Clinical Study of Seven Patients with Infarction in Territories of the Anterior Inferior Cerebellar Artery

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Background: The prominent features of anterior inferior cerebellar artery (AICA) infarction are vertigo, cerebellar ataxia, and impaired hearing. The present study investigated neurological characteristics associated with AICA infarction. *Materials and Methods:* The locations of infarcts in 7 patients (age, 32-72 years) with AICA infarction were divided into the lower lateral pons, the middle cerebellar peduncle (MCP), and the cerebellum. *Results:* Ischemic lesions were located in the MCP in 6 patients, spread to the lower lateral pons in 3, and involved the cerebellum in 4 patients. Standing posture and gait were impaired in all patients. Five and 4 patients had impaired hearing and vertigo, respectively. Two patients had only symptoms of labyrinthine disease, and 1 had these symptoms accompanied by impaired hearing. The symptoms in 2 patients with the lesion in the lateral pons were consistent with those in Gasperini syndrome. Two of 3 patients without vertigo had ataxia of the extremities. Stenosis of the vertebral artery or basilar artery in 5 patients indicated that the etiology was branch atheromatous disease. *Conclusions:* The most prominent symptom of truncal and gait ataxia and the frequent association between vertigo and impaired hearing were consistent with the characteristics of AICA infarction. Two patients without vertigo had ataxia of the trunk and extremities that might have been due to involvement of the dorsal spinocerebellar tract in the inferior cerebellar peduncle. **Key Words:** Anterior inferior cerebellar artery—impaired hearing—vertigo—truncal ataxia—dorsal spinal cerebellar tract.

The anterior inferior cerebellar artery (AICA) reaches the lateral region of the lower lateral pons, the middle cerebellar peduncle (MCP), and the anteroinferior area of the cerebellum,[1-5] and supplies the inner ear through the labyrinthine artery which is a branch of the AICA.[6-8]

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Received September 25, 2016; revision received November 19, 2016; accepted November 25, 2016.

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1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2016.11.118

The prominent neurological findings of AICA infarction are vertigo and cerebellar ataxia that are often accompanied by hearing loss and/or tinnitus.[3,9] The MCP was the most impaired territory after AICA infarction.[4,10] The nuclei of the abducens, facial, and vestibulocochlear nerve are located in the lateral area of the lower pons.[4] Infarction of the AICA including ischemia in the lateral area of the lower pons results in peripheral facial nerve palsy, hearing anomalies on the ipsilateral side, a superficial sensory disturbance in the ipsilateral face and contralateral body/extremities, and cerebellar ataxia.[3,4] Although AICA infarction can be considered in patients with these symptoms of the cranial nerves in the lower lateral pons as well as cerebellar signs,[9,11,12] the clinical features vary according to the extent of an AICA infarct. Recent reports have associated vertigo and truncal ataxia (postural instability) with AICA infarction in the absence of other neurological findings and are referred to as

isolated vertigo-type AICA.[9-14] The clinical features of this type mimic those in peripheral vestibular diseases, namely, pseudo-labyrinthine disease.[11,14] These 2 conditions are difficult to differentiate, based on neurological findings.[9-14] Here, we studied the characteristics of AICA infarction, based on data from 7 patients.

Patients and Methods

Characteristics of the Enrolled Patients

We analyzed the neurological and magnetic resonance imaging (MRI) findings of 7 patients (age, 32-72 years; male, n = 6) with AICA infarction who were admitted to Nihon University School of Medicine, Itabashi Hospital, between 2002 and 2010. The elapsed time between the onset of the initial symptoms and MRI assessment ranged from 15 hours to 5 days. Brain infarction was diagnosed, based on areas of high intensity in diffusion-weighted images. The extent of infarcts was identified from findings of fluid-attenuated recovery inversion and T_2-weighted images.

