Selective coverage of the left subclavian artery without revascularization in patients with bilateral patent vertebrobasilar junctions during thoracic endovascular aortic repair

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Objective: **The primary purpose of the current study was to evaluate the safety and effectiveness of selective coverage of the left subclavian artery (LSCA) without revascularization during thoracic endovascular aortic repair (TEVAR) in patients with bilateral patent vertebrobasilar junctions. The secondary purpose was to assess morphologic change of the vertebral artery (VA) after the procedure.**

Methods: **Among 126 patients who underwent TEVAR between 2006 and 2011, 29 patients requiring LSCA coverage without preemptive revascularization were retrospectively analyzed in this study. The patients were a mean age of 63.1 years (range, 45-84 years). The mean follow-up period was 19.9 months (range, 1-63 months). Bilateral patent vertebrobasilar junctions were evaluated by contrast-enhanced computed tomography (CT), time-of flight magnetic resonance angiography, or conventional angiography. Neurologic complications, such as spinal cord ischemia (SCI) or cerebrovascular accidents, were analyzed. Preprocedural and postprocedural changes in VAs were evaluated on follow-up contrast-enhanced CT.**

Results: **The overall 30-day mortality was 6.9% (2 of 29). None of the patients had SCI or a stroke of posterior circulation alone. Cerebrovascular accidents from embolic infarctions occurred in two patients (7.4%). Transient left arm ischemic symptoms were present in five patients (18.5%), but none required secondary interventions. Delayed development of type I endoleak occurred due to stent deformity in one patient, who underwent surgery. One patient required reintervention after the 10-month follow-up contrast-enhanced CT showed a pseudoaneurysm had developed at the distal margin of the previously placed stent graft. Hypertrophy of the right VA after TEVAR was seen in seven of 27 patients (25.9%); two patients showed bilateral hypertrophy of VAs.**

Conclusions: **LSCA coverage without revascularization can be safely performed during TEVAR in patients with bilateral patent vertebrobasilar junctions. Hypertrophy of the right VA was noted in 25.9% of patients after LSCA coverage. (J Vasc Surg 2013;57:1311-6.)**

Thoracic endovascular aortic repair (TEVAR) has emerged as a promising therapeutic alternative to conventional open aortic replacement; however, it requires suitable proximal and distal landing zones for stent graft anchoring.[1-3] Coverage of the left subclavian artery (LSCA) during TEVAR is performed in up to 40% of procedures.[4,5] The safety and effectiveness of revascularization of the LSCA before coverage are still controversial. Some authors suggest that LSCA coverage without revascularization is significantly related to devastating complications, such as spinal cord ischemia (SCI) or compromised hindbrain circulation and posterior watershed infarction. Meanwhile, other studies have revealed that LSCA coverage without prior transposition or bypass of the LSCA is generally safe, but the bypass or LSCA transposition procedures themselves are burdensome in some patients.[6-9]

The primary purpose of the current study was to evaluate the safety and effectiveness of selective coverage of the LSCA without revascularization during TEVAR in patients with bilateral patent vertebrobasilar junctions. Considering few reports have described computed tomography (CT) findings of the vertebral artery (VA) after coverage of the LSCA, the secondary purpose was to assess morphologic change of the VA after the procedure.

METHODS

The Institutional Review Board approved the entire study. Of the 126 patients who underwent TEVAR between 2006 and 2011 at our institution, 38 with LSCA coverage were retrospectively enrolled. Nine of

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Author conflict of interest: none.

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The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.
0741-5214/$36.00

http://dx.doi.org/10.1016/j.jvs.2012.10.110

these patients were excluded because they underwent LSCA revascularization before TEVAR.

Preoperative analysis. Bilateral patent vertebrobasilar junctions in all patients were evaluated by contrast-enhanced CT, time-of-flight magnetic resonance angiography (TOF MRA) or by conventional angiography when the circle of Willis was not included in the contrast-enhanced CT.

