Central Corneal Thickness and Thickness of the Lamina Cribrosa and Peripapillary Sclera in Monkeys

In the Ocular Hypertension Treatment Study, central corneal thickness (CCT) has been recognized as a significant risk factor for progression of ocular hypertension to primary open-angle glaucoma. Consequently, Herndon et al demonstrated that CCT was inversely correlated with the amount of glaucomatous optic nerve damage at the time of referral of the patient. Several investigations confirmed that a thin cornea was a risk factor for development and progression of glaucoma. It has remained unclear whether a thin cornea was a clinical risk factor because the falsely low intraocular pressure measurements were not corrected for their dependence on CCT or whether a thin cornea was additionally a structural risk factor potentially due to an association with a thin lamina cribrosa. According to biomechanical considerations, a thin lamina cribrosa may be a risk factor for increased glaucoma susceptibility. We therefore conducted this study to assess whether corneal thickness is associated with the thickness of the lamina cribrosa.

Methods. The histomorphometric study included 22 monkey eyes (Macaca mulatta) that had undergone a temporary experimental occlusion of the central retinal artery. On anteroposterior histological sections through the pupil and the central optic disc region, thicknesses of the cornea and lamina cribrosa were measured in the center, at the periphery, and between the center and the periphery. Additionally, the peripapillary scleral thickness was determined at the optic disc border within the optic nerve meninges and just outside the optic nerve meninges. For outlining the borders of the lamina cribrosa, care was taken to differentiate the anterior lamina cribrosa surface from overlying glial tissue and to delineate the posterior lamina cribrosa from the optic nerve. The reproducibility of the technique had been evaluated in a previous study in which 10 randomly selected histological optic disc sections were reevaluated 10 times. The coefficient of variation was 0.14.

Results. The mean (SD) corneal thickness was 675 (114) μm in the center and 875 (107) μm in the corneal periphery. The mean (SD) thickness of the lamina cribrosa was 203 (46) μm in the center and 225 (53) μm in the periphery. The mean (SD) peripapillary scleral thickness was 251 (36) μm within the optic nerve meninges and 407 (63) μm outside the optic nerve meninges. The CCT was statistically not associated with the thickness of the lamina cribrosa in the optic disc center (P = .31) (Figure 1) or in the periphery of the optic nerve head (P = .29), nor was it associated with the thickness of the sclera within the optic nerve meninges (P = .41) (Figure 2) or outside the optic nerve meninges (P = .16). In a similar manner, the peripheral corneal thickness was statistically not associated with the thickness of the lamina cribrosa.
crista cribrosa in the optic disc center ($P=.73$) or in the periphery of the optic nerve head ($P=.55$), nor was it associated with the thickness of the sclera within the optic nerve meninges ($P=.23$).

**Comment.** The results suggest that in nonglaucomatous monkey globes, the CCT and the peripheral corneal thickness are not significantly correlated with the thickness of the lamina cribrosa in the center or at the periphery of the optic nerve head. They are also not associated with the thickness of the peripapillary sclera inside the optic nerve meninges or just outside the meninges. Confirming findings from studies on human globes, our study makes one infer that an assumed relationship between the CCT and glaucoma susceptibility may not be explained by corresponding anatomy between corneal thickness and histomorphometry of the optic nerve head.

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**Financial Disclosure:** None reported.

**Funding/Support:** This work was supported by grant EY-1576 from the National Institutes of Health (Dr Hayreh) and in part by unrestricted grants from Research to Prevent Blindness, Inc, New York, New York.


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**Chronic Localized Fibrosing Vasculitis of the Eyelid**

Chronic localized fibrosing vasculitis (CLFV) is a rare entity of unknown etiology. To our knowledge, it has never been reported to occur in the eyelid.

**Report of a Case.** A 42-year-old man had gradually progressive, painless swelling in the left lower eyelid for 2 years. There was no history of systemic illness, drug intake, or insect bite. On examination, there was a nontender, firm, nodular mass in the left lower eyelid (Figure 1). The palpebral conjunctiva appeared normal. There were no features of orbital involvement. Ocular examination did not reveal any abnormalities in either eye, and best-corrected visual acuities were 20/20 OU. Systemic examination results were normal.

Computed tomographic scan of the orbit showed a homogeneous, minimally enhancing, relatively well-defined soft-tissue lesion in the eyelid with no evidence of orbital extension. Paranasal sinuses were normal. Results of a detailed systemic workup performed to rule out autoimmune or infectious systemic disease were within normal limits.

Incisional biopsy of the lesion was performed. Histopathological examination showed features of chronic leukocytoclastic vasculitis with a mild to moderate diffuse infiltrate consisting predominantly of lymphocytes and fewer, scattered neutrophils, eosinophils, and plasma cells (Figure 2A). The inflamed stroma contained granulation tissue, marked proliferation of small vessels, and an extensive onion-skin pattern of fibrosis of blood vessels (Figure 2B). This picture was punctuated by foci of active leukocytoclastic vasculitis, characterized by fibri-noid necrosis of vessel walls in which eosinophils, neutrophils, and nuclear debris were present (Figure 2C). No lymphoid hyperplasia or granulomas were identified. These histological features were consistent with CLFV. The patient was started on topical tacrolimus ointment and is currently being followed up.

**Comment.** Chronic localized fibrosing vasculitis, an entity that was first described by Carlson and LeBoit in 1997, is characterized by a nonspecific inflammatory reaction with vasculitis of small vessels with a distinctive concentric pattern of fibrosis. Although CLFV may be considered a variant of inflammatory pseudotumor of the skin, there are distinct differences. The presence of vasculitis and leukocytoclasis (nuclear debris) and the absence of dense plasma cell infiltrate in our case clearly distinguished it from cutaneous inflammatory pseudotumor. The distinction between the 2 entities may also have prognostic implications as CLFV (classified as vasculopathic reaction pattern