Left Ventricular Myocardial T1 Values and Extracellular Volume Fraction in Asymptomatic Subjects: Variations According to Left Ventricular Segments and Correlation with Cardiovascular Risk Factors

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Research Article

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Abstract

Purpose: To evaluate the normal range and variation in pre-contrast (preT1) and post-contrast (postT1) myocardial T1 values and extracellular volume fraction (ECV) according to left ventricular (LV) segments and to check for correlations between them and known cardiovascular risk factors.

Methods: This study included 233 asymptomatic subjects (210 men and 23 women; aged 54.1±6.0 years) who underwent cardiac magnetic resonance imaging with preT1 and postT1 mapping on a 1.5-T scanner. T1 values and ECVs were compared among LV segments, age groups, and sex, and correlated with renal function. Based on the presence of hypertension (HTN) and diabetes mellitus (DM), the subjects were subdivided into the control (n=121), HTN (n=58), DM (n=25), and HTN and DM (HTN-DM) groups (n=29).

Results: T1 values and ECV showed significant differences between the basal septal and lateral segments ($p \leq 0.001$) and between the mid-septal and mid-lateral segments (PreT1 $p \leq 0.003$, postT1 and ECV $p<0.001$). Among subgroups according to the HTN and DM status, the HTN-DM group showed a significantly higher ECV (0.260±0.023) than the control (0.240±0.021, p=0.011) and HTN (0.241±0.024, p=0.041) groups. Overall postT1 and ECV of the LV had significant correlation with the estimated glomerular filtration rate (r = 0.19, p=0.038 for postT1; r = -0.23, p=0.011 for ECV).

Conclusion: Septal segments show higher preT1 and ECV but lower postT1 than lateral segments at the mid-ventricular and basal levels. ECV is significantly affected by cardiovascular risk factors such as HTN, DM, and decreased renal function, even in asymptomatic subjects.

Introduction

Post-contrast T1 mapping using the modified look-locker inversion recovery (MOLLI) sequence is a promising method for assessing myocardial pathologic conditions. Reports have demonstrated that native T1 values and extracellular volume expansion on cardiac magnetic resonance imaging (CMR) correlate closely with the histological quantification of myocardial fibrosis [1–5]. The extracellular volume fraction (ECV) is calculated from a combination of native and post-contrast measurements and reflects the volume fraction of extracellular space on a region-of-interest (ROI) basis. Thus, native T1 and extracellular volume are sensitive to different compartments within the myocardium and may reflect different aspects of pathological changes in cardiac diseases [6].

Recent reports have described a possible correlation between myocardial fibrosis and cardiovascular risk factors, such as chronic renal disease, hypertension (HTN), and diabetes mellitus (DM) [8, 9]. To date, normal T1 values and ECV and their regional difference have been documented in a relatively small population [10]. However, they have not been evaluated in a segmental manner, which could serve as reference values to evaluate localized myocardial abnormalities. According to a meta-analysis, pooled means of T1 values and ECV in normal subjects showed almost the same ECV and heterogeneous T1 values and HTN yielded native T1 values and ECV similar to normal subjects [11].

Therefore, this study aimed to report the normal range and variation in pre-contrast and post-contrast myocardial T1 values (preT1 and postT1, respectively) and ECV according to left ventricular (LV) segments and evaluate any correlation between T1 values and known cardiovascular risk factors, such as HTN, DM, and decreased renal function in asymptomatic subjects.

Materials And Methods

This retrospective study was approved by the Institutional Review Board, and the need for informed consent was waived. The authors who have no relationships with the industry controlled the inclusion of all data and information in this article. We evaluated the records of consecutive asymptomatic subjects who underwent CMR for health screening at the Health Promotion Center of our institution between March 2014 and October 2015. We excluded subjects with one or more following criteria; poor imaging quality (n = 36) or cooperation (n = 40), lack of optimal T1 mapping (n = 22), and previous history of myocardial infarction (n = 5), localized abnormality in T1 maps and late gadolinium enhancement imaging suspicious for myocardial infarction or cardiomyopathy (n = 12) LV hypertrophy defined as LV wall thickness >13 mm at least 1 segment or indexed end-diastolic mass >91 g/m^2 in males and >77 g/m^2 in females (n = 9) [12, 13]. A total of 233 subjects were included (Figure 1).

CMR protocol

All patients underwent CMR using a 1.5-T MR system (Magnetom Avanto, Siemens Healthineers) with a 32-channel phased-array receiver coil during repeated breath-holds. After localization, cine images of the LV mass were acquired using a steady-state free precession (SSFP) sequence with 8–10 contiguous short-axis slices to cover the entire LV with a slice thickness of 6 mm and a 4-mm gap. Standard delayed gadolinium-enhanced imaging was acquired using the phase-sensitive inversion recovery (PSIR) technique after injecting 0.15 mmol/kg gadobutrol (Gadovist; Bayer Healthcare) in 10–12 continuous short-axis images of 6 mm thickness with a 4-mm slice gap. Inversion delay times were typically 280–360 ms. Three short-axis images were acquired using the MOLLI sequence before and 15 min after administering gadolinium (Figure 2).

Typical imaging parameters for MOLLI were as follows: two non-selective inversion pulses; number of acquisitions after each inversion pulse, 5, 3; number of recoveries before the second inversion pulse, 3; SSFP single-shot readout in mid-diastolic phase; field of view, 350 × 300 mm^2; acquisition matrix, 192 × 150; slice thickness, 8 mm; flip angle, 35°; repetition time, 2.43 ms; echo time, 1.01 ms; bandwidth, 1085 Hz/pixel; minimum inversion time, 120 ms; inversion time increment, 80 ms; generalized autocalibrating partially parallel acquisition factor, 2.

