Double bundle branch reentrant ventricular tachycardia ablation in a patient on ventricular assist device support

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Introduction

Bundle branch reentrant ventricular tachycardia (BBRVT) is characterized by a unique, fast (200–300 beats/min), monomorphic wide complex tachycardia associated with syncope, hemodynamic compromise, and cardiac arrest. BBRVT is a reentrant VT with a well-defined reentry circuit, incorporating the right bundle branch (RB) and left bundle branch (LB) as obligatory limbs of the circuit, connected proximally by the His bundle (HB) and distally by the ventricular septal myocardium. It is often challenging to diagnose, usually requiring a His bundle recording and specific pacing maneuvers.[1] The overall incidence has been reported to be between 3.5% and 6% of VTs in different series, and up to 20% among patients with nonischemic cardiomyopathy undergoing electrophysiologic studies and ablation.[2] Another type of macroreentry in the His/Purkinje system is the interfascicular VT. This type of reentry is extremely rare; when it does occur, it is most commonly seen in patients with coronary artery disease, specifically those with anterior myocardial infarction with some degree of conduction block in the anterior or posterior fascicules. In these patients, right bundle branch block (RBBB) is complete and bidirectional, so true BBRVT cannot occur. Additionally, there is slow conduction in the apparently blocked fascicle. Of note, interfascicular reentrant VT can develop in patients following ablation of the RB for the treatment of BBRVT.[3] Especially high incidence of BBRVT has been reported in patients on ventricular assist device support.[4] Left ventricular assist devices (LVAD) implanted as destination therapy constitute a treatment option in patients with advanced heart disease who are not candidates for heart transplantation. VTs are common in patients with continuous-flow LVAD, especially in patients with a history of VT prior to the implantation of the device, and have been associated with increased morbidity and mortality.[5] In this new clinical scenario, ablation is proposed as a very promising treatment tool. Herein, we report a case of coexisting classical BBRVT and a second VT utilizing the left anterior fascicle (LAF) in a patient with ischemic cardiomyopathy and severe left ventricular dysfunction on ventricular assist device support.

KEY TEACHING POINTS

- Bundle branch reentrant ventricular tachycardia is not uncommon in patients with left ventricular assist devices. Previous anterior myocardial infarction, ventricular enlargement, and conduction disturbances favor this kind of ventricular tachycardia.
- When facing a suspected bundle branch ventricular tachycardia, a careful analysis of cycle length oscillations and the demonstration of spontaneous changes in His-His electrogram intervals preceding changes in ventricular electrogram intervals can help confirm the diagnosis.
- Using multipolar catheters with closely spaced electrodes may help in recording conduction system electrograms, thus allowing improvement in the diagnostic yield of mapping.

Case report

The patient is a 67-year-old man with history of ischemic cardiomyopathy and advanced heart failure with very low (20%) left ventricular ejection fraction. Ten years before admission he had suffered an inferoposterior myocardial infarction treated with stent in the right coronary artery. The

