

Isolated astasia manifested by acute infarct of the anterior corpus callosum and cingulate gyrus



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ABSTRACT

Astasia, the inability to stand and walk, in the absence of weakness or ataxia, is uncommon and unique. This syndrome is easy to misdiagnose as vestibulocerebellar disease. We report a patient with anterior callosal and cingulate infarction presenting as isolated astasia. A 65-year-old hypertensive man was admitted to our hospital with marked truncal instability. On neurological examination, he was unable to stand and walk independently. When asked to stand up from a sitting position, he had to grasp something nearby to propel himself upwards, and could not use his axial muscles. He collapsed when support was withdrawn. The patient showed no signs of amnesia, aphasia, apraxia or hemiasomatognosia. Diffusion weighted imaging at 3 days after onset demonstrated an acute infarct in the anterior part of the right-sided corpus callosum and cingulate gyrus. Magnetic resonance angiography revealed an occlusion of the right anterior cerebral artery. We discuss the possible mechanisms of astasia due to anterior callosal and cingulate infarction.

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1. Case report

A 65-year-old right-handed Chinese man was admitted to our hospital with a suddenly inability to stand and walk; when attempting to stand, he leaned to the left side and could not stand unsupported. In contrast, he was still able to use chopsticks to eat. He also had a pulsing headache on the top of his head and was suffering visual hallucinations, such as seeing his granddaughter standing beside his bed. The man had a 10 year history of hypertension and did not take anti-hypertensive drugs regularly. During the 5 days prior to onset, he had two instances of a transient difficulty in standing, each lasting for about 10 minutes, but did not seek medical care.

On examination, his blood pressure was 180/109 mmHg. He had normal consciousness and orientation. Impairment of higher functions, such as amnesia, aphasia, apraxia and hemiasomatognosia, were not noted. Muscle strength and tone of the extremities were normal. However, he had marked truncal instability and was unable to stand or walk unassisted. His ability to sit was relatively preserved. When asked to stand up from a sitting position, he needed to grasp something nearby to propel himself upwards, and could not use his axial muscles. He fell to the left or backwards when support was withdrawn. In contrast with cerebellar ataxia, the gait was not broad-based. Finger-to-nose and heel-knee test were normal on both sides. No sensory deficits were observed. All the tendon reflexes were normoactive, with no pathological reflexes.

The laboratory tests including routine electrolytes, renal function, glycosylated hemoglobin, homocysteine, coagulation function, antiphospholipid and anti-neutrophil cytoplasmic antibody were normal. Low density lipoprotein cholesterol was 3.72 mmol/L (normal range: 1.94–3.61 mmol/L). Transthoracic echocardiography showed marked left ventricular hypertrophy. Carotid ultrasound demonstrated increased intima-media thickness of the bilateral carotid arteries and less than 50% stenosis of proximal internal carotid arteries. On admission, an emergency brain CT scan was nor-

mal. Three days after onset, brain MRI showed an acute infarct in the right-sided genu and body of the corpus callosum and the anterior part of cingulate gyrus (Fig. 1A, B). Magnetic resonance angiography revealed an occlusion of right anterior cerebral artery and multiple stenoses of the bilateral middle cerebral arteries and posterior cerebral arteries (Fig. 1C).

The patient was treated with aspirin, atorvastatin, edaravone and antihypertensive agents. His symptoms improved gradually. One month later, he was able to walk independently.

2. Discussion

This patient's presentation was characterized by marked truncal instability and he was unable to stand or walk unassisted. When asked to stand up, he used distal limb muscles rather than axial muscles. This presentation could not be explained by limb weakness, ataxia, involuntary movements, or sensory disorders, because he displayed no such symptoms or signs. Instead, the clinical picture of our patient was compatible with astasia as described by Masdeu and Gorelick [1].

