

# Extensive Venous Obstruction Caused By a Permanent Pacemaker Lead: A Case Report

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## Introduction

The venous approach is the most common method for permanent pacemaker lead implantation, because of its ease and safety (1). Venous thrombosis and stenosis at the implantation site are the most common complications with the incidence varying between 30-45% (2-4). It is a late complication, but rarely it may occur very early (5). Stenosis and thrombosis of the superior vena cava (SVC) are severe life-threatening complications. Although generally remaining silent clinically, sometimes venous thrombosis causes life-threatening complications, one of which is pulmonary embolism (PE) (6).

This report describes a case with pacemaker lead induced SVC syndrome, that was very extensive and complicated with PE.

## Case Report

The patient was an 80-year-old man with a history of coronary artery disease. He had received an internal mammary graft to his left anterior descending coronary artery in 1999. His past medical history revealed nothing but a bladder tumor treated by radiotherapy in 1996. He had received a left pectoral single lead permanent VDD pacemaker due to syncope complete atrioventricular block in 2001.

The patient was admitted to the hospital with a 1 month history of progressive dyspnea and significant worsening of his clinical status. During his first examination, he was dyspneic and cyanotic. His face, neck, arms and upper chest were swollen. Engorged venous collaterals were apparent in his upper chest. The pacemaker pocket, the generator itself and lead functions were normal. Computed tomographic imaging of the thorax disclosed occluded SVC with extensive venous collaterals but no external mass producing lesion. He underwent digital subtraction angi-

ography which showed total occlusion of the subclavian vein, the brachiocephalic trunk and the SVC with extensive venous collateral formation (Fig 1-2). The venous system of the neck was evaluated by Doppler ultrasound which disclosed a totally occluded right and 90% occluded left internal jugular veins. On admission, the coagulation status was normal. Heparin was started in a patient to maintain an aPTT level twice the control value. Increasing symptoms of dyspnea suggested preceding episodes of PE that was confirmed by multiple perfusion defects on a lung scan. Despite his advanced age, intravenous streptokinase infusion was started due to the extensive thrombosis and deteriorating clinical course under heparin treatment. But the drug had to be stopped at the fifteenth minute of infusion when he developed serious dyspnea, bronchospasm, aphasia and neurologic deficits. The cranial computerized tomography (CT) was normal and the symptoms recovered promptly. He was kept on heparin and started on warfarin. Within 10 days, his symptoms and findings improved considerably. A control Doppler ultrasonography of the neck revealed partial resolution in both jugular veins. Upon considerable clinical stabilization, he was discharged on warfarin.

## Discussion

Permanent pacemaker lead induced venous complications are common. Superior vena cava syndrome is a life-threatening venous complication, fortunately it occurs rarely (7). The pathogenesis of thrombosis after implantation of a permanent pacemaker is not clear. Without stenosis, pacemaker lead induced thrombosis tends to occur early, usually within the first year. When venous thrombosis occurs more than 1 year after implantation of a permanent transvenous pacemaker it is usually associated with venous stenosis (8). In the stenotic venous area

the venous collaterals decrease the blood flow rate which may predispose the patient to thrombus formation (9). Most patients with chronic venous thrombosis remain asymptomatic because the collaterals provide adequate venous drainage. Symptomatic pacemaker induced venous thrombosis is usually associated with acute venous thrombosis or occlusion of venous collaterals. The initial therapy for early pacemaker induced venous occlusion is intravenous administration of heparin and warfarin. Thrombolytic therapy has been used for the initial management of lead induced acute thrombosis. Thrombolysis may be successful when initiated within 3 weeks of symptom onset (10). Streptokinase and recombinant tissue plasminogen activator have been shown to be successful in the dissolution of thrombosis associated with transvenous pacing leads and in the treatment of superior vena cava obstruction (11,12). Heparin alone appears to be effective only in the mildest cases (13). Long-term warfarin usually lifelong is generally advocated in any patient who has had pacemaker-associated thrombosis (10,14,15). The extensive thrombosis in our case seems to have responded to an anticoagulant regimen of heparin and warfarin, at least initially.

The initial treatment is anticoagulation and/or thrombolysis. If these fail to clear thrombosis, the other options are surgery, venoplasty or stenting. Chia et al. (16) described a case of SVC obstruction due to previous pacemaker leads, bypassed using the intact native azygous vein. Many clinical studies described bypass conduits. Inoue et al. (7) described pacemaker lead induced left innominate vein thrombosis that produced SVC syndrome and it was successfully treated using a spiral saphenous vein graft bet-

ween the left internal jugular vein and right atrium. First Chiu et al. (17) described a case with SVC syndrome and it was reconstructed using a spiral vein graft in 1974. In the recent report Doty et al. (18) described a case using a spiral vein graft and provided good long-term patency of 90% during follow-up. Among many types of conduits that have been utilized, autogenous vein grafts have been reported to yield the best long-term patency profile (19). But the surgical treatment of the permanent pacemaker lead induced SVC syndrome requires thoracotomy which is the major disadvantage of the method. Venoplasty is the other therapeutic option for thrombosis of the SVC (19-20). Most of the described cases of SVC syndrome after permanent pacemaker implantation have been due to thrombosis of the SVC that occurred between 1 and 15 months after the procedure implantation (21). Kastner et al. (22) reported a case of SVC syndrome treated with balloon venoplasty with a 6 month angiographic patency and they think that stenting can be reserved for a failed balloon venoplasty. Patency rates of angioplasty alone have not been compared to stenting for the treatment of venous thrombosis. Many series reported better early and intermediate results with stenting (23-25). Chan et al. (26) described a case of percutaneous treatment of pacemaker associated SVC syndrome. In their article they used Excimer laser for pacemaker lead extraction with further venous dilatation and stent placement, and new pacemaker lead implantation through same venous access. But long-term results of percutaneous intervention in this setting is still not clear. Owing to patient's old age, associated medical problems (in particular the renal insufficiency) and impressive response to anticoagu-



**Figure 1-2. Digital subtraction angiography findings of total occlusion of the subclavian vein, the brachiocephalic trunk and the SVC with extensive venous collateral formation**

lant therapy, in our case, we used neither surgery nor percutaneous intervention as the therapeutic modality.

In conclusion, SVC obstruction with transvenous pacing leads is unusual, however it can cause significant morbidity and mortality. Anticoagulation is the mainstay treatment but surgical and percutaneous interventional approaches have to be kept in mind.

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