Clinical Nephrology, Vol. 81 – No. 4/2014 (238-246)

clinical nephrology

Original

ISSN 0301-0430

DOI 10.5414/CN108148
e-pub: January 15, 2014

The effect of strict volume control on cardiac biomarker and arterial stiffness in peritoneal dialysis patients

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Key words
peritoneal dialysis – strict volume – hypertension

Abstract. Introduction: Arterial stiffness is a risk marker for cardiovascular events in peritoneal dialysis (PD) patients. Strict volume control strategy has been shown to result in better cardiac functions and control of hypertension in these patients. The aim of the study was to identify the determinants of arterial stiffness and evaluate the changes in cardiac biomarkers in PD patients under strict volume control strategy. Methods: 58 PD patients were enrolled into this prospective observational study. Arterial stiffness determined by aortic pulse wave velocity (PWV), echocardiography, ambulatory blood pressure and NT-pro-BNP levels were measured at baseline and at first year. Results: The mean age of the patients was 46.4 ± 14 years. 30 patients were on automated PD (APD) and 28 on continuous ambulatory PD (CAPD) group. In both groups, there were significant differences in PWV values at baseline and at the end of the study. A similar decrease was observed with NT-proBNP and PWV levels. In addition, a significant improvement was found in echocardiographic parameters in all patients. Comparison of APD and CAPD groups with respect to change in one year, showed no difference in echocardiographic findings, while the reduction in PWV, NT-proBNP and blood pressure values was higher in the CAPD group. Conclusions: In PD patients, strict volume control leads to a reduction in NT-pro-BNP levels, better control of blood pressure and significant improvements in cardiac functions and arterial stiffness.

Received
July 16, 2013;
accepted in revised form
October 4, 2013

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Introduction

Cardiovascular disease (CVD) remains the leading cause of mortality in end-stage renal disease (ERSD) patients treated with dialysis, accounting for ~ 50% of all deaths in this group of patients [1]. In these patients, cardiovascular mortality is mainly caused by nontraditional risk factors such as inflammation, increased oxidative stress, anemia and altered calcium and phosphorus metabolism as well as traditional risk factors such as hypertension, diabetes, dyslipidemia, and smoking [2, 3].

Arterial stiffness (AS) is one of the most important contributors to this high cardiovascular burden. Both hemodialysis (HD) and peritoneal dialysis (PD) patients have stiffer arteries compared to controls of the same age and blood pressure [4]. The underlying mechanisms of AS in PD patients are unclear, but arterial calcification, activation of the renin-angiotensin-aldosterone system, sympathetic nervous system overactivity, and elevated levels of oxidative stress, inflammation and hypervolemia are believed to play a role [5].

Hypertension is also a well-known contributor to the increased cardiovascular morbidity and mortality in these patients. Several studies have shown that strict volume control strategy may be an effective method for achieving better outcomes including blood pressure in dialysis patients [6, 7]. Additionally, recently Demirci et al. [8] demonstrated that efficient blood pressure control may contribute to preserved or reduced arterial stiffening in PD patients.

Cardiac biomarkers have also been very extensively evaluated in the assessment of CVD. Brain natriuretic peptide (BNP) and N-Terminal-pro BNP (NT-proBNP) are among the biological markers of left ven-

tricular dysfunction that have been associated with CVD [9]. Recent studies suggest that BNP and NT-pro-BNP are predictive for mortality and CV outcomes in dialysis patients [10]. In addition, recently Chen et al. [11] demonstrated that NT-pro-BNP levels are associated with arterial stiffness in renal transplant patients.

Several studies have shown that hypertension, hypervolemia, dyslipidemia, hyperglycemia and hormonal dysfunction are contributing causative factors for increasing AS in PD patients [12, 13, 14]. However, the association between strict volume control with cardiac biomarker and arterial stiffness is not yet clear. Thus, in this prospective study we aimed to determine the effect of strict volume control on arterial stiffness and NT-pro-BNP levels in PD patients and compare the impact of PD type (APD or CAPD).

