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Clinical short communication

Spontaneous, headshaking, and positional nystagmus in post-lateral medullary infarction dizziness

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ABSTRACT

Background and purpose: Lateral medullary infarction (LMI) sometimes causes long-lasting dizziness. However, the characteristics of nystagmus in patients with post-LMI dizziness are unknown. We undertook a prospective, comparative study of nystagmus in patients with and without post-LMI dizziness to determine the characteristic pattern of nystagmus of chronic post-LMI dizziness.
Methods: We evaluated and compared nystagmus under spontaneous, head-shaking, and positional testing conditions in 12 patients with post-LMI dizziness and in 6 patients without post-LMI dizziness.
Results: In the dizziness group, contralateral spontaneous nystagmus, ipsilateral head-shaking nystagmus, and horizontal direction-changing geotropic positional nystagmus were observed in patients in whom the LMI had occurred <60 days previously (subacute period). In patients with dizziness in whom the LMI had occurred >90 days previously (chronic period), the nystagmus was ipsilateral under all conditions. In the non-dizziness group, ipsilateral nystagmus was observed in 1 of the 2 subacute patients only after head-shaking and in 1 of the 4 chronic patients only during positional testing.
Conclusions: Ipsilateral nystagmus observed under all spontaneous, head-shaking, and positional testing conditions characterizes chronic post-LMI dizziness.

1. Introduction

Lateral medullary infarction (LMI), or Wallenberg syndrome, is a well-known vascular syndrome, usually caused by occlusion of the posterior inferior cerebellar artery or vertebral artery [1]. Vertigo and/or dizziness, usually associated with other neurologic symptoms such as gait instability, dysphagia, hoarseness, impaired facial and contralateral limb/body sensation, is common in the acute phase of LMI [2]. Dizziness can persist in the chronic phase of LMI [3]. However, the characteristics and mechanism of chronic post-LMI dizziness are not well described.

In LMI, various patterns of nystagmus have been reported. Spontaneous contralateral beating nystagmus (beating away from the lesion side), presumably originating from ipsilateral central vestibular disruption [4], are frequently observed. Ipsilateral beating nystagmus (beating toward the lesion side) is also seen after head shaking [5]. Although head-shaking nystagmus is caused by various mechanisms, ipsilateral beating nystagmus following head-shaking in LMI is thought to result from ipsilateral damage of cerebellar inhibition on the velocity-storage mechanism of the vestibulo-ocular reflex (VOR) [5,6].

To determine the characteristic pattern of nystagmus in patients with chronic post-LMI dizziness, we conducted a prospective, comparative study to evaluate spontaneous, head-shaking, and positional nystagmus in patients with and without post-LMI dizziness.

2. Methods

Because our institution, Yokohama Brain and Spine Center, is an acute stroke care and rehabilitation center, not only acute but also chronic stroke patients are hospitalized. Among these patients, between September 2014 and August 2015, we recruited 18 consecutive patients with pure LMI for participation in the study. All 18 were enrolled regardless of the time from LMI onset, but patients with a history of another neurologic or psychologic disease were excluded. Patients with a definite diagnosis of a peripheral vestibular disorder, such as benign paroxysmal positional vertigo, vestibular neuritis, or Ménière's disease,

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Table 1
Study patients, including their clinical characteristics and study variables.

Patient/Age/Sex	Time from LMI onset (days)	Dizziness (DHI score)	Lesion side	Spontaneous nystagmus (velocity, °/s; amplitude, °)[a]	Head-shaking nystagmus (velocity, °/s; amplitude, °)[a]	Positional nystagmus (velocity, °/s; amplitude, °)[a]	Sensory topography
1/43/M	4	+ (NE)	L	Contralateral (4.53; 4.10)	Ipsilateral (9.18; 8.97)	Geotropic (10.67; 13.64)	Contralateral face Contralateral limbs
2/53/M	4	+ (NE)	R	Contralateral (1.45; 2.99)	Ipsilateral (8.17; 4.31)	Ipsilateral (7.21; 6.16)	Ipsilateral face Contralateral upper limb
3/41/M	7	+ (NE)	R	Contralateral (3.44; 3.41)	Ipsilateral (4.87; 4.54)	Geotropic (12.49; 4.34)	Ipsilateral face Contralateral limbs
4/49/M	12	+ (NE)	L	Contralateral (2.66; 6.52)	Ipsilateral (2.58; 3.24)	Geotropic (6.04; 5.84)	Contralateral upper limb
5/58/M	25	+ (26)	R	Contralateral (3.35; 6.30)	Ipsilateral (9.01; 8.36)	Geotropic (2.99; 6.01)	Ipsilateral face
6/50/M	57	+ (28)	L	Contralateral (3.59; 5.62)	Ipsilateral (6.50; 6.58)	Geotropic (2.51; 5.16)	Contralateral limbs
7/80/M	90	+ (48)	R	Ipsilateral (1.81; 2.62)	Ipsilateral (8.58; 3.72)	Ipsilateral (4.89; 3.00)	Ipsilateral face
8/67/M	102	+ (50)	R	Ipsilateral (5.02; 4.32)	Ipsilateral (9.68; 5.00)	Ipsilateral (3.89; 5.72)	Ipsilateral face Contralateral limbs
9/54/M	461	+ (48)	R	Ipsilateral (1.68; 4.91)	Ipsilateral (2.90; 5.88)	Ipsilateral (2.93; 5.44)	Ipsilateral face Contralateral limbs
10/45/M	711	+ (20)	R	Ipsilateral (2.54; 3.31)	Ipsilateral (5.58; 5.37)	Ipsilateral (4.75; 5.81)	Ipsilateral face Contralateral limbs/body
11/61/M	1245	+ (28)	L	Ipsilateral (3.34; 8.80)	Ipsilateral (5.84; 8.68)	Ipsilateral (10.95; 10.39)	Contralateral limbs
12/48/F	1607	+ (20)	L	Ipsilateral (3.84; 2.49)	Ipsilateral (6.82; 5.67)	Ipsilateral (2.15; 2.76)	Ipsilateral face Contralateral limbs/body
13/66/M	34	− (NE)	L	Contralateral (6.84; 6.41)	None	Geotropic (4.04; 3.05)	Ipsilateral face
14/49/F	35	− (0)	L	Contralateral (1.50; 2.19)	Ipsilateral (2.65; 2.08)	Contralateral (3.07; 3.29)	Ipsilateral face Contralateral limbs
15/46/M	368	− (0)	L	None	None	None	Ipsilateral face
16/63/M	1008	− (0)	L	Contralateral (1.79; 2.38)	None	Ipsilateral (1.55; 2.30)	Ipsilateral face Contralateral limbs
17/55/M	1327	− (0)	R	None	Contralateral (3.84; 8.67)	None	Ipsilateral face Contralateral limbs/body
18/59/M	1927	− (0)	L	Contralateral (2.42; 3.94)	Contralateral (6.24; 6.40)	Contralateral (4.86; 5.68)	Contralateral limbs/body

