Impact of Dialysis Modality on Left Ventricular Geometry in End Stage Renal Disease Patients

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A Dissertation Submitted to The Graduate School of

Ajou University in Partial fulfillment of The Requirements for

The Degree of Master of Medicine

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February, 2014

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-ABSTRACT-

Impact of Dialysis Modality on Left Ventricular Geometry in End Stage Renal Disease Patients

Background: Left ventricular hypertrophy (LVH) is an independent risk factor for morbidity and mortality in patients with end stage renal disease (ESRD). This study aimed to evaluate impact of dialysis modality on LV geometry by using echocardiography.

Methods: We retrospectively evaluated change in LV mass index (LVMI), relative wall thickness (RWT) and LV geometry, assessed by 2D transthoracic echocardiography, in patients starting dialysis while hospitalized from 2002 to 2012. Patients were classified into hemodialysis (HD) group and peritoneal dialysis (PD) group. Follow-up duration performing echocardiogram was 15±7months in HD group and 17±8 months in PD group. LV geometry was divided into 4 groups as normal LV geometry, concentric remodeling, concentric LVH and eccentric LVH according to LVMI and RWT using cutoff values of 115 (men) or 95 (women) g/m² for LVMI and 0.42 for RWT, both men and women. Severity of LVH was followed ASE recommendation (for men; mild: 116-131g/m^2, moderate: 132-148 g/m², severe: ≥149 g/m², for women; mild: 96-108 g/m², moderate:109-121 g/m², severe: ≥122 g/m²) of LVMI. The multivariate analysis was performed to evaluate the independent predictors 10% reduction of LVMI

Results: 84 patients with HD (age 55±13years, 51% male) and 36 patients with PD (age 50±14years, 61% male) were enrolled. Before initiation of dialysis, there were no significant difference of ejection fraction, LVMI, RWT (56±14% vs. 56±14%, p=0.810; 166.7±46.0g/m² vs. 167.8±54.6g/m², p=0.910; 0.46±0.10 vs. 0.46±0.08, *p*=0.960, respectively). Concentric LVH was most common in both groups before initiating

dialysis (58% vs. 64%). In HD group, there was no difference in LVMI (166.7±46.0g/m^2 vs. 165.5±47.2g/m^2, *p*=0.799) at follow-up, PD group had significant reduction of LVMI (164.8±54.6g/m^2 vs. 145.1±43.1g/m^2, *p*=0.021). Incidence of 10% reduction of LVMI was higher in PD group (33% vs. 56%, p=0.026). Predominance of concentric LVH did not changed in both groups at follow-up (61% vs. 61%). Patients with PD had decrease of severe LVH 67% to 50% and increase of normal LV 6% to 22% but it was no significant difference (*p*=0.090). By multivariable linear regression for predicting 10% LVMI regression, Independent predictors were presence of PD (odds ratio[OR]:2.119, 95% confidence interval [CI]:1.041-4.736, *p*=0.048), Diabetes mellitus (OR:0.464, 95%CI: 0.203-8.213, *p*=0.033), coronary artery disease (OR:0.339, 95%CI: 0.339-8.213, *p*=0.037), serum calcium (OR:0.579, 95%CI:0.293-1.134, *p*=0.011).

Conclusion: Otherwise there was no difference in prevalence of LV geometry pattern on baseline and follow up echocardiography, PD patients had reduction of LVMI and PD was an independent predictor for LVMI reduction

Keyword: Hypertrophy, Geometry, End stage renal disease

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I. INTRODUCTION

Cardiovascular complications are major cause of morbidity and mortality in patients with end stage renal disease (ESRD)(Morton, 1998). Progression of left ventricular hypertrophy (LVH) was associated with 62% 3-year cardiovascular events risk in hemodialysis patients (Zoccali et al., 2004). In 1992, Ganau et al.(Ganau et al., 1992) was classified LV geometry with normal, concentric remodeling, concentric hypertrophy, eccentric hypertrophy. There are some reports describing predominance of concentric LVH (40-63%) in ESRD patients (Levin et al., 1996; London and Parfrey, 1997; Ha et al., 1998; Li et al., 2009) and patients with ESRD had known to progress of LVH after starting dialysis, There was no data comparing longitudinal change of LV geometry after start of dialysis in hemodialysis (HD) and peritoneal dialysis (PD). This study aimed to investigate impact of dialysis modality on LV geometry by using echocardiography.

II. Material and Method

A. Patents

Study population consisted of impending end stage renal disease patients referred to our hospital for starting dialysis form 2002 and 2012, we enrolled all patients who started dialysis and maintained one modality. We included patients who performed echocardiography at least one month prior to start dialysis and performed follow-up echocardiography within 6-36months. We excluded patients who had the following exclusion criteria: age less than 18 years, overt cardiac comorbidities (dilated cardiomyopathy, hypertrophic cardiomyopathy, restrictive cardiomyopathy, severe arrhythmia, symptomatic aortic stenosis or more than moderate aortic and mitral regurgitation). Finally 120 patents were recruited into present study, comprising 84 HD patients and 36 PD patients.

