Disappearance of an Oscillating Intraluminal Thrombus in the Carotid Artery Demonstrated by Ultrasonography

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Abstract

In a patient with acute cardioembolic stroke, ultrasonographic studies demonstrated the disappearance of an oscillating intraluminal thrombus lodged at the carotid bifurcation. Following the commencement of immediate anticoagulation, the thrombus completely dissolved over two weeks without further deterioration in the patient's symptoms. Neurosonographic studies are useful for the detection and follow-up of an intraluminal thrombus in acute stroke patients undergoing anticoagulant therapy.

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Key words: intraluminal thrombus, ultrasonography, carotid artery

Introduction

In patients with acute ischemic stroke, cerebral angiographic studies occasionally identify an intraluminal thrombus within the major cerebral arteries. An intraluminal thrombus can lead to complete arterial occlusion and distal embolization, and as a result is considered as a threatening finding (1, 2). However, a standard treatment protocol for an intraluminal thrombus has not yet been established.

Neurosonographic studies are a real-time, noninvasive technique to evaluate the extra- and intracranial arteries. They can be easily performed at the bedside and are suitable for follow-up examinations.

Using duplex carotid ultrasonography and transcranial Doppler (TCD), we herein present the findings of an oscillating intraluminal thrombus within the carotid artery in a patient with acute cardioembolic stroke and its disappearance while undergoing anticoagulant therapy.

Case Report

The patient was a 64-year-old man with dilated cardiomyopathy. He had no history of hypertension, diabetes mellitus, hyperlipidemia, or smoking, and he had never received antithrombotic therapy previously.

The patient abruptly developed weakness in the left extremities, and was admitted to our hospital the following day. He also had an episode of a transient weakness in the right extremities 10 days before admission. On admission, his blood pressure was 110/60 mmHg with a regular heart rate of 64 beats/min. Neurological examinations revealed mild dysarthria and left hemiparesis.

Laboratory blood tests revealed the following: leukocyte count, 6,310/ μ l; erythrocyte count, 4,530,000/ μ l; hemoglobin, 12.1 g/dl; hematocrit, 37.9%; platelet count, 138,000/ μ l; C-reactive protein, 5.85 mg/dl; prothrombin time expressed as an international normalized ratio, 1.10; activated partial thromboplastin time, 42 seconds; fibrinogen, 529 mg/dl; antithrombin III, 80.0%; protein C, 83.6%; fibrin degradation products, 12 μ g/ml; D-dimer, 2.7 μ g/ml; and thrombin-antithrombin III complex, 21.6 μ g/l. Chest X-rays demonstrated an enlargement of the heart. Twelve-lead electrocardiogram showed normal sinus rhythm, but paroxysmal atrial fibrillation was detected on 48-hour electrocardiogram monitoring. Transthoracic echocardiography showed enlargement of the left ventricle with severely reduced contraction. No intracardiac thrombi were visualized.

A brain computed tomography (CT) on admission revealed a low-density area in the right parietal cortex (Fig. 1). A duplex carotid ultrasonographic examination using an ATL Ultramark 9 (Advanced Technology Laboratories, Bothell, WA) with the transducer operating at 5 to 10 MHz for B-mode and Doppler functions was performed just after the CT study. A B-mode scan demonstrated a mobile, echogenic intraluminal mass echo in the bifurcation of the right common carotid artery (CCA) through to the proximal site of the internal carotid artery (ICA) (Fig. 2A). The

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Figure 1. Brain computed tomography (CT) on admission reveals a low-density area in the right parietal cortex (arrow).

intraluminal mass echo was homogeneous, soft and elastic, and oscillated synchronously with the cardiac cycle. Color Doppler flow imaging showed anterograde blood flow in the affected part of the ICA in both systolic and diastolic phases. The peak-systolic and end-diastolic flow velocities of the affected ICA were 202.5 cm/sec and 84.5 cm/sec, respectively. Intra-arterial digital subtraction angiography on the same day demonstrated an intraluminal filling defect in the right ICA extending from the carotid bifurcation to the cervical portion and complete occlusion of the right external carotid artery (Fig. 3). Neither abnormal flow delay nor opacification was detected in the intracranial arteries ipsilateral to the affected side.

We diagnosed the patient as having a cardioembolic stroke. About six hours after the angiography, anticoagulant therapy was started with intravenous heparin infusion to maintain the activated partial thromboplastin time approximately 1.5 times the pretreatment level. Oral administration of warfarin replaced the heparin treatment seven days after the admission, and the intensity was adjusted to an international normalized ratio between 2.0 and 2.5.

