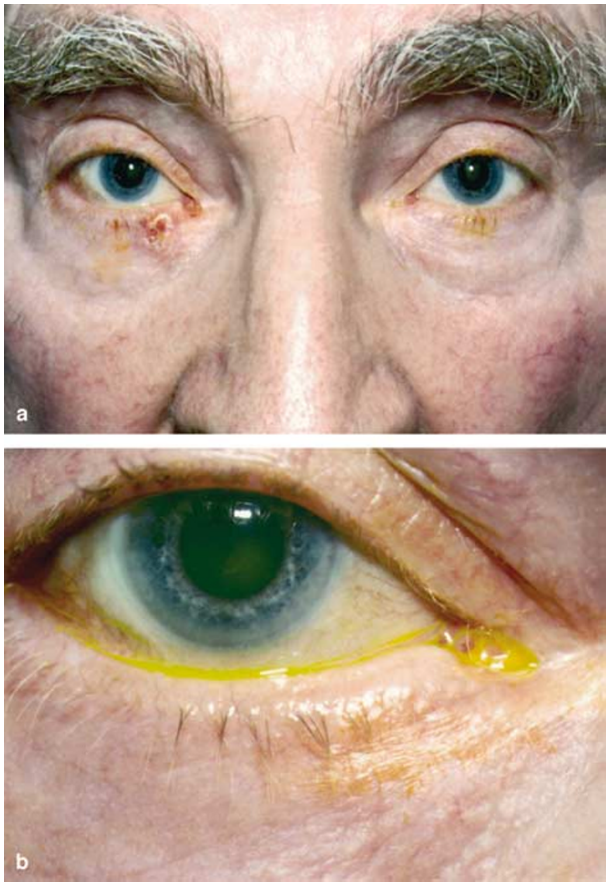

Figure 1 (a) The basal cell carcinoma on the lower lid as seen on presentation in January 1997. (b) Complete spontaneous regression of the basal cell carcinoma in July 1997.

Discussion

Basal cell carcinoma (rodent ulcer), the most common tumour affecting the eyelids, is responsible for considerable morbidity owing to its locally invasive nature.[1] Arising from the basal layer of the epidermis, it is responsible for 85–90% of lid malignancies, two-thirds of which are seen in the lower lid.[1,2]

Diagnosis is made on the clinical appearance and confirmed by histology, the adenoid and metatypical types being the most common differentiated forms.[3] The usual course of the disease is a gradual enlargement of the lesion with underlying tissue destruction necessitating treatment. Treatment options include cryotherapy, radiation, chemotherapy, laser ablation and electrodessication, but surgical excision is the most widely accepted treatment of choice.[4]

Regression of epithelial skin tumours like keratoacanthoma, familial self-healing epithelioma, melanoma and basal cell carcinoma has been reported in dermatology.[5,6] An immune response mediated by CD4+ T lymphocytes through the release of cytokines[6,7] has been postulated to be the causative mechanism. Partial regression of basal cell carcinomas has been reported in about 50% of skin lesion;[5] however, a complete regression of the lesion as in the present instance has, to the best of our knowledge, never been reported in ophthalmic literature.

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Sir,

Delayed allergic reaction to hyaluronidase: a rare sequel to cataract surgery

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Peribulbar anaesthesia using lignocaine or bupivicaine is frequently used in ophthalmic surgery. Hyaluronidase is often used as an adjunct to aid dispersal of these agents.[1–4] We report a case of orbital inflammation secondary to delayed postoperative hyaluronidase allergy leading to visual loss.

Case report

A 77-year-old lady with advanced primary open angle glaucoma treated by bilateral trabeculectomies developed a cataract in her left eye, reducing her best-corrected visual acuity to 6/24 part. Previous uneventful right phacoemulsification under local anaesthesia had improved vision to 6/9 despite advanced bilateral glaucomatous field loss (not shown). She was also known to suffer from osteoarthritis, angina, atrial fibrillation, hypertension, but no known allergies.

She underwent uneventful sutureless left phacoemulsification and lens implant under local anaesthesia. At 24-h postoperatively her left eye was comfortable and quiet, with unaided vision of 6/18. After 28-h later she presented with periorbital swelling of the left eye associated with nausea and vomiting of 4 h duration.

Her visual acuity was 6/12 in the right eye and hand movements in the left eye. Her left eyelids were tender and very oedematous being unable to spontaneously open her eye (Figure 1). Intraocular pressures (IOP) were 12 and 20 mmHg, respectively. The pupils were equal and reactive with no evidence of a relative afferent pupil defect. Left ocular motility was restricted in all directions of gaze with 3 mm of axial proptosis. Slit lamp biomicroscopy revealed a clear cornea and quiet anterior chamber. The left optic disc was pale, fully cupped with a splinter haemorrhage.

CT scan confirmed left-sided proptosis with preseptal oedema, increased intraconal soft tissue density, and minimal extra-ocular muscle enlargement. The paranasal sinuses were clear (Figure 2). A full blood count, erythrocyte sedimentation rate, electrolytes, and blood glucose were normal. Blood cultures were negative, but a conjunctival swab grew coagulase negative Staphylococcus (sensitive to coamoxyclav).

