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CLINICAL STUDY

Brain natriuretic peptide is a biomarker of atrial fibrillation in hemodialysis patients

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ABSTRACT

Objectives: Atrial fibrillation (AF) is the most common persistent arrhythmia and has adverse outcomes in hemodialysis patients. It is obscure the relationship between BNP and AF in hemodialysis patients. This study investigated the interventionable predictors of AF in hemodialysis patients.

Methods: In this retrospectively observational cohort study, a total of 205 hemodialysis patients were screened and 140 were enrolled. The anthropometrics, laboratory parameters, electrocardiogram and echocardiogram were collected. The patients were divided into three groups based on the tertiles of BNP value. Kaplan–Meier curves were used to compare the incidence of AF among different BNP groups. Restricted cubic spline curves and receiver operator characteristic curves were drawn, and Cox proportional hazards models were applied to identify the predictive value of BNP and the other related factors for incident AF.

Results: During the 5-year follow-up period, 33 (23.6%) individuals developed incident AF. The incidence of AF increased significantly with an increase in the BNP. Cox proportional hazards models indicated that age, dialysis vintage, left atrial diameter, ultrafiltration rate, hs-CRP and BNP were independent risk factors for incident AF. The hazard ratios of BNP (per 100 pg/mL) were 1.038 (95% confidence interval, 1.012–1.064, $p = 0.004$). BNP had a predictive value for the occurrence of AF (area under the curve = 0.734).

Conclusions: BNP was a good predictor of incident AF in hemodialysis patients. Higher BNP had an increasing adverse event rate of AF. Further research should be needed to clarify the best reference range of BNP in hemodialysis patients.

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Atrial fibrillation; hemodialysis; brain natriuretic peptide; ultrafiltration rate; predictor

Introduction

Atrial fibrillation (AF) is the most common persistent arrhythmia in clinical practice, with adverse outcomes, including a higher risk of ischemic stroke [1,2], heart failure, hospitalization, and mortality, which significantly increases the medical burden, and this also applies to maintenance hemodialysis (MHD) patients. Epidemiological surveys have shown that the incidence of AF in patients with end-stage renal disease is 12.1/1000 person-years, which is 1.66 times higher than that in patients with chronic kidney disease and 2.42 times higher than that in the general population [3]. The incidence of AF is rising [4,5], and recent data suggest that paroxysmal AF can be detected in as many as 40% of MHD individuals [6]. There are not only traditional risk factors, such as hypertension, diabetes, left ventricular hypertrophy, and left atrial enlargement, but also some nontraditional risk factors, such as toxin accumulation, anemia, micro-inflammation, and volume load in MHD patients. All the above factors lead to cardiac enlargement and myocardial fibrosis, and promote the occurrence and development of AF.

Brain natriuretic peptide (BNP) is a peptide hormone synthesized and secreted by cardiomyocytes when ventricular load and ventricular wall tension change. It is a biomarker that reflects volume status in various clinical settings, including for nondialysis and dialysis chronic kidney disease [7–10], which is easy to measure and highly reproducible. Contrary to earlier theories that BNP is secreted mainly from the ventricular myocardium, it has been reported that the left atrium, not the left ventricle, is the main source of BNP in patients with AF. In the general population, BNP can predict the new-onset AF [11], degree of AF progression and cardiac function, and increasingly secreted in AF patients without heart failure [12]. However, it is inconsistent whether plasma BNP reflects fluid overload [13–16], and the relationship between BNP and AF, and the normal reference range of BNP is obscure in MHD

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patients. In view of the limited studies on AF, and the poor outcomes of AF in MHD patients, it is clear that research is sorely needed to identify vulnerable patients and investigate potentially modifiable risk factors for incident AF.

Materials and methods

Participants

The study was a single-center retrospective longitudinal observational cohort study. Patients were recruited in the Department of Nephrology, Beijing Chao-Yang Hospital, Capital Medical University between September 2019 and August 2024. The inclusion criteria were age ≥ 18 years, and dialysis vintage ≥ 6 months. The exclusion criteria were: (1) patients with evidence of historical AF prior to the study, (2) serious valvular heart disease and heart failure (NYHA ≥ grade II), (3) history of hyperthyroidism, (4) acute stage of cardiac-cerebrovascular disease, and (5) missing data. Figure 1 shows a flow diagram of patient selection.

The study protocol, conforming to the ethical guidelines of the Helsinki Declaration, was approved by the Ethics Committee of Beijing Chao-Yang Hospital, Capital Medical University, approval number 2023-ke-14. All participants gave informed written consent.

All enrolled patients underwent hemodialysis 3 times per week and for 3.5–4 h per treatment. All patients used a standard bicarbonate-based ultrapure dialysate (500 mL/min), and blood flow ranged from 200 to 300 mL/min. The dialysate prescription was as follows: sodium 138-140 mmol/L, potassium 2.5 mmol/L, calcium 1.25-1.5 mmol/L, magnesium 0.5 mmol/L, and bicarbonate 35 mmol/L. All patients were treated with disposable synthetic biocompatible dialyzer membranes (polysulfone hollow fiber dialyzer membranes) and heparin or low molecular weight heparin anticoagulant. Water for hemodialysis and related therapies meets the People's Republic of China pharmaceutical industry standards YY 0572-2015.

