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CASE REPORT

CLINICAL CASE

Scar-Related Monomorphic Ventricular Tachycardia After Treated Right Ventricular Metastatic Diffuse Large B-Cell Lymphoma

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ABSTRACT

A patient with ventricular tachycardia (VT) and right ventricular (RV) metastatic diffuse large B-cell lymphoma had persistent RV gadolinium enhancement following chemotherapy and disease remission. Electrophysiology study demonstrated inducible sustained monomorphic VT requiring subcutaneous implantable cardioverter-defibrillator implantation. This highlights the arrhythmogenic potential of residual scar after resolution of cardiac masses. (J Am Coll Cardiol Case Rep 2024;29:102369) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 48-year-old woman who presented to the emergency room was admitted because she reported experiencing dyspnea and palpitations. On physical examination, she was afebrile, her blood pressure was 127/82 mm Hg, her heart rate was 96 beats/min, and her oxygenation was 98% on room air. Cardiac examination revealed no appreciable murmurs with no jugular venous distension. There were normal breath sounds on auscultation.

LEARNING OBJECTIVES

- To recognize VT as a clinical presentation in patients with metastatic cardiac disease.
- To evaluate the utility of ICD placement as a mode for secondary prevention in adequately treated cancer patients who have experienced metastatic tumor-induced VT and who maintain good prognosis with life expectancy >1 year.

PAST MEDICAL HISTORY

The patient had a history of diffuse large B-cell lymphoma diagnosed 7 years earlier and treated with chemotherapy (RCHOP: rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone) and 6 cycles of everolimus. She experienced recurrence with metastasis to the right ventricle (RV) 5 years after the initial diagnosis, and she sustained monomorphic VT, necessitating cardioversion. She underwent autologous stem cell transplantation 6 months after identification of her cardiac metastases.

DIFFERENTIAL DIAGNOSIS

The differential diagnoses for her presenting dyspnea and palpitations included atrial or ventricular

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ABBREVIATIONS AND ACRONYMS

CMR = cardiac magnetic resonance

EPS = electrophysiology study

ICD = implantable cardioverter-defibrillator

RV = right ventricular

VT = ventricular tachycardia

arrhythmia, pulmonary embolism, systemic infection, pericardial effusion in the setting of malignancy, and volume depletion. Specifically, wide-complex tachycardia differential diagnoses would include VT and supraventricular tachycardia with aberrancy or pre-excitation.

