Bypass grafting to the third segment of the vertebral artery for symptomatic extensive vertebrobasilar atherosclerotic disease

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ABSTRACT

Symptomatic vertebrobasilar atherosclerotic disease is rarely encountered but represents a high-risk factor for recurrent transient ischemic attack or stroke. Posterior strokes are usually associated with embolism or hemodynamic impairment. Extensive disease involving the V3 and V4 segments of the vertebral artery (VA) remains infrequent, and optimal management is limited owing to the infrequency of this disease. We illustrate the case of a 65-year-old man who presented with recurrent transient episodes of dizziness with acute onset of instability, nausea, and left visual blurring. Magnetic resonance imaging findings of the head were normal, and computed tomography angiography revealed severe atherosclerotic disease of both VAs, with proximal occlusion of the right VA and multiple tight stenoses of the left VA at the V1 and V4 segments. Duplex ultrasound found markedly reduced anterograde flow in the VAs and basilar arteries and nonsignificant stenosis of the internal carotid arteries. Optimal medical treatment led to a decrease of transient symptoms. However, the patient developed a cerebellar infarction in the left posteroinferior cerebellar artery territory with left VA V4 segment occlusion. Surgical revascularization of the right VA was decided by the multidisciplinary team. Through an anterolateral approach of the right VA V3 segment, revascularization was performed using a common carotid artery-to-right VA bypass using a reversed saphenous vein graft. The patient fully recovered and was free of symptoms during the next 14 months of follow-up. In the case of extensive VA occlusive disease, surgical reconstruction of the distal VA using a bypass from the common carotid artery represents an option to improve hemodynamics and/or eliminate an embolic source of posterior stroke on a case-by-case basis. (J Vasc Surg Cases Innov Tech 2023;9:101260.)

Keywords: Bypass grafting; Open surgical repair; Posterior circulation strokes; Vertebrobasilar insufficiency

Posterior circulation strokes account for ~30% of all ischemic strokes, and the main cause is vertebrobasilar atherosclerotic disease, in addition to cardiac sources of embolism.[1] Although artery-to-artery embolism is the main mechanism of posterior circulation ischemic strokes, hemodynamic impairment is frequently present and creates the condition for an embolic occlusion that could not be compensated for by collateral vessels.[2] Specific anatomic settings, such as bilateral severe vertebral artery (VA) stenosis, unilateral stenosis with the contralateral VA ending in a posteroinferior cerebellar artery, and atresic posterior communicating arteries, increase the risk of recurrent posterior stroke.[3] This emphasizes the need for posterior circulation revascularization in such patients, usually in the VA proximal segment. Distal VA revascularization is infrequent, with indications including tumor involvement, dynamic compression, intrinsic lesions (ie, stenosis, occlusion), and aneurysms.[4-6] We illustrate this with a case of severe symptomatic hemodynamic compromise in the vertebrobasilar circulation treated by open surgical revascularization. The patient provided written informed consent for the report of his case details and imaging studies.

CASE REPORT

A 65-year-old man with a history of ankylosing spondylitis, human immunodeficiency virus seropositivity, and hypertension was referred to a transient ischemic attack (TIA) clinic with round-the-clock access because of recurrent transient episodes of dizziness. His symptoms had started 1 month before his admission and occurred two to three times daily. He reported no triggering factors and that the attacks could occur while standing or lying down but not with a change

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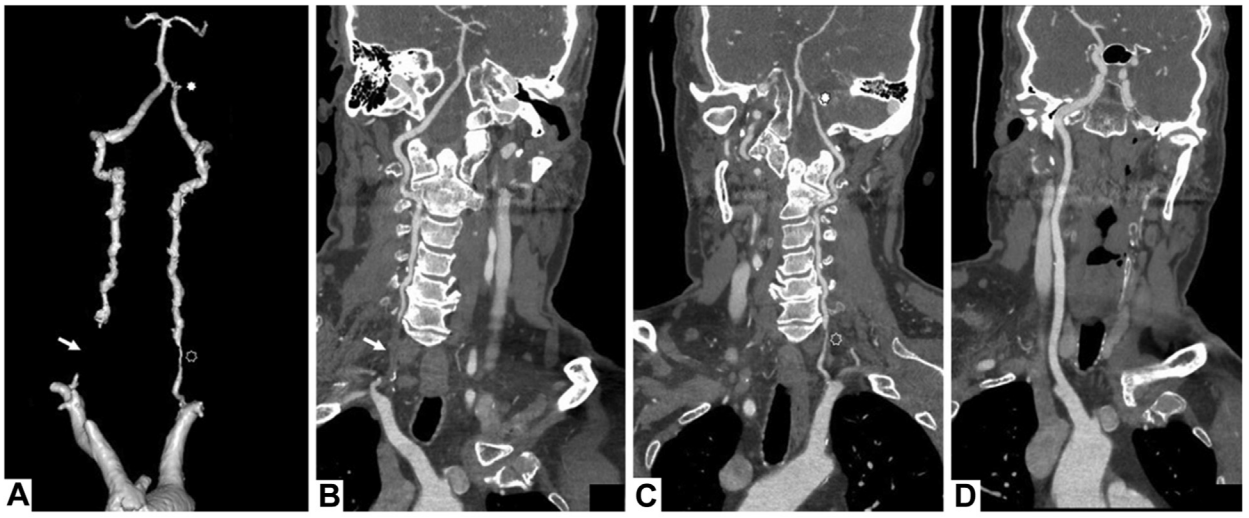