Methods

Study population

This single-center, prospective, observational study was performed in patients undergoing PD at the Nephrology Department of the Medical Faculty of Erciyes University, Turkey, for a period of 23 months between March 2011 and December 2012. All participants provided written informed consent. The study was approved by the university and local ethics committee and performed in accordance with the Declaration of Helsinki.

In our center, the majority (~ 90%) of dialysis patients are on PD. 224 PD patients are currently being followed. We screened 88 consecutive adult PD patients. Of these, 14 patients preferred not to be enrolled; another 16 patients were excluded because of 14 major cardiovascular diseases as assessed by history and transthoracic echocardiography and 2 had atrial fibrillation. Finally, a total of 58 PD patients were enrolled in this study. CAPD patients were dialyzed using the Baxter Twin Bag (Baxter Healthcare SA, Castlebar, County Mayo, Ireland) and Fresenius A.N.D.Y Plus or Stay-safe systems. (Fresenius Medical Care GmbH, Bad Homburg, Germany). APD patients were treated with Baxter Home-Choice or Fresenius PD Night cyclers.

Biochemical measurements

Ten milliliter blood samples were taken with subjects in a seated position following a 20-minute rest after 12 hours of fasting. Glucose, creatinine, and lipid profiles were determined by standard methods. Tripotassium EDTA based anticoagulated blood samples were drawn to measure complete blood count (Sysmex K-1000 auto analyzer, Block Scientific, USA) within 30 minutes of sampling.

Natriuretic peptide measurement

After a resting period of 20 minutes, 10 mL EDTA blood samples were drawn from antecubital veins for (NT-proBNP) measurement. The specimens were centrifuged at 4 °C at 1,500 rpm for 5 minutes; supernatant plasma was used for NT-proBNP measurement. NT-proBNP levels were measured with a commercially available electrochemiluminescence immunoassay (Elecsys® proBNP assay, Roche Diagnostics Corporation; Indianapolis, IN, USA).

Echocardiography

All participants were examined at inclusion using a Vivid 7 instruments (GE Medical Systems, Milwaukee, WI, USA) with a 2.5-MHz transducer and harmonic imaging. The echocardiographies were performed in the echocardiography laboratory at baseline by a cardiology specialist and according to the recommendations of the American Society of Echocardiography. Echocardiographic examinations were conducted in the left lateral decubitus position using parasternal long-short axis and apical views. At least three consecutive beats in sinus rhythm were recorded, and the average values were taken. The LV end-diastolic and end-systolic dimensions (LVEDD and LVESD), interventricular septal and posterior wall thicknesses (IVSd and LPWd) were measured from M-mode images of the left ventricle generated in the long-axis view with the cursor at the tip of the mitral valve leaflets. The LV ejection fraction was calculated using the formula: LVEF % = (LVEDVLVESV)/LVEDV × 100. The left

ventricular mass (LVM) was calculated using the formula: LVM (in g) = 0.8 {1.04 [(LVDD+PWD+IVSD)^3 – IVSD^3]} + 0.6.

Pulse wave velocity

Vascular studies were performed in a quiet, temperature controlled room with subjects resting in a supine position. Systolic and diastolic blood pressures were measured in duplicate using a semi-automated, non-invasive oscillometric sphygmomanometer, following a 10 minutes rest period. Pulse wave measured in the carotid and femoral arteries using a Pulse wave velocity (PWV) machine (Micro Medical Pulse Trace, Rochester, UK) in accordance with the manufacturer's recommendations. Briefly, the transducers were positioned over the carotid and femoral arteries, always on the right side of the body. PWV was automatically calculated by measuring the time for the pulse wave to travel between the carotid and femoral arteries. All measurements were performed over 15 heart beats by a single operator blinded to the patient's grouping exposure.

