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Successful Reimplantation of a Passive-Fixation Ventricle Lead Perforating the Chest Wall

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ABSTRACT

Delayed perforation of a passive fixed pacemaker lead is a rare complication after pacemaker implantation and is associated with increased morbidity and mortality. We report the case of an 82-year-old patient who presented with a delayed perforation of the right heart wall, the pericardium, and the chest wall by a passive-fixation ventricular lead 14 months after pacemaker implantation. The lead was uneventfully extracted transvenously and repositioned in the right ventricle with good pacing and sensing.

BACKGROUND

Lead perforation after pacemaker implantation is an uncommon complication and generally occurs within the first postoperative month. Delayed perforation by the ventricle lead (ie, >1 month after implantation) is a rare and potentially dangerous complication [Ellenbogen 2002]. Right ventricle perforations have usually been described after implantation of active-fixation leads [Lopes 2007]. We report the asymptomatic perforation of a passive-fixated lead through the right ventricle wall, the pericardium, and the chest wall in an 82-year-old patient, and we describe our management of this clinical situation.

CASE REPORT

An 82-year-old patient was referred to our department for the management of a dislocation of a right ventricular lead after routine control of her permanent pacemaker in the outpatient facility in a cardiology department. Her dual-chamber pacemaker (Vitatron T70 DR; Vitatron, Düsseldorf, Germany) had been implanted 14 months before presentation because of sick sinus syndrome. Postoperative and short-term (3 months) testing of the pacemaker demonstrated the correct function of both leads; however, a routine interrogation of the pacemaker at 14 months postoperatively showed a ventricular exit block and loss of sensing of the ventricular lead.

Clinically, the patient was asymptomatic and had experienced no syncope episodes after implantation. An electrocardiographic evaluation showed a sinus rhythm with predominantly atrial stimulation. In addition, a 24-hour Holter electrocardiographic evaluation also showed a sinus rhythm with atrial stimulation and normal intrinsic conduction to the ventricle. There were no pauses longer than 2.3 seconds. A chest radiograph showed migration of the right ventricular lead beyond the cardiac silhouette into the left lateral chest wall (Figure 1). Chest computed tomography scanning revealed perforation of the right ventricle wall and pericardium, as well as migration of the passive-fixed lead through the subcutaneous fatty tissue of the chest wall (Figure 2).

Reintervention was scheduled. Although a light chronic pericardial effusion and partial adherences were observed in

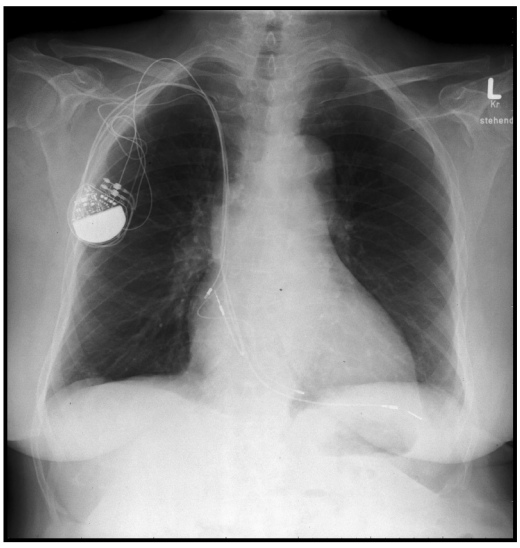

Figure 1. Posteroanterior chest radiograph of the patient showing the ventricular lead beyond the cardiac silhouette.

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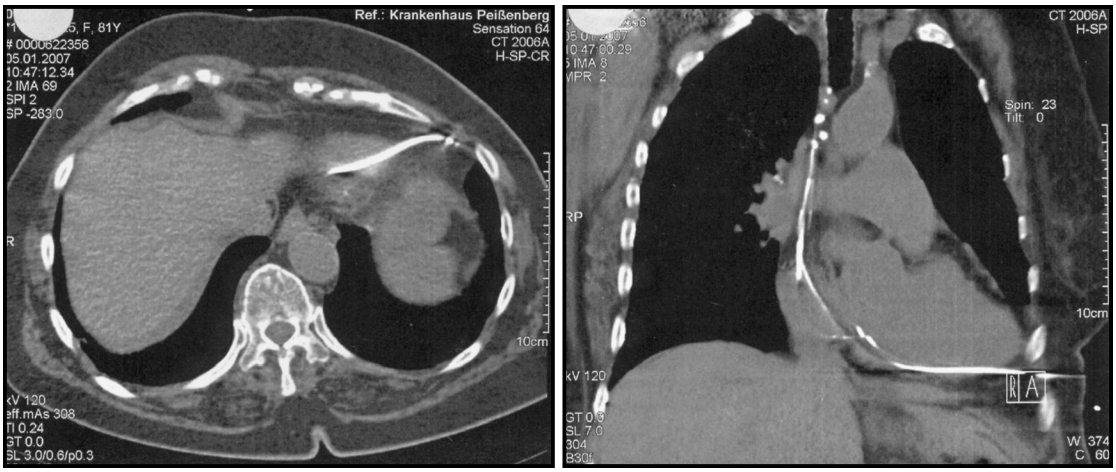

