

Hypesthetic Ataxic Hemiparesis in a Thalamic Lacune

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Right hemiparesis with an ipsilateral hypesthesia and ataxia developed in a 57-year-old man. Magnetic resonance imaging showed a left thalamic lacune bordering the medial portion of the posterior limb of the internal capsule. This finding implicated some pathogenetic mechanism of ataxic hemiparesis. (*Stroke* 1989;20:819–821)

Fisher designated the syndrome of weakness and pyramidal signs on one side combined with an ipsilateral cerebellar-like ataxia as ataxic hemiparesis.¹ In several subsequent reports, the upper pons, posterior limb of the internal capsule, corona radiata, midbrain, thalamus, and parietal lobe have been demonstrated as sites of the lesions.^{1–10} Since the classic thalamic syndrome was first described by Dejerine and Roussy,¹¹ a few cases of ataxic hemiparesis from contralateral thalamic lesions have been reported.^{7–9,12,13} We report a case of hypesthetic ataxic hemiparesis with a lacunar infarct in the contralateral thalamus as seen on magnetic resonance imaging (MRI).

Case Report

A 57-year-old man was admitted to Seoul National University Hospital for a poorly controlled blood sugar level. He did not smoke or drink, but the patient had been suffering from diabetes mellitus for 5 years. His blood pressure was 140/90 mm Hg, and diabetic retinopathy was detected in both fundi. The rest of his neurologic examination, other than a moderately impaired vibratory sensation in both feet and hypoactive deep tendon reflexes, was normal. While his electrocardiogram and serum albumin level (3.9 g/dl) were normal, proteinuria (1103 mg/day) and glucosuria were detected. His fasting and 2-hour-postprandial blood glucose levels were 222 and 322 mg/dl, respectively. This high blood glucose level was controlled with an oral hypoglycemic agent.

In the evening of the 14th hospital day when he was preparing for a stroll, a tingling sensation suddenly developed in his right arm and leg, fol-

lowed by weakness and clumsiness in his right hand. On examination he was alert and well-oriented with a normal neuropsychological assessment. There was a mild right-sided weakness of his involved limbs mainly in his arm, but no facial weakness was observed. Heel-to-shin and great toe-to-finger tests showed a marked ataxia on the right side, and the patient had some difficulty in accomplishing the finger-to-nose test due to a mild dysmetria. Impairment in rapid alternate movements of his right limbs and in the vibratory sensation and joint position of his right hand and foot were elicited, while touch and pain sensations were reduced by 40–50% in his right trunk, arm, and leg and on the right side of his face. As observed previously, on the left side only the vibratory sensation of his foot was reduced. Sensation approached normal near the midline of his trunk. On walking, the patient staggered and nearly fell to his right, but no cerebellar tremor or truncal ataxia were evident. His tendon reflexes remained symmetrically decreased, but his right plantar response was extensor. A brain computed tomogram taken approximately 15 hours later (Figure 1) showed an area of decreased attenuation in the left thalamus.

The following day his weakness began to improve gradually, but the ataxia and hypesthesia did not. On the 14th day after the onset of symptoms, the patient complained of an even more severe tingling sensation and pain on his right side. A T2-weighted MRI (repetition time 3000 msec, echo time 80 msec) taken on the 20th day (Figure 2) showed a high-signal lesion in the left thalamus bordering the adjacent medial portion of the posterior limb of the internal capsule. There were no additional high-signal lesions in the supratentorial area or in the posterior fossa. Two months later, the patient regained his strength and his toes were again flexor; however, he continued to complain of painful paresthesia on his right side, especially of his thigh. Ataxia of his right leg also remained.

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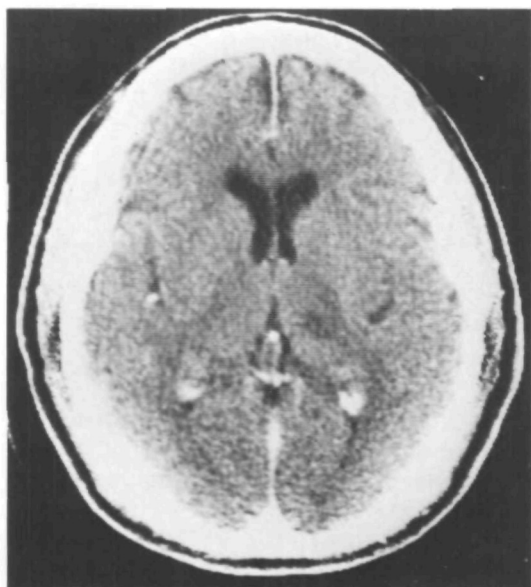


FIGURE 1. Computed tomogram shows low-density area in left thalamus of 57-year-old man with right hemiparesis and ipsilateral hypesthesia and ataxia.

