Pure Sensory Stroke
Clinical–Radiological Correlates of 21 Cases
Jong Sung Kim, MD

Background: Although pure sensory stroke is a relatively common lacunar syndrome, the responsible lesions are often unidentified because of their small size. I reported 21 cases of pure sensory stroke in which the lesions could be identified by head computed tomography and/or magnetic resonance imaging and correlated the clinical findings with the radiological lesions.

Summary of Report: Eleven patients had thalamic strokes. Lacunes confined to the posterolateral part of the thalamus were found in nine cases, and hemorrhages of relatively large size were found in two. Five patients showed a loss of all sensory modalities, but six with very small lacunes showed minor or restricted sensory changes. Seven patients with lacunes or hemorrhages in the lenticulocapsular region or corona radiata showed abnormalities of spinothalamic tract sensation. Two patients with a small lacune and a hemorrhage in the pontine tegmentum showed a selective sensory deficit of the medial lemniscal type. One patient with a small cortical infarct showed a cortical sensory loss that was preceded by cortical sensory transient ischemic attacks.

Conclusions: Pure sensory stroke can occur with lesions in various areas of the somatosensory system. Hemisensory deficits of all modalities usually are associated with a relatively large lacune or hemorrhage in the lateral thalamus, whereas tract-specific or restricted sensory changes suggest very small strokes in the sensory pathway from the pons to the parietal cortex. (Stroke 1992;23:983–987)

Key Words • computed tomography • magnetic resonance imaging • pure sensory stroke

Pure sensory stroke (PSS) is a well-defined clinical entity in which hemisensory symptoms predominate without other major neurological signs. While Fisher reported PSS to be the most common lacunar syndrome, more recent studies showed that it is the second or fourth most common. Although thalamic stroke is the most frequent cause of PSS, nonthalamic strokes involving the brain stem, internal capsule, or cerebral cortex are also reported to produce PSS. Because of their small sizes, the lesions producing PSS are often difficult to identify. Fisher previously analyzed the clinical features of 135 patients with PSS and allied conditions but could not localize the lesions. Although computed tomography (CT) may be helpful, it is often unable to confirm lesions. With the advent of magnetic resonance imaging (MRI), small strokes causing PSS are more easily identified. However, until recently there has been no clinical–radiological study of PSS, which prompted me to report 21 cases in which clinical manifestations correlated with radiological (CT and/or MRI) findings.

Subjects and Methods
I identified 24 patients with pure hemisensory stroke who showed appropriate lesions on head CT (GE/9800) and/or MRI at Asan Medical Center between September 1989 and December 1991. The MRI studies were performed using a 1.5-T superconducting magnet (GE). Axial T2-weighted (repetition time/echo time [TR/TE], 2,500/80), T1-weighted (TR/TE, 600/20), and sagittal T1-weighted scans were generated with a slice thickness of 5 mm. Three patients had more than one lesion contralateral to the sensory deficit and were thus excluded. All 21 patients were examined by the author (patients 12 and 19 were previously reported). In 14 patients CT identified the lesions. In four of these patients MRI was performed later to confirm the presence of the lesion and the absence of other lesions, whereas only MRI was performed in seven patients. The clinical symptoms and signs were correlated with the radiological localization. The size of the stroke was classified as large, small, or very small when the longest diameter of the lesion was >1.5 cm, 0.5–1.5 cm, or <0.5 cm, respectively.

Results
Twenty-one patients (12 men and 9 women, aged 46–74 [mean, 57] years) were studied; the details of each patient are summarized in Table 1. Risk factors for stroke included hypertension in 16, diabetes mellitus in three, and hyperlipidemia in three. Patient 2, who had coronary heart disease, was positive for both lupus anticoagulant and anticardiolipin antibody, and patient 3 had pancreatic cancer.

