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# Positional transient loss of consciousness and hemispheric deficits in the setting of severe four-vessel extracranial cerebrovascular disease

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## ABSTRACT

Carotid artery stenosis typically causes hemispheric neurologic effects by atheroembolism. Nonhemispheric symptoms, such as syncope, are generally not attributable to extracranial carotid disease. This report describes a 62-year-old woman with severe bilateral carotid artery stenosis, right vertebral artery occlusion, and severe left vertebral artery stenosis who presented with transient loss of consciousness and unilateral weakness when upright. Her symptoms resolved after right carotid endarterectomy. Whereas vertebrobasilar insufficiency alone can cause syncope, in the case of severe multivessel cerebrovascular disease, unilateral carotid revascularization was successful in treating the patient's transient loss of consciousness, suggesting global cerebral hypoperfusion as the cause. (*J Vasc Surg Cases and Innovative Techniques* 2019;5:461-6.)

**Keywords:** Carotid stenosis; Vertebrobasilar; Hypoperfusion; Positional

## CASE REPORT

A 62-year-old woman with a history of tobacco abuse, chronic type B aortic dissection, and hypertension presented with several months of positional presyncope, syncope, left-sided weakness, and facial droop. She described resolution of her symptoms after lying supine for several minutes. The patient had adapted by remaining essentially bedbound.

On examination, she was normotensive but with a left brachial systolic pressure 40 mm Hg lower than on the right. She was neurologically intact while supine. After sitting upright, she developed dizziness, lethargy, left-sided weakness, and facial droop that resolved after lying supine. She did not have any evidence of orthostasis as measured by right brachial pressures.

Computed tomography angiography demonstrated diffuse moderate stenosis of the right common carotid, 90% to 99% stenosis (North American Symptomatic Carotid Endarterectomy Trial [NASCET] criteria) of the right internal carotid, diffuse high-grade stenosis of the left common carotid, and 90% to 99% stenosis of the left internal carotid artery (Figs 1 and 2). The right vertebral artery was occluded at its origin with reconstitution of the V2 segment, and the left vertebral artery had severe stenosis in the V1 and V2 segments. The first part of the left axillary artery was also occluded. The circle of Willis was intact (Fig 3).

Magnetic resonance imaging of the brain showed multiple small infarcts bilaterally in the watershed areas, more significant on the right (Fig 4). Of note, computed tomography angiography of the chest, abdomen, and pelvis showed the patient's chronic type B aortic dissection to be unchanged from previous scans in our system.

Given the patient's constellation of findings, we concluded that there was global cerebral hypoperfusion, including symptomatic right internal carotid artery stenosis. The patient was continued on dual antiplatelet and high-dose statin therapies, and urgent right carotid endarterectomy was offered as the initial step in revascularization. Our standard practice favors open endarterectomy over stenting in most circumstances, and the patient was agreeable to this.

Intraoperatively, we noted diffuse disease of the right common carotid, severe disease of the internal carotid extending to the skull base, and moderate disease at the external carotid artery origin. Because there was considerable redundancy of the internal carotid artery, after mobilization, we were able to obtain high exposure just with gentle traction on the vessel. We placed a shunt past the distal aspect of the lesion. With a shunt in place, we performed an extensive endarterectomy of the right common and internal carotid arteries. We also noted a focal, ulcerated plaque with fresh thrombus eroding through the posterior wall of the carotid bulb, which we resected and repaired primarily (Fig 5). Finally, we performed patch angioplasty with a bovine pericardial patch.