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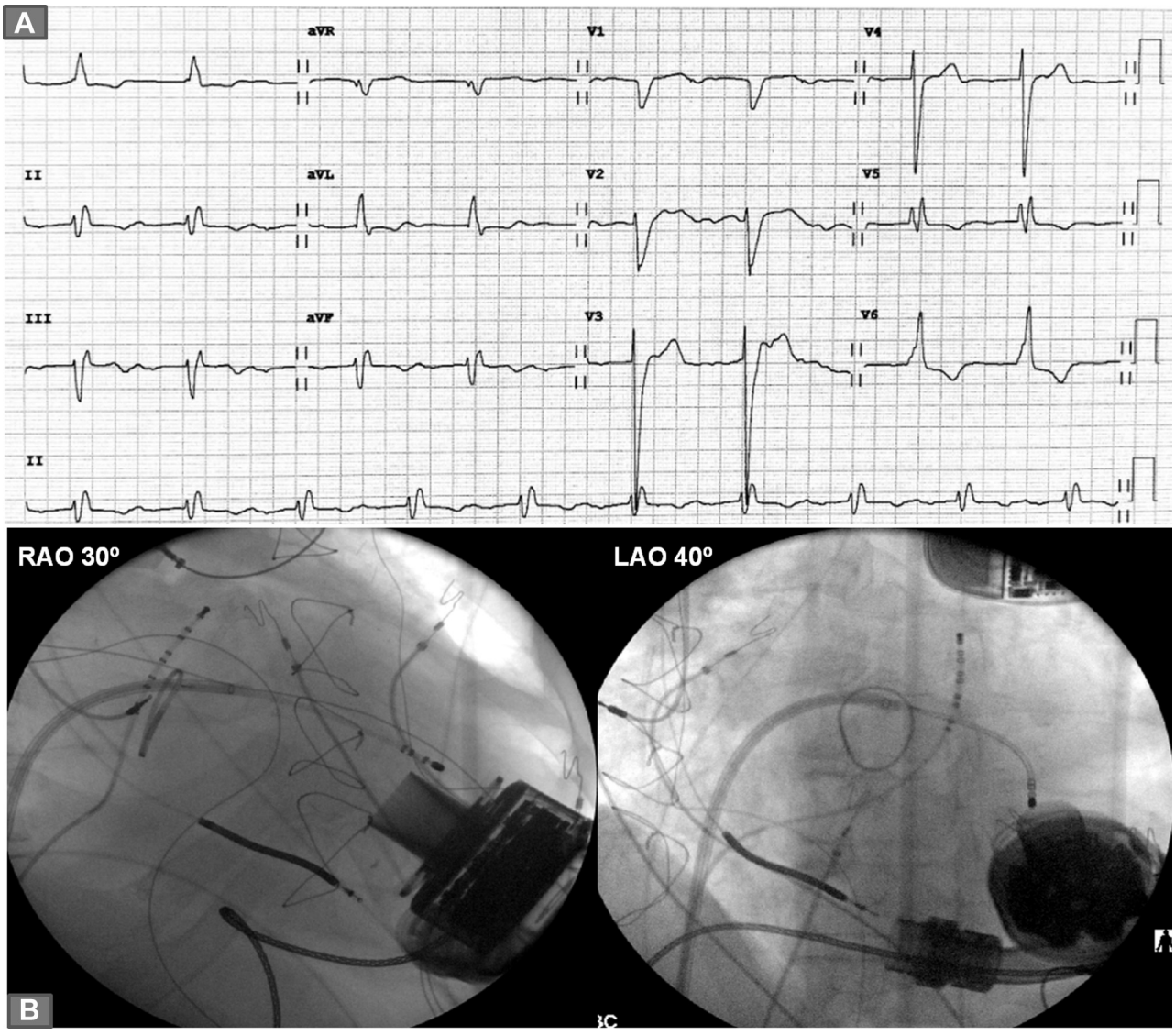

Figure 1 **A:** Twelve-lead electrocardiogram during sinus rhythm before left ventricular assist device implant. **B:** Right anterior oblique (RAO) and left anterior oblique (LAO) projection during ablation.

catheterization also showed total occlusion of the left anterior descending artery (not suitable for bypass) and severe lesion of the circumflex artery. Subsequently a unique bypass on the left circumflex artery was performed. Two years before admission a double-chamber implantable cardioverter-defibrillator was implanted in primary prevention and the patient suffered from VT episodes treated with shock during the follow-up. Owing to progressive and drug-resistant heart failure, the patient was admitted in view of the need for advanced heart failure therapy evaluation. He was not amenable to heart transplant, so it was decided to implant an LVAD as destination therapy (HeartMate 3, Abbott, Chicago, IL). Forty-eight hours after the implant the patient presented multiple sustained VT episodes with heart rate of 140–150 beats per minute (bpm), poorly tolerated as they determined mean arterial pressure decrease, right ventricular (RV) dysfunction, suction phenomena, and low flow of the LVAD. Arrhythmic episodes showed to be resistant to antiarrhythmic drugs (intravenous amiodarone and procainamide) and not responsive to antitachycardia pacing, requiring electrical cardioversion.

Basal electrocardiogram (ECG) showed sinus rhythm with very long PR interval of 400 ms and wide QRS (180 ms) with left bundle branch (LBB)block (Figure 1A). Of note, after the implant of the LVAD, a constant and persistent noise in all but 1 ECG leads was observed, related to the continuous movement of the LVAD pump (Figures 2A and 3A).

