Successful Catheter Ablation of Focal Left Atrial Tachycardia Originating From the Mitral Annulus Aorta Junction

Kiyoshi Otomo,1 MD, Koji Azegami,1 MD, Takeshi Sasaki,1 MD, Mihoko Kawabata,1 MD, Kenzo Hirao,1 MD, and Mitsuaki Isobe,1 MD

SUMMARY

Focal left atrial tachycardias (AT) originating from the mitral annulus-aorta (MA-Ao) junction are rare and their mechanisms are unclear. We report a 35-year-old male with successful ablation of an exercise-induced focal AT due to triggered activity originating from the MA-Ao junction. The AT occurred spontaneously during treadmill exercise testing and was easily induced by an atrial extrastimulus and atrial burst pacing after intravenous administration of isoproterenol. The AT was terminated by an atrial extrastimulus as well as a bolus of 5 mg of adenosine 5′-triphosphate. The coupling intervals of the extrastimuli that induced the AT were positively correlated with the interval between the extrastimuli and the first beat of the AT, suggesting the triggered activity as a tachycardia mechanism. The AT was successfully eliminated by a focal ablation at the MA-Ao junction with the earliest atrial activation where fractionated atrial potentials were recorded. The MA-Ao junction should be recognized as an important arrhythmogenic region. (Int Heart J 2006; 47: 461-468)

Key words: Triggered activity, Adenosine 5′-triphosphate, Sub-aortic curtain, Delayed afterdepolarization, Isoproterenol, Paroxysmal atrial tachycardia, Remnant, Developing conduction system

FOCAL left atrial tachycardias (ATs) originate preferentially from the pulmonary veins and rarely from the left atrial appendage or mitral annulus (MA).1-4) Focal left ATs originating from the MA-Aorta (MA-Ao) junction have been rarely reported and their mechanisms are unclear.1-4) We report a male with an exercise-induced focal left AT originating from the MA-Ao junction that was presumed to be due to the triggered activity and that was successfully eliminated by focal radiofrequency catheter ablation.
CASE REPORT

A 35-year-old male without any significant past illnesses presented at our institution complaining of a 3-year history of exercise-induced palpitations. Chest x-rays, an echocardiogram, and laboratory data were normal. An electrocardiogram (ECG) at rest showed normal sinus rhythm (heart rate 80/min, PQ: 0.17 sec, QRS: 0.95 sec, QT: 0.32 sec) with frequent premature atrial contractions (PACs). Ventricular preexcitation was absent. During treadmill exercise testing, long R-P' and narrow QRS tachycardia occurred spontaneously with a maximal heart rate of 250/min (Figure 1). A Holter ECG showed frequent PACs occurring exclusively during the daytime (13,035 beats/day). The patient was sent to the electrophysiology laboratory since he requested ablative therapy.

The electrophysiological study was performed under local anesthesia in a nonsedated state. Four electrode catheters were positioned at the right atrial appendage, His bundle region, coronary sinus (CS), and apex of the right ventricle. During the baseline state, a stable sinus rhythm was present (AA: 750 msec,

![Figure 1](image_url). The body-surface ECGs of the AT occurring spontaneously during treadmill exercise testing (Bruce protocol). The sustained AT occurred at stage II and the AT rate rose from 147 to 250/min. See the text for details.
AH: 78 msec, HV: 45 msec). During ventricular pacing, decremental ventriculo-
atrial conduction was observed with the earliest atrial activation at the His bundle
region and it was demonstrated to be retrograde atrioventricular (AV) nodal con-
duction by paraHisian pacing\(^5\) (not shown). The tachycardia was not induced by
programmed atrial or ventricular stimulations in the control state. After intrave-
rous administration of 1.0 \(\mu\)g/min isoproterenol, the tachycardia was reproduc-
ibly induced by premature atrial stimulation (S1S1:350 msec, S1S2:320-340

