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ORIGINAL PAPER

Associations of B-type natriuretic peptide (BNP) and dialysis vintage with CMRI-derived cardiac indices in stable hemodialysis patients with a preserved left ventricular ejection fraction

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Abstract

To assess left ventricular myocardial native T1/T2 values and systolic strain and their associations with B-type natriuretic peptide (BNP) and dialysis vintage in hemodialysis (HD) patients with a preserved left ventricular ejection fraction (LVEF). Forty-three HD patients with end-stage renal disease (ESRD) but a preserved LVEF (≥ 50%) and 28 healthy volunteers were enrolled. BNP was measured at the time of cardiac magnetic resonance (CMR) measurements. Global native T1 and T2 values were significantly higher in the HD patients (native T1: 1056 ± 32 ms vs. 1006 ± 25 ms, $p < 0.001$; T2: 50 ± 3 ms vs. 46 ± 2 ms, $p < 0.001$) than in the controls. The mean peak global circumferential strain (GCS) and global longitudinal strain (GLS) were both significantly reduced in the HD patients compared with the controls (GCS: − 13 ± 3 vs. − 16 ± 3, $p < 0.001$; GLS: − 12 ± 4 vs. − 15 ± 3, $p = 0.001$). In the HD patients, the global native T1 value showed a positive correlation with the global T2 value ($r = 0.311$, $p = 0.042$) and significant correlations with GCS ($r = 0.564$, $p < 0.001$) and GLS ($r = 0.359$, $p = 0.018$). Significant positive correlations were found between lg BNP levels and T2 values ($r = 0.569$, $p < 0.0001$) and the left atrial volume index (LAVI) ($r = 0.536$, $p = 0.012$). GLS showed significant positive correlations with the LVMI ($r = 0.354$, $p = 0.020$) and dialysis vintage ($p = 0.026$; $r = -0.339$) in the HD patients. HD patients with a preserved LVEF have increased native T1/T2 values and decreased strain compared to controls. T2 values and the LVAI were positively associated with BNP in HD patients.

Keywords Cardiovascular disease · Hemodialysis · Magnetic resonance imaging · Strain · Fibrosis

Introduction

Chronic kidney disease (CKD) and end-stage renal disease (ESRD) have high mortality rates despite significant advances in hemodialysis (HD), and cardiovascular disease (CVD) is a major cause of morbidity and mortality in HD

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patients [1–3]. Left ventricular hypertrophy (LVH) is a very common cardiac finding in ESRD patients [4]. Additionally, postmortem and biopsy studies have demonstrated that patients with ESRD have high levels of fibrosis, which is associated with an increased risk of sudden cardiac death [2, 5]. Previous studies revealed that this pattern of fibrosis is greater in HD patients than in patients with milder CKD and is progressively more severe with increasing dialysis vintage [1, 3]. However, other studies [6, 7] showed that HD patients have better LV systolic function than CKD patients before HD. The cardiovascular morphology and function in long-term HD patients are not well described. Since the LV ejection fraction (EF) is often preserved (EF ≥ 50%) in most HD patients with LVH [8], early detection and treatment of myocardial abnormalities and dysfunction is essential for the prevention and management of cardiomyopathy in HD patients.

The use of cardiac magnetic resonance imaging (CMRI) T1 mapping to quantify diffuse myocardial fibrosis has been validated in many previous histological studies [9–11]. Some recent studies revealed that HD patients had increased native myocardial T1 values and decreased strain compared with healthy subjects [12, 13], but they cannot exclude the possibility of an effect of myocardial edema from fluid shifts on native T1 values [12]. Evaluating T2 values is a good technique for detecting myocardial edema [14, 15].

In addition, the level of B-type natriuretic peptide (BNP), which is synthesized in the ventricular myocardium in response to ventricular diastolic and wall stress, is an independent predictor of cardiovascular death and overall mortality in patients undergoing dialysis [16]. The BNP level was reported to be increased in HD patients [17] and in patients with CKD not yet requiring dialysis therapy [18]. However, little is known regarding the associations between BNP and CMRI-derived cardiac indices in stable HD patients.

Consequently, we aimed to assess LV myocardial native T1/T2 values and systolic strain and their associations with traditional markers of increased cardiac risk, namely, BNP and dialysis vintage, in HD patients with a preserved left ventricular ejection fraction (LVEF).

Materials and methods

Study population

We prospectively recruited 43 HD patients with a preserved LVEF (≥ 50%) and 28 healthy volunteers of similar age, sex and body mass index (BMI) from the nephrology department of Wuhan Union Hospital and the community, respectively.

The inclusion criteria for HD patients were maintenance hemodialysis for 4-h sessions three times a week for ≥ 3 months; clinically confirmed CKD; age between 30 and 80 years; no clinical manifestations of chest pain, dyspnea, and palpitations; no history of heart disease (congenital heart disease, coronary artery disease, valvular heart disease or cardiomyopathy); and normal electrocardiographic manifestations. The inclusion criteria for the controls were as follows: age from 30 to 80 years; no history of heart disease, hypertension, diabetes or hyperlipidemia; normal physical examination; and normal electrocardiographic manifestations. The exclusion criteria for HD patients were a history of peritoneal dialysis or renal transplant with an abnormal LVEF (< 50%) by CMR; an inability to undergo MRI scanning (due to metal implants, severe claustrophobia); poorly controlled hypertension, blood glucose or blood lipids; and

Table 1 Clinical characteristics of HD patients

Variable	All HD patients (n = 43)
Primary renal diagnosis (n, %)	
Chronic nephritis	16 (37.2)
Uarthritis	5 (11.2)
Diabetic nephropathy	7 (16.3)
Polycystic kidney disease	3 (7.0)
Drug-induced renal damage	2 (4.7)
Hypertensive nephrosclerosis	3 (7.0)
Unknown cause	3 (7.0)
Other causes	4 (9.3)
Dialysis vintage (mo)	39 ± 35
Hypertension (n, %)	35 (81.4)
SBP (mmHg)	155 ± 22
DBP (mmHg)	81 ± 13
Serum biochemistry	
BNP (pg/ml)	389.2 ± 178
Hemoglobin (mg/dl)	104 ± 16
PTH (mg/dl)	372 ± 191
Calcium (mg/dl)	2.3 ± 0.2
Phosphorus (mg/dl)	1.9 ± 0.5
Calcium–phosphorus product	46.7 ± 22.3
Albumin (mg/dl)	38 ± 3
Creatinine (mg/dl)	929 ± 113
Medical and drug history	
ACEIs/ARBs	17 (39.5)
Diuretics	10 (23.3)
CCBs	15 (34.9)
β-blockers	11 (25.6)
Statins	8 (18.6)

Data are presented as the mean ± SD, n (%)

SBP systolic blood pressure, *DBP* diastolic blood pressure, *BNP* B-type natriuretic peptide, *PTH* parathyroid hormone, *LVH* left ventricular hypertrophy, *ACEIs* angiotensin-converting enzyme inhibitors, *ARBs* angiotensin II receptor blockers, *CCBs* channel blockers

