Herz 2018 · 43:548–554
https://doi.org/10.1007/s00059-017-4598-6
Received: 5 March 2017
Revised: 18 May 2017
Accepted: 21 June 2017
Published online: 13 July 2017

Y. Karabağ[1] · I. Rencuzogullari[1] · M. Çağdaş[1] · S. Karakoyun[1] · M. Yesin[2] · M. Uluganyan[3] · M. O. Gürsoy[4] · İnanç Artaç[1] · Doğan İliş[1] · Tayyar Gökdeniz[5] · S. Ç. Efe[6] · O. Taşar[7] · H. İ. Tanboğa[8]

[1] Department of Cardiology, Kafkas University Medical Faculty, Kars, Turkey
[2] Department of Cardiology, Kars Harakani State Hospital, Kars, Turkey
[3] Department of Cardiology, Yedikule Chest Diseases and Chest Surgery Education and Research Center, Istanbul, Turkey
[4] Department of Cardiology, Gaziemir State Hospital, İzmir, Turkey
[5] Department of Cardiology, Medipol University Medical Faculty, İstanbul, Turkey
[6] Department of Cardiology, Ağrı State Hospital, Ağrı, Turkey
[7] Department of Cardiology, Elazığ Education and Research Hospital, Elazığ, Turkey
[8] Department of Cardiology, Ataturk University Medical Faculty, Erzurum, Turkey

Association between BNP levels and new-onset atrial fibrillation

A propensity score approach

New-onset atrial fibrillation (NOAF), a common complication of acute ST-segment elevation myocardial infarction (STEMI), has a reported incidence of 2.3–21% [1]. NOAF is a strong predictor of poor prognosis after STEMI and an independent predictor of mortality [2–4]. Although the exact cause of NOAF has not been identified, ischemia, reduced atrial perfusion, and increased left ventricular end-diastolic pressure (LVEDP) and left atrial (LA) pressure are considered to be the most likely underlying mechanisms [5, 6].

To date, several clinical and laboratory parameters have been found to be associated with NOAF in patients with STEMI. The plasma level of B-type natriuretic peptide (BNP), a neurohormone released from ventricular myocytes in response to acute volume and/or pressure overload, is associated with the severity of left ventricular dysfunction, impaired hemodynamic parameters, and increased LVEDP [7, 8]. The plasma BNP level is an independent predictor of short- and long-term prognosis, reduced left ventricular ejection fraction (LVEF), and negative remodeling in patients with STEMI [9, 10]. Although reduced LVEF is associated with NOAF development in patients with STEMI, data regarding the relationship between the plasma BNP level and NOAF development are limited. The aim of the present study was to evaluate the predictive value of plasma BNP levels in NOAF development in STEMI patients treated with primary percutaneous coronary intervention (pPCI).

Patients and methods

Study population

We retrospectively enrolled 1,928 patients with STEMI who underwent pPCI at Kafkas University, Turkey, and Erzurum Ataturk University, Turkey, between January 2010 and September 2016. STEMI was defined on basis of the following criteria: ongoing ischemic symptoms (within 12 h of presentation); typical increase or decrease in cardiac biomarkers; a new ST elevation in two or more contiguous leads, with readings of at least 0.2 mV in leads V_1, V_2, and V_3, or at least 0.1 mV in the remaining leads; or newly developed left bundle-branch block pattern [11]. Coronary angiography and pPCI were performed according to standard protocols. Before the procedure, all patients received anticoagulant therapy with unfractionated heparin and dual-antiplatelet therapy with aspirin and clopidogrel. Coronary blood flow patterns before and after primary pPCI were thoroughly evaluated on the basis of thrombolysis in myocardial infarction (TIMI) flow grade, using grades 0, 1, 2, and 3 [12]. Patients with atrial fibrillation (AF) diagnosed or documented prior to admission (66 patients) were excluded from the study. The electrocardiograms (ECGs) of all patients included in the study showed normal sinus rhythm on admission. Additionally, patients treated with emergent coronary artery bypass graft surgery (CABG) or with a previous history of CABG (163 patients), and patients with clinical and long-term follow-up data missing from hospital files and computer records (642 patients), were excluded from the study. Thus, the final study population comprised 1,057 patients. Long-term follow-up data were obtained from hospital records and telephone interviews. Information from the Statistics Institute and the Registrar of Birth Office was used to determine if the unreachable patients were deceased. The study protocol was reviewed and approved by the Local Ethics Committee of the Kafkas University in accordance with the Declaration of Helsinki.

