Scientific Research and Essays Vol. 7(1), pp. 8-11, 9 January, 2012
Available online at http://www.academicjournals.org/SRE
DOI: 10.5897/SRE11.177
ISSN 1992-2248 ©2012 Academic Journals

*Full Length Research Paper*

# The clinical characteristics of the benign paroxysmal positional vertigo associated with sudden sensorineural hearing loss

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Accepted 22 November, 2011

**To explore the clinical characteristics of the benign paroxysmal positional vertigo (BPPV) associated with sudden sensorineural hearing loss (SSHL) (through retrospective analysis of 19 cases from June 2008 to June 2010) and improve the diagnosis and treatment. Nineteen cases (7 male and 12 female, aged 46 to 68 years old) of BPPV associated with SSHL from June 2008 to June 2010 were retrospectively analyzed. All the patients were diagnosed SSHL by history and pure tone threshold test. Positional paroxysmal vertigo was diagnosed by Hallpike test or roll test. Epley maneuver or Barbecue roll maneuver were used to treat according to the type of BPPV, and the efficacy was evaluated by symptom and Hallpike test. 63.2% cases were female patients in the study; BPPV occurred after SSHL in all of the cases (all in the hearing loss ears), of which 15 cases were posterior semicircular canal lithiasis and 4 cases were horizontal semicircula canal lithiasis (only 1 case was cupula lithiasis); 15 out of 19 patients had BPPV within 2 weeks after SSHL; BPPV was cured in all cases after several times of posture treatments. BPPV can result from SSHL. A possible mechanism may be the inner ear circulation disorder or virus infection that lead to otolith falling off. Most cases of BPPV occurred in the posterior semicircular canal of the diseased ear. Otolith reposition is an effective treatment for BPPV secondary to SSHL.**

**Key words:** Benign paroxysmal positional vertigo, sudden sensorineural hearing loss, otolith.

## INTRODUCTION

Sudden sensorineural hearing loss and benign paroxysmal positional vertigo (BPPV) are common peripheral vertigo diseases. BPPV accounts for 20 to 30% of all vertigo diseases (Dix and Hallpike, 1952). BPPV can occur as primary or idiopathic disease, but can also be secondary disease to some inner ear diseases such as idiopathic sudden deafness, Meniere's disease, vestibular neuritis, posterior circulation ischemia or systematic postoperative complications. We found that there are certain clinical characteristics on BPPV secondary to SSHL. Herein we summarize our findings from the retrospective analysis of the 19 cases of BPPV associated with SSHL.

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## PATIENTS AND METHODS

This study was approved by Ethics Committee of Sun Yat-Sen University and informed consent was obtained from all patients. The case group comprised of 19 hospitalized patients in the authors' department from June 2008 to June 2009, including 7 male and 12 female aged from 46 to 68, with an average age at 54. The 19 cases of BPPV associated with SSHL contained 4 cases of horizontal semicircular canal BPPV and 15 cases of posterior semicircular canal diagnosed by postural test. Diagnosis and treatment of SSHL was based on the standard according to the guidelines issued by American Medical Association (2007) (Anne and Lorne, 2007). Diagnosis of BPPV was based on the results of Dix-Hallpike test and roll test, conforming to the standard from American Academy of Otolaryngology-Head and Neck Surgery Foundation (2008) (Bhattacharyya et al., 2008). Posture-induced test was performed routinely on each SSHL patient in our department and patients with positive-result receive repositioning treatments thereafter. Patients with posterior semicircular canal BPPV received otolith reposition using Epley maneuver (Epley,

**Table 1. 19** cases distributed in different auditory related characters.

| | | |
|---|---|---|
| Period between BPPV and SSHL | <14 day | 15 |
| | >14 day | 4 |
| Type of acoustic curve | Total deafness | 5 |
| | Flat type | 9 |
| | Hf type | 3 |
| | Lf type | 2 |
| With spontaneous vertigo | Y | 16 |
| | N | 3 |
| Otoconia side | Hearing loss side | 19 |
| | Opposite side | 0 |
| Semicircular canals | PSC | 15 |
| | HSC | 4 |
| Prognosis of BPPV | Natural cure | 4 |
| | Reposition treatment | 15 |

**Table 2.** The relation between the type of acoustic curve and otoconia side.

| Type of acoustic curve (n) | | Otoconia side | |
|---|---|---|---|
| | | PSC (n) | HSC (n) |
| Total deafness | 5 | 5 | 0 |
| Flat type | 9 | 8 | 1 |
| Hf type | 3 | 1 | 2 |
| Lf type | 2 | 1 | 1 |

PSC: posterior semicircular canal; HSC: horizontal semicircular canal; Hf: high frequency and Lf: low frequency.

