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The benefit of salt restriction in the treatment of end-stage renal disease by haemodialysis

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Abstract

Background. Most haemodialysis (HD) centres use antihypertensive drugs for the management of hypertension, whereas some centres apply dietary salt restriction strategy. In this retrospective cross-sectional study, we assessed the effectiveness and cardiac consequences of these two strategies.

Methods. We enrolled all patients from two dialysis centres, who had been on a standard HD programme at the same centre for at least 1 year. All patients underwent echocardiographic evaluation. Clinical data were obtained from patients' charts. Centre A ($n = 190$) practiced 'salt restriction' strategy and Centre B ($n = 204$) practiced antihypertensive-based strategy. Salt restriction was defined as managing high blood pressure (BP) via lowering dry weight by strict salt restriction and insistent ultrafiltration without using anti-hypertensive drugs.

Results. There was no difference regarding age, gender, diabetes, history of cardiovascular disease and efficiency of dialysis between centres. Antihypertensive drugs were used in 7% of the patients in Centre A and 42% in Centre B ($P < 0.01$); interdialytic weight gain was significantly lower in Centre A (2.29 $\pm$ 0.83 kgversus 3.31 $\pm$ 1.12 kg, $P < 0.001$). Mean systolic and diastolic blood pressures were similar in the two centres. However, Centre A had lower left ventricular (LV) mass (indexed for height$^{2.7}$: 59 $\pm$ 16 versus 74 $\pm$ 27 g/m$^{2.7}$, $P < 0.0001$). The frequency of LV hypertrophy was lower in Centre A (74% versus 88%, $P < 0.001$). Diastolic and systolic functions were better preserved in Centre A. Intradialytic hypotension (hypotensive episodes/100 patient sessions) was more frequent in Centre B (11 versus 27, $P < 0.01$).

Conclusions. This cross-sectional study suggests that salt restriction and reduced prescription of antihypertensive drugs may limit LV hypertrophy, better preserve LV functions and reduce intradialytic hypotension in HD patients.

Keywords: echocardiography; haemodialysis; hypertension; left ventricular hypertrophy; salt restriction

Introduction

Cardiovascular disease is the leading cause of death in end-stage renal disease (ESRD) patients treated by maintenance haemodialysis (HD). Hypertension is present in the majority of patients on HD and is an important risk factor for cardiovascular diseases, especially those affecting the heart. In clinical practice today, the control of hypertension in this population is usually achieved by the use of antihypertensive drugs [1].

However, there are still a few centres that apply the drug-free control of hypertension approach using a 5–6 g dietary salt intake per day pioneered by Scribner in 1961 [2] and based upon the original work of Kempner [3]. Blood pressure (BP) control is achieved by lowering the end of dialysis body weight (dry weight) until optimal BP is reached. Shaldon confirmed the results of Scribner in 1963 [4] and described a new phenomenon of a delayed further drop in BP without a change in dry body weight now known as the lag phenomenon [5, 6]. We, together with Tassin and Seattle, have previously reported independently that optimal BP control can be achieved in >90% of ESRD patients using this method, with superior results when compared to drug control with an unrestricted salt intake, in an uncontrolled manner [7,8]. We, therefore, compared the effect of these two strategies on BP control in two HD centres using these contrasting treatment approaches.

Methods

This retrospective cross-sectional study included 423 HD patients, who had been treated by three times per week HD (scheduled as 12 h/week) at the same centre for at least a year, from two dialysis centres operated by

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Fresenius Medical Care in Turkey. Centres were compared by clinical, echocardiographic and laboratory data. All patients underwent echocardiography for cardiac evaluation in a midweek interdialytic day. Clinical data were collected retrospectively from patients' charts. Twenty-nine patients were then excluded after the collection of data because of the technically inadequate echocardiographic images. The study protocol was approved by local ethics committees, and informed consent was obtained from all participants.

