deepening of the anterior chamber and an improvement in outflow facility.[4] Chronic post-operative uveitis may lead to hypotony.[5] However, in our patient the reduction in IOP following phacoemulsification was more profound than described in previous studies. In addition, hypotony did not develop until 6 weeks after the operation. There was no significant anterior chamber activity to suggest chronic uveitis. Withdrawal of latanoprost led to complete resolution of choroidal detachment and hypotony. It therefore appears likely that latanoprost had a contributory role in the profound hypotony in this patient, possibly working synergistically with the post-phacoemulsification effect in lowering the IOP.

Latanoprost is known to work by increasing the uveoscleral outflow[1] and its pressure-lowering effect does not rely on the episcleral pressure of the eye. It can exert its pressure-lowering effect even when the IOP is lower than the episcleral pressure. Our patient was known to respond very well to latanoprost, which reduced the IOP by 14 mmHg (or over 50%) from baseline. The increase in outflow facility following phacoemulsification may have been enhanced by the pharmacological effect of latanoprost,[6] resulting in the profound hypotony experienced by our patient in the immediate post-operative period. Although Scherer *et al.*[6] described latanoprost as a safe and effective method for reducing the IOP following cataract extraction, our case suggests that clinicians should be aware of the potential complication in glaucoma patients, particularly if there has been a previous history of good response to latanoprost.

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Sir,

Neovascularisation in a patent chorioretinal anastomosis

Laser-induced chorioretinal venous anastomosis (CRA) has been advocated as a possible treatment option in patients with non-ischaemic central retinal vein occlusions (CRVO).[1] However, problems have arisen with regard to the formation of the anastomosis and with the development of complications. We describe a patient who developed neovascularisation directly from a patent anastomosis, several months after the procedure was performed.

Case report

A 57-year-old non-insulin-dependent diabetic presented with an acute onset of blurring of vision in his left eye. He had previously been followed up in the diabetic clinic and his minimal diabetic retinopathy had been stable for the last 9 years. His visual acuity had dropped in the left eye from 6/5 to 6/36 and in the right eye was 6/5. There was no afferent pupillary defect or iris neovascularisation of the left eye. Gonioscopy showed no angle neovascularisation. Fundal examination showed evidence of significant macular oedema associated with occlusion of the central retinal vein.

Fundus fluorescein angiography confirmed the non-ischaemic nature of the vein occlusion and 4 weeks after initial presentation he underwent laser-induced chorioretinal anastomosis. Argon laser (green) set to 50 μm spot size, 0.1 s duration, 3 W power was used, placing an anastomosis inferonasally. Two anastomoses were made since success of the first could not be determined; there were no early complications from the anastomosis formation.

Six weeks after the anastomosis his vision had improved to 6/12 with a marked reduction in the macular oedema. Functionality of the anastomosis was determined by rapid sequence fundus fluorescein angiography that showed evidence of trilaminar venous flow.

His vision remained stable at 6/12 and he was followed up at regular intervals. Ten months after the anastomosis was made he developed neovascularisation from the anastomosis site (Fig. 1). Functionality of the anastomosis both clinically and by fluorescein angiography indicated that it was patent (Fig. 2).

He underwent sectoral retinal photocoagulation using the argon (green) laser. The new vessels regressed successfully and up to 1 year follow-up he maintains 6/12 vision and has not experienced any further complications.

Comment

The occlusion of the central vein by an intraluminal venous thrombosis may produce an ischaemic or non-ischaemic CRVO. Patients with non-ischaemic CRVO associated with poor visual acuity and significant macular oedema are more at risk of progression to the

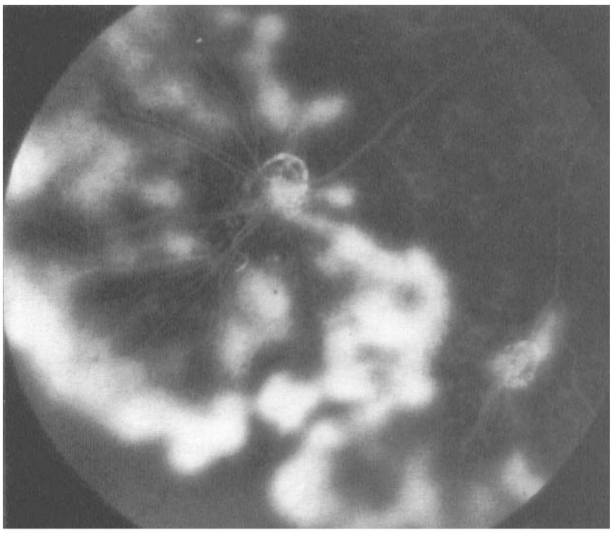

Fig. 1. *Prominent neonvascularisation from the anastomosis site.*

ischaemic type.[2] The median time for this transition to occur is 4.5 months.[3] The aim of the anastomosis is to decompress the retinal venous circulation and hence instigate an improvement in the visual acuity by reducing the macular oedema.[5] This may also reduce the rate of conversion to the ischaemic variant.[5]

