CardioRenal Medicine

Cardiorenal Med 2018;8:271–284
DOI: 10.1159/000490395
Received: April 4, 2018
May 25, 2018
Published online: July 25, 2018

Original Paper

Left Ventricular Strain and Dyssynchrony in Young and Middle-Aged Peritoneal Dialysis Patients and Healthy Controls: A Case-Matched Study

Fei Shi[a] Sheng Feng[b] Jing Zhu[a] Yanni Wu[a] Jianchang Chen[a]

[a]Department of Cardiology, The Second Affiliated Hospital of Soochow University, Suzhou, China; [b]Department of Nephrology, The Second Affiliated Hospital of Soochow University, Suzhou, China

Keywords
Layer-specific strain · Dyssynchrony · Left ventricle function · Uremia · Young and middle-aged people · Two-dimensional speckle-tracking imaging · Peritoneal dialysis

Abstract
Background: This study aimed to evaluate the role of two-dimensional speckle-tracking imaging (2D-STI) and myocardial layer-specific analysis in evaluating early left ventricular (LV) myocardial function and systolic dyssynchrony in young and middle-aged uremic patients undergoing peritoneal dialysis (PD). ***Methods:*** We enrolled 31 PD patients aged ≤65 years with preserved LV ejection fraction (LVEF, ≥54%) as the PD group and 49 age-matched healthy people as the control group. Echocardiography was used to assess the left atrial diameter index (LADI, LAD/BSA), LV mass index (LVMI), LVEF, peak early diastolic velocity/late diastolic velocity (E/A) (measured by pulsed Doppler), and peak early diastolic velocity (by pulsed Doppler)/peak velocity of the early diastolic wave (by pulsed-wave tissue Doppler) (E/e′). Next, we used 2D-STI and myocardial layer-specific analysis to obtain longitudinal strains (LS) of the endocardium (LSendo), the myocardium (LSmyo), the epicardium (LSepi), and the global myocardium (GLS). Then, we measured the postsystolic index (PSI) to evaluate LV myocardial function. Time to peak LS (TTP) and peak strain dispersion (PSD) from 17 consecutive segments were assessed to quantify LV dyssynchrony. ***Results:*** Compared with the controls, PD patients had significantly increased LADI (p = 0.041), LVMI (p = 0.000), and E/e′ (p = 0.009), but reduced

Fei Shi and Sheng Feng contributed equally to this article.

Jianchang Chen
1055 Sanxiang Road
Suzhou, Jiangsu Province 215004 (China)
E-Mail chenjcsz@126.com

LVEF (p = 0.000) and E/A (p = 0.000). The average values of GLS (GLS avg) (p = 0.01) and GLS of the apical 2-chamber view (p = 0.003), including the LSendo (p = 0.024), LSmyo (p = 0.024), and LSepi (p = 0.032), were significantly decreased in patients with PD compared with controls. In PSI, segments of LS were markedly delayed in the anterior septum (p = 0.047), anterior (p = 0.000) and septum wall (p = 0.024) from basal segments, anterior wall (p = 0.001) from middle segments, and anterior (p = 0.024) and inferior (p = 0.024) wall from apical segments. Moreover, PSD was significantly increased in PD patients (p = 0.015), while TTP was evidently delayed in the anterior septum (p = 0.004), anterior (p = 0.000) and posterior (p = 0.042) wall from basal segments, and inferior wall (p = 0.048) from apical segments. ***Conclusions:*** Despite preserved LVEF, young and middle-aged PD patients developed LV dysfunction and myocardial systolic dyssynchrony earlier compared with controls.

Introduction

Peritoneal dialysis (PD) benefits from its advantages of preserving residual renal function with low cost and greater maneuverability outside the hospital, and is becoming a popular choice for renal replacement therapy in uremic patients. Related studies have shown that the number of uremic patients who die from cardiovascular events accounts for about 50% of the total deaths of dialysis patients [1]. Because of several traditional risk factors, including hypertension, diabetes, dyslipidemia, and smoking, and some nontraditional factors like protein carbamylation, oxidative stress, growth factors, and cytokines, cardiovascular diseases in young and middle-aged PD patients may develop earlier than in healthy age-matched people [2], contributing to a higher incidence of cardiovascular diseases and mortality. Therefore, it is of considerable significance to identify the early changes of heart function in young and middle-aged PD patients using effective screening methods. Early intervention will be needed to prevent the progression of heart failure and to reduce cardiovascular mortalities. The myocardial layer-specific analysis technique based on the two-dimensional speckle-tracking imaging (2D-STI) can evaluate the deformability of each layer of myocardium under tension. It is accurate, noninvasive, and independent of angle, and can reflect the whole and local myocardial function. Therefore, in this study, we used 2D-STI and myocardial layer-specific analysis to evaluate early left ventricular (LV) myocardial function and systolic dyssynchrony in young and middle-aged uremic patients at an early stage of PD. There are few related studies that have been reported, especially in China.

Methods

Study Subjects

We enrolled 31 young and middle-aged uremic patients who underwent PD catheter insertion in our PD center from June 2015 to June 2017.The inclusion criteria were as follows: (1) patient age ≤65 years (according to the report of the 2017 WHO meeting) [3], (2) progression to end-stage renal disease, (3) underwent PD regularly for 3–18 months, (4) the basic rhythm was sinus rhythm, and (5) the LV ejection fraction (LVEF) was ≥54% [4]. The exclusion criteria included congenital heart disease, hypertensive heart disease, valvular heart disease, cardiomyopathy of any other origin, heart block (right or left branch block, ventricular pre-excitation syndromes, etc.), pacemaker implantation, having had an acute myocardial infarction or acute heart failure, regional wall motion abnormality detected by echocardiography, any other nonrenal heart diseases, incomplete clinical data, and unsatisfactory echocardiographic images. Furthermore, 49 age-matched healthy people were chosen for the normal control group, disqualifying those with heart and kidney disease by confirmation with electrocardiogram, echocardiography, routine urine examination, renal function (normal serum creatinine and blood urea nitrogen), etc.

