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 which regrettably are a case of a 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 all 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 anticoagulant drug screening. 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 obscurations. These symptoms progressed to daily bilateral obscurations of vision 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 normal papilloedema, with peripheral constriction of the fields but normal visual acuity. Neuro-ophthalmological examination confirmed clinical hearing loss in the left ear, with no evidence of retrocochlear pathology. 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 frequencies ranging from 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 result of the second 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.1-4 It has been suggested that the Weber test lateralised to the right.1 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 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 fibrous otosclerotic 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,5-7 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 loss 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.

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 severe 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 rising to 26 mg/dl and serum creatinine 2·3 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). In the second day, serum urine nitrogen reached 48 mg/dl and creatinine 9·3 mg/dl. Because of deteriorating renal function she was referred to the Venereal Disease Research Laboratory and received three courses of haemodialysis.
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