Fig 1. Three-dimensional reconstruction **(A)** and curvilinear reconstructions of the preoperative computed tomography angiography (CTA) of the right vertebral artery (VA; **B**), left VA **(C)**, and right internal carotid artery (ICA; **D**) showing occlusion of the right V1 segment (*arrow*), a tight atherosclerotic stenosis of the left V1 (*empty star*), and a tight stenosis of the left V4 (*full star*).

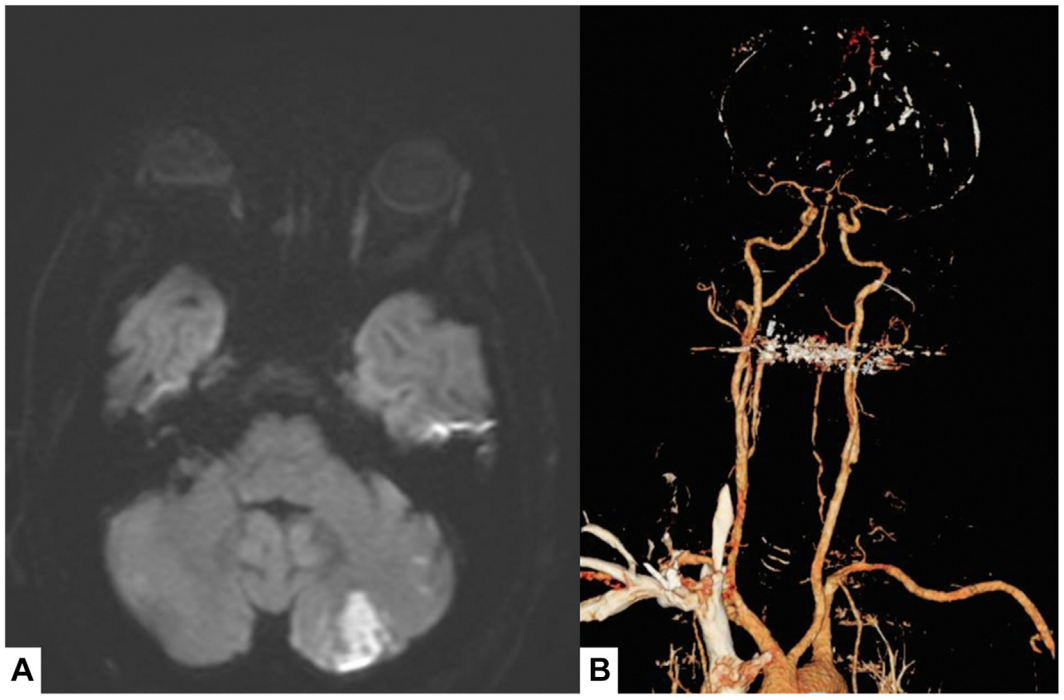

Fig 2. A, Diffusion-weighted magnetic resonance image showing left hemispheric cerebellar acute ischemic stroke. **B,** Computed tomography angiography (CTA) revealed a new occlusion of the V4 segment of the left vertebral artery (VA).

in head position. He described the acute onset of instability, nausea, left visual blurring, and painful contracture below the external occipital protuberance that spread into the back of the neck and resolving rapidly within 30 seconds. The neurologic examination findings were normal between attacks.

The magnetic resonance imaging findings of the head were normal, and computed tomography angiography (CTA) revealed severe atherosclerotic disease of the vertebral arteries (VAs; Fig 1, *A*). The right VA displayed proximal occlusion on its V1 segment (Fig 1, *B*). The left VA had multiple atherosclerotic stenoses, including two tight ones at its V1 and V4 segments

