Determinants of Left Ventricular Mass and Hypertrophy in Hemodialysis Patients Assessed by Cardiac Magnetic Resonance Imaging

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Background and objectives: Left ventricular hypertrophy (LVH) is an independent risk factor for premature cardiovascular death in hemodialysis (HD) patients and one of the three forms of uremic cardiomyopathy. Cardiovascular magnetic resonance (CMR) is a volume-independent technique to assess cardiac structure. We used CMR to assess the determinants of left ventricular mass (LVM) and LVH in HD patients.

Design, setting, participants, & measurements: A total of 246 HD patients (63.8% male; mean age 51.5 ± 12.1 yr) underwent CMR on a postdialysis day. LVM was measured from a stack of cine loops and indexed for body surface area (LVM index [LVMI]). Demographic, past biochemical, hematologic, and dialysis data were collected by patient record review. Results up to 180 d before CMR were collected. LVH was defined as LVMI >84.1 g/m^2 (male) or >76.4 g/m^2 (female).

Results: A total of 157 (63.8%) patients had LVH. LVH was more common in patients with higher predialysis systolic BP, predialysis pulse pressure, and calcium-phosphate product (Ca × PO$_4$). Furthermore, LVH was significantly associated with higher end-diastolic and systolic volumes and lower ejection fraction. There were positive correlations with LVMI and end-diastolic and systolic volumes. There were weak positive correlations among LVMI, mean volume of ultrafiltration, and Ca × PO$_4$. Using multivariate linear and logistic regression (entering one BP and cardiac variable), the independent predictors of LVMI and LVH were end-diastolic volume, predialysis systolic BP, and Ca × PO$_4$.

Conclusions: The principal determinants of LVM and LVH in HD patients are end-diastolic LV volume, predialysis BP, and Ca × PO$_4$.

Patients with ESRD, particularly those who require hemodialysis (HD), have an increased risk for premature cardiovascular disease (CVD) (1). Left ventricular hypertrophy (LVH) is a common feature of patients with ESRD, a component of uremic cardiomyopathy and an independent risk factor for sudden cardiac death, heart failure, and cardiac arrhythmias in the general population and HD patients (2–4).

Studies that have assessed independent predictors of left ventricular mass index (LVMI; corrected for body surface area [BSA]) and LVH in patients with ESRD have used echocardiography and implicated factors such as hypertension, reduced blood vessel compliance, anemia, phosphate control, and dosage of dialysis (5–7); however, accurate echocardiographic estimation of LVMI in patients with ESRD is difficult because of large variation in intravascular (and hence intraventricular) volume during the interdialytic period and during dialysis. Geometric assumptions made during calculation of LVMI from conventional M-mode echocardiography dimensions result in greater inaccuracies as a result of geometric LV distortion in patients with LVH and ESRD.

Cardiac magnetic resonance (CMR) imaging provides more detailed, volume-independent measurement of cardiac structure and has been thoroughly validated using human autopsy and animal specimens (8,9). This technique has been established as the most accurate noninvasive method of assessing ventricular dimensions in patients, including those with stage 5 chronic kidney disease (10,11). In particular, measurements obtained by echocardiography tend to overestimate LVMI, particularly at higher values, when compared with CMR (12). Furthermore, pilot studies in patients with ESRD, using CMR to identify myocardial changes of uremic cardiomyopathy, have so far shown a significant reduction in long-term survival similar to previous echocardiography findings (13 and unpublished data). Thus, the aim of this study was to assess by CMR the determinants of LVMI and LVH in a cohort of HD patients.

Materials and Methods

Patients

Since 2002 (10,11,13), we have used CMR as part of the standard assessment of patients for renal transplantation. The renal transplant unit at the Western Infirmary (Glasgow, Scotland, UK) provides transplant services to a population of 2.8 million people in the west of Scotland. The transplant waiting list has 300 to 400 patients at any given time; approximately 100 to 120 new patients are placed on the waiting list, and approximately 70 adult transplants are performed annually. We assessed 246 patients who had been receiving thrice-weekly maintenance HD for at least 3 mo.