Data collection

The recorded demographic data of patients included gender, age, height, dialysis vintage (duration of dialysis therapy prior to study entry), primary kidney disease, history of complication (such as diabetes mellitus, hypertension, heart failure, coronary heart disease), and beta-blocker antihypertensive drugs. Dialysis-related data were collected, including dry weight, dialysis time per treatment, average ultrafiltration volume (AF patients were recorded for 6 months before the onset of AF, and patients without AF were recorded for 6 months before the end of study), and vascular access (arteriovenous fistula or central venous catheterization). The ultrafiltration rate (UR) was calculated as follows: [UR (ml/kg/h) = ultrafiltration volume (ml) ÷ dry weight (kg) ÷ dialysis time (h)].

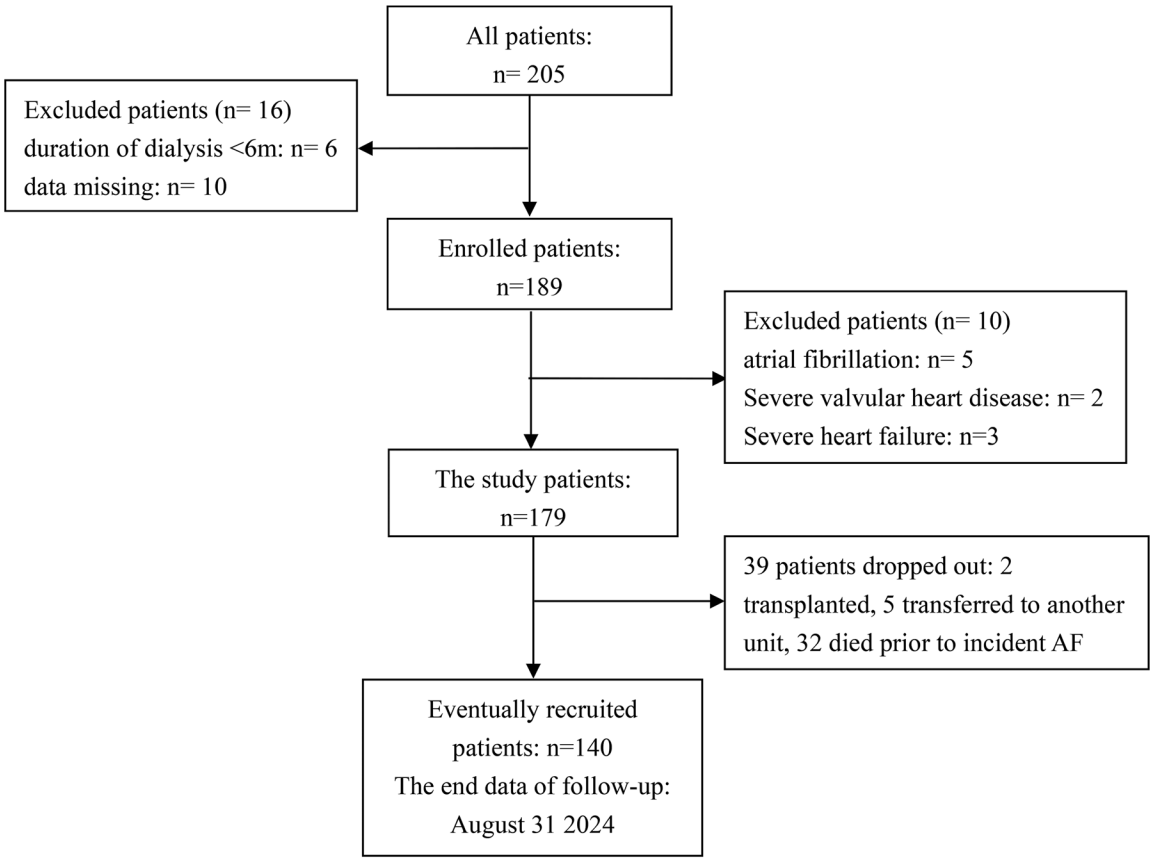

Figure 1. Flow diagram of recruited patients.

The Body Mass Index (BMI) was calculated as follows: BMI (kg/m^2) = dry weight (kg) ÷ height^2 (m^2).

All laboratory parameters were measured by automated and standardized methods on the mid-week dialysis day before hemodialysis, including routine blood tests, blood biochemistry, high sensitivity C-reactive protein (hs-CRP), intact parathyroid hormone (iPTH), β_2-microglobulins (β_2-MG), and BNP. BNP was measured every 3 months and the average of the observation period. The other indicators were baseline data. The transthoracic echocardiogram was performed, and left atrial diameter, left ventricular end-diastolic diameter, left ventricular ejection fraction, and pulmonary arterial hypertension were recorded. All patients underwent a 12-lead electrocardiogram or 24-h dynamic electrocardiogram at least once during every hemodialysis. If the patient had palpitation suffocation or arrhythmia, an electrocardiogram should be performed to confirm the diagnosis.

