Patterns of Lateral Medullary Infarction

Vascular Lesion–Magnetic Resonance Imaging Correlation of 34 Cases

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Background and Purpose—Correlation of MRI findings with various vascular pathologies has rarely been attempted in patients with lateral medullary infarction (LMI). The aim of the present study was to correlate the diverse MRI lesions with the vascular lesions seen on conventional cerebral angiography in LMI.

Methods—The subjects included 34 patients with LMI who underwent both MRI and conventional angiography. We analyzed the risk factors, clinical features, MRI findings, and angiography results. The size of the infarction was also measured. We attempted to correlate the MRI findings with the vascular lesions shown in the angiograms.

Results—Presumed causes for infarction were atherothrombosis in 19 patients, arterial dissection in 8, cardiogenic embolism in 3, moyamoya disease in 1, small-vessel disease in 1, and embolism of unknown source in 2. Isolated posterior inferior cerebellar artery (PICA) disease (n=8) was usually associated with atherothrombosis and correlated with thin, round, or diagonal band–shaped lesions in the lateral-superficial area of the caudal medulla and/or dorsolateral portion of the rostral-middle medulla. Short-segment distal vertebral artery (VA) disease (n=9) was usually due to atherothrombosis and correlated with small lateral caudal and/or medium-sized, diagonal band–shaped rostral-middle medullary lesions. There were 13 patients with long-segment VA disease sparing (n=8) or involving (n=5) the proximal part of the VA with concomitant occlusion of the PICA in 7 patients. This vascular lesion produced either large MRI lesions extending ventrally (n=5; 4 were associated with VA dissection) or small lesions mimicking those produced by isolated PICA disease (n=8; 6 were associated with atherothrombosis and 1 patient had moyamoya disease). These large MRI lesions characteristically produced bilateral or contralateral trigeminal sensory involvement. Normal angiogram (n=4; 3 patients were presumed to have cardiac embolism, one lesion was associated with small-vessel infarction) was associated with small, round lesions that produced minor and fragmentary symptoms. Among these subgroups, the size of the infarct in the patients with long-segment VA disease due to dissection was significantly larger than that of the patients with other vascular lesions.

Conclusions—Our data suggest that the heterogeneous MRI lesions (and consequent clinical syndromes) of LMI are correlated with diverse angiographic findings, which in turn are due to different pathogenic mechanisms: etiology, location and size of the involved vessels, speed of the lesion development, and status of collateral channels. Generally, infarcts related to multiple vessel involvement, dissection, and poor collateral circulation are larger than those associated with single-vessel disease, long-standing atherothrombosis/cardiac embolism, and good collateralization. **(*Stroke*. 1998;29:645-652.)**

Key Words: medulla oblongata ■ cerebral infarction ■ magnetic resonance imaging ■ angiography ■ thrombosis ■ dissection

With the advent of MRI, medullary infarctions are now easily recognized,[1–6] and recent studies[4,6] have shown that the MRI-identified lesions are quite diverse and generally correlate with heterogeneous clinical syndromes in these patients. However, whether the diverse MRI lesions have any implication for different etiopathogeneses has not been studied sufficiently. Although one recent study[6] tried to address this issue, vascular status was analyzed for the most part with MR angiography, which according to our experience frequently failed to provide us with reliable information regarding the status of the PICA. Therefore, whether etiopathogenetic heterogeneity correlates with diverse medullary lesions remains to be explored. In the present study, we describe 34 patients with LMI in whom MRI showed appropriate lesions, and we attempted to correlate the MRI findings with the conventional angiography results.

Subjects and Methods

At the Asan Medical Center, we examined 64 patients with clinically suspected LMI between September 1994 and May 1997. Sixty-one of them underwent MRI; axial T2 (repetition time, 2500 ms; echo time, 80 ms), proton-density, and gadolinium-enhanced T1-weighted scans were performed in the horizontal plane at 3-mm intervals from the medulla to the midbrain. A sagittal T1-weighted image was also obtained. Of these patients, we selected 34 in whom (1) MRI showed an appropriate medullary lesion and (2) conventional (transfemoral) angiography was performed. There is no standardized selection criteria

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Selected Abbreviations and Acronyms

AICA = anterior inferior cerebellar artery
LMI = lateral medullary infarction
PICA = posterior inferior cerebellar artery
VA = vertebral artery

for performing conventional angiography in our hospital, but generally patients who were elderly, had a poor clinical status, or did not give consent did not undergo this study. Patients who underwent MR angiography were not included in the present study because the results of this technique were considered insufficient in the precise evaluation of the PICA.[6] Risk factors for stroke such as hypertension, diabetes mellitus, current cigarette smoking, habitual alcohol drinking (more than 2 times a week or binge drinking), and heart disease were recorded. Electrocardiography was performed in all patients, and those who were <50 years of age or without conventional vascular risk factors underwent transthoracic and transesophageal echocardiograms.

Because no patients showed findings of a fusiform aneurysm or a double lumen sign characteristic of vertebral dissection in angiogram or in the axial cross-sectional MRI images,[7,8] we arbitrarily defined the etiopathogenesis as follows.

Probable dissection was defined as (1) obvious history of recent (within 1 week) head/neck trauma or sudden neck rotation (chiropractic manipulation, golf practice, yoga, etc), (2) concurrent severe neck or occipital pain, (3) no evidence of atherosclerotic vascular changes on angiogram, and (4) angiographic findings of an elongated, usually tapered stenosis/occlusion in the involved artery[9,10]; possible dissection was defined as above except that there was no definite history of recent head/neck trauma/rotation.

Atherosclerosis (or atherothrombosis) was defined as present in patients (1) with at least one conventional risk factor for atherosclerosis, (2) with angiographic evidence of atherosclerotic vascular lesion, and (3) who do not fit the category of dissection.

