was 92 mm/h and C-reactive protein was 96 (range 0–10 mg/l). Repeat imaging showed a frontal lobe abscess (Figure 2c and d) and an intraspinal subdural empyema extending from T12 to S2 (Figure 2e). Pus aspirated from an L4-5 fenestration failed to culture any organisms and blood cultures were also negative. The patient was treated with intravenous cefotaxime (2 g q.d.s.) and metronidazole (500 mg q.d.s.), together with phenytoin prophylaxis.

With 3 weeks of antibiotic therapy, there was a rapid clinical improvement, resolution of the intracerebral abscess, and a significant reduction in her proptosis (Figure 1b) with maintenance of vision. The patient declined the possibility for surgical repair of her craniofacial anomaly.

Comment

Our patient demonstrated clear enlargement of her encephalocoele over the decade under our care, to such a size that it probably obstructed maxillary antral drainage, this causing an infective sinusitis with spread into the neighbouring (abnormal) central nervous system. Since intranasal encephaloceoles are a known predisposition to recurrent meningitis,[3] it is very important to be aware of the association of varices with encephalocoeles.[2]

Surgical management of distensible orbital varices is very difficult and these hamartomas are best left alone unless optic neuropathy, severe cosmetic deformity, or bleeding become a problem. Although some undergo a slow, painless enlargement over years, the acute episode of painful proptosis (because of haemorrhage or thrombosis) would appear to be somewhat more common.

To our knowledge, the occurrence of widespread intracranial infection presenting with painful enlargement of orbital varices has not been previously described. Although rare, this is a potentially fatal condition that can be successfully treated with appropriate antibiotics.

Acknowledgements

Proprietary interest: None.

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BJL Burton[1], N Islam[1], GT Plant[2,3] and GE Rose[1]

[1]Orbital Service, Moorfields Eye Hospital
City Road, London EC1V2PD, UK

[2]Neuro-ophthalmology Department
Moorfields Eye Hospital, London

[3]National Hospital for Neurology and Neurosurgery
Queen Square London, UK

Correspondence: GE Rose
Tel: +44 20 7253 3411x2034
Fax: +44 20 7566 2019
E-mail: Geoff.rose@moorfields.nhs.uk

Sir,

Vertical augmented transposition surgery
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Recently, Foster[1] has described an augmented transposition operation for use in horizontal muscle deviation caused by VI nerve palsy and type 1 Duane syndrome. We have reported the dramatic benefit that can be produced by the procedure.[2] While the possibility of using a similar approach for large vertical deviations has been mentioned,[3] no case reports or series have been published.

We report our experience in using the Foster modification of transposition surgery in a patient with acquired superior rectus palsy.

Case report

An 8-year-old boy was referred to the strabismus service because of a vertical squint. The family reported that recently his right eye tended to drop downwards especially on right gaze, that he adopted a markedly abnormal head posture of chin elevation, and tended to close one eye when reading. There was no past ocular history and his general health prior to presentation had been good. He was not taking medication and there was no relevant family history of ocular problems.

On initial examination, visual acuities were right 6/5 and left 6/4. There was a right hypotropia measuring 20Δ at 1/3 m and 12Δ at 6 m with a 4Δ esotropia. He also had right upper lid retraction and limitation of right eye dextroelevation (Figure 1a). While he attempted to maintain alignment with a head posture, there was evidence of right eye suppression as tested by Bagolini glasses. The remainder of the eye and neurological examination was unremarkable.

MRI, liver function tests, thyroid function tests, full blood count, and plasma viscosity were normal. A diagnosis of isolated superior rectus palsy was made.

He was reviewed at monthly intervals. After 6 months, his deviation had increased to 30Δ right hypotropia at near and distance. Following discussion with the family, surgery was performed despite the changing size of the deviation in the hope of preserving binocular function.

Forced duction test at surgery revealed no restriction. Adjustable 6 mm right inferior rectus recession and 5 mm superior rectus resection were performed. A 2 mm adjustment of the inferior rectus (ie a final recession of 4 mm) was utilised to achieve orthophoria for near and distance.

At 10 days postoperatively, he had developed an 11Δ right hypotropia. At 2 months following surgery, his deviation had returned to the preoperative state of 35Δ right hypotropia.

After 6 months, a stable situation had been reached and a cosmetic procedure was requested. A full muscle transposition of the medial and lateral recti was combined with posterior fixation sutures to the horizontal recti 7 mm posterior to the insertion of the superior rectus muscle.

