Delayed Pericardial Effusion Due to Perforation of the Right Ventricular Outflow Tract by an ICD Lead

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Abstract

A delayed pericardial effusion developed in a recipient of a cardioverter defibrillator (ICD). After an uneventful implant procedure and postoperative recovery, the patient suffered loss of appetite and fatigue, and was re-admitted to the hospital 48 days later. Her vital signs were stable and cardiac silhouette on chest roentgenogram was normal. However, blood cell counts and chemistry revealed the presence of anemia and liver dysfunction, an echocardiogram showed a diffuse pericardial effusion, and computed tomography suggested that the ICD lead, screwed in the right ventricular outflow tract, had perforated the wall. In order to make a prompt diagnosis and initiate timely corrective treatment, the physician in charge of long-term follow-up should remember that a pericardial effusion can be delayed and accumulate in the absence of typical signs of cardiac tamponade after ICD lead implantation.

Key words: implantable cardioverter defibrillator, lead perforation, pericardial effusion

(Inter Med 49: 389-392, 2010)
(DOI: 10.2169/internalmedicine.49.2936)

Introduction

The positive results of recent clinical trials have expanded the indications for implantation of cardioverter defibrillators (ICD) (1, 2), and the number of ICD recipients is increasing worldwide. Lead perforation, a rare complication of ICD and pacemaker implantation, usually occurs during or soon after the procedure, and is typically associated with manifestations of cardiac tamponade (3, 4). We recently observed a patient who suffered from delayed accumulation of a pericardial effusion due to perforation of the right ventricular (RV) outflow tract (OT) by an ICD lead. The patient returned to our hospital 48 days after ICD implant, complaining of fatigue and loss of appetite.

Case Report

A 42-year-old woman was re-admitted to our hospital. She had been treated elsewhere with atrovastatin for hyperlipidemia for approximately 3 years before she began suffering from daytime palpitations and fainting, occurring once or twice a week. In October 2007, she visited another hospital for recurrent palpitation, and a diagnosis was made of sustained, monomorphic ventricular tachycardia (VT) at a cycle length of 330 ms, with right bundle branch block pattern and inferior axis QRS morphology. She was transferred to our hospital, where the VT was terminated with intravenous procainamide. Her height was 162 cm, body weight 38 kg, resting blood pressure 96/62 mmHg, heart rate 58 bpm, and her physical examination was normal. Echocardiogram, blood cell count and chemistry, and 12-lead electrocardiogram (ECG) were also normal (Fig. 1A). No late potentials were observed on signal-averaged ECG.

Cardiac catheterization and electrophysiologic studies were performed with her informed consent. Intracardiac pressures, coronary angiograms, right and left ventriculograms were normal, and the left ventricular ejection fraction was 60%. AH and HV intervals during sinus rhythm measured 65 and 45 ms, respectively. Sinus node function and atrioventricular conduction were normal. Sustained VT with the same QRS morphology as that observed during

Received for publication September 24, 2009; Accepted for publication November 10, 2009
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clinically documented tachycardia was induced by programmed RV extrastimulation, with an immediate fall of the systolic blood pressure to <60 mmHg. Intravenous infusion of isoproterenol facilitated the induction of the VT and shortened its cycle length. The earliest activation during VT was mapped to the antero-lateral portion of the mitral annulus, with a local endocardial electrogram recorded 15 ms prior to the onset of the QRS complex, and an epicardial electrogram recorded via the coronary sinus, 30 ms before the QRS onset. Radiofrequency current (50-55°C, 30-50 W) was applied three times unsuccessfully at the endocardial site. No attempt was made to ablate the VT from inside the coronary sinus or from the epicardial surface.

Treatment with mexiletine, 125 mg i.v., and verapamil, 120 mg p.o. daily, were ineffective or intolerable, and the patient underwent implantation of an ATLAS+DR dual chamber ICD (St. Jude Medical Inc., St. Paul, MN) connected to models 1580/60 RIATA® RV and 1642T/52 ISOFLEX S® right atrial (RA) leads (St. Jude). Since this patient was a thin, small Japanese woman, we advanced the ICD lead into the RVOT in order to obtain a more suitable position of the RV and SVC defibrillation coils in the heart. We first attempted to place the ICD lead tip at the septum of the RVOT, but stable placement was unobtainable probably due to the small heart of this patient. Therefore, the RV lead tip was screwed in the anterior aspect of the OT, and the RA lead was placed in the appendage (Fig. 2A). The R wave amplitude was 10.3 mV, RV capture threshold 1.5 V/0.4 ms, RV lead impedance 365 ohms, P wave amplitude 1.3 mV, RA capture threshold 0.5 V/0.4 ms, and RA lead impedance 490 ohms.

