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Neuro-Otological Aspects of Cerebellar Stroke Syndrome

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Cerebellar stroke is a common cause of a vascular vestibular syndrome. Although vertigo ascribed to cerebellar stroke is usually associated with other neurological symptoms or signs, it may mimic acute peripheral vestibulopathy (APV), so called pseudo-APV. The most common pseudo-APV is a cerebellar infarction in the territory of the medial branch of the posterior inferior cerebellar artery (PICA). Recent studies have shown that a normal head impulse result can differentiate acute medial PICA infarction from APV. Therefore, physicians who evaluate stroke patients should be trained to perform and interpret the results of the head impulse test. Cerebellar infarction in the territory of the anterior inferior cerebellar artery (AICA) can produce a unique stroke syndrome in that it is typically accompanied by unilateral hearing loss, which could easily go unnoticed by patients. The low incidence of vertigo associated with infarction involving the superior cerebellar artery distribution may be a useful way of distinguishing it clinically from PICA or AICA cerebellar infarction in patients with acute vertigo and limb ataxia. For the purpose of prompt diagnosis and adequate treatment, it is imperative to recognize the characteristic patterns of the clinical presentation of each cerebellar stroke syndrome. This paper provides a concise review of the key features of cerebellar stroke syndromes from the neuro-otology viewpoint. **J Clin Neurol 2009;5:65-73**

Key Words cerebellar stroke, vertigo, hearing loss, pseudo-APV, head impulse test.

Introduction

The cerebellum is about one tenth the size of the cerebrum and is located in the posterior fossa, beneath the tentorium cerebelli. Its effect on brain function is often relatively minor compared to cerebrum. The name "cerebellum" literally means "little brain." From a clinical point of view, the main function of the cerebellum is the coordination of movements. Cerebellar infarction is a common cause of a vascular vestibular syndrome and vertigo/dizziness is the most common manifestation of cerebellar stroke. It is usually accompanied by other cerebellar symptoms or signs. Since dizziness/vertigo may occur in isolation without accompanying neurological symptoms or signs, however, a mistaken diagnosis of acute peripheral vestibular disorders might be made. Whereas acute labyrinthine disorders are usually benign and self-limited, vascular injuries of the cerebellum may develop a mass effect. Large cerebellar infarction can cause brainstem compression, and acute hydrocephalus a few days after the onset of symptoms. Furthermore, although small cerebellar infarction generally has a benign prognosis, isolated cerebellar infarction usually results from emboli originating from the heart or great vessels, and recurrent emboli should be treated because a further brainstem infarction due to recurrent emboli can be life-threatening. For the purpose of prompt diagnosis and adequate treatment, it is imperative to recognize the characteristic patterns of the clinical presentation of each cerebellar stroke syndrome. Therefore, this paper provides a concise review of the key features of each cerebellar stroke syndrome focusing on the neuro-otological aspects.

Isolated Episodic Vertigo of Vascular Cause

Transient ischemia within the vertebrobasilar circulation (i.e., vertebrobasilar insufficiency) is a common cause of episodic vertigo in elderly patients. The vertigo is usually accompanied by other neurological symptoms or signs. It is typically abrupt in onset, and usually lasting several minutes.[1] Earlier reports[2,3] emphasized that isolated vertigo, when present for more than several weeks, is rarely due to vascular events. However, recent studies[1,4,5] reported contradictory findings. Grad and Baloh[1] reported that of patients with vertigo due to vertebrobasilar insufficiency, 62% had at least one isolated episode of

