International Journal of Cardiology 354 (2022) 63–67

Contents lists available at ScienceDirect

International Journal of Cardiology

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

Cardiac MRI after first episode of acute pericarditis: A pilot study for better identification of high risk patients

Edoardo Conte [a,b,*], Cecilia Agalbato [a], Gianfranco Lauri [a], Saima Mushtaq [a], Alessia Dalla Cia [a], Alice Bonomi [a], Marco Guglielmo [a], Andrea Baggiano [a,c], Margherita Gaudenzi-Asinelli [a], Gualtiero Colombo [a], Chiarella Sforza [b], Piergiuseppe Agostoni [a], Gloria Tamborini [a], Emilio Assanelli [a], Gianluca Pontone [a], Mauro Pepi [a], Antonio Brucato [d], Daniele Andreini [a,d]

[a] Centro Cardiologico Monzino, IRCCS, Milan, Italy
[b] Università di Milano, Deptartment of Biomedical Sciences for Health, University of Milan, Milan, Italy
[c] Department of Clinical Sciences and Community Health - Cardiovascular Section, University of Milan, Milan, Italy'
[d] Università di Milano, Department of Biomedical and Clinical Sciences "Luigi Sacco", Fatebefratelli Hospital, Milan, Italy

ARTICLE INFO

Keywords:
Acute pericarditis
Cardiac MRI
Prognosis

ABSTRACT

Background: Cardiac magnetic resonance (CMR) was proposed as an accurate non-invasive tool to evaluate pericardial inflammation. Aim of the present study was to evaluate the role of CMR early in the course of the first episode of acute pericarditis.

Material and methods: A clinical registry of consecutive patients who underwent clinical indicated CMR due to pericardial disease from January 2014 to January 2020 was screened. We analyzed patients with the clinical diagnosis of first episode of acute pericarditis needing hospitalization less than 7 days before CMR. Outcome measures were obtained using a single combined end-point, defined as pericardial event, including all the following: recurrent pericarditis, chronic constrictive pericarditis, surgery for pericardial disease.

Results: Twenty-six patients meet the study criteria and were enrolled. A mean follow-up of 34 ± 7 months was obtained and a second episode of pericardial event were recorded in 9 patients. At multivariate analysis adjusted for propensity score, based on clinical significative variable (younger age and higher CRP) the association between pericardial inflammation identified by CMR (positive late gadolinium enhancement on pericardium) and recurrence of pericardial events was confirmed [OR (95%CI) 8.94 (1.74–45.80), $p = 0.008$].

Conclusion: Pericardial inflammation identified by CMR, with LGE images, has a prognostic value independently from clinical and bio-humoral variables.

1. Introduction

Acute pericarditis is an inflammatory pericardial syndrome that could be associated with pericardial effusion [1]. Most patients with acute pericarditis have good prognosis and, if appropriate therapy is introduced, they could be managed in an outpatient setting. However, when high-risk factors are present the hospitalization is suggested as this subgroup of patients may have higher risk of acute and future complications as reported in a landmark paper published in 2007 [2]. More specifically, 35–40% of patients hospitalized for acute pericarditis have recurrent episodes of symptomatic pericardial inflammation. Traditionally, after the first episode, irrespective to disease severity, no specific therapeutic approach is suggested but second- or third-line therapies are reserved to patients with incessant or recurrent pericarditis.

Recently, cardiac magnetic resonance (CMR) has been suggested as an accurate non-invasive tool for anatomical and functional assessment of pericardium [3,4] as it may provide important insights on inflammatory activity affecting the pericardial sheets even in the absence of pericardial effusion [3]. More specifically, T2-weighted images and post-contrast sequences (late-gadolinium enhancement, LGE) positive for signal-hyperintensity on the pericardial leaflets are suggestive for

Abbreviations: CMR, cardiac magnetic resonance; HR, hazard ratio; LGE, late gadolinium enhancement; CRP, C-reactive Protein.

* Corresponding author at: Centro Cardiologico Monzino, IRCCS, Milan, Italy.
E-mail address: edoardo.conte@ccfm.it (E. Conte).

https://doi.org/10.1016/j.ijcard.2022.03.007
Received 30 November 2021; Received in revised form 17 February 2022; Accepted 7 March 2022
Available online 12 March 2022

residual pericardial inflammatory activity.

