Early Detection of Perforation of the Right Ventricle by a Permanent Pacemaker Lead

Hye Kyung Park, MD, Hye Seung Ahn, MD, Ban Suck Lee, MD, Hye Jin Won, MD, Young Sup Byun, MD, Choong Won Goh, MD, Byung Ok Kim, MD, Kun Joo Rhee, MD and Byoung Kwon Lee, MD

Division of Cardiology, Department of Internal Medicine, Sanggye Paik Hospital, Inje University Medical College, Seoul, Korea

ABSTRACT

Ventricular perforation is a rare complication of permanent cardiac pacemaker implantation. We report here on a 68-year-old woman with a dual chamber permanent pacemaker that had been implanted one month earlier, and she suffered cardiac perforation from the pacemaker lead. Frequent follow-up via 12-lead surface electrocardiography and chest radiography and the proper work-up for pacemaker implantation are needed for detecting rare complications after pacemaker implantation. (Korean Circulation J 2007;37:453-457)

KEY WORDS: Cardiac pacemaker; Complications.

Introduction

Myocardial perforation is a rare complication following pacemaker implantation with using contemporary leads.1-3) Patients with myocardial perforation that is related to the pacemaker electrodes may present with various symptoms, from being asymptomatic to displaying cardiac tamponade. While most of these perforations have been reported to occur within one month after implantation, delayed myocardial perforation is very rare.4) We report here on one case of finding on follow-up a right ventricular perforation that was caused by the ventricular electrode one month after uneventful implantation of a permanent pacemaker. We surgically removed the electrode and repaired the perforated right ventricular wall.

Case

A 68-year-old woman was admitted at our hospital because of her syncope. She had hypotension with a blood pressure of 80/40 mmHg and her electrocardiograph (ECG) showed junctional rhythm without a sinus p wave and the longest pause was 3.45 sec (Fig. 1A, B). Therefore, we diagnosed her with sick sinus syndrome and she underwent implantation of a dual chamber permanent pacemaker (Medtronic Kappa KD903, USA) for treating her sick sinus syndrome. Active fixation lead (Medtronic 4068, USA) and passive fixation lead (Medtronic 4092, USA) were used for the atrial and ventricular leads, respectively. The routine laboratory findings, including complete blood cell counts and blood chemistry, were all normal. Her echocardiogram showed normal chamber sizes, a normal left ventricular ejection fraction (LVEF) and no regional wall motion abnormality. No significant coronary disease was noted on the coronary angiogram. Three days after the procedure, she was discharged without any complications.

Chest radiographs showed normal positioning of the electrodes until 32 days after the procedure (Fig. 2A-D). Yet we found that the sensitivity of the ventricle leads until 16 days after the procedure was decreased from 11.2 to 4.8 mV, and the pacing threshold during the period progressively increased from 0.25 to 4.0 V (Table 1). So, we changed her pacemaker mode to AAI on post-procedure day 16, and the sensitivity and pacing threshold of the ventricle leads was not improved on the following check-up. Therefore, we decided to reposition the ventricular lead. On post-procedure day 36, she complained palpitation and chest discomfort. Chest radiographs showed that the ventricular lead was straightened and displaced left and laterally downward (Fig. 3A, B). ECG showed normal ventricular function without any other
related findings. The sensitivity of the ventricle lead was 4.0 mV and pacing was not captured (Table 1). The chest computed tomography (CT) scan demonstrated clear evidence of migration of the ventricular lead out of the heart onto the chest wall and diaphragm (Fig. 4A, B). We surgically removed the ventricular lead that had penetrated through the RV free wall and protruded 2 cm out of the diaphragmatic reflection of the pericardium along the apex of the RV free wall side. Interestingly, the pericardial space was clear without any effusion or hema-

![Fig. 1](image1.png)

**Fig. 1.** The initial electrocardiograph (ECG) revealed junctional rhythm without a sinus p wave (A) and the longest pause was 3.45 sec (B).

