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## Review

# Identification and management of right ventricular perforation using pacemaker and cardioverter-defibrillator leads: A case series and mini review

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## ABSTRACT

Right ventricular perforation is a rare but serious complication of permanent pacemaker and implantable cardioverter-defibrillator implantation, with a reported prevalence rate of 0.1–6%. Generally, there is a high incidence of asymptomatic lead perforation with otherwise normal function. Some patients present with a stabbing chest pain and shortness of breath or pacemaker malfunction. However, in some cases, tamponade or adjacent tissue injury may be seen. The exact risk factors for lead perforation are not yet clear. Furthermore, there are many controversies in the management of lead perforation. Extraction of an asymptomatic, incidentally detected, chronically perforating lead does not seem to be necessary. Patients with symptoms or device malfunction will require treatment appropriate for their problem.

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## 1. Case reports

### 1.1. Case 1

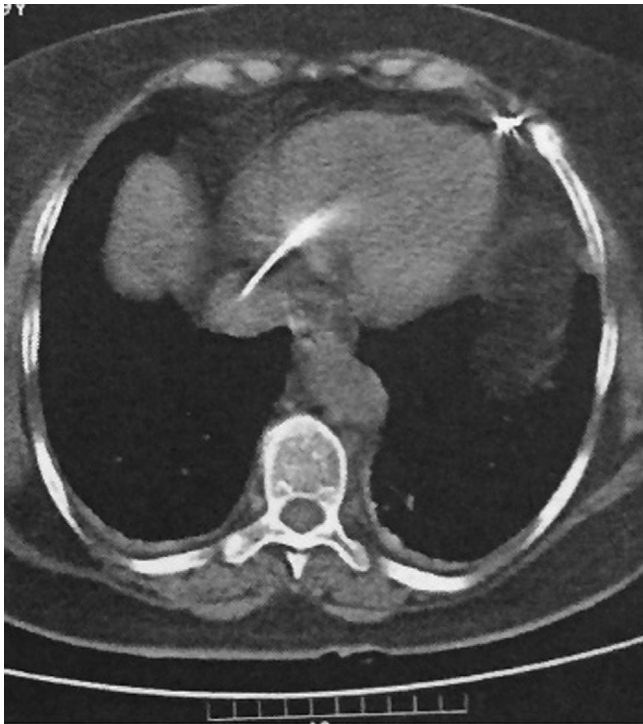
A 67-year-old woman, 2 months after uncomplicated implantation of a dual chamber permanent pacemaker (PPM) for

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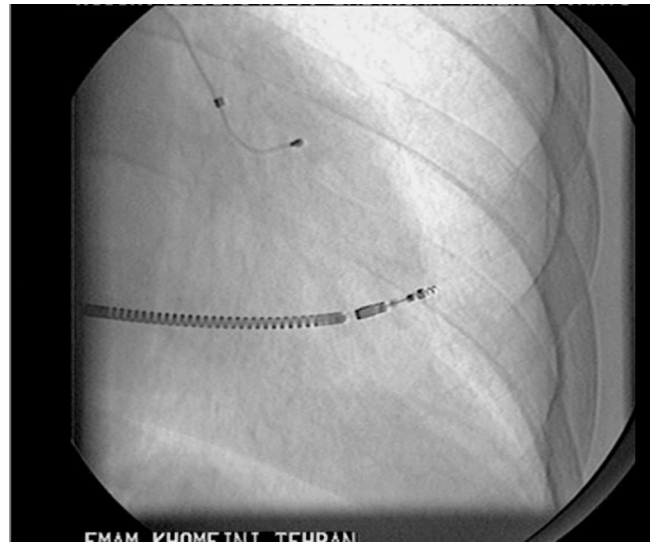


