Repeated Brain Infarction Caused by Atherosclerosis of the Bovine Aortic Arch Successfully Treated With Arch Replacement

—Case Report—

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Abstract

A 62-year-old man presented with repeated embolic infarction over the last 3 years. Computed tomography angiography of the aortic arch revealed that the patient had a common trunk for the innominate and left carotid arteries, the so-called bovine aortic arch, with stenosis extending to both the left common carotid artery and innominate artery. Since antiplatelet therapy was not adequate for prevention of recurrent infarction, total replacement of the aortic arch was performed. Since then, no further ischemic events have manifested. Endovascular procedures are not safe enough for atherosclerotic lesions in the aortic arch, especially in a patient with bovine aortic arch, so surgical aortic arch replacement should be considered.

Key words: bovine aortic arch, atherosclerosis, brain infarction, computed tomography angiography, arch replacement

Introduction

The pathogenesis of brain infarction is quite complicated and the most common types are carotid artery atherosclerosis; cardiogenic infarction, which is usually derived from atrial fibrillation; and lacunar infarction caused by small artery occlusion. Recently, atherosclerotic disease of the aortic arch has been recognized as a risk factor of ischemic stroke. An autopsy study has suggested that ulcerated plaques in the aortic arch may be involved in causing brain infarction, especially in patients with uncertain etiology. Clinical data show that aortic atherosclerotic plaques with a thickness ≥4 mm have high risk for embolization. Atherosclerotic disease of the aortic arch should be regarded as a potential source of cerebral emboli.

The most common aortic arch branching pattern consists of three major vessels, the innominate artery, left common carotid artery, and left subclavian artery. The second most common pattern has the innominate artery and left carotid artery originating from a common trunk, the so-called bovine aortic arch. This anatomic variation of the aortic arch occurs during embryological development. The incidence of bovine aortic arch varies between racial groups, at approximately 8–13% in previous cadaver reports.

We report a case of ischemic stroke caused by artery-to-artery embolism from the atherosclerotic bovine aortic arch. Combined antiplatelet therapy was not sufficient for secondary stroke prevention, and repeated formation and rupture of plaques at the bovine aortic arch occurred. Surgical removal of the atheroma and aortic arch replacement were performed and showed good efficacy.

Case Report

A 60-year-old man had presented with left hemiparesis and was treated in another hospital 2.5 years previously. His past medical history included bladder cancer treated with endoscopic removal and abdominal aortic aneurysm treated with aortic replacement. Magnetic resonance (MR) imaging revealed cerebral infarction in the right middle and posterior cerebral artery territories. MR angiography showed transient basilar artery occlusion, which later recanalized, and an incidental anterior communicating artery aneurysm. The embolic source was not detected even by additional carotid and heart echographical examinations. He was then discharged with mild dysesthesia and slight cognitive dysfunction. Treatments with antiplatelet (clopidogrel 75 mg) and statin (pitavastatin 1 mg) was continued for prevention of new onset of stroke.

He was admitted to our hospital with mild left hemiparesis and mild consciousness disturbance. From his past history, cerebral embolism of cardiac origin was strongly
suspected. Electrocardiography showed sinus rhythm. Transthoracic echocardiography did not detect heart failure or any cardiogenic embolic sources, and transtho- racic ultrasonography of the neck did not reveal explanatory atherosclerotic stenosis of the cervical carotid arteries. Laboratory tests did not show any specific abnormalities. Diffusion-weighted MR imaging showed several small high density spots in the right middle and posterior cerebral artery territories, and right vertebral artery territory (Fig. 1A). Computed tomography (CT) angiography revealed severe stenosis and two ulcers with red thrombus of the common trunk for the innominate and left carotid artery, the so-called bovine aortic arch, and severe stenosis of the common trunk that extended to both the left common carotid and innominate arteries (Fig. 2A). He recovered fully from the neurological deficits in several days and was discharged. The antithrombotic therapy was intensified (clopidogrel 75 mg, aspirin 100 mg, and cilostazol 200 mg) with the addition of ethyl icosapentate (1800 mg).

Three months later, he presented with dressing apraxia and mild consciousness disturbance. MR imaging revealed a new infarction in the right cerebellum (Fig. 1B). CT angiography showed that the shape of the plaque in the common trunk of the bovine aortic arch had slightly changed (Fig. 2B). Digital subtraction angiography (DSA) was not performed because the catheterization procedures might cause further embolism from the common trunk of the bovine arch. Since antithrombotic therapies were not sufficient for the prevention of recurrent infarction and endovascular procedures were not safe, we decided to perform operative repair of the affected vessels. Total replacement of the aortic arch was performed and the single common trunk of the bovine arch was replaced with two synthetic vascular prostheses (Fig. 2C). We used selective

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**Fig. 1** A: Diffusion-weighted magnetic resonance images at the second ischemic attack showing several small high intensity lesions in the right middle and posterior cerebral artery territories and right vertebral artery territory. B: Diffusion-weighted magnetic resonance image at the third ischemic attack showing a new lesion in the right cerebellum (arrow).
cerebral perfusion as an adjunct therapy, with the modified isolation technique to prevent embolic stroke.\textsuperscript{17}

The removed bovine aortic arch had severe stenosis and large ulcers with red thrombus (Fig. 3). Histological examination of the atheroma showed severe atherosclerotic membrane with cholesterol crystals, histiocytes, and multinucleated giant cells. Hemosiderotic and hemorrhagic changes were also found but the calcification was not severe. No cerebrovascular event was observed during and after the operation. No further ischemic events have manifested for more than 9 months with less intense antiplatelet therapy (aspirin 100 mg and cilostazol 200 mg).