![Fig. 2](image2.png)

**Fig. 2.** The chest posteroanterior and lateral films showed no significant interval change of the dual-chamber pacemaker electrodes, with the tip of each lead positioned in the right atrium and right ventricle, between two days after the procedure (A, B) and post-procedure day 32 (C, D). PA: posteroanterior, Lt: left.
After disconnecting from the generator, gentle and steady traction resulted in lead extraction to an outward direction from the RV apex. On her preoperative EKG, the AV conduction was normal (Fig. 6), so a new ventricular lead was not implanted at that time. The AAI mode for the pacemaker was maintained. The patient was discharged after surgical removal of the lead. She has been being well without any complication during the two year follow-up period.

**Discussion**

Pacemakers and implantable cardioverter-defibrillators are used to treat a variety of heart rhythm problems. The usual complications for most patients undergoing implantation of these devices are ecchymosis, hematoma at the incision site and mild chest discomfort, but these are usually temporary. But the procedure-related major complication such as perforation of the major arteries or of the heart itself, pneumothorax and sepsis can be fatal complications. Lead perforation is a rare complication with an incidence of 0.3% to 1.2% and moreover, late perforation is much more uncommon. Early or delayed perforation of a ventricle might cause clinical symptoms. Dyspnea, palpitation or chest pain due to pericardial effusion, hemothorax and extracardiac muscle stimulation can develop. Also, the failure to pace or sense appropriately, and also ventricular arrhythmia might cause syncope or sudden cardiac death. However, a review of the medical literature showed that...
perforation may not have any symptoms. Chest discomfort and palpitation for a few seconds at the one month follow-up visit were the only symptoms in our case.

In cases of suspected lead perforation, many physicians can usually use work-up tools such as device analyzers, chest radiographs, ECG, echocardiography and fluoroscopy. The combined use of these devices will usually allow a physician to arrive at a convincing diagnosis.

There was no abnormality on the follow-up ECG and chest radiographs, and there were no clinical symptoms during the first two weeks in our case. Two weeks after this, we detected the change of the sensitivity and pacing threshold of the ventricular lead. The usual interval of the follow-up schedule for patients with a new pacemaker is between two weeks and one month after the procedure. But we changed the follow-up visits to a weekly schedule to check her pacemaker function and found the ventricular perforation five weeks after pacemaker implantation. Therefore, frequent check-ups for examining the pacemaker device with short regular follow-up intervals under a high index of suspicion would be recommended for the early detection of pacemaker dysfunction. Noticeable position change on the chest radiography may not be possible even after finding failure to pace. computed tomography (CT) can be a very useful adjunct to the other imaging modalities during the evaluation of possible lead perforation. Also, in the case of planning surgical treatment, CT scan can give exact information on lead location and its relation to adjacent organs.

The reasons for perforating a ventricle by the pacemaker lead might arise from many factors related to the patients or the procedure. The factors related to patients are mainly due to a weakened ventricle from dilated cardiomyopathy, ischemic cardiomyopathy or other myocardial inflammatory disease. A tough manipulation of the myocardial leads during pacemaker implantation belongs to the procedure-related factors when using a stiff stylet or an active fixation type lead. It assumed that lead perforation occurred in our patient because the movement of the ventricular lead near the right ventricular free wall would give an axial force to the apex when the right heart contracts. We suggest that a perforating ventricular lead should be removed even there is no other complication because there is the potential risk of cardiac tamponade.

This case shows the potential for ventricular perforation by the pacemaker lead even after uneventful implantation of a passive fixation lead. It also reveals that late perforation by the pacing leads might occur without pacing electrode stimulation of the chest musculature or without any pericardial effusion being present. A frequent follow-up schedule might be recommended when abnormal findings are noted on the immediate results of medial testing. We suggest for such cases as ours that a high index of suspicion is important even when the routine chest radiograph and surface ECG are normal.

Fig. 5. The thoracotomy view showing the exposed pericardial sac. The tip of the ventricular lead (arrow) protruded through the right ventricle apex without causing pericardial effusion or hematoma.

Fig. 6. The AV conduction was normal (Fig. 6. ECG). AV: atrioventricular, ECG: electrocardiograph.
REFERENCES


