CLCS Resident Writing Competition Winner

Microbial keratitis with extended wear: A multifactorial mechanism

Stephanie Fromstein, O.D.

Extended wear of contact lenses is the main risk factor for microbial keratitis.\(^1\) With the advent of silicone hydrogel lenses and their associated increased oxygen permeability, there was a widely held hope that the incidence of this potentially serious complication would decrease. As many studies have since revealed, this is not the case.\(^2\) The question remains, then: how do contact lenses affect disease pathogenesis, and how is this process exacerbated by extended wear?

Contact lenses are a vessel for microbial attachment, with silicone hydrogel lenses showing higher adhesion rates of classic disease-causing agents than hydrogels.\(^3\) Adhesion is an instigating factor in the disease process and directly associated with corneal infection.\(^4\) Lens colonization also has the ability to form biofilms. This conglomerate of bacteria and extracellular matrix has enhanced density and resistance to antimicrobials and is closely associated with the development of microbial keratitis.\(^2,4\) Importantly, this phenomenon is noted only after a period of continuous lens wear, suggesting an adaptation period that “primes” bacteria for infection.\(^2,5\) This bacterial acclimatization is not possible over the time course of daily-wear regimens.\(^2\)

Contact lenses also influence the ocular surface and its innate defenses. The lens material itself dampens intrinsic defenses while up-regulating pro-inflammatory factors, thus increasing epithelial vulnerability to attack.\(^2\) Lenses also affect the volume and composition of the tear film, which may disrupt the protective mechanisms in a way that has yet to be fully elucidated.\(^2,6,7\)

Clearly, the pathogenesis of microbial keratitis is complex and multifactorial, involving interplay between ocular physiology, contact lenses, and the environment. What appears evident, however, is that each mechanism would be exacerbated by extended wear. It is not surprising, then, that extended wear remains the principal risk factor for infection, and we should look to minimize its impact by approaching the underlying interactions outlined above.

References:

*Dr. Stephanie Fromstein is the current Cornea and Contact Lens resident at the Illinois College of Optometry. She graduated with honors from Nova Southeastern University. Her clinical interests include specialty contact lens fittings, as well as emerging lens designs and materials.*

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