Defining the target for stereotactic radioablation of ventricular tachycardia: The combination of cardiac imaging and electrocardiographic information matters



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Introduction

Stereotactic arrhythmia radioablation (STAR) is a relatively novel noninvasive approach to treat ventricular tachycardia (VT) refractory to conventional antiarrhythmic and catheter ablation—based therapy. There is high uncertainty regarding the optimal size and the exact location of the target volume. We report a case of practically incessant slow intramural septal VT that was treated successfully with STAR. Using a combination of noninvasive electrocardiographic imaging (ECGI) to define the VT exit site and cardiac contrastenhanced computed tomography (CE-CT) to identify the scar, achieving a good result with a discrete target volume was possible.

Case report

A 65-year-old man with mixed ischemic–nonischemic cardiomyopathy, previous acute myocardial infarction and NYHA class III/IV heart failure (HF). Because of a left ventricular ejection fraction (LVEF) of 17% and a left bundle branch block (LBBB), a defibrillator with cardiac resynchronization therapy (CRT-D) was implanted in 2012. He underwent atrioventricular node ablation in 2021 for persistent atrial fibrillation after two failed catheter ablation procedures. Afterward, the LVEF increased to 30%, and the patient was in NYHA class II. He was given amiodarone for multiple episodes of nonsustained VT. Amiodarone was suspended in January 2022 because of hyperthyroidism.

In December 2022, he was admitted to the hospital for an arrhythmic storm, with recurrent syncope and the occurrence of eight shocks from the CRT-D due to fast VT. No electrocardiogram (ECG) of the VT was available.

KEYWORDS STAR; Ventricular tachycardia ablation; ECGI; Noninvasive ablation; Intramural origin (Heart Rhythm Case Reports 2025;11:74–78)

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KEY TEACHING POINTS

- Stereotactic arrhythmia radioablation (STAR) is a relatively novel and noninvasive approach to treating ventricular tachycardia refractory or unsuitable for catheter ablation.
- A trend toward a reduction of the target volume has been reported, resulting in fewer adverse events and better efficacy.
- This case report describes a method of precise integration of electrophysiologic and anatomic data with STAR planning to reduce target volume.

A CE-CT scan using ADAS 3D software (Galgo Medical SL, Barcelona, Spain) showed a wide left ventricle lateral wall scar and a small intramural scar at the basal interventricular septum. The patient underwent substrate ablation because the VT was not tolerated, with an endocardial and epicardial approach for left ventricle lateral wall scar homogenization by eliminating all local abnormal ventricular activity within the low-voltage areas (Figure 1). He was discharged with sotalol (80 mg twice a day). The VT recurred 2 months later, and a second ablation was performed in April 2023 with a left ventricular assist device Impella 5.5 (Abiomed, Danvers, MA) to provide hemodynamic support during VT mapping and pulsed-field ablation (CENTAURI PEF System, Galvanize Electrophysiology, San Carlos, CA; Figure 1). The VT did not relapse, but there was a worsening of the HF with an LVEF of 17%, and the patient remained hospitalized for 2 weeks, ineligible for heart transplantation because of severe pulmonary hypertension.

Six months later, he was admitted to the hospital again due to aggravated HF. An ECG performed at admission showed a slow VT at 130 bpm, with a superior axis and precordial transition at V4 (Figure 2A). An electrical cardioversion was performed because of symptomatic HF, and mexiletine was added.

