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Clinical-Computed Tomographic Correlations of Lacunar Infarction in the Stroke Data Bank

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Lacunar stroke was diagnosed in 337 (26%) of the 1,273 patients with cerebral infarction among the 1,805 total in the Stroke Data Bank. We analyzed the 316 patients with classic lacunar syndromes. Among these, 181 (57%) had pure motor hemiparesis, 63 (20%) sensorimotor syndrome, 33 (10%) ataxic hemiparesis, 21 (7%) pure sensory syndrome, and 18 (6%) dysarthria-clumsy hand syndrome. No striking differences were found among the risk factors for the lacunar subtypes, but differences were found between lacunar stroke as a group and other types of infarcts. Compared to 113 patients with large-vessel atherosclerotic infarction, those with lacunar stroke had fewer previous transient ischemic attacks and strokes. Compared to 246 with cardioembolic infarction, patients with lacunar stroke more frequently had hypertension and diabetes and less frequently had cardiac disease. We found a lesion in 35% of the lacunar stroke patients' computed tomograms, with most lesions located in the internal capsule and corona radiata. The mean infarct volume was greater in patients with pure motor hemiparesis or sensorimotor syndrome than in those with the other lacunar stroke subtypes. In patients with pure motor hemiparesis and infarcts in the posterior limb of the internal capsule, there was a correlation between lesion volume and hemiparesis severity except for the few whose infarct involved the lowest portion of the internal capsule; in these patients severe deficits occurred regardless of lesion volume. Taken together, the computed tomographic correlations with the syndromes of hemiparesis showed only slight support for the classical view of a homunculus in the internal capsule. (*Stroke* 1991;22:175-181)

Lacunes as a distinct form of stroke have been based on a small number of cases clinically characterized by a few distinctive syndromes explained at autopsy by a small, deep infarct caused by a special change in small arteries brought about by the chronic effects of hypertension.¹ The small size and special locations of lacunes seemed to make their syndromes distinctive and subject to clinical diagnosis. Large lesions in the same locations or small lesions in

other locations seemed less likely to produce the syndromes.² Although these principles have been sorely criticized over the years, with few exceptions³ autopsy data have been sparse and most publications have been based on studies of small cohorts in which the patients' brain lesions were imaged by computed tomography (CT) or magnetic resonance imaging (MRI). Failing autopsy data, studies based on brain imaging can at least address the issues of risk factors and reliability of the syndromic correlation for the lesion, even if the underlying vascular pathology remains unknown for these individual cases.

Ours appears to be the largest of such clinical and radiographic series. Heretofore, the Oxfordshire Community Stroke Project⁴ was the largest prospective clinical study, with 515 community-based cases of stroke contacted clinically 4 days after onset. A lacunar syndrome was found in 108 patients, of whom 104 underwent CT. Arboix and Martí-Vilalta⁵ recently reported 227 cases studied in Catalonia but did not describe the timing or extent of the clinical contacts.

Subjects and Methods

The Stroke Data Bank is a prospective observational study begun in 1983, with clinical and labora-

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tory data collected during the acute and follow-up phases in patients hospitalized for acute stroke in institutions where personal observations by the investigators were possible. This collaborative study involved the Biometry and Field Studies Branch of the National Institute of Neurological Disorders and Stroke as the statistical coordinating center and four academic centers: University Hospital of Boston University Medical Center, Michael Reese Hospital and Medical Center, University of Maryland Hospital, and the Neurological Institute of Columbia University. A full description of the Stroke Data Bank can be found elsewhere.⁶ Each patient with acute stroke was examined by one of the Stroke Data Bank investigators ≤ 1 week (median time to initial clinical examination 46 hours) after onset, and most patients underwent initial and subsequent CT (median time to initial CT 20 hours after onset). The weakness scales measured weakness bilaterally for the tongue, face, shoulder, hand, hip, and foot. The weakness score ranged from 0 (normal) to 60. At the time of hospital discharge, a diagnosis was determined taking into account all the available data. Information was collected on each patient concerning the details of medical, neurologic, and social history, the results of general and neurologic examinations and laboratory studies, and the final diagnosis, with special procedures for detecting complications, stroke evolution, stroke recurrence, and death.