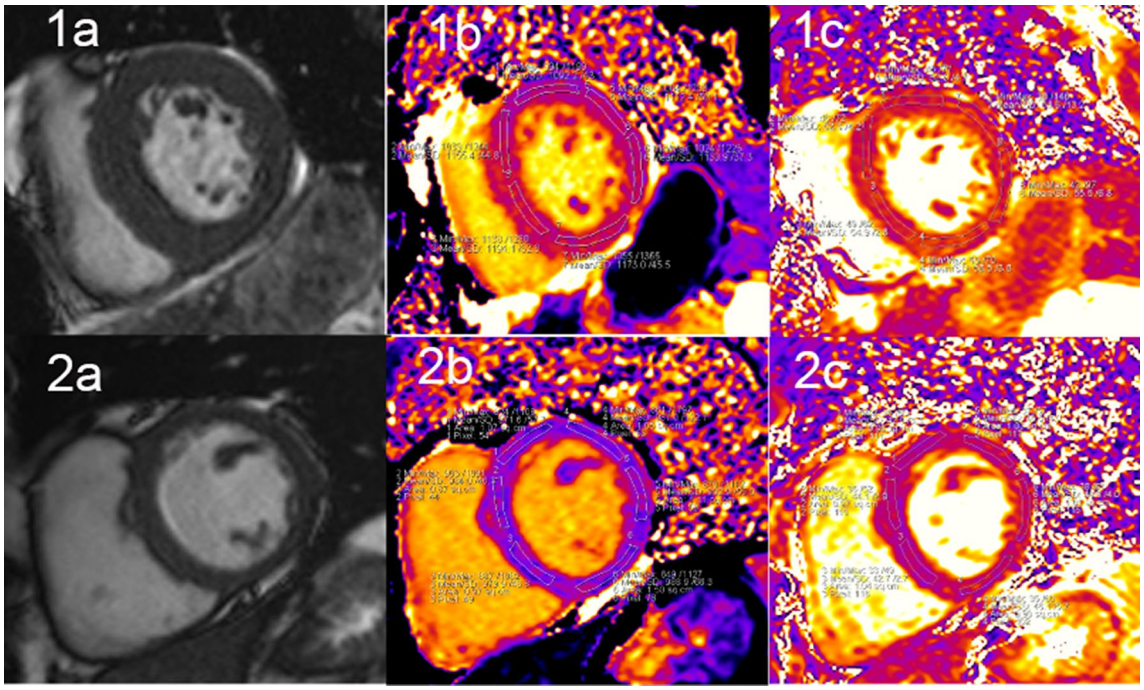

Fig. 1 Examples of end-diastolic cine images and corresponding T1 and T2 parametric maps in 1 hemodialysis (HD) patient and 1 control. The parts labeled **1a** show the left ventricular middle short-axis segment, and the parts labeled **1b** are the native T1 parametric maps at the same slice position in the same patient. The parts labeled **1c** are the T2 parametric maps at the same slice position in the same patient. The parts labeled with 1 correspond to a 64-year-old HD patient. The mean global T1 value is 1139 ms, and the mean global T2 value is 54 ms. The parts labeled **2a** show the left ventricular middle short-axis segment, and the parts labeled **2b** are the native T1 parametric maps at the same slice position in the same person. The parts labeled **2c** are the T2 parametric maps at the same slice position in the same person. The parts labeled with 2 correspond to a 65-year-old healthy volunteer. The mean global T1 value is 998 ms, and the mean global T2 value is 45 ms

inadequate CMR image quality. Data regarding demographics, medical comorbidities, dialysis vintage, hematology, and serum biochemistry were collected prospectively (Table 1). The HD patients were divided into three groups according to dialysis vintage: group A (dialysis vintage ≤ 3 years; n = 21), group B (dialysis vintage > 3 years and ≤ 5 years; n = 11) and group C (dialysis vintage > 5 years; n = 10). This study was approved by the ethics committee of the Tongji Medical College of Huazhong University of Science and Technology. The present study was conducted in accordance with the Helsinki Declaration. We confirm that all methods in the study were performed in accordance with the relevant guidelines and regulations. Written informed consent was obtained from all participants.

HD patient biomarkers and other clinical parameters

Data for parameters tested in blood collected at the time of imaging were obtained from electronic records, including hemoglobin, albumin, C-reactive protein, phosphate, parathyroid hormone (PTH), glucose, and predialysis creatinine and potassium levels; the urea reduction ratio; lipid profiles; BNP levels; and each HD participant's medical and dialysis history. Blood pressure was measured in the patients before an HD session. Patients were defined as hypertensive when the average systolic blood pressure (SBP) was greater than 140 mmHg. BMI was calculated by dividing dry weight (kg) by body height $(m)^2$. The plasma BNP level was determined by radioimmunoassay using a Triage immunofluorescence diagnostic instrument produced by Biosite, USA.

CMRI scanning protocol

Patients and controls were scanned with a 1.5-T MRI scanner (MAGNETOM Area, Siemens Healthcare, Erlangen, Germany) with vector electrocardiogram gating and 18-channel phased array surface coils. Dialysis patients were all scanned on nondialysis days but not after a long break; thus, all scans were conducted between 18 and 24 h after the most recent dialysis session [12]. Cells of the LV long axis and short axis (coverage from the base to the top region) were obtained by a balanced steady-state free parade (b-SSFP) sequence. The cine acquisition parameters were as follows: repetition

