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

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RUPAM BORGOHAIN
HARI RADHAKRISHNA
AJAY KUMAR SINGH
SURATH MOHANDAS
Department of Neurology,
Nizam's Institute of Medical Sciences,
Punjagutta, Hyderabad—500 482,
India
J JAGANMOHAN REDDY
Department of Imageology

Correspondence to: Dr Rupam Borgohain, Department of Neurology, Nizam's Institute of Medical Sciences, Punjagutta, Hyderabad—500 482, India.

- 1 Saunders RC, Murray EA, Mishkin M. Further evidence that amygdala and hippocampus contribute equally to recognition memory. *Neuropsychologia* 1984;22:785–96.
- 2 Press GA, Amaral DG, Squire LR. Hippocampal abnormalities in amnesic patients revealed by high resolution magnetic resonance imaging. *Nature* 1989;341:54–7.
- 3 Stephens RB, Stilwell DB. Arteries and veins of the human brain. Springfield, IL: CC Thomas, 1969:124–67.
- 4 Babin E, Megret M. Variations in the drainage of the basal vein. *Neuroradiology* 1973;6:154–61.
- 5 Levine SR, Twyman RE, Gilman S. The role of anticoagulation in cavernous sinus thrombosis. *Neurology* 1988;38:517–22.

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 neuroimaging, analysis of CSF, and additional ancillary investigations. Recently this *Journal* has carried a series of reports describing rare associated cranial nerve palsies.^{1–3} 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 contraceptives 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. Lumbar puncture produced 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 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 functions, creatine, protein, venereal disease research laboratory test, and anticardiolipin antibody. OKP-Humphrey field analysis showed pronounced peripheral field constriction. The initial management was

withdrawal of the oral contraceptive pill. Diuretic treatment with chlorthalidone was commenced and dietetic referral arranged.

These measures initially resulted in considerable symptomatic improvement. After several months, however, the headaches recurred with increasing visual obscurations. 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 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 lateralised to the right. The Rinne test was positive bilaterally. Pure tone audiometry showed a mild left sided conductive hearing loss, with an air bone gap ranging between 5 and 20 dB at all frequencies tested (250 to 4000 Hz). Tympanometry showed normal middle ear pressure and compliance bilaterally. Repeat lumbar puncture subsequently confirmed raised pressure of 29 cm CSF. On the day after the lumbar puncture 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 lesions have recently been reported.^{1–3} It has been suggested that potential mechanisms 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 either of the above.³

In this case there was not only a close 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 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 temporary coexistent middle ear 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,^{4–5} 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 loss⁵ 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.⁴ 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.

P J DORMAN
M J CAMPBELL
Department of Neurology
A R MAW
Department of Otolaryngology,
Bristol Royal Infirmary,
Upper Maudlin Street,
Bristol BS2 8HW, UK

Correspondence to: Dr P J Dorman, Neurosciences Trials Unit, Department of Clinical Neurosciences, University of Edinburgh, Bramwell Dott Building, Crewe Road, Edinburgh EH4 2XU, UK.

- 1 Zachariah SB, Jimenez L, Zachariah B, Prockop LD. Pseudotumour cerebri with focal neurological deficit. *J Neurol Neurosurg Psychiatry* 1990;53:360–1.
- 2 Davie C, Kennedy P, Katifi HA. Seventh nerve palsy as a false localising sign. *J Neurol Neurosurg Psychiatry* 1992;55:510–1.
- 3 Davenport RJ, Will RG, Galloway PJ. Isolated intracranial hypertension presenting with trigeminal neuropathy. *J Neurol Neurosurg Psychiatry* 1994;57:381.
- 4 Sismanis A. Otological manifestations of benign intracranial hypertension syndrome: diagnosis and management. *Laryngoscope* 1987;97:1–17.
- 5 Tandon PN, Sinha A, Kacker RK, Saxena RK, Singh K. Auditory function in raised intracranial pressure. *J Neurol Sci* 1973;18:455–67.

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.¹ 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 and creatinine rising to 16 mg/dl and 3 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 µg/ml (none present in normal subjects). Oliguria 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 and received three courses of haemodialysis.

Nerve conduction studies

Nerve	Latency (ms) onset	Velocity (m/s)	Amplitude (mV) distal/proximal	Minimum F- latency (ms)	SNAP amplitude (μ V)
Motor:					
R median	4.0	44.3	7.0/6.7	30.4	
R ulnar	4.0	54.4	6.7/7.1	28.9	
R peroneal	3.9	46.1	4.1/4.7	57.2	
R tibial	3.7	43.9	11.8/9.5	52.7	
Sensory:					
R sural	3.2	43.8			10.7
R median	3.0	46.7			25.1
R ulnar	2.9	48.3			21.0
R radial	2.0	50.0			20.1

SNAP = Sensory nerve action potential; R = right.

Urine output increased to 2000 ml in the next six days. Audiometry on the third day suggested severe sensorineural hearing loss. On day 7 she complained of prickling, tingling, and numbness of the distal limbs, extreme over the feet. The sensory problems became even worse two weeks later.

The patient was fully conscious but had a severe hearing deficit. Her limbs were movable but with generalised hyporeflexia, bilateral plantar flexor responses, impaired sensation to 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 sensory problems one year after the ingestion. Six months later, muscular strength, reactive tendon reflexes, and sensation in all modalities were normal. She could hear the telephone ringing without aid. The sural nerve conduction studies showed low amplitudes of sensory action potential but normal latencies.

