

Corneal Thickness after MyoRing Implantation for Keratoconus

Albert Daxer

ABSTRACT

Purpose: To analyze the corneal thickness development after MyoRing implantation as a tissue-related indicator of keratoconus progression.

Materials and methods: Twelve eyes suffering from keratoconus were treated for visual rehabilitation by means of MyoRing implantation into a corneal pocket. The postoperative development of the corneal thickness at the thinnest point was analyzed. Follow-up ranged from 16 to 74 months (mean 37 months).

Results: Out of the 12 eyes only one eye showed a thinner cornea at the last follow-up 60 months after MyoRing implantation compared to the last preoperative data. None of these 12 eyes required an additional intervention like corneal cross-linking, explantation or keratoplasty. Four eyes showed even thickening of the cornea during the postoperative period.

Conclusion: MyoRing implantation for keratoconus does not only allow visual rehabilitation. It also seems to stop the progression of the disease by creating a new biomechanical equilibrium within the tissue which eliminates a constant stimulus that drives the vicious circle of progression.

Keywords: Keratoconus, MyoRing, Corneal thickness, Progression, Corneal cross-linking, PocketMaker.

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INTRODUCTION

Keratoconus is characterized by progressive loss of vision. Corneal cross-linking (CXL) is able to stop the progression of the disease in a high percentage of cases.¹ MyoRing implantation has been demonstrated to be safe and effective in visual rehabilitation of mild, moderate and advanced keratoconus.^{2,3} Single short-term follow-up's report increase of

corneal thickness after MyoRing implantation.^{4,5} It is not clear, however, whether MyoRing implantation alone have a positive impact on long-term corneal stability. Here, I shall present preliminary clinical data on the development of corneal thickness after MyoRing implantation as a tissue-related indicator for progression of the disease.

MATERIALS AND METHODS

Twelve eyes of 12 patients were treated for mild, moderate and advanced keratoconus. Age of the patients ranged from 18 to 37 years (mean 29 years) and postoperative follow-up time ranged from 16 to 74 months (mean 37 months). MyoRing implantation (Dioptex GmbH, Austria) was performed after creating a corneal pocket by means of the PocketMaker Ultrakeratome (Dioptex GmbH, Austria) as described elsewhere.^{2,3} Minimal corneal thickness (CT) and K-value at the steepest point (K_{max}) were measured using Pentacam (Oculus GmbH, Germany). Changes between preoperative and postoperative corneal thickness ranging between +10 microns and -10 microns were considered insignificant and qualified as no change in corneal thickness.

RESULTS

The preoperative and postoperative data are shown in Table 1.

Out of the 12 eyes, one eye (8.33%) showed further thinning, seven eyes (58.33%) remained unchanged and 4 eyes (33.33%) thickened after MyoRing implantation. Eye 3 appeared to have a postoperative progression by 41 microns 60 months after MyoRing implantation. All 12 eyes, however, had a significantly improved visual acuity and were happy with their refractive results which did not worsen postoperatively and no further intervention such as corneal cross-linking (CXL) was required.

Case no. 12 is an exemplary case showing a constant corneal thickness. The patient presented the first time in my practice at the age of 19 years with the refraction $-1.25s = -1.25c \times 160^\circ$ and corrected distance visual acuity (CDVA) of 1.0 (20/20). At that time there were no clinical signs of keratoconus and, therefore, no topography was performed but glasses were prescribed. Three years later the patient, now 22 years of age, presented with the refraction $-4.25s = -6.25c \times 5^\circ$, CDVA of 0.5 (20/40) and UDVA of 0.1 (20/200) with

Director and Professor

GutSehen Eye Center, Ybbs; Department of Ophthalmology
Medical University of Innsbruck, Austria

Corresponding Author: Albert Daxer, Director and Professor
GutSehen Eye Center, Stauwerkstrasse 1, 3370 Ybbs, Austria
Phone: +43741253110, Fax: +4374125311020, e-mail:
daxer@gutsehen.at

Table 1: Age, last follow-up (FU), preoperative corneal thickness (preop CT), postoperative corneal thickness (postop CT) and the changes between preoperative and postoperative corneal thickness (Δ CT) of 12 eyes treated by means of MyoRing implantation

Eye	Age (years)	FU (months)	Preop CT (microns)	Postop CT (microns)	Δ CT (microns)
1	27	30	479	486	7
2	27	31	498	508	10
3	34	60	517	476	-41
4	18	74	514	518	4
5	23	16	448	448	0
6	34	21	466	488	22
7	35	20	470	486	16
8	33	45	511	501	-10
9	37	23	431	422	-9
10	30	49	381	401	20
11	31	18	392	423	31
12	23	62	436	434	-2
Mean	29	37	464	466	2
Std.dev.	6	20	47	39	20

