Research Article

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Baseline and Follow-up Cardiac Magnetic Resonance Findings in Pediatric Acute Myocarditis: A Single-center Experience

Demet Kangel[1], Ahmet Saki Oğuz[1], Halise Zeynep Genç[1], Yiğit Can Kartal[2], Serçin Özkök[2], İbrahim Cansaran Tanıdır[3], Erkut Öztürk[1]

[1]University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital, Clinic of Pediatric Cardiology, İstanbul, Türkiye
[2]University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital, Clinic of Radiology, İstanbul, Türkiye
[3]Memorial Hospital, Clinic of Pediatric Cardiology, İstanbul, Türkiye

Abstract

Objectives: Acute myocarditis in children has a broad clinical spectrum, and cardiac magnetic resonance (CMR) is an important non-invasive tool for diagnosis and follow-up. This study aimed to evaluate baseline and follow-up CMR findings in pediatric acute myocarditis and their relationship with clinical and laboratory markers of myocardial injury and inflammation.

Materials and Methods: This single-center retrospective study included pediatric patients who underwent CMR for clinically suspected acute myocarditis. Demographic characteristics, presenting symptoms, laboratory data, electrocardiographic and echocardiographic findings, and CMR parameters were collected. Follow-up CMR was available in a subset of patients to assess interval changes in myocardial tissue characteristics, including native T1 and T2 mapping values, extracellular volume (ECV), late gadolinium enhancement (LGE), and left ventricular ejection fraction (LVEF).

Results: Baseline CMR was performed in 139 pediatric patients with suspected acute myocarditis. Median LVEF was 50% interquartile range (IQR, 33–58): reduced systolic function (LVEF <55%) was observed in 64.7% of the cohort. LGE was identified in 91.4% of patients, with a median extent of 4% (IQR, 3–5). Follow-up CMR performed in 40 patients

Address for Correspondence: Demet Kangel, University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital, Clinic of Pediatric Cardiology, İstanbul, Türkiye
e-mail: demetkangel@gmail.com **ORCID:** orcid.org/0009-0002-5360-7044
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showed significant improvements in LVEF, native T1 and T2 relaxation times, ECV, and LGE extent (all $p<0.05$). All 40 patients had LGE at baseline, and LGE remained detectable in 32 patients (80%) at follow-up.

Conclusion: Multiparametric CMR provides comprehensive insight in pediatric acute myocarditis. Significant improvement in ventricular function and tissue characterization parameters was observed at follow-up, although LGE remained detectable in 80% of patients. These findings support the role of CMR in both the initial evaluation and follow-up of pediatric myocarditis.

Keywords: Myocarditis, pediatric, cardiac magnetic resonance, late gadolinium enhancement, T1 Mapping, extracellular volume

Introduction

Acute myocarditis is an inflammatory disease of the myocardium with a highly variable clinical presentation, ranging from mild, non-specific viral symptoms to chest pain, ventricular dysfunction, cardiogenic shock, life-threatening arrhythmias, or sudden cardiac death in previously healthy children[1-4]. Although the true incidence of pediatric myocarditis is difficult to determine because many cases remain subclinical or misdiagnosed, epidemiological studies estimate an annual incidence of 1–2 per 100,000 children and myocarditis remains an important, though frequently under-recognized, cause of sudden unexpected death in young people[5-7].

In pediatric myocarditis, infections are the most common underlying cause, with enteroviruses, adenovirus, parvovirus B19, human herpesvirus-6, and influenza viruses among the frequently identified pathogens, although the distribution of individual agents varies across regions, age groups, and diagnostic eras[1-3,8-10]. Emerging respiratory viruses, most notably SARS-CoV-2, have further contributed to the evolving epidemiology of the disease[3].

Historically, the diagnosis of myocarditis relied on endomyocardial biopsy and the Dallas criteria. However, endomyocardial biopsy is limited by its invasive nature, sampling error, and low sensitivity, especially in children[1,11]. Accordingly, current recommendations from the American Heart Association and the American College of Cardiology support a multimodal diagnostic strategy that integrates clinical assessment, cardiac biomarkers, electrocardiography, echocardiography, and importantly, cardiac magnetic resonance (CMR) imaging[2,4]. CMR has become a pivotal tool in diagnosing myocarditis because it enables noninvasive, whole-heart assessment of myocardial edema, hyperemia, necrosis, and fibrosis. With the introduction of the 2018 updated Lake Louise Criteria, multiparametric CMR, including T1 and T2 mapping and extracellular volume (ECV) analysis, has significantly improved the sensitivity and specificity for detecting active myocardial inflammation[4,12,13].

The utility of CMR in children has expanded considerably over the past decade. Multicenter data indicate that the use of CMR in pediatric myocarditis has increased substantially in recent years, reflecting its growing role in clinical decision-making[14]. In this context, T1 and T2 mapping techniques are particularly valuable in children, as they enable the detection of diffuse myocardial inflammation that may not be apparent on late gadolinium enhancement (LGE) imaging[15,16]. Moreover, mapping values correlate with biochemical markers of myocardial injury and may help differentiate acute from subacute or resolving disease[15,17]. In addition to baseline assessment, follow-up CMR provides important information on myocardial recovery, with normalization of mapping values often preceding improvement in LGE or ventricular function[12,18,19].

