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Inferior vena cava diameter determines left ventricular geometry in continuous ambulatory peritoneal dialysis patients: an echocardiographic study

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Abstract

Background. Changes in left ventricular (LV) geometry are frequent in patients with continuous ambulatory peritoneal dialysis (CAPD). Geometric adaptation of LV to various stimuli was reported to have adverse prognosis. This study aimed to identify independent risk factors, which contribute to the development of LV geometric remodelling in CAPD patients.

Methods. The left ventricles of 69 CAPD patients were classified echocardiographically into four different geometric patterns on the basis of LV mass and relative wall thickness. With respect to volume factor, we measured inferior vena cava (IVC) diameter and its decrease on deep inspiration [collapsibility index (CI)] by echocardiography. We modelled a stepwise multiple regression analysis to determine the predictors of LV geometry.

Results. All four geometric models of LV were identified in our group of 69 CAPD patients. Eccentric left ventricular hypertrophy (eLVH) was observed in 32 (46%), concentric LVH (cLVH) in 19 (28%), normal geometry (NG) in 10 (14%) and concentric remodelling (CR) in eight (12%) CAPD patients. Mean IVC index of the eLVH group ($10.72 \pm 2.19\,mm/m^2$) was significantly higher than corresponding indexes of NG ($7.90 \pm 1.54\,mm/m^2$), CR ($8.51 \pm 1.28\,mm/m^2$) and cLVH ($8.04 \pm 2.00\,mm/m^2$) groups ($P < 0.001$ for each comparisons). The eLVH group also had significantly lower mean CI value (0.48 ± 0.11) than CR (0.58 ± 0.09) and cLVH (0.57 ± 0.07) groups (ANOVA $P = 0.008$). Stepwise multiple regression analysis revealed that IVC index, CI and haemoglobin were the independent predictors of LV geometric stratification ($R^2 = 0.36$, $P < 0.001$).

Conclusion. Hypervolaemia, identified by IVC index and CI, and anaemia contribute independently to LV geometry in CAPD patients. Echocardiography as a non-invasive tool is not only useful to determine LV geometry, but also to assess the volume status of CAPD patients.

Keywords: continuous ambulatory peritoneal dialysis; inferior vena cava; ventricular remodelling

Introduction

Death rates from cardiac disease are between 10 and 20 times higher in dialysis patients than in the general population [1]. Cardiac mortality and morbidity in dialysis patients usually result from cardiomyopathy and/or ischaemic heart disease. Cardiomyopathy may be manifest as concentric left ventricular hypertrophy (cLVH), left ventricular (LV) dilatation with compensatory hypertrophy [eccentric left ventricular hypertrophy (eLVH)], or systolic dysfunction. Cardiomyopathy is frequently observed in dialysis patients, and together with indices of LV geometry, are independent adverse predictors of mortality. Left ventricular mass (LVM) is prognostically dominant in cLVH, whereas cavity size predicts outcome in patients with LV dilatation [2].

Dialysis patients have many risk factors for both volume and pressure overload. In end-stage renal disease (ESRD) treated by dialysis, fluid overload and arterial hypertension often contribute to a combination of eccentric and concentric hypertrophy, which may be influenced both by inadequate volume and blood pressure (BP) control [3]. LV dilatation in dialysis

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patients is commonly observed in states of chronic volume overload, whereas pressure overload leads primarily to an increase in LVM. According to Laplace's law, wall stress is directly proportional to pressure and LV internal radius, and inversely proportional to LV wall thickness. Volume overload results in an enlargement of ventricular chamber with increased wall thickness to counterbalance increased radius, so that radius/wall thickness ratio remains within normal limits. In states of pressure overload, the increase in wall tension is offset by a disproportionate increase in LV wall thickness at normal chamber radius, reflected on echocardiography as cLVH.

