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Utility of native T1 mapping and myocardial extracellular volume fraction in patients with nonischemic dilated cardiomyopathy: A systematic review and meta-analysis

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ABSTRACT

Background: Cardiac magnetic resonance imaging (CMR) based T1 mapping and extracellular volume fraction (ECV) are powerful tools for identifying myocardial fibrosis. This systematic review and *meta*-analysis aims to characterize the utility of native T1 mapping and ECV in patients with non-ischemic cardiomyopathy (NICM) and to clarify the prognostic significance of elevated values.

Methods: A literature search was conducted for studies reporting on use of CMR-based native T1 mapping and ECV measurement in NICM patients and their association with major adverse cardiac events (MACE), ventricular arrhythmias (VAs), and left ventricular reverse remodeling (LVRR). Databases searched included: Ovid MEDLINE, EMBASE, Web of Science, and Google Scholar. The search was not restricted to time or publication status.

Results: Native T1 and ECV were significantly higher in NICM patients compared to controls (MD 78.80, 95 % CI 50.00, 107.59; $p < 0.01$; MD 5.86, 95 % CI 4.55, 7.16; $p < 0.01$). NICM patients who experienced MACE had higher native T1 and ECV (MD 52.87, 95 % CI 26.59, 79.15; $p < 0.01$; MD 6.03, 95 % CI 3.79, 8.26; $p < 0.01$). There was a non-statistically significant trend toward higher native T1 time in NICM patients who experienced VAs. NICM patients who were poor treatment responders had higher baseline native T1 and ECV (MD 40.58, 95 % CI 12.90, 68.25; $p < 0.01$; MD 3.29, 95 % CI 2.25, 4.33; $p < 0.01$).

Conclusions: CMR-based native T1 and ECV quantification may be useful tools for risk stratification of patients with NICM. They may provide additional diagnostic utility in combination with LGE, which poorly characterizes fibrosis in patients with diffuse myocardial involvement.

1. Introduction

Nonischemic Cardiomyopathy (NICM) is a heterogeneous classification of several myocardial disorders represented by both structural and functional abnormalities in the absence of significant flow-limiting coronary artery disease (CAD). The prevalence of NICM is estimated at approximately 40 to 50 cases per 100,000 people worldwide and is associated with significantly increased morbidity and mortality through progressive pump failure and fatal arrhythmias [1].

Appropriate risk stratification of patients with NICM is essential and an area that is rapidly evolving. Advancements in cardiac magnetic resonance imaging (CMR) have allowed for greater potential to qualitatively assess the myocardium in NICM patients and to identify characteristics that may be associated with adverse cardiovascular outcomes. Late gadolinium enhancement (LGE), a marker of focal fibrosis, has been shown to strongly predict future adverse cardiovascular events in patients with cardiomyopathy [2–4]. However, it is a suboptimal marker for identifying diffuse interstitial fibrosis, which is often involved in the development of NICM.

Myocardial T1 mapping and extracellular volume fraction (ECV) are novel methods by which diffuse myocardial pathology can be evaluated on CMR; however, their use in NICM remains unclear. In this paper, we summarize the evidence linking the utility and prognostic role of T1 mapping and ECV with adverse outcomes in patients with NICM.

Abbreviations: CAD, coronary artery disease; CMR, cardiac magnetic resonance imaging; ECV, extracellular volume; HF, heart failure; ICM, ischemic cardiomyopathy; LGE, late gadolinium enhancement; MACE, major adverse cardiovascular events; MOLLI, modified Look–Locker inversion recovery; NICM, nonischemic cardiomyopathy; SCD, sudden cardiac death; shMOLLI, shortened modified Look-Locker Inversion recovery; VA, ventricular arrhythmias.

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2. Methods

2.1. Data search

This systematic review was performed in adherence to the guidelines of the PRISMA statement (Preferred Reporting Items for Systematic Reviews and Meta-analyses). The review was performed using a pre-planned protocol in September 2022. The primary endpoint was major adverse cardiac events (MACE). Secondary endpoints included ventricular arrhythmias (VA) and left ventricular reverse remodeling (LVRR). MACE was defined as all-cause mortality, cardiac transplantation, heart failure (HF) hospitalization. LVRR was defined as an improvement in left ventricular ejection fraction (LVEF) by more than 10 %. VAs were defined as the combined incidence of sustained ventricular tachycardia, sudden cardiac death, and appropriate implantable cardioverter-defibrillator shocks.

2.2. Search strategy

A systematic search was conducted using Ovid MEDLINE, EMBASE, Scopus, Web of Science, and Google Scholar for relevant literature that reported an association between native T1 and ECV in CMR with MACE and LVRR. The search was not restricted to time or publication status. Two independent reviewers (MT and DG) performed an electronic search using the following keywords: “extracellular volume”, “ECV”, “T1”, “nonischaemic”, “nonischemic”, “nonischemics”, “cardiomyopathy”, “dilated”. The references of the included studies, other systematic reviews, and *meta*-analyses were further manually reviewed to obtain a comprehensive list of studies. After identifying relevant studies, the full texts of the selected articles were examined by both reviewers based on inclusion criteria. Disagreements were resolved through discussion and consensus.

2.3. Study selection

Studies were selected using the PICO format to include those that studied patients with NICM (Population), compared increased ECV and native T1 values (Intervention) to normal ECV and native T1 values (Comparison), and assessed for clinical endpoints described above (Outcomes). Studies that did not distinguish between mixed ischemic and NICM patient populations were excluded. Studies that specified the inclusion of hypertrophic cardiomyopathy and amyloid patients as NICM were excluded.