Despite the increasing adoption of CMR in pediatric myocarditis, real-world data on the spectrum of multiparametric CMR findings and their relationship with clinical and laboratory markers remain limited. Studies focusing on both baseline and follow-up CMR in children are particularly scarce. Given the heterogeneous presentation and variable clinical course of pediatric myocarditis, ranging from complete recovery to progression toward chronic myocardial dysfunction, comprehensive CMR evaluation can provide valuable insights into myocardial tissue changes throughout its course. This study aimed to characterize baseline multiparametric CMR findings, evaluate follow-up imaging changes, and assess the association of these findings and changes with markers of myocardial injury and inflammation in a single-center cohort of pediatric patients with acute myocarditis. Our findings may help further define the role of CMR in the diagnostic and longitudinal assessment of children with myocarditis.

Materials and Methods

Study Design and Patient Population

A retrospective, single-center study was conducted involving pediatric patients hospitalized with a diagnosis of acute myocarditis who underwent CMR between August 1, 2022, and November 1, 2025. The study included patients aged 1 month to <18 years.

The diagnosis of acute myocarditis was established based on clinical presentation, elevated cardiac biomarkers (troponin and/or CK-MB), electrocardiographic or echocardiographic abnormalities, and supportive CMR findings interpreted according to the 2018 updated Lake Louise Criteria (requiring at least one T2-based marker of myocardial edema and one T1-based marker of non-ischemic injury)[12], in accordance with current consensus recommendations[1,4]. Endomyocardial biopsy was not performed in any patient. Left ventricular systolic function was assessed by echocardiography using M-mode (Teichholz-derived) left ventricular ejection fraction. Reduced systolic function was defined as an ejection fraction of <50% on echocardiography and of <55% on CMR.

Patients with pre-existing congenital heart disease requiring surgical or catheter-based intervention (except for hemodynamically insignificant lesions), known cardiomyopathy, prior myocarditis, or systemic inflammatory, autoimmune, or metabolic disorders that could mimic myocarditis, as well as those with non-diagnostic or incomplete CMR examinations or contraindications to CMR or gadolinium-based contrast agents, were excluded. Patients with multisystem inflammatory syndrome in children (MIS-C)–associated cardiac involvement were also excluded, and no cases of MIS-C or vaccine-associated myocarditis were identified during the study period.

During the study period, 155 patients with acute myocarditis undergoing CMR were assessed for eligibility; 11 were excluded because CMR was not performed, and 5 were excluded because of incomplete data, yielding a final baseline cohort of 139 patients, of whom 40 (28.8%) underwent follow-up CMR. The patient selection process is summarized in Figure 1.

The study protocol was approved by the Ethics Committee of University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital (approval no: 2025/355; November 26, 2025). The study was conducted in accordance with the Declaration of Helsinki. The ethics committee waived the requirement for informed consent due to the retrospective nature of the study.

Cardiac Magnetic Resonance Acquisition and Analysis

All CMR examinations were performed using a 1.5-T Ingenia scanner (Philips Healthcare, Best, the Netherlands). In children younger than 6 years of age, imaging was performed during free breathing with motion correction applied, and sedation was used when necessary, according to institutional pediatric anesthesia protocols. Older children underwent breath-hold, electrocardiogram-gated acquisitions.

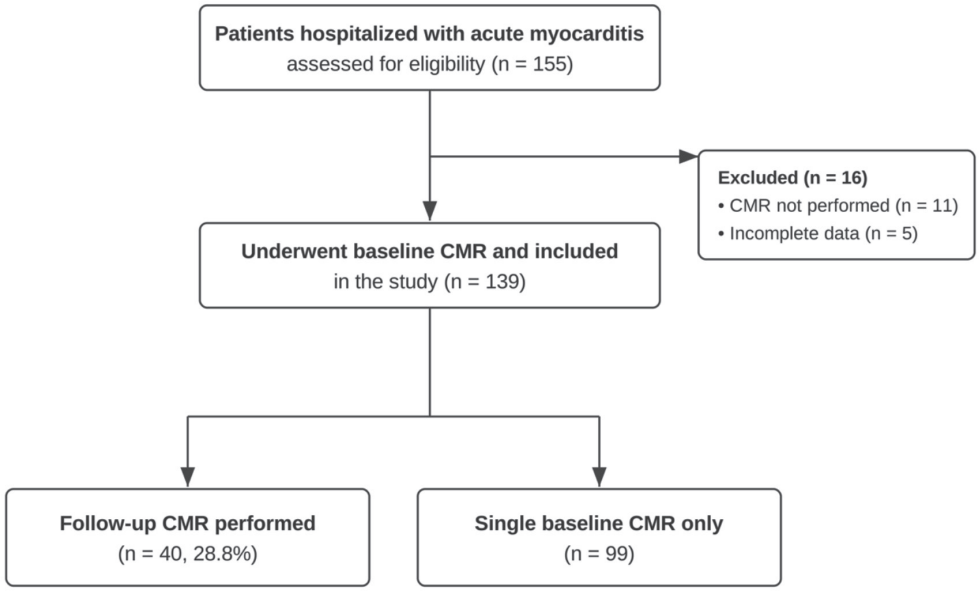

Figure 1. STROBE flow diagram of patient selection. Of 155 patients hospitalized with acute myocarditis who were assessed for eligibility, 16 were excluded (11 without CMR and 5 with incomplete data), yielding 139 patients who underwent baseline CMR. Follow-up CMR was performed in 40 patients (28.8%), while 99 patients had a single baseline study

