Figure 2 Focal macular electroretinogram (FMERG, top), full field electroretinogram and visual evoked response (VEP) from both eyes (bottom). Top: Upper three recordings show the photopic α - and β -waves, and lowest recordings show the oscillatory potentials (Ops). The stimulus spot size was 5°, 10° and 15° as indicated in the figure. Left and centre: FMERGs recorded during the acute phase. The ERGs of the left eye (centre) are reduced compared with those of the right eye. Right: FMERGs recorded on the following day showing the recovery of all components of the left eye. The α -wave is not affected as much as the β -waves and Ops. Bottom left: Bright-flash ERG showed no significant difference in the two eyes even in the oscillatory potentials which are sensitive to retinal ischaemia. Bottom right: The VEPs showed no significant difference between the two eyes. The stimulus onset of full field ERG and VEP is indicated by arrows.

appearance; multifocal ERGs or FMERGs are useful in determining the site of the pathology.

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In-vivo scanning of Ascher intrastromal corneal ring opacity

Case report

An 80-year-old man was seen with abnormal corneal appearances. He was asymptomatic with

Figure 1 Right eye: intrastromal corneal ring 0.1 mm in width, 7.8 mm in vertical diameter.

no previous ocular, medical or drug history. Visual acuities were 6/6 in both eyes unaided; with ocular examination revealing bilateral 0.1 mm wide white intrastromal corneal ring opacities 7.8 mm and 7.7 mm in diameter in the right and left eyes, respectively (fig 1). Corneal sensation was normal with no evidence of thinning, scarring or vascularisation. The remaining ocular examination was normal. Blood tests for lipid, protein and autoimmune markers were normal. Corneal pachymetry and topography was unremarkable. Cross-sectional imaging of the cornea with anterior segment optical coherence tomography (Visante OCT, Carl Zeiss Meditec, Inc., USA) revealed opacification affecting the entire thickness of the stroma (fig 2). Confocal microscopy (HRT II/Rostock Cornea Module; Heidelberg Engineering GmbH, Germany) of the ring opacity revealed microdot extracellular deposits within the corneal stroma (fig 3). The epithelium and endothelium of the cornea appeared normal. Ocular examination of the patient's sister was unremarkable, with another sister unavailable for examination. There has been no progression with over 12 months follow-up with the patient remaining asymptomatic.

Comment

Originally reported by Ascher¹ in 1963, intracorneal rings are rare,^{2–8} occurring both unilaterally

Figure 2 Optical coherence tomography of left cornea: highly reflective deposits are seen throughout the entire thickness of the corneal stroma.

Figure 3 Confocal microscopy of left cornea (depth 352 μm): microdeposits are visible as sparkling corpuscles spread within the extracellular tissue of the stroma. Collagen is seen located in the centre of the image with surrounding keratocytes.

or bilaterally, varying in both ring width and diameter in patients 25–80 years of age. Haematological and autoimmune investigations have been normal and family members have not been affected. Pachymetry and specular microscopy have been unremarkable.^{3–4} Bopp and Laqua⁵ described minute greyish dots distributed within outer stromal layers forming an arcus lipoides-like circular band on biomicroscopy. In contrast, Melles *et al.*⁶ could not detect corneal deposition on biomicroscopy but commented on acoustic reflectivity similar to corneal tissue.

We performed optical coherence tomography, which demonstrated the full thickness involvement of the corneal stroma. Confocal microscopy of the ring opacity revealed the presence of highly reflective extracellular opacities located within the affected stroma.

Hypotheses for their aetiology include an unknown corneal dystrophy or degeneration.⁶ Bron² suggested that the rings represent an immunological reaction, with stromal damage at the site of previous antigen–antibody interactions, although their discreteness and stability would argue against this. The appearance of the rings, particularly on confocal microscopy, would suggest that they represent anomalous exogenous or endogenous extracellular microdeposits in the corneal stroma. Their apparent stability, discrete positioning in the midperipheral cornea and lack of any associated infiltrative or inflammatory response is possibly suggestive of altered collagen fibril formation.

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Figure 1 (A) Retinal vein diameters (arbitrary units) in the temporal inferior vessel arcade and the temporal superior vessel arcade and as mean of all measurements in 24 patients with retinal vein occlusions without treatment during follow-up. (B) Retinal vein diameters (arbitrary units) in the temporal inferior vessel arcade and the temporal superior vessel arcade and as mean of all measurements in 21 patients with retinal vein occlusions receiving an intravitreal injection of approximately 20 mg triamcinolone acetate.