Both centres were located in the western part of Turkey where the traditional dietary salt consumption was similar. Centre A ($n = 190$) used a strategy consisting of salt restricted diet (5 g/day) and intensive ultrafiltration to maintain pre-dialysis BP <140/90 mmHg without antihypertensive medication. Water intake was not restricted. If hypotension and/or cramps occurred during the period of dry weight reduction while pre-dialysis BP was still >140/90 mmHg, a test dose of oral captopril was given on a non-dialysis day in order to estimate whether the high BP was renin dependent. If BP decreased to <140/90 mmHg after 60 min, an angiotensin-converting enzyme inhibitor (ACE-I) or an angiotensin receptor blocker was started as the antihypertensive medication. Otherwise, development of intradialytic hypotension and/or cramps was considered as a sign that the ultrafiltration rate exceeded the refilling rate rather than as evidence of reaching dry weight, and hence the reduction of dry weight was continued. Retrospective evaluation of patients' charts revealed that additional ultrafiltration sessions were temporarily applied 1–7 times (mean 2.9 ± 1.5 sessions) as a tool to decrease the extracellular fluid volume in 25% of cases, usually within 2 months following admission to the centre. Consequently, the percentage of patients requiring antihypertensive drugs in Centre A was <10% [8]. Centre A has been practicing salt restriction strategy for BP control since the foundation of the centre in 2000. In Centre B ($n = 204$), antihypertensive drugs were used to control hypertension unless oedema was present. Hypotension and/or cramps which developed during ultrafiltration were interpreted as having achieved dry weight. Although salt restriction is also recommended in this centre, it was not emphasized insistently.

The following variables were obtained retrospectively from patient charts in order to assess population equivalence: age, gender, diabetes, tobacco use, history of cardiovascular disease, family history of hypertension, height, pre-dialysis body weight–dry weight–interdialytic weight gain (all weight parameters were averaged for the last three measurements before echocardiographic evaluation), intradialytic hypotension episodes, presence of anuria (diuresis <100 mL/day), serum creatinine, urea reduction rate [100 × (1 − pre-dialysis urea/post-dialysis urea)], haemoglobin and dose of erythropoietin, ferritin, transferrin saturation, serum albumin and use of antihypertensive drugs. All biochemical analyses were performed at the same laboratory from the blood samples drawn within the week, when echocardiography was performed (Architect c8000, Abbott-Diagnostics, Illinois, USA).

Pre-dialysis BP measured manometrically, was averaged for the last three HD sessions within the week when echocardiography was performed. Hypertension was defined as systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg. Symptomatic intradialytic hypotensive episodes, defined as a drop in systolic BP >20 mmHg requiring a saline infusion, were recorded from the last 10 HD sessions on patients' charts, and the results were expressed for each centre as the number of episodes per 100 HD sessions.

Echocardiographic examinations were performed according to the recommendations of the American Society of Echocardiography [9–10] on the day between 2 HD days (2.5 MHz transducer, Envisor C, Philips, Netherlands). All patients in Centre A underwent echocardiography at the end of August 2006 and Centre B's patients on the first days of September 2006. All echocardiographic examinations were recorded on CD and assessed at study completion by the same cardiologist. Standard echocardiography analysis included two-dimensional, M-mode and Doppler flow measurements. The following measurements were taken: left ventricular (LV) internal systolic and diastolic dimensions, thickness of the posterior wall and the interventricular septum, left atrium and aorta's systolic internal dimensions. All the measurements were indexed for body surface area (m^2), and normal ranges were considered according to Feigenbaum's appendix [11]. LV mass was calculated using the equation described by Devereux [12]. The LV mass index was calculated by dividing the LV mass by height squared to 2.71; LV hypertrophy was defined as the LV mass index >50 $g/m^{2.7}$ in males and 47 $g/m^{2.7}$ in females [13]. The LV mass index calculation was reliable in 168 patients in Centre A and 195 patients in Centre B because of the quality of the echocardiographic images. The left atrial volume was calculated using the area–length technique. Using this technique, the area of the left atrium was measured by planimetry of both apical views (A1 and A2). A linear dimension was measured from the centre of the mitral annulus to the superior border of the chamber (L). The left atrial volume was then calculated as [(0.85 × A1 × A2) ÷ L] [14–15] (normal volume: 38 ± 10 mL for men, 32 ± 10 mL for women) [16].

