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NT-proBNP for prognostic and diagnostic evaluation in patients with acute coronary syndromes

Vladimir Zdravkovic[1], Violeta Mladenovic[2], Maja Colic[3], Dragic Bankovic[3], Zorica Lazic[2], Marina Petrovic[2], Ivan Simic[1], Slavko Knezevic[2], Sveto Pantovic[4], Aleksandar Djukic[2], Nemanja Zdravkovic[3]

[1]Department of Cardiology, Clinical Centre Kragujevac, Faculty of Medical Sciences, University of Kragujevac, Kragujevac, Serbia
[2]Clinic for Internal Medicine, Clinical Centre Kragujevac, Kragujevac, Serbia
[3]Centre for Molecular Medicine and Stem Cell Research, Faculty of Medical Sciences, University of Kragujevac, Kragujevac, Serbia
[4]Department of Gynaecology and Obstetrics, Clinical Centre of Serbia, Belgrade, Serbia

Abstract

Background and aim: N terminal-proB-type natriuretic peptide (NT-proBNP) is synthesised and secreted from the ventricular myocardium. This marker is known to be elevated in patients with acute coronary syndromes (ACS). We evaluated NT-proBNP as a significant diagnostic marker and an important independent predictor of short-term mortality (one month) in patients with ACS.

Methods: NT-proBNP and cardiac troponin I (cTI) were assessed in 134 consecutive patients (median age 66 years, 73% male) hospitalised for ACS in a cardiological university department. The patients were classified into ST-elevation ACS (STE-ACS, n = 74) and non-ST-elevation ACS (NSTE-ACS, n = 60) groups based on the ECG findings on admission. Patients with Killip class ≥ II were excluded.

Results: The serum level of NT-proBNP on admission was significantly higher (p < 0.0005), while there was no difference in cTI serum level in the NSTE-ACS patients compared to STE-ACS patients. There was a significant positive correlation between NT-proBNP and cTI in the NSTE-ACS (r = 0.338, p = 0.008) and STE-ACS (r = 0.441, p < 0.0005) patients. There was a significant difference in NT-proBNP (p < 0.0005) and cTI (p < 0.0005) serum level between ACS patients who died within 30 days or who survived after one month. The increased NT-proBNP level is the strongest predictor of mortality in ACS patients, also NT-proBNP cut-point level of 1,490 pg/mL is a significant independent predictor of mortality.

Conclusions: We demonstrated the differences and the correlation in the secretion of NT-proBNP and cTI in patients with STE-ACS vs. NSTE-ACS. Our results provide evidence that NT-proBNP is a significant diagnostic marker and an important independent predictor of short-term mortality in patients with ACS.

Key words: acute coronary syndromes, NT-proBNP, troponin I, prognosis

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INTRODUCTION

B-type natriuretic peptide (BNP), cardiac neurohormone, and its N-terminal fragment (NT-proBNP) are synthesised and secreted from the ventricular myocardium. It is well known that stimulus for their release is the increase in left ventricular wall stress [1, 2]. These markers are known to be elevated in patients with acute coronary syndromes (ACS). In this regard, ventricular dysfunction and/or myocardial ischaemia per se can cause an increase in cardiac NT-proBNP expression followed by augmented secretion [3–6]. Also, these markers are closely linked to the prognosis as a powerful predictor of both short and long-term mortality [7–10]. It has been shown that BNP and NT-proBNP levels predict heart failure and death after myocardial infarction (MI) [11, 12].

Cardiac troponin (cT) is a regulatory protein which consists of three subunits I, T and C. Cardiac troponin I (cTI) is the biomarker of choice for the detection of myocardial necrosis as it is more specific and sensitive than classic cardiac enzymes (creatine kinase, CK, CK-MB) [13]. Also, the determination of cTI is useful for estimating the extent of necrosis. For the patients with ACS, cTI is a prognostic indicator and allows the stratification of risk of cardiac events and mortality [14].

