Right Coronary Artery Fistula as a Result of Delayed Right Atrial Perforation by a Passive Fixation Lead

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Delayed lead perforation (DLP) is an uncommon complication of permanent pacemaker and defibrillator implantation, especially that of the right atrium (RA).1 Lead perforation is considered delayed when it occurs >30 days after implantation. The incidence of DLP has been reported to be ~0.8% and is more common in elderly individuals.1,2 With the yearly increase in implanted devices, and the advance in imaging modalities, this complication is more likely to be encountered. To our knowledge, delayed right atrial perforation involving a passive fixation lead remains extremely rare and has never been reported. We describe a case of an asymptomatic right atrial DLP resulting in a pseudoaneurysm and right coronary artery (RCA) fistula, diagnosed incidentally 53 months after the implantation of a passive atrial lead.

Our patient is a 67-year-old man with a medical history of hypertension, diabetes mellitus, end-stage renal disease on hemodialysis, coronary artery bypass grafting, and ischemic cardiomyopathy. The patient had a dual-chamber implantable cardioverter-defibrillator implanted for primary prevention of sudden cardiac death. A passive fixation lead (5594 CapSure SP Novus, Medtronic; Indianapolis, IN) was implanted in the RA appendage, and the right ventricular lead (6949 Sprint Fidelis, Medtronic) was implanted in the apical septum without difficulty. The initial atrial pacing threshold was 0.5 V at 0.4 ms and 1.5 V at 0.4 ms for the ventricular lead. The initial parameters remained unchanged at regular 4-month follow-ups for the first 2 years. At the 2-year follow-up visit, the RA pacing threshold increased to 1 V at 0.5 ms and progressed to 1.5 V at 0.5 ms 3 months later. A serial chest x-ray film did not show any change in the lead position during or after this period (Figure 1). Unknown to us, the patient underwent a noncontrast computed tomographic (CT) scan of the chest in the pulmonary clinic for pulmonary nodules during the same period. The scan showed an ~4.5-cm mass at the level of the atrioventricular groove in the vicinity of the right atrial lead.

During a regular visit 4 years after implantation, implantable cardioverter-defibrillator telemetry showed long episodes of nonsustained ventricular tachycardia. Because of this finding, the patient underwent elective coronary angiography to assess graft patency. Coronary angiography revealed total occlusions of native coronary vessels and an occluded saphenous graft to the RCA. While injecting the native RCA, an unusual filling mass was noticed. This mass was ~7×4 cm, had a saclike appearance with slow clearance of the contrast, and appeared to communicate with the RCA through a fistula. The right atrial lead of the implantable cardioverter-defibrillator appeared to be in contact with the mass on cine angiography images (Figure 2). A multidetector cardiac CT scan confirmed that the lead tip was perforating the RA and protruding into the anterior margin of the mass. The mass measured 7.1×5 cm on this CT scan.

Because of the interval increase in the size of the mass, the patient underwent surgery. An intraoperative transesophageal echocardiogram further defined the mass (Figure 3). On surgical exploration, the mass was confirmed to be a right atrial perforation, with a pseudoaneurysm secondary to lead perforation (Figure 4). The surgical procedure consisted of ligation of the RCA, evacuation of the pseudoaneurysm from the thrombus, resection of the distal right atrial lead, and closure of the right atrial perforation. The patient did well postoperatively.
Discussion

Several predictors of lead perforation have been reported, including age, sex, body mass, type and location of leads, and anticoagulation therapy. Risk factors for late perforation are less understood, although active fixation (screw in) and long-term anticoagulation remain as possible culprits. Active atrial fixation leads have grown in popularity because of the advantage of the decrease in the dislodgment rate. However, these leads carried a higher risk of perforation when compared with passive fixation. Conversely, there are few reports suggesting similar perforation rates for both active and passive leads. In our patient, the passive fixation atrial lead was implanted without apparent dislodgment by chest x-ray film.

The diagnosis of atrial DLP is probably underreported because most patients remain asymptomatic. A change in pacing threshold and impedance can help in the diagnosis, but several studies have shown no significant difference in these parameters between the perforated and nonperforated leads. Although the pacing thresholds gradually increased in our patient, the absence of symptoms and no change in lead position on chest x-ray film delayed the diagnosis significantly. The initial CT findings at 2 years after implantation, which coincided with increasing atrial threshold, should have raised some suspicion of lead complication; however, we were unaware of the CT scan findings at that time.

Pericardial irritation is reported as the mechanism of DLP by active fixation leads. In our case of passive fixation lead, the mechanism is not clear. We believe that the lead must have eroded into the right atrial wall at the AV groove in the vicinity of the RCA. Repetitive mechanical irritation of the lead at such a position led to the fistulous connection with the RCA. Because of the low pressure in the RA and the small cross-sectional area of the perforation, progression of the mass was slow.

The management of asymptomatic DLP remains controversial. Although few reports showed the safety of a conservative management, others demonstrated unfavorable outcome. Fortunately, the rate of tamponade and death is low with this type of perforation. In our case, the RA mass significantly increased in size, which argues against the conservative approach.

In conclusion, DLP of the RA with a passive fixation lead has never been reported. The diagnosis of DLP is challenging because of lack of symptoms and low sensitivity of the usual imaging modalities, such as chest x-ray film. However, with the advances in imaging technology, this entity should be more readily detected. The increase in pacing threshold in the absence of symptoms and no change in lead position on chest x-ray film may raise the suspicion of DLP. Further testing to exclude DLP can prevent serious consequences, especially if repositioning or extraction of the lead is required.

Disclosures

None.

References


Key Words: delayed lead perforation ■ passive fixation leads ■ pseudoaneurysm ■ coronary fistula ■ pacing thresholds
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