Postoperatively, the patient was neurologically intact. At the 1-month postoperative visit, she reported sitting upright routinely, without any focal deficits and no additional episodes of loss of consciousness. Although the hypoperfusion symptoms were resolved, we counseled the patient on treatment of the asymptomatic high-grade left internal carotid artery stenosis. We discussed hybrid endarterectomy and retrograde stenting of the common carotid for the left side, but the patient was reluctant to undergo a second open procedure. There was also

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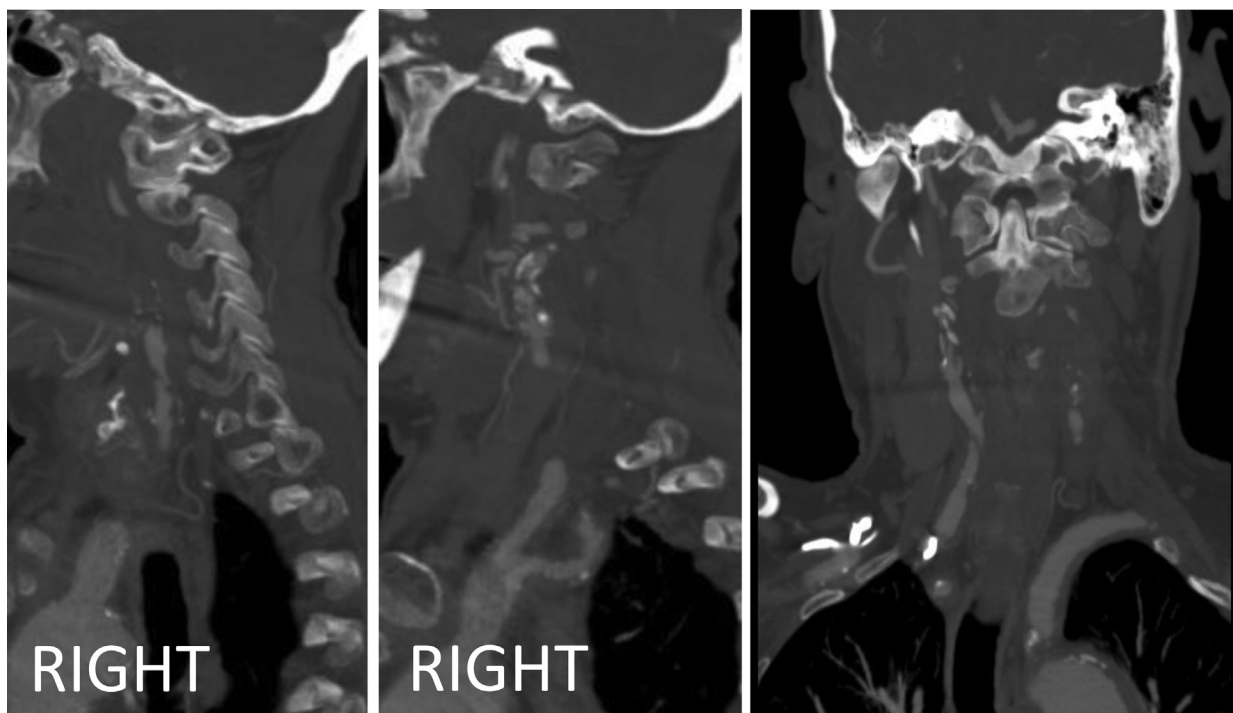
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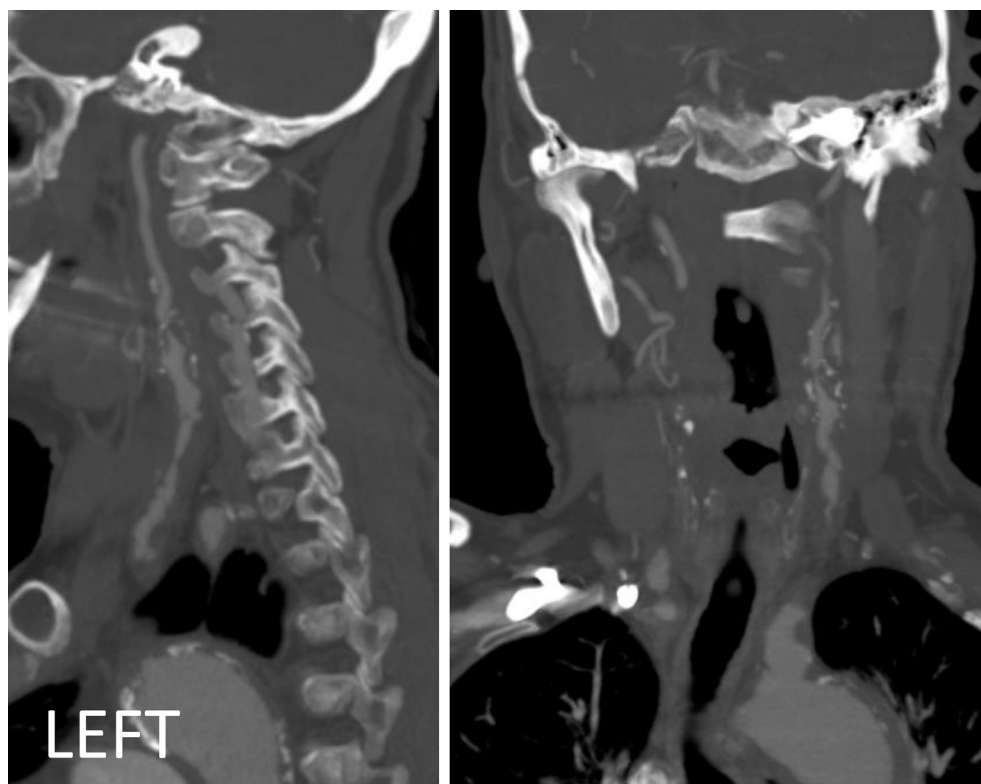
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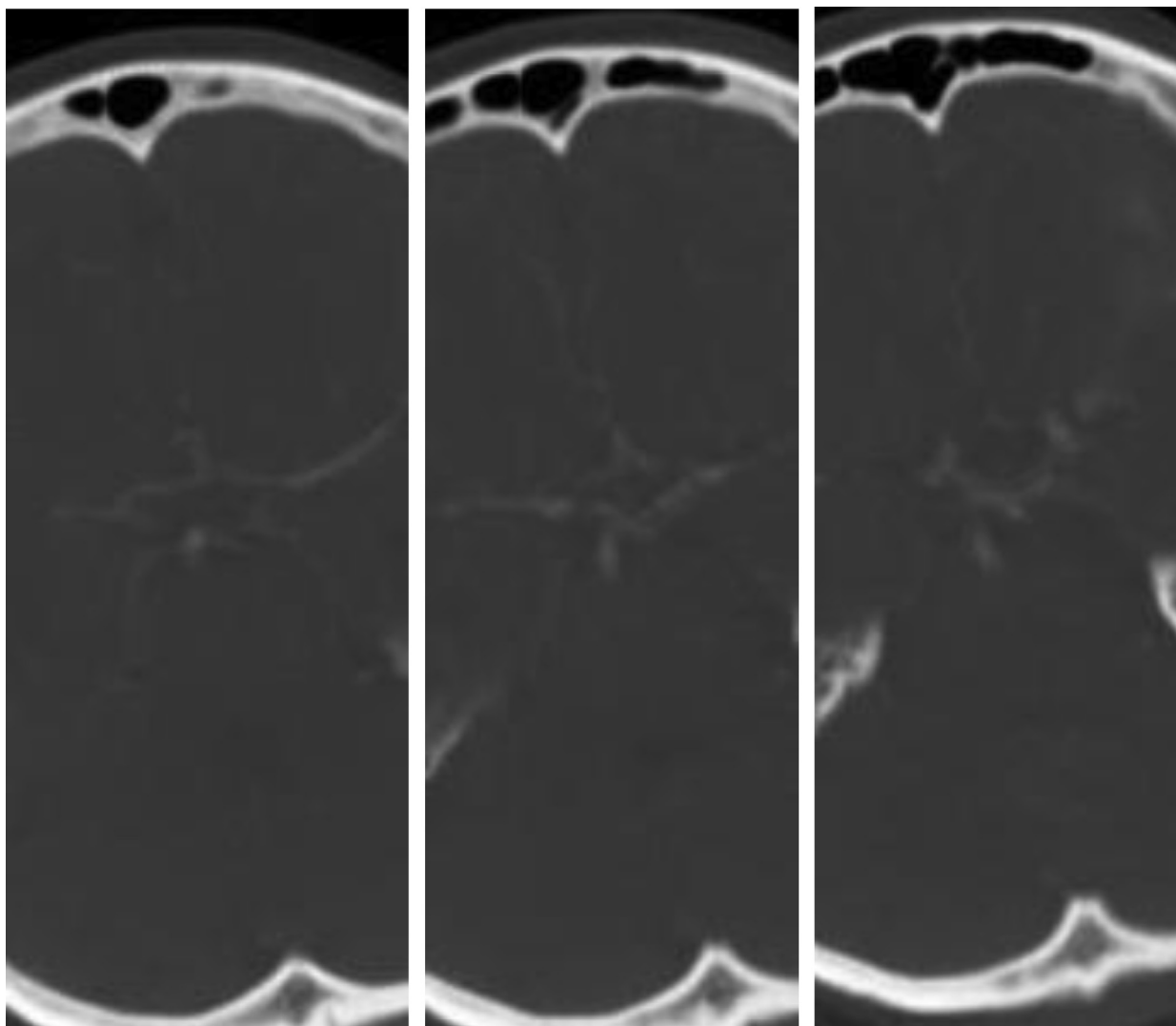
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**Fig 1.** Preoperative computed tomography angiogram demonstrating severe, multifocal stenosis of the right internal carotid artery.



