Diffusion-Weighted Imaging Identifies a Subset of Lacunar Infarction Associated With Embolic Source

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Background and Purpose—Small infarcts in the territory of penetrator arteries were described as causing a number of distinct clinical syndromes. The vascular pathophysiology underlying such infarcts is difficult to ascertain without careful pathological study. However, the occurrence of multiple, small infarcts, linked closely in time but dispersed widely in the brain, raises the possibility of an embolic mechanism. The current study determines the frequency and clinical characteristics of patients with well-defined lacunar syndromes and the diffusion-weighted imaging (DWI) evidence of multiple acute lesions.

Methods—Sixty-two consecutive patients who presented to the emergency room with a clinically well-defined lacunar syndrome were studied by DWI within the first 3 days of admission.

Results—DWI showed multiple regions of increased signal intensity in 10 patients (16%). A hemispheric or brain stem lesion in a penetrator territory that accounted for the clinical syndrome (“index lesion”) was found in all. DWI-hyperintense lesions other than the index lesion (“subsidiary infarctions”) were punctate and lay within leptomeningeal artery territories in the majority. As opposed to patients with a single lacunar infarction, patients with a subsidiary infarction more frequently \( (P < 0.05) \) harbored an identifiable cause of stroke.

Conclusions—Almost 1 of every 6 patients presenting with a classic lacunar syndrome has multiple infarctions demonstrated on DWI. This DWI finding usually indicates an identifiable cause of stroke and therefore may influence clinical decisions regarding the extent of etiologic investigations and treatment for secondary prevention. \( (Stroke. 1999;30:2644-2650.) \)

Key Words: embolism • lacunar infarction • magnetic resonance imaging, diffusion-weighted

Lacunar infarctions are small brain lesions (0.2 to 15 mm³) caused by occlusion of arteries that arise at abrupt angles from the large arteries of the circle of Willis or from the basilar artery.¹,² The penetrator vessels range from 40 to 900 μm in diameter and immediately enter the brain tissue after their origin. Occlusion of such vessels gives rise to a number of well-described clinical syndromes, sometimes with rapidly fluctuating symptoms. Patients may present with severe motor or sensory deficits, disproportionate to the small size of the brain lesion.

Lacunar infarctions are caused by a variety of different mechanisms. In most instances, the primary pathological event (microatheroma formation or lipohyalinosis) is limited to the small penetrator vessels themselves (so-called “microvascular” or “small-vessel” disease). Microatheroma, plaques of foam cells occluding the proximal segments of relatively larger-size penetrating vessels (300 to 900 μm in diameter), is the most common cause of lacunes identified in pathological studies.¹⁻³ Lipohyalinosis, a cerebral vasculopathy associated with untreated, long-lasting hypertension, designates replacement of the muscle and elastic laminae by collagen and dep, which in some places are sufficient to cause occlusion of vessels with diameters of <200 μm.¹,²,⁴ Also, an atheroma lining the parent vessel (basilar artery, middle cerebral artery) may occlude the origin of a penetrator and lead to a lacunar infarction.²⁻⁵⁻⁷ Current diagnostic technology does not allow the identification of either microatheroma or lipohyalinosis as the cause of lacunar infarction. Angiography may help diagnose atheroma in the parent vessel from which the occluded vessels arises. A small embolic particle may also, on occasion, lodge in a penetrating artery and result in lacunar infarction.²⁻³ Accurate identification of such patients is critical because the underlying embolic source may have the potential to give rise to a subsequent larger embolus that causes more extensive brain injury.

Because infarcts lose signal intensity over a period of days to a few weeks on DWI, this imaging modality conveys temporal as well as spatial information about the ischemic event. 
lesion. In studying patients with classic lacunar syndromes by DWI, we frequently observed typical acute small lesions in the territory of penetrating arteries. On occasion, however, 1 or 2 additional acute lesions were also found. These multiple DWI lesions might lie in different vascular territories, perhaps in the opposite hemisphere, which suggests that embolism might be the operative mechanism of the lacunar syndrome. In the present study, we determined the frequency and the clinical characteristics of this DWI finding in a consecutive series of patients presenting with a classic lacunar syndrome.

