STUDY OF VERTIGO RELATED TO HYPOTENSION : CLINICAL FEATURES AND PATHOLOGY

Fumihide Ito, Hideo Kamada, Masahiko Muroi

Department of Otolaryngology, Gunma University

Abstract : A diagnostic treatment was given to 18 patients with vertigo accompanied by hypotension. Neither audiometry nor equilibrium tests revealed diagnostic evidence for vertigo in these patients. In 17 patients (94.4%), vertigo disappeared within two weeks after the initiation of treatment for cerebrovascular circulatory disorders. An improvement or a tendency toward improvement was achieved in bilateral reduced hearing in the low-tone region which was detected on audiograms during the initial examination.

The clinical features were as follows :

- 1) Vertigo is rotatory or floating with short duration and occurs spontaneously or upon moving.
- 2) Although patients are not aware of impaired hearing, a reduction in hearing in the low-tone region is observed at a similar level on audiograms of both ears.
- 3) Tinnitus aurium or congested feeling in the ears, if present, does not appear or disappear accompanying vertigo.
- 4) Transient nystagmus findings, suggesting a central vestibular disorder, are occasionally observed in nystagmus tests.
- 5) Directional preponderance (DP) is occasionally obtained in caloric testing.

A circulatory disorder in the vertebrobasilar artery for which treatment for improving cerebrovascular circulation is effective, was considered as the pathogenesis of vertigo in patients with the above-mentioned clinical features.

Key words : Vertigo, Hypotension, Cerebrovascular circulatory disorder, Impaired hearing
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INTRODUCTION

Patients with vertigo not accompanied by cochlear symptoms rarely visit an otolaryngologist first. Such patients are usually referred to an otolaryngologist by a neurologist or a neurosurgeon due to suspicion of an internal ear disease and because various examinations, including magnetic resonance imaging (MRI), reveal no findings suggesting a cause of vertigo. In these patients, even hearing tests or equilibrium tests do not demonstrate any findings for diagnosing a disease responsible for vertigo. Since patients with vertigo often have hypotension, otolaryngologists treat them based on the assumption of a circulatory disorder, in the vertebrobasilar artery, as a cause of vertigo.

In general, other brainstem symptoms frequently serve as a basis for diagnosis in patients with vertigo

due to circulatory disorders of the vertebrobasilar artery. It is thus difficult to diagnose patients who complain only of vertigo. Clarification of clinical features of such patients is important in diagnosis as well as treatment. We gave a diagnostic treatment to patients with vertigo that seemed to be caused by a circulatory disorder in the vertebrobasilar artery related to hypotension. In this paper, we report their clinical features with a discussion about the pathology of vertigo.

SUBJECTS AND METHODS

Eighteen patients with vertigo and hypotension in whom audiometry, equilibrium tests (including spontaneous nystagmus tests, positional nystagmus tests, position change nystagmus tests, and caloric tests) and occasionally even MRI revealed no findings that all-

owed a diagnosis of vertigo-causing disease were selected as subjects from patients referred to the Department of Otolaryngology, Gunma University with a suspected internal ear disease from January 1996 to December 1996. Hypotension was defined as a systolic pressure of 110 mmHg or lower¹⁾. Of 18 patients, 13 (72.2%) were female. All patients were under 50 years with an average age of 33.8 years. The systolic pressure was under 100 mmHg in 8 patients (44.4%) and was 100 mmHg or higher in 10 patients (55.6%) (Table.1).

The diagnostic treatment consisted of combined administration of dilazep dihydrochloride (Comelian) and adenosine triphosphate disodium (Adetphos), both of which had an effect of increasing cerebral blood flow. This treatment began on the initial examination day for all patients. Amezinium metilsulfate (Risumic), a vasopressor, was added to the treatment, if necessary.

The clinical progress after initiation of the treatment was observed by changes in the number of patients with such symptoms as vertigo, tinnitus aurium and congested feeling in the ear based on results of questionnaires. The exact probability test and the Student t-test were used to analyze improvement in vertigo and hearing.

Table.1 Subjects of the Study

Item		Number of patients (%)
Sex	Male	5 (27.8)
	Female	13 (72.2)
Age (years)	<19	2 (11.1)
	20-29	4 (22.2)
	30-39	4 (22.2)
	40-49	8 (44.4)
Systolic blood pressure (mmHg)	<100	8 (44.4)
	100-110	10 (55.6)

RESULTS

Table.2 summarizes clinical symptoms and test findings for all patients. Vertigo was rotatory or floating, and transient and recurrent. The duration of vertigo varied from several seconds to several minutes. A cochlear symptom, which was tinnitus aurium or a congested feeling in the ear, was observed in 9 of 18 patients (50.0%). This symptom did not appear or disappear accompanying vertigo. In nystagmus tests, positional nystagmus was observed in 2 patients (11.1%) and vertical positional nystagmus was observed in 2 patients (11.1%), which were all transient. Although caloric tests revealed no canal paresis (CP) in any of the 18 patients, directional preponderance (DP) was found in 6 (33.3%). Patients were not aware of impaired hearing, but a reduction in hearing, but a reduction in hearing in the low-tone region at a similar level was detected on audiograms for both ears in all patients. The difference in hearing between both ears was within 10 dB.

