Original Article

Evaluating The Diagnostic Utility of NT-Pro BNP in Cardiac Patients for Renal Dysfunction: A Prospective Study

Sadaf Ali[1*], Fozia Jamal[2], P. Usha Kiran[3]

[1,2]Assistant Professor, Department of Biochemistry, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, Uttar Pradesh, India.
[3]Professor and Head, Department of Biochemistry, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, Uttar Pradesh, India.

Abstract

Background: Patients with heart failure have an increased risk of acute renal impairment. Renal dysfunction is common in heart failure due to similar risk factors, leading to worse outcomes if both are present together. NT-pro BNP levels are raised in heart failure, which plays a role in the development of complications in the kidneys and leads to increased mortality and morbidity. To evaluate renal dysfunction using NT-pro BNP among heart failure patients in Moradabad and to correlate NT-pro BNP levels with the severity of heart failure and kidney function. **Material and Methods:** 102 individuals with a diagnosis of heart failure participated in a prospective trial. Ejection fraction and Echo findings were used to evaluate cardiovascular functioning. Serum NT-pro BNP and KFT were analyzed. Patients were grouped into three groups, each based on NT-pro BNP levels, Ejection fraction, and eGFR values. Statistical tools used were Pearson's correlation coefficient, ANOVA, and multiple linear regression. **Results:** Based on NT-pro BNP levels, a statistically significant difference was found only in serum levels of urea, creatinine, uric acid, calcium, and chloride between the three groups. There was a substantial negative connection ($p<0.05$) between NT-pro BNP and blood calcium and a significant positive correlation ($p<0.05$) with serum urea, creatinine, uric acid, phosphorus, and potassium. Serum levels of NT-proBNP, urea, creatinine, uric acid, and phosphorus were statistically significantly different across the three groups of patients based on eGFR. The multiple linear regression results showed that EF, NT-pro BNP, and GFR had a moderately significant collective influence ($F(2, 70) = 13.52$, $p < .001$, $R2 = 0.28$, $R2adj = 0.26$). EF ($t = -2.387$, $p = .020$) and NT-pro BNP ($t = -5.006$, $p < .001$) were identified as significant predictors in the model after closer examination of the individual factors. **Conclusion:** Screening patients for NT-pro BNP levels and correlating them with kidney function may aid in formulating strategies to prevent further mortality and help target molecular mechanisms to prevent further complications.

Keywords: NT-pro BNP, Kidney Function, Heart Failure, Ejection Fraction.

Received: 02 January 2026 **Revised: 20 January 2026** **Accepted: 03 February 2026** **Published: 14 February 2026**

Introduction

One of the main worldwide health issues is heart failure, leading to increased mortality and morbidity. Current global estimates indicate that 38 million people in the world are living with heart failure. According to some studies, cardiovascular mortality rates are higher than those of some cancers. [1] The prevalence of heart failure in India is 1% of the adult population, which is nearly 8-10 million patients. [7] The most frequently observed co-morbidities in these patients are diabetes mellitus, hypertension, and dyslipidemia, further contributing to complications. Patients with heart failure are more likely to get acute renal impairment. [3] Renal dysfunction in heart failure patients is common due to similar risk factors, leading to worse outcomes if both coincide than if one alone. [4,5] Heart failure, along with CKD, may result in significantly poor outcomes. [6,16] Data on the prevalence, risk elements, routine indicators, and outcome of renal impairment in patients with heart failure are limited. This study was done to fill the gap between routine markers and early markers of renal dysfunction in heart failure patients. Renal dysfunction in heart failure occurs due to decreased cardiac output, which reduces renal perfusion and increases central venous pressure, leading to venous congestion in the kidneys. These changes affect hemodynamic, neuro-hormonal, and inflammatory pathways. Diuretic resistance and renal tamponade hypothesis also contribute to further kidney damage in HF patients.[8] BNP and NT-proBNP are significant indicators of cardiovascular death and morbidity in middle-aged and older persons, according to several studies.[1–4] In recent years, these peptide-based assays have emerged as valuable prognostic indicators to evaluate how well HF patients respond to treatment.[9–12] Of the two natriuretic peptides, NT-proBNP is often used in clinical practice.[13] Increased levels of NT-pro BNP

