imaging either with CT or MRI to rule out mesial temporal infarcts should be mandatory before giving anticoagulant treatment.

We thank Dr K Niranjani Reddy for help in the neuropsychological assessment of the patient.

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Hearing loss as a false localising sign in raised intracranial pressure

Benign intracranial hypertension (BIH) is an idiopathic disorder characterised by headache and visual disturbances with papilloedema (unilateral or bilateral), in which a space occupying lesion or infective processes have been excluded by neuro-imaging, analysis of CSF, and additional ancillary investigations. Recently this Journal has carried a series of reports describing rare associated cranial nerve palsies.1,2,3 We report a case of left sided hearing loss occurring in conjunction with worsening symptoms of raised intracranial pressure in a patient with established BIH that resolved after lumbar puncture.

In December 1993, a 24 year old woman presented to the eye casualty department with a week's history of headache—worse on bending—nausea with intermittent vomiting, and visual blurring. She had been taking oral contraceptive pills but there was no other relevant drug or medical history. Examination showed obesity (95 kg/155 cm), bilateral papilloedema, and normal visual acuity although testing fields by confrontation showed a small central scotoma affecting the right eye and decreased temporal fields in the left eye. Computed tomography, brain MRI, and MR angiography were normal. Venous pressure testing was normal and clear and colourless CSF with an opening pressure of 36 cm CSF. The CSF constituents were normal, with a protein concentration of less than 0·1 g/l and 2·5 white cells per cubic mm. The CSF glucose to plasma ratio was normal. The following peripheral blood indices were also normal: full blood count, viscosity, urea and electrolytes, liver function tests, thyroid function tests, creatine, protein, venereal disease research laboratory test, and anticoagulation antibody. OKP-Humphrey field analysis showed pronounced peripheral field constriction. The initial management was withdrawal of the oral contraceptive pill. Diuretic treatment with chlorothalidone was commenced and dietetic referral arranged.

These measures initially resulted in considerable symptomatic improvement. After several months, however, the headaches recurred with increasing visual obscuration. These symptoms progressed to daily bilateral obscurations of vision for up to a minute despite diuretic treatment. In association with this she had noted increasing impairment of hearing in the left ear, with no associated tinnitus, in the week before readmission. Repeat neurological examination on readmission showed a left sided chronic papilloedema, with peripheral constriction of the fields but normal visual acuity. Neuro-otological examination confirmed clinical hearing loss in the left ear. The Weber test was normal. Pure tone audiometry showed a mild left sided conductive hearing loss, with an air bone gap ranging between 25 and 20 dB at 500 and 4000 Hz. A gap of 2500 Hz was 10 dB. Tympanometry showed normal middle ear pressure and compliance bilaterally. Repeat lumbar puncture subsequently confirmed raised pressure of 29 cm CSF. On the second occasion of lumbar puncture after the patient noted normalisation of the hearing acuity in the left ear. Repeat audiometry and tympanometry were performed showing resolution of the previously noted mild conductive hearing loss and unchanged tympanometry.

Abducens nerve palsies are described in between 9% and 36% of cases of benign intracranial hypertension as false localising signs. Other cranial nerve palsies occurring with this disorder are rare, but oculomotor, trochlear, trigeminal, and facial nerve palsies have been previously reported.4,5 It has been suggested that for these occurrences are direct compression of the nerve root by cerebral tissue, traction of the nerve by caudal displacement of the brainstem, or vascular disturbance as a consequence of the raised ICP.5 In this case there was not only a cause relation between the worsening symptoms of raised intracranial pressure and the development of left sided hearing loss but also rapid normalisation of hearing acuity on reduction of the raised intracranial pressure, suggesting that the hearing loss may have been a pressure related phenomenon. In the current case, however, the audiometric pattern was not indicative of a conductive hearing loss. A potential explanation is to infer an increase in the perilymphatic fluid pressure transmitted through the cochlear aqueduct as a result of the rise in CSF pressure. This might dampen the movement of the stapedial footplate and of the round window membrane giving a small conductive hearing loss. The alternative explanation of a small acoustic gap in the middle ear with effusion is unlikely given the normal tympanometry.

Although otological manifestations have been previously reported in raised intracranial pressure of varying aetiologies including benign intracranial hypertension,5,6 hearing loss is not normally considered in the context of false localising signs. It may be under recognised given the typically mild nature of the hearing loss9 and the wide prevalence of hearing loss in the normal population.

In a series of 20 patients with benign intracranial hypertension the commonest otological manifestations were objective pulsatile tinnitus and low frequency hearing loss.5 Both these symptoms improved transiently in all patients after lumbar puncture. In the longer term, these symptoms responded well to weight reduction and treatment with diuretics. Wider awareness of this association will allow such patients to avoid unnecessary investigation and benefit from appropriate explanation and reassurance.

**Letters to the Editor**

Bromate intoxication with polyneuropathy

Acute bromate intoxication is a rare event in neurological clinics. Previous reports described renal failure and hearing deficit in addition to nausea, vomiting, haemolytic anaemia, depressed consciousness, and seizure. We report a woman who attempted suicide by taking a hair permanent wave preparation. This resulted in acute bromate intoxication and she developed renal failure, deafness, and toxic polyneuropathy.

