What Causes False Clinical Prediction of Small Deep Infarcts?

Jan Lodder, MD; John Bamford, MD; Jaap Kappelle, MD; Jelis Boiten, MD

Background and Purpose Our goal was to identify factors that play a role in false clinical diagnosis of small deep infarcts.

Methods In 350 prospectively registered patients with a first supratentorial ischemic stroke, we clinically differentiated between lacunar and nonlacunar syndromes. Using computed tomography (CT), we distinguished small deep and territorial infarcts and also recorded leukoaraiosis and asymptomatic infarcts. Degree of initial handicap, potential source of cardioembolic stroke, and hypertension were also noted.

Results One hundred forty-seven patients had a lacunar and 203 a nonlacunar syndrome. Forty-two (12%) had a lesion visualized by CT that was compatible with a recent infarct but was considered inappropriate for the clinical syndrome: nineteen had a nonlacunar syndrome but a small deep infarct, and 23 had a lacunar syndrome but a territorial infarct. Patients with a nonlacunar syndrome but a small deep infarct were more severely disabled (a modified Rankin scale rating of 5) (odds ratio [OR], 4.31; 95% confidence interval [CI], 1.25 to 14.88) and had a cardioembolic source (OR, 4.07; 95% CI, 1.04 to 15.95), leukoaraiosis (OR, 3.79; 95% CI, 1.32 to 10.05), or asymptomatic infarcts visualized by CT (OR, 4.13; 95% CI, 1.45 to 11.71) compared with 124 patients with a correctly diagnosed small deep infarct. Twelve of 19 patients with a nonlacunar syndrome but a small deep infarct had a lesion in the left hemisphere, and 9 of these 12 had "aphasia." Patients with a lacunar syndrome but a territorial infarct more often had a cardioembolic source (OR, 4.02; 95% CI, 1.15 to 14.03) and a pure motor syndrome (OR, 4.52; 95% CI, 1.55 to 13.18) than those with lacunar syndrome but a small deep infarct, although 21 (91%) were in the right hemisphere. Of the first 103 patients with lacunar stroke diagnosed by two of the study neurologists, 5 had an inappropriate lesion compared with 14 of the later 40 diagnosed by colleagues without a specific interest in cerebrovascular diseases (OR, 0.09; 95% CI, 0.03 to 0.26).

Conclusions (1) Diagnosis of lacunar syndromes should not be influenced by deficit severity or the presence of a potential cardiac source of embolism. (2) Speech disorders should carefully be influenced by deficit severity or the presence of a potential cardiac source of embolism. (3) Routine tests of nondominant higher functions may be inadequate. (4) Doctors interested in cerebrovascular neurology have a lower failure rate in differentiating small deep infarcts from territorial infarcts than those less well-trained or interested in neurology. (5) Among the lacunar syndromes, pure motor syndrome may be the least specific predictor of a small deep infarct. (Stroke. 1994;25:86-91)

Key Words • cerebral infarction • diagnosis • lacunar infarction
longer than 24 hours were registered prospectively at the University Hospital Maastricht, as described elsewhere. Patients with signs or symptoms that indicated a brain stem location of the lesion were excluded, mainly because of low CT positivity and reliability caused by frequent artifacts in this region, whereas some symptoms that point to a possible brain stem location, eg, vertigo, may be difficult to differentiate from nonvascular causes. Routine investigations included standard blood and urine tests, electrocardiogram, chest roentgenogram, and CT. Echocardiography, 24-hour electrocardiographic monitoring, and cerebral angiography were performed in selected cases.