There is ample evidence that a significant proportion of continuous ambulatory peritoneal dialysis (CAPD) patients demonstrate latent overhydration, because exact estimation of true dry body weight is difficult on clinical grounds alone. Measurement of the diameter of inferior vena cava (IVC) and its decrease on deep inspiration [collapsibility index (CI)] by echocardiography allows an accurate assessment of volume status in dialysis patients [4]. Echocardiography also allows cardiac structure and function to be assessed non-invasively. LV geometry may be classified into four groups on the basis of LVM and relative wall thickness (RWT): normal geometry (normal mass and normal RWT) (NG), concentric remodelling (normal mass and increased RWT) (CR), cLVH (increased mass and increased RWT) and eLVH (increased mass and normal RWT).

No studies have been done to directly relate LV geometry to factors which may predispose to LV geometric remodelling in patients with CAPD. Therefore, a cross-sectional study with 69 CAPD patients was undertaken in an attempt to answer the following questions: (i) what was the prevalence of geometric models among CAPD patients? (ii) What were the most important factors independently related to the development of geometric models in CAPD patients? We measured IVC diameter as a marker of volume status.

Patients and methods

Study design and patients

Ninety-one patients, who were on CAPD for at least 12 months at Marmara University Hospital, were screened for this cross-sectional study. Seventeen patients with previous myocardial infarction ($n=4$), coronary artery bypass surgery ($n=1$), history of congestive heart failure ($n=5$), severe valvular heart disease ($n=2$), pulmonary disease ($n=2$) and atrial fibrillation ($n=3$) were excluded. Additionally, five patients were not enrolled because of technical difficulties during echocardiographic recording. Sixty-nine CAPD patients, of whom 32 were female and 37 were male, constituted the final study group. Informed written consent was obtained from each participant. The mean age of the study group was 46 ± 15 years, which ranged from 17 to 79 years. The aetiologies of chronic renal failure of the final 69 CAPD patients were diabetes mellitus in eight, chronic glomerulonephritis in 23, tubulointerstitial nephritis in seven, hypertensive nephrosclerosis in 13, miscellaneous in four and unknown in 14 patients.

Echocardiography of the left ventricle

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. 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 [5]. Left ventricular mass (LVM) was calculated using the thick-wall prolate-ellipsoidal model with correction based on a necropsy validation study by Devereux *et al.* [6]: $0.832 \times [(\mathrm{LVID}+\mathrm{IVST}+\mathrm{PWT})^3-\mathrm{LVID}^3]+0.6$. LVM was considered as an unadjusted variable and normalized for body surface area (BSA) as left ventricular mass index (LVMI). BSA was calculated by using the formula: $0.007184 \times [\text{weight (kg)}]^{0.425} \times [\text{height (cm)}]^{0.725}$. LVMI partition values of 131 g/m^2 for men and 100 g/m^2 for women were used as the upper gender-specific normal [7].

RWT of the LV was calculated as $2 \times (\mathrm{PWT} / \mathrm{LVID})$. LV geometric patterns were defined according to the method of Ganau *et al.* [8]. A partition value of 0.44 for RWT was used for both men and women to represent the 99th percentile value in normal subjects. NG was present when LVMI and RWT were normal, normal LVMI and increased RWT was classified as CR, increased LVMI but normal RWT identified eLVH, and increases in both variables identified cLVH.

Measurement of inferior vena cava diameter

Measurements of IVC diameter and LV were done on different days within a week by different echocardiographers, who were blind to the previous echocardiographic data of the patient, provided that the body weight of the patient was equal. Echocardiographic examination of IVC was performed in mid-day after complete emptying of the peritoneal dialysate and in a supine position after 10 min of rest. The transducer was placed in the subxiphoidal region and long and short axis views of the IVC were obtained just below the diaphragm in the hepatic segment. An M-mode echocardiogram with simultaneous electrocardiographic monitoring was recorded. IVC diameter was measured before the P-wave on the ECG to avoid interference with a-wave and v-wave on the venous pressure curve. IVC diameter was measured during expiration and maximal inspiration, avoiding valsalva-like manoeuvres. This method was described previously [4]. IVC diameter was indexed for BSA as IVC index. CI was defined as: (maximal diameter on expiration – minimal diameter on deep inspiration) / maximal diameter on expiration. All vasoactive medications were discontinued 16 h before the investigations were done. The intraobserver variability was 2.6% for IVC diameter and 1% for CI.