The 25 year old woman (body weight 57 kg) took 7·5 g sodium bromate in the suicide attempt in 1992. Nausea, vomiting, and diarrhoea developed rapidly. Stomach lavage and irrigation by activated charcoal were carried out at a local hospital. Tinnitus and dizziness occurred five hours later, followed by deafness. Blood urea nitrogen was 10 mg/dl (normal 7–20 mg/dl) and serum creatinine 1·4 mg/dl (normal 0·5–1·2 mg/dl for women). Oliguria was noted on the second day, with blood urea nitrogen increasing to 16 mg/dl and 3·5 mg/dl respectively. The urine sediment disclosed 3–5 red blood cells per high power field (normal 0–2), and 45–50 white blood cells per high power field (normal <5 per high power field). Proteinuria was present (protein >3 mg/dl but without casts). Serum bromide (Br−) concentration was 85 mg/l (none present in normal subjects). After 10 days, serum creatinine was resistant to furosemide. On the third day, serum urea nitrogen reached 48 mg/dl and creatinine 9·3 mg/dl. Because of deteriorating renal function she was referred to the Veterans General Hospital, which received three courses of haemodialysis.
Nerve conduction studies

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Latency (ms)</th>
<th>Velocity (m/s)</th>
<th>Amplitude (mV)</th>
<th>Minimum F- latency (ms)</th>
<th>SNAP amplitude (μV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R median</td>
<td>4.0</td>
<td>44.3</td>
<td>7.0-7.5</td>
<td>30.4</td>
<td>52.0</td>
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<tr>
<td>R ulnar</td>
<td>8.0</td>
<td>54.4</td>
<td>6.7-7.1</td>
<td>28.9</td>
<td>52.7</td>
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<tr>
<td>R peroneal</td>
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<td>46.1</td>
<td>4.1-4.7</td>
<td>57.2</td>
<td></td>
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<tr>
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<td>5.0</td>
<td>43.9</td>
<td>11.6-9.5</td>
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</tr>
<tr>
<td>Sensory:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R radial</td>
<td>3.2</td>
<td>43.8</td>
<td></td>
<td>10.7</td>
<td></td>
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<td>5.0</td>
<td>46.7</td>
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<td>R ulnar</td>
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<tr>
<td>R radial</td>
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<td>50.0</td>
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<td></td>
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</tr>
</tbody>
</table>

SNAP = Sensory nerve action potential; R = right.

Urine output increased to 2000 ml in the first 6 hours but was less than 100 ml per hour the following day. On day 7 she complained of pricking, tingling, and numbness of the distal limbs, extreme over the feet. The sensory problems worsened over the next few days. On day 7 she complained of pricking, tingling, and numbness of the distal limbs, extreme over the feet. The sensory problems worsened over the next few days.

Hearing problems were more difficult to describe, but she could hear the pinprick and light touch at the distal end of the limbs, and intact position and vibration sensations. Nerve conduction studies performed one month later showed decreased nerve conduction velocities and reduced amplitude of compound muscle action potentials and sensory action potentials in the limbs (table). No fibrillation potentials or positive sharp waves were found during needle electromyographic examination. Seven months later the distal limbs continued to be numb. Serum urea nitrogen was 26 mg/dl and serum creatinine was 2.2 mg/dl. The patient had recovered from her renal failure after seven weeks of conservative management. However, she remained deaf. The pathogenesis of hearing loss in renal failure patients presenting with deafness is not yet fully understood. Hearing problems could also occur as a result of ischemic stroke delayed in onset after internal carotid artery (ICA) occlusion.

Embolism across the circle of Willis

Embolism is one of the proposed causes of ischaemic strokes delayed in onset after internal carotid artery (ICA) occlusion. The embolus may arise from a “tail” of thrombus that lies at the top of the ICA, from the thrombus from the ICA, or from the thrombus from the ipsilateral external carotid artery passing through pial vessels of the ophthalmic artery. Another mechanism could be embolisation across the carotid siphon. Transcranial Doppler ultrasoundography in four patients with internal carotid artery occlusion detected embolic signals in the middle cerebral artery ipsilateral to the internal carotid occlusion. After surgery of the opposite stenosed internal carotid artery no signal was detected.

We describe a patient with clinical, radiological, and ultrasonographic evidence of left hemispheric infarction by embolic migration through the anterior communicating artery.

A 74 year old right handed man underwent right carotid endarterectomy six years earlier for right transient cerebral ischaemia. Conventional arteriography showed a stenosis greater than 70% of the right internal carotid artery and an occlusion of the left internal carotid artery. Six years later an ischaemic stroke was discovered that required transluminal angioplasty. During the procedure, as the auxillary catheter was removed from the aorta, the patient sustained a right hemispheric infarction. On admission to the stroke unit of the Salpêtrière Hospital two hours later, the patient was awake, but on neurological examination there were no comprehension of any order or oral expression. He had a right hemiparesis with Babinski’s sign, right hemianopia, and hypoesthesia. General physical examination, a chest radiograph, routine laboratory investigations, and ECG were normal. Brain CT showed two recent areas of hypodensity taking the contrast in the right frontal and left frontoparietal regions to correspond respectively to pial vessel infarcts in the territory of the right and left MCA (figure). Colour Doppler ultrasound of the neck vessels confirmed the old occlusion of the left internal carotid artery and showed a stenosis of 80% of the right internal carotid artery with a “tail” of fresh thrombotic material floating in the lumen. Peribortal directional Doppler detected no reverse flow in the ophthalmic artery. Transcranial Doppler ultrasonography showed full irradiation of the left middle cerebral artery by the anterior communicating artery with an acceleration of velocity in the right middle cerebral artery. Several mechanisms could be responsible for the ischaemic stroke in this patient. Firstly, it could be caused by embolic migration through an extracranio- orbital anastomosis. The source of embolic material could be directly the left external carotid artery.
Bromate intoxication with polyneuropathy.

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