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Ataxic Hemiparesis in Patients with Primary Pontine Hemorrhage

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SUMMARY Two patients with small primary pontine hemorrhage developed a syndrome identical to ataxic hemiparesis, one of the lacunar syndromes of Fisher. The possible mechanisms of the homolateral cerebellar signs, pyramidal signs, and dysarthria are discussed. Lesions of the ipsilateral pontine nuclei may be responsible for the homolateral ataxia.

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IN 1965, Fisher and Cole¹ described a cerebrovascular syndrome, they called homolateral ataxia and crural paresis, characterized by cerebellar-like ataxia, weakness and pyramidal signs involving the limbs of the same side, the leg more than the arm. In 1978, based on the clinicopathological results in three patients with this syndrome, Fisher² identified an old infarct cavity in the basis pontis at the level of the junction of the upper one-third and lower two-thirds on the side opposite the neurological deficit as the causative lesion. He also proposed the designation "ataxic hemiparesis" for this syndrome. Although this syndrome has been recognized as one of the lacunar syndromes of Fisher et al.,^{1, 3-5} lesions other than lacunes, such as tumors or demyelinating processes, could conceivably produce the syndrome.

Recently we examined 2 patients with primary pontine hemorrhage, that had a syndrome compatible with the ataxic hemiparesis of Fisher.

Case Report

Case 1

A 55-year-old hypertensive man, noted the sudden onset of dysarthria and weakness of the left leg and the left arm following occipital heaviness and nausea on November 3, 1981. He was unable to walk because of unsteadiness. Three hours later he was admitted to Tokai University Hospital. On admission his blood

pressure was 160/110 mm Hg. He was alert and well oriented. The neck was supple. The left eye was artificial due to trauma in his youth; visual fields and extraocular movement of his right eye were normal. No pupillary abnormalities were detected. There was fine horizontal nystagmus on left lateral gaze. Speech was dysarthric. The nasolabial fold on the left was flattened; mild left hemiparesis was present particularly in the distal portion of the left leg. Deep tendon reflexes were normal, but Babinski sign was positive on the left. Sensation was normal. There was left dysidiadochokinesis, and the finger-nose-finger test, shin-tapping test and heel-shin test showed impairment out of proportion to his weakness.

CT scan on the day of admission showed a small recent hematoma in the right dorsal part of the rostral basis pontis (fig. 1). ECG and EEG examination were normal. Retrograde vertebral angiography was normal.

After admission, nystagmus rapidly disappeared, and the dysarthria and the left hemiparesis decreased in severity. CT scan on November 26 was now normal. Though the ataxia on the left side gradually improved, slight cerebellar signs still persisted with positive Babinski sign at discharge (December 7, 1981).

Case 2

This 39-year-old woman had been well until the evening of March 20, 1982, when she noticed dysarthria following a floating sensation for several minutes. She could not pick up a coin using her left hand because of clumsiness. Shortly afterwards, she noticed unsteadiness in walking. On admission, about four hours after onset, her blood pressure was 180/110 mm Hg. She was alert and well oriented. Neck was supple. Oculomotor functions and pupils were normal. The left nasolabial fold was shallower and the tongue devi-

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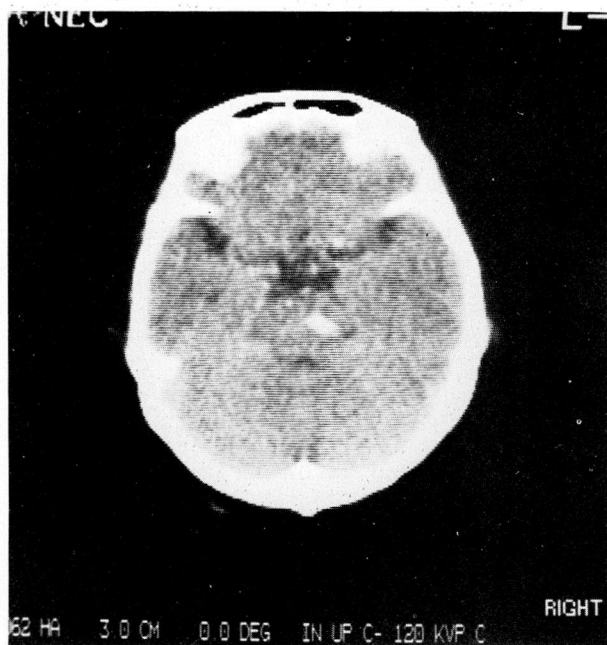


FIGURE 1. Unenhanced CT scan of case 1 on the day of admission shows a small fresh hematoma located in the right dorsal part of the rostral basis pontis.

