

Figure 2. EGM from the atrial and ventricular leads checked during implantation

EGM - electrogram

While confronting a patient in whom the DDD pacemaker interrogation shows nearly 100% ventricular sensing, but electrocardiographic monitoring shows ventricular pacing, the most important probabilities are: a) Dislodgement of the atrial lead into the ventricle and ventricular capture by it; b) Inappropriate connection of the leads to the generator (atrial lead being connected to the ventricular channel of the generator and vice versa). But after ruling out these probabilities, simply by a chest X-ray and pacemaker analysis, the only remaining explanation is that the ventricle is already paced by the ventricular safety pacing mechanism and the generator considers it as V-sensing rather than V-pacing.

Conclusion

In patients with total ventricular sensing in the pacemaker interrogation, ensuring the proper connection of the leads and ruling out the lead dislodgement is necessary. In case both conditions were ruled out, ventricular safety pacing can be the rational diagnosis.

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Catheter ablation of electrical storm triggered by monomorphic ventricular ectopic beats after myocardial infarction

Miyokart enfarktüsü sonrası gelişen monomorfik ventriküler erken atımların tetiklediği elektriksel fırtınanın kateter ablasyonu

Introduction

Electrical storm is a life threatening situation that involves recurrent episodes of ventricular arrhythmias. It is defined as 3 or more sustained episodes of ventricular tachycardia (VT), ventricular fibrillation (VF) or appropriate implantable cardioverter-defibrillator shocks during 24 hours (1). We report a patient who had drug-refractory, repetitive polymorphic VTs after myocardial infarction (MI) which could only be managed by radiofrequency ablation (RF) ablation of triggering ventricular premature beats (VPCs).

Case Report

A 62-year-old female patient admitted to hospital with inferior MI after 12 hours of symptom onset. Patient immediately underwent successful primary percutaneous intervention. Echocardiogram revealed left ventricular ejection fraction of 40% with segmental wall motion abnormality.

Four days after the revascularization, she suddenly developed recurrent and sustained polymorphic VTs triggered by monomorphic VPCs (Fig. 1). There was no electrolyte imbalance and no recurrent ischemic event. Coronary angiography was also repeated but no significant lesion was observed. She was not taking any QT prolonging medication and QT interval was normal. Combination therapy of amiodarone and metoprolol was ineffective to suppress arrhythmias. Patient was deeply sedated and mechanically ventilated. Overdrive pacing and intra-aortic balloon pump counter pulsation were also tried to stop electrical storm. Despite all these interventions several electrical cardioversions were required (21 times in last 24 hours). Therefore, patient was transferred to electrophysiology laboratory to attempt catheter ablation of the VPCs triggering the polymorphic VTs.

Left ventricle (LV) was accessed retrogradely across the aortic valve (7.5 F Navistar D curve irrigated tip catheter, Biosense Webster).

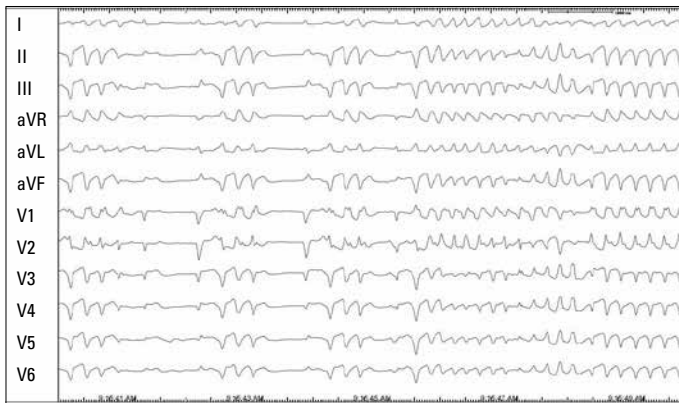


Figure 1. Twelve-lead surface ECG of ventricular premature contractions initiating polymorphic ventricular tachycardia
ECG - electrocardiogram