INVESTIGATIONS

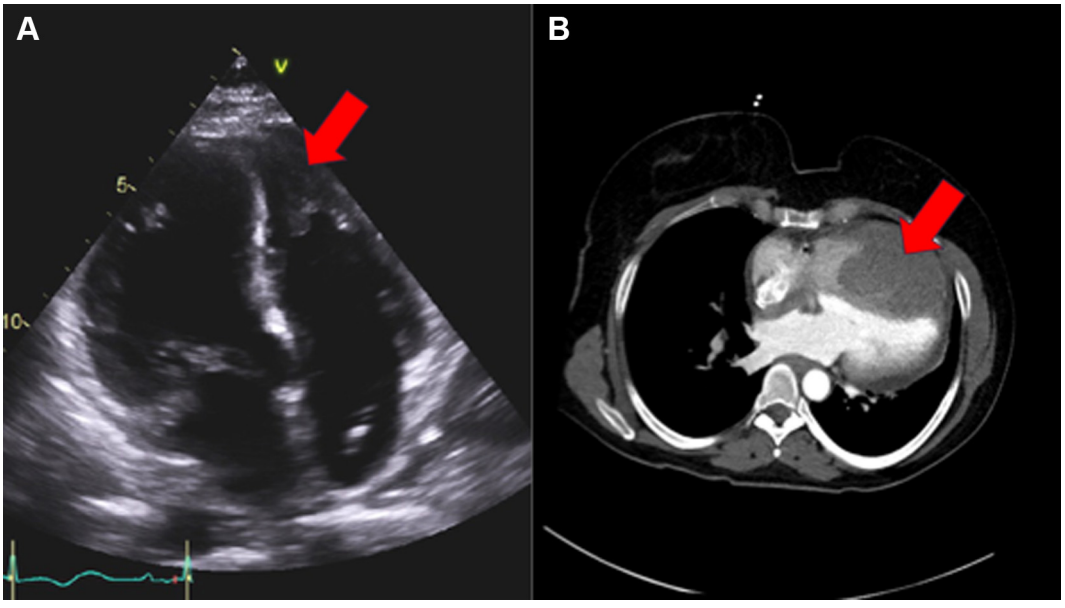
The initial electrocardiogram showed sinus rhythm with an inferior infarct pattern and anterolateral T-wave inversions. Computed tomography of the chest revealed a RV mass suggestive of metastasis, given her history of diffuse large B-cell lymphoma. An echocardiogram depicted an ejection fraction of 65% with no significant valvular disease or wall motion abnormalities and confirmed the presence of a mass in the RV apex (Figure 1). Biopsy of the mass and analysis of the biopsy specimen confirmed recurrent aggressive B-cell lymphoma. Cardiac magnetic resonance (CMR) further characterized the 8.4 × 9.2 × 7 cm RV mass. The patient was transferred to the intensive care unit for rituximab administration. During treatment, the patient had episodes of sustained monomorphic VT. Nonsustained VT with similar morphology was captured (Figure 2). She was started on an amiodarone infusion and converted to sinus rhythm. She was discharged with a Life-Vest, oral maintenance amiodarone, and metoprolol succinate with plans to undergo subsequent sessions of chemotherapy with RV mass surveillance. Two months later and after 1 cycle of RICE (fosfomycin, carboplatin, etoposide, and rituximab), repeated CMR showed a decrement in the RV mass size to 4.2 × 6.4 × 4.6 cm. Six months later, a surveillance CMR demonstrated complete resolution of the RV mass (Figure 3), with delayed enhancement involving the mid to apical RV inferior wall, overall reflecting treated disease with residual scar.

MANAGEMENT

The late gadolinium enhancement on CMR raised the question whether the residual myocardial scar after treatment of her malignancy had predisposed her to scar-mediated re-entrant VT and sudden cardiac death. Although she had not experienced VT recurrence since her RICE therapy in the index hospitalization, she was referred for an electrophysiology study (EPS) for risk stratification for VT recurrence.

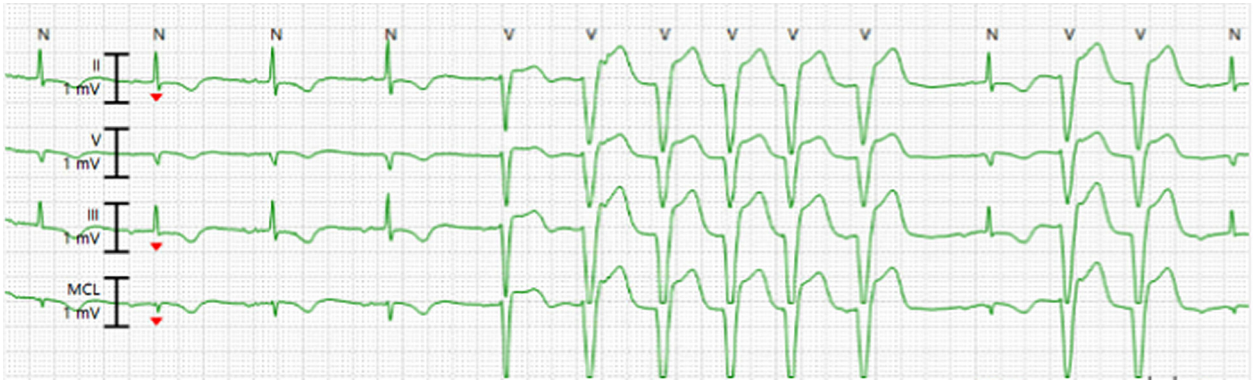
A complete EPS was performed. No VT was seen at a drive train of 600 milliseconds with several extrastimuli. At a drive train of 400 milliseconds, with

