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CLINICAL STUDY

BMI, spKt/V, and SBP but Not DBP Are Related to LVH in Chinese Maintenance Hemodialysis Patients

XueSen Cao[1], JianZhou Zou[1], Jie Teng[1], YiHong Zhong[1], Jun Ji[1], ZhangWei Chen[2], ZhongHua Liu[1], Bo Shen[1] and XiaoQiang Ding[1]

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Abstract

Objective: Left ventricular hypertrophy (LVH) is the strongest predictor of cardiovascular mortality, the leading cause of death in hemodialysis (HD) patients. This study aims to identify the potential risk factors for LVH in HD patients. *Methods*: Exactly, 164 patients (84 men and 80 women) who had been on HD treatment for at least 6 months were enrolled. Clinical data were collected. Anthropometric measurements, biochemical analyses, and echocardiography were performed. The risk factors were determined by multivariate linear and logistic regression. *Results*: In all the patients, the prevalence of LVH was 66.5%. The patients with LVH had higher body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure, and lower single-pool kt/V (spKt/V) compared with those without LVH. Multivariate linear regression showed that BMI ($\beta = 7.608$, $p = 0.014$), SBP ($\beta = 9.462$, $p = 0.001$), and spKt/V ($\beta = -14.226$, $p = 0.024$) were independently correlated with left ventricular mass index (LVMI). Multivariate logistic regression showed the same results that BMI ($\beta = 7.193$, $p = 0.032$), SBP ($\beta = 9.382$, $p = 0.02$), and spKt/V ($\beta = -12.535$, $p = 0.001$) were independently correlated with LVH. *Conclusions*: In Chinese maintenance hemodialysis patients, BMI, single-pool Kt/V (spKt/V), and SBP were independently correlated with left ventricular mass index and were independent risk factors for LVH.

Keywords: Body mass index, spKtV, systolic blood pressure, left ventricular hypertrophy, hemodialysis

INTRODUCTION

In end-stage renal disease (ESRD), the prevalence of cardiac disease is very high.[1] It is the leading cause of death among maintenance hemodialysis (MHD) patients. Left ventricular hypertrophy (LVH) is a common complication that contributes substantially to high cardiovascular mortality and morbidity in ESRD.[2] The prevalence of LVH is also very high, ranging from 60% to 80%.[3,4] It can occur very early in the progression of chronic kidney disease and may be present at the beginning of dialysis treatment.[5]

LVH is currently considered the strongest predictor of cardiovascular and total mortality in the hemodialysis (HD) population. Up to now, there has been no consensus on the risk factors related to LVH. This study was conducted on MHD patients in China to identify potentially modifiable risk factors for better intervention in future.

PATIENTS AND METHODS

Patients

Briefly, 164 patients (84 men and 80 women) who had been on HD treatment for at least 6 months (median duration of dialysis treatment, 48 months; interquartile range, 24–78 months) were enrolled from Blood Purification Center, Zhongshan Hospital, Fudan University, Shanghai, China. These patients represented about 90% of the whole HD population in our center. The enrollment was completed within 1 month from 15 July to 15 August 2009. In our center, all HD patients were advised to have a high-protein diet (at least 1.2 g/kg/d, with mainly animal protein). The study was approved by the Ethical Committee, Zhongshan Hospital, Fudan University, and all the patients provided written informed consent.

Patients were treated thrice weekly with standard bicarbonate dialysate (Na^+ 138.0 mmol/L, HCO_3^- 32.0 mmol/L, K^+ 2.0 mmol/L, Ca^{2+} 1.25 mmol/L,

Mg^{2+} 0.5 mmol/L) by low-flux HD using 1.4 m² dialyzers with synthetic membranes (BLS514SD, Sorin Group Italia, Mirandola, Italy; Polyflux14L, Gambro Dialysatoren GmbH, Hechigen, Germany). The blood flow was 200–280 mL/min, and dialysate flow was 500 mL/min. Dry weight was targeted in every patient to achieve an edema-free state.

