that the interval should be 3 months for the first 3 years of treatment.

A lack of communication between the neurologists and ophthalmologists was stated, by 75% of respondents, as the most common reason for noncompliance with the screening guidelines. This probably relates to the failure of neurologists to refer patients for screening prior to commencing them on Vigabatrin and also to the lack of involvement of the ophthalmologist in the follow-up of these patients.

Surprisingly, a small proportion of respondents cited that an increase in workload as a reason for noncompliance with the RCO guidelines, in spite of the relatively small number of patients requiring screening. Those respondents who were involved in screening patients were performing less than five visual fields a year.

There is a disparity between current clinical practice and the RCO guidelines. None of the respondents had carried out an audit of their clinical practice regarding screening of patients on Vigabatrin. While patient numbers may be few, continued audit and clinical data collection should be encouraged in accordance with RCO guidelines. Although the exact pathogenesis of these field defects is not known, the need to screen patients on Vigabatrin is well established. Screening is important if the field defects are caused by an idiosyncratic reaction to the medication, particularly as only a certain group of patients will be affected.

It is disappointing that despite the RCO publishing guidelines, more than a third of the consultant ophthalmologists responding to the survey were unaware of their existence. Our survey of clinical practice indicates that there is only a moderate agreement with the current guidelines. A joint guideline issued by ophthalmologists and neurologists regarding screening for Vigabatrin-associated field defects may help bridge the gap between the specialties and achieve wider agreement and compliance.

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Sir,

Reply to Kumar and Jivan—Vigabatrin-related visual field defects

The results of the postal survey by Kumar and Jivan on screening for Vigabatrin-related visual field defects has yielded some interesting data reflecting the application of guidelines in the real world. Busy clinicians are naturally averse to extra service load and there are always communication issues between hospital specialists. However, it is worth pointing out that Vigabatrin has now largely been replaced by newer agents without this side effect for the control of refractory epilepsy, so the number of epileptic patients with visual impairment from this phenomenom is limited. Although the field loss is not treatable, recognition and appropriate visual impairment registration is still beneficial to the patient.

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Sir,

Corneal injury from a fishing line: a new mechanism

Fishhooks[1] and sinkers[2,3] are well recognised to cause severe trauma to lids and cornea.[4] Other sequelae to such injuries include retinal detachment[5–7] and endophthalmitis.[1] In the commercial fishing sector, fish bile and fish picks (used in handling heavy fish and crates) are a form of occupational eye morbidity.[8,9]

We report the findings in a patient who, while fishing for salmon (*Salmo salar)* on a river, was struck in the eye by the fishing line alone, apparently without involvement of the hook, a mechanism we believe not to have been reported before. We discuss the mechanisms of injury and treatment options.

Case report

A 52-year-old man presented with an injury to his left eye sustained while fishing for salmon on a river. He was struck on his left eye when he lost control of the back cast. It was only the moving line that hit him and not the treble hook fly. Systematic questioning revealed no significant past ophthalmic or medical history.

At presentation, the visual acuity (VA) in the right eye was 6/6 and hand movements in the left eye. A linear partial thickness lid laceration involved the medial portion of both left upper and lower lids, and it was superficial to the lacrimal canaliculus. The wound edges were well apposed and did not require suturing. The corneal injury was a well-defined linear partial thickness laceration involving the nasal portion from which a thin strand of monofilament nylon (fluoro-carbon) was removed (Figure 1a). Seidel's test was negative. The anterior chamber (AC) was slightly shallower nasally and clouded with hyphaema. The pupil was slightly dilated, and the iris appeared traumatised nasally. There was no relative afferent pupillary defect. The lens showed early cataractous changes with mild phacodonesis. Posterior segment details were not clear because of the haziness of the cornea, hyphaema, and lens changes. B-scan ultrasonography showed choroidal haemorrhage nasally consistent with shallow anterior chamber leading to ciliary body rotation.

He was managed conservatively on topical ofloxacin 0.3% q.d. for 10 days, topical prednisolone 0.5% q.d. for 2 weeks and oral ciprofloxacin 750 mg b.d. for 1 week. A formal vitreo-retinal and ultrasound opinion was sought from a tertiary centre, and this confirmed supra-choroidal haemorrhage only.

After 10 days VA had improved to 6/18 LE. Both lids and corneal laceration were almost healed. The choroidal haemorrhage was noted to be resolving.

By 3 months, the VA had become reduced to 6/60 LE. The IOP was 12 mmHg in LE. The cataract was now moderately dense and with mild phacodonesis. Gonioscopy showed multiple peripheral anterior synaechiae and 2 clock hours of angle recession in the inferonasal quadrant. The choroidal haemorrhage had resolved.

A decision to proceed to elective cataract surgery was taken and biometry was performed based upon different techniques of assessing keratometry. On axis phacoemulsification with PCIOL acrylic lens (Sensar OptiEdge™) and intracapsular tension ring (Morcher™: Type 14c MR–1420) was performed successfully without further loss of zonular integrity. There has been no evidence of secondary glaucoma or vitreoretinal problem in the postoperative period. A 3-month postoperative review revealed stable refraction: LE:$-0.25/+1.00\times10=6/5$. He has been discharged from our care with the relevant cautions for vigilance regarding long-term sequelae. He has been advised to visit his optician on yearly basis for IOP check.

Comment

We believe this is first report of an injury from a fishing line rather than fishhook. The eye suffered from injury by two mechanisms: laceration and blunt trauma. We presume the laceration was due to cheese wiring effect and thermal changes from friction of a thin, but tough, line. The line is also partly abrasive (braided leader). It is apparent that while elements of the line might have relatively low mass (although some salmon lines, especially the sinking type used here have significant mass relative to an eye), it travels at high speed and hence carries significant momentum, and this is directly related to the high speed through the air that is necessary for casting the line. The evidence of blunt trauma is the presence of cataract, choroidal haemorrhage, and angle recession.

The corneal scarring sustained induced irregular astigmatism that potentially affected biometry calculation. The true corneal power measurement may be quantitatively assessed with keratometry (Von Helmholtz type, eg Bausch & Lomb or Reichert, Java-Schiotz or hand-held Nidek™). Alternatively, computerised corneal topography or contact lens over refraction may be used to assess corneal power. Cua