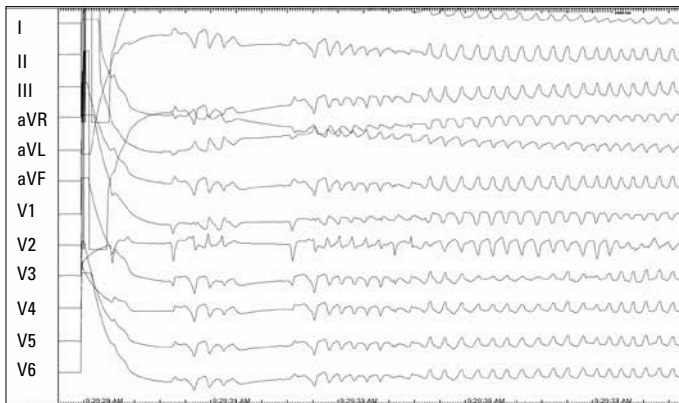


Figure 2. Some of the ventricular tachycardia episodes were being reinitiated by the same ventricular premature contractions shortly after DC shocks
DC - direct current

Electro anatomical mapping system (Carto 3, Biosense Webster, Diamond Bar, Ca, USA) was used to create LV map. Initially rapid activation map of presumed location of VPCs was created. VT episodes were so frequent that some of them were being reinitiated by the same VPCs shortly after DC shocks (Fig. 2). Although it was not detailed, activation mapping revealed the possible origin of VPCs at inferior septum near apex (Fig. 3). At these sites, low amplitude and high frequency Purkinje like potentials preceding VPCs were observed (Fig. 4). But they were not constant and hard to target for ablation. Pace mapping was also applied to localize the origin of VPCs. Areas with earliest activation and similar paced QRS morphology were identified as a target for RF ablation. After 5 RF applications VPCs disappeared and electrical storm stopped. Programmed ventricular stimulation with three extra stimuli failed to induce any tachyarrhythmia and the patient was brought to intensive care unit. Antiarrhythmic drug therapy continued. Patient had septicemia during follow up and had 2 VF episodes during febrile spells, which were not triggered by a VPC. On the 10th day of administration she had an ischemic stroke. Unfortunately, we lost her due to septic shock on the 22nd day of administration.

Discussion

Persistent electrical storm after acute myocardial infarction (MI) is very dramatic condition and sometimes can only be managed by radio-frequency (RF) ablation. The Heart Rhythm Society and the European Heart Rhythm Association support early ablation of recurrent VT (2).

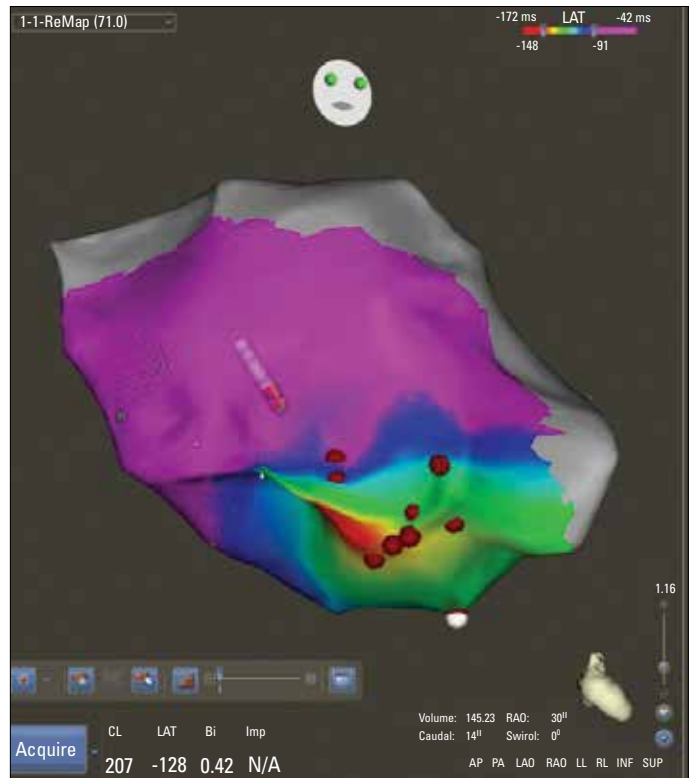


Figure 3. Electroanatomic activation map (right anterior oblique view) during ventricular premature contractions. The earliest activation region was at inferior septum near apex of the left ventricle and it was close to successful ablation site

