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Management of patients with myocarditis and arrhythmogenic phenotype

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Acute myocarditis (AM) is an inflammatory condition of the myocardium that may lead to severe complications, including acute heart failure and life-threatening ventricular arrhythmias (VAs). In-hospital VAs are estimated to affect 2.5% of adult patients with AM. Recent insights suggest a genetic predisposition to develop VA in a subset of patients with AM. This review will focus on arrhythmogenic manifestations of AM, highlighting risk stratification for VA after an acute episode and the contribution of genetic factors, emphasizing the need to integrate clinical, imaging, and genetic findings. In addition, prognostic information derived from cardiac magnetic resonance imaging will be discussed, pointing out the association between VA and the presence, extension, and septal localization of late gadolinium enhancement. The overlap between inherited arrhythmogenic and inflammatory cardiomyopathies will be explored, with specific attention to the identification of desmosomal gene variants, which are associated with recurrent myocarditis-like episodes and a higher risk of VA. Cardiac sarcoidosis, giant cell myocarditis, and immune checkpoint inhibitors-related myocarditis will be discussed as a paradigm of inflammatory cardiomyopathies with increased arrhythmic burden. Finally, the clinical challenges of managing patients with AM and arrhythmogenic presentation will be tackled, looking at indications for implantable cardioverter defibrillators after the acute phase.

Introduction

Acute myocarditis (AM) is an inflammatory condition of the myocardium and the cardiac conduction system that may lead to severe complications, including acute heart failure and life-threatening ventricular arrhythmias (VAs). It can result from infections, immune activation, or exposure to toxic substances, with recent reports suggesting a genetic predisposition.[1] Ventricular arrhythmias, including ventricular tachycardia (VT), ventricular fibrillation (VF), and sudden cardiac death (SCD), are among the most serious complications of AM. The epidemiological burden of VAs in individuals with AM may be underestimated, as myocarditis is a significant yet often unrecognized cause of SCD, particularly in young individuals, where post-mortem examinations attribute 6–10% of SCD cases to AM, with VAs presumed to be the primary mechanism.[1] Among 27 129 myocarditis hospitalizations, cardiac arrest and VF were observed in ∼2.5% of adult patients based on a Nationwide Inpatient Sample database that analysed the primary discharge diagnosis of myocarditis in the United States between 2007 and 2014.[2] In-hospital VA prevalence can rise sharply to 47% in specific clinical scenarios, such as patients presenting with cardiogenic shock, like in fulminant myocarditis.[3] Specific subtypes of AM carry a higher risk of VAs, independently of their initial clinical presentation, such as giant cell myocarditis (GCM), cardiac sarcoidosis (CS), and immune

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checkpoint inhibitor (ICI)-associated myocarditis.[1] A genetic predisposition can further amplify the risk of VAs, contributing to electrical instability both during the acute inflammatory phase and long-term follow-up.[4,5] The identification of numerous clinical markers increased our ability to predict patients at risk of VA events after an acute episode, even if it must be considered that the VA risk after an episode of AM is low.[6] For instance, in a multicentre retrospective study that assessed 248 patients with AM confirmed by histology or cardiac magnetic resonance imaging (CMRI), 7 (2.8%) patients had VA episodes (1 SCD, 1 aborted SCD, and 5 sustained VT episodes) after a median follow-up of 4.7 years.[6]

Risk stratification and predictors of events of ventricular arrhythmia after the acute phase

