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Subclavian Vein Stenosis/Occlusion Following Transvenous Cardiac Pacemaker and Defibrillator Implantation: Incidence, Pathophysiology and Current Management

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Abstract Subclavian vein stenosis is a common, but usually asymptomatic, complication following cardiac device placement. In addition to reviewing the literature on incidence, pathogenesis and management options for this important clinical problem, we describe two cases of symptomatic subclavian vein occlusion following pacemaker/defibrillator placement and successful treatment with venoplasty and stenting.

Keywords subclavian vein, stenosis, pacemaker, complication, venoplasty

The number of pacemakers and defibrillators implanted in the United States and worldwide continues to increase. In 2009 alone, there were more than 1 million pacemaker insertions or replacements and more than 300,000 defibrillator implants. These numbers continue to grow along with an aging population and expanding indications for device therapy.¹ Subclavian vein stenosis/occlusion following transvenous cardiac device placement is a known complication of the procedure.^{2,3} It is an important clinical problem to recognize, especially with the increasing numbers of pacemaker and defibrillator implantations. Subclavian vein stenosis can lead to symptoms such as ipsilateral arm edema, paresthesia and pain in a minority of patients. Acute thrombosis related to the cardiac device is rare but most often symptomatic. Chronic thrombosis/stenosis is more common but usually asymptomatic.

Subclavian vein stenosis related to an intracardiac device, even when asymptomatic, can lead to problems with lead revision or device exchange.^{2,3} Variable incidence has been reported in the literature, but up to 50% of patients may develop at least moderate subclavian vein stenosis, defined in most studies as a greater than 50% luminal narrowing by contrast venography. Even with more severe luminal narrowing

(greater than 70%), most patients remain asymptomatic due to venous collateral formation.⁴⁻⁸ Risk factors for subclavian vein stenosis have not been well elucidated. Various management strategies have been employed, including anticoagulation, lead extraction, percutaneous venoplasty with or without stenting, and surgical bypass. However, no consensus regarding the optimal treatment of this problem exists, and there is limited evidence to support one approach over another.

Herein we review the literature in an effort to further understand what questions have been answered and areas where further study is important for the management of this unique vascular complication. Also provided are descriptions of two cases of symptomatic subclavian vein occlusion associated with transvenous pacemaker and defibrillator leads that were treated at our institution with successful percutaneous venoplasty and stenting.

Illustrative Case #1 (Figure 1)

A 67-year-old man with a history of idiopathic dilated cardiomyopathy and paroxysmal atrial fibrillation on rivaroxaban anticoagulation (with documented compliance) underwent placement of a left-sided biventricular implantable cardioverter defibrillator (Viva XT, Medtronic Inc., Minneapolis, MN) one year prior to presentation. There was no prior history of clotting disorders or venous thromboembolism. The procedure and immediate postoperative course were without complications.

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Figure 1. *A: Subclavian venography demonstrating total occlusion in the presence of a biventricular implantable cardioverter-defibrillator with collateral formation. B: Balloon venoplasty was performed. C: Significant recoil can be seen on postvenoplasty angiography. D: After stenting the lesion, only minimal residual stenosis is evident.*

One month prior to presentation, the patient saw his primary care physician with a complaint of isolated, painful left arm and hand swelling. The patient also noticed visible venous collaterals over the left shoulder and pectoral area. Upper extremity venous ultrasound raised concern for significant left subclavian vein stenosis or occlusion but was limited in its evaluation due to the presence of existing pacemaker leads and the left clavicle. The patient was advised to undergo further evaluation with a venogram.

Left subclavian venography accessed via the left brachial vein demonstrated a total occlusion of the left subclavian vein near the site of entry of the pacemaker wires. There was difficulty in crossing the lesion from the arm with multiple catheters and wires. Femoral venous access was obtained and an attempt was made to cross from a retrograde approach. Ultimately, using retrograde injections, a support catheter (Quick-Cross®, Spectranetics Corp., Colorado Springs, CO) and stiff guidewire were used to successfully cross the lesion from an antegrade approach. The lesion

Figure 2. *A: Subclavian venography demonstrating total occlusion in the presence of a single-chamber pacemaker. B: Balloon venoplasty. C: Significant recoil is shown on postvenoplasty angiography. D: After stenting the lesion, only minimal residual stenosis is evident.*

was dilated at the lead insertion site with 5×40 -mm and 10×40 -mm balloons, and brisk antegrade flow was restored. However, with the removal of the wire, repeat angiography demonstrated reocclusion due to recoil at the lesion site. After consultation with the patient's electrophysiologist, a decision was made to proceed with stenting with the full understanding that the implanted pacemaker leads would be trapped behind the stent. A 12×60 -mm stent (LifeStar®, Bard Peripheral Vascular Inc., Tempe, AZ) was deployed and postdilated using a 10-mm balloon, with an excellent angiographic result.

Illustrative Case #2 (Figure 2)

A 79-year-old man with a history of hypertension, post-surgical revascularization coronary artery disease and persistent atrial fibrillation on warfarin anticoagulation underwent implantation of a single-chamber pacemaker (Accent™, St. Jude Medical Inc., St. Paul, MN) one year prior to presentation for tachycardia-bradycardia syndrome. There was no history of previous venous thromboembolism or clotting disorder, and the patient

was compliant with anticoagulation. The procedure and immediate postprocedure course were uncomplicated.