Brain infarction was defined as rapidly developing clinical signs of focal disturbance of cerebral function, lasting longer than 24 hours or leading to death, with no apparent cause other than that of vascular origin; CT in these patients was normal or showed an area of low attenuation compatible with the clinical symptoms and signs, or autopsy showed a relevant infarct. When neither CT nor autopsy were available we used the Guy's Hospital Diagnostic Score, which predicts with a greater than 90% probability that the stroke was caused by infarction if the score is below 4.16

On admission, patients had detailed clinical examination, and the distinction of whether the clinical syndrome was lacunar or nonlacunar was made before a CT diagnosis was available. We distinguished the following cases as lacunar syndromes: pure motor syndrome (PMS), sensorimotor syndrome (SMS), pure sensory syndrome (PSS), and dysarthria-clumsy hand syndrome/ataxic hemiparesis (DCHS/AH), as defined elsewhere12 (see Appendix 1). A nonlacunar syndrome was recognized when there were signs of dysphasia, visual field deficit, visuospatial problems, neglect, apraxia, or forced gaze alone or together with unilateral motor or sensory deficit. A small deep infarct was defined as a subcortical, sharply margined hypodense lesion with a diameter of less than 20 mm on CT. A territorial infarct was defined as a hypodense lesion compatible with a territory supplied by the main stem or the cortical or medullary branches of one of the three large cerebral arteries. Large subcortical lesions were included in the latter group because of similar pathogenesis.17-19 A lesion visualized by CT was considered "compatible" if located in the hemisphere the signs and symptoms originated from, whereas the radiologically estimated age was consistent with the time of stroke (old lesions being more hypodense, more sharply delineated, or showing signs of retraction of brain structures toward the lesion site).

Territorial infarcts were divided into three groups by presumed cause: cardioembolic, atherothrombotic, and a separate category of "rare" causes. Territorial infarcts were considered to be caused by cardioembolic embolism in the presence of one or more potential cardiac embolic sources: chronic or intermittent atrial fibrillation, left ventricular myocardial infarction less than 6 weeks before stroke, prosthetic aortic or mitral valve, endocarditis, cardiomyopathy, mitral stenosis, left ventricular aneurysm, and left ventricular thrombus. Territorial infarcts were considered to be caused by large-vessel atherothrombosis in the absence of any specific cause such as a potential cardiac source of embolism, with or without significant carotid lesions revealed by noninvasive testing. No effort was made to distinguish between in situ thrombosis and artery-to-artery embolism. Rare causes included vasculitis, arterial dissection, fibromuscular dysplasia, and hematological disorders. At presentation, disability was measured by the modified Rankin scale, in which Rankin 5 indicates "severely disabled."

Leukoaraiosis was defined as focal or diffuse hypodensities in the periventricular or deep white matter not involving the cortex and not compatible with territorial infarction.21 Although we noted different sites of leukoaraiosis (around the frontal or occipital horn, the centrum semiovale, or combinations of these), in the present study only the presence of leukoaraiosis regardless of its extension was analyzed. CT scans were independently reviewed by two neuroradiologists with specific interest in cerebrovascular disorders without knowledge of clinical data. If there was disagreement about the presence of an infarct or leukoaraiosis, the CT was scored as negative for that particular item. The size of the territorial infarct was scored as "large" if the entire area supplied by the anterior or posterior cerebral artery was involved or the complete or larger part of the middle cerebral artery; "moderate" when a larger branch area was involved; and "small" when the lesion affected a small branch area. Large subcortical infarcts were always scored as moderate-sized.

We compared the following four groups: patients with a nonlacunar syndrome but a small deep infarct on CT (NLSSDI), those with a lacunar syndrome but a territorial infarct on CT (LSTI), patients with a lacunar syndrome and a small deep infarct or no specific lesion on CT (LSSI), and those with a nonlacunar syndrome and a territorial infarct or no specific lesion on CT (NLSTI). The first 252 patients of the study population were all seen by one of the two Maastricht authors, who have previously reported the positive and negative predictive values of the lacunar syndromes in diagnosing small deep infarcts; in the later 98 patients, the clinical information was mainly obtained by junior doctors or sometimes by staff neuroradiologists without a specific interest in cerebrovascular disease. This allowed us to study the effect of experience on the accuracy of diagnosing the infarct type from the clinical syndrome. Differences between groups were analyzed using odds ratios (ORs) with a 95% confidence interval (CI) and χ2 analysis, both with Yates’ correction.22

