SHORT REPORTS

Swimming goggles causing severe eye injuries

In 1976 in this hospital, which serves a population of about 1.5 million, we have seen three cases of severe eye injuries caused by a popular type of swimming goggle. The children affected were pulling the goggles forward in order to clear them of steam, when they slipped out of their wet fingers.

Case reports

Case 1—A 13-year-old girl was hit in the right eye by a swimming goggle. On examination half an hour later the visual acuity of the injured eye was 6/60 and in the left eye 6/6. There was a penetrating injury about 8 mm in length across the centre of the cornea; the anterior chamber was flat and contained a blood clot preventing a view of the fundus. The wound was sutured under general anaesthesia the same day. The eye was treated with a pad, and topical cycloplegics and antibiotics, and later steroids, but remained irritable for several weeks. Some 13 weeks after the injury the eye had settled well and the wound had healed (see figure). The visual acuity at this stage was counting fingers at one foot, owing to dense cataract. Future treatment may improve this result, but some visual loss will be permanent.

Case 2—A 13-year-old boy was hit in the right eye by a swimming goggle three days before he was examined, complaining of a "bloodshot" eye and blurred vision. On examination the visual acuity of the injured eye was 6/9 and of the left eye 6/5. There was 1.3 settled hyphaema present and slight traumatic mydriasis; and on examination of the fundus no abnormality was seen. Ten days later the eye had settled completely and the visual acuity was 6/5.

Case 3—A 14-year-old boy was hit in the right eye by a swimming goggle a week later. Examination of the fundus showed a 6-mm (predominantly lymphocytes, 61 mm³), protein 1.1 g/l, and sugar 3.1 mmol/l (56 mg/100 ml). Viral encephalitis was diagnosed. Her condition deteriorated alarmingly, however, and she was transferred.

On arrival in our intensive care unit the patient had a decerebrate posture and responded only to painful stimuli. Rectal temperature was 38.3 °C, generalised convulsions with tonic rotation of the neck were evident, there was pronounced cervical rigidity, and Kernig’s sign was present. The optic discs looked normal. Respiratory function was depressed, and the required intubation and assisted ventilation. Lumbar puncture showed the CSF to contain 23-10⁶ white cells (predominantly lymphocytes, 61 mm³), protein 115 g/l, and sugar 5.9 mmol/l (106 mg/100 ml) (blood sugar 8.1 mmol/l; 146 mg/100 ml). Further CSF studies showed Wassermann reaction negative, Lane curve 432222100, and no organisms, virus, or mycoplasmas. Haemoglobin was 11.9 g/dl, white cell count 14.10⁹/l (14000/mm³); 98% neutrophils, MCV 74 fl (um²), erythrocyte sedimentation rate 90 mm in first hour, platelet count 490.10⁵/l, and Paul-Bunnell reaction negative. Urea, electrolytes, liver function values, and plasma amylase were all within normal limits. Blood culture was sterile. Chest and skull x ray pictures and an EMI scan were normal. An electroencephalogram was devoid of normal activity and showed changes compatible with a severe encephalitic process.

In view of the severity of the illness and signs of generalised toxicity intravenous ampicillin was started. The raised MCV was found to be due to autogglutination of the patient’s cells in the Coulter counter. A spot test confirmed the presence of cold haemagglutinin. Tetracycline treatment by nasogastric tube was begun when M. pneumoniae was suspected as the causative organism.

The patient gradually recovered and the endotracheal tube was removed 48 hours later. She was eventually discharged home two months after the onset of her illness with no residual neurological deficit. At outpatient follow-up she appeared to be well physically but still had some impairment of intellectual function and remained very depressed.

Comment

To my knowledge there have been no reports of similar injuries caused by swimming goggles, which are worn by many children. The goggles concerned were made by different manufacturers, but the design was roughly the same, and all had a strong elastic strap. It is not possible to be certain which part of the goggle struck the eyes, and it may not always have been the same part. Because of the elasticity of the adjustable strap it is possible to pull the goggles 30-40 cm away from the face, and on release they spring back with considerable force. It would seem safer, although more inconvenient, to use a less elastic or non-elastic adjustable strap, or even a Velcro fastener if possible.

