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CASE REPORT

Arrhythmogenic Substrate Ablation in a Patient With Brugada Syndrome and Electrical Storm

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Abstract

Despite being a rare condition, Brugada syndrome (BrS) poses a significant risk of arrhythmic death, and managing associated ventricular arrhythmias is particularly challenging. Here, we present a case where percutaneous ablation targeted an arrhythmogenic substrate in a patient with BrS and recurrent ventricular fibrillation (VF). Epicardial mapping revealed abnormal potential areas in the right ventricle's outflow tract and free wall. Injection of warmed saline into the pericardial space caused further delay in local electrograms and expanded the abnormal potential area. Radiofrequency applications in this region successfully eliminated abnormal electrograms, leading to the disappearance of the coved-shape ST-segment elevation in leads V1-V2. No recurrence of ventricular arrhythmias was reported in a twelve-month follow-up.

Introduction

Brugada syndrome (BrS) is a hereditary disease belonging to the family of cardiac channelopathies that increases the risk of serious ventricular arrhythmias or even sudden death in patients without identified structural heart disease.[1] Recent research indicates that BrS is linked with interstitial fibrosis and decreased expression of intercellular gap junctions on the epicardial surface of the right ventricular outflow tract (RVOT).[2] Percutaneous catheter ablation to modify this epicardial substrate is emerging as a potentially effective intervention for cases of drug-refractory ventricular tachycardia/ventricular fibrillation (VT/VF). In Brazil, this approach is even more crucial due to the limited availability of the drug of choice, quinidine.[3] However, despite its promise, there remains a scarcity of data on this subject.[4]

Keywords

Catheter Ablation; Brugada Syndrome; Epicardial Mapping; Ventricular Tachycardia.

Case description

This case study describes epicardial radiofrequency catheter ablation of an arrhythmogenic substrate in a 54-year-old man with spontaneous BrS type I (Figure 1) who experienced recurrent VT and syncope. Multiple appropriate therapies were triggered by the implantable cardioverter defibrillator (ICD) (30 shocks in six months with two hospitalizations due to an electrical storm).

The procedure was conducted under general anesthesia with invasive blood pressure monitoring. Access to the epicardium was achieved via an anterior subxiphoid puncture using a 17 G-Tuohy needle, following established techniques.[5] High-density multipolar mapping of the epicardial surface was performed using a PENTARRAY® mapping catheter (Biosense Webster, Dmond Bar, CA, USA) coupled with voltage mapping (CARTO®; Biosense Webster). Due to the unavailability of sodium channel blockers (ajmaline, flecainide, procainamide) to enhance phenotypic expression and improve mapping accuracy by increasing fractionated potentials, we opted to infuse 50 ml of warm saline solution (39-40 ºC) into the pericardial space.[6] Specific reports have shown that this technique slows conduction in diseased areas and accelerates it in healthy ones, thus helping to identify areas of interest. Areas of late, fractionated, and prolonged potentials were noted on the epicardial substrate map and were shown to be confined to the free wall and RVOT. The infusion of warm saline extended the areas of late potentials to lower areas of the epicardial region of the RV[7] (Figure 2). Endocardial mapping

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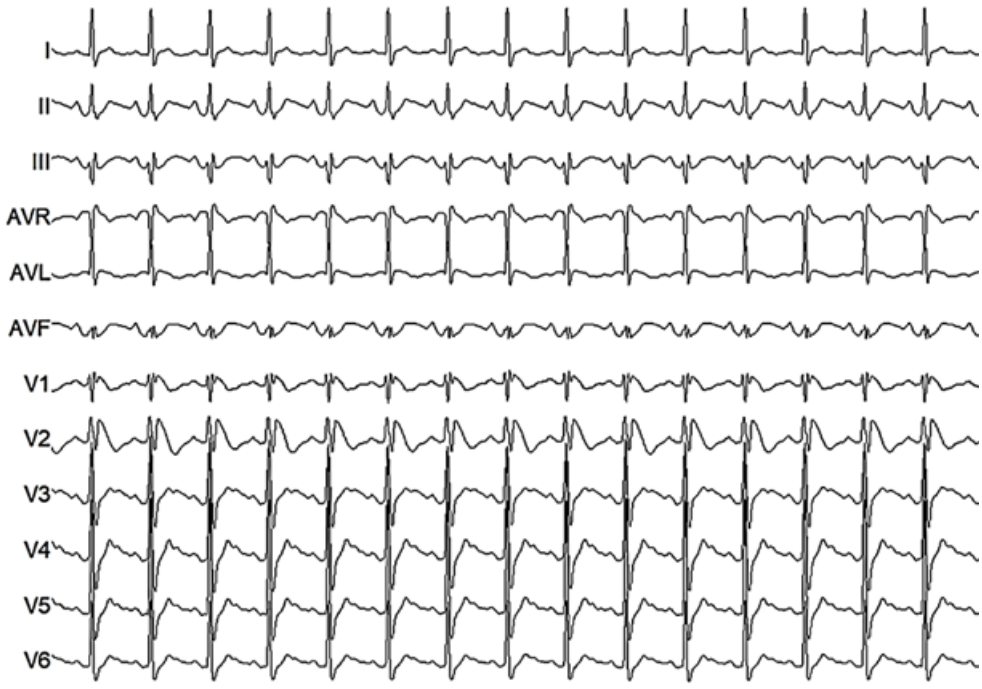

