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Prognostic value of short-term follow-up B-type natriuretic peptide levels after hospital discharge in patients with acute myocardial infarction

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ABSTRACT

Background: Elevated B-type natriuretic peptide (BNP) levels in patients hospitalized for acute myocardial infarction (AMI) are associated with heart failure and mortality. However, the role of BNP after hospital discharge is not clear. Therefore, we assessed the relationship between short-term follow-up BNP levels and clinical outcomes including all-cause mortality and major adverse cardiovascular events (MACE) in patients with AMI after hospital discharge.
Methods: From a prospective single-center percutaneous coronary intervention (PCI) registry, a total of 442 out of 2157 patients with AMI who had measurements for both initial and follow-up BNP levels within 2 months after discharge were retrospectively enrolled. Patients were divided into 4 groups (low-low, high-low, low-high, and high-high) according to their follow-up log-transformed BNP median values.
Results: The median follow-up period was 441 days (interquartile range [IQR], 362–861 days). Logistic regression analysis demonstrated that short-term follow-up BNP level was a significant predictor for all-cause mortality (odds ratio [OR], 2.265; 95% confidence interval [CI], 1.455–3.527) and MACE (OR, 1.43; 95% CI, 1.101–1.858) after adjustments for covariates. The initial BNP level did not predict both all-cause mortality and MACE. The group with high initial and high follow-up BNP levels was significantly associated with all-cause mortality (OR, 3.465; 95% CI, 1.122–10.700).
Conclusions: Short-term follow-up BNP level after hospital discharge was a powerful prognostic marker for all-cause mortality and MACE in patients with AMI. The combination of short-term follow-up BNP level with initial BNP level was a better predictor of all-cause mortality.

1. Introduction

Fibrous tissues replace damaged myocardial tissues as a result of inflammatory reactions and reparative processes [1,2]. This cardiac remodeling is characterized by progressive contractile dysfunction and neurohormonal activation following the release of vasoactive natriuretic peptides [3]. BNP is a highly sensitive and specific indicator of the size of myocardial infarction (MI), which can help to determine the degree of cardiovascular risk [4,5]. If left ventricular dysfunction is detected by elevated plasma BNP levels during the early phase of acute MI (AMI), a close clinical follow-up and image study with the consideration of potential therapeutic strategy are needed in order to improve the long-term clinical outcome [5]. Because the pattern of BNP change is dynamic in acute stages of AMI and depends on the condition of the heart during the follow-up period, BNP levels after 3–4 weeks of AMI have been suggested to be a reliable prognostic marker [6]. There are two typical patterns according to the time course after AMI. One is a monophasic pattern with one peak, and the other is a biphasic pattern with two peaks [7]. Several studies have found that a higher BNP level is associated with the biphasic pattern. Whereas the first peak is caused by the secretion of BNP stimulated by several humoral factors, such as angiotensin-II, interleukin-1, and endothelin-1, the second peak is related to a progressive infarct expansion and subsequent ventricular remodeling [6]. BNP has been considered to be one of the most important factors for predicting cardiovascular outcomes [8]. Although repeated measurements of BNP in a short-term period could be helpful to predict future events, the role of short-term follow-up BNP level has not been clearly established in patients with AMI. Therefore, this study aimed to assess the prognostic value of the short-term follow-up BNP level after hospital discharge in patients with AMI.

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2. Methods

2.1. Study population

Data were retrospectively analyzed using a prospective single center percutaneous coronary intervention (PCI) registry. Among 5398 patients from March 2007 to December 2014, 2157 patients were diagnosed with AMI. Patients without AMI, creatinine ≥2 mg/dL, and missing information on the initial or follow-up BNP level within 2 months after hospital discharges were excluded (Supplemental Fig. 1). A total of 442 patients were enrolled in this study. AMI was diagnosed as the third universal definition of MI [9]. Baseline left ventricular ejection fraction (LVEF) was measured with the biplane Simpson's method by transthoracic echocardiography. Patients were divided into 4 groups according to the initial and follow-up log-transformed BNP median levels (low-low, high-low, low-high, and high-high). The study protocol was approved by our Institutional Review Board. All patients provided written informed consents. The study endpoints were all-cause mortality and major adverse cardiovascular events (MACE), a composite endpoint of all death, any MI, and any revascularization.