Patients initially presenting with life-threatening VAs are at higher risk of VA events after the acute phase compared with the general population of patients with AM.[7] An international registry including 156 patients presenting with VA (median age 44 years; 77% male; histologically proven diagnosis in 62.8% of cases) who were discharged showed a recurrence rate of VAs of 37.2% during follow-up. The median time from discharge to the recurrence was 8 months, and 60.3% of VAs occurred within the first year after the initial episode of AM.[7] The presence of sustained VT (in contrast to VF) was a marker of risk for arrhythmic recurrence [hazard ratio (HR) of 2.90], in combination with other two CMRI parameters: late gadolinium enhancement (LGE) involving ≥2 myocardial segments (HR of 4.51) and the absence of positive short tau inversion recovery (STIR) signals, which means lack of myocardial oedema (HR 2.59).[7] The combinations of these three factors provided a reliable predictive model with an area under the curve of 0.80.[7] Counterintuitively, the absence of oedema, a CMRI hallmark of myocarditis, emerged as a predictor of future VA. The lack of oedema could suggest that the episode that led to the initial hospitalization was primarily an arrhythmic event with troponin release in the setting of an inherited cardiomyopathy with extensive myocardial scar instead of a proper myocarditis with significant myocardial inflammation. Even if it must be recognized that STIR sequences have substantial limitations in detecting inflammation and T2-mapping sequences that can increase the ability to identify myocardial inflammation was rarely used in the 156 patients of this registry. Thus, it could be that myocarditis with extensive scarring and low levels of inflammation can be more prone to arrhythmias. In the 2022 European Society of Cardiology (ESC) Guidelines for the management of patients with VAs and the prevention of SCD, the experts recommend considering an implantable cardioverter defibrillator (ICD) before hospital discharge in patients experiencing haemodynamically unstable VT or VF during the acute phase of myocarditis (Class IIa, Level of Evidence C).[8] Recent studies analysing VA risk in patients with AM and possible event predictors summarized in *Table 1* support the guidelines' indications.[7,9] Nevertheless, it should be noted that VF/VT can occur in patients with primarily a severe pump failure (fulminant myocarditis) requiring temporary mechanical circulatory supports (t-MCS). In this specific setting, it could be that VAs are consequences of the cardiogenic shock and exposure to high doses of catecholamines and should be differentiated by forms where primarily VAs caused haemodynamic instability. In patients with haemodynamically not-tolerated sustained monomorphic VT or tolerated sustained monomorphic VT occurring in the chronic phase of myocarditis, ICD implantation is recommended with Class I and IIa, respectively (level of evidence C).[8] In particular, in a study including 56 patients with biopsy-proven myocarditis who underwent ICD implantation for primary (57%) or secondary (43%) prevention, the presence of sustained VT on admission (HR 13) was identified as the main predictor of VA recurrence treated by ICD intervention (in 45% of cases after a median time of 5.4 years), while the extension of fibrosis detected by low potentials at the bipolar endocardial mapping had a significantly independent HR of 1.2.[10] Data on the benefit of the wearable cardioverter defibrillator for primary prevention of SCD early after AM are sparse, and no recommendations can be currently made.[8]

Role of genetics

Estimates of patients with an identifiable genetic background in AM vary widely, ranging from 3% to 31%,[1,4,5] mainly due to differences in inclusion criteria.[5] Likely, the methodologically most sound perspective study identified myopathic cardiac gene variants in ∼3.1% of consecutive cases in a cohort of 230 patients with AM, all diagnosed by CMRI [median age of 33 years, 84% men and median left ventricular (LV) ejection fraction (EF) of 63%] compared with 0.4% of a control group.[5] This finding was driven by *DSP* (gene encoding for desmoplakin) truncating variants in patients with normal LVEF and VA.[5] Another multicentre study on 36 patients (median age of 24 years, male 67.7%, median LVEF of 59%) with AM and pathogenic or likely pathogenic desmosomal gene variants (DGVs) showed that *DSP* was the gene involved in 88.9% of cases.[4] In this study, VT/VF occurred in 5 (13.9%) patients with DGV after a median follow-up of 3.6 years, with an estimated incidence rate of VAs of 3.59 per 100 patient-years, conversely among 25 patients with AM and a negative genetic test for DGV no VA occurred after a median follow up of 2.2 years.[4] Pathogenic of likely pathogenic DGV and septal LGE on CMRI were associated with the main study endpoint that included risk of death, VAs, recurrent myocarditis, and heart failure with an HR of 4.2 and 3.3, respectively. In contrast, LVEF < 50% on CMRI was not associated with the main endpoint.[4] These findings emphasize the importance of genetic testing in managing patients with AM and inflammatory cardiomyopathies aimed to avoid SCD. Key red flags to identify potential patients with positive genetic testing include a family history of AM, cardiomyopathies or SCD, personal recurrent episodes of AM, VA or high burden of premature ventricular complexes (PVCs) on non-sustained VT at initial presentation, septal LGE or ring-like LGE on first CMRI, persistent troponin release and high PVC burden on ECG ambulatory monitoring beyond the acute phase.[4,5] Diffuse low voltages of the QRS on the initial ECG,

Table 1 Selected studies investigating ventricular arrhythmic risk patients with in acute myocarditis.