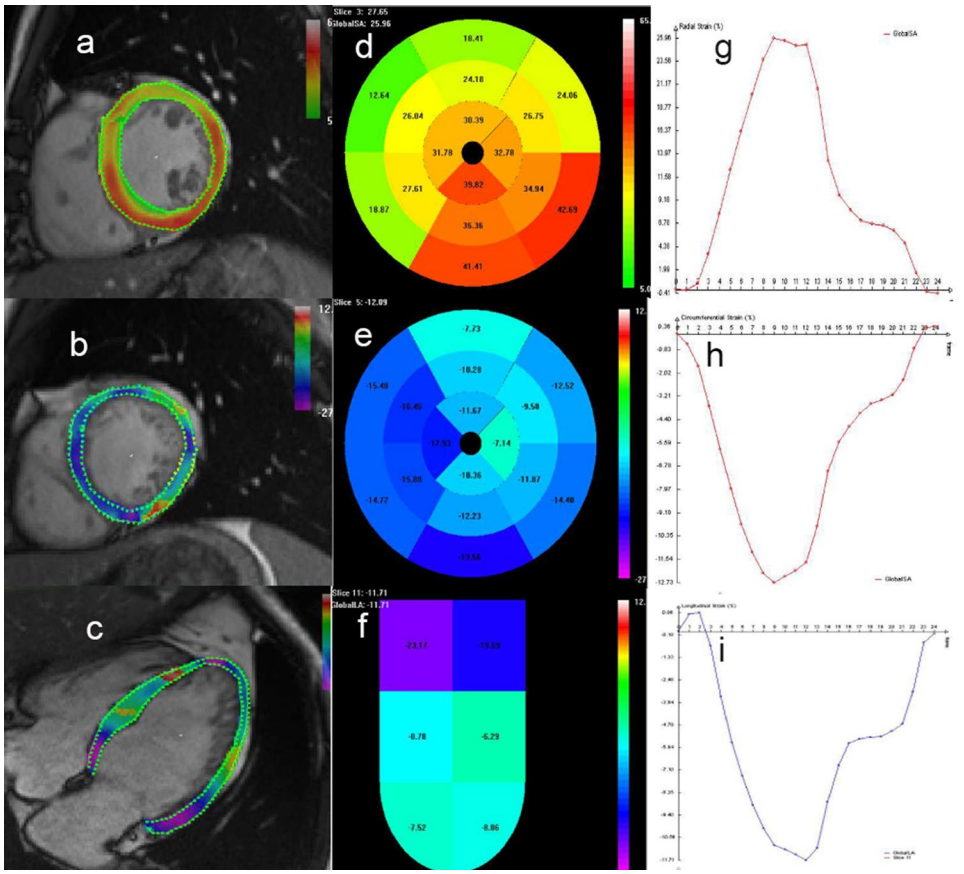

Fig. 2 Diagram of the peak systolic strain analysis of the left ventricular myocardium in a healthy volunteer by HDA software. The colored tissue-tracking maps from radial (**a**), circumferential (**b**), and longitudinal (**c**) strain analyses are shown on the left. The longitudinal (**d**), circumferential (**e**), and radial (**f**) strain values in a 16-segment model are displayed in the middle. The longitudinal (**g**), circumferential (**h**), and radial (**i**) strain–time curves in a cardiac cycle are shown on the right

time, 2.93 ms; echo time, 1.16 ms; slice thickness, 6 mm; flip angle, 80°; field of view, 340 × 255 mm; and matrix, 256 × 205.

The prototype modified look-locker inversion recovery (MOLLI) sequence was used to generate native T1 maps at the base, middle and top levels of the LV short axis. The T1 mapping acquisition parameters were as follows: repetition time, 3.89 ms; echo time, 1.12 ms; slice thickness, 8 mm; flip angle, 35°; field of view, 360 × 270 mm; and matrix, 256 × 192. The T2 values of the LV myocardium were measured on a T2 map generated using a single-shot SSFP technique prepared by T2. The T2 mapping acquisition parameters were as follows: repetition time, 3.244 ms; echo time, 1.35 ms; slice thickness, 8 mm; flip angle, 70°; field of view, 360 × 75 mm; and matrix, 192 × 83.

Assessment of cardiac volume and function

A commercial postprocessing software program (Argus, Siemens Healthineers) was used offline to analyze cardiac structure and function. Cardiac volumetric and functional parameters were quantified based on manual delineation of the endocardial and epicardial borders using a stack of continuous short-axis slice cine images (after excluding papillary muscles from the myocardium). The LV end diastolic volume (EDV), end systolic volume (ESV), EF, stroke volume (SV), Cardiac Index (CI) and myocardial mass index (LVMI) were obtained automatically. In addition, left atrial volumes (LAVs) were calculated according to the biplane area-length method (LAV = [0.85 × (2-chamber area) × (4-chamber area)]/L, where L is the shortest dimension between the above two chambers) [19]. All of the above measurements were indexed to body surface area (BSA).

Native T1 and T2 mapping measurement

Native T1 and T2 values were measured by manually delineating regions of interest in the mid-layer of the myocardium of the basal, middle and apical LV segments. The 16 regions of interest in each volunteer were drawn based on the American Heart Association 16-segment model [20] (Fig. 1). The susceptibility to motion artifacts of each individual part was evaluated. Any segments with artifacts affecting the measurements were eliminated. After any segment was removed, the global T1/T2 time was calculated from the average to calculate all the remaining values. To determine the reproducibility of myocardial strain measurements, the same images of 20 randomly selected individuals were repeatedly measured by the same observer and another blinded observer independently.

Table 2 Clinical and MRI characteristics of the study population

Variable	HD (n = 43)	Control (n = 28)	*p* values
Age (years)	59 ± 11	61 ± 7	0.463
Male (n, %)	28 (65.1)	14 (50)	0.227
BMI (kg/m^2)	22 ± 3	24 ± 2	0.068
HR (bpm)	73 ± 9	68 ± 10	0.025*
LVEF (%)	61 ± 8	64 ± 8	0.766
LVMI (g/m^2)	100 ± 33	58 ± 10	< 0.001*
LVEDV (mL)	76 ± 25	54 ± 10	< 0.001*
LVESV (mL)	33 ± 20	20 ± 7	0.001*
SV (ml/m^2)	43 ± 11	34 ± 5	< 0.001*
CI (ml/m^2)	3.2 ± 0.8	2.3 ± 0.5	< 0.001*
Peak ejection rate (EDV/s)	3.5 ± 1.0	3.6 ± 0.7	0.508
Peak filling rate (EDV/s)	3.5 ± 1.2	3.6 ± 0.8	0.482
LAVI (mL/m^2)	42 ± 21	36 ± 13	0.009
Global T1 (ms)	1056 ± 32	1006 ± 25	< 0.001*
Septal T1 (ms)	1066 ± 38	1015 ± 26	< 0.001*
Midseptal T1 (ms)	1068 ± 46	1022 ± 29	< 0.001*
Global T2 (ms)	50 ± 3	46 ± 2	< 0.001*
Septal T2 (ms)	50 ± 3	46 ± 2	< 0.001*
Midseptal T2 (ms)	50 ± 3	45 ± 3	< 0.001*
GRS (%)	39 ± 12	43 ± 10	0.126
GCS (%)	− 13 ± 3	− 16 ± 3	< 0.001*
GLS (%)	− 12 ± 4	− 15 ± 3	0.001*

All data are expressed as the mean ± SD, percentage (number of participants), or median (interquartile range), as appropriate

HD hemodialysis patients, *BMI* Body Mass Index, *HR* heart rate, *LVEF* left ventricular ejection fraction, *LVMI* Left Ventricular Mass Index, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *SV* stroke volume, *CI* Cardiac Index, *LAVI* Left Atrial Volume Index, *GRS* global radial strain, *GCS* global circumferential strain, *GLS* global longitudinal strain

*$p < 0.05$ between groups

Myocardial systolic strain measurement

Three-dimensional tissue tracking was performed offline using dedicated commercial software (TrufiStrain, version 2.0; Siemens Healthcare, Erlangen, Germany) based on heart deformation analysis (HDA). The short-axis cine images and two long-axis cine images were imported into this software. The LV endocardial and epicardial contours at the end of diastole were manually delineated on the short-axis and two long-axis cine images. The trabeculae and papillary muscles were included in the LV cavity. The LV global longitudinal strain (GLS), global circumferential strain (GCS), global radial strain (GRS) and early systolic strain rate were calculated by automatically tracking contours in each cardiac cycle (Fig. 2). To determine the reproducibility of the myocardial strain measurements, the same images from 15 randomly selected individuals were repeatedly measured by the same blinded observers.