vertigo, and in 19% vertigo was the initial symptom. Moreover, 26% had canal paresis to caloric stimulation, indicating a permanent damage to the peripheral vestibular system involving the inner ear or vestibular nerve. Subsequent studies[5,6] also reported similar results: of 29 patients with vertebrobasilar insufficiency, 21% had episodic vertigo for at least 4 weeks as the only presenting symptom.[5] I recently reported three patients with anterior inferior cerebellar artery (AICA) infarction who experienced isolated episode of recurrent vertigo, fluctuating hearing loss, and/or tinnitus (similar to Meniere's disease) as initial symptoms 1-10 days prior to the infarction.[6] All of these data suggested that isolated episodic vertigo with or without auditory symptom can be the only manifestation of transient ischemia within the vertebrobasilar circulation. Isolated vertigo especially can occur when there is a stenosis of the caudal or middle portion of the basilar artery (presumably close to the AICA origin) or widespread slow vertebrobasilar flow on MRA.[4,6] However, it is still unclear whether isolated episodic vertigo originate from the brain or the inner ear. When isolated vertigo occurs in transient ischemia of the peripheral vestibular labyrinth,[1] the superior part of the vestibular labyrinth may selectively be vulnerable to ischemia, possibly due to the small caliber of the anterior vestibular artery (AVA) and little collateralization.[7] Patients with AVA infarction may subsequently develop typical episodes of benign paroxysmal positional vertigo; these have been ascribed to ischemic necrosis of the utricular macule and release of otoconia into the posterior canal. Since the posterior canal is supplied by the posterior vestibular artery, a branch of the common cochlear artery, it may be spared in AVA infarction.[7,8] Although isolated episodic vertigo can occur as a manifestation of vertebrobasilar insufficiency, long-lasting (>6 months) recurrent episodes of vertigo without other symptoms are almost never caused by vertebrobasilar disease.

Three Cerebellar Ischemic Stroke Syndromes

There are three major cerebellar arteries: the posterior inferior cerebellar artery (PICA), the AICA, and the superior cerebellar artery (SCA). After supplying branches to the brainstem, each of these arteries supplies the part of the cerebellum indicated by its name.

Acute vestibular syndrome due to anterior inferior cerebellar artery territory cerebellar infarction

Clinical anatomy

The AICA is an important artery for vascular supply to the peripheral and central vestibular structures. AICA is the most variable of the long circumferential cerebellar arteries and has the smallest zone of supply within the cerebellum. AICA commonly originates from the lower half of the basilar artery and usually supplies the inner ear, lateral pons, middle cerebellar peduncle, and anterior inferior cerebellum including the flocculus.[9] Since AICA always supplies the lateral pontine tegmentum and middle cerebellar peduncle, AICA territory infarcts usually involve the brainstem and are virtually never limited to the cerebellum itself whereas infarcts in the territory of the PICA or SCA usually involve only the cerebellum. There are common anatomic variants, in which AICA dominance on one side and PICA dominance on the opposite side are commonly seen in normal person. At times either the AICA or PICA is absent or hypoplastic, in which case one AICA-PICA supplies the usual territory of both arteries. The internal auditory artery (IAA) is a usual branch of AICA and supplies the eight cranial nerve, the cochlea and vestibular labyrinth. In terms of collateral circulation, in addition to the dorsolateral pons and middle cerebellar peduncle, which are known to be sensitive to ischemia, inner ear is also particularly vulnerable to ischemia since it is supplied entirely by the IAA that is an end artery with minimal collaterals from the otic capsule and has complete absence of collateral circulation.[1,10,11] By contrast, the retrocochlear eight nerve has an abundant collateral blood supply arising from the lateral medullary artery, the arteries supplying the adjacent dura matter and the petrous bone, and the inferior lateral pontine artery.[12-14] A typical pattern of AICA territory infarction on brain MRI is shown in Fig. 1.

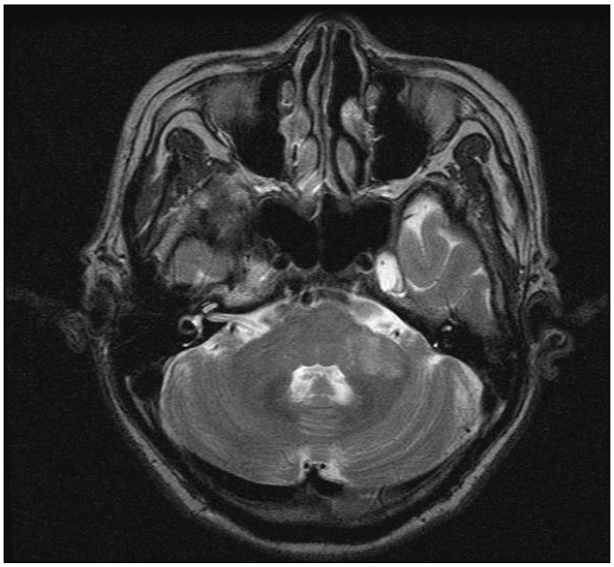

Fig. 1. MRI finding in a patient with AICA artery territory infarction. T2-weighted MRI scan of the brain demonstrated hyperintense foci involving the left middle cerebellar peduncle. AICA: anterior inferior cerebellar artery.

