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ORIGINAL ARTICLE

Interdialytic blood pressure variability and all-cause mortality in patients undergoing maintenance hemodialysis: a multicenter study using DialysisNet

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Abstract

Background/aims In this study, we aimed to analyze all-cause mortality according to interdialytic blood pressure variability (BPV) in patients undergoing hemodialysis.

Methods Data on predialysis blood pressure (BP) and clinical information were extracted from four dialysis units through the DialysisNet system, which enables efficient hemodialysis management using common data elements. Interdialytic BPV was evaluated as the coefficient of variation (CV) of predialysis BP at each dialysis session over a 12-month period. The CV of systolic BP (SBP) and diastolic BP (DBP) was divided into tertiles. The primary outcome was all-cause mortality according to the CV of predialysis SBP, which was analyzed using Cox regression analysis.

Results The data of 357 patients undergoing hemodialysis were analyzed. Compared with the first SBP CV tertile, the third tertile showed significantly increased all-cause mortality after adjustment (hazard ratio [HR], 2.11; 95% confidence interval [CI] 1.04–4.24). Compared with the first DBP CV tertile, the third tertile showed significantly increased mortality in univariable analysis (HR, 2.18; 95% CI 1.10–4.30) but not in multivariable analysis (HR, 1.88; 95% CI 0.89–3.95).

Conclusions Increased interdialytic BPV in patients undergoing hemodialysis is associated with all-cause mortality. This was more prominent in SBP than in DBP. Particular attention should be paid to large BPVs in older adults, women, and patients with a relatively longer dialysis vintage.

Keywords Blood pressure · Blood pressure variability · Dialysis · Hemodialysis · Mortality

Introduction

High blood pressure (BP) is a leading risk factor for cardiovascular diseases [1, 2]. In particular, in patients with chronic kidney disease, who have many other risk factors, hypertension is a major contributor to the development of cardiac, cerebral, and vascular complications [1]. Traditionally, the goal of BP management has been to control mean BP, which is achieved by office BP measurement.

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However, the importance of BP variability (BPV) has been increasingly emphasized recently [3–7].

BP results from a complex interplay of environmental, physical, and emotional factors, and cardiovascular regulatory mechanisms attempt to maintain BP homeostasis to ensure adequate organ perfusion. The size and patterns of these BP changes are defined as BPV [8]. BPV is divided into very short-term (beat-to-beat), short-term (within 24 h), mid-term (day-to-day), and long-term (visit-to-visit) categories based on the measurement interval [9], with each category having different mechanisms. Short-term BPV can be influenced by autonomic modulation, arterial compliance, and the effect of humoral, rheological, and emotional factors. Long-term variability is less well understood; however, arterial stiffness may play a role [10]. Previous studies have shown that visit-to-visit variability has a greater impact on mortality and cardiovascular outcomes in the general population [6, 7].

In patients undergoing hemodialysis (HD), BP is difficult to control because it can be influenced by various pathophysiologies, including excess sodium level and volume, the renin–angiotensin–aldosterone and sympathetic nervous systems, and changes in the arterial and venous vasculature [11–13]. Previous studies have reported outcomes based on visit-to-visit BPV in patients undergoing dialysis. In the HEMO study cohort, higher visit-to-visit variability in predialysis systolic BP (SBP) was associated with a higher risk of all-cause mortality [14]. In another study, higher visit-to-visit variability in predialysis SBP was associated with an increased risk of stroke [15].

The DialysisNet system is a dialysis center management program for doctors based on the Health Avatar Platform, designed for managing patients undergoing dialysis [16]. DialysisNet can function as a common data registry that enables real-time, multicenter data collection. Patients undergoing HD usually visit the dialysis unit repeatedly at intervals of 2–3 days. Therefore, dialysis centers need to monitor patients' BP, and DialysisNet facilitates the extraction of serially repeated data. In this study, we aimed to examine the association between visit-to-visit long-term BPV (interdialysis BPV) and all-cause mortality in patients undergoing HD in several HD centers using multicenter HD patient data obtained through DialysisNet.

