Complication of Pacemaker Implantation: An Atrial Lead Perforation

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1. Introduction

The implantation of permanent pacemakers is increasing year by year, and these devices are constantly being improved. Atrial lead perforation is an infrequent but a critical complication of pacemaker implantation. Recently, there have been increasing reports on the complication with the advances made in imaging modalities, and moreover, computed tomography scans can reveal delayed atrial lead perforation even in asymptomatic patients. However, the details of this complication remain unclear, and it is difficult to decide an appropriate strategy for the patients. This chapter thus focuses on this rare but increasing complication of atrial lead following pacemaker implantation.

2. Overview of cardiac perforation following pacemaker implantation

Acute and late complications from pacemaker implantation occur in a variable percentage of patients, ranging 3.2% to 7.5% (Ellenbogen et al., 2003; Healey et al., 2006; Lamas et al., 2002). Cardiac perforation, which can lead to pericarditis, tamponade, or even death, is one of the important complications. The incidence of perforation after permanent pacemaker is reportedly between 0.3% and 1.2% (Aizawa et al., 2001; Ellenbogen et al., 2003; Mahapatra et al., 2005). Most patients with a perforation complain of chest pain, dyspnea, and hypotension, thus making such symptoms important clues to an accurate diagnosis. Abnormal sensing or pacing parameters, and abnormal signs in chest radiography or echocardiography also indicate cardiac perforation. Almost all such instances tend to occur within 1 month after surgery, and the extraction of the lead is recommended when it is identified (Khan et al., 2005). However, some reports have also described successfully managed cases without extraction (Henrikson et al., 2006; Mahapatra et al., 2005).

3. Predictors of cardiac perforation following pacemaker implantation

There are several independent predictors of cardiac perforation following permanent pacemaker implantation. Multivariate analysis of 4280 permanent pacemaker implantations at the Mayo Clinic revealed that the use of a temporary pacemaker, helical screw leads, and steroids are the independent predictors of a perforation, and elevated right ventricular systolic pressure is protective against perforation (Mahapatra et al., 2005). The risk of using screw-in leads has also been demonstrated in other case reports (Akyol et al., 2005; Dilling-
A recent review article proposed several candidates in addition to the risk factors of perforation; the type and the location of the leads, the heart muscle characteristics, anticoagulation therapy, patient age, gender, and body mass (Rydelwska et al., 2010). However, the Mode Selection in Sinus Node Dysfunction trial, which was a prospective randomized trial included 2010 patients with sinus node dysfunction, and a report by Laborderie et al., which was a retrospective study from a French institution, could not demonstrate any predictors for cardiac perforation after pacemaker implantation (Ellenbogen et al., 2003; Laborderie et al., 2008), and therefore the early prediction or identification of such conditions continues to be a challenge.

4. Active-fixation atrial leads

An atrial lead is essential for dual chamber pacing, but dislodgement of this lead is not infrequent (Ellenbogen et al., 2003; Lamas et al., 2002). In order to reduce the dislodgement rate, active fixation (screw-in) leads have been developed and have grown in popularity because of their reliability and the relative ease of placement at sites with the optimal pacing and sensing thresholds, adding to lower dislodgement rates. However, active fixation leads are associated with rare complications, including pericarditis, atrial lead perforation, pericardial effusion with or without cardiac tamponade, and death. The leads increase the chance of perforating the thin-walled right atrium, which averages 2mm in wall thickness (Hirschl et al., 2007), compared to passive fixation. Several risk factors may be responsible for the increased complication rate of screw-in leads (Srivathsan et al. 2003). Variations in the anatomy of the right atrium, such as an extremely thin-walled or multi-lobed atrial appendage may therefore play a role in the perforation. Previous reports have suggested that the implantation of active-fixation leads in the right atrial free wall is one of the risk factors responsible for increasing pericardial complications compared to the right atrial appendage, however a study that included 1021 consecutive patients demonstrated that the atrial lead tip in 3 of 4 cases of pericarditis after pacemaker implantation were directed anteromedially to the area of the right atrial appendage (Sivakumaran et al., 2002). A prospective randomized study showed a similar frequency of lead tip positioning in the right atrial appendage and lateral atrial wall among patients with pericardial complications (Luria et al., 2007). In addition, lead factors, such as the design and stiffness of the helix may differ between manufactures and could be important. The experience of the operator regarding pacemaker implantation is equally important. Over-screwing during atrial lead fixation, abrupt lead withdrawal without unscrewing, and distal positioning of the stylet while screwing should be avoided.

