Radiology Case Reports 18 (2023) 3710–3715

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Differentiation between mild and severe myocarditis using multiparametric cardiac magnetic resonance ☆,☆☆

Hideo Arai, RT[a,*], *Masateru Kawakubo, PhD*[b], *Kenichi Sanui, RT*[a], *Hiroshi Nishimura, MD, PhD*[a], *Toshiaki Kadokami, MD, PhD*[a]

[a] *Fukuokaken Saiseikai Futsukaichi Hospital, 3-13-1 Yumachi Chikushino, Fukuoka 818-8516, Japan*
[b] *Department of Health Sciences, Faculty of Medical Sciences, Kyushu University, Japan*

ARTICLE INFO

Article history:
Received 28 March 2023
Revised 20 July 2023
Accepted 21 July 2023

Keywords:
Myocarditis
T2 mapping
Ventricular hypertrophy
Right ventricular function
Left ventricular function
Left atrial function

ABSTRACT

The pathophysiology of myocarditis is associated with mild inflammation and may progress silently, or in severe cases such as fulminant myocarditis, may lead to sudden hemodynamic compromise. An invasive myocardial biopsy is generally required for a definitive myocarditis diagnosis. Alternatively, cardiac magnetic resonance (CMR), which evaluates myocardial characteristics and cardiac function, can be used as a noninvasive tool for diagnosing myocarditis. We describe the cases of a 49-year-old woman with mild acute eosinophilic myocarditis and a 48-year-old man with severe acute lymphocytic myocarditis. CMR was performed during the acute and convalescent phases in both cases. Compared with mild myocarditis, CMR in severe myocarditis showed higher T2 values and decreased left ventricular and atrial volumes and strains; however, the right ventricular strain was preserved. Late gadolinium enhancement showed faint contrast enhancement in the whole and strong enhancement in the local myocardium. Follow-up CMR showed recovery from myocardial inflammation and cardiac function. Some late gadolinium enhancement persisted whereas acute inflammation-associated enhancement disappeared. This case report highlights the differences between the cardiac parameters of patients with mild and severe myocarditis. Severe myocardial inflammation can be caused by severe heart failure owing to the concurrent reduction of cardiac function and compliance. Additionally, preserved right ventricular strain may predict cardiac function recovery in acute myocarditis. Noninvasive and repeatable CMR provides information on myocardial characteristics, cardiac function,

☆ Acknowledgments: This work was supported by the Japan Society for the Promotion of Science [KAKENHI grant number JP20K16729]. We would like to thank Editage (www.editage.com) for English language editing.
☆☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.
* Corresponding author.
E-mail address: fukuoka.hideo@gmail.com (H. Arai).
https://doi.org/10.1016/j.radcr.2023.07.048

and hemodynamics in a single scan at that time, which is useful not only for diagnosis but also for severity assessment and patient management in acute myocarditis.

Introduction

Acute myocarditis, induced by viral infections, chemical agents, drugs, or autoimmunity, results in myocardial cell dysfunction [1]. The clinical course of myocarditis is associated with mild inflammation and may progress silently, or in severe cases such as fulminant myocarditis, may lead to sudden hemodynamic compromise [2]. Follow-up monitoring for inflammation and hemodynamics is important to patient care because myocardial inflammation may persist after patients with acute myocarditis have survived the acute phase. Generally, a definitive myocarditis diagnosis requires an invasive endocardial biopsy, which can sometimes result in complications such as cardiac tamponade [3]. Alternatively, the Lake Louise Criteria with cardiac magnetic resonance (CMR) imaging can be used for noninvasive myocarditis diagnosis [4]. Additionally, CMR can clearly show both ventricular myocardia without radiation exposure and can provide repeatable biventricular features and functional motion values in inflammatory and hemodynamic follow-ups [5,6]. Therefore, CMR can be used as a noninvasive, comprehensive diagnostic tool for cardiac diseases. However, patients with acute myocarditis are not always hemodynamically stable in the acute phase, thus CMR examination is often difficult, and there are few follow-up reports. We hypothesized that CMR-derived multiparameters could reveal differences in characteristics between mild and severe cases of patients with acute myocarditis undergoing various processes. In this report, we describe 2 cases in which CMR was useful in differentiating the severity of myocarditis and in observing the subsequent recovery process.