Address for correspondence: Dr. Sadaf Ali,
Assistant Professor, Department of Biochemistry, Teerthanker Mahaveer Medical College & Research Centre, Moradabad, UP.
E-mail:mir.sadaf.ali@gmail.com
ORCID ID :https://orcid.org/0000-0002-4974-1331

DOI:
10.21276/amit.2026.v13.i1.365

How to cite this article: Ali S, Jamal F, Kiran PU. Evaluating Diagnostic Utility of NT-Pro BNP in Cardiac Patients for Renal Dysfunction: A Prospective Study. Acta Med Int. 2026;13(1):428-432.

in the bloodstream may indicate volume overload,[19] left ventricular dysfunction,[18] and cardiovascular disease.[17] The main aim of our study is to evaluate NT-pro BNP levels and KFT and to correlate NT-pro BNP levels with renal dysfunction among heart failure patients. Evaluation of NT-pro BNP levels in CVD is essential in identifying and designing treatment modalities to reduce CKD. To diagnose and assess dyspneic patients for early initiation of heart failure medication, NT-proBNP testing may be a useful tool. According to several studies, people with chronic kidney disease (CKD) are more likely than the general population to develop left ventricular hypertrophy, ischemic heart disease, cardiac arrhythmias, and valvular calcification, all of which contribute to heart failure with a maintained left ventricular ejection fraction.[4-7]

Early screening, especially for cardiac markers like NT-pro BNP, and application of preventive strategies in heart failure patients may help to prevent renal dysfunction in these patients.[14,15]

Although NT-pro BNP is understood to be the chief biomarker for heart failure, very few studies have shown the correlation between renal functioning and NT-pro BNP levels in heart failure patients. The current study was conducted to assess NT-pro BNP levels in patients with heart failure and to establish a correlation between these levels and renal function test results. The association between these factors may help inform preventive strategies for renal dysfunction in patients with heart failure. In individuals with congestive heart failure (CHF), both NT-pro BNP and renal function are predictive markers of survival. It is uncertain, therefore, how BNP, renal function, and heart failure as an emergency diagnosis are related. Significant renal impairment affects therapy and results for most patients treated with acute decompensated heart failure.

Materials and Methods

Study design and settings: A prospective observational study was done on 102 patients diagnosed with heart failure. Patients admitted with heart failure diagnosed with echocardiography in the age group of 18-80 years were included in the study. Patients with existing kidney disorders, stroke, and other critical illnesses were excluded from the study. Ejection fraction and Echo findings were used to assess cardiovascular functioning. Serum NT-pro BNP levels and kidney function tests, including urea, creatinine, uric acid, phosphorus, and electrolytes, were evaluated in these patients using the Vidas analyzer and the Beckman DxC 700 auto analyzer, respectively.

Statistical analysis: The data were collected and compiled in Excel. Patients were grouped into three groups each based on NT-pro BNP levels (three groups), ejection fraction values (three groups), and eGFR (calculated using the CKD-EPI formula) values separately. Data were obtained after seeking permission from the Institutional Ethical Committee. Correlations of NT-pro BNP levels with KFT and ejection fraction were analysed. Statistical analysis was performed using tools such as Pearson's correlation coefficient for correlation, ANOVA for comparisons, and multiple linear regression for predictive modelling.

Ethical consideration: Ethical clearance was obtained from the Institutional Ethics Committee of the college vide number TMU/IEC/2024-25/FACULTY/02.

