

RESEARCH ARTICLE

Histopathological Study of Primary Pterygium

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ABSTRACT

Background: A graded series of ocular surface changes has been described throughout the bulbar conjunctiva. The most advanced changes occurring directly over the pterygium surface, confirming that pterygium is indeed an ocular surface disorder.

Purpose: Pterygium is characterized as invasive, proliferative fibro-vascular altered conjunctival tissue. The extensive vascular network is likely to significantly contribute to the progression of the disease. The present study aims to correlate the findings obtained from studying the epithelium of true primary pterygium by impression cytology technique and microscopic picture.

Design: Prospective clinical study.

Methods: This study is a prospective study designed to analyze data of 20 eyes of 20 patients presenting to the Department of Ophthalmology at “Ain Shams University” and at the “Research institute of ophthalmology” with clinically diagnosed pterygium.

Results: Histopathological study identified pathological change in epithelium and subepithelial fibrovascular tissue of the pterygium.

Conclusion: Histopathological examination of the epithelium of pterygia revealed epithelial changes most prominent directly above the head of pterygium.

Keywords: Epithelium, Histopathology, Pterygium

Introduction

Pterygium is a common ocular surface disease in subtropical countries and is a triangular fibrovascular sub-epithelial ingrowth of degenerative bulbar conjunctival tissue over the limbus onto the cornea [1].

It typically develops in patients who have been living in hot climates and may represent a response to ultraviolet exposure and to other factors such as chronic surface dryness. Histologically, a pterygium shows elastotic degenerative changes in vascularized sub-epithelial stromal collagen. Pterygia encroach onto the cornea and invade the Bowman layer [2].

Epidemiological evidence suggests that chronic sunlight exposure without ultraviolet (UV) radiation protection a crucial role [3].

A pterygium consists of three distinct parts: the cap, the head and the body/tail. The cap or leading edge is a flat zone on the cornea that consists mainly of fibroblasts that invade and destroy Bowman’s membrane. The head is a vascular area that lies behind the cap and is firmly attached to the cornea. The body/tail are the mobile areas of the bulbar conjunctiva, which can be easily dissected from the underlying tissue. Stocker’s line, which is iron deposition in the basal layer of corneal epithelium

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anterior to the cap, indicates that the pterygium is chronic [4]. Squamous metaplasia with goblet cells was observed in specimens obtained from pterygia [5].

Common histological features observed included a proliferative and locally invasive front of pterygium epithelium that abruptly transitioned into corneal epithelium at the advancing edge. At the junction between the pterygium epithelium and normal cornea, the stroma was often characterized by feeder blood vessels that preceded the fibroblastic stroma. The advancing pterygium edge was demarcated by a fragmented Bowman's layer. Goblet cell hyperplasia was prominent in pterygium epithelium, compared with autologous normal conjunctiva. Feeder vessels extending along the length of the lesion were regularly noted, as well as sub-epithelial neovascularization. Stromal elastosis and both intra- and sub-epithelial and intravascular inflammation were present in 60% of cases [6].

A graded series of ocular surface changes has been described throughout the bulbar conjunctiva. The most advanced changes occurring directly over the pterygium surface, confirming that pterygium is indeed an ocular surface disorder [7].

Methods

This study is designed to analyze data of 20 eyes of 20 patients presenting to the Department of Ophthalmology at "Ain Shams University" and at the "Research institute of ophthalmology" with clinically diagnosed pterygium.

The patients will undergo post-operative histopathology and microscopic study.

Study parameters include

- Age and sex.
- Complete ophthalmic examination.
- Slit lamp examination of pterygium to detect any of exclusion criteria.
- Microscopical study after surgical excision.

Results

Histopathological results

See Figures 1-10.

