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Peri-Mitral Atrial Tachycardia Using the Marshall Bundle Epicardial Connections

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ABSTRACT

OBJECTIVES The aim of this study was to determine whether re-entrant circuits were associated with the ligament of Marshall (LOM).

BACKGROUND Peri-mitral atrial tachycardias (PMATs) following pulmonary vein isolation (PVI) or mitral valve surgery are common.

METHODS Six PMATs involving epicardial circuits were identified from 38 patients. Of these, 4 PMATs involved the LOM (PMAT-LOM, mean cycle length 308 ± 53 ms), as confirmed by the insertion of a 2-F electrode in the vein of Marshall (VOM). All patients underwent PVI and mitral isthmus ablation. The PMAT-LOMs were diagnosed based on left atrium (LA) activation maps that covered <90% of tachycardia cycle length (TCL), and a difference between the post-pacing interval and TCL that was: 1) ≤20 ms at the VOM, the ridge between the left pulmonary vein and appendage, the anterior wall of the LA, and along the 6 to 11 o'clock direction of the mitral annulus; and 2) >20 ms at the distal coronary sinus (CS), the posterior wall of the LA, and the mitral isthmus ablation line (or noncapture). Catheter ablation was performed at the ridge for all PMAT-LOMs.

RESULTS Three tachycardias were successfully terminated at the ridge, which showed continuous fractionated potential lasting >100 ms, confirming the bidirectional block of Marshall bundle (MB)-LA connections. The remaining tachycardia required ablation for the CS-MB connections, confirming bidirectional block of CS-MB connections.

CONCLUSIONS PMAT-LOMs following PVI or valve surgery accounted for up to 11% of PMATs. The bidirectional block of either MB-LA or CS-MB connections is required to eliminate PMAT-LOMs. (J Am Coll Cardiol EP 2016;2:27–35)

The ligament of Marshall (LOM) is an epicardial vestigial fold that contains the vein of Marshall (VOM) and a myocardial sleeve called the Marshall bundle (MB). The LOM has previously been implicated as a source of focal activity initiating atrial fibrillation (AF) (1–3). Radiofrequency catheter ablation (RFCA) has emerged as an effective therapy for patients with AF (4–6). Peri-mitral atrial tachycardia (PMAT) commonly develops after pulmonary vein isolation (PVI) or mitral valve surgery (7). The creation of a linear lesion from the mitral annulus (MA) to the left inferior PV, in the so-called mitral isthmus (MI), is the most common ablation strategy for PMAT. Achieving a complete ablation line (defined by a bidirectional conduction block across the MI line) is critical to eliminate PMAT, but doing so can be technically difficult and can require coronary sinus (CS) ablation of up to 70% (8). In rare cases, re-entrant circuits of PMAT involve an epicardial connection (9). This study was conducted to clarify the characteristics of PMATs that use epicardial connections, especially the LOM (PMAT-LOMs).

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ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation

AT = atrial tachycardia

CFAE = complex fractionated atrial electrogram

CS = coronary sinus

LA = left atrial/atrium

LAA = left atrial appendage

LOM = ligament of Marshall

MA = mitral annulus

MB = Marshall bundle

MI = mitral isthmus

PMAT = peri-mitral atrial tachycardia

PPI = post-pacing interval

PVI = pulmonary vein isolation

RFCA = radiofrequency catheter ablation

TCL = tachycardia cycle length

VOM = vein of Marshall

METHODS

STUDY POPULATION. This study consisted of 38 consecutive patients with PMAT who underwent PVI for AF or mitral valve surgery in the Saitama Medical Center of Jichi Medical University and Tokyo Metropolitan Hiroo Hospital from March 2009 to September 2014. Of these patients, 28 underwent ablation for AF (paroxysmal AF n = 12; persistent AF lasting ≤1 year n = 2; long-standing persistent AF lasting >1 year n = 14) and 10 underwent mitral valve surgery (replacement n = 7; plasty n = 3). All patients gave signed informed consent before the procedure.