The pathophysiology of ACS involves the disruption of vulnerable plaque and thrombus formation, which produces severe myocardial ischaemia and downstream embolisation in the coronary vascular bed, leading to subendocardial or transmural necrosis [15]. The clinical spectrum of ACS consists of ST elevation MI (STE-ACS) and non-ST elevation MI (NSTE-ACS) or unstable angina, which are classified from the acute phase electrocardiography (ECG) changes and the development of myocardial necrosis. STE-ACS is caused by acute total coronary occlusion, whereas NSTE-ACS is associated with vulnerable plaque and subocclusive thrombosis [16].

Since the extent of myocardial necrosis is an important determinant of the risk of death, it is important to identify serum markers to predict prognosis. It is well known that cTI, in the triage of patients with unstable coronary disease, may identify those at greater risk for adverse cardiac events [17]. As previously mentioned, recent clinical studies have shown that the degree of BNP or NT-proBNP elevation can be used to predict future cardiac events and survival [11, 12]. But significantly augmented elevation of NT-proBNP in NSTE-ACS patients compared to a not significant difference in elevation of cTI in STE-ACS patients has indicated the possibility that factors other than infarct size are more influential in NTSE-ACS patients [18]. Differences in the clinical utility and pathophysiological implication of NT-proBNP and cTI in patients with STE-ACS vs. NSTE-ACS have been investigated in a few studies [18–21].

In this study, we demonstrated the differences and the correlation in the secretion of NT-proBNP and cTI in patients with STE-ACS vs. NSTE-ACS. Taken together, these results provide evidence that NT-proBNP is a strong and independent diagnostic and prognostic marker in patients with ACS.

METHODS
Patients and design
Two hundred and five consecutive patients with acute chest pain or dyspnoea admitted to the Coronary Care Unit of the Clinical Centre Kragujevac, Serbia, were prospectively included in this observational study from January 2011 until November 2012. The inclusion criteria were a diagnosis of ACS, defined as unstable angina according to Braunwald's classification or acute MI according to the redefined ESC/ACC Committee criteria [22]. All patients with ACS underwent coronary angiography. STE-ACS patients underwent primary percutaneous coronary intervention with stent implantation. NSTE-ACS patients had a coronary angiographic finding with one or two atherosclerotic affected blood vessels and culprit lesion always stent treated. The exclusion criteria were: patients who had had cardiopulmonary resuscitation before admission, and the presence of overt pump failure (≥ Killip class II were excluded to focus on the effect of myocardial ischaemia on the release of cardiac markers). These exclusions allowed us to focus on the effect of myocardial ischaemia per

se on the release kinetics of NT-proBNP or a serum creatinine level > 2.0 mg/dL. All patients underwent standard 12-lead ECG immediately after admission, and blood samples were taken for biochemical measurements.

The patients were classified into STE-ACS and NSTE-ACS groups based on the ECG findings on admission. As demographic data, the history of hypertension, hyperlipidaemia, diabetes mellitus, gender, age, smoking, previous MI and positive family history were investigated on admission.

Diagnostic criteria for STE-ACS and NSTE-ACS. Patients with ST segment elevation at the J point in two or more consecutive leads (with the cut-off point being > 0.2 mV in leads V_1, V_2, or V_3, and > 0.1 mV in the other leads) were defined as having STE-ACS. Patients with ST segment depression, T wave inversion, or no ECG abnormalities were defined as having NSTE-ACS.

The study protocol was approved by the regional Ethics Committee and all patients gave their written informed consent.

Measurement of serum NT-proBNP level
Blood samples were collected from 134 patients (36 women and 98 men, median age 66 years) with ACS on admission in tubes containing Na2-EDTA (1.5 mg/mL). Samples were placed immediately in ice-cold water and the tubes were then centrifuged at 4,000 rpm for 15 min at 4°C.

