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Study of Respiratory Variations of Mitral Valve Diastolic Flow in Hemodialysis Patients

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Abstract

The respiratory variations in mitral valve (MV) Doppler flow in hemodialysis (HD) patients have not been investigated and the normal echocardiographic value is used as a reference for HD patients. The present study evaluated the respiratory variation in MV Doppler flow in HD patients to determine if it has a unique pattern. In this prospective cohort study, echocardiography was performed before and 6 h after dialysis. The transmitral spectral Doppler E wave was measured during inspiratory and expiratory phases. The percent changes in the E wave were calculated pre- and post-dialysis. The means of the percent variation in the MV inspiratory and expiratory E wave pre- and post-dialysis were 56 ± 7% and 44 ± 1.1%, respectively, with a significant reduction after dialysis (P = .000). There was a significant positive correlation between post-dialysis ΔE wave % change and post-dialysis % change in weight (r = .318; P = .000). The respiratory changes in the MV E wave in HD patients were higher than the normal reference values. This marked variation could be explained by fluid overloading in HD patients.

Keywords

respiratory variations, mitral valve diastolic flow, hemodialysis, volume status in hemodialysis

Introduction

Hemodialysis (HD) patients have high cardiovascular (CV) morbidity and mortality, and their adjusted risk of death is about 10–20 times higher than that of the general population.[1] Echocardiography is a noninvasive imaging technique that has been extensively used to study the morphological and the functional cardiac changes in HD patients.[2] The echocardiographic parameters that are commonly measured are left ventricular (LV) systolic, LV diastolic function, right ventricle (RV) function, and pulmonary hypertension. The changes in these parameters have been linked to both traditional CV risk factors, which are more prevalent in HD patients, and uremic milieu-related factors.[3]

Respiration induces cyclic physiological modification of intracardiac hemodynamics. Moreover, as the pericardium has a constraining effect on the combined volume of the four cardiac chambers, respiratory variation in intrapericardial pressure results in reciprocal variation in the filling of both ventricles.[4] With inspiration, intrathoracic and intrapericardial pressures decrease.[5] This augments right ventricular filling and stroke volume and, as the total pericardial space is limited, a compensatory decrease in left ventricular stroke volume occurs in inspiration. With expiration, the opposite occurs. Under normal circumstances, the Doppler peak velocity of mitral valve (MV) inflow varies by ≤ 15% with respiration and tricuspid inflow by ≤ 25%.[5]

The respiratory variation in MV Doppler flow in HD patients has not been investigated, and the normal adult referenced echocardiographic value of respiratory variation in MV Doppler flow is used as an echocardiographic reference for HD patients.[5] However, HD patients have unique hemodynamics related to the pre-dialysis fluid overloading and the cyclic changes in their volume status induced by ultrafiltration.[6]

The present study aimed to evaluate the respiratory variation in MV Doppler flow in HD patients and to assess any relation between these variations and volume-related parameters.

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Patients and Methods

Study Design

This was a prospective cohort study, carried out on HD patients who underwent regular HD in the Nephrology Unit.

Ethical Considerations

The study was approved by the Ethical Committee (No: 17101878 - October 18, 2016). Written informed consent was obtained from the patients before their inclusion; illiterate participants gave their consent by fingerprints.

Patients

This study was carried out on 118 regular HD patients from January 2018 to September 2018. Eligible patients were adults aged ≥18 years.

Exclusion criteria were patients with poor echo window, non-sinus rhythm, congenital heart disease, rheumatic heart disease, left ventricular systolic dysfunction (ejection fraction; EF < 40%), impaired RV systolic function (tricuspid annular plane systolic excursion (TAPSE) < 1.5 cm), pericardial effusion, obviously thickened and/or echogenic pericardium, calcific pericardium with lateral chest x-ray, patients with chronic lung disease (either by history, abnormal imaging, abnormal arterial blood gases, or pulmonary function test), mediastinal syndrome, and patients with pneumonia or respiratory distress.

