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CLINICAL STUDY

Effects of Altered Volume Loading on Left Ventricular Hemodynamics and Diastolic Filling During Hemodialysis

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ABSTRACT

Background. Changes in the circulating volume associated with hemodialysis (HD) resulted in alternations of left ventricular (LV) filling. However, previous studies offered conflicting findings. This study thus evaluated the impact of HD on LV diastolic filling indices and hemodynamics. *Materials and Methods.* Forty patients with end-stage renal disease were studied by Doppler echocardiography immediately before and after HD. The cardiac size, volume and mass were determined by M-mode and two-dimensional echocardiography. LV diastolic filling parameters and hemodynamics were assessed from mitral inflow using Doppler echocardiography. *Results.* Left atrial and LV dimension, LV volume, and LV mass decreased significantly after HD ($p<0.001$). Cardiac output declined from 5.74 ± 1.37 to 4.98 ± 1.27 L/min ($p<0.001$), whereas, the ejection fraction remained unchanged. HD elicited marked changes in the early diastolic E (95.1 ± 20.5 to 70.3 ± 18.2 cm/s, $p<0.001$) and late atrial filling A velocities (104.3 ± 20.9 to 88.9 ± 16.9 cm/s, $p<0.001$). In addition, correction of the deceleration time of E and isovolumic relaxation time prolonged significantly ($p=0.011$ and $p<0.001$, respectively). *Conclusions.* Findings in this study indicate that HD altering the loading condition significantly influenced the LV diastolic function and hemodynamics. Moreover,

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Doppler echocardiography provides an effective means of assessing the effects on LV diastolic filling and hemodynamics during HD.

Key Words: Hemodialysis; End stage renal disease; Echocardiography; Diastolic filling.

INTRODUCTION

Cardiovascular disease is the leading cause of morbidity and mortality among patients with end-stage renal disease (ESRD) on maintenance hemodialysis (HD).[1,2] Such patients have been found to have left ventricular (LV) hypertrophy, irrespective of blood pressure, and LV diastolic dysfunction with normal systolic function.[3–6] Having been validated and refined, Doppler echocardiography is the major clinical modality for noninvasively evaluating diastolic filling patterns.[7–10] Recent studies have demonstrated the impact of HD on LV diastolic filling.[6,11–17] However, they have yielded contradictory results. Therefore, this study investigates how altered volume loading affects LV diastolic filling indices during HD. The changes of heart size, volume, mass and hemodynamics are addressed as well.

MATERIALS AND METHODS

Subjects

The study population consisted of 40 patients (22 females and 18 males), with a mean age of 52.7 years old (range of 15 to 71 years old) who were on maintenance HD. Exclusion criteria included the presence of atrial fibrillation and bundle branch block, any degree of atrioventricular block, significant mitral or aortic valve disease, pericardial disease and inadequate echocardiographic imaging. All subjects gave the informed consent.

Hemodialysis

All study patients underwent their routine HD three times weekly. HD session was performed for 4 hours using a dialyzer, with blood flow rate 250–300 mL/min and dialysate flow 500 mL/min. Composition of dialysate was 140 mEq/L of sodium ion, 2.0 mEq/L of potassium ion, 3.0 mEq/L of calcium ion, 1.0 mEq/L of magnesium ion, 105 mEq/L of chloride, 35 mEq/L of bicarbonate. Heparin was used as an anticoagulant. Dry weight was defined as the weight on which the patient had a heart size (chest X-ray) as close as possible to normal, which was clinically tolerated, and/or the weight below which despite high dialysate sodium, the patient developed cramps and/or orthostatic hypotension in the interdialytic interval. The end point of dialysis was the attainment of the patients' determined ''dry weight.''

Echocardiography

Transthoracic echocardiography was performed using commercially available ultrasound equipment (System FiVe, GE Vingmed, Horten, Norway) with a 3.5 MHz phased array transducer. All patients were examined in the left decubitus position within 30 minutes before and immediately after HD.

Left atrial, aortic and LV diameters, and wall thickness were measured from two-dimensional targeted M-mode echocardiography using the criteria of the American Society of Echocardiography.[18] Fractional shorting was calculated using the standard formula.[19] Next LV end-diastolic, end-systolic volumes and ejection fraction were computed using a modified Simpson biplane method.[19] LV mass was then determined by Devereux formula,[20] and LV mass index was calculated by LV mass/body surface area (BSA).[21]

The mitral flow velocities were recorded with pulsed-wave Doppler with the sample volume place at the tip of mitral valve from the apical four-chamber view. From the mitral inflow velocity curve the following measurements were obtained: peak mitral inflow velocity at early (E) and late (A) diastole, deceleration time (DT) of E, duration of E and A, and velocity-time integral (VTI) of E and A. Next, the isovolumic relaxation time (IVRT) was measured from aortic valve closure to mitral valve opening. All durations were corrected and divided by RR interval$^{1/2}$. Stroke volume was then determined as described elsewhere.[22] Cardiac output (CO) was obtained by multiplying the stroke volume by heart rate and cardiac index (CI) was calculated by dividing CO by BSA. The total vascular resistance (TVR) expressed as unit of resistance was calculated by TVR=mean BP/CO. Average values of echocardiographic indices, as obtained from three consecutive cardiac cycles, were used for analysis.