Ambulatory blood pressure measurements

The 24-hour blood pressure monitoring was performed using a Del Mar Medical Pressurometer Model P6 (Del Mar Reynolds, Irvine, CA, USA) and the results were assessed using the manufacturer's computer software. Ambulatory measurements were conducted once every 15 minutes from 7 AM until 11 PM, and once every 30 minutes from 11 PM until 7 AM Evaluation was performed taking the mean values of day and night blood pressures into account. Hypertension was considered to be present if the systolic pressure was > 140 mmHg and/or diastolic pressure was > 90 mmHg, or if the individual was taking antihypertensive medication.

Strict volume control strategy

In our center, strict volume control is used for PD patients. Our aim is to maintain blood pressure below 130/80 mmHg and cardiothoracic index below 48%. We recommend patients limit salt intake with diet (NaCl intake is 4 – 5 g/day). At every visit, the patient is questioned about salt intake and physical examination is performed to assess volume status (blood pressure measurement, pretibial edema examination and thoracic auscultation). Patients are allowed to drink as much as their thirst requires, as long as their salt intake is restricted. If required, we increase dose of diuretic agents. We use hypertonic PD solutions or icodextrin in patients with volume overload.

Statistical analysis

Continuous variables were tested for normal distribution by the Kolmogorov-Smirnov test. All continuous parameters either reported data as mean ± standard deviation or median, and compared using Students t-test. All categorical variables are reported as percentages and compared using χ^2-test. Pre-peritoneal dialysis and post-peritoneal dialysis hemodynamic and echocardiographic parameters were compared using the Wilcoxon signed-rank test or the paired Student t-test as appropriate. Pearson correlation coefficients were calculated to examine the degree of association between variables. A p-value < 0.05 was considered as significant, and the confidence interval (CI) was set to 95%. All statistical analyses were performed using SPSS version 15 (SPSS, Inc., Chicago, IL, USA).

Results

58 PD patients were enrolled in the study. The mean age of the patients was 46.4 ± 14 years. 10% were diabetic and 72% were on antihypertensive drugs (20% on diuretics). Only one patient was treated with 3.86 (or 4.25)% glucose containing PD solution; the remaining on 2.27 (or 2.5)% or 1.36 (or 1.5)% glucose PD solution.

There were 30 patients in the APD group and 28 in the CAPD group. The mean age of the patients were 45.2 ± 11 years and 47.6 ± 16 years in the automated APD and CAPD groups, respectively. There were no statistically significant differences between the two groups with respect to age, gender,

Table 1.: Baseline demographical and biochemical features of the study population.

Parameters	Overall (n = 58)	APD group (n = 30)	CAPD group (n = 28)	p
Age (years)	46.4 ± 14	45.2 ± 11	47.6 ± 16	0.53
Gender (F/M)	24/34	13/17	11/17	0.75*
Hemoglobin (g/L)	10.4 ± 1.0	10.5 ± 0.9	10.4 ± 1.1	0.87
Platelet count (×1,000/mm^3)	299 ± 70	295 ± 56	304 ± 83	0.63
White blood cell count (10^3/uL)	7.2 ± 1.6	6.9 ± 1.6	7.5 ± 1.6	0.21
D/P Creatinine	0.76 ± 0.1	0.78 ± 0.1	0.73 ± 0.10	0.06
Plasma fasting glucose (mg/dL)	82.7 ± 12	80.9 ± 12	84.6 ± 12	0.27
Phosphorus (mg/dL)	4.2 ± 0.8	4.1 ± 0.7	4.3 ± 0.9	0.36
Calcium (mg/dL)	8.9 ± 0.5	8.9 ± 0.4	9.0 ± 0.5	0.45
Fasting total cholesterol (mg/dL)	184 ± 36	186 ± 38	182 ± 34	0.66
Fasting LDL cholesterol (mg/dL)	120 ± 29	121 ± 32	119 ± 26	0.77
Fasting triglyceride (mg/dL)	152 ± 46	158 ± 24	150 ± 33	0.41
Hs- CRP (mg/L)	9.9± 3.8	10.7 ± 3.2	9.6 ± 4.4	0.47
Serum albumin level (g/dL)	3.3 ± 0.5	3.4 ± 0.5	3.1 ± 0.4	0.32

*p = χ^2-value.