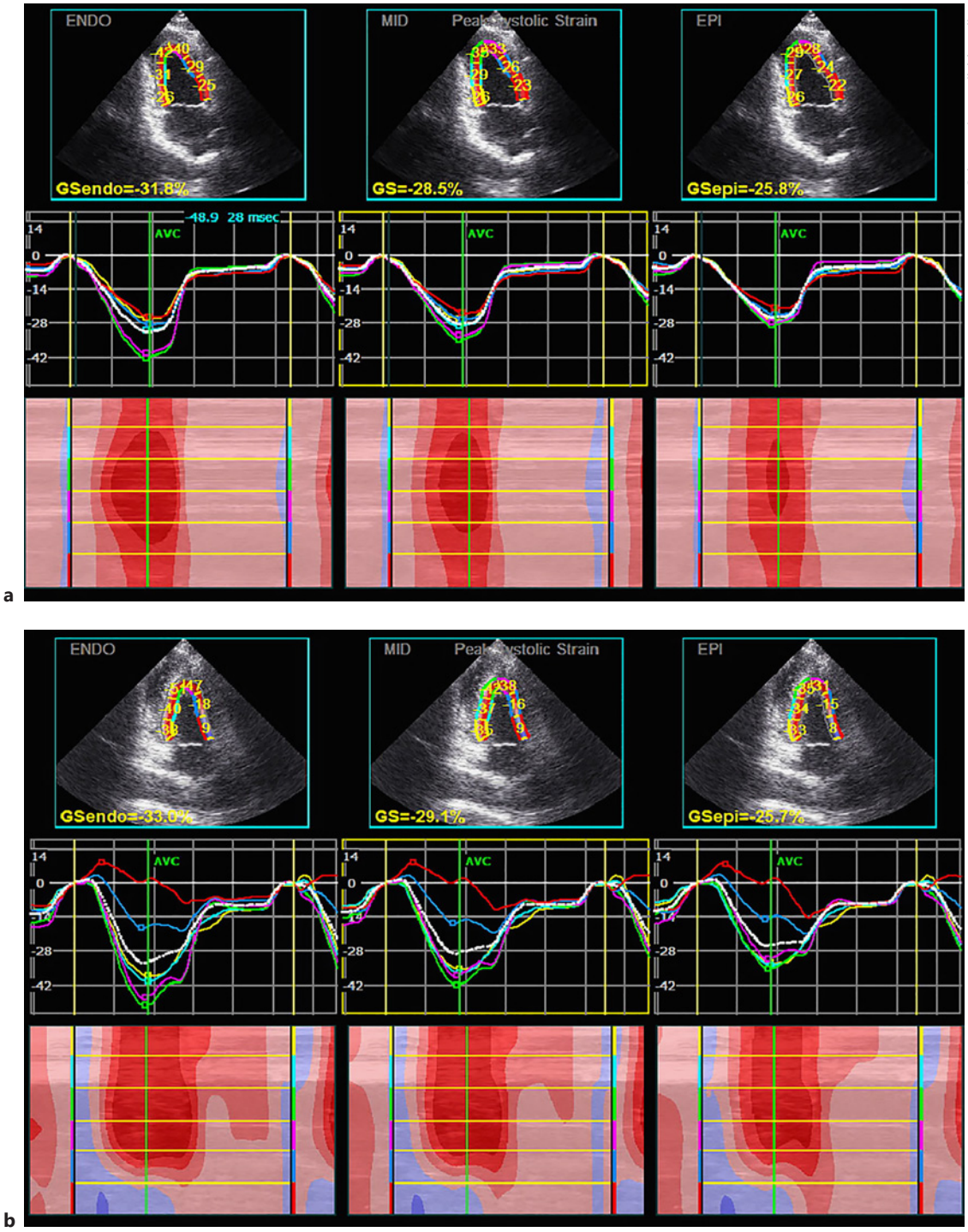

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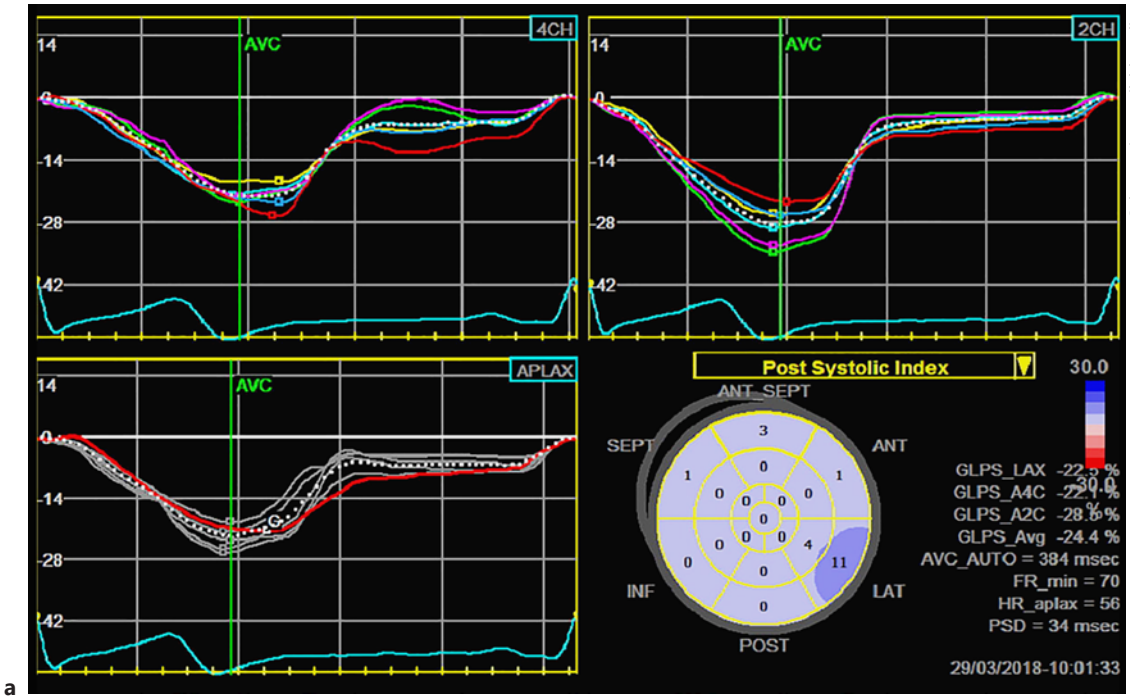
Fig. 1. Left ventricular (LV) longitudinal strain curves yielded by the two-dimensional speckle-tracking method and myocardial layer-specific analysis for endocardial, myocardial, and epicardial layers. **a** Control group. **b** PD group.