Table 1 Demographic, clinical, laboratory, and coronary angiographic characteristics of all patients, patients with NOAF, and patients without NOAF

	All patients (*N* = 1,057)		New-onset atrial fibrillation (NOAF)				*p*
			Patients without NOAF		Patients with NOAF		
Age (years)	57	±13	56	±12	62	±13	0.004
Male gender, *n* (%)	846	(80)	804	(80.5)	42	(72.4)	0.135
Diabetes mellitus, *n* (%)	254	(24)	230	(23)	24	(41.4)	0.001
Hypertension, *n* (%)	474	(44.8)	435	(43.5)	39	(67.2)	<0.001
Dyslipidemia, *n* (%)	425	(40.2)	407	(40.7)	18	(31.0)	0.142
Peripheral arterial disease, *n* (%)	186.0	(17.6)	170.0	(17.0)	16.0	(27.6)	0.040
Family history, *n* (%)	230	(21.8)	222	(22.2)	8	(13.8)	0.130
Smoking, *n* (%)	556	(52.6)	535	(53.6)	21	(36.2)	0.01
Previous medication							
ASA, *n* (%)	26	(2.5)	24	(2.4)	2	(3.4)	0.617
Clopidogrel, *n* (%)	2	(0.2)	2	(0.2)	0	(0)	0.733
Beta-blocker, *n* (%)	86	(8.1)	77	(7.7)	9	(15.5)	0.034
ACEI or ARB, *n* (%)	231	(21.9)	217	(21.7)	14	(24.1)	0.665
Statin, *n* (%)	197	(18.6)	190	(19)	7	12.1	0.186
SBP (mm Hg)	134	±35	133	±34	132	±40	0.792
Heart rate (bpm)	77	±17	77	±16	82	±21.4	0.084
Killip class >1 on admission (%)	218	(20.6)	193	(19.3)	25	(43.1)	<0.001
Hemoglobin (g/dl)	13.6	±1.9	13.6	±1.8	13	±2.4	0.041
C-reactive protein (mg/dl)	10.8	5.8–18.3	9.8	5.7–17.6	18.7	12–32.5	0.001
eGFR (ml/min)	85.7	±26.5	86.4	±26.1	73.4	±30.2	0.002
Peak CK-MB (U/l)	176	94.00–322	169	91–303	365	211–444	<0.001
Peak troponin I (ng/ml)	84	38.8–187	78	36–181.5	171.5	89.6–278	<0.001
BNP pg/ml	72.5	38–135	70.7	36.7–129	161	72.3–432	<0.001
LVEF (%)	46.4	±8.7	46.9	±8.5	39.2	±8.8	<0.001
LMCA disease	15	(1.4)	15	(1.5)	0	(0)	0.347
IRA of LAD (ml/min)	568	(53.7)	533	(53.4)	35	(60.3)	0.478
Post-PCI TIMI grade 3, *n* (%)	913	(86.4)	873	(87.4)	40	(69)	<0.001
Duration of telemetric monitoring (days)	2	1.50–3.00	2	1.50–3.00	3	2.00–5.00	<0.001

ASA acetyl salicylic acid, *ACEI* angiotensin-converting enzyme inhibitor, *ARB* angiotensin II receptor blocker, *SBP* systolic blood pressure, *eGFR* estimated glomerular filtration rate, *CK-MB* creatine kinase-myocardial band, *LVEF* left ventricular ejection fraction, *IRA* infarct-related artery, *LAD* left anterior descending, *LMCA* left main coronary artery

Data collection

Baseline clinical and demographic characteristics and patient medical history data were obtained from the hospital records. Complete blood count and blood biochemical parameters were measured in all patients on admission. Blood samples were retested for troponin T and creatine kinase myocardial band (CK-MB) every 6 h until peak levels were detected, and these tests, together with hemograms and creatinine tests, were repeated daily thereafter. BNP plasma concentrations were measured using a microparticle enzyme immunoassay with an AQT90 FLEX analyzer (Radiometer Medical Aps, Åkandevej, Denmark) on admission. The ECGs, which were obtained via surface ECG or monitor/defibrillator records (with an ECG readout of at least 30 s), were recorded at admission, in the intensive care unit 48–72 h after pPCI, and during hospitalization. NOAF was defined as arrhythmia developing after hospital admission that included irregular RR intervals on ECG; absence of identifiable P waves, with an unidentifiable isoelectric line; and atrial rhythm of >300 bpm [13]. LVEF, defined as the postprocedural ejection fraction, was assessed using a modified Simpson's method [14].

