Left Ventricular Hypertrophy Patterns and Cardiovascular Outcome in Peritoneal Dialysis Patients

Periton Diyalizi Hastalarında Sol Ventriküler Hipertrofi Paternleri ve Kardiyovasküler Sonuçlar

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ABSTRACT Objective: Left ventricular hypertrophy (LVH) is highly predictive of cardiovascular (CV) events in dialysis patients. The impact of different left ventricular (LV) geometric patterns on cardiovascular outcome has not been well established. The aim of this prospective observational study was to define the patterns of LVH and find out their impact on future cardiovascular events in patients on continuous ambulatory peritoneal dialysis (CAPD). **Material and Methods**: Patients were followed up between 1999 and 2006 for cardiovascular events. Left ventricular mass and geometric patterns were determined by echocardiography. Kaplan-Meier method and Cox regression analysis were used to evaluate the impact of LV geometric patterns on CV event free survival. **Results:** Sixty-five CAPD patients were enrolled in the study and were followed for 76 ± 15 months. There were a total of 16 (14 fatal, two non-fatal) CV events. Mean event-free survival time for patients without LVH was 79 ± 3 months (95% CI of 73 to 86 months). The mean CV event-free survival for the patients with concentric LV hypertrophy and eccentric hypertrophy was 75 ± 3 months (95% CI of 69 to 81 months), and 76 ± 3 months (95% CI of 69 to 82 months) respectively. Patients with eccentric [hazard ratio (HR): 20.2] and concentric (HR:18.7) LV hypertrophy had shorter time to CV event when compared to the patients without LV hypertrophy (p= 0.042). **Conclusion:** Left ventricular hypertrophy is a marker of poor cardiovascular prognosis irrespective of its pattern. Both eccentric and concentric LV hypertrophy are associated with shorter CV event-free survival compared to normal geometry and concentric remodeling.

Key Words: Peritoneal dialysis, continuous ambulatory; hypertrophy, left ventricular

ÖZET Amaç: Sol ventrikül hipertrofisi (SVH) diyaliz hastalarında kardiyovasküler (KV) olaylar açısından yüksek prediktif değerdedir. Farklı sol ventrikül (LV) geometrik paternlerinin kardiyovasküler sonuçlara etkisi çok iyi bilinmemektedir. Bu prospektif gözlemsel çalışmada LVH paternlerini tanımlamak ve sürekli ayaktan periton diyalizi (SAPD) hastalarında gelecekteki kardiyovasküler olaylar üzerindeki etkilerini araştırmak amaçlandı. **Gereç ve Yöntemler**: Hastalar 1999 ve 2006 yılları arasında kardiyovasküler olaylar açısından izlendi. Sol ventrikül kitlesi ve geometrik paternleri ekokardiyografi ile tespit edildi. LV geometrik paternlerinin KV olaysız sağ kalıma etkisini değerlendirmek için Kaplan-Meier yöntemi ve Cox regresyon analizi kullanıldı. **Bulgular:** Altmış beş SAPD hastası çalışmaya alındı ve 76 ± 15 ay boyunca takip edildi. Toplam 16 (14 ölümcül, iki ölümcül olmayan) KV olay meydana geldi. LVH olmayan hastalar için ortalama olaysız sağ kalım süresi 79 ± 3 ay (%95 güvenlik indeksi, 73-86 ay) idi. Konsantrik sol ventrikül hipertrofisi ve ekzantrik hipertrofisi olan hastalar için ortalama KV olaysız sağ kalım 75 ± 3 ay (%95 güvenlik indeksi, 69-81 ay) ve 76 ± 3 ay (%95 güvenlik indeksi, 69-82 ay) idi. Eksantrik [risk oranı (HR): 20.2] ve konsantrik (HR: 18.7) sol ventrikül hipertrofisi olan hastalar, sol ventrikül hipertrofisi olmayan hastalarla karşılaştırıldığında KV olaylar daha kısa sürede ortaya çıktı (p= 0.042). **Sonuç:** Sol ventrikül hipertrofisi, paterninden bağımsız olarak kötü kardiyovasküler prognoz göstergesidir. Hem eksantrik hem konsantrik sol ventrikül hipertrofisi, normal geometri ve konsantrik remodeling ile karşılaştırıldığında daha kısa KV olaysız sağkalım ile ilişkilidir.