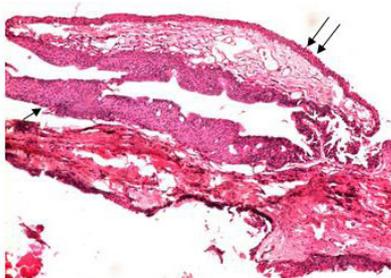


Figure 1: Pterygium (arrow) with part of conjunctiva lying on (double arrows) (H and E 500X).

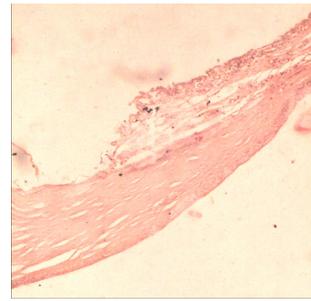


Figure 2: Pterygium attached to part of cornea (H and E 125X).

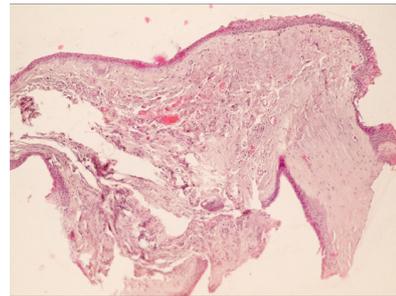


Figure 3: Classic pterygium (H and E 125X).

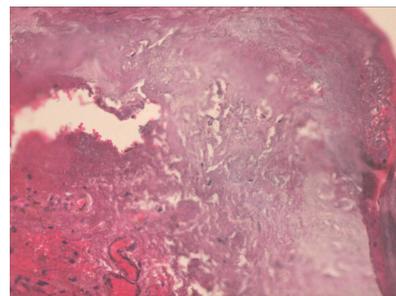


Figure 4: Pterygium elastosis (H and E 500X).

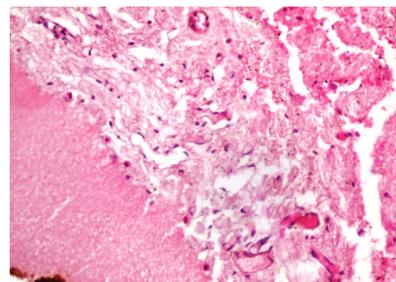


Figure 5: Pterygium with blood clot infiltrated by chronic inflammatory cells, with degenerated collagen (H and E 500X).

Discussion

This work identified goblet cell hyperplasia and squamous metaplasia with epithelial hyperplasia that is consistent with Wang and Chan findings in IC samples obtained from pterygia surfaces and that reported conjunctival squamous metaplasia and goblet cells without atypical cells [7,8].

Chui *et al.* [9] stated that common histological features observed included a proliferative and locally invasive front of pterygium epithelium that abruptly transitioned into corneal epithelium at the advancing edge. At the

junction between the pterygium epithelium and normal cornea, the stroma was often characterized by feeder blood vessels that preceded the fibroblastic stroma. The advancing pterygium edge was demarcated by a fragmented Bowman's layer. Goblet cell hyperplasia was prominent in pterygium epithelium, compared with autologous normal conjunctiva. Feeder vessels extending the length of the lesion were regularly noted, as well as sub epithelial neovascularization. Stromal elastosis and both intra- and sub epithelial and intravascular inflammation were present in 60% of cases which were almost the same results in this study.

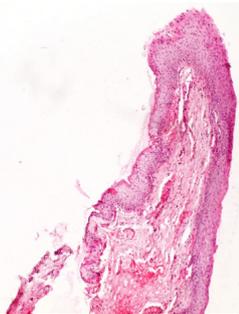


Figure 6: Pterygium formed of stratified squamous epithelium as a cover, and a core of connective tissue stroma with multiple dilated vessels (H and E 125X).

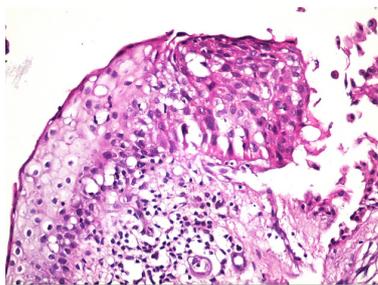


Figure 7: Pterygium exhibiting mild dysplastic changes (H and E 500X).