DHI, Dizziness Handicap Inventory; + dizziness present; − dizziness absent; NE, not examined; VOR, vestibulo-ocular reflex; C-VEMP, cervical vestibular evoked myogenic potential; O-VEMP, ocular vestibular evoked myogenic potential.
Fr = {(VOR gain in darkness) − (VOR gain during fixation)}/(VOR gain in darkness) × 100(%).
[a] Nystagmus are shown with the mean slow-phase velocities (degree/s, °/s) and amplitudes (degree, °).
[b] Mean ± SD in age-matched normal controls (n = 16) are shown in parentheses as normative values.

were also excluded. Ischemic lesions in the lateral medulla were confirmed by diffusion-weighted MRI (DWI) performed within 1 day after LMI onset. The 18 patients were divided into 2 groups according to the presence or absence of dizziness: 12 with dizziness (dizziness group) and 6 patients without dizziness (non-dizziness group). For the purpose of the study, LMI was classified as subacute or chronic, depending on the time that had passed since LMI onset, with subacute LMI referring to the stroke phase 0–60 days after LMI onset and chronic stroke referring to the stroke phase beyond 60 days. The severity of patients' dizziness was scored according to the Dizziness Handicap Inventory (DHI), which consists of 25 questions representing the impact on daily life in 3 domains: the physical, functional, and emotional domains [7]. The DHI was applied only during the chronic phase of LMI because it is intended only for evaluation of chronic dizziness. Although dizziness in the subacute phase patients was not scored, all patients with dizziness in the subacute phase could not walk without assistance because of unsteady feeling.

Spontaneous nystagmus, head-shaking nystagmus, and positional nystagmus were evaluated in both the dizziness group and the non-dizziness group. Nystagmus was recorded in darkness by means of video-oculography performed with the use of Frenzel goggles and a built-in charge-coupled device camera with infrared illumination. Head-shaking nystagmus was induced by application of a passive head-shaking maneuver; the patient's head was manually shaken horizontally in a sinusoidal fashion at a rate of about 2.8 Hz with an amplitude of about ±10° for 15 s [5]. In addition to nystagmus, the horizontal VOR, cervical vestibular evoked myogenic potential (C-VEMP), and ocular (O)-VEMP were evaluated.

Horizontal VORs were recorded in darkness by means of a video-oculography-based VOR recording and analysis system (IRN-2, Morita Mfg. Corp., Kyoto, Japan) [8], with the patient seated on a rotating armchair with a headrest (S-2, Nagashima Medical Instruments Corp., Tokyo, Japan). The chair was programed to rotate in sinusoidal fashion for 30 s (frequency, 0.6 Hz; amplitude, 60°). VOR gain, i.e., the eye velocity to head velocity ratio, was determined. After recording of the VOR, fixation suppression of the VOR was tested by asking the patient to fixate on a target (a red circle, 1 cm in diameter) located 50 cm from the eyes and rotating with the armchair. The fixation suppression rate (Fr) was calculated on the basis of the VOR gain in darkness (G) and the VOR gain during fixation (Gf) according to the following equation: Fr = (G − Gf)/G × 100(%) [9]. C-VEMP was recorded from the tonically contracting ipsilateral sternocleidomastoid muscle during monoaural stimulation with 105-dB, 500-Hz short tone bursts (MEB 2312 testing system, Nihon Kohden, Tokyo, Japan; bandwidth 20–2000 Hz, 200 averaged signals). Latencies of the first positive wave (p13) and second negative wave (n23) and peak-to-peak p13–n23 amplitude were measured [10]. O-VEMP was recorded from the contralateral lower eyelid (inferior oblique muscles) during stimulation with 105-dB, 500-Hz short tone bursts (MEB 2312, Nihon Kohden; bandwidth 20–2000 Hz, 200 averaged signals). The patient was instructed to