Anahtar Kelimeler: Periton diyalizi, sürekli ayaktan; hipertrofi, sol ventriküler

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Cardiovascular (CV) disease is the major cause of death in dialysis patients, and survival in patients on continuous ambulatory peritoneal dialysis (CAPD) is similar to that in hemodialysis (HD) patients.[1,2] In a previous study by Wang et al., cardiac and cerebrovascular causes accounted for 65% of the mortality in end-stage renal disease (ESRD) patients receiving long-term peritoneal dialysis.[3]

Left ventricular hypertrophy (LVH) is common in patients with renal insufficiency even before they progress to dialysis, and the prevalence of LVH correlates with the degree of renal function impairment.[4,5] Left ventricular hypertrophy is known to be highly prevalent in both CAPD and HD patients[6,7] and the presence of LVH has been confirmed as an important determinant of survival in dialysis patients, as in the general population.[8-10]

Hemodynamic and non-hemodynamic factors are implicated in the maintenance and aggravation of LVH in ESRD. The balance between the two fundamental hemodynamic stimuli (pressure and volume overload) determines the type of geometric development of the left ventricle.[11] Four different patterns (normal,concentric remodeling, eccentric hypertrophy, concentric hypertrophy) of left ventricular (LV) geometry has been described.[12] Eccentic left ventricular hypertrophy (eLVH) refers to increased LV mass with LV dilatation, whereas concentric left ventricular hypertrophy (cLVH) denotes increased LV mass with normal LV cavity volume. LV dilatation is commonly observed in states of volume overload and pressure overload and it leads primarily to increase in LV mass.[13] Dialysis patients have a number of risk factors for both volume and pressure overload. Although the prevalence of the concentric and eccentric form of LVH was similar in the two dialytic modalities, CAPD patients were found to be more volume expanded and display more severe LVH than HD patients, in a study done by Enia et al.[14] In a population-based sample of subjects without CV disease it was shown that subjects with cLVH had the poorest prognosis.[15] The influence of different LV geometric patterns on prognosis were studied in patients with hypertension and coronary artery disease, and patients with cLVH appeared to have the poorest prognosis.[16,17] According to a large body of evidence, LVH is accepted as a strong predictor of survival and cardiovascular events in dialysis patients, but the impact of different patterns of LVH on CV prognosis has not been well elucidated. We aimed to investigate the prevalence of LVH, the distribution of LV geometric patterns and their impact on cardiovascular events in CAPD patients.

MATERIAL AND METHODS

STUDY DESIGN AND PATIENTS

This study was started in 1999 and carried out in the CAPD Unit of Marmara University Hospital, İstanbul. Patients were eligible for entry to the study if (a) they were on CAPD for at least six months and (b) they had a technically satisfactory echocardiogram. Patients with cancer, severe valvular heart disease, severe pulmonary disease and history of previous cardiovascular events which are described below were not included. After baseline registration of clinical data and echocardiographic examination, each patient was monitored for CV events until December 2006. Each patient was followed up until the first cardiovascular event. Study protocol was approved by the local ethics committee and informed written consents were obtained from all participants. Eighty-four CAPD patients were monitored and 66 were found to be eligible for recruitment. Thirteen patients with previous myocardial infarction (n= 4), coronary artery bypass surgery (n= 1), history of congestive heart failure (n= 5), atrial fibrillation (n= 3) were excluded. Five patients were not enrolled due to technically poor echocardiographic study. One patient was lost to follow-up. Sixty-five CAPD patients constituted the final study group. Four patients who moved to another CAPD center throughout the study period were contacted by telephone to get information about their outcome. Other patients were followed by their routine monthly visits and by medical records. Peritoneal dialysis treatment was mainly performed by manual exchanges of Dianeal® 1.36% solutions. If the patients were volume overloaded, Dianeal® 2.27% and 3.86% solutions were prescribed.

Cardiovascular outcome events were prespecified as; sudden cardiac death, fatal and nonfatal myocardial infarction, new-onset angina, arrhythmias requiring hospitalization, severe heart failure requiring hospitalization, fatal and nonfatal stroke. Official hospital documents and standart international criteria were used for the definition of outcome events. Sudden cardiac death was defined as a witnessed death from cardiac causes that occured within an hour after the onset of acute symptoms. Myocardial infarction was diagnosed on the basis of typical rise and fall of cardiac enzymes (CK-MB, troponin T) with either ECG changes indicative of ischemia or ischemic symptoms. New-onset angina was defined as typical chest pain symptoms with objective evidence of ischemic ECG changes. Stroke was diagnosed on the basis of rapid onset of localizing and persistent neurological deficit in the absence of any other disease process explaining the symptoms.

Laboratory measurements were done by standard procedures. Parathyroid hormone levels were determined using radioimmune assay (Sigma-Aldrich Laboratories, The Woodlands, TX, USA).