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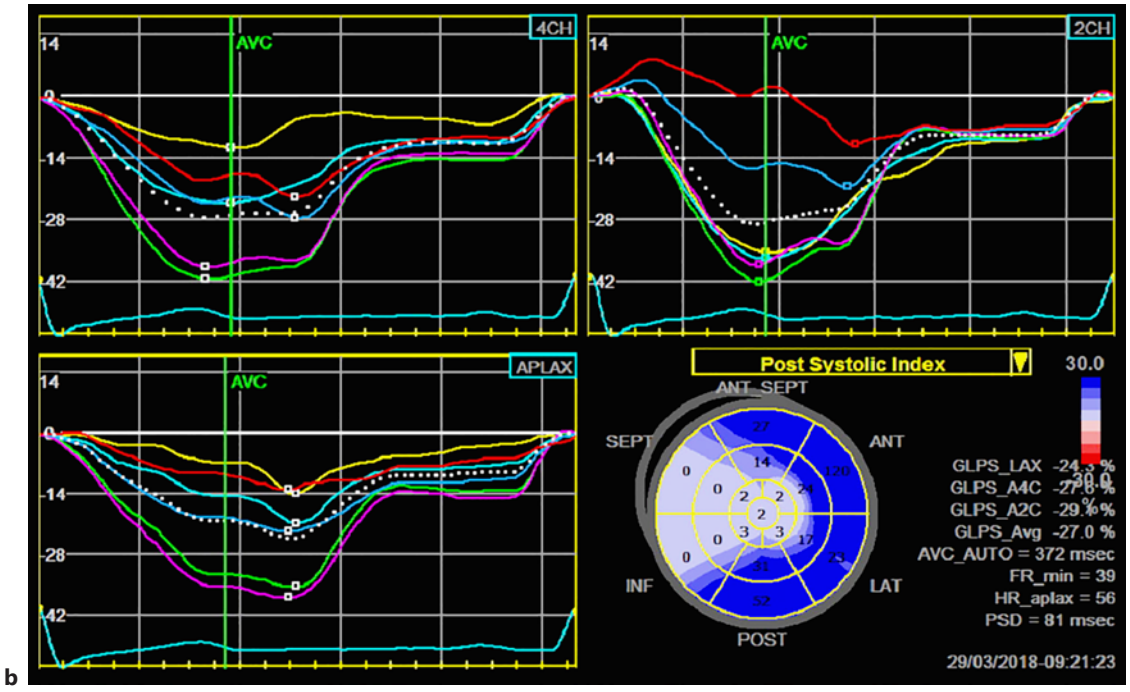

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Fig. 2. Bull's-eye images by the two-dimensional speckle-tracking method for the postsystolic index (PSI) for the multiple LV segments. **a** Control group. **b** PD group.

Study Methods

Conventional Echocardiography

All echocardiographic measurements were acquired with Model GE Vivid E9, using an M5S phased array transducer with a transmission frequency of 2.0–4.5 MHz. Images were taken of the parasternal view with patients lying in the left decubitus position. We recorded the left atrial diameter (LAD), LV internal diameter at end-diastole (LVIDD), LV internal diameter at end-systole (LVIDS), interventricular septal thickness (IVST), and LV posterior wall thickness (LVPWT). These were corrected by the body surface area (BSA) to derive the LAD index (LADI, LAD/BSA), LVIDD index (LVIDD/BSA), and LVIDS index (LVIDS/BSA). Then, we used the chamber diameter and wall thickness (LVPWT and IVST) to calculate the LV mass: LV mass (g) = 0.8 × 1.04 × [(LVIDD + LVPWT + IVST)^3 – LVIDD^3] + 0.6 [4]. The LV mass index (LVMI) was defined as the LV mass/height$^{2.7}$: >52 g/m$^{2.7}$ in men, and >47 g/m$^{2.7}$ in women, as suggested by the 2013 ESH/ESC guidelines [5]. LVEF was calculated using the modified Simpson method from the apical 4- and 2-chamber views, and the normal systolic function was defined as an LVEF ≥54% [4]. The peak early diastolic velocity (E) and late diastolic velocity (A) of the mitral orifice were measured by pulsed Doppler from the apical 4-chamber view. Then, the E/A ratio was calculated. The peak velocity of the early diastolic wave (e′) was measured by pulsed-wave tissue Doppler, with the sample volume close to the mitral valve annulus in the apical 4-chamber view in the lateral wall. We calculated the E/e′, and defined E/A <1 or E/e′ >13 as the presence of LV diastolic dysfunction, according to the recommendation from the American Society of Echocardiography [5]. All echocardiographic measurements were performed by experienced echocardiographic technicians who were blinded to the clinical conditions.

