Diffusion-Weighted MRI in Acute Lacunar Syndromes
A Clinical-Radiological Correlation Study

Wouter J. Schonewille, MD; Stanley Tuhrim, MD; Michael B. Singer, MD; Scott W. Atlas, MD

Background and Purpose—Clinical-radiological correlation studies in lacunar syndromes have been handicapped by the low sensitivity of CT and standard MRI for acute small-vessel infarction and their difficulty in differentiating between acute and chronic lesions.

Methods—We prospectively studied 43 patients presenting with a classic lacunar syndrome using diffusion-weighted MRI, a technique with a high sensitivity and specificity for acute small-vessel infarction.

Results—All patients were scanned within 6 days of stroke onset. An acute infarction was identified in all patients. Pure motor stroke was associated with lesions in the posterior limb of the internal capsule (PLIC), pons, corona radiata, and medial medulla; ataxic hemiparesis with lesions in the PLIC, corona radiata, pons, and insular cortex; sensorimotor stroke with lesions in the PLIC and lateral medulla; dysarthria–clumsy hand syndrome with lesions in the PLIC and caudate nucleus; and pure sensory stroke with a lesion in the thalamus. Supratentorial lesions extended into neighboring anatomic structures in 48% of the patients.

Conclusions—Lacunar syndromes can be caused by lesions in a variety of locations, and specific locations can cause a variety of lacunar syndromes. Extension of lesions into neighboring structures in patients with lacunar syndromes appears to be more frequent than previously described in studies using CT and standard MRI. (Stroke. 1999;30:2066-2069.)

Key Words: diffusion ■ lacunar infarction ■ magnetic resonance imaging ■ stroke, acute

The diagnosis of small-vessel or lacunar infarction was traditionally based on clinical presentation. Autopsy studies provided a basis for clinical-pathological correlation but were limited in a condition with such low mortality.

Recent studies have shown diffusion-weighted MRI (DW MRI) to be a highly sensitive and specific diagnostic technique for acute small-vessel infarction. The purpose of this study was to evaluate the clinical-radiological correlation in patients presenting with a classic lacunar syndrome with the use of DW MRI.

Subjects and Methods
From August 1, 1996, to June 30, 1997, we prospectively obtained conventional fast spin-echo imaging and DW MRI in 43 patients presenting with the acute onset of 1 of 5 classic lacunar syndromes: pure motor stroke, sensorimotor stroke, ataxic hemiparesis, dysarthria–clumsy hand syndrome, or pure sensory stroke. The procedures followed were in accordance with institutional guidelines; all patients consented to participation. All patients were examined by a stroke neurologist within 5 days of symptom onset and within 24 hours of presentation to the hospital and scanned within 6 days (mean, 53.3 hours) of symptom onset. We used a 1.5-T GE Signa Horizon MR scanner modified with hardware for echoplanar imaging. Multislice single-shot, spin-echo echoplanar diffusion imaging (δ=31 ms, Δ=36.6 ms, repetition time/echo time=10 000/99 ms) was performed with diffusion sensitivity b=1000 s/mm². The diffusion gradients were applied sequentially in 3 orthogonal directions to generate 3 sets of axial DW MR images. All images were obtained with 5-mm-thick sections with 2.5-mm interslice gaps and a 24-cm field of view for all scans. Interpretations were made with the use of all 3 sets of DW images. The DW MR images were read by 2 experienced neuroradiologists (M.B.S., S.W.A.), who were unaware of the patient’s neurological findings but knew the patient had suffered an acute stroke. Criteria for the diagnosis of acute infarction on DW MRI included focal high intensity on at least 1 diffusion-weighted image, judged not to be due to normal anisotropic diffusion or magnetic susceptibility artifact. In all cases the precise neuroanatomic location of such lesions was noted. All judgments were made by consensus of the 2 readers.

Following Fisher, pure motor stroke was defined as the presence of weakness in face, arm, and leg (with involvement of at least 2 body parts) in the absence of objective sensory deficit, ataxia out of proportion to weakness, diplopia, nystagmus, or persistent vertigo. If objective sensory findings were present in addition to weakness in the face, arm, and leg, the patient was categorized as having a sensorimotor stroke. Ataxic hemiparesis was defined as the presence of ataxia out of proportion to weakness in face, arm, and leg (with involvement of at least 2 body parts) in the absence of objective sensory deficit, diplopia, nystagmus, or persistent vertigo. If the ataxia was limited to the hand in addition to a prominent dysarthria, with or without facial