Table.3 shows changes in the number of patients with vertigo or cochlear symptoms from initiation of the treatment to 4 weeks later. Vertigo disappeared within 2 weeks after initiation of the treatment in 17 of 18 patients (94.4%). There was no significant difference in the improvement rate for vertigo between patients with a different nature or duration of the symptom ($p>0.50$, respectively). Tinnitus aurium and congested feeling in the ear largely improved within 4 weeks after initiation of the treatment. Since hearing was similar in both ears even after the treatment (the difference was within 10 dB), hearing in the left ear was selected for evaluation. Figure 1 illustrates changes in average hearing levels between the initial examination and 2 weeks after initiation of the treatment (the difference was within 10 dB). The average hearing levels for 125, 250, 500, 1,000, 2,000, 4,000 and 8,000 Hz

Table.2 Clinical Symptoms and Test Findings

Item			Number of patients (%)
Vertigo	Nature	Rotatory	11 (61.1)
		Floating	7 (38.9)
	Duration	Less than 1 minute	10 (55.6)
		1 minutes or longer	8 (44.4)
	Duration of present disease	Less than 1 month	6 (33.3)
		1 month-2 months	5 (27.8)
		greater than 2 months	7 (38.9)
Cochlear symptoms	Tinnitus aurium		6 (33.3)
	Congested feeling in the ear		3 (16.7)
Nystagmus	Positional nystagmus caused by directional changes		2 (11.1)
	Vertical positional nystagmus		2 (11.1)
Caloric test	Normal		12 (66.7)
	DP		6 (33.3)

Table.3 Changes in the Number of Patients with Clinical Symptoms

			Day of initial examination	1 weeks after initiation of treatment	2 weeks after initiation of treatment	4 weeks after initiation of treatment
Vertigo	Nature	Rotatory Floating	11 7	3 1	0 1	0 1
	Duration	Less than 1 minute 1 minute or longer	10 8	2 2	0 1	0 1
Cochlear symptoms	Tinnitus aurium		6	6	4	0
	Congested feeling in the ear		3	2	1	1

Fig.1 Hearing level of left ear (18 patients)

at the initial examination were 28.3(SD=9.2), 25.8(SD=9.1), 18.1(SD=7.5), 11.1(SD=6.3), 10.8(SD=5.8), 9.2(SD=6.1) and 12.5 dB (SD=8.4), respectively. Two weeks after initiation of the treatment, the levels were 13.3(SD=5.7), 11.9(SD=5.7), 9.4 (SD=5.4), 7.8(SD=6.2), 8.9(SD=6.5), 6.9(SD=6.0) and 9.4 dB (SD=7.3), respectively. The t-test revealed that improvement in hearing 2 weeks after initiation of the treatment was significant at 125,250 and 500 Hz ($p < 0.001$, respectively). Although an improvement or tendency toward improvement in hearing was observed in the low-tone region for both ears in all the patients, none of the patients were aware of any improvement in hearing.

DISCUSSION

Although attempts have been made to apply vestibular function tests, such as fixation nystagmus and positional nystagmus tests using electronystagmography as well as caloric tests, to diagnose circulatory disorders in the vertebrobasilar artery²⁾, none of the tests provide a definite basis for diagnosis. In general, other brainstem symptoms frequently serve as a basis

for diagnosis in patients with vertigo due to circulatory disorders of the vertebrobasilar artery³⁾. It is thus difficult to diagnose patients, like ours, who complain only of vertigo. Vertigo is a common symptom in patients with circulatory insufficiency of the vertebrobasilar artery. The incidence of vertigo was 58% reported by Williams et al.⁴⁾, 74% by Bradshaw et al.⁵⁾, and 63% by Marshall⁶⁾. Fisher⁷⁾ reported that 88 of 112 patients with vertebrobasilar artery occlusion exhibited vertigo and that approximately one forth of the patients had vertigo alone as an initial symptom, with no other brainstem symptoms. Vertigo is one of the most common symptoms in circulatory disorders in the vertebrobasilar artery and may be the sole symptom for it. We believe that there are a rather large number of patients with circulatory disorders of a severity that exhibits vertigo alone. Kubo³⁾ reported that vertigo was often rotatory or floating, lasted for several seconds to several minutes, and occurred spontaneously or following positional changes when vertebrobasilar insufficiency was caused by reversible functional changes. These findings agreed well with the nature and duration of vertigo found in our patients. Nystag-

