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Original Article

Prognostic and Diagnostic Value of ProBNP in Iranian Patients With Chest Pain at a Tertiary Cardiovascular Center

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ABSTRACT

Background: Diagnosis and early treatment of the cardiac causes of chest pain are of particular importance. This study aimed to investigate the association between NT-pro-BNP levels as a cardiac marker and the prognosis of patients with chest pain.

Methods: All patients visiting the emergency department of a tertiary cardiovascular center with chest pain between October 2016 and March 2017 were evaluated for eligibility. Demographic data, proBNP levels, final diagnosis on angiography, echocardiography, and other symptoms were recorded.

Results: A total of 222 patients at a mean age of 59.0±14.8 years were studied. Totally, 127 patients (57.2%) were male. A significant inverse relationship was found between proBNP levels and the left ventricular ejection fraction (r= -0.316; P<0.001). NT-proBNP levels showed a significant elevation in patients with abnormal size and function of the right ventricle, with regional wall motion abnormalities, and with valvular heart diseases (P<0.05). The BNP level in patients with abnormal angiographic results was 1148.5 (405.3–3214.0), significantly higher than that in patients with normal results (545.0: 90.3–2807.8; P=0.009). The level of this marker in patients with obstructive coronary artery disease (1192.0: 438.8–3233.0) was significantly higher than that in patients with non-obstructive coronary artery disease (620.0: 108.0–2792.0; P=0.001). BNP>841 pg/mL had a sensitivity of 92.9% and a specificity of 47.9% in identifying cases at risk of complications.

Conclusions: NT-proBNP could be a good diagnostic and prognostic marker for patients with chest pain complaints. Measuring this marker upon arrival can help identify patients with cardiac diseases. It is recommended to evaluate patients with elevated levels of this marker for earlier diagnosis and treatment. ***(Iranian Heart Journal 2023; 24(2): 23-34)***

KEYWORDS: NT-proBNP, Chest pain, Prognostic value

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Chest pain (CP) is a major challenge for physicians due to its possible fatal causes. [1] The source of CP can be cardiac (CCP) or non-cardiac (NCCP). The cause of CP of non-cardiac origin can be pulmonary, gastrointestinal, musculoskeletal, or psychological. [2] CP has a wide range of potential causes, about 22% of which is myocardial ischemia. [3] For a better diagnosis of CP, 3 criteria are considered: substernal CP, exertional CP, and CP relieved by rest or nitroglycerin sublingual tablets. [1] If there are all 3 criteria, it is considered typical chest pain (TCP), if there are 2 criteria, it is considered atypical chest pain (ATCP), and if only 1 of the criteria is observed, it is regarded as non-angina. [4,5] Nonetheless, the patterns of CP with cardiac origin differ in type and severity, making diagnosis challenging. On the other hand, the early diagnosis of underlying causes leads to timely management and plays a key role in reducing complications. [6,7]

Prohormone of brain natriuretic peptide (BNP) is continuously produced in small quantities by the heart. ProBNP degradation results in the release of 2 products, one of which is a 32-amino acid peptide called "BNP", which is a biologically active, and a non-biologically active N-terminal-pro brain natriuretic peptide (NT-proBNP). NT-proBNP is increased more in older patients, due to exacerbated diastolic dysfunction, increased heart size, and other underlying causes. [8] The principal function of BNP is to help maintain and control blood volume and regulate blood flow throughout the body. It also prevents the retention of excess sodium and water in the body. BNP levels rise in response to increased myocardial stretch and wall stress, as well as ischemia. [9] Serum BNP levels are associated with the severity of heart failure (HF) and have a prognostic value shown to be effective in various studies. [10] High serum BNP levels are a stronger predictor of death in women than men. [11] Plasma BNP levels are used clinically to guide the management of patients with HF and cardiac dysfunction and are also drawn upon as prognostic indicators that can help clinicians adjust their therapy strategy. [11]

CCP has more mortality, morbidity, and cost to patients and society. In the present study, we sought to evaluate the prognostic value of proBNP in patients with CP as a prognostic marker.