T1 mapping post-processing

The T1-maps were generated automatically by the scanner after the acquisition with MOLLI. To correct for residual cardiac and respiratory motion between individual inversion-recovery images, a non-rigid

motion correction algorithm [14] was used, and T1 values were calculated on a pixel-wise basis by performing a PSIR non-linear curve fitting using the three-parameter signal model.

Image analysis

Image acquisition and analysis were done in a manner consistent with the Society for Cardiovascular Magnetic Resonance and CMR Working Group of the European Society of Cardiology consensus statement [5]. Image analysis was performed by two experienced cardiac radiologists (CMR experience of 7 years and 12 years, respectively) blinded to the clinical information. ROIs were drawn on 16 LV segments and blood pools (Figure 2) manually. ROI drawing excluded any areas of late gadolinium enhancement (LGE) consistent with LGE at the right ventricular insertion point. All original images were assessed for artifacts due to susceptibility and cardiac or respiratory motion. Each motion-corrected series was evaluated for correct image alignment and each map was evaluated as to whether the original images were transformed to an acceptable map [15].

ECV calculation

ECV was calculated using the following equations:

$\Delta R1$ = 1/T1 time post-contrast - 1/T1 time pre-contrast,

$\lambda = \Delta R1_{myocardium} / \Delta R1_{blood}$,

$ECV = \lambda * (1 - Hct)$.

Hct refers to the hematocrit measured in venous blood sampled on the same day of the MRI scan acquisition.[5, 16, 17].

Clinical information

The clinical information of the subjects was obtained from medical records and laboratory findings on the day of CMR acquisition. Body mass index (BMI) was calculated as weight (kg) divided by the square of the height (m^2). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured after the subject had rested for at least 5 min. Blood samples were collected from the antecubital vein after overnight fasting. Total cholesterol, high-density lipoprotein, triglyceride (TG), fasting plasma glucose (FPG), and serum creatinine levels were measured using enzymatic or colorimetric methods. The estimated glomerular filtration rate (eGFR) was calculated using a formula from the Modification of Diet in Renal Disease study: eGFR (mL/min/1.73 m^2) = 186.3 × (Cr) - 1.154 × (age) - 0.203 × (0.742 for women) (11). Information on the presence of HTN, type 2 DM, smoking, and alcohol consumption was obtained from interviews. Body surface area (BSA) was calculated as follows: 0.20247 × height (m)$^{0.725}$ × weight (kg)$^{0.425}$ (Dubois formula). HTN was diagnosed when a person's SBP in the office or clinic was ≥140 mmHg and/or their DBP was ≥90 mmHg following repeated examinations [18]. DM was diagnosed using the following criteria: occasional plasma glucose value of ≥200 mg/dL (≥11.1 mmol/L), FPG of ≥126 mg/dL (7.0 mmol/L) (fasting time 8–12 h), oral glucose tolerance test 2-h value in venous plasma

≥200 mg/dL (≥11.1 mmol/L), glycosylated hemoglobin (HbA1c) ≥6.5% (≥48 mmol/mol Hb), and impaired fasting glucose for the fasting glucose range of 100–125 mg/dL (5.6 mmol/L- 6.9 mmol/L) in venous plasma [19].

T1 values and ECV were compared among age groups and between sexes. According to their status of cardiovascular risk factors, subjects were divided into four subgroups: 1) HTN group, 2) DM group, 3) HTN and DM (HTN-DM) group, and 4) control group. The control group was defined as subjects who did not have HTN and DM. We evaluated whether there were any differences in T1 values and ECV between the subgroups.

Statistical analysis

All analyses were conducted using SPSS 26 (IBM, Chicago, United states) and Rex (Version 3.0.3, RexSoft Inc., Seoul, Korea). Categorical variables were reported as percentages and continuous variables are presented as means with standard deviations. The differences between continuous variables were analyzed using the independent or paired t-test, Wilcoxon rank sum test and the Mann Whitney U-test was used to analyze the differences between non-continuous variables. According to normality of distribution, analysis of variance or Kruskal-Wallis test was applied for multiple group comparison. The relationships between T1 values and cardiovascular risk factors were studied using simple correlation analysis with Pearson correlation coefficient (r). Inter-observer agreement was quantified using intraclass correlation coefficients (ICCs) for all subjects. The threshold for statistical significance was set at $p = 0.05$ in the two-tailed tests.

Results

The baseline characteristics and CMR LV measurements of the subjects and subgroups divided by their status of cardiovascular risk factors are shown in Table 1. The age of total subjects ranges from 40 to 80 years. Except for BMI, blood pressure, and HbA1c, there was no statistically significant difference between subgroups with respect to each characteristic. The HTN and HTN-DM groups showed higher BMI, SBP, and DBP, and the DM and HTN-DM groups demonstrated higher HbA1c than the rest subgroups ($p < 0.05$).

Table 1
Baseline characteristics and cardiac magnetic resonance measurements of study population

