ULTRASTRURAL APPEARANCE OF THE CORNEA IN ABSOLUTE GLAUCOMA

By

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INTRODUCTION

Glaucoma is a disease characterised by elevation of the intraocular pressure. It is well known that this rise in the intraocular pressure (IOP) is always due to tissue changes which reduces the normal outflow of aquous humours (except in rare cases of hypersecreton glaucoma).

Irrespective of the mechanism causing the rise of IOP, the changes induced in the ocular tissues are usually characteristic. One of the tissues to be affected clinically by the rise of IOP is the cornea.

The purpose of this study is to describe the ultrastructural changes in the cornea in cases of absolute glaucoma due to different causes.

MATERIAL AND METHODS

Three corneas from enuculated eyes diagnosed as absolute glaucoma with different histories were examined.

Case No. 1.

A 46 year old male was seen as an emergency with red eye. Tension was extremly high with deep anterior chamber, old keratic precipitates and only perception of light. He was diagnosed as absolute glaucoma secondary to injury in childhood and received retrobulbar alcohol. Pain recurred again after three months by which time the eye was completly blind and was then enucleated.

Case No. 2

A 67 year old female had cataract extraction; six months later she showed evidence of damage of the epithelium of the cornea probably due to decreased tear secretion. A year later, she developed open angle glaucoma with rapid deterioration of vision and the eye was eventually enucleated.

Case No. 3

A case of long standing uncomplicated glaucoma in a male 65 year

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old, blind-registered for four years. After repeated retrobulbar injection of alcohol for the past three years, the eye being painful and blind was encleated.

Immediately after enucleation, the the cornea was excised and fixed in 4% cold cacodylate buffered glutaraldehyde, post fixed in 2% osmium teroxide, dehydrated and embedded in arladite. Thin sections were double stained with uranyl acetate and lead citrate and examined with Zeis EM/10 electron microscope.

RESULTS

All three cases showed similar ultrastructural changes, though the severity of the changes varied from one case to the other.

In all instances, the epithelium was markedly edematous both intercellularly and intracellularly. The intercellular spaces were dilated and the cells were only attached to each other at the desmosomal junctions (fig. 1). The cytoplasmic organelles, especially the mitochondria, were dilated and swollen and the cytoplasmic tonofibrils were clumped (fig. 2). These changes were sometimes marked that, in the central part of cornea of case no. 1, the epithelium was missing over a great part and only fragments of the basal cells were found to be attached to the basement membrane of the epithelium (fig. 3).

Bowman's layer was found to be separated from the basement membrane of the epithelium by a space containing bundles of fine collagen fibrils (1000A in diameter) and arranged mostly perpendicular to the surface of the cornea, together with basement membrane-like material (fig. 4).

The stroma, generally, showed moderate degree of edema, keratitis and some scarring at the posterior part (fig. 5).

The endothelium, on the other hand, showed marked degree of ultrastructural changes. The cells appeared metabolically active with abundant mitochondria, long chains of rough endoplasmic reticulum as well as well developed Golgi complex. The nucleus was found to lose its cubical shape and had a more or less spindle shape. The cells, themselves, had a flattened appearance and two layers of these modified endothelial cells were frequently seen at the posterior part of the cornea (fig. 6a). The adjacent endothelial cells were found to be attached to each other by normal junctional complex. In addition, groups of collagen fibrils (150 A in diameter) were seen inside these cells. The amount of fibrils varied from one case to the other and, at some places the cells were nearly full of these fibrils as in case no 3 (fig. 7). Similar groups of collagen fibrils were also seen outside these cells mainly near their anterior border. Moreover, groups of fine filaments (50 to 100 A in diameter) and fusiform banded bodies (5 to 8 u thin

with periodicity of 1000A) were also seen underneath these cells (fig. 6b &fig. 7). These collagen fibrils had no organized collagenous lamellea and were mainly seen to run along the anterior surface of these cells.

Various types of leukocytes, mainly lymphocytes, were commonly seen attached to the posterior surface of the endothelium most commonly in case no. 1 (fig.. 6a).

