and myoglobin, which indicates the tumour exhibits skeletal muscle differentiation.

Over the past decades, great progress has been made in the treatment of RMS. The 5-year survival rates increased from 25% in 1970 to 73%, as shown in the IRS-IV reported in 2001.[4] As most RMS is sensitive to chemotherapy and radiotherapy, exenteration is now rarely performed except for advanced or recurrent cases. The amount of tissue removed during biopsy is controversial but most ophthalmologists believe only a small biopsy is needed as orbital RMS has a favourable prognosis following radiation and chemotherapy, regardless of the amount of tissue removed. However, some prefer complete, or near complete, surgical removal when that can be achieved without major damage to vital structures like the optic nerve and extraocular muscles.[5]

Our patient with pleomorphic RMS did not respond to chemotherapy and the tumour grew rapidly. Excision of the tumour was the only option to check the relentless growth of the tumour. Although childhood RMS in general has been shown to respond to chemotherapy in IRS trials, most of these RMS are of either the embryonal or alveolar histological types. Studies showed that the presence of pleomorphic cell type is associated with poorer response to chemotherapy and prognosis.[6,7]

In summary, this case illustrates our experience in the management of a rare case of palpebral pleomorphic RMS, which failed to respond to chemotherapy and required complete surgical excision to control the disease process.

Acknowledgements

We have no financial interest in this case and this case has never been published or presented elsewhere.

References

1 Rousseau P, Flamant F, Quintana E, Voute PA, Gentet JC. Primary chemotherapy in rhabdomyosarcomas and other malignant mesenchymal tumors of the orbit: results of the International Society of Pediatric Oncology MMT 84 Study. *J Clin Oncol* 1994; **12**: 516–522.
2 Shields CL, Shields JA, Honavar SC, Demirci H. Clinical spectrum of primary ophthalmic rhabdomyosarcoma. *Ophthalmology* 2001; **108**: 2284–2292.
3 Shields JA, Shields CL. Rhabdomyosarcoma: review for the ophthalmologist. *Surv Ophthalmol* 2003; **48**(1): 39–57.
4 Crist WM, Anderson JR, Meza JL, Fryer C, Raney RB, Ruymann FB *et al*. Intergroup rhabdomyosarcoma study IV: results for patients with nonmetastatic disease. *J Clin Oncol* 2001; **15**: 3091–3102.
5 Lawrence Jr W, Hays DM. Surgical lessons from the Intergroup Rhabdomyosarcoma Study. *Natl Cancer Inst Monogr* 1981; **4**: 159–163.
6 Kodet R, Newton Jr WA, Hamoudi AB, Asmar L, Jacobs DL, Maurer HM. Childhood rhabdomyosarcoma with anaplastic (pleomorphic) features. A report of the Intergroup Rhabdomyosarcoma Study. *Am J Surg Pathol* 1993; **17**(5): 443–453.
7 Komdeur R, Klunder J, van der Graaf WT, van den Berg E, de Bont ES, Hoekstra HJ, Molenaar WM. Multidrug resistance proteins in rhabdomyosarcomas: comparison between children and adults. *Cancer* 2003; **97**(8): 1999–2005.

M Alhady, CT Ngo, AK Tan, GK Swethadri and CN Chua

The Ophthalmology Unit, Universiti Malaysia Sarawak (UNIMAS), Kuching, Sarawak, East Malaysia

Correspondence: CN Chua,
Ophthalmology Unit,
Universiti Malaysia Sarawak (UNIMAS),
Lot 77, Seksyen 22 Kuching Town Land District,
Jalan Tun Ahmad Zaidi Adruce, 93150 Kuching,
Sarawak, East Malaysia
Tel: +128 908 877;
Fax: +128 908 877.
E-mail: chuaoxford@hotmail.com

Department where the work was carried out:
Sarawak General Hospital, Kuching
Tel: +012 8908877;
Fax: +60 82 422564.
E-mail: chuaoxford@hotmail.com

Eye (2007) **21,** 291–293. doi:10.1038/sj.eye.6702541; published online 1 September 2006

Sir,

Cessation of migraines in a woman with low-tension glaucoma following the use of latanoprost: a favourable side effect?

