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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.

Contents

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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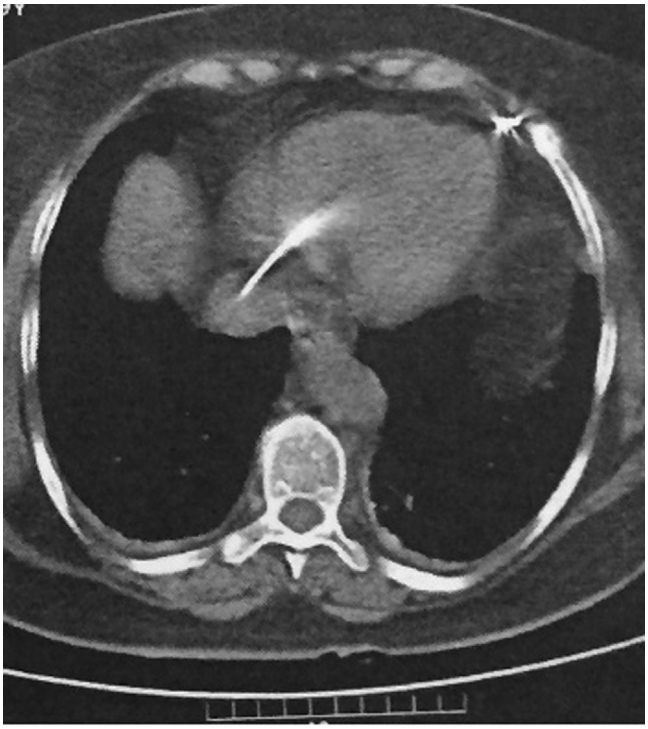

Fig. 1. Chest computed tomography. Perforation of the right ventricular apex 2 months after pacemaker implantation.

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 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.

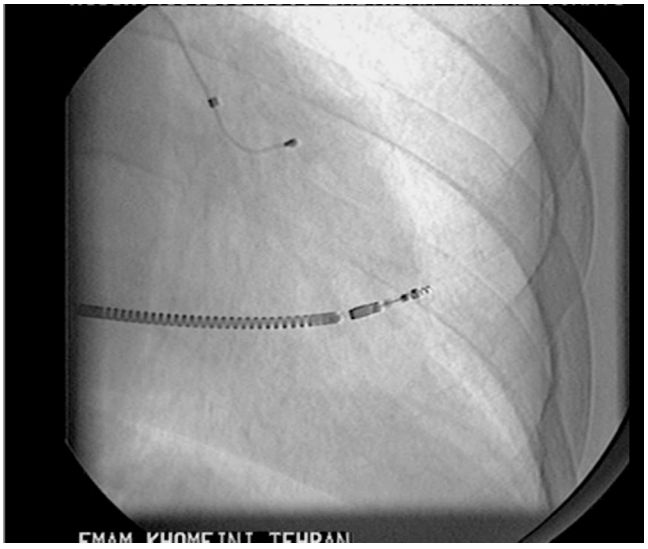

Fig. 2. Fluoroscopy view. Right ventricle lead penetration to the pericardium 3 months after cardiac resynchronization therapy implantation.

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

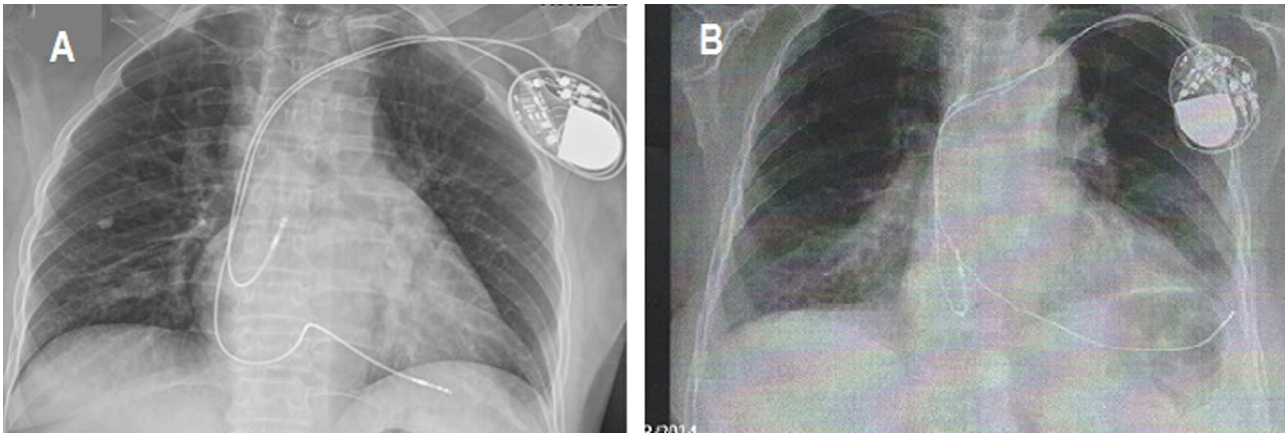

Fig. 3. Chest radiography. A, Chest radiography at the time of implantation; B, Chest radiography 4 weeks later showed the right ventricle lead in the left pleural space.