DISCUSSION

Although the examined cases of glaucoma had different causes, the pathological changes tend to increase their resembelence to one another as the condition becomes more advanced. When glaucoma had become absolute, it is certainly one disease so that at the end, all types may present almost identical signs and symptoms, Moreover, it has to be remembered always that corneas studied for pathological changes, in case of glaucoma, are essentially taken from eyes removed as a terminal stage in the disease and that it could be justifiable to speak of the pathology of absolute glucoma of different eitiology.

The present electron microscopic study revealed no significant structural difference in all three examined cases. There was a basment membrane underneath the endothelial layer together with groups of collagen fibrils and fine filaments. The findings are similar to those reported by Iwamoto et al. (1861) in Fuch's dystrophy. The changes in the endotheial cells in glaucoma resembled type I abnormal cells of Fuch's dystrophy. Moreover, a basement membrane of this type had been noted to cover the trabecular meshwork following contusion (Wolff et al. 1962) associated with clouding and edema of the cornea, a condition which is commonly seen in absolute glaucoma. Howevere, one must differentiate between two sorts of corneal haziness : The first appears promptly when the IOP rises and disapeares as soon as it falls and the second, more lasting and true form, is found in the long standing and absolute glaucoma. According to Cogan (1951), the first type is due to temporary dislodgment of the intracellular fluid due to stretching of the cornea caused by the rise of the IOP. As to the cause of the second type, the present ultrastructural study showed abnormalities in the endothelial lining of the cornea which can lead to disturbance of its function. These abnormalities of the endothelium is likely to be caused by the injurious effect of the continuous rise of IOP, presumbly resulting in fibroblastic metaplasia of these cells with the resultant formation of a basement membrane and collagenous material in the posterior part of Descement's membrane.

As the endothelium is known to be responsible for the state of detergescence of the cornea, the edema of all layers of the cornea reported in the present study can represent the second type of edema known to occur in glaucoma. Thus it can be

concluded that this edema is not primarily due to the elevation of the IOP but rather secondary to the pathological changes affecting the posterior part of the cornea, mainly the endothelium.

The concept of fibroblastic potential in the endotheial cells had been previously reported by Stocker (1953) as well as Chi et al. (1962). In addition, since the endothelium is probably mesodermal in origin, it would not seem imposible that under certain conditions, it might aquire properties of connective tissue.

SUMMARY

The effect of rise of IOP on the different layers of the cornea was studied. Increase in the IOP results in fibroblastic metaplasia of the corneal endothelium with deposition of collagenous material in the posterior part of Descemt's membrane. These pathological changes in the cornea were found to be associated with a significant degree of edema of all the cellular components of the cornea.

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LEFENDS FOR FIGURES

Fig. 1. A micrograph showing marked edema of the epithelum with widening of the intercellular spaces (S) and separation of the cells from each other except at the desmosomal junctions (ć) (X 10000)

Fig. 2. Part of an epithelial cell from the basal layer of the epithelium of the cornea showing wide intercellular spaces (S), dilatation of the mitochondria (mit) and clumping of the tonofibrils (T). N. nucleus, (rer.) rough endoplasmic reticulum. (X 16000).

Fig. 3. Bowman's layer (BoL) with remnants of the basal cell layer (Ep) loosely attached to it. (X 16000).

Fig. 4. Bowman's layer (BoL) is separated from the basal epithelium (Ep) by a space filled with fine collageous fibrils (col) and basement membrane-like material (bm-L). (X 16000)

Fig. 5. The stroma is edematous but its lamellae remain reasonably regular Lower left corner shows part of Descemet's membrane (DM). (X 4000).

Fig. 6a. The endothlium here is separated into two layers of cells (I & II) with

intervening groups of collagen fibrils(*). A basement membrane lines the endothelial cells and separates if from the underlying Descemet's membrane (DM). The endothelial cells contain long chains of rough endopasmic reticulum (rer). L : lymphocyte, (AC): anterior chamber. (X 10000).

Fig. 6b. A higher magnification of fig. 6a showing the collagen fibrils and the basement membrane between the two layers of endothelium. (X 25000).

Fig. 7. Part of the endothelial cell (En) showing collagen fibrils in their cytoplasm and a basement membrane (bm) separting it form the underlying Descemet's membrane (DM). (AC) : anterior chamber. (X 16000).

Fig. 8. An endotheilail cell with a spindle-shaped nucleus (N) and contains collagen fibrils in its cytoplasm. Fusiform banded bodies (arrow) lie in the posterior part of Descement's membrane.

