



The Aortomitral Continuity Challenge

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Abstract

Premature Ventricular Contractions (PVC) is a frequent finding in the general population, especially in patients with structural cardiac disease. Nonetheless, data regarding aortomitral continuity as substrate for PVC in normal hearts is still limited. We present 3 cases of young patients without structural heart disease, with frequent symptomatic PVC, without clinical improvement after anti-arrhythmic drugs treatment. All patients were submitted to an electrophysiological study that identified the aortomitral continuity as the earliest site of PVC activation. After a successful ablation, all the patients improved their clinical status. These cases and literature review of the aortomitral continuity substrate highlight the indications, efficacy, safety and limitations of catheter ablation.

Keywords: Ventricular premature contractions; Aortomitral continuity; Left ventricular outflow tract; Radiofrequency ablation

Introduction

In the absence of structural heart disease, the presence of Premature Ventricular Contractions (PVC) or Ventricular Tachycardia (VT) is frequently located in the Right Ventricular Outflow Tract (RVOT) or Left Ventricular Outflow Tract (LVOT). The complex relationships between the LVOT and the surrounding structures and its unique electrophysiological properties can limit the physician approach. Regarding the LVOT, PVC can arise from some sites surrounding the LVOT, such as the aortic root and aortic cusps, the mitral annulus, the superior basal septum, the epicardium and the Aortomitral Continuity (AMC) [1]. The AMC is defined as the continuation of the anteromedial aspect of the mitral annulus to the aortic valve; it consists of a curtain of fibrous tissue extending from the anterior mitral valve leaflet to the left and noncoronary aortic cusp. The AMC is connected with the left and right fibrous trigones to ventricular myocardium and the right fibrous trigone to the membranous ventricular septum [2] (Figure 1).

The knowledge of the electrophysiological properties of the LVOT arrhythmias are incomplete [3], and the ECG does not allow the precise localization of the PVC origin [4]. AMC and PVC are uncommon and have limited literature information, which may difficult mapping and ablation procedure. We describe three patients with symptomatic AMC PVC refractory to Anti-Arrhythmic Drugs (AAD), who were submitted to catheter ablation that result in a complete PVC suppression.

Case Series

Case 1

Thirty-three years-old female complaining of frequent palpitations. The ECG showed frequent PVC with inferior axis (Figure 2), with more than 50000 monomorphic PVC on 24 h Holter monitoring. Transthoracic echocardiography and myocardial perfusion scintigraphy excluded structural heart disease or ischemia. An Electrophysiological Study (EPS) with 3D electroanatomic endocardial mapping was guided by the Ensite NavX/Velocity mapping system (Abbot, St Paul, USA). Through a 7F arterial short sheath in the right femoral vein, an irrigated-tip therapy ablation catheter (SmartTouch Thermocool; Biosense) was inserted *via* transeptal approach for mapping and ablation. Clinical PVC was sustained during mapping and the 3-Dimensional geometry identified the AMC as the PVC substrate (Figure 3A). Radiofrequency (RF) ablation was then targeted at the AMC site, with a power range from 30W to 40W, with a maximum temperature limited to 40°C, and a flow of 12 ml/min. PVC elimination was achieved on the third RF attempt (Figure 3B). Three weeks after, the patient had recurrence of symptoms with frequent PVC. A new EPS was performed, with a 3D electroanatomic mapping guided by the CARTO³ system (Biosense Webster, USA) and the earliest activation site was located on the mitral annulus, in the AMC (Figure

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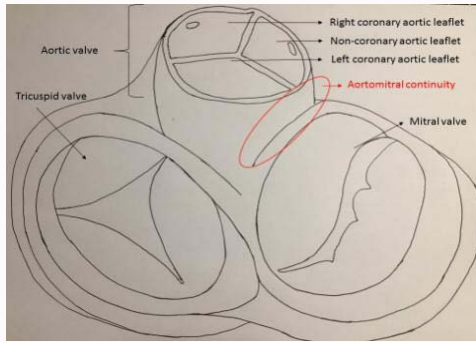


Figure 1: Schematic representation of the aortomitral continuity and the surrounding structural. The aortomitral continuity is localizing between the anteromedial aspects of the mitral annulus to the aortic valve.



