Isolated Vertigo Caused by Anterior Inferior Cerebellar Artery Infarction—A Case Report

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*Anterior inferior cerebellar artery (AICA) infarct occurs infrequently. Peripheral facial palsy, deafness, tinnitus, and trigeminal sensory deficit are common presentations. Vertigo as the only clinical feature of AICA infarct is rare. A 46-year-old woman with an acute vertigo attack is presented. She had risk factors for cerebrovascular diseases. At the time of infarction, she developed nystagmus, gait ataxia, and limb dysmetria. Magnetic resonance imaging (MRI) on T2-weighted images showed hyperintense lesions in middle cerebellar peduncle, anterior inferior cerebellum, and flocculus corresponding to the distribution of AICA. Since nystagmus and gait ataxia do not distinguish between the central or peripheral causes of vertigo, AICA infarct could masquerade as peripheral vestibular lesions. A high suspicion of AICA infarction should be considered in managing patients with acute vertigo and limb dysmetria, especially in patients with risk factors for stroke. (**Tzu Chi Med J** 1999; **11**: 279-283)*

Key words: ***vertigo, AICA infarct, hypertension, diabetes mellitus, magnetic resonance imaging***

INTRODUCTION

Vertigo is a common symptom, usually due to the disturbance in the peripheral or central pathways of the vestibular system. Although vertigo often occurs with other neurologic symptoms, it can occur in isolation. Patients with cerebellar infarction can present with vertigo and resemble peripheral vestibulopathy [1]. The anterior inferior cerebellar artery (AICA) supplies inner ear, the anterolateral pons, middle cerebellar peduncle, anterior inferior cerebellum, and the flocculus [2]. Ischemia of any of these structures may lead to vertigo. In AICA infarcts, brainstem signs predominate [3]. Vertigo presented as the sole symptom of an AICA infarct is rare. A patient with isolated vertigo and AICA infarction with no brainstem symptoms or signs is described.

CASE REPORT

A 46-year-old married woman with a 5-year history of hypertension and diabetes had an acute episode of vertigo, nausea, and vomiting at 1:00 a.m., on May 29, 1998. She was hospitalized for sustained vertigo. She had no history of stroke or transient ischemic at-

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tacks. Her blood pressure was 200/110 mmHg and pulse rate was 76 beats per minute. No carotid bruits were audible. Neurologic examination showed isocoric pupils, prompt light reflex, intact visual field, full extraocular movement, and bilateral equal palpebral fissure. There was, however, a horizontal gaze-evoked nystagmus in the rightward position. Trigeminal sensation, facial motor, hearing, swallowing, and tongue movement were normal. She had right limb dysmetria and gait ataxia. The muscle strength was normal in all limbs. Deep tendon reflexes were normal. Babinski signs were absent. All sensory modalities were normal. Liver, renal function, lipid study and electrolytes test results were all within reference ranges. Fasting blood glucose level was 272 mg%.

Electrocardiographic and echocardiographic findings revealed no cardiac source of emboli. The magnetic resonance imaging (MRI) of the brain disclosed hyperintense lesions on T2-weighted images in the right anterior inferior cerebellum, right middle cerebellar peduncle and right flocculus (Fig.1, 2). The magnetic resonance angiography (MRA) revealed a hypoplastic right vertebral artery (Fig. 3). Transcranial doppler (TCD) demonstrated normal velocity in basilar and bilateral vertebral arteries. Brainstem auditory evoked potential (BAEP) tests revealed prolonged interpeak interval of right wave I-III. Pure tone audiometry was normal.

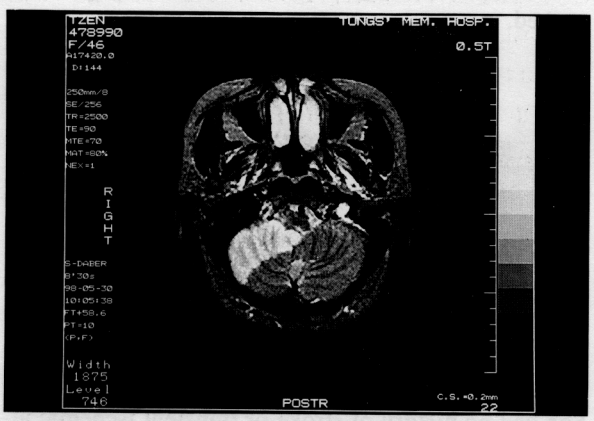

Fig. 1. Axial view: T2-weighted image of MRI reveals an infarction of the right anterior inferior area of the cerebellum.

