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ORIGINAL ARTICLE

Potential Value of Native T1 Mapping in Symptomatic Adults with Congenital Heart Disease: A Preliminary Study of 3.0 Tesla Cardiac Magnetic Resonance Imaging

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Abstract

The native T1 value at 3.0 Tesla is a sensitive marker of diffuse myocardial damage. We evaluated the clinical usefulness of native T1 mapping in symptomatic adults with congenital heart disease (CHD), particularly in the systemic right ventricle (RV). Prospectively, 45 consecutive symptomatic adults with CHD were enrolled: 20 with systemic RV and 25 with tetralogy of Fallot underwent cardiac magnetic resonance (CMR) imaging at 3.0 Tesla. The Modified Look-Locker Inversion recovery sequence was used for T1 mapping. Cardiovascular events in the systemic RV were defined as heart failure and tachyarrhythmia. Brain natriuretic peptide (BNP) and indexed systemic ventricular end-diastolic volume were significantly higher in the systemic RV group. The native T1 value and extracellular volume (ECV) of the septal and lateral walls were higher in the systemic RV group, suggesting high impairment of the myocardium in the systemic RV group. There was a strong correlation between the native T1 value and ECV of the septum ($r=0.58$, $P=0.03$) and lateral wall ($r=0.56$, $P=0.046$) in the systemic RV group. Seven patients with systemic RV had cardiovascular events. In univariate logistic regression analysis, BNP and native T1 values of the insertion point were important for predicting cardiovascular events. The native T1 value at 3.0 Tesla may be a sensitive, contrast-free, and non-invasive adjunct marker of myocardial damage in CHD and predictive of cardiovascular events in the systemic RV.

Keywords T1 mapping · Native T1 · Congenital heart disease · Systemic right ventricle

Abbreviations

CHD	Congenital heart disease
LGE	Late gadolinium enhancement
CMR	Cardiac magnetic resonance
BNP	Brain natriuretic peptide
ECV	Extracellular volume
HCM	Hypertrophic cardiomyopathy
DCM	Dilated cardiomyopathy
RV	Right ventricle
CCTGA	Congenitally collected transposition of the great arteries
TGA	Transposition of the great arteries
TOF	Tetralogy of Fallot
ROI	Region of interest
LV	Left ventricle
EDV	End-diastolic volume
EF	Ejection fraction
PAH	Pulmonary arterial hypertension

Introduction

T1 mapping techniques in cardiac magnetic resonance (CMR) imaging allow quantitative tissue characterization in the myocardium, adding new information to that provided by T1-weighted techniques such as late gadolinium enhancement (LGE) imaging. For focal fibrosis, LGE provides excellent delineation of lesions with some means of quantifying their volume [1]; however, LGE does not give a T1 estimate and may not be able to identify widely distributed or diffuse

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myocardial diseases. These are the obvious limitations of LGE techniques. Mapping of the longitudinal relaxation time (T1) and extracellular volume (ECV) offers a means of identifying and quantifying pathological changes in myocardial tissue, including diffuse changes that may be invisible to LGE [1–3]. Use of myocardial T1 mapping is now widespread in various cardiomyopathies and infiltrative diseases [3–5]. In other words, myocardial T1 mapping is a non-invasive modality to assess ECV for estimating diffuse myocardial fibrosis, by measuring myocardial and blood T1 relaxation time before and after contrast enhancement.

Several T1 mapping studies in congenital heart disease (CHD) gave the highest priority to the assessment of ECV; however, adults with CHD sometimes have chronic renal failure, and the contrast medium administration is contraindicated. Furthermore, flow measurements are often very complex and time-consuming; therefore, not all patients can tolerate the large amount of time required for scanning with gadolinium. As an alternative, native T1 values may be a sensitive, contrast-free, and non-invasive CMR marker of myocardial damage. Recently, some studies in non-CHD patients reported that native myocardial T1 values of 3.0 Tesla CMR imaging provide better discrimination between normal and diffusely diseased myocardium, including hypertrophied cardiomyopathy (HCM) and dilated cardiomyopathy (DCM) [3–5]. It is important to detect potential myocardial impairment in systemic right ventricle (RV) impairment, such as congenitally collected transposition of the great arteries (CCTGA), transposition of the great arteries (TGA), and single ventricle. The reason is that patients with systemic RV have anatomically vulnerable myocardia that are intolerant of systemic pressure for long periods of time. To optimize the timing of interventions, including surgical valve replacement and/or cardiac resynchronization therapy, visualization of ventricular tissue deterioration may be clinically useful.

