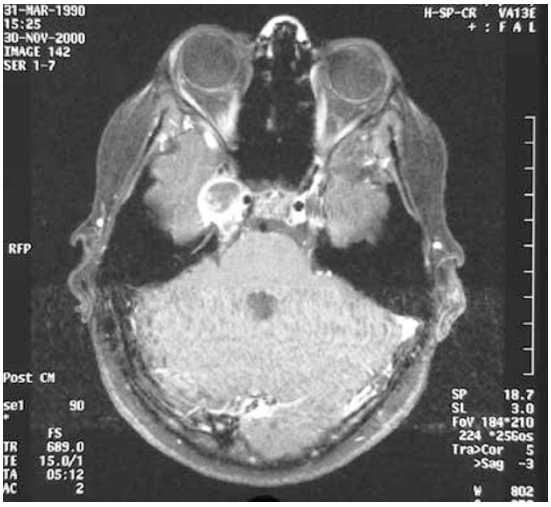

Figure 2 Gadolinium-enhanced axial T1-weighted MRI shows a mass at the right petrous apex extending forward into Meckel's cave and the cavernous sinus with intense inhomogeneous peripheral ring enhancement.

Ocular motility had not been restricted. A plexiform schwannoma of the thorax had also been excised.

On ophthalmologic examination, visual acuity measured 20/50 in the right and 20/25 in the left eye. Ocular motility testing showed extensive limitation of right abduction. Sensory function of the right trigeminal nerve was markedly diminished.

At 2 weeks after a CT scan of the head—that notably had been reported as unremarkable—a subsequent MRI detected an extraaxial mass at the right petrous apex extending forward into Meckel's cave and the cavernous sinus (Figure 2). This tumour had originated from the right trigeminal nerve and was subtotally removed in a one-stage procedure. Histological examination confirmed the diagnosis of schwannoma. Concurrence of juvenile cataract and schwannomas led to the suspicion of NF-2. Genetic testing revealed a nonsense-mutation of the NF-2 gene.

Comment

Further refinement of the diagnostic systems[2] for NF-2 in both children and adults is of great importance for early detection of this debilitating disease. NF-2 should be included in the differential diagnosis in patients with isolated schwannomas comprising a close look for further features typical of NF-2 and the use of genetic testing as a diagnostic tool.

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Sir,

Cataract surgery using phacoemulsification may reactivate angiogenic growth factors in ocular ischaemic syndrome

Neovascularisation is a recognised complication of intraocular surgery performed during retinal ischaemia.[1] We describe its occurrence in a patient with severe carotid artery atherosclerosis and ocular ischaemic syndrome who underwent phacoemulsification and intraocular lens implantation. To our knowledge this phenomenon has not been previously reported in ocular ischaemic syndrome.

Case

A 65-year-old man with bilateral ocular ischaemic syndrome due to carotid atherosclerosis underwent left cataract surgery.[2–5] Vision was 2/60 in the left eye due to

posterior subcapsular lens opacity. Vision in the right eye was no perception of light due to extensive rubeosis refractory to several previous laser treatments.[4]

Past medical history included severe carotid occlusive disease considered unsuitable by the vascular surgeons for endarterectomy, and which had rendered both eyes rubeotic.[6] Duplex scanning 18 months before surgery had shown reverse flow of normal velocity in both ophthalmic arteries.[7] Panretinal photocoagulation (PRP) had been undertaken by an ophthalmic surgeon to the left eye 3 years previously, with additional fill-in laser administered up to 2 years prior to cataract surgery, ensuring regression of neovascularisation in the left eye by 10 months before phacoemulsification.[4] Figure 1 displays the fundus fluorescein angiogram (FFA) of the patient's left eye following PRP, showing a grossly ischaemic fundus due to total occlusion of the ipsilateral carotid artery. There was no history of diabetes.

