Improvement in left ventricular ejection fraction after radiofrequency catheter ablation of premature atrial contractions in a 23-year-old man

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Introduction

Arrhythmia-induced cardiomyopathy has been reported with a range of sustained supraventricular tachycardia including atrial fibrillation, atrial tachycardia, inappropriate sinus tachycardia,[1] dual atrioventricular nodal nonreentrant tachycardia,[2] and double fire tachycardia, as well as frequent premature ventricular contractions (PVCs).[3] Although the hallmark of both tachycardia-mediated and PVC-induced cardiomyopathy is resolution upon treating the inciting arrhythmia,[4] underlying mechanisms likely differ based on differing time course of onset and associated structural changes.[5,6] We present a patient with frequent symptomatic premature atrial contractions (PACs) who had normalization of left ventricular (LV) systolic dysfunction upon successful PAC catheter ablation. To date, we are aware of only one prior report of PAC-mediated cardiomyopathy.[7]

Case report

A 23-year-old man with no known significant past medical history presented with a 9-year history of palpitations associated with lightheadedness. He was a competitive basketball player and developed early-onset degenerative knee disease, requiring him to refrain from vigorous exercise for several months. During elective knee surgery, frequent extrasystoles were noted, prompting further cardiac workup. On 12-lead electrocardiography frequent PACs with varying degrees of aberrant conduction were noted (Figure 1), with an overall burden of 19.9%. An echocardiogram at this time revealed a dilated LV (end-diastolic diameter 5.9 cm) with globally reduced LV ejection fraction (LVEF) of 40% (Figure 2), with mild mitral and tricuspid regurgitation. Other chambers and valves were of normal structure and function. Cardiac magnetic resonance imaging demonstrated no evidence of myocardial edema or abnormal contrast enhancement. He endorsed mild exertional dyspnea without orthopnea and occasional chest fluttering associated with lightheadedness, but no chest discomfort or loss of consciousness. Clinical examination was normal except for elevated body mass index of 35.40 kg/m^2. He was commenced and uptitrated on beta-blocker therapy but reported no improvement in his symptoms after 4 weeks, with ongoing frequent ectopy on surface electrocardiography (Figure 1B). Given the severity of his symptoms, an electrophysiological study with possible PAC ablation was recommended.

KEY TEACHING POINTS

- Frequent premature atrial contractions (PACs) can result in a functional dilated cardiomyopathy.
- Catheter ablation of the PACs results in improvement in ventricular function.
- The risk for and factors predisposing to PAC-mediated cardiomyopathy are unknown.

At the start of the procedure, the patient was in sinus rhythm with frequent PACs, which were conducted with variable levels of aberrancy, most commonly in a right bundle branch block pattern but occasionally in a left bundle branch block pattern. The P-wave morphology was positive in lead I, biphasic with an initial positive vector in lead V_1, and negative in lead III (Figure 3A). The patient was placed under general anesthesia owing to intolerance of mild and conscious sedation. The femoral vessels were accessed under ultrasound guidance and multielectrode catheters were placed at the His bundle position, within the coronary sinus and right ventricular apex, using fluoroscopic guidance. Under general anesthesia, the PACs were no longer evident; thus, mapping was performed using isoproterenol infusion at 1 mcg/min to 5 mcg/min, coupled with burst atrial pacing and 1 g of

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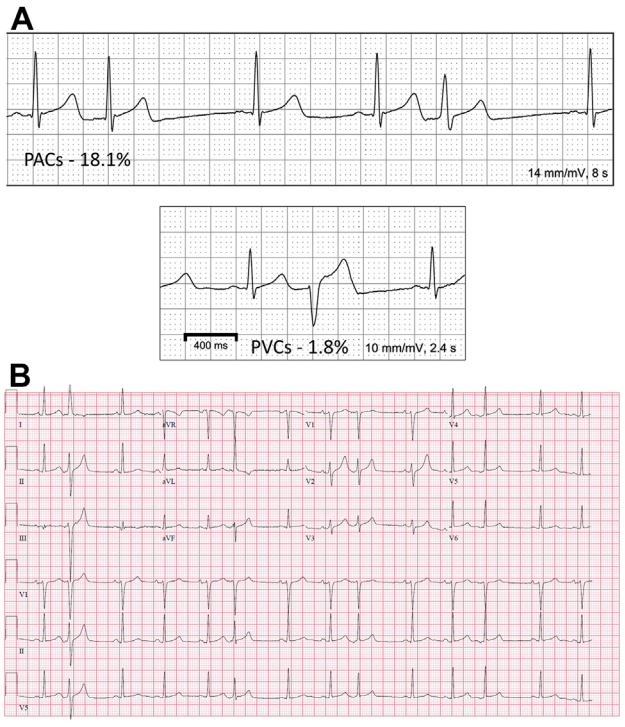

Figure 1 Echocardiographic data upon presentation to electrophysiology clinic. **A:** Sample of our patient's frequent premature atrial contraction (PAC) morphologies (*top*) and premature ventricular contraction (PVC) morphology (*bottom*) from ambulatory heart rate monitor. **B:** The 12-lead electrocardiogram on presentation to clinic on beta-blocker therapy.

