Cardiology Journal

VIA MEDICA

ORIGINAL ARTICLE

DOI: 10.5603/cj.97866

ISSN 1897–5593
eISSN 1898–018X

A real-life clinical application of cardiac magnetic resonance imaging in patients with acute myocarditis — one-center observational retrospective study

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Abstract

Background: *The diagnosis of acute myocarditis is complex, with cardiac magnetic resonance (CMR) being a recommended diagnostic method. This study aimed to evaluate the real-life use of CMR in the diagnosis of acute myocarditis and to correlate CMR results with the degree of myocardial damage.*

Methods: *This is a retrospective, observational tertiary single-center study of 90 consecutive patients (F/M: 18/72, mean age: 39 ± 14 years) hospitalized between 2015–2022 with a clinical diagnosis of acute myocarditis. The study population was divided into two groups: patients who underwent CMR+ and those who did not undergo CMR — In the CMR+ group, various sequences, including T1/T2-weighted imaging, late gadolinium enhancement (LGE), and mapping techniques, were used to assess myocardial inflammation and damage.*

Results: *CMR was performed in 39 patients (43.3%, F/M: 10/29, mean age: 41 ± 16 years). In this group, myocardial edema (increased T2 signal intensity) was detected in 29 patients, and LGE (signal intensity 2 standard deviations cabove normal on T1 images) was found in 39 patients. Diagnosis based on Lake Louise Criteria was possible in 29 cases. Edema negatively correlated with TnT levels ($r = –0.412$, $p < 0.05$) and positively with the number of LGE segments ($r = 0.372$, $p < 0.05$). Significant correlations were found between LVEF and LGE mass ($r = –0.360$, $p < 0.05$), and maximal TnT levels ($r = –0.38$, $p < 0.05$). CMR+ patients had lower myocardial damage markers and CRP concentrations compared to CMR– patients.*

Conclusions: *CMR is underused in diagnosing acute myocarditis. Myocardial damage markers correlate with CMR-detected edema and volumetric measures, but not LGE extent. More research is needed to enhance risk assessment and treatment.*

Keywords: acute myocarditis, cardiac magnetic resonance, late gadolinium enhancement, region of interest mass, Lake Louise Criteria

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Received: 16.10.2023 Accepted: 15.09.2024 Early publication date: 13.11.2024

Introduction

Acute myocarditis is an inflammatory disease characterized by myocardial edema, necrosis, and inflammatory cell infiltration. Despite a high mortality rate of up to 50%, its classification, diagnosis, and treatment are still being developed [1]. Clinical presentation and diagnostic/therapeutic processes vary widely between centers.

The gold standard for diagnosis is myocardial biopsy, but its reliability is limited due to difficulty in sampling the diseased area. Its invasive nature and potential complications further restrict its use, reserving the procedure for high-risk patients (e.g., those with cardiogenic shock or suspected eosinophilic or giant-cell myocarditis) and not recommended for low-risk cases [2].

While the cause and clinical manifestations of myocarditis are often unclear, myocarditis can be clearly visualized with cardiovascular magnetic resonance imaging (CMR). CMR is a non-invasive imaging modality that allows for the assessment of both volumetric values and myocardial changes. Using CMR both dimensions and function of the heart chambers assessment and the various tissue characterization techniques are available. Tissue characterization in CMR involves different imaging sequences to assess the composition and condition of the myocardium. T1-weighted imaging, with the assessment of late gadolinium enhancement (LGE), allows visualization of permanent damage to the myocardium due to the replacement of myocardial cells by fibrous tissue. T2-weighted imaging shows myocardial edema, reflecting reversible myocardial damage and potentially present even in the absence of LGE. Extracellular volume fraction (ECV) calculation, though not the focus of this study, is another marker of myocardial tissue remodeling and provides a physiologically intuitive unit of measurement [2–5].

The Lake Louise Criteria (LLC) are a set of diagnostic criteria established to standardize the diagnosis of myocarditis using CMR. The updated LLC established LGE and T2 weighted imaging techniques and demonstrated the growing importance of quantitative mapping in diagnosing myocarditis [2, 5].

According to the updated LLC, CMR findings are consistent with myocarditis if at least two of the following criteria are met: a regional or global increase in myocardial signal intensity on T2-weighted images consistent with edema; an early increase in global gadolinium myocardial gain factor between myocardium and skeletal muscles in T1-weighted images; or at least one focus of non-ischemic regional redistribution on inverse resting gadolinium-enhanced T1-weighted images [6, 7].