5. Differences among atrial lead types

Several types of atrial leads have been available, and active-fixation leads have advantages and disadvantages. However, there is limited data to compare the atrial leads for the choice of fixation (passive or active) or lead shape (J-shape or straight) (Van Herendael & Willems, 2009). A randomized comparison between 2 active-fixation, steroid-eluting, polyurethane-insulated, bipolar atrial lead models that differed only in shape (J-shape or straight) showed equally favorable performance profiles for 1 year of follow-up. Dislodgments were only reported in the straight lead group in 5.9% of cases, while no dislodgments occurred in the J-
shaped lead group. The rates of exit block and lead malfunction tended to be higher in the J-shaped group. Pericardial complications occurred in both groups in 1% of cases (Glikson et al., 2000). Moreover, those groups were followed over a 5-year period. Lead macrodislodgment occurred in the straight lead group in another 1.9% of cases during the additional follow-up, and lead malfunction and excessive pacing thresholds without dislodgment occurred in the J-shaped lead group in 10.7% of cases and in the straight lead group in 3.8% of cases (Luria et al., 2005). A prospective randomized comparison of the performance of J-shaped atrial leads with or without active-fixation revealed significantly lower pacing thresholds in the passive-fixation group at implantation, and this difference persisted at 1-year follow-up. The duration of fluoroscopy during the implantation procedure was significantly shorter in the passive-fixation group. Dislodgments were only reported in the passive-fixation group in 2% of cases, while pericardial complications occurred only in the active-fixation group in 6% of cases (Luria et al., 2007). Another report also showed early dislodgment requiring subsequent lead repositioning to occur in 2.4% of passive-fixation leads, but in none of the active-fixation leads. The incidence of pericarditis following implantation of J-shaped active-fixation leads was 5% (Sivakumaran et al., 2002). Passive-fixation leads are reported to have an excellent reliability and a very low incidence of atrial lead perforation (Glikson et al., 1999), while no difference in the J-shaped leads and straight leads in passive-fixation was demonstrated (Krupienicz et al., 2000).

6. Late lead perforation following pacemaker implantation

Late complications of pacemaker implantation that are well recognized include infection, failure of the atrial or ventricular lead to pace or sense appropriately, erosion of the pulse generator, and subclavian vein thrombosis. Delayed lead perforation has been defined as migration and perforation after one month of implantation. This complication has been reported to occur in 0.1-0.8% of pacemaker and 0.6-5.2% of implantable cardioverter defibrillator implantations (Khan et al., 2005; Polin et al., 2006), while recent progress in diagnostic imaging has increased the number of case reports on late lead perforation. Computed tomography is becoming a gold standard used for the diagnosis of a perforation, and asymptomatic perforation cases identified on the scans have been described extensively in the literature. A retrospective investigation of 100 consecutive patients with permanent pacemakers or implantable cardiac defibrillators who underwent multidetector computed tomography revealed that 15% of patients had a lead perforation, and the perforation rate of active- and passive-fixation atrial leads were 12% and 25%, respectively (Hirschl et al., 2007). This common phenomenon was confirmed by an autopsy study. Myocardial perforation or penetration by an electrode was recognized in 5.3% of 111 autopsy cases of patients 60 years of age or over with an implanted pacemaker. The perforation rate was 27.3% in active-fixation atrial leads, and 0% in 10 passive leads. All the atrial leads perforated through the right atrial appendage but did not reach the outside of the pericardium (Ishikawa et al., 1999). In the diagnosis of delayed lead perforation, failure of pacing or sensing of the lead is an important clue. A recent report revealed that detection of lead dysfunction by an automatic home-monitoring system had fast and possibly life-saving capabilities for severe lead perforation (Spencer et al., 2007). Usually, the lead parameters, in particular the pacing threshold, will show a significant change following lead perforation, while many reports have demonstrated normal electrophysiological parameters. Hirschl et al. showed
that perforated leads did not show significant difference from nonperforated leads in the impedance, and the pacing threshold of all the perforated leads except for one was categorized as low (Hirschl et al., 2007). A larger part of the electrode may have been in contact with the atrial myocardium, resulting in a lack of change in the lead parameters. Therefore, we should be aware that pacemaker malfunction may indicate perforation, but normal parameters do not exclude a perforation. Risk factors for late perforation have not yet been fully defined, although Polin et al suggested that active fixation leads and anticoagulation therapy may represent predictors for the long-term development of a perforation (Polin et al., 2006). Freedom from symptoms also does not exclude the possibility of there being a perforation, as almost all of the patients were asymptomatic. Therefore, an important question remains how we follow these patients with pacemakers and track down such delayed perforation cases. Interestingly, late lead perforation is characterized by a low rate of tamponade or death (Khan et al.; 2005), although the mechanism underlying subclinical late perforation has not been elucidated.