Methods

All patients underwent echocardiography with an HDI 5000 instrument (Philips Medical Systems, Bothell, Washington, USA) equipped with a broadband sector array transducer (Frequency range: 4–2 MHz). All echocardiographic examinations were carried to the patients before the HD sessions and within 6 h after the sessions. Standard echocardiography was carried out at the Internal Medicine Department and were based on apical four and two-chamber views. LV dimensions were calculated using 2D guided M-mode calculations. The mean value of three measurements of the technically best cardiac cycles was used from each examination. LV EF was measured by 2D guided M-mode calculations. LV diastolic function was calculated using: transmitral E wave velocity using pulsed Doppler with a sample size of .7 mm just above MV leaflets tips, lateral MV annular e' wave measured by tissue Doppler with a sample size of 4.0 mm at the level of lateral MV annulus, and E/e' velocity ratio. RV systolic function was measured as TAPSE using M-mode.

During quiet breathing, the peak transmitral spectral Doppler E wave was measured during inspiratory and expiratory phases. After at least 3 respiratory cycles with the aid of plethysmographic breath cycle chest adhesive electrodes of a separate monitor (GE Medical Systems, Milwaukee, Wisconsin, USA), the peak of the best and complete E wave velocity envelope in each respiratory phase was measured.

The pre- and post-dialysis respiratory percent changes of MV E wave were calculated as follows: expiratory MV E wave – inspiratory MV E wave/expiratory MV E wave x 100. Post-dialysis difference in the percent changes (Δ E wave % changes) was calculated as follows: pre-dialysis percent changes of MV E wave – post-dialysis percent changes of MV E wave/pre-dialysis percent changes MV E wave x 100. While the pre- and post-dialysis respiratory percent changes of TV E wave were calculated as follows: inspiratory TV E wave – expiratory TV E wave/inspiratory TV E wave x 100.

Hemodialysis/Ultrafiltration Volume Data

HD was performed using Fresenius 5008 therapy system (Fresenius Medical Care, Bad Homburg vor der Höhe, Germany). The average duration of dialysis was about 4 h. Dialysate temperature was kept constant at 37 C with dialysate ion-concentrations consisting of Na^+ 140 mmol/L, HCO^{3-} 38 mmol/L, K^+ 2.0 mmol/L, and Ca^{2+} 1.25 mmol/L. The filters used were PF 170 or PF 210 from Gambro (Deerfield, Illinois, U.S). The average blood flow was 300 mL/m. Dialysate flow was set at 500 mL/m. Blinded resident nephrologists, guided by the standard HD unit protocols, estimated the ultrafiltration volumes of the included patients.

Data Collection

Patient demographic data and medical history including age, sex, and comorbid diseases were recorded. Body mass index (BMI) was calculated as (weight (kg)/height^2 (m)). The following clinical variables were measured before and within 6 h after dialysis: systolic and diastolic blood pressure by mercurial sphygmomanometer in a head and arm supported supine position after 10 min of rest; heart rate; the presence of lower limb edema; the presence of congested neck veins; and the presence of basal lung rales. Venous blood samples were collected from the studied patients before and after the end of dialysis sessions to measure the following: serum levels of creatinine (mg/dl), urea (mg/dl), Hb (g/dl), Na (mmol/l), and K (mmol/l) levels. The transducer (frequency range: 4-2 MHz) was used to measure inferior vena cava (IVC) diameter. The probe was placed in the subxiphoid location when the patients were supine. The IVC was measured 2 cm caudal to the junction point of the hepatic vein and IVC. Both the inspiratory and respiratory diameters were detected by measuring the vein lumen at 1 respiratory cycle, from 1 interior wall to the opposite interior wall. Percent changes of these variables were calculated as follows: pre-dialysis value – post-dialysis value/post-dialysis value %.