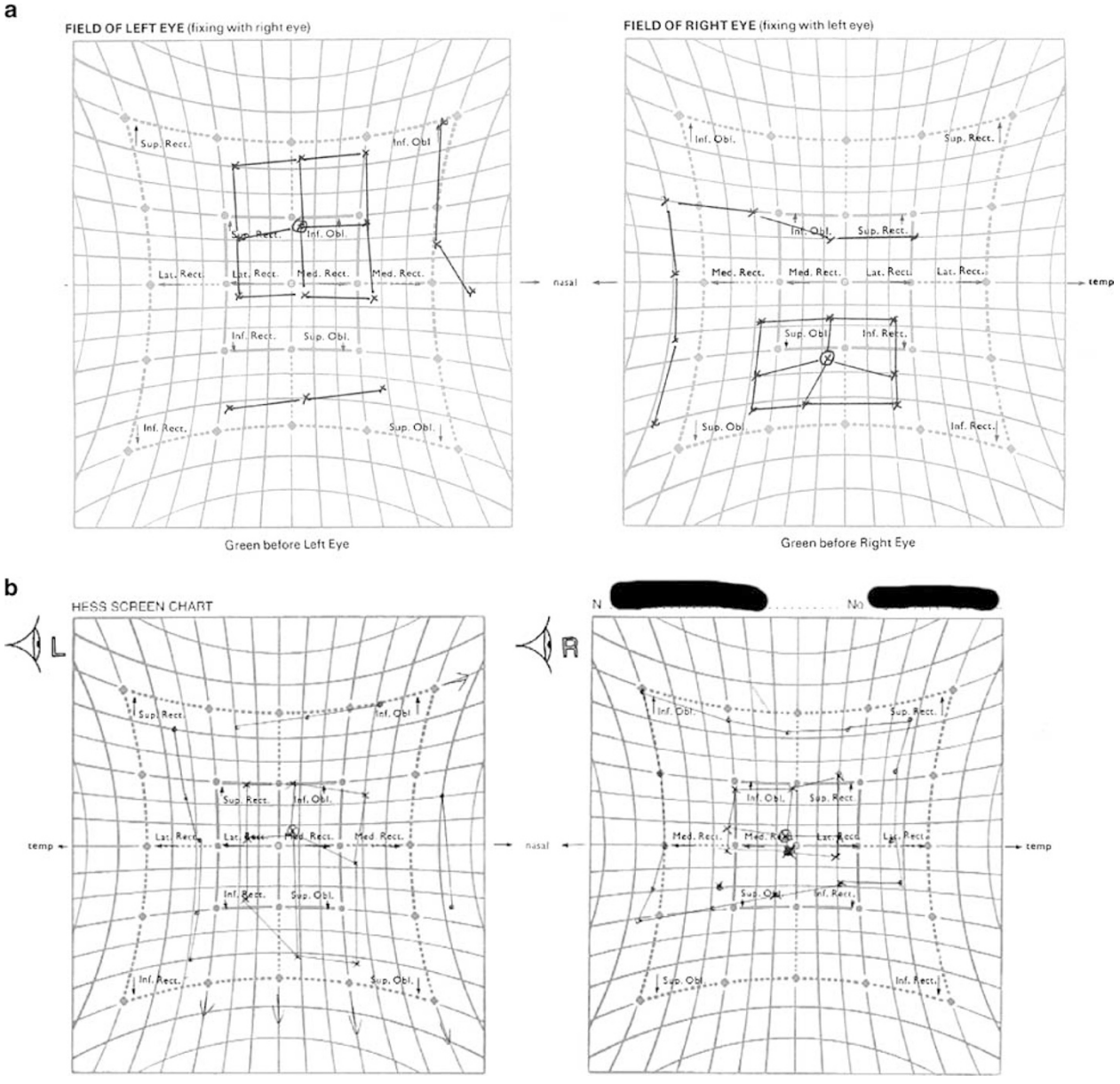

Figure 1 HESS charts: (a) Preoperatively, there is an isolated limitation of superior rectus muscle function. (b) Postoperatively, there is limitation of downgaze in the operated eye. Consequently, there is diplopia in marked downgaze.