At present no data exist as regards the prognostic role of CMR in patients hospitalized for the first episode of acute pericarditis. Indeed, previous studies [5–8] were focused on patients with recurrent or incessant pericarditis. Of note, in these studies the time from acute onset of pericardial disease and CMR was often unspecified, introducing potential bias related to a less effective assessment of pathological findings at CMR in case of longer delay between symptoms onset and imaging evaluation.

Aim of the present pilot study was to evaluate the role of CMR early in the course of the first episode of acute pericarditis presenting with high risk factors and in which hospitalization was clinically indicated in order to better identify patients who may merit stricter follow-up and more aggressive therapy to reduce risk of future pericardial complications.

2. Material and methods

2.1. Study population

A clinical registry of consecutive patients who underwent clinical indicated CMR due to pericardial disease from January 2014 to January 2020 at Centro Cardiologico Monzino, IRCCS, Milan Italy was screened. From this registry including 152 patients, those who reached all the following inclusion criteria were enrolled in the present retrospective study: 1) clinical diagnosis of first episode of acute pericarditis according to ESC guidelines [1] needing hospitalization less than 7 days before CMR due to the presence of at least one of major high risk criteria according to ESC guidelines: Fever>38 °C, subacute onset, large pericardial effusion, cardiac tamponade, lack of response to aspirin or NSAIDs after at least 1 week of therapy, myopericarditis, immunosuppression, trauma, oral anticoagulation therapy; 2) available ECG at the time of diagnosis less than 7 days before CMR; 3) available bio-humoral data (at least white blood cell, hemoglobin, platelets count, Tn-I and CRP) obtained less than 7 days before CMR; 4) available transthoracic echocardiography performed less than 7 days before CMR. Medical therapy and bio-humoral data were retrieved from hospital records during index hospitalization. Electrocardiograms were reviewed and the following categorial findings were recorded: PR segment depression, ST-depression, ST-elevation, negative T waves. The presence of at least 1 of these ECG alterations was used to consider the patient status as positive for ECG changes.

2.2. Imaging modalities

Transthoracic echocardiography was performed in all patients according to clinical routine and were re-evaluated blinded to clinical and CMR data. More precisely, a complete standard 2DTTE was performed according to clinical laboratory practice and international recommendations [9]. The presence and entity of pericardial effusion was recorded and measured in millimeters together with sing of constriction defined as any of the following: e' med/e'lat > 1, paradoxical septal movement during forced inspiration, E/A typical pattern during respiratory phases. Large pericardial effusion was defined as >20 mm. Transthoracic echocardiography was considered to be positive for pericardial disease when pericardial effusion and/or any sign of constriction were identified.

All patients underwent clinically indicated CMRs that were carried out using dedicated cardiac software, phased-array surface receiver coils, and ECG triggering. A stack of short axis bSSFP images encompassing both ventricles from base to apex was used for biventricular volumes and mass and systolic function assessments together with identification and quantification of pericardial effusion. Pre-contrast T1-weighted images for identification and quantification of pericardial thickening and STIR T2-weighted for identification of myocardial and/or pericardial inflammation/edema were performed. LGE images were obtained 8–12 min after intravenous injection of gadolinium contrast agent (0.1 to 0.2 mmol/kg body weight). Moreover, real-time cine images during forced inspiration were obtained to identify the presence of biventricular interdependence, suggestive for constrictive physiology. As previously suggested, CMRs were re-evaluated blinded to clinical and echocardiographic data for the following findings [10–14]: pericardial effusion (large pericardial effusion>20 mm), pericardial hyperintensity signal detected on T2-w, pericardial hyperintensity signal detected on LGE images. Quantitative evaluation of pericardial LGE was performed using dedicated post-processing tools, as previously described [7].

2.3. Follow-up

Follow-up information were obtained by clinical visits in all patients and hospital records were screened for clinical events as well. All records were analyzed by medical staff blinded to previous clinical, bio-humoral and imaging data. The occurrence of recurrent pericarditis, chronic constrictive pericarditis, surgery for pericardial disease defined according to ESC guidelines [1] were recorded. Outcome measures were obtained using a single combined endpoint, defined as pericardial event, including any of the following: recurrent pericarditis, chronic constrictive pericarditis, surgery for pericardial disease.

