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Evaluation of arterial stiffness between peritoneal dialysis and hemodialysis in patients with renal replacement therapy

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ABSTRACT

Objectives: The aortic stiffness index beta (ASI-β), calculated non-invasively with the pressure change caused by arterial strain and volume changes on echocardiography, shows a strong correlation with invasive measurements of arterial stiffness. The aim of this study was to compare arterial stiffness and distensibility between peritoneal dialysis and hemodialysis and patients in renal replacement therapy (RRT).

Methods: 108 consecutive patients under RRT (peritoneal dialysis and hemodialysis) were analyzed in cross-sectional and observational study design. The aortic stiffness index beta (ASI-β) was calculated for each group.

Results: The mean age of the patients in the study was 58.2 ± 11.1 years, and 49 (45.4%) of the patients were female and 59 (54.6%) were male. Age, gender, comorbid rates, and levels of blood pressure and heart rate did not differ between the peritoneal dialysis and hemodialysis groups. Blood pressure levels and heart rate. Mean aortic strain (5.6 ± 1.9 vs. 9.4 ± 2.8, $p < 0.001$) and median distensibility (1.5 vs. 2.9 cm, $p < 0.001$) were lower in the peritoneal dialysis group than the hemodialysis group, while median ASI-β (11.6 vs. 6.2, $p < 0.001$) and mean E/e' (10.6 ± 2.9 vs. 9.2 ± 2.3, $p = 0.006$) were higher in the peritoneal dialysis group. The rate of concentric hypertrophy was higher in the peritoneal dialysis group (47.5% vs. 23.5%, $p = 0.005$).

Conclusions: Peritoneal dialysis patients have higher arterial stiffness and lower distensibility levels compared to hemodialysis patients. Therefore, patients with peritoneal dialysis may be more prone to diastolic dysfunction, cardiovascular disease, and events.

Keywords: Aortic stiffness index, renal replacement therapy, cardiovascular disease, peritoneal dialysis, hemodialysis

Patients with end-stage renal disease (ESRD) are predisposed to cardiovascular disease such as left ventricular hypertrophy, heart failure, and acute myocardial infarction [1]. Traditional cardiovascular risk factors like advanced age, diabetes mellitus, smoking, hypertension, dyslipidemia, ventricular hypertrophy, and physical inactivity are common in these patients. ESRD also increases the risk of developing coronary heart disease. Moreover, accelerated atherosclerosis may contribute to the pathophysiology of ESRD [2].

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Arterial stiffness is an important predictor of atherosclerosis, cardiovascular diseases and events [3]. Considered the gold standard, pulse wave velocity (PWV) is a non-invasive, reproducible and simple method of assessing arterial stiffness. [4]. It has been reported that arterial stiffness is increased in patients with renal failure [5]. The type of dialysis has different effects on the cardiovascular system. Peritoneal dialysis (PD) may accelerate atherosclerosis and increase the risk of cardiovascular disease or events because it is associated with the accumulation of excess volume in the body [6]. Previous studies have provided conflicting results that arterial stiffness measured by PWC is similar, low, or high in PD and hemodialysis (HD) patients [7]. There is therefore a need for further research into the impacts of the dialysis type used on arterial stiffness.

The aortic stiffness index beta (ASI-β), calculated in a non-invasive way with the change in pressure caused by arterial strain and volume changes on echocardiography, is highly correlated with invasive arterial stiffness measures [8]. Increased ASI-β has been demonstrated to be an indicator of cardiovascular morbidity and mortality in patients with PD. [9]. The aim of this study was to compare arterial stiffness and distensibility between HD and PD and patients on renal replacement therapy.

METHODS

Study Population

The present study analyzed a total of 108 patients who were receiving renal replacement therapy at the Nephrology Clinic of a tertiary referral hospital and who presented to the Cardiology Clinic for various reasons were evaluated in cross-sectional design. The patients with previous coronary heart disease, myocardial infarction, heart failure, rheumatic diseases, asthma, pulmonary embolism, inflammatory disease, peripheral artery disease, chronic obstructive pulmonary disease, cerebrovascular disease, liver diseases and cancer were excluded from the study.