At this stage, our differential diagnosis included orbital cellulitis, allergic orbital inflammation, and retrobulbar haemorrhage.

The episode was initially managed as orbital cellulitis, since there was no evidence of allergy 24 h after surgery and the use of systemic steroids could predispose to cavernous sinus thrombosis.

Eight-hourly intravenous coamoxyclav 1.2 g and metronidazole 500 mg were commenced. Chlorpheniramine 10 mg was also administered intravenously twice daily along with intravenous fluids and analgesia.

After 24-h later, systemic improvement was apparent, although the ocular features remained unchanged. The following day, she was noted to have developed a left relative afferent pupil defect. The intraocular pressure of the left eye was 21 mmHg, for which 0.5% apraclonidine was given.

Over the next 2 days, the ocular and systemic signs improved and the intravenous antibiotics were switched to oral. At 1 week, the proptosis had regressed and she was able to open her left eye. Ocular motility was full, but her left visual acuity remained at hand movements. She was discharged on topical prednisolone 1%, timolol 0.25% and brimonidine 0.2% to the left eye.

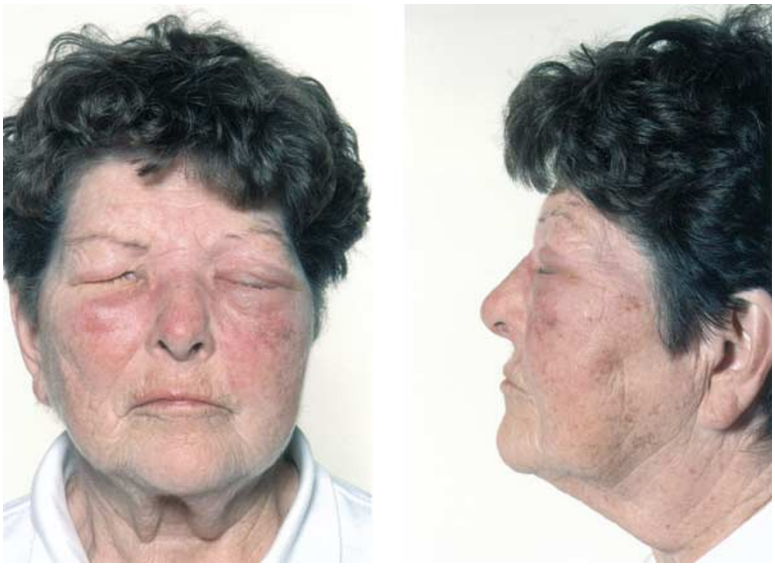

Figure 1 Hyperaemic and swollen left eyelids with inability to spontaneously open the left eye.

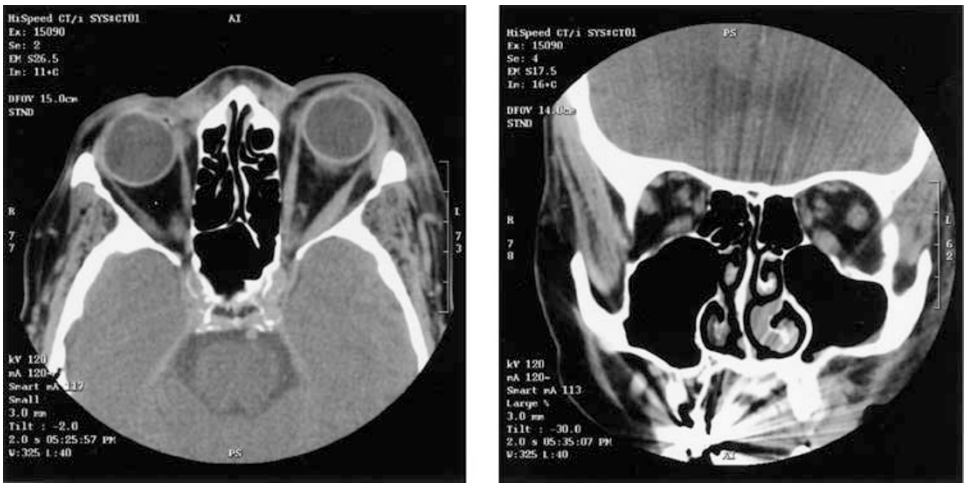

Figure 2 CT scan of both orbits. Left (axial): left proptosis with preseptal oedema, increased intraconal soft tissue density, minimal extraocular muscle and lacrimal gland enlargement. Right (coronal): Minimal extraocular muscle enlargement and clear paranasal sinuses.

After 2 weeks later her left visual acuity remained hand movements, with an IOP of 17 mmHg and a fully cupped optic disc. She was referred for allergy testing.

She was skin-prick tested for lignocaine, bupivicaine, hyaluronidase, adrenaline, mepivicaine, prilocaine, rubber, latex, and house dust mites. Skin-prick testing was strongly positive for hyaluronidase but negative for all other allergens.