In this study, we focused on native myocardial T1 values at 3.0 Tesla in symptomatic adults with CHD, particularly in the systemic RV, and we evaluated the potential value of using this method for detecting myocardial damage.

Materials and Methods

We prospectively enrolled 45 consecutive symptomatic adult CHD patients who were in New York Heart Association functional class ≥ 2: 20 consecutive adult patients with systemic RV (such as TGA and CCTGA) and 25 consecutive adult patients with tetralogy of Fallot (TOF) who had undergone intracardiac repair. Each patient underwent CMR imaging at Tokyo Women's Medical University between March 2018 and March 2019. Cardiovascular events in the systemic RV were defined as heart failure requiring hospitalization, tachyarrhythmias requiring medication, and ablation and/or implantation of a cardiac defibrillator within the previous 2 years. A blood test was performed within 3 months, and catheterization was performed within 2 years of CMR imaging. Twenty-five age-matched, healthy adults were also enrolled as a control group. These controls underwent CMR imaging for screening of palpitations and non-specific chest pain and showed normal results on all tests including electrocardiography, brain natriuretic peptide (BNP), transthoracic echocardiography, CMR and/or coronary magnetic resonance, and/or computed tomography angiography. No controls had hypertension, diabetes mellitus, or ischemic heart disease.

All participants underwent CMR imaging with a 3.0-Tesla whole-body imager (Ingenia 3 T; Philips Healthcare, Best, the Netherlands) equipped with dual-source, parallel radiofrequency transmission, and 32-element cardiac phased-array coils for radiofrequency reception. The Modified Look-Locker Inversion recovery sequence was used for T1 mapping. Mid-ventricular short-axis slices were scanned for myocardial T1 values before and after contrast imaging. Other scan parameters were as follows: field of view: 360 mm, matrix size: 128 × 256, SENSE factor: 2, repetition time: 2.7 ms, echo time: 1.26 ms, slice thickness: 8 mm, flip angle: 10°, TFE factors: 33, and shot mode: single-shot. Post-contrast imaging was performed approximately 12–15 min following administration of 0.1 mmol/kg gadoteric meglumine (Magnescope; Guerbet, Tokyo, Japan). Hematocrit was determined from a venous blood sample following CMR imaging. An elliptical region of interest (ROI) was drawn in the septal free walls and insertion points of the left ventricle (LV) and the RV on mid-ventricular short-axis images, avoiding regions with artifacts and ensuring that there was neither pooled blood nor epicardial fat in the ROI (Fig. 1). ECV was calculated based on pre- and post-contrast T1 values and hematocrit, as proposed by Flett et al. [6].

The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki. A priori approval was received from the institution's human research committee, and the ethical committee of our hospital approved our study. All patients provided informed consent for undergoing CMR imaging.

Statistics

Statistical analyses were performed using one-way analysis of variance. Continuous variables were assessed using Pearson's or Spearman's correlation coefficient. Logistic regression analysis was used to assess important parameters of cardiovascular events. Intra- and interobserver variabilities were assessed using intra- and interclass correlation coefficients, absolute values, and 95% confidence intervals.

