INNOVATIVE TECHNIQUES

COMPLEX CASE STUDY

Ablation of Ventricular Ectopy Arising from the Anterolateral Papillary Muscle: Utility of Intracardiac Echocardiography

CHRISTIAN PERZANOWSKI, MD, FACC, FHRS

Electrophysiology, Tampa General Hospital/Bay Area Cardiology, Brandon, FL

ABSTRACT. Idiopathic ventricular arrhythmias from the papillary muscles have been infrequently observed. The unusual location of the arrhythmogenic site lends itself to a technically challenging procedure. This case describes the value of the CARTOSOUND platform in mapping and ablating ventricular ectopy of anterolateral papillary muscle origin.

KEYWORDS. idiopathic ventricular tachycardia, papillary muscle, ventricular ectopy.

Introduction

Idiopathic ventricular tachycardia are a heterogeneous group of arrhythmias which tend to occur in the absence of any known structural cardiac abnormalities. The symptoms can be severe and poorly tolerated.1 The responsible mechanisms appear to be primarily triggered activity, with enhanced automaticity and microreentry accounting for a minority of cases. This accounts for varied presentations ranging from premature ventricular contractions to non-sustained ventricular tachycardia.2 Nearly all locations in both ventricles are known to harbor potentially arrhythmogenic tissue. Far less common however are ventricular arrhythmias originating from the papillary muscles.3 Even less frequent are foci exiting from the anterolateral papillary muscle (APM). Based on the available evidence, the following case describes the approach used to diagnose, map and ablate ventricular arrhythmia arising from the APM.

Case

A 46-year-old man was referred for highly symptomatic unifocal premature ventricular contractions (PVCs). The patient who was otherwise in good health began to experience disabling symptoms including angina and dizziness during the arrhythmic episodes. The onset and duration of the arrhythmia were variable, often occurring nocturnally and throughout the day as well. Holter monitoring recorded frequent bigeminy, and less frequently transient ventricular tachycardia. There was no family history of sudden death. The electrocardiogram was notable for PVCs with a right bundle branch block pattern and an inferior axis often appearing in bigeminy, or trigeminy; the QRS duration was 160 ms (Figure 1). Coronary computed tomography angiography excluded coronary artery disease. Echocardiography suggested mild ventricular impairment, with a left ventricular ejection fraction between 45% and 50%. Treatment with B-blockade was poorly tolerated due to hypotension. The patient was subsequently referred for electrophysiology study with possible catheter ablation.

The patient was brought to the electrophysiology suite in a fasting non-sedated state. The patient had not been treated with any antiarrhythmic or atrioventricular nodal blocking agents. Based on the electrocardiographic pattern, there was a high suspicion for APM origin.3,4 Based on previous reports of the intrinsic challenges in ablating the papillary muscles, a transseptal approach was chosen.5 A 10 French intracardiac echocardiography (ICE) probe (CARTOSOUND™, Biosense Webster, Diamond Bar, CA) was inserted via the left femoral vein to ascertain catheter contact and better visualize the papillary muscles. Moreover, a three-dimensional electroanatomic map was created with the CARTOSOUND™
Figure 1: The presenting electrocardiogram indicates sinus rhythm with unifocal ventricular ectopy. The right bundle branch morphology implies a left ventricular origin.

Figure 2: At the earliest site of activation, the timing of local electrogram to QRS was –0 ms. The double arrows highlight the multicomponent nature of the complex local electrogram. This was confirmed by intracardiac echocardiography to be a site of chordal insertion to the anterolateral papillary muscle. The single arrow contrasts a less remarkable appearance of the local electrogram in sinus rhythm. Electrode pairs are marked from proximal to distal. CS: coronary sinus; HIS: adjacent to septal tricuspid leaflet; RV: right ventricle; ABL: ablation catheter. Sweep speed is 200 mm/s.
platform. Additionally, a decapolar catheter was introduced into the coronary sinus via the right internal jugular approach, and a wide spaced decapolar catheter was placed near the right ventricular apex from the right femoral vein. The procedure was performed under general anesthesia with sevoflurane. Following induction, the patient remained in a non-paralyzed state. Sedation was lightened to permit spontaneous respiration and facilitate the appearance of spontaneous unifocal ventricular ectopy (VE).