intravenous calcium gluconate. With this, the clinical PACs were brought out frequently enough to allow for activation mapping. Electroanatomic mapping was performed with the CARTO system (Biosense Webster, Diamond Bar, CA) using a force-sensing irrigated ablation catheter (THERMOCOOL SMARTTOUCH SF). The earliest activation of the PACs was localized to the distal arcuate ridge at the anterior superior interatrial septum (Figure 3B, red arrow) and occurred 25–30 ms prior to atrial activation. Radiofrequency (RF) catheter ablation was delivered at this site. The PAC P-wave morphology slightly changed and a new focus of early activation was noted more inferiorly along the arcuate ridge, just superior to the anteromedial tricuspid annulus (Figure 3B, blue arrows). After RF ablation at this site, PACs were no longer inducible. Postablation electrophysiology study was normal. Upon waking the patient up from sedation, no PACs were seen and the patient remained in normal sinus rhythm. The patient's postprocedural course was uneventful. Metoprolol was discontinued upon discharge home after the procedure. At 3 months follow-up, the patient reported resolution of palpitations and exertional limitation, having returned to exercising about 1 week after his ablation. Repeat ambulatory cardiac monitoring at this time demonstrated only rare isolated PACs, with 69 recorded over a 4.5-day period (burden of less than 1%), and no PVCs. Transthoracic echocardiogram performed at the 3-month follow-up demonstrated improvement in LV

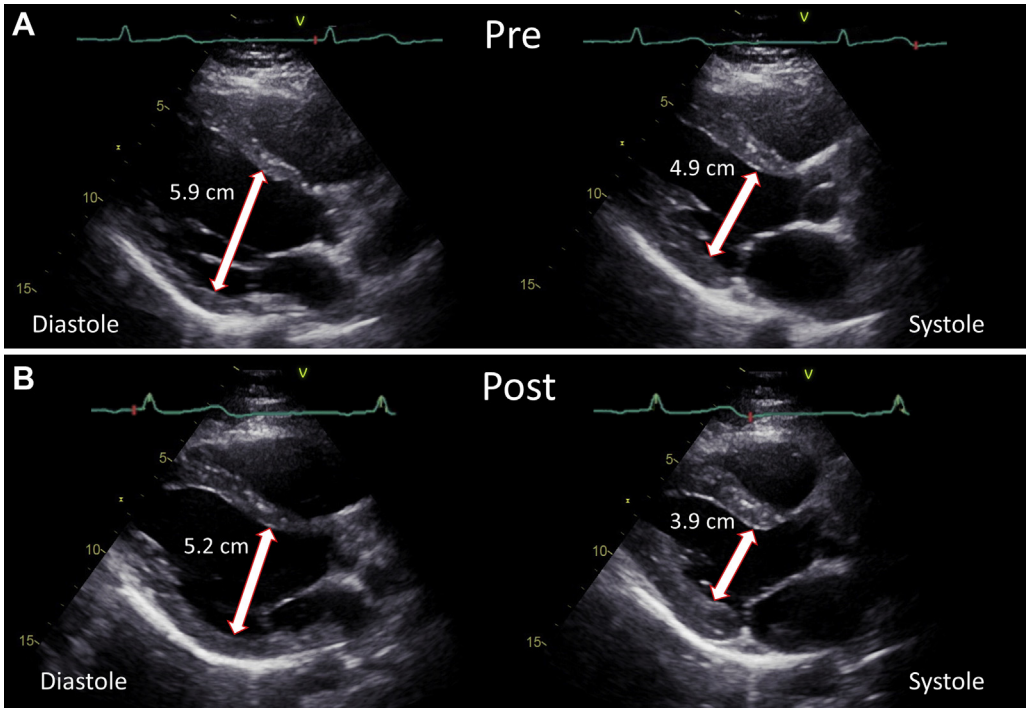

Figure 2 Transthoracic echocardiography before and after ablation. Samples of parasternal long-axis images from transthoracic echocardiography before and after radiofrequency ablation of premature atrial contractions. **A:** The preablation left ventricle (LV) was dilated, with end-diastolic diameter of 5.9 cm, and demonstrated depressed ejection fraction of 40%. **B:** After ablation, there was improvement in LV dimensions, with end-diastolic diameter decreasing to 5.2 cm, and LV ejection fraction improved to 50%.

