Roll of Mapping and Radiofrequency Catheter Ablation in Brugada syndrome

By Andrés Ricardo Pérez-Riera MD PhD

Until recently, the management of patients who have survived sudden cardiac death (SCD) has focused on treating the consequences by an implantable cardioverter-defibrillator (ICD). However, such therapy remains restricted in many countries, is associated with a prohibitive cost to the community, High instrumental cost and the potentially large number of candidates will significantly impact hospital budgets (1) and may be a cause of significant morbidity in patients with frequent episodes of electrical storms (ES). Recurrent ventricular tachycardia/Ventricular fibrillation (VT and/or VF) followed by frequent ICD shocks might thus put patients in a painful.

Evidence emerging from the study of fibrillation both in the atria and the ventricle suggests an important role for triggers arising from the Purkinje network or the RVOT in the initiation of VF. Initial experience in patients with idiopathic ventricular fibrillation (IVF) and even those with VF associated with LQTS BrS and IVF suggests that long term suppression of recurrent VF may be feasible by the elimination of these triggers. With the development of new mapping and ablation technologies, and greater physician experience, RFCA of VF, with the ultimate aim of curing such patients at risks of SCD, are not an unrealistic approach (2;3).

Haissaguerre et al (4), localized by mapping the earliest endocardial activity and by focal RFCA of PTV/VF in tree patients with BrS. The authors conclude that triggers from the Purkinje arborization or the RVOT have a crucial role in initiating VF associated with BrS and LQTS. These can be eliminated by focal RFCA.

In a highly symptomatic 18-year-old-male with BrS, frequent episodes of VF, fast PVT, and fast S-MVT were observed. The episodes were classified as VT or VF and as a consequence received appropriate therapies with the ICD. Precipitating VPBs that were stored in the ICD memory and on the ECG exhibited the same morphology as frequent isolated VPBs. During the electrophysiological (EPS) study, right and left atrial tachycardia with one-to-one atrioventricular conduction were induced and successfully ablated. VF was ablated using the same non contact mapping (NCM) system-triggering VPBs from RVOT (5).

Yu e col; presented a case of recurrent syncope diagnosed as recurrent VF by an implanted loop recorder (ILR). The VF was eliminated by RFCA of triggering VPCs (6).

Lim et al reported a case of epicardial RFCA in a combined BrS and inferior early repolarization syndrome. Patient with recurrent defibrillator therapy for spontaneous VF. Electroanatomic

mapping and RFCA were achieved with remote magnetic navigation. Highly fractionated electrograms were seen epicardial in the anterior RVOT and at the anterior-inferior right ventricle. RFCA of the RVOT region resulted in resolution Brugada type 1 ECG pattern. The inferior early repolarization persisted despite RFCA of the inferior right ventricular epicardium. The patient remained event free at 12-months follow-up (7).

Epicardial substrate modification appears to be more effective than endocardial-only approach in preventing VT/VF. Persistent or recurrent J-ST elevation appears to represent a marker of failure of RFCA. This approach seems to be an acceptable strategy for patients with BrS and VT/VF. Epicardial substrate modification appears to be more effective than endocardial-only approach in preventing VT/VF. Persistent or recurrent J-ST elevation appears to represent a marker of failure of RFCA (8).

The early attempts at catheter ablation to treat BrS patients were limited to a few reported cases of patients with electrical storms. The former approach was designed to target the initiating PVCs that trigger VF with RFA at the endocardial site of the RVOT (9). However, this approach did not prove successful in all cases, because BrS patients rarely have PVCs frequently enough to allow mapping and to provide a precise target for ablation. The first epicardial RFA of the RVOT was followed by pVT/VF inducibility in 22% cases, while BrS pattern disappeared after procedure in 3 above 9 patients at follow-up (10). A recent report evaluated 14 BrS patients with ICD for targeting fragmented and delayed potentials and low voltage areas in the basal condition and after flecainide test. In a short time, data reported 100% pattern disappearance under basal conditions and after drug challenge, no pVT/ VF inducibility, and no more episodes (11). Although larger studies with longer follow-up are required, these results provide new insights into the EPS mechanisms of BrS; however, areas of uncertainty regarding the clinical outcome after embellishment of the ventricular repolarization have been already observed in the history of cardiac electrophysiology, when digoxin administration, which shortens the QT segment (12) was not associated with better outcome in LQTS patients (13). Moreover, whether the elimination of the phenotypic pattern of the BrS will result in less arrhythmic events during follow-up remains to be established (14). Finally, it should be considered that in patients in whom BrS is associated with the early repolarization pattern/syndrome, selective ablation of the anterior RV epicardium (including the RVOT) is not ameliorative (15).

Shelke et al studied 5 patients (4 males, age-23 to 32 years) with BrS and electrical storm (ES) despite being on isoprenaline infusion and cilostazol underwent 3 dimensional electroanatomic mapping and RFA. VF was easily inducible in two patients. Voltage map of RV was created in sinus rhythm in all patients. Substrate modification of RVOT was performed endocardially in one patient, both endocardial and epicardial in three and only epicardially in one. Brugada pattern gradually resolved over one week in all patients post procedure. These patients completed follow up of median 40 months. One patient had inappropriate shock due to AF, one had an episode of VF and appropriate shock 24 months after the RFCA. The remaining 4 patients had no device therapy or ventricular arrhytmias in device log on follow up. Abnormal myocardial substrate is observed in RVOT among patients with BrS. Substrate modification in these patients may abolishType 1 ECG Brugada pattern and prevents spontaneous VAs on long term follow up (16)

Following. HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes: document endorsed by HRS, EHRA, and APHRS Catheter ablation may be considered (Class IIb) in patients with diagnosis of BrS and history of arrhythmic storms or repeated appropriate ICD shocks additionally also following this consensus. BrS is diagnosed in patients with ST-segment elevation with type 1 morphology ≥2 mm in ≥1 lead among the right precordial leads V1, V2, positioned in the 2nd, 3rd or 4th intercostal space occurring either spontaneously or after provocative drug test with intravenous administration of Class I antiarrhythmic drugs. 2. BrS is diagnosed in patients with type 2 or type 3 ST-segment elevation in ≥1 lead among the right precordial leads V1, V2 positioned in the 2nd, 3rd or 4th intercostal space when a provocative drug test with intravenous administration of Class I antiarrhythmic drugs induces a type I ECG morphology (17). Implantation of a cardioverter defibrillator (ICD) is first-line therapy for BrS patients presenting with prior cardiac arrest or documented VT. A pharmacological approach to therapy is recommended in cases of electrical storm, as an adjunct to ICD and as preventative therapy. The goal of pharmacological therapy is to produce an inward shift to counter the genetically-induced outward shift of ion channel current flowing during the early phases of the ventricular epicardial action potential. This is accomplished by augmentation of I_{Ca}using b-adrenergic agents or phosphodiesterase III inhibitors or via inhibition of I_{to}. RFCA of the RVOT epicardium is effective in suppressing arrhythmogenesis in BrS patients experiencing frequent appropriate ICD-shocks. Understanding of the pathophysiology and approach to therapy of BrS has advanced considerably in recent years, but there remains an urgent need for development of cardio-selective and ion-channel-specific I_{to}blockers for treatment of BrS(18).

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