An unusual cause of benign paroxysmal positional vertigo

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Abstract

Benign paroxysmal positional vertigo (BPPV) is a self-limiting condition characterized by vertigo and nystagmus induced by certain head positions. The most common causes of BPPV are post-traumatic following head injury and post-viral labyrinthitis. We present an interesting case of BPPV following an otherwise uneventful neurosurgical removal of a parietal osteoma using hammer and chisel. Caution should be exercised during such procedures as disabling vertigo can result for a considerable period of time.

Key words: Vertigo, benign positional; Head injuries; Neurosurgery

Case report

A 40-year-old lady presented with a positionally-related vertigo dating from the neurosurgical removal of a left-sided parietal osteoma.

She had originally presented with a 25-year history of a nontender, bony, hard, fixed lump over the left parietal region. It had slowly grown to 7 cm in diameter. Her past medical history was insignificant and a complete neurological examination including cranial nerves and cerebellar function were entirely normal at this stage. Skull X-rays and CT scan suggested this swelling to be an osteoma (Figure 1). This was surgically removed using a hammer and chisel. The operation was otherwise uneventful but on the third day post-operatively she developed severe positionally-related dysequilibrium for which she was referred to the ENT department.

When she was seen by us she denied any previous history of ear disease or vertigo. There was no history of hearing loss or tinnitus. A general ENT examination and a pure tone audiogram were unremarkable but the Dix and Hallpike (1952) positional testing elicited a typical rotatory nystagmus on left ear down position with the fast component in the clockwise direction. It had a latency of a few seconds, lasted less than 30 seconds, and was associated with a subjective sensation of vertigo. It could not be elicited on repeated testing. A diagnosis of benign paroxysmal positional vertigo was made. The patient was followed-up at regular intervals and the vertigo improved gradually over the next year.

Discussion

Benign paroxysmal positional vertigo is characterized by

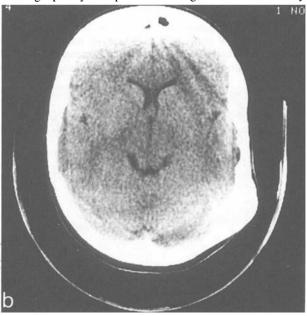


Fig. 1

Left parietal osteoma displayed by: (a) skull X-ray; (b) axial CT scan.

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bursts of nystagmus associated with vertigo provoked by a particular head position (Dix and Hallpike, 1952). The nystagmus is predominantly rotatory in the position of forward gaze. In the right ear down position the rotation is to the right (counterclockwise quick component). If the rotation occurs in the left ear down position the rotation is to the left (clockwise quick component). With the eyes directed toward the uppermost ear the nystagmus is sometimes vertical and upward beating (Schuknecht, 1969). The nystagmus has a latency of a few seconds and is associated with a sensation of violent vertigo. It lasts for no more than half a minute and fatigues with repeated testing (Barany, 1921; Dix and Hallpike, 1952).

In a recent study of 240 patients with BPPV, the most common identifiable causes were head trauma (17 per cent) and viral labyrinthitis (15 per cent). The former group had the onset of BPPV within three days of well documented head trauma (Baloh *et al.*, 1987).

The first description of BPPV was by Barany (1921), who attributed the condition to a disorder of the otolith organ, as it was induced by change in head position relative to gravity (Barany, 1921). This was supported by Dix and Hallpike (1952) who found unilateral degeneration of utricular macula at necropsy in a typical case of BPPV.

Schuknecht (1969) suggested a variety of aetiological factors. These included spontaneous degenerative changes in the vestibular labyrinth, labyrinthine concussion from head injury, otitis media, ear surgery and occlusion of the anterior vestibular artery. Barber (1964) noted positional vertigo occurring in 47 per cent of 47 patients with longitudinal fracture of the temporal bone and in 20.8 per cent of 77 head injuries of comparable severity without skull fracture.

Schuknecht (1969) also suggested that the possible mechanism for the development of positional vertigo following head injury is disruption of the utricular otolithic membrane and release of otoconia into the endolymph of the pars superior. These otoconia are free to respond to gravitational force and

settle into the ampulla of the posterior semicircular canal. Change in head position can then forcibly displace the ampulla. Schuknecht's cupulolithiasis could provide a reasonable explanation for most of the clinical features of positional vertigo of the benign paroxysmal type.

In the unusual case presented here it would appear that direct removal of a large osteoma with hammer and chisel caused sufficient trauma to result in symptoms more commonly associated with a severe head injury. Although fortunately a self-limiting condition in the majority of cases, BPPV may nonetheless be extremely disabling to the patient. When removing such large benign bony skull tumours it would appear prudent to minimize the possibility of such sequelae by using a less traumatic technique such as an electric or pneumatic drill of the type used by otologists.

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