2.2. Measurement of BNP

Blood samples were obtained at initial admission and within 2 months after hospital discharge. The median follow-up period was 26 days (interquartile range [IQR]: 15–29). Approximately 3–5 mL of blood samples were collected in plastic tubes coated with ethylene diamine tetraacetic acid. Samples were centrifuged and quantitatively measured with a point-of-care fluorescence immunoassay (Biosite, San Diego, CA, USA). The reference range of the BNP levels was 5–5000 pg/mL.

2.3. Statistical analysis

Continuous variables are expressed as mean ± standard deviation or median (IQR). Categorical variables are expressed as frequency and percentage. Highly skewed BNP levels were log-transformed to normalize the distribution. Groups were analyzed using one-way analysis of variance or the Chi-square test or Fisher's exact test. Kruskal-Wallis test was used to compare medians. After checking the predictive value of covariables in

Table 1
Baseline characteristics among 4 groups according to initial and follow-up B-type natriuretic peptides (BNP) level.

	Low-low N = 231	High-low N = 65	Low-high N = 63	High-high N = 83	p Value
Age, years	60.2 ± 11.2	70.2 ± 9.9	67.4 ± 11.5	72.7 ± 9.5	<0.001
Male	198 (85.7)	38 (58.5)	43 (68.3)	35 (42.2)	<0.001
BMI, kg/m^2	24.8 ± 3.3	23.9 ± 2.7	24.1 ± 3.0	23.2 ± 3.3	0.001
Medical history					
Hypertension	112 (48.7)	46 (70.8)	43 (68.3)	54 (65.1)	0.001
Diabetes mellitus	62 (27)	24 (36.9)	22 (34.9)	40 (48.2)	0.005
Dyslipidemia	47 (20.4)	15 (23.1)	13 (20.6)	17 (20.5)	0.973
CKD	0 (0)	0 (0)	1 (1.6)	5 (6)	0.001
Previous MI	13 (5.7)	4 (6.2)	6 (9.7)	7 (8.8)	0.572
Previous PCI	25 (10.9)	9 (13.9)	7 (11.1)	14 (16.9)	0.526
Old CVA	7 (3.1)	2 (3.2)	5 (8.2)	4 (4.9)	0.328
Current smoking	167 (72.9)	31 (48.4)	39 (62.9)	38 (47.5)	<0.001
Clinical diagnosis					
STEMI	141 (61)	16 (24.6)	55 (87.3)	41 (49.4)	<0.001
NSTEMI	90 (39)	49 (75.4)	8 (12.7)	42 (50.6)	<0.001
Initial vital sign					
SBP, mmHg	128.1 ± 30.5	133.3 ± 25.1	125.4 ± 33.4	126.9 ± 29.3	0.481
DBP, mmHg	76.1 ± 18.2	76.6 ± 15.3	73.6 ± 19.7	73.7 ± 16.0	0.596
Heart rate, rpm	75.7 ± 18.4	78.7 ± 20.1	74.1 ± 21.8	86.2 ± 20.8	<0.001
Disease extent					0.538
1-vessel disease	96 (41.6)	19 (29.2)	22 (34.9)	24 (28.9)	
2-vessel disease	76 (32.9)	26 (40)	22 (34.9)	27 (32.5)	
3-vessel disease	57 (24.7)	20 (30.8)	19 (30.2)	31 (37.3)	
Culprit lesion					
Left main	3 (1.3)	3 (4.6)	1 (1.6)	4 (4.8)	0.201
LAD	77 (33.6)	29 (44.6)	31 (49.2)	42 (50.6)	0.012
LCX	42 (18.3)	11 (16.9)	9 (14.3)	11 (13.3)	0.719
RCA	107 (46.7)	22 (33.8)	22 (34.9)	26 (31.3)	0.043
