Case Report

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Bilateral cerebral infarction associated with severe arteriosclerosis in the A1 segment: a case report

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Abstract

Large artery atherosclerosis and cardioembolism are the two major subtypes of ischemic stroke. We herein describe a 75-year-old man with acute complete cerebral infarction in the typical territories of the bilateral anterior cerebral artery (ACA) and left middle cerebral artery. Brain magnetic resonance angiography showed that the right A1 segment of the ACA was affected by severe arteriosclerosis and that the right ACA other than the A1 segment was compensated by the left ACA through the anterior communicating artery. Acute cardioembolism only occluded the left anterior circulation but simultaneously blocked the right ACA due to decompensation. We presume that the bilateral cerebral infarctions were caused by chronic atherosclerosis and acute cardioembolism.

Keywords

Bilateral cerebral infarction, atherosclerosis, cardioembolism, anterior cerebral artery, middle cerebral artery, ischemic stroke, magnetic resonance angiography

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Introduction

Large artery atherosclerosis and cardioembolism are the two major subtypes of ischemic stroke in the Trial of Org 10172 in Acute Stroke Treatment classification, which indicates the etiology for each

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stroke subtype.[1] Large atherosclerotic cerebral infarction mainly occurs in patients of advanced age with chronic hypertension. However, cardioembolic cerebral infarction, which accounts for approximately one-quarter of all cerebral infarcts, may occur in patients of any age.[2] We herein report a rare case of complete cerebral infarction in the typical territories of the bilateral anterior cerebral artery (ACA) and left middle cerebral artery (MCA) caused by a combination of chronic atherosclerosis and acute cardioembolism. This case is being reported to highlight the fact that we should pay attention to acute bilateral cerebral infarction caused by a mixture of the two etiologies or anatomic variation.

Case report

A 75-year-old man presented with a sudden onset of unconsciousness, quadriplegia, and persistence epilepsy progressing from the right upper limb to the whole body. He had a medical history of hypertension that was poorly controlled with irbesartan.

On physical examination, his heart rate was 70 beats/minute with atrial fibrillation. His blood pressure was 175/84 mmHg. A neurological examination showed light coma, left-gaze deviation, right central facial paralysis, and quadriplegia (motor power grade 0 in the right extremity, 1 in the left upper extremity, and 2 in the left lower extremity). His deep tendon reflexes were 4+ on the right side and 3+ on the left side. Both Chaddock's sign and Babinski's sign were positive. An electrocardiogram demonstrated atrial fibrillation. The laboratory findings for thrombolysis revealed no contraindications, and brain computed tomography at 2 hours after symptom onset showed no hemorrhage. Venous thrombolysis using alteplase (not mechanical thrombectomy because of economic limitations) was started 2.5 hours after symptom onset. On day 10 of hospitalization, brain magnetic resonance imaging (MRI) revealed complete acute cerebral infarction in the typical territories of the bilateral ACA and left MCA (Figure 1(a) and (b)). Head and neck magnetic resonance angiography demonstrated severe arteriosclerosis in the right internal carotid artery, right MCA, and A1 segment of the right ACA (Figure 1(c) and (d)).

About 1 month later, the patient's level of consciousness recovered to stupor and his motor weakness partially improved (motor power grade 2 in the bilateral upper extremities and 3 in the bilateral lower extremities). Finally, the patient was transferred to the rehabilitation department for a physical rehabilitation program.

This study was carried out in accordance with the recommendations of the ethics committee of the First Affiliated Hospital of Sun Yat-Sen University. The patient gave written informed consent in accordance with the Declaration of Helsinki. The protocol was approved by the ethics committee of the First Affiliated Hospital of Sun Yat-Sen University (No. 58 Zhong Shan Er Lu, Guangzhou, China). Because of the patient's low level of consciousness and motor weakness, his caretaker signed the consent form and gave consent to publish the case report.

Discussion

Bilateral ACA territory infarction, especially acute complete bilateral infarction, is rare.[3] Bilateral ACA territory infarction revealed by MRI may be caused by frequent arterial atherosclerotic infarction, cardioembolism, or other reasons; however, frequent infarction usually exhibits a different course on MRI. The A1 segment of the ACA is reportedly missing in 0.2% to 2.0% of autopsy cases.[4] Some cases of acute bilateral caudate nucleus infarction are considered to be related to the variant A1 segment.[5,6] In our patient, head and neck

