Case Report

Intradural vertebral endarterectomy with nonautologous patch angioplasty for refractory vertebrobasilar ischemia: Case report and literature review

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Abstract

Background: The natural history of patients with symptomatic vertebrobasilar ischemic symptoms due to chronic bilateral vertebral artery occlusive disease is progressive, and poses significant challenges when refractory to medical therapy. Surgical treatment options depend largely on location and characteristics of the atheroma (s), and generally include percutaneous transluminal angioplasty (PTA) with or without stent placement, posterior circulation revascularization (bypass), extracranial vertebral artery reconstruction, or vertebral artery endarterectomy.

Case Description: We present the case of a 56-year-old male with progressive vertebrobasilar ischemia due to tandem lesions in the right vertebral artery at the origin and intracranially in the V4 segment. The contralateral vertebral artery was occluded to the level of posterior inferior cerebellar artery (PICA) and posterior communicating arteries were absent. Following PTA and stent placement at the right vertebral artery origin, the patient was successfully treated with intradural vertebral artery endarterectomy (V4EA) and patch angioplasty via the far lateral approach. Distal endovascular intervention at the V4 segment proved not technically feasible after multiple attempts.

Conclusions: V4EA is an uncommonly performed procedure, but may be considered for carefully selected patients. The authors' techniques and indications are discussed. Historical outcomes, relevant anatomic considerations, and lessons learned are reviewed from the literature.

Key Words: Endarterectomy, patch angioplasty, vertebral stenosis, vertebrobasilar ischemia

INTRODUCTION

Symptomatic intracranial vertebral artery (VA; V4 segment) thrombo-occlusive disease, its natural history, and associated treatment strategies have not been as rigorously studied in comparison to stroke phenomena originating from the internal carotid artery.[6,8,10,22,29]

Surgical interventions for patient's refractory to medical therapy consist principally of open arterial bypass,

or percutaneous transluminal angioplasty (PTA) and stent placement. Posterior fossa bypass procedures are technically demanding with significant risk of morbidity, particularly in the setting of graft occlusion.[5,14]

Intradural vertebral (V4 segment) endarterectomy (V4EA) is an uncommonly performed revascularization alternative. Despite inherent advantages, the applicability of V4EA is limited due to narrow indications for a highly selected population of patients, limited historical data, and multiple anatomic and technical constraints. We present a case of symptomatic, refractory intracranial VA occlusive disease treated successfully via V4EA with patch angioplasty after failed endovascular intervention. Operative technique and the prior literature are briefly reviewed to formulate the best indications and strategies for obtaining improved outcomes.

CLINICAL PRESENTATION

Presentation

A 56-year-old male was transferred to the inpatient neurology service from an outside institution with complaints of progressive and intermittent vertigo, ataxia, left-sided weakness, and tinnitus for the prior 10 days. The patient's symptoms were initially postcoital, but without associated headache. He additionally endorsed a single similar episode 6 months earlier that was self-limited. His past medical history was remarkable for hypertension and hyperlipidemia. He took no medications, and did not smoke or use tobacco.

Initial neurological examination revealed normal mental status, cranial nerve findings, motor examination, reflexes, sensation, drift, and cerebellar signs (including no evidence of dysdiadochokinesia). Magnetic resonance imaging (MRI) evaluation from the outside institution displayed evidence of multiple discrete foci of diffusion restriction in the inferior right cerebellar hemisphere. Computed tomography (CT) angiography obtained on admission at our institution showed absent left vertebral flow below posterior inferior cerebellar artery (PICA) and right VA origin stenosis [Figure 1]. The remainder of his extracranial and intracranial vasculature was unremarkable. Standard laboratory evaluations, including hyper-coagulability studies (including factor V Leiden, activated protein C/S) were negative. The patient was heparinized, monitored to avoid hypotension, and endovascular consultation was obtained by the admitting service.