Table 1. Baseline characteristics of study subjects.

Number	40
Age, y	52.7±13.6
Male	18 (45.0%)
Etiology of chronic renal failure	
Chronic glomerulonephritis	26 (65.0%)
Diabetes mellitus	8 (20.0%)
Systemic lupus erythematosus	3 (7.5%)
Polycystic kidney disease	1 (2.5%)
Renal cancer	1 (2.5%)
Bladder cancer	1 (2.5%)
Associated cardiovascular disease	
Hypertension	25 (62.5%)
Coronary artery disease	2 (5.0%)
Heart failure	1 (2.5%)
Vasoactive medications	
Beta-blocker	6 (15.0%)
Alpha and beta-blocker	7 (17.5%)
Calcium channel blocker	10 (25.0%)
Angiotensin II blocker	5 (12.5%)
Diuretics	4 (10.0%)
Nitrate	9 (22.5%)
Body height, cm	159.2±7.0
Body weight, kg	59.2±10.7
Body surface, m^2	1.61±0.16

Assessment of Reproducibility

Intraobserver and interobserver reproducibilities were assessed in 10 randomly selected subjects. The latter involved the second observer acquiring as well as measuring the data. Variability was expressed as the mean percentage error, derived as the absolute difference between the two sets of observations and divided by the mean of the observations. All data were presented to the observers in a blind, random manner; they were completely independent of each other. The mean intraobserver difference was 2.6%, and the mean interobserver difference was 5.1%.

Statistical Analysis

Values are presented as mean±SD for continuous variables and as percentage for categorical data. Paired t-tests were conducted to compare intra-individual changes of paired data, which variables included hemodynamic, M-mode, two-dimensional and Doppler echocardiographic measurements before and after HD. $P<0.05$ was considered statistically significant. The statistical software SPSS 11.0 ([SPSS 2003]) was used here for statistical analysis.

RESULTS

Patient Characteristics

44 HD patients were initially screened for the study, of which four were excluded (2 with atrial fibrillation and 2 with inadequate echocardiographic imaging). Table 1 presents clinical characteristics of the final study group of 40 patients. The etiology of renal failure was chronic glomerulonephritis in 26 patients, diabetes mellitus in 8, lupus nephritis in 3, polycystic kidney disease in 1, renal cancer in 1, and bladder cancer in the remaining one. 25 patients had a history of hypertension, 2 had coronary artery disease, and one had heart failure. Vasoactive medications prescribed included

Table 2. Effects of hemodialysis on heart size, left ventricular volume and mass.

	Before	After	*P*
Aorta, mm	31.5±3.7	30.4±3.5	0.009
Left atrium, mm	37.6±6.1	31.8±5.8	<0.001
LVOT, mm	20.3±1.7	19.9±1.5	0.018
Interventricular septum, mm	12.6±3.0	11.8±2.9	0.021
LVPW, mm	10.3±1.9	10.1±1.8	0.476
LVEDD, mm	48.3±5.5	43.0±5.9	<0.001
LVESD, mm	28.8±5.7	25.0±6.6	<0.001
FS,%	40.7±7.8	42.5±9.0	0.156
LVEDV, mL	111.0±29.7	85.3±28.4	<0.001
LVESV, mL	33.6±16.8	25.2±17.5	<0.001
LV mass, g	215.3±66.7	171.0±65.7	<0.001
LV mass index, g/m^2	133.7±39.5	107.7±40.9	<0.001

LVOT: left ventricular outflow tract; LVPW: left ventricular posterior wall; LVEDD: left ventricular end-diastolic dimension; LVESD: left ventricular end-systolic dimension; FS: fractional shortening; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume; LV: left ventricular.

Table 3. Hemodynamic changes before and after hemodialysis.