Subgroup	Control group (n=121, 51.9%)	HTN (n=58, 24.9%)	DM (n=25, 10.7%)	HTN-DM (n=29, 12.4%)	p-value	Total (n=233)
Baseline characteristics						
Age (years)	53.2 ± 4.98	55.8 ± 7.56	54.5 ± 5.56	54.5 ± 6.16	0.051	54.1 ± 5.97
Male	108 (87.8%)	53 (93.0%)	22 (91.7%)	27 (93.1%)	0.111	210 (90.1%)
BSA (m^2)	1.83 ± 0.13	1.83 ± 0.15	1.79 ± 0.12	1.90 ± 0.17	0.076	1.83 ± 0.14
BMI (kg/m^2)	24.3 ± 2.11	25.4 ± 2.53*‡	23.9 ± 2.54	26.7 ± 3.40*‡	<0.001	24.8 ± 2.59
Heart rate (bpm)	66.0 ± 10.4	69.0 ± 12	68.5 ± 10.4	67.7 ± 11.3	0.330	67.2 ± 11.0
SBP (mmHg)	114 ± 14.3	125 ± 17.3*‡	114.6 ± 13.0	122 ± 17.4*‡	<0.001	117 ± 16.0
DBP (mmHg)	76.0 ± 9.32	81.9 ± 10.2*‡	73.6 ± 9.41	79.0 ± 8.05‡	<0.001	77.6 ± 9.76
Hematocrit (%)	44.1 ± 3.19	44.7 ± 2.99	44.9 ± 2.78	44.3 ± 2.73	0.773	44.3 ± 3.04
Hb (g/L)	15.0 ± 1.19	15.2 ± 1.12	15.3 ± 1.22	15.1 ± 1.06	0.656	15.1 ± 1.16
Hb A1c (%)	5.42 ± 0.22	5.30 ± 0.40	6.42 ± 0.53*†	6.21 ± 0.48*†	<0.001	5.60 ± 0.36

Abbreviations: BMI, body mass index; CI, cardiac index; DM, diabetes mellitus; DBP, diastolic blood pressure; ED MASSi, indexed end-diastolic myocardial mass; EDVi, indexed end-diastolic volume; EF, ejection fraction; ESVi, indexed end-systolic volume; GFR, glomerular filtration rate; Hb, hemoglobin; HTN, hypertension; SBP, systolic blood pressure; SVi, indexed stroke volume.

Continuous values are presented as mean ± standard deviation.

The proportion of a numeric value is described in parenthesis.

* p <0.05 versus control group

† p <0.05 versus HTN group

‡ p <0.05 versus DM group

Subgroup	Control group (n=121, 51.9%)	HTN (n=58, 24.9%)	DM (n=25, 10.7%)	HTN-DM (n=29, 12.4%)	p-value	Total (n=233)
GFR (mL/min/1.73 m^2)	83.5 ± 11.3	81.8 ± 15.6	83.6 ± 14.4	85.0 ± 10.2	0.720	83.3 ± 12.7
Hyperlipidemia	70 (57.9%)	37 (63.8%)	11 (44.0%)	19 (65.5%)	0.269	137 (58.8%)
Hypertension	0	58 (100%)	0	29 (100%)		87 (37.3%)
Diabetes	0	0	25 (100%)	29 (100%)		54 (23.2%)
Left ventricular measurements						
EF (%)	65.75 ± 4.81	65.70 ± 5.79	65.25 ± 6.52	67.78 ± 6.07	0.378	65.9 ± 5.43
EDVi (mL/m^2)	70.01 ± 10.22	67.79 ± 11.00	68.36 ± 8.24	67.68 ± 8.02	0.439	69.0 ± 9.99
ESVi (mL/m^2)	24.16 ± 5.74	23.43 ± 6.25	23.90 ± 6.09	22.10 ± 5.85	0.391	23.7 ± 5.92
SVi (mL/m^2)	59.21 ± 10.48	62.70 ± 11.78	54.82 ± 11.08	55.17 ± 10.98	0.097	59.1 ±11.2
CI (L/min/ m^2)	3.01 ± 0.44	3.01 ± 0.50	3.06 ± 0.28	3.22 ± 0.46	0.167	3.04 ± 0.45
ED MASSi (g/m^2)	61.35 ± 10.89	65.45 ± 11.56	60.61 ± 9.16	63.24 ± 8.94	0.114	62.5 ± 10.8

Abbreviations: BMI, body mass index; CI, cardiac index; DM, diabetes mellitus; DBP, diastolic blood pressure; ED MASSi, indexed end-diastolic myocardial mass; EDVi, indexed end-diastolic volume; EF, ejection fraction; ESVi, indexed end-systolic volume; GFR, glomerular filtration rate; Hb, hemoglobin; HTN, hypertension; SBP, systolic blood pressure; SVi, indexed stroke volume.

Continuous values are presented as mean ± standard deviation.

The proportion of a numeric value is described in parenthesis.

* p <0.05 versus control group

† p <0.05 versus HTN group

‡ p <0.05 versus DM group

Segmental analysis of T1 values

The mean T1 values and ECV with standard deviations for all 16 American Heart Association segments are shown in Supplementary Table 1 and Figure 3. The overall ICC for each segmental T1 value was 0.82 (p <0.001). The ICC (0.64–0.80) for T1 value of each apical segment were significantly poorer than that (0.79–0.88) of each basal and mid-ventricular segment (p <0.001, respectively). PreT1 and postT1 values and ECV of global LV myocardium were 989 ± 41 ms, 454 ± 38 ms, and 0.245 ± 0.024, respectively.

PreT1 and ECV revealed significantly higher values in the septal wall than in the lateral wall at the basal and mid-ventricular levels of the LV myocardium (p <0.05, Table 2). However, postT1 values in the septal wall were significantly lower than those in the lateral wall at the basal and mid-ventricular levels of the LV myocardium (p <0.05, Table 2).

Table 2
Comparing T1 values and ECVs between the septal and lateral walls

	PreT1			PostT1			ECV		
	Septal	Lateral	p	Septal	Lateral	p	Septal	Lateral	p
Basal	992 ± 50	980 ± 52	0.001	458 ± 45	471 ± 40	<0.001	0.240 ± 0.031	0.225 ± 0.026	<0.001
Middle	998 ± 48	985 ± 61	0.003	454 ± 39	463 ± 40	<0.001	0.242 ± 0.028	0.236 ± 0.035	<0.001
Apical	989 ± 84	1011 ± 77	<0.001	433 ± 50	430 ± 44	0.150	0.270 ± 0.048	0.278 ± 0.047	0.005
Mean	985 ± 48	992 ± 49	0.014	448 ± 41	454 ± 38	<0.001	0.251 ± 0.029	0.246 ± 0.028	0.001

Abbreviations: ECV, extracellular volume fraction; PreT1, pre-contrast myocardial T1 value; PostT1, post-contrast myocardial T1 value

Values are presented as mean ± standard deviation.