Locations of Infarcts and Vascular Findings

Infarcts identified from diffusion-weighted, fluid-attenuated inversion recovery, and T_2-weighted images generated by a 1.5-Tesla MRI instrument were grouped according to location in the lower lateral pons, MCP, and cerebellum. Vascular findings of the posterior circulation were evaluated using magnetic resonance angiography (MRA) in 5 patients and by digital subtraction angiography in 1. The etiology of infarction comprised cardiogenic embolism associated with emboligenic heart disease, and atherothrombotic large vessel disease with angiographic evidence of stenotic and occluded arteries of the posterior circulation in the absence of emboligenic heart disease. Large vessel disease was taken as branch atheromatous disease (BAD) when stenosis was located in the proximal region of the basilar artery (BA) or the distal region of the vertebral artery (VA), and as artery-to-artery embolism when stenosis was located in the proximal region of the VA.

Clinical Findings of 7 Patients

The medical charts of the patients were reviewed to identify initial symptoms and neurological findings upon admission. Vascular risk factors were also identified. A board-certified specialist in neurology performed neurological examinations. Nystagmus was assessed using the confrontation test.

Results

Distribution of the Infarcts

Tables 1 and 2 show the distribution of infarcts. The MCP and the cerebellum were the sites of infarction in 2 patients (patients 1, 5) and the infarct in 1 was bilateral (patient 1). Infarcts were located in the lower lateral pons and the MCP in 2 patients (patients 2, 6) and in lower lateral pons, MCP, and cerebellum in 1 patient (patient 3). Infarcts located in the bilateral MCP of 1 patient had not spread to the lower lateral pons or the cerebellum (patient 4) and 1 patient had an infarct only of the cerebellum (patient 7).

Neurological Findings

Table 1 shows the neurological findings. Among 4 patients with vertigo (patients 3-5, 7), 1 had dizziness (patient 4) and 3 had rotatory vertigo (patients 3, 5, 7). All 7 patients had impaired standing ability and gait. Three patients had extreme truncal ataxia (lateropulsion or inability to stand) (patients 1, 5, 7). Ataxia of the extremities was unilateral (patients 3, 4, 6) and bilateral in 1 (patient 1). One patient with bilateral ischemic lesions had unilateral ataxia of the extremities (patient 4). Among 6 patients with nystagmus (patients 1, 3-7), the type was horizontal toward the unilateral direction in 3 patients (patients 1, 4, 5) and horizontal gaze-evoked toward the bilateral direction in 3 others (patients 3, 6, 7). One patient had Bruns nystagmus (patient 3). Eye movement was impaired in 2 patients (patients 1, 6) and 1 had lateral gaze palsy (patient 1). The other patient showed abducens nerve palsy on the side ipsilateral to the ischemic lesion and contralateral esotropia (patient 6). Two patients had peripheral facial nerve palsy on the side ipsilateral to the ischemic lesion (patients 2, 6). Impaired hearing in 5 patients (patients 2-4, 6, 7) comprised hearing loss in 4 (patients 2-4, 6) and tinnitus without hearing loss in 1 (patient 7). Two patients had the Horner sign (patients 1, 6). One patient had mild hemiparesis on the side contralateral to the ischemic lesion (patient 2). Two patients had a superficial sensory disturbance in the side of the face ipsilateral to the lesion (patients 2, 6). A sensory disturbance below the neck was distributed on the side contralateral to the lesion, indicating an alternating pattern.

Initial Symptoms of the 7 Patients

Table 3 shows the initial symptoms of the patients. Standing and gait were the most frequently impaired in 5 patients (patients 1, 3-5, 7), 4 of whom also had vertigo (rotatory, n = 3; dizziness, n = 1). Two patients each had hearing loss and tinnitus (patients 3, 7). A sensory disturbance was identified in 3 patients (patients 2, 3, 6), among whom it disappeared upon admission in 1 patient (patient 3) (Table 1).

Vascular Findings and Vascular Risk Factors

Table 1 shows the vascular findings and risk factors. Stenosis was located in the proximal region of the BA