The Aberrant Vertebral Artery With Aortic Arch Pathology

A discussion of the indications for revascularization and technical tips for preservation.

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Aberrant origin of the vertebral artery (VA) is an uncommon anatomic variant that usually has no clinical significance, except when treating cerebrovascular or aortic arch pathology. When the VA origin is located in the intended area of replacement or zone of endovascular coverage, critical steps must be taken to preserve its perfusion in certain clinical situations. This article discusses indications and techniques used to preserve the aberrant VA associated with aortic arch pathology.

EMBRYONIC AND ANATOMIC CONSIDERATIONS

The aorta develops during the third week of gestation from a dorsal and ventral aortic bud that gives rise to six paired branchial arch arteries. The right dorsal aortic root and right ductus arteriosus regress from the normal left aortic arch, and any deviation results in a variation in arch anatomy. The most common variants include bovine anatomy with a common brachiocephalic trunk, coarctation of the aorta, and left VA (LVA) arising from the arch, which occur in 10% to 22%, 5% to 7%, and 2% to 6% of the population, respectively.[1]

According to Yuan:

> The VA develops between 33 and 55 days during intrauterine life and is normally formed by the longitudinal anastomoses linking the seven cervical intersegmental arteries. The cervical intersegmental arteries obliterate soon after, except the seventh intersegmental artery, which develops into the subclavian artery involving the origin of the VA. In some cases, the anastomosis between the sixth and seventh intersegmental arteries does not develop on the left side and the sixth intersegmental artery remains, in which case the LVA arises from the aortic arch between the left common carotid artery and left subclavian artery (LSA).[1]

Yuan reviewed 1,286 cases of aberrant VA origins and found that most involve a single LVA (84.7%) and originate from the aortic arch (97.4%) between the left carotid artery (LCA) and LSA (81.9%). Other anatomic variations included aberrant right VA (RVA) in 12.5%, dual origin in 4%, and extra-arch origins, which mostly were on the right side (32.4%), coming off the right carotid artery (85%).[1]

Meila et al retrospectively reviewed the CTAs of 539 patients and found that most (94.2%) LVAs originated from the LSA, and in nearly all cases, the LVAs entered the foramen transversarium at C6. In addition, 6.3% of LVAs originated from the aortic arch and entered the foramen transversarium either at C4, C5, or C7 but never at C6.[2]

INDICATIONS FOR PRESERVATION

Although there are no clear recommendations on when to revascularize the VA, posterior circulation strokes or vertebrobasilar insufficiency have been reported with coverage of the LSA during thoracic endovascular aortic repair (TEVAR), usually due to low flow or occlusion of the VA.[3] Therefore, indications to preserve perfusion of the VA can be extrapolated from published indications to preserve perfusion to the LSA. Woo et al described indications for revascularization and techniques to revascularize the LSA and preserve the LVA during TEVAR. Indications for revasculariza-

TABLE 1. INDICATIONS FOR VERTEBRAL ARTERY REVASCULARIZATION
• Dominant vertebral artery
• Incomplete circle of Willis
• Diseased contralateral vertebral artery
• Extensive aortic coverage
• Inability to evaluate the circle of Willis prior to intervention

tion include a dominant LVA (present in 50% of the population); incomplete circle of Willis, especially the posterior circulation (found in more than 50%); diseased or atretic RVA; or inability to evaluate the anatomy of the vertebrobasilar circulation prior to intervention.[4] Because some of the blood supply to the spine arises from the proximal VA, extensive coverage of the aorta is also considered an indication for preservation to prevent spinal cord ischemia. In cases of aberrant LVA with planned aortic coverage, concomitant LSA revascularization is usually indicated, especially with the absence of collateralization. Other than VA revascularization for atherosclerotic VA disease published in the neurosurgery literature,[5] most evidence for VA revascularization in the setting of thoracic aorta pathology is in the form of case reports[6-10] or as part of recommendations to expand the zone coverage proximally in review articles.[11] Table 1 summarizes the indications for VA revascularization.

TECHNICAL CONSIDERATIONS: CASE EXAMPLES

The following clinical scenarios describe VA transposition to maintain flow to the posterior circulation prior to intervention.

Case 1

A 76-year-old woman (former smoker) with hypertension, hyperlipidemia, chronic obstructive pulmonary disease, and a history of a cerebrovascular accident with residual dysarthria was referred to our vascular clinic after a thoracic aortic aneurysm (TAA) was found on workup for chest and lower back pain after a motor vehicle accident. A CTA revealed an irregularly shaped TAA with a maximum diameter of 6 cm involving the origin of the LSA, as well as a dominant aberrant LVA originating directly off the aortic arch between the LCA and LSA (Figure 1). A staged repair was planned that included a left carotid-subclavian bypass and LVA transposition, followed by TEVAR and LSA embolization the following day.

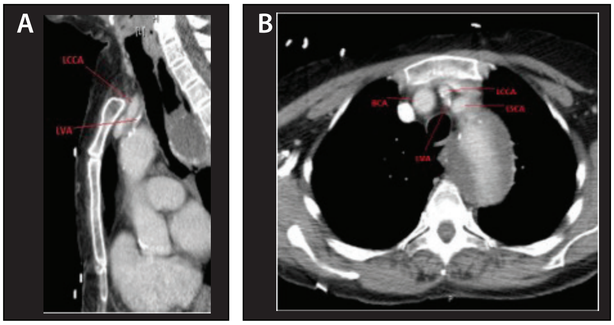

Figure 1. Sagittal view of the LVA originating from the aortic arch between the LCA and LSA (A). Axial view showing part of the aneurysmal aorta (B).