B. Dialysis

The dialysis modality was determined by clinical decision of nephrologist and patients. All HD patients received a conventional twice or thrice-weekly HD and all PD patients received continuous ambulatory peritoneal dialysis.

C. Physical parameters & Laboratory parameters

Baseline physical parameters (blood pressure, heart rate, height and weight) were calculated as average value of all recording taken during the month before initiating the dialysis. For the follow-up physical parameters, in hemodialysis patients physical parameters were obtained by measured value at pre-dialysis period preceding the follow-up echocardiography. In PD patients the blood pressure, heart rate and weight were obtained by averaging measured value during the month preceding follow-up

echocardiography. /Blood sampling for the biochemical measurements were performed before echocardiography. Laboratory parameters were used data done before following dialysis in HD patients. In PD patients, we used parameters performed at a routine follow-up visit day. Biochemical parameters such as hemoglobin, serum creatinine, serum uric acid, serum albumin, serum calcium, serum phosphate and intact parathyroid hormone were measured. Corrected calcium was calculated as follows:

Corrected calcium (mg/dL) = total calcium+ (0.8 x (4.0-albumin[in g/dL])

D. Echocardiographic parameters

Echocardiographic measurements were performed based on the recommendations of American Society of Echocardiography (ASE) (Lang et al., 2005). Follow-up echocardiography was performed during the dialysis interval in HD patients and with an empty abdomen in PD patients. Ventricular dimensions were assessed through 2-D guided M-mode tracing. Echocardiographic parameters including left ventricular end diastolic diameter (LVEDD), inter ventricular septum diastolic thickness (IVSDT) and left ventricular posterior wall thickness (PWT) were measured at end-diastole. LV mass(LVM) was calculated according to Devereux formula (Devereux et al., 1994):

$$LVM\ (g) = 0.8 \times (1.04 \times [(LVEDD + IVSDT + PWT)^3 - (LVEDD)^3] + 0.6$$

LVM index (LVMI) was obtained by dividing LVM by BSA.

Relative wall thickness (RWT) was calculated by following formula:

$$RWT = 2 \times PWT/LVEDD$$

LVH was defined according to ASE recommendation (male, LVMI>115g/m^2; female, LVMI>95g/m^2) and classified as concentric LVH when RWT is>0.42, eccentric LVH

when RWT ≤0.42. Concentric remodeling was considered when RWT >0.42 with normal LVMI. Severity of LVH was followed ASE recommendation(for men; mild: 116-131g/m^2, moderate: 132-148 g/m^2, severe: ≥149 g/m^2, for women; mild:96-108 g/m^2, moderate:109-121 g/m^2, severe: ≥122 g/m^2. Regression of LVH defined as 10% reduction of LVMI on follow-up echocardiography compared to the baseline value.

E. Statistics

Continuous variables were expressed as mean± standard deviation (SD), catergrized variables were expressed as number and percentage. Inter-group, intra-group statistical comparisons used the paired and unpaired *t*-tests and chi square test. Multivariable linear regression was performed to identify independent predictors of regression of LVH. A series of variables were selected as independent variables in this model, such as age, sex, modality of dialysis, presence of diabetes mellitus (DM), coronary artery disease, previous myocardial infarction (MI), LV dysfunction (ejection fraction<45%) , SBP, HR, serum hemoglobin, calcium-phosphate product. For dialysis modality HD was defined as 1 (used as reference) and PD as 2. A two-tailed *p* value less than .05 considered significant. All analyses were performed using SPSS version 18.0 statistical software (SPSS Inc, Chicago, IL, USA).

III. Results

A. Baseline characteristics

HD group was older than PD group (55±13years vs. 49±14years). Briefly, there were no significant differences between the two groups in sex, duration of dialysis, comorbidities, medications and laboratory parameters (Table 1). Baseline echocardiographic parameters were no significant differences between two groups except E/E' was higher in PD group (7.8±9.0 vs. 18.3±7.1, p=0.001). Concentric LVH was most common in both groups (58.8% vs. 63.9%) and distribution of LV geometry did not differ (Table 2).

Table 1. Baseline characteristics.

	Hemodialysis (n=84)	Peritoneal dialysis (n=36)	*p*
Age, years	55±13	49±14	0.027
Male , n (%)	43 (51%)	22 (61%)	0.241
Mean f/u duration, months	15±7	17±8	0.293
Height, cm	163±9	165±8	0.222
Weight, Kg	63±11	63±12	0.980
Body surface area, g/m^2	1.67±0.18	1.68±0.19	0.593
Systolic blood pressure, mmHg	155±26	151±18	0.422
Pulse-pressure, mmHg	71±21	66±18	0.168
Heart rate, bpm	81±11	85±12	0.082
Hypertension, n (%)	72 (85%)	30 (83%)	1.000
Diabetes, n (%)	57 (67%)	22 (61%)	0.538
Dyslipidemia, n (%)	19 (22%)	5(14%)	0.330
Any smoking, n (%)	24 (28%)	13 (36%)	0.397
Current smoking, n (%)	16 (19%)	6 (17%)	0.805
Coronary artery disease, n (%)	15 (18%)	7 (19%)	1.000
Previous Myocardial infarction, n (%)	5 (6%)	0	0.320
Previous PCI, n (%)	12 (14%)	7 (19%)	0.585
Previous PAOD, n (%)	2 (2%)	0	0.579
Previous CVA, n (%)	1 (1%)	3 (8%)	0.078
Medication			
RAS blocking agent	41 (48%)	13 (36%)	0.319
ACEi, n (%)	9 (11%)	4 (11%)	1.000
ARB, n (%)	32 (38%)	9 (25%)	0.211
β-blocker, n (%)	29 (34%)	21 (58%)	0.016
CCB, n (%)	59 (69%)	24 (67%)	0.831
Statin, n (%)	17 (20%)	5 (14%)	0.458
Laboratory data			
Serum creatinine, mg/dL	9±4	11±5	0.143