Figure 3B: Electrocardiogram after a successful radiofrequency ablation, without premature ventricular contractions.



Figure 2: Initial electrocardiogram before the electrophysiological study with frequent Premature Ventricular Contractions with left bundle branch block QRS morphology and inferior ECG axis.



Figure 4: 3D mapping of the re-ablation of the portion of the mitral annulus in the aortomitral continuity.

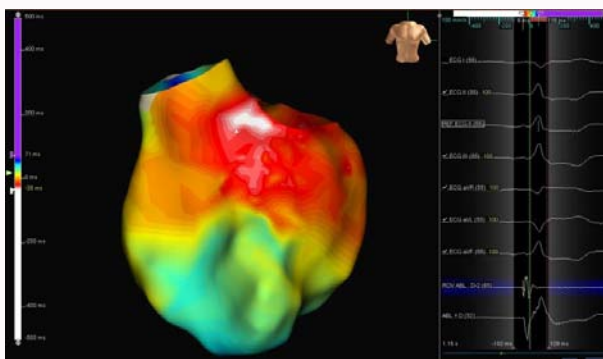


Figure 3A: 3D mapping of the left ventricular outflow tract and the first ablations application on the aortomitral continuity. Yellow mark represents the bundle of His.



Figure 5: Initial electrocardiogram of the patient 2 before the electrophysiological study with frequent premature ventricular contractions with an inferior axis, positive QRS in V1 and negative QRS in DI.

4). Power was delivered at 35W with a maximum temperature limited to 40°C, which resulted in immediate PVC suppression. The duration of RF was 300 sec, with a fluoroscopy time of 4 min. There were no recurrences in the following 6 months.

Case 2

A 53 years-old female presented in the cardiology outpatient clinic complaining of fatigue and frequent palpitations. Transthoracic echocardiography and cardiac magnetic resonance imaging confirmed absence of structural heart disease. The ECG showed frequent PVC, with an inferior axis, positive QRS in V1 and negative QRS in DI, with more than 15000 monomorphic PVC on

Holter monitoring (Figure 5). The EPS, using an irrigated-tip ablation catheter for 3D electroanatomic endocardial mapping was guided by the Ensite NavX/Velocity mapping system (Abbot, St Paul, USA), and identified the PVC earliest site on the anterolateral mitral annulus at AMC (Figure 6). Two RF attempts were performed with a power of 35W and a total RF time of 60 sec. After further mapping, a third RF

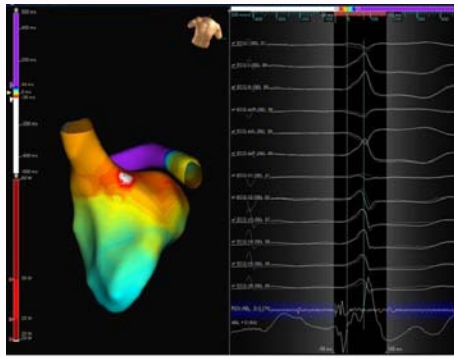


Figure 6: 3D mapping of the left ventricular outflow tract with an identification of the anterolateral mitral annulus at the aortomitral continuity as substrate.

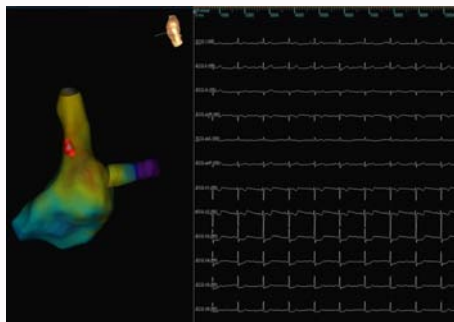


Figure 7: Electrocardiogram after a successful radiofrequency application, without premature ventricular contractions in the patients 2.