Probable cardiogenic embolism was defined as the presence of concurrent emboligenic heart disease (atrial fibrillation, prosthetic valve, sick sinus syndrome, valvular disease, cardiomyopathy, recent myocardial infarction) without risk factors for atherothrombosis; possible embolism was defined as patent foramen ovale with right to left shunt without risk factors for atherothrombosis.

Small-vessel disease was defined as (1) presence of hypertension, (2) age >50 years, (3) no emboligenic heart disease, and (4) normal angiogram.

The patients' MRI findings were copied from the original film (T2-weighted axial image) by one of the authors (J.S.K.) who was blinded to the angiogram findings. According to the previous criteria,[4] the level of medulla shown in MRI was categorized as rostral, middle, and caudal. The size (area) of an infarct was measured with the Leica Q-500 MC image analyzer (Cambridge Ltd) and was presented as the cross-sectional area of infarction/whole medullary area at that segment ×100 (%). When there were two cuts of MRI demonstrating the lesion, the larger lesion was used for the analysis. All data regarding the average size of the infarction were expressed as mean±SD. Comparison of the size of the lesion among different subgroups (see below) was done by Wilcoxon rank sum tests, with the use of the SAS statistical package (version 6.0). Angiography results were schematically drawn by another author (J.H.L.) who was blinded to the MRI findings. They were also reviewed by a neuroradiologist (C.G.C.) who was unaware of the MRI findings.

Results

General Features

The demographic characteristics, risk factors, and clinical features of 34 patients are summarized in the Table. There were 26 men and 8 women aged from 28 to 73 years (mean, 50 years). The presumed pathogenetic mechanisms of infarction were atherothrombosis in 19 patients, arterial dissection in 8 (probable 5, possible 3), small-vessel disease in 1 (patient 32), and moyamoya disease in 1 (patient 26). Cardiogenic embolism was considered in 3 (probable 1, possible 2). Although 2 additional patients had patent foramen ovale with right to left shunt, they were included in the group of atherothrombosis because they had multiple risk factors and atherosclerotic changes on angiogram. One (patient 3) had decreased serum free protein S, and in 1 (patient 14) the etiopathogenesis was unclear. These 2 patients were considered to have an embolism of unknown source. Except for 1, all patients with arterial dissection were younger than 50 years.

The patients' major neurological symptoms/signs were vertigo/dizziness (88%), gait ataxia (88%), Horner's sign (88%), nystagmus (71%), nausea/vomiting (65%), dysphagia (62%), and hoarseness (41%). Sensory manifestations included crossed pattern (ipsilateral trigeminal–contralateral hemibody/limb) in 11, contralateral trigeminal pattern in 10, bilateral trigeminal pattern in 4, each of which probably was due to an involvement of the descending trigeminal tract, the ascending secondary trigeminal tract, and both tracts, respectively.[4] Isolated hemibody/limb sensory involvement and isolated trigeminal sensory changes were noted in 6 and 2 patients, respectively. One patient did not show any sensory abnormalities.

Angiographic Findings and MRI-Angiogram Correlation

The patients' MRI findings and angiographic results were combined and are presented in Figs 2, 4, 6, and 8. The majority of the patients had distal VA disease (stenosis or occlusion). Stenosis >50% was considered significant in this study. For further clarification, the length of the stenotic/occlusive segment was classified as "short" when the involved area was <2 cm and "long" when it was longer than 2 cm. In general, there was isolated PICA stenosis/occlusion in 8 patients (23.5%), isolated VA disease in 13 (38.2%), and involvement of both VA and PICA in 9 (26.5%). In 4 patients (11.8%) no angiographic abnormalities were seen.

Isolated PICA Disease

There were 8 patients with isolated PICA disease (4 stenosis, 4 occlusion; patients 1 through 8, Figs 1 and 2).

MRI Findings

In this group, the MRI lesion was generally small, thin (except patient 2), and located at various levels: caudal (n=2), middle (n=3), rostral (n=1), caudal and middle (n=1), and middle and rostral (n=1) medulla. It represented a small lesion involving the lateral caudal (patients 5 and 6)/dorsal middle-rostral (patients 5 and 7) medulla or a diagonal band–shaped lesion involving the posterolateral medulla (patients 1, 3, and 4). Cerebellar involvement (medial PICA territory) was seen in only 1 patient (patient 5).

Clinical Manifestations

The patients' symptoms were usually mild and often fragmentary. Sensation in the face was retained in patients 5 through 7, and patient 8 did not have sensory symptoms.

Presumed Pathogenesis

Six patients had atherothrombosis, and 1 had possible dissection. Patient 6 with possible dissection had a small aneurysm at