2D-STI and Layer-Specific Strain Analysis

Dynamic 2D sonographic images of 3 cardiac cycles were obtained from the standard apical long-axis view, and 4- and 2-chamber views. These images were digitally stored on hard disks for myocardial strain analysis using offline software EchoPAC 201. Manual tracings of the endocardial border during end-systole in 3 apical views were performed to measure longitudinal strain (LS), and the system automatically determined the tracking quality for each analyzed segment. The LV wall was automatically divided into 3 layers as follows: the inner third (endocardial), the middle third (myocardial), and the outer third (epicardial) view. The system then yielded LVLS curves for each of the 3 layers (Fig. 1). Next, each view was divided into 6 segments, finally, the LV was divided into basal, middle, and apical segments, and each segment was divided into the anterior septum, anterior, lateral, posterior, and inferior and posterior septum wall, totaling 17 segments. By averaging the LS of the 3 apical views, the global LS for each myocardial layer was obtained, and then the postsystolic index (PSI) in all 17 segments of the LV were directly reflected by a bull's-eye view automatically to evaluate LV myocardial function (Fig. 2).

Myocardial Dyssynchrony

The system automatically recorded time to peak LS (TTP) and peak strain dispersion (PSD) to evaluate LV myocardial systolic dyssynchrony (Fig. 3).

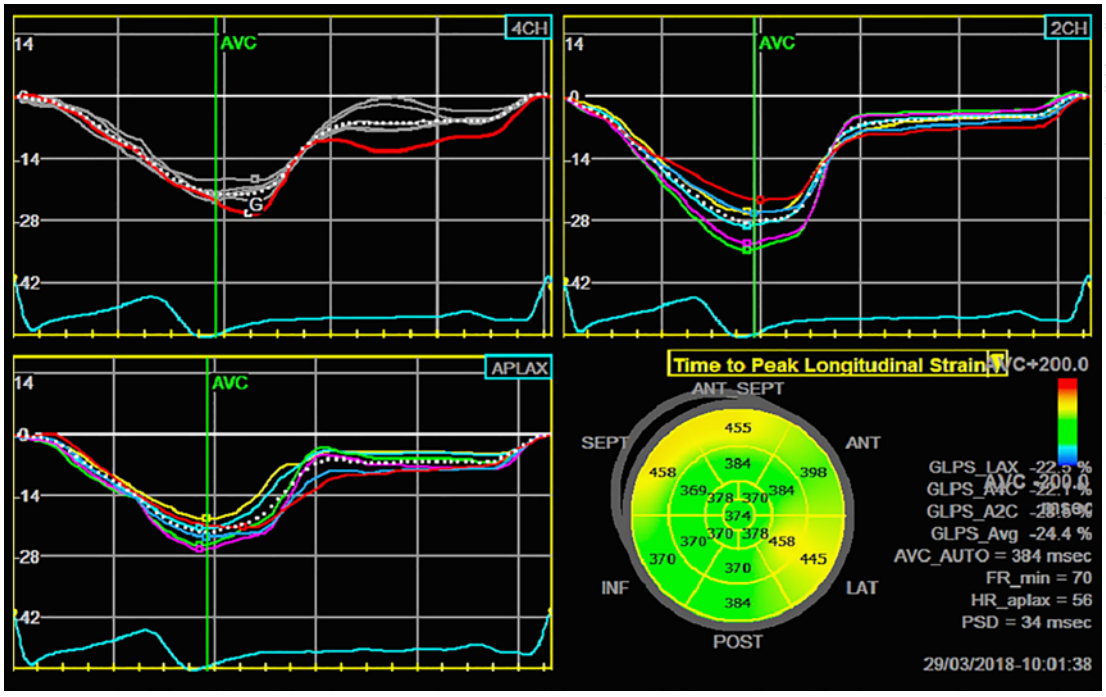
Statistical Analysis

All data and measurements are reported as mean ± SD. The independent sample test was used to compare normally distributed continuous variables. Pearson correlation analysis was performed to determine the association between PSI and TTP in the PD group. Statistical significance was considered as a p value less than 0.05. All statistical calculations were performed using SPSS 22.0 (IBM SPSS, Somers, NY, USA).

Results

Clinical and Conventional Echocardiographic Characteristics

The baseline characteristics of the study subjects are shown in Table 1. A total of 31 patients were included, and their average PD time was 11 months. In our study, no significant differences were found in subjects' age (p = 0.321) and BMI (p = 0.584), while systolic blood pressure (p = 0.001), diastolic blood pressure (p = 0.000), serum creatinine (p = 0.000), and blood urea nitrogen (p = 0.000) levels in PD patients were significantly increased. The under-