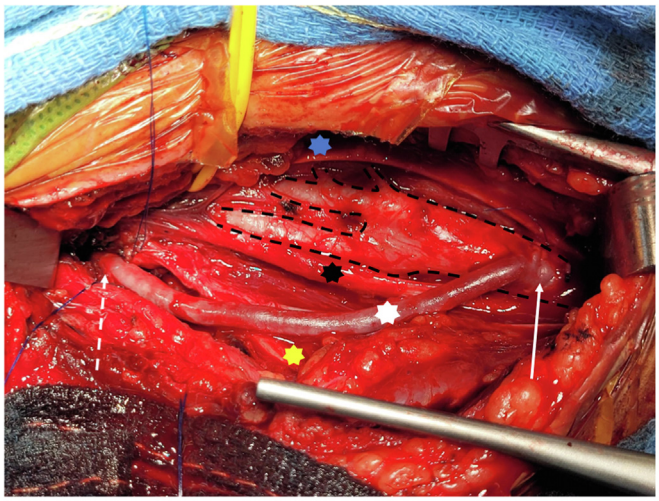

Fig 3. Intraoperative view after saphenous vein bypass grafting. Anterolateral approach of the right vertebral artery (VA) with a retrojugular approach of the carotid artery bifurcation. *Black dotted line* indicates right common carotid artery bifurcation; *blue star,* internal jugular vein moved medially; *black star,* vagus nerve; *yellow star,* accessory spinal nerve; *white star,* reversed saphenous vein bypass; *white arrow,* proximal anastomosis performed on right common carotid artery; *white dotted arrow,* distal anastomosis on V3 segment of the right VA. The anterior branch of the C2 root has been sectioned. Both ends are towed by stitches on each side of the distal anastomosis.

(Fig 1, *C*). Duplex and color flow Doppler ultrasound examinations revealed anterograde flow in the VAs and basilar arteries, but markedly reduced, and no left or right carotid artery lesion (Fig 1, *D*).

TIAs related to severe large artery atherosclerosis were diagnosed, but their mechanism was uncertain. We first hypothesized a hemodynamic mechanism because the flow was severely impaired in the whole posterior circulation and the transient episodes were strictly stereotyped. His antihypertensive therapy was stopped, aspirin was continued, and a revascularization procedure was scheduled. The intensity and frequency of the transient symptoms decreased; however, 5 weeks later, the patient developed a symptomatic occlusion of the V4 segment of the left VA (Fig 2, *A*) with a new cerebellar infarction in the left posteroinferior cerebellar artery territory (Fig 2, *B*), leading us to believe, a posteriori, that the TIAs were of atherothrombotic origin. Considering the symptoms and severity of the atherosclerosis process in the vertebrobasilar circulation, open surgical revascularization of the right VA was decided, and the patient consented to the procedure.

Surgical technique. With the patient under general anesthesia, VA exposure was performed in the V3 segment through a retrojugular anterolateral approach, as described by George and Laurian.[7,8] A skin incision was drawn on the anterior aspect of the sternocleidomastoid muscle to the level of the mastoid process on its upper part. The internal jugular vein was moved medially. The right common carotid artery (CCA) was dissected. The vagus nerve was carefully respected. The accessory spinal nerve was dissected and mobilized. The right VA was exposed between both transverse processes of the first and second cervical vertebrae (C1 and C2). The levator scapulae muscle, which inserts on the transverse process of C1 vertebra, was carefully cut. It allowed for exposure of the anterior branch of the C2 nerve, which needs to be sectioned with no clinical consequences. The VA was exposed for almost 2 cm (Fig 3). An 8- to 10-cm segment of the crural great saphenous vein was harvested. The venous collateral vessels were ligated using 6-0 polypropylene suture. An intravenous bolus of 50 UI/kg of sodium heparin was injected before arterial cross-clamping. The right terminal CCA was clamped, with the systolic blood pressure maintained at >140 mm Hg. A 10-mm longitudinal arteriotomy was performed, and a lateral–terminal anastomosis between the CCA, and the reversed saphenous vein graft was created using a 6-0 polypropylene running suture. After standard purges, the carotid artery clamps were released, and the saphenous vein bypass was clamped. The VA was cross-clamped using two adjustable pressure bulldog clips. A 5-mm longitudinal arteriotomy was performed on the VA, and a terminal–lateral anastomosis created using 8-0 polypropylene running suture. After the purges and clamp release, an immediate intraoperative Doppler ultrasound examination was performed.

Postoperative course. The patient recovered fully immediately after revascularization with disappearance of the preoperative symptoms. The right cranial nerve test findings were normal. He was free of recurrent events during the next 14 months of follow-up. Postoperative duplex ultrasound and CTA demonstrated bypass patency with no complications (Fig 4, *A* and *B*).