2.4. Data extraction

Two reviewers (SS and MT) independently extracted the study data using a predefined data extraction sheet. Variables that were extracted from the studies included: Lead author, year of publication, study design, mean follow-up duration, mean age, gender, average LVEF, left ventricular end-diastolic volume index (LVEDVI), CMR mapping method, percentage of patients with LGE, ECV and native T1 values, and number of patients with clinical endpoints based on ECV and native T1 values.

2.5. Statistical analysis

Meta-analysis was performed using Cochrane Review Manager [47]. Majority of studies reported baseline ECV and native T1 values in patients who subsequently did or did not experience clinical endpoints. Statistical analysis was performed with results reported as: mean difference (MD), upper and lower limits for 95 % confidence interval, and Z-value. Heterogeneity was determined by I2 with 0 % to 40 % defined as low, 40 % to 70 % defined as moderate, 70 % to 100 % defined as high heterogeneity. A fixed effect model was used when heterogeneity was low to moderate or if sample sizes were small. A random effect model was used when heterogeneity was high and sample sizes were large. Statistical significance was considered with a P-value < 0.05, and all tests were 2-sided.

3. Results

3.1. Literature search and study selection

We identified 58 potentially eligible studies from our initial literature search (Fig. 1). After reviewing all studies in full text, 24 studies were identified to be eligible for *meta*-analysis based on the inclusion and exclusion criteria listed above.

3.2. Study and patient characteristics

This *meta*-analysis included prospective and retrospective cohort studies (Tables 1 and 2). A total of 2,966 patients (862 with NICM and 2,106 controls) were included in studies reporting native T1 and ECV values in patients with NICM compared to healthy controls. A total of 1,286 patients (193 with MACE and 1,093 without MACE) were included in studies reporting baseline native T1 and ECV values in NICM patients who on follow-up experienced MACE compared to NICM patients who did not experience MACE. A total of 2,213 patients (598 with LVRR and 1,615 without LVRR) were included in studies reporting baseline native T1 and ECV values in NICM patients who on follow-up had LVRR compared to NICM patients who did not have LVRR. The mean follow-up duration was 17 months.

3.3. Comparison of native T1 and ECV values of NICM patients and healthy controls

Patients with NICM had a significantly higher native T1 time compared to healthy control patients (MD 78.80, 95 % CI 50.00, 107.59; $p < 0.01$). Subgroup analysis of native T1 time, based on method of T1 mapping sequence, demonstrated this result to be statistically significant regardless of whether the modified Look–Locker inversion recovery (MOLLI) or shortened modified Look-Locker Inversion recovery (shMOLLI) method was used (Fig. 2). Patients with NICM had significantly higher ECV compared to healthy control patients (MD 5.86, 95 % CI 4.55, 7.26; $p < 0.01$). Subgroup analysis by LGE demonstrated this result to be significant regardless of whether LGE was absent or present (Fig. 3). The heterogeneity was high for both native T1 time and ECV ($I^2 = 93$ %; $I^2 = 85$ %) and could not be addressed through subgroup analysis; thus, a random effects model of analysis was used.

3.4. Association of native T1 time and ECV values with MACE and VAs

NICM patients who experienced MACE, defined as all-cause mortality, need for transplant, or HF hospitalization on follow-up, had statistically significant higher baseline native T1 time (MD 52.87, 95 % CI 26.59, 79.15; $p < 0.01$), and heterogeneity was moderate ($I^2 = 46$ %) (Fig. 4). NICM patients who experienced MACE had statistically significantly higher ECV compared to NICM patients who did not experience MACE (MD 6.03, 95 % CI 3.79. 8.26; $p < 0.01$), and heterogeneity was high ($I^2 = 86$ %) (Fig. 5). NICM patients who experienced VAs did not have a statistically significantly higher baseline native T1 time compared to patients who did not experience VAs (MD 39.28, 95 % CI −7.63, 86.20; $p = 0.10$), and heterogeneity was high ($I^2 = 80$ %) (Fig. 6). There was inadequate information in the studies reporting on MACE to perform further subgroup analysis by individual endpoints.

3.5. Association of native T1 time and ECV with LVRR

NICM patients who were poor treatment responders and did not have LVRR, had significantly higher native T1 times and ECV values compared to treatment responders (MD 40.58, 95 % CI 12.90, 68.25; p

