CLINICAL SCIENCE

Effect of maintenance hemodialysis on diastolic left ventricular function in end-stage renal disease

Mustafa Duran,[I] Aydin Unal,[II] Mehmet Tugrul Inanc,[III] Fatma Esin,[III] Yucel Yilmaz,[III] Ender Ornek[I]

[I]Etlik Ihtisas Research and Educational Hospital, Department of Cardiology, Ankara, Turkey. [II]Erciyes University Medical Faculty, Department of Nephrology, Kayseri, Turkey. [III]Erciyes University Medical Faculty, Department of Cardiology, Kayseri, Turkey.

PURPOSE: To analyze the effect of maintenance hemodialysis on left ventricular diastolic function in patients with end-stage renal disease.

METHODS: Study population consisted of 42 patients with end-stage renal disease. Before an arteriovenous fistula was surgically created, the patients were evaluated by conventional and Doppler echocardiography and Doppler tissue imaging. Then, the patients undergoing hemodialysis treatment when the arteriovenous fistula was compleated. After the first hemodialysis session (mean 76.14 $\pm$ 11.37 days) the second echocardiographic evaluations were performed.

RESULTS: Mean age was 58 $\pm$ 13 years and 21 (%50) of the patients were female. After maintenance hemodialysis treatment; peak early (E) and peak late (A) diastolic mitral inflow velocities and E/A ratio were not significantly change however the deceleration time of E wave and left atrial diameter were significantly increased. Also there was no change in the early (Em) and late (Am) diastolic myocardial velocities and Em/Am ratios of lateral and septal walls of left ventricular. E/Em ratio was decreased insignificantly. Pulmonary vein velocities and right ventricular functions are remained almost unchanged after hemodialysis treatment.

DISCUSSION: The acute and long-term effect of hemodialysis on left ventricular diastolic function is unclearly. Patients with end-stage renal disease treatment with hemodialysis via arteriovenous fistula experience a variety of hemodynamic and metabolic abnormalities that predispose to alterations in left and right ventricular functions. The present study showed that left ventricular diastolic function except left atrial diameter and right ventricular functions were not significantly change, however left ventricular systolic functions were impaired after maintenance hemodialysis treatment in patients with end-stage renal disease.

CONCLUSION: It has been suggested that echocardiographic parameters are useful markers for evaluation of left ventricular and right ventricular functions in patients with end-stage renal disease. However, in patients with end-stage renal disease treated with hemodialysis, repeated assessment of echocardiographic examinations to observe serial changes in left and right ventricular functions are not yet well established. In this study, we showed that acute changes of volume status and electrolytes and autonomic regulation by hemodialysis session did not affect left ventricular diastolic and right ventricular functions in a relatively long term.

KEYWORDS: End-stage renal disease; Left ventricular diastolic function; Echocardiography; Hemodialysis.

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E-mail: mduran2@gmail.com

Tel.: 0090 505 3911620

INTRODUCTION

Cardiovascular complications are the most important cause of death in patients with end stage renal disease (ESRD) on hemodialysis treatment.[1,2] Left ventricular hypertrophy (LVH) and left ventricular (LV) systolic and diastolic dysfunction are the most common cardiovascular abnormalities and associated with increased morbidity and mortality in patients with ESRD.[3] These cardiac abnormalities may result from a variety of mechanisms including uremia, fluid retention, chronic volume overload, pressure overload, renal anemia, high-flow arteriovenous shunting, and hyperparathyroidism.[3-5] The effects of hemodialysis (HD), peritoneal dialysis and arteriovenous fistula (AVF) to the cardiac abnormalities are not clear yet.[6,7]

Arteriovenous fistula, which was used for vascular access in patients with ESRD, increases stroke volume load on the left ventricle and this may contributes to LVH and may results in LV systolic and diastolic dysfunction with time.[8] Also the presence of an AVF reduces systemic vascular

resistance.[9] Studies after AVF closure because of complications suggest that AVF may leads to progression of LVH and high cardiac output.[10]

Numerous echocardiographic techniques can be used to evaluate LV diastolic function. LV diastolic filling is analyzed from recordings of mitral inflow Doppler velocities. The velocity of the septal and lateral myocard, which has been shown to reflect the rate of myocardial relaxation, can be recorded with tissue Doppler imaging (DTI). Pulmonary vein flow velocities contribution to recognize diastolic dysfunction. Also the left atrial (LA) diameter and volume reflect the burden of LV diastolic filling.[11,12]

However, the pathophysiology of LV diastolic dysfunction and the contribution of AVF and hemodialysis to LV diastolic function remain unclearly in patients with ESRD. We aimed to investigate the effect of maintenance HD treatment on LV systolic and diastolic function in patients with ESRD.

