cardioverter defibrillator was implanted after a first episode of bad tolerated VT. In the past year, he was hospitalized several times for appropriate shocks, including two episodes of electrical storm, leading to RF ablation indication. Predominant VT morphology was well tolerated and incessant (Figure 1D). According to the presence of chronic left apical thrombus, we decided to perform three-dimensional ventricular mapping and RF ablation using intracardiac echography image integration system (CartosoundTM system). This allowed us to clearly individualize apical thrombus in the scar zone, and thus to perform an independent three-dimensional map of it (Figure 1A–C).

During electrophysiological study, many episodes of spontaneous and sustained VT occurred (Figure 1D), allowing an intracardiac mapping of the tachycardia. An emerging point with good entrainment and concealed fusion criteria was then identified (Figure 1E, G, green point).

RF ablation of a border, septoapical zone, of the aneurysm reduced tachycardia, and then non-inducibility confirmed successful VT ablation (Figure 1F, G, green point). RF ablation was performed under live control of catheter position, to avoid manipulation of catheter in the apical thrombus (Figure 1H).

CartosoundTM is a new system of image integration using intracardiac echography (ICE). The use of the specific ICE catheter SoundstarTM allows integration of the ICE images in the CartoTM system, and thus a three-dimensional ‘non-invasive’ reconstruction of the chamber anatomy. Furthermore, soundstar catheter tip contains both a navigation sensor (same as Carto™), and ultrasound phased array probe (same as in the Acunav™ catheter) for visualization of the catheter in the CartoTM system.

Thanks to this ‘non-invasive’ anatomical mapping, we obtained a fast and precise three-dimensional anatomy of the left ventricle, including scar and thrombus (which would be impossible to obtain with ‘conventional’ mapping). This was safe because anatomical mapping did not require to move catheter in the apical thrombus.

In addition, live control of the catheters during RF ablation enhanced security of the procedure, by limiting RF ablation, and catheter manipulation in the apical thrombus.

It is important to note, however, that the technique cannot totally avoid thrombus manipulation during RF ablation phase, because all the catheters and specially the RF catheter tip cannot be fully visualized during all the procedure. In this case, thrombus was present already in 2004, and thus may have been organized, so we hypothesized that the risk of fragmentation was low. We have to emphasize the isolated nature of this procedure, and the potential risk of catheter manipulation in a fresh and/or mobile thrombus.

Conclusion
This case underlines a good indication of Cartosound systemTM in VT ablation. Indeed, even if this system demonstrated safety and efficacy in VT ablation, cost of the procedure, including expensive and single use intracardiac two-dimensional ultrasound catheter (Soundstar™) limits indications. Hence, in some cases where manipulation catheters in the chambers could be dangerous (such as intracardiac thrombus, or left appendage tachycardia for example), use of Cartosound systemTM might be a good solution.

Conflict of interest: None to declare.

Reference

CASE REPORT
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Idiopathic left ventricular tachycardia with dual electrocardiogram morphologies in a single patient

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A 29-year-old female with a documented ventricular tachycardia exhibiting two different electrocardiogram (ECG) morphologies in the precordial leads was referred for catheter ablation. We describe the mechanism of the dual ECG morphologies in this case.
Case report

A 29-year-old female with a documented ventricular tachycardia (VT) exhibiting two different electrocardiogram (ECG) morphologies in the precordial leads was referred for catheter ablation. One of the ECG morphologies exhibited a complete right bundle branch block (CRBBB) pattern (VT1) and the other an incomplete right bundle branch block (ICRBBB) pattern (VT2) (Figure 1A and B, respectively). The patient has been otherwise healthy lacking any structural heart disease. A VT with a QRS morphology identical to that of VT1 was induced by programmed ventricular stimulation. The intracardiac recordings revealed a diastolic potential (P1) and presystolic Purkinje potential (P2) during VT1 and the reversal of those potentials during sinus rhythm at a site on the left ventricular apical-inferior septum (Figure 1D and E, respectively). Pace mapping at that site demonstrated a similar QRS morphology as the clinically documented VT2 (the limb leads were identical to VT2, but the other precordial leads were identical to a fusion complex composed of VT1 and VT2) and that pacing triggered VT1 (Figure 1C). After an ablation targeting that site, no further tachycardias were inducible. On the other hand, VT2 could never be induced throughout the entire procedure.