Blood Pressure Measurements
Blood pressure was estimated by averaging all predialysis blood pressure during the month before this study (12 measurements in total).

Anthropometric Measurements and Clinical Data Collection
Height and weight were measured while participants were barefoot and wearing light clothes only. The body mass index (BMI) was calculated as weight (kg) divided by height (m) squared. Demographic and clinical data were collected, including age, gender, smoking history, underlying kidney disease, dialysis duration, comorbidity, initial urine output at the onset of HD, and current urine output, on which urine reduction rate was determined.

Biochemical Measurements
Blood sampling was performed during a midweek nondialysis day 8–10 am after 30 min of quiet rest in semirecumbent position. Serum albumin, pre-albumin, hemoglobin, blood urea nitrogen, creatinine, calcium, phosphorus, lipids, homocysteine, ion, ferritin, and transferrin were measured using standard methods in the routine clinical laboratory. The concentrations of high-sensitivity C-reactive protein and β_2-microglobulin were determined using immunoturbidimetry assay, and concentrations of intact parathyroid hormone (iPTH) were measured using electrochemiluminescence immunoassay.

Echocardiography
Transthoracic echocardiographic examinations were conducted using a Philips echocardiographic machine (Philips IE33, Eindhoven, the Netherlands) with a 3.5-MHz multiphase array probe by a single experienced cardiologist during a midweek nondialysis day, within 2 h after blood sampling. Measurements of the left ventricular internal dimension, interventricular septal thickness, and posterior wall thickness were made at end diastole according to the recommendations of Penn Convention. Left ventricular mass was calculated with the Devereux formula,[6] and left ventricular mass index (LVMI) was obtained by dividing left ventricular mass by height in meters raised to the power of 2.7. The left ventricular ejection fraction was determined by two-dimensional echocardiography.

Statistical Analysis
All data were expressed as mean ± SD or median (interquartile range) appropriately. To compare two groups of normal variables, independent samples t-test was used, whereas for skewed and categorical variables Mann–Whitney U and chi-square test were performed, respectively. One-way ANOVA and Kruskal–Wallis test were employed for analyzing multigroup of variables. Linear and logistic regressions were performed to identify the independent risk factors for LVMI and LVH. Covariates with $p < 0.1$ in the univariate regression were selected for multivariate regression analysis. Two-sided $p < 0.05$ was considered significant. All analyses were performed using SPSS version 17.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

Patient Characteristics
One hundred and sixty-four HD patients (84 men and 80 women) participated in this study. The mean age was 57.0 ± 13.8 years. The median dialysis duration was 48 (24–78) months. The initial and current urine output were 21.1 ± 11.4 and 0 (0–5.05) mL/kg/24 h, respectively. The delivered dialysis dose measured by single-pool kt/V (spKt/V; second-generation Daugirdas equation) was 1.4 ± 0.3 or by urea reduction rate was 0.7 (0.6–0.7). Of the 164 patients, 52 had smoking history, 8 coronary heart disease, 20 type 2 diabetes mellitus, 20 cerebral infarction, and 6 cerebral hemorrhage. On echocardiographic examination, valvular diseases were found in nine patients (one moderate aortic regurgitation, two mild aortic regurgitation, one mild aortic stenosis, three mild mitral regurgitation, and two mild tricuspid regurgitation). Altogether 119 patients were on antihypertensive treatment (32 on monotherapy with calcium channel blocker, angiotensin-converting enzyme inhibitor, angiotensin-II receptor blocker, β-blocker or α-blocker, and 87 on double or triple therapy with various combinations of these drugs), whereas 123 patients were taking oral calcium salt, with calcitriol prescribed according to the serum iPTH level. Only 12 patients were taking statin lipid-lowering drugs. All patients received erythropoietin treatment, and intravenous iron was used when necessary. The underlying kidney diseases are listed in Table 1.

