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Pretarsal and marginal orbicularis oculi muscle fiber changes in trachomatous cicatricial entropion: histopathological evaluation

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ABSTRACT: Purpose. *To evaluate the histopathological changes of pretarsal and orbicularis muscle fibers in trachomatous cicatricial entropion.*
Methods. *Orbicularis muscle tissue specimens were histopathologically evaluated in 17 eyes of 11 cases in which anterior lamellar reposition and/or wedge-shaped tarsal resection or Wies procedure were performed.*
Results. *Degeneration of orbicularis muscle fibers, atrophy, connective tissue increase between muscle fibers and edema were observed in 13 tissue specimens. Muscle fiber changes were more commonly observed in cases with severe entropion, in which Wies procedure was performed.*
Conclusions. *These histopathological changes, which may develop secondary to other structural changes that can cause entropion in the eyelid and weaken the orbicularis muscle, may be a co-factor influencing the severity of entropion more than causing entropion alone. (Eur J Ophthalmol 1999; 9: 89-92)*

KEY WORDS: Trachoma, Cicatricial entropion, Orbicularis oculi muscle, Histopathological evaluation

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INTRODUCTION

Trachoma is one of the most common causes of preventable blindnees. The reson for blindness in trachoma is corneal scarring due to entropion and trichiasis. In this immunopathological disease caused by *Chlamydia trachomatis*, severe scar tissue formation is observed in the tarsal conjunctiva as a result of relapsing infections and superinfections of other microorganisms. In this clinical situation where there is a total thickening of the eyelid, the main change is seen in the conjunctiva and tarsal plate (1). Eyelids and eyelashes distort towards the globe because of developing cicatrization and vertical contracture of posterior eyelid lamella (2). There are few studies reported, which investigate the histopathology of trachomatous entropion, however, these contain changes in the palpebral conjunctiva, tarsal plate and Meibomian glands. In cicatricial entropion, which has a very complex and dynamic mechanism, the normal balance between anatomic and physiologic factors which keep the eyelid in the normal position are damaged. Posterior lamellar changes are not alone; they occur together with secondary anterior lamellar tissue changes. It is the purpose of this study to report the histopathological evaluation of muscle fiber changes in cases that underwent surgery for moderate cicatricial entropion.

METHODS

Between 1996 and February 1998 a total of 17 eyes in 11 patients who had been submitted to surgery with

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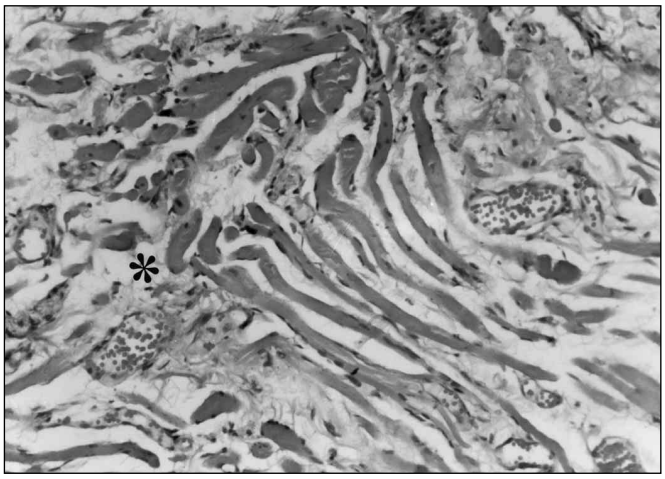

◀ **Fig. 1 -** *Edema (*) occasional muscle fibre atrophy and thinning are seen between orbicularis oculi muscle fibres which do not have morphological structure in case with trachomatous entropion (Hematoxylin-eosin, x200).*

Fig. 2a - *Dense fibrosis around orbicularis oculi muscle fibres (*) and replacement of muscle fibres with connective tissue in some areas. (Hematoxylin-eosin, x200).*
Fig. 2b - *Connective tissue increase with abundant collagen is seen with Masson-trichrome dye in the same region (arrow). (Masson-trichrome, x100).*

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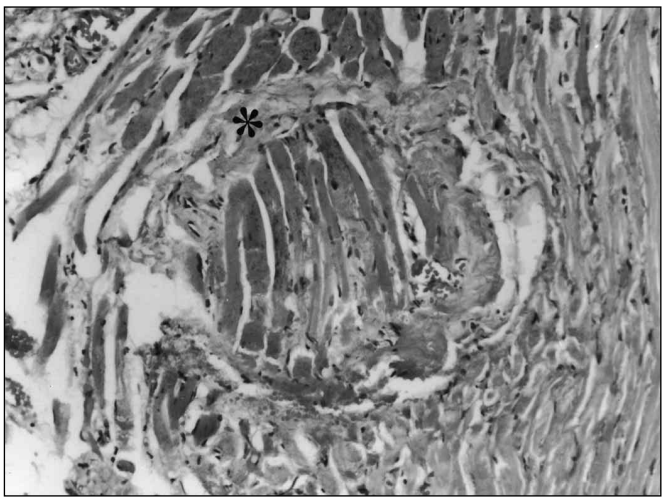

