Cardiogenic Embolism Producing Crescendo Transient Ischemic Attacks

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Lateralizing, repetitive transient ischemic attacks are characteristic of symptomatic carotid bifurcation atherosclerotic plaques. We report a case in which a cardiogenic embolus, after lodging at the left carotid bifurcation, produced crescendo episodes of expressive aphasia and mild right upper extremity weakness. Complete neurological recovery was achieved following emergent carotid embolectomy and endarterectomy. This case demonstrates that the laminar nature of internal carotid blood flow may result in the localization of embolic events to a single region of the cerebral vasculature, regardless of the source lesion in the carotid artery. The role of endoluminal techniques in the diagnosis and management of such lesions is discussed.

Cardiogenic emboli are a common source of stroke, causing up to 60% of all ischemic strokes.¹⁻³ Distribution of these emboli throughout the peripheral vasculature is variable. Therefore, while solitary transient ischemic attacks (TIAs) arising from a cardiac embolic source are not uncommon, a clinical presentation of repetitive TIAs affecting the same hemisphere directs the vascular surgeon toward the diagnosis of unstable carotid bifurcation plaque. We present a report of a cardiogenic embolus that lodged at the left carotid bifurcation. Emboli subsequently released from this clot produced repetitive episodes of expressive aphasia and right hand weakness until successful treatment by emergent thromboembolectomy and endarterectomy was undertaken.

CASE REPORT

An 81-year-old woman with a history of hypertension and irregular heartbeat was brought to our emergency department after family members noted the sudden onset of expressive aphasia and right

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facial droop at home. Initial neurological exam was unremarkable. Electrocardiography (EKG) demonstrated new onset of atrial fibrillation with left bundle branch block and poor rate control. Noncontrast head computed tomography (CT) demonstrated age-related atrophy without acute intracranial abnormalities. Although her symptoms had resolved during transit, they recurred during her emergency department evaluation, and she was admitted to the neurology service for observation. Detailed examination revealed expressive aphasia, right facial droop, and right hand weakness with associated pronator drift. Over the next 12 hr, despite aspirin therapy, the patient experienced multiple transient recurrences of the same constellation of neurological findings, with resolution within 10 min in each instance. A carotid duplex examination was obtained the following morning. Velocities were consistent with an 80-99% left internal carotid stenosis, with note made of a mobile component of the bifurcation lesion.

The vascular surgery service was then consulted. The patient was heparinized, and an emergent cerebral angiogram was obtained, demonstrating a large saddle embolus lodged at the left carotid bifurcation (Figs. 1 and 2). The patient was transported directly to the operating suite.

Under general anesthetic, incision was made along the anterior border of the left sternocleidomastoid muscle, and exposure of the distal left internal carotid and proximal left common carotid was obtained while leaving the bifurcation untouched. After verifying adequate anticoagulation,

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Fig. 1. Anteroposterior angiogram of the left carotid artery, demonstrating a large saddle embolus lodged at the carotid bifurcation.

the internal carotid artery was clamped several centimeters distal to the angiographic extent of the saddle embolus, followed by clamping of the proximal common carotid artery. The bifurcation was then rapidly dissected out, and the external carotid artery was controlled. The bifurcation was opened, and the large embolus was rapidly evacuated. A Sundt shunt was then placed, with Doppler verification of shunt flow performed every 3 min until removal. No cerebral monitoring was used. Inspection of the bifurcation demonstrated a mild to moderate degree of smooth fibrocalcific plaque. Endarterectomy of the fibrocalcific plaque at the bifurcation was performed, followed by patch closure with saphenous vein harvested from the groin.

Full anticoagulation was continued throughout the perioperative period, with conversion from intravenous heparin to warfarin (Coumadin). The patient's neurological exam performance had returned to normal by postoperative day 1, and the remainder of her recovery was uneventful. She was



Fig. 2. Lateral angiogram of the left carotid artery, demonstrating a large saddle embolus lodged at the carotid bifurcation.

discharged on warfarin, with an international normalized ratio (INR) maintained between 2.0 and 3.0. She remains asymptomatic 18 months after the procedure.

DISCUSSION

The distribution of cardiogenic emboli within the arterial tree is highly variable, even between initial and subsequent embolic events in a single patient.^{4,5} In distinction, initial and subsequent TIAs produced by carotid bifurcation plaques usually affect the same regional cerebrovascular territory, as hypothesized by Millikan et al.^{6,7} and demonstrated by the elegant experiments of Whisnant⁸ and Gacs et al.⁹ In particularly symptomatic patients, this process produces crescendo TIAs.¹⁰

However, other carotid pathologies can produce repetitive TIAs, including internal carotid artery dissection and carotid body tumors.^{11,12} This patient's unusual presentation confirms that any

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embolizing lesion within the carotid circulation may preferentially embolize to a single region of the cerebral vasculature, mimicking the behavior of symptomatic carotid bifurcation plaques.

Anticoagulation remains the standard treatment of cardiogenic emboli due to atrial fibrillation. Although a solitary embolic event in the setting of untreated atrial fibrillation is usually treated with prompt anticoagulation, investigation of carotid arterial pathology should not be neglected. Should subsequent ipsilateral TIAs be detected, as in this patient's presentation, a thorough evaluation of the cervical and intracranial circulation is mandatory.

The decision to pursue diagnostic cerebral angiography requires the clinician to weigh the risks and benefits of the procedure. In our patient, the duplex finding of a mobile lesion at the bifurcation suggested an unusual pathology. Angiography confirmed the presence of a saddle embolus, clearly defined its local extent, and ruled out the propagation of thrombus into the major intracranial arteries. When strong suspicion of thrombus at the carotid bifurcation is present, arch aortography must be obtained at the start of the angiographic procedure, to ensure that selective common carotid catheterization can be performed without dislodging proximal thrombi. In certain instances, adequate views of the carotid arteries may be obtained at arch aortography, obviating the need for selective catheterization. If selective angiography is required, it must be accomplished using low-flow, low-volume contrast injections, to minimize the chance of thrombus fragmentation and distal embolization.

Carotid angioplasty and stenting in this patient was contraindicated for several reasons. Passage of a distal cerebral protection device (filter or occlusion balloon) through the near-occlusive thrombus at the bifurcation might have resulted in distal embolization of the clot prior to deployment of the protection device. In addition, the heavy burden of thrombus would have overwhelmed the filling capacity of any of the filter-type cerebral protection devices. Furthermore, the extension of thrombus into both external and internal carotid arteries would have prohibited the safe reversal of internal carotid artery flow using the ArteriA device (Arteria Medical Sciences, San Francisco, CA) as wire passage into the external carotid artery is required to provide adequate support for guiding the ArteriA catheter into the common carotid artery. Lesser degrees of in situ thrombus noted during carotid stenting procedures have been successfully managed with intraarterial administration of glycoprotein IIb/IIIa inhibitors and lytic agents, but these medications may increase the risk of intracranial hemorrhage.^{13,14}

Recently, investigators have questioned the applicability of carotid stenting in the elderly. Most prominently, the Carotid Revascularization Endarterectomy versus Stenting Trial (CREST) investigators presented data from the lead-in phase of that randomized trial, demonstrating a disturbingly high rate of periprocedural stroke and death (13%) for octogenarians. Further data are required to clearly define the indications for carotid angioplasty and stenting, particularly in the elderly.¹⁵

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