Long pentraxin-3 measured at late phase associated with GRACE risk scores in patients with non-ST elevation acute coronary syndrome and coronary stenting

Koroner stent uygulanan ST yükselmesiz akut koroner sendromda geç dönem ölçülen "long pentraxin-3"ün GRACE risk skoru ile ilişkisi

Serkan Saygı, M.D., Bahadır Kırılmaz, M.D., İstemihan Tengiz, M.D.,[#] Uğur Önsel Türk, M.D.,[†] Hicran Yıldız, M.D., Nurullah Tüzün, M.D.,[†] Emin Alioğlu, M.D.,[#] Gülden Sönmez Tamer, M.D.,[¶] Ertuğrul Ercan, M.D.[#]

Department of Cardiology, Çanakkale Onsekiz Mart University Faculty of Medicine, Çanakkale;
[#]Department of Cardiology, Izmir University Faculty of Medicine, İzmir;
[†]Department of Cardiology, Central Hospital, İzmir;
[¶]Department of Microbiology and Clinical Microbiology, Kocaeli University Faculty of Medicine, Kocaeli

ABSTRACT

Objectives: We analyzed pentraxin 3 (PTX3) levels and the relation of PTX3 levels with GRACE risk scores in 39 patients with non-ST elevation acute coronary syndrome (ACS) and stabile angina after stenting.

Study design: Seventeen patients with ACS and 22 patients with stabile angina who underwent coronary stenting were included in the study. PTX3 levels were measured serially at admission, at the 8th hour and at the 24th hour after stenting.

Results: While diabetes and hypertension were more frequent in the stabile angina group, leukocyte counts were significantly higher in the ACS group. PTX3 levels measured at the 8th hour were significantly higher in the ACS group compared to the stabile angina group (p=0.003). Strong correlations were observed between 24th hour PTX3 levels and GRACE scores calculated for risk of death and death/MI at admission (in-hospital/to 6 months), and for risk of death/MI at discharge to 6 months (R=0.571, p=0.01, R=0.564, p=0.01; R=0.558, p=0.02, R=0.512, p=0.03; R=0.653, p=0.004, respectively).

Conclusion: The serum PTX3 levels may provide important information for the early risk stratification of patients with ACS who underwent coronary stenting.

ÖZET

Amaç: Kararlı anjina ve ST yükselmesiz akut koroner sendrom (AKS) tanısıyla perkütan koroner girişim (PKG) yapılmış 39 hastada ardışık ölçülen "pentraxin-3" (PTX3) düzeyleri ve bu düzeylerin GRACE risk skoru ile ilişkisi incelendi.

Çalışma planı: Çalışmaya perkütan koroner stent takılan ST yükselmesiz AKS tanılı 17, kararlı anjina tanılı 22 olgu alındı. Tüm hastalarda girişim öncesi, girişim sonrası 8. ve 24. saatte PTX3 ölçümü için kan örnekleri alındı.

Bulgular: Kararlı anjinalı grupta diabetes mellitus ve hipertansiyonlu olgu sayısı anlamlı olarak daha yüksek iken, AKS grubunda lökosit sayısı anlamlı olarak daha yüksek saptandı. Sekizinci saat ölçülen PTX3 düzeyleri anlamlı olarak AKS grubunda daha yüksekti (p=0.003). AKS'li olgularda 24. saat PTX3 düzeyleri ile GRACE başvuru hastane içi ve 6 ay ölüm-kardiyak ölüm-ME ve GRACE taburculuk kardiyak ölüm-ME risk oranları arasında güçlü korelasyon saptandı (sırasıyla, p=0.02, R: 0.558; p=0.03, R=0,512; p=0.01, R=0,571; p=0.01, R=0.564; p=0.004, R=0.653).

Sonuç: Serum PTX3 düzeyleri koroner stent takılan AKS'li olgularda erken risk sınıflaması açısından önemli bilgiler verebilir.

