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Acute Myocarditis and Inflammatory Cardiomyopathies: Insights From Cardiac Magnetic Resonance Findings

Francesco Lauriero[1] | Camilla Vittoria Vita[2] | Alessio Perazzolo[2] | Giovanni Sanseverino[2] | Eleonora Moliterno[2] | Giuseppe Rovere[1] | Riccardo Marano[1,2] | Luigi Natale[1,2]

[1]Department of Diagnostic Imaging, Oncological Radiotherapy and Hematology, Fondazione Policlinico Universitario Agostino Gemelli IRCCS, Rome, Italy | [2]Department of Radiological and Haematological Sciences–Section of Radiology, Università Cattolica del Sacro Cuore, Rome, Italy

Correspondence: Francesco Lauriero (francesco.lauriero@guest.policlinicogemelli.it)

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ABSTRACT

Myocardial inflammation encompasses a broad spectrum of conditions, including acute myocarditis, chronic inflammatory cardiomyopathy, and several overlapping entities that differ in clinical presentation, pathophysiology, and progression. These conditions range from self-limiting acute inflammation to chronic myocardial injury and dysfunction. The etiologic classification of myocardial inflammation highlights the complexity of its pathogenesis, involving direct tissue damage, immune-mediated mechanisms, and environmental triggers. Cardiac magnetic resonance (CMR) imaging has become a central diagnostic tool in the assessment of myocardial inflammation, providing precise characterization of myocardial tissue, assessing cardiac function, and stratifying prognosis. Advanced techniques such as T1 and T2 mapping and extracellular volume quantification have further expanded its diagnostic capabilities. This review highlights the essential role of CMR in diagnosing myocardial inflammation, recognizing various imaging findings associated with different underlying causes, and informing clinical management. The standardization of CMR protocols, along with advancements in imaging techniques and strengthened interdisciplinary collaboration, represents a fundamental step toward improving diagnostic accuracy, patient outcomes, and the understanding of the broad spectrum of myocardial inflammatory diseases.

1 | Introduction

Acute myocarditis (AM) and chronic inflammatory cardiomyopathy (I-CMP) represent two distinct conditions along the spectrum of myocardial inflammation, differing in timing, clinical presentation, and pathophysiology. AM refers to the diffuse or focal inflammatory infiltration of the myocardium, typically triggered by a direct pathogenic insult or an overactive immune response [1]. It usually manifests within the first month of symptom onset and is often characterized by elevated high-sensitivity troponin levels and evidence of myocardial edema on cardiac magnetic resonance (CMR) imaging.

In contrast, I-CMP is a histologic and functional diagnosis defined by hypokinetic cardiomyopathy, which may present in either dilated or nondilated forms [2]. This persistent condition, lasting more than 1 month, may result from the progression of one or more episodes of AM—whether diagnosed or

Abbreviations: ADs, auto-immune diseases; AM, acute myocarditis; CMR, cardiac magnetic resonance; CTDs, connective tissue diseases; CTRCD, cancer treatment–related cardiac dysfunction; DCM, dilated cardiomyopathy; ECV, extracellular volume fraction; EGE, early gadolinium enhancement; EM, eosinophilic myocarditis; EMB, endomyocardial biopsy; FT, feature tracking; GLS, global longitudinal strain; ICIs, immune checkpoint inhibitors; I-CMP, chronic inflammatory cardiomyopathy; IR, inversion recovery; LGE, late gadolinium enhancement; LLC, Lake-Louise criteria; LV, left ventricle; MACE, major adverse cardiac event; NDLVC, nondilated left ventricle cardiomyopathy; RV, right ventricle; RWMA, regional wall motion abnormalities; STIR, short tau inversion recovery.

TABLE 1 | Spectrum of myocardial inflammation categorized by timing, key features, and histological findings, highlighting transitions from acute to chronic stages.

	Fulminant Myocarditis	Acute Myocarditis	Subacute Myocarditis	Chronic Myocarditis	Chronic Inflammatory Cardiomyopathy
Timing	Rapid onset		Intermediate (1-3 months)	Transitional	Persistent (>1 month)
Key Features	Rapid progression with severe hemodynamic compromise (low-output syndrome, cardiogenic shock) Often requires inotropic or MCS support High mortality risk	High-sensitivity troponin levels Evidence of edema on CMR performed within four weeks, or positive FDG-PET imaging	Intermediate phase between acute myocarditis and chronic inflammatory cardiomyopathy May represent a healing phase of inflammatory response	Ongoing inflammation without active necrosis or myocyte abnormalities Non-dilated or mildly dilated cardiomyopathy phenotype	Clinical phenotype of hypokinetic either dilated or non-DCM that can be associated with arrhythmogenic substrate
Histology	Diffuse inflammatory infiltrates +++ Necrosis ++ Large multinuclear cells infiltrate (GCM)	Inflammatory infiltrate++ Fibrosis +/- Necrosis ++	Inflammatory infiltrate +/- Fibrosis +/- Necrosis +/-	Inflammatory infiltrate +/- Fibrosis ++ Necrosis - Myocyte abnormalities - -	Inflammatory infiltrate/- Focal/diffuse fibrosis ++ Necrosis - - Myocyte abnormalities ++

missed—that have resulted in myocardial damage and systolic dysfunction [3].

Table 1 categorizes myocardial inflammation based on a temporal framework that includes AM, I-CMP, and other distinct entities. These additional conditions represent intermediate stages or variations of myocardial inflammation with varying degrees of severity. These overlapping conditions underscore the complexity of myocardial inflammation and the importance of tailored diagnostic and therapeutic approaches.

Myocarditis can be broadly classified into the following two main categories based on etiology: infectious and noninfectious (Figure 1). The noninfectious group broadly includes autoimmune disease (AD), toxic, and genetic forms; among these, viral-mediated heart damage is by far the most common cause of myocarditis [1].

The clinical presentation of inflammatory myocardial involvement is highly heterogeneous as the symptoms can range from transient ECG changes to severe, life-threatening conditions such as cardiogenic shock and ventricular arrhythmias [4]. Prompt recognition of AM and I-CMP is important as it can impact patient management and outcome and its diagnosis is based on a combination of clinical and diagnostic criteria [1, 5].

Endomyocardial biopsy (EMB) is still considered the gold standard for definitive diagnosis of myocarditis but has limitations due to the sampling of endocardial and subendocardial layer of the myocardium, usually not involved in myocarditis, sampling errors, and variability in sample interpretations [6]. Additionally, the invasive nature of EMB and its low sensitivity compared with cardiac explant at autopsy reduce its employment in clinical practice [1]. Despite these challenges, EMB should be strongly considered in cases of AM presenting with cardiogenic shock (i.e., fulminant myocarditis), evidence of high-grade AV block, or malignant ventricular arrythmia [5, 7]. According to two recent international expert consensus documents, EMB is also indicated in several other settings: suspected immune checkpoint inhibitor (ICI)–induced myocarditis; I-CMP with persistent or relapsing release of myocardial necrosis markers, particularly when associated with suspected or known auto-immune disorders; AM or I-CMP associated with peripheral eosinophilia; dilated cardiomyopathy (DCM) with recent-onset HF that is refractory to standard treatment [3, 8, 9]. Given the limitations of EMB, a noninvasive approach, especially with CMR, is increasingly being used to diagnose myocarditis or guide the EMB [10]. Imaging findings can also aid in identifying other potential diagnosis with similar clinical presentations, such as coronary syndrome or stress-induced cardiomyopathy [11]. However, EMB remains essential when CMR lacks sufficient diagnostic accuracy to guide treatment or differentiate pathological processes. It is particularly indicated when CMR findings indicate myocardial inflammation but cannot establish specific etiologies such as eosinophilic myocarditis (EM), sarcoidosis, or giant cell myocarditis [3, 12–14].

American Heart Association (AHA) recommends one or more cardiac imaging techniques for the assessment of patients with suspicion of myocardial inflammation.

Among these, transthoracic echocardiography remains the most widely used due to its accessibility, affordability, and utility in

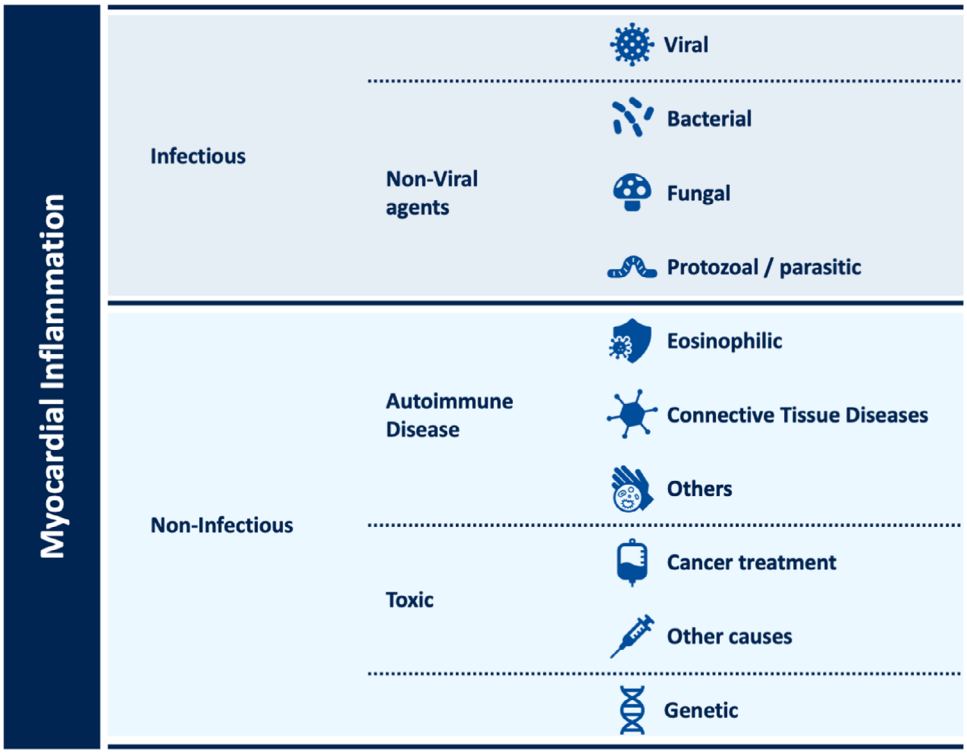


FIGURE 1 | Etiology of myocardial inflammation.

assessing cardiac function and structure [12]. Positron emission tomography (PET/CT) provides valuable insights into inflammatory processes, enabling a more precise identification of patients who may benefit from further diagnostic procedures; while PET/CT offers important information for risk stratification, its use is limited by radiation exposure and the risk of false positive [15, 16].