Methods

Compliance with standard models and development of a multicenter trial concept in DialysisNet

To effectively manage HD data, we identified common data elements for HD information (CDEHI). The CDEHI metadata were parsed into the metadata registry on the Health Avatar Care Platform based on the representational state transfer (REST) protocol. The DialysisNet system can be plugged into and played on the Care Platform. The CDEHI metadata in DialysisNet were exchanged and created as a real-time registry for HD information based on the Continuity of Care Record standards [16]. Details of the DialysisNet design have been described previously [16–18].

Study design and data collection

The DialysisNet system was used to obtain information on patients undergoing HD from a multicenter study. A total of 357 adult patients undergoing maintenance HD were recruited from four dialysis centers between January and December 2016. Participants were adult patients aged 18 years or older who underwent maintenance HD at the institution for ≥ 8 months, ≥ 2 times per week, and ≥ 8 times per month. Hospitalized patients and in-hospital HD data were excluded. Baseline demographic data, including age, sex, underlying cause of end-stage kidney disease, comorbidities, and anthropometric measurements, were recorded at enrollment. Blood samples were collected before the start of each HD session, typically at 2-day intervals, following the standardized protocols of each participating center. BP measured before the start of each HD session was collected at each center over a period of 1 year. At each regular HD visit, predialysis SBP and diastolic BP (DBP) were measured by trained dialysis nurses in accordance with the practice protocol of each dialysis center. The measurements were made on the upper arm opposite the HD vascular access after the patient had rested for approximately 5 min upon arriving at the dialysis unit and before the initiation of HD. This study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was approved by the Institutional Review Board of each participating center. Informed consent was waived due to the retrospective design of the study.

BPV

BPV was assessed as visit-to-visit BPV (interdialysis BPV). Interdialysis BPV was evaluated as the coefficient of variation (CV) of predialysis BP at each HD session over a 12-month period. The CV was calculated as the standard deviation (SD) divided by the mean.

Study outcome

The primary outcome was all-cause mortality until August 2019, according to the CV of predialysis BP. The CV of predialysis SBP was divided into tertiles, and mortality was

compared. We further analyzed all-cause mortality using the CV of predialysis DBP.

Statistical analysis

Data were expressed as mean ± SD or median (interquartile range) for continuous variables and as number (percentage) for categorical variables. Baseline characteristics and laboratory findings were compared using analysis of variance or the Kruskal–Wallis test for continuous variables and the chi-square test for categorical variables. CV was divided into tertiles according to predialysis SBP and DBP. The first tertile (T1) was used as the reference group, and the all-cause mortality in each group was compared. Cumulative survival curves for all-cause mortality were generated using the Kaplan–Meier method, and between-group survival was compared using the log-rank test. A Cox proportional hazards regression model was used to assess the independent relationship between CV and all-cause mortality. The analyses were adjusted for baseline confounders, including age, sex, body mass index (BMI), underlying comorbidities, and laboratory tests. Three models were designed, and the results of the Cox analysis are presented as hazard ratio (HR) and 95% confidence intervals (CIs). Changes in HR based on the CV were analyzed using a restricted cubic spline curve. Subgroup analysis was performed, and subgroups were defined by age (< 65 versus ≥ 65 years), sex (male vs. female), diabetes mellitus (DM) status (DM vs. non-DM), hypertension (HTN) status (HTN vs. non-HTN), and dialysis vintage (< 24 vs. ≥ 24 months). Dialysis vintage was divided by the median value for all patients. Statistical analyses were performed using SPSS for Windows (version 27.0; IBM Corp., Armonk, NY, USA) and R software (version 4.3.1; R Project for Statistical Computing, Vienna, Austria).

