Case Reports

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)

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taxic hemiparesis, first described by Fisher and Cole1 in 1965, consists of hemiparesis and cerebellar ataxia on the same side of the body.2 Ataxic hemiparesis is usually caused by lacunar infarction in the contralateral posterior limb of the internal capsule, in the pons, or in the corona radiata1-4 but may also be caused by a hemorrhage or a tumor.5-7 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.
从对侧的背外侧核通过前庭小脑-丘脑皮层纤维传递到对侧的皮质区域。这一纤维束通过丘脑损伤阈而受损。急性期时，由于对侧皮质锥体束的压迫，可出现表现为对侧的肢体偏瘫，而对侧小脑性共济失调暂时性的缓解。作者的病例中，对侧的皮质锥体束可能由于对侧小脑性共济失调而受压迫，且MRI显示没有涉及皮质锥体束。需要进一步的研究来明确这一临床症状的机制。
Ataxic hemiparesis following thalamic infarction.
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