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Sir,

Bilateral Ptosis, Tonic Pupils and Abducens Palsies Following *Campylobacter jejuni* Enteritis

There is a well-described relationship between *Campylobacter jejuni* gastroenteritis and subsequent Guillain–Barré syndrome. Some variants involving ophthalmoplegia[1] have been described including Miller–Fisher syndrome[2] and one reported case of isolated bilateral abducens palsies.[3] We report a case of bilateral ptosis, tonic pupils and bilateral abducens palsies following *Campylobacter jejuni* infection.

Case Report

A 21-year-old woman presented to the ophthalmology department with diplopia and ptosis of approximately 10 days' duration. Two weeks earlier she had suffered an episode of gastroenteritis proven to be due to *Campylobacter jejuni* by positive stool culture; she was treated with a course of ciprofloxacin and she recovered within a week.

On examination, she had normal visual acuities. Both pupils were noted to be dilated, with sluggish reaction to light and a tonic reaction to a near target. Vermiform movements of the irides were noted on slit lamp examination. She had a moderate degree of ptosis bilaterally. Examination of the ocular movements showed limitation of abduction of both eyes to just beyond the midline. Horizontal jerk nystagmus

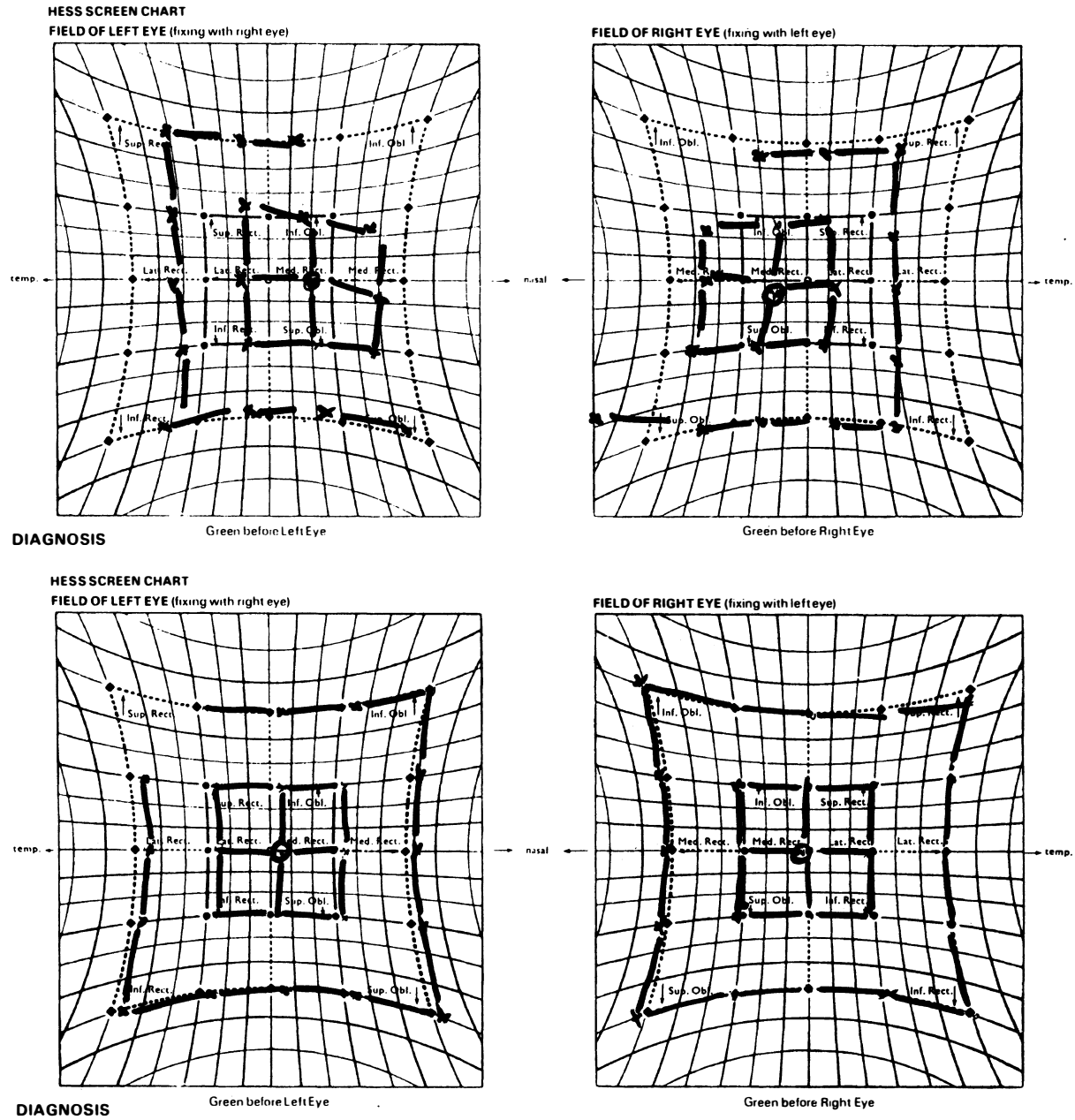

Fig. 1. *Serial Hess charts.*

was noted on attempted lateral gaze. There were no other neurological signs; specifically, she was not ataxic and deep tendon reflexes were intact.

Guttae pilocarpine 0.1% instilled into both eyes produced pupillary constriction showing denervation supersensitivity. Serial Hess charts were recorded (Fig. 1). Skeletal muscle autoantibody and acetylcholine receptor antibody analysis proved negative.

The ptosis resolved spontaneously within 4 weeks and the ocular movements were normal by 8 weeks: she was noted still to have tonic pupils at 12 weeks and these remained unchanged 6 months after initial presentation.

Discussion

Since Rhodes and Tattersfield[4] first reported a case of Guillain–Barré syndrome following enteric infection with *Campylobacter jejuni* in 1982, there have been 21 more reports of individual patients and series of patients with Guillain–Barré or its variants preceded by *C. jejuni* infection. Other specific infectious agents associated with Guillain–Barré include *Mycoplasma pneumoniae*, hepatitis B virus, cytomegalovirus, Epstein–Barr virus, varicella-zoster virus, rubeola virus and HIV. The development of a tonic pupil has also been reported following infectious disorders and pupillary function can be affected in Miller–Fisher syndrome.

