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Letter to the Editor Re: "The Relationship Between Keratoconus Stage and the Thickness of the Retinal Layers"

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Dear Editor,

We read with interest the recent study conducted by Özsaygılı et al.[1] regarding the relationship between the keratoconus (KC) stages and the thickness of the retinal layers. The authors concluded that patients with more advanced KC had a thicker inner nuclear layer (INL) and thinner retinal pigment epithelium (RPE) layer. They hypothesized different potential mechanisms for changing these two retinal layers in KC eyes. The main novelty of this work was the investigation of retinal layers of KC patients separately. We would like to congratulate the authors on their informative research. However, the conclusion of the study suffers from some shortcomings.

In the study by Özsaygılı et al.[1], patients with myopia lower than -6.00 D were included. However, the authors attributed the significant changes in the INL and RPE to the different biochemical, oxidative, genetic, and cellular mechanisms in a KC eye. Nevertheless, in KC patients, the induced myopic refractive error might be a compelling factor producing compensatory changes in the retinal layers due to the resultant optical defocus. For example, Liu et al.[2] investigated the effect of myopic refractive error on the thickness of the retinal layers in the healthy population and concluded that various myopic refractions (from mild to high degrees) could significantly affect the profile of the retinal layers of normal subjects. The more progression in myopia, the more changes in the profile of the retinal layers. Although Özsaygılı et al.[1] measured the refractive state of their patients with manifest refraction, they did not clearly state the range and mean values of the refractive errors of their study subjects. Furthermore, other influential factors on retinal thickness including diurnal variations, segmentation errors, gender, and body mass index were not considered as confounding factors.[3]

In addition to the mentioned likely causes for changes in the retinal layer profile in KC patients, there are two possible neurophysiological explanations. The first theory relates to the induced Stiles-Crawford phenomenon of the first type (SCE-type I) in KC corneas. In KC patients, a kind of multifocality occurs in the affected cornea that could contribute to the reduced luminous strength of incoming light on the retina in a way similar to light entering at the border of the pupil.[4] This phenomenon may stimulate SCE-type I and induce changes to the profile of the retinal layers. Another potential mechanism is a compensatory photostasis phenomenon in KC eyes. This effect is the long-term adaptation of the retinal photoreceptors (rod cells) to the changes in lighting conditions of the eye. KC eyes face sub-normal optical function and the light rays reaching the retina are dimmed. This effect causes some degree of prolonged image degradation and results in light deprivation. Subsequently, photostasis of the

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photoreceptors acts as a compensatory mechanism and could result in photoreceptor elongation.[5]

According to the above explanations, the conclusion of the manuscript could not be restricted to the KC patients. In practice, the significant changes in the INL and RPE layers could be as a compensatory response to other associated factors including induced myopia secondary to the KC, not exclusively the KC disease itself.

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Authorship Contributions

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