Case report

A 64-year-old woman presented to eye casualty with a second episode of right dacryocystitis. The visual acuity was 6/6 bilaterally. She was given a 7 day course of oral amoxicillin 500 mg t.d.s. with flucloxacillin 250 mg q.d.s. and was reviewed when the infection had settled. Syringing showed patent canaliculi with regurgitation and she was listed for dacryocystorhinostomy (DCR) under local anaesthesia.

In the anaesthetic room the patient was sedated with 2.5 mg of intravenous midazolam. Two drops of amethocaine were instilled into both eyes. Two puffs of 2% lignocaine spray were applied to the right nasal passage. A nasal pack of 5% cocaine with adrenaline was placed in the right nasal antrum. A local anaesthetic mixture containing 4 ml of 2% lignocaine with 1:200 000 adrenaline and 4 ml of 0.75% bupivacaine was administered to three sites using a 25 mm, 25 G needle in a standard fashion.[2] The infraorbital nerve was infiltrated with 3 ml of anaesthetic, the skin of the side of the nose with 2 ml, and 3 ml was injected medially, lateral to the caruncle with a peribulbar technique. The patient experienced some discomfort during the administration of the anaesthetic, but this was mild. However, once the initial skin incision was made, the patient complained of pain that was not relieved by further infiltration of anaesthetic to the skin and periosteum. It was necessary to convert to a general anaesthetic, and the operation was completed without further incident.

The following day she was noted to have a mid-dilated pupil on the right, with a corrected visual acuity of 6/9. Fundoscopy showed a raised appearance of the temporal and inferior retina and choroid, the macula and disc were normal and there were no retinal haemorrhages. The possibility of globe perforation was raised at this point. One week later there had been a deterioration in the visual acuity to 6/36 and there was vitreous haemorrhage with a nasal suprachoroidal haemorrhage. The suprachoroidal haemorrhage resolved over the next week and at this stage retinal haemorrhages with scarring and vitreous incarceration were seen two disc diameters nasal to the optic disc. This was felt to represent a needle track from a perforating injury and she was admitted for vitrectomy and endolaser to the area of injury. Post-operatively, the retina remained flat and she achieved a final visual acuity of 6/9.

Comment

During the last 10 years DCR surgery has increasingly been performed under local anaesthesia[3,4] because of the benefits of this technique to patient health and the move towards outpatient surgery. The local anaesthetic technique usually involves a medial canthal peribulbar injection.[2] Ocular perforation is a recognised complication of retrobulbar and peribulbar anaesthesia for cataract surgery[1,5,6] but has not been reported during anaesthesia for DCR.

The incidence of penetrating injury is thought in part to be due to globe shape, with myopic eyes being at greater risk. Vohra and Good[7] suggest, however, that a medial canthal approach is the safest, especially in larger globes.[7] This is because of a reduction in the equatorial width to axial length ratio in high degrees of axial myopia. Inflammation of the tissues surrounding the usual landmarks, for example following dacryocystitis, as in this patient, can alter the anatomy of the injection site and increase the risk of perforation. Meyer[8] reports some success with topical anaesthetic techniques which would eliminate the risk of penetrating ocular injury.

Early diagnosis and treatment of ocular perforations are essential for a good visual outcome[6,9] and therefore there should be a high index of suspicion in those cases where the injections are excessively painful, or ineffective, or if there is hypotony of the globe or a decrease in visual acuity. DCR surgery has been effectively performed under local anaesthesia for many years[4] but care must always be taken during intraconal anaesthetic injections.

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Sir,

Rapid development of visual field defects associated with vigabatrin therapy

Vigabatrin, a selective irreversible gamma-aminobutyric acid (GABA) aminotransferase inhibitor, is a new antiepileptic drug that is generally well tolerated with few side-effects. However, bilateral permanent visual