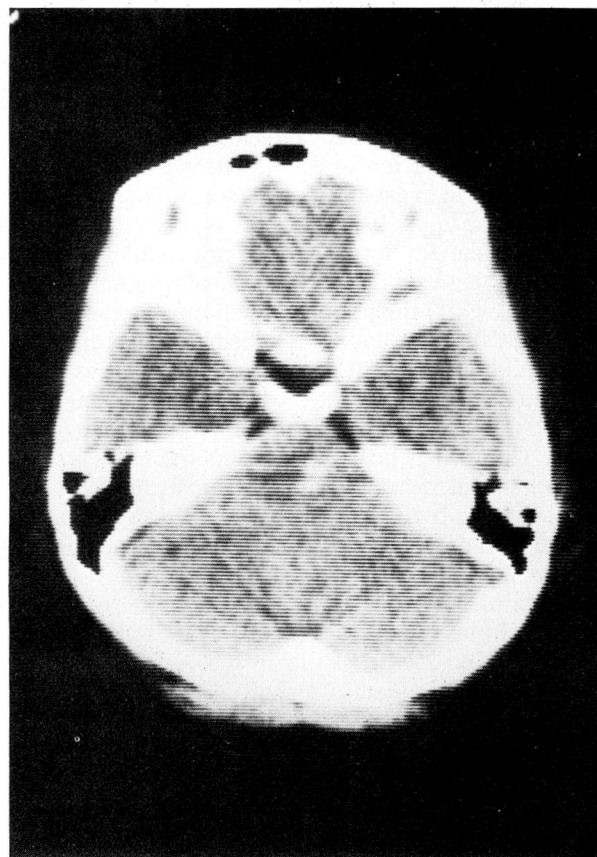


FIGURE 2. Unenhanced CT scan of case 2 on the day of admission shows a small hematoma located in the right dorsolateral aspect of the pons.

ated to the left. Her speech was slightly dysarthric but swallowing was normal. Sensation was intact. There was slight weakness of the left upper and lower extremities. Deep tendon reflexes were symmetrically hypoactive but Babinski sign was positive on the left. The finger-nose-finger test revealed terminal hesitation on the left side, and the shin-tapping test and heel-shin test were slightly ataxic on the left, showing more impairment than would be expected from her weakness. She could not stand or walk without support.

CT scan, on the day of admission, showed a small localized high density area in the right dorsolateral aspect of the junction of the upper and the middle basis pontis (fig. 2); no cerebellar or supratentorial lesions were detected. Angiographic study of the vertebrobasilar system revealed no pathologic findings.

After admission her neurological symptoms and signs improved. CT scan obtained on March 29 showed resolution of the high density lesion in the pons. In early April the left hemiparesis and the ataxia on the same side were markedly improved, and she was discharged without residual neurological deficits on April 10, 1982.

Discussion

Case 1 was the first report of ataxic hemiparesis resulting from small pontine hemorrhage.* Although ataxic hemiparesis has been recognized as one of the

lacunar syndromes,⁷ we emphasize that small pontine hemorrhage must be considered, as well as small infarctions, tumors or demyelinating lesions, as a possible causative lesion of ataxic hemiparesis.

The number of patients with ataxic hemiparesis in whom the responsible lesion had been confirmed by autopsy or by CT scan is quite small.^{2, 8-11} The possible lesions already proposed are small infarction at the upper basis pontis,^{2, 9} or at the superior portion of the posterior limb of the internal capsule^{8, 11} or mass lesion in the rostral midbrain.¹⁰ Although it has been suggested that small infarctions at the posterior limb of the internal capsule could produce ataxic hemiparesis on the basis of CT scan,^{8, 11} there might be small coexistent infarctions in the brainstem which could not be detected by conventional CT scans. Bendheim and Berg¹⁰ reported that interruption of the corticospinal tract and cerebellofugal pathways by a mass lesion situated at the ventrolateral aspect of the rostral midbrain could also produce contralateral hemiparesis and cerebellar signs.

The second problem concerning ataxic hemiparesis is the mechanism of the development of homolateral pyramidal and cerebellar signs resulting from one small lesion. How can a unilateral lesion of the basis pontis produce only contralateral cerebellar signs? Regarding the mechanism underlying the contralateral

*After we reported Case 1 at the 80th Regional Meeting (Kanto district) of Japanese Society of Neurology and we prepared the paper for publication including Case 2 which we examined immediately after the Meeting, Schnapper⁶ reported a case with pontine hemorrhage presenting as ataxic hemiparesis in this Journal.

cerebellar signs, Fisher² speculated that either the pontine nuclei sending fibers to the opposite cerebellum were damaged or crossing fibers from the opposite pontine nuclei were interrupted, but he did not conclude why the cerebellar signs were not bilateral in either case. In each of Fisher's cases the lesion was very small, and in each of our cases too, the hematoma located in the upper basis pontis was rather small. The reasons for the development of contralateral cerebellar signs from these small lesions in the basis pontis might be as follows; 1) the pontine nuclei may be more vulnerable or they are more localized than the crossing fibers, and/or 2) ipsilateral cerebellar signs resulting from the interruption of the crossing fibers are apt to be promptly compensated for by the majority of the intact fibers. The former possibility seems more likely, and thus we consider that a lesion of the ipsilateral pontine nuclei is probably responsible for homolateral ataxia. Further clinico-pathological studies are necessary to confirm this hypothesis.

The third interesting problem is the side of lesion and dysarthria. The lesions in all 3 cases of Fisher and in the present 2 cases were in the right brainstem. Among those 5 patients, 4 showed dysarthria, while ataxic hemiparesis due to left brainstem lesions¹⁰ did not reveal speech disturbance. This is in agreement with the results of Lechtenberg and Gilman,¹² who

suggested that speech function was most commonly affected by damage to the left cerebellar hemisphere.

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