

Correlation of inflammation due to cardiac sarcoidosis by positron emission tomography-computed tomography imaging and endocardial voltage mapping in a patient with recurrent ventricular tachycardia

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We present a patient with cardiac sarcoidosis and ventricular tachycardia who had correlative analyses of electrophysiologic, positron emission tomography (PET) imaging and pathologic findings. Voltage maps correlated with inflammation and not just scar by PET imaging with tissue pathology confirming presence of inflammatory sarcoid lesions. This case highlights a role for metabolic PET imaging in guiding catheter ablation of sarcoid-associated ventricular tachycardia.

Case report

A 48-year-old man with cardiomyopathy due to cardiac sarcoidosis presented with recurrent ventricular tachycardia (VT). He underwent an electrophysiology study with mapping which demonstrated low voltage (≤ 0.5 mV) regions (shown as red regions in Figure 1A and B) along the mitral and tricuspid annulus and septum. Catheter ablation (marked by red dots in Figure 1A and B) was performed by targeting sites with fractionation, late diastolic potentials and excellent pace maps (sample site marked by white arrow in Figure 1A).

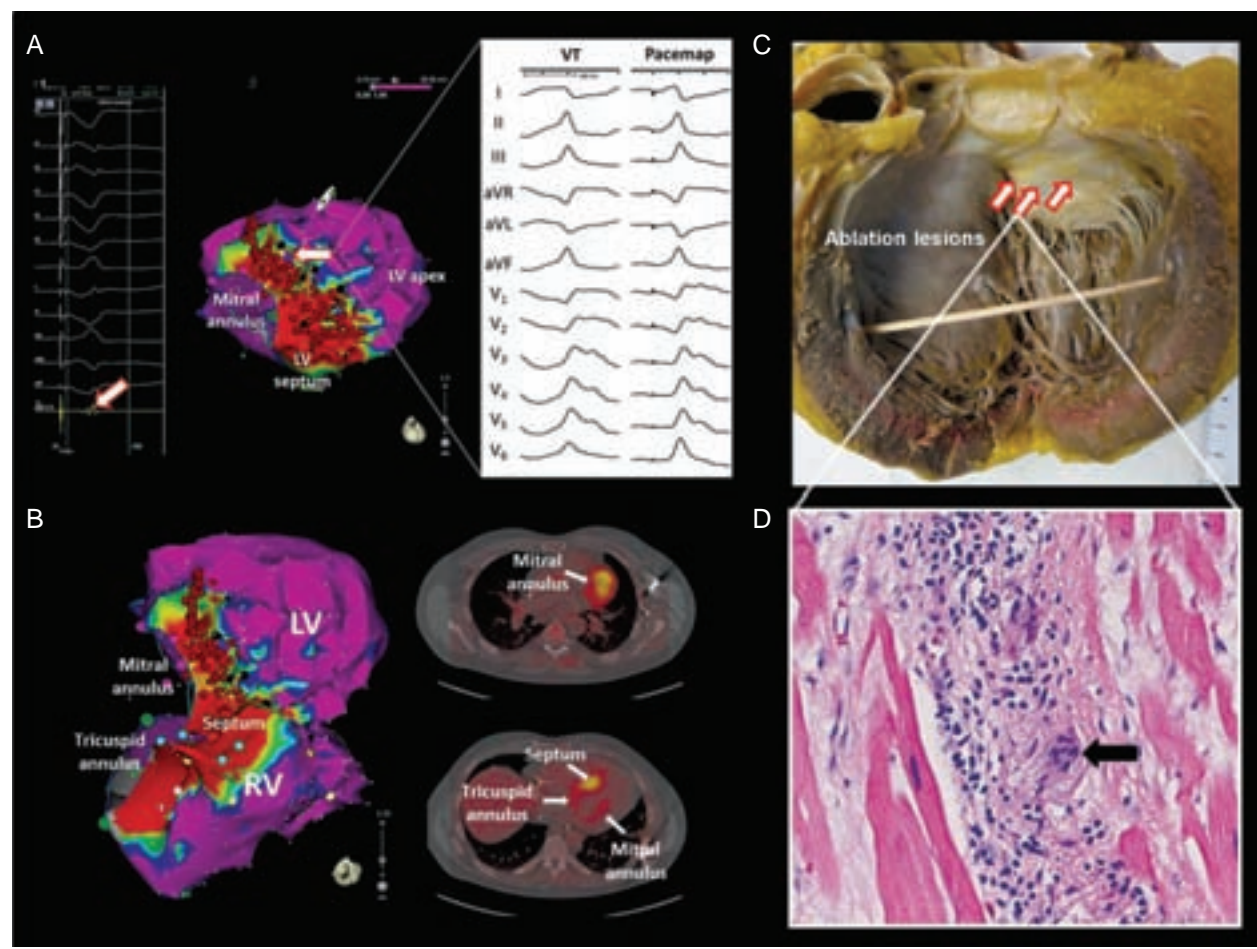


Figure 1 A. Endocardial left ventricular voltage map (middle panel) highlighting fractionated potentials (left panel) and excellent pace map (right panel) B. Left and right ventricular voltage map and correlation with PET-CT images. C and D. Gross and microscopic pathology of targeted anterior mitral annular sites.

Cardiac positron emission tomography (PET)-computed tomography (CT) scan with N-13 ammonia and F-18 fluorodeoxyglucose (FDG) radiotracers was performed > 2 weeks post-ablation to assess sarcoid disease activity. Areas of active sarcoid disease with increased inflammation and decreased perfusion by PET-CT (i.e. perfusion–inflammation mismatch) (*bright regions* in *Figure 1B*) correlated with low-voltage regions encountered during voltage mapping. At 10 months following ablation, the patient was free of VT but underwent cardiac transplantation due to refractory heart failure. Tissue pathology of areas targeted for VT ablation such as the anterior mitral annulus (ablation lesions marked by *white arrows* in gross pathology shown in *Figure 1C*) revealed presence of multinucleated giant cells (marked by *black arrow* in *Figure 1D*), lymphocytic infiltration and interstitial fibrosis consistent with inflammation and granuloma remnants.

Catheter ablation can be an effective adjunct therapy to implantable cardioverter-defibrillators in patients who present with VT due to cardiac sarcoid.¹ Ablation of sarcoid-related VT involves lesion delivery along the scar border targeting isthmus and exit sites.² Our case illustrates close correlation of low-voltage regions associated with VT with not just scar, but rather areas of active inflammation identified by cardiac PET imaging in a patient with sarcoidosis. This was further confirmed by tissue pathologic analysis. Cardiac PET with both N-13 ammonia, which assesses myocardial perfusion, and F-18 FDG, which assesses inflammation, has become a powerful tool in the characterization of cardiac sarcoid.³ Using dual radiotracer PET imaging, early-stage cardiac sarcoid lesions demonstrate normal or decreased perfusion and increased F-18 FDG uptake while late-stage lesions show matched decreases in both perfusion and F-18 FDG uptake. Metabolic cardiac PET imaging may have a role in guiding catheter ablation of VT due to cardiac sarcoidosis.

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