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Sir,

Bilateral idiopathic orbital inflammatory syndrome with grossly elevated creatinine kinase levels

Idiopathic orbital inflammatory syndrome (IOIS) is a heterogeneous group of disorders characterized by orbital inflammation without any identifiable cause.[1,2] It commonly affects the EOMs, particularly the lateral rectus,[3] lacrimal gland,[4] and cavernous sinus.[5] Classically it presents with unilateral pain, swelling, proptosis, diplopia, and reduced vision. We report the first case of bilateral IOIS involving all EOMs with an unusually grossly elevated creatinine kinase (CK) level and with a favourable response to steroids.

Case report

A previously healthy 7-year-old girl presented with 10 days of binocular vertical diplopia and bilateral eyelid swelling. Visual acuity (VA) was 6/6 in both eyes. She had a left ptosis, exotropia, and hypertropia. EOM demonstrated the limitation of elevation, adduction, and abduction (Figure 1a). Anterior and posterior segments of both eyes were normal and she was diagnosed with double elevator palsy. A week later, both ptosis and hypertropia had switched to the right. EOM limitation persisted. A week later, she had bilateral periorbital oedema and left proptosis. VA was 6/6 OD and 6/12 OS with a left RAPD. ESR was 69 mm/h, CRP 27 mg/l, and CK 4300 IU/l. There was no hepatosplenomegaly or rash. Muscle strength was MRC grade 5. CT scan showed the enlargement of all EOMs bilaterally (Figure 1b) and MRI confirmed EOM swelling from the muscle origin to insertion (Figure 1c). Acetyl-choline receptor antibodies, anti-mitochondrial antibodies, ANA, dsDNA antibodies, pANCA and cANCA cardiolipin antibodies, TFTs, thyroid antibodies, Rh factor, and ACE were all negative. She was diagnosed with bilateral IOIS and treated with intravenous and then oral steroids with complete clinical recovery. The patient remains well 2 years on.

Comment

To the best of our knowledge, this is the first report of bilateral IOIS in a child with grossly elevated levels of CK. Thus, IOIS should not be excluded on the basis of laterality and age. CK levels should be considered, alongside a high index of suspicion with a low threshold for MRI in order to prevent irreversible visual loss from optic nerve compression.

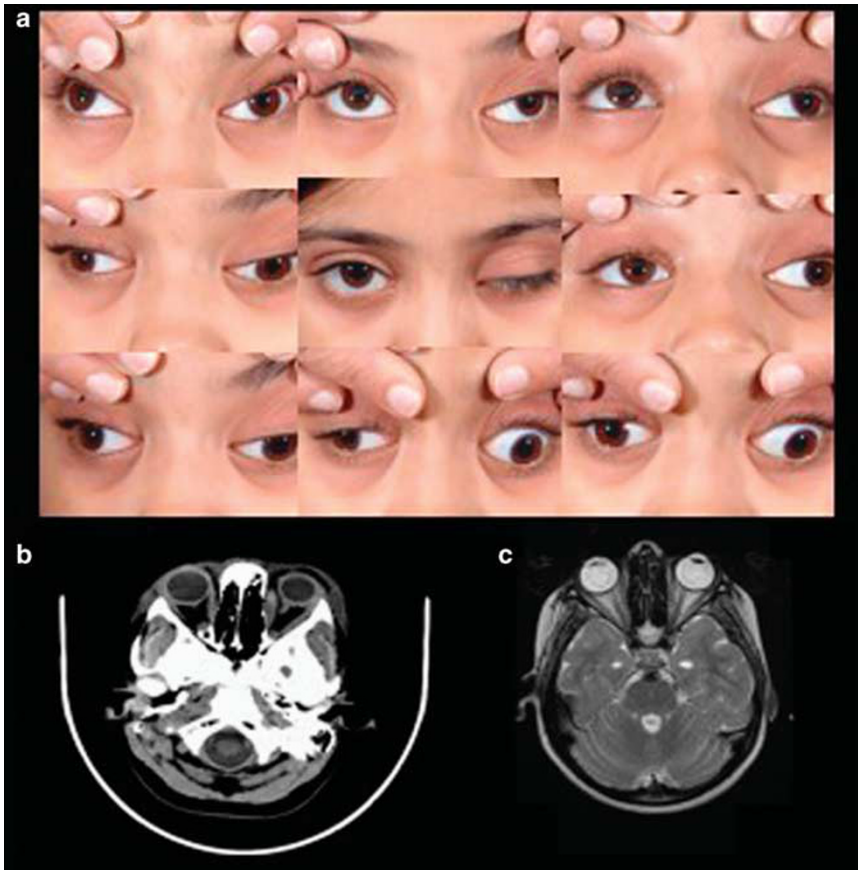

Figure 1 (a) Nine positions of gaze demonstrating the limitation of EOM. (b) CT scan demonstrating enlarged EOM. (c) MRI scan demonstrating enlarged EOM.

Conflict of interest
The authors declare no conflict of interest.

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Sir,

A case of lorazepam (Ativan)-induced accommodation paresis

Sudden loss of accommodation can result from numerous etiologies, including head trauma, encephalitis, oculomotor nerve palsy, uveitis, and viral diseases.[1] Many drugs, such as tricyclic antidepressants and phenothiazines, also cause acute accommodation paresis.[2] However, to our knowledge, lorazepam-induced accommodation paresis has never been reported.

Case report
A 23-year-old man complained of near-sight disturbance. He had been taking lorazepam (Ativan) 1.5 mg/day and fluoxetine 10 mg/day during the past 2 weeks because of depression. At 5 days after taking the medicine, the near-sight disturbance suddenly developed. He visited our hospital 2 weeks after taking the medicine.

Best-corrected visual acuity was 1.0 in both eyes. The anterior segment and posterior segment revealed negative findings. The alternate cover test showed 16 prism diopter exotropia in far and near distance. The near point of accommodation (NPA) was 16 cm in the right eye and 19 cm in the left eye, and sluggish pupillary reflex was noted in both eyes. The near-sight disturbance was relieved by near glasses correction. He stopped taking lorazepam 7 days after the first visit, and the NPA was 14 cm in his right eye and 12 cm in his left eye at 15 days after cessation of lorazepam. He restarted sedative medication with diazepam 4 mg/day, instead of lorazepam. Two months after cessation of lorazepam, NPA was recovered (6 cm in both eyes) and pupillary reflex was normal.

Comment
Lorazepam is a benzodiazepine widely used to treat anxiety disorder and as an amnesic, sedative/hypnotic, anticonvulsant, and muscle relaxant.[3] The common side effects of lorazepam are light-headedness, drowsiness, and daytime tiredness.[4] Speeg-Schatz *et al*[5] studied the effects of lorazepam on visual acuity, binocular vision, and accommodation. The results of their investigation showed that lorazepam had no effect on visual acuity or accommodation, but it impaired oculomotor balance, meaning that it reduced convergence and divergence amplitude without impairing accommodation. In the present case, near-sight disturbance with prolonged NPA was noted and the symptom was relieved after cessation of lorazepam. Thus, lorazepam can induce accommodation insufficiency.

We should consider accommodation paresis when patients who have taken lorazepam complain of near-sight disturbance because short-term usage of lorazepam may induce accommodation paresis.

Conflict of interest
The authors declare no conflict of interest.

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Sir,

Reply to Spiteri Cornish and Reddy

We read the excellent review by Spiteri Cornish and Reddy[1] describing the management of periocular capillary haemangiomas using propranolol and the current available evidence supporting this.

Our experience in Southampton has been very positive. We have a cohort of seven patients (two orbital,