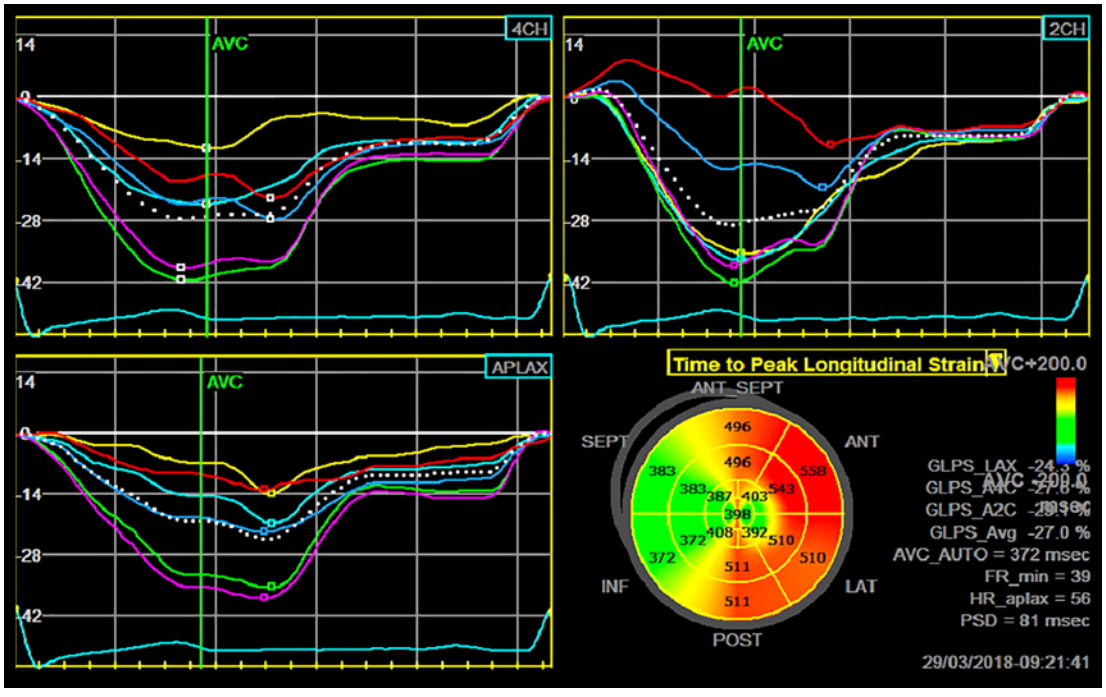


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Fig. 3. Bull's-eye images by the two-dimensional speckle-tracking method for the time to peak longitudinal strain (TTP) for the multiple LV segments and their peak strain dispersion (PSD). **a** Control group. **b** PD group.

Table 1. Comparison of clinical characteristics in patients and healthy controls

Variable	Case	Control	*p* value
Patients	31	49	
Age, years	43±9 (28–59)	41±9 (24–59)	0.321
Male gender	17 (54.8)	18 (36.7)	
PD time, months	11 (3–18)		
Diabetes	5 (16.1)		
Smoking	12 (38.7)		
BMI	23.04±2.80	22.57±3.17	0.584
SBP, mm Hg	137.2±24.77#	119.7±11.13	0.001
DBP, mm Hg	84.80±11.22#	74.84±8.64	0.000
CREA, μmol/L	909.1±339.39#	61.66±12.81	0.000
UREA, mmol/L	20.43±7.49#	4.930±1.12	0.000
Cause of ESRD			
Glomerulonephritis	17 (54.8)		
Diabetic nephropathy	5 (16.1)		
Hypertensive nephrosclerosis	4 (12.9)		
Polycystic kidney	2 (6.5)		
Nephrotic syndrome	1 (3.2)		
Unknown	2 (6.5)		
Medication use			
Diuretics	17 (54.8)		
RAAS blockers	17 (54.8)		
Ca^{2+} channel blockers	23 (74.2)		
α-Receptor blockers	21 (67.7)		
β-Receptor blockers	13 (41.9)		

Values are presented as mean ± SD, median (interquartile range), or number of subjects (%). PD, peritoneal dialysis; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; CREA, serum creatinine; UREA, blood urea nitrogen; ESRD, end-stage renal disease; RAAS, renin-angiotensin-aldosterone system. # $p < 0.01$.

Table 2. Comparison of conventional echocardiography parameters in patients and healthy controls

Variable	Case ($n = 31$)	Control ($n = 49$)	*p* value
LAD, mm	39.5±7.0#	32.2±3.9	0.000
LAD/BSA, mm/m^2	23.9±3.3*	20.6±1.9	0.041
LVIDD, mm	50.6±6.6#	45.1±4.1	0.000
LVIDD/BSA, mm/m^2	30.8±3.6	29.7±2.2	0.511
LVIDS, mm	33.2±5.8#	28.1±3.3	0.000
LVIDS/BSA, mm/m^2	20.2±3.7	18.3±1.6	0.261
IVST, mm	11.1±2.4#	8.30±1.2	0.000
LVPWT, mm	10.3±1.6#	8.27±1.1	0.000
LVEF, %	63.9±6.0#	67.9±5.2	0.000
LVMI, g/$m^{2.7}$	56.0±19#	31.0±4.4	0.000
E/A	0.90±0.3#	1.40±0.5	0.000
E/e’	8.10±3.2#	6.40±1.5	0.009

Values are presented as mean ± SD. LAD, left atrial diameter; LADI, left atrial diameter index; LVIDD, left ventricular internal diameter at end-diastole; LVIDS, left ventricular internal diameter at end-systole; IVST, interventricular septal thickness; LVPWT, left ventricular posterior wall thickness; BSA, body surface area; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; E/A, peak early diastolic velocity/late diastolic velocity (by pulsed Doppler); E/e’, peak early diastolic velocity (by pulsed Doppler)/peak velocity of the early diastolic wave(by pulsed-wave tissue Doppler). * $p < 0.05$, # $p < 0.01$.