Serum calcium, mg/dL	7±1	8±1	0.069
Serum phosphate, mg/dL	6±2	6±2	0.114
Ca x PO4	46±14	44±16	0.513
Serum uric acid, mg/dL	9±2	9±3	0.476
Serum albumin, g/dL	3 ±1	3±1	0.522
Serum hemogrobin, g/dL	8±2	8±2	0.443
Serum iPTH, pg/mL	276±273	262±251	0.845

Values are mean ± standard deviation (SD)
HD= hemodialysis, PD= peritoneal dialysis, PCI= percutaneous coronary intervention, PAOD= peripheral artery obstructive disease, CVA= cerebrovascular accident, ACEi= angiotensin converting enzyme inhibitor, ARB= angiotensin receptor blocker, CCB= calcium channel blocker, iPTH= intact parathyroid hormone

Table 2. Baseline echocardiographic parameters.

	Hemodialysis(n=85)	Peritoneal dialysis (n=36)	*p*
EF,%	56±14	56±14	0.810
<45% of EF ,n(%)	19 (22%)	7 (19%)	0.812
LV mass, g	277±81	271±73	0.683
LV mass index, g/m^2	166.7±46.0	168±55	0.910
RWT	0.46±0.10	0.46±0.08	0.960
LVEDD, mm	54±6	54±5	0.874
LVESD, mm	37±9	36±7	0.731
IVSDT, mm	12 ±2	12±3	0.604
PWT, mm	12±2	12±2	0.465
LA, mm	44±6	45±6	0.548
LV geometry			0.893
Normal, n (%)	7 (8%)	2 (6%)	0.725
Concentric remodeling n (%)	3 (4%)	0	0.554
Concentric hypertrophy n (%)	50 (59%)	23 (64%)	0.686
Eccentric hypertrophy n (%)	26 (31%)	11 (31%)	1.000

Values are mean ± SD or n (%)

EF= ejection fraction, LV= Left ventricle, RWT= relative wall thickness, LVEDD= LV early diastolic dimension, LVESD= LV early systolic dimension, IVSDT= interventricular septum diastolic thickness, PWD= posterior wall dimension, LA= Left atrium

B. Comparison in change of laboratory and echocardiographic parameters after dialysis

Both groups showed significant reduction of BSA (1.66±0.18 g/m^2 vs. 1.63±0.02 g/m^2, p<0.001; 1.68±0.19 g/m^2 vs. 1.63±0.19 g/m^2, p<0.001, respectively), improvement of anemia (8.3±1.8 g/dL vs. 10.0±1.6g/dL p=0.001; 8.1±1.5 vs. 10.1±1.7, p<0.001) at follow-up (Table 3.). In HD group, Significant reductions of serum calcium-phosphate product and intact PTH (46.3±14.0 vs. 38.1±17.0, p<0.001; 277.3±81.1 pg/mL vs. 165.5±188.3 pg/mL, p=0.012) were observed. Other clinical indexes were no difference between two groups after starting dialysis. LVEDD was reduced in both groups after dialysis but there was no difference between the inter-groups. Figure 1. showed individual ΔRWT and ΔLVMI over time. Only PD patients showed significant regression of LVMI (164.8±54.6 g/m^2 vs. 145.1±43.1 g/m^2, *p*=0.021) (Figure 1.). Distributions of LV geometry were no significant change at follow-up in both groups (Figure 2.,Table 4). Severe hypertrophy was predominant on baseline and follow-up echocardiography in both groups. Patients with PD had decrease of severe LVH 66.7% to 50% and increase of normal LV 5.6% to 22.6% but it was no significant difference (*p*=0.090) (Fig. 3.). Incidence of >10% regression of LVMI was higher in PD patients (32.9% vs. 55.6%, p=0.026) (Figure 4.). By multivariable linear regression for predicting 10% LVMI regression, Independent predictors were presence of DM, CAD, PD, serum calcium (R^2=0.438, *p*<0.001) (Table 5.).

Table 3. Comparison of changes in clinical indexes and echocardiographic parameters.