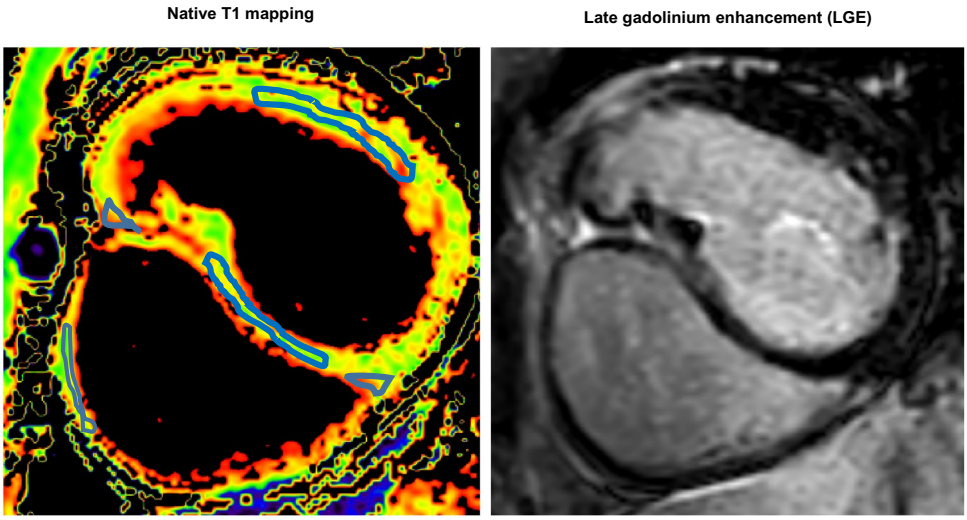

Fig. 1 Native T1 mapping and late gadolinium enhancement in adults with congenitally corrected transposition of the great arteries. A ellipse region of interest (ROI) was drawn in the septal, free walls, and insertion points of the LV and the RV at mid-ventricular short-axis images, avoiding regions with artifact and ensuring that neither blood pool nor epicardial fat in the ROI

Table 1 Patients' characteristics in adults with systemic RV, TOF, and controls

	Systemic RV 20 patients	TOF 25 patients	Controls 25 patients	3群	Systemic RV vs TOF
Age (years)	35.4 ± 8.3	36.3 ± 10.6	37.8 ± 11.7	NS	NS
Male	11 (55.0%)	10 (40.0%)	5 (50.0%)	NS	NS
Type of TOF repair	CCTGA 12 Unrepaired CCTGA Conventional Rastelli TGA Senning/Mustard 8	TAP 8 Rastelli/PVR 12 Resection/valvotomy 5	–	–	–
NYHA≧3	5 (25.0%)	3 (12.0%)	–	–	–
BNP (pq/ml)	102.0 ± 80.5	50.8 ± 41.2	–	–	< 0.05
SPO2	95.3 ± 5.1	98.3 ± 1.3	98.6 ± 1.9	NS	< 0.05
SBP (mmHg)	111.3 ± 22.8	111.8 ± 15.3	126.5 ± 20.9	NS	NS
CMR Systemic ventricular EDVI (ml/m^2)	111.4 ± 39.6	76.4 ± 21.1	76.5 ± 13.5	< 0.001	< 0.001
Systemic ventricular EF (%)	44.1 ± 11.3	49.6 ± 10.6	57.8 ± 6.9	< 0.001	NS
Pulmonary ventricular EDVI (ml/m^2)	78.6 ± 27.5	142.8 ± 44.6	83.9 ± 14.6	< 0.001	< 0.05
Pulmonary ventricular EF (%)	55.4 ± 11.8	49.8 ± 8.3	50.3 ± 10.1	NS	NS
Cath Systemic ventricular EDP (mmHg)	11.5 ± 4.4	10.8 ± 3.9	–	–	NS
Pulmonary ventricular EDP (mmHg)	10.1 ± 3.8	9.0 ± 3.5	–	–	NS
CI (L/min/m^2)	2.6 ± 0.8	3.0 ± 0.7	–	–	NS
mPA (mmHg)	17.0 ± 5.7	16.4 ± 7.1	–	–	NS

RV right ventricle, *TOF* tetralogy of Fallot, *TAP* transannular patch, *CCTGA* congenitally corrected transposition of the great arteries, *TGA* transposition of the great arteries, *TAP* transannular patch, *PVR* pulmonary vascular replacement, *NYHA* New York heart association, *BNP* brain natriuretic peptide, *SPO2* peripheral capillary oxygen saturation, *SBP* systolic blood pressure, *CMR* cardiac magnetic resonance, *EDVI* indexed end-diastolic volume, *EF* ejection fraction, *LGE* late gadolinium enhancement, *EDP* end-diastolic pressure, *CI*: cardiac index, *mPA* main PA pressure