Statistical Analysis

The statistical analysis was performed using SPSS (version 22.0, SPSS Inc., Chicago, IL, USA). The Kolmogorov–Smirnov test was used to test normality. The continuous variables were presented as the means ± SD or median and range, and categorical variables were presented as frequency and percentage. The Chi-square test and Fisher Extract test were used to compare qualitative parameters. The paired t-test was used to determine changes between quantitative parameters before and after dialysis in the case of parametric variables and the Wilcoxon Signed-Rank test in the case of non-parametric variables. Spearman correlation was used to test the presence of correlation between Δ E wave % change and % change of weight. We randomly selected 20 echocardiograms to assess variability. The echocardiography variables were measured by two independent echocardiographers. Interobserver variability (the mean of absolute differences between two measurements) in the evaluation of peak transmitral spectral Doppler E wave measured during inspiratory and expiratory phases was 4.8 ± 2.8% and 6.9 ± 6.3%, respectively. Univariate regression analysis was used to detect the relations between Δ E wave % changes and other variables, while multivariate regression analysis was used to detect the independent predictors of Δ E wave % changes. A 2-sided $P < .05$ was considered statistically significant.

Table 1. Demographic data and risk factors of the study population.

	No. (118)	%
Age		
<50	47	39.8
50–60	39	33.1
>60	32	27.1
Female	50	42.4
BMI:		
Normal (< 25 kg/m^2)	74	62.7
Overweight (> 25 kg/m^2)	44	37.3
Risk factors:		
HTN	84	71.2
D.M	48	40.7
Smoking	44	37.3

BMI: Body mass index (kg/m^2), HTN: systemic hypertension, DM: diabetes mellitus

Results

Demographic Characteristics

The mean age of the study population was 53.5 ± 9.5 years. Other demographic data are shown in Table 1.

Clinical and Laboratory Data

The mean ultrafiltration dose was 3.27 ± 1.53 L/session. A part of heart rate, which showed a significant reduction after dialysis, there were statistically significant reductions of post-dialysis systolic blood pressure (BP), diastolic BP, weight, urea, creatinine, and serum K^+. While there was no case with pulmonary congestion after dialysis and no significant reduction regarding lower limb edema, the presence of pleural effusion, serum Na^+, and hemoglobin (Hb) (Table 2).

Pre- and Post-Dialysis Echocardiographic and IVC data

There were significant reductions in all cardiac chamber dimensions, inspiratory and expiratory trans-mitral and trans-tricuspid Doppler flow waves, pulmonary artery systolic pressure (PASP), E/e' ratio, and both inspiratory and expiratory IVC diameters. While there were significant increases in LV EF, wave velocity, e' wave velocity, and TAPSE after dialysis (Table 3).

Respiratory Changes

The means of the percent variation in the MV inspiratory and expiratory E pre- and post-dialysis were 56 ± 7% and 44 ± 1.1%, respectively, with a significant reduction after dialysis; P = .000

Table 2. Clinical and laboratory data pre- and post-dialysis.

	Pre-dialysis	Post-dialysis	p
Systolic BP (mmHg), *mean ± SD*	137.4 ± 14.8	120.1 ± 8.8	.000
Diastolic BP (mmHg), *mean ± SD*	89.1 ± 9.6	78.6 ± 5.7	.000
Heart rate (beats/min), *mean ± SD*	73.4 ± 4.7	80.5 ± 5.9	.000
Lower limb edema, *n (%)*	11 (9.3%)	11 (9.3%)	1.000
Pleural effusion, *n (%)*	6 (5.1%)	6 (5.1%)	1.000
Pulmonary congestion, *n (%)*	30 (25.4%)	0 (.0%)	.000
Weight (kg), *mean ± SD*	71.4 ± 6.9	68.6 ± 6.8	.000
Creatinine (mg/dl), *mean ± SD*	10.4 ± 2.2	4.8 ± 1.0	.000
Urea (mg/dl), *mean ± SD*	138.4 ± 25.0	59.6 ± 13.5	.000
Hb (g/dl), *mean ± SD*	8.9 ± 0.9	8.7 ± 0.8	.203
Na (mmol/l), *mean ± SD*	137.4 ± 3.9	136.9 ± 1.7	.180
K (mmol/l), *mean ± SD*	4.6 ± 0.5	3.3 ± 0.2	.000

BP: blood pressure, Hb: hemoglobin, Na: serum sodium, K: serum potassium,

Table 3. Echocardiographic data pre- and post-dialysis.