Comparisons of T1 values and ECV for age and sex

PreT1 and postT1 values and ECV of global LV myocardium in male were 984 ± 40 ms, 457 ± 36 ms, and 0.241 ± 0.021, respectively. Those in female were 1029 ± 25 ms, 443 ± 51 ms, and 0.280 ± 0.026, respectively. PreT1 and ECV were significantly higher in women than in men in all LV segments (p <0.05, Figure 3, Supplementary Table 1), except for the apical inferior segment (p = 0.053). However, postT1 showed no significant difference between male and female subjects (p ≥0.061).

There was no statistically significant difference in T1 values and ECV of the four age groups divided into quantiles (group 1 ≤ 50 years, n = 68; 50 years < group 2 ≤ 53 years, n = 58; 54 years < group 3 ≤ 56 years, n = 51; group 4 > 56 years, n = 56) (994 ± 46, 987 ± 40, 990 ± 45, and 991 ± 33 for preT1, p = 0.77; 451 ± 33, 454 ± 34, 446 ± 43, and 449 ± 43 for postT1, p = 0.95; 0.243 ± 0.029, 0.245 ± 0.027, 0.246 ± 0.021, and 0.245 ± 0.030 for ECV, p = 0.41, respectively) and no significant correlation (p >0.46 for T1

values and ECV). In total population nor each sex group, there was no significant linear correlation between the age and T1 values or ECV (preT1, p = 0.58; postT1, p = 0.88; ECV, p = 0.45).

Relations between T1 values and cardiovascular risk factors

A dot scatter plot of ECV with error bars indicating two standard deviations from the average reveals that the HTN-DM group showed significantly higher ECV (0.260 ± 0.023) than the control group (0.240 ± 0.021, p = 0.011) and the HTN group (0.241 ± 0.024, p = 0.041) (Figure 4). There was a tendency for the ECV to increase among the subgroups in the following order: control, HTN, DM, and HTN-DM groups. BMI nor dyslipidemia did not affect T1 values and ECVs statistically in the total population and each subgroup. PostT1 and ECV of the LV in the control group showed significant correlations to eGFR (r = 0.19 p = 0.038 for postT1; r = -0.23, p = 0.011 for ECV; Figure 5). PreT1 demonstrated no significant correlation to eGFR (p = 0.100).

Discussion

In this study, we presented reference values for native T1 relaxation time and ECV according to LV segments in asymptomatic subjects using a 1.5 T MR system. We found segmental variation in the myocardial tissue composition using CMR T1 mapping in asymptomatic subjects. Females showed higher preT1 and ECV than males. The HTN-DM group showed a significantly higher ECV than the control and HTN groups.

Myocardial fibrosis is commonly associated with cardiac hypertrophy and failure and is associated with worsening ventricular systolic function, abnormal cardiac remodeling, and increased ventricular stiffness in animal models [20]. Although endocardial biopsy is the most specific procedure for measuring myocardial fibrosis, it is invasive, and its sensitivity is low due to sampling errors [21]. CMR T1 mapping may serve as a noninvasive biopsy and collagen volume fraction (CVF) was associated with preT1 and ECV in an animal model of myocardial fibrosis [22, 23].

In a pooled summary of 954 healthy subjects from the literature, 3T showed longer T1 than 1.5T with MOLLI variant sequences by approximately 100-200 ms. ECV of the healthy subjects was consistent among the studies (25-27%), irrespective of subject factors, sequences, vendors, and contrast types [11]. In a recent study, normal myocardial T1 reference ranges were reported as follows; at 3-T: 1129-1309 ms and at 1.5-T: 933-1020 ms (males) and 965-1054 ms (females), which tend to be similar to our findings [24].

Our finding included segmental variations in T1 values and ECV, especially between septal and lateral segments. Motion during the T1 map acquisition may induce a poor T1 model fit and falsely deviated T1 values and ECV, because T1 maps are generated from a sequential series of images [25]. Lateral wall is more vulnerable to motion artifacts which may be a potential cause of the lower preT1 and ECV and the higher postT1 in the lateral segments [10, 26, 27]. Considering low reproducibility of apical T1 values, it may be reasonable to measure T1 values in basal to middle septal walls [27].

Sex hormones may influence myocardial structure and function [28, 29]. Especially, a previous study demonstrated that estradiol attenuates myocardial hypertrophy [30]. Our observations of higher preT1 and ECV of females than those of males also suggest that sex hormones affect myocardial histology.

There is inconsistency in association of age with T1 values and ECV in the published studies [10, 26, 31–34]. Rauhalamm et al. (28) and Liu et al. (26) demonstrated a linear correlation between age and T1 values and ECV. According to Rosmini et al., native myocardial T1 decreased slightly with age, while ECV did not change with age (29). In our study, aging was not a significant factor affecting T1 values and ECV, which is consistent with a previous study by Dabir et al. (25).

Our results showed that there was no significant difference in ECV between the HTN and control groups. According to previous reports, T1 mapping revealed increased diffuse myocardial fibrosis in well-controlled hypertensive patients, but the increase was small and only occurred with LV hypertrophy (LVH) [35]. Our findings are consistent with the literature, since LVH was contained in the exclusion criteria of our study. Patients with HTN LVH had higher ECV and longer native T1 than HTN non-LVH and control subjects [6, 35]. According to Venkatesh et al., in a study of 1,813 subjects who underwent CMR, HTN-induced remodeling was related to enhanced replacement and diffuse fibrosis [36]. In an animal study, the ECV and native T1 value of the myocardium in 18 hypertensive swine increased over three months, even though no LGE was found at any of the imaging times [23].