CMR, however, stands out as the preferred imaging tool in this context, thanks to its unique capacity for noninvasive, radiation-free tissue characterization.

2 | CMR for Myocardial Inflammation Assessment

CMR has emerged as a noninvasive gold-standard method for the diagnosis of myocardial inflammatory involvement [17]. It offers unique advantages, including high sensitivity for detecting myocardial inflammation, especially when performed within 2–3 weeks of symptom onset, as there is well-documented evidence that edema tends to decline 4 weeks after disease onset [3, 18]. By providing a detailed assessment of myocardial structures and functions, CMR serves as a fundamental diagnostic modality for differentiating myocarditis from other causes of myocardial injury, particularly in patients with nonobstructive coronary artery disease. While it is highly sensitive for infarct-like presentations of myocarditis, its sensitivity is lower for cardiomyopathy-like and arrhythmia presentations [19]. Additionally, the implementation of high-sensitivity troponins has provided a valuable adjunct to CMR, enhancing the noninvasive diagnosis of AM and enabling the detection of low-risk cases [20].

Although CMR has a limited diagnostic accuracy in identifying the specific etiology of myocardial inflammation, it offers non-invasive imaging that can accurately assess myocardial inflammation and is now considered the first-line modality to confirm suspected inflammatory myocardial disease for uncomplicated cases with preserved or mildly reduced left ventricular ejection fraction (LVEF) [3].

CMR also plays a fundamental role in therapy monitoring, although there is a lack of data on the optimal timing for follow-up CMR to assess therapeutic response in myocardial inflammation. A follow-up MRI at 3–6 months following the diagnosis is recommended to evaluate for ongoing inflammation and to assess the extent of late gadolinium enhancement (LGE). If LGE does not resolve in the long term, it is indicative of scar [3, 21]. The essential role of CMR in detecting myocardial inflammatory involvement is also highlighted in the recent proposed "Stages of Myocarditis" from the expert consensus of the American College of Cardiology, which also identifies future challenges, such as the detection of myocardial inflammatory involvement in asymptomatic patients [22].

Despite its advantages, CMR has some limitations, including difficulties performing scans on patients with hemodynamic instability or subjects under mechanical ventilation, complicating its use in acute heart failure scenarios [20].

2.1 | The Diagnostic Value of the Updated Lake Louise Criteria in Myocarditis

CMR findings of myocardial inflammation are commonly evaluated using the **Lake-Louise Criteria (LLC)**. Initially established

in 2009, the original LLC targeted myocardial inflammation by detecting edema on T2-weighted images and identifying hyperemia or necrosis through early gadolinium enhancement (EGE) and LGE [23]. While these criteria showed good specificity (87%), their sensitivity was limited (80%), particularly for diffuse inflammation and across different CMR setups [24].

In 2018, the LLC was updated to overcome these limitations, incorporating T1 and T2 mapping for enhanced tissue characterization, significantly improving the sensitivity compared to the original LLC, while maintaining a consistently high specificity [25]. This revision removed the EGE requirement, instead requiring at least one T1-based criterion -increased myocardial T1 relaxation times, extracellular volume fraction (ECV), or LGE- alongside one T2-based criterion -increased myocardial T2 relaxation times, visible myocardial edema, or increased T2 signal intensity ratio [20]. Standalone T1-based criteria showed a sensitivity of 90.0% and specificity of 76.9%, while T2-based criteria showed a sensitivity of 84.6% and specificity of 88.5%. When combined, these criteria provided even greater diagnostic accuracy, underscoring their added value [25]. The inclusion of T1 and T2 mapping also broadened the utility of the LLC by enabling noncontrast imaging and reducing susceptibility to artifacts, which was a common limitation of earlier techniques . Notably, certain combinations, such as T2 mapping with LGE, achieved excellent diagnostic performance (90%) in detecting acute myocardial inflammation, while T1 mapping combined with LGE yielded even higher accuracy (96%). Even the presence of only one criterion may support the diagnosis of myocardial inflammation when considered in the appropriate clinical context, though with lower specificity [11].

The updated Society of Cardiovascular Magnetic Resonance (SCMR) standard protocol for the assessment of myocarditis is shown in Table 2 [26].

2.2 | T2-Based Criteria

Tissue edema is a hallmark of inflammation that is often focal in the setting of myocarditis, although diffuse edema can also be identified.

T2-weighted short tau inversion recovery (STIR) sequence is particularly useful in detecting edema, with high diagnostic accuracy for focal edema. The limitations of the STIR sequence, such as artifacts from prolonged acquisition times and low signal-to-noise ratios, reduce its accuracy [27]. To mitigate these limitations, some studies have proposed normalizing myocardial signal intensity to skeletal muscle, identifying edema when the T2 signal intensity ratio is equal to or greater than 2.0 [28]. However, this approach may be less sensitive in cases of associated myositis [29].

T2 mapping sequences overcome these limitations by providing quantitative, reproducible T2 relaxation times for each voxel, producing a color-coded map that detects both focal and global myocardial edema with higher sensitivity.

T2 mapping offers substantial advantages in differentiating between acute inflammatory conditions and chronic myocardial damage, as it is highly sensitive and specific for detecting edema [30]. Additionally, it is strongly correlated with the extent of myocardial injury and prognosis, as persistent elevation of T2 values beyond the acute phase can indicate ongoing inflammation and predict adverse outcomes such as heart failure and major cardiac events (MACEs) [31]. However, to guarantee the reliability and consistency of the results, it is crucial to implement standardization across imaging protocols and equipment.

2.3 | T1-Based Criteria

LGE is an essential criterion for detecting myocardial injury by leveraging differences in T1 relaxation properties of tissues following gadolinium administration. Gadolinium accumulates in the interstitial space of areas affected by injury, necrosis, or fibrosis, while it rapidly clears from healthy myocardial tissue. This accumulation results in high signal intensity on inversion-recovery (IR) images, enabling clinicians to distinguish damaged tissue from unaffected myocardium.

LGE is a robust, independent predictor of both cardiac and all-cause mortality in patients with myocarditis, particularly in the post-acute phase when T2 has reached a stable state [32]. The pattern of LGE in patients with myocarditis is most commonly subepicardial or mid-wall and often in a linear configuration. The basal inferolateral wall is the most commonly affected region for LGE in viral myocarditis. Other frequently involved areas include the basal anterior septum, mid inferolateral wall, and the basal-to-mid inferior wall. Transmural enhancement and more widespread LGE have been observed, especially in severe cases such as fulminant and giant cell myocarditis. Specific patterns, such as mid-wall septal enhancement, have been demonstrated to be particularly associated with an increased risk of MACE and poorer patient outcomes [33].

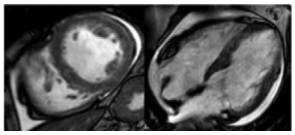
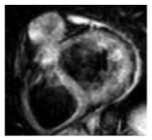
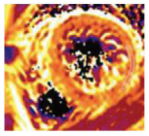
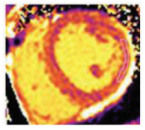
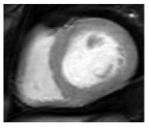
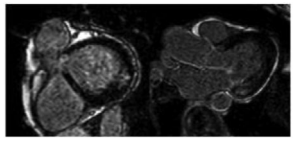
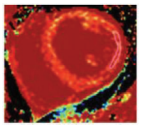
The LGE sequence has certain drawbacks, including its vulnerability to artifacts, challenges in detecting diffuse fibrosis, and its inability to differentiate between acute and chronic myocardial injury, which has led to the adoption of complementary techniques such as mapping sequences for enhanced assessment of myocardial inflammation and fibrosis [27].

Native T1 mapping measures the intrinsic myocardial T1 relaxation time before contrast administration, allowing quantitative assessment of myocardial tissue properties and detecting pathophysiological changes without contrast agents [34].

Its role in identifying myocardial inflammation is linked to its ability to reveal a complex interplay of intracellular and extracellular edema, hyperemia, capillary leakage, and myocyte necrosis [27]; however, elevated T1 values can also indicate fibrotic regions where extracellular space expansion occurs due to chronic myocardial damage. This overlap between active inflammation and fibrosis underscores the need for complementary T2-weighted imaging for clearer interpretation.

ECV, calculated by comparing myocardial and blood-pool T1 values pre- and post-contrast (adjusted for hematocrit), provides additional diagnostic insight; however, its accuracy may be limited during early disease phases, where intracellular edema

TABLE 2 | Updated 2020 SCMR standard protocol for myocarditis.

CMR imaging protocol- tissue changes during myocardial inflammation					
CMR Sequences			**Planes**	**Evaluation/ Detection**	**Examples**
Cine-SSFP			SA and long axis	Myocardial dysfunction	
T2w		T2w imaging	SA	Myocardial edema	
		T2 mapping	SA	Myocardial edema	
T1w	Pre-contrast	Native T1 mapping	SA	Myocardial edema, necrosis and fibrosis	
	Post-contrast	EGE (optional)	SA	Hyperemia (increased vascular permeability, capillary leak)	
		LGE	SA and long axis	Myocardial necrosis and fibrosis	
		Enhanced T1 mapping/ ECV	SA	Myocardial edema, necrosis and fibrosis	

predominates over interstitial edema, potentially keeping ECV within normal ranges [35].

A significant advantage of T1 mapping and ECV over conventional LGE is its sensitivity to diffuse interstitial fibrosis, where LGE is less effective. Native T1 mapping also avoids potential adverse effects related to gadolinium-based agents, making it particularly useful for patients with contraindications to contrast agents [35].

Elevated T1 and ECV values are negative prognostic markers in myocarditis, linked to increased myocardial damage and adverse cardiac outcomes [36].

