No Harmful Effect of Dialysis-Induced Hypotension on the Myocardium in Patients Who Have Normal Ejection Fraction and a Negative Exercise Test

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Key Words

Dialysis-induced hypotension • Biventricular systolic and diastolic functions • Ejection fraction

Abstract

Background: We investigated the effects of dialysis-induced hypotension (DIH) on the myocardium of patients who have a normal ejection fraction and normal treadmill stress tests.

Methods: This study included 26 end-stage renal disease (ESRD) patients with DIH, 30 ESRD patients without DIH (non-DIH), and 30 control subjects. Mitral-myocardial systolic velocity (MSV), the mitral E'/A' ratio, the left ventricle filling pressure index (E/E' ratio), tricuspid-MSV, and the tricuspid E'/A' ratio were calculated. **Results:** Biventricular systolic and diastolic functions were impaired in dialysis patients. The mitral and tricuspid MSV were similar between DIH and non-DIH patients (8.03 ± 0.90 cm/s vs. 8.31 ± 1.68 cm/s, $p = 0.896$, and 13.27 ± 2.97 cm/s vs. 13.15 ± 2.37 cm/s, $p = 0.980$). Mitral and tricuspid E'/A' were similar between DIH and non-DIH patients (1.30 ± 0.53 vs. 1.16 ± 0.56 , $p = 0.695$, and 0.70 ± 0.24 vs. 0.68 ± 0.33 , $p = 0.976$). Likewise, the E/E' ratio was similar between DIH and non-DIH patients (8.20 ± 2.83 vs.

8.28 ± 2.53 , $p = 0.990$). **Conclusion:** Although biventricular systolic and diastolic function is impaired in dialysis patients compared to controls, DIH episodes did not have an adverse effect on the myocardial functions.

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Introduction

Cardiovascular disease is an important cause of mortality in patients undergoing maintenance dialysis, accounting for approximately one half of all deaths [1]. Cardiac failure is common in patients with end-stage renal disease (ESRD) [2]. In addition to direct myocardial depression from a circulating toxin associated with uremia, chronic renal failure has the potential to cause myocardial dysfunction through chronic hypertension [3], premature atherosclerosis due to abnormal carbohydrate and lipid metabolism [4], cardiomyopathy associated with iron overload [5] and volume overload injury due to chronic anemia, hypervolemia, and iatrogenic arteriovenous fistulas [6, 7].

Additionally, there is increasing evidence implicating dialysis-induced hypotension (DIH) in the pathophysiology of 'uremic cardiomyopathy'. Intradialytic hypotension increases the risk of myocardial hypoperfusion, leading to segmental ischemia inducing segmental left ventricular (LV) dysfunction [8], a process known as myocardial stunning [9]. Myocardial stunning is cumulative and leads to irreversible LV dysfunction in ischemic heart disease [10, 11].

The aim of our study was to investigate the systolic and diastolic function in dialysis patients who have a normal LV ejection fraction and a normal treadmill exercise stress test. Furthermore, we investigated the harmful effects of recurrent DIH episodes on myocardial tissue, and LV systolic and diastolic function.

Methods

This study included 26 ESRD patients with DIH, 30 ESRD non-DIH patients, and 30 control subjects. Exclusion criteria were: severe morbid obesity (BMI >35); moderate or severe heart valve insufficiency and/or stenosis; congenital heart disease; atrial fibrillation; left bundle branch block; established coronary artery disease (CAD); a history of myocardial infarction, unstable angina pectoris, angiographically proven significant coronary stenosis (more than 50% luminal stenosis in at least one coronary artery in coronary angiography) or previous revascularization, or an abnormal exercise test; LV ejection fraction <50%; symptomatic heart failure; poor echocardiographic image quality; stroke; chronic obstructive pulmonary disease, or an orthopedic or musculoskeletal disorder.