Stroke risk factors and findings on clinical and laboratory examinations were compared for groups of patients with lacunar, large-vessel atherosclerotic, and cardioembolic infarctions. More detailed definitions of the stroke diagnostic subtypes can be found elsewhere.⁷ Lacunar infarction was diagnosed in a patient presenting with a lacunar syndrome and a CT taken ≤ 10 days after stroke onset that disclosed a small, deep infarct or a normal scan; cardiac and carotid noninvasive testing, when performed, did not demonstrate a hemodynamic or embolic source of stroke; and if angiography was performed, the major ipsilateral cerebral arteries were normal or showed unrelated findings. While no rigid definitions were set a priori, in general, hypertension included any blood pressure of $>160/95$ mm Hg; diabetes was diagnosed when the fasting venous plasma glucose concentration was >140 mg/dl on at least two separate occasions; and cardiac disease included coronary heart disease, congestive heart failure, evidence of left ventricular hypertrophy, and rhythm abnormalities, particularly atrial fibrillation.

The lacunar infarction group was further subdivided into lacunar syndrome subgroups. The classic lacunar syndromes recognized for this clinical study were pure motor hemiparesis, sensorimotor syndrome, ataxic hemiparesis, pure sensory syndrome, and dysarthria-clumsy hand syndrome. Lesion location and volume, hemiparesis profile, and weakness score were examined for each lacunar syndrome subgroup. For the pure motor hemiparesis and sensorimotor syndrome subgroups, the hemiparesis pro-

file was enumerated based on infarct location in the posterior limb of the internal capsule and the corona radiata. The posterior limb of the internal capsule was further subdivided into the anterior, medial, and posterior thirds. Lesion location and volume were recorded by sketching each lesion in the appropriate anatomic location and relative size on the Stroke Data Bank CT form.⁸ Sketches were drawn for all CT sections in which the lesion appeared. Once it was complete, a plastic transparency with a cartesian grid was placed over each sketch. Each specific grid element was converted to relative brain volume; the average volume per grid element was 1.822 ml. Adding the volumes for each grid element containing the lesion resulted in an estimate of the total lesion volume.⁶ In those cases in which more than one lacune was documented by CT, the clinical-CT analyses were based on the first lesion recorded in the Stroke Data Bank.

Results

Lacunar infarction was diagnosed in 337 (26%) of the 1,273 patients with infarction (71%) in the cohort of 1,805 patients with stroke. The median age of the lacunar infarction group was 66 years. Almost half were men, and 59% were black. This study analyzed the 316 patients classified as having classic lacunar syndromes. The 21 remaining cases were classified as having the infrequent syndromes of pure hemichorea-hemiballismus and presumed basilar branch occlusion.

Comparisons were made among the infarction groups for conventional risk factors (Table 1). Hypertension and diabetes prior to stroke were significantly more frequent in the lacunar infarction group than in the cardioembolic infarction group ($p=0.001$). The frequency of treated hypertension was similar in all three groups. Previous strokes and transient ischemic attacks (TIAs) were less frequent in the lacunar infarction group than in the large-vessel atherosclerotic infarction group ($p=0.001$), but the temporal distribution of TIAs did not differ between them. As expected by the definitions, cardiac disease was more prevalent in the cardioembolic infarction group. The clinical onset of the deficit on awakening was more frequent in the lacunar infarction group than in the other groups. Although risk factors for lacunar stroke differed from those for cardioembolic and (slightly) large-vessel atherosclerotic infarction, no difference in risk factors was found among the five recognized lacunar syndromes. (Table 2).

