REVIEW

Recent Data on Epicardial Ablation of Ventricular Tachycardia in Nonischemic Heart Disease

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Epicardial ablation has been adopted during the last years, mainly as a supplementary technique after a failed endocardial ablation procedure, both in patients with ischemic and nonischemic ventricular tachycardias (VTs). Sosa and colleagues were the first who described the percutaneous subxiphoidal puncture to approach the epicardial space in 1996.[1] Using the 3D electroanatomic mapping systems, the endocardial and epicardial substrate mapping have become feasible during the same procedure. Because of its complexity and its potential risks this process is performed only in high experienced centers by skilled operators with a large number of VT ablation procedures.

Endocardial ablation in patients with left ventricular nonischemic cardiomyopathy (NICM) has shown worst outcome compared with ablation in ischemic cardiomyopathy. The main reason seems to be the progressive nature of the disease and the presence of epicardial and intramural slow conduction areas forming reentry circuits. The pattern of fibrosis and scar in NICM is not predictable as in ischemic cardiomyopathy where it follows the distribution of the coronary artery disease. Data from the study of Hsia et al[2] in patients with NICM and VT episodes supported that the critical endocardial low voltage substrate was located mainly in the basal and perivalvular area. In this study, epicardial mapping was performed only in few patients revealing abnormal fragmented potentials but data from later studies highlighted the important of epicardial substrate in NICM.[2] Cano et al[3] performed both endocardial and epicardial mapping in 22 patients either because of failed endocardial ablation or because of electrocardiographic signs suggesting epicardial localization of the exit point. Electroanatomic mapping revealed extended epicardial low voltage areas in the majority of the patients (about 82%) which were located mostly in basal left ventricular lateral wall. However, Haqqani et al[4] described 31 of 266 patients with NICM (11.6%) who had septal involvement, mainly in the basal region, without lateral low voltage areas.

Identification of epicardial substrate

Electrocardiographic criteria identifying VT epicardial origin

During the last years several electrocardiographic criteria have been proposed for the diagnosis of an epicardial VT. The most important study was published from Berruezo et al [6] suggesting the following criteria for left ventricular epicardial origin: pseudodelta wave ≥34 milliseconds, intrinsicoid deflection in V2 ≥85 milliseconds and an R/S complex duration ≥121 milliseconds. These parameters were derived after epicardial pacing mainly in postinfarction patients and their specificity and sensitivity was found that was affected from the left ventricular paced region. A later published paper suggested that from these parameters only the intrinsicoid deflection ≥85 milliseconds was consistent with the diagnosis of epicardial VT in patients with NICM.[6] A specific algorithm for the NICM epicardial VTs was published in 2010 by Valles and colleagues[7] suggesting as diagnostic criteria the absence of a q wave in the inferior leads, a pseudo delta wave ≥75 milliseconds, a maximum deflection index (defined as the time from the beginning of the QRS to the earliest maximal deflection in any of the precordial leads divided by the maximal QRS duration >0.59 and the presence of a q wave in lead I (Figure 1). Moreover, recently was reported that even during sinus rhythm the presence of R wave ≥0.15 mV in V1, S wave ≥ 0.15 mV in V6 and/or a S/R ratio >0.20 mV in V6 reveal epicardial origin of VT in patients with NICM.[8]

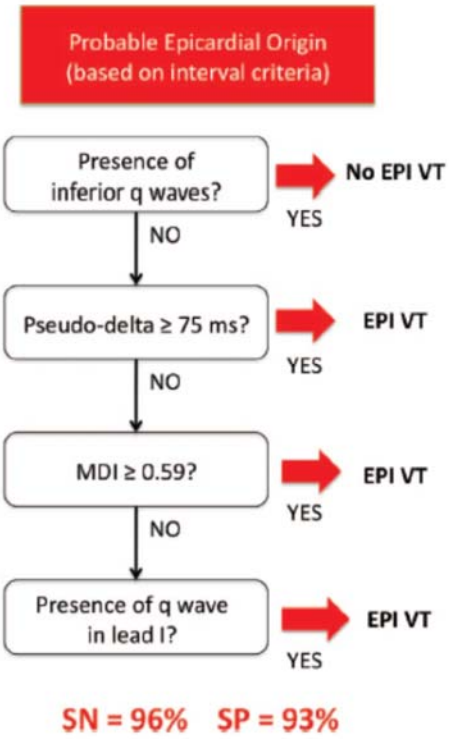

Figure 1. Four-step algorithm for the identification of an epicardial VT in NICM as proposed by Valles et al[7]. MDI: maximum deflection index, SN: sensitivity, SP: specificity

