paralysis. Essential thrombocythaemia is a rare condition with an incidence of around 1–2.5 per 100 000 per year. It affects both sexes and is most common in the sixth and seventh decades. More than one-half of the patients are asymptomatic when the condition is detected on a routine full blood count.[1] Untreated, patients are at increased risk of cerebrovascular accidents, deep vein thrombosis, and gastrointestinal tract bleeding. Neurologic symptoms are common and include headache, paresthesiae, and amaurosis fugax.[2] A small number of case reports of central retinal vein occlusion and central retinal artery occlusion in essential thrombocythaemia have been published.[3–6]

The differential diagnosis of a persistently high platelet count includes polycythaemia vera (increased red cell mass in the presence of normal iron stores), chronic myeloid leukaemia (presence of the Philadelphia chromosome) and agnogenic myeloid metaplasia (prominent marrow fibrosis).

Essential thrombocythaemia is known to affect the microvasculature and we hypothesise that the thrombocytosis contributed to the ischaemic third nerve palsy in our patient. Although this patient had increased blood pressure at presentation, two large studies have shown that hypertension is not an independent risk factor for ocular motor nerve palsies.[7,8] While we accept that hypertension may have played a contributory role, we believe that the presence of headache (a common symptom in patients with essential thrombocythaemia) and the rapid resolution of symptoms following initiation of treatment support our hypothesis that essential thrombocythaemia was a precipitating, if not sole causative factor, for the third nerve palsy in this case.

Reports of third nerve palsy secondary to other haematologic abnormalities are also very uncommon. However, it is interesting to note that, in the two reported cases of third nerve palsy secondary to monoclonal gammopathy,[9,10] pain was a presenting feature. This may suggest involvement of the third nerve in the cavernous sinus as sensory fibres from the ophthalmic division of the fifth nerve join the third nerve in the lateral wall of the cavernous sinus. Essential thrombocythaemia should be included in the differential diagnosis of ischaemic ocular motor nerve palsies. This case also illustrates the importance of routine haematology work-up in cases of spontaneous ocular motor nerve palsies.

References

1 Imbert M, Pierre R, Thiele J, Vardiman JW, Brunning RD, Flandrin G. Essential thrombocythaemia. In: Jaffe ES, Harris NL, Stein H, Vardiman JW (eds) *WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues*. IARC Press:Lyon, 2001 pp 39–41.
2 Jabaily J, Iland HJ, Laszlo J, Massey EW, Faguet GB, Briere J *et al*. Neurological manifestations of essential thrombocythemia. *Ann Intern Med* 1983; **99**: 513–518.
3 Yoshizumi MO, Townsend-Pico W. Essential thrombocythemia and central retinal vein occlusion with neovascular glaucoma. *Am J Ophthalmol* 1996; **121**(6): 728–730.
4 Imasawa M, Iijima H. Multiple retinal vein occlusions in essential thrombocythemia. *Am J Ophthalmol* 2002; **133**(1): 152–155.
5 Liu M, Lee AG, Rice L, Lambert HM. Bilateral retinal vascular occlusive disease in essential thrombocythemia. *Retina* 1999; **19**(6): 563–564.
6 Strassman I, Silverstone BZ, Seelenfreund MH, Sheer A, Berson D. Essential thrombocythemia: a rare cause of central retinal artery occlusion. *Metabol Pediatr Syst Ophthalmol* 1991; **14**: 18–20.
7 Patel SV, Holmes JM, Hodge DO, Burke JP. Diabetes and hypertension in isolated sixth nerve palsy: a population-based study. *Ophthalmology* 2005; **112**(5): 760–763.
8 Jacobson DM, McCanna TD, Layde PM. Risk factors for ischemic ocular motor nerve palsies. *Arch Ophthalmol* 1994; **112**: 961–966.
9 Bogousslavsky J, Steck AJ. Bilateral third nerve palsy and anterior ischemic optic neuropathy. *Neuro-ophthalmology* 1986; **6**: 117–120.
10 Ruggles KH, Massey EW. Pupil-sparing oculomotor palsy with IgG momoclonal paraproteinemia. *Ann Ophthalmol* 1981; **13**: 875–876.