Angiography was performed in 56 (18%) of the lacunar stroke patients, with normal findings in 25 (44%) or clinically unrelated findings on the symptomatic (nine, 16%) and/or asymptomatic (24, 43%) side. Lesions in the major arteries ipsilateral to the infarct, including stenosis of the major intracranial or extracranial arteries and two cases of extracranial carotid occlusion, were found in 22.8% of the hypertensive patients and 5.8% of the normotensive patients.

TABLE 1. Distribution of Stroke Risk Factors by Infarction Group

	LAC (n=316)	ATH (n=113)	EMB (n=246)	P	
				LAC vs ATH	LAC vs EMB
Age (mean±SD yr)	65±0.6	64±1.0	68±0.9		
Risk factor					
Hypertension (7)	75%	74%	60%	0.90	0.001
Treatment					
No	30%	21%	16%
Yes	45%	53%	44%
Systolic blood pressure					
≥160 mm Hg	49%	38%	30%	...	0.001
Mean±SD	166±1.8	156±2.7	150±1.9		
Diastolic blood pressure					
≥90 mm Hg	52%	35%	34%	...	0.001
Mean±SD	96±1.1	91±1.5	87±1.2		
Diabetes (6)	26%	29%	17%	0.503	0.001
Blood sugar					
≥140 mg/100 ml	26%	45%	40%	...	0.001
Mean±SD	140±4.8	154±7.9	154±4.9		
Cardiac disease	24%	32%	77%	0.091	0.001
Claudication (11)	5%	11%	5%	0.049	0.082
Previous TIA (46)	13%	40%	13%	0.001	0.097
1–7 days	6%	22%	5%
8–30 days	3%	7%	2%
1–6 months	2%	7%	3%
>6 months	2%	4%	3%
Previous stroke (15)	19%	39%	29%	0.001	0.005
Deficit on awakening	38%	25%	27%

LAC, lacunar infarction; ATH, large-artery atherosclerotic infarction; EMB, cardioembolic infarction; *p*, χ^2 with 1 degree of freedom; TIA, transient ischemic attack; ..., missing data among all three groups.

A CT scan was done ≤30 days after stroke onset in 310 (98%) of the 316 lacunar stroke patients, mostly ≤48 hours after admission. Abnormalities, some not clinically related and some (e.g., atrophy and hydrocephalus) not stroke, were found in 152 patients (49%). A relevant infarct was found in 93 patients (30%) on the first CT scan. This frequency did not differ among the various lacunar syndrome subgroups, nor did lesion volume. Only four patients showed more than one related lesion. More than one CT scan was performed in 92 (29%) of the 316

lacunar stroke patients, which increased the yield of a clinically related infarct to 35%.

Pure motor hemiparesis was found in 181 (57%) of the patients with lacunar syndromes, with a positive CT scan in 62 (34%). Average lesion volume was 4.0 ml. The most frequent sites of infarcts were the posterior limb of the internal capsule and the genu (34%) and the corona radiata (20%) (Table 3); average volumes of lesions in these locations were 2.3 and 2.9 ml, respectively. In this subgroup, transient sensory symptoms occurred in 16 patients (9%) de-

TABLE 2. Stroke Risk Factors by Lacunar Syndrome Subgroup

Risk factor	Lacunar syndrome				
	PMH (n=181)	SMS (n=63)	AH (n=33)	PSS (n=21)	DCH (n=18)
Hypertension (1)	76	71	67	80	78
Diabetes	30	14	30	24	17
Claudication (2)	27	33	18	19	28
Previous TIA (12)	12	9	6	33	24
Previous stroke (5)	17	20	21	19	22

PMH, pure motor hemiparesis; SMS, sensorimotor syndrome; AH, ataxic hemiparesis; PSS, pure sensory syndrome; DCH, dysarthria-clumsy hand syndrome; TIA, transient ischemic attack; (), missing data among all five subgroups. Data are percent.

