CASE REPORTS

Successful treatment by balloon venoplasty and stent insertion of obstruction of the superior vena cava by an endocardial pacemaker lead

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Abstract

A 63 year old man with symptomatic obstruction of the superior vena cava associated with an indwelling pacemaker was successfully treated with balloon venoplasty and stent insertion. He was symptom free with normal pacemaker function nine months later.

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Venous thrombosis and stenosis secondary to transvenous pacemaker leads is common. Clinical manifestations of such complications are unusual and the superior vena caval syndrome is rare. We describe a patient who presented with superior vena caval syndrome and in whom treatment with balloon venoplasty and stent insertion successfully relieved the clinical signs and symptoms.

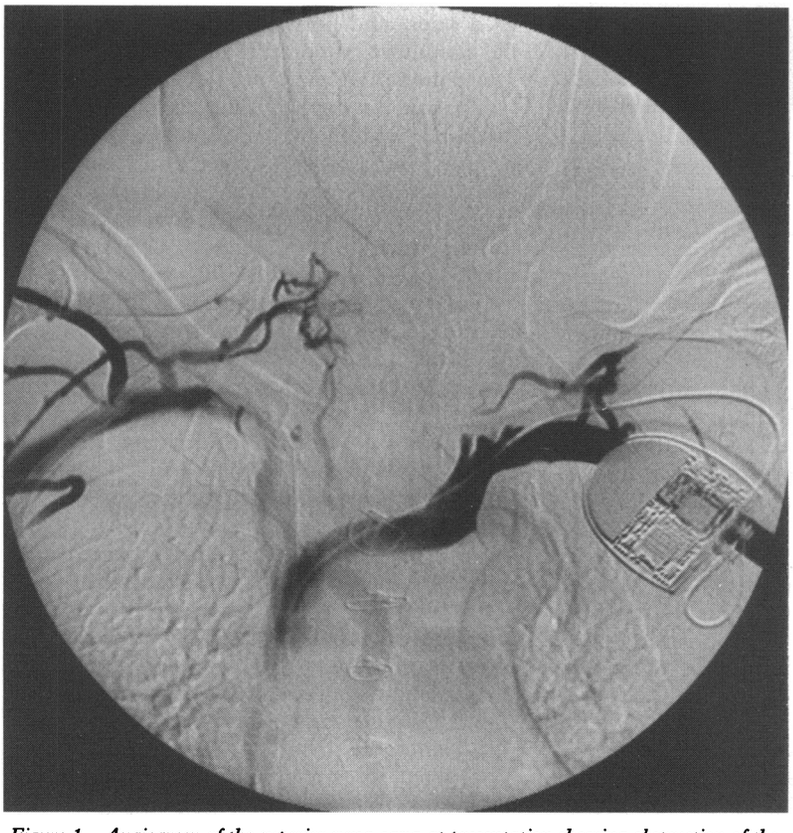

Figure 1 Angiogram of the superior vena cava at presentation showing obstruction of the superior vena cava and dilatation of the azygos vein.

Case report

A 63 year old man presented to the pacemaker clinic for routine review. He volunteered a two week history of sudden onset of facial swelling, headache, snoring, and swollen eyelids. On examination he had signs of obstruction of the superior vena cava.

He had initially presented in 1980 with paroxysmal atrial fibrillation. Antiarrhythmic therapy caused symptomatic bradycardia and in 1983 a VVI pacemaker was implanted through the right subclavian vein. In 1985 he underwent coronary artery bypass grafting for symptomatic three vessel coronary disease. A rash that developed postoperatively was attributed to aspirin. He continued to complain of paroxysmal palpitation and in 1989 was referred to another centre where his pacemaker was upgraded to a dual chamber system that used the original ventricular lead. This system became infected and was explanted. The ventricular lead could not be removed and was capped and buried. An AAIR system was placed through the left subclavian vein.

At admission the results of a full blood count and a coagulation and thrombophilia screen were normal. The chest *x* ray showed the pacemaker on the left side of the chest with the wire entering the left subclavian vein. The right-sided ventricular wire was still in place with its proximal end cut. The aorta was not folded but otherwise the mediastinal contour was within normal limits.

Angiography of the superior vena cava after simultaneous injection of contrast into the veins in both arms showed stenoses in both subclavian veins at the sites of wire insertion. The stenosis on the right was severe. The superior vena cava was patent but tightly stenotic and its appearance suggested the presence of thrombus. A distended azygos vein was readily opacified indicating considerable obstruction of the superior vena cava (fig 1). Computed tomography did not show any mediastinal mass that could be causing extrinsic compression.

