Pure Motor Hemiparesis and Sensorimotor Stroke

Accuracy of Very Early Clinical Diagnosis of Lacunar Strokes

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Background and Purpose Clinical differentiation of lacunar from nonlacunar strokes in the very early phase could help to exclude patients with lacunar stroke from pharmacologic trials designed for nonlacunar strokes, namely, those with thrombolytic agents. In a continuous series of acute ischemic stroke patients, we evaluated how accurately a clinical diagnosis of pure motor hemiparesis or sensorimotor stroke formulated in the first hours from onset predicts a lacunar stroke documented by cerebral computed tomography or by autopsy.

Methods We examined 517 patients (299 men, 218 women; mean±SD age, 67±10 years) within 12 hours (mean±SD, 6.1±3.2 hours) of the event. At hospital admission, we observed 151 (29%) patients with pure motor hemiparesis and 68 (13%) patients with sensorimotor stroke.

Results Computed tomography or autopsy was compatible with a lacunar stroke (ie, detection of a lacune or permanent negative computed tomography) in 170 (33%) patients, of whom 123 (72%) had pure motor hemiparesis and 47 (28%) had sensorimotor stroke. This led to a sensitivity of 72%, a specificity of 72%, a positive predictive value of 56%, and a negative predictive value of 84%. Overall positive predictive value of pure motor hemiparesis was 58% (60% for two areas and 58% for three areas involved), and that of sensorimotor stroke was 51% (87% for two areas and 40% for three areas involved). By separately evaluating the sides of lesions, we found a positive predictive value of 46% for right-side infarcts and of 72% for left-side infarcts. Right-side lesions constituted 51% of lesions in lacunar syndrome patients with lacunar stroke, 76% in those with nonlacunar stroke, 19% in nonlacunar syndrome patients with lacunar stroke, and 31% in those with nonlacunar stroke (P<.0001). During the first days of hospital stay we observed a deterioration of 21% of lacunar syndrome patients with nonlacunar stroke and an improvement of 49% of nonlacunar syndrome patients with lacunar stroke, with appearance and disappearance of symptoms of cortical involvement, respectively. The examination of these patients after the occurrence of these clinical changes would have led to a daily increase of the positive predictive value up to a maximum of 66% at day 7.

Conclusions Pure motor hemiparesis and sensorimotor stroke diagnosed within 12 hours of the event are poorly predictive of lacunar strokes. Hence, the very early identification of these syndromes cannot be used for patient selection in therapeutic trials. (Stroke. 1994;25:92-96.)

Key Words • cerebral ischemia • diagnostic imagery • lacunar infarction • pure motor stroke

In continuous series of stroke patients, lacunar strokes constitute approximately 25% of cases.1-7 They have spontaneous benign clinical evolution more frequently than nonlacunar strokes,5,8,9 probably do not share the same pathogenetic mechanisms,10 and require different clinical management.7 Thus, the inclusion of patients with lacunar stroke in large pharmacologic trials, particularly those with thrombolytic agents, could lead to treatment of possibly different targets with the same compound, and this could mask the expected effects of the drug being tested. Therefore, the early clinical identification of lacunar strokes and possibly their exclusion from randomization (or, conversely, their inclusion in specifically designed trials) appear to be of the utmost relevance for a correct evaluation of the efficacy of any kind of treatment.11 This would also apply to daily clinical management.

Different studies report that lacunar syndromes predict from 50% to 95% of lacunar strokes.2,7,12-16 This large variability might be explained by noncomparable groups of patients studied or by the presumable selection bias of noncontinuous series, as suggested by the excess of lacunar strokes sometimes reported.13,15 However, the common feature of all these studies is that the clinical picture was evaluated at the time of its complete expression, ie, 24 hours (and more) after stroke onset, when every clinically tailored therapeutic intervention is pointless.19

In this prospective study of a continuous series of acute ischemic stroke patients hospitalized within 12 hours of clinical onset of the event, we evaluated the accuracy of pure motor hemiparesis (PMH) and sensorimotor stroke (SMS) syndromes in predicting lacunar strokes identified by either cerebral computed tomography (CT) or autopsy.