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Correspondence: Dr. Serkan Saygı. Çanakkale Onsekiz Mart Üniversitesi Araştırma ve Uygulama Hastanesi, Kardiyoloji Kliniği,17110 Kepez, Çanakkale, Turkey.
Tel: +90 286 - 263 59 50 e-mail: serkankard@gmail.com

Studies have clearly demonstrated the association of inflammation with the initiation and progression of atherosclerosis. Risk stratification in the early phase of acute coronary syndromes is essential for determining optimal therapy and predicting poor outcomes.

The short pentraxin C-reactive protein has been used as a diagnostic and prognostic tool after coronary stenting.[1,2] The PTX protein family is divided into two subfamilies, long and short PTX.[3] CRP is a member of the short PTX family and is one of the most studied inflammatory markers. PTX3 is the first identified long PTX. Distinct from CRP, which is synthesized by hepatocytes, PTX3 is synthesized directly by cells located in the atherosclerotic lesion, including vascular endothelial cells, smooth muscle cells, fibroblasts, and macrophages.[4] Expression of PTX3 is controlled by pro-inflammatory cytokines, such as interleukin-1 and tumor necrosis factor-α.[5] Because of the relation between PTX3 and CRP, the role of PTX3 in cardiovascular diseases has been investigated. Studies have shown that the level of PTX3 in the blood increases during acute coronary syndrome.[6] Additionally, PTX3 measured during the early stage was found to be a prognostic tool in patients with acute myocardial infarction.[7] The Global Registry of Acute Coronary Events (GRACE) risk scoring system was designed to predict the risk of death, either in-hospital or after discharge, for patients with ACS. The GRACE risk scoring system was shown to precisely predict cardiovascular outcome for patients with ACS.[8]

Recently, it has been shown that, of the parameters determining the GRACE risk scores, Killip class is independently associated with PTX3 concentrations in subjects with unstable angina, non-ST elevation MI, and ST elevation MI.[9] Coronary stenting is the most efficient therapy for ACS. However, there is no information on the prognostic value of PTX3 levels in acute coronary syndrome patients treated with coronary stenting.

In this study, we aimed to investigate the prognostic value of serum PTX3 levels in patients with unstable angina pectoris/non-ST elevation MI who underwent coronary stenting, using the GRACE risk scoring system. We also planned to measure serum PTX3 levels serially at baseline (before coronary intervention), at the 8th hour, and at the 24th hour to determine the time interval in which PTX3 concentration is closely related with poor cardiovascular outcomes.

Abbreviations:

ACS	*Acute coronary syndrome*
CRP	*C-reactive protein*
GRACE	*Global Registry of Acute Coronary Events*
MI	*Myocardial infarction*
PTX	*Pentraxin*

PATIENTS AND METHODS

Subjects

Informed consent was obtained from all patients before enrollment, and the protocol was approved by the local ethics committee of Çanakkale Onsekiz Mart University, School of Medicine. A total of 39 patients (17 patients with non-ST elevation ACS, 22 patients with stable angina) treated with percutaneous coronary stenting between February and August 2010 were included in the study. Unstable angina/non-ST elevation MI was defined as the presence of typical chest pain lasting more than 10 minutes and accompanied with ST segment depression or transient ST segment elevation, T wave inversion in at least two contagious leads, or cardiac troponin I elevation. All 17 patients with ACS were admitted to the hospital within 24 hours after the onset of chest pain.

The stable angina group consisted of 22 patients with angiographically documented significant luminal narrowing in at least one coronary artery that required percutaneous coronary intervention. Patients with persistent ST segment elevation, a new left bundle branch block, renal failure serum (creatinine >2.0 mg/dl), malignancy, or acute/chronic inflammatory disease were excluded from the study.

Blood sample collection and laboratory analyses

Venous blood samples were obtained from all patients before intervention (0th hour/baseline) and both 8 hours and 24 hours after coronary stenting to measure serum PTX3 levels. Blood samples were collected in ethylenediaminetetraacetic (EDTA) tubes and separated by centrifugation. The serum was stored at -70°C. Serum PTX3 was measured

using a commercially available enzyme-linked immunosorbent assay kit (Perseus Proteomics Inc., Tokyo, Japan).

