Role of retrograde conduction from the left bundle to right bundle in determining the morphology of premature ventricular contractions triggering ventricular fibrillation

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Introduction

Ventricular fibrillation (VF) after an acute myocardial infarction (AMI) is a relatively common occurrence, with a significant number of cases being triggered by premature ventricular contractions (PVCs) originating from the Purkinje network.[1] Komatsu and colleagues demonstrated the efficacy of catheter ablation of after an AMI by targeting the triggering PVC while using pace mapping to identify the ablation target.[2] An accurate identification of the PVC morphology during catheter ablation is imperative for a successful procedure. However, PVCs may not always manifest during the procedure, potentially owing to the effects of sedation or the sensation of pain and associated discomfort resulting from catheter manipulation. Another challenge during the ablation is the presence of multiple PVC morphologies, including a "nontriggering" PVC morphology that does not require ablation. Therefore, distinguishing between triggering PVCs and nontriggering PVCs is crucial.[3]

This study presented a case of VF and ventricular tachycardia (VT) occurring during the subacute phase of myocardial infarction. The triggering PVC originated from an abnormal Purkinje fiber in the left ventricular (LV) septum, with a significant preceding interval from the Purkinje potential to the triggering PVC. Interestingly, a narrower nontriggering PVC morphology was also observed when the firing from the abnormal Purkinje conducted retrogradely from the left bundle to the right bundle. Conversely, the triggering PVC appeared only in the absence of retrograde conduction from the left bundle and was followed by delayed local reentrant ventricular excitation at the LV septum. During the procedure, no episodes of VF or sustained VT were observed. However, a single episode of spontaneous nonsustained VT (NSVT) occurred after the appearance of the triggering PVC with a Purkinje potential intervening among ventricular excitations. Moreover, programmed ventricular stimulation from the LV septum exclusively induced NSVT when the paced QRS morphology exhibited a triggering PVC morphology. That observation suggested that the retrograde conduction block from the left bundle branch to the right bundle branch may have been associated with the initiation of the NSVT or VF in this particular case.

KEY TEACHING POINTS

- Ablation of triggering premature ventricular contractions (PVCs) has shown efficacy in the management of ventricular fibrillation.
- The morphology of triggering PVCs can vary depending on the presence or absence of retrograde conduction from the left bundle.
- The appearance of a triggering PVC morphology was consistently associated with the induction of nonsustained ventricular tachycardia during programmed ventricular stimulation.

Case report

A 62-year-old male patient was initially referred to another hospital owing to myocardial infarction involving the left anterior descending coronary artery. Percutaneous coronary intervention (PCI) was performed 8 days later. However, subsequent VF episodes occurred, leading to the insertion of venoarterial extracorporeal membrane oxygenation (VA-ECMO). During this period, in-stent thrombosis was identified, necessitating repeat PCI. VF reoccurred after the

KEYWORDS Catheter ablation; Ventricular Fibrillation; Purkinje; Acute Myocardium Infarction
(Heart Rhythm Case Reports 2024;10:63–67)

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https://doi.org/10.1016/j.hrcr.2023.10.024

