Adenosine-Sensitive Focal Reentrant Atrial Tachycardia Originating From the Mitral Annulus–Aorta Junction

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Adenosine-sensitive reentrant atrial tachycardia (AT) has been recognized to originate from the confined area of either the right or left atrioventricular nodal regions. We describe a case with adenosine-sensitive focal AT which was successfully ablated at the uncommon focus located at the mitral annulus-aorta junction. The mode of AT initiation during the atrial extrastimulus suggested as the mechanism tachycardia reentry; AT was terminated by a bolus of 2 mg of adenosine 5'-triphosphate. These electrophysiological features are possibly associated with a substrate involved in the mitral annulus-aorta junction with node-like properties that is responsive to adenosine.

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Introduction

The mitral annulus-aorta (MA-Ao) junction, where the left atrium is continuous through the subaortic curtain with the musculature of the anterior mitral leaflet, has recently been recognized as a unique source of atrial tachycardia (AT). AT is rare, and its characteristics are yet to be elucidated.

Case Report

A 63-year-old female was referred for catheter ablation to treat supraventricular tachycardia. She had a 13-year history of paroxysmal palpitation with dizziness. The heart was structurally normal. A 12-lead electrocardiogram (ECG) revealed sinus rhythm with right bundle branch block (Figure 1A). ECG during tachycardia demonstrated a long R-P' tachycardia with two-to-one or one-to-one atrioventricular (AV) conduction (Figure 1B). The P-wave morphology during the tachycardia was isoelectric in leads I, aVR, V5, and V6, positive in leads II, III, and aVF, negative in lead aVL, and biphasic with negative followed by positive in leads V1-V4.

An electrophysiological study was performed. The patient was on no antiarrhythmic drugs for 2 weeks prior to the procedure. Electrode catheters were positioned in the high right atrial (HRA) septum, His bundle region (HIS), coronary sinus (CS), and right ventricular apex (RVA). The most proximal electrode of the CS catheter was positioned at the CS ostium. At baseline, the patient exhibited normal AH (110 ms) and HV (43 ms) intervals with a sinus cycle length of 610 ms (Figure 2A). Retrograde AV nodal conduction was present with the earliest activation recorded at the His bundle region during the RVA
pacing (Figure 2B). The tachycardia (cycle length 345 ms) was reproducibly induced by an atrial extrastimulus (S1S1 550 ms, S1S2: 330–360 ms) from HRA and was terminated by a single atrial stimulus. There was a significant negative correlation between the coupling interval of the extrastimulus (S1S2) and the interval between the extrastimulus and the first beat of the tachycardia (S2Ae) (S2Ae = −0.45 × S1S2 + 558, R² = 0.99, P = 0.007) (Figure 3). An intravenous administration of 2 mg of adenosine 5′-triphosphate prolonged the tachycardia cycle length and terminated the tachycardia reproducibly without causing AV block.

Ventricular overdrive pacing during the tachycardia demonstrated AV dissociation without tachycardia entrainment; an A-A-V response was observed after discontinuation of the pacing (Figure 2C). The atrial activation sequence during the tachycardia was different from sinus rhythm and retrograde VA conduction; the atrial potential was earliest at the His bundle region where the potential was 4 ms behind the onset of the P wave, followed by the potential at the proximal HRA septum. Accordingly, the tachycardia was diagnosed as an adenosine-sensitive reentrant AT.

The CS catheter was advanced distally because it was possible that earlier activity would be present on the left atrial side. The earliest atrial activation was then recorded at the most distal pair of electrodes of CS, and the potential preceded P wave onset by 10 ms, which was suggestive of a left atrial focus. Subsequently, transseptal puncture was done, and the endocardial activation mapping of the left atrium was performed by a 4-mm-tip ablation catheter. Finally, the earliest atrial activation was obtained at the 11 o’clock position of the mitral annulus in the left anterior oblique projection, the bipolar electrogram preceded the onset of the P wave by 40 ms, and a uni-polar electrogram exhibited a QS pattern (Figure 4A). The activation from this site radiated in all directions as shown by the electrograms at CS, the HRA septum and HIS, and the range of activation times including the other part of the atrium was less than the tachycardia cycle length. These findings were consistent with focal AT. At this location the motion of the catheter was synchronized to the motion of the CS catheter, indicating that the tip of the catheter was located at the mitral annulus.21 Left ventriculogram confirmed that the catheter was located at the MA-Ao junction (Figure 4B). A single radiofrequency current application at this site terminated the AT within 2 seconds without transient acceleration. The AT became no longer inducible. The patient has been free from the recurrent tachycardia without antiarrhythmic drugs for the subsequent 12 months.