CT examinations were performed from the vertex to the level of the common femoral artery with a slice thickness of 2 mm. VAs were measured by contrast-enhanced CT at the maximum diameter in the intradural segment proximal to the anastomosis with the basilar artery. When contrast-enhanced CT did not include the circle of Willis, only the foraminal segments of VAs in the transverse processes were measured. Although there is no clear definition of VA hypoplasia, it was defined in this study as a maximum lumen diameter of <2 mm on contrast-enhanced CT or TOF MRA. With conventional angiography, hypoplasia was regarded as a VA thickness less than half that of the contralateral VA. Patent vertebrobasilar junctions were categorized into five types depending on the dominance and hypoplasia of the VA[1]: symmetric, left dominant, right hypoplasia, right dominant, and left hypoplasia (Fig 1).

Stent graft procedure. Of the 29 patients, 16 (55.2%) underwent the stent graft procedure with only local anesthesia, and 13 (44.8%) had intravenous anesthesia as well as local anesthesia. Bilateral common femoral artery access was obtained using the preclosure technique with two Perclose ProGlide devices (Abbott Vascular, Santa Clara, Calif). Peri-interventional heparin (5000 IU) was given intravenously. A conduit for access was not required.

Amplatzer vascular plugs (AGA Medical Corp, Plymouth, Minn) were used to prevent type II endoleaks in patients with direct LSCA involvement of the aneurysm or aneurysms with wide necks. SEAL thoracic (S&G Biotech Inc, Seongnam, Korea; n = 17) or Valiant Captivia (Medtronic, Minneapolis, Minn; n = 12) stent grafts with diameters of 26 to 46 mm and lengths of 80 to 219 mm were implanted.

Blood pressure was not brought down during deployment of the stent graft. In the event a type I endoleak occurred, a large, compliant, low-pressure Coda balloon (Cook Inc, Bloomington, Ind) inflated with a hand-held syringe was used in patients with an aortic aneurysm, but not in patients with dissection after the blood pressure was brought down using nitroprusside or right ventricular pacing. When the stent graft coverage of the descending thoracic aorta was >20 cm in length, cerebrospinal fluid drainage was performed before TEVAR.

Complications and follow-up assessment. Data on neurologic complications, such as SCI or cerebrovascular accidents (CVA), and non-neurologic complications after TEVAR were gathered from medical records. To be included in our analysis, CVA and SCI had to be newly diagnosed by a neurologist with symptoms lasting >72 hours. Contrast-enhanced CT was obtained ≤1 month after the procedure and annually thereafter if there were no remarkable abnormalities, such as immediate migration or endoleak, that would require repeat intervention or closer observation. If contrast-enhanced CT revealed type I or II endoleak without other associated abnormalities at the 1-month follow-up, 3- and 6-month follow-up CT examinations were performed.

Preprocedural and postprocedural changes in VA maximum diameter were evaluated at the intradural or foraminal segments on follow-up contrast-enhanced CT. VA hypertrophy was defined as a VA of at least 1 mm larger than it was before the procedure.

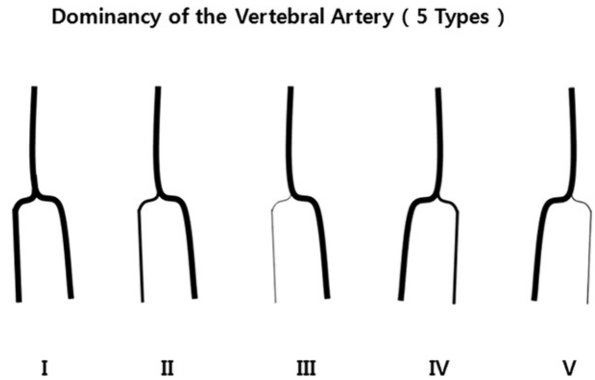

Fig 1. Vertebral artery (VA) dominance: *I,* symmetric; *II,* left dominant; *III,* hypoplasia of the right VA; *IV,* right dominant; and *V,* hypoplasia of the left VA.