	Before	After	*P*
Systolic BP, mm Hg	144.8±31.7	123.7±28.2	<0.001
Diastolic BP, mm Hg	79.9±11.3	71.7±13.4	<0.001
Heart rate, min^{-1}	78.1±11.5	83.4±11.9	0.002
Body weight, kg	59.2±10.7	56.6±10.4	<0.001
LVEF,%	72.7±8.5	73.4± 9.5	0.335
Stroke volume, ml	73.7±14.5	60.0±13.9	<0.001
Stroke volume index, mL/m^2	46.1±9.4	38.2±9.6	<0.001
Cardiac output, L/min	5.74±1.37	4.98±1.27	<0.001
Cardiac index, $L/min/m^2$	3.60±0.96	3.19±0.92	0.001
TPR, $dyn.sec.cm^{-5}$	1497±466	1513±475	0.800

BP: blood pressure; LVEF: left ventricular ejection fraction; TPR: total peripheral resistance.

beta-blockers (6 patients), alpha- and beta-blockers (7 patients), calcium channel blockers (10 patients) angiotensin II blockers (5 patients), diuretics (4 patients), and nitrates (9 patients). The mean body height was 159.2±7.0 cm, mean body weight of 59.2±10.7 kg, and mean BSA of 1.61±0.16 m2.

Heart Size, LV Volume, and Mass

Table 2 lists the effects of HD on heart size, LV volume and mass. After HD, the dimension of aorta, left atrium and LV outflow tract significantly decreased. Similarly, there was a significant reduction in LV end-diastolic and end-systolic dimension, as well as volume ($p<0.001$). However, the slight increase of LV fractional shorting was statistically insignificant (40.7±7.8% to 42.5±9.0%, $p=0.16$). On the other hand, both LV mass and LV mass index decreased by about 20% ($p<0.001$).

Hemodynamics

Table 3 summarized the hemodynamic changes, indicating that HD incurred a reduction in systolic and

Table 4. Effects of hemodialysis on left ventricular diastolic filling parameters.

	Before	After	*P*
E, cm/s	95.1±20.5	70.3±18.2	<0.001
A, cm/s	104.3±20.9	88.9±16.9	<0.001
E/A ratio	0.93±0.21	0.80±0.20	<0.001
DT of E, ms	192.2±50.0	212.9±64.9	0.044
DTc of E, ms	217.0±50.4	247.3±69.3	0.011
IVRT	90.1±23.9	106.6±22.7	<0.001
IVRTc	101.4±23.4	124.7±24.3	<0.001
E dur, ms	244.9±43.0	240.6±59.2	0.627
E dur.c, ms	276.5±38.7	280.1±61.8	0.701
A dur, ms	151.8±27.6	153.3±22.9	0.805
A dur.c, ms	171.7±29.2	179.9±28.1	0.236
E dur/A dur ratio	1.66±0.36	1.60±0.43	0.486
E+A dur, ms	378.0±97.9	355.6±83.5	0.126
E+A dur.c, ms	422.7±80.9	412.3±74.1	0.409
E_{VTI}, cm	13.90±3.10	10.54±3.56	<0.001
A_{VTI}, cm	9.25±2.36	8.24±1.75	0.003
E_{VTI}/A_{VTI} ratio	1.59±0.54	1.32±0.48	0.003

E: peak mitral inflow velocity at early diastole; A: peak mitral inflow velocity at late diastole; DT: deceleration time; DTc: correction of DT; IVRT: isovolumic relaxation time; IVRTc: correction of isovolumic relaxation time; E dur: duration of E; E dur.c: correction of E duration; A dur: duration of A; A dur.c: correction of A duration; E+A dur: total duration of E and A; E+A dur.c: correction of total duration of E and A; E_{VTI}: velocity-time integral of E; A_{VTI}: velocity-time integral of A.

diastolic blood pressure ($p<0.001$), as well as an increase in heart rate ($p=0.002$). Following dialysis, body weight significantly decreased (59.2±10.7 to 56.6±10.4 kg, $p<0.001$). Although LV ejection fraction remained unchanged after HD ($p=0.335$), the stroke volume (73.7±14.5 to 60.0±13.9 ml, $p<0.001$), stroke volume index (46.1±9.4 to 38.2±9.6 mL/m^2, $p<0.001$), CO (5.74±1.37 to 4.98±1.27 L/min, $p<0.001$) and CI (3.60±0.96 to 3.19±0.92 L/min/m^2, $p<0.001$) all reduced significantly. However, TPR increased slightly, which was statistically insignificant.

LV Diastolic Filling

Table 4 summarized the effects of HD on LV diastolic filling parameters. According to this table, mitral inflow velocity E (95.1±20.5 to 70.3±18.2 cm/s, $p<0.001$), A (104.3±20.9 to 88.9±16.9 cm/s, $p<0.001$) and E/A ratio (0.93±0.21 to 0.80±0.20, $p<0.001$) decreased during HD, all of which were statistically significant. The correction of DT of E and IVRT increased significantly (217.0±50.4 to 247.3±69.3 msec, $p=0.011$ and 101.4±23.4 to 124.7±24.3 msec, $p<0.001$ respectively). However, the duration of E, A, E and A, as well as the ratio of E/A duration remained constant (p=ns). The VTI of transmitral flow E (13.90±3.10 to 10.54±3.56 cm, $p<0.001$) and A (9.25±2.36 to 8.24±1.75 cm, $p=0.003$) decreased significantly after HD, with a significant reduction in the ratio of E_{VTI}/A_{VTI} (1.59±0.54 to 1.32±0.48, $p=0.003$).

