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CORRESPONDENCE

Sir,

Radial Descemet's membrane folds as a sign of pterygium traction

Pterygium is a triangular[1] fibrovascular neoformation[2] arising from the conjunctiva and encroaching the cornea.[1] This condition is often responsible for irregular with-the-rule astigmatism related to mechanical traction or pooling of tears in front of the pterygium head.[3] We report a case that outlines the role played by mechanical traction on corneal topographic and structural changes.

Case report

A 77-year-old female patient presented with the complaint of a decreased visual acuity on the right side. The patient had undergone uneventful phacoemulsification with posterior chamber lens implantation a year previous to examination. Central corneal curvature measured with an automated keratometer (Nikon Speedy K) was 11.04 mm (175°)/ 8.45 mm (85°) OD and 8.32 (85°)/8.08 (175°) OS. Best-corrected visual acuity OD was 6/12 with a +1.25 correction.

Slit-lamp examination on the right side showed a mildly fleshy nasal pterygium, graded T2 in Tan's classification[4] with a 3 mm horizontal encroachment onto the cornea (Figure 1). Three radial Descemet's membrane folds were visible in front of the pterygium head (Figure 1). The semilunar fold was displaced temporally and its direction was oblique rather than vertical (Figure 1). The rest of the examination on the right side was unremarkable except for pseudophakia. Examination on left side only showed a nuclear cataract limiting corrected visual acuity to 6/18.

After 2 weeks, the patient underwent a right pterygium excision followed by conjunctival autograft under local anaesthesia. The day after surgery, the radial Descemet's membrane folds had disappeared (Figure 2). At 6 months after surgery, no recurrence was noted. Corneal curvature measured with the same method as preoperatively was 8.43 (85°)/8.10 (175°). The patient achieved 6/7.5 with a +1.25 correction.

Comment

Pterygium is known to cause traction on the cornea,[3] resulting in irregular, hemimeridian with-the-rule astigmatism.[3,5] In our patient, traction resulted in Descemet's membrane radial folds. It is possible that the intraocular pressure decrease that occurred during the phacoemulsification procedure, performed a year previous to initial examination, favoured the folds development through less corneal resistance to pterygium traction. Disappearance of the folds on the first day following pterygium removal proves that the folds were related to pterygium traction on the cornea. Traction relief led to a complete keratometric resolution of with-the-rule astigmatism[6] and even a shift to a moderate against-the-rule astigmatism making eventually corneal curvatures quite similar on both sides. In our patient, fibrovascular traction was the cause of structural and topographic corneal changes even though the pooling of tears at the pterygium head is known to play an important role on corneal topography in most cases.[7]

Even though preoperative automated keratometry showed a high with-the-rule astigmatism on the right side, the patient's subjective refraction was spherical. It is known, however, that the induced irregular astigmatism caused by pterygia is not always apparent by subjective refraction.[8]

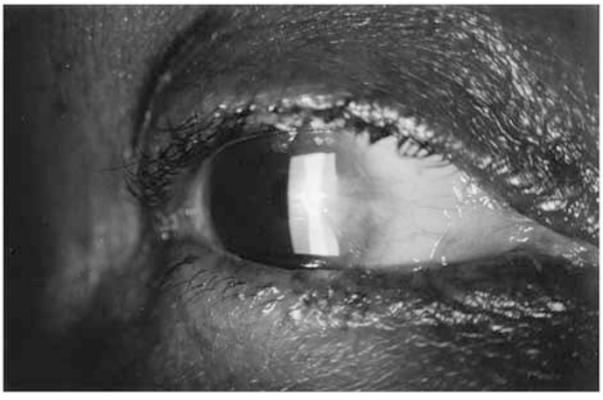

Figure 1 Descemet's folds radiate from the pterygium head and are best observed when the patient looks temporally as the traction exerted on the cornea is increased. The semilunar fold is displaced temporally and obliquely tilted. This ectopia is another consequence of the pterygium traction.

Figure 2 Descemet membrane folds disappeared the day following pterygium removal followed by conjunctival autograft. This photograph, taken a week after surgery shows complete resolution of the folds. Best-corrected visual acuity is 6/7.5.

Visual recovery with a spherical subjective refraction 6 months after pterygium removal retrospectively proves that irregular hemimeridian[3] astigmatism (with presumably a much flatter cornea on the nasal side) and probably the Descemet's membrane folds themselves were the cause for patient's decreased visual acuity.

In the case of extended pterygia, fibrous tissue creates strong adhesions with the medial canthus structures[5] resulting in a traction effect at both pterygium extremities.[5] In our opinion, the semilunar fold temporal ectopia is thus explained.

Buratto reports having observed, in very advanced pterygia, fine Descemet's membrane striae when the patient is asked to look temporally, this manoeuvre resulting in a dynamic corneal deformation.[5] Our patient presented proper radial Descemet's membrane folds visible in primary position of gaze and slightly more obvious in abduction. In our opinion, this sign is extremely rare as the presented patient is the only one in which the authors observed it. This sign may sometimes be overlooked in cases involving inner cornea visualisation difficulties. Patients with a large hood or with recurrent pterygia in whom stromal opacities sometimes remain at the excision site may represent such cases.

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E Denion, P-H Dalens, P Huguet, J Petitbon and M Gérard

Ophthalmology Department
Cayenne Hospital
French Guiana, France

Correspondence: E Denion
Hôpital de Cayenne
Service d'ophtalmologie
Rue des Flamboyants
BP 6006
97306 Cayenne Cedex
French Guiana, France
Tel: +33 05 94 39 50 12
Fax: +33 05 94 39 50 22
E-mail: eric.denion@caramail.com

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Sir,

A late presentation of ocular quinine toxicity managed with a combination of vasodilatory treatments

Case report

A 39-year-old female patient presented to our A + E Department 12 h after taking an overdose of quinine sulphate (300 mg tablets × 28).

She complained of blurred vision and nausea. Blood pressure was 105/55 mmHg, pulse 144 min^{-1}, ECG showed sinus-tachycardia. Intravenous fluids, antiemetics, and 50 g of activated charcoal were administered.

After 6 h, the right visual acuity (RVA) deteriorated to hand movements. The left eye was amblyopic, pre-existing acuity being perception of light (PL). She was referred to ophthalmology the next day.

At the first ophthalmology review (40 h after overdose), visual acuity was PL bilaterally with inaccurate projection. Pupils were dilated with sluggish direct and consensual responses. Fundoscopy showed attenuated arteries, retinal pallor and normal discs (Figures 1 and 2). Fluorescein angiogram (FFA) demonstrated normal arm-to-circulation time with bilateral rapid disc filling in the choroidal phase. Arterio-venous transit time was within normal limits with no evidence of damage to the outer blood retinal barrier. Oral nimodipine (60 mg × 6 day^{-1}) was started.

Vision remained unchanged by the fourth admission day, so intravenous clonidine infusion (300 μg/24 h) was administered for 24 h in HDU. Right stellate ganglion block (SGB) (ropivicaine1% 10 ml) was performed 1 hr after the start of the infusion without complication.

On the seventh admission day RVA started to improve. By day 9, RVA was 6/12 unaided, 6/9 with pinhole. The left amblyopic eye remaining PL. Fundoscopy showed arteriolar attenuation with pale discs. Right colour vision was impaired (17/21 Ishihara plates correct) with grossly constricted visual field.