Discussion

Previous studies reported a bidirectional tachycardia originating in the atrioventricular junction with a tachycardia-dependent right bundle branch block morphology and alternating conduction over the anterior and posterior fascicles of the left bundle. In those reports, the alternating conduction through the left anterior or posterior fascicle resulted in a left- or right-axis deviation in the limb leads during every other beat. On the other hand, in this case, the axes in the limb leads were similar, but the QRS morphology in the precordial leads differed...
between the two VTs, that is, a CRBBB pattern (VT1) and ICRBBB pattern (VT2), respectively. The intracardiac electrograms during VT1 revealed that the His bundle and Purkinje potentials were recorded with a sequence of P1 → P2 → His (Figure 1D). Although the pace mapping did not completely match with VT2 (Figure 1C), the QRS morphology in the precordial leads was more likely a result of fusion beats consisting of VT1 and VT2. Moreover, pacing at that site could trigger VT1, and the fact that the catheter ablation eliminated the two VTs at that site suggests that the two VTs may have used a common pathway as part of the reentrant circuit. As a result, both VTs were no longer inducible after the catheter ablation at that site. In general, the left posterior fascicle (LPF) runs inferiorly from the His bundle and courses along the posterior papillary muscle, and also the RBB runs along the lower portion of the septal band reaching the moderator band and then the anterolateral papillary muscle. We speculated that the reentrant circuits of VT1 and VT2 might have shared a common pathway, and the circuit of VT2 might have involved the LPF and part of the RBB in the interventricular septal wall (Figure 1F).

Conflict of interest: none declared.

References

CASE REPORT

Pseudo-pacemaker syndrome in a young woman with first-degree atrio-ventricular block

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First-degree atrio-ventricular (AV) block is defined as a PR interval longer than 200 ms. If too long, it can become clinically relevant and may mimic a pacemaker syndrome. We report the case of a young woman with a long PR interval, probably congenital, with episodes of syncope and dizziness since childhood. Pseudo-pacemaker syndrome is rare and is a Class IIa recommendation for a pacemaker implantation. A dual-chamber pacemaker was implanted and short AV delay was programmed, with rapid clinical improvement.

Introduction

First-degree atrio-ventricular (AV) block is defined as a delay in transmission of an impulse from the atria to the ventricles resulting in a PR interval longer than 200 ms.1 It can be due to anatomical or functional impairment in the conduction system. It can be observed in healthy children and athletes and is usually asymptomatic. However, in some cases, loss of AV synchrony because of a marked prolongation of the PR interval may cause important haemodynamic alterations, with subsequent decrease in cardiac output and symptoms of heart failure, mimicking a pacemaker syndrome, so-called ‘pseudo-pacemaker syndrome’.

Case

A 40-year-old female consulted the emergency department for nausea, vomiting, and syncope in the setting of gastroenteritis for 2 days. Her clinical history revealed dizziness, palpitations, and several episodes of syncope at rest during childhood. She also mentioned experiencing dyspnoea on exertion for a few years. The physical examination revealed only neck vein distension with intermittent ‘a canon waves’ without other signs of heart failure. Heart rate and blood pressure were normal. The electrocardiogram (ECG) showed a first-degree AV block with a PR interval of 480 ms (Figure 1). The echocardiography, coronary angiogram, and cardiac magnetic resonance imaging were all normal. Auto-immune, endocrinologic, metabolic, and serological investigations were equally normal. The exercise test with Bruce’s protocol was maximal for load (10.2 METs) but submaximal for heart rate (157 bpm, 87% of theoretic maximal heart rate) and was stopped for reason of dyspnoea. Electrocardiogram during stress test showed constant PR interval (Figure 2) with gradual shortening of the RR interval, leading to a certain degree of chronotropic insufficiency. Complete electrophysiological study was not available in our hospital, but we performed rapid atrial stimulation. This revealed a Wenckebach point at 130 bpm and a 2:1 AV block point at 150 bpm. After 1 mg atropine, Wenckebach block appeared

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