Left eye phacoemulsification and intraocular lens implantation was technically uncomplicated, used a superior clear corneal incision, and utilised routine settings for phacopower, vacuum, and aspiration. Postoperative fundal examination on the day of surgery revealed old PRP burns with no evidence of visible new vessels. However, despite a steady improvement in vision to 6/9 corrected at 3 weeks following surgery, surprisingly, small new vessels on the optic disc were noticed. Vision further improved to 6/6 with correction during the course of the next month. There was no change in visual field, evidence of optic neuropathy or anterior ischaemic optic neuropathy which might have produced pseudoneovascularisation.

Close follow-up was implemented, and after assessment of systemic risk factors, conservative treatment was continued owing to absence of haemorrhage, intraocular inflammation or proliferation of new vessels beyond the disc. At 1 year following surgery only tiny stumps of new vessels could be seen at the disc. Throughout this period new vessels had been confined to the disc, with no evidence of involvement of the retina itself or the iris. Left vision has remained 6/6 with correction for the entire period following the initial postoperative phase.

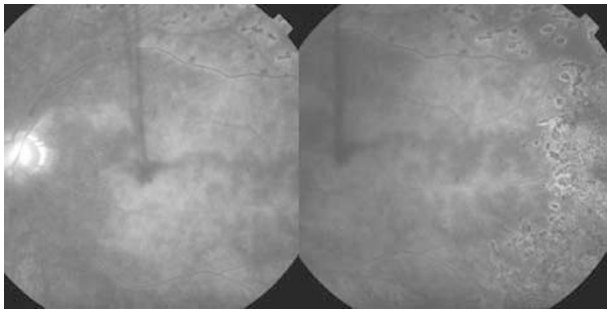

Figure 1 FFA showing the grossly ischaemic fundus with no arterial inflow into the retina as well as laser-induced scars due to PRP.

Discussion

Following intraocular surgery, reactivation and exacerbation of neovascularisation has been described with other ischaemic retinal conditions. This has been highlighted most often with diabetic retinopathy.[1] Cataract surgery has also been reported to exacerbate subretinal neovascularisation in age-related macular degeneration.[8] The phenomenon has hitherto not been reported with ocular ischaemic syndrome. This may be owing to the rarity of cases as many patients with ocular ischaemic syndrome do not undergo cataract surgery owing to a poor visual prognosis in many of them, and as the condition is not in itself common.[2–5] However, in this patient early effective treatment with laser therapy in the years prior to cataract surgery had left the patient with good vision.

Generally, ocular sequelae of carotid artery ischaemia such as slow flow retinopathy can be reduced or even reversed with effective joint management by a team of ophthalmic and general vascular surgeons, provided each has experience in and is aware of the interaction between ocular and carotid pathologies.[4,9] However, progression to ocular ischaemic syndrome tends to carry a very guarded prognosis.[2–5] Local treatment with PRP can induce regression of rubeosis iridis—nevertheless, visual prognosis is unpredictable, and while it is poor for most patients, as this case shows a good visual outcome is still feasible through effective use of PRP by ophthalmologists.[5] Carotid endartectomy by a specialist in vascular surgery can help stabilise ocular ischaemic syndrome and improve visual outcome.[4,6] Superficial temporal to middle cerebral artery bypass has also been performed by vascular surgeons and/or neurosurgeons, who have claimed some benefit in ocular ischaemic syndrome.[7] Other issues of relevance include, during ophthalmic surgery, the use of a clear corneal incision anterior to the superior rectus muscle to remain in avascular corneal tissue. This incision is less likely to disrupt the fragile blood supply to the eye.

There are six important features in this case that make it plausible that the neovascularisation was indeed related to the surgery. First, there was no progression of neovascularisation in the affected eye for 2 years following the cessation of argon laser therapy to the retina, suggesting stable ocular ischaemia prior to surgery. Second, treatment had caused neovascularisation to resolve by 10 months before cataract surgery, which also suggests that the status of

ocular ischaemia had been stable preoperatively. Third, absence of any ocular inflammation for 2 years before surgery also suggests stable ocular ischaemia prior to surgery. Fourth, laser photocoagulation on FFA (Figure 1) which was adequate enough to cause regression of new vessels clinically. Fifth, a plausible temporal association between neovascularisation and cataract surgery, owing to new vessels becoming visible on fundoscopy 3 weeks following cataract surgery. Sixth, biological plausability on the basis of occurrence of reactivation and exacerbation of neovascularisation following surgery in other ischaemic retinal conditions.