Although the LLC, first published in 2009 and updated in 2018, are the recommended criteria for the definitive diagnosis of clinically suspected acute myocarditis, they are not always applied in practice [8]. There are several reasons for this. Firstly, the use of LLC is most relevant in the acute phase of the disease, and applying it in different stages of myocarditis might result in divergent or even misinterpreted evaluations. Secondly, CMR findings can vary depending on the experience and protocols of the CMR laboratory, leading to inconsistencies in interpretation. Furthermore, logistical challenges such as the availability of CMR technology and trained personnel can also limit the routine application of LLC. In most cases, a history of viral infection and laboratory tests for myocardial damage should be interpreted together with imaging results from methods such as echocardiography and CMR. However, using retrospective data, we aim to summarize the tertiary center experience in CMR use. This will contribute data on improving the myocarditis diagnostic process, especially since, despite recent advances in imaging techniques, the diagnosis, monitoring, and prognostication of patients in this clinical setting remain challenging.

The study aimed to evaluate real-life CMR use in the diagnosis of acute myocarditis and refer CMR results to the degree of myocardial damage.

Methods

Patient population

This study was approved by the research ethics board. It is a retrospective and observational study of 90 consecutive patients (F/M: 18/72, mean age: 38.7 ± 14.2 years) hospitalized in the 1st Department of Cardiology in a tertiary cardiovascular centre between 2015 and 2022 with a clinical diagnosis of acute myocarditis. The clinical diagnosis of acute myocarditis was based on case history, markers of inflammation (hsCRP) and myocardial injury (troponin, CK-MB levels), imaging non-invasive methods (echocardiography and CMR), and coronary angiography ruling out coronary artery disease. In this study, ECG findings of patients were not described, as they did not play a significant role in the present investigation.

Patients with contraindications for CMR (acute heart failure, cardiogenic shock, respiratory insufficiency, eGFR < 30 mL/kg/1.73 m^2, claustrophobia), patients with previous myocarditis and comorbidities

significantly influencing heart function (coronary artery disease, valve heart disease, congenital heart disease, cardiomyopathies), patients with LGE suggestive of myocardial ischemia/infarction (subendocardial or transmural) were excluded from the analysis.

Clinical characteristics included: demographic data, BMI (body mass index), duration of hospitalization, co-morbidities (RTI — respiratory tract infection; systemic hypertension), heart rate, blood tests (maximal levels of CRP, troponin T, CK-MB, D-dimer, GFR, fasting glucose), echocardiographic parameters (LVEF — left ventricle ejection fraction; LVESD — LV end-systolic volume; LVEDV — LV end-diastolic volume; LA area — left atrial area; RA area — right atrial area).

Taking into consideration CMR use, the study population was divided into two groups: those who underwent CMR (CMR+) and those who did not (CMR–). Comparisons of the clinical characteristics of the study groups as well as the analysis of the CMR results were done. The CMR results were analysed regarding the following aspects: number of LV LGE occupied segments, markers of myocardial damage (maximal levels of CRP, troponin T, CK-MB, LVEF in TTE and CMR, RVEF in CMR), and pharmacotherapy implemented during hospitalization and administered at the moment of CMR imaging (including: angiotensin-converting-enzyme inhibitors/ACEI, angiotensin receptor neprilysin inhibitor/ARNI, mineralocorticoid receptor antagonist/MRA, loop diuretics and beta-blockers).

Patients with mildly expressed symptoms of myocarditis in the current study refer to those presenting with minimal symptoms, such as exercise limitation classified as NYHA I/II, non-specific chest pains, and no resting dyspnea or edema.

CMR protocol and imaging analysis

CMR imaging was obtained during the first 10 days of hospitalization. The CMR images were acquired on 1.5-T systems (Optima MR450w, GE Healthcare) with a dedicated phased-array cardiac coil or body matrix coil using an electrocardiography-gated breath-hold protocol. Cine-CMR sequences included steady-state free precession (SSFP) imaging, while T2-weighted imaging sequences used a triple inversion recovery technique. Diagnosis of myocarditis was based on cine-CMR, T2-weighted imaging, and T1-weighted late gadolinium enhancement imaging.