Statistical analysis

SPSS version 22.0 (SPSS Inc., Chicago, Ill.) was used for statistical analysis. Continuous and categorical variables are expressed as mean ± standard deviation and percentages, respectively. Differences in patient characteristics between the patients with and without NOAF were analyzed using the *t* test or Mann–Whitney *U* test for continuous variables, and the chi-square test for categorical variables. Because the study was nonrandomized,

Herz 2018 · 43:548–554 https://doi.org/10.1007/s00059-017-4598-6

Y. Karabağ · I. Rencuzogullari · M. Çağdaş · S. Karakoyun · M. Yesin · M. Uluganyan · M. O. Gürsoy · İ. Artaç · D. İliş · T. Gökdeniz · S. Ç. Efe · O. Taşar · H. İ. Tanboğa

Association between BNP levels and new-onset atrial fibrillation. A propensity score approach

Abstract

Background. New-onset atrial fibrillation (NOAF), a common complication of acute ST-segment elevation myocardial infarction (STEMI), is associated with a poor prognosis. Several clinical and laboratory parameters are reported to be associated with NOAF in patients with STEMI. The aim of the present study was to evaluate the predictive value of plasma B-type natriuretic peptide (BNP) levels for NOAF development and long-term prognosis in STEMI patients undergoing primary percutaneous coronary intervention (pPCI).

Patients and methods. We retrospectively enrolled 1,928 patients with STEMI who underwent pPCI. After applying exclusion criteria, 1,057 patients were retained in the final study population. Patients with NOAF were compared with patients without NOAF in the entire study population and in a matched group.

Results. Patients with NOAF had a significantly higher average plasma BNP level (161 pg/ml, range: 72.3–432) than patients without NOAF in the study population (70.7 pg/ml, range: 70–129; $p < 0.001$) and in the matched group (104.6 pg/ml, range: 47.2–234.5; $p = 0.014$). Furthermore, the plasma BNP level was found to be an independent predictor of NOAF development (odds ratio [OR]: 1.003; 95% confidence interval [CI]: 1.000–1.005; $p = 0.034$) and mortality in the long-term follow-up (OR: 1.004; 95% CI: 1.002–1.006; $p < 0.001$).

Conclusion. The present study found that a high plasma BNP level was significantly associated with NOAF development in STEMI patients, and was an independent predictor of NOAF development and all-cause mortality during long-term follow-up, regardless of other NOAF risk factors.

Keywords

B-type natriuretic peptide · Atrial fibrillation · ST-segment elevation myocardial infarction · Prognostic factors · Percutaneous coronary intervention

Zusammenhang zwischen BNP-Werten und neu aufgetretenem Vorhofflimmern. Ein Propensity-Score-Ansatz

Zusammenfassung

Hintergrund. Neu aufgetretenes Vorhofflimmern („new-onset atrial fibrillation", NOAF), eine häufige Komplikation des akuten ST-Strecken-Hebungs-Infarkts (STEMI), geht mit einer schlechten Prognose einher. Verschiedene klinische und Laborparameter sollen mit NOAF bei STEMI-Patienten in Zusammenhang stehen. Ziel der vorliegenden Studie war es, den prädiktiven Wert der Werte für BNP („B-type natriuretic peptide") für das Auftreten von NOAF und die Langzeitprognose bei STEMI-Patienten nach primärer perkutaner Koronarintervention (pPCI) zu untersuchen.

Patienten und Methoden. Retrospektiv wurden 1928 Patienten mit STEMI und nach pPCI in die Studie aufgenommen. Nach Anwendung der Ausschlusskriterien verblieben letztlich 1057 Patienten in der Studienpopulation. Dabei wurden in der gesamten Studienpopulation und einer entsprechend ausgewählten Gruppe Patienten mit NOAF im Vergleich zu Patienten ohne NOAF betrachtet.

Ergebnisse. Die Patienten mit NOAF wiesen einen signifikant höheren durchschnittlichen Plasma-BNP-Wert auf (161 pg/ml, Spannbreite: 72,3–432) als Patienten ohne NOAF in der Studienpopulation (70,7 pg/ml, Spannbreite: 70–129; $p < 0{,}001$) und in der gematchten Gruppe (104,6 pg/ml, Spannbreite: 47,2–234,5; $p = 0{,}014$). Darüber hinaus stellte sich der Plasma-BNP-Wert als ein unabhängiger Prädiktor des Auftretens von NOAF (Odds Ratio, OR: 1,003; 95%-Konfidenzintervall, 95%-KI: 1,000–1,005; $p = 0{,}034$) und der Mortalität während der Langzeitnachbeobachtung heraus (OR: 1,004; 95%-KI: 1,002–1,006; $p < 0{,}001$).

Schlussfolgerung. In der vorliegenden Studie wurde festgestellt, dass ein hoher Plasma-BNP-Wert in signifikanter Weise mit dem Auftreten von NOAF bei STEMI-Patienten in Zusammenhang steht und sich als unabhängiger Prädiktor des Auftretens von NOAF und der Gesamtmortalität während der Langzeitnachbeobachtung, unabhängig von Risikofaktoren für NOAF, herausstellte.