According to Levelt et al., there were no significant differences in native myocardial T1 values and ECV between the patients with DM and the control subjects, indicating the absence of fibrosis in a similar context as our study [37]. Furthermore, our study showed HTN-DM group showed significantly high ECV than the control and HTN groups, which provides a clue about a synergetic effect on ventricular fibrosis of DM and HTN. DM appears to enhance fatty acid metabolism, decrease glucose oxidation, and change intracellular signaling, leading to cardiac structure and function alterations, based on prior research and our study [38–40]. Ventricular fibrosis is a common structural and histological hallmark of DM, primarily owing to the deposition of collagen and advanced glycation end-products resulting from altered intracellular signaling and metabolic disturbances [41]. In a study of 135 patients with DM, ECV was significantly different from that of control subjects, while native T1 was not significantly different [42]. ECV values correlated with HbA1c levels in DM [43]. The group with HbA1c $\geq$7.0 had a significantly higher ECV value than the control subjects and the lower HbA1c group (HbA1c <7.0). There was no statistically significant difference in the native T1 value and postT1 value among the three groups [43]. The lack of significance between the control and DM groups in ECV can be explained by Hb A1c below 7.2 in our research population.

In our study, in the control group, GFR significantly correlated not with preT1 linearly but with postT1 proportionally and ECV inversely. This interesting finding suggests that postT1 may be associated with the degrees of renal clearance of contrast agent. Increased renal clearance of contrast agent could facilitate washout of contrast agent in myocardium and then increase myocardial postT1 values [44, 45].

According to Edward et al., patients with chronic kidney disease had increased native T1 values and ECV compared with the control and hypertensive subjects [7].

The limitations of this study include the retrospective cross-sectional study design, the relatively small number of subjects with DM (n = 25), DM and HTN (n = 29), and female subjects (n = 23), and a relatively narrow range of age distribution concentrated in 50s resulting from the consecutive data collection. Low ICCs for measurement of apical segments are ascribed to small ROIs and image inhomogeneity associated with motion.

In conclusion, there were significant differences between the T1 values and ECV of the septal and lateral walls at the mid-ventricular and basal levels in asymptomatic subjects. Females had significantly higher preT1 and ECV values than males. ECV is significantly affected by cardiovascular risk factors such as HTN, DM, and decreased renal function, even before cardiovascular symptoms develop.

Declarations

Author contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Moon Young Kim, Soo Jin Cho, and Yeon Hyeon Choe. The first draft of the manuscript was written by Moon Young Kim and Yeon Hyeon Choe and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Figures