Figure 1 – 12-lead ECG (standard positions) showing Brugada type I pattern (coved-shape) in right precordial leads (V1-2).

did not demonstrate abnormal potentials. Coronary angiography confirmed a safe distance from the main coronary arteries. We conducted radiofrequency ablation (30-40W; 10-20g; 10-20s) over a broad area encompassing the epicardial free wall and RVOT (Figure 3), aiming to eliminate identified late potentials, leading to the resolution of the coved-shaped ST-segment elevation in leads V1–V2 (Figure 4). Immediately following ablation, the remaining electrocardiographic pattern showed alterations in ventricular repolarization that did not meet the criteria for a classic Brugada type II (saddleback) pattern.

After 40 days, the electrocardiogram (ECG) returned to a normal appearance, suggesting that some of the immediate changes might have been due to pericardial inflammation resulting from the extensive treatment area. This normalized ECG persisted throughout all subsequent outpatient evaluations over the following 12-month follow-up (Figure 5). During this period, there were no documented instances of ventricular arrhythmias, ICD shocks, or syncope.

Conclusion

The modification of the arrhythmogenic substrate within the epicardial region of the RVOT to treat recurrent ventricular arrhythmias in patients with type I BrS remains underutilized, yet evidence supporting its effectiveness is increasing. While the optimal mapping and ablation technique is still debated, targeting the elimination of late potentials appears to be a promising strategy with durable outcomes. Infusion of warmed saline solution expanded the area of potential abnormalities beyond the RVOT, thus partially mitigating the unavailability of intravenous sodium channel blockers.

Author Contributions

Conception and design of the research, acquisition of data and analysis and interpretation of the data: Ponte Filho AD, Tavora RV; writing of the manuscript and critical revision of the manuscript for intellectual content: Ponte Filho AD, Tavora RV, Costa IP, Rezende AGS, Santos Neto FR.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