CMR: Cardiac magnetic resonance

The protocol comprised cine steady-state free precession imaging for ventricular function; native T1 mapping (MOLLI 5(3)3) before and after contrast; T2 mapping (multi-echo GraSE) in basal, mid, and apical short-axis slices; and LGE imaging with a phase-sensitive inversion-recovery sequence 10 minutes after intravenous administration of gadolinium-based contrast (0.15 mmol/kg). Detailed acquisition parameters are provided in Supplementary Table S1.

All measurements were performed on a dedicated workstation (IntelliSpace Portal 11.2, Philips Healthcare) by an experienced radiologist (S.O.) trained in congenital cardiac imaging, who had performed more than 1500 cardiac magnetic resonance imaging examinations and was blinded to clinical details. LVEF was derived from short-axis cine images by tracing endocardial contours across all cardiac phases, with papillary muscles and trabeculations included in the ventricular blood pool. For native T1 and T2 mapping, endocardial and epicardial contours were manually drawn, excluding the blood pool and epicardial fat, and global myocardial values were generated within the defined myocardial mask. LGE was quantified on phase-sensitive inversion-recovery images using a semi-automated full-width at half-maximum thresholding method, with hyperenhanced myocardium defined as signal intensity ≥50% of the maximal intensity within the region of enhancement, and expressed as a percentage of left ventricular myocardial mass. ECV was calculated from pre- and post-contrast myocardial and blood-pool T1 values and same-day hematocrit using the formula ECV = (1 − hematocrit) × (ΔR1myocardium/ΔR1blood).

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 23.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as median with interquartile range (IQR), and categorical variables were presented as number and percentage.

Comparisons between patients with and without follow-up CMR were performed using the Mann-Whitney U test for continuous variables and the chi-square test or Fisher's exact test for categorical variables, as appropriate. Paired comparisons between baseline and follow-up CMR findings were performed using the Wilcoxon signed-rank test for continuous variables and the McNemar test for categorical variables. The influence of the follow-up interval on the degree of was assessed using Spearman's correlation between the follow-up interval and the paired change in each CMR parameter. For paired comparisons, effect sizes were reported as the matched-pairs rank-biserial correlation; 95% confidence intervals for the median change were also provided. Associations between laboratory markers (peak troponin, peak C-reactive protein (CRP), and NT-proBNP) and baseline CMR tissue parameters (native T1, T2, ECV, and LGE extent) were assessed using Spearman rank correlations. Because myocardial native T1 varies with age in children, partial Spearman correlations adjusted for age were used as the primary analysis. 95% confidence intervals were derived using the Fisher z-transformation. LGE extent was analyzed across the full cohort, and LGE-negative studies were assigned an extent of 0%. To account for multiple comparisons within the correlation analysis, p-values were adjusted using the Benjamini-Hochberg false-discovery-rate procedure, and these associations were considered exploratory. A two-sided p-value <0.05 was considered statistically significant.

Results

During the study period, 139 pediatric patients hospitalized with acute myocarditis underwent baseline CMR. Follow-up CMR was available in 40 patients. Baseline demographic and clinical characteristics did not differ significantly between patients with and without follow-up imaging (Table 1). At baseline CMR, however, patients who underwent follow-up had a higher prevalence of LGE (100% vs. 87.9%, p=0.019) and higher T2 values (p=0.024), and showed non-significant trends toward greater LGE extent and pericardial effusion, whereas laboratory markers and other functional parameters were comparable (Supplementary Table S2). Thus, patients with more pronounced baseline myocardial involvement were preferentially selected for follow-up imaging.

The median age of the overall cohort was 12.4 years (IQR, 3.4–16.2), and 98 patients (70.5%) were male. The most common presenting symptoms were chest pain (61.2%), dyspnea (29.5%), abdominal pain (20.9%), fever (18.0%), and palpitations (13.7%). Baseline demographic and clinical characteristics of the study population are summarized in Table 1.

At baseline CMR, left ventricular systolic function was mildly reduced overall, with a median LVEF of 50% (IQR, 33–58). Reduced LVEF (<55%) was observed in 90 patients (64.7%). LGE was present in 127 patients (91.4%), with a median extent of 4% (IQR, 3–5); pericardial effusion was identified in 22 patients (15.8%). Detailed baseline CMR findings are provided in Table 2.