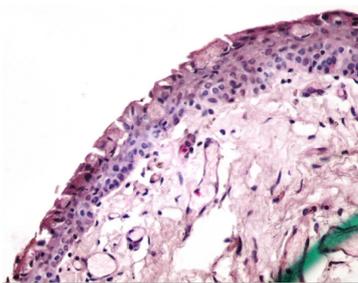


Figure 8: Pterygium formed of stratified squamous epithelium as a cover, and a core of connective tissue stroma with multiple dilated vessels (H and E 500X).

This thesis identified several previously documented histopathological features in pterygia. Common findings included a prominent migratory front of actively proliferating and locally invasive epithelium with evidence of Bowman's layer dissolution, which were found in two studies by Di Girolamo *et al.* [10,11]. These authors found that these features are mediated by the activity of UV-induced matrix metalloproteinase.

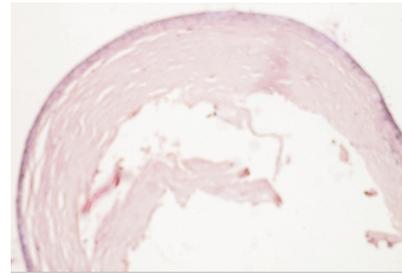


Figure 9: Control cornea (H and E 125X).

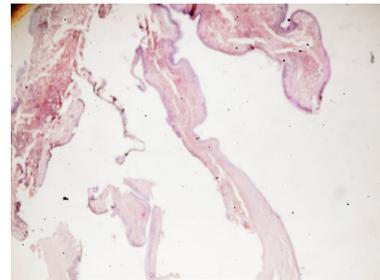


Figure 10: Conjunctival - corneal junction (H and E 125X).

Other features included a reactive fibrovascular stroma with evidence of elastosis, and intravascular, sub epithelial, and intraepithelial leukocyte infiltration, likely mediated through UV-induced cytokines and growth factors [12,13]. These findings are congruous with the results of the current study, which detected elastosis in 65% and chronic inflammatory cells in 100% of cases.

According to the results of the present study, pterygium is mostly covered by altered conjunctival epithelium that exhibited squamous metaplasia with epithelial hyperplasia in 40% of cases, which is in accordance with literature data of Dzunic *et al.* [14].

This study demonstrated elastosis (histopathologic evidence of solar injury) observed in 65% patients, confirming sun damage to the ocular tissue. This finding helps explain the rate of pterygia among sun-exposed workers and farmers, since sunlight exposure is a common etiological factor in the disease [15].

McKelvie *et al.* [5] have reported elastosis in 100% of their specimens, while a previous Brazilian study found elastosis in 81.4% [16]. In comparison, Tabrizi reported elastosis in 50% of the cases [17], whereas, Barros *et al.* [18], observed elastosis in all pterygia.

Golu found areas of hyperplasia, prone to pseudokeratinization. More than two thirds of patients with pterygium showed numerous goblet cells, either diffusely scattered or clustered in the form of intraepithelial glands [19]. These findings are consistent with our results of squamous metaplasia 40%, epithelial hyperplasia 40% and goblet cell hyperplasia 45%.

Chan *et al.* [7] results showed squamous metaplasia in 73.2% of pterygium cases, compared to our results of 40% of cases. The increased goblet cell density over the pterygium is similar to our results.

This work identified chronic inflammatory cells in 100% of our cases. Anguria showed that chronic inflammatory cells were found to be scattered unevenly in all samples. Mild chronic inflammation has a tendency to be more frequent than severe inflammation in pterygia [20].

Conclusion

Analysis of morphological characteristics of pterygium with the gold standard histopathologic examination is essential but the drawback is that histopathology is invasive.

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