

Regression of left ventricular hypertrophy after conversion to nocturnal hemodialysis

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Background. Left ventricular hypertrophy (LVH) is an independent risk factor for mortality in the dialysis population. LVH has been attributed to several factors, including hypertension, excess extracellular fluid (ECF) volume, anemia and uremia. Nocturnal hemodialysis is a novel renal replacement therapy that appears to improve blood pressure control.

Methods. This observational cohort study assessed the impact on LVH of conversion from conventional hemodialysis (CHD) to nocturnal hemodialysis (NHD). In 28 patients (mean age 44 ± 7 years) receiving NHD for at least two years (mean duration 3.4 ± 1.2 years), blood pressure (BP), hemoglobin (Hb), ECF volume (single-frequency bioelectrical impedance) and left ventricular mass index (LVMI) were determined before and after conversion. For comparison, 13 control patients (mean age 52 ± 15 years) who remained on self-care home CHD for one year or more (mean duration 2.8 ± 1.8 years) were studied also. Serial measurements of BP, Hb and LVMI were also obtained in this control group.

Results. There were no significant differences between the two cohorts with respect to age, use of antihypertensive medications, Hb, BP or LVMI at baseline. After transfer from CHD to NHD, there were significant reductions in systolic, diastolic and pulse pressure (from 145 ± 20 to 122 ± 13 mm Hg, $P < 0.001$; from 84 ± 15 to 74 ± 12 mm Hg, $P = 0.02$; from 61 ± 12 to 49 ± 12 mm Hg, $P = 0.002$, respectively) and LVMI (from 147 ± 42 to 114 ± 40 g/m², $P = 0.004$). There was also a significant reduction in the number of prescribed antihypertensive medications (from 1.8 to 0.3, $P < 0.001$) and an increase in Hb in the NHD cohort. Post-dialysis ECF volume did not change. LVMI correlated with systolic blood pressure ($r = 0.6$, $P = 0.001$) during nocturnal hemodialysis. There was no relationship between changes in LVMI and changes in BP or Hb. In contrast, there were no changes in BP, Hb or LVMI in the CHD cohort over the same time period.

Conclusions. Reductions in BP with NHD are accompanied by regression of LVH.

Key words: daily hemodialysis, home hemodialysis, hypertension, extracellular fluid volume, blood pressure, end-stage renal disease.

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Cardiovascular disease is the leading cause of death in patients with end-stage renal disease (ESRD) [1]. In patients undergoing conventional (4 hours, 3 times per week) hemodialysis, left ventricular hypertrophy (LVH) has been identified as an independent risk factor for mortality [2]. Different authors have estimated the prevalence of LVH in the general dialysis population at between 70 and 80% [3]. Although the exact pathophysiology of LVH in ESRD is unknown, hypertension, poor extracellular fluid (ECF) volume control, anemia, and uremia have been implicated in this process [2, 4].

Nocturnal hemodialysis (NHD), a novel mode of renal replacement therapy, provides eight to ten hours of hemodialysis during sleep six to seven nights per week. This mode of dialysis appears to lower blood pressure (BP) in dialysis patients [5, 6], but the reason(s) why blood pressure falls is unclear. Possible mechanisms include a decrease in ECF volume, vasodilation and abolition of nocturnal obstructive sleep apnea [7, 8]. Uremia control is superior to that achieved with conventional hemodialysis (CHD) [9]. NHD may therefore represent a beneficial alternative mode of renal replacement therapy for the ESRD population.

The purpose of this study was to determine left ventricular mass following conversion of ESRD patients from CHD to NHD. Patients receiving self-care home hemodialysis also were studied for comparison. Our specific aim was to determine whether any observed reduction in left ventricular mass related to one or more of lower BP, improvement in anemia or a decrease in ECF volume.

METHODS

All patients participating in the Nocturnal Hemodialysis demonstration project in Toronto for at least two years from November 1993 onward were included in this observational cohort study. The control cohort was drawn from patients eligible for NHD and enrolled in the To-

ronto General Hospital self-care home dialysis program for a minimum of one year from 1993 onward.