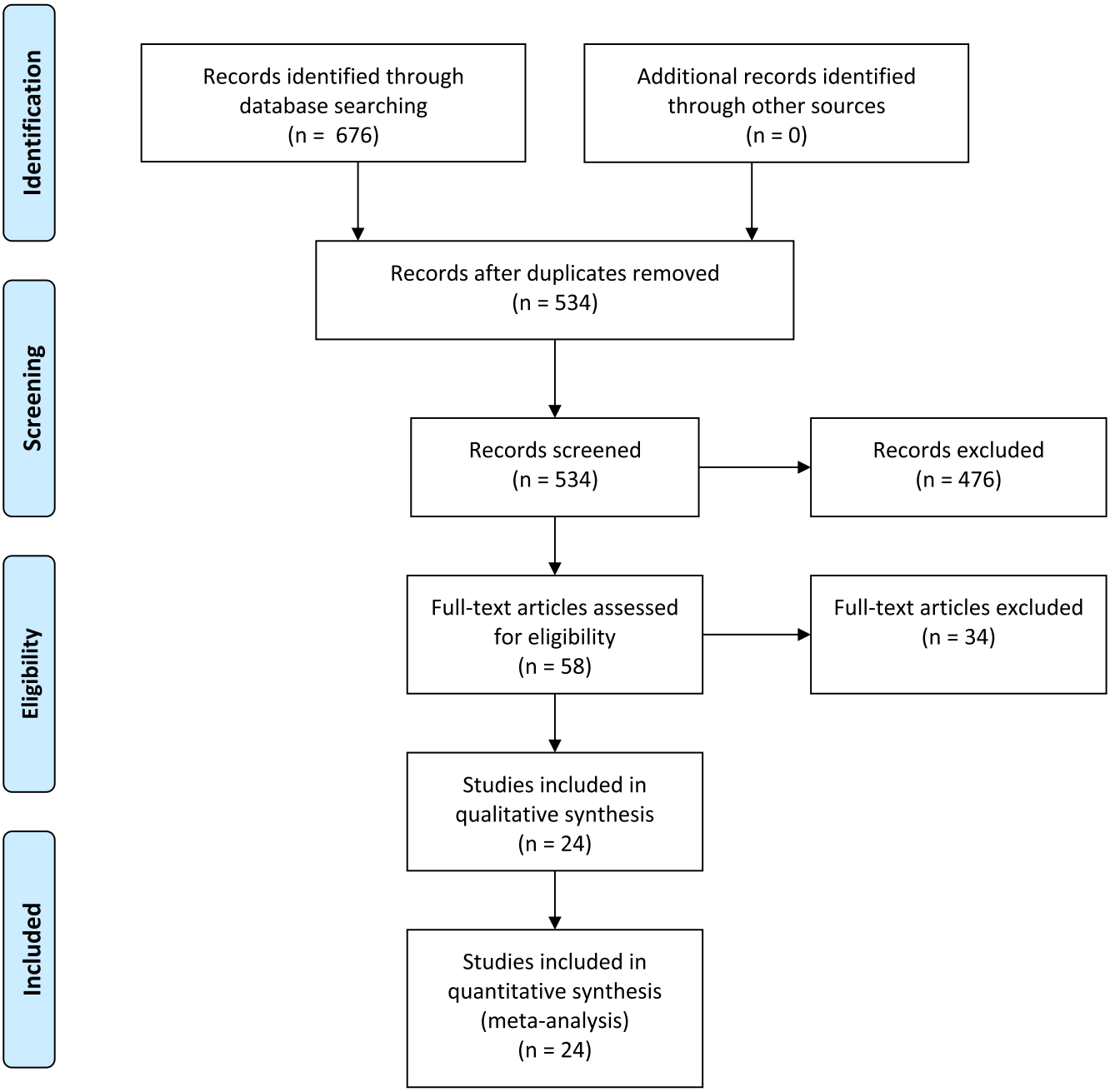

Fig. 1. PRISMA Flow Diagram. Flow diagram depicting study selection for inclusion based on the PRISMA statement for reporting systematic reviews and *meta*-analyses.

< 0.01; MD 3.29, 95 % CI 2.25, 4.33; $p < 0.01$) (Figs. 7 and 8). The heterogeneity was high for native T1 time, but low for ECV value ($I^2 =$ 78 % and $I^2 =$ 25 %, respectively).

4. Discussion

This *meta*-analysis evaluated the role of native T1 mapping and ECV in prognostication of patients with NICM and demonstrated that both techniques can be used as effective tools for further risk stratification. The main findings were as follows: (a) Compared to healthy controls, NICM patients have higher native T1 and ECV values; (b) Higher native T1 and ECV values are associated with significantly higher risk of MACE and a trend toward higher risk of VA; (c) Elevated native T1 and ECV values may identify NICM patients who are likely to be non-responders to medical and device therapies.

Traditionally, LVEF has been considered the leading determinant for risk stratification of patients with heart failure with reduced ejection fraction. While this association has been well established in patients with ischemic cardiomyopathy, the benefits have been less clear in NICM patients. LVEF may not consistently predict adverse cardiovascular outcomes, and with appropriate medical and device therapies, patients with severe systolic dysfunction can experience drastic improvement in their LVEF [5,6]. Thus, it is imperative to identify methods for further risk stratifying patients with NICM. T1 mapping and ECV have a direct relationship with diffuse myocardial fibrosis and in turn may serve as independent predictors with prognostic value given their ability to approximate adverse outcomes in NICM.

Although there has been considerable evidence to demonstrate the utility of LGE in identifying patients who are at higher risk for worse prognostic outcomes, its role is limited in detecting diffuse myocardial

Table 1
Demographic data of studies comparing NICM patients to healthy controls: LGE: late gadolinium enhancement, **LVEDVI:** left ventricular end-diastolic volume index (indexed by body surface area), **LVEF:** left ventricular ejection fraction, **MOLLI:** modified look-locker inversion recovery, **N/A:** not available, **NICM:** non-ischemic cardiomyopathy, **SASHA:** saturation recovery single-shot acquisition, **shMOLLI:** shortened modified look-locker inversion recovery.

Name	Year	Mean Age (Years)	Male (%)	Mean LVEF (%)	Mean LVEDVI	% with LGE	Mapping Method
Dass [30]	2012	57	50	37	91	66	shMOLLI
Puntmann [27]	2013	44	65	34	110	33	MOLLI
Siepen [31]	2014	54	67	31	142	48	MOLLI
Puntmann [17]	2014	N/A	N/A	39	123	23	MOLLI
Barison [32]	2015	59	71	41	121	44	N/A
Hong [33]	2015	53	65	24	160	0	MOLLI
Chen [5]	2016	64	95	22	N/A	N/A	MOLLI
Mordi [22]	2016	47	100	48	101	25	MOLLI
Puntmann [34]	2016	50	62	47	109	27	MOLLI
Goebel [26]	2016	50	78	30	N/A	N/A	MOLLI
Costello [25]	2017	51	57	31	131	0	SASHA
Youn [35]	2017	52	61	25	159	70	MOLLI
Inui [13]	2018	60	67	27	132	61	MOLLI
Chen [36]	2019	47	72	20	202	80	MOLLI
Cui [37]	2019	45	74	17	196	67	MOLLI
Vita [38]	2019	49	62	43	114	35	MOLLI
Mondy [28]	2020	38	42	N/A	N/A	N/A	shMOLLI
Rubis [39]	2021	55	89	29	N/A	N/A	MOLLI
Xu [16]	2021	46	68	25	171	45	MOLLI
Di Marco [40]	2022	59	66	42	111	41	MOLLI
Gao [41]	2022	44	50	38	114	0	MOLLI
Kinoshita [15]	2022	63	81	37	N/A	42	MOLLI