Clinical Features and Risk Factors of Patients

No/Sex/Age	V/D	NS	GA	N/V	DP	HS	HN	FP	Sensory Pattern	Risk Factors
1/M/59	+	+	++	+	+	−	+	+	Crossed	HT, DM
2/M/54	+	+	++	+	−	−	+	−	BT	HT, SM, AL
3/F/41	+	−	+	−	++	−	+	−	CT	Prot S def
4/M/44	+	+	+	+	+	+	+	−	CT	SM
5/M/39	+	+	++	+	−	−	+	−	Body/limb	HT, SM, AL
6/M/49	+	+	++	+	−	−	+	−	Body/limb	HT, AL
7/M/54	+	−	+	−	++	+	+	+	Body/limb	HT, DM, SM, AL
8/M/73	−	−	−	−	++	−	−	−	No	HT
9/F/57	+	+	++	+	−	−	+	−	Crossed	HT
10/F/48	+	+	++	+	+	+	+	−	Crossed	HT, DM
11/F/48	+	+	++	+	−	−	+	−	Crossed	HT
12/M/58	+	+	++	+	+	+	+	−	Crossed	HT, SM, AL
13/M/59	+	+	−	−	−	−	+	+	CT	HT, DM, AL
14/M/51	−	−	−	−	+	−	+	−	Body/limb	HT
15/F/62	+	+	+	−	++	−	+	−	Crossed	HT, DM, CHD
16/M/39	+	+	++	−	++	+	+	+	CT	HT, SM, AL
17/M/49	+	+	++	+	−	−	−	−	BT	HT, SM, PFO
18/M/42	+	+	++	−	++	+	+	+	BT	HT, SM
19/M/52	+	+	+	+	++	−	+	−	CT	HT, SM
20/F/30	+	−	++	+	−	−	−	−	Crossed	HT, PFO
21/M/49	+	+	+	+	+	+	+	−	CT	DM, AL
22/M/52	+	+	++	+	+	+	+	−	Crossed	HT, SM
23/M/55	+	−	+	+	++	+	+	+	BT	SM
24/M/42	+	+	++	+	++	+	+	+	CT	...
25/M/59	+	+	++	−	+	+	+	−	Crossed	HT, SM
26/F/57	+	+	++	+	−	−	+	−	Crossed	Moya moya
27/M/59	+	+	++	+	−	−	+	−	CT	HT, DM, SM
28/M/38	+	+	++	+	+	−	+	−	Body/limb	HT, SM
29/M/50	+	+	++	+	+	+	+	−	Crossed	SM, AL
30/M/39	+	+	++	+	+	+	+	−	CT	HT, SM, AL
31/M/46	+	−	+	+	−	−	+	−	CT	PFO
32/M/49	+	−	+	−	−	−	−	−	Face only	EHD
33/M/28	+	−	−	−	−	−	+	−	Face only	PFO
34/M/60	−	−	+	−	++	+	+	+	Body/limb	SM, AL

V/D indicates vertigo/dizziness; NS, nystagmus; GA, gait ataxia; N/V, nausea/vomiting; DP, dysphagia; HS, hoarseness; HN, Horner's sign; FP, facial paresis; +, present; ++, present to a severe degree[4]; −, absent; Crossed, crossed sensory pattern; BT, bilateral trigeminal pattern; CT, contralateral trigeminal pattern; HT, hypertension; DM, diabetes mellitus; SM, cigarette smoking; AL, habitual alcohol drinking; CHD, coronary heart disease; PFO, patent foramen ovale; and EHD, emboligenic heart disease.

the PICA-VA junction area. One did not have a stroke risk factor but had decreased serum protein S level and was categorized as having an embolism without obvious source.

Short-Segment VA Stenosis

Of the 9 patients with short-segment VA stenosis, there were 3 with and 6 without PICA occlusion (patients 9 through 17, Figs 3 and 4). The vascular lesion was located below (n=2) or above (n=3) the PICA origin. In 3 (patients 15 through 17), the lesion was at the PICA orifice and occluded the PICA. In 1 (patient 14), the PICA was considered to be absent congenitally because it was unidentifiable in both sides and the AICA was predominantly supplying the lower part of the cerebellum. In this patient the lesion appeared to be a small filling defect rather than a stenosis, suggesting an embolic disease, but we were unable to localize the embolic source.

MRI Findings

The lesions were variously located at the caudal (n=1), middle (n=1), rostral (n=1), caudal and middle (n=3), and middle and rostral (n=2) medulla. They were characterized by lateral-superficial lesions at the caudal medulla (patients 9, 12, and 16) or posterolateral lesions at the middle-rostral medulla. Generally, the proximal (lower) vascular lesion was correlated with a caudal medullary lesion (patients 9 and