DISCUSSION

Large artery atherosclerosis is the cause of cerebellar infarctions in approximately one half of cases,[9-12] and one half of these cases could be the consequence of hypoperfusion related to intracranial VA (ICVA) occlusive disease.[2,9] The origin of the extracranial VA (ECVA) is one of the most prevalent location of posterior circulation atherosclerosis,[12] present in ~30% of patients with posterior circulation stroke.[9] The etiology of posterior strokes appears to be a combination of embolic and hemodynamic phenomena in the vast majority of cases.[2] In the present case, the symptoms were first thought to result from hemodynamic impairment of the posterior circulation. The mechanism of the second onset of symptoms was acute thrombosis of the left VA on a preexisting stenosis, complicated by cerebellar stroke. The present case represents an example of a heterogeneous etiology of posterior strokes.

Concomitant internal carotid artery (ICA) and VA stenosis are frequent; however, patients presenting for combined ICA and VA reconstruction remain at high risk of postoperative stroke.[13] These patients are possibly more sensitive to the effects of ischemia during clamping

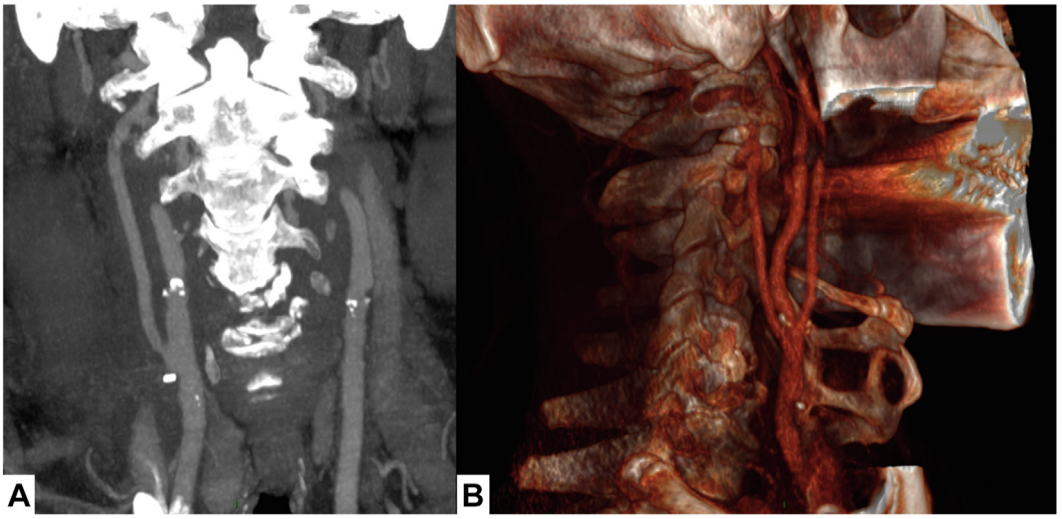

Fig 4. A, Multiplanar reconstruction of computed tomography angiography (CTA) at 6 months postoperatively showing patency of the venous bypass and distal vertebral artery (VA) revascularization. **B,** CTA with volume-rendering technique reconstruction revealing three-dimensional anatomic relations of the distal anastomosis between the transverse processes of the first and second cervical vertebrae.

and to reperfusion injury.[13] Sequential ICA and VA revascularization can be considered for such patients.[13]

Revascularization procedures using stenting or surgery (ie, transposition, bypass, endarterectomy) for ECVA and ICVA atherosclerotic stenosis have been reported in case series, but large randomized trials are lacking. The performance of a large randomized controlled trial of posterior stroke is compromised by the rarity of the disease and heterogeneity of the clinical presentation due to anatomic variations, effectiveness of the collateral vessels, and number of stenotic lesions.

In the case of occlusive disease of the V1 segment of the VA not suitable for endovascular repair, open revascularizations modalities include direct transposition of the VA into the common carotid artery, trans-subclavian VA endarterectomy, or vein bypass from the subclavian artery.[14] Such repairs were not indicated for our patient because of bilateral long occlusive disease in the V1 segments. Tandem ICVA and ECVA severe atherosclerotic stenosis, occurs in ~25% of patients.[9] In such cases, hemodynamic TIA related to the ECVA has rarely been reported.[9] Because of the precarious hemodynamic status of the posterior circulation, his symptomatic status, and the need to maintain antihypertensive therapy in the long term, we chose revascularization of the right distal VA. The surgical options for distal ECVA revascularization include transposition of the VA into the ICA and bypass grafting.[14] Revascularization using transposition of the external carotid artery or occipital artery are options if no suitable graft is available.[13] Regarding the size of the VA in the V3 segment and the better patency of a venous bypass, a reversed great saphenous vein graft remains the choice for a bypass between the common carotid artery and the V3 segment of the VA, above C2, as described by George (a neurosurgeon) and Laurian (a vascular surgeon).[7]

CONCLUSIONS

Severe symptomatic hemodynamic compromise in the vertebrobasilar circulation is rarely encountered but can cause disabling stroke despite advances in medical management. Revascularization of the distal ECVA using a venous graft provides a safe and efficient option for these patients on a case-by-case basis.

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