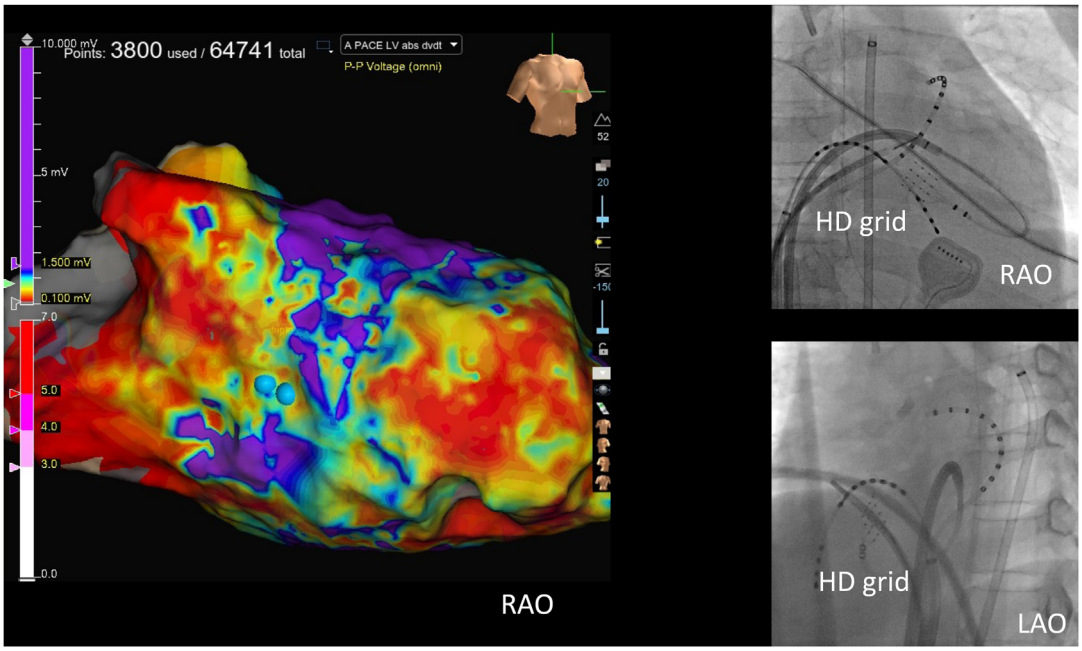

Figure 1 The left ventricular (LV) septum exhibited a relatively preserved voltage area surrounded by infarcted basal and apical regions. The fluoroscopic image depicts the placement of the HD Grid catheter (Abbott, Abbott Park, IL) on the LV septum. LAO = left anterior oblique, RAO = right anterior oblique.

repeat PCI and removal of the VA-ECMO, prompting the reinsertion of the VA-ECMO. The patient was referred to our hospital at this stage owing to frequent VF storms. The patient exhibited normal sinus rhythm at baseline, but frequent PVCs were observed and were identified as triggers of VT and VF (Supplemental Figures 1 and 2). Catheter ablation was attempted during the support of the VA-ECMO device.

Despite the absence of PVCs at the beginning of the procedure at baseline, PVCs with multiple morphologies emerged following intravenous isoproterenol administration. Two different PVC morphologies were observed, one with left bundle branch block morphology and the other with right bundle branch block morphology. Notably, both PVCs were significantly preceded by abnormal Purkinje potentials, which were detected using an HD Grid Mapping Catheter (Abbott, Abbott Park, IL) placed on the LV septum. The LV septum exhibited a relatively preserved voltage area between the infarcted basal and apical regions on the 3D mapping (Figure 1). The triggering PVC displayed a right bundle branch block morphology characterized by distinctive qR pattern in lead V_1. Prior to the occurrence of the PVC, an abnormal Purkinje potential was observed, which appeared 212 ms earlier than the PVC and was separated by an interval of 206 ms from the preceding local LV excitation (Figure 2A). The coupling interval between the preceding sinus beat and PVC was 520 ms; and following the PVC, a delayed local reentrant ventricular excitation was observed in the LV. Notably, right bundle potentials were absent from the recordings of the catheter placed in the right ventricle (RV) during the PVC.

Conversely, a nontriggering PVC characterized by an rS pattern in lead V_1 was observed and was preceded by an abnormal Purkinje potential at the same location on the LV septum (Figure 2B). The Purkinje potentials preceding the nontriggering PVC demonstrated a shorter interval to the subsequent PVC (88 ms) and shorter coupling interval to the preceding sinus beat (452 ms) but a longer interval from the preceding LV excitation (260 ms) as compared to the triggering PVC. Unlike the triggering PVC, no delayed ventricular excitation on the LV septum was observed after the nontriggering PVC. However, a right bundle branch potential was recorded during the nontriggering PVC, suggesting retrograde conduction from the left bundle to the right bundle. Compared to the triggering PVC, the narrower width of the nontriggering PVC was attributed to retrograde conduction from the left bundle to the right bundle. The difference in the ventricular activation observed on the HD grid between the triggering PVC and nontriggering PVC suggested variations in the propagation patterns within the LV septum during those 2 PVCs.

