Possible feature of intrascar ventricular tachycardia: A case of recording constant diastolic potential within extensive scar despite irregular pleomorphic QRS manifestation

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Introduction

The primary mechanism underlying ventricular tachycardia (VT) in structural heart disease is scar-related reentry. The circuit for scar-related reentrant VT usually involves the outer loop of the healthy myocardium and the critical isthmus within a scar or border zone. However, with only 1 reported case of "intrascar VT,"[1] understanding the essential VT reentrant circuit completed within a low-voltage scar remains limited. Moreover, there have been no documented reports of pleomorphic VT associated with "intrascar VT." In this study, we encountered a unique case of suspected "intrascar VT" where the QRS-QRS interval and QRS morphology on the surface electrocardiogram changed and demonstrated a pleomorphic manifestation despite the constant cycle lengths (CL) of diastolic potential (DP) within the low-voltage scar.

Case report

A 71-year-old man with a history of lateral myocardial infarction (Supplemental Figure 1) and an implantable cardioverter-defibrillator was transferred to our institution for catheter ablation of multiple implantable cardioverter-defibrillator shocks due to VTs. The QRS complex morphology displayed polymorphic features through 2 main types of QRS (VT1, defined as right bundle branch block type and positive concordance; and VT2, defined as right bundle branch block type with a V_5 transition) waveforms that periodically appeared and subsided, as shown in Figure 1.

KEY TEACHING POINTS

- While ventricular tachycardia (VT) has a pleomorphic manifestation, constant cycle lengths of diastolic potentials can represent a critical isthmus of VTs.
- Intrascar VT, where the whole circuit is included in an extensive scar, can show the pleomorphic VT exits.
- In "intrascar VT" with a pleomorphic manifestation, even though the diastolic potential was recorded, it could not always indicate the critical isthmus. Therefore, it is essential to identify the critical isthmus or the entire circuit and administer appropriate ablation treatment.

During VTs, a 1-liner decapolar catheter (1 mm electrode size and 2-8-2 mm interelectrode spacing; DECANAV; Biosense Webster, Diamond Bar, CA) was positioned at the lateral part of the left ventricle, marked as # in Figure 2A within a low-voltage area defined by a bipole voltage <0.5 mV. This positioning revealed potential with a constant CL, and an identical waveform was consistently recorded during diastole (Figure 3; DP). The DP-QRS interval showed variations between the VT1 and VT2 (Figure 3). During VTs, identical morphology of VT1 was recorded with latency from the pacing site, where a constant DP was registered (Supplemental Figure 2). We ablated this DP site during the mixed appearance of VT1 and VT2, which resulted in the termination of VTs with only a single application of ablation. Subsequently, VTs were no longer induced (Supplemental Figure 3).

KEYWORDS QRS morphology; Pleomorphic ventricular tachycardia; Intrascar ventricular tachycardia; Ischemic cardiomyopathy; Catheter ablation

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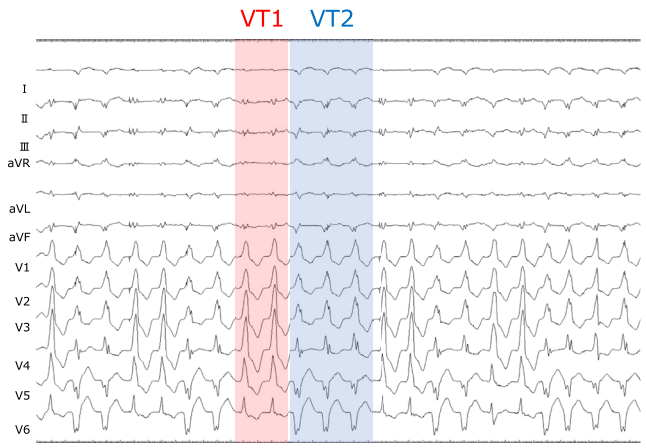

Figure 1 Twelve-lead electrogram illustrating pleomorphic-like ventricular tachycardia (VT). They were approximately classified as VT1 and VT2.

Discussion

There are reports indicating that the CL of VT can vary with the oscillation of DP CLs.[2] However, there have been no reports suggesting that the CLs and sequences of DPs are constant, despite the presence of 2 main types of QRS morphologies with irregular CL. Regardless, this is a fascinating case involving the establishment of an essential circuit solely within the scar.

Estimated circuit of VTs

The instability in morphologies and CL of VTs, coupled with the mapped area covered by low voltage, could have limited the mapping points that 3D mapping systems can obtain, preventing the acceptance of detailed potentials (Supplemental Video 1). Additionally, entrainment maneuvers were not applicable in cases of CL changes. While nonreentrant tachycardia, localized reentry, or complex VTs related to circuit composition cannot be ruled out, some clues toward understanding simple VT mechanisms have been acquired. First, DPs with CL and waveform remained constant in low-voltage areas, supporting the notion that the critical pathway is stable with regular excitation. Furthermore, ablation at the most upstream region of the DP, where fragmented potentials fulfilled a relatively wide range of the CL, was recorded very close to the ablation site, resulting in the termination of the pleomorphic VTs with only 1 burn application, capturing crucial parts of those VTs (Supplemental Figure 3).