Table 2
Demographic data of studies reporting the association of ECV and native T1 values with clinical outcomes in NICM patients: LGE: late gadolinium enhancement, **LVEDVI:** left ventricular end-diastolic volume index (indexed by body surface area), **LVEF:** left ventricular ejection fraction, **MOLLI:** modified look-locker inversion recovery, **N/A:** not available, **NICM:** non-ischemic cardiomyopathy, **shMOLLI:** shortened modified look-locker inversion recovery, **STONE:** slice-interleaved T1 mapping.

Name	Year	Mean Age (Years)	Male (%)	Mean LVEF (%)	Mean LVEDVI	% w/LGE	Mapping Method
Dass [30]	2012	57	50	37	91	66	shMOLLI
Puntmann [27]	2013	44	65	34	110	33	MOLLI
Puntmann [17]	2014	N/A	N/A	39	123	23	MOLLI
Siepen [31]	2014	54	67	31	142	48	MOLLI
Puntmann [34]	2016	50	62	47	109	27	MOLLI
Chen [5]	2016	64	95	22	N/A	N/A	MOLLI
Goebel [26]	2016	50	78	30	N/A	N/A	MOLLI
Youn [35]	2017	52	61	25	159	70	MOLLI
Costello [25]	2017	51	57	31	131	0	ShMOLLI
Nakamori [42]	2018	53	72	33	132	33	STONE
Inui [13]	2018	60	67	27	132	61	MOLLI
Chen [36]	2019	47	72	20	202	80	MOLLI
Vita [38]	2019	49	62	43	114	35	MOLLI
Mondy [28]	2020	38	42	N/A	N/A	N/A	shMOLLI
Rubis [39]	2021	55	89	29	N/A	N/A	MOLLI
Xu [16]	2021	46	68	25	171	45	MOLLI
Kitagawa [14]	2022	57	65	25	N/A	40	MOLLI
Kinoshita [15]	2022	63	81	37	N/A	42	MOLLI
Gao [41]	2022	44	50	N/A	N/A	N/A	MOLLI

fibrosis [7,8]. LGE relies on the direct comparison of enhanced myocardium to normal myocardium; however, identification of diffuse fibrosis is difficult given normal myocardium is absent for reference [9,10]. In contrast, T1 mapping and ECV are neither dependent on normal myocardium for reference, nor are they dependent on local differences in image contrast [11]. Both T1 mapping and ECV have demonstrated consistency in detecting diffuse fibrosis when compared to endomyocardial biopsy results [11,12]. Additionally, routine surveillance with T1 mapping and ECV may assist with predicting treatment response, as well and improvement in cardiac function [13–17]. One study demonstrated the prognostic role of ECV where, over a median follow-up of 1.7 years, elevated ECV was associated with increased risk for heart failure hospitalizations and death [18]. Moreover, several studies have confirmed the prognostic potential of T1 mapping and ECV in predicting CV outcomes[19–21].

Lastly, it must be noted that abnormal T1 and ECV results can be attributed to other disease processes affecting the myocardium, including myocarditis, hypertrophic cardiomyopathy, and cardiac amyloidosis. For example, there is robust evidence demonstrating an increase in ECV and T1 values in patients with hypertrophic cardiomyopathy when compared to both athletic hearts and normal healthy controls [22–24]. Thus, the elevation in native T1 relaxation time and ECV may serve as general markers for myocardial pathology, but additional workup is required to identify a specific etiology.

There are several limitations to our current *meta*-analysis which must be addressed. One limitation is the heterogeneity present between studies included for analysis. We believe that this is likely a result of differences in the CMR acquisition techniques, as well as the criteria used to define NICM. T1 relaxation time is dependent on several factors, including the strength of the magnetic field and the acquisition technique employed. Although there are two common acquisition techniques, such as MOLLI and shMOLLI pulse sequences, there is no

