Pure Sensory Stroke Caused by a Pontine Infarct
Clinical, Radiological, and Physiological Features in Four Patients

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Background and Purpose We conducted this retrospective study to evaluate the clinical, neuroradiological, and neurophysiological findings in patients with pure sensory stroke due to pontine lacuna.

Summary of Report Four patients with pontine lacuna, three men and one woman (mean age, 64.5 years; range, 55 to 75 years), were evaluated. Magnetic resonance images were obtained with a 0.5-T superconducting magnet using the SE technique. Short-latency somatosensory evoked potentials were evaluated by unilateral stimulation of the median nerve at the wrist. These tests were done at a mean of 22.5 months (range, 9 to 34 months) after symptom onset. Deep sensory disturbances were present in one half of the patient's body with no other neurological deficits found. The sensory deficit was characterized by a prolonged period of refractory dysesthesia and a discrepancy between the superficial and deep sensory disturbances. Lesions were localized in the medial lemniscus of the middle and lower pons, with a sparing of the spinothalamic tracts. The central conduction times of short-latency somatosensory evoked potentials were prolonged (patients 1, 2, and 4), and the amplitudes were significantly reduced (patients 1 and 2) when stimulating the paresthetic-sided median nerve (contralateral side of the lesion) compared with stimulating the other side.

Conclusions In all cases, the clinical and radiological findings indicated a dysfunction of the medial lemniscal tract in the pons. The observed somatosensory evoked potentials were probably related to the persistent refractory dysesthesias present in these patients. (Stroke. 1994;25:1512-1515.)

Key Words • evoked potentials, somatosensory • pons • magnetic resonance imaging

Pure sensory stroke is a well-defined clinical entity in which hemisensory symptoms predominate with the absence of other major neurological signs.1 Fisher1 attributed this syndrome to a lacunar infarct in the ventroposterior nucleus of the thalamus. Although thalamic stroke is the most frequent cause of pure sensory stroke, nonthalamic strokes involving the brain stem,2-5 internal capsule,6,7 or cerebral cortex8 are also reported to produce pure sensory stroke.9 We present the findings in four patients with pure sensory stroke due to pontine lacuna and discuss their characteristics.

Subjects and Methods

Four Japanese patients with pure sensory stroke who showed pontine lesions on magnetic resonance imaging (MRI) were diagnosed and treated at the Toride Kyodo General Hospital between June 1990 and September 1991. MRI was performed in each case with a 0.5-T superconducting magnet (Resona, Yokokawa Medical) using the SE technique. T1-weighted (repetition time [TR], 380 milliseconds; echo time [TE], 15 milliseconds) and T2-weighted (TR, 2000 milliseconds; TE, 100 milliseconds) scans were performed in the axial plane.

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The deficits in vibration and position senses gradually improved, but the refractory dysesthesia persisted in the left half of his body, especially in the upper limb and trunk, even though the patient had been treated with a variety of pharmacologic agents such as carbamazepine, imipramine, maprotiline, sulpiride, and mecobalamin. Two years later the dysesthesias were still present and severe enough to cause sleep disturbance, but he had learned to ignore them.

Fasting blood sugar (FBS) concentration was 100 mg/dL. Total cholesterol (TC), high-density lipoprotein (HDL) cholesterol, and triglyceride (TG) levels were 131 mg/dL, 48 mg/dL, and 82 mg/dL, respectively. The serum lipoprotein(a) (Lp[a]) concentration was 10 mg/dL.

SSEPs produced by stimulation of the median nerve were evaluated 28 months after the clinical onset. The latency of N13 (recorded from the fifth cervical vertebral electrode) was almost equal on both sides (15.3 milliseconds versus 15.2 milliseconds), but the central conduction time (CCT; N13 to N20) was prolonged (5.70 milliseconds versus 6.42 milliseconds) and the amplitude was significantly reduced when stimulating the left median nerve as compared with the right (Fig 2).