RESULTS

Patient demographics and comorbidities are listed in Table I. The study enrolled 29 of 38 patients with LSCA coverage without preemptive revascularization. Among nine patients excluded, six underwent revascularization of LSCA due to a previous coronary bypass graft from the left internal mammary artery. Of the remaining three patients (9.4%), two demonstrated no communications between the left and the right VA and one patient had multifocal atherosclerotic calcifications with stenosis in the intradural segment and the origin of the right VA, resulting in LSCA bypass surgery.

Among the 29 patients (19 men, 10 women; mean age, 63.1; range, 45-84 years), 19 received stent grafts for thoracic aortic aneurysm and 10 for aortic dissection. The mean follow-up period was 19.9 months (range, 1-63 months). Emergency procedures were performed in two patients (6.9%), of which one had traumatic aortic rupture and the other had aortic dissection with dynamic obstruction of abdominal branch vessel. The remaining 27 cases were elective (93.1%). The 10 dissection cases were chronic. Bilateral patent vertebrobasilar junctions were confirmed by conventional angiography in 14, contrast-enhanced CT in 13, or TOF MRA in two. Patent vertebrobasilar junctions were categorized as symmetric VAs (n = 12),

Table I. Patient demographics (N = 29)

Variable	*Median ± SD or No. (%)*
Age, years	63.1 ± 14.6
Sex	
Men	19 (65.5)
Women	10 (34.5)
Emergency	2 (6.9)
Elective	27 (93.1)
Traumatic	2 (6.9)
Nontraumatic	27 (93.1)
Comorbidities	
Hypertension	24 (82.8)
Diabetes	3 (10.3)
Coronary artery disease	5 (17.2)
Stroke	4 (13.8)
End-stage renal disease	2 (6.9)
Peripheral vascular disease	1 (3.4)
Previous aortic operation	2 (6.9)
Abdominal aortic aneurysm	1 (3.4)

SD, Standard deviation.

left dominant (n = 7), right dominant (n = 6), left VA hypoplasia (n = 2), or right VA hypoplasia (n = 2).

There were no technical failures of stent graft placement during TEVAR. Vascular plugs for occlusion of the LSCA were used in 15 patients, and LSCA coverage with the stent graft alone was performed in the remaining 14. Four of 29 patients (13.8%) underwent cerebrospinal fluid drainage before TEVAR. Two patients underwent carotid-to-carotid bypass surgery to ensure suitable proximal landing zones. One patient underwent simultaneous abdominal and thoracic aortic aneurysm treatment with a stent graft and was successfully treated without neurologic complications.

Complications. The overall 30-day mortality was 6.9% (2 of 29). One patient died of recurrent massive hemoptysis due to chronic destructive lung disease. The other patient was treated emergently due to traumatic aortic rupture but died of multiorgan failure 7 days after stent graft placement. No deaths or complications were related to aneurysm rupture.

None of the patients had SCI or stroke of the posterior circulation alone. CVA occurred in two patients (7.4%). The first patient was a 69-year-old man who experienced right-sided weakness and dysarthria after the procedure. Magnetic resonance imaging (MRI) revealed multifocal infarctions in the pons, cerebellum, right temporal occipital lobe, and the left parietal lobe. The second patient was a 51-year-old man who developed a medial gaze palsy after the procedure. MRI demonstrated focal infarctions in the right frontal lobe and the left cerebellum. The medial gaze palsy completely resolved after 1 month. Both patients had symmetric VAs.

Left arm symptoms were noted in five patients (18.5%), including arm claudication (n = 3), tingling sensation upon elevation of the left arm (n = 2), and pain (n = 1), all of which were tolerable at rest, spontaneously resolved at median of 5 months (range, 1-12 months), and none of which required secondary interventions.

