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

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2 Pruss GA, Amaral DG, Squire LR. Hippocampal abnormalities in amnesic patients revealed by high resolution magnetic

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. 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 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. A 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, create, protein, venereal disease research laboratory test, and anticoagulopin 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. 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-oto-

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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 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, 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). She 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.
Urine output increased to 2000 ml in the first hour on hemodialysis on the third day after admission, suggesting severe sensorineural hearing loss. On day 7 she complained of pricking, tingling, and numbness of the distal limbs, extreme otodynia. The sensory problem could not be resolved by dialysis.

The patient was fully conscious and had a severe hearing deficit. Her limbs were moveable but with generalised hyporeflexia, bilateral plantar flexor responses, impaired sensation in pin prick 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 amplitudes 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 two weeks later.

Six months later, muscular strength, active 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.

\[ SNAP = \text{Sensory nerve action potential; } R = \text{right.} \]

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.1,2 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 pricking 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 dichloro-phenyl care hair lotion and a bromate solution (either 2%-4% potassium bromate or 10%-20% sodium bromate).2 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 oxidises 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 methaemoglobinemia, 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 hyporeflexia. Measurement of the serum concentration of bromate is currently not possible. Hence, we measured the bromide concentration of the patient instead of bromate, as noted should be present in renal samples. 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.

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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 the middle cerebral artery (MCA) after surgery of the opposite stenosed internal carotid artery no signal was detected.3 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 months later the right renal artery stenosis was discovered that required transluminal angioplasty. During the procedure, as the auxiliary catheter was removed through the aorta, the patient sustained retrograde embolisation of a clot from an atrial thrombus into the ICA. On admission to the stroke unit of the Salpêtrière Hospital two hours later, the patient was awake, but on neurological examination there were no signs of weakness of any order or oral expression. He had a right hemiparesis with Babinski's sign, right hemianopia, 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 vessels 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 narrow band of fresh thrombotic material floating in the lumen. Peribulbar 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 aneurysm. The source of embolic material could be directly the left external carotid