Sir,

Mistaken eye drops and subsequent instillation of superglue

A 60-year-old man presented himself to the casualty department after accidentally instilling superglue into his eyes. He had traditionally put eye drops in himself in the evening. On this occasion he had mistaken his wife's fingernail glue for the eye drops as it stood on the bedside cabinet. The bottles were very similar in reduced light, as both were a dropper design for delivery and the same compact size.

Once the glue, of which the major constituent was cyanoacrylate, contacted his eye it caused immense sudden pain causing him to close his eye more. The glue then set quickly and thus he presented with a permanently closed eye. The upper lid was adherent to the cornea, as when the eye movements were tested the lid moved.

He was followed up and after two consultations he was able to fully open his eye and his vision returned to normal. This outcome was also found in two other studies where 6 and 14 patients were followed up.[1,2]

Suggestions to avoid this type of accident include: childproof bottles,[3] different colours of cap for different drugs, a Braille warning on bottles, vertical ribs on glue bottles to signify a warning, posters in the A and E department, and giving glues a different odour to alert the user. This case adds weight to previous reports indicating that this accident still occurs and is very distressing to the patient, who becomes instantly blind in the injured eye.

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Sir,

Rimexolone-induced intraocular pressure elevation

Topical administration of corticosteroids inhibits the inflammatory response to mechanical, chemical or immunological agents. Their precise mechanism of action is unknown, but the effects of corticosteroids include inhibition of inflammatory mediator production, inhibition of leucocytes and phagocyte migration in the acute inflammatory response, and inhibition of fibroblast proliferation and collagen deposition.[1] Corticosteroids are effective in suppressing the inflammatory process, but are also associated with undesirable side-effects which include the elevation of intraocular pressure (IOP) in susceptible (steroid-responsive) patients. Rimexolone 1% ophthalmic suspension is a recently developed topical corticosteroid with effective anti-inflammatory properties as well as a reduced risk of increased IOP.[2,3] We present a case report of a patient with markedly elevated IOP associated with the use of 1% rimexolone suspension.

Case report

A 52-year-old woman with a history of toxic epidermal necrolysis has been attending our institution since 1992. As a result of her condition she developed dry eyes which required punctal occlusion and eye lubricants, including autologous serum drops. She had marked keratinisation of the tarsal conjunctiva especially in the left eye, which necessitated mucous membrane grafting. Her condition was further complicated by metaplastic eyelashes, which were treated with cryotherapy. She subsequently developed a corneal ulcer in the left eye which persisted for a long period despite aggressive treatment. During the healing stage of the ulcer, 0.5% prednisolone sodium phosphate (Predsol) three times daily was commenced. The baseline IOP was 14 mmHg in the right eye and 12 mmHg in the left, and gonioscopy revealed open angles. During the course of her treatment she was found to be a steroid responder. The baseline IOP increased by 16–20 mmHg with Predsol three times daily. Because of the need to continue using topical corticosteroids, she was instructed to use timolol 0.5% twice daily. After a few months of treatment the IOP started increasing again. Fundoscopy revealed glaucomatous optic disc cupping in the left eye only, and perimetry demonstrated glaucomatous field defect. Latanoprost was added to her treatment, which helped in reducing the IOP.

When rimexolone 1% suspension became available, Predsol was discontinued and she was asked to use rimexolone three times daily instead. Before initiating rimexolone, the IOP was 26 mmHg in the left eye. Two weeks later the IOP went down to 14 mmHg. The IOP was maintained within the normal range until about 3 months later when it was found to be 42 mmHg. Rimexolone was stopped and on follow-up examination 1 week later the IOP returned to 15 mmHg. Rimexolone was reintroduced 3 weeks later and again the IOP increased to 40 mmHg, but on this occasion the rise occurred within 2 weeks of treatment. Rimexolone was discontinued, and the IOP went down to 14 mmHg after 1 week. The IOP in the right eye (which did not receive any corticosteroid) remained normal and fundoscopy showed a healthy disc throughout her clinic visits.