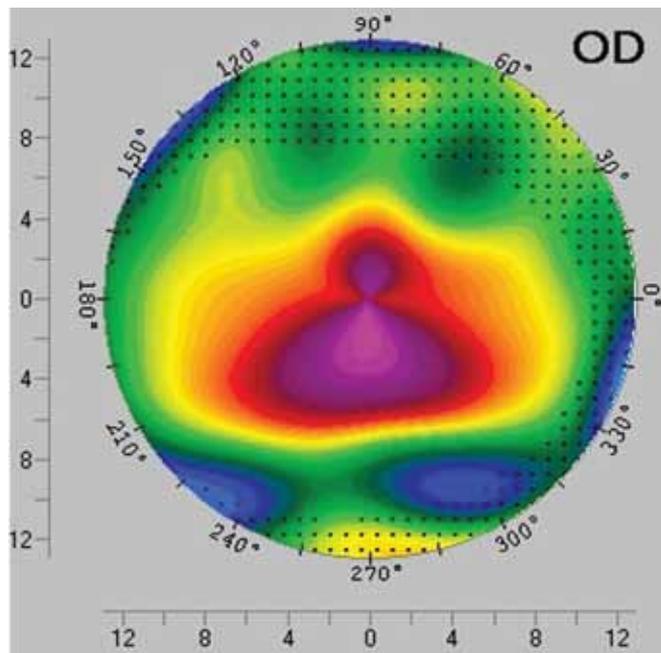


Fig. 1: Preoperative sagittal map of eye 12

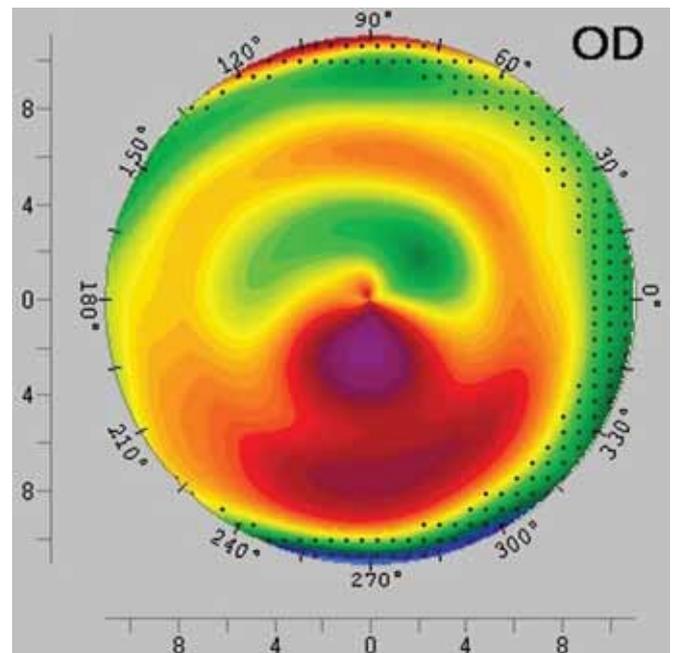


Fig. 2: Sagittal map of eye 12—one year after MyoRing implantation

K_{max} (steepest point) of 60.2 diopters and CT (thinnest point) of 436 microns (Fig. 1). Two months later MyoRing implantation was performed without any intra- or postoperative problems. One year after MyoRing implantation K_{max} was 52.1 dioptres and CT was 425 microns (Fig. 2). Five years after surgery the patient had UDVA of 0.8, CDVA of 0.9 with $+0.5s = -1.75c \times 10^\circ$, K_{max} of 50.7 and CT of 434. The topography 5 years after surgery is shown in Figure 3. Corneal thickness remained, therefore, unchanged compared to the preoperative state immediately before surgery.

Case no. 3 is the exemplary case for postoperative appearance of further corneal thinning without the requirement of further intervention. Preoperatively, the patient presented

with $-0.50s = -4.0c \times 95^\circ$, CDVA 0.6, UDVA 0.02, K_{max} 81.3 dioptres and CT 517 microns at the right eye (Fig. 4). One year after surgery, the eye showed $-4.5s = -4.0c \times 5^\circ$, CDVA 0.7, UDVA 0.6, K_{max} 79.3 dioptres and CT 468 microns (Fig. 5). Five years after MyoRing implantation the eye had $-3.5s = -2.25c \times 5^\circ$, CDVA 0.8, UDVA 0.6, K_{max} 76.2 and CT 476 (Fig. 6). The time between the last preoperative measurement of CT and surgery was 3 months.

