Letters

What Is the Cause of Corneal Astigmatism?

For the past several years, I have been involved with the clinical applications of several modalities that induce biomechanical changes in the cornea, specifically microwave thermokeratoplasty and ultraviolet A cross-linking. My colleagues and I have used these modalities singularly and in combination on both normal and keratoconic corneas.

After observing numerous pre- and postoperative topographies, I noticed an interesting pattern starting to emerge, prompting me to begin to think of corneal biomechanics in a different way. What I have observed is that, when a biomechanical intervention is applied to a cornea, the behavior of a keratoconic "cone" is very similar to the behavior of an astigmatic topographic elevation. For me, it raised the question, what is the cause of astigmatism? While we all know what the manifestation of astigmatism is, the etiology is not known. So, I posit the following: what if a localized weakening of the cornea causes astigmatism?

The clinical observations supporting the theory certainly fit. When an astigmatic cornea that otherwise appears to be normal is cross-linked, there is always enhanced flattening of the astigmatic component, just as in the case of a cross-linked keratoconic cornea. Is astigmatism just subclinical keratoconus?

It is well known that, when preselecting a LASIK candidate's suitability, preexisting astigmatism is always a concern for analysis as to shape and location. This is because we believe there is some correlation between astigmatism and possible ectasia. Is it logical to correlate this with a preexisting weakness?

In terms of biomechanics, we know that the pressure on the posterior cornea is completely isotropic, and we do not observe any abnormal thinning of a normal cornea in the region of astigmatism. Why would an isotropic force pressing against an isotropic mechanical surface induce an anisotropic shape?

Currently, when treating myopic astigmatism with a laser, we remove more tissue on the raised area. Alternatively, we often use relaxing incisions to flatten the astigmatic area. Both of these techniques certainly have a localized weakening effect. Will they also exacerbate a preexisting weakness?

I do not pretend to know all of the ramifications of our current therapies if my conjecture is, in fact, correct. It is only a conjecture based on many, many observations by someone not classically trained as an ophthalmologist. Nonetheless, I believe that it is an intriguing question and certainly one I will be investigating. I hope this food for thought sparks some interesting conversation.

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Alarmed About Generics

With many physicians' recent shift to generic ophthalmic products, clinicians must begin to police these agents themselves, because it seems obvious that the normal agencies do not. Every ophthalmologist remembers the problems several years ago with generic diclofenac and the associated corneal melting. We have now seen three cases of corneal problems with generic ketorolac and bromfenac associated with the use of these agents around the time of cataract surgery.

That stated, we do not understand why generic ophthalmic medications are only required to show bioequivalence or a comparable rate and extent of absorption. Branded drugs must show preclinical data to establish efficacy and safety in both animal and clinical models, whereas generics are not required to undergo the same rigorous requirements for ophthalmic products. Obviously, all physicians have seen problems with generic formulations. The most obvious is generic prednisolone acetate 1%. Most ophthalmologists realize that the generic is not equivalent to the branded product in terms of efficacy and potency. Assessing bioavailability with systemic medications through blood testing is much easier than monitoring bioavailability in the eye.[1] It is for this reason that we believe generics should be held to the same testing regimens as their branded counterparts with regard to ophthalmic preparations. The preparations are comparable with regard to their chemistry and manufacturing, but their inactive ingredients can vary considerably. Preservatives, pH adjusters, thickening agents, and buffers can differ.[1]

What can physicians do? As with diclofenac, they can begin reporting cases to the appropriate agencies and pushing for generics to be held to the same standards as their branded counterparts. Practitioners should keep clinical photographs and document cases well, because the manufacturers of these products will deny it is in fact their product until adequate proof is given. For the time being, physicians should monitor their patients' use of generic nonsteroidal anti-inflammatory drugs with a watchful eye. We think we will see more of these problems in the near future.

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1. Cantor LB. Ophthalmic generic drug approval process: implications for efficacy and safety. *J Glaucoma*. 1997:6(5):344-359.