

# Right ventricular free-wall scar: an exceptional source of post-infarction ventricular tachycardia. A case report

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## Background

In patients with coronary artery disease, ventricular tachycardia (VT) is usually related to left ventricular (LV) post-infarction scars.

## Case summary

A case of a 78-year-old man with post-infarction VT originating from the right ventricular (RV) free wall is described. Following recurrent episodes of VT with left bundle branch block morphology and left superior axis deviation, a patient with prior myocardial infarction was submitted to catheter ablation. Two areas of abnormal bipolar electrograms were observed at 3D electroanatomical mapping: one located at the basal aspect of the posterior and postero-septal LV, and the other one extending from the antero-lateral to the posterior mid-basal RV free wall. Ventricular late potentials (LPs) were recorded within both scars, but only pacing from those located in the RV resulted in long stimulus-to-QRS latency and optimal pace-mapping. Accordingly, this substrate was deemed the culprit of the clinical VT. Radiofrequency catheter ablation aimed at eliminating all LPs recorded from both scars was effective in preventing VT recurrences at follow-up.

## Discussion

A post-infarction RV free-wall scar may exceptionally be responsible of VT occurrence. Right ventricular mapping should be considered in selected cases based on 12-lead electrocardiogram VT morphology and prior RV infarct.

## Keywords

Right ventricular infarction • Ventricular tachycardia • Cardiac mapping • Catheter ablation • Myocardial infarction • Case report

## Learning points

- Post-infarction scars located at the right ventricular free wall may be the substrate of monomorphic, re-entrant ventricular tachycardia.
- Right ventricular electroanatomical substrate mapping should be performed when a right ventricular origin of the tachycardia is suspected, and is valuable in identifying the location and the extent of the abnormal, arrhythmogenic myocardium.

## Introduction

The right ventricle (RV) is frequently the chamber of origin of scar-related ventricular tachycardias (VTs) occurring in patients with arrhythmogenic RV cardiomyopathy (ARVC) or cardiac sarcoidosis. Conversely, in patients with coronary artery disease, VT is usually related to left ventricular (LV) post-infarction scars. The RV involvement in the VT mechanism is uncommon and mainly limited to the right aspect of the interventricular septum.<sup>1–5</sup> This report describes a

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case of post-myocardial infarction, scar related, VT that was effectively treated by radiofrequency (RF) catheter ablation performed at the RV free wall.

## Timeline

Time	Events
25 years before presentation	Acute, ST elevation, infero-posterolateral myocardial infarction treated with thrombolysis i.v.
At presentation	Emergency Department admission for haemodynamically stable (arterial blood pressure: 90/60 mmHg; O <sub>2</sub> saturation: 94%) sustained ventricular tachycardia (VT) at 210 b.p.m.
8 days after presentation	Cardioverter/defibrillator implantation
11 days after presentation	Electrical storm (three effective cardioverter/defibrillator shocks, 10 ineffective, and one effective antitachycardia pacing attempt) due to recurrent episodes of the clinical VT. Amiodarone administration (i.v.) started.
13 days after presentation	Catheter ablation of VT arising from a post-ischaemic right ventricular free-wall scar
4 months after discharge	Patient asymptomatic; uneventful follow-up

