Spectrum of Lateral Medullary Syndrome
Correlation Between Clinical Findings and Magnetic Resonance Imaging in 33 Subjects

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Background and Purpose
Computed tomography is insufficient in evaluation of medullary lesions. Although lateral medullary infarction is a relatively common type of cerebrovascular disease, detailed correlation between clinical findings and magnetic resonance imaging (MRI) has not yet been reported.

Methods
We studied 33 consecutive patients with lateral medullary infarction who showed appropriate MRI lesions and correlated their clinical findings with the MRI results.

Results
Gait ataxia (88%), vertigo/dizziness (91%), nausea/vomiting (73%), dysphagia (61%), hoarseness (55%), Horner sign (73%), and facial (85%) and hemibody (94%) sensory changes were frequent clinical findings. MRI results showed that the lesions located in the rostral part of the medulla were usually diagonal band-shaped and were associated with more severe dysphagia, hoarseness, and the presence of facial paresis, whereas the caudal lesions, situated usually in the lateral surface of the medulla, appeared to correlate with more marked vertigo, nystagmus, and gait ataxia. Nausea/vomiting and Horner sign were common regardless of the lesion location, and lesions involving more mediodorsally correlated with facial sensory change on the contralateral side of the lesion.

Conclusions
Analysis of MRI findings in rostrocaudal and dorsoventral aspects allows us, although not unequivocally, to make anatomicoclinical correlations in the evaluation of patients with lateral medullary stroke syndrome. (Stroke. 1994; 25:1405-1410.)

Key Words: cerebral infarction • lateral medullary syndrome • magnetic resonance imaging • stroke assessment

Computed tomography (CT) usually fails to identify medullary vascular lesions. With the advent of magnetic resonance imaging (MRI), brain stem ischemic strokes can be more definitively evaluated.1,2 Ross et al3 found MRI-identified lesions in their 4 patients with lateral medullary syndrome (LMS), and Bogousslavsky et al4 described 6 patients who had small vertebrobasilar territory infarcts with good clinical-MRI correlation. Recently Sacco et al5 analyzed 33 patients with LMS and described the MRI findings as abnormal in 20 of 22 patients in whom MRI was performed. However, they failed to correlate the diverse clinical manifestations with MRI findings, and a detailed clinical-MRI correlation study remains to be reported. In the present study we describe 33 patients with LMS in whom MRI showed appropriate lesions and attempt to correlate their clinical findings with the results of MRI.

Subjects and Methods
At Asan Medical Center (Seoul, South Korea), MRI scan was performed in 37 patients with clinically suspected LMS from June 1990 to December 1993. In this anatomicoclinical correlation study, 4 patients were excluded: 2 with equivocal lesions, 1 with additional pontine lesions, and 1 with bilateral medullary lesions. The remaining 33 patients showed unilateral lesions mainly involving the dorsolateral portion of the medulla oblongata. Some patients with lesions extending beyond the dorsolateral portion were included. The 33 patients comprised 22 men and 11 women (age range, 36 to 71 years [mean, 59 years]). Risk factors for stroke included hypertension in 24, diabetes mellitus in 8, cigarette smoking in 8, and cardiac disease in 3. No significant risk factors were identified in 3 patients. All patients except 2 were examined within 5 days after the onset of stroke. Their main clinical symptoms and signs were evaluated as follows: vertigo/dizziness: — (absent), + (present); nystagmus: — (absent), + (present) on extreme gaze, ++ (present on forward gaze); gait ataxia: — (absent), + (present but able to walk), ++ (unable to walk); nausea/vomiting: — (absent), + (present); dysphagia: — (absent), + (mildly present), ++ (needs nasogastric tube for feeding); hoarseness: — (absent), + (present); Horner sign: — (absent), + (present); facial palsy: — (absent), + (present); hemiparesis: — (absent), + (present); and sensory dysfunction (depicted in Fig 1).

MRI studies were performed using a 1.5-T superconducting magnet (GE). Axial T2 (repetition time [TR], 2500 milliseconds; echo time [TE], 80 milliseconds) scan was performed in horizontal plane at 5- or 6-mm intervals from the medulla to the midbrain. T1-weighted (TR, 600 milliseconds; TE, 20 milliseconds) axial and sagittal images were also obtained. Coronal sections were done in two cases. Evaluation of the lesions generally depended on T1-weighted axial cuts of the medulla, which are imaged at three different levels: the upper medulla, characterized by posterior lateral bulging of the restiform body (Fig 2, top left panel); the middle medulla, characterized by nodular lateral surface due to the olivary nucleus (Fig 2, top right panel); and the lower medulla, characterized by a relatively round figure with closed fourth ventricle (Fig 2, bottom left panel). The patients’ MRI findings were evaluated in rostrocaudal and dorsoventral aspects.