Coronary angiographic analysis and interventional procedure

All patients in the study underwent selective coronary artery angiography after appropriate patient preparation. Coronary angiographic examination was performed using the Judkins method, via the femoral or radial artery approach, and according to the American College Cardiology/American Heart Association/Society for Cardiovascular Angiography and Interventions 2005 Guideline.[10] Quantitative analyses of angiographic views were completed using an automated edge detection system (GE Medical Systems).

The following variables were measured: minimum lumen diameter (mm), reference lumen diameter (mm) and degree of stenosis. All angiograms were evaluated by two experienced cardiologists blinded to the study. A scoring method developed by Gensini was used to assess the severity of coronary artery lesions.[11]

The GRACE risk score of each patient with acute coronary syndrome was calculated using the following parameters: age, heart rate, systolic blood pressure, serum creatinine level, Killip class, presence of cardiac arrest at admission, ST segment deviation, and elevated cardiac enzymes.[8] The risk scores for all causes of death and cardiac death/MI, in-hospital and to 6 months, at admission and at pre-discharge were separately calculated for patients with ACS. Coronary stent implantation was performed using the standard Judkins technique via the femoral or radial approach. All patients were pre-medicated with 10000 IU of heparin administered intravenously before the procedure. A bare metal stent was placed after optimal or suboptimal lesion dilation. After stent implantation, ACS patients received a heparin infusion (maximum 1000 IU/h) to maintain an appropriate ACT.

Statistical analysis

Statistical data analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 15.0 (SPSS Inc., Chicago, IL, USA). Numerical variables were expressed as mean and standard deviation, and frequencies were expressed as number and percentage. p-values ≤ 0.05 were accepted as statistically significant. Linear regression analysis was performed to verify correlations between GRACE risk scores in the ACS group and serum levels of PTX3 measured at baseline, at the 8th hour, and at the 24th hour.

RESULTS

Baseline characteristics of, and laboratory findings for, the 39 patients (22 stable angina, 17 ACS) who underwent percutaneous coronary stenting are summarized in Table 1. While the numbers of hypertensive, diabetic, and overweight patients were significantly higher in the stable angina group compared to the ACS group (p=0.01, p=0.03, and p=0.02, respectively), blood leukocyte and hemoglobin levels were higher in the ACS group (p=0.001 and p=0.01, respectively). Mean Gensini scores of the stable angina and the ACS group were similar. The PTX3 levels of the two groups were compared (Table 1, Fig. 1). The baseline mean PTX3 levels were similar for both groups. At the 24th hour, the mean PTX3 level of the ACS group was higher than that of the stable angina group, but the difference was not statistically significant. The mean PTX3 level measured at the 8th hour was significantly higher for the ACS group compared to the stable angina group (ACS group, 17.4±11.3; stable angina group, 9.9±9.4; p=0.003).

Linear regression analysis was performed between the GRACE scores and the PTX3 levels of the ACS group. No correlation was found between mean PTX3 levels measured at baseline or at the 8th hour and GRACE risk scores. Significant correlations were observed between the mean PTX3 level at the 24th hour and GRACE scores calculated for risk of death and death/MI at admission (in-hospital and to 6 months), and for risk of death/MI at discharge to 6 months (Fig. 2; risk of death in-hospital at admission, R=0.571, p=0.01; risk of death/MI in-hospital at admission, R=0.564, p=0.01; risk of death at admission to 6 months, R=0.558, p=0.02; risk of death/MI ad admission to 6 months, R=0.512, p=0.03; risk of death/MI at discharge to 6 months, R=0.653, p=0.004).

DISCUSSION

In this study, we found strong correlations between 24th hour serum PTX3 levels and GRACE risk scores of patients with non-ST elevation ACS who underwent coronary stenting. The 8th hour mean PTX3 level of the ACS group was significantly different from that of the stable angina group; however, surprisingly, no correlation was observed between 8th hour PTX3 levels and GRACE scores of ACS patients. Previous reports have demonstrated the relation of PTX3 to acute inflammatory response and myocardial infarction.[3,7,12] It has been shown that PTX3 blood levels peak within eight hours of the onset of symptoms of myocardial infarction.[6] In accordance with previous reports, the 8th hour mean PTX3 level of the ACS group was found to be significantly higher compared to the stable angina group in our study. Additionally, the 24th hour mean PTX3 level was higher in the ACS