	Hemodialysis			Peritoneal dialysis			
	Baseline	Follow-up	*p*	Baseline	Follow-up	*p*	Follow-up HD vs. PD, *p*
Weight, Kg	63±11	59±12	<0.001	63±12	64±11	0.548	0.065
BSA, g/m^2	1.66±0.18	1.63±0.02	<0.001	1.68±0.19	1.69±0.17	0.679	0.075
SBP, mmHg	155±26	151±28	0.449	151±18	149±21	0.496	0.533
Pulse pressure, mmHg	71±21	73±25	0.615	66±18	63±18	0.403	0.026
Heart rate,bpm	81±11	82±15	0.631	85±12	82±14	0.302	0.834
Serum creatinine, mg/dL	9±1	8±4	0.007	11±5	11±5	0.644	0.001
Serum calcium,mg/dL	8±1	9±1	<0.001	8±1	9±1	0.002	0.960
Serum phosphate,mg/dL	6±2	4±2	<0.001	6±2	5±2	0.063	0.041
Serum hemoglobin, g/dL	8.3±1.8	10.0±1.6	<0.001	8.1±1.5	10.1±1.7	<0.001	0.825
Ca x P	46 ±14	38±17	<0.001	47±17	44±16	0.446	0.057
Serum uric acid, mg/dL	9±2	6±2	<0.001	9±3	6±2	<0.001	0.020
Serum albumin, g/dL	3±1	4±1	<0.001	3±0	3±1	0.676	0.001
Serum hemoglobin, g/dL	8±2	10±2	<0.001	8±2	10±2	<0.001	0.825
Serum iPTH, pg/dL	289±218	166±188	0.012	237±268	2089±287	0.443	0.283
EF ,%	56±14	56±13	0.849	56±14	59±15	0.370	0.312
LV mass, g	277±81	267±75	0.254	271±73	245±73	0.093	0.106
LVmass index, g/m^2	167±46	165±48	0.799	165±55	145±43	0.021	0.024
RWT	0.45±0.08	0.47±0.10	0.047	0.46±0.08	0.47±0.11	0.155	0.851
LVEDD, mm	54±6	53±6	0.033	54.0±4.6	51±6	0.018	0.222
LVESD, mm	37±9	37±8	0.623	36±7	34±10	0.077	0.180
IVSDT, mm	12±2	12±2	0.288	12±3	12±2	0.78	0.156
PWT, mm	12±2	12±2	0.619	12±2	12±2	0.869	0.475
LA, mm	44±6	43±8	0.709	45±6	42±7	0.182	0.506

Values are mean ± SD or n (%)

SBP= systolic blood pressure, BSA= body surface area, iPTH= intact parathyroid hormone, EF= ejection fraction, LV= Left ventricular, RWT= relative wall thickness, LVEDD= LV end diastolic diameter, IVSDT= interventricular septum diastolic thickness

