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Echocardiographic Parameters as Cardiovascular Mortality Predictors in Chronic Hemodialysis Patients

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Abstract

Background: Hemodialysis (HD) patients have high rates of cardiovascular (CV) mortality. Although structural and functional echocardiographic alterations in HD patients have been the subject of several survival analysis studies, the prognostic value of these alterations is not well established. The aim of this study was to determine the prognostic value of echocardiographic parameters in chronic HD patients. Objectives: One hundred eighteen HD patients were clinically evaluated and underwent Doppler echocardiography, being followed for 45.7 ± 13.6 months. The outcome measures were CV mortality. The predictive value of echocardiographic variables was evaluated by Cox regression model and survival curves were constructed using the Kaplan-Meier method and log-rank test to compare them. Results: CV diseases accounted for 46.4% of all deaths during the follow-up period. We found that the event-free survival rates in one and two years were 96.5% and 83.0%, respectively. Diabetes and E/e' ratio were predictors of CV outcome by multivariate analyses. Conclusion: Diabetes and diastolic dysfunction are independent predictors of CV mortality in chronic HD patients.

Keywords

Diastolic Dysfunction, Echocardiography, Hemodialysis, Cardiovascular Mortality, Prognosis

1. Introduction

The annual mortality rates in hemodialysis (HD) patients are high. According to the dialysis census of the USA,

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the survival of HD patients in the country was 77.4% in one year and 34.2% in five years, from 1999 to 2003 [1]. In Japan, the annual crude mortality was 10.2% in 2011[2].

Cardiovascular (CV) diseases account for approximately 40% - 50% of all deaths in HD patients. Moreover, these patients are often hospitalized and CV diseases account for approximately one third of hospital admissions [3].

Structural and functional alterations detected by echocardiography, such as left ventricular (LV) hypertrophy and systolic and diastolic dysfunction, are very prevalent in the HD population. Doppler echocardiographic diagnosis of these abnormalities has been an important step for the characterization of individuals with higher CV risk [4] [5].

Several studies have reported to determine the prognostic value of alterations such as LV hypertrophy and systolic dysfunction in HD patients [6]-[8]. However, studies evaluating the predictive value of diastolic dysfunction in this population are scarce. Thus, the objective of this study was to determine the prognostic value of echocardiographic parameters in chronic HD patients.

2. Methods

2.1. Study Design and Population

This is an observational and prospective cohort study. The subjects of this study were 118 maintenance HD patients (83 men and 35 women) treated in the Dialysis Unit of Hidaka Hospital (Gunma, Japan). Each patient underwent HD three times weekly (4 hours/day). Blood pressures (BP) were measured with a mercury sphygmomanometer with the patient in the supine position after 10 to 15 minutes of rest, and mean values for 1 month at enrollment were used for the analysis. This study was in compliance with the Declaration of Helsinki and was approved by the Institutional Review Board of Hidaka Hospital. All subjects gave their informed consent.

Inclusion criteria were patients aged 20 years and older, undergoing HD therapy for at least three months. All patients in this study had no history of previous CV diseases. Exclusion criteria were: recent history (less than six months) of acute myocardial infarction, percutaneous or surgical revascularization, unstable angina or cerebrovascular accident, decompensated congestive heart failure; severe valvular disease; pulmonary hypertension, BP > 160/110 mmHg, uncontrolled atrial fibrillation or complex ventricular arrhythmia, uncontrolled blood sugar levels, malignancies, active infection; irregular dialysis regimen; incapacity to obtain informed consent from the patient and inadequate echocardiographic window.

Patients were clinically evaluated and underwent a Doppler echocardiography during the period from March to December 2007, with an interval < 30 days between the two procedures. Subsequently, they were followed regularly until December 2013 or until the occurrence of outcome.

