Comment

Central nervous system (CNS) involvement is a rare manifestation of CIDP, and its precise incidence is unclear. Mendell *et al.*,¹ Pakanlis *et al.*,² Uncini *et al.*³ and Ohtake *et al.*⁴ reported a total of 11 of 55 consecutive patients with clinical signs or symptoms of CNS involvement. Optic neuropathy was found in 8 of these 11 patients. In general, these clinical features are indistinguishable from those of multiple sclerosis. In addition to clinical CNS findings, subclinical involvement can be demonstrated by VEP, brainstem auditory evoked response (BAER), electroencephalography (EEG) and MRI. In these four studies there were 17 patients with abnormal results of evoked potentials or MRI but no CNS symptoms or signs noted. In these 17 patients, VEPs were abnormal in 11, BAERs in 2 and EEGs in 1. MRI showed changes suggestive of CNS demyelination in 7 patients. According to the VEP findings of these reports, the optic nerves seem to be most frequently affected in CIDP patients.

The mainstay of CIDP treatment is corticosteroid, for instance prednisolone 60–100 mg per day for 2–4 weeks, then gradually tapered to 5–20 mg every other day. The beneficial effect of corticosteroid is to speed recovery and to prevent relapse. This disease is also responsive to plasma exchange or intravenous infusion of high dose of immunoglobulin.

Optic neuropathy is one of the clinical manifestations in CIDP with CNS involvement, but its course, therapy and prognosis are not well documented in the literature. Our patient initially suffered from PNS demyelination, which was followed by optic neuropathy about 1 year later. Under treatment with corticosteroid, his PNS symptoms and signs subsided significantly and there has been no relapse to date. However, recurrent episodes of optic neuropathy occurred within 4 months in spite of pulse therapy with methylprednisolone. The different courses and responses to corticosteroid between the myelinopathy in the PNS and CNS suggest a possible difference in their immunopathogenetic mechanisms.

In our report, intravenous corticosteroid accelerated the recovery of visual function but did not prevent the subsequent attack of optic neuropathy. It needs to be further investigated whether CIDP-related CNS demyelination is also responsive to other therapies of CIDP, such as plasmapheresis or intravenous infusion of immunoglobulin.

Although CIDP is classified as a demyelinating disorder of the PNS, it does cause clinical or subclinical CNS problems, especially optic neuropathy, and needs cooperation between neurologists and ophthalmologists for more effective treatment.

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Sir,

Extensive globe rupture from blunt trauma: a case with a hope

Ocular trauma is a commonly encountered presentation to eye casualty. The majority of the injuries are minor, but over 10% of patients will lose useful vision in the injured eye.¹ Various aspects of serious ocular trauma, including the demography, prognostic variables, histopathological characteristics and role of vitrectomy, have been studied to identify features predictive of outcome and establish useful guidelines for management. We report a patient with extensive globe rupture who, despite the presence of uveal prolapse at scleral wound and an incomplete scleral wound closure, made a good visual recovery, which was against that predicted by outcome features.

Case report

A 28-year-old man presented to the eye casualty with his left eye having been hit by a metal bar at work. His visual acuity was perception of light on the left. Ocular examination revealed a full-thickness corneal laceration starting at the 9 o'clock hour position, extending downwards to the 7 o'clock hour position of the cornea and into the sclera, where it was obscured by extensive

subconjunctival haemorrhage. There was a total hyphaema precluding anterior chamber view with very soft digital globe pressure.

He underwent exploration and primary repair, where the scleral rupture was found to have extended down to the origin of the optic nerve, and the posterior edge of the laceration could not be visualised. There was prolapse of some uveal tissue along the scleral laceration but no evidence of vitreous incarceration. Primary repair of the corneal and scleral wound was performed to the limits of visualisation. A portion of the posterior extension (post-equator) of the scleral perforation was left unsutured.

He made a slow and uncomplicated visual recovery over the next few months requiring no secondary surgical procedure and achieving a best corrected vision of 6/18 with a refraction of -3.25 DS/+0.50 DC $\times 110^\circ$ in the left eye after a year.

Comment

A direct blow to the eye and surrounding tissues causes a blunt, contusional injury from simple 'black eye' to severe intraocular disruption, including rupture of the globe. An open-globe rupture is defined as a full-thickness wound of the eye wall (through and through injury of the sclera or cornea or both).² Full-thickness globe ruptures usually occur at the cornea-scleral limbus, under the insertion of one of the rectus muscles or around the entrance of the optic nerve posteriorly. There is often loss of the lens and prolapse of the uvea and vitreous at the time of the injury.¹

In the acute phase of presentation, detailed examination of the extent of the ocular injury is often difficult. Therefore the standard practice for most ophthalmologists has been to repair ocular wounds, whatever the initial vision and extent of injury. Scleral ruptures require primary closure as soon as possible, in order to restore the normal anatomical relationship of uveal tract or vitreous in the wound. This should allow orderly wound healing without the complication of abnormal fibrocellular ingrowth,³ a common finding in enucleated ocular specimens, which would hinder any further surgical procedures.

Advice and prediction on the prognosis at the early stage of injury, even after a primary repair, is often difficult. Various studies have therefore examined factors that are predictive of outcome, to facilitate decision-making on the necessity for enucleation and to avoid the development of sympathetic ophthalmia. In the study by Esmali *et al.*,³ predictors of excellent final visual acuity (6/9 or better) were: initial visual acuity of 6/60 or better, wound location anterior to the plane of insertion of the four rectus muscles, wound length 10 mm or less, and a sharp mechanism of injury. In contrast, predictors of poor vision were initial visual acuity of light perception or worse, wounds extending posterior to the rectus muscle insertion plane, wound length greater than 10 mm, and blunt or missile injury.

The nature and extent of ocular injury sustained by our patient would completely fit the latter group, which would mean a very grave prognosis and the likelihood of enucleation, as recommended by previous studies.^{3,4} Despite sustaining a large globe rupture, the presence of uveal prolapse at the scleral wound and an incomplete scleral wound closure, the patient made an excellent visual recovery. This case shows that the scleral texture could be much more resilient than expected and with its rich blood supply, self-healing is possible if the wound was opposable with no loss of tissue. The other predictor of favourable outcome in our patient was the moderate degree of prolapse but 'replaceable' uveal tissue at the wound, which did not require excision.

The study by Pieramici *et al.*⁴ supported the findings of Esmali *et al.*; in addition they found a decreasing trend for enucleation following severe ocular trauma compared with 20 years ago. The development of sympathetic ophthalmia in the other eye has always been a concern with penetrating injuries, but this possibility is not a good reason to proceed to enucleation. Indeed, sympathetic ophthalmia is extremely rare, with a prevalence of 0.1–0.3% in patients following traumatic ocular injuries, and is now considered to be a treatable condition using steroids and cytotoxic agents.⁵

In conclusion, assessment of serious ocular trauma and the early prediction of prognosis are often difficult despite the availability of known features predicting outcome. Each individual patient should be assessed carefully at the time of primary surgery, and enucleation to prevent sympathetic ophthalmia should be considered as a much later option.

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