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Acute Effects of Hemodialysis on Left and Right Ventricular Function Evaluated by Doppler Tissue Imaging

Hemodiyalizin Sağ ve Sol Ventrikül Fonksiyonlarına Akut Etkisinin Doku Doppler Görüntüleme ile Değerlendirilmesi

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ABSTRACT

Purpose: Doppler tissue imaging (DTI) allows noninvasive assessment of both left ventricular (LV) and right ventricular (RV) function. The aim of this study was to evaluate the effect of hemodialysis (HD) on LV and RV function using DTI.

Method: Our study group included 30 patients on chronic HD program (mean age 45 15 years). Myocardial (Sm, Em, Am) and annular velocities (Ea, Aa) were measured in several cardiac territories before and after HD.

Results: After HD, Ea significantly reduced from 10.8 3.4 cm/s to 9.6 2.4 cm/s ($p = 0.029$). Patients exhibited a lower Em following HD in all measured territories. Em/Am ratio was also reduced for each LV wall investigated after HD in all measured territories. At the RV segments, Sm, Em, and Am decreased significantly in all measured territories. Em of the anterior wall was positively related to ultrafiltration volume ($r = 0.25$, $p = 0.006$), whereas the decrease of Sm of RV basal segment correlated with a decrease of diastolic blood pressure ($r = 0.23$, $p < 0.01$).

Conclusion: Our data indicate that a single HD session is associated with acute changes of systolic and diastolic parameters of LV and RV.

Keywords : tissue Doppler imaging; hemodialysis; left ventricular function; right ventricular function.

ÖZET

Amaç: Doku Doppler görüntüleme, sağ ve sol ventrikülün fonksiyonlarının non-invazif değerlendirilmesine olanak verir. Çalışmamızın amacı, hemodiyalizin sağ ve sol ventrikül fonksiyonlarına akut etkisini non-invazif olarak doku Doppler görüntüleme aracılığıyla değerlendirmektir.

Yöntem: Çalışmamızda kronik hemodiyaliz proğramına devam eden 30 hasta (ortalama yaş: 45 ± 15 yıl) incelendi. Hemodiyaliz öncesi ve sonrasında farklı kardiyak bölgelerden miyokardiyal (Sm, Em, Am) ve annüler (Ea, Aa) velositeler doku Doppler aracılığıyla ölçüldü.

Bulgular: Hemodiyaliz sonrası, Ea anlamlı olarak 10.8 ± 3.4 cm/s'den 9.6 ± 2.4 cm/s' ye düştü ($p = 0.029$) Ölçüm yapılan tüm kardiyak segmentlerde hemodiyaliz sonrası ölçülen Em değeri anlamlı olarak daha düşük bulundu. Ölçüm yapılan her kardiyak segmentte ayrı ayrı olmak üzere Em/Am oranı hemodiyaliz sonrasında anlamlı derecede düştü. Sağ ventrikül değerlendirmesinde ölçüm yapılan tüm segmentlerde Sm, Em ve Am anlamlı olarak hemodiyaliz sonrası düştü. Ön duvar Em değeri, ultrafiltrasyon volümüyle pozitif ilişkili saptanırken; ($r = 0.25$, $p = 0.006$) sağ ventrikül bazal segmentinde Sm değerindeki düşüş diyastolik kan basıncı düşüşüyle korele bulundu. ($r=0.23$, $p<0.01$)

Sonuç: Verilerimiz, tek bir hemodiyaliz seansında da sağ ve sol ventrikülün sistolik ve diyastolik parametrelerinde akut değişimlerin olduğunu göstermiştir.

Anahtar Kelimeler: Doku Doppler görüntüleme, hemodiyaliz, sol ventrikül fonksiyonları, sağ ventrikül fonksiyonları.

INTRODUCTION

Cardiovascular mortality is high in patients with end stage renal disease (ESRD) on hemodialysis (HD)[1]. In addition, HD per se can stress cardiac function because of the acute hemodynamic changes in blood volume, arterial pressure, electrolytes and sympatho-vagal balance[2]. Contradictory data exist about the HD-induced changes on ventricular diastolic function. Some studies reported that HD improves left ventricular (LV) function[3-4] on the other hand other studies found impairments[5-6], whereas others concluded that the effect of HD on echocardiographic parameters of LV filling are primarily related to changes in volume status rather than to HD-related metabolic alterations[7-8]. Evidence exists that LV function is changed in chronic uremic patients; however data about the acute changes on right ventricular (RV) function are not clear.

