Effect of Peritoneal Dialysis Treatment on Left Ventricular Systolic and Diastolic Functions in Patients with End-Stage Renal Disease

Son Dönem Böbrek Yetmezliği Olan Hastalarda Sol Ventrikül Sistolik ve Diyastolik İşlevleri Üzerine Periton Diyalizinin Etkisi

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ABSTRACT

OBJECTIVE: To analyze the effect of peritoneal dialysis (PD) treatment on left ventricular systolic and diastolic function in patients with end-stage renal disease (ESRD).

MATERIAL and METHODS: The study population consisted of 51 patients with ESRD. Before a PD catheter was inserted, the patients were evaluated by echocardiography and Doppler tissue imaging (DTI). Then, a PD catheter was inserted. After 6 months, the second echocardiographic evaluations were performed. Left ventricular systolic and diastolic function parameters were compared.

RESULTS: The mean age was 47 ± 13 years and 38 (74.5%) of the patients were male. No significant difference was found in echocardiographic parameters including ejection fraction, fractional shortening, left ventricular mass, left ventricular mass index, left ventricular posterior wall thickness, inter ventricular septal thickness, left atrial diameter, early diastolic filling/late diastolic filling ratio before and after the period of PD. Left ventricular end-systolic diameter and left ventricular end-diastolic diameter values were significantly lower found in the period after PD.

CONCLUSION: Our findings appear to reflect somewhat the favourable changes in LV diastolic and systolic functions in PD patients.

KEY WORDS: End-stage renal disease, Left ventricular diastolic function, Left ventricular systolic function, Peritoneal dialysis

ÖZ

AMAÇ: Çalışmanın amacı, son dönem böbrek yetmezliği (SDBY) olan hastalarda sol ventrikül sistolik ve diyastolik fonksiyonlar üzerine periton diyalizi (PD) tedavisinin etkisini değerlendirmekti.

GEREÇ ve YÖNTEMLER: Çalışmaya SDBY olan 51 hasta alındı. PD kateteri takılmadan önce, hastalar ekokardiyografi ve doku Doppler görüntüleme (DDG) ile değerlendirildi. Daha sonra PD kateteri takıldı. Altı ay sonra ikinci bir ekokardiyografik inceleme daha yapıldı. Sol ventrikülün sistolik ve diyastolik işlevleri karşılaştırıldı.

BULGULAR: Hastaların ortalama yaşı 47 ± 13 yıldı ve hastaların 38 (%74,5) tanesi erkekti. PD başlamadan önce ve PD başlandıktan 6 ay sonra bakılan ejeksiyon fraksiyonu, fraksiyonel kısalma, sol ventrikül kitlesi, sol ventrikül kitle indeksi, sol ventrikül arka duvar kalınlığı, ventriküller arası septum kalınlığı, sol atrium çapı, erken diyastolik dolum/geç diyastolik dolum oranını içeren ekokardiyografik parametreler açısından anlamlı fark saptanmadı. Sol ventrikül sistol sonu çapı ve sol ventrikül diyastol sonu çapı PD sonrası dönemde anlamlı olarak daha düşüktü.

SONUÇ: Bizim bulgularımız PD hastalarında sol ventrikül diyastolik ve sistolik fonksiyonlarda bir miktar olumlu değişikliğin olduğunu yansıtır gibi görünmektedir.

ANAHTAR SÖZCÜKLER: Son dönem böbrek yetmezliği, Sol ventrikül diyastolik işlevi, Sol ventrikül sistolik işlevi, Periton diyalizi

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INTRODUCTION

Cardiovascular complications are the most important cause of death in patients with end-stage renal disease (ESRD) (1). The most frequent cardiovascular disorders in patients with chronic kidney disease (CKD) are left ventricular hypertrophy (LVH) and left ventricular (LV) systolic and diastolic dysfunction, which are related with increased morbidity (2,3). There are several mechanisms underlying these disorders; uremia itself, hypervolemia, hypertension, anemia, and hyperparathyroidism (4,6). The effect of peritoneal dialysis (PD) on the cardiac abnormalities is not clear yet (7). We aimed to investigate the effect of PD treatment on cardiac functions in patients with ESRD.

MATERIAL and METHODS

This prospective study was performed at the Department of Nephrology of Erciyes University Medical School between December 2008 and May 2010. The study protocol was approved by the local ethics committee. The study procedures were approved by all patients.