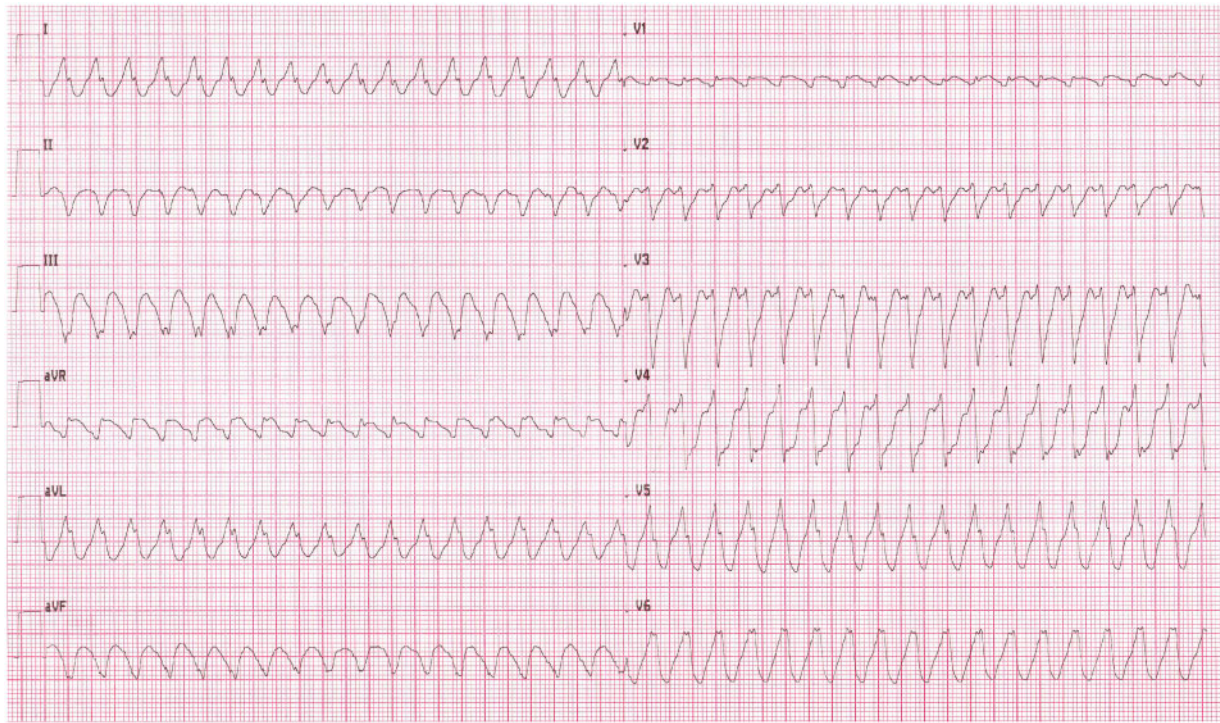
## Case presentation

A 78-year-old man with prior, ST elevation, infero-posterolateral myocardial infarction treated with systemic thrombolysis was admitted to the Emergency Department for prolonged palpitations associated with shortness of breath. Other cardiovascular risk factors included arterial hypertension, diabetes, and mixed dyslipidaemia. Surface 12-lead electrocardiogram (ECG) showed VT at 210 b.p.m. with left bundle branch block morphology, left superior axis deviation, and precordial transition in lead V5 (Figure 1). Despite symptoms, VT was haemodynamically tolerated: arterial blood pressure measured 90/60 mmHg, oxygen saturation was 94%, and there were no signs of pulmonary or peripheral stasis. Tachycardia was terminated by electrical cardioversion and a single-chamber cardioverter-defibrillator (ICD) was subsequently implanted. Pre-implant 2D-echo evaluation (Supplementary material online, Video S1) showed an aneurysm located at the basal aspect of the infero-posterior LV free wall, a moderate mitral valve insufficiency, and a globally preserved LV function (LV ejection fraction: 50%). A moderate RV dysfunction with a systolic bulging of the lateral RV free wall was also noted (Supplementary material online, Video S2). At angiographic evaluation, a dominant right coronary artery was chronically occluded at its origin, while the left branches had stenoses (70–85% vessel narrowing)

