(Fig. 1); however, there were no external skin injuries. There was no light perception through the lids. Both a skull radiograph (Fig. 2) and a CT scan were performed. They clearly showed that a metal signet ring was sitting in the posterior part of the maxillary sinus. There was marked haemorrhage, surgical emphysema and enophthalmos with a possible fracture of the roof of the maxillary sinus, but no fracture of the anterior wall of the maxillary sinus was seen on CT. He underwent examination under anaesthesia and removal of the foreign body by the maxillofacial surgeons via the mouth through the gums (Caldwell-Luc approach). With the exception of grossly oedematous conjunctiva, the eyeball was otherwise normal. Four weeks later there was complete resolution of the periorbital and conjunctival edema. The visual acuity remains NPL in an enophthalmic eye and the optic disc is pale. Visual field assessment of the right eye and psychiatric evaluation were normal, and no further attempts at self-harm have occurred.

Comment

Drug-induced psychosis is not common and can result in self-mutilation.[1–3] Self-ocular injury has been described in such settings.[3,4] In the present case the patient insisted that he introduced the ring to relieve a sense of imminent suffocation. The local soft tissue and bony destruction together with the final destination of the ring give some indication of the forces exerted. Self-introduction of foreign bodies into the sinuses is exceedingly rare.[5] In this case the likely route of entry of the ring into the maxillary sinus was through the inferior conjnctival sac, through the orbital floor, and into the posterior maxillary sinus. The cause of blindness was presumed to be secondary to compression of the optic nerve within the optic canal as a result of oedema and haemorrhage.

Two other unlikely possibilities, which have not been reported before, exist. The ring, having been pushed through the inferior orbital floor into the posterior part of the maxillary sinus, then disrupted the orbital floor of the maxilla and caused primary transectional damage to the intraorbital portion of the optic nerve. The other is that the ring may have been introduced into the maxillary sinus through the anterior wall. The force involved and subsequent bleeding may have caused the maxillary sinus to explode upwards into the orbital cavity and cause compression of the optic nerve secondary to haemorrhage and oedema. The acute treatment in this case would then be to perform an immediate lateral canthoplasty.

However, it is more likely that neural dysfunction occurred secondary to compression within the optic canal as a result of oedema and haemorrhage. In this instance treatment options include high-dose steroid treatment and optic nerve sheath decompression.[6] No treatments have been proved effective in a randomised controlled trial, but patients treated with corticosteroids or a combination of corticosteroids seem to have a better visual prognosis.[7,8]

The patient's visual acuity remains NPL in that eye. In a previous case of attempted autoenucleation, good recovery of vision was reported.[9] Visual field assessment of the contralateral eye is advised because of the potential for involvement of the optic chiasm in such a destructive process.[3]

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Sir,

Reduced corneal sensation and severe dry eyes in a child with fetal valproate syndrome

In utero exposure to sodium valproate is linked with a characteristic facial phenotype in which ophthalmic signs are a prominent feature.[1–3] We extend previous observations by reporting here an infant with the 'fetal valproate syndrome' (FVS) associated with severe dry eyes.

Case report

The patient was born via normal vaginal delivery after a 40 week pregnancy which was uncomplicated except for maternal anticonvulsant therapy. Her mother, aged 25 years, started having grand mal epilepsy aged 11 years and was initially treated with phenobarbitone. Due to

poor control, sodium valproate monotherapy was begun at the age of 17 years. Throughout the pregnancy she was prescribed 2300 mg/day and had no seizures during this time.

At birth the patient was noted to have a posterior cleft palate. Evaluation in the genetic clinic revealed a number of additional facial abnormalities including a small mouth, short philtrum, epicanthic folds and infraorbital grooves. Chromosome analysis was normal. This combination of abnormalities was therefore felt most likely to be secondary to maternal ingestion of sodium valproate during the pregnancy.

The patient first attended the ophthalmology department aged 8 months with a history of a sticky left eye since birth. Probing showed a blocked nasolacrimal duct on this side and her symptoms subsequently subsided. Aged 24 months she was noted to be photophobic. Examination under anaesthesia found no evidence of glaucoma, fundal or ocular surface abnormalities. She remained photophobic and at 32 months developed a widespread bilateral superficial punctate keratopathy. Minimal small follicles were noted at that time. Bacterial and viral conjunctival swabs were negative. Although the follicles resolved by her next visit 2 weeks later, the superficial punctate keratitis continued to persist, despite successive trials of topical corticosteroids and lubricants. At 5 years she was still mildly photophobic. The pre-corneal tear film was now noted to be markedly reduced, especially on the left, and associated with a filamentary keratopathy on this side (Fig. 1). Treatment with Viscotears was recommenced with limited clinical and symptomatic improvement. At 7 years the child was able to comply with Schirmer's test, which was 2 mm on the right and 0 mm on the left at 5 min. Tear function index[4] was 8 on the right and 0 on the left. Corneal sensation was tested with a wisp of cotton. This was noted to be absent in the left eye and markedly reduced in the right. Trigeminal nerve function was otherwise intact. There was no evidence of any ocular conditions known to cause symptoms of dry eye or reduced sensation. The child remained systemically well throughout. After an unsuccessful trial of intense topical lubricants, the decision was made to occlude the lower punctum of each lid. A punctal plug was inserted on the right. On the left side, a slit-like abnormality of this punctum prevented plug insertion. Permanent occlusion by cautery was performed. Currently the patient is asymptomatic on topical lubricants with resolution of the keratitis.

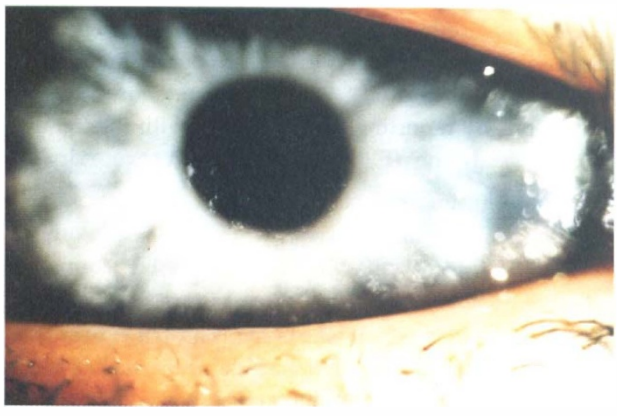

Fig. 1. *The left eye at age 5 years showing a filamentary keratopathy.*

Comment

Sodium valproate is a widely used anticonvulsant, *in utero* exposure to which is associated with a distinctive pattern of congenital malformations referred to as FVS.[5] Affected infants typically have a combination of facial features giving rise to a characteristic facial phenotype. These facial features include a number of ophthalmic signs, most commonly epicanthic folds and an infraorbital groove.[5]

In our patient, while both epicanthic folds and an infraorbital groove were present, the predominant ophthalmic problem was dry eye secondary to poor corneal sensation.[6] Dry eye is relatively uncommon in children and only rarely produces symptoms of photophobia.[7] In the absence of any features of recognised causes for this problem,[7] an association between dry eye and FVS is suggested. Punctal abnormalities have also not previously been described in relation to FVS. This defect was only noted by us when punctal occlusion was considered, and was thought unlikely to have arisen as a consequence of the probing performed at 8 months of age.

Mothers taking sodium valproate during pregnancy are at an increased risk of having children with congenital malformations.[5] Eye signs are common but can easily be overlooked.[1] The finding of dry eye and poor corneal sensation in our patient suggests that these features may also constitute part of the FVS.

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