Repeatability analysis

To determine the reproducibility of myocardial native T1 and T2 value and strain measurements, 15 individuals were randomly selected for repeatability analysis. The analysis was performed independently by two experienced radiologists, with one observer measuring the values again 2 weeks later. The measured data were used for consistency evaluations within and between observers. The mean values between the observers were taken as the results.

Statistical analysis

Statistical analyses of all data were carried out using SPSS software (SPSS 21.0 for Windows, IBM, Chicago, IL, USA). For all continuous data, the normality of the distribution was assessed using the Kolmogorov–Smirnov test. Normally and nonnormally distributed data and categorical variables are expressed as the means ± standard deviations, medians (interquartile ranges) and frequencies (percentages), respectively. The independent-sample Student's *t* test was used to compare two groups of normally distributed variables, and the chi-square test was used to compare categorical variables. Normally distributed variables were analyzed by Pearson's correlation, and nonnormally distributed data were analyzed by the Spearman correlation (log-transformed BNP levels and PTH). Multiple linear regression analyses were performed to identify determinants of myocardial native T1/T2 values and GLS in patients with HD. All candidate variables ($p < 0.10$ in univariable linear regression and without collinearity) were entered into a multiple stepwise regression model. Clinical characteristics and CMRI findings were compared among groups A, B and C by one-way ANOVA. A p value < 0.05 (two-tailed) was considered statistically significant.

Results

Clinical characteristics of the study population

A total of 71 subjects were enrolled: 43 HD patients and 28 healthy volunteers. The baseline demographic characteristics of the HD patients and the prescribed medications used in this group are shown in Table 1. The controls had no cardiovascular or systemic diseases and a normal electrocardiogram. The controls were not treated for hypertension or hypercholesterolemia and were not taking regular medications.

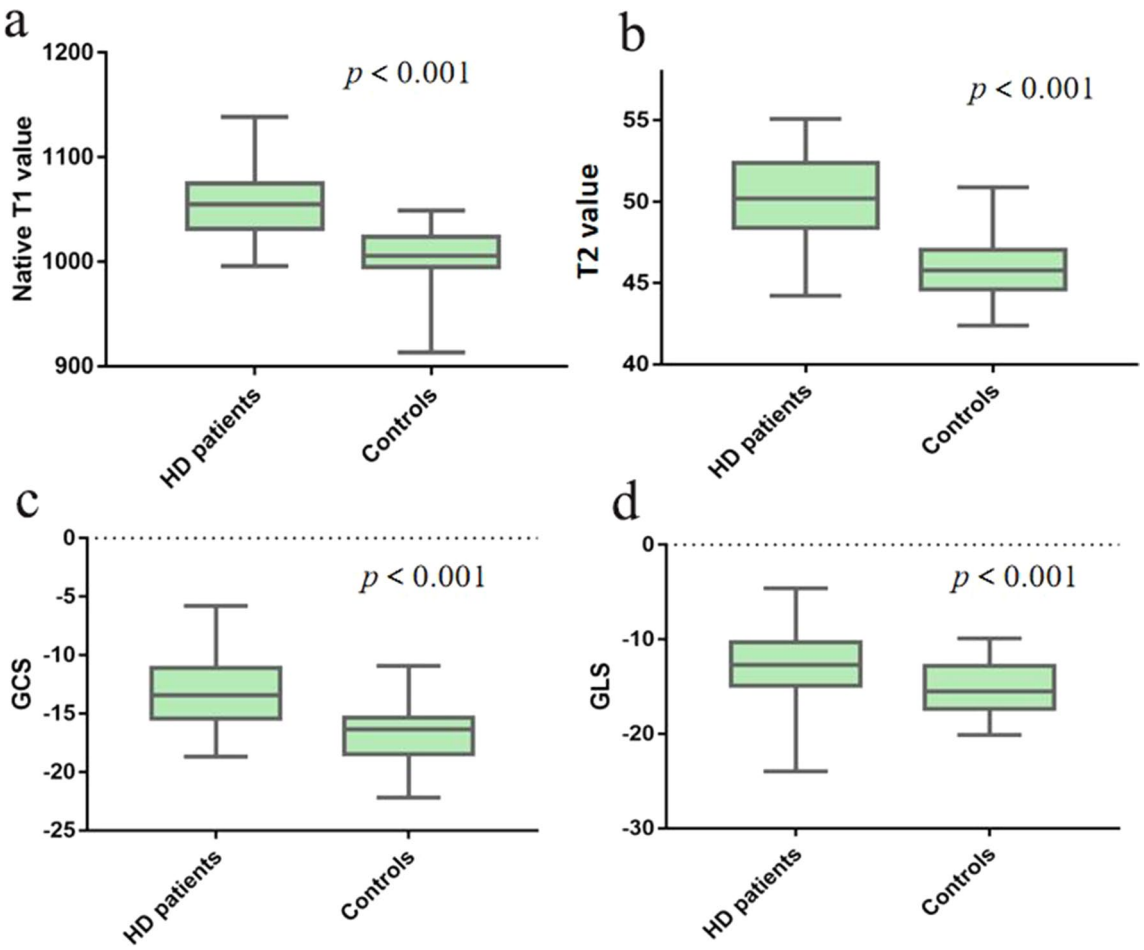

Fig. 3 Comparison of the mean native myocardial T1 (**a**) and T2 (**b**) values, peak global circumferential strain (**c**) and peak global longitudinal strain (**d**) between healthy controls and hemodialysis (HD) patients

Mass, volume and function of the left ventricle

Compared with those in the controls, the LAVI, LVMI, EDV and ESV were significantly increased (LAVI: 42 ± 21 vs. 36 ± 13, $p = 0.009$; LVMI: 100 ± 33 vs. 58 ± 10 g/m^2, $p < 0.001$; EDV: 76 ± 25 vs. 54 ± 10 mL/m^2, $p < 0.001$; ESV: 33 ± 20 vs. 20 ± 7 mL/m^2, $p < 0.001$) in the HD patients. However, no significant difference in the LVEF was found between the two groups (61 ± 8 vs. 68 ± 10%, $p = 0.766$). The lg BNP levels were significantly associated with the LAVI ($r = 0.536$, $p = 0.012$).

Native T1 and T2 values and strain

The global native T1 and T2 values were significantly higher in the HD patients than in the controls (native T1: 1056 ± 32 ms vs. 1006 ± 25 ms, $p < 0.001$; T2: 50 ± 3 ms vs. 46 ± 2 ms, $p < 0.001$) (Table 2) (Fig. 3a, b). Furthermore, we found that the native T1 values and T2 values of 16 segments of the left ventricle in the HD patients were higher than those in the healthy controls (Fig. 4a, b). The native T1 and T2 values showed no significant differences between the septal and nonseptal regions in the HD patients or in the controls (native T1: 1066 ± 38 ms vs. 1052 ± 31 ms, $p = 0.062$; T2: 50 ± 3 ms vs. 50 ± 3 ms, $p = 0.269$) (Table 2). The mean peak GCS and GLS were both significantly reduced in the HD patients compared with the controls (GCS: − 13 ± 3 vs. − 16 ± 3, $p < 0.001$; GLC: − 12 ± 4 vs. − 15 ± 3, $p = 0.001$) (Fig. 3c, d). No difference in GRS was found between the HD and control groups (39 ± 12 vs. 43 ± 10, $p = 0.126$) (Table 2).