Laboratory measurements

Whole blood counts and blood chemistry were analysed by standard laboratory procedures. Intact parathyroid hormone

(PTH) levels were determined using radioimmunoassay (Sigma-Aldrich Laboratories, The Woodlands, TX, USA). Kt/V_{urea} was calculated from total loss of urea nitrogen in the spent dialysate using the Watson equation [9]. Peritoneal equilibration test (PET) was performed as previously described [10]. Serum albumin concentration was measured by the bromocresol green method.

Statistical analysis

All calculations were done using SPSS and GraphPad Prism computer programs. Data were expressed as mean ± SD. Comparisons between clinical, laboratory and echocardiographic characteristics of CAPD patients with different LV geometric patterns were done using Kruskal–Wallis one-way ANOVA. *Post hoc* comparisons between pairs of means were made by using Dunns with downward adjustment of the alpha level to compensate for multiple comparisons. Stepwise multiple regression analysis was performed to define the predictors LV geometric patterns. The LV geometric adaptation was included into the multiple regression equation by assigning numerals to represent geometric models. The LV geometric changes were dummy coded as 0 for concentric remodelling (CR and cLVH) and 1 for eccentric remodelling (eLVH) according to RWT, with the exclusion of NG. A two-tailed *P*-value of < 0.05 was considered significant.

Results

All four geometric models of LV were identified in our group of 69 CAPD patients. Most frequently, 32 (46%) demonstrated eLVH. Concentric LVH was observed in 19 (28%), NG in 10 (14%) and CR in eight (12%) CAPD patients.

Clinical and laboratory characteristics of the patients with four different LV geometric patterns were presented in Table 1. Age, gender, BMI, duration of CAPD, number of antihypertensive medications, serum albumin level, total and renal weekly Kt/V_{urea} did not differ significantly between groups. On the other hand, systolic BP (SBP), daily total ultrafiltration (UF), haemoglobin, haematocrit and dialysate-to-plasma ratio of creatinine (Cr D/P) were different between the groups. Mean SBP was significantly higher in the cLVH group (165 ± 28 mmHg) than in the NG group (141 ± 20 mmHg; $P = 0.02$). Mean daily total UF volume was higher in the eLVH group (1570 ± 437 ml) than in the cLVH group (1215 ± 376 ml; $P = 0.02$). The eLVH group had significantly lower mean haemoglobin (10.2 ± 1.6 g/dl) and haematocrit levels (30.5 ± 4.1%) compared with the NG group (11.7 ± 1.2 g/dl and 34.4 ± 3.0%, respectively; $P = 0.01$ for both). Mean Cr D/P was higher in the eLVH group than in the NG group (0.66 ± 0.11 *vs* 0.55 ± 0.08; $P = 0.02$).

Echocardiographic data of the groups were presented in Table 2. Mean IVC index of the eLVH group (10.72 ± 2.19 mm/m^2) was significantly higher than corresponding indexes of NG (7.90 ± 1.54 mm/m^2), CR (8.51 ± 1.28 mm/m^2) and cLVH (8.04 ± 2.00 mm/m^2) groups ($P < 0.001$ for each comparisons). The eLVH group also had a significantly lower mean CI value (0.48 ± 0.11) than CR (0.58 ± 0.09) and cLVH (0.57 ± 0.07) groups (ANOVA $P = 0.008$).