Subjects and Methods

Over the period of 25 months from July 1996 and through July 1998, 62 consecutive patients presented to the emergency room with a well-defined lacunar syndrome. Each patient was examined by a neurologist soon after arrival. An intracerebral hemorrhage was excluded by CT in each in the emergency room. The medical history and patient’s symptoms were carefully reviewed. For the purposes of the current study, we included patients with the following “classic” lacunar syndromes: ataxic hemiparesis, dysarthria-clumsy hand, pure motor hemiparesis, pure sensory stroke, and sensorimotor syndrome. Absent were aphasia, apraxia, agnosia, monoplegia, isolated memory impairment, homonymous hemianopsia, seizures, and stupor. Though these conservative criteria exclude some other lacunar syndromes, these 5 classic syndromes have been linked most firmly by pathology to lacunar lesions.8–13

In each patient, time and mode of onset of symptoms were established. The latter was classified as sudden onset with maximum deficit, gradual onset with progressive worsening, or fluctuating or stuttering onset. The following cerebrovascular risk factors were recorded: hypertension, diabetes mellitus, hyperlipidemia, atrial fibrillation, smoking, previous transient ischemic attack or stroke, coronary artery disease, and peripheral vascular disease. To identify potential mechanism of cerebral infarction, a set of diagnostic tests was performed that included blood chemistry, blood cell counts, and electrocardiography in all patients; many also underwent carotid duplex ultrasonography, transcranial Doppler ultrasonography, MR angiography (MRA), special coagulation tests, transthoracic echocardiography, MR imaging was performed within 3 days of symptom onset, on a 1.5-Tesla whole-body scanner (GE Sigma) with echo-planar capabilities (Advanced NMR Systems). We used conventional MR sequences as well as single-shot echo-planar imaging (EPI), as previously described.13 Using 3 orthogonal acquisitions, we sampled the “trace” of the diffusion tensor, which is spatially invariant and previously described.15 Using 3 orthogonal acquisitions, we sampled the “trace” of the diffusion tensor, which is spatially invariant and previously described.15

Results

DWI was entirely normal in 9 of the 62 patients (14%) admitted with a classic lacunar syndrome; the symptoms had completely resolved by the time of imaging in 8 of them. In an additional patient with pure motor hemiparesis, DWI did not reveal a lesion in the territory of a penetrating vessel but did show a small inferior, and a second small superior, lesion in the motor strip.

Patients With Single Acute Infarct

Forty-two of 62 patients (68%) had a single hyperintense lesion in the territory of deep hemispheric or brain stem penetrating. There were 25 male and 17 female patients. The mean±SD age was 67±13 years (range 42 to 89 years). All but 1 patient had at least 1 cerebrovascular risk factor; 36 patients were on medications for hypertension, 13 for diabetes mellitus, and 19 for hypercholesterolemia. One patient had atrial fibrillation, and 5 had a previous history of ischemic stroke. Pure motor hemiparesis (PMH) was the most common presenting syndrome (21 patients). Nine patients had ataxic hemiparesis (AH), seven had sensorimotor stroke (SMS), three dysarthria-clumsy hand syndrome (DCHS), and two, pure sensory syndrome (PSS). Within the first 24 hours, the clinical deficit gradually worsened in 15 and fluctuated in nine patients. The mean±SD time between symptom onset and DWI study was 39±23 hours. All DWI lesions were localized in a region considered appropriate for the symptoms. DWI showed a hyperintense lesion in the corona radiata and/or lenticular nucleus in 17 patients, internal capsule in 6, thalamus in 10, and pons in 9. Echocardiography was performed in 31 patients (74%): transthoracic in 29 and transesophageal in 2. They revealed a potential source of cardiac emboli in 6 patients; left ventricular apical akinesis and hypokinesis in 2, patent foramen ovale in 2, and asymmetric septal hypertrophy and mild aortic valve stenosis in 1 each. Thirty-seven patients (88%) had at least 1 vascular study to evaluate the extracranial and intracranial brain vessels. Continuous-wave Doppler ultrasonography of the neck vessels was performed in 30 patients (71%), MRA in 27 (64%), and transcranial Doppler ultrasonography in 29 (69%). Overall, vascular studies revealed mild ipsilateral extracranial internal carotid artery stenosis (>30% diameter reduction) in 1 patient with a corona radiata lesion, and midbasilar artery stenosis in 1 and mild atherosclerotic changes in another with a pontine lacune. Three had acute lesions in the posterior circulation (pons or thalamus), with internal carotid artery stenosis (>70% diameter reduction) of the extracranial (2 patients) or the siphon (1 patient) segments; none had fetal-type posterior cerebral artery on MRA.