Subjects and Methods

From January 1984 to December 1991, patients with a first-ever acute supratentorial stroke hospitalized in the emergency unit of the 1st University Hospital of Rome were referred to the Stroke Unit of the Department of Neurological Sciences.
TABLE 1. Clinical Presentation According to Type of Lesion at Cerebral Computed Tomography or Autopsy

<table>
<thead>
<tr>
<th>Clinical Syndrome</th>
<th>Compatible With Lacunar Stroke</th>
<th>Noncompatible With Lacunar Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lacune</td>
<td>Negative CT</td>
</tr>
<tr>
<td>PMH/SMS</td>
<td>31 (6)</td>
<td>92 (18)</td>
</tr>
<tr>
<td>Nonlacunar</td>
<td>11 (2)</td>
<td>36 (7)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>42 (8)</td>
<td>128 (25)</td>
</tr>
</tbody>
</table>

Numbers in parentheses are percentages. CT indicates computed tomography; PMH, pure motor hemiparesis; and SMS, sensorimotor stroke.

Neurological examination at hospital admission allowed us to identify PMH or SMS involving at least two of three areas (face, arm, and leg) without signs or symptoms of cortical involvement (ie, aphasia, neglect, or hemianopia).2 Gaze palsy and consciousness disturbances were also considered signs of cortical involvement because our analysis was restricted to patients affected by supratentorial strokes. Patients with ataxic hemiparesis, dysarthria–clumsy hand syndrome, and pure sensory stroke are not included in this study. Neurological examination was repeated daily during the first 15 days of hospital stay to monitor changes of clinical status. All patients underwent an initial plain CT scan within 48 hours of the event to exclude previous lesions and lesions other than infacts. A second CT scan was performed within 15±2 days. Initially we used a Siemens Siretom 2000 scanner, and after 1986 we used a Siemens-Somatom CR high-resolution scanner. Four-millimeter-thick slices were scanned in the examination of the posterior fossa up to the chiasmatic cistern, and 8-mm slices were used above that level. Patients who died before the second CT scan underwent pathological examination. Site and size of lesions were defined on the second CT scan or by autopsy. We distinguished between (1) lacunar infarctions: subcortical sharply margined infarcts ≤1.5 cm in diameter and permanently negative CT scans (irrespective of clinical presentation) and (2) nonlacunar infarctions: cortical, subcortical (>1.5 cm in diameter), or combined infarcts with partial or total involvement of supratentorial arterial territories.

We evaluated the accuracy of PMH and SMS syndromes in predicting lacunar strokes by computing (1) sensitivity: percentage of PMH and SMS patients with lacunar stroke among all patients with lacunar stroke; (2) specificity: percentage of nonlacunar syndrome patients with nonlacunar stroke among all patients with nonlacunar stroke; (3) positive predictive value: percentage of PMH and SMS patients with lacunar stroke among all patients with PMH and SMS; and (4) negative predictive value: percentage of nonlacunar syndrome patients with nonlacunar stroke among all patients with nonlacunar syndrome. We took account both of clinical presentation at hospital admission and clinical changes that occurred during hospitalization before the second CT scan or autopsy was performed.

Univariate analysis was performed by using the χ² test.

Results

In 8 years of study 627 consecutive acute stroke patients were hospitalized. Among them, 110 (17.5%) were excluded because of intracerebral hemorrhage (n=44; 40%), tumors (n=4; 4%), subtentorial infarcts (n=32; 29%), transient ischemic attacks (n=16; 15%), endarterectomy (n=8; 7%), and previous lesions on CT scan (n=6; 5%). Globally 517 patients were then prospectively studied (299 men, 218 women; mean±SD age, 67±10 years). Admission neurological evaluation, performed within 6.1±3.2 hours of stroke (mean±SD), provided evidence of 151 (29%) PMH and 68 (13%) SMS patients, whereas the remaining 298 (58%) subjects presented nonlacunar syndromes.