This study was performed in 60 patients undergoing PD due to ESRD. None of the patients underwent hemodialysis (HD) before they enrolled in this study. Before a PD catheter was inserted for PD, the patients, who signed an informed consent form before the first echocardiographic evaluation, were evaluated by echocardiography. Then, a PD catheter was inserted into all patients. After 6 months, the second evaluation was performed by echocardiography. Four of the 60 patients died during follow-up. Five patients could not be reached by telephone (loss to follow up). Finally, 51 patients completed the study. Before beginning of PD, some patients could not underwent HD whereas the remaining patients, who had an acute dialysis indication (hyperpotasemia, pulmonary edema, uremic encephalopathy and pericarditis, and metabolic acidosis) could underwent HD by a temporary dialysis catheter. Before initial echocardiographic evaluation, clinical normovolemia was provided in all patients.

The inclusion criteria were the following: new diagnosis of ESRD, sinus rhythm, LV ejection fraction (EF) above 50%, no history of myocardial infarction, and no evidence of valvular disease. The exclusion criteria were the following: myocardial ischemia, coroner artery disease, pericardial disorder, systolic heart failure (i.e.EF of less than 50%), valvular heart disease, cerebrovascular disease, and chronic obstructive pulmonary disease. Body mass index (BMI) was defined as weight in kilograms divided by height in square meters.

The rate of change of variables before PD treatment (baseline) and after PD treatment (post-treatment) (delta, Δ) was calculated by the following formula;

Δ = (pretreatment value – posttreatment value) / (pretreatment value) x 100

Bioelectrical Impedance Analysis

All patients were examined using bioelectrical impedance analysis to estimate the ratio of extracellular water (ECW) to total body water (ECW/TBW). The control group of our previously study was used as the control group for determine the mean ECW/TBW (8). The patients were classified according to volume statuses: hypervolemia (above +2 SD from the mean value), normovolemia (between +2 SD and -2 SD), and hypovolemia (below -2 SD from the mean value) (8).

Echocardiographic Evaluations

The echocardiographic studies were performed in 3 cardiac cycles before and after PD treatment using a Vivid 7 Dimension (General Electric Healthcare Company, Milwaukee, WI, USA) with a 3-MHz transducer in the left lateral position (9). Analysis was performed according to the guidelines of the American Society of Echocardiography recommendations (10).

Left ventricular end-diastolic and end-systolic diameters (LVEDD, LVESD) and end-diastolic interventricular septum and posterior wall thicknesses (IVSEDD, PWEDD) were measured by the M-mode in the parasternal long-axis view (9). EF was calculated according to the Teicholz formula (11). To evaluate the diastolic functions, the mitral inflow velocities were evaluated from the apical 4-chamber view. Pulmonary vein flow velocities were obtained from the right posterior pulmonary vein in the apical view. The left atrium (LA) diameter was measured in the parasternal long-axis view. Right ventricle (RV) early (E) and late (A) ventricular inflow velocities were measured by pulse wave Doppler by placing the sample volume between the tips of the tricuspid valve in the apical 4-chamber window. Diastolic filling was 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 (PVAr) were recorded. The early diastolic velocity of the lateral mitral annulus (Ea), which has been shown to reflect the rate of myocardial relaxation, was recorded with Doppler tissue imaging (DTI) (9). IVSEDD, PWEDD, and internal dimensions were used to calculate LV mass (LVM) using the following equation: LVM = 1.04 x 0.8 [(LV wall thicknesses + internal dimension)-(internal dimension)] + 10.6 g (9). LVH was defined as the LV mass index (LVMI), which was calculated with LV mass in grams divided by the body surface area in square meters, higher than 116.0 for men and 104.0 for women (12).

Blood Samples

Blood samples were taken from all patients for laboratory examinations, including complete blood count, serum glucose, blood urea nitrogen (BUN), serum bicarbonate, arterial pH, creatinine, calcium, phosphorus, alkaline phosphatase, albumin, intact parathyroid hormone (iPTH), and high sensitive C-reactive protein (hs-CRP) levels, and total lipid profile.

All biochemical and bioelectrical impedance analysis evaluations were performed on the same day on which echocardiographic evaluation was performed.

Statistical Analysis

SPSS 16.0 software was used for the statistical analysis. The Kolmogorov-Smirnov test was used for normality analysis of quantitative variables. Continuous variables with normal distribution were presented as mean ± standard deviation. Statistical analysis for the parametric variables was performed by the paired t test. Median value was used where normal distribution was absent. The Wilcoxon signed-rank test was used to compare nonparametric variables. The qualitative data were defined as percentages. The McNemar test was performed for categorized variables. Statistical analysis for the parametric variables was performed by one-way ANOVA with Scheffe's post-hoc test between three groups. The Kruskal-Wallis test was used to compare the nonparametric variables. Then, the Mann-Whitney U-test with Bonferroni correction was used to assess differences among the groups. A P value <0.05 was considered statistically significant.