I am grateful to the surgeons of this hospital for permission to report these cases, and to Mr C Hood for photography.

(Accepted 22 December 1976)

Eye Department, Royal Infirmary Edinburgh, Edinburgh EH3 9HA
F JONASSON, CAND MED ET CHR, DO, registrar

Raised mean cell volume and meningoencephalitis associated with Mycoplasma pneumoniae infection

Mycoplasma pneumoniae infections are not usually associated with disorders of the central nervous system (CNS). The organism, however, should be considered as a causative agent in all cases of acute neurological dysfunction, particularly in young patients. In the following case the diagnosis was suspected owing to a raised mean cell volume (MCV) resulting from cold agglutination of the patient’s cells in the Coulter counter.

Case report

A 30-year-old housewife was transferred to this hospital in April 1976. She had been admitted to Ashford Hospital, Middlesex, three days earlier with a two-week history of depression, diplopia, and hesitancy of micturition after a "flu-like" illness. Examination of the cerebrospinal fluid (CSF) at Ashford had shown 61 10⁶ white cells (predominantly lymphocytes, 61 mm³), protein 115 g/l, and sugar 5.9 mmol/l (106 mg/100 ml). Viral encephalitis was diagnosed. Her condition deteriorated alarmingly, however, and she was transferred.

On arrival in our intensive care unit the patient had a decerebrate posture and responded only to painful stimuli. Rectal temperature was 38.3 °C, generalised convulsions with tonic rotation of the neck were evident, there was pronounced cervical rigidity, and Kernig’s sign was present. The optic discs looked normal. Respiratory function was depressed, and the required intubation and assisted ventilation. Lumbar puncture showed the CSF to contain 23 10⁶ white cells (predominantly lymphocytes, 61 mm³), protein 115 g/l, and sugar 5.9 mmol/l (106 mg/100 ml) (blood sugar 8.1 mmol/l; 146 mg/100 ml). Further CSF studies showed Wassermann reaction negative, Lane curve 432222100, and no organisms, virus, or mycoplasmas. Haemoglobin was 11.9 g/dl, white cell count 14 10⁹/l (14000/mm³); 98% neutrophils, MCV 74 fl (um²), erythrocyte sedimentation rate 90 mm in first hour, platelet count 490 10⁵/l, and Paul-Bunnell reaction negative. Urea, electrolytes, liver function values, and plasma amylase were all within normal limits. Blood culture was sterile. Chest and skull x ray pictures and an EMI scan were normal. An electroencephalogram was devoid of normal activity and showed changes compatible with a severe encephalitic process.

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The patient gradually recovered and the endotracheal tube was removed 48 hours later. She was eventually discharged home two months after the onset of her illness with no residual neurological deficit. At outpatient follow-up she appeared to be well physically but still had some impairment of intellectual function and remained very depressed.

Comment

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Eye Department, Royal Infirmary Edinburgh, Edinburgh EH3 9HA
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An initial *M pneumoniae* complement-fixing antibody titre was 1/1028; two weeks later the titre was again 1/1028, indicating a recent infection.

Comment

Four types of CNS disorder have been described in association with *M pneumoniae* infections—meningoencephalitis, psychosis, meningitis, and radiculopathy. Antecedent or concurrent pulmonary disease is common but not uniformly present.\(^2\) The nature of the neurological lesion is not clear but direct invasion of the CNS is unlikely. A neurotoxin has been suggested but has never been isolated from cases in man.\(^3\) The diagnosis is suspected by the detection of cold haemaggutinins\(^4\) (discovered in the present case owing to an abnormal MCV) and is confirmed by measuring either serial complement-fixing or growth-inhibiting antibody titres. A definite diagnosis is important because, although antimicrobial treatment is ineffective,\(^5\) the prognosis for a full recovery is generally good.

I thank Dr Maurice Gross for permission to report this case, and Professor H P Lambert for helpful advice.