**Discussion**

Severe atheromas of the aortic arch are regarded as one of the important risk factors for embolic cerebral infarction.\textsuperscript{11,10} However, this important mechanism of ischemic stroke is often poorly recognized.\textsuperscript{14,15} Therefore, we must consider the possibility of aortogenic embolism, if cardio- genic embolic sources and/or cervical and intracranial arterial stenoses are not detected. Especially if the lesions are found in territories supplied by multiple major vessels, evaluation of the aortic arch is mandatory. In our case, ulcerated and mobile plaques were present in the common trunk of the bovine aortic arch that caused repeated infarctions in the territories of multiple major vessels.

MR imaging, including diffusion-weighted imaging, and MR angiography are routinely performed for the diagnosis of cerebral infarction. However, MR angiography of the aortic arch is not accurate enough because of motion artifacts.DSA has been regarded as the most accurate diagnostic procedure for cerebrovascular diseases. However, DSA has a higher risk of embolism for patients with atheroma in the aortic arch. Transesophageal echocardiography (TEE) is an established procedure for the diagnosis of aortogenic embolism.\textsuperscript{1,11,16} However, TEE cannot easily evaluate lesions around the branching of the innominate and left carotid arteries from the aortic arch even with experienced operators. In addition, TEE is an invasive procedure that is not tolerated by all patients. CT angiography of the aortic arch is noninvasive except for the use of contrast medium and relies less on the operator’s skill. Multidetector-row spiral CT is efficient for investigation of anatomical conformation and detection of atherosclerosis of the aortic arch.\textsuperscript{5,6} In our case, CT angiography could reveal the anatomical variation and severe stenosis of the aortic arch. Moreover, sequential CT angiography could also reveal the dynamic morphological changes of the plaque, which suggested repeated formation and rupture of the plaque.

Optimal treatments for the secondary prevention of cerebral infarction derived from aortic arch atherosclerosis have not been established. A combination of antiplatelet agents and/or anticoagulation therapy has been generally used.\textsuperscript{20} Some nonrandomized controlled observational case series have shown benefit in treating mobile plaques. Warfarin was reported efficacious in preventing stroke in patients with mobile aortic atheroma.\textsuperscript{29} On the other hand, anticoagulation was reported in association with new or worsening atheroemboli, possibly because of bleeding into the plaques.\textsuperscript{7} Therefore, warfarin was not used in the present case. Antiplatelet treatment with aspirin has a substantial effect on the risk reduction of recurrent stroke. The Antiplatelet Trialists’ Collaboration meta-analysis showed a reduction of the 3-year risk of serious vascular events from 22% to 18%, a relative risk reduction of 22% with a number needed to treat of 25.\textsuperscript{17} The international randomized-controlled trial, Aortic Arch Related Cerebral Hazard Trial (ARCH) has already commenced. This trial suggests the superiority of the combination of clopidogrel plus aspirin to only warfarin in preventing recurrent vascular events in patients with transient ischemic attack, minor stroke, or peripheral embolism associated with severe aortic arch atheroma.

Treatment with statin, a 3-hydroxy-3-methylglutarylcoenzyme A reductase inhibitor, has decreased the risk of recurrent stroke in patients with severe aortic plaques. Simvastatin reduced the vessel wall area and increased lumen area in patients with asymptomatic carotid or aortic atheroma.\textsuperscript{6} In an observational study of 519 patients, statin therapy reduced the incidence of stroke and other embolic events in patients with severe thoracic aortic plaque on TEE.\textsuperscript{19} Further studies are needed for clarifying the role of statin therapy in preventing cerebral ischemic diseases derived from the aortic arch atherosclerosis.

Endovascular procedures are not safe enough for atherosclerotic lesions in the aortic arch, especially in patients with a bovine aortic arch from which both common carotid arteries arise.\textsuperscript{6} One of the difficulties is that both common carotid arteries have to be occluded for distal protection during the procedure. For those patients, surgical aortic arch replacement should be considered if intensified medical treatments are not effective for preventing further ischemic events.

Surgical treatment has not been commonly performed for aortogenic brain infarction and its efficiency is not well recognized. Aortic arch replacement was successful in a patient who had recurrent embolization and plaque-associated aortic arch thrombi.\textsuperscript{22} However, replacing the atheromatous proximal aorta is a high-risk procedure,\textsuperscript{10} and the risk of perioperative morbidity must be weighed against the risk of recurrent stroke. With recent progress in cardiovascular surgery, the operative outcomes of such cases have been greatly improved. In the present case, we successfully replaced the atheromatous proximal aorta using the isolation technique. Therefore, surgical aortic arch replacement is recommended for patients with poor response to medical treatments for preventing new onset ischemic events.

**References**


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