As anticipated, programmed ventricular stimulation did not induce arrhythmia. However, the VE appeared spontaneously and frequently with the previously described sedation technique and without exogenous adrenergic stimulation. Following induction, the patient remained in a non-paralyzed state. Sedation was lightened to permit spontaneous respiration and facilitate the appearance of spontaneous unifocal ventricular ectopy (VE).

As anticipated, programmed ventricular stimulation did not induce arrhythmia. However, the VE appeared spontaneously and frequently with the previously described sedation technique and without exogenous adrenergic stimulation. Following induction, the patient remained in a non-paralyzed state. Sedation was lightened to permit spontaneous respiration and facilitate the appearance of spontaneous unifocal ventricular ectopy (VE).

Figure 3: Intracardiac echocardiography depicts a long axis view of the ablation catheter crossing the mitral valve and in contact with the proximal anterolateral papillary muscle, at the site of earliest activation (arrow). The chords are faintly seen. LA: left atrium; a: anterior septum; i: inferior septum.

Figure 3: Intracardiac echocardiography depicts a long axis view of the ablation catheter crossing the mitral valve and in contact with the proximal anterolateral papillary muscle, at the site of earliest activation (arrow). The chords are faintly seen. LA: left atrium; a: anterior septum; i: inferior septum.

As anticipated, programmed ventricular stimulation did not induce arrhythmia. However, the VE appeared spontaneously and frequently with the previously described sedation technique and without exogenous adrenergic stimulation. Following a standard transseptal catheterization with an SL-1 sheath, systemic anticoagulation was administered targeting ACT between 300 and 350 s. A 3.5 mm open irrigated tip catheter (Thermacool™, Biosense Webster) was used for mapping the left ventricle. Activation mapping localized the earliest activation to \(-0\) ms to the APM. ICE imaging visualized this focus to be near the origin of the chordae tendinae (Figure 2). At this location, the local electrogram was noted to be complex and multifractionated (Figure 3). Notably, Purkinje potentials preceding the complex electrogram were absent (Figure 4). Pace mapping was observed to be 12/12 at this location. Radiofrequency ablation was administered with 35 watts with 30-s applications. More aggressive ablation was tempered by concerns of potential damage to the chords. At no time did mitral regurgitation occur. The VE did not recur during the remainder of the case. At 6-month follow-up, the patient remained off medications and endorsed a marked reduction in symptom burden. He qualified for “80–90%” improvement despite residual VE being observed. A follow-up echocardiogram 15 months later indicated no loss of systolic function and the absence of any mitral regurgitation.

Discussion

Ventricular arrhythmia from the APM are uncommon, yet challenging to ablate. Yamada et al\(^3\) had suggested
one difficulty in ablation may be related to a deep endocardial origin. The papillary muscles are complex structures, and prior publications have been limited to small series of patients. The precise locations of the ablation sites along the papillary muscles have not been systematically detailed. It has been reported in the published literature that the thicker bases have been primarily targeted without the consistent use of ICE. However, in a separate publication, Yamada et al. report using ICE for papillary muscle ventricular tachycardia, including CARTOSOUND to guide ablation. The overall success rates for papillary muscle ablation have ranged from 42% to 78%. In one series, open irrigated or 8-mm tips were required in repeat procedures to maintain a successful result. The use of this modality on enhancing the success rate will need to be studied on a larger scale.

Yamada et al. published a brief series of six patients with VE arising from the APM. The base and mid-portion were found to contain the source of the ectopy. This isolated case demonstrates that exit sites may be found at the insertion of the chordae tendineae. It is noteworthy that the clinical implications of overzealous ablation could be mitral regurgitation secondary to severed chords. Despite a cautious approach, significant symptom reduction was observed.

Conclusions

Idiopathic ventricular arrhythmias originating from the APM are uncommon. This case highlights the use of the CARTOSOUND™ intracardiac echocardiography platform to precisely map an active focus near the emergence of the chordae tendineae. A satisfactory clinical result was obtained.

References


Figure 4: The electroanatomic map in the left anterior oblique view highlighting the earliest activation (red markers). Note the QS morphology of the unipolar electrogram (asterisk). On the MAP1-2 channel, the multicomponent appearance of the local electrogram can be appreciated.