Examining the Lacunar Hypothesis With Diffusion and Perfusion Magnetic Resonance Imaging

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Background—The clinical diagnosis of subcortical cerebral infarction is inaccurate for lesion location and pathogenesis. Clinically suspected small perforating artery occlusions may be embolic infarcts, with important implications for investigation and treatment. New MRI techniques may allow more accurate determination of the stroke mechanism soon after admission.

Methods—In a prospective series of 106 patients evaluated with acute diffusion-weighted MRI (DWI) and perfusion-weighted MRI (PWI) within 24 hours of stroke, we enrolled 19 with a lacunar syndrome. On the basis of the topography, DWI and PWI findings, and outcome T2 MRI, we determined whether the mechanism of infarction was single perforating vessel occlusion or large artery embolism.

Results—Thirteen patients had pure motor stroke, 2 had ataxic hemiparesis, and 4 had sensorimotor stroke. Six patients had lacunes on MRI, none with PWI lesions. Four patients had subcortical and distal cortical infarcts on DWI. Nine had solitary restricted striatocapsular infarcts. Seven of these 9 had PWI studies, 5 with PWI lesions. The presence of a PWI lesion reliably differentiated striatocapsular from lacunar infarction for solitary small subcortical infarcts (P=0.03).

Conclusion—DWI and PWI altered the final diagnosis of infarct pathogenesis from small perforating artery occlusion to large artery embolism in 13 of 19 patients presenting with lacunar syndromes. Lacunes cannot be reliably diagnosed on clinical grounds. (Stroke. 2002;33:2019-2024.)

Key Words: cerebral infarction ■ magnetic resonance imaging ■ stroke

The lacunar hypothesis states that particular clinical syndromes are caused by small infarcts of the brain, owing to single perforating artery occlusion, the result of local microatheroma and thrombosis of that single vessel.1 The pathological foundations for our understanding of lacunes in the modern era were laid by Fisher,2 and subsequent clinico-radiological studies with computed tomography (CT) have supported to some extent the validity of the lacunar hypothesis.3–5 Clinical diagnosis is inaccurate, however, particularly for pure motor stroke,6 and the lacunar hypothesis has been questioned by many authors, most notably Millikan and Futrell,7,8 who point out the considerable evidence in favor of embolism accounting for small deep infarcts in many patients. The possible role of embolism in patients suspected of having lacunar infarction is important from the point of view of investigation and treatment.

Recent studies using diffusion-weighted MRI (DWI) have shown that some patients with suspected subcortical infarcts have cortical lesions,9 and multiple small subcortical infarcts on DWI indicate an embolic source of cerebral ischemia.10–13 In contrast to multiple DWI abnormalities, the stroke etiology in patients with solitary small DWI lesions is more difficult to determine. One early study of DWI in subcortical infarction found a solitary lesion consistent with a lacune in 37 of 39 patients,14 but in a later report by the same group lesions extending beyond lacunar sites were described in 24 of 43 patients enrolled with a lacunar syndrome. Only 1 of these was a cortical lesion, and many of the nonlacunar type involved the capsule and an adjacent structure. No MRI images were included, and volumetric analysis was not performed.15

Volumetric measurements alone, in which the upper volume limit for a lacune should be ~1.8×10³ mm³,16 will not necessarily differentiate acute lacunar from striatocapsular infarction as there may be some overlap in their dimensions on DWI. Given the embolic mechanism of striatocapsular infarcts, perfusion imaging may provide valuable supplementary information.17 In particular, a perfusion defect larger than the DWI lesion might not be expected from single perforator occlusion, even when the DWI lesion is of lacunar dimensions. This is not to say that there is no perfusion lesion accompanying a lacune, but that hypoperfusion of one single
perforating artery may be beneath the resolution of current MRI perfusion imaging methods. There are no studies addressing this issue. Combined DWI and magnetic resonance angiography (MRA) has been shown to improve diagnostic accuracy with respect to stroke subtype in one recent study.13

There is no study looking at the combination of diffusion and perfusion imaging in suspected lacunar infarction. We aimed to assess the influence of combined DWI and perfusion-weighted MRI (PWI) on the final diagnosis of infarct pathogenesis in patients admitted with lacunar syndromes.

