often ignored in clinical practice. We report a case that highlights the need for constant vigilance in the context of such ophthalmic therapy.

Case report

A 66 year old white man, on treatment with topical latanoprost for primary open angle glaucoma, required further reduction of intraocular pressure, in the pursuit of which topical brinzolamide was added to one eye only.

At review 3 months later, the patient reported having stopped brinzolamide on his own initiative because of lethargy and a bad taste in the mouth. The subject had worn a silver chain around his neck for many years; interestingly, this chain had turned black within 2 days of starting topical brinzolamide. The chain was professionally cleaned and then re-worn, immediately upon which it turned black once again (fig 1).

Adequate control of intraocular pressure was subsequently established with a combination of topical travoprost, timolol, and pilocarpine.

Comment

The clinical history strongly indicates the development of metabolic acidosis in response to topical brinzolamide, a recognised consequence of therapy with carbonic anhydrase inhibitors. The blackening of the silver chain was caused by oxidation of the silver, most likely as a result of exposure to acidic sweat, a homeostatic mechanism to normalise the pH of the extracellular fluid. Incontrovertible objective verification of such disturbance of the acid-base balance would necessitate re-challenging the patient with the drug along with arterial blood gas analysis, a measure that was deemed excessive and inappropriate on the grounds of patient safety.

It would appear remarkable that such acidosis developed in response to an apparently small dose of medication, the drug having been instilled twice daily in only one eye—until the pharmacodynamics of topical ocular medications are studied. It is well recognised that topically administered drugs are rapidly absorbed into the systemic circulation.[1] Manual lacrimal sac compression has been advocated as a means of reducing systemic absorption, on the basis that drugs are absorbed more effectively from the ciliated columnar (respiratory) epithelium of the nasal passages—the effects of such lacrimal compression are, however, variable.[2 3] Lacrimal compression also does not affect drug absorption across the vascular conjunctiva, which has been estimated at being 10 times faster than the transcorneal route. Transconjunctival absorption also competitively reduces absorption across the cornea, thus depotentiating any ocular therapeutic effect.

It has been estimated that less than 5% of an instilled dose of a topical drug is absorbed intraocularly.[4] The achievement of a desired ocular therapeutic effect therefore often necessitates higher concentrations and frequent instillations of a drug, with the attendant increased risk of systemic effects. In this context it is also clinically significant that topical eye drops, when absorbed, do not undergo first pass metabolism, bypassing as they do the liver and gaining direct entry into the systemic circulation.

We submit that the common tendency to consider eye drops as representing miniscule and systemically insignificant doses of therapeutic agents is therefore ill founded—the prescription of eye drops merits comparable weight and consideration as that of systemic medication.

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doi: 10.1136/bjo.2005.075622

Accepted for publication 21 August 2005

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Figure 1 The blackened silver medallion.

Anterior ischaemic optic neuropathy after rotator cuff surgery

Perioperative visual loss has been associated with many types of non-ocular surgery.[1] Risk factors include combinations of prolonged surgical times, hypotension, anaemia due to blood loss, or prone positioning.[2] We are not aware of another report of perioperative visual loss in which the only risk factor was hypotension.

Case report

A 55 year old man had left rotator cuff surgery. He was supine, surgical time was 3 hours, and blood loss was minimal. Postoperatively he experienced ''kaleidoscopic'' vision in the inferior visual field of his left eye which cleared. Four weeks later he had right rotator cuff surgery. Again he was supine, surgery lasted 2–3 hours, and blood loss was 100 ml. After recovery he had ''kaleidoscopic'' vision in the same eye. It did not resolve and his vision continued to deteriorate prompting him to seek ophthalmological care.

His medical history was significant for treated hyperlipidaemia. He quit smoking 35 years earlier. Visual acuity was 20/20 in the right eye and counting fingers in the left. He identified 9/12 colour plates in the right eye and 0/12 in the left. He had a left afferent papillary defect. The right optic disc was