The patient was given intravenous streptokinase and treatment with intravenous heparin was started. His symptoms settled and angiography of the superior vena cava was repeated. There was a web-like stricture within the superior vena cava just beneath the level of the azygos vein and beyond this the

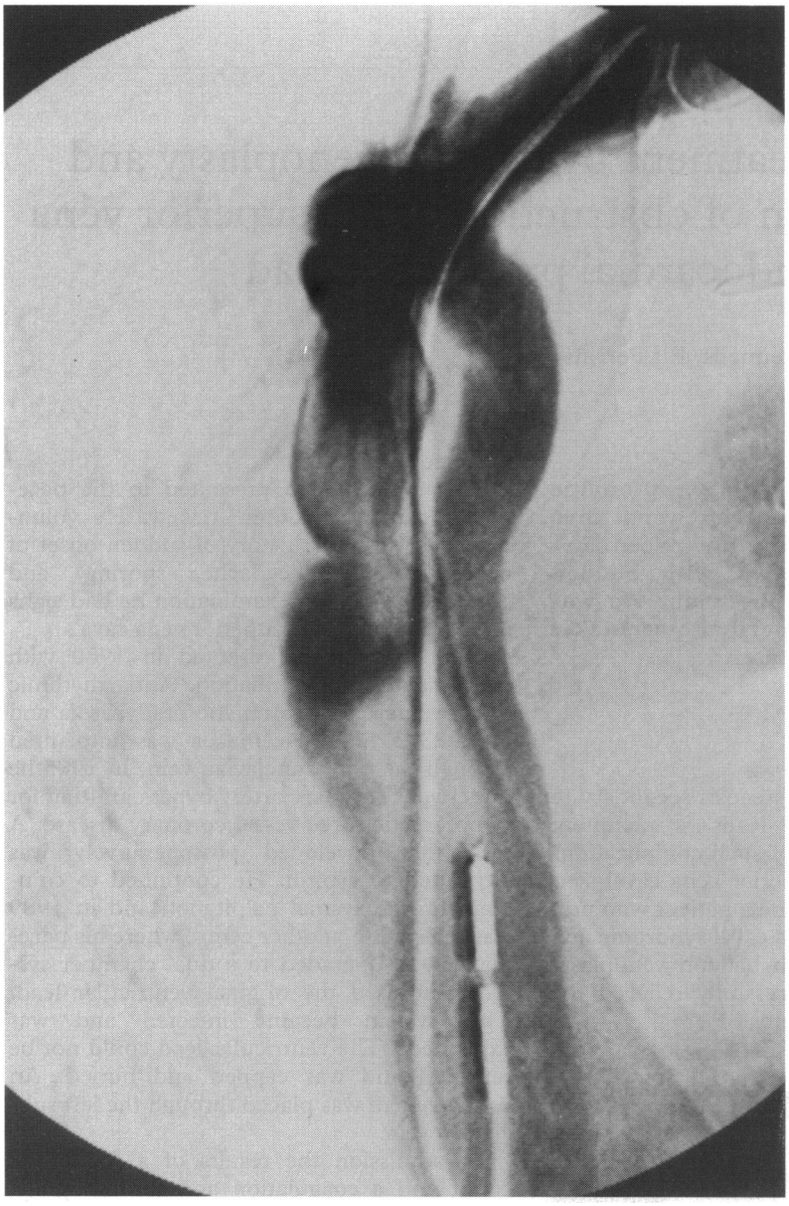

Figure 2 Angiogram of the superior vena cava after thrombolysis, showing a fibrotic web below the azygos vein and a pacing wire lying outside the lumen of the superior vena cava at the level of the stenosis.

superior vena cava was irregularly stenotic as far as the right atrium (fig 2). We dilated the diseased segment of superior vena cava with a 10 mm balloon catheter introduced from the right femoral vein. A 16 mm self-expanding metallic stent (Wallstent, Medivent) was then deployed across the dilated segment from the right atrial orifice to the level of the azygos vein. The patient was pre-treated with dextran, dipyridamole, and heparin. Postoperatively treatment with intravenous heparin was continued and treatment with warfarin was started. Aspirin was not used during the pre and post stent regimens because of the history of allergy.

The patient was readmitted seven days after discharge with a recurrence of symptoms. Angiography of the superior vena cava was repeated and showed thrombus within the stented portion of the superior vena cava. The effects of warfarin were reversed with vitamin K to allow administration of alteplase to lyse the clot. After thrombolysis treatment with heparin and warfarin was re-started. Inadequate antiplatelet therapy was thought to have contributed to the recurrent thrombosis and because the history of aspirin intolerance was equivocal the patient was challenged with aspirin. There were no ill effects and regular treatment with aspirin was started.

His symptoms abated and he was free of symptoms at follow up six months later. Follow up angiography of the superior vena cava showed that the stent remained in position and was widely patent. There was a filling defect within the lumen of the stent (fig 3) but the absence of collateral vessels, including the previously enlarged azygos system, and the excellent flow seen at the time of the procedure suggested that there was no haemodynamically significant obstruction within the stent. His pacemaker function was normal throughout the investigations.

Discussion

Venous thrombosis is a well recognised complication of transvenous pacing leads that occurs in 30–45% of patients.[1] The most common sites of thrombosis are the subclavian, axillary, and innominate veins and the superior vena cava.[1] Complete occlusion is less common and may be clinically silent because of the development of a collateral circulation. Superior vena caval syndrome secondary to pacemaker-induced thrombosis is uncommon. Goudenovos *et al* reported only one case in 3100 implants over a 10 year period at their institution and a further three cases referred to them from elsewhere.[2] Another study cited an incidence of four in 1000 cases.[3] Multiple leads, particularly severed ones, and previous infection seemed to be the most important aetiological factors. Our patient had had both.