CMR Technique and Analysis

Non–gadolinium-based contrast CMR was performed using a 1.5 Tesla magnetic resonance imaging scanner (Sonata; Siemens, Erlangen, Germany). Scans were consistently performed 24 h after the end of the last dialysis session. Long-axis pilot scans were obtained through the apex of the left ventricle, aligning it with the center of the mitral valve, and then 8-mm-thick short-axis images were obtained. A fast imaging with steady-state precession sequence was then used to acquire cine images in long-axis planes (vertical long axis, horizontal long axis, LV outflow tract) followed by sequential short-axis LV cine loops (8-mm-slice thickness, 2-mm gap between slices) from the atrioventicular ring to the apex. Slices were contiguous (*i.e.* with no gap). Imaging parameters, which were standardized for all patients, included repetition time/echo time/flip angle/voxel size/field of view of 3.14 ms/1.6 ms/60°/2.2 × 1.3 × 8.0 mm/340 mm. LVM was analyzed by two observers, who were blinded to patient clinical characteristics, from short-axis cine loops using manual tracing of epicardial and endocardial end-systolic and end-diastolic contours. End-systolic volume (ESV) and end-diastolic volume (EDV) and LVM were calculated using analysis software (Argus; Siemens). Values were compared with published normal values: LVH was defined as LVMI (LVM/BSA) >84.1 g/m^2 (male) or >76.4 g/m^2 (female), and LV systolic dysfunction was defined as LV ejection fraction (LVEF) <55%. LV dilation was defined as EDV/BSA >111.7 ml/m^2 (male) or 99.3 ml/m^2 (female) or ESV/BSA >92.8 ml/m^2 (male) or 70.3 ml/m^2 (female) (14).

Clinical and Blood Result Data Collection

Demographic information and clinical (including drug) history were recorded at the time of CMR. Retrospective review of the electronic patient record was performed to retrieve HD information, including immediate pre-HD and post-HD systolic and diastolic BP (SBP and DBP, respectively), dosage of ultrafiltration (UF), dialysis adequacy using urea reduction ratio (URR), and biochemical and hematologic blood results. Information was collected at 30 d intervals up to 180 d before CMR and mean results were calculated as described previously (15). Calcium-phosphate product (Ca × PO$_4$) was also calculated from blood results collected. URR was calculated using an established protocol (16).

Statistical Analysis

Statistical analyses were performed using SPSS 15.0 (SPSS, Chicago, IL). Graphs were drawn using GraphPad Prism 4.00 (GraphPad Software, San Diego, CA). Data are expressed as mean ± SD. Correlations between LVMI and other factors were evaluated by Pearson correlation coefficient. Variables that were identified by univariate analysis as significant predictors were entered into a backward stepwise linear and logistic regression model to identify independent predictors of LVMI and LVH, respectively. Because of significant interdependence, SBP and DBP were entered separately into the model. Only one cardiac parameter (EDV/BSA, ESV/BSA, or LVEF) was entered for the same reason.

Results

Clinical and Cardiac Parameters

A total of 246 HD patients were assessed by CMR between 2002 and 2008. LVH was present in 157 (63.8%) of patients. The demographic, HD, blood, drug, and cardiac data are presented in Table 1.

Factors Associated With LVH

Higher pre-HD SBP, pre-HD pulse pressure (PP), and Ca × PO$_4$ were significantly associated with LVH (pre-HD SBP 144.6 ± 20.0 [LVH] *versus* 138.1 ± 24.3 mmHg [no LVH; *P* = 0.02]; pre-HD pulse pressure 64.4 ± 16.5 mmHg [LVH] *versus* 59.1 ± 17.6 mmHg [no LVH; *P* = 0.02]; and Ca × PO$_4$ 3.05 ± 1.00 [LVH] *versus* 2.72 ± 1.00 mmol^2/L^2 [no LVH; *P* = 0.03], respectively; Table 1). Serum phosphate and mean UF dosage were higher in the LVH group of patients, but these were not statistically significant. When comparing both groups, there were no significant differences in age, gender, body mass index, cardiovascular history, duration on HD, mean post-HD SBP and DBP, and mean pre-HD DBP and URR. Furthermore, LVH was not significantly associated with a difference in mean hemoglobin, albumin, adjusted serum calcium, or parathyroid hormone (PTH) measured during the 180 d before CMR. There was no significant difference in drug use between both groups.

