Case presentation

We present the case of an 82-year-old woman with longstanding hypertrophic obstructive cardiomyopathy (HOCM). The patient began to suffer from effort dyspnea in 2008. During the same year she had several episodes of rapid paroxysmal atrial fibrillation (PAF). She was treated with various drug combinations (β-blocker, verapamil, and amiodarone) that failed to prevent PAF recurrence. During 2009 her clinical condition deteriorated and she started suffering from dyspnea during mild exercise. In August 2009 she received oral disopyramide in an attempt to control the PAF and reduce the left ventricular outflow tract (LVOT) gradient. However, the gradient did not decrease and recurrent PAF still occurred, while marked QT prolongation (up to 500 ms) was observed that prompted drug discontinuation. In addition, due to marked sinus bradycardia, she underwent implantation of a dual-chamber pacemaker in August 2010. Eight days following pacemaker implantation, she complained of extreme weakness and dizziness that was attributed to asynchronous right ventricular (RV) pacing. Atrioventricular delay (AVD) optimization did not improve the LVOT gradient, and the pacemaker was programmed to DDD mode (50/min) with AVD of 400 ms to reduce the contribution of RV pacing to the minimum. The patient still remained severely disabled and agreed to undergo transcatheter ablation of septal hypertrophy (TASH). Baseline PR interval was 185 ms, and the QRS complexes were narrow. Baseline echocardiogram showed normal LV chamber size, hypercontractility of the hypertrophic heart with left ventricular ejection fraction of 65%, posterior wall thickness of 14 mm, and intraventricular septum thickness of 20 mm. LVOT gradients were 80–110 and 130–140 mmHg during rest and during Valsalva, respectively. Coronary
angiography showed irregular arteries without significant obstruction. On September 28, 2010, she underwent direct alcohol injection to the second septal branch. This was followed by chest pain for a few minutes and resulted in immediate decrease in LVOT gradient (100, 150, and 190 mmHg during rest, Valsalva and postventricular extrasystolic beat, respectively, before TASH compared with 15 mmHg during the ischemic period and post-alcohol injection). Post-procedure peak creatine phosphokinase (CPK) and troponin I were 1234 U/l and 3.09 ng/ml, respectively. The post-TASH electrocardiogram showed sinus rhythm with prolonged PR interval (248 ms), right bundle branch block (RBBB), QRS width of 158 ms with additional STT changes. The echocardiogram performed the day after TASH revealed a septum thickness of 22 mm and a LVOT gradient of 54 and 98 mmHg during rest and Valsalva, respectively. A few hours after the procedure, the monitor recording at the intensive cardiac-care unit (ICCU) and pacemaker interrogation showed pacemaker behavior of undersensing of the RV electrogram (Figure 1a). The pacemaker interrogation (Figure 1b) revealed an R wave of 0.8 mV, below the programmed RV sensitivity of 2.5 mV and much lower than the R-wave value 1 day before the procedure (10.7 mV). The RV impedance fell from 490 ohm before TASH to 340 ohm after TASH (Figure 1b) while the pacing threshold was stable at 0.8 mV. The atrial parameters did not change. The pacemaker was programmed to AAI mode, and the patient was closely monitored for the next 10 days. An echocardiogram performed 10 days after the TASH revealed septum thickness of 19 mm with LVOT gradient of 10 and 15 mmHg during rest and Valsalva, respectively. On the last echocardiography (June 13, 2011) the LV ejection fraction was 65%, the septum and LV posterior wall thickness were 17 and 14 mm, respectively and the LVOT gradient was 16 mmHg. During the last visit to the pacemaker clinic (September 13, 2011), she reported a marked clinical improvement, and for the first time the R wave started to rise to 3 mV (see details in Figure 1c).

Commentary

HOCM is characterized with dynamic LVOT obstruction. A subset of patients with HOCM may develop symptoms of dyspnea, angina, and syncope, and present an increased risk of heart failure and sudden death. For those patients who have severe LVOT obstruction and drug-refractory symptoms, dual-chamber pacemaker implantation and surgical or non-surgical septal reduction therapy have been shown to be effective. TASH has shown an efficacy similar to surgical septal myectomy.6 During TASH, an injection of ethanol into one or two septal branches induces localized septal myocardial infarction, which results in immediate LVOT gradient reduction due to septal dysfunction followed by septal thinning and gradual continuous reduction of the LVOT gradient.8 However, TASH is associated with a relatively high complication rate (15–50%).9 The most frequent complication is complete heart block requiring permanent pacemaker implantation.6,8,9,11,12 TASH may also result in serious and fatal complications, including acute myocardial infarction, coronary dissection, tamponade, ventricular arrhythmias, infective endocarditis, and death.6,8–12