Results

Three hundred seventy-three patients were registered with a first-ever supratentorial brain infarct, of whom 12 had "rare" stroke causes and were therefore excluded from the present study. Eleven patients with a nonlacunar syndrome who did not undergo CT scanning were also excluded. Thus, 350 patients remained for the study. One hundred forty-seven (42%) had a lacunar syndrome and 203 (58%) a nonlacunar syndrome. Forty-two (12%) had a lesion visualized by CT that, although compatible with a recent infarct, was considered completely inappropriate for the clinical syndrome. Table 1 depicts the sensitivity and specificity, positive and negative predictive values, and the overall accuracy of the lacunar syndromes as a clinical test. Table 2 shows the clinical and radiological findings in the four groups. Table 3 shows the ORs with 95% CI resulting from the comparison between groups. In 52 of the 143 patients with a small deep infarct (36%; 95% CI, 28 to 44), and in 37 of the 207 patients with territorial infarct (18%; 95% CI, 13 to 23), no presumed symptomatic infarct could be visualized. CT was performed at a
TABLE 2. Number of Patients With Different Clinical and CT Features

<table>
<thead>
<tr>
<th></th>
<th>NLSSDI (n=19)</th>
<th>LSTI (n=23)</th>
<th>LSSDI (n=124)</th>
<th>NLSTI (n=184)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean), y</td>
<td>72</td>
<td>66</td>
<td>67</td>
<td>72</td>
</tr>
<tr>
<td>Males</td>
<td>10 (53)</td>
<td>12 (52)</td>
<td>68 (55)</td>
<td>105 (57)</td>
</tr>
<tr>
<td>Rankin 5</td>
<td>6 (32)</td>
<td>5 (22)</td>
<td>12 (10)</td>
<td>88 (48)</td>
</tr>
<tr>
<td>Cardiac embolism</td>
<td>5 (26)</td>
<td>6 (26)</td>
<td>10 (8)</td>
<td>75 (41)</td>
</tr>
<tr>
<td>Right hemisphere</td>
<td>7 (37)</td>
<td>21 (91)</td>
<td>64 (52)</td>
<td>77 (42)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>11 (58)</td>
<td>10 (43)</td>
<td>60 (48)</td>
<td>84 (48)</td>
</tr>
<tr>
<td>PMS</td>
<td>...</td>
<td>18 (78)</td>
<td>55 (44)</td>
<td>...</td>
</tr>
<tr>
<td>SMS</td>
<td>...</td>
<td>5 (22)</td>
<td>42 (34)</td>
<td>...</td>
</tr>
<tr>
<td>DCHS/AH</td>
<td>...</td>
<td>0</td>
<td>23 (19)</td>
<td>...</td>
</tr>
<tr>
<td>PSS</td>
<td>...</td>
<td>0</td>
<td>3 (2)</td>
<td>...</td>
</tr>
<tr>
<td>Territorial infarct size</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large</td>
<td>...</td>
<td>0</td>
<td>42 (23)</td>
<td>...</td>
</tr>
<tr>
<td>Moderate</td>
<td>...</td>
<td>7 (30)</td>
<td>80 (44)</td>
<td>...</td>
</tr>
<tr>
<td>Small</td>
<td>...</td>
<td>5 (22)</td>
<td>16 (9)</td>
<td>...</td>
</tr>
<tr>
<td>Large subcortical</td>
<td>...</td>
<td>11 (48)</td>
<td>...</td>
<td>9 (5)</td>
</tr>
<tr>
<td>Leukoaraiosis (any)</td>
<td>11 (58)</td>
<td>5 (22)</td>
<td>33 (27)</td>
<td>39 (21)</td>
</tr>
<tr>
<td>Asymptomatic infarcts (any)</td>
<td>11 (58)</td>
<td>7 (30)</td>
<td>31 (25)</td>
<td>58 (32)</td>
</tr>
</tbody>
</table>