Results

The research comprised 102 individuals with heart failure. The statistical analysis for the test parameters is as follows:

Table 1: Comparison of KFT based on NT-pro BNP

Parameters	NT-BNP (<4000) N= 32	NT-BNP (4000-20000) N = 18	NT-BNP (>20000) N = 24	p-value
NT-pro BNP (pg/mL)	1518 (456-2713)	7181 (4804-13046)	23631 (22207-24702)	<0.001**
Urea (mg/dL)	33.5 (27.2-55.4)	72.8 (48.3-122.9)	81 (64.9-120)	<0.001**
Creatinine (mg/dL)	0.8 (0.6-1.28)	1.97(0.9-2.49)	2.53 (1.39-5.95)	<0.001**
Uric acid (mg/dL)	5.5 (4.2-7.05)	8.25 (6.23-11.4)	8.45 (6.93-12.93)	<0.001**
Calcium (mg/dL)	8.74 ± 0.89	8.11 ± 0.66	8.09 ± 0.80	0.006*
Phosphorus (mg/dL)	4.2 (3.5-4.6)	4.05 (3.43-4.73))	4.7 (3.1-7.8)	0.232
Sodium (mmol/L)	136 (132.5-138.5)	136 (129-140)	136 (131-139)	0.885
Potassium (mmol/L)	3.94 ± 0.74	4.42 ± 0.84	4.37 ± 1.08	0.118
Chloride (mmol/L)	94.44 ± 5.15	101.93 ± 7.68	101.27 ± 7.33	0.011*

Based on NT-pro BNP, patients were divided into 3 categories (32 patients had NT-pro BNP <4000, 18 had 4000-20000, and 24 had>20000) and compared for KFT. A statistically significant difference was found only in serum levels of urea, creatinine, uric acid, calcium, and chloride between the groups ($p < 0.05$). The mean and standard deviation were used to display regularly distributed data, whereas the median and interquartile range were used to display non-normally distributed data. ANOVA was applied to compare groups for normally distributed data. The Kruskal-Wallis test was used for group comparisons with non-normally distributed data.

Table 2: Correlation of NT-pro BNP with KFT among patients with heart failure

Parameters	r-value (correlation coefficient)	p-value
NT-pro BNP and Urea	0.402	0.000**
NT-pro BNP and Creatinine	0.484	0.000**
NT-pro BNP and Uric acid	0.202	0.043*
NT-pro BNP and Calcium	-0.246	0.013*
NT-pro BNP and Phosphorus	0.250	0.012*
NT-pro BNP and Sodium	-0.121	0.229
NT-pro BNP and Potassium	0.219	0.028*
NT-pro BNP and Chloride	0.179	0.074

**. Correlation is significant at the 0.01 level (2-tailed)
*. Correlation is significant at the 0.05 level (2-tailed)

NT-pro BNP was shown to have a substantial negative connection with serum calcium ($p<0.05$) and a significant positive association with serum urea ($p<0.01$), serum creatinine ($p<0.01$), serum uric acid ($p<0.05$), serum phosphorus ($p<0.05$), and serum potassium ($p<0.05$). However, NT-pro BNP showed no correlation with either serum sodium or chloride ($p>0.05$).

Table 3. Comparison of biochemical parameters based on EF

Parameters	EF (>50%) N= 32 (Mean ± SD)	EF (41-49%) N = 18 (Mean ± SD)	EF (<40%) N = 24 (Mean ± SD)	p-value
NT-pro BNP (pg/mL)	10203.69 ± 420.44	10285.78 ± 410.06	13967.54 ± 490.12	0.324
Urea (mg/dL)	84.82± 9.92	69.90 ± 8.56	69.388 ± 2.92	0.437
Creatinine (mg/dL)	2.71 ± 0.99	1.92 ± 0.62	1.65 ± 0.40	0.180
Uric acid (mg/dL)	7.89 ± 1.25	13.08 ± 2.11	8.29± 1.59	0.207
Calcium (mg/dL)	7.96 ± 1.65	8.21 ± 0.74	8.57 ± 0.48	0.182
Phosphorus (mg/dL)	5.12 ± 0.57	4.20± 0.31	4.66 ± 0.72	0.387
Sodium (mmol/L)	134.09 ± 5.96	135.61 ± 7.71	134.72 ± 4.48	0.697
Potassium (mmol/L)	4.27 ± 0.80	4.30 ± 0.08	4.19 ± 0.93	0.915
Chloride (mmol/L)	99.24 ± 7.10	100.73 ± 9.54	100.10 ± 5.29	0.777

Based on EF levels, patients were divided into 3 categories (32 patients had EF >50%, 18 had 41-49%, and 24 had <40%) and compared for KFT. There was no statistically significant difference in any kidney function test parameter between the groups ($p > 0.05$).