Methods

Patients

Patients with a sudden-onset focal neurological deficit consistent with hemispheric ischemic stroke were recruited prospectively from the Stroke Service of the Royal Melbourne Hospital where 400 patients with acute stroke are admitted each year. The period of the recruitment was 50 months. Stroke onset was defined as the last time the patient was known to be without a neurological deficit. Patients were excluded if they had cerebral hemorrhage, preexisting significant nonischemic neurological deficits (including dementia or extrapyramidal disease), or a history of prior stroke that would hamper interpretation of clinical and radiological data. There were no age, gender, or handedness exclusions. The study was performed with the approval of the Clinical Research and Ethics Committee at our institution, and written informed consent was obtained from the patient or next of kin. All patients had MRI studies within 24 hours of stroke onset, and all had outcome T2-weighted MRI studies at 3 months. All clinical assessments were performed by a neurologist or neurology resident without knowledge of the imaging results. The clinical diagnosis was made in the emergency department by either the neurologist or neurology resident trained in the differentiation of subcortical from cortical infarcts. No formal assessment of interobserver agreement was made between any 2 examiners, but the diagnosis of all patients was reviewed by the admitting stroke neurologist who either assessed the patient in the emergency department or reviewed the patient within 24 hours of admission. Later information from neuroimaging or nonacute neurological or neuropsychological assessments did not affect the acute clinical infarct classification.

All MRI scans were obtained using a 1.5-T echo-planar imaging (EPI)—equipped whole-body scanner (Signa Horizon SR 120; General Electric) using a standard protocol as previously described.17–20

Diffusion Imaging

DWI was obtained using a multislice, single-shot spin-echo EPI sequence. Slice thickness was 6 mm with a 1-mm gap, with the number of slices set to include the whole brain (average of 15). Matrix size was 256 × 128 (128 × 128 for the first 7 patients), field of view was 40 × 20 cm, and TR/TE was 6000/100 ms (TE was 75 ms for the first 5 patients). Diffusion gradient strength was varied between 0 and 22 mT/m, resulting in 5 b values of increasing magnitude from 0 up to 1200 s/mm². The diffusion gradient was applied in 3 orthogonal directions (x, y, and z), and an average of these measurements was calculated to give the trace of the diffusion tensor.

The protocol was changed after the first 22 patients, with TR/TE of 10 000/100 ms, and the diffusion gradient strength varied so that there were 3 b values of increasing magnitude from 0 to 1000 s/mm². The diffusion sensitizing gradient was applied in 6 directions (xy, xz, yz, x, y, and z), however, for the purposes of this study, analyses were performed from the average of the measurements taken in the x, y, and z orthogonal directions.

Perfusion Imaging

Perfusion images were obtained using dynamic first-pass bolus tracking of gadolinium diethylenetriamine penta-acetic acid (Gd-DTPA) using an EPI spin-echo sequence with a TR/TE of 2000/70 ms. The Gd-DTPA bolus (0.2 mmol/kg) was administered by manual intravenous injection over ~5 seconds using 1 large-bore cannula in the antecubital fossa in the first 18 patients recruited, and subsequently an EPI gradient-echo sequence with a TR/TE of 2000/70 ms was used with the Gd-DTPA bolus (0.1 mmol/kg) being administered by a power injector (Spectris MR injector, MEDRAD) at a rate of 5 mL/s. A total of 10 slices were obtained, with a slice thickness of 6 mm with a 1-mm gap, a matrix of 256 × 128, and a field of view of 40 × 20 cm. Images were obtained at 40 time points per slice, with a total imaging time of 1 minute 21 seconds. The concentration-time curve obtained was processed on a voxel-by-voxel basis, allowing determination of an observed or relative mean transit time map, where the relative mean transit time is related to the sum of the true mean transit time plus the injection time.

Magnetic Resonance Angiography

Phase-contrast MRA was performed as previously described.19

Postprocessing of MR images was performed using customized software based on commercial image analysis applications (Advanced Visualization Systems) using an Indigo R10000 workstation (Silicon Graphics Inc). DWI volumes were measured using the maximum diffusion sensitivity isotropic image because this showed the greatest contrast between the hyperintense infarct and the surrounding tissue. The edge of the DWI or PWI lesion was identified visually and regions of interest were outlined using a manual pixelwise method. The area of the region of interest was multiplied by the slice thickness plus the interslice gap and then summed. Analyses used the average of 2 measurements taken on separate occasions by 2 neurologists trained in the technique and blinded to clinical data. Intra- and interobserver agreement was high with variability <10%.