The demographic, anthropometric, and clinical characteristics and biochemical parameters were given in Table 2. The average BMI was 22.0 ± 3.1 kg/m², and patients with LVH had higher BMI than those without LVH (22.4 ± 3.4 vs. 21.2 ± 2.2 kg/m², $p = 0.047$). In both groups, the average blood pressure was 136.6 ± 16.8/90.0 ± 9.8 mmHg. Patients with LVH had significantly higher systolic blood pressure (SBP) and diastolic blood pressure (DBP) compared with those without LVH (140.2 ± 14.6/83.5 ± 9.7 vs. 129.5 ± 18.5/80.0 ± 9.2 mmHg, $p < 0.0001$ and $p = 0.004$, respectively). No significant difference was found among the biochemical parameters. With regard to dialysis adequacy, we found that patients without LVH

Table 1. The underlying kidney diseases of 164 MHD patients.

Underlying kidney disease	n	Percentage
Glomerular disease	76	46.3
Diabetic nephropathy	13	7.9
Hypertensive nephropathy	13	7.9
Polycystic kidney disease	14	8.5
Aristolochic acid nephropathy	6	3.7
Lupus nephritis	4	2.4
Chronic pyelonephritis	3	1.8
Gouty nephropathy	3	1.8
Multiple myeloma nephropathy	2	1.2
Penicillamine-induced nephropathy	1	0.6
Obstructive nephropathy	1	0.6
Solid renal tumor	1	0.6
Unknown	27	16.5

had higher spKt/V (1.5 ± 0.3 vs. 1.3 ± 0.3, $p < 0.0001$) and urea reduction rate [0.71 (0.66–0.77) vs. 0.66 (0.61–0.73), $p < 0.001$] compared with those with LVH, whereas there was no significant difference in initial and current urine output, urine reduction rate, smoking history, or dialysis duration between patients with and without LVH ($p = 0.359$, $p = 0.729$, $p = 0.591$, $p = 0.386$, and $p = 0.17$, respectively).

Prevalence of LVH

LVH was defined by a LVMI of >47 $g/m^{2.7}$ in women or >50 $g/m^{2.7}$ in men.[7] The height-based indexing was specifically chosen to minimize any potential distortion attributable to extracellular volume expansion. According

Table 2. The demographic, anthropometric, clinical characteristics, and biochemical parameters of 164 MHD patients.