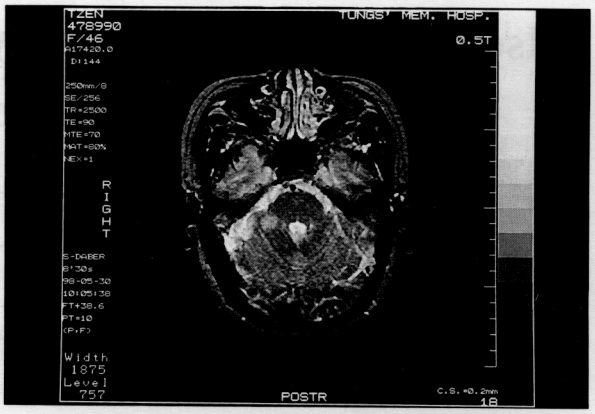

Fig. 2. Axial view: T2-weighted image of MRI shows an infarction in the right middle cerebellar peduncle and flocculus.

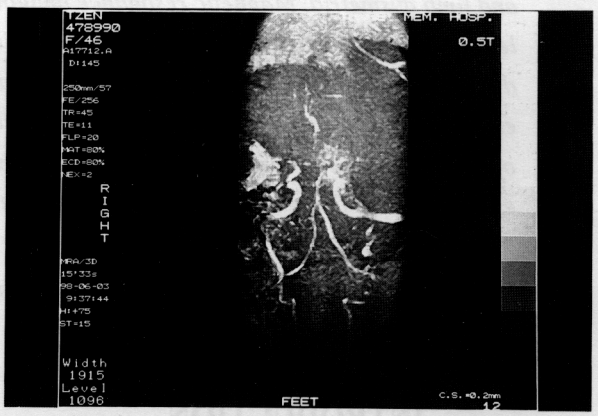

Fig. 3. Coronal view: MRA demonstrates a hypoplastic right vertebral artery and the patent basilar artery.

The vertigo subsided after 7 days of hospitalization. She showed mild improvement in nystagmus and right limb coordination during 2 months of follow-up.

DISCUSSION

Vertigo, defined as an illusion of movement, indicates an imbalance within the vestibular system. Vestibular neuronitis, labyrinthitis, benign positional vertigo, and Meniere's syndrome are common causes of pe-

ripheral vertigo. Transient vertebrobasilar ischemia, posterior fossa tumors, multiple sclerosis, and cerebellar infarction account for the cases of central vertigo. Peripheral vestibular dysfunctions are usually of short duration and are characterized by severe, often paroxysmal vertigo accompanied by auditory symptoms. Nystgmus is present and unidirectional. On the contrary, central vestibular dysfunctions are prolonged. Signs of brainstem and cerebellum are present. Vertigo is less severe and nystagmus can be unidirectional [4]. My patient had acute persistent vertigo and cerebellar signs which were similar to those for central lesions. Ataxia, dysmetria, and a history of hypertension and diabetes suggested cerebellar infarct. The MRI study confirmed the diagnosis.

The cerebellum is supplied by the superior cerebellar artery (SCA), the AICA, and the posterior inferior cerebellar artery (PICA). In PICA infarcts, a triad of vertigo, headache, and gait imbalance predominate. In SCA infarcts, gait disturbance predominates and vertigo occurs less frequent [5]. In AICA infarcts, the full syndrome includes vertigo, nystagmus, tinnitus, hearing loss, peripheral facial palsy, Horner's syndrome, ipsilateral trigeminal sensory loss, contralateral pain and temperature sensory loss, dysmetria, and hemiataxia [3, 6]. Trigeminal sensory deficit, severe peripheral facial palsy, and vestibulocochlear dysfunction are the characteristics of infarction in the AICA territory [3].

In my patient, the cerebellar infarct did not involve the brainstem and the vestibular connection to flocculus was mainly supplied by the AICA [7], thus vertigo became the only clinical manifestation. Oas and Baloh [8] described two patients with isolated vertigo attacks due to transient ischemia with final progression into the AICA infarct. They emphasized a combination of peripheral symptoms, including hearing loss and tinnitus, and the central finding of facial sensory loss and hemiataxia subsequent to the infarction at lateral pons and middle cerebellar peduncle. The cause of isolated vertigo in my patient was due to a unique central lesion, which was different from the cause in the patients of Oas and Baloh.