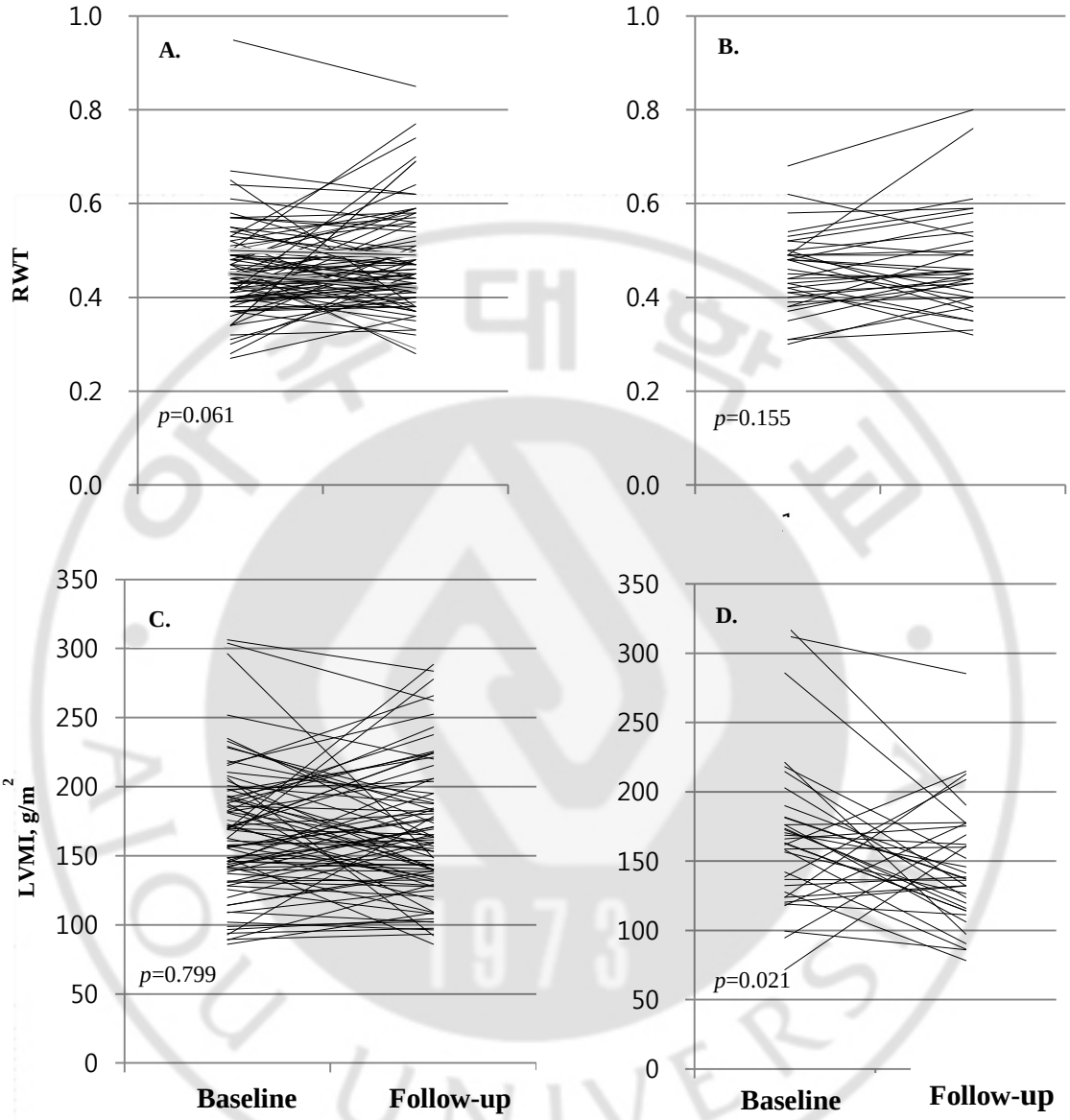

Fig. 1. Individual ΔRWT and ΔLVMI over time.

ΔRWT (A.) and ΔLVMI (C.) in Hemodialysis patients. ΔRWT (B.) and ΔLVMI (D.) in Peritoneal dialysis patients. RWT= relative wall thickness, LVMI= left ventricular mass index.

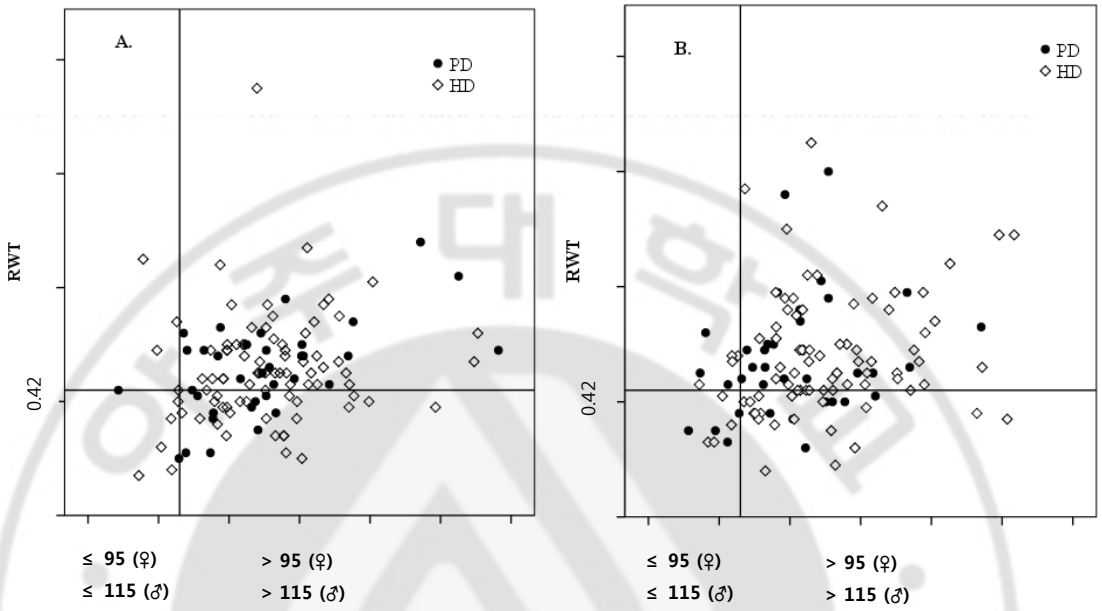

Fig. 2. Distributions of LV geometry of Baseline (A.) and follow-up echocardiography (B.).

HD= hemodialysis, PD= peritoneal dialysis.

Table 4. Comparison of LV geometry

	Baseline	follow-up	*p*	Baseline	follow-up	*p*
	Concentric remodeling			Concentric hypertrophy		
HD, n (%)	3 (4%)	4 (5%)	1.000	49 (58%)	51 (61%)	0.876
PD, n (%)	0	3 (8%)	0.239	23 (64%)	22 (61%)	1.000
p	0.553	0.427		0.685	1	
	Normal			Eccentric hypertrophy		
HD, n (%)	6 (7%)	4 (5%)	0.535	11 (31%)	25 (30%)	1.000
PD, n (%)	2 (6%)	4 (11%)	0.674	26 (31%)	7 (19%)	0.415
p	0.722	0.239		1	0.270	

Values are mean ± SD or n (%)
HD= hemodialysis, PD= peritoneal dialysis, LV= left ventricle