RESULTS

The mean age of the 51 patients was 47 ± 13 years; 38 (74.5%) of the 51 patients were male. The etiology of ESRD was diabetes mellitus in 20 (39.2%), hypertension in 11 (21.6%), glomerulonephritis in 3 (5.9%), amyloidosis in 3 (5.9%), polycystic kidney disease in 2 (3.9%), obstructive nephropathy in 1 (2.0%), and unknown in 11 (21.6%) patients.

The comparison of clinical and laboratory findings of the patients before and after PD treatment are summarized in Table I. Levels of hemoglobin, serum bicarbonate, corrected

Table I: Comparison of clinical and laboratory findings of the patients before and after PD treatment.

Parameter	Before PD	After PD	p value
Body mass index (kg/m^2)	24.78± 3.57	24.86 ± 4.16	0.729
Systolic blood pressure (mmHg)	135.0 ± 20.1	131.0 ± 22.8	0.275
Diastolic blood pressure (mmHg)	82.8 ± 10.9	84.2± 15.8	0.570
Heart rate (beats/min)	83.4 ± 7.0	82.9 ± 3.5	0.595
ECW/TBW ratio	0.40 ± 0.07	0.37 ± 0.05	0.062
Volume status			0.133
Hypervolemic (%)	16 (31.3)	9 (17.6)	
Normovolemic (%)	34 (66.7)	41(80.4)	
Hypovolemic (%)	1 (2.0)	1 (2.0)	
White blood cell count (mm^3)	8042 ± 254	7430 ± 203	0.066
Hemoglobin (g/dL)	9.82 ± 1.49	11.84 ± 1.83	0.001
Glucose (mg/dL)	98 (45-493)	101 (54-458)	0.249
Blood urea nitrogen (mg/dL)	76.9 ± 30.59	55.5 ± 17.9	0.001
Creatinine (mg/dL)	7.98 ± 8.37	6.76 ± 3.16	0.318
pH value	7.42 ± 0.08	7.45 ± 0.07	0.084
Serum bicarbonate (mEq/L)	21.40 ± 5.14	23.80 ± 4.47	0.017
Corrected calcium (mg/dL)	8.74 ± 0.99	9.30 ± 0.99	0.004
Phosphorus (mg/dL)	5.12 ± 1.55	4.10 ± 1.10	0.001
Corrected calcium x phosphorus (mg^2/dL^2)	44.11 ± 11.87	38.14 ± 10.99	0.016
Alkaline phosphatase (IU/L)	85.0 (4.6-468.0)	51 (4.9-561.0)	0.052
Albumin (g/dL)	3.28 ± 0.63	3.37 ± 0.57	0.421
Intact parathyroid hormone (pg/mL)	206.1 (12.9-1460.0)	303.3 (30.5-742.0)	0.083
High sensitive C-reactive protein (mg/dL)	9.56 (3.0-151.0)	21.85 (5.4-192.2)	0.006
Total cholesterol (mg/dL)	180.4 ± 45.0	209.8 ± 73.6	0.001
Triglyceride (mg/dL)	142.1 ± 54.1	192.2 ± 106.2	0.001
Low-density lipoprotein (mg/dL)	115.1 ± 50.5	137.8 ± 63.2	0.014
High-density lipoprotein (mg/dL)	41.98±13.71	43.50±13.69	0.357

PD: Peritoneal dialysis, **ECW:** Extracellular water, **TBW:** Total body water.

calcium, hsCRP, total cholesterol, triglyceride, and low-density lipoprotein were significantly higher after starting of PD treatment than before PD treatment. On the other hand, levels of BUN and phosphorus, and corrected calcium x phosphorus were significantly lower after starting of PD treatment than before PD treatment. There was no significant difference between the 2 time periods in terms of BMI, systolic and diastolic blood pressures, heart rate, ECW/TBW ratio, volume status, white blood cell count, levels of glucose, creatinine, alkaline phosphatase, albumin, iPTH, and high-density lipoprotein, and pH value.

Table II shows comparison of M-mode echocardiographic findings of the patients before and after PD treatment. LVESD and LVEDD were significantly decreased after PD compared with the baseline values. On the other hand, values of EF, fractional shortening, IVSEDD, PWEDD, left atrial diameter, LVM, and LVMI and presence of LVH were not significantly changed after PD treatment compared with the baseline values.