Electroanatomic mapping

In patients with structural heart disease remarkable bipolar voltage attenuation is usually recorder in areas characterized as fibrotic or scar. Patients with NICM presenting with VT commonly have fragmented endocardial electrograms (EGMs) and late potentials, suggesting reentry circuit areas with nonuniform and delayed electrical activation. Substrate mapping during VT ablation includes commonly the combination of voltage and pace mapping, especially in patients with well tolerated stable VTs. Bipolar EGMs amplitude lower than 1.5 mV is indicative of scar although the sensitivity of this cut-off threshold is not high.[9] The role of unipolar endocardial recordings in the identification of epicardial scar in patients with NICM seems to be significant, as it has proved that a value ≤8.27 mV in endocardial surface predicts a corresponding epicardial scar.[10] In patients with arrhythmogenic right ventricular cardiomyopathy/ dysplasia (ARVC/D), this cut-off value for unipolar EGMs for the assessment of epicardial involvement has been proposed to be <5.5 mV.[11]

There are many barriers in the epicardial voltage mapping process and subsequent ablation coming mainly from the poor contact between the catheter tip and the tissue due to the possible existence of pericardial adhesions and/or epicardial fat. Furthermore, the epicardial scar is often found adjacent to the epicardial coronary arteries or left phrenic nerve which crosses along the left ventricular free wall, thus requiring particular attention to avoid potential complications.

Magnetic resonance imaging (MRI)

The first study that demonstrated a correlation between the extent of transmural scar and the arrhythmogenic risk in patients with NICM was published from Nazarian et al.[13] Patients with 25-75% extent of scar in MRI predicted inducible VT in the electrophysiology study. Bogun et al[14] demonstrated that the "delayed enhanced MR imaging" technique could partly identify the location of scar, endocardial or epicardial, in pre-ablation MRI to identify the potential successful ablation sites.

Clinical outcomes

The efficacy of ablation in patients with NICM has significantly improved with the introduction of epicardial ablation additionally to the endocardial approach. The incidence of the abnormal fragmented and low voltage epicardial and endocardial electrograms has been reported to be higher in patients with inducible VTs compared to those without inducible VTs.[12] Sojema et al[15] performed epicardial ablation in 29% of the total patients after the failure of the endocardial procedure and they found that in these patients the scar area war larger in the epicardium compared to endocardium. After a follow up of one year the survival free of VT was 54%. Similarly, the study of Cano et al[3] showed greater epicardial scar in conjunction with wider and more fragmented EGMs. During a follow up of 18±7 months 78% of the patients who underwent epicardial ablation were free of arrhythmia suggesting high percentage of abnormal epicardial tissue. Schmidt et al[16] performed epicardial ablation in 15 patients with NICM who had undergone a failed endocardial procedure and VT was not inducible in 75% of them.

Combined endocardial and epicardial ablation provides better outcomes in patients with NICM and recurrent VT episodes and ICD shocks. The lateral basal free wall seems to be the most common area with low voltage and slow conduction properties, facilitating the reentry mechanism. Performing these operations requires special attention and experience in order to eliminate the arrhythmogenic burden and to avoid possible complications.

