Medical Science

Case report

Innominate Steal Syndrome: A Two Patient Case Report and Review

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Abstract: Like subclavian artery disease, innominate artery disease can result in reversal of flow in the ipsilateral vertebral artery. Unlike subclavian artery disease, innominate artery disease can also affect cerebral perfusion in the ipsilateral carotid territory, is less well characterized in the literature, and is more challenging to revascularize. Herein, we present two cases of 'innominate steal' and a summary of relevant literature focusing on innominate artery disease.

1.   Introduction

Subclavian steal phenomenon is the reversal of vertebral artery flow due to an ipsilateral proximal subclavian or innominate artery stenosis. Reversal of flow in the vertebral artery ipsilateral to the subclavian or innominate stenosis is caused by a pressure drop distal to the stenosis that causes blood flow to course down its pressure gradient from the basilar artery to the ipsilateral arm. Subclavian steal syndrome is the presence of subclavian steal phenomenon associated with symptoms of vertebrobasilar insufficiency, such as vertigo, dizziness, diplopia, ataxia, and nausea [1].

Subclavian steal phenomenon is often asymptomatic, as an intact circle of Willis enables adequate perfusion of the posterior cerebrum despite flow reversal in a vertebral artery. Similarly,

though a marker for atherosclerotic disease, the natural history of subclavian steal phenomenon/syndrome is thought to be relatively benign with a low incidence of ipsilateral posterior circulation cerebrovascular events [2].

Subclavian steal phenomenon/syndrome is more commonly encountered on the left as left subclavian artery atherosclerosis is 3–5 times more common than right-sided disease for unknown reasons [3]. When vertebral flow reversal occurs on the right, it can be a result of either subclavian or innominate artery stenosis or occlusion. Innominate artery disease can cause not only vertebral flow reversal but also flow reversal in the ipsilateral carotid system. Flow reversal in both the right vertebral and carotid systems has been termed 'double subclavian steal', 'innominate steal', and 'cerebral steal' [4]. We will refer to it as 'innominate steal'.

Compared to subclavian steal, innominate steal is less well characterized. It was first described clinically in 1966 [5]. It is less common than subclavian steal phenomenon, but the proportion of patients who are symptomatic is likely higher with innominate versus subclavian steal. Both surgical and endovascular treatment of innominate obstructive disease are more complex than for subclavian disease [6–10].

Herein, we present two cases of innominate steal syndrome, review the clinical manifestations and imaging findings of this disorder, describe treatment options, and summarize the sparse relevant literature.

2. Case 1

A 55 year-old male presented with episodes of dizziness and nausea that had worsened over the past 6 months. The dizzy spells were associated with visual "black spots" and were worse while working with his right hand. He had a history of hypertension, hyperlipidemia, tobacco abuse, and peripheral arterial disease and had undergone innominate artery stenting for symptoms of vertebrobasilar insufficiency three years prior. His medications included aspirin, amlodipine, and atorvastatin. He was a former smoker. On physical examination, the systolic blood pressure in the left arm was 125 mmHg, and the systolic blood pressure in the right arm was 80 mmHg. The pertinent pulse exam is described in Table 1.

Table 1. Pulse exam of patient 1 and 2.

	Patient 1		Patient 2	
Level	Right	Left	Right	Left
Superficial Temporal	0	2+	0	2+
Carotid	0	2+	0	2+
Brachial	0	2+	0	2+
Radial	0	2+	0	2+

