

Case report

Case of progressive hyperopia due to flattening of cornea

Hiromasa Hirai, Shinji Maruoka, Tadanobu Yoshikawa, Nahoko Ogata*

Department of Ophthalmology, Nara Medical University, Nara, Japan



ARTICLE INFO

Keywords:

Hyperopia
 Irregular astigmatism
 Corneal shape
 Flattened cornea
 Anterior segment optical coherence tomography
 Higher order aberrations

ABSTRACT

Purpose: To report a case that had a progressive decrease in the visual acuity caused by a progressive increase in the hyperopia due to a flattening of the cornea.

Observations: A 50-year-old woman complained of decreased vision in both eyes. Her decimal best-corrected visual acuity was 1.2 in the right and 0.5 in the left eyes, and the refractive error (spherical equivalent) was +3.75 diopters (D) for the right eye and +6.5 D for the left eye. Slit-lamp examinations showed clear corneas but Descemet's folds and fine pigmentations and opacities were present beneath the corneal epithelium in both eyes. Analysis of the corneal shape by anterior segment optical coherence tomography showed that the corneas were flattened, and the corneal refractive power was decreased in both eyes. The large values of the higher-order aberration in the cornea and total eye displayed bilateral irregular astigmatism. She obtained good vision by wearing hard contact lenses but her refractive power continued to decrease for at least 1 year.

Conclusions: and Importance: We report a rare case of progressive hyperopia and irregular astigmatism due to a flattening of the cornea. Folds were present in Descemet's membrane but the cornea was not edematous.

1. Introduction

Progressive myopia caused by changes in the corneal curvature is well known as in cases of keratoconus.¹ However, progressive hyperopia is rare and has not been reported except after refractive surgery or due to corneal scars. We report a case of progressive hyperopia due to a flattening of the cornea that presented with no corneal edema but with Descemet's folds.

1.1. Case report

A 50-year-old woman noticed a progressive decrease in her vision in both eyes beginning 2 years before our initial examination. She was prescribed +1.0 diopters (D) spectacles and obtained good vision but her visual acuity progressively decreased even while wearing the glasses. She visited our hospital to try to determine the cause of her progressive decrease in her vision.

Our initial examination showed that she had no history of ocular or systemic diseases. Her decimal best-corrected visual acuity (BCVA) was 1.2 with +2.50–1.25 × 90 in the right eye and 0.5 with +6.50–1.25 × 90 in the left eye. The intraocular pressure was normal in both eyes. The refractive error (spherical equivalent) determined by an auto refractometer (ARK1a[®], NIDEK, Aichi, Japan) was +3.75 D in the right eye and +6.5 D in the left eye. Both of her eyelids and conjunctivas were normal. Slit-lamp examinations showed that the corneas

were almost clear with no edema. However, Descemet's folds were present in the central area and fine pigments and opacities were detected in the superficial stroma of the cornea in both eyes (Fig. 1). The anterior chamber was of normal depth and clear, the lens was transparent, and no abnormalities were observed in the fundus of both eyes.

Anterior segment optical coherence tomography (AS-OCT, Spectralis OCT[®], Heidelberg Engineering, Heidelberg, Germany) showed several hyperreflective regions beneath the corneal epithelium and deep corneal stroma in both eyes (Fig. 2). The central corneal thickness was 541 μm in the right eye and 539 μm in the left eye as determined by a single rotating Scheimpflug camera (Pentacam[®], Oculus, Wetzlar, Germany, Fig. 3). Both values were within the normal range. The refractive power in the central portion of the cornea was 40.3 D in the right eye and 37.5 D in the left eye (Pentacam[®], Fig. 4A and B) which are both weaker than the average refractive power of the central cornea of about +43 D.² AS-OCT showed that both the anterior and posterior surfaces of the corneas were flattened bilaterally. She had bilateral irregular astigmatism, i.e., the values of the higher-order aberration were large in both the cornea and total eye bilaterally and especially in the left eye, determined by a wavefront analyzer (KR-1W[®], TOPCON, Tokyo, Japan, Fig. 5). She was prescribed hard contact lenses and her BCVAs improved to 1.0 in both eyes.

After one year, a progression of the decrease in the corneal refractive power was determined in both eyes. The refractive power in the central portion decreased from 40.3 D to 37.6 D in the right eye and

* Corresponding author. Department of Ophthalmology, Nara Medical University, 840 Shijo-cho, Kashihara, Nara 634-8522, Japan.
 E-mail address: ogata@naramed-u.ac.jp (N. Ogata).