Infarct Subtype Definition

The lacunar syndromes were the following: (1) pure motor hemiparesis, weakness of face, arm, and leg without sensory symptoms or signs and without evidence of dysphasia; (2) pure sensory stroke, hemianesthesia without cortical signs or weakness; (3) sensorimotor stroke, a combination of the above; (4) ataxic hemiparesis, ataxia of the arm and leg with ataxia out of proportion to the weakness; and (5) dysarthria clumsy hand syndrome.21

The final infarct pathogenesis was determined on the basis of a combination of the infarct topography on acute DWI and the final outcome T2 volumes. The acute DWI reliably revealed recent infarction location and number, and differentiated any old ischemic lesions from those to be analyzed. Where there were multiple lesions on DWI, the infarct was attributed to embolism. Solitary infarcts were classified as lacunes, attributable to single perforating artery occlusion, if the 3-month T2 MRI volume was <1.8 × 10⁶ mm³. This cutoff volume was based on the usual definition of an upper diameter limit of 15 mm for lacunes,22 assuming lacunes are spherical.16 Infarct topography was deemed consistent with a lacune for spherical or spheroidal infarcts of the above dimensions, and including cylindrical infarcts of the pons such as paramedian perforating artery territory infarcts. Infarcts including the putamen, caudate, and capsule were classified as striatocapsular infarcts. Smaller lenticto-lostriate artery territory infarcts larger than 1.8 × 10⁶ mm³ on 3-month T2 MRI were classified as restricted striatocapsular infarcts.23 Such infarcts are most often lateral striatocapsular, and most obviously affect the posterior one-third or one-half of the putamen. The outcome T2 MRI provided the volumes for the final diagnostic classification, as the traditional size criteria are taken from pathological studies that examined mostly old infarcts. Furthermore, it is known that acute DWI lesions with PWI deficits expand in the first 72 hours,18,24 whereas lacunes have been shown to shrink slightly on outcome T2 follow-up, 1 to 5 months later.19
TABLE 1. Clinical and MRI Volume Data

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<th>Age</th>
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</table>

PMS indicates pure motor stroke; SC, striatocapsular infarct; Lac, lacune; Put, putamen; Caps, capsular; MB, midbrain; Thal, thalamus; Occip, occipital; AH, ataxic hemiparesis; AF, atrial fibrillation; HT, hypertension; CCF, cardiac failure; DM, diabetes; ICA, internal carotid artery; Hyperchol, hypercholesterolaemia; LA, left atrium; and PFO, patent foramen ovale.

Statistical Analysis

Figures expressed as means are appended ± SD. The Fisher exact test was used for categorical variables. Results were considered significant at \(P<0.05\).

Results

Nineteen (18%) of 106 patients presented with typical lacunar syndromes (Table 1). Five patients of 111 presenting within 24 hours with acute cerebral infarction had been excluded as a result of previous stroke and residual neurological deficit. There were 11 women and 8 men with mean age 68.5±9.9 years. Thirteen patients had pure motor stroke, 2 had ataxic hemiparesis, and 4 had sensorimotor stroke. One patient with pure motor hemiparesis had a contralateral sixth nerve palsy as a result of a single perforating artery territory pontine infarct (patient 13). This would be consistent with Fisher’s21 lacunar syndrome number 11 in the table included in his review of lacunar infarcts. No patient had dysarthria clumsy hand syndrome. The patient with the largest striatocapsular infarct at presentation (patient 17) had no cortical signs on routine bedside testing but was subsequently found to have subtle inattention to formal neuropsychological tests of visual function.

Four of the 19 patients had recognized sources of embolism, atrial fibrillation, carotid stenosis, and patent foramen ovale, and all had striatocapsular or cortical infarction or both, consistent with an embolic pathogenesis. None of the 6 patients with an MRI final diagnosis of lacunar infarction had a recognized source of embolism. Eleven patients had a history of hypertension, 4 of the 6 with lacunes and 7 of the remaining 13 with embolic infarcts. One patient had had a previous contralateral stroke caused by a lacune (patient 3, Table 1).