Five hundred twenty-two patients with ESRD were evaluated for the determination of DIH during a 6-month run-in period at five hemodialysis (HD) centers. After the 6-month run-in phase and excluding patients based on the enrollment criteria, 26 patients with DIH who had been undergoing 4-hour sessions of HD 3 times a week for ≥ 2 years were enrolled. We also included 30 age- and sex-matched patients undergoing HD without DIH from this cohort with a similar diabetes mellitus and hypertension prevalence. The control group included 30 patients without renal failure (serum creatinine <1.2 mg/dl). Diabetes was diagnosed according to World Health Organization criteria [12]. Participants were classified as hypertensive if their resting systolic blood pressure (SBP) was ≥ 140 mm Hg and/or their diastolic blood pressure (DBP) was ≥ 90 mm Hg or if they took antihypertensive medications. Hypertensive patients who were on a medication which may affect the heart rate (verapamil, diltiazem, β -blockers) quit taking these drugs 1 week before the treadmill stress test. All study patients underwent a thorough clinical, anthropometric, and laboratory examination. Blood samples were obtained the following day after HD before the cardiac stress test and echocardiographic examination. The height and weight of all subjects were measured and the BMI was calculated as weight (kg) divided by height² (m).

As previously reported, DIH was defined as the presence of one or both of the following conditions on more than 30 occasions

among 50 previous sessions of dialysis, [13]: (i) a fall of SBP to less than 90 mmHg during dialysis, and/or (ii) a fall of SBP of more than 25% from the start of dialysis associated with symptoms related to hypotension, including dizziness, vomiting, nausea, and muscle cramps. BP was measured by trained nursing staff, on the same arm, intermittently at 30-min intervals, with a mercury sphygmomanometer, with the patients seated in multiadjustable chairs. The start and end positions were semi-recumbent. When sudden symptomatic hypotension occurred, BP measurements were repeated in the Trendelenburg position, and hypertonic saline boluses were given as needed. The ultrafiltration rate (% body weight reduction) during dialysis sessions was adjusted according to the presumed dry weight (assessed as the postdialysis patient's weight when normotensive and free of edema). During the follow-up period, patients received standard HD using a bicarbonate bath and substituted with cellulose and synthetic membranes. Blood flow rates ranged from 350 to 400 ml/min, the dialysate flow rate was 500 ml/min, and the dialysate temperature was 36°C. Ultrafiltration was linear and the ultrafiltration rate was adapted to reach the dry weight in the preset dialysis time. No sodium or blood volume profiling was carried out. The dialysate contained 140 mmol/l Na⁺, 1 mmol/l K⁺, 33 mmol/l HCO₃⁻, 105 mmol/l Cl⁻, 1.25 mmol/l Ca₂⁺, 0.25 mmol/l Mg₂⁺, and 11 mmol/l glucose. Kt/V was calculated from the Daugirdas formula [14].

Exercise Testing

The modified Bruce standard protocols used symptom-limited treadmill testing during which leaning on handrails was explicitly not allowed [15, 16]. With each stage of exercise and recovery (first 3 min), data on symptoms, rhythm, heart rate, blood pressure, workload in metabolic equivalents (METs), and ST-segment changes were collected. As per laboratory protocol, exercise could be stopped for marked (>2.5 mm) ST-segment depression, chest pain, exercise SBP >250 mmHg, and ventricular tachycardia. The exercise ECG was considered positive if there was 0.1 mV or more of J point depression and the ST-segment was flat or downward sloping 0.08 s after the J point. The treadmill exercise test was interpreted as negative in patients who did not meet the ST depression criteria or who did not develop ST elevation or typical chest pain during the stress test.

Echocardiographic Measurements

Each patient underwent standard transthoracic two-dimensional echocardiography and exercise testing between 10.00 h and 16.00 h on the next day of HD. All echocardiographic assessments were performed by two operators who were blinded to the clinical and laboratory results of the study group. Vivid 3-dimensional echocardiography equipment (GE Vingmed, Horten, Norway) with a 2.5-MHz phased-array transducer was used for each study subject. LV dimension and wall thickness were measured from 2-dimensional guided M-mode echocardiographic tracings at the mid-chordal level on the parasternal long axis view. The M-mode traces were recorded at a speed of 50 mm/s. Ejection fraction was calculated using the Teicholz formula. The LV mass was estimated using the anatomically validated formula of Devereux et al. [17]. It was indexed for body surface area to estimate the LV mass index.

