Case Report

Sinus node dysfunction with interatrial conduction delay observed after left atrial myxoma resection through the superior septal approach

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abstract
We report on a 64-year-old female patient who underwent cardiac surgery for left atrial myxoma, using the superior septal approach with large atrial septal wall resection and patch closure. The superior septal approach is reported to be a relatively safe method for preventing the development of sinus node dysfunction after cardiac surgery. However, this patient developed sinus node dysfunction after surgery and required the implantation of a permanent pacemaker. Moreover, in this case, determining the appropriate positions of the pacemaker leads was difficult because of the presence of a large conduction delay in the interatrium. Selecting the appropriate atrioventricular delay settings was important in order to achieve proper sequential contractions between the left atrium and the left ventricle.

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1. Introduction

Left atrial myxoma is the most frequently occurring type of heart tumor [1–3]. Patients often require pacemaker implantation after cardiac surgery for treatment of myxomas [1–3]. The superior septal approach (SSA) is one of the surgical approaches used for myxoma resection. Cases that require pacemaker implantation after SSA have been reported to be very rare [4,5]. We herein describe the case of a patient who required pacemaker implantation after surgery with the SSA for the treatment of a left atrial myxoma. Moreover, this case involved a complex endocardial electrogram that affected the pacemaker implantation procedure.

2. Case report

The patient was a 64-year-old woman with a history of hypertension, who experienced a near-syncopal episode after developing transient palpitations and dyspnea. A 12-lead electrocardiogram (ECG) (Fig. 1b) and a Holter ECG recorded upon her arrival at our hospital showed the presence of a normal sinus rhythm with no significant arrhythmias. Normal sinus nodal function and normal atrioventricular (AV) node conduction were estimated according to the Holter ECG findings of a maximum heart rate of 150 bpm and maximum 1:1 AV conduction rate of > 120 bpm. In addition, coronary angiography showed no significant stenosis. However, the echocardiographic findings revealed the presence of a large left atrial tumor. Surgery was performed under cardiopulmonary bypass. Use of the SSA and removal of a large part of the atrial septum followed by patch closure were required in order to resect the tumor because the tumor was large, multilobular, myxomatous, and attached to the thick stalk arising from the interatrial septum near the fossa ovalis.

A 12-lead ECG recorded after surgery revealed the presence of sinus arrest with backup ventricular pacing by epicardial temporary pacing leads (Fig. 1a and c), and sometimes showed an ectopic atrial rhythm or prolonged R–R intervals following the termination of paroxysmal atrial fibrillation. Therefore, a diagnosis of bradycardia–tachycardia syndrome was made. Because the ectopic atrial rhythm and sinus arrest were sustained, and the patient did not recover a normal sinus rhythm within 2 weeks of the operation, a permanent pacemaker (REPLY™ DR; Sorin Group, Milan, Italy) was implanted into the left precordial region.
The atrial bipolar lead (model 5076-45 cm; Medtronic Inc., MN, USA) was screwed into the free wall of the right atrium. The atrial pacing threshold was 0.5 V/0.5 ms, lead impedance was 604 Ω at 5 V, and P wave amplitude was 0.6 mV at the implanted site. Concerning the position of the atrial lead, no other location in the right atrium satisfied the standard sensing and pacing threshold criteria. Pacing thresholds and P-wave amplitudes were more than 5 V/0.5 ms and 0.1 mV at the high septal wall; more than 5 V/0.5 ms, 0.3 mV at the posterior septum; more than 5 V/0.5 ms, 0.2 mV around the right atrial appendage; and more than 5 V/0.5 ms, 0.4 mV at the cavo-tricuspid isthmus. The ventricle bipolar lead (model Screwvine-52 cm; Japan Lifeline Co., Ltd., Tokyo, Japan) was screwed into the septum of the right ventricle. A 12-lead ECG recorded during pacemaker implantation showed an ectopic atrial rhythm with a very broad P-wave morphology (Fig. 1d). Moreover, the timing of the intrinsic waves at the atrial and ventricular leads was almost simultaneous (Fig. 2a). Although the atrial lead on the free wall was located apart from the AV valve, we could not rule out the possibility that the intrinsic wave of the atrial lead was a far-field potential of the intrinsic R wave. To address this point, intravenous adenosine triphosphate (ATP) (20 mg) was administered to induce an AV block (Fig. 2b). As expected, the pacing from the atrial lead captured only the atrium and not the ventricle (Fig. 2b). In addition, electromapping of the right atrium and the coronary sinus (CS), using the atrial pacemaker lead, revealed that the CS ostium was the earliest site of activation of the ectopic P rhythm in the right atrium and that the activation sequence in the CS was distal to the proximal site. These