LV systolic function was assessed by LV ejection fraction and fractional shortening. LV diastolic performance was evaluated by using pulsed-wave Doppler ultrasound from mitral inflow tract in an apical four-chamber view. All Doppler measurements are given as the average values of three consecutive cardiac cycles. The peak early diastolic flow velocity (E), the peak atrial filling velocity (A), mitral deceleration time (from peak E wave to baseline) and isovolumic relaxation time (time interval from the aortic closing component of the second heart sound to the onset of mitral diastolic flow) were measured. As all these mitral inflow pulsed-wave Doppler measurements could be affected by preload (volume) [17], we evaluated LV tissue Doppler imagings (TDI) in order to increase the diagnostic accuracy of diastolic dysfunction [18,19]. TDI velocities of longitudinal mitral annular motion were recorded at septal and lateral annular borders. Spectral pulsed-wave Doppler was used with instrument setting adjusted to record the high amplitude/low velocity myocardial signals. The peak-systolic (Sm), early-diastolic (Em) and late-diastolic (Am) TDI velocities over mitral annulus were measured.

Table 1. Clinical and laboratory data of the haemodialysis centres

	Centre A ($n = 190$)	Centre B ($n = 204$)	*P*-value
Age (years)	54 ± 16	53 ± 15	ns
Female (%)	48	44	ns
Primary disease (%)			
Diabetes	21.5	26.9	ns
Hypertension	17.8	21.0	ns
Glomerulonephritis	14.7	11.7	ns
Polycystic kidney disease	5.2	5.8	ns
Other	19.4	18.6	ns
Unknown	21.0	15.6	ns
History of cardiovascular disease (%)	22	30	ns
Tobacco use (%)	34	29	ns
Family history of hypertension (%)	25	31	ns
Body surface area (m^2)	1.69 ± 0.18	1.66 ± 0.23	ns
Body mass index (kg/m^2)	24.2 ± 4.4	24.5 ± 3.9	ns
Duration of HDs (months)	52 ± 47	62 ± 43	ns
Urea reduction rate (%)	75 ± 8	75 ± 7	ns
Kt/V	1.42 ± 0.18	1.41 ± 0.16	ns
Anuric patients (%)	91%	79%	ns
Cardiothoracic ratio	0.46 ± 0.05	0.49 ± 0.08	0.018
Serum albumin (g/dL)	3.93 ± 0.36	3.86 ± 0.28	0.026
High-sensitive CRP (mg/dL)	1.57 ± 3.35	1.19 ± 2.21	ns
Haemoglobin (g/dL)	11.4 ± 1.6	10.4 ± 1.5	0.0001
Ferritin (ng/mL)	693 ± 364	705 ± 679	ns
Transferrin saturation (%)	26 ± 12	25 ± 13	ns
Use of erythropoietin (%)	37.9	45.1	ns
Dose of erythropoietin (U/kg/week)	103 ± 32	111 ± 43	ns

Values are expressed as mean ± SD unless otherwise defined.
ns: non-significant; HD: haemodialysis.

SPSS (Chicago, IL, USA) for Windows (Version 13.0) was used for statistical analysis. Data are presented as percentages for discrete variables and as mean ± SD for continuous variables. A *P*-value of <0.05 (two-sided) was regarded as statistically significant. Comparisons between centres were made either by the *t*-test or by the Mann–Whitney test based on the distribution pattern of the variables. The chi-square test was performed to test the differences in proportions. Linear correlation analysis was performed to investigate the association of cardiac parameters and clinical data, by using Pearson correlation testing (or Spearman correlation test when the data were not normally distributed or had ordered categories—coefficient rs). Stepwise multiple regression analysis was used to define the predictors of the LV mass index.

