different ages, with a central focus of methemoglobin, a peripheral rim of hemosiderin, an adjacent focus of acute or subacute haemorrhage and minimal or no enhancement on gadolinium administration.[7] The patient in the present report had MRA on two occasions, both of which were reported to be normal. Angiography is typically silent with these lesions. This may be because the small caliber of feeding arteries, and their slow circulation, causes dilution of contrast medium, or extensive thrombosis or both.[1]

In other cases, MRI may suggest a meningioma or a venous anomaly as in the present case. Other possibilities include a haematoma, calcified glioma or a venous angioma.[1] Unlike similar lesions in the orbit, intracranial angiomas have no true capsule, although compression from an expanding lesion produces a gliotic pseudocapsule. Microscopically their walls are composed of collagen with tongues of fibrillary neuroglia which penetrate the lesion.[1]

Cavenous haemangiomas may have an autosomal dominant inheritance. Not all lesions require surgery. Those presenting with seizures and minimal or stable visual loss can be observed. Surgery is advocated for those with severe or progressive visual loss.

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Sir,

Central retinal vein occlusion associated with Sturge Weber syndrome

Eye (2002) **16,** 657–659. doi:10.1038/sj.eye.6700120

Central retinal vein occlusion associated with Sturge Weber syndrome is rare, to our knowledge there are only two other reported cases in the literature.[1,2] We report such a case.

Case report

A 22-year-old male patient with left-sided Sturge Weber syndrome and well-controlled ipsilateral glaucoma, presented to the eye casualty department with a left central retinal vein occlusion (Figure 1). The visual acuity was 6/36 in the left eye and intraocular pressure 18 mmHg. The cornea was clear and there was no evidence of rubeosis iridis. A relative afferent pupilliary defect was noted prior to dilated fundoscopy, which revealed the typical features of a central retinal vein occlusion. Routine blood investigations, including a thrombophilia screen, were all normal, and antithrombin III and plasminogen levels were all within normal limits.

The patient suffering from Sturge Weber syndrome originally presented to the eye department at 9 years of age, with an elevated intraocular pressure of 34 mmHg in the left eye, the right was 20 mmHg. The visual acuity was 6/6 in each eye and the optic discs were healthy (Figure 2). The left corneal diameter was 2 mm greater than the right. A visual field test revealed a right homonymous hemianopia, which was attributed to Sturge Weber syndrome. He was known to be epileptic and controlled with sodium valproate and carbamazepine. There was no other significant medical history. A diagnosis of Sturge Weber

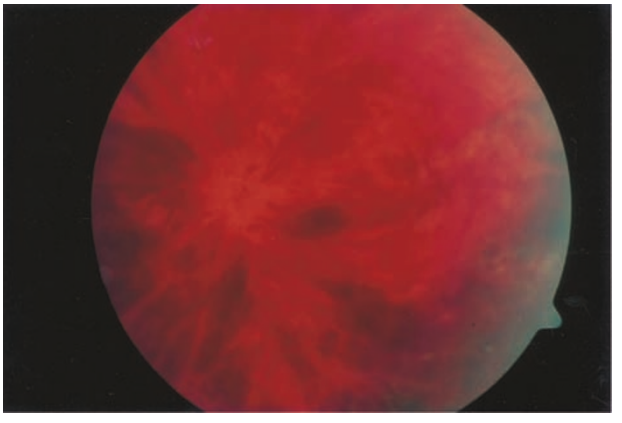

Figure 1 Central retinal vein occlusion in the left eye.

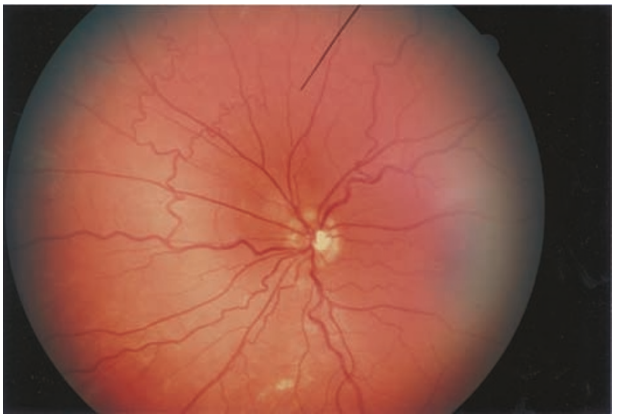

Figure 2 The left eye with a healthy optic disc and diffuse choroidal haemangioma.

syndrome-related glaucoma was made and treated with guttae timoptol 0.5%. Over the following 13 years, the intraocular pressure in the left eye remained well controlled.

Three weeks after presentation with the CRVO, he developed an acute painful red eye. Examination revealed an elevated intraocular pressure of 52 mmHg and rubeosis iridis. A diagnosis of neovascular glaucoma was made and the eye was treated with guttae pred forte 1% q.d.s., guttae atropine 1% b.d. and oral acetozolamide 250 mg q.d.s. Extensive panretinal photocoagulation (PRP) was performed. Unfortunately despite maximal treatment, the IOP remained elevated, which required cyclodiode laser treatment. Over the next 4 months, two further treatments of laser PRP were needed and cyclodiode laser treatment was repeated. At last review the eye was pain free with an IOP of 30 mmHg on guttae timoptol. The vision was counting fingers.

Discussion

Central retinal vein occlusion is a condition more commonly attributed to the elderly, when it is associated with arteriosclerosis, hypertension, hyperlipidaemia, hypercoagulability and uncontrolled glaucoma.[3] It is less common in patients under 50 years of age when in the vast majority of cases, there is no known association with systemic disease.[4] However, in the case of bilateral and recurrent CRVO, a systemic cause should be suspected, especially a hyperviscosity or hypercoagulation state, such as abnormal platelet function or anticoagulant protein deficiencies.[5,6] Local ocular conditions such as uncontrolled glaucoma and optic nerve drusen[4,7] should also be excluded in unilateral cases, as these conditions have been reported in association with CRVO.