Fig. 1. Twelve-lead electrocardiogram recordings. Electrocardiograms after myxoma operation. Prolonged periods of asystole following ectopic atrial rhythm (a), twelve-lead electrocardiograms recorded before myxoma surgery (b), immediately after myxoma surgery (c), during pacemaker implantation (d), and after pacemaker implantation (safe RR mode, rate 70–130 ppm) (e). A normal sinus rhythm was recorded on admission (b). However, sinus arrest with backup pacing with a temporary epicardial pacemaker (a), junctional rhythm (d), or ectopic atrial rhythms (wide P waves) (d) were recorded after surgery for myxoma through the superior septal approach. When the pacemaker rhythm was set to the AAI mode (safe RR), the atrial pacing/ventricular sensing (ApVs) was 98% (e). ope: operation, PMI: pacemaker implantation.
results suggest that the ectopic P rhythm originated from the left atrium (most likely from the lateral wall), and the conduction time of the ectopic P wave from the origin to the tip of the atrial lead on the free wall of the right atrium was very long (Fig. 3a). In addition, the conduction time of the right free wall to the left atrium was also very long (Fig. 3a). These observations suggested a severe conduction delay between the left and right atria.

Next, we evaluated the interatrial and interatrioventricular synchrony because a long contraction time between the right and left atria was observed during the SSA procedure, dissection of the atrial septum, and patch closure. We were primarily concerned with determining whether the timing of the left atrium and left ventricle contractions was appropriate. The pacemaker was programmed in the DDDR mode at a rate of 70 to 120 ppm, and with an AV delay of 155 ms (nominal AV delay interval). Bradycardia due to the ectopic atrial rhythm was the basic cardiac rhythm observed when the pacing was stopped transiently, and was sustained for at least 1 week. Because of the conduction and contraction delay between the left and right atria, this almost nominal setting or shorter AV delay settings resulted in atrial pacing/ventricular pacing (ApVp) and simultaneous contractions of the left atrium and the left ventricle (Fig. 3b). To address this problem, the pacing mode was set to the AAI mode. Under this setting, the mitral valve inflow pulse Doppler measured by the echocardiogram showed no truncated A wave, which was seen in the setting with an AV delay of 50–155 ms. These results indicated that the sequential contractions of the left atrium and the left ventricle were favorable under prolonged AV delay (Fig. 3b). When either the long AV delay setting or the safe RR mode was set to ON, the atrial pacing rate was 98% and the ventricular pacing rate was 0%. These settings also showed favorable left atrium-left ventricle sequential contraction (Fig. 3b). No problems were observed in the safe RR mode during the 1-year follow-up period. A schematic summary of cardiac conductions under ectopic atrial rhythm or atrial pacing of this patient was shown in Fig. 4.

3. Discussion

Arrhythmias such as atrial fibrillation and incisional flutter after cardiac surgery for congenital heart defect or benign tumor
are very common [6]. Sinus nodal dysfunction, which requires permanent pacemaker implantation, also commonly occurred with the original Maze procedure [7]. However, the recently modified Maze procedure significantly reduced the need for pacemaker implantation (6%) [8,9]. This improvement was the result of the decreased number of resected arteries supplying the

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**Fig. 3.** Effects of intra-atrial conduction delay on the sequential contraction between the left atrium and the left ventricle. Wide P wave under ectopic atrial rhythm originating from the lateral wall of the left atrium and the right atrial lateral pacing. (a) Blood flow across the mitral valve assessed by pulse Doppler echocardiogram under various atrioventricular (AV) delay settings and (b) ApVp (AV delay, 50 ms, 125 ms, and 155 ms) showed a truncated A wave by simultaneous left atrium–left ventricle contraction, but not in ApVs.

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**Fig. 4.** Schematic model of this case. A schematic model of this case under the ectopic atrial rhythm originating from the left atrium (a). An ectopic P rhythm originating from the left atrium (P) slowly conducted to the lateral wall of the right atrium (P). However, the rhythm from the left atrium to the ventricles via the atrioventricular node (R) was normal. As a result, the timing of right atrial (P) and ventricular sensing (R) was almost simultaneous (a). A schematic model of this case under atrial pacing/ventricular pacing (ApVp) during the DDD mode (b). Stimulation of the lateral wall of the right atrium (A) slowly conducted to the left atrium. The rhythm from the lateral wall of the right atrium to the ventricle (R) was almost normal. As a result, the timing of left atrium and left ventricle contractions (R) was very close. Appropriate atrioventricular delay settings such as long AV delay or the safe R mode should be selected. RV: right ventricle, RA, right atrium, LA: left atrium.
A case of sinus node dysfunction requiring pacemaker implantation, after myxoma resection through the SSA. Performing the SSA with wide resection of the atrial septum could cause severe sinus node dysfunction. Moreover, our patient experienced conduction delays between the atria, and contraction dyssynchrony between the atria and the ventricles. Pacemaker settings for such patients should be carefully selected to prevent left atrium–left ventricle contraction dyssynchrony owing to interatrial conduction delays.

Conflict of interest
No conflict of interest declared.

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References