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.

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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.

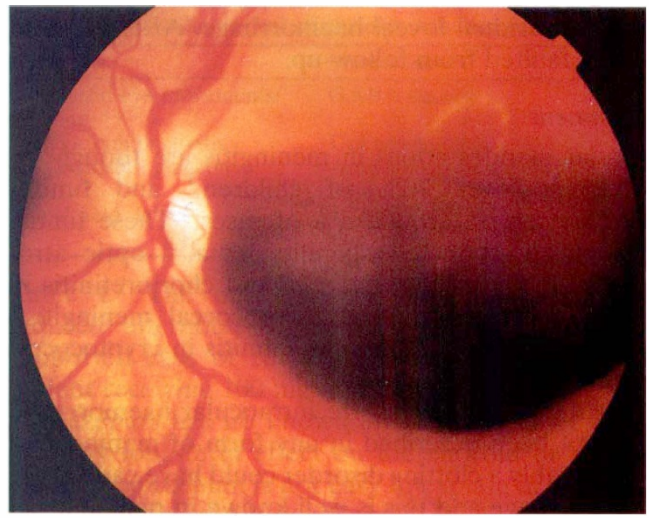

Fig. 1. *The large subretinal and vitreous haemorrhage in the left eye.*

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

a small residual foveal haemorrhage. After this visit she defaulted from follow-up.

Comment

Ocular manifestations in meningitis are thought to occur in over 70% of children,[1] and fundus involvement in around 5% of cases.[2] These fundal complications include papilloedema,[1,2] optic atrophy,[1,2] temporal disc pallor[1,2] and chorioretinitis in both tuberculous[1–3] and cryptococcal meningitis,[4] and panuveitis and endophthalmitis in cryptococcal meningitis.[4]

As far as we are aware intraocular haemorrhage has not been described in any form of meningitis – conceivably a clotting disorder could have caused the haemorrhage and this is well known in meningococcal meningitis,[5] but clotting studies were normal throughout the patient's illness.

Intraocular haemorrhage is a well-recognised complication of subarachnoid haemorrhage;[6] it is thought to be caused by the transmission of intracranial pressure along the optic nerve with subsequent nerve sheath dilation and rupture of dural and bridging vessels. Retinal venous hypertension brought on by obstruction of the central retinal vein may be the source of the haemorrhage.[7]

It seems conceivable that our patient could have had a rise in intracranial pressure at some stage in her illness and this could have precipitated a retinal venous haemorrhage. Although the cerebrospinal fluid pressure is often not taken in the acute situation, the presence of drowsiness can indicate raised intracranial pressure.

Whatever the underlying cause for the haemorrhage, resolution was spontaneous (up to the time of default) and it would appear that the initial management of these situations should be conservative.

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Sir,

Maculopathy Associated with Diazepam

There is a well-known affinity between diazepam and mammalian retina,[1,2] but there have been no reports of associations between the drug and retinal degenerations. We present such a case, and discuss the evidence suggesting that this may be more than a chance association.

Case Report

A 40-year-old male Caucasian was referred by his optician after asymptomatic macular abnormalities were found on routine examination. He had no ocular history and no family history of ocular disease. He had a long history of anxiety complicated by excessive alcohol intake, and had been taking up to 30 mg daily of diazepam for the previous 7 years. In addition he had received short courses of tricyclic antidepressants, monoamine oxidase inhibitors, fluoxetine and propranolol. Other benzodiazepines such as temazepam had also been used in the past.

The patient was emmetropic and saw 6/9 in the right eye and 6/6 in the left. Both fundi showed macular atrophic changes with scattered hyperpigmentation (Fig. 1). Fluorescein angiography confirmed retinal pigment epithelial degenerative change (Fig. 2).

On further examination 3 months later there was no change. The patient did not attend for electro-oculography (EOG) and electro-retinography (ERG) and has evaded follow-up despite our efforts since.

Discussion

Diazepam has been commonly prescribed to treat anxiety, insomnia, epilepsy and muscle spasms over the last 25 years. In recent years long-term therapy has been discouraged due to well-publicised central side-effects including tolerance and dependence, and difficulties in drug withdrawal.[3] Doses of 6–15 mg