Prognostic Value of Left Atrial Volume Index in Hemodialysis Patients

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Summary

Objective: To evaluate the prognostic value of left atrial volume index (LAVi) in the clinical course of hemodialysis (HD) patients, compared with previously established echocardiographic and clinical parameters.

Methods: Echocardiograms were obtained from 118 hemodialysis patients, who were then followed for 19 ± 8 months. Study endpoint was a composite of all-cause mortality and nonfatal cardiovascular events. Cox multivariate analysis was used do assess the independent prognostic value of LAVi.

Results: On univariate analysis, LAVi and other clinical and echocardiographic parameters were predictive of prognosis. Multivariate analyses showed that LAVi was an independent predictor of prognosis (hazard ratio 1.03 per ml/m^2, 95% confidence interval: 1.01 to 1.05, p=0.014), and added incremental information to the model containing traditional predictors of cardiovascular risk, such as left ventricular mass, ejection fraction, and clinical variables (p=0.02).

Conclusion: LAVi is an independent predictor of prognosis in HD patients, providing incremental information to traditional clinical and Doppler echocardiographic data. (Arq Bras Cardiol 2007;88(6):568-575)

Key words: Hypertrophy, left ventricular; renal dialysis; risk assessment; echocardiography, Doppler.

Introduction

Cardiovascular disease is the leading cause of death in chronic renal failure patients on renal replacement therapy with hemodialysis (HD)[1,2]. The excess cardiovascular risk in this group is caused by the interaction between traditional risk factors for cardiovascular disease and those related to chronic kidney disease (CKD)[1]. Even though these patients have accelerated atherosclerosis[3], structural cardiac changes such as hypertrophy and left ventricular dilation (resulting in diastolic and systolic dysfunction) are associated with the incidence of congestive heart failure and high morbidity and mortality rates[4,5]. For many decades now Doppler echocardiography has been widely used to evaluate cardiac structure and function, and thus has played a key role in characterizing individuals at higher cardiovascular risk. Former studies using the method to predict cardiovascular risk in hemodialysis population focused primarily on the role of hypertrophy[4,6] and left ventricular (LV) systolic dysfunction[7,8]. It has been recently postulated that left atrial (LA) dilation, best represented by planimetric determination of LA volume index (LAVi) on two-dimensional echocardiography[9], is related to the duration of left ventricular diastolic dysfunction[10,11], and is a powerful marker of cardiovascular risk in the general population[11,12] and in some clinical populations as well[13-16]. As LV diastolic function seems to be impaired in most HD patients, even in those asymptomatic[5], we speculate that LAVi may be useful in stratifying cardiovascular risk in this group. This study was designed to assess the prognostic value of LAVi in HD patients, compared with previously established clinical and Doppler echocardiographic parameters.

Methods

Population - Study patients were recruited among those routinely treated in the hemodialysis unit of our university. CKD patients on maintenance hemodialysis for at least one month who agreed to undergo echocardiogram were deemed eligible. Exclusion criteria were the following: malignancies, active infection, nonsinus rhythm, pericardial effusion, and evidence of major valvular heart disease (presence of prosthetic valve, any degree of mitral or aortic stenosis, and more than mild degree of mitral, aortic, or tricuspid regurgitation). The Institutional Research Ethics Committee approved the study protocol, and all patients signed an informed consent. All subjects underwent HD in Altra Touch dialysis machines (Althin, Miami, Florida, Fl, USA) equipped with cellulose-acetate dialyzers regulated to a blood flow rate of at least 200ml/min and a dialysate flow rate between 300 and 400 ml/min containing standard preparation. "Dry weight" estimates aiming at the total volume to be removed by ultrafiltration are performed routinely in our institution by means of clinical signs of hydration, blood pressure behaviour during session, and periodic bioimpedance assessment. Parameters measured at the time of examination included systolic and diastolic blood pressure, heart rate, plus body

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height and weight. Body surface area was calculated according to DuBois & DuBois's simplified formula (0.20247 x weight$^{0.425}$ x height$^{0.725}$)[18]. Body mass index was calculated by dividing weight (kg) by squared height (m). Those with BMI > 30 were considered obese.