Table 1. Comparisons of clinical and laboratory characteristics of CAPD patients with four different LV geometric patterns

	NG ($n = 10$)	CR ($n = 8$)	cLVH ($n = 19$)	eLVH ($n = 32$)	*P*
Age (years)	44 ± 8	51 ± 18	47 ± 15	46 ± 16	NS
Gender (female/male) (*n*)	5/5	3/5	12/7	12/20	NS
Body mass index (kg/m^2)	24 ± 2	24 ± 3	25 ± 3	24 ± 5	NS
Duration of CAPD (months)	26 ± 12	26 ± 18	30 ± 14	22 ± 14	NS
SBP (mmHg)	141 ± 20	161 ± 29	165 ± 28[a]	148 ± 21	0.02
DBP (mmHg)	88 ± 17	101 ± 15	102 ± 16	93 ± 14	NS
Antihypertensive medications (*n*)	0.5 ± 0.7	0.8 ± 0.7	0.9 ± 0.8	0.9 ± 0.7	NS
Daily urine volume (ml)	537 ± 503	370 ± 444	321 ± 378	572 ± 590	N.S.
Daily total ultrafiltration (ml)	1537 ± 611	1145 ± 560	1215 ± 376	1570 ± 437[b]	0.02
Weekly 3.86% dextrose (l)	3.6 ± 4.1	3.2 ± 2.2	4.8 ± 4.6	5.1 ± 6.7	NS
Haemoglobin (g/dl)	11.7 ± 1.2	11.6 ± 1.2	10.4 ± 1.9	10.2 ± 1.6[c]	0.01
Haematocrit (%)	34.4 ± 3.0	35.0 ± 4.3	30.8 ± 5.9	30.5 ± 4.1[c]	0.01
Serum calcium (mg/dl)	9.3 ± 0.9	9.2 ± 0.7	9.5 ± 0.9	9.2 ± 0.9	NS
Serum phosphorus (mg/dl)	5.0 ± 1.2	4.9 ± 1.3	5.4 ± 1.1	5.3 ± 1.5	NS
Serum albumin (g/dl)	4.1 ± 0.5	3.9 ± 0.4	3.9 ± 0.5	3.7 ± 0.5	NS
Parathormone (pg/ml)	447 ± 556	171 ± 212	266 ± 240	286 ± 338	NS
Patients on rHuEpo (%)	80	75	74	84	NS
Dose of rHuEpo (U/week)	4625 ± 2200	4333 ± 1966	6500 ± 3525	6037 ± 3216	NS
Creatinine clearance (l/week/1.73 m^2)	67 ± 23	63 ± 19	56 ± 17	68 ± 31	NS
Kt/V_{urea} (weeks)	2.3 ± 0.4	2.1 ± 0.4	2.0 ± 0.3	2.2 ± 0.5	NS
Renal Kt/V_{urea} (weeks)	0.42 ± 0.44	0.55 ± 0.73	0.19 ± 0.25	0.44 ± 0.55	NS
PET drainage volume (ml)	2583 ± 185	2452 ± 149	2540 ± 141	2467 ± 202	NS
Cr D/P (PET)	0.55 ± 0.08	0.62 ± 0.09	0.60 ± 0.10	0.66 ± 0.11[c]	0.02
Peritoneal transport type by PET (L/LA/H/HA) (*n*)	2/6/0/2	1/4/0/3	3/10/0/6	2/14/5/11	NS

Kt/V_{urea}, urea clearance × time normalized by total body water, the volume of distribution of urea; L, low; LA, low-average; H, high; HA, high-average transporters; NS, not significant.
Post hoc comparisons: [a]cLVH *vs* NG; [b]eLVH *vs* cLVH; [c]eLVH *vs* NG.

Table 2. Echocardiographic comparisons between four different LV geometric patterns in CAPD patients