Table 4: Correlation of EF with other biochemical parameters among patients with heart failure

Parameters	r-value (correlation coefficient)	p-value
EF and NT-pro BNP	0.131	0.26928
EF and Urea	0.0343	0.7613
EF and Creatinine	0.2081	0.0621
EF and Uric acid	-0.165	0.1413
EF and Calcium	-0.1333	0.235
EF and Phosphorus	0.0696	0.5369
EF and Sodium	-0.0317	0.9033
EF and Potassium	0.0574	0.6107
EF and Chloride	-0.0639	0.5709

**. Correlation is significant at the 0.01 level (2-tailed)
*. Correlation is significant at the 0.05 level (2-tailed)

EF showed no correlation with any kidney function parameters. ($p > 0.05$)

Table 5: Comparison of other parameters based on GFR

Parameters	GFR (>60 mL/min/1.73m2) N= 33 (Mean ± SD)	GFR (39-59mL/min/1.73m2) N = 16 (Mean ± SD)	GFR (<30mL/min/1.73m2) N = 24 (Mean ± SD)	p-value
EF (%)	45.75 ± 12.06	40.93 ± 9.34	47.5 ± 11.70	0.201104.
GFR (mL/min/1.73m^2)	92.15 ± 7.56	44.18 ± 8.17	16.95 ± 7.98	0.00001**
NT-pro BNP (pg/mL)	6965.93 ± 298.73	12473.31 ± 243.78	17313.25 ± 985.10	0.000253*
Urea (mg/dL)	43.42 ± 19.14	85.16 ± 10.14	114.89 ± 14.51	0 .00001**
Creatinine (mg/dL)	0.81 ± 0.24	1.63 ± 0.31	4.39 ± 1.62	0 .00001**
Uric acid (mg/dL)	6.27 ± 2.60	14.88 ± 2.66	9.71 ± 3.40	0.020907*
Calcium (mg/dL)	8.58 ± 0.77	8.00 ± 0.93	7.85 ± 0.89	0.0522
Phosphorus (mg/dL)	3.79 ± 0.01	4.85 ± 0.90	5.95 ± 0.95	0.001095*
Sodium (mmol/L)	135.5 ± 5.12	134.96 ± 4.90	133.35 ± 7.52	0.402536
Potassium (mmol/L)	4.043 ± 0.75	4.38 ± 0.92	4.45 ± 0.65	0.19393
Chloride (mmol/L)	98.61 ± 6.64	100.20 ± 6.24	101.43 ± 8.40	0.342388

Based on GFR, patients were divided into 3 categories (33 patients were found to have GFR >60mL/min/1.73m2, 16 patients had 39-59mL/min/1.73m2, and 24 patients had (<30mL/min/1.73m2 and compared for KFT. Serum levels of NT-proBNP, urea, creatinine, uric acid, and phosphorus varied significantly across groups ($p < 0.05$).

Table 6: Correlation of GFR with other parameters among patients with heart failure

Parameters	r-value (correlation coefficient)	p-value
GFR and EF	-0.032	0.786177
GFR and NT-pro BNP	-0.4741.	0.000023**
GFR and Urea	-0.6967	0.00001**
GFR and Creatinine	-0.7262	0.00001**

GFR and Uric acid	-0.2296	0 .050697*
GFR and Calcium	0.259	0.026925*
GFR and Phosphorus	-0.4761	0.000021**
GFR and Sodium	0.1746	0.139566
GFR and Potassium	-0.2457	0.036148*
GFR and Chloride	-0.147	0.214589

**. Correlation is significant at the 0.01 level (2-tailed)

*. Correlation is significant at the 0.05 level (2-tailed)

GFR showed significant correlations with NT-pro BNP, urea, creatinine, uric acid, calcium, phosphorus, and serum potassium ($p<0.05$), and was not significantly correlated with serum sodium, chloride, or ejection fraction ($p>0.05$).

