EuroIntervention 2015;10:e1-e7 published online e-article March 2015

# How should I treat a complex left subclavian artery stenosis involving the vertebral artery in a patient with subclavian steal syndrome and left internal mammary artery bypass graft?

Tareq Ibrahim[1]*, MD; Nicolas Langwieser[1], MD; Michael Dommasch[1], MD; Moritz Wildgruber[2], MD; Karl-Ludwig Laugwitz[1,3], MD

*1. Department of Angiology, 1. Medizinische Klinik und Poliklinik, Klinikum rechts der Isar der Technischen Universität München, Munich, Germany; 2. Institute of Diagnostic and Interventional Radiology, Klinikum rechts der Isar der Technischen Universität München, Munich, Germany; 3. DZHK (German Centre for Cardiovascular Research), partner site Munich Heart Alliance, Munich, Germany*

*Invited Experts: Hans Krankenberg[1], MD, PhD; Marco Roffi[2], MD; Alberto Cremonesi[3], MD*

*1. Department of Angiology, Cardiovascular Center Bad Bevensen, Bad Bevensen, Germany; 2. Division of Cardiology, University Hospital, Geneva, Switzerland; 3. GVM Care and Research, Interventional Cardiovascular Unit, Cotignola, Italy*

*The accompanying supplementary data are published online at: http://www.pcronline.com/eurointervention/82nd_issue/236*

## CASE SUMMARY

**BACKGROUND:** A 68-year-old male smoker presented with progressive symptoms of vertebrobasilar insufficiency and angina. His past medical history included arterial hypertension, diabetes mellitus, dyslipidaemia as well as diffuse coronary artery disease including left main disease. Of note, he had undergone coronary bypass surgery 12 years earlier utilising the left internal mammary artery.

**INVESTIGATION:** Physical examination, laboratory tests, duplex ultrasound imaging, contrast-enhanced magnetic resonance imaging and coronary angiography.

**DIAGNOSIS:** Severe bifurcation stenosis of the left subclavian and vertebral artery with consecutive subclavian steal syndrome and myocardial ischaemia.

**MANAGEMENT:** Bifurcation T-stenting using a self-expandable bare metal and a coronary drug-eluting stent.

**KEYWORDS:** bare metal stent, drug-eluting stent, peripheral intervention, subclavian artery, subclavian steal syndrome, T-stenting technique, vertebral artery

## PRESENTATION OF THE CASE

A 68-year-old male smoker presented with progressive dizziness, vertigo and temporary diplopia existing for two months. Six weeks previously, he had suffered from a sudden drop attack. Moreover, he complained about exertional angina during the last quarter of the year. Apart from that, he had no other symptoms. His past medical history included arterial hypertension, diabetes mellitus, dyslipidaemia as well as diffuse coronary artery disease including left main disease. Of note, he had undergone coronary bypass surgery 12 years earlier utilising the left internal mammary artery (LIMA) on the left anterior descending (LAD) coronary artery and two venous grafts on the left circumflex artery (LCX) and right coronary artery (RCA). He was on a long-term medication consisting of acetylsalicylic acid, simvastatin, bisoprolol, ramipril, and metformin. On clinical examination left carotid and subclavian bruits were noted. He was found to have a 55 mmHg difference in upper extremity systolic blood pressure with 125 mmHg on the right and 70 mmHg on the left arm. Pulses in the lower extremities were intact with a systolic blood pressure of 160 mmHg on both legs. ECG and the laboratory tests showed no abnormalities.

Non-invasive imaging using duplex ultrasound suggested diffuse atherosclerotic disease of the extracranial arteries, including severe stenosis of the proximal left subclavian artery (SA) (>250 cm/s) with alternating flow within the left vertebral artery (VA) under resting conditions **(Figure 1A)**, of the innominate artery (≈200 cm/s) as well as of the left internal carotid artery (≈200 cm/s), and occlusion

**Corresponding author: Department of Angiology, 1. Medizinische Klinik und Poliklinik, Klinikum rechts der Isar der Technischen Universität München, Ismaningerstrasse 22, 81675 München, Germany. E-mail: t.ibrahim@lrz.tu-muenchen.de*



DOI: 10.4244/EIJV10I11A236

of the right carotid artery with left to right crossflow via the anterior cerebral artery on transcranial Doppler examination. Clinical assessment by a neurologist confirmed symptomatic SA stenosis with consecutive subclavian steal syndrome in this patient with diffuse supra-aortic vessel disease. For further evaluation contrast-enhanced magnetic resonance imaging was performed revealing the complex stenosis of the left SA at the mid segment involving the left vertebral artery origin (**Figure 1B**, **Figure 1C**).