LVEF, %	53.7 ± 9.1	53.3 ± 11.2	48.2 ± 8.6	45.2 ± 11.1	<0.001
Total stent number	1.7 ± 0.9	1.8 ± 0.8	1.7 ± 0.8	2.0 ± 1.0	0.020
Mean stent diameter, mm	3.17 ± 0.44	3.04 ± 0.42	3.16 ± 0.46	2.97 ± 0.36	0.003
Total stent length, mm	39.1 ± 23.9	41.0 ± 21.7	40.4 ± 24.1	47.6 ± 23.6	0.068
Laboratory findings					
Hemoglobin, g/dL	14.4 ± 1.8	13.3 ± 2.2	13.7 ± 1.7	12.4 ± 1.7	<0.001
Total cholesterol, mg/dL	177.4 ± 40.6	175.8 ± 49.3	172.9 ± 36.9	166.4 ± 47.4	0.256
Triglyceride, mg/dL	129.8 ± 81.3	110.5 ± 57.6	131.4 ± 93.8	112.0 ± 119.9	0.223
HDL-cholesterol, mg/dL	44.6 ± 10.7	43.6 ± 10.5	43.8 ± 9.8	43.5 ± 14.0	0.847
LDL-cholesterol, mg/dL	113.6 ± 36.6	110.4 ± 40.8	112.8 ± 32.7	103.8 ± 42.9	0.260
Creatinine, ng/mL	0.86 ± 0.22	0.92 ± 0.27	0.91 ± 0.29	1.02 ± 0.36	<0.001
Glucose, mg/dL	154.1 ± 59.4	167.3 ± 80.7	181.6 ± 102.9	178.4 ± 83.6	0.019
CK-MB, ng/mL	4.82 (1.55, 25.75)	10.64 (4.78, 25.47)	4.28 (1.83, 31.12)	10.58 (3.98, 35.66)	0.002
Troponin I, ng/mL	0.515 (0.085, 4.045)	4.520 (1.530, 9.160)	0.445 (0.06, 3.490)	4.590 (0.89, 20.31)	<0.001
Initial BNP, pg/mL	17.9 (8.3, 45.1)	245.8 (179.0, 417.5)	48.0 (23.7, 89.5)	611.0 (322.6, 1271.3)	<0.001
Follow-up BNP, pg/mL	57.0 (29.5, 104.2)	114.3 (56.6, 176.7)	397.4 (274.5, 709.8)	535.8 (366.9, 808.9)	<0.001
Initial log BNP	2.93 ± 1.04	5.70 ± 0.66	3.74 ± 0.83	6.49 ± 0.90	<0.001
Follow-up log BNP	3.97 ± 0.85	4.54 ± 0.70	6.08 ± 0.53	6.38 ± 0.69	<0.001
Discharge medication					
Aspirin	216 (96.4)	62 (96.9)	60 (95.2)	80 (97.6)	0.921
Clopidogrel	194 (89.4)	56 (90.3)	54 (88.5)	72 (88.9)	0.989
Prasugrel	15 (6.9)	5 (8.1)	2 (3.3)	3 (3.7)	0.560
Statin	204 (91.1)	57 (89.1)	54 (85.7)	74 (90.2)	0.659
RAS blocker	174 (75.3)	51 (78.5)	44 (69.8)	68 (81.9)	0.363
Beta blocker	201 (89.7)	53 (82.8)	55 (87.3)	70 (85.4)	0.448

Data are presented as mean ± SD, median (interquartile range), or n (%).
BMI, body mass index; MI, myocardial infarction; PCI, percutaneous coronary intervention; CVA, cerebrovascular accident; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; SBP, systolic blood pressure; DBP, diastolic blood pressure; LAD, left anterior descending; LCX, left circumflex; RCA, right coronary artery; LVEF, left ventricular ejection fraction; HDL, high density lipoprotein; LDL, low density lipoprotein; CK-MB, creatine kinase MB fraction; BNP, B-type natriuretic peptide; RAS, renin-angiotensin system.

Table 2
Logistic regression analysis of initial and follow-up B-type natriuretic peptides (BNP) for predicting all-cause mortality and major adverse cardiovascular events.