Ten months following pacemaker implantation, the patient saw his primary care physician for isolated left arm and hand swelling and development of dilated venous collaterals over the left chest area. A duplex ultrasound was ordered at that time and demonstrated slow flow in the left subclavian vein, raising suspicion of stenosis or occlusion of the subclavian vein. A venogram was recommended.

Left subclavian venography accessed via the left brachial vein demonstrated a total occlusion of the left subclavian vein near the entry site of the pacemaker lead. Using an angled guidewire, we were able to cross the occlusion. Venoplasty was then performed with an 8 × 40-mm balloon with multiple inflations up to 10 atmospheres. An 8 × 40-mm noncompliant balloon was then used up to 14 atmospheres, given significant recoil at the lesion site after initial venoplasty. Because of unsatisfactory flow across the lesion, a 12 × 40-mm stent (LifeStent®, Bard Peripheral Vascular) was deployed and postdilated with an 8-mm noncompliant balloon, with an excellent angiographic result.

At 1-month follow-up, both patients had complete resolution of the arm swelling, and the subcutaneous venous collaterals had completely disappeared.

Pathophysiology

Venous thrombosis and stenosis were reported in the literature shortly after the first transvenous pacemakers were implanted. Early and late venous thrombosis has been described.^{5,6} Late thrombosis is often asymptomatic, the result of chronic fibrosis around the device leads that enables collaterals to form. Acute venous thrombosis is less common and often symptomatic. Most of the modern histopathologic data has focused on the complex inflammatory and fibrotic changes that occur at the endocardial lead interface. Autopsy studies have identified similar inflammatory and fibrotic changes at the insertion site of the device leads in the vein.^{9,10} Dense fibrotic adhesions are routinely encountered when lead removal is necessary and have formed the basis for the development of excimer laser lead extraction.¹¹ This fibrosis can occur more frequently at characteristic anatomic sites like

the lead insertion point, venous bifurcation sites and the costoclavicular space. It is not well characterized why particular anatomic sites are more prone to stenosis, but the cause is likely multifactorial and related to endothelial injury from insertion as well as repetitive mechanical trauma at bifurcation sites or where bony compression may occur. Two studies have suggested that temporary pacemaker insertion prior to permanent implant and prior pacemaker insertion before defibrillator placement were risk factors associated with stenosis, presumably due to repetitive vessel trauma.^{7,12}

Epidemiology

The incidence of subclavian vein stenosis after device implantation varies widely in the literature, ranging from 30% to 50%.^{4-8,12,13} Some of this variation depends on the modality used to diagnose the problem, i.e. ultrasound versus contrast venography. Other reasons for the variation are likely diverse study populations that vary considerably in size, both retrospective and prospective data acquisition and nonstandard definitions of significant stenosis.

Multiple studies in which routine postimplant venography was performed have demonstrated significant stenosis rates. Many studies have defined the degree of stenosis as mild (<50%), moderate (50–70%) and severe (>70%). Antonelli et al.⁵ performed consecutive venography on 40 patients and found significant venous stenosis in 23% at a mean postprocedure follow-up of 4 months. Studies with longer follow-up have reported rates as high as 50%. Da Costa and colleagues⁷ prospectively studied venography at 6 months postimplant in 202 patients. Greater than moderate stenosis was identified in 51% of patients. Most of the patients in these studies were asymptomatic, thought to be secondary to collateral venous circulation that develops as the stenosis gradually worsens. The incidence of symptomatic subclavian vein stenosis in patients with cardiac devices is estimated at 1–5%. However, this may be an underreported clinical problem.

Risk factors for the development of subclavian vein stenosis have been described but are not well characterized. In Da Costa et al.'s study of both symptomatic and asymptomatic patients, low ejection fraction and a prior temporary pacemaker conveyed

increased risk. There are conflicting data on whether the number of leads present conveys greater risk. Other studies have investigated but found no link in a variety of patient- and device-specific factors. These include age, sex, pacemaker or defibrillator device, time from lead implant, lead type or material (silicone or polyurethane) and entry site, among others.^{7,13}

Clinical Characteristics and Diagnosis

Symptomatic patients with chronic lead-related subclavian vein stenosis/occlusion often present more than one month after device implantation with painful arm swelling that may be associated with cyanosis and visible venous collaterals over the ipsilateral chest and upper extremity. Rarely, pulmonary embolism can be a presenting symptom as reported in Da Costa et al.'s prospective study.⁷ The majority of patients remain asymptomatic due to venous collateral formation. Symptoms may develop due to inadequate collaterals or their acute obstruction, an acute thrombotic event on an existing moderate venous stenosis in which collaterals have not formed, or the presence of an ipsilateral arteriovenous fistula in dialysis patients.¹⁴