Results

The overall quality of dialysis was the same in the two centres, except the different salt intake policy. All cases were scheduled for 12 h weekly HD in three sessions, with the same membrane (polysulphone; FX series high-flux and HP series low flux). The duration of HD sessions (Centre A 231 ± 11 min, Centre B 227 ± 14 min), use of high-flux dialyser (Centre A 58%, Centre B 61%), dialysate sodium concentrations (138 ± 3 mmol/L in both) and dialysate calcium concentrations (Centre A 1.60 ± 0.14 mmol/L, Centre B 1.62 ± 0.12 mmol/L) did not differ among the centres. Table 1 summarizes the clinical and laboratory data of the centres. There was no difference regarding age, gender, the prevalence of diabetes, distribution of primary diseases, the duration of HD, cardiovascular disease history, tobacco use and hypertension in the family history. Figure 1 shows the similarity between the two centres' populations with regard to frequency distribution curves for HD duration and age. The efficiency of dialysis (estimated by Kt/V), use of erythropoietin, ferritin levels, transferrin saturation and high-sensitive CRP levels were similar in both centres. Serum albumin and haemoglobin levels were significantly higher in Centre A compared to Centre B (Table 1).

BP data are given in Table 2. The antihypertensive drug utilization rate was significantly lower in Centre A than in Centre B. Interdialytic weight gain was significantly lower in Centre A, where salt restriction was repeatedly emphasized. There was no significant difference between the mean systolic and diastolic BP levels obtained by the two methods of treatment. The number of symptomatic intradialytic hypotension episodes was significantly higher in Centre B.

Table 3 outlines the echocardiographic data of patients in the centres. Regarding cardiac structure, Centre A had a smaller mean left atrial volume index than Centre B. LV end-diastolic and end-systolic diameters indexed for body surface area were larger in Centre B, indicating cardiac dilatation. The LV mass index and frequency of LV hypertrophy were significantly higher in Centre B compared to Centre A. LV systolic functions were better preserved in Centre A, as evidenced by significantly higher ejection fraction and fractional shortening values.

Although the *E*/*A* ratio was comparable between the centres, the more sensitive markers of diastolic dysfunction, such as isovolumetric relaxation time, deceleration time and duration of the mitral inflow *A* wave, were significantly prolonged in patients of Centre B, indicating deterioration of the diastolic functions (Table 3). The left atrial volume index, which has been accepted as a marker of the chronicity of LV diastolic dysfunction [20], was also significantly higher in Centre B, showing the advanced degree of LV diastolic impairment. Moreover, the Em/Am ratio obtained from TDI of the mitral valve annulus was significantly higher in Centre A, reflecting better-preserved diastolic functions.

Among the variables studied, the LV mass index was correlated positively with systolic BP ($r = 0.14$, $P = 0.001$), interdialytic weight gain ($r = 0.2$, $P = 0.001$) and age ($r = 0.3$, $P = 0.001$) in both centres. The LV mass index was inversely related to haemoglobin ($r = -0.2$, $P = 0.001$) and albumin levels ($r = -0.2$, $P = 0.01$). Linear multiple regression analysis identified only systolic BP, age and interdialytic weight gain as independent variables associated with the LV mass index ($P = 0.0001$). The left atrial volume index was positively correlated with systolic BP ($r = 0.3$, $P = 0.001$) and inversely with haemoglobin ($r = -0.23$, $P = 0.002$). The duration of dialysis was not associated with any of the echocardiographic parameters including the LV mass index and LV diastolic function indices.