METHODS

Patients

The present retrospective cross-sectional and diagnostic value study investigated the prognostic value of proBNP in patients with CP. All patients visiting the Emergency Department of Rajaie Cardiovascular Medical and Research Center in Tehran with a complaint of CP between October 2016 and March 2017 were enrolled in the study after the provided informed and written consent. Patients' information, including age, sex, occupation, final diagnosis, and other related symptoms was collected. CP diagnosis was based on the patient's own statements. Patients with acute coronary syndromes, septic shock, and atrial fibrillation with rapid ventricular response were excluded from the study.

Electrocardiography (ECG), Echocardiography, and Coronary Angiography (CAG)

Standard 12-lead ECG (GE MAC1200, GE Healthcare, Fairfield, Connecticut, USA) was assessed at a paper speed of 25 mm/s. Twelve-lead ECGs were examined by 2 independent observers, blinded to clinical data. Additionally, transthoracic echocardiographic examinations were done using a 5-1 MHz transducer (Philips CX50; Philips, Amsterdam, Netherlands). The

conventional technique was used to analyze the angiographic data of all the patients.

Biomarker Measurement

After the collection of venous blood from all patients, serum NT-proBNP levels were assessed with an Elecsys 2010 analyzer (a commercially available electrochemiluminescent sandwich immunoassay) (Elecsys proBNP; Roche Diagnostics, Mannheim, Germany) at the Laboratory of Rajaie Cardiovascular Medical and Research Center. The amount of proBNP after the test was expressed in pg/mL. The BNP assay analytical range was 5–35000 pg/mL (0.6–4130 pmol/L), defined as the limit of detection and the maximum of the master curve.

Statistical Analysis

The SPSS software, version 25, was used to analyze the data. Due to the non-normal distribution of proBNP, non-parametric tests (ie, the Mann–Whitney *U* and the Kruskal–Wallis) were employed to compare quantitative variables. The Spearman correlation test was used to evaluate the correlation between proBNP and quantitative variables. The χ^2 test was applied to compare nominal variables. Receiver operating characteristic (ROC) analysis and the area under the curve (AUC) were utilized to evaluate the diagnostic value of proBNP. A *P* value <0.05 was considered a significant level.

RESULTS

Patient characteristics

The study population consisted of 222 patients with CP at a mean ± standard deviation (SD) age of 59.0±14.8 years. Additionally, 127 (57.2%) were male, and 95 (42.8%) were female. The risk factors of coronary artery disease (CAD) were hypertension in 140 patients (63.1%), family history of CAD in 98 (44.3%), diabetes mellitus in 87 (39.2%), dyslipidemia in 77 (34.8%), and smoking in 74 (33.5%).

CP types were TCP in 77 patients (34.7%) and ATCP in 145 (65.3%). Dyspnea was reported in 129 patients (58.4%) and syncope in 6 (2.7%). A previous history of CAD (22.1%) and chronic kidney disease (19.4%) was the most common underlying disease. The mean systolic and diastolic blood pressures were 124.0±13.3 mm Hg and 78.8±9.1 mm Hg, respectively. The mean heart rate of the patients was 79.0±11.5 bpm, and the mean oxygen saturation level (O_2Sat) was 96.7±1.8%.

ECG, Echocardiography, and CAG

Eight patients underwent pulmonary computed tomography angiography, and 176 patients underwent CAG. The angiographic results were normal in 63 patients (28.4%), mild CAD in 29 (13.1%), single-vessel disease in 31 (14.0%), double-vessel disease in 30 (13.5%), and triple-vessel disease in 61 (27.5%). Eight patients (3.6%) with pulmonary thromboembolism were reported (Table 2). The median length of hospital stay was 3 (2–5) days. The treatment plan considered in 142 patients (64%) was a medical follow-up, in 54 (24.3%) percutaneous coronary intervention (PCI), in 19 (8.6%) coronary artery bypass graft surgery, and in 7 (3.2%) catheter-directed thrombolysis. Twenty-eight patients (12.6%) had at least 1 complication; 4 patients (1.8%) had cardiac shock, 17 (7.7%) had decompensated HF, 4 (1.8%) had cardiac tamponade, 1 (0.5%) had hemodialysis, 6 (2.7%) had bleeding during the first 72 hours as hematoma at the site of angiography, and 6 (2.7%) had gastrointestinal bleeding.