Table III shows the effects of PD on LV diastolic function parameters. E value and PVS/PVd ratio were significantly decreased after PD compared with the baseline values. There was no significant difference between values after PD and the baseline values in terms of other LV diastolic function parameters including values of A, Em, Am, DT, IVRT, PVS, PVd, PVAr, and PVAr duration, and ratios of E/A, Em/Am, and PVS/PVd.

Table II: Comparison of M-mode echocardiographic findings of the patients before and after PD treatment.

Parameter	Before PD	After PD	p value
LV ejection fraction (%)	61.6 ± 7.8	62.9 ± 10.0	0.373
Fractional shortening (%)	34.15 ± 5.63	35.07 ± 7.61	0.426
LV end-systolic diameter (cm)	3.62 ± 0.66	3.26 ± 0.71	0.001
LV end-diastolic diameter (cm)	5.23 ± 0.71	4.99 ± 0.68	0.029
IVS end-diastolic diameter (cm)	1.23 ± 0.20	1.16 ± 0.24	0.086
PW end-diastolic diameter (cm)	1.16 ± 0.17	1.12 ± 0.20	0.272
Left atrial diameter (mm)	36.1 ± 5.1	36.7 ± 6.2	0.519
LV mass (g)	178.8 ± 57.7	169.6 ± 62.9	0.325
LV mass index (g/m^2)	99.7 ± 30.4	94.9 ± 33.3	0.359
Presence of LV hypertrophy (%)	15 (29.4)	12 (23.5)	0.607

PD: Peritoneal dialysis, **LV:** Left ventricular, **IVS:** Interventricular septum, **PW:** Posterior wall.

Table III: Comparison of Doppler echocardiography and Doppler tissue imaging findings of the patients before and after PD treatment.

Parameter	Before PD	After PD	p value
E (cm/s)	80.4 ± 26.2	66.1 ± 22.7	<0.001
A (cm/s)	90.1 ± 24.5	83.6 ± 19.9	0.075
E/A ratio	0.94 ± 0.37	0.84 ± 0.41	0.175
Em (cm/s)	9 (3-130)	10 (3-150)	0.666
Am (cm/s)	10 (3-180)	12 (3-111)	0.063
Em/Am ratio	0.89 (0.21-4.00)	0.73 (0.05-5.83)	0.102
DT (ms)	107.5 ± 49.1	125.8 ± 55.0	0.051
IVRT (ms)	108.7 ± 27.2	111.9 ± 56.5	0.725
PVS (cm/s)	58.3 ± 22.6	53.8 ± 17.9	0.201
PVd (cm/s)	51.1 ± 14.8	57.1 ± 19.3	0.050
PVS/PVd ratio	1.16 ± 0.36	1.02 ± 0.40	0.044
PVAr (cm/s)	38 (23-123)	42 (22-140)	0.462
PVAr duration (ms)	218.3 ± 50.3	223.0 ± 55.2	0.683

PD: Peritoneal dialysis, **E:** Peak early diastolic mitral inflow velocity, **A:** Peak late diastolic mitral inflow velocity, **Em:** Early diastolic myocardial velocity, **Am:** Late diastolic myocardial velocity, **DT:** Deceleration time, **IVRT:** Isovolemic relaxation time, **PVS:** Pulmonary vein peak systolic velocity, **PVd:** Pulmonary vein peak diastolic velocity, **PVAr**: Pulmonary vein peak atrial reversal velocity.

Table IV: Inflow velocities measured from tricuspid valve and right ventricle free myocardial tissue Doppler velocity changes after PD treatment.

Parameter	Before PD	After PD	p value
Tricuspid E (ms)	53.4 ± 15.7	56.0 ± 23.1	0.479
Tricuspid A (ms)	54.0 ± 18.1	58.5 ± 14.3	0.173
Tricuspid E/A ratio	1.07 ± 0.42	0.99 ± 0.39	0.340
Right ventricular free wall			
Em (cm/s)	11 (4-90)	12 (4-82)	0.155
Am (cm/s)	15 (6-60)	21 (5-117)	0.002
Em/Am ratio	0.63 (0.17-15.00)	0.56 (0.20-3.28)	0.061
Sm (cm/s)	13 (7-99)	14 (6-90)	0.528

PD: Peritoneal dialysis, **E:** Peak early diastolic tricuspid inflow velocity, **A:** Peak late diastolic tricuspid inflow velocity, **Em:** Early diastolic myocardial velocity, **Am:** Late diastolic myocardial velocity, **Sm:** Systolic myocardial velocity.

The comparisons of RV functions are summarized in Table IV. Am value was significantly increased after PD treatment compared with baseline value. However, there was no significant difference between values after PD and the baseline values in terms of other RV function parameters.