Subsequent coronary angiography demonstrated a patent venous graft on the dominant and proximal occluded RCA, a filiform stenosis of the left main artery with occlusion of the proximal LAD and a rather small LCX with occlusion of a marginal branch. The venous graft supplying the marginal branch was also occluded. Angiography of the left SA depicted the severe stenosis at the curvature of the SA with an alternating flow in the nearby VA as well as an antegrade flow in the LIMA graft supplying the LAD **(Figure 1D)**.

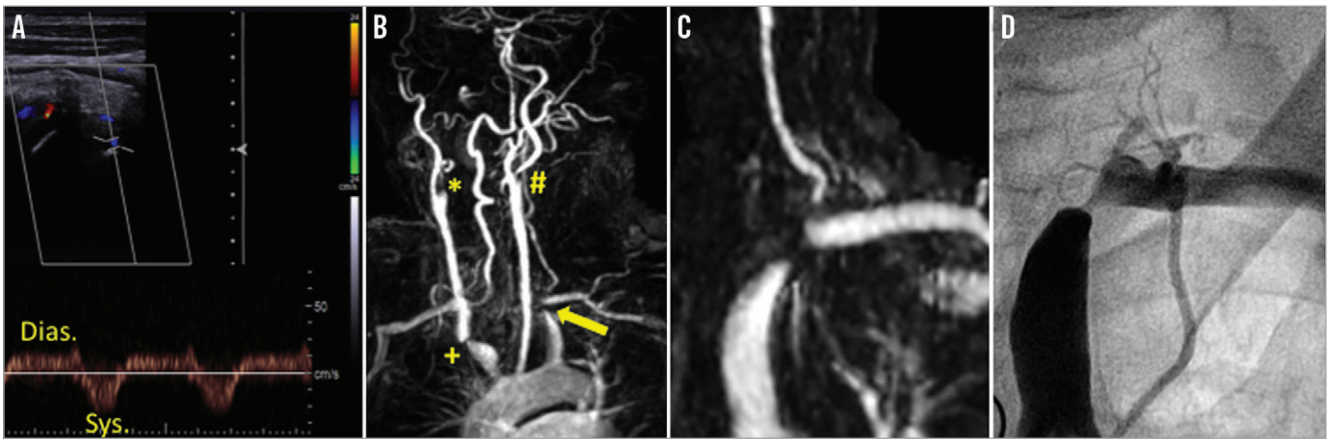


**Figure 1.** *Subclavian steal syndrome caused by advanced supra-aortic disease. A) Colour-coded Doppler ultrasound imaging of subclavian steal syndrome demonstrating an alternating flow within the extracranial left verterbral artery (VA) under resting conditions with antegrade flow in the diastolic (Dias.) and retrograde flow in the systolic (Sys.) phase due to severe stenosis of the proximal left subclavian artery (SA). B) MR angiography of the supra-aortic arteries revealing filiform stenosis of the left SA (arrow) as well as the concomitant lesions including occlusion of the right (*) and stenosis of the left internal carotid artery (#) and of the innominate artery (+). C) Magnification of the MRA demonstrates the close relation of the high grade stenosis of the left SA and the nearby stenosis of the VA. D) Conventional angiography clearly demonstrates the SA stenosis and the retrograde perfused nearby VA as well as the antegrade perfused LIMA origin.*

EuroIntervention 2015;10:e1-e7 published online e-article March 2015

# How would I treat?

**THE INVITED EXPERT'S OPINION**

Hans Krankenberg*, MD, PhD

*Department of Angiology, Cardiovascular Center Bad Bevensen, Bad Bevensen, Germany*

In this patient, over the years, multiple cardiovascular risk factors led to an extensive atherosclerosis and caused an intermittent subclavian steal syndrome.

The patient's symptoms are probably due to vertebrobasilar and myocardial ischaemia as a consequence of an alternating flow in the vertebral artery and in the LIMA graft as a result of a pressure gradient caused by a severe stenosis of the proximal left subclavian artery. However, embolisation of atherosclerotic material as a cause of neurological events cannot be entirely excluded[1].