Case report

From December 2016 to May 2022, we enrolled 2 patients who underwent CMR twice, once in the acute phase and once in the convalescent phase, in a group of patients diagnosed with acute coronary syndrome in our hospital, without predominant stenosis from coronary angiography and with acute myocarditis from myocardial biopsy. The study was approved by the institutional review board and was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from each patient.

Cine, T2-weighted, T2 mapping, and late gadolinium enhancement (LGE) of CMR sequences were performed with electrocardiography-gating using a 3.0-T magnetic resonance imaging (MRI) system (Ingenia, Philips Healthcare, Best, The Netherlands) equipped with a 33-mT/m maximum gradient strength, 120-T/ms slew rate, and a 32-channel phased-array receiver coil. T2 value was analyzed using SYNAPSE VINCENT software (Fujifilm Co, Tokyo, Japan). Cardiac function was analyzed using Extended Workspace (Philips Healthcare, Best, The Netherlands). Strain was analyzed using an in-house-developed off-line feature-tracking application that had been validated in a previous clinical study [7]. The CMR sequence parameters were as follows:

Cine: repetition time (TR)/echo time (TE), 3.0/1.49 ms; flip angle (FA), 50°; slice thickness, 6 mm; field of view (FOV), 350 × 350 mm; acquisition matrix, 176 × 201; SENSE (sensitivity encoding) factor, 2.5; and heart phases, 20 phases/beat.

T2-weighted: TR/TE, 2 heartbeats/70 ms; FA, 90°; FOV, 350 × 350 mm; slice thickness, 10 mm; acquisition matrix, 236 × 162; SENSE factor, 2; and fat suppression, short tau inversion recovery.

T2 mapping: TR/TE, 1 heartbeat/first TE=1.5 ms, delta TE=10 ms; number of echo, 5 echoes; FA, 15°; FOV, 350 × 350 mm; slice thickness, 10 mm; acquisition matrix, 176 × 173; SENSE factor, 2.

LGE: TR/TE, 3.2 ms/1.5 ms; FA, 15°; FOV, 350 × 350 mm; slice thickness, 10 mm; acquisition matrix, 236 × 172; SENSE factor, 3.2; and images were acquired 10-15 minutes after injection of gadolinium-DO3A-butriol (Gadovist, Bayer, Germany; 0.1 mmol/kg). The inversion time was optimized to null the signal in the normal myocardium.

Case 1. Eosinophilic myocarditis with mild inflammation: A 49-year-old woman was admitted to the emergency department with throat tightness and molar and right shoulder pain, associated with ST-segment elevation on leads V2 and V3 of a 12-lead electrocardiogram (ECG) (Fig. 1). Two years prior, she had started raising guinea pigs, 6 months after which she developed symptoms of asthma and started receiving inhalation therapy. Laboratory test results showed that troponin, creatinine kinase (CK), and C-reactive protein (CRP) levels and white blood cell (WBC) and eosinophil counts were elevated (Table 1). Echocardiography revealed mildly impaired left ventricular (LV) function. Furthermore, coronary angiography (CAG) did not reveal significant coronary artery stenosis (CAS). Subsequently, a myocardial biopsy confirmed eosinophil infiltration in the edematous myocardial interstitium, and eosinophilic myocarditis was diagnosed. Treatment was initiated with 30 mg/d of prednisolone (steroid).

Case 2. Lymphocytic myocarditis with severe inflammation: A 48-year-old man was admitted to the emergency department with cough, dyspnea, and chest pain associated with poor R-wave progression on a 12-lead ECG (Fig. 1). Laboratory tests demonstrated elevated troponin, CK, and CRP levels and WBC counts (Table 1). Echocardiography revealed severe LV function impairment and diffuse myocardial hypertrophy. After ruling out significant CAS with a CAG, a myocardial biopsy revealed a prominent inflammatory cell infiltrate consisting mainly of lymphocytes, and lymphocytic myocarditis was diagnosed. Treatment was initiated with 2 mg/d of perindopril (ACE-I) and 12.5 mg/d of eplerenone (aldosterone antagonist).

