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Percutaneous Management of Non Malignant Superior Vena Cava Syndrome Using IN.PACT Paclitaxel Coated Balloon

Omer Iftikhar[1], Eileen Gajo[1], Westby Fisher[1], Jonathan Gilbert[1] and Michael H. Salinger[1*]

[1]Department of Cardiology, NorthShore University Health System, USA

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***Corresponding author:** Michael H. Salinger, MD, Department of Cardiology, NorthShore University Health System, USA, Tel no: 847-570-2450, Fax no: 847-570-1865; E-mail: msalinger@northshore.org*

Abstract

Superior Vena Cava (SVC) syndrome is an uncommon but known complication of Implantable Cardioverter Defibrillator (ICD) and pacemaker lead placement. In general 0.2 to 3% of all implants are associated with symptomatic SVC stenosis. Stent placement is a viable option for benign SVC syndrome in the absence of indwelling pacing leads. The risk of perforation and lead fracture increases when large numbers of leads are present chronically. We describe here a case of patient with SVC syndrome secondary to chronic ICD and pacemaker lead placement and a novel use of IN.PACT™ paclitaxel drug coated balloon to treat the SVC obstruction with a favorable clinical result.

Keywords: Interventional cardiology; Balloon venoplasty; Drug coated balloon

Introduction

Superior Vena Cava (SVC) syndrome occurs when there is external or internal obstruction of the SVC prior to its entry into the Right Atrium (RA) [1]. Before the discovery of antibiotics, most of the cases of SVC syndrome were from infections, particularly syphilitic aortic aneurysm and tuberculosis [2]. The syndrome is now more often associated with malignancies, most commonly lung cancer and Non-Hodgkin Lymphoma (NHL) [3].

With increasing use of pacemakers and defibrillators, the benign causes of SVC syndrome are increasing. Benign causes of SVC syndrome account for about 20-30% cases of the disease [4]. In general 0.2 to 3% of all implants including pacemakers, Implantable Cardioverter Defibrillator (ICD) and Biventricular pacemakers are associated with symptomatic SVC stenosis [5]. Silent venous thrombosis, without any clinical symptoms, is found on routine imaging in as many as 30% of all the implants [6]. We describe here a case of patient with markedly symptomatic SVC syndrome secondary to chronic ICD and pacemaker lead placement and a novel use of IN.PACT™ paclitaxel-coated balloon to dilate the SVC obstruction.

Case Presentation

74 year old female with a past medical history of coronary artery disease (previous myocardial infarction at age 31) sustained ventricular tachycardia with cardiac arrest requiring ICD placement, paroxysmal atrial fibrillation, remote pulmonary embolism and ischemic cardiomyopathy with Ejection Fraction (EF) of 13% presented with complaints of facial swelling, periorbital edema, and right arm swelling. About 5 months prior, the patient had her ICD upgraded to a biventricular ICD given her significantly reduced EF and New York Heart Association (NYHA) class IV symptoms of heart failure. At the time, the decision was made to forego removal of her previous ICD wires given that she had the leads in for a significant amount of time and removal would be a complex procedure with significant risk of vessel or cardiac perforation. No stenosis or thrombosis of the SVC or subclavian veins was found on the venogram done at the time of the previous device upgrade. The patient tolerated the pacemaker upgrade procedure well and she was discharged to home the same day. Months later, the patient started having complaints of facial swelling, head pressure and upper extremity swelling with intermittent throat tightness. She initially saw her primary care physician and was given steroids and her sacubitril/valsartan Entresto®) was stopped as the symptoms were thought to be a reaction to the drug. After several weeks, discontinuation of Entresto and allergy treatments did not alleviate her symptoms. Other relevant tests that were obtained included chest X-ray that showed mild cardiomegaly and presence of pacemaker leads in the SVC (Figure 1).