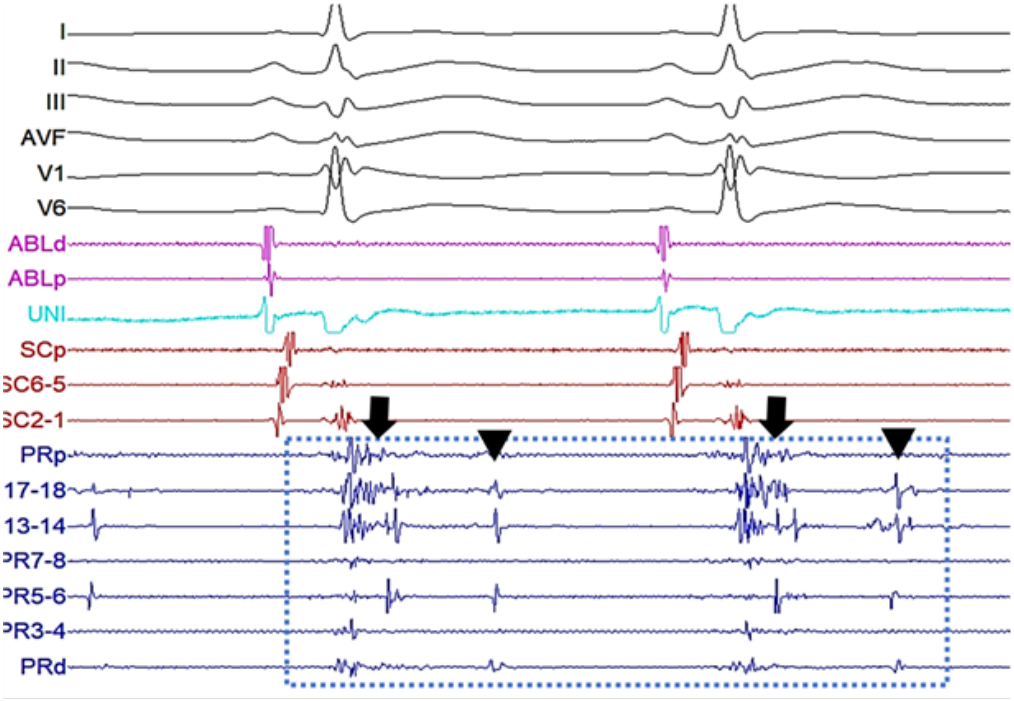

Figure 2 – Recording showing electrocardiographic leads + intracavitary electrograms from the coronary sinus (proximal, mid, and distal) and from the epicardial region of the right ventricle, recorded using a high-density catheter (PENTARRAY®; Biosense Webster, Diamond Bar, CA, USA). Highlighted (dashed box) are the fragmented potentials (arrows) and late potentials (arrowheads). Recording speed: 150 mm/s.
SCp: proximal coronary sinus; SC6-5: medial coronary sinus; SC2-1: distal coronary sinus; PR: pentarray; PRp: proximal pentarray; PRd: distal pentarray.

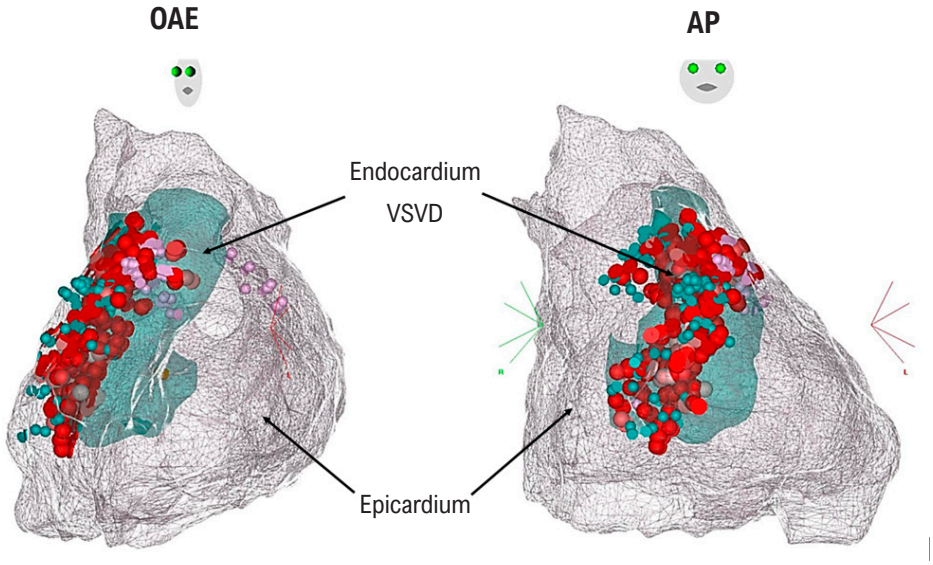

Figure 3 – Electroanatomic mapping of the epicardial region (gray) and endocardial region of the right ventricle (RV) (green). The points indicate the locations where radiofrequency ablation was applied in the epicardial region, extending from the outflow tract to the free wall of the RV, and correspond to the locations where late potentials were detected.
VSVD: RV outflow tract; OAE: left anterior oblique; AP: anteroposterior.