Case 2
A 63-year-old man with a history of hypertension developed numbness in the right half of his body early one morning. His blood pressure was found to be 160/90 mm Hg on the following day, when neurological examination revealed the presence of dysesthesia in the right face, arm, leg, and upper trunk. The dysesthesias were especially intense in the forehead, forearm, upper breast, and foot on the right side. The distortion of body schema was not present. There was a decrease in vibration and position senses in the upper breast and limbs on the right side, but the response to pinprick and temperature senses was essentially preserved. Motor function and reflexes were normal, and there was no cranial nerve palsy. Computed tomography (CT) on the same day showed normal findings, but 4 days later, T$_2$-weighted MRI (TR, 2000 milliseconds; TE, 100 milliseconds) demonstrated a lesion of high intensity in the left paramedian pontine tegmentum (Fig 3). Twelve months later the distribution and intensity of dysesthesias were gradually reduced, but the numbness was still present in the right forearm and toes despite treatment with a variety of pharmacologic agents such as carbamazepine, imipramine, mecobalamin, and mexiletine.

FBS concentration was 102 mg/dL. TC, HDL cholesterol, and TG levels were 155 mg/dL, 56 mg/dL, and 56 mg/dL, respectively.
mg/dL, respectively. The serum Lp(a) concentration was 52.8 mg/dL. SSEPs were performed 19 months after the clinical onset. The latency of N13 was almost equal on both sides (13.6 milliseconds versus 13.9 milliseconds), but the CCT (N13 to N20) was prolonged (7.02 milliseconds versus 6.30 milliseconds) and the amplitude was significantly reduced in response to stimulating the right median nerve versus the left.

Case 3
This 75-year-old man who had no history of hypertension suddenly developed numbness on the left cheek, tongue, and fingers when he was playing gateball (the favorite sport of the elderly in Japan). His blood pressure was 138/80 mm Hg on admission. The next day on admission, neurological examination revealed marked dysesthesia and a slight impairment of vibration and position senses in the corner of the mouth, tongue, palm, and thumb on the left side. The left fingers, especially the thumb, felt burned, and the palm felt full, swollen, and hot. The tongue and lip on the left side also felt burning dysesthesia. According to the distortion of body schema, he transiently perceived that his left arm was attached to the lumbar region for several days. Response to pinprick, touch, and temperature was normal. Motor paresis and cranial nerve palsy were absent. Reflexes were normal, and Babinski’s sign was absent. T1-weighted MRI (TR, 380 milliseconds; TE, 15 milliseconds) performed 2 weeks after symptom onset demonstrated a lesion of low intensity in the lower right pons (Fig 4). Three months later the numbness of the left fingers and palm gradually disappeared, but the dysesthesia and paresthesia in the corner of the mouth was still present. Fifteen months later the numbness of the face had gradually reduced.

FBS was 114 mg/dL. TC, HDL cholesterol, and TG levels were 194 mg/dL, 58 mg/dL, and 146 mg/dL, respectively. The serum Lp(a) concentration was 46.0 mg/dL. SSEPs performed 34 months after the onset of symptoms showed an essentially equal CCT (N13 to N20) and amplitudes on both sides.

Case 4
A 65-year-old woman with a history of hypertension had a sudden onset of numbness on the right side of the body, especially her right leg. No other symptoms were present. The neurological examination on the following day showed numbness involving the check, jaw, arm, breast, and leg on the right side. Numbness of the limbs was more severe distally than proximally. Vibration and position senses were slightly impaired in the right jaw, elbow, breast, and foot. The distortion of body schema was not present. There was bilateral hyperreflexia, but Babinski’s sign was absent. Motor function impairment and cranial nerve palsy were also absent. T1-weighted MRI (TR, 380 milliseconds; TE, 15 milliseconds) performed 1 week after the onset demonstrated a lesion of low intensity in the left pons (Fig 5). Two years later the dysesthesias of face, arm, and breast gradually disappeared, but the burning numbness of the right leg was still present.

FBS was 88 mg/dL. TC, HDL cholesterol, and TG levels were 185 mg/dL, 55.3 mg/dL, and 96 mg/dL, respectively. The serum Lp(a) concentration was 35.6 mg/dL. SSEPs were performed 9 months after the onset of symptoms. At that time the amplitude was almost equal on both sides, but the CCT (N13 to N20) was prolonged (6.48 milliseconds versus 6.18 milliseconds) in response to stimulation of the right median nerve compared with that on the left.

