LETTERS TO THE EDITOR

Isolated intracranial hypertension presenting with trigeminal neuropathy

Isolated intracranial hypertension (pseudotumour cerebri) is an idiopathic condition characterised by raised intracranial pressure in the absence of a cerebral mass lesion or hydrocephalus. By definition symptoms and signs are restricted to those of raised pressure including papilloedema and abducens nerve palsies. Other cranial nerve palsies in association with this disease are rare and we report a case presenting with headache and unilateral trigeminal sensory disturbance.

In December 1991, a 20-year-old woman presented to the casualty department with headache and numbness initially affecting the right side of the lips before spreading to involve the right side of the face. She had restarted the oral contraceptive pill two months previously after a one year break. There was no other relevant history.

On examination she was obese with impaired sensation to light touch and pinprick affecting all three branches of the right trigeminal nerve together with an ipsilateral attenuated corneal reflex. Trigeminal motor function was intact and there were no other neurological signs; in particular, fundoscopic examination was normal.

Her symptoms persisted and on review seven days later, examination of the optic fundi revealed bilateral haemorrhagic papilloedema with enlarged blind spots but normal visual acuity; the trigeminal sensory signs remained unchanged. She was admitted for further investigations. A contrast-enhanced CT brain scan was normal and lumbar puncture revealed an opening pressure of 390 mm CSF. The cerebrospinal fluid was acellular with a protein content of 0·3 g/l. Her symptoms improved rapidly after the lumbar puncture and within 48 hours facial sensation had returned to normal and the headache had resolved completely. The contraceptive pill was stopped and she was discharged on acetozolamide. At review a week later examination confirmed normal trigeminal sensory function and resolving papilloedema. The lumbar puncture was repeated and the opening pressure was 190 mm of CSF with normal CSF constituents. Six weeks later her optic discs appeared normal and the acetozolamide was gradually withdrawn. The patient has since been reviewed regularly and has remained asymptomatic for 12 months.

Suggested diagnostic criteria for isolated intracranial hypertension comprise a raised CSF pressure of normal constituents, a normal cranial CT image, and symptoms and signs of raised intracranial pressure alone.[1] The patient in this case satisfied the first three criteria and no other explanation for the trigeminal sensory loss was discovered on examination or investigation. The close temporal relation between the reduction of CSF pressure and a resolution of symptoms and signs suggests that the trigeminal sensory loss may have been a pressure related phenomenon.

Trigeminal palsies may occur as false localising signs secondary to brain tumours.[2] The postulated mechanisms for this occurrence are direct compression of the trigeminal root by cerebral tissue, traction of the nerve by caudal displacement of the brainstem, or vascular disturbance secondary to the first two insults. Whereas abducens nerve palsies are well recognised in association with isolated intracranial hypertension (9%-36% of cases),[3] involvement of other cranial nerves has been described infrequently. Oculomotor,[4] trochlear,[5] and facial nerve[6] palsies have all been reported, most recently in this journal.[7] To our knowledge only one case report of an isolated trigeminal lesion in association with isolated intracranial hypertension exists.[3] This patient presented with a six year history of intermittent symptoms culminating in 12 months of recurrent facial pain. The late development of headache and papilloedema provided the diagnostic clue with resolution of symptoms and signs on appropriate treatment. The current case is a more acute presentation of trigeminal disturbance with numbness rather than pain. Numbness of the face was also reported by Zachariah *et al*[8] in a patient with isolated intracranial hypertension but the presence of an ipsilateral hemiparesis suggested a higher level of cerebral involvement.

Our report reinforces the fact that patients with isolated intracranial hypertension may present with disturbance of trigeminal function, albeit rarely. We concur with Davie *et al*[7] that the rapid resolution of symptoms and signs after lumbar puncture in these cases suggests that the signs are falsely localising in nature and as such are compatible with the diagnosis. Recognition of this association may avoid unnecessary investigation in such patients.

