

Parietal Ataxic Hemiparesis

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Key Words

Ataxia
Hemiparesis
Parietal lobe

Abstract

Ataxia in the parietal lobe is rarely described. A case of ataxia hemiparesis due to a parietal lobe infarction is described and pathophysiology is discussed
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A combination of ipsilateral corticospinal tract signs and cerebellar ataxia was designated by Fisher [1] as ataxic hemiparesis. Pathologic study showed an old lacunar infarction in the basis pontis at the level of junction of the upper one-third and lower two-thirds on the opposite side of the neurologic deficit. The following is a report of case of ataxic hemiparesis with a lesion in the parietal lobe as seen on the computerized tomography (CT) scan.

Case Report

A 61-year-old white male with non-insulin-dependent diabetes mellitus developed paresthesia of the left upper limb in February, 1982. The carotid angiogram at that time showed bilateral mild atherosclerotic changes at the carotid bifurcation. Two-dimensional echocardiography showed a clot in the anterior leaflet of the mitral valve. He was treated with intravenous heparin and later on with Warfarin for six months. He was stable till November 2, 1985, when he developed tingling and numbness of the left upper extremity along with weakness of the left hand. On examination the patient was awake and alert. There was flattening of the left nasolabial folds. There was slight drift of the left upper extremity with slight weakness (-5/5 on Medical Research Council Scale) of the left upper extremity. The patient had slightly impaired pain sensation on the left upper extremity along with agraphesthesia and astereognosis. The position sense of the left fingers was slightly impaired while the vibration sense was normal. The tendon reflexes were symmetrical and the left plantar reflex was extensor. The patient had marked finger to nose ataxia which was out of proportion to the weakness

and sensory changes. There was no significant change in the patient's ataxia with eyes open or closed, and the Romberg sign was negative. A CT scan of the head showed a well-defined area of decreased density in the right parietal region consistent with ischemic infarction (fig. 1).

Discussion

Infarction found in posterosuperior part of the internal capsule, adjacent to the corona radiata, was thought to be responsible for ataxic hemiparesis in the autopsied case of Fisher and Cole [2]. They felt that the clinicopathologic correlation was unsatisfactory because of presence of multiple infarctions in other locations. In 1978, Fisher [1] described three cases of ataxic hemiparesis with an old infarct cavity in the basis pontis at the level of junction of the upper one-third and lower two-thirds on the opposite side of the neurologic deficit. The infarct was thought to be secondary to occlusion of penetrating arteries as the basilar artery was patent in each case. All three patients were hypertensive. Subsequently there are several reports of ataxic hemiparesis with varieties of pathologic lesions [infarction, ref. 3-14; hemorrhage, ref. 15-17; tumors, ref. 12, 14, 18] in different locations [internal capsule, ref. 3, 4, 6, 8, 10, 11, 17; corona radiata, ref. 5, 14; thalamus, ref. 7; midbrain, ref. 18; basis pontis, ref. 9, 10, 13, 15] as shown by CT scan of the brain.

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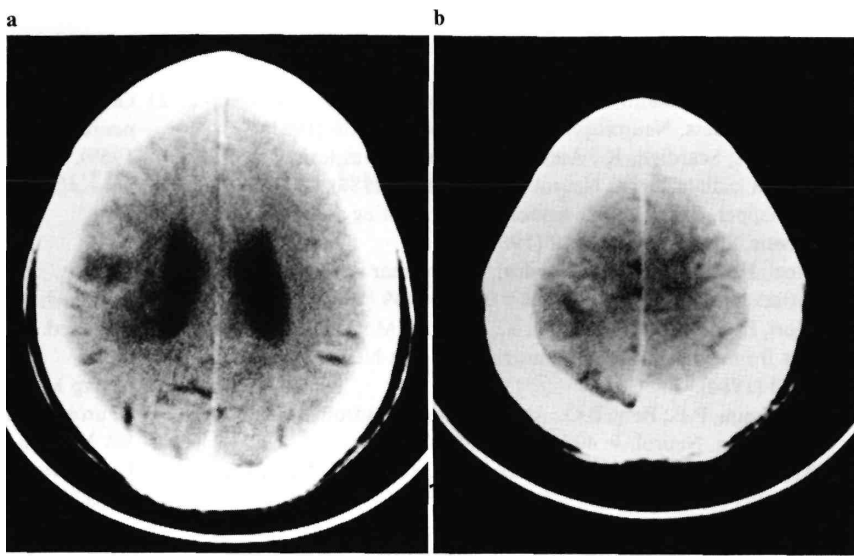


Fig. 1. CT scan showing an infarction in the right parietal region (a, b).

In our patient the CT scan of the brain (fig. 1) showed a recent ischemic infarction in the parietal region. The patient had mild left hemiparesis with ataxia of the left upper and lower extremities and parietal lobe signs characterized by tactile inattention, mild agraphesthesia and astereognosis. Ataxia in parietal lobe lesions has been described since 1916 [quoted by Appenzeller and Hanson, ref. 19], however, there are only infrequent reports in English language. Appenzeller and Hanson [19] described two patients with parietal ataxias, one of whom had an infarction in the arm area of the parietal lobe in the postcentral gyrus [19]. Critchley [20] described two patients with unilateral ataxia and parietal lobe lesion. In both cases there was no sensory loss. One patient was thought to have biparietal softening while the other patient had a large parietal lobe abscess at autopsy. The reason for cerebellar ataxias in lesion of the parietal lobe is uncertain. Impaired proprioception is thought to be responsible for ataxia but some patients with parietal ataxia do not have sensory impairment on clinical examination. Ataxia may be related to lack of spatial orientation in the patients with parietal lobe lesion or related to destruction of corticopontine (parietopontine) fibers destined for the cerebellum. Also, it is believed that parietal lobe constitutes a higher center related to vestibular and cerebellar nuclei [21]. We believe that ataxic hemiparesis in our patient was due to recent infarction seen on the CT scan in the parietal area. Ataxic hemiparesis produced by the lesion in different areas of the brain can be recognized clinically by the presence of other associated signs and symptoms and CT scan [10]. The presence of nystagmus, dysarthria and normal sensation favors pontine localization and presence of sensory abnormalities, while absence of dysarthria and nystagmus favors supratentorial location for ataxic hemiparesis. Ataxic hemiparesis is a syndrome caused by a variety of pathologic processes in the different regions of the brain.

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