2.4. Statistical analysis

Continuous variables were presented as mean ± SD and categorical variables as absolute numbers and percentages. When not normally distributed, continuous variables were expressed as median (interquartile range). Cox regression analysis was used in order to evaluate the relationship between, bio-humoral, clinical and imaging variables and outcomes at univariate analysis. Significative variables were evaluated at multivariate analysis. More precisely the relationship between significative 'imaging' variables at univariate analysis and the event was evaluated with multivariate Cox models, adjusted for propensity score, based on the significative clinical variables at univariate analysis. Finally, events-free survival rates as a function over time were obtained by the Kaplan-Meier method and compared using the log-rank test.

The total population of 26 patients included in the study, with a mean events rate for the entire population of 0.35, enabled to identify two different populations with an event rate delta of 0.5, with a type 1 error of 5% and a type II error of 20%. Statistical significance was defined as a $p < 0.05$.

3. Results

From a prospective clinical registry of consecutive patients underwent CMR due to pericardial disease ($n = 152$), 49 patients had CMR for non-inflammatory pericardial disease, 40 subjects had CMR more than 7 days after diagnosis and 37 subjects had CMR examination for recurrent pericarditis. Accordingly, these patients were excluded as pre-specified inclusion criteria. Thus, a total of 26 patients with first episode of acute pericarditis needing hospitalization were finally enrolled (Supplementary Fig. 1). Among patients enrolled 22 had idiopathic pericarditis, 2 had previous radiotherapy, 1 had rheumatoid arthritis and 1 post-myocardial infarction pericarditis. They all fulfilled the high-risk criteria for which hospitalization is recommended [1,2]. Mean follow-up was 34 ± 7 months during which 9 pericardial events (8 recurrent pericarditis and 1 chronic constrictive pericarditis) were recorded in 9 different patients.

The mean population age was 58.3 ± 15.5 years (male/female: 16/10). Patients with pericardial events at follow-up resulted to be younger (50.3 ± 15.7 vs. 62.6 ± 14.7, $p = 0.032$) without any other differences between the two groups for other clinical variables at baseline (Supplementary Table 1). More precisely, all subjects included in the study had elevated CRP (177.1 ± 104.7 vs. 118.6 ± 87.2 mg/dL, for patients with vs without pericardial events respectively, $p = 0.156$) and the

majority of patients had idiopathic pericarditis (88.8% vs. 82.2%, $p = 0.667$), while one third of subjects included in the study had positive troponin-I (33.3% vs. 35.3%, $p = 0.921$) (Supplementary Table 1). Almost all patients were treated with non-steroidal anti-inflammatory therapy (100% vs. 88.2% for patients with vs without pericardial events respectively, $p = 0.294$) and more than half of subjects enrolled were treated with dual association therapy (66.6% vs. 64.7%, $p = 0.912$); on the contrary, as expected, only a minority of patients received a triple association therapy (11.1% vs. 5.9%, $p = 0.604$) (Supplementary Table 1). All patients correctly completed therapy as prescribed in accordance with ESC guidelines on pericardial disease [1].

Among imaging characteristics, identification of pericardial effusion both at transthoracic echocardiography (88.8% vs. 55.5% with vs. without pericardial events respectively, $p = 0.123$) and at CMR (66.6% vs. 47%, $p = 0.349$) did not result to be predictive of future pericardial events. On the contrary, transthoracic echocardiography positive for pericardial disease (100% vs. 55.5% for patients with vs without pericardial events respectively, $p = 0.027$) and positive LGE on pericardium at CMR (66.7% vs. 11.7%, $p = 0.005$) were more common among subjects with pericardial events at follow-up. On the contrary, positive LGE on myocardium was not more common among patients with pericardial events at follow-up (Supplementary Table 2).

