• Comment and Response •

Comment on "Post photorefractive keratectomy corneal ectasia"

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Dear Editor,

It is with interest that I read this paper[1] of two patients with ectasia after photorefractive keratectomy (PRK), particularly the second case of a 54-year-old man who 17y after successful PRK developed ectasia in his left eye 3mo after undergoing bariatric surgery. I very much thank the authors for reporting this case. What is particularly intriguing is that 7y previously, and 10y after PRK, this gentleman was emmetropic in this eye with a central corneal thickness of 560 μm and a normal post myopic PRK topography, albeit with what appeared to be a small central island. If this patient has developed progressive ectasia after such a long period of stability, then this has important implications for the hundreds of thousands of patients undergoing laser kerato-refractive procedures annually and the increasing numbers of patients undergoing bariatric surgery.

Based on the evidence the authors have been able to present this far, there might be three possible scenarios to explain the changes seen in this patient. The first is that this could be, as suggested, progressive ectasia due to biomechanical changes induced by possible hormonal changes induced bariatric surgery. This would be unexpected, but not impossible, given the passage of time after PRK, the patient age, the central location of the steeping, the lack of any previous reports of corneal ectasia after bariatric surgery and the fact that although hormonal changes do occur after bariatric surgery they are typically associated with a normalization of values[2]. Other possible scenarios are the development of degenerative changes in the corneal epithelium generated by nutritional/hormonal changes induced by bariatric surgery, unmasking either an underlying central island, which were common with first generation Excimer lasers and apparently present on the topographic map 7y previously, or a long-standing subclinical ectasia that has been present and stable for years and conceivably present even before PRK.

I wonder whether the authors could present further information to allow your readers to evaluate this case further. The author's quite rightly point out the abnormal changes seen on the Belin/Ambrosio Enhanced Ectasia Display (BAD) in this

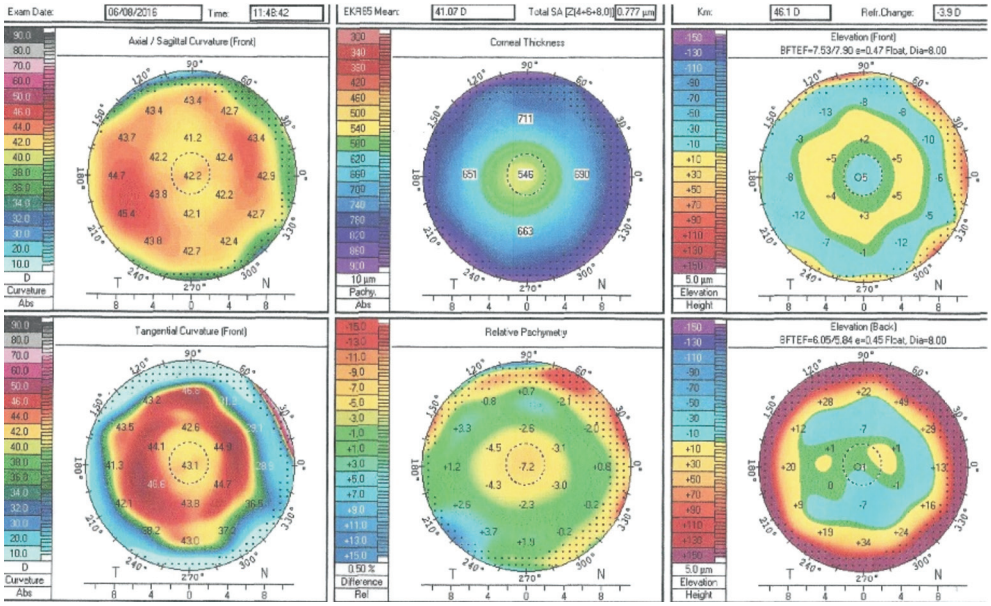

Figure 1 Tomographic maps of a 54-year-old lady who underwent PRK for low myopia 14y previously There were no features of ectasia with no obvious anomalies on the posterior elevation maps seen on the Holladay report display.

