corrected visual acuity was 20/25 and the angles were equally open OU with light pigmentation OD and moderate pigmentation OS, and without synechiae OS, but the IOP was 26 mm Hg OS on Timolol.

Comment

We found two reports of ectopia lentis in patients with SWS,^{2,3} one of which may have had traumatic lens dislocation.³ Glaucoma in SWS is typically similar to congenital glaucoma in cases presenting from birth to 2 years, or is due to increased episcleral venous pressure later in life.¹ ACG has been reported in patients with ectopia lentis,^{4,5} and some authors advocate lensectomy in the management of ACG associated with ectopia lentis.⁵ In our patient, lensectomy widened the angle but did not resolve the IOP elevation. Pigment dispersion may have occurred because of friction between the subluxed lens and the iris pigment epithelium, or intermittent pupillary block. Our patient had SWS associated with ectopia lentis that caused both ACG and pigment dispersion; these findings are rare ocular complications of SWS.

Conflict of interest

The authors declare no conflict of interest.

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Sir, Idiopathic uveal effusion syndrome causing unilateral acute angle closure in a pseudophakic patient

Pseudophakic angle closure is a rare occurrence. Most frequently it occurs secondary to pupil block with iris bombe, capsular bag distention syndrome, or aqueous misdirection, with an anterior shift of the iris-lens diaphragm. We present a case of acute angle closure in a

Figure 1 Van Herrick examination of the right eye showing iridocorneal contact (a); in the left eye anterior chamber depth is >25% of the corneal thickness (b). On gonioscopy, no angle structures were visible in the right eye (c). There was a normal configuration in the left eye (d).

pseudophakic patient without pupil block caused by an idiopathic uveal effusion syndrome (IUES).

Case report

A pseudophakic 65-year-old lady presented with a 1-day history of pain around the right eye approximately 2 years after uncomplicated cataract surgery. Examination revealed a visual acuity of 6/9 and intraocular pressure (IOP) of 32 mm Hg. The anterior chamber was quiet with no posterior synechiae or axial shallowing. Peripheral iridocorneal contact was visible. Gonioscopy showed 360° angle closure (Figure 1). The IOL was in the bag, with the posterior capsule apposed to its posterior surface. Fundal examination showed folds in the choroid without serous retinal detachment. The patient was normotensive at presentation and not on antihypertensives. An ultrasound B-scan and OCT confirmed annular choroidal effusions and angle closure (Figure 2). There was no evidence of posterior scleritis, scleral thickening, or choroidal masses. The patient was not nanophthalmic and a CT scan of the orbits showed no masses, myositis, or vascular malformations. She had a

background of diabetes and epilepsy. There were no recent changes to her medications that comprised metformin 500 mg BD, pregabalin 50 mg TDS, repaglinide 1 mg BD, and lamotrigine 100 mg BD, none of which is known to cause uveal effusion. She had never taken topiramate. Full blood count, renal, liver and thyroid function tests, autoantibody screening, immunoglobulins, and plasma electrophoresis were unremarkable.

The patient was treated with a single dose of acetazolamide 500 mg, topical atropine 1%, and timolol 0.25%. The drainage angle opened with cycloplegia and the IOP was lowered to 13 mm Hg. Consideration of sclerectomy was made, but the condition fully resolved over the next month without intervention. A fluorescein angiogram performed 2 weeks after presentation showed no evidence of vasculitis.

Comment

Spontaneous uveal effusions are rare and can be caused by inflammation (scleritis/myositis), infection, malignancy, or an idiosyncratic response to drugs (when they are typically bilateral).^{1,2} IUES is a diagnosis of

Figure 2 (a–d) B-scan transverse mode of the right eye highlights an anterior choroidal effusion (arrow) on temporal and nasal gaze. (a, b) Anterior segment OCT demonstrates iridocorneal contact and closed drainage angle in the affected right eye (c) and the unaffected left eye (d).

exclusion and typically occurs in middle-aged men. The pathogenesis remains controversial and is thought to be related to abnormalities in the sclera resulting in albumin accumulation.³ To our knowledge unilateral pseudophakic acute angle closure secondary to IUES has not been reported. Pseudophakic angle closure is rare and can be caused by a variety of different mechanisms.⁴ Careful assessment of peripheral iris configuration and symmetry of anterior chamber depth point to the diagnosis.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Association between intraocular pressure and adherence: is there one?

The paper by Ajit *et al*¹ that was recently published in *Eye* described a new methodology of graphically presenting adherence data, using an electronic dosing monitor. The concept of using such a device to provide meaningful, graphical information is potentially favourable to the clinician in identifying patterns of adherence. However, the idea of collecting this information for all patients is probably unrealistic and unlikely to be cost-effective.

Lowering intraocular pressure (IOP) to reduce or halt the progression of visual field loss is the only currently available intervention for patients with glaucoma. Measuring IOP to assess efficacy has been standard practice ever since topical anti-glaucoma therapy was commenced. If a therapeutic regimen is adhered to, a reduction in IOP would be expected on repeat measurement *a priori*. Theoretically, therapeutic outcome would be both an objective and a practical measure of adherence. However, to date there is no consistent

evidence of a relationship between adherence and IOP.² Failure to identify such a relationship could be explained by the lack of a quantified correlation, or could be attributed to the methodological quality of the studies examining such a correlation being poor. However, it is more likely that the complexities of assessing the level of IOP due to individual differences (types of glaucoma and diurnal variance), together with regression to the mean, lead to 'noisy data'.

The methodology used by Ajit *et al*¹ stated that 100 patients at their initial interview had their IOP recorded. However, no further discussion of the IOP data was done in the paper. Assuming that Ajit *et al* had the intention of collecting IOP data to study any relationship between IOP and adherence, it would be interesting if their findings could be published, particularly given the potential value to the glaucoma clinician of learning how IOP measures might correlate with adherence.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Response to Cate and Broadway

We thank you for providing us an opportunity to respond to the letter by Cate and Broadway¹ concerning our paper on patterns of adherence to hypotensive therapy that was recently published in *Eye*.² Our paper tried to address the issue of adherence in a way that was clinically more meaningful than simple percentage figures that are commonly reproduced in the literature. As part of this project we also collected pre- and post-treatment IOPs although we did not report on them within the *Eye* paper.

In their letter Cate and Broadway highlight that the routine use of an electronic dosing monitor is unrealistic (especially so now that Alcon has discontinued the supply of such aids) and asks whether a measure of IOP reduction can be used as a surrogate measure for adherence. We have now undertaken an analysis of pre- and post-treatment IOPs and can report that there is