Insert : shows high power magnification of the fusiform banded body and collagen fibrils inside and outside the cell. (X 1000) & (X 25000).

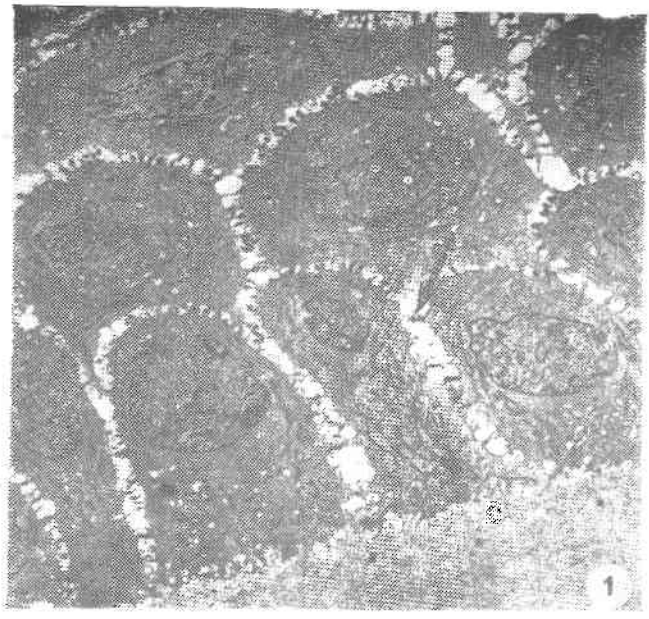


Fig. (1)

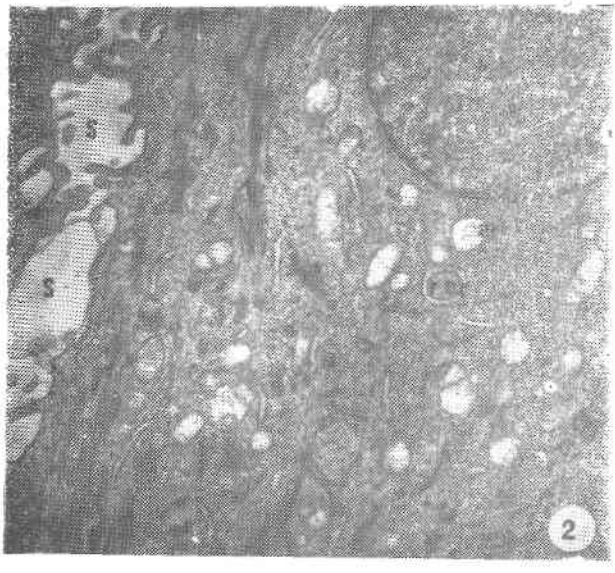


Fig. (2)

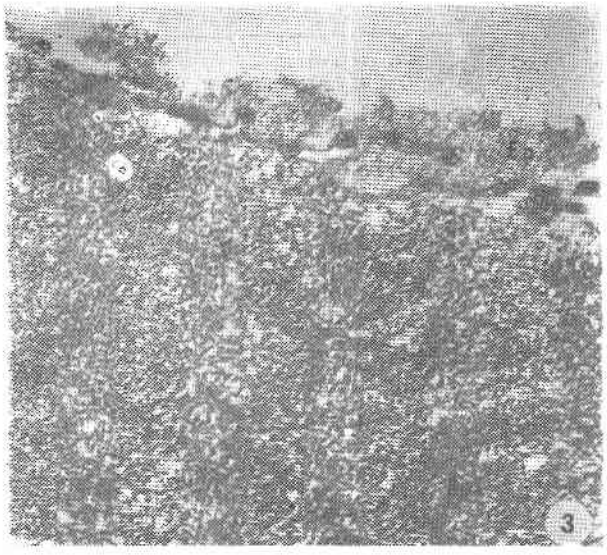
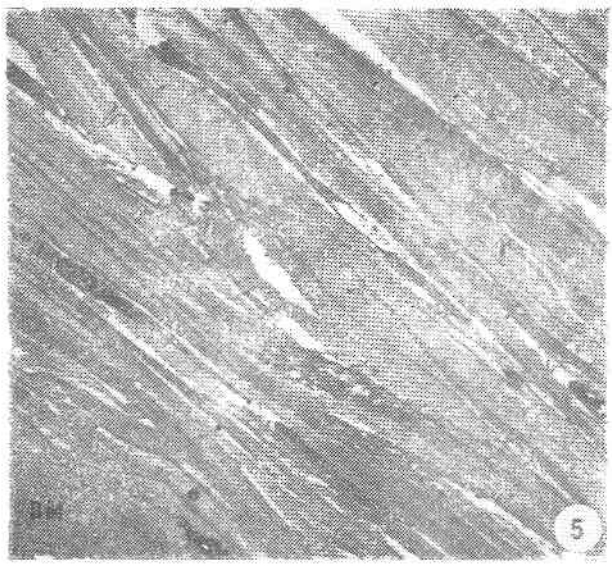