In general, a subclavian artery stenosis increases cardiovascular mortality. In particular, the involvement of the LIMA graft, supplying the LAD, puts the life of our patient at risk. Therefore, the first and most important treatment objective is the revascularisation of the subclavian artery. According to the ESC guidelines and following a previously reported favourable five-year primary patency rate of 89% and a 95% secondary patency rate in those patients with symptomatic restenosis[2], we would choose the endovascular-first approach.

Access to the subclavian artery would be achieved through the common femoral artery, using a 6 Fr sheath and a 0.035" guidewire. A proper placement of the stent to cover the stenosis fully but not to compromise the vertebral artery or the LIMA bypass is mandatory. For that reason, we would choose a balloon-expandable stent, which is not likely to move during expansion as can happen with self-expanding nitinol stents.

From our point of view, **Figure 1D** shows a sufficient filling of the vertebral artery origin, no stenosis but only an unsteadiness concerning the vessel wall. An ostial stenosis of the vertebral artery may have been simply pretended at the MRT **(Figure 1C)** because of its angulated take-off. Nonetheless, stenting of the subclavian artery may compromise the adjacent vertebral artery, as well as the LIMA graft. In order to deal with that, we would secure both vessels by introducing 0.018" guidewires transbrachially. This would allow for kissing balloon angioplasty or provisional stenting in case of flow-limiting lumen constriction. Furthermore, the transbrachial sheath would enable additional angiography in order to visualise the take-offs of the vessels precisely.

In a second step, a few weeks after the initial procedure and dependent on symptoms and the general condition of the patient, the moderate left internal carotid artery stenosis might be considered for revascularisation in order to prevent stroke. The right internal carotid artery occlusion may have worsened symptoms of the subclavian steal but does not itself require intervention because there is a sufficient left to right cross flow.

The patient's cardiovascular risk factors have to be controlled, and a dual antiplatelet therapy for 30 days would be advisable.

## Conflict of interest statement

The author has no conflicts of interest to declare.

*Corresponding author: Department of Angiology, Cardiovascular Center Bad Bevensen, Römstedter Straße 25, 29549 Bad Bevensen, Germany. E-mail: h.krankenberg@hgz-bb.de*

# How would I treat?

## THE INVITED EXPERTS' OPINION

Marco Roffi[1]*, MD, FESC, FACC; Alberto Cremonesi[2], MD

*1. Division of Cardiology, University Hospital, Geneva, Switzerland; 2. GVM Care and Research, Interventional Cardiovascular Unit, Cotignola, Italy*

This 68-year-old patient presented with multisite artery disease characterised by an advanced involvement of the supra-aortic trunks as well as of the coronary arteries. The 2011 European Society of Cardiology (ESC) guidelines on the diagnosis and treatment of peripheral artery diseases recommend, as a general rule, to treat the symptomatic territory first in patients requiring multisite revascularisation[3]. The patient described has coronary-subclavian steal syndrome, and the treatment of the left subclavian artery (SA) stenosis should lead to an improvement of both vertebrobasilar symptoms – by re-establishing an antegrade perfusion in the left vertebral artery (VA) – and exertional angina – by improving the flow to the LIMA graft[4]. While a surgical approach to subclavian steal syndrome – using axillo-axillary or carotid subclavian bypasses – has been described with good long-term patency outcomes[5], in most centres these lesions are approached percutaneously. In the present case, the advanced supra-aortic disease (i.e., additional significant stenosis of the innominate artery, occlusion of the right internal and stenosis of the left internal carotid arteries) precludes surgical options anyway. Based on the limited images at our disposal, we do not believe that the origin of the left VA is significantly stenosed, though arising very close to the SA lesion. Of note, MRA – which is a flow study and not an anatomic study – may overestimate lesions in vascular territories at low flow (such as at the origin of the left VA in the presence of alternating flow).