7. Management of a late lead perforation

A proper management strategy for a late lead perforation remains controversial and should vary among individuals. Altered pacemaker parameters and pericardial complications are the main factors that should be used to decide the strategy. Pacing or sensing failure requires lead repositioning or a new lead insertion for appropriate functioning of the pacemaker. Cardiac tamponade caused by lead perforation requires emergency percutaneous pericardiocentesis with placement of a drainage catheter. After the stabilization, percutaneous extraction in the operating room with echocardiographic monitoring during and/or after the procedure with the cardiosurgical team backup is one of recommended strategies that can be used instead of conventional open heart lead removal (Geyfman et al., 2007; Laborderie et al., 2008), although this management is classified as a class III indication in the Heart Rhythm Society expert consensus (Wilkoff et al., 2009). Khan et al. reported successful removal of atrial leads in 2 cases of delayed lead perforation with cardiac surgery backup (Khan et al.; 2005). On the other hand, Polin et al. reported that 4 of 5 patients with cardiac tamponade that occurred over 30 days after pacemaker implantation were successfully managed conservatively without lead manipulation at a mean follow-up of 31 months (Polin et al., 2006). Henrikson et al. also demonstrated that a patient with an asymptomatic atrial lead perforation 2 weeks after the implantation was doing well at 1-year follow-up without lead extraction (Henrikson et al., 2006). Although the conservative strategy seems to be reasonable for patients without need of lead repositioning, one case report showed chronic severe pericarditis following acute pericarditis after pacemaker implantation resulted in the lead extraction (Ellenbogen et al., 2002), and another reported an asymptomatic patient with a perforated atrial lead that had to have the lead removed 2 years after the pacemaker implantation (Trigano & Caus, 1996). In addition, a recent report demonstrated that a successful surgical repair in a patient with a perforated right atrial lead migration into the right lung 1 year after the replacement of atrial lead (O’Neill et al., 2010). Although there was no imaging evidence of perforation just after the implantation in these cases, the perforation might have occurred either during or soon after the operation, and thereafter develop progressively over a longer period. A lack of long-term follow-up data
remains an important concern that may influence the use of conservative management without lead extraction.

8. Our experience

We have recently reported an asymptomatic case of atrial lead perforation which developed 5 years after pacemaker implantation (Sadamatsu et al., 2009). The patient underwent a pacemaker implantation for sick sinus syndrome via the right subclavian vein (Fig.1A). Because of right breast cancer, the pacemaker and the leads were removed, and new ones were implanted via the left subclavian vein (Fig.1B). Although computed tomography scans, which we examined retrospectively, had already clearly demonstrated a perforation 9 months after the replacement (Fig.2B), another 3 years had passed until we actually noticed the complication because she remained asymptomatic and the lead parameters did not change. Transient pacing failure and the imaging findings (Fig.1EF) made us the diagnosis of perforation, but the patient was asymptomatic and the rhythm became atrial fibrillation. We therefore managed her condition conservatively by switching the mode from DDD to VVI at first. However, the lead perforation progressed (Fig.1GH, Fig.2CD) and, as a result, open surgery was performed to remove the lead. The lead penetrated the pericardium enclosed in fibrous adhesions. This case suggests that computed tomography for evaluating lead perforation is useful and may be the most effective modality for detection, especially at the earliest possible stage of this complication. Moreover, this case throws some doubt on the safety of conservative management without extraction, and also supports the use of a management algorithm (Ellenbogen et al., 2002; Geyfman et al., 2007), for either the extraction or repositioning of the perforated lead under either fluoroscopic or echocardiographic guidance.