2.2. Doppler Echocardiogram

The echocardiograms were performed on echocardiography equipment, an Aplio XV (TOSHIBA, Tokyo, Japan) ultrasound imager equipped with a 2.2/4.4 MHz (harmonics) phased-array 3S transducer during continuous electrocardiographic recording as previously described [9]. The examinations were performed in the interdialytic period, within 24 hours after the dialysis session by a single medical professional, trained and skilled in echocardiography, with patients at rest and in left lateral decubitus position. Echocardiographic measurements followed the recommendations of the American Society of Echocardiography [10]-[12] and, for each variable, at least three cycles were analyzed.

The assessment of LV geometry was obtained by two-dimensional image, with the following variables: left ventricular end-diastolic diameter of (LVDD) and left ventricular end-systolic diameter (LVDS). The left ventricular mass (LVM) was calculated using the formula proposed by Devereux *et al*. [13] and then indexed to body surface area (BSA), to obtain the left ventricular mass index (LVMI = LVM/BSA). LV hypertrophy (LVH) was diagnosed when LVMI was >115 g/m^2 for men and >95 g/m^2 for women. The left ventricular ejection fraction (LVEF) was calculated by the method described by Teichholz *et al*. [14].

Mitral flow was measured in apical four-chamber view by pulsed Doppler. The sample was positioned between the distal ends of the mitral valve leaflets, and then the following variables were obtained: early (E) and late (A) transmitral diastolic velocities, E/A ratio. Tissue Doppler was performed in the apical four-chamber view to obtain the velocities of the mitral annulus. The sample was placed at the junction of the LV lateral wall

with the mitral annulus [15], and then early (e') diastolic velocities of the mitral annulus were identified, as well as the E/e' ratio.

LV dilatation was defined when the LVEDD was >59 mm for men and >53 mm for women. Systolic dysfunction was considered when the EF was <50%. LV diastolic function was classified into four patterns: normal, abnormal relaxation (mild diastolic dysfunction), pseudonormal (moderate diastolic dysfunction) and restrictive (severe diastolic dysfunction). It was considered abnormal relaxation when E/A < 1; restrictive pattern when E/A > 2 and pseudonormal pattern when E/A was >1 and <2 in association with E/e' > 10 [16].

2.3. Clinical and Laboratory Data

Clinical data, including age, gender, comorbidities and dialysis vintage were obtained from detailed analysis of medical records and interviews with the patient and the attending physician, when necessary. Before performing each Doppler echocardiogram, BP was measured and anthropometric data and ratios were obtained (weight, height, BSA, body mass index), which were measured according to standard procedures and using suitable materials. The body mass index (BMI) was calculated by dividing weight (kg) by squared height (m). The BSA was obtained using the formula of Dubois and Dubois [17]. All biochemical measurements were performed by a single laboratory, located in Hidaka Hospital, and data were collected from patients' charts.

2.4. Outcomes

The primary endpoint or CV mortality was defined by death from CV causes (including sudden death, myocardial infarction and cerebrovascular accidents). The secondary outcomes included overall mortality. Outcomes were obtained from monthly review of medical documentation, including medical records and death certificates, as well as communication with the physician and patient's relatives. Patients who underwent kidney transplant or who switched dialysis modality were censored in the study.

2.5. Statistical Analysis

Quantitative variables are reported as means ± SD or median and categorical variables as percentages. For comparison of proportions between groups with and without outcome, we used the Chi-square test and for comparison of quantitative variables, Student's *t*-test for independent samples. To estimate the hazard ratios (HR), we performed univariate analysis using Cox proportional hazards model and then, variables with $p < 0.10$ were included in the multivariate analysis using the same model. Survival curves were constructed using the Kaplan-Meier method and log-rank test was used to compare survival curves in univariate analysis. The significance level was defined as $p < 0.05$.

3. Results

The study population consisted of 83 (70.3%) men and 35 (29.7%) women, with a mean age of 62.5 ± 11.5 years, ranging from 21 to 76 years. The clinical, biochemical and echocardiographic characteristics of the population are listed in **Table 1**.