A postimplantation syndrome, including fever, was noted in seven patients (25.9%), and they were subsequently managed with conservative treatment. A hematoma at the puncture site was noted in one patient (3.7%).

Follow-up contrast-enhanced CT. Type I endoleaks after stent graft placement were noted in five patients (18.5%), including one with delayed development at 4.5 years due to stent deformity. Four of these patients experienced spontaneous resolution at a median follow-up of 5 months (range, 1-12 months). In one patient, we initially found no endoleak after the procedure, but delayed development of a type I endoleak occurred due to stent deformity with increasing aneurysmal sac size, and the patient subsequently underwent open surgery without LSCA revascularization. One patient required reintervention after the 10-month follow-up contrast-enhanced CT showed pseudoaneurysm had developed at the distal margin of the previously placed stent graft on the descending thoracic aorta. A type II endoleak from the LSCA was encountered in one patient (3.4%) who underwent LSCA coverage without a plug, which closed spontaneously.

Change of the VA on follow-up CT scan. Serial changes of the hypertrophied right VA diameter after TEVAR are summarized in Table II. Seven of 27 patients (25.9%) presented with hypertrophy of the right VAs after TEVAR, including four of 15 instances (26.6%) at the foraminal segment and three of 12 (25%) at the intradural segment (Fig 2). Two patients had bilateral VA hypertrophy. Mean diameter of VAs in patients with hypertrophy changed from 3.5 ± 0.9 to 4.7 ± 0.9 mm after the procedure. VAs were increased as fast as 1 month after TEVAR and grew up to 36 months. Aortographies revealed reversal of left VA flows immediately after TEVAR in all patients, with or without embolization of the LSCA using a plug.

DISCUSSION

The endovascular repair of thoracic aortic pathologies offers a minimally invasive alternative to open repair and is increasingly replacing the traditional open surgical repair of diseases of the descending thoracic aorta.[1,2] TEVAR can be complicated by the requirement of suitable proximal and distal landing zones for stable stent graft fixation. It has been suggested that 2 cm of normal aortic wall are required for an adequate and stable seal[10,11]; however, many aortic pathologies affect the area near the LSCA, limiting the proximal landing zone site without proximal vessel coverage. It has been reported that about 40% of LSCAs should be covered during TEVAR.[4,5]

There is controversy regarding the safety of LSCA coverage without revascularization before TEVAR because the process may alter the circulation of the vertebrobasilar system and upper extremity. A multicenter registry analysis by Buth et al[12] suggested that the incidence of paraplegia due to SCI and stroke was higher in patients requiring LSCA coverage (8.4%) than in patients who received prophylactic revascularization (0%). Other studies have

Table II. Serial changes of the hypertrophied right VA diameter after TEVAR

		VA diameter, mm						
			At follow-up month					
Segment	*Pt*	*Initial*	*1*	*3*	*6*	*12*	*24*	*36*
Foraminal (n = 4/15)	2	3.2	4	4.1	4.2	4.2	4.2	4.2
	4	3.8	4.2	...	...	4.8	...	...
	13	3.8	4.3	...	...	4.3	5.2	5.3
	17	4.8	5.2	...	...	5.2	5.5	5.8
Intradural (n = 3/12)	8	4.6	5.5	...	...	6.1	...	...
	11	1.7	...	3	3	3	...	...
	23	2.6	3.6	...	...	4	...	...

TEVAR, Thoracic endovascular aortic repair; *VA,* vertebral artery.

suggested the risk of stroke and paraplegia if the LSCA is covered without preceding or simultaneous revascularization.[13,14] A study by Tiesenhausen et al,[9] however, revealed no signs of malperfusion after LSCA coverage without revascularization. They noted that surgical treatments performed with bypass or LSCA transposition correlated with a mortality of 1.2% to 5%, thereby increasing the overall risk of TEVAR. Furthermore, whether routine revascularization results in a reduction in perioperative stroke complications is not clear.