With respect to the technical aspects, we would approach this intervention by a retrograde femoral access. The patient would receive aspirin and 5,000 IU of unfractionated heparin. Using a 5 Fr Judkins right diagnostic catheter to engage the left SA, a 0.035" floppy hydrophilic wire (e.g., Glidewire®; Terumo Corp., Tokyo, Japan) should be able to pass the lesion and be followed by the diagnostic catheter across the lesion. The floppy wire should then be exchanged for a 0.035" long stiff wire (e.g., Supra Core®; Abbott Vascular, Santa Clara, CA, USA). The short femoral sheath is then exchanged for a 90 cm long 6 Fr sheath (e.g., Flexor Shuttle Select® Guiding Sheath; Cook Medical, Bloomington, IN, USA), which is engaged over the wire at the origin of the SA. An angiogram with digital subtraction function and store-image function is performed. Balloon predilatation is performed with an undersized 0.035" over-the-wire (OTW) balloon (e.g., 5.0×40 mm Admiral Xtreme™; Invatec-Medtronic, Minneapolis, MN, USA). Using the stored ground image as reference, a self-expanding nitinol stent is deployed across the origin of the VA (e.g., 8.0×60 mm, S.M.A.R.T® Vascular Stent; Cordis, Johnson & Johnson, Warren, NJ, USA) and post-dilated (e.g., 7.0×40 mm Admiral). In our experience, there is no need to place a filter emboli protection device in the VA as cerebral embolic events in this setting (i.e., flow reversal in the VA) are exceedingly rare. In case of a stenosis of the origin of the VA artery we would consider performing the procedure using instead a long 7 Fr sheath and either securing the VA with a 0.014" wire – in case of moderate lesion of the VA ostium – or stenting the origin of the VA (e.g., with a coronary stent) before stenting the SA (T stent) if the VA stenosis is severe.

Last but not least, this patient has advanced internal carotid disease – probably asymptomatic – which requires attention. In the presence of a contralateral carotid occlusion an asymptomatic revascularisation of carotid stenosis of ≥70% should be considered. In this setting, carotid artery stenting should be favoured over endarterectomy, as the presence of contralateral carotid occlusion increases the risk of stroke with surgery but not with stenting[6].

## Conflict of interest statement

The authors have no conflicts of interest to declare.

**Corresponding author: Interventional Cardiology Unit, University Hospital, Rue Gabrielle Perret-Gentil 4, 1211 Geneva 14, Switzerland. E-mail: marco.roffi@hcuge.ch*

EuroIntervention 2015;10:e1-e7 published online e-article March 2015

# How did I treat?

## ACTUAL TREATMENT AND MANAGEMENT OF THE CASE

In accordance with the interdisciplinary vascular board at our institution, endovascular revascularisation of the left SA was recommended. For antegrade percutaneous intervention, an 8 Fr sheath was inserted through the right groin and 5,000 IU of heparin was administered. The left SA was cannulated with an 8 Fr Hockey Stick guiding catheter. The initial angiogram demonstrated the stenosis at the curvature of the SA **(Figure 2A, Moving image 1)**, which was passed with a long 0.018-inch V-18 ControlWire™ (Boston Scientific Corporation, Natick, MA, USA) and predilated with a 4.0×40 mm PowerCross™ balloon at 8 atm (ev3 Europe, Paris, France). Hereafter, the VA showed antegrade perfusion revealing the close relation of the SA and VA stenosis **(Figure 2B, Moving image 2)**, on which we decided to intervene by bifurcation T-stenting.

The V-18 ControlWire was exchanged for a 0.035-inch standard wire to achieve higher stability and to allow precise positioning of a 7.0×40 mm self-expanding EverFlex™ stent (ev3 Europe), which was implanted in the SA across the VA ostium and terminating prior to the LIMA origin. Post-dilatation was carried out by 7.0×20 mm FoxCross balloon at 6 atm (Abbott Vascular, Beringen, Switzerland), whereas the standard wire was exchanged for the previously used 0.018-inch V-18 ControlWire to allow double wiring through the 8 Fr catheter **(Figure 2C)**. The subsequent angiogram showed the primary result within the SA and the stenotic but patent VA origin **(Figure 2D, Moving image 3)**. An additional 0.014-inch Pilot 200 guidewire (Abbott Vascular, Diegem, Belgium) was used to probe the left VA through the SA stent struts. Predilatation of the VA ostium was performed with a 2.5×20 mm coronary balloon. Thereafter, a 4.0×12 mm coronary DES, Resolute Integrity (Medtronic, Galway, Ireland), was advanced through the SA stent struts into the VA while a 6.0×20 mm PowerCross balloon (ev3 Europe) was placed in the axillary artery. The VA stent was positioned to cover its ostium while protruding into the SA and was deployed at 14 atm **(Figure 3A, Figure 3B, Moving image 4)**. After deflation, the stent balloon was slightly retrieved and kissing balloon angioplasty of the bifurcation was carried out with both balloons at 10 atm **(Figure 3C)**. Final angiography demonstrated widely patent bifurcation stents with no residual stenosis **(Figure 3D, Moving image 5)**. The post-procedural hospital course was significant for resolution of patient symptoms and he was discharged two days later on a dual platelet inhibition therapy for six months' duration.

