Old but not Vain: Two Cases of Refractory Polymorphic Ventricular Tachycardia associated with Coronary Artery Disease successfully treated with Quinidine

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**Recommended Citation**

Tit-Kei Ng, Tommy King-Him Ma, Chi-Chun Au, Janice Woon-Yan Wong, Jenny Nga-Lai Chow, Andrew Vincent Li, Adrian Yin-Cheung Luk, Sze-Wah Lai, Candy Ming-Yan Cheuk, Jaclyn Chi-Lin Chan, Li-Wah Tam, Old but not Vain: Two Cases of Refractory Polymorphic Ventricular Tachycardia associated with Coronary Artery Disease successfully treated with Quinidine *Journal of the Hong Kong College of Cardiology* 2023;29(5) [https://doi.org/10.55503/2790-6744.1496](https://doi.org/10.55503/2790-6744.1496)

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This case report is available in Journal of the Hong Kong College of Cardiology: https://www.jhkcc.com.hk/journal/vol29/iss5/5
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CASE REPORT

Old but not Vain: Two Cases of Refractory Polymorphic Ventricular Tachycardia Associated with Coronary Artery Disease Successfully Treated with Quinidine

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Abstract

Post-myocardial infarction polymorphic ventricular tachycardia (VT) triggered by injured Purkinje fibres can cause VT storms that are refractory to various treatment even after successful reperfusion or revascularization. We report recurrent polymorphic VT in two post-myocardial infarction patients after percutaneous coronary intervention. Both of them responded readily to Quinidine. We also discussed the pathophysiology of this arrhythmia and the treatment options for it, namely catheter ablation and anti-arrhythmic drugs.

Keywords: Myocardial infarction, Myocardial ischemia, Quinidine, Ventricular tachycardia

Case 1

A 43-year-old gentleman with history of 20-pack-year of cigarette smoking and newly diagnosed diabetes mellitus, was admitted in early 2022 for congestive heart failure with chest discomfort. ECG showed ST segment depression in lateral leads and T wave inversion in inferior leads. His echocardiogram showed left ventricular ejection fraction (LVEF) of around 30%. He was treated as non-ST elevation acute coronary syndrome (NSTEMI). Soon after admission he ran a deteriorating course and was in cardiogenic shock requiring intubation and inotropic support. Urgent coronary angiogram was done showing triple vessel disease with acute distal right coronary artery (RCA) thrombotic lesion. The RCA was intervened, and the left anterior descending (LAD) chronic total occlusion was also intervened in a staged manner. An Impella device was also placed for circulatory support. He was given dialysis support due to acute renal failure.

However after revascularization, he developed recurrent polymorphic ventricular tachycardia (VT) (Figure 1), all preceded by a premature ventricular complex (PVC) of relatively narrow QRS with a relatively short coupling interval (~400 ms) (Figure 2). He was subsequently put on amiodarone and temporary transvenous pacing was commenced. Stellate ganglion block was also performed. Nevertheless, there was still recurrence of polymorphic VT necessitating defibrillation, and he remained in critical condition. Quinidine 200mg q6h was started. There was no more defibrillation after starting the drug.

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https://doi.org/10.55503/2790-6744.1496
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Figure 1. Polymorphic VT triggered by Purkinje PVC. Note the short coupling interval of the initiating PVC and the normal baseline QTc, which speak against Torsade de Pointe. VT, ventricular tachycardia; PVC, premature ventricular complex.

Figure 2. 12-lead ECG showing Purkinje PVC of RBBB configuration with short intrinsicoid deflection. PVC, premature ventricular complex; RBBB, right bundle branch block.
patient could be weaned off from Impella support and temporary pacing was removed subsequently. He ran a satisfactory recovery course. A dual-chamber ICD was implanted when he was stabilized haemodynamically and electrically. All anti-arrhythmic drugs were stopped before discharge. Upon device follow up, there was no ventricular arrhythmia episode. PVC count was 0.1%.

