From the Southern Association for Vascular Surgery

Cough-induced transient ischemic attack treated with revascularization of the external carotid artery

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A 65-year-old man presented with right arm and face weakness associated with severe coughing fits. A computed tomography angiogram revealed an occlusion of the left common and internal carotid arteries and an incomplete circle of Willis. An arch angiogram demonstrated reconstitution of the left external carotid artery, which collateralized with the intracranial left internal carotid artery. The patient underwent left subclavian-to-external carotid artery bypass with reversed saphenous vein, with complete resolution of symptoms over 1 year of follow-up. Cough-induced hemispheric transient ischemic attack is a rarely described hemodynamic phenomenon that can be managed with revascularization of the external carotid artery in select patients. (J Vasc Surg 2014;60:1657-60.)

We report a patient with the rare presentation of cough-induced hemispheric transient ischemic attack (TIA). The patient's symptoms resolved after revascularization of the external carotid artery (ECA).

CASE REPORT

The patient is a 65-year-old man who presented with recurrent episodes of transient right hand weakness and right facial droop that were concurrent with severe coughing episodes lasting seconds to minutes. The patient had a history of chronic bronchitis, and the right-sided symptoms had been occurring every 1 to 2 weeks for the last several months. On physical examination, the patient was normotensive, grossly neurologically intact, and no carotid bruits were detectable. Bilateral brachial and radial pulses were palpable and equal.

A carotid duplex examination showed moderate 40% to 59% stenosis (peak systolic velocity, 192 cm/s; end diastolic velocity, 73 cm/s) of the right internal carotid artery (ICA) and an occluded left common carotid artery (CCA) and ICA. Retrograde flow was visualized at the origin of the left ECA. The right vertebral artery had antegrade flow, and the left vertebral artery had bidirectional flow, suggestive of a proximal left subclavian artery stenosis.

A computed tomography angiogram (CTA) of the head and neck showed a bovine arch with moderate stenosis at the origin of the left subclavian artery. The left CCA was occluded just distal to its origin. The right vertebral artery was dominant and widely patent. A focal stenosis was noted at the origin of the left vertebral artery. The CTA of the head demonstrated an incomplete circle of Willis, with absent posterior communicating arteries bilaterally, and an atretic anterior communicating artery (Fig 1).

We therefore hypothesized that the patient had cerebral hypoperfusion that was exacerbated by coughing and manifesting as hemodynamic TIAs. To better assess whether the left side of the brain was dependent on cross-hemispheric collaterals vs collaterals from the ipsilateral ECA, we performed a carotid and cerebral angiogram (Figs 2 and 3).

The angiogram confirmed that collaterals from the left ECA were reconstituting the intracranial left ICA and that there was minimal cross-filling of the left hemisphere from the right ICA. A left subclavian angiogram also revealed a moderate stenosis at the left subclavian origin. We therefore brought the patient to the operating room for a left subclavian-to-ECA bypass with reversed saphenous vein.

After the systolic blood pressure was augmented to >160 mm Hg and the patient was fully heparinized, the left ECA was transected at the carotid bifurcation, and an endarterectomy of the ECA origin was performed. The remainder of the ECA was widely patent. There was some back bleeding.

The bypass was created in an end-to-side fashion to the left subclavian artery distal to the origin of the vertebral artery and in an end-to-end fashion to the ECA. The occluded carotid bulb was then oversewn. Because of the moderate proximal left subclavian stenosis with bidirectional flow in the left vertebral artery, angioplasty and stenting with a balloon-expandable bare-metal stent of the left subclavian origin was performed to prevent steal syndrome and to ensure good inflow for the bypass graft.

