Endo-Epicardial mapping for ventricular tachycardia based on old myocardial infarction to identify tachycardia circuits involved intramural myocardium: A case report

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**Introduction**: Radiofrequency catheter ablation is a therapeutic option for recurrent ventricular tachycardia (VT) in ischemic cardiomyopathy. Usually endocardial mapping is performed for ablation of the VT in patients with old myocardial infarction (OMI); however, in some cases, epicardial mapping may be required to identify tachycardia circuits that can only be explained in three dimensional (3D) rather than 2D. We report a case in which epicardial approach was used to successfully ablate the VT involving an intramural/epicardial substrate.

**Methods**: N/A

**Result**: A 66-year-old man who had a history of anterior-septal OMI visited our hospital for respiratory distress. We did cardioversion because electrocardiogram indicated monomorphic ventricular tachycardia (VT). Electrophysiological study and catheter ablation for monomorphic VT were performed. Voltage map of left ventricle (LV) during right ventricular (RV) pacing indicated that there was scar and low voltage area from the LV septum to the apex. Activation map of the LV for induced VT1 (LBBB/superior, TCL 320 ms.) showed a centrifugal pattern from the apical septal wall and local activation time did not cover for tachycardia cycle length (TCL) of VT1. Entrainment pacing revealed that the post pacing interval (PPI) from a centrifugal pattern point of LV endocardium exceeded the TCL by 20 ms with manifest fusion. VT1 was terminated by ablation of that site; however, VT2 (RBBB/superior, TCL 340 ms.) was induced. Because there is no evidence for the endocardial circuit of VT1, we tried to evaluate VT2 from both the endocardium and the epicardium. We created an activation map of VT2 from the epicardium by subxiphoid approach. The earliest activation site was at the left ventricular apex, and then the excitation propagation was directed to the base of the anterior wall, exciting the conduction delay site of the anterior wall as a figure of eight. Despite adding the endocardial mapping, the endocardial and epicardial activation did not cover all phases of the tachycardia, and the epicardial reentry with partial intramural circuit was suspected. Unfortunately, the VT2 changed to VT3 (RBBB/superior, TCL 330ms) during mapping without detailed pacing evaluation. The propagation of
VT3 showed that a tachycardia circuit in which the activation sequence of “common channel” was reversed to that of VT2. This epicardial common channel site was ablated during VT3, and the tachycardia stopped. After that, the procedure was terminated without being induced any VT by programmed stimulation.

**Conclusion**: We experienced that, as VT based OMI, endocardial and epicardial approach were effective to identify 3D reentrant circuit of VT, where involvement of intramural site. Adding epicardial mapping and ablation may improve outcomes for ischemic VT involving intramural/epicardial circuit.