Percentages are shown in parentheses. NLSSDI indicates nonlacunar syndrome but a small deep infarct; LSTI, lacunar syndrome but territorial infarct; LSSDI, lacunar syndrome with small deep infarct; NLSTI, nonlacunar syndrome with a territorial infarct; PMS, pure motor syndrome; SMS, sensorimotor syndrome; DCHS/AH, dysarthria-clumsy hand syndrome/ataxic hemiparesis; and PSS, pure sensory stroke.

median of 5 (range, 0 to 201) days after the onset of stroke. Sixty-five percent of the patients had CT within 1, 85% within 2, and 91% within 3 weeks after stroke. There was no statistically significant difference in the timing of CT between falsely and correctly diagnosed infarct subgroups.

Patients with NLSSDI more often were severely disabled (Rankin 5) and had a potential cardiac source of embolism, leukoaraiosis, or one or more asymptomatic infarcts on CT when compared with correctly diagnosed small deep infarcts (LSSDI). Twelve of these 19 patients with NLSSDI had infarction in the left hemisphere; 9 of these 12 were diagnosed as having some degree of aphasia on clinical examination. In 3 patients the small deep infarct was located in the head of the caudate nucleus (2 on the right side), and in the left thalamus in 1 patient. The factors in patients with NLSSDI resembled those in patients with correctly diagnosed territorial infarcts (NLSTI), except that they had leukoaraiosis significantly more often, and one or more asymptomatic infarcts (Table 3). Patients with LSTI differed from those with correctly diagnosed small deep infarcts (LSSDI) in that they had a potential source of cardiogenic embolism significantly more often.

TABLE 3. Comparison of Different Infarct Subgroups by Odds Ratio

<table>
<thead>
<tr>
<th></th>
<th>NLSSDI Versus</th>
<th>LSTI Versus</th>
<th>NLSTI Versus</th>
<th>LSTI Versus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male:Female</td>
<td>0.92 (0.07-12.07)</td>
<td>0.84 (0.05-12.95)</td>
<td>0.90 (0.37-2.19)</td>
<td>0.82 (0.14-4.65)</td>
</tr>
<tr>
<td>Rankin 5</td>
<td>4.31 (1.25-14.88)</td>
<td>0.50 (0.15-1.69)</td>
<td>2.59 (0.62-10.83)</td>
<td>0.30 (0.10-0.90)</td>
</tr>
<tr>
<td>Cardiac embolism</td>
<td>4.07 (1.04-15.95)</td>
<td>0.52 (0.14-1.93)</td>
<td>4.02 (1.15-14.03)</td>
<td>0.48 (0.16-1.49)</td>
</tr>
<tr>
<td>Right hemisphere</td>
<td>0.55 (0.16-1.89)</td>
<td>0.81 (0.08-8.27)</td>
<td>9.48 (2.54-38.12)</td>
<td>14.59 (4.25-50.12)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.47 (0.55-3.88)</td>
<td>1.64 (0.47-5.68)</td>
<td>0.82 (0.12-5.41)</td>
<td>0.92 (0.38-2.19)</td>
</tr>
<tr>
<td>Leukoaraiosis</td>
<td>3.79 (1.32-10.05)</td>
<td>5.11 (1.91-13.65)</td>
<td>0.77 (0.08-7.35)</td>
<td>1.03 (0.76-1.39)</td>
</tr>
<tr>
<td>Asymptomatic infarct (any)</td>
<td>4.13 (1.45-11.71)</td>
<td>3.41 (1.18-9.87)</td>
<td>1.31 (0.21-8.38)</td>
<td>0.95 (0.45-2.02)</td>
</tr>
<tr>
<td>PMS</td>
<td>...</td>
<td>...</td>
<td>4.52 (1.55-13.18)</td>
<td>...</td>
</tr>
<tr>
<td>SMS</td>
<td>...</td>
<td>...</td>
<td>0.54 (0.14-2.05)</td>
<td>...</td>
</tr>
</tbody>
</table>

95% confidence interval in parentheses. An odds ratio greater than 1 indicates more frequent in NLSSDI or LSTI; confidence interval lower limit greater than 1 or upper limit less than 1 indicates difference with P<.05.