Schlüsselwörter

Natriuretisches Peptid vom B-Typ · Vorhofflimmern · ST-Strecken-Hebungs-Infarkt · Prognostische Faktoren · Perkutane Koronarintervention

a logistic regression model with propensity scores (exact matching) was created with variables that were shown to be associated with NOAF in patients with STEMI to balance patient characteristics and to perform propensity-matched analysis of the patients with and without NOAF. The variables used in this model were as follows: age, sex, diabetes mellitus (DM), hypertension (HT), smoking, LVEF, C-reactive protein (CRP) level, Killip class on admission, systolic BP, infarct-related artery (IRA), postprocedural TIMI grade, and peak CK-MB. In order to evaluate the value of variables in predicting NOAF, we performed logistic regression (stepwise backward elimination) analysis. Receiver operating characteristics (ROC) curve analysis of plasma BNP levels was performed to calculate the optimal cut-off value for predicting NOAF. Survival curves were calculated using the Kaplan–Meier method, and statistical significance was assessed using log-rank tests. Cox regression analyses were used to identify associations between BNP level and all-cause long-term mortality.

Results

The study population consisted of 1,057 STEMI patients (mean age: 57 ± 13 years; 80% male) who underwent pPCI. NOAF developed in 58 patients (5.4%) during the index hospitalization. Demographic,

Table 2 Demographic, clinical, laboratory and coronary angiographic characteristics of patients with NOAF and without NOAF (*p* value in matched group)

	Patients with NOAF (*n* = 57)		Patients without NOAF (*n* = 58)		*p*
Age (years)	60	±12.7	62	±13.3	0.648
Male gender, *n* (%)	45	(80.5)	42	(72.4)	0.356
Diabetes mellitus, *n* (%)	23	(41.5)	24	(41.4)	0.993
Hypertension, *n* (%)	35	(61)	39	(67.2)	0.521
Dyslipidemia, *n* (%)	17	(29.3)	18	(31.0)	0.851
Family history, *n* (%)	8	(14.6)	8	(13.8)	0.906
Peripheral arterial disease, *n* (%)	17.0	(29.3)	16.0	(27.6)	0.855
Smoking, *n* (%)	26	(46.3)	21	(36.2)	0.311
Previous medication					
ASA, *n* (%)	3	(4.9)	2	(3.4)	0.722
Clopidogrel, *n* (%)	1	(2.4)	0	(0)	0.232
Beta-blocker, *n* (%)	3	(4.9)	9	(15.5)	0.097
ACEI or ARB, *n* (%)	11	(19.5)	14	(24.1)	0.586
Statin, *n* (%)	10	(17.1)	7	(12.1)	0.482
SBP (mm Hg)	134	±41	132	±40	0.770
Heart rate (bpm)	74	±21	82	±21	0.081
Killip class >1 on admission (%)	17	(31.7)	25	(43.1)	0.251
Hemoglobin (g/dl)	13.7	±1.7	13	±2.4	0.065
C-reactive protein (mg/dl)	11	5.9–23.2	18.7	12–32.5	0.138
eGFR (ml/min)	80.2	±32.8	73.4	±30.2	0.296
Peak CK-MB (U/l)	189	114–442	365	211–444	0.200
Peak troponin I (ng/ml)	94	56.7–211.7	171	89.6–278	0.114
BNP pg/ml	104.6	47.2–234.5	161	72.3–432	0.014
LVEF (%)	42.6	±9.6	39.2	±8.8	0.079
LMCA disease	1	(2.4)	0	(0)	0.232
IRA of LAD, *n* (%)	35	(61)	35	(60.3)	0.498
Post-PCI TIMI 3 grade, *n* (%)	48	(85.4)	40	(69)	0.140
Duration of telemetric monitoring (days)	3	2.00–3.50	3	2.00–5.00	0.103

ASA acetyl salicylic acid, *ACEI* angiotensin-converting enzyme inhibitor, *ARB* angiotensin II receptor blocker, *SBP* systolic blood pressure, *eGFR* estimated glomerular filtration rate, *CK-MB* creatine kinase-myocardial band, *LVEF* left ventricular ejection fraction, *IRA* infarct-related artery, *LAD* left anterior descending, *LMCA* left main coronary artery

clinical, laboratory, and coronary angiographic characteristics of all patients, patients with NOAF, and patients without NOAF are listed in **Table 1**. Plasma BNP levels were higher in patients with NOAF than in patients without NOAF (161 pg/ml, range: 72.3–432, vs. 70.7 pg/ml, range: 70–129; $p < 0.001$). Compared with patients without NOAF, patients with NOAF were older and had a more frequent history of DM, HT, and smoking. Furthermore, compared with patients without NOAF, patients with NOAF had a higher Killip class on admission; longer telemetric monitoring time; lower postprocedural TIMI grade; higher white blood cell (WBC) count; higher levels of C-reactive protein (CRP), peak CK-MB, and troponin; and lower hemoglobin, estimated glomerular filtration rate (eGFR), and LVEF.

