Isolated intracranial hypertension presenting with trigeminal neuropathy

Isolated intracranial hypertension (pseudo-tumour cerebri) is an idiopathic condition characterised by raised intracranial pressure in the absence of a cephalic 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 vomiting 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 pin-prick 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.

The patient was reviewed 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 H2O. 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 acetazolamide. 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 Six weeks later her optic discs appeared normal and the acetazolamide was gradually withdrawn. The patient has since been reviewed regularly and has remained asymptomatic for 12 normal.

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. The patient in this case satisfied the first three criteria and no other explanation for the trigeminal sensory loss was discovered on investigation. The 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. The postulated mechanisms for this occurrence include 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), involvement of other cranial nerves has been described in a few isolated cases.5,6 and facial nerve palsies have all been reported, most recently in this journal. To our knowledge only one case report of an isolated trigeminal lesion in association with isolated intracranial hypertension exists. This patient presented with a six year history of intermittent symptoms culminating in 12 months of recurrent facial pain. The late development of CSF with normal constituents. Our report reinforces the fact that patients with isolated intracranial hypertension may present with disturbance of trigeminal function, albeit rarely. We cannot agree with Davie et al7 that the resolution of symptoms and signs after lumbar puncture in these cases suggests that the signs are falsely localising in nature and as such are of uncertain utility. Recognition of this association may avoid unnecessary investigation in such patients.

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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 had gained lumbar intrathecal baclofen for spasticity associated with dysaesthetic pain in his extremities, after a stroke. The pain was suppressed appreciably with a small dose (25 g) of intrathecal baclofen, which did not affect the spasticity. Noting this experience, we investigated the effect of intrathecal baclofen in five patients with central pain after a stroke.

The patients were admitted to the neurological ward for the investigation, and we were informed that the patients had been treated medically without significant pain relief. Intravenous morphine was also ineffective. No patient had taken 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 highest treatment 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 the other injections. The saline 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 injection. The ethics 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 dysesthetic pain in his left upper and lower limbs for five years. The cause was a small haemorrhage in the right posterior thalamus. Alloodynia to light touch and anaesthesia to pin-prick was noted on the left side of his body. A bolus of intrathecal baclofen (50 g) was given and he was then allowed to walk around as usual. After one hour he complained of considerable reduction (2/10) of the leg pain and after four hours the arm pain was relieved (3/10). The alloodynia was also relieved but anaesthesia to pin-prick 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 lorn. Her pain was in the right arm and leg. It was constant pain of dysesthetic and burning nature. Anaesthesia to pin-prick was noted in the painful area but allodynia was not observed. We gave 50 g 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 could be detected for about 12 hours. The patient gave baclofen five times and increased the dose gradually, up to 150 g, which resulted in pain relief of longer duration. Her gait improved steadily with this dose. Placebo was not effective.

Case 3
A 57-year-old woman had severe burning pain in her right extremities that developed...
two months after a small infarction in the left thalamus. She stated that the pain was constant, burning, and unbearable. There was pronounced allodynia to light touch and cold stimuli. Pinprick showed hyperesthesia. At first we gave 50 μg of baclofen intrathecally, with no pain relief. The dose was gradually increased to 150 μg but she reported no pain relief. There was transient urinary retention.

Case 4
A 62-year-old man had central pain due to cerebral haemorrhage in the left corona radiata. His pain was in the distal parts of the right upper and lower extremities. There was no allodynia. Hyperesthesia to pinprick was noted in the right half of his body. A bolus of 50 μg of intrathecal baclofen resulted in considerable pain reduction in an hour (1-2/10), that developed in the upper and lower limbs at the same time. The effect continued for about 12 hours. This response was confirmed three times with repeat intrathecal injection at the same dose. There was no sensory change with the injection.

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Effect of immobilisation on position and movement sense of the knee
It has been suggested that joint position and movement sense can be improved by practice or by specialised therapeutic techniques such as exercises.1 A change in motor imbalance after immobilisation may be attributed to proprioceptive deconditioning. To investigate these theories we used established measurement techniques2 to test normal knees in 18 patients after a minimum two weeks of immobilisation. Ten patients had been immobilised in a full length leg plaster after fractures of the ankle region and eight were on strict bed rest due to injuries of the unstented leg. Results were compared with those of 30 controls with no history of immobilisation.

Movement sense was tested by determining the threshold of perception of slow joint movement at a velocity of 0·5° per second. A motor extended or flexed the knee (starting position 35° flexion) via a system of pulleys connected to the extended and flexed joint subjects by a canvas sling wrapped around an inflatable boot. Four randomised tests were carried out in flexion and extension ranges. Subjects signalled appreciation of knee joint movement by depressing a hand-held switch. The range of movement traversed before detection (threshold angle) was extrapolated from the output of potentiometers placed in parallel with the main pulley circuit.

Position sense was tested by measuring the margin of error in the reproduction of the previously held knee joint positions. Tests were carried out within the range 20°-50° of knee flexion—the normal arc of movement during walking. This condition was operated—namely, active reproduction of an active movement, active reproduction of a passive movement, and passive reproduction of a passive movement. Tests involving active movement were not carried out on the plaster group because of potential effects of muscle weakness and lack of coordination on results. Achievement of target positions was signalled by depressing a hand-held switch.

Results were recorded directly onto computer disc and analysed with two-tailed t tests and one way analysis of variance. Tests of movement sense did not show any significant differences either between or within groups (figure). Movement was detected within a mean of 2°. There were no significant differences in results for position sense either between or within groups. Subjects were accurate to within a mean of 4°. There was a trend, which reached significance in controls (n = 13, p < 0·001), for greater inaccuracy in active reproduction of a passive movement compared with active reproduction of an active movement and passive reproduction of a passive movement.

In conclusion, results for controls were comparable with those of other studies on the knee joint.2,3 No significant differences were detected in position or movement sense between the knees of subjects experiencing altered mobility and weight-bearing conditions and normal controls. The applicability of the results of these tests to normal functional movement is uncertain and warrants further investigation. Results suggest, however, that functional deficits in non-neurological subjects after a period of altered weight-bearing and mobility of the knee joint may be due to factors other than adaptation of position and movement sense mechanisms. Position and movement sense seem to be resistant to changing physical states. The rationale of therapeutic techniques that purport to improve position and movement sense in neurologically intact patients should be reconsidered.
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