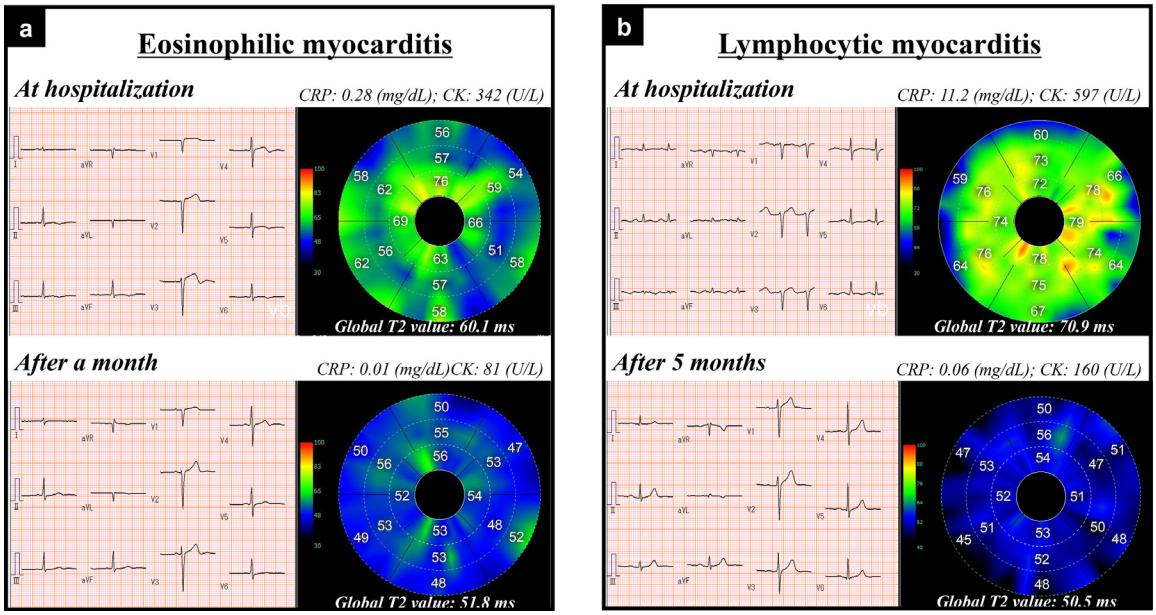

Fig. 1 – Electrocardiogram and T2 mapping in patients with myocarditis during acute and convalescent phases. The normal myocardial T2 value in our hospital is 47 ms. Eosinophilic myocarditis with mild inflammation (left side): Electrocardiogram shows ST-elevation in V2, V3, and V4 leads during the acute phase. T2 mapping shows slightly high and normal T2 values in the entire left ventricle during the acute and convalescent phases, respectively. Lymphocytic myocarditis with severe inflammation (right side): Electrocardiogram shows poor R-wave progression in the acute phase. T2 mapping shows an extremely high T2 value in the whole left ventricle during the acute phase and a normal T2 value in the convalescent phase.