There were 6 isolated subcortical infarcts with a final diagnosis of lacunar infarction. The positive predictive value of a clinical diagnosis of lacunar infarction in the 19 patients was 32%. The average lacune volume on 3-month T2 MRI was 0.57±0.27×10$^3$ mm$^3$. Three other patients had acute DWI lesion volumes <1.8×10$^3$ mm$^3$ but had subsequent infarct expansion and final diagnoses of striatocapsular infarction (patients 2, 8, and 19; see Methods, Table 1, and Figure 1). Six further patients had isolated restricted striatocapsular infarction with acute DWI and outcome T2 volumes >1.8×10$^3$ mm$^3$. Four patients had both subcortical and cortical infarction on acute DWI (Figure 2).

For the isolated solitary subcortical hemisphere infarcts, there was a significant association between the presence of an acute PWI lesion and a larger 3-month T2 infarct volume (>1.8×10$^3$ mm$^3$), consistent with restricted striatocapsular infarction. Of the 7 patients with restricted striatocapsular infarcts who had a PWI study, 5 had PWI lesions. Three of these patients with PWI lesions larger than DWI had acute DWI lesion volumes <1.8×10$^3$ mm$^3$, but had subsequent infarct expansion. No patient with lacunar infarction as the final diagnosis had an acute PWI deficit (\(P=0.03\), Fisher’s exact test; see Table 2.) Acute PWI lesions were present in all 4 patients with cortical infarcts. The MRA did not show occlusion of the middle cerebral artery (MCA) or posterior circulation arteries in any of the 19 cases.
Discussion

This is the first report detailing the role of PWI in determination of the pathogenesis of infarction in patients presenting with lacunar syndromes. Of 19 patients with clinical presentations typical of lacunar infarction, 13 had MRI findings not consistent with lacunar infarction as a result of occlusion of a single perforating vessel. This conclusion was most obvious when multiple DWI lesions were demonstrated within a large artery territory. In the MCA territory this was typically a simultaneous small striatocapsular infarct and a distal small cortical infarct. This pattern is most likely to be due to thrombus in the MCA, occluding the origins of a number of lenticulostriate penetrating arteries, with subsequent fragmentation and distal cortical branch occlusion. Embolism is usually the cause of such proximal thrombotic MCA occlusions.25–27 This series of lacunar syndrome patients may not be representative of patients encountered routinely in an emergency department, given the small sample taken from >800 stroke admissions, but the percentage of lacunar presentations within this larger MRI series (18%) is consistent with many prospective registries of stroke subtype. One previous series has described cortical infarcts in 2 of 23 patients presenting with lacunar syndromes,9 but another group found only 1 cortical infarct in 43 patients with lacunar syndromes.15 The early clinical assessment in our study may have accounted for missing subtle signs of dysfunction of cortical modalities, but although there is good evidence that stroke subtype definition within 12 hours is less accurate than at a later time point,28–30 the very small cortical infarcts seen on DWI in the 4 patients with combined cortical and subcortical infarcts were not likely to be clinically detectable. This early misdiagnosis is certainly relevant, however, to the larger striatocapsular infarcts (see below).

Solitary infarcts in the lenticulostriate distribution, larger than lacunes on acute DWI, constituted restricted striatocapsular infarcts (see Methods). The clear elucidation of the topography and mechanism of striatocapsular infarction occurred only in the CT era.25–27 They are typically comma shaped and large enough to include the caudate head, putamen, and intervening internal capsule, dwarfing the typical lacune with dimensions on the order of 45×40×20 mm.25 However, smaller lesions do occur. Fisher’s “giant lacunes,” infarcts >10 mm in diameter,2 were probably striatocapsular infarcts for the most part.23,31 In the only formal subcortical infarct classification so far proposed, size was not deemed the only determinant of lacunes and striatocapsular infarcts, limited forms of which were allowed to occur.22 In one CT study of pure motor hemiparesis, a subset of the lesions were clearly striatocapsular infarcts.32 In such cases misclassified as lacunes, early recanalization of the occluded MCA has been postulated as the explanation for the brief or inapparent cortical dysfunction that is typical of large striatocapsular infarcts.23 A restricted striatocapsular infarct involving the posterior putamen is depicted in cartoon form (Reference 33, Figure 1, panel 1) in a transcranial Doppler study of MCA embolic occlusion, and a pathological specimen (Reference 23, Figure 12.1) in an important series of striatocapsular infarcts, all typical of infarcts seen regularly not only on MRI but also on CT in routine clinical practice. Such infarcts are not lacunes and could not be accounted for by single perforating artery occlusion. In a series of 43 patients presenting with lacunar syndromes,15 9 patients had infarction of at least 2 striatocapsular elements, most commonly posterior limb of internal capsule and putamen. Without seeing the infarct topography they encountered, one could only speculate as to whether they were seeing similar types of infarct.