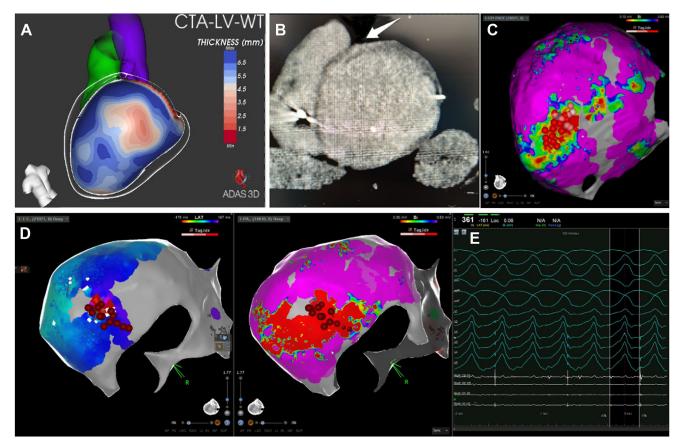


Figure 1 A: Contrast-enhanced computed tomographic scan before first ablation displays an ischemic scar at the lateral wall. **B:** Late contrast enhancement in the septum (*arrow*). **C:** First substrate-guided ventricular tachycardia (VT) ablation, both endocardial and epicardial. **D:** Second ablation activation and voltage map with pulsed-field ablation applications (*dots*). **E:** Hemodynamically unstable VT and electrogram at the ablation site.

The 12-lead electrocardiogram let to the origin of VT being suspected as originating from the septum. At this time, the patient was highly symptomatic. A 24-hour Holter recording showed an almost incessant slow VT below the implantable cardioverter defibrillator (ICD) detection rate (Figure 2B). The CE-CT scan was repeated, displaying identical findings, such as the scar at the basal anterior septum (Figure 2C). The VT was induced with noninvasive programmed stimulation (NIPS) using the ICD, which showed a 12-lead matching with the clinical VT (Figure 2D). The ECGI identified the VT exit point in the basal anterior septum at the place of the septal scar (Figure 2D). The superior axis of the clinical VT made us question the ECGI location, and the case was reviewed by other experts blinded to the scar area, confirming the origin of the tachycardia from the basal anterior septum. Because of concerns regarding the potential inability to reach the suspected intramural site of VT origin, the patient's NYHA class with LVEF of 17%, and the reluctance about a third VT catheter procedure, an ablation with STAR was proposed to the patient.

Materials and methods

The treatment approach was previously approved by a multidisciplinary team of cardiac electrophysiologists,

cardiologists, radiologists, radiation oncologists, and medical physicists.

The patient provided informed written consent. A customized immobilization vacuum cushion (Bluebag System; Elekta, Stockholm, Sweden) was used to ensure the same patient position for both the deep inspiration breath-hold (DIBH) CE-CT and the DIBH planning CT (PL-CT) that were performed on the same day. The clinical target volume (CTV) for ablation was delineated during a multidisciplinary contouring session, using the CE-CT scan to define all cardiac structures and scars based on myocardial wall thickness (<5 mm) and LCE involving >50% of the myocardial thickness. The CTV included the whole transmural myocardium encompassing the scar. However, an ECGI was performed with the VIVO system (Catheter Precision, Fort Mill, SC), as described previously,³ to confirm the VT exit point (Figure 2). The CE-CT was co-registered with the PL-CT using the Monaco treatment planning system (Elekta, Stockholm, Sweden). A planning target volume (PTV) was then created by adding a margin of 5 mm in three dimensions to the CTV to compensate for the heart motion. The organs at risk including lungs, healthy heart, left and right coronary arteries, ascending aorta, pulmonary artery, esophagus, stomach, and spinal cord were delineated (Figure 3). Because the patient already had an atrioventricular block and a