Angiographic findings and procedures
Digital subtraction angiography revealed tandem 90% stenosis at the right VA origin and near occlusion (~99% stenosis) more distally just beyond the V3/V4 junction. The right PICA failed to opacify. The left VA was occluded at the origin, and did not reconstitute until the level of PICA via limited opacification from the contralateral circulation. Posterior communicating arteries, notably, were not apparent on the angiogram or pre-procedure CT angiography. Allcock's test, however, was not performed.
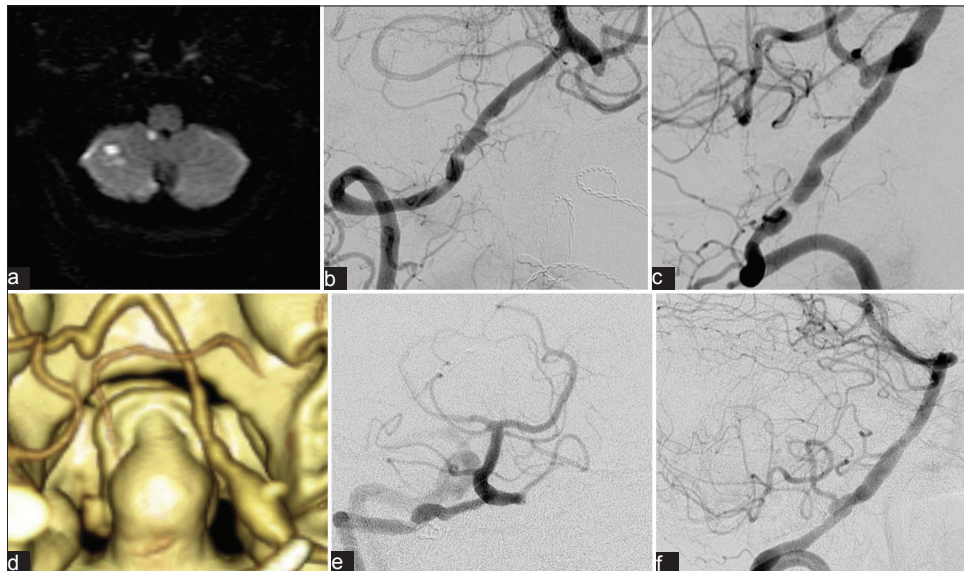

Figure 1: (a) Preoperative axial MRI diffusion-weighted imaging depicting multiple, scattered, and punctate areas of restricted diffusion within the right cerebellar hemisphere. A large territory brainstem or cerebellar stroke was not evident. (b) Anteroposterior (AP) and (c) lateral preoperative DSA displaying severe, near-total occlusive focal plaque at the V4 segment. The ipsilateral PICA does not opacify. (d) 3D-reconstructed images from the postoperative CT-angiogram revealing patency and slight dilation at the endarterectomy site. A single clip at the transected posterior meningeal branch is also seen. (e) Postoperative AP and (f) lateral DSA 10 months postsurgery reveal patency without significant change at the endarterectomy site

The right VA origin stenosis was successfully treated with deployment of a 4.0 × 12 mm balloon-mounted coronary stent (Multi-Link Vision, Abbott Vascular, Abbot Park, IL). The patient was loaded and subsequently maintained on clopidogrel and aspirin. However, further attempts to navigate a microwire and guide catheter beyond the stent and distal to the intracranial stenosis were unsuccessful due to proximal vessel tortuosity. The patient awoke without new neurological deficit. Repeat attempts via right brachial access the following day were similarly unsuccessful, and further attempts at treatment of the distal stenosis were aborted after concern for subintimal migration of the guidewire. Final angiographic runs did not reveal clear evidence of arterial dissection.

The patient again awoke without deficit, and CT imaging was negative for evidence of gross new ischemia or hemorrhage, but diffusion MRI showed numerous small hits in the cerebellar hemispheres. The patient's symptoms became increasingly labile over the following 2 weeks postprocedure in the hospital. Symptoms referable to the right PICA territory (as previous) were reproduced with systolic blood pressures less than 180 mmHg or slight deviation from the recumbent position. The senior author (PN) was then consulted for possible posterior-fossa revascularization. Clopidogrel was discontinued after the patient was transitioned to full heparin anticoagulation in anticipation of surgery.

