A Challenging VT Ablation with a Large Cardiac Tumor

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Abstract

Ventricular tachycardia (VT) normally occurs from an abnormal structural substrate. We report a case in which VT was caused by a large tumor in the interventricular septum. Surgical intervention was not an option due to the location of the tumor and its proximity to the coronary arteries. The patient underwent ablation and upgrade to CRT before ultimately receiving a heart transplant.

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Background: We describe an unusual case of ventricular tachycardia (VT) caused by a massive, benign cardiac tumor in an adult.

Case presentation: A 66-year-old man with history of cardiac fibroelastoma resected at the age of 13 presented with ventricular tachycardia (VT) storm. He initially developed VT at the age of 39, underwent catheter ablation resulting in RBBB, and had an ICD placed. Recently, episodes of slow VT (˜ 120 bpm) terminated by antitachycardia pacing became so frequent as to contribute to heart failure despite treatment with dronedarone and mexiletine. He was transferred to our center for VT ablation.

Case management:

Prior to the procedure, cardiac CT revealed a large tumor, which involved the interventricular septum and most of the LV anterior wall. The LV was somewhat compressed but with normal LV ejection fraction. A small amount of ventricular tissue could be appreciated between the right coronary cusp (RCC) and the tumor (Figure 1). Surgical resection was deemed not feasible due to the tumor's size and its juxtaposition to the coronary arteries. When not in VT, the patient's functional capacity was very good, and it was felt that cardiac transplant could be deferred if VT control could be achieved.

At EP study, left ventricular voltage mapping revealed an overall normal endocardial bipolar voltage (> 1.5 mV), but a large area of decreased (<8.3 mV) unipolar voltage extending from the LVOT onto the basal anterior wall, corresponding with the area of the tumor. Two hemodynamically tolerated VTs were induced by programmed stimulation (PES) (Figure 2). Entrainment mapping was limited by spontaneous VT termination and cycle length oscillations.

VT1 had RBBB morphology and activation mapping demonstrated a focal breakout at the mid lateral LV where signals were simultaneous with the QRS onset and entrainment was consistent with a bystander site

near the exit (Figure 2). This was consistent with an epicardial or an intramural component to the reentry circuit. Following spontaneous termination, pace mapping from the leftward aspect of the right coronary cusp (RCC) resulted in a perfect pace match to VT1 with a long Stim-QRS interval, potentially consistent with a proximal isthmus site (Figure 3A). Ablation in the RCC abolished this VT.

VT2 had LBBB morphology, and activation mapping demonstrated endocardial breakout in the same region as the putative proximal isthmus for VT1, just below the RCC, with activation proceeding toward the septum (Figure 2). Pace mapping here resulted in a perfect pace match (Figure 3A) with relatively short stim-QRS and ablation abolished the VT, consistent with a distal isthmus site. Ablation here also resulted in complete AV block. Further ablation was performed in the peri-aortic region extending toward the mid ventricle in the region of low unipolar voltage (Figure 3B). No VT was inducible following ablation with up to three extrastimuli following two drive trains (400 and 600ms). He recovered well after the procedure. ICD was upgraded to a CRT system and he has remained free of recurrent VT at 60 day follow-up . Cardiac PET/CT performed approximately 6 weeks after ablation did not demonstrate any metabolic activity in the tumor, consistent with a benign process.

He underwent evaluation for heart transplantation and was eventually transplanted successfully at another institution approximately four months following the ablation procedure. A biopsy from the cardiac mass was obtained following transplantation which revealed benign fibroblastic proliferation admixed with collagen, focal chronic inflammation, and occasional calcifications.

Conclusions: We speculate that this large tumor was the result of a slowly growing cardiac fibroma that, perhaps from pressure, resulted in fibrosis that extended deep to the endocardium in the anterior wall creating the substrate for reentry. This presented as two VT circuits that shared a common isthmus between the mass and the aortic root which could be ablated from the endocardium.

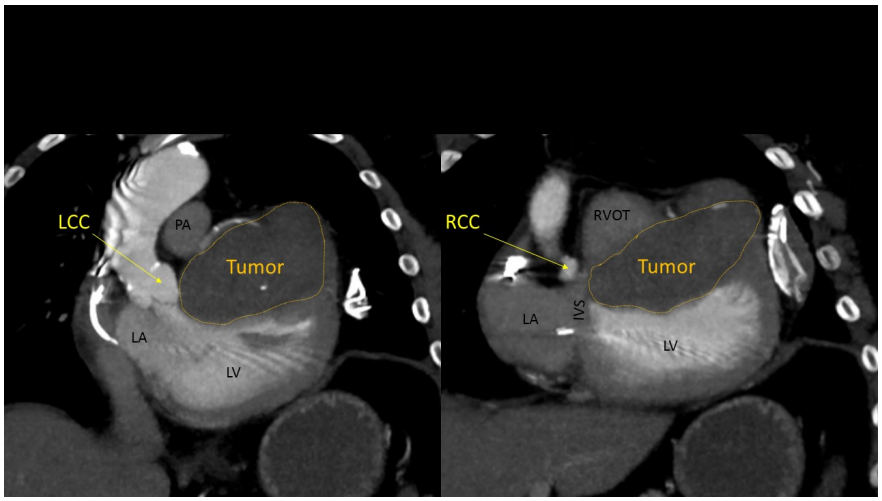

Figure 1 Cardiac CT revealed a large, septal LV tumor with a thin rim of intervening tissue between the tumor and the aortic valve (right image). RCC: right coronary cusp, LCC: left coronary cusp, IVS: interventricular septum.

