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The authors have no proprietary interest in any of the materials or techniques used in this study.

Sir,

Use of Pilocarpine following Hyphaema-Related Ocular Hypertension

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With the wide range of antiglaucoma drugs available to us now, the main use of pilocarpine is in the management of primary angle closure glaucoma. It is also useful in paediatric patients with pseudophakic glaucoma or in the short term following goniotomy. In the current literature, pilocarpine is not recommended for the treatment of hyphaema-related ocular hypertension. We have found it to be useful in a number of cases of raised intraocular pressure secondary to hyphaema which were refractory to other forms of treatment.

Case reports

Case 1 A 37-year-old man was admitted with an extensive left hyphaema following trauma to the eye with an exploding cartridge. The intraocular pressure (IOP) was 34 mmHg. He was commenced on oral acetazolamide, apraclonidine, and dorzolamide. He rebled 3 days later and the IOP rose again. It remained high at 30–40 mmHg for 5 days. Latanoprost was added with no effect. On day 6 following the rebleed the IOP was 52 mmHg, so a washout was performed under general anaesthesia (GA). Postoperatively, the IOP was 42 mmHg. Aqueous was released from the paracentesis on four occasions but the pressure rose within hours on each occasion. He remained on maximum medical treatment. After 3 days, pilocarpine 1% q.i.d. was commenced and the IOP fell to 22 mmHg on day one and 10 mmHg on day 2. All medications apart from pilocarpine were discontinued after 1 week. The pilocarpine was discontinued after a further 2 weeks and the IOP remained low. On gonioscopy he was noted to have angle recession with some areas of peripheral anterior syncechiae.

Case 2 A 35-year-old man was admitted with hyphaema and secondary ocular hypertension (OHT) of 52 mmHg following a blow to the eye. He was treated with topical steroid, cycloplegics, apraclonidine, betaxalol, and oral acetazolamide. The IOP returned to normal and the hypotensive treatment was reduced to betaxalol. Following a rebleed on day three, the IOP remained at 30–40 mmHg for 7 days in spite of recommencement of apraclonidine and maximum dose oral acetazolamide. The addition of latanoprost after 2 days had no effect and mannitol on day 6 reduced the IOP for less than 24 h. On day 8 after the rebleed he had a washout under GA. In spite of an initial drop to 10 mmHg the IOP remained high for 3 days (28–40 mmHg). No reduction was made in his IOP-lowering medications apart from discontinuing latanoprost. Pilocarpine 4% qid was added and the IOP fell over the next 3 days to 12 mmHg. All medications apart from pilocarpine were discontinued. The pilocarpine was discontinued 3 weeks later and the pressure remained at 15 mmHg. He was noted to have angle recession when the view of the angle improved sufficiently to allow gonioscopy.

Case 3 A 19-year-old man was admitted with a right hyphaema and IOP of 38 mmHg following a blow to the right eye. He was commenced on topical steroid, cyclopentolate, acetazolamide, and betaxalol which lowered the IOP. He rebled 2 days later and the IOP remained high at 30–40 mmHg for 5 days, in spite of the addition of apraclonidine and latanoprost. We commenced pilocarpine 2% q.i.d. on day 6 and stopped cyclopentolate and latanoprost. The IOP dropped from 38 to 23 mmHg after 2 days on pilocarpine. The other ocular hypotensive medications were discontinued 3 days after starting pilocarpine. The IOP remained low on pilocarpine alone at 12 mmHg after the other medications were discontinued. There was no evidence of angle recession on gonioscopy.

Comment

Current mangement of hyphaema emphasises the importance of cycloplegia, because of the concurrent anterior chamber activity, to break any pupillary block[1] and to allow posterior segment examination as soon as there is a clear view. In the current literature, pilocarpine is not recommended for ocular pressure lowering.[1,2] However, some cases of secondary glaucoma can be refractory to all treatment, including AC washout. In this small series, pilocarpine appeared to lower the IOP where all other forms of treatment failed. Prior to the development of the wide range of antiglaucoma medications that are available today, pilocarpine and acetazolamide were the only agents for treating hyphaema-related OHT.[3] Pilocarpine and homatropine were tried in combination to speed up resorption of the

hyphaema. This increased the rebleed rate, possibly because of excessive pull on the traumatised blood vessel.[4]

Cases 1 and 2 required a washout for persistently high IOP. The IOP remained high for a few days. This may have been because of trabeculitis or the residual erythrocytes within the trabecular meshwork. The use of latanoprost in the early stages of IOP control in these patients may have been counterproductive because of its tendency to weaken the blood aqueous barrier. Pilocarpine drops were commenced to reduce outflow resistance by constricting the pupil and mechanically opening up the trabecular meshwork. It appears that pilocarpine reduced the IOP when conventional treatment failed. In all three patients, the IOP remained low on pilocarpine alone when all other medications were discontinued. We discontinued the other medications at an earlier stage in cases 2 and 3 following our experience of its efficacy. Perhaps the IOP was going to fall anyway, as in the first two cases the AC had been washed out and the delay in reduction of IOP was secondary to residual RBCs within the trabecular meshwork. The outflow resistance may have returned to normal even without pilocarpine 2–3 days following washout. However, the third patient did not have a washout and there was visible macrohyphaema in the AC when the effect of pilocarpine was noted.

Pilocarpine is generally felt to be contraindicated in hyphaema because its miotic effect is undesirable in the presence of active inflammation but the mechanical effect may help lower the IOP. We have found pilocarpine to be effective if the elevated IOP is refractive to conventional treatment in the second or third week. The number of patients is small and there may be other factors influencing the fall in IOP, mainly the timing of the introduction of pilocarpine. It would be necessary to examine the effect of pilocarpine in a greater number of patients to confirm that it is effective in the control of persistent hyphaema-related OHT.

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Sir,

Abnormal lens shape on CT in a patient with Aniridia
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CT is a useful tool in detecting and localizing intraocular and orbital foreign bodies.[1] However, total reliance on scan information may be injudicious. We present a case that illustrates an unusual CT appearance of a congenitally subluxed lens.

Case report

A 40-year-old man was referred to the neuro-ophthalmology department complaining of a subjective reduction in vision. He was diagnosed with aniridia at birth that has an autosomal dominant inheritance within his pedigree.

Visual acuity was CF (counting fingers) RE and HM (hand movements) LE; this had been stable for 2 years. He had peripheral corneal scarring and bilateral subluxed lenses (left more than right). The lens zonules were intact superiorly but stretched and missing inferiorly. There was an anterior cortical and posterior subcapsular cataract in both lenses and the intraocular pressures were 14 mmHg RE, 12 mmHg LE. Fundal examination showed bilateral macular hypoplasia, and both discs were pale. In view of his recent symptoms and disc pallor, a CT scan was arranged.

CT, 2 mm axial sections along the meatoinfraorbital plane through the orbits, showed evidence of bilateral hypoplastic optic nerves. It also showed that the lens shape appeared reversed, that is, the anterior lens surface was more and the posterior lens surface less convex (Figure 1).

Comment

Aniridia (absence of iris) was first described by Barrata in 1818. This panocular disorder is bilateral in 98% of cases, two-thirds are familial (autosomal dominant), one-third sporadic.[2] The sporadic variant is associated with extraocular pathology, for example, Wilms tumour and a high incidence of deletion at 11p13.[3] Glaucoma, cataracts, dislocated lenses (due to a molecular defect of the zonules), corneal defects and optic nerve/macular hypoplasia are commonly found.[4]