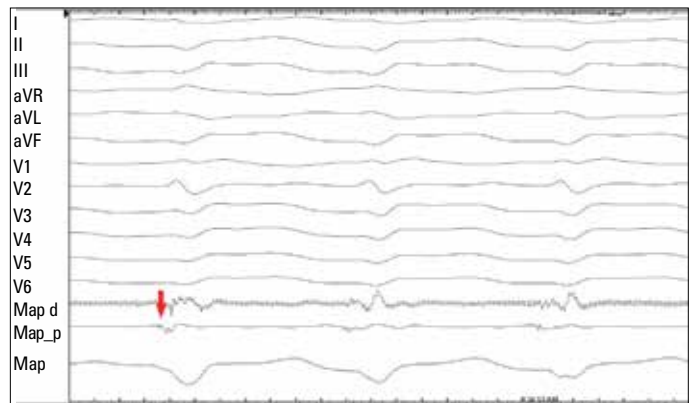


Figure 4. Mapping at the site of earliest activity during ventricular tachycardia. Note that the low amplitude and high frequency Purkinje like potentials (arrow) precedes the ventricular signals
Map - ablation catheter

Ablation technique is depending on the mechanism of arrhythmia. Most monomorphic VTs in the presence of ischemic heart disease are due to electrical wave front reentry around a scar tissue. These mechanisms allow identifying critical isthmuses and ablation (3, 4). In contrast, the mechanisms responsible for polymorphic VT are poorly understood. Ventricular premature contractions (VPCs) originating from Purkinje system has been shown to be associated with polymorphic VTs and electrical storms after MI (5-7).

Emerging evidence in patients with polymorphic VT has identified that the Purkinje arborization is a dominant source of triggers initiating arrhythmias (8). Purkinje fibers are more resistant to ischemia

than myocardial cells and endocardial fibers may be nourished from cavity blood (9). These surviving Purkinje fibers in infarct region demonstrate enhanced automaticity and triggered activity which may cause polymorphic VT when coupled with prolonged action potential duration (10). In current studies, most of the VPCs originating from Purkinje network were located in the border-zone of MI (5-7). These studies has shown that ablation of these triggers was able to eliminate arrhythmias. Similar results were also demonstrated for patients early after MI (6).

However, it is not always easy to find and abolish Purkinje potentials during electrical storm. For instance, we were not able to localize Purkinje potentials constantly because of repetitive hemodynamically unstable VTs. During the procedure we observed Purkinje like potentials where the earliest endocardial activation regions of VPCs were. After successful RF applications we didn't observe these signals. Our report is result of a single case and more studies are needed to elucidate the mechanisms of polymorphic VT after MI. Unfortunately, follow-up period was too short due to concomitant diseases and this is an obvious limitation of our report.

Conclusion

Catheter ablation plays increasingly important role in management of electrical storm after MI. RF ablation is indicated in recurrent polymorphic VT or VF when specific triggers can be targeted (2). In these cases accelerated ablation approach may help reaching to a safe harbor.

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Successful ablation of cavo-tricuspid isthmus dependent atrial flutter in a patient with Senning operation

Senning operasyonlu hastada kavo-triküspit istmus bağımlı atriyal flutterin başarılı ablasyonu

Introduction

Atrial flutter is a common complication late after atrial switch operation for transposition of the great arteries. It is usually cavotricuspid isthmus (CTI) dependent (1). Radiofrequency catheter ablation (RFCA) targeting CTI region may eliminate flutter (2). Access to targets for ablation may be limited by anatomy and by surgically placed obstacles. We report a case in which bidirectional CTI block achieved under computed tomography (CT) and electro-anatomic mapping (EAM) system guidance terminated the tachycardia.

Case Report

A 16-year old boy with a history of surgical palliation of d-transposition of the great arteries, a normal systolic ejection fraction, and symptomatic drug refractory atrial flutter was referred for an electrophysiological study and ablation procedure. At the age of 3 months he had undergone a Senning (atrial switch) operation. A baffle was surgically constructed within atria for directing systemic venous blood across the mitral valve into the left ventricle (systemic venous ventricle) and