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Decreased retinal vein diameter after intravitreal triamcinolone for retinal vein occlusions

Recent studies have suggested that macular oedema caused by central retinal vein occlusion may be treated by intravitreal triamcinolone acetate.^{1–5} In these investigations, macular oedema and retinal thickening markedly decreased and visual acuity temporarily increased after the intravitreal application of triamcinolone acetate. The purpose of the present study was to evaluate whether the diameters of the retinal veins change after the intravitreal injection of triamcinolone acetate. It may give hints for alterations in the retinal haemodynamics after the intravitreal application of triamcinolone acetate.

Care report

The comparative non-randomized clinical interventional investigation included a study group of 21 patients (21 eyes) with central retinal vein occlusion (n = 9) or branch retinal vein occlusion (n = 12) who received an intravitreal injection of approximately 20 mg triamcinolone acetate as described recently.³ Mean age was 69.3 ± 8.6 years, mean refractive error was 0.36 ± 1.12 diopters, and mean follow-up was 8.9 ± 5.9 months (range 2.1–22.2 months). A control group consisting of 24 patients (24 eyes) with central retinal vein occlusion (n = 9) or branch retinal vein occlusion (n = 15) did not receive any treatment. Mean age was 61.8 ± 14.3 years, mean refractive error was 0.32 ± 1.81 diopters, and mean follow-up was 7.6 ± 5.8 months (range 2.0–23.3 months). On fluorescein angiograms obtained at baseline and during the follow-up, the diameters of the affected veins were measured at the inferotemporal and superotemporal optic disc border, above and below the fovea, and between the optic disc and the infrafoveal location or the suprafoveal location.

Figure 2 Scattergram showing the change in retinal vein diameter related to the change in visual acuity during follow-up in 21 patients with retinal vein occlusions receiving an intravitreal injection of approximately 20 mg triamcinolone acetate; the relationship was statistically not significant (p=0.18).

The measurements were obtained in relative size units.

At baseline, the study group and control group did not vary significantly in age (p = 0.09), gender (p = 0.56), refractive error (p = 0.34), and follow-up (p = 0.43), nor in the diameter measurements of the retinal veins (p>0.30). In the study group and in the control group, retinal vein diameters decreased significantly during follow-up (table 1; fig1). The amount of decrease in the retinal vein diameters did not vary significantly between the study groups (p>0.15; table 1). Visual acuity increased significantly from 0.95 ± 0.60 logarithm of the minimum angle of resolution (logMAR) to 0.78 ± 0.69 logMAR (p = 0.006; 95% confidence intervals (CI) –0.28, –0.05) in the study group, and did not change significantly in the control group (0.51 ± 0.36 logMAR versus 0.44 ± 0.35 logMAR; p = 0.17; 95% CI –0.17, 0.03). In the study group, the change in retinal vein diameter was

statistically not associated with a change in visual acuity (p>0.15) (fig 2).

Comment

The results suggest that in patients with retinal vein occlusions, a triamcinolone-associated increase in visual acuity is statistically independent of a concurrent decrease in retinal vein diameter. In agreement with other studies, it may be an indication that the anti-oedematous effect of intravitreal triamcinolone occurs at other vascular levels, such as the capillary bed, than at the level of the large retinal veins. As the decrease in retinal vein diameters did not vary significantly between the treatment group and the control group, one may infer that intravitreal triamcinolone may not have a marked effect on retinal vein dilatations, and indirectly, on the retinal vein outflow resistance, in eyes with retinal vein occlusions.

Table 1 Diameters of retinal veins in relative size units (mean ± SD) at baseline of the study and during follow-up in 45 patients with retinal vein occlusions who either received an intravitreal injection of triamcinolone acetate (study group) or who did not receive any treatment (control group)

	Baseline	Follow-up	p Value	95% CI
Study group				
n	21			
Temporal inferior vessel arcade	0.125 ± 0.032	0.111 ± 0.027	0.031	–0.023, –0.001
Temporal superior vessel arcade	0.135 ± 0.029	0.114 ± 0.025	0.002	–0.030, –0.008
Total	0.130 ± 0.025	0.113 ± 0.022	0.001	–0.024, –0.007
Control group				
n	24			
Temporal inferior vessel arcade	0.127 ± 0.035	0.104 ± 0.021	<0.001	–0.032, –0.010
Temporal superior vessel arcade	0.126 ± 0.034	0.100 ± 0.027	<0.001	–0.037, –0.015
Total	0.126 ± 0.029	0.102 ± 0.021	<0.001	–0.033, –0.015

CI, Confidence interval.
p Value, Statistical significance of difference between baseline values and measurements obtained during follow-up.