Sir,

Primary surgical management in a case of subtotal iridodialysis

Iridodialysis is the separation of the iris base from the ciliary body and the scleral spur. The iris root is the thinnest and weakest part of iris anatomy, making it vulnerable to ocular trauma.[1] We describe a case where traumatic iridodialysis was sustained resulting in secondary glaucoma. Surgical intervention involving an anterior chamber washout and removal of necrotic iris brought about control of the intraocular pressure.

Case report

A 66-year-old man sustained blunt trauma to his right eye whilst repairing his garage door. Visual acuity in this eye was hand movements. On slit-lamp examination an iridodialysis of 330° was present, as was a microscopic hyphaema (Fig. 1). There was a vitreous haemorrhage, with no fundal view. The lens was subluxed posteriorly and cataractous. The iris appeared to be necrotic. There was zonular dehiscence except inferiorly where the iris was still attached. The intraocular pressure was 40 mmHg. An ultrasound scan showed a flat retina. The patient was treated with topical steroids and beta-blockers, together with systemic acetazolamide and mannitol. None of this served to reduce the intraocular pressure, which fluctuated between 40 and 60 mmHg. A paracentesis was performed and the dialysed iris tissue removed. Due to significant damage to the lens zonule, the lens was removed using an intracapsular technique. An anterior vitrectomy was then performed. By the next day the intraocular pressure was 14 mmHg, and remained controlled 4 months later. By this time, most of the vitreous haemorrhage had cleared, and the patient was achieving 6/12 vision using a coloured aphakic contact lens.

Comment

Romen and Singer[2] described a case of persistent secondary glaucoma despite good visual outcome following traumatic iridodialysis. Possible mechanisms for the development of glaucoma include angle recession, trabeculitis, trabecular damage, or debris in the anterior chamber. Surgical techniques have been formulated to enable repair of even a large iridodialysis, but with viable iris tissue.[3]

We suggest that the cause of the pressure rise in this case was inflammation from necrotic tissue in the anterior chamber. The rapid resolution of the glaucoma precludes any significant trabecular trauma. Lenticular damage may have been a contributory factor, but the lens was not obstructing the drainage angle.

In cases of subtotal iridodialysis, where the iris tissue is unlikely to be viable, we suggest early removal of necrotic tissue to avoid secondary glaucoma.

If removal of the iris is inevitable, problems of both cosmesis and glare result. Several strategies have been employed to overcome these problems. Implantation of an iris diaphragm has been described, and so has corneal tattooing.[4,5] In cases where a cataract has developed and the lens zonule is stable, frosted intraocular lenses have been implanted with success.[6]

