Research Highlight

Corneal Nerve Damage In Microbial Keratitis

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Barriers that rapidly oppose potential corneal pathogens include innate immunity, tears that form a barrier and contain antimicrobial substances, and blink reflex that helps to spread tears over the cornea and mechanically sweep away pathogens. These protective mechanisms are controlled by nerves that can both stimulate and inhibit immune functions, stimulate tear production, and regulate blink reflex. In this issue of *IOVS*, Cruzat et al.[1] demonstrate that corneal infections by a variety of pathogens are associated with loss of corneal nerves. By using laser in vivo confocal microscopy and corneal sensitivity measurements, they demonstrate that patients with microbial keratitis caused by bacteria, fungi, and *Acanthamoeba* all show significant loss of the corneal nerves and blink reflex in the clinically affected eye. This observation extended previous reports of corneal nerve loss in eyes of patients and mice with herpes keratitis.[2,3] Of particular interest was the observation that nerve loss and leukocytic infiltration was not confined to the clinically affected cornea, but also extended to the contralateral clinically unaffected cornea. Although the mechanism(s) of nerve damage was not defined, these studies demonstrate that one cannot ignore the clinically unaffected eye of patients with microbial keratitis. Whether contralateral nerve damage reflects a subclinical infection or sympathetic damage remains to be determined. Inflammation contributes significantly to corneal pathology associated with microbial keratitis. Therefore, it is also important to determine if inflammation is the cause or the effect of nerve damage; the possible contribution of neurotrophic inflammation and exposure keratopathy to corneal pathology associated with microbial keratitis; and to what extent blinking in the contralateral eye and consensual blinking in the infected eye are affected. Also critically important is understanding if nerve damage is reversible, as suggested by a recent study,[3] and what controls corneal re-innervation.

References

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