Nonetheless, interpreting native T1 and ECV values remains challenging due to factors such as age, sex, imaging sequences, and magnetic field strength or different scanners and vendors. Thus, standardization of imaging protocols and acquisition techniques is critical to enhance the reproducibility and reliability of T1 mapping outcomes.

2.4 | Additional Findings

Although not required for diagnosis under the revised LLC, additional CMR criteria for myocardial inflammatory involvement can provide relevant diagnostic and prognostic insights.

In severe cases, **global left ventricular (LV) dysfunction** and regional wall motion abnormalities (RWMA) can be identified at CMR. Patients with fulminant myocarditis typically present with a markedly reduced LVEF at the time of admission that usually improves rapidly during hospitalization but remains lower than in nonfulminant cases at long-term follow-up [37]. Global systolic dysfunction (LVEF < 40%) serves as a critical prognostic marker, with persistent dysfunction linked to higher

mortality and rehospitalization rates [36]. Functional recovery largely occurs in the first weeks, emphasizing the urgency of therapeutic interventions.

Myocardial strain quantifies the deformation of the heart muscles detecting changes in the measurement of the tissue between relaxed and contracted states [38]. The most widely used CMR technique in strain measurements is feature tracking (FT). FT has emerged as a promising tool for evaluating myocardial function in myocarditis and inflammatory cardiomyopathies; however, its routine application in clinical practice is still limited [20]. Recent studies highlight impaired strain rates even in patients with a preserved EF, with GLS holding prognostic value as strongly correlating with worse outcomes, including MACE [39, 40].

Pericardial inflammation is a notable feature in certain cases of myocarditis. CMR plays a key role in assessing pericardial involvement, with findings such as pericardial effusion, thickening, high T1 or T2 values on mapping sequences and LGE, helping to differentiate inflammatory causes from other cardiac conditions. When present, concomitant pericarditis is most observed involving the pericardium adjacent to areas of inflamed myocardium, although it can also be diffuse. The presence of pericardial effusion or thickening may indicate a more severe form of the inflammatory process, which is often seen in fulminant myocarditis [37].

3 | Pathogenesis of Myocardial Inflammatory Involvement and Its Correlation With CMR Findings

The pathogenesis and progression of AM and I-CMP to symptomatic heart failure is thought to involve a multiphase process driven by a complex interplay of genetic, epigenetic, auto-immune, and environmental factors. The immune-mediated response plays a recognized and extensive role as a key driver of myocardial involvement; indeed, inflammation—regardless of the primary etiology that triggered it (infectious, auto-immune, drug-related, genetic, etc.)—can create a self-perpetuating "vicious cycle," leading to progressive myocardial damage [3, 13]. In acute myocardial inflammatory involvement, evidence suggests both direct myocardial damage from causative agents (predominantly of viral origin) and indirect auto-immune mechanisms; its CMR correlates are represented by the T2 and T1 criteria included in the LLC [20]. These processes can occur in individuals with or without genetic predisposition, presenting as familial or sporadic cases, respectively [1, 41]. The indirect auto-immune mechanism of damage can be triggered by infectious and noninfectious etiologic agents, including in the context of systemic ADs [42]. Most patients recover completely after AM; however, some may develop localized fibrotic scarring or diffuse fibrotic replacement (respectively, evidence of subepicardial or mid-wall LGE and a diffuse increase in native T1 mapping and ECV values).

In a minority of patients, the inflammatory response may persist or recur, with or without persistent direct damage and involving an auto-immune mechanism, leading to a chronic inflammatory process with progressive development of fibrotic scarring and diffuse fibrotic replacement:

TABLE 3 | Most common viruses associated with myocardial inflammation.

Viral Tropism	Virus
Cardiotropic	Adenovirus Enterovirus (coxsackieviruses, echoviruses)
Vasculotropic	Parvovirus V19
Lymphotropic	Cytomegalovirus; Epstein-Barr virus; Human herpesvirus 6
Cardiotoxic	Hepatic C Virus; Influenza viruses
ACE2-tropic	Coronaviruses (MERS-CoV, SARS-CoV, SARS-CoV-2)

- if the inflammatory process persists, CMR will continue to show edema on T2-weighted sequences;
- in cases of recurrent inflammatory episodes, follow-up CMR scans may reveal fluctuations in signs of cardiac edema, with possible alternation between normal and pathological T2-weighted intensity and T2 mapping values.

More frequently, in chronic inflammatory processes, there may be a possible progression toward DCM or, alternatively, a nondilated left ventricle cardiomyopathy (NDLVC) [11, 43, 44].

Graphical abstract illustrates the key concepts of the correlation between the pathogenesis of myocardial inflammation and the CMR findings.

4 | CMR Findings in the Different Categories of Myocardial Inflammatory Involvement

CMR findings show substantial overlap between different causes of myocardial inflammatory involvement, making it essential to consider clinical features in order to achieve an accurate diagnosis.

4.1 | Infectious Myocardial Inflammation

4.1.1 | Viral Agents

Viruses are the most common cause of myocardial inflammatory involvement [2, 45]; despite the high prevalence of viral causes, viral serology is not routinely recommended due to its low diagnostic yield, leaving the etiology of myocarditis often classified as idiopathic [1]. Regarding the pathogenesis of cardiac damage, a clear classification should distinguish viruses that directly infiltrate cardiac tissue (such as cardiotropic and vasculotropic viruses) or indirectly affect it through lymphotropic activity, from viruses that do not necessarily infect cardiac cells but may cause cardiac injury and impaired contractility by triggering a cytokine storm or an immune response via molecular mimicry [2, 42]. The most common viruses associated with myocardial inflammatory involvement are divided into five categories, as shown in Table 3 [2].

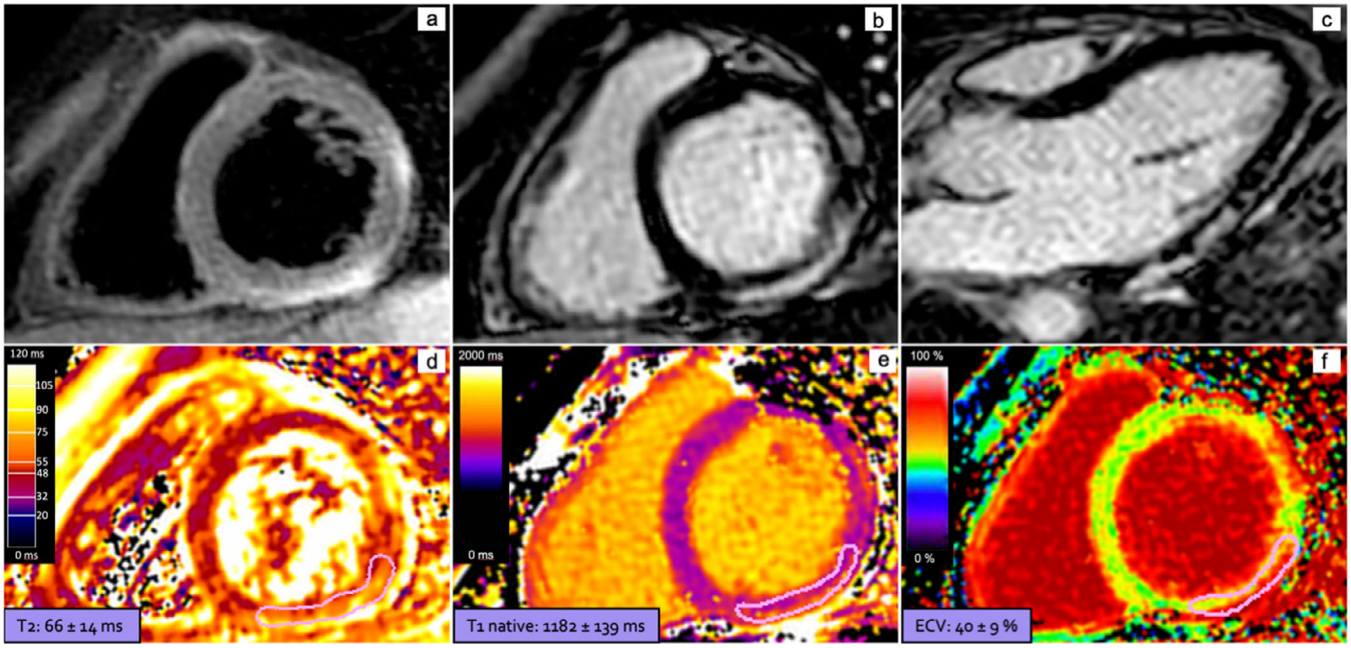


FIGURE 2 | Acute myocarditis in a 21-year-old patient presenting with acute chest pain and fever. CMR shows focal edema (a, STIR image) and subepicardial LGE in the LV basal inferolateral and inferior segments (b), (c) corresponding to high myocardial native T1, ECV, and T2 values (d)–(f). CMR, cardiac magnetic resonance; ECV, extracellular volume; LGE, late gadolinium enhancement; LV, left ventricular; STIR, short tau inversion recovery.

CMR identified the presence of edema associated with active inflammation through high T1 and T2 values on mapping sequences or increased signal intensity on T2-weighted sequences. This edema is typically distributed from the epicardium to the mid-wall. In contrast, LGE imaging frequently demonstrates enhancement in these edematous areas, typically sparing the subendocardium, which is associated with acute necrotic tissue. Additionally, evidence of concomitant inflammatory processes within the pericardium may be observed, manifesting as pericardial thickening and enhancement on LGE imaging, and occasionally as increased signal intensity on T2-weighted sequences. The segments most commonly affected in post-viral myocarditis are the basal to mid-septum and, most frequently, the basal to mid-lateral walls (Figure 2). The reasons for this pattern remain unclear but may reflect the direct spread of infection from the overlying pericardium through associated lymphatics. Indeed, the lateral wall is less likely to be surrounded by extensive epicardial fat and has closer contact with the pericardium above [13, 20]. Myocardial involvement has the potential to result in either regional or global LV dysfunction. Nevertheless, despite significant tissue damage, there may be only a limited effect on cardiac contractility, as the endocardial myocytes, which are essential for normal ventricular function, are relatively preserved [13]. As edema subsides and necrotic cells are cleared and replaced by collagen or fibrous tissue, the injured area gradually diminishes [46].

