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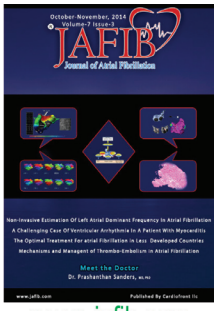

# A Challenging Case Of Ventricular Arrhythmia In A Patient With Myocarditis: ICD Yes/No After Ablation

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**Abstract**

In patients with myocarditis, early diagnosis and appropriate therapy are mandatory, as well as close clinical follow-up with particular regard to progression of disease and ventricular arrhythmia recurrences. The management of ventricular arrhythmias should follow current guidelines for ICD implantation, but new therapeutic options could be evaluated in these patients, such as combined epicardial/endocardial ablation and external wearable defibrillator. Particularly, depressed left ventricular ejection fraction (LVEF) represents the only risk marker for sudden cardiac death currently used in myocarditis, although the use of a single risk factor has limited utility. On this regard, combined analysis of myocardial tissue structure by cardiac magnetic resonance (CMR) and endomyocardial biopsy, in association with resting cardiac systolic function, could improve predictive accuracy for Sudden Cardiac Death (SCD) in patients with myocarditis.

## Introduction

The clinical presentation of myocarditis is heterogeneous, encompassing clinically silent conditions, acute coronary syndrome-like conditions, new-onset heart failure (HF) and life-threatening conditions such as cardiogenic shock, ventricular arrhythmias and SCD.[1-5] The mortality rate of acute myocarditis is 15%-20%.[6-8] A recent position statement of the ESC defined three different nosological entities: myocarditis, inflammatory and dilated cardiomyopathy.[9] Progression from myocarditis to dilated cardiomyopathy seems to occur predominantly in patients with histologically confirmed chronic inflammation,[10] but the specific rate of ventricular arrhythmic events in these three different presentations is unknown.

## Case Report

In October 2011, a 33 year-old man presented to our institution with palpitations arising while playing soccer. He had neither cardiovascular risk factors nor a family history of SCD. In 1995, he had suffered from acute pericarditis treated with non steroidal anti-inflammatory drugs and corticosteroids. On admission in the emergency room, physical examination revealed a heart rate of 183 bpm, normal blood pressure (125/70 mm Hg) and no symptoms of heart failure. A 12-lead electrocardiogram (ECG) showed a monomorphic ventricular tachycardia (VT) with right bundle branch block (RBBB) morphology and left axis deviation (Fig. 1). The VT could not be stopped by either intravenous lidocaine or amiodarone, and it was interrupted by electrical cardioversion (single 200J DC shock). Blood tests before DC shock revealed elevated high-sensitive troponin T levels (0.50 ng/mL, upper limit: 0.014 ng/mL). Echocardiography showed mild left ventricular (LV) systolic dysfunction (LVEF 45%) and dilation (end-diastolic volume: 140 mL) as well as LV wall motion abnormalities of the posterior-inferior and lateral walls. Coronary artery disease was ruled out by coronary angiography. Cardiac magnetic resonance (CMR) (Philips Achieva 1.5T, Eindhoven, NL) revealed mild LV dysfunction (LVEF=47%) and severe hypokinesia associated with circumferential subepicardial delayed enhancement (DE) as a result of myocardial-pericardial recurrent inflammatory involvement, more evident at the inferior basal LV wall, as well as intramyocardial DE of the septum (Fig. 2). Cardiotropic viral serology and autoantibody serum testing were negative.

Because of incessant monomorphic VT, refractory to multiple antiarrhythmic therapy (amiodarone, lidocaine, magnesium sulphate, beta blockers), the patient underwent electroanatomical mapping and radiofrequency catheter ablation with the CARTO-3 System (Biosense Webster Inc., Diamond Bar, CA, USA). Unipolar and bipolar LV endocardial mapping demonstrated the absence of scar tissue areas (voltage cut off 8 and 0.5 mV, respectively) (Fig. 3A, 3B). Clinical VT was induced and the activation map showed the VT exit site in the LV posterior wall (basal segment) and diastolic potentials in the LV inferior septum (basal segment). No late potentials were recorded. Endocardial ablation was performed during VT with

