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.

B. N. Roberts, MRCOphth
P. V Mills, FRCOphth
N. Hawksworth, FRCOphth
Cardiff Eye Unit
University Hospital of Wales
Heath Park
Cardiff
South Glamorgan CF4 4XW, UK

References

1. Wroe SJ, Blumhardt LD. Acute polyneuritis with cranial nerve involvement following *Campylobacter jejuni* infection (letter). J Neurol Neurosurg Psychiatry 1985; 48:593.
2. Kohler A, de Torrente A, Inderwildi B. Fisher's syndrome associated with *Campylobacter jejuni* infection. Eur Neurol 1988;28:150–1.
3. Krujic RA, Lampe AS, Endtz H. Bilateral abducens paresis following *Campylobacter jejuni* enteritis. J Infect 1992;24:215–6.
4. Rhodes KM, Tattersfield AE. Guillain–Barré syndrome associated with *Campylobacter* infection. BMJ 1982;285: 173–4.
5. Mishu B, Blaser MJ. Role of infection due to *Campylobacter jejuni* in the initiation of Guillain–Barré syndrome. Clin Infect Dis 1993;17:104–8.
6. Kaldor J, Speed BR. Guillain–Barré syndrome and *Campylobacter jejuni*: a serological study. BMJ 1984; 288:1867–9.

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

recovery the following day and the visual acuity returned to normal (6/6 unaided). The topical medications were continued for a week.

A 10-year-old boy was admitted for a urological examination under general anaesthesia. The patient had Emla cream applied to the dorsum of both hands. The child, like the case above, was found an hour later to have a red, watering eye; the ocular surface and surrounding skin was anaesthetic, but he was not complaining of any discomfort. The occlusive dressing was not intact and the contents had exuded out. It was presumed that the Emla cream was responsible and the patient was immediately treated by irrigating the eye with normal saline solution. Examination of the eye later revealed mild conjunctival injection, no limbal ischaemia, a large corneal abrasion and a normal intraocular pressure. The patient was treated with guttae betamethasone 0.1% six times dialy and cyclopentolate 1% b.d. for 1 week. The boy made a full recovery 2 days later; visual acuity returned to normal (6/5 unaided).

Discussion

Emla cream is an alkaline formulation (pH 8–9) to allow the penetration of the local anaesthetic agents. Each gram of cream contains 25 mg lidocaine base and 25 mg prilocaine, in a eutectic mixture as an oil–water emulsion, its other main component being sodium hydroxide (7%).[2] Emla cream has been used for skin anaesthesia for minor lid surgery;[3] no mention was made of any long-term side-effects but some patients had conjunctival injection and corneal staining. Emla cream is an effective topical anaesthetic but, as illustrated in these two cases, can mask a chemical ocular injury, due to lack of pain or discomfort, unless the other signs of injury are noted, and can thus lead to delay in presentation.

A. K. Brahma
C. Inkster

Manchester Royal Eye Hospital
Manchester M13 9WH, UK

References

1. Clarke S, Radford M. Topical anaesthesia for venepuncture. Arch Dis Child 1986;61:1132–4.
2. Emla Cream 5%, Astra Pharmaceuticals Ltd. ABPI data sheet compendium. London: Datapharm Publications, 1993–4:107.
3. Gotsis SS, Volonaki OM, Theodossiadis GP. Percutaneous anaesthesia with a lignocaine–prilocaine cream (Emla) for eyelid skin surgery. Br J Ophthalmol 1994; 78:209–10.

Sir,

Retinal Haemorrhage in Meningitis

Fundal complications in meningitis occur in around 5% of cases, varying from disc abnormalities to chorioretinitis. We report a case of subretinal haemorrhage in a patient with meningococcal meningitis. Intraocular haemorrhage is known to occur after subarachnoid haemorrhage but, as far as we know, there has been no association of intraocular haemorrhage with bacterial meningitis.

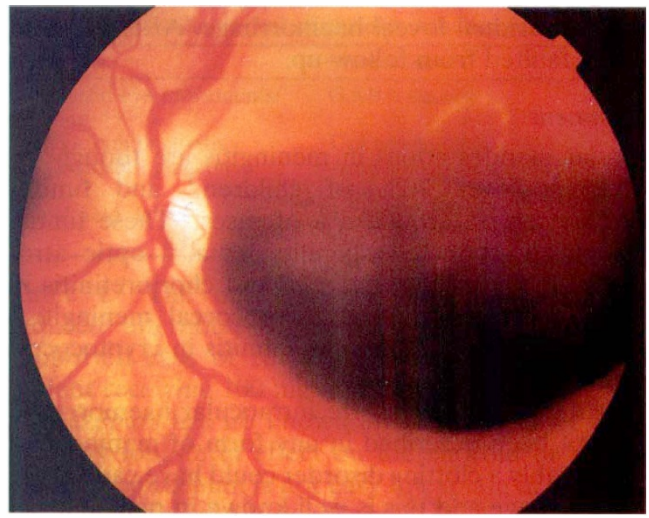

Fig. 1. *The large subretinal and vitreous haemorrhage in the left eye.*

Case Report

A 12-year-old girl was admitted as an emergency under the paediatrician service with a 1 day history of generalised headache and vomiting. On examination, she was found to be drowsy and pyrexial with a stiff neck; a presumptive diagnosis of meningitis or encephalitis was made. Her pupil reactions were described as normal but there was no comment on funduscopy, she had no purpura and clotting was normal. She had an immediate CT scan which was normal (with normal ventricular size), and a subsequent lumbar puncture that yielded a turbid fluid; the pressure was not measured – as is common in paediatric patients. A diagnosis of bacterial meningitis was made and high-dose intravenous penicillin started immediately.

Two days later *Meningococcus* was grown from the cerebrospinal fluid. The patient made a slow but steady recovery until 6 days after admission, when she complained of blurred vision in her left eye and she was referred to the eye department.

When seen in the eye department she had a visual acuity of 6/9 in her right eye but only counting fingers in the left; there was no afferent pupillary defect but dilation revealed a large subretinal and vitreous haemorrhage in the left eye (Fig. 1) but a normal right fundus. One month after the initial event her vision remained at counting fingers and she developed a left convergent squint; the subretinal blood was unchanged. Eight weeks after admission her vision improved to 6/9 and funduscopy revealed only