

CASE REPORT

Corneal Thinning after Contact Lens-related Infective Keratitis

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ABSTRACT

Purpose: To describe two cases of contact lens-related infectious keratitis with secondary corneal thinning.

Material and methods: Case reports and review of the literature.

Results: The first case is of a 13-year-old girl, contact lens user, admitted for acute corneal infection in her right eye (OD). Seven years after treatment, she presented with corneal thinning and flattening with uncorrected distance visual acuity of 20/30 OD; thinnest value was 363 μ m in OD and 513 μ m in the left eye (OS). The second case is a 22-year-old male, cosmetic contact lens user, first presented with severe keratitis in OD. One year after treatment, the eye was quiet with moderate corneal opacity; corneal thickness was 228 μ m OD and 561 μ m OS. Ectasia was identified due to the protrusion and steepening with an irregular curvature pattern. Rigid gas permeable (RGP) contact lens fitting enabled visual acuity of 20/25 OD.

Conclusion: Corneal melt with secondary stromal loss and thinning may lead or not to secondary ectasia. Visual rehabilitation is possible with RGP contact lenses despite corneal opacity. The risk of infectious keratitis among contact lenses users should be considered.

Keywords: Corneal ulcers, Ectasia, Contact lenses.

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INTRODUCTION

Corneal ectasia is characterized by thinning and protrusion. Ectatic diseases comprise a range of disorders involving either primary disease conditions such as keratoconus, keratoglobus and pellucid marginal degeneration or secondary ectatic conditions, such as keratectasia after LASIK.^{1,2} Ectasia may also develop after trauma or corneal infection. The condition occurs when there is biomechanical failure so that the normally round dome-shaped cornea progressively assumes a cone-like bulge or forward protrusion, a response of a thin and weak cornea to the normal pressure of the eye.^{3,4} Thinning occurs primarily in the stromal layers. The bulging can lead to an irregular shape or astigmatism of the cornea and usually results in loss of visual acuity which can make even simple daily tasks, such as driving, watching television or reading, difficult to perform.⁵

Corneal remodeling may occur through epithelial thinning and modulation, leading to partial homogenization of the anterior corneal surface for maintaining visual acuity.⁶ Remodeling may occur following riboflavin ultraviolet (UV)-A induced corneal collagen cross-linking (UV-A), but also following an event that results in focal corneal scarring, such as corneal infection. In a highly irregular cornea, the benefit of the flattening effect of a scar may outweigh the increase in aberrations and light scatter.⁷

CASE REPORT

The first case is of a 13-year-old girl who was admitted in the Ophthalmology Department of Centro Hospitalar de Entre o Douro e Vouga, Feira, Portugal, for acute corneal infection in her right eye (OD). She had started wearing contact lens 4 months before and her distance corrected visual acuity (DCVA) was 20/20 both eyes (OU), with a minus sphere of 2.5D. She had no history of corneal disease or trauma. Slit-lamp examination showed central epithelial defect with stromal infiltration and hypopyon. Microbiological examinations were performed from corneal scrapings

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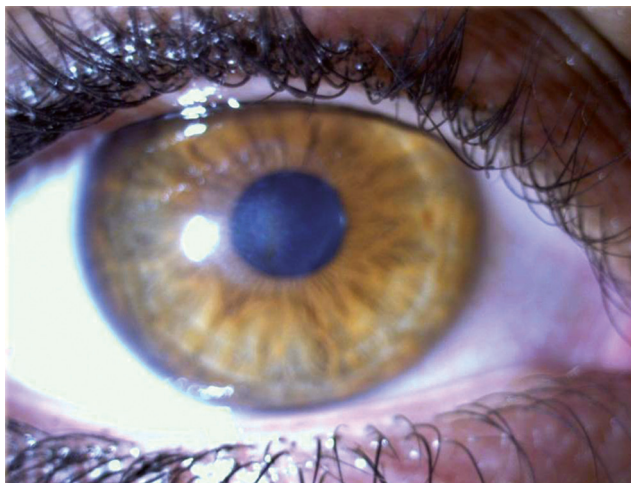


Fig. 1: Residual subepithelial corneal opacity in the right eye (Case 1)

and were positive for *Pseudomonas aeruginosa*. Empiric antibiotic treatment was started with ophthalmic solutions of Cefazolin and Tobramycin, with good clinical response. After healing epithelial defect and having the infectious focus under control, mild topical steroids (*Fluorometholon acetate*) were used to reduce corneal scarring.

Seven years later, she presented with a slight residual subepithelial corneal opacity (Fig. 1). Uncorrected distance visual acuity (UDVA) was 20/30 OD and 20/200 in the left eye (OS). Manifest refraction was a -2.5 cylinder leading to 20/30 in OD and -5.00 sphere in OS, leading to 20/20. Pentacam® showed a thinnest spot in the pachymetry map of 363 μm OD and 513 μm OS and reduced central medium keratometry of 37.0D OD compared to 43.6D OS (Fig. 2).