Thalamic Strokes
Eleven patients (cases 1–11) had thalamic strokes, of which nine were ischemic (lacune) and two were hemorrhagic. Eight lesions were demonstrated by head CT

From the Department of Neurology, College of Medicine, University of Ulsan, Asan Medical Center, Seoul, Korea.
Address for correspondence: Jong Sung Kim, MD, Department of Neurology, Asan Medical Center, 388-1, Poongnap Dong, Songpa-Ku, Seoul 138-040, Korea.
Received January 1, 1992; accepted March 3, 1992.
TABLE 1. Clinical Features of 21 Patients With Pure Sensory Stroke

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex/Age</th>
<th>Risk factors</th>
<th>Nature</th>
<th>Site</th>
<th>Size</th>
<th>Sensory deficits</th>
<th>Identification</th>
<th>Remark</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F/52</td>
<td>HT</td>
<td>I</td>
<td>R Thal</td>
<td>S</td>
<td>+ + + + +</td>
<td>CT</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>M/55</td>
<td>CHD, APL</td>
<td>I</td>
<td>L Thal</td>
<td>S</td>
<td>+ + - + -</td>
<td>CT</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M/55</td>
<td>Cancer</td>
<td>I</td>
<td>R Thal</td>
<td>S</td>
<td>+ + + + nc</td>
<td>CT</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>M/52</td>
<td>HT</td>
<td>H</td>
<td>L Thal</td>
<td>L</td>
<td>+ + + - +</td>
<td>CT</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>F/58</td>
<td>HT</td>
<td>H</td>
<td>L Thal</td>
<td>L</td>
<td>+ + + + nc</td>
<td>CT</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>M/55</td>
<td>HT</td>
<td>I</td>
<td>R Thal</td>
<td>VS</td>
<td>- - - - -</td>
<td>MRI</td>
<td>Faciobrachial distribution</td>
</tr>
<tr>
<td>7</td>
<td>M/50</td>
<td>HT, DM</td>
<td>I</td>
<td>L Thal</td>
<td>VS</td>
<td>- - - - -</td>
<td>CT</td>
<td>Cheiro-oral syndrome</td>
</tr>
<tr>
<td>8</td>
<td>F/64</td>
<td>HT</td>
<td>I</td>
<td>R Thal</td>
<td>VS</td>
<td>+ - + - -</td>
<td>MRI</td>
<td>Cheiro-oral and four toes</td>
</tr>
<tr>
<td>9</td>
<td>F/57</td>
<td>HT</td>
<td>I</td>
<td>R Thal</td>
<td>VS</td>
<td>- - - - -</td>
<td>MRI</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>M/56</td>
<td>DM</td>
<td>I</td>
<td>R Thal</td>
<td>VS</td>
<td>+ - - - -</td>
<td>MRI</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>M/46</td>
<td>HT</td>
<td>I</td>
<td>L Thal</td>
<td>VS</td>
<td>- - + + +</td>
<td>CT</td>
<td>Restricted to digits</td>
</tr>
<tr>
<td>12</td>
<td>M/47</td>
<td>HT, HL</td>
<td>I</td>
<td>L Post IC</td>
<td>VS</td>
<td>+ + - - -</td>
<td>CT, MRI</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>M/56</td>
<td>HT</td>
<td>I</td>
<td>R Post IC</td>
<td>S</td>
<td>+ + - - -</td>
<td>CT</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>M/61</td>
<td>HT</td>
<td>H</td>
<td>L Post IC</td>
<td>VS</td>
<td>+ + - - -</td>
<td>MRI</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>M/55</td>
<td>HT</td>
<td>H</td>
<td>L Post IC</td>
<td>VS</td>
<td>+ + - - -</td>
<td>MRI</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>M/55</td>
<td>HT</td>
<td>I</td>
<td>L CR</td>
<td>VS</td>
<td>+ + - - -</td>
<td>CT, MRI</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>F/59</td>
<td>HT</td>
<td>I</td>
<td>L CR</td>
<td>S</td>
<td>- - - - -</td>
<td>CT</td>
<td>Sensory TIA</td>
</tr>
<tr>
<td>18</td>
<td>F/55</td>
<td>HL</td>
<td>I</td>
<td>L CR</td>
<td>VS</td>
<td>+ - - - -</td>
<td>MRI</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>F/67</td>
<td>HT</td>
<td>H</td>
<td>R Pons</td>
<td>VS</td>
<td>- - + + +</td>
<td>CT, MRI</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>F/74</td>
<td>HT</td>
<td>I</td>
<td>R Pons</td>
<td>S</td>
<td>- - + + +</td>
<td>CT, MRI</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>F/65</td>
<td>DM, HT</td>
<td>I</td>
<td>R Parietal S</td>
<td>-</td>
<td>- - + + +</td>
<td>MRI</td>
<td>Sensory TIA</td>
</tr>
</tbody>
</table>