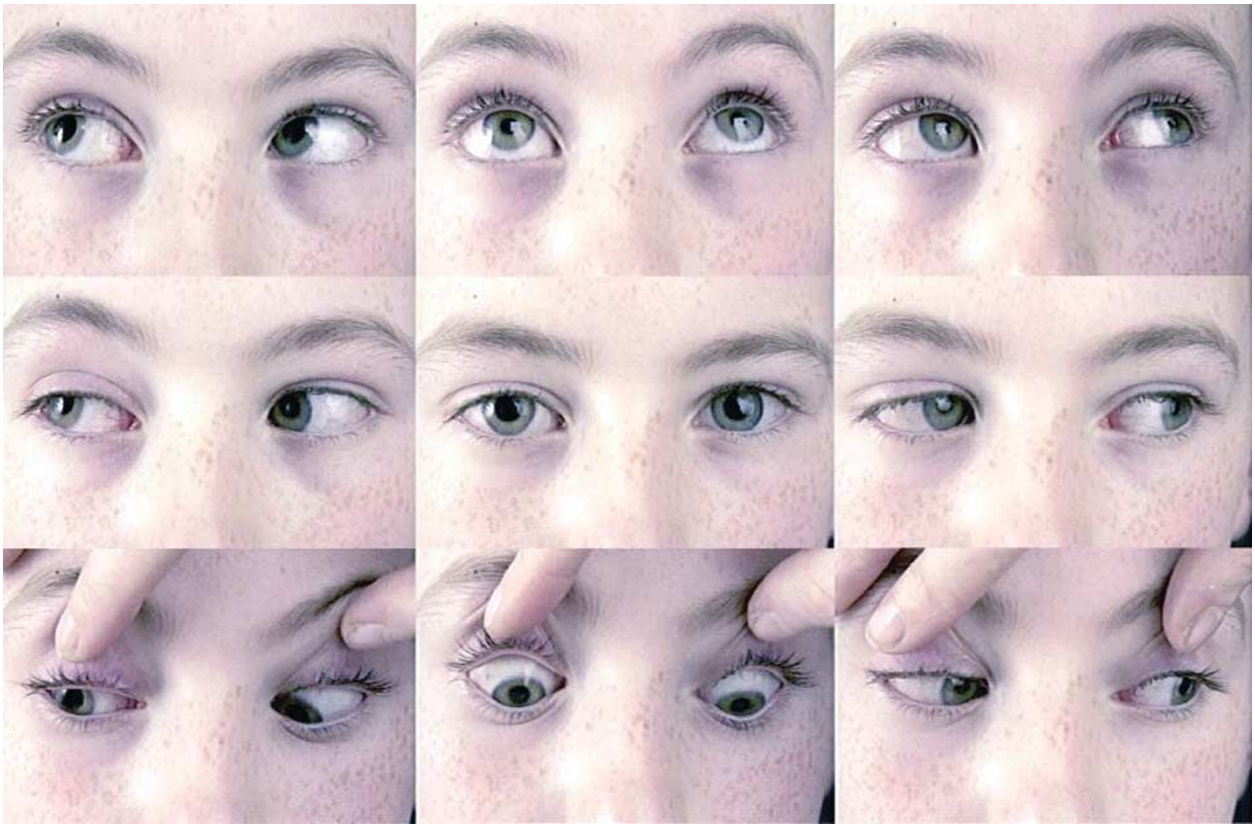

Figure 2 Postoperative alignment in nine positions of gaze.

Postoperatively, a 5Δ right hypotropia was present that drifted to orthophoria over 2 weeks. Stereopsis of 60″ was now present. An initial 4 mm ptosis gradually resolved over the next 3 weeks. Ocular motility has remained unaltered 5 months following surgery. However, there is limitation of right downgaze (Figures 1b and 2).

Comment

Large incomitant vertical deviations present a challenge to the strabismus surgeon, particularly if an attempt is made to preserve binocularity.

The results of conventional transposition surgery are often disappointing.[1,6] The value of a Foster procedure has been clearly demonstrated before in the management of horizontal palsies,[1,2] but there is little information regarding use of the technique for vertical strabismus.

In our case, augmented vertical transposition was used following a return of the deviation after initial standard resection/recession, which may explain the presence of limitation in downgaze postoperatively. It is also possible that the posterior fixation suture may also contribute to the limitation in downgaze postoperatively, although we would need to perform forced duction testing to elucidate this further. In retrospect, use of an augmented transposition procedure at the initial surgery may have been the optimal initial management.