Throughout the procedure, no spontaneous VF episodes were observed. However, a single episode of spontaneous NSVT occurred, initiated by the triggering PVC that lacked an RV potential, with alternating excitation of the LV and Purkinje potentials (Figure 3A). Subsequent ventricular programmed stimulation from the LV septum using the HD Grid catheter consistently induced NSVT, mimicking the earlier observed spontaneous NSVT characteristics with alternating excitation of the LV and Purkinje potentials. Notably, the initial beat of the induced NSVT exhibited a morphology similar to that of the triggering PVC, corresponding to the refractory period of the retrograde conduction from the left bundle (Figure 3B). Conversely, we consistently observed that no NSVT occurred when the retrograde conduction

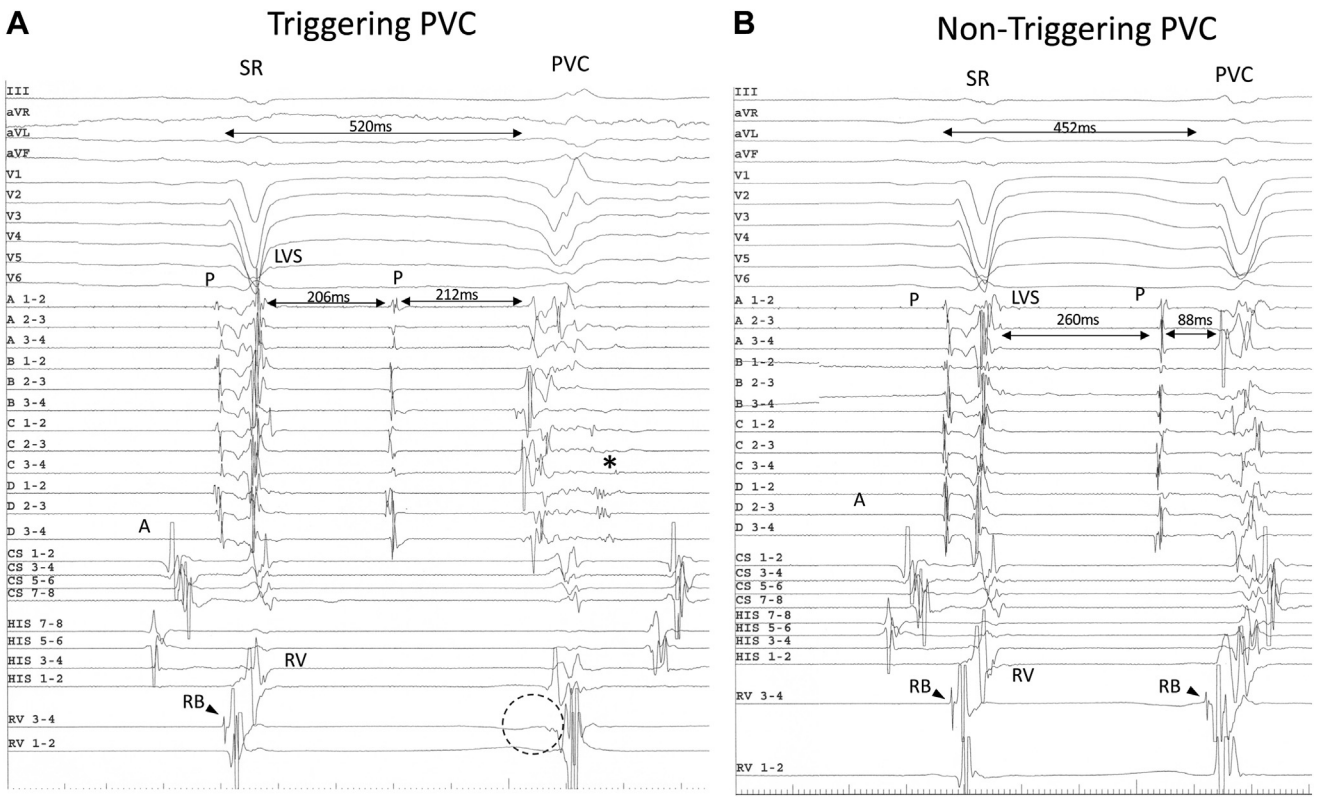

Figure 2 **A:** The triggering premature ventricular contraction (PVC) exhibited a right bundle block morphology with a distinct qR pattern in lead V_1. An abnormal Purkinje potential preceded the PVC by 212 ms. The coupling interval from the preceding sinus beat to the PVC was 520 ms. The local left ventricular (LV) potential exhibited a delayed "local reentrant" ventricular excitation following the PVC (*). Notably, no right bundle potentials were recorded by the catheter placed in the right ventricle during the PVC (dotted circle). **B:** A nontriggering PVC displayed an rS pattern in V_1 with a preceding abnormal Purkinje potential on the LV septum. The coupling intervals from the Purkinje potential to the PVC and preceding sinus beat to the PVC were shorter (88 ms and 452 ms, respectively) but longer from the preceding local LV excitation to the Purkinje potential (260 ms) than the triggering PVC. No delayed ventricular excitation on the LV septum followed the nontriggering PVC, yet a right bundle branch potential was recorded during the PVC, indicating retrograde conduction from the left bundle. The narrower morphology of the nontriggering PVC is attributed to retrograde conduction from the left bundle to the right bundle. (A–D represents each spline of the HD grid.) A = atrium; CS = coronary sinus; HIS = His bundle electrogram; LVS = left ventricular septum; P = Purkinje potential; RB = right bundle; RV = right ventricle; SR = sinus rhythm.