The echocardiographic findings of the patients can be seen in Table 3.

ProBNP levels

The median (IQR) level of NT-proBNP in the study population was 1047.5 (317.8–

3092), which was in the normal range in 34 patients (15.3%) and abnormal in 188 (84.7%).

The results of the Spearman correlation test indicated a direct and significant relationship between age and the NT-proBNP level (r=0.182; P=0.007). The BNP level was significantly different between patients with and without dyspnea (P=0.006). The BNP level in patients with chronic kidney disease was significantly higher than that in other patients (Table 1).

No significant relationships were observed between BNP and systolic blood pressure (r= -0.042, P=0.536), diastolic blood pressure (r=0.065; P=0.335), heart rate (r=0.013; P=0.849), and O_2Sat (r=0.12; P=0.074). Comparisons of the Dunn test results between the 2 groups showed that the BNP level in patients with normal right ventricular size was significantly lower than that in patients with mild enlargement (adjusted P=0.026) and moderate enlargement (adjusted P=0.004). No significant difference was found in the comparison of the other 2 groups (adjusted P=1.000). In addition, the BNP level in patients with normal right ventricular function was significantly lower than that in patients with mild dysfunction (adjusted P<0.001), moderate dysfunction (adjusted P<0.001), and severe dysfunction (adjusted P<0.001).

Significant inverse relationships were found between the BNP level and the CK-MB level after 6 hours, lipid profile (triglyceride, cholesterol, and low-density lipoprotein), and the hemoglobin level. The relationships between BNP and renal markers (blood urea nitrogen and creatinine), white blood cells, and inflammatory factors (C-reactive protein, the neutrophil-to-lymphocyte ratio, and the platelet-to-lymphocyte ratio) were direct and significant.

A pairwise comparison of the diagnostic groups showed that the BNP level in patients with normal angiography was significantly lower than in patients with triple-vessel disease (adjusted P=0.013). Using the ROC analysis, the area under the curve (AUC) for the diagnosis of abnormal angiographic findings was 0.613 (95% CI, 0.525 to 0.701), showing the poor diagnostic value of BNP (P=0.009): A cut point >475 pg/mL had a sensitivity of 70.0% and specificity of 43.5%, whereas a cut point >2017pg/mL had a sensitivity of 36.9% and specificity of 71.0% (Fig. 1a).

AUC for the BNP level in the diagnosis of obstructive CAD was 0.631 (95% CI, 0.553 to 0.708), showing the poor diagnostic value of BNP (P=0.001): A cut point >590 pg/mL had a sensitivity of 70.5% and a specificity of 50.0%, while a cut point of 975 pg/mL had a sensitivity of 60.7% and a specificity of 59.8% in distinguishing obstructive CAD from non-obstructive CAD (Fig. 1b).

A direct and significant relationship was found between the BNP level and the length of hospital stay (r=0.351; P<0.001). The BNP level in patients with complications during hospitalization was 2951.5 (1190.3–5744.0), significantly higher than that in patients without complications (936.0: 278.5–2784.8; P<0.001). In the ROC analysis, AUC was 0.731 (95% CI, 0.644 to 0.817), indicating the relatively good diagnostic value of BNP in identifying cases at risk of complications (P<0.001): A cut point > 841 pg/mL had a sensitivity of 92.9% and a specificity of 47.9%. (Fig. 1c).

Table 1: BNP level in patients with CP

		BNP Level		*P* value
		Yes	No	
Comorbidity	CAD	1280.0 (344.5-4117)	1021.0 (294.5-2947.0)	0.19
	MI	501.5 (226.0-3603.8)	1052.5 (341.8-3102.0)	0.537
	Stroke	1820.5 (556.0-3286.5)	1031.5 (297.5-3083.0)	0.317
	CKD	1790.0 (577.0-4694.0)	980.0 (233.0-2796.0)	0.006
	COPD	985.0 (469.5-3009.8)	1052.5 (297.5-3102.0)	0.649
	Dyslipidemia	1079.5 (3055.5-325)	1046 (311.5-3115)	0.647
	HTN	986.5 (2801.5-363.5)	1188.5 (3745.8-201)	0.791
	DM	1021 (3233.0-383)	1066 (290.0-3024)	0.489
Family History of CAD		1072.5 (305.3-3083)	1022 (326.8-3111)	0.759
Smoking		1144.5 (2529.3-263.5)	1011 (3139-326)	0.871
Dyspnea		1374 (3442.5-438.5)	604 (2241.0-198)	0.006
CP		**TCP**	**ATCP**	0.424
		1042 (374-3608)	1049 (271.5-2852)	
Sex		**Female**	**Male**	0.385
		938 (2796-296)	1175 (3233-353)	

