Lacunar Syndrome Due to Intracerebral Hemorrhage

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SUMMARY It has been recognized that small intracerebral hemorrhage not uncommonly produced lacunar syndromes. In this study, we examined cases of intracerebral hemorrhage presenting as lacunar syndromes. Of 174 cases with recent intracerebral hemorrhage, 19 presented with a lacunar syndrome: 4 presented with pure motor hemiparesis, 5, ataxic hemiparesis, 3, dysarthria-clumsy hand syndrome, 7, sensorimotor stroke, and, none, pure sensory stroke. The sites of hemorrhage were capsular in 11, putaminal in 6, and pontine in 2. In these 19 patients, 17 were hypertensive, and the signs characteristic of parenchymal hemorrhage, e.g., gradual onset, headache, nausea, vomiting and stiff neck, were absent or very rare. Computed tomography revealed that one third of the patients had one or more non-symptomatic lacunae in the basal ganglia, the corona radiata or the anterior limb of the internal capsule. These observations suggest that hypertensive intracerebral hemorrhage causes lacunar syndrome more often than previously considered and is apt to manifest ataxic hemiparesis and sensorimotor stroke. Computed tomography is the only way of differentiating hemorrhagic "lacunar" syndrome from lacunar infarct.

Methods

Our clinical material consisted of 174 cases with recent spontaneous intracerebral hemorrhage (within 48 hours from stroke onset) admitted in the Hyogo Brain and Heart Center at Himeji between August 1981 and November 1983. Acute stroke patients of all types visiting at our hospital were, without exception, examined by neurologists or neurosurgeons and performed immediate CT examination. All the patients with intracerebral hemorrhage were hospitalized and underwent detailed neurological examination. A clinical diagnosis of a lacunar syndrome was made by staff neurologists including the authors before the end of the third day. The patients presenting as one of the following lacunar syndromes were selected: (1) Pure motor hemiparesis, (2) Pure sensory stroke, (3) Ataxic hemiparesis, (4) Dysarthria-clumsy hand syndrome, and (5) Sensorimotor stroke. The specific inclusion-exclusion criteria for each lacunar syndrome was based on Fisher et al. and summarized in table 1. The absence of diminished alertness, aphasias, intellectual change, neglect syndromes, visual field defect, pupillary abnormalities and higher nervous dysfunction was Fisher did not exclude small intracerebral hemorrhage as a cause of the lacunar syndromes, but he failed to find any case of hemorrhage in his series of pathological study except for one patient with pure sensory stroke. However, in the era of computed tomography (CT), it has become evident that small non-lacunar cerebral lesions also produce the identical syndromes. Other than lacunar infarct, small intracerebral hemorrhage seems most likely to be responsible for the syndromes. A number of case reports of hemorrhage presenting as one of the four classic lacunar syndromes (to our knowledge, 18 CT-documented and 1 autopsyed cases) have appeared. Despite the recognition of the hemorrhagic cause of lacunar syndromes, no systematic investigations have been carried out. The aims of this study are, first, to examine the frequency of appearance of lacunar syndromes in intracerebral hemorrhage, second, to clarify what is the diagnostic point differentiating hemorrhagic "lacunar" syndrome from lacunar infarct, and finally, to analyze the clinical CT correlation.

Results

Among the 174 patients with acute intracerebral hemorrhage, there were 19 patients who exhibited a...
TABLE 1 Inclusion/exclusion Criteria for Each Lacunar Syndrome

<table>
<thead>
<tr>
<th>Syndrome</th>
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<tr>
<td>Pure motor hemiparesis</td>
<td>Weakness of the face and the arm and leg on one side of the body. Not including monoparesis.</td>
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<tr>
<td>Pure sensory stroke</td>
<td>Paresthesia or hypesthesia, or both involving the arm, leg and usually the face.</td>
</tr>
<tr>
<td>Ataxic hemiparesis</td>
<td>Hemiparesis, which is more severe in the lower extremity, and ipsilateral hemiataxia. Occasionally accompanied by paresthesia or mild hypesthesia, or both.</td>
</tr>
<tr>
<td>Dysarthria-clumsy hand syndrome</td>
<td>Slurred speech, facial weakness, dysphagia and minor clumsiness of the hand. Obvious weakness or ataxia of the arm and leg not accompanying.</td>
</tr>
<tr>
<td>Sensorimotor stroke</td>
<td>Both weakness and hypesthesia with paresthesia involving the face, arm and leg on one side of the body, without accompanying deficits. Not including cases with involvement of only face and hand, or hand and leg.</td>
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The absence of diminished alertness, aphasias, intellectual change, neglect syndromes, visual field defect, pupillary changes, ocuolomotor disturbances and seizures is fundamental condition for each lacunar syndrome.

lacunar syndrome. Four had pure motor hemiparesis, 5, ataxic hemiparesis, 3, dysarthria-clumsy hand syndrome, 7, sensorimotor stroke and none, pure sensory stroke. Ten were male. The patient's ages ranged from 39 to 81 years, with a mean of 60.6 years.