Results

Baseline characteristics

The baseline demographic and clinical characteristics of the 357 patients according to SBP CV tertiles are presented in Table 1. The mean age was 62.9 ± 13.2 years, 198 (55.5%) were male, and the median dialysis vintage was 24 (interquartile range: 5–68) months. In total, 206 (57.7%) patients had DM and 284 (79.6%) had HTN as an underlying condition. The mean predialysis SBP and DBP were 144.6 ± 16.4 mmHg and 72.5 ± 10.8 mmHg, respectively. The CV ranges for the T1, second tertile (T2), and third tertile (T3) were 0.06 to 0.10, 0.11 to 0.12, and 0.13 to 0.19, respectively. There was no significant difference in mean predialysis SBP and DBP among the three groups. Patients in the T3 group were older ($P = 0.010$), had a higher prevalence of underlying DM ($P = 0.017$), and had lower serum albumin levels ($P = 0.010$) than those in the T1 group. The baseline demographic and clinical characteristics, according to the tertiles of the CV values of DBP, are presented in Supplementary Table S1. Patients in the T3 group were predominantly female ($P = 0.008$) and had lower serum albumin levels ($P = 0.043$), with no significant differences in other characteristics between the groups.

Primary outcome

During a median follow-up of 44.0 (31, 44) months, 55 (15.4%) of 357 patients died, with the most common cause of death being infection (n = 26 [47.3%]), followed by cardiovascular disease (n = 15 [27.3%]) and malignancy (n = 6 [10.9%]) (Table 2). There were no significant differences in causes of death according to the CV tertiles. Regarding the CV of SBP, the numbers of patients who died in the T1, T2, and T3 groups were 14 (10.3%), 16 (15.4%), and 25 (21.4%), respectively. Kaplan–Meier analysis showed that the T3 group had a higher all-cause mortality than the other groups (log-rank P-value = 0.031; Fig. 1). The three models of the multivariable Cox analysis revealed that the T3 group had a significantly higher mortality risk than the T1 group (HR, 2.11; 95% CI 1.04 to 4.24; $P = 0.037$; Table 3). Figure 2 shows the restricted cubic spline curve for the HR of all-cause mortality according to the CV values of SBP obtained using multivariable Cox regression analysis. Even after further adjustment for the SBP mean value in Model 3, the T3 group still had a significantly higher mortality risk than the T1 group (HR, 2.34; 95% CI 1.16 to 4.72; $P = 0.018$).

In the CV tertile groups, according to DBP, the numbers of patients who died in the T1, T2, and T3 groups were 13 (9.6%), 19 (19.4%), and 23 (18.7%), respectively. There were no significant differences in causes of death according to the DBP CV tertiles (Supplementary Table S2). Kaplan–Meier analysis showed that the T1 group had a lower all-cause mortality than the other groups (log-rank P-value = 0.032; Fig. 3). In the Cox regression analysis, the T2 (HR, 2.32; 95% CI 1.14 to 4.69; $P = 0.020$) and T3 (HR, 2.18; 95% CI, 1.10 to 4.30; $P = 0.025$) groups had a higher all-cause mortality than the T1 group in the univariable analysis. However, this trend was not significant in the multivariable analysis (Table 4).

Subgroup analysis

The all-cause mortality results of the subgroup analyses for SBP variability are shown in Table 5. There was no significant interaction between SBP variability and each subgroup in terms of mortality outcomes. However,