While the short-term prognosis of AM is generally favorable, an estimated 10%–20% of adults go on to develop complications, including I-CMP, DCM, congestive heart failure, and sudden cardiac death [47]. The most common viruses involved in myocardial injury are adenoviruses and enteroviruses, which are cytolytic and lead to complete recovery without residual injury in about 50% of patients with virus-induced myocarditis, resulting in healed myocarditis [48]. However, persistent viral presence or an ongoing auto-immune response in the myocardium can lead to I-CMP [2, 49].

As with other forms of viral myocarditis, the pathophysiology of SARS-CoV-2-related myocarditis is thought to comprise a combination of direct viral damage, cytokine release, and cardiac injury resulting from the host's immune response [50]. Recent studies indicate a relatively high incidence of elevated T2 values, suggesting myocardial edema, in patients who have recovered from symptomatic COVID-19; this may be related to the inhibition of the ACE2 receptor by SARS-CoV-2, as ACE2 plays a role in regulating vascular permeability during acute injury [51, 52]. The development of this myocardial edema could be mistakenly classified as myocardial inflammation. Additional long-term imaging studies with clinical outcomes are necessary to thoroughly evaluate the relationship between COVID-19 and myocarditis and to determine their clinical significance.

Although no definite causal relationship has been established, **vaccine-related** myopericarditis has been reported sporadically following some vaccinations. A recent data analysis in the United States, covering all vaccinations licensed for use from 1990 to 2018, found that only 0.1% of cases with physician-diagnosed myopericarditis were related to vaccine [53]. Recently, several cases have been reported as adverse events associated with vaccination with novel messenger ribonucleic acid (mRNA) COVID-19 vaccines, with a probable auto-immune/hypersensitivity mechanism [54, 55]; a worldwide analysis reported a rate of 9.23 cases per 100 000 person-years of myocarditis/myopericarditis diagnosed concerning mRNA COVID-19 vaccines, with the highest observed rate in males aged 18–24 years, at 53.76 per 100 000 person-years [56].

A recent retrospective cohort study investigated the differences in CMR findings between vaccine-associated myocarditis and COVID-19 or other causes of myocarditis; the study found a higher LVEF and less extensive LGE in patients with vaccine-associated myocarditis, even after controlling for age, sex, and time from symptom onset to CMR. The most common location of LGE in all groups was subepicardial at the basal inferolateral wall, although septal involvement was less common in vaccine-associated myocarditis [57].

4.1.2 | Nonviral Agents

In certain populations and regions of the world, specific nonviral infections, such as bacterial, fungal, and protozoal/parasitic agents, remain important causes of inflammatory myocardial involvement; unfortunately, there is limited data on CMR findings, and no distinct pattern has emerged for this etiology [11, 13]. In the context of bacterial infections, auto-immune alterations following untreated streptococcal infection and Borrelia burgdorferi (Lyme disease) infections, endemic in some regions of the Northern Hemisphere, remain significant [58, 59].

An important parasite endemic to Latin America is Trypanosoma cruzi (Chagas disease), which causes myocardial tissue damage through multiple mechanisms: autonomic nervous system derangements, microvascular disturbances, parasite-dependent myocardial aggression, and immune-mediated myocardial injury [60]. In Chagas disease, LGE is observed in up to 70% of patients and it is most frequently observed in the LV apex, as well as in the apical inferior and lateral walls and the basal-to-mid inferolateral wall. LGE typically manifests in mid-wall or subepicardial regions and is less frequently observed in subendocardial or transmural areas; often accompanied by the presence of apical aneurysms [61]. Chronic or recurrent (tropical) parasitic infections can lead to eosinophilic endomyocarditis and may ultimately be responsible for some cases of endomyocardial fibrosis associated with restrictive cardiomyopathy [62].

4.2 | Noninfectious Myocardial Inflammation

4.2.1 | Auto-Immune Diseases

ADs encompass a broad spectrum of conditions that impact the cardiovascular system. AM and chronic I-CMP are often linked to systemic or organ-specific auto-immune disorders. An intercurrent infection can trigger immune activation that exacerbates the underlying immune condition, potentially involving the heart. Identifying the underlying condition linked to myocarditis is crucial, as targeted treatment can mitigate the risk of progression, particularly since AM frequently presents as an early sign of a broader systemic inflammatory or AD [63].

Conventional imaging methods often fail to detect early cardiac involvement in auto-immune disorders due to limited tissue characterization capabilities. This limitation can lead to underdiagnosis of early myocardial damage, potentially progressing to dangerous MACE. However, CMR offers a noninvasive alternative with superior diagnostic and prognostic capabilities for assessing cardiovascular involvement in AD patients. A variety of auto-immune disorders is associated with myocardial inflammation and can be classified according to the predominant inflammatory cell type or the underlying systemic disease, as illustrated in the algorithm in Figure 1. This categorization supports a tailored approach to diagnosing and managing each auto-immune subtype. Although CMR findings are often similar across different subtypes, there can be distinct patterns of LGE distribution in terms of extent, distribution, and/or localization.

4.2.1.1 | Eosinophilic Myocarditis. EM is a rare and potentially life-threatening form of myocardial inflammation, characterized by eosinophilic infiltration into cardiac tissue and often associated with peripheral eosinophilia [64]. It is linked to various conditions, from hypersensitivity and ADs to neoplasia and infections [65]. The etiology in many cases remains unknown, leading to an idiopathic classification. Clinical presentations range from mild to severe, with acute presentations like fulminant myocarditis and life-threatening arrhythmias, more common in hypersensitivity-associated EM, or chronic restrictive cardiomyopathy (also called Loeffler cardiomyopathy) [66, 67]. Definitive diagnosis requires EMB although CMR can provide supportive evidence by detecting typical findings [23, 68] (Table 4 and Figure 3).

4.2.1.2 | Auto-Immune Connective Tissue Diseases. Connective tissue diseases (CTDs) are a group of chronic, auto-immune conditions characterized by an immune response targeting self-antigens, often triggered by genetic predisposition and environmental factors, ultimately leading to tissue and organ damage [69]. While CTDs commonly affect organs such as the musculoskeletal system, skin, kidneys, lungs, and central nervous system, cardiac involvement—particularly myocarditis—remains underdiagnosed and is not always included in classification criteria. However, myocardial inflammation has been observed frequently in autopsies, with up to 40%–50% prevalence in patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE), and similarly high rates in systemic sclerosis (SSc), Sjögren's syndrome, polymyositis (PM), and dermatomyositis (DM) [70–73]. This discrepancy underscores the need for improved noninvasive detection. Despite the introduction of targeted therapies, cardiovascular disease remains a significant contributor to reduced life expectancy in patients with CTDs. The conventional screening techniques for heart failure, which include electrocardiogram (ECG), TTE, as well as serum cardiac biomarkers, such as high-sensitivity troponin and NT-proBNP, are not always sufficient to detect the early involvement of subclinical inflammation. Incorporating CMR into standard evaluation protocols could enable earlier detection and improved management of cardiac involvement in CTDs.

4.2.1.2.1 | Rheumatoid Arthritis. RA is a chronic auto-immune inflammatory disease affecting joints and multiple organs, including the heart and cardiovascular system, leading to diverse complications such as accelerated atherosclerosis, valvular heart disease, and myocarditis, the latter occurring in approximately 6% of cases [74, 75]. RA-associated cardiac disease frequently manifests in patients with active extra-articular disease, elevated rheumatoid factor titers, and systemic vasculitis [75]. Subclinical myocardial involvement is a common occurrence

TABLE 4 | Main findings of the most common eosinophilic myocarditis.

	Idiopathic EM	Hypersensitivity-associated EM	Eosinophilic granulomatosis with polyangiitis (EGPA)-associated EM	HES-associated EM
Prevalence ***26% other causes**	36% 	**34%** 	**13%** 	**9%** 
Key Features	Eosinophilic myocardial infiltration of unknown cause Symptoms: dyspnea, chest pain, elevated troponin levels indicating myocardial injury Variable progression, depending on severity	Triggered by drugs (e.g., anticonvulsants, antibiotics) Symptoms: chest pain, dyspnea, fever Can range from mild myocarditis to fulminant necrotizing myocarditis Elevated risk of cardiac arrest	Multisystem disease with vasculitis, asthma, and eosinophilia Symptoms: dyspnea, chest pain, systemic involvement Cardiac complications is the leading cause of mortality	Persistent eosinophilia Symptoms: dyspnea, chest pain, pericardial effusion High morbidity and mortality from severe cardiac complications
Typical MR Findings	Subendocardial LGE	Subendocardial LGE	Variable LGE patterns (mid-wall, epicardial, transmural) Subendocardial LGE (when diffuse, in severe cases may indicate Löffler endocarditis, +/- thrombi)	Diffuse subendocardial LGE Frequent presence of intracardiac thrombi

in RA, including focal and diffuse myocardial fibrosis and inflammation. These have been demonstrated to be present frequently even at the time of diagnosis. CMR imaging plays an essential role in the identification of subclinical cardiac involvement in RA. Both symptomatic and asymptomatic patients with RA exhibit higher native T1/T2 mapping and ECV values compared to controls. It also shows RMWA with reduced GLS at the mid-ventricular level, with LGE often located in the basal and mid-ventricular inferolateral walls [76]. The presence of myopericarditis in CMR can precede the development of relapse and subsequent congestive heart failure in RA patients, which is a poor prognostic indicator in this patient population [77].

4.2.1.2.2 | Systemic Lupus Erythematosus. SLE is a chronic auto-immune disorder that affects various organs, including the cardiovascular system. Cardiac involvement is a common occurrence, with over 50% of SLE patients presenting with heart-related issues, particularly myocarditis, which is clinically overt in up to 15% of cases. However, autopsy studies reveal that subclinical myocarditis is much more common in SLE, affecting 30%–50% of patients [78–80]. It is postulated that myocarditis in the context of SLE is an immune complex–mediated vascular phenomenon, therefore subendocardial fibrosis resulting from severe microvascular ischemia may be observed. Myocarditis in SLE can present with a similar spectrum of CMR findings to infectious myocarditis, including epicardial to mid-wall fibrosis of the basal to mid-lateral wall and mid-wall fibrosis of the interventricular septum [81]. T1, T2, and ECV values are usually elevated, even in the absence of overt symptoms [82] (Figure 4). Among these, mapping parameters have been demonstrated to offer prognostic value, as they correlate with disease activity and treatment response on follow-up CMR [83].