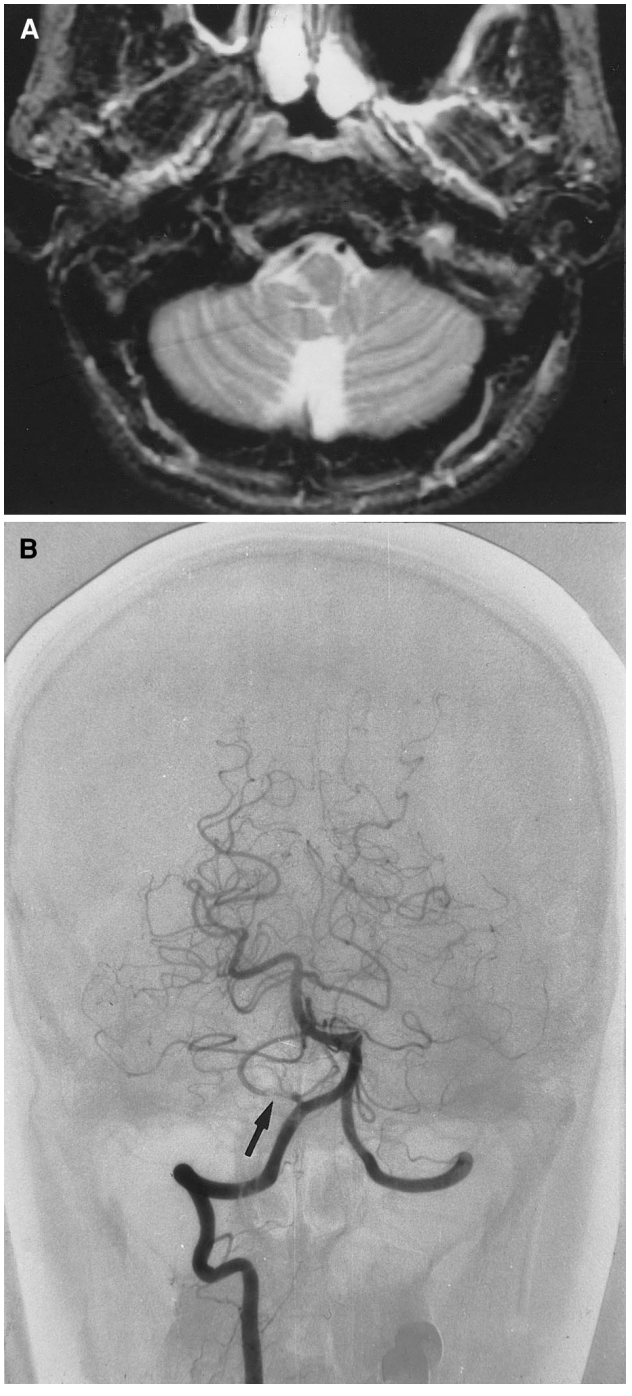

Figure 1. Patient 6. A, T2-weighted MRI shows an infarct in the right caudal medulla. B, Angiogram shows a focal stenosis in the proximal portion of the PICA (arrow) caused by possible dissection.

10), and distal vascular lesions tended to produce rostral lesions (patients 13 and 14; patient 12 was an exception). Cerebellar involvement was not seen in any patient.

Clinical Manifestations

The lesions tended to produce classic lateral medullary syndrome (for example, 5 patients had a classic crossed sensory pattern).

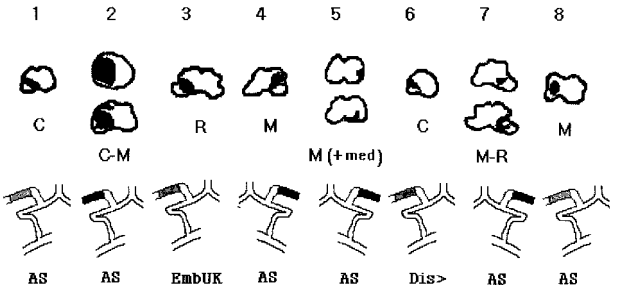

Figure 2. MRI and angiographic findings of patients with PICA stenosis/occlusion. The size of PICA was exaggerated for clear viewing. Dotted area indicates stenosis and dark area indicates occlusion. Number indicates patient number; C, caudal; M, middle; R, rostral; +med, cerebellar involvement of medial PICA territory; AS, atherosclerosis; EmbUK, embolism of unknown source; and Dis>, possible dissection.

Presumed Pathogenesis

Six patients had atherothrombosis, 2 had arterial dissection (1 probable and 1 possible), and 1 had an embolism of unknown source.

Long-Segment VA Disease Sparing the Proximal Portion

Long-segment VA disease sparing the portion proximal to the C1 segment of the VA was seen in 8 patients (2 stenosis, 6

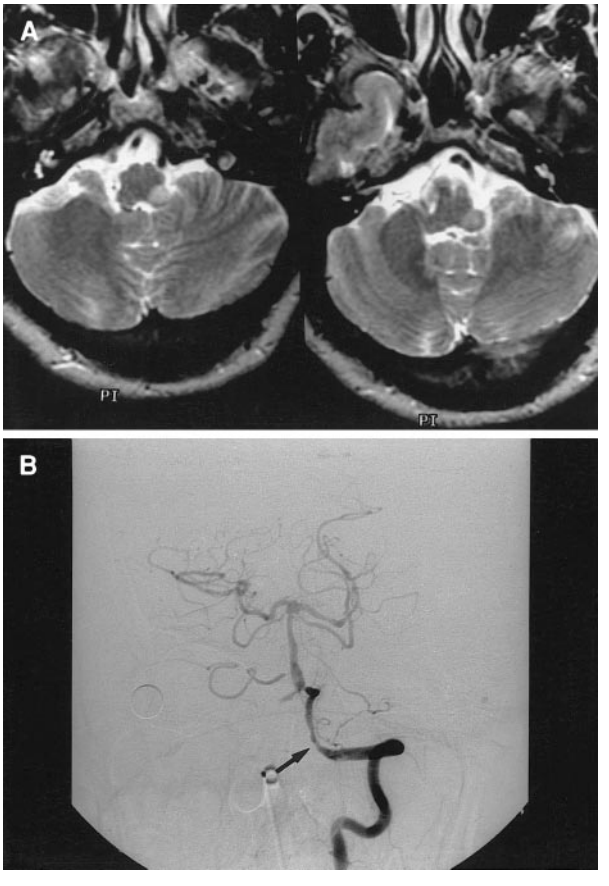

Figure 3. Patient 15. A, T2-weighted MRI shows an infarct in the left middle-rostral medulla. B, Angiogram shows a focal stenosis in the left VA (arrow) with nonvisualization of the PICA, which was probably caused by atherothrombosis. There also was significant stenosis in the proximal portion of the right AICA.