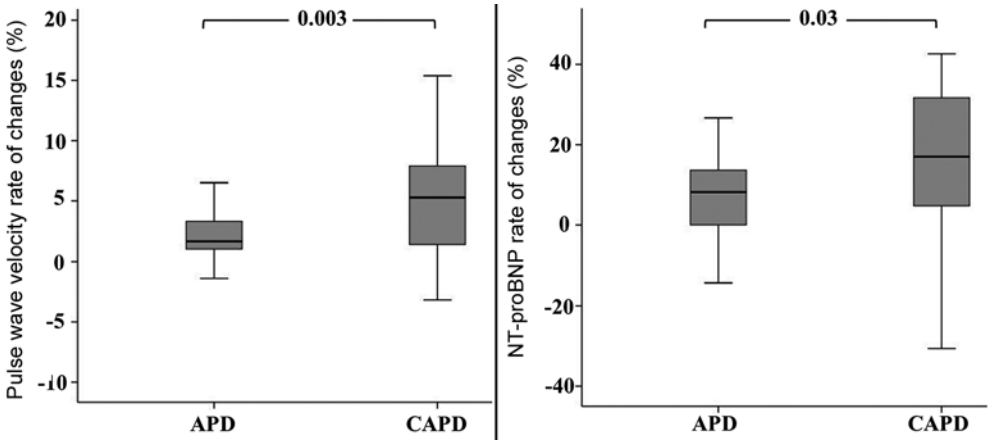

Figure 1. Comparison of PWV values between APD and CAPD group showing the changes according to the basal measurements.

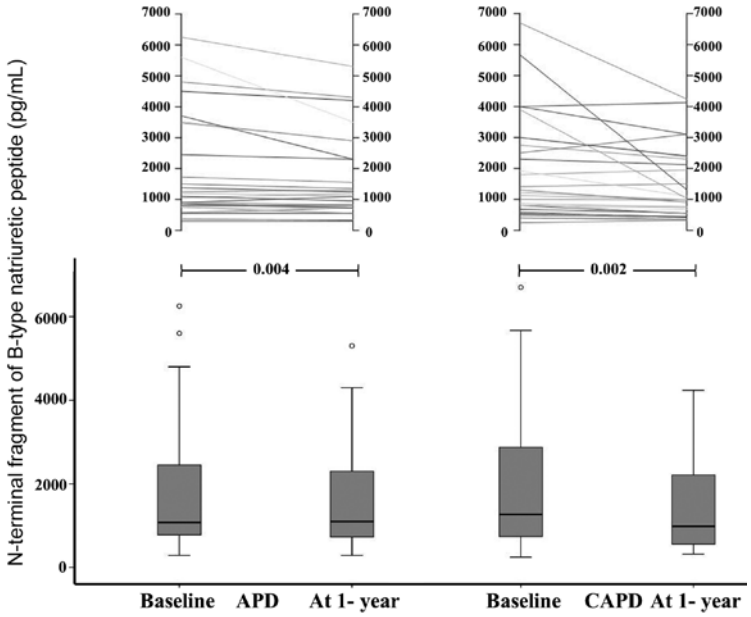

Figure 2. Graph showing the changes of NT-proBNP levels in APD and CAPD groups at baseline and after 1 year.

mean daily residual urinary volume, blood glucose level, cholesterol levels, albumin, C-reactive protein (CRP) levels, and D/P creatinine levels. The demographics of the patients are summarized in Table 1.