Table 1 also shows LV cardiac parameters measured: Ejection fraction, EDV/BSA, and ESV/BSA. LVH was significantly associated with lower LVEF, higher end-diastolic and systolic LV volumes, LV systolic dysfunction, and LV dilation.

Correlations with LVMI

There were strong positive correlations between LVMI and EDV/BSA (*r* = 0.60, *P* < 0.001; Figure 1a) and ESV/BSA (*r* = 0.54, *P* < 0.001) and negative correlation with LVEF (*r* = −0.37, *P* < 0.001). In addition, there were weaker but significant correlations with mean dosage of UF (*r* = 0.18; *P* = 0.005; Figure 1b) and Ca × PO$_4$ (*r* = 0.13, *P* = 0.05; Figure 1c). Factors that were close to statistical significance were post-HD DBP (*r* = 0.13, *P* = 0.06) and mean pre-HD SBP (*r* = 0.11, *P* = 0.08; Figure 1d). Mean UF correlated significantly with EDV/BSA (*r* = 0.22, *P* = 0.001; Table 2).

Determinants of LVH and LVMI

Univariate followed by multivariate logistic and linear regression analyses were performed to determine predictors of LVH and LVMI, respectively (Tables 3 and 4). Univariate analyses identified EDV/BSA, mean Ca × PO$_4$ product, and mean pre-HD SBP as factors that were significantly associated with presence of LVH. Multivariate logistic regression modeling (only entering one BP variable and cardiac parameter because of their significant interdependence) similarly identified EDV/BSA, pre-HD SBP, and Ca × PO$_4$ as the most robust independent predictors of LVH.

Linear regression analysis was performed to create the most robust predictive model for LVMI (Table 3). Factors considered in the model were those that were identified as significant (or close) correlates with LVMI. As before, because of their close interdependence, BP variables and cardiac parameters were