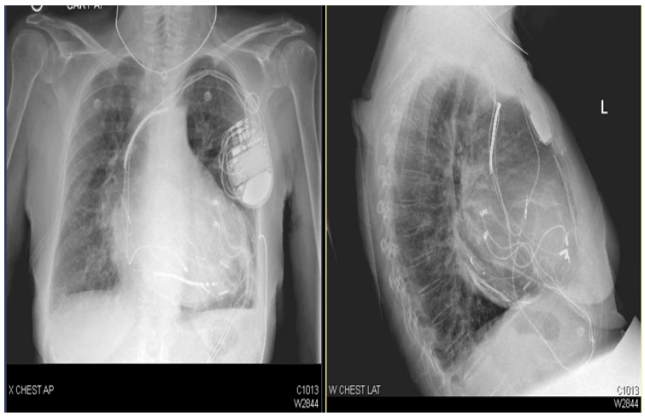

Figure 1: Chest X-ray (Anteroposterior and lateral view) showing location of the multiple transvenous leads in the SVC and right atrium

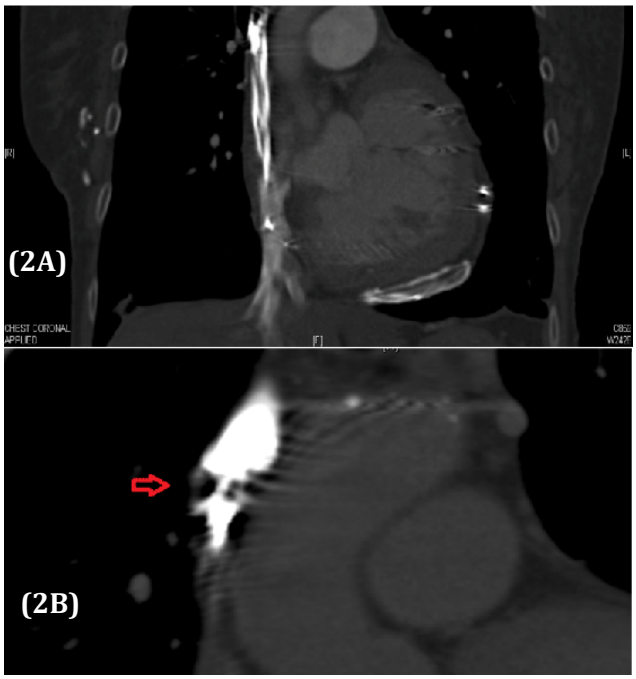

Figure 2: (A) Non contrast coronal section of the CT chest showing multiple pacemaker leads at the SVC and right atrium and (B) contrast coronal imaging demonstrating high grade stenosis or occlusion at the RA/SVC junction with lead artifact making distinction between high grade stenosis and occlusion difficult

A transthoracic echocardiogram (TTE) showed pacing leads in the right heart chamber without any evidence of thrombus in the right atrium. Computed tomography (CT) of the chest was also obtained which demonstrated the multiple leads and a high grade narrowing or obstruction of the SVC as it entered the right atrium (Figure 2a and 2b). Collateral flow was demonstrated into the azygous and hemiazygous systems of veins. Ultimately, lead extraction and stenting were felt to represent excessive risk due to her limited cardiac reserve. The decision was made to proceed with attempted balloon dilatation of the SVC using a paclitaxel coated balloon.

The Procedure

Needle entry of the right internal jugular (IJ) vein with a 5 French introducer was accomplished without difficulty using ultrasound guidance and micropunture technique. Right IJ venous pressures were recorded and selective SVC venography performed. Initial SVC pressure was 26 mmHg. The SVC was shown to be 100% occluded just below the level of the left subclavian vein. The left subclavian vein was patent with very large intrathoracic collaterals presumed to be superior intercostal/hemiazygous veins (Figure 3a). After review of the venography, percutaneous transluminal venoplasty (PTV) of the occluded SVC was performed. The obstruction was crossed using a 0.035-angled hydrophilic guide wire via a 4F multipurpose catheter. The 4F catheter was then advanced to the RA and after confirming appropriate intra-atrial position angiographically, was used to exchange for a 0.035 extra support guide wire. The 4F system was exchanged over the extra support guide wire for a 23 cm long 7F sheath, which was positioned in the mid RA. A 4mm x 6 cm Mustang balloon was then advanced via the sheath and guide wire. After positioning the sheath more proximally the area of obstruction in the SVC was dilated with the 4 mm balloon to a maximal pressure of 6 atm with resolution of deformity on the balloon. Because of tapering present at the tip of sheath, the technique of advancing the sheath beyond the obstruction to deliver the balloon was chosen in an attempt to minimize risk of distal embolization. A 7 mm X 80 cm IN.PACT Paclitaxal eluting balloon was then positioned across the lesion and inflated at 8 atm pressure (Figure 3b). A 10 x 4 Opti Pro balloon was then used to perform serial overlapping 5 and 10 min inflations at 4 atm attempting to modify the potential for elastic recoil and relying on the effects of the drug coated balloon to reduce post dilatation intimal fibrosis. Post dilatation, SVC pressure was reduced to 13 mmHg and the stenosis was reduced to 50% with brisk anterograde flow (Figure 3c). The procedure was well tolerated and there were no complications or evidence clinically of distal embolization. The right IJ sheath was removed and pressure applied to achieve hemostasis. Estimated blood loss was less than 50 ml.