After adjustment for age and correction for multiple comparisons, higher peak troponin and higher peak CRP were each associated with greater baseline LGE extent ($\rho=0.39$, $q<0.001$; $\rho=0.22$, $q=0.020$, respectively). Higher NT-proBNP levels were associated with higher native T1 ($\rho=0.36$, $q<0.001$), higher T2 ($\rho=0.28$, $q=0.007$), and greater LGE extent ($\rho=0.32$, $q=0.001$). Correlations with ECV did not reach statistical significance (n = 128). Unadjusted troponin– and CRP–native T1/T2 correlations were negative, but attributable to age confounding and disappeared after age adjustment (Table 3).

Among the 40 patients who underwent follow-up CMR, both ventricular function and myocardial tissue characteristics showed significant improvement over time. The median interval between baseline and follow-up imaging was 144 days (IQR, 63–200). Median LVEF increased from 54% (IQR, 38–60) at baseline to 58% (IQR, 44–63) at follow-up ($p<0.001$). Native T1 values, T2 relaxation times, ECV, and LGE extent decreased significantly on repeat imaging ($p<0.05$ for all). Although the prevalence of LGE declined from 100% to 80%, residual LGE remained detectable in 32 patients at follow-up. In addition, the number of involved myocardial

Table 1. Baseline demographic, clinical, and laboratory characteristics of the study population

Variable	Follow-up (n=40)	No follow-up (n=99)	p-value
Demographic characteristics			
Age, years, median (IQR)	13.3 (4.5–15.6)	11.8 (2.5–16.5)	0.48
Male, n (%)	26 (65%)	72 (72.7%)	0.48
Clinical presentation			
Chest pain, n (%)	26 (65%)	59 (59.6%)	0.69
Palpitations, n (%)	5 (12.5%)	14 (14.1%)	1.00
Dyspnea, n (%)	12 (30%)	29 (29.3%)	1.00
Fever, n (%)	8 (20%)	17 (17.2%)	0.88
Abdominal pain, n (%)	8 (20%)	21 (21.2%)	1.00
Electrocardiographic findings at admission (overall cohort, n=139)			
Normal ECG, n (%)	72 (51.8%)		
ST-segment elevation, n (%)	33 (23.7%)		
Other ST-T abnormalities, n (%)	21 (15.1%)		
Arrhythmia, n (%)	13 (9.4%)		
Echocardiographic findings at admission (overall cohort, n=139)			
Normal echocardiography, n (%)	85 (61.2%)		
Reduced systolic function, n (%)	54 (38.8%)		
Pericardial effusion, n (%)	22 (15.8%)		
Time intervals (days) (overall cohort, n=139)			
Symptom onset to hospital admission, median (IQR)	2 (2–5)		
Hospital admission to baseline CMR, median (IQR)	10 (5–19)		
Troponin normalization time, median (IQR)	5 (4–10)		
Laboratory findings (overall cohort, n=139)			
Peak troponin, ng/L, median (IQR)	243 (114–449)		
C-reactive protein, mg/L, median (IQR)	16.4 (3.9–48.35)		
Procalcitonin, ng/mL, median (IQR)	0.09 (0.06–0.18)		
NT-proBNP, pg/mL, median (IQR)	729 (155–14648)		
Viral testing (n=137)	**n (%)**		
Positive viral test	73 (52.5% of cohort; 53.3% of tested)		
- Rhinovirus	24 (17.5%)		
- Adenovirus	8 (5.9%)		
- SARS-CoV-2	7 (5.1%)		
- Other viruses	34 (24.8%)		

Data are presented as median (IQR) or n (%), CMR: Cardiac magnetic resonance, ECG: Electrocardiography, IQR: Interquartile range, NT-proBNP: N-terminal pro-B-type natriuretic peptide; SARS-CoV-2: Severe acute respiratory syndrome coronavirus 2

segments decreased modestly but significantly, and the frequency of pericardial effusion was significantly reduced. Detailed paired comparisons are summarized in Table 4. The magnitude of change was independent of the follow-up interval for LVEF, T2, ECV, and LGE extent (all p>0.3); for native T1, a longer interval was associated with a greater reduction (ρ=−0.38, p=0.026), consistent with progressive reduction over time. Temporal changes in the LGE pattern are illustrated in Figure 2. The corresponding pattern transition matrix is provided in Supplementary Table S3. In the paired follow-up subgroup, higher baseline peak troponin was associated with greater residual LGE extent at follow-up (ρ=0.39, p=0.012) and showed a non-significant trend toward persistent LGE

(median 282 vs. 145 ng/L; p=0.088), whereas peak CRP and NT-proBNP were not associated.