FIGURE 1 Initial Diagnostic Imaging



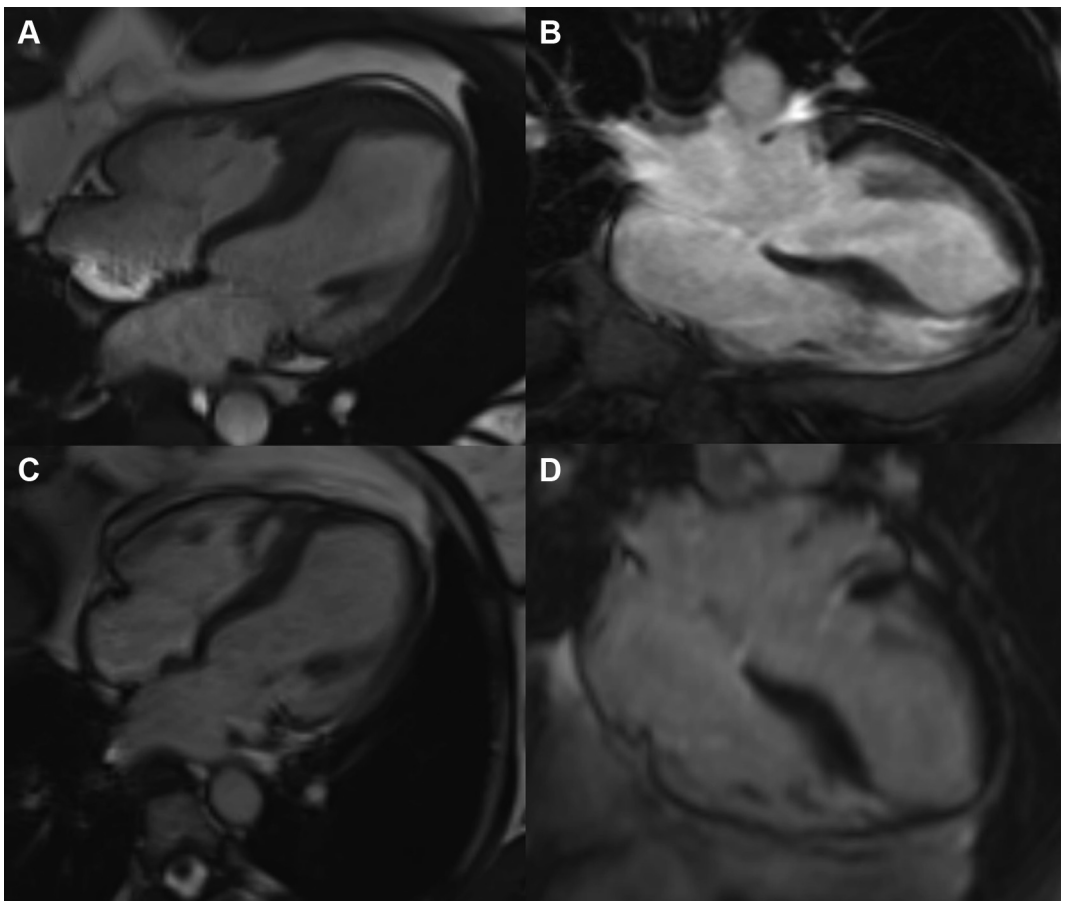
(A) Mayo Clinic format echocardiogram demonstrating right ventricular apical mass (arrow). (B) Computed tomography of chest: axial image demonstrating right ventricular apical mass (arrow).