7120 SJM) was implanted in the interventricular septum. Open pericardial drainage was performed. The 18-month follow-up was uneventful.

2. Discussion

RV perforation is a rare but serious complication of PPM and ICD implantation. It is diagnosed when at least the tip of a passive fixation lead or the screw of an active fixation lead passes through the myocardium and extends into the pericardial cavity [1]. This complication may develop acutely (i.e., less than 24 h) after device implantation or in a subacute or chronic fashion [2].

2.1. Incidence

Different incidence rates have been reported in the literature. Before 2000, the rate of RV lead perforation in PPM and ICD was reported to be 0.1–0.8% and 0.6–5.6%, respectively [3]. In the OPTIMUS registry (designed to monitor the long-term performance of SJM leads), RV perforation was observed in 0.33% of the cases with ICD leads and 0.5% of the cases with PPM leads [4]. However, since this complication may be asymptomatic in some patients, the prevalence is probably underestimated in many studies. CT evaluation of patients has shown a prevalence of 6% [5].

2.2. Clinical presentations

Although the rate of cardiac tamponade is higher in acute RV lead perforation, the presentation is different in the late form. Most patients present with a stabbing chest pain and shortness of breath [2,6]. Severe pericardial effusion and tamponade due to leakage from a low-pressure chamber (e.g., RV) is observed less than anticipated, perhaps due to a combination of the self-sealing properties of the ventricle wall by muscle contraction, fibrosis, or by the lead itself [3,6]. PPM malfunction, such as pacing or sensing failure, is also common. Perforation of the left liver lobe due to the migration of the lead through the left hemidiaphragm [7], pneumonia and hemothorax as a result of lead migration into the anterior left pleural space [8], chest muscle twitching [9], hiccups [10], chest wall hematoma [11], and rib perforation [12] have all been reported in previous articles.

2.3. Risk factors

Owing to the very low incidence of RV perforation and the conflicting results of previous reports, the exact risk factors are not clear. For example, in some reports, the incidence of lead perforation seems to be higher with active lead fixation but other studies found an equal incidence for both passive and active leads [13,14].

Although activating the lead in the right ventricular apex results in greater stability, screwing the electrode into the thick ventricular septum not only preserves the normal activation sequence, but also reduces the risk of perforation and consequent tamponade. Moreover, it is known that the free wall location of the RV lead increases the risk of perforation [3,14–16]. In some cases, perforation occurs when significant pressure is perpendicularly transferred to the heart wall by using a straight stylet during implantation, and when an excessive loop of the lead is left and generates more tension in the cardiac wall [17].

The lead itself may be another predictor. There are reports of higher perforation rates with Riata ST 7000 SJM, especially in the first 6 weeks of implantation, which might point to the structural properties of these leads [18–21]. The design characteristics of the lead, the stiffness of the tip with a smaller diameter, especially if the lead body construction applies forward pressure on the lead in the case of an excess loop, may contribute to perforation by increasing the force applied to an area. In order to decrease the rate of perforation, newer leads have softer and more flexible tips so their force on the wall decreases [22].

Anticoagulant or steroidal drug consumption in the first 7 days post-implantation, old age, female sex, and low body weight are other predictors of this complication in some studies [6,17,23]. A high right ventricular systolic pressure (more than 35 mmHg) is associated with a lower rate of cardiac perforation, which may be due to associated RV hypertrophy [23].

Thin heart muscle itself may favor perforation. Akyol et al. reported that in a patient with myotonic muscular dystrophy and dilated cardiomyopathy, a PPM lead caused right ventricular perforation [24]. A blunt chest trauma, especially soon after implantation, may also predispose the patient to cardiac perforation [25,26].

2.4. Diagnosis

Most late perforations are asymptomatic and suspicion is raised after detecting device malfunction upon routine analysis. A diagnosis of ventricular lead perforation may be confirmed with chest radiography, fluoroscopy, echocardiography, or chest CT scan.

When the lead migrates too far from the heart, chest radiography and fluoroscopy can easily detect the problem. However, in cases with minimal perforation of the heart, these tests are often non-diagnostic.

Two and three dimensional echocardiography can also be helpful for the detection of pacing wire perforation when the path

of the wire is visualized in the spatial orientation of the echocardiography beam [27]. Post-implantation pericardial effusion detected via echocardiography can be a sign of lead perforation, but there are other mechanisms that can cause effusion such as traumatic inflammation of the myocardium and pericardium from the lead screw, or irritation of the visceral pericardium via immune mediated mechanisms [1,28]. Although three-dimensional echocardiography has a lower spatial and temporal image resolution than a two-dimensional technique, because of the high echogenicity of the lead and the sharp interface between the intracavitary blood and the surface of the wire, this technique can improve the accuracy of the lead perforation detection because of its more realistic and comprehensive view of the intracardiac structures [27].