Results
Neurological Symptoms and Signs
The patients’ neurological symptoms and signs are summarized in Fig 1. Vertigo/dizziness (30 [91%]), gait
FIG 1. Chart showing clinical and radiological features of 33 patients. RF indicates risk factors; V/D, vertigo/dizziness; NS, nystagmus; GA, gait ataxia; N/V, nausea/vomiting; DS, dysphagia; HS, hoarseness; HN, Horner sign; FP, facial paresis; HE, hemiparesis; SEN, sensory abnormality; MRI, magnetic resonance imaging; Cbll, cerebellar lesion; Angio, angiographic vascular lesion; HT, hypertension; SM, cigarette smoking; CD, cardiac disease; DM, diabetes mellitus; HL, hyperlipidemia; +, present (see "Subjects and Methods" for more specific definitions); -, absent; C, contralateral to lesion; I, ipsilateral to lesion; U, upper; cl, classic; Ig, large; vm, ventromedial; an, anterior; pl, posterolateral; M, middle; Is, lateral-superficial; hs, hemi; L, lower; and np, not performed.
ataxia (29 [88%]), nausea/vomiting (24 [73%]), nystagmus (22 [67%]), Horner sign (24 [73%]), dysphagia (20 [61%]), hoarseness (18 [55%]), and facial (28 [85%]) and hemibody (31 [94%]) sensory changes were frequent clinical manifestations. Mild facial paresis of the central type on the side ipsilateral to the lesion was seen in 12 patients (36%), and 4 had mild hemiparesis, 1 on the side ipsilateral to the lesion. Although facial sensory change ipsilateral to the lesion was usual, 9 patients showed facial sensory dysfunction on the side contralateral to the lesion, and 1 had bilateral facial sensory abnormalities. In 4 patients facial sensory change was restricted to the ophthalmic division of trigeminal (V1) area, and in 2 there was no facial sensory change. In 2 patients there was no definite sensory dysfunction in either the face or the body.

Imaging and Vascular Studies

Eight patients had lesions in the upper medulla, 8 in the middle medulla, and 9 in the lower medulla. Four had lesions in the upper and middle medulla, and 4 in the middle and lower medulla. The lesions in the upper and the middle medulla were usually diagonal band-shaped and situated in the posterolateral portion of the medulla. Diagonal band-shaped lesions sparing the most posterolateral portion were most common and therefore classified as the classic type (Fig 3, top panel). Diagonal band-shaped lesions situated more ventromedially were classified as the ventromedial type (Fig 3, top panel), which was defined when the center of the lesion was located inside the medial half of a line drawn as in Fig 3, top panel. Lesions encompassing the most posterolateral surface of the medulla were designated as posterolateral. However, a few lesions were large enough to encompass the ventromedial and posterolateral part of the medulla and were designated as large. In some patients (usually those with lower medullary lesions), the lesions were located in the lateral surface of the medulla and were classified as the lateral-superficial type (Fig 3, bottom panel). In a few patients, additional or extended lesions were seen in the anterior half of the medulla; these were classified as anterior and hemi types, respectively. This classification was made by one of the authors, who was blind to the patients' clinical findings.

Twelve patients showed lesions in the upper medulla (4 of them had concomitant middle medullary lesions). Generally, the lesions in the upper medulla were thick and diagonal band-shaped. They were classified as classic in 4, classic and anterior in 1, ventromedial in 2, large in 3, and posterolateral in 2. Sixteen had lesions in the middle medulla (4 had lesions in the upper medulla and 4 had lesions in the lower medulla concomitantly). Lesions were classified as classic in 4, ventromedial in 1, posterolateral in 6, posterolateral and anterior in 1, hemi in 1, and lateral-superficial in 3. Thirteen had lesions in the lower medulla. The lesions were most often located superficially: lateral-superficial in 8, posterolateral in 1, posterolateral and anterior in 1, classic in 1, and ventromedial in 2.