Informed consent was obtained from the subjects and the study protocol was approved by the local ethics committee of the institute (Decision No: 2022-17/9). The study protocol conforms to the tenets of the Declaration of Helsinki.

Demographic, laboratory and echocardiographic data were collected from all patients. Hypertension was defined as blood pressure > 140/90 mmHg in repeated measurements or use of antihypertensive medication, and diabetes mellitus was defined as fasting plasma glucose ≥ 126 mg/dL or use of antidiabetic medication.

Laboratory Measurements

Blood samples were taken from all patients in the morning after an overnight fast. The levels of hemoglobin (photometrically), platelets (impedance method), highly sensitive C-reactive protein (hs-CRP) (immunoturbidimetric method) were determined. Lipid panels [triglycerides, total cholesterol (enzymatic colorimetric method), high-density lipoprotein (HDL) (homogeneous enzymatic colorimetric method)] and complete blood counts were measured using a Beckman Coulter LH 780 device (Mervue, Galway, Ireland). Low-density lipoprotein (LDL) levels were calculated using the Friedewald formula.

Echocardiography Measurements

Echocardiographic data were obtained by an experienced cardiologist using the Vivid S5-dimensional cardiovascular ultrasound system (General Electric Vingmed, Horten, Norway). Standard American Society of Echocardiography guidelines for images and techniques were followed. ASI-β was used as a substitute indicator of arterial stiffness and calculated in the following way:

$$ASI\text{-}\beta = \ln(SBP/DBP)/[(Asd-Add)/Add]$$

(ASI-β = Aortic Stiffness Index beta, ln = Logarithm Natural BP, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, Asd = Asending Aorta Systolic Diameter, Add = Asending Aorta Diastolic Diameter)

Statistical Analysis

The Kolmogorov-Smirnov test was used to assess the normality of the numerical data. According to the distribution pattern, continuous variables were presented as mean ± standard deviation or median with quartiles (Q1-Q3), and Student's T-test or Mann-Whitney U-test was used for comparisons between groups. Categorical variables were expressed as numbers and percentages, and comparisons between groups were

assessed using chi-squared and Fisher exact tests. Pearson and Spearman correlation analyses were used for the relationship between ASI-β and numerical data. A p value < 0.05 was considered to be statistically significant. All statistical analyses were carried out using IBM SPSS Statistics for Windows 20.0 (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 108 consecutive patients were analyzed in the study, including 49 (45.4%) females and 59 (54.6%) males with the mean age of 58.2 ± 11.1 years. Demographic characteristics did not differ between the PD and HD groups. The rate of spironolactone user was higher in the PD group, for other drug users, there were no significant differences between the groups. Blood pressure levels and heart rate were similar between groups. The detailed demographic and clinical data of the patients were presented in Table 1.

Mean LDL (158.1 ± 19.8 vs. 147.5 ± 25.5 mg/dL, p = 0.026) and median triglyceride (220 vs 1975 mg/dL, p = 0.045) was higher in PD group than the HD group. Other laboratory results did not differ significantly among the groups (Table 2).