Doppler echocardiogram - All examinations were performed by a single examiner (S.H.B.) on interdialytic days (Tuesday or Thursday only) between noon and 6 PM, as previously recommended[19]. M-mode, two-dimensional and Doppler echocardiography (pulsed, continuous, color, and tissue imaging) were performed using an HDI 3000 system (ATL-Philips Ultrasound Systems, Bothell, Washington, USA) equipped with a 2,5-4 MHz transducer capable of operating with fundamental and second-harmonic imaging. According to the Penn convention[20], the following linear measurements were obtained: interventricular septal thickness, posterior wall thickness, LV end-diastolic and end-systolic diameters. The end-diastolic diameter upper limit was established at 55 mm. Left ventricular mass was calculated using Devereux's formula[21] and indexed to body height raised to the 2.7th power; hypertrophy was diagnosed when LV mass index (LVMi) was greater than 51 g/m^{2,7} [22]. In order to assess LV geometric pattern, relative wall thickness was calculated using the formula: (2 x mean wall thickness)/LV end-diastolic diameter, where mean thickness = (interventricular septal thickness + posterior wall thickness at diastole)/2. Reference value was 0.45, discriminating eccentric (below 0.45) from concentric (above 0.45) hypertrophy[23]. Concentric remodeling was defined by normal LVMi associated with increased relative wall thickness. Left ventricular systolic function was assessed by calculating ejection fraction using the Teichholz[24] method, the limit of which was defined as 50%. Mitral inflow velocities were measured in the apical four-chamber view with pulsed-Doppler sample placed between the leaflet tips of the mitral valve; at this time patients were instructed to breathe in a calm, controlled manner. According to American Society of Echocardiography recommendations[25], the following parameters were measured: early rapid filling (E) velocity, atrial contraction velocity (A), E/A ratio, and E-wave deceleration time (DT). DT < 140 ms, associated with a restrictive filling pattern, was considered abnormal[26]. Mitral annular velocities measured by tissue Doppler were recorded in the apical four-chamber view, with a 1- to -2 mm sample volume placed at the junction of LV septal[27] and lateral[28] walls with the mitral annulus. Both early (E') and late (A') diastolic mitral annular velocities were determined, in addition to E'/A' and E/E' ratios. Diastolic dysfunction was defined as: 1) E/A < 1 (abnormal relaxation); 2) E/A > 2 (restrictive flow); 3) E/A between 1 and 2 in association with E/E' > 10 (pseudonormalization)[29]. Left atrial size was assessed by M-mode measurement of anteroposterior dimension (abnormal when > 40 mm), as previously recommended[30], and also by LA volume calculated by using two-dimensional planimetry with the biplanar Simpson's rule[9] on the frame just before mitral valve opening. Left atrial volume index (LAVi) was calculated by the LA volume to body surface area ratio[12]; values greater than 32 ml/m^2 are suggestive of increased cardiovascular risk[11,13,14]. All Doppler echocardiographic measurements presented in the study correspond to the average of three cardiac cycles.

Survival analysis - Demographics, the presence of comorbidities (history of diabetes mellitus, hypertension, acute myocardial infarction, angina documented by coronary angiography, stroke, and clinical diagnosis of heart failure), drugs in use, and routine laboratory tests (hemoglobin, albumin, calcium, and phosphorus) were determined based on a thorough analysis of the medical chart combined with an interview with the patient or the attending physician. Study patients were prospectively followed from June 2003 to July 2006 or until an endpoint occurred. The study's composite endpoint was all-cause mortality and nonfatal cardiovascular events. The following events were observed: 1) death; 2) new coronary event, defined as nonfatal acute myocardial infarction or angina pectoris with coronary stenosis > 50% on coronary angiography; 3) ischemic or hemorrhagic stroke, transient ischemic attack excluded; 4) clinical diagnosis of congestive heart failure requiring hospitalization. These events were researched through periodic review of medical documentation, including clinical records and death certificates, in addition to contact with the doctor and patient's relatives. In case of multiple events in a single patient, only the first event was considered. Patients who underwent renal transplantation were censored in the analysis.