Case 2

A 67-year-old gentleman with a history of 40-pack-year of cigarette smoking presented in mid-2021 for congestive heart failure. Echocardiogram showed impaired LVEF ~40% and akinetic anterior wall and apex, with a suspected left ventricular (LV) thrombus. Subsequent cardiac MRI confirmed the presence of LV thrombus with evidence of inducible ischemia. Percutaneous coronary intervention (PCI) was scheduled but he developed acute NSTEACS presenting with acute crushing chest pain before PCI. After admission, there were episodes of polymorphic VT triggered by a relatively narrow PVC requiring repeated defibrillation. The patient was initially given boluses of intravenous amiodarone and magnesium sulphate, and he was put on temporary pacing. Urgent PCI to the culprit LAD was performed. However, despite successful revascularization and ongoing amiodarone infusion, there were still multiple early coupled (~400 ms) Purkinje PVCs (Figure 3), some of which triggered polymorphic VT mandating defibrillation and resulting in VT storm. Since the presence of LV thrombus precluded catheter PVC ablation, the patient was then prescribed Quinidine Sulphate 400mg Q6H. After the Quinidine treatment, there was no further polymorphic VT nor Purkinje PVC. No further defibrillation was needed. The dose of Quinidine was then gradually tailed down to 200 mg QID.

After the patient was free from ventricular arrhythmias for a few days, a dual-chamber ICD was implanted. The patient was then discharged with antiplatelet agents, warfarin (for his LV thrombus), standard heart failure medication and Quinidine.

Upon follow-up visit 2 months after discharge, the patient was in NYHA class I, remained well and ambulatory. His device interrogation showed <1% ventricular pacing. There was no ventricular arrhythmia episode. PVC count from the device was less than 1%. Quinidine was continued. It was planned to stop Quinidine if device interrogation later showed no ventricular arrhythmias recurrence.

Discussion

Post-myocardial infarction spontaneous polymorphic VT in the absence of myocardial ischemia is well described in several case series [1–4]. It may occur 3–8 days after myocardial infarct or coronary revascularization. Not uncommonly, once it occurs, it can cause clusters of arrhythmic storms which is difficult to treat as conventional antiarrhythmic drugs are generally ineffective.

This polymorphic VT is usually initiated by ventricular extrasystoles displaying short coupling interval. These short-coupled ectopic beats originate from ischemia-resistant Purkinje fibres that survive, though injured, in areas of myocardial scar or the scar border. Animal studies [5] have shown that these surviving post-myocardial infarction Purkinje fibres had altered electrophysiological properties, e.g., shortened action potential duration, creating optimal condition for reentry. Some of these Purkinje fibres may even exhibit spontaneous diastolic depolarizations (early afterdepolarizations) and give rise to extrasystoles. Hence, these fibres may act both as triggers and as substrate for initiation and maintenance of polymorphic VT.

In those early series describing this arrhythmia, the patients were treated as a last resort with catheter ablation, where focal Purkinje-related triggers arising from the scar border zone at the left ventricular septum were targeted. However, catheter ablation during this VT storm is often more easily said than done. Frequently, polymorphic VT/or ventricular fibrillation will be triggered by the Purkinje’s PVC, precluding accurate mapping of its origin. Also, various mechanical circulatory supports which are usually necessary in this circumstance can impose difficulties on vascular access and catheter manipulation, adding further challenge to the procedure. It is not surprising that in the Komatsu’s series [4], only 5 out of 18 patients having ventricular fibrillation storm could survive even after Purkinje PVC ablation. Therefore, catheter ablation is not the universal answer for all the patients, not to mention there are patients having contraindication to catheter ablation, like the presence of LV thrombus.

Recently, Viskin et al [6] reported the success of Quinidine in treating this group of patients. In his series, 43 patients with this arrhythmia were identified. 22 of them received Quinidine, and out of these 22 patients, 17 of them experienced ventricular fibrillation storm. All of them responded to Quinidine quickly and dramatically. Impressively all 22 patients receiving Quinidine including those...
experiencing ventricular fibrillation storm were discharged alive, and none of them required bailout catheter ablation. The safety profile was also promising. Serious adverse effects occurred in 3 patients which resolved upon drug discontinuation.

Quinidine is a Class 1A antiarrhythmic drug. Among its different channels blocking effect, Quinidine in particular is a strong blocker of the transient outward potassium channels, which are highly expressed in Purkinje fibers [7]. This could explain the specific action of Quinidine in preventing Purkinje fibre-related arrhythmia.

To conclude, though generally considered as contraindicated in organic heart disease, Quinidine may be lifesaving for patients with coronary artery disease developing arrhythmic storms attributable to polymorphic VT. Compounded to its efficacy in treating

Figure 3. Polymorphic VT initiated after a Purkinje PVC. There was no “short-long” cycle preceding the onset of the VT. VT, ventricular tachycardia; PVC, premature ventricular complex
other arrhythmic storms in primary electrical disorders like Brugada syndrome and early repolarization syndrome, Quinidine stocking in every acute hospital's pharmacy should be seriously considered.

**Funding**

None.

**Conflict of interest**

None declared.

**Acknowledgements**

None.

**References**