Pi, pinprick; T, touch; V, vibration; Po, position; C, cortical; F, female; M, male; HT, hypertension; CHD, coronary heart disease; APL, antiphospholipid antibody syndrome; DM, diabetes mellitus; HL, hyperlipidemia; I, infarct; H, hemorrhage; R, right; L, left; Thal, thalamus; Post, posterior; IC, internal capsule; CR, corona radiata; S, small; L, large; VS, very small; nc, not checked; CT, computed tomography; MRI, magnetic resonance imaging; TIA, transient ischemic attack.

and three by MRI. In all ischemic strokes, the lesions were confined to the posterolateral part of the thalamus probably involving the ventral posterolateral nucleus (Figure 1). The two hemorrhagic strokes were relatively large in size.

Five patients with thalamic stroke showed sensory deficit of both spinothalamic and medial lemniscal types, whereas six with very small lacunes (cases 6-11) did not. Patients 6 and 7 complained of an unpleasant numbness over the face and arm without an objective sensory deficit, while patient 10 showed a mildly decreased pinprick sense only. Patient 8, who had decreased pinprick and vibration senses restricted over the left perioral and cheek area, suffered numbness in those areas as well as the hard palate and palm. Patient 9 had painful dysesthesia over the same area as patient 8 but additionally had numbness in the first four toes. Patient 11, who initially had mildly decreased position and vibration senses in the right arm and leg, soon showed sensory changes restricted to the fingers.

Case 1. A hypertensive 52-year-old woman developed sudden numbness in the left arm and leg. On admission, her blood pressure was 160/110 mm Hg with regular pulse and no carotid bruits. Neurological examination showed normal motor function and reflexes. There was a decreased sensation to pinprick, touch, vibration, and position over the entire left side of her body. Graphesthesia and stereognosis were also impaired in the left limbs. Head CT performed 3 days after the onset showed an infarct in the posterolateral part of the right thalamus (Figure 1). The hypesthesia and unpleasant numbness continued.

Case 11. This 46-year-old hypertensive man suddenly felt heaviness in his right leg and complained of a gait difficulty. When he was first examined 2 days later his blood pressure was 160/100 mm Hg, and he showed mildly decreased vibration and position senses in his right arm and leg. The next day he complained of numbness restricted to the right distal fingers. Sensory examination showed impaired position sense in all right fingers and decreased vibration sense restricted to the area distal to the first interphalangeal joints. However, pinprick and touch senses were spared. Head CT showed a small infarct in the left thalamus (Figure 2).

Lenticulocapsular or Corona Radiata Strokes

Four patients (cases 12-15) with lenticulocapsular strokes were identified. Patients 12 and 13 had lacunes in the lentiform nuclei abutting the posterior portion of the posterior limb of the internal capsule, and patients 14 and 15 had very small hemorrhages near the middle third of the posterior limb of the internal capsule. Three patients showed very small (cases 16 and 18) or small (case 17) lacunes in the deep white matter of the middle
FIGURE 1. Computed tomography shows infarct in posterolateral part of right thalamus.

cerebral artery territory, probably involving the corona radiata. All of the patients with lenticulocapsular or corona radiata stroke showed a decreased sense of spinothalamic modality and suffered from a long-lasting, unpleasant hemiparesis some time after the initial events. Vibration and position senses were spared in all of these patients. Patient 18 had suffered repeated episodes of sensory transient attacks.

Case 13. A 56-year-old hypertensive man awoke with numbness in the left limbs. Neurological findings were normal except for decreased pinprick and temperature senses over the entire left side of his body. His objective sensory dysfunction gradually improved, but the unpleasant hemiparesis continued. Initial brain CT was negative, but a repeat scan performed 3 months after the onset showed a small infarct in the right lentiform nucleus abutting the posterior part of the posterior limb of the internal capsule (Figure 3).

Brain Stem Strokes

Two women with pontine strokes had PSS. One had a small hemorrhage, and the other had an infarct in the tegmentum. Both showed an isolated sensory deficit of the medial lemniscal type.

Case 19. This 67-year-old hypertensive woman suddenly felt numb in the left half of her body. In the emergency room her blood pressure was 230/140 mm Hg. Cranial nerve and motor functions were normal. Vibration and position senses in the left limbs were decreased and were worse in the lower extremity. Pinprick and temperature were normally perceived. When standing, she veered to the left. T1-weighted MRI performed 2 days after the onset demonstrated a small hemorrhage in the right pontine tegmentum (Figure 4).

Cerebral Cortical Strokes

One patient with cortical infarct showed PSS. Interestingly, the PSS was preceded by multiple pure cortical sensory transient ischemic attacks.

Case 21. A 65-year-old hyperglycemic and hyperlipidemic woman developed intermittent abnormal sensation and weak feelings in the left arm 7 days before admission. These episodes occurred two or three times a day and lasted usually less than an hour. During those attacks she could walk, but veered to the left, and the posture of her left arm became awkward. Neurological examination showed a normal, obese woman, but during the attacks she showed a clumsy left hand with markedly decreased vibration and position senses and abnormal stereognosis and graphesthesia. However, pinprick and temperature senses were spared. A similar but less marked sensory abnormality was present in her left leg.

FIGURE 2. Computed tomography shows small infarct in left thalamus.

FIGURE 3. Computed tomography shows infarct in right lentiform nucleus bordering posterior limb of internal capsule.
FIGURE 4. T1-weighted magnetic resonance image (repetition time/echo time, 600/20) shows high-signal intensity in right pontine tegmentum corresponding to location of medial lemniscus.

In addition, her left arm was observed to assume an abnormal posture: her elbow became gradually flexed to 90° with her wrist and all fingers slightly flexed. She did not notice this abnormal posture unless told, after which she could promptly correct it. During admission, the recurrent attacks led her to suffer PSS of cortical sensory deficit, which lasted about 5 days. Three days after admission T2-weighted and gadolinium-enhanced T1-weighted MRIs showed a small, linear, high-signal intensity in the cortical and subcortical areas of the right parietal lobe (Figure 5). An angiogram was refused.

Discussion

As determined in previous investigations, the thalamus was the site most frequently involved in this series. Five patients showed an impairment of all sensory modalities including pinprick, touch, vibration, and position senses. Three patients had small infarcts (lacune) confined to the posterolateral part of the thalamus, and two had relatively large hemorrhages. Six patients with very small lacunes showed restricted sensory changes: paresthesia without objective sensory signs (cases 6, 7, and 9), mild impairment in pinprick sense only (case 10), and selective impairment in vibration and position senses (case 11).

Although thalamic strokes were usually reported to produce deficits of all sensory modalities, Landi et al described a patient with decreased pinprick sense only, whereas Sacco et al reported a patient with a selective loss of the position sense. Therefore, small lacunes strategically placed in the thalamus may cause tract-specific sensory deficits. According to previous studies, the medial lemniscal fibers are projected densely to the caudal division of the ventral posterolateral nucleus, whereas the spinothalamic fibers diffusely enter the posterior thalamic nucleus, the ventral posterolateral nucleus, and the central lateral nucleus. Presumably the small lacune in patient 11 was situated within the caudal part of the ventral posterolateral nucleus, but this structure was spared in patients 6, 7, 9, and 10.

The patients with very small lacunes tended to show restricted distribution of sensory abnormality, in the faciobrachial area in patients 6 and 7 and the distal fingertips in patient 11. Patients 8 and 9 showed so-called “cheiro-oral syndrome”: numbness restricted over the corner of the mouth, hard palate, and hand. The present study and the previously reported thalamic strokes with cheiro-oral sensory syndrome support the alleged topographic presentation in the ventrolateral nucleus of the thalamus in which sensory areas projected from face and hands are closely related, although why the first four toes were also affected in patient 9 remains unexplained.

There have been a few reports of PSS caused by capsular or corona radiata strokes. Groothuis et al suggested that to produce PSS, the lesion should occupy the posterior quarter of the posterior limb of the internal capsule, where sensory tracts without motor fibers are believed to be located. In the present study, four patients with lenticulocapsular stroke with PSS were identified: two (cases 12 and 13) had lacunes abutting the posterior quarter of the posterior limb, and two (cases 14 and 15) showed lesions bordering the middle of the posterior limb, suggesting that small lesions in this area could also escape the descending motor tract. Moreover, in this series three patients had lacunes in the corona radiata of the middle cerebral artery distribution. A similar patient with a corona radiata lacune in the middle cerebral artery territory was previously reported by Rosenberg and Koller, and Chamorro et al described a patient with PSS who had a CT-identified lacune in the anterior limb of the internal capsule.
It is noticeable that all of the patients in the present study with capsular or corona radiata stroke showed decreased pinprick and touch senses without impaired vibration or position senses. The patient described by Groothuis et al. who showed an additional deficit in the medial lemniscal sensory modality had lesions in the posterior part of the posterior limb adjacent to the thalamus, whereas the patients in the present study showed lesions situated in the lateral part of the internal capsule or corona radiata. Therefore, it may be postulated that the medial lemniscal sensory pathways run posteriorly and medially within the posterior limb of the internal capsule, whereas those of spinothalamic modalities ascend more laterally and diffusely into the corona radiata.

I observed two patients with brain stem stroke, one with a lacune and another with a hemorrhage in the pontine tegmentum. Both revealed sensory deficits of pure lemniscal modality caused by discrete lesions situated in the medial lemniscus. A patient with similar clinical features was previously described by Graveleau et al., but patients with isolated spinothalamic modality impairment or with only subjective sensory symptoms have also been described. Therefore, strategically located small brain stem strokes tend to produce sensory deficit of a single tract type. Recently, brain stem strokes causing chiro-oral-sensory syndrome were also described. 7, 8

Finally, I described a patient with cortical infarct who showed markedly impaired cortical sensation. Derouesné et al. reported a similar case with a small cortical infarct in the middle cerebral artery territory. It is unique and of interest that patient 21 had pure cortical sensory transient ischemic attacks. This patient’s abnormal posture during the sensory transient ischemic attack was probably due to a loss of position sense in the involved limbs.

In conclusion, the clinical-radiological correlation of 21 cases in this study strongly supports the previous assumption that strokes anywhere in the sensory tract can produce PSS. Sensory deficits of all modalities in one half of the body usually are associated with a relatively large lacune or hemorrhage in the area of the posterothalamic thalamus, whereas tract-specific or restricted sensory abnormalities usually correlate with a very small lesion located in the sensory pathway.

Acknowledgment

I wish to thank Professor Onyou Hwang for her helpful suggestions in preparing this manuscript.

References

Pure sensory stroke. Clinical-radiological correlates of 21 cases.

J S Kim

*Stroke.* 1992;23:983-987
doi: 10.1161/01.STR.23.7.983

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

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/23/7/983

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/