Discussion

Since Fisher and Cole¹⁴ described the homolateral ataxia and crural paresis observed in 14 stroke patients, varieties of pathologic processes such as infarction, hemorrhage, and tumor have been identified as causes of the lesions,^{3,4,8,15-17} with the upper pons, posterior limb of the internal capsule, corona radiata, midbrain, thalamus, and parietal lobe strongly suspected of being sites of the lesions.^{2-10,12,13,18} Thus far, many different mechanisms for ataxia have been postulated, depending on the site involved. Involvement of the cell bodies and synaptic structure in the basis pontis is thought to be responsible for contralateral ataxia,¹⁸ whereas

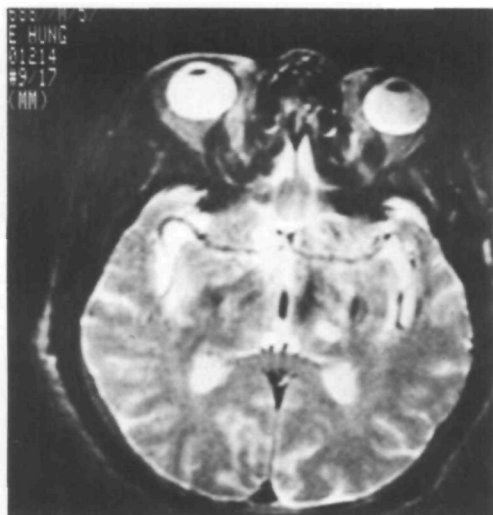


FIGURE 2. Magnetic resonance image shows high-signal lesion in left thalamus of 57-year-old man with right hemiparesis and ipsilateral hypesthesia and ataxia.

interruption of the descending corticospinal fibers and the cerebellar tract in and around the red nucleus may be responsible in midbrain lesions.^{3,14} In capsular lesions, involvement of the corticopontine fibers or the dentatorubrothalamocortical pathway in the posterior limb of the internal capsule may cause ataxia,^{5,6} whereas ataxia with parietal lobe infarction might be related to impaired proprioception, lack of spatial orientation, or destruction of the corticopontine fibers.¹⁰

In the case of a thalamic lesion, it has been postulated that an isolated lesion of the ventrolateral nucleus is responsible for contralateral ataxia,⁷ but involvement of the corticopontine fibers in the posterior limb of the internal capsule might also contribute to ataxia.

In our case, MRI showed a high-signal lesion in the ventral nuclear group of the left thalamus abutting the adjacent posterior limb of the internal capsule. There seems to be a very close similarity between our MRI findings and the autopsy findings described by Mohr et al¹⁹ of a lacune in the ventral posterior nucleus of the left thalamus with pallor in the adjacent internal capsule. Furthermore, our case suggests that ataxia may be related to the lesion involving the dentatorubrothalamocortical pathway in the ventral nucleus of the thalamus and partly to extension to the corticopontine fibers in the internal capsule. Hypesthesia must have been caused by the lesion in the ventral posterolateral and ventral posteromedial nucleus of the thalamus, whereas the transient hemiparesis may have been the result of transient edema involving the posterior limb of the internal capsule.

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References

1. Fisher CM: Ataxic hemiparesis. *Arch Neurol* 1978;35:126-128
2. Huang CY, Lui FS: Ataxic hemiparesis, localization and clinical features. *Stroke* 1984;15:363-366
3. Benhaum PE, Berg BO: Ataxic hemiparesis from a midbrain mass. *Ann Neurol* 1987;9:405-407
4. Perman GP, Racy A: Homolateral ataxia and crural paresis. *Neurology* 1980;30:1013-1015
5. Ichikawa K, Tustumishika A, Fujoka A: Capsular ataxic hemiparesis. *Arch Neurol* 1982;39:528-529
6. Iragui VJ, McCutchen CB: Capsular ataxic hemiparesis. *Arch Neurol* 1982;39:528-529
7. Bogousslavsky J, Regli F, Ghika J, Feldmeyer JJ: Painful ataxic hemiparesis. *Arch Neurol* 1984;41:892-893
8. Verma AK, Mahe Shwarl MC: Hypesthetic-ataxic-hemiparesis in thalamic hemorrhage. *Stroke* 1986;17:49-51
9. Murthy JMK: Ataxic hemiparesis—Ventrolateral nucleus of the thalamus: Yet another site of lesion (letter). *Stroke* 1988;19:122
10. Yagnik PM, Dhaduk V, Huen L: Parietal ataxic hemiparesis. *Eur Neurol* 1988;28:164-166
11. Dejerine J, Roussy G: Le syndrome thalamique. *Rev Neurol* 1906;14:521-532

12. Garcin R: Syndrome cérébellothalamique par lésion localisée du thalamus. *Rev Neurol* 1955;93:143–149
13. Garcin R, Lapresle J: Incoordination cérébelleuse du membre inférieur par lésion localisée de la région interne du thalamus contralatéral. *Rev Neurol* 1969;120:5–13
14. Fisher CM, Cole M: Homolateral ataxia and crural syndrome: A vascular syndrome. *J Neurol Neurosurg Psychiatry* 1968;27:48–55
15. Kobatake K, Shinohara Y: Ataxic hemiparesis in patients with primary pontine hemorrhage. *Stroke* 1983;14:762–764
16. Mori E, Yanadori A, Kudo Y, Tabuchi M: Ataxic hemiparesis from small capsular hemorrhage. *Arch Neurol* 1984;41:1050–1053
17. Fisher CM: Lacunar strokes and infarcts. *Neurology* 1982;32:871–876
18. Nabatame H, Fukuyama H, Akingnchi I, Kameyama M, Nishimura K, Torizuka K: Pontine ataxic hemiparesis studied by a high-resolution magnetic resonance imaging system. *Ann Neurol* 1987;21:204–207
19. Mohr JP, Kase CS, Meckler RJ, Fisher CM: Sensorimotor stroke due to thalamo-capsular ischemia. *Arch Neurol* 1977;34:739–741

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