Latanoprost is a phenyl-substituted isopropyl ester of prostaglandin F_{2a}, effective in low-tension glaucoma (LTG), lowering intraocular pressure (IOP), improving pulsatile ocular blood flow, and increasing ocular perfusion pressure.[1–2] Adverse topical effects such as anterior uveitis, cystoid macular oedema, conjuctival hyperaemia, and pigmentation of intra- and extraocular structures have been reported.[3] Systemic side effects are rare but may occur such as facial rash, cardiovascular effects,[4] and headaches.[5]

We report a favourable side effect of latanoprost in a woman with LTG. She reported complete cessation of her migraines after the use of latanoprost. She remained free of episodes of migraines for more than 1 year. Satisfactory challenge and rechallenge were performed.

Case report

A 57-year-old Caucasian woman, with a history of frequent migraines (every other day to once a week), low blood pressure (LBP), and peripheral vasospasm was referred with suspected glaucoma. Her best-corrected visual acuity was 6/6 in either eye. Clinical examination revealed findings consistent with a diagnosis of LTG. The patient was commenced on latanoprost monotherapy.

The IOP was reduced more than 30% from baseline. In addition the patient happily mentioned that as using latanoprost she had not experienced any new episodes of migraines although these were very frequent before she started the medication. The patient remained free of episodes of migraine for more than 1 year. There were no work, nutritional, or environmental factors that had changed during this year.

She subsequently developed ocular discomfort with latanoprost and her topical treatment was changed to brinzolamide. She suffered four episodes of migraine over a period of 1 month while she was using brinzolamide. She requested to have her topical therapy changed to latanoprost. No new episodes of migraines occurred when she reverted back to using latanoprost.

The patient later carried out a challenge and rechallenge on her own accord and her migraines once again recurred when she stopped latanoprost.

Comment

LTG is a distinct entity of primary open-angle glaucoma.[1] LBP and peripheral vasospasm are established risks factors for LTG.[1,6] Latanoprost is effective in LTG, lowering IOP, improving pulsatile ocular blood flow, and increasing ocular perfusion pressure.[1]

Many theories of migraine pathogenesis have been proposed, but the exact mechanism is still not understood. Usually the attack is started by one or more triggers.[7]

According to the literature,[5] latanoprost may rarely cause headaches in normal, with no peripheral vasospasm, subjects. However it has never been known to aggravate headaches in known migraine sufferers.

It is known that prostaglandin F_{2a} causes relaxation of the ciliary muscle which increases uveoscleral outflow.[2,8] This may relieve the ciliary muscle spasm which can cause pain referred to the brow and as a result can cause migraine-type headache.

In a comparative study involving brain magnetic resonance imaging,[9] patients with LTG were found to have a greater extent of cerebral infarcts and corpus callosum atrophy. This may imply a greater degree of neuronal degeneration, possibly on an ischaemic basis in LTG. It is known[10] that in the human ophthalmic artery, endothelium-derived nitric oxide, and endothelin are very potent modulators of vascular tone, suggesting that they play an important role in the regulation of local blood flow in the eye.

Prostaglandin F_{2a} is a known vasoconstrictor.[4] Systemic use of prostaglandin F_{2a} has been implicated in cardiac arrhythmias and vasospastic angina.[4,11] Sumatriptan, the most extensively investigated antimigraine drug,[7] is a known vasoconstrictor.

Although the plasma half-life of local applied latanoprost is short we postulate that the main mechanism that may have caused cessation of the migraines is via a slight systemic vasoconstriction.

Migraine relief after the use of latanoprost in patients with LTG has not been previously described. However, a prospective study with large series of patients and controls may be necessary in order to determine this favourable side effect.

