

Eyelid Alterations in Involutional Ectropion

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Abstract

Purpose: To describe the histological features of eyelid tissue in involutional ectropion carriers.

Methods: An interventional case series study was performed evolving 52 involutional eyelid ectropion carriers submitted to Tarsal strip surgery. A full-thickness eyelid fragment was evaluated by light microscope and results were statistical analyzed.

Results: The conjunctival substantia propria had an inflammatory mononuclear process. Meibomian glands and eyelash follicles were enlarged. Inflammatory cells permeated the glands and eyelashes follicles. Orbicularis muscle fibers were replaced by fibrosis in some areas. Solar elastosis and epithelial squamous cell metaplasia were also observed in the skin epithelium.

Conclusion: Eyelid ectropion is associated with important lid margin alterations affecting the glands, eyelashes, and orbicularis muscle. Inflammation and fibrosis compromise and disturb eyelid function.

Keywords: Lid; Involutional ectropion; Histology; Clinicopathological study

Introduction

Involutional eyelid ectropion is a very common eyelid positional deformity. The literature generally focuses on surgical management and little is known about histological changes associated with this chronic eyelid malposition.

Lack of lid contact with the ocular bulb is related to many important physiologic alterations. The eyelid glands do not have the same function and it is very common to have associated inflammatory processes in the eyelid margin, including meibomitis, blepharitis, misdirected lashes as well as secondary changes to the ocular surface, as keratitis and keratoconjunctivitis.

Many times these changes do not solve when the margin is surgically replaced to its normal position, suggesting the eyelid alterations do not recover with repositioning.

Microscopic histology of full thickness surgical eyelid ectropion and entropion specimens showed chronic inflammation and scarring in the both situations. [1] The main histological features associated with ectropion are: collagen degeneration, tarsal plate elastosis, increased adipose tissue in the tarsus and capsulopalpebral fascia, subacute inflammation and epidermalization of the tarsal conjunctiva, focal degeneration, fibrosis and elastosis of the pretarsal orbicularis and Riolan's muscle, and arteriosclerosis of the marginal artery. [2,3]

Trichiasis and misdirected eyelashes associated with ectropion suggest more alterations in the pilous follicles and orbicularis muscle in this area.

The purpose of this study is to show the histological features of eyelid tissue in involutional ectropion carriers.

Materials and Methods

This is an interventional case series report. Fifty-two involutional eyelid ectropion carriers were submitted to surgical eyelid ectropion correction by Tarsal Strip procedure.

After surgery the full-thickness eyelid fragments removed from

each patient were fixed in 10% formaldehyde for at least 2 hours, and then embedded in paraffin. Systematic serial sections were obtained and stained with hematoxylin/eosin and Masson-Trichromium. The same pathologist evaluated all samples by light microscopy.

The majority of patients were male (75.9%) up to 60 years old (92.6%).

Results were statistical analyzed according to frequency of occurrence.

Results

Eyelid fragments removed in the Tarsal strip surgery consisted in full thickness eyelid portions, containing epidermis, dermis, eyelid glands, eyelashes follicles, orbicularis muscle, tarsus and conjunctival tissue.

Histological evaluation showed keratinized conjunctiva and inflammatory cells in the conjunctival substantia propria in all specimens (100.0%) and surrounding Meibomian glands and the eyelashes follicles in the majority of the patients (63.4%) (Figure 1).

The Meibomian eyelid glands were frequently enlarged and hyperplastic (76.9%) as well as the eyelashes follicles (67.5%) (Figures 1,2).

The connective tissue between the glands and follicles increased. The orbicularis muscle fibers were replaced by fibrosis in 92.6% of the

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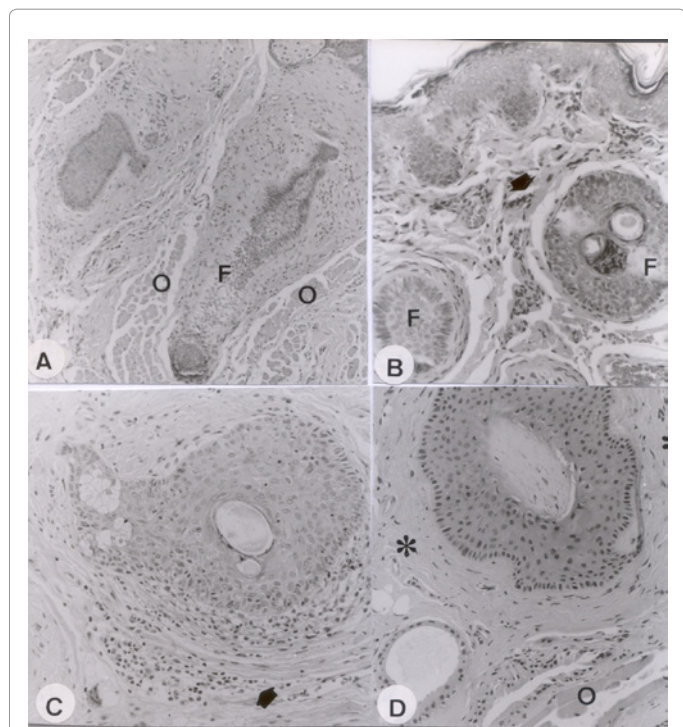


Figure 1: Lower lid of an involuntional ectropion carrier: (A) The conjunctival substantia propria with inflammatory infiltrate (arrow) (HEX20); (B) Meibomium glands (M) surrounded by orbicularis muscle (O) and inflammatory infiltrate in the interstium (HEX40); (C) Inflammatory cells and fibrosis (*) around the eyelid glands (HEX40); (D) Fibrosis (*). Orbicularis muscle (O) around glands (HE X100).