Results

Basic Characteristics and CMR Imaging Data (Table 1)

BNP and indexed systemic ventricular end-diastolic volume (EDV) were significantly higher in the systemic RV group than in the TOF group. Peripheral capillary oxygen saturation and indexed pulmonary ventricular EDV were significantly lower in the systemic RV group than in the TOF group. There was no significant difference in age, blood pressure, systemic or pulmonary ejection fraction (EF), end-diastolic pressure, cardiac index, or mean pulmonary arterial pressure between the systemic RV and TOF groups.

T1 Mapping Data at 3.0 Tesla (Table 2) (Fig. 2)

Systemic ventricular native T1 values in the systemic RV group, pulmonary ventricular native T1 values in the TOF groups, and insertion point native T1 values were prolonged compared to those in the control group. Systemic and pulmonary ventricular ECV in the systemic RV and TOF groups were higher than those in the control group. Septal and lateral native T1 and ECV in the systemic RV group were higher than those in TOF group. There was a strong correlation between native T1 values and ECV of the septum ($r=0.58$, $P=0.03$) and lateral wall ($r=0.56$, $P=0.046$) in the systemic RV group.

Cardiovascular Events in Symptomatic Patients with Systemic RV (Table 3)

Seven patients with systemic RV had cardiovascular events: 4 patients had heart failure and 5 had atrial arrhythmias (tachycardia/atrial flutter/atrial fibrillation, overlapped) within the previous 2 years. There were no episodes of sustained ventricular tachycardia or death. In univariate logistic regression analysis, BNP and native T1 values of the insertion point were important parameters predicting cardiovascular events. Age, lower RV EF, and septal native T1 values also showed the tendency of important markers ($P=0.06$, 0.08, and 0.06, respectively) but were not significant statistically.

Reproducibility

The intraclass correlation coefficients for the interventricular septum, RV insertion points, and LV free wall T1 values were 0.82, 0.79, and 0.80, respectively. The interclass correlation coefficients for the septum, RV insertion points, and LV free wall T1 values were 0.80, 0.76, and 0.75, respectively.

Table 2 T1 mapping data

	Systemic RV 20 patients	TOF 25 patients	Control 25 patients	3群	Systemic RV vs TOF
Systemic ventricular septal native T1 (ms)	1319.3 ± 71.9	1242.2 ± 92.3	1198.1 ± 62.2	< 0.001	< 0.05
Systemic ventricular lateral native T1 (ms)	1315.6 ± 80.2	1201.9 ± 80.9	1190.0 ± 76.1	< 0.001	< 0.05
Pulmonary ventricular native T1 (ms)	1334.8 ± 103.8	1332.6 ± 123.8	1224.5 ± 72.2	< 0.05	NS
Insertion point native T1 (ms)	1350.9 ± 78.1	1272.6 ± 84.3	1227.6 ± 45.3	< 0.001	< 0.05
Systemic ventricular septal ECV (%)	29.6 ± 2.5	26.8 ± 4.8	21.7 ± 3.7	< 0.001	< 0.05
Systemic ventricular lateral ECV (%)	29.8 ± 1.9	25.4 ± 3.7	21.8 ± 3.8	< 0.001	< 0.05
Pulmonary ventricular ECV (%)	32.4 ± 5.1	33.5 ± 8.5	27.4 ± 1.8	< 0.001	NS