From the apical four-chamber view, pulsed-wave Doppler recordings of the mitral inflow were obtained with the sample volume placed at the tips of the mitral valve leaflets. The following

Table 1. Baseline clinical and biochemical parameters of all patients

Variables	DIH (n = 26)	Non-DIH (n = 30)	Control (n = 30)	p
Age, years	44.15 ± 12.63	43.70 ± 11.70	43.37 ± 13.62	0.973
Males, n (%)	9 (34.6)	12 (40.0)	17 (56.7)	0.215
Diabetes mellitus, n (%)	7 (26.9)	8 (26.7)	7 (23.3)	0.940
Hypertension, n (%)	6 (23.1)	6 (20.0)	7 (23.3)	0.943
Baseline SBP ^a , mm Hg	116.15 ± 30.89	142.53 ± 21.56	131.67 ± 17.30	<0.001
Baseline DBP ^a , mm Hg	72.69 ± 17.56	86.50 ± 16.44	85.03 ± 9.93	0.001
BMI	22.33 ± 4.22	23.32 ± 4.06	24.92 ± 3.48	0.011
Hematocrit, %	34.92 ± 1.38	34.73 ± 1.43	38.06 ± 3.61	<0.001
Blood urea, mg/dl	73.39 ± 11.31	70.06 ± 9.97	38.10 ± 3.88	<0.001
Serum creatinine, mg/dl	7.64 ± 0.74	7.41 ± 0.68	0.96 ± 0.09	<0.001
Potassium, mmol/l	4.19 ± 0.29	4.30 ± 0.35	4.14 ± 0.28	0.139
Calcium, mg/dl	9.06 ± 0.37	9.17 ± 0.41	9.16 ± 0.32	0.560
Phosphorus, mg/dl	5.49 ± 0.39	5.64 ± 0.57	3.73 ± 0.19	<0.001
Albumin, g/dl	3.96 ± 0.25	4.01 ± 0.23	4.07 ± 0.25	0.205
Dialysis duration, months	73.12 ± 41.60	72.37 ± 59.02	–	0.392
Mean Kt/V urea value	1.32 ± 0.09	1.33 ± 0.07	–	0.462
Weight reduction, %	3.21 ± 0.44	3.08 ± 0.52	–	0.317

^a Post hoc analysis of the groups is given in Results.

parameters were measured by pulsed-wave Doppler: peak velocities of early (E) and late diastolic filling (A), and deceleration time. The ratio of early diastolic to late diastolic mitral inflow velocities was calculated (E/A). All Doppler signals were recorded with a chart recorder set at 100 mm/s. The averages of 3 cycles were used.

The TDI program was set to pulsed-wave Doppler mode. Filters were set to exclude high frequency signals. Gains were minimized to allow a clear tissue signal with minimal background noise. The TDI of the diastolic and systolic velocities was obtained from the apical 4-chamber view. A 1.5-mm sample volume was placed at the lateral corner of the mitral and tricuspid annulus. Analysis was performed for early (E') and late diastolic velocity (A') and myocardial systolic velocity (MSV) and the E'/A' ratio.

LV systolic function was evaluated with mitral MSV, and diastolic function was evaluated with mitral inflow E/A ratio and mitral E'/A' ratio parameters. In addition, E/E' was calculated as an indicator of the left ventricle filling pressure. Right ventricular systolic function was assessed with tricuspid MSV. Right ventricular diastolic function was evaluated with tricuspid E'/A' since this parameter is relatively less affected by the preload condition [18].

Statistical Analysis

All statistical analyses were performed using SPSS 11.5 (SPSS Inc., Chicago, Ill., USA). Data are presented as means ± SD for each group of measurements. The frequency distribution of categorical variables was tested using a 2 × 2 contingency table. Continuous variables of two groups were compared using Student's t test, and the Mann-Whitney U test was used when a non-normal distribution was present. ANOVA and Kruskal-Wallis tests were used during the comparison of three groups with each other depending on the normal distribution of the data. For the post hoc test, we used the Tukey HSD (honestly significant differ-

ence) test for the variables that were significant in ANOVA, and the Mann-Whitney U test for the variables that were significant in the Kruskal-Wallis test. Correlations between the variables were tested by Pearson's correlation analysis. In all statistical analyses, two-tailed p values <0.05 were considered statistically significant.