After adjustment for heart rate (HR), the native T1 and T2 values and peak GCS and GLS were still significantly different between the two groups (Tables 3, 4).

Factors associated with myocardial native T1 and T2 values

The global native T1 value was correlated with gender ($r = 0.328$, $p = 0.032$). The global native T1 value showed a

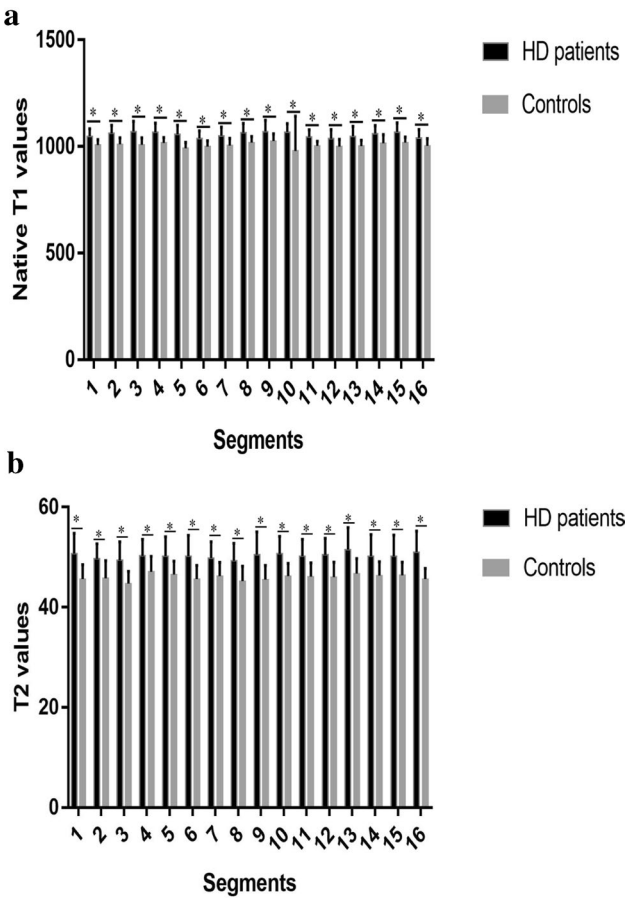

Fig. 4 Comparison of the native T1 values (**a**) and T2 values (**b**) of 16 segments of the left ventricular myocardium in hemodialysis (HD) patients and healthy controls, $*p < 0.05$

Table 3 MRI characteristics of study population adjusted for HR

Variable	HD (n = 43)	Control (n = 28)	*p* values
LVEF (%)	60 ± 1.2	63 ± 1.5	0.693
LVMI (g/m^2)	99 ± 4.1	58 ± 5.1	< 0.001*
LVEDV (ml)	77 ± 3.2	54 ± 4.0	< 0.001*
LVESV (ml)	33 ± 20	20 ± 7	0.001*
SV (ml/m^2)	43 ± 11	34 ± 5	< 0.001*
CI (ml/m^2)	3.2 ± 0.8	2.3 ± 0.5	< 0.001*
Peak ejection rate (EDV/s)	3.2 ± 0.1	3.5 ± 0.1	0.428
Peak filling rate (EDV/s)	3.5 ± 1.2	3.6 ± 0.8	0.482
LAVI (mL/m^2)	42 ± 3.2	37 ± 1.5	0.010*
Global T1 (ms)	1055 ± 4.5	1008 ± 6.0	< 0.001
Septal T1(ms)	1062 ± 3.4	1014 ± 5.6	< 0.001*
Midseptal T1 (ms)	1067 ± 6.1	1020 ± 4.2	< 0.001*
Global T2 (ms)	50 ± 0.4	46 ± 0.5	< 0.001*
Septal T2(ms)	50 ± 0.3	46 ± 0.4	< 0.001*
MidseptalT2(ms)	50 ± 0.4	45 ± 0.6	< 0.001*
GRS (%)	39 ± 1.7	43 ± 2.1	0.105
GCS (%)	− 13 ± 0.5	− 17 ± 0.6	< 0.001*
GLS (%)	− 13 ± 0.5	− 15 ± 0.6	0.001*

All data are expressed as the mean ± SD, percentage (number of participants), or median (interquartile range), as appropriate

HD hemodialysis patients, *HR* heart rate, *LVEF* left ventricular ejection fraction, *LVMI* Left Ventricular Mass Index, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *SV* stroke volume, *CI* Cardiac Index, *LAVI* Left Atrial Volume Index, *GRS* global radial strain, *GCS* global circumferential strain, *GLS* global longitudinal strain

$*p < 0.05$ between groups

positive correlation with the global T2 value ($r = 0.311$, $p = 0.042$) (Fig. 5a) as well as significant correlations with GCS ($r = 0.564$, $p < 0.001$) and GLS ($r = 0.359$, $p = 0.018$) in the HD patients but not in the control subjects. Significant positive correlations were found between the T2 value and GCS ($r = 0.346$, $p = 0.023$) and lg BNP(log-transformed) levels ($r = 0.569$, $p < 0.0001$) (Fig. 5b). On multivariable analysis, the independent determinants of the native T1 value were gender ($\beta = 0.365$, $p = 0.007$) and lg BNP levels ($\beta = 0.365$, $p = 0.007$) (model $R^2 = 0.271$) (Table 6). The independent determinant of the T2 value was the lg BNP level ($\beta = 0.545$, $p < 0.001$) (model $R^2 = 0.280$).

Factors associated with myocardial strain

GLS was found to be positively correlated with LVEF and the LVMI and negatively correlated with dialysis vintage in HD patients (LVEF: $r = -0.344$, $p = 0.024$; LVMI: $r = 0.354$, $p = 0.026$; $r = -0.339$, $p = 0.020$) (Fig. 5c, d). GRS showed a significant positive correlation with LVEF in the HD patients (GRS: $r = 0.548$, $p < 0.001$). GCS showed a significant negative correlation with LVEF in the HD patients (GCS: $r = -0.385$, $p = 0.011$) (Table 5). On multivariable analysis, the independent determinant of GLS was dialysis vintage ($\beta = -0.339$, $p = 0.029$) (model $R^2 = 0.094$) (Table 6).

In addition, GLS was independently correlated with dialysis vintage (standardized $r = -0.321$, $p = 0.044$). GLS was increased in the HD patients in group C compared with that in group A ($p = 0.008$) and group B ($p = 0.011$) patients (Fig. 5d). No significant differences in sex, BMI, age, EF, EDV, ESV, LVMI or native T1/T2 values were identified between the three groups (Table 7).

Intra- and interobserver reproducibility

The intraclass correlation coefficients (ICCs) in the intraobserver analysis were 0.977, 0.978, 0.980, 0.967, and 0.923 for the native T1 and T2 values, GRS, GCS, and GLS, respectively. The ICCs in the interobserver analysis were 0.967, 0.969, 0.971, 0.956 and 0.912 for the native T1 and T2 values, GRS, GCS, and GLS, respectively.