### Table 2. PWI Lesion Frequency in the Two Hemisphere Infarct Subtypes for Solitary Lesions

<table>
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<tr>
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<th>PWI⁻</th>
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</thead>
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<td>5</td>
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<tr>
<td>Striatocapsular</td>
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</table>

*P*=0.03 (Fisher exact test, one tailed). PWI⁺ indicates PWI lesion present; PWI⁻, PWI lesion not present.

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**Figure 1.** Patient 2. Top, Acute DWI showing a bifid infarct topography but with volume within lacunar limits. Middle, Acute PWI lesion (arrows). Bottom, day 4 DWI showing expansion of the infarct to dimensions and topography of a restricted striatocapsular infarction. h indicates hours; d, days.

**Figure 2.** Patient 18. Top, Acute DWI showing striatocapsular and cortical infarction (arrow). Middle, Acute PWI. Bottom, DWI at 72 hours. h indicates hours; d, days.
In the acute stage then (<24 hours), DWI alone may not be able to differentiate lacunes from restricted striatocapsular infarcts. Although none of the infarcts with a final diagnosis of lacune had a PWI lesion, 3 patients had acute infarcts of lacunar dimensions on DWI but with a larger acute PWI deficit (patients 2, 14, and 19; see Figure 1). All 3 infarcts expanded to lesions larger than lacunes at 3 months. For small acute DWI lesions, the presence of a larger acute PWI deficit may be a reliable determinant of an embolic mechanism of cerebral infarction on initial MRI studies soon after the onset of stroke. In 1 patient (patient 16, Table 1), the 3-hour diffusion lesion of $2.5 \times 10^3$ mm$^3$ had a smaller PWI lesion of $1.0 \times 10^3$ mm$^3$ and the outcome lesion shrank to an outcome T2 volume of $1.9 \times 10^3$ mm$^3$. The lack of expansion may have related to early reperfusion and the resolution of acute infarction edema.

In the patient just discussed and in patients 4, 8, and 19, the volumes of the outcome lesions were certainly close enough to the stated volume cutoff of $1.8 \times 10^3$ mm$^3$ that they may actually have been lacunes rather than smaller restricted striatocapsular infarcts due to embolism. This is particularly so for patients 4 and 8, considering that neither of them had a PWI lesion. The absence of a PWI lesion in these and the lacunar infarcts is likely to relate to the resolution of the technique, inasmuch as it is likely that there would be a region of hypoperfusion associated with even the smallest infarction.

None of the 19 patients had occlusion of the MCA or other cerebral artery on MRI. This is likely to be one of the factors giving rise to the milder strokes in these patients; the striatocapsular patients must at some point before assessment have had MCA embolic occlusion, but spontaneous recanalization has spared them from large territorial infarction and other small infarcts with an embolic etiology constitute even the most carefully chosen clinical set of “lacunar” infarcts.

Two other recent DWI studies suggest this. With the possibility of occult cardiac embolism due to intermittent atrial fibrillation, and with the emergence of other proximal pathologies as significant potential sources of embolism, decisions about further investigation should not necessarily be based solely on clinical bedside examination for cortical signs. Further prospective studies of patients with lacunar syndromes are needed to assess the routine clinical implications of this study, but for patients with lacunar syndromes acute diffusion and perhaps perfusion MRI may be an important modality for obtaining an early accurate diagnosis of the infarct type and mechanism.

Acknowledgments

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References

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