Table 3. Comparison of LS in patients and healthy controls

Variable	Layer-specific strain, %		
	Case (n = 31)	Control (n = 49)	p value
4CH-LSendo	–23.53±4.20	–23.19±4.22	0.782
4CH-LSmyo	–20.63±3.91	–20.28±3.87	0.760
4CH-LSepi	–18.20±3.70	–17.85±3.62	0.747
4CH-GLS	–20.63±3.91	–21.57±4.42	0.447
2CH-LSendo	–21.82±5.23*	–25.23±4.66	0.024
2CH-LSmyo	–19.08±4.70*	–22.17±4.23	0.024
2CH-LSepi	–16.80±4.30*	–19.50±3.94	0.032
2CH-GLS	–19.08±4.70#	–23.23±4.06	0.003
LAX-LSendo	–22.31±5.52	–23.84±3.09	0.216
LAX-LSmyo	–19.82±5.10	–21.18±2.92	0.238
LAX-LSepi	–17.69±4.76	–18.92±2.83	0.261
LAX-GLS	–19.86±5.09*	–22.63±2.84	0.018
GLS avg	–19.85±3.72*	–22.46±2.35	0.010

Values are presented as mean ± SD. LS, longitudinal strain; GLS, global longitudinal strains; GLS avg, the average value of GLS; 2CH, 2-chamber view; 4CH, 4-chamber view; LAX, long-axis view; endo, endocardium; myo, myocardium; epi, epicardium. * $p < 0.05$, # $p < 0.01$.

lying causes of end-stage renal disease were glomerulonephritis in 54.8%, hypertensive nephrosclerosis in 12.9%, diabetic nephropathy in 16.1%, polycystic kidney in 6.5%, nephrotic syndrome in 3.2%, and not identified in 6.5% of patients. Overall, 74.2% of the patients were using Ca^{2+} channel blockers, 54.8% were receiving angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, and 67.7% were using α-receptor blockers (Table 1).

The conventional echocardiography parameters are shown in Table 2. LADI (LAD/BSA) (p = 0.041), LVMI (p = 0.000), and E/e′ (p = 0.009) in PD patients were significantly increased while LVEF (p = 0.000), E/A (p = 0.000) were significantly reduced. There was no significant difference in LVIDD/BSA (p = 0.511) between the groups (Table 2).

2D-STI and Layer-Specific Strain

Layer-specific strain in patients and controls is shown in Table 3, and PSI is shown in Table 4.

Global Endocardial, Myocardial, and Epicardial LS. The average value of GLS (GLS avg) was significantly decreased (p = 0.010), especially in the apical 2-chamber view. GLS was also significantly decreased (p = 0.003) and showed a decreasing trend from endocardium (LSendo) to epicardium (LSepi) (p = 0.024, p = 0.032, respectively) (Table 3).

Segment LS at Basal, Middle, and Apical LV. In PSI, segments of LS were markedly delayed in the anterior septum (p = 0.047), anterior (p = 0.000) and septum (p = 0.024) wall from basal segments, anterior wall (p = 0.001) from middle segments, and anterior (p = 0.024) and inferior (p = 0.024) wall from apical segments (Table 4).

Myocardial Dyssynchrony

TTP was significantly delayed in the anterior septum (p = 0.004), the anterior (p = 0.000) and posterior (p = 0.042) wall from basal segments, and the inferior wall from apical segments (p = 0.048) (Table 4). PSD was significantly increased in young and middle-aged uremic PD patients (p = 0.015) (Table 5).

Table 4. Comparison of PSI and TTP in patients and healthy controls

Variable	PSI, %			TTP, ms		
	Case (n = 31)	Control (n = 49)	p value	Case (n = 31)	Control (n = 49)	p value
bas (ANT-SEPT)	21.7±33.4*	5.10±10.7	0.047	452±80#	384±65	0.004
bas (ANT)	15.9±14.3#	2.40±4.40	0.000	450±60#	376±58	0.000
bas (LAT)	9.30±12.0	7.60±14.4	0.658	413±58	380±66	0.073
bas (POST)	6.30±10.2	3.90±8.80	0.401	402±53*	376±33	0.042
bas (INF)	2.10±3.40	6.60±13.9	0.191	368±57	392±58	0.167
bas (SEPT)	4.80±6.90*	1.60±2.60	0.024	378±58	371±49	0.646
mid (ANT-SEPT)	7.10±17.7	0.67±2.10	0.055	392±79	362±44	0.096
mid (ANT)	7.80±10.6#	0.94±2.40	0.001	406±85	368±51	0.054
mid (LAT)	3.80±6.00	3.90±7.20	0.956	390±70	404±101	0.560
mid (POST)	3.80±6.90	1.20±2.30	0.060	393±56	371±31	0.084
mid (INF)	1.30±2.80	2.60±7.60	0.483	361±55	368±48	0.644
mid (SEPT)	1.50±4.00	0.60±1.60	0.338	364±63	354±39	0.563
ap (ANT)	5.40±12.5*	0.10±0.30	0.024	381±69	354±32	0.070
ap (LAT)	1.20±2.50	5.80±16.2	0.247	367±51	375±42	0.561
ap (INF)	4.00±8.40*	0.39±1.10	0.024	386±67*	358±28	0.048
ap (SEPT)	1.80±5.20	1.80±4.00	0.997	361±47	365±35	0.736
ap (ap)	3.10±6.80	2.00±4.70	0.559	374±50	363±30	0.356

Values are presented as mean ± SD. ANT-SEPT, anterior septum wall; ANT, anterior wall; LAT, lateral wall; POST, posterior wall; INF, inferior wall; SEPT, septum wall; bas, basal segment; mid, middle segment; ap, apical segment; PSI, postsystolic index; TTP, time to peak longitudinal strain. * $p < 0.05$, # $p < 0.01$.