**Fig 2.** Preoperative computed tomography angiogram showing severe, multifocal stenosis of the left internal and common carotid arteries.



**Fig 3.** Preoperative axial computed tomography angiogram demonstrating the patient's intact circle of Willis (cephalad to caudad).

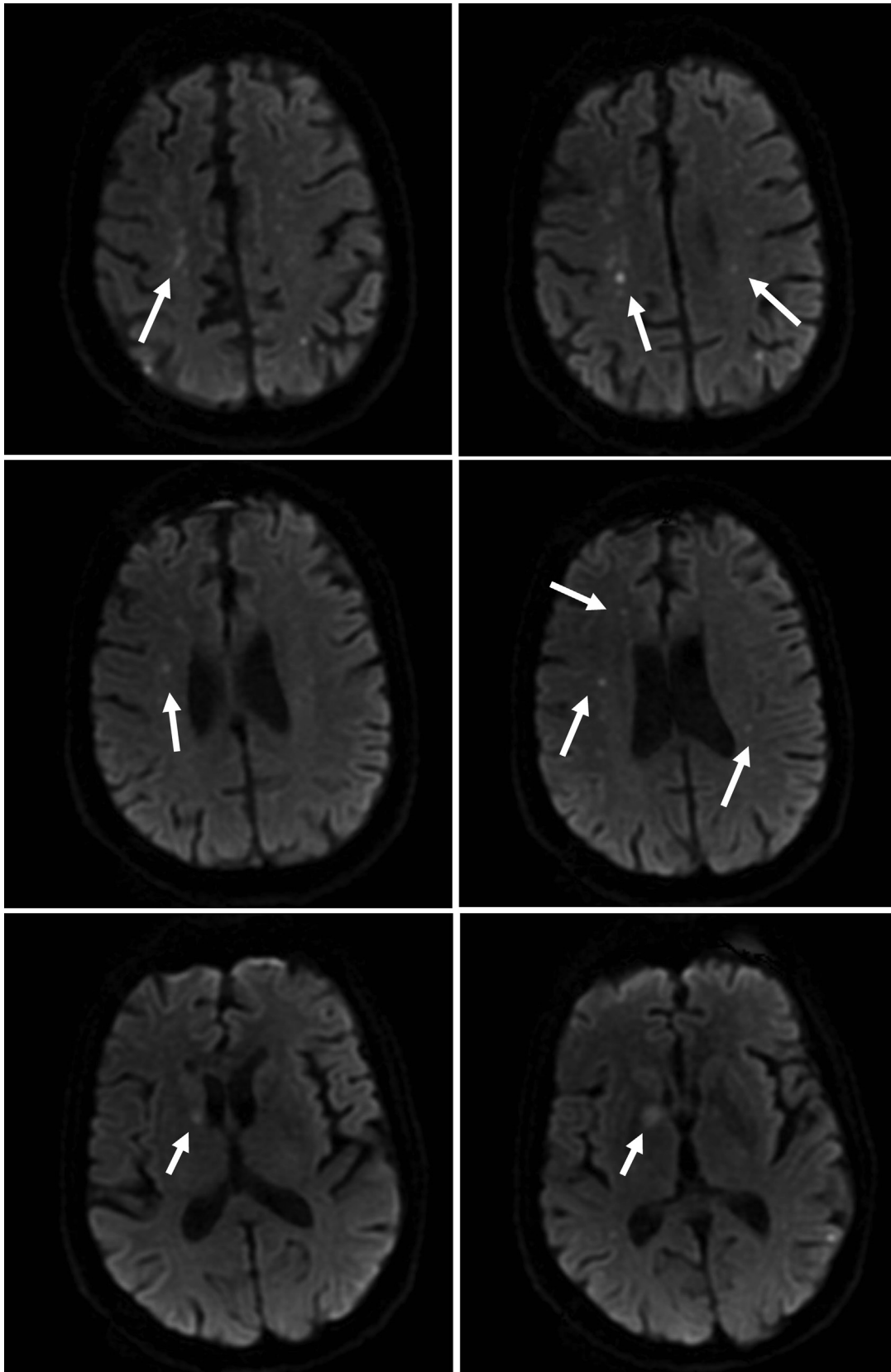
an ongoing evaluation by otolaryngology for long-standing voice hoarseness, which revealed a right vocal cord polyp with decreased mobility. These factors led to a shared decision that transfemoral left carotid artery stenting was most appropriate. The patient consented to publication of this report.

## DISCUSSION

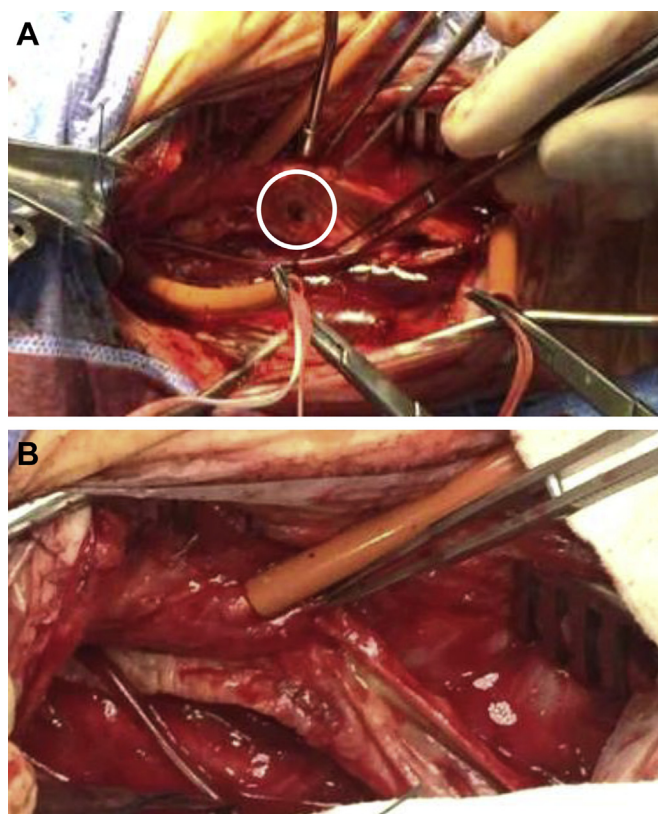
We report a case of positional loss of consciousness and focal neurologic deficits associated with severe multivessel extracranial cerebrovascular disease that was alleviated with right carotid endarterectomy. Carotid artery stenosis typically is manifested with hemispheric deficits caused by embolization from atherosclerotic plaques,<sup>1</sup> and revascularization is performed to prevent embolic stroke.<sup>2</sup> However, severe extracranial cerebrovascular occlusive disease can cause transient ischemic attacks and ischemic strokes in patients with reduced cardiac function or shock.<sup>3</sup> In addition, a link has been