In the both groups, there were significant differences in PWV values at baseline and at first year. Similarly, significant decreases were observed in NT-proBNP and PWV levels at the end of the first year in both groups. When the two groups were compared with respect to AS, echocardiographic measurement and NT-pro-BNP levels, significant decreases were seen in NT-pro-BNP (Figure 1) and PWV levels (Figure 2) in both groups at the end of the first year. Regarding echocardiographic parameters, significant reductions were found in IVSD and PWD levels, contrary to a significant increase in LVEF. Additionally, there was a significant decrease in all ambulatory

Table 2. The results of the study in all CAPD patients.

Parameters	All PD patients (n = 58)			
	Baseline	At 1-year	Rate of change, %	p
NT-proBNP (pg/mL)	1,112.5 (245, 6,700)	1,007 (287, 5,300)	11.5	< 0.001[a]
PWV (m/sec)	8.87 ± 1.7	8.53 ± 1.5	3.3	< 0.001
LVSD (mm)	30.8 ± 3	30.3 ± 3	1.2	0.06
LVDD (mm)	47.0 ± 3	46.5 ± 2	1.0	0.007
IVSD (mm)	11.6 ± 2	11.1 ± 1	3.2	< 0.001
PWD (mm)	11.3 ± 2	10.6 ± 1	5.5	< 0.001
LVEF (%)	62.3 ± 6	64.2 ± 5	–3.2	< 0.001
Average 24-h systolic BP (mmHg)	129.4 ± 10	125.9 ± 8.2	2.4	< 0.001
Average daytime systolic BP (mmHg)	136.6 ± 13	132.6 ± 9	2.6	< 0.001
Average nighttime systolic BP (mmHg)	122.2 ± 8	119.3 ± 7	2.2	< 0.001
Average 24-h diastolic BP (mmHg)	90.0 ± 5	84.2 ± 5	6.4	< 0.001
Average daytime diastolic BP (mmHg)	97.4 ± 6	91.5 ± 5	5.9	< 0.001
Average nighttime diastolic BP (mmHg)	82.7 ± 4	76.9 ± 4	6.8	< 0.001
Average 24-h mean BP (mmHg)	103.2 ± 5	98.1 ± 5	4.8	< 0.001
Average daytime mean BP (mmHg)	110.5 ± 6	105.2 ± 5	4.7	< 0.001
Average nighttime mean BP (mmHg)	95.8 ± 4	91.0 ± 5	5.0	< 0.001

NT-proBNP = the N-terminal fragment of B-type natriuretic peptide; PWV = pulse wave velocity; LVSD = Left ventricular end-systolic diameter; LVDD = Left ventricular end-diastolic diameter; IVSD = Interventricular septum diameter; PWD = Posterior Wall diameter; LVEF = Left ventricular ejection fraction. [a]nonparametric analysis.

Table 3. The results of the study in APD and CAPD patients.

