J.H.	Ji Hoe	Heo
B.O.	Byung Ok	Choi

Department of Neurology, Yonsei University College of Medicine, Seoul, Korea

Hemiataxia-Hypesthesia in Thalamic Hemorrhage Significance of Sensory Deficit Patterns and Presence or Absence of Weakness

Ji Hoe Heo, MD, Department of Neurology, Yonsei University College of Medicine, CPO Box 8044, Seoul (Korea)

Hemiataxia-hypesthesia without weakness has rarely been reported in thalamic hemorrhage [1–3]. We report 2 patients with thalamic hemorrhage who showed hemiataxia-hypesthesia without weakness and who demonstrated different patterns of sensory deficit. We also discuss the significance of sensory deficit patterns as well as the significance of the presence or absence of weakness towards localizing a thalamic lesion.

Case Reports

Patient 1. A 59-year-old right-handed hypertensive man had sudden loss of consciousness for 2 h. His case history included an operation 12 years prior due to an aneurysmal rupture, and an intracerebral hemorrhage 2 years prior, which had resulted in minimal right spastic hemiparesis. Two days later, at admission, he complained of headache, dizziness, and blurred vision. He was alert and well oriented, but showed dysarthria. Although there were mild ptosis and miosis in the left eye, both pupils were reactive to light. Skew deviation was noticed, and the right eyeball was limited in its ability to gaze upward. Muscle power and tone were both normal throughout the left side of his body. Although a sensory examination revealed a mild decrease in pain and light touch sensations on the left side of his body, position and vibration sensations were normal. He showed a tendency to fall to the left side. Both the finger to nose test and the heel to shin test revealed marked incoordination and terminal tremor on his left side. Rapid alternate movement in the left hand was severely impaired. A brain CT scan revealed a hemorrhage in the right thalamus (fig. 1a). The patient improved gradually. One year later, he showed mild hypesthesia and moderate cerebellar ataxia on his left side.

Patient 2. A 67-year-old right-handed diabetic man developed sudden headache, dizziness, numbness in the left hand, and equilibrium disturbance. Three days later, at admission, muscle power, deep tendon reflexes, and muscle tone were normal. But sensory examination revealed a slight decrease in pain, light touch, position and vibration sensation on his left side. Rapid alternate movement in the left hand showed mild dysdiadokokinesia. He showed a tendency to fall to the left side during straight-line walking tests. A brain CT scan revealed a hemorrhage in the right thalamus (fig. 1b). Two weeks later, he showed neither dysdiadokokinesia nor a tendency to fall. A sensory examination revealed improved but still mild left hemihyp-esthesia.

Discussion

Our 2 patients showed hemiataxia-hypesthesia without weakness, although the clinical picture of patient 1 included thalamic-subthalamic dysfunction. Interestingly, they showed different patterns of sensory deficits. Patient 1 revealed a reduction in pain and touch

Fig. 1. Both computerized tomographic scans of patients 1 (a) and 2 (b) show a high-density lesion in the thalamus, consistent with a hemorrhage.

Short Reports

243 sensation, while patient 2 showed decreased sensation in pain, touch, vibration and position. Upon reviewing previously reported cases of thalamic ataxia, in addition to our 2 cases, deficits in pain sensation were found to be the most common pattern, with all but one patient showing such a pattern, while deficits in proprioceptive sensations were found to be the least common [1–8]. This disparity strongly indicates that the spinothalamic pathway was more frequently involved than the lemniscal pathway in these patients. Anatomical studies have shown that the spinothalamic terminations overlap the cerebellorubrothalamic pathway in the VPLo nucleus, while the lemniscal pathway, which terminates in the VPLc nucleus, does not [9]. These anatomical findings explain why patients with thalamic ataxia have sensory deficits more frequently in pain than in proprioception. Thus, when an acute stroke patient with ataxia does not show a deficit in pain sensation, it is unlikely that the lesion is in the thalamus.

Although Melo and Bogousslavsky [4] and Solomon et al. [2] suggest that a hemiataxia-hypesthesia has a localizing value in thalamic strokes, Solomon et al. [2] do not differentiate between patients with weakness and those without. However, hemiataxia-hypesthesia with weakness has been reported in lesions in the thalamus as well as in the internal capsule [8] and parietal lobe [10]. In the acute stage of a stroke, therefore, hemiataxia-hypesthesia with transient weakness by the thalamic lesion may be indistinguishable from that by the capsular lesion. We believe that all reported cases with hemiataxia-hypesthesia without weakness have had lesions confined to the thalamus [1–8]. We therefore suggest that hemiataxia-hypesthesia without weakness has a higher localizing value than that with transient weakness.

In conclusion, in stroke patients with ataxia, we suggest that an ataxia associated with deficit in pain sensation without weakness is more suggestive of and specific to a thalamic lesion than that with other sensory deficit patterns, or with weakness, or with both.

References

Gutrecht JA, Zamani AA, Pandya DN: Lacunar thalamic stroke with pure cerebellar and proprioceptive deficits. J Neurol Neurosurg Psychiatry 1992;55:854–856.
Solomon DH, Barohn RJ, Bazan C, Grissom J: The thalamic ataxia syndrome. Neurology 1994;44:810–814.
Verma AK, Maheshwari MC: Hypesthetic-ataxic-hemiparesis in thalamic hemorrhage. Stroke 1986;17:49–51.
Melo TP, Bogousslavsky J: Hemiataxia-hypesthesia: A thalamic syndrome. J Neurol Neurosurg Psychiatry 1992;55:581–584.
Caplan LR, DeWitt LD, Pessin MS, Gorelick PB, Adelman LS: Lateral thalamic infarcts. Arch Neurol 1988;45:959–964.
Melo TP, Bogousslavsky J, Moulin T, Nader J, Regli E: Thalamic ataxia. J Neurol 1992;239:331–337.
Lee NS, Roh JK, Myung HJ: Hypesthetic ataxic hemiparesis in thalamic lacune. Stroke 1989;20:819–821.
Helgason CM, Wilbur AC: Capsular hypesthetic ataxic hemiparesis. Stroke 1990;21:24–33.

Asanuma C, Thach WT, Jones EG: Distribution of cerebellar terminations and their relation to other afferent terminations in the ventral lateral thalamic regions of the monkey. *Brain Res Rev* 1983;5:237–265.

10 Yagnik PM, Dhaduk V, Huen L: Parietal ataxic hemiparesis. *Eur Neurol* 1988;28:164–166.