A cerebrovascular duplex ultrasound showed flow reversal in the right vertebral artery and right common and internal carotid arteries (ICA) without stenosis in the visualized extracranial cervical vessels bilaterally (Figure 1). A computerized tomography (CT) angiogram confirmed severe ostial stenosis of the innominate artery at the site of the prior stent. After informed consent, the patient was brought to the catheterization laboratory for endovascular treatment for symptomatic innominate artery stenosis.
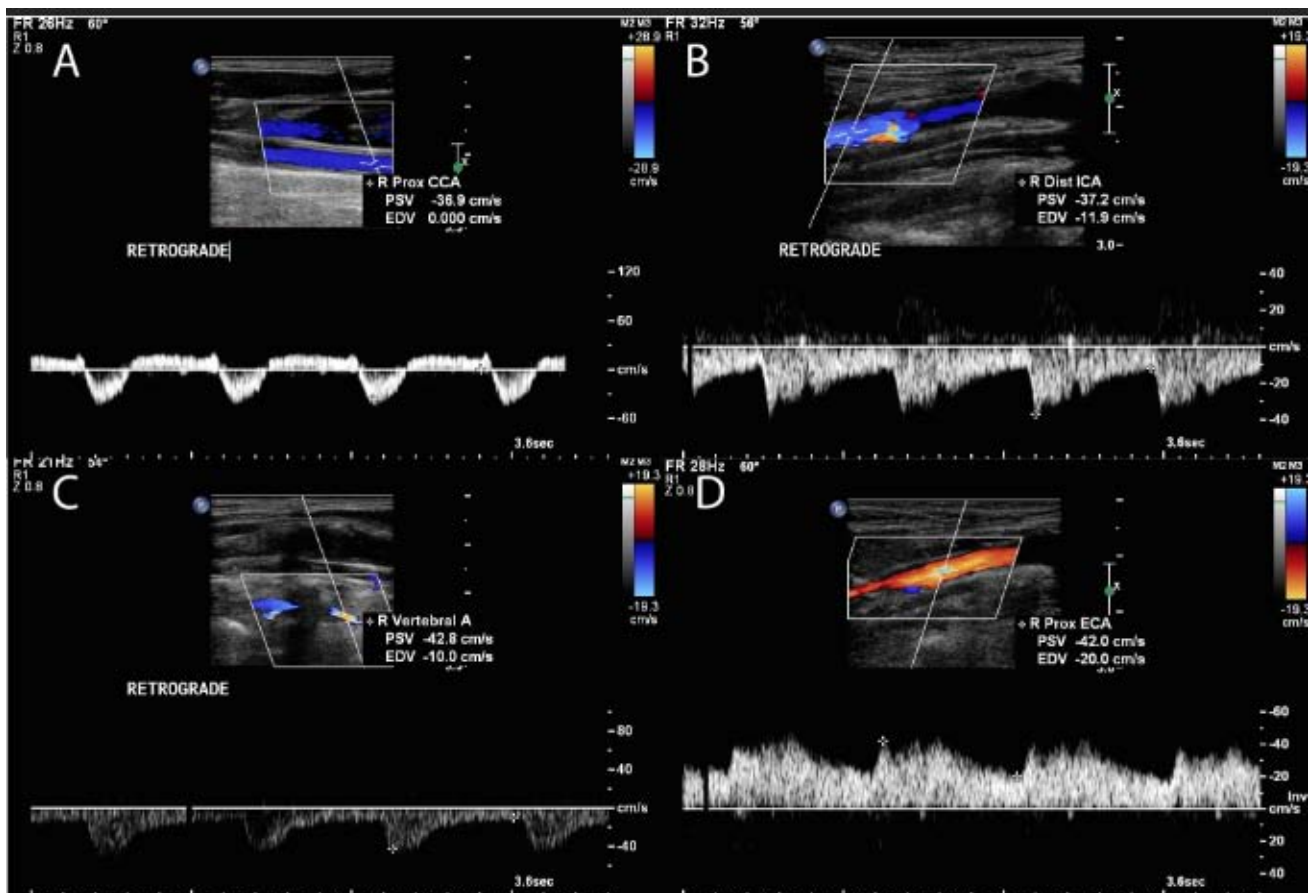

Figure 1. Composite of spectral doppler waveforms demonstrating flow reversal (blue arrow) in (A) right common carotid (B) right internal carotid (C) right vertebral artery and blunted antegrade flow (red arrow) in the (D) right external carotid.