The patient presented 2 different and repetitive VT morphologies (Figures 2A and 3A). The first VT (VT1) showed wide complex tachycardia at 140 bpm, LBB morphology, QRS width 180 ms, and QRS morphology almost identical to basal sinus rhythm ECG in both precordial and limb leads. This VT was considered to suggest a classical bundle branch reentry mechanism. The second VT (VT2), at 160 bpm, inferior axis, showed initial positivity in V_1, biphasic in V_3, and mostly positive in V_4, pointing to a left mid-anterior origin, according to recently described criteria.[6]

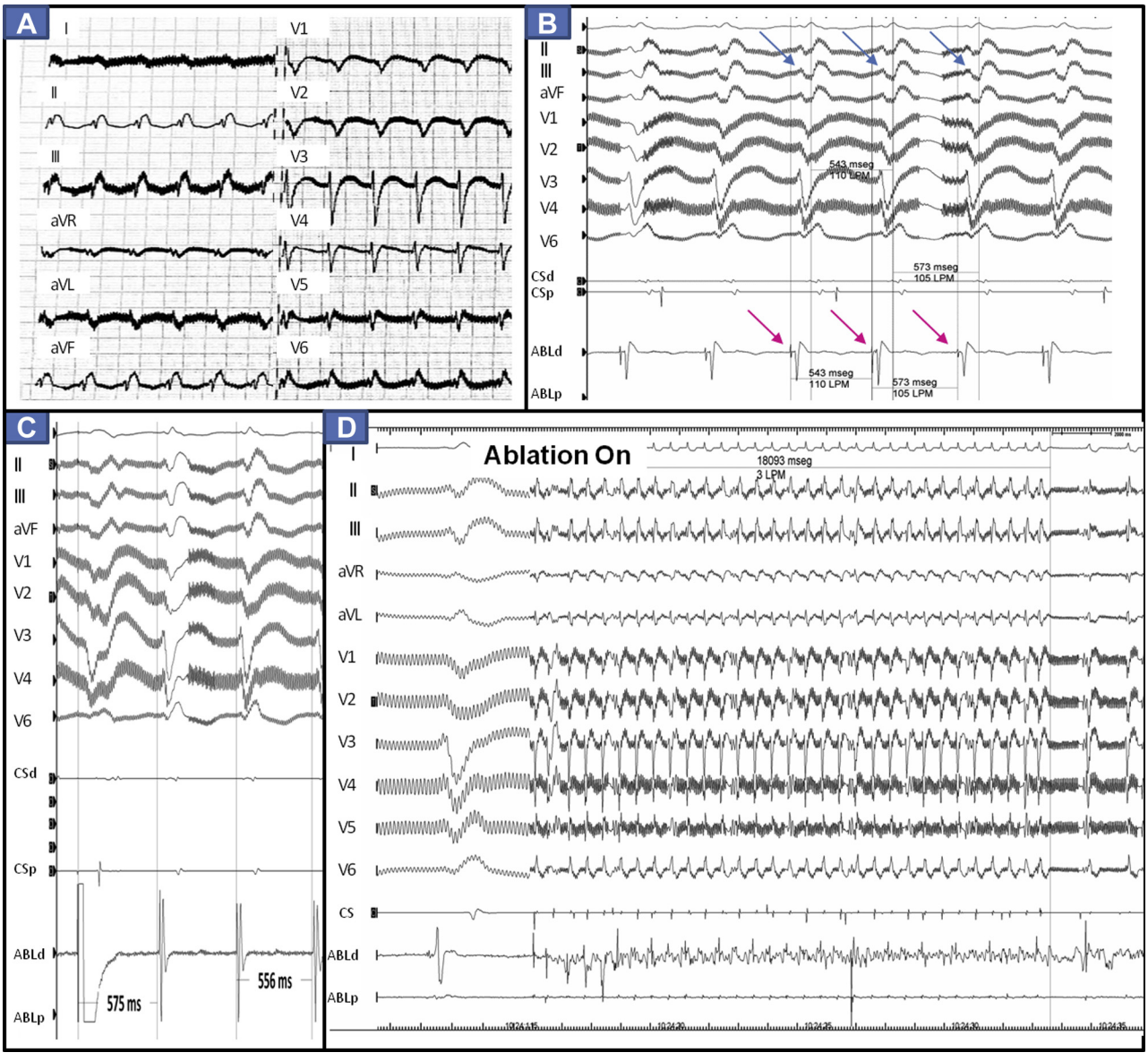

Figure 2 **A:** Twelve-lead electrocardiogram of the first ventricular tachycardia (VT1). **B:** Spontaneous changes in RB-RB potential intervals preceding changes in V-V. **C:** entrainment from the right ventricular apex showing short postpacing interval – tachycardia cycle length. **D:** VT1 ablation by ablation of the right bundle (VT stops with an early beat of the same morphology).