mus findings suggesting a central vestibular disorder in 4 of 18 patients (22.2%) during an initial examination seems to be due to transient ischemia of the vestibular nuclei. DP obtained in 6 patients (33.3%) is also considered to be attributable to central vestibular disorders. Patients were not aware of impaired hearing, but a reduction in hearing in the low-tone region in both ears was observed on audiograms for all patients. Sasano et al.⁸⁾ examined 7 patients with sensorineural deafness of one ear, considered to be caused by circulatory insufficiency of the vertebrobasilar artery and found deafness in the other ear that seemed to be intact. They reported that impaired hearing was attributable to a circulatory disorder in the internal ear, denying the possibility of endolymphatic edema, based on the finding that changes in hearing were larger in the low-tone region during the observation period and also based on electrocochleography results. In our study, since hearing was almost the same for both ears, the affected side could not be identified. A reduction in hearing in the low-tone region in both ears also seems to be due to an internal ear blood flow disorder caused by circulatory disorders of the vertebrobasilar artery. It is highly probable that such reductions in hearing in the low-tone region may be overlooked because they are mild, do not differ from ear to ear and are not accompanied by any awareness of impaired hearing.

Kawata et al.⁹⁾ reported that the effect of adenosine triphosphate disodium in improving cerebrovascular circulation was enhanced by a concomitant use of dilazep dihydrochloride. These two drugs were also administered to the patients in our study. Ameszium metilsulfate (Risumic), a vasopressor, was added to the treatment if necessary, because hypotension is one of the important factors in circulatory disorders. We¹⁰⁾ previously reported that the same combined therapy markedly improved symptoms in 4 of 10 patients (40.0%) with Ménière's disease and in 9 of 12 patients (75%) with circulatory insufficiency of the vertebrobasilar artery or other cerebrovascular circulatory disorders. The improvement rate in this study was significantly higher than that for Ménière's disease ($p=0.007$), but there was no significant difference in the rate between this study and the previous study ($p=0.325$). Such a difference in the improvement rate seems to be due to a difference in the pathology of the subjects. Our subjects were patients with vertigo which was considered to be caused by circulatory disorders of the vertebrobasilar artery. They were referred to our department since symptoms were not alleviated in spite of treatment for vertigo, so the presence of an internal ear disease was suspected. In most cases, vertigo was

first alleviated after improving cerebral circulation through treatment. In patients whom transient vertigo frequently occurred, a reduction in hearing in the low-tone region in both ears was found on audiograms. It should be noted that these findings improved or tended to improve following alleviation of the symptoms.

In cerebral blood vessels, unlike peripheral blood vessels, there is a cerebral circulation autoregulation mechanism that maintains blood flow at a constant level against changes in blood pressure¹¹⁾. This regulation mechanism is considered to be closely related to the autonomic nervous system¹²⁾. In addition, the cerebrovascular autoregulation functions of the internal carotid artery and the vertebrobasilar artery differ. The latter is inferior to a function that maintains blood flow against changes in blood pressure, so that the vertebrobasilar artery is more readily affected by a reduction in blood pressure, leading to a reduction in blood flow in the cerebellum and the brain stem. Matsunaga¹³⁾ stated that the vestibular nucleus was most sensitively affected by abnormal hemodynamics of the vertebrobasilar artery. Even if blood flow is maintained at a constant level, cerebral circulatory disorders may occur. This seems to be closely associated with hypotension or cerebral arterial sclerosis. These angiopathies are important factors for depressed cerebral circulation autoregulation.

The relationship between blood pressure and cerebral circulation disorders depends on age and other individual factors. The subjects of this study were patients with systolic pressure of 110 mmHg or lower. It is likely that these patients had a latent circulatory disorder in the vertebrobasilar artery due to hypotension, which was not severe enough to exhibit symptoms on its own, and that adding a decrease in cerebral perfusion pressure due to some cause to the circulatory disorder, induced transient ischemia in the vestibular nuclei. It is considered that this ischemia caused transient vertigo and that a blood flow disorder in the internal ear caused a reduction in hearing in the low-tone region.

Except patients with vertigo due to obvious cerebral infarction or cerebral hemorrhage, most patients with vertigo due to cerebral circulation disorders exhibit transient symptoms with little cause, resulting in a difficult diagnosis. To diagnose a patient with vertigo alone as a circulatory disorder in the vertebrobasilar artery, other vertigo-causing diseases should be ruled out. Nystagmus findings suggesting a central vestibular disorder are rarely obtained as a basis for diagnosis in patients with extremely short vertigo. A reduction in hearing in the low-tone region in both

ears on audiograms at a similar level was considered to be a finding suggesting circulatory disorders in the vertebrobasilar artery.

CONCLUSION

A diagnostic treatment was given to 18 patients with vertigo accompanied by hypotension. Neither audiometry nor equilibrium tests revealed diagnostic evidence for vertigo in these patients. The clinical features were as follows:

- 1) Vertigo is rotatory or floating with a short duration, and occurs spontaneously or upon moving.
- 2) Although patients are not aware of impaired hearing, a reduction in hearing in the low-tone region is observed at a similar level on a audiograms of both ears.
- 3) Tinnitus aurium or congested feeling in the ear, if present, does not appear or disappear accompanying vertigo.
- 4) Transient nystagmus findings, suggesting a central vestibular disorder, are occasionally observed in nystagmus tests.
- 5) DP is occasionally obtained in caloric testing.

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