The cause of end-stage renal disease was attributed to chronic glomerulonephritis in 54 cases, diabetic nephropathy in 53 cases, hypertensive nephrosclerosis in 6 cases, polycystic kidney disease in 3 cases and other diseases in 2 cases.

The mean follow-up was 45.7 ± 13.6 months and during this period, there were twenty-eight deaths and thirteen cardiovascular deaths. CV diseases accounted for 46.4% of all deaths. Other causes of deaths were septic shock in five cases, malignancies in three cases, gastrointestinal bleeding in three cases and unknown origin in four cases. The event-free survival rates in one and two years were 96.5% and 83.0%, respectively.

Figure 1 shows the Kaplan-Meier curves for the occurrence of all-cause deaths in HD patients with (n = 53) and without diabetes (n = 65). There was no significant difference between the two groups. **Figure 2** shows the survival curves free of CV mortality in the two groups. The event-free survival rate in the diabetic group was significantly lower than that of the non-diabetic group ($p = 0.006$).

Table 2 shows the comparison of clinical and echocardiographic characteristics between groups with and without CV deaths. Patients with CV outcome had a higher prevalence of diabetes, higher values of BMI and serum non-HDL-cholesterol and triglyceride, and lower values of diastolic BP and high values of E/e'.

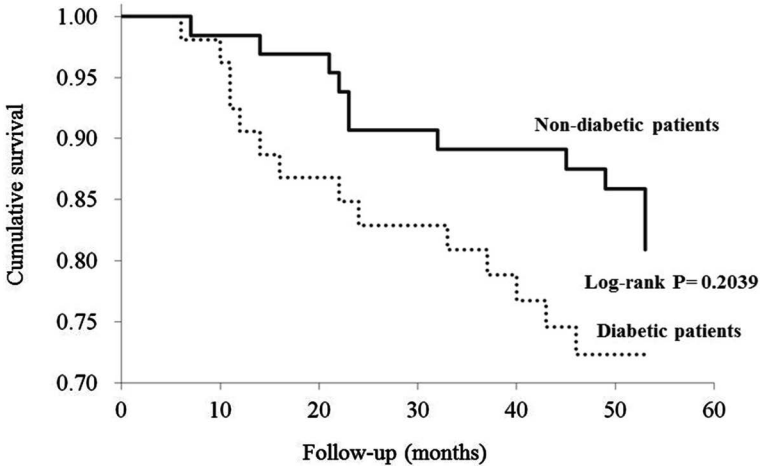

Figure 1. Kaplan-Meier curves for the occurrence of death in HD patients with (n = 53) and without diabetes (n = 65).

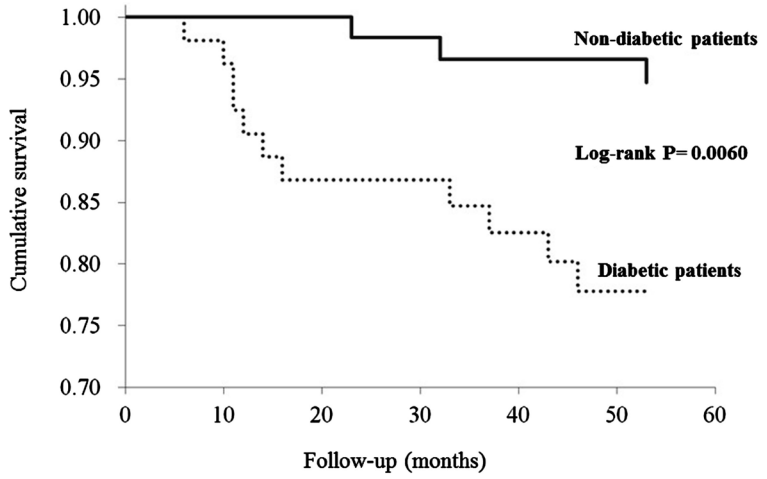

Figure 2. Kaplan-Meier curves for event-free survival from cardiovascular disease in HD patients with (n = 53) and without diabetes (n = 65).