CP, Chest pain; TCP, Typical chest pain; ATCP, Atypical chest pain; CKD, Chronic kidney disease; COPD, Chronic obstructive pulmonary disease; HTN, Hypertension; DM, Diabetes mellitus; CAD, Coronary artery disease

Table 2: CAG findings in patients with CP

	CAG	N (%)	Median (IQR)	*P* value
Indication	PPCI NSTEMI UA Elective	12 (6.9) 52 (29.9) 49 (28.2) 61 (35.1)	530.0 (365.0-3391.0) 1787.0 (448.3-3598.5) 920.0 (296.5-1548.5) 1499.0 (331.0-4489.0)	0.173
Result	Normal Abnormal	63 (28.4) 159 (71.6)	545.0 (90.3-2807.8) 1148.5 (405.3-3214.0)	0.009
	Normal Mild Single-vessel disease Double-vessel disease Triple-vessel disease PTE	63 (28.4) 29 (13.1) 31 (14.0) 30 (13.5) 61 (27.5) 8 (3.6)	542.0 (88.0-2799.0) 700 (194.5-2435.5) 1002.0 (383.0-1932.0) 977.0 (433.0-3644.8) 1784 (507.5-3507.0) 2588.5 (1628.5-7987.3)	0.004
	Obstructive CAD Non-obstructive CAD	100 (45.0) 122 (55.0)	1192 (438.8-3233.0) 620 (108.0-2792.0)	0.001

CP, Chest pain; CAD, Coronary artery disease; PPCI, Primary percutaneous coronary intervention; NSTEMI, Non–ST-elevation myocardial infarction; UA, Unstable angina; PTE, Pulmonary thromboembolism

Table 3: Echocardiographic findings in patients with chest pain

Echocardiography		Mean ± SD	ProBNP Level	
			r	*P* value
Ejection fraction		13.2 ± 36.8	-0.316	<0.001
Systolic pulmonary artery pressure		14.3 ± 34.7	0473	<0.001
		N (%)	Median (IQR)	p-value
Regional wall motion abnormality	Yes No	107 (48.4) 115 (51.6)	14458.0 (418.0-3438.0) 801.5 (150.3-2807.8)	0.008
Heart valve disease	Yes No	84 (37.8) 138 (62.2)	2599.0 (1085.8-5784.3) 503.0 (181.0-1906.3)	<0.001
Right ventricular Size	Normal Mild Enlargement Moderate	159 (71.9) 32 (14.5) 21 (9.5)	870.0 (232.0-2278.0) 2017 (524.0-5357.3) 2704 (1359.5-6107.0)	0.001

	Enlargement Severe Enlargement	9 (4.1)	2780 (458.5-3097.0)	
Right ventricular function	Normal Mild dysfunction Moderate dysfunction Severe dysfunction	92 (41.6) 49 (22.2) 63 (28.4) 17 (7.7)	412 (109.5-1180.3) 1748 (501.0-5447.5) 1656 (938.0-3233.0) 3123 (1909.0-5058.0)	<0.001

