Atherothrombotic Cerebellar Infarction
Vascular Lesion–MRI Correlation of 31 Cases

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Background and Purpose—Correlation of MRI findings with atherosclerotic vascular lesions has rarely been attempted in patients with cerebellar infarction. The aim of this study was to correlate the MRI lesions with the vascular lesions seen on conventional cerebral angiography in cerebellar infarction.

Methods—The subjects included 31 patients with cerebellar infarcts who underwent both MRI and conventional cerebral angiography. We analyzed the risk factors, clinical findings, imaging study, and angiography results. We attempted to correlate MRI lesions with the vascular lesions shown in the angiograms.

Results—The vascular lesions seen on angiograms were subdivided into 3 groups: large-artery disease (n = 22), in situ branch artery disease (n = 6), and no angiographic disease with hypertension (n = 3). The proximal segment (V1) lesions of vertebral artery were the most common angiographic features in patients with large-artery disease in which stroke most commonly involved the posterior inferior cerebellar artery (PICA) cerebellum. The V1 lesions with coexistent occlusive lesions of the intracranial vertebral and basilar arteries were correlated with cerebellar infarcts, which had no predilection for certain cerebellar territory. The intracranial occlusive disease without V1 lesion was usually correlated with small cerebellar lesions in PICA and superior cerebellar artery (SCA) cerebellum. The subclavian artery or brachiocephalic trunk lesion was associated with small cerebellar infarcts. The in situ branch artery disease was correlated with the PICA cerebellum lesions, which were territorial or nonterritorial infarct. No angiographic disease with hypertension was associated with small-sized cerebellar infarcts within the SCA, anterior inferior cerebellar artery, or SCA cerebellum.

Conclusions—Our study indicates that the topographic heterogeneity of cerebellar infarcts are correlated with diverse angiographic findings. The result that large-artery disease, in which nonterritorial infarcts are more common than territorial infarcts, is more prevalent than in situ branch artery disease or small-artery disease, suggest that even a small cerebellar infarct can be a clue to the presence of large-artery disease. (Stroke. 1999;30:2376-2381.)

Key Words: angiography | cerebellar infarction | magnetic resonance imaging

With the advent of MRI, it has become possible to diagnose cerebellar infarctions, even very small-sized infarctions, and delineate their topography with high sensitivity.1,2 Infarcts can be divided into territorial infarcts (ie, in the territory of the posterior inferior cerebellar artery [PICA],4-6 anterior inferior cerebellar artery [AICA],7,8 and superior cerebellar artery [SCA];9,10) and nonterritorial infarcts that are small cerebellar infarcts,<2 cm in diameter.11,12 Whether these MRI findings correlate with various atherosclerotic vascular lesions in posterior circulation remains to be explored. We attempted to correlate the vascular lesions shown in the angiogram with the MRI.

Subjects and Methods

From January 1998 through April 1999 we prospectively collected data from patients with cerebellar infarction at Kyungpook National University Hospital. Of these patients, we selected 31 patients in whom (1) MRI showed an appropriate cerebellar lesion and (2) transfemoral conventional angiography was performed. All patients had been diagnosed by MRI; axial T2 (repetition time 2500 ms; echo time 80 ms), axial T1, and gadolinium-enhanced T1-weighted scans were performed in the horizontal plane at 3-mm intervals from the medulla to the midbrain. There is no standardized selection criteria for performing conventional angiography in our hospital, but angiography is not usually performed in patients who have a known cardiac source of embolism, are elderly, or would not give consent.

We used the following definitions for vascular risk factors. Arterial hypertension was defined in 2 ways: (1) if the patient was being treated with antihypertensive drugs or his or her medical record gave such a diagnosis, (2) 2 blood pressure recordings with both a systolic blood pressure >160 mm Hg and a diastolic blood pressure >95 mm Hg. A patient was defined as a smoker if he or she was a current smoker in the last 12 months. Diabetes mellitus was defined in 2 ways: (1) by history if the patient had this diagnosis and...
(2) if there were at least 2 fasting glucose concentrations >7.8 mmol/L (140 mg/dL). Hypercholesterolemia was diagnosed if the patient had this diagnosis and was receiving treatment or if a fasting cholesterol level was >6.21 mmol/L (>240 mg/dL).

