with transient leakage of fluid into and around the optic nerve head[3,4] or to deeper optic nerve head vascular compromise and axoplasmic flow disruption.[1]

Retinal new vessels are a recognised, if uncommon, clinical finding in patients with diabetic papillopathy,[2,4–6] and non-perfusion has been demonstrated in up to 50% of patients using fluorescein angiography.[2] However, associated disc neovascularisation is rare: to our knowledge it has been reported in only three patients, occurring several weeks after the observation of diabetic papillopathy in each case.[2,7] Our patient was unusual in presenting with signs of diabetic papillopathy and disc new vessels concurrently. In her case, the disc new vessels were clearly visible on slit-lamp biomicroscopy and were highlighted by the fluorescein angiography as shown in Fig. 1. However, in cases of less florid neovascularisation, distinction from the surface telangiectatic vessels characteristic of diabetic papillopathy may be more difficult and requires careful observation of the disc vessel pattern. New vessels of ischaemic origin tend to be more randomly orientated and elevated above the plane of the retina and disc, whereas the fine telangiectatic vessels of diabetic papillopathy are more radially orientated and lie within the disc and retina. Fluorescein angiography tends not to be helpful because profuse leakage from the disc vessels can occur in both conditions.[3]

From a management standpoint, diabetic papillopathy should be a diagnosis of exclusion, following investigation of other causes of swollen discs. This report highlights the importance of careful funduscopy to identify coexisting severe diabetic retinopathy and, in particular, optic disc neovascularisation.

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Sir,

Severe hypotony following cataract extraction in a patient on latanoprost

Latanoprost is a topical ocular hypotensive medication (prostaglandin $F_{2\alpha}$ analogue) which works via the uveoscleral pathway.[1] To the best of our knowledge, choroidal detachment caused by latanoprost in a glaucoma patient following cataract surgery has not been described. We report the following case to illustrate a potential side-effect of latanoprost.

Case report

A 74-year-old man with a past ophthalmic history of uncontrolled primary open angle glaucoma, underwent a right trabeculectomy in 1994 and a repeated right trabeculectomy augmented with 5-fluorouracil in 1995. In 1997, his intraocular pressure (IOP) became uncontrolled at 26 mmHg. Latanoprost was then commenced, with a dramatic effect in reducing the IOP from 26 to 12 mmHg in the right eye.

He underwent an uneventful right phacoemulsification with posterior implant under local anaesthetic in November 1999. The pre-operative visual acuity was 6/9 in the right eye with an IOP of 16 mmHg. His immediate post-operative period was unremarkable, with a visual acuity of 6/9 and an IOP of 16 mmHg on latanoprost and bethamethasone at 1 week. He presented 6 weeks post-operatively with a painful right eye. Examination revealed a visual acuity of 6/24 in the right eye and an IOP of 0 mmHg. The Seidel test was negative. The anterior chamber was deep and quiet. Fundal examination showed a large choroidal detachment in the supranasal and supratemporal areas. Latanoprost was discontinued and prednisolone 0.5% 4 times a day was started in the right eye. Four weeks later, the visual acuity in the right eye recovered to 6/9 with an IOP of 19 mmHg, and a complete resolution of choroidal effusion. At 3 months follow-up his IOP in the right eye remained controlled at 12 mmHg without treatment.

Comment

Our case illustrates a patient with poorly controlled glaucoma whose IOP stabilised on latanoprost treatment but who later developed profound hypotony following cataract extraction.

The pressure-lowering effect of phacoemulsification has been described in a number of studies, typically in the range of 1.1–2.5 mmHg.[2] In another study, the pressure-lowering effect of phacoemulsification in glaucoma patients was not effective until a year later.[3] The mechanism by which phacoemulsification lowers IOP is unclear, but it is thought to be related to the

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