	Pre-dialysis	Post-dialysis	p
MV inspiratory E wave (m/s), *mean ± SD*	1.19 ± .16	.93 ± .28	.000
MV expiratory E wave (m/s), *mean ± SD*	1.25 ± .20	1.00 ± .44	.000
MV E wave respiratory variations (%), *mean ± SD* *	56 ± 7 (36 - 74)+	44 ± 1.1 (14 - 68)+	.000
A (m/sec), *mean ± SD*	.83 ± .25	.99 ± .20	.000
e' (m/sec), *mean ± SD*	.05 ± .02	.16 ± .27	.000
E/e', *mean ± SD*	13.83 ± 6.11	6.64 ± 3.40	.000
TV inspiratory E wave (m/s), *mean ± SD*	2.34 ± .21	1.43 ± .21	.000
TV expiratory E wave (m/s), *mean ± SD*	1.64 ± .27	1.01 ± .24	.000
TV E wave respiratory variations (%) ^, *mean ± SD*	67 ± 18 (41-81)+	54 ± 17 (21-71)+	.000
LAD (cm), *mean ± SD*	3.69 ± .67	3.00 ± .62	.000
RVEDD (cm), *mean ± SD*	1.97 ± .17	1.77 ± .18	.000
LVEDD (cm), *mean ± SD*	56.75 ± 7.86	49.40 ± 6.15	.000
LVESD (cm), *mean ± SD*	36.36 ± 5.11	32.67 ± 5.04	.000
LVEF (%), *mean ± SD*	56.0 ± 5.6	67.7 ± 4.8	.000
TAPSE (cm), *mean ± SD*	2.50 ± .44	2.77 ± .56	.001
End inspiratory IVCD (cm), *mean ± SD*	1.21 ± .32	1.01 ± .33	.000
End expiratory IVCD (cm), *mean ± SD*	1.77 ± .30	1.44 ± .30	.000
PASP (mmHg), *mean ± SD*	20.25 ± 2.66	15.30 ± 2.49	.000

MV: Mitral valve, TV: tricuspid valve, LAD: Left atrium diameter, RVEDD: Right ventricle end-diastolic diameter, LVEDD: Left ventricle end-diastolic diameter, LVESD: Left ventricle end-systolic diameter, LVEF: Left ventricular ejection fraction, TAPSE: Tricuspid annular plain systolic excursion, IVCD: Inferior vena cava diameter, PASP: Pulmonary artery systolic pressure.* MV respiratory percent changes; mean, standard deviations, and range.^ TV respiratory percent changes; mean, standard deviations, and range.+ range.

(Figure 1) (Table 3). The post-dialysis Δ MV E wave % change after dialysis was 22.74 ± 14.98 (−15.25-70.21). The means of percent variation in the TV inspiratory and expiratory E pre- and post-dialysis were 67 ± 18% and 54 ± 17%, respectively, with a significant reduction after dialysis (P = .000). The post-dialysis Δ TV E wave % change after dialysis was 31.21 ± 17.38 (−13.62-82.31) (Table 3).

Correlation and Regression Analysis

Spearman correlation showed a significant positive correlation between post-dialysis Δ E wave % change and % change of weight (Figure 2). Moreover, univariate analysis showed a significant relation between post-dialysis Δ E wave % change and percent change of weight, systolic BP, diastolic BP, plasma urea, Hb, serum Na+, and both inspiratory and expiratory IVC diameters (Table 4). Multivariate analysis showed that the independent predictors of post-dialysis Δ E wave % change were the change of weight, systolic BP, diastolic BP, plasma urea, creatinine, Hb, serum Na^+, serum K^+, LV EDD, PASP, and both inspiratory and expiratory IVC diameters (Table 5).

Discussion

The main findings of the present study are: (1) the means of the percent respiratory variation in MV E wave were 56 ± 7% pre-HD, and 44 ± 1.1% post-HD, (2) there was a significant reduction in Δ E wave % change after HD, (3) the post-HD Δ E wave % change was significantly related to volume-load reduction parameters after HD.