Results

Fifty-six HD patients (mean age 43.9 ± 11.8 years) and 30 controls (mean age 43.4 ± 13.6 years) were included in this study. Baseline clinical and biochemical parameters of the study population are presented in table 1. All groups were similar in terms of age, gender, prevalence of diabetes and hypertension, antihypertensive medication profile, serum potassium, serum calcium, and serum albumin levels. The control group had increased BMI compared to the patients on HD (p = 0.011). The DIH group had lower baseline SBP compared to non-DIH patients and controls (116.15 ± 30.89 mm Hg vs. 142.53 ± 21.56 mm Hg, p < 0.001, and 116.15 ± 30.89 mm Hg vs. 131.67 ± 17.30 mm Hg, p = 0.042). The baseline SBP of non-DIH patients and the control group were similar (142.53 ± 21.56 mm Hg vs. 131.67 ± 17.30 mm Hg, p = 0.180). The DIH group had lower baseline DBP compared to the non-DIH and control patients (72.69 ± 17.56 mm Hg vs. 86.50 ± 16.44 mm Hg, p = 0.002, and 72.69 ± 17.56 mm Hg

Fig. 1. Correlations between baseline SBP and the LV mass of dialysis patients.

Table 2. Structural echocardiographic parameters of all patients

Echocardiographic parameters	DIH	Non-DIH	Control	p
IVS-D, cm	1.05 ± 0.15	1.06 ± 0.12	0.97 ± 0.17	0.082
PW-D, cm	0.96 ± 0.14	0.95 ± 0.10	0.86 ± 0.16	0.012
LVEDD, cm	4.74 ± 0.41	4.72 ± 0.27	4.59 ± 0.36	0.240
LVESD, cm	3.10 ± 0.44	3.04 ± 0.32	2.90 ± 0.39	0.135
LVEF, %	63.9 ± 4.9	64.6 ± 2.5	63.9 ± 3.4	0.703
RVEDD, cm	3.23 ± 0.34	3.17 ± 0.28	3.12 ± 0.34	0.460
LV mass ^a , g	169.2 ± 41.1	169.3 ± 38.2	138.6 ± 47.8	0.009
LV mass index ^a g/m ³	108.7 ± 27.6	104.0 ± 27.6	71.6 ± 21.2	<0.001

IVS-D = Interventricular septum diastolic thickness; PW-D = posterior wall diastolic thickness; LVEDD = left ventricle end-diastolic diameter; LVESD = left ventricle end-systolic diameter; LVEF = left ventricular ejection fraction; RVEDD = right ventricle end-diastolic diameter. ^a Post hoc analysis of the groups is given in Results.

vs. 85.03 ± 9.93 mm Hg, $p = 0.008$). Baseline DBP were similar between the non-DIH and control groups (86.50 ± 16.44 mm Hg vs. 85.03 ± 9.93 mm Hg, $p = 0.923$).

The structural echocardiographic parameters of all patients are presented in table 2. Dialysis patients had increased LV wall thickness, LV mass, and mass index compared to the control group. There was no difference be-

tween the DIH and non-DIH groups in terms of LV mass and mass index ($p > 0.05$). The LV ejection fraction was similar in the three groups ($p = 0.703$).

The Doppler measurements of all patients are presented in table 3. The DIH and non-DIH groups had reduced mitral myocardial systolic velocities compared to the controls (8.03 ± 1.90 cm/s vs. 10.78 ± 3.21 cm/s, $p < 0.001$, and 8.31 ± 1.68 cm/s vs. 10.78 ± 3.21 cm/s, $p < 0.001$). The mitral MSV values of the DIH and non-DIH subgroups were statistically similar (8.03 ± 1.90 cm/s vs. 8.31 ± 1.68 cm/s, $p = 0.896$). The non-DIH group had a reduced mitral E'/A' ratio compared to the controls (1.16 ± 0.56 vs. 1.70 ± 0.81, $p = 0.005$). The mitral E'/A' ratio values of the DIH versus non-DIH and DIH versus control subgroups were statistically similar (1.30 ± 0.53 vs. 1.16 ± 0.56, $p = 0.695$, and 1.30 ± 0.53 vs. 1.70 ± 0.81, $p = 0.062$). The DIH and non-DIH groups had reduced mitral inflow E/A ratios compared to the controls (0.88 ± 0.31 vs. 1.21 ± 0.37, $p = 0.001$, and 0.91 ± 0.26 vs. 1.21 ± 0.37, $p = 0.002$). The mitral inflow E/A ratio values of the DIH and non-DIH subgroups were statistically similar (0.88 ± 0.31 vs. 0.91 ± 0.26, $p = 0.927$). The DIH and non-DIH groups had increased E/E' ratios compared to the controls (8.20 ± 2.83 vs. 6.21 ± 1.87, $p = 0.009$, and 8.28 ± 2.53 vs. 6.21 ± 1.87, $p = 0.004$). The E/E' ratio values of the DIH and non-DIH subgroups were statistically similar (8.20 ± 2.83 vs. 8.28 ± 2.53, $p = 0.990$).