Table 1 Baseline characteristics according to SBP CV tertiles

Characteristics	All	CV Tertiles of SBP			*P* value
		T1	T2	T3	
		0.06–0.10	0.11–0.12	0.13–0.19	
	N = 357	(*n* = 136)	(*n* = 104)	(*n* = 117)	
Age, mean ± SD	62.9 ± 13.2	60.2 ± 13.3	64.4 ± 12.9	64.7 ± 12.9	0.010
Sex, male, *n* (%)	198 (55.5)	81 (59.6)	59 (56.7)	58 (49.6)	0.268
Primary cause of CKD, *n* (%)					0.482
DM	187 (52.4)	61 (44.9)	55 (52.9)	71 (60.7)	
GN	39 (10.9)	16 (11.8)	12 (11.5)	11 (9.4)	
PKD	9 (2.5)	4 (2.9)	2 (1.9)	3 (2.6)	
HTN	70 (19.6)	33 (24.3)	18 (17.3)	19 (16.2)	
Others	52 (14.6)	22 (16.2)	17 (16.3)	13 (11.1)	
Dialysis vintage, median, (Q1, Q3), month	24 (5, 68)	35 (8, 72)	17 (1, 59)	18 (2, 70)	0.018
BMI, kg/m^2	22.4 ± 4.0	22.1 ± 4.3	22.3 ± 3.9	22.7 ± 3.8	0.514
SBP, mmHg	144.6 ± 16.4	145.5 ± 17.3	145.6 ± 17.0	142.7 ± 14.9	0.308
DBP, mmHg	72.5 ± 10.8	74.2 ± 11.5	71.4 ± 10.8	71.5 ± 9.7	0.067
Comorbidities, *n* (%)					
DM	206 (57.7)	66 (48.5)	63 (60.6)	77 (65.8)	0.017
HTN	284 (79.6)	108 (79.4)	82 (78.8)	94 (80.3)	0.962
Laboratory test					
WBC, cells/mm^3	6700 ± 2600	6400 ± 2200	6500 ± 2300	7200 ± 3100	0.021
Hemoglobin, g/dL	10.4 ± 1.5	10.5 ± 1.3	10.1 ± 1.4	10.4 ± 1.8	0.148
Albumin, g/dL	3.8 ± 0.4	3.9 ± 0.4	3.8 ± 0.4	3.7 ± 0.5	0.010
Phosphorus, mg/dL	4.7 ± 1.6	4.8 ± 1.6	4.6 ± 1.5	4.5 ± 1.6	0.261
Corrected calcium, mg/dL	9.0 ± 0.7	9.1 ± 0.7	8.9 ± 0.7	9.0 ± 0.8	0.257

Categorical variables are reported as numbers (percentages), and continuous variables are reported as mean ± standard deviation

CV coefficient of variation, *CKD* chronic kidney disease, *DM* diabetes mellitus, *GN* glomerulonephritis, *PKD* polycystic kidney disease, *HTN* hypertension, *BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *WBC* white blood cell

Table 2 Cause of death according to SBP CV tertiles

Cause of death, *n* (%)	Total	T1	T2	T3	*P* value
Total	55 (15.4)	14 (10.3)	16 (15.4)	25 (21.4)	0.052
Cardiovascular	15 (27.3)	7 (50.0)	4 (25.0)	4 (16.0)	0.126
Infection	26 (47.3)	5 (35.7)	10 (62.5)	11 (44.0)	
Malignancy	6 (10.9)	0 (0)	1 (6.3)	5 (20.0)	
Others	6 (10.9)	2 (14.3)	0 (0)	4 (16.0)	
Unknown	2 (3.6)	0 (0)	1 (6.3)	1 (4.0)	

CV coefficient of variation, *SBP* systolic blood pressure

when stratified and analyzed within each subgroup, the results were as follows. The HR for mortality in the SBP T3 group, compared with the T1 group, significantly increased in those aged ≥ 65 years (HR, 2.38; 95% CI 1.05 to 5.38; *P*=0.038), women (HR, 3.81; 95% CI 1.01 to 14.38; *P*=0.049), patients without DM (HR, 4.21; 95% CI 1.06 to 16.73; *P*=0.041), and those with dialysis vintage ≥2 years (HR, 2.94; 95% CI 1.14 to 7.60; *P*=0.026). Subgroup analyses of DBP showed no differences in mortality among the subgroups (Supplementary Table S3).

Discussion

In the present study, patients in the third SBP CV tertile were older, had a higher prevalence of DM, and had lower serum albumin levels. Compared with the first SBP CV tertile, the T3 showed significantly increased all-cause mortality after adjustment. Subgroup analysis showed higher mortality in older adults, women, patients without DM, and those with longer dialysis vintage in the T3 group. In the analysis of predialysis DBP, all-cause mortality significantly increased in the T2 and T3 in the univariable analysis; however, the difference was not significant after adjustment in the multivariable analysis.