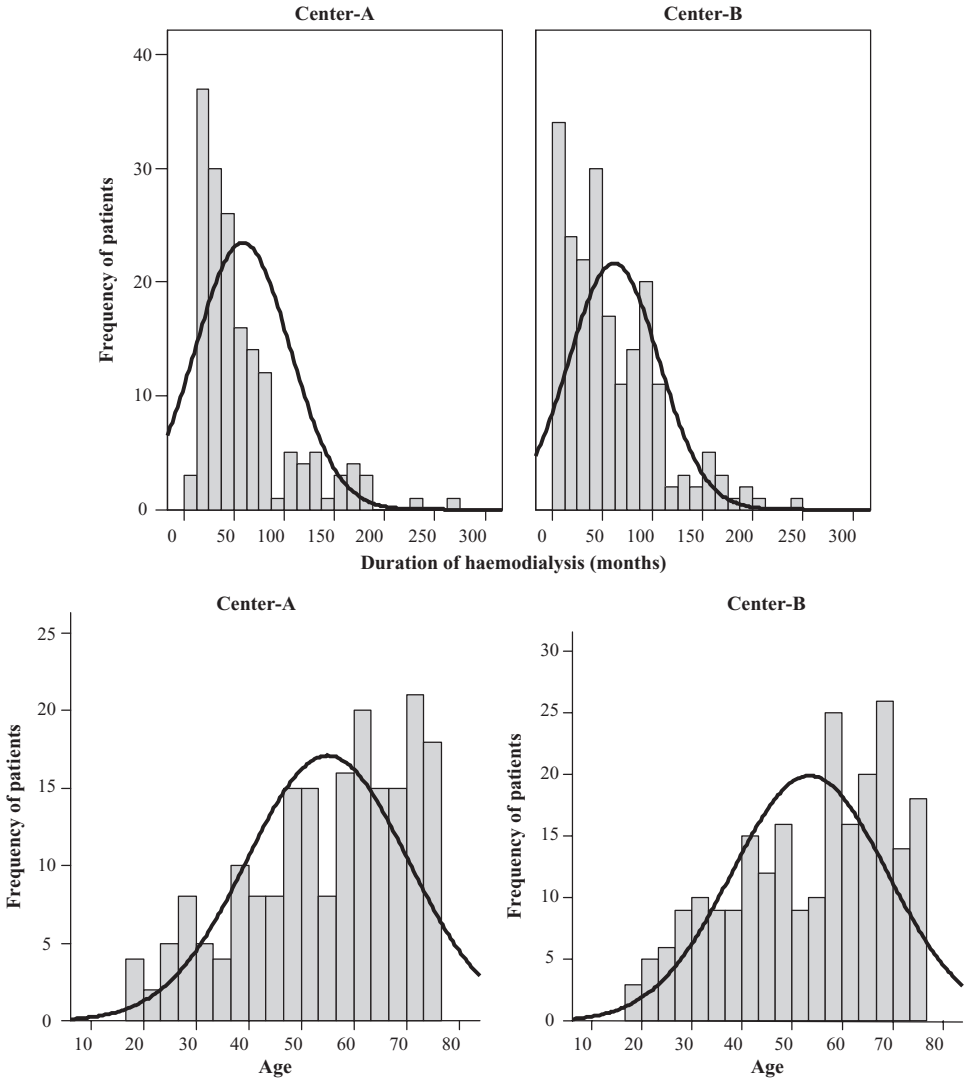

Fig. 1. The frequency distribution curves for the duration of haemodialysis and age of the centres.

Discussion

Hypertension is associated with adverse cardiovascular outcomes and significantly increased mortality in HD patients [21]. Despite the widespread use of antihypertensive medication in the HD population, most of the patients on chronic HD treatment are still hypertensive. In a cohort of 2535 prevalent HD patients, hypertension defined as systolic BP $\geq$150 mmHg and/or diastolic BP $\geq$85 mmHg was found in 86% of cases [1]. In addition, antihypertensive drug regimens did not prevent the progression of LV hypertrophy [22].

In contrast to these data, better results have been reported by centres using a 5 g or lower salt diet and long hours of dialysis. The experience with long overnight dialysis sessions (8–10 h, three times a week) was developed by Tassin [7], following its pioneering initial introduction by Shaldon in 1963 [4]. We have strictly adhered to the low-salt diet regimen with relatively good patient compliance without employing long hours of dialysis. We believe that there is enough evidence based upon survival data and rehabilitation to justify this approach of 3 $\times$ 4–5 h HD/week, provided that a strict adherence to the dietary salt restricted regimen is followed as judged by an interdialytic weight gain of 1.5 kg/70 kg of body weight in the anuric patient. We would suggest that this is the gold standard for rehabilitation and long-term survival of the ESRD patient today [23].