FIGURE 2 **Telemetry Tracing**



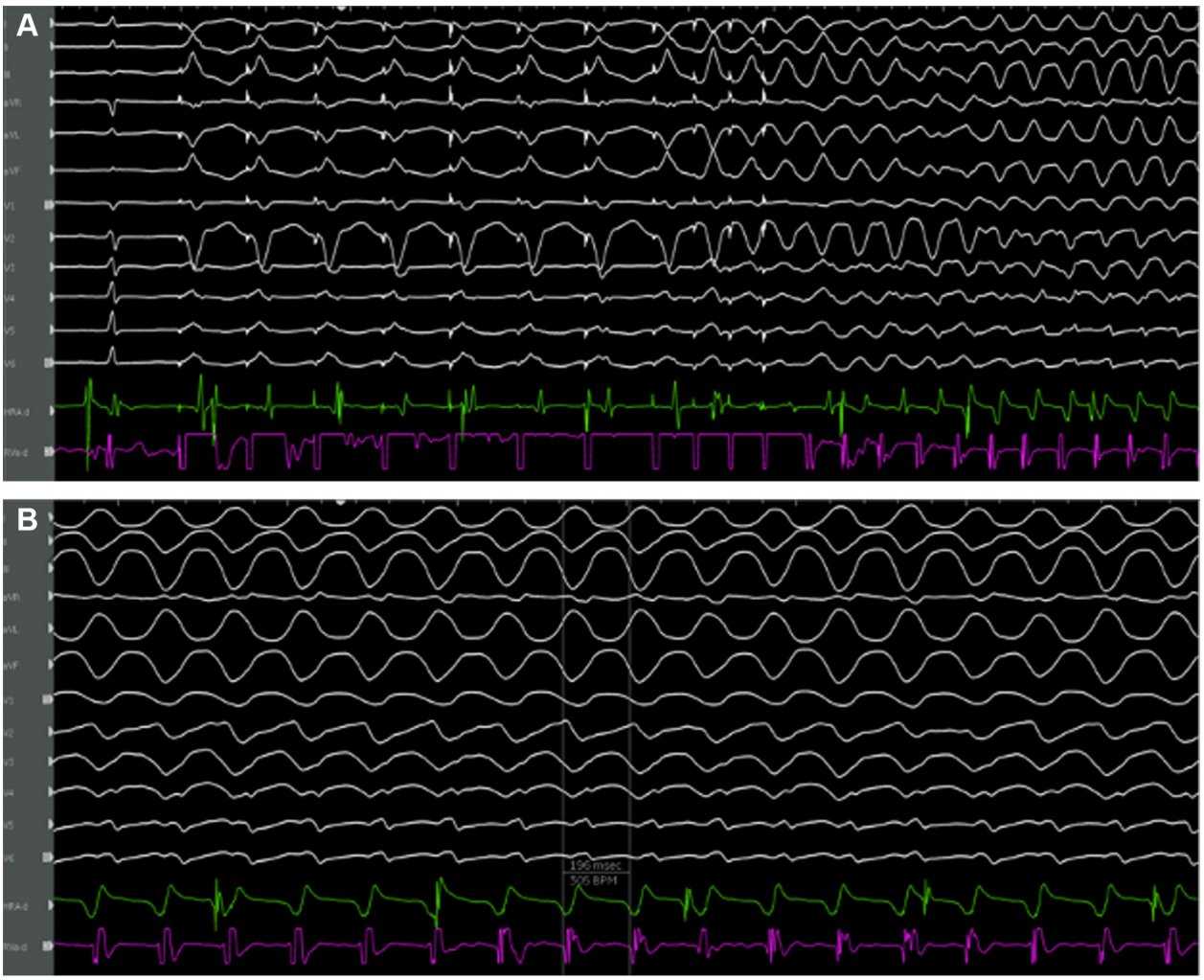
Strip showing a fused premature ventricular contraction triggering a nonsustained run of ventricular tachycardia with a left bundle, superior axis morphology.

FIGURE 3 **Cardiac Magnetic Resonance on Presentation and After Treatment**



(Left) At presentation; (right) after treatment. (A) Cine 4-chamber view of RV apical mass. (B) LGE in RV free wall and apical septum. (C) Cine 4-chamber view showing near resolution of RV apical mass. (D) LGE in the apical septum present, but less pronounced in the RV free wall. LGE = late gadolinium enhancement; RV = right ventricular.