Patients With Multiple Acute Infarcts

DWI showed multiple regions of increased signal intensity in the remaining 10 of 62 patients (16%). There were 5 male and
5 female patients. Mean ± SD age was 65 ± 15 years (range 36 to 85 years). Table 1 shows cerebrovascular risk factors, type of lacunar syndrome, and cardiac and cerebrovascular findings. In this subgroup, there was no patient whose deficit fluctuated; the clinical deficit worsened progressively within the first 24 hours in 3 patients and was sudden in onset then stable in 7 patients. None of the 10 patients experienced any transient alteration in consciousness. A stroke mechanism, most often embolic, could be identified in 8 patients: atrial fibrillation in 2, and 1 each with severe left ventricular hypokinesis, mobile aortic arch atheroma, severe stenosis in the vertebrobasilar junction, patent foramen ovale, atheroma plaque in the ipsilateral carotid bulb, and cocaine-associated vasculopathy. In the remaining 2 (Table 1, patients 7 and 9), a definite causative mechanism could not be identified; MRA revealed only minimal disease of both carotid bifurcations in each patient.

DWI was performed with a mean ± SD delay of 38 ± 33 hours after symptom onset in patients with multiple infarctions; there were two hyperintense lesions in eight patients, and three hyperintense lesions in 2 (Table 2). At least 1 of the hyperintense lesions was within the territory supplied by either a brain stem or a deep hemispheric perforator. The deep brain stem lesion location correlated well with the clinical symptom in all patients (“index lesion”); their dimensions on DWI ranged between 5 mm and 20 mm. Brain stem paramedian territories were involved by the index lesion in 5 patients, putamen and adjacent corona radiata in 3, thalamus in 1, and posterior limb of the internal capsule in 1. DWI-hyperintense lesions other than the index lesion were termed “subsidiary infarctions.” They lay within the same circulation as the index lesion (ie, either within the posterior circulation or within the anterior circulation of the same hemisphere) in 6 patients and within different circulations in 4 patients (ie, one lesion within the anterior and the other within the posterior circulation). Subsidiary infarctions involved cerebral leptomeningeal territory in 5 patients (Figure 1), cerebellar leptomeningeal territory in 2 (Figure 2), and brain stem or deep hemispheric perforator territory in 3 (Table 2).

### Table 1. Clinical Characteristics and Etiologic Findings

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age, y</th>
<th>Clinical Syndrome</th>
<th>Risk Factors</th>
<th>ECG</th>
<th>Echocardiography</th>
<th>TCD</th>
<th>Carotid-Vertebral Ultrasoundography</th>
<th>MRA</th>
<th>Other Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>72</td>
<td>L AH</td>
<td>HT, DM, H C, CHD, S</td>
<td>AF</td>
<td>Papillary muscle calcification*</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>75</td>
<td>R PMH</td>
<td>HT, CHD</td>
<td>AF</td>
<td>ND</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>36</td>
<td>L SMS</td>
<td>S</td>
<td>N</td>
<td>N†</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Urine positive for cocaine</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>50</td>
<td>R PMH</td>
<td>HT, DM, C HD</td>
<td>N</td>
<td>Severe inferobasal hypokinesis*</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>64</td>
<td>L AH</td>
<td>HT, DM, H C, S</td>
<td>N</td>
<td>N*</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Atheroma in the R carotid bulb</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>85</td>
<td>L PMH</td>
<td>HT</td>
<td>N</td>
<td>PFO*</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>53</td>
<td>R SMS</td>
<td>HT, CHD, S</td>
<td>N</td>
<td>N†</td>
<td>N</td>
<td>Atherosclerotic changes in both carotid bifurcations</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>77</td>
<td>L PMH</td>
<td>HT, DM</td>
<td>N</td>
<td>N*</td>
<td>R vertebral and proximal basilar stenosis</td>
<td>R vertebral stenosis</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>80</td>
<td>R DCHS</td>
<td>DM, PS</td>
<td>N</td>
<td>N*</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Atherosclerotic changes in both carotid bifurcations</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>65</td>
<td>R PSS</td>
<td>S</td>
<td>N</td>
<td>Mitral valve strands and mobile aortic atheroma†</td>
<td>ND</td>
<td>ND</td>
<td>N</td>
<td></td>
</tr>
</tbody>
</table>

