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Inner-Ear Function Test in Cases of Posterior Canal–Type Benign Paroxysmal Positional Vertigo

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Abstract: Otolaryngologists typically perform diagnoses and offer medical treatment for vestibular dysfunction. This vestibular dysfunction manifests as benign paroxysmal positional vertigo (BPPV), Ménière's disease, vestibular neuronitis, and so on. The etiology of BPPV is still not clear, so in this article we discuss inner-ear function, etiology, and factors related to BPPV. We examined by pure-tone audiometry and hot and cold caloric tests patients whom we identified as having diagnosed posterior canal–type BPPV. We observed canal paresis at a high rate on the affected side ($p < .01$). The term of recovery at the first treatment was longer in patients with canal paresis as compared to those without. Deterioration of hearing level was observed more frequently on the affected side ($p < .01$). The horizontal semicircular canal and cochlea are important potential sites of lesions affecting posterior canal–type BPPV, and the posterior circular canal and otolith are already considered to be sites of affecting lesions.

Key Words: audiometry; benign paroxysmal positional vertigo; canal paresis

Dizziness or vertigo is a comparatively common symptom found in various diseases. We examined patients who reported dizziness or vertigo (or both) on medical examination and received treatment. Otolaryngologists typically perform diagnoses and offer medical treatment for vestibular dysfunction. This vestibular dysfunction manifests as benign paroxysmal positional vertigo (BPPV), Ménière's disease, vestibular neuronitis, and so on.

BPPV is a disease characterized by an equilibrium dysfunction that was first reported by Bárány [1] in 1921 and then was described in detail and named *benign paroxysmal positional vertigo* by Dix and Hallpike [2] in 1952. BPPV is one of the common diseases of equilibrium dysfunction and is characterized by nystagmus on the positional nystagmus test. The diagnosis of BPPV by the positional nystagmus test is not difficult. However, this BPPV can take the form of an attack without an evident inducing factor.

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At present, the etiology of BPPV is still not clear, so in this article we discuss inner-ear function, etiology, and the factor related to the BPPV.

SUBJECTS AND METHODS

The subjects were 80 patients who visited Kitasto University from April 1994 until March 2006. They were given the diagnosis of posterior canal–type BPPV on the basis of standard diagnostic criteria provided by the Ministry of Health, Labour, and Welfare [3] and were examined by pure-tone audiometry and cold caloric tests. We excluded those suffering from sudden deafness or Ménière's disease and considered to have an inner-ear dysfunction. The average onset age was 52.9 ± 13.6 years (range, 24–79 years), and the gender distribution was 29 men and 51 women.

At the first visit, we performed a spontaneous nystagmus test and a gaze nystagmus test with an examination of eye movement behind Frenzel glasses, together with the usual ear, nose, and throat inspection and the common neurological examination. We conducted a caloric test after confirming the disappearance of positional nystagmus.

Equilibrium Function Tests

As stated, using the positional nystagmus test, we checked spontaneous nystagmus and gaze nystagmus. We also performed the positional nystagmus test behind Frenzel glasses. In addition, we performed a positional nystagmus test by the Dix and Hallpike method and performed a caloric test with 5 ml of 20°C water. Maximum slow-phase velocity within 10°/sec revealed canal paresis (CP).

Audiometry (Pure-Tone Audiometry)

The difference in the audiogram between the right and left ears was defined as positive when the difference was more than 30 dB on the average in six frequencies, except for 125 Hz. The term of recovery at first treatment was defined as the period before disappearance after confirming nystagmus for the first time. We analyzed the data by paired or unpaired t tests and χ^2 tests. We considered p values of less than .05 to be significant.

RESULTS

Affected Side

Of the total patient group, 35 cases were left-sided and 45 cases were right-sided.