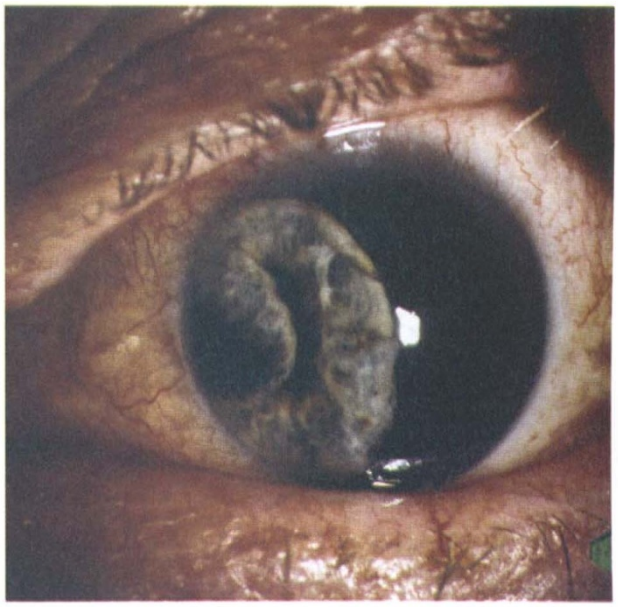

Fig. 1. *Colour photograph showing subtotal iridodialysis.*

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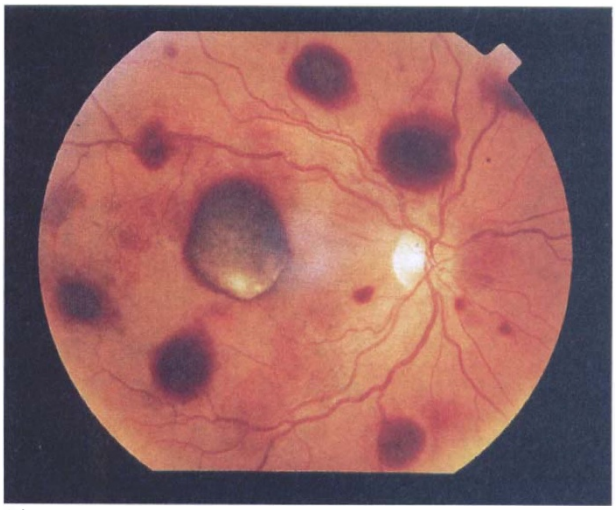
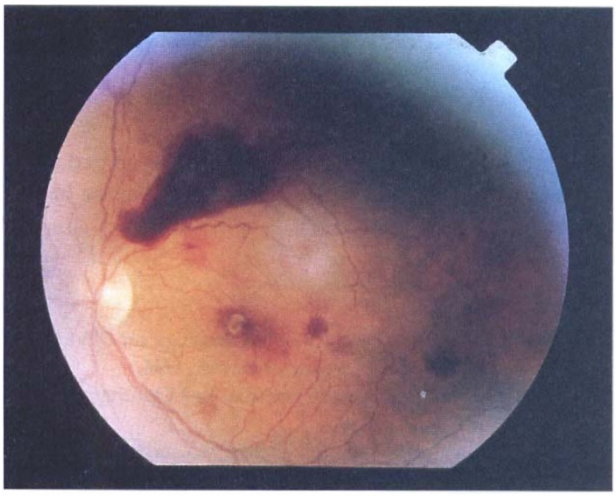
Sir,

Treatment of preretinal Valsalva haemorrhages with neodymium:YAG laser

Valsalva retinopathy is caused by a sudden increase in intrathoracic or intra-abdominal pressure against a closed glottis (Valsalva's manoeuvre) and is characterised by rupture of superficial retinal capillaries



(a)



(b)

Fig. 1. *Retinal haemorrhages in the right (a) and left (b) eyes at presentation.*

classically causing 'cannonball-shaped' haemorrhages.[1] Valsalva retinopathy is one cause of a sudden subhyaloid haemorrhage; however, more common causes include proliferative diabetic retinopathy and a retinal artery macroaneurysm. These haemorrhages usually resolve over several months,[2] but some reports have cited the use of laser to aid resolution in cases of diabetic and macroaneurysmal preretinal haemorrhage.[3–5]

Case report

A 44-year-old Caucasian woman was referred for an ophthalmic assessment due to decreased visual acuity in both eyes. This patient had been diagnosed with papillary carcinoma of the thyroid and 7 weeks earlier had had a total thyroidectomy and left functional neck dissection. She appeared to make a satisfactory recovery; however, 5 days later she suffered a respiratory arrest. She was resuscitated and thrombolysed with intravenous streptokinase for a suspected pulmonary embolus, before being transferred to intensive care, where she was ventilated by continuous positive pressure ventilation (CPPV). She remained rather hypotensive with electrocardiographic ischaemic changes, and developed

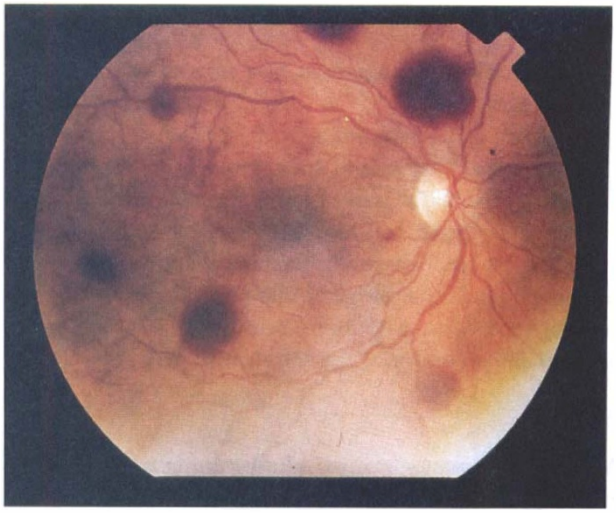


Fig. 2. *One week after Nd:YAG laser treatment the right foveolar haemorrhage has completely cleared.*

renal failure, for which she received haemofiltration. After 3 weeks of intensive care she was allowed to self-ventilate. Once able to communicate she complained of blurred vision in both eyes.

Her best corrected visual acuity was counting fingers in the right eye and 6/36 in the left eye, with no relative afferent pupillary defect. Visual field tests showed a central visual field defect in both eyes. Examination of the anterior segment was unremarkable; however, the fundus examination revealed numerous retinal haemorrhages in both eyes (Fig. 1), some flame-shaped in the nerve fibre layer, and several large preretinal round 'cannonball' haemorrhages. One of the latter covered the foveola in the right eye accounting for the poor visual acuity. The visual acuity in the left eye was decreased due to several small intraretinal foveal haemorrhages.

The right foveolar haemorrhage was treated with an Ocular Instruments Q-switched Nd:YAG laser (five pulses of 3 mJ) using an OMRA-HM Mainster High Magnification laser lens. The laser was aimed at the inferior edge of the haemorrhage away from the foveola. The haemorrhage immediately began to disperse and had completely cleared 1 week later (Fig. 2). The patient's visual acuity improved to 6/12 in the right eye and 6/36 in the left eye.