The VIDAS NT-proBNP is an automated quantitative test for use on the VIDAS instruments for the determination of NT-proBNP in human serum or plasma (lithium heparin) using the enzyme-linked fluorescent assay (ELFA) technique. The assay principle combines a one step immunoassay sandwich method with a final fluorescent detection (ELFA). The sample is transferred into the well containing anti NT-proBNP antibody (conjugate) labelled with alkaline phosphatase. The intensity of the fluorescence is proportional to the concentration of antigens present in the sample. The functional sensitivity, defined as the lowest concentration of NT-proBNP that can be measured with an inter-assay coefficient of variation of 20%, is < 50 pg/mL. The VIDAS NT-proBNP measurement range is 20–25,000 pg/mL.

Measurement of serum cardiac troponin I level
Blood samples were collected from 134 patients (36 women and 98 men, median age 66 years) with ACS on admission in tubes containing Na2-EDTA (1.5 mg/mL). Samples were placed immediately in ice-cold water and the tubes were then centrifuged at 4,000 rpm for 15 min at 4°C.

The VIDAS cTI ultra assay is an aid in the diagnosis of MI. The assay principle combines a one step immunoassay sandwich method with a final fluorescent detection (ELFA). The measurement values of the VIDAS cTI ultra kit range from 0.01 to 30 μg/L. The analytical detection limit, defined as the smallest concentration of cTI which is significantly different from the zero concentration with a probability of 95%, is < 0.01 μg/L.

Table 1. Prevalence of risk factors in patients with acute coronary syndromes

Risk factor	NSTE-ACS (n = 60)	STE-ACS (n = 74)
Smoking	67%	58%
Hypertension	76%	73%
Lipids in blood	78%	80%
Previous myocardial infarction	17%	16%
Diabetes mellitus	24%	22%
Positive family history	58%	57%
Gender (male)	62%	73%

NSTE-ACS — non-ST elevation acute coronary syndromes; ST-ACS — ST elevation acute coronary syndromes

Table 2. Serum levels of cTI and NT-proBNP in NSTE-ACS and STE-ACS patients

ACS	NSTE-ACS (n = 60)	STE-ACS (n = 74)	P
Cardiac troponin I [μg/L]	3.48	3.50	0.552
25th–75th percentile [μg/L]	2.23–4.49	1.55–4.33	
NT-proBNP [pg/mL]	1,358	566	< 0.0005
25th–75th percentile [μg/L]	682–2,448	247–1,431	

ACS — acute coronary syndromes; NSTE-ACS — non-ST elevation acute coronary syndromes; ST-ACS — ST elevation acute coronary syndromes; NT-proBNP — N terminal-pro-B-type natriuretic peptide

Statistical analysis

The NSTE-ACS and STE-ACS groups were compared by the Mann-Whitney U test. The cTI and NT-proBNP were expressed as median (25th to 75th percentile) and other continuous variables were expressed as mean ± standard deviation. The differences of percentages were compared by the χ^2 test. The cTI and NT-proBNP levels on admission were compared between NSTE-ACS and STE-ACS patients. Spearman's correlation coefficients were calculated to assess the relationships between NT-proBNP and cTI. Multivariate binary logistic regression was applied to identify independent predictors of mortality. Receiver operating characteristic (ROC) curves analysis was used to investigate the prognostic value of NT-proBNP. A p value less than 0.05 was considered statistically significant.

RESULTS

There were no differences in the prevalence of risk factors in patients with NSTE-ACS and STE-ACS. In this series, 134 patients with ACS were included: 60 (44.8%) had NSTE-ACS and 74 (55.2%) had STE-ACS. Patients with pump failure (Killip class ≥ II) were excluded in order to focus on the myocardial ischaemia per se. The mean age was 66.32 ± 10.26 years, and 98 (73.1%) patients were men. There was no statistical significance in the average age between genders (women vs. men 67.6 ± 10.35 vs. 65.8 ± 10.23 years, p > 0.05). The prevalence of risk factors for the patients in the NSTE-ACS and STE-ACS groups is shown in Table 1. The majority of the patients were smokers with hypertension, hyperlipidaemia and a positive family history for ischaemic coronary desease. There were no differences in smoking, hypertension, hyperlipidaemia, previous MI, diabetes mellitus or positive family history between these two groups.