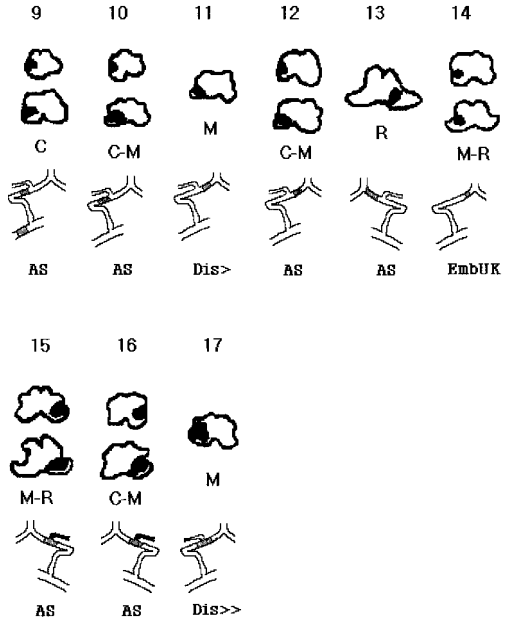

Figure 4. MRI and angiographic findings of patients with short-segment VA disease without (patients 9 through 14) and with (patients 15 through 17) PICA occlusion. Dis≫ indicates probable dissection; other abbreviations are defined in Fig 2.

occlusion; patients 18 through 25, Fig 5 and Fig 6, upper row). In this group, the long-segment distal VA was involved up to the vertebrobasilar junction or PICA orifice. In 3 patients (patients 23 through 25), PICA was occluded by the diseased VA, whereas in others (n=5) the PICA was spared. Among those patients, 3 (patients 20 through 22) had a high PICA origin (therefore the vascular lesion was located below the PICA origin), and 1 (patient 18) had a low PICA origin (the lesion was above the PICA).

MRI Findings

The lesion was located in the middle medulla in 4 patients, caudal and middle medulla in 3, and middle and rostral medulla in 1. One (patient 25) with concomitant PICA involvement had scattered infarcts in the cerebellum. Five patients (patients 18 through 20, 23, and 24) had large infarcts encompassing the posterolateral-ventromedial part of the medulla, whereas in 3 the lesions were relatively small.

Clinical Manifestations

This group was characterized by relatively frequent bilateral trigeminal (n=3) or contralateral trigeminal (n=2) sensory pattern. However, the 3 patients with small lesions showed a crossed sensory pattern.

Presumed Pathogenesis

Mechanisms included arterial dissection in 5 patients (probable dissection in 4, possible dissection in 1) and atherothrombosis in 3. Two of the 3 patients with atherothrombosis (patients 22 and 25) had lesions that were relatively small.

Long-Segment VA Occlusion Including the Proximal Portion

Long-segment VA occlusion including the portion proximal to the C1 segment was seen in 5 patients (patients 26 through 30,

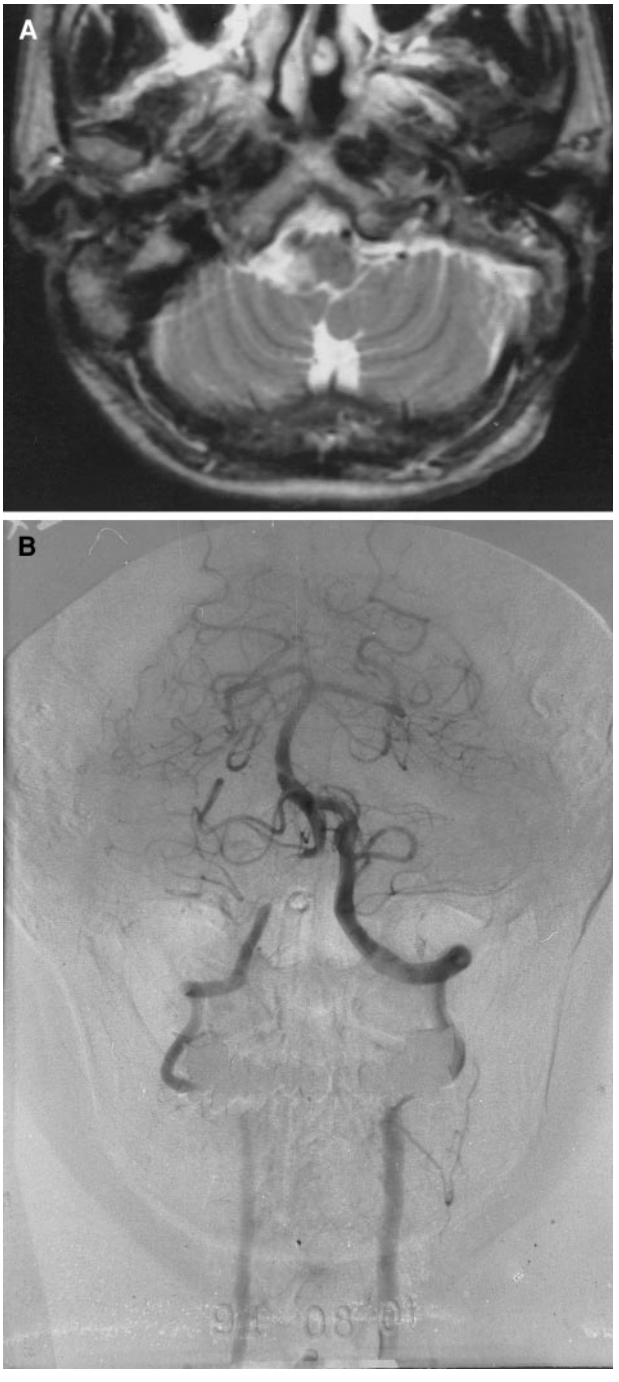

Figure 5. Patient 20. A, T2-weighted MRI shows an infarct in the right middle medulla. B, Angiogram shows a long-segment occlusion in the right VA that was probably caused by dissection.

Fig 7 and Fig 6, lower row). In this group, the vascular lesions ranged from the origin of the VA to the distal part of the vessel. In 4 patients, the lesion occluded the PICA, whereas in 1 the lesion ended just distal to the origin of the PICA.