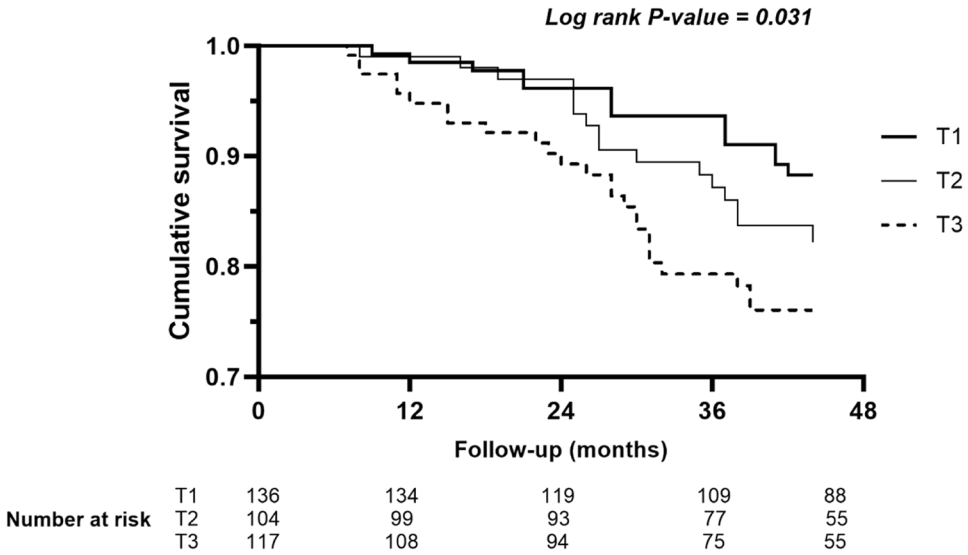


Fig. 1 Kaplan–Meier survival curve for all-cause mortality according to SBP CV tertiles. *CV* coefficient variation, *SBP* systolic blood pressure

Table 3 All-cause mortality according to SBP CV tertiles

	CV tertile of SBP	Events, *n* (%)	Model 1		Model 2		Model 3	
			HR (95% CI)	*P* value	HR (95% CI)	*P* value	HR (95% CI)	*P* value
All-cause mortality	T1	14 (10.3)	Reference		Reference		Reference	
	T2	16 (15.4)	1.56 (0.76, 3.21)	0.222	1.13 (0.52, 2.48)	0.757	1.05 (0.47, 2.33)	0.902
	T3	25 (21.4)	2.34 (1.22, 4.50)	0.011	2.22 (1.10, 4.45)	0.026	2.11 (1.04, 4.24)	0.037

Model 1: Unadjusted

Model 2: Adjusted for age, sex, BMI, DM, HTN

Model 3: Model 2 + adjusted for hemoglobin, albumin, phosphorus

CV coefficient of variation, *SBP* systolic blood pressure, *HR* hazard ratio, *CI* confidence interval, *BMI* body mass index, *DM* diabetes mellitus, *HTN* hypertension

Since the 2010 study on visit-to-visit BPV and stroke risk [6], many studies have been conducted on BPV in the general population. In various studies, increased BPV was associated with increased all-cause mortality and cardiovascular events, and this association was more evident with long-term than with short-term BPV [3, 4, 7, 9]. There have been various studies on BPV in patients undergoing HD and the general population. According to a previous meta-analysis published in 2021, interdialytic BPV is used to represent long-term BPV in patients undergoing HD and is associated with all-cause and cardiovascular mortality [19]. A previous study showed that short-term BPV could be a mediator promoting an adverse cardiovascular profile [20]. However, most studies have primarily focused on interdialytic systolic BPV, with higher BPVs being associated with increased all-cause mortality [7, 14, 20–23], increased cardiovascular events [7, 14, 22], and increased stroke risk [15]. Our finding that higher SBP variability was associated with higher all-cause mortality is similar to those of previous studies. However, DBP did not show a clear relationship, and previous studies have reported inconsistent results. Some studies on patients undergoing dialysis have reported that increased DBP variability is associated with an increased risk of all-cause mortality [19, 21] and stroke [15], whereas others have found no relationship [23]. In a study of cardiovascular outcomes in patients with DM, the risk was higher with increasing DBP variability, especially when the variability was below the mean pressure ("dips"). This may be related to diastolic coronary hypoperfusion [5]. In this respect, for DBP in patients undergoing HD, interdialytic variations in DBP may be less influential than intradialytic BP changes.

