Effect of Capsular Infarct Size on Clinical Presentation of Stroke

Young Ho Sohn, MD, Byung In Lee, MD, Il Nam Sunwoo, MD, Ki Whan Kim, MD, and Jung Ho Suh, MD

We reviewed the medical records and cranial computed tomograms of 74 patients with acute capsular infarcts to investigate the correlation between infarct size and clinical symptoms. Average infarct size varied significantly by clinical syndrome; patients with sensorimotor stroke had the largest infarcts, patients with pure motor hemiparesis had middle-sized infarcts, and patients with ataxic hemiparesis or the dysarthria-clumsy hand syndrome had the smallest infarcts. Although it has been proposed that the type of lacunar syndrome is determined entirely by the infarct location, our results suggest that infarct size is another important factor influencing the clinical presentation of lacunar syndromes. (Stroke 1990;21:1258-1261)

The introduction of computed tomography (CT) made it much easier to identify small deep infarcts and to correlate their size and location with clinical variables. It has been reported that the size of deep cerebral infarcts affects the severity of clinical symptoms but not the type of lacunar syndrome. However, Nelson et al. reported that all infarcts in patients with the dysarthria-clumsy hand syndrome were < 4 ml in volume, whereas 31% in patients with pure motor hemiparesis and 50% in those with pure sensory stroke were larger than that. Allen et al. documented that lesions in patients with sensorimotor stroke were larger than those in patients with pure motor hemiparesis. These reports suggest that the size of small deep infarcts might have a role in determining the type of clinical syndrome and prompted us to analyze the clinical features and CT findings of 74 patients with acute capsular infarcts.

Subjects and Methods

We reviewed the medical records and cranial CT scans of 74 patients with CT-verified acute capsular infarcts who were admitted to the Yonsei University Medical Center in Seoul, Korea between January 1, 1983, and March 31, 1989. Inclusion criteria were symptoms of one of the recognized lacunar syndromes (pure motor hemiparesis, sensorimotor stroke, pure sensory stroke, ataxic hemiparesis, or the dysarthria-clumsy hand syndrome) in the absence of cortical deficits and cranial CT scans taken ≤10 days after onset showing only one ischemic lesion in the capsular region (internal capsule, basal ganglia, corona radiata, or adjacent areas) consistent with the patient's recent clinical symptoms. We excluded patients with only transient neurologic symptoms lasting <24 hours or multiple lesions on cranial CT scans.

All CT examinations were performed both before and after the intravenous injection of 150 ml of 60% meglumine iothalamate, using either a GE 9800 scanner (Milwaukee, Wis.) with a 256x256 matrix and a 10 mm slice thickness or a Philips Tomoscan 310 scanner (Eindhoven, The Netherlands) with a 256x256 matrix and a 9 mm slice thickness. Infarct volume was estimated according to the method of Nelson et al.

We classified the lacunar syndrome in our patients according to Fisher's criteria as pure motor hemiparesis, sensorimotor stroke, or ataxic hemiparesis/dysarthria-clumsy hand syndrome. We disregarded the transient symptoms found only at the time of onset but considered the clinical features that persisted for ≥72 hours after admission. We regarded ataxic hemiparesis and the dysarthria-clumsy hand syndrome as a single syndrome because practical differentiation between them was often difficult. We classified infarct location on the CT scans according to Kashihara and Matsumoto with some modification as anterior (low-density areas mainly within the head of the caudate nucleus, the anterior limb of the internal capsule, and the lenticular nucleus; Figure 1, left), posterior (low-density areas mainly within the genu and the posterior limb of the internal capsule; Figure 1, right), or superior (low-
density areas mainly lateral to the body of the lateral ventricle; Figure 1, bottom).

The size of infarcts in each lacunar syndrome was analyzed by one-way analysis of variance. A value of $p<0.05$ was considered to indicate significance.

**Results**

Among the 74 patients with CT-verified acute capsular infarcts, 44 (59.5%) were men and the other 30 (40.5%) were women. Their ages ranged from 23 to 91 (mean±SD 60.7±12.9) years. Infarct volume ranged from 0.16 to 6.93 (mean±SD 1.72±1.75) ml.