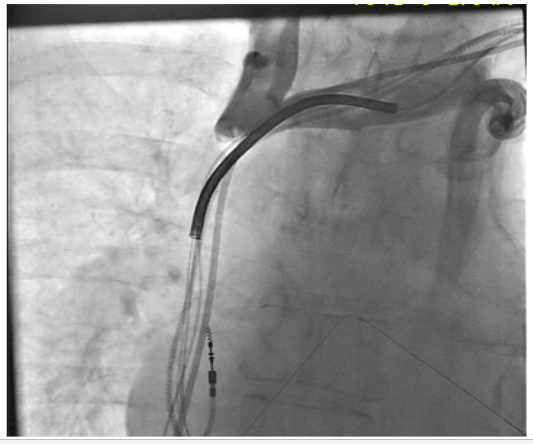

Figure 3a: Venography showing occluded SVC and venous drainage through the hemi-azygous system of veins

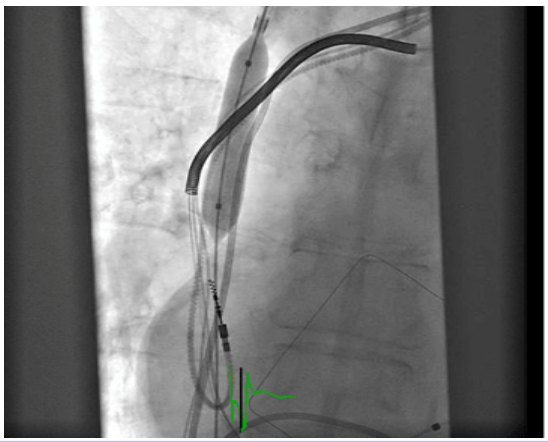

Figure 3b: Percutaneous transluminal venoplasty of the occluded SVC using IN.PACT Paclitaxal eluting balloon

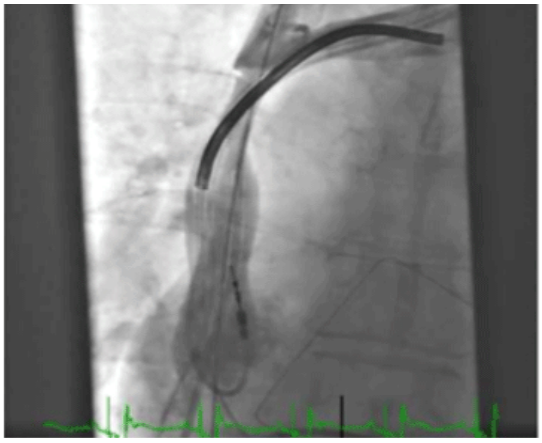

Figure 3c: Venography post PTV demonstrated restored patency with a moderate residual stenosis and normal anterograde flow

The patient was admitted for monitoring after her procedure. She was given a clopidogrel load of 600 mg prior to the procedure. Heparin bridging was used until she reached a goal therapeutic INR range between 2.0-3.0 on Coumadin. Total length of stay in the hospital was 3 days. As per the manufacturer's recommendation the patient was put on dual antiplatelet therapy with aspirin and clopidogrel for one month. Coumadin was chosen as anti-coagulation for her atrial fibrillation because of possibility of reversal in case of uncontrolled bleeding. In addition, after res-establishing antegrade flow venography appeared to demonstrated residual thrombus at the treatment site. After 1 month, clopidogrel and Coumadin were discontinued, aspirin was continued and rivaroxaban was started, as was the sacubitril/valsartan. She has been followed clinically and has had no complaints or neck or arm swelling and unchanged normal pacemaker function now 8 months post therapy.