Doppler tissue imaging (DTI) is a non-invasive Doppler echocardiographic technique that allows quantitative, objective, and highly reproducible measurements of LV systolic and diastolic velocities. DTI is considered as a useful diagnostic tool for early detection of LV dysfunction[9] and for the assessment of acute changes in LV function[10]. DTI parameters have also been shown to be less load dependent, especially the isovolumetric phase velocities. This is of particular importance for HD patients in whom both pre-load and afterload change as the hydration status fluctuates. DTI can also give additional information on global and regional systolic and diastolic RV function in HD patients in comparison with conventional echocardiography[11].

The aim of the study was to evaluate the effect of a single HD session on LV and RV systolic and diastolic function by using pulsed wave DTI.

MATERIALS and METHODS

Study Population

Thirty patients (18 males, aged 45 ± 15 years) with ESRD on maintenance HD program (for 30 ± 26 months) were recruited from the Dialysis Unit of our institution. Exclusion criteria were functional class NYHA II-IV, absence of sinus rhythm, pericardial effusion, significant valvular disease, coronary artery disease (CAD), congestive heart failure, acute illness, and neoplasia.

Past medical history of patients were obtained from the hospital database. Diagnoses of CAD and heart failure were on the basis of clinical history, findings of definite ECG changes of ischemia or previous myocardial infarction, echocardiographic findings of regional wall motion abnormality, and angiographic evidence of significant CAD.

Routinely HD treatment was performed using high biocompatibility membranes lasting four hours, three times a week. Delivered dialysis dose as assessed by Kt/V, was 1.51 ± 0.16 and the normalized protein catabolic rate was 1.2 ± 0.3 g/kg per day.

Informed consent was obtained from each subject entering the study. The investigation conforms to the Declaration of Helsinki.

Blood sample was drawn by the afferent line before the beginning of the HD. Serum levels of urea, creatinine, potassium, sodium, calcium, phosphate, and hemoglobin were determined using the standard methods of our laboratory.

Conventional 2-dimensional and Doppler echocardiography

Ultrasound examinations were performed before and 1 hour after a HD session with an Acuson XP-128 (Acuson Computer Sonography,

Mountain View, CA, USA) machine. The echocardiograms were evaluated according to the recommendations suggested by the American Society of Echocardiography[12].

Two dimensional and Doppler parameters were acquired and recorded. At least three consecutive heart beats in each view were acquired. Standard 2-dimensional echocardiographic measurements including LV end-diastolic and end-systolic dimensions, end-diastolic and end-systolic wall thicknesses of the interventricular septum, and LV posterior wall were measured from M-mode images. The ejection fraction (EF) was calculated by modified Simpson's method. Also the velocities of early diastolic inflow (E) and late atrial inflow (A), E/A ratio, and isovolumetric relaxation time (IVRT) were measured using pulsed wave Doppler with the sample volume positioned at the tips of the mitral valve leaflets in the apical 4 chamber view.

Doppler tissue imaging

Pulsed wave DTI was performed using a special software package available on the Acuson XP-128. This method is capable of providing measurements of ventricular wall motion velocity by positioning the sample volume within the myocardium. The Doppler program was set to the pulsed mode with a sample volume of 4 mm. We performed a pulsed-wave DTI with optimal gain settings to minimize noise. DTI parameters of LV lateral wall, basal interventricular septum, RV segments (apical, mid, and basal), and mitral annulus (lateral and septal) were obtained from the apical 4 chamber views, whereas those of LV inferior and anterior wall segments were obtained from apical two chamber views. Myocardial velocities were measured in systole (Sm) and in early (Em) and late (Am) diastole.

Mitral annular diastolic velocities were also measured. Early (Ea) and late annular (Aa) diastolic velocities, Ea/Aa ratio, and IVRTa were recorded. All parameters were measured during three consecutive cardiac cycles and the average values were calculated. All measurements were made offline by a single observer blinded to the patient details before and one hour after an effective HD session.

Statistical analysis

Statistical analysis was performed using SPSS 9.0 (SPSS for windows 9.0, Chicago, IL). Numeric values were expressed as mean ± SD. Parametric or non-parametric tests were used for comparison of data according to the distribution of the data. Statistical analysis was performed using the one-way analysis of variance (ANOVA), Student's t-test, correlation and multiple regression analysis. Differences were considered significant when $p < 0.05$.