Reported complication rates are inconsistent due to sample size limitations and heterogeneity in patient selection criteria. A 2009 consensus statement from the Society of Vascular Surgery described the "very low" quality of existing evidence regarding performance of LSCA revascularization in patients undergoing TEVAR.[15] According to a meta-analysis, revascularization of LSCA demonstrated no protective effect for CVA and had only a questionable protective role for SCI.[16] Owing to the lack of evidence about the potential role of LSCA revascularization, conservative patient selection is used to avoid unnecessary operations. Some authors propose prophylactic revascularization only in select cases of an aberrant right subclavian artery, previous myocardial revascularization with the left internal mammary artery, critically stenosed carotid or VA, occluded contralateral VA, functional compromise of the circle of Willis, an arteriovenous shunt for hemodialysis in the left arm, and in left-handed patients.[17,18]

Criado et al[10] described the general safety of LSCA coverage without revascularization, even without prior transposition or bypass; however, they recommended ascertaining the angiographic patency of the contralateral VA beforehand. Preoperative contrast-enhanced CT, MRI, or angiography may be helpful in selecting candidates for adjunct LSCA revascularization.[11]

We also evaluated VA anatomy in our study, specifically focusing on the communication between both VAs as they join to form the basilar artery. Currently, our contrast-enhanced CT protocols for aortic pathology cover the entire area, from the whole brain to the common femoral arteries, with a 2-mm slice thickness. This detail makes it unnecessary to do conventional angiography and enables us to quickly determine whether LSCA coverage without revascularization can be safely performed. In addition, we suggest that our study's findings are more applicable for patients with traumatic aortic pathology who require an emergency decision of stent graft placement because these patients often undergo LSCA coverage without revascularization, even with little information about their VA anatomy.

Our study enrolled 29 of 38 patients and excluded nine patients. Six underwent a previous coronary artery bypass graft from the left internal mammary artery, so we did not need to evaluate vertebral communications. Three of 32 patients (9.4%) needed bypass surgery of the LSCA; of these, two did not have patent vertebrobasilar junctions, and one had multifocal atherosclerotic calcifications with stenosis in the intradural segment and the origin of the right VA, with patent communication between the left and right VAs. Without a doubt, we believe that the entire right VA, as well as the vertebrobasilar junctions, should be patent in instances of LSCA coverage without revascularization.

Theoretically, the posterior communicating arteries may play a role in collateral supply as well when the left VA is occluded. In this study, however, we investigated vertebrobasilar junctions on axial images, on which the VAs were well visualized and patency was easier to evaluate, compared with the posterior communicating arteries. In addition, the VA is generally larger than the posterior communication artery and could contain more collateral supply than the posterior communication artery when the left VA is occluded.

We did not perform duplex ultrasound imaging to document the directionality of VA flow. However, all patients underwent aortographies immediately after TEVAR, which revealed reversal of left VA flows. Two patients in our study had stroke events after TEVAR, and MRI revealed multifocal infarcts in the anterior and posterior circulation territory. These strokes were likely due to atheroembolization from the aortic arch because the risk of embolic stroke during thoracic aortic stent graft placement is inherent due to manipulation of wires, catheters, and devices in the aortic arch. However, we would like to acknowledge that in multifocal infarctions in the anterior and posterior circulation, lesions in the posterior circulation might be related with simultaneous ischemia with embolic infarcts in anterior circulation.

Covering the LSCA can lead to type II endoleaks due to retrograde perfusion from the LSCA into the aneurysm sac or the false lumen of the dissection. This potential complication is a commonly cited justification for LSCA revascularization. We use vascular plugs to prevent type II endoleaks in patients with direct LSCA involvement of the aneurysm or aneurysms with wide necks. Only one of 14 patients in this study who underwent LSCA coverage without a plug presented with a type II endoleak, which resolved spontaneously. None of the patients treated with vascular plugs for LSCA coverage had type II endoleaks.