PATIENTS AND METHOD

Patient Population

The prospective study was performed between January 2007 and February 2008 in Erciyes Univercity Medical Faculty Hospital. Study population consisted of 42 patients with ESRD. Before an AVF was surgically created for HD, the patients were evaluated by conventional and Doppler echocardiography. Then, an AVF was surgically created in the patients, who started HD via AVF when the AVF was compleated. After the first HD session (mean 76.14 $\pm$ 11.37 days) the second echocardiographic evaluations were performed. The patients were dialyzed via AVF three times a week for four hours. The second evaluations were performed 6-8 hours after the last HD session to avoid from acute volume loading. LV systolic and diastolic function parameters were obtained. The local ethics committee approved the study, and informed consent was obtained for each patient.

The patients, who had new diagnosis ESRD and planned HD via AVF, sinus rhythm, LV ejection fraction (EF) above 50%, no history of myocardial infarction, and no evidence of valvular disease included to the study. The patients with cerebral vascular disease, clinical and electrocardiographic evidences of myocardial ischemia, history of coronary artery disease, pericardial disease, heart failure (EF<50%), valvular heart disease and chronic pulmoner disease were excluded from the study. The patients had no signs or symptoms of heart failure at any time in the study.

Clinical and laboratory data were obtained from the patients on the day of echocardiographic examination before creation of AVF. These examinations were repeated after beginning of the HD session.

Echocardiographic evaluations

Echocardiographic studies were performed by the same cardiologist (MD) over three cardiac cycles before and after creation of AVF by Vivid 7 Dimension (General Electric Healthcare Company, Milwaukee, WI, USA) with a 3 MHz transducer in the left lateral position. Analysis was performed according to the guidelines of the American Society of Echocardiography recommendations.[13]

Left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD) and septal-posterior wall thicknesses were measured by M-mode in the parasternal long-axis view and EF was calculated according to the Teicholz formula via these measurements.[14] To evaluation of diastolic properties of the left ventricle, the mitral inflow velocities and Doppler tissue imaging (DTI) were evaluated from the apical four-chamber view. Pulmonary vein flow velocities were obtained from the right posterior pulmonary vein in the apical view. The LA diamater was measured in the parasternal long-axis view.[15]

Diastolic filling is classified on the basis of the peak early (E) and late (A) diastolic mitral inflow velocities, E/A ratio, E wave deceleration time (DT) and isovolemic relaxation time (IVRT). Also pulmonary vein flow velocities: systolic velocity (PVS), diastolic velocity (PVd) and atrial flow reversal velocity (PVa) were recorded. The early diastolic myocardial velocity (Em) and late diastolic myocardial velocity (Am) were recorded from midsegment of the lateral and septal myocardial walls with DTI.[12,13] Septal Em value was used to measure E/Em ratio. Right ventricle early (E) and late (A) ventricular inflow velocities were measured by pulsed wave Doppler placing the sample volume in between the tips of the tricuspid valve in the apical four chamber window.

End-diastolic left ventricular septal and posterior wall thicknesses (IVSEDD, PWEDD) and internal dimensions were used to calculate left ventricular mass by using the following equation: left ventricular mass = 1.04×0.8 [(left ventricular wall thicknesses + internal dimension) – (internal dimension)] + 0.6 g. Left ventricular hypertrophy was defined as left ventricular mass index (LVMI), which was calculated with left ventricular mass in grams divided by body surface area in square meters, higher than 116.0 for men and 104.0 for women.[16]

Statistical Analysis

SPSS 15.0 statistic software was used for the statistical analysis. The Kolmogorov-Smirnov test was used to determine normality of distributions of variables. Continuous variables with normal distribution were presented as mean $\pm$ standard deviation. Median value was used in variables without normal distribution. The qualitative variables were given as percent and the correlation between categorical variables was investigated by the χ^2 test. To compare variables before an AVF creation and after an AVF creation, paired t test (for the parametric variables), Wilcoxon test (for the nonparametric variables), and McNemar test (for categorized variables) were performed. The correlation analysis was evaluated by the Pearson's correlation test for parametric variables and by Spearman's correlation test for nonparametric variables. P value of <0.05 was considered to be significant.

