

FOCAL HYPERMETABOLIC LEFT VENTRICULAR CARDIOMYOPATHY: AN UNDERDIAGNOSED LIFE-THREATENING ARRHYTHMOGENIC DISEASE

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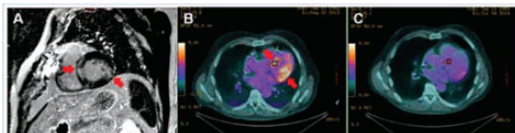
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Purpose of the Study: Ventricular arrhythmias (VA), atypical flutter and/or conduction disorders may be life-threatening manifestations of Cardiac Sarcoidosis (CS), whose etiologic assessment represents a challenge encompassing biological and histological findings as well as imaging biomarkers. Considering the frequent virus-negativity, autoreactive immunopathogenesis besides CS may cause a progressive arrhythmogenic cardiac fibrosis whose clinical presentation remains poorly defined.

Methods: We report a series of 21 consecutive pts (57 ± 13.4 y, n = 4 females) addressed at our tertiary university center because of VA, atypical flutter and/or conduction disorders, who underwent a comprehensive diagnostic work-up including coronary angiography (CA) and Cardiac Magnetic Resonance (CMR) in those without an ICD. 18F-FDG PET scan (PET) was performed in pts with normal CA and evidence for delayed enhancement (DE) within the left ventricle (LV) at CMR. Pts displaying focal hypermetabolic activity at PET underwent directed cardiac biopsies and an extensive blood testing searching for autoimmunity and viral serologies.

Summary: DE within the LV was observed in 18 pts with normal CA and no contraindication for CMR. A DE within the interventricular septum suggestive of CS was reported in 11 (52%) pts only. A PET, performed in 21 pts, revealed a hypermetabolic LV activity in 20 pts (95%) of whom four (19%) displayed a focal hypermetabolic right ventricle activity as well. The figure below illustrates an example of septal and lateral DE at CMR (A) and focal hypermetabolic LV activity at PET (B). Laboratory tests were not contributive except for three (14%) positive Tuberculosis spots indicating latent infection. Directed myocardial biopsies, performed in 19 pts, were abnormal in 14 (74%) cases consisting in two (14%) CS only and two (14%) lymphocyte myocarditis. The 10 remaining pts showed fibrosis. Despite the lack of criteria fulfilling the diagnosis of CS, immunosuppressive treatment has been introduced in 14 (67%) pts up to now, resulting in complete suppression of focal hypermetabolic activity in nine (64%) pts and a partial reduction in the remaining five (36%) ones (Figure C).

Conclusions: A multi-modality assessment based on CMR and FDG-PET allowed the detection of a new focal hypermetabolic syndrome sharing several features with CS including a favorable response to immunosuppressive treatment. In contrast to CS, there are no biological markers indicative of systemic inflammation.



Conflict of interest: none

STRICTER CRITERIA FOR LEFT BUNDLE BRANCH BLOCK DIAGNOSIS DO NOT IMPROVE PATIENT SELECTION FOR CRT

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Purpose of the study: Cardiac resynchronization therapy (CRT) was proved to be effective in patients with heart failure and left bundle branch block (LBBB). Recently, new ECG criteria have been proposed for the diagnosis of LBBB. These criteria are stricter than the current American Heart Association (AHA) criteria and thus increase the specificity of LBBB diagnosis. We assessed the rate of echocardiographic response to CRT in patients who did and did not meet new criteria (Strict-LBBB).

Method used: Consecutive patients who received CRT defibrillators were enrolled in the CRT MORE registry. Patients with no-LBBB QRS morphology according to AHA, atrial fibrillation, right bundle branch block and right ventricular pacing were excluded from the analysis. Strict-LBBB was defined as: QRS ≥ 140 ms for men and ≥ 130 ms for women, QS or rS in V1-V2, mid-QRS notching or slurring in ≥ 2 contiguous leads. Patients showing a relative decrease of $\geq 15\%$ in left ventricular end systolic volume (LVESV) at 12 months were defined as responders.

Summary of results: Among 335 patients with AHA LBBB, 131 (39%) had Strict-LBBB. Patients with and without Strict-LBBB showed comparable baseline characteristics except for QRS duration (166 ± 20 ms vs 152 ± 25 ms, $p < 0.001$). At 12-month evaluation responders were 205 (61%), 85 (65%) patients had Strict-LBBB and 120 (59%) had no Strict-LBBB ($p = 0.267$). On multivariate analysis, history of atrial fibrillation, larger LVESV, and presence of mid-QRS notching in ≥ 1 lead (OR 1.96; 95%CI 1.04 to 3.70, $p = 0.038$) were independently associated with the echocardiographic response.