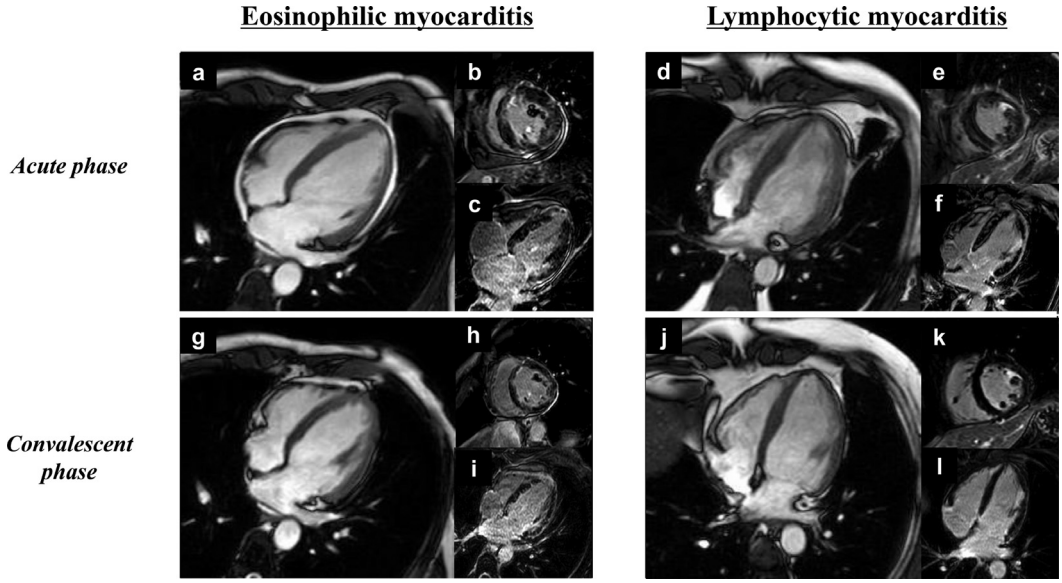

Fig. 2 – Cine cardiac magnetic resonance and late gadolinium enhancement in the acute and convalescent phases. Case 1. Eosinophilic myocarditis with mild inflammation (left side): Cine CMR shows pericardial effusion and dilated LV volume in the acute phase (a) and slight residual pericardial effusion and reduced LV volume in the convalescent phase (g). LGE shows strong CE in the anteroseptal and faint non-ischemic CE in the circumferential myocardium in the acute (b and c) and convalescent phases (h and i). Case 2. Lymphocytic myocarditis with severe inflammation: Cine CMR shows reduced LV volume due to centric LV hypertrophy in the acute phase (d) and normal myocardial thickness and volume in the convalescent phase (j). LGE shows faint nonischemic CE in the circumferential myocardium (e and f) and strong CE in the anterolateral myocardium (e) in the acute phase, with loss of the faint CE (k and l) and persistent strong CE (k) in the convalescent phase. CMR, cardiac magnetic resonance; LV, left ventricular; LGE, late gadolinium enhancement; CE, contrast enhancement.

Table 1 – Patient characteristics, laboratory tests results, and cardiac function by cardiac magnetic resonance during hospitalization and follow-up.

	Eosinophilic myocarditis			Lymphocytic myocarditis		
	Baseline	Follow-up (a month)	Normal range	Baseline	Follow-up (5 months)	Normal range
Characteristics						
Age	49			48		
Sex	Female			Male		
Height (cm)	159	159		169	169	
Weight (kg)	51	48		57	57	
BMI (kg/m^3)	20.2	19.0		20.0	20.0	
Laboratory data						
Troponin (ng/L)	1.061		0-0.1	≧0.1		0-0.1
CK (U/L)	342	81	51-209	597	160	51-209
CRP (mg/dL)	0.28	0.01	0-0.14	11.15	0.06 (After 15 days)	0-0.14
WBC (10^3/μL)	10.4	7.9	3.3-8.6	12.9	5.5	3.3-8.6
NT pro BNP (pg/mL)	3640	222	125≦	14,259	19	125≦
Cardiac MRI						
Heart rate (bpm)	69	50		86	67	
LV analysis			Mean (SD)			Mean (SD)
EDVI (mL/m^2)	92.9	87.8	73 (12)	72.8	83.3	79 (15)
ESVI (mL/m^2)	53.8	43.9	25 (7)	48.5	42.9	29 (9)
SVI (mL/m^2)	39.1	43.8	49 (8)	24.2	40.4	52 (10)
EF (%)	42.1	49.9	66 (7)	33.3	48.5	64 (8)
CO (L/min)	4.3	2.9	4.5 (0.9)	3.4	4.5	5.6 (1.1)
CI (L/min/m^2)	2.8	2.0	2.9 (0.5)	2.1	2.7	3.0 (0.6)
MI (g/m^2)	74.0	65.0	49 (10)	75.0	43.6	62 (11)
LVCS (%)	-9.2	-16.3	-23.0 (0.7)	-10.5	-16.3	-23.0 (0.7)
LVLS (%)	-8.2	-11.9	-20.1 (0.4)	-4.4	-10.4	-20.1 (0.4)
LA analysis						
Volume (mL)	50.4	39.4	64 (18)	32.1	39.5	72 (20)
VI (mL/m^2)	33.4	26.8	39 (11)	19.5	23.9	38 (11)
LA Strain (%)	17.9	19.0	40.2 (8.5)	9.8	18.1	37.6 (10.2)
RV analysis						
EDVI (mL/m^2)	72.9	63.3	74 (12)	63.5	75.4	83 (13)
ESVI (mL/m^2)	30.8	23.6	26 (8)	37.2	39.5	29 (9)
SVI (mL/m^2)	42.1	39.7	48 (7)	26.2	35.9	54 (8)
EF (%)	57.8	62.7	66 (7)	41.4	47.7	66 (7)
CO (l/min)	4.4	2.9	4.4 (1.0)	3.7	4.0	5.6 (1.4)
CI (L/min/m^2)	2.9	2.0	2.8 (0.6)	2.2	2.4	3.0 (0.7)
FAC (%)	56.5	62.3	>35	59.9	50.1	35>
RV strain (%)	-35.1	-47.4	-21.8 (0.8)	-38.4	-40.5	-21.8 (0.8)