RESULTS

Of the study subjects 18 were men and 12 were women. The cause of ESRD was unknown in 8 patients, while 5 patients had diabetes mellitus, 5 patients had hypertension, 4 patients had chronic glomerulonephritis, 3 patients had nephrolithiasis, 1 patient had vesicouretheral reflux, 1 patient had pyelonephritis, 1 patient had polycystic renal disease, 1 patient had amyloidosis, and 1 patient had Alport syndrome.

Twenty-one patients were on anti-hypertensive medication. Calcium antagonists were the drug of choice in 9 patients, angiotensin-converting enzyme inhibitors in 5 patients, angiotensin II receptor antagonist in 3 patients, beta-adrenergic blockers in 2 patients, and alpha-adrenergic blockers in 2 patients. Clinical characteristics of the study population are given in Table 1.

As shown in Table 1, systolic and diastolic blood pressures were significantly decreased (from 133 ±17 to 120 ± 19 mmHg and from 82 ±13 to 75 ±12 mmHg respectively, $p < 0.0001$), whereas heart rate was increased (from 77 ±11 to 85 ±15 pbm, $p = 0.001$) after HD. Following HD, no significant difference was observed in EF (59 ± 8 % vs 61± 10 %, $p > 0.05$), whereas LV end-diastolic volume (from 101 ± 29 to 78 ± 23 cc, $p <$

0.0001) and end-systolic volume (from 39 ± 16 to 31 ± 14 cc, p = 0.001) significantly decreased as well as stroke volume (from 60 ± 21 to 47 ± 14 cc, p = 0.001) (Table 1).

Effect of HD on mitral inflow parameters is summarized in Table 2. After HD, E velocity and E / A ratio significantly decreased; however, A velocity and IVRT remained unchanged (p > 0.05). Effect of HD on mitral annular pulsed-wave DTI parameters is summarized in Table 2. After HD, Ea significantly reduced from 10.8 ± 3.4 to 9.6 ± 2.4 (p = 0.029), without significant change in Aa, Ea/Aa ratio, and IVRTa (p > 0.05). Table 3 shows comparisons of pre- and post- HD myocardial velocities obtained from different LV walls. Sm did not change significantly after HD except for the LV lateral wall. At the LV lateral wall, Sm increased from 7.3 ± 1.6 to 9.1 ± 3.2 cm/s, p=0.006, after HD. Em and Em/Am ratio significantly decreased (p < 0.05, for all LV walls), without a significant change in Am (p > 0.05) when compared with pre-HD values.

At the RV segments, Sm, Em, and Am decreased significantly (p < 0.05, for all RV segments), however, Em/Am did not demonstrate a significant change (p > 0.05) after HD (Table 4). Linear regression analysis demonstrated a dependency of Em of the anterior wall upon ultrafiltration volume (r = 0.25, P < 0.006) and Sm of the RV basal segment upon diastolic blood pressure after HD (r = 0.23, p < 0.01).

No statistically significant correlation was found between DTI-derived systolic and diastolic measurements and changes in serum electrolyte levels, systolic blood pressure, heart rate, hematocrit, and calcium-phosphate plasma levels following HD.

Table 1: Hemodialysis-Related Changes in Echocardiographic and Hemodynamic Variables and Biochemical Data.

	Before HD	**After HD**	**p**
SBP (mmHg)	133 ± 17	120 ± 19	< .0001
DBP (mmHg)	82 ±13	75 ± 12	< .0001
HR (beats/min)	77 ± 11	85 ± 15	001
Weight (kg)	64.5 ± 12	61.5 ± 12	< .0001
BSA (kg/m2)	1.70 ± 0.16	11.66 ± 0.16	< .0001
EF	59 ± 8	61 ± 10	.18
LVEDV (cc)	101 ± 29	78 ± 23	< .0001
LVESV (cc)	39 ± 16	31 ± 14	.001
LVSV	60 ± 21	47 ± 14	.001
Urea (mg/dl)	80 ± 15	-	-
Creatinine (mg/dl)	9.4 ± 3.1	-	-
Hemoglobin (g/dl)	10.8 ± 1.7	-	-
Sodium (mEq/l)	136 ± 4	-	-
Potassium (mEq/l)	5.2 ± 0.8	-	-
Phosphorus (mg/dl)	4.9 ± 1.9	-	-
Calcium (mg/dl)	10.9 ± 9.2	-	-