Discussion

In this single-center cohort of pediatric patients with acute myocarditis, baseline CMR frequently demonstrated ventricular dysfunction, LGE, and pericardial effusion. The prevalence of LGE in our cohort appears relatively high compared with previously reported pediatric series, which may be related to differences in patient selection and timing of imaging[15,20,21]. Among patients who underwent repeat imaging, both ventricular function and myocardial tissue characterization parameters improved significantly over time, consistent with previous studies reporting gradual

Table 2. Baseline cardiac magnetic resonance findings in the study cohort (n=139)

Variable	Value
Ventricular function	
Left ventricular ejection fraction (%), median (IQR)	50 (33–58)
Reduced LVEF (<55%), n (%)	90 (64.7%)
Tissue characterization	
Native T1, ms, median (IQR) (n=128)	1130 (1044–1201)
T2 relaxation time, ms, median (IQR) (n=130)	62 (53–69)
Extracellular volume (%), median (IQR) (n=128)	36 (30–46)
Late gadolinium enhancement	
LGE present, n (%)	127 (91.4%)
LGE extent (%), median (IQR) [range] (n=139)	4 (3–5) [1-32]
LGE extent >10%, n (%)	9 (6.5%)
Additional findings	
Pericardial effusion, n (%)	22 (15.8%)

CMR: Cardiac magnetic resonance, IQR: Interquartile range, LGE: Late gadolinium enhancement, LVEF: Left ventricular ejection fraction

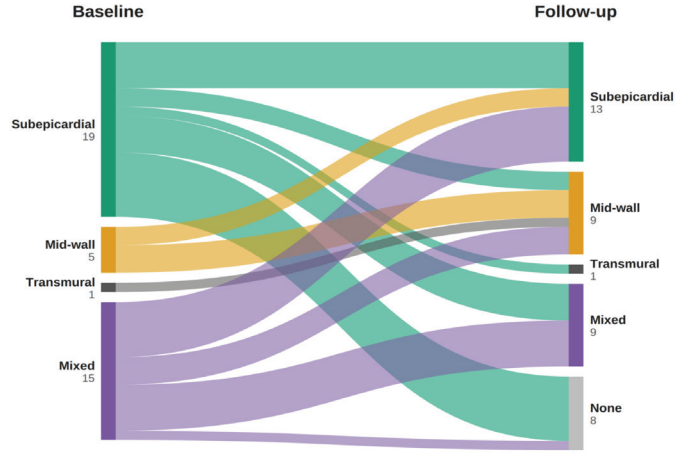

Figure 2. Temporal changes in late gadolinium enhancement (LGE) distribution patterns between baseline and follow-up cardiac magnetic resonance imaging. The Sankey-style diagram illustrates transitions in myocardial LGE patterns among pediatric patients with acute myocarditis. Most patients demonstrated reductions in both the extent and number of myocardial segments involved at follow-up, whereas a substantial proportion showed persistent LGE despite overall improvement in ventricular function and tissue characterization parameters

Table 3. Age-adjusted correlations between laboratory markers and baseline CMR parameters (n=139)

Biomarker	CMR parameter	n	Unadj. ρ	Age-adjusted ρ (95% CI)	q
Peak troponin	Native T1	128	−0.28	+0.09 (−0.09, +0.27)	0.556
	T2	130	−0.20	−0.00 (−0.19, +0.18)	0.985
	ECV	128	−0.06	+0.06 (−0.12, +0.24)	0.798
	LGE extent	139	+0.36	**+0.39 (+0.24, +0.52)**	**<0.001***
Peak CRP	Native T1	128	−0.20	+0.03 (−0.15, +0.21)	0.905
	T2	130	−0.15	−0.02 (−0.20, +0.17)	0.921
	ECV	128	−0.09	−0.04 (−0.22, +0.14)	0.905
	LGE extent	139	+0.23	**+0.22 (+0.06, +0.38)**	**0.020***
NT-proBNP	Native T1	128	+0.62	**+0.36 (+0.19, +0.51)**	**<0.001***
	T2	130	+0.42	**+0.28 (+0.10, +0.44)**	**0.007***
	ECV	128	+0.21	+0.09 (−0.09, +0.27)	0.556
	LGE extent	139	+0.16	**+0.32 (+0.16, +0.46)**	**<0.001***

*ρ: partial Spearman correlation coefficient adjusted for age, q: Benjamini–Hochberg false-discovery-rate–adjusted p-value; *q<0.05. Unadjusted ρ shown for transparency; the negative unadjusted troponin/CRP–T1/T2 correlations reflected age confounding and were abolished after adjustment, CMR: Cardiac magnetic resonance, CRP: C-reactive protein, ECV: Extracellular volume, LGE: Late gadolinium enhancement, CI: Confidence interval, FDR: False discovery rate*

Table 4. Comparison of baseline and follow-up cardiac magnetic resonance findings (n=40)

Variable	Baseline	Follow-up	Effect size	p-value
LVEF (%)	54 (38–60)	58 (44–63)	r = 1.00	<0.001
Native T1 (ms) (n=34)	1118 (1046–1209)	1037 (1005–1137)	r = −0.61	0.002
T2 (ms) (n=34)	62 (48–86)	52 (46–64)	r = −0.51	0.013
ECV (%) (n=33)	42 (30.8–51)	30.6 (27-36)	r = −0.76	<0.001
LGE extent (%), among LGE-positive	4 (4–6)	3 (2–5)	r = −0.99	<0.001
LGE present, n (%)	40 (100%)	32 (80%)	—	0.008
Number of involved segments	2 (2–4)	2 (1–3)	r = −0.53	0.031
Multi-segment involvement, n (%)	31 (77.5%)	21 (52.5%)	—	0.006
Pericardial effusion, n (%)	10 (25%)	0 (0%)	—	0.002
Myocardial localization (septal/lateral/inferior/anterior/apical/multiple/none)	7/17/6/0/2/8/0	4/16/8/0/1/3/8	—	N/A
Myocardial pattern (subepicardial/mid-wall/transmural/mixed/none)	19/5/1/15/0	13/9/1/9/8	—	N/A