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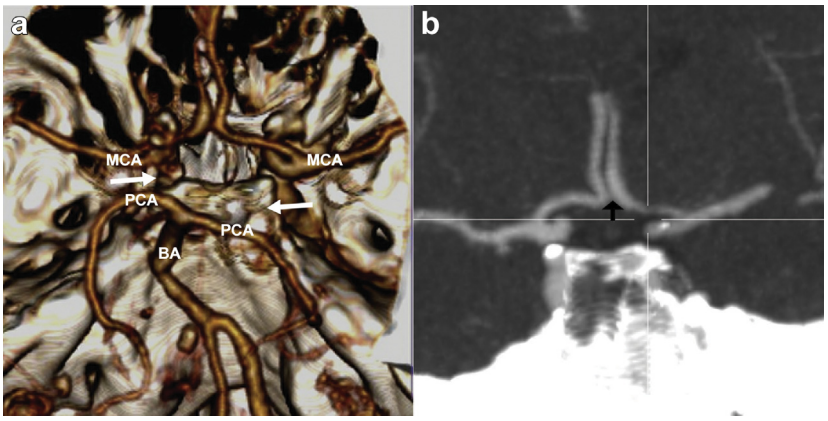

Fig 1. a, A computed tomography angiogram (CTA) of the head revealed an incomplete circle of Willis. The *arrows* indicate absent posterior communicating branches. *BA,* Basilar artery; *MCA,* middle cerebral artery; *PCA,* posterior cerebral artery. **b,** The anterior communicating artery (*black arrow*) was barely detectable.

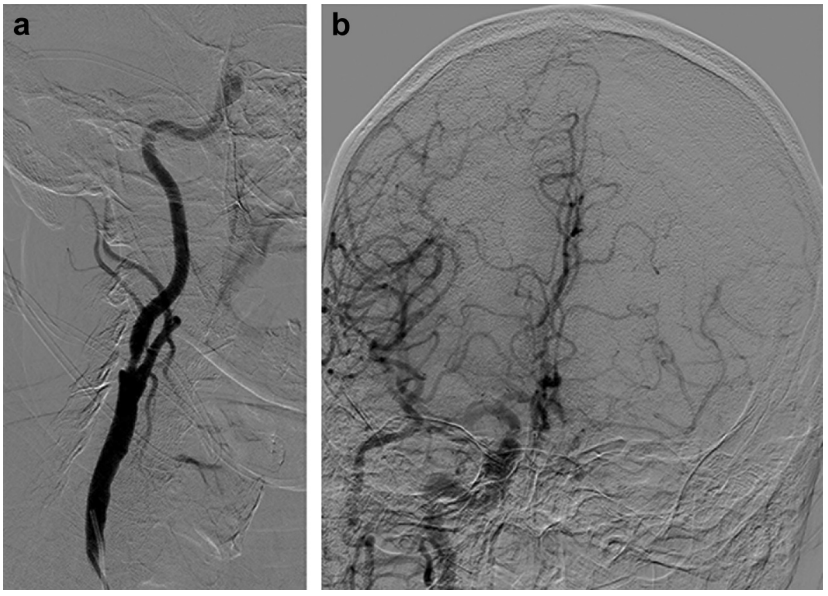

Fig 2. a, A right carotid angiogram confirmed moderate (<50%) stenosis of the right internal carotid artery (ICA). **b,** A cerebral angiogram from the right side demonstrated minimal cross-hemispheric filling from the right to the left.

Immediately after the operation, the patient had complete resolution of his right-sided weakness that occurred during coughing episodes. He was discharged to home 3 days postoperatively, without any incident.

Follow-up duplex examinations at 1 month and 6 months showed antegrade flow in the left ECA, and a CTA at 11 months demonstrated a patent bypass graft and subclavian stent. Assessments of perfusion of the left hemisphere during clinical follow-up out to 1 year have been significant for complete resolution of neurologic symptoms, despite multiple recurrent episodes of bronchitis. The patient consented to the publication of this case report.

DISCUSSION

Hemodynamic strokes have been defined as TIAs or stroke symptoms associated with certain precipitating circumstances, such as orthostatic hypotension (ie, shaking-limb TIA), exercise, eating, coughing, and antihypertensive medications.[1] These events commonly divert blood flow to other organ systems, thus resulting in decreased cerebral blood flow. The similar phenomenon of cough syncope resulting from extracranial carotid disease has been reported,[2] and one case report documented cough-induced TIA secondary to ICA occlusion was managed with permissive hypertension.[3]

Coughing is believed to result in cerebral arterial vasoconstriction secondary to hypercapnia, decreased venous return, and decreased cardiac output secondary to Valsalva.[2] These events together can lead to global cerebral hypoperfusion, and in some patients, the phenomenon of cough syncope, or as in this patient, a TIA. To our knowledge, this is