4.2.1.2.3 | Systemic Sclerosis. SSc is a CTD characterized by immune activation, auto-antibody production, and macrophage involvement. These mechanisms contribute to endothelial dysfunction, low-grade inflammation, and fibrosis, driving widespread vascular damage and multiorgan involvement [84, 85]. Postmortem studies have revealed significant cardiac abnormalities in over half of SSc patients often asymptomatic during life but associated with poor prognosis when symptomatic, with a 2-year mortality rate reaching 60% [86]. CMR frequently detects SSc-related cardiac involvement, initially presenting as edema and progressing to fibrosis affecting both ventricles, potentially causing conduction abnormalities [87, 88]. Pericardial involvement, though typically mild, can lead to significant complications such as large pericardial effusion or even tamponade, which may subsequently lead to constriction [89]. Patchy, mid-wall, linear LGE in the LV wall is a characteristic CMR finding that indicates fibrosis typical of SSc myocarditis [90] (Figure 5). Additionally, subendocardial fibrosis resulting from severe microvascular ischemia has been observed, with the extent of this fibrosis varying from focal to circumferential [91]. Elevated T1 mapping and ECV values have been identified in SSc patients, with T1 values demonstrating a correlation with left atrial volume indices and LV diastolic dysfunction [92].

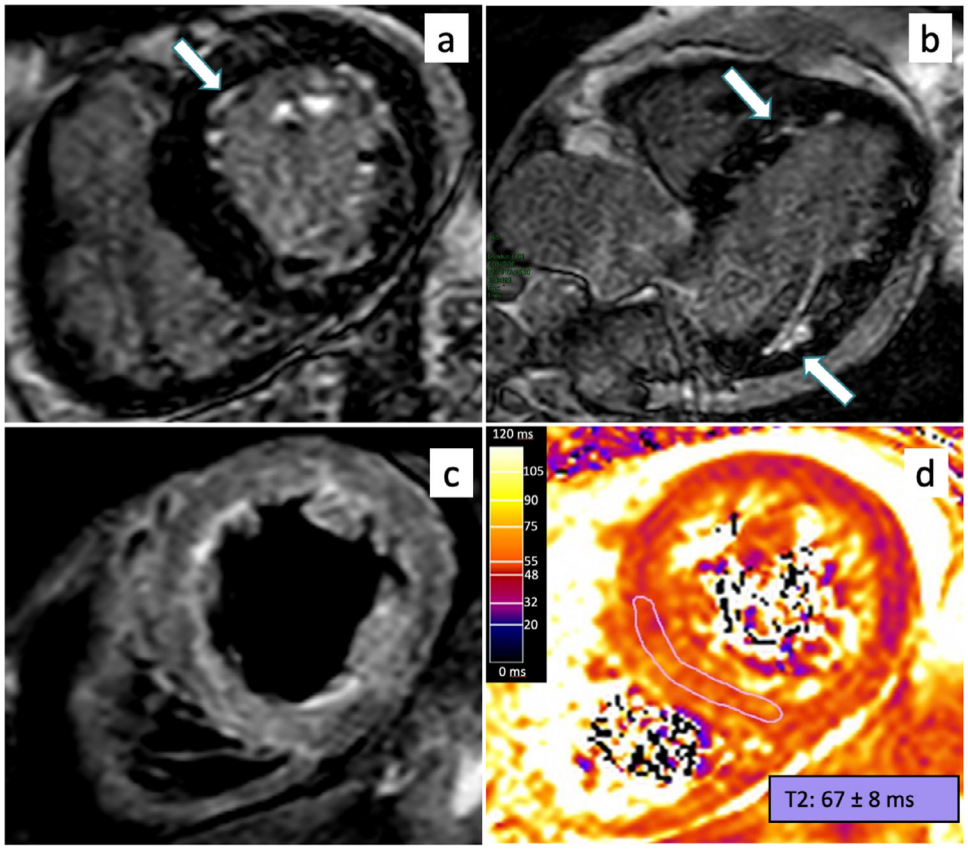


FIGURE 3 | Acute endomyocarditis in eosinophilic granulomatosis with polyangiitis (EGPA). LGE images display patchy subendocardial LGE (arrows in a, b) in the basal anterolateral wall, mid-cavity, and apical regions, along with increased T2 signal in STIR sequence (c) and increased T2 values on mapping (d). LGE, late gadolinium enhancement; STIR, short tau inversion recovery.

4.2.1.2.4 | Polymyositis and Dermatomyositis. PM and DM are idiopathic inflammatory myopathies (IIM) defined by chronic inflammation of skeletal muscle tissue, often associated with vasculitis, focal fibrosis, intimal proliferation, and medial vessel sclerosis. These pathological changes contribute to both skeletal muscle and cardiac damage [93, 94]. Cardiac involvement is a major contributor to mortality, accounting for up to 20% of deaths [95, 96]. Myocarditis, present in approximately 8% of patients, often develops insidiously, complicating timely diagnosis and management [97]. Postmortem studies further reveal myocarditis in around 30% of asymptomatic DM patients [98]. Pericarditis affects 4%–25% of PM/DM patients but is generally asymptomatic and hemodynamically insignificant [99]. CMR reveals signs of myocardial inflammation in 50%–62% of cases, with LGE observed in up to 65% of PM and 54% of DM cases, often as patchy mid-wall LGE in the lateral septal walls of the LV [100]. Newly diagnosed IIM patients exhibit significantly elevated T1 and T2 relaxation times and ECV, indicative of myocardial inflammation [101].

4.2.1.3 | Others AD. Vasculitis can manifest in a multitude of forms, encompassing large-caliber vessels such as those affected in giant cell arteritis and Takayasu arteritis, as well as medium-sized vessels affected in polyarteritis nodosa. Moreover, vasculitis can affect small vessels, as observed in granulomatosis with polyangiitis, microscopic polyangiitis, eosinophilic granulomatosis with polyangiitis, or vessels of varying sizes, as seen in Bechet's disease. **Behçet's disease** is an idiopathic, multisystem vasculitis that characteristically affects small, medium, and large arteries and veins. However, it can also directly involve all layers of the heart, presenting with pericarditis, myocarditis, and endocarditis [102]. CMR can identify myocardial fibrosis in Behçet's disease, with findings similar to those of the other inflammatory myocardial diseases. It can cause aseptic endomyocarditis of the right ventricle (RV) leading to subendocardial fibrosis and large thrombi formation.

Sarcoidosis is an idiopathic granulomatous disease characterized by multisystem involvement [13]. Among the organs involved in systemic sarcoidosis, cardiac involvement occurs in 2%–3% of patients, though postmortem studies show up to 25% of those with extra-cardiac sarcoidosis (CS) may be affected [103, 104]. The disease progresses through the following three distinct stages: edema, granulomatous inflammation, and fibrosis. Although CS lacks a unique imaging pattern, typical findings often include LGE in the subepicardial and mid-wall regions, particularly along the basal and mid-septum and inferolateral walls. One of the most distinctive features is the "Hook Sign," in which LGE extends into the RV from the insertion points in the septal wall of the LV, serving as a crucial indicator of CS [105–107]. CMR findings have been demonstrated to be of critical importance in predicting malignant arrhythmias and sudden cardiac death and have been shown to possess prognostic value in the assessment of LV and RV LGE [108, 109]. In some cases, CS can mimic arrhythmogenic cardiomyopathy or ischemic myocardial infarction, as LGE patterns may appear sub-endocardial or transmural. Elevated native T1,

