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CASE REPORT

Post-LASIK Ectasia associated with Pigmentary Glaucoma: Tomographic and Biomechanical Characterization

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ABSTRACT

Aim: To report a case of bilateral post-laser *in situ* keratomileusis (LASIK) ectasia associated with pigmentary glaucoma (PG), in which intraocular pressure (IOP) assessment had been relentlessly underestimated by Goldmann's applanation tonometry (GAT).

Materials and methods: Case report and prospective literature review.

Results: The patient presented for second opinion regarding ectasia in the right eye, while ectasia was diagnosed in the left eye. Uncorrected visual acuity was 20/40 oculus dexter (OD) and 20/200 oculus sinister (OS). Distance corrected visual acuity was 20/20 in OD and 20/50 OS; GAT was 18/18 mm Hg. Slit-lamp biomicroscopy was relevant for a well-positioned superior hinge cornel flap and Krukenberg's spindle in both eyes, and Fleischer's ring in the left eye. Corneal compensated IOP was 47.8 and 43.8 mm Hg in OD and OS as measured by the ocular response analyzer (ORA; Reichert, Buffalo, New York, USA). The biomechanicalcompenated (IOPb) was 62.9 mm Hg OD and unmeasured OS by Corvis ST (Oculus; Wetzlar, Germany), which also demonstrated attenuated corneal deformation OU. Pentacam confirmed corneal ectasia in both eyes, being considerably worse in OS. One day after initiation of a fixed combination of beta blocker and carbonic anhydrase inhibitor, the patient noted significant improvement of vision with reduction of IOPb to 13 and 13.5 mm Hg in OD and OS respectively. Patient referred improvement in quality of vision and there was marked reduction on corneal scatter, thickness, and curvature.

Conclusion: Pigment dispersion syndrome (PDS) and PG may coexist with corneal ectasia. Careful consideration of the corneal impact on IOP assessment is mandatory when evaluation patients after laser vision correction (LVC). The IOP normalization may improve corneal ectasia in patients with coexisting glaucoma or ocular hypertension.

Keywords: Ectasia, Keratoconus, Pigmentary glaucoma.

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INTRODUCTION

Corneal ectasia is an uncommon but serious vision-threatening complication of keratorefractive surgery.[1] It is characterized by a progressive topographic steepening associated with deterioration of visual acuity. As the number of patients who have refractive surgery increases, more cases of ectasia are being recognized.

Iatrogenic ectasia usually occurs within the first year of surgery, although cases have been reported up to 10 years postoperatively.[2] From the reported cases, a variety of risk factors were proposed, including younger age, high myopia, low residual stromal bed thickness, and low preoperative corneal thickness altering corneal biomechanics.[3]

Even though the role of corneal biomechanical properties in ectatic corneal diseases is well established, studies suggest that it also has a crucial role in glaucoma. This is because of the relation of corneal properties and the accuracy of IOP measurement, as well as the inherent risk of glaucomatous damage to the optic nerve.[4] Corneal biomechanical factors can influence the accuracy of GAT and other tonometers. Although valuable in estimating glaucoma risk, central corneal thickness (CCT) is a suboptimal surrogate for the mechanical bending characteristics of the cornea, and it is but one component of a complex interaction between the viscous and elastic properties of the cornea, which ultimately combine to influence IOP measurements in not entirely predictable ways.[5,6]

We report a case of bilateral corneal ectasia 9 years post-LASIK, coexisting with PG. The very high IOP had not been detected, as it was considerably underestimated by GAT. In addition, the case illustrates the impact of IOP on corneal transparency and geometry.

CASE REPORT

A 31-year-old man was referred due to progressive visual acuity loss for the last 6 months in OS due to post-LASIK ectasia and suspicious of ectasia in OD. The patient had no records of previous ocular disease and a negative family history for glaucoma or keratoconus.

The patient reported bilateral LASIK 9 years ago for treating moderate myopia. No detailed information was available regarding flap, ablation, or refractive treatment, nor preoperative corneal parameters.

Uncorrected visual acuity was 20/40 OD and 20/200 OS. Distance corrected visual acuity was 20/20 (–2.00 –1.50 × 160) in OD and 20/50 (1.25 –3.75 × 125) in OS.

