

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.

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Sir,

Reply to Spiteri Cornish and Reddy

We read the excellent review by Spiteri Cornish and Reddy¹ 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,

four periocular, and one brow haemangioma). We used a starting dose of 1 mg/kg per day in three divided doses, increasing it to 2 mg/kg per day after 1 week if tolerated. Six of our patients have responded very well to the treatment and have had no side effects to date. The propranolol was stopped in one case owing to loss of appetite, which coincided with the commencement of propranolol.

One point we wished to make is to raise awareness of the fact that propranolol elixir can be dispensed in four concentrations (5 mg/5 ml, 10 mg/5 ml, 40 mg/5 ml and 50 mg/5 ml). We have had one drug error where the prescription was correctly written, but the dispensing community pharmacist gave the 50 mg/5 ml propranolol. This resulted in the infant being given 10 times the prescribed dose. He was admitted for monitoring with no ill effect. We feel it is important that parents are made aware of the different formulations available so that similar possible errors are prevented.

Conflict of interest

The authors declare no conflict of interest.

Reference

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Sir,

A case of African crystalline maculopathy

We read with great interest the report¹ describing an isolated case of crystalline maculopathy in a North-East African individual from Egypt. West African Crystalline Maculopathy (WACM) has only been seen in a single population isolate to date (The Yoruba and Igbo tribes of West Africa), making a genetic predisposition likely. While the genetic makeup of Egyptians is interesting, genetically, they are very different to the West Africans; a combination of geographical location and history of modern and ancient migration have resulted in a complex and diverse gene pool of European, Middle Eastern, and African genetic characteristics.²

The central foveal crystalline deposits in this case are indeed interesting; however, we believe it differs from the described features of WACM. Firstly, the appearance and distribution of the crystals are different from that seen with published cases of WACM. Our previously published series, as well as of other authors have shown WACM to consist of intra-retinal crystals (Figure 1),^{3–5} whereas, in this case, the crystals appear very localised to the inner retina at the foveola. Commonly in WACM, there is also concurrent co-pathology that affects the blood-retinal barrier such as diabetes.

The authors have described and excluded other causes of crystalline deposits in this report; therefore, although we believe this case does not exhibit the recognised changes seen in WACM, it could be a different phenotype. However, it is difficult to make further comment from just a single index case. We now have accumulated an unpublished case series of over 50 patients with WACM, who all, without exception, originate from West Africa, and exhibit the described features. It is premature to consider a new label for this condition from just a single albeit interesting case.

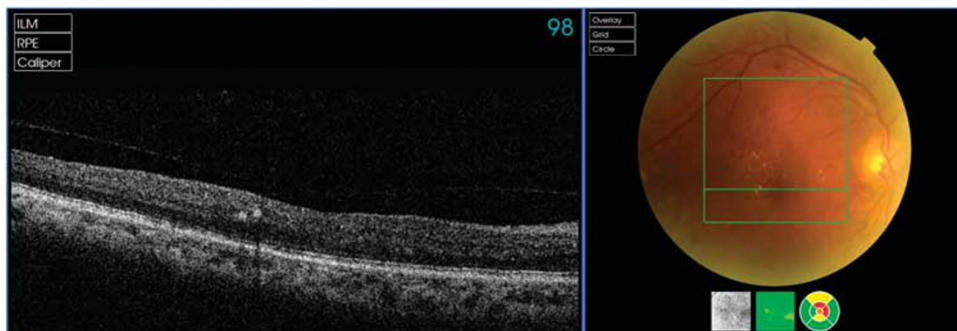


Figure 1 Fundus photograph demonstrating West African Crystalline Maculopathy, with OCT image showing retinal localisation of crystals.