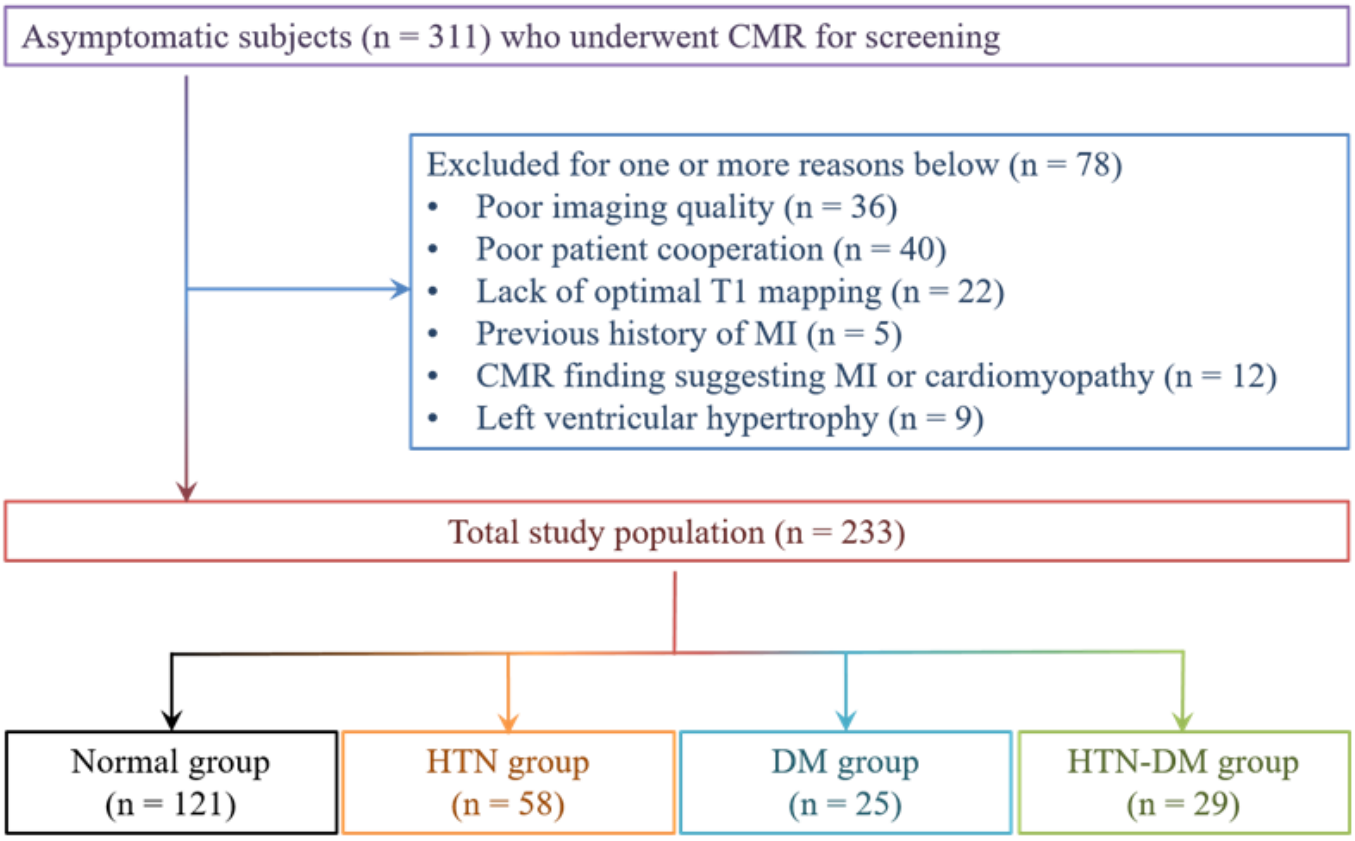

Figure 1

Flow chart of study population enrollment. The total study population was divided into four groups according to cardiovascular risk factors. The control group was defined as subjects who did not have hypertension (HTN) and diabetes mellitus (DM). Abbreviations: CMR, cardiac magnetic resonance imaging; MI, myocardial infarction.

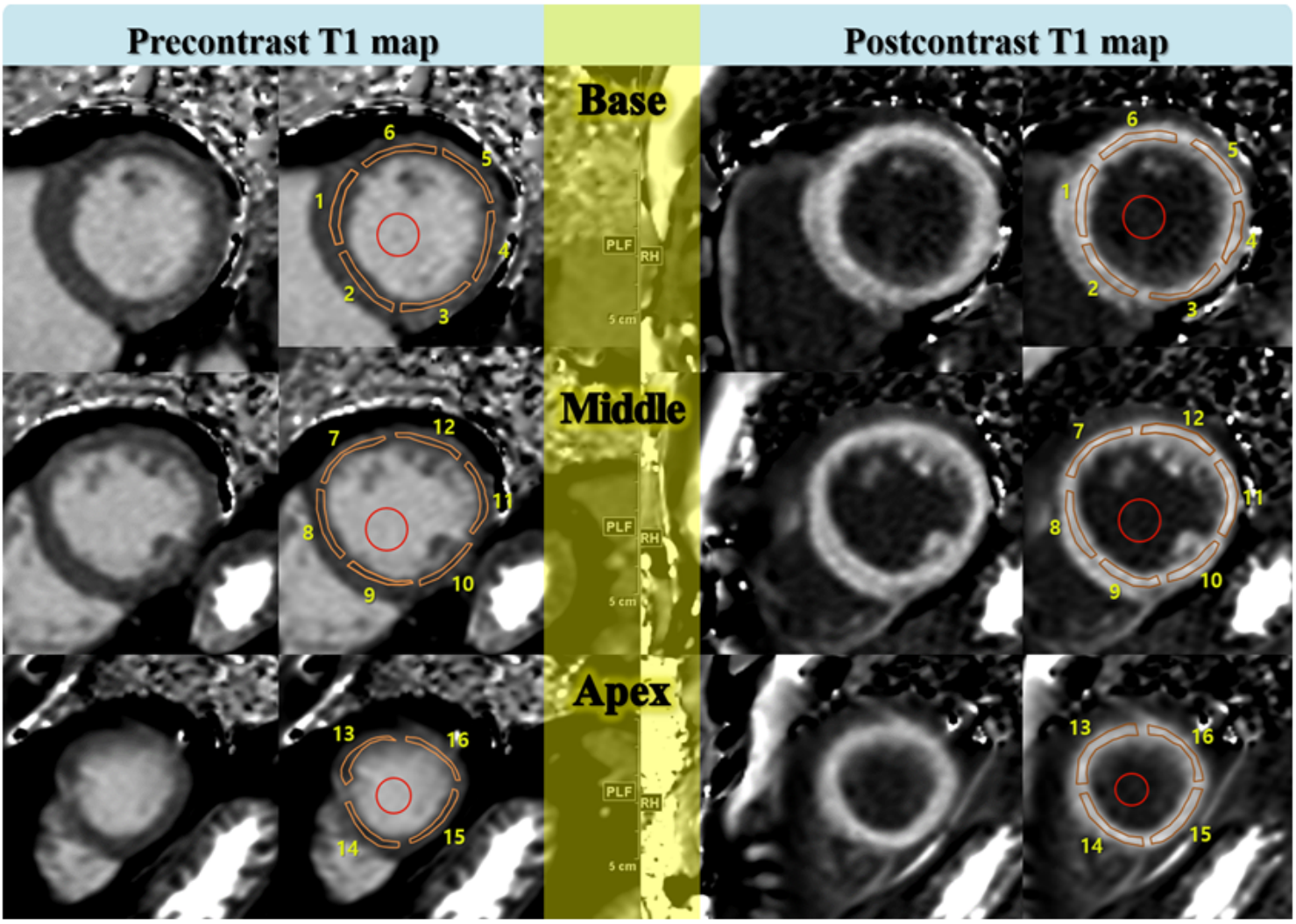

Figure 2

Short-axis slices of pre-contrast and post-contrast reconstructed T1 maps. The corresponding regions of interest on left ventricular segments and cavity blood (red circles) were drawn on T1 maps in a 54-year-old male in the control group.