BMI, body mass index; CI, cardiac index; CO, cardiac output; EDVI, end-diastolic volume index; EF, ejection fraction; ESVI, end-systolic volume index; FAC, fractional area change; LA strain, left atrium strain; LVCS, left ventricular circumferential strain; LVLS, left ventricular longitudinal strain; MRI, magnetic resonance imaging; MI, mass index; RV strain: right ventricular strain; SVI, stroke volume index; VI, volume index.

CMR imaging findings: Acute-phase CMR imaging was performed on days 2 and 5 for cases 1 and 2, respectively, after achieving hemodynamic stability. Table 1 shows the CMR parameters. Figure 2 shows the cine CMR and LGE of both cases in the acute (Fig. 2a–f) and convalescent phases (Fig. 2g–l). Figure 3 shows the cine CMR and T2-weighted imaging of both cases in the acute (Fig. 3a–f) and convalescent phases (Fig. 3g–l). The normal ranges for CMR parameters were obtained from previous studies [8–10]. The normal myocardial T2 value at our hospital is 47 ms. Acute global T2 values for cases 1 and 2 were 60.1 and 70.9 ms, respectively, showing inflammation throughout the left ventricle and a high value for case 2 especially (Fig. 1 upper sides of a and b). Both cine CMR images in the acute phase showed severely impaired LV ejection fraction (Case 1: 42.1% vs Case 2: 33.3%) and LV strain (Case 1: LV circumferential strain (LVCS), -9.2%; LV longitudinal strain (LVLS), -8.2% vs Case 2: LVCS, -10.5%; LVLS, -4.4%); however, the right ventricular wall motion was normal (Case 1: right ventricular fractional area change (RVFAC), 56.5% vs Case 2: RVFAC, 59.9%) (Table 1). Case 2, with severe inflammation, showed bi-ventricular hypertrophy and reduced left atrial (LA) strain of 9.8% much lower than the 17.9% in Case 1. LGE showed faint nonischemic contrast enhancement (CE) in the intimal, middle, and adventitial layers of the circumferential myocardium and strong local CE in both cases (Fig. 2b, c, e, and f). Convalescent CMR for cases 1 and 2 was performed after 1 and 5 months, respectively, and the laboratory test results were within the normal range (Table 1). Subsequent T2 mapping revealed no signs of myocardial inflammation (Case 1: 51.8 ms; Case 2: 50.5 ms; Fig. 1 lower side of a and b), while cine CMR imaging showed myocardial weight reduction and cardiac function recovery. Some LGE remained (Fig. 2h, i, k, and l); however, LGE due to acute inflammation disappeared in Case 2 (Fig. 2k and l).