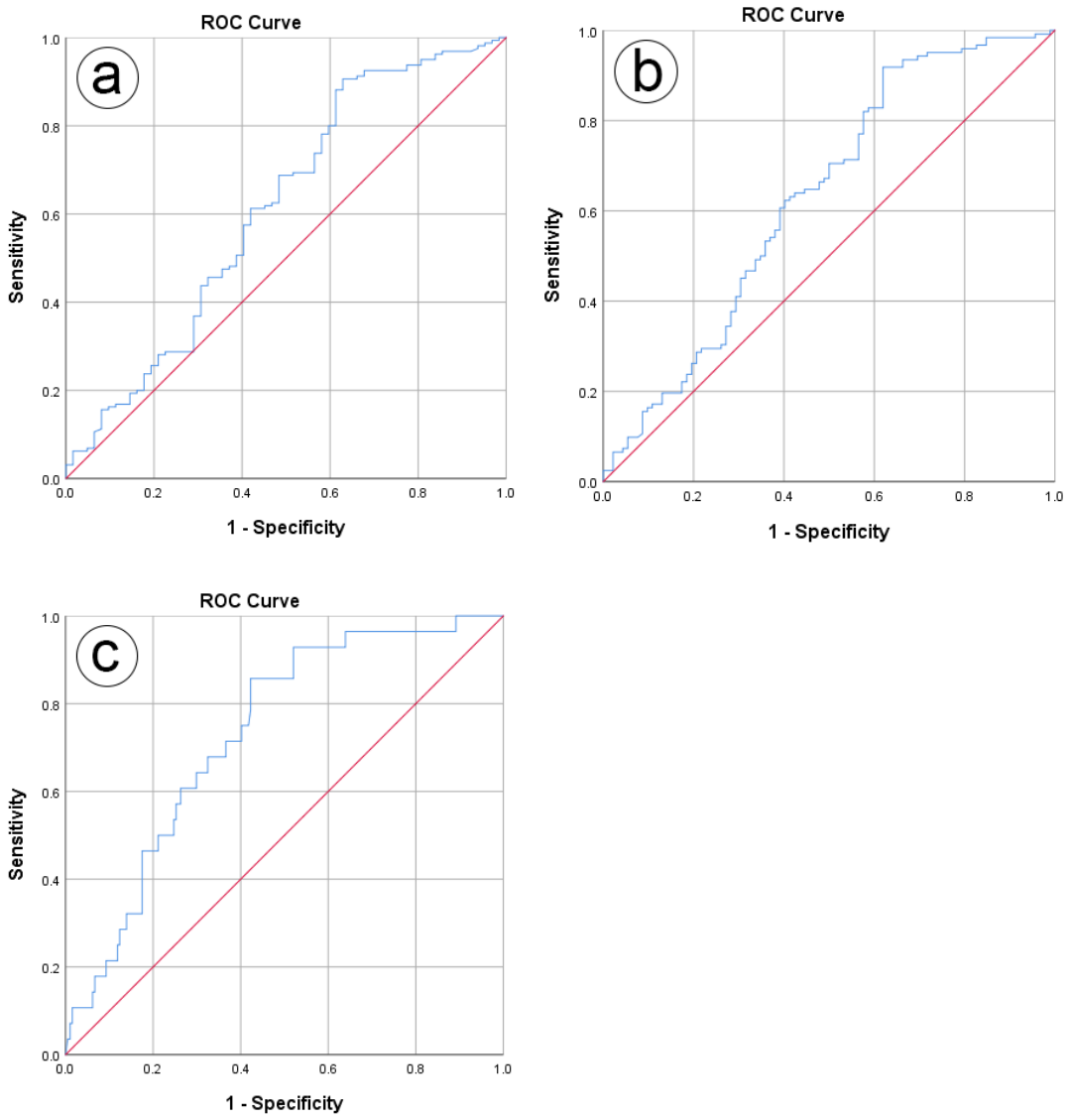

Figure 1: a) The image illustrates the ROC curves for the BNP diagnostic value in identifying abnormal angiographic cases. **b)** The image shows the ROC curve for the diagnostic value of BNP in identifying obstructive CAD cases in angiography. **c)** The image illustrates the ROC curve for the BNP value in identifying cases at risk of complications during hospitalization.
ROC, Receiver operating characteristic; BNP, Brain natriuretic peptide; CAD, Coronary artery disease