ECHOCARDIOGRAPHIC STUDY

Two-dimensional guided M-mode echocardiography was performed by standard methods using an ultrasound system (Ultramark 9; Advanced Technology Laboratories, Bothell, WA, USA) with a 2.25 MHz transducer. Examinations were performed with an empty stomach. Left ventricular internal dimension (LVID), interventricular septal thickness (IVST) and posterior wall thickness (PWT) were measured at end-diastole according to the American Society of Echocardiography recommendations.[18] Left ventricular mass (LVM) was calculated using the formula described by Devereux et al.[19]: $0.832 \times [(LVID + IVST + PWT)^3 - LVID^3] + 0.6$. Left ventricular mass was then normalized for body surface area (BSA) as left ventricular mass index (LVMI). Left ventricular mass index partition values of 131 g/m^2 for men and 100 g/m^2 for women were used as the upper gender-specific normal.[20] Relative wall thickness (RWT) of the LV was calculated at the end-diastole as 2 x (PWT / LVID). Left ventricular geometric patterns were defined according to the method of Ganau et al.[21] A partition value of 0.44 for RWT was used for both men and women. Normal geometry was present when LVMI and RWT were normal. Normal LVMI and increased RWT were classified as concentric remodeling. These two formed the noLVH group. Increased LVMI with a normal RWT identified eLVH (LVH with left ventricular dilatation). Increases in both LVMI and RWT were classified as cLVH (LVH without left ventricular dilatation). All echocardiographic examinations were performed by the same physician.

STATISTICAL ANALYSIS

Data analyses were performed by using SPSS software, version 15.0 (SPSS, Inc., Chicago, IL, USA). For the comparison of clinical and echocardiographic variables between CAPD patients with and without cardiovascular events, Mann Whitney U test or t-test was used for continuous data and Chi square or Fisher's exact test were used for categorical data, where appropriate. Participants were grouped into three categories of LV geometry; eccentric LV hypertrophy, concentric LV hypertrophy and the combination of normal geometry and concentric remodeling (noLVH group; reference category). For univariate analysis, event-free survival was calculated and log rank test was performed. Survival probabilities of different groups were calculated by Kaplan-Meier method and Cox regression analysis, which was performed by enter method. Cox regression model included gender, body mass index, systolic and diastolic blood pressures, hemoglobin, albumin, LDL-cholesterol, LV mass index, smoking status and the presence of hypertension, coronary artery disease, diabetes mellitus, hyperlipidemia and peripheral artery disease in addition to LV geometry types. A p value of <0.05 was considered significant.

RESULTS

Sixty five CAPD patients (mean age 46 years, 55% men) were followed for CV events. Baseline clinical and demographic characteristics of the study population are presented in Table 1. Hypertensive

nephropathy and glomerulonephritis were the most frequent primary renal diseases. Seven patients had previously documented coronary artery disease, five had diabetes mellitus and 55 had hypertension. All of the patients were in good blood pressure control throughout the study. Fifty two (80%) patients were on erythropoetin treatment, mean hemoglobin level was 10.6 g/dl (range, 6.9 to 14.0). Nineteen patiens (29%) were anuric. Patients were on CAPD treatment for 19±12 months (range, 6 to 43) before entering to the study.

After a mean follow-up period of 76 ± 15 months (range, 15 to 84 months), two patients had nonfatal CV events, 14 patients died because of CV events and seven patients died of non-CV events. Cardiovascular mortality was responsible for 67% of the total mortality. Five of the seven CAPD patients with previously documented coronary artery disease had CV events. Cardiovascular events and other causes of death in CAPD patients are listed in Table 2. Ten patients were switched to hemodialysis and four patients had renal transplantation during follow-up. No CV event was observed in those patients.

As listed in Table 3, CAPD patients with CV events were older and had higher prevalance of coronary artery disease compared with the patients without CV events. Forty five patients had LVH (69%), of these 21(47%) had eccentric and 24 (53%) had concentric type of hypertrophy. In the noLVH group (n= 20), 13 (65%) had normal geometry and seven (35%) had concentric remodeling. Cardiovascular events tended to occur in a higher proportion of patients with (14/45,31%) than without (2/20,10%) left ventricular hypertrophy (p= 0.068). Eighty eight percent (14/16) of patients with CV events had LVH. Patients with concentric remodeling (n= 7) did not experience any CV event during follow-up. Two of the patients with normal geometry (n= 13, 15%), seven of the patients with concentric LV hypertropy (n= 24, 29%) and seven of the patients with eccentric LV hypertrophy (n= 21, 33%) reached CV end-points during follow-up.