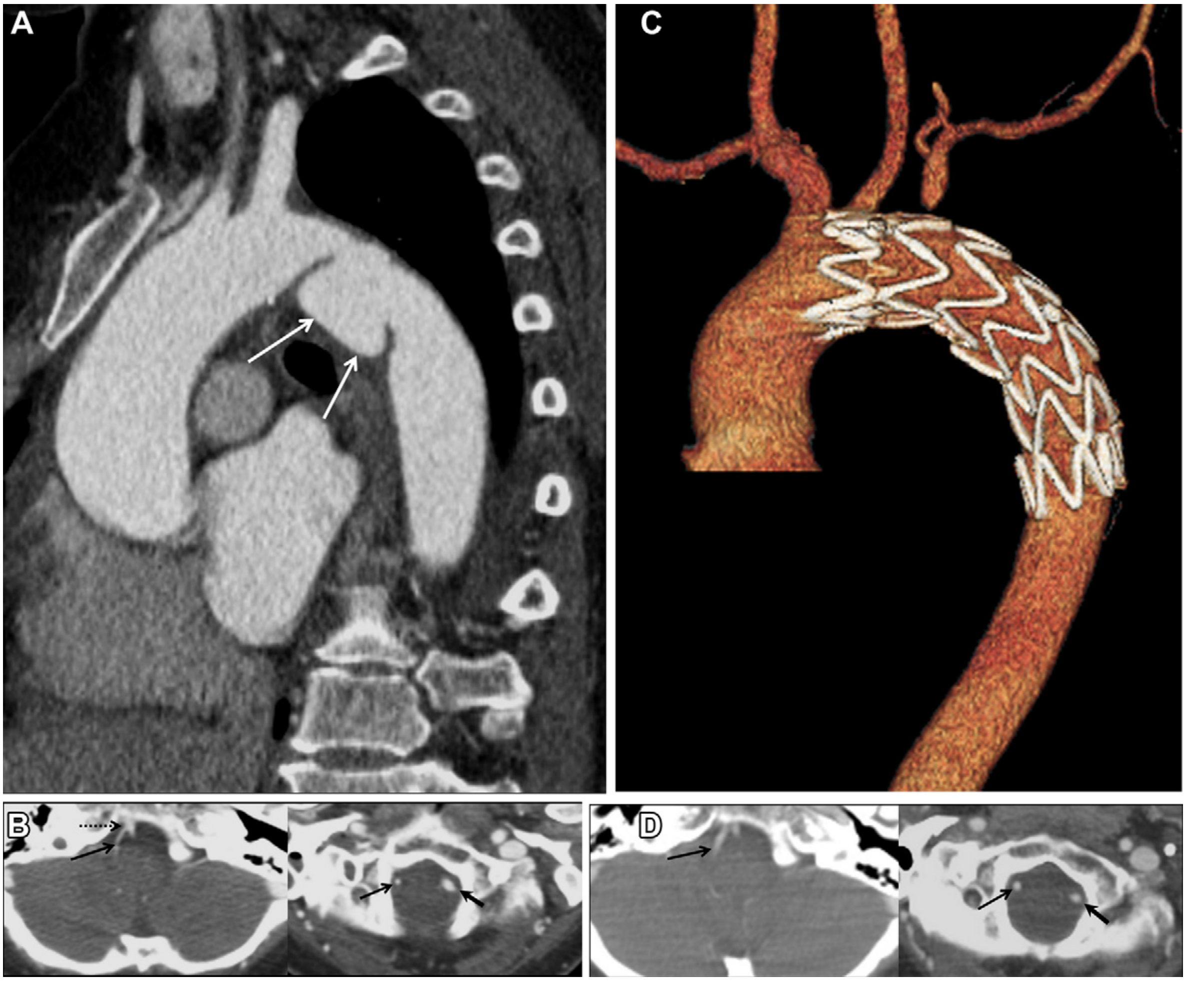

Fig 2. Images are shown for a 73-year-old man with a traumatic aortic pseudoaneurysm. **A,** A contrast-enhanced computed tomography (CT) scan reveals an aortic pseudoaneurysm (*arrows*) at the proximal descending thoracic aorta. **B,** A patent vertebrobasilar junction (*dotted arrow*) is demonstrated. The left vertebral artery (VA) (*thick arrow*) is dominant compared with the hypoplastic right VA (*thin arrow*). **C,** The left subclavian artery (LSCA) is covered, without recanalization, during the stent graft procedure. **D,** A contrast-enhanced CT scan at the 6-month follow-up reveals the right VA (*thin arrow*) hypertrophy that is nearly the same as the left VA (*thick arrow*).

Further studies are needed to determine whether vascular plugs may play a role in preventing type II endoleaks from the LSCA in such patients.

A dominant left VA is typically an indication for revascularization before LSCA coverage during TEVAR[5,15,16]; however, it has recently been suggested that a dominant left VA alone, without other indications, does not require revascularization.[19] We performed LSCA coverage without revascularization in nine patients who had left dominant VAs or hypoplasia of right VAs, and none exhibited SCI or CVA on posterior circulation alone. Although the small study population limits the drawing of a definitive conclusion, there appear to be few serious complications directly related to LSCA coverage without revascularization, even in patients with a left dominant VA, if bilateral vertebrobasilar junctions are patent.

In addition, stent graft placement for abdominal aortic aneurysm is also known as a risk factor of paraplegia during TEVAR with LSCA coverage, but a case in our study implicated that TEVAR with LSCA coverage without revascularization might be carefully performed even in patients treated for AAA if bilateral vertebrobasilar junctions are patent; but, larger studies are needed.