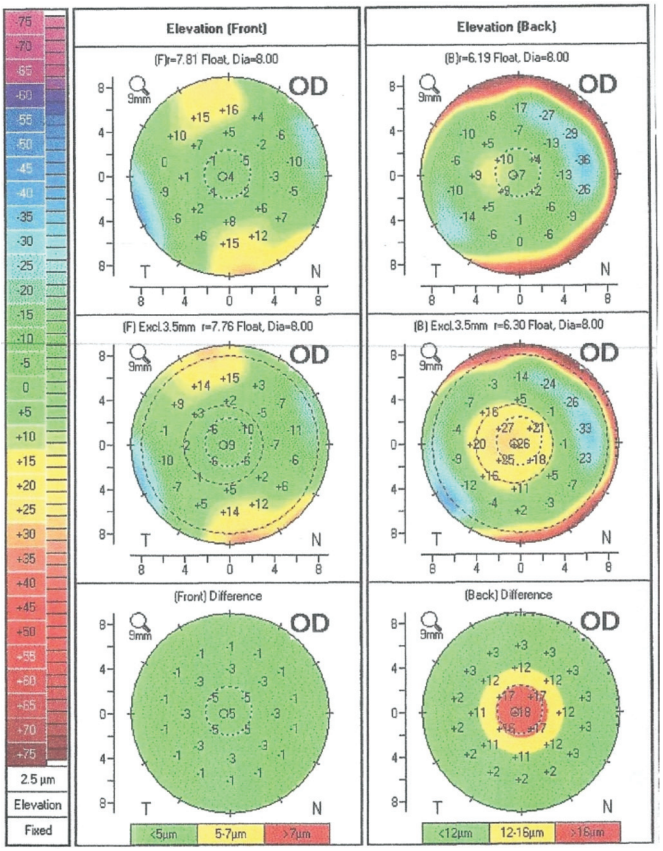

Figure 2 On the Belin/Ambrosio display there are anomalies on the posterior elevation maps despite no evidence of ectasia.

case to support their diagnosis of ectasia in this case. Whilst it is true that the BAD based on Scheimpflug technology is one the most sensitive ectasia screening systems, it probably still requires some validation in abnormally shaped corneas such as those after laser kerato-refractive surgery. I have now seen a few cases of eyes 15-20y after PRK (Figures 1, 2) who show similar posterior elevation changes on BAD, but with no central/paracentral corneal steepening/bow-tie asymmetry, no refractive evidence of ectasia with stable refractions and no induction of astigmatism, with elevation changes within normal limits on the refractive maps and Holladay[3] reports and who have remained stable with over 5y follow-up. Would it therefore be possible to know what were the maximal central posterior elevations on the refractive map displays and Holladay reports and if there has been any evidence of progressive changes consistent with ectasia since this case was reported. It might also be of interest to know if this gentleman's vitamin A and micronutrient levels, including Magnesium, have been measured as such deficiencies have been associated with ectasia development[4-7] as well as epithelial anomalies and whether he had floppy eyelid syndrome in association with his obesity which has also been associated with ectasia, albeit tenuously[8].

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Conflicts of Interest: O'Brart D, None.

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Author Reply to the Editor

Dear Editor,

We wish to thank Prof. O'Brart for appreciating our work on corneal ectasia developed several years after photorefractive keratectomy (PRK). It highlights the actuality of topic that in the next future would constitute another challenge for ophthalmologists in order to understand the possible corneal biomechanical changes in bariatric surgery patients.

This patient claimed visual disturbance few months after bariatric surgery requiring a cylindrical correction. Instrumental examination showed central steepening, red flag on posterior Belin/Ambrosio Enhanced Ectasia Display (BAD), 21 mm posterior elevation in refractive display and 19 mm posterior elevation on Holladay report. Central corneal thickness varied from 550 mm as measured at the first examination with US pachymeter to 508 mm as shown on Pentacam maps. Even if two different systems were used, we believe to consider this variation worthy of attention.

Prof. O'Brart suggests three possible scenarios to explain the changes seen in this patient. Progressive ectasia due to biomechanical changes following bariatric surgery and its hormonal variations, degenerative changes in the corneal epithelium due to nutritional/hormonal changes induced by bariatric surgery, unmasking either an underlying central island and finally a long standing sub-clinical ectasia that has been present and stable for years and conceivably present even before PRK.

The first scenario confirms our hypothesis related to slight biomechanical changes induced by metabolic and hormonal imbalance due to bariatric surgery. As to the hypothesis of a longstanding sub-clinical ectasia previously to the PRK, we feel to exclude it because of the elevation values far higher from normal with visual disturbances onset in concomitance of post-bariatric surgery period and central corneal thinning about 50 µm.

As to the epithelial changes unmasking a central island we have evaluated the epithelial thickness with Swept Source OCT (DRI OCT Triton, Topcon, Japan). The measurements revealed a minimal thinning of central epithelium with respect to the surrounding area. The central values were 39-40 μm and they were of 50-53 μm in paracentral area being slightly thinner on the center with respect to the paracentral area.

Such observation permits to exclude the presence of a central island where the epithelium characteristics are expected to be opposite (Figure 1). In their paper Rocha *et al*[1] described a significant thinning of the central epithelium (apex) which was around 46 μm in average and up to an average value 51-54 μm in the paracentral areas, in eyes with postoperative ectasia. These values were similar to those measured in our patient[1].