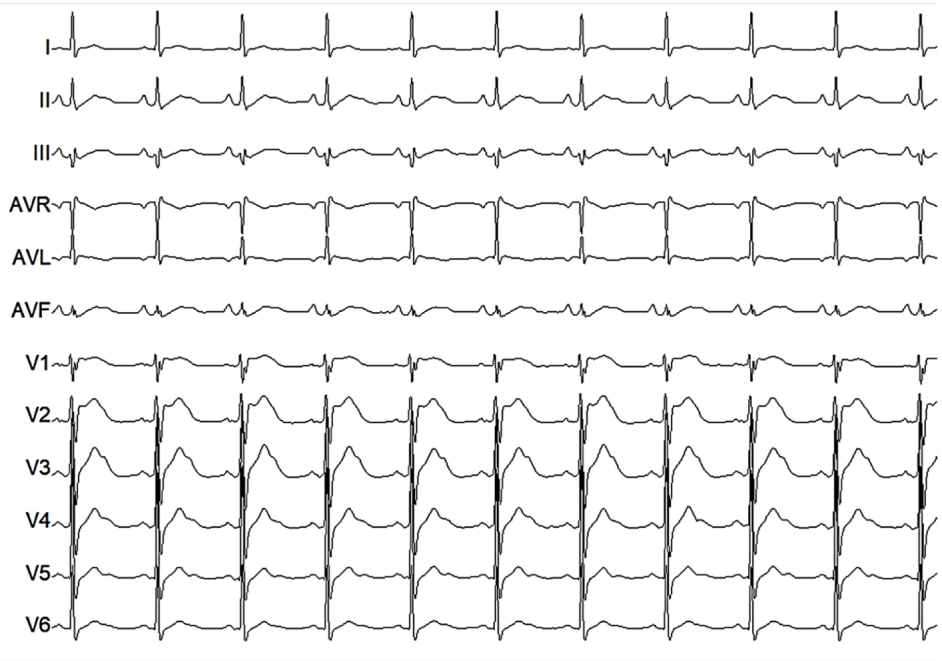

Figure 4 – ECG immediately after epicardial ablation. The "coved-shape" pattern in the precordial leads has disappeared. The remaining changes do not resemble the classic Brugada type II pattern ("saddleback") either.

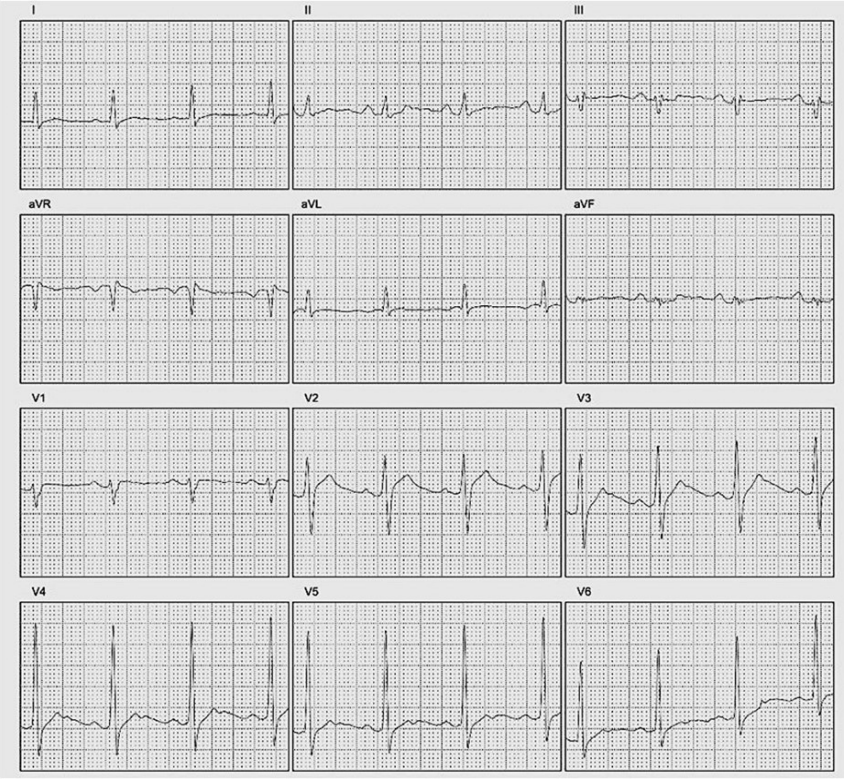

Figure 5 - 12-lead ECG performed 6 months after the ablation. Absence of the "coved-shape" pattern in precordial leads.

Sources of Funding

There were no external funding sources for this study.

Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Huppor under the protocol number 001/2024. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

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