Both left and right ventricles are interdependent because they are enclosed within the pericardium and have the septum in common.[7,8] During spontaneous inspiration, increased venous return to the right heart increases the right ventricular end-diastolic volume (RVEDV) which shifts the septum to the left and reduces left ventricular end-diastolic volume (LVEDV).[9] It is well known that with spontaneous respiration small changes (< 15%) in trans-mitral peak flow velocities occur in healthy subjects.[5]

The present study detects higher percent variations in MV E wave than what has been referenced for normal adults. The exaggerated ventricular interdependence in HD patients could be explained by the increase in the degree of pericardium constrain in HD patients.[9] This could be produced by uremia and other subtle changes in the pericardial tissue that could not be detected by conventional methods.[10] In a constricting pericardium, the interventricular interaction increases, so that with RV volume increase, there is a corresponding decrease in LV volume.[11] Hypervolemia in patients with constrictive pericarditis reduces the respiratory variation of the Doppler inflows and exaggerated ventricular interdependence may not be detected unless preload is reduced by head-up tilt or diuretics.[12] However, the present study showed that respiratory variations in MV E wave were reduced after ultrafiltration which makes the explanation by pericardial constriction unacceptable.

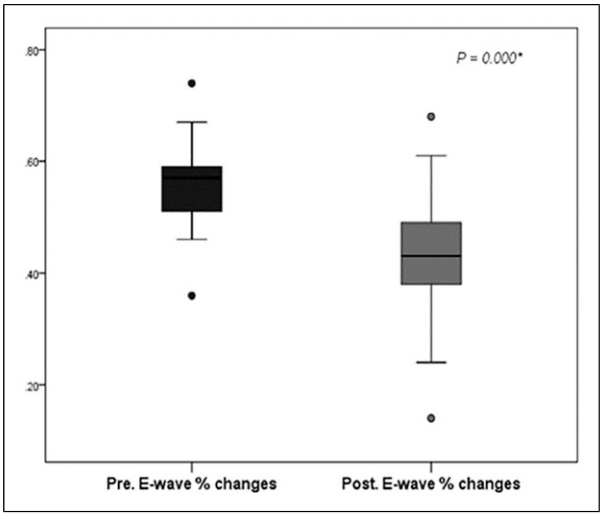

Figure 1. The median and middle quartile of pre- and post-dialysis E-wave percent changes (P = .000) *significant.

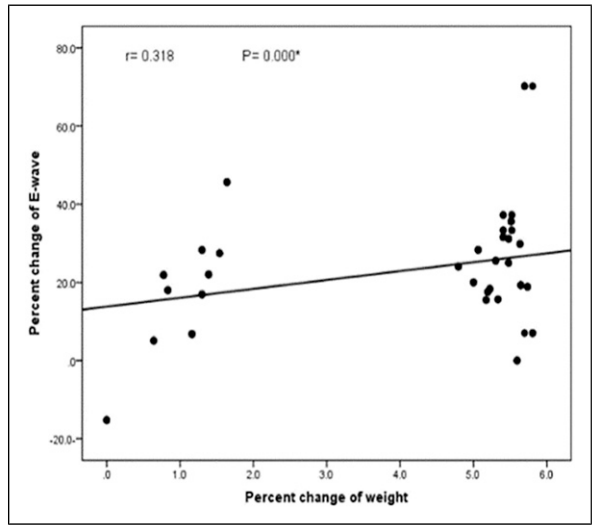

Figure 2. Correlation between ΔE wave % change and weight % change.

Table 4. Univariate linear regression analysis.

			95% CI for B	
	B	p	Lower	Upper
Weight % change (kg)	2.2735	.000	1.027	3.520
Systolic BP % change (mmHg)	.46	.007	.126	.794
Diastolic BP % change (mmHg)	.4735	.002	.176	.771
Creatinine % change (mg/dl)	−.1475	.471	−.550	.255
Urea % change (mg/dl)	−.6395	.009	−1.118	−.161
Hb % change (g/dl)	7.251	.000	4.147	10.355
Na % change (mEq/L)	−1.836	.009	−3.199	−.473
K % change (mEq/L)	.327	.118	−.084	.738
LAD % change (cm)	.3565	.101	−.070	.783
RVEDD % change (cm)	.1845	.367	−.219	.588
LVEDD % change (cm)	.3225	.214	−.188	.833
LVESD % change (cm)	−.5165	.058	−1.051	.018
LVEF % change (cm)	−.366	.060	−.748	.016
End inspiratory IVCD % change (cm)	−1.0035	.000	−1.316	−.691
End expiratory IVCD % change (cm)	1.4575	.000	.933	1.982
PASP % change (mmHg)	−.4975	.172	−1.215	.220

