the vitreous opacification.[2,3,4,7] However, as we have demonstrated, even after vitrectomy, amyloid may infiltrate the trabecular meshwork and iris, resulting in secondary glaucoma.

The ophthalmologist should be aware that ocular amyloidosis may present with a vitreous haze and should be included in the differential diagnosis of vitreous opacification. A full medical and family history is mandatory but may be negative in the case of sporadic ocular amyloidosis. When a vitreous biopsy is considered the sample should be stained specifically for amyloid. Long-term follow-up of patients with proven ocular amyloidosis is mandatory as glaucoma may develop insidiously as a late complication.

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Sir,

Bilateral third-nerve palsy with aberrant regeneration in Guillain–Barré syndrome

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Aberrant regeneration of the third nerve commonly develops several months after third-nerve palsy.[1] Although third-nerve palsy occurs as a complication of Guillain–Barré syndrome in up to 17% of cases, most resolve without sequelae.[2] We report a 9-year-old boy who developed signs of aberrant regeneration of the third nerve as a consequence of Guillain–Barré syndrome.

Case report

A 9-year-old boy presented to a hospital in India in October 1998 with a 1-day history of mild unsteady gait and fever. Past medical and ophthalmic history was unremarkable. Within 14 h he developed a rapidly progressive ascending paralysis of all four limbs and respiratory failure. He required a tracheostomy and ventilation for 1 month. He was treated with immunoglobulins and antibiotics, and after 9 weeks he was transferred to the United Kingdom for further management.

On admission, he was afebrile with a blood pressure of 120/80 and a pulse of 80. He had a flaccid paralysis affecting all four limbs with power grade 3/5 in all groups of muscles. Tendon reflexes were absent and plantar responses were flexor. There was also weakness of the facial muscles with slurred speech, normal swallowing but difficulty in chewing. Sensory examination, bowel and bladder function were normal. Ocular examination showed that visual acuities were 6/12 OD and 6/9 OS. There was no relative afferent pupillary defect. Confrontation fields and colour vision testing were normal. In the primary position of gaze, there was bilateral 3 mm ptosis and a 50 prism diopter (PD) right exotropia with a slight right hypotropia. Adduction was limited in both eyes. Anterior segment examination was unremarkable. Fundoscopy showed a mild optic atrophy on the right but a normal left optic disc.

Magnetic resonance imaging showed no intracranial abnormalities. Nerve conduction studies of the right median nerve, right ulnar nerve and the right lateral popliteal nerve showed results that were consistent with the diagnosis of a generalised demyelinating neuropathy.

At 12 months follow-up, his flaccid paralysis had significantly improved and he was able to stand on his own and just able to walk unsupported. Visual acuities were 6/6 OD and 6/5 OS. The right exotropia with

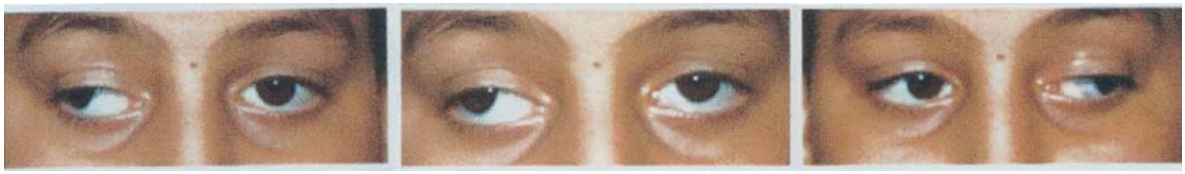

Figure 1 Motility examination reveals bilateral third-nerve palsy with aberrant regeneration.

hypotropia and the bilateral adduction weakness remained and he denied any diplopia. He had bilateral ptosis of 3 mm, but interestingly there was now lid elevation on adduction and lid depression on abduction in both eyes (Figure 1). There were no associated pupil abnormalities. The mild optic atrophy of the right eye was unchanged.

To evaluate the potential benefit of surgery to improve cosmesis, Botulinum toxin A was injected in his right lateral recti muscle. This reduced the right exotropia in primary position to 30 PD with now subjective diplopia. He is due to undergo right lateral rectus recession with medial rectus resection.

Comment

Ophthalmoparesis is the most frequent ocular sign in Guillain–Barré syndrome and third-nerve abnormalities occur in 10–17% of patients.[2,3] The oculomotor nerve aberrant regeneration or misdirection syndrome is believed to result from the extensive growth and misdirection that characterises the regeneration of injured nerve fibres. Ocular findings include elevation of the upper lid on attempted adduction of the eye (pseudo-von Graefe sign), retraction of the globe on attempted vertical gaze and constriction of the pupil on adduction of the eye (pseudo-Argyll Robinson pupil).[1] The pseudo-von Graefe sign results from the growth of medial and inferior rectus axons into the levator. The patient in the current report had bilateral third-nerve palsies with pseudo-von Graefe sign and depression of the lids on abduction.

Aberrant regeneration is well documented in acquired oculomotor palsy, most commonly following aneurysms or trauma and it may occur in congenital third-nerve palsy.[4] There are no previous reports of Guillain–Barré syndrome, to our knowledge, with ophthalmoplegia leading to aberrant regeneration.