Table 5. Comparison of PSD in patients and healthy controls

Group	n	PSD
Case	31	53.6±17.6*
Control	49	39.7±19.6
p value		0.015

Values are presented as mean ± SD. PSD, peak strain dispersion. * $p < 0.05$.

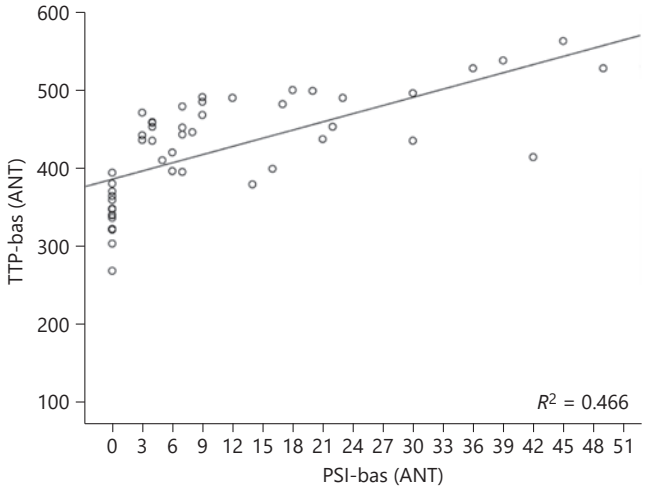
The Relationship between PSI and TTP

Our study showed that PSI and TTP had a strong correlation in the anterior and septum wall from basal segments and the anterior wall from middle segments (p = 0.000), and a moderate correlation in the anterior septum (p = 0.004), the posterior wall (p = 0.000) from basal segments, the anterior septum wall from middle segments (p = 0.000), and the anterior and inferior wall from apical segments (p = 0.000) (Fig. 4).

Discussion

Cardiovascular disease is the leading cause of death in late-stage uremic patients. Because of the increase of pressure and volume overload, early cardiac function damage is mainly due to compensatory hypertrophy of the myocardium in PD patients [6]. At that time, there is no obvious change in cardiac cavity size and systolic function, and it is still within the normal

Variable	*r*	*p*
bas (ANT-SEPT)	0.404	0.004
bas (ANT)	0.683	0.000
bas (POST)	0.517	0.000
bas (SEPT)	0.602	0.000
mid (ANT-SEPT)	0.507	0.000
mid (ANT)	0.705	0.000
ap (ANT)	0.552	0.000
ap (INF)	0.487	0.000



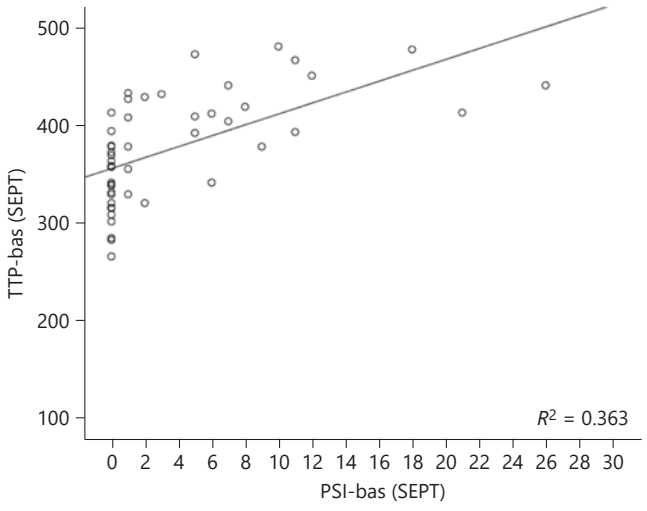


Fig. 4. Association between PSI and TTP. ANT-SEPT, anterior septum wall; ANT, anterior wall; POST, posterior wall; SEPT, septum wall; INF, inferior wall; bas, basal segment; mid, middle segment; ap, apical segment; PSI, postsystolic index; TTP, time to peak longitudinal strain.

range, lacking specific clinical manifestations. Therefore, it is important to evaluate cardiac function in young and middle-aged uremic PD patients at the early stage of PD.

Conventional Echocardiography

In this study, we used conventional echocardiography to show that in the PD group, LVEF is lower than that of normal age-matched people, while LVEF is still in the normal

range. LADI, LVIDD, LVIDS, IVST, LVPWT, and LVMI were increased, while LVIDD/BSA showed no significant difference. It is suggested that systolic dysfunction and LV remodeling exists in the PD group, and due to water and sodium retention, LVIDD/BSA is normal. In PD patients, the main damage to cardiac function is myocardial hypertrophy, which may compensate for an increase in the cardiac afterload, and allow the heart to maintain its normal systolic function and preserved ejection fraction. When the condition continues to decompensate, the LAD, LVIDD, and LVIDS obviously enlarge, and LVEF will reduce. The increased cardiac preload will also result in the false appearance of increased LV output, which makes the LVEF of PD patients within the normal range [1, 7]. Some studies have shown that mitral and aortic regurgitation can reduce the reliability of relevant indicators, and result in poor measurements, in which LVEF is preserved [8, 9]. Taken altogether, LVEF is often affected by heart rate and cardiac preload and afterload, and its value depends on the size and shape of the ventricle, and cannot reflect early and local myocardial damage [10]. In addition, we found that the E/A was significantly decreased while E/e′ was significantly higher in the case group than in the control group, though it was still in the normal range. These results suggest that diastolic function was also impaired in the young and middle-aged PD patients. This may be related to LV compensatory hypertrophy, and thus diastolic dysfunction can be seen in the early stage of PD.