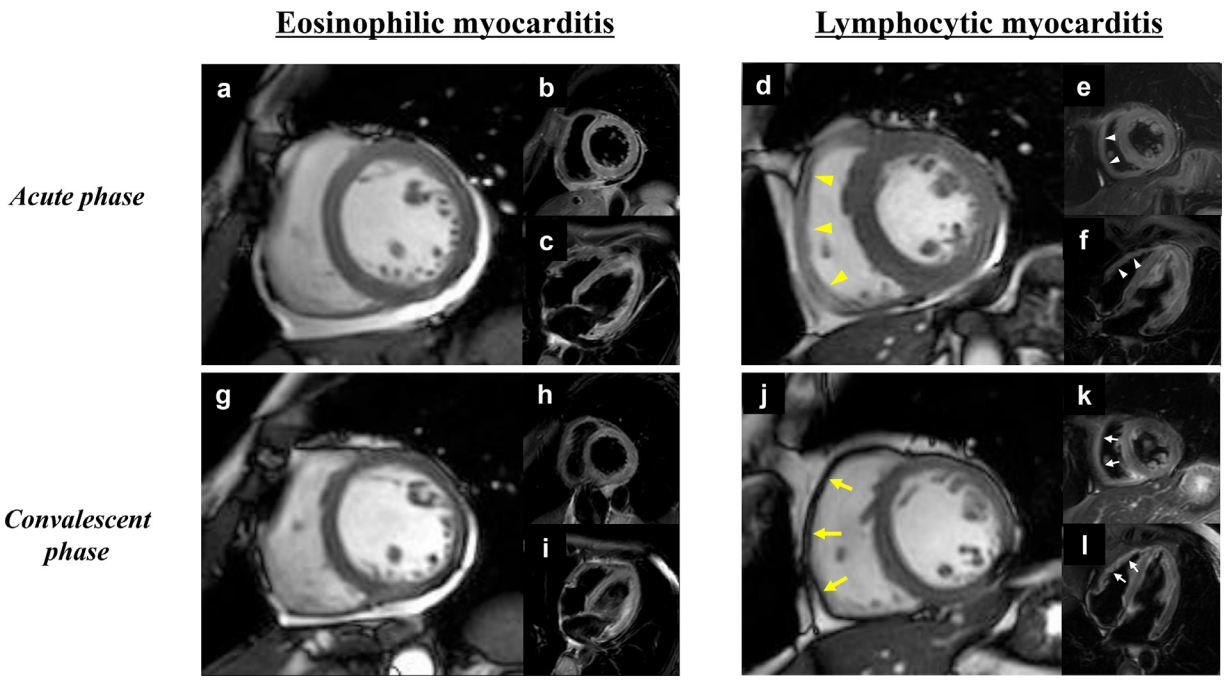

Fig. 3 – Right ventricular myocarditis indicated by cine cardiac magnetic resonance. Case 1. Eosinophilic myocarditis with mild inflammation (left side): No findings of RV myocarditis are observed in cine CMR (a and g) and T2-weighted images (b, c, h, and i) in the acute and convalescent phases. Case 2. Lymphocytic myocarditis with severe inflammation: RV myocarditis can only be observed in the hypertrophied RV myocardium in cine CMR during the acute phase (d; yellow arrowhead). No RV myocarditis is observed in the T2-weighted image in the acute phase (e and f; white arrowhead), cine CMR (j; yellow arrow), and T2W (k and l; white arrow) in the convalescent phase. RV, right ventricular; CMR; cardiac magnetic resonance.

Discussion

An invasive myocardial biopsy is required for a definitive diagnosis of acute myocarditis, and follow-up is necessary because of the varied course of this disease [1,2]. CMR can noninvasively assess myocardial characteristics and function at that time [11]. Moreover, CMR can be repeated since it is noninvasive, making it useful for follow-up. In this case report, we describe the usefulness of CMR parameters for the diagnosis and management of mild and severe myocarditis from the acute to the convalescent phase.

T2-based imaging and T2 mapping revealed inflammation throughout the LV myocardium during the acute phases in both cases. Compared with T2-weighted imaging, T2 mapping is an advanced imaging technique that can quantify edema and, in this case, it facilitated the diagnosis of severe edema and patient follow-up (Figs. 1 and 3) [12]. As in previous reports, elevated T2 values were associated with the severity of left ventricular dysfunction [13]. In addition, T2 mapping is superior to clinical biomarkers as it can additionally indicate the location of myocardial inflammation. Furthermore, T1-based imaging LGE showed nonischemic CE. These T1- and T2-based findings are indicative of myocarditis in the Lake Louise Criteria [4].

