CASE REPORT

Multiple-vein thrombosis and pulmonary embolism after pacemaker implantation treated by thrombolysis

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Abstract This report describes a patient who suffered multiple-vein thrombosis following permanent pacemaker implantation and developed a pulmonary embolism while on anticoagulation treatment, which was successfully treated by thrombolytic therapy.

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KEYWORDS
subclavian thrombosis; pulmonary embolism; thrombolysis; pacemaker

Introduction

Thrombosis of entry vein(s) following permanent pacemaker and ICD implantation alone or with additional atrial and ventricular leads (CRT and multi-chamber pacing), is becoming more widespread due to the increasing rate of implantation, as well as the increasing number of leads implanted with these devices. This report describes a patient with multiple-vein thrombosis following permanent pacemaker implantation who, during anticoagulation treatment, subsequently developed a pulmonary embolism which was successfully treated by thrombolytic therapy.

Case report

A 65-year-old male was admitted with a painful swollen left arm and shoulder. He denied trauma of the extremity, chest discomfort or dyspnoea. Eight months previously a permanent DDDR pacemaker had been implanted via the left subclavian vein due to syncope and high degree AV block. The procedure lasted 37 min and there were no complications. On several follow-up examinations the patient felt well and the pacemaker functioned normally in VDD mode. On admission his physical examination was normal except for the
oedematous left upper limb. The left arm was tender to palpation, the left jugular vein and superficial veins over the proximal part of the limb and shoulder were distended. Laboratory data were unremarkable. Chest X-ray showed a small left pleural effusion. A left subclavian vein thrombosis was suspected and subsequent duplex ultrasonography confirmed the diagnosis. Two days later, venography showed multiple thrombi in basilic, axillary and subclavian veins with venous collaterals around the shoulder (Fig. 1). The patient received subcutaneous enoxaparin 60 mg twice daily for 10 days without any change in his condition, except that he developed sudden dyspnoea accompanied by hypoxia \((PO_2 = 55 \text{ mmHg})\) and respiratory alkalosis \((pH = 7.52, PCO_2 = 24 \text{ mmHg})\). Ventilation-perfusion lung scan showed a large perfusion defect in the base of the left lung and segmental defect on the right side. Echocardiography did not show right ventricle dysfunction and the patient remained haemodynamically stable. Pulmonary embolism was diagnosed. Enoxaparin therapy was discontinued and the patient received 250 000 units of streptokinase intravenously for 30 min, followed by 100 000 units/h for 24 h. Warfarin therapy was initiated concomitant to the streptokinase. On the 2nd day of treatment the unilateral neck vein congestion diminished markedly and 3 days later venography demonstrated a good reperfusion effect (Fig. 2). The patient was maintained on warfarin and 10 months after discharge he felt well with no signs of recurrent vein thrombosis.

**Discussion**

Transvenous pacemaker leads, like any foreign intravascular body, alter venous flow and increase turbulence [1], which may lead to platelet aggregation and fibrin deposition, causing further reduction in the vessel lumen, possibly culminating in total venous occlusion. Vein thrombosis, especially in the subclavian vein, is not uncommon after pacemaker or ICD implantation [1–4]. It is reported on average in 12% (range 2–22%) of patients, from several days to 9 years post-implantation (data collected from 10 publications reporting on a total of 702 patients) [3]. In patients with local infection, the number of occluded subclavian and/or brachiocephalic veins was 27%, while in patients with systemic infection it was twofold (55%) [3]. Acute symptomatic thrombosis was observed in only 5% of these patients; usually vein occlusion is asymptomatic and clinically insignificant [2]. Since access to a partially or completely obstructed vein(s) is complicated, preoperative venography has been advocated before all device lead revision [2]. There is some evidence that difficult and traumatic access at the implant is associated with inflammation,
which may subsequently lead to thrombosis of the entry vein [1,3].

Several additional thrombogenic factors have been discussed in the literature [1,3–7].

Although it would be reasonable to assume that multiple leads could result in a high rate of vein obliteration, this expectation has not been substantiated in the literature. None of seven plethographic studies described a higher incidence of occlusion in patients with multiple leads [3]. A similar number of thrombotic complications have been demonstrated in a prospective echo-Doppler study in 39 patients each with 3 leads and in 9 patients with four leads, compared with 48 age-matched control patients with DDD pacemakers [4]. There is controversy regarding the entry site of implanted leads, i.e. cephalic cut-down versus subclavian puncture [1,3,5]. In a recent report describing 89 patients with pacemakers and ICDs, there was no difference in occlusion of the subclavian vein regarding entry site, or increased risk for occlusion in the case of a second implant procedure [3]. There was also no difference in incidence of thrombosis in the use of polyurethane or silicone type of lead insulation [1].

The time of appearance of occlusion symptoms after pacemaker/ICD implantation ranges from several days to several years [3]. In our patient, clinical signs of thrombosis upper limb veins appeared 8 months after pacemaker implantation and there were no risk factors for thrombotic occlusion of the subclavian vein.

Although asymptomatic pulmonary embolism following pacemaker implantation is common (detected in 15% of patients as a new small perfusion defect(s) on pulmonary scintigraphy within 14-day postoperative period) [6], this ‘laboratory phenomenon’ probably has no clinical significance. Symptomatic pulmonary embolism is a very rare [7] but major and clinically significant complication and its presence in patients with implanted devices should raise suspicion of thrombotic formation around lead(s) from proximal to distal points [9].

There is no firm consensus regarding therapy for catheter-related thrombosis [1,2,7,8]. Specific therapy depends on whether thrombosis or fibrosis is the causative factor, as well as on symptom presentation grade. Therapy will vary from heparin followed by warfarin or thrombolysis to percutaneous angioplasty, with or without stent or open surgery procedure [1–3,7,8]. Anticoagulation has been the therapy of choice based on prevention of clot propagation while allowing for collateral formation although in some cases the existing thrombus could spontaneously resolve without therapy at all. In patients with signs of acute thrombosis of the subclavian vein after pacemaker/ICD implantation, thrombolytic therapy should probably be initiated before anticoagulation. As first line therapy it may be effective not only in

Figure 2 Five days after initiation of streptokinase, no thrombi were found in the axillary and basilic veins. Despite the presence of thrombus, there is appropriate flow through the subclavian vein.
revascularization of occluded veins, but also in potentially preventing delayed morbidity, such as sequelae of chronic post-phlebitic syndrome. It may also prevent fragmentation of the thrombus and consequently prevent pulmonary embolism.

References


