Case Report

A Case Report of Takayasu Arteritis Accompanied with Diabetes Mellitus in a 52-Years-Old Man Diagnosed by Cerebral Infarction

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Abstract

Objective
Takayasu arteritis (TA) is a major vessel inflammatory disease. Cerebral infarction is one of its presenting symptoms. Atypical ischemic stroke is sometimes caused by complicated hemodynamic changes associated with TA.

Conclusion
Cerebral angiography revealed impaired hemodynamics suggestive of TA type I, based on revised Ishikawa diagnostic criteria. However, both CCA occlusions were caused by different mechanisms. Major vessel occlusions by TA-derived vessel wall thickness and atheromatous plaque deposition coexisted in one patient. TA should be taken into account as one of the causes of atypical cerebral infarction.

Keyword: Takayasu Arteritis; Cerebral Infarction; Diabetes Mellitus

Case Report

A 52-year-old man with diabetes mellitus (DM) developed left hemiplegia and dysarthria; he was diagnosed with cerebral infarction. Magnetic resonance imaging showed a cerebral infarction extending from the right corona radiata to the internal capsule. Acute antithrombotic therapy and rehabilitation against neurological disorders were performed. He was then transferred to the rehabilitation hospital at 37 days post-admission with dual antiplatelet therapy (100 mg of aspirin and 75 mg of clopidogrel per day). Diffusion-weighted imaging of MRI showed hyperintensity signal extending from the right corona radiate to the internal capsule (Figure 1A). Time of flight (TOF) image of MRI demonstrated a loss of or reduction in the flow gain at right or left internal carotid artery (ICA), respectively (Figure 1B).

Doppler echo examination showed a macaroni sign in the left common carotid artery (CCA) and atheromatous plaque deposition of the right CCA. Cerebral angiography demonstrated that both CCA and the left subclavian artery were occluded. The right vertebral artery (VA) perfused posterior circulation and supplied reverse flow to the left VA. Collateral flows from VA to occipital artery via meningeal branch made a retrograde stream from the external carotid arteries to the internal carotid arteries. This caused an increase in the serum inflammatory markers.

Introduction

Takayasu arteritis (TA) has vessel wall thickness in major branches of the aorta promoted by inflammatory responses [1,2]. Severe stenosis or occlusion of artery causes cerebral infarction [1-5]. Although the association of type I diabetes mellitus (DM) [6-8] or gestational DM [9] with TA are reported, TA in type II DM is rare. This report presents a rare case of male TA with severe type II DM. Further, the coexistence of different kinds of the occlusive manner in major vessels has been quite rare.

Case Presentation

A 52-year-old man with DM developed left hemiplegia and dysarthria. His conscious level was clear without aphasia. Magnetic resonance imaging (MRI) diagnosed cerebral infarction extending from the right corona radiata to the internal capsule. Acute antithrombotic therapy and rehabilitation against neurological disorders were performed. He was then transferred to the rehabilitation hospital at 37 days post-admission with dual antiplatelet therapy (100 mg of aspirin and 75 mg of clopidogrel per day). Diffusion-weighted imaging of MRI showed hyperintensity signal extending from the right corona radiate to the internal capsule (Figure 1A). Time of flight (TOF) image of MRI demonstrated a loss of or reduction in the flow gain at right or left internal carotid artery (ICA), respectively (Figure 1B).

Doppler echo examination showed a macaroni sign in the left common carotid artery (CCA) and atheromatous plaque deposition in the right CCA with a filamentous vascular lumen (Figure 2). Both CCAs were stumped with no flow. Left external carotid arteries (ECAs) showed reverse flow, since right ECA made weak anterograde flow.

Cerebral angiography showed that both the CCA and left subclavian artery were occluded. The right vertebral artery (VA) perfused...
posterior circulation, as well as supplied reverse flow to the left VA. Collateral flows from both VAs to the occipital artery via meningeal branch made a retrograde stream from ECA to ICA. The anterograde flow of the left ICA perfused not only the left but right anterior circulation area by cross-flow via anterior communicating artery (Acom) (Figure 3). Further, the left subclavian artery was supplied via the reverse flow of the left VA (data not shown). Only the right VA from the innominate artery was kept in the major branch of the aortic arch (Figure 4).

The serological test showed increased C-reactive protein (5.27 mg/dL), hemoglobin-A1c (10.0%), erythrocyte sedimentation rate (34 mm/h) and prolonged activated partial thromboplastin time (38.2 s). Electrocardiogram never detected atrial fibrillation, and echocardiogram never demonstrated abnormal cardiac wall motions.

Discussion
A middle-aged man with type II DM was diagnosed with TA type I according to the revised Ishikawa diagnostic criteria [10]. The cervical vessels echogram showed both CCA were near total occlusion. Although the right CCA had atheromatous plaque deposition, the left CCA had vessel wall thickness with macaroni sign (Figure 2). This result suggested that each CCA occluded by a different manner. Inflammatory reactions chronically occluded the left CCA. During the gradual atheromatous plaque increment which blocked right CCA ICA flow, cross-flow supply via Acom might develop to prevent the wide range ischemic stroke.

The diagnosis of TA was done by cervical vessels echogram, cervical-cerebral angiography and serum examination. Left CCA had isoechoic vessel wall thickness (Figure 2A) suspected to be caused by an inflammatory reaction. In contrast, the right CCA
had hypoechoic plaque deposition of atheromatous thrombus. Major branches of the aorta were impaired at both the CCA and left subclavian artery (Figure 4). The innominate artery kept the flow intact. The right VA from innominate artery perfused the whole brain and left upper limb. In contrast, the pulmonary artery and renal artery had no stenosis. The aortic arch and descending artery had no stenosis or aneurysm. These results from angiography and computed tomography angiography confirmed type I TA by Ichikawa criteria. This patient matched three items of the American College of Rheumatology 1990 criteria: decreased brachial artery pulse, blood pressure difference >10 mmHg and arteriogram abnormality [11]. However, the patient had major vessels stenosis with atheromatous plaque deposition and vessel wall thickness caused by an inflammatory reaction. The coexistence of vessels stenosis by different mechanisms was much more rare. The majority of TA patients are young women, who have a low risk for atherosclerosis. In contrast, middle-aged men with TA are rare. From these reasons, we have little knowledge about the correlation of TA and atherosclerosis. This patient had a high risk for atherosclerosis with severe DM. Though modified hemodynamics by TA kept balance perfusion, progressed atherosclerosis disturbed hemodynamic balance. That explanation is the presumed mechanism of cerebral infarction in this case. Accidental diagnosis of TA was accompanied with cerebral infarction.

Conclusion
A DM carrying patient with cerebral infarction was diagnosed with TA. Atheromatous change occluded both CCA and TA-derived vessels wall thickness, respectively. Although differential diagnosis for TA includes arteriosclerosis, we should take into account the coexistence of TA and DM.
References