on Fisher's *z* transformation. For sex, smoking and toxic chemical exposure, I used two-sided Fisher's exact tests because several expected cell counts were small. To make the magnitude and uncertainty of these categorical associations clearer, I also report conditional odds ratios with exact 95% confidence intervals. The parent thesis preserves the marginal totals and the reported Fisher exact *p* values but does not reproduce the original 2 x 2 cross-tabulations. I therefore reconstructed, for each categorical comparison, the unique 2 x 2 table compatible with the reported margins and Fisher *p* value to the reported three-decimal precision, and calculated a conditional maximum-likelihood odds ratio with an exact 95% confidence interval. I regard these odds ratios as secondary reconstructed estimates rather than independently verified patient-level calculations, so I interpret them cautiously. I examined whether five demographic/environmental factors were associated with four main clinical outcomes, giving 20 bivariate comparisons. I considered the possibility of inflated Type I error but did not apply a formal multiple-comparison adjustment because the analyses were exploratory and hypothesis-generating. As a conservative sensitivity reference, a Bonferroni correction across 20 tests would correspond to  $\alpha=0.0025$ . Because this was a cross-sectional study, I use association language only and do not interpret these analyses as showing temporality or causality. I therefore interpret *p* values together with effect sizes, confidence intervals, sample size and the overall pattern of results rather than as stand-alone evidence.

Sample-size and power calculation

Using Fisher's *z* method for a two-sided test ( $\alpha=0.05$ , 80% power) and a moderate-to-large target correlation of  $r=0.43$ , the calculated requirement was  $n = [(1.96 + 0.84)/\text{atanh}(0.43)]^2 + 3 = 40.1$ , rounded up to 41 participants. I therefore considered the available sample of 41 adequate for detecting moderate-to-large associations, but not for smaller effects. I also expected lower power for Fisher's exact analyses because several categorical subgroups were small and unequal. For this reason, I treated these analyses as exploratory and did not interpret non-significant findings as evidence that no association exists.

Results

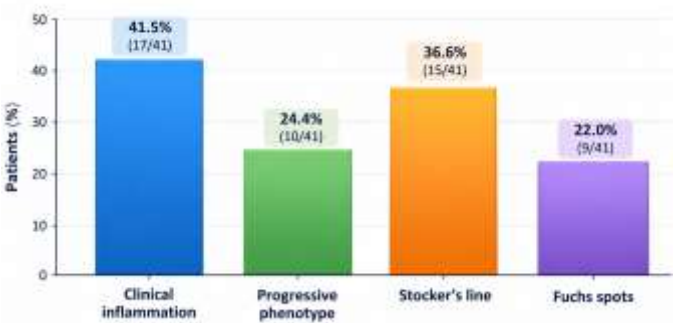
Forty-one patients with primary pterygium were included. Their mean age was  $52.15 \pm 14.36$  years. Twenty-four patients (58.5%) were female

and 17 (41.5%) were male. Mean daily sun exposure was  $5.54 \pm 1.00$  hours. Fifteen patients (36.6%) were smokers, while 27 (65.9%) reported toxic chemical exposure. Clinically, 17 patients (41.5%) had inflamed pterygium. Thirty-one lesions (75.6%) were atrophic and 10 (24.4%) were progressive. Stocker's line was seen in 15 patients (36.6%) and Fuchs spots in 9 (22.0%). Most lesions were advanced in corneal extension, with 37 of 41 (90.2%) classified as Type 3. Forty lesions (97.6%) were nasal.

Table I. Demographic, environmental and clinical characteristics of the study group (n=41)

| Characteristic           | Result                |
|--------------------------|-----------------------|
| Age                      | 52.15 ± 14.36 years   |
| Female                   | 24 (58.5%)            |
| Male                     | 17 (41.5%)            |
| Daily sun exposure       | 5.54 ± 1.00 hours/day |
| Smoking                  | 15 (36.6%)            |
| Toxic chemical exposure  | 27 (65.9%)            |
| Clinical inflammation    | 17 (41.5%)            |
| Atrophic phenotype       | 31 (75.6%)            |
| Progressive phenotype    | 10 (24.4%)            |
| Stocker's line           | 15 (36.6%)            |
| Fuchs spots              | 9 (22.0%)             |
| Type 3 corneal extension | 37 (90.2%)            |
| Nasal location           | 40 (97.6%)            |

Figure 1. Frequency of the main clinical features of primary pterygium in the study group (n=41).