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Sir,

Intrableb triamcinolone acetonide injection after bleb-forming filtration surgery (trabeculectomy, phacotrabeculectomy, and trabeculectomy revision by needling): a pilot study

Bleb-forming filtration procedures, such as trabeculectomy, combined phacotrabeculectomy, and trabeculectomy revision by needling, are surgical options in medically uncontrolled glaucoma. Postoperative topical steroid significantly increased the chance of

filtration success[1–3] by inhibiting fibroblast proliferation.[4] Topical steroid application, however, requires compliance by and dexterity of the patient. Direct injection of triamcinolone acetonide (TA) into the bleb at the conclusion of surgery may be a more direct, sustained, and convenient mode of steroid delivery. The bulk of injected TA also serves as a barrier between the inflamed conjunctiva and sclera. This pilot study evaluates whether intrableb injection of TA is an effective and safe route for steroid application after bleb-forming filtration surgery.

Case reports

In consecutive glaucoma patients undergoing trabeculectomy, phacotrabeculectomy, and needling revision by the same surgeon (CT) during the study period of 3 months, 0. 03 ml of TA (Kenacort-A IM, Bristol-Myers Squibb) (40 mg ml^{-1}) was injected using a bent 27-G needle into the filtration bleb at the conclusion of surgery. The entry site for the injection needle was at least 1 cm from the scleral flap, and covered by the upper eyelid. The injection needle was passed between the conjunctiva and the sclera towards the scleral flap. The TA was injected immediately adjacent and posterior to the scleral flap (Figure 1). Postoperative topical corticosteroid (Pred Forte, Allergan) was prescribed four times a day, because of the uncertain efficacy of intrableb TA injection.

Eleven eyes of 11 patients were recruited (seven men and four women). Mean age $\pm$ standard deviation was 57.4 $\pm$ 19.7 years. Their diagnoses included primary open-angle glaucoma (five eyes) and chronic angle-closure glaucoma (six eyes). Three eyes underwent phacotrabeculectomy (with mitomycin C 0.4 mg ml^{-1} applied to sclera for 3 min), three eyes underwent trabeculectomy (with mitomycin C 0.4 mg ml^{-1} applied to sclera for 3 min), and five eyes underwent needling revision (with single intraoperative subconjunctival injection of 5 mg 5-fluorouracil).

Mean intraocular pressure (IOP) was reduced from 23.7 $\pm$ 7.1 mmHg (range, 9–34 mmHg) preoperatively to 12.2 $\pm$ 5.7 mmHg (range, 5–20 mmHg) at 1 month and 11.9 $\pm$ 5.1 mmHg (range, 5–20 mmHg) at 3 months after surgery. The mean number of topical glaucoma drugs was reduced from 3.4 $\pm$ 1.0 (range, 2–5) preoperatively to 0 in all eyes at both 1 month and 3 months.

There was minimal postoperative anterior segment inflammation in all cases. Microcystic and spongy blebs were achieved in all cases.

All corneas were clear before surgery, and at 1 and 3 months after surgery. There was no statistically significant reduction in corneal endothelial cell count up to 3 months after surgery ($P = 0.11$).

The only complication was persistent subconjunctival TA deposit in one case of needling up to 3 months after surgery (Figure 2), with no other consequences. In this eye, IOP was effectively reduced from 26 mmHg preoperatively to 16 mmHg at 3 months, whereas the number of glaucoma drugs was reduced from 3 to 0. There was no incident of TA suspension entering anterior chamber. There were also no bleb infection, no conjunctival ulceration, and no observable cataract progression in those phakic-operated eyes.

Comments

Giangiacomo *et al*[5] previously described preoperative subconjunctival TA injection for trabeculectomy.

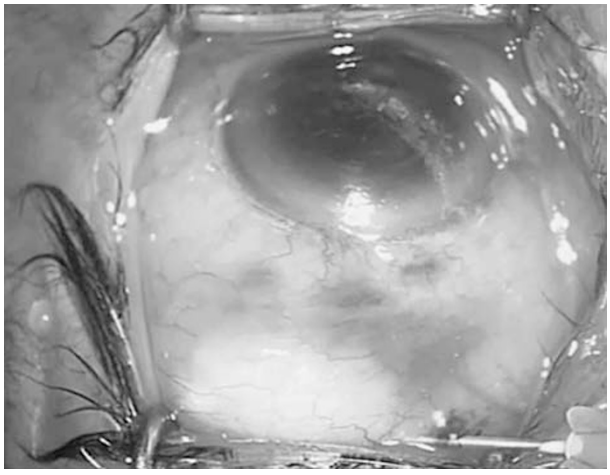

Figure 1 Subconjunctival injection of TA immediate adjacent and posterior to the scleral flap at the conclusion of bleb-forming filtration surgery.