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From the Department of Neurology, Clinica Rotger, Palma de Mallorca, Spain (W.J.S.); and the Departments of Neurology (S.T.) and Neuroradiology (M.B.S., S.W.A.), Mount Sinai Hospital, New York, NY.
Correspondence to Stanley Tuhrim, MD, Mount Sinai School of Medicine, Department of Neurology, Box 1137, One Gustave L. Levy Place, New York, NY 10029. E-mail S_Tuhrim@mssm.edu
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In the 43 study patients who presented with the acute onset of a classic lacunar syndrome, pure motor stroke was the most frequent lacunar syndrome seen (n=20, 47%) followed by ataxic hemiparesis (n=11, 26%) and sensorimotor stroke (n=9, 21%). Two patients presented with the dysarthria–clumsy hand syndrome (5%) and 1 with a pure sensory stroke (2%). DW MRI identified an acute lesion consistent with infarction, without detailing the clinical-anatomic correlation.1,2 Standard MRI is handicapped by its difficulty in differentiating between acute and chronic lesions. Hommel showed approximately equal involvement in face, arm, and leg (1 lesion in the corona radiata and 1 in the PLIC), and 1 patient had more pronounced weakness in the leg (lesion in the PLIC and putamen). Two patients had sparing of the face (1 lesion in the PLIC extending to the corona radiata and 1 lesion limited to the PLIC), and 1 patient had sparing of the leg (pontine lesion). Six patients had accompanying symptoms. Two patients, 1 with a lesion in the PLIC that also involved the lentiform nucleus and another with a lesion in the pons, had a subjective change in sensation of face, arm, and leg but no objective sensory findings. Two patients, 1 with a lesion of the PLIC that also involved the genu and corona radiata and another with a lesion in the pons, had transient vertigo (described as a spinning sensation) at stroke onset. One patient with a lesion in the PLIC complained of dizziness (described as lightheadedness) at stroke onset associated with transient, unilateral perioral numbness. Dysarthria was present in 15 patients. The 5 patients without dysarthria had lesions in the PLIC, corona radiata (n=2), and pons (n=2).

Patients with ataxic hemiparesis (n=11) had lesions in the PLIC in 6 of 11 cases. Lesions were limited to the PLIC in 3 cases. One also involved the corona radiata, 1 the thalamus, and 1 the cerebral peduncle. Two patients had pontine lesions. One patient had a lesion limited to the corona radiata, another had a putaminal lesion, and 1 patient had a small isolated lesion in the insular cortex. Ataxia involved arm and leg equally in 7 patients and was more prominent in the arm in 3 patients. One patient with a lesion in the corona radiata had ataxia limited to the leg. Weakness was mild and involved face, arm, and leg in all cases. Dysarthria was mild in 6 patients and absent in 5. Two patients had subjective sensory complaints, and 1 patient with a lesion involving the thalamus and PLIC presented after an episode of loss of consciousness.

Patients with sensorimotor stroke (n=9) showed involvement of the PLIC in 8 of 9 of cases. Lesions were limited to the PLIC in 5 patients. Two additionally involved the putamen, and 1 also involved the thalamus. The only lesion not involving the PLIC was located in the lateral medulla. Weakness involved face, arm, and leg in all patients. In 2 patients there was sparing of facial sensation (both involving the PLIC). Sensory complaints were more prominent than motor deficits in 2 patients (lesions in the lateral medulla and PLIC), while in 2 other patients the motor deficit was more pronounced (lesions in the PLIC, 1 also involving the putamen). One patient had a small hemorrhage involving the PLIC and putamen.

One patient with the dysarthria–clumsy hand syndrome had a lesion involving the PLIC and putamen and the other a lesion in the caudate nucleus. The patient presenting with a pure sensory stroke had a lesion in the thalamus. The sensory deficit was limited to the face and arm.

### Discussion

Previous clinical-anatomic correlation studies of lacunar infarction have been based primarily on autopsy and CT findings. Recent MRI studies have mainly focused on the higher sensitivity of MRI compared with CT for lacunar infarction, without detailing the clinical-anatomic correlation.1,2 Standard MRI is handicapped by its difficulty in differentiating between acute and chronic lesions. Hommel

### Lacunar Syndromes and Infarct Location

<table>
<thead>
<tr>
<th>Lacunar Syndromes (n=43)</th>
<th>Location</th>
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<tbody>
<tr>
<td>Pure motor stroke (n=20)</td>
<td>PLIC (2)</td>
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<tr>
<td></td>
<td>PLIC+putamen (2), thalamus (2), CR (1)</td>
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<tr>
<td></td>
<td>PLIC+putamen/CR (1), putamen/GP (1)</td>
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<td></td>
<td>PLIC+GP/genu/CR (1)</td>
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<td></td>
<td>Pons (6)</td>
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<td></td>
<td>Medial medulla (1)</td>
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<td></td>
<td>CR (3)</td>
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<tr>
<td>Ataxic hemiparesis (n=11)</td>
<td>PLIC (3)</td>
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<tr>
<td></td>
<td>PLIC+thalamus (1), thalamus/cerebral peduncle (1)</td>
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<td>PLIC+CR (1)</td>
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<td>Pons (2)</td>
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<td>Putamen (1)</td>
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<td>CR (1)</td>
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<td>Insular cortex (1)</td>
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<tr>
<td>Sensorimotor stroke (n=9)</td>
<td>PLIC (5)</td>
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<td>PLIC+putamen (2), thalamus (1)</td>
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<td>Lateral medulla (1)</td>
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<tr>
<td>Dysarthria–clumsy hand (n=2)</td>
<td>PLIC+putamen</td>
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<td>Caudate nucleus+white matter</td>
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<tr>
<td>Pure sensory stroke (n=1)</td>
<td>Thalamus</td>
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</tbody>
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CR indicates corona radiata; GP, globus pallidus; and genu, genu internal capsule.
et al., using standard MRI, were able to identify a lacunar infarction appropriate to clinical features in 89 of their 100 patients presenting with an acute lacunar syndrome. However, 16 patients had more than 1 lesion that could explain their symptoms, and 5 others had such extensive chronic changes that the exact location of the acute lesions could not be determined. Clinical-radiological correlation was therefore possible in only 68 of 100 patients. In all of our patients, the lacunar syndrome corresponded to a single acute lesion identified with DW MRI.