On admission, the systolic blood pressure ranged from 144 to 270 mm Hg and 16 patients had elevated blood pressure with systolic over 160 or diastolic over 95 mm Hg. Fifteen patients had prior history of hypertension and 17 patients were diagnosed to be hypertensive from serial reading of blood pressure during at least two weeks of hospitalization. Thus only 2 were believed to be non-hypertensive. One had prior history of putaminal hemorrhage but had completely recovered without any residual symptoms.

Headache, nausea and vomiting at the onset were rare, headache occurring in 2 and nausea and vomiting in only 1. Dizziness or light-headedness at the onset was reported by 2 patients. The onset of stroke was sudden in 16, smooth and gradual, occurring over a half an hour, in 2, and fluctuating in 1. No patient showed stiff neck, either at the onset or through the course.

Three different types of hemorrhage were found and could be classified according to their location and extension: (1) Capsular hemorrhage, (2) Putaminal hemorrhage, and (3) Pontine hemorrhage.

(1) Capsular Hemorrhage (fig. 1)

A small hemorrhage extended vertically along the posterior limb of the internal capsule. Pallidocapsular and thalamo-capsular hemorrhage were also included in this group. Its size ranged from 1.0 cc to 5.7 cc with a mean of 2.9 cc. Eleven patients belonged to this group; 6 had left-sided lesion and 5, right-sided lesion. Two patients presented with pure motor hemiparesis, presented with ataxic hemiparesis, and 5 presented with sensorimotor stroke.

In the pure motor hemiparesis group, one had a pallido-capsular hemorrhage involving the center of the posterior limb of the internal capsule, and the other had a small hemorrhage extending from the pallidum across the genu and anterior portion of the posterior limb of the internal capsule to anterolateral part of the thalamus. In ataxic hemiparesis group, 3 had a small hemorrhage almost restricted in the posterior and superior portion of the posterior limb of the internal capsule but slightly involving the posterolateral part of the thalamus, and one had a pallido-capsular hemorrhage affecting superior and posterior portion of the posterior limb of the internal capsule. In sensorimotor stroke group, the posterior limb of the internal capsule was apt to be involved at the lowest portion close to the cerebral peduncle, and the thalamus was involved in the considerable extent at its inferior part.

The functional prognosis was generally good; within a few weeks, half of the patients became ambulatory. In the remaining patients, however, it took a few months to become ambulatory with the aid of a cane. Half of the patients with sensorimotor stroke or ataxic hemiparesis were left with mild hypesthesia or numbness on the affected side.

(2) Putaminal Hemorrhage (fig. 2)

A concave lens-shaped hematoma was located in the putamen or between the putamen and the external capsule. Its size ranged from 2.0 cc to 9.8 cc and fell into the smallest portion of the spectrum of putaminal hemorrhage. No ventricular collapse or shift of the midline structures were detected. Its medial extension and upward extension did not or just reach to the adjacent internal capsule or corona radiata. Six cases belonged to this group; 4 had left-sided lesion and 2, right-sided lesion. Two patients were consistent with pure motor hemiparesis, 3, were consistent with dysarthria-clumsy hand syndrome and 1 showed sensorimotor stroke. One of these exhibiting dysarthria-clumsy hand syndrome had a prior left-sided putaminal hemorrhage, opposite to the present hemorrhage, whose CT revealed an irregular hypodense lesion in the anterior putamen consistent with old hemorrhagic scar. The remaining 2 dysarthria-clumsy hand syndrome patients had left-sided hemorrhage only. The difference of lesion site between pure motor hemiparesis and dysarthria-clumsy hand syndrome was not necessarily clear, but the hemorrhage in the latter syndrome appeared to be located anteriorly to that in the former. One case whose lesion involved a part of the internal capsule presented with sensorimotor stroke.

The prognosis of all 6 patients was excellent; all recovered almost fully within a week, except that the sensorimotor stroke patient complained numbness on the affected side as a residual. In one patient with pure motor hemiparesis, the right hemiparesis disappeared.
within the 24 hours after onset, mimicking a TIA episode.

