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New insights into risk stratification of Brugada syndrome from high density epicardial electroanatomic mapping

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Background/Introduction: Brugada syndrome (BrS) has been initially described as a channelopathy with no structural heart disease but a growing body of evidence points towards structural anomalies in the epicardium of right ventricle outflow tract (RVOT-EPI). Since its first description the electrical substrate of BrS has been thought to be a fractionation of the electrograms (EGMs) in the RVOT-EPI. Also there is recent evidence that SCN5A mutation BrS patients might be at increased risk of sudden cardiac death (SCD). No studies correlated high-density mapping substrate with clinical outcomes.

Purpose: The aim of the current study was to compare clinical characteristics of BrS patients with EGMs characteristics from high-density epicardial mapping with Advisor HD-Grid mapping catheter.

Methods: All consecutive patients with BrS who underwent hybrid RVOT-EPI substrate ablation at our University Hospital between April 2018 and March 2021 were retrospectively included in the study. Genetic analysis was performed in all patients and clinical data were collected. BrS was diagnosed following current recommendations. Inclusion criteria were: 1) use of Advisor HD-Grid mapping catheter and 3D-electroanatomical (EAM) mapping system EnSite Precision; 2) acquisition of EAM before and after ajmaline infusion at standard protocol (1 mg/kg in 5 minutes). Bipolar EGMs were considered abnormal (aEGMs) if they met at least 1 of the following: prolonged duration (>80 ms), fragmented components (at least 2

distinct peaks), and/or late potentials extending beyond the end of the QRS complex. aEGMs duration (Ed) was defined as the time from surface QRS onset in D2 to bipolar EGM offset.

Results: Fifteen patients were included in the study. Six patients (40%) had history of SCD or inducible ventricular arrhythmias (VAs) at electrophysiological study and 5 patients (33.3%) had a mutation of SCN5A. The mean number of points of EAMs was 1020 ± 500 . Patients with history of SCD or VAs compared with patients without history had similar Ed before ajmaline [117.5 ms (100.6–132.5) vs 110.5 ms (106.8–114.8), $p=0.72$] but longer Ed after ajmaline [247.5 ms (231.6–273.9) vs 188.0 ms (178.0, 211.5), $p=0.034$]. The best cutoff of Ed after ajmaline to predict VAs history was 233 msec (AUC of the model 0.83, sensitivity: 0.98, specificity: 0.66). Patients with SCN5A mutation compared with patients without had similar Ed before ajmaline [125.2 ms (117.9–132.6) vs 105.0 ms (103.0–119.0), $p=0.24$] but longer Ed after ajmaline [270.0 ms (259.0–280.5) vs 200.8 ms (181.2–224.2), $p=0.037$], figure 1. The best cutoff of Ed after ajmaline to predict SCN5A mutation was 256.5 ms (AUC of the model 0.84, sensitivity: 0.80, specificity: 0.98), figure 2.

Conclusions: High density RVOT epicardial mapping provides data of clinical meaning in risk stratification of BrS patients. These data should be interpreted in the context of a population undergone RVOT-EPI substrate ablation.

