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Mismatch Between Cardiac Perfusion, Sympathetic Innervation, and Left Ventricular Electroanatomical Map in a Patient with Recurrent Ventricular Tachycardia

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 69
Final Diagnosis: Recurrent ventricular tachycardia
Symptoms: Multiple ICD shocks
Medication: —
Clinical Procedure: Ventricular tachycardia ablation
Specialty: Cardiology

Objective: Rare co-existance of disease or pathology

Background: Regional cardiac sympathetic denervation causes electrophysiological heterogeneity and has been found to be a predictor of potentially lethal VT.

Case Report: We present the case of 69-year-old patient admitted with recurrent ventricular tachycardia and a history of anterior myocardial infarction. In line with Tc-99m-MIBI-SPECT perfusion imaging, electroanatomical mapping revealed extensive LV anterior scarring as detected by low-voltage areas. Surprisingly, I-123-MIBG-SPECT showed an extensive deficit of sympathetic innervation inferior (mismatch) and anterolateral (match).

Conclusions: Combination of electroanatomical mapping with tomographic imaging of innervation and perfusion might improve our understanding of the neural trigger of VT after myocardial infarction or substrate-based catheter ablation.

MeSH Keywords: Arrhythmias, Cardiac • Autonomic Denervation • Cardiac Imaging Techniques

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/897412

Background

Regional cardiac sympathetic denervation causes electrophysiological heterogeneity and has been found to be a predictor of potentially lethal VT.

Case Report

A 69-year-old male patient with a history of anterior myocardial infarction in 1988 presented with recurrent ventricular tachycardia (VT) that had to be terminated by multiple shocks of his implantable cardioverter defibrillator. A left ventricular (LV) apex aneurysm and a low LV ejection fraction were confirmed in echocardiography. Resting Tc-99m-MIBI-SPECT reflected the anterior myocardial scar with corresponding perfusion deficit (Figure 1A) which are presented 3-dimensionally (3D) (left column: anterior view; middle column: posterior view) and as polar plot (right column). No high-grade coronary stenosis was found in coronary angiography. I-123-MIBG-SPECT 4 hours after intravenous injection [1] (Figure 1B) showed an extensive deficit of sympathetic innervation inferior (mismatch,#) and anterolateral (match). However, residual innervation could be documented in a basal anterolateral region with severely impaired perfusion (reverse mismatch,*). Electroanatomical mapping (by using image integration with fluoroscopy), in line with perfusion, supported extensive LV anterior scarring as detected by low-voltage areas (Figure 1C). Note also the electroanatomical polar plot (custom-made software) supporting scarring in the left anterior descending coronary artery perfusion