Plot of the data according to Δ CT vs follow-up period (Fig. 7) and Δ CT vs Preop CT (Fig. 8) did neither show a correlation between the change of CT and the postoperative follow-up period nor between the change of CT and the preoperative CT.

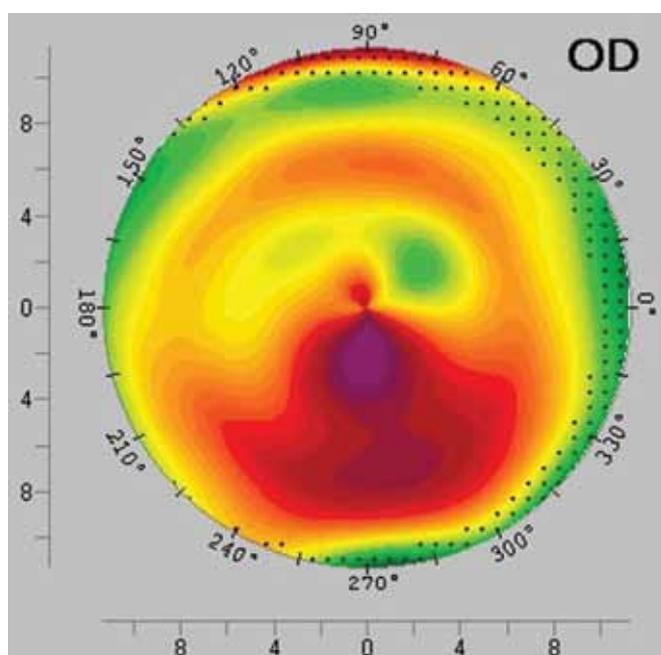


Fig. 3: Sagittal map of eye 12—five years after MyoRing implantation

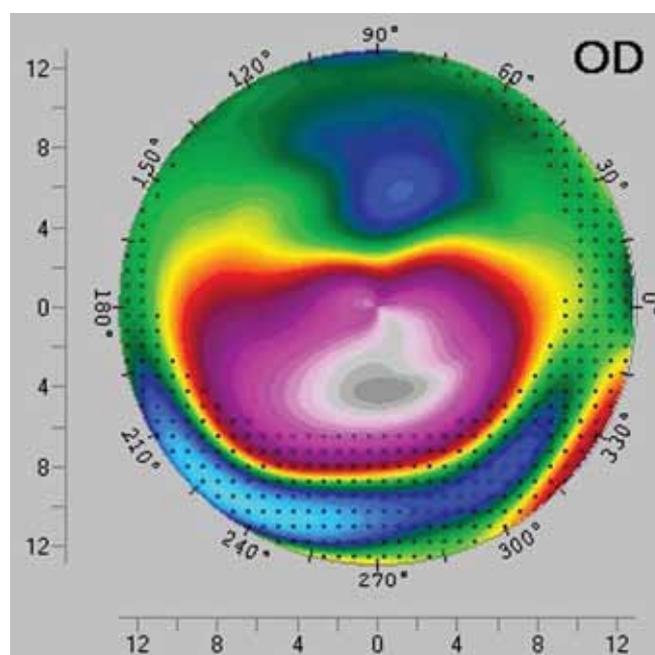


Fig. 4: Preoperative sagittal map of eye 3

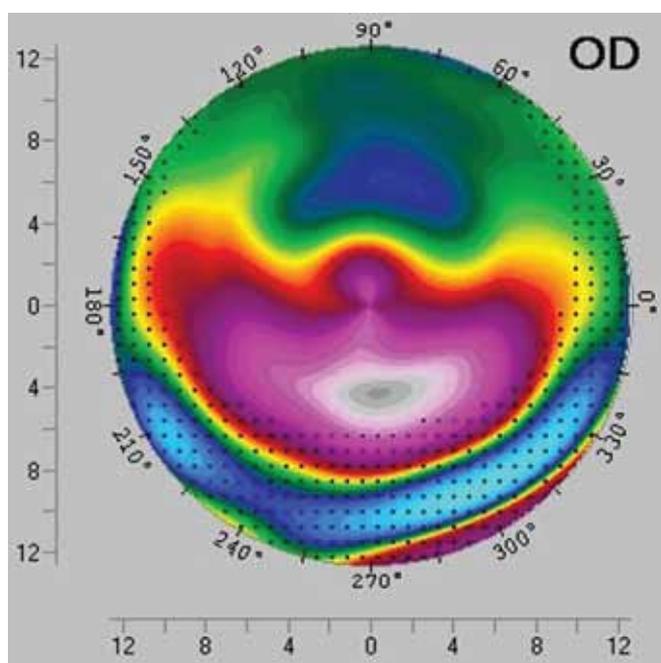


Fig. 5: Sagittal map of eye 3—one year after MyoRing implantation

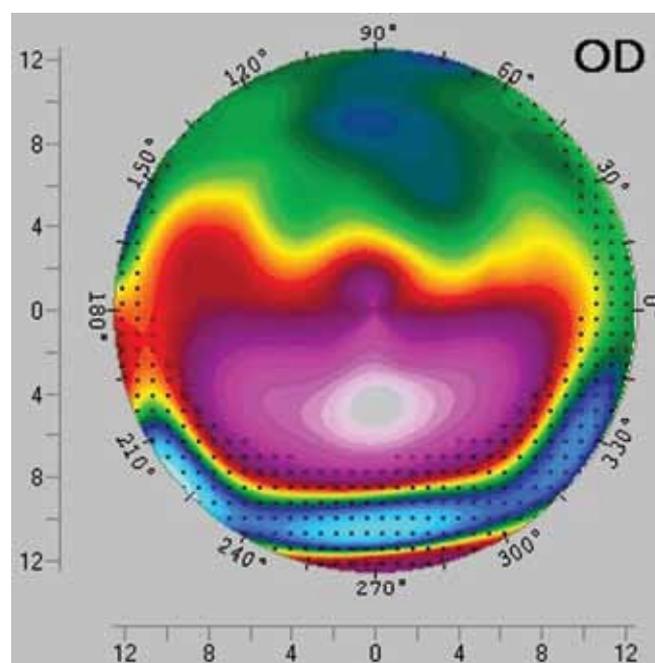


Fig. 6: Sagittal map of eye 3—five years after MyoRing implantation

DISCUSSION

The progression of keratoconus seems to be associated with selective proteolytic activity which alters the regular orthogonal matrix pattern of the corneal lamellae.⁶ Are these ultrastructural changes, which are obviously associated with the biomechanical weakening of the tissue somehow triggered? Is there an initial event that can start the keratectic process? Can we learn from the presented data about basic mechanisms of keratoconus progression?

