EP CASE REPORT

Successful bailout of refractory ventricular fibrillation originating from the moderator band using bipolar ablation in a patient with short-coupled variant of torsade de pointes

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A 33-year-old woman was transferred to our institute with ventricular fibrillation (VF) after a few beats of torsade de pointes (TdP). The QT interval in sinus rhythm was 360 ms, and the trigger premature ventricular contraction (PVC) had morphologies suggestive of left bundle branch block with coupling intervals of 260 ms. Although the recurrent VF was refractory to intravenous amiodarone, lidocaine, landiolol, and percutaneous stellate ganglion block, intravenous infusion of verapamil suppressed the PVCs. Neither a structural abnormality on echocardiography and cardiac magnetic resonance imaging nor coronary stenosis on computed tomography coronary angiography were present. The patient underwent implantable cardioverter defibrillator implantation due to short-coupled variant of TdP (sc-TdP), and also received oral verapamil. However, an electrical storm of VF recurred 10 months postoperatively. The morphology of the trigger PVC was that of a left bundle branch block with left axis deviation, which was refractory to intravenous verapamil (*Figure 1A*). Catheter ablation targeting the trigger PVC was performed, and the earliest activation was recorded at the lateral aspect of the apex of the right ventricle. Multiple radiofrequency (RF) applications at the earliest activation site decreased the PVC, and neither TdP nor VF occurred at the end of the procedure. However, the VF recurred immediately after the index procedure, and a repeat procedure was performed. The earliest activation site was recorded at the lateral aspect of the right ventricular apex, 32 ms earlier than the QRS onset (*Figure 1B*). The ablation catheter was located at the free-wall insertion of the moderator band on intracardiac echocardiography (ICE, *Figure 1C*). Multiple RF at a maximum of 50 W were applied to the moderator band but failed to suppress the PVC. Epicardial mapping was performed, and this revealed that the opposite site of the endocardial earliest activation site was later than the QRS onset. Bipolar ablation was attempted between the endocardial earliest activation site (4 mm tip irrigation catheter) and the corresponding epicardial site (4 mm tip non-irrigation catheter). Six bipolar RF applications at 25 W were delivered,

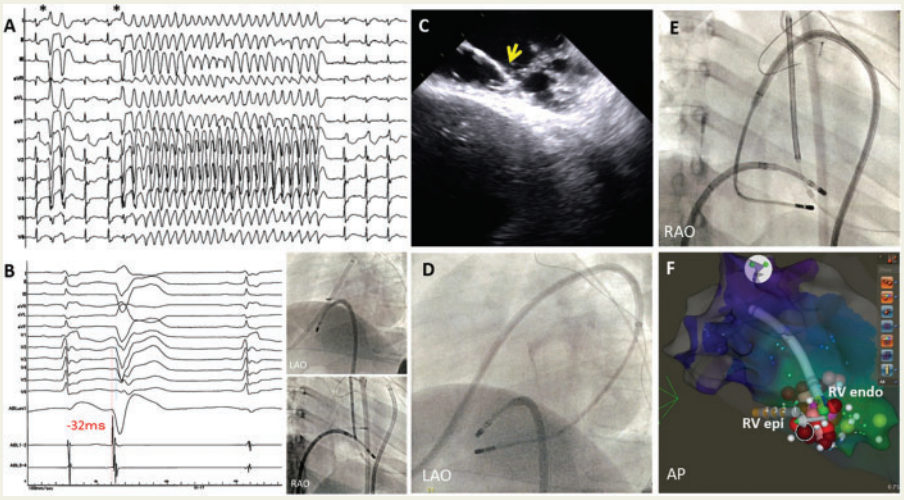

Figure 1 (*A*) Electrocardiogram monitor recording of ventricular fibrillation (VF) after a few beats of torsade de pointes (TdP). Note the identical morphology of the preceding two premature ventricular contractions (PVCs), before the initiation of the TdP (asterisks). (*B*) Intracardiac tracing and fluoroscopic images in left anterior oblique (LAO) and right anterior oblique (RAO) projection during the redo ablation targetting the trigger PVC. The earliest activation was recorded at a lateral aspect of the apex of the right ventricle, which was 32 ms earlier than the QRS onset of the PVC. (*C*) Intracardiac echocardiography showed that the ablation catheter was located on the free-wall insertion of the moderator band, where the earliest activation was recorded. (*D–F*) Fluoroscopic images (*D* and *E*) and the three-dimensional electroanatomical map (*F*) of the bipolar ablation. Note that the 4 mm tip irrigation catheter was located at the endocardial earliest activation site, while the 4 mm tip non-irrigation catheter was located at the corresponding epicardial site.

each for 30 s, while confirming that the impedance drop to within 30 Ω, and this completely eliminated the PVC (*Figure 1D-F*). The patient was free from electrical storms of VF 10 months after the redo procedure.

sc-TdP is a rare form of polymorphic ventricular tachycardia in patients without structural heart disease or prolonged QT interval, that is triggered by PVCs with a remarkably short coupling interval of $\leq$300 ms,[1] and very little is currently known about its pathogenesis. The moderator band can be a source of idiopathic ventricular arrhythmias.[2] Steinfurt *et al.*[3] have recently demonstrated that sc-TdP predominantly originated from the free wall insertion of the moderator band and its Purkinje network, and catheter ablation using three-dimensional mapping and ICE provided favourable long-term outcomes. Our case suggested not only the unique electrophysiological feature of the moderator band as a source of the trigger PVC in sc-TdP but also the challenging nature of the endocardial ablation, possibly due to the intramural origin of the trigger PVC in the moderator band and/or the difficulty in catheter stability and contact on the moderator band, which may have been compensated by the larger lesions created by the bipolar ablation.

Conflict of interest: none declared.

References

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