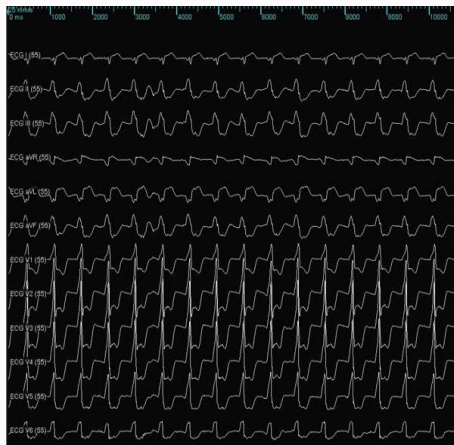


Figure 8: Initial electrocardiogram of the patient 3 before the electrophysiological study with a sustained ventricular tachycardia with an inferior axis, positive QRS in V1 and negative QRS in DI and a VL.

application was delivered on the substrate site, with a power delivered at 35W, a maximum temperature of 43°C, a flow 12 ml/min and 2.5 min of fluoroscopy time, obtaining total PVC suppression (Figure 7).

Case 3

A 56 years-old male referred to the cardiology outpatient clinic due frequent palpitations. Transthoracic echocardiography also without structural heart disease. Holter monitory and ECG showed frequent runs of sustained VT, with an inferior axis, negative QRS in a VL and DI, with a positive QRS in V1 (Figure 8). The EPS, using an irrigated-tip ablation catheter for 3D electroanatomic endocardial mapping was guided by the Ensite NavX/Velocity mapping system

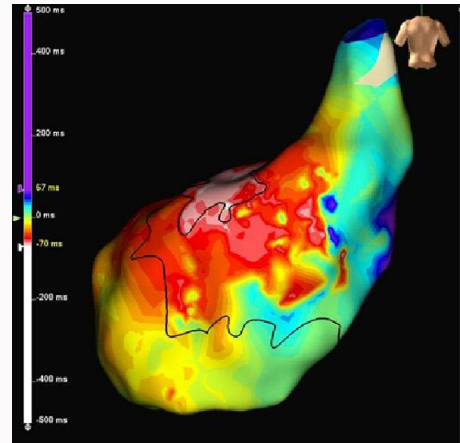


Figure 9: Patient 3 3D mapping of the left ventricular outflow tract with an identification of the anterolateral mitral annulus at the aortomitral continuity as substrate.

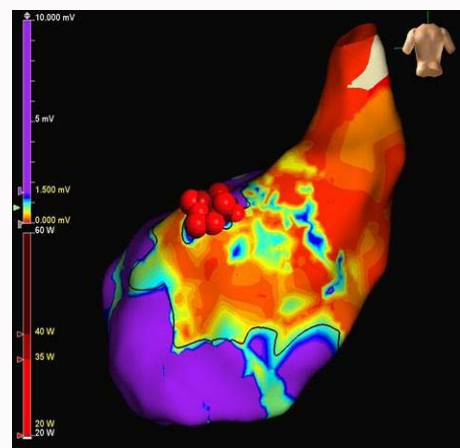


Figure 10: Electrocardiogram after a successful radiofrequency application, without premature ventricular contractions or sustained ventricular tachycardia in the patient 3.

(Abbot, St Paul, USA), showed the PVC earliest activation site on the anterolateral portion of the mitral annulus in the AMC (Figure 9). RF ablation was targeted at the substrate site, with a power of 30W, with a maximum temperature limited of 40°C, a flow of 12 ml/min. PVC were immediately eliminated during the first RF application (Figure 10).

Discussion

AMC is a complex fibrous tissue that can include remnant of the atrioventricular conduction system (dead-end tract). These cells have histological and electrophysiological properties, like the cardiac conduction cells, a characteristic that may favor idiopathic AMC PVC [5,6]. This fact explains the capacity of the AMC cells to generate an electrical potential and its propagation for the ventricle resulting in PVC. Also, this aspect may allow us to consider the AMC as site with high arrhythmogenic potential [1,7], since its cells can had singular electrophysiological characteristics compared with the surrounding LVOT structures [3,6]. Apparently, there are no myocardial cells composing the AMC [3]. Yet, the entire nature of the AMC arrhythmogenic substrate remains a mystery.

Usually, patients with frequent outflow tract PVC have structurally normal heart. However, PVC may be an early signal of

cardiomyopathies that should be clarified before any approach [8]. PVC originated from the LVOT occur more frequently in men, rise with age and maybe register as non-sustained or sustained runs of VT [9]. PVC cause several symptoms, as palpitations, fatigue or heart failure. Another form of presentation is asymptomatic Left Ventricular (LV) dysfunction (directly or indirectly associated to the PVC) [10].