Surgical management and technique [Video 1]

Two weeks following the initial angiogram, the patient underwent right far lateral craniotomy in anticipation of occipital artery-PICA bypass as a first choice or endarterectomy as a second. Following placement of a lumbar drainage catheter, the patient was padded, placed in the radiolucent head-frame, and secured in the park-bench position. Somatosensory evoked potentials (SSEP) and electroencephalography baselines were obtained prior to incision. The patient's blood pressure was supported at preoperative levels with neosynephrine throughout the procedure.

A standard semilunar "hockey-stick" style myocutaneous flap was employed. The occipital artery was identified and partially dissected distally during the exposure. Prior to skin incision, audible flow could not be demonstrated along the course of the occipital artery, and the caliber of the artery ultimately proved feasible but suboptimal for bypass. Bony removal, including C1 laminectomy, proceeded in the standard fashion. Limited condylar drilling under microscopic visualization was performed to facilitate improved dural/vascular mobilization, eliminate any mechanical constraints, and optimize visualization and trajectory to the VA. The dura was incised along the superior and medial borders of the craniotomy, and tacked laterally.

Details and tenets of the far lateral approach have been reviewed elsewhere, and are beyond the scope of the current report.

Following arachnoidal opening and cerebrospinal fluid (CSF) egress, the cerebellar hemispheres were allowed to relax with the aid of gravity. No fixed retractors were used during the procedure. The VA was identified intracranially and dissected above the origin of PICA proximally down to its dural cuff. The proximal dentate ligaments were also transected to facilitate improved mobilization of the brainstem. A marked color change of the VA wall consistent with atheromatous plaque was clearly identifiable proximal to the PICA origin, at the level of the twelfth nerve. Indocyanine green (ICG) angiography confirmed limited flow distal to the site of the plaque.

The dural cuff around the VA was then opened, and the entire circumference of the VA was freed from the dura. The remainder of the V3 segment along the sulcus arteriosus of the posterior C1 arch was skeletonized for proximal control, and venous bleeding was controlled with gentle tamponade and surgifoam. A posterior meningeal branch immediately proximal to the dural cuff was identified and clipped (later transected) for use in back-bleeding and assessment of patency. The occipital artery was deemed to be very small and of poor quality in relation to the VA. Therefore, we elected intraoperatively to proceed with arteriotomy and endarterectomy, reserving the occipital (OA)-to-PICA bypass option only in case a salvage procedure would be required.

Prior to endarterectomy, the field was irrigated until clear; systemic heparin was administered, and anesthesia was directed to achieve burst suppression with propofol for cerebral protection. Distal and proximal clips were applied, a linear arteriotomy was made, and then extended with Pott's scissors. Endarterectomy was performed without complication in the standard fashion with a Penfield 6 microdissector and microforceps [Figure 2]. The plaque was firm, hard, and well-organized, with only a small focal area of ulceration. It tailed out proximally
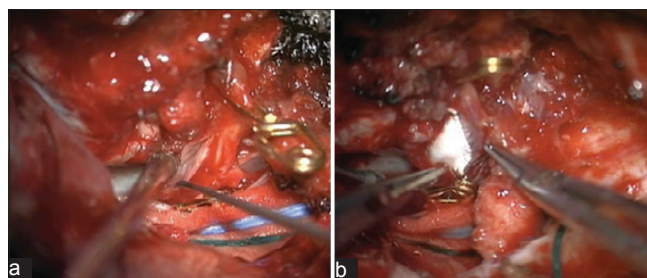

Figure 2: Intraoperative photographs displaying (a) completion of the endarterectomy using micro-forceps and 6-Penfield microdissector and (b) completion of the nonautologous patch angioplasty. Used with permission from Barrow Neurological Institute

and distally relatively smoothly. No evidence of arterial dissection was observed. The lumen was irrigated with heparinized saline, and the arteriotomy was closed with 2 limbs of running 10-0 prolene suture. Following proximal clip removal, minimal bleeding at the loose suture-line was apparent. ICG administration revealed patency to the level of the posterior meningeal branch, but luminal obstruction was consistent with thrombus formation at the site of the endarterectomy. The suture line was re-opened, thrombectomy performed, perfusion briefly re-established, and definitive closure was reattempted. Repeat ICG revealed similar findings to the previous. Systemic hypertension was maintained throughout the exposure, augmented during clip occlusion, and slightly relaxed following completion of the endarterectomy. Burst suppression was maintained throughout the procedure.