Labyrinthine infarction as an important sign for the diagnosis of anterior inferior cerebellar artery territory infarction

IAA infarction mostly occurs due to thrombotic narrowing of either the AICA itself, or of the basilar artery at the orifice of the AICA.[9] Occlusion of the IAA causes a sudden loss of both auditory and vestibular functions, resulting in acute onset of hearing loss and vertigo, so-called labyrinthine (inner ear) infarction. When a labyrinthine infarction occurs, infarction of the brainstem and/or cerebellum in the territory of the AICA is usually associated. However, AICA infarction rarely causes sudden hearing loss and vertigo without brainstem or cerebellar signs (i.e., isolated labyrinthine infarction), in which case an acute infarct may still be seen on brain MRI.[15] Hearing loss is a less widely appreciated and has been traditionally considered as a less common sign of AICA territory infarction. This may be explained by two facts. First, patients may not be aware of their hearing loss during an attack of vertigo and vomiting when the unilateral hearing loss is mild or the associated vertigo is severe. Second, neurologists do not included the audiogram as a routine diagnostic tool for the evaluation of AICA infarction. However, a recent report[16] showed that 11 out of 12 patients (92%) with AICA infarction showed labyrinthine infarction, which is clinically diagnosed by sudden sensorineural hearing loss on pure tone audiogram (PTA) and canal paresis to standardized bithermal caloric tests. Subsequently there have been other reports[17-19] emphasizing that AICA infarction commonly accompanied inner ear involvement and labyrinthine infarction is an important sign for the diagnosis of AICA infarction. Hearing loss is usually permanent, but dizziness and imbalance gradually improve with central compensation. However, a recent report[20] showed that, in addition to vertigo, most patients with labyrinthine infarction show improved hearing loss partially or completely.

Isolated labyrinthine infarction as a harbinger of incoming anterior inferior cerebellar artery territory infarction

Since the blood supply to the inner ear arises from the IAA, partial ischemia in the AICA territory could lead to an isolated acute audiovestibular loss. Several case reports[21-24] have shown that labyrinthine infarction may be served as an impending sign of incoming AICA territory infarction. A recent study[25] reported that approximately 8% (4/43) of patients with documented AICA territory infarction on brain MRI experienced an isolated audiovestibular disturbance (i.e., sudden onset of vertigo and hearing loss) with an initially normal MRI, and subsequently suffered from other neurological symptoms or signs indicative of AICA territory infarction. All of these studies suggest that the clinician should keep in mind the differential diagnosis of a posterior circulation stroke when caring for patients with acute peripheral-type audiovestibular syndrome, especially in patients with vascular risk factors and vertebrobasilar compromise on brain MRA, even though the classic brainstem or cerebellar signs are absent and MRI does not demonstrates acute infarction in the brain. Since the inner ear is not well visualized on routine MRI, a definite diagnosis of labyrinthine infarction is not possible unless a pathological study is done. It should be keep in mind that clinicians should consider all of the clinical evidence when attempting to determine the etiology of the acute audiovestibular syndrome rather than emphasizing that MRI is the best way to distinguish a viral (i.e., labyrinthitis) from a vascular (i.e., labyrinthine infarction) etiology.[26]

Characteristic pattern of vestibular dysfunction in anterior inferior cerebellar artery territory infarction

