Arteriovenous Fistula Rescue via Endovascular Treatment of Ipsilateral Subclavian Artery Stenosis Using a Retrograde Wiring Approach from the Fistula Access Site

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ABSTRACT: Arteriovenous fistula (AVF) failure in hemodialysis patients due to a low flow state induced by ipsilateral subclavian artery stenosis (SAS) is uncommon. However, when encountered, the SAS can be managed via traditional femoral arterial access and antegrade percutaneous intervention, thereby restoring flow to the AVF. We describe here a unique case where antegrade wiring and intervention of the left SAS was not feasible. Instead, we achieved successful AVF rescue through accessing the fistula, retrograde SAS wiring with externalization of the wire via the femoral artery, and subsequent antegrade SAS angioplasty and stenting.

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Arteriovenous fistulas are the preferred route for hemodialysis access in patients with end-stage renal disease. These surgically constructed fistulas, usually between the radial artery and the cephalic vein, have proven to be the best permanent vascular access for hemodialysis based on durability and low risk of complications when compared to other forms of vascular access.1 Reasons for AVF complications include lack of patency, incomplete maturation, and insufficient flow.2 The fistula flow depends on high arterial volume flow, which should be greater than 200 mL/min to maintain adequate patency and use during dialysis.2 A stenosis in any part of the circuit (arterial inflow or venous outflow) can limit forward flow. While venous problems are the most common reasons for access dysfunction, arterial stenosis at the anastomotic site can occur in a minority (up to 4%) of patients.3,4 Arterial inflow disease can also extend to central arteries, particularly the ipsilateral brachiocephalic or subclavian artery. One study in patients with digital hand ischemic syndrome showed that up to 14% of patients had isolated inflow stenosis proximal to the AVF as the cause of fistula dysfunction.5 This is usually attributable to accelerated atherosclerotic disease, as many patients with an AVF for chronic renal failure also have significant atherosclerotic risk factors. If left untreated, patients can develop symptoms related to a steal phenomenon or progressive AVF failure with an inability to complete dialysis.

CASE REPORT
A 76-year-old female with type II diabetes, hypertension, hyperlipidemia, aspirin allergy, and end-stage renal disease secondary to diabetes and hypertension was receiving dialysis 3 times a week via a right-sided, tunneled permcath. She had a brachiocephalic AVF placed in her left arm in May 2011 with anticipated maturity in July 2011. No preoperative assessment of arterial inflow to the left arm was performed. After July 2011, only a faint thrill was palpable in the AVF and multiple attempts at dialysis through the fistula were unsuccessful. She continued dialysis through her pre-existing permcath and was referred to cardiology for renal transplant evaluation. At this time, she reported symptoms of intermittent arm claudication and chest pain. She denied
any neurologic symptoms, episodes of presyncope, syncope, or heart failure.

On exam, her BP was 140/80 mmHg in the right arm and was not checked in the left arm due to the AVF. A harsh, systolic bruit was noted at the base of the left neck and over the left clavicle. A transthoracic echocardiogram revealed moderate mitral regurgitation and a preserved ejection fraction. A left upper extremity ultrasound suggested a high-grade stenosis in the left subclavian artery and evidence of subclavian steal with retrograde flow in the left vertebral artery. A nuclear exercise stress test revealed severe reversible perfusion defects in the anterolateral and inferolateral territories.

In September 2011, after aspirin desensitization, the patient was brought to the cardiac catheterization laboratory for coronary and left subclavian angiography via right femoral access. The second obtuse marginal and diagonal arteries were severely diseased and treated with drug-eluting stents. Aortic arch angiography revealed type II aortic arch and a subtotally occluded, calcified lesion at the origin of the left subclavian artery (Figure 1). A staged percutaneous intervention of the left subclavian artery was planned to improve symptoms of arm claudication and to rescue the AVF, which was compromised secondary to inflow disease.

Although traditionally an antegrade approach for endovascular treatment of SAS is commonly used, several factors deemed this approach unfavorable in this case. The patient had peripheral vascular disease and aortoiliac tortuosity, which could have led to unfavorable guide catheter support. Coupled with an ostial, calcified, and angulated subclavian lesion, we anticipated difficulty in crossing the near total occlusion of the subclavian artery. Therefore, a modified retrograde approach was planned, where the initial access site would be in the brachial artery inflow segment of the AVF. After successful retrograde wiring of the lesion, the wire would be snared and externalized via the femoral sheath. This would provide adequate wire support to subsequently complete the intervention from an antegrade approach. Femoral access to perform the coronary interventions was gained without difficulty given that the guide catheter was placed leftward around the entire arch and had extra support from the ascending aorta when engaged in the left main artery. In addition, coronary intervention was performed in lesions that were not subtotally occluded and did not have acute angulations, which facilitated easy wiring.