TABLE 3. Infarct Location by Lacunar Syndrome Subgroup

Location	Lacunar syndrome				
	PMH (n=181)	SMS (n=63)	AH (n=33)	PSS (n=21)	DCH (n=18)
Basal ganglia	22	19	8	0	0
Thalamus	2	9	0	67	0
Anterior limb of internal capsule	15	6	15	33	11
Genu	8	7	15	0	0
Posterior limb of internal capsule	26	31	23	0	11
Corona radiata	20	22	31	0	28
Pons	1	0	0	0	0
Cerebellum	0	0	0	0	11
Other	6	6	8	0	39

PMH, pure motor hemiparesis; SMS, sensorimotor syndrome; AH, ataxic hemiparesis; PSS, pure sensory syndrome; DCH, dysarthria-clumsy hand syndrome. Data are percent.

spite a normal sensory examination; four of these patients showed an infarct on CT scan with an average volume of 1.4 (range 0.9–2.4) ml.

Sensorimotor syndrome occurred in 63 patients (20%) and was the second most frequent syndrome. The CT scan was positive in 29 patients (46%) with an average lesion volume of 4.3 ml. The lesions were scattered throughout the subcortical region but occurred mainly in the posterior limb of the internal capsule and the corona radiata (Table 3). Average volume was 3.2 ml for the capsular lesions and 6.2 ml for those in the corona radiata; both these volumes are larger than the corresponding values for the pure motor hemiparesis subgroup.

Ataxic hemiparesis accounted for 33 patients (10%). The hemiparesis profile showed face-arm-leg involvement in four, shoulder-thigh in three, face-arm in two, and arm-leg in one. The 10 positive CT scans (31% of 32 patients examined) had an average lesion volume of 2.7 ml and showed infarcts scattered throughout the motor pathway.

Pure sensory syndrome occurred in a mere 21 patients (7%), with even fewer positive CT scans (three, 14%) and the smallest average lesion volume (1.8 ml). Two lesions were located in the thalamus and one in the anterior limb of the internal capsule (Table 3). Angiography was normal in the seven patients with pure sensory syndrome in whom it was performed.

Dysarthria-clumsy hand syndrome was the least frequent lacunar syndrome (18 patients, 6%), with a positive CT scan in five (28%) and an average lesion volume of 2.3 ml. Infarcts were found in the anterior and posterior limbs of the internal capsule, the genu, the corona radiata, and the centrum semiovale. The hemiparesis profile showed an isolated facial weakness in two patients and a faciobrachial paresis in three.

The hemiparesis profile was variable in 42 of the 117 patients with pure motor hemiparesis or sensorimotor syndrome who had lesions in the posterior limb of the internal capsule or the corona radiata (Table 4). Lesions confined to the posterior limb of the internal capsule involved the face, arm, and leg in 21 of 26 patients, the arm and leg (sparing the face) in three, the face alone in one, and the arm alone in one. The distribution of the weakness was proportional in the arm and leg in 12, more proximal in three, distal in one, and disparate in nine. In the latter patients, the weakness varied between the arm and leg.

The hemiparesis profile was compared with lesion location in the posterior limb of the internal capsule (Table 4). Face-arm-leg involvement was found in patients with lesions in each third of the posterior limb of the internal capsule, as was arm and leg involvement with sparing of the face. Isolated facial

TABLE 4. Hemiparesis Profile in PMH and SMS Patients With Posterior Internal Capsule and Corona Radiata Lesions

	Profile					
Lesion location	Face-arm-leg	Face-arm	Arm-leg	Face only	Arm only	Total
Posterior limb of internal capsule	21	0	3	1	1	26
Anterior third	4	0	0	0	0	4
Medial third	3	0	0	1	1	5
Posterior third	14	0	3	0	0	17
Corona radiata	9	4	1	1	1	16
Total	30	4	4	2	2	42

PMH, pure motor hemiparesis; SMS, sensorimotor syndrome. Data are number of patients.

palsy or brachial monoplegia also occurred with single lesions in the medial third.