The most common pattern of vestibular dysfunction in AICA territory infarction was a combination of peripheral (i.e., unilateral canal paresis), and central ocular motor or vestibular signs (i.e., asymmetrically impaired smooth pursuit, bidirectional gaze-evoked nystagmus, or impaired modulation of the vestibular responses using visual input). These findings can be explained by the fact that AICA constantly supplied the peripheral vestibular structures such as the inner ear and vestibulocochlear nerve, in addition to the central vestibular structures.[9] As a result, in contrast to other cerebellar artery territory infarction, complete AICA infarction usually results in combined peripheral and central vestibular damages in addition to hearing loss, facial weakness, limb and facial sensory loss, gait ataxia, and cerebellar dysmetria. Sincee ischemia of any structures supplied by AICA can lead to vertigo, determining the responsible site (s) for the prolonged vertigo seems difficult in individual patient with AICA infarction. However, as noted above, most patients with AICA infarction had a unilateral weakness to caloric stimulation, suggesting that the vertigo was from the dysfunction of the peripheral vestibular structure at least in part. On the other hand, some patients showed normal caloric response, indicating that the in these patients vertigo may have resulted from ischemia to the central vestibular structures. Overall, prolonged vertigo in AICA infarction mostly results from ischemia to both the peripheral and central vestibular structures.

Spectrum of audiovestibuar loss in anterior inferior cerebellar artery territory infarction

It is well known that acute audiovestibular loss commonly occurs in acute ischemic stroke in the distribution of the AICA, but the detailed spectrum of audiovestibular dysfunction has

not been systematically studied in AICA infarction. Two dizziness clinics (from Keimyung and Seoul National Universities) investigated the pattern of audiovestibuar loss in AICA infarction. Eighty-two consecutive patients with AICA infarction diagnosed by MRI completed a standardized audiovestibular questionnaire and underwent a neuro-otological evaluation including bithermal caloric tests and PTA. As noted in Table 1, all but two (80/82: 98%) patients had acute prolonged (lasting more than 24 hours) vertigo and vestibular dysfunction of peripheral, central, or combined origin. The most common pattern of audiovestibular dysfunctions was the combined loss of auditory and vestibular function (*n*=49; 60%); selective loss of vestibular (*n*=4; 5%) or cochlear (*n*=3; 4%) function was rarely observed. We could classify AICA infarction into seven subgroups according to the patterns of neurotological presentations (Table 2): 1) acute prolonged vertigo with audiovestibular loss (*n*=35), 2) acute prolonged vertigo with audiovestibular loss preceded by an episode (s) of transient vertigo/auditory disturbance within 1 month before the infarction (*n*=13), 3) acute prolonged vertigo and isolated auditory loss without vestibular loss (*n*=3), 4) acute prolonged vertigo and isolated vestibular loss without auditory loss (*n*=4), 5) acute prolonged vertigo, but without documented audiovestibular loss (*n*=24), 6) acute prolonged vertigo and isolated audiovestibular loss without any other neurological symptoms/signs (*n*=1), 7) non-vestibular symptoms with normal audiovestibular function (*n*=2). Above findings suggested that infarction in the AICA territory mostly present with vertigo with a broad spectrum of audiovestibular dysfunctions. Considering the low incidence of selective cochlear or vestibular involvement in AICA infarction, vascular compromise appears to give rise to combined loss of auditory and vestibular functions while viral illness commonly presents as an isolated vestibular (i.e., vestibular neuritis) or cochlear loss (i.e., sudden deafness).

Table 1. Frequencies of audiovestibular dysfunctions in 82 patients with AICA territory infarction

	Frequency (*n*=82)
Vertigo as a presenting or main symptom at the time of AICA infarction	98% (80/82)
Central ocular motor or vestibular signs*	96% (79/82)
Vestibular labyrinth infarction	65% (53/82)
Cochlear infarction	63% (52/82)
Combined vestibulo-cochlear infarction	60% (49/82)
No auditory or vestibular infarction	32% (26/82)
Isolated vestibular infarction without cochlear involvement	5% (4/82)
Isolated cochlear infarction without vestibular involvement	3% (3/82)
Non-vertigo symptom as a presenting or main symptom at the time of AICA infarction	2% (2/82)
Isolated audiovestibular loss without central symptoms or signs	1% (1/82)

*Asymmetrical abnormalities of pursuit or optokinetic nystamus, gaze-evoked bidirectional nystagmus, or impaired modulation of the vestibular response using visual input.
AICA: anterior inferior cerebellar artery.