Figure 2. Thorax computed tomography scans showing the ventricular lead perforating the right ventricle and the pericardium (left) and the lead's tip in an intercostal space (right).

the computed tomography scan, retraction and reimplantation of the old ventricular lead were deemed feasible. The operation was performed with cardiac surgical backup and was successful. The ventricular lead was gently retracted during transesophageal echocardiography monitoring and repositioned into the apex of the right ventricle. The condition of the patient remained stable. Serial echocardiographic monitoring in the intensive care unit revealed no increase in pericardial effusion.

The patient was discharged in good condition on the second postoperative day after an echocardiographic control evaluation was made, a chest radiograph appeared normal (Figure 3), and correct pacemaker function was confirmed.

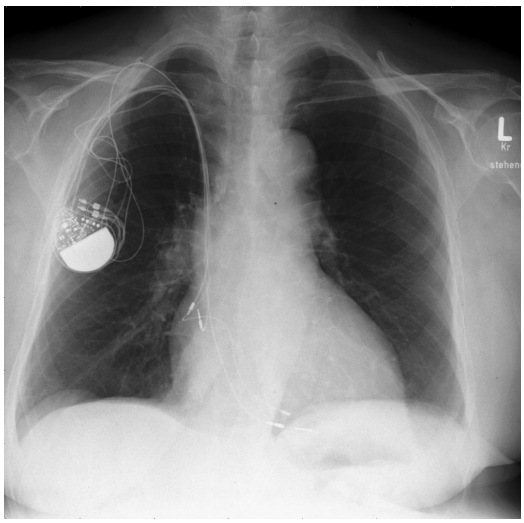

Figure 3. Posteroanterior chest radiograph of the patient at discharge.

DISCUSSION

Acute myocardial perforation following dual-chamber pacemaker implantation has been reported to occur in less than 1% of cases. It typically occurs at the time of implantation and rarely occurs after the first 24 hours. Delayed ventricle perforation (>1 month after pacemaker implantation) is an even rarer complication and is potentially dangerous [Kiviniemi 1999].

Clinical presentation is heterogeneous, varying from pericardial effusion with cardiac tamponade to symptoms of chest pain. In our case, the patient was asymptomatic, and the diagnosis was made accidentally in a routine control evaluation of her pacemaker 14 months after her operation. In such cases, the presence of defects in sensing/pacing or impedance in any of the leads after pacemaker interrogation must lead to the suspicion of abnormal lead positioning, and imaging analysis should be instituted for further diagnosis. Such imaging should consist primarily of chest radiographs, but computed tomography scanning is frequently necessary to determine the exact location of the lead as well as the anatomic structures affected [Hirschl 2007].

The optimal management of this clinical situation has not been defined, and different strategies have been proposed. It is important to define whether the lead must be removed and to ascertain the best method for removal if it is necessary. Conservative procedures would include the fixation of the lead and transvenous implantation of another ventricular lead in the right ventricular outflow tract to ensure pacemaker function. Open-chest surgery offers more safety for extracting the lead; however, surgical extraction is an invasive procedure with associated risks and a longer hospital stay.

The removal and repositioning of the perforated lead or the implantation of a new one are less invasive than open-chest surgery; however, these interventions should be performed

with echocardiographic monitoring, general anesthesia, and with a cardiac surgical backup [Selcuk 2006]. The method of lead fixation may influence the therapeutic decision. Active-fixation leads can be extracted transvenously with a low risk of complications; however, tine leads often have bulky tips that increase the risk of tissue damage during extraction. Therefore, cutting the lead's tips and posterior transvenous extraction of the electrode are recommended.

In our patient, transvenous extraction was performed uneventfully under echocardiographic monitoring, and the lead produced good sensing/pacing values after it was repositioned in the right ventricle. Our decision to reposition the lead was supported by our assessment that further migration of the perforated lead could cause bleeding or structure damage and of the relative risk of causing venous damage by retracting a tined lead. Repositioning the lead allows these risks to be avoided.

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