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1 Ahlskog JE, O'Neill BP. Pseudotumour cerebri. *Ann Intern Med* 1982;**97**:249-56.
2 O'Connell JEA. Trigeminal false localising signs and their causation. *Brain* 1978;**101**: 119-42.
3 Hart RG, Carter JE. Pseudotumour cerebri and facial pain. *Arch Neurol* 1982;**39**:440-1.
4 McCammon A, Kaufman HH, Sears ES. Transient oculumotor paralysis in pseudotumour cerebri. *Neurology* 1981;**31**:182-4.
5 Helpern JI, Gordon WH. Trochlear nerve palsy as a false localising sign. *Ann Ophthalmol* 1981;**13**:53-6.
6 Kiwak KJ, Levine SE. Benign intracranial hypertension and facial diplegia. *Arch Neurol* 1984;**41**:787-8.
7 Davie C, Kennedy P, Katifi HA. Seventh nerve palsy as a false localising sign. *J Neurol Neurosurg Psychiatry* 1992;**55**:510-1.
8 Zachariah SB, Jimenez L, Zachariah B, Prockop LD. Pseudotumour cerebri with focal neurological deficit. *J Neurol Neurosurg Psychiatry* 1990;**53**:360-1.

Spinal intrathecal baclofen suppresses central pain after a stroke

Central pain due to cerebral stroke is one of the most difficult of all pain syndromes to ameliorate. Medical treatment is usually unsatisfactory and surgical intervention in the thalamus or the midbrain may be indicated. We had experience of a patient who was given lumbar intrathecal baclofen for spasticity associated with dysaesthesic pain in his extremities, after a stroke. The pain was suppressed appreciably with a small dose (25 μg) of intrathecal baclofen, which did not affect the spasticity. Encouraged by this experience, we investigated the effect of intrathecal baclofen in five patients with central pain after a stroke.

The patients were admitted to the neurosurgical ward for this investigation. All the patients had been treated medically without significant pain relief. Intravenous morphine was also ineffective. No patient had been taking oral baclofen. Intrathecal baclofen was given once a day through a lumbar puncture at the L3-4 level. The patients were asked to report their subjective pain hourly with a 10-grade score, in which 0 = no pain and 10 = pain at the pretreatment level. The injection was repeated three to five times during a week. Normal saline was used once to exclude a placebo effect. The placebo was given between baclofen injections. The baclofen solution for intrathecal use was supplied by Ciba Geigy Corporation (Basel, Switzerland). The original solution (0.5 mg/ml) was diluted 10 times with normal saline for bolus injection. The experimental nature of this investigation and its possible risk were explained to the patients and the family and informed consent to the use of baclofen for central pain was obtained. The procedure was approved by the ethics committee of the Tokyo Women's Medical College.

Case 1

A 60-year-old man had had severe constant dysaesthetic pain in his left upper and lower limbs for five years. The cause was a small haemorrhage in the right posterior thalamus. Allodynia to light touch and anaesthesia to pinprick were noted on the left side of his body. A bolus of intrathecal baclofen (50 μg) was given and he was then allowed to walk around as usual. After one hour he reported considerable reduction (2/10) of the leg pain and after four hours the arm pain was relieved (3/10). The allodynia was also relieved but anaesthesia to pinprick was not affected. The pain relief lasted for about 24 hours. We repeated the same procedure twice and obtained a consistent response. Placebo gave no pain relief. He reported a transient headache after the injection.

Case 2

A 57-year-old woman had had intractable pain for 20 years after a small haemorrhage in the left pons. Her pain was in the right arm and leg. It was constant pain of dysaesthetic and burning nature. Anaesthesia to pinprick was noted in the painful area but allodynia was not observed. We gave 50 μg of intrathecal baclofen, which resulted in good pain reduction (4/10). The pain relief started from the leg and then progressed to the upper arm in three hours. There was no objective change in sensation. The effect continued for about 12 hours. We gave baclofen five times and increased the dose gradually, up to 150 μg, which resulted in pain relief of longer duration. Her gait became unsteady with this dose. Placebo was not effective.

Case 3

A 57-year-old woman had a severe burning pain in her right extremities that developed