Conclusion: Recently proposed stricter criteria for LBBB diagnosis did not improve the identification of CRT responders. Among ECG variables, only the presence of mid-QRS notching in ≥ 1 lead was associated with the echocardiographic response.

Conflict of interest: none

VECTORCARDIOGRAPHIC QRS AREA IDENTIFIES DELAYED LEFT VENTRICULAR LATERAL WALL ACTIVATION DETERMINED BY ELECTROANATOMIC MAPPING IN CANDIDATES FOR CARDIAC RESYNCHRONIZATION THERAPY

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Introduction: Delayed left ventricular lateral wall (LVLW) activation is considered the electrical substrate underlying LV dysfunction amenable to cardiac resynchronization therapy (CRT). The purpose of this study was to assess LVLW activation in CRT candidates using coronary venous electroanatomic mapping (EAM) and to investigate whether the QRS area (QRSAREA) on the vectorcardiogram (VCG) can identify delayed LVLW activation.

Methods: 51 consecutive CRT candidates (29 LBBB, 15 IVCD, 7 RBBB) underwent intraprocedural coronary venous EAM using EnSite NavX. VCGs were constructed from preprocedural digital 12-lead ECGs using the Kors method. QRSAREA was assessed and compared to QRS duration and 5 different LBBB definitions.

Results: Delayed LVLW activation (activation time $> 75\%$ of QRS duration, example in figure) occurred in 38 of 51 patients (29/29 LBBB, 8/15 IVCD, 1/7 RBBB). QRSAREA was larger in patients with than in patients without delayed LVLW activation ($108 \pm 42 \mu\text{Vs}$ vs $51 \pm 27 \mu\text{Vs}$, $P < .001$), and identified delayed LVLW activation better than QRS duration (area under the curve 0.89 [95% confidence interval 0.79-0.99] vs 0.49 [95% confidence interval 0.33-0.65]). QRSAREA $> 69 \mu\text{Vs}$ diagnosed delayed LVLW activation with a higher sum of sensitivity (87%) and specificity (92%) than any of the LBBB definitions. Of the different LBBB definitions, the European Society of Cardiology textbook definition performed best with sensitivity of 76% and specificity of 100%.

Conclusion: Coronary venous EAM can be used during CRT implantation to determine the presence of delayed LVLW activation. QRSAREA is a noninvasive alternative for intracardiac measurements of electrical activation, which identifies delayed LVLW activation better than QRS duration and LBBB morphology.

Conflict of interest: none

THE IMPACT OF LEFT VENTRICULAR SCAR ON THE ACUTE HEMODYNAMIC IMPROVEMENT WITH MULTISITE LEFT VENTRICULAR PACING

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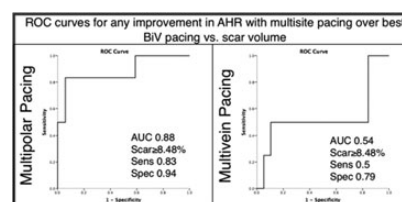
Purpose: Multisite biventricular (BiV) pacing may offer additional acute hemodynamic benefits to traditional BiV pacing either through multivein pacing (MVP) or multipolar pacing (MPP) with a multielectrode left ventricular (LV) lead within one vein.

Our objective was to test whether the presence, amount and distribution of scar impacts the degree of acute hemodynamic response (AHR) with multisite pacing.

Method: Multi-center study, where patients underwent an acute hemodynamic study as part of their BiV procedure. LV dp/dtmax was measured to determine % change from baseline pacing (AAI) compared to 'optimal' BiV pacing. MPP and MVP in the same patients. All patients had late gadolinium enhanced CMR imaging to assess for scar burden and distribution.

Summary of Results: Twenty-four LBBB patients completed the study (83% male; QRS width 171 ± 20 ms; 38%/62% NYHA class II/III; and 58% ischemic). Scar volume was $6.0 \pm 7.0\%$ with 6/24 patients having a scar volume $> 8.48\%$. MPP and MVP displayed similar AHR when compared to best BiV site (BBV). There was a statistically significant correlation between the difference in AHR (MPP-BBV) versus scar volume ($R = 0.49$, $p = 0.02$), however the mean AHR benefit gained was only $3.9 \pm 5.3\%$ for MPP in those that improved. There was no correlation between MVP AHR improvement over BBV and scar volume ($R = 0.111$, $p = 0.62$). Scar volume of 8.48% predicted an improvement in AHR with MPP (sensitivity 83% specificity 94%); this was not the case with MVP pacing (figure 1). The presence of the multielectrode lead in scar also predicted MPP AHR improvement ($p = 0.04$). The presence of either MV lead in scar did not predict MVP AHR improvement.

Conclusion: Greater scar volume and the distribution of scar at the pacing site may increase the likelihood of AHR improvement with MPP but not MVP over BBV.



Conflict of interest: none