The underlying pathology is often fibrotic stenosis. Endothelialisation of pacing leads occurs from an early stage[1] and when it is excessive connective tissue proliferation can cause clinically significant reduction in the luminal diameter of the vessel. Collateral formation can further reduce flow within the vessel and subsequent thrombosis ultimately may lead to occlusion.

Thrombosis is a dynamic process and the history in this case suggested a recent acute event. This is borne out by the response to thrombolysis. With an underlying stenosis, however, recurrence seemed likely. There are reports of successful angioplasty of venous stenoses complicating pacemaker leads[4] and also of successful resolution of pacemaker-induced superior vena caval syndrome.[5] The recurrence rate after angioplasty is considerable and higher in arterialised venous grafts and dialysis shunts than arteries. Stenoses in dialysis shunts have a one year patency rate of only 45% after angioplasty[6] and the restenosis rate for venous bypass grafts is up to 54%.[7] Venous stenoses are often fibrotic and more liable to recoil. Balloon angioplasty generally has not been successful in the longer term.[8]

We thought that restenosis was likely in this

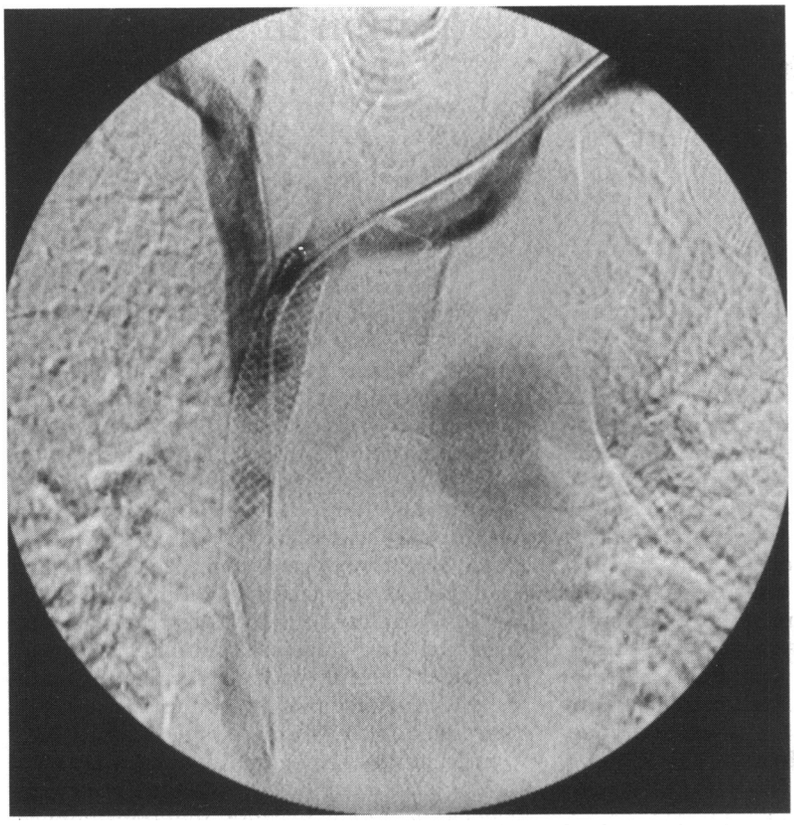

Figure 3 Repeat angiogram of the superior vena cava six months after stent insertion. The stent is patent and there is a filling defect on the medial wall.

situation and elected to place a stent as a primary definitive procedure. The subsequent relapse illustrates the importance of adequate adjuvant antithrombotic therapy. In retrospect, we should have used aspirin in the first instance and should have obtained a greater degree of anticoagulation with warfarin before treatment with heparin was stopped. Since aspirin treatment was started after rethrombolysis the patient has remained symptom free. We decided to continue long-term treatment with warfarin because he also has paroxysmal atrial fibrillation.

We considered the possibility that the stent may interfere with pacemaker lead function but thought that this was unlikely because the lead would be well ensheathed with endothelium at this stage and because the angiogram clearly showed that it was lying outside the lumen of the superior vena cava (fig 2).

We were concerned to see a filling defect on the follow up angiogram. This may indicate the organisation of thrombus after the initial acute occlusion and provided that thrombosis is not continuing it is unlikely to cause any clinical problems. Another possibility is progressive intimal hyperplasia. This is more pronounced in stented veins than in stented arteries[8] but does not seem to present a significant clinical problem. Ultimately the stent may occlude as the native superior vena cava did. Primary surgical repair would be difficult in a patient who has already had bypass grafts.

In conclusion superior vena caval syndrome associated with a pacemaker lead is rare but causes distressing symptoms. This case shows that if obstruction is diagnosed promptly thrombolysis will relieve the symptoms, that angioplasty can be used successfully to dilate underlying venous stenoses, that intravascular stents can be placed safely in the presence of venous pacing leads, and that scrupulous attention to anticoagulation regimens is required to maintain patency.

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