Data on myocardial edema and LGE were analyzed by a specialist with many years of experience in cardiac magnetic resonance imaging. Due to differences in protocols over the evaluated period, pulmonary congestion was not consistently revealed in all exams and thus was not included in the analysis.

In the present study, the number and percentage of occupied left ventricular (LV) LGE segments were counted using the Cardiac VX program in a short-axis projection. American Heart Association (AHA) 16-segment model for segmentation was used.

For image analysis, T2-weighted imaging was used to detect myocardial edema by measuring the signal intensity, which is considered elevated if it is more than two standard deviations (2SD) above that of normal myocardium. LGE imaging was performed to detect areas of fibrosis, with LGE defined as regions with signal intensity greater than 2SD above that of normal myocardium.

To specifically determine the amount of LGE, the region of interest (ROI) mass (exact weight of LGE in the heart muscle) and ROI % (ratio of ROI mass to LV mass) were calculated. For the program to count the number of occupied segments in a given examination, the endocardium was marked, then the border of the epicardium, thus separating the myocardium. Also marked were the intersection point between the LV and right ventricle (RV), the so-called threshold, and proceeded in the same way in each segment.

The enhancement pattern in LGE was also assessed, with particular attention to non-ischemic patterns such as mid-wall, epicardial, or patchy enhancement, which are characteristic of myocarditis. The ROI was selected by manually outlining the area of hyperenhancement on LGE images, ensuring it was above the 2SD threshold relative to normal myocardium.

These detailed methods allowed quantification of the extent of myocardial damage and to correlate it with clinical and laboratory findings, providing a comprehensive assessment of myocarditis in the patient population.

Statistical analysis

The study population was first dichotomized into 2 groups of patients who underwent CMR and those who did not. Clinical characteristics and outcomes were compared between groups. Continuous variables were presented as mean ± standard deviation or median ± inter quartile (IQ) and categorical as absolute values and percentages. Normality was verified using the Shapiro–Wilk test. The comparisons of groups were based

on students' two-sample t-tests or nonparametric Mann–Whitney U tests, as appropriate. The differences in proportions between groups were analysed using the $\chi 2$ test. A p-value ≤ 0.05 was considered statistically significant for all tests. To analyse the correlation, the Pearson's and/or Spearman's rank correlation coefficients were used. All other analyses were performed using MedCalc® version 20.015 software.

Results

Clinical characteristic: CMR(+) vs. CMR(–) groups

Baseline characteristics of groups of patients with CMR vs. those without CMR done during the diagnostic process were summarized in Table 1. Patients who underwent CMR (CMR+) were more likely to have hypertension, lower heart rates, and lower levels of markers of myocardial injury and inflammation (TnT, CK-MB, CRP) compared to those who did not undergo CMR (CMR–). They also had different cardiac structural parameters, with higher LVEDV and larger LA areas. The differences in medication use were not statistically significant between the groups (Figure 1).

CMR results — general data

In the CMR(+) group, the oedema was revealed in 29 (74%) and LGE in 39 (100%) patients. It allowed for the diagnosis of acute myocarditis based on LLC in 29 (74%) cases.

Several LV segments with LGE were also analysed — the mean number of LGE-occupied LV segments was 11.6 ± 2.3 (range: 6–16). The average value of ROI mass was 10.2 ± 13.4 g, and ROI % was 9.19 ± 0.6%.

The volumetric CMR parameters of the whole CMR (+) group were as follows: the LVEF was 51.8% ± 11.8. The LV mass value was estimated to be 128 ± 45.7 g/m^2. Mean LV ESV and LV EDV were respectively: 83.8 ± 53.2 and 162 ± 54.3 mL; the mean stroke volume (SV) was 78.7 ± 19mL. The right ventricle volumetric parameters were as follows: right ventricular EF (RVEF): 57.5% ± 6.3, RV end-systolic volume (RVESV): 53 ± 16.6 mL, RV end-diastolic volume (RVEDV): 120.2 ± ± 32.4 mL.

CMR results- number of LGE-occupied segments and clinical characteristics

Patients were divided into two subgroups according to LGE-occupied segments. The number of LV segments with LGE ranged from 6–10 in 12 (30.8%) and 11–16 in 27 (69.2%) patients. There were no differences regarding clinical characteristics between the subgroups, particularly in the biochemical markers of myocardial damage.

A comparison of the subgroups is presented in Table 2.