ECV extracellular volume

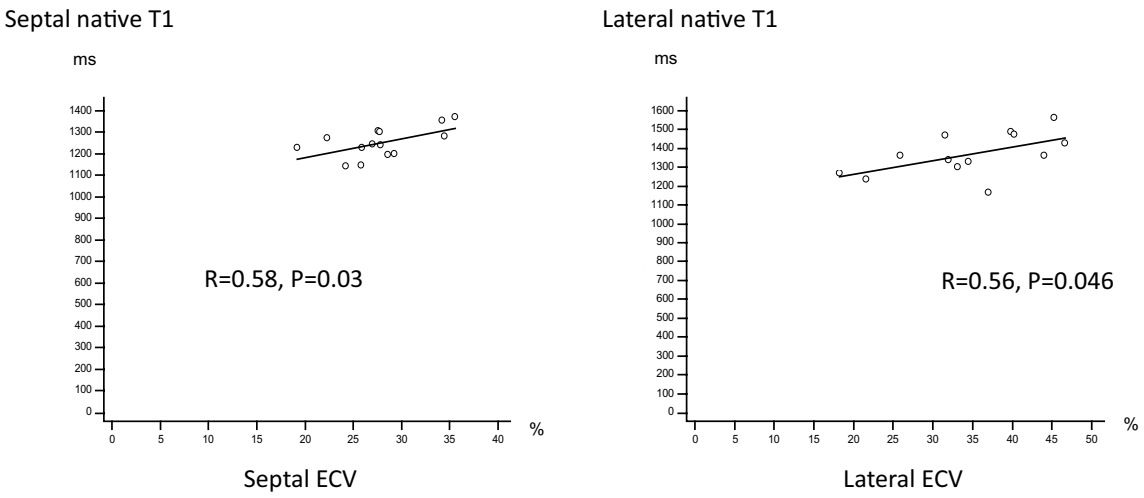

Fig. 2 Correlation between native T1 value and ECV in systemic RV. There was a good correlation between native T1 value and ECV of the septum ($r=0.58$, $P=0.03$) and of the lateral wall ($r=0.56$, $P=0.046$) in the systemic RV

Table 3 Univariate logistic regression analysis of cardiovascular events in adults with systemic RV

	Odds ratio	95% CI	*P* value
Age (years)	1.20	0.99–1.44	0.06
BNP (pq/ml)	1.03	1.005–1.05	0.02
RVEDVI (ml/m^2)	1.03	0.99–1.06	0.09
RVEF (%)	0.89	0.78–1.01	0.08
Insertion point native T1 (ms)	1.03	1.001–1.07	0.043
Septal native T1 (ms)	1.05	0.99–1.09	0.06
Systemic ventricular native T1 (ms)	1.01	0.99–1.03	0.12
Pulmonary ventricular native T1 (ms)	1.006	0.99–1.02	0.29

RV right ventricle, *BNP* brain natriuretic peptide, *EDVI* indexed end-diastolic volume, *EF* ejection fraction, *LGE* late gadolinium enhancement

Discussions

This is a preliminary study of native T1 mapping and its potential clinical usefulness in adults with CHD. Our results showed that (1) the septal and lateral native T1 values and ECVs were higher in the systemic RV group than in the TOF group (2) there was a strong correlation between native T1 values and ECV in the systemic RV group, and (3) native T1 values of the insertion point, as well as BNP, were relevant predictive factors for cardiovascular events in the systemic RV group using 3.0 Tesla CMR imaging.

Native T1 Values in CHD and the Systemic RV in Particular

Generally, myocardial damage in CHD can be caused by volume and/or pressure overload, prolonged cyanosis before intracardiac repair in the childhood, and longer cross-clamp times during surgery [7–10]. Systemic ventricular myocardium deterioration is common in middle-aged adults with systemic RV [11]. Systemic RV function with significant tricuspid regurgitation becomes more easily deteriorated; however, it is difficult to assess genuine ventricular damage only by EF using echocardiography or CMR cine images because of Frank-Starling's law [12]. Therefore, additional parameters are needed to evaluate real myocardial damage proactively.

Before evaluating T1 map, we should know the following details. Normal myocardium is composed of three major compartments: intracellular, intravascular, and interstitial [2]. The intracellular compartment is the largest of the three in normal myocardium and consists predominantly of myocytes, but also includes fibroblasts, endothelial cells, and smooth muscle cells. The interstitial and intravascular compartments are often referred to as the ECV. In the majority of cardiovascular diseases such as extracellular edema, interstitial replacement, or infiltrative fibrosis, the ECV becomes expanded, primarily due to expansion of the interstitial component [2, 3]. Native T1 mapping refers to the acquisition of T1 maps without the use of contrast agents. In edema and other fibrotic conditions, expansion of the extracellular space results in an increase of native T1 values as well.