Data are presented as median (IQR) or n (%).
Effect sizes are the matched-pairs rank-biserial correlation (r) for continuous variables (Wilcoxon signed-rank test); categorical variables were compared with the McNemar test. LGE extent is reported among LGE-positive patients at each time point; for the paired comparison, LGE-negative studies were assigned an extent of 0%. The matched-pairs rank-biserial correlation reflects the consistency of the direction of change rather than its magnitude. An r of 1.00 for LVEF indicates that all 40 patients showed an increase in LVEF at follow-up (median change +4 percentage points; range +3 to +9), with no patient showing a decrease or no change.
CMR: Cardiac magnetic resonance, ECV: Extracellular volume, IQR: Interquartile range, LGE: Late gadolinium enhancement, LVEF: Left ventricular ejection fraction

resolution of inflammatory CMR findings[18,20]. Follow-up CMR was performed at a median of 144 days after baseline CMR, and although LGE prevalence, extent, and segmental involvement decreased, residual enhancement remained detectable in a substantial proportion of patients, consistent with prior studies showing that LGE may regress over time but frequently does not completely resolve in the early follow-up period[17-19]. These findings suggest that myocardial recovery on imaging may lag behind functional improvement, particularly during early follow-up[17,20]. Importantly, the follow-up subgroup was not representative of the entire cohort: patients who underwent repeat imaging had more pronounced baseline abnormalities, including a higher prevalence of LGE (100% vs. 87.9%, p=0.019) and higher T2 values (p=0.024), indicating that repeat CMR was preferentially performed in more severely affected children. This has two implications. First, the significant regression in tissue parameters observed here occurred despite this enrichment, indicating that imaging improvement occurs even among the more severely affected patients. Second, because children with milder baseline involvement were underrepresented, the magnitude of change reported here should not be extrapolated to the entire cohort or to unselected pediatric myocarditis populations, and the proportion of patients with persistent LGE may differ in such populations.

Cardiac magnetic resonance plays a central role in the diagnosis of acute myocarditis by enabling comprehensive assessment of myocardial inflammation, edema, and injury[12,22]. The updated Lake Louise Criteria, incorporating parametric mapping techniques such as native T1, T2, and ECV, have substantially improved diagnostic accuracy compared with conventional imaging approaches[12,15,23]. These techniques allow detection of both focal and diffuse myocardial involvement, overcoming the limitations of LGE, which primarily reflects regional injury[12,15]. In pediatric populations, where clinical presentation may be heterogeneous and noninvasive diagnostic tools are particularly important, CMR provides a valuable framework for diagnosis and disease characterization[2,20,24]. Our findings, demonstrating a high prevalence of abnormal tissue characterization parameters at baseline, further support the value of multiparametric CMR in capturing the extent of myocardial involvement in acute pediatric myocarditis.

LGE has been consistently associated with myocardial injury and has emerged as an important imaging marker with potential prognostic implications in acute myocarditis[12,25]. Previous studies have shown that the presence and extent of LGE may be associated with adverse clinical outcomes, including persistent ventricular dysfunction and the development of chronic myocardial damage[20,26]. In pediatric populations, although long-term outcome data remain limited, persistent LGE on follow-up imaging has been reported even in patients with clinical recovery, suggesting that residual enhancement may be observed even after clinical recovery[19,21,27]. Our findings support the concept that LGE represents a dynamic process reflecting ongoing myocardial remodeling rather than a static marker of irreversible injury, particularly in the early phase of recovery. Because clinical outcomes were not assessed, the prognostic significance of the residual enhancement observed in our cohort cannot be determined, and its persistence should not be equated with an adverse prognosis in the absence of outcome data.

Beyond their descriptive value, our tissue-characterization findings were associated with circulating markers of myocardial injury and wall stress. After adjustment for age, higher peak troponin and CRP were associated with greater LGE burden, and higher NT-proBNP was associated with higher native T1 and T2 and greater LGE extent, supporting a biological link between the intensity of the acute inflammatory insult and the degree of detectable myocardial involvement[18,19]. Higher baseline troponin was also associated with greater residual LGE at follow-up, consistent with the concept that more severe acute injury predisposes to residual replacement fibrosis[16,26]. Because age was strongly associated with both native T1 and peak troponin in our cohort, consistent with reports of age-related variation in pediatric myocardial T1[28], these associations were adjusted for age; unadjusted analyses were confounded by age and would have yielded biologically implausible inverse correlations, underscoring the importance of age adjustment in pediatric CMR studies. These associations should be regarded as exploratory, and quantitative LGE assessment in children remains constrained by dependence on the thresholding technique, partial-volume effects in the thin pediatric myocardium, and the absence of validated age-specific reference ranges[13,23].