Serum level of NT-proBNP on admission was significantly higher in the NSTE-ACS patients compared to STE-ACS patients, while there was no difference in serum level of cardiac troponin I. Unlike the serum level of cTI, serum level of NT-proBNP on admission was significantly higher (p < 0.0005) in NSTE-ACS patients compared to STE-ACS patients. In patients who had NSTE-ACS, the serum level of NT-proBNP was 1,358 pg/mL (interquartile range 682–2,448 pg/mL), and in patients who had STE-ACS the serum level of NT-proBNP level was 566 pg/mL (interquartile range 247–1,431 pg/mL). Additionally, there was no significant difference (p = 0.552) in the serum level of cTI between NSTE-ACS patients and STE-ACS patients (Table 2).

There was a significant positive correlation between serum levels of NT-proBNP and cardiac troponin I in the NSTE-ACS and in the STE-ACS patients. The results of the relation between serum levels of NT-proBNP and cTI showed an important positive correlation in the NSTE-ACS and STE-ACS patients (Fig. 1).

The significant positive correlation was observed between serum level of NT-proBNP and cTI in the NSTE-ACS (r = 0.338, p = 0.008) group and in the STE-ACS (r = 0.441, p < 0.0005) group.

There were significant differences in serum levels of NT-proBNP and cardiac troponin I between ACS patients who survived and who died after one month. NT-proBNP serum level was higher among patients who died than among those who were alive after 30 days. NT-proBNP serum level in patients who died was 2,130 (590–2,685) pg/mL vs. 680 (318–1,259) pg/mL in patients who survived one month after ACS. The difference of NT-proBNP serum level was statistically significant (p < 0.0005) between patients who had died after one month and those who survived (Fig. 2A).

Additionally, NT-proBNP serum level in the subgroup of patients who had non-ST elevation and who died was 2,725 (1,628–3,131) pg/mL vs. 882 (600–1,582) pg/mL in patients who survived one month after ACS. The difference of NT-proBNP serum level was statistically significant between patients who died after one month and those who survived (Fig. 2B).

NT-proBNP serum level in the subgroup of patients with ST elevation and who died was 1,673 (574–2,368) pg/mL vs. 375 (215–962) pg/mL in patients who survived one month after ACS. The difference of NT-proBNP serum level was sta-
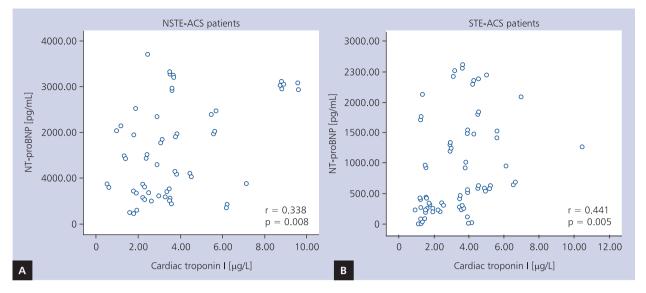
Figure 1. Significant positive correlation between serum level of N terminal-pro-B-type natriuretic peptide (NT-proBNP) and cardiac troponin I in the non-ST elevation acute coronary syndromes (NSTE-ACS) group (**A**) and in the ST elevation acute coronary syndrome (STE-ACS) group (**B**)

tistically significant (p < 0.0005) between patients who died after one month and those who survived (Fig. 2C).

cTI serum level in patients who died was 3.8 (1.89–5.05) μg/L vs. 3.12 (1.75–3.87) μg/L in patients who survived one month after ACS. The difference of cTI serum level was statistically significant (p = 0.027) between patients who died after one month and those who survived (Fig. 2D). There were no significant differences (p > 0.05) between the subgroup of patients who had MI with ST elevation and the patients who had non-ST elevation MI.