Dependent variable: ΔE wave % changesB = Unstandardized CoefficientsBP: blood pressure; Hb: hemoglobin; Na: serum sodium; K: serum potassium; LAD: Left atrium diameter; RVEDD: Right ventricle end-diastolic diameter; LVEDD: Left ventricle end-diastolic diameter; LVESD: Left ventricle end-systolic diameter; LVEF: Left ventricular ejection fraction; IVCD: Inferior vena cava diameter; PASP: Pulmonary artery systolic pressure.

Another explanation of the exaggerated ventricular interdependence in HD patients could be that: the interdialytic weight gain could mainly have a predominant effect on the RV which causes its significant inspiratory expansion that in turn makes an inspiratory shift in the IVS shifts to the LV. This explanation is supported by the significant reduction in the percent variation after dialysis. However, this explanation is not supported by any previous work and should be a focus of further studies.

It is well known that echocardiographic measurements are affected by the changing LV loading conditions during HD.[13-16] The present study could point to another cardiac overloading consequence of interdialytic weight gain which is the exaggerated ventricular interdependence. The hemodynamic effect of this exaggerated respiratory variation, especially before HD, may add another cause of the impairment of the LV diastolic function in HD.[13-18] Chronic impairment in LV filling can lead to pulmonary congestion which in turn can cause secondary pulmonary

Table 5. Multiple linear regression analysis.

			95% CI for B	
	B	p	Lower	Upper
Weight % change (Kg)	.741	.000	.421	1.061
Systolic BP % change (mmHg)	−.184	.004	−.308	−.060
Diastolic BP % change (mmHg)	.225	.000	.117	.333
Creatinine % change (mg/dl)	.2665	.000	.149	.384
Urea % change (mg/dl)	−.406	.000	−.533	−.279
Hb % change (g/dl)	.7325	.315	−.706	2.171
Na % change (mEq/L)	−.3045	.085	−.652	.043
K % change(mEq/L)	−.1505	.002	−.246	−.055
LAD % change (cm)	−.1555	.001	−.247	−.064
RVEDD % change (cm)	−.0825	.198	−.209	.044
LVEDD % change (cm)	−.3345	.000	−.454	−.215
LVESD % change (cm)	−.146	.043	−.287	−.005
LVEF % change (%)	.2335	.001	.095	.372
End inspiratory IVCD % change (cm)	−1.94	.000	−2.027	−1.853
End expiratory IVCD % change (cm)	2.9555	.000	2.752	3.159
PASP % change (mmHg)	−.237	.016	−.430	−.044

Dependent variable: ΔE wave % changesB = Unstandardized CoefficientsBP: blood pressure; Hb: hemoglobin; Na: serum sodium; K: serum potassium; LAD: Left atrium diameter, RVEDD: Right ventricle end-diastolic diameter; LVEDD: Left ventricle end-diastolic diameter; LVESD: Left ventricle end-systolic diameter; LVEF: Left ventricular ejection fraction; IVCD: Inferior vena cava diameter; PASP: Pulmonary artery systolic pressure.

hypertension. The current findings may unveil one of the hidden mechanisms of idiopathic pulmonary hypertension in HD patients named group 5 'unexplained pulmonary hypertension by the world health organization.[19,20]

In the present study, there was a significant improvement in lung rales post-dialysis. This reflects the effective value of ultrafiltration as an LV unloading therapy and it is in concordance with the American Heart Association/American College of Cardiology and European society of Cardiology guidelines that consider ultrafiltration as a reasonable therapy in decompensated heart failure patients with unresolved congestion who cannot withstand optimal medical therapy and/or hyponatremia.[21,22]

Ultrafiltration during HD comprises intravascular volume mobilization using osmotic or hydrostatic pressure.[23] It has been reported that cardiac chambers dimensions decrease in response to ultrafiltration[13-16,24,25] in agreement with the findings of the present study.