Right common femoral artery access was obtained. On arch aortography, there was evidence of retrograde filling of the right vertebral and right common and internal carotid arteries (Supplemental Figure 1). Selective innominate angiography was performed but poor contrast opacification of the innominate artery immediately distal to the stenosis raised concern for a lack of 'landing zone' for the distal portion of a stent (Figure 2). Thus, left carotid angiography was performed to elucidate the distal innominate anatomy via retrograde filling of the right CCA (Supplemental Figure 1).
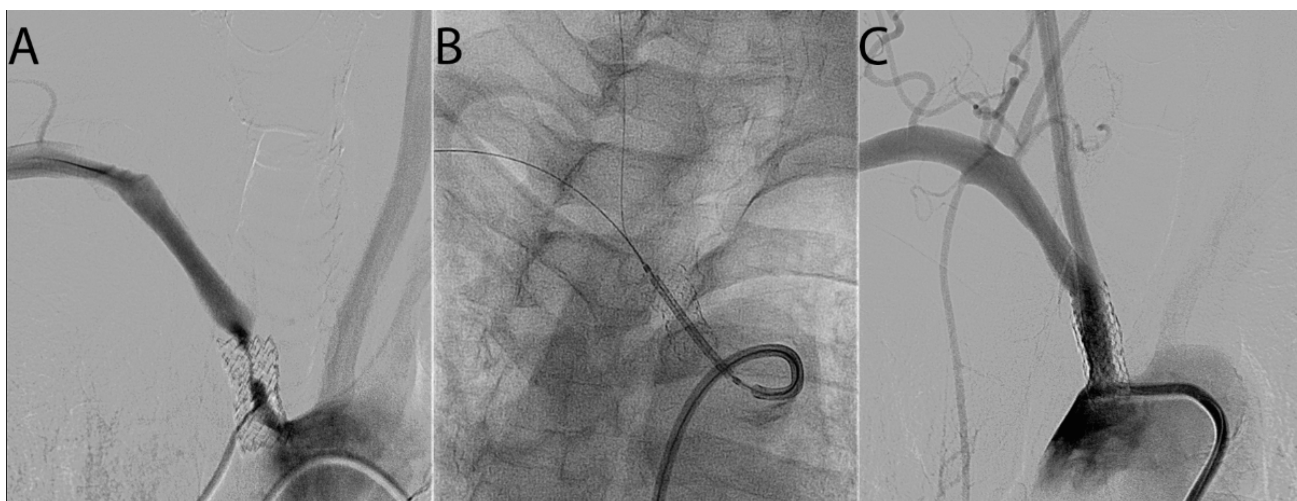
Figure 2. (A) Baseline angiogram of severe innominate artery restenosis without antegrade right common carotid artery flow. (B) Express stent across the innominate stenosis advanced over both wires in the right common carotid and subclavian artery. (C) Final angiogram of the innominate artery after stenting with antegrade flow in the right common carotid artery.

A 4F Berenstein catheter (Angiodynamics; Latham, NY) was telescoped through an 8F H1 guide catheter (Cook Medical; Bloomington, IN). The innominate artery was engaged with the diagnostic catheter. A Prowater Flex guide wire (Asahi; Nagoya, Japan) was advanced past the stenosis to the right brachial artery. The 4F Berenstein catheter was advanced over the wire to the origin of the innominate artery, keeping the catheter tip proximal to the stenosis. The H1 guide catheter was advanced over catheter/wire combination to the origin of the innominate artery.

A 3.0×30 mm Sprinter balloon (Medtronics; Minneapolis, MN) was used to pre-dilate the innominate in-stent restenosis (ISR). The Sprinter balloon was exchanged for a Quick-cross 0.018-inch (Spectranetics; Colorado Springs, CO) catheter which was used to exchange the 0.014-inch guidewire for the stiffer 0.018-inch Hi-Torque Steelcore wire (Abbott Laboratories; Abbott Park, IL) in the brachial artery. A 315 cm Barewire (Abbott Laboratories) was advanced to the distal cervical internal carotid artery, and a 4.0–7.0 mm NAV6 Emboshield (Abbott Laboratories) was deployed in the distal cervical internal carotid artery. An 8.0×27 mm (0.035) Express stent (Boston Scientific, Natick, MA) was advanced over both wires but could not be deployed across the innominate, likely due to the 'nose' of the stent catheter abutting the common carotid-right subclavian carina. The 8.0×27 mm Express was exchanged for a 7.0×19 mm 0.018'-inch Express stent that was advanced over the Barewire and deployed across the innominate ISR. The stent was post dilated with an 8.0×20 mm Optapro balloon (Cordis; Bridgewater Township, NJ). On final angiography, there was no residual stenosis, no evidence of cerebral embolization, and antegrade flow in the right vertebral and carotid arteries was present (Figure 2; Supplemental Figure 1). The patient had no complications and was discharged home the next day. Close follow-up of his blood pressure was

pursued by daily phone calls over the next week due to his risk of cerebral hyperperfusion syndrome. At a 30-day follow-up visit, he had resolution of his neurological symptoms.

