Editorial

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Papillary Muscle Ventricular Tachycardia: Another Zigsaw Puzzle to Be Solved

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Idiopathic ventricular arrhythmias (VAs) with diverse mechanisms have been described in the left ventricle (LV). The predilection sites for these VAs are LV outflow tract, aortic cusps, mitral annulus, or epicardium (LV summit) and epicardial venous tissue (LV crux). Recently, idiopathic ventricular tachycardias (VT) or premature ventricular contractions (PVC) originating from the papillary muscles (PM) in the LV have been reported as a distinct clinical syndrome.[1-7]

The elegant studies by Rawling et al.[8] showed that the PM has a rich network of Purkinje fibers, that the distribution of subendocardial Purkinje to ventricular muscle electrical coupling is spatially inhomogeneous, and that the junctional regions themselves have variable degrees of electrical coupling. They indicated that ventricular muscle activation by the Purkinje network occurs only at discrete, localized regions near the PM base.[8]

The PM has been known to be implicated in arrhythmogenesis of both idiopathic left VAs and VAs due to structural heart diseases.[1-7] Experimental studies showed that the PM is important in the generation and the maintenance of reentry during VT and VF as a spiral wave anchoring site that stabilizes wave conduction.[9] These findings were confirmed again by Pak et al.[10] Idiopathic VAs from the PM are uncommon and rare in a sustained form.[4] They are not reproducibly induced by programmed stimulation and sensitive to catecholamines, which suggest triggered activity or abnormal automaticity as its mechanism. They occur more commonly from the posterior papillary muscle (PPM) than anterior papillary muscle (APM) as shown in prior studies[2)4-6] as well as in the study of Ban et al.[7]

The PM VAs should be differentiated from all VAs originating from LV outflow tract, aortic cusp, mitral annulus, LV epicardium, and even VT seen in patients with structural heart diseases. All these forms of VAs can be differentiated easily from the PM VAs by electrocardiographic morphology, or with the aid of sophisticated mapping technique such as entrainment and electro-anatomical mapping. The fascicular VT involving the fascicles of the specialized conduction system with either focal or reentrant mechanisms should also be differentiated since they have a similar QRS morphology.[2] Fascicular VTs had a right bundle branch block pattern and rsR' morphology in lead V1, in contrast to the PM VAs having a R or qR morphology in lead V1. The fascicular VT showed typical left anterior hemiblock or posterior hemiblock including discrete Q waves in leads I and aVL or III, II, and aVF, respectively.[2] In fascicular VAs, presystolic Purkinje potentials were identified at all effective ablation sites of the interventricular septum. The PM VAs had a broader QRS complex than that of the fascicular VT.[2)4] The PM VAs from the PPM displayed a right bundle branch block with superior axis while PM VAs originating from the APM had an inferior axis. The presence of an R/S ratio $\leq$1 in lead V6 and a QRS duration >160 ms suggests the PM VAs of APM and PPM origin, respectively.[4] These would be an additional clue to differentiate PM VAs from LV fascicular VT.

In this issue of the Korean Circulation Journal, Ban et al.[7] tried to find specific electrophysiological characteristics which can predict a successful outcome after radiofrequency catheter ablation (RFCA) of VAs originating from PM in the LV. Eight patients (67%) among 12 patients who had VT or PVCs of LV PM origin (4.2% of total 284 consecutive patients with idiopathic VAs) showed "high-amplitude discrete potentials". Seven out of 8 patients showing "high-amplitude discrete potentials" at the ablation site had a successful outcome (85.7%), while the remaining 4 patients who showed low-amplitude

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fractionated potentials at the ablation site experienced VAs recurrence. Second, the mean duration from onset to peak downstroke (Δt) of unipolar electrogram was significantly longer in the successful group than the recurrence group (58±8 ms vs. 37±9 ms, p=0.04). And slow downstroke >50 ms of the initial Q wave of unipolar electrogram at ablation sites was also significantly associated with successful outcome (85.7% vs. 25.0%, p=0.03).

It is noteworthy that the authors found the 2 new electrophysiological markers which can predict the successful outcome after RFCA; 1) presence of the "high-amplitude discrete potentials" defined as >1.0 mV at ablation sites, 2) duration from onset to peak downstroke (Δt) of unipolar electrogram >50 ms.

The authors speculated that the proximity of ablation catheter to arrhythmogenic foci is relevant to the differences between low and high amplitude potentials. In other words, high-amplitude potentials might reflect near field activities of the subendocardial VA origin in the PM, whereas low-amplitude fractionated potentials might reflect far field activities of the VA origins deep inside the PM. The so-called "high-amplitude discrete potentials" used in this study seemed to be equivalent to the term "a sharp ventricular prepotential" in other studies,[4)6)] in which prepotentials were recorded at the successful ablation site during the PM VAs in only about 40% of the patients who were successfully ablated. Thus, whether the "high-amplitude discrete potential" observed at ablation sites implies a pathognomonic marker which can localize the VAs origin or not need to be tested in further studies. The possible explanation for slow downstroke of the initial Q wave in unipolar electrocardiogram was that this might reflect the slow propagation of wavefront from the foci of the arrhythmogenic PM to surrounding myocardium.

Observations on the presence of Purkinje potentials at the effective ablation site during both sinus rhythm and the PM VAs showed some discrepancies - from none to 45% - depending on the investigators.[2)4-7)] Even though Purkinje potentials were recorded during sinus rhythm at the site of successful ablation, the local ventricular muscle potential preceded the putative Purkinje potential during PVCs, suggesting that PVCs originate from the ventricular muscle rather than the conduction system,[1)] whereas the presence of preceding Purkinje potentials were associated with a successful outcome in other study.[5)] However, the lack of high-frequency potentials preceding the VT may not exclude involvement of Purkinje fibers deep relative to the endocardial surface.

Interestingly, heterogeneous uptake of gadolinium with delayed enhancement during magnetic resonance (MR) imaging was observed in arrhythmogenic PM in post-infarction VAs originating from the PM,[3)] and even in patients without a structural heart disease.[2)5)] However, it is difficult to draw a firm conclusion on the correlation between abnormal uptake in the PM on MR imaging and arrhythmogenecity due to the scarcity of data. The role of mechanosensitive stretch-activated channels in PM arrhythmogenesis would form an another area of research and should be addressed in future studies.

In terms of RFCA treatment, the success rate is reported to be rather low due to the difficulty in maintaining a stable contact of the catheter tip with the PM and the site of origin located somewhat deep relative to the endocardium.[6)] For these reasons, ablation can be challenging, and irrigated catheter ablation may be necessary to achieve long-term success.

In conclusion, the 2 predictors proposed by Ban et al.[7)] in this study would be helpful to find appropriate ablation sites. However, they should be validated in a rather large cohort with the PM VAs. A lot needs to be done in order to elucidate the role and electrophysiological characteristics, including the interaction between the Purkinje network and the PM in PM arrhythmogenesis.

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