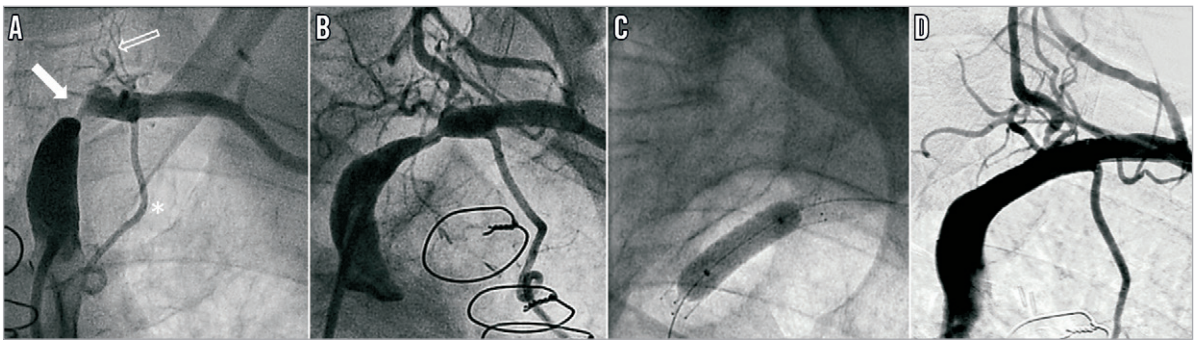


**Figure 2.** *Endovascular treatment of the left subclavian artery stenosis. A) Baseline angiography demonstrating the stenosis of the left SA (closed arrow), the nearby retrograde perfused VA (open arrow), and the LIMA bypass (*). B) Post-dilatation angiography revealing VA flow reversal with stenotic vessel origin. C) Self-expanding stent implantation within the SA with low-pressure post-dilatation. D) Post-implantation angiography with normal flow in the SA and preserved patency of the VA.*

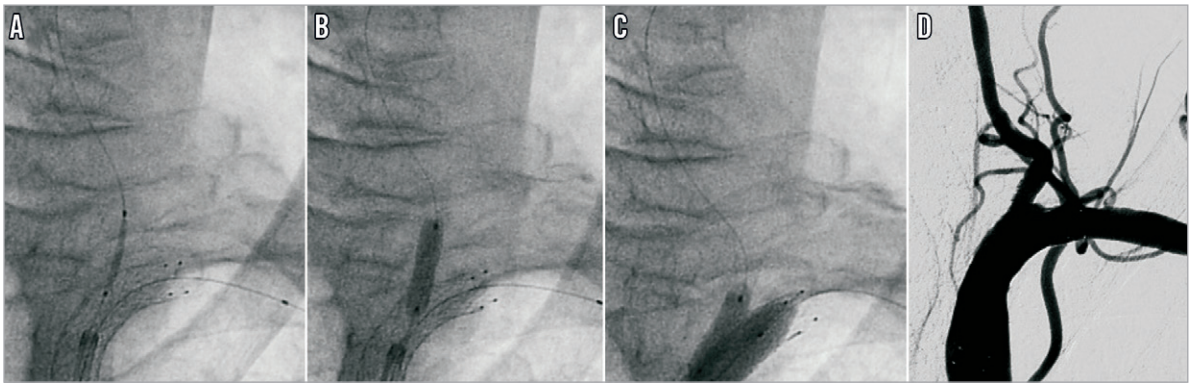


**Figure 3.** *Endovascular treatment of the left vertebral artery stenosis. A) Placement of a DES within the VA through the SA stent struts with a second balloon being positioned in the axillary artery. B) Stent deployment with full coverage of the VA ostium. C) Kissing balloon angioplasty of the bifurcation after retraction of the vertebral and axillary artery balloon. D) Final angiogram of bifurcation T-stenting with restored normal flow in both the SA and VA.*