However, there is an atypical feature in this case in the spontaneous regression of the new vessels. For this reason we considered the possibility that the new vessels on the disc could be a new form of 'pseudoneovascularization', a condition that has been described in diabetic patients with anterior ischaemic optic neuropathy where 'due to disc oedema, the disc is covered with a network of prominent fine vessels, mimicking neovascularization and thus leading to erroneous diagnosis of proliferative retinopathy.'[10] However, the patient had neither diabetes nor anterior ischaemic optic neuropathy, the new vessels were not as fine as in pseudoneovascularisation, and most importantly in this context there had been no disc oedema, thus excluding disc swelling as a cause of obscuration of the optic disc vessels. Interestingly, spontaneous regression of neovascularisation has been reported in retinal ischaemia due to diabetes.[11] No explanation for the latter phenomenon has been established.[11] However, spontaneous resolution of new vessels in diabetic retinal ischaemia has thus far not been linked to surgery.[11]

Following phacoemulsification, breakdown of the blood–retinal barrier is more likely in patients such as ours who received over 1 J of energy from the phacoprobe.[12] Levels of growth factors are also affected by phacoemulsification. In diabetics phacoemulsification is associated with increased aqueous levels of VEGF, IL-6, and protein.[13] In ocular ischaemic syndrome due to carotid artery atherosclerosis the retina shows immunoreactivity for inducible nitric oxide synthase, which is also implicated in cerebral ischaemic damage, diabetic vasculopathy and angiogenesis.[14] Study of morphology as well as immunoreactivity for glial fibrillary acidic protein and vimentin suggest that nitric oxide synthase induced by retinal ischaemia localises to the Muller glial cells.[14] High concentrations of nitric oxide produced by inducible nitric oxide synthase could contribute to both microvascular remodelling during angiogenesis as well as neurotoxicity in both ocular ischaemic syndrome and diabetic retinopathy.

This case suggests a possible association between reactivation of neovascularisation and phacoemulsification in ocular ischaemic syndrome due to carotid atherosclerosis. As this has not previously been reported, further studies are needed to assess the feasibility of a large series, and the strength of any association to be assessed and clarified. This subject merits further attention.

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Sir,

Ocular perforation during peribular injection
Reply

I read with concern a case report by Gauba *et al*[1] describing a case of ocular perforation and intra vitreal injection of depomedrone during peribulbar injection.

This is a most unfortunate and frightening complication of a routine injection.[2]

However, it should be appreciated that such complications will become more common at the hands of ophthalmologists who are rapidly becoming deskilled in the valuable art of making periocular injections.

Until a few years ago the retrobulbar/epibulbar/peribulbar injections to effect akinesia and anaesthesia for ocular surgery were made by the ophthalmologists. This practice has now been passed on to anaesthetists/nurse practitioners. As a direct consequence of this the ophthalmologists have lost an opportunity to develop expertise in making such injections. The ophthalmologists are now required to make such injections on rare occasions as in the case described by Gauba *et al*.[1]

The present training programmes do not give ophthalmologists ample opportunities to practice and develop this most useful skill of making such injections. It should therefore come as no surprise that ophthalmologists have lost the ability to appreciate whether the needle is in the vicinity of the globe or is inside it. Ophthalmologists should make some serious attempt to reclaim the art of retro/peri/epibulbar injections to minimise and eliminate such unfortunate complications.

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Sir,

The author replies

The correct and safe technique of peribulbar/retrobulbar injection, as alluded to in the original article, should indeed be emphasized in the training of junior ophthalmologists. However, in the current environment of blunt injection techniques, most periocular injections, including steroid administration, can be performed safely and efficaciously by a subtenon approach.[1]

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