ELECTROPHYSIOLOGICAL STUDY AND ABLATION PROCEDURE. Antiarrhythmic drugs, excluding amiodarone, were discontinued ≥5 half-lives prior to ablation. The electrophysiological study was performed during continuous intravenous administration of propofol (3 to 6 mg/kg/min). A decapolar or multipolar catheter (Inquiry Luma-Cath, St. Jude Medical, Tokyo, Japan, or BeeAT, Japan Lifeline Co., Ltd., Tokyo, Japan) was inserted via the right subclavian vein to the CS. The left atrium (LA) and PV were explored via transseptal catheterization with 2 or 3 long sheaths. PV mapping was performed using a circular mapping catheter (Inquiry Optima, St. Jude Medical or Lasso, Biosense Webster, Inc., South Diamond Bar, California). A 3.5-mm irrigated-tip catheter (ThermoCool Navistar, Biosense Webster or Cool Path, St. Jude Medical) and a 3-dimensional anatomic mapping system (CARTO, Biosense Webster or EnSite NavX and Velocity, St. Jude Medical) were used for mapping and ablation.

AF ABLATION STRATEGY. Ablation for AF was performed as previously described (10). Briefly, in patients with paroxysmal AF and persistent AF lasting ≤1 year, circumferential PVI was performed. If AF was present, sinus rhythm was restored by internal or external cardioversion. The target in this phase was the elimination of all PV potentials; once the target was attained, continuous intravenous isoproterenol (4 μg/min) was administered, a 20- to 40-mg bolus of adenosine triphosphate was injected, and further RFCA was performed to eliminate any reconduction of PV potentials or adenosine triphosphate-provoked acute dormant PV conduction. The endpoint of PVI was the establishment of a bidirectional conduction block between the LA and PV. If reproducible non-PV foci–initiated AF was identified, RFCA was attempted to eliminate the non-PV foci.

In the patients with long-lasting persistent AF lasting >1 year, PVI was performed during AF, followed by mapping of complex fractionated atrial electrograms (CFAEs) of the LA. CFAEs were defined as previously reported (11). Subsequently, linear ablation of a roof and MI line was performed. If AF persisted after linear ablation, LA CFAE ablation was performed. If the AF still persisted in this phase, internal or external cardioversion was performed. If the AF converted to atrial tachycardia (AT) after either linear or CFAE ablation, or both, mapping and ablation were performed.

DIAGNOSIS AND ABLATION OF PMAT. Linear ablation of the MI was performed for induced or spontaneously occurring PMAT. PMAT was diagnosed using a 3-dimensional anatomic mapping system with the entrainment pacing technique. A difference between the post-pacing interval (PPI) and tachycardia cycle length (TCL) of ≤20 ms from the 4, 8, and 12 o'clock positions in the LA or CS along MA. Left-side PVI was performed in patients without AF. Subsequently, RFCA was applied from the 4 o'clock position of the MA to the bottom left-side PV with 25 to 35 W. If endocardial ablation was unable to successfully achieve a complete MI conduction block (defined by bidirectional conduction block across the MI line), further RFCA application was delivered within the CS opposite of the endocardial MI line with 20 to 25 W.

DIAGNOSIS OF PMAT USING LOM. PMATs using epicardial connections were diagnosed with a 3-dimensional anatomic mapping system with the entrainment pacing technique. PMATs using epicardial connections were diagnosed based on: 1) an LA activation map of a 3-dimensional anatomic mapping system that covered <90% of the TCL; 2) a difference between PPI and TCL from the LA 6 to 11 o'clock positions of the MA that was ≤20 ms; 3) a difference between PPI and TCL from the LA 4 o'clock position of the MA or prior MI line that was >20 ms or noncapture with high-output pacing (20 mA × 2 ms); and 4) a difference between PPI and TCL from the multiple sites of CS that was ≤20 ms. Added to these observations, PMAT-LOM was diagnosed based on: 1) a difference between PPI and TCL from the 2-F octopolar electrodes (EPstar Fix, Japan Lifeline) inserted into the VOM that was ≤20 ms; 2) a difference between PPI and TCL from the CS distal beyond the bifurcation of VOM that was >20 ms; and 3) a difference between PPI and TCL from the ridge (defined as the area between the left-side PVs and left atrial appendage [LAA]) that was ≤20 ms.