

An unusual complication of cochlear implant: benign paroxysmal positional vertigo

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Abstract

Three days after the initial fitting of the cochlear device a 40-year-old woman complained of severe rotational vertigo following head movements associated with neurovegetative symptoms. Otoneurological evaluation revealed a horizontal paroxysmal positional nystagmus beating towards the lowermost ear, induced by rolling the patient's head from supine both to the right or to the left lateral position suggesting the diagnosis of benign paroxysmal positional vertigo of the left horizontal semicircular canal. The nystagmus characteristics were the same whether the cochlear device was switched on or off. The hypothesis of an otolith dislodging due to the electrical stimulation during the initial fitting is discussed.

Key words: Cochlear implant; Vertigo; Semicircular canals

Introduction

A weakness or absence of vestibular function can be found in patients with profound bilateral sensorineural hearing loss who are potential candidates for cochlear implantation (Black, 1977; Black *et al.*, 1987; van den Broek *et al.*, 1993; Magnusson *et al.*, 1995) although the presence of vestibular symptoms are quite rare because vestibular lesions are generally well compensated.

Following cochlear implant, the risk of loss of vestibular function may be estimated at between 31–60 per cent (Mangham, 1987; van den Broek *et al.*, 1993; Brey *et al.*, 1995; Huygen *et al.*, 1995) and the occurrence of vestibular symptoms is quite uncommon (Pyman *et al.*, 1990; van den Broek *et al.*, 1993).

Usually symptoms spontaneously recover within a few days (Hoffman and Cohen, 1995) and are usually represented by transient post-operative dizziness due to surgery (Clark, 1988; van den Broek *et al.*, 1993; Huygen *et al.*, 1995) or to electrical stimulation of the vestibular (saccular) nerve with device use (Black, 1980; Magnusson, 1995).

The presence of rotatory vertigo is very unusual and it has been reported as a perilymph gusher in 0.1 per cent of the patients (Hoffman and Cohen, 1995) or in Menière-like disease (Graham and Dickins, 1995).

This case reports a horizontal canal benign paroxysmal positional vertigo (HC-BPPV) as a post-operative complication of cochlear implant.

Case report

A 40-year-old female affected by a 10 years long lasting progressive profound sensorineural hearing loss of unknown origin, was implanted in the left ear with a Nucleus 22 channel device. Pre-operative vestibular assessment included electronystagmography (ENG), computerized dynamic posturography (Di Girolamo *et al.*, 1998) and harmonic acceleration test. No spontaneous or positional nystagmus was observed. ENG with bithermal

caloric irrigation, was performed with 250 ml of water for 40 seconds (30°C and 44°C), and showed a well-compensated bilateral vestibular hypoexcitability.

A global postural impairment specifically of the vestibular component was recorded by dynamic posturography.

The 22 channel intra-cochlear device was implanted on the left side in March 1997 using a vertical postaural incision, cortical mastoidectomy, posterior tympanotomy and cochleostomy. Dacron ties and bone cement were used to anchor the electrode array. The surgical procedure was uneventful. A good insertion was obtained with 22 functional plus five supporting rings inside the cochlea. Post-operative recovery and wound healing were satisfactory.

Vestibular tests and dynamic posturography showed no modification of the vestibular function in the implanted ear.

After six weeks she underwent her initial fitting of the cochlear device. Three days later the patient complained of severe rotational vertigo following head movements associated with neurovegetative symptoms.

Otoneurological evaluation revealed a horizontal paroxysmal positional nystagmus beating towards the lowermost ear, induced by rolling the patient's head from supine both to the right or to the left lateral position.

The nystagmus, more evident when the head was turned towards the implanted side, showed a latency of about four seconds, duration ranging from 30 to 40 seconds.

Clinical features suggested the diagnosis of benign paroxysmal positional vertigo of the left horizontal semicircular canal.

The nystagmus characteristics were the same with the implant switched on or off. After two rehabilitative manoeuvres according to Lempert and Tiel Wilck (1996) a complete resolution of the vertigo was obtained. No recurrence has been observed up to now.

Discussion

Clinical features of BPPV of the horizontal semicircular canal have been well described (Dix and Hallpike, 1952;

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McClure, 1985; Pagnini *et al.*, 1989). BPPV seems to be caused by utricular otoconial debris both adherent to the cupula or free to move within the endolymph of the involved semicircular canal. Based upon nystagmus characteristics it is possible to identify the side and the affected semicircular canal (Lanska and Remler, 1997; Fife, 1998).

The pathogenesis of BPPV may be idiopathic, post-traumatic, post-infectious or due to vascular disorders (Baloh *et al.*, 1994).

The specific clinical features of BPPV observed in our patient can hardly be explained by a stimulation of vestibular receptors due to the electrophysiological changes as suggested by Pyman *et al.* (1990), and Van den Broek *et al.* (1993). Direct electrical stimulation of the vestibular nerve as reported by Black *et al.* (1980), who described vestibular symptoms strictly correlated with frequency, duration and amplitude of the stimulus caused by the location of the electrode near to the saccular nerve, can be excluded, because the symptoms and positional nystagmus were independent of the cochlear device status (ie. whether switched on or off).

The onset of BPPV as a consequence of trauma during otoneurological surgery as suggested by Hughes and Proctor (1997) and Andaz *et al.* (1993) should be excluded by the long time interval between surgery and the onset of vertigo (one month).

An incorrect surgical introduction of the electrodes causing end organ damage, as demonstrated by Clark *et al.* (1988) in post mortem studies can be excluded by post-operative imaging and by the normal functioning of the device.

A case of acute rotational vertigo following cochlear implant with fluctuating electrical thresholds (Luetje *et al.*, 1993; Graham and Dickins, 1995) was reported as autoimmune-mediated endolymphatic hydrops.

Perilymphatic gusher as a rare but major post-operative complication is present in 0.1 per cent of Hoffman and Cohen's series (1995).

The correlation between the activation of the cochlear implant and the onset of vertigo suggests the hypothesis of an otolith dislodging due to the electrical stimulation that is necessary during the initial fitting. Although such an hypothesis cannot be supported by any electrophysiological data it seems very closely event-related.

Moreover, considering the high annual incidence of BPPV (100/100.000) a causal association cannot be excluded (Mizukoshi *et al.*, 1988; Froehling *et al.*, 1991).

The vestibular function must always be carefully investigated before cochlear implantation and cochlear implant candidates should be advised of the possibility of post-operative vestibular effects following cochlear implantation. Inner ear damage must be as far as possible avoided during electrode introduction within the cochlea. If vestibular symptoms occur after surgery BPPV should be considered.

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