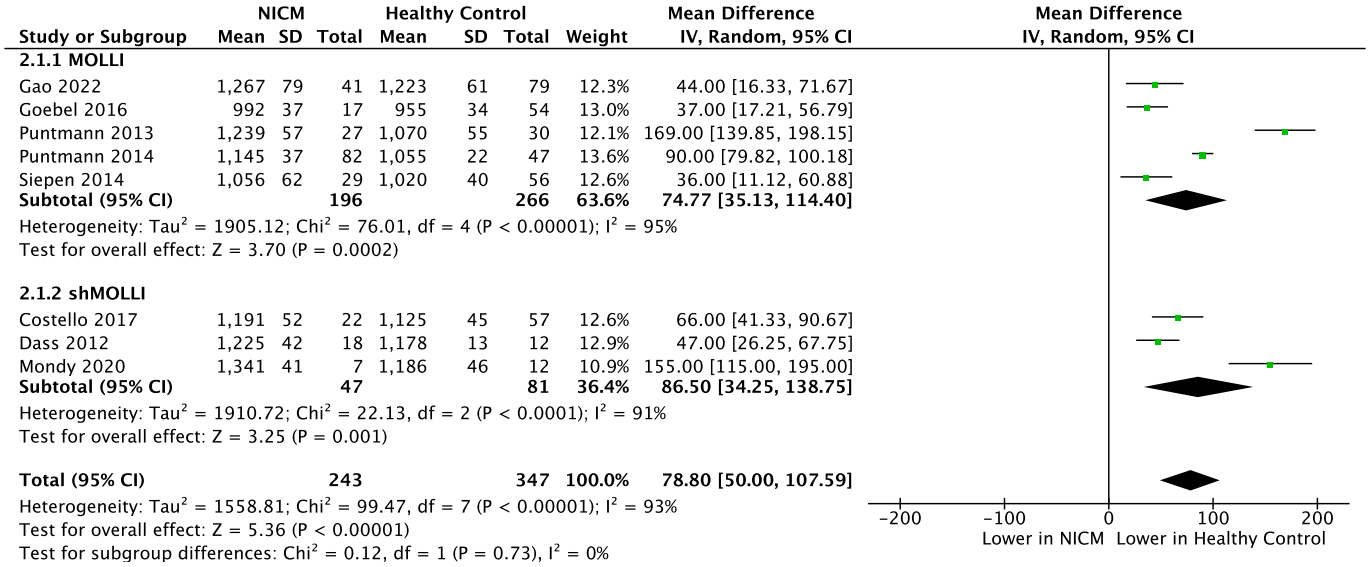

Fig. 2. Comparison of native T1 time between NICM patients and healthy control patients. Native T1 time was significantly higher in patients with NICM with a mean difference of 78.80 (95 % confidence interval 50.00, 107.59; $p < 0.01$).

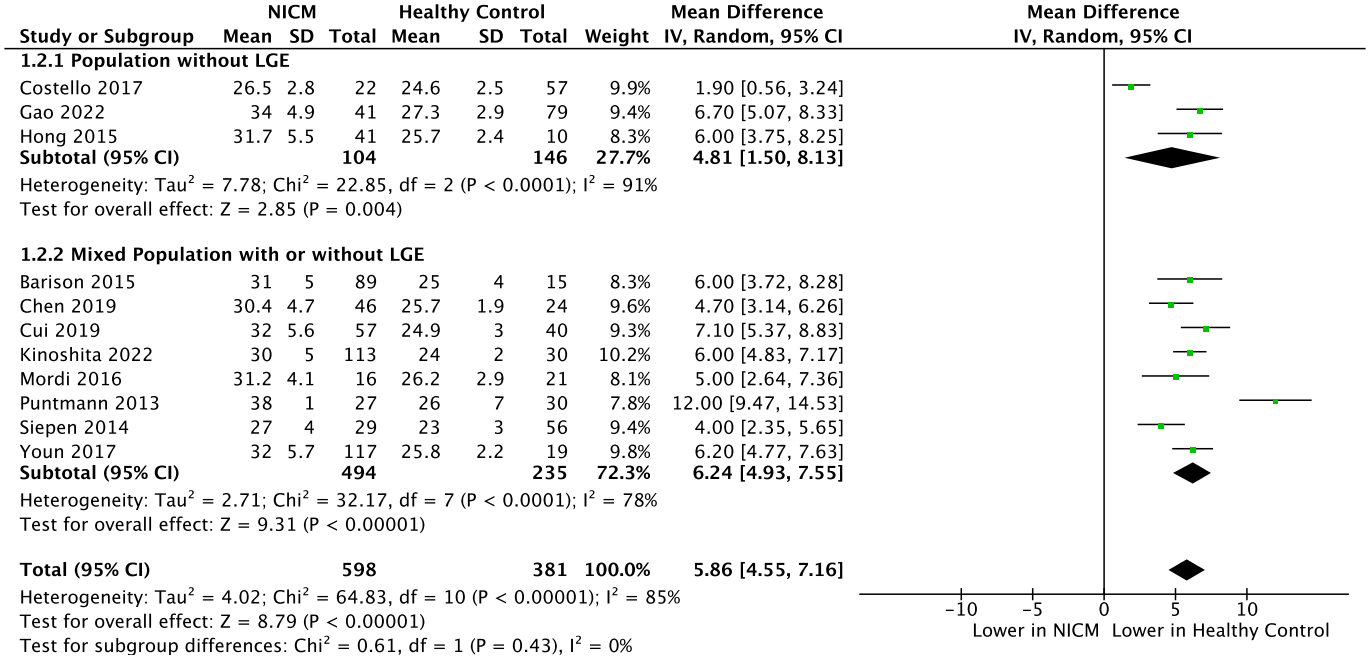

Fig. 3. Comparison of ECV between NICM patients and healthy control patients. ECV was significantly higher in patients with NICM with a mean difference of 5.86 (95 % confidence interval 4.55, 7.16; $p < 0.01$).

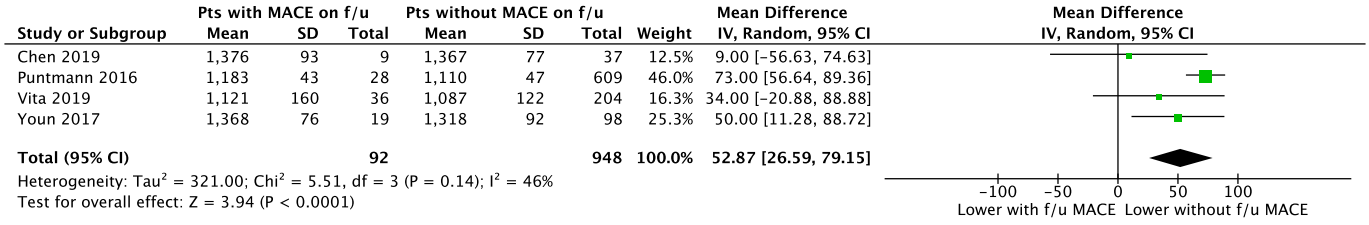

Fig. 4. Association of native T1 time with risk of MACE in NICM patients. Baseline native T1 time was statistically significantly higher in NICM patients who subsequently experienced MACE compared to NICM patients who did not experience MACE with a mean difference of 52.87 (95 % confidence interval 26.59, 79.15; $p < 0.01$).

