Introduction

In the cerebellar infarction caused by AICA (anterior inferior cerebellar artery) or medial branch of PICA occlusion, the vertigo of labyrinthine type can be manifested as only symptom, while in pontocerebellar infarction caused by AICA occlusion, brain-stem signs predominate. Since the blood supply to the inner ear and the vestibulocochlear nerve arises from AICA, a combination of symptoms and signs caused by vestibulocochlear nerve lesions and brainstem and cerebellar lesions may be characteristics of the AICA infarction syndrome. Vertigo preceding infarction and mimicking labyrinthine lesions may have resulted from transient ischemia to the inner ear or the vestibular nerve. We now describe a case with AICA infarction presenting as vertigo mimicking labyrinthine lesions, with the involvement of eighth root entry zone on brain magnetic resonance imaging (MRI).
Case Report

A 56-year-old female was admitted to the emergency room with vertigo and hearing loss in right ear. She complained repetitive paroxysmal vertigo attacks, which was aggravated by positional change, since several days ago. On past medical history, she had diabetes mellitus and hypertension. Several years ago, she experienced sensorineuronal hearing loss on her left ear. There were no remarkable findings in her family history. Vital signs were normal and physical examination revealed no specific abnormalities. On neurological examination, her mental status was alert. Left beating jerky torsional and horizontal nystagmus was detected. She had a tendency falling and past pointing to right side. Motor and sensory examination were normal and deep tendon reflex was intact. There was no pathologic reflex. Eye tracking test and optokinetic pattern test(OKP) showed no abnormal findings. Left-beating direction fixed spontaneous horizontal nystagmus was observed in all of gaze, positional and positioning nystagmus tests. Caloric test showed no response in the right ear. Audiometry documented absent auditory function on both sides. Brain magnetic resonance images showed high signal intensity in the anterolateral region of the right inferior pons on T2-weighted images(Fig. 1). Serological test for syphilis was negative. Leukocyte count, the level of uric acid, triglycerid, and the rate of platelet adhesion slightly increased. The levels of her antiphospholipid antibody(IgG: 7.23 GPL unit/mL, IgM: 23.41 MPL unit/mL)
unit/mL) and anticardiolipin antibody (IgM: 14.7 MPL unit/mL) increased. One day after admission, right side severe peripheral facial palsy developed (Fig. 2) with dysmetria on her right hand. Her paroxysmal positional vertigo with nystagmus disappeared. On blink reflex with right supraorbital nerve stimulation, ipsilateral R1, and R2 responses were not detected, but contralateral R2(30.4 ms) response was normal. With left supraorbital nerve stimulation, ipsilateral R1(10.8 ms) and R2(30.4 ms) responses were normal, but contralateral R2 response was not detected (Fig. 3). On facial nerve conduction velocity during right facial nerve stimulation, prolonged terminal latency(4.4 ms) and decreased CMAP amplitude (560 uv) were noted on right. And during left facial nerve stimulation, normal terminal latency and CMAP amplitude were noted (Fig. 3). TCD showed increased flow velocity(76~80 cm/sec) and pulsatility index in the basilar artery system (Fig. 4). Conventional angiography revealed atherosclerotic change with luminal narrowing on proximal segment of basilar artery and narrowing of proximal segment of right AICA and loop of right AICA at the site of right internal auditory canal (Fig. 4). She was treated with intravenous heparin and her symptoms improved slowly.

**Discussion**

The syndrome of the AICA territory infarction includes vertigo, vomiting dysarthria, ipsilateral hearing loss with tinnitus, massive peripheral facial palsy, trigeminal sensory loss, Horner’s syndrome, ipsilateral dysmetria of the limbs, and contralateral loss of temperature and pain sensation sparing the face). These symptom and signs are mainly linked to the involvement of brainstem and middle cerebellar peduncle structure. When an infarction is limited to the cerebellar hemisphere, vertigo may be the only clinical manifestation. Rubenstein et al. reported cases of cerebellar infarct in AICA territory presented with an isolated labyrinthine type syndrome. This can be compared with cases of infarction in PICA territory also producing an isolated labyrinthine type syndrome. The cerebellar syndrome caused by the infarc-

![Fig. 3. Facial NCV(A) and Blink reflex(B: right, C: left).](image)

In her blink reflex, during right supraorbital nerve stimulation, ipsilateral R1, and R2 responses were not detected, and contralateral R2 response was normal. During left supraorbital nerve stimulation, ipsilateral R1 and R2 responses are normal, and contralateral R2 response was not detected. During right facial nerve stimulation, prolonged terminal latency and decreased CMAP amplitude were noted, and during left facial nerve stimulation, normal terminal latency and CMAP amplitude were noted.
tion in the AICA territory could be linked to lesions of the middle cerebellar peduncle, the restiform body, the ventral spinocerebellar tract or the cerebellar hemispheres\(^7\). The cerebellar signs are never isolated. The vestibular syndrome of infarction in AICA territory can be of the peripheral type because AICA gives rise to the internal auditory artery. The labyrinthine syndrome that follows infarction in the AICA territory could be explained by occlusion of the internal auditory artery or involvement of the vestibular nuclei in the pontine tegmentum, the eighth nerve in the lateral pontine area, or the flocculus and its connections\(^7\). Small branches of AICA supply facial and vestibulocochlear nerves in the CPA and the internal auditory canal(ICA) via recurrent penetrating artery\(^8\). These recurrent penetrating arteries(RPA) supply ventral part of the anterolateral pons\(^9\). The root entry zone of the facial and vestibulocochlear nerves had a rich network formed between medullary artery, AICA, and the inferior lateral pontine artery\(^9\). Thus, the anterolateral region of pons, middle cerebellar peduncle and the inner ear are the areas most commonly affected in the AICA occlusion. Part of the intrapontine vestibular nerve segment run close to the intrapontine segment of the facial nerve\(^9\). An ischemic insult to this region, for example, hypoperfusion in the territory of a long penetrating basilar artery branch, may simultaneously involve the intrapontine segments of the facial and vestibular nerves, which explains ipsilateral facial palsy and vestibular paresis. Because of collateral vessels, the root entry zone of the seventh and eighth nerves is often spared. But in our case, RPA occlusion brought about the root entry zone of the seventh and eighth nerves in the AICA territory. There was also concomitant lower basilar artery stenotic lesion. PICA dominance was not specified and it is possible that concomitant occlusive disease of the vertebrobasilar system may have altered collateral arterial supply.

We experienced isolated episodes of vertigo for several days before accompanied neurological deficits localized to inferior anterolateral pons. Neuro-otologic testing provided additional clues to the localization of the lesion in our patient. The patient had a loss of caloric response on lesion side(right side) at the time of infarction indicating a peripheral vestibular lesion. Audiometry showed abnormal function on lesion side (right side). Twenty-four hours later a peripheral facial palsy developed. Facial nerve conduction velocity and blink reflex revealed right facial neuropathy. Facial palsy was severe and permanent. MRI identified a

\[\text{Fig. 4. TCD(A) and conventional angiography(A, B). On conventional angiography, atherosclerotic change with luminal narrowing on proximal segment of basilar artery(white arrow) and narrowing of proximal segment of right AICA appeared and Loop of right AICA at the site of right internal auditory canal was seen, but its branch was not visualized(black arrow). TCD showed increased flow velocity(76–80 cm/sec) and pulsality index(6) in the basilar artery system.}\]
localized area of infarction in the inferior anterolateral pons although it could not exclude the inner ear lesions. We report a case with vestibular and facial nerve root entry zone infarction in AICA territory presenting as vertigo, which mimic labyrinthine lesion.

REFERENCES