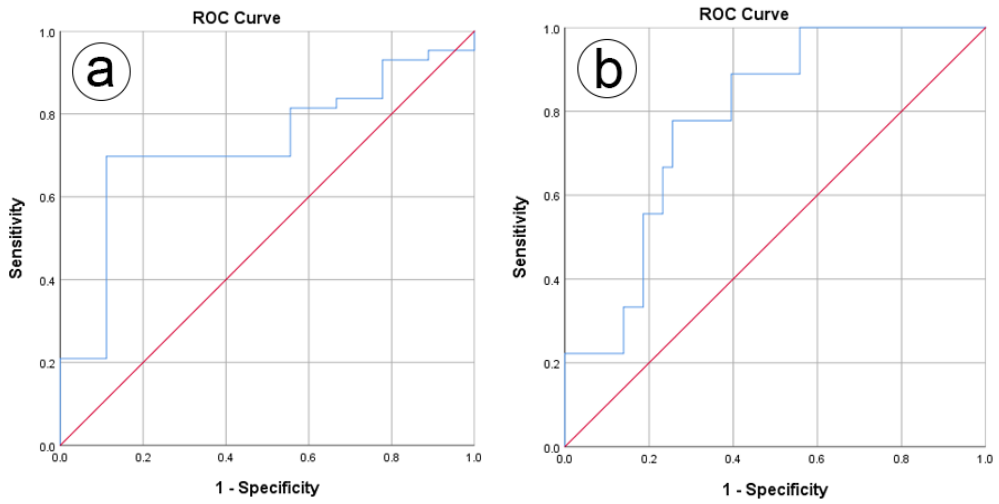

Figure 2: a) The image shows the ROC curve for the diagnostic value of BNP in identifying obstructive CAD in patients with NSTEMI indication. **b)** The image demonstrates the ROC curve for the diagnostic value of BNP in identifying complications during hospitalization in patients with NSTEMI indication.

ROC, Receiver operating characteristic; BNP, Brain natriuretic peptide; CAD, Coronary artery disease; NSTEMI, Non–ST-elevation myocardial infarction

Correlation between BNP and angiographic outcomes in patients with primary PCI

The results of angiography in all primary PCI patients were abnormal and obstructive CAD; therefore, only the prognostic value of proBNP for complications in patients in this group was evaluated. The median (IQR) BNP level was 3567.0 (440.0–3567.0) in 3 patients with complications and 418.0 (344.5–2530.0) in 9 uncomplicated patients (*P*=0.165).

Correlation between BNP and angiographic outcome in patients with Non–ST-elevation myocardial infarction (NSTEMI)

The relationship between BNP and the angiographic outcome of patients with NSTEMI was abnormal in 47 patients (90.4%) and normal in 5 (9.6%). The median (IQR) BNP level was 884.0 (595.5–3153.5) in patients with normal angiography and 1853.0 (442.0–3649.0) in those with abnormal results (*P*=0.566). The median (IQR) BNP level was 884.0 (595.5–3153.5) in patients with a normal diagnosis, 261.0 (154.8-677.8) in patients with mild CAD, 1463.0 (447.8–10251.0) in patients with single-vessel disease, 2594.0 (1367.5–6340.0) in patients with double-vessel disease, and 1821.5 (476.8–3497.5) in patients with triple-vessel disease (*P*=0.112). The median (IQR) BNP level in patients with obstructive CAD was 1944.0 (480.0–3711.0), significantly higher than that in patients with non-obstructive CAD (776.0: 261.0 to 901.5; *P*=0.034).

ROC analysis for the diagnostic value of BNP in identifying obstructive CAD showed that AUC was 0.726 (95% CI, 0.560 to 0.892), indicating a relatively good diagnostic value for the BNP level (*P*=0.034): A cut point >960.5pg/mL had a sensitivity of 69.8% and a specificity of 88.9%.

The median (IQR) BNP level in patients with complications was 3649.0 (2223.5–13972.0), significantly higher than that in uncomplicated patients (1021.0: 383.0–3024.0; *P*=0.008). The AUC in the ROC curve was 0.783 (95% CI, 0.643 to 0.923), showing a relatively good diagnostic value for the BNP level (*P*=0.008): A cut point

>2544.5 pg/mL had a sensitivity of 77.8% and a specificity of 74.4.