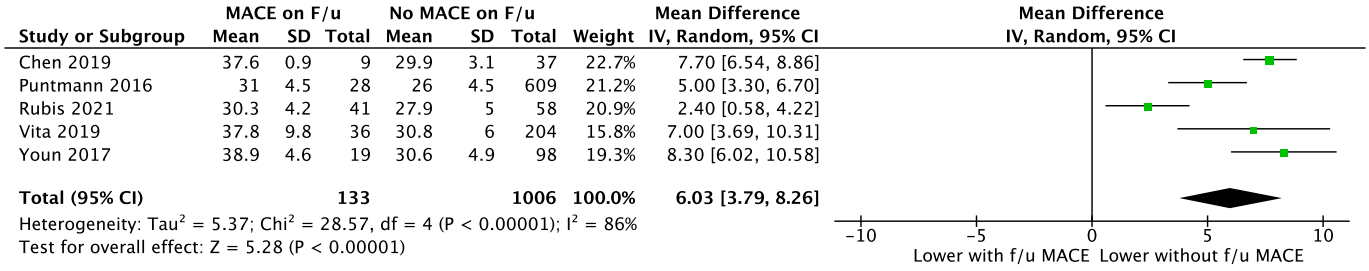

Fig. 5. Association of ECV with risk of MACE in NICM patients. ECV was statistically significantly higher in patients who experienced MACE during follow-up compared to patients who did not experience MACE with a mean difference of 6.03 (95 % confidence interval 3.79, 8.26; $p < 0.01$).

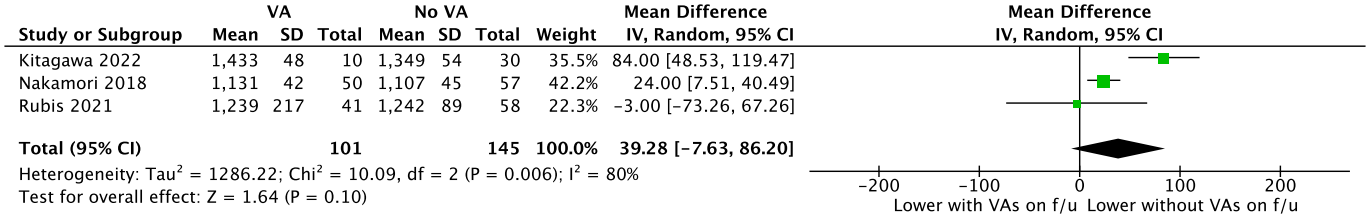

Fig. 6. Association of native T1 time with risk of VAs in NICM patients. There is a trend toward higher baseline native T1 time in NICM patients who subsequently experienced VAs compared to NICM patients who did not experience VAs, however this association was not statistically significant with a mean difference of 39.28 (95 % confidence interval −7.63, 86.20; $p = 0.10$).

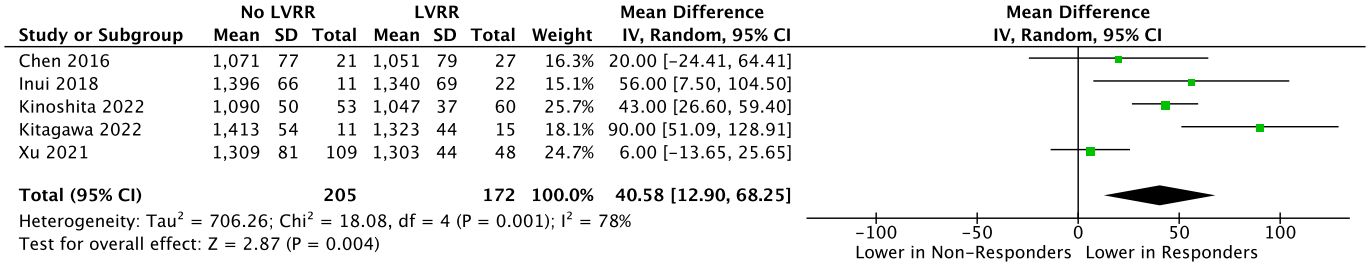

Fig. 7. Association of native T1 time with LVRR Patients who did not have LVRR had significantly higher native T1 time compared to patients who had LVRR on follow-up with a mean difference of 40.58 (95 % confidence interval 12.90, 68.25; $p < 0.01$).

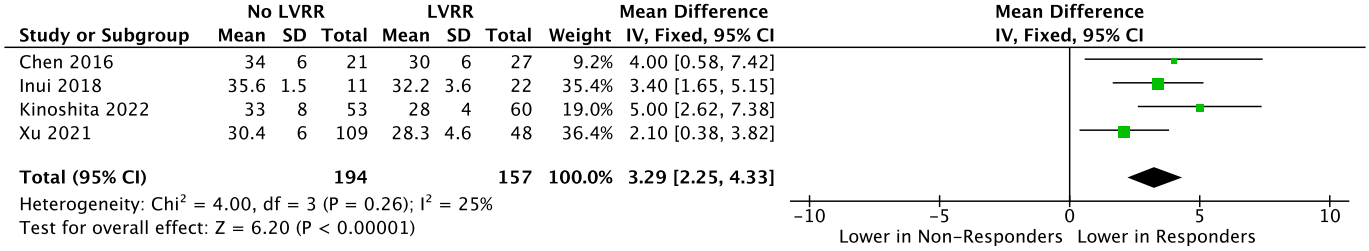

Fig. 8. Association of ECV with LVRR Patients who did not have LVRR had significantly higher ECV compared to patients who had LVRR on follow-up with a mean difference of 3.29 (95 % confidence interval 2.25, 4.33; $p < 0.01$).

standardization of acquisition method; moreover, additional methods are currently being established and evaluated [25–28]. Based on these factors, high heterogeneity was expected and addressed through both subgroup analysis and through use of a random effects model, which accounted for differences in study design and measurements between studies.