9. Mechanism underlying the delayed pericardial complications

The precise mechanism responsible for such late progression remains unclear, and the mechanism(s) for delayed pericardial effusion and tamponade also have not been elucidated. Surgical and autopsy findings of late atrial lead complications did not reveal any perforation in the atrial wall, which had been observed in the imaging findings (Aizawa et al., 2001; Ishikawa et al., 1999; Kono et al., 2008). These observations seem consistent with the low rate of tamponade or death in late perforation and the successful management with repositioning of the perforated atrial lead. Several recent reports have led to some speculation about the mechanism of pericardial complications. One of the proposed causes is that atrial perforation with partial protrusion of the distal aspect of the fixation screw into the pericardial cavity causes pericardial irritation (Sivakumaran et al., 2002). Another possibility is torsion by the perforated helix on the visceral pericardium during cardiac contraction, which opens a perforation gap allowing for intermittent oozing of blood into the pericardial space out of the right atrium (Geyfman et al., 2007). In addition, a perforation might be sealed by a combination of the lead itself, muscle contraction, and fibrosis, because of the small cross-sectional area of the perforation and the low pressure of the right atrium (Hirschl et al., 2007). On the other hand, the atrial leads in some cases perforate the pericardium completely, and further perforation of the leads would harm other organs, such
as the lungs. It is also possible that constant mechanical pressure from the screw could also ultimately culminate in the occurrence of a sudden late myocardial perforation (Ellenbogen et al., 2002).

Fig. 1. Chest X-rays. Compared to the image before the replacement of the pacemaker (A), the image taken 1 week after the replacement (B) showed only a focal protrusion from the right side of cardiac silhouette (arrowheads) and the lateral image (C) did not show any abnormalities. The image taken 2 years later had no serial change (D), however the image from 3 years and 9 months later (a lateral view; F) clearly showed atrial lead protrusion, while the lead changed to tip-tilted on the frontal view (E). One year later, both of frontal (G) and lateral views (H) demonstrated the progression of atrial lead protrusion. The arrows indicate the distal tip of the atrial lead. (From Sadamatsu K, et al. (2009). Progressive atrial lead perforation developed 5 years after pacemaker replacement. J Cardiol, 53,150-153)

10. Conclusion and future research

Atrial lead perforation, especially late onset perforation, is an infrequent phenomenon. However, recent imaging modalities have revealed that the complication is not as rare as has been previously reported, and the available data and knowledge are limited. The use of active-fixation atrial leads is an independent predictor for cardiac perforation following pacemaker implantation, even though the dislodgment rate is low. In addition, active-fixation leads may also be a risk factor for late lead perforation. In the diagnosis of the perforation, patients are often asymptomatic, and the lead parameters are also normal in many cases. Computed tomography can reveal the perforated lead, however the imaging examination is not appropriate for the routine follow-up. Therefore an important question remains how we follow these patients with pacemakers and track down such perforation cases. Although the proper management strategy remains controversial, percutaneous
Fig. 2. Computed tomography. (A) Before the replacement of the pacemaker, the scan showed right breast cancer (asterisk) and an atrial lead (arrows). (B) Nine months after the replacement, the new atrial lead (arrows) was screwed in almost the same position as the previous one, and had perforated the right atrial appendage. (C, D) Four years later, consecutive scans clearly showed the progression of the perforated atrial lead (arrows). (From Sadamatsu K, et al. (2009). Progressive atrial lead perforation developed 5 years after pacemaker replacement. J Cardiol, 53,150-153)

extraction of the perforated lead in the operating room with a cardiosurgical team backup may be appropriate. Conservative management without extraction is also one of the proposed strategies, however, the long-term safety of this strategy is unknown. Therefore, we should be aware of the potential occurrence of late atrial lead perforation in daily practice for pacemaker follow-up, and large multicenter investigations with long-term follow-up are needed to clarify the details and outcome of this complication.

11. References


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patient management: this document was endorsed by the American Heart Association (AHA). *Heart Rhythm*, 6, 1085-1104

The book focuses upon clinical as well as engineering aspects of modern cardiac pacemakers. Modern pacemaker functions, implant techniques, various complications related to implant and complications during follow-up are covered. The issue of interaction between magnetic resonance imaging and pacemakers are well discussed. Chapters are also included discussing the role of pacemakers in congenital and acquired conduction disease. Apart from pacing for bradycardia, the role of pacemakers in cardiac resynchronization therapy has been an important aspect of management of advanced heart failure. The book provides an excellent overview of implantation techniques as well as benefits and limitations of cardiac resynchronization therapy. Pacemaker follow-up with remote monitoring is getting more and more acceptance in clinical practice; therefore, chapters related to various aspects of remote monitoring are also incorporated in the book. The current aspect of cardiac pacemaker physiology and role of cardiac ion channels, as well as the present and future of biopacemakers are included to glimpse into the future management of conductions system diseases. We have also included chapters regarding gut pacemakers as well as pacemaker mechanisms of neural networks. Therefore, the book covers the entire spectrum of modern pacemaker therapy including implant techniques, device related complications, interactions, limitations, and benefits (including the role of pacing role in heart failure), as well as future prospects of cardiac pacing.

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