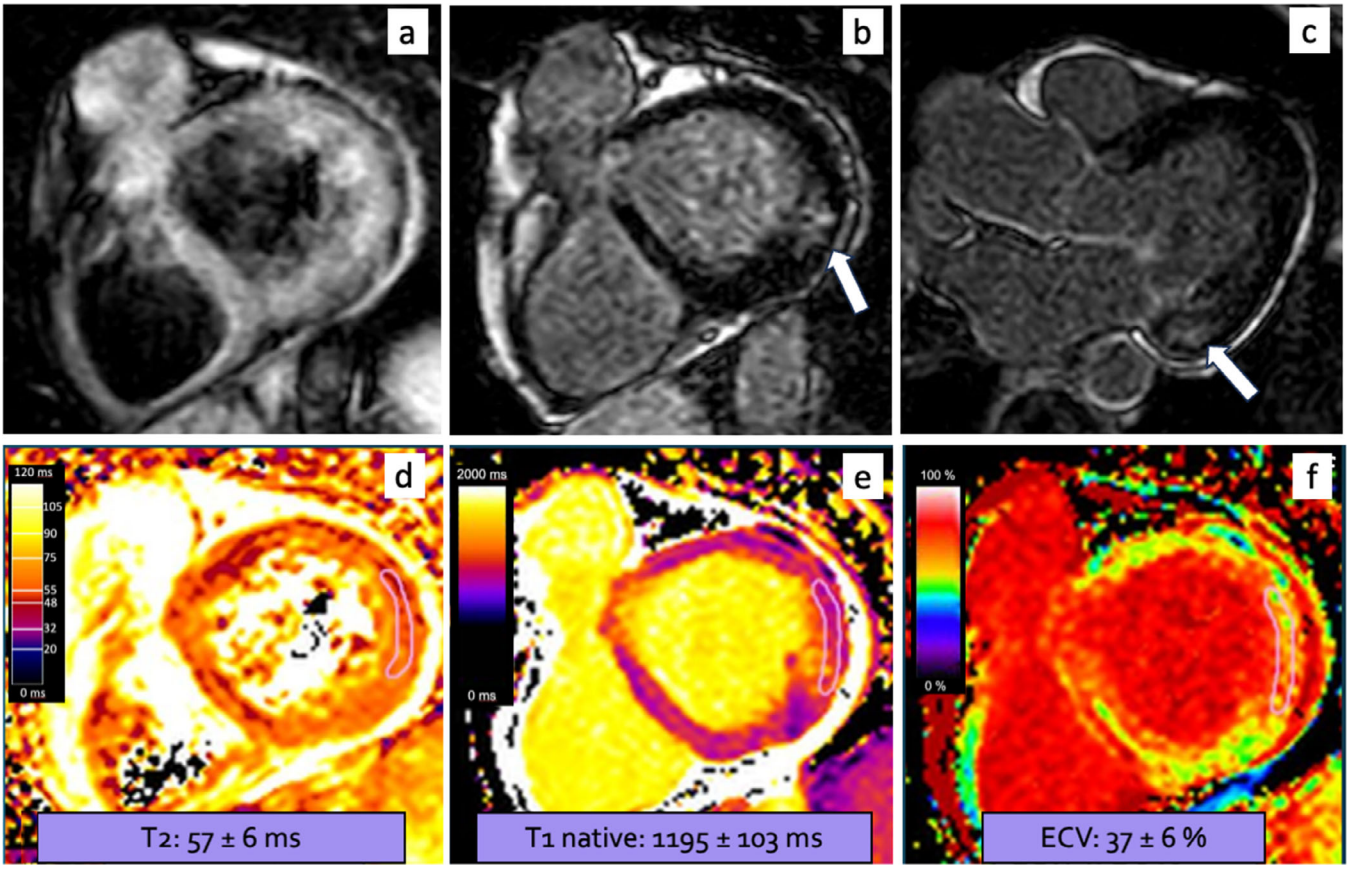


FIGURE 4 | Acute myocarditis in lupus eritematosus (LES). STIR images (a) show increased signal in the LV basal lateral wall with associated focal mid-wall LGE (arrows in images b, c) and high myocardial native T1, ECV, and T2 mapping values (d)–(f). ECV, extracellular volume; LGE, late gadolinium enhancement; LV, left ventricular; STIR, short tau inversion recovery.

T2, and ECV values, along with abnormal post-contrast T1 values, are common in CS, even without LGE, and may indicate early cardiac involvement [110, 111]. Crouser et al. found significantly higher myocardial T2 values on mapping sequences among 50 consecutive patients investigated for cardiac sarcoid compared to healthy controls [111]. T2 cutoff of 59 ms demonstrated a sensitivity of 54% and a specificity of 100%. Cine images can reveal ventricular dilation, a reduced ejection fraction, and regional wall thickening or thinning.

PET-CT imaging plays a pivotal role in assessing sarcoidosis by identifying areas of active inflammation (Figure 6), offering improved detection of the active phase of CS [112]. Increased T2 signal and LGE have been demonstrated to correspond to regions taking up 18F-FDG, with reduced uptake following corticosteroids, indicating active inflammation. However, LGE is also present in regions without 18F-FDG uptake, suggesting the presence of fibrotic lesions. Therefore, an increased T2 signal may reflect active inflammation, whereas LGE may represent either active inflammation or fibrosis [24].

Positron emission tomography–magnetic resonance imaging (PET-MRI) represents an emerging modality that combines the metabolic imaging capabilities of PET with the high-resolution anatomical and functional details provided by MRI. This hybrid approach has demonstrated potential for enhanced diagnosis, disease activity assessment, therapy monitoring, and prognosis in CS, offering complementary information that improves sensitivity in early subclinical stages and aids in patient management decisions [113].

4.2.2 | Toxic Myocardial Inflammation

Myocardial inflammation can be triggered by a plethora of pharmacological agents, toxins, and physical factors, which act via a multitude of pathological mechanisms [1]. Their CMR findings are nonspecific and include evidence of myocardial edema, myocardial interstitial fibrosis on T1 mapping and ECV, and subepicardial to mid-wall enhancement on LGE.

4.2.2.1 | Cancer Treatment–Related Cardiac Dysfunction. The application of CMR in cardio-oncology has become a topic of significant recent scientific interest, as a considerable number of oncology drugs, particularly those of an immunotherapeutic nature, have demonstrated the potential for adverse cardiac effects. In this context, CMR can assist in the diagnosis, prognostication, and the provision of guidance for the management of cancer treatment–related cardiac dysfunction (CTRCD) [114, 115]. Among the various oncological treatments causing cardiotoxicity, the most frequent and well-studied are anthracyclines, ICIs, and thoracic radiation therapy.

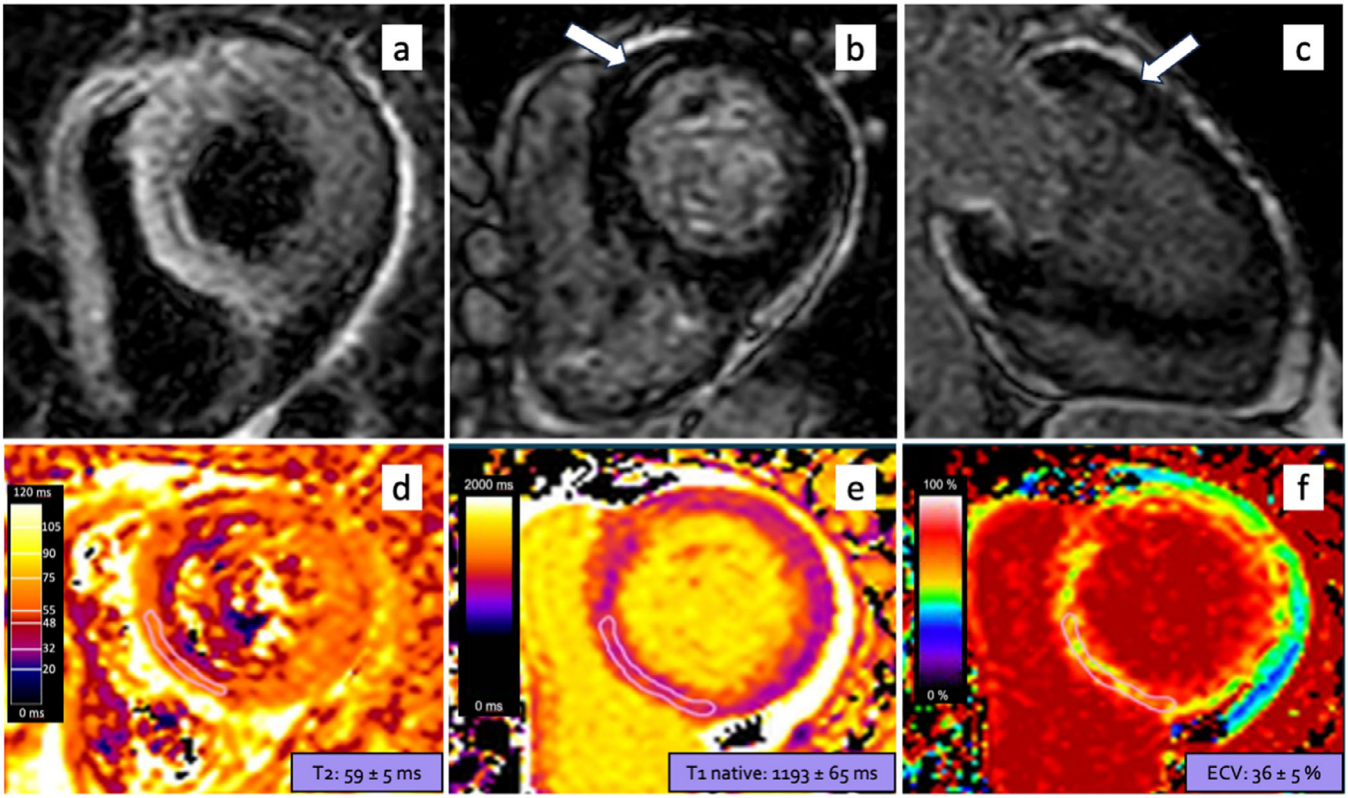


FIGURE 5 | Acute myocarditis with pericardial involvement in systemic sclerosis (SSc). STIR images (a) reveal increased signals in the interventricular septum and anterior LV wall. LGE images (b), (c) show focal mid-wall enhancement (arrows), in the LV basal anterior segment. High myocardial native T1, ECV, and T2 mapping values (d)–(f) are observed on the basal LV septum. Thickening and increased inferior and lateral pericardial sheets signal intensity in STIR and LGE (a)–(c). ECV, extracellular volume; LGE, late gadolinium enhancement; LV, left ventricular; STIR, short tau inversion recovery.

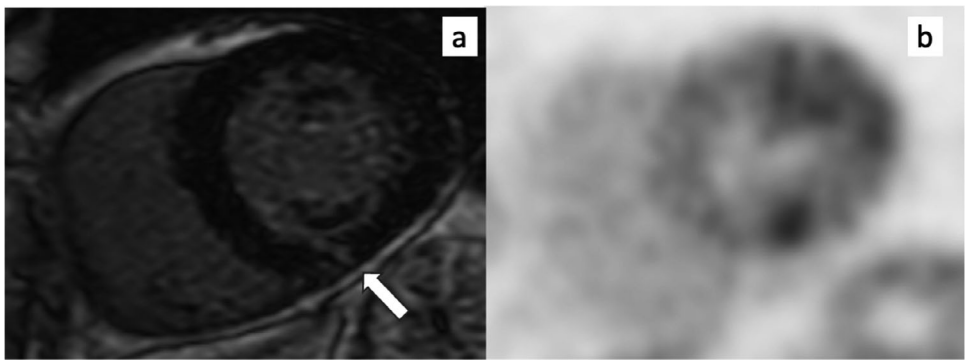


FIGURE 6 | Cardiac sarcoidosis. CMR (a) shows a focal area of LGE in the basal infero-septal wall (arrow), which corresponds to an area of increased uptake of the metabolic tracer at PET-CT (b). CMR, cardiac magnetic resonance; LGE, late gadolinium enhancement; PET-CT, positron emission tomography-computed tomography.