Astasia has been reported to be associated with lesions of the thalamic ventrolateral region, supplementary motor area, posterior cingulate cortex, or midbrain tegmental region [2–5]. However, there are no reports in the English literature of astasia following anterior callosal or cingulate lesion to our knowledge. In our patient, diffusion weighted imaging showed an acute infarct in the anterior corpus callosum and cingulate gyrus, suggesting anterior callosal and cingulate infarction may cause astasia. However, the underlying mechanisms are unknown. Fastigial fibers of the vestibulocerebellar pathway project to the medial ventrolateral nucleus of the thalamus, which in turn projects fibers to the vestibular cortex [1,2]. Disruption of these connections may lead to impairment of balance and gait. Moreover, effective walking requires coordination between various levels of the nervous system and frontal executive functions [6]. The bilateral frontal lobes connect with each other through the anterior part of the corpus callosum [7]. Infarction of the anterior corpus callosum may impair integration of bilateral frontal executive function for walking, causing disorders of balance and gait.

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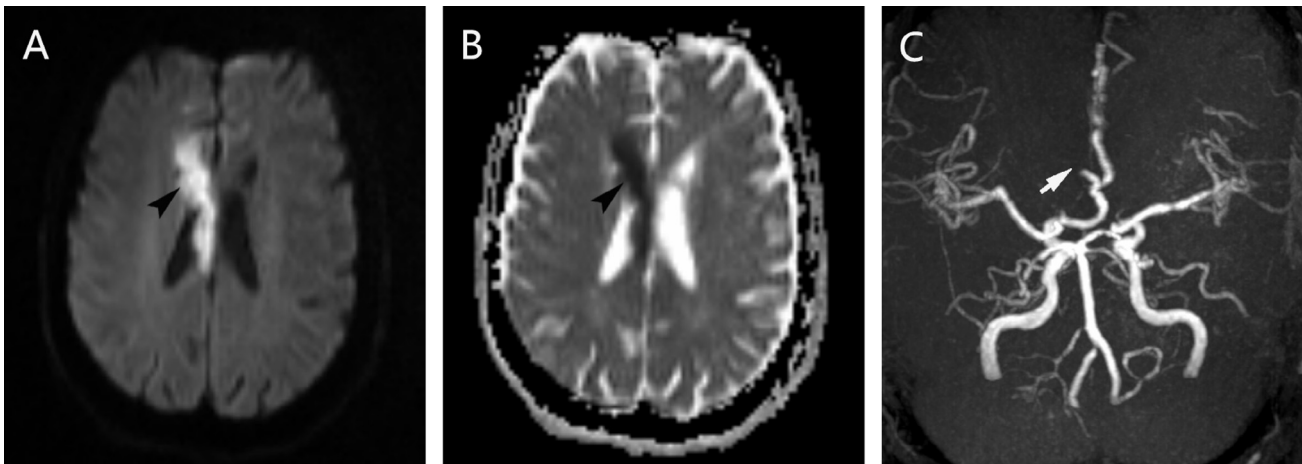


Fig. 1. Axial diffusion weighted imaging MRI shows high intensity in the anterior part of the right-sided corpus callosum and cingulate gyrus (A, arrowhead), with corresponding apparent diffusion coefficient maps (B, arrowhead). Magnetic resonance angiography demonstrates occlusion of the right anterior cerebral artery (C, arrow).

In previous case reports of astasia caused by a callosal lesion, disconnection syndromes such as left hand apraxia, agraphia, and tactile anomia have been observed concurrently [8,9]. However, our patient had no signs of disconnection syndrome. We noted that one patient with corpus callosum astrocytoma in a previous report had isolated astasia-abasia initially. As the tumor involved the whole corpus callosum, she developed an apraxic disorder. Thus, the type of lesion causing astasia with disconnection syndrome may involve the entire corpus callosum. More cases are needed to investigate the correlation of clinical manifestation and radiological results.

Astasia, presenting as isolated inability to stand and walk, is easily misinterpreted as a symptom of vestibulocerebellar disease. To differentiate from vestibulocerebellar disease, patients with astasia do not exhibit a broad-based gait or ataxia. Moreover, other symptoms indicating cerebral hemisphere disorders may appear concurrently with astasia. Our patient had visual hallucinations after stroke onset, suggesting the gait disorder was not due to a vestibulocerebellar lesion.

Conflicts of Interest/Disclosures

The authors declare that they have no financial or other conflicts of interest in relation to this research and its publication.

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