Study	Characteristics	N.	Population	Endpoint	Predictors of events
Rav-Acha et al.,[9] JACC Clin Electrophysiol, 2024 PMID: 38661603	Multicentre, retrospective. Median FU 5.5 years.	69	AM verified by CMRI or EMB with VA at presentation	Long-term recurrence of SVA or death	Pre-discharge LVEF < 50%, initial sustained monomorphic VT
Cannatà et al., Front Card Med, 2022 PMID: 36312271	Single-centre, retrospective. Median FU 53 months	199	AM confirmed with CMRI	Composite of all-cause mortality, resuscitated SCD, appropriate ICD therapy	Arrhythmias at presentation (AV block, SVA, or aborted SCD)
Gentile et al.,[7] Eur J Heart Fail, 2021 PMID: 34196079	International, multicentre, retrospective. Median FU 23 months	156	AM verified by CMRI or EMB with life threatening VA at presentation	SCD or successfully treated VF or SVT	SVT at presentation, LGE involving ≥ 2 segments or negative STIR at first CMRI
Casella et al., Heart Rhytm, 2021 PMID: 33348060	Single-centre, retrospective. Median FU 2 years	144	Prior myocarditis and VA at presentation	VA recurrence	Presence of anteroseptal scar identified at CMRI and electroanatomic mapping
Peretto et al., J Clin Med, 2021 PMID: 34768662	Single-centre, prospective. Median FU 3.7 years	104	AM verified by EMB with VA at presentation	VA recurrence	Anteroseptal LGE and 'chronically active' myocarditis at EMB (presence of fibrosis, myocyte hypertrophy, nuclear atypia)
Sasko et al., Cardiology, 2021 PMID: 33550300	Single-centre retrospective. Median FU 4.7 years	51	AM verified by CMRI and EMB and life-threatening VA	VA recurrence with appropriate ICD intervention	No predictors identified
Pelargonio et al.,[10] JACC Clin Electrophysiol, 2020 PMID: 32439044	Single-centre retrospective. Median FU 74 months	56	AM verified by EMB and ICD implantation before discharge	Appropriate ICD intervention	SVT on admission and the extension of the areas of low potentials at electroanatomic mapping
Rosier et al., J Clin Med, 2020 PMID: 32244983	Single-centre retrospective. Median FU 3 years	68	AM verified by CMRI or history of AM and evidence of LGE at CMRI and ICD implantation in secondary prevention	Appropriate ICD intervention on VT or VF	SVA during the acute phase

AM, acute myocarditis; AV, atrio-ventricular; CMRI, cardiac magnetic resonance imaging; EMB, endomyocardial biopsy; FU, follow-up; LGE, late gadolinium enhancement; LVEF, left ventricular ejection fraction; ICD, implantable cardioverter defibrillator; SCD, sudden cardiac death; STIR, short tau inversion recovery; SVT, sustained ventricular arrhythmias; SVT, sustained ventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia.

especially on the limb leads, can be another finding that, among patients with AM and preserved LVEF, could suggest an extensive LGE pattern and the presence of a genetic background (*Figure 1*).

Inflammatory features in arrhythmogenic cardiomyopathy—hot phases vs. acute myocarditis

Inflammation is increasingly recognized as a key feature of inherited cardiomyopathies, including arrhythmogenic cardiomyopathies (ACM). However, its role in the pathophysiology of these conditions remains uncertain, as it may represent either a heightened response to minor inflammatory stimuli or an integral component of the disease process itself. Pathological studies consistently document inflammatory infiltrates along with the characteristic fibrofatty replacement of the myocardium in patients with ACM.[5] The presence of inflammatory cells and necrosis, often correlated with more severe structural abnormalities, suggests a contributory role of inflammation in ACM development. Most cases of ACM are linked to pathogenic or likely pathogenic variants in proteins of the cardiac intercalated disc, particularly desmosomal proteins. Notably, 4–15% of ACM patients experience *hot phases* of recurrent myocarditis-like episodes characterized by chest pain, elevated troponin levels, and imaging evidence of myocardial inflammation resembling AM.[5,11] The true prevalence of myocardial injury in ACM is likely underestimated due to frequent misdiagnosis, and *DSP* mutations are disproportionately associated with myocarditis-like episodes. Among 107 patients with