Univariate analysis confirmed a significative protective association between older age [OR 0.93 (95%CI 0.87–0.98), $p = 0.021$] and pericardial events, while elevated CRP [OR 1.01 (95%CI 1.00–1.01), $p = 0.025$] resulted to be associated with higher risk of pericardial events (Table 1). On the contrary, neither pericarditis etiology [OR 0.91 (95% CI 0.11–7.62), $p = 0.914$], ECG modifications [OR 0.46 (95%CI 0.12–1.77), $p = 0.269$] or myocarditis involvement [OR 1.29 (95%CI 0.31–5.44), $p = 0.726$] resulted to be associated with pericardial events at follow-up. AS regards pericardial effusion identified both at echocardiography [OR 2.34 (95%CI 0.28–19.23), $p = 0.427$] and CMR [OR 1.82 (95%CI 0.43–7.71), $p = 0.413$] it was not associated with higher risk of pericardial events. On the contrary both T2-weighed [OR 31.82 (95%CI 3.34–302), $p = 0.003$] and LGE images [OR 7.38 (95%CI 1.76–30.88), $p = 0.006$] positive for pericardial inflammation were predictive of pericardial events (pericarditis recurrences and chronic constrictive pericarditis diagnosis) (Table 1). At multivariate analysis only positive LGE at CMR was confirmed to be associated with pericardial events at follow-up [ORE (95%CI) 8.94 (1.74–45.80), $p = 0.008$), even when adjusted for propensity score based on clinical significative variable (both younger age and elevated CRP) (Table 2). Of interest, Kaplan-Meyer curve well outlined the higher incidence of pericardial events at follow-up of patients with pericardial LGE and positive T2-weighted images on pericardium (Fig. 1A and B respectively). More precisely, patients with positive LGE or T2 weighted images on pericardium had a significative (log-rank $p < 0.001$) lower probability of pericardial events-free survival at follow-up (19% and 32%, respectively) when compared with patients free from pericardial inflammation at CMR (89% and 82%, respectively).

Table 1
Clinical and imaging characteristics associated to pericardial events at univariate analysis.

	Univariate analysis OR (CI 95%)	p
Clinical Characteristics		
Age (y)	0.93 (0.87–0.98)	0.021
Sex (M)	1.10 (0.27–4.46)	0.889
Chest pain	1.16 (0.13–9.73)	0.887
Friction rub	-*	-*
Dyspnea	1.42 (0.28–7.08)	0.667
Fever >38 °C	2.5 (0.65–9.61)	0.178
Subacute onset, n (%)	2.68 (0.61–10.97)	0.181
Lack of response to NSAIDs, n (%)	1.27 (0.33–4.85)	0.723
Immunosuppression, n (%)	1.82 (0.21–15.27)	0.605
Idiopathic pericarditis	0.91 (0.11–7.62)	0.914
Myopericarditis	1.29 (0.31–5.44)	0.726
ECG changes	0.46 (0.12–1.77)	0.269
Bio-humoral data		
CRP peak	1.01 (1.00–1.01)	0.025
WBC	1.00 (0.99–1.00)	0.253
Hb	0.78 (0.51–1.17)	0.233
Abnormal Tn-I	0.52 (0.12–2.21)	0.378
Echocardiographic data		
Pericardial effusion presence	2.34 (0.28–19.23)	0.427
Large pericardial effusion	0.36 (0.04–3.09)	0.358
Pericardial thickening	2.26 (0.26–18.91)	0.452
Sign of constriction	3.19 (0.58–17.49)	0.179
Echocardiography positive for pericardial disease	3.55 (0.43–28.96)	0.236
Cardiac MRI data		
Pericardial effusion presence	1.82 (0.43–7.71)	0.413
Large pericardial effusion	0.46 (0.05–4.01)	0.487
Positive myocardium T2w images	0.95 (0.11–7.91)	0.967
Positive pericardium T2w images	31.82 (3.34–302)	0.003
Positive myocardium LGE	1.89 (0.45–7.92)	0.382
Positive pericardium LGE	7.38 (1.76–30.88)	0.006
LGE myocardium mass	0.98 (0.87–1.01)	0.752
LGE pericardium volume	1.04 (1.00–1.09)	0.034

* The only patient with friction rub was free from event at follow-up.

4. Discussion

We evaluated the prognostic role of pericardial inflammation at CMR in a very selected population of patients hospitalized for a first episode of severe acute pericarditis who underwent CMR no more than 7 days after hospitalization. The main finding of our pilot study was that pericardial inflammation identified by CMR, with positive pericardial LGE, had a prognostic value independently from clinical and bio-humoral variables. More specifically, positive pericardial LGE at CMR enabled to identify a subgroup of patients with first episode of pericarditis, but with very high incidence of pericarditis recurrences at follow-up (81% of patients with positive pericardial LGE had pericardial event at follow-up). Cremer et al. [15] suggested that pericardial LGE is caused by neovascularization of pericardial layers during pericardial inflammation. Consequently, a higher degree of neovascularization, highlighted by positive pericardial LGE, may have a role in enhancing the auto-inflammatory response that may sustain pericarditis recurrences [16–17–18] (Fig. 2). More specifically, neovascularization, has been previously describe at hystopatological studies as indicative of an ongoing, dynamic active inflammatory reaction [16] On the contrary, the absence of significative pericardial neovascularization may be protective, reducing auto-inflammatory response, despite similar CRP elevation in the acute phase. Of interest, T2-weighted images positive on pericardium, suggestive for pericardial edema, were not associated to pericardial events at follow-up when corrected for CRP at multivariate analysis; this finding may support the hypothesis that positive LGE is associated to pericardial neovascularization and not only to pericardial inflammation. Of interest, among clinical variable, younger age was associated with future pericardial events at univariate analysis, supporting the role of immune response in promoting recurrencies. It should

Table 2
Clinical and Imaging characteristics associated to pericardial event at multivariate analysis.