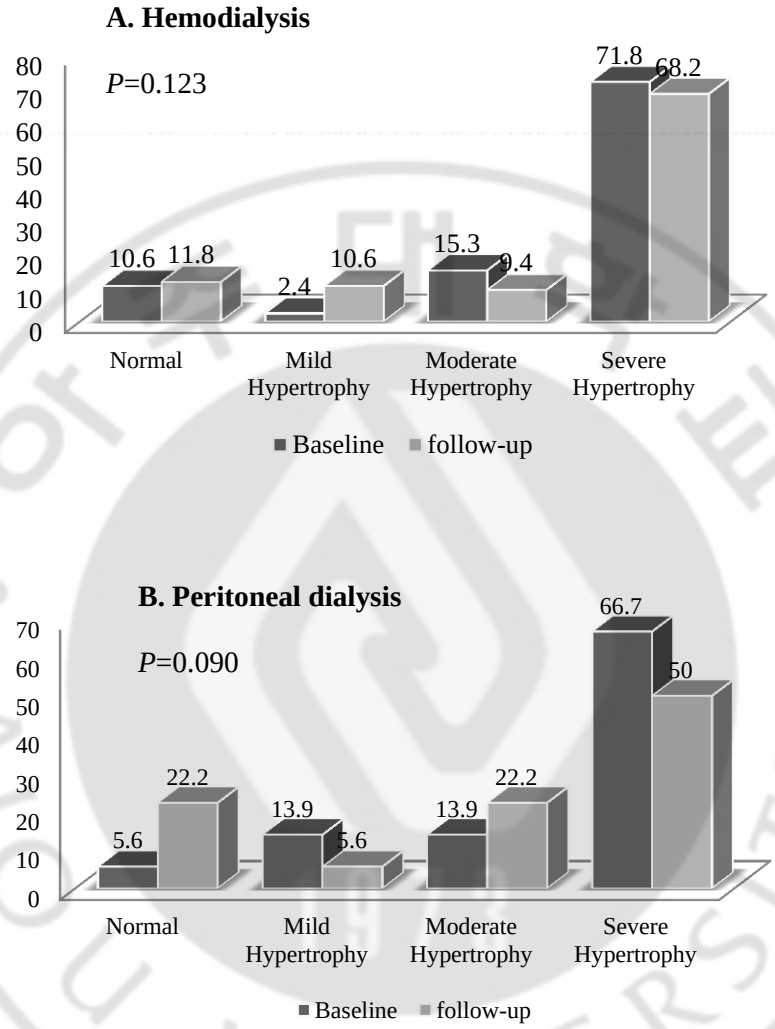

Fig 3. Comparison of severity of LVH at baseline and follow-up echocardiography

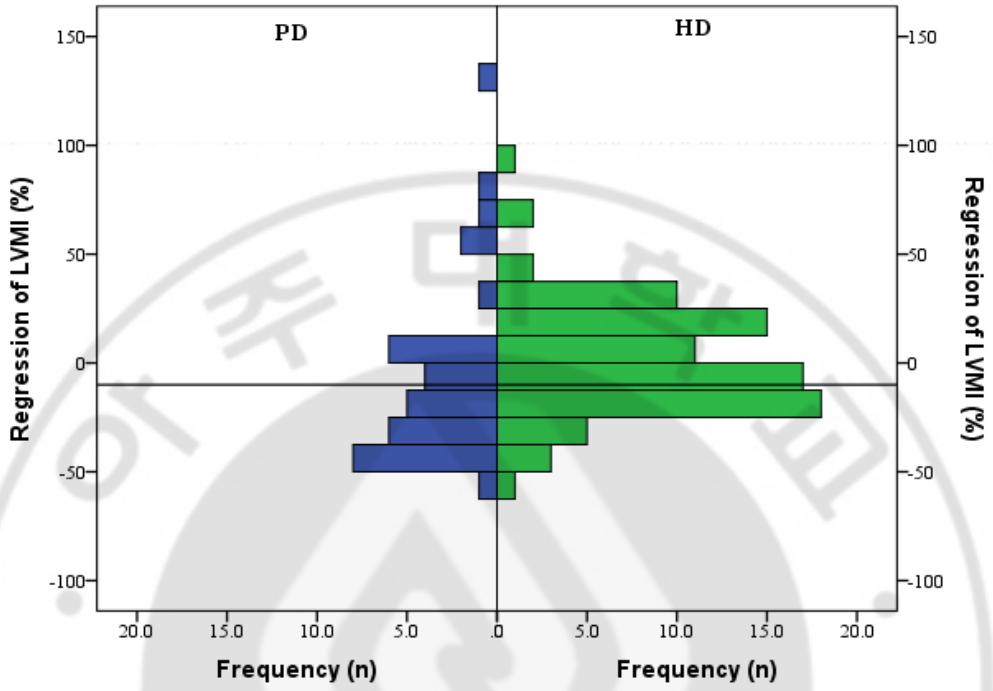

Fig 4. Incidence of LVH regression

LVH regression defined as 10% reduction of LVMI (black line) on follow-up echocardiography compared to the baseline value. LVH= left ventricular hypertrophy, LVMI= LV mass index.

Table 5. The output from a multivariable linear regression to predict >10% of LVMI regression.

