Acquired posterior keratoconus

ROBERT WILLIAMS

From Moorfields Eye Hospital, City Road, London EC1V 2PD

SUMMARY A case of acquired posterior keratoconus is presented. As in developmental cases the vision was moderately reduced and the condition was not progressive. Posterior keratoconus is usually developmental in origin, but trauma has been implicated in some cases.

Posterior keratoconus is a rare corneal disorder characterised by non-inflammatory thinning of the cornea, resulting in a conical protuberance of the posterior surface. The anterior surface remains regular, so differentiating the condition from anterior keratoconus. It is usually considered to be a developmental abnormality.

Case report

A white male was known previously to have had 6/6 vision in both eyes, unaided, and with normal globes. The eyes had been assessed for occupational purposes. At the age of 25 years he suffered a right penetrating injury from the impact of a metal spring. The resulting small corneal perforation did not require surgical treatment, but a secondary cataract developed. This was treated by needling via a limbal approach. At the age of 27 years he was assessed for contact lens correction of his right aphakia. His right visual acuity was then 6/18 with refraction +10-00 D sph −1-50 D cyl axis 30° BVD 11 mm, and keratometry 8-10 mm axis 30°, 7-60 mm axis 120°. The ocular media were clear and the optic disc and macula also appeared normal. The left eye had a visual acuity of 6/6 unaided; keratometry was 8-20 mm axis 30°, 8-15 mm axis 120°, and the anterior segment was normal. Photokeratometry showed the anterior corneal surface to be regular. The central corneal thickness was 0-52 mm in both eyes; in the right this was at the temporal edge of the cone, and the corneal thickness at the cone apex was 0-23 mm.

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Fig. 1 Slit-lamp photograph of acquired posterior keratoconus.

Correspondence to R Williams, FRCS, Worthing Hospital, Park Avenue, West Sussex BN11 2DH.
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Discussion

Posterior keratoconus was described by Butler in 1927. The posterior corneal protuberance is mostly localised, central, and associated with posterior stromal opacity. The visual loss is not progressive and is only moderate. If marked visual loss occurs, another cause must be sought, as has been illustrated by a case of Schocket et al. A Fleischer ring may be present.

Posterior keratoconus is usually considered to be a developmental defect because half the cases are bilateral, and it has been reported in two siblings and also in a parent and child. Krachmer and Rodrigues consider it to belong to the spectrum of anterior cleavage syndromes, as it is frequently associated with other anterior segment anomalies.

Trauma has been proposed as an alternative cause, but before the present case there has been little to support this theory. A history of trauma or ocular disease was present in a minority of cases in one review. However, there were no details of the trauma or the prior visual acuity or corneal appearance, so the history of trauma may be coincidental.

This case shows that some cases of posterior keratoconus may be acquired as a result of trauma. The mechanism in such cases may involve an oblique penetrating injury, with more splitting and destruction of the inner corneal layers than the outer. Alternatively localised damage to Descemet’s membrane or the corneal endothelium may be involved. Either mechanism is likely to be modified by later corneal remodeling. In this case keratometry showed an increase in radius of corneal curvature and the refraction altered, but there was no progression of the visual loss.

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References


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