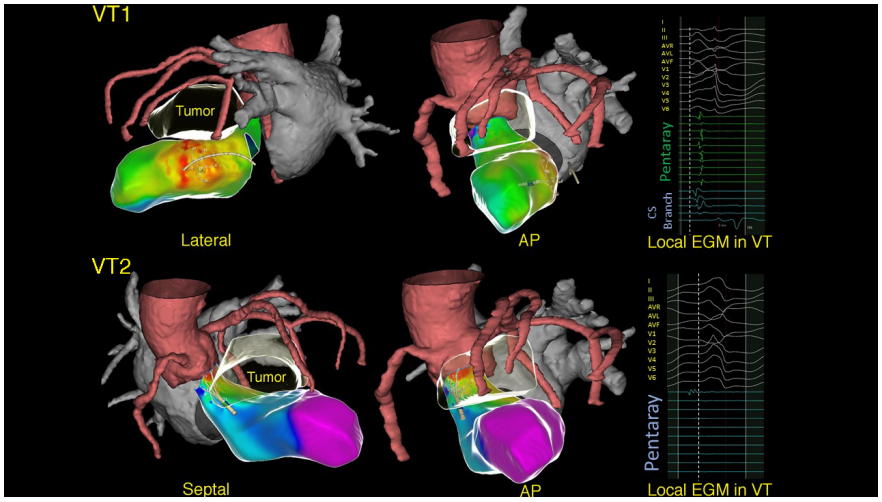

Figure 2 *Top panel:* VT1 activation map that revealed an endocardial breakout pattern at the lateral LV. Local signal there was simultaneous with QRS onset suggestive of an intramural or an epicardial component to the isthmus. *Bottom panel:* VT2 activation map which revealed a slightly presystolic signal just underneath the right coronary cusp consistent with a potential exit site.

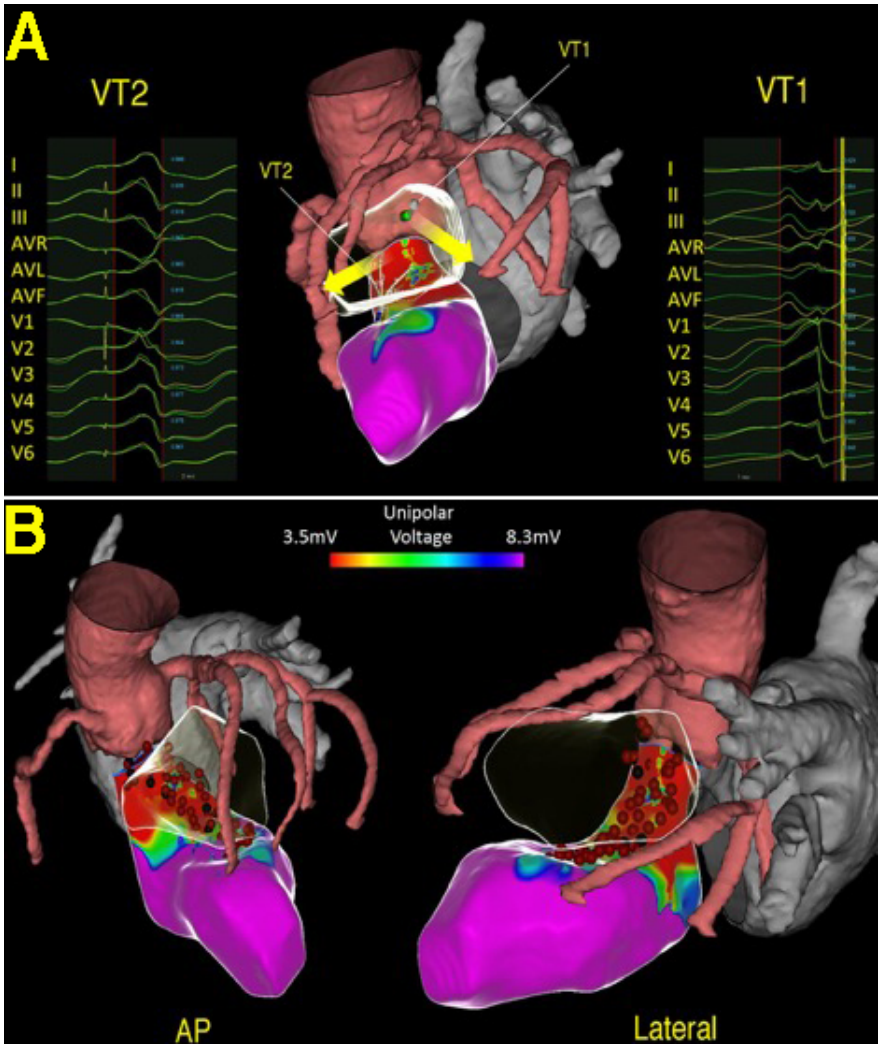

Figure 3 Pace mapping in the periaortic region resulted in excellent pace match for both VT1 and VT2 (Panel A). These regions were targeted for ablation which was extended apically to include the unipolar low voltage areas abutting the tumor (Panel B)