Table 1. Distribution of demographic and clinic findings of study populations

Variables	Peritoneal dialysis n = 40	Hemodialysis n = 68	*p value*
Demographic findings			
Age (years)	59.8 ± 11.3	58.7 ± 10.9	0.618
Male gender, n (%)	22 (55.0)	37 (54.4)	0.999
BSA (m^2)	1.8 ± 0.1	1.9 ± 0.2	0.200
BMI (kg/m^2)	24.9 ± 3.0	25.0 ± 3.6	0.842
Smoking, n (%)	22 (55.0)	45 (66.2)	0.306
Hypertension, n (%)	28 (70.0)	43 (63.2)	0.533
Diabetes mellitus, n (%)	10 (25.0)	17 (25.0)	0.999
Hyperlipidemia, n (%)	25 (62.5)	50 (73.5)	0.281
Use of drugs, n (%)			
Beta blocker	18 (45.0)	21 (30.9)	0.152
ACEi	17 (42.5)	23 (33.8)	0.413
CCB	25 (62.5)	33 (48.5)	0.169
Spironolactone	15 (37.5)	9 (13.2)	0.007*
Alpha blocker	4 (10.0)	1 (1.5)	0.118
Number of drugs	2 (0-3)	1 (0-2)	0.957
Duration of CRF, months	46 (29-74)	45 (30-66)	0.573
Clinic finding			
Heart rate (bpm)	73.4 ± 3.6	73.2 ± 3.5	0.741
Systolic BP (mm Hg)	146.5 ± 25.2	143.5 ± 22	0.523
Diastolic BP (mm Hg)	79.1 ± 12.3	81.4 ± 12.6	0.356
Pulse pressure (mm Hg)	67.4 ± 21.2	62.1 ± 16.7	0.154
MAP (mm Hg)	101.6 ± 14.6	102.1 ± 14.4	0.848

Numerical variables were shown as mean ± standard deviation or median (Q1-Q3). Categorical variables were shown as number (%). BSA = body surface area, BMI = body mass index, ACEi = angiotensin converting enzyme inhibitor, CCB = calcium channel blockers, BP = blood pressure, CRF = chronic renal failure, MAP = mean arterial pressure

Table 2. Laboratory findings of study populations

Variables	Peritoneal dialysis n = 40	Hemodialysis n = 68	*p value*
Hemoglobin (g/dL)	13.3 ± 1.4	13.3 ± 1.2	0.796
Leukocyte ($\times 10^3/\mu L$)	5.8 ± 1.5	6.1 ± 2.1	0.300
Neutrophil ($\times 10^3/\mu L$)	3.8 ± 0.7	3.9 ± 1.0	0.509
Platelet ($\times 10^3/\mu L$)	319.3 ± 77.5	312.4 ± 85.1	0.678
HbA1C (%)	5.7 ± 0.8	5.6 ± 0.6	0.508
Glucose (mg/dL)	109 (92-150)	98 (87-121)	0.103
Sodium (mmol/L)	139.2 ± 7.2	136.0 ± 13.5	0.181
Potassium (mmol/L)	4.6 ± 0.4	4.4 ± 0.5	0.173
Calcium (mg/dL)	8.6 ± 0.2	8.7 ± 0.2	0.074
Phosphorus (mmol/L)	4. ± 1.1	4.7 ± 1.2	0.404
Albumin (g/dL)	39.3 ± 6.6	38.4 ± 6.2	0.471
Cholesterol (mg/dL)	252.7 ± 24.3	243.0 ± 30.9	0.092
LDL (mg/dL)	158.1 ± 19.8	147.5 ± 25.5	0.026*
HDL (mg/dL)	40.5 ± 11.0	44.7 ± 14.0	0.103
Triglyceride, mg/dL	220(180-263)	197 (160-236)	0.045*
Hs-CRP (mg/dL)	5 (1.3-9.0)	3 (1.5-7.6)	0.377
Ferritin (ml/ng)	649.2 ± 200.3	705.2 ± 203.7	0.168
Parathyroid hormone (pg/mL)	312 (222-367)	240 (130-345)	0.720

Numerical variables were shown as mean ± standard deviation or median (Q1-Q3). HbA1C = hemoglobin A1C, LDL = low-density lipoprotein, HDL = high-density lipoprotein, Hs-CRP = high-Sensitivity C-Reactive Protein

Mean left ventricle (LV) end diastolic volume, mean LV end systolic volume, mean LV diastolic diameter and mean LV systolic diameter levels were lower in the PD group, while mean interventricular septum diameter and mean posterior wall diameter levels was higher. Mean aortic strain (5.6 ± 1.9 vs. 9.4 ± 2.8, $p < 0.001$) and median distensibility (1.5 vs. 2.9 cm, $p < 0.001$) were lower in the PD group than the HD group, while median ASI-β (11.6 vs. 6.2, $p < 0.001$) and mean E/e' (10.6 ± 2.9 vs. 9.2 ± 2.3, $p = 0.006$) were higher in the PD group. The rate of concentric hypertrophy was higher in the PD group (47.5% vs. 23.5%, $p = 0.005$) (Table 3).