Patients received hemodialysis at home for eight to ten hours every night. Vascular access was achieved through either a long-term internal jugular catheter or an arteriovenous fistula. A dialysate flow rate of 100 to 500 mL per minute and F40, F50 or F80 polysulfone dialyzers (Fresenius Medical Care, Lexington, MA, USA) were used. In the self-care home dialysis program, each patient received hemodialysis for four hours, three times per week via similar vascular access. A dialysate flow rate of 500 to 750 mL per minute and F80 polysulfone dialyzers were used.

In both cohorts, clinical assessment, including weight, height, BP and hemoglobin (Hb) concentration was performed initially and every three months. Parathyroid hormone (PTH) was measured initially and every six months. Seated BP was measured during clinic visits by physicians or nurses after five minutes of rest. All BP measurements were obtained with the same calibrated sphygmomanometer. Echocardiographic data were obtained annually and interpreted blindly.

Prescribed cardiovascular medications were documented. These included diuretics, beta-blockers, angiotensin converting enzyme inhibitors, angiotensin receptor antagonists, digitalis, calcium channel blockers, and vasodilators, were documented. The dose of erythropoietin (EPO) prescribed also was documented.

In the NHD cohort, ECF volume was estimated by single-frequency bioelectrical impedance analysis [10] at baseline (prior to conversion) and annually thereafter. Electrodes were placed on the wrist and foot. Limbs with AV access were avoided. The RJL systems device (model, BIA-101Q; RJL Systems Inc., Clinton TWP, MI, USA) with Fluid and Nutrition Analysis Software 3.2 (RJL Systems Inc.) was used to compute ECF volumes. ECF volume was measured at baseline two to three hours after conventional dialysis, and in the morning after a regular session of nocturnal hemodialysis.

Left ventricular mass was calculated from two-dimensional (2D) echocardiographic images according to the formula of Devereux and Reichek [11]. The left ventricular mass index (LVMI) was derived by correcting the left ventricular mass for a body surface area of 1 m². Left ventricular hypertrophy was defined as LVMI >131 g/m² in males and >100 g/m² in females, as per the Framingham Study [12]. Fractional shortening (FS), an index of left ventricular systolic function, also was assessed.

The primary outcome measures were changes in LVMI, BP, ECF volume and Hb between baseline and the last recorded value for each cohort. Descriptive analyses are presented as mean \pm standard deviation. The paired Student *t* test was used for comparison of continuous variables within each cohort. The Student *t* test was used for comparison of continuous variables between

the two cohorts. Repeated analysis of variance was used for multiple comparisons of a continuous variable within a group. Linear correlation was used to investigate potential associations between variables of interest. All statistical tests were two-tailed with a *P* value less than 0.05 taken to indicate significance. The SPSS-10 software program (SPSS Inc., Chicago, IL, USA) was used for all statistical analyses.

RESULTS

Twenty-eight patients from the NHD cohort and thirteen patients from the CHD group met these inclusion criteria for analysis. Their baseline demographics are summarized in Table 1. There was no age difference between NHD and CHD cohorts (44 ± 7 vs. 52 ± 15 , $P > 0.05$). BP was adequately controlled in both groups. The interval between the first and last clinic measurement was similar (NHD: 3.4 ± 1.2 years vs. CHD: 2.8 ± 1.8 years, $P > 0.05$). Body mass index, BP, antihypertensive medication use, Hb and LVH values were similar in the two groups (Table 1).

Primary outcomes are summarized in Table 1. With NHD, there was a significant fall in BP but no change in post-dialysis ECF volume. The need for antihypertensive therapy also decreased yet there were significant reductions in systolic, diastolic, mean arterial and pulse pressure. There was a significant reduction in LVMI (from 147 ± 42 g/m² to 114 ± 40 g/m², respectively, $P = 0.004$) after conversion to nocturnal hemodialysis. At baseline, 7 of 28 (25%) patients in the NHD group and 5 of 13 (38%) patients in the CHD group had normal left ventricular mass. After conversion to NHD, 20 of 28 (71%) patients achieved normal left ventricular mass by Framingham criteria. In contrast, only 4 of 13 (31%) in the CHD group patients had normal left ventricular mass. Regression of LVMI was achieved through significant reductions in end-diastolic diameter, septal wall thickness and posterior wall thickness. At baseline, FS was similar in the two groups and tended to increase with long-term NHD. Hb also increased in the NHD cohort despite a trend toward lower EPO requirements. PTH tended to decrease in the NHD cohort. In contrast, there were no changes in LVMI, BP, Hb, EPO requirements, PTH or prescription of antihypertensive therapy in the CHD group.