**Disclosures:**
None.

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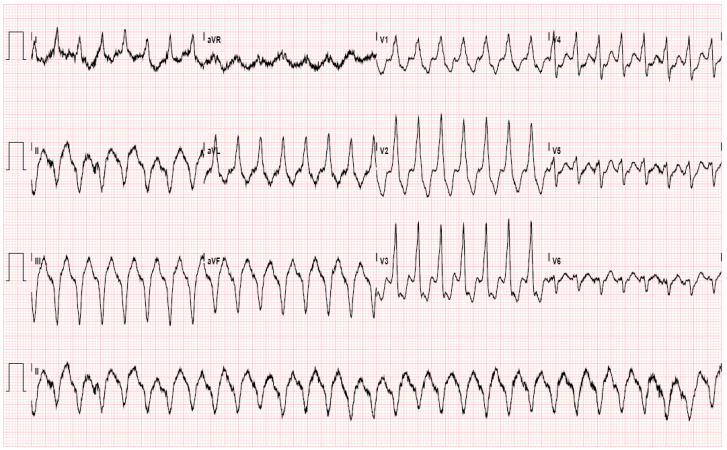

**Figure 1:** 12-lead ECG during ventricular tachycardia with superior axis and right bundle branch block morphology.

multiple consecutive RF pulses from the LV posterior basal wall to the basal interventricular septum, with repeated interruptions of the VT. No further VT or VF was inducible after ablation. LV endomyocardial biopsy (EMB) performed before ablation showed acute lymphocytic myocarditis (active myocarditis). Amplification of viral genome by real-time polymerase chain reaction was negative. Consequently, oral prednisone was added to metoprolol, ACE inhibitors and diuretics because of suspected autoimmune myocarditis.

At 3 and 6 months follow up, the ventricular ectopic burden on 24h-Holter monitoring progressively increased to 20% and 35%, respectively. In 2013, the patient was re-admitted because of sustained monomorphic VTs at the Holter ECG monitoring (ventricular rate 180 bpm, RBBB morphology) without haemodynamic instability or heart failure symptoms. A new CMR confirmed mild LV dilation (end-diastolic volume 139.3 mL/m$^2$; end-systolic volume= 81,7 mL/m$^2$) and demonstrated a further reduction in LV systolic function (LVEF=40%). An almost transmural extent of DE in the inferior basal wall, subepicardial DE in lateral wall, and a

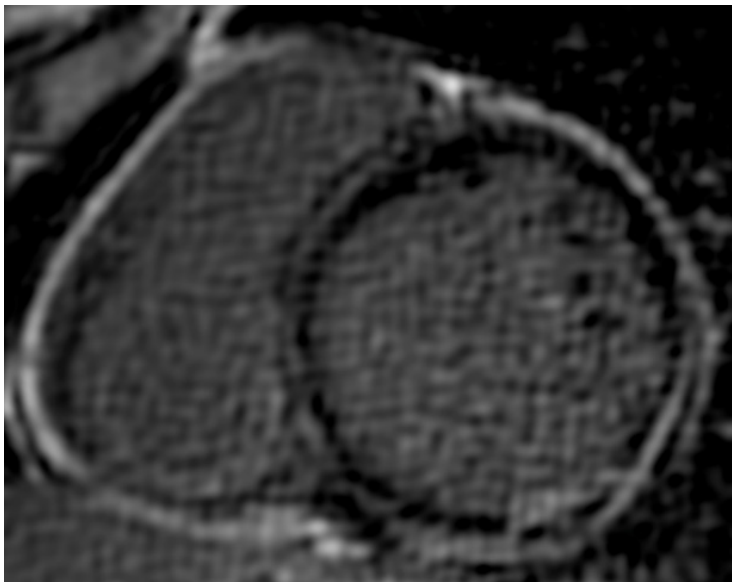

**Figure 2:** Cardiac MRI (2011) 2D Inversion Recovery Turbo Field Echo (IRTFE) image at the basis showing circumferential enhancement of the left ventricle and thick inferior subepicardial delayed enhancement (DE).