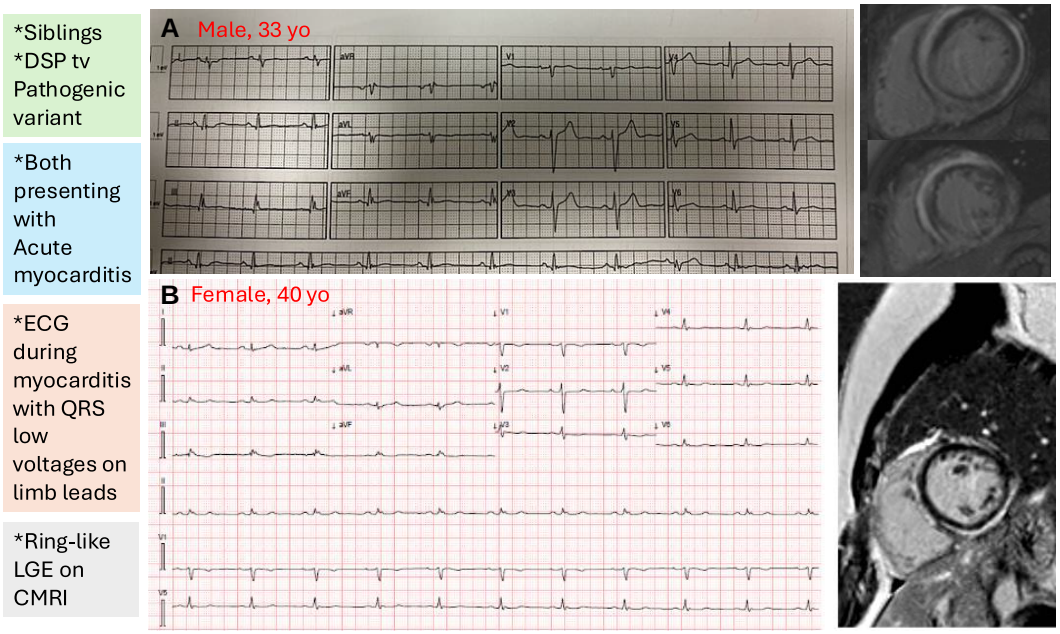

Figure 1 Two siblings with DSP truncating variants with acute myocarditis. ECG on admission is characterized by low QRS voltages on limb leads and diffuse late gadolinium enhancement on cardiac magnetic resonance imaging. Both experience ventricular arrhythmias. The patient in (A) had a cardiac arrest due to ventricular fibrillation during the initial hospitalization for acute myocarditis. His sister (B) had a haemodynamically stable sustained ventricular tachycardia treated by an implantable defibrillator that was placed in primary prevention.

DSP-related cardiomyopathies, episodes of myocardial injuries documented by troponin elevation were reported in 15% of cases, while among 81 patients with *PKP2*-related cardiomyopathy, no episodes of myocardial injuries were observed.[11] In both groups of patients, independent of the type of ACM (*DSP* or *PKP2*), VTs were frequently reported (28% of patients with *DSP*-related cardiomyopathy and 30% of patients with *PKP2*-cardiomyopathy).[11] Thus, it remains to be fully established whether inflammatory episodes increase the risk of VA. Among patients with *DSP*-related cardiomyopathy in the presence of LVEF < 45% and the LGE on CMRI, an ICD implantation could be considered as primary prevention of SCD based on the 2023 ESC guidelines for the management of cardiomyopathies (IIa, level of evidence C).[12] It must be recognized that differentiation between a hot phase of an ACM and CS can be challenging because both can present with frequent VAs and extensive LGE patterns on CMRI. Finally, it must be acknowledged that the term *hot phase* in patients with known inherited cardiomyopathy or *AM with a genetic background* can be seen as equivalent terms. However, the latter remarks that myocarditis comes first in individuals in whom cardiomyopathy is not previously identified.