After establishing a lumbar drain and neuromonitoring, the LSA and LCA were controlled through a supraclavicular incision in their usual anatomic locations. The LVA was dissected posterior to the LCA and as proximal as possible to allow transposition. After systemic heparinization, the LVA was transected and ligated as proximal as possible. Special attention was made to avoid losing control of the proximal stump of the LVA in the chest. The LVA was then transposed, avoiding kinks or twists, and anastomosed to the posterior surface of the LCA in an end-to-side configuration using 7-0 Prolene suture (Ethicon, a Johnson & Johnson company). Blood flow was reestablished to the LVA (Figure 2). The carotid-subclavian bypass was then performed using a 6-mm Dacron graft, which was tunneled in a retroscalene fat pad position.

Staged endovascular repair of the TAA with zone 2 coverage was then performed using a 40- X 36- X 150-mm Valiant Captivia (Medtronic) proximal main piece. The proximal LSA was successfully embolized using five 12-mm Nester coils (Cook Medical) to

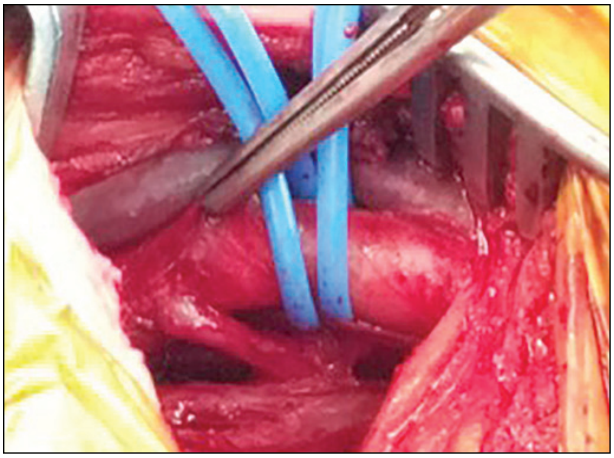

Figure 2. LVA transposed to the posterior surface of the left common carotid artery.

Figure 3. CTA with three-dimensional reconstruction of the LVA transposition at 1-year follow-up.

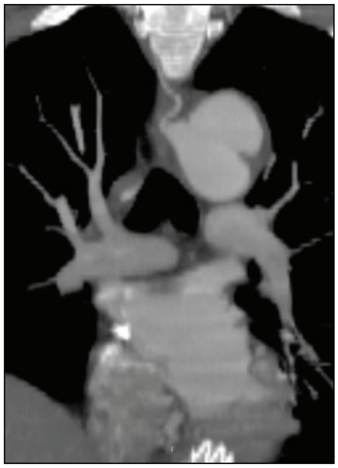

Figure 4. Coronal CTA showing the aberrant retroesophageal RVA coming off a diseased thoracic aorta.

avoid a type II endoleak. A completion angiogram showed a patent left common carotid artery and LVA and carotid-subclavian bypass with no endoleak.

Postoperatively, the patient was neurologically intact, and the lumbar drain was removed on postoperative day 2. The remainder of the patient's hospital course was uneventful, and the patient was discharged in a stable condition on postoperative day 5. Follow-up at 1 year demonstrated a shrinking aneurysm sac with no endoleak and a patent LVA as well as LCA-LSA bypass (Figure 3).

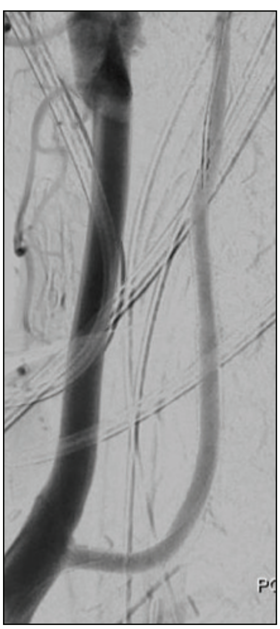

Figure 5. Intraoperative selective angiogram of the RVA after transposition showing a patent artery with normal flow.

Case 2

The case patient had a dominant aberrant retroesophageal RVA associated with an aortic arch aneurysm that required zone 2 coverage. The patient had a previous infrarenal EVAR, which is another indication for preservation of the VA. The origin of this patient's VA was from a diseased descending thoracic aorta (Figure 4). A staged LCA-LSA bypass was planned, followed by RVA transposition and TEVAR. The VA transposition was performed using a similar technique as previously mentioned in case 1, but from a right supraclavicular approach. Because preoperative imaging demonstrated that the proximal VA was diseased, the transposition and TEVAR was planned as a single-stage procedure to protect from proximal stump blowout, and the VA was angiographically evaluated after transposition (Figure 5). The patient recovered with no neurologic sequelae and was discharged on postoperative day 5. Follow-up at 6 months confirmed patent transposition.

CONCLUSION

To maintain posterior cerebral perfusion, VA artery transposition is indicated in certain clinical situations prior to covering or ligating its origin as part of treatment of aortic arch pathology. Transposition of this vessel to the adjacent carotid artery is technically feasible and safe. ■

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