Recurrent shocks from implantable cardiac defibrillator implanted 6 months ago. What is the mechanism?

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1 | CASE

A 51-year-old gentleman reported thumping sensations in the chest in the morning when he was preparing to go to work. He was diagnosed to have received appropriate shocks for ventricular tachycardia (VT) and was commenced on amiodarone. About 1 week later, the patient was admitted to our Centre for Palpitations. Device check revealed that he received five shocks from his implantable cardiac defibrillator (ICD) in a single morning 1 week ago.

Patient had history of ischemic cardiomyopathy and received a dual chamber ICD (Boston Scientific Inogen) for primary prevention of sudden cardiac death 6 months ago. His baseline 12 lead ECG showed sinus rhythm with narrow QRS complex. ICD check showed stable and normal device parameters. VT zone was programmed 170-199 beats per minute (BPM) with no therapies. Fast VT zone was programmed 200-249 BPM with antitachycardia pacing (ATP) followed by 41J shocks. Ventricular fibrillation (VF) zone was programmed 250 BPM with ATP followed by 41J shocks.

Figures 1 and 2 showed the device tracings of the events. What was the cause of his symptoms and how would you manage the patient?

2 | COMMENTARY

2.1 | Interpreting the intracardiac electrocardiogram

Figure 1A started with sinus tachycardia (ST) of tachycardia cycle length (TCL) 445 ms, followed by two early ventricular electrograms (vEGM) ("1-3" in Figure 1B), then atrial EGM (aEGM) leading to tachycardia initiation. Looking at farfield EGM, the vEGM “1” appeared earlier than expected (by 48 ms) and also had similar morphology to “2” and “3,” suggesting that it is also a ventricular ectopy (VE) or fused beat rather than a conducted vEGM.

During tachycardia (Figure 1B), the far field morphology was similar to the conducted vEGM during sinus rhythm and dissimilar to those of VE, suggesting it was a narrow complex tachycardia. The TCL of this tachycardia fell into the VT zone, hence channel markers labeled each vEGM as “VT.” Atrial cycle length fell into AF zone, hence aEGM was labeled as AF. About eight out of 10 vEGM were in the VT zone, so ICD detected it as VT episode (marker channel labeled as V-Epsd) and started “Attempt 1.”

The atrial and ventricular TCL initially remained stable at 270-278 ms (Figure 1B). Therapy was withheld because tachycardia had 1:1 AV relationship, ventricular TCL was stable, and morphology matched that of SVT as determined by Boston Scientific’s proprietary Rhythm ID match. However, tachycardia gradually accelerated into VF zone, hence some of the vEGM has been relabeled as VF (Figure 2C). Eight out of 10 vEGM fell into VF zone, so morphology was no longer used as a discriminator (hence subsequent vEGM labeled as “RID-”). During tachycardia in VF zone where RID- is in place, it confirmed with six out of 10 vEGM, Figure 2C. ICD confirmed tachycardia as VF (marker channel “V-Detect”) and delivered ventricular overdrive pacing (ATP) via eight burst paced beats (marker channel “VP”) as labeled “3.” We noticed that far field vEGM morphology changed after ATP, but the VA relationship remained unchanged (160 ms), suggesting it is the same tachycardia with morphology change (Figure 2C). The change in farfield vEGM was likely due to aberration as a result of ventricular overdrive pacing during ATP.
In Figure 2C, after delivery of ATP, the first vEGM was ignored (marker channel "- -"), which was the normal response of the Boston Scientific algorithm. It assessed that two out of three vEGM are fast, and so it regarded ATP as failed, hence it continued with attempt 1 therapy, which in this case was high-energy shock (marker channel "Chrg"). In Figure 2D, charge was completed (marker channel "Chrg"). Two out of three confirmation showed tachycardia was still present, so energy was delivered (marker channel "41J Shk" and vertical line across atrial and ventricular channels). The first vEGM was ignored (marker channel "- -"), followed by two out of three vEGM, which were fast, so ICD was classified as failure of shock and attempt 2 of therapy commenced (Figure 2E). Looking closely, however, there was a change in VA relationship and slow down in VV cycle length (CL) for the 4 vEGM ("4- 7") immediately post shock. Post shock, the first return beat ("4") was suggestive of a junctional beat (V and A are on time), followed by 3 VE ("5- 7") before initiation of the tachycardia with the same VA relationship of 160 ms (Figure 2E). This was reminiscent of how this tachycardia initiated! Tachycardia far field morphology had now changed after the first shock and tachycardia had accelerated to 265- 270 ms. However, the VA relationship remained unchanged (160 ms), suggesting it is the same tachycardia with morphology change due to aberration, which was a result of the ICD shock (Figure 2E). The same cycle recurred for the remainder four shocks with the final shock finally resulting in termination of tachycardia after the fifth shock.

2.2 | Applying clinical electrophysiology principles

This tachycardia had the same ventricular morphology as that of sinus rhythm both from far field morphology and by ICD morphology discrimination (Figure 1A and B). During ATP (ventricular overdrive pacing), vEGM was accelerated without resetting the atrial TCL and the tachycardia continued. This demonstrated dissociation of the ventricle from the atrium without termination of tachycardia, proving that the ventricle was not part of the tachycardia. This ruled out VT with 1:1 conduction to the atrium. It also ruled out dual tachycardia (VT and atrial flutter) as it was very improbable to have atrial and ventricular CL to be so tightly coupled. It also ruled out atrioventricular reentry tachycardia (AVRT) as atrium and ventricular will be linked during AVRT. Tachycardia was consistently initiated and reinitiated by VE and by vEGM-aEGM-vEGM (VAV) response during ATP, ruling out atrial flutter (AFL) and AT.

Atypical atrial ventricular nodal reentry tachycardia (AVNRT) was the most likely diagnosis. The VA interval (160 ms) was too long for typical AVNRT. AVNRT is a reentrant tachycardia in which both atrium and ventricle are not part of the circuit, hence it is possible for the ventricle to be dissociated from the atrium during ATP response. Initiation of tachycardia from VE is generally more common than initiation via atrial ectopy (AE) in atypical AVNRT. This is postulated to be due to block in retrograde fast pathway, but there is still conduction retrogradely via slow pathway, then conduct down fast pathway antegradey. This is in contrast with typical AVNRT, which is typically initiated by AE.

2.3 | Interventions

We raised the fast VT zone to 220 BPM so that ICD will withhold therapy should AVNRT recur as the discriminators had worked well until it accelerated into VF zone. We subsequently performed an electrophysiology study (EPS), which confirmed the diagnosis of atypical AVNRT. During EPS, atypical AVNRT could only be initiated via ventricular extrastimuli, reflecting the same initiation we saw in the device tracing. Slow pathway was successfully modified and the patient did not have any further recurrence.
CONCLUSION

Applying electrophysiology principles to device tracings is important to make an accurate diagnosis. In this case, we were able to avoid an unnecessary drug (amiodarone) and help the patient to avoid future inappropriate shocks.

CONFLICT OF INTEREST

Authors declare no conflict of interests for this article.

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