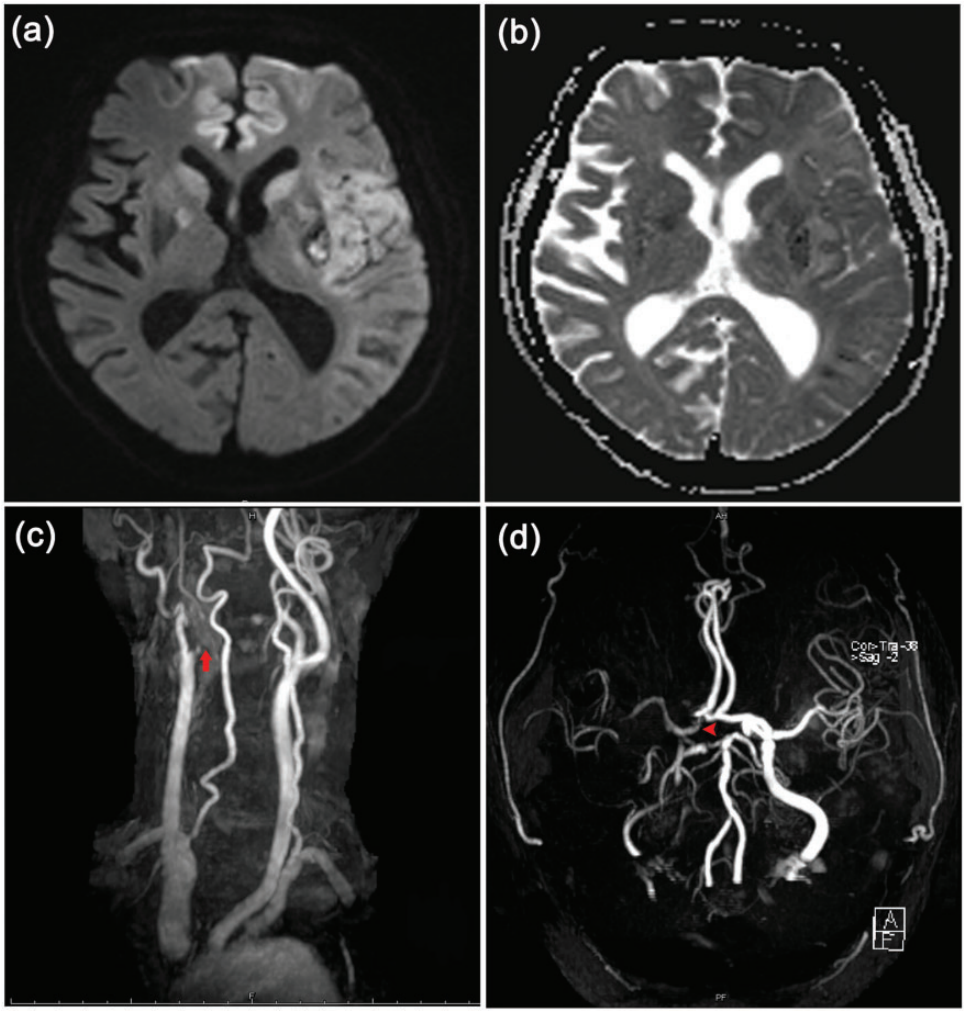

Figure 1. (a) Brain magnetic resonance imaging revealed increased signal intensities in the bilateral anterior cerebral artery (ACA) territory and left middle cerebral artery (MCA) territory on diffusion-weighted imaging. (b) Brain magnetic resonance imaging demonstrated decreased signal intensities in the bilateral ACA territory and left MCA territory on apparent diffusion coefficient-calculated images. (c) Neck magnetic resonance angiography demonstrated severe arteriosclerosis and almost complete occlusion at the initial part of the right internal carotid artery (arrow). (d) Head magnetic resonance angiography revealed severe arteriosclerosis at the C4 to C6 segment of the right internal carotid artery, right MCA, and A1 segment of the right ACA (arrowhead).

magnetic resonance angiography revealed no obvious anatomic variation at the A1 segment; however, the blood supply in the right anterior circulation was poor. The severe arteriosclerosis in the right A1 segment of the ACA, internal carotid artery, and MCA was likely due to chronic hypertension because the patient had a medical history of poorly controlled hypertension. As a result, the blood in the right

ACA and A1 segment was compensated by the left ACA through the anterior communicating artery.

No absolute criteria for differentiating cardioembolic infarction from atherothrombotic infarction are available, especially in patients with multiple risk factors. However, some clinical features are suggestive of cardioembolic infarction, including sudden onset of a maximal deficit, a decreased level of consciousness at symptom onset, and massive but not lacunar infarction as shown by neuroimaging.[7,8] In the present case, the patient presented with a sudden onset of unconsciousness, quadriplegia, and persistence epilepsy and showed massive infarction on subsequent neuroimaging. Moreover, we detected atrial fibrillation upon admission and hospitalization in our institution. Although some patients with atrial fibrillation have significant carotid artery disease, not all strokes in these patients are cardioembolic in origin.[9] However, our patient demonstrated severe arteriosclerosis in the right rather than left anterior circulation. Cerebral infarction attributed to atherothrombotic stroke would likely be located in the right hemisphere, not the bilateral hemispheres. Hence, atrial fibrillation could well have been the direct cause of the embolic infarction in the territories of the left MCA and left ACA in this patient. Once the left ACA had become occluded, the blood in the right ACA, which relied on compensation from the left ACA, became blocked and caused the infarction. Therefore, we presume that the acute complete bilateral cerebral infarctions were a result of chronic atherosclerosis and acute cardioembolism.

In this case, the patient presented with quadriplegia and unconsciousness and therefore may have easily been misdiagnosed with infarction of the brain stem. However, simultaneous bilateral cerebral hemisphere infarctions due to an anatomic variation or chronic atherosclerosis may also cause quadriplegia. Infarction of the cortical branches of the ACA usually causes contralateral hemiparesis with leg predominance, which is associated with involvement of paracentral lobule.[10] In contrast, predominant weakness of the contralateral arm and face is attributed to occlusion of the ACA perforators.[11] The right hemiplegia in this patient was ascribed to infarction of the ACA and MCA, but the weakness in the left upper and left lower extremities were caused by infarction of the ACA perforating and cortical branches, respectively.

This case highlights the importance of paying close attention to the compensation of the collateral circulation due to long-term atherosclerosis. In such patients, acute cardioembolism on one side may cause atherosclerosis-associated bilateral cerebral infarction.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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