To account for the marked differences between the number of patients with and without NOAF and to eliminate the effects of the variables that were previously found to be associated with NOAF, we performed propensity score matching. In all, 57 patients were selected from among the patients without NOAF, and matched with 58 patients with NOAF. In the matched population, we found that the plasma BNP level was significantly higher in patients with NOAF than in patients without NOAF (161.0 pg/ml, range: 72.3–432.0, vs. 104.6 pg/ml, range: 47.2–234.5,; $p = 0.014$). In addition, no statistically significant difference was found between patients with and without NOAF in terms of duration of telemetric monitoring (3 days, range: 2.0–3.5, vs. 3 days, range: 2.0–5.0; $p = 0.103$).The demographic, clinical, and laboratory characteristics of the matched group are listed in **Table 2**.

In the matched group, the plasma BNP level was significantly associated with NOAF (odds ratio [OR]: 1.003; 95% confidence interval [CI]: 1.000–1.005; $p = 0.034$) and was found to be an independent predictor of NOAF development. ROC curve analysis was performed to determine the plasma BNP cut-off value for predicting NOAF in the matched group. The cut-off value of the plasma BNP level for NOAF prediction was 262.66 pg/ml, with a sensitivity of 41.4% and a specificity of 82.9% (area under the curve: 0.623; 95% CI: 0.510–0.731; $p = 0.041$; **Fig. 1**).The positive predictive value of the plasma BNP level was 73.68% with a 95% CI of 63.99–81.53; the negative predictive value was 49.15% with a 95% CI of 40.61–57.74.

The mean follow-up time was 33.16 ± 13.2 months. Eighteen patients (15.7%) died during the long-term follow-up. The incidence of all-cause long-term mortality in patients with NOAF was significantly higher than that in patients without NOAF – 14 patients (24.1%) vs. 4 patients (7%); $p = 0.015$ (**Fig. 2**) – and the plasma BNP level was identified as an independent predictor of mortality in the long-term follow-up period (OR: 1.004; 95% CI: 1.002–1.006; $p < 0.001$).

Discussion

In this study, we found that STEMI patients with NOAF had a higher plasma BNP level, which was shown to be a strong independent predictor of NOAF development during hospitalization. Furthermore, to our knowledge, this is the first study to show that the plasma BNP level

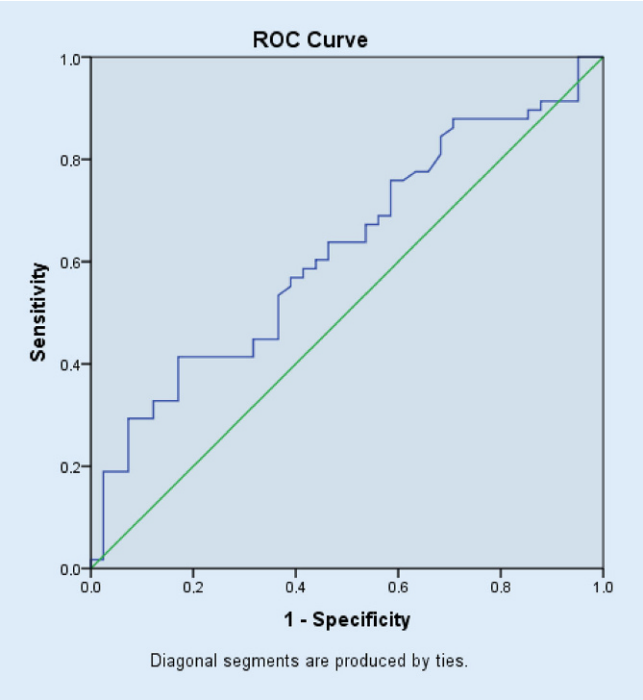

Fig. 1 ◀ ROC graphic depicting best cut-off value of B-type natriuretic peptide (BNP) level for new-onset atrial fibrillation (NOAF) prediction. The cut-off value of BNP level for NOAF prediction was 262.6 with a sensitivity of 41.4% and a specificity of 82.9% (AUC: 0.621; 95% CI: 0.510–0.731; $p = 0.041$)