RESULTS

Demographic and clinical characteristics of the patients with ESRD are shown in Table 1. Mean age was 58 $\pm$ 13 years and 21 (%50) of the patients were female. The most known causes of ESRD were diabetes mellitus and hypertension, respectively. Twenty-eight of the 42 patients with ESRD were taking anti-hypertensive treatment before AVF. These anti-hypertensive agents were not modifieted or interrupted and shown in Table 1.

Comparison of biochemical and clinical findings between before and after HD treatment are shown in Table 2. Levels

Table 1 - Demographic and clinical characteristics of patients with end-stage renal disease (n: 42).

Age (year)	58 ± 13
Gender	
Female	21 (50%)
Male	21 (50%)
The cause of end-stage renal disease	
Diabetes mellitus	14 (33.3%)
Hypertension	8 (19%)
Glomerulonephritis	3 (7.2%)
Obstructive uropathy	4 (9.6%)
Amyloidosis	3 (7.2%)
Polycystic kidney disease	1 (2.3%)
Unknown	9 (21.4%)
Presence of diabetes mellitus	15 (35%)
Presence of hypertension	28 (66%)
Smoking	10 (23%)
Body mass index (kg/m^2)	27.3 ± 5.0
Diastolic blood pressure (mm Hg)	83 (60-90)
Systolic blood pressure (mm Hg)	135(90-150)
Medications for hypertension (n)	
Calcium antagonists	21
Beta blockers	6
ACE inhibitors	3
Angiotensin-II receptor blockers	4

of serum hemoglobin, triglyceride, albumin, calcium, and alkaline phosphatase were significantly increased after maintenance HD treatment compared to baseline values. However, levels of low density lipoprotein, blood urea nitrogen, uric acid, and phosphorus and calcium x phosphorus product were significantly decreased after creation of AVF and HD treatment compared to baseline values. There was no significant difference with regard to other parameters including white blood cell count, total cholesterol, high density lipoprotein, serum creatinine, glucose, parathyroid hormone, C-reactive protein, body mass index, blood pressure of diastolic and systolic between before and after maintenance HD treatment.

Table 2 - Comparison of laboratory and clinical findings of the patients before and after creation of AVF.

Parameter	Before AVF	After AVF	p
White blood cell count (mm^3)	7.9 ± 3.4	7.4 ± 2.6	0.351
Hemoglobin (g/dL)	9.8 ± 1.4	11.2 ± 1.3	0.001
Total cholesterol (mg/dL)	172.1 ± 40.9	165.8 ± 44.3	0.307
HDL (mg/dL)	31.5 ± 9.7	32.6 ± 12.3	0.553
LDL (mg/dL)	112.4 ± 29.5	99.2 ± 31.2	0.012
Triglyceride (mg/dL)	139.8 ± 78.3	170.8 ± 79.6	0.008
Blood urea nitrogen (mg/dL)	72.3 ± 32.4	50.3 ± 24.5	0.001
Creatinine (mg/dL)	7.1 ± 3.9	6.1 ± 3.3	0.050
Uric acid (mg/dL)	7.4 ± 1.9	6.2 ± 1.7	0.001
Albumin (g/dL)	2.9 ± 0.7	3.3 ± 0.6	0.003
Calcium (mg/dL)	8.1 ± 0.8	8.8 ± 0.6	0.003
Phosphorus (mg/dL)	5.4 ± 1.7	4.3 ± 1.4	0.001
Calcium×phosphorus (mg^2/dL^2)	48.7 ± 13.7	41.6 ± 12.9	0.004
Glucose (mg/dL)	107(46-264)	102(73-456)	0.218
Alkaline phosphatase (IU/L)	83(40–308)	94(44-585)	0.059
Parathyroid hormone (pg/mL)	185(6-1289)	138(22-1014)	0.383
C-reactive protein (mg/dL)	10(3-148)	8(3-110)	0.737
Body mass index (kg/m^2)	27.3 ± 5.0	27.2 ± 4.9	0.528
Diastolic BP (mm Hg)	83(60-90)	81(60-95)	0.389
Systolic BP (mmHg)	135(90-150)	131(100-150)	0.574

AVF: arteriovenous fistula, HDL: High density lipoprotein, LDL: Low density lipoprotein, BP: Blood pressure

Table 3 - Comparison of M-mode echocardiographic findings of the patients before AVF creation and after AVF creation.

