Cochleovestibular Dysfunction Caused by Cerebrovascular Diseases

Ágnes Szirmai

Semmelweis University, Department of Otolaryngology and Head and Neck Surgery, Budapest, Hungary

Abstract: When a vascular disorder of the cochleovestibular system is considered, the diagnosis is based on exclusion of other diseases. As arteries of the cochleovestibular system cannot be directly visualized, physicians must deduce vascular cochleovestibular disease from the vascular risk factors and vascular lesions of other territories. With my colleagues at Semmelweis University, I analyzed neurootological and audiological examination results in 65 patients with vascular vertigo. Cochleovestibular system disorders can be considered to be of vascular origin if the examinations exclude other diseases, if the patients have vascular risk factors, and if other territories of the brain accessible for imaging methods show vascular disorders.

Key Words: auditory brainstem response; cochleovestibular dysfunction; vascular vertigo; vertebrobasilar insufficiency

The entire cochleovestibular system's blood supply comes from the vertebrobasilar system. Although the main vessels of the brainstem and the circle of Willis can be seen in angiograms, arteries of the cochleovestibular system cannot be observed with either transcranial Doppler sonography or magnetic resonance angiography (MRA). When cochleovestibular dysfunction of vascular origin is considered, diagnosis is based on exclusion of other diseases.

Vertigo often is caused by vertebrobasilar insufficiency. Baloh [1] distinguishes several symptoms of vascular lesion. In labyrinthine infarction and in lateral pontomedullary infarction, a patient suffered from sensorineural hearing loss and, in all of these syndromes, suffered from vertigo. Several authors have written about sensorineural hearing loss in cerebrovascular disorders [2–5]. In their reports, occlusion of small-caliber arteries due to severe arteriosclerosis or slow blood flow could be diagnosed.

Vertebrobasilar ischemia sometimes produces a combination of central and peripheral vestibular symptoms (e.g., antero-inferior cerebellar artery and postero-inferior cerebellar artery infarctions). These arteries supply overlapping territories of the brainstem and cerebellum and the peripheral labyrinth via the labyrinthine artery [6].

The aim of the investigation reported here was to diagnose cochleovestibular dysfunction in patients with vascular vertigo.

PATIENTS AND METHODS

In the neurootological department of Semmelweis University, we analyzed the data from 65 patients with vertebrobasilar insufficiency. The mean age of the patient group was 54 years (range, 45–64 years). Patients with Ménière's disease, benign paroxysmal positional vertigo, multiple sclerosis, or intracranial space-occupying lesions were excluded from the study. The patients had occipital headache, minutes-long rotatory vertigo after head movement, drop attacks, occasional transient diplopia, and sensorineural hearing loss. Some patients had vertiginous attacks that lasted a few days and were accompanied by vomitus, severe occipital headache, and perioral facial numbness. Most of the patients demonstrated several risk factors (Table 1); one-half had multiple risk factors that predisposed them to cerebrovascular diseases.

Reprint requests: Ágnes Szirmai, MD, PhD, Semmelweis University, Department of Otolaryngology and Head and Neck Surgery, H-1083, Szigony u 36, Budapest, Hungary. Fax: 36-1-333-3316; E-mail: szirmai@fulo.sote.hu

Table 1. Risk Factors

Risk Factors	Patients (%)
Multiple risk factors	53
Hypertension	40
Diabetes mellitus	15
Hypercholesterolemia	40
Arteriosclerosis	33
Thrombophilia	10
Obesity	47
Estrogen therapy	9
Cervical spondylosis	84
Psychiatric disorder	19

We performed a complete neurological examination, Doppler sonography, magnetic resonance imaging (MRI), and MRA, all of which verified the vascular origin and excluded space-occupying lesions and multiple sclerosis. All patients had normal eardrum and middle-ear function. We examined the cochleovestibular function of all patients by separate audiological and vestibular function tests. Cochlear function tests included pure-tone audiometry, acoustic reflex threshold and decay, and brainstem evoked response audiometry. Vestibular tests included statokinetic tests, examination of spontaneous nystagmus, smooth-pursuit eye movement tests, and the bithermal caloric test. The nystagmus examination was carried out by computer-based electronystagmography. The caloric nystagmus (RW: right-ear irrigation with warm stimulus; LW: left-ear irrigation, warm stimulus; RC: right-ear irrigation, cool stimulus; LC: left-ear irrigation, cool stimulus) patterns were analyzed based on the average slow-phase velocity using the Jongkees formula: caloric weakness = [(RW + RC) − (LW + LC)] × 100/(RW + RC + LW + LC); directional preponderance = [(RW + LC) − (LW + RC)] × 100/(RW + RC + LW + LC).

RESULTS

A breakdown of patient symptoms is shown in Table 2. Two-thirds of the patients (45/65) had sensorineural hearing loss, and most experienced rotatory vertigo. Nearly one-half of the patients suffered from tinnitus (35/65) and headache (34/65).

Table 2. Symptoms of Patients

Symptoms	Patients (%)
Unsteadiness	26
Rotatory vertigo	65
Normal hearing	32
Unilateral mild hearing loss	21
Unilateral severe hearing loss	26
Bilateral hearing loss	21
Tinnitus	53
Headache	47

Table 3. Brainstem Evoked Response Audiometry Results

Results	Patients (%)
No I wave	21
Increased V; latency	21
Increased I–V; IPL	25
Increased I–III; IPL	8
Irregular waves	8
Normal	17

IPL = interpeak latency.