Parameters	APD group (n = 30)				CAPD group (n = 28)					
	Baseline	At 1-year	Rate of change, %	p^1	Baseline	At 1-year	Rate of change, %	p^1	p^2	p^3
NT-proBNP (pg/mL)	1,075 (287, 6,250)	1,005 (280, 5,300)	6.4	**0.004**[a]	1,263 (245, 6,700)	984 (320, 4,236)	15.3	**0.002**[a]	0.66[a]	**0.03**[a]
PWV (m/sec)	8.7 ± 1.7	8.5 ± 1.5	1.4	**0.002**	9.0 ± 1.7	8.4 ± 1.4	5.2	**< 0.001**	0.56	**0.003**[a]
LVSD (mm)	30.6 ± 3	30.2 ± 4	0.91	0.38	31.1 ± 3	30.5 ± 2	1.6	0.06	0.59	0.09[a]
LVDD (mm)	46.5 ± 4	46.3 ± 3	0.96	0.47	47.6 ± 2	46.7 ± 3	1.7	0.06	0.23	0.09[a]
IVSD (mm)	11.3 ± 1	11.0 ± 1	2.3	**0.01**	12.0 ± 2	11.3 ± 3	4.2	**0.001**	0.18	0.08[a]
PWD (mm)	11.0 ± 1	10.5 ± 1	3.1	**0.008**	11.7 ± 2	10.7 ± 1	5.2	**0.001**	0.11	0.06[a]
LVEF (%)	62.2 ± 6	63.5 ± 6	–2.4	**0.002**	62.5 ± 5	65.0 ± 4.3	–4.1	**< 0.001**	0.83	0.10[a]
Average 24-h systolic BP (mmHg)	130 ± 10	127 ± 8	2.0	**0.001**	128 ± 13	124 ± 7	2.9	**0.001**	0.46	0.96[a]
Average daytime systolic BP (mmHg)	137 ± 14	134 ± 10	1.8	**0.01**	136 ± 12	131 ± 8	3.4	**0.001**	0.76	0.33[a]
Average nighttime systolic BP (mmHg)	123 ± 8	120 ± 6	2.1	**< 0.001**	120 ± 9	117 ± 7	2.3	**0.002**	0.19	0.57[a]
Average 24-h diastolic BP (mmHg)	90 ± 5	85 ± 4	5.2	**< 0.001**	90 ± 4	83 ± 5	7.6	**< 0.001**	0.93	**0.03**[a]
Average daytime diastolic BP (mmHg)	97 ± 5	92 ± 5	5.2	**< 0.001**	97 ± 6	90 ± 6	6.6	**< 0.001**	0.86	0.42[a]
Average nighttime diastolic BP (mmHg)	82 ± 5	78 ± 4	5.1	**< 0.001**	82 ± 4	75 ± 5	8.6	**< 0.001**	0.96	**0.007**[a]
Average 24-h mean BP (mmHg)	103 ± 5	99 ± 4	3.9	**< 0.001**	102 ± 5	96 ± 5	5.7	**< 0.001**	0.57	**0.02**[a]
Average daytime mean BP (mmHg)	110 ± 6	106 ± 5	4.0	**< 0.001**	110 ± 7	104 ± 5	5.4	**< 0.001**	0.74	0.23[a]
Average nighttime mean BP (mmHg)	96 ± 4	92 ± 4	3.9	**< 0.001**	95 ± 3	89 ± 5	6.1	**< 0.001**	0.41	**0.01**[a]

NT-proBNP = the N-terminal fragment of B-type natriuretic peptide; PWV = pulse wave velocity; LVSD = Left ventricular end-systolic diameter; LVDD = Left ventricular end-diastolic diameter; IVSD = Interventricular septum diameter; PWD = Posterior Wall diameter; LVEF = Left ventricular ejection fraction; p^1 = between baseline and at 1-year variables within study groups; p^2 = between APD group and CAPD group at baseline; p^3 = between APD and CAPD groups rate of changes at 6-month; [a]nonparametric analysis.