Atrial fibrillation

Heart rhythm was monitored pre-dialysis and every hour during dialysis. When the patient had palpitation, suffocation, or arrhythmia in auscultation, a 12-lead electrocardiogram was performed to confirm the diagnosis. The patients had palpitation in dialysis intervals, electrocardiogram was performed in time. When the diagnosis is definite, the researcher will record it in the medical record promptly. An ECG recording of AF lasting for more than 30s can be diagnosed as AF.

Study endpoint

The starting time of the selected data was September 2019, the end event was incident AF, and the study deadline was August 2024.

Statistical analyses

Normally distributed continuous variables were expressed as the mean ± standard deviation (SD), and compared using an unpaired Student's *t*-test. Non-normally distributed variables were shown as medians (P_{25}, P_{75}), and compared using the Mann–Whitney *U* tests. Categorical variables were expressed as numbers (percentages) for each item and used chi-square analysis. Kaplan–Meier curves were performed, and logarithmic rank tests were computed to compare the incidence of AF among different BNP groups. The gradually include covariates for model adjustment analysis were conducted to the impact of different BNP groups on the occurrence of AF. Cox proportional hazard models were used to calculate the relative risk of incident AF, hazard ratios (HR), and 95% confidence intervals (95% CI). The 'enter' and 'conditional forward stepwise' methods were used for univariate and multivariate analyses, respectively. The association between levels of BNP and incident AF was evaluated on a continuous scale with restricted cubic spline curves based on Cox proportional hazards models. The receiver operator characteristic (ROC) curves were drawn, and the area under the curve (AUC) was used to evaluate the predictive power of the models.

All statistical analyses were performed using SPSS software version 23.0 (IBM SPSS, Chicago, IL) and R version 4.4.2. All tests were conducted two-sided, and $p < 0.05$ were considered statistically significant.

Results

Participant characteristics

Figure 1 displays a flow diagram of the study cohort selection. A total of 140 MHD patients were included in the study. The study patients had an average age of 56.9 ± 11.5 years, dialysis vintage of 101 (54, 206) months, and 63.6% were male. The cohort had an average follow-up duration of 60 (29, 60) months. During the 5-year follow-up period, 33 individuals developed incident AF (the incidence rate of AF was 23.6%).

Comparison of baseline indicators between AF and non-AF groups

The present study compared demographic data, dialysis-related data, and laboratory parameters between the AF group and non-AF group. Age, dialysis vintage, UR, serum ferritin, hs-CRP, BNP, and left atrial diameter were higher in the AF group than in the non-AF group. The height, blood urea nitrogen, and left ventricular ejection fraction were lower in the AF group than in the non-AF group. The percentage of coronary heart disease and pulmonary arterial hypertension in the AF group was significantly greater than that in the non-AF group. Table 1 displays the baseline study cohort characteristics of all patients.

Comparison of the incidence of AF among different BNP groups

To evaluate the impact of BNP on AF, all patients were divided into three groups based on tertiles of BNP as follows: (Q1, < 89 pg/mL; Q2, 89–245 pg/mL; Q3, >245 pg/mL). Figure 2 displays the distribution of the incident AF according to different BNP groups. As shown in Figure 3, Kaplan–Meier survival analyses revealed the incident rate of AF increased significantly with an increase in the BNP. Log-rank tests demonstrated that the differences among the three groups were statistically significant ($\chi^2 = 15.319$, $p < 0.001$).

The unadjusted hazard of AF was the highest among patients in group Q3 (Table 2). When comparing different BNP groups to a fixed reference of group Q1, the group of higher BNP was associated with higher hazard of AF, and this association was persisted in gradually adjusted models (Table 2).

Cox proportional hazard models for incident AF

Univariate Cox proportional hazards models were performed, and Table 3 presented the univariate associations between

Table 1. Comparison of clinical characteristics between patients with and without AF.