For age and daily sun exposure, I found only small to modest associations, and the confidence intervals were wide. For age, Spearman *r* ranged from -0.051 to 0.226 across the four clinical outcomes. The

largest estimate was for Stocker's line ( $r=0.226$ , approximate 95% CI -0.088 to 0.499;  $p=0.156$ ). For daily sun exposure,  $r$  ranged from -0.050 to 0.136. All approximate 95% confidence intervals included zero, so the data were compatible with both small negative and small positive associations. For sex, smoking and toxic chemical exposure, I assessed whether each factor was associated with the clinical outcomes using exact Fisher tests because of the sparse cells, and I present conditional odds ratios with exact 95% confidence intervals in Table II. The confidence intervals were wide and all included the null value of 1, showing substantial uncertainty. The closest result was toxic chemical exposure with progressive rather than atrophic phenotype (conditional OR=0.24, exact 95% CI 0.04 to 1.32;  $p=0.064$ ). Because the original thesis did not retain the 2 x 2 cross-tabulations, I derived these categorical odds ratios from the unique tables compatible with the reported margins and Fisher p values to the reported precision; I therefore treat them as secondary reconstructed estimates. Because I examined 20 bivariate comparisons without a formal multiplicity adjustment, I also treat every p value as exploratory and do not interpret an isolated p value as definitive evidence for or against a relationship.

Table II. Effect estimates and exact tests for demographic and environmental factors in relation to clinical morphology

| Clinical feature      | Age r (95% CI); p                | Sun exposure r (95% CI); p       | Sex OR (95% CI); exact p              | Smoking OR (95% CI); exact p          | Toxic exposure OR (95% CI); exact p   |
|-----------------------|----------------------------------|----------------------------------|---------------------------------------|---------------------------------------|---------------------------------------|
| Clinical inflammation | 0.023 (-0.287 to 0.328) p=0.886  | -0.050 (-0.352 to 0.262) p=0.756 | OR 1.02 (95% CI 0.24 to 4.37) p=1.000 | OR 0.91 (95% CI 0.20 to 3.94) p=1.000 | OR 0.38 (95% CI 0.08 to 1.71) p=0.189 |
| Progressive phenotype | -0.051 (-0.353 to 0.261) p=0.753 | 0.089 (-0.225 to 0.386) p=0.579  | OR 0.38 (95% CI 0.06 to 1.98) p=0.270 | OR 1.21 (95% CI 0.20 to 6.46) p=1.000 | OR 0.24 (95% CI 0.04 to 1.32) p=0.064 |
| Stocker's line        | 0.226 (-0.088 to 0.499) p=0.156  | -0.006 (-0.313 to 0.302) p=0.972 | OR 0.31 (95% CI 0.06 to 1.34) p=0.749 | OR 1.25 (95% CI 0.27 to 5.59) p=0.749 | OR 1.06 (95% CI 0.23 to 5.22) p=1.000 |

|             |                                 |                                 |                                        |                                       |                                       |
|-------------|---------------------------------|---------------------------------|----------------------------------------|---------------------------------------|---------------------------------------|
|             |                                 | p=0.102                         |                                        |                                       |                                       |
| Fuchs spots | 0.018 (-0.291 to 0.324) p=0.913 | 0.136 (-0.179 to 0.426) p=0.397 | OR 1.54 (95% CI 0.27 to 11.21) p=0.711 | OR 0.43 (95% CI 0.04 to 2.75) p=0.445 | OR 1.05 (95% CI 0.18 to 7.72) p=1.000 |

Age and sun exposure are reported as Spearman  $r$  with approximate 95% confidence intervals and two-sided p values. For categorical comparisons, I report conditional odds ratios with exact 95% confidence intervals followed by two-sided Fisher's exact p values. Odds ratios compare female with male, smoker with non-smoker, and toxic exposure present with absent; for outcomes, the comparison is feature present versus absent, except progressive phenotype, which is progressive versus atrophic. Because the parent thesis does not reproduce the original 2 x 2 cross-tabulations, I reconstructed the unique 2 x 2 table compatible with each pair of reported marginal totals and Fisher p value to the reported three-decimal precision. I therefore regard the categorical odds ratios as secondary reconstructed estimates and interpret them cautiously. All p values are unadjusted for the 20 exploratory bivariate comparisons.