Site and size of lesions were defined on the second CT scan for 465 patients and by autopsy in the remaining 52 individuals. In total, CT scan or autopsy was compatible with a lacunar stroke in 170 (33%) patients and noncompatible with a lacunar stroke in the remaining 347 (67%) patients. Of the 170 patients with lacunar stroke, 123 (72%) had presented PMH or SMS and 47 (28%) a nonlacunar syndrome (Table 1). Therefore, we had a sensitivity of 72% (confidence interval [CI], 65% to 79%), a specificity of 72% (CI, 67% to 77%), a positive predictive value of 56% (CI, 49% to 67%), and a negative predictive value of 84% (CI, 80% to 88%).

Table 2 shows the pattern of neurological deficit among patients with PMH and SMS. The positive predictive value was 58% (CI, 49% to 67%) for PMS patients with three areas affected and 60% (CI, 46% to 74%) for those with two areas involved, as opposed to 40% (CI, 27% to 53%) and 87% (71% to 100%), respectively, for SMS patients. Overall the positive predictive values of PMH and SMS were 58% (CI, 50% to 66%) and 51% (CI, 39% to 63%), respectively.

By separately examining the sides of lesions (Table 3), the positive predictive value of PMH and SMS fell to 46% (CI, 38% to 54%) for right-side infarcts and

TABLE 2. Pattern of Neurological Deficit Among Patients With Pure Motor Hemiparesis and Sensorimotor Stroke According to Type of Lesion at Cerebral Computed Tomography or Autopsy

<table>
<thead>
<tr>
<th>Clinical Syndrome</th>
<th>Compatible With Lacunar Stroke</th>
<th>Noncompatible With Lacunar Stroke</th>
<th>Positive Predictive Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PMH</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Areas</td>
<td>28</td>
<td>19</td>
<td>28/47=60%</td>
</tr>
<tr>
<td>3 Areas</td>
<td>60</td>
<td>44</td>
<td>60/104=58%</td>
</tr>
<tr>
<td>Total</td>
<td>88</td>
<td>63</td>
<td>88/151=58%</td>
</tr>
<tr>
<td>SMS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Areas</td>
<td>14</td>
<td>2</td>
<td>14/16=87%</td>
</tr>
<tr>
<td>3 Areas</td>
<td>21</td>
<td>31</td>
<td>21/52=40%</td>
</tr>
<tr>
<td>Total</td>
<td>35</td>
<td>33</td>
<td>35/68=51%</td>
</tr>
</tbody>
</table>

PMH indicates pure motor hemiparesis; SMS, sensorimotor stroke.
reached 72% (CI, 62% to 82%) for left-side infarcts. Patients with right-side infarcts accounted for 51% (63/123) of PMH/SMS patients with lacunar strokes, 76% (73/96) of PMH/SMS patients with nonlacunar strokes, 19% (9/47) of nonlacunar syndrome patients with lacunar strokes, and 31% (79/251) of nonlacunar syndrome patients with nonlacunar strokes (P < .0001).

Among the 219 PMH/SMS patients, 43 (20%) presented a clinical deterioration during hospitalization. Of them, 23 (19%) subjects belonged to the group of 123 patients with lacunar strokes and showed an impairment of motor or sensorimotor deficit, whereas 20 (21%) individuals belonged to the group of 96 patients with nonlacunar strokes and developed speech deficits (n = 6) or consciousness disturbances (n = 14) (Table 1). Had we examined these latter patients after deterioration, we would have allocated them to the group of nonlacunar syndrome patients with nonlacunar strokes, with an increase of positive predictive value to 62% (123/199) (CI, 55% to 69%).

In the group of 47 nonlacunar syndrome patients with lacunar strokes, signs and symptoms of nonlacunar lesion were represented by aphasia (motor, n = 22; receptive, n = 2; global, n = 10), neglect (n = 2), hemianopia (n = 3), and involvement of only one area (n = 8). CT scan was permanently negative in 36 cases. By excluding these patients from analysis, we would have obtained a sensitivity of 92% (123/134) (CI, 85% to 95%) and a negative predictive value of 96% (251/262) (CI, 94% to 98%), whereas the positive predictive value would not have been modified. Of the total 47 patients, 23 (49%) improved in the following days, and their cortical signs and symptoms cleared (15 cases of motor aphasia, 2 cases of receptive aphasia, 5 cases of global aphasia, and 1 case of hemianopia). Thus, had we examined these patients after improvement we would have classified them as PMH or SMS syndromes with lacunar strokes, with an increase of sensitivity to 86% (146/170) (CI, 81% to 91%), of negative predictive value to 91% (251/275) (CI, 88% to 94%), and of positive predictive value to 60% (146/242) (CI, 54% to 66%).