Table 4 Univariate correlation coefficients for native T1 and T2 values in HD patients

Variable	Global T1 (ms)		Global T2 (ms)	
	R value	*P* value	R value	*p* value
Age (years)	− 0.114	0.467	0.237	0.127
Sex	0.328	0.032	− 0.018	0.907
BMI (kg/m^2)	0.053	0.734	− 0.230	0.138
Dialysis vintage (mo)	0.039	0.805	0.137	0.381
SBP (mmHg)	0.120	0.442	0.242	0.118
DBP (mmHg)	− 0.145	0.354	0.196	0.208
Lg BNP (pg/ml)	0.267	0.084	0.545	< 0.0001*
Hemoglobin (mg/dl)	− 0.125	0.424	0.074	0.638
PTH (mg/dl)	0.027	0.862	− 0.056	0.722
Calcium (mg/dl)	0.022	0.897	0.062	0.713
Phosphorus (mg/dl)	0.199	0.231	0.103	0.538
Calcium-phosphorus product	− 0.006	0.972	0.078	0.618
Albumin (mg/dl)	0.203	0.506	0.005	0.987
Creatinine (mg/dl)	0.021	0.897	− 0.152	0.343
HR (bpm)	0.104	0.509	0.021	0.896
LVAI	0.224	0.149	0.252	0.103
LVEF (%)	− 0.125	0.423	0.147	0.347
LVMI (g/m^2)	0.027	0.863	0.015	0.925
LVEDV (ml)	0.008	0.959	0.142	0.365
LVESV (ml)	− 0.038	0.807	0.071	0.650
SV (ml/m^2)	0.089	0.572	0.192	0.218
CI (ml/m^2)	0.081	0.607	0.185	0.235
Peak ejection rate (EDV/sec)	− 0.075	0.635	0.085	0.588
Peak filling rate (EDV/sec)	0.023	0.884	0.079	0.614
Global T1 (ms)	–	–	0.311	0.042*
Global T2 (ms)	0.311	0.042*	–	–
GRS (%)	− 0.380	0.012*	0.037	0.815
GCS (%)	0.564	0.000*	0.346	0.023*
GLS (%)	0.359	0.018*	0.043	0.783

All data were analyzed using Person correlation

All data are expressed as the mean ± SD, percentage (number of participants)

HD hemodialysis patients, *BMI* Body Mass Index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *BNP* B-type natriuretic peptide, *Lg BNP* log transformation BNP levels, *PTH* parathyroid hormone, *HR* heart rate, *LVEF* left ventricular ejection fraction, *LVM* Left ventricular mass, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *SV* stroke volume, *GRS* global radial strain, *GCS* global circumferential strain, *GLS* global longitudinal strain

*$p < 0.05$ between groups

Discussion

ESRD patients with a preserved LVEF have been shown to have increased myocardial fibrosis [2, 5]. In this study, native T1 values were significantly higher and were associated with decreased GCS and GLS in the HD patients compared with the healthy controls, which is consistent with previous studies [12, 13]. Increased T1 values have been shown to be related to interstitial fibrosis in many previous histological studies [9–11]. However, native T1 values have been shown to be increased in several other diseases, including myocardial edema [21], acute myocardial infarction, amyloidosis [22], and nonischemic cardiomyopathy [23]. Because no patients in our HD group had a history of amyloidosis, no differences in the in rates of previous myocardial infarction were found between the HD and control groups. Thus, these conditions would be unlikely to interfere with our results. However, we cannot exclude the possibility of fluid shifts in myocardial edema affecting the native T1 values. Moreover, we found that the global T2 value was significantly higher in the HD patients than in the controls, although Graham et al. [24] showed that the reproducibility of native T1 mapping was excellent and unrelated to changes in markers of fluid status in HD patients. We found a positive weak but significant correlation between T2 values and native T1 values. Furthermore, Marlies et al. [25] demonstrated that native T1 values were significantly associated with fluid status, suggesting that chronic fluid overload may also modulate increased T1 values independent of fibrosis. Therefore, the high native T1 and T2 values in our study suggested that not only fibrosis but also interstitial edema or inflammation may potentially have a nonnegligible influence on HD patients. One possible mechanism is chronic inflammation of the heart in HD patients, which results in an increase in cardiomyocyte water exudation [26]. In addition, a significant positive correlation was found between the T2 value and GCS, indicating that myocardial edema was associated with decreased wall compliance.

BNP levels were reported to be increased in HD patients [17] and in patients with CKD not yet requiring dialysis therapy [18]. Moreover, previous studies found positive correlations between BNP levels and the LVMI and LVEF in HD patients [27, 28], suggesting that BNP levels are a good predictor of LV dysfunction. In the present study, positive correlations were found between BNP levels and T2 values and the LVAI in HD patients. High T2 values reflect myocardial edema or inflammation [14, 15], and the LAVI was proven to be markedly increased in ESRD patients and independently associated with LVH and LV systolic and diastolic dysfunction. Therefore, BNP may be a useful biomarker for detecting myocardial abnormalities and LA remodeling in HD patients.

To date, the effect of HD therapy on cardiac function in ESRD patients remains controversial. In our study, dialysis vintage was found to be independently correlated with GLS. Furthermore, we found that the HD patients in group C had markedly increased GLS values compared with those in the other two groups. One previous study showed that