The site chosen for the anastomosis is usually inferiorly, at least three disc diameters away from the disc.[1] However, if anatomical variants of the venous circulation are present then separate anastomotic sites will be needed to ensure adequate venous decompression. Argon laser with 50 μm spot size, 0.1 s duration and power of 1.5–2.5 W was initially advocated to disrupt the wall of the vein and rupture the underlying Bruch's membrane[1] at the same time. However, due to success rates of only 33%, a power of 2.5–3 W with the argon laser to rupture Bruch's membrane and a second spot for the retinal vein is recommended.[4] Alternatively the more difficult retinal vein may be ruptured successfully with the Nd:YAG laser (3–5 mJ).[4]

Immediate complications, i.e. haemorrhages of the retinal vein, vitreous and choroid, can be controlled by pressure from the contact lens, resolve by 2 weeks,[1] but may persist as the laser site may rebleed. Late complications include preretinal and subretinal fibrosis,[5] distal vein closure,[1,5] proximal segment narrowing,[6] choroidal neovascular membrane formation[7] and retinal neovascularisation.[8]

This patient developed retinal new vessels in the presence of a functioning chorioretinal anastomosis. Previous cases of reported retinal neovascularisation have occurred in patients with non-functioning CRAs or where only a segment of the retina was drained by the anastomosis.[8] Distal closure of the vein is a risk factor for neovascularisation and is thought to occur within the first 8 weeks of the laser application.[5] Since an anastomosis takes on average 7 weeks to become functional, distal closure that occurs in the 4–6 week[6] period after the laser treatment may become stabilised after the anastomosis becomes functional.

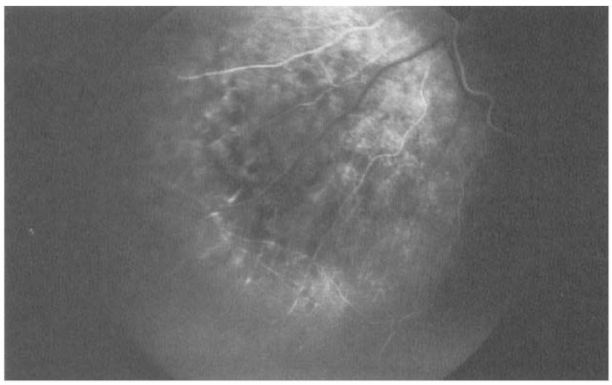

Fig. 2. *Preferential filling of the anastomosis in the early venous phase of fluorescein angiography, indicating functionality.*

The authors believe that early closure of the vein was initially stabilised by the anastomosis but latent closure triggered more ischaemia and the neovascularisation in this case. We could not completely rule out a secondary ischaemic mechanism driven by the patient's underlying diabetic retinopathy, even though this had been stable for some time. The formation of neovascularisation so long after the creation of the anastomosis indicates a further complication of this procedure and the need for careful consideration of this treatment in diabetic patients.

As complications are potentially sight-threatening, formation of a CRA should only be attempted in patients in whom it is felt a clear benefit is expected. Suitable patients should be defined as those in whom an improvement in vision could be expected from a reduction in macular oedema or those who demonstrated a progressive reduction in visual acuity in the milieu of a non-ischaemic CRVO.[1] The presence and the need for treatment of complications emphasise the need for careful follow-up of these patients after laser surgery.

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Sir,

Partial facial paralysis following temporal artery biopsy

A superficial temporal artery (STA) biopsy is often required to confirm the diagnosis of giant cell arteritis (GCA) prior to committing the patient to a long course of corticosteroid therapy.[1] It is considered a benign procedure with a low complication rate. However, complications can occur as in any surgical procedure, including infection, bleeding, haematoma formation, wound dehiscence and scarring. Recently, we encountered a patient who developed a partial facial paralysis manifested by brow ptosis following a STA biopsy.