MRI Findings

The lesions were located in the caudal medulla in 3 patients, caudal and middle medulla in 1, and middle and rostral medulla in 1. The infarct was generally small in size and variously located: lateral-superficially (patients 28 and 29) or posterolat-

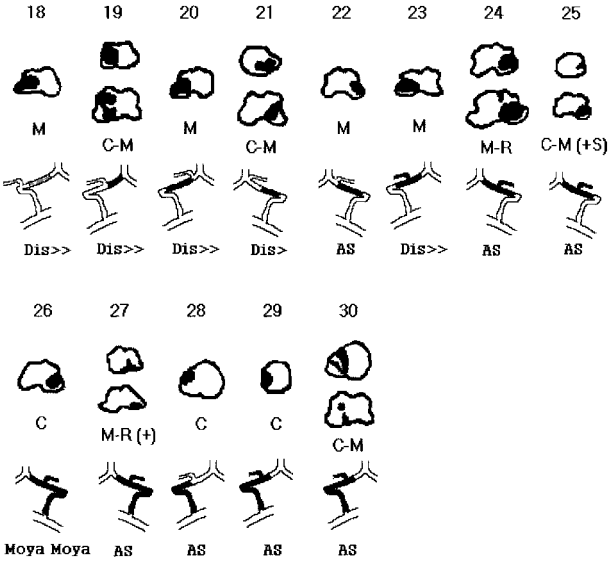

Figure 6. MRI and angiographic findings of patients with long-segment VA disease without (patients 18 through 25) and with (patients 26 through 30) proximal VA involvement. +S indicates scattered cerebellar infarction; +, cerebellar infarction with whole PICA territory; and Dis≫, probable dissection. Other abbreviations are defined in Fig 2.

erally in a diagonal-band shape (patients 26 and 30) at the caudal medulla. Lesions also involved the most dorsal part of the middle-rostral medulla in patients 27 and 30. Cerebellar involvement was seen in 1 patient (whole PICA territory).

Clinical Manifestations

The patients showed marked gait ataxia and mild dysphagia. The sensory pattern was heterogeneous.

Presumed Pathogenesis

Pathogenesis included atherothrombosis in 4 patients and moyamoya disease (complete occlusion of both distal carotid arteries with typical rete mirabile) in 1 patient (patient 26).

Normal Angiographic Findings

Angiographic findings were normal in 4 patients (Figs 8 and 9).

MRI Findings

The lesions were small in size, relatively round, and heterogeneously located. The cerebellum was spared in all.

Clinical Manifestations

There was a paucity of symptoms, and sensory signs were often fragmentary; 2 patients had ipsilateral sensory changes limited to the face.

Presumed Pathogenesis

The causes included cardiac embolism in 3 patients (probable 1, possible 2), and small-vessel infarction in 1 patient.

Size of Infarct in Each Subgroup

The average sizes of infarct in patients with PICA disease, short-segment VA disease, long-segment disease with proximal VA involvement, and normal angiogram were 14.14±8.95%, 15.78±7.11%, 10.63±6.05%, and 9.19±3.71%, respectively, and were not significantly different from each other. However,

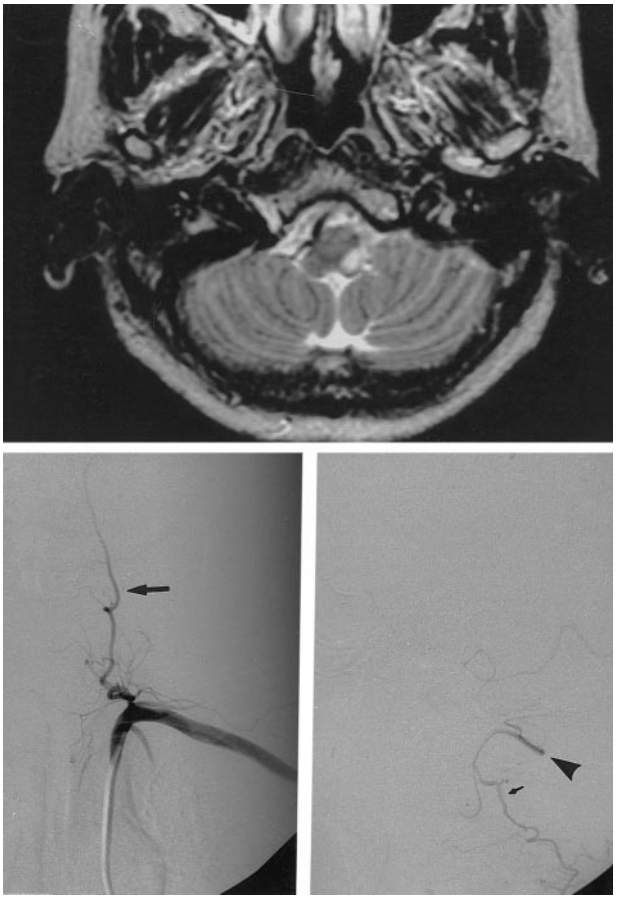

Figure 7. Patient 26. Top, T2-weighted MRI shows an infarct in the left middle medulla. Bottom, Angiogram shows nonvisualization of the left VA. Thyrocervical trunk (arrow) is shown with prominent collaterals (right panel). A part of the upper cervical portion of the VA (arrowhead) was faintly visualized through collaterals from the deep cervical branches (arrow), but the distal part of the VA was not visualized, suggesting occlusion (left panel). This patient had a bilateral distal internal carotid artery occlusion with rete mirabile consistent with moyamoya disease.

the size of the MRI lesion in patients with long-segment VA disease sparing the proximal VA (average size, 27.78±9.28%) was significantly larger than those of all other groups ($P<.05$, in all). In the patients with long-segment VA disease, we also attempted to compare the infarct size of the patients with dissection (patients 18 through 21 and 23) with that of the patients with atherosclerosis (patients 22 and 24 through 30). We found that the former was significantly larger than the latter ($P<.05$). In patients with short-segment VA disease, the average infarct size of the patients with concomitant occlusion of PICA was larger (23.83±2.74%) than in those without (average, 11.76±9.07%), which, however, was not significantly different on statistical analysis.