NLSSDI indicates nonlacunar syndrome but small deep infarct; LSTI, lacunar syndrome but territorial infarct; LSSDI, lacunar syndrome with small deep infarct; NLSTI, nonlacunar syndrome with territorial infarct; PMS, pure motor syndrome; and SMS, sensorimotor syndrome.
and a clinical syndrome of PMS. Twenty-one of the 23 were located in the right hemisphere. Fewer LSTI patients had an initial Rankin grade 5 than those with NLSTI. Of the 73 patients with a diagnosed PMS syndrome, 18 (25%; 95% CI, 15 to 35) had a territorial infarct visualized by CT (Table 2). Five of the 47 patients with SMS had a territorial infarct (11%; 95% CI, 2 to 20) compared with none of the 23 DCHS/AH cases. Eleven of the 23 LSTI patients presented with a large subcortical infarct versus 9 of the 184 NLSTI patients. Patients with NLSTI more often had an infarct of large size than patients with LSTI (OR, 8.80; 95% CI, 1.30 to 59.66). Of the first 149 patients with a territorial infarct diagnosed by JL or J Boiten in the first part of the study, 11 had a lacunar syndrome compared with 12 of the 58 examined by others (OR, 0.31; 95% CI, 0.12 to 0.78; \( P < 0.02 \)), whereas of the first 103 patients with a small deep infarct diagnosed by JL and J Boiten, 5 did not have a lacunar syndrome compared with 14 of the subsequent 40 examined by others (OR, 0.09; 95% CI, 0.03 to 0.26; \( P < 0.001 \)).

**Discussion**

In approximately 1 of 10 cases with a first symptomatic brain infarct, the clinical syndrome did not predict the correct infarct type in our series. The mistake rate was similar for lacunar syndromes in predicting small deep infarcts and for nonlacunar syndromes in predicting territorial infarcts. Theoretically, the mistake rate may have been higher because in both groups a number of infarcts were not visualized by CT. The timing of CT may be relevant for the infarct detection rate. Some lesions may not be detected by early CT. CT in our study was done at a median of 5 days after the onset of stroke, whereas 45% of our patients underwent CT later than 1 week and most of them (91%) within 3 weeks after the onset of stroke. There was no difference in the timing of CT between falsely and correctly diagnosed infarct subgroups; therefore, bias in this respect is unlikely. Of the first 109 patients with small deep infarcts we were able to repeat CT in 26 of 46 with an initial negative CT. Five of these 26 had small deep infarcts. No territorial infarcts were detected, so it is unlikely that most cases with a negative early CT are in fact undetected territorial infarcts. In addition, MRI studies show that most of CT-negative cases with a lacunar syndrome had a small deep lesion, often located in the brain stem.\(^{13,23,24}\) Therefore, it is unlikely that in CT-negative cases the infarct type is more often incompatible with small-vascular vasculopathy than in those in whom the lesion is visualized by CT. However, not all of our patients with normal CT may have had supratentorial infarction, which was the aim of our study. Because we excluded patients with obvious brain stem signs, the infarcts located in this region were most probably small deep (lacunar) infarcts and are clinically similar to those located in the basal ganglia.\(^4\) Most clinicians faced with a case of lacunar syndrome would assume it to be a “carotid distribution” event and treat the patient accordingly. Therefore, from a pragmatic viewpoint it seems reasonable to include such CT-negative cases in a study such as the present one. Nonetheless, the overall accuracy of the lacunar syndromes in predicting a small deep infarct was 88% (95% CI, 85 to 91), which supports the lacunar hypothesis.

Patients with NLSSDs were more often severely disabled (Rankin 5) and had more often a potential source of cardiogenic embolism compared with LSSDs patients but were similar in this respect to NLSTI patients. This suggests that these two factors, which may more readily be associated with territorial rather than small deep infarction, influenced the doctors’ decision in classifying the syndrome as nonlacunar. However, patients with LSTI had an even higher frequency of a potential source of cardiogenic embolism than LSSD patients, but this did not cause the doctors to consider a nonlacunar rather than a lacunar syndrome. A lesser degree of deficit in the LSTI compared with NLSTI patients may also have directed the diagnosis toward lacunar syndrome.