Case 1 (with mild inflammation) had a compensatory increase in heart rate and chamber enlargement to maintain the cardiac output (CO). In contrast, these changes were not observed in case 2 (with severe inflammation). This may be a result of progressive heart failure; severe myocardial edema causes excessive cardiac hypertrophy due to tissue expansion and subsequently leads to restricted LV enlargement due to reduced myocardial compliance. The acute phase-stroke volume is severely decreased due to the reduced LV volume and wall motion and is reportedly an indicator of poor prognosis [14].

The Forrester heart failure severity classes recorded for cases 1 and 2 were I and IV, respectively. In addition, the severely decreased LA volume of 32.1 mL and LA strain of 9.8% during the acute phase in case 2 may be explained by a rapid increase in LV pressure due to decreased LV compliance and contractility or by LA inflammation alone. It has been reported that LA strain and LV end-diastolic pressure show a good correlation and that a decreased LA strain may indicate increased preload due to severe myocarditis [15]. During the convalescent phase, the LA strain improved to the normal range and the heart failure severity to Forrester class I in case 2. In addition, during the acute phase in case 2, right ventricular (RV) wall thickening was observed on cine CMR imaging (Fig. 3d), which may be the result of RV myocarditis. The prevalence of RV myocarditis in patients with clinically suspected myocarditis has been reported to be 17.8% [16]. RV myocarditis is scarcely reported due to the RV imaging restriction when using echocardiography as the standard diagnostic modality. The CMR showed that preserved RV strain parameters were observed in both severe and mild cases. Despite the presence of severe heart failure caused by extremely high inflammation in case 2, cardiac function recovered after 5 months. Recently,

RV strain has been reported to be an independent prognostic factor for acute myocarditis [17]. In the present case, preserved RV strain in the acute phase may have contributed to the recovery of cardiac function in a patient with myocarditis.

In patients with suspected acute coronary syndromes, when coronary artery stenosis is ruled out, the diagnosis is often difficult to make. However, the ability to capture myocardial characteristics and function with CMR, as in previous reports, is of great help in diagnosis and treatment [18]. In addition, CMR has a wide field of view, making it possible to analyze both ventricles and atria. Furthermore, because there is no radiation exposure, scans can be repeated, which is useful in the management of acute myocarditis requiring follow-up. The limitations of this study are the small number of patients and the lack of long-term follow-up. Further study is required to examine our findings with more patients and long-term follow-ups.

Acute myocarditis with varied courses is a difficult disease to diagnose and manage. Noninvasive and repeatable CMR provides information on myocardial inflammation, fibrosis, cardiac function, and hemodynamics in a single scan at that time, which is useful not only for diagnosis but also for severity assessment and patient management with acute myocarditis.

Patient consent

The authors confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with the COPE guidance.

REFERENCES

[1] Tschöpe C, Cooper LT, Torre-Amione G, Van Linthout S. Management of myocarditis-related cardiomyopathy in adults. Circ Res 2019;124:1568–83. doi:10.1161/CIRCRESAHA.118.313578.

[2] Law YM, Lal AK, Chen S, Čiháková D, Cooper LT Jr, Deshpande S, et al. Diagnosis and management of myocarditis in children: a scientific statement from the American Heart Association. Circulation 2021;144:e123–35. doi:10.1161/CIR.0000000000001001.

[3] Halvorsen S, Mehilli J, Cassese S, Hall TS, Abdelhamid M, Barbato E, et al. 2022 ESC Guidelines on cardiovascular assessment and management of patients undergoing noncardiac surgery. Eur Heart J 2022;43:3826–924. doi:10.1093/eurheartj/ehac270.

[4] Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U, et al. Cardiovascular magnetic resonance in nonischemic myocardial inflammation: expert recommendations. J Am Coll Cardiol 2018;72:3158–76. doi:10.1016/j.jacc.2018.09.072.

[5] Cochet H, Iriart X, Allain-Nicolaï A, Camaioni C, Sridi S, Nivet H, et al. Focal scar and diffuse myocardial fibrosis are independent imaging markers in repaired tetralogy of Fallot. Eur Heart J Cardiovasc Imaging 2019;20:990–1003. doi:10.1093/ehjci/jez068.