Mean CV event-free survival time for the CAPD patients with normal geometry and concentric remodeling (noLVH group) was 79 ± 3 months (95% CI of 73 to 86 months). The probability of CV event-free survival for noLVH group was %90. The corresponding mean CV event-free survival time for the patients with concentric LV hypertrophy

TABLE 1: Clinical and demographic characteristics of the study population (n= 65).

Age, years	46 ± 15*
Men / women, n	36 (55%) / 29 (45%)
Duration of CAPD, months	19±12
Primary renal disease	
Diabetic nephropathy	7 (11%)
Glomerulonephritis	16 (25%)
Hypertensive nephropathy	19 (29%)
Genetic	7 (10%)
Tubulointerstitial	4 (6%)
Other	7 (11%)
Unknown	5 (8%)
Diabetes	5 (8%)
Hypertension	55 (85%)
Hyperlipidemia	38 (58%)
Coronary artery disease	7 (11%)
Smoking	30 (45%)
Peripheral artery disease	3 (4%)

*Data expressed as mean ± SD.
CAPD: Continuous ambulatory peritoneal dialysis.

TABLE 2: Cardiovascular events and other causes of death in study patients (n= 65).

Outcome	n
Nonfatal cardiovascular events	
Arryhthmia	1
Myocardial infarction	1
Subtotal	2
Fatal cardiovascular events	
Sudden cardiac death	6
Myocardial infarction	1
Heart failure	1
Stroke	6
Subtotal	14
Other causes of death	
Sepsis	6
Gastrointestinal bleeding	1
Subtotal	7

TABLE 3: Comparisons of clinical and echocardiographic variables in CAPD patients with and without subsequent cardiovascular (CV) events.

Variables	No CV event (n=49)	CV event (n=16)	p value
Age, years	44 ± 14	53 ± 17	0.04
Men, n (%)	25 (51)	11 (69)	0.22
BMI, kg/m^2	24 ± 4	24 ± 4	0.33
Cigarette smoking, n (%)	22 (45)	7 (44)	0.94
Diabetes, n (%)	2 (4)	3 (19)	0.09
Coronary artery disease, n (%)	2 (4)	5 (31)	<0.01
Hyperlipidemia, n (%)	29 (59)	9 (56)	0.84
Hypertension, n (%)	43 (88)	15 (94)	0.45
Systolic blood pressure, mm Hg	151 ± 28	158 ± 15	0.28
Diastolic blood pressure, mm Hg	95 ± 17	99 ± 15	0.45
LDL-cholesterol, mg/dl	128 ± 38	123 ± 44	0.54
HDL-cholesterol, mg/dl	41 ± 12	43 ± 13	0.89
Triglycerides, mg/dl	149 ± 59	179 ± 91	0.25
Hemoglobin, g/dl	11 ± 2	11 ± 2	0.94
Albumin, g/dl	3.9 ± 0.4	3.6 ± 0.7	0.11
Parathormone, pg/ml	326 ± 382	165 ± 164	0.27
Calcium, mg/dl	9.3 ± 0.8	9.2 ± 1.0	0.91
Phosphorus, mg/dl	5.3 ± 1.3	5.0 ± 1.2	0.45
Urine volume, ml/day	395 ± 459	619 ± 641	0.23
Echocardiographic data			
LVEDD, cm	5.0 ± 0.7	5.2 ± 0.7	0.26
IVST, cm	1.2 ± 0.2	1.3 ± 0.2	0.25
PWT, cm	1.1 ± 0.2	1.2 ± 0.1	0.47
RWT, cm	0.48 ± 0.11	0.48 ± 0.12	0.84
LVMI, g/m^2	133 ± 39	146 ± 26	0.11

Data expressed as mean ± SD.

CAPD: Continuous ambulatory peritoneal dialysis, BMI: Body mass index, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, , LVEDD: Left ventricular end-distolic diameter, IVST: Interventricular septal thickness, PWT: Posterior wall thickness, RWT: Relative wall thickness, LVMI: Left ventricular mass index.

was 75 ± 3 months (95% CI of 69 to 81 months) and it was 76 ± 3 months (95% CI of 69 to 82 months) for the patients with eccentric LV hypertrophy. The probability of CV event-free survival for the patients with LV hypertrophy was %71. Prognostic factors for disease free survival by univariate analysis are shown in Table 4. After adjusting for confounders in the Cox regression model, as illustrated in Figure 1a, CAPD patients with LV hypertrophy had shorter time to CV event compared to the patients without LV hypertrophy (Hazard ratio (HR)= 19.4, 95% CI for HR= 1.3-295, p= 0.033). Patients with eccentric (HR of 20.2) and concentric LV hypertrophy (HR of 18.7) had significantly shorter time to CV events compared to the patients with normal geometry and concentric remodeling (p= 0.042 for both) (Table 5 and Figure 1b). In addition, CAPD patients with diabetes mellitus, hypertension and history of coronary artery disease had shorter time to CV events regardless of the geometry type (Table 5).