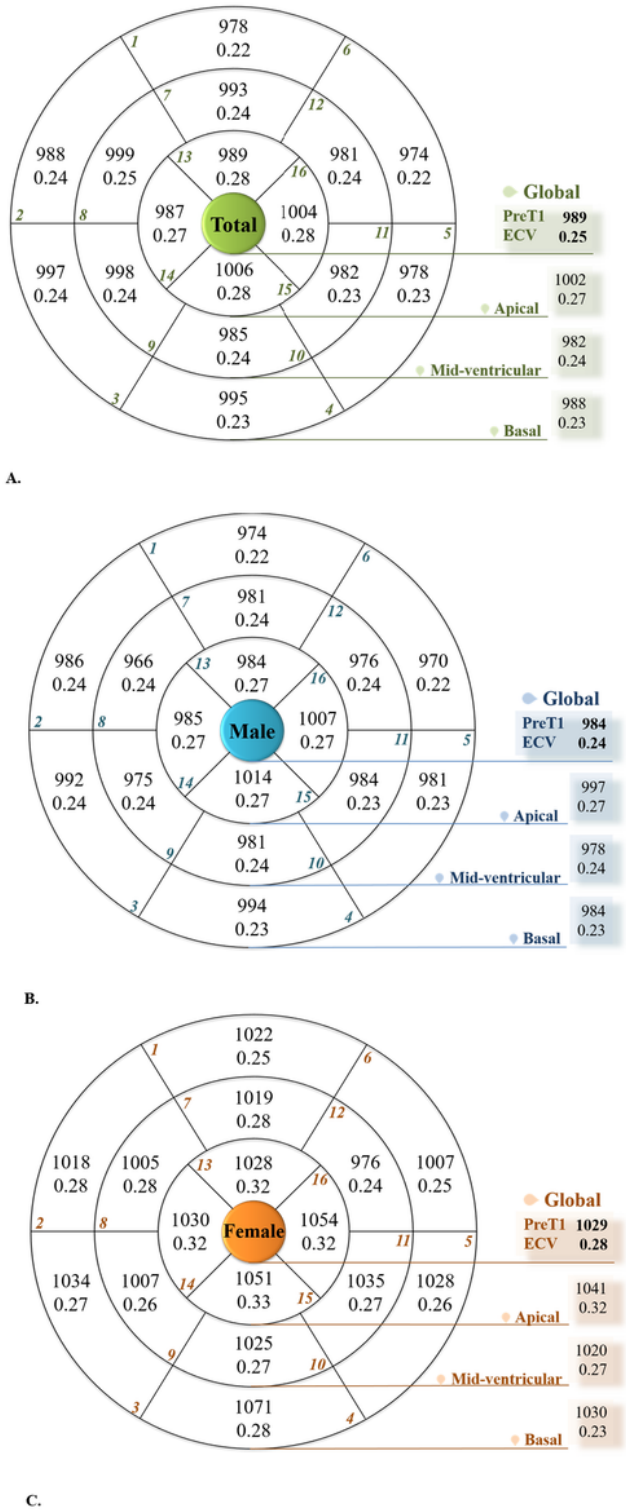

Figure 3

Mean segmental pre-contrast T1 value and mean extracellular volume fraction (ECV) of the left ventricle (LV). A. Total study population. B. Male. C. Female. Pre-contrast T1 value and ECV were significantly higher in women than in men in global LV segments ($p < 0.05$) except apical inferior segment.