Statistical analysis - Data are presented as mean and standard deviation (parametrically distributed continuous variables), median (nonparametrically distributed continuous variables), and percentage (categorical variables). Differences between groups were determined using the unpaired Student's t test, the Mann-Whitney test, or the chi-square test. LAVi independent prognostic value was tested using Cox multivariate survival analysis. Initially, groups with and without the study endpoint were compared, in order to select variables for proportional risk analysis. The E/E' ratio was specifically included in the model because an earlier study showed that this index was associated with increased mortality in candidates for renal transplantation[31]. Significant univariate predictors were added to the Cox model (entry and retention set at a significance level of 0.1 and 0.05, respectively) in several steps, simulating the clinical reasoning). Thus, the first step used clinical and biochemical variables as baseline risk factors. Subsequently, traditional Doppler echocardiographic variables were added. The last steps consisted of the sequential introduction of E/E' ratio and LAVi as continuous variables. The increase in predictive power at each step was assessed by change in chi-square value. Kaplan-Meier curves were plotted for LAVi as categorical variable, using a partition value of 32 ml/m^2. The statistical significance level was set at 5% ($p < 0.05$).

Data were processed by using "SPSS 13.0 for Windows" (SPSS INC, Chicago, Illinois).

Results

One hundred and thirty-one subjects were assessed. Nine patients were excluded (three because of arrhythmias, three because of major mitral regurgitation, one because of aortic stenosis, and one because of pericardial effusion), and four patients moved to another city and were lost to follow-up. Table 1 summarizes demographic, clinical, biochemical,

and Doppler echocardiographic characteristics of the 118 remaining patients that made up this study population. Our group comprised 64 men and 54 women, mean age 48 ± 15, and median HD duration of 22 months (1 to 120). CKD etiology was attributed to hypertensive nephrosclerosis (40%), chronic glomerulonephritis (24%), diabetic nephropathy (19%), policistic kidney (8%), chronic pyelonephritis (6%), and other conditions (3%). Most patients (76%) were on antihypertensive drugs, particularly angiotensin-converting enzyme inhibitors (45%), beta-blockers (19%), alpha-blockers (13%), calcium channel antagonists (13%), and angiotensin receptor blockers (10%), alone or combined.

Previous cardiovascular disease - Forty-eight patients (41%) had history of heart disease (heart failure in 32%, myocardial infarction in 5%, and angina in 3%). Moreover, 2% had history of stroke, and none had peripheral arterial disease. Eleven percent of the patients were obese.

Table 1 - Primary baseline characteristics of the study population

	Total (n = 118)
Age (years)	48 ± 15
Male gender (%)	54
Body mass index	24 ± 5
Time on dialysis (months)	22*
Systolic BP (mmHg)	140 ± 25
Diastolic BP (mmHg)	86 ± 12
Previous heart disease (%)	41
Diabetes Mellitus (%)	19
Hemoglobin (g/dl)	10.8 ± 3.1
Albumin (mg/l)	3.8 ± 0.6
Ca x P product	51 ± 22
LVEDD (mm)	50 ± 7
LVMi (g/m$^{2.7}$)	93 ± 39
Ejection fraction (%)	61 ± 8
Left atrium	
Anteroposterior dimension (mm)	36 ± 6
LAVi (ml/m^2)	34 ± 15
Doppler	
E/A	1.0 ± 0.7
DT (ms)	190 ± 9
E/E'	10 ± 4

Dada presented in mean ± SD, percentages, or median with variation. BP - blood pressure; Ca x P - calcium x phosphorus; LVEDD - left ventricular end-diastolic diameter; LVMi - left ventricular mass index; LAVi - left atrial volume index; E - early transmitral filling velocity; A - transmitral flow velocity during atrial contraction; DT - deceleration time; E/E' - ratio between E and early diastolic mitral annular velocity. *median.

