There are many causes of lens and capsule pigmentation including drugs (chlorpromazine, chloroquine)[5] and metals (copper, iron, silver, gold),[6] but our patient had no history of ingestion of these compounds and had no other ocular signs of either siderosis or chalcosis. Axial pigmentary stippling as described by Vogt[7] also has to be considered in the differential diagnosis but has a different appearance to that seen in our patient with the pigment deposits being much finer.

It is difficult to postulate a mechanism by which DMSO could cause pigmented deposits in the lens. Histological examination of animal eyes reveals that DMSO itself does not accumulate in the lens although significant concentrations can be found in cornea, aqueous, vitreous and sclera.[8] It could be postulated that metabolic products of DMSO rather than DMSO itself are responsible. The major biochemical changes reported in DMSO-affected lenses are decreased concentrations of urea, uric acid, glutathione and amino acids, with an increase in albuminoids.[9] DMSO has been reported to cause a loss of gamma-crystallin and an increase in water-insoluble protein in the lens.[10] None of these changes, however, could be expected to lead to pigmentary changes. We cannot comment on how the plasma level of DMSO in the animals that developed lens changes compares with that in humans receiving bladder instillations, as neither the maufacturer nor we could find evidence of these data.

To date, many thousands of patients have been treated with DMSO bladder instillations and the Medicines Control Agency has received five reports of eye disorders: two 'abnormal vision', one myopia and two blurred vision (personal communication with Britannia Pharmaceuticals). Whilst we have no definite mechanism as to the cause of the pigment deposits, we believe they should be looked for when screening patients using DMSO bladder instillations.

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Sir,

Corneal burn secondary to amyl nitrite

Amyl nitrite is a volatile vasodilator and is often abused by inhalation to produce a 'high'.[1] Standard toxicology advice at present states that only transient superficial injuries are to be expected following splash contamination to the eyes. Reports of such injuries are rare.[2,3] We report an unusual case of a moderate corneal burn following exposure to the liquid form of amyl nitrite.

Case report

A 37-year-old woman presented following a splash injury to her eyes with the liquid form of amyl nitrite 11 h previously. She irrigated her eyes several hours after the initial injury before attending casualty complaining of stinging and decreased visual acuity particularly affecting the right eye. There was no other ocular or medical history of note.

Despite having a neutral pH on presentation, she was further irrigated with at least 2 l of normal saline. Subsequent examination revealed a visual acuity (VA) of 6/24 improving to 6/12 with pinhole (PH) in the right eye (RE) and 6/12 improving to 6/9 with PH in the left eye (LE). There was marked erythema and oedema of her right eyelids. Anterior segment examination of the RE showed 270° of perilimbal ischaemia with conjunctival chemosis and nasal conjunctival epithelial loss. There

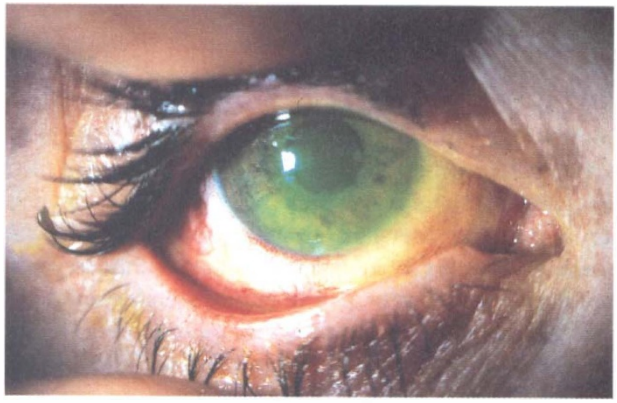

Fig. 1. *Anterior segment photograph of the right eye showing marked perilimbal ischaemia.*

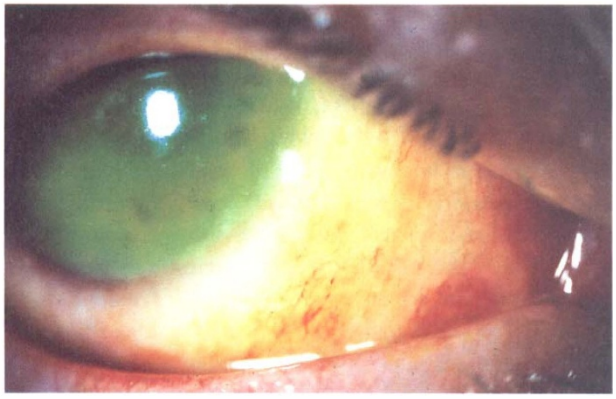

Fig. 2. *Magnified anterior segment view of the right eye showing perilimbal ischaemia and peripheral corneal sloughing nasally.*

was total loss of corneal epithelium and peripheral corneal sloughing nasally (Figs. 1, 2). The LE showed only mild superficial punctate keratopathy. Iris details were still visible in both eyes. There was no anterior chamber activity and intraocular pressures were within normal limits. Fundal examination was unremarkable. A diagnosis of a moderate chemical burn was made and this was managed with topical predsol 0.5%, chloramphenicol and cyclopentolate 1% drops in the first instance. Topical ascorbate was added the following day and the predsol drops were tapered and then stopped after 7 days to prevent inhibition of corneal re-epithelialisation.

Two days later, the patient was symptomatically better, VA was 6/36 improving to 6/12 PH in the RE and unchanged in the LE. Anterior segment examination of the RE revealed decreased conjunctival chemosis but at least 180° of perilimbal ischaemia. There was evidence of epithelial regeneration with a healing edge advancing from the temporal aspect of the cornea. Examination of the LE showed some residual superficial punctate lesions.

One week later, examination of the left eye was unremarkable and there was almost complete healing of the corneal epithelium in the right eye with evidence of limbal revascularisation. Over the following 2 weeks, the patients' vision had returned to normal and there was no residual perilimbal ischaemia or any evidence of an ocular surface abnormality.

Comment

Amyl and butyl nitrites are widely abused by inhalation as aphrodisiacs. They are thought to heighten sexual stimulation and produce a 'high' and are sold in liquid form as room odorisers, or in small glass ampoules known as 'poppers'.[4,5] In external contact with the eye, amyl nitrite vapour is known to induce stinging and transient lacrimation. Liquid amyl nitrate tested on normal rabbit eyes has been shown to cause only slight superficial injury. There is only one previous reference reported in 1913 regarding severe corneal injury where the liquid form of amyl nitrite was involved.[2] The damage was thought to be due to the decomposition of amyl nitrite to nitric acid. This may be the mechanism in our patient although this is impossible to confirm as the bottle was empty on presentation. Another possibility is that the damage may be due to contaminants, which are not uncommon when dealing with substances of abuse.

It is interesting to note that limbal ischaemia can be a consequence of exposure to many different types of chemical agent. This is thought to occur secondary to the degree of necrosis suffered by the ocular tissues during the contact period with the chemical. Deeper penetrating agents such as alkalis cause more necrosis and as a result a greater likelihood of limbal ischaemia, often despite copious irrigation.[6]

Schirner *et al.*[7] used electron microscopy and energy-dispersive X-ray analysis to look at conjunctival tissue in severe eye burns and showed traumatic contamination with calcium in calcium hydroxide burns. This was to be expected, but they also showed that in other types of burn there was particulate contamination with other metals such as iron, aluminium, nickel and zinc. These were thought to produce a prolonged inflammatory response in the eyes involved.

This case highlights the fact that severe injuries to the eye can occur secondary to amyl nitrate and/or its contaminants when there is direct ocular contact. Patients should be advised to irrigate their eyes immediately and attend a casualty as a matter of urgency.

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