from the left bundle to the right bundle was still present during programmed stimulation from the LV septum (Supplemental Figure 3). This relationship between the inducibility of the NSVT and absence of retrograde conduction was consistently observed and reproducible. Extensive ablation was performed, targeting the Purkinje potentials in the LV septal region, which coincided with the scar border area. Following the ablation procedure, no PVCs were observed, and no episodes of NSVT could be induced. Subsequently, the patient was successfully weaned off VA-ECMO and discharged a few weeks later. The patient remained free from any recurrence of VF or VT for 1 year.

Discussion

In this case, we performed a catheter ablation of VF-triggering PVCs originating from the Purkinje network on the LV septum after an AMI. Interestingly, we observed distinct QRS morphologies of the PVCs, depending on the presence or absence of retrograde conduction from the left bundle to the right bundle, despite both being triggered by Purkinje potentials originating from the same location on the LV septum. The morphology of the spontaneous NSVT episodes closely resembled that of the triggering PVCs. Notably, NSVT was induced during programmed ventricular stimulation only when the paced QRS morphology transitioned from the nontriggering PVC morphology to the triggering PVC morphology, coinciding with block of the retrograde conduction from the left bundle to the right bundle after reaching the refractory period.

Morphology of the triggering PVC

Catheter ablation targeting PVCs that trigger VF after an AMI can effectively suppress VF. During such procedures, abnormal Purkinje potentials preceding the PVCs are frequently observed and are the primary ablation targets.[4] These Purkinje potentials often originate from the infarcted area near to the His-Purkinje system. In the present case, the Purkinje fibers responsible for the triggering PVCs were found to originate from the LV septum, specifically within the border zone of the infarcted region.[5] Although catheter ablation targeting Purkinje potentials has been proven effective, it is not uncommon for triggering PVCs