Variables	All ($n=140$)	AF group ($n=33$)	non-AF group ($n=107$)	$t/z/\chi^2$	p
Male [*n* (%)]	89 (63.6)	17 (51.5)	72 (67.3)	2.710	0.147
Age (years)	56.9 ± 11.5	61.9 ± 11.7	55.4 ± 12.4	2.705	0.008[a]
HD vintage (months)	101.0 (53.8, 206.0)	192.0 (85.5, 256.0)	92.0 (40.0, 199.0)	−3.366	0.001[a]
Height (cm)	170 (163, 174)	168 (161, 172)	170 (164, 174)	−2.141	0.032[a]
Weight (kg)	62.88 ± 12.29	61.09 ± 9.21	63.43 ± 12.92	−0.955	0.341
BMI (kg/m^2)	21.96 ± 3.49	21.93 ± 2.94	21.96 ± 3.65	−0.051	0.959
AVF [n (%)]	121 (86.4)	28 (84.8)	93(86.9)	0.092	0.774
Hypertension [*n* (%)]	115 (82.1)	28 (84.8)	87 (81.3)	0.215	0.431
DM [*n* (%)]	48 (34.3)	15 (45.5)	33 (30.8)	2.391	0.144
CHD [*n* (%)]	76 (54.3)	23 (69.7)	53 (49.5)	4.132	0.048[a]
HF [*n* (%)]	21 (15.0)	8 (24.3)	13 (12.1)	2.893	0.100
β-BA [*n* (%)]	92 (65.7)	20 (60.6)	72 (67.3)	0.500	0.532
UR (ml/kg/h)	11.2 ± 3.1	12.4 ± 2.6	10.9 ± 3.1	2.517	0.013[a]
Hb (g/L)	113.6 ± 11.3	110.7 ± 10.4	114.5 ± 11.5	−1.668	0.098
ALB (g/L)	41.5 ± 3.2	41.4 ± 2.9	41.6 ± 3.2	−0.312	0.756
sFerr (μg/L)	100.7(52.4, 232.7)	167.7(65.6, 338.2)	81.8(45.6, 195.3)	−2.096	0.036[a]
BUN (mmol/L)	21.5 (17.9, 25.6)	19.4(16.3, 23.6)	22.1(18.4, 26.2)	−2.230	0.026[a]
UA (μmol/L)	410.0 (366.0, 465.0)	400.0 (360.8, 428.5)	411.5 (369.8, 472.0)	−1.170	0.242
cCa (mmol/L)	2.29 ± 0.23	2.34 ± 0.23	2.28 ± 0.22	1.269	0.207
P (mmol/L)	1.97 ± 0.59	1.91 ± 0.49	1.98 ± 0.62	−0.611	0.542
FBG (mmol/L)	5.21 (4.49, 6.46)	5.15 (4.45, 6.48)	5.24 (4.51, 6.46)	−0.182	0.856
hs-CRP (mg/L)	2.86 (1.36, 6.86)	5.19 (2.59, 13.74)	2.50 (1.22, 6.15)	−3.442	0.001[a]
iPTH (pg/mL)	278.1 (126.5, 375.2)	293.3 (88.8, 455.7)	264.6 (131.5, 368.4)	−0.590	0.555
β_2M (mg/L)	39.8 ± 11.5	42.2 ± 11.1	39.0 ± 11.6	1.371	0.173
BNP (pg/mL)	144.0 (67.3, 311.0)	285.0 (150.0, 1399.5)	117.0 (58.0, 258.0)	−4.082	<0.001[a]
LAD (mm)	37.9 ± 6.02	41.82 ± 5.35	36.81 ± 5.73	4.452	<0.001[a]
LVEDD (mm)	48 (45, 51)	49 (46, 52)	48 (44, 51)	−1.357	0.175
LVEF (%)	66 (62, 70)	64 (50, 68)	67 (63, 71)	−2.861	0.004[a]
PAH [*n* (%)]	29 (20.7)	17 (51.5)	12 (11.2)	24.941	<0.001[a]

Values are expressed as mean ± standard deviations, medians (interquartile ranges), or number (percentage), as appropriate.

AF: atrial fibrillation; HD: hemodialysis; BMI: body mass index; AVF: arteriovenous fistula; DM: diabetes mellitus; CHD: coronary heart disease; HF: heart failure; β-BA: beta-blocker antihypertensive drugs; UR: ultrafiltration rate; Hb: hemoglobin; ALB: albumin; sFerr: serum ferritin; BUN: blood urea nitrogen; UA: uric acid; cCa: corrected serum calcium; P: serum phosphorus; FBG: fasting blood glucose; hs-CRP: high-sensitivity C-reactive protein; iPTH: intact parathyroid hormone; β_2M: β_2-microglobulins; BNP: brain natriuretic peptide; LAD: left atrial diameter; LVEDD: left ventricular end-diastolic diameter; LVEF: left ventricular ejection fraction; PAH: pulmonary arterial hypertension.

[a]$p<0.05$.

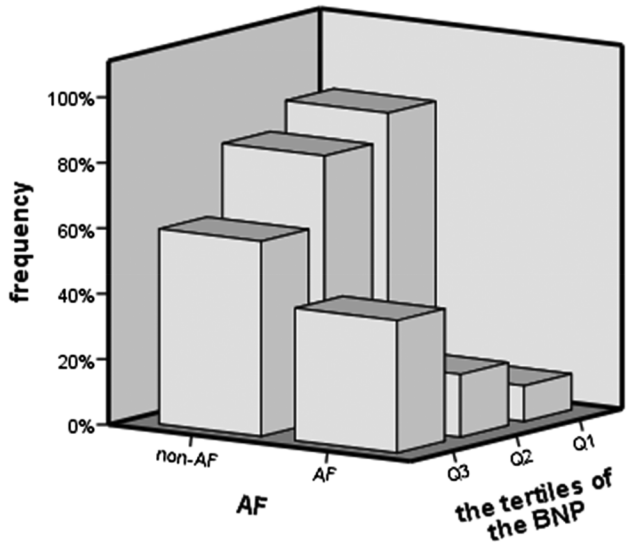

Figure 2. The incidence rate of AF in the different BNP tertiles. The number of AF/total patients in each tertile was 5/46, 9/47 and 19/47, respectively (χ^2= 12.041, $p=0.001$).

incident AF and other covariates. The potential variables in univariate Cox proportional hazards models ($p<0.03$) were entered into a multivariate Cox proportional hazards model with the conditional forward stepwise method. The result indicated that age, dialysis vintage, left atrial diameter, UR, hs-CRP, and BNP were independent risk factors for on-set AF. The hazard ratios of BNP (per 100 pg/mL) and UR (per 1 mL/kg/h) were 1.038 (95% CI, 1.012–1.064, $p=0.004$) and 1.242 (95%CI, 1.050–1.469, $p=0.011$) for an incident of AF, respectively (shown in Table 3).