Twelve of the 19 NLSSDs were located in the left hemisphere, and 9 of these 12 had some type of speech disturbance that was viewed as a sign of “cortical” dysfunction. Subcortical lesions sometimes produce dysphasia\(^{25–27}\) or other cortical signs such as denial of handicap,\(^{28}\) neglect,\(^{29,30}\) affective and mnemonic disturbances,\(^{31,32}\) or frontal lobe dysfunction.\(^{33}\) Our patients’ speaking difficulties may have been labeled dysphasia simply because the lesion was located in the left hemisphere, because only 4 of these 9 NLSSD cases were located in the areas associated with dysphasia, such as the thalamus or the caudate nucleus. The higher frequency of leukoaraiosis and asymptomatic infarcts in the NLSSD patients may also have contributed to the appearance of cortical symptoms; when the brain is already lesioned by leukoaraiosis or prior asymptomatic infarction, a small deep infarct possibly elicits more extensive functional disturbances than those caused by the small deep infarct alone. Matsubayashi et al\(^ {34} \) argued that neurobehavioral function disturbances may be provoked in this way.

In 21 of the 23 patients with an LSTI, the infarct was located in the right hemisphere. The major reason for this misdiagnosis is probably doctors’ neglect of right-hemispheric cortical symptoms, which are less easily detected by the routine neurological examination, probably because of the absence of language dysfunction as a discriminator between territorial and small deep infarcts in right-hemispheric stroke. This problem has been recognized previously in a study of the “lacunar history.”\(^ {35}\) Clinical discrimination between territorial and small deep infarcts might be improved by adding a standard battery for right-hemispheric cortical dysfunction to the routine neurological examination. Another cause of error was that 11 of the 23 LSTI cases had large deep territorial (striatocapsular) infarcts, which, because of their usual pathogenesis, were classified as territorial.\(^ {17,18,19}\) Such infarcts have also presented with lacunar syndromes in other series.\(^ {36,37}\)

DCHS/AH syndrome was a more accurate predictor of a small deep infarct than some suggest.\(^ {38}\) We agree with others who have found that SMS is more likely to be related to small deep than territorial infarction.\(^ {1,39,40}\) Of all four lacunar syndromes PMS had the lowest association with a small deep infarct. This contrasts with the view of others who considered PMS the prototype lacunar syndrome and argued that it should be studied as a separate group but concurs with the findings of Chimowitz et al.\(^ {14,36,41,44}\) The inclusion of patients with a monoparesis in the PMS syndrome group, as done by others, cannot be the reason that some of our patients...
with PMS had a territorial lesion visualized by CT because we classified monoparesis as a cortical syndrome; however, the “two out of three” rule in PMS as we defined it may be less accurate than we thought and may need to be evaluated more precisely.\textsuperscript{12,44-46} Bogousslavsky et al\textsuperscript{14} initially found a cerebral hemorrhage or a territorial infarct in two thirds of PMS cases, whereas 17% of all patients with hemorrhages in their series presented with PMS.\textsuperscript{14} However, they studied only PMS and HSS with a lesion visible on CT. Considering these findings together with our data suggests that leaving out SMS and DCHS/AH when studying the relation between lacunar syndromes and infarcts biases the results toward unfavorable correlations. In a later study from the Lausanne Stroke Registry, a PMS was caused by hemorrhage in only 9 of 255 patients.\textsuperscript{46}

The two Maastricht investigators, whose aim was to establish the validity of lacunar syndromes, made fewer mistakes in the earlier part of the present study than their less-experienced junior colleagues or less-interested staff members in the later part.\textsuperscript{12} Obviously, consensus on what is meant by certain clinical syndromes and practical training may improve a clinician’s chance of correctly predicting the type of infarct from the signs and symptoms.