DISCUSSION

This study of dialysis patients without known CAD has demonstrated that ASI-β levels, reflecting arterial stiffness, were significantly higher in patients with PD compared to patients with HD. This also provided new insights, as this is the first study comparing ASI-β as a surrogate of arterial stiffness in patients with PD and HD. ASI-β offer different implications that may allow it to be used as a potential screening tool for high-risk dialysis patients at risk for cardiovascular disease and events.

Approximately 40% of dialysis patients have cardiovascular disease prior to dialysis, and CAD accounts for the majority of hospitalizations for cardiovascular reasons in these patients [10]. An accelerated atherosclerosis process caused by mechanisms such as increased inflammation, oxidative stress, sympathetic activation, and endothelial dysfunction associated with ESRD plays a role in the pathophysiology [2]. Arterial stiffness, an important predictor of both atherosclerosis and cardiovascular events, physiologically increases with age and in-

Table 3. Echocardiography measurements in haemodialysis and peritoneal dialysis patients

Variables	Peritoneal dialysis n = 40	Hemodialysis n = 68	*p value*
LVEF (%)	57.2 ± 3.4	56.6 ± 5.4	0.553
sPAB (mm Hg)	32.8 ± 5.6	32.3 ± 8.4	0.876
LV EDV (mL/m^2)	57.0 ± 8.8	62.8 ± 9.5	0.002*
LV ESV (mL/m^2)	24.4 ± 4.2	27.2 ± 5.4	0.006*
LVDD (mm)	45.9 ± 1.7	47.7 ± 2.8	< 0.001*
LVSD (mm)	25.4 ± 3.1	27.1 ± 3.3	0.009*
IVSD (mm)	11.3 ± 1.1	10.5 ± 1.0	0.001*
PWD (mm)	10.6 ± 1.1	9.8 ± 1.1	0.001*
Aortic strain (×10^2)	5.6 ± 1.9	9.4 ± 2.8	< 0.001*
Distensibility (cm)	1.5 (1.3-2.0)	2.9 (2.5-3.5)	< 0.001*
ASI-β	11.6 (9.1-14.7)	6.2 (5.0-7.3)	< 0.001*
RWT (mm)	0.48 ± 0,04	0.42 ± 0.03	< 0.001*
LV mass index (g/m^2)	106.4 ± 14.5	93.0 ± 19.3	< 0.001*
LAVI (mL/m^2)	32.3 ± 4.5	32.0 ± 4.3	0.708
LV diastolic function			
Peak E velocity (cm/sec)	87.8 ± 18.3	85.4 ± 15.6	0.462
Peak A velocity (cm/sec)	66.2 ± 15.6	68.0 ± 14.1	0.521
E/A	1.4 (1.3-1.7)	1.4 (1.3-1.6)	0.174
DT, ms	180.9 ± 41.8	193.2 ± 49.8	0.192
Peak e' (cm/s)	8.5 ± 1.8	9.6 ± 2.1	0.005*
Peak a' (cm/s)	12.2 ± 2.6	11.4 ± 2.6	0.128
S'	10.2 ± 1.8	10.6 ± 2.1	0.292
E/e'	10.6 ± 2.9	9.2 ± 2.3	0.006*
LV remodeling, n (%)			
Normal geometry	8 (20.0)	32 (47.1)	0.005*
Concentric remodeling	13 (32.5)	16 (23.5)	
Eccentric hypertrophy	0	4 (5.9)	
Concentric hypertrophy	19 (47.5)	16 (23.5)	