Discussion

Permanent cardiac pacing and ICD placements are relatively simple and benign procedures that have results in thousands of lives being saved and improvement of symptoms in a large number of patients. The procedure is however associated with a number of complications including infections, pneumothorax and thrombosis. SVC syndrome is a known complication of the procedure that occurs due to fibrosis caused by the pacemaker leads. The greater the number of leads, the greater the chance of having significant fibrosis and subsequent SVC obstruction [7].

Treatment options for SVC syndrome include steroids, radiation therapy, chemotherapy, anticoagulants, stenting and surgical repair depending on the etiology of the obstruction. Interventional techniques preferred for malignancy associated SVC syndrome includes stenting whereas open surgical repair is reserved for patients with recurrent severe symptoms. Overall recurrence rates with SVC stents range from 0 to 40%; but in most cases, patency is restored with re-intervention [8]. In case of pacemaker leads associated SVC syndrome, stent placement and balloon venoplasty are viable options. The limitation of traditional balloon venoplasty is a high recurrence rate [9]. Our institution has had limited but favorable outcomes with the use of Drug Coated Balloon (DCB) therapy in juxtacardiac lesions such as recurrent pulmonary venous stenosis [10]. Based on our past experience and risk of lead fracture from stent placement over the indwelling leads and the risks of attempted chronic multiple lead extraction, the decision was made to approach treatment in this patient using the IN.PACT™ Admiral Drug-Coated Balloon (DCB). Our patient presented with facial swelling and peri-orbital edema while on rixaroxaban for her atrial fibrillation which was considered a treatment failure for medical therapy, hence the decision was made to pursue DCB balloon therapy versus conservative management with anticoagulation. This is a primary endovascular therapy whose main indication is for angioplasty after pre-dilatation of de novo or restenotic lesions up to 180 mm in length in superficial femoral or popliteal arterial disease. The paclitaxel coating on the balloon causes inhibition of neo-intimal growth through stabilization of microtubules by preventing their depolymerization during final G2/M phases of cell division. The overall effect increases blood flow and reduces thickening of the artery wall by delivering the drug to the vessel wall. Favorable outcomes have been seen in SFA lesions treated with IN.PACT balloon vs conventional PTCA. In the IN.PACT SFA trial's 24-month results, DCB showed significantly higher primary patency when compared with PTA (78.9% vs 50.1%; $p < 0.001$). The rates of clinically directed target vessel revascularization (CD-TLR) were 9.1% and 28.3% ($p < 0.001$) for the DCB and PTA groups, respectively. The rate of vessel thrombosis was low (1.5% DCB vs. 3.8% PTA; $p = 0.243$), with no new events reported between 1 and 2 years [11]. The use of drug-coated balloons on the venous circulation has also been studied. The smooth muscle cells in the veins are more responsive to effects of anti-proliferative agents as compared to their arterial counterparts [12]. A recent randomized controlled trial showed improved patency of failing hemodialysis vascular access when paclitaxel coated balloon angioplasty was used for vascular stenosis with cumulative target lesion primary patency of 70% with drug coated balloon versus 25% with conventional balloon angioplasty (HR 0.30, 95% CI 0.12 to 0.71, $p < 0.006$) [13]. Massmann et al. retrospectively reviewed 27 patients with hemodialysis fistula associated central venous stenosis and found that paclitaxel coated balloon angioplasty showed statistically significant longer freedom period from target vessel revascularization compared to conventional balloon angioplasty [14].

To our knowledge this is the first case of benign SVC syndrome secondary to chronic lead placement that has been treated with IN.PACT drug coated balloon. To date, now 8 months post procedure, the patient remains free of any reoccurrence of signs and symptoms of SVC syndrome and has continued normal pacemaker lead function. Long term follow-up and further studies are needed to evaluate the role of IN.PACT balloon in the treatment of benign SVC syndrome secondary to lead placement.

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