Numerous examples of brain lesions other than small deep infarcts have been used to argue that predicting such infarcts from certain clinical syndromes is fallacious. However, these “examples of proof” against a clinico-pathological correlation in small deep infarcts are neurological rarities rather than the results of well-defined prospective series. The fact that not all small deep infarcts would result in a lacunar syndrome and vice versa\textsuperscript{11} is not surprising and does not disagree with Fisher’s original suggestion that lacunar infarcts can usually be recognized from certain clinical syndromes. Involvement of the basal ganglia might result in what we usually consider “cortical” symptoms. However, deeply located lesions in specific sites may provoke such symptoms.\textsuperscript{25-29} Some investigators presume that lowered distant cortical blood flow is the underlying cause of these symptoms in such lesions.\textsuperscript{30,33,47-49} Generally, an interruption in various neural pathways is thought to be responsible for the cortical dysfunction.\textsuperscript{26,30-33,48,49} Whether a lowered cortical perfusion is the cause or a mere epiphenomenon of interrupted neuronal cortical projections is unclear. However, if it is an experienced or interested clinician who makes the distinction between lacunar or nonlacunar syndrome, the prediction is fairly accurate. Different recent series showed that both positive and negative predictive values of the lacunar syndromes are in such a range that they can be considered excellent clinical tests.\textsuperscript{2,12,50}

Some factors emerge from our data that promote the clinical misdiagnosis of different infarct subtypes. When examining a stroke patient, the physician should realize that it is the clinical features making up the clinical syndromes and not associated pathologies (such as the presence of a cardioembolic source) that should be used to predict the CT findings. If a lacunar syndrome is diagnosed on the basis of a pure left motor hemiparesis, the examiner should seek evidence of nondominant hemisphere cortical dysfunction such as visuospatial perceptual disturbances, sensory and visual inattention, and discriminatory sensory loss. We value accurate differentiation of brain infarct subtypes by clinical diagnosis, because CT fails to visualize a rather substantial number of both small deep and, although to a lesser degree, territorial infarcts, whereas the differentiation of infarct subtypes is valuable for both clinical management and in clinical trials. With increasing interest in stroke treatments in the acute phase, classifications based on imaging “holes in the brain” are likely to become less relevant. Clinical skills should not be discarded on the altar of imaging technology.

**Appendix 1**

**Definition of Lacunar Syndromes**\textsuperscript{1}

A constellation of clinical symptoms and signs are present at the time of maximal deficit after a single cerebrovascular event. The presence of a visual field defect, evidence of higher cerebral dysfunction (eg, dysphasia, visuospatial disturbance, predominantly proprioceptive sensory loss) during standard clinical testing, or features that clearly localize the lesion in the vertebrobasilar distribution (eg, gaze palsies or crossed deficits, though not nystagmus or dysarthria) exclude the diagnosis of lacunar syndrome.

**Pure Motor Syndrome**

A unilateral, pure motor deficit involving at least two of three areas (face, arm, or leg) and affecting the whole limb in patients with brachiofacial or brachiocrural weakness. Sensory symptoms may be present at the time of onset, but there should not be any objective sensory loss on standard clinical testing.

**Pure Sensory Syndrome**

A sensory deficit (which may be diagnosed even when there is no objective sensory loss on standard clinical testing) involving at least two of three areas (face, arm, or leg). In patients with brachiofacial and brachiocrural symptoms, the whole limb should be affected. The sensory deficit may include all modalities equally or may spare proprioception.

**Ataxic Hemiparesis**

A syndrome of ipsilateral corticospinal and cerebellar-like dysfunction without other features that clearly localize to the posterior circulation. This includes cases with predominantly dysarthria and clumsiness of the hand (dysarthria–clumsy hand syndrome).

**Sensorimotor Syndrome**

A syndrome of ipsilateral motor and objective sensory loss, involving at least two of three areas (face, arm, or leg). In patients with brachiofacial or brachiocrural deficit, the whole limb must be involved. The sensory deficit may involve all modalities equally or may spare proprioception.

**References**


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