At the slit-lamp biomicroscopy, patient had a well-positioned and clear LASIK flap with superior hinge in both eyes. There was very mild corneal edema and Krukenberg's spindles on both eyes (Fig. 1). Fleisher's ring was evident in OS; IOP (GAT) was 18 mm Hg in both eyes (2:15 pm). Corneal compensated IOP was 47.8 and 43.8 mm Hg in OD and OS respectively, as measured by the ORA (Reichert, Buffalo, New York, USA). The IOPb was 62.9 mm Hg OD and unmeasured OS by Corvis ST (Oculus; Wetzlar, Germany), which also demonstrated

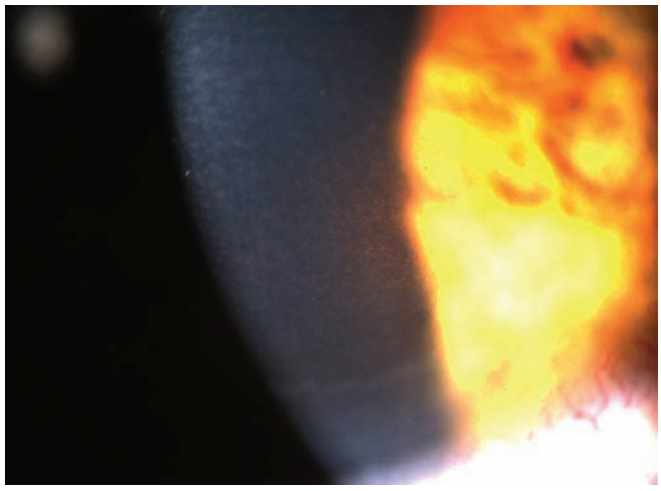


Fig. 1: Right eye presenting mild corneal edema and Krukenberg's spindle. Margin of LASIK flap is also noticeable

attenuated corneal deformation OU (Fig. 2). Pentacam HR corneal tomography (Oculus; Wetzlar, Germany) showed corneal ectasia in both eyes, but considerably worse in OS (Figs 3A and 4A). Ectasia OD was confirmed based on the posterior elevation, with an elevation of 21 µm the thinnest point using the best fit sphere for 8 mm. Fundoscopic exam showed a linear cup disk/ratio of 0.3 OD and 0.7 OS.

The diagnosis of PG associated with corneal ectasia was established. The patient was asked to return 1 day after initiation of a fixed combination of beta blocker and carbonic anhydrase inhibitor [Azorga® (brinzolamide 1% and timolol maleate 0.5%); Alcon; Fort Worth, TX, US].