Doppler echocardiographic changes - Figure 1 shows the most prevalent echocardiographic changes found in our group. Twenty-six patients had LV dilation (22%), 106 had hypertrophy (90%), 21 had systolic dysfunction (18%), and 96 had diastolic dysfunction (81%). According to the relative wall thickness, the most frequent geometric change was concentric hypertrophy (77%), followed by eccentric hypertrophy (13%), and concentric remodeling (4%). Only 6% of the patients showed normal LV geometry. Thirty-two patients (27%) were diagnosed with LA dilation by M-mode echocardiography. Mean LAVi was 34 ± 15 ml/m^2 (median 31, ranging from 17 to 89), and this index was greater than 32 ml/ m^2 in 50% of the study sample.

Prognosis - During the mean follow-up of 19 ± 8 months, there were 20 deaths and 17 nonfatal cardiovascular events, resulting in an overall endpoint rate of 31%. Deaths were categorized as cardiovascular (12 deaths, seven from acute myocardial infarction, four from sudden death, and one from hemorrhagic stroke) or infectious (eight). Nonfatal cardiovascular events included three cases of angina, one case of acute myocardial infarction, ten hospital admissions for decompensated heart failure, and three cases of ischemic stroke. Table 2 shows differences among subgroups with and without endpoint. Patients who experienced study events were older (56 ± 15 vs 45 ± 13, p < 0,001); were on HD for a longer period (median 24 months vs 11 months, p = 0.014); had history of heart disease (heart failure plus infarction and angina: 73% vs 12%, p < 0.001) and diabetes (35% vs 12%, p = 0.004); showed lower serum albumin concentration (3.4 ± 0.4 vs 3.9 ± 0.6, p = 0,02), and were mostly male (67% vs 48%, p = 0.047). No differences were found in body mass index (p = 0.5), systolic blood pressure (p = 0.7), diastolic blood pressure (p = 0.8), hemoglobin (p = 0.2), and calcium-phosphorus product (p = 0.4). On echocardiogram, the subgroup of patients that reached the study endpoint had greater LV diastolic diameter (53 ± 6 vs 48 ± 7 mm, p < 0.001), LVMi (109 ± 44 vs 85 ± 34 g/m$^{2.7}$, p = 0.002), LA anteroposterior dimension (39 ± 6 vs 34 ± 6 mm, p = 0.001), and LAVi (42 ± 15 vs 30 ± 14 mL/m^2, p < 0.001). Consistent with these findings, this subgroup showed lower ejection fraction and higher E/E' (57 ± 9 vs 64 ± 6%, p <

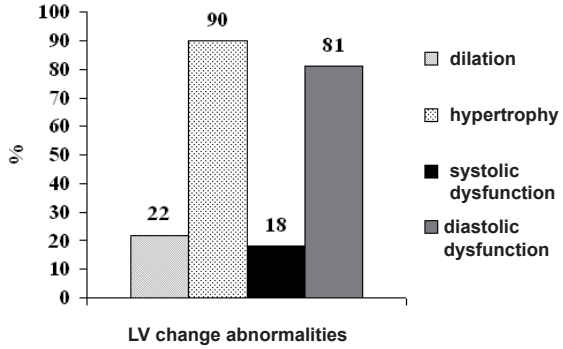

Fig. 1 - Percentage of main abnormalities in LV anatomy and function on echocardiogram. LV - left ventricle.

Table 2 - Comparison of demographic, clinical, biochemical and Doppler echocardiographic data of patients with and without the study endpoint

	Without endpoint (n=81)	With endpoint (n=37)	p value
Age (years)	45 ± 13	56 ± 15	<0.001
Male gender (%)	48	67	0.047*
BMI	24 ± 6	25 ± 5	0.5
Time of dialysis (months)	11	24	0.014†
Diabetes mellitus (%)	12	35	0.004*
Previous heart disease (%)	12	73	<0.001*
Systolic BP (mmHg)	152 ± 26	148 ± 24	0.7
Diastolic BP (mmHg)	88 ± 12	86 ± 11	0.8
Hemoglobin (g/dl)	11 ± 3.3	10±2.1	0.2
Ca x P product	49 ± 22	56 ± 18	0.2
Albumin (mg/l)	3.9 ± 0.6	3.4 ± 0.4	0.02
LVEDD (mm)	48 ± 7	53 ± 6	<0.001
LV dilation (%)	15	30	0.05*
Ejection fraction (%)	64 ± 6	57 ± 9	<0.001
LVMi ($g/m^{2.7}$)	85 ± 34	109 ± 44	0.002
E/A	0.98 ± 0.4	1.5 ± 1	0.01
DT (ms)	197 ± 62	175 ± 51	0.05
DT < 140 ms (%)	14	24	0.18*
E'/A'	0.79 ± 0.3	0.80 ± 0.3	0.7
E/E'	8 ± 4	13 ± 4	<0.001
E/E' > 15 (%)	17	33	0.02*
LAD (mm)	34 ± 6	39 ± 6	0.001
LA dilation on M-mode (%)	22	38	0.08*
LAVi (ml/m^2)	30 ± 14	42 ± 15	<0.001
LAVi > 32 ml/m^2 (%)	36	81	<0.001*