(*Accepted 6 January 1977*)

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**Pregnancy and lactation after pituitary surgery**

We report the clinical and biochemical features of a patient with a prolactin-secreting pituitary adenoma who conceived after transsphenoidal surgery and who lactated in the puerperium despite unusually low postpartum serum prolactin concentrations.

**Case report**

A 34-year-old woman presented to the endocrine clinic in November 1973 with a six-month history of amenorrhoea (after 10 years' oligomenorrhoea) and a five-year history of gradually increasing body hair. She was a thin woman (54.8 kg) with normal secondary sexual development and mild hirsutism of the face, trunk, and limbs. The results of investigations at this time included normal basal gonadotrophin concentrations, thyroxine, testosterone, and 24-hour urine oestrogen steroids and oestrone. At the time of presentation she was in her 30th week of gestation and a normal-sized, non-stressed fetus was palpated. The 24-hour urine oestrogen excretion was 273 ng. An x-ray film was reported as normal. She was given two courses of clomiphene citrate but failed to ovulate. At routine follow-up in August 1974, a serum prolactin concentration measured for the first time was found to be very high, at 350 μg/l (normal <15 μg/l VLS-1). Again, the concentrations of gonadotrophins, both basal and LH-RH-stimulated concentrations, were within the normal range. Tomography of the pituitary fossa showed an asymmetrical enlargement consistent with the presence of a prolactin-secreting pituitary tumour. A lumbar air encephalogram (AEG) showed no evidence of suprasellar extension of the tumour.

In July 1975 a transphenoidal adenomectomy was performed (Mr R A Williams). Postoperatively her prolactin level was 11.4 μg/l; gonadotrophin, TSH, and ACTH secretion were normal. Ovulatory menstruation resumed within a month of surgery. In 1976, her last menstrual period was on 15 October 1975, and pregnancy was confirmed in December 1975. She had an uncomplicated pregnancy and delivered a normal child on 28 July. Postnatally her breasts became engorged and she was able to breast feed normally until six weeks postpartum, when she elected to change to bottle feeding. Prolactin concentrations throughout the pregnancy remained at about 15 μg/l and did not change significantly in the puerperium (see figure). Despite adequate lactation there was no rise in prolactin in response to suckling at one week postnatally. There was a small increase in prolactin after TRH (see figure).

**Discussion**

The association of hyperprolactinaemia and amenorrhoea, irrespective of the presence of galactorrhoea, is now well established, and this patient shows many of the typical features of this syndrome.\(^6\) In this case the finding of a very high prolactin concentration was associated with the presence of radiological changes of a pituitary tumour. Treatment of the patient with a radiologically apparent prolactinoma who wishes to become pregnant is controversial\(^7\) but at this centre transphenoidal surgery is the treatment of choice when the AEG indicates that there is minimal or no suprasellar extension.\(^8\) After removal of the adenoma her serum prolactin concentration fell to within the normal range; there was no evidence of hypopituitarism; and ovulatory menstruation resumed without further treatment within a month of surgery. This illustrates that the reproductive disorder of hyperprolactinaemic amenorrhoea is readily repaired by effective reduction of prolactin, whether by medical or surgical means.

In the normal woman prolactin concentrations rise steadily during pregnancy, to reach levels of about 100 μg/l in the third trimester, and remain high in the week after delivery. Thereafter, basal levels may return to normal, but, for two to three months suckling produces a steep rise in prolactin concentrations.\(^9\) In our patient prolactin concentrations remained relatively low, and unchanged, during pregnancy and the puerperium and suckling at one week postpartum failed to produce a rise of prolactin. Nevertheless, this patient was able to lactate adequately. Lactation may occur without a rise in prolactin concentrations, as in the case of some patients who have galactorrhoea,\(^1\) and in normal women studied late in the puerperium.\(^3\) The reason for this is not yet entirely clear, though recently it has been postulated that prolactin can induce and maintain its own receptor population in mammary tissue.\(^1\) Hence the concentration of 15 μg/l in this patient was possibly sufficient to maintain an adequate number of prolactin receptors in the breast, and thereby sustain lactation.

The reagents for the prolactin assay were generously supplied by the National Pituitary Agency of the United States of America.


(*Accepted 20 December 1976*)

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