persistence of intramyocardial DE in the interventricular septum were found, suggesting worsening disease progression (Fig.4). The patient underwent endo-epicardial mapping and ablation of the arrhythmia. The absence of scar tissue areas was confirmed (Fig. 5A). Epicardial bipolar mapping was performed with a high-density mapping catheter (PentaRay NAV, Biosense Webster) inserted via a subxyphoid approach, with evidence of a scar area of 19.7 cm$^2$ (voltage cut-off, 1.0 mV) in the posterior wall (basal and medial segments) (Fig. 5B); an area of late potentials was detected in the posterior wall (from basal to apical segment) (Fig. 5C). Activation mapping of the inducible clinical VT showed early anticipation of the local electrogram to the surface QRS (48 ms) in the epicardial posterior basal wall, corresponding to the area of late potentials (Fig. 5D, 5E). After coronary angiography ruled out the course of coronary vessels within the target area, the ablation was performed in this area until late potentials were completely abolished and VT could no longer be reinduced. CARTO-guided left ventricular EMB was performed in the endocardial basal segment of the LV posterior wall (Fig. 6A, 6B) and showed diffuse interstitial fibrosis with focal oedema suggestive of scar; immunohystochemistry did not reveal lymphocyte infiltration (Fig.7A, 7B). Genotype testing was negative for cardiotropic viruses. The patient was discharged on beta-blockers without ICD, with an echocardiographic evidence of LVEF 49%. There was no recurrent VT during a 6-month follow-up period.

## Discussion

This case report highlights the importance of early aetiological diagnosis in patients with acute myocarditis and the need for a close follow-up in patients with active myocarditis and ventricular arrhythmias. The diagnosis of myocarditis should be obtained early, as indicated by the ESC statement on myocarditis, by integration of ECG Holter, myocardiocytolysis markers, CMR and echo evidence of functional and structural abnormalities and confirmed by EMB.[9] On this regard, the prognosis in myocarditis patients varies according to the underlying aetiology.[11, 12]

Myocarditis may cause sustained ventricular arrhythmias as its first clinical manifestation, both in its acute phase, due to inflammatory infiltration and myocyte necrosis, and in its chronic phase, due to immune reaction, fibrosis, and resulting ventricular electric remodelling. The case in this report could represent different stages of myocarditis (1995 pericarditis, 2011 acute active myocarditis

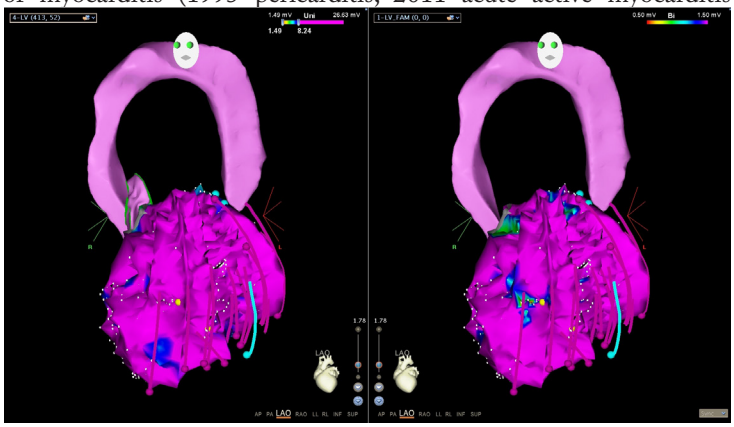

**Figure 3:** Endocardial left ventricular mapping before ablation (october 2011). Endocardial voltage unipolar (3A) and bipolar map (3B) of the left ventricle in left anterior oblique view. Impedance map (3C) of left ventricle in left anterior oblique view with geometry reconstruction guided by intracardiac echocardiography.

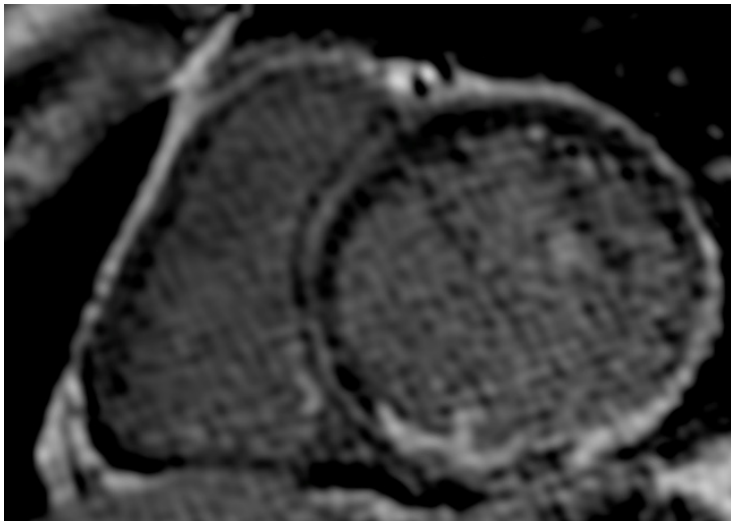

**Figure 4:** Cardiac MRI (2013) 2D Inversion Recovery Turbo Field Echo (IRTFE) image at the same level as Fig. 2. Clear circumferential DE is still detectable. In comparison to the 2011 image, the subepicardial DE involvement is increased, almost transmural, in the left ventricular inferior wall.