Patients undergoing HD visit the dialysis unit two to three times a week. Because BP monitoring is essential for patients undergoing HD, BP is repeatedly measured during every dialysis session. Many previously reported studies have been based on < 6 months of data [23, 24]. Using DialysisNet, repeated real-time BP data can be accumulated

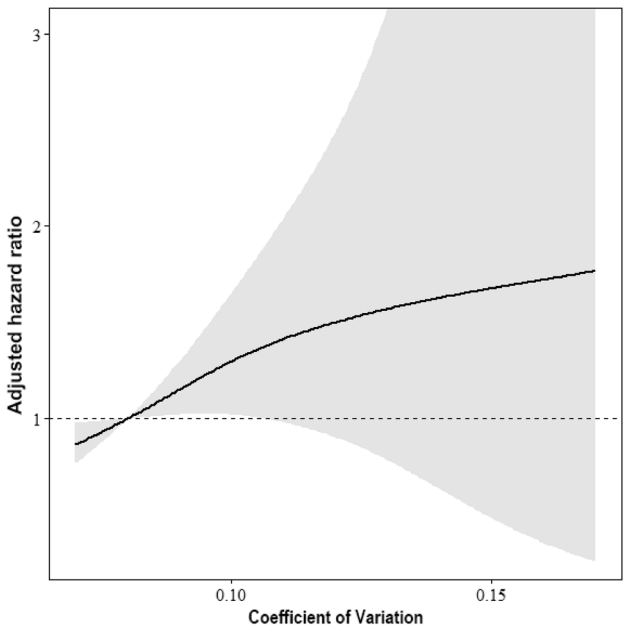


Fig. 2 The restricted cubic spline curve for the adjusted hazard ratio according to the CV of SBP using multivariable Cox regression analysis. *CV* coefficient variation, *SBP* systolic blood pressure

and collected continuously. The present study was conducted by collecting long-term, multicenter consecutive BP data for 1 year using DialysisNet. This continuously collected data, obtained through DialysisNet, was evaluated for long-term BPV, including seasonal changes. Previous studies have also analyzed long-term changes using serial laboratory results (hemoglobin, potassium, and others) from patients undergoing HD obtained through DialysisNet [17, 18]. In the present study, continuously collected data obtained through such a multicenter common data model are expected to be more reflective of actual patient BPV than sporadically collected data.

During HD, patients are exposed to rapid fluid and osmolality shifts that cause the BP to fluctuate [15], which affects intradialytic BPV. The key causes of increased BP during the interdialytic interval may be weight gain and increased peripheral resistance due to excessive activity of the sympathetic nervous system and vascular calcification [11]. In the present study, patients in the third tertile were older and had diabetes, which may be associated with vessel stiffness. BPV may increase under diabetic conditions, similar to the results of previous studies [14, 23]. Hyperglycemia increases the

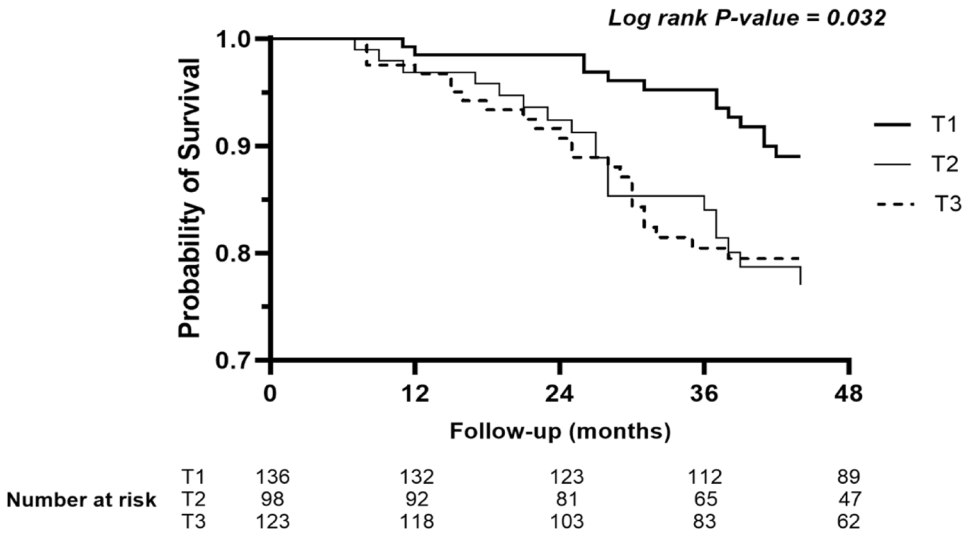


Fig. 3 Kaplan–Meier survival curve for all-cause mortality according to DBP CV tertiles. *CV* coefficient variation, *DBP* diastolic blood pressure