Comment

Valsalva retinopathy in this case most likely occurred during the prolonged resuscitation after respiratory arrest or alternatively during episodes of coughing or straining whilst being ventilated. The problem may have been exacerbated by thrombolysis and anticoagulation. Spontaneous clearing of the premacular haemorrhage usually occurs but may take several months. In view of this patient having reduced vision in both eyes and because of her poor life expectancy, we elected to perform Nd:YAG laser puncture of the posterior hyaloid face on the premacular right fundal haemorrhage. This cleared the prefoveal haemorrhage providing rapid visual recovery. Ophthalmoscopy and fluorescein

angiography (acknowledged to be the most sensitive test for laser-induced retinal damage[6]) confirmed that no iatrogenic damage had occurred. Although this treatment has had some success with diabetic and macroaneurysmal preretinal haemorrhages, to our knowledge it has not been used before in treating Valsalva preretinal haemorrhages.

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Sir,

Spontaneous acute scleritis and scleral necrosis in choroidal malignant melanoma

Necrosis of choroidal malignant melanoma is not unusual and may rarely be accompanied by necrosis of intraocular tissues.[1] However, the sclera is relatively resistant to necrosis and acute scleritis with necrosis, secondary to choroidal malignant melanoma, has not been reported to the best of our knowledge.

Case report

A 58-year-old Caucasian man presented with loss of vision in the right eye of 3 weeks' duration. The visual acuity in the right eye was perception of light. Examination revealed a subretinal pigmented mass elevating the retina and normal intraocular pressure. A clinical diagnosis of choroidal malignant melanoma was made.

Three weeks later he developed a painful red eye with vitreous haemorrhage, hyphaema and an elevated intraocular pressure of 35 mmHg. The ultrasonogram

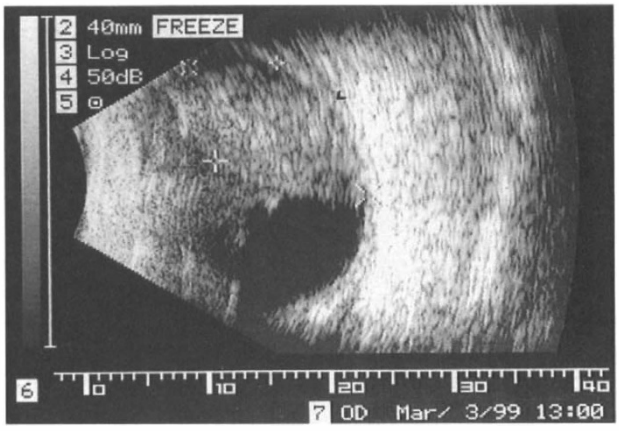

Fig. 1. *Ultrasonogram showing scleral thickening overlying the tumour mass (arrowhead).*

showed an 8 × 16 mm choroidal mass with features suggestive of melanoma (Fig. 1). The sclera overlying the tumour mass was thickened, but there was no evidence of extrascleral extension (Fig. 1). In view of the tumour mass and intractable pain, the eye was enucleated. There was no evidence of systemic metastasis. There was no history or clinical evidence of systemic collagen vascular diseases. Serological screening for connective tissue diseases including rheumatoid arthritis was negative.

Pathological findings. Macroscopic examination revealed a firm globe measuring 22 mm in diameter. A light-brown tumour, 14 × 5 mm in cross-section, was present in the inferior part of the posterior segment. There was no extrascleral extension (Figs. 2, 3). Microscopic examination revealed a choroidal malignant melanoma, which was almost entirely necrotic. Immunohistochemistry revealed expression of neurone-specific enolase within the tumour and focal expression of HMB45 and S100, particularly in the better-preserved cells around the edge of the tumour. The tumour did not demonstrate a lymphocytic response. Both the anterior and posterior segments of the eye were filled with a proteinaceous exudate in which fresh haemorrhage was seen (Fig. 2). The retina was totally detached and showed reactive microcystic changes and gliosis.

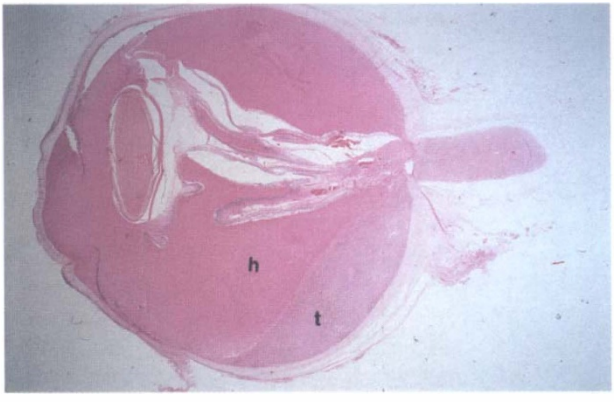

Fig. 2. *Plan view of the globe demonstrating a choroidal melanoma. The detached retina is seen running anteroposteriorly and there is a proteinaceous exudate and fresh haemorrhage. h, Haemorrhage; t, tumour. (Original magnification* ×8)