Data presented in mean ± SD, percentages or median. BMI - body mass index; BP - blood pressure; Ca x P - calcium x phosphorus; LVEDD - left ventricle end-diastolic diameter; LVMi - left ventricle mass index; E/A - ratio between early transmitral filling and atrial contraction velocities; DT - deceleration time; E'/A' - ratio between early and late diastolic mitral annulus velocity; LAD - left atrial anteroposterior dimension; LAVi - left atrial volume index. *percentages compared using chi-square test; † medians compared using Mann-Whitney test; means compared using Student's t test for the others.

0.001; and 13 ± 4 vs 8 ± 4, p < 0.001, respectively). The frequency of deceleration time (DT) < 140 ms was similar in the subgroups (p = 0.2), but there was a statistical trend toward shorter DT in patients who reached endpoints (175 ± 51 vs 197 ± 62 ms, p = 0.05). After the most significant predictors were selected (Table 2), the basic clinical model included age, male gender, previous heart disease, diabetes, time on HD, and albumin. Subsequently, we added ejection fraction, LVMi, and DT to the model, followed by E/E' and, finally, LAVi. In order to prevent statistical colinearity, we sought not to use correlated variables, such as LV end-diastolic diameter, which plays a role in ejection fraction and LVMi calculation.

Independent prognostic predictors, with respective hazard ratios and 95% confidence interval (CI), are listed in Table 3. After LAVi was added to the analysis, ejection fraction, LVMI and E/E' were no longer independent predictors of the study endpoint. In the final model, LAVi was the sole independent echocardiographic predictor of combined death and nonfatal cardiovascular events, with hazard ratio of 1.03 per ml/m^2 (95% CI, 1.01-1.05, p = 0.014) from 32 ml/m^2. Among the

other variables, the presence of previous heart disease and time on HD (over 21 months) reached statistical significance. The incremental value provided to the model by LAVi is shown in Figure 2 (chi-square increased from 69 to 77 in the final step, p = 0.02).The endpoint rate was significantly higher in patients with LAVi > 32 ml/m², compared with the subgroup with LAVi ≤ 32 ml/m² (51% vs 12%, respectively, p < 0.001). Figure 3 shows adjusted Kaplan-Meier curves for endpoint-free survival using the partition value of 32 ml/m². Annual event-free survival was estimated at 98% for patients with LAVi ≤ 32 ml/m² vs 79% for those with LAVi > 32 ml/m² (p < 0.001). Table 4 depicts the main clinical, Doppler echocardiographic, and biochemical features according to this partition value. Patients with LAVi > 32 ml/m² had more heart failure and LV echocardiographic abnormalities, such as hypertrophy, systolic dysfunction, and increased E/E'; in addition they were predominantly male. Conversely, no differences were found in age, biochemical parameters and prevalence of hypertension, diabetes, and obesity. Death rate was higher among those with LAVi > 32 ml/m² (30.5% vs 3.4%, p < 0.001). Among 59 patients with LAVi ≤ 32 ml/m², only two died, in contrast with 18 death among 59 patients with LAVi > 32 ml/m².