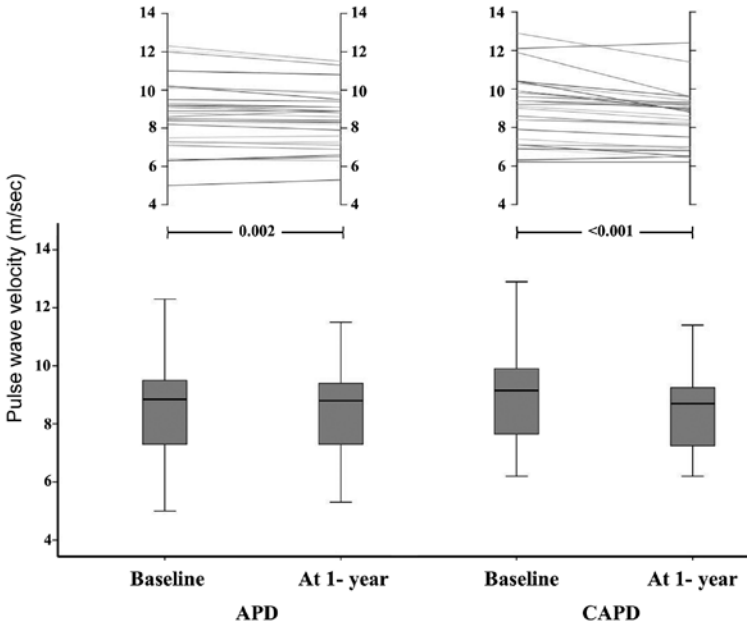

Figure 3. Comparison of rate of changes in NT-proBNP levels and PWV values in both groups.

blood pressure parameters (Table 2, 3). Use of antihypertensive drugs regressed to 24% at the end of the first year, considered to be related to the strict volume control strategy. At the end of the first year, diuretic agents were used in 15% and 5 patients were on 3.86 (or 4.25)% glucose PD solution.

In the CAPD group, significant decreases were observed in NT-proBNP (Figure 1) and PWV levels (Figure 2) at the end of the first year. Regarding echocardiographic findings, significant reductions were found in IVSD and PWD levels, while there was a significant increase in LVEF. In addition, significant reductions were seen in all ambulatory blood pressure parameters (Table 2, 3). Comparison of APD and CAPD groups with respect to changes during the first year period, no difference was found in echocardiographic parameters despite reductions in PWV, NT-proBNP (Figure 3) and higher blood pressure levels in the in the CAPD group (Table 3).

Statistically significant correlations between NT-proBNP, PWV and blood pressure values were also observed (Data not shown).

Discussion

Cardiovascular events are common in PD patients and arterial stiffness is an important contributor to increased CV burden in these patients. In this prospective study, we demonstrated that serum NT-pro-BNP levels decrease by strict volume control strategy in PD patients and echocardiographic findings and arterial stiffness significantly improve in CAPD patients.

Cardiovascular mortality is the most frequent cause of mortality in PD patients. Hypertension, diabetes, obesity and smoking are traditional risk factors in this population. On the other hand, gradual decrease in residual diuresis leads to hypervolemia in PD patients, resulting in increased frequency of cardiovascular events [15]. Moreover, increased glucose load secondary to PD contributes to insulin resistance and development of an atherogenic lipid profile. The presence of glucose degradation products in dialysis solutions, leading to local formation of advanced glycation end products, is also specific to PD [16].

In the recent years, arterial stiffness became an emerging issue in the pathophysiology of CV diseases in both renal disease and non-renal disease populations. Measures of aortic PWV have been shown to be powerful predictors for survival in HD patients [17]. Recently Sipahioglu et al. [18] demonstrated that arterial stiffness is an independent risk predictor of adverse CV outcome in PD patients. Increasing PWV in PD patients is reported to be significantly associated with age, malnutrition, and peritoneal transport status [19, 20]. Additionally, Demirci et al. conducted a study in which blood pressure was the main determinant of AS and AS progression in PD patients [8].

Hypertension is a major risk factor for the increased CV burden rates of PD patients. A multicenter study from Italy showed that 88% of 504 patients on PD were hypertensive [21]. In PD patients, hypertension is largely related with increased volume load. Overhydration, which develops easily in the absence of residual urine volume, is probably the most important cardiovascular risk factor in PD patients [22]. Increases in systolic blood pressure cause elevations in end-systolic stress subsequently leading to cardiac hypertrophy and higher myocardial oxygen requirement. The NECOSAD study showed that for PD patients, increased systolic blood pressure was related with increased mortality rate [23]. This was con-

firmed by another study from the UK [24]. Therefore, overhydration should be avoided in such patients. Strict volume control strategy results in better cardiac functions and control of hypertension in PD patients. Gunal et al. [25] conducted a study in 78 patients undergoing PD and reported significant improvements in blood pressure levels with strict volume control strategy despite no antihypertensive therapy use. In addition, Asci et al. [6] found significant decreases in blood pressure levels and left ventricular hypertrophy by strict volume control in 56 PD patients. Furthermore, Kircelli et al. [26] reported that strict volume control strategy decreased mortality rate and improved technical survival in PD patients.