	NG ($n=10$)	CR ($n=8$)	cLVH ($n=19$)	eLVH ($n=32$)	P
LVID at end-diastole (cm)	4.89 ± 0.49	4.37 ± 0.52	4.75 ± 0.68	5.48 ± 0.48[a]	<0.001
IVST at end-diastole (cm)	1.03 ± 0.20	1.14 ± 0.18	1.44 ± 0.19[b]	1.23 ± 0.18	<0.001
PWT at end-diastole (cm)	0.90 ± 0.11	1.10 ± 0.11	1.30 ± 0.16[b]	1.11 ± 0.10	<0.001
RWT (cm)	0.37 ± 0.05[c]	0.51 ± 0.07	0.56 ± 0.10	0.41 ± 0.03	<0.001
LVMI (g/m^2)	97 ± 15	101 ± 19	158 ± 40[d]	149 ± 27[d]	<0.001
IVC diameter (mm)	13.6 ± 2.8	14.6 ± 3.2	13.3 ± 2.5	19.0 ± 3.8[a]	<0.001
IVC index (mm/m^2)	7.90 ± 1.54	8.51 ± 1.28	8.04 ± 2.00	10.72 ± 2.19[a]	<0.001
CI	0.55 ± 0.11	0.58 ± 0.09	0.57 ± 0.07	0.48 ± 0.11[e]	0.008

Post hoc comparisons: [a]eLVH *vs* NG, CR and cLVH; [b]cLVH *vs* NG, CR and eLVH; [c]NG *vs* CR and cLVH; [d]cLVH and eLVH *vs* NG and CR; [e]eLVH *vs* CR and cLVH.

Table 3. Stepwise multiple regression analysis for the determination of predictors of the LV geometric stratification in CAPD patients

Independent variables	β (coefficient)	t-test value	P
IVC index	0.32	3.08	0.003
Haemoglobin (g/dl)	−0.28	−2.69	0.009
CI	−0.22	−2.14	0.036

$R^2=0.36$, $P<0.001$.
Out of the model: systolic blood pressure ($P=0.91$); daily total ultrafiltration volume ($P=0.44$); haematocrit ($P=0.77$) and Cr D/P ($P=0.06$).

We modelled a stepwise multiple regression analysis to define the independent determinants of LV geometry. SBP, daily total UF volume, haemoglobin, haematocrit, Cr D/P, IVC index and CI were included into the model. IVC index, haemoglobin and CI were found to be independent predictors of LV geometry ($R^2=0.36$; $P<0.001$; Table 3).

Discussion

The present study provides observational evidence that: (i) CAPD patients had a prevalence of abnormal LV geometry of 86% and LVH of 74%; (ii) hypervolaemia, assessed by IVC index and CI, and anaemia were independent factors which contribute to LV geometric stratification in CAPD patients. These risk factors were derived from multivariate analysis and are independent of each other and of other determinants of cardiac disease, such as SBP, daily total UF and Cr D/P. Hypervolaemia and anaemia appear to be the key independent predictors of cardiac structural abnormalities in this study.

CAPD patients have many risk factors for both volume and pressure overload. Volume overload and hypertension often contribute to a combination of eccentric and cLVH, which may be influenced both by inadequate volume control and anaemia. The heterogeneity of the LV geometry in CAPD patients was a consequence of volume factors and anaemia according to our study. But several other potential factors, such as genetic influences, neurohormonal activation and growth factors were not tested.

The higher prevalence of eLVH in the present and as well as in previous studies is caused by volume factors and anaemia. Echocardiographic examination of IVC is a simple, quick, and non-invasive method for the assessment of intravascular volume status. Cheriex *et al.* [4] showed that both IVC index and CI correlated well with central venous pressure, and IVC index significantly correlated with total blood volume. Linear regression to right atrial pressure defined overhydration (mean right atrial pressure >7 mmHg) as a caval diameter >11.5 mm/m^2 or a CI as <40%. According to this definition, 50% of our CAPD patients with eLVH were actually hypervolaemic. There are other indicators of volume status in dialysis patients. Atrial natriuretic peptide (ANP) and its second messenger, cyclic guanosine monophosphate (cGMP), are biochemical markers with high sensitivity for the volume overloaded state [11]. On the other hand, the meaning of low levels of the markers and the specificity for detecting the underhydrated state is unknown. Biochemical marker levels are influenced by cardiac or valvular dysfunction. Measurement of electric conductivity (bioimpedance) is another non-invasive tool that has proven to be useful for detecting and managing both the over- and underhydrated state in haemodialysis [12]. Unlike biochemical markers and IVC, bioimpedance can additionally determine interstitial and intracellular fluid status.