Anthracyclines have well-documented chronic effects that can lead to cardiac damage [116, 117]; making it essential to recommend serial surveillance using imaging and biomarkers for both symptomatic and asymptomatic patients, given the lifelong risk of CTRCD [115]. CMR can detect alterations in cardiac function and myocardial damage by identifying edema, focal fibrosis/scarring, and diffuse fibrosis through conventional sequences, which enhance its sensitivity in detecting acute involvement and post-therapy myocardial fibrotic remodeling [114, 118–122] (Figure 7); additional studies are needed to determine whether early initiation of cardioprotective therapy, based on abnormal T1, T2, and ECV values, results in improved patient outcomes. The use of strain imaging is increasingly gaining traction in CMR, owing to its ability to facilitate the early detection of CTRCD. This approach is further supported by evidence indicating that echocardiographic GLS can serve as a valuable reference for guiding cardioprotective therapy [115, 123]. The precision of mass estimation by CMR enables the identification of patients with

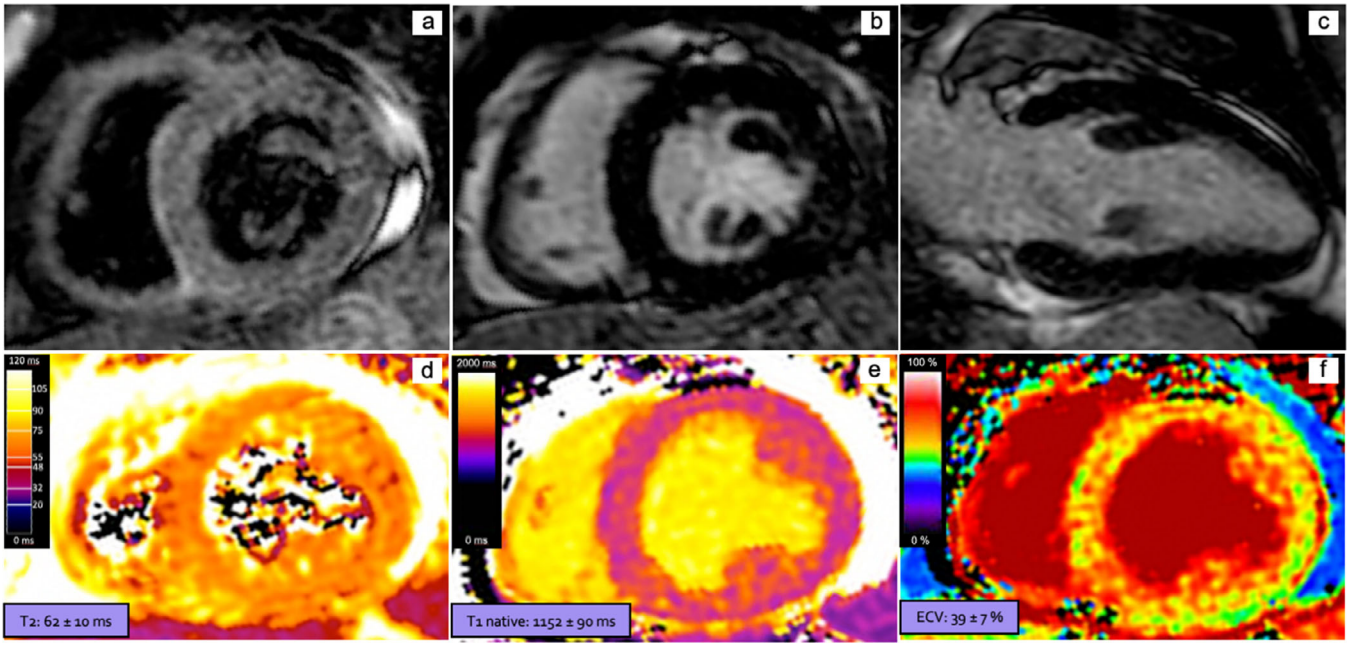


FIGURE 7 | Fifty-nine-year-old patient undergoing treatment for B-cell non-Hodgkin lymphoma with doxorubicin, vincristine, and cyclophosphamide. STIR images (a) show diffuse hyperintensity with associated elevated global myocardial native T1, ECV, and T2 values (d–f; mean values of the mid-ventricular segments are shown in the figures). No focal delayed enhancement was observed on LGE imaging (b), (c). ECV, extracellular volume; LGE, late gadolinium enhancement; STIR, short tau inversion recovery.

reduction cardiac mass after anthracycline therapy, who, despite exhibiting preserved LVEF, manifest an exacerbation of heart failure symptoms [124].

The most extensively and recently studied area in cardio-oncology involves **ICI**; myocarditis is an uncommon but serious complication of ICI therapy, with an incidence ranging from 0.1% to 1% [125–127]. In cases of ICI-associated myocarditis, comprehensive evaluation with CMR can be instrumental in detecting cardiac involvement even before the onset of clinical symptoms, abnormalities in laboratory biomarkers (such as troponin and proBNP), or the development of reduced ejection fraction observed on echocardiography [128]. The incidence of LGE and edema detected by T2-weighted STIR is significantly lower than that observed in AM not related to ICI [36, 129]; a recent analysis found that LGE was present in less than 50% of ICI-associated myocarditis cases, and 42% of cases showed neither LGE nor an elevated T2-weighted STIR signal [127]. These findings suggest that in suspected ICI-associated myocarditis, the absence of LGE or a normal T2-weighted STIR signal on CMR does not rule out the diagnosis, as both are dependent on local variations in fibrosis or inflammation to become qualitatively apparent. Therefore, mapping techniques may play a key role in identifying early myocardial changes before LGE becomes evident. In this setting, T1 mapping has proven to be more sensitive in detecting inflammatory myocardial involvement compared to T2 mapping [127, 130]. Emerging evidence suggests that septal LGE and elevated native T1 mapping values provide significant prognostic information, serving as predictors of major adverse cardiovascular events (MACE) [130, 131]. On the other hand, CMR-FT has demonstrated a potential role in the early detection and prognostic stratification of ICI myocarditis [132, 133]. These findings emphasize the critical role of CMR in the early detection and prognostic assessment of ICI-associated myocarditis. Furthermore, prolonged treatment with immunosuppression resulted in reduced T1/T2 values, demonstrating that CMR may also serve as an alternative strategy to monitor treatment response and determine the optimal timing for discontinuing immunosuppression [128].

Radiotherapy, through a cascade of mechanisms primarily driven by oxidative stress and inflammatory responses, causes cardiac damage and fibrotic remodeling, resulting in structural and functional alterations [134, 135]. While cardiomyocytes are relatively resistant to radiation, endothelial cells are highly sensitive and play a key role in initiating indirect damage to cardiomyocyte [136, 137]. During chest radiotherapy, CMR can evaluate volume and functional changes, which become more pronounced when the damage is permanent, typically observed during longer follow-up periods (>20 years) or in patients treated with older RT techniques and larger radiation fields [138, 139]. Unlike systemic therapy with more homogeneous heart distribution, radiation doses vary spatially, depending on tumor proximity to the heart, with a sharp dose drop-off outside the treatment area. Fibrotic remodeling is dose-dependent, evident as both LGE and elevations in native T1 values and ECV; specifically, LGE has been correlated with the LV mean dose, with >30 Gy identified as a threshold beyond which progressive increases in LGE are observed and a segmental ECV analysis has shown an average increase of 0.136% per Gray of mean segmental dose [140–142]. Abnormal GLS has been observed to precede changes in LVEF, with the earliest effects reported over shorter follow-up periods (e.g., 13 ± 2 months) in patients receiving concurrent treatments such as chemoradiotherapy, where the combined cardiotoxic effects of chemotherapy and radiotherapy may amplify damage [38, 143]. Detecting radiotherapy-induced cardiac toxicity at an early stage using CMR can enhance the chances for clinical

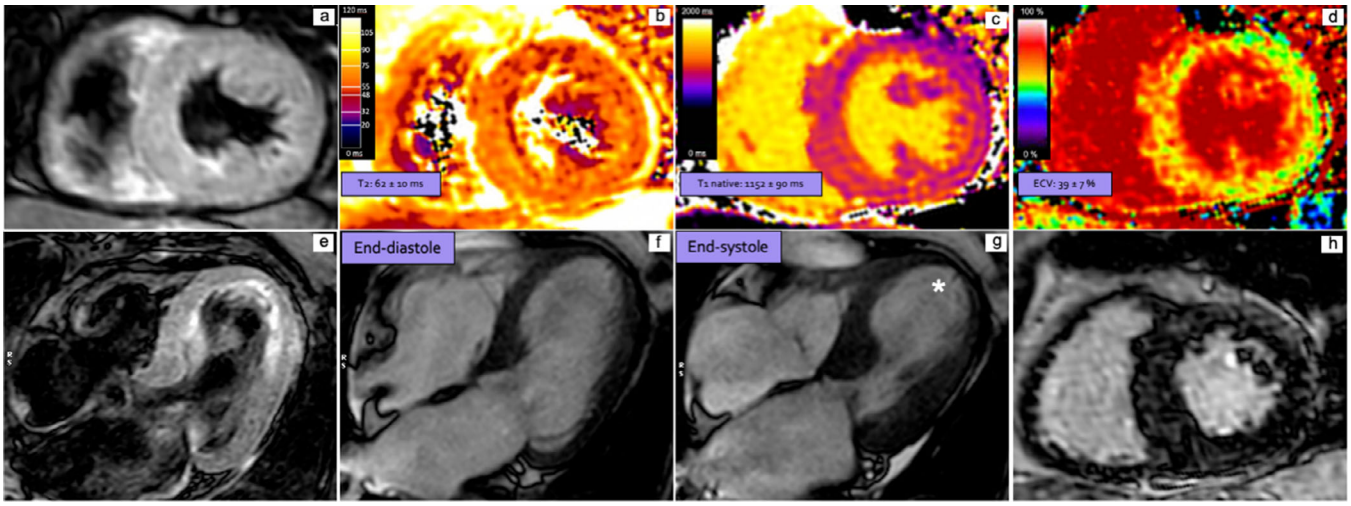


FIGURE 8 | Sixty-year-old woman with Takotsubo cardiomyopathy. CMR demonstrates diffuse myocardial edema in the mid-apical LV walls (a and e, STIR). Mapping reveals elevated global myocardial native T1, ECV, and T2 values (d–f; mean values of the mid-ventricular segments are shown in the figures). Cine-SSFP images in the horizontal long-axis view (f and g) show the typical apical "ballooning" of the LV (* in g) in the end-systolic phase. No focal delayed enhancement was observed on LGE imaging (h).

diagnosis and timely intervention, potentially preventing irreversible damage [144]. The further cardiac side effects related to radiotherapy, such as pericarditis and acceleration of coronary artery disease, are beyond the scope of this review.

