

Fig. 1. Appearance of the eye injury caused by a fish-hook.

nously and the patient was carefully monitored for signs of endophthalmitis. Visual acuity was 20/200 right eye on discharge. Two years later visual acuity was 20/30 right eye with mild corneal scarring without any other complications.

Discussion

Penetrating fish-hook injuries are a rare and potentially devastating trauma. Fishing organisations should be aware of the importance of prevention (e.g. sunglasses) and first aid in eye injuries. In our case further risk for infection was the live bait used.

There are at least four known techniques for the extraction of a fish-hook from the eye, each of which has advantages and disadvantages. We suggest that a large rusty fish-hook, susceptible to a wide range of unusual pathogens, which injures the eye outside of the central cornea, should be removed through an additional corneal incision. This minimises the risk of further damage to internal structures during specialised repair by backing the hook out through the entrance wound. Residual corneal rust stains derived from the fish-hook, the risk of endophthalmitis or foreign bodies during the use of wire cutters to transect the hook are minimised as well. The technique described by us has in our case yielded an excellent long-term visual outcome. It is of further interest to compare the method of backing the hook through the entrance wound and our incision technique for hook removal.

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Sir,

Dacryocystitis

Dacryocystitis is inflammation of the lacrimal sac. In his 1944 paper on dacryocystitis, Gifford¹ stated that this disease is a 'sort of no-man's land', neglected by both ophthalmologists and rhinologists. With regard to the aetiology of dacryocystitis, this statement is as true today as it was then.

If untreated or inadequately treated, dacryocystitis can act as a potent reservoir of pyogenic infection. Its complications include mucocoele formation, conjunctivitis, corneal ulceration, endophthalmitis and orbital cellulitis. The American Academy of Ophthalmology² therefore recommend that this condition 'should be corrected prior to performing any intraocular surgery'.

In southern India dacryocystitis is a common problem constituting a considerable drain on resources. In 1993, the Aravind Eye Hospital in Madurai performed 10 822 operations for ophthalmic conditions other than cataract. Of these, 12.7% were dacryocystectomy operations, the vast majority due to chronic dacryocystitis. Fig. 1 shows the age and sex distributions of patients presenting to the Out-Patients Department of the Aravind Eye Hospital (Paying Section) who were clinically diagnosed as having dacryocystitis. The most striking feature is the large number of patients below 45 years of age. An additional unexpected finding is the male predominance over 65 years of age: a finding not accounted for in the literature on dacryocystitis. We assume that the Aravind Eye Hospital is representative of the extent of the problem in the community on account of the hospital's strong commitment to rural outreach programmes.

We therefore conclude that the number of cases of dacryocystitis in relatively young patients may constitute a major problem in developing countries. The reasons for this remain obscure. We strongly recommend that dacryocystitis be the subject of considerably more research in future, with a view to prevention.

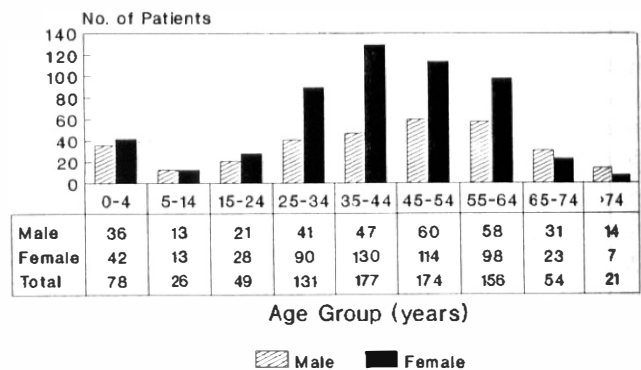


Fig. 1. Outpatient cases of unilateral/bilateral dacryocystitis by age and sex. Data from Aravind Eye Hospital (Paying Section), 1993.

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Sir,

Myasthenia Gravis with Pupillary Involvement

Myasthenia gravis is an autoimmune disorder affecting the nicotinic acetylcholine receptors at the neuromuscular junction of skeletal muscle. The resulting loss of functional receptors impairs transmission of signals at the neuromuscular junction leading to weakness and increased fatigability in those muscles. Myasthenia gravis can occur in generalised or ocular forms, but patients with both types frequently present to the ophthalmologist initially with symptoms of diplopia and/or ptosis. We present a case where atypical signs confounded the diagnosis.

Case Report

A 15-year-old girl presented with a 5 day history of vertical and horizontal diplopia and right ptosis. She was otherwise fit and well and had no past medical history of note.

On examination visual acuity was 6/6 right and left. The right pupil (3.5 mm) was larger than the left (2 mm) and was slow to constrict (and redilate) on direct and consensual light stimulation. There was limitation of right elevation, adduction and depression, but left extraocular movements were full. She had 4 mm of ptosis of the right lid. The optic discs and posterior poles were healthy.

Neurological examination (including deep tendon reflexes) was otherwise normal. Full blood count and film were normal, as was random blood glucose. Autoantibody screen including acetylcholine receptor antibody assay and syphilis serology were all negative, and thyroid function tests were normal. Magnetic resonance imaging (MRI) of the brain and orbit was normal; so too was an MRI angiogram. A lumbar puncture was performed, and results of cerebrospinal fluid analysis were unremarkable.

Her symptoms gradually improved over the Christmas period, but on her return to school the ptosis and diplopia worsened, especially by the end of the day. A test dose of edrophonium was given intravenously with immediate but temporary resolution of the ptosis and diplopia, confirming our

suspicion of myasthenia gravis. She has been asymptomatic on oral physostigmine for the last 3 months. Her right pupil is decreased to 2.5 mm in diameter but is still sluggish in its reaction to light. No hypersensitivity to g. Pilocarpine 0.125% has developed. Right extraocular movements are full and there is only 1 mm residual ptosis.

Comment

Myasthenia gravis, in its generalised or ocular form, is understood not to affect the pupil or accommodation since these functions are subserved by non-striated muscle.¹ However, pupillographic analysis² and slit lamp biomicroscopy studies³ have demonstrated that a high proportion of myasthenic patients have abnormal pupillary function. Furthermore Dutton *et al.*⁴ have demonstrated fatigability of pupil constriction in bright light in a series of 11 patients with myasthenia gravis. The absence of nicotinic receptors on the iris musculature means pupillary involvement is difficult to explain. The site of dysfunction may lie elsewhere, perhaps at the nicotinic receptors of the ciliary ganglion. Research by Watanabe *et al.*⁵ suggests possible dysfunction at the neuronal nicotinic acetylcholine receptors in the central nervous system of patients with myasthenia gravis. It has been suggested that seronegative myasthenia gravis affecting only oculobulbar musculature may represent a separate disease entity in which an autoimmune response is not implicated.⁶ In such a case symptoms and signs may not be limited to those attributed to dysfunction at the neuromuscular junction of skeletal muscle. Clearly 'myasthenia gravis' is incompletely understood and further research is being undertaken.

In the meantime we note that pupillary involvement does not exclude the diagnosis of this treatable disease. Its early consideration as a differential diagnosis may prevent unnecessary and invasive investigations and avoid delay in the administration of appropriate therapy.

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