In addition to the medullary lesions, 7 patients showed infarcts in the cerebellum. Fourteen had CT scan before MRI examination, which universally failed to localize medullary lesions but detected concomitant cerebellar infarcts in 3 patients. Seven patients had angiography, which showed vascular abnormalities in 4
patients: vertebral artery stenosis or occlusion in 3 and severe basilar artery stenosis in 1.

Clinical-MRI Correlation

To elucidate the clinical difference between rostral and caudal lesions, we divided the patients into three groups according to rostrocaudal aspect of the lesion identified by MRI.

Rostral Group

This group included patients with lesions in the upper medulla or both upper and middle medulla (patients 1 through 12; n=12). All patients in the rostral group had dysphagia and needed nasogastric tube insertion for feeding, and 5 of them had to use it for more than 3 months. All patients exhibited hoarseness. Six patients had no nystagmus, and only 1 had nystagmus on forward gaze. Gait ataxia was present in 11 and was severe in 8. Horner sign was present in 10 patients, and ipsilateral facial paresis was observed in 10. Ten patients had vertigo/dizziness; 8 had nausea/vomiting. Four patients (2 ventromedial, 2 large) showed facial sensory change on the side contralateral to the lesion. One (patient 11, classified as large) showed bilateral facial sensory dysfunction.

Caudal Group

This group included patients with lesions in the lower medulla or lower and middle medulla (patients 21 through 33; n=13). The patients tended to have lesions in the superficial area. No patients had dysphagia or hoarseness, except for 2 (patients 21 and 23) with concomitant middle medullary lesion who reported transient dysphagia. Vertigo and dizziness were noted by all and were usually severe. Gait ataxia, detected in 12 patients, was severe in 11. Six showed nystagmus on forward gaze. Nine had nausea/vomiting, and 9 showed Horner sign. Facial paresis was observed in 1 patient. Two (1 ventromedial, 1 posterolateral-anterior) showed facial sensory change on the side contralateral to the lesion.

Middle Group

This group included patients with middle medullary lesions (patients 13 through 20; n=8). This group exhibited intermediate characteristics. Six had dysphagia, but only 2 needed a nasogastric tube. Six had hoarseness, 7 had vertigo/dizziness, and 7 reported nausea/vomiting. Horner sign and facial paresis were seen in 5 and 1, respectively. Three patients (1 ventromedial, 1 hemi, and 1 classic) had facial sensory change on the side contralateral to the lesion.

Discussion

The clinical characteristics of our patients are generally similar to those described in recent clinical studies of
LMS. In our study we attempted to correlate the clinical manifestations with MRI results. Although it is difficult to classify the various lesions satisfactorily, we attempted to divide the cases with reference to the rostrocaudal aspects to elucidate possible clinical difference.

The most distinguishing rostrocaudal symptomatic difference was dysphagia, which was distinctly more severe in the rostral group than in the caudal group. Patients with midline medullary lesions showed an intermediate degree of severity. The different degrees of severity of dysphagia may be explained in several ways. Dysphagia in medullary stroke is caused by involvement of the nucleus ambiguous, a vertical columnar structure extending to the level of pyramidal decussation. This structure, seen in the middle portion of the medulla (Fig 3), may have been more frequently involved in the rostral group because lesions in this group were generally thick and tended to involve the ventral portion of the medulla. The lesions of the caudal group usually involved the superficial area and may have spared the more medially located nucleus ambiguous. However, patients with deep lesions in the caudal group (patients 27 and 32) also showed no dysphagia. Possibly only a caudal part of the nucleus ambiguous, a portion not directly related with visceral efferent fibers, may have been involved in this group. Whatever the actual explanation, caudal group patients have a more benign prognosis in terms of aspiration. As expected, hoarseness, another symptom related to nucleus ambiguous involvement, was also more marked in the rostral group than in the caudal group.

Nystagmus, gait ataxia, and vertigo/dizziness were apparently more severe in patients in the caudal group, although the latter symptoms were not objectively graded in this study. Nystagmus in LMS is attributed to involvement of the vestibular nuclei or their connections to the cerebellum, which begin at the caudal part of the inferior cerebellar peduncle, in the posterolateral medulla. A focal lesion in this area was reported to cause severe vestibulococular symptoms. The vestibulocerebellar pathway runs through the juxtarestiform body, a part of the inferior cerebellar peduncle, in the posterolateral medulla. These areas tended to be spared in the patients with upper medullary lesions, among whom 6 of 8 did not show nystagmus. Gait ataxia, a very common sign in our series, is attributed to involvement of either the restiform body or the spinocerebellar pathway. In the lower medulla, both of these structures are located in the lateral surface, which may have been frequently involved in patients in the caudal group (Fig 3, bottom panel). The involvement of the cerebellum, detected in 7 patients, does not appear to augment the severity of gait ataxia.