On the day following the admission, we performed trans-temporal TCD bilaterally for 30 minutes using a DWL Multidop X with the transducer operated at 2.5 MHz. Two microembolic signals (MES) were detected from the right

A

C

B

Figure 2. Duplex carotid ultrasonography on admission demonstrates a mobile, homogeneous intraluminal mass echo in the bifurcation of the right CCA through to the proximal ICA (arrow) on longitudinal B-mode scan (A). Following the commencement of immediate anticoagulant therapy, successive ultrasonographic studies on the 8th day (B) and the 11th day (C) of admission show that the intraluminal mass echo gradually decreased in size.

Figure 3. Cerebral angiography performed on the day of admission demonstrates a long, intraluminal filling defect in the right ICA extending from the bifurcation of the CCA to the cervical portion of the ICA (arrows) and complete occlusion of the right external carotid artery.

middle cerebral artery. Transesophageal echocardiography, performed on the same day, detected neither intracardiac thrombi nor right-to-left shunts nor atherosclerotic lesions of the aortic arch.

Follow-up carotid ultrasonographic examinations showed the intraluminal mass echo in the right carotid artery gradually decreased in size (Fig. 2B, 2C) and disappeared completely within two weeks of hospitalization. The peak-systolic and end-diastolic flow velocities of the affected ICA on the 11th day of admission were 39.6 cm/sec and 18.2 cm/sec, respectively. MES were no longer detected from

Figure 4. On day 28 of hospitalization, the right ICA appears normal on MR angiography.

bilateral middle cerebral arteries on follow-up TCD studies on the 7th day. Diffusion-weighted MR imaging of brain on the 25th day did not demonstrate fresh infarcts and MR angiography on the 28th day showed normal appearance of the right ICA (Fig. 4). The patient had no additional episodes of brain ischemia and was discharged without neurological deficits on the 30th day of admission.

Discussion

We recently reported that, in some patients with acute cardioembolic ICA occlusion, carotid ultrasonography demonstrated a mobile, echogenic intraluminal mass echo synchronizing with cardiac cycle in the proximal ICA (3, 4). We termed this finding “oscillating thrombus” and emphasized that it is a finding specific to acute embolic ICA occlusion. Although the intraluminal mass echo in the present case was like an “oscillating thrombus”, it did not occlude the ICA but became lodged at the carotid bifurcation. This is different

from the previous reports and is unique to the present case. We believe that the intraluminal mass echo was a thromboembolus from the heart lodging at the carotid bifurcation because the patient had obvious emboligenic heart disease such as dilated myopathy and atrial fibrillation but no demonstrable atherosclerotic diseases from the aortic arch through to the ipsilateral CCA. The thrombus may have subsequently propagated in an antegrade fashion into the ICA during the acute stage.

It has been reported that MES are rarely detected in patients with cardioembolic stroke as compared to those with carotid artery disease (5), and that bilateral MES are suggestive of a cardioembolic origin (6). The MES in the present case indicated that the microemboli originated from the thrombus lodged at the carotid bifurcation rather than from the heart, because the MES were detected unilaterally from the right MCA distal to the thrombus and no intracardiac thrombi were evident on transesophageal echocardiography. Detection of MES arising from an intraluminal thrombus supports the concept that intraluminal thrombi have a propensity to produce secondary, distal embolism (1, 2).

Some authors regard the presence of an intraluminal thrombus as an indication for urgent thromboendarterectomy. However, this is a questionable strategy. The perioperative complication rate was high in patients with intraluminal thrombus, accounting for 20 to 30% (7–9). On the other hand, resolution of thrombi was observed without any additional neurologic deficits in the majority of patients receiving only antithrombotic therapy (7, 8). In the present case, the intraluminal thrombus decreased in size and eventually disappeared during anticoagulant therapy. This is probably due to the relative predominance of plasma fibrinolytic activity over anticoagulation-inhibited thrombin activity (10).

Conclusion

In a patient with acute cardioembolic stroke, disappearance of a thromboembolus from the heart lodging at the carotid bifurcation was observed using a series of ultrasonographic studies. Within two weeks after starting immediate

anticoagulant therapy, the thrombus completely dissolved without any additional neurologic symptoms. Neurosonographic studies may be a useful tool for detection and follow-up of an intraluminal thrombus in acute stroke patients undergoing anticoagulant therapy.

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