Table V shows comparison of demographic, clinical, laboratory and echocardiographic findings of the patients according to left ventricular hypertrophy status (stabile or improved or deteriorated) after PD treatment. There were significant differences among 3 groups in terms of Δ intact parathyroid hormone, Δ LV end-diastolic diameter, and Δ LV mass index. On the other hand, there was no significant difference among the 3 groups with regard to other parameters.

DISCUSSION

It has been suggested that echocardiography is the best useful technique for predicting the development of ventricular dysfunction (13). However, in patients with chronic kidney disease (CKD) treated with PD or HD, serial changes in ventricular functions have not yet been well established. There are very few studies evaluating the effects of dialysis treatment on cardiac function in patients with ESRD. Most of these studies have investigated LVH.

Patients undergoing dialysis have a 10–20-fold increase in cardiovascular mortality compared to the general population (14). Cardiovascular disease accounts for approximately half the deaths among the patient population (15). High risk of cardiovascular death in individuals with CKD is related with several causes. First of all, other major cardiovascular risk factors including dyslipidemia, hypertension, smoking, diabetes mellitus, and impaired LV functions can accompany CKD. In addition, reduction in renal function may be an indicator of advanced vascular damage. Renal failure is also associated with non-traditional risk factors such as inflammation and homocysteinemia. These risk factors may directly contribute to increased cardiovascular mortality (16). In addition, cardiac functions are affected from hemodynamic, toxic, metabolic, and vascular factors in CKD. Factors such as hypervolemia, anemia, hypertension, endothelial dysfunction, renin-angiotensin system are more in the foreground. With the effect of these factors, LV dilatation, reduced EF, increased LVMI, and diastolic dysfunction can develop in patients with CKD.

LVH is one of the most common cardiac abnormalities in patients with CKD. This condition causes ventricular arrhythmias and abnormalities in coronary microcirculation. LVH increases the relative risk of cardiac mortality and all-cause mortality 3.7 times in patients receiving therapy for ESRD (17). In a study performed by Koc et al. it was found that a significantly greater LVMI in CAPD patients with uncontrolled hypertension. This increase in LVMI was explained by the presence of hypervolemia (18). Observational studies have demonstrated that the prevalence of LVH was lower in PD compared to conventional HD (19). Its prevalence according to the Framingham criteria was 68.8% in HD patients and 45.2% in PD patients (19). In the present study, we observed that the prevalence of LVH was 29.4% before starting PD and 23.5% after PD treatment. This probably results from the fact that the number of hypervolemic patients in our study was low. Furthermore, before PD catheter insertion we performed HD in some patients who had hypervolemia symptoms and findings such as pretibial edema, hepatomegaly, tachycardia, dyspnea, orthopnea, and paroxysmal nocturnal dyspnea, until clinical normovolemia was obtained. Thereafter, an initial echocardiographic evaluation was performed. Dialysis treatments (conventional HD three times a week or PD) do not give rise to full regression of LVH in the majority of patients with ESRD (20). Similarly, in the present study, LVMI and the prevalence of LVH decreased somewhat, but full regression of LVH was not obtained.

Table V: Comparison of demographic, clinical, laboratory, and echocardiographic findings of the patients according to left ventricular hypertrophy status (stable or improved or deteriorated) after PD treatment.

	Left ventricular hypertrophy			
	Stabile (n: 36)	**Improved (n: 9)**	**Deteriorated (n: 6)**	**p value**
Age (year)	46 ± 13	47 ± 13	54 ± 17	0.448
Male/female (%)	28 (77.8)/8 (22.2)	7 (77.8)/2 (22.2)	3 (50.0)/3 (50.0)	0.341
Presence of diabetes mellitus	13 (36.1)	5 (55.6)	2 (33.3)	0.538
Smoking	17 (47.2)	6 (66.7)	3 (50.0)	0.579
Changes of volume status				0.300
Stabile (%)	30 (83.3)	6 (66.7)	4 (66.7)	
Improved (%)	5 (13.9)	3 (33.3)	1 (16.7)	
Deteriorated (%)	1 (2.8)	-	1 (16.7)	
Use of RAS blocker (%)	9 (25.0)	4 (44.4)	1 (16.7)	0.414
Use of beta-blocker (%)	10 (27.8)	3 (33.3)	2 (33.3)	0.924
Δ hemoglobin	+20 (-14– +68)	+20 (-9 – +60)	+19 (-4 – +53)	0.877
Δ corrected calcium x phosphorus	-19(-67 – +81)	-8 (-54 – +80)	+10 (-41 – +44)	0.546
Δ albumin	+1.7 (-34 – +92)	+3 (-22 – +15)	+9 (-32 – +61)	0.764
Δ low-density lipoprotein	+12 (-62 – +161)	+42 (-2 – +161)	+18 (-27 – +46)	0.075
Δ intact parathyroid hormone β	+13 (-100 – +5568)	+36 (-63 – +179)	+129 (+50 – +460)	0.035
Δ high sensitive C-reactive protein	-28 (-98 – +890)	0 (-90 – +405)	-3 (-68 – +7)	0.916
Δ LV end-systolic diameter	-4 (-38 – +21)	-25 (-45 – +7)	-5(-35 – +27)	0.071
Δ LV end-diastolic diameter γ	-7 (-29 – +28)	-15 (-24 – +2)	+8 (-15 – +50)	0.021
Δ LV ejection fraction	-1 (-29 – +38)	+15 (-6 – +39)	+9 (-25 – +38)	0.057
Δ systolic blood pressure	0 (-72 – +64)	-7 (-33 – +16)	-6 (-31 – +36)	0.523
Δ diastolic blood pressure	0 (-25 – +63)	0 (-70 – +13)	-6 (-25 – +15)	0.440
Δ ECW/TBW ratio	-4 (-61 – +48)	0 (-28 – +46)	-13 (-38 – +29)	0.134
Δ LV mass index*βγ	-7 (-51 – +55)	-33 (-62 – -9)	+61 (+21 – +127)	<0.001