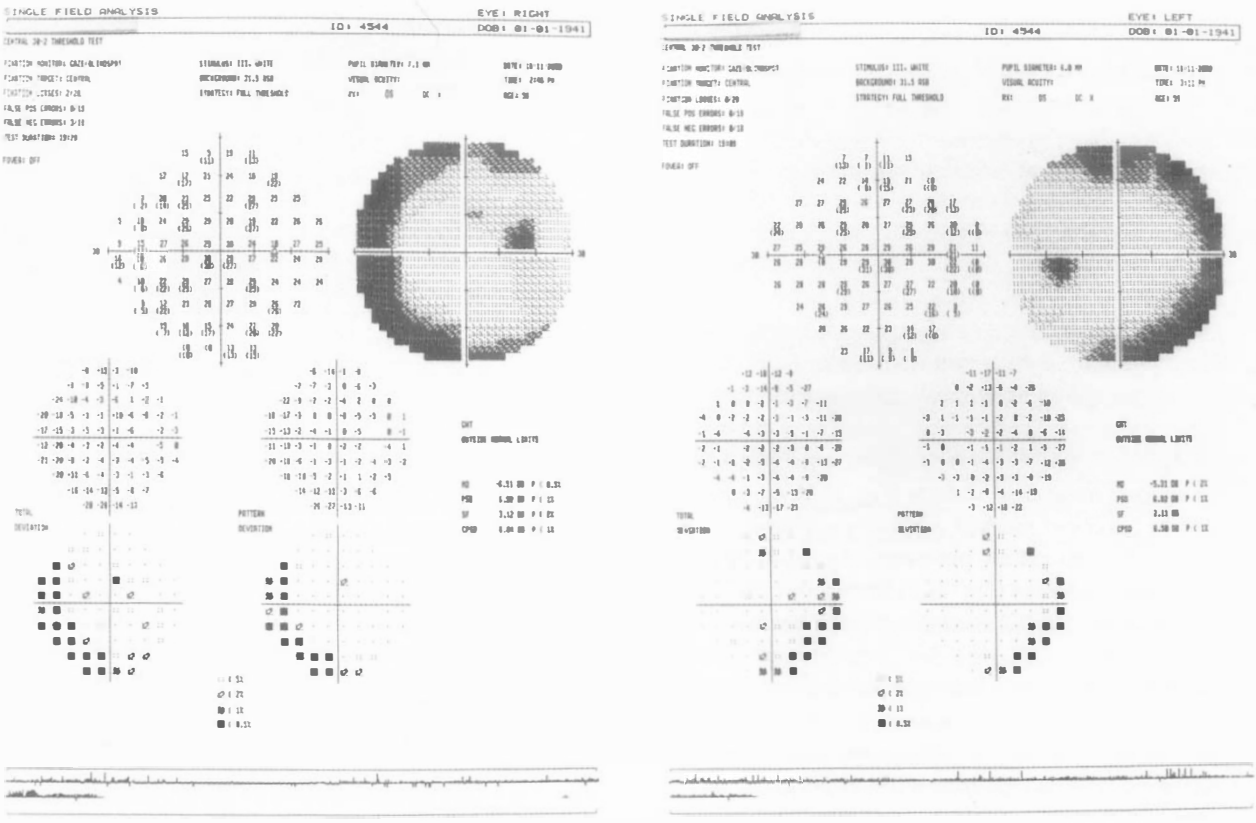

Fig. 1. *Most recent automated 30-2 visual field test results showing symmetric binasal field defects: (a) right eye, (b) left eye.*

field defects in association with the use of vigabatrin consistently continue to be reported.[1–3] We describe a woman who started to experience visual abnormalities including field defects as early as 6 months following vigabatrin intake. Because no visual field testing was performed before the introduction of vigabatrin in our patient, we presume that this drug is the most likely cause of the later documented visual field changes.