Lesions isolated in the corona radiata (16 cases) revealed a variety of hemiparesis syndromes, including face, arm, and leg (nine), face and arm (four), and arm and leg alone, face alone, and arm alone in one each (Table 4). The weakness affected the arm and leg equally in four cases, the shoulder and hip (proximal) more than the wrist and ankle (distal) in five, distal portions of the limbs in one, and the arm and the leg differently in five.

Discussion

Our present study was directed toward issues of diagnosis of the lacunar syndromes. Previous reports from this cohort have already shown a frequency of worsening during hospitalization comparable to that for other types of infarction,⁹ an almost nonexistent acute mortality,¹⁰ and recurrence rates of 2.2% at 30 days¹¹ and 18% at 1 year.¹²

Our study, like most recent reports, contains no autopsy data and leaves one unable to guess the vascular pathology responsible for any infarct documented by CT. However, within the limits imposed by our material, the lack of difference in lesion volume between those with long-standing hypertension and normotensive patients failed to corroborate the impression formed from histologic studies that large, symptomatic infarcts are usually associated with long-standing hypertension.¹³

In this cohort, diabetes was a significant risk factor for lacunar infarction, a finding contrary to the observations of others.¹⁴ An initial blood sugar concentration of >140 mg/100 ml was more frequently found in the large-vessel atherosclerotic infarction group than in the lacunar infarction group of the Stroke Data Bank. Although the first poststroke systolic and diastolic blood pressures were higher and the frequency of untreated hypertension was greater in the lacunar infarction group, we found little evidence to separate lacunar stroke from other types of brain infarction on the basis of hypertension. Our findings agree with those of other modern studies showing that lacunar syndromes are common in normotensive patients.^{15,16} We speculate that the effect of increasing therapeutic attention to hypertension has made it less a factor for lacunar strokes than it may have been formerly and that improvements in brain imaging by CT and MRI have allowed better detection of small, deep lesions during life. Like the Oxfordshire Community Stroke Project,¹⁷ we found lacunar stroke and large-vessel atherosclerotic infarction to share many risk factors, except that previous TIAs were more frequent in the latter group. We did not observe the "flurry" pattern of TIA described elsewhere.¹⁸ As expected, lacunar and cardioembolic infarction showed clear differences in risk factors, including a more frequent history of stroke in the latter group ($p=0.005$). The lacunar syndromes did not differ in specific risk factors,

suggesting that they share common mechanisms of occurrence.

The percentage of positive CT scans (35%) was similar to that in some series^{4,5,19} but lower than that in others using different selection criteria.^{20,21} Although a minority of our patients underwent angiography, the low frequency of major arterial disease found agreed with that found in other studies²² and was similar to that found in an asymptomatic group of patients of similar age and with similar risk factors.²³ We infer from these findings that the morbidity of angiography may weigh against its use because it is not likely to uncover a responsible lesion in a major artery in such cases. Pure motor hemiparesis was reconfirmed as the most frequent syndrome²⁴ in persons with small, deep infarcts. This purely motor stroke involved the face, arm, and leg on one side in the absence of other findings in the sensory, visual, or higher cortical functions.²⁵ A few patients had some additional sensory symptoms at stroke onset,²⁶ but these added symptoms did not separate this group by lesion location or volume. A relation between total weakness score and lesion volume was found for capsular infarcts except in the inferior part of the internal capsule supplied by the anterior choroidal artery, where severe deficits were produced by small infarcts, presumably as a result of the fiber density at this level.^{27,28} The two cases of isolated brachial or facial weakness with capsular infarction are clear exceptions to the rule that monoplegias are a reliable sign of surface infarction.^{19,20,24}

Sensorimotor syndrome, the second most frequent, has been reported in autopsy studies as being due to infarction in the thalamus,²⁹⁻³¹ posterior limb of the internal capsule,³ and pons.³ The genu/posterior limb of the internal capsule or the corona radiata was the site of lesions found in our study, with 60% of the patients with sensorimotor syndrome having a lesion volume similar to that in patients with pure motor hemiparesis.