BNP[a]	Model	All-cause mortality		MACE	
		OR (95% CI)	p Value	OR (95% CI)	p Value
Initial	Crude model	1.507 (1.221–1.860)	0.0001	1.166 (1.013–1.343)	0.033
	Model 1	1.335 (1.046–1.704)	0.0204	1.1 (0.932–1.298)	0.262
	Model 2	1.225 (0.946–1.585)	0.123	1.034 (0.868–1.231)	0.708
	Model 3	1.135 (0.874–1.474)	0.342	1 (0.837–1.196)	0.997
	Model 4	1.125 (0.862–1.468)	0.386	1.004 (0.837–1.206)	0.962
Follow-up	Crude model	2.611 (1.856–3.675)	<0.001	1.587 (1.291–1.952)	<0.001
	Model 1	2.502 (1.715–3.651)	<0.001	1.557 (1.242–1.951)	<0.001
	Model 2	2.357 (1.540–3.608)	<0.001	1.445 (1.119–1.865)	0.005
	Model 3	2.213 (1.444–3.392)	<0.001	1.411 (1.092–1.825)	0.009
	Model 4	2.265 (1.455–3.527)	<0.001	1.43 (1.101–1.858)	0.007

Model 1: adjusted for age and sex.
Model 2: adjusted for Model 1 and LVEF.
Model 3: adjusted for Model 2 and hemoglobin.
Model 4: adjusted for Model 3, BMI and creatinine.
MACE, major adverse cardiovascular events; OR, Odds ratio; CI, confidence interval; LVEF, left ventricular ejection fraction; BMI, body mass index.
[a] BNP values were log-transformed.

the univariate logistic regression analysis (Supplemental Table 1), a multivariate logistic regression analysis including significant covariables was performed to predict all-cause mortality and MACE according to the initial, follow-up BNP, and 4 BNP groups. Predictive values were tested with an unadjusted crude model and adjusted models with confounding variables; Model 1 adjusted for age and sex, Model 2 adjusted for Model 1 and LVEF, Model 3 adjusted for Model 2 and hemoglobin, and Model 4 adjusted for Model 3, body mass index (BMI), and creatinine. Kaplan-Meier survival curves and log-rank test were used to compare the survival rates among the 4 different BNP groups according to all-cause mortality and MACE. A p value <0.05 was considered statistically significant. Data analyses were performed with SAS 9.4 version (SAS Inc., Cary, NC, USA) and IBM SPSS Statistics for Windows, version 20.0 (IBM Corp, Armonk, NY).

3. Results

3.1. Baseline characteristics according to the 4 BNP groups

Table 1 summarizes the baseline characteristics of the patients. The median follow-up was 441 days (IQR, 362–861 days). Thirty-three (7.5%) patients passed away before their clinical follow-up. The mean age was 65.0 ± 12.0 years, and 314 (71.0%) patients were male. The median values of the initial and short-term follow-up BNP level after discharge were 55.9 (IQR: 15.2, 215.0) and 120.4 pg/mL (IQR: 49.1, 296.8), respectively. The high-high BNP group was older, increased number of females, higher incidence of diabetes, and increased heart rate. The baseline LVEF was significantly lower in the high-high BNP group. There were no differences of discharge medications including renin-angiotensin system blocker and beta blocker.

3.2. Association between BNP and all-cause mortality and MACE

A logistic regression analysis demonstrated that short-term follow-up BNP level was significantly associated with all-cause mortality (odds ratio [OR], 2.611; 95% confidence interval [CI], 1.856–3.675) and MACE (OR, 1.587; 95% CI, 1.291–1.952) in the crude model (Table 2). After adjusting the covariables in the multiple logistic regression analysis, follow-up BNP level was still a significant independent predictor for all-cause mortality (OR, 2.265; 95% CI, 1.455–3.527) and MACE (OR, 1.43; 95% CI, 1.101–1.858). However, the initial BNP level had no significant predictive value for all-cause mortality and MACE in the adjusted model.