The presented data suggest that MyoRing implantation alone may have the power to stop progression of keratoconus

without additional CXL. What we obviously do when implanting a MyoRing into a corneal pocket is to change the shape of the cornea and, therefore, the distribution of forces acting within the tissue. In a steeper cornea, the shear stress between two adjacent collagen lamellae for instance should be increased compared to a flatter situation. Since, the MyoRing is placed in a pocket which is larger than the diameter of the implant and not captured in a tunnel like segments are, the cornea can find a new biomechanical equilibrium around the implant and resting biomechanically neutral in the postoperative corneal shape. Is it possible that this new

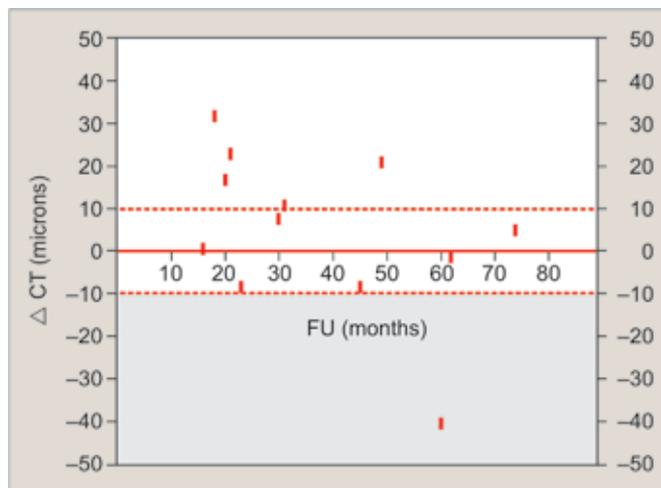


Fig. 7: Change of corneal thickness (Δ CT) in microns between the preoperative and postoperative measurement vs the related postoperative follow-up period (FU) in months. The shaded (dotted) area marks the area of significant progression of corneal thickness