Finally, by cumulatively taking into account all the mentioned clinical changes (ie, deterioration of PMH/SMS patients with nonlacunar strokes and improvement of nonlacunar syndrome patients with lacunar strokes), the positive predictive value would have presented a daily increase up to a maximum of 66% (146/222) (CI, 60% to 72%) at day 7 (Figure).

![Bar graph shows changes of positive predictive value of pure motor hemiparesis (PMH) and sensorimotor stroke (SMS) syndromes from hospital admission to day 7 of hospital stay, taking into account the improvement of 49% (23/47) of nonlacunar syndrome patients with lacunar strokes (with disappearance of cortical signs and symptoms) and the deterioration of 21% (20/123) of PMH/SMS patients with nonlacunar strokes (with appearance of signs and symptoms of cortical involvement).](#)

### Table 3. Clinical Presentation According to Type and Side of Lesion

<table>
<thead>
<tr>
<th>Clinical Syndrome</th>
<th>Compatible With Lacunar Stroke</th>
<th>Noncompatible With Lacunar Stroke</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>PMH/SMS</td>
<td>Right 63</td>
<td>Left 60</td>
<td>Right 73</td>
</tr>
<tr>
<td>Nonlacunar</td>
<td>Right 9</td>
<td>Left 38</td>
<td>Right 79</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>72</strong></td>
<td><strong>98</strong></td>
<td><strong>152</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Right side</th>
<th>Left side</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity</td>
<td>63/72=87%</td>
<td>60/98=61%</td>
</tr>
<tr>
<td>Specificity</td>
<td>79/152=52%</td>
<td>173/195=89%</td>
</tr>
<tr>
<td>Positive predictive value</td>
<td>63/136=46%</td>
<td>60/83=72%</td>
</tr>
<tr>
<td>Negative predictive value</td>
<td>79/88=90%</td>
<td>172/210=82%</td>
</tr>
</tbody>
</table>

PMH indicates pure motor hemiparesis; SMS, sensorimotor stroke; and CI, confidence interval.
Discussion

These data on a large consecutive series of patients with first-ever acute ischemic stroke show that PMH and SMS, when observed very early after onset of signs and symptoms, globally predicted lacunar strokes in only 56% of cases. The type of lacunar syndromes included in this study, frequency of right and left side infarcts in the different groups, allocation of patients with permanently negative CT scans, and the very early neurologcal observation after stroke onset may account for our results.

Patients admitted to our Stroke Unit are affected by moderate to severe motor or sensorimotor deficits with or without signs and symptoms of cortical involvement; they represent the typical patients usually enrolled in pharmacologic trials. Therefore, of the five most frequent lacunar syndromes,2 16 ataxic hemiparesis, dysarthria–clumsy hand syndrome, and pure sensory stroke were not considered in this study because they are characterized by mild or nonmotor deficits. From this point of view our study is not directly comparable to previous studies.2 16 Because these clinical features constitute approximately up to 20% of all lacunar syndromes2 16 and show a high predictivity for lacunar strokes,2 13 we may observe that their absence in the present study partially explains the lower accuracy of the clinical diagnosis of lacunar stroke when compared with other studies.2 16

It has recently been proposed that in patients with PMH the assumption of a lacunar etiology should be applied only to those with global involvement of the face, upper limb, and lower limb.19 In our cohort we did not find differences of predictivity among PMH patients with two or three areas involved, even when hypertension was taken into account (data not shown). On the other hand, by applying the same analysis to SMS patients, we found the highest predictivity in those with two areas involved. Nevertheless, we adopted the criteria of Bamford et al2 16 for definition of lacunar syndromes to avoid introducing further discrepancies between our study and previous studies.2 16

In regard to side of lesions, among PMH/SMS patients with nonlacunar strokes we found a preponderance of right-side infarcts compared with the other groups, probably because of the difficulties in identifying signs and symptoms of visuospatial disturbances in hyperacute stroke patients. However, by separately examining patients with right- and left-side infarcts, we observed that up to one third of patients with left-side infarcts can present without patent signs and symptoms of cortical involvement.