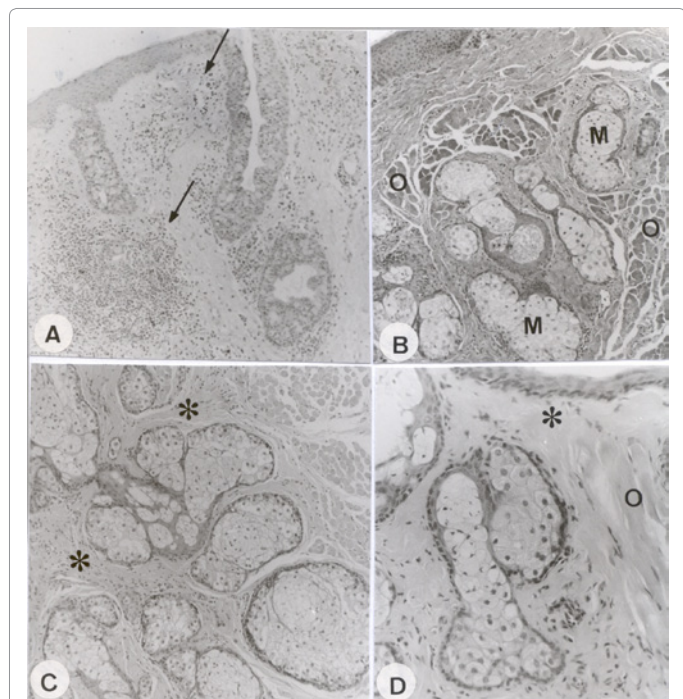


Figure 2: Lower lid of an involuntional ectropion carrier: (A) Lashes follicles (F) surrounded by orbicularis muscle (O) (HEX20); (B) Pilous follicles surrounded by inflammatory cells (large arrow) (HE X40); (C) Inflammatory cells (large arrow) around the lash follicle (HEX40); (D) Fibrosis (*) around the lash follicle. Orbicularis muscle (O) (HEX100).

analyzed areas, mainly when located around the lashes follicles and glandular tissue (Figure 2).

Solar elastosis (65.3%) and epithelial squamous cell metaplasia (55.7%) were also observed in the skin epithelium.

Discussion

There are few studies about the histology alterations due to eyelid ectropion. One of them focuses the tarsus and orbicularis muscle [2] and another one the role of the capsulo-palpebral fascia and lower lid retractors in the genesis of the eyelid ectropion [4]. Nevertheless the lid margin was not the main subject of those authors, our findings are consistent and very similar to another study about eyelid histopathology in ectropion and entropion carriers [3].

Another study is about the orbicularis muscle and tracomatous cicatricial ectropion,⁵ an entity with very different genesis to involuntional ectropion.

Our study confirmed previously described histological aspects [1,2,3] and supports clinical suspicions that important changes exist in the ectropion lid margin. Congested glands having inflammatory cells, clinically seen as meibomitis which is associated to ectropion were present in all our cases. Conjunctiva metaplasia provokes glandular obstruction and enlargement succeeded by stasis and inflammation, frequent alterations seem in chronic longstanding ectropion and resulting in exposure and irritation.

It was also very common to observe inflammatory cells around the eyelash follicles. The alterations in the conjunctiva substantia propria were very interesting – fibrosis and significant changes in Riolan’s muscle, a portion of the orbicularis muscle which involved eyelash follicles, supporting them. Nevertheless the Riolan’s muscle was seen without alteration in ectropion carriers in the past, [2] our study confirmed it is often replaced by fibrosis which might be the cause of misdirected eyelashes observed in the ectropion carriers.

Anatomical factors at the orbicularis muscle are supposedly evolved in ectropion eyelid development [6]. Probably lid laxity is secondary to orbicularis atrophy, degeneration, and fibrosis combined with tarsal degeneration secondary to fragmentation of collagenous fibers and elastogenesis by aging fibroblasts. Besides the fibrotic muscle degeneration observed in the present study, using specific staining method (like van Gieson) it will be easier to visualize differentiation between the elastic and collagen fibers.

According to others, orbicularis atrophy or fibrosis might be related to arteriosclerotic and partially occluded marginal artery suggesting an ischemic basis to the muscle atrophy [2]. However, the chronic inflammation is very common and probably will contribute to orbicularis weakness in the ectropion carriers making the possibility to have ischemia or paralysis primarily with inflammatory changes contributing probably in later stages.

Cutaneous changes such as solar elastosis were also frequent, probably due to the fact that most of our patients were light skinned and living in a Brazilian solar environment. Cutaneous elastosis may lead lid laxicity worsening the lid position. Tarsal conjunctiva grows beyond the gray line, probably because the conjunctival tissue is exposed to the solar radiation.

Our study did not notice whether one eye or both eyes were operated in each patient and do not note the degree of ectropion related to the histopathology alterations detected. Furthermore, it would be

interesting to know if there is any sign of chronic marginal blepharitis on the fellow eye also.

Other important points to figure out in next studies evolving histopathology and eyelid position alterations would be the condition of elastic fibers and goblet cell loss, since the last one is a marker of the chronic inflammation in the tarsal and bulbar conjunctiva.

Even when the eyelid is surgically repositioned, lid alterations frequently remain probably because all histological alterations are permanent and will not change because of lid position correction.

Therefore, definitive histological alterations might be the reason for persistent inflammatory eyelid conditions (meibomitis, blepharitis) and eyelash misdirection, even after successful eyelid ectropion correction surgery.

Conclusion

A lack of eyelid opposition to the ocular bulb promotes many important alterations, including glandular and eyelash follicles

enlargement, secretion stasis, and inflammatory tissue reaction in both glandular tissue and the substantia propria, resulting in fibrosis which compromises the orbicularis muscle and disturbs eyelid function.

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