Because we attempted to focus on the atherosclerotic vascular lesion, patients were excluded who had a known cardiac source of embolism such as recent myocardial infarction (<3 weeks), known atrial fibrillation with or without mural thrombus, mitral stenosis or prosthetic valve, dilated cardiomyopathy, sick sinus syndrome, acute bacterial endocarditis, or patent foramen ovale. All patients had electrocardiography and transthoracic echocardiography.

Vascular lesions seen on conventional cerebral angiography were classified as follows.

Large-artery disease was defined as present in patients (1) with at least 1 conventional risk factor for atherosclerosis, (2) with angiographic evidence of vertebral or basilar artery atherosclerotic lesion, and (3) without a cardiac source of embolism. Large-artery disease was subdivided into V1 disease, V1 lesions with tandem intracranial occlusive disease, intracranial occlusive disease without V1 lesion, and subclavian artery or brachiocephalic trunk disease.

In situ branch artery disease included infarcts in the territory of a cerebellar artery in the absence of large artery lesions and of a cardiac source of embolism and in the presence of both at least 1 conventional risk factor for atherosclerosis and angiographically documented atherosclerotic lesion of 1 cerebellar artery (PICA, AICA, or SCA). This group was subdivided into PICA, AICA, and SCA disease.

No angiographic disease with hypertension was defined as (1) presence of hypertension, (2) no cardiac source of embolism, (3) normal angiogram in posterior circulation.

The patients’ MRI findings were copied from the original film (T2-weighted axial image of MRI) by one of the authors who was blinded to the angiographic findings. We used previously published templates to locate cerebellar infarcts.11,12 Angiographic results were schematically drawn by a neuroradiologist who was blinded to the MRI findings.

**Results**

**General Features**

We summarized the demographic characteristics, risk factors, and clinical features of 31 patients in the Table. There were 24 men and 7 women, 42 to 80 years of age (mean 59 years).
The patients’ most common symptoms were vertigo/dizziness (90%). Overt brain stem symptoms/signs (eg, Horner’s syndrome, hearing loss, tinnitus, limb weakness, sensory loss, facial palsy, dysphagia, or altered consciousness) were noted in 11 patients.

Correlation of Angiographic Results With MRI Findings

The patients’ MRI findings and angiographic results were combined and are presented in Figures 1, 2, and 3. There was large-artery disease in 22 patients, in situ branch artery disease in 6 patients, and no angiographic disease with hypertension in 3 patients.

We used the previously suggested method for measuring the arterial stenosis: \[ \% \text{stenosis} = \left(1 - \frac{D_{\text{stenosis}}}{D_{\text{normal}}} \right) \times 100, \]
where \(D_{\text{stenosis}}\) = diameter of the artery at the site of the most severe stenosis and \(D_{\text{normal}}\) = diameter of the artery at the widest, nontortuous segment that had parallel margins.13

Stenosis >50% was considered significant in this study.

Large-Artery Disease

There were 22 patients with large-artery disease (patients 1 through 22, Figure 1).

In this group, territorial infarcts were seen in 10 patients. It represented the SCA territory (n=3), AICA territory (n=5), and PICA territory (n=7). Of these 10 patients, 2 patients (patients 3 and 10) had all 3 cerebellar territories simultaneously involved. Patient 6 showed PICA and AICA territorial infarcts. Pons (n=2), midbrain (n=1), and medulla (n=3) were also involved in 4 patients. Multiple (at least 2 lesions) small-sized cerebellar infarcts in the SCA territory were also seen in patients 12 and 14. Nonterritorial infarcts were seen in 12 patients. It represented small lesions in the SCA territory (n=6), AICA territory (n=4), and PICA territory (n=5). Patients 13 and 19 showed multiple small-sized cerebellar lesions in the SCA and PICA territories. Multiple small lesions were also seen in the SCA and AICA territories in patient 8. Midbrain plus pons lesions (n=1) and medullar lesion (n=1) were observed in patient 22 and patient 2, respectively.
The proximal segment (V1) disease of vertebral artery was seen in 8 patients (patients 1 through 8). Six patients had V1 unilateral stenosis and 2 had unilateral occlusions. Stroke most commonly involved the PICA cerebellum (5 [63%] of 8 patients), of which 80% were territorial lesion. The AICA cerebellum (50%) and SCA cerebellum (38%) were also involved. Dizziness and ataxia were the most common symptoms. Only 1 patient had brain stem symptoms.