Table 1. Group baseline characteristics and pentraxin-3 levels

	Acute coronary syndrome (n=17)			Stable angina pectoris (n=22)			
	n	%	Mean±SD	n	%	Mean±SD	*p*
Age			56±14			60±8	NS
Female (%)	6	35		8	36		NS
Diabetes mellitus (%)	1	6		10	45		**0.03**
Hypertension (%)	5	30		15	68		**0.01**
Body mass index (kg/m^2)			25±2			28±4	**0.02**
Family history of CAD (%)	7	41		8	36		NS
Hyperlipidemia (%)	7	41		15	68		NS
Smoking (%)	13	76		12	54		NS
History of MI (%)	4	23		8	36		NS
History of PCI (%)	1	6		6	27		NS
History of CABG (%)	0			0			NS
Previous medication							
Aspirin (%)	15	88		20	90		NS
Clopidogrel (%)	6	35		14	63		NS
Statin (%)	13	76		18	81		NS
Beta blocker (%)	11	64		13	60		NS
Laboratory findings							
White blood cell (mm^3/dl)			10±4			8±1	**0.001**
Hemoglobin (gr/dl)			13±2			12±2	**0.01**
Platelets (/mm^3)			225±76			214±63	NS
Fasting glucose (mg/dl)			110±25			126±44	NS
Creatinine			0.9±0.2			0.9±0.2	NS
Total cholesterol			191±53			171±44	NS
LDL-cholesterol			116±44			99±33	NS
NYHA class			2.4±0.7			1.6±0.7	NS
Gensini score			39±35			24±20	NS
Pentraxin-3 level baseline			8.8±8.1			9.3±10	NS
Pentraxin-3 level 8th hour			17.4±11.3			9.9±9.4	**0.003**
Pentraxin-3 level 24th hour			14.8±12.1			9.4±9.8	0.08 (NS)

CAD: Coronary artery disease; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass graft; NYHA: New York Heart Association; NS: Not significant; p≤0.05 considered significant.

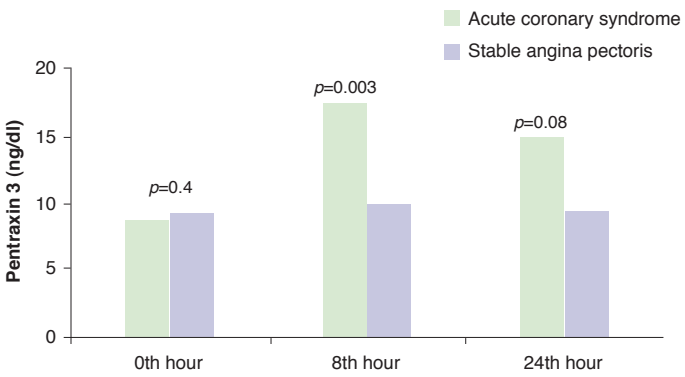

Figure 1. Comparison of baseline (0th hour), 8th hour, and 24th hour mean pentraxin 3 levels of each group. ($p \leq 0.05$ considered significant).

group; however, the difference was not statistically significant. Previous reports have analyzed PTX3 levels measured in the early phase of acute cardiac events. Latini et al.[7] demonstrated that PTX3 levels, measured at a median of three hours after symptom onset, predicted three-month mortality for ST elevation MI patients. Another study of 16 ACS patients investigated the diagnostic value of PTX3 and demonstrated the diagnostic sensitivity and specificity of PTX3 for ACS in the early stage.[13] The evidence clearly demonstrated not only the diagnostic sensitivity, but also, the prognostic value of PTX3 measured in the early phase of ACS. In addition to the baseline and 8th hour measurements, serum PTX3 levels were measured at the 24th hour of the acute cardiac event in this study.