Correlation between BNP and angiographic outcomes in patients with unstable angina

Angiographic results were abnormal in 46 (93.9%) and normal in 3 (6.1%) patients with unstable angina. The median (IQR) BNP level was 489.0 (34.0–489.0) in patients with normal angiographic results and 920.5 (296.8–1503.3) in patients with abnormal results (*P*=0.770). The median (IQR) BNP level was 489.0 (34.0–489.0) in patients with a normal diagnosis, 353.0 (73.5–2596.5) in patients with mild CAD, 579.5 (248.0–1116.5) in patients with single-vessel disease, 966.5 (646.0–2229.5) in patients with double-vessel disease, and 1280.0 (712.5–1945.0) in patients with triple-vessel disease (*P*=0.173). In addition, the median (IQR) BNP level in patients with obstructive CAD was 971.0 (423.5–1548.5), not significantly different from patients with non-obstructive CAD (421: 71.3–3434.3; *P*=0.143). The median (IQR) BNP level was 1639.0 (469.5–8382.0) in patients with complications and 846.0 (296.3–1260.5) in patients without complications (*P*=0.261).

Association between the BNP level and angiographic outcomes in elective patients

Angiographic results were abnormal in 27 (44.3%) and normal in 34 (55.7%) elective patients. The median (IQR) BNP level was 1509.5 (125.3.0–3965.3) in patients with normal outcomes in angiography and 1175.0 (577.0–4694.0) in patients with abnormal outcomes (*P*=0.561). The median (IQR) BNP level was 1509.5 (125.3.0–3965.3) in patients with normal diagnosis, 1079.0 (577.0–3460.0) in patients with mild CAD, 1932.0 (710.5–6883.5) in patients with single-vessel disease, 705.5 (236.0–705.5) in patients with double-vessel disease, and 4694.0 (673.0–9979.0) in patients with triple-vessel disease (*P*=0.604). Furthermore, the median (IQR) BNP level in patients with obstructive CAD 1553.5 (542.5–4694.0) did not significantly differ from that of the non-obstructive CAD group (1499.0: 220.0–3655.0; *P*=0.404). The median (IQR) BNP level in patients with complications was 2796.0 (1122.0–4736.0) and 1349.0 (235.0–4400.0) in uncomplicated patients (*P*=0.175).

DISCUSSION

The relationship between the level of NT-proBNP marker and CP was investigated in patients visiting the Emergency Department of Rajaie Cardiovascular Medical and Research Center. The mean age of the patients in this study was 59 years, and more than half of them were male. We found a direct and significant relationship between age and NT-proBNP, but no significant relationship was found between sex and NT-proBNP.

A well-known relationship exists between age and the NT-proBNP level. [8,12] Nevertheless, contradictory findings exist regarding the relationship between NT-proBNP and the sex of patients. [8,12] In contrast to the results of our study, some studies have shown higher levels of NT-proBNP in females. [8,12] Estrogen in women apparently regulates the NT-proBNP level, such that in tandem with aging, the difference in the estrogen level narrows between men and women and perhaps even disappears in ages over 80. [8,13]

While a prior investigation reported that a higher level of NT-proBNP was associated with smoking, diabetes mellitus, hypertension, and dyslipidemia, [13] our results demonstrated no significant associations between the NT-proBNP level and vascular risk factors, including a family history of CAD, smoking, dyslipidemia, hypertension, and diabetes mellitus. Likewise, Kotecha et al [14] found no

associations between smoking and diabetes mellitus and elevated levels of NT-proBNP.

We found high levels of NT-proBNP in patients with chronic kidney disease compared with other patients; however, a history of CAD, MI, stroke, and chronic obstructive pulmonary disease had no significant relationship with the NT-proBNP level. Previous studies have also shown no association between a history of CAD and MI and elevated levels of NT-proBNP. [14]

In our study, 65% of the patients had ATCP, and 58.4% had dyspnea. We did not find a significant difference between NT-proBNP levels in patients with ATCP and TCP. Still, the NT-proBNP level was significantly higher in patients with dyspnea, which may be indicative of cardiac causes due to increased NT-proBNP levels. [15-17]