The neural substrate for nausea and vomiting, common symptoms of LMS, is not clearly defined. Whereas Peterman and Siekert suggested that nausea and vomiting are attributed to lesions of the vestibular nuclei, Currier et al stated that these symptoms are related to involvement of the medullary vomiting center, which may be identical to the nucleus ambiguous. In our series symptoms of nausea/vomiting were not specifically associated with the presence of dysphagia or nystagmus. The neural substrate for these symptoms may be related to both of the above structures. Horner syndrome, a sign related to the descending sympathetic pathways in the lateral reticular formation, is also common in our series regardless of lesion location.

Although dissociated sensory abnormality seen in classic LMS is usual in our series, 9 patients showed facial sensory change on the side contralateral to the lesion. All had lesions involving the ventromedial or anterior part of the medulla (ventromedial in 4, large in 2, hemi in 1, classic in 1, and posterolateral-anterior in 1), which would involve the ascending trigeminal sensory tract located in the medial-ventral portion of the medulla. (Fig 3, top panel). One patient (patient 11) had bilateral facial sensory change, probably because of a wide lesion involving both the descending and ascending trigeminal sensory tract. Two patients (patients 19 and 25) did not reveal any sensory abnormalities, probably because of sparing of the spinothalamic and trigeminal pathways. Three (patients 28, 30, and 31) showed hemihypesthesia without facial sensory change. Facial sensation was spared in 7 of 39 patients in the study of Currier et al, who suggested that the descending trigeminal sensory tract may be spared in ventrally situated lesions. Our patients, however, had lateral-superficial lesions in the lower medulla, suggesting that the descending trigeminal tract located posteromedially (Fig 3, bottom panel) may have been spared. Four of our patients had facial sensory change restricted to the V1 area: 2 in the side ipsilateral and 2 in the side contralateral to the lesions. Because the V1 area is located most ventrally in the descending trigeminal tract, Currier et al thought that this sensory pattern may be associated with a ventrally located lesion. However, the patients with ventromedial-type lesions did not reveal this type of sensory change in our series, and the mechanism of restricted facial sensory deficit in patients 7 and 17 remains unclear. Restricted sensory change of the V1 area on the side contralateral to the lesion (patients 29 and 32) may be due to selective involvement of the lateral part of the ascending trigeminal tract, where the sensory fibers from the upper part of the face are located.

Facial paresis ipsilateral to the lesion was observed in 12 patients (36%) in our series. Fisher and Tapia reported an autopsy-proven case of LMS associated with a severe peripheral type of facial paralysis, which was caused by extension of the lesion to the lower pons, involving intra-axial facial nerve fascicles. In our series no patients showed obvious lesions in the pons, and all had a mild, usually transient facial paresis of the central type. Currier et al and Sacco et al noted weakness of the facial muscles in 51% and 42% of their patients, respectively, and suggested that aberrant, looping corticobulbar fibers may have been involved in these patients. Most of our patients with facial paresis had lesions in the rostral medulla, suggesting that the aberrant fibers, if they exist, may not descend to the level of the lower medulla, although 1 patient (patient 27) with a caudal lesion of the ventromedial type also showed facial paresis. Finally, 4 of our patients showed mild hemiparesis. All except 1 had lesions extending anteriorly, suggesting that the corticospinal tract was involved. Of the 4, 1 (patient 29) showed hemiparesis on the side ipsilateral to the lesion. In this patient a coronal cut of the MRI showed that the lesion extended into the upper cervical cord (Fig 4), which probably involved the pyramidal tract after decussation.
In conclusion, our study illustrates that the clinical and toponographic spectra of LMS are diverse, and MRI analysis in rostrocaudal and dorsoventral aspects allows us, although not unequivocally, to make clinical-MRI correlations. Generally, the rostral lesions are diagonal band-shaped and correlate with severe dysphagia, hoarseness, and the presence of facial paresis, whereas caudal lesions, usually involving lateral superficial areas, appear to correlate with more marked nystagmus, vertigo, and gait ataxia. Nausea/vomiting and Horner sign are common regardless of the lesion location. Lesions extending ventromedially correlate with contralateral facial sensory change, whereas anteriorly located lesions are associated with hemiparesis.

References
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