Parameter	Before AVF	After AVF	p
LV end-diastolic diameter (mm)	49.0 ± 4.7	50.6 ± 4.6	0.033
LV end-systolic diameter (mm)	33.8 ± 4.4	35.9 ± 5.1	0.010
LV ejection fraction (%)	59.6 ± 5.6	56.9 ± 5.8	0.023
IVS end-diastolic diameter (mm)	11.0 ± 1.5	11.7 ± 1.9	0.013
PW end-diastolic diameter (mm)	10.5 ± 1.5	11.4 ± 1.8	0.002
Left atrial diameter (mm)	34.9 ± 4.0	36.3 ± 4.1	0.011
LV mass (g)	200.6 ± 56.6	225.6 ± 55.0	0.003
LV mass index (g/m^2)	112.3 ± 29.2	126.0 ± 25.2	0.002
Presence of LV hypertrophy	18 (42%)	30 (71%)	0.003

AVF: arteriovenous fistula, LV: Left ventricular, IVS: Interventricular septum, PW: Posterior wall

The baseline echocardiographic measurements and the changes in these parameters after maintenance HD treatment are presented in Table 3. In the comparison of echocardiographic findings; LVEDD, LVESD, IVSEDD, PWEDD, LA diameter, LVM, LVMI, and presence of LVH were significantly increased, however, EF value was significantly decreased after creation of AVF and HD treatment compared to baseline value.

Table 4 shows comparisons of conventional Doppler echocardiography and DTI findings. In our study, E wave DT was significantly increased after HD treatment compared to baseline value. There was no significant difference with regard to other mitral inflow velocities including E value, A value, E/A ratio, and IVRT. Parameters of DTI such as values of septal and lateral wall Em, Am and Em/Am ratio were not significantly change. There was no significant difference with pulmonary vein flow velocities; PVS, PVd, PVS/d ratio, and PVAr between before and after maintenance HD treatment.

Table 4 - Comparison of Doppler echocardiography and Doppler tissue imaging findings of the patients before and after HD treatment.

	Before HD	After HD	p
E (cm/s)	80.2 ± 26.6	72.5 ± 23.3	0.069
A (cm/s)	82.6 ± 26.0	83.2 ± 25.8	0.881
E/A ratio	1.0 ± 0.4	0.9 ± 0.4	0,228
DT (ms)	141.3 ± 41.4	162.8 ± 40.9	0.002
IVRT (ms)	96.0 ± 19.5	97.0 ± 19.9	0.816
PVS (cm/s)	58.5 ± 12.9	55.3 ± 14.1	0.183
PVd (cm/s)	50.8 ± 15.0	48.1 ± 14.0	0.316
PVS/PVd ratio	1.2 ± 0.3	1.1 ± 0.3	0,862
PVAr (cm/s)	36.9 ± 12.2	38.8 ± 10.6	0.359
Lateral Sm (cm/s)	7.7 ± 2.6	7.2 ± 2.3	0.309
Lateral Em (cm/s)	8.1 ± 3.4	8.0 ± 2.6	0.806
Lateral Am (cm/s)	10.1 ± 3.3	9.3 ± 2.9	0.112
Lateral Em/Am ratio	0.8 ± 0.4	0.9 ± 0.4	0.447
Septal Sm (cm/s)	6.9 ± 1.7	6.3 ± 1.5	0.05
Septal Em (cm/s)	6.6 ± 2.3	6.1 ± 1.9	0.237
Septal Am (cm/s)	8.8 ± 2.6	8.8 ± 2.4	1
Septal Em/Am ratio	0.7 ± 0.3	0.7 ± 0.4	0.708
E/Em ratio	13.8 ± 7.2	12.2 ± 4.5	0.114

HD: hemodialysis, E: peak early diastolic mitral inflow velocity, A: peak late diastolic mitral inflow velocity, DT: deceleration time, IVRT: isovolemic relaxation time, Sm: systolic myocardial velocity, Em: early diastolic myocardial velocity, Am: late diastolic myocardial velocity, PVS: pulmonary vein peak systolic velocity, PVd: pulmonary vein peak diastolic velocity, PVAr: pulmonary vein peak atrial reversal velocity

Table 5 - Inflow velocities measured from tricuspid valve and right ventricle free myocardial wall tissue Doppler velocity changes after maintenance hemodialysis treatment.