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IMAGES IN CARDIOLOGY

Echocardiographic Findings in Carcinoid Syndrome

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A 57-year-old Caucasian female was referred to our clinic due to hypertension, flushing, and diarrhea. She had a medical history of hypertension and paroxysmal atrial fibrillation. On physical examination, the patient had a heart rate of 70 bpm and a respiratory rate of 12 breaths/min. Her temperature was 37°C and her blood pressure was 120/80 mmHg. Cardiac examination revealed a left parasternal holosystolic murmur, and a palpable right ventricular heave. Lung auscultation was unremarkable. From the initial biochemical exam she had no specific abnormalities. The ECG showed sinus rhythm, negative T-waves in leads III, $V_{1\text{-}5}$. The transthoracic echocardiography study revealed a left ventricle with normal size and normal systolic function and dilatation of the left atrium, whereas the right cardiac chambers were dilated with thickened, immobile leaflets of the tricuspid and pulmonic valve, leading to malcoaptation and severe tricuspid and pulmonic regurgitation (Fig. 1-5). The clinical and echocardiographic findings raised the suspicion of carcinoid heart disease. Abdominal computed tomography (CT) demonstrated hepatic metastases and the patient was treated with chemotherapy and with the somatostatin analog octreotide.

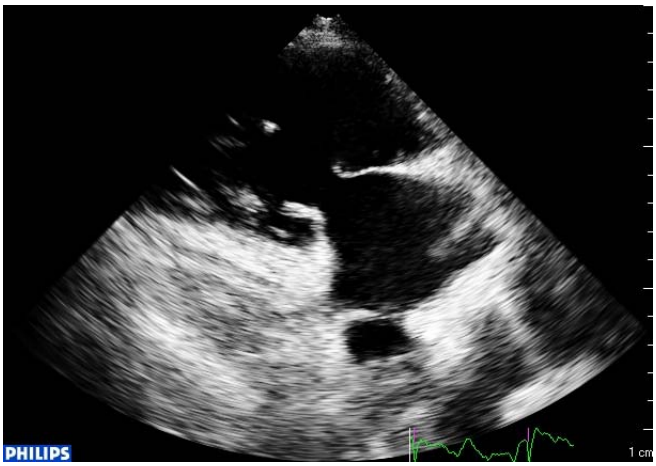

Figure 1. Right ventricular inflow view in systole showing thickened, immobile and retracted anterior and septal leaflets of tricuspid valve**.**

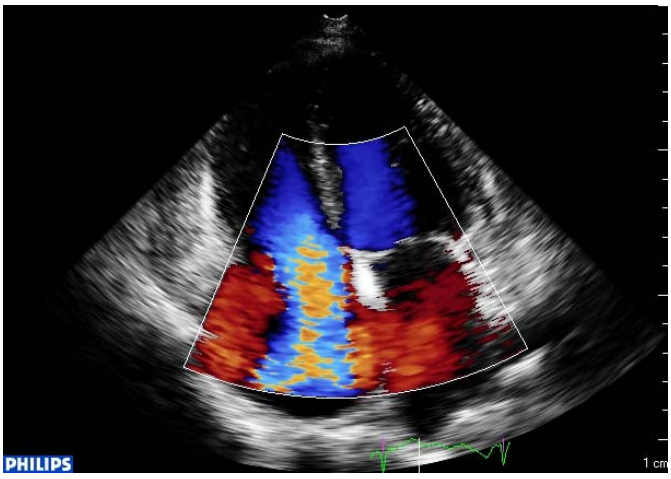

Figure 2. Apical four-chamber view: color Doppler demonstrates severe tricuspid valve regurgitation.

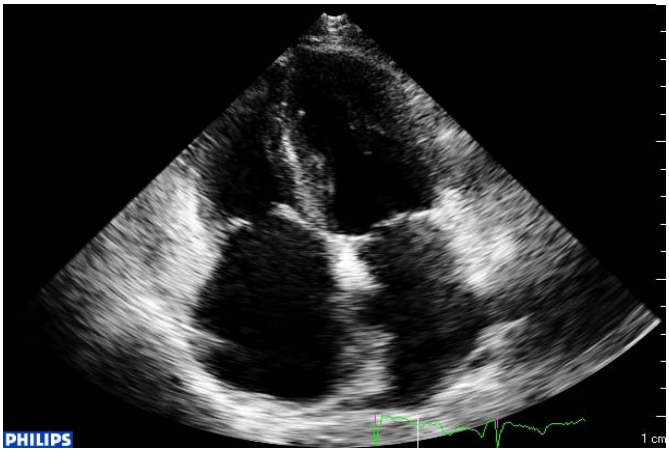

Figure 3. Apical 4-chamber view in systole: opened and retracted tricuspid valve (left); mitral valve is closed (right)