Discussion

Although various patterns of MRI lesions and vascular pathologies have been previously shown to be associated with LMI, how they are correlated with each other remains unexplored. Ours is the first attempt to analyze the location, shape, and size of the MRI-identified lesions with conventional angiographic findings in a relatively large number of patients. The results

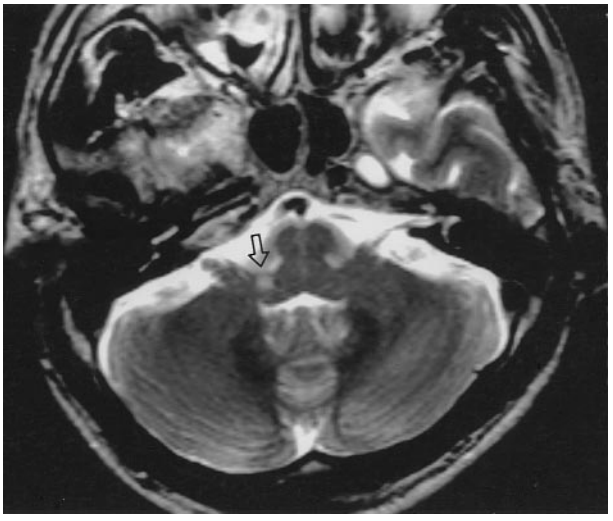

Figure 8. Patient 32. T2-weighted MRI shows a small infarct in the right rostral medulla (arrow) that was probably caused by cardiogenic embolism. Angiogram findings were normal in this patient.

provided basic insight as to how the heterogeneous vascular lesions are related with the various patterns of infarcts (and consequent clinical syndromes) occurring in the lateral medulla.

In this study, angiography demonstrated isolated PICA disease in 23.5% of patients, VA disease in 38.2%, involvement of both VA and PICA in 26.5%, and normal results in 11.8%. Previously, Fisher et al[11] reviewed the pathological findings of 42 patients with LMI (26 from the literature and 16 of their own) and found that PICA disease, VA disease, and involvement of both arteries were seen in 14.3%, 38.1%, and 26.2%, respectively. In 19% of these patients, occluded vessels were not found. Thus, our results were similar to those of Fisher et al, except that isolated PICA disease was more frequent and normal findings were less frequent. This may be attributed to selection bias; conventional angiography was likely to have been performed in patients who were expected to have a gross vascular lesion. This selection bias may also explain, at least in part, the younger average age of the patients (50 years) compared with patients with stroke in general in our hospital (62 years), as well as the relatively frequent arterial dissection in our series (24%) compared with the previous study (14% in the series of Vuilleumier et al[6]). Thus, the prevalence of each of the pathogenetic mechanisms for LMI shown in our study may not be generalizable.

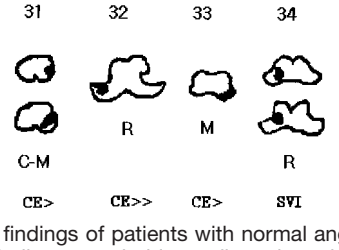

Figure 9. MRI findings of patients with normal angiographic results. CE≫ indicates probable cardiogenic embolism; CE>, possible cardiogenic embolism; and SVI, small-vessel infarction. Other abbreviations are defined in Fig 2.

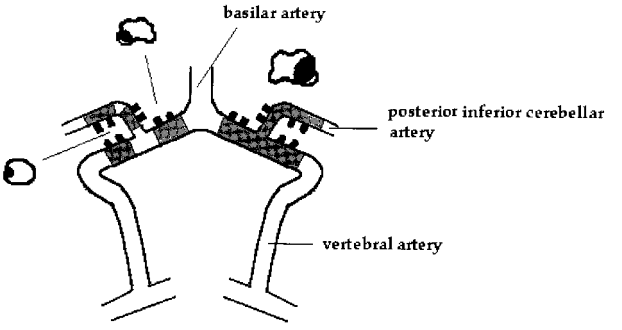

Figure 10. Schematic drawing of the vascular lesion (dotted area) and resultant MRI lesion.

We found that isolated PICA disease was associated with relatively small, thin lesions in the lateral caudal and/or dorsolateral middle-rostral portion of the medulla. The lesion was located at various rostral-caudal levels of the medulla, which may result from various levels of PICA origin at the VA,[11] various lengths of the ascending loop of PICA,[12] different degree of collateralization from other vessels such as the VA or the AICA,[11] and different levels of the occlusion of penetrating branches from the PICA (Fig 10). The most dorsal part of the caudal medulla was usually spared. This area (supplied by the posterior spinal artery, a relatively distal branch of the PICA[13]) may easily be spared, probably because atherosclerotic changes are generally more severe in the proximal than the distal part of the PICA. Regardless of the explanation, the heterogeneous lesion location is consistent with heterogeneous clinical manifestations (such as sensory pattern), but the clinical symptoms were generally mild, probably because of the thinness of the lesions. The fact that the dorsal part of the caudal medulla, in which the descending trigeminal tract/nuclei are located,[13] is frequently spared may explain the absence of trigeminal sensory symptoms in patients 5 through 8. Our data illustrate that PICA territory is not strictly confined to a certain part of the medulla, as suggested by previous authors.[6,13] Furthermore, cerebellar involvement was noted in only 1 of 8 patients with isolated PICA disease, illustrating the effectiveness of the collateral circulation in the cerebellum through the AICA or the superior cerebellar artery.[12]

In our study, VA stenosis or occlusion was the most common angiographic feature. Short-segment VA stenosis tended to produce a medium-sized, diagonal band–shaped lesion usually confined to the dorsolateral part of the rostral-middle medulla or lateral superficial portion of the caudal medulla. It seems that the level of stenosis at the VA determines the rostrocaudal level of the MRI lesion. The lesion usually produced classic lateral medullary syndrome with a crossed sensory pattern. The MRI lesion of short-segment VA disease does not appear to be distinctly different from that produced by PICA disease in its morphology and size, suggesting that territories supplied by branches from the PICA and VA frequently overlap (Fig 10). Furthermore, although statistical significance was not reached, the average size of the infarct was larger in patients with concomitant PICA occlusion than that in those with spared PICA, which may reflect an involvement

of both arteries leading to a relatively large lesion due to poor collateral circulation.