Interestingly, seven of 27 patients (25.9%) in our study demonstrated hypertrophy of the right VA. VAs increased in size as fast as 1 month later after TEVAR and grew up to 36 months. Two of these had bilateral hypertrophy of the VAs on the follow-up CT scan. Two patients had hypoplastic right VAs before treatment, but after TEVAR, the right VAs became larger than the left VAs. These findings may result from right VA compensation for reduced LSCA blood flow. To our knowledge, few other reports have examined VA changes after LSCA coverage documented on CT scan.

The incidence of left arm ischemia is reported between 0% and 36%.[20] Overall, left upper extremity symptoms occur in ∼20% of patients who undergo TEVAR and LSCA coverage without prior revascularization. Five of 27 patients (18.5%) in our study had left arm symptoms, but no patients required a secondary intervention.

The present study has a few limitations. The retrospective study design, without comparing a nonrevascularized group with revascularized group, may have yielded biased results. As a second limitation, preprocedural and postprocedural VA diameters of intradural segments were not compared in all patients. Some patients enrolled in our study underwent preprocedural CT imaging that did not cover the level of the circle of Willis, and their VA diameters were compared among only foraminal segments after TEVAR on follow-up CT scan, even though a similar rate of VA hypertrophy was demonstrated with foraminal segments as intradural segments. Another limitation is the relatively small number of patients; further large, multicenter studies are needed.

CONCLUSIONS

LSCA coverage without revascularization can be safely performed during TEVAR in patients with bilateral patent vertebrobasilar junctions. Hypertrophy of the right VA was noted in 25.9% of patients after LSCA coverage.

AUTHOR CONTRIBUTIONS

Conception and design: DL
Analysis and interpretation: ML, MK
Data collection: ML, JW
Writing the article: ML, MK, DL
Critical revision of the article: DL, MK, DC
Final approval of the article: YY, TL, DC, YK
Statistical analysis: ML
Obtained funding: Not applicable
Overall responsibility: MK

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Submitted Sep 10, 2012; accepted Oct 25, 2012.