One surprising result from our analysis was that native T1 values were not significantly different between patients who experienced VAs compared to those who did not. These results are in stark contrast to those seen with LGE, in which focal fibrosis > 15 % and fibrosis location along the lateral, septal or mid LV walls were significantly higher in patients experiencing VAs [5,29]. Our results, however, do show a trend toward higher native T1 values in NICM patients who experienced VAs; the lack of statistical significance is likely due to low statistical power within the small sample sizes seen in the three included studies. Thus, additional high-quality studies, with larger sized cohorts, are required for further evaluation of the association between native T1 values and risk of VAs.

5. Conclusions

Given the significant morbidity and mortality associated with NICM, it is imperative to establish useful tools to further risk stratify patients. Although limited by heterogeneity, this *meta*-analysis substantiates the utility of native T1 mapping and ECV in patients with NICM, and identifies the association between increased native T1 relaxation time and ECV measurements with risk for all-cause mortality, HF hospitalization, need for cardiac transplant, and poor response to guideline directed medical and device therapy for HF. The prognostic utility of ECV measurements and T1 mapping in predicting risk of VAs remains to be further elucidated.

Funding

None.

Guidelines statement

The systematic review was conducted with a protocol in accordance with the Preferred Reporting of Items for Systematic review and Meta-Analysis (PRISMA) statement.

Declaration of competing interest

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.