Table 4 All-cause mortality according to DBP CV tertiles

	CV tertile of DBP	Events, *n* (%)	Model 1		Model 2		Model 3	
			HR (95% CI)	*P* value	HR (95% CI)	*P* value	HR (95% CI)	*P* value
All-cause mortality	T1	13 (9.6)	Reference		Reference		Reference	
	T2	19 (19.4)	2.32 (1.14, 4.69)	0.020	1.88 (0.89, 3.97)	0.097	1.71 (0.80, 3.66)	0.168
	T3	23 (18.7)	2.18 (1.10, 4.30)	0.025	1.78 (0.86, 3.68)	0.121	1.88 (0.89, 3.95)	0.096

Model 1: Unadjusted

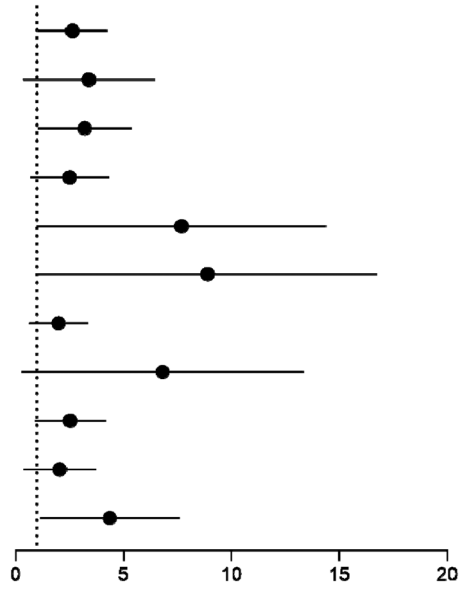
Model 2: Adjusted for age, sex, BMI, DM, HTN

Model 3: Model 2 + adjusted for hemoglobin, albumin and phosphorus

CV coefficient variation, *DBP* diastolic blood pressure, *HR* hazard ratio, *CI* confidence interval, *BMI* body mass index, *DM* diabetes mellitus, *HTN* hypertension

Table 5 Subgroup analysis of all-cause mortality according to SBP CV tertiles

Subgroup	Events, *n* (%)	Unadjusted HR(95% CI)	Adjusted HR (95% CI)	Adjusted HR (95% CI)	*P* for interaction
Overall	25 (21.4)	2.34 (1.22, 4.50)	2.11 (1.04, 4.24)		
Age < 65	5 (9.8)	1.62 (0.47, 5.59)	1.52 (0.36, 6.45)		0.611
Age ≥ 65	20 (30.3)	2.21 (1.01, 4.85)	2.38 (1.05, 5.38)		
Male	12 (20.7)	1.68 (0.74, 3.80)	1.74 (0.70, 4.33)		0.269
Female	13 (22.0)	4.76 (1.36, 16.73)	3.81 (1.01, 14.38)		
DM (-)	9 (22.5)	4.81 (1.48, 15.64)	4.21 (1.06, 16.73)		0.269
DM (+)	16 (20.8)	1.44 (0.65, 3.17)	1.47 (0.64, 3.37)		
HTN (-)	4 (17.4)	2.33 (0.43, 12.73)	1.96 (0.29, 13.35)		0.836
HTN (+)	21 (22.3)	2.32 (1.14, 4.72)	1.94 (0.90, 4.18)		
Vintage < 2 years	10 (15.9)	1.82 (0.41, 4.09)	1.21 (0.39, 3.73)		0.523
Vintage ≥ 2 years	15 (27.8)	2.97 (1.30, 6.80)	2.94 (1.14, 7.60)		



HR is the result of the T3 group compared with the T1 group

The model was adjusted for age; sex; BMI; underlying comorbidities (DM and HTN); and albumin, hemoglobin, and phosphorus levels

HR, hazard ratio; CI, confidence interval; DM, diabetes mellitus; HTN, hypertension; BMI, body mass index

activity of the local renin-angiotensin system and the expression of angiotensin receptors on the vascular wall, leading to hypertrophy and stiffening of the arterial wall [25]. The prevalence of hypertension increases with age; in particular, the increase in SBP compared with DBP is remarkable due to arterial stiffening [26, 27].