Table 7: Multiple linear regression analysis of EF, NT-pro BNP with e GFR as dependent variable

	Coeff	SE	t-stat	Stand Coeff	p-value
Ln (EF)	-0.767	0.3213	-2.387	-0.247	0.0197011*
Ln (NT-pro BNP)	-0.262	0.0523	-5.006	-0.519	0.000017**

Multiple linear regression results showed that EF and NT-BNP, had a moderately significant collective influence ($F(2, 70) = 13.52$, $p < .001$, $R2 = 0.28$, $R2adj = 0.26$). EF ($t = -2.387$, $p = .020$) and NT-pro BNP ($t = -5.006$, $p < .001$) were identified as significant predictor in the model after closer examination of the individual factors.

Discussion

When heart failure worsens, ventricles, or the lower chambers of the heart, produce a substance called pre-pro BNP. Pre-pro BNP is a 134-amino acid peptide. When cleaved, it yields 108 amino acids and a 26-amino-acid signal peptide. With subsequent cleavage, there is production of NT-pro BNP, which is a 76 amino acid N-terminal pro-B-type natriuretic peptide. [24]

BNP stimulates natriuretic peptides, which, in turn, cause diuresis, renin and aldosterone inhibition, vasodilation, and fibrosis inhibition. Organs with strong blood flow, such as the kidneys, liver, and muscles, passively eliminate NT-pro BNP. In healthy people, the kidneys eliminate 15–20% of BNP and NT-proBNP; this renal clearance is maintained in the presence of significant kidney failure. NT-pro BNP increases in the setting of heart failure. However, the non-cardiac conditions in which it increases are kidney diseases, advanced age, stroke, and critical illnesses, independent of heart failure. Our findings from [Table 1] confirm that NT-pro BNP levels are markedly elevated in heart failure patients with renal dysfunction, supporting earlier studies. [20-24] Renal dysfunction worsens the prognosis in heart failure, with mechanisms including neuro-hormonal activation, reduced renal perfusion, and venous congestion. [8] Elevated NT-pro BNP in these patients may be both a marker of cardiac strain and a reflection of impaired renal clearance. According to [Table 2] of the results, NT-pro BNP showed a significant positive correlation with serum urea, creatinine, uric acid, phosphorus, and serum potassium. However, there was a negative correlation with serum calcium ($p<0.05$). The observed negative correlation with calcium might be explained by secondary hyperparathyroidism and altered calcium-phosphate metabolism in CKD. [23] According to Andreas Luchner et al. BNP and NT-proBNP are markedly influenced by renal dysfunction. [25] Rajat Tagore et al. state that more and more CKD patients would need early use of retardation techniques and co-morbidity screening; in particular, renal failure in these individuals seems to have a major impact on cardiac BNP. In CKD, BNP may be a better biomarker for cardiac dysfunction. [26] Renal failure raises BNP and NT-proBNP concentrations; the degree of change also depends on LVEF and sex, according to Pornpen Srisawasdi et al. However, our results from [Table 3 and 4] showed no statistically significant difference or correlation between the kidney function test parameters and ejection fraction across the groups. Our findings from [Table 5 and 6] confirm that there was a statistically significant difference in serum levels of NT-proBNP, urea, creatinine, uric acid, and phosphorus between the groups ($p < 0.05$). GFR showed substantial correlations with NT-pro BNP, urea, creatinine, uric acid, calcium, phosphorus, and serum potassium ($p<0.05$), and was not significantly correlated with serum sodium, chloride, or ejection fraction ($p>0.05$). Therefore, measuring GFR would be a better predictor of renal function, as done in this study. GFR estimation may be useful for detecting more cases of renal dysfunction at an early stage. The long-term prognosis of AHF patients is better predicted when GFR and NT-pro BNP are combined. Early detection of these high-risk individuals may enable medical professionals to adjust treatment plans, thereby improving patients' clinical outcomes. [28] According to [Table 7] of the results from the regression analysis, we found that NT-pro BNP is a better predictor of renal function than left ventricular ejection fraction in cardiac patients. This may be the reason for the statistically less significant difference when we have compared various biochemical parameters based on ejection fraction. Additional prospective studies are required, although using the impact of renal failure, defined by stage, in conjunction with NT-pro BNP and eGFR may enhance the rationale for diagnosis and monitoring. [27] These results underscore the need for integrated cardiac and renal care. Early measurement of NT-pro BNP in heart failure patients could help identify those at risk of developing renal dysfunction, prompting earlier interventions to prevent further morbidity and mortality. Significant renal impairment affects therapy and results for most patients treated with acute decompensated heart failure.[28]