1980). Briefly, seated patient with head placed 45° to the affected side lay supine with the head gesture. The head tilted 30° downward, turned to the neutral position and then slowly to 45° to the asymptomatic side. Then the head and body were rotated until facing downward 45°, after which the patient was brought to sitting position, head turned forward with chin down 30°. Each position was kept for 1 to 2 min or until vertigo subsided. Patients with lateral semicircular canal BPPV were treated by Barbecue roll maneuver (Lempert and Tiel-Wilck, 1996). Briefly, seated patient lay supine promptly.

The head tilted 90° to the asymptomatic side. Then the body was rotated 180° to the asymptomatic side with head tilted to the neutral position. The body was further rotated 180° to the initial supine position and the patient then sat up. Patients received at least 6 months follow-up after in-hospital treatments.

## RESULTS

In this group, there were 12 females and 7 males. In all of the cases, BPPV occurred after SSHL, of which 15 cases were posterior semicircular canal lithiasis (all in the hearing loss ears) and 4 cases were horizontal semicircular canal lithiasis (1 case was cupulalithiasis in the hearing ear). In this study, all patients were cured after 3 to 4 times of posture treatments (Tables 1 and 2).

## DISCUSSION

From the anatomical structures study, the vestibule and the cochlea have an intently relationship, for example, about 28 to 57% of patients who have sudden sensorineural hearing loss (SSHL), also have vestibular symptoms (Rauch, 2008). Some cases of BPPV are primary, there is no obvious precipitating factor, and some of BPPV are secondary which is concurrent with SSHL in some cases. Katsarkas (Katsarkas and Kirkham, 1978) reported that there was 5% SSHL patient who had concurrent BPPV. Park et al. (2001) analyzed 125 cases

of acute hearing loss patients, 6% of which were associated with BPPV in the posterior semicircular canal. Mikael et al. (2000) and Rambold et al. (2004) reported BPPV cases which were SSHL related in the posterior semicircular canal and lateral semicircular canal, respectively. Wu et al. (2005) also reported 35 cases of BPPV concurrent with SSHL. In this paper, after the study of 19 cases of BPPV associated with SSHL, the clinical features of the secondary BPPV are summarized and discussed as follows:

**Secondary BPPV occurred to those SSHL patients with spontaneous vertigo**

Most of the patients of secondary BPPV with SSHL had the spontaneous vertigo. In this study, 16 out of 19 patients underwent spontaneous vertigo attacks after hearing loss. The hearing curve for patients with spontaneous vertigo mostly showed a flat curve (9/19) or deaf hearing curve (5/19). For the aforementioned 16 cases, BPPV mainly occurred after spontaneous vertigo symptoms showed apparent remission. Peripheral vestibular vertigo includes spontaneous vertigo and positional vertigo and so on. There must be some certain factors that affected both cochlea and vestibule since SSHL associated with spontaneous and vestibule and cochlea are closely related in anatomy structure study. Xin et al. (2010) believed that the occurrence of spontaneous vertigo right after SSHL was caused by poor peripheral vestibular compensation after the inner ear lesions. As the brain and brainstem established fast physiological adaptation and compensation for asymmetric static signals from two vestibular peripheral organs, the spontaneous vertigo usually underwent remission within a short period of time. Different from spontaneous vertigo, positional vertigo may be due to the abnormal metabolism of the otolith structure and otolith fall-off if further developed, caused by the SSHL contributing factors.

But this pathologic change of otolith may take a longer period of time to develop, so positional vertigo often occurred after spontaneous vertigo.

**BPPV mainly occurred within 2 weeks after SSHL**

All the patient in this study underwent BPPV after their hearing loss, and 15 out of 19 patients had BPPV within 2 weeks after SSHL, the remaining 4 cases occurred in one or two months after SSHL. Although it has been reported in certain case that BPPV occurred more than half a year after SSHL, this kind of case was rarely seen. Xin et al. (2010) and the opposite situation which secondary SSHL occurred within a short period after BPPV's occurrence had not been observed either. We believe that otolith structure is different from ear hair cells, inner ear hair cells are more sensitive to a variety of possible factors leading to SSHL, so the pathogenesis is such that at the early stage, the damage of inner ear hair cells occurred and hearing declined, while at later stage, the impairment for otholith structure appeared which caused secondary BPPV. This is the reason of BPPV and SSHL do not synchronized in the event of time. For the phenomenon that BBPV mainly occurred within two weeks after SSHL, further observation and research are needed.