In the present study, the highest tertile of SBP variability was associated with increased all-cause mortality compared with the lowest tertile. DBP variability was associated with higher all-cause mortality in the univariable analysis; however, this association was not statistically significant in the multivariable model. In previous animal studies, rats undergoing sinoaortic denervation exhibited significant increases in BPV without significant changes in mean BP, leading to biventricular hypertrophy and increased atherogenesis, possibly due to impaired endothelin-dependent relaxation [28–30]. These findings suggest that BPV can influence clinical outcomes. SBP variability primarily reflects arterial stiffness and impaired baroreflex function, which are strongly associated with left ventricular hypertrophy, heart failure, and, ultimately, higher mortality risk. In contrast, DBP variability may impact coronary perfusion and reflect peripheral vascular resistance; however, its effect on clinical outcomes appears less pronounced than that of SBP variability. Notably, previous studies have reported inconsistent associations between DBP variability and adverse outcomes, whereas SBP variability has shown a more robust and consistent relationship with mortality. The actual mechanisms underlying these differences remain unclear; however, our results align with those of existing literature, supporting the notion that SBP variability has a stronger impact on mortality outcomes than DBP variability in patients undergoing HD.

In the subgroup analyses, mortality according to BPV was higher in female patients and those with a relatively longer dialysis vintage. The sex difference may be due to differences in arterial wall stability, which can affect interdialytic BPV. Estrogen is involved in the production of elastin and collagen, which affect arterial wall remodeling. Older women, especially postmenopausal women, have higher aortic stiffness and pulsatile arterial load than men, which appear to play a role in the predominance of isolated systolic hypertension, uncontrolled hypertension, and heart failure with preserved ejection fraction [31]. Arterial stiffness can increase with dialysis vintage [32]. In patients with decreased renal function, chronic inflammation, oxidative stress, and various endocrine abnormalities contribute to the development of arterial stiffness [33, 34]. In particular, increased arterial stiffness has been reported in patients undergoing HD compared with patients not undergoing HD [34]. Volume overload during HD may directly or indirectly cause endothelial dysfunction, thereby affecting arterial rigidity and left ventricular hypertrophy [35]. In the present study, subgroup analysis revealed that non-diabetic patients with higher SBP variability had higher mortality risk. The exact reasons for this significant impact remain unclear; however, we offer the following explanation. Given that 65% of patients in the T3 group had diabetes, it is plausible that in the already high-risk diabetic subgroup, BPV had a relatively diminished effect as an additional risk factor for mortality.

The strength of our study is that we used DialysisNet to continuously collect and analyze real-time BP data from patients undergoing dialysis over a long period of time. The integration of longitudinal BP data from multiple institutions underscores the potential of a standardized common data model for dialysis research. By leveraging repeated real-time data, our study provides a more comprehensive understanding of BPV patterns in patients undergoing HD, which may aid in refining BP management strategies. In addition, the use of common data elements within DialysisNet ensured standardized data collection across dialysis centers, enhancing data quality and comparability. Our study demonstrates the feasibility of using DialysisNet for large-scale, retrospective analyses of data from patients undergoing HD. However, the present study has some limitations. The number of cardiovascular outcomes was insufficient, making comparisons difficult. Another limitation was the unavailability of drug information.

In conclusion, increased interdialytic BPV in patients undergoing HD is associated with all-cause mortality. This was more prominent in SBP than in DBP. Particular attention should be paid to large BPVs in older patients, women, and patients with a relatively longer dialysis vintage.

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Declarations

Conflict of interest None declared.

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