AH indicates ataxic hemiparesis; PMH, pure motor hemiparesis; SMS, sensory motor stroke; PSS, pure sensory stroke; DCHS, dysarthria–clumsy hand syndrome; HT, hypertension; DM, diabetes mellitus; HC, hypercholesterolemia; S, smoking; CHD, coronary heart disease; PV, previous stroke; AF, atrial fibrillation; N, normal; ND, not done; PFO, patent foramen ovale; and TCD, transcranial Doppler sonography.

*Transthoracic and †transesophageal echocardiography were performed as noted.

### Table 2. Locations of Hyperintense Lesions on DWI

<table>
<thead>
<tr>
<th>Patient</th>
<th>Index Infarction</th>
<th>Subsidiary Infarction(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>R pontine paramedian</td>
<td>L frontal cortical</td>
</tr>
<tr>
<td>2</td>
<td>L pontine paramedian</td>
<td>L occipital cortical</td>
</tr>
<tr>
<td>3</td>
<td>R thalamus</td>
<td>R cerebellar cortical</td>
</tr>
<tr>
<td>4</td>
<td>L posterior limb of IC</td>
<td>L frontal white matter</td>
</tr>
<tr>
<td>5</td>
<td>R putamen and CR</td>
<td>R frontal cortical</td>
</tr>
<tr>
<td>6</td>
<td>R putamen and CR</td>
<td>L midbrain</td>
</tr>
<tr>
<td>7</td>
<td>L pontine paramedian</td>
<td>L occipital cortical</td>
</tr>
<tr>
<td>8</td>
<td>R pontine paramedian</td>
<td>L cerebellar cortical</td>
</tr>
<tr>
<td>9</td>
<td>L pontine paramedian</td>
<td>L frontal cortical (2 adjacent lesions)</td>
</tr>
<tr>
<td>10</td>
<td>L putamen and CR</td>
<td>L lateral thalamus</td>
</tr>
</tbody>
</table>

IC indicates internal capsule; CR, corona radiata.
Subsidiary lesions were punctate (ie, <5 mm in diameter) in 6, and <10 mm in diameter in all but 1 patient (patient 4, 20 mm).

There were no statistically significant differences between patients with single infarction and those with multiple infarctions on DWI in terms of age, sex, presence of individual risk factors, mean number of risk factors, mean time to MRI, clinical syndrome, mode of onset, and number of etiologic investigations (echocardiography, carotid duplex ultrasonography, MRA). Patients with subsidiary infarctions more frequently harbored an underlying embolic source than did patients with single lesion (P<0.05).

Discussion

C. Miller Fisher first raised the possibility of embolism as a cause of lacunar infarction. In his study of 11 capsular infarcts in 10 autopsies, the perforating arteries running into the infarction did not exhibit any pathological change in the lumen or within the vessel wall in 2 cases, at 1 month and 6 years, respectively, after the clinical event. Embolism was postulated as the mechanism, because embolic material might disappear over time whereas complete resolution of atheroma was unlikely. In another patient he examined 6.5 years after the stroke, there was a nonocclusive microatheroma in a penetrating vessel, reducing its lumen from 400 μm to 150 μm. The cause of infarction was uncertain, yet embolism remained a possibility.

The idea of embolic lacunar infarction remained dubious because of the seemingly low likelihood of an embolus entering a vascular territory that received such a small portion of the cerebral blood flow. Moreover, the sharp angle that penetrating vessels make with the parent vessel renders it more likely that an embolus would be directed toward the leptomeningeal arteries, into which the main stream of laminar flow is aimed. On the other hand, embolism was still a consideration as the cause of lacunar infarction in rare patients with, for example, subacute bacterial endocarditis. Likewise, Cacciatore and Russo described 2 patients with PMH that developed during cardiac angiography in one and during arch aortography in the other; 2 weeks later, CT revealed a low-density lesion in the posterior limb of the contralateral internal capsule in the former and was normal in the latter.