---

**Figure 2.** A: Induction of the narrow QRS and long R-P’ tachycardia (PR/RP = 100/290 msec,
AA/AH/HA = 390/84/306 msec) by a premature atrial stimulation after intravenous administration
of isoproterenol. B: Termination of the tachycardia by a single atrial extrastimulus (S2) delivered
from the CS. All figures are shown in msec. See the text for details. CS indicates coronary sinus;
HBE, His bundle electrogram; LA sept., left interatrial septum; Lat. MA, lateral mitral annulus;
and RA sept., right interatrial septum.
msec, Figure 2A) and burst atrial pacing (300-350 msec) without a “jump-up” in AH interval, and was terminated by a single atrial extrastimulus (Figure 2B). An intravenous bolus of 5 mg of adenosine 5'-triphosphate (ATP) during sustained tachycardia lengthened the tachycardia cycle length and subsequently terminated the tachycardia (not shown). Each episode of the tachycardia was often nonsustained and the cycle length was variable (310-400 msec). The earliest atrial activation during the tachycardia was found simultaneously at the His bundle region and the distal CS and preceded the onset of the P wave on the surface ECG by 43 msec (Figure 2A). The atrial activation sequence during the tachycardia was different from that during the sinus rhythm and the retrograde AV nodal conduction (not shown). Based on the above-mentioned findings, the tachycardia was diagnosed as an AT. The P waves of the AT in the surface ECG were generally lower in amplitude than those during sinus rhythm, and their polarities were negative in I, aVR and aVL, positive in II, III and aVF, biphasic with negative followed by positive deflection in V1-V5, and isoelectric in V6 (Figure 3A). At the induction of the AT with premature atrial stimulation, the coupling intervals of the extrastimuli and the intervals between the extrastimuli and the first AT beats were pos-

**Figure 3.** A: Body-surface ECGs during sinus rhythm and the AT. The vertical dotted line indicates the onset of the P waves during the AT. **B:** Fluoroscopic views of the successful ablation site. **C:** Body-surface and intracardiac ECGs during the AT with the ablation catheter located at the MA-Ao junction where the earliest atrial activation was recorded. The longer dotted vertical line indicates the earliest atrial activation and the shorter one indicates the onset of the P waves in the body-surface ECG. See the text for details. RAO indicates right anterior oblique view; LAO, left anterior oblique view; RA, right atrium; HB, His bundle; and Abl, ablation catheter. Other abbreviations as in Figure 2.
The whole left atrium was explored by an ablation catheter inserted via a patent foramen ovale because the true earliest activation site was suspected to be within the LA. The earliest atrial activation during the AT was found at the 11 o'clock direction of the MA in the left anterior oblique projection (Figure 3B), where wide (48 msec) and fractionated atrial potentials with low amplitude (0.48 mV) and an A/V ratio of approximately 1.0 could be recorded. The local atrial potential preceded the onset of the P wave in V1 by 62 msec (Figure 3C) with local unipolar recording of the QS pattern (Figure 3C). A single radiofrequency energy application at this site terminated the AT without any recurrence of the AT or the PAC. Aortography confirmed the successful ablation site was located at the MA-Ao junction (not shown). The Holter ECG performed after the ablation showed a marked reduction in the daily number of PACs (pre- versus postablation: 13,035 versus 3 beats/day) and a treadmill exercise test induced neither AT nor PAC. During a postablation follow-up period of 7 months, no recurrence has been observed.