RAS: Renin-angiotensin-aldosterone system, **LV:** Left ventricular, **ECW:** Extracellular water, **TBW:** Total body water.
* There is a significant difference between the stable group and improved group.
β There is a significant difference between the stable group and deteriorated group.
γ There is a significant difference between the improved group and deteriorated group.

Hypervolemia is an important factor in the pathogenesis of LVH. Strict control of extracellular volume is the highest priority in the management of LVH. In the present study, although not statistically significant, the improvement of volume status was marked in patients who had improvement of LVH. Similarly, Δ ECW/TBW ratio changed negatively in patients who had deterioration of LVH.

Normal LV diastolic function is defined as the normal end-diastolic volume of heart at rest and during exercise not increasing over 12 mmHg for diastolic pressure (21). LV diastolic dysfunction is known to increase mortality (22). As parameters measured by pulse wave Doppler are affected by the preload of the heart, tissue Doppler measurements, which are partially affected by preload, have been used for the diagnosis of diastolic dysfunction (23,24). Diastolic dysfunction, which is identified by both pulse-wave Doppler and tissue Doppler, has been observed in patients receiving dialysis therapy for ESRD in many studies (25,26). When patients with CKD were compared to healthy subjects, a lower mitral E/A ratio, Em/Am ratio

and Em velocity were found (27-29). However investigators reported that LV and RV functions did not change in patients with CKD after long-term HD treatment in a few studies (9). In this study, we observed that LV end-diastolic and end-systolic diameters were significantly lower after PD treatment. This probably results from correction of anemia, uremia, acidosis, hyperphosphatemia, as well as hypervolemia, in which the improvement was not statistically significant.

In this study the mitral E/A ratio was found below 1 both before and after PD treatment (early stage of diastolic dysfunction). There was no significant difference between values after PD and the baseline values in terms of mitral E/A ratio. Systolic functions were also evaluated by EF and fractional shortening and we did not find significant changes in LV systolic functions after PD treatment.

Hypervolemia is one of the causes of diastolic dysfunction and is common in PD patients with high peritoneal permeability. Irrespective of peritoneal permeability in PD patients, deterioration in diastolic functions cannot only be explained by hypervolemia alone. Correlates of diastolic dysfunction are age, diabetes mellitus, hypertension, and LV mass, rather than dialysis adequacy or abnormal endothelial function (30). Many factors including anemia, uremic toxins, hormonal imbalance, hyperparathyroidism, hypercalcemia, amyloid deposition, valvular and pericardial abnormalities, the renin-angiotensin system activation, diabetes mellitus, and hypertension are associated with diastolic dysfunction in patients with CKD. In this study, there was no significant difference between the 2 time periods in terms of ECW/TBW ratio and volume status. However, there were significant improvements in the hemoglobin concentration, acid-base balance, and calcium-phosphorus status. On the other hand, a significant increase in inflammation, which was evaluated by measuring hs-CRP level, was observed after PD treatment.

During ventricular diastole, the LA is directly exposed to LV filling pressure. Left atrium pressure and dimensions increase whenever preload increases or LV compliance decreases (9). Therefore, increased LA size and volume may reflect the duration and severity of diastolic dysfunction (31). In patients with CKD, the LA parameters are reported as a marker of chronic diastolic dysfunction (9), however some investigators have shown that LA parameters were similar in the group of healthy volunteers (32). Another important observation of our study was that the mean LA dimension was not significantly changed after PD treatment.