In our center, we apply strict volume control strategy in all eligible PD patients. The aim is to keep blood pressure below 130/80 mmHg and the cardio-thoracic index below 48%. In the current study, strict volume control was performed for PD patients and a significant improvement was observed in ambulatory blood pressure monitoring and echocardiographic findings of patients within a period of 1 year. It is known that PWV predicts cardiovascular events in PD patients [27]. In the current study, significant reductions were achieved in PWV values by strict volume and blood pressure control strategy in both CAPD and APD groups. Our result are in line with the study of Demirci et al. [8] who reported that increased PWV is a predictor of increased blood pressure in PD patients. They suggested that successful control of blood pressure (mean SBP and DBP of 121 ± 24 and 77 ± 14 mm/Hg, respectively), and risk of progression of AS by 12.4% for every 1 mmHg increment in mean arterial pressure.

In the current study, it is clearly demonstrated that the results are better in the CAPD group with respect to changes not only in blood pressure values but also in echocardiographic findings and PWV. Although the underlying cause of the findings is not clearly understood, Demirci et al. [8] found higher PWV in the APD group. This result can be explained with the fact that ultrafiltration by using APD may be relatively lower than CAPD patients because of dry abdomen during the day and higher peritoneal transport characteristics in patients with APD group.

Ultrafiltration failure occurs in about one-third of PD patients and may easily lead to hypertension and overhydration. This overhydration leads to increased atrial dilatation, left ventricular hypertrophy and myocardial release of natriuretic peptides [28]. B-type natriuretic peptides are synthesized by cardiac myocytes to counteract increased ventricular wall stress. They are secreted in the form of prohormone and are biologically divided into active hormone (BNP) and NT-proBNP, which contains inactive N-terminal [29].

Studies indicate that BNP levels are higher in patients with renal failure in comparison with those without renal failure [30]. NT-pro-BNP levels are found to be related to left ventricular hypertrophy in patients who undergo PD [31]. In the current study, NT-pro-BNP levels significantly decreased with strict volume control strategy in PD patients at the end of the first year period. This reduction was more remarkable in the CAPD group, and the finding indicate a relation to volume control strategy. The relation between NT-pro-BNP levels and arterial stiffness is not clear. It is believed that NT-proBNP is associated with AS since it is known that hypervolemia is related to arterial stiffness under ordinary conditions. The study conducted by Caliskan et al. [32] found no relation between NT-proBNP levels and PWV, while Chen et al. [11] determined a significant relation in renal transplant patients. In the current study, NT-proBNP levels appear to be related to blood pressure and PWV and this finding can be explained with hypervolemia-associated blood pressure and increased stiffness.

There are some limitations to the current study. The volume condition of patients was not assessed with bio-impedance spectroscopy and urinary salt excretion was not addressed. Such measurements could not be done due to technical drawbacks although they were planned at baseline. We used routine control indicators such as blood pressure values, findings of physical examination (pretibial edema, pulmonary auscultation findings etc.) and lung X-ray (cardiothoracic index). Nevertheless, the reductions in NT-proBNP levels and the correlation of echocardiography and stiffness findings indicate that we followed a good volume control strategy for patients.

In conclusion, for PD patients, strict volume control led to reductions in NT-pro-BNP levels, better controlled blood pressure values and significant improvements in cardiac functions and arterial stiffness. Findings were more remarkable in CAPD patients and randomized studies are needed to clarify this issue.

Conflict of interest

The authors declare no conflict of interest.

Funding

No funding has been received for this study.

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