Discussion

The present focal AT has two uncommon aspects. First, the site of origin was located at the MA-Ao junction. Second, the mechanism of the AT was possibly focal reentry, and the AT was adenosine-sensitive.
At the MA-Ao junction, a subaortic curtain supports three structural components: the left coronary cusp, noncoronary cusp, and the anterior leaflet of the mitral valve. The arrhythmogenicity of this site has been explained as follows. The leaflet musculature at this site has been shown to have action potential with AV nodal-type characteristics. These properties may be explained by calcium-dependent cells, which are responsive to adenosine and similar to AV nodal cells. Furthermore, embryological study suggested that remnants of the developing conduction system may be the substrate of the tachycardia. These findings suggest that the MA-Ao junction may cause both reentry and triggered activity. Clinically, the mechanism of the AT originating at the MA-Ao

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\[ R^2 = 0.99 \]
\[ P = 0.007 \]

Figure 2
Intracardiac electrograms during sinus rhythm (A), at ventricular pacing with retrograde AV nodal conduction (B), and at the cessation of ventricular overdrive pacing during the tachycardia (C). The atrial activation sequence during the tachycardia was different from that at sinus rhythm and the retrograde VA conduction. The atrial potential was earliest at the His bundle region (arrows), where the potential was 4 ms behind the onset of the P wave, followed by the potential at the proximal HRA septum. Ventricular overdrive pacing during the tachycardia demonstrated AV dissociation without tachycardia entrainment; the tachycardia showed an A-A-V response upon cessation of ventricular pacing.


Figure 3
Linear regression analysis of the relation between the coupling interval of the extrastimulus (S1S2) and the interval between the extrastimulus and the first beat of the tachycardia (S2Ae). A significant inverse correlation was observed (R^2: 0.99, P: 0.007).
junction may not be uniform. Gonzalez et al. combined the data of 10 cases to determine the triggered activity based on the mode of initiation. However, Kistler, et al. concluded that the mechanism is unclear. The present focal AT is unique in the way that reentry was suggested based on the mode of initiation.

From the above arrhythmogenic properties of the MA-Ao junction, certain characteristics of the focal reentrant AT emerge. In 1997, Iesaka et al. proposed a clinical entity of adenosine-sensitive reentrant AT due to focal reentry involving the AV node and/or AV nodal transitional tissues without any involvement of the AV nodal pathway originating in the vicinity of the apex of Koch’s triangle. The AT can be ablated at the earliest atrial activation site in the right superoseptal area. Recently, a left variant form of the adenosine-sensitive reentrant ATs was reported; the circuit of the AT may involve the leftward AV nodal transitional tissue, and the ATs are amenable for ablation from the noncoronary aortic sinus or the left coronary aortic sinus. The AT in the present case shares some characteristics of this entity: the AT was sensitive to a small dose of adenosine (2 mg), and the mechanism was focal reentry. Apparently, the site of origin of the present AT differs from those of the others, which were located within the confined area of either the right or left para-Hisian regions. Thus, the present AT may have arisen from a substrate with similar properties to the “para-Hisian type” of adenosine-sensitive focal reentrant ATs.

Limitation needs to be addressed regarding the mechanism of the AT. An inverse relationship between the coupling interval of the atrial extra-stimulus and the first beat of the tachycardia is suggestive, but not a specific criterion for reentry. Entrainment of the tachycardia would have shown convincing evidence. In conclusion, the adenosine-sensitive focal reentrant AT may originate from the MA-Ao junction. The AT may share a substrate with node-like properties that is responsive to adenosine with the previously recognized “para-Hisian type” of adenosine-sensitive focal reentrant AT.
References