Recently, the fate of embolic material was tested in animals. Seventy thousand to 1 million microspheres (ranging from 31 to 92 μm in mean diameter) were injected into the internal carotid artery of monkeys over a 10-minute period, and histological brain sections were obtained postmortem. Depending on microsphere size, 1.4% to 6% of the injected spheres entered small penetrating vessels. In human stroke, this may translate into massive shower of emboli, as can be seen during cardiac and aortic operations, in cholesterol embolization from ulcerated atheroma, and in paradoxical fat or air embolism. An in vivo model of embolic shower from an arterial site was produced by Futrell et al. A photochemical lesion was created in the rat carotid artery, resulting in rapid formation of a platelet thrombus. Multiple small foci of brain infarction occurred, ranging from 2 to 8 in number and from 0.2 to 1.7 mm in diameter. Overall, 9 of 44 lesions in 12 rats were within the territory of penetrating arteries.
A similar combination of cortical and subcortical infarctions was also observed in cases with autopsy-confirmed cholesterol embolism from aortic atheroma. In a pathological study of 16 patients with aortic atheroma, Soloway and Aronson demonstrated cholesterol crystals within the leptomeningeal cortical and cerebellar branches in all and in a small artery in the basal ganglia in one. Laloux and Brucher reported the case of another patient with a dissecting aneurysm of the aorta who was found at autopsy to have multiple lacunar infarcts in the thalamus, putamen, and caudate nuclei as well as in multiple cortical foci. Vessels as small as 14 \( \mu \text{m} \) were loaded with cholesterol crystals. These autopsy findings, as well as the animal data, suggest that if a penetrating vessel is occluded by a small embolus, it is expected that many more small emboli will also have traveled to cortical vessels. Depending on the differential probability of infarction due to such small emboli in different vascular territories, multiple brain lesions will be expected. Because of its lack of collateral flow pathways, the penetrator territory is possibly more susceptible to infarction on entry of a very small embolus compared with the leptomeningeal territory. Such a regional difference in infarction rate due to tiny emboli could explain our finding of only 1 or 2 cortical lesions for each penetrator lesion as opposed to the greater ratio expected from studies of embolus distribution. Furthermore, our highly restrictive patient selection criterion of presentation with a classic lacunar syndrome might also have led to this low ratio of leptomeningeal/penetrator territory infarctions. Because of the inherent inability of autopsy studies to establish the ages of chronic lesions, it is difficult to firmly relate the superficial and the lacunar lesions to each other when they are found together; they may have occurred separately in time due to the coincidental presence of both small-vessel occlusive disease and cerebral embolism. Neither do standard imaging studies (CT and conventional MRI) provide temporal evidence that a penetrator territory lesion identified on imaging is the cause of the acute syndrome. Lacunar infarctions usually do not exhibit obvious edema, mass effect, or contrast enhancement that might mark them as “acute.” Moreover, silent, chronic lesions, mainly in the white matter, are noted in 29% to 47% of patients with acute stroke scanned by CT or conventional MRI. Therefore, it is difficult to be certain which lacunar infarction is the acute lesion and causally related to symptoms and which is chronic and not associated with the clinical syndrome.

DWI differs from CT and conventional MRI in that it provides information about multiplicity of cerebral infarctions as well as establishes a temporal association between symptoms and the lesion(s). In acute brain ischemia, extracellular water moves intracellularly, resulting in a decrease in the apparent diffusion coefficient (ADC) of water, which contributes to the increased signal on DWI. In the subacute period—after approximately 1 to 2 weeks—initially reduced ADC values return to baseline. Only acute to subacute lesions appear hyperintense on DWI. Thus, this characteristic allows differentiation of new lesion(s) from old asymptomatic infarction(s). Moreover, DWI identifies small subcortical and brain stem lesions better than conventional MRI because of the high lesion-to-background signal ratio. Most lacunar lesions, as well as the subsidiary lesions we observed in this study, were small (Table 2).

In this study of 62 patients presenting with a classic lacunar syndrome, 16% had multiple infarctions on DWI in either a single (6 patients) or different (4 patients) vascular territories.