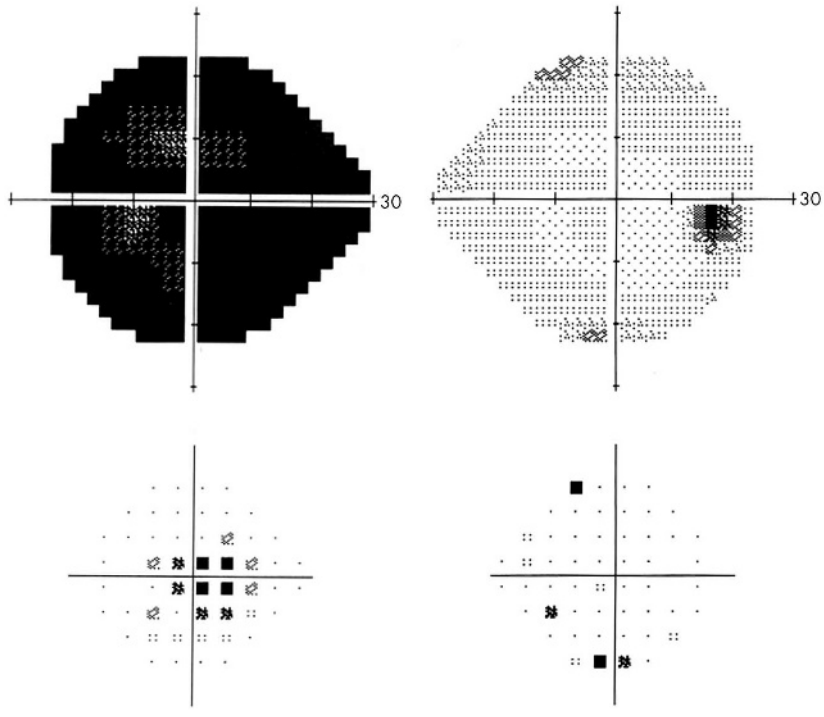

Figure 1 Humphrey automated perimetry after the second surgery and loss of vision in the left eye.

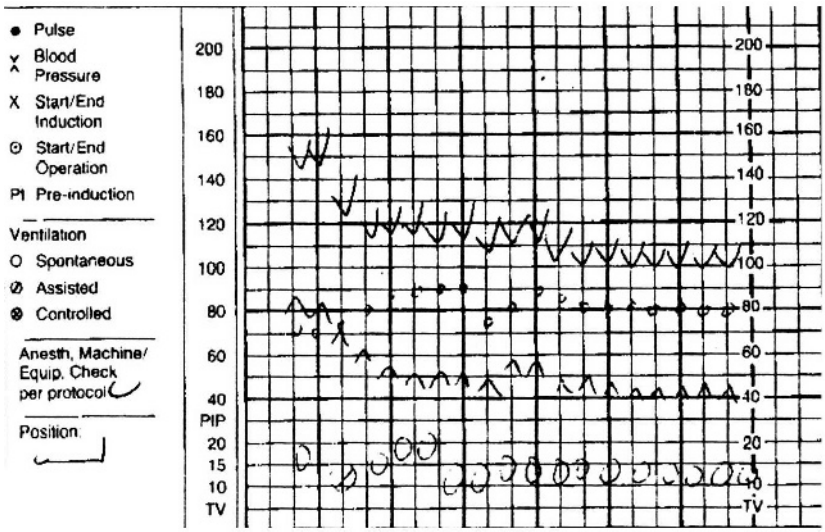

Figure 2 Intraoperative anaesthesia record showing 80 minute drop in mean arterial pressure. Small boxes, 10 minutes; Large boxes, 30 minutes; downward pointing arrowheads, systolic pressure; upward pointing arrowheads, diastotic pressure.

congenitally anomalous, the left pale and swollen. Visual fields revealed an inferior arcuate defect in the left eye.

Comment

Many types of non-ophthalmic surgery have been associated with anterior ischaemic optic neuropathy (AION).[1] Our patient's preoperative risk factors included treated hyperlipidaemia and anomalous nerves. During surgery he did not have any of the established risk factors for perioperative non-AION with the exception of prolonged hypotension. His MAP was reduced by 41.6% for 80 minutes. The previous surgery on the patient's left side decreased the MAP only 28%.

It is likely that this hypotension combined with anomalous nerves led to his optic nerve infarction. The perfusion pressure of the eye drops linearly with the mean arterial blood pressure.[3] If there are areas of atherosclerosis or watershed zones present in a particular optic nerve a significant drop in the perfusion pressure to the eye could result in ischaemia. Of interest, he experienced transient visual obscurations after his first surgery with a smaller drop in his MAP, perhaps signifying a vascular system susceptible to hypotensive episodes.