with VT, 2013 chronic myocarditis with VT recurrence). In the acute phase, as recommended by 2006 ESC/AHA guidelines on ventricular arrhythmias and SCD, treatment is usually largely supportive. Even in chronic myocarditis, therapy is confined mostly to anti¬arrhythmic drugs, with limited efficacy, and to implantable cardioverter-defibrillator (ICD) for higher-risk cases, such as those with haemodynamically unstable VT and aborted sudden death.[13, 14, 15, 16]

## ICD Implantation

The management of ventricular arrhythmias in these patients should follow current guidelines.[13, 17-21] Since acute myocarditis often represents a transient condition from which recovery is common, ICD implantation in this phase is not indicated.[9,13] Analysis of the IMAC-2 patient cohort emphasizes the dynamic nature of LV function in some patients newly-diagnosed non-ischaemic cardiomiopathy, as well as in patients with myocarditis.[22] In myocarditis complicated by VT or VF, antiarrhythmic drugs or ICD implantation have not yet been investigated in controlled trials.

To date, stratification for SCD mainly relies on LVEF, regardless of the underlying cardiac disease. Particularly, 2008 guidelines for Device–Based Therapy strongly recommend to rule out the reversible causes for transient LV dysfunction and to postpone ICD implantation after a period of optimal medical therapy. The 2013 appropriate use criteria for ICD therapy classified ICD therapy as appropriate in non-ischaemic cardiomyopathy after >3 months on guideline-directed therapy for LVEF≤40% and NYHA Class I-III symptoms.[23] According to the recent HRS/ACC/AHA expert consensus statement, ICD implantation for primary prevention between 3 and 9 months can be useful in selected patients with non-ischemic cardiomyopathy who are unlikely to recover LV function.[21] Patients with giant cell myocarditis might benefit from ICD implantation during this period, as this drug-refractory myocarditis presents with a virulent course.[24]

Numerous investigations proved that a reduced LVEF significantly increases the risk of SCD.[16,25,26,27] However, LVEF as a standalone risk stratification marker has major limitations, particularly considering that: (i) the majority of SCD cases occur in patients with preserved or moderately reduced LVEF, (ii) relatively few patients with reduced LVEF will benefit from an ICD (most will never experience a life-threatening arrhythmic event, others have a high risk for non-sudden death), (iii) a reduced LVEF is a risk factor for both sudden and non-sudden death, (iv) patients with potentially reversible cause of cardiomyopathy such as myocarditis were not enrolled in clinical trials.[21, 28, 29] Immunohystological evidence of inflammation without the presence of viral genome in endomyocardial specimens, as in our case, was an independent predictor of survival.[10] Cardiac magnetic resonance has become an established diagnostic tool for acute myocarditis and recent papers demonstrated that late gadolinium enhancement (LGE) is associated with adverse outcome in patients with acute myocarditis[6, 7, 8] Particularly, in a subgroup of patients with more severe myocardial involvement, LGE can be used as a prognostic tool for all-cause and cardiac mortality.[8] On the other hand, patients with a diagnosis of myocarditis who do not have heart failure on admission would have a low risk for cardiovascular events, as suggested in a recent paper by De Stefano et al.[6] These data need to be confirmed in multicentre studies. The indication for ICD remains controversial, because acute myocarditis may heal completely. Our patient was not implanted with an ICD in the acute phase of