Cardiac magnetic resonance imaging

Septal or anteroseptal scar identified by LGE on CMRI and the risk of VAs after myocarditis have been confirmed by independent studies.[6,9] Clinical presentation and CMRI-derived markers have emerged as robust prognostic tools for patient stratification after AM.[1,6] In a study including 69 patients with AM and initial VA (mean age of 44 years, 67% male) with a median follow-up of 5.5 years, 39% experienced a VA (24 cases) or death (1 case), and initial monomorphic VT (HR of 5.17), pre-discharge LV systolic dysfunction on echocardiogram (HR of 4.57) and anteroseptal LGE on CMRI were significantly associated with the endpoint. A significant correlation was found between initial sustained monomorphic VT and the presence of anteroseptal LGE on CMRI.[9] In another abovementioned study, including 248 patients with AM, the presence of septal LGE (HR of 9.2) emerged together with reduced LVEF (<50%) on CMRI (HR of 12.4) as good predictors of major cardiac events (composite of cardiac death, heart transplantation, aborted SCD, sustained VA, and HF hospitalization). Septal LGE on initial CMRI was observed in 28.6% of patients with AM, while LVEF < 50% was observed in 12% of cases.[6] Nevertheless, the complicated clinical presentation by LV systolic dysfunction, or VA on admission, or fulminant presentation was a key factor in identifying patients with cardiac events even after discharge with an HR of 35.8. Uncomplicated clinical presentation preserved LVEF and no LGE septal involvement on initial CMRI-identified patients without events in a 4.7-year follow-up.[6] As previously mentioned, other markers of VA risk based on CMRI are no oedema based on STIR sequences and LGE involving ≥2 myocardial segments.[7,10]

Specific inflammatory cardiomyopathies with increased ventricular arrhythmia burden

Specific subtypes of AM are associated with a heightened risk of VA. Among patients with CS, the three primary cardiac manifestations are LV systolic dysfunction, atrioventricular conduction abnormalities, and VAs. Complete atrioventricular block typically develops