**DISCUSSION**

**Tachycardia mechanism:** Reports regarding focal left ATs arising from the MA-Ao junction are sparse and their mechanisms are unclear. In previous reports,
Figure 5. **A**: Macroscopic (A) and microscopic specimens of the MA-Ao junctional area in a human heart (B) and the intracellular action potentials recorded at the MA-Ao junctional area. A: A specimen of a fibrous skeleton of a human heart, viewed from the apex (upper) and anterior (lower). The MA-Ao junction is indicated by the arrowheads (upper) and the white dots (lower). B: The left atrial side of the MA-Ao junction is covered by myocardial cells contiguous with the left atrial myocardium. The dotted ellipse indicates the MA-Ao junctional area. C: The gradual morphological changes in the intracellular action potentials from the left atrial myocardial cells to the myocardial cells at the anterior mitral leaflet. Note the shallower resting membrane potential and the lower $dV/dt$ of phase 0 from the myocardial cells at the mitral annulus-aortic junction and the anterior mitral leaflet, resembling the characteristics of AV nodal cells. AA indicates aortic annulus; Ao, aorta; AV, aortic valve; AML, anterior mitral leaflet; CFB, central fibrous body; LA, left atrium; LCC, left coronary cusp; LFT, left fibrous trigon; LV, left ventricle; MA, mitral annulus; NCC, noncoronary cusp; RCC, right coronary cusp; RFT, right fibrous trigon; SL, septal leaflet of the tricuspid valve; TA, tricuspid annulus; and TV, tricuspid valve. (A and B: Modified from Anderson RH, Becker AE. Cardiac Anatomy. An Integrated Text and Colour Atlas. London: Gower Medical Publishing, 1980. C: Modified from reference 6.)
the underlying mechanisms of similar ATs were considered to be microreentry or triggered activity. In earlier experimental studies, the MA-Ao junction was found to be arrhythmogenic with respect to atrial arrhythmias due to catecholamine-induced delayed afterdepolarization. Chen, et al reported 9 patients with focal ATs due to triggered activity characterized by both induction and termination of the AT by programmed atrial stimulations, enhanced AT induction by isoproterenol infusion, AT termination by adenosine, and the delayed afterdepolarizations demonstrated by monophasic action potential recordings. In our case, the mechanism of the focal left AT was presumed to be due to triggered activity, judging from the direct relationship between the coupling interval of the extrastimulus and the interval from the extrastimulus to the first tachycardia beat, enhanced inducibility by isoproterenol infusion and the ATP-sensitivity, although the microreentry involving the atrial tissue with AV node-like electrophysiological properties could not be completely ruled out from the possible mechanisms.

Arrhythmogenic substrate: In previous reports, the foci of the ATs arising from the MA had a propensity to be localized to the superior aspect, consistent with the MA-Ao junctional region. The MA-Ao junctional region, also known as the "sub-aortic curtain", is where the mitral and aortic annuli conjugate and is sandwiched between the right and left fibrous trigons (Figure 5A and B). It is reported that, on the left atrial side of the MA-Ao junction, there are myocardial cells contiguous with the left atrial myocardium which further extend into the anterior mitral leaflet. Wit, et al reported that the myocardial cells at the MA-Ao junction exhibited AV node-like electrophysiological properties and gave rise to catecholamine-induced triggered activities due to the delayed afterdepolarizations (Figure 6C). In a recent study using mouse embryos, Gonzalez, et al demonstrated the existence of the remnants of the developing specialized conduction system at the MA-Ao junction and they postulated that these remnant tissues can be a possible substrate of this kind of arrhythmia.

Limitations: The microreentry involving the tissues with AV node-like electrophysiological properties cannot be completely excluded from the tachycardia mechanisms, because the entrainment pacing was not performed due to the nonsustenance of the AT. Linear regression analysis of the relationship between the intervals from the extrastimuli to the first AT beats and the coupling interval of the extrastimuli that induced the ATs showed a positive correlation with a much steeper inclination in the regression line (Figure 4, y = 4.3x - 989.33) than that reported previously (y = 0.9852x + 16.63). In our case, the cycle lengths of induced ATs were variable (330, 350, and 370 ms at coupling intervals of 320, 330, and 340 ms, respectively). Hence, the exaggerated responses of the return cycles to the changes in the coupling intervals might be due to variable cycle lengths of the ATs induced at each coupling interval. Furthermore, the AT was
induced only at 3 coupling intervals with a narrow induction zone (20 ms), which might have enhanced the influence of the changes in the AT cycle length on the inclination of the regression line. Judging from the low amplitude of the local ventricular electrogram (0.52 mV), the successful ablation site might be slightly deviated to the left atrial side, instead of right on the MA. The successful ablation site in this study resembles that of previous reports regarding similar ATs\(^1,3\) with regard to the fluoroscopic position and the characteristics of the local electrograms, possibly suggesting that the site of AT origin might be located on the atrial side of the MA-Ao junction in this clinical entity. In this era of electroanatomical mapping, the case presented here should have been mapped properly in order to visualize the entire atrial activation sequence.

**Conclusions:** It is concluded that the MA-Ao junction may be one of the arrhythmogenic regions and should be recognized as a possible AT origin during catheter ablations of focal left ATs.

**REFERENCES**