	Multivariate analysis OR (CI 95%)	p
Corrected for age*		
Positive T2 on pericardium	15.93 (1.53–165.76)	0.020
Positive LGE on pericardium	8.58 (1.67–43.93)	0.009
Corrected for CRP*		
Positive T2 on pericardium	26.85 (1.64–439.24)	0.021
Positive LGE on pericardium	5.27 (1.10–25.07)	0.036
Corrected for both age and CRP*		
Positive T2 on pericardium	11.51 (0.45–295.37)	0.140
Positive LGE on pericardium	8.94 (1.74–45.80)	0.008

* Propensity score adjusted.

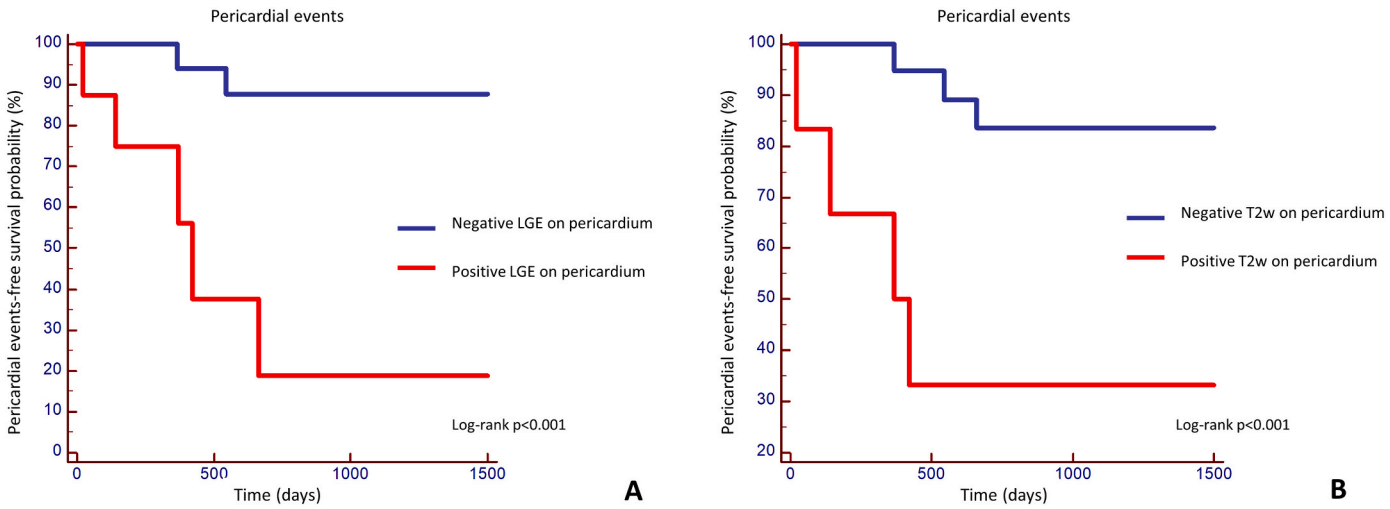

Fig. 1. Kaplan-Meyer curves analysis.
Subjects enrolled were stratified according to the presence of pericardial LGE (Fig. 1A) and positive T2 weighted images (Fig. 1B). Both T2 weighted images and pericardial LGE supporting presence of pericardial inflammation is significatively associated with higher rate of pericardial events at follow-up.