Fig. (3)

Fig. (4)



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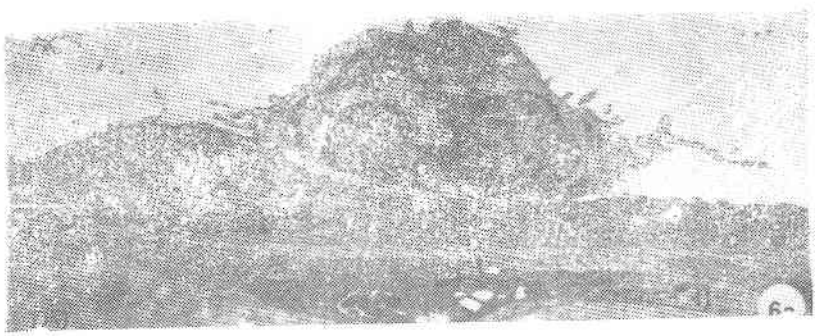

Fig. (6a)

Fig (6b)

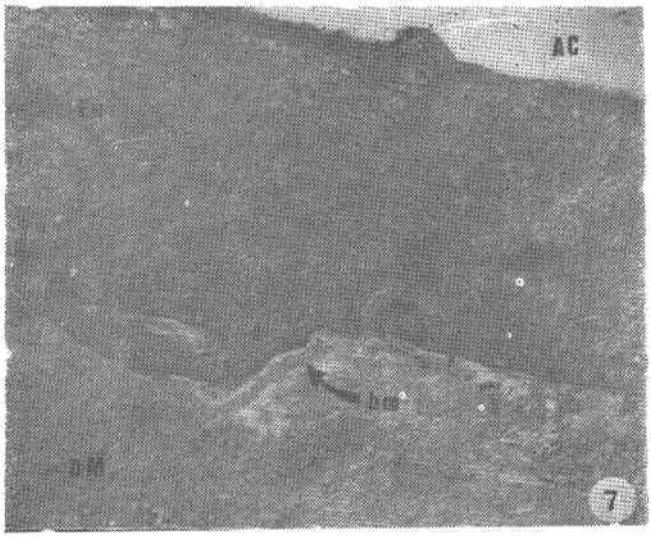

Fig. (7)

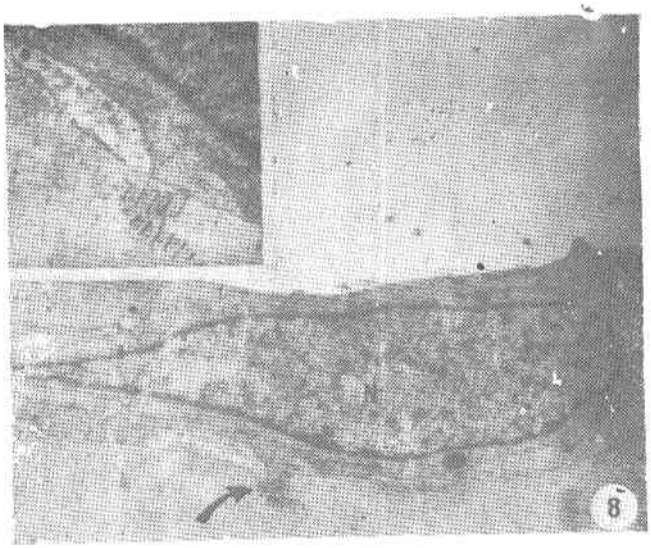

Fig. (8)