Strong correlations were found between the 24th hour PTX3 levels and the GRACE risk scores, including risk of all causes of death at admission in-hospital/to 6 months, and risk of cardiac death/MI at admission and at discharge in-hospital/to 6 months in our study. The GRACE risk scoring system was found to precisely predict mortality for patients with ACS, including ST elevation MI, non-ST elevation MI and unstable angina pectoris.[8] The GRACE risk scores were calculated using age, heart rate, Killip class, history of MI, systolic blood pressure, ST segment depression, serum creatinine level, cardiac enzyme elevation, cardiac arrest at admission, in-hospital percutaneous intervention, and past history of MI at discharge.[8] Most previous reports have investigated PTX3 levels obtained in the early stage of acute cardiac events. Although PTX3 blood levels peak during the early stage, we hypothesized that the prognostic value of PTX3 measured after the peak time had been underestimated. The enhanced plasma levels of PTX3 at the 24th hour could reflect the inflammatory burden in atherosclerotic lesions. Kotooka et al.[14] analyzed 20 patients undergoing elective coronary stenting. The relative increase in PTX3 at the 24th hour after coronary stenting was found to be the most powerful predictor of late lumen loss. They concluded that the 24th hour PTX3 level may be a useful marker for the evaluation of inflammatory reactions.[14] In contrast, patients with non-ST elevation ACS were analyzed in our study. Even so, PTX3 levels measured at the 24th hour were strongly correlated with GRACE risk scores supporting the results of Kotooka et al.

Prognostic evaluation of ACS patients is essential to estimate high-risk groups and to determine optimal therapy. Cardiac troponins, NT-proBNP, and CRP have been shown to predict cardiovascular events in patients with ACS.[15-17] Long PTX3 is structurally related to CRP, which is one of the most investigated biomarkers for acute cardiac events. Previous reports have shown that elevated levels of CRP during ACS are associated with adverse cardiac events.[18,19] Despite the presence of accumulated data, there are still uncertainties about the diagnostic and predictive value of CRP in ACS. The level of CRP measured at the early phase of an acute cardiac event is not helpful in estimating cardiovascular prognosis. Additionally, CRP levels increase in a wide variety of conditions