Long-segment VA diseases were divided into those with sparing of the proximal part of the VA and those without. According to the MRI–vascular lesion correlation, however, it seems more reasonable to divide VA disease into that producing a large MRI lesion and that associated with a small lesion. The long-segment VA disease associated with a large lesion involving the whole dorsolateral-ventromedial part of the medulla was most often caused by dissection. The large lesion was probably caused by simultaneous occlusion of multiple branches of the VA with or without PICA involvement (Fig 10). The clinical hallmark of the large MRI lesions was bilateral or contralateral trigeminal sensory involvement that was caused by concomitant involvement of the medial-ventrally located ascending secondary trigeminal fibers.[4] On the other hand, patients with atherothrombotic disease had small MRI lesions despite extensive vascular disease, which may be attributed to previously established collateralization in patients with slowly progressive atherothrombosis or moyamoya disease (Fig 7). The MRI lesion in these patients was similar in shape and size to that produced by isolated PICA disease, suggesting that the lateral medulla supplied by the medial PICA may have poor collateral circulation.[12] Thus, it seems that not only the length of the involved VA segment but also the speed of development of the vascular lesion determine the eventual size of the infarct and consequent clinical syndromes.

Finally, patients with normal angiogram frequently had a cardiac source of embolism, and the embolic occlusion appears to have been recanalized at the time of angiography. The successful recanalization may have resulted in a small infarct restricted to the most vulnerable area, producing fragmentary symptoms in this group. Small dorsal lesions affecting a limited area produced isolated facial sensory changes in patients 32 and 33 by selective involvement of the descending trigeminal tract/nuclei (Fig 8). According to previous authors[6] and our own experiences, patients with cerebellar infarction may exhibit symptoms of LMI without visible lesion in the medulla. These cases were omitted in our series because we considered only the patients with MRI-identified medullary lesions. Thus, the prevalence of cardiogenic embolism may have been underestimated in our study.

In summary, our study illustrates that LMI is a heterogeneous condition associated with various MRI lesions and diverse vascular pathologies. Isolated PICA disease usually produces thin lesions at various rostrocaudal levels leading to mild symptoms, and short-segment VA disease is associated with classic diagonal band–shaped lesions confined to the lateral-posterior medulla leading to classic symptom complexes. Both are associated with atherothrombotic vascular disease. Long-segment VA disease is associated with either large MRI lesions or lesions mimicking isolated PICA disease that are most often related to dissection and atherothrombosis, respectively. Patients with normal angiogram often had emboligenic cardiac disease and had relatively small lesions producing fragmentary symptoms. These principles may be much too simplified to account for the complexity of LMI and are indeed open to further verification through analysis of more cases.

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References

1. Ross MA, Biller J, Adams HP, Dunn V. Magnetic resonance imaging in Wallenberg's lateral medullary syndrome. *Stroke*. 1986;17:542–545.
2. Bogousslavsky J, Fox AJ, Barnett HJM, Hachinski VC, Vinitski S, Carey LS. Clinico-topographic correlation of small vertebrobasilar infarct using magnetic resonance imaging. *Stroke*. 1986;17:929–938.
3. Sacco RL, Freddo L, Bello JA, Odel JG, Onesti ST, Mohr JP. Wallenberg's lateral medullary syndrome: clinical-magnetic resonance imaging correlations. *Arch Neurol*. 1993;50:609–614.
4. Kim JS, Lee JH, Suh DC, Lee MC. Spectrum of lateral medullary syndrome: correlation between clinical findings and magnetic resonance imaging in 33 subjects. *Stroke*. 1994;25:1405–1410.
5. Kim JS, Kim HG, Chung CS. Medial medullary syndrome: report of 18 new patients and a review of the literature. *Stroke*. 1995;26:1548–1552.
6. Vuilleumier P, Bogousslavsky J, Barth F. Infarction of the lower brainstem: clinical, aetiological and MRI-topographical correlations. *Brain*. 1995;118: 1013–1026.
7. Shimoji T, Bando K, Nakajima K, Ito K. Dissecting aneurysm of the vertebral artery: report of seven cases and angiographic findings. *J Neurosurg*. 1984;61:1038–1046.
8. Yamaura A, Watanabe Y, Saeki N. Dissecting aneurysms of the intracranial vertebral artery. *J Neurosurg*. 1990;72:183–188.
9. Hart RG. Vertebral artery dissection. *Neurology*. 1988;38:987–989.
10. Mokri B, Houser W, Sandok BA, Piepgras DG. Spontaneous dissections of the vertebral arteries. *Neurology*. 1988;38:880–885.
11. Fisher CM, Karnes WE, Kubik CS. Lateral medullary infarction: the pattern of vascular occlusion. *J Neuropathol Exp Neurol*. 1961;20:323–379.
12. Goodhart SP, Davison C. Syndrome of the posterior inferior and anterior inferior cerebellar arteries and their branches. *Arch Neurol Psychiatry*. 1936; 35:501–524.
13. Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of human brain: brainstem and cerebellum. *Neurology*. 1996;47:1125–1135.