

TEVAR for Iatrogenic Injury of the Distal Aortic Arch after Pacemaker Implantation

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Introduction: We report the endovascular treatment of aortic arch injury due to direct puncture during pacemaker implantation.

Report: After pacemaker implantation a 74-year-old woman showed a progressive decrease in haematocrit with elevation of cardiac troponin-I. Coronary angiography revealed the malposition of the catheters introduced through the aortic wall. The atrial lead was placed in the left circumflex coronary artery. Computed tomography scan confirmed distal aortic arch perforation. A Medtronic-Valiant stent-graft was implanted in the distal aortic arch while the two catheters were removed. A new VVI pacemaker was implanted and, 3 days later, the patient underwent percutaneous coronary intervention (PCI) on the dissected left circumflex artery. Four days later the patient was discharged. One-year computed tomography scan showed successful repair of the injured aorta.

Discussion: Endovascular stent grafting has emerged as a less invasive therapeutic alternative to treat traumatic or iatrogenic injuries of the distal aortic arch.

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INTRODUCTION

The rate of acute complications after pacemaker implantation is 4–5% and mostly related to operator experience.¹ Some of these complications can be dramatic, especially those related to venous access (haemothorax, pneumothorax, venous thrombosis, etc.) or to lead malpositioning (ventricular or atrial perforation with cardiac tamponade). All these complications are fully described elsewhere in the literature.¹

Herein we report treatment with thoracic stent-graft implantation (thoracic endovascular aortic repair [TEVAR]) of a rarely described dramatic complication after pacemaker implantation: an aortic injury due to direct puncture of the distal aortic arch with malplacement of the two pacing leads in the left ventricle and in the left circumflex artery.

REPORT

A 74-year-old woman was transferred from a local hospital and admitted to our emergency unit for an evolving haemothorax and increased cardiac troponin-I and creatine kinase, with progressive systemic hypotension and syncopal episodes. She referred fatigue and dyspnoea without chest pain.

A dual chamber pacemaker with DDD mode had been implanted 48 hours before through a left subclavian access.

Blood tests showed a progressive decrease in haematocrit with significant elevation of blood level markers for myocardial infarction, namely cardiac troponin I > 100 ng/ml and creatine phosphokinase MB isoenzyme > 30 ng/ml. Chest X-ray showed a large haemothorax. The transthoracic echocardiography revealed a marked hypokinesia of the inferolateral wall of the left ventricle, in the areas supplied by the left circumflex artery (LCX), and identified malplacement of the two pacing leads in the left ventricle crossing the aortic valve. A mild aortic regurgitation was observed and the presence of the large haemothorax was confirmed. Emergent coronary angiography revealed the malposition of the two pacemaker catheters introduced through the aortic wall. The ventricular lead was positioned at the apex of the left ventricle, while the atrial lead was placed in the left circumflex coronary artery, as depicted in Fig. 1B. Fortunately, the two branches of the left coronary artery issued from the aorta by separate ostia. The increased elevation of the markers for myocardial injury and myocardial hypokinesia were due to LCX occlusion.

Accurate diagnosis of the direct aortic wall injuries was established by a contrast-enhanced spiral computed tomography (CT) scanner. The three-dimensional volume rendering showed perforation of the distal aortic arch wall below the left subclavian artery (LSA) origin (Fig. 1A).

We considered it necessary and urgent to remove the malpositioned leads, and treat the aortic injury. The transthoracic echocardiography indicated the absence of thrombus on the pacing leads. As the patient was considered

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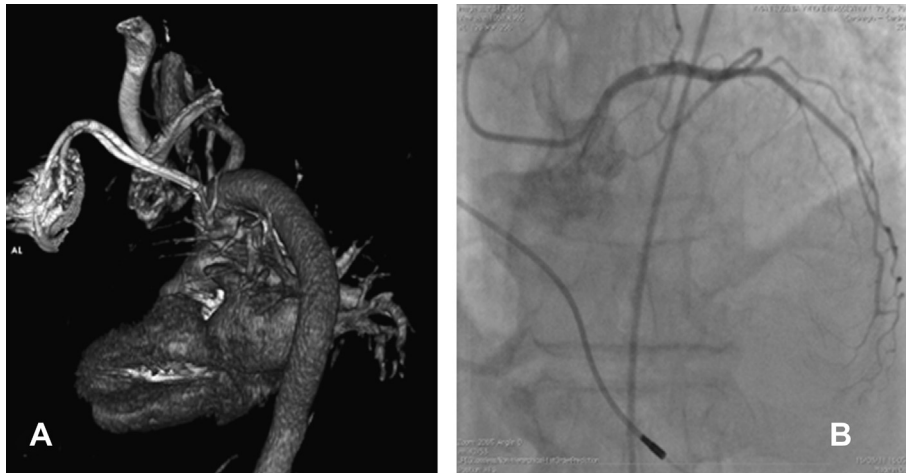


Figure 1. (A) Volume-rendered image showing perforation of the distal aortic arch; (B) coronary angiography showing leads malposition.

unfit for conventional surgery because of the evolving haemothorax and myocardial ischaemia with severe ventricular hypokinesia, we opted for endovascular treatment with thoracic stent–graft placement. The criteria to assess anatomical suitability for device implantation were respected.