Subclavian steal syndrome (SSS) is a vascular phenomenon in which a steno-occlusive lesion of the proximal SA causes retrograde flow in the ipsilateral VA away from the brain stem, subsequently causing vertebrobasilar insufficiency. The prevalence of SSS in a recently published large series of ultrasound exams of the extracranial neck vessels yielded 5.4%, whereas the majority of patients were asymptomatic[7]. However, symptomatic patients may present with neurological features or with myocardial ischaemia in case of ipsilateral IMA-coronary bypass graft as in our patient. It is generally agreed that symptomatic lesions of the SA should be revascularised.

Endovascular therapy by means of angioplasty and stenting has evolved as a safe and effective treatment for symptomatic lesions of the SA with very low rates of complications and excellent long-term results with primary one-year patency rates of about 90%[2]. Mostly, SA lesions are found in the proximal vessel segment; however, lesions located in the mid-vessel segment involving the VA pose a special challenge due to potential plaque shift and risk of stroke. Intervention of the extracranial VA stenosis is generally recommended in patients with symptomatic lesions refractory to modification of cardiovascular risk factors and antiplatelet therapy or in asymptomatic patients in the presence of contralateral VA hypoplasia or occlusion[8]. Although our patient does not fit the above-mentioned indications, this case represents a rare clinical scenario where the primary indication for intervention is SA ischaemia and the VA is being stented to preserve its patency and to improve cerebral haemodynamics.

The T-stenting technique was originally reported for the treatment of plaque shift in bifurcation coronary artery stenosis and consists of the placement of two stents in a T-shape configuration[9]. Our endovascular approach is distinguished by stenting of the SA prior to the VA since visualisation, and thus accurate coverage, of the VA ostium may be facilitated. Furthermore, if the VA stent is implanted first and protrudes into the SA lumen, passage of a stent into the SA may pose technical difficulties. From our point of view, the use of a self-expanding instead of a balloon-expandable stent within the mid segment of the SA offers several advantages. First, this stent type allows preservation of vessel flexibility at the crest of the SA. Second, self-expanding stents usually achieve good vessel apposition without high-pressure post-dilatation, thus avoiding maximal plaque shift, which may preserve VA patency. On the other hand, interventional experience shows that the precise placement of a self-expandable stent may be more challenging as compared to a balloon-expandable stent, since these types of stent usually tend to move forward slightly during deployment.

Balloon-expandable coronary stents are well established in the endovascular treatment of occlusive extracranial VA disease. While the procedural success rate is extremely high with very low complication rates, in-stent restenosis is a relevant concern. Evidence from case series of plain VA stenting support the notion that the use of DES reduces the rate of restenosis as compared to bare metal stents (BMS); however, no randomised data are currently available.

In a recent review article, the mean restenosis rate after DES as compared to BMS averaged 13% vs. 26%, respectively[10]. Comparable results with a significant reduction of in-stent restenosis have recently been described for DES in a large patient cohort from China[11], as well as in a meta-analysis by Langwieser et al[12].

Apart from the stent type used for bifurcation stenting of complex SA/VA lesions, the interventional technique with sufficient plaque coverage of the VA ostium may be of major importance for both technical success and long-term outcome of VA stenosis. To cover the VA ostium fully, we deployed the stent at the level of the far side vessel origin with protrusion into the SA lumen at the proximal site and performed final kissing balloon inflation with high pressures to allow for optimal vessel apposition of both stents.

Although bifurcation stenting techniques have been well described in coronary artery stenosis, they have scarcely been described in cases of supra-aortic vessel procedures[13,14]. The T-stent technique is suitable for bifurcation lesions with a side branch of 90 degree angle and in which the vessel size differs significantly

between the main and the side branch, which is usually the case in SA/VA lesions. This technique has been described recently in a case with a SA/VA bifurcation stenosis using two balloon-expandable stents[13]. To our knowledge, our case is the first report of a dedicated T-stenting using a self-expanding stent in the SA and a balloon-expandable DES in the VA. Although the long-term consequences of our endovascular approach are not known and need further investigation, we believe that this dedicated stent technique is an interesting therapeutic option to be considered in complex SA lesions involving the VA origin.

## Conflict of interest statement

The authors have no conflicts of interest to declare.