Table 1. Clinical, drug, and cardiac data for patients

Parameter	All Patients ($n = 246$)	No LVH ($n = 89$)	LVH ($n = 157$)	P
Age (yr; mean $\pm$ SD)	51.4 $\pm$ 12.1	51.8 $\pm$ 12.2	51.2 $\pm$ 12.1	0.20
Male gender (n [%])	157 (63.8)	55 (61.8)	102 (65.0)	0.62
BMI (kg/m^2; mean $\pm$ SD)	25.6 $\pm$ 4.6	25.2 $\pm$ 4.2	25.8 $\pm$ 4.8	0.45
Ischemic heart disease (n [%])	51 (20.7)	21 (23.6)	30 (19.1)	0.40
Diabetes (n [%])	156 (63.4)	58 (65.2)	98 (62.4)	0.67
Chronic heart failure (n [%])	15 (6.1)	5 (5.6)	10 (6.4)	0.81
Cerebrovascular disease (n [%])	20 (8.1)	10 (11.2)	10 (6.4)	0.18
Peripheral vascular disease (n [%])	18 (7.3)	7 (7.9)	11 (7.0)	0.80
Hypertension (n [%])	223 (90.7)	79 (88.8)	144 (91.4)	0.44
Duration on HD (yr; mean $\pm$ SD)	3.0 $\pm$ 3.0	2.9 $\pm$ 3.1	3.0 $\pm$ 2.9	0.89
UF volume (L; mean $\pm$ SD)	2.2 $\pm$ 1.1	2.1 $\pm$ 0.9	2.3 $\pm$ 1.2	0.08
Pre-HD SBP (mmHg; mean $\pm$ SD)	142.3 $\pm$ 21.8	138.1 $\pm$ 24.3	144.6 $\pm$ 20.0	0.02
Post-HD SBP (mmHg; mean $\pm$ SD)	132.3 $\pm$ 23.1	129.2 $\pm$ 24.8	133.8 $\pm$ 21.9	0.14
Pre-HD DBP(mmHg; mean $\pm$ SD)	79.8 $\pm$ 14.0	79.1 $\pm$ 16.4	80.3 $\pm$ 12.4	0.52
Post-HD DBP(mmHg; mean $\pm$ SD)	73.3 $\pm$ 13.8	72.3 $\pm$ 15.9	73.9 $\pm$ 12.5	0.36
Pre-HD PP (mmHg; mean $\pm$ SD)	62.5 $\pm$ 17.1	59.1 $\pm$ 17.6	64.4 $\pm$ 16.5	0.02
Post-HD PP (mmHg; mean $\pm$ SD)	58.9 $\pm$ 17.6	57.1 $\pm$ 18.9	60.0 $\pm$ 16.8	0.22
URR (%; mean $\pm$ SD)	70.8 $\pm$ 7.5	67.6 $\pm$ 11.9	70.7 $\pm$ 6.4	0.64
Hemoglobin (g/dl; mean $\pm$ SD)	11.3 $\pm$ 1.3	11.3 $\pm$ 1.5	11.3 $\pm$ 1.3	0.95
Adjusted Ca (mmol/L; mean $\pm$ SD)	2.31 $\pm$ 0.20	2.29 $\pm$ 0.20	2.31 $\pm$ 0.20	0.61
Phosphate (mmol/L; mean $\pm$ SD)	1.27 $\pm$ 0.40	1.20 $\pm$ 0.40	1.32 $\pm$ 0.50	0.06
Ca $\times$ PO$_4$ (mean $\pm$ SD)	2.93 $\pm$ 1.00	2.72 $\pm$ 0.90	3.05 $\pm$ 1.00	0.03
Albumin (mean $\pm$ SD)	37.3 $\pm$ 5.9	37.9 $\pm$ 6.1	36.9 $\pm$ 5.8	0.24
PTH (mean $\pm$ SD)	32.5 $\pm$ 28.7	34.3 $\pm$ 28.3	31.4 $\pm$ 29.2	0.54
Aspirin (n [%])	97 (39.4)	38 (42.7)	59 (37.6)	0.43
Warfarin (n [%])	11 (4.5)	3 (3.8)	8 (5.1)	0.53
ACEI/ARB (n [%])	61 (24.8)	23 (25.8)	38 (24.2)	0.78
Diuretic (n [%])	50 (20.3)	23 (25.8)	27 (17.2)	0.11
CCA (n [%])	73 (29.7)	25 (28.1)	48 (30.6)	0.68
α Adrenoceptor blocker (n [%])	17 (6.9)	9 (10.1)	8 (5.1)	0.14
β Adrenoceptor blocker (n [%])	106 (43.1)	36 (40.4)	70 (44.6)	0.53
ESA (n [%])	196 (79.7)	74 (83.1)	122 (77.7)	0.46
Vitamin D analogue (n [%])	99 (40.2)	35 (39.3)	64 (40.8)	0.83
Ejection fraction (%; mean $\pm$ SD)	64.7 $\pm$ 13.3	68.5 $\pm$ 7.9	62.6 $\pm$ 15.1	0.001
LVMI (g/m^2; mean $\pm$ SD)	99.4 $\pm$ 30.0	67.2 $\pm$ 10.8	116.9 $\pm$ 31.3	0.001
EDV/BSA (ml/m^2; mean $\pm$ SD)	77.8 $\pm$ 31.1	58.2 $\pm$ 18.3	88.7 $\pm$ 34.7	0.001
ESV/BSA (ml/m^2; mean $\pm$ SD)	29.6 $\pm$ 21.9	19.7 $\pm$ 10.8	36.5 $\pm$ 27.5	0.001
LVSD (n [%])	45 (18.3)	6 (6.7)	39 (24.8)	0.001
LV dilation (n [%])	39 (15.8)	2 (2.2)	37 (23.6)	0.001

Comparisons between patients with and without LVH are shown. Tests of significance are t test and χ^2 test. ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor II blocker; BMI, body mass index; CCA, calcium channel antagonist; ESA, erythropoiesis-stimulating agent; LVSD, left ventricular systolic dysfunction; MAP, mean arterial pressure; PP, pulse pressure.

entered separately into the multivariate model. Using a backward stepwise model, the most significant model ($R^2 = 0.49$) generated included EDV/BSA, mean pre-HD SBP, and Ca $\times$ PO$_4$ as significant predictors of LVMI.