Our results demonstrated a significant inverse relationship between the BNP level and lipid profile (triglyceride, cholesterol, and low-density lipoprotein) and hemoglobin levels. Similarly, many studies have shown that cholesterol levels could be significantly higher in patients with normal levels of NT-proBNP than in patients with increased NT-proBNP. [14,18,19] In contrast, Yang et al [13] showed a direct relationship between the low-density lipoprotein level and increased NT-proBNP. The exact cause of low levels of NT-proBNP in patients with dyslipidemia has yet to be elucidated. [19]

We observed a significant relationship between BNP and renal function markers (blood urea nitrogen and creatinine), white blood cells, C-reactive protein, the neutrophil-to-lymphocyte ratio, and the platelet-to-lymphocyte ratio. Unlike our study, Kotecha et al [14] found no significant relationships between C-reactive protein and NT-proBNP. Some studies on patients with HF have also failed to show a link between C-reactive protein and NT-proBNP. [20] The discrepancy between the results of our study and other previous studies requires future studies to determine the association between NT-proBNP and inflammatory markers and prognosis in patients with coronary artery disease.

One of the principal objectives of the current study was to investigate the relationship between NT-proBNP and echocardiographic findings. We found a significant inverse relationship between the NT-proBNP level and the left ventricular ejection fraction on echocardiography. The association between NT-proBNP and systolic pulmonary artery pressure was direct and significant. Moreover, the NT-proBNP level exhibited a significant rise in patients with abnormal size and function of the right ventricle, patients with regional wall motion abnormality, and patients with heart valve dysfunction. These findings confirm the increase in NT-proBNP levels in patients with anatomical and functional heart disorders. The inverse relationship between the left ventricular ejection fraction and the NT-proBNP level has been demonstrated in many previous studies. [12,14] Chiming in with our study, several studies have shown increased NT-proBNP levels in patients with right ventricular enlargement and dysfunction, as well as increased pulmonary pressure. [21-23]

Another principal objective of the present study was to investigate the relationship between NT-proBNP and angiographic findings and prognosis in patients with CP. Our findings showed a significant difference in the NT-proBNP level: The level was significantly lower in patients with a normal angiographic diagnosis than in those with a triple-vessel diagnosis. In general, patients with abnormal angiographic diagnoses had much higher NT-proBNP levels than those with normal outcomes. NT-proBNP levels above 475 pg/mL had a sensitivity of 70% and a specificity of 43.5% for the detection of abnormal angiographic results. We divided the study population into obstructive CAD and non-obstructive CAD groups. NT-

proBNP levels were significantly higher in the obstructive CAD group than in the non-obstructive CAD group. Based on elevated plasma NT-proBNP levels >590 pg/mL as an indicator to diagnose obstructive CAD, a sensitivity of 70.5% and a specificity of 50% were obtained. The relationships between the NT-proBNP level and the length of hospital stay and complications during hospitalization were direct and significant. Patients with complications had much higher levels of NT-proBNP than other patients. In ROC analysis, BNP >841 pg/mL had a sensitivity of 92.9% and a specificity of 47.9% in identifying cases at risk of complications.

Consistent with the findings of our study, the diagnostic and prognostic value of BNP in patients with suspected cardiac complications with and without HF has been demonstrated in some previous studies. [13,24,25] Yang et al [13] showed a relationship between higher BNP levels and mixed calcified coronary plaques and significant coronary stenosis. Some investigations have reported more cases of repeated coronary stenosis in patients with higher levels of BNP. [26-28] In a study by Azevedo et al, [24] patients with CP complaints, higher BNP levels were reported in patients with ischemic myocardial perfusion scintigraphy than in other non-ischemic patients. Kawabe et al [29] showed high levels of BNP in patients with obstructive CAD compared with patients with non-obstructive CAD.

In conclusion, our findings showed that the NT-proBNP level could be a good diagnostic and prognostic marker for patients with CP complaints. Measuring this marker on arrival can help identify cardiac abnormalities. Due to the significant association between NT-proBNP and abnormal findings in echocardiography and angiography, it is recommended to evaluate patients with elevated NT-proBNP levels for earlier diagnosis and treatment. In addition, NT-proBNP can be very helpful in identifying patients at risk of complications during hospitalization.

Acknowledgments

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Conflict of Interest

None of the authors declared any conflicts of interest.

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