**Fig. 1.** Chest computed tomography. Perforation of the right ventricular apex 2 months after pacemaker implantation.

complete atrioventricular (AV) block, was referred to our hospital owing to the recurrence of her symptoms and the development of new onset chest pain that was exacerbated by deep breathing. The 12-lead surface electrocardiography (ECG) showed a normal sinus rhythm, complete heart block, and evidence of ventricular lead undersensing and failure to capture. The PPM interrogation showed no R wave and no ventricular capture at the maximum output. Leads impedances were within the normal range. The right atrial (RA) lead (Capsure fix Medtronic 4076) function was normal. Echocardiography showed minimal pericardial effusion. Chest computed tomography (CT) confirmed perforation of the right ventricular (RV) apex by the RV lead (Fig. 1). In the operating room, with full hemodynamic monitoring and cardiac surgery backup, the PPM pocket was opened and, after deactivation, the RV lead (Capsure fix Medtronic 4076) in the apex was extracted through the left subclavian vein and a similar new active fixation lead (Capsure fix Medtronic 4076) was implanted in the right ventricular septum. No symptoms of tamponade were observed during or after the procedure in 8 months of follow-up.

### 1.2. Case 2

A 60-year-old woman with known non-ischemic cardiomyopathy with a left ventricular ejection fraction of 20% was scheduled for cardiac resynchronization therapy-implantable cardioverter defibrillator (CRT-ICD) due to exertional dyspnea despite guideline-directed medical therapy. During the procedure, RA (Tendril STS 1888 Saint Jude Medical [SJM]), RV (Durata 7120 SJM), and coronary sinus (CS) (Quick flex 1258 SJM) leads were implanted in the RA appendage, RV apex, and lateral branch of the CS, respectively. Sensing and pacing parameters were within the normal range. Follow-up device interrogation at 1 and 3 months was normal, but RV lead capturing was lost (even at 7.5 V) and the R wave amplitude decreased (1.2 mV) at the 6 months post-implantation although the patient was completely asymptomatic. The fluoroscopy findings indicated RV lead penetration of the



**Fig. 2.** Fluoroscopy view. Right ventricle lead penetration to the pericardium 3 months after cardiac resynchronization therapy implantation.

pericardium (Fig. 2), but there was no pericardial effusion on echocardiography. Multislice CT of the thoracic cavity confirmed the position of the RV lead to be outside the heart in the pericardium. Follow-up visit and echocardiography findings at one year were unremarkable. We implanted a new 58-cm simple bipolar lead (Capsure fix Medtronic 4076) in the RV septum and decided to abandon the previous RV lead without removing it and kept the patient on close follow-up. Over the course of 2 years of follow-up, the patient was asymptomatic and echocardiography showed no pericardial effusion.

### 1.3. Case 3

An 85-year-old woman experienced episodes of syncope, and during one of the episodes, sinoatrial node arrest was documented. She successfully underwent dual chamber PPM with a diagnosis of sick sinus syndrome (RA lead: Tendril STS 1888 SJM in the RA appendage, RV lead: Tendril STS 1888 SJM in the RV apex) (Fig. 3A). Four weeks later, the patient complained of left-sided pleuritic chest pain and left hemithorax pulsation. PPM interrogation revealed normal RA lead function but a decrease in R wave amplitude and failure to capture (even at the maximal voltage). Chest radiography showed the RV lead in the left pleural space (Fig. 3B). Considering that the patient did not have any episodes of atrioventricular block, only the migrated RV lead was unscrewed and totally retracted and the mode of the generator changed to AAI. Echocardiography performed immediately and 2 days after RV lead removal showed no pericardial effusion. The 18-month follow-up was uneventful.

### 1.4. Case 4

A 75-year-old man, with hypertrophic cardiomyopathy, underwent dual chamber ICD (Ellipse SJM) with a good sensing and capturing threshold (RA lead: Tendril STS 1888 SJM, RV lead: Durata 7120 SJM). On the next day, R wave amplitude and capturing thresholds were 5 mV and 2.5 V at 0.5 ms, respectively. Two weeks later, the R wave amplitude was 2 mV with no ventricular capturing even at high voltages. Echocardiography showed a massive pericardial effusion, with impending subsequent tamponade. The patient was brought to the cardiovascular hybrid operating room. Under general anesthesia and transesophageal guidance, the RV lead was retracted and another ICD lead (Durata

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