Numerical variables were shown as mean ± standard deviation or median (Q1-Q3). Categorical variables were shown as number (%). LVEF = Left ventricle ejection fraction, sPAB = systolic pulmonary artery pressure, LVEDV = left ventricle end-diastolic volume, LVESV = left ventricle end-systolic volume, LVDD = left ventricle diastolic diameter; LVSD = left ventricle systolic diameter, IVSD = interventricular septum diameter; PWD = posterior wall diameter, ASI = aortic stiffness index, RWT = relative wall thickness, LAVI = left atrial volume index, DT = deceleration time

travascular pressure [8]. On the other hand, arterial stiffness is affected by the course of diseases like diabetes mellitus, hypertension, and ESRD, which plays a role in accelerating atherosclerosis [11]. However, the question of which type of dialysis leads to atherosclerosis or cardiovascular disease remains controversial. Previous epidemiological studies reported that the rates of hypertension and diabetes mellitus were similar in patients with HD and PD, whereas rate of CAD was lower in patients with PD and interestingly, PD

patients were associated with increased mortality [12, 13]. In contrast, a recent epidemiological study by Sun *et al*. [14] matched PD and HD patients for age, sex, duration of dialysis treatment, and comorbid conditions, and showed that PD was associated with lower cardiovascular events. These matching parameters play an important role in the acceleration of atherosclerosis, including arterial stiffness [15]. In the current study, there was no significant difference in dialysis duration, as well as demographic and comorbid characteristics between PD and HD patients, but susceptibility to atherosclerosis trended toward PD.

The inconsistency observed in the epidemiological studies mentioned above was also present in the conflicting results of previous studies evaluating arterial stiffness in PD and HD patients [15-18]. In a study measuring PWV in vivo and epigastric artery stiffness in vitro, arterial stiffness was not significantly different in PD and HD patients [16]. In another study, PWV values measured in both the carotid-radial and dorsalis pedis arteries were not significantly different between PD and HD patients. [15]. Similar results were reported in another study of PD and HD patients with similar age and comorbid conditions [17]. In contrast to these studies, increased carotid-femoral PWV has been shown to be associated with increased vascular endothelial dysfunction in PD patients [18]. Another study reported that arterial stiffness measured by brachial- ankle PWC was higher in HD patients [19]. This was supported by the results of a study of serial measurement of brachial- ankle PWV [20]. These inconsistencies between studies may be related to differences in dialysis treatment times between groups, different measurement methods such as carotid-femoral PWV, carotid-radial PWV, OR brachial-ankle PWV, or high measurement values of diagnostic devices [21].

In the current study, ASI-β levels were higher than the range of 4.2-5.3 previously reported only in PD patients [9, 22]. This may be related to the lower mean age range of previous studies. Compared to HD patients, PD patients had higher levels of ASI-β. The type of hemodialysis may contribute to the atherosclerotic process by having different effects on the cardiovascular system. In the early years of treatment, PD, which offers advantages such as hemodynamic stability, continuous ultrafiltration, and the absence of an arteriovenous fistula, may allow prolonged maintenance of residual renal function and diuresis and better fluid and blood pressure control [23]. This is consistent with study results showing no change in serial PWV measurements in PD patients over a 1-year follow-up [24, 25]. In the following periods, with the decrease in peritoneal ultrafiltration capacity and residual diuresis, hypervolemia may develop and blood pressure control may deteriorate [23]. In addition, the presence of higher LDL and triglyceride levels in patients with PD may lead to an exaggerated inflammatory response due to the accumulation of atherogenic lipids and uremic toxins associated with the biocompatibility of the peritoneal fluid [18]. This may lead to an acceleration of atherosclerosis and an increase in the risk of cardiovascular disease or events. [17].

The higher rate of LV concentric hypertrophy of PD patients was consistent with previous studies [26, 27]. LV concentric hypertrophy associated with increased arterial stiffness [28], has been associated with fluid overload in PD [29]. It has been suggested that higher LV myocardial index in PD patients may indicate chronic hypervolemia, which may impair arterial distensibility function [30]. Consistent with this hypothesis, PD patients had low distensibility levels. Volume status, blood pressure, and arterial stiffness/distensibility affect afterload and preload and decrease coronary perfusion. This may lead to diastolic dysfunction [31]. The fact that the E/e' ratio, which is an indicator of diastolic dysfunction, was higher in PD patients supported the susceptibility of these patients to diastolic dysfunction.