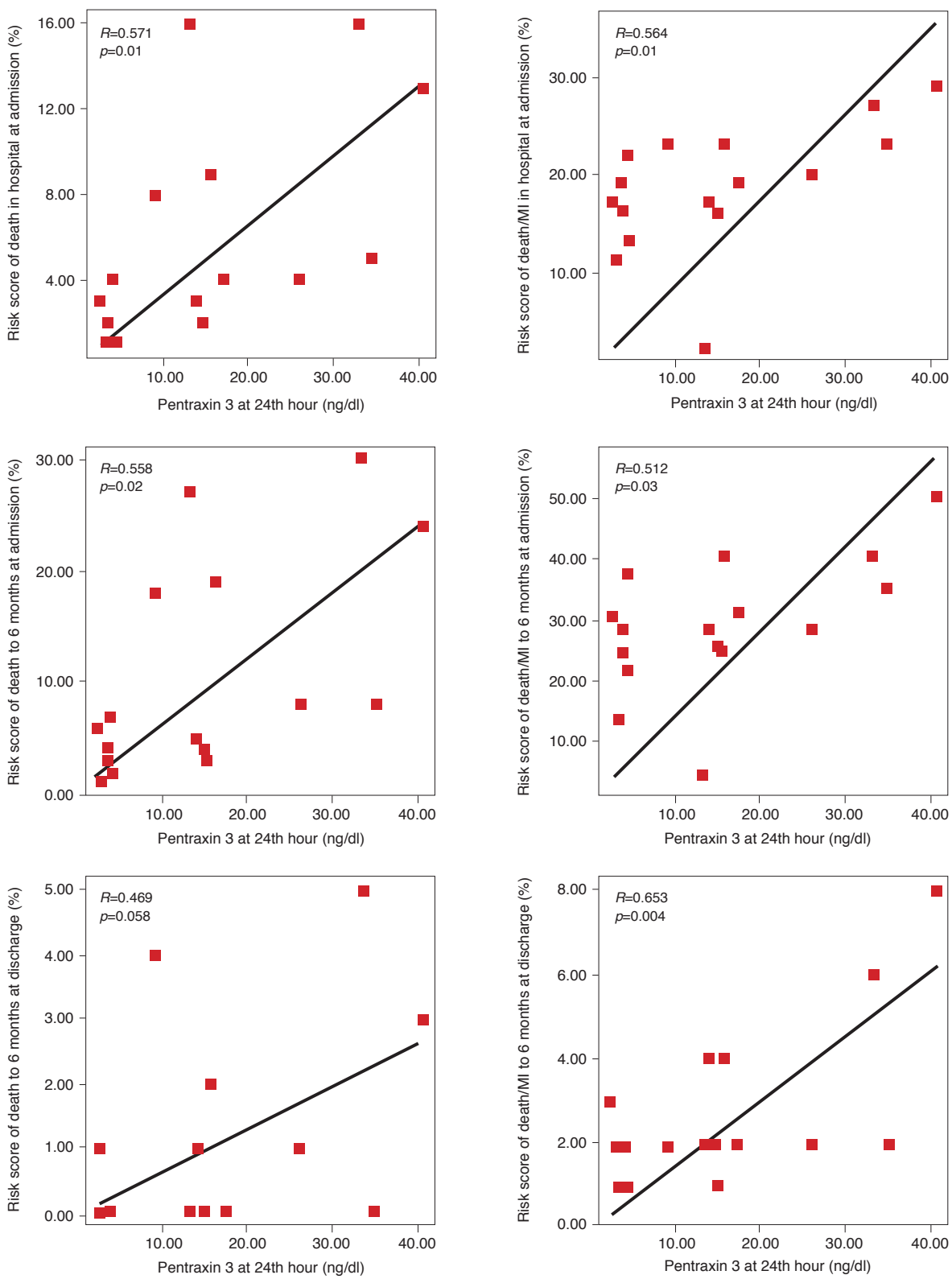

Figure 2. Linear regression analysis of 24th hour mean PTX3 level and GRACE scores, including risk of all causes of death and death/MI at admission in-hospital and to 6 months, and risk of all causes of death and death/MI at discharge to 6 months.

(acute inflammation, malignancy, chronic inflammatory disorders, vasculitis), a finding which is considered the most important disadvantage of CRP. Furthermore, the relation between elevated CRP concentrations and the presence of coronary artery disease risk factors limits the predictive value of CRP. In a recent report, PTX3, but not high sensitivity CRP, was found to be an independent predictor of cardiac events in a stepwise multivariate Cox regression analysis that included 18 well-known clinical and biochemical predictors of ACS.[20] Distinct from CRP, PTX3 is synthesized specifically by cells located in the atherosclerotic lesion,[4] so PTX3 is a specific biomarker for ACS, unlike CRP. PTX3 concentrations measured during acute cardiac events also provide clinicians with an estimate of the long-term prognosis for patients at the early phase of ACS. This evidence demonstrates the superior diagnostic and prognostic value of PTX3 compared to CRP.

Limitations

Our study has limitations. Because of the small sample size, we used the GRACE risk scoring system to estimate the cardiovascular outcomes. However, the GRACE risk scoring system was shown to precisely predict the cardiovascular outcomes of patients with ACS; clinical follow up and mortality analysis would strengthen our results. Nine of 17 patients with ACS had elevated troponin I concentrations. We did not observe cardiac enzyme elevation due to coronary stenting in the ACS group. However, the changes in cardiac enzymes before and after percutaneous coronary intervention in the stable angina group were not analyzed which can be accepted as a limitation. The cross-sectional nature of the study might also be accepted as a limitation.

In conclusion, the mean 8th hour serum PTX3 level was significantly higher in the non-ST elevation ACS patients compared with the stable angina patients. Additionally, the 24th hour mean PTX3 level was higher in the ACS group; however, the difference was not statistically significant. The strong correlation between mean 24th hour PTX3 level and GRACE risk score demonstrated in our study is remarkable. This result demonstrates that elevated PTX3 concentrations can serve as an early indicator of poor prognosis in patients with non-ST elevation ACS who underwent coronary stenting.

Conflict-of-interest issues regarding the authorship or article: None declared

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Key words: Acute coronary syndrome; angina pectoris; atherosclerosis C-reactive protein; GRACE risk score; myocardial infarction; pentraxin.

Anahtar sözcükler: Akut koroner sendrom; angina pektoris; ateroskleroz; C-reaktif protein; GRACE risk skoru; miyokart enfarktüsü; pentraksin.