(3) Pontine Hemorrhage (fig. 3)
A very small hemorrhage was located in the upper-mid-pons. One patient had mild hemiparesis, mild hemi-hypesthesia to pin-prick and light touch, numbness and severe ataxia contralateral to the lesion and were consistent with ataxic hemiparesis. The patient had gaze evoked horizontal nystagmus. The other patient had mild hemiparesis and hemi-hypesthesia contralateral to the hemorrhage and were consistent with sensorimotor stroke. The hemorrhage of the two patients appeared to be similar in location, locating around the tegmentum rather than in the basis pons, and size (0.3 cc).

The prognosis was good; both became ambulatory two weeks after onset. Mild ataxia and numbness of the hand persisted in the former and paresthesia of chetro-oral type remained in the latter.

In 6 of the 19 patients with a hemorrhagic "lacunar" syndrome, CTs revealed one or more small round or oval hypodense lesions in the basal ganglia, the corona radiata or the anterior limb of the internal capsule, which suggested non-symptomatic lacunae. Among the 6 patients who had lacunae, 5 belonged to the capsular hemorrhage group and one to the pontine hemorrhage group.

**Discussion**
In the present series, of 174 cases of spontaneous intracerebral hemorrhage, 19 (10.9%) presented with a lacunar syndrome. This incidence of hemorrhagic "lacunar" syndrome is higher than previously considered. Before the introduction of CT, intracerebral hemorrhage was believed to produce severe symptoms and therefore, such a small hemorrhage producing a lacunar syndrome might have been misdiagnosed as a lacunar infarct. As lacunar syndrome was frequently diagnosed on clinical ground alone even in the era of CT,23 such misdiagnoses may still be present. The early studies using CT on hypertensive putaminal or thalamic hemorrhage24, 25 failed to describe the occurrence of the 4 classic lacunar syndromes. During the past 5 years, a number of cases of intracerebral hemorrhage presenting as lacunar syndromes have been described.10, 12-23 Nowadays one should recognize that small intracerebral hemorrhage is one of the likely causes of lacunar syndrome.

The high incidence of ataxic hemiparesis and sen-
A lacunar syndrome from intracerebral hemorrhage/Mori et al

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FIGURE 2. Contiguous CT sections of patients with lacunar syndrome due to putaminal hemorrhage. (Top) A patient with pure motor hemiparesis. The hemorrhage is located in the left putaminal region, just reaching to the internal capsule. (Middle) A patient with dysarthria-clumsy hand syndrome. The hemorrhage is located in the left anterior putaminal region, not involving the internal capsule. (Bottom) A patient with sensorimotor stroke. The hematoma is located in the right putaminal region with slight involvement of the posterior limb of the internal capsule.

sensormotor stroke and low incidence of pure motor hemiparesis and pure sensory stroke in intracerebral hemorrhage is noteworthy. Previous studies suggested that pure motor hemiparesis was the most frequently encountered lacunar syndrome, as often as 50% of symptomatic lacune.11, 24, 27, 28 Fisher9 considered that pure sensory stroke was the most common manifestation. However, in our series of hemorrhagic "lacunar" syndrome, only 21% had pure motor hemiparesis and none pure sensory stroke. Conversely, ataxic hemiparesis and sensorimotor stroke was rare manifestation in the previous studies of lacunar infarct (below 10%),24, 27, 28 but in our series, 26% and 37% showed them respectively. For the capsular lesion, the difference of manifestation between the two causes is more remarkable.

The present study suggested that in differentiating the small hemorrhage presenting with lacunar syndromes from lacunar infarct, presence of stiff neck, headache, nausea and vomiting is quite useless. The incidence of hypertension in our patients with hemorrhagic "lacunar" syndrome was 90% and was slightly higher than that in patients with lacunar infarct in previous reports (57–75%),11 but elevated blood pressure on admission is of little help for differential diagnosis. Although we did not perform a lumbar puncture, it would not be expected to yield bloody CSF because of the small size of hematoma and its location apart from the subarachnoid space and the ventricular system. Although gradual evolution would be most likely mode of onset in intracerebral hemorrhage,24 most of our patients with hemorrhagic "lacunar" syndrome showed sudden onset. As there was neither expansion of hematoma nor marked edema because of its small size, the symptoms would be maximal at onset. Since lacunar infarct would show sudden onset as well as step-wise progression,21 it seems difficult to differentiate the two from mode of onset.