FIGURE 4 Electrophysiologic Study



Induction of monomorphic VT with ventricular extrastimuli up to S4 of 240/210/200 milliseconds with a drive train of 400 milliseconds delivered from the RV base (outflow tract). No ventriculoatrial conduction is present. (A) The entire drive train is visualized before the tachycardia cycle length stabilizes. (B) Left bundle, left superior axis morphology with a precordial transition is seen in V_5, suggesting an exit close to the inferoapical septum consistent with the site of residual LGE. Additionally, there is wobble in cycle length (185-208 milliseconds) with ventriculoatrial dissociation. LGE = late gadolinium enhancement; RV = right ventricular; VT = ventricular tachycardia.

triple extrastimuli (240/210/200 milliseconds), an initially polymorphic VT was induced organizing into ventricular flutter. VT morphology was a left bundle, left superior axis (III > II) with negative concordance. There was also discordance in the leads aVR and aVL (aVL > aVR) consistent with a breakout in the inferior RV septum (Figure 4). The patient's condition became hemodynamically unstable, necessitating synchronized direct current cardioversion with 360 joules. Thus, a subcutaneous implantable cardioverter defibrillator (ICD) was placed, she was maintained on metoprolol tartrate to aid in suppressing VT recurrence, and she was discharged from the hospital.

DISCUSSION

Myocardial metastases are diagnosed with increasing frequency with the increased use of cross-sectional imaging modalities. The reported incidence ranges from 2% to 18%, the most frequent causes being lymphoma, melanoma, and lung cancer.[1,2] CMR is the preferred modality for detecting and characterizing

pericardial and myocardial infiltration by tumors with the use of late gadolinium enhancement to assess for scar that may heighten the risk of ventricular arrhythmia.[3] The utility of CMR in this setting can be extrapolated from the 2017 ACC/AHA/HRS guidelines, which give a class IIa recommendation for use in patients with nonischemic cardiomyopathy.[4]

The treatment of ventricular arrhythmias in this setting has frequently been limited to the use of antiarrhythmic agents, and surgical excision can be considered if the overall clinical picture and operative risk are acceptable.[5] The placement of ICDs in these patients remains controversial because the life expectancy in this population is cited at around 4 months at the time of metastatic disease identification.[6] To our knowledge, only 1 patient documented in the literature received an ICD for refractory ventricular arrhythmia despite optimal chemoradiation treatment of a primary cardiac lymphoma.[7] ICD implantation appears to be a reasonable option in patients who have completed treatment for primary lymphoma with evidence of tumor regression yet continue to have recurrent VT.

The mechanism of VT in patients with cardiac tumors is speculative. Although direct tumor invasion to the myocardium or conduction system could promote abnormal automaticity or triggered activity, scar forms the basis of re-entry.[8,9] EPS risk stratification in patients with nonischemic cardiomyopathy or infiltrative disease is a class IIa recommendation and could confirm the heightened risk in patients with significant residual scar demonstrated on CMR.[4] Our patient's long-term prognosis exceeded 1 year after successful treatment of her recurrent lymphoma; thus, the implantation of a subcutaneous ICD was warranted to reduce her risk of sudden cardiac death. The choice of subcutaneous implantation was driven by the advantages of low risk of systemic infections, low risk of vascular injury or pneumothorax, decreased risk of lead failure in an active younger patient, and no indication for pacing.[10]

FOLLOW-UP

Six months after ICD placement, the patient continued to take metoprolol tartrate for VT suppression, and subsequent interrogation showed no interval episodes of VT. Repeated CMR showed no evidence of progression or recurrence, inasmuch as no tracer-avid malignant lesion was detected.

CONCLUSIONS

Residual ventricular fibrosis despite metastatic heart disease remission may continue to carry an increased risk of malignant ventricular arrhythmias and sudden death. Such patients should be followed up meticulously, and risk stratified and treated accordingly. In this situation and despite a scarcity of evidence, there was a significant role for ICD placement.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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