Along the direction of vascular branches, the AICA separates into premeatal recurrent penetrating artery (RPA), internal auditory artery (IAA), postmeatal RPA, and terminal branches. Premeatal RPA supplies the anterolateral pons. IAA supplies the inner ear. Postmeatal RPA supplies the middle cerebellar peduncle. AICA terminal branches supply the anterior inferior cerebellum and flocculus [8]. MRI of my patient identified infarctions in the middle cerebellar peduncle, anterior inferior cerebellum and flocculus, implying the occlusion of postmetal RPA.

Previous reports [3,5] pointed out that the causes of the three cerebellar arteries infarctions are different. SCA infarct is mostly cardioembolic, PICA infarct is equally divided between embolic and atherosclerotic causes, while AICA infarct often relates to atherothrombosis of caudal basilar artery or the rostral vertebral artery. The clinical findings in my patient showed the atherothrombosis risk factors: hypertension and diabetes, but did not show evidence of cardioembolism. These are consistent with the report of Amarenco and Hauw [3].

Amarenco et al [9] divided AICA infarcts into two types, pure AICA infarcts and AICA plus infarcts. The pure type developes secondarily to basilar artery plaques or microatheroma, extending into and obstructing the origin of the AICA. Patients with pure AICA infarcts are hypertensive, diabetic, but have no prodromata. In contrast, the AICA plus type have basilar artery occlusion at the AICA level, which is compensated by flow from the PICA and posterior cerebral artery. Patients with AICA plus infarcts are hypertensive and usually have a history of transient ischemic attacks. My patient with hypertension and diabetes shows patent basilar artery on MRA, normal basilar artery velocity in TCD, but do not have prodromata. These findings conform to pure AICA infarcts. The thrombus may have propagated to occlude a postmeatal RPA.

An overlapping exists between the territories of the PICA and the AICA, since there is a balance in caliber between these two arteries. When the PICA is hypoplastic, the AICA can have a maximal extent, taking over

the territory of the PICA to supply the whole anteroinferior portion of the cerebellum. It is also true that the anteroinferior portion of the cerebellum can be supplied by the PICA or AICA [10]. The infarction involves the middle cerebellar peduncle which is the core of the AICA territory [3]. The AICA infarct in my patient was diagnosed because the middle cerebellar peduncle was involved. Atkinson [2] described two variations in AICA anatomy. An anastomosis between AICA and PICA is classic AICA anatomy with both arteries of equal dominance. Another common variant is the AICA dominance on one side associated with the ipsilateral hypoplastic vertebral artery and contralateral PICA dominance. MRA of my patient displayed a hypoplastic right vertebral artery. The AICA anatomy in my patient belonged to the latter variation.

The BAEP examination is sensitive in detecting lesions of the auditory pathways of the brainstem. The presence of the prolonged interpeak interval of wave I-III in my patient suggested lateral pontine dysfunction which may have been caused by ischemia or edema.

In conclusion, AICA infarct is an infarction of pontocerebellar distribution. When an infarction is limited to the cerebellar hemisphere, vertigo may be the only isolated symptom which might be misdiagnosed as an inner ear disease. In addition, cerebellar infarcts are difficult to diagnose with computed tomography (CT) [11], therefore, AICA infarcts may be ignored clinically. Taking into account some clues such as the duration of attacks, the age of the patient, the vascular risk factors, the cranial nerves palsy, and the cerebellar signs, it is possible to predict the precise cause of isolated vertigo.

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由前下小腦動脈梗塞引起的暈眩症一病例報告

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前下小腦動脈梗塞不多見。周邊顏面神經麻痺、耳聾、耳鳴及三叉感覺異常為其常見的病徵，但只以暈眩症表現的前下小腦動脈梗塞更是罕見。本文報告一位急性暈眩症的46歲女性病人，她有腦血管疾病的危險因子及眼球震顫，步履運動失調和肢體辨距障礙。核磁共振掃瞄顯示前下小腦動脈所支配的中小腦腳，前下小腦及小葉產生梗塞徵象。由眼球震顫和步履運動失調無法區分中樞性或周邊性之暈眩症，因此前下小腦動脈梗塞與週邊前庭病變可產生臨床相似的症狀。有腦中風危險因子的病人，若有急性暈眩症和肢體辨距障礙時，應考慮到前下小腦動脈梗塞引起之可能性。(**慈濟醫學** 1998; **11**: 279-283)

關鍵語：暈眩症，前下小腦動脈梗塞，高血壓，糖尿病，核磁共振

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