The prognosis for young patients suffering from CRVO is generally good, with most cases resolving spontaneously over the following 6 months, however the final visual outcome, is related to the amount of retinal ischaemia. Neovascular glaucoma occurs in approximately 20% of patients and is treated with panretinal photocoagulation.[4]

A patient suffering from Sturge Weber syndrome (encephalofacial angiomatosis), typically suffers from the quartet, of port wine staining (naevus flammeus) of the face roughly in the distribution of the first, second or third divisions of the trigeminal nerve, ipsilateral intracranial haemangioma, choroidal haemangioma, and congenital glaucoma. Incomplete forms exist.[8]

We know of only two previous reports of CRVO in association with Sturge Weber syndrome, one of which describes a patient who also had disc drusen in addition to Sturge Weber syndrome.[1,2] Our patient had been maintained at an intraocular pressure, which, in this young patient, prevented the development of any optic nerve fibre damage. Despite this, the intraocular pressure may have been high enough to predispose to CRVO in an eye with the vascular anomalies of Sturge Weber syndrome.

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Sir,

The origin of posterior vitreal gas bubbles following the high velocity impact of a metallic fragment

Eye (2002) **16,** 659–660. doi:10.1038/sj.eye.6700154

Intraocular foreign bodies are a common and potentially devastating sequelae to the impact of high velocity solid objects upon the globe. Here we report the unusual finding of small gas bubbles closely related to the location of the foreign body, but well away from the site of entry. The possible origins of the bubbles are discussed in relation to the clinical picture.

Case report

A 25-year-old male was admitted having been hit in the right eye by an object dislodged whilst hammering a metal machine punch. The patient complained of right visual blurring and his initial Snellen visual acuities were 6/9 in the right eye and 6/5 in the left. Ocular examination revealed a 1.5 mm conjunctival laceration immediately temporal to the limbus, with surrounding subconjunctival haemorrhage obscuring view of the underlying sclera. There was a mild cellular response in the anterior chamber, and intraocular pressures were 11 and 19 mmHg in the right and left eyes respectively. Fundoscopy revealed a small area of pre-retinal haemorrhage overlying the infero-nasal vessels, with blood extending from this site antero-inferiorly into the vitreous (Figure 1). Of particular note were a number of bubbles lying adjacent to the retina in the posterior vitreous, and superior to the above retinal lesion. These bubbles were only present in this one location, and slowly reabsorbed over the next 48 h. Plain radiographs of the orbits suggested the presence of a radio-opaque foreign body within the right eye, and this was supported by CT scan. The patient was therefore commenced on oral ciprofloxacin 750 mg b.d. and two hourly Predforte drops, together with topical Chloramphenicol and Atropine. By day 4 a metallic foreign body could be observed within the clot overlying the area of

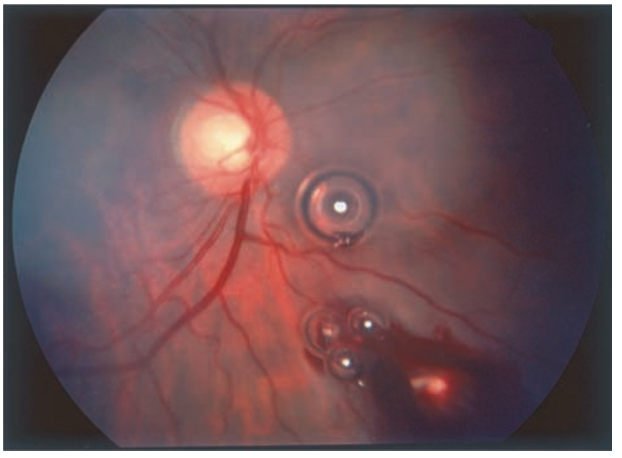

Figure 1 Fundus photograph taken on day 1 showing area of preretinal and intravitreal haemorrhage with associated gas bubbles.

preretinal haemorrhage, and this was subsequently removed during a pars plana vitrectomy (Figure 2). Although the postoperative recovery was complicated by further vitreous haemorrhage, the patient made a good visual recovery with an acuity of 6/9 when discharged 6 months later.

Comment

The presence of free gas bubbles within the vitreous following the passage of a high velocity fragment has not been previously reported to our knowledge, and it is interesting to speculate on the origins of the bubbles seen in this patient. Free gas bubbles associated with both *Clostridia* sp and *Bacillus cereus* panophthalmitis have been reported, but such cases involved extensive inflammation with necrosis throughout the globe.[1–3] In this case there was no evidence of vitritis and the bubbles were present within 1 h of injury, and it is therefore likely that the presence of the bubbles was

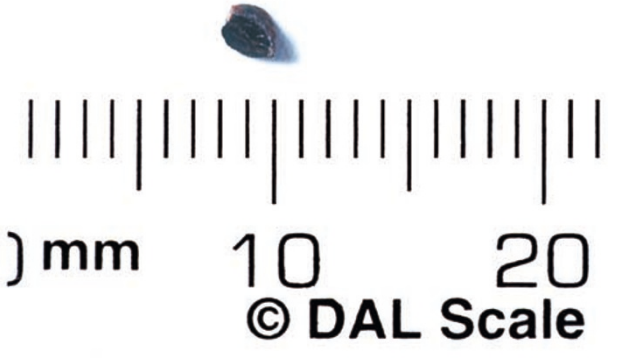

Figure 2 The metallic foreign body recovered from the patient.