cases of ipsilateral cerebral hemispheric involvement have been reported, common sites including the basofrontal region, posterior fossa and meninges.

Archer *et al.*[5] studied retinal arteriovenous malformations *per se* with fundus fluorescein angiography and ophthalmodynamometry to elucidate structural abnormalities, and grouped them according to the nature and extent of the arteriovenous communications.

A review of the literature also reveals that retinal arteriovenous malformations are capable of growth,[6] haemorrhage,[5] sclerosis[7] and thrombosis.[8,9] These changes can affect vision profoundly by causing optic atrophy, as can a compressive effect of the lesion.

Orbital manifestations, when they do occur, include enlargement of the optic foramen and, occasionally, proptosis, which may or may not become pulsatile.

Dermatological lesions can occur in a minority of cases and take the form of ipsilateral skin naevi, in the distribution of the trigeminal nerve.

Conclusion

Our young patient has Wyburn-Mason syndrome by virtue of ipsilateral retinal and orbital arteriovenous malformations. To our knowledge, such an association without any lesion in the brain has not been reported previously.

Although retinal arteriovenous malformations can sometimes be confused with inherited retinal arteriolar tortuousity or even congenital retinal macrovessels, in our case, the typical age of presentation and the unilateral involvement suggest otherwise.

Intervention, when contemplated, may comprise embolisation (frequently used to treat central nervous system arteriovenous malformations), laser photocoagulation or proton beam radiation, the latter being described in maxillary and mandibular lesions.

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Sir,

Primary acute angle closure glaucoma associated with suprachoroidal fluid in three Chinese patients

Separation of the choroid from overlying sclera may occur with hypotony, inflammation, neoplasia, panretinal laser photocoagulation,[1] nanophthalmos and uveal effusion syndrome.[2] Clinically detectable choroidal detachments associated with profound reductions in intraocular pressure are described after trabeculectomy, bleb needling or even topical treatment.[3–5]

Acute primary angle closure glaucoma (APACG) demands prompt, aggressive lowering of raised intraocular pressure to break the attack and minimise optic nerve damage and ischaemic sequelae. We describe three patients with suprachoroidal fluid collections following such treatment. As part of a larger prospective study we examined all patients with APACG attending the Singapore National Eye Centre over 18 months. Ultrasound biomicroscopy (UBM)[6] examinations (Humphrey Instruments, San Leandro, CA) were performed within 24 h of presentation, before peripheral laser iridotomy but after medical treatment.

Case reports

All three patients presented with a history of periorbital pain and blurred vision of between 1 day's and 2 weeks' duration. They all had raised intraocular pressure with signs of APACG and closed anterior chamber angles on gonioscopy (Table 1). All were female. Treatment with intravenous and oral acetazolamide, mannitol, topical pilocarpine, timolol and steroids successfully broke the attacks.

UBM demonstrated suprachoroidal fluid collections in all three cases (Fig. 1), the maximum depths of which are shown in Table 1. Patient 1 had the largest collection, extending over the full 360° circumference of the globe and posteriorly beyond the equator, whereas those in patients 2 and 3 were shallower and did not reach to the equator. All uninvolved eyes had narrow but open angles and no abnormality on UBM. All three fluid collections resolved within 5 days.

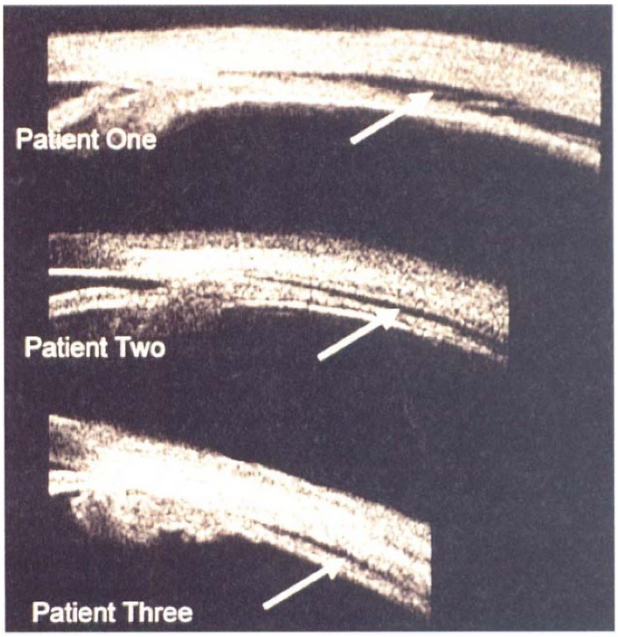

Fig. 1. *Composite ultrasound biomicroscopy images showing suprachoroidal fluid collections (arrowed).*