Second, regarding the activation of VTs, when positioning the linear decapolar catheter along the scar area, the DP on bipole pairs 9-10 preceded the DP on bipole pairs 1-2. This pattern represents a gradient from the apex to the basal LV toward the exits of VT1 at the base of the LV lateral (Supplemental Figure 4). As VT2 appeared, the DP-QRS interval shortened, indicating that the excitation propagated from different sites to the nonscar region before reaching the

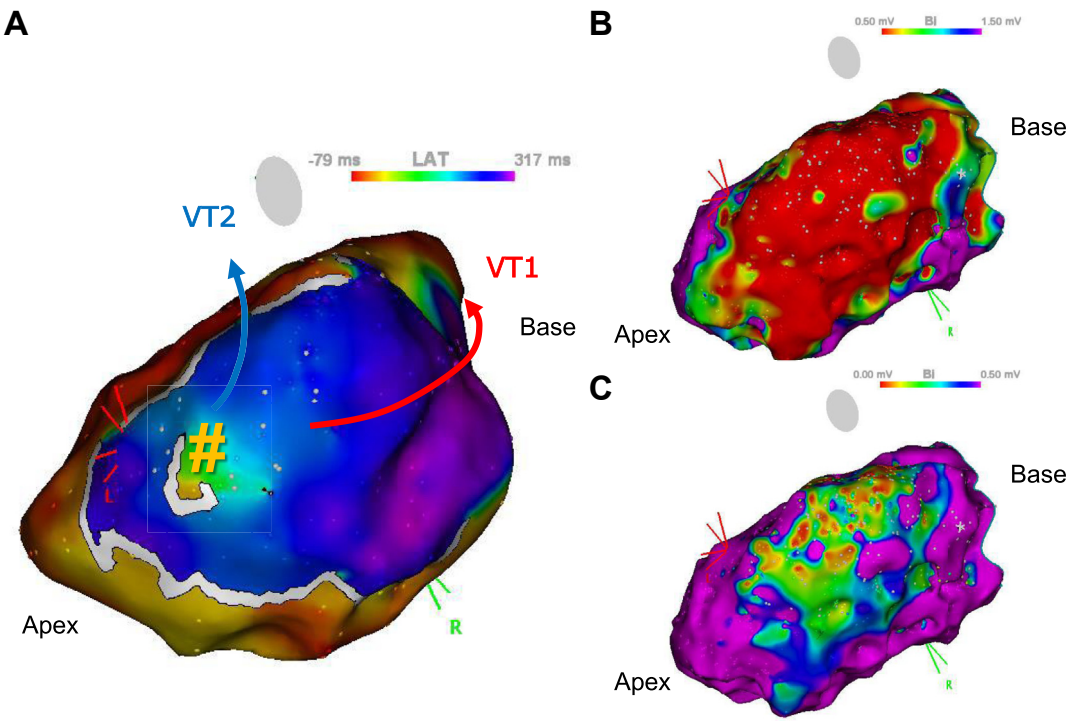

Figure 2 **A:** A schematic of the ventricular tachycardia (VT) circuit, with # indicating the estimated common isthmus area of VTs. The red and blue arrows indicate the propagation of VT1 and VT2 to nonscar area, respectively. The instability in the propagation of the blue arrow, representing VT2, is expected to be the most significant factor in the pleomorphic manifestation. **B:** The bipolar voltage map of the left ventricle (LV) during sinus rhythm. The purple area indicates a preserved bipolar voltage area of $\geq$1.5 mV. The scar area, colored red, shows a bipole voltage ($<$0.5 mV), while the scar border zone area, colored orange, yellow, green, and blue, successively, features a voltage of 0.5–1.5 mV. **C:** The dense scar bipolar voltage map of the LV during sinus rhythm, with adjustment of color threshold to 0–0.5 mV in panel B. The dense scar area, colored red, orange, yellow, green, and blue, successively, features a voltage of 0–0.5 mV.