Limitations

The main limitation of this study was the small sample size and the single-center design of the study. Another important limitation was the lack of serial measurement parameters, especially arterial stiffness. This may limit the prognostic significance of changes in arterial stiffness in pre-dialysis patients for cardiovascular risk assessment.

CONCLUSION

PD patients have higher arterial stiffness and lower

distensibility levels compared to HD patients. Therefore, patients with PD may be more prone to diastolic dysfunction, cardiovascular disease, and events

Authors' Contribution

Study Conception: TG, DT, SA; Study Design: TG, DT, SA; Supervision: TG, DT; Funding: TG, SA; Materials: TG, SA; Data Collection and/or Processing: TG, DT; Statistical Analysis and/or Data Interpretation: TG, DT; Literature Review: TG, SA; Manuscript Preparation: TG, DT, SA and Critical Review: TG, DT, SA.

Conflict of interest

The author disclosed no conflict of interest during the preparation or publication of this manuscript.

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REFERENCES

1. Aoki J, Ikari Y. Cardiovascular disease in patients with end-stage renal disease on hemodialysis. Ann Vasc Dis 2017;10:327-37.
2. Carracedo J, Alique M, Vida C, Bodega G, Ceprian N, Morales e, et al. Mechanisms of cardiovascular disorders in patients with chronic kidney disease: a process related to accelerated senescence. Front Cell Dev Biol 2020;8:185.
3. Garnier AS, Briet M. Arterial stiffness and chronic kidney disease. Pulse (Basel) 2016;3:229-41.
4. Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D, et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications. Eur Heart J 2006;27:2588-605.
5. Olczyk P, Malyszczak A, Golebiowski T, Letachowicz K, Szymcak A, Mazanowska O, et al. Arterial stiffness assessed by oscillometric method in kidney transplant, predialysis, and dialysis patients. Transplant Proc 2020;52:2337-40.
6. Kocyigit I, Sipahioglu MH, Orscelik O, Unal A, Celik A, Abbas SR, et al. The association between arterial stiffness and fluid status in peritoneal dialysis patients. Perit Dial Int 2014;34:781-90.
7. Fischer EC, Zocalo Y, Galli C, Wray S, Bia D. Arterial stiffness and renal replacement therapy: a controversial topic. Int J Nephrol 2015;2015:729609.
8. Cho JY, Kim KH. Evaluation of arterial stiffness by echocardiography: methodological aspects. Chonnam Med J 2016;52:101-6.
9. Sipahioglu MH, Kucuk H, Unal A, Kaya MG, Oguz F, Tokgoz B, et al. Impact of arterial stiffness on adverse cardiovascular outcomes and mortality in peritoneal dialysis patients. Perit Dial Int 2012;32:73-80.
10. Cheung AK, Sarnak MJ, Yan G, Berkoben M, Heyka R, Kaufman A, et al. Cardiac diseases in maintenance hemodialysis patients: results of the HEMO Study. Kidney Int 2004;65:2380-9.
11. Candido JSA, Camelo LV, Mill JG, Lotufo PA, Ribeiro ALP, Duncan BB, et al. Greater aortic stiffness is associated with renal dysfunction in participants of the ELSA-Brasil cohort with and without hypertension and diabetes. PLoS One 2019;14:e0210522.
12. Ganesh SK, Hulbert-Shearon T, Port FK, Eagle K, Stack AG. Mortality differences by dialysis modality among incident ESRD patients with and without coronary artery disease. J Am Soc Nephrol 2003;14:415-24.
13. Stack AG, Molony DA, Rahman NS, Dosekun A, Murthy B. Impact of dialysis modality on survival of new ESRD patients with congestive heart failure in the United States. Kidney Int 2003;64:1071-9.
14. Sun CY, Li CY, Sung JM, Cheng YY, Wu JL, Kuo YT, et al. A comparison of the risk of acute myocardial infarction in patients receiving hemodialysis and peritoneal dialysis: A population-based, propensity score-matched cohort study. Atherosclerosis 2020;307:130-8.
15. Sigrist M, Bungay P, Taal MW, McIntyre CW. Vascular calcification and cardiovascular function in chronic kidney disease. Nephrol Dial Transplant 2006;21:707-14.
16. Chung AW, Yang HH, Kim JM, Sigrist MK, Brin G, Chum E, et al. Arterial stiffness and functional properties in chronic kidney disease patients on different dialysis modalities: an exploratory study. Nephrol Dial Transplant 2010;25:4031-41.
17. Strozecki P, Donderski R, Kardymowicz A, Manitius J. Comparison of arterial stiffness in end−stage renal disease patients treated with peritoneal dialysis or hemodialysis. Pol Arch Med Wewn 2012;122:33-9.
18. Covic A, Goldsmith DJ, Florea L, Gusbeth-Tatomir P, Covic M. The influence of dialytic modality on arterial stiffness, pulse wave reflections, and vasomotor function. Perit Dial Int 2004;24:365-72.
19. Chang JH, Yoon SJ, Han SH, Shin SK, Jeon DW, Yang JY, et al. The impact of dialysis modality on arterial stiffness in patients with end-stage renal disease. Ren Fail 2010;32:947-53.
20. Mimura T, Takenaka T, Kanno Y, Aoki H, Ohshima J, Suzuki H. Comparison of changes in pulse wave velocity in patients on continuous ambulatory peritoneal dialysis and hemodialysis one year after introduction of dialysis therapy. Adv Perit Dial 2005;21:139-45.
21. Rajzer MW, Wojciechowska W, Klocek M, Palka I, Brzozowska-Kiszka M, Kawecka-Jaszcz K. Comparison of aortic pulse wave velocity measured by three techniques: Complior, SphygmoCor and Arteriograph. J Hypertens 2008;26:2001-7.
22. Zapolski T, Wysokinski A, Janicka L, Grzebalska A, Ksiazek A. Aortic stiffness and valvular calcifications in patients with end-stage renal disease. Pol Arch Med Wewn 2008;118:111-8.
23. Segall L, Covic A. Cardiovascular disease in haemodialysis and peritoneal dialysis: arguments pro haemodialysis. Nephrol Dial Transplant 2007;22:59-63.
24. Stompor T, Rajzer M, Sulowicz W, Dembinka-Kiec A, Janda