Despite the growing use of CMR in the evaluation of acute myocarditis, data regarding the comprehensive assessment of both baseline and follow-up multiparametric CMR findings in pediatric populations remain limited. Most available studies have focused either on diagnostic performance or on selected imaging markers, with relatively few addressing the temporal evolution of myocardial tissue characteristics using a multiparametric approach[19-21,27,29]. In this context, our study provides a detailed evaluation of functional and tissue-level changes, integrating ventricular function, mapping parameters, and LGE characteristics in a pediatric cohort. The combined assessment of baseline abnormalities and their evolution over time offers a more comprehensive understanding of myocardial involvement and recovery patterns in children with acute myocarditis. These findings may help inform follow-up imaging strategies, although prospective studies with longer follow-up and systematically collected clinical outcomes are required before persistent imaging abnormalities can be used for risk stratification. Although our study did not specifically evaluate clinical outcomes, the observed improvement in imaging parameters alongside persistent LGE may provide insight into ongoing myocardial remodeling and may warrant further investigation.

Study Limitations

This study has several limitations. Its retrospective, single-center design may limit generalizability. Because CMR—including the tissue markers used in the Lake Louise Criteria—contributed to the diagnosis, the reported prevalences of CMR abnormalities reflect incorporation bias rather than diagnostic accuracy. The cohort also represents a CMR-imaged subset of all clinically diagnosed patients. Patients who underwent follow-up CMR had greater baseline myocardial abnormality (higher

LGE prevalence and T2 values), indicating that follow-up preferentially included more severely affected patients; the follow-up findings may therefore overrepresent patients with greater baseline involvement and not generalize to the full cohort. The follow-up interval was also not standardized, which may have influenced the degree of recovery observed. Methodologically, left ventricular function by echocardiography was estimated using the M-mode Teichholz method, which, together with a higher normal reference range, greater sensitivity, and a different threshold of CMR, likely explains the lower prevalence of systolic dysfunction on echocardiography; ventricular volumes and some mapping parameters were not available in all patients; CMR was interpreted by a single reader, so interobserver reproducibility was not assessed. The retrospective categorization of admission electrocardiograms may underestimate transient abnormalities. Most importantly, long-term clinical follow-up data—including functional status, arrhythmic events, hospitalization, and the need for heart failure therapy—were not available. Therefore, the residual LGE observed at follow-up cannot be related to clinical outcome, and its prognostic significance in this cohort remains undetermined; this, together with the exploratory nature of the association analyses, should be taken into account when interpreting the persistent imaging abnormalities reported here.

Conclusion

Multiparametric CMR imaging provides a comprehensive assessment of myocardial involvement in pediatric acute myocarditis. Significant improvement in ventricular function and tissue characterization parameters is observed over time. However, residual abnormalities, particularly LGE, may persist during follow-up. These findings suggest that imaging recovery may lag behind the recovery of ventricular function. CMR may therefore be a valuable tool not only for diagnosis but also for follow-up evaluation in this patient population, although the optimal timing and the prognostic value of persistent imaging abnormalities require prospective evaluation.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Ethics Committee of University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital (approval no: 2025/355, date: November 26, 2025).

Informed Consent: Informed consent was not required due to the retrospective nature of the study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Kangel D, Oğuz AS, Genç HZ, Öztürk E, Concept: Kangel D, Özkök S, Tanıdır İC, Öztürk E, Design: Kangel D, Kartal YC, Tanıdır İC, Data Collection and/or Processing: Kangel D, Oğuz AS, Özkök S, Tanıdır İC, Analysis and/or Interpretation: Oğuz AS, Öztürk E, Literature Search: Kangel D, Genç HZ, Writing: Kangel D, Tanıdır İC.

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Supplementary Tables: https://d2v96fxpocvxx.cloudfront.net/beb8919b-f013-4ea1-b1c8-40332e840fe1/content-images/46c5218b-0dde-47c8-89f3-b4bbfc8e29d8.pdf

References

1. Caforio AL, Pankuweit S, Arbustini E, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2013;34:2636-48.
2. Law YM, Lal AK, Chen S, et al. Diagnosis and management of myocarditis in children: a scientific statement from the American Heart Association. Circulation. 2021;144:e123-e35.
3. Ammirati E, Moslehi JJ. Diagnosis and treatment of acute myocarditis: a review. JAMA. 2023;329:1098-113.
4. Drazner MH, Bozkurt B, Cooper LT, et al. 2024 ACC Expert consensus decision pathway on strategies and criteria for the diagnosis and management

of myocarditis: a report of the American College of Cardiology Solution Set Oversight Committee. J Am Coll Cardiol. 2025;85:391-431.

