CASE REPORT: TOTAL SUDDEN HEARING LOSS WITH BENIGN PAROXYSMAL POSITIONAL VERTIGO

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Introduction

Sudden sensorineural hearing loss (SSNHL) is defined as a hearing loss of at least 30 dB over three contiguous frequencies occurring in less than 3 days [1]. Vertigo and profound hearing loss are considered poor prognostic factors in SSNHL [2-4]. The most common diseases associated with vertigo in SSNHL include BPPV, vestibular neuropathy, central vertigo, and non-specific dizziness [3]. The following case report is about a patient with sudden onset vertigo followed by total sudden sensorineural hearing loss in one ear, with nausea and vomiting upon changing her head position to left when lying down in bed. Her symptoms and results of examination are discussed.

Case history

The patient was a 67-years-old Chinese female. She presented with sudden onset vertigo of 2 weeks with unsteady walking and described her sensation as “everything was whirling”. Vertigo made her feel nauseated, and she would feel a little better after vomiting and would be able to close her eyes and go into asleep. Vertigo episodes would last for about one hour without hearing loss or tinnitus. Vertigo stopped several days later without medical intervention, but started again 3 days before presentation with feeling “left ear blocked and unable to hear the mobile phone”. Her vertigo had persisted and would aggravate upon changing her head position to left when lying down.

This patient denied a history of ear infections, hearing loss or flu. She did not have noise exposure or head trauma history, or family history of hearing loss. Small kidney stones had been found and treated several years ago without recurrence. In general, this patient was in good health, with no report of high blood pressure or abnormal blood glucose.

Clinical data

This patient was first seen by an otolaryngologist at the Sixth Affiliated Hospital of Sun Yat-Sen University (SYSU) on May 21st, 2012, and referred to our audiological clinic facility for further evaluation.

The audiometric test (AC40 clinical audiometer, TDH39 earphone, Interacoustics, Inc) revealed total sensorineural hearing loss in left ear and normal hearing in right ear (Fig.1). The acoustic immittance measurement (AT235h impedance audiometer, Interacoustics, Inc) revealed type A tympanograms in both ears, but ipsilateral and contralateral acoustic reflexes in left ear were absent.

A Dix-Hallpike positional test provoked vertigo and nystagmus when the patient was moved from a sitting to supine position, with the head tipped 45 degrees toward the floor with left ear downward. The nystagmus had a latency of a few seconds and fatigued after approximately 30-40 seconds. It was torsional with the fast phase beating toward the left and adapted with repeated testing. Optic fixation reduced severity of the nystagmus.

Electrocochleargraphy (EcochG) was obtained using alternating clicks (Eclipse, Interacoustics, Inc). Ear canal skin was carefully prepared. Disposable electrodes were placed on the forehead (ground), vertex and mastoid. The recording electrode was gold foil attached to the insert phone tubing, and linked to the preamplifier via an alligator clip. Stimuli were presented at, 11.3/sec for 1000 sweeps. Summating potential (SP) and Action potential (AP) and baseline (BLst: Baseline start, BLe: Baseline end) were labeled (Fig.2). The SP/AP area ratio and amplitude ratio were 1.443 and 0.273 in right ear, and 4.948 and 1.103 in left ear, respectively. The AP of

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left ear showed a broad and low amplitude waveform.

MRI of the brain and brainstem reported no retrocochlear pathology. Distortion product otoacoustic emissions (DPOAEs) were measured, with two pure tone stimuli $f_1$ and $f_2$ presented at 65 dB SPL and 55 dB SPL respectively (frequency ratio $f_2/f_1 = 1.22$). The maximal amplitudes of $2f_1-f_2$ were obtained from 500 Hz to 8000 Hz. The test time of every frequency lasted for 90 sec. The pass criterion at each stimulus frequency was when the DPOAE signal to noise ratio was equal to or above 7 dB. The right ear passed while no $2f_1-f_2$ responses were evoked in the left ear. The patient was admitted and treatment administered after finishing these tests.