In patients with CKD, LV volumes and dimensions may decrease or increase or may not change according to the previous studies (33,34). LV dimensions and wall thicknesses may increase because of anemia, hypertension, systemic vascular resistance, volume overload and presence of an AVF in patients with CKD (9). In our study, LVEDD and LVESD were decreased after PD treatment but PWEDD, LVMI and EF did not change after PD treatment.

In conclusion, our findings appear to reflect somewhat the favourable changes in LV diastolic and systolic functions in PD patients.

Limitations

The results were based on a small number of patients, and our findings should be validated with further studies. It is possible that we could not fully control for all confounding variables because of the multifactorial causes of diastolic dysfunction.

REFERENCES

1. Jardine AG, McLaughlin K: Cardiovascular complications of renal disease. Heart 2001; 86:459-466
2. Schärer K, Schmidt KG, Soergel M: Cardiac function and structure in patients with chronic renal failure. Pediatr Nephrol 1999; 13: 951-965
3. Chen SC, Chang JM, Liu WC, Huang JC, Tsai JC, Lin MY, Su HM, Hwang SJ, Chen HC: Echocardiographic parameters are independently associated with increased cardiovascular events in patients with chronic kidney disease. Nephrol Dial Transplant 2012; 27:1064-1070
4. Scharer K, Schmidt KG, Soergel M: Cardiac function and structure in patients with chronic renal failure. Pediatr Nephrol 1999; 13:951-965
5. Mitsnefes MM, Daniels SR, Schwartz SM, Meyer RA, Khoury P, Strife CF: Severe left ventricular hypertrophy in pediatric dialysis: Prevalence and predictors. Pediatr Nephrol 2000; 14:898-902
6. London G: Pathophysiology of cardiovascular damage in the early renal population. Nephrol Dial Transplant 2001; 16:3-6
7. Hüting J, Alpert MA: Progression of left ventricular hypertrophy in end-stage renal disease treated by continuous ambulatory peritoneal dialysis depends on hypertension end hypercirculation. Clin Cardiol 1992; 15:190-196
8. Unal A, Sipahioglu M, Oguz F, Kaya M, Kucuk H, Tokgoz B, Buyukoglan H, Oymak O, Utas C: Pulmonary hypertension in peritoneal dialysis patients: Prevalence and risk factors. Perit Dial Int 2009; 29:191-198
9. Duran M, Unal A, Inanc MT, Kocyigit I, Oguz F, Ocak A, Ozdogru İ, Kasapkara A, Karakaya E, Oymak O: Hemodialysis does not impair ventricular functions over 2 years. Hemodial Int 2011; 15:334-340
10. Devereux RB, Lutas EM, Casale PN, Kligfield P, Eisenberg RR, Hammond IW, Miller DH, Reis G, Alderman MH, Laragh JH: Standardization of M-mode echocardiographic left ventricular anatomic measurements. J Am Coll Cardiol 1984; 4:1222-1230
11. Teichholz LE, Kreulen T, Herman MV, Gorlin R: Problems in echocardiographic volume determinations: Echocardiographic-angiographic correlations in the presence of absence of asynergy. Am J Cardiol 1976; 37:7-11