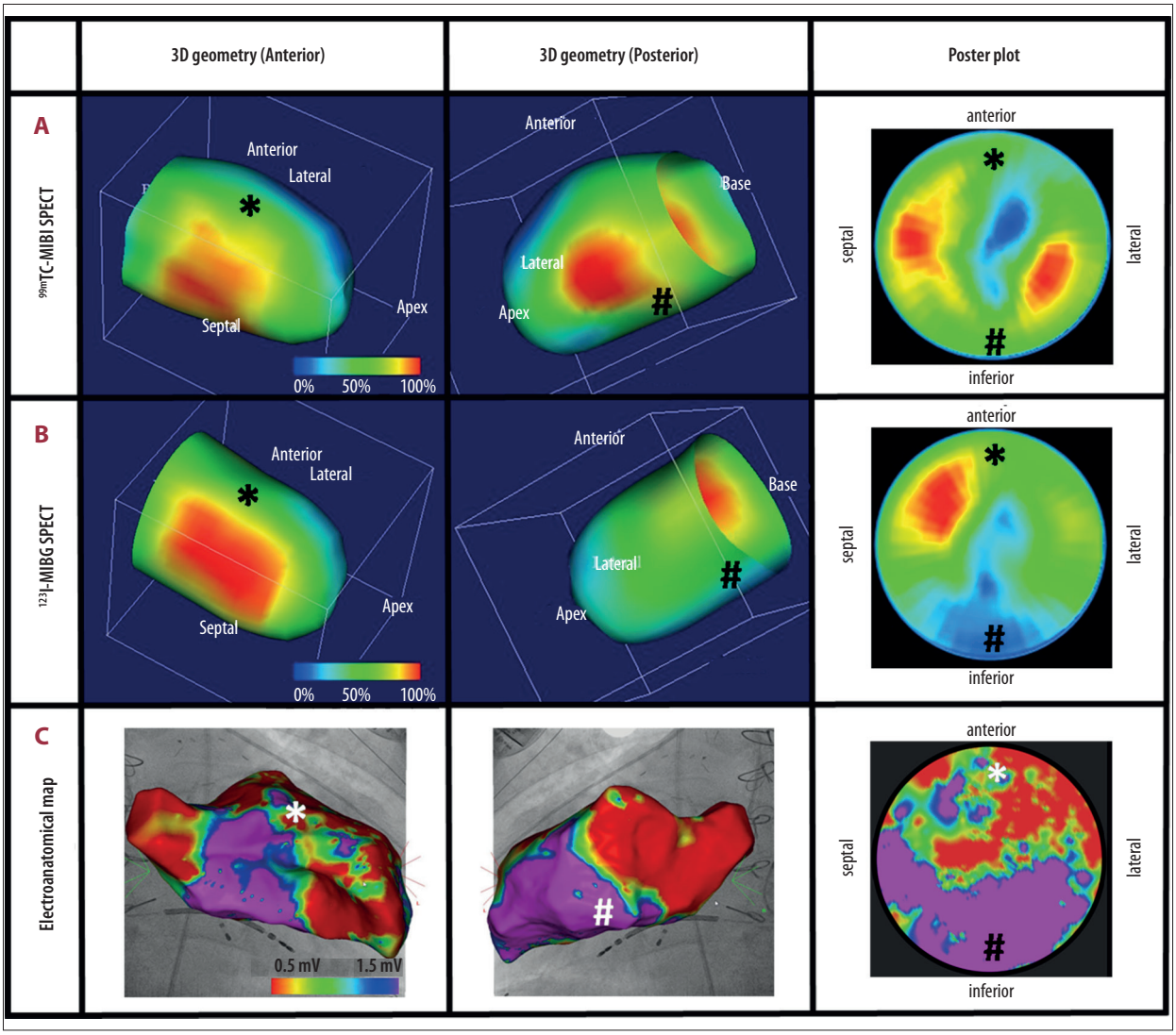

Figure 1. Mismatch between cardiac imaging modalities. (**A**) Tc-99m-MIBI-SPECT perfusion imaging reveals areas of reduced anterior and inferior perfusion. (**B**) I-123-MIBG-SPECT imaging demonstrates reduced sympathetic innervation at the inferior left ventricle. (**C**) Electroanatomical mapping depicts extensive LV anterior scarring as detected by low-voltage areas.

area. Substrate-based ablation was performed within the anterior myocardial scarring (low-voltage areas) [2]. Despite abolition of all signals indicating local abnormal ventricular activation, the patient again experienced a VT of midseptal origin (207 bpm, cycle length 290 ms) remote from the myocardial scar, which had to be additionally treated by radiofrequency catheter ablation.

Discussion

Denervation of inferior areas is known to occur in patients after modulating parts of the autonomic/sympathetic intracardiac nervous system located at the posterior wall of the left atrium during pulmonary vein isolation [3]. The present sympathetic innervation of the anterior non-perfused scar area might mirror a partial re-innervation 27 years after myocardial infarction. Regional cardiac sympathetic denervation causes electrophysiological heterogeneity in the myocardium [4,5] and has been found to be a predictor of potentially lethal VT [6–8]. Inhomogeneity in LV sympathetic innervation has also been described in areas remote from post-myocardial infarction scarring but is yet not fully understood [9].

Conclusions

Combination of electroanatomical mapping with tomographic imaging of innervation and perfusion might improve our understanding of the neural trigger of VT after myocardial infarction or substrate-based catheter ablation.

Conflicts of interest

None

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