Bhatti and Enneking described decreased vision and ophthalmoparesis following rotator cuff surgery but the patient did not have non-AION and the vision eventually recovered.[4] In addition, after a review of Medline, we did not find any other cases of perioperative visual loss in whichh the only risk factor was prolonged hypotension. This underscores the importance of hypotension as an independent risk factor for perioperative visual loss.

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doi: 10.1136/bjo.2005.081455

Accepted for publication 24 August 2005

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Bilateral intraocular involvement in Lemierre's syndrome

First described in 1936, Lemierre's syndrome is an oropharyngeal infection characterised by septic thrombophlebitis of head and neck veins, complicated by dissemination of septic emboli to pulmonary and systemic sites.[1] Ophthalmic involvement in this syndrome is extremely uncommon, having been reported previously in a patient with retro-orbital involvement and proptosis,[2] and more recently as a case of endogenous endophthalmitis.[3] Here, we describe a case of bilateral intraocular involvement in this interesting and rare disease.

Case report

A previously healthy 14 year old African American female presented to the emergency department with cough, dyspnoea and tachypnoea, and a pulse oximeter reading of 70% on room air. A chest *x* ray demonstrated diffuse bilateral pulmonary infiltrates and a right sided pleural effusion. Empirical treatment with intravenous vancomycin 1 g intravenously every 12 hours and cefotaxime 1 g intravenously every 8 hours was initiated. She was transferred to the intensive care unit for treatment and further examination. Her laboratory studies were remarkable for a rapid decline in her haemoglobin from 12.6 g/dl to 8.6 g/dl and a drop in platelet count from 57 000×10^9/l to 35 000×10^9/l.

Computed tomography (CT) of the neck revealed a left parapharyngeal abscess adjacent to a clot in the left internal jugular vein (fig 1). Multiple other lesions, thought to be septic emboli, were found systemically, including in her liver, spleen, and lungs. Subsequent blood cultures were positive for *Fusobacterium necrophorum*. Her antibiotic regimen was changed to clindamycin 300 mg intravenously every 6 hours.

Initial ophthalmic examination was significant for visual acuity of light perception in the right eye and 20/150 in the left with normal pupillary responses, intraocular pressure, and anterior segments. Indirect ophthalmoscopy revealed a dense vitreous haemorrhage in the right eye, precluding any view of the retina, and a preretinal haemorrhage in the left eye centred over the macula. B-scan ultrasonography of the right eye demonstrated vitreous haemorrhage and a subretinal mass with moderate internal reflectivity (fig 2). Three weeks after her initial presentation, her platelet count normalised and blood cultures were negative, but the dense vitreous haemorrhage is the right eye persisted with light perception vision. Subsequently, a pars plana vitrectomy was performed on the right eye, from which Gram stains and cultures were negative. Intraoperatively, the subretinal mass

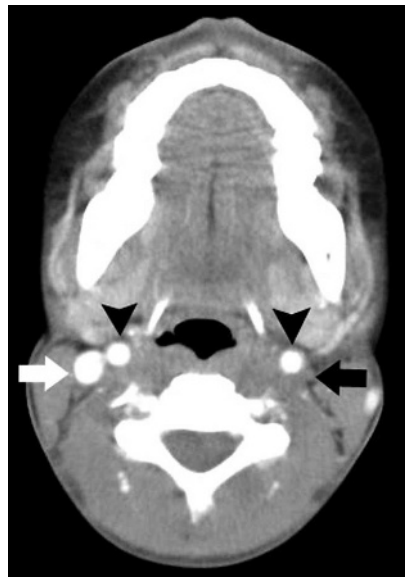

Figure 1 Computed tomography (CT) scan with intravenous contrast of the neck at the level of the hyoid bone. Note the patency of the carotid arteries bilaterally (black arrowheads). On the right, the internal jugular vein is patent (white arrow), whereas on the left the internal jugular vein is thrombosed (black arrow).

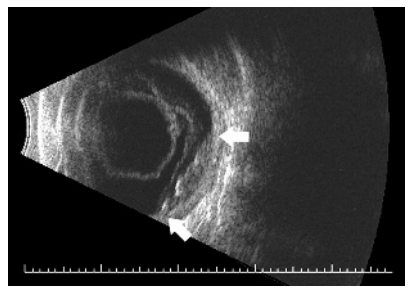

Figure 2 B-scan ultrasound of the right eye demonstrates a subretinal mass of moderate internal reflectivity (white arrows) accompanied by vitreous haemorrhage.