Increased serum level of NT-proBNP was the strongest predictor of mortality in ACS patients. Multivariate binary logistic regression was used to identify independent predictors of mortality (variables: gender, age, glycaemia, C-reactive protein [CRP], cTI, total cholesterol, HDL, LDL, triglyceride, NT-proBNP, type of ACS). Multivariate binary logistic regression showed (Table 3) that the strongest predictors of mortality were increasing serum levels of NT-proBNP (p < 0.005) and CRP (p < 0.05), unlike cTI (p = 0.586) and glycaemia (p = 0.194).

ROC curve analysis, sensitivity and specificity were determined as continuous data where the area under the curve (AUC) is the biggest then its sensitivity and the specificity are the best. This analysis showed that the accuracy of relative changes in NT-proBNP level are highly sensitive and specific (Fig. 3). The sensitivity of NT-proBNP was 0.674, and the specificity was 0.852 (AUC = 0.768 ± 0.048, p < 0.0005).

NT-proBNP cut-point level was a significant independent predictor of mortality. It was shown that NT-proBNP at a cut-point of 1,490 pg/mL was highly sensitive (67.4%) and specific (85.2%) and was a significant independent predictor

of mortality (Fig. 4). When we analysed short-term mortality (one month) in patients with NT-proBNP < 1,490 vs. NT-proBNP ≥ 1,490, it was statisticaly significant (p < 0.0005). Additionaly, we found that positive predictive value was 70.5% (31/44), and negative predictive value 83.3% (75/90) (Table 4).

DISCUSSION

In several studies on patients with STE-ACS and NSTE-ACS, the elevation of BNP and NT-proBNP has been observed [19–21]. The STE-ACS group with transmural infarction had a larger infarct size than the NSTE-ACS group in these studies. cTI I is a marker of myofibril damage and is elevated in proportion to infarct size per se [22, 23]. In contrast, NT-proBNP has been found to be higher in NSTE-ACS patients than in STE-ACS patients despite lower values of cTI [19, 23]. Patients with pump failure greater than Killip class II were excluded, which suggests that factors other than infarct size or pump failure had a fundamental influence on the elevation of NT-proBNP. In the present study, we demonstrated that there was no significant difference in cTI levels between STE-ACS and NSTE-ACS patients, as was shown in the previous study. Further, the values of NT-proBNP were significantly higher in the NSTE-ACS than in the STE-ACS group (Table 2).

Galvani et al. [20] reported similar findings in their multicentre study of patients with ACS. Myofibril marker cTI was not significantly higher in STE-ACS patients compared to NSTE-ACS patients. Conversely, NT-proBNP was significantly higher in NSTE-ACS patients than STE-ACS especially within 3 h of onset, suggesting a larger ischaemic insult despite the smaller extent of myocardial necrosis compared to STE-ACS patients. Weber et al. [21] reported similar findings: the highest
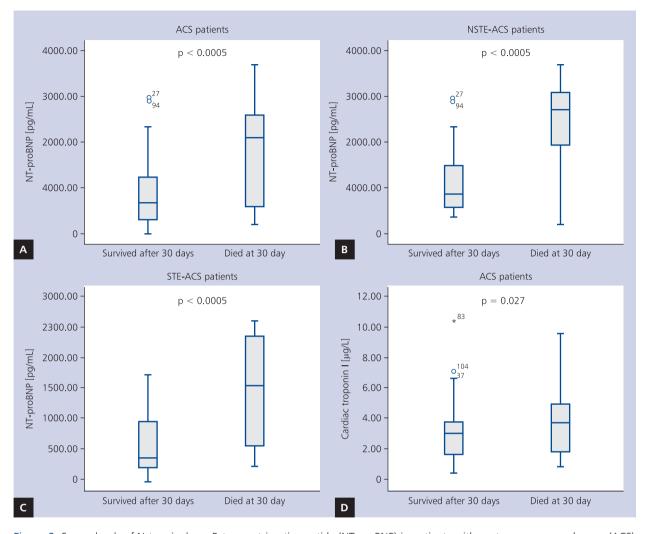
Figure 2. Serum levels of N-terminal pro-B-type natriuretic peptide (NT-proBNP) in patients with acute coronary syndromes (ACS) who survived after one month and who died (**A**). Serum level of NT-proBNP in NSTE-ACS (**B**) and in STE-ACS (**C**) patients who survived after one month and who died. Serum levels of cardiac troponin I among patients with ACS who survived after one month and who died (**D**)