This cohort is one of the few large enough to address issues of classical clinical correlation with brain lesion location. The classical anatomic view of the corticospinal tract organized somatotopically in the posterior limb of the internal capsule (with fibers to the upper extremity in the anterior third, fibers to the trunk in the medial third, and fibers to the lower limb in the posterior third) was not well substantiated by our 26 patients with isolated posterior limb of the internal capsule infarcts and pure motor hemiparesis or sensorimotor syndrome. All four patients with a lesion in the anterior third showed involvement of the face, arm, and leg, with the leg and arm equally weak in two. Isolated involvement of the arm or face was reported in two cases with a lesion in the middle third, but other syndromes included the limbs affected more than the face and vice versa. Patients with infarcts in the posterior third also showed inconstant syndromes. Although the face was frequently spared, the expected increasing involvement of the leg was not found with more posteriorly placed

lesions, nor did such patients have the face, arm, and leg affected to comparable degrees. Attempts to relate the severity of weakness with the volume of a lesion in the corona radiata were frustrated by the great variety of results obtained. Although the highest frequency of partial hemiplegias was associated with corona radiata infarcts, the responsible lesions did not differ in volume from those located in the internal capsule. These findings argue against views that partial hemiplegias are more frequent with infarcts in the corona radiata because of the smaller size of the vessels that supply this territory.²⁰ We suggest that the presumed separation of the corticofugal motor fibers better explains the incomplete hemipareses than does the volume of supracapsular infarcts.

The scatter of lesions in our 10 patients with ataxic hemiparesis and a positive CT scan agree with autopsy literature, which describes such a syndrome in patients with a lesion in the posterior limb of the internal capsule,³² corona radiata,³³ anterior limb of the internal capsule,³⁴ thalamus,³⁵ and pons.³⁶ In other words, the syndrome seems not to predict the lesion locus. Future MRI studies may improve this shaky data base.

Our material adds a little to the scanty information on pure sensory syndrome: two thalamic infarcts 2.1 ml in volume and one in the anterior limb of the internal capsule 1.2 ml in volume. Autopsy documentation has been made for thalamic³⁷ and corona radiata³⁸ infarcts, while infarcts in the posterior limb of the internal capsule^{39,40} have been detected by CT. Pure sensory syndrome with an anterior capsular lesion could be explained by disruption of the anterior thalamic radiation, although a coexistent lesion undetected by CT might be responsible. Angiograms have been negative with this syndrome.⁴¹

Dysarthria-clumsy hand syndrome has been encountered in patients with lesions in a variety of locations, including the anterior limb of the internal capsule,⁴² the genu,⁴³ and the pons.⁴⁴ A recent MRI series concluded that if rigid clinical criteria are used, dysarthria-clumsy hand syndrome predicts a lesion in the contralateral basis pontis.⁴⁵ In our study, the dysarthria-clumsy hand and ataxic hemiparesis syndromes have many similarities. Unless pathologic or MRI series prove the opposite, it is our impression that these two clinical syndromes can be viewed as one entity.

In our study, the classic lacunar syndromes formed a homogeneous group in terms of risk factors, frequency of a positive CT scan, lesion volume, and lesion location. Our study also shows that lesion location and volume are not reliable predictors of the deficit. Unlike a recent MRI-based prospective study,⁴⁶ we found clinically important but statistically nonsignificant differences in volume among the different lacunar syndromes. As a group, these syndromes appear worthy of separate distinction since an invasive workup using angiography is rarely productive, worsening is uncommon after day 5 (perhaps

allowing early discharge for those who are ambulatory), recurrence rates are comparable to those for other syndromes, and recurrent strokes (usually in another brain region) are frequent but likely to also be of the lacunar type. It remains unknown whether medical therapy of the type used for cardioembolic infarction can improve the prognosis for recurrence in the lacunar syndromes, but the formerly held view that the use of anticoagulants is risky because the lesions are due to chronic hypertension no longer seems justified.

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