Prof. O'Brart reports a personal experience with patients who showed similar posterior elevation changes on BAD but without central steepening, no refractive evidence, no astigmatism induction with elevation changes within normal limits on the refractive maps and Holladay report. In our experience, we have never observed red flags in the posterior elevation maps in BAD in normal population (preoperatively) or after excimer laser ablation. This unexpected observation induced us to investigate the patient in detail in consideration of the contemporary visual disturbance and recent bariatric surgery. The our patient exhibited a slight astigmatism induction, central steepening and thinning with posterior elevation values far higher than normal that we believe is not so low to be ignored.

As to BAD reliability, is actually one the most sensitive ectasia screening system based on Scheimpflug technology and provides information on both anterior and posterior corneal surface. Belin *et al*[2] state that "the posterior cornea is an earlier indicator of ectatic change or ectasia susceptibility and when combined with full pachymetric data serve as more sensitive screening tool then anterior topography and ultrasound pachymetry combined". We wish to stress the BAD of the posterior corneal surface that exhibits red circle with 21 μm elevation is considered as abnormal[2-4].

As to the maximal central posterior elevation it was 21 mm on the refractive map displays (Figure 2), and on Holladay reports it was 15 mm at first examination (Figure 3) and 19 mm in 2017 (Figure 4) being out of normal limits.

Relatively to the progression we did not observe significant variations, except 4 μm worsening of the posterior elevation on Holladay report, that could not be significant at the moment. In our patient only minimal corneal change occurred, but it appeared as a self-limited, fruste secondary ectasia that should be monitored. Once occurred, the corneal changes were not progressive, probably due to biomechanical characteristics of the eye in over 60 years old subjects. For this reason we have speculate the biomechanical changes in the perioperative period characterized by important nutritional and

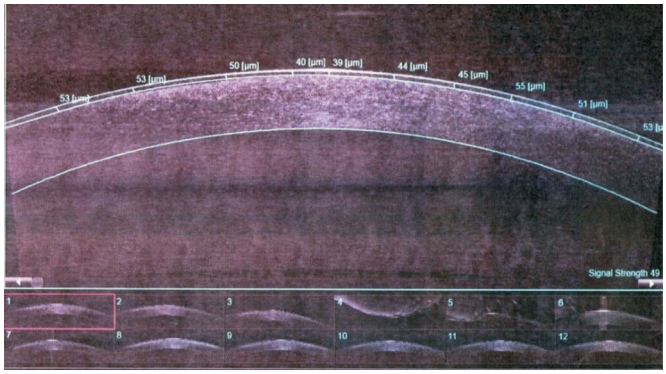

Figure 1 Swept Source OCT epithelium measurement.

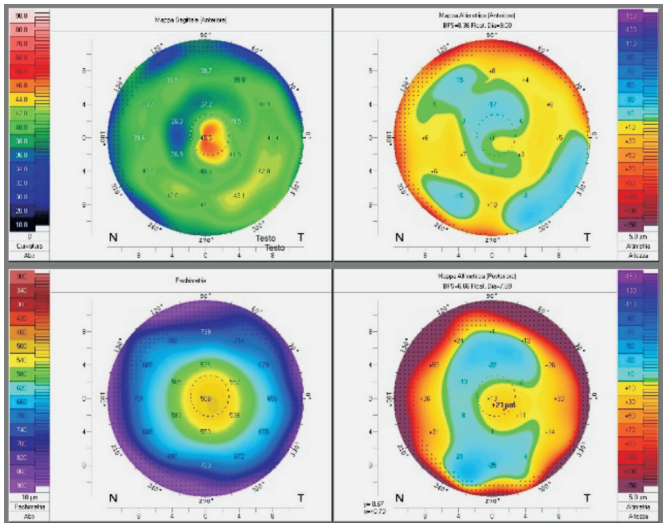

Figure 2 Refractive map display.

hormonal imbalance that, as reported, might lead to corneal biomechanical changes. We believe that such problem needs to be highlighted, considering wide bariatric surgery diffusion all over the world with remarkable number of refractive surgery patients.

As to Vitamin A and micronutrient levels, the patient did not complain any ocular surface disturbance and he exhibited normal tear film with normal corneal transparency and tear layer distribution with BUT over 15s in both eyes. For this reason, Vitamin A dosage and micronutrient levels were not measured. Patient referred no supplements intake. His bariatric surgeon did not prescribe him any supplement therapy.