Conclusion

Simultaneous measurement of NT-pro BNP and kidney function

parameters in heart failure patients provides valuable prognostic information. Such screening could guide timely interventions to prevent the progression of cardio-renal syndrome. Therefore, NT-pro BNP levels may be useful for predicting AKI risk and for management and fluid balance, along with eGFR values. This may be useful for risk stratification in these patients and also for prognostic use in ESRD patients.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Braunwald E. Heart failure. J Am CollCardiol. 2013; 61(3):391–403.
2. Bekmurodovna TK, Erkinovna EN, Gadaevich GA, Oripovna DN, Abdujalolovna KF. Comorbid states in patients with chronic heart failure: regional level of the problem (preliminary study). J Cardiovasc Dis Res. 2020;11(2):59–65.
3. Chittineni H, Miyawaki N, Gulipelli S, Fishbane S. Risk for acute renal failure in patients hospitalized for decompensated congestive heart failure. Am J Nephrol. 2007;27(1):55–62.
4. Ahmed A, Campbell RC. Epidemiology of chronic kidney disease in heart failure. Heart Fail Clin. 2008;4(4):387–399.
5. Bansal N, Katz R, Robinson-Cohen C, Odden MC, Dalrymple L, Shlipak MG, et al. Absolute rates of heart failure, coronary heart disease, and stroke in chronic kidney disease: analysis of 3 community-based cohort studies. JAMA Cardiol. 2017; 2(3):314–318.
6. Heywood JT, Fonarow GC, Costanzo MR, Mathur VS, Wigneswaran JR, Wynne J; ADHERE Scientific Advisory Committee and Investigators. High prevalence of renal dysfunction and its impact on outcome in patients hospitalized with acute decompensated heart failure. J Card Fail. 2007; 13(6):422–430.
7. Huffman MD, Prabhakaran D. Heart failure: epidemiology and prevention in India. Natl Med J India. 2010; 23(5):283–288.
8. Mullens W, Abrahams Z, Francis GS, Sokos G, Taylor DO, Starling RC, et al. Elevated intra-abdominal pressure in advanced decompensated heart failure. J Am CollCardiol. 2008; 51(3):300–306.
9. Januzzi JL Jr, Camargo CA, Anwaruddin S, Baggish AL, Chen AA, Krauser DG, et al. The N-terminal Pro-BNP Investigation of Dyspnea in the Emergency Department (PRIDE) study. Am J Cardiol. 2005;95(8):948–954.
10. Casals G, Azzalini L, Tomas C, Alvarez-Contreras L, Roura G, Ferreira-Gonzalez I, et al. Admission B-type natriuretic peptide retains prognostic value in patients with acute coronary syndrome and preserved left ventricular ejection fraction. Int J Cardiol. 2012;158(3):459–460.
11. Verschuren F, Bonnet M, Benoit MO, Thys F, Brimioulle S, Legrand V, et al. Prognostic value of pro-B-type natriuretic peptide in acute pulmonary embolism. Thromb Res. 2013; 131(6):e235–e239.
12. García-Alvarez A, Regueiro A, Hernández J, et al. Additional value of B-type natriuretic peptide for discrimination of patients at risk for mortality after non-ST-segment elevation acute coronary syndrome. Eur Heart J Acute Cardiovasc Care. 2014; 3(2):132–140.