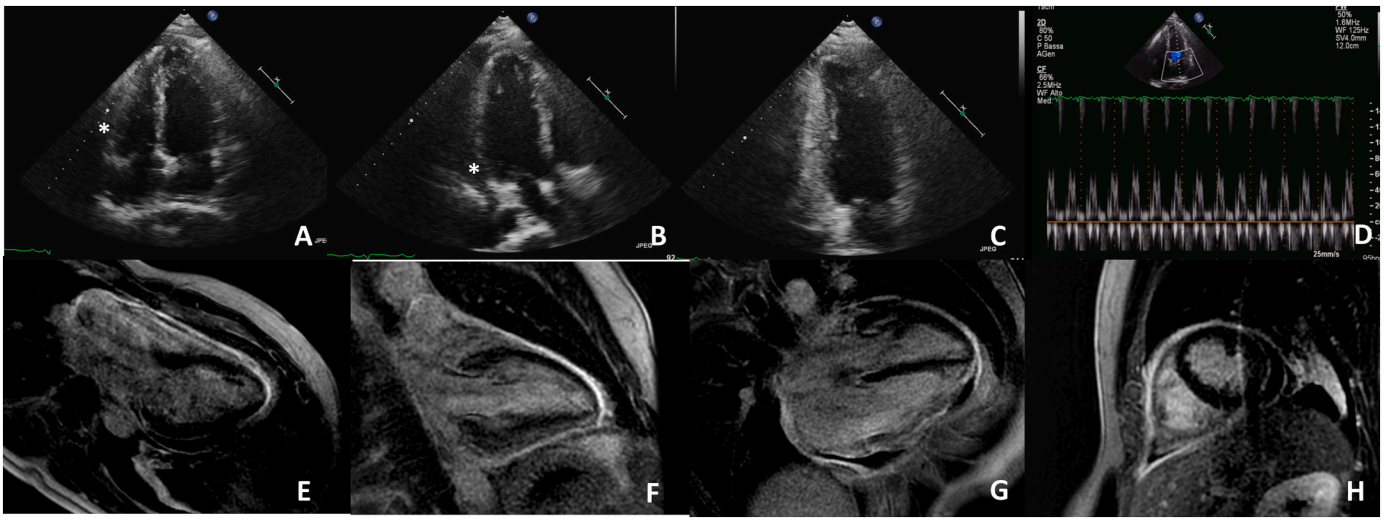

Fig. 2. Case example.
A 34 year old male patients with first episode of acute pericarditis. Transthoracic echocardiography (panel A, B and C) showed mild pericardial effusion ("asterix *" in Panel A and B) without any sign of constriction (panel D). In panel E, F, G and H hyperintensity signal on pericardial sheets at LGE is well evident. The patients had pericarditis recurrence 1 year later.

be underlined that, in the absence of residual inflammation and/or pericarditis recurrences, pericardial LGE is expected to resolve at follow-up after appropriate medical therapy [3]. Thus, in the absence of an event a second cardiac MRI would not be appropriate as clinical (absence of symptoms), bio-humoral (normal CPR) and echocardiographic data (no constriction/pericardial effusion) could be considered as enough to identify a low-risk population in which cardiac MRI would turn out to be negative for pericardial inflammation in 100% of cases.

Of interest, even if no previous studies addressed the role of T1 and T2 mapping in patients with pericarditis, a recently published case series suggested potential role of T1 mapping in distinguishing inflamed pericardial (high signal—bright image) from epicardial fat (low signal—dark image) vs traditional LGE sequences, potentially improving diagnostic performance of cardiac MRI in this setting [19].

Medical therapy for pericarditis is not formally guided by disease severity, but second- or third-line therapies are reserved only to those patients with recurrences [13–20]. However, recurrent pericarditis is indeed an adverse clinical event causing disabling symptoms and early identification of patients with higher risk of recurrences could be of potential clinical interest. In a study by Imazio et al. [2] clinical characteristics (i.e., severe pericardial effusion, fever>38 °C etc.) have been already suggested for identification of patients with first episode of acute pericarditis at higher risk of recurrent pericarditis. Taking into consideration that CMR could not be proposed in all patients with acute pericarditis [14], we included in the present study only patients with at least one of previously described high risk clinical characteristics (Fever >38 °C, subacute onset, large pericardial effusion, cardiac tamponade, lack of response to aspirin or NSAIDs after at least 1 week of therapy, myopericarditis, immunosuppression, trauma, oral anticoagulation therapy), needing hospitalization during acute pericarditis according to ESC guidelines [1]. In this very selected setting of acute pericarditis, characterized by a high rate of recurrence, CMR could be proposed in order to better identify a group of patients at very high risk of recurrence [5]. This very high-risk subgroup may be evaluated for aggressive therapy early in the course of the first episode of pericarditis as previously suggested for patients with recurrent pericarditis [12], for

example with early use of anti-IL 1 drugs. However, no medical therapy schemes could be inferred from the present study.