Discussion

Premature CVD is the most common cause of mortality in patients with ESRD. Although the risk of accelerated coronary artery disease and myocardial infarction is increased in this population, the major increased cardiovascular risk is attributable to heart failure and to sudden, presumed arrhythmic cardiac death (17). The atypical nature of CVD in this population is underscored by the futility of interventions, such as statin therapy, that are effective in other high-risk populations (18). This has prompted the identification of alternative risk factors and therapeutic targets. Of these,
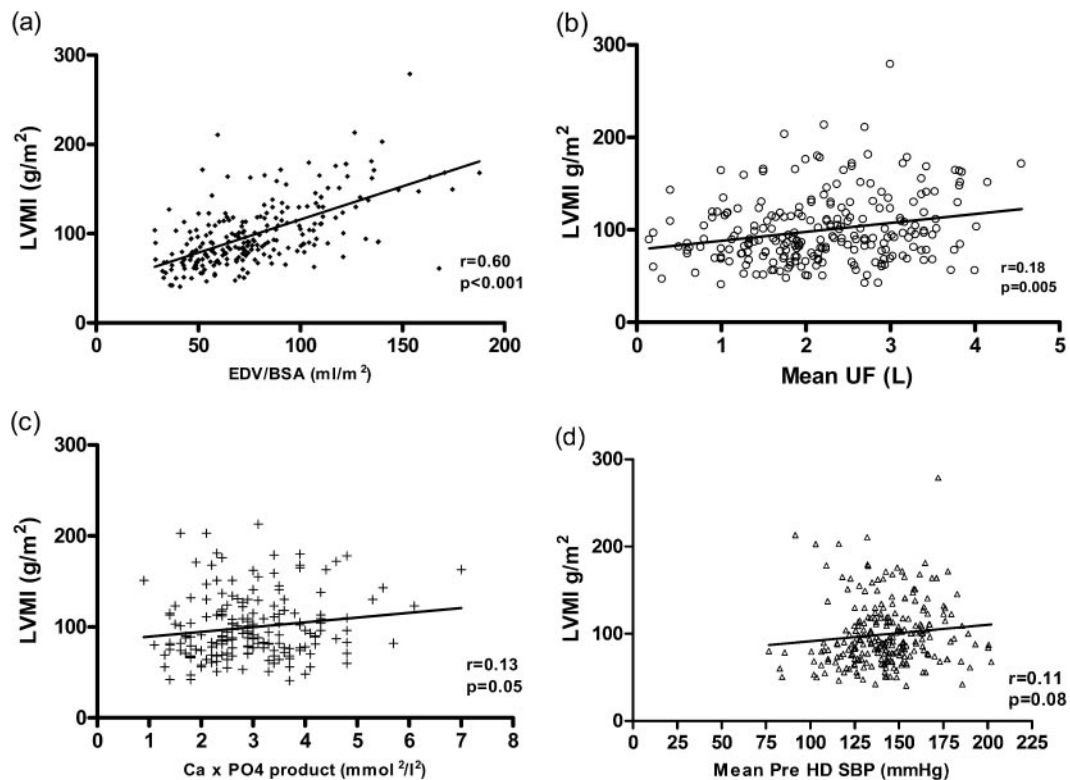
Figure 1. Scatterplot demonstrating correlation between LVMI and (a) EDV/BSA, (b) Mean UF, (c) Ca × PO$_4$ product, and (d) mean pre-HD SBP (Pearson's correlation coefficient).