References

[1] M.B. Codd, et al., Epidemiology of idiopathic dilated and hypertrophic cardiomyopathy. A population-based study in Olmsted County, Minnesota, 1975-1984, Circulation 80 (3) (1989) 564–572.
[2] E. Wu, et al., Infarct size by contrast enhanced cardiac magnetic resonance is a stronger predictor of outcomes than left ventricular ejection fraction or end-systolic volume index: prospective cohort study, Heart 94 (6) (2008) 730–736.
[3] K.C. Wu, et al., Late gadolinium enhancement by cardiovascular magnetic resonance heralds an adverse prognosis in nonischemic cardiomyopathy, J. Am. Coll. Cardiol. 51 (25) (2008) 2414–2421.
[4] O. Catalano, et al., Late gadolinium enhancement by cardiovascular magnetic resonance is complementary to left ventricle ejection fraction in predicting prognosis of patients with stable coronary artery disease, J. Cardiovasc. Magn. Reson. 14 (1) (2012) 29.
[5] Z. Chen, et al., Focal but not diffuse myocardial fibrosis burden quantification using cardiac magnetic resonance imaging predicts left ventricular reverse modeling following cardiac resynchronization therapy, J. Cardiovasc. Electrophysiol. 27 (2) (2016) 203–209.
[6] A. Aleksova, et al., Natural history of dilated cardiomyopathy: from asymptomatic left ventricular dysfunction to heart failure–a subgroup analysis from the Trieste cardiomyopathy registry, J. Cardiovasc. Med. (Hagerstown) 10 (9) (2009) 699–705.
[7] E. Donal, et al., Multimodality imaging in the diagnosis, risk stratification, and management of patients with dilated cardiomyopathies: an expert consensus document from the European Association of Cardiovascular Imaging, Eur. Heart J. Cardiovasc. Imaging 20 (10) (2019) 1075–1093.
[8] J.A. McCrohon, et al., Differentiation of heart failure related to dilated cardiomyopathy and coronary artery disease using gadolinium-enhanced cardiovascular magnetic resonance, Circulation 108 (1) (2003) 54–59.
[9] C. Parsai, et al., Diagnostic and prognostic value of cardiovascular magnetic resonance in non-ischaemic cardiomyopathies, J. Cardiovasc. Magn. Reson. 14 (1) (2012) 54.
[10] T. Rogers, et al., Standardization of T1 measurements with MOLLI in differentiation between health and disease–the ConSept study, J. Cardiovasc. Magn. Reson. 15 (1) (2013) 78.
[11] P. Haaf, et al., Cardiac T1 mapping and extracellular volume (ECV) in clinical practice: a comprehensive review, J. Cardiovasc. Magn. Reson. 18 (1) (2016) 89.
[12] N. Mewton, et al., Assessment of myocardial fibrosis with cardiovascular magnetic resonance, J. Am. Coll. Cardiol. 57 (8) (2011) 891–903.
[13] K. Inui, et al., Extracellular volume fraction assessed using cardiovascular magnetic resonance can predict improvement in left ventricular ejection fraction in patients with dilated cardiomyopathy, Heart Vessels 33 (10) (2018) 1195–1203.
[14] T. Kitagawa, et al., Native myocardial T1 value in predicting 1-year outcomes in patients with nonischemic dilated cardiomyopathy experiencing recent heart failure, Int. Heart J. 63 (3) (2022) 531–540.
[15] M. Kinoshita, et al., Native T1 heterogeneity for predicting reverse remodeling in patients with non-ischemic dilated cardiomyopathy, Heart Vessels 37 (9) (2022) 1541–1550.
[16] Y. Xu, et al., Myocardial tissue reverse remodeling after guideline-directed medical therapy in idiopathic dilated cardiomyopathy, Circulation Heart Failure 14 (1) (2021) e007944.
[17] V.O. Puntmann, et al., Aortic stiffness and interstitial myocardial fibrosis by native T1 are independently associated with left ventricular remodeling in patients with dilated cardiomyopathy, Hypertension 64 (4) (2014) 762–768.
[18] E.B. Schelbert, et al., Myocardial fibrosis quantified by extracellular volume is associated with subsequent hospitalization for heart failure, death, or both across the spectrum of ejection fraction and heart failure stage, J. Am. Heart Assoc. 4 (12) (2015).
[19] F. Fröjdh, et al., Extracellular volume and global longitudinal strain both associate with outcomes but correlate minimally, J. Am. Coll. Cardiol. Img. 13 (11) (2020) 2343–2354.
[20] A. González, et al., Myocardial interstitial fibrosis in heart failure: Biological and translational perspectives, J. Am. Coll. Cardiol. 71 (15) (2018) 1696–1706.
[21] A. Seno, et al., Prognostic value of T1 mapping and feature tracking by cardiac magnetic resonance in patients with signs and symptoms suspecting heart failure and no clinical evidence of coronary artery disease, J. Am. Heart Assoc. 11 (2) (2022) e020981.
[22] I. Mordi, et al., T1 and T2 mapping for early diagnosis of dilated non-ischaemic cardiomyopathy in middle-aged patients and differentiation from normal physiological adaptation, Eur. Heart J. – Cardiovasc. Imaging 17 (7) (2015) 797–803.
[23] P.P. Swoboda, et al., Assessing myocardial extracellular volume by T1 mapping to distinguish hypertrophic cardiomyopathy from athlete's heart, J. Am. Coll. Cardiol. 67 (18) (2016) 2189–2190.
[24] D.M. Sado, et al., Cardiovascular magnetic resonance measurement of myocardial extracellular volume in health and disease, Heart 98 (19) (2012) 1436–1441.
[25] B.T. Costello, et al., SASHA versus ShMOLLI: a comparison of T1 mapping methods in health and dilated cardiomyopathy at 3 T, Int. J. Cardiovasc. Imaging 33 (10) (2017) 1551–1560.
[26] J. Goebel, et al., Can native T1 mapping differentiate between healthy and diffuse diseased myocardium in clinical routine cardiac MR imaging? PLoS One 11 (5) (2016) e0155591.
[27] V.O. Puntmann, et al., Native T1 mapping in differentiation of Normal myocardium from diffuse disease in hypertrophic and dilated cardiomyopathy, J. Am. Coll. Cardiol. Img. 6 (4) (2013) 475–484.
[28] V.C. Mondy, S.B. Peter, R. Ravi, Native T1 mapping in diffuse myocardial diseases using 3-tesla MRI: An institutional experience, Indian J Radiol Imaging 30 (4) (2020) 465–472.
[29] C. Schiau, et al., Myocardial fibrosis as a predictor of ventricular arrhythmias in patients with non-ischemic cardiomyopathy, In Vivo 35 (3) (2021) 1677–1685.
[30] S. Dass, et al., Myocardial tissue characterization using magnetic resonance noncontrast T1 mapping in hypertrophic and dilated cardiomyopathy, Circ. Cardiovasc. Imaging 5 (6) (2012) 726–733.
[31] F. aus dem Siepen, et al., T1 mapping in dilated cardiomyopathy with cardiac magnetic resonance: quantification of diffuse myocardial fibrosis and comparison with endomyocardial biopsy, Eur. Heart J. – Cardiovasc. Imaging 16 (2) (2014) 210–216.
[32] A. Barison, et al., Prognostic significance of myocardial extracellular volume fraction in nonischaemic dilated cardiomyopathy, J. Cardiovasc. Med. (Hagerstown) 16 (10) (2015) 681–687.
[33] Y.J. Hong, et al., Extracellular volume fraction in dilated cardiomyopathy patients without obvious late gadolinium enhancement: comparison with healthy control subjects, Int. J. Cardiovasc. Imaging 31 (1) (2015) 115–122.
[34] V.O. Puntmann, et al., T1-mapping and outcome in nonischemic cardiomyopathy: all-cause mortality and heart failure, J. Am. Coll. Cardiol. Img. 9 (1) (2016) 40–50.
[35] J.-C. Youn, et al., Contrast-enhanced T1 mapping-based extracellular volume fraction independently predicts clinical outcome in patients with non-ischemic dilated cardiomyopathy: a prospective cohort study, Eur. Radiol. 27 (9) (2017) 3924–3933.
[36] R. Chen, et al., The comparison of short-term prognostic value of T1 mapping with feature tracking by cardiovascular magnetic resonance in patients with severe dilated cardiomyopathy, Int. J. Cardiovasc. Imaging 35 (1) (2019) 171–178.
[37] Y. Cui, et al., Myocardial extracellular volume fraction measurements with MOLLI 5(3)3 by cardiovascular MRI for the discrimination of healthy volunteers from dilated and hypertrophic cardiomyopathy patients, Clin. Radiol. 74 (9) (2019) 732.e9–732.e16.
[38] T. Vita, et al., Comparing CMR mapping methods and myocardial patterns toward heart failure outcomes in nonischemic dilated cardiomyopathy, J. Am. Coll. Cardiol. Img. 12 (8 Pt 2) (2019) 1659–1669.
[39] P.P. Rubiś, et al., Extracellular volume is an independent predictor of arrhythmic burden in dilated cardiomyopathy, Sci. Rep. 11 (1) (2021) 24000.
[40] A. Di Marco, et al., Extracellular volume fraction improves risk-stratification for ventricular arrhythmias and sudden death in non-ischaemic cardiomyopathy, Eur. Heart J. – Cardiovasc. Imaging (2022).
[41] Y. Gao, et al., Early detection of myocardial fibrosis in cardiomyopathy in the absence of late enhancement: role of T1 mapping and extracellular volume analysis, Eur. Radiol. (2022).
[42] S. Nakamori, et al., Increased myocardial native T(1) relaxation time in patients with nonischemic dilated cardiomyopathy with complex ventricular arrhythmia, J. Magn. Reson. Imaging 47 (3) (2018) 779–786.