Table 1. Characteristics of the study group

Group characteristics	CMR (+)* n = 39	CMR(–)* n = 51	P-value
Sex [F/M], n [%]	10 (25.6%)/ /29 (24.6%)	8 (15.7%)/ /43 (84.3%)	NS
Age [years]	40.8 ± 16.2	37.1 ± 14.3	NS
BMI [kg/m^2]	24.4 ± 4.2	24.3 ± 5.5	NS
Duration of hospitalization [days]	6.3 ± 3	5 ± 1.6	NS
Comorbidities			
RTIs, n [%]	11 (28%)	29 (56%)	0.05 < p < 0.10
Hypertension, n [%]	33 (84.6%)	18 (35%)	0.001
Heart rate [bpm]	68.51 ± 12.4	78.5 ± 16.9	< 0.005
Blood tests			
TnT [μg/mL]	0.36 ± 0.7	0.70 ± 1.2	< 0.01
CK-MB [IU/L]	20.41 ± 11.6	43.84 ± 24.6	< 0.005
CRP [mg/L]	16.48 ± 27.8	58.76 ± 72.1	< 0.001
Glucose level [mg/dL]	96.33 ± 19.1	107.18 ± 26.1	0.05 < p < 0.10
eGFR [mL/ /min/1,73m^2]	112.93 ± 38	120 ± 39	NS
Standard TTE parameters			
LVEF [%]	49.15 ± 0.5	53.18 ± 0.5	NS
LVESV [mL]	89.24 ± 53.0	93.92 ± 34.7	< 0.005
LVEDV [mL]	138.59 ± 55.2	114.51 ± 34.7	< 0.01
LA area [cm^2]	19.6 ± 4.3	17.3 ± 3.3	< 0.05
RA area [cm^2]	14.9 ± 2.7	15.4 ± 3.1	NS
Medicines			
ACEI, n [%]	29 (74%)	23 (46%)	NS
ARNI, n [%]	8 (20%)	3 (6%)	NS
MRA, n [%]	23 (59%)	15 (29%)	NS

*CMR(+) patients diagnosed with myocarditis who had CMR done during their hospitalization; CMR(–) patients diagnosed with myocarditis who did not have CMR done during their hospitalization; ACEI — angiotensin-converting-enzyme inhibitors, ARNI — angiotensin receptor neprilysin inhibitor, CK-MB — creatine kinase-MB, CMR — cardiac magnetic resonance, CRP — C-reactive protein, GFR — glomerular filtration rate, LA area — left atrium area, LESV — left ventricular end-systolic volume, LVEDV — left ventricular end-diastolic volume, LVEF — left ventricular ejection fraction, MRA — mineralocorticoid receptor antagonist, RA area — right atrium area, RTIs — respiratory tract infections, TnT — troponin T

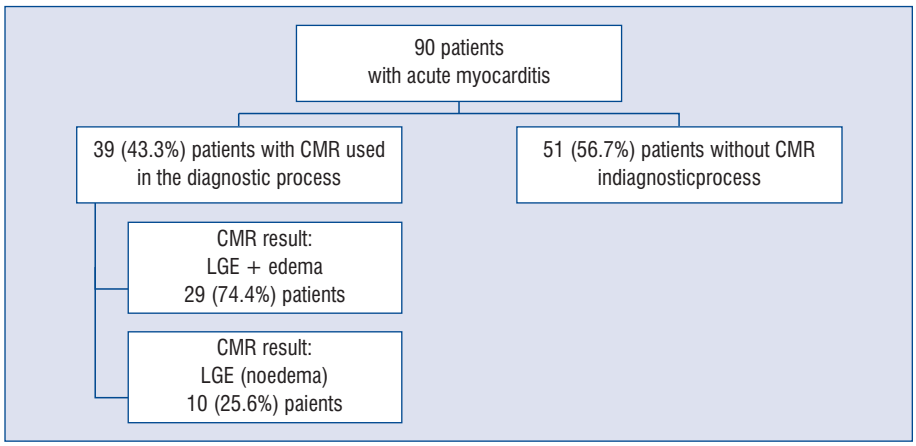

Figure 1. CMR in the diagnostic process of patients with acute myocarditis; CMR — cardiac magnetic resonance; LGE — late gadolinium enhancement

Table 2. Comparison of the subgroups of patients based on the number of LGE occupied LV segments in CMR

