Pregnancy-related exacerbation of iatrogenic keratectasia despite corneal collagen crosslinking

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Abstract

Iatrogenic keratectasia after laser in situ keratomileusis (LASIK) represents a serious complication of refractive laser surgery. We describe a woman who developed bilateral iatrogenic keratectasia during her first pregnancy 26 months after LASIK. Corneal collagen crosslinking (CCL) with riboflavin and ultraviolet-A was performed in March 2005 (right eye) and April 2005 (left eye). This treatment stopped the progression and even caused the keratometric steepness to regress over a postoperative follow-up of 22 months, as demonstrated by preoperative and postoperative corneal topographies and maximum K-readings. During the patient's second pregnancy, the keratectasia exacerbated. To our knowledge, this is the first case showing exacerbation of keratectasia despite CCL and, as the exacerbation occurred only during pregnancy, suggesting that hormonal changes might affect corneal biomechanical stability.

Reference


DOI : 10.1016/j.jcrs.2008.02.036
PMID : 18571094

Available at:
http://archive-ouverte.unige.ch/unige:22477

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Iatrogenic keratectasia after laser in situ keratomileusis (LASIK) represents a serious complication of refractive laser surgery. We describe a woman who developed bilateral iatrogenic keratectasia during her first pregnancy 26 months after LASIK. Corneal collagen crosslinking (CCL) with riboflavin and ultraviolet-A was performed in March 2005 (right eye) and April 2005 (left eye). This treatment stopped the progression and even caused the keratometric steepness to regress over a postoperative follow-up of 22 months, as demonstrated by preoperative and postoperative corneal topographies and maximum K-readings. During the patient’s second pregnancy, the keratectasia exacerbated. To our knowledge, this is the first case showing exacerbation of keratectasia despite CCL and, as the exacerbation occurred only during pregnancy, suggesting that hormonal changes might affect corneal biomechanical stability.


Accepted for publication February 20, 2008.

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Neither author has a financial or proprietary interest in any material or method mentioned.

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following 2 years, keratectasia not only remained stable but also regressed, as demonstrated by preoperative and postoperative corneal topographies and maximum K-readings and keratoconus indices (Figure 1, B). Concomitant with the decrease in the maximum K-readings and the regularization of central astigmatism, the BSCVA increased to 20/40 with \(-3.75\) \(-2.75 \times 85\) in the right eye and 20/32 with \(-4.75\) \(-2.5 \times 145\) in the left eye in October 2006. Optical pachymetry showed the expected crosslinking-induced decrease to 346 \(\mu\)m and 330 \(\mu\)m, respectively.

In January 2007, the patient began her second pregnancy and in the sixth gestational month (June 2007), she noted a sudden deterioration of vision in the right eye. Corneal topographies showed a progression of keratectasia in the right eye until the end of the second pregnancy. Maximum K-readings reached levels similar to those before CCL (Figure 1, C). In the left eye, the BSCVA was unchanged, and the topographies were stable.

The patient had never smoked and had no history of allergies or extensive eye rubbing. Her body mass index was between 22.2 and 25.5.

**DISCUSSION**

Induced keratectasia after LASIK and after photorefractive keratectomy has been widely reported in recent years. Randleman et al.\(^4\) have shown that the most significant risk factor for the development of iatrogenic keratectasia is preoperatively undetected abnormal topographies. Other reported cases of induced keratectasia show no preoperative signs of corneal abnormalities. Furthermore, the postoperative time to occurrence of the first signs of keratectasia varies; in some cases, keratectasia occurred up to 36 months after LASIK.\(^5\) Our case shows late onset of keratectasia and is highly unusual because of the temporal accord with the patient’s pregnancies.

There is growing evidence that changes in estrogen levels may play a role in LASIK-induced keratectasia: Estrogen receptors have been identified in the cornea,\(^6\) and exposure to estrogen reduced the biomechanical stability of the cornea in an experimental study.\(^7\) These data suggest that changes in estrogen levels may reduce the biomechanical stability of the cornea, which leads to the onset of iatrogenic keratectasia after LASIK. However, Randleman et al.\(^4\) showed a statistically significantly higher risk for men to develop iatrogenic keratectasia.

Until recently, the only therapeutic option for LASIK-induced keratectasia was keratoplasty. Recently, several groups have reported that CCL with riboflavin and ultraviolet-A can stop and even partially reverse progressive LASIK-induced keratectasia.\(^8,9\) In our case, CCL stabilized the ectasia and led to a partial regression of maximum K-readings over a period of 2 years. However, during the patient’s...
second pregnancy, the keratectasia exacerbated. One might speculate that this exacerbation was due to a “poor” or partial crosslinking effect. The actual amount of the crosslinked stroma cannot be evaluated because no tool provides direct measurement of the CCL effect. However, for several years following the first CCL treatment, the patient’s topographies showed stabilization of the keratectasia. A poor crosslinking effect is therefore highly unlikely.

It is unclear why the left eye showed no progression during the second pregnancy. A possible explanation is that the residual stromal bed thickness was slightly greater than in the right eye, which developed keratectasia. In such borderline cases, minimal biomechanical differences, along with pregnancy-induced changes in estrogen levels, might determine whether a cornea develops keratectasia.

In conclusion, changes in the estrogen level during pregnancy might represent a previously unrecognized risk factor for the induction of keratectasia after LASIK surgery. To our knowledge, this is the first case to demonstrate that CCL may not be able to stabilize iatrogenic keratectasia in borderline cases.

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