Table 3. Binary logistic regression for possible independent predictors of mortality

	Multivariate analysis OR (95% CI)	P
Cardiac troponin I	0.927	0.586
NT-proBNP	1.001	< 0.005
CRP	1.012	< 0.05
Glycaemia	0.911	0.194

Multivariate binary logistic regression shows that increasing NT-proBNP and CRP proved to be independent predictors of mortality; CRP — C-reactive protein; NT-proBNP — N terminal-pro-B-type natriuretic peptide; OR — odds ratio; CI — confidence interval

values for NT-proBNP on admission were found in patients with NSTE-ACS compared to patients with STE-ACS.

On the other hand, Ogawa et al. [18] showed that cTI levels on admission were significantly higher in STE-ACS patients compared to NSTE-ACS. When NT- proBNP levels were assessed, the median plasma level was 1,976 (489––3,097) pg/mL in the NSTE-ACS group, while the respective values were 98 pg/mL in the STE-ACS group. When the values were compared, NT-proBNP levels were significantly higher in the NSTE-ACS patients (NSTE-ACS vs STE-ACS, p = 0.0132). Interestingly, those values are almost consistent with our data: 1,358 pg/mL in NSTEMI patients. Such early increases would reflect the amount of ischaemic insult to the myocardium rather than the actual extent of myocardial damage or degree of heart failure. Myocardial ischaemia per se could be another mechanism leading to elevation of NT-proBNP, besides the presence of ventricular dysfunction. It could be also possible that early NT-proBNP elevation in NSTE-ACS patients may reflect the consequences of repeated episodes of myocardial
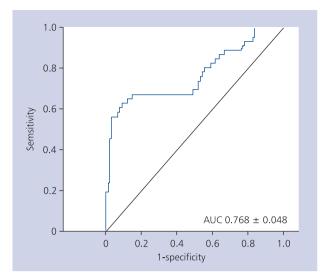
Figure 3. Receiver-operating characteristic (ROC) curves analysis showing the accuracy of relative changes in N terminal--pro-B-type natriuretic peptide (NT-proBNP) level, with a highly significant area under the curve (AUC). NT-proBNP was highly sensitive and specific as the predictor of mortality
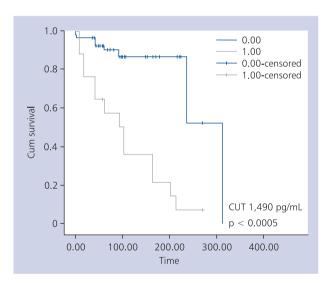

Figure 4. Survival function in acute coronary syndromes patients for cut-point level of N terminal-pro-B-type natriuretic peptide (NT-proBNP)

Table 4. Predictive value of N terminal-pro-B-type natriuretic peptide (NT-proBNP) in acute coronary syndromes in patients

	Survived after 30 days	Died within 30 days
NT-proBNP < 1,490	75	15
NT-proBNP ≥ 1,490	13	31

ischaemia, both symptomatic and asymptomatic, occurring in the past several hours or days [24, 25].