Variable	β (coefficient)	95% CI	*p*
PD	2.119	1.041-4.736	0.048
CAD	0.339	0.339-8.213	0.037
DM	0.464	0.203-8.213	0.033
Serum calcium	0.576	0293-1.134	0.011

The initial model included following variables: age, sex, modality of dialysis, presence of diabetes mellitus (DM), coronary artery disease, previous myocardial infarction, heart failure (ejection fraction [EF] <45%) , systolic blood pressure, diastolic pressure, heart rate, serum hemoglobin, serum calcium, calcium-phosphate product, serum intact PTH. variables of volumetric data included multivariate analysis were selected by p value < 0.02 of correlation analysis. LVMI= Left ventricular mass index, PD= peritoneal dialysis, CAD= coronary artery disease, PTH= parathyroid hormone, R^2=0.438, p<0.001.

IV. Discussion

LVH assessed by echocardiography was known as risk factor of cardiovascular disease apart from the conventional risk factors (Levy et al., 1990). ~70 to 80% of patients with stage 4 to 5 CKD have LVH before the initiation of dialysis. Systolic arterial hypertension and elevated pulse pressure are strongly associated with LVH in patients with advanced CKD, suggesting that fluid overload , increase of systolic blood pressure, arterial stiffness play a role in progression to LVH (Paoletti et al., 2005), in addition anemia, secondary hyperparathyroidism and chronic micro-inflammation had known to contribute developing LVH (Harnett et al., 1994). In this study, 90% of patients had LVH in pre-dialysis period and among them, 70.2% had severe LVH. We conducted subgroup analysis to evaluate effect of LVH on cardiovascular events (composite with cardiac death, myocardial infarction, coronary artery revascularization). There was no significant difference, but patients with LVH had higher incidence of cardiovascular events (23.0% vs. 9.5% p=0.239).

A few studies had conducted comparing the impact of dialysis modality on LV remodeling. According to Previous study, on patients with PD had lower incidence of LVH in PD patients after short duration of dialysis by maintaining residual renal function and more adequate volume control (Tian et al., 2008), in contrast Enia et al. reported long-term PD patients had severe LVH compared to HD patients because long-term PD had deterioration of residual renal function and progressive loss of peritoneal ultrafiltration capability (Enia et al., 2001). But other two studies showed higher prevalence of LVH in HD patients at short-term and long term duration of dialysis (Hiramatsu et al., 2007; Tian et al., 2008). These results are consistent with ours. Hiramasu et al. compared longitudinal changes in echocardiographic parameter but they based on this study just 26 patients, and Tian et al. conducted cross sectional study. Our study is based on patients with ‘real world’ practice of dialysis, though echocardiography follow-up duration was shorter than those studies. And we had

conducted study with larger number of patients and compared longitudinal change of echocardiographic and clinical parameter. After initiating dialysis, patients in both groups did not reduced systolic blood pressure after dialysis. Pulse-pressure was lower in PD groups after dialysis (72.5±24.7 vs. 63.3±18.3 p=0.026). Though prevalence of LVH was similar in both groups (88% vs.77.8%, *p*=0.166) and severe LVH was predominant, There was significant reduction of LVMI in patients with PD (165.5±47.2 vs. 145.1±43.1 p=0.028). Tian et al. showed more appropriate control of ECW in PD group and Hiramasu et al showed more stable cardiac parameters after long term dialysis. We did not checked ECW but more regression of LVEDD showed indirect evidence of appropriate ability of volume control in PD. Besides there was improvement of anemia, intact PTH and serum calcium-phosphate in HD group, there was no reduction of LVMI. HD have some co-factors affecting LVH in addition to blood pressure and volume status. Previous study reported that large flow arterio-venous fistular contributed to progression of LVH by lowering vascular resistance, resulting in an increase of venous return. And conventional thrice-weekly diffusive hemodialysis, excessive ultrafiltration and inter-dialytic hypotension associated with myocardial injury (Burton et al., 2009).

LV geometry classified by LVMI and RWT as 4 types, it determined by predominant pathogenetic factor whether afterload related factor or preload related factor (Ganau et al., 1992). Afterload related factors are systemic arterial resistance, elevated systolic (and diastolic) arterial blood pressure, and large vessel compliance and increased afterload lead to myocardial cell thickening and concentric LVH. Preload related factors are expansion of intravascular volume (water and salt loading), anemia, large flow arterio-venous fistula. Increase of preload cause lengthening of myocardial cell and eccentric LVH. (Krumholz et al., 1995). But it is difficult to anticipate change of LV geometry in patients with ESRD after initiating dialysis because of afterload related factor and preload related factor are not independent and they are closely related to each other in ESRD patients. Many studies demonstrated a predomination of

concentric LVH (40-63%) in ESRD (London and Parfrey, 1997; Ha et al., 1998; Li et al., 2009). Our result consisted with previous studies, most common type was concentric hypertrophy in both groups before and after dialysis. Perhaps predominance of concentric hypertrophy is because continued higher rate of HTN. Though significant geometric change did not observed in both group, there were reduction of eccentric LVH (30.6% to 19.4%) and increase of normal geometry (5.6% to 11.1%) in PD group. To define the impact of dialysis modality on LV geometry should be more patients and long term data. And insufficient correction of confounding factor for LVH such as SBP, anemia and serum calcium-phosphate might influence no change of LV geometry in both groups. London et al. reported 10% regression LVM reduce 28% cardiovascular mortality. Previous study reported factors associated with LV mass reduction as younger age, lower pulse pressure, higher GFR (McMahon et al., 2004). According to our results, patients with PD had lower pulse pressure and significantly reduced LVMI. And PD and absence of CAD remained independent predictor for ≥10% LVMI regression (London et al., 2001).