Comment

The relatively new ophthalmic preparation, rimexolone, is a synthetic non-fluorinated corticosteroid. Structurally, it contains a methyl group at the C-21 position. Omission of the hydroxyl group at C-21 and substitution of a

methyl group may reduce the propensity for inducing increased IOP, as demonstrated with fluorometholone and medrysone.[1]

Topical corticosteroids are frequently used in everyday practice by the ophthalmologist. Although they are effective in suppressing the inflammatory response, corticosteroids should be used with caution because of their undesirable side-effects. Elevation of the IOP is a relatively common side-effect encountered with topical administration, especially in patients with primary open angle glaucoma, diabetes mellitus and myopia.[1] It is due to reduced ocular outflow facility. Johnson *et al.*[4] studied the ultrastructure of the trabecular meshwork in eyes with corticosteroid-induced ocular hypertension. Their findings were consistent with previous reports of the accumulation of the characteristic extracellular fingerprint-like material, resembling basement membrane, in the trabecular meshwork.[5]

The results from two multicentre clinical studies in patients with anterior uveitis indicated that rimexolone 1% is as effective as prednisolone acetate 1% in reducing inflammation, as determined by a reduction in baseline anterior chamber flare and cells.[2] Leibowitz *et al.*[3] compared the IOP-elevating potential of rimexolone 1% and 0.1% fluorometholone in patients who were known steroid responders. The results showed that rimexolone has a low IOP-elevating potential, comparable to that of fluorometholone. When compared with 0.1% dexamethasone sodium phosphate and 1.0% prednisolone acetate, rimexolone 1% produced a clinically significant lower elevation in IOP, as well as a longer interval before the responses.

On examination of our patient there was no evidence of secondary glaucoma and gonioscopy showed open angles. Since she has been confirmed to be steroid-responsive and experienced elevation of IOP in the left eye only, it is reasonable to conclude that the IOP elevation was due to rimexolone. We feel that clinicians should be made aware of this complication in view of the marked elevation in IOP and the hazardous effects that can follow.

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Sir,

Posterior segment changes secondary to late yaws

Yaws is a contagious, non-venereally transmitted disease caused by the *Treponema pallidum* subspecies *pertenue*. It is characterised by a relapsing clinical course with involvement of bone, skin and mucous membranes. Whether or not any neurological and ophthalmic involvement occurs in the teritary stage of yaws is still being debated in the literature.

Most review articles and textbooks are of the view that yaws does not involve the nervous system or eyes.[1] However, other reports which have specifically looked at the clinical associations of late yaws disagree. A study of a large group of patients with late yaws in Venezuela showed ocular signs including disc pallor, perivascular sheathing, optic atrophy and both acute and chronic inflammatory retinopathies.[2] In addition, a single case report in the literature documents a man with optic atrophy which was attributed to late yaws.[3]

Yaws used to be one of the most common tropical diseases. In the 1950s an estimated 50–150 million people were thought to be affected worldwide, the largest reservoir being Central Africa.[1] However, a series of successful health campaigns by the World Health Organization and UNICEF between 1954 to 1963 meant that yaws was considered to be almost extinct.[4] Yet since the early 1980s, several countries have started to witness a resurgence of yaws.[5] Recently, several cases of imported yaws and endemic syphilis have been described in Europe. In an era of increasing world travel, the diagnosis of non-venereal treponematoses must therefore not be forgotten, given an appropriate clinical situation.[1]

Case report

A 54-year-old woman presented with a 3 month history of bilateral, progressive visual loss. This patient was of African origin, having spent her early years in Zimbabwe, before emigrating to Europe at the age of 20.

Although she presented with a complex social history, she had not been sexually active in recent years. On examination, she was found to have bilateral anterior uveitis, papillitis and gross macula oedema (serous detachment). No vitritis was present and there was no other retinal vasculitis in conjunction with the papillitis. In addition, it was noted that there were no acute retinal haemorrhages present.