Comment

Adverse reactions to periocular injections may arise either from the anaesthetic compound itself or from the mechanical manipulation of the needle.[1]

Three previous case reports of hyaluronidase allergy were available on literature review. The onset of allergic response reported was found to be variable, ie immediate (intraoperative), early (within a few hours), intermediate (within a few days), and late (within weeks). The present case had an intermediate onset.

All but one of the cases reported to date had a previous uneventful retrobulbar anaesthetic containing hyaluronidase. Primary sensitisation seemed a prerequisite to such an allergic response. The variability in onset of symptoms following exposure to the offending allergen and response to skin testing would suggest that type I and type IV hypersensitivity might both contribute to this response.

Kempeneers *et al*[1] reported a series of five patients with hyaluronidase-induced orbital pseudotumour as a complication of retrobulbar anaesthesia. Four of the five patients were sensitive to hyaluronidase on intradermal testing of all anaesthetic components. Four of the five patients were exposed to retrobulbar anaesthesia for the second time while one was subjected to it for the first time. The onset of symptoms of orbital inflammation ranged from a few hours to a few weeks. Lymphocytic transformation tests (LTT) revealed a delayed hypersensitivity reaction.

Minning *et al*[5] reported a case of intraoperative hyaluronidase allergy simulating an expulsive choroidal haemorrhage. This patient had a previous uneventful retrobulbar injection containing hyaluronidase. Skin testing confirmed an allergy to hyaluronidase. Taylor *et al*[6] also reported a similar case of intraoperative hyaluronidase allergy during intracapsular cataract extraction. Allergic angioedema following local anaesthesia for dental and ophthalmic surgery has been described, the causative allergen being hyaluronidase.[7]

This case is the first case report of hyaluronidase allergy with peribulbar anaesthesia for sutureless phacoemulsification and intraocular lens implant rather than extracapsular cataract surgery.

This case demonstrates that despite being less common than infection, allergy can account for delayed postoperative orbital inflammation following intraocular surgery. Detection of an allergy to hyaluronidase suggests that systemic steroids may have been of benefit once an infective focus had been ruled out. Secondly, there is a case for avoiding the use of hyaluronidase in patients who have had previous periocular local anaesthesia, particularly if the optic nerve is vulnerable, as in patients with advanced glaucoma.

Proprietary interests: None.

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Sir,

Iris heterochromia after vertical squint surgery in a patient with SLE

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Anterior segment ischaemia is a known complication of squint surgery particularly in those involving more than two recti muscles. Certain factors can increase this risk such as sickle-cell anaemia and hyperviscosity syndrome. To the best of our knowledge, there has been no reported case of anterior segment ischaemia following squint surgery in a patient with systemic lupus erythematosus (SLE). We present a case of iris heterochromia as the only feature of anterior segment ischaemia following two-recti-muscle surgery in a patient with SLE.

Case report

A 45-year-old lady diagnosed with SLE for 22 years was referred to the clinic with irritable and gritty eyes. She was diagnosed with keratoconjunctivitis sicca and prescribed ocular surface lubricants. During the consultation, she also mentioned a long-standing diplopia that she was able to control by tilting her head. There was no history of previous squint surgery. Recently however, the diplopia has become symptomatically worse following an episode of patching of the left eye for recurrent corneal erosion.

Examination revealed an abnormal head posture with head turn to the right, head tilt to the left shoulder and chin up. The right hypertropia measured 13 dioptres for near and 20 dioptres for distance. Hess chart and eye movements indicated a possible bilateral Brown's syndrome with the left eye more affected than the right (Figure 1a).

She was listed for squint surgery in the hypertropic eye. Traction tests under anaesthesia revealed limitation of both eyes on moving the globe up and consistent with bilateral Brown's syndrome. She underwent right inferior rectus resection 5 mm and right superior rectus recession 4 mm on an adjustable suture. The superior rectus was pulled forward on adjustment.

After a postoperative period of 8 weeks, the squint measured 3 dioptres of right hypotropia and there was a marked reduction of abnormal head posture (Figure 1b). The patient had noticed that her right eye was a lighter colour and the pupil mis-shapen. Slit-lamp examination showed segmental iris stromal atrophy superiorly and inferiorly corresponding to the site of the operated muscles (Figure 2). Anterior segment examination was otherwise unremarkable with normal intraocular pressure. Iris fluorescein angiography was done but was unhelpful on account of her dark irides.[3] Her sectoral iris hypochromia remains unchanged to date, despite a trial of topical and systemic steroids.

Comment

SLE is an autoimmune disease with multiorgan involvement, skin, joints and vasculature being the most commonly involved. It occurs worldwide (0.1% incidence in the UK[1]) and has a female preponderance. The presence of autoantibodies such as anti-double-stranded DNA and antismooth muscle antibodies is supportive but not diagnostic of the condition. The ocular manifestations of this disease include keratoconjunctivitis sicca, retinal vasculitis, iritis, optic neuritis, and papilloedema. Ocular motility disorders can originate from anywhere—from the cerebral cortex to the extraocular muscles.

Squint surgery involving recti muscles can permanently disrupt the anterior ciliary arteries in any patient and can lead to anterior segment ischaemia. Studies have documented that procedures involving the vertical recti are at the greatest risk of precipitating this.[2] Further