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; BSA, body surface area; LVEF, left ventricular ejection fraction; LVEDV, left ventricular end diastolic volume; LVESV, left ventricular end systolic volume; LVSV, left ventricular stroke volume

Table 2: Hemodialysis-related changes in mitral inflow and diastolic mitral lateral annular velocities

	Before HD	After HD	p
E (cm/s)	74 ± 24	53 ± 18	< .0001
A (cm/s)	76 ± 20	72 ± 18	.21
E/A ratio	1 ± 0.7	0.8 ± 0.2	.048
IVRT (ms)	74 ± 18	79 ± 15	.24
Ea (cm/s)	10.8 ± 3.4	9.6 ± 2.4	.029
Aa (cm/s)	12.4 ± 3.1	11.3 ± 2.4	.052
Ea/Aa ratio	0.9 ± 0.4	0.9 ± 0.3	.5
IVRTa (ms)	83 ± 23	83 ± 29	.79

E, early diastolic mitral inflow velocity; A, late diastolic mitral inflow velocity; IVRT, isovolumetric relaxation time; Ea, early diastolic mitral annular velocity; Aa, late diastolic mitral annular velocity, IVRTa, annular isovolumetric relaxation time.

Table 3: Pulsed Doppler tissue imaging parameters of left ventricular walls in patients before and after hemodialysis

		Before HD	After HD	p
Lateral wall	Sm (cm/s)	7.3 ± 1.6	9.1 ± 3.2	.006
	Em (cm/s)	11.7 ± 4.3	9.8 ± 3.4	.026
	Am (cm/s)	10.5 ± 2.8	10.3 ± 2.8	.82
	Em/Am ratio	1.1 ± 0.4	0.9 ± 0.3	.01
IVS	Sm (cm/s)	8 ± 2.2	7.6 ± 2.1	.19
	Em (cm/s)	8.7 ± 2.3	6.3 ± 1.6	< .0001
	Am (cm/s)	9.8 ± 2.6	9.2 ± 2.5	.054
	Em/Am ratio	0.9 ± 0.3	0.7 ± 0.2	< .0001
LV inferior wall	Sm (cm/s)	8.2 ± 2.2	8.4 ± 2.3	.62
	Em (cm/s)	9.9 ± 3.9	7.8 ± 2.4	.002
	Am (cm/s)	10.4 ± 2.8	10.5 ± 2.6	.81
	Em/Am ratio	1.0 ± 0.5	0.7 ± 0.2	< .0001
LV anterior wall	Sm (cm/s)	7.4 ± 2.8	9.3 ± 3.4	.061
	Em (cm/s)	10.3 ± 2.9	8.2 ± 2.6	.009
	Am (cm/s)	9 ± 2	1 9.1 ± 3.0	.9
	Em/Am ratio	1.1 ± 0.4	0.9 ± 0.3	.016

HD, hemodialysis; LV, left ventricle, Sm, myocardial systolic velocity; Em, early diastolic myocardial velocity; Am, late diastolic myocardial velocity

Table 4: Hemodialysis-related changes in right ventricular segments

		Before HD	After HD	p
RV apical segment	Sm (cm/s)	.4 ± 3.4	8.1 ± 3.2	.016
	Em (cm/s)	10.2 ± 3.3	8 ± 3.1	.001
	Am (cm/s)	11.9 ± 4.7	10.1 ± 3.2	.016
	Em/Am ratio	0.9 ± 0.4	0.8 ± 0.3	1
RV mid segment	Sm (cm/s)	12.3 ± 2.8	10.2 ± 3.3	< .0001
	Em (cm/s)	12.2 ± 3.8	9.5 ± 2.6	< .0001
	Am (cm/s)	16.6 ± 5.7	13.3 ± 4.4	.001
	Em/Am ratio	0.8 ± 0.2	0.7 ± 0.3	.72
RV basal segment	Sm (cm/s)	14.5 ± 3.5	12.5 ± 4.1	.003
	Em (cm/s)	14.2 ± 3.8	10.3 ± 3.5	< .0001
	Am (cm/s)	18.9 ± 6.1	15 ± 4.4	< .0001
	Em/Am ratio	0.8 ± 0.2	0.7 ± 0.3	.25

HD, hemodialysis; RV, right ventricle; Sm, myocardial systolic velocity; ; Em, early diastolic myocardial velocity; Am, late diastolic myocardial velocity

DISCUSSION

In the present study, HD-related volume reduction resulted in significant decrease in E and E/A ratio. These findings were similar to those observed in previous studies evaluating the effect of HD on Doppler echocardiographic parameters of LV filling[7,13]. Preload reduction is the main mechanism that accounts for acute changes in Doppler diastolic indices during HD[14].