Previous reports from Framingham have demonstrated that LVM has a continuous and graded association with cardiovascular outcomes [13]. The prognostic information from the geometric classification derived largely from differences in LVM between the groups. In the Framingham population-based sample of subjects without cardiovascular disease, knowledge of LV geometry provided little prognostic information beyond that available from LVM and traditional cardiovascular risk factors [14]. On the other hand, standard classification, based merely on combinations of LVMI and RWT, may be inefficient at quantifying risk in populations where LV dilatation is common. The standard geometric classification was shown to be independently associated with cardiac death in chronic dialysis patients free of symptomatic heart disease, with the adjusted relative risk of 1.26 [15]. According to an echocardiographic prognostic

classification system proposed by Foley *et al.* [15], in dialysis patients with LV dilatation and normal systolic function, high cavity volume ($>120\,ml/m^2$) was independently associated with late mortality (>2 years after starting dialysis therapy), with the adjusted relative risk being 17.14. LVMI was of no prognostic significance in this group. LVMI was associated with late mortality only in patients with normal cavity volume and high LVM in the mentioned study.

Anaemia is a frequent finding in ESRD patients and the influence of anaemia on LV structure has been demonstrated previously by a variety of studies. Anaemia contributed independently to LV geometric stratification in our study. Low haemoglobin was also shown to be an independent risk factor for LV dilatation and LV hypertrophy, and predicts death in patients with ESRD [16,17]. There was a tendency toward more severe anaemia in the group who developed eLVH in our study. It has been reported that each 1 g/dl decrease in the mean haemoglobin level was independently associated with a 42% increased likelihood of having LV dilatation on a second echocardiogram in a prospective cohort of 432 ESRD patients [16]. In addition, each 1 g/dl decrease in the mean haemoglobin level was independently associated with cardiac failure and death (relative risk, 1.14). Amelioration of anaemia with erythropoietin has been reported to reduce cardiac size and wall thickness, and improve cardiac function in chronic haemodialysis patients [18]. But normalization of haemoglobin did not induce regression of overt LV dilatation or cLVH in haemodialysis patients in the prospective study conducted by Foley *et al.* [19].

The cross-sectional design and low sample size are important limitations of this study. Another limitation of the present study is that the renin-angiotensin-aldosterone system (RAAS) and sympathetic overactivation were not evaluated for their effects on LV geometry. A previous study reported high plasma renin activity and plasma aldosterone concentrations in hypertensive patients with cLVH [20]. However, the impact of neurohormonal activation inherent in uraemia and the role of messenger systems, growth factors and cytokines in LV structural and functional alterations in chronic renal failure remain to be evaluated. There are also difficulties with the use of M-mode echocardiography in the measurement of IVC and CI, because measurements may be inaccurate as a result of the beam angle. The inter- and intraobserver variability for IVC was $<5\%$ and $<2.5\%$, respectively, and for the CI $<10\%$ and $<2.5\%$, respectively, in the basic work done by Cheriex *et al.* [4]. The intraobserver variability was small and it was 2.6% for IVC and 1% for CI in our study. Finally, measurements of IVC diameter and LV were done on different days in our study. There may be a major concern that volume status and LVID measurements could differ when measured on different days. We assumed a nearly identical haemodynamic situation, when measurements were done within a week and the body weight of the patient was the same.

In conclusion, volume factors, such as IVC index and CI, and anaemia contribute to LV geometric stratification in CAPD patients. Correction of hypervolaemia together with reversal of anaemia may reduce echocardiographic disease and improve prognosis in CAPD patients. Due to the frequent occurrence of fluid retention in patients with CAPD, measurements of IVC diameter may help to guide the physicians for CAPD therapy. Further prospective studies examining the effects volume removal and amelioration of anaemia on LV geometry are warranted.

Conflict of interest statement. None declared.

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