Discussion

The main finding in my exploratory analysis was that I did not detect statistically significant associations between the demographic or environmental factors examined and the clinical appearance of primary pterygium. However, my sample had limited power for small effects, so I do not interpret the absence of statistical significance as evidence that these factors are unrelated to pterygium morphology. This does not mean that sunlight is unimportant in pterygium. Current evidence still supports ultraviolet exposure as a major factor in the development of the disease [11,12]. Zeng and colleagues also found that solar radiation and several air pollutants were related to increased pterygium visits in a large population study. A recent global analysis estimated a substantial worldwide burden and again showed important variation with age and geography. My study addressed a different and much narrower question because all 41 participants already had unilateral primary pterygium and had been selected for surgical excision[13]. Therefore, my data cannot determine whether the exposures studied cause or contribute to the development of pterygium in the general population.

One reason may be that most patients in this study had similar levels of sunlight exposure. Daily sun exposure was concentrated near the higher end of the recorded range, so there was limited variation between patients. This makes it difficult to show a dose-response relationship within a small group. The clinical appearance of pterygium is also likely to depend on local tissue changes. Ha and Kim showed that different pterygium morphologies were related to different dry-eye findings [14]. Han and colleagues found measurable vascular differences between mild and severe lesions [15]. Yun and colleagues reported a relationship between complement C3 and stromal severity [16], while Rahman and colleagues found greater tear-film and meibomian gland changes with higher pterygium grade [17]. Other recent studies using clinical grading, histopathology, anterior-segment OCT and tissue analysis also support a link between morphology, inflammation and structural change [18]. Toxic chemical exposure gave the closest categorical result for progressive rather than atrophic phenotype (conditional OR=0.24, exact 95% CI 0.04 to 1.32; Fisher's exact  $p=0.064$ ). I do not consider this a positive or definitive finding because the confidence interval is wide and includes 1, and the progressive group contained only 10 patients. The other categorical odds ratios were also imprecise, with all exact 95% confidence intervals including 1. In addition, I performed 20 bivariate comparisons. I considered multiple-comparison adjustment but did not apply a formal correction because the analysis was exploratory. Because my study was cross-sectional, I interpret these findings only as possible associations and not as evidence of temporality or causality. I therefore interpret the effect estimates, their confidence intervals and the overall pattern of results together rather than treating any isolated  $p$  value as evidence of a definitive relationship. Recent research also shows why a simple clinical label may not explain every aspect of pterygium. Histopathological examination can occasionally identify unexpected epithelial or neoplastic changes [19], and newer molecular reviews describe important roles for angiogenesis, extracellular-matrix remodeling and inflammatory pathways [12,20]. Morphological measurements have also been linked with postoperative corneal shape and optical recovery, while large clinical series continue to show that presentation and surgical outcome vary between patients [21]. These findings support the idea that environmental exposure is only one part of the overall clinical picture. My study has some limitations. The sample was small, especially for progressive pterygium and Fuchs spots, which limited the precision of the estimates and the ability to detect small effects. My exposure measurements were also limited. I recorded sun

exposure only as average hours per day and could not distinguish occupational from recreational exposure or estimate cumulative or lifetime ultraviolet exposure. Smoking was recorded only as smoking status, without current/former classification, duration, cigarettes per day or pack-years. Toxic chemical exposure was recorded only as present or absent; the type of chemical, source, route, duration, frequency and intensity were not available. Because of these limitations, I could not examine dose-response relationships for smoking or chemical exposure, and some exposure misclassification is possible. Most lesions were nasal and Type 3, so I could not compare those variables properly. I did not fit a multivariable model because the small number of events in some outcomes would have produced unstable adjusted estimates. The parent thesis did not retain the original  $2 \times 2$  cross-tabulations for the categorical analyses. I therefore reconstructed the categorical odds ratios from the unique tables compatible with the reported marginal totals and Fisher  $p$  values to the reported precision; these should be regarded as secondary reconstructed estimates rather than independently verified patient-level effect estimates. I also tested 20 bivariate associations without a formal multiplicity adjustment, so I treated the analysis as exploratory and interpreted the results from the effect sizes, confidence intervals and overall pattern rather than nominal  $p$  values alone. In addition, all 41 participants had been selected for surgical excision, so my study population represents surgical/clinically significant pterygium rather than all patients with pterygium. This selection limits generalizability. The clinical distribution was also highly unbalanced: 37 of 41 lesions (90.2%) were Type 3, only 4 were Type 2, none were Type 1, and 40 of 41 lesions (97.6%) were nasal with only one temporal lesion. I therefore did not have enough variation to make meaningful comparisons across corneal extension grades or between nasal and temporal sites. Within this selected group of 41 surgical patients, my findings suggest that the demographic and environmental factors I examined were not clearly associated with the clinical morphology recorded before excision. However, because every participant already had unilateral primary pterygium and was selected for surgery, I cannot use these findings to make conclusions about all Pakistani adults, about patients who are not selected for surgical excision, or about the development of pterygium in the general population. The sample size was small and the clinical groups were unequal. Although 41 participants provided approximately 80% power to detect moderate-to-large correlations (about  $r=0.43$ ), my study was underpowered for smaller effects, and several categorical comparisons were sparse. I did