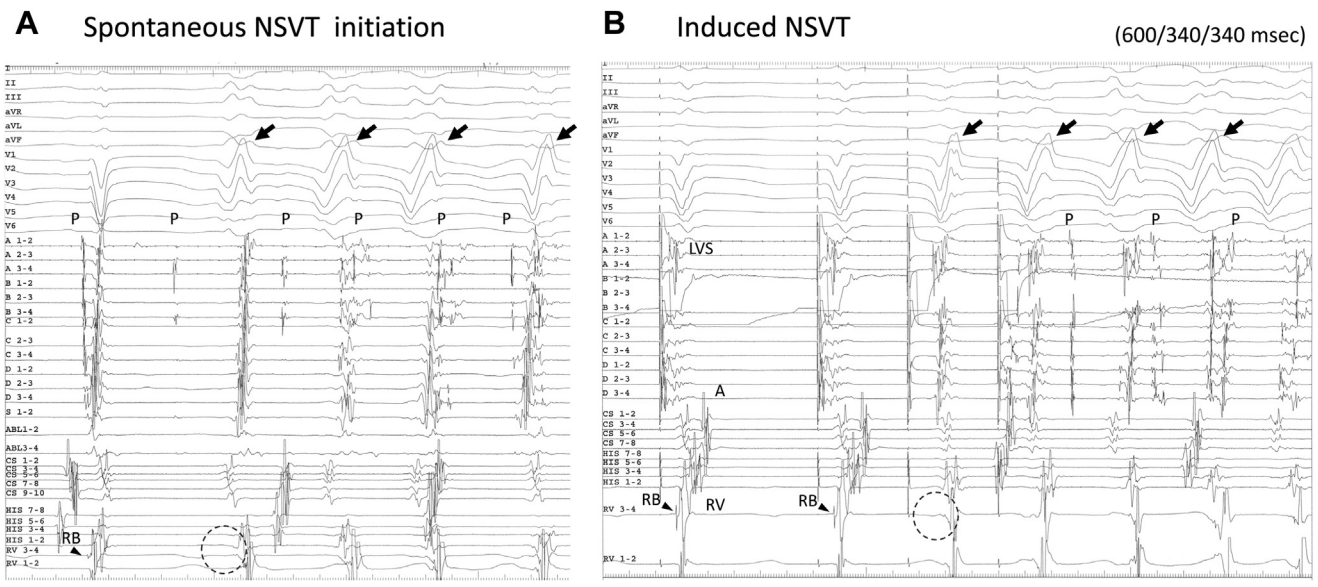

Figure 3 **A:** A single episode of spontaneous nonsustained ventricular tachycardia (NSVT) was observed during the procedure, initiated by the triggering premature ventricular contraction (PVC). The triggering PVC lacked a right ventricular potential and involved Purkinje intervention within the ventricular excitation. **B:** The induced NSVT by programmed ventricular stimulation (600/340/340 ms) mimicked the characteristics of the earlier observed spontaneous NSVT, with the involvement of Purkinje fibers. Interestingly, the initial beat of the induced NSVT displayed a morphology resembling that of the triggering PVC, corresponding to the refractory period of the retrograde conduction from the left bundle. A = atrium; CS = coronary sinus; HIS = His bundle electrogram; LVS = left ventricular septum; P = Purkinje potential; RB = right bundle; RV = right ventricle.