DISCUSSION

This study has demonstrated that HD-related volume reduction incurred changes in heart size, volume, mass and hemodynamics. In addition, HD affects LV diastolic filling parameters as well. These findings indicate that a reduction in preload accounts for changes in heart size, volume and LV diastolic filling indices, and further influence on hemodynamics.

Dialysis-Induced Changes of Diastolic Filling

In this study, HD-induced acute volume reduction impacted significantly mitral inflow parameters, primarily affecting early LV diastolic filling (reduction in mitral inflow E velocities). These findings resembled those in previous studies that evaluated the effect of HD on echocardiographic parameters of LV filling.[6,11–17] However, those studies yielded conflicting results in terms of variations in DT of E, late diastolic A velocities, and IVRT after HD.

Rozich et al.[11] performed echocardiographic evaluation before and after HD of 16 patients with ESRD, indicating that the rate and extent (VTI) of early rapid LV diastolic filling velocities E were significantly reduced, as well as the IVRT prolonged; conversely, late atrial-assisted filling A velocities only changed slightly. Sztajzel et al.[13] observed similar findings in 12 patients undergoing HD. On the other hand, Agmon et al.[17] analyzed Doppler echocardiography in 13 patients receiving HD, indicating that after HD both mitral inflow E and A velocities decreased significantly; however, IVRT did not. However, in our series, E and A velocities declined and IVRT prolonged significantly after HD. With respect to the DT of E velocities, Sadler et al.[12] observed that it prolonged markedly after HD in 24 patients. This study confers those findings. In contrast, previous studies offered conflicting results.[14,15]

Mechanism of Altered Diastolic Filling During HD

Changes in diastolic filling after HD in this study were namely significant reduction in early E and late A velocities, slowing of DT, prolongation of IVRT and decreased in VTI of diastolic filling waves. Consequently, the ratios of E/A and E_{VTI}/A_{VTI} decreased substantially during HD. The proportion of LV filling during the early and late diastolic phases depends on the elastic recoil, rate of myocardial relaxation, chamber compliance, atrial pressure, and heart rate.[17,24,26–29] The status of these variances is the result of the interaction among the cardiac disease process, baseline diastolic properties, and volume status. The changes of LV diastolic filling observed after HD may reflect two mechanisms: a reduction in left atrial pressure with decreased transmitral pressure gradient and impairment of LV relaxation. With respect to the first mechanism, ultrafiltration during HD markedly reduced the circulating blood volume with a concomitant reduction in left atrial pressure. Correspondingly, Kinet et al.[23] noted the lowering of the left atrial pressure during HD as well as a reduction in pulmonary capillary wedge pressure from an average of 12.5 mm Hg to 3.1 mm Hg. Decreased left atrial pressure also reduced the difference in pressure between left atrium and ventricle. Courtois et al.[24] observed that the ability of inferior vena caval occlusion to suddenly reduce left atrial pressure significantly altered the transmitral pressure relation and, in doing so, profoundly affected the transmitral

Doppler flow velocity profile during both the early and late phases of diastole. With respect to the second mechanism, the higher serum calcium concentrations detected by patients during HD may have affected myocardial relaxation.[25] correspondingly, this study also found a significant positive correlation between the percentage change in serum calcium after dialysis and the increase in IVRT. Importantly, diastolic filling in the LV is an extremely complex process, particularly with respect to the rate of LV relaxation, myocardial stiffness, pericardial constraint, and ventricular interaction.[24,26–29]

Dialysis-Induced Hemodynamic Changes

Four distinct and interrelated factors, i.e., preload, afterload, contractility and heart rate determine and regulate CO. In this study, HD significantly reduced preload but mildly increased the heart rate. Notably, ejection fraction and TPR did not change indicating that the changes of CO and stroke volume after HD were largely owing to preload reduction, irrespective of afterload, contractility and heart rate.

CONCLUSIONS

This study demonstrated that HD alters the loading conditions that significantly change LV diastolic function and hemodynamics. Doppler echocardiography is a highly promising means of assessing the effects of various protocols of HD on LV filling and ejection. Results of this study may also provide direct evidence for a potentially important mechanism whereby some patients become hemodynamically unstable during routine HD. Further study is warranted to identify those patients who are likely to become hemodynamically brittle during HD.

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