

## Evaluation of pentraxin-3 level and its related factors in patients undergoing primary percutaneous coronary intervention

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### Original Article

#### Abstract

**BACKGROUND:** Inflammation has an important role in the development and progression of atherosclerosis, and acute phase proteins such as pentraxin-3 (PTX3) can be deployed in determining the prognosis of coronary artery disease (CAD). So the purpose of this paper was to evaluate the PTX3 level and its related factors in patients undergoing primary percutaneous coronary intervention (PCI).

**METHODS:** In this cross-sectional study, the PTX3 levels were determined for 100 patients with ST-elevation myocardial infarction referred to the Modarres Hospital, Tehran, Iran. Checklist included demographic data [age, gender, history of myocardial infarction (MI)] and characteristics of heart disease (type of MI, culprit, and pre-dilation). PTX3 was measured for all patients before PCI.

**RESULTS:** In this study, the mean age of the participants was 58.7 (11.4). Global registry of acute coronary events (GRACE) score was higher in the group with abnormal PTX3 levels ( $P = 0.008$ ). The number of the involved vessels ( $P = 0.005$ ), MI type ( $P = 0.05$ ), and the need for PCI all had a significant relation with abnormal PTX3 levels. The increased levels of PTX3 received higher Killip class, lower ejection fraction, and higher GRACE score. The group with abnormal PTX3 had a significant difference in platelet counts ( $P = 0.018$ ) in comparison with the group with normal level of PTX3.

**CONCLUSION:** Currently, the biomarkers are highly important in the field of cardiovascular diseases. The diagnostic and prognostic importance of PTX3 as a new marker has been underscored in recent studies. Differentiating between high-risk patients with acute cardiac infarction and low-risk ones through their clinical signs is difficult.

**Keywords:** Pentraxin-3, Percutaneous Coronary Intervention, Atherosclerosis, Prognosis, Coronary Artery Disease

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#### Introduction

Inflammatory process plays a key role in the creation and development of atherosclerosis.<sup>1</sup> As a result of atherosclerosis process, the plaque formation of fibrosis-lipid (atheroma) grows gradually by aging and causes artery stenosis and inflammation, thus leading to the creation of coronary artery disease.<sup>2</sup>

Researchers have reported different factors involved in the instability and detachment of the

plaque, which are as follows: factors associated with lipids such as low density lipoprotein (LDL) and lipoprotein (LP), those associated with oxidative stress such as glutathione peroxidase and myeloperoxidase, those associated with the acute phase of inflammation and the platelets and white blood cells (WBC) regulators as biomarkers, and proteolytic enzymes such as matrix metalloproteinase.<sup>3</sup> Pentraxin-3 (PTX3) is a multi-subunit glycoprotein in acute phase of inflammation

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that can be synthesized by endothelial cells, monocytes, macrophages, and myeloid cells.<sup>2</sup> PTX3 is released from the platelet-activated neutrophils in coronary artery disease (CAD)<sup>4</sup> and through the process of inflammation, plays a role even as a prognostic factor for CAD.<sup>5</sup> The remarkable point is that PTX3 production has also been reported in the context of coronary atherosclerotic lesions.<sup>6</sup> A number of studies have indicated that high levels of serum PTX3 in unstable angina<sup>5</sup> have been associated with negative consequences after myocardial infarction (MI).<sup>7</sup> Compared with the high-sensitivity C-reactive protein (hs-CRP), PTX3 acts as a more specific factor of atherosclerosis and coronary vascular inflammation and has been independently associated with the progress of the atherosclerosis and coronary artery disease.<sup>8</sup> In one study by Wang et al. in 2014 on patients with angina who underwent percutaneous coronary intervention (PCI), the role of PTX3 was examined in predicting the elevation in cardiac troponin levels after the PCI. PTX3 levels were significantly high in patients whose troponin levels were three times higher than normal after 24 hours. In addition to the other risk factors, with logistic regression analysis, PTX3 was the most independent risk factor to increase the troponin levels after PCI.<sup>9</sup>

However, the accurate role of PTX3 in the pathogenesis of the inflammatory cardiovascular disease requires a comprehensive study. In patients with acute coronary syndrome (ACS) especially those under PCI, there are no clinically useful biomarkers for prognosis. