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
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 dysesthesia over the same area as patient 8 but 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 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

### 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>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>F/52</td>
<td>HT</td>
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<td>+</td>
<td>+</td>
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<td>2</td>
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<td>CHD, APL</td>
<td>L Thal</td>
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<td>+</td>
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<td>+</td>
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<tr>
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<td>S</td>
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<td>L Thal</td>
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<td>L CR</td>
<td>VS</td>
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<td>F/55</td>
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<td>R Parietal</td>
<td>S</td>
<td>-</td>
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</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.
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 hemiparesthesia 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 hemiparesthesia 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.
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.\(^\text{24}\) described a patient with decreased pinprick sense only, whereas Sacco et al.\(^\text{27}\) 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.\(^\text{27}\) 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\(^\text{13,26–28}\) support the alleged topographic presentation in the ventro-lateral nucleus of the thalamus\(^\text{34}\) 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. Groothius et al.\(^\text{17}\) 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,\(^\text{23}\) and Chamorro et al.\(^\text{4}\) 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 mediodiencephalic. 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 cheiro-oral sensory syndrome were also described.

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 posterolateral thalamus, whereas tract-specific or restricted sensory abnormalities usually correlate with a very small lesion located in the sensory pathway.

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References

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