No eye was nanophthalmic on A-scan ultrasound examination (Table 1). Fundus examination revealed a markedly cupped disc (CD ratio 0.9) in patient 1 and signs of an old branch retinal vein occlusion (BRVO) in patient 2.

Visual acuity at 6 months was poor in patient 2, possibly due to the old BRVO. Visual field examination showed profound glaucomatous field loss in patient 1 (presumably due to previous underlying chronic PACG) and field loss consistent with an old inferior BRVO in patient 2. There were no long-term effects on vision attributable to the suprachoroidal fluid.

Comment

Separation of the choroid from overlying sclera may occur when fluid accumulates in the suprachoroidal space at a greater rate than it can drain via the choroidal or trans-scleral routes, probably as a transudate from choroidal capillaries.[2] In accordance with Starling's law this may arise from increased vessel permeability (inflammation, neoplasia), increased intravascular pressure and uveal congestion (scleral buckling procedures, nanophthalmos, uveal effusion syndrome, caroticocavernous fistulae[2,6,7] or from decreased intraocular pressure that reduces the hydrostatic pressure gradient, thus reducing outflow. It is likely that there is a similar underlying mechanism of reduced hydrostatic pressure gradient across choroidal capillaries.

This is the first instance in which the development of suprachoroidal fluid has been seen as a consequence of APACG or its treatment. Other causes of a rapid and profound reduction in intraocular pressure, such as trabeculectomy, are associated with such collections but usually as a result of hypotony.[3–5,8] It also is possible that the markedly raised intraocular pressures (mean 40.3 mmHg) led to ciliary body and/or choroidal ischaemia and a secondary inflammatory response with alteration in capillary permeability. It is of interest that the eyes with higher initial intraocular pressures had larger fluid collections. The normotensive 'fellow' eyes showed no sign of suprachoroidal fluid and had only a modest drop in IOP from normal pressures due to the systemic treatment (mean 13.6–10.6 mmHg). This suggests either that it requires a larger absolute fall in IOP to alter the fluid dynamics sufficiently to cause a suprachoroidal fluid collection or that an additional (possibly ischaemic) mechanism is involved.

It cannot be entirely excluded that suprachoroidal fluid existed before the acute attack. Previous reports have described acute angle closure *secondary* to choroidal effusion or cilio-choroidal detachment.[9–12] For example, Quinlan and Hitchings[12] report a series of three patients in whom choroidal detachments due to posterior scleritis resulted in anterior rotation of the ciliary body and secondary acute angle closure glaucoma. In contrast to reported cases the acute attacks in our patients resolved with standard miotic and hypotensive therapy and had no evidence for coexisting pathology (minimum 6 months follow-up). There was no significant anterior rotation of the ciliary body on UBM to cause secondary angle closure (as seen by Liebmann and Pavlin) and the fluid collections are far smaller than previously described. It is not possible to rule out that these are cases of secondary ACG occurring as the result of small, transient, idiopathic effusions in susceptible eyes which had partially resolved at the time of the examination.

Several factors may explain the absence of previous descriptions: APACG patients receive pilocarpine, preventing peripheral fundal examination until peripheral iridotomy has been performed – by which time small fluid collections may have resolved. UBM identifies shallow collections in the absence of clinically detactable choroidal detachment[8] and these are uncommon, seen in only 3 of 75 cases (4%).

In summary, APACG may be complicated by secondary suprachoroidal fluid collections, probably as a result of rapid decompression with aggressive hypotensive therapy.

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Table 1. *Patient details*

Patient no.	Age (years)	Presenting IOP (mmHg)	IOP after treatment (mmHg)	Axial length (mm)	Depth of fluid collection (maximum, mm)	Time to resolution (days)
1	62	70	8	22.37	0.40	4
2	71	40	10	21.19	0.25	4
3	57	30	14	21.28	0.14	2

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