References

1 Kamal D, Hitchings R. Normal tension glaucoma. A practical approach. *Br J Ophthalmol* 1998; **82**: 835–840.
2 Ravinet E, Mermoud A, Brignoli R. Four years later: a clinical update on latanoprost. *Eur J Ophthalmol* 2003; **13**(2): 163–175.
3 Warmar RE, Bullock JD, Balla D. Cystoid macular oedema and anterior uveitis associated with latanoprost. *Ophthalmology* 1998; **105**: 263–268.
4 Rajan MS, Syam P, Liu C. Systemic side effects of topical latanoprost. *Eye* 2003; **17**: 442–444.
5 Weston BC. Migraine headache associated with latanoprost. *Arch Ophthalmol* 2001; **119**: 300–301.
6 Pache M, Dubler B, Flammer J. Peripheral vasospasm and nocturnal blood pressure dipping—two distinct risk factors for glaucomatous damage? *Eur J Ophthalmol* 2003; **13**(3): 260–265.
7 Ferrari MD. Migraine. *Lancet* 1998; **351**: 1043–1051.
8 Mishima HK, Shoge KS, Takamatsu MT, Kiutsi Y, Tanaka J. Ultrasound biomicroscopic study of ciliary body thickness after topical application of pharmacologic agents. *Am J Ophthalmol* 1996; **121**: 319–321.
9 Ong K, Farinelli A, Billson F, Houang M, Stern M. Comparative study of brain magnetic resonance imaging findings in patients with low-tension glaucoma and control subjects. *Ophthalmology* 1995; **102**(11): 1632–1638.
10 Haefliger IO, Flammer J, Luscher TF. Nitric oxide and endothelin-1 are important regulators of human ophthalmic artery. *Invest Ophthalmol Vis Sci* 1992; **33**(7): 2340–2343.
11 Shigematsu S, Niwa H, Saikawa T. Vasospastic angina induced by prostaglandin F2 alpha. *Br Heart J* 1993; **69**: 361–365.

DA Karagiannis, P Venkatesh and N Kirkpatrick

Gloucestershire Eye Unit, Cheltenham, Great Britain

Correspondence: DA Karagiannis, Gloucestershire Eye Unit,

Sandford Road,
Cheltenham,
GL53 7AN,
Great Britain
Tel: +44 7789 755930;
Fax: +44 1242 272585.
E-mail: dimitrioskaragiannis@doctors.org.uk

Eye (2007) **21,** 293–295. doi:10.1038/sj.eye.6702548;
published online 1 September 2006

Sir,

Large metallic fragment found in the angle of anterior chamber after phacoemulsification, and its removal

The presence of intraocular metallic fragments after phacoemulsification surgery is common. Some times the tip of the second instrument can break off and dislodge, this might not be revealed in immediate postoperative period. We report an interesting case in which a large metallic fragment was found at the angle of the anterior chamber on post-phacoemulsification examination. This was later removed under gonioscopic visualisation.

Case report

A 78-year-old male underwent left eye phacoemulsification. During surgery, at the end of lens removal, the tip of the second instrument appeared to have broken off (Figure 1). The way the tip of the second instrument broke is not known. The procedure otherwise went well. Despite thorough search, the metallic fragment was not found either inside the eye or in tubing cassette. X-ray of orbits did not show the presence of intraocular foreign body.

Figure 1 Lens manipulating instruments, the right extreme instrument with blunt bulged tip and narrow neck, the middle instrument with broken head from our case, compared with, left extreme, another instrument with no narrow weak point.

On postoperative examination, visual acuity in the left eye was 6/5 and the eye was settling down quite well. However, on gonioscopy, a metallic fragment was found located in the inferior angle of left anterior chamber (Figure 2).

The fragment was removed with the aid of gonioscope under operating microscope (Figure 3). The postoperative course was completely uneventful.

Comment

The presence of intraocular metallic fragments is not uncommon after phacoemulsification.[1] Such particles are believed to derive from the phaco-tip surface;[1,2] from irrigation–aspiration ducts;[3] and from contact of phaco-tip with the second instrument.[4] Second

Figure 2 Gonioscopy of the inferior angle of the anterior chamber showing large metallic fragment.

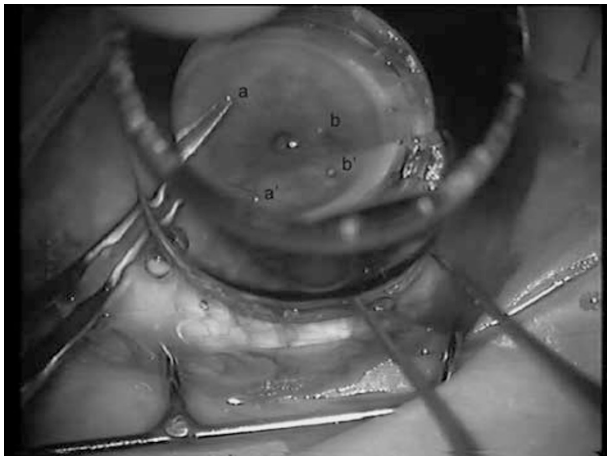

Figure 3 Removal of the metallic fragment with the aid of gonioscope. Forceps holding the fragment with its reflection in the gonio-mirror is seen (a, a′). On the right side of the picture artefact, air bubble with its reflection is seen (b, b′).