The hallmark of Guillain–Barré syndrome is segmental demyelination of peripheral nerves, with mononuclear infiltration and oedema. The exact pathogenic mechanism underlying the association between *C. jejuni* infection and Guillain–Barré syndrome has not been established. Several theories have been forwarded: myelin destruction may be mediated by a direct toxic effect or by an immunopathogenetic mechanism. Mishu and Blaser[5] suggest that perhaps only a few *C. jejuni* strains are capable of triggering immunologically mediated myelin destruction. They also suggest a specific genetic predisposition. Kaldor and Speed[6] propose that *C. jejuni* antibodies may trigger demyelination by an immunological cross-reaction between *C. jejuni* and neural tissue, or that a specific enterotoxin produced by *C. jejuni* may cause direct neural damage. A cell-mediated immune mechanism is another possibility.

This case may represent a limited variant of Miller–Fisher syndrome. The development of tonic pupils is interesting as the neural lesion must be in the ciliary ganglion or the post-ganglionic parasympathetic pathway. This would seem to suggest that a mechanism other than myelin destruction has occurred in this case.

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Sir,

Alkaline Chemical Ocular Injury from Emla Cream

Emla cream is widely used to decrease the pain of intravenous cannulation in children[1] by producing anaesthesia of the overlying skin, by applying it topically under an occlusive dressing. We report here two children who had features of an alkali eye injury due to accidental self-application of Emla cream to the eye.

Case Reports

A 7-year-old boy was admitted for an elective general surgical procedure. Before the surgery 2 g of Emla cream, under an occlusive dressing, was applied to the dorsum of one of the hands. Approximately 30 minutes later, the nursing staff noted that the patient was rubbing his right eye which was red and watering. The patient was not complaining of pain or discomfort. The occlusive dressing that covered the Emla cream was found to have ruptured and the cream had exuded out, and some was present on the cheek. It was assumed that the Emla cream was responsible for the patient's symptoms. A quick initial examination of the patient revealed an anaesthetic ocular surface and thus irrigation of the eye was begun with normal saline solution. Further findings on examination were conjunctival injection, a corneal abrasion and a normal intraocular pressure. The patient was treated with guttae betamethasone 0.1% q.d.s. and guttae cyclopentolate 1% b.d. The child made a full