Sir,

Decompression retinopathy following laser peripheral iridoplasty for acute primary angle-closure

Peripheral iridoplasty using either argon or diode laser has been reported highly effective in lowering the intraocular pressure (IOP) in acute primary angle-closure (APAC).[1] Rapid reduction of the IOP during APAC can result in decompression retinopathy.[2,3] We report two cases of decompression retinopathy following laser peripheral iridoplasty in two patients with APAC.

Case reports

Case 1: A 57-year-old Chinese woman presented with a 4-day duration bilateral APAC after taking antitussive (dextromethorphan HBr, phenylpropanolamine, promethazine). The vision was 6/20 and the IOP was 68 mmHg in both eyes. She had no history of hypertension but the blood pressure was transiently elevated to 177/100 mmHg at presentation. She had migraine treated with panadol and tenormin. She was also an α-thalassaemia trait. Her complete blood count and clotting profile were normal. She was treated with immediate argon laser peripheral iridoplasty, tiomolol, and pilocarpine eyedrops without systemic carbonic anhydrase inhibitor. The IOP was lowered within 30 min to 34 and 30 mmHg in the right and left eye, respectively. In 2 h, the IOP was 16 mmHg both eyes. Fundal examination at that time showed normal optic nerve head appearance in both eyes. On the next day before laser peripheral iridotomy was performed, the patient complained of a central scotoma in her left eye and the vision dropped to 6/120 while the right eye was 6/15. Examination revealed scattered pre- and intraretinal haemorrhages with involvement of the macula in her left eye and disc oedema in both eyes (Figure 1a and b). The haemorrhages and the disc oedema resolved completely