Stent–graft placement was performed in a hybrid operating room. The patient received general anaesthesia and mechanical ventilation. Through left femoral artery access a Medtronic Valiant stent–graft (Medtronic AVE, Santa Rosa, CA, USA) was implanted in the distal aortic arch covering the LSA origin, while the two catheters were removed from the pocket of subclavian access. At the same time a new VVI pacemaker was implanted through a right subclavian access.

Subsequent aortography confirmed the adequacy of the treatment and the absence of bleeding from the aortic wall, while the coronary angiography showed the dissection with a subocclusion of the LCX artery.

We decided not to treat the coronary lesion at the same time in order to reduce the risk of bleeding from the aorta

following administration of antiplatelets. After TEVAR the patient was transferred to the intensive care unit for management of her haemodynamic instability due to the myocardial ischaemia.

After confirming absence of bleeding from the aortic lesion, 3 days later the patient underwent percutaneous coronary intervention with a drug-eluting stent on the dissected LCX (Fig. 2B).

Four days after surgery the patient was transferred to a rehabilitation center. One-month CT scan showed successful repair of the injured aorta.

At 1-year follow-up the patient was in good condition, showing no signs of steal phenomena due to the intentional occlusion of the LSA. The CT scan confirmed successful repair of the injured aorta (Fig. 2A).

DISCUSSION

Malposition of a pacemaker lead in the left ventricle is a complication rarely described in the literature.² The lead can be introduced into the left ventricle through a patent

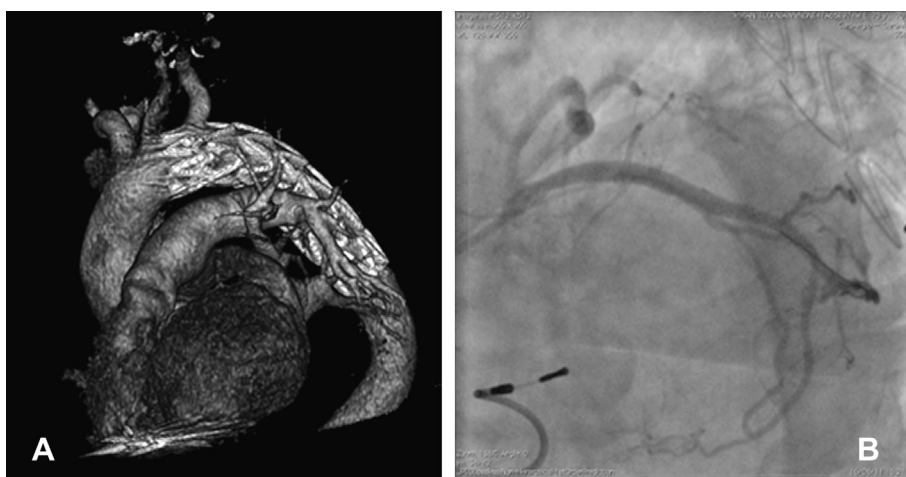


Figure 2. (A) One-year computed tomography scan confirming successful repair of the injured aorta; (B) coronary angiography after percutaneous coronary intervention.

foramen ovale or an atrial septal defect.² More rarely, the lead can be inserted through the aorta for an inadvertent puncture of the subclavian artery³ or through a direct puncture of the aortic arch.⁴

In the literature we found only one case of a pacemaker lead malpositioned in the left ventricle through direct puncture of the aorta treated with conventional surgery through a median sternotomy to achieve direct closure of the aorta.

This complication usually becomes obvious through the appearance of symptoms or at follow-up several months later. Malpositioned leads are easily recognisable by two-dimensional echocardiography or by chest X-ray³ revealing the aberrant course of the ventricular lead into the aorta.

Patients with malpositioned leads in the left ventricle can be asymptomatic or develop symptoms secondary to systemic embolization. Asymptomatic patients can be treated with a lifelong anticoagulant, such as Warfarin, to prevent thrombo-embolic complications,⁵ which occur in approximately 40% of patients,⁵ reserving surgical extraction for symptomatic patients or during concomitant surgery in order to avoid potential mobilisation of thrombotic material during lead manipulation.² Surgical removal requires median sternotomy or thoracotomy, or a less invasive approach using a right anterolateral thoracotomy.

Removal of the malpositioned lead was required in our patient as she had developed a large haemothorax, symptoms indicating malplacement of one of the leads in the LCX, and a demonstrated absence of thrombotic material.

In treating this particular aortic injury we opted for an endovascular strategy because the patient was considered

unsuitable for conventional surgery given her current clinical conditions.

CONCLUSION

TEVAR has emerged as a less invasive therapeutic alternative in treating iatrogenic injuries, mostly in high-risk patients, of the distal aortic arch.

CONFLICT OF INTEREST

None.

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