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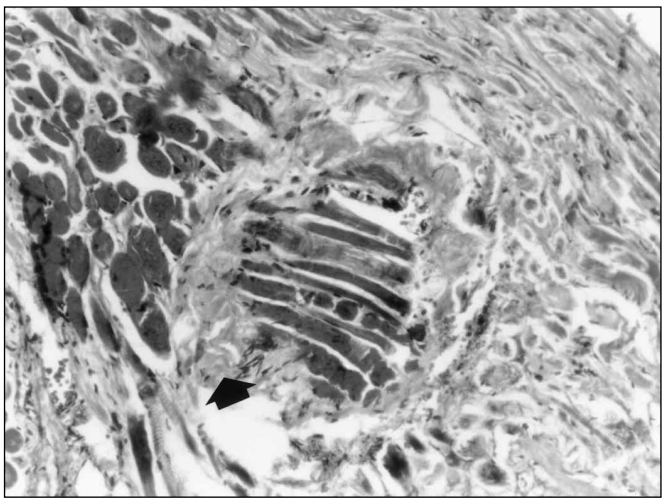

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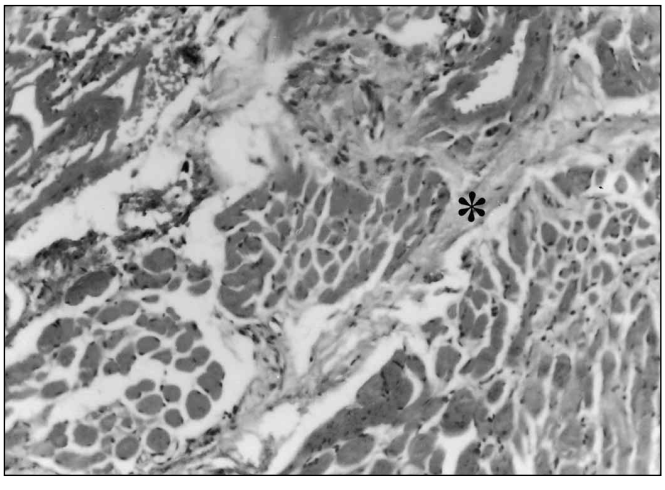

Fig. 3 - *Connective tissue invasion and edema are seen between the muscle fibres (*). (Hematoxylin-eosin, x200).*

a diagnosis of moderate degree trachomatous upper eyelid entropion were evaluated. There were 6 females and 5 men, and age ranged from 51 to 77 years (mean 58 years).

Wies operation (the procedure is to split the lid transversly to create a fibrous tissue scar barrier which prevents the upward movement of the preseptal muscle; this is combined with everting sutures which shorten the lower lid retractors and transfer their pull to the upper border of the tarsus) (3) was performed in 6 eyes, while anterior lamellar reposition and recession with wedge resection was performed in 11. Pretarsal orbicularis oculi muscle tissue specimens from the medial region of the upper eyelid were fixated in 10% formaline for 72 hours, after standard paraffin embedding and serial sectioning, and later dyed with hematoxylin, and Masson trichrome

specimens were microscopically examined and photographed.

For the purposes of a histopathological evaluation, pyknosis of nucleus in striated muscle fibers, degeneration findings such as karyorhexis and karyolysis, loss of cell details and striation, fragmentation of intracytoplasmic formations and membrane structures, presence of edema and connective tissue between muscle fibers, arrangement and integrity of fibers were investigated.

RESULTS

In 8 out of 17 specimens from eyelid muscle tissue, findings of degeneration, loss of striation in some areas, prominent connective tissue increase between muscle fibers and edema were seen. It was also observed that muscle fibers had lost their normal appearance and volume, and were fragmented in some places, and deformed muscle cells were observed to have altered nucleus-cytoplasm ratio and indistinct cell borders (Fig. 1). Connective tissue was seen to be increased in muscle tissue, muscle fibers were invaded and destroyed by fibroblasts extending between them, and had lost their normal configuration (Figs. 2a, 2b, 3). Changes seen in muscle tissue which was poorly vascularized were thought to be partially due to ischemia.

Mild atrophic changes, an increase in connective tissue between fibers and edema were observed in specimens from 5 eyes in which the changes were milder.

In cases where Weiss surgery had been performed, degenerative findings were severe (4 cases) and mild (2 cases). Degenerative findings were severe (4 cases), mild (3 cases), or obsent (4 cases) in 11 eyelids with moderate entropion.