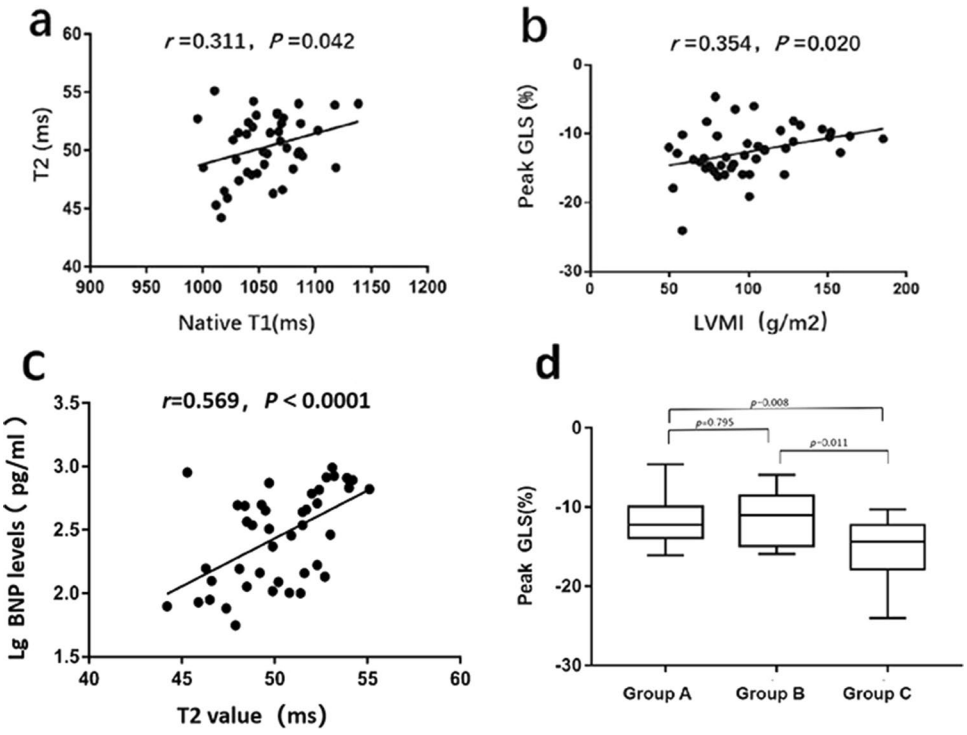

Fig. 5 **a** The relationship between native myocardial T1 and T2 values in hemodialysis patients; **b** the relationship between peak global longitudinal strain and the left ventricular mass index; **c** the relationship between native myocardial T2 values and lg BNP level in hemodialysis patients; **d** comparison of peak global longitudinal strain among the groups of different dialysis vintages (group A: dialysis vintage ≤ 3 years; group B: dialysis vintage > 3 years and ≤ 5 years; and group C: dialysis vintage > 5 years)

the process of progressive LVH continues after initiation of dialysis therapy, especially in the first year [3]. In a large-sample study, Kamyar et al. [29] demonstrated that greater fluid retention between two subsequent hemodialysis treatment sessions is associated with a higher risk of all-cause and cardiovascular death in HD patients. However, overhydration and accumulation of uremic toxins may influence the development of LVH and LV dysfunction in patients with chronic renal failure, suggesting that HD treatment may improve cardiac function by uremic toxin removal. Hayashi et al. [30] reported that myocardial function improves after one session of HD. Meanwhile, Liu et al. [6] showed that HD patients have better LV systolic function than moderate-advanced CKD patients by STE, and the better LV systolic function in HD patients may be due to the removal of uremic toxins [6]. Subsequently, Elaine et al. [7] also found that both GLS and the strain rate increased in patients following 6 months of HD. This result may also be due to an improvement in LV function or improved control of cardiovascular risk factors in the long dialysis vintage groups. Nevertheless, without a correlative analysis of the tissue, we cannot be certain that changes in GLS reflect an improvement in myocardial tissue abnormalities. Moreover, a possible explanation for more favorable findings in long-term survivors than in short-term HD patients is survival bias as patients with more severe cardiovascular characteristics died sooner while on dialysis [31]. Further studies are required to investigate the long-term effect of stable HD therapy on the cardiac function of patients, as well as the possible mechanisms.

Our study has some limitations. First, this was a single-center study with a relatively small cohort, and larger studies are required to confirm our findings and identify any potential prognostic benefits. Second, the lack of a histological correlation in this study is a limitation as this analysis could have provided further information. However, this limitation reflects the decreasing use of endomyocardial biopsy in routine clinical practice. Additionally, biopsy is limited by sampling error. Third, diastolic dysfunction and echocardiographic parameters were not available for our enrolled subjects. Finally, although our subjects included some HD patients with hypertension, which affects myocardial structure and function, we excluded patients with poorly controlled hypertension. Associations between blood pressure and CMR-derived cardiac indices were also not observed because of our exclusion of patients with poorly controlled hypertension according to the exclusion criteria. Howevver, a multivariable analysis was performed to exclude this confounding factor.

Table 5 Univariate correlation coefficients for GRS, GCS and GLS in HD patients

Variable	GRS (%)		GCS (%)		GLS (%)	
	R value	*P* value	R value	*P* value	R value	*P* value
Age (years)	0.343	0.024*	− 0.162	0.3000	− 0.087	0.579
Sex	− 0.056	0.723	− 0.009	0.956	0.024	0.877
BMI (kg/m^2)	− 0.043	0.786	0.055	0.728	0.146	0.350
Dialysis vintage (mo)	0.289	0.060	− 0.102	0.515	− 0.339	0.026*
SBP (mmHg)	0.054	0.729	0.120	− 0.145	0.242	0.118
DBP (mmHg)	0.201	0.196	0.442	0.354	0.196	0.208
Hemoglobin (mg/dl)	0.074	0.639	− 0.019	0.904	0.069	0.660
LgBNP (pg/ml)	0.288	0.061	− 0.066	0.676	− 0.118	0.450
PTH (mg/dl)	0.148	0.342	− 0.011	0.943	0.005	0.973
Calcium (mg/dl)	0.046	0.783	0.109	0.515	− 0.150	0.368
Phosphorus (mg/dl)	0.185	0.267	− 0.186	0.263	0.120	0.472
Calcium-phosphorus product	0.054	0.733	− 0.004	0.980	0.059	0.706
Albumin (mg/dl)	− 0.530	0.063	0.131	0.669	0.578	0.038*
Creatinine (mg/dl)	0.238	0.135	− 0.209	0.190	− 0.063	0.697
HR (bpm)	− 0.039	0.802	0.094	0.548	0.108	0.652
LVAI	0.187	0.230	− 0.131	0.402	0.067	0.671
LVEF (%)	0.548	0.000*	− 0.348	0.022*	− 0.344	0.024*
LVMI (g/m^2)	− 0.267	0.084	0.149	0.339	0.354	0.020*
LVEDV (ml)	− 0.039	0.802	0.094	0.548	0.108	0.491
LVESV (ml)	0.016	0.917	− 0.109	0.486	− 0.094	0.549
SV (ml/m^2)	0.264	0.087	− 0.205	0.188	0.013	0.936
CI (ml/m^2)	0.183	0.239	− 0.140	0.369	0.079	0.615
Peak ejection rate (EDV/sec)	0.011	0.942	0.122	0.437	0.099	0.528
Peak filling rate (EDV/sec)	0.338	0.027	− 0.128	0.415	− 0.063	0.686
Native T1 (ms)	− 0.380	0.012*	0.564	0.000*	0.359	0.018*
T2 (ms)	0.037	0.815	0.346	0.023*	0.043	0.783
GRS (%)	− 0.67	–	− 0.675	0.000*	− 0.623	0.000*
GCS (%)	5	0.000*	–	–	0.504	0.001*
GLS (%)	0.623	0.000*	− 0.505	0.001*	–	–

All data are expressed as the mean ± SD, percentage (number of participants), or median (interquartile range), as appropriate

HD hemodialysis patients, *BMI* Body Mass Index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *BNP* B-type natriuretic peptide, *Lg BNP* log transformation BNP levels, *PTH* parathyroid hormone, *HR* heart rate, *LVEF* left ventricular ejection fraction, *LVM* Left ventricular mass, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *SV* stroke volume, *GRS* global radial strain, *GCS* global circumferential strain, *GLS* global longitudinal strain

[a]Analyzed using Spearman correlation, all others using Person correlation

*$p < 0.05$ between groups

Conclusion

In conclusion, HD patients with a preserved LVEF have increased native T1/T2 values and decreased strain compared to controls. T2 values and the LVAI were positively associated with BNP in HD patients. Native T1/T2 mapping and strain may have the potential to quantify the severity of early cardiomyopathy in HD patients and monitor the progress of myocardial abnormalities with HD therapy. Future research with larger sample sizes should focus on investigating the long-term effect of stable HD therapy on the cardiac function of patients, as well as the possible mechanisms.