The impact of NHD on LVH regression was detected by one year and was sustained thereafter (Table 2). Post-dialysis ECF volume was unchanged after conversion to NHD. There were no significant correlations between changes in LVMI and changes in systolic BP, pulse pressure, Hb or post-dialysis ECF volume. In the NHD patients, there was a significant relationship between the final LVMI and the final systolic blood pressure ($r = 0.6$, $P = 0.001$; Fig. 1).

Table 1. Primary outcome variables in chronic (CHD) and nocturnal (NHD) hemodialysis

Variables	CHD (N = 13)		NHD (N = 28)	
	Initial	Final	Initial	Final
LVMI g/m^2	142 \pm 33	150 \pm 56 ^b	147 \pm 42	114 \pm 40 ^a
SBP $mm\ Hg$	136 \pm 25	131 \pm 20	146 \pm 20	122 \pm 13 ^a
DBP $mm\ Hg$	82 \pm 13	80 \pm 15	84 \pm 15	74 \pm 12 ^a
PP $mm\ Hg$	54 \pm 22	51 \pm 17	61 \pm 12	49 \pm 12 ^a
MAP $mm\ Hg$	100 \pm 15	97 \pm 14	104 \pm 16	90 \pm 11 ^a
BMI kg/m^2	25.0 \pm 4.0	25.1 \pm 4.1	24.9 \pm 5.1	25.4 \pm 4.9
ECFV L			17.1 \pm 4.0	16.9 \pm 3.8
PTH $pmol/L$	40.7 \pm 36.2	40.1 \pm 30.0	34.9 \pm 55.0	16.6 \pm 24.3
Hb g/dL	11.7 \pm 14	11.9 \pm 1.1	10.7 \pm 1.3	11.8 \pm 1.6 ^a
EPO $IU/week$	5500 \pm 3471	8111 \pm 5043	10372 \pm 8065	8090 \pm 6832
Anti-BP meds	1.5	1.5	1.8	0.3 ^a
	ACEI: 27%	ACEI: 33%	ACEI: 19%	ACEI: 11%
	BB: 27%	BB: 27%	BB: 23%	BB: 78%
	CCB: 27%	CCB: 27%	CCB: 26%	ARB: 11%
	Vaso: 13%	Vaso: 7%	Vaso: 14%	
	Diuretic: 6%	Alpha: 7%	NTG: 8%	
EDD mm	51.4 \pm 4.6	50.6 \pm 9.1	50.7 \pm 7.8	48.4 \pm 7.0 ^a
ESD mm	35.0 \pm 6.9	33.9 \pm 8.6	32.3 \pm 9.7	30.5 \pm 8.1
FS %	34 \pm 5	32 \pm 8	37 \pm 11	41 \pm 15
SWT mm	10.9 \pm 1.2	11.4 \pm 1.3	10.9 \pm 2.4	9.6 \pm 2.2 ^a
PWT mm	10.4 \pm 1.6	11.1 \pm 1.5	10.8 \pm 2.1	9.4 \pm 2.4 ^a

Values are presented as mean \pm standard deviation. Abbreviations are: ACEI, angiotensin-converting enzyme inhibitor; Alpha, alpha receptor blocker; Anti-BP meds, antihypertensive medications; ARB, angiotensin receptor blocker; BB, beta receptor blocker; CCB, calcium channel blocker; DBP, diastolic blood pressure; ECFV, extracellular fluid volume; EDD, end-diastolic diameter; ESD, end-systolic diameter; FS, fractional shortening; Hb, serum hemoglobin concentrations; LVMI, left ventricular mass index; MAP, mean arterial blood pressure; NTG, nitrates; PP, pulse pressure; PTH, parathyroid hormone; PWT, posterior wall thickness; SBP, systolic blood pressure; SWT, septal wall thickness.