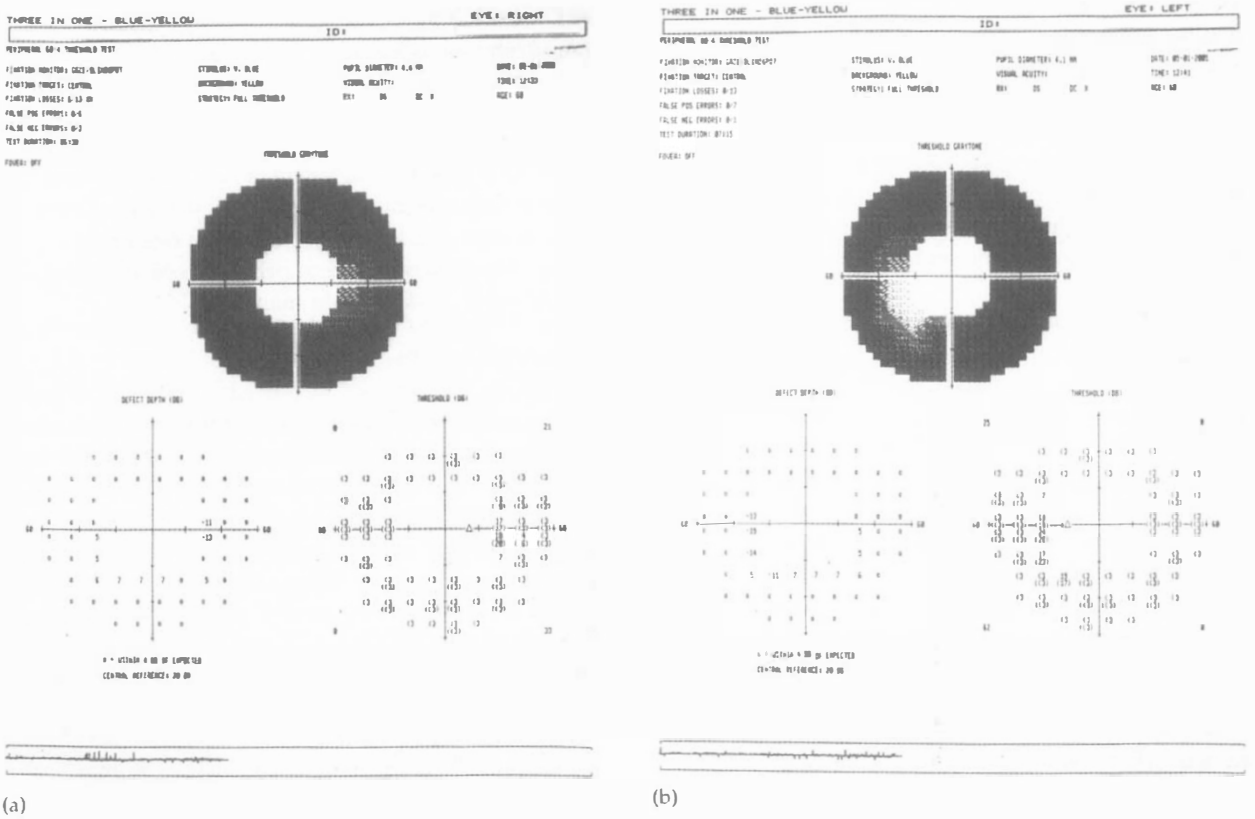

Fig. 2. *The 60-4 blue-on-yellow perimetry results demonstrate bilateral dense annular scotomas: (a) right eye, (b) left eye.*

Case report

A 60-year-old otherwise healthy woman has been treated for epilepsy for 10 years. The first drug she was prescribed was carbamazepine, and 2 years ago vigabatrin was added at a dose of 500 mg q.i.d. A year later sodium valproate was also added. Six months after she started vigabatrin she developed photophobia, flickering lights and saw 'shadows at both sides'. On ocular examination her visual acuity was 20/20 both eyes. She could easily identify Ishihara plates. There was no afferent pupillary defect. Anterior segments and fundi were within normal limits. Intravenous fluorescein angiography did not reveal any abnormality. Automated field testing using the 30-2 program disclosed bilateral crescent-shaped nasal field defects (Fig. 1). A 60-4 full threshold test showed marked annular peripheral field defects; 60-4 blue-on-yellow perimetry demonstrated denser annular scotomas (Fig. 2). Scotopic and photopic electroretinography (ERG) recordings were normal. On electro-oculography (EOG) the Arden ratios were clearly reduced (42% and 30% for the right and left eye respectively). Vigabatrin was tapered to 500 mg t.i.d. Photophobia and flashing lights have resolved. However, the visual field defects persisted without progression. The patient is currently checked every 3 months.

Comment

Visual field defects reportedly develop in 30% of adult patients and in up to 65% of children taking vigabatrin.[2,3] These defects are usually symmetric, absolute and binasal, sparing the temporal field.[1] The concentric contraction of visual fields does not change or improve when the medication is discontinued.[4] Currently, the visual field changes are explained by the accumulation of GABA in the Mueller cells and the GABA transaminase inhibition of the rod bipolar cells in the peripheral retina.[5] This is further supported by the fact that most observed ERG abnormalities involved scotopic b-waves, produced by Mueller cells.[5] As in our patient, there is often a reduced Arden ratio on EOG that is yet to be explained.[1,5]

Some authors believe that the effect is cumulative over a prolonged period of treatment and found a strong correlation between the percentage of visual field changes and total dose of the drug.[2,3] It was suggested that a daily dose of 1500 mg or more would be likely to cause visual field defects.[2] In children, field defects were observed from 1 year up to 6.5 years after the start of vigabatrin.[3] Others did not find any relationship between the dose and duration of therapy and the risk of developing visual field losses.[5] The emergence of this side-effect as early as 6 weeks after the institution of vigabatrin could suggest an idiosyncratic response as well.[6] Our case favours the latter point of view.

The majority of studies in the current literature were conducted on patients who had been taking vigabatrin for many years, and in most of these patients it was not possible to determine exactly when the visual field changes first started. The patient reported here demonstrates that visual field loss may occur soon after the institution of vigabatrin therapy.

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Sir,

Primary tuberculosis of the conjunctiva

Conjunctival tuberculosis is now a very rare condition in the developed world, although it was not uncommon in the nineteenth and the early part of the twentieth century. We present a case of primary conjunctival tuberculosis in a 34-year-old man.

Case report

A 34-year-old male Caucasian General Practitioner with no past medical history presented with a papillomatous lesion in the right upper tarsal conjunctiva. There had been a mucoid discharge for the previous 3 months. The lesion was excised and the patient was treated with topical chloramphenicol. Histopathological examination revealed the inflamed conjunctival tissue with occasional giant cells in the underlying fibrous tissue associated with lipoid material. The appearances were consistent with a diagnosis of chalazion.

The lesion recurred 3 months later, was excised, and the base cauterised. Histopathological examination revealed the presence of mixed acute and chronic inflammatory cells. There were areas of granuloma