DISCUSSION

Left ventricular hypertrophy is a common finding and strong predictor of cardiovascular mortality and morbidity in dialysis patients. Both hemodynamic and non-hemodynamic factors are responsible for the development of LVH. Left ventricular hypertrophy may result from volume and pressure overload.[22] Hypertension is highly prevalent in dialysis patients and overactivity of the sympathetic system is a consistent feature.[23,24] Hypervolemia and hypercirculatory state together with anemia may account for increased LV mass even in normotensive dialysis patients. Anemia, arteriovenous fistula, hyperparathyroidism,increased arterial stiffness together with high peripheral resistance are accepted as potential factors influencing development of cardiac hypertrophy.[24-27] Moreover, the importance of preserving residual renal function (RRF) on LVH and cardiovascular outcome was emphasized by Wang et. al.,[1] LVH was believed to have an inverse relationhip with RRF. Inflammation has also been closely linked with arterial stiffening, LVH and LV dilatation, and systolic dysfunction in CAPD patients.[3]

The balance between the two fundamental hemodynamic stimuli (pressure and volume) determines the predominant type of geometric development of the left ventricle.[11,28] In states of pressure overload, the resultant increase in wall tension is offset by an increase in LV wall thickness with normal cavity volume (concentric hypertrophy), whereas LV dilation is commonly observed in states of volume overload and many patients with LV dilation have increased LV mass (eccentric hypertrophy).[13,29]

TABLE 4: Prognostic factors for disease free survival by univariate analysis.

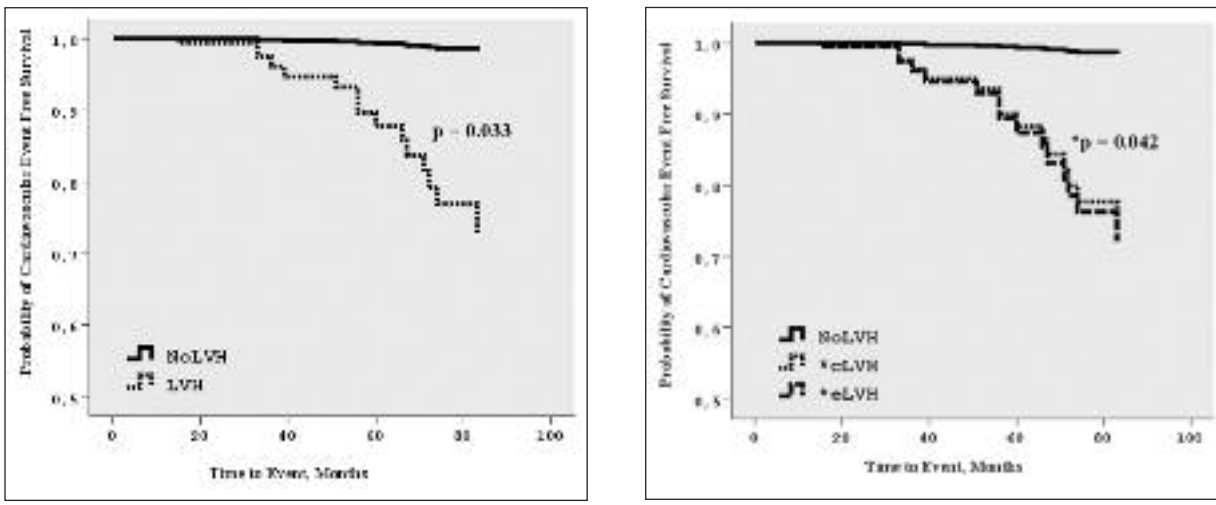
	Number of patients	Number of events	Mean DFS (months)	95% confidence interval	Log rank	p value
Gender						
Female	29	5	80±2	76-84	1.75	0.186
Male	36	11	74±3	68-80		
Age						
<45	36	4	82±1	80-84	8.27	0.004
>45	29	12	70±4	63-78		
BMI(kg/m^2)						
<25	42	9	79±2	76-82	0.88	0.645
25-30	17	5	70±6	58-83		
>30	6	2	77±2	69-86		
SBP(mmHg)						
≤140	17	1	80±4	72-88	3.43	0.064
>140	48	14	75±2	71-80		
DBP(mmHg)						
<90	20	4	75±5	66-84	1.05	0.592
90-100	22	4	80±2	77-84		
>100	23	7	75±3	69-81		
Hypertension						
No	7	1	79±4	71-88	0.41	0.520
Yes	58	15	76±2	72-80		
LVH						
No	20	2	79±3	73-86	2.96	0.085
Yes	45	14	75±2	71-80		
LVH type						
cLVH	24	7	75±3	69-81	3.02	0.220
eLVH	21	7	76±3	69-82		
normal	20	2	79±3	73-86		
Diabetes						
No	60	13	79±2	76-82	7.05	0.008
Yes	5	3	54±12	30-77		
Hyperlipidemia						
No	27	7	76±3	69-82	0.06	0.799
Yes	38	9	77±2	73-82		
CAD						
No	58	11	79±2	76-82	13.10	0.0001
Yes	7	5	58±9	40-76		
Smoking						
no	36	9	78±2	73-83	0.0005	0.982
yes	29	7	75±3	69-82		
Hb(g/dl)						
<10	26	6	78±3	72-84	0.80	0.669
10-12	28	8	74±3	67-80		
<12	11	2	81±2	78-84		