^a $P < 0.05$ compared to within-group initial value

^b $P < 0.05$ compared to between group final value

Table 2. Changes of principal variables in relation to time of nocturnal hemodialysis

	LVMI g/m^2	Hb g/dL	ECFV L
Baseline	147 \pm 42	10.7 \pm 1.3	17.1 \pm 4.0
Year 1	130 \pm 33 ^a	11.1 \pm 1.2	16.2 \pm 3.3
Year 2	106 \pm 32 ^a	11.7 \pm 1.6	16.2 \pm 3.4
Year 3	102 \pm 19 ^a	11.8 \pm 1.9	15.8 \pm 3.0

Values are presented as mean \pm standard deviation. Abbreviations are: ECFV, extracellular fluid volume; Hb, serum hemoglobin concentration; LVMI, left ventricular mass index.

^a $P < 0.05$ vs. baseline

DISCUSSION

Dialysis patients with LVH have higher mortality rates than those with normal ventricular mass [13]. In the present study, we observed regression of LVH after patients on CHD were converted to NHD. In contrast, LV mass did not change in those patients who remained on CHD. Conversion to nocturnal dialysis also resulted in a clinically important reduction in blood pressure and in the prescription of antihypertensive therapy.

The absence of any significant change in post-dialysis ECF volume, and the strong correlation between the lower SBP and LVMI after prolonged NHD, point to a significant role for hypertension in the pathogenesis of LVH in this population. Indeed, in 432 dialysis patients followed serially, a mean arterial blood pressure of greater than 106 mm Hg was strongly associated with the develop-

ment of LVH [14], and in a population of 72 pre-dialysis patients with chronic renal failure, one year of antihypertensive therapy with angiotensin converting enzyme inhibitors reduced the prevalence of cardiac hypertrophy by 20% [15]. Superior BP control also is thought to be the principal factor contributing to the regression of cardiac hypertrophy in patients receiving short daily dialysis [16–18]. Our findings are consistent with this conclusion. By contrast, CHD, in the present study had no impact on either BP or LVMI.

The absence of significant correlation between changes in LVMI, and changes in either BP, Hb or post-dialysis ECF volume following conversion to NHD indicates that the pathogenesis of LVH in dialysis patients is likely multifactorial, and does not depend on the change of any unique variable. Changes in ventricular architecture in this ESRD population, and reverse remodeling following long-term NHD, may provide additional insight into the relative roles of blood pressure and ECF volume expansion mediating these changes. Studies of hypertensive patients with primary hypertension implicate blood pressure load in the development of concentric LVH (increased wall thickness with increased LV mass), and in addition, increased plasma volume in those with eccentric LVH (normal wall thickness with increased LV mass) [19, 20]. As with previous reports in the ESRD population [21], our patients at baseline had primarily eccentric LVH. Following the conversion to NHD, there

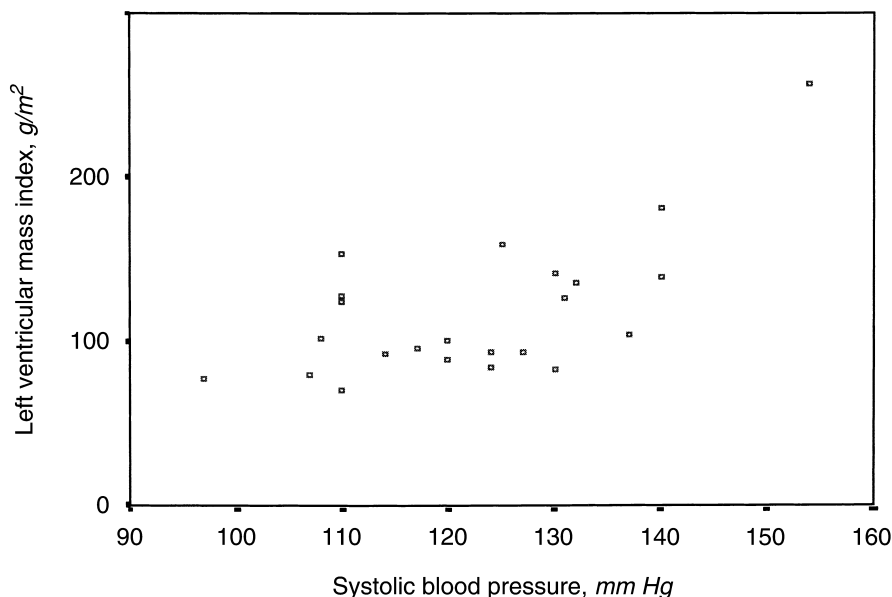


Fig. 1. Correlation between post-nocturnal hemodialysis (NHD) systolic blood pressure (SBP) and current left ventricular mass index (LVMI) ($r = 0.6$; $P = 0.001$).