The relative frequencies of the classic lacunar syndromes in our study were comparable to those of previous studies. Pure motor stroke was the most frequent lacunar syndrome, followed by ataxic hemiparesis, sensorimotor stroke, dysarthria–clumsy hand syndrome, and pure sensory stroke. Twenty-seven patients had a lesion limited to 1 anatomic structure. Patients with lesions limited to the PLIC presented with pure motor stroke, ataxic hemiparesis, and sensorimotor stroke. Purely pontine lesions were associated with pure motor stroke and ataxic hemiparesis, and lesions in the corona radiata were associated with pure motor stroke and ataxic hemiparesis.

The lesion locations demonstrated by DW imaging in this series of patients included virtually all those previously ascribed to specific lacunar syndromes and demonstrated several previously unrecognized sites as well. Pure motor hemiparesis has been ascribed in pathological studies to small infarctions involving the pons, medullary pyramid, and internal capsule. Studies based on CT scanning have emphasized capsular and corona radiata lesions, consistent with the low sensitivity of this imaging modality for brain stem lesions. We found lesions limited to each of these areas and in addition found lesions involving the PLIC and neighboring structures. Similarly, ataxic hemiparesis was initially ascribed on the basis of pathological study to the basis pontis, but studies based on CT scanning have emphasized the corona radiata and PLIC. An early report of the use of MRI in acute stroke illustrated the bias of CT scanning in this regard, and foreshadowed the superiority of MRI, in describing a case of a patient with an ataxic hemiparesis whose CT scan demonstrated a (presumably new) basis pontis lesion that accounted for all his symptoms. The lacunar syndrome of sensorimotor stroke was initially described on the basis of a pathological study of an infarct that straddled the lateral thalamus and PLIC, but subsequent pathological studies described a patient with sensorimotor stroke who had a lesion limited to the putamen and another with a lesion involving the thalamus, putamen, and PLIC. We found examples of each of these patterns. A CT-based study emphasized larger caudatoputaminal lesions. We did not find any examples akin to these, but 1 of our patients had a lesion in the medial medulla, a region not previously associated with this syndrome, presumably involving the pyramid tract and medial lemniscus. The dysarthria–clumsy hand syndrome was initially ascribed to an infarct in the basis pontis on the basis of a single autopsied case. Subsequent CT-based studies have revealed basis pontis as well as internal capsule and corona radiata lesions. We did not find a pontine lesion; however, our 2 patients had lesions that included the putamen in one instance and the caudate in the other, but both also involved white matter tracts. Our 1 patient with pure sensory stroke had a lesion confined to the thalamus, as originally described by Fisher.

Previous authors have described the extension of lacunar infarctions to neighboring structures on autopsy and CT scan but not to the extent that we observed. In our study, 16 of 33 (48%) of supratentorial lesions showed involvement of nuclei or tracts adjacent to those classically described as giving rise to these syndromes. This involvement was associated with each type of lacunar syndrome we studied except pure sensory stroke. Lesions of the putamen were seen in patients with pure motor stroke, sensorimotor stroke, and dysarthria–clumsy hand syndrome. Thalamic involvement was seen in patients with pure motor stroke, ataxic hemiparesis, and sensorimotor stroke, and involvement of the corona radiata was seen in patients with pure motor stroke and ataxic hemiparesis.

Of the 10 patients with a supratentorial lesion and a moderate to severe deficit, 7 had a lesion extending into neighboring structures. All 3 severely affected patients showed such involvement. Although the significance of this appearance on DW MRI is uncertain, these data suggest that extension of lacunar infarction into neighboring structures, as seen on DW MRI, may be associated with more severe deficits.

As suggested in previous studies, the presence of a lacunar syndrome was not indicative of a single underlying mechanism of stroke. One patient with a sensorimotor syndrome had a small basal ganglia hemorrhage, while another patient with ataxic hemiparesis had atrial fibrillation and a small cortical lesion suggestive of embolic infarction.

Our study confirms previous findings that the classic lacunar syndromes as used in clinical practice describe a heterogeneous group of patients. They are most frequently caused by deep ischemic lesions, but they can also be due to superficial or hemorrhagic lesions. The same lacunar syndrome can be caused by lesions in a variety of locations, and lesions in apparently the same anatomic location can give rise to a variety of lacunar syndromes. Because of its high sensitivity and specificity, DW MRI is a helpful tool for the clinical-pathological study of lacunar syndromes.

Acknowledgment

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References

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