The V1 lesions with tandem intracranial occlusive disease were seen in 6 patients (patients 9 through 14). Four had unilateral V1 stenosis, 1 had unilateral V1 occlusive lesion, and 1 had bilateral V1 stenosis. Intracranial lesions involved intracranial vertebral artery (n = 3) or both intracranial vertebral and basilar arteries (n = 3). Of the 6 patients who showed evidence of intracranial vertebral artery lesions, 5 had unilateral occlusion and 1 had bilateral lesions (a combination of occlusion and stenosis). Of the 3 patients who had basilar artery lesions, 2 had basilar occlusion and 1 had a combination of stenosis and occlusion. In these patients with tandem lesions, 3 patients had pontine lesions, 3 patients had medullar lesions, and 1 had a midbrain lesion. Cerebellar lesions tended to be distributed within 3 cerebellar territories without any predilection. Three patients showed multiple bilateral small cerebellar lesions in the PICA cerebellum, the SCA cerebellum, or both (patients 12, 13, and 14). Brain stem symptoms were present in 5 (83%) of 6 patients. Three patients had altered consciousness.

Intracranial occlusive disease without V1 lesion was seen in 5 patients (patients 15 through 19). Two had unilateral intracranial vertebral artery lesions (stenosis in one and a combination of stenosis and occlusion in the other). One patient had bilateral intracranial vertebral artery occlusions. Two patients had unilateral intracranial vertebral artery lesion and coexistent basilar artery stenosis. Four of 5 patients had cerebellar lesions in the PICA and SCA cerebellum, of which most were small infarcts. Dizziness and ataxia were the most common symptoms.

The subclavian artery or brachiocephalic trunk disease was seen in 3 patients (patients 20 through 22). One had a unilateral occlusion and 2 had unilateral stenosis. One patient who had a concomitant rostral basilar artery occlusion showed subclavian steal syndrome. Midbrain, pons, and PICA territory nonterritorial lesions were seen in this patient. Two patients had small cerebellar lesions in the SCA territory. The patient showing subclavian steal syndrome had brain stem symptoms and altered consciousness. Others had dizziness, dysarthria, and ataxia.

In large-artery disease, cerebellar infarcts with multiple small lesions (at least 2 small lesions in different territories) were seen in 3 patients (patients 8, 13, and 19), each one in V1 disease, in V1 lesions with tandem intracranial occlusive disease, and in intracranial occlusive disease without V1 lesion.

In Situ Branch Artery Disease

There were 6 patients with in situ branch artery disease (patients 23 through 28, Figure 2). In this group, territorial infarcts were seen in 3 patients. It represented the PICA territory in all patients. Of these 3 patients, 2 had coexistent medullar lesions. Nonterritorial infarcts were seen in 3 patients. The small cerebellar lesions were present in the SCA territory (n = 1) and PICA territory (n = 2). Of these 3 patients, 2 had medullar lesions. The SCA was involved in 5 patients (stenosis in 4 patients, occlusion in 1 patient). Of these 5 patients, 3 patients had territorial lesions and 2 had small cerebellar lesions. Vertigo and dizziness were the most common symptoms. Four (80%) of 5 patients showed brain stem signs. The SCA causing a small lesion in its territory was involved in 1 patient. Dysarthria and limb dysmetria were prominent in this patient.

No Angiographic Disease With Hypertension

There were 3 patients with no angiographic disease (patients 29 through 31, Figure 3). In this group, the small cerebellar infarcts were present in the PICA, AICA, and SCA territories. Multiple small lesions were seen in the PICA and SCA territories in 1 patient. All patients showed normal angiograms in the posterior circulation. Dizziness was the most common symptom. Two patients had multiple old lacunar infarcts in basal ganglia, thalamus, and centrum semiovale. One patient had a significant stenosis of the right internal carotid artery.

Discussion

Although causes and mechanisms of cerebellar infarction have been previously studied,2–12,14 how the diverse MRI
findings correlate with vascular lesions has not been studied sufficiently. Our study is an attempt to analyze the location and size of the MRI-identified lesions with conventional angiographic findings in a relatively large numbers of patients. The results led us to ascertain how the heterogeneous vascular lesions are related to the diverse patterns of cerebellar infarcts.