As previously reported, there is an obvious correlation between the serum cTI level and the wall motion score during the acute phase of acute MI [26] or other major haemodynamic derangements [27]. Also, a correlation between the cTI level and left ventricular ejection fraction was found in a previous study [18]. These findings suggest that cTI is in correlation with ejection fraction, as well as in correlation with left ventricular systolic dysfunction. Our results did not show a significant difference between STE-ACS and NSTE-ACS in cTI serum level. On the other hand, NT-proBNP was statistically significantly higher in NSTE-ACS patients, and it seems that dysfunction is not the only mechanism that leads to NT-proBNP releasing. This is probably the evidence for ischaemia as the cause for NT-proBNP releasing.

Plasma levels of NT-proBNP may be a new way to detect silent myocardial ischaemia [28], as our results proposed. In this regard, the study by Goetze et al. [3, 6] strongly suggests that acute myocardial hypoxia/ischaemia per se stimulates BNP expression and the release of a newly synthesised proBNP peptide. In that study, NT-proBNP levels were markedly elevated in ACS patients, especially in the NSTE-ACS group, and could be an early sensitive marker of myocardial ischaemia that rises much higher than expected than the cTI levels in NSTE-ACS patients, and even in the absence of heart failure [23].

As mentioned in the introduction, NT-proBNP appears as a unifying feature that is independent of other biochemical markers and is a powerful and independent determinant of the short-term cardiac risk in patients with ACS [29, 30]. Our study showed that the difference of median NT-proBNP concentrations between patients who died after one month and who survived was statistically significant (Fig. 2A).

Biochemical markers are useful for the prediction of future cardiovascular events in patients with ACS. The independent as well as the combined prognostic value of elevated Tn and NT-proBNP on the Thrombolysis In Myocardial Infarction (TIMI) risk score and on the short-term prognosis were evaluated in a cohort of ACS patients. The TIMI risk score as expected had a positive and strong correlation with elevated cTI, but had no correlation with the elevation of NT-proBNP. In this respect, some results have shown that the contribution of cTI in predicting 30-day death/MI is more important, whereas the role of NT-proBNP was attenuated [31]. However, the highest risk of death from any cardiovascular cause within 30 days of follow-up was significantly higher when all biomarkers were elevated [32, 33].

Our study showed that the median NT-proBNP level for patients who died is statistically significantly higher than in patients who survived one month after ACS (Fig. 2A). We also showed that the median cTI level for patients who died is statistically significantly higher than in patients who survived one month after ACS (Fig. 2B). Furthermore, we showed that NT-proBNP is a better predictor of mortality than cTI.

Looking at the bigger picture, among the biomarkers measured at randomisation, using multivariate binary logistic regression, we demonstrated that increasing NT-proBNP and CRP proved to be the strongest independent predictors of mortality, unlike cTI and glycaemia (Table 3). However, adjusting by biomarkers, NT-proBNP concentration was the strongest independent predictor of in-hospital ($p < 0.005$) mortality.

Therefore, NT-proBNP demonstrated the strongest association to the composite end point of death/MI. Our results showed that NT-proBNP at a cut-point of 1,490 pg/mL was highly sensitive (67.4%) and specific (85.2%), and is a significant independent predictor of mortality (Fig. 4). This is, to the best of our knowledge, the first study to evaluate the cut-point value of NT-proBNP in patients with ACS.

CONCLUSIONS

NT-proBNP is an early sensitive marker of myocardial ischaemia that rises much higher in the earlier phase compared to a novel marker of myocardial damage such as troponin, especially in NSTE-ACS patients. NT-proBNP assessment should be considered in clinical practice for risk stratification of patients with normal cTI. Furthermore, our study clearly shows that an elevated level of NT-proBNP has a close correlation to disease severity in patients with ACS, which underlines its utility as a biochemical marker for the screening of cardiovascular disorders.

To the best of our knowledge, this is the first study to demonstrate that NT-proBNP is highly sensitive and specific, and is a significant independent predictor of short-term (one month) mortality.