The time lag between symptoms and is of utmost importance to obtain appropriate information. Indeed, in a study enrolling 128 patients who underwent CMR for recurrent pericarditis, a time delay of more than 4 weeks in performing CMR after symptoms onset was reported to attenuate CMR findings, irrespective to severity of pericarditis [8]. Of note all subjects enrolled in the present study underwent CMR before no more than 7 days after diagnosis.

In any case it must be underlined that CMR evaluation should not be considered as an alternative to clinical and echocardiographic diagnostic evaluation and prognostic stratification, but CMR has a complementary role [21]. Indeed, echocardiography positive for pericardial disease was more common among patients with recurrent pericarditis at follow-up in the present study but did not results to be significative at univariate analysis, possibly due to the limited number of patients enrolled.

The main limitation of the present study is the low number of patients enrolled, even if population power calculation supported results presented in the present manuscript. On this regard, the most important drawback was that we could not perform statistical analysis according to pericarditis etiology. Moreover, the retrospective nature of the study may have introduced potential bias in the clinical selection of patients who underwent CMR, that may represent those with more severe disease. However, it should be acknowledged that the potential clinical interest in performing CMR during the first episode of pericarditis is limited to patients with major clinical risk factors as defined by international guidelines and consensus papers [1–21–23]. Finally, therapy may have been guided by CMR results even if no significant differences in medical therapy were identified between patients with vs. without pericardial events at follow-up.

5. Conclusion

Pericardial inflammation evaluated by CMR identifies patients at higher risk of recurrent pericarditis in a population of patients with first episode of acute pericarditis needing hospitalization. Taking into consideration that recurrent pericarditis is itself an adverse clinical event causing disabling symptoms, further research trials are needed to evaluate the use of second- and third-line therapy early in the course of first episode of acute pericarditis, maybe in those patients with pericardial inflammation at CMR.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2022.03.007.