Otolith dysfunction in anterior inferior cerebellar artery territory infarction

Ocular tilt reaction (OTR), a sign of vestibular dysfunction in the roll plane, is characterized by the triad of conjugate ocular torsion, skew deviation, and head tilt. It is typically caused by damage to the brainstem tegmentum.[27] Recent studies[28,29] have showed that AICA infarction also can cause the partial OTR (i.e., ocular torsion/skew deviation without head tilt) associated with a deviation of the subjective visual vertical (SVV). Ipsiversive ocular torsion accompanying skew deviation was found in 6 patients with hearing loss and caloric weakness to caloric stimulation, whereas 3 patients with normal audiovestibular response showed contraversive ocular torsion only.[29] There was no difference in MRI findings in both groups with AICA territory infarction.[29] This phenomenon may result from infarction of the inner ear or the root entry zone of the eighth cranial nerve. Thus, the peripheral vestibular structure with inner ear probably plays a crucial role in determining the direction of ocular torsion associated

Table 2. Patterns of audiovestibular loss in 82 patients with AICA territory infarction

	Group 1 (*n*=35)	Group 2 (*n*=13)	Group 3 (*n*=3)	Group 4 (*n*=4)	Group 5 (*n*=24)	Group 6 (*n*=1)	Group 7 (*n*=2)
Presented with vertigo	+	+	+	+	+	+	-
Combined audiovestibular loss	+	+	-	-	-	+	-
Isolated auditory loss	-	-	+	-	-	-	-
Isolated vestibular loss	-	-	-	+	-	-	-
Normal audiovestibular function	-	-	-	-	+	-	+
Associated with ocular motor dysfunction	+	+	+	+	+	-	-
Associated with other neurological symptoms or signs	+	+	+	+	+	-	+
Prodromal audiovestibular disturbance	-	+	-	-	-	-	-

AICA: anterior inferior cerebellar artery.

with AICA territory infarction.[29]

Acute vestibular syndrome due to posterior inferior cerebellar artery territory cerebellar infarction

Clinical anatomy

The PICA most often arises from the vertebral artery, and rarely from the basilar artery. The common trunk of the PICA gives rise to a medial branch and a lateral branch. The medial PICA supplies the inferior vermis including the nodulus and uvula, and the inferior cerebellar hemisphere.[30] Since the nodulus, a critical site for modulating the vestibulo-ocular reflex, is constantly supplied by the medial PICA, infarcts limited to the medial PICA infarction may present as purely vestibular syndromes with severe vertigo, nausea, vomiting, and postural instability.[30,31] Because the lateral PICA usually supplies the caudal portion of the lateral cerebellar hemisphere, which is known to be related to the limb coordination, infarcts limited to the lateral PICA can lead to dysmetria on ipsilateral limb, gait disturbance, and hypotonia of the ipsilateral arm and leg without accompanying severe vertigo or vomiting.[32] A typical pattern of PICA territory infarction on brain MRI is shown in Fig. 2.

Pseudo-acute peripheral vestibulopathy associated with medial posterior inferior cere-bellar artery territory cerebellar infarction

The classic medial PICA cerebellar ischemic stroke syndrome is characterized by severe vertigo, vomiting, prominent axial (body) lateropulsion, dysarthria, and limb dysmetria. The key structure responsible for vertigo is the nodulus, which is strongly connected to the ipsilateral vestibular nucleus and receives direct projections from the labyrinth.[32,33] Functionally, nodulovestibular Purkinje fibers have an inhibitory effect on the ipsilateral vestibular nucleus.[33,34] Since extremity ataxia can be minimal or absent with mPICA cerebellar infarction particularly if the infarcts is small,[35,36] the clinical pattern of cerebellar infarction in the territory of the medial PICA can mimic APV. As many as 25% of patients with vascular risk factors who presented to an emergency medical setting with isolated severe vertigo, nystagmus and postural instability have a cerebellar infarction in the territory of the medial PICA.[37] Recent case reports[38,39] also described an unique clinical presentation of a vascular vestibular syndrome that is characterized by severe vertigo, ipsilesional spontaneous nystagmus, and contralesional axial lateropulsion due to the medial PICA cerebellar infarction, in which the clinical symptoms were similar to those of APV contralateral to the side of lesion on brain MRI. All of these reports suggest that patients with vertigo associated with medial PICA territory cerebellar infarction are often misdiagnosed with APV.