Case report

A 63-year-old woman was referred to the neuro-ophthalmology service for evaluation of possible ocular involvement due to GCA although she was visually asymptomatic. Her past medical history was notable for hypothyroidism, colon cancer, rheumatoid arthritis and atrial fibrillation.

On examination visual acuity was 20/20 in both eyes. Pupils were equal with no relative afferent pupillary defect. Extraocular movements were normal. Slit-lamp biomicroscopy and intraocular pressures were normal. The optic nerves were normal OU. External examination revealed subtle left-sided brow ptosis at rest (Fig. 1, left), an inability to raise the left eyebrow with contraction of the frontalis muscle (Fig. 1, middle) and normal orbicularis oculi function bilaterally (Fig. 1, right). No further tests were recommended.

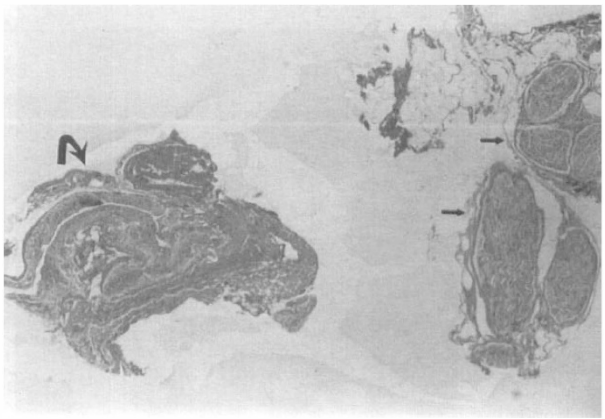

Fig. 2. *Low-power view (×13.2) of the pathological specimen demonstrating a small piece of artery (curved arrow) with predominantly nerve (arrows).*

One month prior to our evaluation, at the discretion of her primary physician, a general surgeon was consulted for a STA biopsy because of an elevated Westergren erythrocyte sedimentation rate (59 mm/h), left temporal headache and pain on mastication. Intraoperative reports indicate there was an estimated 200 cm^3 of blood loss. Prior to surgery an arterial pulse was identified with the aid of a vascular ultrasound in the left temporal scalp region. After an unsuccessful first attempt with dissection that penetrated the deep facial layers, a second separate incision was made anteriorly, and after further dissection a small arteriolar branch was identified and collected along with a 'second specimen around it'. The pathological specimen from the STA biopsy demonstrated a small segment of artery with no signs of inflammation and normal facial nerve (Fig. 2).

A short trial of oral corticosteroids was unsuccessful in resolving her symptoms and eventually the headaches improved spontaneously.

Comment

GCA is a systemic vasculitis with involvement of medium-sized and large vessels. The only definitive diagnostic modality is the pathological demonstration of

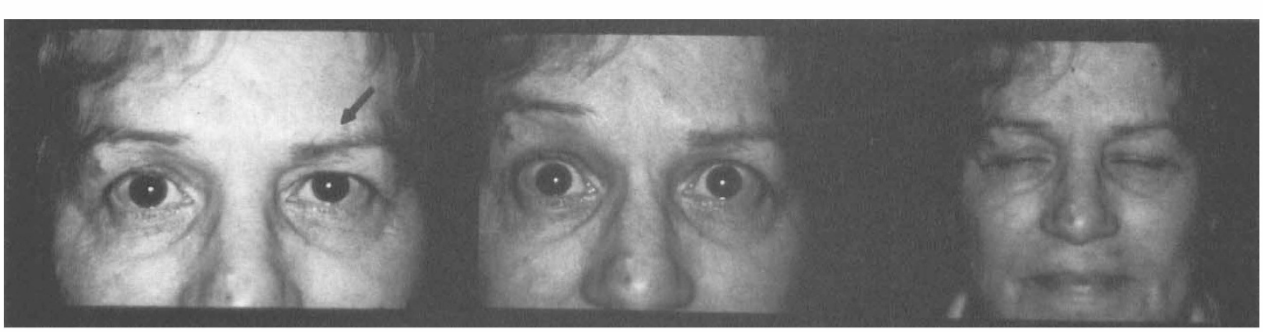

Fig. 1. *Left: Subtle left-side brow ptosis at rest (arrow). Middle: Note the inability to raise the left eyebrow with contraction of the frontalis muscle, demonstrating a partial facial nerve paresis. Right: Orbicularis oculi function intact bilaterally*