Comment

Sanichlor in a 1:1000 dilution solution is a convenient method of preparing a chlorinated solution for disinfection. It is a known respiratory, skin and ocular irritant, which is featured in the list of hazardous chemicals by the International Labour Organization.² Bronchial hyperresponsiveness and asthma has been reported³⁻⁷ with chlorine gas inhalation and chloramine T, an organic disinfectant. Chloramines are formed when chlorine combines with organic matter in tissues. Chloramines have also been reported⁸⁻¹⁰ to cause allergic contact urticaria. The above-reported asthma and contact dermatitis in our ophthalmologists can be ascribed to exposure to chlorine and its derivatives like chloramines, the actual mechanism possibly being a combination of mucosal irritation and sensitisation towards these substances. These cases illustrate the potential for severe adverse effects with Sanichlor, which is still in use as a disinfectant for tonometer heads in some eye units in the UK. Hydrogen peroxide 3% appears to be a safe and acceptable alternative, while awaiting a reliable and user-friendly form of disposable tonometer heads for use in ophthalmology.

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Sir,

Central retinal artery occlusion following staphylococcal orbital cellulitis
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Visual loss following orbital cellulitis can still occur despite prompt diagnosis and management. The development of retinal and/or ciliary artery occlusion following orbital cellulitis has not been well documented.¹⁻³ We report a case of rapidly progressive staphylococcal orbital cellulitis resulting in a central retinal artery occlusion.

Case report

A 51-year-old Indian male presented to the eye department with a 24-h history of progressive left periorbital pain and swelling accompanied by fever and chills. His right visual acuity was 6/4 and left visual acuity was hand movements. Examination of the left eye revealed a pustular lesion at the inner aspect of the upper lid with periorbital swelling, complete ptosis, marked proptosis and haemorrhagic chemosis (Figure 1a). Anterior segment examination demonstrated corneal oedema (Figure 1b). Fundus examination showed patchy filling of the tributaries of the central retinal artery and vein, superficial retinal opacification at the posterior pole and the absence of a cherry red spot at the macula (Figure 1c).

Intravenous acetazolamide, ocular massage and anterior chamber paracentesis were unsuccessful at restoring retinal perfusion. His left visual acuity deteriorated to no perception of light. Magnetic resonance imaging and computerized tomography demonstrated soft tissue density infiltrate extending

Figure 1 (a) Pustular lesion at the inner aspect of the left upper lid with periorbital swelling, complete ptosis, marked proptosis and haemorrhagic chemosis. (b) Corneal oedema with folds in Descemet's membrane. (c) Thickened pale retina most prominent at the macula with patchy filling of the tributaries of the central retinal artery and vein with cattletracking in places. (d) Fundus fluorescein angiography (1 week after presentation) shows the appearance of a central retinal artery occlusion, predominantly affecting the lower half of the retina. The upper half of the retina fills incompletely.

anteriorly around the left globe with no sub-periosteal abscess, clear sinuses and no intracranial involvement. Both swab and fine-needle aspiration taken for the pustular lesion at the inner aspect of the left upper lid grew *Staphylococcus aureus* and his orbital cellulitis responded to intravenous flucloxacillin and metronidazole. Full blood count revealed only an increase in neutrophils and monocytes during the acute illness with inflammatory markers returning to normal after the infection had resolved. Immunological and haematological investigations were all normal. His past medical history and review of systems were noncontributory. There was no evidence clinically and from investigation that he was immunocompromised. His fundus fluorescein angiogram 1 week after the event confirmed his retinal vascular occlusion with normal choroidal perfusion (Figure 1d). Six weeks later, fundal examination did not demonstrate disturbances in the retinal pigment epithelium consistent with choroidal ischaemia.

Comment

Acute arterial occlusion is an unusual but known complication of orbital cellulitis.^{2,4} It has been

demonstrated that following orbital inflammation, occlusion may occur at the level of the central retinal artery² or occasionally at the ophthalmic artery.⁴⁻⁶ In central retinal artery occlusion, there is typically a cherry red spot at the macula.^{1,2} In our case, there was retinal whitening at the posterior pole, which is usually seen in ophthalmic artery occlusion.^{1,4} Interestingly, the fluorescein angiogram, which was carried out 1 week after the event, demonstrated central retinal artery occlusion but the choroidal perfusion was deemed to be normal. This was supported by a fundal appearance 6 weeks later showing no evidence of previous choroidal ischaemia. It seems that the clinical appearance of a white posterior pole may not always signify ophthalmic artery occlusion.

The origin of the infection was believed to be a pustular lesion on the inner aspect of the upper lid which grew *Staphylococcus aureus*. This is a known cause of orbital cellulitis, but its association with arterial occlusion, to the best of our knowledge, has not been documented.

In conclusion, we report a case of rapidly progressive staphylococcal orbital cellulitis in a healthy middle-aged male resulting in the rare complication of central retinal artery occlusion.

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