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Targeting the Hidden Substrate Unmasked by Right Ventricular Extrastimulation Improves Ventricular Tachycardia Ablation Outcome After Myocardial Infarction

Marta de Riva, MD,[a] Yoshihisa Naruse, MD, PhD,[a] Micaela Ebert, MD,[a] Alexander F.A. Androulakis, MD,[a] Qian Tao, PhD,[b] Masaya Watanabe, MD, PhD,[a] Adrianus P. Wijnmaalen, MD, PhD,[a] Jeroen Venlet, MD,[a] Charlotte Brouwer, MD,[a] Serge A. Trines, MD, PhD,[a] Martin J. Schalij, MD, PhD,[a] Katja Zeppenfeld, MD, PhD[b]

ABSTRACT

OBJECTIVES This study sought to determine whether ablation of hidden substrate unmasked by right ventricular extrastimulation (RVE) improves ablation outcome of post-myocardial infarction (MI) ventricular tachycardia (VT).

BACKGROUND In patients with small or nontransmural scars after MI, part of the VT substrate may be functional and, in addition, masked by high-voltage far-field signals arising from adjacent normal myocardium.

METHODS In 60 consecutive patients, systematic analysis of electrograms recorded from the presumed infarct area was performed during sinus rhythm, RV pacing at 500 ms, and during a short-coupled RV extrastimulus. Sites showing low-voltage, near-field potentials with evoked conduction delay in response to RVE were targeted.

RESULTS In 37 (62%) patients, ablation target sites located in areas with normal voltage during sinus rhythm were unmasked by RVE (hidden substrate group). These patients had better left ventricular function (36 ± 11% vs. 26 ± 12%; $p = 0.003$), smaller electroanatomical scars (<1.5 mV), and smaller dense scars (<0.5 mV) (median 59 and 14 cm^2 vs. 82 and 44 cm^2; $p = 0.044$ and $p = 0.003$) than did those in whom no hidden substrate was identified (overt substrate group). During a median follow-up of 16 months, 13 (22%) patients had VT recurrence. Patients with hidden substrate had a lower incidence of VT recurrence (12-month VT-free survival 89% vs. 50% in patients with overt substrate; $p = 0.005$). Compared with a historical cohort of 90 post-MI patients matched for left ventricular function and electroanatomical scar area, in whom no RVE was performed, patients in the hidden substrate group had a higher 1-year VT-free survival (89% vs. 73%; $p = 0.039$).

CONCLUSIONS Hidden substrate ablation unmasked by RVE improves ablation outcome in post-MI patients with small or nontransmural scars. (J Am Coll Cardiol EP 2018;4:316–27) © 2018 by the American College of Cardiology Foundation.

From the [a]Department of Cardiology, Leiden University Medical Center, Leiden, the Netherlands; and the [b]Department of Radiology, Division of Image Processing, Leiden University Medical Center, Leiden, the Netherlands. The Department of Cardiology Leiden receives unrestricted research and fellowship grants from Edward Lifesciences, Boston Scientific, Medtronic, and Biotronik. Dr. Naruse was supported by an overseas research fellowship from the Japan Society for the Promotion of Science) and a 2014 to 2015 fellowship from the Japanese Heart Rhythm Society-European Heart Rhythm Association (sponsored by Biotronik). The authors have reported that they have no relationships relevant to the contents of this paper to disclose.
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Substrate-based ablation for post-myocardial infarction (MI) ventricular tachycardia (VT) relies on: 1) delineation of low bipolar voltage (BV) areas (<1.5 mV) by electroanatomical (EA) mapping; and 2) identification of electrograms (EGMs) consistent with slow conduction or conduction block within low-voltage areas during sinus rhythm (SR) (1,2). Mapping accuracy to detect scar may, however, be limited by the current use of ablation catheters with large electrodes and wide interelectrode spacing, leading to far-field contamination of local EGMs (3). This phenomenon may be particularly relevant in patients with smaller and subendocardial scars, in whom parts of the arrhythmogenic substrate may be obscured by high-voltage far-field signals arising from adjacent normal myocardium. Of note, this scar pattern is often encountered in contemporary patients undergoing early reperfusion therapy, and is associated with fast and poorly tolerated VT (4,5). The inability of BV mapping during SR to delineate nontransmural scars has been demonstrated by head-to-head comparison of voltage maps with contrast-enhanced magnetic resonance imaging (CE-MRI) (5). Changing the activation wavefront by continuous ventricular pacing may identify low-voltage areas that are not evident during SR (6). However, using EGM amplitude as the only guide may lead to unnecessary ablation, with potential damage of viable myocardium contributing to cardiac function. Critical sites for VT should exhibit additional electrophysiological characteristics, most importantly functional conduction delay or conduction block.