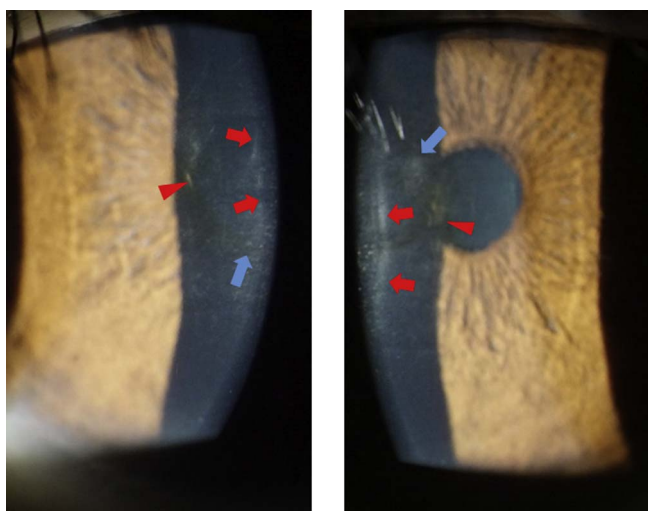


Fig. 1. Photographs of the anterior segment of the eyes (Left: right eye, Right: left eye). Corneas are clear but Descemet's folds are present in the central area (red arrows). Fine pigments (red arrowheads) and opacities (blue arrows) can be seen in the superficial stroma of the cornea in both eyes. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

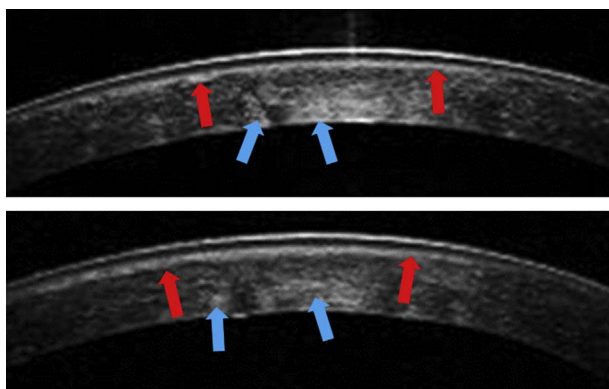


Fig. 2. Anterior segment optical coherence tomographic images (upper, right eye; lower, left eye). A highly reflective layer can be seen under the epithelium (red arrows) and several highly reflective regions can be seen in the corneal stroma (blue arrows) of both eyes. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

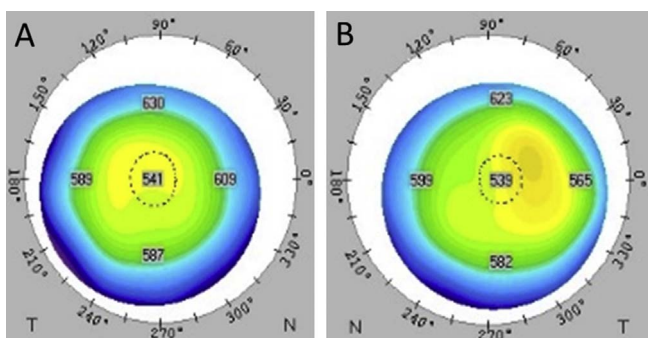


Fig. 3. Corneal thickness determined by Pentacam®. The central corneal thickness was 541 μm in the right eye (A) and 539 μm in the left eye (B). Both values were within the normal range.

37.5 D to 36.1 D in the left eye. These results demonstrated that the corneal flattening was continuing in spite of the hard contact lens wear (Fig. 4C and D).

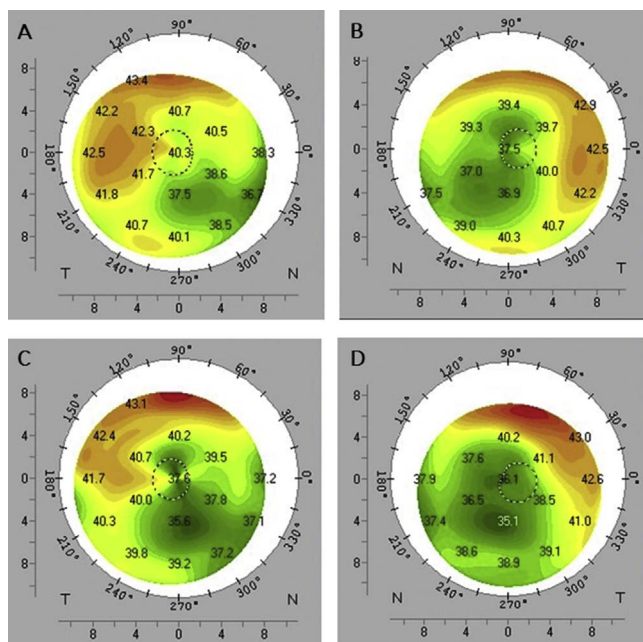


Fig. 4. Corneal topographic map (true net power). A (right eye) and B (left eye) from her first visit. C (right eye) and D (left eye) are from the latest examination (1 year later). The refractive power in the central portion decreased from 40.3 D to 37.6 D in the right eye and 37.5 D to 36.1 D in the left eye. Corneal flattening is progressing.