Some studies suggest that mitral annular early diastolic velocity behaves as preloadindependent index of left ventricular relaxation[15,16] however some studies report that it is preload-dependent[14,17]. In our study, HD-related volume reduction resulted in significant decrease in Ea, but no significant change in Ea/Aa ratio. These findings resembled previous studies suggesting that mitral annular velocities detected by DTI are preload dependent[14,17] Agmon et al.[14] and Dincer et al.,[17] reported a significant decrease of DTI-derived velocities of diastolic myocardial function after HD. Ie et al.,[18] compared LV diastolic function in 10 patients before and after HD, and they found that volume overload before HD resulted in underestimation of the degree of diastolic dysfunction. However, Bauer et al.[19] reported that DTI-derived measurements were not significantly affected by HD and Hayshi et al.[11] showed an improvement in cardiac function after HD. These discrepancies could be related to differences in the methodology, changes in heart rate, in pre-load and after-load and in the clinical condition of the patients. The potential influence of load-related changes in myocardial velocities has been addressed in some studies. Oguzhan et al.,[7] studied the effect of HD-related volume reduction on DTI diastolic velocities. They found that LV lateral wall Em and Em / Am ratio and septal Em significantly reduced after HD and these results are in agreement with ours. But they did not study other walls of the LV.

In other study, Govind et al.,[3] studied differing myocardial response to a single HD session in ESRD with and without diabetes mellitus and CAD. They reported that a single HD session improves LV function only in ESRD without coexistent DM and / or CAD. In our study there were only 5

diabetic patients and we excluded patients with CAD Our results suggest that LV loading conditions may affect DTI diastolic myocardial velocities as well as DTI mitral annular diastolic velocities. On the other hand, we did not find a HDinduced worsening of the LV systolic function. Neither EF, nor Sm of LV wall segments were significantly changed after HD. In addition, Sm of the lateral wall significantly increased after HD. These results are concordant with the study mentioned above[3]. Improved LV systolic function in ESRD could be explained by the fact that there is improved myocardial perfusion, which is not only because of removal of fluid, solutes and toxins[11], but also because of improvements in myocardial interstitial edema[20].

Although there are plenty of data about the differences in RV DTI velocities by preload reduction, only a few studies regarding RV function are found in the literature. Arinc et al.,[21] investigated the effects of intravascular volume reduction by ultrafiltration on RV functions by DTI in 27 dialysis patients. They suggest that RV systolic and diastolic velocities were not affected or only minimally affected by preload reduction in HD patients.

However, in our study, systolic and early and late diastolic velocities of RV segments decreased significantly but early/late ratio did not change significantly after HD. The observed changes in RV systolic function may simply reflect decreased contractility due to a leftward movement on the Frank Starling curve due to reduced preload after HD. Acute DTI changes in uremic patients after HD treatment may be explained by several mechanisms which determine LV structural changes leading to impairment of LV relaxation. A possible mechanism is the sympathetic hyperactivity occurring during HD[22]. The unbalanced hyper-sympathetic response is due to body fluid depletion in relation to the ultrafiltration rate. In conclusion, our data indicate that a single hemodialysis session is associated with acute changes of LV diastolic and RV systolic and diastolic function. This seems to be related to the ultrafiltration volume and then to the interdialytic weight gain. These findings suggest that cardiac functions should be assessed in a relatively normovolemic state, namely at the end of the HD and that the limitation of interdialytic weight gain and a low ultrafiltration volume are important factors for the cardioprotection of HD patients.

Study Limitations

DTI-derived parameters are limited by rotational and tethering effects of an actively contracting and relaxing heart. Recently developed new DTI modalities which measures segmental tissue deformation, may overcome this limitation.

Although all measurements were made offline by a single observer blinded to the patient details, we did not establish data for inter- and intra-observer variations. We have studied only one randomly chosen HD session and hence it is difficult to reflect on the natural history of progression of LV dysfunction in these patients.A more important study would be to evaluate changes in LV and RV performance at the initiation of dialysis and several months after initiation of HD in those who were undergoing HD for the first time.

The validity of our study is limited by the relatively small number of patients. Therefore,these findings have to be confirmed in larger prospective studies.

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