## References


1. Potter BJ, Pinto DS. Subclavian steal syndrome. *Circulation*. 2014;129:2320-3.
2. De Vries JP, Jager LC, Van den Berg JC, Overtoom TT, Ackerstaff RG, Van de Pavoordt ED, Moll FL. Durability of percutaneous transluminal angioplasty for obstructive lesions of proximal subclavian artery: long-term results. *J Vasc Surg*. 2005;41:19-23.
3. European Stroke Organisation, Tendera M, Aboyans V, Bartelink ML, Baumgartner I, Clément D, Collet JP, Cremonesi A, De Carlo M, Erbel R, Fowkes FG, Heras M, Kownator S, Minar E, Ostergren J, Poldermans D, Riambau V, Roffi M, Röther J, Sievert H, van Sambeek M, Zeller T; ESC Committee for Practice Guidelines. ESC Guidelines on the diagnosis and treatment of peripheral artery diseases: Document covering atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries: the Task Force on the Diagnosis and Treatment of Peripheral Artery Diseases of the European Society of Cardiology (ESC). *Eur Heart J*. 2011;32:2851-906.
4. Faggioli G, Pini R, Cremonesi A, Grattoni C, Longhi M, Mauro R, Castriota F, Stella A. Endovascular treatment of late coronary-subclavian steal syndrome. *J Thorac Cardiovasc Surg*. 2014;148:2112-6.
5. Song L, Zhang J, Li J, Gu Y, Yu H, Chen B, Guo L, Wang Z. Endovascular stenting vs. extrathoracic surgical bypass for symptomatic subclavian steal syndrome. *J Endovasc Ther*. 2012;19:44-51.
6. Roffi M, Mukherjee D, Clair DG. Carotid artery stenting vs. endarterectomy. *Eur Heart J*. 2009;30:2693-704.
7. Labropoulos N, Nandivada P, Bekelis K. Prevalence and impact of the subclavian steal syndrome. *Ann Surg*. 2010;252:166-70.
8. Ernemann U, Bender B, Melms A, Brechtel K, Kobba J, Balletshofer B. Current concepts of the interventional treatment of proximal supraaortic vessel stenosis. *Vasa*. 2012;41:313-8.
9. Carrie D, Karouny E, Chouairi S, Puel J. "T"-shaped stent placement: a technique for the treatment of dissected bifurcation lesions. *Cathet Cardiovasc Diagn*. 1996;37:311-3.
10. Cremonesi A, Roffi M, Carvalho De Campos Martins E, Castriota F. Subclavian, brachiocephalic and vertebral artery interventions, in The PCR-EAPCI textbook - Percutaneous interventional cardiovascular medicine; Volume III, Chapter 42. Europa Edition; 2012.
11. Song L, Li J, Gu Y, Yu H, Chen B, Guo L, Zhang J. Drug-eluting vs. bare metal stents for symptomatic vertebral artery stenosis. *J Endovasc Ther*. 2012;19:231-8.
12. Langwieser N, Buyer D, Schuster T, Haller B, Laugwitz KL, Ibrahim T. Bare metal vs. drug-eluting stents for extracranial vertebral artery disease: a meta-analysis of nonrandomized comparative studies. *J Endovasc Ther*. 2014;21:683-92.
13. Biria M, Tadros P, Gupta K. Subclavian-vertebral artery bifurcation stenting using drug-eluting stents: a report of two cases using different techniques. *J Invasive Cardiol*. 2007;19:E156-9.
14. Roguin A, Alhaddad IA. Crush stenting of bifurcational left subclavian-vertebral artery stenosis. *Catheter Cardiovasc Interv*. 2004;62:393-5.


## Online data supplement

**Moving image 1.** Baseline angiography demonstrating the stenosis of the left SA (closed arrow), the nearby retrograde perfused VA (open arrow), and the LIMA bypass (*).

**Moving image 2.** Post-dilatation angiography revealing VA flow reversal with stenotic vessel origin.

**Moving image 3.** Conventional angiography clearly demonstrates SA stenosis and the retrograde perfused nearby VA as well as the antegrade perfused LIMA origin.

**Moving image 4.** Placement of a DES within the VA through the SA stent struts with a second balloon being positioned in the axillary artery.

**Moving image 5.** Final angiogram of bifurcation T-stenting with restored normal flow in both the SA and VA.