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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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.

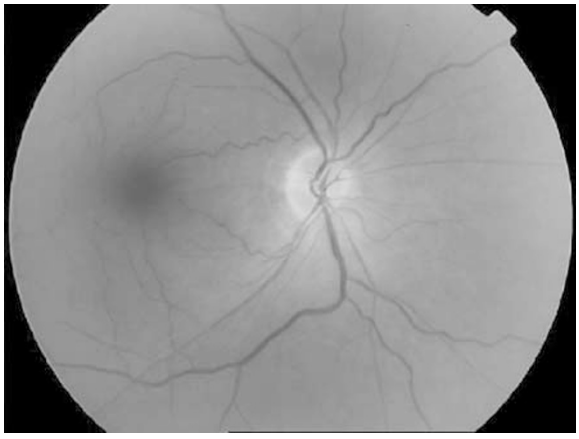

Figure 1 Colour fundus photograph of right eye.

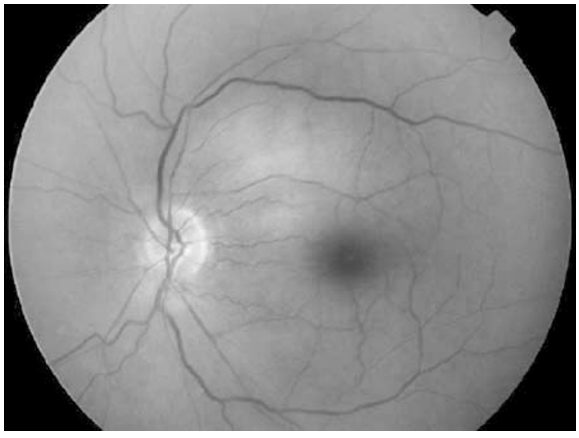

Figure 2 Left eye of case of ocular quinine toxicity 40 h after overdose demonstrating marked arteriolar attenuation and retinal pallor.

After 1 month, RVA was 6/9 + 2 with pinhole, LVA was PL. Optic discs were atrophic and arteries attenuated. She was registered blind due to single grossly constricted visual field.

Comment

Ocular quinine toxicity produces symptoms of blurred/loss of vision, defective colour vision, and constricted visual field. Boland's study of 165 cases reported visual disturbance in 42% of cases[1]. Symptoms are likely to occur after a single dose of >4 g and death can occur with doses of 8 g.[2]

Proposed mechanisms of quinine toxicity include direct toxic damage to neuroretina or ischaemia secondary to arterial vasoconstriction.[3] The resulting generalised vasospasm without alteration of the blood-retinal barrier accounts for the angiographic features seen in Figures 1 and 2.

Our case presented late (>40 h) for ophthalmology assessment with reduced visual acuity, dilated, sluggish pupils, and retinal arterial attenuation. As vasodilatory treatments such as intravenous nitrates have been reported to improve outcome,[4] we administered a combination of vasodilatory treatments—oral nimodipine, clonidine infusion, and SGB. Favourable outcome has been reported with SGB when administered early.[5] Risk of complications including haemo/pneumothorax, intra-arterial injection, phrenic or recurrent-laryngeal block must be considered. In our patient, a unilateral block was performed late without complication on the side of the nonamblyopic eye.

Right visual acuity improved 48 h after the above treatment continuing over the next 2 days. It was not possible to quantify the effect of treatment on the left eye due to pre-existing amblyopic.

Interventional case reports in the literature to date have only involved early intervention, often before the onset of symptoms. Our case illustrates that a combination of therapeutic interventions to improve retinal blood flow may aid in recovery, even in cases presenting late.

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