The second case is a 22-year-old male, cosmetic contact lens user, first presented with severe keratitis in OD. Significant worsening was reported 24 hours after topical

empiric antibiotic therapy, with ciliary injection, stromal infiltration and hypopyon. Microbiologic work up revealed *Pseudomonas aeruginosa*. There was only modest clinical improvement despite intensive topical treatment with fortified antibiotics (Vancomycin and Gentamicin). Empiric treatment for *Acanthamoeba* was initiated with no resolution of keratitis, when he presented to Contarini Eye Clinic, Rio de Janeiro, Brazil (Fig. 3). After 4 negative corneal scrapings, empiric antifungal therapy was initiated with a good clinical response. After healing epithelial defect and reducing stromal infiltrate, topical steroids and immunosuppressive agents (Tacrolimus) were used to minimize stromal scarring. One year later, the eye was quiet and patient was referred to the Instituto de Olhos Renato Ambrósio, Rio de Janeiro, Brazil for considering therapeutic surgery.⁸ At ophthalmological evaluation he presented with moderate corneal opacity in OD (Fig. 4). UDVA was 20/150 in OD and 20/25 in OS and best spectacle corrected visual acuity was 20/40 OD and 20/20 OS. Total corneal thickness was 228 μm in OD and 561 μm in OS. Significant protrusion and irregular curvature in the right eye were observed by corneal tomography (Fig. 5). Due to the severe thinning, no therapeutic corneal ablation was considered. Before keratoplasty, patient was referred for contact lens fitting trial. Rigid gas permeable fitting enabled visual acuity of 20/25 in OD.

DISCUSSION

The breakdown of the unique corneal defenses through alterations in the natural biology of the epithelium, such as those reported during contact lens wear, predispose to infection.⁹ Contact lens-related microbial keratitis is the most visually devastating complication associated with contact

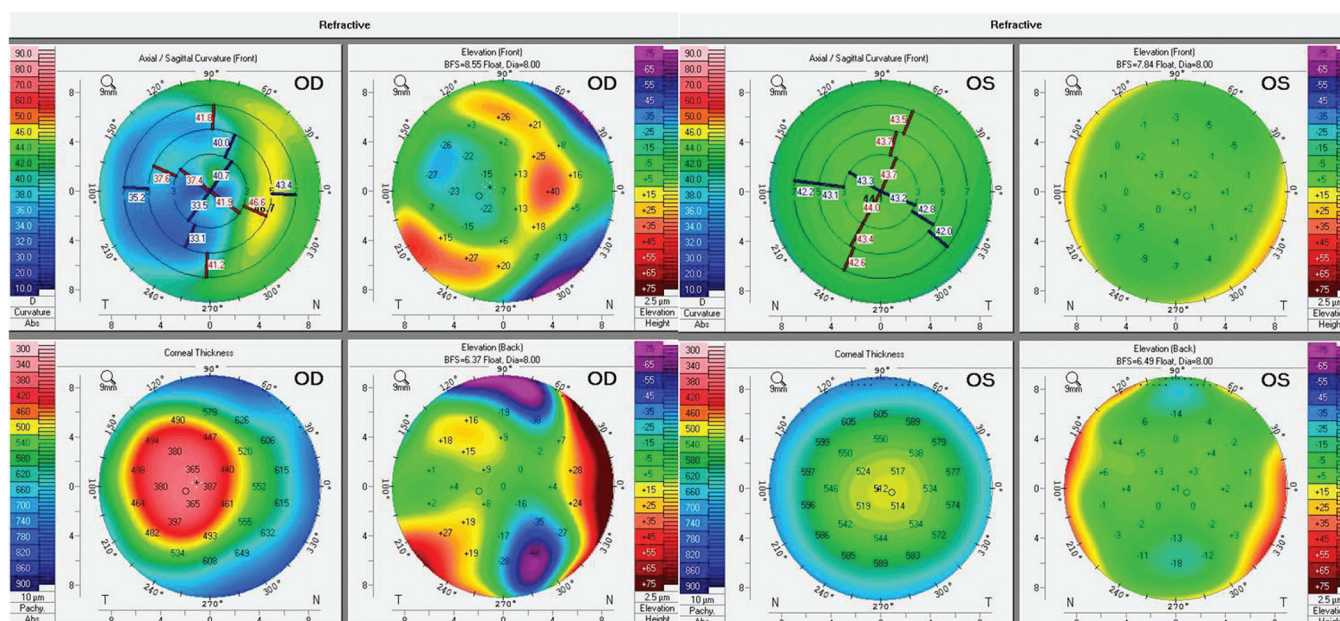


Fig. 2: Pentacam® four maps refractive (Case 1)