In the current work, there were significant reductions in both IVC inspiratory and expiratory diameters after dialysis. A meta-analysis[26] reported that IVC diameter is consistently low in hypovolemic status when compared with euvolemic. Moreover, IVC diameter was significantly decreased after blood donation.[27] However, during the management of heart failure, there were no significant changes in the IVC size with time.[28] This could be explained by the phenomenon of vascular refilling which means that rapid vascular refill could mask the IVC diameter changes related to volume loss. In the current work, measurement of IVC diameter was carried out within 6 h, which could be fast enough to detect the rapid volume changes before the refill phenomenon could mask it.

Conclusion

The percent respiratory variation in the MV E wave was higher than the normal adult referenced percentage both pre- and post-HD. This marked variation is attributed to fluid overloading and could explain the LV diastolic dysfunction and be used to the assess loading status in HD patients.

Study Limitations and Recommendations

The present study has some limitations. Firstly, it was conducted on HD patients without a control group to compare the respiratory variation between the included patients and the used reference value. Secondly, motion independent imaging should have been included in the study to depict evidence of septal shift during respiratory phases which would confirm the ventricular interdependency. Thirdly, the work was conducted on a mono-ethnic population and its results cannot be used as a generalized reference, so, we recommend studying this phenomenon on a multi-ethnic scale. Fourthly, subclinical pericardial constrain is still a remarkable confounder, so, we recommend using multidetector CT or cardiac MRI to detect any pericardial abnormalities in HD patients to be sure they do not have any subtle pericardial constraint before inclusion. Lastly, the present work was single-center non–outcome-based study.

We suggest conducting a multi-center prognosis-based study to detect the prognostic value of this phenomenon in HD patients.

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Declaration of Conflicting Interests