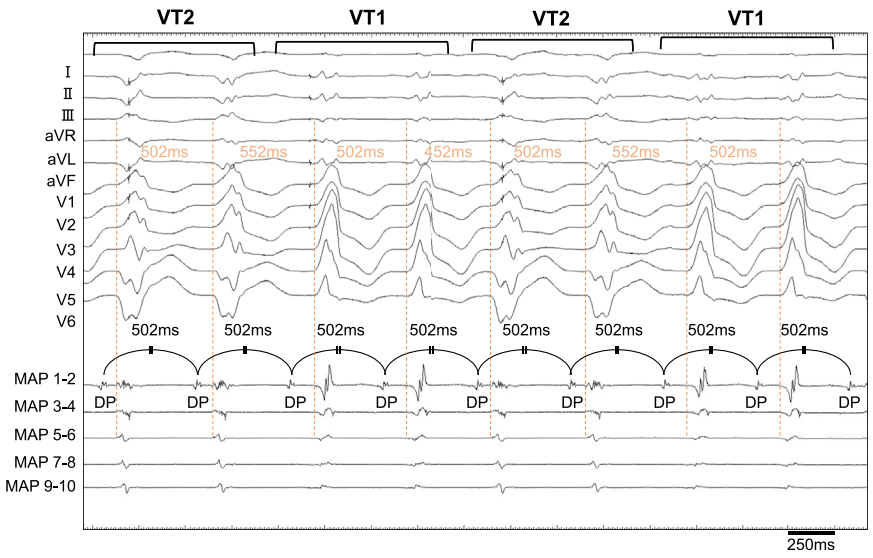

Figure 3 Intracardiac electrogram during ventricular tachycardia (VT) in a low-voltage area. Intracardiac recordings revealed a diastolic potential (DP) with constant cycle lengths, consistently recorded during diastole. The DP-QRS interval differs between VT1 and VT2. MAP = mapping catheter.

LV base. Interestingly, interrupting the circuit usually prolongs the tachycardia circuit and reduces the tachycardia CL; however, in this case, the CL of the DP remained constant. When the same catheter was placed in a nonscarred area of the anterior LV, it showed that the activation of VT2 occurred from lateral to the anterior, contrasting with VT1 (as indicated by the blue arrowhead shown in Supplemental Figure 5).

Based on these findings, the term "intrascar VT,"[1] where all critical isthmuses of the circuits are completed within the low-voltage scar, is strongly indicated. The instability in the propagation of excitation outside the scar area may account for the presence of 2 types of QRS waveforms. In particular, the conduction from the mid-LV scar to the surrounding healthy myocardium was unstable. When conduction was interrupted, it resulted in excitation propagated solely from the basal LV to the healthy myocardium, leading to a VT1 waveform. While there are 2 main sites of excitation propagation from the scar area to the nonscar area, this conduction instability is expected to further complicate the QRS waveform. A schematic of the proposed circuit is shown in Figure 2A.

According to recent reports, most scar-related reentrant VTs have 3-dimensional construction.[3] In this case, the converging main isthmus recorded in Figure 2A (at #) and the DP recorded in the decapolar catheter (Supplemental Figure 4) are considered to be near-field from the endocardium, as well as the magnetic resonance imaging findings of late gadolinium enhancement in the subendocardium (Supplemental Figure 1) and the catheter being located in the center of the scar. However, the potential recorded during the QRS phase may be a border zone in voltage or far-field potential from the midmyocardium or border zone. Nevertheless, since it was relatively difficult to precisely identify the entire essential circuit and determine near-field or far-field potentials, it is not entirely clear whether this VT involved a so-called 3-dimensional circuit.

Speculation on the mechanism of pleomorphic VTs in relation to "intrascar VT"

Pleomorphic VTs exhibit varied manifestations stemming from a shared VT circuit linked to preferred exit sites.[4] In such a circuit, the maintenance of VT typically involves conduction instability owing to factors such as refractory periods, anisotropic conduction, and conduction velocity.[5] The changes within DP could result in alterations to the tachycardia CL. In this case, it was possible to record DPs that occupied some CL using a single catheter, suggesting that conduction instability primarily occurred at the exit phase rather than at the isthmus formation. Given the constancy of DPs, despite pleomorphic QRS morphology, it can be implied that the conduction instability is more likely to occur at the exit phase, within the scar or surrounding border zone, perhaps even more so in this case of "intrascar VT," where the VT circuit is exclusively completed within the scar (Figure 2B and 2C). Hence, "intrascar VT" and pleomorphic VT are speculated to have a strong relationship.

In this case, the potential activation occurred within the diastolic interval, which was not always part of the critical isthmus. Consequently, it is important to identify the critical isthmus by obtaining an overview of the entire circuit, not only the site of the DP recording. Therefore, the diagnosis of "intrascar VT" is crucial because it can avoid the need for extensive scar modification and unnecessary ablation applications. However, it is noteworthy that the definition provided by Wakamatsu and colleagues[1] is based on an entrainment maneuver, and limitations arise when this maneuver is impossible owing to the changing exit patterns and pleomorphic manifestation, as observed in this case. Thus, there could be merit in redefining it when it concerns diastolic occupancy.

Conclusion

The case involves a relatively large scar and unstable conductivity to the surrounding nonscar region. This scenario could illustrate a manifestation of pleomorphic VT, all stemming from a totally shared common isthmus and solely contained within the scar, a condition referred to as "intrascar VT."

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at [https://doi.org/10.1016/j.hrcr.2024.07.007].

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