Guillain–Barré syndrome is an acute inflammatory demyelinating polyneuropathy. It is the most common cause of acute generalised paralysis with an annual incidence of 0.4–2 cases per 100 000 population.[2,5] In 40% of cases it presents after a flu-like illness, involving cytomegalovirus in about 15% of all cases, Epstein–Barr virus in about 5% and Mycoplasma in 5%.[6] Allergic reactions account for about 10% of cases usually following the administration of lithium, streptokinase and botulinum toxin A injection. It classically presents as an ascending motor paralysis that may progress to involve the respiratory and extraocular muscles in severe cases. The weakness is relatively symmetrical and usually progresses for 1–3 weeks with subsequent slow improvement over several months.[5] The mortality rate is 3–8%, typically from complications such as sepsis, respiratory failure and pulmonary emboli.[6] Only 15% of cases recover completely and about 65% have persistent minor neurological disturbances. Several studies found that children have a better prognosis than adults. The history, physical signs and nerve conduction studies of our patient were consistent with the diagnosis of Guillain–Barré syndrome.

Increased attention to the eyelid manifestations of the Guillain–Barré syndrome has shown a variety of eyelid abnormalities including lid lag and lid retraction.[7,8] Optic nerve involvement may be a result of either optic neuritis or papilloedema.[9,10] The latter occurs in 4–6% of patients with Guillain–Barré syndrome and is believed to be caused by either impairment of CSF reabsorption secondary to high protein level or by cerebral oedema.[9] Visual loss at presentation can be very severe, but most patients usually have good recovery. Paralysis of accommodation has also been reported.[11]

Several reports have suggested that aberrant regeneration following injury to the third nerve is not fully explained by the misdirection of regenerating nerve fibres. They suggest that the central mechanism seems more plausible with disruption of the synapses of the third-nerve nuclei.[12] It is possible that both peripheral and intramedullary mechanisms are involved simultaneously. We report that aberrant regeneration of the third nerve should be added to the list of neuro-ophthalmological complications of Guillain–Barré syndrome.

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Sir,

Factitious keratoconjunctivitis (not another case of ocular Munchausen's syndrome)

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We report the case of a young female patient who self-induced a chronic keratoconjunctivitis by inserting tissue paper and cotton wool into her conjunctival fornices.

Case report

A 17-year-old woman was referred to our outpatient department with a 2-month history of left conjunctivitis and mild periorbital oedema. Initial unaided visual acuities were 6/9 in each eye. Right ocular examination was normal. Left ocular examination revealed intense papillary conjunctivitis, mainly involving the inferior tarsal and bulbar conjunctiva, with multiple punctate erosions of the inferior cornea (Figure 1a). Mild left periorbital oedema and a mucous discharge were also present. There were no prior ophthalmic problems. She suffered from hay fever and worked in a factory as a fish processor. Our initial differential diagnosis included allergic, toxic, or infective conjunctivitis.

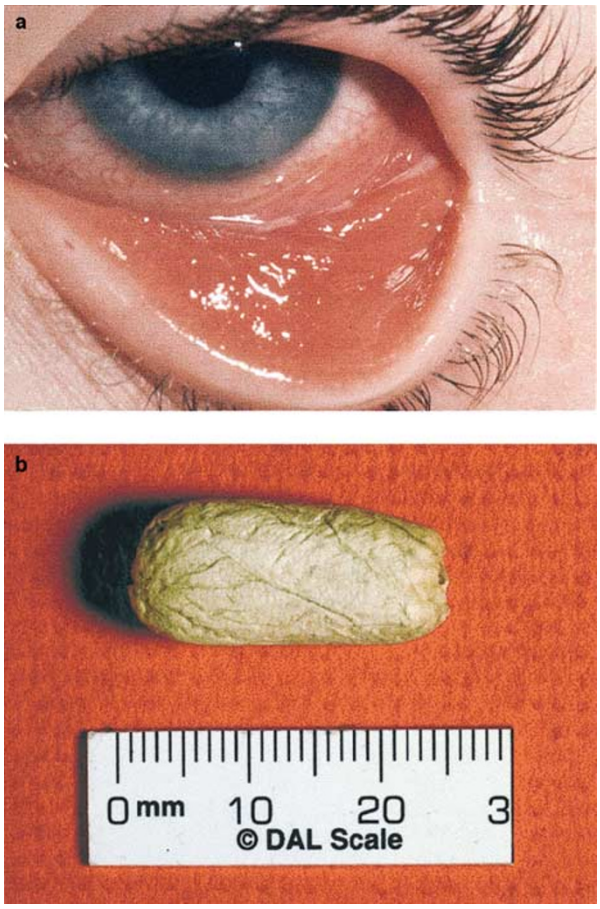

Figure 1 (a) Inferior keratoconjunctivitis (lid held down by photographer). (b) Tissue paper foreign body.

In the following 2 months repeated conjunctival swabs were negative for bacteria, viruses and chlamydia. Despite courses of topical steroids and antibiotics (both preserved and unpreserved), and a trial of systemic erythromycin, her conjunctivitis persisted and inferior corneal subepithelial infiltrate with neovascularisation developed. Conjunctival biopsy showed changes of chronic inflammation only. Her full blood count, urea and electrolytes, serum immunoglobulins, C-reactive protein and autoantibodies were all normal.

One month later her left keratoconjunctivitis improved but she developed identical signs in her right eye.