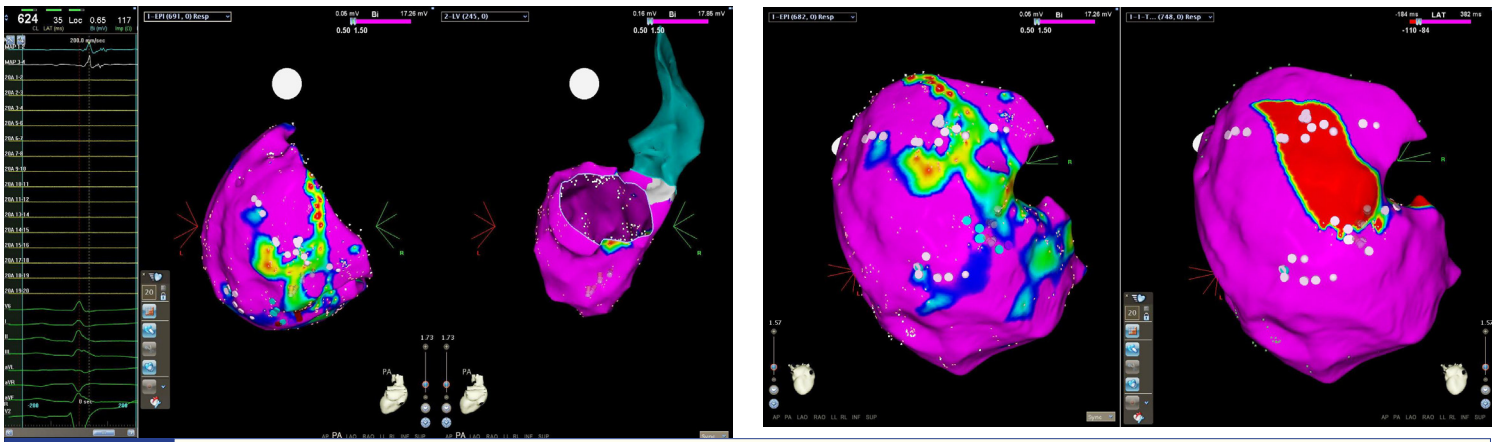

**Figure 5:** Endocardial/epicardial mapping before ablation (October 2013). Endocardial bipolar voltage (5A) and epicardial bipolar voltage (5B) of the left ventricle in posterior-anterior view; late potentials (5C) during mapping in epicardial posterior region of the left ventricle in posterior view (pink points); epicardial activation map (5D) with "early meets late" in basal posterior region of left ventricle synchronized with epicardial bipolar map of the same region, including late potentials (pink points) (5E).

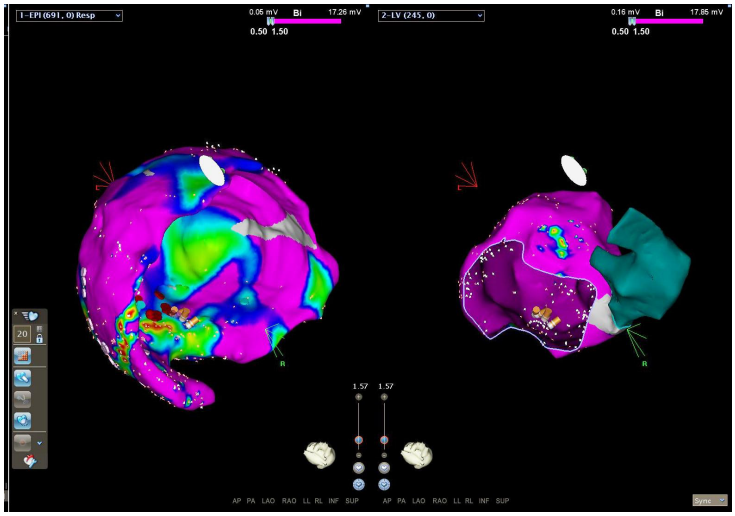

**Figure 6:** CARTO guided endomyocardial biopsy before ablation (October 2013). Left ventricular endocardial biopsy was guided by electroanatomic bipolar map (Carto System) in basal segment of LV posterior wall (tip of bioptome localized in "boderzone area" with voltage ranging between 0.5-1 mV) (6A). The endocardial map was synchronized with epicardial map (6B) (site of ablation).