**BPPV occurred mainly in posterior semicircular canal of SSHL ear**

For the 19 patients in this study, the ears for BPPV were highly consistent with the ears for SSHL, 15 out of 19 cases were otolithiasis in the posterior semicircular canal in the hearing loss ears and 4 out of 19 cases were otolithiasis located in the horizontal semicircular canal in the hearing loss ears. The highly consistent of the location for the two diseases would relate to the factors which caused SSHL. BPPV in the posterior semicircular canal occurred in patients who showed flat hearing curve and those with deaf hearing curve (13 out of 15 cases). The current study suggested that SSHL with vertigo was related with the circular disorders in inner ears (Wu et al., 2010). According to the inner ear blood supply area, internal auditory artery has three main branches; 1) the cochlear artery to supply cochlear apex, 2) the vestibular artery to supply utricle, the anterior and the lateral semicircular canal, 3) the vestibulocochlear artery to supply for the posterior semicircular canal, saccule, and cochlear base. That is, the cochlea and the posterior semicircular canal are supplied by the same artery; poor blood supply to this area may cause ischemic changes for the physiological functions of the cochlea and the posterior semicircular canal. Since inner ear hair cells are more sensitive to ischemia, hearing dysfunction always happen earlier than vestibular dysfunction; as a result, the abnormal metabolism of otolith which leads to BPPV in semicircular canal. H Rambold also believed that BPPV in posterior semicircular canal may be associated with abnormal blood circulation in the inner ear (Rambold et al., 2004). In addition, the inner ear infections can also harm the inner ear by affecting the inner ear local microcirculation, sometimes the two interacts. During their pathology study in the temporal bone specimens for SSHL patients, Vasama et al., (2000) found degeneration of the spiral ligament, stria vascularis, saccule and hair cells and pathological changes of the inner ear infection such as hydrolabyrinth. Potentials in their study showed that the impact of inner ear infection was focused on saccule and horizontal semicircular canal (Myogenic et al., 2005).

In their anatomy for the temporal bone specimens from BPPV with Meniere's disease, Norimasa et al. (2009) found the existence of a large number of basophilic

deposits (fall off otolith) in the semicircular canal chamber and ridge cap on the involvement side of membranous labyrinth and high incidence of this phenomenon occurred in the side of semicircular canals where hydrolabyrinth occurred. Then whether the factors responsible for the inner ear circulation disorder and inner ear infections may also cause ischemia changes in otolith structure and hydrolabyrinth, further resulting in BPPV with SSHL, remains for further in-depth study.

### A few can self-healing, majority can be cured by reposition treatments

Our previous study found that BPPV associated with Meniere's disease was more stubborn to be treated, and often required multiple and repeated reposition treatments, some patients who showed good control of symptoms of Meniere's disease still need multiple reposition treatments or even surgery to recover (Peng et al., 2010), while the treatment for BPPV caused by SSHL differs from the former, clinical observations found that some BPPV showed the tendency of self-healing within a short period of time and the majority of BPPV associated with SSHL showed satisfactory results after 1 to 3 times reposition treatments, the only exception was for BPPV associated with total hearing loss, which need multiple reposition treatments, yet was still much easier to be cured compared with BPPV associated with Meniere's disease. We analyzed the possible reasons for this characteristics as: after the inner ear blood circulation disorders or ear infections and other possible mechanisms causing inner ear damage, the active treatments given to improve the microcirculation in patients and systemic corticosteroids treatments and other active treatments, combined with the reversibility of otolith structure which exists self healing tendency after virus infection, are the two major reasons for the good results in reposition treatments, which were applied after SSHL patients gained stable hearing. Gross et al. (2000) believed that due to the durable effect of stagnant water on both otolith structure and the membrane in membranous labyrinth, the otolith reposition process for BPPV associated with Meniere's disease was more complex.

In conclusion, we summarized the general clinical characteristics of BPPV secondary to SSHL through the retrospective analysis of 19 cases in our medical practice. With the accumulation of medical cases and the progress of related research, we will have deeper understanding of this disease.

### ACKNOWLEDEGMENT

This study was supported by Ministry of Education New Faculty Foundation (20090171120082) and Guangdong Province Medical Science Foundation (B2009075).

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