Caloric Test

We observed CP in 32 of 160 ears: Of 80 ears affected with posterior canal–type BPPV and typical nystagmus, we found CP in 23 and, of 80 unaffected ears, we found CP in 9. Among these, three cases of CP were bilateral. We observed CP at a high rate on the affected side ($p < .01$). The term of recovery at first treatment was 58.3 ± 59.9 days in patients exhibiting CP (n = 29) and 33.3 ± 22.6 days in patients without CP (n = 51). The term of recovery at the first treatment was longer in patients with CP as compared to those without CP ($p < .01$).

Audiometry

We observed an audiogram difference between the right ear and the left ear in 23 of 80 patients. We observed deterioration of the hearing level in the affected ear in 17 patients and in the unaffected ear in 6 patients and observed deterioration of hearing level more frequently on the affected side ($p < .05$). The term of recovery at first treatment was 51.7 ± 60.2 days in cases exhibiting deterioration of hearing level (n = 24) and 55.5 ± 130.3 days in cases without deterioration of hearing level (n = 56). Between both groups, the difference in the days required for recovery at the time of first treatment was not significant.

Complications

Complications observed among the 80 patients were as follows: hypertension, 8; hyperlipidemia, 6; ischemic heart disease, 4; diabetes mellitus, 4; subarachnoid hemorrhage, 1; and arterial fibrillation, 1. Some patients exhibited multiple complications, so the total number of patients experiencing complications was 21 of 80.

The term of recovery at first treatment was 57.4 ± 33.7 days on average in patients with complications (n = 21) and 36.8 ± 42.5 days on average in patients with no complications (n = 59). The term of recovery at first treatment was longer in patients with complications as compared to that in patients without complications ($p < .01$).

DISCUSSION

The otolith and posterior semicircular canal are generally considered to be sites of lesions affecting BPPV. On the basis of results obtained, we found CP on the affected side at a high rate, and functional disorder of the horizontal semicircular canal was suggested to be an important factor affecting BPPV and the otolith and posterior semicircular canal (previously indicated). In other words, dysfunction of the inner ear itself is considered to be an important factor affecting BPPV.

The term of recovery at first treatment was longer in patients with CP than in those with no CP. The results suggest that a functional disorder of the semicircular canal exerts considerable influence on BPPV and on the days required for recovery. Indications suggest that CP may have prolonged the days required for recovery.

According to our results, differences in audiometry may indicate the inner-ear dysfunction. This result suggested to us the need to estimate the functional disorder of the cochlea.

We previously reported [4] atherosclerosis in the background of patients with vestibular dysfunction including BPPV. In this article, we report that the recovery terms at first treatment for patients with complications were longer in comparison to those for patients without complication. General estimates suggest that complications such as hypertension, diabetes mellitus, and hyperlipidemia promote atherosclerosis. This suggests that BPPV warrants close attention when it presents. Factors that delay recovery remain to be determined, but inner-ear dysfunction based on vascular problems and a disturbance of otolithic absorption should be considered.

Lindsay and Hemenway [5] advocated circulatory disorder in the etiology of BPPV owing to degeneration

caused by occlusion of the anterior vestibular artery. Dix and Hallpike [2] reported degeneration of the otolith and temporal bone on the basis of pathological examination. According to these reports, the dysfunction of the otolith is a major factor in BPPV.

With inner-ear dysfunction and microcirculatory damage generated by atherosclerosis, sensory epithelial dysfunction will occur, and detachment of the otolith from the otolithic membrane will progressively worsen. Consequently, a disorder of the otolithic absorption in vestibular dark cells may occur. Furthermore, a floating otolith may become a cause of onset of BPPV.

Prolongation of the recovery time required from first treatment may be caused by such conditions as inner-ear dysfunction and microcirculatory damage generated by atherosclerosis. We believe that a close inspection for this condition and management of factors that promote atherosclerosis as well as recognition of atherosclerosis as a potential factor in patients with vestibular dysfunction are important considerations if vestibular dysfunction recurrence is to be avoided.

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