Discussion
The characteristics of sensory deficits in pure sensory stroke due to pontine infarct include a prolonged refractory dysesthesia and a discrepancy between superficial and deep sensory disturbances. In these patients, the dysesthesia was severe enough to cause sleep disturbances. The deficits of vibration and position senses gradually improved, but the refractory dysesthesia had been persistent for a long time. Fisher11 reported that the objective sensory disturbances were mild in the pure sensory stroke, and therefore endeavoring neurological examination revealed faint decreased sensations. Patients with pure sensory stroke suffered primarily from subjective sensory disturbances, such as dysesthesia and paresthesia, and suffered in part from objective sensory disturbances, such as position and vibration sense dis-
turbances. Fisher therefore suggested that the pure sensory stroke is mainly pure paresthetic stroke.11

Robinson et al.12 investigated SEPs in pure sensory stroke, and reported that SEPs were normal in all 11 cases of pure sensory stroke and that CT was normal in 10. In only one case did CT show a lesion in right thalamus.12 In our study we used MRI to detect small brain stem lesions. Because CT is inferior to MRI for detecting smaller and faint lesions, Robinson et al might have missed some brain stem, thalamic, and other lesions. Karnaze et al.13 evaluated 49 patients who had suffered hemispheric or thalamic ischemia and reported that SSEP abnormalities correlated with the presence of sensory deficit and the degree of neurological deficit. They discussed that the fact that CCT delays and absent N19-P22 components correlated strongly with the severity of the neurological deficit; the CCT therefore may prove to have prognostic value in stroke.13 In our SSEP studies, the CCTs were prolonged in cases 1, 2, and 4, and the amplitudes were significantly reduced in cases 1 and 2. These neurophysiological findings were probably related to the characteristics of sensory disturbances in pure sensory stroke, such as persistent refractory dysesthesia.

The other characteristic of sensory deficit in pure sensory stroke due to a pontine infarct is the discrepancy between superficial and deep sensory disturbances. In the four cases presented, vibration and position senses were decreased, but the responses to pinprick and temperature were essentially preserved. These clinical symptoms indicated a dysfunction of the medial lemniscal tract with no dysfunction of the spinothalamic tract. The lesions in our patients were localized in the medial lemniscus of the middle pons (cases 1, 2, and 4) and the lower pons (case 3), with sparing of the spinothalamic tracts. A discrepancy between the superficial and deep sensory disturbances is common in pure sensory stroke caused by a pontine lacuna. It is due to the anatomic location of the medial lemniscal tract, which is separate from the spinothalamic tract in thepons.14

Fisher14 analyzed 135 patients in which numbness, either episodic or persistent, involved one side of the body; he tentatively proposed that isolated paresthesias of the face, arm, and leg indicate thalamic involvement and that numbness of cerebrovascular brain stem origin does not assume any of these patterns. In our patients with brain stem lesions, the dysesthesias involved face, arm, leg, and breast in cases 1, 2, and 4. In case 1, the dysesthesias extended to neck, shoulder, and abdomen. The characteristic of syndrome-lesion correlation in pure sensory stroke due to pontine infarct may be the distribution of its sensory disturbance. The thalamic lesion causes isolated sensory involvement of all three parts, face, arm, and leg.15 The pontine lesion causes sensory involvement that includes trunk (breast and abdomen) as well as face, arm, and leg. The lesion in case 3 was quite large, but the syndrome was quite circumscribed. Case 4 had a small lesion with wide-spread syndrome and greatest complaints in the leg. The cause of this disproportion between syndrome and lesion remains unknown, but the neurophysiological findings by SSEP may be related to this condition. SSEPs showed normal findings in case 3, but in case 4 the CCT was prolonged when stimulating the paresthetic-sided median nerve. SSEPs correlate with the severity of the neurological deficit and sensory abnormalities after cerebral ischemia.13 Case 3, a 75-year-old man, manifested a cheiro-oral syndrome, which is typically provoked by lesions located in the thalamus and cortex. Takezawa et al.14 reported a 69-year-old woman with a cheiro-oral syndrome caused by a pontine lacuna. The onset of the brain stem cheiro-oral syndrome is thought to involve the medial lemniscus, which has a body-localization sequence in the upper and lower limbs.17 It spreads horizontally on both sides of the paramedian pontine tegmentum. The ascending fibers from the lower limb are located on the outside, while those from the upper limb are located on the inside. The abdominal pathway of the trigeminal nerve, which transmits the epicritic sensation of the face, ascends close to the rear inside of these fibers.17

References


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