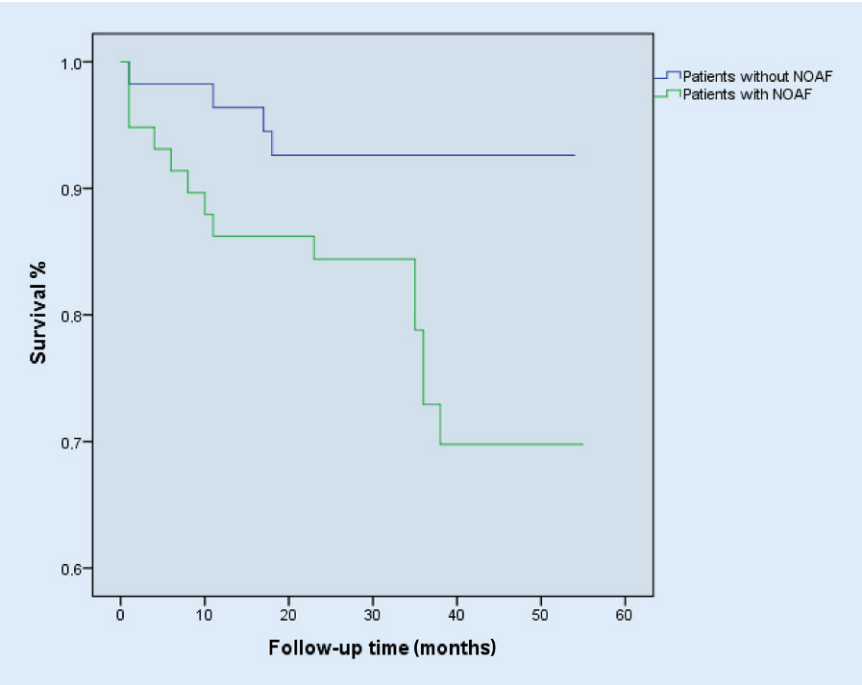

Fig. 2 ▲ Kaplan–Meier survival curve

in patients with STEMI is a predictor of NOAF development and a predictor of mortality in long-term follow-up, independent of other NOAF risk factors.

Any AF event that newly developed during the hospitalization of patients with acute STEMI is associated with a worse prognosis and with higher short-, mid-, and long-term mortality rates in patients undergoing thrombolysis or pPCI [2, 3, 15]. Several clinical parameters have been associated with NOAF development in patients with STEMI. Consistent with the results of previous studies [1–5], we found that the following were associated with increased NOAF development in all study groups: older age; history of HT, DM, or smoking; higher Killip class; and extensive myocardial damage (higher peak CK-MB, reduced LVEF, imperfect reperfusion, and TIMI <3). In the present study, we also observed that patients with NOAF had a reduced eGFR. Although the data are insufficient to show a definite relationship between reduced eGFR and AF in patients with STEMI, persistent inflammation and oxidative stress, frequent observations of ECG abnormalities (prolonged PR and QT intervals), and continuous activation of the renin–angiotensin–aldosterone system in patients with renal failure may facilitate AF development [16]. Additionally, in our study, peripheral arterial disease (PAD) was observed at a significantly higher frequency in patients with NOAF. Previous studies have shown that the frequency of PAD is twofold higher in patients with AF [17]. Although PAD is also an independent predictor of AF in women [18], this is the first study to show that PAD is associated with NOAF in STEMI irrespective of sex. Moreover, in the present study, patients with NOAF had low hemoglobin and high CRP levels, which is consistent with the results of previous studies [19–21].

In addition to being directly associated with systolic or diastolic dysfunction, plasma BNP levels are also associated with acute coronary syndromes, advanced age, renal dysfunction, pulmonary disease, AF, and high-output states such as sepsis and intensive inflammation [22]. In the present study, plasma BNP levels were higher in patients with NOAF; this finding could be attributed to myocardial dysfunction or additional factors such as advanced age, extensive myocardial damage or inflammation, and decreased eGFR. Asanin et al. have previously demonstrated the relationship between BNP and NOAF [19] in patients with STEMI. In their study, the patients were divided into low and high BNP groups according to the most useful BNP cut-off level for the prediction of NOAF development. Patients in the high BNP group had a greater number of NOAF risk factors, including older age, DM, anterior wall infarction,

>1 Killip class on admission, higher troponin level, and lower eGFR and LVEF. In the present study, to eliminate the effect of these clinical factors on NOAF development and plasma BNP levels, we compared NOAF patients with 57 matched subjects selected from patients without NOAF according to clinical factors that were shown to be associated with NOAF development (age, DM, HT, smoking, LVEF, IRA, Killip class on admission, systolic BP, postprocedural TIMI grade, and peak CK-MB) by using propensity score matching. Statistical analysis of the matched population revealed that only the plasma BNP level was higher in patients with NOAF. Thus, regardless of other NOAF risk factors, the plasma BNP level was shown to be an independent predictor of NOAF in patients with STEMI.

The relationship between BNP and NOAF could be explained by several mechanisms. STEMI leads to alterations in LV composition, architecture, and myocardial stiffness, resulting in changes in diastolic function [23]. Diastolic dysfunction with elevated LVEDP is associated with high plasma BNP levels [8] and leads to a restrictive filling pattern after myocardial infarction, which could facilitate AF development in patients with STEMI. Moreover, even after successful reperfusion of the epicardial coronary artery, high LVEDP results in decreased perfusion pressure, impaired microvascular flow and subendocardial oxygen delivery, and sympathetic and neurohormonal activation [24–26]. Consequently, these patients are more likely to develop atrial ischemia [27], high atrial pressure [28], and autonomic nervous system alterations [29] that cause structural and functional remodeling in atria.