3.3. Association between the 4 BNP groups, all-cause mortality, and MACE

The high-high BNP group (high initial and follow-up BNP levels) was an independent predictor for all-cause mortality (OR, 3.465; 95% CI, 1.122–10.700) in adjusted model 4 (Table 3). A Kaplan-Meier survival analysis showed the high-high BNP group had a significantly higher

Table 3
Logistic regression analysis for predicting all-cause mortality and major adverse cardiovascular events according to 4 B-type natriuretic peptides (BNP) groups.

Model	BNP[a]	All-cause mortality		MACE	
		OR (95% CI)	p Value	OR (95% CI)	p Value
Crude model	Low-low	1		1	
	High-low	NA	0.968	0.604 (0.224–1.633)	0.321
	Low-high	2.934 (0.979–8.795)	0.055	2.679 (1.354–5.301)	0.005
	High-high	8.275 (3.462–19.783)	<0.001	3.125 (1.693–5.770)	<0.001
Model 1	Low-low	1		1	
	High-low	NA	0.963	0.521 (0.186–1.454)	0.213
	Low-high	1.946 (0.606–6.247)	0.263	2.408 (1.187–4.882)	0.015
	High-high	5.815 (2.168–15.599)	<0.001	2.738 (1.361–5.509)	0.005
Model 2	Low-low	1		1	
	High-low	NA	0.962	0.052 (0.177–1.422)	0.195
	Low-high	1.917 (0.569–6.461)	0.294	2.073 (0.987–4.354)	0.054
	High-high	4.238 (1.415–12.688)	0.01	2.155 (1.010–4.598)	0.047
Model 3	Low-low	1		1	
	High-low	NA	0.962	0.488 (0.172–1.386)	0.178
	Low-high	2.008 (0.592–6.806)	0.263	2.036 (0.971–4.272)	0.06
	High-high	3.597 (1.191–10.860)	0.023	1.992 (0.926–4.286)	0.078
Model 4	Low-low	1		1	
	High-low	NA	0.961	0.495 (0.173–1.415)	0.190
	Low-high	1.974 (0.580–6.721)	0.277	2.057 (0.977–4.333)	0.058
	High-high	3.465 (1.122–10.700)	0.031	2.084 (0.945–4.598)	0.069

MACE, major adverse cardiovascular events; OR, odds ratio; CI, confidence interval.
[a] BNP values were log-transformed.