Discussion

In recent years, echocardiographic assessment of the left atrium and its relationship with cardiovascular risk have been revalued. The most relevant finding of the present study is that LAVi may provide independent prognostic and incremental information to traditional clinical and echocardiographic data of HD patient population. Of all echocardiographic parameters assessed, LAVi was shown to be the best predictor of clinical course in this group, known to be at high cardiovascular risk. Left ventricular mass, recognized as a strong marker of mortality in this population[4,6], was no longer an independent predictor of endpoint after LAVi was included in the analysis. The presence of increased LAVi was also more prevalent than other risk indicators, such as ejection fraction and DT. This finding corroborates previous reports indicating that LAVi is a more effective parameter than systolic function analysis in predicting death in patients with acute myocardial infarction[13,14] and dilated cardiomyopathy[15,16].

Some speculations may be made to understand LAVi's remarkable prognostic power. It is known that a positive correlation exists between LA size and LV passive properties[32]. The progressive deterioration of myocardial diastolic properties causes an elevation in LV filling pressures. Consequently, LA pressure rises, causing it to dilate[32]. Therefore, it is highly likely that LA size acts as a marker of diastolic dysfunction chronicity. In keeping with this proposition, we recently reported that LAVi is less sensitive to acute changes in preload than pulsed Doppler-derived parameters of mitral flow[33]. Additionally, we have demonstrated that LAVi is correlated with diastolic dysfunction severity in HD patients[34] and is more effective in detecting pseudonormalization of mitral flow, compared with various previously tested indices[35]. Thus, unlike mitral flow-derived Doppler indices, known to be volume-dependent and provide transient information about ventricular filling[33,36], LAVi appears to be a more stable marker of diastolic function, related to the cardiovascular "track record" built over time. This may also explain its predictive superiority over the E/E' ratio, the most reliable non-invasive index for estimating LV end-diastolic pressure[31,37], but responsive to acute preload changes.

It is likely that in clinical cohorts, with significant prevalence of cardiovascular disease, left atrial dilation may result not only from the long-standing diastolic dysfunction but also

Table 3 - Independent predictors of prognosis according to Cox multivariate analysis

	HR	95% CI	p value
Previous heart disease	1.91	1.79-1.96	< 0.001
Time on dialysis (months)	1.03	1.01-1.04	0.006
LAVI (per ml/m²)	1.03	1.01-1.05	0.014

HR - hazard ratio; CI - confidence interval; LAVI - left atrial volume index.

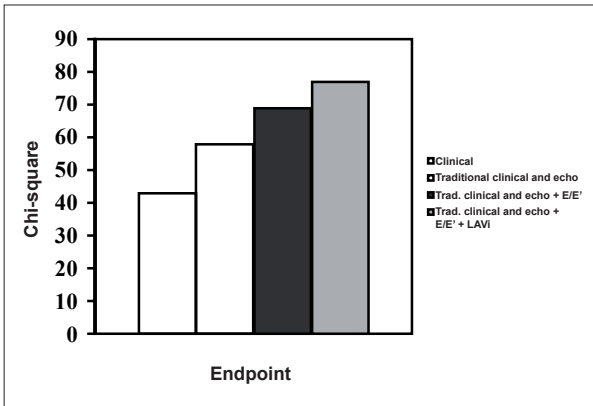

Fig. 2 - Increase in the prognostic predictive power of left atrial volume index (LAVi). Traditional echocardiographic data include left ventricular mass index, ejection fraction, and deceleration time. E/E', ratio between early transmitral filling velocity and early diastolic mitral annulus velocity.

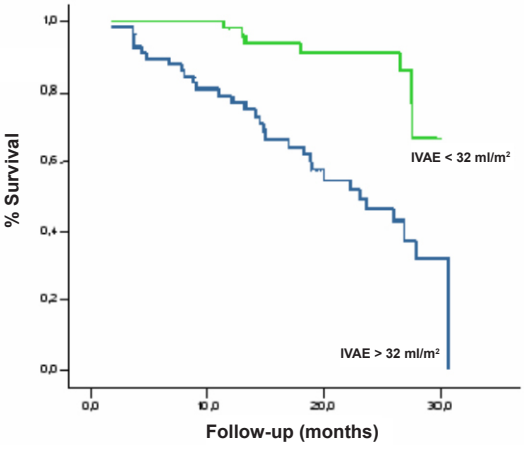

Fig. 3 - Kaplan-Meier curves for endpoint-free survival based on a partition value of left atrial volume index (LAVi) of de 32 mL/m² (p < 0.001).