Our patient underwent the TASH procedure after a DDD pacemaker that was implanted due to severe sinus node dysfunction and that DDD pacing failed to reduce LVOT gradient. In contrast, the TASH procedure resulted in marked clinical and hemodynamic improvement. The procedure was complicated by the occurrence of RBBB with first-degree AV block. In addition, our patient developed a unique pattern of loss of RV sensing post-TASH procedure without significant changes in the impedance or the pacing threshold of the RV lead. To our knowledge this is the first report documenting this complication. The loss of sensing appears a few hours after the procedure (Figure 1a,b), the R wave dropped from 10.7 mV to 0.8 mV, leading to pacemaker behavior of RV malsensing. The RV impedance dropped from 490 to 340 ohm (Figure 1b,c), and further stabilized (Figure 1c); the pacing threshold did not change (0.8 V). The RV loss of sensing might have occurred due to migration of the ethanol injected in the second septal branch to the RV apex area, where the RV lead tip was located (Figure 2). It was shown that coronary collateralization can develop immediately after a brief coronary occlusion of a normal artery.13 Agarwal et al.14 reported a case of apical myocardial injury caused by collateralization of the septal artery during ethanol septal ablation. In that case, the ethanol migrated due to collateralization between the first septal branch and the distal left anterior descending (LAD) coronary artery that led to apical myocardial injury. It might be that in the present case a collateralization of a second septal branch of the LAD coronary artery to the distal right coronary artery resulted in local myocardial injury around the RV lead tip and loss of the local electrogram (Figure 2). The mechanism by which the RV resulted in a loss of sensing without significant changes in the impedance or the pacing threshold remains unclear. Testing the electrode with both bipolar and unipolar configurations reveals the same pattern of loss of RV sensing without any changes in RV impedance or pacing threshold, locating the RV lead failure to the distal pole and RV tissue contact.

The proposed mechanism of complete heart block post ethanol septal ablation is injury of the His–Purkinje system.11 In our case the loss of R-wave sensing may be related to further injury around the RV lead tip. Faber et al.15 suggested DDD pacemaker implantation prior to ethanol septal ablation in patients with HOCM and left bundle branch block. Although our case demonstrates just loss of sensing, it might be that TASH could also result in a loss of capture in already implanted patients that may necessitate reposition or reimplantation of the RV lead in case of post-ethanol septal ablation atrioventricular block (AVB).

Finally, Figure 1c demonstrates the R-wave fluctuation during follow-up. It is evident that the R wave post-TASH ranged from 0 to 1.4 mV during the 8 months of follow-up, but for the first time climbed to 3 mV (above the routine programmed ventricular sensitivity) at the
Figure 1: Pacemaker interrogation of long-term P and R wave and right atrial and right ventricular (RV) lead impedance. (a) Pacemaker electrogram recording demonstrates inappropriate ventricular pacing due to loss RV lead sensing due to dropped R wave. (b) Interrogation on January 6, 2011 shows the R wave drop to 0.8 mV just after the transcoronary ablation of septal hypertrophy (TASH) procedure. (b) Interrogation on September 13, 2011 shows the low voltage of the R wave (below 1.5 mV) for the first time on August 17, 2011 (8 months after the TASH procedure), an R wave of 3 mV that stayed stable afterwards.
Figure 2: Fluoroscopy of the left coronary system. (a) Before the transcoronary ablation of septal hypertrophy (TASH) procedure, a big septal branch bifurcated to three sub-branches. (b) Post-TASH procedure, an occluded first sub-branch of the septal artery is shown.
patient’s last visit. This observation may suggest a regenerative process in the injured area around the RV lead tip.

In summary, our present case report describes the first case of impaired RV lead tip and RV tissue contact occurring after ethanol septal ablation. This complication was characterized by lead sensing loss without significant effect on the impedance and the pacing threshold values of the RV lead. We conclude that in situ devices (pacemakers and implantable cardioverter-defibrillators) should be interrogated post TASH, as alterations in sensing and possibly pacing threshold may occur, even if the lead is far from the targeted septal perforator.

References