Variable	6–10 LGE occupied segments, n = 12	11–16 LGE occupied segments, n = 27	P-value
Sex [F/M]	3 (25%)/9 (75%)	7 (25.9%)/20 (74.1%)	NS
Age [years]	39.5 ± 13.3	41.3 ± 17.6	NS
BMI [kg/m^2]	23.2 ± 2.3	24.9 ± 4.7	NS
Duration of hospitalization [days]	5.3 ± 1.9	6.7 ± 3.4	NS
Comorbidoties			
RTIs, n [%]	2 (17%)	9 (33%)	NS
Hypertension, n [%]	10 (83%)	23 (85%)	NS
Heart Rate [bpm]	69.7 ± 11.2	68 ± 13	NS
Blood tests			
TnT [μg/mL]	0.56 ± 1.0	0.26 ± 0.4	NS
CK-MB [IU/L]	22.75 ± 16	19.37 ± 9	NS
CRP [mg/L]	0.33 ± 20.2	0.52 ± 30.9	NS
eGFR [ml/min/1,73m^2]	124.13 ± 36.8	108 ± 38.3	NS
Standard TTE parameters			
LVEF [%]	47.1 ± 11.7	53.6 ± 6.9	NS
LVESV [mL]	94.4 ± 59.7	61.4 ± 14.8	< 0.005
LVEDV [mL]	159.62 ± 62.9	134.5 ± 18.8	< 0.01
LA area [cm^2]	21.43 ± 5.0	16.13 ± 2.4	NS
RA area [cm^2]	15.84 ± 4.8	12.92 ± 1.4	NS
CMR			
ROI MASS [g]	6.44 ± 5.8	12.24 ± 15.5	NS
ROI [%]	6.97 ± 5.5	10.79 ± 10.4	NS
Edema, n [%]	7 (58%)	22 (82%)	NS
Medicines			
ACEI, n [%]	8 (68%)	22 (80%)	NS
ARNI, n [%]	0 (0%)	5 (19%)	NS
MRA, n (%)	7 (58%)	16 (59%)	NS
Loop diuretic, n [%]	3 (22%)	0 (0%)	NS
Beta-blocker, n [%]	7 (58%)	22 (81%)	NS

ACEI — angiotensin-converting-enzyme inhibitors, ARNI — angiotensin receptor neprilysin inhibitor, CK-MB — creatine kinase-MB, CMR — cardiac magnetic resonance, CRP — C-reactive protein, GFR — glomerular filtration rate, LA area — left atrium area, LESV — left ventricular end-systolic volume, LVEDV — left ventricular end-diastolic volume, LVEF — left ventricular ejection fraction, MRA — mineralocorticoid receptor antagonist, RA area — right atrium area, RTIs — respiratory tract infections, TnT — troponin T

Table 3. Data dependency between different variables

CMR variables:	CMR: LGE — ROI MASS	P-value	CMR: edema	P-value	CMR: LVEF	P-value
Blood tests						
TnT	r = 0.05	NS	**r = –0.41** r_s = –0.23	< 0.05	**r = –0.33** **r_s = –0.38**	0.05
CK-MB	r = –0.04	NS	r = –0.27	NS	r = 0.19	NS
CRP	r = 0.04	NS	r = 0.04	NS	r = –0.07	NS
Left ventricle ejection fraction						
LVEF (TTE)	**r = –0.57** **r_s = –0.47**	< 0.05	r = –0.19	NS		
LVEF (CMR)	**r = –0.36** **r_s = –0.40**	< 0.05	r = –0.13	NS		
Medicines						
ACEI	r = –0.06	NS	r = –0.21	NS	r = 0.28	NS
ARNI	r = 0.14	NS	r = 0.23	NS	**r = –0.45** **r_s = –0.45**	< 0.05
MRA	r = 0.28	NS	r = 0.23	NS	**r: –0.33** **r_s: –0.33**	< 0.05
Loop diuretics	r = 0.38	< 0.05	r = 0.09	NS	**r = –0.66** **r_s = –0.55**	< 0.05
Beta-blockers	r = 0.13	NS	r = –0.08	NS	r = –0.23	NS

ACEI — angiotensin-converting-enzyme inhibitors, ARNI — angiotensin receptor neprilysin inhibitor, CK-MB — creatine kinase-MB, CMR — cardiac magnetic resonance, CRP — C-reactive protein, LVEF — left ventricular ejection fraction, MRA — mineralocorticoid receptor antagonist, p — probability value, r — Pearson correlation coefficient, rs — Spearman's rank correlation coefficient, TnT — troponin T, TTE — transthoracic echocardiogram

CMR results and markers of myocardial damage

Relationships between the following CMR results: LVEF, ROI mass, and oedema in regards to the markers of myocardial damage and inflammation were analysed.