Figure 2. Patient 3. Axial DWI demonstrates a hyperintense signal in the territory of a right thalamic penetrator (index lesion; top row) and another hyperintense lesion in the right cerebellar cortex (subsidiary lesion; bottom).
Those with multiple infarctions on DWI were more likely to harbor an identifiable stroke mechanism than those with a single lacunar infarction (80% versus 24%; P<0.05). None had a fluctuating deficit, which might be expected to occur more commonly in thrombotic lesions. Indeed, 9 of 42 patients with single infarction in a penetrator territory had fluctuating symptoms. Embolism was suspected in 7 of 10 patients with multiple infarctions. A cardiac source was found in 5 patients (patients 1, 2, 4, 6, and 10); DWI hyperintense lesions were in different vascular territories in three (patients 1, 6, and 10). An artery-to-artery embolus was suspected in 2 patients (patients 5 and 8); DWI hyperintense lesions were in the same vascular territory distal to the vascular lesion. Two remaining patients (patients 7 and 9) with pontine lesions had only minimal atheromatous disease in the carotid bifurcation. The final patient had normal cardiac and vascular investigations but had a urine test positive for cocaine, which raised the suspicion of a cocaine-associated vasculopathy. Though impossible to validate, it is also possible that a subset of the lacunar infarctions in the single-lesion group nevertheless might have been caused by embolism.

Multiple hyperintense lesions on DWI, located in different vascular territories, occurring simultaneously, or closely related in time, strongly indicate an embolic stroke mechanism. However, whether DWI lesions occur due to showers of multiple emboli or recurrent emboli linked closely in time remains unknown. Other theoretical possibilities include bilateral or unilateral watershed infarctions, or diffuse thrombotic or inflammatory processes (such as thrombotic thombocytopenic purpura, granulomatous angiitis, antecedicaloplon syndrome) that lead to multiple small-vessel occlusions within a short period of time. However, only 1 of our patients was thus classified (patient 3, who may have had a cocaine-related stroke). It is also conceivable that multiple infarctions in diverse penetrator territories (such as in patients 4, 6, and 10) may have occurred from simultaneous microatheromatous or lipohyalinotic occlusion of these vessels. However, this explanation could not account for the temporally linked occurrence of both penetrator and leptomeningeal territory infarcts (7 of 10 patients). To remain all-inclusive, the possibility of both a lacunar infarction due to intrinsic small-vessel disease and a cortical infarction due to embolism, occurring at different times but still within the 7-to-14-day time frame of DWI hyperintensity, should also be considered.

Multiple hyperintense lesions on DWI are not an uncommon finding. Within the time frame of this study, among 443 patients with infarction on DWI, 43 patients (9.7%) had multiple DWI hyperintense lesions located within different vascular territories (bihemispheric or involving both anterior and posterior circulations). Although hyperintense lesions in the perforating vessel territories were also seen in most, only 4 of 43 patients presented with a classic lacunar syndrome. There is currently no clinical or laboratory means to conclusively identify embolism as the cause of infarction in patients presenting with a classic lacunar syndrome. One approach depends on finding a potential source of emboli. A number of studies reported variable rates of detection of embolic sources, however, almost all used different investigative batteries, which may have influenced the likelihood of establishing a stroke mechanism. Clinically significant extracranial atherosclerosis or major cardioembolic sources were present in some patients with lacunar infarction, but approximately 2 to 3 times less frequently than among patients with cortical infarctions. Nevertheless, demonstration of an embolic source per se is not sufficient to establish a causal relationship between a clinical lacunar syndrome and a particular cardiac or arterial abnormality.

In common clinical practice, patients who present to the emergency room with a classic lacunar syndrome (as opposed to presentation with symptoms or signs suggestive of a cortical infarction) often receive the diagnosis of intrinsic small-vessel occlusive disease, with high probability of excellent recovery and recommendation for antiplatelet agents for secondary prevention. In such patients, low priority may be placed on extensive cardiac or arterial testing for other causes of stroke. Our data, in concert with that of other published studies, indicate that—although small—there is a definite risk of a lacunar syndrome occurring from an embolus. It is not possible to identify such patients prospectively based only on clinical features at presentation (eg, demographic features, clinical syndrome, mode of onset, and symptom progression). The use of DWI to uncover subsidiary infarctions in patients with lacunar syndromes should prompt the physician to search for an underlying embolic source and tailor a secondary stroke prevention strategy to treat the underlying cause.

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