4.2.2.2 | CMR in Other Causes of Toxic Myocardial Inflammation. In the context of toxic and pharmacological nononcological substances, particular emphasis is placed on certain substances of abuse, with cocaine being the most studied due to its relatively frequent harmful cardiovascular effects. Furthermore, considerable focus has been directed towards the investigation of clozapine-induced myocarditis (CIM), a significant adverse event that limits the use of clozapine, the most effective treatment for schizophrenia [145]. Although CIM is rarely associated with clozapine treatment (1%–3%), it can be fatal, with a mortality as high as 50% [146, 147]. Therefore, its early recognition is fundamental.

Cocaine-related cardiovascular complications can be either acute or chronic and include both ischemic and nonischemic events [148]. The mechanism underlying cocaine's cardiovascular toxicity is primarily linked to its sympathomimetic effects and its blockade of voltage-dependent K+ and Na+ channels [148, 149]. CMR can provide a valuable assessment of myocardial damage caused by cocaine use in both acute and chronic cardiac complications. It is particularly useful for evaluating the extent and progression of myocardial injury in symptomatic cocaine users; additionally, CMR should be recommended as a screening tool for long-term cocaine users, even in the absence of symptoms [148]. Acute complications may present as myocarditis-like conditions (edema and LGE with mid-wall or subepicardial distribution, without a specific predilection for a particular cardiac region) or as ischemic damage (edema and LGE have subendocardial or transmural distribution); some patients may simultaneously exhibit involvement of all three myocardial layers within the same study [13, 150, 151]. In the chronic setting, CMR can detect abnormalities even in asymptomatic cocaine users, including regional LV hypokinesia, mild dilation of the LV or RV, and fibrosis (observed in 73% of subjects and resulting from silent ischemic cardiac events, exhibiting both ischemic and nonischemic patterns) [152]. In both acute and chronic settings, mapping techniques may offer significant advantages in the detection of myocardial involvement; however, research in this population remains limited, with current evidence mainly highlighting increased ECV in asymptomatic cocaine users [153].

CMR can detect **catecholamine-induced myocardial inflammation** in patients with pheochromocytoma. A systematic CMR study involving patients with pheochromocytoma, compared to healthy and hypertensive controls, showed that this condition can result in focal or diffuse fibrosis and persistent impairment in systolic and diastolic function, even after curative surgery due to subclinical catecholamine myocarditis; these effects exceed those observed in hypertensive heart disease alone, highlighting the direct impact of catecholamine toxicity [154]. A similar pathophysiology is observed in **Takotsubo syndrome** and its associated variants, which may be regarded as a form of acute catecholaminergic myocardial stunning. Also in these cases, CMR enables the identification of RWMA and features of myocardial inflammation. In the classical pattern, seen in 50%–80% of cases depending on the series, RWMA is characterized by apical and circumferential mid-ventricular hypokinesia with basal hypercontractility [155]. CMR tissue characterization helps identify areas of high T2w signal indicative of edema, which often shows a diffuse or transmural distribution corresponding to RWMA. LGE is typically absent but may occasionally appear during the acute phase, resolving during follow-up and without significant prognostic relevance and relation to clinical presentation; persistent apical transmural LGE has been reported only rarely (Figure 8) [155–157].

4.2.3 | Genetic Cardiomyopathies

In the context of cardiomyopathies, a link between genetic abnormalities and susceptibility to inflammation/immune response

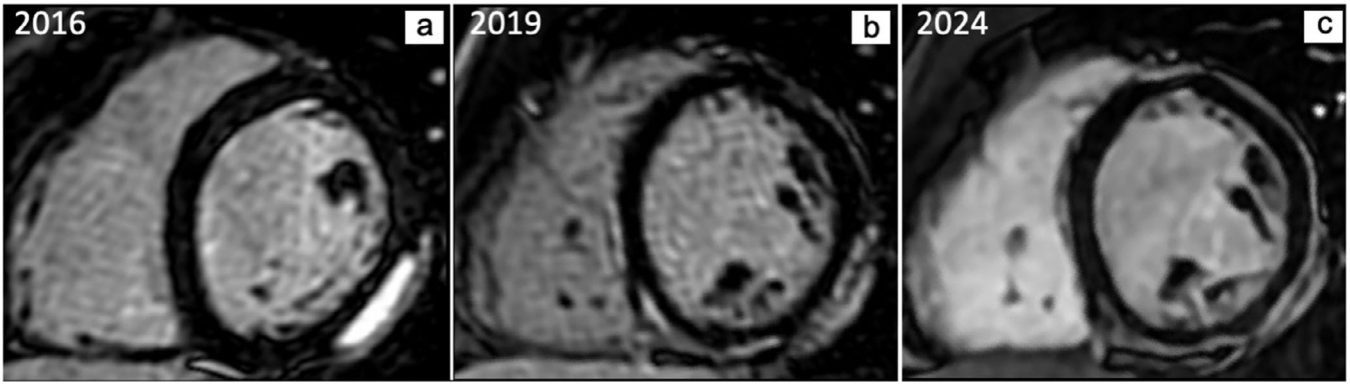


FIGURE 9 | Genetic cardiomyopathy. Progressive enlargement of subepicardial LGE in a young patient with recurrent chest pain associated with mild troponin elevation. Initially interpreted over the years as recurrent myocarditis, the diagnosis was later revised to desmoplakin cardiomyopathy following genetic testing.

has been proposed, with the strongest evidence observed in the conditions formerly known as arrhythmogenic cardiomyopathy, which have now been reclassified in the ESC guidelines on cardiomyopathies [3, 44]. Without full penetrance, a genetic disorder—predominantly caused by desmosomal gene variants and less commonly by nondesmosomal gene variants—leads to an abnormal response of cardiac myocytes to mechanical stress, resulting in myocyte loss and inflammation. Although inflammation is recognized as a key driver in the pathogenesis of cardiomyopathies with a genetic etiological component, the exact role of inflammatory and immune responses, whether triggered by internal factors or external agents such as viruses, remains incompletely understood [158–161]. In addition to identifying the different types of myocardial damage present in these cardiomyopathies, CMR can assess the myocardial inflammatory involvement associated with the so-called "hot phase" of certain genetic cardiomyopathies by detecting edema [162]. The inflammatory involvement in genetic cardiomyopathies is so pronounced that it is not uncommon for myocarditis or recurrent myocarditis to be misdiagnosed, with the underlying genetic condition not being identified until later (Figure 9) [163, 164].

Inflammation also plays a recognized pathogenic role in storage diseases such as amyloidosis and Fabry disease (FD), linked to the toxicity/damage these deposits cause in myocardial tissue; the edema associated with the inflammatory process contributes to the findings observed during CMR tissue characterization.

In FD, a lysosomal storage disorder leading to the accumulation of glycolipids, CMR identifies the following three progressive phases [165–167]:

- Accumulation phase: begins in childhood, characterized by progressively lower native T1 values without left ventricular hypertrophy (LVH) or LGE;
- Inflammation and/or hypertrophy phase: features low T1 values, initial LVH (predominantly in males), and evidence of inflammation on T2 mapping, primarily in the basal inferolateral segment, often accompanied by LGE;
- Fibrosis and/or impairment phase: marked by increasing native T1 values (pseudo-normalization), LGE, and wall thinning, predominantly in the basal inferolateral segment.

Amyloidosis, characterized by extracellular deposition of fibrillary proteins, primarily presents in the following two forms: immunoglobulin light-chain amyloidosis (AL) and transthyretin amyloidosis (ATTR). Of these, the only hereditary form is ATTR, specifically its subcategory ATTRv, which is caused by an autosomal dominant mutation in the TTR gene. In contrast, both wild-type ATTR (ATTRwt) and AL amyloidosis are not inherited. ATTRwt occurs due to the misfolding of the wild-type TTR protein, commonly seen in elderly individuals, while AL amyloidosis results from a clonal plasma cell disorder in which misfolded immunoglobulin light chains are produced and deposited as amyloid fibrils in various tissues [168]. CMR demonstrates variable distribution of LGE and a diffuse increase in T2 mapping, native T1 mapping, and ECV, with ECV shown to be the single best parameter to differentiate cardiac amyloidosis from other LVH [169]. CMR can also provide valuable insights into the differentiation between AL and ATTR amyloidosis, thanks to higher T2 mapping values in AL and thanks to differing patterns of LGE distribution (a subendocardial preference in AL compared to the diffuse, often transmural distribution seen in ATTR) [169]. Compared to FD, the inflammatory involvement in amyloidosis is less pronounced but remains a consistent contributor to its pathophysiology [170, 171]. The deposition of fibrils in cardiac tissue may lead to fluid retention and mild cardiotoxic effects, resulting in myocardial edema detectable via T2 mapping; the prolonged T2 values observed in AL amyloidosis versus ATTR amyloidosis are potentially attributable to greater fluid retention and increased cardiotoxicity in AL amyloidosis [169, 172, 173].

There is also evidence of myocardial inflammation in hypertrophic cardiomyopathy associated with sarcomere protein gene mutations. Researchers have proposed that factors such as cardiomyocyte disarray, sarcomere damage, mitochondrial oxidative stress, and microvascular disease may act as triggers for the onset of early inflammation in this condition [174].

5 | Conclusion

CMR has transformed the evaluation of myocardial inflammation, providing detailed tissue characterization and functional assessment. Its implementation has refined the diagnosis of AM and I-CMP, allowing differentiation from other

myocardial pathologies and aiding in prognostication. However, CMR findings show substantial overlap between different causes of myocardial inflammatory involvement, making the integration of clinical features essential for accurate diagnosis. Despite advances, challenges remain in standardization, early detection in the acute setting, and comprehensive evaluation of multiple etiologies. This review underscores the need for a multidisciplinary approach that combines clinical insights and imaging findings to improve diagnostic accuracy, risk stratification, and management of inflammatory myocardial disease.

Acknowledgments

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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