to cease occurring during the procedure, making ablation challenging. In such scenarios, accurately identifying the PVC morphology is crucial when performing ablation with the assistance of pace mapping. Moreover, since multiple morphologies of PVCs can manifest during the procedure, distinguishing between triggering and nontriggering PVCs becomes imperative for successful ablation. In our case, both triggering and nontriggering PVCs originated from the Purkinje network on the LV septum. However, the triggering PVCs exhibited a shorter coupling interval from the Purkinje firing to the preceding sinus beat than the nontriggering PVCs, resulting in a longer interval to the occurrence of the PVC. Conversely, nontriggering PVCs had a longer coupling interval but a shorter interval to the PVC itself. Notably, retrograde conduction from the left bundle to the right bundle was exclusively observed in nontriggering PVCs, resulting in a narrower PVC width. In contrast, no retrograde conduction within the cardiac conduction system was observed during the triggering PVCs. Instead, a subsequent delayed local ventricular potential was observed. To the best of our knowledge, our case represents the first demonstration of retrograde conduction from the left bundle to the right bundle originating from an abnormal Purkinje firing of PVC. The presence or absence of retrograde conduction is closely associated with variations in the PVC morphology. Therefore, when performing ablation of triggering PVCs in cases of VF, it is crucial to record the His bundle potential or bundle branch potential to assess the presence or absence of retrograde conduction, which can significantly influence the PVC morphology. This recommendation is based on actual clinical needs and is not merely for research purposes. Furthermore, it has been noted that abnormal Purkinje potentials often display a substantial preceding interval prior to the occurrence of the PVC/NSVT during the procedure. This suggests that the "breakout" site of PVC/NSVT may be distant from the site of the Purkinje potential and may also be influenced by conduction delays within the Purkinje and LV scar.[6] Considering these findings, it is crucial to prioritize the ablation of the Purkinje potential itself when it is clearly mapped and localized during the procedure, as demonstrated in the current case. Conversely, in situations where the Purkinje potential was not documented, and clinical PVCs were not recorded during the procedure, it is beneficial to refer to the morphology of the intracardiac electrogram on the implanted implantable cardioverter-defibrillator when available in patients.

VT and VF as Purkinje reentry

Recently, it has been reported that most ventricular activities driving the onset of VF arise from the Purkinje network and myocardial substrate.[7] As the "Purkinje entry" concept indicates, Purkinje fibers are more often becoming targets of catheter ablation of VF.[8] One notable feature in the current case was that only the triggering PVC, which was not associated with the retrograde conduction from the left bundle to the right bundle, exhibited a delayed local ventricular potential indicating local reentry after the PVC. Furthermore, a spontaneous NSVT manifested with a first beat resembling the triggering PVC. Similarly, the induced NSVT only occurred when the paced QRS morphology resembled the triggering

PVC, coinciding with the absence of retrograde conduction from the left bundle to the right bundle. Firing from a Purkinje fiber with a shorter coupling interval to the preceding LV excitation, which is more susceptible to retrograde conduction block from the left bundle owing to its refractory period, can result in a further conduction delay in both Purkinje fiber and the infarcted left ventricle, thus inducing NSVT by the triggering PVC. This explanation can also be extended to programmed ventricular stimulation on the LV septum, where a shorter coupling interval with a triggering PVC morphology can exclusively induce NSVT. The disparities in the LV propagation within the HD grid between the triggering PVC and nontriggering PVC provided further support for this explanation, emphasizing the variations in the propagation patterns within the LV septum during triggering PVCs and nontriggering PVCs. On the other hand, the consistent noninducibility of NSVT by a nontriggering PVC morphology, both spontaneously and during programmed ventricular stimulation on the LV septum, gave rise to an alternative hypothesis concerning the occurrence of NSVT and a triggering PVC morphology. This hypothesis proposes that the retrograde conduction from the left bundle to the right bundle encounters and disrupts any reentrant activity within the LV septum, thereby preventing the establishment of sustained reentry.

In spontaneous NSVT, there was no precise correlation between the P-P interval and the V-V interval. We classified this tachycardia as VT owing to its nearly identical morphology and tachycardia cycle length. However, the mechanism of this VT aligns with the contemporary concept of "organized VF," characterized by irregular cycle length but driven by a reentry mechanism. It is important to note that the relationship between Purkinje activity and the local electrogram is not a strict one-to-one correspondence.[7]

Acknowledgments

We thank John Martin for his linguistic assistance in writing the manuscript.

Funding Sources: None.

Disclosures: No conflicts of interest related to this topic.

Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.10.024.

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