The univariate Cox model for CV mortality is shown in **Table 3**. At univariate analysis, dialysis vintage, diabetes mellitus, BMI, serum levels of non-HDL-cholesterol and triglyceride, and E/e' ratio were significantly associated with CV mortality. Multivariate analysis included variables: dialysis vintage, diabetes, systolic and diastolic BP, BMI, HDL- and non-HDL-cholesterol, triglyceride and E/e' ratio. In the final regression model, dialysis vintage, diabetes, systolic BP and E/e' showed themselves to be independent risk factors for CV mortality (**Table 4**).

4. Discussion

This study showed free survival rates of overall mortality of 94.9% in one year and 87.2% in two years. These rates are comparable to those found by Siqueira *et al*. [18], who found survival rates of 96.5% and 83.0% in one and two years, respectively. Data from dialysis censuses show survival rates of 88.6% in one year and 79.1% in two years in Europe [19] and 77.4% in one year and 63% in two years in the USA [1]. The patient characteristics included in this study could explain the differences found. We studied patients without history of previous CV diseases, which is uncommon in this population. Furthermore, the average LVEF was preserved in the subjects studied, characterizing a population of more stable patients from the point of view of LV systolic function.

CVD accounted for 46.4% of all deaths during follow-up period. The proportion found is similar to those reported in studies on all-cause and CV mortality in HD patients [2] [6]. However, multicenter studies, such as the

Table 1. Clinical and echocardiographic characteristics of the study population.

Characteristics	Value (n = 118)
Age (years)	62.5 ± 11.5
Male gender (%)	83 (70.3%)
Dialysis vintage (months)	79 (14 - 131.4)
Diabetes (%)	53 (44.9%)
Systolic BP (mmHg)	146.3 ± 17.8
Diastolic BP (mmHg)	75.0 ± 10.4
BMI (kg/m^2)	21.6 ± 3.9
Albumin (g/dL)	3.6 ± 0.3
Hemoglobin (g/dL)	10.4 ± 0.9
Total cholesterol (mg/dL)	147.6 ± 31.0
HDL cholesterol (mg/dL)	45.1 ± 12.0
Non-HDL cholesterol (mg/dL)	102.5 ± 31.6
Triglyceride (mg/dL)	112.2 ± 83.8
Calcium (mg/dL)	8.7 ± 0.6
Phosphorus (mg/dL)	5.2 ± 1.1
Intact-PTH (pg/mL)	220.0 ± 162.5
LVDD (mm)	46.9 ± 5.6
LVEF (%)	66.8 ± 10.7
LAD (mm)	38.5 ± 6.7
LVMI (g/m^2)	109.4 ± 32.5
E/A	0.8 ± 0.3
E/e'	15.3 ± 6.2

Data are expressed as mean ± standard deviation, median (range) or percentage. BP, blood pressure; BMI, body mass index; HDL, high-density lipoprotein; PTH, parathyroid hormone; LVDd, left ventricular end-diastolic diameter; EF, ejection fraction; LAD, left atrial diameter; LVMI, left ventricular mass index; E, early diastolic transmitral velocity; A, late diastolic transmitral velocity; e', early diastolic velocity of mitral annulus.

Table 2. Comparison of clinical and echocardiographic characteristics according to the presence of cardiovascular outcome.