LVEF correlated negatively with maximal TnT levels ($r = –0.38$, $p < 0.05$). The presence of oedema correlated negatively with TnT levels ($r = –0.41$, $p < 0.05$) and positively with the number of LGE-occupied segments ($r = 0.37$, $p < 0.05$).

There was a significant correlation between LGE ROI mass and LVEF in CMR ($r = –0.36$, $p < 0.05$) as well as in TTE ($r = –0.57$, $rs = –0.47$, $p < 0.05$). The correlation between ROI mass and the number of LGE-occupied segments ($r = 0.50$, $rs = 0.45$, $p < 0.05$) was found.

No statistical significance was found concerning correlations between the CK-MB, CRP and CMR parameters (p = NS) (Table 3).

CMR results and pharmacotherapy

Relationships between the following CMR results: LVEF, ROI mass, and oedema concerning pharmacotherapy were analysed.

There were negative correlations between LVEF in CMR and administration of ARNI ($r = –0.45$, $p < 0.05$), MRA ($r = –0.33$, $p < 0.05$) and loop diuretics ($r = –0.66$, $p < 0.05$); and positive correlation between ROI MASS and loop diuretics intake ($r = = 0.38$, $p < 0.05$; but $rs = 0.22$, $p > 0.05$) (Table 3).

Discussion

Diagnosis of acute myocarditis is complex and requires different diagnostic tools. Both case history of viral infection and laboratory imaging tests are critical for the management [9–11].

In the study, the focus was on the evaluation of CMR application in the diagnosis of acute myocarditis. The present findings revealed that real-life CMR use in this setting was limited to patients with less pronounced clinical symptoms and laboratory tests. Also correlated were the CMR results with the extent of myocardial damage.

The current study compared subjects with and without CMR use. Patients' CMR(+) were characterized by less specific symptoms that ambiguously confirmed the diagnosis. Moreover, in these patients, the results of laboratory tests were less overt, and in some cases, also inconclusive — lower levels of troponin, CK-MB and CRP. It was suspected that in these patients CMR was necessary to confirm myocarditis.

The gold standard for the diagnosis of acute myocarditis is myocardial biopsy [13]. However, due to its low availability, high cost, and invasiveness, CMR is a more accessible and safer method. CMR is useful in the clinical decision-making process to take appropriate steps in stratifying patients' health risk. According to the updated LLC, CMR is the primary method for detecting signs of acute myocarditis and other markers of myocardial damage associated with myocarditis [14]. Optimal CMR imaging should include visualization of LGE, oedema and congestion.

According to available research, a lack of data on congestion constitutes a limitation of this study. Due to the marked sequence in CMR, LGE and oedema were determined in the documented laboratory. In the group of patients with CMR, 100% had LGE and 74% had oedema which allowed for diagnosis based on LLC in 29 cases (74%). In all patients a non-ischemic pattern of LGE was found.

According to the present study, the laboratory test results such as troponin, CK-MB and CRP levels did not correlate with LGE presence and the number of LV segments involved. This finding is consistent with some publications [14–16]. It should be underlined, that LGE is present both in the acute phase of myocarditis and can be a result of previous processes that may explain the current results. On the other hand, CMR was performed in patients with mildly abnormal laboratory tests — in such groups a confirmation of relationships is more difficult. In addition, the lack of a direct correlation between the presence of LGE and the level of laboratory markers of inflammation may indicate the ability of CMR to reveal features of myocardial inflammation, which, for some reason, cannot be reflected in routine blood tests. These reasons include the temporal dissociation between blood marker levels and imaging findings, the focal nature of myocardial damage that might not significantly affect systemic blood markers, and the possibility of subclinical inflammation that does not alter routine blood test results.

The data on LGE are of different clinical value. The prognostic potential of LGE in the population of patients with suspected myocarditis has already been demonstrated in many studies [17–20]. LGE is a better predictor of cardiac death and all-cause mortality compared to other functional CMR measures, including LVEF [21].