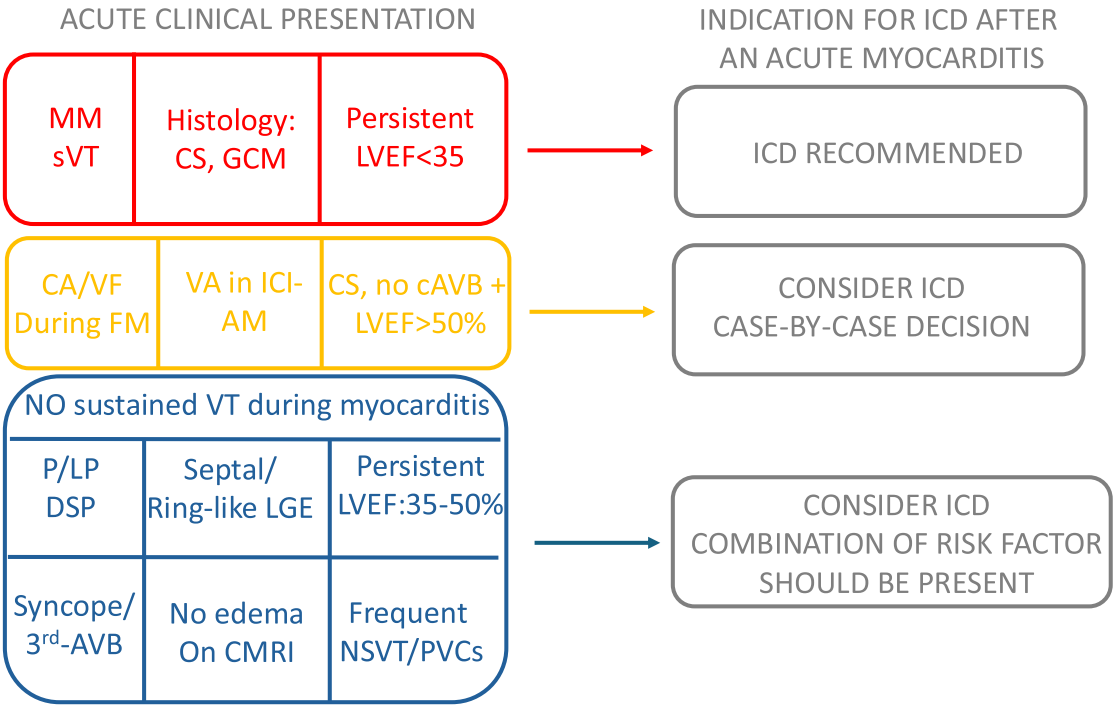

Figure 2 When consider an implantable cardioverter defibrillator after an episode of acute myocarditis. AM, acute myocarditis; AVB, atrio-ventricular block; CA, cardiac arrest; CMRI, cardiac magnetic resonance imaging; CS, cardiac sarcoidosis; FM, fulminant myocarditis; GCM, giant cell myocarditis; LGE, late gadolinium enhancement; LVEF, left ventricular ejection fraction; ICI, immune checkpoint inhibitors; MM, monomorphic; NSVT, non-sustained ventricular tachycardia; P/LP, pathogenic/likely pathogenic; PVC, premature ventricular complexes; sVT, sustained ventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia.

during the acute inflammatory phase, whereas sustained VT more often arises in the advanced stages of the disease. Studies estimate that ~60% of patients with CS experience arrhythmic events, including VT and VF. Moreover, research indicates that 16–35% of patients under 60 years of age presenting with complete atrioventricular block or unexplained VAs are later diagnosed with previously unrecognized CS.[13] Independent predictors of VAs in CS include LVEF <35%, high-degree atrio-ventricular (AV) block, and right ventricular or LV scarring. International guidelines recommend ICD implantation for primary prevention not only in patients with CS and an LVEF ≤ 35% (Class I, Level B) but also in patients with an indication for permanent cardiac pacing (Class IIa, Level C), significant myocardial scarring on CMRI after acute inflammation has resolved (Class IIa, Level B), or a LVEF between 35% and 50% with inducible sustained monomorphic VT during programmed electrical stimulation.[8] Corticosteroid therapy remains the mainstay of treatment for CS, while methotrexate is often employed as a second-line agent.[13]

Similarly, ICI-AM is frequently marked by life-threatening VAs, with severe arrhythmias reported in up to 15% of cases in a series that included 147 patients, where sustained VT occurred in 10.9%, VF in 2.7% and torsade de pointes in 1.4%.[14] Complete heart block and life-threatening VAs co-occurred in 11/147 (7.5%). Patients who experience a VA as a complication of ICI-AM had a 30-day mortality of 55% compared to 22% in patients with ICI-AM without VAs. Corticosteroids were the treatment of choice in 118 (80.2%) out of 147 cases of ICI-AM. Immunosuppression is considered effective in treating this type of myocarditis, even if mortality remains high.[1] Indication for ICD as secondary prevention after AM in this specific population remains debateable, and a case-by-case decision is reasonable based on life expectancy and progression of cancer. In this registry, only 14% of patients who survived after an ICI-AM complicated by VAs received an ICD.[14] GCM represents a rapidly progressive and necrotizing form of myocarditis with a poor prognosis, including an ~85% rate of death or heart transplantation within 3 years.[1] A study that specifically analysed the risk of SCD and VAs in 51 patients with GCM showed a 41% risk of SCD or VAs at 1 year and 55% at 5 years. Among 31 patients who were implanted with an intra-cardiac ICD, 17 (55%) received 117 appropriate antiarrhythmic therapies by the device without SCD.[15] Immunosuppressive therapy, initiated promptly, is crucial for improving outcomes. Anti-T-lymphocyte-based therapies (e.g. anti-thymocyte globulin) and calcineurin inhibitors have shown efficacy in achieving clinical remission; nevertheless, based on these data, most patients discharged after a GCM receive an ICD.

Conclusions

Managing VA risk in patients with AM requires a personalized approach that takes into consideration clinical, imaging, genetic, and histologic data (*Figure 2*). Despite advances in diagnostic tools and risk stratification, significant gaps remain in translating these

insights into standardized approaches. Future research should focus on defining risk prediction models by integrating emerging evidence and genotype-specific knowledge. Moreover, prospective studies are needed to evaluate the efficacy of targeted immunomodulating therapies in reducing arrhythmic burden in myocarditis patients. Most indications for ICD implantation are based on expert opinion. Further data are required from large registries to identify patients that can benefit most, especially among patients with AM and preserved LVEF with a genetic background.

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Data availability

No new data were generated or analysed in support of this research.

Disclaimer

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