Case Report

Selective Proprioceptive Loss From a Thalamic Lacunar Stroke

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In an elderly woman a small thalamic infarct, documented by computed tomography and nuclear magnetic resonance imaging, caused proprioceptive loss contralaterally without impairment of other sensory modalities. This patient, the first so reported, demonstrates the anatomic separation of spinothalamic and dorsal column/medial lemniscus sensory modalities in the human thalamus. (Stroke 1987;18:1160-1163)

Pure sensory stroke was first described in 1965; the lesion was in the thalamus. In similar cases responsible infarcts and hemorrhages were localized by computed tomography (CT) or autopsy to the thalamus, thalamocortical radiations, thalamocortical projection area, midbrain, or corona radiata. Symptoms included transient or persistent numbness or paresthesias, and there was either normal or decreased sensation in the face, arm, and leg. Spinothalamic modalities have been more often impaired than dorsal column modalities. We describe a unique case in whom unilateral proprioceptive loss was the only clinical abnormality after a contralateral thalamic infarct.

Case Report

A 91-year-old right-handed woman with a history of hypertension, syncope, and premature ventricular contractions suddenly developed difficulty using her left side. She could not rise from a chair without falling, and she could not walk. She believed that she was weak in the left arm and leg and denied any sensory symptoms.

On admission, blood pressure was 180/90 mm Hg and pulse was 70 beats/min with occasional skipped beats. There was a 2/6 apical systolic ejection murmur. She was alert and oriented with normal speech, repetition, naming, reading, writing, and comprehension. Memory was poor with no recall of 3 objects after 10 minutes. There was no neglect, agnosia, asomatognosia, or constructional apraxia. Cranial nerve functions including visual fields, eye movements, and facial sensation were normal. There was downward drift of the left arm. Strength was difficult to assess; left arm and leg muscles tended to give way abruptly, and performance was better when she looked at the limb being tested. Tendon reflexes were less active on the left, and there was a left Babinski’s sign. Sensation to light touch, pain, cold, and vibration was exquisitely normal on the right and left, but proprioception was severely impaired on the left, even at the shoulder and hip. Stereognosis and graphesthesia were poor in the left hand. There was no extinction to double simultaneous stimulation. Ataxia in the left arm was worse when she closed her eyes. She could stand alone, but because of unsteadiness she needed assistance to walk.

CT scan without contrast on admission showed cerebral atrophy. CT with and without contrast on the third hospital day disclosed areas of decreased attenuation in the right and left thalamus (Figure 1). An electroencephalogram showed diffuse mild slowing. Nuclear magnetic resonance imaging (T2 weighted) confirmed the right lateral thalamic lesion, as well as a left medial thalamic lesion and multiple foci of increased signal intensity in the periventricular white matter diffusely, consistent with subacute arteriosclerotic microangiopathy (Figure 2).

Except for a syncopal episode attributed to cardiac arrhythmia, she improved steadily and was transferred to the rehabilitation medicine service. At no time did she experience spontaneous pain or other “positive” sensory symptoms.

Discussion

This patient had a thalamic infarct with pure hemisensory loss consisting of severely impaired proprioception in the absence of lateral spinothalamic dysfunction on clinical examination. Her “weakness” and ataxia were probably due to the proprioceptive loss. In contrast to other reported patients, she had no symptoms of altered sensation, perhaps because there was no loss of pain, touch, or temperature perception.

Previous cases of pure sensory stroke have resulted
Some patients have had mild impairment of dorsal column modalities, usually with other sensory loss as well. In 4 patients proprioceptive loss accompanied loss of touch and pain sensation. In 6 patients spinothalamic modalities were involved but proprioception was spared. In only 1 other patient dorsal column modalities (proprioception, stereognosis, graphesthesia, 2-point discrimination) were involved with sparing of spinothalamic modalities (pain, temperature, light touch). That patient, in contrast to ours, noted subjective "numbness" in his hand and foot, and sensory loss was restricted to his fingers and toes; a parietal cortical and subcortical infarct was present. There have been no reported cases of proprioceptive loss without other sensory abnormalities after a thalamic stroke.

The left medial thalamic infarct, in the region of the centromedian and parafascicular nuclei, was not clinically evident. These nuclei receive afferent fibers mainly from forebrain derivatives and project principally to putamen. The right lateral thalamic infarct presumably involved that portion of the ventral thalamus that receives projections from the dorsal column/medial lemniscus, sparing spinothalamic projections.

Cytoarchitectural studies have demonstrated separate projections from these pathways to the ventral posterior lateral (VPL) nucleus of the thalamus. In addition to somatotopic organization of the ventroposterior nucleus of the thalamus with a dorsoventral shift corresponding to a proximal-to-distal representation of the body surface, there is a shift in receptive field properties. Neurons activated by pressure or joint movement are located in regions of the VPL nucleus distinct from those activated by light tactile stimuli.

Morphologic studies of the monkey thalamus have demonstrated that lemniscal fibers enter the diencephalon along the medial edge of the medial geniculate complex. Although there is some evidence of termination in the medial geniculate, pulvinar, and suprageniculate nuclei, the primary terminal zone is confined to the morphologically distinct caudal division of the ventral posterior lateral nucleus (VPLc). No terminals extend into the oral division (VPLo).

Spinothalamic fibers enter posteriorly to the medial lemniscus and bypass the medial geniculate complex, terminating more diffusely in the ventral, posterior, and intralaminar thalamic nuclei. As opposed to the dense homogeneous dorsal column termination in the VPL, there is an uneven sparse distribution in the outskirts of the VPL nucleus and no comparable abrupt stop at the VPLc–VPLo border.

This case provides a clinicopathologic correlation of the separation of sensory functions in the thalamus by demonstrating that a lacunar infarct strategically placed in the thalamus may cause discrete sensory mo-
FIGURE 2. Left: Heavily $T_2$-weighted nuclear magnetic resonance image (NMRI) 3 weeks after admission demonstrates right lateral thalamic infarct in region of the ventral posterior lateral nucleus (VPL). Right: Same NMRI examination at higher level demonstrates more medially placed left thalamic infarct in region of the centro-medial nucleus (CM). Bottom: Anatomical map of thalamic nuclei for correlation shows VPL and CM.
Table 1. Summary of Pure Sensory Stroke Lesions Identified by Autopsy or CT

<table>
<thead>
<tr>
<th>Author, year</th>
<th>Symptoms</th>
<th>Abnormal sensory signs</th>
<th>Stereography</th>
<th>Lesion site</th>
<th>Identification</th>
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<tbody>
<tr>
<td>Fisher, 65</td>
<td>Paresthesias</td>
<td>+ - - - -</td>
<td>VP thalamic infarct</td>
<td>Autopsy</td>
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<tr>
<td>Groothuis et al, 77</td>
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<td>Post-internal capsule hemorrhage, thalamocortical radiations</td>
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<tr>
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<td>Thalamic and occipital infarct</td>
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<tr>
<td></td>
<td>Numbness</td>
<td>+ + + + -</td>
<td>Thalamic hemorrhage</td>
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<tr>
<td></td>
<td>Numbness</td>
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<td>Thalamocortical radiations infarct</td>
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<td>Thalamic infarct</td>
<td>CT</td>
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</table>

Pp, pinprick; Lt, light touch; Vib, vibration; Jps, joint position sense; Stereo/graph, stereognosis and graphesthesia; +, impaired; −, unimpaired; ?, unknown.

Acknowledgments

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References


Key Words: pure sensory stroke • lacune • thalamus • cerebrovascular disease