Permanently negative CT scans were a factor in 25% of cases, a percentage that is in agreement with the literature.2 19 20 While negative scans of patients with lacunar syndromes are generally considered compatible with a lacunar lesion,2 16 our choice to also extend this assumption to the negative scans of patients with nonlacunar syndromes could be debated. However, had we excluded these cases from analysis, the positive predictive value would not have improved. On the other hand, our decision was supported by the notion that lacunes can underlie cortical signs and symptoms.2 16

The cause could be a functional disconnection between subcortical infarcted areas and their cortical projec-

tions, which was also demonstrated for lesions of lacunar size.2 21 This phenomenon is potentially reversible24 and could also explain the disappearance of cortical signs and symptoms observed during the first days of hospital stay in approximately one half of our nonlacunar syndrome patients with lacunar strokes.

Clinical changes were also observed in one fifth of PMH/SMS patients with nonlacunar strokes, who developed cortical signs and symptoms (ie, speech or consciousness disturbances). This emphasizes the role of the time interval between stroke onset and first neurologic evaluation. In fact, in our cohort the positive predictive value of PMH and SMS ranged from 56% at hospital admission to 66% after the aforementioned clinical changes had occurred.

In conclusion, PMH and SMS are poorly predictive of lacunar strokes14 16 when patients are evaluated within 12 hours of the event. Unfortunately, the first hours after stroke onset are the most decisive hours for current therapeutic attempts.17 Hence, although theoretically desirable, it is practically difficult to exclude patients with a lacunar infarction from acute ischemic stroke trials such as those with thrombolytic agents. In other words, lacunar infarctions cannot be an exclusion criterion in clinical trials simply because they cannot be reliably diagnosed on clinical grounds early after stroke onset. A post hoc search for lacunar strokes, combining clinical description and CT scan or autopsy data, and a separate evaluation of their clinical evolution might be more useful to disclose the effects of the drug being tested.

Acknowledgments

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References


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tomography in patients presenting with lacunar syndromes. Stroke. 
tomography in patients with lacunar stroke in the carotid territory. 
14. Ghika J, Bogousslavsky J, Regli F. Infarcts in the territory of the deep 
15. Boiten J, Lodder J. Lacunar infarcts: pathogenesis and validity of 
Etiology of motor or sensory stroke: a prospective study of the 
17. Fieschi C, Argentino C, Lenzi GL, Fantozzi L, Sacchetti ML, Pace 
A, Rasura M, Bastianello S, Borzooi L, Zanette EM, Buttinelli C, 
Giubilei F, Fantano P. Therapeutic window for pharmaceutical 
311-320.
20. Brott T, Marler JR, Olinger CP, Adams HP, Tomsick T, Barsan W, 
Bille R, Eberle R, Hertzberg V, Walker M. Measurements of acute 
cerebral infarction: lesion size by computed tomography. Stroke. 
manifestations of pathologically verified lacunar infarction. Stroke. 
1989;20:990-999.
22. Takano T, Kimura K, Nakamura M, Fukunaga R, Kusunoki M, 
Etsisi H, Matsumoto M, Yoneda S, Abe H. Effect of small deep 
hemispheric infarction on the ipsilateral cortical blood flow in man. 
23. Perani D, Vallar G, Cappa S, Messa C, Fazio F. Aphasia and 
neglect after subcortical stroke: a clinical/cerebral perfusion study. 
Recovery from aphasia and neglect after subcortical stroke: neu-
ropsychological and cerebral perfusion study. J Neurol Neurosurg 
Pure motor hemiparesis and sensorimotor stroke. Accuracy of very early clinical diagnosis of lacunar strokes.
D Toni, R Del Duca, M Fiorelli, M L Sacchetti, S Bastianello, F Giubilei, C Martinazzo and C Argentino

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