ECG-gated non-contrast cardiac CT, especially when performed with the new generation of multidetector helical scanners, provides an excellent visualization of the heart and can be the most important diagnostic method to confirm myocardial perforation not detected by other modalities. It can evaluate the intracardiac lead position and even small amounts of pericardial effusion. However, the metal edges cause streaks surrounding the electrode tip, known as the “star artifact”, which affect the quality of the image and make it difficult to detect the lead tip [29–31].

Due to concerns about catastrophic complications, especially in older devices, magnetic resonance imaging (MRI) is not recommended for the detection of lead perforation and is only performed with certain safety protocols in patients with other definite indications for MRI. However, with new generations of MRI-conditional devices, this imaging modality, with fewer lead artifacts compared with CT, may become the gold standard for the detection of lead perforation in the future [32].

2.5. Management

The optimal management of lead perforation is still unclear. There is a high incidence of asymptomatic lead perforation with otherwise normal function. According to our practice and the opinion of some experts, the extraction of an asymptomatic, incidentally detected, chronically perforated lead does not appear to be necessary [5,6]. However, some prefer to extract these leads [3,17]. On the other hand, patients with symptoms or device malfunction will require appropriate revisions for their problem. Hemodynamically stable patients with postimplantation pericardial effusion, but normal device function, rarely require any therapy but should be followed closely due to the risk of cardiac tamponade [1,28]. On occasion, reprogramming the device is all that is needed [33,34].

Cardiac tamponade, as a late complication of device placement, is exceedingly rare. The strategy for this fatal complication depends on the experience of the team, dynamics of the symptoms, and hemodynamic status. Successful management with closed pericardiocentesis and a pericardial drain in place has been reported. Surgical intervention seems to be the treatment of choice in the case of other visceral injuries, hemodynamic instability, rapid progression of pericardial effusion, or if closed pericardiocentesis fails [35]. In this setting, the surgeon can repair the sites of perforation with a patch or sutures [14]. Careful hemodynamic and echocardiographic monitoring is necessary in both types of management (closed or surgical intervention) because delayed re-tamponade could develop as the site of perforation may not be fully closed or the surgeon may not have been able to identify or repair the location of the cardiac muscle perforation.

In the case of symptomatic heart perforation or lead malfunction without significant pericardial effusion, the lead may be removed manually by direct traction with the aid of a regular stylet, after retraction of the active fixation screw. This procedure should be performed in a hybrid operating room under TEE observation with a cardiac surgical team on standby. The leads may be repositioned to a new site [6].

Our cases exemplify the different aspects of clinical presentation, the diagnostic challenges, and treatment of lead perforation.

In the first case, the patient was pacemaker-dependent, so she became symptomatic after lead perforation and loss of ventricular capture. Chest CT definitively proved the RV lead tip was located in the pericardium. The lead was retracted and a new lead was implanted in the septum to reduce the risk of recurrence.

Our second case was asymptomatic and found incidentally during ICD analysis. Chest CT was the main tool used in the diagnosis of this complication. Considering the fact that the patient was asymptomatic, 6 months had passed since implantation at the time of diagnosis, and ICD lead retraction is not as simple and safe as the pacemaker lead, we abandoned the previous RV ICD lead and implanted a new simple pacing lead in the septum to perform the impaired function of the ICD lead (RV lead pacing) and used the coils of previous lead.

In the third case, chest radiography demonstrated the complication and there was no need for chest CT. Our treatment in this case was retraction of the RV lead because the patient had sinus node dysfunction rather than AV block and the atrial lead was working properly. In this case, we decided not to pose another risk (e.g., pneumothorax, hemothorax, lead dislodgement) to the patient by introducing a new RV lead. However, it should be noted that a single chamber atrial pacemaker is not a routine practice these days.

The fourth case was an example of a life threatening complication, i.e., tamponade. Our assumption was that retraction of the ICD lead may aggravate pericardial leakage; therefore, we performed this procedure in the cardiovascular hybrid room rather than the catheterization laboratory. Similar to the third case, there was no need for chest CT.

3. Conclusion

Late RV perforation is a rare but serious complication of PPM and ICD implantation. It may be completely asymptomatic or present as a life-threatening condition such as tamponade. ECG-gated non-contrast cardiac CT is one of the best diagnostic modalities. Optimal management is still controversial. Treatment for an incidentally detected, chronically perforating lead does not appear to be necessary. In symptomatic patients or cases with lead malfunction, most of the time, lead removal in the hybrid operating room can be safely performed. However, in cases with other visceral injuries, surgical intervention may be necessary.

Conflict of interest

All authors declare no conflict of interest (or financial relationships) related to this study.

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