Ataxic Hemiparesis Following Thalamic Infarction

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We describe a 73-year-old man with ataxic hemiparesis following infarction of the ventrolateral nucleus of the thalamus demonstrated by computed tomography and magnetic resonance imaging. Cerebellar ataxia was most likely due to interruption of the dentatorubrothalamicocortical fibers at the level of the injured ventrolateral nucleus. Hemiparesis was probably caused by local edema compressing the corticospinal tract in the adjacent posterior limb of the internal capsule. We believe this to be the first reported case of classic ataxic hemiparesis following thalamic infarction. (Stroke 1990;21:339–340)

Ataxic hemiparesis, first described by Fisher and Cole in 1965, consists of hemiparesis and cerebellar ataxia on the same side of the body. Ataxic hemiparesis is usually caused by lacunar infarction in the contralateral posterior limb of the internal capsule, in the pons, or in the corona radiata but may also be caused by a hemorrhage or a tumor. We report a case of ataxic hemiparesis in which computed tomography (CT) and magnetic resonance imaging (MRI) showed an infarct in the contralateral ventrolateral nucleus of the thalamus.

Case Report

A 73-year-old man suddenly developed dysarthria and weakness of his right arm and leg with unsteadiness of gait. There was no history of hypertension, diabetes, cardiac disease, or peripheral vascular disease. On examination the next day, he had a regular pulse of 76/min. Blood pressure was 150/80 mm Hg. There were no carotid bruits, and cardiac examination was normal. He was alert and fully oriented.

Neurologic examination showed a slight right-sided hemiparesis with an extensor plantar response. Finger-to-nose and heel-to-shin tests showed cerebellar-type right-sided dysmetria, hypermetria, and intention tremor out of proportion to the degree of weakness. Sensation was normal. His hemiparesis disappeared after 4–5 days, while his ataxia persisted. After 4 weeks there was only clumsiness of his right hand, with slight ataxia on the finger-to-nose test. Results of electrocardiography, 24-hour electrocardiographic (Holter) monitoring, echocardiography, and duplex carotid sonography were normal. Serum cholesterol concentration was slightly elevated.

CT scan on day 4 revealed a hypodense lesion in the ventrolateral nucleus of the contralateral thalamus, compatible with a recent infarction (Figure 1). MRI 1 year later showed the same infarct in the left thalamus (Figure 2). There were no other lesions visible in the internal capsule, corona radiata, brainstem, or cerebellum.

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FIGURE 1. Computed tomogram showing recent hypodense lesion in ventrolateral part of thalamus, with involvement of ventrolateral nucleus. Right side of body appears on left side of figure.
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FIGURE 2. Magnetic resonance image 1 year later showing same lesion in left thalamus.

Discussion

"Painful ataxic hemiparesis" following thalamic infarction and "hypesthetic ataxic hemiparesis" in thalamic hemorrhage and infarction have been reported.\(^8\)\(^-\)\(^10\) Sole unilateral cerebellar ataxia has been described following contralateral thalamic infarction.\(^11\)\(^-\)\(^12\) Murthy\(^13\) described a case of ataxic hemiparesis following thalamic infarction, but the lesion appeared to be located in the head of the caudate nucleus instead of in the thalamus.\(^14\) CT and MRI in our patient showed an infarct in the contralateral thalamus. To our knowledge, this is the first reported case of classic ataxic hemiparesis following infarction strictly confined to the thalamus.

The lesion was located in the ventrolateral part of the thalamus, with involvement of the ventrolateral nucleus. From experimental evidence it was concluded that the ventrolateral nucleus receives fibers from the contralateral cerebellar dentate nucleus (the dentatorubrothalamic projection).\(^15\) From the ventrolateral nucleus, fibers run to the sensorimotor cortex.\(^15\) Cerebellar ataxia in our patient was most likely caused by interruption of the dentatorubrothalamic corticospinal fibers at the level of the injured ventrolateral nucleus. Hemiparesis was probably caused by initial local edema compressing the adjacent corticospinal tract in the posterior limb of the internal capsule because MRI did not reveal involvement of the internal capsule. The hemiparesis clearing much more rapidly than the hemiataxia supports this assumption.

Our case illustrates that the ventrolateral part of the thalamus takes part in the cerebellar projection to the sensorimotor cortex, disruption of which at different levels can induce the classic lacunar syndrome of ataxic hemiparesis.

References


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