[6] Yang L, Zhang L, Cao S, Gao C, Xu H, Song T, et al. Advanced myocardial characterization in hypertrophic cardiomyopathy: feasibility of CMR-based feature tracking strain analysis in a case-control study. Eur Radiol 2020;30:6118–28. doi:10.1007/s00330-020-06922-6.

[7] Kawakubo M, Nagao M, Ishizaki U, Shiina Y, Inai K, Yamasaki Y, et al. Feature-tracking MRI fractal analysis of right ventricular remodeling in adults with congenitally corrected transposition of the great arteries. Radiol Cardiothorac Imaging 2019;1:e190026. doi:10.1148/ryct.2019190026.

[8] Kawel-Boehm N, Hetzel SJ, Ambale-Venkatesh B, Captur G, Francois CJ, Jerosch-Herold M, et al. Reference ranges ("normal values") for cardiovascular magnetic resonance (CMR) in adults and children: 2020 update. J Cardiovasc Magn Reson 2020;22:87. doi:10.1186/s12968-021-00815-3.

[9] Vo HQ, Marwick TH, Negishi K. MRI-derived myocardial strain measures in normal subjects. JACC Cardiovasc Imaging 2018;11:196–205. doi:10.1016/j.jcmg.2016.12.025.

[10] Truong VT, Palmer C, Wolking S, Sheets B, Young M, Ngo TNM, et al. Normal left atrial strain and strain rate using cardiac magnetic resonance feature tracking in healthy volunteers. Eur Heart J Cardiovasc Imaging 2020;21:446–53. doi:10.1093/ehjci/jez157.

[11] Zhang L, Awadalla M, Mahmood SS, Nohria A, Hassan MZO, Thuny F, et al. Cardiovascular magnetic resonance in immune checkpoint inhibitor-associated myocarditis. Eur Heart J 2020;41:1733–43. doi:10.1093/eurheartj/ehaa051.

[12] Sprinkart AM, Luetkens JA, Träber F, Doerner J, Gieseke J, Schnackenburg B, et al. Gradient Spin Echo (GraSE) imaging for fast myocardial T2 mapping. J Cardiovasc Magn Reson 2015;17:12. doi:10.1186/s12968-015-0127-z.

[13] Spieker M, Haberkorn S, Gastl M, Behm P, Katsianos S, Horn P, et al. Abnormal T2 mapping cardiovascular magnetic resonance correlates with adverse clinical outcome in patients with suspected acute myocarditis. J Cardiovasc Magn Reson 2017;19:38. doi:10.1186/s12968-017-0350-x.

[14] Hiramitsu S, Morimoto SI, Kato S, Uemura A, Ohtsuki M, Kato Y, et al. Significance of transient left ventricular wall thickening in acute lymphocytic myocarditis. Heart Vessels 2007;22:25–9. doi:10.1007/s00380-006-0933-1.

[15] Wakami K, Ohte N, Asada K, Fukuta H, Goto T, Mukai S, et al. Correlation between left ventricular end-diastolic pressure and peak left atrial wall strain during left ventricular systole. J Am Soc Echocardiogr 2009;22:847–51. doi:10.1016/j.echo.2009.04.026.

[16] Aquaro GD, Negri F, De Luca A, Todiere G, Bianco F, Barison A, et al. Role of right ventricular involvement in acute myocarditis, assessed by cardiac magnetic resonance. Int J Cardiol 2018;271:359–65. doi:10.1016/j.ijcard.2018.04.087.

[17] Bernhard B, Schnyder A, Garachemani D, Fischer K, Tanner G, Safarkhanlo Y, et al. Prognostic value of right ventricular function in patients with suspected myocarditis undergoing cardiac magnetic resonance. JACC Cardiovasc Imaging 2023;16:28–41. doi:10.1016/j.jcmg.2022.08.011.

[18] Cau R, Bassareo P, Deidda M, Caredda G, Suri JS, Pontone G, et al. Could CMR tissue-tracking and parametric mapping distinguish between Takotsubo syndrome and acute myocarditis? A pilot study. Acad Radiol 2022;29(Suppl 4):S33–9. doi:10.1016/j.acra.2021.01.009.