I systemically reviewed the clinical findings of 240 patients with isolated cerebellar infarction to identify the best tool for differentiating cerebellar infarction from APV.[40] Approximately 11% (25/240) of patients with isolated cerebellar infarction had isolated vertigo only, and most (24/25: 96%) had an infarct in the medial PICA territory including the nodulus. The key findings differentiating isolated vertigo associated

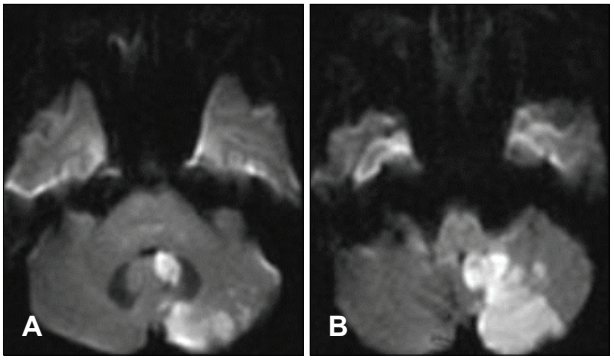

Fig. 2. MRI finding in a patient with infarction in the territory of the medial branch of the PICA. Diffusion-weighted axial images (A and B) of the brain MRI showed an acute infarct in the left medial caudal cerebellum. PICA: posterior inferior cerebellar artery.

Table 3. Vestibular findings and imbalance in 24 patients with "pseudo-APV" associated with medial PICA territory cerebellar infarction

	Patients
Head thrust test	Normal
SN	15
GEN	
Typical*	13
Unidirectional†	4
Gaze to only lesion side	7
Asymmetrical pursuit‡	6
Asymmetrical OKN	4
Canal paresis	None
Imbalance	
Grade	
1	7
2	1
3	16
Direction	
Lesion side	17
Healthy side	7

*Direction-changed bidirectional gaze-evoked nystagmus that the intensity was maximal when gaze to the lesion side, †Direction-fixed unidirectional gaze-evoked nystagmus beating toward the side of lesion, ‡Ipsilateral impairment of smooth pursuit with frequent corrective saccade. Canal paresis defined as side differences more than 25% at bithermal caloric stimulation.
APV: acute peripheral vestibulopathy, PICA: posterior inferior cerebellar artery, SN: spontaneous nystagmus, GEN: gaze evoked nystagmus, OKN: optokinectic nystagmus.

with medial PICA infarction from APV were the normal head impulse and caloric test results in cerebellar infarction (Table 3). Since the head impulse test can be performed at the bedside without special equipments, it is invaluable for separating pseudo-APV due to cerebellar infarction. Physicians who evaluate stroke patients should be trained to perform and interpret the results of the head impulse test. The prominent cerebellar signs, particularly severe axial instability and direction changing gaze-evoked nystagmus (occurred in 71% and 54%, respectively in the aforementioned series)[40] can also help in the differential, but these findings are less reliable and the findings in some patients with central vertigo are similar to those with peripheral vertigo. The significance of head impulse test for differentiating cerebellar stroke from APV has been confirmed by another recent paper[41] that showed that a negative head impulse test (i.e., normal vestibulo-ocular reflex) is strongly suggestive of a central lesion with a pseudo-APV presentation.

For patients with spontaneous prolonged vertigo, in addition to the obvious cases with associated neurological symptoms or signs, MRI to rule out medial PICA territory cerebellar infarction should be considered in 1) older patients presenting with isolated spontaneous prolonged vertigo, in 2) any patient with vascular risk factors and isolated spontaneous prolonged vertigo who has a normal head impulse test, and in 3) any patient with isolated spontaneous prolonged vertigo who has a direction-changing gaze-evoked nystagmus or severe gait ataxia with falling in the upright posture.[42,43]

Although small PICA territory cerebellar infarction generally has a benign prognosis, isolated PICA territory cerebellar infarction usually results from emboli originating from the heart or great vessels,[43] and recurrent emboli require appropriate treatments. Cerebellar infarction causes brain swelling in up to 25% of cases; PICA territory infarcts are more likely to produce a mass effect than SCA territory infarcts.[44,45] Large PICA territory cerebellar infarction can cause brainstem compression, hydrocephalus, cardiorespiratory complications, coma, and death.[46]