3.   Case 2

A 43 year-old male was hospitalized with heart failure after a recent 3-vessel coronary artery bypass graft surgery, ascending aorta replacement, and failed aortic valve replacement due to a small annulus. He was found to have severe left ventricular outflow obstruction, aortic valve stenosis and regurgitation, and findings consistent with constrictive pericarditis. He underwent redo sternotomy with aortic valve replacement with annulus enlargement and epicardial stripping. He was also known to have an innominate artery occlusion; however, this lesion was not addressed during the above surgeries as it was felt to be asymptomatic.

Post-operatively, he was noted to have positional episodes of left arm and leg weakness with confusion upon standing. These episodes were reliably reproduced with position change and were not correlated with orthostatic hypotension. His pulse exam is described in Table 1. CT angiography demonstrated complete occlusion of the proximal innominate artery. Cerebrovascular duplex ultrasound demonstrated biphasic (antegrade and retrograde) flow in the right internal carotid artery and retrograde flow in the right vertebral artery, findings consistent with innominate steal physiology. Neurologic consultation was obtained and felt his clinical presentation was consistent with innominate steal syndrome. His providers felt innominate revascularization was indicated. Due to two prior thoracotomies, endovascular treatment was preferred over surgery.

After informed consent, he was brought to the catheterization laboratory for possible endovascular treatment. Via the left common femoral artery, ascending aortography demonstrated a common origin of the innominate and left common carotid artery, reconstitution of the right subclavian artery from retrograde filling of the right vertebral artery, and antegrade filling of the right common carotid artery from the reconstituted right subclavian artery (Supplemental Figure 2). Selective innominate angiography with a VITEK catheter (Cook) also revealed a 50% stenosis of the origin of the left common carotid. Via the right radial artery, a Navicross catheter (Terumo; Tokyo, Japan) was advanced to the distal cap of the innominate occlusion. Simultaneous contrast injections were performed through the VITEK in the innominate artery and the Navicross in the right subclavian artery (Figure 3). Due to the proximity of the proximal cap of the occlusion to the left common carotid takeoff, simultaneous stenting of the left CCA and innominate was planned.

The VITEK catheter was telescoped through an 8F HI guiding catheter (Terumo). The left CCA was engaged with the VITEK catheter. An angled Glidewire (Terumo) was advanced through the VITEK catheter into the left common carotid artery and the VITEK catheter was advanced into the common carotid. The H1 guide was advanced to the origin of the left CCA. The glidewire was exchanged for an 0.018-inch workhorse wire.

Via the right radial artery, a 6F 80 cm Shuttle sheath (Cook) was advanced to the distal cap of the innominate artery occlusion. The occlusion was crossed from the right radial access with a 6F JB1 (Cook)

catheter (through the Shuttle sheath) and a straight stiff glidewire (Terumo). From the radial sheath over a 0.035-inch workhorse wire, the innominate lesion was predilated with a 5.0×20 mm Optapro balloon. Simultaneous kissing stents were then deployed in the innominate [7.0×27 mm LD Express biliary stent (Boston Scientific)] and left common carotid arteries [7.0×19 mm Express SD stent (Boston Scientific)] (Figure 3; Supplemental Figure 2) with a good angiographic result.
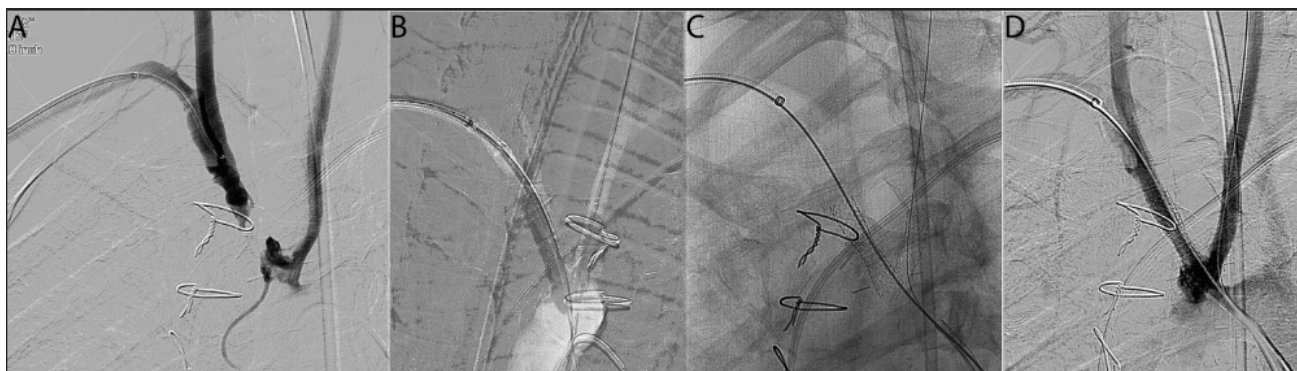