DISCUSSION

The normal shape and appearance of the eyelids are principally maintained by partial mechanical rigidity of the tarsal plates, stretch and tonus of orbicularis oculi muscle fibers laying over the tarsal plates, which are in complete harmony with the ocular surface. There is a physiologic balance between the anterior lamella as a functional unit, consisting in skin and obicularis oculi muscle, and the posterior lamella, which is formed by the tarsus tightly adhered to the palpebral conjunctiva (4).

Cicatricial entropion is secondary inversion of eyelid margin and cilia by vertical contracture and distortion of the posterior eyelid lamella caused by a variety of reasons. It is seen more often and produces more serious problems to the globe in the upper lid (5).

Dense inflammatory reaction and subsequent scar formation are explained by immunopathologic mechanisms more than by the direct cytopathological effect of *Chlamydia* (6). Surgical treatment is indicated for cicatricial entropion. Enabling the lid to come into a normal position by changing the strength balance between the anterior and posterior lamellae, by removing as much scar tissue as possible, by replacing scar tissue, if needed, by using a graft in order to elongate a retracted posterior lamella, are the fundamental principles of this surgery. The most important step in treatment is defining the severity of the pathology, as this provides the basis for surgical planning (5).

The anterior lamella and orbicularis oculi muscle are also affected in trachomatous cicatricial entropion, in which the posterior lamella is primarily affected. The first change observed in the eyelids is a narrowing of the distance between the tarsal interductal line, beginning from the middle eyelid region and the medial conjunctival margin. Thickening of the tarsal plate increases with fibrosis which begins in the subconjunctival tissue like thin spindles and originates from fibroblasts around the blood vessels, to increase with cicatrization of necrotic lymph follicles. Subepithelial contractile fibrous membrane which consists of vertical collagen bands causes the tarsus to rotate and mucocutaneous junction to displace towards the globe. This means that anterior lamellar structures displace inferoposteriorly (2).

Orbicularis muscle function can cause changes in the severity of entropion, and trichiasis in cases with conjunctival scarring. It is pointed out that entropion and trichiasis are less common with orbicularis oculi muscle weakness. Orbicularis oculi muscle strenght has an effect on the development of entropion in cases with cicatrized posterior lamella; there may be structural and functional changes in the orbicularis oculi muscle when it accompanies primary or secondary pathology together with posterior lamellar changes. Pretarsal and marginal fibers can actively take part in rotation of tarsal tissue towards the globe, or they may be passively affected. A period with an increase in interfibrillar con-

nective tissue, secondary degeneration, shortening of fibers and decreasing muscle strength may follow a period of temporary weakness caused by an increase in the orbicularis oculi muscle interfibrillar space and a change in their direction during early contracture when the posterior lamella rotates towards globe (7).

A long-lasting entropion with trichiasis may cause secondary blepharospasm by activating a neurogenic mechanism due to chronic corneal irritation. In serious cases the condition becomes irreversible, and orbicularis muscle fibers also containing pretarsal fibers change irreversibly. Outcoming contracture increases the severity of entropion. In this situation, wide excision of orbicularis muscle fibers is required (8). Reflex or essential blepharospasm can initiate entropion or increase the severity of the present condition (4). This is mostly related to pretarsal muscle fibers, because type 2 fibers responsible for phasic activity in rapid eyeblinking are present mostly in the pretarsal region (9). There may be changes in the ratio of these fibers, and there may also be structural changes along with enzyme abnormalities in entropion. Moreover, it has been determined that ischemic tissue changes are more common in entropion than ectropion (10).

In our study, we found muscle fiber degeneration in many of the pretarsal orbicularis oculi muscle fiber specimens. In addition to muscle cell degeneration, the regularity and appearance of normal striated muscle was distorted, and there were configuration changes in favor of connective tissue. Ischemic findings, like degeneration and atrophy, connective tissue among muscle fibers and edema were observed. It is difficult to explain whether these changes are primary trachomatous changes that derive from the conjunctiva and tarsus, or anterior lamellar changes secondary to posterior lamellar changes.

Histopathological changes in some specimens were more intense than expected more than secondary changes. However, there are no findings concerning primary disease of the orbicularis oculi muscle in trachoma reported. It is hard to thoroughly explain histopathological changes with reactive blepharospasm. Contracture and fibrosis are expected to be seen following a period of hypertrophy, but there was no such finding in our cases.

Involutional changes may have an effect on the continuing trachomatous period. Senile and ischemic alterations in the anterior lamella may cause changes in the situation as a result of their physiologic balancing role. But this is far from explaining the situation alone.

Consequently, during the period of cicatricial trachomatous entropion structural changes are seen in the orbicularis oculi muscle. It is our belief that a large series of ultrastructural and electrophysiological studies are needed in order to reveal these changes, which may be a cause, a result, or both.

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