In this study, angiography demonstrated large-artery disease in 71%, in situ branch artery disease in 19.4%, and no angiographic disease with hypertension in 9.6%. Amarenco et al11 in their series found large-artery disease in 25 patients and in situ atherosclerotic occlusion in 8 patients as a mechanism of cerebellar infarct, suggesting a prevalence of large-artery disease over branch disease. However, several potential biases in our selection of patients may limit comparison with other studies and populations of studies. Conventional angiography was likely to have been performed in patients who were expected to have a gross vascular lesion. In addition, the Kyungpook National University Hospital is a referral center for patients with stroke. Hence, patients in our registry may have a larger percentage of diagnostically complex or unusual conditions compared with typical patients admitted to the hospital for stroke. Thus the prevalence of each of the pathogenetic mechanisms for cerebellar infarcts shown in our study may not be generalizable. Despite these limitations, we found that large-artery disease as an atherothrombotic cause of cerebellar infarcts was the most common angiographic finding in our study.

In our study, V1 disease of the vertebral artery was the most common angiographic feature in large-artery disease. Wityk et al15 described the most common site of infarction in patients with V1 lesion as the PICA cerebellum, the SCA territory cerebellum, and the occipital and temporal lobes. Caplan et al16 found that the most frequent recipient sites of intra-arterial embolism were the intracranial vertebral artery–PICA region and the distal basilar artery and its SCA and posterior cerebral artery branches. We found that the PICA cerebellum was the most common site of infarction in our study. Notably, the AICA cerebellum was second in this study. No patient had intracranial vertebral artery occlusion ipsilateral to the V1 lesion. Thus patients with V1 thrombus had artery-to-artery embolism to the PICA, AICA, and SCA, presumably. Considering that there are few data about the accuracy of MR angiography (MRA) in assessing the proximal vertebral artery, that MRA did not prove to be specific in grading stenoses of intracranial vertebrobasilar system,17 and that V1 lesions are common in patients with large-artery disease in our study, we speculate that conventional angiography should be considered to assess the proximal vertebral artery in evaluating patients with atherothrombotic cerebellar infarcts. The prevalence of territorial infarction in patients with V1 lesion might be related to the size of vessel involvement and status of collateral vessels. Dizziness and ataxia were the most common symptoms, as described by previous authors.15

The V1 lesions associated with coexistent occlusive lesions of the intracranial vertebral and basilar arteries were the second most common in large-artery disease. Cerebellar lesions tended to be distributed within 3 cerebellar territories without any predilection and tended to be multifocal. Half the patients had pons lesion. A previous study showed that patients with tandem intracranial occlusive lesions had the PICA cerebellum and pons infarcts typically and had multifocal lesions commonly.15 The difference in cerebellar lesion distribution may be attributed to relatively small numbers of patients with tandem lesions in our study compared with the previous study. The mechanism for stroke in this group could be explained by hypoperfusion caused by intracranial vascular lesion in most patients, but the potential contribution of the V1 lesion to either diminished blood flow or artery-to-artery embolization is uncertain. Half the patients in this group had altered consciousness with subsequent poor outcome.

Patients with intracranial occlusive disease without V1 lesions had small cerebellar lesions in PICA and SCA cerebellum in most cases. The infarcts could be due to hemodynamic effects of the intracranial lesions or artery-to-artery embolization.

Patients with subclavian or brachiocephalic trunk disease showed small cerebellar lesions that were presumed to be caused by artery-to-artery embolism except for 1 patient with subclavian steal syndrome. Reversal of blood flow in extracranial vertebral artery is a common, usually benign vascular disorder, which only occasionally produces cerebrovascular events.18 Failure of intracranial collateral circulation through the circle of Willis may lead to the development of cerebrovascular events in any of the territories involved in patients with subclavian steal syndrome. The small cerebellar infarct in our patient with reversed vertebral flow might result from restricted collateral blood flow to the basilar artery caused by rostral basilar artery occlusion combined with microembolism originating from areas of low flow velocity within vertebrobasilar arteries where activation of thrombogenesis was suspected to represent a major risk factor.