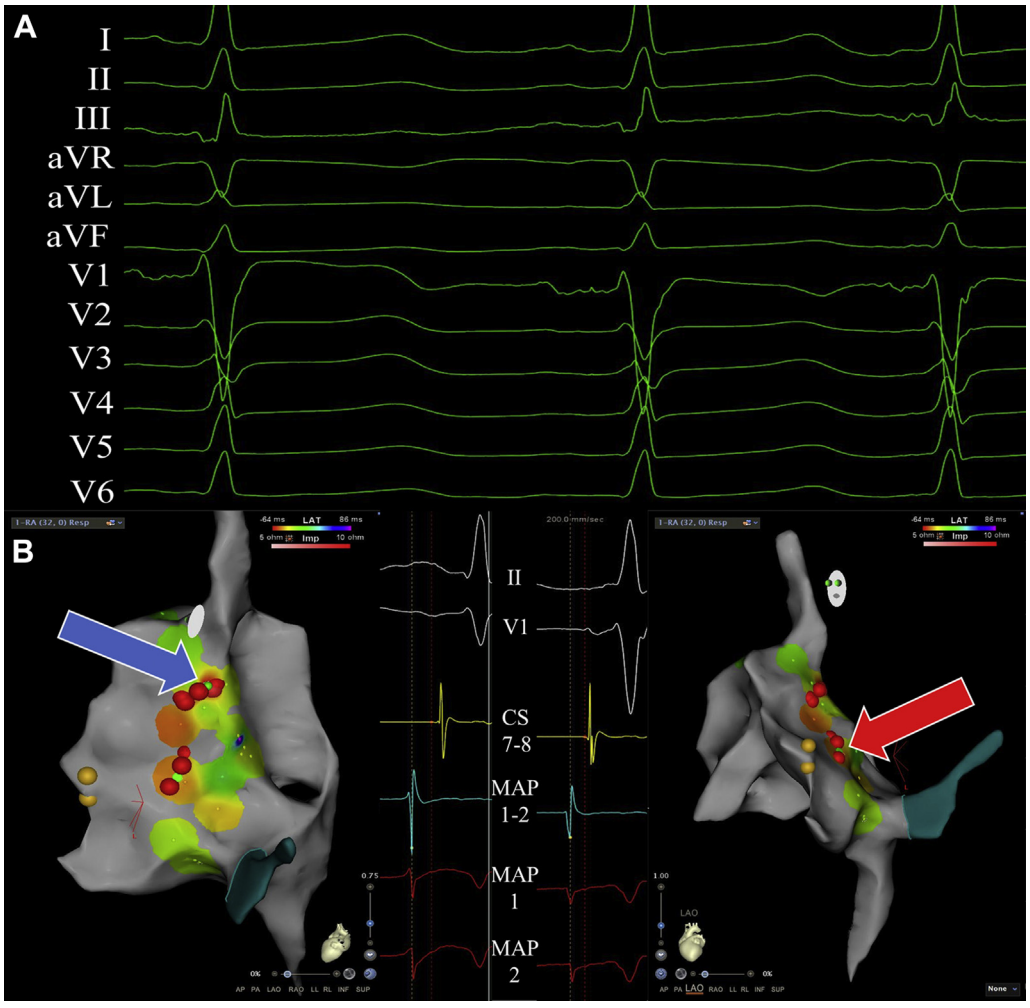

Figure 3 Intraprocedural surface electrocardiogram and electroanatomic mapping. **A:** Surface electrocardiogram prior to electroanatomic mapping confirmed P-wave morphology was positive in lead I, biphasic with an initial positive vector in lead V_1, and negative in lead III (premature atrial contraction generates the third QRS complex). **B:** Two sites were ablated (*blue arrow* and *red arrow*), viewed from a posterolateral viewpoint (*left*) and left anterior oblique view (*right*). *Yellow points* denote His electrogram positions. *Red points* and *green points* denote ablation sites. Unipolar and bipolar electrograms demonstrating points of earliest activation are shown adjacent to their respective ablation sites. The left electrogram corresponds to the ablation target denoted by the *blue arrow* and the right electrogram corresponds to the ablation target denoted by the *red arrow*.

dilation (end-diastolic diameter 5.2 cm) and systolic function (LVEF 50%, Figure 2) with resolution of mitral and tricuspid regurgitation.

Discussion

PACs generally are a benign phenomenon. However, in some circumstances, they may be pivotal in the development of sustained supraventricular arrhythmia. The efficacy of catheter ablation of atrial fibrillation–triggering PACs arising from the pulmonary veins is well recognized.[8] At a population level, PACs are associated with atrial fibrillation,[9,10] stroke, cardiovascular disease, and increased mortality.[11] There are few studies that discuss the burden of PACs on LV systolic dysfunction. Our case demonstrates clear evidence of PAC-mediated cardiomyopathy, causing LV dilation, and systolic dysfunction, associated with a high burden of PACs, which improved upon successful PAC ablation. These PACs were primarily aberrantly conducted, which likely contributed to this presentation. The findings on cardiac magnetic resonance imaging are consistent with other reports of PVC-induced cardiomyopathy, in which late gadolinium enhancement is rarely seen.[12] To the best of our knowledge, only 1 prior case of PAC-mediated cardiomyopathy has been published.[7] The frequency with which frequent PACs result in cardiomyopathy is unknown, nor are the associated risk factors for its development in the face of frequent PACs. We speculate that this patient's predisposition to cardiomyopathy was through frequent aberrant conduction, with a resulting mechanism similar to PVC-mediated cardiomyopathy.

Conclusion

We present a case of young man with PAC-induced tachycardiomyopathy that improved after successful PAC RF catheter ablation. The risk for and factors predisposing to PAC-mediated cardiomyopathy are unknown.

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