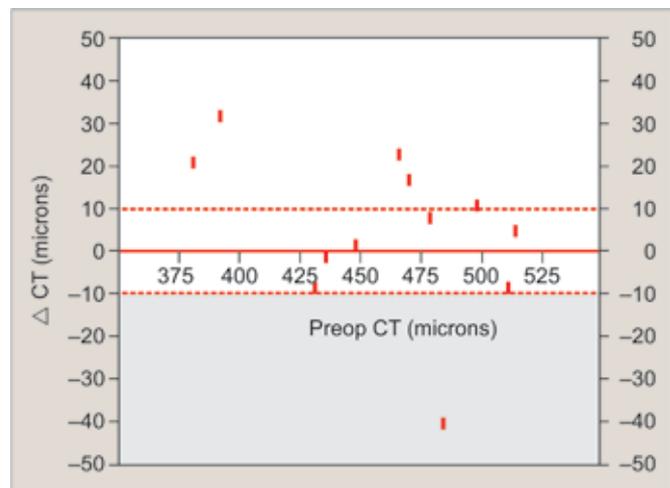


Fig. 8: Change of corneal thickness (Δ CT) in microns between the preoperative and postoperative measurement vs the corneal thickness preoperatively (Preop CT) in microns. The shaded (dotted) area marks the area of significant progression of corneal thickness

biomechanical equilibrium in combination with the new and flatter corneal shape results in a force distribution within the tissue which can stop the progression by eliminating a stimulus for progression and breaking a force driven causal loop?

In order to get an idea to answer this question, we can perhaps learn about related mechanisms from another connective tissue where the force distribution within the tissue can result in a remodeling of the shape of the tissue: the bone. In the bone model, we know that depending on the force distribution within the tissue osteoclastic or osteoplastic activity exists in order to reshape the tissue according to the biomechanical requirements. The morphogenetic control systems responsible for overall bone shape are related to its responsiveness to altered conditions of stress, so that the bone structure is reorganized in a manner which resists the predominating mechanical stresses.⁷ These fundamental mechanisms define, e.g. also the concept of Orthodontology.

Taking the bone model into consideration one may consider the progression of keratoconus as a kind of vicious circle which can be broken either by freezing the actual tissue shape (CXL) or by breaking the vicious circle via the elimination of the stimulus that drives the vicious circle and bringing the cornea in a new biomechanical equilibrium (MyoRing).

This view can be supported by analysing, e.g. the postoperative course of eye 12 as described in the results section above. In this case, there was a dramatic and rapid progression of the disease before MyoRing implantation with K_{\max} of 60.2 dioptres and CT of 436 microns which resulted in K_{\max} of 52.1 dioptres and CT of 425 microns 1 year after MyoRing implantation. The data improved further between postoperative year 1 and postoperative year 5 to K_{\max} 50.7 dioptres and CT 434 microns. This patient showed rapid progression preoperatively and no progre-

ression postoperatively including the last follow-up more than 5 years after MyoRing implantation.

One case (eye 3) is a most unusual case for several reasons. First, it is the only one in the presented series which had a significant reduced CT postoperatively compared to the preoperative measurements. Second, the patient was postoperatively higher myopic than preoperative despite of flattening of the cornea. Third, the preoperative relation between high CT, high K_{\max} and good CDVA was very unusual and fourth, the postoperative relation between high CT, high K_{\max} , good CDVA and very good UDVA was and is most unusual. Five years after MyoRing implantation eye 3 had a CT which was 41 microns less than that measured at the last preoperative examination. There was, however, no thinning during the 4 years between 1 year after surgery and 5 years after surgery. The cornea thickened even insignificantly from 468 to 476 microns within this 4 years postoperative period. Moreover, also refraction, UDVA, CDVA and K_{\max} improved during this postoperative period of time. Unfortunately, the last CT measurement preoperatively was 3 months before surgery and no CT measurement immediately before surgery is available. Therefore, it may not be unlikely that at least a part of the difference between preoperative and postoperative CT can be related to preoperative progression in this case, which should have happened between the date of last preoperative CT measurement and the date of surgery.

Although the cause and underlying mechanisms of keratoconus development and progression are unknown it seems that behavior (e.g. eye rubbing), environmental factors (e.g. dry and hot climate such as in deserts) and genetic factors play a major role.⁸ In agreement with the presented data after MyoRing implantation and the above mentioned discussion of underlying mechanisms one may propose that in the presence of a genetic predisposition, factors like eye rubbing or

unfavorable environment may act as a trigger to start the disease which, according to the ultrastructural changes and force distribution within and between the collagen lamellae, starts and perpetuates a vicious circle of progressive ultrastructural tissue destruction and which appears clinically as progression of the disease. One may conclude from the presented data that bringing the cornea in a new biomechanical equilibrium by means of MyoRing implantation into a corneal pocket a stop of progression by breaking a progression inducing vicious circle will happen.

Further clinical as well as ultrastructural studies are required to clarify this issue in more details.

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