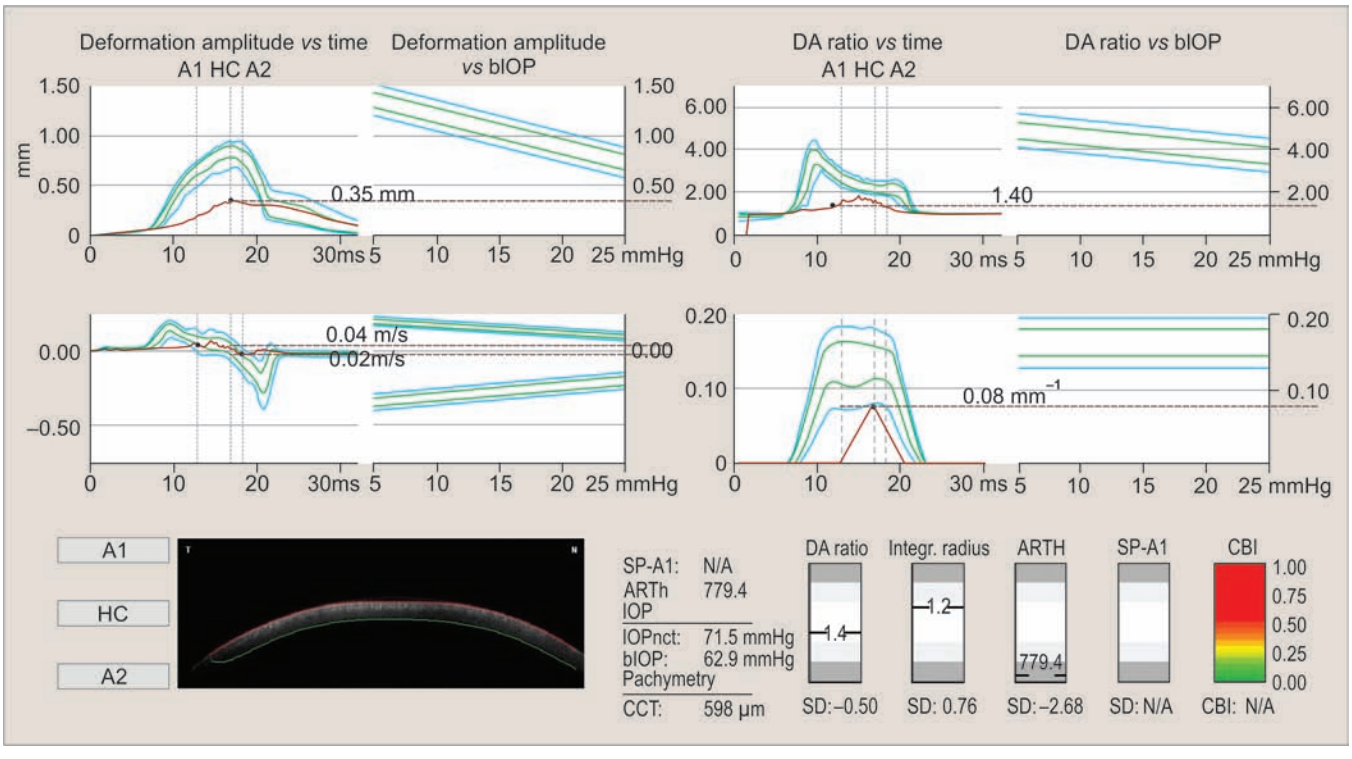
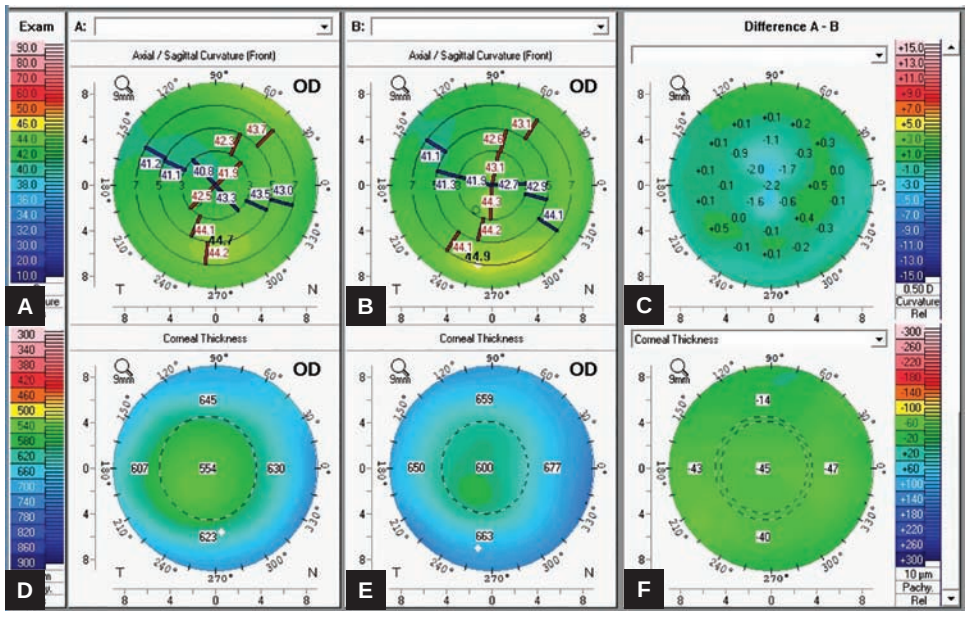
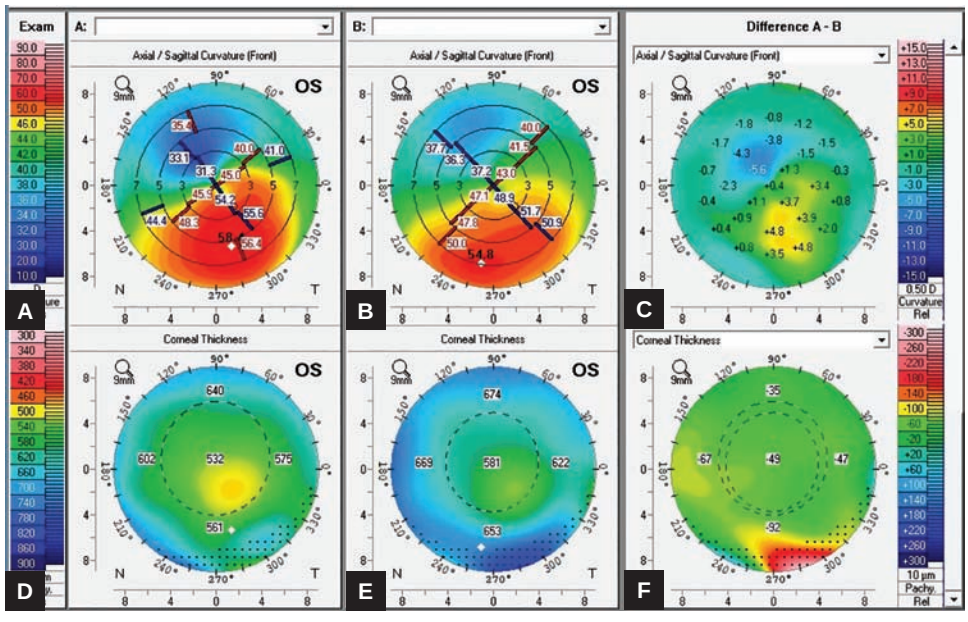