In October 2011, the patient returned to the cardiac catheterization laboratory...
for the subclavian artery intervention. A 6 Fr 10 cm Pinnacle introducer sheath (Terumo Interventional Systems) was placed via the right common femoral artery. Left brachial artery access, adjacent to the inflow segment of the AVF, was achieved using a micropuncture needle and subsequent placement of a 5 Fr 10 cm Glidesheath (Terumo Interventional Systems). A fistulogram through the brachial sheath revealed a patent arterial inflow and venous outflow of the native fistula without any evidence of local disease (Figure 2). The right CFA sheath was then exchanged for a 7 Fr 65 cm Destination sheath (Terumo). After sheath exchange, 3500 units of intra-arterial unfractionated heparin were administered and an activated clot time of 270 was maintained throughout the procedure. A 0.035" x 260 cm Angled Glidewire (Terumo) was advanced through a 0.035" x 135 cm Quick-Cross catheter (Spectranetics) via the brachial sheath to the site of the subclavian lesion, which was successfully crossed in a retrograde fashion. A 35 mm Amplatzer Gooseneck Snare Kit (EV3) was delivered through the femoral sheath and the distal tip of the Angled Glidewire was snared into the femoral Destination guiding sheath. After snaring the wire, the Angled Glidewire was externalized through the femoral sheath and exchanged for a more supportive 0.035" x 300 cm SupraCore wire (Abbott Vascular).

At this point, traditional percutaneous intervention of the left subclavian artery was performed via the femoral antegrade approach over the SupraCore wire. The ostial lesion was predilated with a 5 x 20 mm FoxCross balloon (Abbott Vascular) at 10 atm (Figure 3). Follow-up views revealed a second obstructive lesion in the mid portion of the subclavian artery. The mid lesion was treated with an 8 x 40 mm Zilver 635 self-expanding stent (Cook Medical). The ostial calcified lesion was then treated with a 9 x 25 mm Express Biliary LD balloon-expandable stent (Boston Scientific) at 8 atm. Post-procedural views revealed a widely patent left subclavian artery with brisk flow down the left arm and no further angiographic evidence of retrograde flow in the left vertebral artery (Figure 4).

The patient remained hemodynamically stable throughout the procedure without any complications. Immediate palpation of the left AVF post procedure revealed an easily palpable thrill, which was previously very faint. The next day, the patient was able to undergo successful hemodialysis via the left AVF for the first time since its construction. The patient was maintained on daily aspirin and clopidogrel antiplatelet therapy after the procedure.

**DISCUSSION**

Prior case reports have described significant symptoms related to subclavian stenosis in patients undergoing hemodialysis via an ipsilateral AVF. In a report by Lee et al., a patient had a prior coronary artery bypass graft utilizing a left internal mammary artery. The high flow demand from the poorly functioning AVF caused a coronary and vertebral steal phenomenon leading to extreme symptoms of angina and dizziness. In this report, all symptoms were relieved by percutaneous stenting of the ostial subclavian lesion via a transfemoral approach. The authors noted that brachial or radial approaches to subclavian revascularization were not adopted due to possible trauma and damage to the left arm AVF.

As described in this report, access and retrograde wiring from the brachial segment adjacent to the inflow of the AVF to aid in subclavian revascularization can safely be performed. We elected to access the brachial artery adjacent to the inflow arm of the AVF so that we could complete a fistulogram to ensure patency of the inflow and outflow segments. Although the retrograde procedure is more complex, anatomic barriers and lesion characteristics may prevent revascularization through a transfemoral approach in certain cases. Furthermore,
progressive AVF failure would have been inevitable in this case without endovascular or surgical revascularization.

The complications that can arise from brachial access include the theoretical risk that arterial puncture near the AVF site can lead to iatrogenic stenosis from injury-induced intimal hyperplasia, as well as the potential development of pseudoaneurysms and hematomas at the access site. These concerns are the main reason that transvenous outflow access is preferred over the antegrade inflow brachial access when diagnosing and treating local lesions in native fistulas. The risks of arterial injury can be minimized by using a micropuncture needle for initial access to the brachial artery, a 4 Fr to 5 Fr Glidesheath in the brachial artery and being able to complete the intervention via the 6 Fr to 7 Fr transfemoral approach. We performed the procedure via the 5 Fr brachial sheath, which was used solely to assist with initial wiring of the complex subclavian lesion; however, a 4 Fr system could have been used with 0.014” to 0.018” guidewires and supporting catheters. The second step of the procedure including the angioplasty and stenting of the subclavian lesions were performed via the 7 Fr femoral sheath. The intent of this strategy was to minimize any trauma or arterial complications in any segment of the inflow arm to the AVF. In addition, utilizing the 7 Fr femoral route for the second step of the procedure facilitated accurate positioning of the stent as we were able to achieve better lesion visualization with injection of contrast through a 7 Fr system during stent positioning and deployment.

The alternative treatment for symptomatic subclavian stenosis is surgical endarterectomy or carotid subclavian bypass, which also carries a low morbidity and mortality risk, although the risk is higher than in an endovascular approach. For our case, a surgical approach would have likely carried higher periprocedural risks than an endovascular approach given other comorbidities including coronary artery disease and recent coronary stenting. In general, stenting compared to surgery for treatment of SAS is associated with equivalent long-term patency rates with fewer post-procedural complications; however, careful scrutiny of this literature may slightly favor the surgical approach in terms of long-term patency rates.

While the true incidence of subclavian artery disease in dialysis patients is unknown, this case highlights the importance of peripheral vascular pre-screening prior to AVF placement in this high-risk peripheral arterial disease patient population. This can be accomplished by a careful history and physical examination with subsequent noninvasive imaging studies if clinically indicated. This case also highlights the technical feasibility of a retrograde approach to ipsilateral SAS intervention from the brachial AVF access site.

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