In order to examine the association of BNP and incident AF in a more flexible manner, we conducted analyze the association of BNP as a cubic spline and incident AF. This analysis showed the hazard of incident AF increased monotonically as increasing BNP (Figure 4).

Predictive power of the independent risk factors for AF

ROC curves were drawn for the above risk factors. In descending order, the AUC was BNP [0.734 (95% CI: 0.638–0.831)], left atrial diameter [0.727 (95% CI: 0.631–0.824)], dialysis vintage [0.697 (95% CI: 0.599–0.795)], hs-CRP [0.696 (95% CI: 0.596–0.797)], age [0.643 (95% CI: 0.537–0.748)] and UR [0.636 (95%CI: 0.534–0.738)], respectively.

BNP and UR, the modifiable risk factors, were combined for the prediction of incident AF. Figure 5 illustrates that the AUC of the combining model was increased to 0.740 (95% CI: 0.647–0.834)].

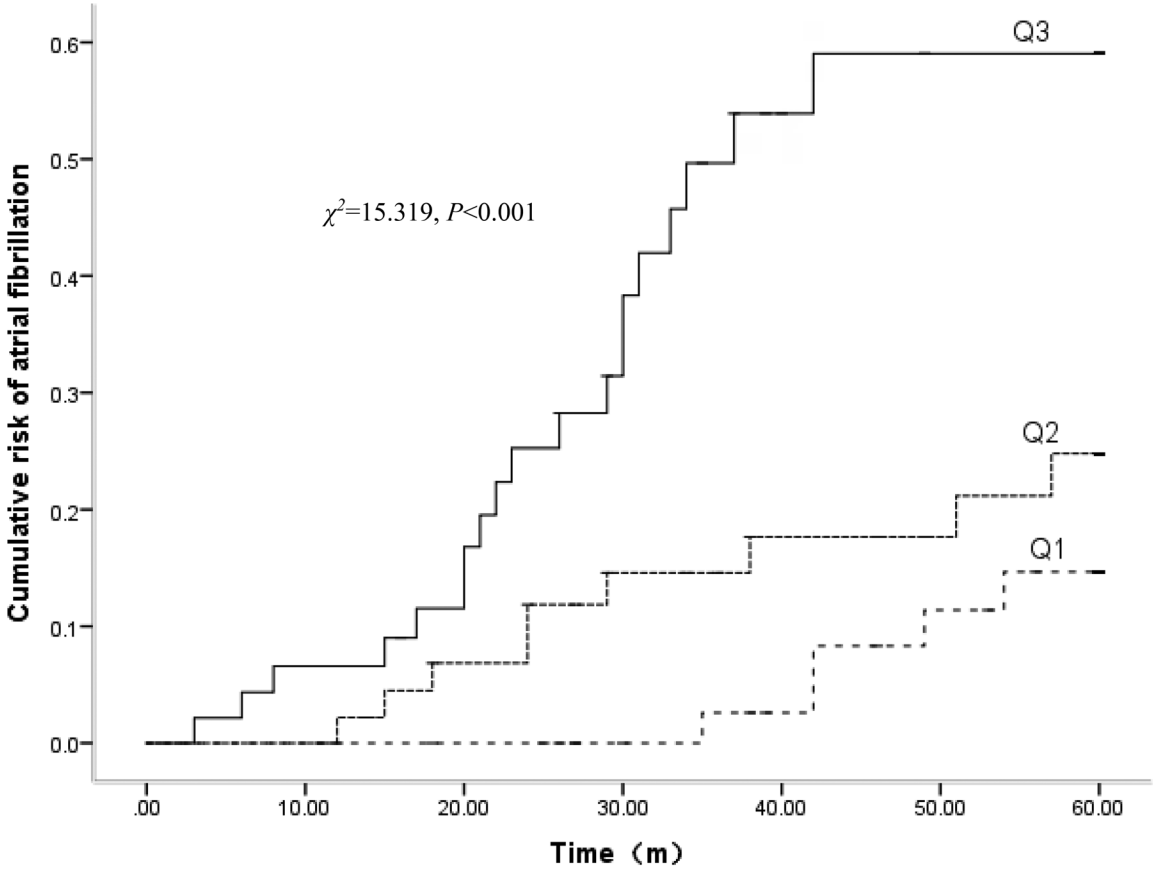

Figure 3. Kaplan–Meier Plots for the incidence rate of AF in the different BNP groups (log-rank test).

Table 2. Associations of atrial fibrillation with different BNP groups.