13. Akasheva DU, Plokhova EV, Tkacheva ON, et al. Age-related changes of left ventricular diastolic function, NT-proBNP level, and their association with leukocyte telomere length. Kardiologiia. 2015;55(5):59–65.
14. Kellett J. Prediction of mortality in patients with suspected heart failure by brain natriuretic peptide concentrations >100 pg/mL. Heart. 2006;92(10):1512–1513.
15. Nadir MA, Witham MD, Szwejkowski BR, Struthers AD. Meta-analysis of B-type natriuretic peptide's ability to identify stress-induced myocardial ischemia. Am J Cardiol. 2011;107(5):662–667.
16. Niizuma S, Iwanaga Y, Yahata T, Miyazaki S. Reno-cardiovascular biomarkers: managing chronic kidney disease and cardiovascular disease. Front Cardiovasc Med. 2017;4:1–11.
17. Khan IA, Fink J, Nass C, Chen H, Christenson R, deFilippi CR. N-terminal pro-B-type natriuretic peptide and B-type natriuretic peptide for identifying coronary artery disease and left ventricular hypertrophy in chronic kidney disease patients. Am J Cardiol. 2006; 97(10):1530–1534.
18. Satyan S, Light RP, Agarwal R. Relationships of N-terminal pro-B-natriuretic peptide and cardiac troponin T with left ventricular mass, function, and mortality in asymptomatic hemodialysis patients. Am J Kidney Dis. 2007; 50:1009–1019.
19. Paniagua R, Ventura MDJ, Díaz MA, et al. NT-proBNP, fluid overload, and dialysis modality as predictors of mortality in ESRD patients. Nephrol Dial Transplant. 2010;25:551–557.
20. Maisel AS, Krishnaswamy P, Nowak RM, et al. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. N Engl J Med. 2002; 347:161–167.
21. Januzzi JL Jr, van Kimmenade R, Lainchbury J, et al. NT-proBNP testing for diagnosis and short-term prognosis in acute destabilized heart failure. J Am CollCardiol. 2005;45:1101–1109.
22. McCullough PA, Duc P, Omland T, et al. B-type natriuretic peptide and renal function in heart failure. Am J Kidney Dis. 2003; 41(3):571–579.
23. McCullough PA, Kellum JA, Haase M, et al. Cardiorenal syndromes: pathophysiology and treatment. Am J Kidney Dis. 2011; 58(3):399–403.
24. Savoj J, Becerra B, Kim JK, et al. Cardiovascular disease in chronic kidney disease: prevention and treatment. CurrOpinNephrolHypertens. 2019; 28(6):513–522.
25. Luchner A, Burnett JC Jr, Jougasaki M, et al. Long-term pattern of brain natriuretic peptide and N-terminal pro-brain natriuretic peptide in the general population. Eur J Heart Fail. 2013;15(8):859–867.
26. Tagore R, Ling LH, Yang H, Daw HY, Chan YH, Sethi SK. Natriuretic peptides in chronic kidney disease. Clin J Am SocNephrol. 2008; 3(6):1644–1651.
27. Srisawasdi P, Vanavanan S, Charoenpanichkit C, Kroll MH. The effect of renal dysfunction on BNP and NT-proBNP. Am J ClinPathol. 2010; 133:14–23.
28. Wang J, Tan GJ, Han LN, Bai YY, He M, Liu HB. Prognostic value of NT-proBNP and glomerular filtration rate in acute heart failure. Front Cardiovasc Med. 2020;7:1–10.