5. Arola A, Pikkarainen E, Sipilä JO, et al. Occurrence and features of childhood myocarditis: a nationwide study in Finland. J Am Heart Assoc. 2017;6:e005306.
6. Vasudeva R, Bhatt P, Lilje C, et al. Trends in acute myocarditis related pediatric hospitalizations in the United States, 2007–2016. Am J Cardiol. 2021;149:95-102.
7. Harris KM, Mackey-Bojack S, Bennett M, et al. Sudden unexpected death due to myocarditis in young people, including athletes. Am J Cardiol. 2021;143:131-4.
8. Kindermann I, Kindermann M, Kandolf R, et al. Predictors of outcome in patients with suspected myocarditis. Circulation. 2008;118:639-48.
9. Hutchinson Z, Law Y. Myocarditis in children: diagnosis and management. JHLT Open. 2025:100332.
10. Yao Z, Liang M, Zhu S. Infectious factors in myocarditis: a comprehensive review of common and rare pathogens. Egypt Heart J. 2024;76:64.
11. Cooper LT, Baughman KL, Feldman AM, et al. The role of endomyocardial biopsy in the management of cardiovascular disease: a scientific statement from the American Heart Association, the American College of Cardiology, and the European Society of Cardiology Endorsed by the Heart Failure Society of America and the Heart Failure Association of the European Society of Cardiology. Eur Heart J. 2007;28:3076-93.
12. Ferreira VM, Schulz-Menger J, Holmvang G, et al. Cardiovascular magnetic resonance in nonischemic myocardial inflammation: expert recommendations. J Am Coll Cardiol. 2018;72:3158-76.
13. Schulz-Menger J, Bluemke DA, Bremerich J, et al. Standardized image interpretation and post-processing in cardiovascular magnetic resonance-2020 update: society for cardiovascular magnetic resonance (SCMR): board of trustees task force on standardized post-processing. J Cardiovasc Magn Reson. 2020;22:19.
14. O'Halloran CP, Robinson JD, Watanabe K, et al. Magnetic resonance imaging in pediatric myocarditis: trends and associations with cost and outcome. JACC Cardiovasc Imaging. 2022;15:1230-8.
15. Isaak A, Bischoff LM, Faron A, et al. Multiparametric cardiac magnetic resonance imaging in pediatric and adolescent patients with acute myocarditis. Pediatr Radiol. 2021;51:2470-80.
16. Grigoratos C, Di Bella G, Aquaro GD. Diagnostic and prognostic role of cardiac magnetic resonance in acute myocarditis. Heart Fail Rev. 2019;24:81-90.
17. Aquaro GD, Ghebru Habtemicael Y, Camastra G, et al. Prognostic value of repeating cardiac magnetic resonance in patients with acute myocarditis. J Am Coll Cardiol. 2019;74:2439-48.
18. Luetkens JA, Homsi R, Dabir D, et al. Comprehensive cardiac magnetic resonance for short-term follow-up in acute myocarditis. J Am Heart Assoc. 2016;5:e003603.
19. Yukcu B, Aydemir MM, Balci M, et al. Baseline and follow-up cardiac magnetic resonance imaging findings in children with acute myocarditis and factors associated with late gadolinium enhancement. J Clin Med. 2024;14:189.
20. Martins DS, Ait-Ali L, Khraiche D, et al. Evolution of acute myocarditis in a pediatric population: An MRI-based study. Int J Cardiol. 2021;329:226-33.
21. Dubey S, Agarwal A, Nguyen S, et al. Persistence of late gadolinium enhancement on follow-up CMR imaging in children with acute myocarditis. Pediatr Cardiol. 2020;41:1777-82.
22. Kotanidis CP, Bazmpani M-A, Haidich A-B, et al. Diagnostic accuracy of cardiovascular magnetic resonance in acute myocarditis: a systematic review and meta-analysis. JACC Cardiovascular Imaging. 2018;11:1583-90.
23. Cornicelli MD, Rigsby CK, Rychlik K, et al. Diagnostic performance of cardiovascular magnetic resonance native T1 and T2 mapping in pediatric patients with acute myocarditis. J Cardiovasc Magn Reson. 2019;21:40.
24. House AV, Grosse-Wortmann L. Cardiac magnetic resonance imaging assessment of myocardial disease in children and adolescents. Cardiovasc Diagn Ther. 2025;15:888-97.
25. Popa A, Cionca C, Agoston R, et al. The role of magnetic resonance imaging in risk stratification of patients with acute myocarditis. Diagnostics. 2024;14:1426.
26. Gannon MP, Schaub E, Grines CL, et al. State of the art: evaluation and prognostication of myocarditis using cardiac MRI. J Magn Reson Imaging. 2019;49:e122-e31.
27. Malek LA, Kamińska H, Barczuk-Falęcka M, et al. Children with acute myocarditis often have persistent subclinical changes as revealed by cardiac magnetic resonance. J Magn Reson Imaging. 2020;52:488-96.
28. Alsaied T, Tseng SY, Siddiqui S, et al. Pediatric myocardial T1 and T2 value associations with age and heart rate at 1.5 T. Pediatric Cardiol. 2021;42:269-77.
29. Pitak B, Opgen-Rhein B, Schubert S, et al. Cardiovascular magnetic resonance in children with suspected myocarditis: current practice and applicability of adult protocols. Cardiol Young. 2022;32:1957-65.