Sir,

An unusual case of severe penetrating ocular injury with the shank of a triplet fish hook

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Fortunately, penetrating ocular injuries with fish hooks are rare. These injuries are usually caused by the fish hook prongs penetrating the ocular tissues. We report a rather unusual case of severe ocular injury caused by the shank of a triplet fish hook.

Case report

A 46-year-old woman presented to the eye casualty with a three-pronged steel fish hook embedded in her left eye. She had been fly fishing using a 20 Gauge, 20 mm long salmon hook. While whipping the line back to cast her hook, she felt a blow on her eye as if she had been hit with a stone. She cut the line close to the hook, but did not attempt to remove the hook. The shank of the fish hook, with an intact fly had penetrated the medial bulbar region 5 mm posterior to the limbus. All the three prongs were lying just outside the globe, with one barb embedded in the lower lid. The visual acuity was hand movements in the affected eye. X-rays showed penetration of 18 mm of the shank of the hook into the tissues (Figure 1), with no bony injury.

Under general anaesthesia, the barb was released from the lower lid by blunt dissection to spread the orbicularis, rather than the traditional method of cutting the hook and pushing the barb through the tissues as it was felt that this might disturb the shank of the hook and cause more damage to the eye. The shank was grasped outside the sclera with forceps and carefully withdrawn. Prolapsing uveal tissue and vitreous were excised and the scleral perforation measuring 4 mm in length was closed with 9-0 nylon. No shank exit wound was found. The patient received intravenous antibiotics on induction of general anaesthesia and also postoperatively. A left relative afferent pupillary defect was noted. B-scan ultrasound demonstrated a disorganised vitreous and massive choroidal haemorrhage with overlying retinal detachment.

Vitreoretinal surgical repair was undertaken, 12 days following primary repair, with signs of choroidal haemorrhage liquefaction on ultrasound. The liquified choroidal haemorrhages were drained with an anterior chamber infusion and small sclerostomies over the pars plana. A three-port pars plana vitrectomy was then carried out. This revealed an incomplete posterior vitreous detachment with a giant retinal tear nasally from 9.30 to 12 o'clock position, extending to within 1 mm of the optic nerve head. Most of the supranasal retina was

Figure 1 X-rays showing the extent of ocular penetration of the triplet fish hook.

absent having been avulsed by the fish hook in the original injury with some incarceration of the remaining retina in the scleral wound. The vitreous was detached up to the vitreous base using aspiration with heavy liquids to stabilise the posterior retina during the procedure. A relieving retinotomy was performed locally around the retinal incarceration site. Profuse intraocular bleeding during this procedure was controlled by temporarily elevating the intraocular pressure. Subretinal haemorrhage was evacuated and indirect laser applied to the retinal edges. After heavy liquid/air exchange, an encircling 277 buckle was placed and a silicone oil exchange carried out. The lens remained fairly clear during the procedure and was spared.

Postoperatively, the retina remained attached and the remaining choroidal haemorrhage resolved. At 4 weeks

postoperatively, the patient managed 6/36 unaided vision improving to 6/12 with a pinhole. Unfortunately, 2 months postoperatively she developed macular pucker and increasing tautness of her nasal giant retinal tear edge with a local area of subretinal fluid. There was also increasing cataract formation. A revision vitrectomy was carried out combined with phacoemulsification and intraocular lens implantation. The macular membrane was peeled, further relieving retinotomies carried out on the nasal retinal edge and the silicone oil replaced. After further postoperative laser, 4 months following this procedure, the silicone oil was removed and at 6 months follow-up, her best corrected vision was 6/24 with a stable attached retina.

Comment

Previous reports describe penetrating ocular injuries caused by fish hook prongs.¹⁻⁴ In our case, the shank of a triplet fish hook caused severe ocular damage, which to date has required further ocular surgery with moderate visual outcome. We emphasise the potentially sight threatening nature of this recreational pastime and the need for protective eyewear.

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TS Kalyanasundaram¹, Diana Depla² and David Steel³

¹Ophthalmology Department
Burnley General Hospital, Burnley
Lancashire, BB10 2PQ, UK

²Ophthalmology Department
Cumberland Infirmary
Carlisle CA2 7HY, UK

³Sunderland Infirmary
Sunderland SR2 9HP, UK

Correspondence: TS Kalyanasundaram
Tel: +44 1282 474162
E-mail: tskskalyan@yahoo.com

Sir,

Bilateral hypopyon following streptokinase treatment for acute myocardial infarction

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Streptokinase, a commonly used thrombolytic agent, is a nonenzymatic protein produced by beta-haemolytic streptococci. It has been associated with several allergic reactions including bronchospasm, urticaria and anaphylaxis.¹ We report an unusual case of bilateral uveitis with hypopyon following streptokinase therapy for myocardial infarction (MI).

Case report

A 74-year-old Caucasian gentleman was admitted with acute central chest pain of 3 hours duration and electrocardiogram was diagnostic for an anterior MI. Immediate medical management involved intravenous diamorphine 5 mg and cyclizine 50 mg. He was subsequently thrombolysed with 1.5 million units of intravenous streptokinase. Within 24 hours of receiving treatment, he had developed bilateral sore red eyes and reduced vision, worse in his left eye. However, he was only referred to the ophthalmology department 72 hours later, by which time his cardiovascular status had been stabilised and his ocular symptoms had actually begun to improve.

His past ocular history was significant for mild left amblyopia, ocular hypertension and bilateral retinoschisis. Best corrected visual acuity (BCVA) was 6/9 in the right eye (RE) and counting fingers in the left eye (LE). Ocular examination revealed bilateral conjunctival injection and anterior uveitis with hypopyon. The hypopyon measured 0.8 mm RE and 2 mm LE. Intraocular pressures were 22 mmHg RE and 24 mmHg LE. The vitreous in his RE was quiet and the fundus showed inferotemporal retinoschisis, but there was no fundus view of his LE. Ultrasound examination of his LE showed no signs of inflammatory activity. The patient had no clinical signs or symptoms of systemic vasculitis and no previous history of allergies. He was treated with intensive topical steroids and cycloplegics to which he had a prompt response and his BCVA improved to 6/6 RE, 6/12 LE by the second day of treatment. He subsequently made a complete visual recovery by the fourth week of treatment and his BCVA was 6/6 RE and 6/9 LE.

Comment

So far, seven cases of acute anterior uveitis following streptokinase therapy have been reported to the Committee on Safety of Medicines (personal communication). Of these, only one case had bilateral hypopyon.² The pathogenesis of streptokinase-induced