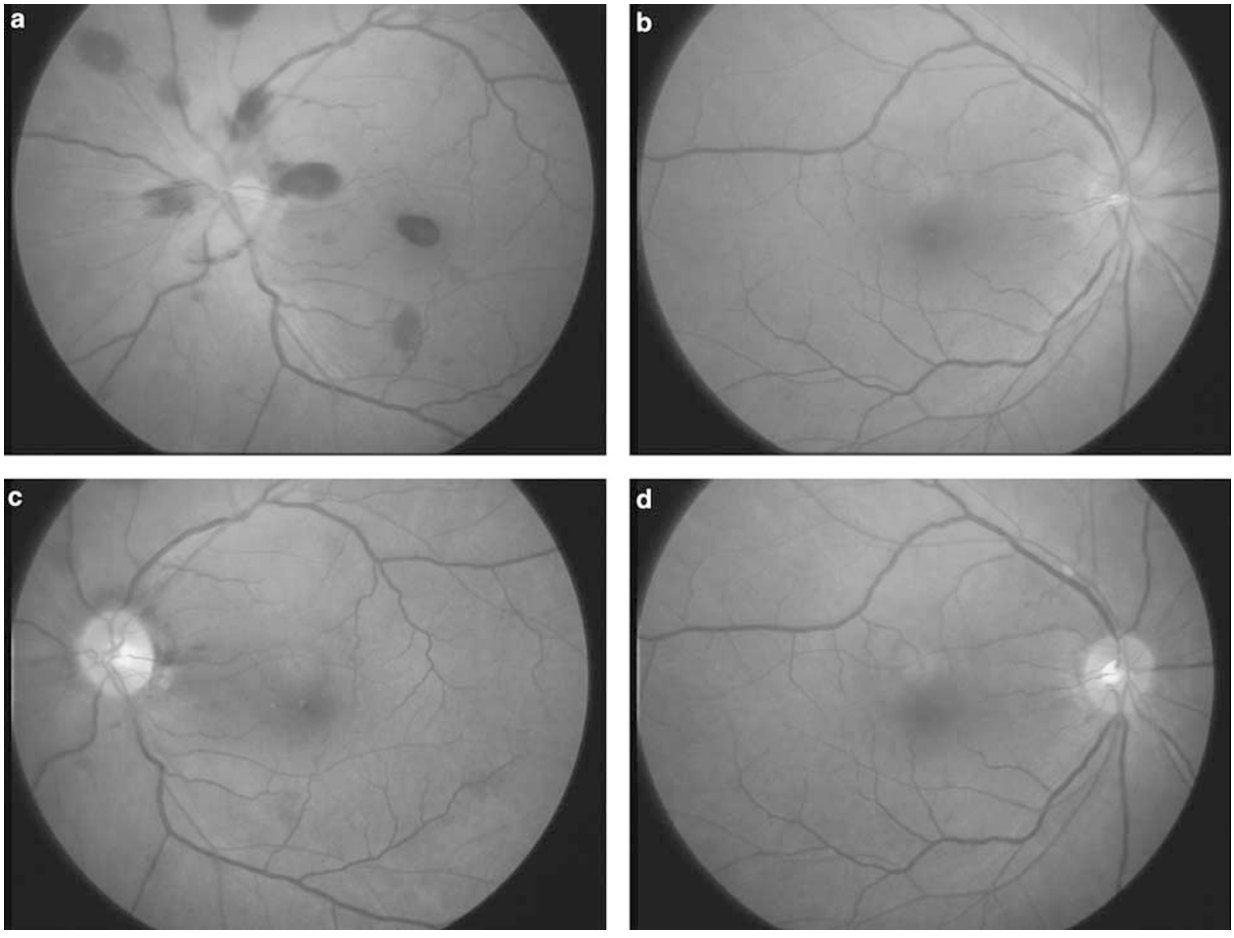

Figure 1 (a) Left eye showing disc oedema and scattered retinal haemorrhages involving the macula. (b) Right eye showing disc oedema. (c) Left eye showing resolution of the retinal haemorrhages and disc oedema. (d) Right eye showing resolution of disc oedema.

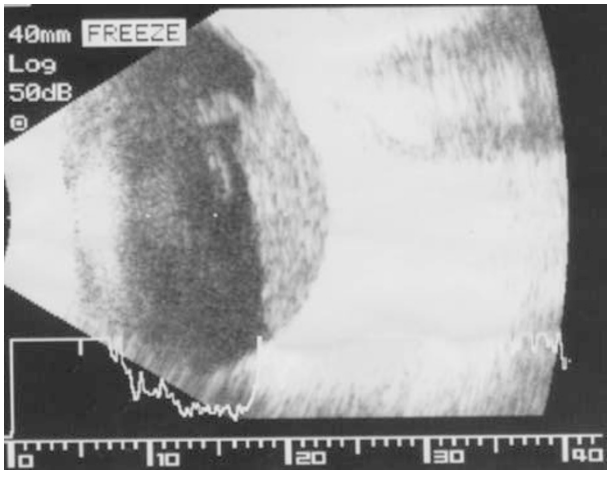

Figure 2 Left eye B scan ultrasound showing preretinal haemorrhage.

after 4 weeks and the left eye vision returned to 6/15 (Figure 1c and d).

Case 2: A 57-year-old Chinese woman presented with a 12-h duration APAC in her left eye. She was healthy and blood investigations were normal. The vision at presentation was 6/60 and the IOP was 68 mmHg. She was treated with topical timolol, piolcarpine, and systemic aceazolamide. However, the IOP remained at 52 mmHg after 6 h. Diode laser peripheral iridoplasty was performed. The IOP decreased to 28 mmHg in 1 h. Fundal examination 2 h later showed a normal optic disc. On the next day before laser peripheral iridotomy was performed, she was found to have preretinal and vitreous haemorrhages in her attacked eye over the macula (Figure 2). At 4 months after the attack there was still residual blood clot in the vitreous. The vision returned to 6/9 and the IOP was 11 mmHg.

Comment

Decompression retinopathy has been reported in APAC following laser peripheral iridotomy.[2,3] It was believed that the haemodynamic change associated with the sudden decrease in the markedly elevated IOP produced the retinal haemorrhage. Laser peripheral iridoplasty has been shown to lower the IOP rapidly in APAC.[1] As a result of the rapid IOP reduction, it carries the risk of decompression retinopathy. The retinal and the vitreous haemorrhage in our patients could be explained by the sudden reduction of IOP. The disc oedema of the first patient could be explained by a forward shift of the lamina cribrosa with acute obstruction of the axonal flow from rapid reduction of the IOP. Our first patient had migraine and was also an α-thalassaemia trait both of which were associated with haematological and vascular abnormality. Whether these were the predisposing factors for the decompression retinopathy is unknown. In previous reports of decompression retinopathy following laser peripheral iridotomy in APAC, both patients were female in the 50–60 years age group. Our two patients are also females and in the 50 s. Female gender may not be a true risk factor and its predominance may be explained by the fact that APAC is more common in female.

Although decompression retinopathy usually presents with retinal haemorrhages and resolves spontaneously within a few weeks, it can lead to preretinal and vitreous haemorrhage and may take months to resolve. In our two patients, there was complete recovery of the visual acuity and there was no residual visual field defect at their last follow-up. Waheeb *et al*[3] reported a case of decompression retinopathy following laser peripheral iridotomy and recommended significant IOP reduction before laser treatment. In our cases, the retinal haemorrhages and disc oedema occurred soon after laser peripheral iridoplasty before laser peripheral iridotomy was performed. This illustrated that the rapid IOP reduction itself might be the cause of the decompression retinopathy and lowering the IOP significantly before laser peripheral iridotomy may not necessarily prevent its occurrence. Although urgent IOP reduction is crucial in preventing irreversible nerve damage in APAC, the potential risk of decompression retinopathy from rapid IOP reduction using laser peripheral iridoplasty should be born in mind. We recommend thorough fundal examination after the acute attack is aborted.