Figure 3. (A) Baseline simultaneous antegrade and retrograde injections demonstrating a short segment of occlusion (arrow) involving the innominate artery. Note the left common carotid origin and 'bovine' configuration. (B,C) Simultaneous kissing stent deployment (arrow) in the innominate and the right common carotid artery. (D) Final angiogram demonstrating no residual stenosis of the stented arteries.

The patient tolerated the procedure well without apparent complications. Post-procedure he had no further episodes of what was felt to be innominate steal syndrome. He was discharged on hospital day 25.

4.   Discussion

The cause of innominate artery disease is most frequently atherosclerosis. Other potential etiologies include Takayasu arteritis, giant cell arteritis, radiation, and fibromuscular dysplasia [11]. Clinical manifestations of innominate steal can include symptoms referable to the posterior circulation (dizziness, vertigo, ataxia, diplopia, bilateral limb weakness), symptoms referable to ICA territory ischemia (e.g., global cerebral hypoperfusion, amaurosis, aphasia, left hemiparesis), or exertional right upper extremity discomfort [6,11]. Some suggest that symptoms of cerebral hypoperfusion in innominate stenosis typically occur in the setting of additional occlusive disease in the cerebral circulation [12]. However, there are small case series of patients with hemispheric symptoms in the setting of innominate stenosis that have demonstrated objective evidence of reduced regional cerebral blood flow (rCBG) by SPECT imaging in the absence of other intra or extra cranial disease [13]. Isolated innominate disease was present in both cases presented above.

The prevalence of clinically significant innominate stenosis is likely low. In 4748 patients with suspected cerebrovascular disease referred for angiography, the prevalence on innominate stenosis was only 2.5% [1]. Of 20,000 patients undergoing duplex ultrasonography in a single institution in Europe, only 3 were found to have hemispheric steal [2]. The prevalence of individuals with innominate steal phenomenon who are symptomatic is likely even lower. The natural history of asymptomatic innominate steal is unknown. However, in the absence of data to the contrary, there exists concern for ischemic watershed stroke in the right cerebral hemisphere in patients with right hemispheric steal syndrome as well as the risk of cerebral embolic events.

The presence of innominate stenosis is suggested by physical exam findings such as diminished right radial and brachial pulses, blood pressure decrement in the right versus left arms, diminished carotid and superficial temporal pulses, and right supraclavicular bruit. CT or magnetic resonance angiography can confirm the presence of stenosis. While typically not useful for imaging below the clavicle, Duplex ultrasonography can characterize the physiologic significance of an innominate stenosis by examining the flow patterns in the right vertebral and carotid arteries.

Partial or complete reversal of flow in the ipsilateral vertebral artery is a hallmark feature of innominate artery stenosis and has been well described in the setting of subclavian steal phenomenon [2]. Spectral Doppler flow patterns in the common, internal, and external carotid arteries distal to a severe innominate stenosis are more variable, likely reflecting the multiple potential collateral pathways for reconstituting the carotid circulation [2,4,15]. If the carotid system is reconstituted via the Circle of Willis, there will be flow reversal in the right internal and external carotid arteries. If the carotid system is reconstituted via the external carotid artery, there will be reversal of flow in the external and common carotid arteries. If the carotid system is reconstituted by the reconstituted subclavian (via the right vertebral artery), the spectral Doppler waveforms will be antegrade but blunted. The patient in case one had Doppler findings consistent with severe steal phenomenon evident by complete flow reversal in the ipsilateral vertebral, common carotid artery (CCA), and internal carotid artery (ICA) while the patient in case two had reversal of flow in the ipsilateral vertebral artery and biphasic flow in the right common carotid artery. The flow patterns of the patient in case one was also clearly demonstrated by selective angiography of the left CCA that showed retrograde flow of contrast in the right ICA.

The majority of patients with innominate artery stenosis have atherosclerotic disease and should receive aggressive secondary prevention measures. Clinical practice guidelines give a Class IIA recommendation (the weight of evidence is in favor of usefulness) for innominate revascularization for symptomatic disease or when an ipsilateral internal mammary artery is needed for coronary bypass based on expert opinion [11].