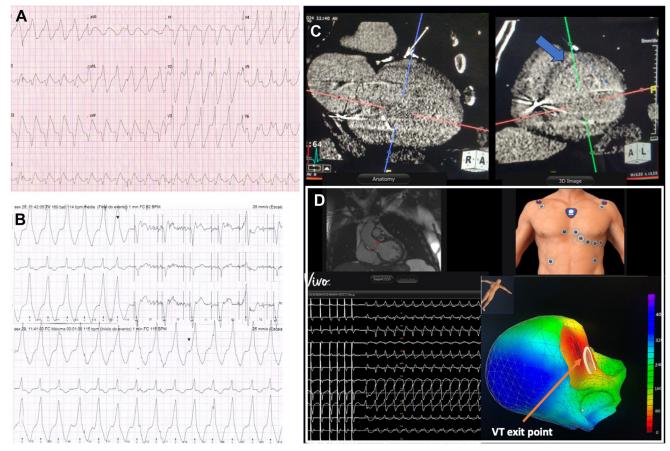


Figure 2 A: Clinical ventricular tachycardia (VT). **B:** Twenty-four-hour Holter monitor showing biventricular pacing alternating with slow VT at a rate of 114 bpm. **C:** Contrast-enhanced computed tomographic (CE-CT) scan showing late enhancement in the basal anterior septum (*white arrow*). **D:** VIVO workflow: clinical VT induced with noninvasive programmed stimulation and CE-CT scan; 3-dimensional image of the torso with VIVO patches and VT exit point (*orange arrow*).

CRT-D, no constraints were taken regarding the His-bundle and the LBB. The PTV was kept as small as possible.

The STAR treatment planning was generated in the Monaco planning system to deliver a total dose of 25 Gy to the PTV (19.5 mL) at the anterior basal septum (Figure 3) in a single fraction, with the patient in the immobilization vacuum cushion. The aim was to achieve 95% coverage of the PTV region while the constraints of the organs at risk were fulfilled. The radiation was delivered in two full arcs with flattening filter-free beams of 6 MV photons from an Elekta Versa HD Linear Accelerator (Elekta). The delivery was performed with respiratory control using DIBH (Catalyst HD system; C-RAD). The treatment data are displayed in Figure 3.

The CRT-D parameters were stable after therapy. The patient was kept in the hospital, monitored for 24 hours, and discharged the next day with the usual medication, including oral anticoagulation. The 12-lead ECG showed normal biventricular pacing. Programming of CRT-D after therapy included three zones: (1) ventricular fibrillation zone with a single anti-tachycardia pacing (ATP) followed by shocks; (2) VT zone set at 120 bpm, with six ATPs followed by shocks; and (3) VT monitor zone (no therapy) set at 100 bpm.

Three days after STAR, the patient was readmitted because of the recurrence of VT in the monitoring zone, associated with aggravated heart failure. The VT was converted to sinus rhythm with NIPS. He stayed in the hospital for 1 week, during which the VT did not recur. The ICD was programmed to a lower detection in the VT zone at 110 bpm. One week later, he had another episode of VT detected by the ICD and treated with ATP. After a 4-month follow-up, the patient was asymptomatic without acute adverse events or new episodes of VT or HF. The LVEF remained at 17%, the Holter did not show any episode of VT, and the results of a thoracic CT scan performed at 3 months were normal.

Discussion

Although still a bailout strategy⁴, STAR offers a successful noninvasive option for treating repetitive slow VT. In this case, the ECGI was a valuable tool that played a fundamental role in identifying the VT origin within the scar, whereas the 12-lead ECG suggested a more inferior location. Identifying an intramural origin of the VT was pivotal for the decision to treat the patient with STAR. Although many strategies have been described to improve the success of septal ablation, we