CMR volumetric parameters of the entire CMR(+) group showed a mean LVEF of 51.8%, which indicated, in some patients, a mild impairment of cardiac function. The group of patients with myocarditis was characterized by increased LV mass, estimated at a mean value of 128 g, and increased LV ESV and LV EDV which corresponds with the process of myocardial remodelling. The SV was within the normal range (range: 60–100 mL). The right ventricular ejection fraction was 57.5%. RVESV and RVEDV were also elevated, which may suggest right ventricular involvement in myocarditis.

The study also examined the relationship between CMR volumetric results and laboratory markers of inflammation and myocardial damage. LVEF showed a negative correlation with maximal levels of TnT. This points to the fact that impaired heart function was associated with higher troponin T levels, reflecting greater myocardial damage.

Oedema also correlated negatively with TnT levels which is in concordance with the fact that oedema represents ongoing myocardial damage [5]. In addition, oedema was positively correlated with the number of segments occupied by LGE. It indicates a relationship between oedema and the degree of myocardial involvement.

The timing of the laboratory tests and CMR was carefully coordinated. Blood tests, including measurements of TnT, CK-MB, and CRP levels, were conducted at the initial presentation of the patients and were repeated at 24-hour intervals for up to 72 hours to monitor changes over time. CMR was performed within 48–72 hours after the initial presentation to capture the acute phase of myocardial inflammation and damage. This approach allowed for a comprehensive assessment of the dynamic changes in both laboratory markers and imaging findings.

In the present study, the relationship between CMR results and pharmacotherapy was also determined. LVEF in CMR showed a negative correlation with the use of MRA, ARNI and loop diuretics. Interestingly, ROI mass showed a positive correlation with the intake of loop diuretics. This may suggest that patients with a more advanced inflammatory process and LV involvement require more intensified medical management. According to the literature data, CMR LVEF is a better predictor of treatment intensification than LGE [22]. Intensified therapy in the present study typically involved combination therapy, higher dosages of medications, and longer treatment periods.

Echocardiography (TTE) plays a crucial role in the initial assessment of patients with suspected myocarditis. It is often the first imaging modality used and can provide valuable information on ventricular function, wall motion abnormalities, and

the presence of pericardial effusion. TTE findings can influence the clinical decision-making process by prompting further investigation with more advanced imaging techniques like CMR.

The current study as a retrospective one-centre analysis has some limitations. There were limited number of patients analysed. However, several exclusion criteria were used and the group examined was well selected. Thanks to it the final results reflect the clinical importance of the problem of acute myocarditis and real-life low frequency of CMR use. It can be suspected that in some cases with acute heart failure, the CMR was not done because of the critical state of the patients. Due to differences in the protocols over the evaluated period, the congestion was not revealed in all exams and thus, it was not included in the analysis.

Conclusions

The present study reveals that although CMR is a valuable tool in diagnosing acute myocarditis, its use in real-world clinical practice is often limited to patients with milder symptoms. It was found that in patients with acute myocarditis, markers of myocardial damage are associated with oedema observed in CMR and CMR volumetric parameters, but they do not correlate with the presence or extent of LGE.

It should be noted, however, that this study had a relatively small number of patients, which may limit the robustness of the conclusions. Other limitations include the lack of long-term follow-up and the potential for selection bias. Further research is needed to address these limitations, validate these findings, and expand our understanding of the role of CMR in myocarditis. Larger studies with more diverse patient populations and extended follow-up periods will help improve risk assessment, guide therapeutic decisions, and ultimately enhance clinical management for patients with myocarditis.

Author contributions: Bartosz Gruchlik: analysis of CMR results, co-author of the work concept, coordination of each stage of work creation; Agnieszka Nowatorska: responsible for preparing the introduction; Sylwia Ścibisz-Brenkus: responsible for the preparation of the methodology section; Martyna Nowak: responsible for the development of the research results; Wiktor Werenkowicz: responsible for the development of the research results; Małgorzata Niemiec: responsible for developing the discussion; Andrzej Swinarew: help in analyzing the results of the study; Barbara Mika: help in analyzing the results of the study; Wojciech Wróbel: analysis of CMR results; Maciej Haberka: analysis of CMR results; Bartłomiej Stasiów: help in analyzing the results of the study; Katarzyna Mizia-Stec: author of the concept of work, coordinating each of the stages of work.

Conflict of interest: None declared.

Funding: None declared.

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