2D-STI and Layer-Specific Strain Analysis

Layer-Specific Strain of LV. Some studies showed that GLS is stable, reproducible, and used as a load-independent measure of LV systolic function [4, 11]. It has a good correlation with LVEF, but is more sensitive than LVEF, can evaluate myocardial systolic function early, and can effectively reflect abnormal local myocardial blood supply [12, 13]. This study showed that the average value of GLS (GLS avg) decreased, indicating that these patients had early systolic dysfunction; this is consistent with the results of our conventional cardiac ultrasound measurements. LS in the anterior and inferior wall of the 2-chamber view was significantly decreased as well. Owing to the disorder of calcium and phosphorus metabolism, and the deposition of blood calcium, coronary artery calcification was easily found in uremic patients [14]. Coronary artery calcification mainly invades the main trunk and branches, with the left anterior descending branch being the most commonly invaded, followed by the right coronary artery and the left circumflex branch. The left anterior descending branch mainly supplies the anterior wall, the inferior wall, and the anterior septum wall [4], which contributes to ischemic injury and a decrease in LS of the corresponding myocardium in the 2-chamber view. Our study also proved that the LS decreased gradually from the endocardium to epicardium, with the most significant decrease in LSendo, exactly as in previous studies [15]. Due to the uneven thickness of the LV wall in the 3 layers of myocardium, compared with the epicardial layer, the systolic intensity of the endocardium was obviously higher, and the endocardial layer needed greater oxygen consumption; therefore, when perfusion was compromised, the endocardial layer was more vulnerable to ischemia [16]. Additionally, artery calcification often occurred in the intima membrane of the blood vessel, and endocardial fibers mainly consisted of longitudinal fibers, jointly causing LSendo to significantly decrease.

Segment LS at Basal, Middle, and Apical LV. The PSI is calculated as (postsystolic peak longitudinal strain – end-systolic strain)/end-systolic strain × 100% [17]. Many studies [18–21] showed that PSI can be seen in myocardial ischemia and hypertrophic cardiomyopathy, with overloaded LV volume, becoming a good predictor of regional myocardial dysfunction. In addition, PSI persists after stress due to prolonged stunning or myocardial hibernation, and can detect chronic ischemia in nonstress conditions and predict the degree of early myocardial damage without obvious cardiac insufficiency. In our studies, the PSI was signif-

icantly higher than that of the controls in the following: the anterior septum, anterior and septum wall from basal segments, anterior wall from middle segments, and anterior and inferior wall from apical segments. This indicates that in young and middle-aged uremic PD patients, early myocardial impairment occurred in the anterior wall, inferior wall, and septum wall. This is consistent with the finding that the GLS mainly decreased in the anterior and inferior walls in the 2-chamber view of LV myocardium.

Myocardial Dyssynchrony

TTP refers to the peak time from the beginning of the QRS wave to the peak of the LS extracted from the 17 segments of the myocardium. PSD is the standard deviation of the peak time of the 17 segments of the myocardium. TTP is widely used as a marker of LV mechanical dyssynchrony [22]. In our study, TTP was evidently delayed, and PSD was significantly increased, suggesting that PD patients have LV systolic dyssynchrony at the early stage of PD. Studies have shown that LV mechanical dyssynchrony may not only be caused by abnormal electrical activation, but may also be caused by inhomogeneous myocardial tissue damage, inhomogeneous ventricular wall structures, and abnormal loading conditions [23]. Because of the effects of long-term high afterload, renal toxicity, and volume imbalance (which worsens myocyte hypertrophy, fibroblast overgrowth, and the change of extracellular matrix substances), the intra- and intercellular electrical transmissions become delayed. Delayed myocardial contraction may occur in myocardial ischemia, stunning, or hibernation [24]. Furthermore, LV mechanical dyssynchrony may cause regional myocardial injury, and lead to inefficient LV contraction. Therefore, TTP and PSI were abnormal in almost the same myocardium in our study, and had a good positive correlation with each other. Due to the different structure of the three layers of myocardium and the calcification in different coronary arteries, the anterior wall, the inferior wall, and the septum wall were most commonly invaded. Our study also proved that TTP was evidently delayed in the anterior, the anterior septum, and the posterior wall from basal segments, and the inferior wall from apical segments.

Our study has its strengths. First, we collected complete data on young and middle-aged PD patients and the normal control group, including clinical indices and echocardiography data that consisted of the conventional echocardiography parameters, the LS of three layers and 17 segments, and thus the parameters reflecting dyssynchrony. One limitation of our study was the small sample size, and the fact that it was only a single-center observational and cross-sectional study. Furthermore, the follow-up time was not long enough. We need to continue to enlarge our sample size, follow up these patients, and trace the changes in related clinical indices with the prolongation of PD time, making our study results more reliable and prospective.

In summary, despite preserved LVEF, young and middle-aged uremic PD patients developed LV dysfunction and myocardial systolic dyssynchrony earlier compared with controls. Layer-specific evaluation of the LV function provided improved efficacy in discriminating early LV impairment. This may allow clinicians to track the progression of LV dysfunction in young and middle-aged uremic PD patients, and thus facilitate early medical intervention, such as further lowering the blood pressure to keep it more stable, stricter control of volume load, further adjustment of blood calcium and phosphorus levels, early application of RAAS blockers to improve ventricular remodeling, and better correction of anemia, which may provide PD patients with a primary or secondary preventive treatment.

Acknowledgments

This study was funded by the Department of Cardiology and Nephrology of the Second Affiliated Hospital of Soochow University. We thank the staff of the Echocardiography Room and Peritoneal Dialysis Center of The Second Affiliated Hospital of Soochow University for their help with data collection.

Statement of Ethics

This study was approved by The Second Affiliated Hospital of Soochow University's ethics committee.

Disclosure Statement

The authors certify that none of them has any financial or other conflicts of interest in connection with this paper.

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