Brainstem evoked response audiometry results were pathological except in approximately 16.8% of cases, or 11 of 65 (Table 3). No patient had a normal vestibular system, 37% (24/65) had a peripheral vestibular lesion, and 52% (34/65) had a central vestibular lesion; 11% of patients (7/65) suffered from combined vestibular lesion. In 32% of patients (21/65), we found no spontaneous nystagmus but saw nystagmus in a narrow sense in 26% (17/65), and we detected gaze nystagmus in 42% of the patients (27/65).

Using the Romberg test, we found deviation to each direction in 26.3% (17/65) and ataxia in 57.9% (38/65), but the statokinetic test results were normal in 15.8% (10/65). In 44 patients (68.4%), we did not find positional nystagmus. We could provoke the peripheral type of positional nystagmus (e.g., benign paroxysmal positional vertigo) in only 10.5% of patients, whereas the central type was found in almost one-fourth of the patients (14/65).

An analysis of the caloric test results based on the Jongkees formula revealed pure caloric weakness in 47.4% of the patients (31/65), but we saw pure directional preponderance in only 15.8% (10/65) and a combined lesion in 36.8% (24/65). The degree of caloric weakness and directional preponderance is seen in Table 4.

Table 4. Calorigram Parameters: Severity of Caloric Weakness and Directional Preponderance

Parameter	Patients (%)
Severity of caloric weakness (%)	
Up to 25 (normal)	35
26–40	18
41–70	29
70–100	18
Severity of directional preponderance (%)	
Up to 25 (normal)	40
26–40	14
41–70	28
70–100	18

Table 5. Evaluation Results

Examination	Patients with Pathological Findings (%)	Patients with Normal Results (%)
Neurootology	100	0
BERA	83	17
Doppler sonography	60	40
Risk factors	58	42
MRI and MRA	63	37
Audiology	72	28
SPECT	95	5

BERA = brainstem evoked response audiometry; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging; SPECT = single-photon emission computed tomography.

Caloric weakness or canal paresis occurs in peripheral lesions, whereas directional preponderance is characteristic of central vestibular lesion. In central lesions, the increased average slow-phase velocity of nystagmus is characteristic, owing to destroyed central inhibiting mechanisms. In a few cases, typical positional nystagmus formed the basis of the diagnosis of central vestibular lesion, although the nystagmographic results were normal.

We examined the ratio of pathological findings of MRA, MRI, Doppler sonography of the internal carotid and vertebral arteries, brainstem evoked response audiometry, and otoneurology (Table 5). Neurootological examination results were pathological in every case. Auditory brainstem response showed cochleovestibular disorders in 83% (54/65), and carotid and vertebral artery Doppler results were pathological in 60% (39/65). MRI and MRA showed several pathological findings in 41 cases. Although 20 patients had normal hearing, the detailed audiological examination results, including acousticofacial reflex patterns, were normal in only 18 patients. Single-photon emission computed tomography of the brain was pathological in most of the patients with vertebrobasilar insufficiency.

DISCUSSION

Vertebrobasilar ischemia can often be responsible for a wide range of central, peripheral, and combined vestibular syndromes. As arteries of the cochleovestibular system cannot be visualized directly, we have to arrive at the diagnosis of vascular cochleovestibular disease on the basis of vascular risk factors and vascular lesions of other territories. In cerebrovascular patients, neurootological examination is the most sensitive procedure by which to evaluate vascular lesion of the brainstem.

Auditory brainstem response and vestibular examination showed several lesions of brainstem function and labyrinthine function. In most of the patients we evaluated, we could observe multiple risk factors for cerebrovascular disorders.

Cochleovestibular system dysfunction can be considered to be vascular in origin if the examinations exclude other diseases, if the patients demonstrate vascular risk factors, and if other territories of the brain accessible for imaging methods show vascular disorders.

REFERENCES

1. Baloh RW. Vertebrobasilar insufficiency and stroke. *Otolaryngol Head Neck Surg* 112:114–117, 1995.
2. Doyle KJ, Fowler C, Starr A. Audiologic findings in unilateral deafness resulting from contralateral pontine infarct. *Otolaryngol Head Neck Surg* 114:482–486, 1996.
3. Huang MH, Huang CC, Ryu SJ, Chu NS. Sudden bilateral hearing impairment in vertebrobasilar occlusive disease. *Stroke* 24:132–137, 1993.
4. Yamasoba T, Kikuchi S, O'Uchi T, et al. Sudden sensorineural hearing loss associated with slow blood flow of the vertebrobasilar system. *Ann Otol Rhinol Laryngol* 102: 873–877, 1993.
5. Yamasoba T, Kikuchi S, O'Uchi T, et al. Magnetic resonance angiographic patients with slow vertebrobasilar blood flow. *Acta Otolaryngol Suppl (Stockh)* 520:153–156, 1995.
6. Brandt T. Vascular Vertigo. In *Vertigo (Its Multisensory Syndromes)*. London: Springer-Verlag, 2000:303–305.