Author statement

All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

References

[1] Y. Adler, P. Charron, M. Imazio, et al., 2015 ESC guidelines for the diagnosis and management of pericardial diseases: the task force for the diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC) endorsed by: the European Association for Cardio-Thoracic Surgery (EACTS), Eur. Heart J. 36 (42) (2015) 2921–2964.
[2] M. Imazio, E. Cecchi, B. Demichelis, et al., Indicators of poor prognosis of acute pericarditis, Circulation. 115 (21) (2007) 2739–2744.
[3] M. Chetrit, B. Xu, D.H. Kwon, et al., Imaging-guided therapies for pericardial diseases, JACC Cardiovasc. Imaging 13 (6) (2020) 1422–1437.
[4] P. Rajiah, A. Canan, S.S. Saboo, C.S. Restrepo, M.A. Bolen, MRI of the pericardium, Radiographies. 39 (7) (2019 Nov-Dec) 1921–1922.
[5] A. Kumar, K. Sato, B.R. Verma, et al., Quantitative assessment of pericardial delayed hyperenhancement helps identify patients with ongoing recurrences of pericarditis, Open Heart 5 (2) (2018), e000944.
[6] N. Aldweib, V. Farah, R.W.W. Biederman, Clinical utility of cardiac magnetic resonance imaging in pericardial diseases, Curr. Cardiol. Rev. 14 (3) (2018) 200–212.
[7] A. Kumar, K. Sato, E. Yzeiraj, et al., Quantitative pericardial delayed Hyperenhancement informs clinical course in recurrent pericarditis, JACC Cardiovasc. Imaging 10 (11) (2017) 1337–1346.
[8] M. Imazio, E. Pivetta, S. Palacio Restrepo, et al., Usefulness of cardiac magnetic resonance for recurrent pericarditis, Am. J. Cardiol. 125 (1) (2020) 146–151.
[9] R.M. Lang, L.P. Badano, V. Mor-Avi, et al., Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging, Eur. Heart J. Cardiovasc. Imaging 16 (2015) 233–270.
[10] A.M. Taylor, S. Dymarkowski, E.K. Verbeken, et al., Detection of pericardial inflammation with late-enhancement cardiac magnetic resonance imaging: initial results, Eur. Radiol. 16 (2006) 569–574.
[11] M. Chetrit, B. Xu, B.R. Verma, A.L. Klein, Multimodality imaging for the assessment of pericardial diseases, Curr. Cardiol. Rep. 21 (5) (2019 Apr 16) 41.
[12] M.C. Alraies, W. AlJaroudi, H. Yarmohammadi, T. Yingchoncharoen, A. Schuster, A. Senapati, M. Tariq, D. Kwon, B.P. Griffin, A.L. Klein, Usefulness of cardiac magnetic resonance-guided management in patients with recurrent pericarditis, Am. J. Cardiol. 115 (4) (2015 Feb 15) 542–547.
[13] N. Aldweib, V. Farah, R.W.W. Biederman, Clinical utility of cardiac magnetic resonance imaging in pericardial diseases, Curr. Cardiol. Rev. 14 (3) (2018) 200–212.
[14] B. Cosyns, S. Plein, P. Nihoyanopoulos, O. Smiseth, S. Achenbach, M.J. Andrade, M. Pepi, A. Ristic, M. Imazio, B. Paelinck, P. Lancellotti, European Association of Cardiovascular Imaging (EACVI); European Society of Cardiology Working Group (ESC WG) on myocardial and pericardial diseases. European Association of Cardiovascular Imaging (EACVI) position paper: multimodality imaging in pericardial disease. Eur heart J Cardiovasc, Imaging. 16 (1) (2015 Jan) 12–31.
[15] P.C. Cremer, A. Kumar, A. Kontzias, C.D. Tan, E.R. Rodriguez, M. Imazio, A. L. Klein, Complicated pericarditis: understanding risk factors and pathophysiology to inform imaging and treatment, J. Am. Coll. Cardiol. 68 (21) (2016 Nov 29) 2311–2328.
[16] A.O. Zurick, M.A. Bolen, D.H. Kwon, et al., Pericardial delayed hyperenhancement with CMR imaging in patients with constrictive pericarditis undergoing surgical pericardiectomy: a case series with histopathological correlation, JACC Cardiovasc. Imaging 4 (2011) 1180–1191.
[17] G. Lopalco, D. Rigante, L. Cantarini, M. Imazio, A. Lopalco, G. Emmi, V. Venerito, M. Fornaro, B. Frediani, M. Nivuori, A. Brucato, F. Iannone, The autoinflammatory side of recurrent pericarditis: enlightening the pathogenesis for a more rational treatment, Trends Cardiovasc. Med. S1050-1738 (20) (2020 May 3) 30060–30068.
[18] A. Brucato, M. Imazio, P.C. Cremer, Y. Adler, B. Maisch, G. Lazaros, M. Gattorno, A. L.P. Caforio, R. Marcolongo, G. Emmi, A. Martini, A.L. Klein, Recurrent pericarditis: still idiopathic? The pros and cons of a well-honoured term, Intern. Emerg. Med. 13 (6) (2018 Sep) 839–844.
[19] T.B. Mano, H. Santos, S.A. Rosa, B. Thomas, L. Baquero, Cardiac magnetic resonance in the assessment of pericardial abnormalities: a case series, Eur. Heart J. Case Rep. 5 (12) (2021 Nov 8) ytab444.
[20] A. Klein, P. Cremer, A. Kontzias, M. Furqan, R. Tubman, M. Roy, M.Z. Lim-Watson, M. Magestro, US database study of clinical burden and unmet need in recurrent pericarditis, J. Am. Heart Assoc. 10 (15) (2021 Aug 3), e018950.
[21] M. Pepi, M. Muratori, Echocardiography in the diagnosis and management of pericardial disease, J. Cardiovasc. Med. (Hagerstown) 7 (7) (2006 Jul) 533–544.
[22] V. Jain, G. Chhabra, M. Chetrit, A. Bansal, F. Berglund, B. Montanè, M.M. Furqaan, S.P. Bachour, A. Kontzias, A.V. Forte, W.S. Aronow, M. Imazio, P.C. Cremer, A. L. Klein, Role of non-invasive multimodality imaging in autoimmune pericarditis, Eur. Heart J. Cardiovasc. Imaging (2021 Aug 1), https://doi.org/10.1093/ehjci/jeab131 jeab131.
[23] M. Chetrit, B. Xu, D.H. Kwon, J. Ramchand, R.E. Rodriguez, C.D. Tan, C.L. Jellis, D. R. Johnston, R.D. Renapurkar, P.C. Cremer, A.L. Klein, Imaging-guided therapies for pericardial diseases, JACC Cardiovasc. Imaging 13 (6) (2020 Jun) 1422–1437.