DFS: Disease free survival, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, LVH: Left ventricular hypertrophy, CAD: Coronary artery disease, Hb: Hemoglobin.

TABLE 5: Significant variables in Cox regression

Variables	Hazard Ratio (HR)	95% CI for HR Lower	Upper	p value
Diabetes mellitus	27.9	3.2	241	0.003
Hypertension	58.0	1.3	259	0.036
Eccentric LV hypertrophy*	20.2	1.1	368	0.042
Concentric LV hypertrophy*	18.7	1.1	316	0.042
Coronary artery disease	12.8	1.1	155	0.044

*The reference category for the group including eccentric and concentric LV hypertrophy is normal geometry and concentric remodeling.
Cox regression model included age, gender, body mass index, systolic and diastolic blood pressures, hemoglobin, LDL-cholesterol, LV mass index, smoking status and the presence of hypertension, coronary artery disease, diabetes mellitus, hyperlipidemia and peripheral artery disease in addition to LV geometry types.
LV: Left ventricular.



a) b)

FIGURE 1: Cardiovascular event free survival curves for continuous ambulatory peritoneal dialysis patients with respect to LV hypertrophy (a) and LV hypertrophy patterns (b).
*Compared with the reference category (normal geometry and concentric remodeling).
LVH: left ventricular hypertrophy, cLVH: concentric lef ventricular hypertrophy, eLVH: eccentric left ventricular hypertrophy.

In a previous study, we examined the factors which contributed to the development of left ventricular hypertrophy and geometric remodeling in CAPD patients.[30] Systolic blood pressure was significantly higher in the cLVH group compared the normal geometry group. Daily total ultrafiltration volume was higher in the eLVH group than in the cLVH group. The eLVH group had significantly lower hemoglobin levels, and higher dialysate-to-plasma creatinine ratio than the normal geometry group. Hypervolemia, identified by echocardiographic measurement of inferior vena cava index and collapsibility index, and anemia were found to contribute independently to LV geometry.[30]

Progressive cardiac enlargement, particularly left ventricular dilation with compensatory hypertrophy, continues after starting dialysis therapy.[22] Most of the additional cardiac enlargement occurs in the first year of dialysis therapy and intervention beyond one year may be relatively ineffective.[22] Thus, screening every patient for LVH at the beginning of dialysis with echocardiography may be relevant for risk assessment.

Enia et al., reported that the prevalence of the concentric and eccentric form of LVH was similar in CAPD patients.[14] In a study by Foley et al., baseline echocardiographic classification, based on LV mass and cavity volume, was the strongest predictor of late mortality.[13] In the population based Framingham Study, knowledge of LV geometry provided little prognostic infomation beyond that available from LV mass and traditional risk factors.[15] In other studies, non-dialysis patients with cLVH were shown to have the poorest CV prognosis.[16,17] The impact of different patterns of hypertrophy on cardiovascular outcome of dialysis patients has not been adequately studied. Zoccali et al., stated that the geometric pattern of LVH was a significant correlate of systolic dysfunction and this alteration evolved faster in dialysis patients with eccentric LVH than in those with concentric LVH or normal LV mass.[31] Eccentric LVH is characterized by LV dilation and by a reduction in myocardial contractility.[11] Compromised systolic function is another predictor of CV complications and there is faster progression of systolic dysfunction in dialysis patients with eLVH.[32]