The presenting lacunar syndrome was pure motor hemiparesis in 46 (62.2%), sensorimotor stroke in 17 (23.0%), and ataxic hemiparesis/dysarthria-clumsy hand syndrome in 11 (14.9%) patients. Pure sensory stroke was not found in any patient with capsular infarcts.

Infarct location was anterior in 28 (37.8%), posterior in 20 (27.0%), and superior in 26 (35.1%) patients. Pure motor hemiparesis was the most frequent lacunar syndrome for infarcts in all three locations. Sensorimotor stroke was more frequent than ataxic hemiparesis/dysarthria-clumsy hand syndrome.
Superior

Posterior

Anterior

(mL)

Size

TABLE 1. Infarct Size by Lacunar Syndrome and Location

Syndrome

<table>
<thead>
<tr>
<th>Location</th>
<th>Pure motor hemiparesis</th>
<th>Sensorimotor stroke</th>
<th>Ataxic hemiparesis/dysarthria-clumsy hand</th>
<th>Size</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Size</td>
<td>n</td>
<td>Size</td>
</tr>
<tr>
<td>Anterior</td>
<td>17</td>
<td>2.67±1.80</td>
<td>5</td>
<td>4.89±1.60</td>
</tr>
<tr>
<td>Posterior</td>
<td>12</td>
<td>0.56±0.70</td>
<td>5</td>
<td>1.59±0.97</td>
</tr>
<tr>
<td>Superior</td>
<td>17</td>
<td>0.86±0.57</td>
<td>7</td>
<td>2.81±2.24</td>
</tr>
<tr>
<td>Total</td>
<td>46 (62.2%)</td>
<td>1.45±1.51</td>
<td>17 (23.0%)</td>
<td>3.06±2.16</td>
</tr>
</tbody>
</table>

Size as mean±SD mL.

*Probability values were obtained by one-way analysis of variance.

Discussion

Recent clinical studies using advanced neuroimaging techniques have significantly modified our classical concept of the lacune as having a limited size (<10 mm in diameter),9 a unique pathogenesis such as lipohyalinosis,9 and strict clinicoanatomic correlations. Clinically significant lacunes are often >10 mm in diameter and more often result from microatheroma, embolism, or large-artery diseases10-12 than from lipohyalinosis. Certain lacunar syndromes are also occasionally caused by lesions in unexpected sites; for example, sensorimotor stroke may be caused by a lesion in the capsular region.11113-14 However, the exclusive relation between lesion location and type of lacunar syndrome has still not changed.

Most previous clinical studies have tried to relate the patient's clinical symptoms to the location of the infarct rather than to its size.1,3,7,13 Since the internal capsule is a very compact structure consisting of many neural pathways of diverse functions, not only the location of the lesion but also its size should be important factors affecting the patient's clinical symptoms. In our study, the distribution of pure motor hemiparesis and other lacunar syndrome types was consistent for different lesion locations. Among the other lacunar syndrome types, the most critical factor differentiating ataxic hemiparesis/dysarthria-clumsy hand syndrome from sensorimotor stroke was infarct size.

Donnan et al11 reported that capsular lesions in patients with incomplete pure motor hemiparesis were smaller than those in persons with complete pure motor hemiparesis, which might be due to selective pyramidal tract involvement. In our study, ataxic hemiparesis/dysarthria-clumsy hand syndrome could have been related to the smaller lesions and more selective pyramidal tract involvement than is found in pure motor hemiparesis. In the dysarthria-clumsy hand syndrome, the corticobulbar tracts might be involved more selectively, with relative sparing of other parts of the pyramidal tract. In ataxic hemiparesis, although the frontotopontine or cerebellocortical fibers are also involved,15 it seems likely that the degree of pyramidal tract involvement is mild. If it had been more severe, the ataxia might have been masked by severe hemiplegia.

Lesion location in patients with sensorimotor stroke was somewhat confusing. The original case proved by pathologic examination demonstrated a lesion in the thalamocapsular area.16 However, several previous reports using CT showed the lesions in persons with sensorimotor stroke to be located in various capsular areas1,11113-14 that were hardly explained by classical neuroanatomic concepts. Krappelle and van Gijn2 reported that capsular lesions in patients with sensorimotor stroke were larger than that. In the posterior and superior locations, all lesions in patients manifesting ataxic hemiparesis/dysarthria-clumsy hand syndrome were <2 mL in volume, whereas all those in patients with sensorimotor stroke were larger than that (Table 2).

