CASE REPORT

Typical atrial flutter mimicking a pacemaker-mediated tachycardia

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Abstract
A 64-year-old man with a history of congestive heart failure secondary to nonischemic cardiomyopathy, mitral and aortic valve replacements, and biventricular cardioverter-defibrillator placement, developed a tachycardia. The tachycardia exhibited a biventricular paced rhythm with a short R-P interval and concentric atrial activation sequence within the coronary sinus, suggesting that the tachycardia might be a pacemaker-mediated tachycardia (PMT). However, the tachycardia was diagnosed as counterclockwise cavotricuspid isthmus (CTI)-dependent atrial flutter (AFL), and linear ablation of the CTI eliminated the tachycardia. This case illustrated that typical AFL can mimic a PMT when there is a severe conduction delay through the CTI.

KEYWORDS
catheter ablation, cavotricuspid isthmus, intra-atrial conduction delay, pacemaker-mediated tachycardia, typical atrial flutter

1 | INTRODUCTION

We describe a case with a typical atrial flutter (AFL) presenting as an unusual form, which might mimic a pacemaker-mediated tachycardia (PMT).

2 | CASE REPORT

A 64-year-old man with a history of congestive heart failure secondary to nonischemic cardiomyopathy, mechanical mitral and aortic valve replacements for rheumatic heart disease and recurrent endocarditis, and a biventricular cardioverter-defibrillator (CRT-D) placement, underwent electrophysiological testing of a supraventricular tachycardia. A preprocedural echocardiogram revealed a mildly reduced left ventricular ejection fraction (50%-55%), normal size of the left atrium, and severely dilated right atrium and ventricle. The supraventricular tachycardia with 1:1 atrioventricular conduction was found by chance during an interrogation of the CRT-D, but the supraventricular tachycardia was too slow and under the detection rate for the CRT-D to detect or record it. During the electrophysiological study, a decapolar catheter was positioned within the coronary sinus (CS) with the most proximal electrode pair at the CS ostium. At baseline, the patient was in a biventricular paced rhythm with a short R-P interval, proximal to distal atrial activation sequence within the CS, and cycle length of 600 ms (Figure 1). A catheter-induced premature atrial contraction (PAC) interrupted the ventricular pacing with the resumption of spontaneous AV conduction (Figure 1). During this transition, the 12-lead electrocardiogram was unable to characterize the P-wave morphologies. The cycle length of the atrial rhythm was the same before and after this transition. Following this, activation mapping was performed during the tachycardia, suggesting that the tachycardia might be a counterclockwise cavotricuspid isthmus (CTI)-dependent AFL (Figure 2). Entrainment pacing from the CTI demonstrated that the postspacing interval was equal to the tachycardia cycle length. Linear ablation at the CTI eliminated the tachycardia. In this case, the CRT-D had been programmed to the DDD mode with a tracking rate of 60-130 bpm, sensed atrioventricular delay of 140 ms, and postventricular atrial refractory period (PVARP) of 300 ms. The atrial lead had been placed on the lateral edge of the right atrial appendage.

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A PMT is an endless-loop reentrant tachycardia, which can only occur in the presence of retrograde ventriculoatrial conduction with dual-chamber or cardiac resynchronization devices programmed to an atrial-synchronous, ventricular pacing mode. If the patient’s pacemaker or defibrillator is programmed to a DDD/R or VDD/R pacing mode and senses a retrogradely induced atrial event outside of the PVARP, it will respond by pacing the ventricle at the end of the AV delay. The repeated cycle of sensing and tracking the retrograde conduction (atrial sense, ventricular pace) is known as PMT. During a PMT, the atria should be retrogradely activated with a concentric atrial activation sequence. In this case, at first glance, the tachycardia with biventricular pacing appeared to be a PMT because the concentric atrial activation within the CS was recorded after the ventricular pace that tracked the atrial activation in the same cycle. However, a PMT was unlikely to have operated in this case because the ventriculoatrial interval during the tachycardia with biventricular pacing was short, and the P waves occurred during the PVARP. Also, because current pacemakers and defibrillators have a PMT termination algorithm that can terminate a PMT quickly, a PMT should not have continued. In this case, the main mechanism of the tachycardia was a typical AFL. However, two different ventricular rhythms were present during the same AFL. During the biventricular paced rhythm, ventricular pacing would have retrogradely caused the AV node to be refractory, allowing the AFL with biventricular pacing to continue. A PAC restored the intrinsic AV conduction. Because the PAC occurred during the PVARP, the CRT-D did not deliver a ventricular pace because it was blind to the PAC. In the meantime, the AV node recovered from being
refractory, and the AFL returning after the PAC could conduct through the AV node.

During the tachycardia with biventricular pacing, the atrial activation was recorded at the CS ostium 148 ms after the ventricular pacing stimulus. The sensed AV delay was 150 ms, and the atrial activation should have been sensed by the atrial lead 150 ms before the ventricular pacing was delivered. Therefore, the difference in the activation time between the right atrial appendage where the atrial lead electrode was placed, and the CS ostium, should have been a sum of 148 and 150, which was equal to 298 ms. In this case, during the typical AFL, the activation time travelling from the anterior aspect of the tricuspid annulus near the atrial appendage to the CS ostium was longer than the sensed AV delay, allowing for an occurrence of the atrial activation within the CS after the ventricular pace. In addition, the slow AFL rate within the tracking rate allowed for 1 to 1 AV synchrony.

The typical AFL in this case was very slow. A slow conduction along the tricuspid annulus can be caused by damaged myocardium, anti-arrhythmic drugs, and a dilated tricuspid annular ring. This patient did not take any anti-arrhythmic drugs. Therefore, the damaged myocardium and dilated tricuspid annular ring that were attributed to nonischemic cardiomyopathy and rheumatic valvular heart disease were likely to be the causes of a very slow typical AFL.

4 | CONCLUSION

This case illustrated that a typical AFL could mimic a PMT when there is a severe conduction delay through the CTI.

CONFLICT OF INTEREST DISCLOSURES

Authors declare no conflict of interests for this article.

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