This study has some limitations. First, this study is observational, retrospective, it includes possibility of incomplete data collection and a dependency on previous recorded data in the medical records. Second this study made up of small number of patients and we did not showed long-term outcome. Third, there is lack of factors relating volume status such as residual renal function and dialysis adequacy. To conduct accurate comparison, it is necessary to correcting aforementioned limitations.

V. Conclusion

We compared longitudinal change of echocardiographic and clinical parameter after start of dialysis in HD and PD patients who received “real world” conventional practice and tried to investigate impact of dialysis morality on LV geometry. Though this study did not showed significant geometric change of LV after start of dialysis, in PD patients had significant LVMI regression and PD was independent predictor for LVMI regression. We could conclude that PD is more feasible than HD aspect of reduction of LVH.

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말기 신부전 환자에서 투석 방법에 따른 좌심실 형태 변화의 영향

배경: 좌심방 비대는 말기 신부전 환자에서 사망률을 증가시키는 독립인자이다. 본 연구는 심장 초음파를 이용하여 투석 방법이 좌심방 형태에 미치는 영향을 밝히고자 한다.

Methods: 후향적으로 2002년에서 2012년 사이 투석을 시작한 환자에서 2D-경흉부 초음파를 이용하여 좌심방 질량지수(LVMI), 상태적 벽두께(RWT), 좌심방 형태의 변화를 조사하였다. 투석 방법에 따라 혈액 투석군과 복막 투석 군으로 나누었다. 추적 심초음파를 시행한 시기는 혈액투석 15±7 개월, 복막투석군이 17±8 개월로 양군에서 차이는 없었다. 좌심실 형태는 LVMI 가 남자는 115g/m^2, 여자는 95g/m^2 이하, RWT가0.42 이하를 정상치로하고, 정상 LVMI 와 RWT 가 0.42이상인 군을 구심성 재구성, LVMI가 정상치 이상인 경우 RWT 이 0.42이상인 경우 구심성 비대, 0.42 이하인 경우 원심성비대로 구분하였다. 좌심실 비대의 중증도는 American society of echocardiography 의 권고에 따라 나눴다 (남자; mild: 116-131g/m^2, moderate: 132-148 g/m^2 , severe: ≥149 g/m^2 , 여자; mild: 96-108 g/m^2 , moderate: 109-121 g/m^2 , severe: ≥122 g/m^2 of LVMI). 10% LVMI 감소의 예측인자를 구하기 위하여 다변량 분석을 이용하였다.

결과: 84명의 혈액투석 환자 (age 55±13years, 50% male) 와 36 명의 복막투석 (age 49±14 years, 62% male) 환자를 대상으로 하였고, 투석

시작 전 양 군에서 좌심실 구축율과 LVMI, RWT 의 차이는 없었고 (56±14% vs. 56 ±14%, p=0.810; 166.7±46.0g/m² vs. 167.8±54.6g/m² , p=0.910; 0.456±0.094 vs. 0.455±0.082, *p*=0.96, respectively), 투석 전 구심성 비대가 가장 많았다. (59% vs. 64%). 투석 후 혈액투석군에서 LVMI 의 변화는 없었고 (166.7±46.0g/m^2 vs. 165.5±47.2g/m^2, *p*=0.799), 복막 투석군에서 LVMI가 의미있게 감소하였고(164.8±54.6g/m^2 vs. 145.1±43.1g/m^2, *p*=0.021), 혈액 투석 군보다 LVMI 가 10% 이상 감소한 비율이 높았다(33% vs. 56%, p=0.026). 추적 심초음파에서 양군 모두 구심성 심비대의 우세는 변하지 않았으나 (61% vs. 61%) 복막 투석 환자군에서 중증 심비대가 의미있지는 않았으나 67% 에서 50%로 감소하였다. 다변량 분석에서 복막투석과 (odds ratio[OR]:2.119, 95% confidence interval [CI]:1.041−4.736, *p*=0.048), 당뇨 (OR:0.464, 95%CI: 0.203−8.213, *p*=0.033), 심혈관질환 (OR:0.339, 95%CI: 0.339−8.213, *p*=0.037), serum calcium (OR:0.579, 95%CI:0.293−1.134, *p*=0.011)이 LVMI 10%이상 감소의 독립인자였다.

Conclusion: 두 군에서 투석 전후 좌심실 형태의 분포의 차이는 보이지 않았으나 복막 투석 환자에서 LVMI 가 의미있게 감소하였으며 복막 투석은 LVMI 10%이상 감소의 독립인자였다.

Keyword: Hypertrophy, Geometry, End stage renal disease