	All patients	LVH Yes	LVH No	p-Value
Age, years	57.0 ± 13.8	56.9 ± 13.7	57.2 ± 14.0	0.87
Gender (male/female)	84/80	57/52	27/28	0.698
BMI, kg/m^2	22.0 ± 3.1	22.4 ± 3.4	21.2 ± 2.2	0.047
Initial urine output, mL/kg/24 h	21.1 ± 11.4	20.5 ± 11.0	22.3 ± 12.0	0.359
Current urine output, mL/kg/24 h	0 (0–5.05)	1.2 (0.5–2.3)	1.0 (0.4–2.2)	0.729
Urine reduction rate, mL/kg/month	1.21 (0.56–2.49)	1.23 (0.58–2.47)	1.21 (0.46–2.52)	0.591
Cigarette smoking (yes/no)	52/112	37/72	15/40	0.386
Dialysis duration, month	48 (24–78)	42 (18–77)	52 (30–82)	0.17
SBP, mmHg	136.6 ± 16.8	140.2 ± 14.6	129.5 ± 18.5	<0.0001
DBP, mmHg	90.0 ± 9.8	83.5 ± 9.7	80.0 ± 9.2	0.004
Albumin, g/L	39.3 ± 3.7	39.3 ± 3.5	39.3 ± 4.0	0.911
Pre-Albumin, g/L	0.34 ± 0.08	0.33 ± 0.08	0.34 ± 0.08	0.467
Hemoglobin, g/L	104.1 ± 15.0	104.4 ± 14.6	103.5 ± 16.0	0.734
BUN, mmol/L	23.6 ± 5.1	23.7 ± 4.6	23.5 ± 6.1	0.863
Creatinine, μmol/L	1018.5 ± 246.8	1014.4 ± 237.9	1026.4 ± 265.4	0.772
Uric acid (UA), μmol/L	434.5 ± 89.0	441.1 ± 77.6	421.7 ± 102.3	0.179
Calcium, mmol/L	2.24 ± 0.20	2.25 ± 0.20	2.23 ± 0.21	0.575
Phosphorus, mmol/L	2.22 ± 0.66	2.27 ± 0.64	2.13 ± 0.69	0.208
Serum iron, μmol/L	11.6 ± 5.9	11.3 ± 6.0	12.2 ± 5.6	0.375
Serum ferritin, ng/mL	139.8 (81.5–284.2)	146.1 (86.0–306.9)	113.3 (69.9–234.5)	0.196
Transferrin, g/L	1.9 ± 0.5	1.9 ± 0.5	1.9 ± 0.4	0.926
iPTH, pg/mL	275.2 (117.8–563.9)	286.0 (143.6–629.9)	215.3 (91.7–545.9)	0.089
hsCRP, mg/L	1.7 (0.7–6.0)	2.2 (0.7–6.2)	1.1 (0.6–3.7)	0.112
Triglyceride, mmol/L	1.5 (1.1–2.1)	1.5 (1.1–2.3)	1.5 (1.2–2.0)	0.868
Total cholesterol, mmol/L	4.6 ± 1.2	4.6 ± 1.1	4.6 ± 1.2	0.915
High-density lipoprotein cholesterol (HDL-C), mmol/L	1.2 ± 0.3	1.2 ± 0.3	1.2 ± 0.4	0.925
Low-density lipoprotein cholesterol (LDL-C), mmol/L	2.6 ± 0.9	2.5 ± 0.9	2.6 ± 1.0	0.401
Apoprotein A (Apo-A), g/L	1.3 ± 0.3	1.3 ± 0.2	1.3 ± 0.3	0.791
Apoprotein B (Apo-B), g/L	0.87 ± 0.21	0.87 ± 0.21	0.86 ± 0.23	0.804
Lipoprotein(a) (Lp (a)), mg/L	167 (114–289)	173 (109–289)	157 (121–301)	0.527
Homocysteine, μmol/L	34.4 (27.7–44.1)	35.4 (27.8–46.0)	32.9 (27.1–40.8)	0.488
β_2-Microglobulin, mg/L	35.6 ± 10.9	35.5 ± 11.6	35.8 ± 9.4	0.878
LVMI, $g/m^{2.7}$	62.4 ± 19.5	68.0 ± 15.6	38.7 ± 7.3	<0.0001
Ejection fraction (EF), %	66.8 ± 6.9	66.3 ± 7.5	67.7 ± 5.5	0.229
spKt/V	1.4 ± 0.3	1.3 ± 0.3	1.5 ± 0.3	<0.0001
URR	0.7 (0.6–0.7)	0.66 (0.61–0.73)	0.71 (0.66–0.77)	<0.001

Notes: Data are reported as mean ± SD or median and interquartile range. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; BUN, blood urea nitrogen; hsCRP, high-sensitivity C-reactive protein; URR, urea reduction rate; UA, uric acid; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Apo-A, apoprotein A; Apo-B, apoprotein B; Lp(a), lipoprotein (a); EF, ejection fraction.
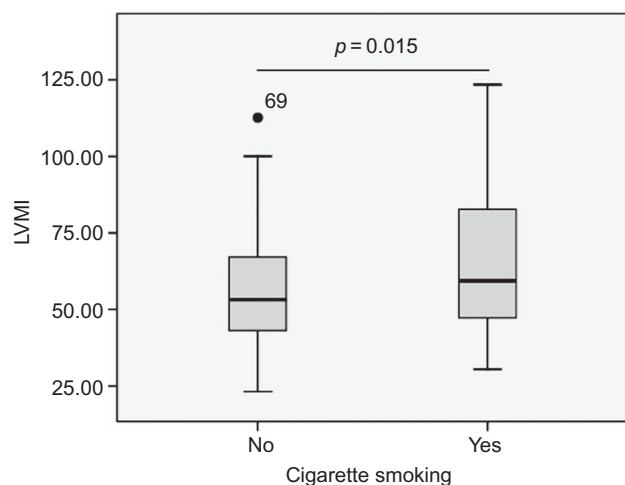
Figure 1. Impact of cigarette smoking on LVMI.