K, Kawacka-Jaszcz K, et al. Trends and dynamics of changes in aortic pulse wave velocity over one-year observation period in patients treated with peritoneal dialysis. Int J Artif Organs 2004;27:904-6.
25. Jung JY, Hwang YH, Lee SW, Lee H, Kim DK, Kim S, et al. Factors associated with aortic stiffness and its change over time in peritoneal dialysis patients. Nephrol Dial Transplant 2010;25:4041-8.
26. Alexandrou ME, Sarafidis P, Mu PT, Sachpekidis V, Papadopoulos C, Loutradis C, et al. Cardiac geometry, function, and remodeling patterns in patients under maintenance hemodialysis and peritoneal dialysis treatment. Ther Apher Dial 2022;26:601-12.
27. Kott J, Reichek N, Butler J, Arbeit L, Mallipattu SK. Cardiac imaging in dialysis patients. Kidney Med 2020;2:629-38.
28. Toprak A, Reddy J, Chen W, Srinivasan S, Berenson G. Relation of pulse pressure and arterial stiffness to concentric left ventricular hypertrophy in young men (from the Bogalusa Heart Study). Am J Cardiol 2009;103:978-84.
29. Di Lullo L, Gorini A, Russo D, Santoboni A, Ronco C. Left ventricular hypertrophy in chronic kidney disease patients: from pathophysiology to treatment. Cardiorenal Med 2015;5:254-66.
30. Tangwonglert T, Davenport A. Changes in extracellular water and left ventricular mass in peritoneal dialysis patients. Kidney Res Clin Pract 2021;40:135-42.
31. Covic A, Gusbeth-Tatomir P, Goldsmith DJ. Arterial stiffness in renal patients: an update. Am J Kidney Dis 2005;45:965-77.