Before foregoing primary closure and converting to bypass, we elected to proceed with one more attempt to (i) reinspect the lumen for evidence of iatrogenic dissection and/or subintimal flap, (ii) trim the arteriotomy walls for any residual thrombogenic debris (e.g. intimal tags) at the arteriotomy line, and (iii) to attempt patch angioplasty given some concern for iatrogenic stenosis during the first two attempts at primary closure. Given concern about the ischemia time, the decision was made not to harvest saphenous vein. A Gore-Tex (W. L. Gore and Associates, Flagstaff, AZ) patch was trimmed and sutured in place with 9-0 prolene, accommodating a slight dilation at the site of the arteriotomy. Frequent and repeated back-bleeding from both the proximal and distal ends of the VA and brisk irrigation with heparinized saline at the anastomosis line was performed at 2-minute intervals during closure to prevent thrombosis [Figure 2]. Backbleeding was relatively brisk from the proximal side, implying some collateral, but the stump pressure was not measured. Following clip removal, patency was demonstrated by suture line bleeding prior to final ligature, distal pulsations, final ICG angiography, and confirmed with Doppler ultrasound. There were no changes in SSEPs throughout the entirety of the procedure.

A final bit of dural sealant (DuraSeal, Covidien, Dublin, Ireland) was placed over the suture lines, and the dura was closed with running 5-0 prolene. As watertight closure at the dural cuff of the VA was not feasible, the dural defect was repaired with a combination of interrupted sutures followed by an onlay patch (Duragen, Integra, Plainsboro, NJ) and additional sealant. The cranial bone flap was replaced with titanium mini-plates, and the wound was closed in the standard layered fashion.

Postoperative course

The patient again awoke without new neurological deficit. Postoperative day one angiogram revealed wide patency at the right VA origin and endarterectomy site. Surveillance MRI revealed new punctate foci of restricted diffusion in the left cerebellar hemisphere and subinsular region. Clopidogrel was resumed and aspirin was continued throughout the perioperative period following the satisfactory MRI. His lumbar drainage catheter, blood pressure parameters, and mobilization restrictions were weaned over the next 3 days. The patient was ultimately discharged to a neurorehabilitation facility at 5 days postoperatively, and returned to work 3 weeks later.

Ten-month follow-up angiography revealed satisfactory posterior circulation perfusion, wide patency at the treated VA segment, and mild (non flow-limiting) in-stent restenosis at the right VA origin [Figure 1]. Aspirin and clopidogrel sensitivity assays were performed, and the patient was transitioned to aspirin therapy alone. At 15-month clinical follow-up, he remained employed, functionally independent without restriction, and neurologically asymptomatic without recurrent transient ischemic attack (TIA) or deficit. Repeat surveillance angiography is planned at 2 years postintervention.

DISCUSSION

Symptomatic intracranial VA (V4 segment) thrombo-occlusive disease, its natural history, and associated treatment strategies have not been as rigorously studied in comparison to stroke phenomena originating from the internal carotid artery.[6,8,10,22,29] The Warfarin-Aspirin Symptomatic Intracranial Disease study calculated a corresponding territory stroke rate of 7.8 events per 100 patient-years despite medical treatment among 31 patients enrolled with symptomatic 50-99% unilateral (26/31) or bilateral (5/31) V4 stenosis.[27] Muller-Kuppers *et al*. reviewed 75 patients from the New England Medical Center Posterior Circulation Registry, and reported that 19% of patients presented with focal (PICA or medullary territory) hypoperfusion symptoms, 32% with distal embolism, and 52% with nonfocal diffuse hypoperfusion related to their V4 disease.[25] Among 42 patients with bilateral V4 stenosis later culled from the same registry, 43% of cases presented with stroke after TIA (s), 12% suffered stroke-related death within the follow-up period (mean 31.4 months), and only 74% maintained a favorable outcome at last follow-up (modified Rankin Scale ≤ 2).[32]