Influence of Cigarette Smoking and Underlying Kidney Disease on LVMI

to this criterion, the prevalence of LVH in our study group was 66.5%.

Of 164 MHD patients, 52 were smokers. The proportion of smokers to nonsmokers in the group with and without LVH were 37/72 and 15/40, respectively ($p = 0.386$, Table 2). But in the whole group, smoking patients had higher LVMI than nonsmoking patients (63.5 ± 20.7 vs. 55.7 ± 18.2 g/m$^{2.7}$, $p = 0.015$, Figure 1).

We investigated the relationship between LVMI and the top four underlying kidney diseases. The group of diabetic nephropathy had the highest LVMI of 73.7 ± 20.6 g/m$^{2.7}$ compared with the group of hypertensive

nephropathy (67.4 ± 18.9 g/m$^{2.7}$), glomerular disease (56.2 ± 19.1 g/m$^{2.7}$), and polycystic kidney disease (48.4 ± 20.0 g/m$^{2.7}$) ($p = 0.002$). Post hoc analysis (Bonferroni method) showed significant difference in LVMI between the groups of diabetic nephropathy and glomerular disease ($p = 0.016$) and of diabetic nephropathy and polycystic kidney disease ($p = 0.005$, Figure 2). But the difference in LVMI between the group of hypertensive nephropathy and other three groups was not significant.

Linear Regression Model for LVMI and Related Factors

Univariate linear regression with LVMI as the dependent variable was performed. We found that BMI ($\beta = 7.982$, $p = 0.042$), SBP ($\beta = 9.513$, $p < 0.0001$), DBP ($\beta = 6.698$, $p < 0.0001$), and iPTH ($\beta = 6.355$, $p = 0.043$) were positively related to LVMI, whereas spKt/V ($\beta = -16.709$, $p = 0.002$) was inversely related to LVMI. There was also a tendency for dialysis duration ($\beta = -6.99$, $p = 0.062$) and serum iron ($\beta = -0.444$, $p = 0.097$) to be inversely related to LVMI. But age, gender, initial and current urine output, urine reduction rate, and other biochemical parameters were not correlated with LVMI. Then multivariate linear regression analysis was performed (backward stepwise method) with LVMI as dependent variable and BMI, SBP, DBP, spKt/V, iPTH, serum iron, dialysis duration, cigarette smoking, and underlying kidney disease as independent variables. The results showed that BMI ($\beta = 7.608$, $p = 0.014$), SBP ($\beta = 9.462$, $p = 0.001$), and spKt/V ($\beta = -14.226$, $p = 0.024$) were independently correlated with LVMI, whereas DBP ($p = 0.936$), iPTH ($p = 0.603$), serum iron ($p = 0.907$), dialysis duration ($p = 0.883$), cigarette
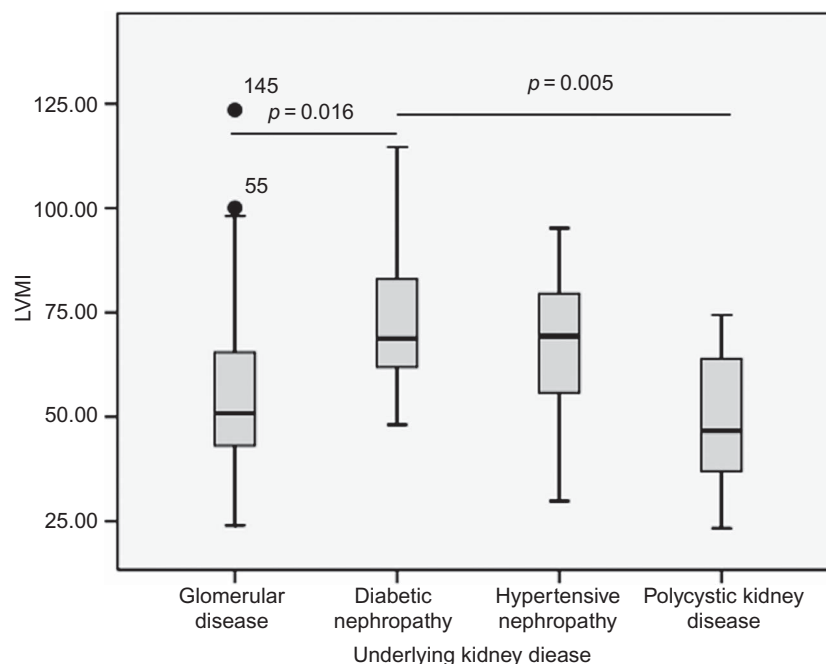