demonstrated between extracranial cerebrovascular disease and decreased vasomotor reactivity within the brain, resulting in susceptibility to infarcts in watershed distributions.<sup>4</sup> In our case, the patient demonstrated nonhemispheric symptoms consistent with vertebrobasilar insufficiency as well as hemispheric symptoms consistent with carotid disease, but both were associated only with an upright position.

Whereas several syndromes of vertebrobasilar insufficiency have been described,<sup>5-7</sup> cases of provoked, reproducible, transient neurologic ischemic symptoms in the anterior circulation are rare. These include syndromes of recurrent episodes of aphasia caused by orthostatic hypotension<sup>8</sup> and reversible unilateral weakness caused by postprandial hypotension<sup>9</sup> and head turning.<sup>10,11</sup> Another case described a patient with multivessel extracranial cerebrovascular disease suffering postural aphasia, focal weakness, and syncope, reflecting ischemia



**Fig 4.** Select axial sequences from preoperative T2-weighted magnetic resonance imaging studies (cephalad to caudad) showing multiple hyperintense foci in both hemispheres, distributed in a periventricular pattern typical of acute watershed infarcts.



**Fig 5.** Intraoperative images demonstrating an ulcerated plaque (circle) eroding through the posterior carotid bulb, before (A) and after (B) primary repair.

affecting multiple vascular territories.<sup>12</sup> None of these patients underwent surgical intervention.

However, surgical revascularization has been described in other cases. A patient with a left carotid occlusion who suffered right upper extremity weakness and facial droop only while coughing was noted to improve after subclavian-external carotid bypass.<sup>13</sup> Two reports have described syncope associated with severe bilateral carotid stenosis successfully treated with carotid revascularization.<sup>14,15</sup> In contrast, we describe the successful surgical treatment of an extracranial vascular lesion leading to resolution of both hemispheric and nonhemispheric positional symptoms.

In our case, the patient suffered reproducible hemispheric and nonhemispheric symptoms, consistent with global cerebral hypoperfusion: in the anterior circulation, manifested as unilateral facial droop and left-sided weakness; and in the posterior circulation, manifested as dizziness and syncope. The four-vessel extracranial occlusive disease was severe enough to produce symptoms when the patient was sitting upright, even in the absence of measurable hypotension or orthostasis. Subsequent imaging confirmed punctate infarcts in watershed territories of the brain consistent with global

cerebral hypoperfusion.<sup>16</sup> The priority was management of the right carotid disease as this was deemed the most unstable and potentially devastating lesion. After unilateral carotid endarterectomy, her postural symptoms completely resolved, presumably secondary to increased global cerebral perfusion rather than isolated vertebrobasilar insufficiency.

## CONCLUSIONS

Severe multivessel extracranial cerebrovascular disease resulting in global hypoperfusion is a rare and poorly described clinical phenomenon. This disease pattern can cause transient ischemic attacks affecting multiple vascular distributions within the brain. This case demonstrates that isolated carotid revascularization may alleviate even the posterior circulation symptoms, resulting in a major improvement in quality of life for patients with this single intervention. Revascularization of the other three vessels should be entertained only if it is otherwise clinically warranted after this first procedure.

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