**Treatment**

The patient was placed on a course of methylprednisolone 500 mg i.v qd for 5 days followed by tapering over 5 days, and Batroxobin injection 5 BU i.v qd, combined with Betahistine 12 mg po tid and Mecobalamin 0.5 mg po tid for 10 days. During the first five days of treatment, she reported improvement of vertigo and she could lie in bed and turn over without worrying about being dizzy. We performed Dix-Hallpike maneuver on her again on the 5th day after treatment, she didn’t show any nystagmus in any position.

On May 31st, 2012, 10 days after her first treatment, the patient received another audiometric test (Fig.1), which showed an obvious improvement across 125 Hz to 8000 Hz in left ear. Patient also reported improved hearing in left ear with the “blocked feeling in her left ear” remitted, although there continued to be a constant “buzzing” tinnitus in left ear day and night. We prescribed mecobalamin 0.5 mg po tid, Betahistine 12 mg po tid and Sibelium 10 mg po qn for one week for her upon discharge.

**Discussion**

Sudden sensorineural hearing loss with BPPV is not rare. The diagnosis of BPPV has been reported in 12.7% of patients with SSNHL [3]. More than half of the idiopathic sudden sensorineural hearing loss patients develop BPPV relatively early, usually within 24 hours after the onset of deafness. In an analysis [5] of the characteristics of concomitant vertigo in patients with sudden deafness, 45.7% patients showed vertigo and hearing loss with time intervals between 1 hour and 24 hours, and 28.3% patients presented vertigo several days after cochlear symptom. The patient in this case experienced vertigo 2 weeks before sudden total hearing loss, and showed BPPV signs during the hearing loss attack. In summary, BPPV is not an uncommon symptom in sudden hearing loss and vertigo patients. In most cases BPPV arises from canalolithiasis, caused by otoconia that have been dislodged from the utricle and are moving freely in the semicircular canals. In our case, it was possible that the hydraulic distension or rupture damaged the otolithic apparatus, leading to the release of otoconia debris which migrated to the semicircular canal where it caused BPPV manifestations.

Important prognostic factors of sudden hearing loss include the presence of vertigo, severity of hearing loss, and the time between onset of hearing loss and treatment [2]. Most reports confirm that vertigo is a negative prognostic factor, and the prognosis in patients with BPPV and elevated audiometric thresholds is very poor [3-8]. Some researchers [3,4] report that many SSNHL patients with BPPV exhibit profound hearing loss, of whom very few achieve complete or significant improvement in response to treatment. Some authors [5] find that the mean duration of treatment for BPPV with SSNHL or unilateral vestibulopathy is longer than for other groups. Some researchers [7,8] find that high inner ear signals on 3D FLAIR on a 3T MRI system in sudden deafness patients are associated with poor chances to recover. In our case, the patient presented with vertigo and BPPV manifestations with total sudden hearing loss, consistent with similar cases in previous reports. Fortunately, the time between onset of hearing loss and treatment in this case was relatively short (3 days). Based on the latest definition of “recovery” [9], the treatment outcome assessment grade in this patient was
C2 (Partial), i.e. PTA (dB HL) within 50% of initial hearing loss or >10 dB improvement of the hearing loss. The low frequency thresholds showed the most prominent recovery. The patient had no more vertigo and aural fullness after treatment, with persistent but tolerable tinnitus. It was worth noting that this patient’s BPPV symptoms resolved after 5 days of pharmacological treatment without any therapy maneuvers. This may indicate that spontaneous recovery of BPPV represents improved prognosis.

Endolymphatic hydrops may change EcochG waveforms by increasing the magnitude of SP. Researchers report that the SP/AP curve area curve ratio is more sensitive than the conventional SP/AP amplitude ratio in revealing hydrops. The cut-off limits for SP/AP amplitude and area curve ratios are 0.46 and 1.94, respectively. Some authors conclude that less patients with nonrecurrent sudden sensorineural hearing loss present with enhanced SP/AP ratio than recurrent sudden sensorineural hearing loss, and that an enhanced SP/AP ratio appears to be a favorable prognostic factor in recurrent idiopathic sudden sensorineural hearing loss. Besides, EcochG gives a definite location of deafness in most patients in whom conventional audiometry has produced equivocal results. In the left ear of the patient in our study, the SP/AP amplitude and area ratios were both abnormal, indicating the cochlea as the location of deafness and a potentially favorable prognosis in response to treatment.

References


(Received September 17, 2013)