Revascularization of innominate disease can be performed percutaneously or surgically. In contrast to subclavian artery obstructive disease, which can be treated extra-anatomically, surgical innominate revascularization often requires a transthoracic approach and carries relatively high morbidity and mortality. In 3 studies of 327 patients undergoing transthoracic revascularization for

disease of the great vessels in the 1990's, the combined perioperative stroke and death rate ranged from 8–16% [16–18]. Thus, endovascular or hybrid techniques are favored currently.

There are no randomized trial data and only sparse observational evidence regarding percutaneous innominate revascularization (Table 2). More reports exist that pool innominate and subclavian outcomes. Across several small series summarized in Table 2 that specifically report innominate interventions, the success rate of angioplasty/stenting is typically 90–100%; the periprocedural neurologic event rate is low but not insignificant (TIA 4.3%, stroke 1%), and the patency is fairly good [19–23].

Table 2. Reports since 2000 Focusing on Endovascular Treatment of Innominate Disease.

Study	Year	N	Technical Success (%)	Neurological Complications	Primary Patency
Paukovitz et. al. (19)	2010	77	93.6%	2.6% TIA	86% at 103 months
Van Hattum et. al. (23)	2007	25	83%	4% TIA	79% at 24 months
Huttl et. al. (21)	2002	89	96%	2% CVA 6% TIA	93% at 117 months
Peterson et. al. (22)	2006	8	100%	0	100% at 12 months
Brountzos et. al. (20)	2004	10	100%	1 TIA	90% at 17 months

Several technical aspects make innominate intervention challenging. In particular, the use of distal embolic protection is desirable at least in theory. However, it can be difficult to maintain such a device, designed for placement in the distal cervical internal carotid artery, in the field of view when treating the innominate. Also, appropriately sized stents are often compatible with a 0.035-inch guidewire, whereas distal embolic protection devices are 0.014-inch systems. Another challenge of innominate intervention can be achieving adequate guiding catheter or sheath support in the aortic arch to cross the lesion and deliver equipment. We attempted to obviate some of these issues in the first case by advancing a 0.018-inch wire to the axillary artery to provide extra guiding catheter support while deploying a stent compatible with the wire of the distal embolic protection system. Such a two-wire approach has been reported in the past [24].

A final technical challenge of note is preserving important side branches. In the second case, the occlusion was 'flanked' by the right and left common carotid arteries, respectively. The use of 'kissing' stents in this case protected the left common carotid artery.

Longitudinal follow-up of patients following innominate revascularization is important given the not insignificant restenosis rates and the potential for neurologic symptoms. Physical examination, including bilateral noninvasive arm blood pressures, is critical for detecting restenosis. Likewise, extracranial vertebral and carotid duplex ultrasound can be used to ensure there is antegrade flow in the right vertebral and carotid arteries. There is no evidence to guide the use of antiplatelet agents in this setting. We favor dual antiplatelet therapy for 3 months and indefinite mono-antiplatelet therapy, thereafter.

5.　　Conclusion

Similar to subclavian steal syndrome, innominate steal can involve vertebrobasilar insufficiency but also can be associated with cerebral ischemia in the right internal carotid territory. Surgical revascularization of innominate disease carries higher morbidity and mortality than that for subclavian artery stenosis. Hence, percutaneous revascularization is often favored. We have presented two cases, which illustrate typical clinical presentations of innominate steal and also highlight several technical challenges of endovascular treatment of this disorder.

Conflict of Interest

None of the authors has conflict of interests.

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Supplement Figure Legends

Supplemental Figure 1

(A) Baseline aortic arch angiography demonstrating severe innominate artery stenosis with retrograde flow in the right common carotid artery via collateral vessels. (B) Selective left carotid angiography demonstrating antegrade flow in the left common carotid artery with collateralization and retrograde flow in the right common carotid. (C) Final selective innominate angiography after stenting showing no residual stensois and antegrade flow in the right common carotid artery.

Supplemental Figure 2

(A) Baseline aortic arch angiography demonstrating occlusion of the innominate artery and 50% stenosis of the origin of the right common carotid artery. The right subclavian artery is reconstituted from retrograde flilling of the right vertebral artery with antegrade filling of the right common carotid artery from the reconstituded subclavian. (B) Final angiography demonstrating good angiographic results with antegrade flow in the right common carotid artery.

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