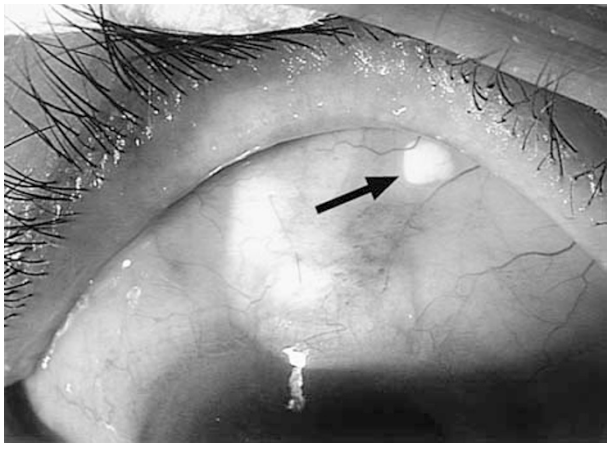

Figure 2 Subconjunctival deposit of white TA powder persisted in one eye for up to 3 months after surgery, but no adverse clinical effects had arisen.

However, they did not employ a standardized timing or dosage for the TA injection. As there is no evidence that injecting TA at 2 days to 1 week before surgery offers additional benefits, we believe our approach of TA injection at conclusion of surgery may be more convenient, and the operative site can be more accurately targeted.

Sterile conjunctival ulceration following subconjunctival TA injection has been reported.[6,7] Exact mechanism was unclear, but underlying autoimmune disease,[7] an anterior interpalpebral injection site,[6] a dose-dependent toxicity, and a bad batch of triamcinolone[6] were proposed as possible causes. Our patients did not have known autoimmune diseases, and our injection site was not exposed in the interpalpebral space. Furthermore, our dosage of TA (1.2 mg) was substantially lower than the dosages routinely used for chronic uveitis (20–40 mg). For these reasons, we believe the risk of conjunctival ulceration associated with our approach should be minimal, although the present series may be too small to address this risk.

In conclusion, intrableb TA injection in bleb-forming filtration surgery is compatible with a desirable clinical outcome, and appears to be safe up to 3 months after surgery. We are evaluating whether intrableb TA injection will offer filtration patients additional clinical benefits when compared to patients receiving topical steroid only, in a randomized controlled trial.

References

1 Araujo SV, Spaeth GL, Roth SM, Starita RJ. A ten-year follow-up on a prospective, randomized trial of postoperative corticosteroids after trabeculectomy. *Ophthalmology* 1995; **102**: 1753–1759.
2 Roth SM, Spaeth GL, Starita RJ, Birbillis EM, Steinmann WC. The effects of postoperative corticosteroids on trabeculectomy and the clinical course of glaucoma: five-year follow-up study. *Ophthalmic Surg* 1991; **22**: 724–729.
3 Starita RJ, Fellman RL, Spaeth GL, Poryzees EM, Greenidge KC, Traverso CE. Short- and long-term effects of postoperative corticosteroids on trabeculectomy. *Ophthalmology* 1985; **92**: 938–946.
4 Blumenkranz MS, Claflin A, Hajek AS. Selection of therapeutic agents for intraocular proliferative disease. Cell culture evaluation. *Arch Ophthalmol* 1984; **102**: 598–604.
5 Giangiacomo J, Dueker DK, Adelstein E. The effect of preoperative subconjunctival triamcinolone administration on glaucoma filtration. I. Trabeculectomy following subconjunctival triamcinolone. *Arch Ophthalmol* 1986; **104**: 838–841.
6 Agrawal S, Agrawal J, Agrawal TP. Conjunctival ulceration following triamcinolone injection. *Am J Ophthalmol* 2003; **136**: 539–540.
7 Allen QB, Lowder CY, Meisler DM. Conjunctival necrosis following the administration of subconjunctival corticosteroid. *Ophthalmic Surg Lasers* 1998; **29**: 779–780.

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Sir,

Iris damage and acute pigment dispersion following photo-epilation

Laser-assisted hair removal (photo-epilation) is becoming an increasingly popular treatment. It relies on the principle of selective photothermolysis whereby use of an appropriate wavelength and pulse duration of light causes injury that is confined to the desired target tissue while sparing surrounding structures.[1,2] We report a case of iris damage and acute pigment dispersion after the use of long-pulsed infra-red (LPIR) alexandrite laser for photo-epilation of the eyebrow.

Case report

A 38-year-old woman presented with left ocular discomfort associated with photophobia, redness, and blurred vision. She had undergone photo-epilation of her eyebrows earlier that day with a 755 nm LPIR alexandrite laser (20 ms pulse duration, 22 J/cm^2 fluence, 10 mm diameter spot). Although treatment to her right eyebrow