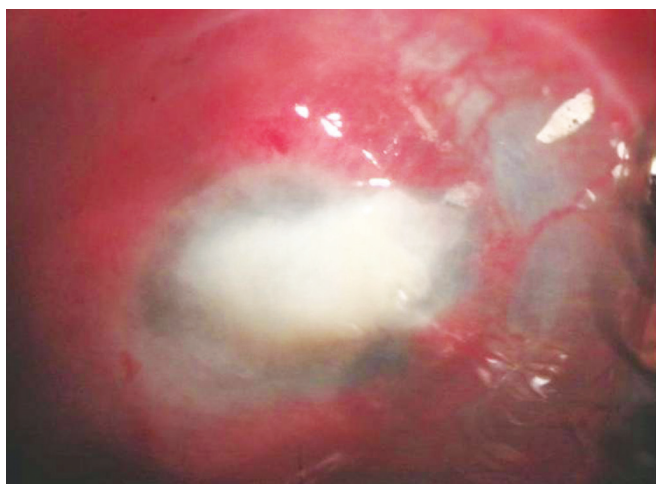


Fig. 3: Right eye biomicroscopy in the first presentation to Contarini Eye Clinic (Case 2)



Fig. 4: Right eye biomicroscopy 1 year after treatment (Case 2)

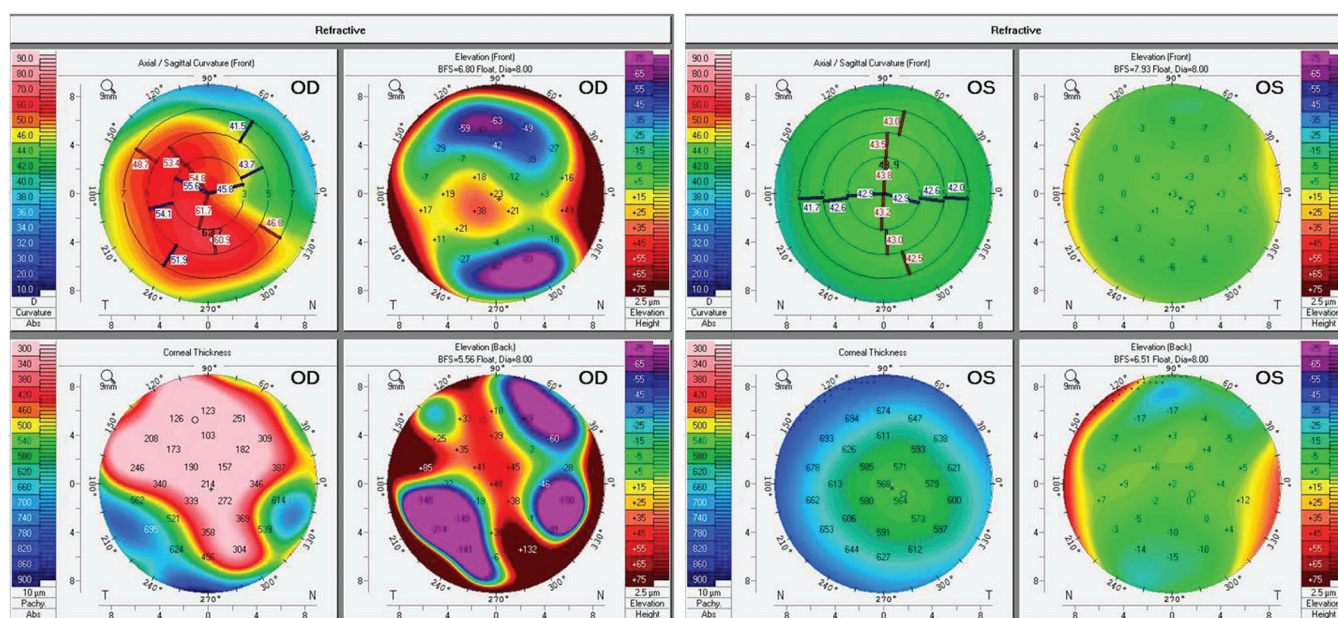


Fig. 5: Pentacam® four maps refractive (Case 2)

lens wear and for more than three decades, *Pseudomonas aeruginosa* has been consistently identified as the primary infectious pathogen.¹⁰⁻¹³ In the healing phase, corneal melt occurs leading to stromal loss and thinning, which may lead to secondary ectasia.

In these two cases, there was thinning due to the corneal melt related to the infection. Secondary ectasia progression occurred in the second case, while the first case had less thinning which did not compromise biomechanical integrity. The result of this process was moderate flattening with partial resolution of myopia, despite of moderate irregularity. Irregular astigmatism with higher order aberrations limits spectacle corrected visual acuity, which could be treated by contact lens or therapeutic corneal surgery.¹⁴

In the second case, corneal thinning was more pronounced leading to biomechanical failure and ectatic progression. Moderate scarring and corneal opacity was also present.

Nevertheless, we obtained good visual outcome with contact lens fitting.

The risk for infectious keratitis among contact lenses users should always be considered. The prevention of lens-induced inflammatory-mediated epithelial surface damage should be the main focus when fitting contact lenses. Patient education has a major role in this prevention process. Epithelial compromise becomes a zero sum game in which lens type, wearing modality and cleaning solution must be optimized.

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