The present study is the first attempt at a cross-sectional comparison of two HD centres with regard to BP control by salt restriction (Centre A) as opposed to antihypertensive drug therapy (Centre B). Although mean systolic and diastolic BP levels were similar in the two centres, the proportion of hypertensive patients was higher in Centre B. More importantly, Centre B patients had a higher LV muscle mass index and prevalence of LV hypertrophy than Centre A patients. The lack of association between survival

Table 2. Blood pressure characteristics of the patients treated in two centres

	Centre A ($n = 190$)	Centre B ($n = 204$)	*P*-value
Use of antihypertensive medication (n = %)	13 (7%)	86 (42%)	0.001
ACE-/I or ARB	8	27	
Calcium channel blocker	1	43	
Beta blocker	2	3	
Furosemide	1	1	
Combination of two medications	1	12	
Interdialytic weight gain (kg)	2.29 ± 0.83	3.31 ± 1.12	0.0001
Interdialytic weight gain (kg for 70 kg man)	2.61 ± 0.98	4.05 ± 1.52	0.0001
Systolic BP (mmHg)	126 ± 15	126 ± 21	ns
Diastolic BP (mmHg)	75 ± 12	76 ± 11	ns
Pulse pressure (mmHg)	51 ± 9	50 ± 12	ns
Systolic BP ≥140 (%)	18	37	0.001
Diastolic BP ≥90 (%)	12	8	ns
Patients with systolic BP ≥140 and/or diastolic BP ≥90 (%)			
At the time of starting the HD programme	78	83	ns
Current situation	19	37	0.001
Intradialytic hypotension (number of episode per 100 HD sessions)	11	27	0.009

Values are expressed as mean ± SD unless otherwise defined.
BP: blood pressure, ns: non-significant.

Table 3. Echocardiographical data of the centres

	Centre-A ($n = 190$)	Centre-B ($n = 204$)	*P*-value
LA indices			
LA index (cm/m^2)	2.40 ± 0.34	2.74 ± 0.53	0.0001
LA volume index (mL/m^2)	29.5 ± 10.0	36.7 ± 21.7	0.0001
LV measurements and indices			
LV diastolic index (cm/m^2)	2.61 ± 0.33	2.97 ± 0.64	0.0001
LV end-systolic index (cm/m^2)	1.60 ± 0.29	1.96 ± 0.47	0.0001
Interventricular septalindex (cm/m^2)	0.79 ± 0.13	0.83 ± 0.14	0.018
Posterior wall index (cm/m^2)	0.76 ± 0.11	0.83 ± 0.11	0.0001
LV ejection fraction (%)	68 ± 10	63 ± 09	0.0001
LV fractional shortening (%)	39 ± 8	35 ± 6	0.0001
LV mass indexed to height$^{2.7}$ (g/m$^{2.7}$)	59 ± 16	74 ± 27	0.0001
LV hypertrophy (%)[a]	124 (74%)	171 (88%)	0.001
Pulsed Doppler parameters			
Mitral-inflow *E* (cm/s)	73 ± 22	76 ± 27	ns
Mitral-inflow *A* (cm/s)	83 ± 18	82 ± 25	ns
Deceleration time (min/s)	0.23 ± 0.06	0.28 ± 0.07	0.0001
Isovolumic relaxation time (min/s)	0.08 ± 0.01	0.12 ± 0.02	0.0001
Mitral-inflow *A*-wave duration (min/s)	0.14 ± 0.02	0.16 ± 0.03	0.0001
E/*A* ratio	0.90 ± 0.31	0.96 ± 0.33	0.076
Mitral valve lateral annulus Ee/Ae (min/s)	0.99 ± 0.43	0.89 ± 0.41	0.034

Values are expressed as mean ± SD.
LA, left atrium; LV, left ventricular; ns, non-significant.
[a]LV hypertrophy was defined as the LV mass index >50 g/m$^{2.7}$ in males and >47 g/m$^{2.7}$ in females.

time on dialysis and LV mass provides further evidence for our previous experience that salt restriction and volume control policy could prevent the development of LV hypertrophy [8]. This result is in contrast to that of others who have reported that LV hypertrophy is a progressive, even an inevitable abnormality in HD patients [22,24,25].