In this study, the prevalence of LVH was 69%, of these 47% had eccentric and 53% had concentric type of LV hypertrophy. In a total of 16 patients with CV events, seven had eLVH, seven had cLVH and only two had no LVH. To assess the impact of hypertrophy patterns on CV outcome, we used Kaplan-Meier method; mean CV event free survival time in the patients with concentric LV hypertrophy was 75 ± 3 months (95% CI of 69 to 81 months) and it was 76 ± 3 months (95% CI of 69 to 82 months) for the patients with eccentric LV hypertrophy. Patients with LVH had poorer outcome compared to patients without, and there was no difference between the two hypertrophy types. CAPD patients with eccentric and concentric LV hypertrophy had a shorter time for a CV event compared to the patients without LV hypertrophy . In a study by Ohashi et al., it was shown that diabetic nephropathy and severe LVH were the two main predictors of CV events.[32] In our study, hypertension, diabetes mellitus and previously documented coronary artery disease were independent variables which were found to be significantly in affecting event free survival by multivariate analysis. Continuous ambulatory peritoneal dialysis patients with above mentioned diseases had shorter time to CV events regardless of the LV geometry type.

This study was done in one center and screening covered all of the registered CAPD patients. Despite the fact that number of patients included were rather small for survival analysis, we believe that our findings may provide additive information regarding the role of LV geometric types as a marker of CV prognosis in CAPD patients. Statistical significance was low due to small number of patients and events, and we believe that a multicenter trial with higher number of subjects would yield more significant results.

CONCLUSIONS

LVH is highly prevalent in CAPD patients and the prevalences of eLVH and cLVH are similar. Left ventricular hypertrophy is an important prognostic marker for poorer CV outcome irrespective of its pattern. Both eccentric and concentric LV hypertrophy are associated with shorter CV event free survival compared to normal geometry and concentric remodeling. The presence of coronary artery disease, hypertension and diabetes mellitus are other independent risk factors that identify patients at higher risk for CV events. Echocardiography is a useful tool in screening CAPD patients for LV mass and geometry. Each patient starting dialysis therapy should be investigated for LVH and patients without LVH at baseline should be screened regularly. Defining the type of LVH may not be necessary. All factors that contribute to LVH must be taken into account, utmost care should be taken of good blood pressue control, correction of hypervolemia and anemia, control of hyperparathyroidism and preservation of RRF . Cardiovascular disease is the major cause of death in peritoneal dialysis patients and those with documented LVH need strict medical assistance and therapy.