not amenable at percutaneous revascularization (marked vessel tortuosity and calcification). At Day 2 and 3 after ICD implant the patient experienced multiple ICD shocks (one and three, respectively) for recurrent episodes of the clinical VT and, thus, amiodarone administration was started and continuously maintained intravenously to achieve electrical stability. Conversely, beta-blocker therapy dosage was not increased to avoid excessive bradycardia. Given the negative impact of an electrical storm on prognosis, catheter ablation procedure was planned. The clinical VT with a cycle length duration of 310 ms was induced at baseline programmed ventricular stimulation (two extrastimuli) and interrupted by overdrive pacing due to poor haemodynamic tolerance. Retrograde LV 3D electroanatomical substrate mapping (CARTO 3; Biosense Webster, Diamond Bar, CA, USA) was initially performed with a 3.5 mm, irrigated-tip ablation catheter (ThermoCool SmartTouch SF Bidirectional; Biosense Webster, Diamond Bar, CA, USA). A 24.5 cm<sup>2</sup> area of abnormal bipolar voltage signals (0.5–1.5 mV) was noted at the basal aspects of the posterior LV free wall and of the posterior interventricular septum (Figure 2). At these levels, a few late potentials (LPs) inscribing beyond the end of the QRS complex were recorded in sinus rhythm, but quick activation mapping performed during induced VT did not show any diastolic activation (earliest LV activation recorded at the interventricular septum) and pace-mapping resulted in paced-QRS morphologies poorly matching the VT morphology. According to these findings, RV substrate mapping was also performed, showing a larger area of abnormal bipolar voltages (34.5 cm<sup>2</sup>) extending from the antero-lateral to the posterior aspect of the mid-basal RV free wall (Figure 2). At the borders and within this area, several isolated or multicomponent LPs (Figure 3A) exhibiting a centripetal gradient of delayed activation (up to 100 ms beyond the end of the QRS complex) were recorded (Figure 3B). Pacing at sites with LPs resulted in variably long stimulus-to-QRS latencies and optimal pace-mapping (i.e. paced-QRS morphology concordant with that of the VT in 12/12 surface ECG leads) was also demonstrated (Figure 3C). No further activation mapping during induced VT was performed because of poor patient compliance. Radiofrequency current (40 W, 40 s, target contact force ≥10 g) was then sequentially delivered at all sites showing LPs up to their complete abolition (Figure 3D). Ablation was finally extended also to LPs recorded at LV substrate mapping. Repeated programmed ventricular stimulation failed to induce any VT, and no acute or peri-procedural complication occurred. The patient was discharged without antiarrhythmic drug therapy and ICD programming was left unchanged. No VT recurrence was observed during a 4-month follow-up. Post-discharge genetic testing did not disclose any mutation associated with ARVC, and <sup>18</sup>F-fluorodeoxyglucose (FDG) positron emission tomography (PET) excluded cardiac inflammatory infiltrates suggestive of sarcoidosis. Given the age of the patient and the absence of anginal symptoms, a medical management of the coronary artery disease was planned.

## Discussion

Although described in an experimental model,<sup>6</sup> post-myocardial infarction, scar related, VTs rarely originate from the RV. In previous case reports and series, catheter ablation solely directed at or further

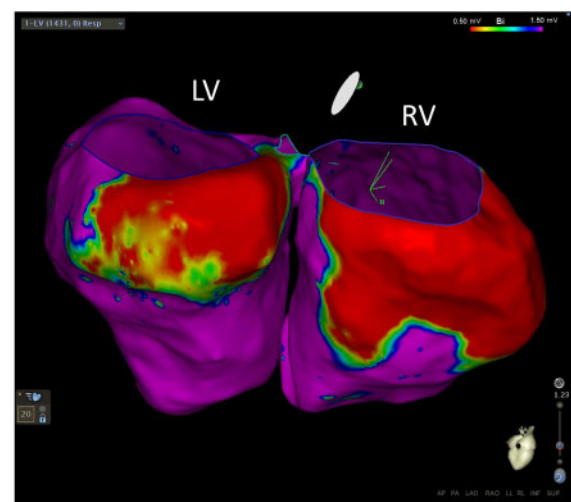


**Figure 1** Twelve-lead electrocardiogram of the clinical ventricular tachycardia at 210 b.p.m., left bundle branch block morphology and left superior axis deviation.