Surgical interventions for patients refractory to medical therapy consist principally of open arterial bypass or PTA and stent placement. Posterior fossa bypass procedures are technically demanding with significant risk of morbidity.[5,14] Low-flow options in the setting of vertebrobasilar insufficiency include OA-to-PICA and superficial temporal artery (STA)-to-superior cerebellar artery (SCA) (or STA-to-PCA) bypasses for proximal and

distal vertebrobasilar stenosis, respectively.[15] OA-to-AICA bypass is less commonly employed in our practice, and PICA-to-PICA anastomosis may also be considered in the setting of unilateral proximal VA or PICA origin disease, which did not apply in this case. Hopkins *et al.* reported resolution of ischemic symptoms in 25 of 25 cases treated with OA-to-PICA (in one case, VA-to-PICA) bypass for stenosis proximal to the PICA origin, and neurological improvement with graft patency in 14 of 18 OA-to-STA/PCA bypass procedures for distal vertebrobasilar disease.[15] The same lead author subsequently published a literature review of OA-to-PICA and STA-to-SCA/PCA bypass procedures citing cumulative graft patency, morbidity, and mortality ranges ranging from 79% to 91%, 22% to 55%, and 12% to 14%, respectively.[14]

Endovascular alternatives are well-reported,[7,13,18,20,21,24,26,30,33,34] less invasive, associated with comparatively low peri-procedural stroke rates according to a recent meta-analysis,[10] and likely hold considerable promise for future innovation. Although technically feasible,[34] intracranial stent placement has also been associated with high rates of symptomatic in-stent restenosis,[12,34] high periprocedural complication rates in selected early experiences specific to the vertebrobasilar circulation,[12,19,30] may be limited occasionally in access by proximal atherosclerotic disease, and has not been convincingly or rigorously shown as superior to medical therapy in some recent publications.[9,11]

Lu *et al.* reported technical success in 24 patients who underwent both intracranial VA and VA origin stenting with a drug-eluting stent and had less than 30% residual stenosis.[21] They documented no stroke or death within 30 days, and only two patients with in-stent restenosis.[21] In a series of eight patients, Rasumussen *et al.* showed a successful outcome of stenting for intracranial VA/vertebrobasilar disease with 7-28% residual stenosis.[30] One patient died the night of the procedure from a large subarachnoid hemorrhage, two developed groin hematomas, one suffered congestive heart failure, and one had a transient encephalopathy.[30] In another study of 16 patients, 2 underwent V4 angioplasty and 2 underwent angioplasty and stenting. Two of the four patients experienced complications related to vessel dissection and one of the four patients died from a subarachnoid hemorrhage following vessel perforation.[13] While the success and feasibility of VA angioplasty with or without stenting has been demonstrated, potential complications related to this treatment or not insignificant and further reports are eagerly awaited.

V4EA is an attractive alternative to other open revascularization strategies, particularly when endovascular management strategies have failed.

Several inherent advantages of V4EA compared with bypass options are worth noting: V4EA preserves the native VA and tributaries, restores near-physiologic flow dynamics, and can typically be salvaged by bypass or thrombectomy[2] if necessitated by technical failure or thrombosis.

Our complete list of indications for V4EA are listed in Table 1, and are generated in part from prior published experience with the procedure [Table 2]. In the current case, we favored V4EA due to the following:

