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CORRESPONDENCE

Sir,

Acute irreversible corticol cataracts in prolonged topical corticosteriod overuse for chronic eczema

We report a unique case of acute irreversible cortical cataracts associated with chronic overuse of topical (dermal) corticosteroids in eczema.

Case report

A 35-year-old gentleman presented with bilateral blurring of vision over 3 weeks. Visual acuity (VA) was 6/9 OD 6/18 OS. Slit-lamp biomicroscopy revealed bilateral cortical cataracts (Figure 1) and no signs of previous uveitis. Intraocular pressure (IOP) were 14 mmHg OD 18 mmHg OS, and Posterior segments unremarkable.

Two weeks later, he returned with worsening of his vision. VA had deteriorated to 6/12 OD 6/24 OS with shallow anterior chambers. IOP increased to 18 mmHg OD and 24 mmHg OS. Gonioscopy showed bilateral narrowed angles. Further questioning revealed a background of chronic facial eczema (Figure 1), from age 13, and treatment with hydrocortisone 1% cream tid, on most days over the past 20 years. Treatment was confined to his face as he had only mild eczema elsewhere. There was no previous use of oral, ocular or inhaled steroids, and no history of diabetes or trauma. He was systemically well and blood tests revealed normal inflammatory markers and random blood glucose levels of 4.5 mmol/l.

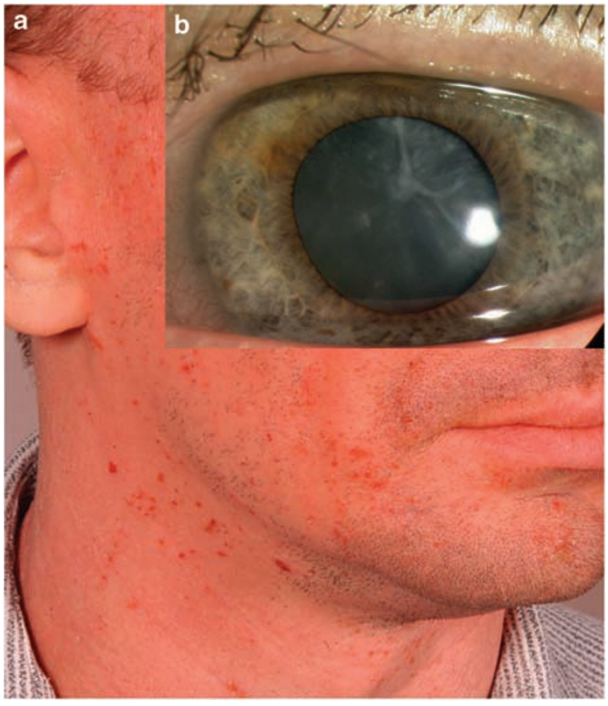

Figure 1 (a) Colour photo showing lichenification of facial skin from prolonged rubbing and scratching secondary to chronic eczema. (b) Colour photo of an acutely swollen lens, cortical cataract, and narrowed anterior chamber.

This gentleman proceeded to have uneventful bilateral sequential cataract surgery, with IOP normalization, attaining VA 6/5 in both eyes.

Comment

Acute irreversible cortical cataracts have previously been associated with Type 1 diabetic patients,[1] but not topical (dermal) steroid use.

Long-term systemic steroid use is a well-known cataractogenic factor. Percutaneous absorption of corticosteroids has been shown by the measured rise of serum cortisol levels.[2] Absorption is enhanced by the increase in skin permeability in corticosteroid-treated skin or chronic inflammation, and plasma levels rise according to the frequency of application.[3] Although topical (ocular) steroid have been shown to penetrate the aqueous humour, there is no direct evidence that dermal steroids raises plasma cortisol levels sufficiently to induce cataracts.

Proposed mechanisms for corticosteroid-induced cataract are dominated by those governing lens osmolytic processes. Corticosteriods inhibit the $Na+$ $K+$ ATPase pump, which actively transports sodium ions across the cell membranes of the lens, allowing water to follow passively.[4] Disruption of this process results in swollen lens fibres, which eventually rupture, causing irreversible opacification. Sorbitol accumulation, inhibition of glucose-6-phosphate dehydrogenase and formation of disulfide-linked protein aggregates have also been implicated.[5]

Patients should be cautioned against overuse of dermal corticosteroids, and warned of potential systemic absorption and possible ocular side effects.

References

1 Florkowski AR, Golden R, Sooch Y, Krishna R. Sudden bilateral visual loss in a diabetic man. *Am J Med Sci* 2005; **329**(2): 99–101.
2 Turpeinen M. Influence of age and severity of dermatitis on the percutaneous absorption of hydrocortisone in children. *Br J Dermatol* 1988; **118**(4): 517–522.
3 Pellanda C, Ottiker E, Strub C, Figueiredo V, Rufli T, Imanidis G *et al*. Topical bioavailability of triamcinolone acetonide: effect of dose and application frequency. *Arch Dermatol Res* 2006; **298**(5): 221–230.
4 Mayman CI, Miller D, Tijerina ML. *In Vitro* production of steroid caratact in bovine lens: part II, measurement of sodium–potasium adenosine triphosphatase activity. *Acta Ophthalmologica* 1979; **57**: 1107–1116.
5 Saether O, Risa O, Cejková J, Krane J, Midelfart A. High-resolution magic angle spinning 1H NMR spectroscopy of metabolic changes in rabbit lens after treatment with dexamethasone combined with UVB exposure. *Graefes Arch Clin Exp Ophthalmol*. 2004; **242**(12): 1000–1007.

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