Predicting NOAF is crucial because NOAF leads to a worse prognosis in patients with STEMI by causing arrhythmic LV contractions and decreasing coronary flow. It also decreases LVEF, leads to heart failure, and exacerbates ventricular arrhythmias [1–6]. Similar to the predictors described in previous studies, measurement of plasma BNP levels may help identify these patients at an early stage of STEMI.

Limitations

NOAF frequency data were unavailable because screening tests were not performed to detect silent AF attacks during hospitalization. The inability to identify silent/asymptomatic paroxysmal AF episodes in patients prior to admission is also a limitation of this study.

Conclusion

The present study demonstrated that a high plasma BNP level was significantly associated with NOAF development in patients with STEMI, and was an independent predictor of NOAF development, regardless of other NOAF risk factors. To our knowledge, this is the first reported study to comprehensively examine the relationship between NOAF development and plasma BNP levels in patients with STEMI.

Corresponding address

Dr. I. Rencuzogullari
Department of Cardiology, Kafkas University Medical Faculty
Kars, Turkey
rencuzog@gmail.com

Compliance with ethical guidelines

Conflict of interest. Y. Karabağ, I. Rencuzogullari, M. Çağdaş, S. Karakoyun, M. Yesin, M. Uluganyan, M.O. Gürsoy, İ. Artaç, D. İliş, T. Gokdeniz, S.Ç. Efe, O. Taşar, and H.İ. Tanboğa declare that they have no competing interests.

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. For this type of study formal consent is not required.

References

1. Schmitt J, Duray G, Gersh BJ, Hohnloser SH (2009) Atrial fibrillation in acute myocardial infarction: A systematic review of the incidence, clinical features and prognostic implications. Eur Heart J 30:1038–1045
2. Crenshaw BS, Ward SR, Granger CB et al (1997) Atrial fibrillation in the setting of acute myocardial infarction: the GUSTO-I experience. Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries. J Am Coll Cardiol 30:406–413
3. Wong CK, White HD, Wilcox RG et al (2003) Significance of atrial fibrillation during acute myocardial infarction, and its current management: insights from the GUSTO-3 trial. Card Electrophysiol Rev 7:201–207
4. Kinjo K, Sato H, Sato H et al (2003) Prognostic significance of atrial fibrillation/atrial flutter in patients with acute myocardial infarction treated with percutaneous coronary intervention. Am J Cardiol 92:1150–1154
5. Gorenek B, Kudaiberdieva G (2012) Atrial fibrillation in acute ST elevation myocardial infarction: clinical and prognostic features. Curr Cardiol Rev 8:281–289
6. Waldecker B (1999) Atrial fibrillation in myocardial infarction complicated by heart failure: cause or consequence? Eur Heart J 20:710–712
7. Wiese S, Breyer T, Dragu A et al (2000) Gene expression of brain natriuretic peptide in isolated atrial and ventricular human myocardium: influence of angiotensin II and diastolic fiber length. Circulation 102:3074–3079
8. Maeda K, Tsutamoto T, Wada A et al (1998) Plasma brain natriuretic peptide as a biochemical marker of high left ventricular end-diastolic pressure in patients with symptomatic left ventricular dysfunction. Am Heart J 135:825–832
9. De Lemos JA, Morrow DA, Bentley JH et al (2001) The prognostic value of B-type natriuretic peptide in patients with acute coronary syndromes. N Engl J Med 345:1014–1021
10. Garcia-Alvarez A, Sitges M, Delgado V et al (2009) Relation of plasma brain natriuretic peptide levels on admission for ST-elevation myocardial infarction to left ventricular end-diastolic volume six months later measured by both echocardiography and cardiac magnetic resonance. Am J Cardiol 104:878–882
11. Steg PG, James SK, Atar D et al (2012) ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J 33:2569–2619
12. Gibson CM, Cannon CP, Daley WL et al (1996) TIMI frame count: a quantitative method of assessing coronary artery flow. Circulation 93:879–888
13. Camm AJ, Kirchhof P, Lip GY et al (2010) European Heart Rhythm Association, European Association for Cardio-Thoracic Surgery Guidelines for the management of atrial fibrillation: the Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC). Eur Heart J 31:2369–2429
14. Lang RM, Bierig M, Devereux RB et al (2005) Recommendations for chamber quantification: a report from the American society of echocardiography's guidelines and standards committee and the chamber quantification writing group, developed in conjunction with the European association of echocardiography, a branch of the European society of cardiology. J Am Soc Echocardiogr 18:1440–1463
15. Pizzetti F, Turazza FM, Franzosi MG et al (2001) GISSI-3 Investigators. Incidence and prognostic significance of atrial fibrillation in acute myocardial infarction: the GISSI-3 data. Heart 86:527–532
16. Watanabe H, Watanabe T, Sasaki S et al (2009) Close bidirectional relationship between chronic kidney disease and atrial fibrillation: the Niigata preventive medicine study. Am Heart J 158:629–636
17. Naccarelli GV, Varker H, Lin J et al (2009) Increasing prevalence of atrial fibrillation and flutter in the United States. Am J Cardiol 104:1534–1539