- Feasible, but suboptimal caliber of the donor occipital artery vessel
- Focal nature of the VA plaque. Previous authors have recommended that the endarterectomy be useful in cases of focal plaque.[2-4] This is intuitive as endarterectomy for long segment plaques involves a large arteriotomy and can potentially put perforators to the brainstem at risk during clamping
- Accessibility of the VA plaque (proximal to the PICA origin). In a series of four cases, Ausman *et al.* reported that two patients with endarterectomies at the level or distal to PICA experienced infarction or VA sacrifice.[4] The more proximal the lesion intradurally, the more relatively surgically accessible the exposure of the lesion becomes from a far lateral approach. As in the current case, we additionally consider preexisting occlusion of PICA as an acceptable preoperative angiographic finding
- Relative freedom of the remainder of the right VA from significant untreated stenosis
- Salvage opportunities could have been employed as a last resort either via an STA-SCA or OA-to-PICA bypass.[15] We chose to expose the occipital artery in this case, providing the opportunity to use it if in the event of an unsuccessful endarterectomy. Given the significant morbidity of the procedure,[14] bypass was reserved only as a bailout strategy
- Some authors reported that V4EA may be indicated in cases of hemodynamic or embolic etiologies[1-3] while others have also find it or suggested

Table 1: Authors' Indications

Refractory to best medical therapy
Symptoms attributable to hemodynamic or embolic etiologies
Compromised collateral circulation (e.g., contralateral VA)
Not amenable to endovascular intervention
High-grade focal stenosis
Lesion proximal to PICA origin or preexisting PICA occlusion with lesion at the level of PICA
Remainder of ipsilateral posterior circulation patent without untreated stenosis
Distal and proximal control accessible via far-lateral approach
Bypass options available for bailout

VA: Vertebral artery, PICA: Posterior inferior cerebellar artery

Table 2: Previously reported cases of vertebrobasilar ischemia treated via V4EA

Series	Patients	Plaque location	Approach (positioning)	Indication/contralateral circulation	Follow up	Outcome
Allen et al., 1981[1]	2	Between PICA and AICA origins	C1 laminectomy and suboccipital craniectomy (sitting)	Refractory VB TIAs/ Patent	~1 and 4 months	Favorable outcome in 1 of 2 cases (no arteriotomy made due to extensive plaque in 1 case)
Ausman et al., 1982[3]*	1	60% stenosis at V3, 40% V4 stenosis	Suboccipital craniectomy, unroofed C1 foramen transversarium (three-quarter prone)	Refractory VB TIAs/VA ends in PICA	7 months	Improved POD #1 CSF leak controlled with lumbar diversion
Ausman et al., 1990[4]*	6	V4 (4 of 6 proximal to PICA origin)	Suboccipital craniectomy (authors preferred three-quarter prone)	Refractory VB TIAs/ Ends in PICA, occluded, stenotic, or hypoplastic	NR (3 patients) 1 perioperative death. 5 and 11 months follow up in remainder	2 of the 4 VEAs proximal to PICA-patent and improved at follow up. 1 patient with VEA at PICA- infarct and death. 1 VEA distal to PICA-VA sacrifice and Wallenburg's
Hopkins et al., 1987[15]†	2	V4, 90 and 95% stenosis proximal to PICA	C1 laminectomy and unilateral craniotomy (lateral decubitus or park-bench position)	Refractory VB TIAs/ Compromised	10 and 16 months	Both asymptomatic and patent LP-Shunt for hydrocephalus
Anson and Spetzler, 1993[2]	8	V4 (7 of 8 proximal to PICA origin)	Authors preferred standard far-lateral craniotomy with posterolateral 1/3 condylar resection (park-bench, 5 patients) versus suboccipital craniotomy and C1 laminectomy suboptimal (3 patients)	Refractory VB TIAs, some with MRI evidence of completed infarct but without fixed neurological deficits/ ends in PICA, occluded, stenotic, hypoplastic	Mean 16 months	5 of 8 patent and asymptomatic immediately postoperative. 2 required postoperative thrombectomy. 7 of 8 improved at last follow up. 12.5% "major complication rate." 3 required LP-shunt for HCP
Morgan et al., 1994[23]	1	V4 stenosis (2 cm plaque length)	C1 hemilaminectomy and suboccipital craniectomy and medial 1/3 condylar resection (prone), saphenous vein patch angioplasty	Refractory VB TIAs with "submaximal L PICA infarct"/V4 stenosis with R PICA territory filled by R AICA	2 months	POD #1 occlusion treated with urokinase, asymptomatic cerebellar stroke