The ablation procedure was performed under conscious sedation. A quadripolar nonsteerable catheter was placed in the RV apex, and a decapolar steerable catheter was placed in the coronary sinus. Endocardial access to the LV cavity was obtained by single transseptal puncture, as the retrograde aortic access in LVAD patients is extremely difficult owing to limited opening of the aortic valve and is also dangerous, in case of thrombus formation on the Valsalva sinuses. The procedure started while on incessant VT (VT2) at 160 ms; the access to the LV cavity was obtained by transseptal puncture guided by transesophageal echocardiography. Subsequently a steerable sheath (Agilis, Abbott) was placed in the left ventricle and a multipolar mapping catheter (HD-Grid, Abbott) was advanced to the zone of interest. Special attention was needed to avoid contact of the catheter tip with the inflow cannula of the LVAD, placed in the apex (Figure 1B). No data about the His-ventricular interval could be obtained owing to incessant VT. During catheter movements VT2 stopped and after few seconds VT1 started. Owing to the suspected RV origin of VT1, the multipoint catheter was moved into the right ventricle and an activation mapping was performed. Of note, we observed presence of the His potential and right bundle potential before every QRS of the VT and we were able to demonstrate that spontaneous changes in H-H interval preceded changes in V-V interval (Figure 2B). Also, there was rapid intrinsicoid deflection in the right precordial leads, suggesting that the initial ventricular activation occurred through the His-Purkinje system. The activation map of the VT showed earliest points in the RV apex area, compatible with classical