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References

1. de Jager DJ, Grootendorst DC, Jager KJ, et al. Cardiovascular and non-cardiovascular mortality among patients starting dialysis. JAMA. 2009;302:1782-1789.
2. Loutradis C, Sarafidis PA, Papadopoulos CE, Papagianni A, Zoccali C. The Ebb and Flow of Echocardiographic Cardiac Function Parameters in Relationship to Hemodialysis Treatment in Patients with ESRD. J Am Soc Nephrol JASN. 2018;29:1372-1381.
3. Cozzolino M, Mangano M, Stucchi A, Ciceri P, Conte F, Galassi A. cardiovascular disease in dialysis patients. Nephrol Dial Transplant. 2018;33:iii28-34.
4. Harvey F, Armstrong WF, Ryan T. Feigenbaum's Echocardiography, 6th edn. Can J Cardiol. 2004;23:100-104.
5. Ginghina C, Beladan CC, Iancu M, Calin A, Popescu BA. Respiratory maneuvers in echocardiography: a review of clinical applications. Cardiovasc Ultrasound;7:42.
6. Ekinci C, Karabork M, Siriopol D, Dincer N, Covic A, Kanbay M. Effects of Volume Overload and Current Techniques for the Assessment of Fluid Status in Patients with Renal Disease. Blood Purification. 2018;46:34-47.
7. Friedberg MK, Redington AN. Right versus left ventricular failure: differences, similarities, and interactions. Circulation. 2014;129:1033-1044.
8. Kroeker CA, Shrive NG, Belenkie I, Tyberg JV. Pericardium modulates left and right ventricular stroke volumes to compensate for sudden changes in atrial volume. Am J Physiol-Heart Circ Physiol. 2003;284:H2247-H2254.
9. Sarkar M, Bhardwaj R, Madabhavi I, Gowda S, Dogra K. Pulsus paradoxus. Clin Respir J. 2018;12:2321-2331.
10. Rehman KA, Betancor J, Xu B, et al. Uremic pericarditis, pericardial effusion, and constrictive pericarditis in end-stage renal disease: Insights and pathophysiology. Clin Cardiol. 2017;40:839-846.
11. Nishimura RA. Constrictive pericarditis in the modern era: a diagnostic dilemma. Heart Br Card Soc. 2001;86:619-623.
12. Oh JK, Tajik AJ, Appleton CP, Hatle LK, Nishimura RA, Seward JB. Preload Reduction to Unmask the Characteristic Doppler Features of Constrictive Pericarditis. Circulation. 1997;95:796-799.
13. Sarafidis PA, Kamperidis V, Loutradis C, et al. Haemodialysis acutely deteriorates left and right diastolic function and myocardial performance: an effect related to high ultrafiltration volumes? Nephrol Dial Transplant. 2017;32:1402-1409.
14. Drighil A, Madias JE, Mathewson JW, El Mosalami H, El Badaoui N, Ramdani B, Bennis A. Haemodialysis: effects of acute decrease in preload on tissue Doppler imaging indices of systolic and diastolic function of the left and right ventricles. Eur J Echocardiogr. 2008;9:530-535.
15. Sadler DB, Brown J, Nurse H, Roberts J. Impact of hemodialysis on left and right ventricular Doppler diastolic filling indices. Am J Med Sci. 1992;304:83-90.
16. Fijalkowski M, Koprowski A, Gruchala M, et al. Effect of Preload Reduction by Hemodialysis on Myocardial Ultrasonic Characterization, Left Atrial Volume, and Doppler Tissue Imaging in Patients with End-stage Renal Disease. J Am Soc Echocardiogr. 2006;19:1359-1364.
17. Graham RJ, Gelman JS, Donelan L, Mottram PM, Peverill RE. Effect of preload reduction by haemodialysis on new indices of diastolic function. Clin Sci (Lond). 2003;105:499-506.
18. Assa S, Hummel YM, Voors AA, et al. Changes in left ventricular diastolic function during hemodialysis sessions. Am J Kidney Dis. 2013;62:549-556.
19. Simonneau G, Robbins IM, Beghetti M, et al. Updated clinical classification of pulmonary hypertension. J Am Coll Cardiol. 2009;54:S43-S54.
20. Fathi R, Isbel N, Haluska B, Case C, Johnson DW, Marwick TH. Correlates of subclinical left ventricular dysfunction in ESRD. Am J Kidney Dis. 2003;41:1016-1025.
21. Hunt SA, Abraham WT, Chin MH, et al. Focused update incorporated into the ACC/AHA 2005 guidelines for the diagnosis and management of heart failure in adults a report of the American college of cardiology foundation/American heart association task force on practice guidelines developed in collaboration with the international society for heart and lung transplantation. J Am Coll Cardiol. 2009;53:e1-e90.
22. Mancia G, Fagard R, Narkiewicz K, et al. ESH/ESC guidelines for the management of arterial hypertension: the task force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). J Hypertens. 2013;31:1281-1357.
23. Bergström J. Ultrafiltration without dialysis for removal of fluid and solutes in uremia. Clin Nephrol. 1978;9:156-164.
24. Bornstein A, Gaasch WH, Harrington J. Assessment of the cardiac effects of hemodialysis with systolic time intervals and echocardiography. Am J Cardiol. 1983;51:332-335.

25. Vaziri ND, Prakash R. Echocardiographic evaluation of the effect of hemodialysis on cardiac size and function in patients with end-stage renal disease. Am J Med Sci. 1979;278:201-206.
26. Dipti A, Soucy Z, Surana A, Chandra S. Role of inferior vena cava diameter in assessment of volume status: a meta-analysis. Am J Emerg Med. 2012;30:1414-1419.
27. Lyon M, Blaivas M, Brannam L. Sonographic measurement of the inferior vena cava as a marker of blood loss. Am J Emerg Med. 2005;23:45-50.
28. Spevack R, Al Shukairi M, Jayaraman D, Dankoff J, Rudski L, Lipes J. Serial lung and IVC ultrasound in the assessment of congestive heart failure. Crit Ultrasound J. 2017;9:7.