	Unadjusted		Model 1		Model 2	
Variables	HR (95% CI)[a]	*p*-value	HR (95% CI)[a]	*p*-value	HR (95% CI)[a]	*p*-value
Group Q1	Reference	–	Reference	–	Reference	–
Group Q2	1.957 (0.656, 5.840)	0.229	1.548 (0.511, 4.691)	0.440	1.822 (0.589, 5.636)	0.298
Group Q3	5.130 (1.911, 13.771)	0.001[b]	3.613 (1.293, 10.095)	0.014[b]	3.477 (1.230, 9.829)	0.019[b]

[a]HR: hazards ratios; 95% CI: 95% confidence interval.
Model 1: adjusted for age, gender, BMI, dialysis vintage. Model 2: adjusted for model 1 plus ultrafiltration rate, high-sensitivity C-reactive protein, left atrial diameter, left ventricular ejection fraction, pulmonary arterial hypertension.
[b]$p < 0.05$.

Discussion

Our single-center observational retrospective cohort study indicated that the prevalence rate of AF in hemodialysis patients was 23.6%, which was similar to the previous reviews [17,18]. The age, inflammation, atrial remodeling, dialysis duration, BNP, and UR were related factors of AF, among which BNP and UR were modifiable and interventional risk factors in hemodialysis patients. Evidence-based data on the relation between BNP and AF was obscure, with most studies investigating the correlation between BNP and dry weight [19]/fluid overload [8,20–22], and the relation between UR and AF [23,24]. Since BNP can be rectified by adjusting the dry weight, it is possible to delay to incident of AF if AF is related to BNP. Therefore, it is important to investigate the relationship between BNP and AF in patients with MHD.

This study found that UR rate was an independent risk factor of AF in hemodialysis patients, and the risk of AF increased by 24.2% as the UR increasing 1 mL/kg/h. Because of the UR taking into account the weight and single dialysis time, we thought that UR was better than the ultrafiltration volume in evaluating the volume load and the fluid rebalance during dialysis. The potential mechanisms of the correlation between UR and AF were as follows. First, higher UR caused overtly rapid fluid removal, it exceeded the vascular refill rate, and the intravascular volume was relatively insufficient, which inhibited the baroreflex. Therefore, sympathetic nerves were activated and catecholamines were released, which ultimately increased susceptibility to AF. Second, relatively insufficient intravascular volume caused by excessively fast UR was prone to complicated with intradialytic hypotension. A large observational study reported that more frequent intradialytic hypotension was associated with higher

Table 3. Cox proportional hazards model of AF (with the conditional forward stepwise method, $p > 0.03$ to remove).

	Univariate predictors		Multivariate predictors	
Variables	HR (95% CI)[a]	*p*-value	HR (95% CI)[a]	*p*-value
Male [*n* (%)]	1.563 (0.789, 3.093)	0.200	–	–
Age (years)	1.043 (1.015, 1.073)	0.003[b]	1.039 (1.007, 1.073)	0.018[c]
HD vintage (years)	1.054 (1.011, 1.100)	0.013[b]	1.045 (1.000, 1.093)	0.049[c]
AVF [*n* (%)]	1.381 (0.532, 3.584)	0.507	–	–
Diabetes [*n* (%)]	2.007 (1.010, 3.989)	0.047	–	–
β-BA [*n* (%)]	0.843 (0.419, 1.695)	0.632	–	–
UR (ml/kg/h)	1.192 (1.049, 1.355)	0.007[b]	1.242 (1.050, 1.469)	0.011[c]
Hemoglobin (g/L)	0.981 (0.953, 1.011)	0.209	–	–
Serum albumin (g/L)	0.960 (0.861, 1.070)	0.459	–	–
sFerr (10μg/L)	1.016 (1.002, 1.029)	0.021[b]	–	0.150
BUN (mmol/L)	0.934 (0.873, 0.999)	0.048	–	–
Uric acid (mmol/L)	0.997 (0.993, 1.002)	0.259	–	–
cCa (mmol/L)	1.880 (0.420, 8.413)	0.409	–	–
P (mmol/L)	0.780 (0.428, 1.423)	0.418	–	–
FBG (mmol/L)	1.024 (0.904, 1.159)	0.713	–	–
hs-CRP (mmol/L)	1.023 (1.009, 1.037)	0.001[b]	1.026 (1.008, 1.043)	0.003[c]
iPTH (pg/ml)	1.001 (0.999, 1.002)	0.409	–	–
β_2M (mg/L)	1.013 (0.982, 1.044)	0.417	–	–
BNP (100pg/mL)	1.057 (1.034, 1.080)	<0.001[b]	1.038 (1.012, 1.064)	0.004[c]
LAD (mm)	1.165 (1.095, 1.241)	<0.001[b]	1.113 (1.035, 1.198)	0.004[c]
LVEDD (mm)	1.082 (1.008, 1.163)	0.030	–	–
LVEF (%)	0.955 (0.932, 0.979)	<0.001[b]	–	0.869
PAH [*n* (%)]	5.355 (2.690, 10.661)	<0.001[b]	–	0.102

[a]HR: hazards ratios; 95% CI: 95% confidence interval.

