CASE REPORT

Stent implantation for the treatment of superior vena cava syndrome related to pacemaker leads

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This is a case-report of two patients with superior vena cava syndrome related to pacemaker leads. Both patients were treated successfully using intravenous stenting.

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Introduction

Severe stenosis or even occlusion of the superior vena cava is an uncommon complication of permanent transvenous pacing[1,2]. This complication is related to thrombosis or fibrosis around the pacemaker leads resulting in superior vena cava syndrome (SVCS). The prevalence of this syndrome has been estimated between 1 per 1000[1] and 1 per 40 000[2] patients with implanted pacemakers. Since the original description of this pacemaker-related syndrome by Wertheimer et al. in 1973[3], no more than 50 cases of SVCS have been reported.

It has been suggested that patients with at least two pacing leads are more than three-fold prone to develop this complication when compared with patients implanted with only one lead[4,5]. As the natural history of SVCS results in death, several treatments have been proposed, including anticogulation[2,6], surgical resection of the thrombotic and fibrous tissue[7], local and systemic thrombolysis[8] but rarely angioplasty with stent[9]. Among 1500 patients implanted in our centre over the last 10 years, We found two patients with SVCS who were successfully treated with venous stenting.

Case 1

A 42-year-old woman with very symptomatic sick sinus syndrome underwent a first pacemaker implantation using an endocardial ventricular lead when she was 26 years old. Lead replacement was performed 7 years later for lead dysfunction. At the age of 40, due to battery depletion, the first pacemaker was replaced. Two months later the generator was extruded. A new pacemaker was then implanted via the right subclavian vein. However, the previous lead introduced through the left cephalic vein could not be extracted by traction and was finally left in situ. Subsequent progress was event-free and without infection. Two years later, the patient had exertional breathlessness and bloating of the head and neck associated with gross distension of the neck veins leading to the diagnosis of SVCS. Venography showed occlusion of the right subclavian vein and a severe stenosis of the proximal superior vena cave. An incomplete dilatation of the stenosis was obtained by inflating a 10 mm long and 10 mm diameter Meditech balloon (Boston Scientific Corporation, U.S.A.) at eight atmospheres inserted via the left femoral vein. An 18 mm long Palmaz stent P188 (Johnson & Johnson Interventional Systems, New Jersey, U.S.A.) was deployed within the stenosis with a 10 mm balloon diameter at eight atmospheres in the lower SVC. An overlapping 30 mm long Palmaz stent P308 implanted at seven atmospheres in the lower SVC using a 14 mm diameter XXL-Meditech balloon extending to the right atrial junction. Both stents were further dilated to 14 mm with the previous
balloon. The post-procedure antithrombotic regimen consisted in 160 mg of aspirin, 500 mg of ticlopidine and 0.4 ml of low molecular weight heparin for 1 month. After 5 years of follow-up, the patient remained totally asymptomatic.

Case 2

An 82-year-old man was admitted in April 2000 with a 3 week history of progressive SVCS. In 1992, the patient had undergone a dual chamber permanent pacemaker implantation through the right subclavian vein for syncope due to paroxysmal infra-Hisian block. Five years later, pacemaker dysfunction was detected. During device replacement, attempts made to remove the atrial lead using different extraction techniques (loop snare, Dotter extraction kit) were unsuccessful and therefore the atrial lead was left in place. A new single lead VDD pacemaker was implanted via the left subclavian vein. In March 2000 he developed progressive swelling of the face, neck, arms and upper chest. Superior vena cavo-angiography through the left femoral vein, demonstrated an occlusion of the left subclavian vein and a sub-total occlusion of the innominate vein. Five days later angioplasty was undertaken. The new venogram revealed complete occlusion of the innominate vein with extensive clot formation behind the occlusion. Through the right femoral vein, a 6-F catheter was advanced into the innominate vein through the occlusion. A bolus of 5000 units of heparin was administered locally, followed by an infusion of 200,000 units of urokinase. A large amount of clot remained in the vessels. A 14 mm diameter balloon XXL-Meditech balloon (Boston Scientific Corporation, U.S.A.) was advanced retrogradely through the right femoral vein between the innominate vein and the SVC where three inflations at six atmospheres were performed. Only incomplete dilation of the stenosis was obtained. A 39 mm long and 10 mm diameter Wallstent (Boston Scientific Corporation, U.S.A.) was deployed within the stenosis. A new inflation with the 14 mm diameter balloon was then performed in the stent with a significant improvement in the vein luminal diameter. The pacing leads were fixed between the vein wall and the stent, without damage. Within 24 h of the procedure, the head and neck congestion had resolved. The post-procedure antithrombotic regimen consisted of 160 mg of aspirin, 500 mg of ticlopidine and 0.4 ml of heparin for 1 month. One year after the procedure, the patient was asymptomatic and the pacemaker functioned normally.

The main result of our study is that stenting of the venous system is a feasible treatment for pacemaker lead-related superior vena cava syndrome.