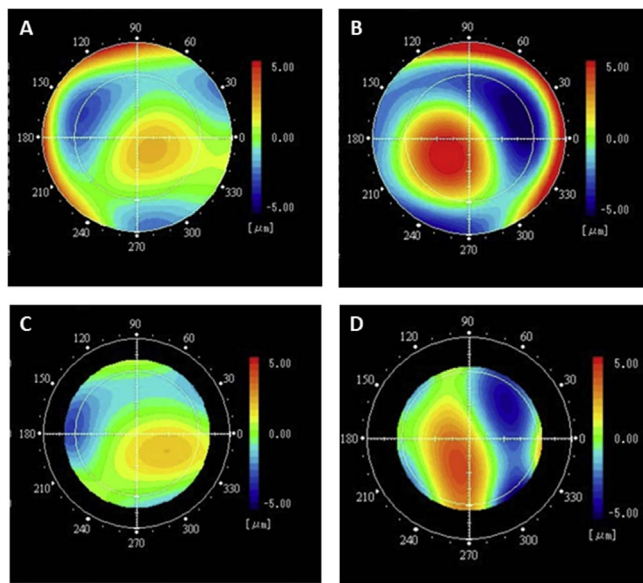


Fig. 5. Maps of corneal and ocular higher-order aberrations. A (upper, left) is the right cornea, B (upper, right) is the left cornea. C (lower, left) is the total aberrations of the right eye, and D (lower, right) is the total aberrations of the left eye. The corneal values of the higher-order aberrations in the left eye (3.237 μm) is larger than that of the right eye (1.562 μm). The total eye value of the higher-order aberrations of the left eye (3.065 μm) is also larger than that of the right (1.451 μm).

2. Discussion

Our results showed that a flattening of the anterior surface of the cornea in both eyes leading to the hyperopia and irregular astigmatism. These changes were the cause of the decreased visual acuities. A search of the PubMed and Medline database failed to find any similar progressive flattening of the cornea.

It is relatively common for hyperopic shifts to occur in eyes with corneal scars such as that after bacterial keratitis or herpetic keratitis or tuberculosis. However, these keratitis are usually unilateral and our

patient presented with fine pigmentations and fine opacities in the corneal stroma bilaterally. Interstitial keratitis due to syphilis is usually bilateral but our patient was negative by blood examinations.

The flattening of the central corneas might result in excessive Descemet's membranes causing the folds in Descemet's membrane. It is well known that Descemet's folds with corneal edema occurs in cases of cataract surgery or infectious keratitis. However, our patient had no history of ocular or systemic diseases, and she never had any injuries of both eyes.

The hyperreflective regions observed by AS-OCT may be due to corneal fibrosis. Central toxic keratopathy (CTK), a rare complication after laser in situ keratomileusis (LASIK) or photorefractive keratectomy (PRK), is characterized by central corneal opacities and hyperopic shifts.^{3,4} Tissue destruction due to abnormal secretion of MMPs has been suggested to be the pathogenesis of CTK.⁴ Our patient had not undergone LASIK or PPK but similar processes of tissue destruction might have occurred. The abnormal secretion of enzymes or proteins from stromal cells, e.g., TGF β -induced proteins (TGF β Ip),⁵ MMPs, and TIMPs⁶ could promote corneal fibrosis which could then result in a flattening of the cornea.

3. Conclusions

We report a rare case of progressive hyperopia and irregular astigmatism due to a flattening of the cornea.

Patient consent

Consent to publish the case report was obtained. This report does not contain any personal information that could lead to the identification of the patient.

Acknowledgments and disclosures

Funding.

No funding or grant support.

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Conflicts of interest

The authors have no financial disclosures relating this topic.

Acknowledgements

None.

Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.ajoc.2018.02.013>.

References

1. Soiberman U, Foster JW, Jun AS, et al. Pathophysiology of keratoconus: what do we know today. *Open Ophthalmol J.* 2017;11:252–261.
2. Jin KW, Shin YJ, Hyon JY. Effects of chalazia on corneal astigmatism : large-sized chalazia in middle upper eyelids compress the cornea and induce the corneal astigmatism. *BMC Ophthalmol.* 2017;17:36.
3. Abdelmaksoud A, Khoo NT, Hanoot H, et al. Bilateral central toxic keratopathy after laser in situ keratomileusis. *BMJ Case Rep.* 2015;10:212423.
4. Davey N, Aslanides IM, Selimis V. A case report of central toxic keratopathy in a patient post TransPRK (followed by corneal collagen cross-linking). *Int Med Case Rep J.* 2017;15:131–138.
5. Murugan E, Venkatraman A, Lei Z, et al. pH induced conformational transitions in the transforming growth factor β -induced protein (TGF β Ip) associated corneal dystrophy mutants. *Sci Rep.* 2016;6:23836.
6. Kenney MC, Chwa M, Alba A, et al. Localization of TIMP-1, TIMP-2, TIMP-3, gelatinase A and gelatinase B in pathological human corneas. *Curr Eye Res.* 1998;17:238–246.