Figure 2. Relationship between underlying kidney diseases and LVMI.

Table 3. Multivariate linear regression model with LVMI as the dependent variable in 164 MHD patients.

Independent variables	β	p-Value
BMI	7.608	0.014
SBP	9.462	0.001
spKt/V	−14.226	0.024
Serum iron[a]		0.907
iPTH[a]		0.603
DBP		0.936
Dialysis duration[a]		0.883
Cigarette smoking		0.958
Underlying kidney disease		0.462

Notes: For the whole model, $F = 13.187$, $p < 0.0001$, $R^2 = 0.739$, and the residual followed Gauss distribution ($p = 0.262$). BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure.
[a]Data $\log_{10}$ transformed.

Table 4. Multivariate logistic regression model with LVH as the dependent variable in 164 MHD patients.

Variables	β	p-Value
BMI	7.193	0.032
SBP	9.382	0.02
spKt/V	−12.535	0.001
DBP		0.363
iPTH[a]		0.614

Notes: For the whole model, Cox and Snell $R^2 = 0.721$. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure.
[a]Data $\log_{10}$ transformed.

smoking ($p = 0.958$), and underlying kidney disease ($p = 0.462$) had no significance (Table 3).

Logistic Regression Model for LVH and Related Factors

First univariate logistic regression with LVH as dependent variable was performed. It was found that BMI ($\beta = 8.141$, $p = 0.019$), SBP ($\beta = 10.043$, $p < 0.0001$), DBP ($\beta = 5.05$, $p = 0.006$), and spKt/V ($\beta = −14.12$, $p = 0.001$) were significant, but iPTH ($\beta = 2.628$, $p = 0.086$) was marginally significant in contrast to the univariate linear regression. Based on the criterion of $p < 0.1$, we selected BMI, SBP, DBP, spKt/V, and iPTH as independent variables. It was also found that the results of multivariate logistic regression model (also backward stepwise method) were consistent with those of multivariate linear regression (Table 4). BMI ($\beta = 7.193$, $p = 0.032$) and SBP ($\beta = 9.382$, $p = 0.02$) were positively correlated with LVH, whereas spKt/V was inversely correlated with LVH ($\beta = −12.535$, $p = 0.001$). So it was suggested in this study that BMI, SBP, and spKt/V were the independent risk factors for LVH.

DISCUSSION

Cardiovascular events are the main cause of death in MHD patients, whereas LVH is a common complication that contributes substantially to high cardiovascular morbidity and mortality in this population. Even in the predialysis population, the prevalence of LVH increases with progressive renal decline.[5] Based on the data from our center, we found that BMI, spKt/V, and SBP were the independent risk factors for LVH.

The association between BMI and mortality is controversial. Most researchers believed that there was a U-shaped or J-shaped relation between BMI and the risk of death in the general population.[8,9] As for the HD population, greater BMI conferred a survival advantage.[10] Patients in the earlier stages of chronic kidney disease appear to represent the transition point between the general population and patients with kidney failure.[11]

Concerning the relationship between BMI and LVH, the majority view was that higher BMI led to acquisition of LVMI and predicted LVH in general population including children and adults.[12,13] In HD population, some studies showed that BMI was positively correlated with LVH[14] as in general population, whereas other studies gave different results.[15] In our study, the patients with LVH had higher BMI than those without LVH. Multivariate linear regression showed that BMI was positively correlated with LVMI, and such a positive correlation was elucidated by multivariate logistic regression.