12. Devereux RB, Wachtell K, Gerdts E, Boman K, Nieminen MS, Papademetriou V, Rokkedal J, Harris K, Aurup P, Dahlöf B: Prognostic significance of left ventricular mass change during treatment of hypertension. JAMA 2004; 292:2350-2356
13. Foley RN, Parfrey PS, Kent GM, Harnett JD, Murray DC, Barre PE: Serial change in echocardiographic parameters and cardiac failure in end-stage renal disease. J Am Soc Nephrol 2000; 11:912-916
14. Foley RN, Parfrey PS, Sarnak MJ: Epidemiology of cardiovascular disease in chronic renal disease. J Am Soc Nephrol 1998; 9(12 Suppl):S16-S23
15. Best PJ, Holmes DR: Chronic kidney disease as a cardiovascular risk factor. Am Heart J 2003; 145:383-386
16. Knight EL, Rimm EB, Pai JK, Rexrode KM, Cannuscio CC, Manson JE, Stampfer MJ, Curhan GC: Kidney dysfunction, inflammation, and coronary events: A prospective study. J Am Soc Nephrol 2004; 15:1897-1903
17. Silberberg JS, Barre PE, Prichard SS, Sniderman AD: Impact of left ventricular hypertrophy on survival in end stage renal failure. Kidney Int 1989; 36:286-290
18. Koç M, Toprak A, Tezcan H, Bihorac A, Akoglu E, Ozener IC: Uncontrolled hypertension due to volume overload contributes to higher left ventricular mass index in CAPD patients. Nephrol Dial Transplant 2002; 17:1661-1666
19. Tian JP, Wang T, Wang H, Cheng LT, Tian XK, Lindholm B, Axelsson J, Du FH: The prevalence of left ventricular hypertrophy in Chinese hemodialysis patients is higher than that in peritoneal dialysis patients. Ren Fail 2008; 30:391-400
20. Glassock RJ, Pecoits-Filho R, Barberato SH: Left ventricular mass in chronic kidney disease and ESRD. Clin J Am Soc Nephrol 2009; 4 (Suppl 1):S79-91
21. Feigenbaum H, Armstrong WF, Ryan T: Evaluation of systolic and diastolic functions of the left ventricle. In: Feigenbaum H, Armstrong WF, Ryan T (eds), Feigenbaum's Echocardiography. 6th ed. Philadelphia: Lippincott Williams & Wilkins, 2005;138-180
22. Foley RN, Parfrey PS, Harnett JD, Kent GM, Murray DC, Barre PE: The prognostic importance of left ventricular geometry in uremic cardiomyopathy. J Am Soc Nephrol 1995; 5:2024-2031
23. Waggoner AD, Bierig SM: Tissue Doppler imaging: A useful echocardiographic method for the cardiac sonographer to assess systolic and diastolic ventricular function. J Am Soc Echocardiogr 2001; 14:1143-1152
24. Oğuzhan A, Arınc H, Abaci A, Topsakal R, Kemal Eryol N, Ozdoğru I, Basar E, Ergin A: Preload dependence of Doppler tissue imaging derived indexes of left ventricular diastolic function. Echocardiography 2005; 22:320-325
25. Kimura H, Takeda K, Tsuruya K, Mukai H, Muto Y, Okuda H, Furusho M, Ueno T, Nakashita S, Miura S, Maeda A, Kondo H: Left ventricular mass index is an independent determinant of diastolic dysfunction in patients on chronic hemodialysis: A tissue Doppler imaging study. Nephron Clin Pract 2011; 117:c67-c73
26. Raizada V, Skipper B, Taylor RA, Luo W, Harford AA, Zager PG, Rohrscheib M, Spalding CT: Left ventricular diastolic function in patients on hemodialysis. J Investig Med 2010; 58:791-795
27. Hacıömeroğlu P, Özkaya O, Günal N, Baysal K: An echocardiographic assessment of cardiac functions and structure in children on dialysis. Renal Failure 2008; 30:147-153
28. Uçar T, Tutar E, Yalçinkaya F, Cakar N, Ozçakar ZB, Atalay S, Uncu N, Kara N, Ekim M: Global left-ventricular function by tissue Doppler imaging in pediatric dialysis patients. Pediatr Nephrol 2008; 23:779-785
29. Çivilibal M, Çalışkan S, Oflaz H, Sever L, Candan C, Canpolat N, Kasapcopur O, Arisoy N: Left ventricular function by 'conventional' and 'Tissue Doppler' echocardiography in paediatric dialysis patients. Nephrology 2009; 14:636-642
30. Fathi R, Isbel N, Haluska B, Case C, Johnson DW, Marwick TH: Correlates of subclinical left ventricular dysfunction in ESRD. Am J Kidney Dis 2003; 41:1016-1025
31. Appleton CP, Galloway JM, Gonzalez MS, Gaballa M, Basnight MA: Estimation of left ventricular filling pressures using two-dimensional and Doppler echocardiography in adult patients with cardiac disease. Additional value of analyzing left atrial size, left atrial ejection fraction and the difference in duration of pulmonary venous and mitral flow velocity at atrial contraction. J Am Coll Cardiol 1993; 22:1972-1982
32. Barberato SH, Mantilla DE, Misocami MA, Gonçalves SM, Bignelli AT, Riella MC, Pecoits-Filho R: Effect of preload reduction by hemodialysis on left atrial volume and echocardiographic Doppler parameters in patients with end-stage renal disease. Am J Cardiol 2004; 94:1208-1210
33. Alpert MA: Cardiac performance and morphology in end-stage renal disease. Am J Med Sci 2003; 325:168-178
34. Meeus F, Kourilsky O, Guerin AP, Gaudry C, Marchais SJ, London GM: Pathophysiology of cardiac disease in hemodialysis patients. Kidney Int Suppl 2000; 76:S140-S147