Fig. 2: Vinciguerra screening report from corvis ST measuring a 71.5 mm Hg IOP in OD, with a very low corneal amplitude deformation



Figs 3A to F: Pentacam OD. Curvature after (A), prior (B) to IOP reduction, and subtraction (C); and thickness after (D), prior (E) to IOP reduction, and subtraction (F)



Figs 4A to F: Pentacam OS. Curvature after (A), prior (B) to IOP reduction, and subtraction (C); and thickness after (D), prior (E) to IOP reduction, and subtraction (F)

The patient noted significant improvement of vision with uncorrected distance visual acuity of 20/25 OD and 20/80 OS. Manifest refraction was not performed at this visit. There was marked reduction of IOPb to 13 and 13.5 mm Hg in OD and OS respectively, with deformation corneal response consistent with the diagnosis of ectasia. Figure 5 demonstrates the Corvis ST data of the right eye in the second visit.

Marked reduction on corneal scatter, thickness, and curvature was noted in both eyes (Figs 3 and 4). Corneal applanation was 2.2D in OD (Fig. 3C) and 5.4D in OS (Fig. 4C). Corneal thinning (reduction of edema) was noted in both eyes (Figs 3F and 4F).

The patient was advised to consider cross-linking in both eyes and keep medical treatment and clinical control for glaucoma. The patient returned to his original city and no follow-up was further obtained.

DISCUSSION

Iatrogenic corneal ectasia is a rare, but very severe complication of LASIK.[7] There are recognized risk factors for the development of ectasia after keratorefractive surgery, including preoperative mild (or forme fruste) keratoconus, thin corneal pachymetry, thick LASIK flap, high corrections, and younger age.[1] But other factors are