**TABLE 2. Distribution of Lacunar Syndromes by Infarct Size and Location**

Syndrome

<table>
<thead>
<tr>
<th>Size (mL)</th>
<th>Pure motor hemiparesis</th>
<th>Sensorimotor stroke</th>
<th>Ataxic hemiparesis/dysarthria-clumsy hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;2</td>
<td>9</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>≤2</td>
<td>8</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Posterior</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;0.5</td>
<td>4</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>≤0.5</td>
<td>8</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Superior</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;0.5</td>
<td>10</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>≤0.5</td>
<td>7</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

Values are number of patients.
described the mechanisms of capsular sensorimotor stroke as resulting from anatomic variations, the effects of surrounding edema, or simultaneous invisible infarcts in different locations. However, these explanations appear to be insufficient, particularly in patients with lenticular sensorimotor stroke.

The exact courses of the thalamocortical sensory projections are still unclear, but they are thought to comprise two systems. In the first system, the essential or specific sensory projection transferring primary sensory input passes through the posterior limb of the internal capsule and the corona radiata, within which the nerve fibers are aggregated compactly. In the second system, the nonspecific sensory projection transferring multimodal sensory input passes via the striatal, limbic, and reticular systems, within which the nerve fibers are dispersed.17 On the basis of these concepts, we speculate on the causes of sensory deficits due to capsular infarcts. In the posterior location, deficits are due to direct involvement of the capsular-specific sensory projection or indirect compressive effects on the thalamus, in the superior location to direct or indirect effects on the radiata-specific sensory projection, and in the anterior location to direct involvement of the nonspecific sensory projection or the radiata-specific sensory projection with upward extension. This explanation may be compatible with our result that lesions in the anterior location are much larger (>2 ml) than those in other locations (>0.5 ml).

Chokroverty and Rubino18 reported that approximately half of patients with pure motor hemiparesis show abnormalities on somatosensory evoked potential tests, suggesting that pure motor hemiparesis might have subclinical sensory deficits and that the capsular sensory fibers might be located close to the motor fibers. Thus, as our results suggest, the larger the capsular lesion, the more the sensory fibers might be involved to manifest clinical sensory deficits.

For patients with pure motor hemiparesis, the infarct was larger in the anterior location than in the posterior and superior locations. The exact pathomechanisms of pure motor hemiparesis occurring in patients with anterior lesions are still unclear, but involvement of the pyramidal tract at its rostral portion or the effect of CT-negative surrounding ischemia have been suggested.19 For either mechanism, it is likely that an infarct in the anterior location manifesting as hemiparesis should be larger than those in the posterior or superior locations, which directly involve the pyramidal tract or the corona radiata, respectively.

The pyramidal tract fibers in the posterior limb of the internal capsule are arranged somatotopically from anterior to posterior in face-arm-leg order. Therefore, in the posterior location, lesions may influence the pattern of pure motor hemiparesis in terms of the degree of face-arm-leg involvement. Donnan et al1 reported that lacunes in the anterior half of the posterior limb of the internal capsule are more likely to involve the face, whereas those in the posterior half are more likely to involve the leg. Among our 12 patients with pure motor hemiparesis and lesions in the posterior location, one of seven with a lesion in the anterior half showed incomplete hemiparesis involving the face and arm and three of five with a lesion in the posterior half showed incomplete hemiparesis involving the arm and leg. These results do not violate the concept of somatotopic distribution of the capsular pyramidal tract fibers, but the number of our cases is too small to be meaningful and further prospective studies are required.

Performed during the acute stage, CT often overestimates the size of an infarct compared with those that are pathologically proven or measured during the subacute or chronic stages.27 However, our selection of acute cases (≤10 days after onset) probably minimized this variability. We propose that in the capsular region, the size of the infarct as well as its location are important factors determining the type of lacunar syndrome.

References


KEY WORDS • cerebral infarction • lacunar infarction
Effect of capsular infarct size on clinical presentation of stroke.
Y H Sohn, B I Lee, I N Sunwoo, K W Kim and J H Suh

Stroke. 1990;21:1258-1261
doi: 10.1161/01.STR.21.9.1258

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/21/9/1258

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/