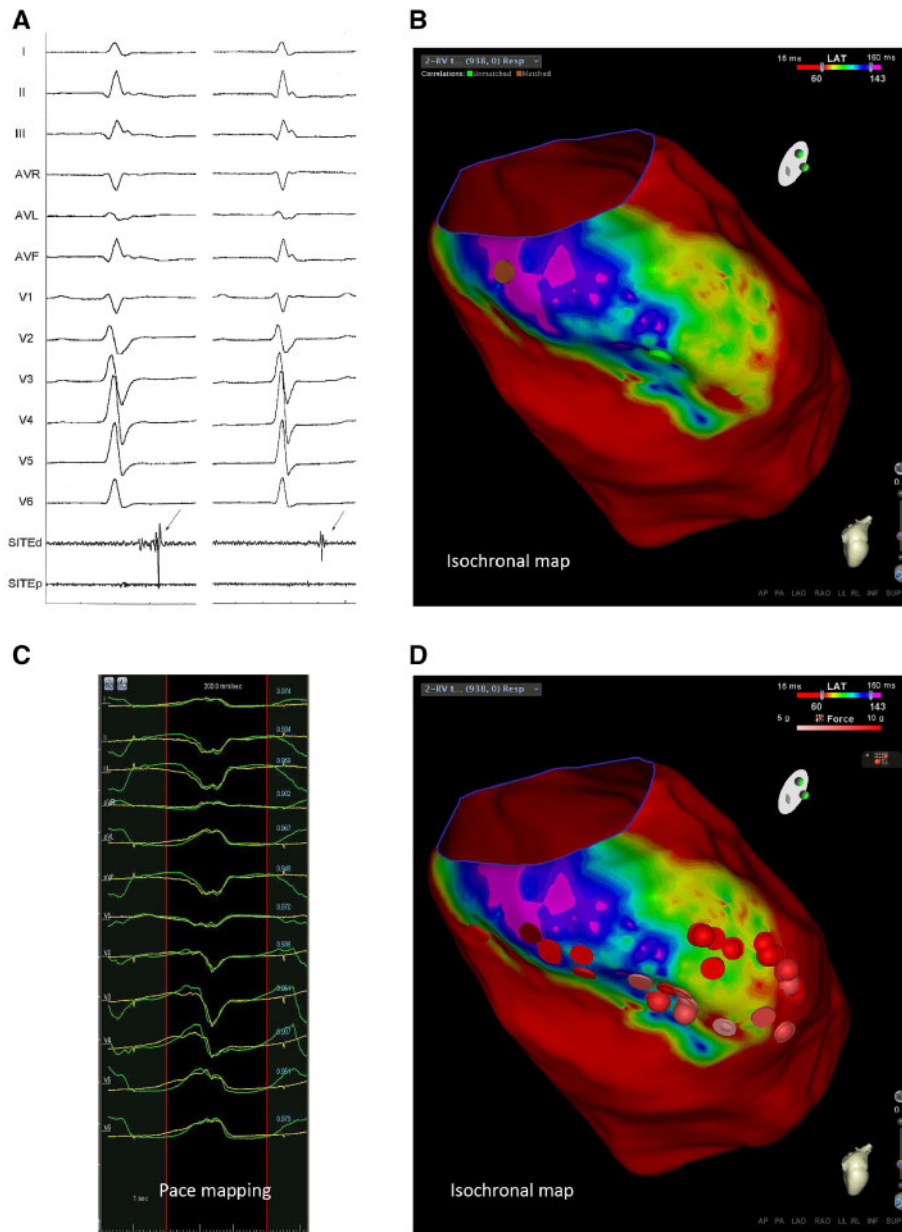
extended to the right side of the interventricular septum demonstrated to be effective in terminating and preventing re-induction of post-infarction VT.<sup>1-4</sup> To our knowledge, this is a very rare case in whom the VT substrate was located at the RV free wall. In fact, apart from a single case report,<sup>5</sup> the involvement of the RV free wall in post-myocardial infarction VT has never been described in other case series,<sup>7</sup> randomized studies,<sup>8</sup> or large multicentre registries.<sup>9</sup>

The very long time elapsed from the acute myocardial infarction and the first documented VT is also unusual. Clinical characteristics predictive of very late occurrence of VT have never been demonstrated, but this event seems more common after an inferior wall myocardial infarction.<sup>10</sup>

In patients with an electrical storm ( $\geq 3$  appropriate ICD interventions occurring in 24 h), a catheter ablation performed in experienced centres is strongly recommended by current treatment guidelines<sup>11</sup> given the positive impact of this procedure on important outcome measures (including improved survival).<sup>9</sup> In this setting, medical therapy, mainly including beta-blocker agents and/or amiodarone, as well as other interventions (i.e. ICD reprogramming, sedation, etc.) should be only considered to acutely manage the patient and to defer catheter ablation once electrical stability has been achieved. Conversely, given the paucity of data from large randomized clinical trials, either amiodarone or catheter ablation are recommended in case of recurrent (or even first occurring) VT causing ICD shock.<sup>11</sup> Therefore, in this setting, the decision basically depends on



**Figure 2** Left and right ventricular electroanatomic bipolar voltage maps showing the presence of two areas of abnormal substrate (electrogram amplitude  $< 1.5$  mV, coded with colours from blue to red). The left ventricular scar is located at the basal aspect of the posterior and postero-septal wall, while the right ventricular scar extends from the antero-lateral to the posterior aspect of the mid-basal free wall. LV, left ventricle; RV, right ventricle.



