Case Report

Mobile Thrombus from a Ruptured Plaque in the Brachiocephalic Artery

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An 86-year-old man with a history of brain infarction was admitted because of recurrent stroke with symptoms of left hemiparesis. Ultrasonography revealed a mobile thrombus in the brachiocephalic artery. Although anticoagulant therapy was commenced, neurologic symptoms deteriorated suddenly and he died on the third day. Autopsy indicated that the thrombus resulted from plaque rupture in the brachiocephalic artery. **Key Words:** Mobile thrombus—brachiocephalic artery—brain infarction—plaque rupture—stroke—autopsy.

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The pathogenesis of atherothrombotic brain infarction can include various entities.¹ We report a patient who developed a severe embolic stroke because of plaque rupture^{2,3} in the brachiocephalic artery.

An 82-year-old man was admitted to our hospital after developing hemiparesis on the left side. Brain magnetic resonance (MR) imaging (MRI) revealed acute infarction in the anterior territory of the right middle cerebral artery (MCA), which was considered to represent artery-toartery embolism from a stenotic lesion at the beginning of the right internal carotid artery (ICA). The left ICA was occluded. Aspirin (100 mg/d) was prescribed. Transthoracic echocardiography showed only mild wall thickness of the left ventricle. Three years later, the patient developed paroxysmal atrial fibrillation. Warfarin was started, but was discontinued because of dermatitis as an adverse effect.

Four years after the initial stroke, he was admitted to our hospital after developing weakness in the left leg and speech impairment one morning. Plasma levels of fibrinolytic markers including fibrin degradation products (53 µg/mL) and D-dimer (28.1 µg/mL) were markedly elevated. Serum level of lipid was within normal range. MRI revealed two fresh infarcts in the cerebral cortex supplied by the right MCA. MR angiography demonstrated occlusion of the right MCA branches. Carotid ultrasonography demonstrated a hypoechoic thrombus moving from the brachiocephalic artery to the beginning of the right common carotid artery and subclavian artery (Fig 1, A). Surgical removal of the thrombus was withheld because of the risk of surgical intervention adversely impacting brain circulation. Intravenous unfractionated heparin (10,000 U/d) was started; the infusion rate was adjusted to maintain activated partial thromboplastin time of 40 to 50 seconds.

In the evening of hospital day 2, the patient developed a sudden decrease in level of consciousness, followed by vomiting and respiratory failure. Blue toe syndrome was seen. Brain MRI the next morning identified bilateral massive infarctions in both the anterior and posterior circulations (Fig 1, *B*). MR angiography revealed total occlusion of the right proximal MCA and proximal basilar artery, adding to occlusion of the left ICA from the previous stroke. The patient died in the afternoon.

Autopsy revealed severe stenosis of the brachiocephalic, right common carotid, and subclavian arteries. In the intra-arterial lumen, ruptured plaque with thrombi was observed (Fig 1, C). Bilateral multiple renal old

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Figure 1. (A) Proximal carotid arteries on duplex ultrasonography (day of admission). Hypoechoic thrombus (arrow) displays oscillation from brachiocephalic artery to right common carotid artery (CCA) and subclavian artery (SCA). (B) Axial image on diffusion-weighted MRI (third day). Whole brain excluding part of right MCA territory, right cerebellum, and medulla oblongata is demonstrated as regions with new infarct. Midline of cerebrum deviates to right side. (C) Section of brachiocephalic artery (left) (Masson-trichrome stain) and its schema (right). Part of fibrous capsule of plaque is broken, and structure with similar stainability as contents of plaque almost completely occluded arterial lumen. Contents of plaque are thought to be soft because they include little fibrous structure.

infarctions were also seen. Organized thrombus was present in the left auricular appendage.

Discussion

This case is rare in two points: the site of mobile thrombus, and mechanism of the thrombus formation. Although mobile thrombus or plaque in the carotid artery⁴⁻⁷ or aorta⁸ has been reported, there are few reports about the brachiocephalic artery.⁹ On the other hand, some authors pointed out plaque disruption as a cause of mobile plaque or thrombus.^{5,6} Because our patient had paroxysmal atrial fibrillation, it was hard to judge whether the thrombus was from the heart or formed originally in the artery. Given that autopsy revealed a ruptured plaque with thrombus, not fibrin-rich thrombus, the thrombus should be formed there.

Treatment for patients with mobile thrombus in a major cerebral artery remains controversial. Some authors recommend thromboembolectomy, particularly for thrombi in the carotid artery,^{4,7} but some have described cases treated using anticoagulant⁶ or antiplatelet agents.⁵ Whether we should have attempted surgical removal, and how much heparin should have been used, remains unresolved.

In this case, multiple embolism of bilateral kidneys and limbs was attributed to thrombus in the left auricular appendage. Thrombotic tendency after plaque rupture in the brachiocephalic artery might have accelerated thrombus formation in the appendage. Conversely, brain infarction was caused by thrombi attaching to a ruptured plaque in the brachiocephalic artery found on autopsy. The occlusion of the left ICA found at the age of 82 years would be caused through atherosclerotic mechanism, given that it probably occurred asymptomatically.

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