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Are Descemet Membrane Ruptures the Root Cause of Corneal Hydrops in Keratoconic Eyes?



EDITOR:

THE RECENTLY PUBLISHED PAPER BY PARKER AND ASSOCIATES¹ on acute corneal hydrops (ACH) in keratoconus is of interest as it reinforces a novel concept on the mechanism of acute hydrops implicating the pre-Descemet layer (Dua's layer, PDL). This was postulated when the first evidence of the PDL was provided² and clinically substantiated by Yahia Chérif and associates,³ when they showed that approximation of the torn PDL with mattress sutures without Descemet membrane (DM) leads to rapid resolution of ACH. Parker and associates¹ have provided further clinical evidence to support the involvement of the PDL in the pathogenesis of ACH. This, however, is not the full story. When the DM by itself or jointly with the PDL is stripped or incised (3-mm incision extending to 80–100 μ m of posterior stroma) in normal eye bank eyes and the pressure raised from 15 mm Hg to 60 mm Hg (with the incised cornea mounted in an artificial anterior chamber), ACH does not result. There are no fluid spaces formed in the corneal stroma (personal observations). On this basis we stated in 2015 that “acute hydrops in keratoconus was a result of a tear in both the DM and the PDL in the background of the abnormal collagen of the corneal stroma in keratoconus.”⁴ The altered stromal matrix and collagen in keratoconus are also important in the occurrence of ACH. An in vitro experiment with keratoconus corneas will be very difficult to perform, but Parker and associates¹ have provided the nearest to that with their in vivo intraoperative observations in keratoconus eyes, elegantly supporting the concept. Acute traumatic and surgical (venting incisions) perforations of normal corneas, which replicate the intraoperative scenario as reported by Parker and associates,¹ could constitute “natural controls” for their observations. ACH does not occur in such situations in normal corneas, pointing to the role of the altered corneal stroma in ACH. Inadvertent intraoperative perforations in keratoconus and traumatic perforations differ from the real-life ACH scenario in which the tear(s) are internal only, not through-and-through. In our experiment the former situation was replicated in normal corneas.

The biomechanical properties of the cornea are altered in keratoconus. The PDL has been shown to contain a high concentration of elastin that is uniformly

distributed through its thickness.^{5,6} In DM elastin is distributed as a dense anterior sheet.⁶ Elastin contributes to the biomechanics of the cornea and is degraded in keratoconus.⁵ The DM is apposed to the PDL and loosely attached to it by interfacial matrix. When the two are torn together as in ACH, the retraction and coiling of the DM, determined by its elastin content and distribution,⁶ pulls the PDL with it, resulting in delayed healing and prolonging resolution of ACH.

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REPLY

WE WERE GRATIFIED TO SEE THAT OUR RECENT ARTICLE has generated so much interest, particularly from some of

the foremost thinkers in the field of the posterior cornea. And, we thoroughly agree with the primary point of their letter; namely, that “acute hydrops in keratoconus was a result of a tear in both the Descemet membrane and the pre-Descemet’s layer in the background of the abnormal collagen of the corneal stroma in keratoconus.”¹ As we learn more about this abnormal collagen structure, it is hoped that more and better treatment options for keratoconus will present themselves.

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