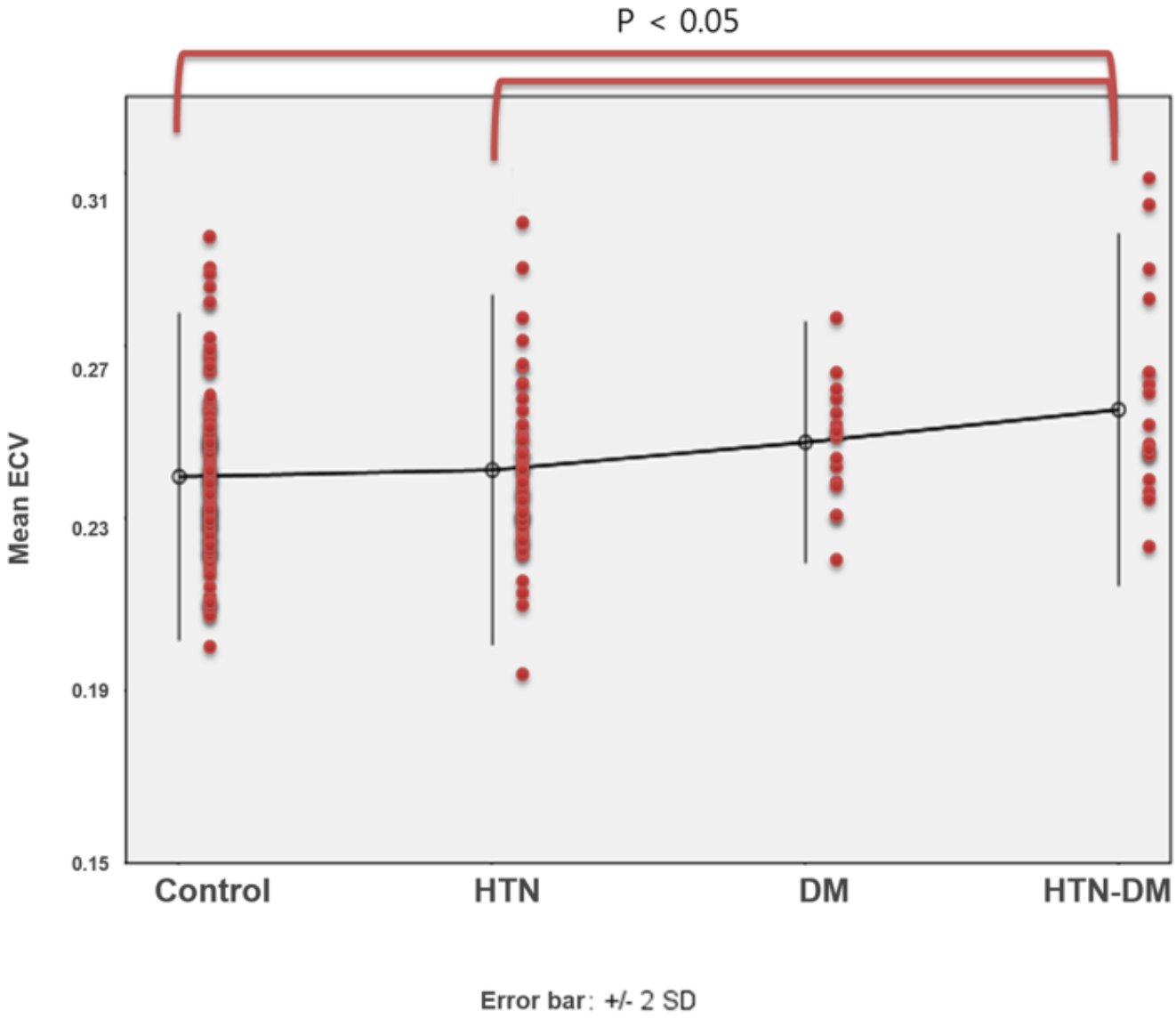

Figure 4

Relation of extracellular volume fraction (ECV) with hypertension (HTN) and diabetes mellitus (DM). HTN-DM group showed significantly higher ECV (0.260 ± 0.023) than control (0.240 ± 0.021, p = 0.011) and HTN groups (0.241 ± 0.024, p = 0.041). Error bars indicate 2 standard deviations (SDs) from average.

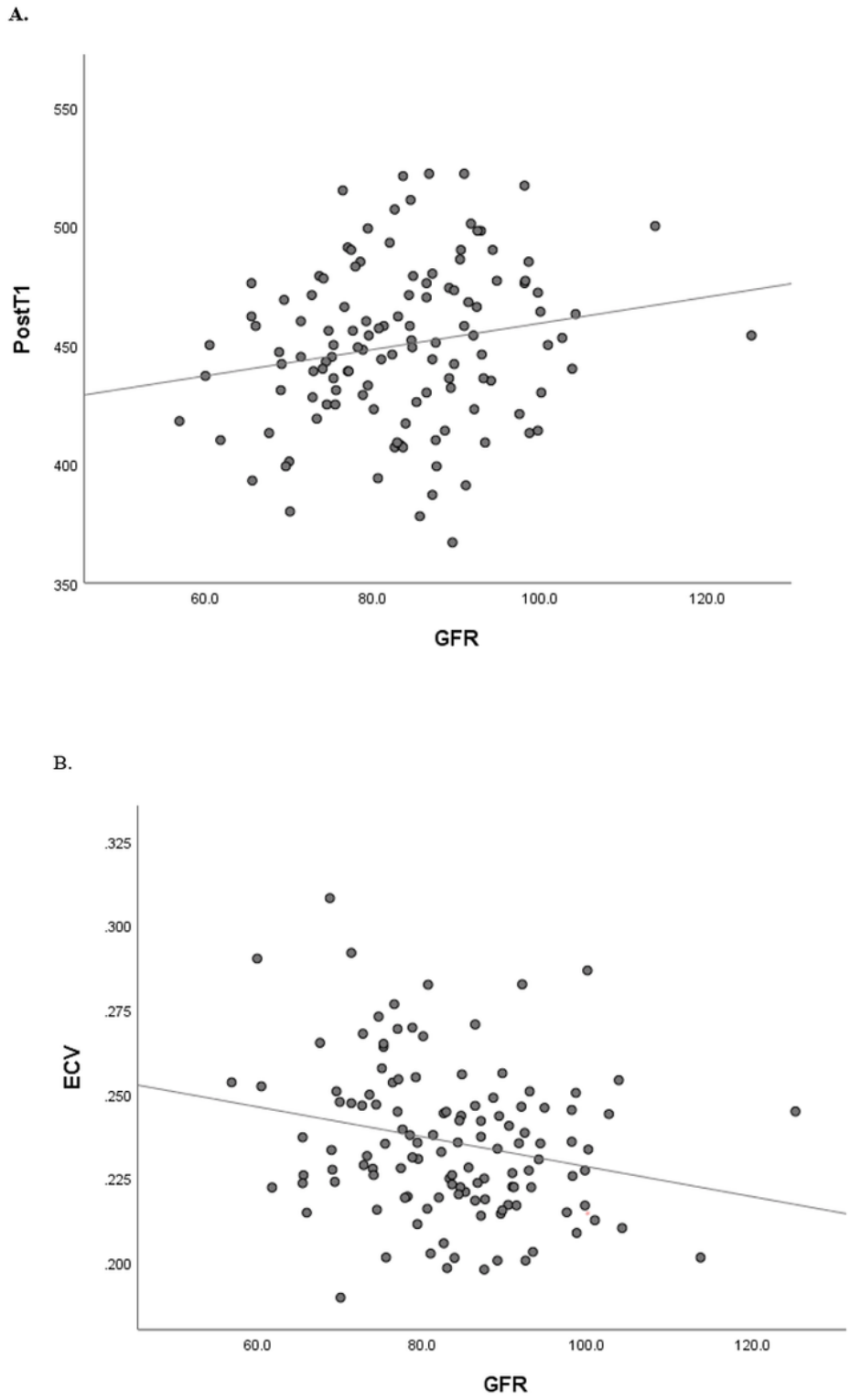

Figure 5

Relation of post-contrast T1 (PostT1) value and extracellular volume fraction (ECV) with glomerular filtration rate (GFR) in the control group. Overall PostT1 and ECV of left ventricle were proportional to GFR (A: r = 0.19, p = 0.038 for PostT1, B: r = -0.23, p = 0.011 for ECV).

Supplementary Files

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- Supplement2110.pdf