The finding that patients from Centre B not only had more LV hypertrophy but also had larger cardiac volume may indicate that these patients were more overhydrated. This might suggest that besides pressure load, increased volume load is an important factor in the development of LV hypertrophy in dialysis. Accordingly, both systolic BP and interdialytic weight gain were independent predictors of LV mass index, besides age.

ACE-I have been shown to regress LV hypertrophy in HD patients, as well as in the general population [26,27]. Despite similar BP control in the two study centres and significantly more utilization of ACE-I in Centre B, there was a low prevalence of LV hypertrophy in Centre A. This might support the important role of salt restriction independently of BP.

Yet, it is evident that HD patients are under constant threat of salt and water retention, and that strict volume control policy diminishes this trend. The fact that Centre A patients had less interdialytic weight gain confirms that they were compliant regarding their restricted dietary salt intake. Although the LV muscle mass index is notably lower in Centre A compared to Centre B, there is still a high prevalence of LV hypertrophy despite good BP and volume control. It would be interesting to examine whether the effect of ACE-I on LV hypertrophy is more pronounced in cases with better volume control. Compliance to the salt restricted diet is the cornerstone of the drug-free treatment of hypertension. The assessment of compliance is achieved by monitoring interdialytic weight gain in the anuric patient. The success of Centre A in decreasing interdialytic weight gain by dietary salt limitation confirms that the interdialytic weight gain is a reliable indicator of compliance and patients are never instructed to restrict water intake. The excellent long-term survival rates of the Tassin group [7] can also be attributed to sodium restriction, as they had prescribed salt restriction (5.0 g/day) and nominal dialysate sodium of 138 mmol/L [7]. In the general population, a reduction in dietary sodium intake to 5–6 g/day has been shown to reduce the incidence of cardiovascular disease by 25% in normotensive subjects with a hypertensive relative (pre-hypertensive) population compared to a similar population whose diet was not restricted in salt intake [28].

The surprising result seen in this study was the significantly lower prevalence of cardiovascular consequences in Centre A when compared to Centre B whose population was treated by antihypertensive drugs without strict salt restriction. Clearly, this suggests that some factor other than BP played a role in the development of the higher incidence of cardiovascular complications in Centre B.

Another important finding of our study is that intradialytic hypotension occurred less frequently in patients treated by restricted salt intake. More frequent use of anti-hypertensive medications and increased ultrafiltration rate, necessitated by higher interdialytic weight gain

due to unrestricted salt intake were possibly the major causes of higher frequency of intradialytic hypotension in Centre B.

The higher levels of haemoglobin observed in Centre A cannot be explained by iron status and erythropoietin use, which were similar in the two centres. We also assessed the possibility that the levels of albumin and haemoglobin could be already different at the beginning of the HD programme in two centres and found no difference between them (data not given). Relatively lower volume of plasma due to better extra-cellular fluid volume control by strict salt restriction in Centre A, which is suggested by the inverse correlation found between haemoglobin and left atrial volume index, may be considered as one of the causes of higher haemoglobin and albumin levels in this centre [29]. Another explanation might be the association between the inflammation, which leads to erythropoietin resistance and reduced albumin synthesis, and volume expansion and unrestricted salt intake [30,31]

The major limitation of the present study is its retrospective cross-sectional nature. Especially the lack of echocardiographical follow-up is an important limitation. Therefore, we cannot rule out a possibility that the LV structure and functions of study cases might be already different when the HD programme was started. However, as both centres were operated by the same organization, the overall quality of dialysis was the same in the two centres except for the different salt intake policy. Our study is hypothesis generating and makes even more compelling the need of performing a properly designed prospective randomized trial based on clinical end-points (mortality, cardiovascular events) is a true priority.

In conclusion, the results of this retrospective study suggest that salt restriction with the reduced use of antihypertensive medication for the management of high BP may reduce the prevalence of LV hypertrophy and LV dysfunction in dialysis patients.

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Conflict of interest statement. None declared.

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