18. Perez MV, Wang PJ, Larson JC et al (2013) Risk factors for atrial fibrillation and their population burden in postmenopausal women: the Women's Health Initiative Observational Study. Heart 99:1173–1178
19. Asanin M, Stankovic S, Mrdovic I et al (2012) B-type natriuretic peptide predicts new-onset atrial fibrillation in patients with ST-segment elevation myocardial infarction treated by primary percutaneous coronary intervention. Peptides 35:74–77
20. Hwang HJ, Ha JW, Joung B et al (2011) Relation of inflammation and left atrial remodeling in atrial fibrillation occurring in early phase of acute myocardial infarction. Int J Cardiol 146:28–31
21. Aronson D, Boulos M, Suleiman A et al (2007) Relation of C-reactive protein and new-onset atrial fibrillation in patients with acute myocardial infarction. Am J Cardiol 100:753–757
22. Daniels LB, Maisel AS (2007) Natriuretic peptides. J Am Coll Cardiol 50:2357–2368
23. St John Sutton M (2008) Quest for diastolic prognostic indicators of clinical outcome after acute myocardial infarction. Circulation 117:2570–2572
24. Kirtane AJ, Bui A, Murphy SA et al (2004) TIMI Study Group. Association of epicardial and tissue-level reperfusion with left ventricular end-diastolic pressures in ST-elevation myocardial infarction. J Thromb Thrombolysis 17:177–184
25. Claeys MJ, Bosmans J, Veenstra L et al (1999) Determinants and prognostic implications of persistent ST-segment elevation after primary angioplasty for acute myocardial infarction: importance of microvascular reperfusion injury on clinical outcome. Circulation 99:1972–1977
26. Van Herck PL, Carlier SG, Claeys MJ et al (2007) Coronary microvascular dysfunction after myocardial infarction: increased coronary zero flow pressure both in the infarcted and in the remote myocardium is mainly related to left ventricular filling pressure. Heart 93:1231–1237
27. Sinno H, Derakhchan K, Libersan D et al (2003) Atrial ischemia promotes atrial fibrillation in dogs. Circulation 107:1930–1936
28. Satoh T, Zipes DP (1996) Unequal atrial stretch in dogs increases dispersion of refractoriness conducive to developing atrial fibrillation. J Cardiovasc Electrophysiol 7:833–842
29. Coumel P (1996) Autonomic influences in atrial tachyarrhythmias. J Cardiovasc Electrophysiol 7:999–1007

Erratum

Herz 2018 · 43:554
https://doi.org/10.1007/s00059-017-4604-z
Published online: 20 July 2017

C. Gecmen[1] · G. G. Gecmen[2] · D. Ece[2] · M. Kahyaoğlu[1] · A. Kalayci[1] · C. Y. Karabay[3] · O. Candan[1] · M. E. Isik[1] · F. Yilmaz[1] · O. Akgun[1] · M. Celik[1] · I. A. Izgi[1] · C. Kirma[1] · S. Keser[2]

[1] Department of Cardiology, Kartal Kosuyolu Heart & Research Hospital, Istanbul, Turkey
[2] Dr. Lutfi Kirdar Education and Research Hospital, Istanbul, Turkey
[3] Dr. Siyami Ersek Chest and Cardiovascular Surgery Hospital, Istanbul, Turkey

Erratum to: Cytopathology of pericardial effusions

Experience from a tertiary center of cardiology

Erratum to:
Herz (2017)
DOI 10.1007/s00059-017-4596-8

In the above mentioned article, the affiliations of four authors were not given correctly. The correct affiliations of all authors are as follows:

C. Gecmen[1], G.G. Gecmen[2], D. Ece[2], M. Kahyaoğlu[1], A. Kalayci[1], C.Y. Karabay[3], O. Candan[1], M.E. Isik[1], F. Yilmaz[1], O. Akgun[1], M. Celik[1], I.A. Izgi[1], C. Kirma[1], S. Keser[2]

[1]Department of Cardiology, Kartal Kosuyolu Heart & Research Hospital, Istanbul, Turkey

[2]Dr. Lutfi Kirdar Education and Research Hospital, Istanbul, Turkey

[3]Dr. Siyami Ersek Chest and Cardiovascular Surgery Hospital, Istanbul, Turkey

The authors and publisher apologize for this mistake.

Corresponding address

C. Gecmen, MD
Department of Cardiology, Kartal Kosuyolu Heart & Research Hospital
34846 Istanbul, Turkey
koronerr@hotmail.com

The online version of the original article can be found under doi: 10.1007/s00059-017-4596-8