Table 2. Correlation between LVMI and clinical, blood, and cardiac parameters

Parameter	r	P
EDV/BSA (ml/m^2)	0.60	<0.001
Mean pre-HD SBP (per mmHg)	0.12	0.08
Ca × PO$_4$ (mmol^2/L^2)	0.13	0.05
Mean UF volume (L/dose)	0.18	0.005
ESV/BSA (ml/m^2)	0.54	<0.001
Stroke volume/BSA	0.46	<0.001
Ejection fraction (%)	−0.37	<0.001
Age (yr)	−0.15	0.10
BMI (kg/m^2)	−0.07	0.23
Duration on HD (yr)	0.06	0.40
Mean post-HD SBP (mmHg)	0.06	0.34
Mean pre-HD DBP (mmHg)	0.10	0.11
Mean post-HD DBP (mmHg)	0.13	0.06
Mean pre-HD PP (mmHg)	0.06	0.38
MAP post-HD PP (mmHg)	−0.02	0.76
URR (%)	0.07	0.78
Hemoglobin (g/dl)	−0.01	0.92
Adjusted Ca (mmol/L)	−0.11	0.12
Phosphate (mmol/L)	−0.03	0.63
Albumin (g/dl)	0.02	0.78
PTH (pmol/L)	−0.03	0.72

BMI, body mass index.

Table 3. Multivariate linear regression entering only significant correlates into the (backward stepwise) model (adjusted R^2 = 0.49)

	B	β	P	95% CI
Constant	−13.5		0.40	−45.33 to 18.3
EDV/BSA	0.80	0.66	<0.001	0.66 to 0.91
Mean pre-HD SBP	0.24	0.13	0.01	0.05 to 0.42
Ca × PO$_4$ product	5.38	0.16	0.003	1.53 to 9.24

B, unstandardized regression coefficient; β, standardized β coefficient; CI, confidence interval. Units of measurement as before.

LVH is common in patients with ESRD and may contribute to arrhythmias as a result of impaired subendocardial perfusion and increased myocardial fibrosis (19). These changes cause chaotic, nonuniform, cardiac myocyte depolarization and repolarization, which, in turn, increase the risk for re-entrant tachyarrhythmias. Thus, identification of factors that are linked to LVH may lead to new treatment strategies to address cardiovascular risk in ESRD.

Several studies have used echocardiography to study the determinants of LVMI and LVH. They identified factors that increase cardiac preload (*e.g.,* salt and water retention, presence of arteriovenous fistulas and anemia), afterload (hypertension, calcific arteriosclerosis), and humoral factors (in-

Table 4. Univariate and multivariate logistic regression analyses determining presence of LVH

Variable	Univariate Analyses			Multivariate Analyses		
	OR	95% CI	P	OR	95% CI	P
EDV/BSA (per ml/m^2)	1.08	1.04 to 1.13	0.001	1.06	1.04 to 1.08	<0.001
Mean Ca $\times$ PO$_4$ (per mmol^2/L^2)	1.37	1.01 to 1.87	0.04	1.74	1.17 to 2.57	0.006
Mean pre-HD SBP (per mmHg)	1.02	1.01 to 1.05	0.04	1.02	1.01 to 1.04	0.01
Age (per year)	0.99	0.99 to 1.02	0.70			
Gender (male *versus* female)	1.55	0.91 to 2.65	0.11			
BMI (per kg/m^2)	0.96	0.89 to 1.03	0.27			
HD duration (per year)	1.01	0.85 to 1.20	0.88			
Ischemic heart disease	1.34	0.67 to 2.60	0.38			
Diabetes	1.20	0.69 to 2.60	0.52			
Chronic heart failure	0.88	0.28 to 2.72	0.82			
Cerebrovascular disease	2.76	0.96 to 6.93	0.09			
Peripheral vascular disease	0.83	0.29 to 2.42	0.74			
Hypertension	0.71	0.30 to 1.70	0.45			
UF volume (per L)	1.26	0.95 to 1.66	0.11			
URR (per %)	1.06	0.91 to 1.23	0.45			
Mean post-HD SBP (per mmHg)	0.99	0.97 to 1.02	0.55			
Mean pre-HD DBP (per mmHg)	0.98	0.95 to 1.01	0.21			
Mean post-HD DBP (per mmHg)	1.01	0.98 to 1.04	0.49			
Mean pre-HD PP (per mmHg)	1.02	1.01 to 1.05	0.05			
Mean post-HD PP (per mmHg)	0.99	0.97 to 1.01	0.47			
Ejection fraction (per %)	0.95	0.88 to 1.03	0.21			
ESV/BSA (per ml/m^2)	0.93	0.85 to 1.02	0.12			
Mean hemoglobin (per g/dl)	0.86	0.60 to 1.23	0.41			
Mean adjusted Ca (per mmol/L)	1.61	0.35 to 7.48	0.54			
Mean serum PO4 (per mmol/L)	2.10	1.00 to 4.29	0.06			
Mean albumin (per g/dl)	1.00	0.96 to 1.05	0.91			
Mean PTH (per pmol/L)	0.99	0.99 to 1.01	0.54			