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We hypothesized that: 1) systematic evaluation of EGMs located in the infarct area during right ventricular extrastimulation (RVE) can identify scar areas with functional conduction delay or block as substrate for VT that are not evident during SR; and 2) ablation targeting this hidden, functional substrate may improve ablation outcome in post-MI patients.

METHODS

Between October 2013 and February 2016, consecutive post-MI patients referred for VT ablation to the Leiden University Medical Center were prospectively included. All patients were treated according to the institutional clinical protocol and provided pre-procedural informed consent.

PRE-PROCEDURAL EVALUATION. Patients underwent a comprehensive clinical evaluation including review of all medical records. Data on past ischemic events, acute reperfusion therapy, prior revascularization, spontaneous ventricular arrhythmias, implantable cardioverter-defibrillator (ICD) interrogation reports, and failed antiarrhythmic drugs (AADs) were collected. All patients underwent coronary angiography or noninvasive stress testing to detect ischemia. Coronary angiograms were reviewed to determine the area supplied by the infarct-related artery. Wall motion abnormalities and left ventricular (LV) function were derived from echocardiograms. If possible, CE-MRI was performed for scar delineation (Online Appendix) (7). Patients scheduled for epicardial ablation underwent electrocardiography-gated cardiac computed tomographic imaging for procedural integration (8). Before ablation, all AADs with the exception of amiodarone were discontinued.

ABLATION PROCEDURE. Before ablation, programmed electrical stimulation was performed (4 drive cycle lengths [CLs] [S1: CL = 600, 500, 400, and 350 ms], 1 to 4 ventricular extrastimuli [S2: ≥200 ms] from 2 RV sites and ≥1 LV site). Positive endpoint of stimulation was induction of sustained VT lasting >30 s or requiring termination because of hemodynamic compromise. Fast VT was defined as a VT with CL ≤ ventricular refractory period + 30 ms (9).

Electroanatomical mapping. All patients underwent LV endocardial mapping. Additional RV or epicardial mapping was performed if appropriate. BV maps were created with a 3.5-mm irrigated-tip catheter (NaviStar ThermoCool; Biosense Webster, Diamond Bar, California) and the CARTO 3 system (Biosense Webster). Standard cutoff values of 1.5 mV for scar, 0.5 mV for dense scar, and 0.5 to 1.5 mV for the border zone were applied. A patchy scar pattern was defined as 2 low-voltage areas separated by areas of preserved voltage (4). CE-MRI-derived scars were integrated with EA maps as previously described (7).

EGM analysis. Bipolar EGMs were filtered at 30 to 500 Hz and displayed at a gain of 0.072 mV/cm and a sweep speed of 200 mm/s on the Prucka EP system (GE Healthcare, Chicago, Illinois). With the mapping catheter in a stable position, EGMs were systematically analyzed during SR, RV pacing at a fixed rate of 500 ms, and the application of a single RV extrastimulus with a coupling interval of 50 ms above the ventricular refractory period. Care was taken to cover the presumed infarct area as derived from imaging data (echocardiogram and CE-MRI) and review of

ABBREVIATIONS AND ACRONYMS

AAD = antiarrhythmic drugs
BV = bipolar voltage
CABG = coronary artery bypass grafting
CE-MRI = contrast-enhanced magnetic resonance imaging
CI = confidence interval
EA = electroanatomical
EDP = evoked delayed potential
EGM = electrogram
HR = hazard ratio
ICD = implantable cardioverter-defibrillator
IQR = interquartile range
LAVA = local abnormal ventricular activity
LP = late potential
LV = left ventricular
LVEF = left ventricular ejection fraction
MI = myocardial infarction
RF = radiofrequency
RV = right ventricular
RVE = right ventricular extrastimulation
SR = sinus rhythm
VT = ventricular tachycardia