Table 6 Independent determinants of native T1 and T2 value and GLS in HD patients

Variable	Unstandardized β	Standardized β	*p* value
Native T1			
Gender	35.378	0.533	0.001*
Lg BNP	24.239	0.490	0.002*
T2 value			
Lg BNP	3.944	0.545	< 0.001*
GLS			
Dialysis vintage	− 0.035	− 0.339	0.029*

BNP B-type natriuretic peptide, *LgBNP* log transformation BNP levels, *GLS* global longitudinal strain

*p value < 0.05

Table 7 Comparison the difference of clinical characteristics and MRI characteristics among groups A, B and C in HD patients

Dependent variable	(I) group	(J) group	Std. error	Sig	95% confidence interval	
					Lower bound	Upper bound
Gender	Group A	Group B	0.181	0.455	− 0.23	0.50
		Group C	0.187	0.923	− 0.40	0.36
	Group B	Group A	0.181	0.455	− 0.50	0.23
		Group C	0.214	0.474	− 0.59	0.28
	Group C	Group A	0.187	0.923	− 0.36	0.40
		Group B	0.214	0.474	− 0.28	0.59
Age	Group A	Group B	4.22680	0.565	− 10.9972	6.0881
		Group C	4.36542	0.418	− 5.2501	12.3956
	Group B	Group A	4.22680	0.565	− 6.0881	10.9972
		Group C	5.00122	0.235	− 4.0806	16.1351
	Group C	Group A	4.36542	0.418	− 12.3956	5.2501
		Group B	5.00122	0.235	− 16.1351	4.0806
BMI	Group A	Group B	1.12113	0.787	− 1.9613	2.5704
		Group C	1.15790	0.368	− 1.2866	3.3938
	Group B	Group A	1.12113	0.787	− 2.5704	1.9613
		Group C	1.32654	0.575	− 1.9319	3.4301
	Group C	Group A	1.15790	0.368	− 3.3938	1.2866
		Group B	1.32654	0.575	− 3.4301	1.9319
EF	Group A	Group B	4.2638	0.459	− 11.804	5.431
		Group C	4.4036	0.315	− 13.379	4.421
	Group B	Group A	4.2638	0.459	− 5.431	11.804
		Group C	5.0450	0.799	− 11.489	8.904
	Group C	Group A	4.4036	0.315	− 4.421	13.379
		Group B	5.0450	0.799	− 8.904	11.489
EVD	Group A	Group B	9.3788	0.929	− 19.792	18.119
		Group C	9.6864	0.904	− 20.748	18.406
	Group B	Group A	9.3788	0.929	− 18.119	19.792
		Group C	11.0972	0.976	− 22.763	22.094
	Group C	Group A	9.6864	0.904	− 18.406	20.748
		Group B	11.0972	0.976	− 22.094	22.763
ESV	Group A	Group B	7.6412	0.687	− 18.543	12.343
		Group C	7.8918	0.470	− 21.703	10.197
	Group B	Group A	7.6412	0.687	− 12.343	18.543
		Group C	9.0412	0.771	− 20.926	15.620
	Group C	Group A	7.8918	0.470	− 10.197	21.703
		Group B	9.0412	0.771	− 15.620	20.926
SV	Group A	Group B	4.0447	0.577	− 5.902	10.447
		Group C	4.1774	0.275	− 3.823	13.063
	Group B	Group A	4.0447	0.577	− 10.447	5.902
		Group C	4.7858	0.626	− 7.325	12.020
	Group C	Group A	4.1774	0.275	− 13.063	3.823
		Group B	4.7858	0.626	− 12.020	7.325
LMI	Group A	Group B	8.2301	0.897	− 15.561	17.706
		Group C	8.5000	0.599	− 12.676	21.682
	Group B	Group A	8.2301	0.897	− 17.706	15.561
		Group C	9.7380	0.727	− 16.251	23.111
	Group C	Group A	8.5000	0.599	− 21.682	12.676
		Group B	9.7380	0.727	− 23.111	16.251

Table 7 (continued)

Dependent variable	(I) group	(J) group	Std. error	Sig	95% confidence interval	
					Lower bound	Upper bound
Native T1values	Group A	Group B	12.0751	0.632	− 30.240	18.569
		Group C	12.4711	0.824	− 27.993	22.417
	Group B	Group A	12.0751	0.632	− 18.569	30.240
		Group C	14.2875	0.832	− 25.829	31.923
	Group C	Group A	12.4711	0.824	− 22.417	27.993
		Group B	14.2875	0.832	− 31.923	25.829
T2 values	Group A	Group B	1.0021	0.794	− 2.288	1.762
		Group C	1.0349	0.276	− 3.234	.949
	Group B	Group A	1.0021	0.794	− 1.762	2.288
		Group C	1.1857	0.463	− 3.276	1.517
	Group C	Group A	1.0349	0.276	− 0.949	3.234
		Group B	1.1857	0.463	− 1.517	3.276
GRS	Group A	Group B	4.2015	0.833	− 7.601	9.382
		Group C	4.3393	.029*	− 18.584	− 1.044
	Group B	Group A	4.2015	0.833	− 9.382	7.601
		Group C	4.9713	.037*	− 20.751	− .657
	Group C	Group A	4.3393	.029*	1.044	18.584
		Group B	4.9713	0.037	0.657	20.751
GCS	Group A	Group B	1.2336	0.420	− 3.497	1.489
		Group C	1.2741	0.618	− 1.935	3.215
	Group B	Group A	1.2336	0.420	− 1.489	3.497
		Group C	1.4596	0.267	− 1.306	4.594
	Group C	Group A	1.2741	0.618	− 3.215	1.935
		Group B	1.4596	0.267	− 4.594	1.306
GLS	Group A	Group B	1.2351	0.795	− 2.819	2.174
		Group C	1.2756	0.008*	1.004	6.160
	Group B	Group A	1.2351	0.795	− 2.174	2.819
		Group C	1.4614	0.011*	0.951	6.858
	Group C	Group A	1.2756	0.008*	− 6.160	− 1.004
		Group B	1.4614	0.011*	− 6.858	− .951

BMI Body Mass Index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *BNP* B-type natriuretic peptide, *Lg BNP* log transformation BNP levels, *PTH* parathyroid hormone, *HR* heart rate, *LVEF* left ventricular ejection fraction, *LVM* Left ventricular mass, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *SV* stroke volume, *CI* Cardiac Index, *GRS* global radial strain, *GCS* global circumferential strain, *GLS* global longitudinal strain

*The mean difference is significant at the 0.05 level

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Data availability The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Compliance with ethical standards

Conflict of interest The authors declare no conflict of interest.

Ethics approval This study was approved by the ethics committee of Tongji Medical College of Huazhong University of Science and Technology.

Informed consent All subjects provided written informed consent.

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