BMI, body mass index; CI, confidence interval; OR, odds ratio; PP, pulse pressure.

cluding PTH and catecholamines) (20,21); however, there are conflicting results, presumably as a result, in part, of the unreliability of echocardiographic measurement of LVMI in HD patients (12). This is the first study to identify independent determinants of LVMI and LVH in HD patients using CMR. The intravascular volume-dependent inaccuracies of echocardiography are avoided by CMR scanning as a result of direct characterization of the LV borders. Our study also differs from previous published data because we obtained serial blood and dialysis data during 180 d as opposed to single results obtained at the time of imaging. Our data show that both LVMI and presence of LVH are associated with and are independently predicted by high end-diastolic LV volumes, higher pre-HD SBP, and Ca $\times$ PO$_4$.

EDV: Increased Cardiac Preload

In our study, EDV/BSA was the strongest parameter that predicted LVMI and LVH in multivariate analyses. This reflects the notion that development of LVH in ESRD is due, in part, to the adaptive response of the left ventricle to long-term volume overload. LV mechanical stretch stimulates cardiac sarcomere production and LV wall hypertrophy in an attempt to reduce wall stress (19); however, as wall thickening worsens, capillary vessels fail to develop adequately and perfuse the myocardium, particularly in the subendocardium. This results in myocyte ischemia and death, followed by myocardial fibrosis, systolic dysfunction, and further ventricular dilation. Our data support this mechanism: LVMI correlates positively with EDV/BSA and ESV/BSA and negatively with LVEF.

Pre-HD BP and Ca $\times$ PO$_4$: Increased Afterload

The other major factors that were associated with LVH in our study are those that have been associated with increased afterload: hypertension and Ca $\times$ PO$_4$. These observations are consistent with many reports of an association between BP and LVH in HD patients (20,22,23). Although measurement of BP before starting and immediately at the end of dialysis is highly variable, dependent on fluid volume status and the hemodynamic effects of dialysis, it remains the clinical standard. Twenty-four-hour ambulatory BP monitoring studies have confirmed these associations (24), but this method is not universally available, is time-consuming, and is uncomfortable for dialysis patients.

Elevated Ca $\times$ PO$_4$ is a risk factor for arterial calcification

and, as a consequence, contributes to BP and the development of LVH. It has been shown to be an independent predictor of cardiovascular and sudden death in patients with ESRD (25). Other measurements and markers of vascular calcification, such as in pulse wave velocity and electron-beam computed tomography, have demonstrated an association with LVH in patients with ESRD (26,27). Previous studies demonstrated a potential role of PTH in promoting myocardial fibrosis (8), but our data showed no correlation between LVMI and PTH.

Anemia as a predictor of LVMI?

Our study did not demonstrate a predictive role of hemoglobin concentration for LVMI. This contrasts with other published data that demonstrated a strong correlation between LVM and anemia (11,28), and the most likely explanation is the higher values and narrower range in our cohort (mean hemoglobin 11.3 g/dl; SD 1.3) compared with other studies.

Conclusions

Although previous studies have examined the determinants of LVH using echocardiography, the use of which is inaccurate in patients with ESRD, this study has confirmed that the main determinants of LVH in ESRD are preload and afterload. We accept these data are limited by the relatively short period (180 d) of collection and absence of repeat CMR scanning of patients. Nonetheless, given the futility of conventional CV risk management (*e.g.* with statins) in this population, LVH offers a therapeutic target and improved BP, fluid volume, and bone mineral control offer interventions with potential to prevent or regress LVH. In addition, CMR offers a high fidelity assessment of LVMI that will allow the use of small populations to power studies of LVH in this population.

Acknowledgments

Disclosures

None.

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