Reduced right ventricular outflow tract strain at cardiac magnetic resonance correlates with low-voltage areas at unipolar electroanatomic mapping in patients with brugada syndrome

M. Ciabatti1, C. Zocchi1, M. Pieroni1, P. Notarstefano1, V. Tavanti2, M. Nesti1, N. Sistl1, C. Fumagalli2, L. Malatesti2, F. Raimondi3, G. Pedrizzetti4, R.H. Chan5, I. Olivotto3, L. Bolognese1

1San Donato Hospital of Arezzo, Cardiovascular Department, Arezzo, Italy
2San Donato Hospital of Arezzo, Radiology Department, Arezzo, Italy
3Careggi University Hospital, Cardiomyopathy Unit, Florence, Italy
4University of Trieste, Department of Engineering and Architecture, Trieste, Italy
5Peter Munk Cardiac Centre, Toronto General Hospital, University Health Network, Toronto, Canada

Funding Acknowledgements: None.

Background: Brugada Syndrome (BrS) was initially described as a channelopathy without underlying structural substrate, but pathological and electroanatomic abnormalities of the right ventricular outflow tract (RVOT), are increasingly recognized, suggesting a possible role of electroanatomic mapping (EAM) in risk stratification of these patients. New echocardiographic and cardiac magnetic resonance (CMR) tools are able to identify subtle structural and functional myocardial abnormalities associated with an increased arrhythmic risk.

Purpose: We aimed to assess the presence of structural and functional abnormalities of RVOT by CMR-derived RV and RVOT feature tracking in BrS patients. We also sought to evaluate the relationship between RVOT functional abnormalities and the presence and extension of RVOT abnormal voltage areas at EAM.

Methods: we retrospectively enrolled BrS patients submitted to CMR comprehensive of RVOT sequences and bipolar and unipolar endocardial EAM. Abnormal voltage areas were defined by the presence of signal amplitude <4mV at unipolar mapping and signal amplitude <1.5mV at bipolar mapping. We included a control group of 15 healthy volunteers submitted to CMR including RVOT sequences.

Results: We studied 16 patients, 12/16 males, mean age 42±9 years. In all cases CMR showed normal left and right ventricular ejection fraction (LVEF 57±4%, RVEF 56±6%). RVOT peak strain values were significantly impaired in BrS patients compared to controls (-17.9±8.6% vs -26.4±9.5%, p=0.011), while RV-GLS did not differ between the two groups (-22.2±4.0% vs 24.6±2.5%, p=0.061). EAM showed the presence of pathological voltages at both unipolar and bipolar mapping in 12 patients, abnormal unipolar and normal bipolar maps in 1 case and both unipolar and bipolar normal maps in 3 patients. Median extent of abnormal voltage areas was 5.3 cm² [IQ 1-4 = 0.6-9.3 cm²] at unipolar and 4.1 cm² [IQ 1-4 = 0.7-7.5 cm²] at bipolar map. RVOT peak strain showed a significant correlation with abnormal voltage areas at unipolar EAM (r=0.546, r²=0.525, p=0.029). Patients with both unipolar and bipolar normal maps (n=3) had normal RVOT strain values compared to controls (-25.7±3.9 vs -26.4±9.5%, p=0.90).

Conclusions: In patients with BrS, RVOT peak strain is significantly reduced compared to controls and is associated with abnormal voltage areas at endocardial unipolar EAM. Noninvasive evaluation of BrS patients with CMR-derived RVOT feature tracking may help to identify patients requiring invasive evaluation with EAM. Prospective studies evaluating the prognostic role of RVOT strain and EAM in BrS patients are needed.
Relation between RVOT feature tracking and extent of pathological areas on unipolar electroanatomic mapping

\[ v = 74.59 + 0.41x + 0.4x^2 + 5.56 \times 10^{-3}x^3 \]

\[ R^2 \text{Cubic} = 0.525 \]
A BrS patient with preserved RVOT-FT values (A) and normal unipolar voltage electroanatomic map (B).
A BrS patient with reduced RVOT-FT (C) values and RVOT low-voltage area at unipolar voltage electroanatomic map (D).