were significant reductions in both LV wall thickness and chamber volume, suggesting a role for reductions in both BP and intravascular volume in this process of regression and reverse remodeling. However, ECF volume, as estimated by bioelectrical impedance analysis (BIA), was not diminished by the change in dialysis mode. We postulate that the magnitude of daily oscillations in ECF and average ECF volume in patients receiving CHD is decreased by NHD. A reduction in this diurnal repetitive mechanical stimulus to hypertrophy, over time, could explain why eccentric hypertrophy was also less prominent following NHD. It is interesting to note that Leenen and colleagues reached a similar conclusion when describing the regression of LVH in 18 hypertensive ESRD patients after they were placed on chronic ambulatory peritoneal dialysis (CAPD) [22]. Taken together, these observations suggest that more continuous modes of renal replacement therapy may promote regression of LVH likely through augmenting both volume and pressure management.

By what mechanism or mechanisms might conversion to NHD lower BP? The hypotensive effect of hemodialysis has been attributed to a reduction in ECF volume [16]. In the Tassin experience, Charzot et al reported that normotension can be achieved by aggressive control of post-dialysis ECF volume [23]. However, our present findings, which indicate that volume control is not necessary for hypotension to occur, are not unique. Savage et al studied 27 chronic hemodialysis patients with 48 hours of ambulatory BP monitoring between two midweek dialysis sessions [24]. They concluded that interdialytic BP changes were not related to interdialytic fluid gain, and emphasized the importance of additional factors beyond

fluid status in mediating these BP changes. Luik et al challenged ten hemodialysis patients with 3 liters of fluid and found no relationship between increases in intravascular volume and BP [25]. Even the Tassin group have suggested that normotension can be achieved if the dialysis time is long enough to ensure removal of possible vasoconstrictor factors [26].

McGregor et al conducted a randomized crossover study to establish if long intermittent dialysis was associated with better BP control than conventional HD [27]. These authors demonstrated a BP lowering effect of long intermittent HD in the absence of any change in dialysis ECFV. Nesrallah et al studied ESRD patients who were randomly assigned to short daily HD or NHD and found that BP fell in both groups of patients (abstract; Nesrallah et al, *J Am Soc Nephrol* 12:273A, 2001). However, post-dialysis ECF volume remained unchanged in the nocturnal dialysis cohort, in contrast to the short daily dialysis cohort in whom there was a fall in post-dialysis ECF volume. Parfrey et al have reported significant LVMI regression upon resolution of uremia by renal transplantation in 32 transplant recipients [28]. Thus, the present study adds to the growing evidence that blood pressure control in dialysis patients is not only related to fluid status, but may also arise from superior elimination of vasoactive and trophic substances such as catecholamines or elements of the renin-angiotensin-aldosterone system.

Normalization of Hb levels, as noted in the present analysis, has been studied previously and found not to increase BP or affect LV mass [29]. Therefore, the significant changes observed after the switch to NHD are more likely to arise from the change in dialysis mode than to the increase in Hb.

The present study indicates that the mode of hemodialysis impacts upon cardiac hypertrophy, but we could not definitively determine the mechanism responsible for LVH regression by NHD. It is reasonable to assume that BP, ECF volume and uremia all contribute to LVH progression in ESRD, and that NHD facilitates the regression of LVH by lowering BP, damping day-to-day fluctuations in ECF volume, removing uremic toxins more efficiently, or by more effective clearance of circulatory vasoconstrictors and molecules with trophic actions. Clinical experience, as reported by Charra, Bergström and Scribner underscores the importance of appropriate dry weight in achieving normotension in ESRD [30]. Since blood pressure control is a major determinant of LVH progression in ESRD, the crucial role of ECF volume in the pathogenesis of hypertension and LVH in ESRD requires further examination. The principal limitation of our study is its observational nature. Further studies to define the underlying pathophysiology as well as prospective trials to determine the impact of reducing LV mass on outcome are required.

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