Acknowledgements

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Sir,

Bilateral juvenile glaucoma with iridotrabecular dysgenesis, congenital ectropion uveae, and thickened corneal nerves

Congenital ectropion uveae (CEU) is a rare, nonprogressive neural crest cell disorder resulting from proliferation of iris pigment epithelium on the anterior surface of iris from the pigment ruff. The association of glaucoma in this primary iris pigment epithelial hyperplasia, which may present at birth, infancy, or later stages in life has been well documented in the literature.[1–6] Systemic associations often reported are neurofibromatosis, facial hemihypertrophy, Prader–Willi syndrome, and Rieger's syndrome.[2]

It is characteristically unilateral with ipsilateral glaucoma, with only one prior case reported of bilateral congenital ectropion uveae with bilateral glaucoma.[4] There is one previous case report of unilateral CEU with associated thickened corneal nerves.[5]

We present here a case of bilateral juvenile glaucoma with iridotrabecular dysgenesis, congenital ectropion uveae, and thickened corneal nerves, which has not been reported earlier.

Case report

A 22-year-old male presented with painless gradual loss of vision in both eyes during the previous 7 years. His best-corrected visual acuities were 20/60 in right eye and 20/80 in left eye. The applanation intraocular pressure (IOP) on examination was 30 mmHg in right eye and 37 mmHg in left eye.

Congenital ectropion uveae was present in both eyes, extending 360° around the pupil to the mid-periphery of the iris, where it extended in a sharply demarcated scalloped border with the rest of the iris having smooth, cryptless appearance (Figure 1a, b). The pupil itself was

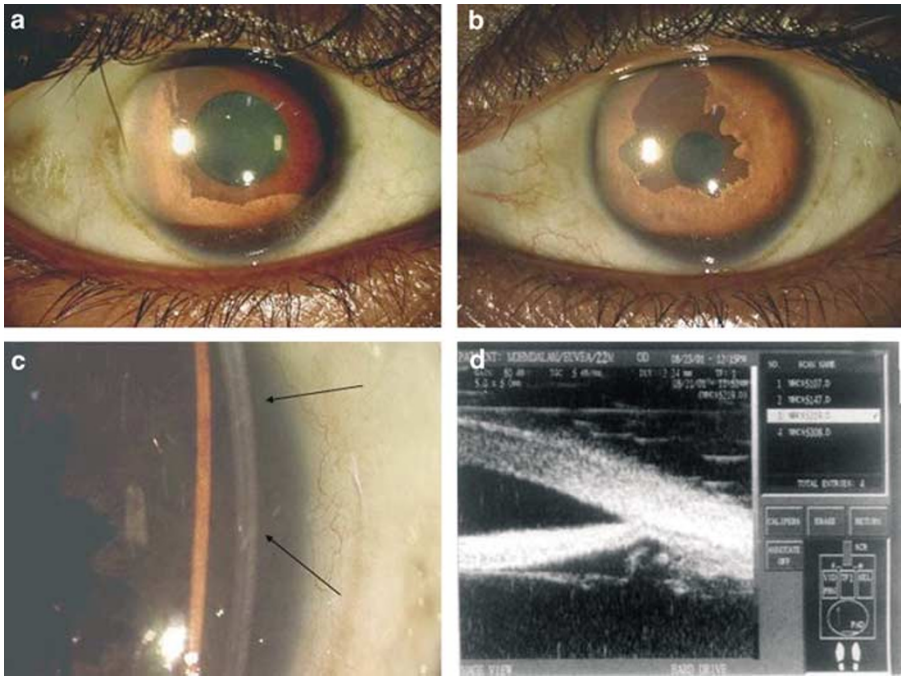

Figure 1 (a) Anterior segment photograph of right eye showing congenital ectropion uveae 360 degrees around the pupil and extending to the superonasal periphery. (b) Anterior segment photograph of left eye showing congenital ectropion uveae extending 360° around the pupil to the mid-periphery of the iris. (c) Anterior segment photograph of left eye (magnification × 25) showing greyish thickened corneal nerves in temporal quadrant (arrows). (d) Ultrasound biomicroscopy showing anterior insertion of iris root in left eye.