ANOVA analysis demonstrated that tricuspid MSV values were statistically reduced in patients on dialysis ($p = 0.042$). However, post hoc analysis did not demonstrate a significant difference between each group ($p > 0.05$). The DIH and non-DIH groups had reduced tricuspid E'/A' ratios compared to the controls (0.70 ± 0.24 vs. 1.08 ± 0.53, $p = 0.002$, and 0.68 ± 0.33 vs. 1.08 ± 0.53, $p = 0.001$). The tricuspid E'/A' ratio values of the DIH and non-DIH subgroups were statistically similar (0.70 ± 0.24 vs. 0.68 ± 0.33, $p = 0.976$). Pearson's correlation analysis including dialysis duration, baseline SBP, baseline DBP, BMI, hematocrit levels, and echocardiographic parameters was performed on the dialysis patients. This analysis demonstrated a moderate strength correlation between the baseline SBP and LV mass ($r = 0.45$, $p < 0.001$) (fig. 1).

Discussion

In our study, we found that biventricular systolic and diastolic functions were impaired in dialysis patients who were free from CAD demonstrated by a negative treadmill stress test and echocardiography. Furthermore, we

Table 3. Doppler measurements of all patients

	DIH	Non-DIH	Control	p
Mitral inflow				
Peak E, cm/s	71.92 ± 18.33	80.00 ± 19.29	76.97 ± 16.22	0.240
Peak A, cm/s	84.62 ± 20.82	89.33 ± 18.18	66.27 ± 14.66	<0.001
E/A ratio ^a	0.88 ± 0.31	0.91 ± 0.26	1.21 ± 0.37	<0.001
DT, ms	224.5 ± 31.8	234.5 ± 34.9	190.8 ± 32.2	<0.001
Lateral mitral annulus				
Myocardial systolic velocity ^a , cm/s	8.03 ± 0.90	8.31 ± 1.68	10.78 ± 3.21	<0.001
E' peak velocity, cm/s	9.19 ± 2.55	10.31 ± 3.15	12.94 ± 4.36	<0.001
A' peak velocity, cm/s	7.60 ± 2.04	9.73 ± 2.41	8.11 ± 1.96	0.001
E'/A' ratio ^a	1.30 ± 0.53	1.16 ± 0.56	1.70 ± 0.81	0.005
Lateral tricuspid annulus				
Myocardial systolic velocity ^a , cm/s	13.27 ± 2.97	13.15 ± 2.37	14.58 ± 1.67	0.042
E' peak velocity, cm/s	9.84 ± 3.11	9.75 ± 2.86	12.41 ± 3.32	0.002
A' peak velocity, cm/s	14.34 ± 3.26	15.92 ± 5.07	12.44 ± 3.22	0.005
E'/A' ratio ^a	0.70 ± 0.24	0.68 ± 0.33	1.08 ± 0.53	<0.001
LV filling pressure				
E/E' ratio ^a	8.20 ± 2.83	8.28 ± 2.53	6.21 ± 1.87	0.002

DT = Deceleration time. ^a Post hoc analysis of the groups is given in Results.

found that DIH episodes were not associated with adverse effects on the myocardium among this specific dialysis patient population.

Uremia increases the myocardial interstitial ground substance by mechanisms independent of hypertension [19]. The hyperparathyroid state and altered vitamin D status found in chronic renal failure contribute to the cardiovascular pathology (fibrosis, cardiomyopathy, myocardial hypertrophy, myocardial ischemia, uremic glucose intolerance, dyslipidemia, hypertension, atherosclerosis, and vascular and cardiac calcifications) and also to the excess mortality from cardiovascular causes [20]. A high prevalence of arteriosclerosis and remodeling of large arteries causes a reduction in arterial compliance, thereby leading to increased SBP, increased pulse pressure, LV hypertrophy (LVH), and decreased coronary perfusion [21]. HD treatment may affect cardiac functions by itself. Increased myocardial calcium content is strongly associated with myocardial dysfunction in patients undergoing dialysis [22]. Similar to previous studies, the dialysis patients had impaired biventricular systolic and diastolic function compared to the control group. Our findings contribute significantly to the literature, especially after excluding the CAD of these patients by echocardiography and exercise stress tests. In addition, the dialysis patients had increased LV mass and LVMI compared to the controls. Patients with DIH had

reduced baseline SBP and DBP levels compared to the non-DIH group; however, both groups had similar LV mass and LVMI levels. These findings represent a multifactorial etiology for the development of LVH in dialysis patients, especially after considering the significant correlation between LV mass and baseline SBP in the correlation analysis.