Partial or complete venous occlusion may occur in up to 30–50% of patients after transvenous pacemaker implantation[11]. However, as the formation of thrombus and fibrosis is usually progressive, it allows development of an adequate venous collateral circulation, explaining why SVCS is rare. Early obstruction usually results from thrombosis in the absence of stenosis, later occlusion may be the result of thrombosis at a site of stenosis[7]. But isolated stenosis without thrombosis has been described[9,10].

Predisposing factors for the development of SVCS may be the presence of more than one electrode[2,5]. Mazetti et al.[1] suggested that abandoned leads whether intact or damaged are not the main cause of SVCS but more probably the difficulties in the implanting procedure. It has also been hypothesized that individual reactions to endothelial irritation may play a role[4].

Infection is a potential trigger of venous thrombosis through inflammatory involvement of the vascular endothelium but is present in only 30% of cases[4].

In SVCS related to pacemaker leads, treatment has been reported to be effective in 88% of cases but one

Figure 1  Venous angiography in the anteroposterior view of patient number 1. (A): Before stenting, a stenosis of superior vena cava is present. (B): The result after stenting.
death has been reported\[12\]. The proposed treatments are thrombolytic agents alone or in combination with surgery\[7,8\]; heparin alone or in association with thrombolysis\[6\]; angioplasty alone \[13\] or associated with thrombolysis and surgery\[4,7,14\].

Long-term efficacy of balloon dilatation of venous stenosis related to pacing electrodes is unknown as only few cases have been reported. However, patency rates as low as 30% at 1 year have been reported in our series of balloon dilatation of benign axillary and subclavian vein stenosis\[16\]. To avoid restenosis, the use of venous stenting in this condition has been described for the first time by Lindsay et al.\[9\]. The safety of this procedure has been demonstrated in patients with SVCS due to benign or malignant compression of the vena cava\[17,18\] with good results. In the presence of obvious thrombosis associated with underlying venous stenosis thrombolytic therapy in combination with balloon dilatation and stenting can be considered. Only four cases of pacing lead related venous stenosis treated by venoplasty and stenting have been reported in the literature and are summarized in Table 1.

In the setting of SVCS related to pacemaker leads, stenting of the venous system may theoretically induce three serious events:

1. The lead fracture either immediately or more probably after a few months as a consequence of the erosion of the lead by the rigid surface of the stent. After 5 years in our first case and 1 year in the second, this event had not occurred and has not been reported in the previous series. Stents without protruding tines seem to be a good choice in order to avoid electrode damage.

2. Inability to remove the leads incorporated between the stent and the vein in case of dysfunction or more importantly infection is a serious issue. This complication has not yet been reported but should be kept in mind as its occurrence would necessitate a difficult surgical operation.

### Table 1  Review of some published cases of venous stenting in patients with superior vena cava syndrome related to pacemaker leads

<table>
<thead>
<tr>
<th>Number of leads</th>
<th>Infection</th>
<th>Stent</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lindsay et al. 1994[9]</td>
<td>3</td>
<td>+ 16 mm diam. Wallstent</td>
<td>6 months</td>
</tr>
<tr>
<td>Francis et al. 1995[10]</td>
<td>2</td>
<td>– 16 mm diam.–60 mm long Wallstent</td>
<td>6 months</td>
</tr>
<tr>
<td>Rosenthal et al. 1996[19]</td>
<td>2</td>
<td>– 16 mm diam.–30 mm long Palmaz</td>
<td>6 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>18 mm diam–40 mm long Palmaz</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>18 mm diam–50 mm long Palmaz</td>
<td></td>
</tr>
<tr>
<td>Melikian et al.[20]</td>
<td>2</td>
<td>– 16 mm diam.–56 mm long Wallstent</td>
<td>3 months</td>
</tr>
<tr>
<td>Personal Case 1</td>
<td>3</td>
<td>– 14 mm diam.–10 mm long Palmaz</td>
<td>60 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>14 mm diam–30 mm long Palmaz</td>
<td></td>
</tr>
<tr>
<td>Case 2</td>
<td>2</td>
<td>– 14 mm diam.–39 mm long Wallstent</td>
<td>6 months</td>
</tr>
</tbody>
</table>

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Figure 2  Venous angiography in the right anterior oblique view of patient number 2. (A): Before stenting, superior vena cava is occluded with the presence of thrombi. (B): After stenting, superior vena cava is widely patent with presence of small thrombi in the innominate vein.

Table 1  Review of some published cases of venous stenting in patients with superior vena cava syndrome related to pacemaker leads
(3) Stent thrombosis: Lindsay et al. reported thrombus within the stented portion of the superior vena cava 7 days later, due to inadequate antiplatelet therapy illustrating that scrupulous attention to antithrombotic regimens is required to maintain patency. These possible complications might explain the apparent reluctance of authors to treat SVCS with stenting. However, our results could help to reassure physicians concerning the use of this procedure.

In conclusion, superior vena cava syndrome related to a pacemaker lead is rare but causes distressing symptoms. Intravenous stents can be placed safely in the presence of venous pacing leads but require adequate adjuvant antithrombotic therapy.

References