The fact that nonterritorial infarcts were found more commonly than territorial infarcts in our patients with large-artery disease suggests that even a small cerebellar infarct can be a clue to the presence of a large-artery lesion in the posterior circulation, including subclavian artery or brachiocephalic trunk, especially in patients with risk factors of atherosclerosis. The distribution and size of cerebellar infarcts presumably depend on the size of the embolus causing the infarct in most cases, but whether the hemodynamic mechanism contributes to the infarct or not is uncertain in patients with bilateral vertebral artery or basilar artery occlusive disease. Amarenco et al11 found that hemodynamic failure was the likely mechanism in 14% of patients with nonterritorial cerebellar infarcts in which coexistence of a small distal embolus could not be ruled out.

In patients with in situ branch artery disease, cerebellar infarcts usually involved the PICA cerebellum consisting of territorial or nonterritorial lesions. Kim et al19 found that cerebellar involvement was uncommon in patients with isolated PICA disease, suggesting the effectiveness of the collateral circulation in the cerebellum through the AICA or the SCA. Thus it appears that the extent of cerebellar lesions in PICA disease depends on the status of collateralization, but in those patients the potential role of embolization to a more distal artery is uncertain. Terao et al20 reported that no patients with PICA infarcts had cerebellar symptoms alone.
Brain stem signs were present in most patients with PICA cerebellar lesions in our study, which may prevent us from localizing PICA cerebellum lesions on clinical grounds in the absence of MRI.

Patients with no angiographic disease with hypertension had small infarcts in the AICA, PICA, or SCA cerebellum. Dizziness was the most common symptom. Small infarcts may be caused by coagulopathy such as thrombocytopenia, polycythemia, disseminated intravascular coagulation, and systemic lupus, but none of our patients had these conditions. A recent study emphasized the common occurrence of very small multiple cerebellar infarcts, of which small territorial infaracts may be end zone infarcts from the involvement of small distal arteries. The first MRI series of cerebellar infarction, which also discussed underlying causes for stroke, showed that multiple small infarcts were associated with vertebralbasilar atherosclerosis. We speculate that whereas multiple small cerebellar infarcts (such as cases 12 and 13) in patients with large-artery disease could be explained by a hemodynamic mechanism or artery-to-artery embolism, small-artery disease was a presumed mechanism in patients with no angiographic disease (patients 29 through 31; patient 29 had multiple small cerebellar infarcts) because this group had hypertension as a risk factor for small-artery disease and had no embolic source from the heart or large arteries. Bernasconi et al found that small-artery disease was uncommon in patients with multiple acute infarcts in the posterior circulation. In their study, the patients with small-artery disease had small-sized cerebellar infarcts and had long-standing hypertension or diabetes mellitus in the absence of potential arterial or cardiac sources of emboli. In Amarenco’s study, small cerebellar infarcts caused by intracranial atheroma were seen in 3 patients in the end artery disease group who had a history of hypertension or diabetes mellitus, or both and multiple other lacunes. Therefore small-artery disease was presumed to be an unusual condition in cerebellar infarcts and was associated with coexistent multiple other lacunes (eg, thalamus or basal ganglia) in patients with hypertension or diabetes mellitus. Small-artery disease was an uncommon cause of cerebellar infarcts in our study, which may be related to arterial anatomic disposition with progressively tapered arteries reaching the deep cerebellar white matter.

In summary, our study illustrates that various MRI findings of cerebellar infarcts correlate with diverse atherothrombotic vascular lesions. The V1 lesion of vertebral artery was the most common angiographic feature in patients with large-artery disease in which stroke most commonly involved the PICA cerebellum, of which most were territorial infarcts. The V1 lesions with coexistent occlusive lesions of the intracranial vertebral and basilar artery are associated with cerebellar infarcts, which tended to be distributed within 3 cerebellar territories without any predilection and tended to be multifocal. The intracranial occlusive disease without V1 lesion is commonly associated with small cerebellar lesions in the PICA and SCA cerebellum. The subclavian artery or brachiocephalic trunk lesion is associated with small cerebellar infarcts. The in situ branch artery disease usually produces PICA cerebellum lesions that are territorial or nonterritorial infarcts. The small-artery disease is associated with small-sized cerebellar infarcts within the SCA, AICA, or SCA cerebellum.

Our findings emphasize the heterogeneity of topographic and vascular lesional aspects of cerebellar infarcts. This suggests that early recognition of these types of atherothrombotic cerebellar infarct may have implications for therapy in the acute phase of stroke.

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References