Characteristics	Cardiovascular mortality		p-value
	No (n = 105)	Yes (n = 13)	
Age (years)	62.5 ± 11.4	61.9 ± 12.7	0.8554
Male sex (%)	72.1	57.1	0.2495
Dialysis vintage (months)	126.9 ± 150.6	61.6 ± 41.2	0.1107
Diabetes (%)	40.4	78.6	0.0070
Systolic BP (mmHg)	147.4 ± 16.7	138.2 ± 24.0	0.0692
Diastolic BP (mmHg)	75.7 ± 10.4	69.7 ± 9.3	0.0431
BMI (kg/m^2)	21.2 ± 2.8	24.1 ± 8.1	0.0084
Albumin (g/dL)	3.7 ± 0.3	3.6 ± 0.3	0.1954
Hemoglobin (g/dL)	10.4 ± 1.0	10.4 ± 0.8	0.8992
Total cholesterol (mg/dL)	146.0 ± 30.9	159.7 ± 30.0	0.1199
HDL cholesterol (mg/dL)	45.8 ± 12.0	39.5 ± 10.9	0.0653
Non-HDL cholesterol (mg/dL)	100.2 ± 31.1	120.2 ± 29.9	0.0249
Triglyceride(mg/dL)	104.4 ± 70.1	170.5 ± 141.7	0.0051
Calcium (mg/dL)	8.8 ± 0.7	8.5 ± 0.5	0.1590
Phosphorus (mg/dL)	5.2 ± 1.1	4.8 ± 0.8	0.1460
Intact-PTH (pg/mL)	223.2 ± 168.1	196.4 ± 115.0	0.5654
LVDD (mm)	46.8 ± 5.6	47.8 ± 5.2	0.5435
LVEF (%)	66.8 ± 9.9	66.4 ± 15.9	0.8760
LAD (mm)	38.2 ± 5.8	40.4 ± 11.5	0.2631
LVMI (g/m^2)	108.5 ± 32.1	116.0 ± 36.0	0.4200
E/A	0.8 ± 0.3	0.9 ± 0.4	0.2024
E/e'	14.9 ± 5.3	18.6 ± 10.0	0.0317

Table 3. Predictors of cardiovascular mortality by univariate analysis of Cox regression model.

Characteristics	HR	95% CI	p
Age (years)	0.995	0.954 - 1.043	0.8144
Male sex (%)	0.555	0.193 - 1.687	0.2862
Dialysis vintage (months)	0.990	0.978 - 0.998	0.0152
Diabetes (%)	5.017	1.565 - 22.193	0.0055
Systolic BP (mmHg)	0.974	0.945 - 1.004	0.0830
Diastolic BP (mmHg)	0.963	0.933 - 1.005	0.0791
BMI (kg/m^2)	1.142	1.030 - 1.237	0.0147
Albumin (g/dL)	0.234	0.034 - 1.698	0.1506
Hemoglobin (g/dL)	1.038	0.578 - 1.710	0.8918
Total cholesterol (mg/dL)	1.012	0.996 - 1.028	0.1318
HDL cholesterol (mg/dL)	0.956	0.911 - 1.001	0.0533
Non-HDL cholesterol (mg/dL)	1.018	1.002 - 1.033	0.0289
Triglyceride(mg/dL)	1.006	1.002 - 1.009	0.0111
Calcium (mg/dL)	0.583	0.264 - 1.284	0.1796
Phosphorus (mg/dL)	0.676	0.374 - 1.151	0.1556
Intact-PTH (pg/mL)	0.999	0.994 - 1.002	0.6106
LVDD (mm)	1.033	0.939 - 1.132	0.4979
LVEF (%)	0.992	0.945 - 1.047	0.7541
LAD (mm)	1.054	0.970 - 1.141	0.2118
LVMI (g/m^2)	1.006	0.991 - 1.021	0.4082
E/A	2.911	0.531 - 12.011	0.2005
E/e'	1.092	1.009 - 1.172	0.0301

Table 4. Predictors of cardiovascular mortality by multivariate analysis of Cox regression model.