Diagnosis may be apparent based on history and physical exam alone. Often, a venous duplex ultrasound will identify a high-grade stenosis or occlusion.¹⁵ Computed tomography is an additional noninvasive option when ultrasound is unavailable or inadequate.¹⁶ Definitive diagnosis is made with contrast venography.^{2,12}

Management

The optimal management strategy for the treatment of patients with subclavian vein stenosis related to a transvenous lead has not been systematically studied. Most of the literature has focused on management options for asymptomatic patients with venous stenosis who need an additional lead or lead revision and in whom the diagnosis is made incidentally.¹⁷⁻²² Options for management of these patients include contralateral access, lead extraction with reimplantation or epicardial lead placement. Surgical approaches for bypass or reconstruction exist but are rarely used in the modern era.²³ An alternative option, percutaneous venoplasty, is being increasingly utilized as a safe and effective method to preserve ipsilateral access even in the setting of chronic occlusion. Worley and colleagues²⁴ reported successful venoplasty in 371

of 373 consecutive patients (over an 11-year period) who were found to have significant subclavian stenosis while undergoing lead revision or upgrade.

The optimal management strategy of the symptomatic patient with subclavian stenosis in which the existing lead is functioning normally is not known. Spittell et al.²⁵ reported early success with balloon venoplasty in the treatment of two patients with symptomatic venous stenosis. Conservative options include watchful waiting for collateral vessels to develop. Anticoagulation and thrombolytic therapy also have been utilized, but they are unlikely to be effective for the chronic fibrotic lesions that often exist in these patients.²³ For the persistent symptomatic patient, device extraction can be considered, but this comes with an approximately 1–2% risk of significant morbidity and mortality, including tamponade, valve injury and hemothorax.¹¹ Venoplasty with or without stenting is an alternative approach that enables the device and leads to stay in place, avoiding the risks of lead extraction as well as preserving ipsilateral access. In Worley et al.'s large, retrospective, single-center review, no procedural complications were noted in 373 venoplasty procedures.²⁴

Many reports in the literature describe the successful treatment of symptomatic patients with superior vena cava stenosis due to transvenous leads.²⁶⁻³⁰ Riley et al.²³ performed a pooled analysis of 104 patients treated for lead-associated superior vena cava syndrome over a period of 39 years. The treatment options included anticoagulation (n=29 patients), thrombolysis (n=11), venoplasty (n=16), surgery (n=23) and stenting (n=25). Stenting was the superior modality, with 5% recurrence over a median of 9.5 months. Surgery had a 12% recurrence rate, venoplasty 23%, anticoagulation 21% and thrombolysis 33%. Complication rates were not reported.

Kalman et al.³¹ reported their single-center experience with a small heterogeneous group of patients with symptomatic venous stenosis treated with a variety of interventional techniques, including directed thrombolysis, venoplasty and stenting as a bail-out option. Of the patients in their cohort, 15% had a pacemaker lead and 15% received a stent. They reported a 70% 2-year patency rate for the

entire group, with no complications noted from the stenting technique. The low rate of stent use is in line with the Heart Rhythm Society expert consensus recommendation to avoid stenting in patients with symptomatic pacemaker-related subclavian stenosis.³² There is concern that stenting can damage device leads and lead to pacemaker malfunction, but this has been reported rarely in the literature.³³

Conclusions

Cardiac device-related subclavian vein stenosis is a common problem that most often remains clinically silent. The two patients presented here were both symptomatic, with venography demonstrating subclavian vein occlusion. Both were approximately one year removed from device implant at the onset of symptoms, a common time frame for this occurrence. One had a single-lead pacemaker and the other a biventricular device. It may be that lead burden is only one of many contributing factors in this complication. Interestingly, both patients were on chronic therapeutic anticoagulation for the treatment of atrial fibrillation. The pathophysiology of lead-related venous stenosis may be unrelated to any acute thrombotic event but more likely is a chronic inflammatory and fibrotic reaction driven by endothelial injury and mechanical trauma. No prospective data exist on the benefit of anticoagulation therapy to prevent venous stenosis surrounding cardiac device implantation; this is an area in need of further study.

Both of our patients were treated with venoplasty and stenting due to unsatisfactory results with balloon inflation alone. The long-term outcomes of this approach are not known, and limited data exist for the use of this approach in the subclavian vein. It has been shown to be a reasonable strategy with good short-term outcomes when used in the superior vena cava with retention of device leads.²² The dense fibrosis of these lesions, causing significant recoil, can lead to venoplasty failure. Aggressive balloon inflation in this setting may lead to perforation. Stenting is a safe alternative that improves procedural success and ipsilateral access. The obvious limitation of this technique is a lack of both short- and long-term data from a larger patient population specifically targeting subclavian vein stenosis. We have shown here that it is a safe and effective approach for the nearly immediate

relief of symptoms in two patients. It is important to know it remains an option for the select group of patients with symptomatic subclavian vein stenosis related to an implanted cardiac device.

Patient-Friendly Recap

- Blockage of blood flow in the subclavian vein is common following placement of a cardiac device.
- This complication is occurring more frequently as the overall use of implanted devices increases.
- A combination of venoplasty and stenting may effectively relieve painful symptoms that afflict some patients.

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Conflicts of Interest

None.

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