HD patients are particularly susceptible to myocardial ischemia. In addition to the high prevalence of coronary artery atheroma [23], diabetic dialysis patients have been shown to have a reduced coronary flow reserve (CFR) in the absence of coronary vessel stenoses [24]. CFR determines the ability to increase blood flow to the myocardium during increased demand, and there is preliminary evidence that the same reduction in CFR is also seen in nondiabetic dialysis patients [25]. In part this may be caused by LVH, present in 74% of patients on dialysis [26], which reduces the CFR and is associated with a myocyte-capillary mismatch [27]. Increased peripheral artery stiffness is also recognized to have an adverse effect on myocardial perfusion and reduces the ischemic threshold [28]; therefore, LVH in tandem with increased vascular stiffness may lead to a propensity to reduced subendocardial blood flow [29]. In addition, dysregulation of blood pressure control due to abnormal baroreflex sensitivity [30] and vasoregulatory failure [31] increases the risk of myocardial hypoperfusion.

DIH is an important complication in HD patients. It occurs in one third of patients on dialysis. This condition is also associated with increased morbidity and decreased overall quality of life [32]. The mechanism of DIH has been explained as the imbalance between a decreased plasma volume during HD and counter-regulatory cardiovascular and neurohormonal mechanisms [33, 34]. These sudden changes in blood pressure may provoke subendocardial ischemia/necrosis or myocardial stunning. The average dialysis period of our study patients was 6 years and CAD was excluded by noninvasive tests. We found that DIH episodes were not associated with an adverse impact on the myocardium among this specific dialysis patient population. Selby et al. [9, 10] reported that a significant number of new LV regional wall motion abnormalities occurred during standard dialysis. The same studies showed that improving the mean blood pressure and reducing DIH with both biofeedback dialysis and cool temperature dialysis was associated with a significant reduction in the number of new regional wall motion abnormalities. The incidence of diabetes mellitus was greater in these studies compared to our patients, and the patients were more heterogenous in terms of ischemic heart disease in the study by Selby et al. [9, 10]. Owen et al. [35] demonstrated the impaired myocardial contractile reserve in patients with DIH by a dobutamine-atropin stress test in their small study. The authors did not observe any ischemic ECG changes or angina suggesting epicardial CAD during this study. The impaired contractile reserve in this study may originate from the insufficient response to the pharmacological agents at the myocardial receptor level since the tissue Doppler MSV values of our dialysis patients were all similar. Our study makes a significant contribution to the literature with its large number of study patients and by excluding CAD with the exercise stress test. The dialysis patients in our study had mild LVH and the prevalence of diabetes was low (26%). These findings indicate that the CFR values might have been

preserved in our study patients. Similarly, the DIH and non-DIH groups had impaired systolic and diastolic functions compared to controls; however, these were similar to each other. This result suggests that myocardial functions play a minor role in the development of DIH for this specific population of patients who have preserved ejection fraction.

Limitations of the Study

We excluded the presence of epicardial stenotic lesions with an exercise stress test in our study group. We did not perform a coronary arteriogram since there was no clinical indication to justify this invasive procedure. Long-term randomized clinical trials on patients with or without critical epicardial CAD are needed to demonstrate the impact of DIH episodes on the myocardial functions. Similarly, randomized follow-up studies according to CFR values are needed in patients in whom CFR was quantitatively measured.

In conclusion, the biventricular systolic and diastolic functions were impaired compared to controls in dialysis patients who had normal LV ejection fraction and negative stress test. We did not observe any adverse effect of dialysis-induced hypotensive episodes on myocardial functions in the subgroup of dialysis patients.

Acknowledgements

The authors are grateful for the excellent nursing support from Yasemin Bozalp and Yeliz Ozkan Cetin during the treadmill cardiac stress tests and nursing care from Serpil Gunduz during the dialysis of the study patients. We appreciate the excellent secretarial assistance of Ayten Bolut during the preparation of this paper.

Disclosure Statement

The authors declare that there are no conflicts of interest.

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