Myocardial fibrosis is not considered rare in the systemic RV [13, 14] and is associated with higher BNP [15]. Elevation of ECV was reported to be associated with adverse clinical outcomes as well [15]. An increased native T1 value and/or ECV reflect the proportionally larger extracellular matrix component subsequent to cardiomyocytes atrophy and/or death [3, 9, 16]. If a contrast-free CMR imaging marker was available, it would be more convenient. Our result showed a strong correlation between native T1 values and ECV; therefore, native T1 values may be an alternative marker of potential myocardial damage in this population as reported in the population with HCM or DCM [4, 5]. In adults with CHD with low pre-test possibility for myocardial impairment or for those in whom contrast administration is contraindicated, native T1 values may serve as an effective screening test.

Some studies also reported that native T1 values and ECV for 1.5 Tesla CMR imaging were higher in the systemic RV than in TOF [15]; therefore, our results were reasonable. A profibrotic state through genetically driven collagen metabolism precedes the overt phenotype with LV hypertrophy or fibrosis visible on LGE in cardiomyopathy [16]; therefore, native T1 values may be adjunct but can be clinically informative. Contrast-free markers of adverse cardiac remodeling may be able to predict therapy response and/or guide treatment strategies. It has been reported that TOF after PVR may improve native T1 value and/or ECV compared to that before PVR; therefore, there is a possibility that native T1 value and/or ECV can be useful parameters of reversible myocardial damage. These parameters may be informative when we decide the optimal timing of tricuspid valve replacement in the systemic RV before irreversible myocardial damage occurs.

Why was the Native T1 Value of the Insertion Points Relevant?

LGE and/or elevated native T1 values of the insertion points are often identified in patients with HCM and pulmonary arterial hypertension (PAH) [17–19]. These findings are considered to represent focal plexiform fibrosis associated with myocardial disarray and increased connective tissue density rather than replacement fibrosis [19–22]. On the other hand, some studies have reported the prognostic significance of them in PAH and HCM [20–22].

In the systemic RV, ventricles become hypertrophied and become a similar shape to HCM. This may be one of the reasons why patients with systemic RV often show elevated native T1 values of the RV insertion point. Another possibility is that there is secondary pulmonary hypertension associated with left-sided heart failure. Not only patients with genuine PAH but also those with secondary PAH show elevated native T1 values of the insertion point [23]. Few studies have focused on native T1 values of the insertion point as well as septum and/or free wall in CHD, so further studies are required.

The Possibility of 3.0 Tesla CMR Imaging in Adults with CHD

Previous CHD studies at 1.5 Tesla mainly focused on ECV [24] rather than native T1 values because there appeared to be no statistical significance in native T1 values among various CHDs. This may be partially due to the technical difference/limitation between 1.5 and 3.0 Tesla. Native T1 values at 1.5 Tesla are reported to underestimate myocardial infarction compared to those at 3.0 Tesla [25]; therefore, native T1 images at 3.0 Tesla in adults with CHD may be more sensitive, but further studies in a large cohort are needed to confirm that these findings are clinically useful.

Limitations

The limitations of this study are the very small population size and the lack of a cox-hazard analysis due to the short follow-up period; therefore, the prognostic values of native T1 mapping remain unknown. This is a preliminary study of native T1 mapping at 3.0 Tesla in adults with CHD, but we believe that this study is clinically informative. Furthermore, the native T1 values of the insertion points were found to be less reproducible than those of the interventricular septum. This may be due to higher T1 heterogeneity in this region leading to greater T1 variation when ROI placement differs.

Conclusions

The native T1 value at 3.0 Tesla may be a sensitive, contrast-free, and non-invasive adjunct marker of myocardial damage in CHD and may be predictive of cardiovascular events in the systemic RV.

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Compliance with Ethical Standards

Conflict of interest The authors of this manuscript declare no relationships with any companies, whose products or services may be related to the subject matter of the article.

Ethical Approval Institutional Review Board approval was obtained. Study subjects or cohorts overlap: No study subjects or cohorts have been previously reported. Methodology: Prospective diagnostic or prognostic study performed at one institution.

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