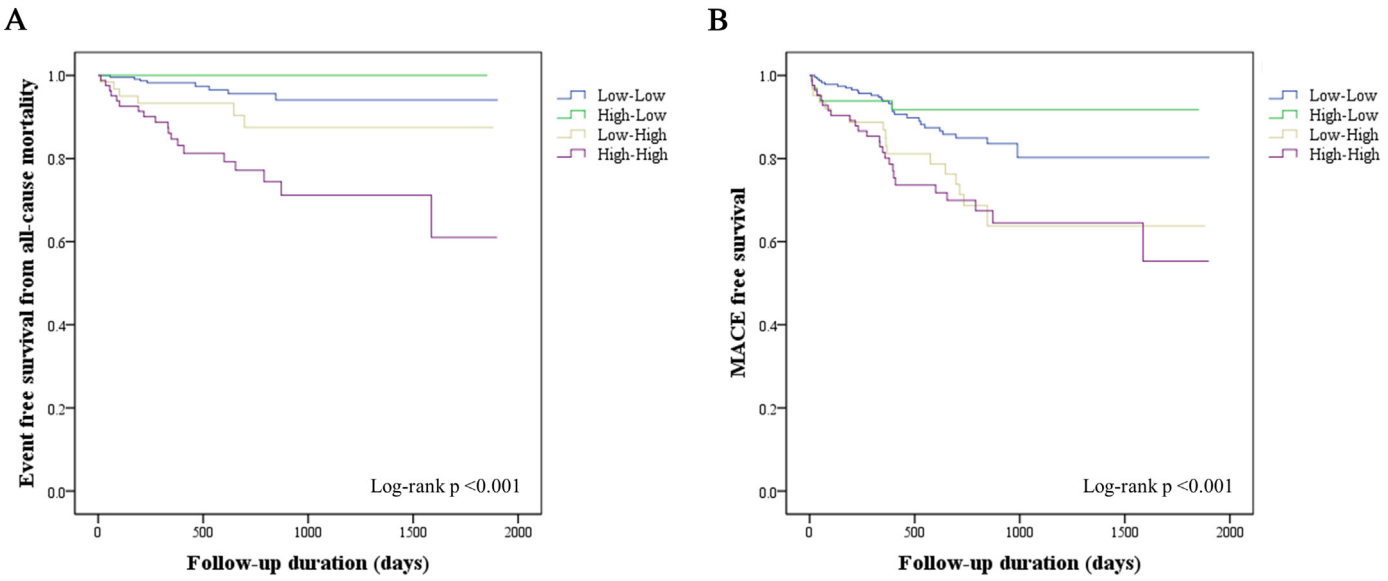

Fig. 1. Kaplan-Meier survival analysis for all-cause mortality (A) and MACE (B) stratified by 4 different BNP groups.

rate of all-cause mortality and MACE compared to the other groups (Log-rank p < 0.001) (Fig. 1).

4. Discussion

In this study, the short-term follow-up BNP level within 2 months after hospital discharge was a significant determinant for predicting all-cause mortality and MACE in patients after AMI. Moreover, the combination of follow-up BNP level with initial BNP level could better predict all-cause mortality. In contrast, the predictive value of the initial BNP level disappeared after adjusting for the covariables.

BNP and NT-proBNP are useful prognostic markers in patients with acute decompensated heart failure (HF), chronic stable HF, acute coronary syndrome, and AMI [6,8,10,11]. However, it is not clear which time period for the BNP measurement would be better for predicting poor clinical outcomes. Omar and Guglin reported that discharge BNP was a better predictor of 6-month mortality than initial BNP [12]. Kociol et al. also demonstrated that discharge BNP was the best predictor among admission, discharge, or change from admission to discharge BNP in patients with HF [13]. When BNP levels were repeatedly measured at least 2 times during hospitalization, the highest follow-up BNP level provided better a predictive value of short-term death [14]. During outpatient follow-up in patients with acute coronary syndrome (ACS), elevated levels of BNP at 4 and 12 months were significantly associated with death and new HF. Moreover, serial changes of BNP levels from baseline to 4 months provided a prognostic value for clinical outcomes [15]. A Norwegian sub-study on district treatment of ST-elevation MI reported that repeated measurements of NT-proBNP at 3 months was a stronger predictor of left ventricular dysfunction than acute measurements at 3 days [16]. In our study, we focused on whether the shorter (2 month) BNP measurement after discharge could predict mortality and MACE. As a result, BNP at 2 months and serial change of BNP were significant predictors for poor clinical outcomes.

Current updated HF guideline recommends that baseline and pre-discharge BNP measurements can be useful for establishing prognosis [17]. However, the role of BNP after discharge has still not been established. Several studies previously reported the prognostic value of BNP after discharge [15,16,18]. Moreover, serial measurements and a combination of initial and short-term follow-up BNP levels have a better prognostic value. Our results suggest that routine check-up of BNP level in a short-term period should be considered in patients with AMI for predicting prognosis. A large-scaled, multicenter prospective study is warranted to confirm this result.

5. Limitations

This study has some inherent limitations. First, this was a single-center retrospective study. Therefore, selection bias and confounding variables could not be controlled. Second, there was no scheduled follow-up image study in most of the patients. Third, this study evaluated only short-term follow-up BNP levels, which might not reflect the longer-term clinical outcomes.

6. Conclusion

Short-term follow-up BNP levels after hospital discharge was a powerful prognostic biomarker for all-cause mortality and MACE in patients with AMI. The combination of short-term follow-up BNP level after discharge with initial BNP level provided better a predictive value for all-cause mortality.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2019.01.026.

Statement of authorship

These authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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Declaration of conflicting interests

The authors report no relationships that could be constructed as a conflict of interest.

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