AF: atrial fibrillation; HD: hemodialysis; AVF: arteriovenous fistula; β-BA: beta-blocker antihypertensive drugs; UR: ultrafiltration rate; sFerr: serum ferritin; BUN: blood urea nitrogen; cCa: corrected serum calcium; P: serum phosphorus; FBG: fasting blood glucose; hs-CRP: high-sensitivity C-reactive protein; iPTH: intact parathyroid hormone; β_2M: β_2-microglobulins; BNP: brain natriuretic peptide; LAD: left atrial diameter; LVEDD: left ventricular end-diastolic diameter; LVEF: left ventricular ejection fraction; PAH: pulmonary arterial hypertension.

[b]$p < 0.03$

[c]$p < 0.05$.

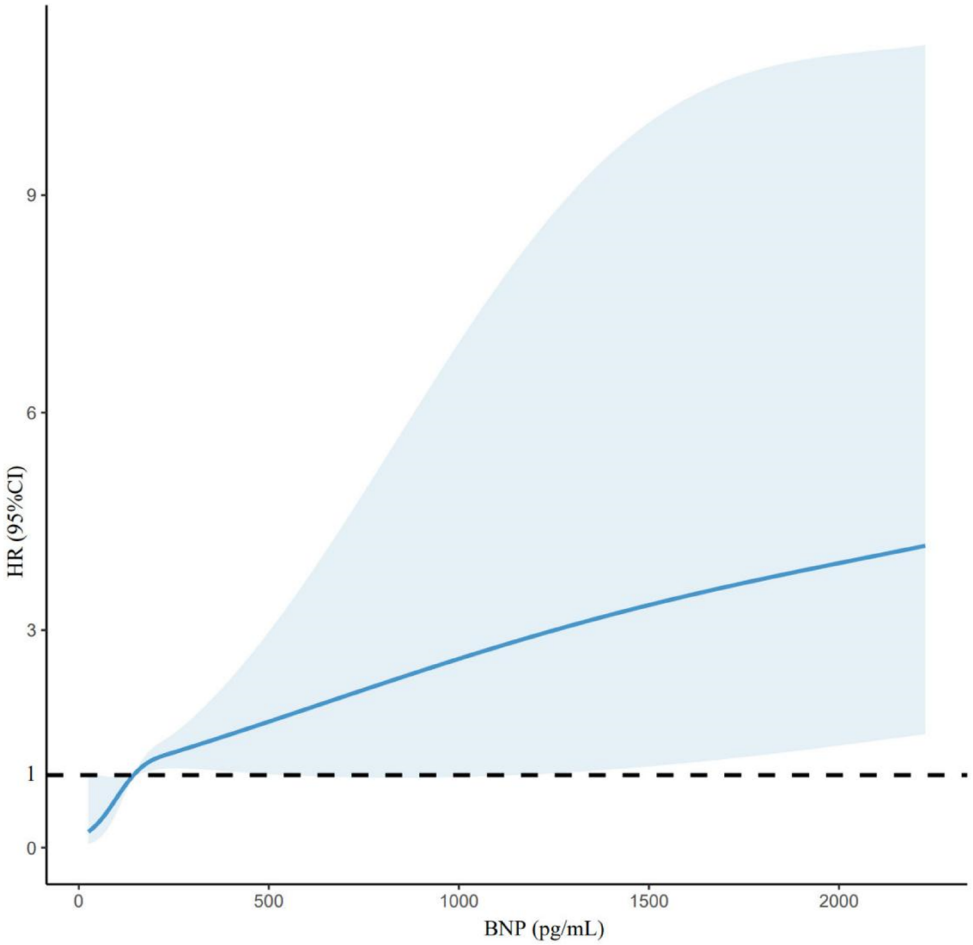

Figure 4. Univariate associations between BNP and incident AF when BNP is modeled as a cubic spline. The solid blue line indicates HRs for incident AF as a function of BNP and the light gray shading represents the associated 95% CIs.