**Figure 3** (A) Examples of late potentials (arrows) with different activation times recorded within the right ventricular free-wall abnormal substrate. (B) Isochronal late activation map (3 ms difference between isochrones) of the right ventricle in sinus rhythm. A centripetal gradient of delayed activation (colour range from red to purple) is evident within the abnormal voltage area. (C) Twelve-lead electrocardiogram recordings during pace-mapping. Pacing at sites showing late potentials (orange dot in B) results in paced-QRS morphologies matching those of the induced ventricular tachycardia in 12/12 electrocardiogram leads with a long stimulus-to-QRS latency (135 ms). (D) Ablation lesions (red and pink dots) deployed within and at the borders of the right ventricular scar.

the patient's clinical profile (i.e. age, type of structural heart disease, advanced heart failure, significant comorbidities, treatment contraindication or tolerability, etc.) and preference.

The RV is involved in 30–50% of patients with LV inferior wall and/or infero-septal myocardial infarction.<sup>12</sup> In the acute phase of RV myocardial infarction, electrical instability frequently occurs as malignant ventricular tachyarrhythmias or electrical storms have been

reported in 38% and 8.3% of the cases, respectively.<sup>13</sup> However, the RV is more resistant to relatively long periods of ischaemia, and it better recovers contractility after reperfusion.<sup>14</sup> These characteristics might account for a lower amount of scarring in the RV following ischaemic injuries. Nonetheless, in the present case, a relatively large, arrhythmogenic, RV free-wall scar was demonstrated by the presence of abnormal electrograms and LPs at electroanatomical

substrate mapping.<sup>15</sup> The topographic location and size of the RV scar is consistent with the proximal occlusion of the dominant right coronary artery.<sup>16</sup> The origin of the clinical VT from the RV can be suspected according to the 12-lead ECG morphology<sup>17</sup> and to the 2D-echo findings, and in these cases electroanatomical mapping should be extended to the RV chamber. Thus, our case highlights the importance of performing programmed ventricular stimulation and of 12-lead ECG recording during clinical or induced VT, even when substrate modification is attempted, in order to not miss uncommon VT substrates. On the other hand, since similar 12-lead ECG VT morphologies can occur in case of an exit site from the left interventricular septum<sup>17</sup> and given the presence of the LV aneurysm, we decided to perform LV mapping as well.

Although we did not perform RV activation mapping during the ongoing VT, the presence of multiple LPs, the optimal pace-mapping associated to long stimulus-to-QRS latency, and the catheter ablation results strongly support the critical role of this RV scar in the tachycardia mechanism. The electroanatomical findings in this case closely resemble those observed in patients with ARVC<sup>18</sup> or cardiac sarcoidosis.<sup>19</sup> However, the former diagnosis can be reasonably excluded according to the age of the patient, his negative familiar history, the lack of typical ECG abnormalities (i.e. epsilon wave, inverted T waves in precordial leads), the negative genetic testing and the angiographic findings.<sup>16</sup> On the other hand, the lack of atrioventricular conduction abnormalities and the negative results of <sup>18</sup>F-FDG PET makes cardiac sarcoidosis very unlikely.<sup>19</sup> The occurrence of frequent VT relapses (including an electrical storm) in the first days after ICD implant could have been fortuitous. However, either the presence of the RV lead or the close proximity of the pacing site to the scar<sup>20</sup> might have also played a role in these events.

Finally, we extended RF ablation also at sites exhibiting LPs in the left-sided scar. Although this decision could be debatable, we did it because of the possible involvement of that areas in other, non-clinical VTs.<sup>21</sup>

## Conclusions

In conclusion, a post-myocardial infarction scar located in the RV free wall may be responsible for VT occurrence. In this case, RF catheter ablation aimed at modifying the arrhythmogenic substrate, was demonstrated to be effective in preventing VT recurrences at short-term follow-up.

## Lead author biography



Massimo Tritto, MD, is Director of Electrophysiology and Cardiac Pacing Unit at Humanitas Mater Domini Hospital. His electrophysiology research focuses on the mechanisms of cardiac arrhythmias, catheter ablation of complex arrhythmias, and resynchronization therapy. He is also an active member of several Italian and European Scientific Societies.

## Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

**Slide sets:** A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

**Consent:** The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** none declared.

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