*The series reported by Ausman et al. (1990) also contains the original case reported by the lead author in 1982. †Approach not specifically stated for all cases. PICA: Posterior inferior cerebellar artery, AICA: Anterior inferior cerebellar artery, VB: Vertebrobasilar, TIA: Transient ischemic attack, POD: Postoperative day, CSF: Cerebrospinal fluid, NR: Not reported, VEA: Vertebral artery endarterectomy, VA: Vertebral artery, LP: Lumbar-peritoneal, HCP: Hydrocephalus

that it would be useful especially for patients with (orthostatic) hypoperfusion phenomenon.[23] We consider either indication to be satisfactory.

The central tenets of our technique are similar to those described by previous authors,[1-4,15,23] but with important minor modifications. Circumferential mobilization of the VA from its dural cuff is useful in selected cases to allow for ideal proximal control, inspection of proximally located stenosis adjacent to the V3/4 junction, to improve working room, and to permit for extension of the arteriotomy (if dictated by a plaque with an unexpected proximal extension). V4EA proceeds in a similar fashion to a standard internal carotid artery endarterectomy, although we exercise additional caution in aggressively removing tags of media and also exercise caution in following distal/proximal atheroma beyond the arteriotomy. As previously highlighted by Ausman et al.,[4] the medial and adventitial layers of the VA thin significantly following dural penetration.[31,35]

In our case, however, appropriate trimming of thrombogenic medial debris at the arteriotomy line

and the small size of the VA necessitated an expansile patch angioplasty due to luminal constriction. Morgan et al.[23] previously reported the only other V4EA with patch angioplasty, in that case, using saphenous vein graft. A nonautologous vascular patch was chosen in the present case due to immediate availability. This represented a viable solution to intraoperative thrombosis at the suture line in this instance, and proved durable without change at 10-month follow-up. There is, however, insufficient evidence to accurately predict the long-term natural history of recurrent vertebrobasilar ischemia in patients treated via V4EA (with or without patch angioplasty). The current patient remains on low-dose aspirin, and regular surveillance angiography is planned.

We agree with Anson and Spetzler that exposure via a far-lateral craniotomy plus C1 laminectomy is preferable to suboccipital access.[2] Advantages of this approach include gravity retraction of the cerebellum with appropriate lesion-side-up positioning, improved lateral-to-medial trajectory to the proximal VA, and

access to the condylar region. Mobilization of the VA at its dural cuff and proximally along the C1 sulcus arteriosus is also readily feasible if required. We additionally consider MR angiography or CT angiography a necessary preoperative adjunct to conventional catheter-based angiography, as the former two modalities permit detailed inspection of vascular-bony relationships at the skull base. As in the present case, the extent of lateral occipital bone removal and condylar drilling can be appropriately tailored to facilitate sufficient exposure.

We find the use of ICG in this case to be an excellent adjunct to micro-Doppler ultrasound to assess the patency of the vessel after endarterectomy, and find it to be a time-saving step over intraoperative digital subtraction angiography (DSA). DSA carries its own stroke risk, significantly increases operative time, and cannot localize the anatomic segment of stenosis directly in the operative field in real time under the operating microscope. The arteriotomy may then be appropriately limited to directly cover only the region of focal pathology, and the repair assessed under direction vision. We have previously found ICG useful in cases of bypass (unpublished data) to interrogate graft patency and have also used it in cases of aneurysms, arteriovenous malformations, and dural fistulae in lieu of or as an adjunct to formal angiography.[16,17,28]

CONCLUSION

The natural history of patients with symptomatic vertebrobasilar ischemic symptoms due to chronic bilateral VA occlusive disease is progressive, and poses significant challenges when refractory to medical therapy. Surgical treatment options depend largely on location and characteristics of the atheroma(s), and generally include PTA with or without stent placement, posterior circulation revascularization (bypass), extracranial VA reconstruction, or VA endarterectomy. We describe a nonautologous patch angioplasty and V4EA in a patient refractory to other treatments. An excellent outcome was demonstrated at 15-month follow-up.

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