LETTERS TO THE EDITOR

Temporary prism treatment of acute esotropia precipitated by fusion disruption

EDITOR.—A period of temporary fusion disruption may lead to decompensation of a pre-existing heterophoria or may precipitate acute acquired concomitant esotropia.¹ Conventional strabismus surgery is usually necessary to permanently restore binocular single vision (BSV), although botulinum toxin has been advocated.² We describe two cases associated with unusual forms of occlusion induced decompensation who achieved fusion after temporary prismatic correction. Patient 1 underwent surgery and patient 2 avoided surgical correction and remarkably regained fusion while wearing base out Fresnel prisms of strength 50 prism dioptres to restore fusion before intended strabismus surgery.

CASE REPORTS

Case 1

A 12-year-old girl with emmetropia was referred with a 2-year history of occasional intermittent horizontal diplopia at all distances. Visual acuities were 6/9 in the right and 6/6 in the left. A moderate esophoria (10 prism dioptres) with delayed recovery at near and a moderate esophoria (14–16 prism dioptres) becoming a right esotropia on dissociation, with diplopia, at distance and far distance was present. Stereoacuity and the prism fusion range were slightly reduced. Orthoptic exercises were prescribed but the patient was lost to follow up.

Four months later, after a new hair cut (Fig 1) in which one eye was regularly occluded, the patient returned with a constant right esotropia with diplopia, measuring 25 prism dioptres at near and distance. A 25 prism dioptre Fresnel prism was fitted on planos. The angle following prism adaptation³ increased to 35 prism dioptres for near and distance. BSV was present when wearing the prisms. The patient wore the prisms for 3 weeks before surgery, and showed no spontaneous recovery.

She underwent a right medial rectus recession of 4.5 mm and lateral rectus resection of 7 mm for the full prism adapted angle. Three months later the patient remains symptom free with a well controlled esophoria (6 prism dioptres).

Case 2

A 7-year-old girl, with no previous history of ocular problems, presented to casualty 15 minutes after putting 'super glue' into her left

eye in mistake for an antibiotic cream. On opening the eye 16 days later, she complained of diplopia. A moderate alternating esotropia with diplopia was present for near and distance (40–45 prism dioptres). No significant refractive error was present and visual acuities were 6/6 in each eye. BSV was achieved with prismatic correction of 15 prism dioptres base out right eye and 35 prism dioptres base out left eye, which was prescribed in Fresnel form. One week later the patient reported single vision with prismatic correction, and BSV was present (Bagolini glasses at near and distance). At times a small esotropia (10 prism dioptres) was present at near with diplopia, and the Fresnel prism was not reduced. The patient missed an interim appointment. Six weeks later the diplopia spontaneously resolved at all distances. Four months later, an esophoria is present (12 prism dioptres at near and 4 prism dioptres at distance), and the patient is asymptomatic.

COMMENT

We describe two patients with acquired esotropia who responded in interesting ways to the preoperative use of temporary Fresnel prisms to promote a period of normal sensorial relations before planned surgical correction.

Preoperative prism adaptation has been shown to be a more successful method of treating acquired esotropia than conventional strabismus surgery.^{3,4} Case 1 regained fusion with prismatic correction, underwent surgery for the prism adapted angle, and has remained asymptomatic since. Patients with decompensated esophoria are ideally suited to this test as BSV can be restored after determining the most appropriate amount of strabismus surgery to perform.

Case 2 became symptom free following temporary prismatic correction of a large angle esotropia. The deviation spontaneously resolved with restoration of binocular vision and stereopsis. This uncommon response of such a large angle esodeviation to temporary prism wear has been described previously in two cases.^{1,5}

Acute acquired concomitant esotropia and complete decompensated esophorias are uncommon types of esodeviation. We would suggest that such patients undergo prismatic correction before considering surgery. The patient then has the advantage of undergoing surgery based on preoperative prism adaptation or may regain binocular single vision without surgery.

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 α Fetoprotein as marker for a case of orbital yolk sac tumour

EDITOR.—We describe a case of primary yolk sac tumour of the orbit. This was treated with chemotherapy. Serum α fetoprotein level was elevated before treatment and fell during the course of chemotherapy. To the best of our knowledge, this is the first case in children with primary yolk sac tumours of the orbit having elevated serum levels of α fetoprotein before treatment.

CASE REPORT

A 6-month-old boy presented with a 2 week history of a swollen left cheek and a 5 day history of a swollen and squinting left eye. There was no other significant medical or ophthalmic history of note.

On examination, he had a marked left axial proptosis, with generalised swelling over his left cheek. There was no discrete palpable mass on the orbit nor were there any palpable lymph nodes. He had a left ptosis, complete ophthalmoplegia, and left relative afferent pupillary defect. Mild optic atrophy was evident. No other abnormalities were found on general examination.

Computed tomographic scan of the head (Fig 1) showed a large tumour involving the lateral wall and floor of the left orbit, extending into the infratemporal fossa invading the left antrum and nasal cavity. No intracranial extension was seen. Blood tests showed a raised serum α fetoprotein at 1345 units per litre (normal <10). Transnasal biopsy

Figure 1 Axial computed tomographic scan of the head showing a large tumour involving the lateral wall and floor of the left orbit, extending into the infratemporal fossa invading the left antrum and nasal cavity.

Figure 1 Case 1. Patient with new hair style where the right eye was regularly occluded.

Figure 2 Biopsy specimen showing tumour with cuboidal – columnar cells arranged in parts around blood vessels (Schiller-Duval bodies). Tumour cells stained for α fetoprotein ($\times 60$).

showed tumour with cuboidal columnar cells, arranged in parts around blood vessels (Fig 2), stained for cytokeratin and α fetoprotein but negative for desmin, confirming the diagnosis of yolk sac tumour. Staging investigation did not show any metastatic disease.

He was given dexamethasone to reduce tumour related oedema. The treatment that followed was chemotherapy, using carboplatin, etoposide, and bleomycin, for which he had four courses with gaps of 3 weeks in between. The child was given two courses

beyond the number required to correct α fetoprotein levels, using it as a marker for monitoring therapy. The first course was given 10 days after presentation, and 3 weeks after that the tumour shrank dramatically; only a left relative afferent pupillary defect and some mechanical restriction of abduction remained on examination.

The α fetoprotein level fell dramatically with treatment – down to 250 units per litre 3 weeks after the first course and to 12 units per litre 3 weeks after the second course. During the 6 month follow up period, the child has

developed normally and remained free of tumour recurrence.

COMMENT

Yolk sac tumour is a malignant germ cell neoplasm. Primordial germ cells migrate from yolk sac to genital ridge. Most tumours are therefore gonadal while aberrant migration may occur. Yolk sac tumours in the orbit are rare.¹ In the orbit, they manifest by a unilateral proptosis with or without restricted ocular motility, loss of vision, papilloedema, and congested retinal vessels.¹ α Fetoprotein had been used as a diagnostic and therapeutic markers for gonadal yolk sac tumours,² but it has yet to be shown that children with primary yolk sac tumour of the orbit have elevated serum levels of α fetoprotein before treatment.³

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