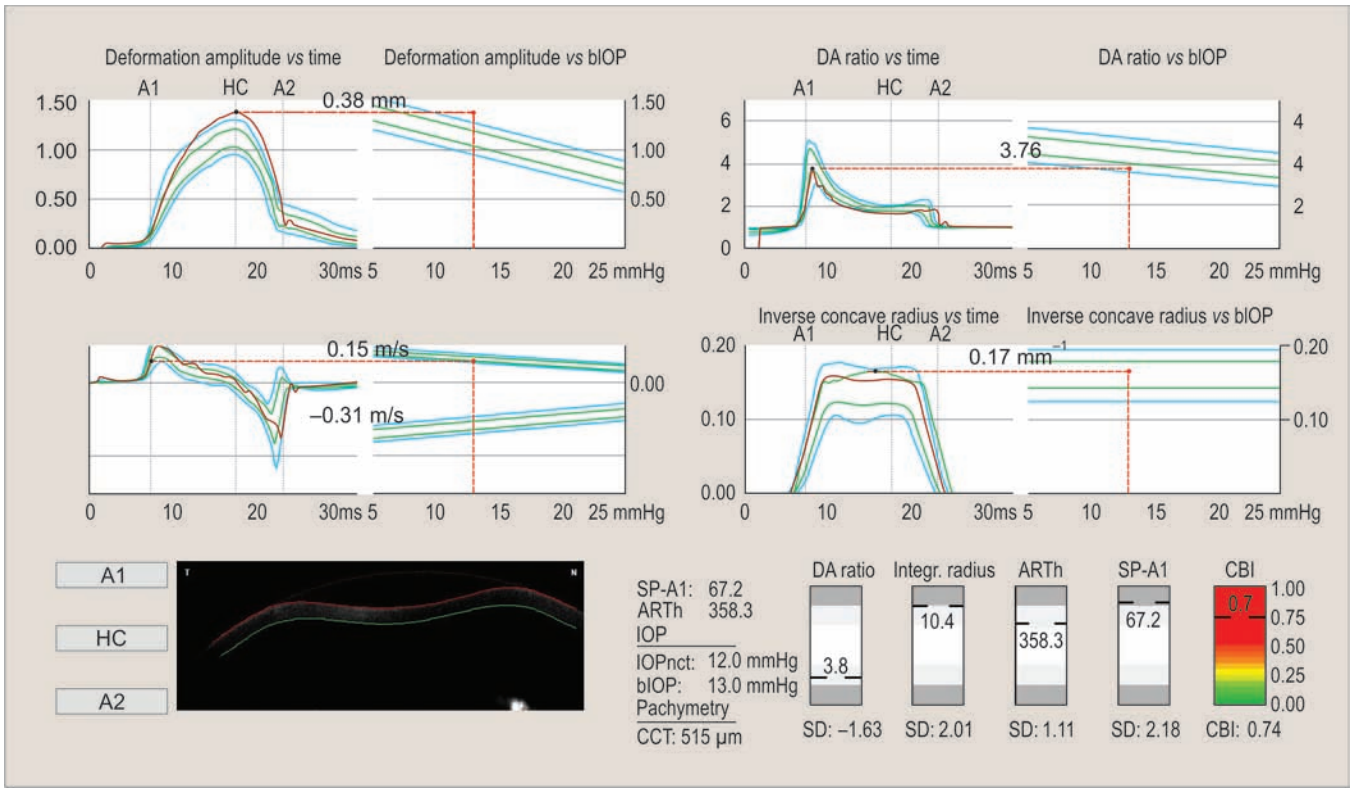


Fig. 5: Vinciguerra screening report from corvis ST OD after IOP reduction

associated with ectasia progression, such as eye rubbing and pregnancy.[1,8]

In fact, there are different mechanisms for ectasia to develop following keratorefractive surgery, including the inherent corneal predisposition to manifesting ectasia and the weakening by the impact from the procedure.[9] The role of postoperative trauma, such as eye rubbing and high IOP may also be very relevant for the biomechanical decompensation.[10]

While biomechanical properties of the cornea are of paramount importance in the keratorefractive surgery evaluation, it also has been related to glaucoma field. This is because of its relation with IOP measurement, but also due to the relation to the risk of glaucomatous damage to the optic nerve. The ORA and Corvis ST are noncontact tonometers that use a pulse of air to apply force to applanate the cornea and measure the IOP and try calculating a corrected IOP value, considering corneal deformation properties.[11]

Several studies investigating the potential utility of the corneal hysteresis measurement in glaucoma have been published, and it seems that low corneal hysteresis, but not CCT or IOP, is associated with progressive glaucomatous optic neuropathy.[5,12]

The most common and accepted method for measuring IOP for over 50 years has been the GAT. Nevertheless, as we better understand corneal biomechanics, its imitations are we increasingly appreciated.[13] Algorithms to correct for CCT to estimate IOP may oversimplify the effects of corneal biomechanics.

Corneal thickness is but one component of a complex interaction between the viscous and elastic properties of the cornea, which ultimately combine to influence IOP measurements in not entirely predictable ways. And even so, as a solo biomechanical component, it may not be reliable, since different corneal responses have been observed in corneas having similar thickness with the same deformation stimuli.[5,11]

Many authors have found an artificial underestimation of IOP measurements by different types of tonometers in eyes that underwent LVC.[14,15] As the number of patients who have LVC increases each day, falsely low IOP due to IOP measuring biomechanical limitations could precariously postpone the diagnosis of glaucoma or can interfere in the adequate control of the disease.[16]

The ophthalmologist who exam post-LASIK patients has to maintain a high index of suspicion for possible ocular hypertension and glaucoma patients, even if the IOP reading is low. The degree of myopic correction (which translates to ablation depth) may seem to be related to reduction in post-LASIK IOP. However, further studies are needed to determine if a critical level of myopic correction is required before a significant change in IOP post-LASIK occurs.[16]

Studies observed that patients with PDS, 5 to 10% will develop PG 5 to 6 years after diagnoses, 15% after 15 years, and 35% after 35 years. As the quiescent phase of PG occurs, the increased angle pigmentation tends to clear, and with this, the reversal of the pigmentary

signs in older patients can lead to a misdiagnosis of primary open-angle glaucoma or normal tension glaucoma when associated with IOP normalization. It also seems that the degree of myopia in PDS patients that develop PG is higher than the degree of myopia in those who do not develop.[17] Interestingly, the relationship between PDS and corneal ectatic disease has not been established yet.

This article illustrates a case of ectasia that was associated with PG in which GAT limitations limited the ability for the diagnosis. This is true that a more careful slit lamp evaluation of the cornea and a comprehensive analysis including fundus exam would provide more information that would facilitate the diagnosis. Nevertheless, the role of novel methods for IOP assessment is unquestionably emphasized in this report. Refractive surgeons must be aware that there are bidirectional influences of IOP and corneal properties assessment.

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