She recovered completely from the procedure and was discharged from the hospital 2 weeks after ICD implantation on a regimen of carvedilol. She returned to our hospital approximately 1 month later, complaining of fatigue and loss of appetite. Her blood pressure was 92/63 mmHg and pulse rate 63 bpm, similar to those observed during her previous admission. However, hematological examinations revealed the presence of anemia (red blood cell count: 300×10⁴ mm³; hemoglobin: 8.8 g/dL), and blood chemistry showed liver dysfunction (GOT 196 IU/L, GPT 307 IU/L, LDH 309 IU/L, ALP 451 IU/L, γ-GTP 103 IU/L). The ECG was similar to that recorded before ICD implantation (Fig. 1B), and a chest roentgenogram showed an increase in the cardiopulmonary ratio from 38 to 43% (Fig. 2B). An echocardiogram showed a diffuse, 2.0 cm wide pericardial effusion (Fig. 3A), and computer tomography suggested that the tip of the ventricular lead had penetrated the pericardial space (Fig. 3B).

Interrogation of the ICD memory showed the presence of multiple VT recurrences, successfully terminated by antitachycardia pacing. The R wave amplitude was 8.0 mV, RV

Figure 1. Twelve-lead electrocardiogram. ECG A and B were recorded at the time of 1st and 2nd admission to our hospital, respectively. Except for the non-specific T wave flattening present on the 2nd admission, the traces were normal and similar.
Figure 2. Posterior-anterior and left lateral chest roentgenograms. A: Obtained during the 1st hospitalization, after ICD implantation, with the RV lead placed in RVOT. B: Obtained at the time of 2nd admission. The cardio-thoracic ratio had increased from 38 to 43%.

Figure 3. A: Echocardiogram shows a diffuse pericardial effusion (arrow). B: Computed tomography of the heart suggests that the tip of the lead (arrow) has perforated the RV wall.

capture threshold 2.0 V/0.4 ms, RV lead impedance 395 ohms, P wave amplitude 2.1 mV, RA capture threshold 0.5 V/0.4 ms, and RA lead impedance 470 ohms. At the time of surgical repair, the tip of the RV lead had advanced onto the epicardial surface. Dark colored bloody effusion in the amount of 400 mL was drained from the pericardial space. The RV and RA leads were removed and the injured myocardium was repaired. A new ICD system was re-implanted 6 weeks later. Since the surgical repair was performed in an OT area, a new ICD lead was screwed into position at the septal site of the RV apex. Over a follow-up of 18 months, the patient has remained clinically stable and asymptomatic, and her laboratory tests have normalized.

Discussion

This case illustrates 2 important observations pertaining to the follow-up of ICD recipients. First, the delayed accumulation of pericardial effusion developed without typical manifestations of cardiac tamponade. Second, an ICD lead
had perforated the RVOT.

Previous studies have reported a 0.5-2.0% incidence of lead perforation associated with implantable pacing devices (3, 4). However, the risk of this complication seems to have increased with the development of recent lead models (5, 6). Symptoms and signs of lead perforation vary depending on the time course and amount of pericardial effusion. Acute pericardial effusion, which occurs during or pending on the time course and amount of pericardial effusion, can cause overt manifestations of cardiac tamponade. On the other hand, patients suffering from a slow accumulation of pericardial effusion may remain asymptomatic, complain of mild symptoms, including dyspnea, cough and chest pain, or develop edema, pacing/sensing failure, or inappropriate ICD discharges (7, 8).

Hirschel et al recently reported lead perforations confirmed by computed tomography in 15% of asymptomatic patients treated with implantable cardiac pacing devices (9).

The present patient complained of loss of appetite and had liver dysfunction, apparently due to an abnormal venous return to the heart. The general fatigue and anemia were probably due to the slow blood loss into the pericardial space. Although neither pacing or sensing failure, nor inappropriate ICD discharges were observed in this patient, mild enlargement of the cardiac silhouette on the chest roentgenogram was another sign of pericardial effusion. Since she was asymptomatic and had normal laboratory examinations when first discharged from the hospital, a slow progression of the lead through the RV myocardium during daily activity seemed to be the probable cause of the delayed accumulation of pericardial effusion.

Perforation is a rare complication following placement of an ICD lead in the RVOT, compared with lead implantation at the RV apex (10, 11). To minimize the risk of perforation, attempts should be made to place the lead in the interventricular septal region, although this may be a challenge in the presence of a small heart, as in the present case. The design, structure, diameter and material of the lead are other factors that may be associated with an increased risk of lead perforation. Recent reports have shown a relatively high incidence of lead perforation with the RIATA series of ICD leads, which was implanted in our patient (12). However, other studies have reported that the incidence of ICD complications in RIATA series was similar to other lead models (13, 14). ICD lead perforation is difficult to anticipate during the operation, and gentle lead manipulation is essential to prevent this complication. Monitoring of the unipolar electrogram from the tip electrode seems to be useful to reduce the risk of perforation; we have used such monitoring during the ICD operation in all cases.

In the present patient, an ICD was implanted for the management of repetitive episodes of palpitation and fainting due to hemodynamically unstable, idiopathic VT of epicardial origin, reproduced at electrophysiological study (15). Since the VT origin was initially believed to be located in the epicardial region, cryoablation would have been preferable to have attempted during the repair operation of the lead perforation. However, such an operation was not performed in this case. Implanting physicians must remember that pericardial effusion due to lead perforation may develop without typical manifestations of cardiac tamponade during the long-term follow-up after ICD implantation.

References