Characteristics	HR	95% CI	p
Dialysis vintage (months)	0.989	0.975 - 0.999	0.0187
Diabetes (%)	7.230	1.553 - 45.036	0.0107
Systolic BP (mmHg)	0.956	0.898 - 0.997	0.0368
Diastolic BP (mmHg)	0.951	0.889 - 1.074	0.3375
BMI (kg/m^2)	1.056	0.934 - 1.181	0.3591
HDL cholesterol (mg/dL)	0.961	0.901 - 1.020	0.1976
Non-HDL cholesterol (mg/dL)	1.023	0.998 - 1.051	0.0768
Triglyceride(mg/dL)	0.999	0.991 - 1.005	0.7132
E/e'	1.100	1.011 - 1.202	0.0267

HEMO and AURORA studies, showed rates of less than 50% of mortality due to CV disease in the dialysis population [19] [20]. These results confirm that CV diseases remain the leading cause of mortality in HD patients. HD patients have a 10 - 20-fold higher risk of CV mortality compared with the general population [21] and this is due to the fact that HD patients are exposed to both traditional as well as non-traditional factors for CV complications [22].

The results of this study suggested that diabetes is related to a higher risk of CV mortality, as previously described [23]. CV disease in diabetic patients is usually attributed to coronary artery disease resulting from an accelerated process of atherosclerosis [24]. Diabetic patients are also more prone to develop congestive heart failure, independently of the presence of coronary artery disease or hypertension [25]. This is probably due to the presence of diabetic cardiomyopathy; there is substantial experimental, pathological, epidemiological, and clinical evidence to support the existence of specific abnormalities in cardiac structure and function in diabetic patients [26].

We have shown that LV diastolic dysfunction is associated with higher CV mortality of HD patients in this study. Nardi *et al*. evaluated how diabetes affected LV geometry and diastolic function in 288 hypertensive patients with chronic kidney disease [27]. They found that diabetes together with renal dysfunction was associated with a worse diastolic function with significantly higher E/e'. Hung *et al*. have recently showed that diabetic HD patients had a more profound LV diastolic dysfunction, as estimated E/e' [28]. The mechanisms and evolution of

diastolic dysfunction are not completely understood, especially in diabetic patients. Several factors affect LV function and transmitral flow: preload, after load, heart rate, LV mass, metabolic and hormonal parameters, myocardial innervation, and microangiopathic lesions [29]. The increased proportion of diabetic patients with moderate or severe LV diastolic dysfunction was not related to the presence of myocardial ischemia or hypertension, because the frequency of these factors was not higher in diabetic HD patients [30].

LV diastolic dysfunction combines relaxation and compliance abnormalities. As a result of poor compliance, a slight increase in preload can induce a sharp increase in LV pressure, leading to congestion. On the other hand, a small increase in filling pressure can reduce systolic ejection volume and cardiac output. These abnormalities might predispose diabetic HD patients to hemodynamic instability and hypotension [31]. Myocardial fibrosis resulting from these processes is a major determinant of LV stiffness and elevated filling pressures, predisposing to the development of LV diastolic dysfunction [32]. In a recently published study, which evaluated 211 HD patients and used conventional Doppler and tissue Doppler criteria to classify diastolic dysfunction, Quiroqa *et al*. [33] have shown that LV diastolic dysfunction is an emergent risk factor for death and CV events in HD patients.

The present study has several limitations. First, the number of patients studied was small and highly selective. Most of the patients in this study had normal LVEF despite six patients that had LVEF below 50%. Patients with normal LVEF without structural heart diseases are difficult to identify, especially HD patients. Additionally, Doppler techniques are highly dependent on investigator. In this study, echocardiographic parameters were recorded by a specialist according to the guideline of American society of Echocardiography. However, this technique allows periodic analysis in patients who have high rates of CV events.

5. Conclusion

Chronic HD patients have high rates of CV mortality. The presence of diabetes and high E/e' are factors potentially related to CV mortality. LV diastolic dysfunction was an independent risk factor for CV mortality, and although further studies are necessary to validate this finding, it is recommended that LV diastolic function evaluation, made through pulsed Doppler and tissue Doppler parameters, included in the evaluation of HD patients. This measure will enable the early detection of HD patients at risk in order to reduce mortality.

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Conflict of Interest

The authors have declared that no conflict of interest exists.

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