It has been well known from pathological and CT studies that lacune involving the internal capsule causes pure motor hemiparesis,1, 12, 15, 27, 29 ataxic hemiparesis,27, 28 dysarthria-clumsy hand syndrome28, 30 and sensorimotor stroke.5, 28, 29 The present study and previous studies suggest that small hemorrhage confined to the same site can also produce identical syndromes. Although it is difficult to discuss the precise location of the lesion responsible for each syndrome, it seems that ataxic hemiparesis results from hemorrhage in the uppermost and posterior part of the posterior limb of the internal capsule and the posterolateral aspect of the
FIGURE 3. A CT section of patient with atoxic hemiparesis due to pontine hemorrhage. A small hemorrhage is located in the left upper-mid-pons around the tegmentum rather than in the basis pontis.

thalamus, sensorimotor stroke from extension of hemorrhage into the lowermost part of the posterior limb of the internal capsule and lower and more medial part of the lateral thalamus, and pure motor hemiparesis from hemorrhage involving only anterior half of the posterior limb of the internal capsule.

Tapia et al\textsuperscript{16} reported a case with pure motor hemiparesis due to putaminal hemorrhage, and Minematsu et al\textsuperscript{20} described a case with dysarthria-clumsy hand syndrome due to putaminal hemorrhage. In the present study, we documented the similar cases. A reason why putaminal hemorrhage without involvement of the internal capsule produces pure motor hemiparesis or dysarthria-clumsy hand syndrome may be attributed to the pressure effects to the internal capsule; in the former syndrome, to the posterior limb of the internal capsule and in the latter syndrome, to the anterior limb and genu of the internal capsule.

Small hemorrhage as well as lacunar infarct in the basis pontis was reported to produce ataxic hemiparesis\textsuperscript{19, 21, 22} and pure motor hemiparesis.\textsuperscript{13} The 2 patients with pontine hemorrhage in this study showed ataxic hemiparesis with sensory impairment or sensorimotor stroke. Because the hemorrhage in both patients appeared to be located in the dorsal part of the upper-mid-pons involving the medial lemniscus and/or spinothalamic tract, sensory impairment was associated.

As for the pathogenesis of small hemorrhage which can cause lacunar syndrome, high association rate of non-symptomatic lacunae is of interest. Fisher\textsuperscript{21, 32} pointed out that cerebral hemorrhage was frequently associated with lacunae, and suggested that small non-symptomatic lacune was most often the result of occlusion of perforating artery by lipohyalinosis, a hypertensive cerebral vasculopathy, which is probably also the source of hypertensive intracerebral hemorrhage. Our result confirmed this Fisher’s notion in clinical material.

In summary, hypertensive intracerebral hemorrhage produces lacunar syndromes more often than previously thought. It seems that CT is the only way of differentiating hemorrhage from infarct. When a patient with a lacunar syndrome is seen, especially ataxic hemiparesis or sensorimotor stroke, one should suspect a small hemorrhage as well as a lacunar infarct.

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References

Cause of Cerebral Infarction in the Carotid Territory. Its Relation to the Size and the Location of the Infarct and to the Underlying Vascular Lesion

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SUMMARY Seventy-three patients with acute nonhemorrhagic stroke in the carotid territory were investigated for the cause of the stroke: middle cerebral artery (MCA) occlusion/stenosis or internal carotid artery (ICA) occlusion/stenosis; embolus from the heart and extra-cranial arteries or thrombosis. The study is prospective and consecutive comprising stroke patients below the age of 75 years, admitted in the acute state i.e. within 3 days after stroke onset. Excluded were patients with intracerebral hematoma, subarachnoid hemorrhage, vertebrobasilar stroke and patients in whom another severe disease was present. Cerebral angiography and CT-scan were performed in all patients within one and two days after admission. CT-scan was repeated 2 weeks and 6 months later.

Forty percent had MCA occlusion, none had MCA stenosis, 12% had ICA occlusion, 14% had severe ICA stenosis (half of these were associated with MCA occlusion) and 41% were without significant MCA/ICA lesions. Twenty-seven percent had large infarcts with a diameter >3 cm; 34% had medium-sized infarcts with a diameter between 1.5 and 3 cm; 21% had small infarcts with a diameter <1.5 cm; 18% had no identifiable infarct on CT-scan. MCA occlusion was responsible for 62% of the large or medium-sized infarcts. ICA occlusion or severe ICA stenosis were responsible for only 27% of the large or medium-sized infarcts. Only 11% of the patients with small or no infarct on CT-scan had significant MCA/ICA lesion.

The frequency of infarction due to embolus from the heart and extracranial arteries was estimated to be about 40%. Another 40% of the infarcts were probably lacunes due to small vessel thrombosis. Only about 20% of the infarcts seemed to be due to large-vessel thrombosis. Emboli from the heart and extracranial arteries gave rise to twice as many major strokes as did thromboses.
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