Table 4 - Primary clinical, biochemical, and Doppler echocardiographic characteristics according to LAVi

Variable	LAVi ≤ 32 ml/m²	LAVi > 32 ml/m²	p value
Number of patients	59	59	
Age (years)	46 ± 14	51 ± 15	0.09
Male gender (%)	44	67	< 0.05
Diabetes Mellitus (%)	14	27	0.1
Hypertension (%)	50	50	0.7
Previous heart disease (%)	12	56	< 0.001
Myocardial infarction (%)	1.5	9.6	0.09
Angina (%)	1.5	5.8	0.4
Heart failure (%)	14	56	< 0.001
Obesity (%)	12	11	0.9
LV hypertrophy (%)	82	100	< 0.001
Systolic dysfunction (%)	6	30	< 0.01
Diastolic dysfunction (%)	68	73	0.68
Hemoglobin (g/dl)	11.3 ± 3.6	10.2 ± 2	0.1
Albumin (mg/l)	3.9 ± 0.6	3.7 ± 0.6	0.9
Ca x P product	48 ± 14	55 ± 29	0.2
E/A	0.93 ± 0.4	1.44 ± 0.9	< 0.01
DT (ms)	192 ± 53	188 ± 67	0.7
E' (cm/s)	9.3 ± 2.8	7.6 ± 1.6	< 0.01
E/E'	7.9 ± 2.6	12 ± 4	< 0.01

Dada presented in mean ± SD, percentages or median with variation. LV - left ventricle; Ca x P - calcium x phosphorus; E - early transmitral filling velocity; A - transmitral flow velocity during atrial contraction; DT - deceleration time; E - ealy diastolic mitral annular velocity.

from other factors associated with worse prognosis. In a study using an animal model of heart failure, Khan and colleagues[38] have demonstrated LA remodeling similar to that found in the ventricular chamber. The neurohumoral activation causes changes in the extracellular matrix, atrial myocites hypertrophy, fibrosis, and progressive dilation of the left atrium[38]. Based on accumulated evidence, it is reasonable to interpret a chronically dilated LA as a result from both diastolic dysfunction burden and remodeling in patients with sinus rhythm but without significant mitral valve disease.

Recently, Tripepi and colleagues[39] published a similar prospective study evaluating 199 patients on HD and 50 on peritoneal dialysis, and found an independent association of LAVi with diastolic function, as well as higher all-cause mortality. This study suggested that the indexation of LA volume by height raised to the 2.7th power added prognostic information and was superior to the indexation by body surface area. We used the body surface approach because a previous population-based study (over 2000 patients) showed that the adjustment to body size using height failed to nullify the gender influence on atrial size, unlike the adjustment to body surface area. Furthermore, the indexation by height to the 2.7th power was described by Simone and colleagues[22] taking LV geometry into account, and was not validated to the LA. Finally, it must be remembered that the indexation method based on body height was superior to that based on body surface area to prevent all-cause mortality, but not cardiovascular event incidence[39].

Limitations - No patient included in this study had changes in segmental contractility in LV basal regions, but 8% had history of coronary artery disease. To optimize accuracy in assessing global diastolic function using tissue Doppler, we used a mean E' value measured at both sides of the annulus[29]. Ejection fraction was calculated based on linear measurements, rather than the planimetric two-dimensional technique. However, as other studies failed to demonstrate the superiority of ejection fraction by planimetry over LAVi in the prognostic evaluation[14,16], and given the relatively small number of patients with history of coronary failure in our sample, it is unlikely that our final result has been

compromised. Patients with arrhythmias and significant valvular heart disease were excluded from our study, thereby eliminating confounding factors; yet, this may have rendered our sample less representative of the HD population.

Conclusion

Concluding, LAVi was an independent predictor of prognosis in patients on renal replacement therapy with hemodialysis, providing additional information to traditional clinical and echocardiographic data. We propose that LAVi be incorporated in the routine assessment of this group of patients to improve the stratification of cardiovascular risk and, thereby, reduce morbidity and mortality rates.

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Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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