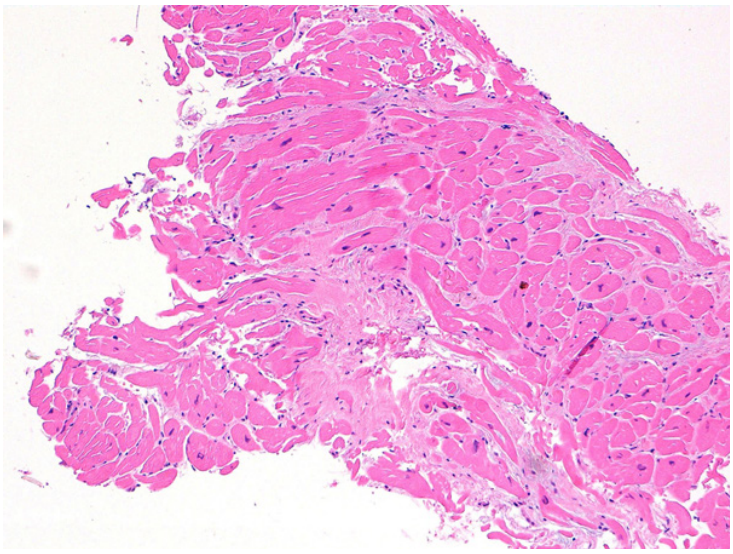

**Figure 7:** Endomyocardial biopsy. Haematoxylin and eosin staining (A,B). At histology endomyocardial biopsy showed myocardium with no evidence of lymphocyte infiltration (A). However, the presence of focal considerable interstitial fibrosis was found (B).

myocarditis (October 2011) due to the evidence of LVEF>40% and because his VT was hemodynamically well-tolerated; in the chronic phase of myocarditis (October 2013), we decided to perform epicardial ablation and to delay ICD implantation because of improved LV systolic function. Bridging with a wearable defibrillator in patients with myocarditis and severe ventricular arrhythmias could solve the transient problem, mostly in patients with previous history of myocarditis complicated by arrhythmias and normal LVEF. There is a single case report study by Prchanau and colleagues documenting the successful use of the Life Vest (Zoll) defibrillator during a post-myocarditis cardiac arrest caused by VF in a young woman with myocarditis, ventricular arrhythmias and preserved LVEF.[31]

**Radiofrequency Catheter Ablation**

In patients with myocarditis, radiofrequency catheter ablation of drug-refractory VT has been demonstrated to be feasible, safe, and effective and this therapeutic option is mentioned in the context of ESC guidelines.[13, 32, 33] Dello Russo et al. found that endocardial ablation was acutely successful in 70% of patients, while in the remaining 30% clinical VT was successfully ablated by epicardial approach.[32] Consequently, epicardial ablation should be considered as an important therapeutic option to increase the ablation success rate. Maccabelli et al. recently supported this evidence indicating a first-line epicardial ablation approach in myocarditis patients.[33] In the short term follow-up, after this combined approach, 77% of patients remained free of VT recurrences at a median follow up period of 23 months.

**Follow Up**

Acute myocarditis recovers in about 50% of cases, however about 25% will develop persistent cardiac dysfunction over time and 12-25% may acutely turn into dilated cardiomyopathy.[1-3, 12] Progression from myocarditis to dilated cardiomyopathy seems to occur predominantly in patients with chronic myocardial inflammation.[10] For this reason, these patients should be closely followed-up with transthoracic echocardiography and Holter ECG or loop recorders. Cardiac magnetic resonance might gain its weight in the prognostic stratification of myocarditis, even in the case of a normal LV function, by detecting life-threatening arrhythmic substrate during the course of the disease. On this regard, Vermes et al. demonstrated that in patients with clinically suspected acute myocarditis, the presence of positive Lake Louise criteria is associated with recovery of LV function.[34] Myocardial oedema as defined by CMR was the strongest parameter, indicating that the observed increase in LVEF may be due to the recovery of reversibly injured oedematous myocardium. Jerish et al. reported spontaneous improvement of LVEF detected by CMR after acute or subacute viral myocarditis.[35] Consequently, clinical follow-up should be associated necessarily with early access to an experienced CMR centre, especially in case of suspected disease progression.

## Conclusion:

In patients with acute myocarditis, early diagnosis and specific therapy are mandatory, as well as close follow-up with particular attention to disease progression and arrhythmia relapse. Cardiac magnetic resonance should be regarded as a potentially leading tool in the risk stratification process after myocarditis, due to its ability to characterize tissue structure. The wearable defibrillator could represent a valuable approach to provide temporary protection from sudden arrhythmic death as a bridge to recovery.

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