	Before HD	After HD	p
Tricuspid E (m/s)	57.4 ± 14.9	54.2 ± 12.3	0.209
Tricuspid A (m/s)	54.5 ± 15.0	57.1 ± 17.9	0.210
Tricuspid E/A ratio	1.1 ± 0.4	1.0 ± 0.3	0.137
RV free wall			
Em	11.7 ± 4.6	10.7 ± 4.2	0.107
Am	16.8 ± 4.3	15.7 ± 4.2	0.197
Em/Am ratio	0.7 ± 0.4	0.7 ± 0.4	0.492
Sm	14.0 ± 3.8	12.8 ± 4.6	0.084

HD: hemodialysis, E: peak early diastolic tricuspid inflow velocity, A: peak late diastolic tricuspid inflow velocity, RV; Right Ventricle, Em: early diastolic myocardial velocity, Am: late diastolic myocardial velocity, Sm: systolic myocardial velocity

Table 5 shows comparisons of RV functions. Tricuspid early (E) and late (A) inflow velocities, E/A ratio and RV free wall myocardial velocities were not significantly change after maintenance HD treatment.

DISCUSSION

In patients with ESRD, it has been suggested that echocardiographic parameters are useful markers for predicting the development of LV dysfunction.[17] However; serial changes of systolic and diastolic functions in ESRD patients with time are not yet well established. In the present study, we have found by conventional and Doppler echocardiography that LV diastolic function and RV functions did not significantly change, however LV systolic function was significantly impaired after starting HD treatment in patients with ESRD.

Patients with ESRD treatment with HD via AVF experience a variety of hemodynamic and metabolic abnormalities that predispose to alterations in LV systolic and diastolic function parameters. Increasing myocardial calcium level, lipid peroxides level, oxidative stres and decreasing antioxidants may affect LV myocardial functions and loading conditions may affect the evaluation of LV functions by echocardiography.[18,19]

The potential acute effect of HD on LV diastolic function has been addressed in several studies. Previous studies demonstrated that an improvement or unchanged or deterioration of LV diastolic functions after HD session.[20-22] In these studies, investigators examined echocardiographic parameters before and immediately after HD session. The acute changes of echocardiographic parameters after HD treatment may be explained by several mechanisms such as the change of serum ionized calcium concentration, sympathetic hyperactivity, increased oxidative stress during hemodialysis treatment and disease of smaller resistance vessels.[21]

Chronic effects of HD on LV diastolic function is unclearly. Studies were reported different results: a significant alterations at LV longitudinal myocardial function parameters assessed by color DTI, improved indices for left ventricular diastolic function and did not change Doppler parameters of mitral inflow and pulmonary venous flow.[23-25] The coexistence of hypervolemia, hypercirculation, LV hypertrophy, and interstitial fibrosis may predispose to LV diastolic dysfunction.

In our study, we evaluate and compare LV functions before and nearly 2½ months after creation of AVF and starting HD treatment. To avoid the acute effect of HD treatment, the second echocardiographic evaluations were performed after 6-8 hours from the last HD session. Although LV systolic function was significantly impaired, diastolic function did not significantly change, except LA diameter, with time after maintenance HD treatment. Indeed we would expect that hemodialysis and AVF cause cardiovascular change and had negative effects on LV systolic and diastolic functions because of methabolic changes, acute volume changes, sympathetic hyperactivity, increased oxidative stress and hyperdynamic state.

During ventricular diastole the LA is directly exposed to LV filling pressure. Therefore, increased LA size and volume may reflect the duration and severity of diastolic dysfunction.[26] Effect of HD on LA dimension could be explained by the factors that influence LV filling. Left atrium pressure increases and resulting in augmented LA dimensions whenever preload increases and/or LV compliance decreases. In patients undergoing HD, the LA parameters reported as a marker of chronic diastolic dysfunction, however some investigators showed that LA parameters were similar in the group of healthy volunteers.[25,27] Another important observation of our study was the mean LA dimension was significantly higher after HD treatment than basaline value. Myocardial velocity by DTI and LA parameters have been proposed as relatively preload independent measurements of diastolic function and more accurately reflects LV diastolic dysfunction.[26,28]