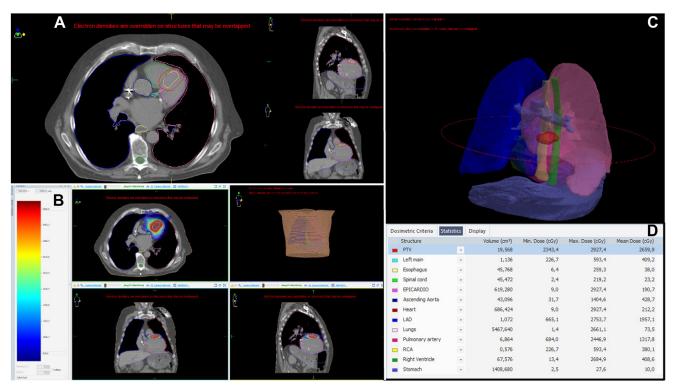


Figure 3 A: Treatment plan simulation. Planning target volume (PTV) contour, delineated by the red contour line, was obtained through an integrated "DIBH" contrast-enhanced computed tomographic (CT) scan with a planning (PL) CT scan. B: PTV and organs at risk contours in the axial, sagittal, and coronal views of the PL-CT scan. Dose distribution in the axial, sagittal, and coronal views of the PL-CT scan. C: Three-dimensional model view of 360° volumetric modulated arc therapy arcs. D: Dose-volume histogram values.

decided against these options due to concerns about another extensive procedure in a critically ill patient and the low success rate.⁵

Radioablation can deliver ablative energy to any internal volume within the body, making it possible to perform transmural ventricular ablation reaching otherwise inaccessible arrhythmogenic substrates. Precise target delineation, ensuring that the critical parts of the VT circuit are incorporated in the PTV, is crucial for the success of the treatment. However, avoiding unnecessary large treatment volumes is also essential because data suggest that irradiating relatively small volumes might be sufficient for VT reduction.⁶

Uncertainty exists regarding the optimal size of the target volume and whether the treatment should be aimed at the clinical VT or if the whole arrhythmogenic substrate should be irradiated.⁷ There is unanimous agreement that this method requires accurate preprocedural identification of the culprit arrhythmogenic substrate. This is accomplished by using scar imaging, concomitant with identifying the site of origin of the VT for which ECGI is promising due to its noninvasive nature.⁸

Manual interpretation of the QRS morphology in VT has been used to help guide STAR. Contemporary studies have shown limited accuracy of QRS morphology analysis (range 39%–82%) compared with ECGI. Often, the breakout of the VT can be remote from the critical isthmus, especially in the septum. In this case, using the 12-lead ECG alone would point to a septal inferior origin, which would have led to a

more inferior extension of the clinical target volume with an increase in the treated volume. Although electroanatomic mapping (EAM) remains the most accurate method of identifying the critical isthmus of the VT, co-registration of the EAM and the PL-CT may be complicated and inaccurate.

Here, we present a case in which the CE-CT scan used for the ECGI and scar identification was performed on the same day using the same immobilization device as the PL-CT scan. This allows a perfect alignment for target planning. When the target area is delineated based exclusively on anatomic premises, as reported by some groups, ¹¹ or when an EAM from previous procedures is used for target planning, the volume is much higher than when the ECGI is used. ¹²

In addition, using the DIBH protocol, delivering radiotherapy only during a prespecified window has been associated with smaller PTVs compared with free-breathing radiotherapy. ¹⁰

The mechanism of radiation injury to the cardiac tissue is not fully understood. Histologic studies have demonstrated the presence of myocardial cell death by apoptosis within the first months, followed by fibrosis. 12,13 However, most clinical cases and series have shown a rapid reduction of arrhythmia burden within days, suggesting an antiarrhythmic mechanism of STAR occurring earlier and different from fibrosis. Changes in the electrophysiologic properties of the circuit might be the answer to this question. 14

Our results demonstrate that the effectiveness of STAR is not due to the development of large areas of fibrosis since success was achieved by treating just a small volume. Although clinical data is still sparse, a correlation between larger PTV size and less favorable results has been suggested. A trend toward a reduction of the PTV has been reported. Over the duration of the ENCORE trial, there was a significant decrease in the delineated target volume over time. 15

Conclusion

This case report underscores the importance of precise delineation of the target for STAR. It describes a method for directly integrating the electrophysiological data, the CE-CT scan, and the pre-STAR PL-CT study. Using the ECGI, the vacuum immobilization device and the same respiratory phase for both examinations ensure the same position, promising greater accuracy in delineating the target.

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