Bromate poisoning has occasionally been described in the medical literature with patients presenting with restlessness, depressed consciousness, and generalised convulsions. It is encountered mainly in children who ingest the agent accidentally and in adults who attempt suicide, and it can produce serious nephrotoxic and ototoxic sequelae.¹⁻³ Hearing problems are seemingly less severe in children, probably because of the potential for neural regeneration. Fatal events have mainly been ascribed to acute renal failure.

The estimated lethal dose of potassium bromate ranges from about 200 to 500 mg/kg of body weight, equivalent to 10 to 25 g per person of average body weight. Our patient ingested about 7.5 g of sodium bromate (130 mg/kg body weight), not lethal but toxic enough to cause renal failure and deafness.

She had tinnitus soon after ingestion, and deafness occurred five hours later. Renal failure developed on the second day, and oliguria responded poorly to diuretics. Renal function improved after management by haemodialysis but hearing was permanently damaged. Renal biopsy was not performed, but renal tubular necrosis in the proximal convoluted tubules, interstitial oedema, and inflammation have been

reported.¹ The pathology of the inner ear is not known.

Our patient had prickling and numbness of the limbs, seven days after ingestion. This sensory discomfort lasted for one year, longer than any previous report, and was accompanied by muscular stretch hyporeflexia. Nerve conduction studies confirmed the clinical findings of sensorimotor polyneuropathy.

Permanent cold wave setting solutions contain a thioglycolate hair care lotion and a bromate solution (either 2%–4% potassium bromate or 10%–20% sodium bromate).³ The first makes the hair flexible by changing the sulphur-sulphur (S-S) bonds of keratin to sulphur-hydrogen (S-H) bonds. The bromate then reoxidises the S-H bonds to form different S-S bonds and curl the hair. Like chlorate, a strong oxidising agent, bromate can interfere with S-H groups in energy generation pathways to result in methaemoglobinaemia, although this may not be manifest.¹ As suggested by Quick *et al*, the kidney and cochlea have similar antigenicity.⁴

Spontaneous recovery may occur as a possible result of reformation of S-S bonds. The primary changes may occur in the Schwann cells and the myelin sheaths, as reported in a case with sural nerve biopsy.⁵

Measurement of the serum concentration of bromate is currently not possible. Hence, we measured the bromide concentration of the patient instead of bromate, as neither should be present in normal subjects. High concentrations of bromide may indicate intoxication by bromide; the detectable concentration of bromide in our patient is attributed to conversion from bromate and can be an indicator of residual bromate.

Prognosis after bromate poisoning is usually poor, but our patient recovered from renal failure after haemodialysis. Her sensory complaints had also disappeared one year later, although she remained partially deaf.

VINCHI WANG

Department of Neurology, Cardinal Tien Hospital, Hsintien, Taipei, Taiwan

KON-PING LIN

CHING-PIAO TSAI

KO-PEI KAO

Department of Neurology, Neurological Institute, Veterans General Hospital, Taipei, Taiwan.

National Yang-Ming Medical College, Taipei, Taiwan, 11217, Republic of China

Correspondence to: Dr Kon-Ping Lin.

1 Dunsley I. Potassium bromate poisoning. *Am J Dis Child* 1947;74:730–4.

2 Matsumoto I, Morizono T, Paparella MM. Hearing loss following potassium bromate: two case reports. *Otolaryngol Head Neck Surg* 1980;88:625–9.

- 3 Lue JN, Johnson CE, Edwards DL. Bromate poisoning from ingestion of professional hair-care neutralizer. *Clinical Pharmacy* 1988;17:66–70.
- 4 Quick CA, Fish A, Brown C. The relationship between cochlea and kidney. *Laryngoscope* 1973;83:1469–82.
- 5 Deshimaru M, Miyagawa T, Sumiyoshi S, Nomura Y. Peripheral nerve disease associated with acute renal failure due to bromate poisoning. *No To Shinkei* 1976;28:807–14.

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.¹ The embolus may arise from a "tail" of thrombus that lies at the top of the ICA,² from thrombus from the "stump" of the ICA,³ or from thrombus from the ipsilateral external carotid artery passing through pial vessels of the ophthalmic artery.⁴ Another mechanism could be embolisation across the circle of Willis. To support this hypothesis, transcranial Doppler ultrasonography in four patients with internal carotid artery occlusion detected embolic signals in the middle cerebral artery ipsilateral to the internal carotid artery occlusion. After surgery of the opposite stenosed internal carotid artery no signal was detected.⁵

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 a right renal artery stenosis was discovered that required transluminal angioplasty. During the procedure, as the axillary catheter was removed through the aorta, the patient sustained a right hemiparesis with aphasia. On admission to the stroke unit of the Salpêtrière Hospital two hours later, the patient was awake, but on neurological examination there was no comprehension of any order or oral expression. He had a right hemiparesis with Babinski's sign, right hemianopsia, and hypoaesthesia. 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 evidence of fresh thrombotic material floating in the lumen. Periorbital directional Doppler detected no reverse flow in the ophthalmic artery. Transcranial Doppler ultrasonography showed full irrigation 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