In patients with ESRD, because of the renal anemia, systemic hypertension, volume overload, and the presence of an AVF with high-flow rates, LV systolic and diastolic diameters, wall thickness and cardiac output are increased and indirectly EF is decreased. Several studies have shown that patients with ESRD before and on dialysis had higher LV volumes and dimensions.[29,30] Increases in LVEDD, LVESD, IVSEDD, PWEDD, and LVMI were found after maintenance HD treatment in our study. Furthermore, in our study, 2% decrease in ejection fraction was observed following the HD treatment. Previous studies demonstrated that either an improvement or unchanged of LV systolic function after HD.[22,31] McIntyre et al. showed that HD treatment was associated with significant reductions in myocardial blood flow. Stress–induced myocardial ischemia occurs in the absence of large-vessel epicardial coronary disease and repetitive episodes of ischemia may lead to LV systolic dysfunction.[31] After HD treatment, several factors such as the semi-quantitative assessment of wall motion and altered loading conditions by HD, limit the evaluation of LV systolic function by echocardiography correctly. In our study LV size was only judged by dimension measures and EF determined by the Teichholz equation. It may be misleading because of the loading contidions. Although we reported significant structural changes in LV systolic function, EF of LV before and after HD session were within normal values. So we do not suggest that these findings would be have a biological impact.

In patients with ESRD, anemia leads to a chronic increase in cardiac output and contributes to diastolic dysfunction.[32] Also worse diastolic function in patients on dialysis was associated with increased serum phosphorus and calcium-phosphorus ion product.[33] In our study, after HD treatment,

serum hemoglobin level significantly increased and phosphorus and calcium-phosphorus ion product were meaningfully decreased.

The pathogenesis of hypertension in ESRD patients is multifactorial. Hypervolemia has been considered a major pathogenetic factor and other factors such as a disturbed hormone profile with an activated renin-angiotensin system, increased catecholamine, vasopressin, endothelin, and perhaps decreased nitrous oxide activity seems to play a role in the high incidence of hypertension in ESRD patients[34]. In our study, the systolic and diastolic blood pressure in ESRD patients did not significantly change after maintenance HD treatment. There were twenty-eight patients, who have hypertension, taking antihypertensive agents before HD treatment in our group and we did not change this agents after creation of AVF. Hemodialysis may change metabolic parameters that affect systolic and diastolic blood pressure and hemodialysis and AVF may change volum status in our patients and as a result the systolic and diastolic blood pressure did not significantly change.

After creation of an AVF in patients with ESRD, because of the reduction in peripheral resistance and increase in sympathetic nervous system activity, stroke volume and heart rate, there is a 10–20% increase in cardiac output. The long-term effects of an AVF creation were left ventricular hypertrophy, high-output cardiac failure, myocardial ischemia, and venous stenosis.[35,36]However, the effect of AVFs on cardiac function, remodeling, and long-term mortality remains unclear. Our study design did not allow separation of the effects of an AVF and HD procedures.

Arinc et al. showed that RV systolic and diastolic velocities detected by DTI were not or only minimally affected by preload reduction in hemodialysis patients.[37] Drighil et al. show that both systolic and diastolic DTI velocities of the RV are preload dependent.[38] In our study RV systolic and diastolic functions did not change after maintenance HD treatment. Although we used echocardiography to evaluate RV functions, echocardiography is not the best method to evaluate RV functions, we would not be able to use other imaging methods and also we did not evaluate autonomic regulation.

There were some limitations of the present study. Firstly, we did not evaluate the volume situation of study patients before and after HD treatment with the objective methods (like evaluation of weight gain) and it is possible that some patients on hemodialysis continued to have significant fluid overload even after HD treatment. Secondly, hemodynamic changes associated with AVF and HD treatment but in our study they can not be differentiated. To separate the effect of HD and AVF, some patients can doing HD with another way like permanent catheter. The role of AVF should be investigated in future studies. Thirdly, the results were based on a small number of patients, and our findings wait further validation. Forthly, the observation period was not long enough to explain long term effects but this period is enough to show relatively long term effects of HD (compaired to acute changes).

In conclusion, our data indicated that LV diastolic function and RV systolic and diastolic functions did not change in patients with ESRD after HD treatment. We showed that acute changes of volume status and electrolytes and autonomic regulation by HD session did not affect LV diastolic functions in a relatively long-term. Also we suggest that these results would be same if applying chronic renal failure patients with hypertrophic hearts.

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