Comment

The differential diagnosis of a ciliary body mass ranges from congenital to acquired lesions, such as melanoma or leiomyoma.¹ In this case the histopathology¹ and immunohistochemistry³ were consistent with the diagnosis of a NPCE adenoma.¹ There is no evidence to suggest malignant transformation occurred, as there was no growth of the tumour over 1 year of follow-up. Therefore, the presence of rubeosis iridis was attributed to the NPCE adenoma.

NPCE adenoma has been shown to be associated with elevated levels of VEGF in both aqueous and vitreous humour.⁴ This was the underlying cause for optic disc neovascularisation and cystoid macular oedema in one case.⁴ In our case, the fundus was not affected but the presumed increased VEGF production resulted in rubeosis iridis.

Plaque brachytherapy is used for the successful treatment of retinal mass lesions with neovascular potential, such as vasoproliferative tumours⁵ and retinal capillary haemangiomas. Intra-cameral anti-VEGF injections are used for the management of rubeosis iridis. This combination treatment is effective in controlling the vasculogenic response of a NPCE adenoma.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Acquired lateral upper lid entropion in a child treated with Botulinum toxin

Acquired entropion in children may happen as a result of ocular scarring following trauma, infection or surgery. Upper lid entropion in children is rare; Botulinum toxin injection has been suggested in literature to correct congenital entropion. Here we report for the first time a case of acquired lateral upper lid entropion in a child who was treated with a single injection of Dysport Botulinum toxin.

Case report

A 30-month-old Caucasian child presented with a 2-week history of eye rubbing and distress. No previous problems were reported. The lashes at the lateral end of the right upper lid were inverted (Figure 1). The lid was easily everted by lateral traction. His parents were instructed to reposition the lid. Unfortunately, the lateral entropion quickly recurred and a week later he was no better. His parents were very keen to avoid any surgery.

Examination under anaesthetic was unremarkable; there was no foreign body or conjunctival scarring. The lid was repositioned and a single injection of 10 U of Dysport Botulinum toxin was given into the lateral part of the pretarsal orbicularis oculi (Figure 2).

The entropion resolved. He has been followed-up for 15 months with only very, very occasional recurrence and that was managed with traction only.

Comment

Both congenital and acquired entropion occur in children. Orbicularis oculi muscle hypertrophy, orbicularis spasm, epiblepharon, and disinsertion of the lower lid retractors are suggested aetiologies of congenital entropion.¹ Acquired entropion is attributed to ocular infection, scarring, previous surgery, and trauma.²

Upper lid entropion in children is rare. An Asian study reported successful surgical repair of upper lid entropion in 11 children.³ Christiansen *et al*⁴ reported

Figure 1 Right eye upper lid entropion.

Figure 2 Injection of Botulinum toxin into the pretarsal orbicularis oculi.

a case of congenital lower lid entropion in a 3-week old baby that responded to 5 U injection of Botulinum toxin, although the type of Botulinum toxin was not stated.

In our case the upper lid entropion did not recur after the Botulinum toxin worn off. We suggest two possible mechanisms, either the cycle of orbicularis spasm due to irritation from the entropion was broken by the treatment or that facial growth changed the balance of the inverting effect of the orbicularis over the stability of the posterior lamellae. Children with epiblepharon commonly improve as the midface grows during childhood.⁵

To our knowledge this is the first reported case of acquired lateral upper lid entropion in a child treated with Botulinum toxin. We suggest that this treatment could be used in children with entropion, where there is no obvious underlying cause.

Conflict of interest

The authors declare no conflict of interest.

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Sir,
Systemic toxicity of topical cyclopentolate eyedrops in a child

Topically applied ophthalmic medications may sometimes get absorbed into the systemic circulation and lead to significant toxicity. We, herein, report a case of systemic toxicity of topical cyclopentolate eyedrops in a young child.

Case report

A 5-year-old boy presented to paediatric OPD with complaint of progressively diminishing distant vision since 2 years of age. He had normal general physical and systemic examination. Ocular movements and light and accommodation reflexes were bilaterally normal. For fundus examination, he was advised to instill 1% cyclopentolate eyedrops, two drops in each eye, three times at 15 min intervals. However, the father inadvertently instilled it five to six times over 2 h. After another 1 h, he reported again to our OPD with altered behaviour, visual hallucinations, and difficulty in walking. He was disoriented, with ataxic gait and slurred speech. Pupils were widely dilated and fixed (effect of cyclopentolate). Rest of the examination was unremarkable.

Based on these findings and their temporal relation to overdose of cyclopentolate eyedrops, a diagnosis of cyclopentolate toxicity was made. His symptoms gradually resolved over the next 6–8 h. He was discharged after 24 h of observation.

Comment

Cyclopentolate is a synthetic anticholinergic agent and causes rapid-onset cycloplegia.¹ Onset is within 30–60 min and effects last up to a day.² Cyclopentolate eyedrops pass readily through nasolacrimal duct and are well absorbed locally as well as systemically through conjunctiva and nasal mucosa. Systemic absorption also occurs through oropharynx, digestive system, and skin.^{3,4}

There have been various reports of systemic toxicity following topical application of cyclopentolate eyedrops.^{3–7} Children are especially prone due to lower body weight. Various manifestations in children include flushing, tachycardia, feeding intolerance, seizures, and drowsiness. Behavioural changes and transient psychotic reactions may also occur.⁷ Physostigmine is the antidote of choice as it readily crosses the blood–brain barrier. Commonly used anticholinesterases such as neostigmine, pyridostigmine, and edrophonium do not cross the