Comparing the results of all previous studies about BMI and outcomes (mortality or LVH), we found there was no consensus especially in the HD population. This might be attributed to the confounding factors. First, the association was distorted owing to preexisting chronic disease and inadequate control for smoking status, because chronic illness and smoking were associated with decreased BMI and increased risk of the outcomes. Second, because of age-related changes in the amount and distribution of body fat, the impact of BMI on risk of mortality or LVH was dependent on the age and follow-up. Greater BMI was associated with higher mortality, but the relative risk declined with age.[16] Third, race was another confounding factor. Many studies found that with regard to the association between BMI and mortality, Asian people were an exception.[11,17] The impact of BMI on LVH was likely to vary according to race. Notably, in our center, diabetic nephropathy accounts for only 7.9% of ESRD, which is lower than 30–50% in developed countries. Furthermore, the prevalence of disturbance of lipid metabolism is relatively low, which may be attributed to the relatively low prevalence of diabetic nephropathy. Last, the impact of BMI may be obscured by physical activity, alcohol use, dietary intake, and other pathologic process, including malnutrition, inflammation, and changes in vitamin D metabolism.

In 1990s, the United States Renal Data System; HEMO Study: the Hemodialysis Study (USRDS) reported that an approximate log-linear relationship of decreasing mortality with increased dose of dialysis

existed within the range of spKt/V 0.8–1.4.[18] But the Hemodialysis (HEMO) Study gave a negative result, that is, neither the increased dose of dialysis (higher than that recommended by current US guidelines, i.e., spKt/V 1.2) nor the use of high-flux dialyzers improved outcomes.[19] As for the relationship between spKt/V and LVH, the limited data showed that spKt/V was not an independent risk factor for LVH.[20] However, when patients converted from conventional HD to daily HD with a longer time and so a higher spKt/V, LVMI decreased significantly.[21] In our study, spKt/V was negatively correlated with LVMI and an independent risk factor for LVH. This result could be explained by good control for volume overload, anemia, and hypertension with a higher spKt/V. Many studies indicated that the relative risk for morbidity and mortality in general and HD population was different concerning race and ethnicity. The association between spKt/V and LVH in Chinese people may be different from previously reported results. More studies are needed to elucidate this problem.

SBP but not DBP was a widely accepted risk factor for LVH in HD population,[22] and time-integrated SBP was an independent predictor of increase in LMVI in HD patients.[23] These results are consistent with our present findings. The increased cardiac afterload is suggested to be the major reason. But other potentially reversible factors such as anemia, volume overload, secondary hyperparathyroidism, uremic toxins, dose of dialysis, and malnutrition may also have an important role in the pathogenesis of LVH. All these confounding factors may have a part to play in the relationship between hypertension and LVH.

Although some studies indicated that active and long-term antihypertensive treatment could decrease LVMI,[24,25] in this study, it was found that the use of antihypertensive medications including calcium channel blocker, angiotensin-converting enzyme inhibitor, angiotensin-II receptor blocker, β-blocker, and α-blocker had no significant relations with LVMI or LVH (data not listed). Multicenter, randomized, double-blind, controlled studies might give a definite answer in the future. Concerning the impact of SBP on LVH, the blood pressure control especially on SBP might be needed to alleviate progression of LVH.

CONCLUSIONS

From this cross-sectional study in Chinese MHD patients, we concluded that BMI, spKt/V, and SBP were independently correlated with LVMI and were the independent risk factors for LVH. The relationships between BMI, spKt/V, and LVH, as revealed in our study, are significantly different from the results of other countries, suggesting that race might be an important factor. Although they had a detrimental effect on LVMI, smoking and underlying kidney disease lost significance in multivariate regression. Studies of large sample sizes remain to be made to further elucidate this problem.

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