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Znaczenie stężenia NT-proBNP w określaniu rokowania i ocenie diagnostycznej u chorych z ostrymi zespołami wieńcowymi

Vladimir Zdravkovic[1], Violeta Mladenovic[2], Maja Colic[3], Dragic Bankovic[3], Zorica Lazic[2], Marina Petrovic[2], Ivan Simic[1], Slavko Knezevic[2], Sveto Pantovic[4], Aleksandar Djukic[2], Nemanja Zdravkovic[3]

[1]Department of Cardiology, Clinical Center Kragujevac, Faculty of Medical Sciences, University of Kragujevac, Kragujevac, Serbia
[2]Clinic for Internal Medicine, Clinical Center Kragujevac, Kragujevac, Serbia
[3]Centre for Molecular Medicine and Stem Cell Research, Faculty of Medical Sciences, University of Kragujevac, Kragujevac, Serbia
[4]Department of Gynecology and Obstetrics, Clinical Center of Serbia, Belgrade, Serbia

Streszczenie

Wstęp i cel: N-końcowy fragment propeptydu natriuretycznego typu B (NT-proBNP) jest syntetyzowany i wydzielany przez miokardium komór serca. Wiadomo, że stężenie tego wskaźnika jest podwyższone u chorych z ostrymi zespołami wieńcowymi (ACS). Autorzy ocenili znaczenie stężenia NT-proBNP jako istotnego wskaźnika diagnostycznego i ważnego niezależnego czynnika prognostycznego śmiertelności krótkoterminowej (w ciągu 1 miesiąca) u chorych z ACS.

Metody: Oznaczono stężenia NT-proBNP i sercowej troponiny I (cTI) u 134 kolejnych chorych (mediana wieku 66 lat, 73% mężczyzn) hospitalizowanych z powodu ACS na oddziale kardiologicznym szpitala uniwersyteckiego. Pacjentów przydzielano do grupy ACS z uniesieniem odcinka ST (STE-ACS, n = 74) lub do grupy ACS bez uniesienia odcinka ST (NSTE-ACS, n = 60) na podstawie EKG wykonanego przy przyjęciu. Chorych w klasie Killipa ≥ II wykluczono z badania.

Wyniki: Stężenie NT-proBNP w surowicy przy przyjęciu do szpitala było istotnie wyższe (p < 0,0005) u pacjentów z NSTE-ACS niż u osób z STE-ACS, natomiast surowicze stężenia cTI były podobne w obu grupach. Stwierdzono istotną dodatnią korelację między stężeniami NT-proBNP i cTI u chorych z NSTE-ACS (r = 0,338; p = 0,008) i u osób z STE-ACS (r = 0,441; p < 0,0005). Stężenia NT-proBNP (p < 0,0005) i cTI (p < 0,0005) w surowicy różniły się istotnie między chorymi z ACS, którzy zmarli w ciągu 30 dni, a pacjentami, którzy żyli dłużej niż miesiąc. Zwiększone stężenie NT-proBNP jest najsilniejszym czynnikiem prognostycznym zgonu u chorych z ACS. Ponadto stężenie NT-proBNP powyżej progowej wartości 1490 pg/mL jest istotnym niezależnym czynnikiem prognostycznym zgonu.

Wnioski: Autorzy wykazali różnice w wydzielaniu NT-proBNP i cTI między chorymi ze STE-ACS i chorymi z NSTE-ACS oraz korelacje między tymi wskaźnikami. Uzyskane przez nich wyniki dowodzą, że NT-proBNP jest istotnym niezależnym wskaźnikiem diagnostycznym i ważnym niezależnym czynnikiem prognostycznym śmiertelności krótkoterminowej u chorych z ACS.

Słowa kluczowe: ostre zespoły wieńcowe, NT-proBNP, troponina I, rokowanie

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Adres do korespondencji:
Nemanja Zdravkovic, MD, PhD, Assistant Professor, Centre for Molecular Medicine and Stem Cell Research, Faculty of Medical Sciences, University of Kragujevac, Svetozara Markovica Street 69, 34000 Kragujevac, Serbia, tel: +38134306800113, fax: +38134306800112, e-mail: zdravkovic_nemanja@yahoo.com
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