It is possible that this patient may require further surgery for the depression deficit, although children can be remarkably tolerant of such difficulties and currently, he is managing extremely well. Late overcorrection and the risk of anterior segment ischaemia caused by multiple operations on extraocular muscles are two other important considerations.

The HESS chart confirms the clinical finding of isolated superior rectus underaction rather than double elevator palsy (Figure 1a). There is paucity of information on the management of idiopathic superior rectus palsy in the literature.[4,5] Despite this, it is a condition that is encountered occasionally in specialist strabismus practice and, if marked, may be an indication for augmented transposition. However, the technique is still in its infancy and only time and experience will delineate its optimal use.

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SRH Mohamed and JR Ainsworth

Birmingham Childrens Hospital
NHS Trust, Steelhouse Lane
Birmingham B4 6NH

Correspondence: JR Ainsworth
Tel: +44 121 333 9999
E-mail: j.r.ainsworth@bham.ac.uk

Sir,

A non-idiopathic case of polypoidal choroidal vasculopathy

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Idiopathic polypoidal choroidal vasculopathy (PCV) is a condition similar to but distinct from age-related macular degeneration. The known underlying ocular predisposing factors for choroidal neovascularisation are absent. Systemic hypertension is a frequent association. We describe a case of PCV where characteristic clinical and angiographic features were precipitated by about of forceful sneezing.

Case report

A 76-year-old Caucasian lady presented to eye casualty complaining of sudden visual distortion in the left eye following a bout of forceful sneezing. She was hypertensive on medication with a blood pressure of 150/86 mmHg at presentation. She had no relevant ocular history. Her best-corrected Snellen visual acuity was 6/9 in each eye. The anterior segments and intraocular pressures were normal. Fundoscopy had revealed some small soft drusen over both maculae. The left had superficial retinal haemorrhages between the disc and the macula and involving the fovea, with surrounding shallow neurosensory retinal detachment. In addition, there was a slightly elevated reddish-orange colour nodule noted at the peripapillary area of the left disc.

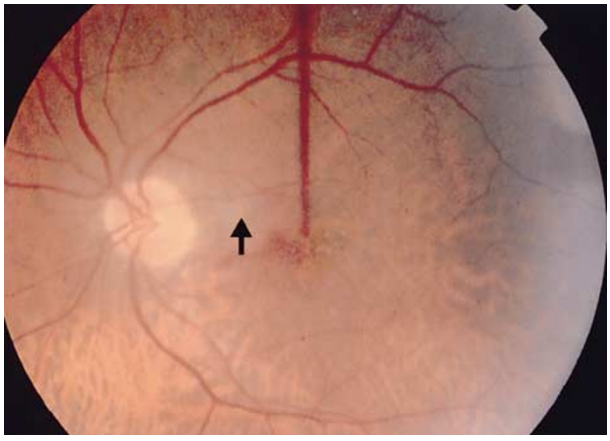

Figure 1 Fundus photograph of the left eye 2 weeks after initial presentation, showing the reddish-orange elevated nodule (arrow) in the peripapillary region with superficial retinal haemorrhages extending towards the fovea. Some soft drusen were also found over the macula.

When reviewed in the specialist clinic 2 weeks later, she reported subjective improvement in her vision to 6/6 in each eye. Previously noted neurosensory retinal detachment had resolved with some residual retinal haemorrhages (Figure 1). The differential diagnosis at this stage was age-related peripapillary choroidal neovascularisation. However, the presence of the visible reddish-orange nodule (Figure 1, arrow) strongly suggested PCV.

Fundus fluorescein (Figure 2a and b) and indocyanine green angiography (Figure 3a and b) performed on the same day had confirmed a single hyperfluorescent lesion (arrows) corresponding to the clinically visible reddish-orange nodule. This lesion was well defined with no sign of active leakage as shown from the late phases of both angiograms (Figure 2b and 3b). There was no evidence of age-related choroidal neovascularisation arising from the soft drusen over the macula area. The diagnosis of PCV was therefore made. Laser treatment was not proposed based on clinical and angiographic findings. At 3 months following the initial presentation, the vision continued to improve to 6/5 in the left eye with complete resolution of the retinal haemorrhages but persistence of the reddish-orange nodule.

Comment

Idiopathic PCV was first described in hypertensive black female subjects with characteristic elevated reddish-orange subretinal peripapillary lesions associated with recurrent haemorrhagic retinal pigment epithelial detachments, retinal or vitreous haemorrhages. [1] More recently, case studies have described similar lesions occurring in the macula, the midperipheral, and