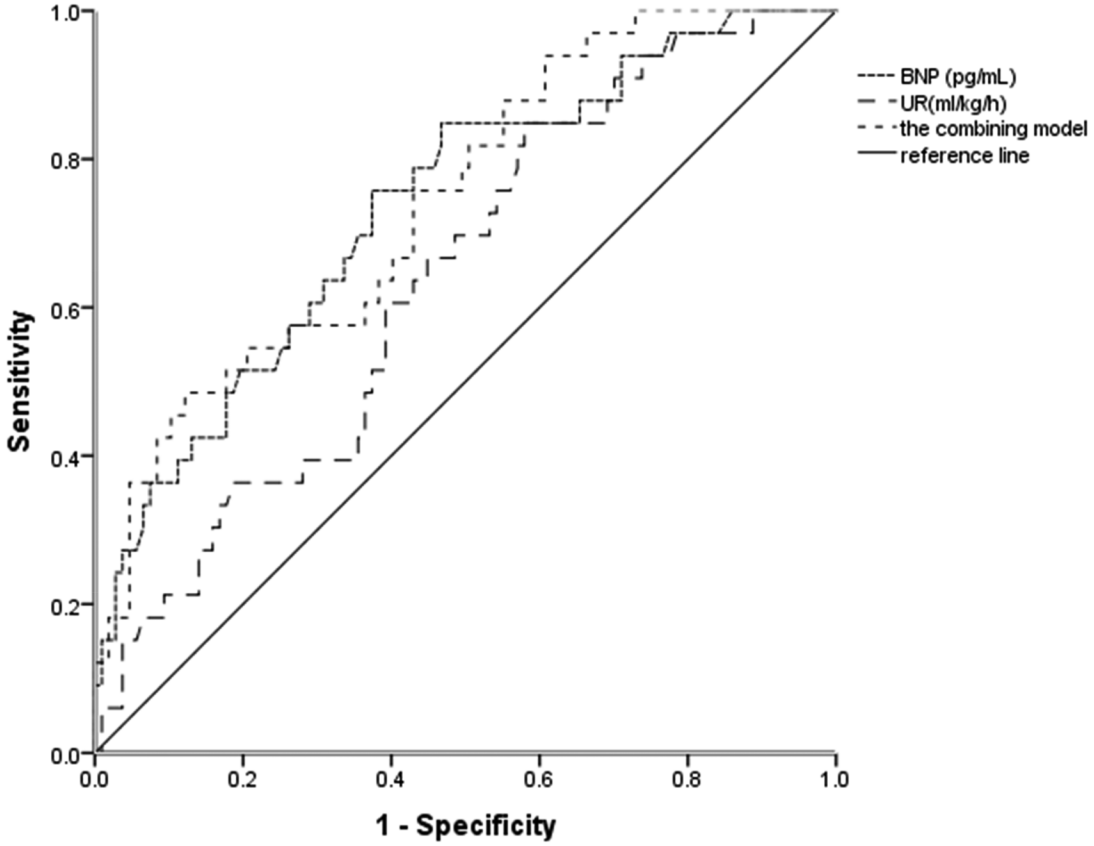

Figure 5. The ROC curves of BNP, UR and their combining model in assessing incident AF. (BNP, AUC = 0.734, sensitivity = 0.758, specificity = 0.626; UR, AUC= 0.636, sensitivity = 0.848, specificity = 0.421; combining model, AUC= 0.740, sensitivity = 0.485, specificity = 0.879).

incidence rates of AF [25]. The echocardiographs revealed cardiac 'stunning' in the setting of higher UF rates that, over time, induced cardiac remodeling and created a cardiac milieu conducive to arrhythmia development [26].

In addition, our study showed that BNP was a risk factor for AF, and in the related incident, AF increased monotonously with increasing BNP. Taskapan et al. considered that the expansion of extra-cellular volume causing myocardial stretching may be the principal cause of increased BNP in MHD patients [27]. Jacobs et al. also reported that BNP concentration had a positive effect on the extracellular water/body weight ratio (reflecting volume overload) [14], and monitoring BNP helped with dry weight adjustment [19]. The volume overload contributed to some inducing both transient and long-standing cardiac structural changes [26]. A longitudinal study demonstrated that BNP was strongly positively related to left atrial volume in hemodialysis patients [28]. Ishigami et al. [29] found that BNP and left atrial diameter had a positive correlation, and BNP was a useful tool for estimating LA enlargement among patients undergoing hemodialysis. Han et al. [30] also pointed out the left atrial volume index was positively associated with BNP in hemodialysis patients. Although the pathophysiology of AF was complex, structural remodeling was a key contributor [31]. Excessive volume load caused atrial traction, left ventricular hypertrophy, and neurohumoral fluid changes, which increased the AF risk. In brief, the left atrium structural changes due to volume overload resulted in increased AF hazards. Meanwhile, hemodynamic instability due to AF resulted in the dialysis being incomplete and assessing correctly dry weight being impossible. Ultimately, these further induced a vicious cycle of interaction in AF and volume overload. Early detection and timely treatment can interrupt this cycle, which may be beneficial for improving both cardiac rhythm and cardiac function in hemodialysis patients.

This study also had some limitations. First, this research was a single-center, retrospective cohort study. The multi-center and larger cohort studies are expected to further confirm the predictive performance of the BNP for AF occurrence and reference range of BNP in hemodialysis patients. Second, our results had limited generalizability to other racial groups, further research is required to determine whether these findings are universal across ethnic groups and countries. Third, this study may miss some paroxysmal AF and asymptomatic patients, reducing the incidence of AF and decreasing the excluded patients with a previous history of AF. But the frequency of medical visits was 3 times per week can mitigate detection bias. Finally, less enrolled patients resulted in the included variables were limited, which may lead to bias. But this study was a preliminary discussion on the relationship between BNP and atrial fibrillation, and further studies are mandatory for the aim of exploiting evidence-based practice and putting into practice targeted at reducing the adverse outcomes of AF.

In conclusion, we demonstrated that a higher UR and BNP were associated with a higher hazard of incident AF. The combined model of UR and BNP had a stronger ability to predict the risk of AF. According to reducing UR by lowering interdialytic weight gains, and adjusting dry weight by monitoring BNP, avoiding volume overload and volume depletion may reduce the incident AF in hemodialysis patients. So as further reduced the occurrence of ischemic stroke and heart failure, improved the clinical outcomes. Our results provided additional evidence regarding the harms of higher BNP and underscored the need for further researches determining the normal reference range of BNP in MHD patients.

Disclosure statement

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Data availability statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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