Otolith dysfunction in posterior inferior cerebellar artery territory cerebellar infarction

Although OTR and its components such as head tilt, ocular torsion, and skew deviation, as well as tilts of the perceived SVV are usually considered as a sign of brainstem dysfunction,[25] recent studies[47,48] have shown that cerebellar dysfunction can also cause partial (incomplete) OTR. One case report[47] described two patients with isolated medial PICA territory cerebellar stroke who showed a contraversive partial OTR (i.e., skew torsion without head tilt) with a contraversive deviation of the SVV. The authors speculated that interruption of nodular inhibitory projections to graviceptive neurons in the ipsilesional vestibular nuclei caused the contraversive conjugate ocular torsion. Lesion of the dentate nucleus can also lead to tilts of the SVV in the contraversive direction (i.e. a vestibular tone imbalance to the contralateral side), whereas cerebellar lesions excluding the dentate nucleus can induce a tone imbalance to the ipsilesional side.[48]

Acute hearing loss associated with non- anterior inferior cerebellar artery (mostly posterior inferior cerebellar artery) territory cerebellar infarction

Although the most commonly infarcted territory on brain MRI associated with acute hearing loss was in the distribution of the AICA, the PICA territory cerebellar infarction can rarely cause acute hearing loss because the IAA sometimes originates from the PICA or directly from the basilar artery.[49] Mazzoni[12] described a PICA origin of the labyrinthine artery in 3 out of 100 temporal bone dissections. More interestingly, in my series, 7 (1%) of 685 patients with vertebrobasilar ischemic stroke (VBIS) had acute unilateral hearing loss associated with non-AICA territory VBIS, in which PICA territory cerebellar infarction (5/7: 71%) was most common affected site.[50] Although the site of injury responsible for hearing loss in these patients can not be confirmed without pathological examination, a detailed auditory function testing indicates a cochlear site of injury.[50] Acute hearing loss associated with non-AICA territory VBIS is probably attributable to damage to the peripheral auditory system with the inner ear that is supplied by the IAA mostly originated from the PICA.[49]

Acute vestibular syndrome due to superior cerebellar artery territory cerebellar infarction

Clinical anatomy

The SCA divides into the lateral and medial branches of SCA after it arises from the basilar artery just proximal to its bifurcation into the posterior cerebral artery.[51] The medial SCA divides into the vermian and the paravermian arteries, as well as into the intermediate (hemispheric) arteries.[51,52] The vermian branches supply the ipsilateral half of the rostral vermis. The hemispheric branches run obliquely, dorsally, and laterally along the rostral cerebellar hemisphere and supply the most of the dorsomedial surface of the rostral cerebellar hemisphere. Rostral lateral cerebellar hemisphere is related predominantly to limb control while the rostral vermis is related to gait and postural control. Functional MRI disclosed that foot movements activated areas within the ipsilateral central lobule of the rostral vermis that are consistently supplied by the medial SCA.[53] A typical pattern of SCA territory infarction on brain MRI is shown in Fig. 3.

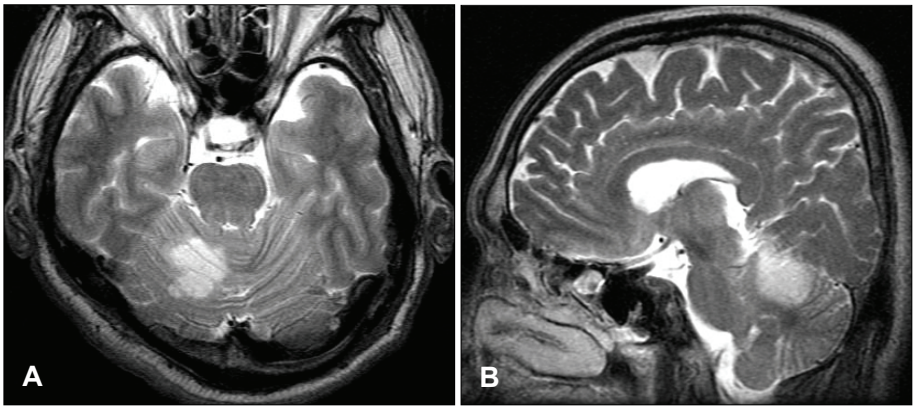

Fig. 3. MRI finding in a patient with infarction in the territory of the medial branch of the SCA. T2-weighted axial (A) and sagittal (B) images of the brain MRI showed an acute infarct involving the rostral paravermal region of the right anterior lobe. SCA: superior cerebellar artery.