All the variants of LVOT arrhythmias seem to have similar QRS morphology characteristics. Nonetheless, some authors advocate that further analysis allows the identification of the precise site, something that would facilitate the procedure [11]. According to Kumagai et al. [4] ECG characteristics of AMC PVC or VT are: R wave without an S wave in almost all the precordial, R/S wave transitional zone in V1 lead, absence of S wave in V6 lead and/or a greater intrinsicoid deflection time. Other authors, identify in the inferior leads the presence of tall R waves, positive QRS in lead I and negative QRS in the superior and lateral basal leads, as well as the qR pattern in V1 lead [12]. Chen et al. [5] stated that an early R/S wave transition in precordial leads with equal R and S wave amplitude in V2, and an R wave in V3 are typical of the anterior AMC location, a finding displayed in our patients' ECG (Figure 2 and 5). On the other hand, an early R/S wave transition in precordial leads, with equal R and S wave amplitude in V2, but high R wave in V1 and V3 suggest a middle AMC location. V1 lead with "qr" morphology on the ECG exhibited high specificity to AMC PVC in the electroanatomic mapping [5]. Our patients exhibited an inferior ECG axis, with an early R/S wave transition in V2. However, the first case had negative QRS in lateral leads, and the second a positive QRS in lateral leads, proving the difficulty of PVC localization by the ECG. The AMC is an ample zone and the substrate localization may be more anterior or posterior, which can promote an early or late transition zone in the precordial leads. This fact can be a reason for the lack of consensus among authors.

LV dysfunction can be induced by frequent PVC, nonetheless there are limited literature focused on this issue, and even less publications describing AMC PVC as a primary responsible for LV dysfunction. AAD can reduce the PVC burden, and, therefore, may be used as therapy, with one or more of the following classes of drugs: β -blockers or non-dihydropyridine calcium channel blockers (diltiazem or verapamil) and/or class III agents (amiodarone, sotalol, dofetilide) [12]. In some cases, these drugs cannot accomplish the PVC control or may even present serious side effects, being important the clinical and ECG monitoring after AAD initiation [10].

Catheter ablation is more efficient than AAD to treat PVC, particularly in monomorphic PVC, and can provide a definite PVC suppression with low complication rates. Another interesting find is in patients with LV dysfunction, that show a high rate of recovery after a successful PVC ablation [9,13]. Nowadays, PVC catheter ablation is a class I recommendation in symptomatic patients PVC/VT refractory to AAD [2,14].

AMC anatomical tissue and the difficulty to access this area can influence the procedure, since may not allow a stable contact between the catheter and the potential substrate. An eventual cause of procedure failure is the epicardial location of the substrate. Nonetheless, the major difficulty during the mapping and ablation maneuvers is the absence of PVC during the EPS that do not allow the substrate site identification.

AAD therapy, autonomic and hormonal response, environmental

factors and sedation can influence the PVC burden during the procedure [8,15]. In the cases in whom PVC are not present during the procedure, pace-mapping is an alternative approach to reproduce the QRS PVC morphology allowing RF ablation [8,9].

A transeptal approach is usually performed, allowing having a better catheter contact and stabilization [8]. The electrograms can be divided into two components: First, the frequent low amplitude potential that precedes the QRS (pre-systolic), and second, a large potential that occurs with the QRS onset. Despite a lower rate of complications described, coronary artery rupture with acute myocardial ischemia is a potential risk, justifying anatomical 3D geometry or angiography before RF application.

Regarding PVC catheter ablation, several settings and energy delivery protocols can be applied according to the type of catheter used the center experience, and operator. General recommendations for LVOT PVC advocate a maximum energy delivery of 30W, with a maximum temperature of 40°C, and an irrigation flow range from 12 ml/min to 20 ml/min. If PVC suppression occurs in the first 10 sec the application should be maintained until a maximum of 120 sec. If not, the application should be suspended followed by another attempt, after PVC remap [9,16].

AMC PVC recurrence is uncommon, even in patients without AAD treatment. Reappearance is more frequent in patients with an epicardial origin [5,8]. The operator experience, anatomic substrate location, PVC burden, the complexity the procedure, and stability and catheter contact can influence the safety and success of this procedure.

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