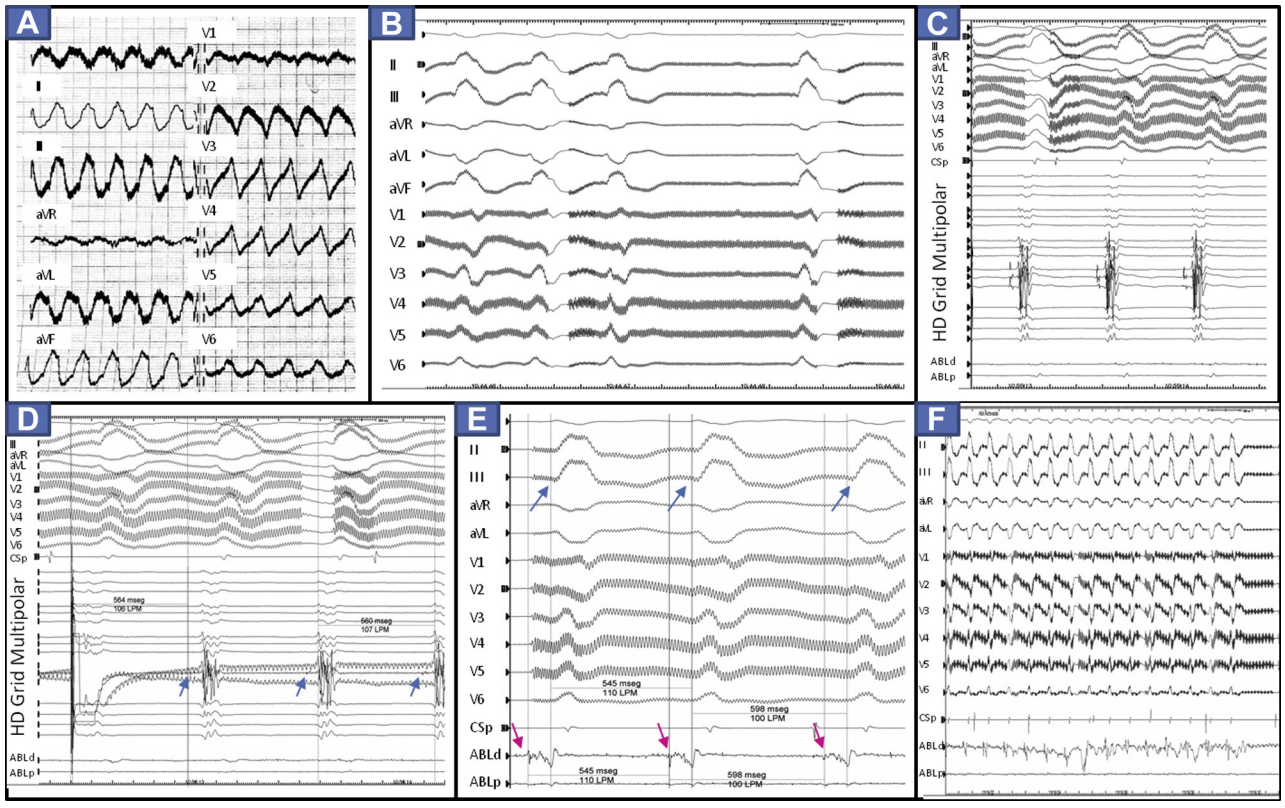

Figure 3 **A:** Twelve-lead electrocardiogram of the second ventricular tachycardia VT2. **B:** Same QRS morphology during VT2 and sinus rhythm. **C:** Left anterior fascicle (LAF) potential during VT. **D:** Postpacing interval – tachycardia cycle length after entrainment from the LAF area. **E:** Spontaneous changes in LAF-LAF potential intervals preceding changes in V-V. **F:** VT2 ablation by ablation of the LAF.

BBRVT. Entrainment from the RV apex showed manifest fusion and short return cycle (Figure 2C). The decision was made to localize and ablate the right bundle branchRadiofrequency lesions were delivered at this location (40 W, 43°C) and the VT stopped after a few seconds, being not reinducible during the rest of the procedure. Soon after the BBRVT ablation VT2 started again, slower than the initial presentation, with cycle length of more than 500 ms. Interestingly, the sinus rhythm QRS after the ablation of the right BBRVT was identical to the VT2 QRS (Figure 3B).

Multipolar mapping during VT2 showed presystolic electrogram in the anteroseptal area, suggesting LAF potential (Figure 3C). Entrainment of the VT by rapid pacing at the LAF area showed occult fusion with a postpacing interval minus tachycardia cycle length of almost zero, suggesting that the LAF area was part of the tachycardia circuit and that the mechanism could be interfascicular VT (Figure 3D). As a confirmation of the LAF involvement, we were able to show that spontaneous changes in the LAF-LAF electrogram intervals preceded changes in the V-V intervals (Figure 3E). Interfascicular VT or an atypical form of fascicular VT utilizing ventricular myocardium as the retrograde limb were both possibilities, as we were not able to demonstrate retrograde activation through the left posterior fascicle. Radiofrequency lesions at the LAF location stopped VT2 immediately.

Subsequently, based on the large anterior, inferior, and inferolateral scar revealed by endocardial voltage mapping, with multiple late potentials and local abnormal ventricular activities, the decision was made to perform substrate modification of the scar. Areas of late potentials were targeted by radiofrequency ablation using a 3.5-mm externally irrigated Tacticath catheter (Abbott) up to a maximum of 45 W and, whenever possible, a contact force of greater than 10 g was achieved. After the ablation of the interfascicular VT the patient presented complete heart block and remained with paced rhythm by the DDD implantable cardioverter-defibrillator. BBRVT was not inducible after the ablation.

Discussion

The case reported here constitutes a unique example of coexisting both classical right BBRVT and a second VT utilizing the left anterior fascicle (atypical fascicular VT or interfascicular VT). Although usually rare, this kind of VT is quite common in patients with LVAD, moreover in cases of previous His-Purkinje system disease and large myocardial scar. The possible origin of VT1 was suggested by the surface ECG and pointed to right BBRVT, while the origin of VT2 was clearer when QRS in sinus rhythm after ablation of the RBB was identical to VT2. The patient

presented a diffuse disease of the conduction system and it was not possible, because of the incessant VT, to locate a more proximal His potential and to be able to demonstrate the typical activation sequence (H-RB-LB)[7]; nevertheless, the spontaneous H-H changes that preceded the V-V interval are very suggestive of this type of tachycardia. Regarding ablation, it has been suggested that ablation of the left bundle in patients with pre-existing LBB block may decrease the incidence of postablation complete heart block,[8] but in this case we preferred the typical ablation approach, targeting the RBB. Owing to the very high heart rate usually reached by these tachycardias and poor hemodynamic tolerance, it is an extremely rare event to be able to perform ablation during tachycardia[7]; therefore the location of the descending branch in both VTs and the demonstration of its participation in the circuit was the reason for applying radiofrequency energy in this location. Indeed, in this case the presence of an LVAD improved the hemodynamic tolerance; even so, RV dysfunction is often observed while on VT, which, especially in the case of high pulmonary pressure, can lead to hypotension and malfunction of the LVAD.[9]

The ablation of ventricular tachycardia in a patient with LVAD presents elements of exceptional complexity, especially those related to access to the left ventricle, the catheter's maneuverability, and the presence of arrhythmic substrates that are usually uncommon; but the good results of ablation of the BBRVT together with the presence of a hemodynamic support device[10] have to support the choice of this type of treatment in selected patients.

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