CASE REPORTS

A large cardiogenic thrombus lodged at the carotid bifurcation mimicking severe carotid stenosis

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Abstract

A 62-year-old woman without vascular risk factors presented with left-sided weakness and numbness. Magnetic resonance imaging (MRI) brain depicted acute right hemispheric infarcts in the cortical and subcortical white matter. Initial MR angiography (MRA) showed large thrombus at the right carotid bifurcation. Valvular atrial fibrillation (vAF) with severe tricuspid regurgitation (TR) was found on two-dimensional echocardiography. Serial follow-up computed tomography angiography (CTA) or MRA at 3, 9, and 15 days after anticoagulation alone showed complete resolution of the thrombus with no neurological deterioration. Our case suggests that prompt institution of anticoagulation alone may result in radiologic resolution of the thrombus with improvement in patient’s clinical status.

INTRODUCTION

Thrombi at the site of extracranial internal carotid artery (ICA) are present more often in patients with severe atheromatous stenosis. They may act as a source of additional emboli, leading to intracranial branch occlusion, or progress locally to complete occlusion of the ICA. The finding of adherent clot within the lumen of the extracranial ICA is commonly thought to require emergency carotid surgery or stenting to prevent neurological deterioration. But the natural history of cardiogenic thromboemboli at extracranial ICA in patients presenting with acute cerebral ischemia might be very different from that of atheromatous stenosis. Regarding acute treatment of thrombi at the ICA, it is currently unclear whether surgical, endovascular (eg stenting) or medical treatment is superior, particularly when the presentation is more than 6-8 hours after onset of symptom. In addition, both surgical and endovascular therapy are associated with peri-procedural complications.

CASE REPORT

A 62-year-old woman presented with acute onset of left sided weakness and dysarthria. Two days prior to her stroke she had transient shortness of breath which resolved spontaneously. She did not have any risk factors or other medical illness. Neurologic examination revealed mild flattening of the left nasolabial fold, mild left sided weakness and left homonymous hemianopia. Sensation for pain and temperature was decreased over the left arm and left leg. She was started on intravenous heparin in another institution one day after symptom onset. MRI brain including T1, T2, fluid-attenuated inversion recovery and diffusion-weighted imaging sequences, revealed acute infaracts in the right middle cerebral artery (MCA) territory (Figure 1). Initial MRA performed in another institution demonstrated severe stenosis/occlusion of the right carotid bifurcation and the right proximal MCA (Figure 2A and B). Transcranial color duplex sonography detected high-grade stenosis in the proximal right MCA. Routine laboratory and coagulation studies were normal. Cardiac evaluation, which included two-dimensional echocardiography and Holter monitoring, showed vAF with severe TR.

Follow-up CTA 3 and 9 days after intravenous anticoagulation showed decreased size of the carotid artery filling defect compared with the first MRA (Figure 3). MRA 15 days after anticoagulation showed nearly complete resolution of the thrombus at the ICA but persistent occlusion of the right proximal MCA (Figure 2C and D). Notwithstanding persistent occlusion of the right proximal MCA, her recovery was uneventful and she was discharged nearly symptom-free.

DISCUSSION

The resolution rate of carotid artery thrombus with
Figure 1. Diffusion-weighted MRI showing acute right MCA territory infarcts.

Figure 2: MRA showed severe stenosis or occlusion of the right carotid bifurcation (A) and the right proximal MCA (B) (arrow). 15 days later MRA demonstrated no abnormality in the right ICA (C), but persistent occlusion of the right proximal MCA (D) (arrow).
anticoagulation alone ranged from 58% to 86%. Histopathology demonstrated embolic and plaque thrombi. The present case was distinguished by thromboemboli at the carotid bifurcation without significant atherosclerosis. The etiology is most likely cardiogenic embolism related to underlying vAF.

There was substantial evidence that thromboembolic material in the carotid artery accounted for her cerebral symptoms. Because this thrombus had a propensity to embolize and might be a prelude to complete arterial occlusion, it was regarded as potentially life-threatening. When we found a severe focal stenosis/occlusion at the right carotid bifurcation on initial MRA, it was difficult to decide whether this might be a surgical emergency. Optimal acute management of carotid artery thrombus remained controversial: some studies suggested emergency thromboendarterectomy whereas others recommended anticoagulation. Endovascular therapy, eg stenting, is a more recent therapeutic modality. Recently several studies showed usefulness of endovascular therapy in patients within 6-8 hours of large vessel ischemic stroke. Nonetheless, their safety or benefit of endovascular stent thrombectomy in those patients with major vessel occlusion after hyperacute stage are still disputable. Our patient presented to us only a day after the onset of symptom.

In our patient, there was neurological improvement with gradual resolution of the thrombi 15 days from symptom onset. Possibly the administration of antithrombotic drugs,
such as heparin and warfarin, prevented further thrombogenesis while the natural process of fibrinolysis proceeded. The proximal right MCA occlusion likely represented pre-existing atherosclerosis rather than persistent thrombosis, in view of resolution of the larger right carotid artery filling defect.

Our case suggests that with thrombi in the carotid artery, medical treatment with full anticoagulation may result in radiologic resolution with neurological improvement.

DISCLOSURE
Conflict of interest: None

REFERENCES