REFERENCES

1. Wang AY. The "heart" of peritoneal dialysis. Perit Dial Int 2007;27(Suppl 2):S228-32.
2. Fenton SS, Schaubel DE, Desmeules M, Morrison HI, Mao Y, Copleston P, et al. Hemodialysis versus peritoneal dialysis: a comparison of adjusted mortality rates. Am J Kidney Dis 1997;30(3):334-42.
3. Wang AY, Wang M, Woo J, Lam CW, Lui SF, Li PK, et al. Inflammation, residual kidney function, and cardiac hypertrophy are interrelated and combine adversely to enhance mortality and cardiovascular death risk of peritoneal dialysis patients. J Am Soc Nephrol 2004;15(8):2186-94.
4. Levin A, Singer J, Thompson CR, Ross H, Lewis M. Prevalent left ventricular hypertrophy in the predialysis population: identifying opportunities for intervention. Am J Kidney Dis 1996;27(3):347-54.
5. Tucker B, Fabbian F, Giles M, Thuraisingham RC, Raine AE, Baker LR. Left ventricular hypertrophy and ambulatory blood pressure monitoring in chronic renal failure. Nephrol Dial Transplant 1997;12(4):724-8.
6. Foley RN, Parfrey PS, Harnett JD, Kent GM, Martin CJ, Murray DC, et al. Clinical and echocardiographic disease in patients starting end-stage renal disease therapy. Kidney Int 1995;47(1):186-92.
7. Harnett JD, Parfrey PS, Griffiths SM, Gault MH, Barre P, Guttman RD. Left ventricular hypertrophy in end-stage renal disease. Nephron 1998;48(2):107-15.
8. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med 1990; 322(22): 1561-6.
9. Parfrey PS, Harnett JD, Griffiths SM, Taylor R, Hand J, King A, et al. The clinical course of left ventricular hypertrophy in dialysis patients. Nephron 1990;55(2):114-20.
10. Silberberg JS, Barre PE, Prichard SS, Sniderman AD. Impact of left ventricular hypertrophy on survival in end-stage renal disease. Kidney Int 1989;36(2):286-90.
11. de Simone G. Left ventricular geometry and hypotension in end-stage renal disease: a mechanical perspective. J Am Soc Nephrol 2003;14(10):2421-7.
12. Ganau A, Devereux RB, Roman MJ, de Simone G, Pickering TG, Saba PS, et al. Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension. J Am Coll Cardiol 1992;19(7):1550-8.
13. Foley RN, Parfrey PS, Harnett JD, Kent GM, Murray DC, Barré PE. The prognostic importance of left ventricular geometry in uremic cardiomyopathy. J Am Soc Nephrol 1995; 5 (12):2024-31.
14. Enia G, Mallamaci F, Benedetto FA, Panuccio V, Parlongo S, Cutrupi S, et al. Long-term CAPD patients are volume expanded and display more severe left ventricular hypertrophy than haemodialysis patients. Nephrol Dial Transplant 2001;16(7):1459-64.
15. Krumholz HM, Larson M, Levy D. Prognosis of left ventricular geometric patterns in the Framingham Heart Study. J Am Coll Cardiol 1995;25(4):879-84.
16. Ghali JK, Liao Y, Cooper RS. Influence of left ventricular geometric patterns on prognosis in patients with or without coronary artery disease. J Am Coll Cardiol 1998;31(7):1635-40.
17. Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. Ann Intern Med 1991;114(5):345-52.
18. Sahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. Circulation 1978;58(6):1072-83.
19. Devereux RB, Alonso DR, Lutas EM, Gottlieb GJ, Campo E, Sachs I, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol 1986;57(6):450-8.
20. Levy D, Savage DD, Garrison RJ, Anderson KM, Kannel WB, Castelli WP. Echocardiographic criteria for left ventricular hypertrophy: the Framingham Heart Study. Am J Cardiol 1987;59(9):956-60.
21. Ganau A, Devereux RB, Roman MJ, de Simone G, Pickering TG, Saba PS, et al. Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension J Am Coll Cardiol 1992;19(7):1550-8.
22. Foley RN, Parfrey PS, Kent GM, Harnett JD, Murray DC, Barre PE. Long-term evolution of cardiomyopathy in dialysis patients. Kidney Int 1998;54(5):1720-5.
23. Converse RL Jr, Jacobsen TN, Toto RD, Jost CM, Cosentino F, Fouad-Tarazi F, et al. Sympathetic overactivity in patients with chronic renal failure. N Engl J Med 1992;327 (27): 1912-8.
24. Amann K, Mandelbaum A, Schwarz U, Ritz E. Hypertension and left ventricular hypertrophy in the CAPD patient. Kidney Int Suppl 1996; 56:S37-40.
25. Silberberg J, Racine N, Barre P, Sniderman AD. Regression of left ventricular hypertrophy in dialysis patients following correction of anemia with recombinant human erythropoietin. Can J Cardiol 1990;6(1):1-4.
26. London GM, De Vernejoul MC, Fabiani F, Marchais SJ, Guerin AP, Metivier F, et al. Secondary hyperparathyroidism and cardiac hypertrophy in hemodialysis patients. Kidney Int 1987;32(6):900-7.
27. Marchais SJ, Guerin AP, Pannier BM, Levy BI, Safar ME, London GM. Wave reflections and cardiac hypertrophy in chronic uremia. Influence of body size. Hypertension 1993;22(6): 876-83.
28. de Simone G. Left ventricular geometry and hypotension in end-stage renal disease: a mechanical perspective. J Am Soc Nephrol 2003; 14(10):2421-7.
29. Huwez FU, Pringle SD, Macfarlane PW. A new classification of left ventricular geometry in patients with cardiac disease based on M-mode echocardiography. Am J Cardiol 1992; 70(6):681-8.
30. Toprak A, Koc M, Tezcan H, Ozener IC, Akoglu E, Oktay A. Inferior vena cava diameter determines left ventricular geometry in continuous ambulatory peritoneal dialysis patients: an echocardiographic study. Nephrol Dial Transplant 2003;18(10):2128-33.
31. Zoccali C, Benedetto FA, Tripepi G, Mallamaci F, Rapisarda F, Seminara G, et al. Left ventricular systolic function monitoring in asymptomatic dialysis patients: a prospective cohort study. J Am Soc Nephrol 2006;17(5):1460-5.
32. Ohashi H, Oda H, Ohno M, Sakata S. Predictors of survival in continuous ambulatory peritoneal dialysis patients: the importance of left ventricular hypertrophy and diabetic nephropathy. Adv Perit Dial 1999;15:87-90.