Table 4. Differentiating between common cerebellar ischemic stroke syndromes focused on neuro-otological aspects

	Vertigo/related structure	CP/related structure	Hearing loss/related structure	Gait ataxia
AICA CI	++/inner ear, 8th nerve, VN, Flocculus	++/inner ear, 8th nerve, VN	++/ inner ear, 8th nerve, Cochlear nucleus	+
Medial PICA CI	++/Nodulus	None	Rare	++
Lateral PICA CI	None	None	None	None
Medial SCA CI	Rare	None	Rare/lateral lemniscus	++
Lateral SCA CI	None	None	None	+

CP: canal paresis, VN: vestibular nucleus, AICA: anterior inferior cerebellar artery, PICA: posterior inferior cerebellar artery, SCA: superior cerebellar artery, CI: cerebellar infarction.

Body lateropulsion as a presenting symptom of medial superior cerebellar artery territory cerebellar infarction

Since the superior cerebellum supplied by the SCA does not have significant vestibular connections, cerebellar infarction in the SCA rarely causes vertigo.[54,55] The vestibulo-ocular portion of the cerebellum is located primarily in the flocculonodular lobes, which are supplied by branches of the AICA and PICA. The low incidence of vertigo in SCA distribution may be a useful clinical distinction from PICA or AICA cerebellar infarction in patients with acute vertigo and limb ataxia.[54,55] Among the broad spectrum of clinical manifestations of SCA territory infarction, infarction in the territory of the lateral SCA is the most common, representing about a half of the cases.[54-56] Lateral SCA territory cerebellar infarction is characterized by dizziness, nausea, unsteadiness, mild truncal ataxia, and severe limb ataxia.[54-56] A recent study suggested that the most prominent clinical presentation in the medial SCA territory cerebellar infarction is severe gait ataxia with a sudden fall or severe veering, observed in 11 (76%) of 14 patients with isolated medial SCA territory cerebellar infarction.[57] Prominent body lateropulsion in isolated medial SCA territory cerebellar infarction may be explained by involvement of rostral vermis that is related predominantly to gait, muscle tone, and postural control.[53] The high incidence of sudden falling with body lateropulsion in the medial SCA cerebellar infarction may be a useful clinical distinction from the lateral SCA cerebellar infarction in patients with acute dizziness and postural instability. Table 4 summarizes the differential points among three common cerebellar ischemic stroke syndromes focused on neuro-otological aspects.

Acute Vestibular Syndrome due to Cerebellar Hemorrhage

Cerebellar hemorrhage is also a common cause of vertigo in older patients, especially in those with hypertension. The initial symptoms of acute cerebellar hemorrhage are vertigo, nausea, vomiting, headache, and prominent body lateropulsion with falling to lesion side. The clinical features are similar to those of acute cerebellar infarction and might be confused with an APV. Patients with cerebellar hemorrhage usually complain of more severe occipital headache and nuchal rigidity than in cerebellar infarction. Approximately 50% of patients lose consciousness within 24 hours of the initial symptoms, and 75% become comatose within 1 week of the onset.[53] The condition is often fatal unless surgical decompression is performed. A widely accepted neurosurgical adage is to evacuate a cerebellar hemorrhage that is more than 3 cm in cross-sectional diameter by CT scan.[59]

Conclusion

Since cerebellar stroke syndrome can produce unique symp-

toms and signs, recognizing the characteristic patterns of each individual cerebellar stroke syndrome is a key to the efficient diagnosis and treatment of this group of patients. The head impulse test is a useful bedside tool for differentiating acute cerebellar infarction from more benign disorders involving the inner ear.

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