not perform multivariable adjustment because of the low number of events in some outcomes [22]. I evaluated 20 bivariate associations and considered the risk of false-positive findings from multiple comparisons. I did not apply a formal Bonferroni, false-discovery-rate or other multiplicity correction because these analyses were exploratory rather than confirmatory; therefore, all reported p values are unadjusted. I used a Bonferroni alpha of 0.0025 only as a conservative sensitivity reference and did not interpret isolated nominal p values as definitive evidence of relationships. For categorical analyses, the parent thesis did not preserve the original 2 x 2 cross-tabulations. The odds ratios and exact 95% confidence intervals shown in Table II were therefore reconstructed from the unique 2 x 2 tables compatible with the reported margins and Fisher p values to the reported precision and should be interpreted cautiously. Exposure assessment was limited because sun exposure was recorded only as average hours per day without separation of occupational and recreational exposure or estimation of cumulative ultraviolet exposure. Smoking was recorded only as smoking status, without current/former classification, duration, cigarettes per day or pack-years. Toxic chemical exposure was recorded only as present or absent, without documentation of the specific agent, source, route, duration, frequency or intensity. I therefore could not evaluate dose-response relationships for these exposures. I consider these findings hypothesis-generating and do not interpret a non-significant result as proof that no association exists. In addition, the parent thesis did not document the number of clinical assessors and no interobserver agreement analysis was reported. The surgical selection of participants is an additional limitation, and the predominance of Type 3 lesions (37/41) and nasal lesions (40/41), with no Type 1 lesions and only one temporal lesion, prevented reliable comparison across clinical grades and sites.

## Conclusion

This cross-sectional study sample of 41 patients with unilateral primary pterygium who were selected for surgical excision, I found that age, sex, daily sun exposure, smoking and toxic chemical exposure were not clearly associated with clinical inflammation, atrophic or progressive phenotype, Stocker's line or Fuchs spots. Because I performed multiple exploratory comparisons without formal multiplicity adjustment, I do not interpret any isolated p value as definitive evidence of a relationship, and because my sample was mainly able to detect moderate-to-large effects, I cannot exclude smaller associations. Because the study was

cross-sectional, I do not interpret these findings as evidence of temporality or causality. I therefore limit my conclusion to these 41 selected surgical patients. I do not generalize these findings to all Pakistani adults, to patients with bilateral, non-surgical or otherwise different pterygium, or to the role of these exposures in the development of pterygium in the general population. Larger, prospectively designed and adequately powered studies would be needed to test these possible associations confirmatorily.

## Authors' Contributions

**Sumera Khalil:** Conceptualization, study design, data collection, histopathological assessment, interpretation of findings, manuscript drafting, critical revision of the manuscript, final approval of the version to be published, and accountability for all aspects of the work.

**Sadia Aman:** Study design, histopathological assessment, data curation, literature review, interpretation of findings, manuscript revision, final approval of the version to be published, and accountability for all aspects of the work.

**Nafeesa Kiran & Sanah Sameen:** Data collection, histopathological assessment, data curation, literature review, manuscript drafting and revision, final approval of the version to be published, and accountability for all aspects of the work.

All authors: Reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work in accordance with ICMJE authorship criteria.

## Ethical Approval

The Ethical Review Committee of the Rawalpindi Medical University, Rawalpindi, Pakistan (RMU/Reg-19/Erc/2611/09/2024).

## Conflict of Interest

The authors declare no conflict of interest.

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