The patient had a mild bilateral floppy eyelid syndrome that was described in clinical records since the first examination and it did not worsened during the follow-up period. As reported, metabolic and hormonal imbalance might lead to corneal biomechanical changes. In fact, pregnancy is included among risk factors associated with iatrogenic ectasia such as eye rubbing and young age[5-6].

We have speculated a hormonal and metabolic imbalance at the origin of corneal changes. We do not know what hormones, enzymes, and which metabolic pathway can be involved in corneal biomechanics. Besides, sex and enteric hormones, peptides and enzymes change in bariatric surgery patients[7]. We do not know the effects of these changes, if there are any,

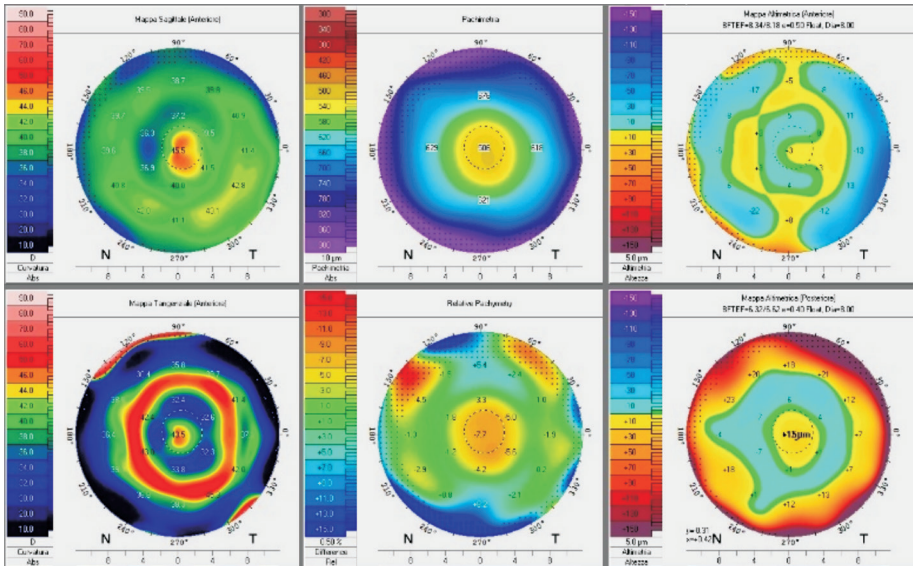

Figure 3 Holladay report in 2015.

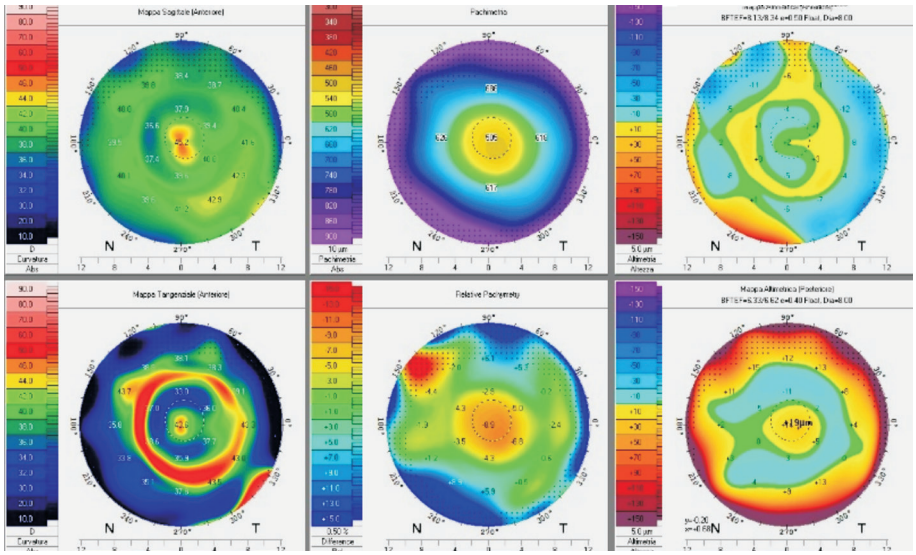

Figure 4 Holladay report in 2017.

on corneal biomechanics and we believe that they deserve furthers insights. These aspects need further investigation, as we have only tried to raise an interest to study such patients and we believe that the exact reason for these abnormalities needs to be clarified.

It should constitute a stimulus for further clinical assessment in bariatric surgery patients, as we wanted it to be. Prof. O'Brart states that there is limited knowledge about such cases and we agree completely with him. That is why we wanted to report our finding and stimulate further studies in this field.

We thank Prof. O'Brart for these comments. His interest confirms that the case reported is uncommon and difficult to understand and that makes it worth of publication.

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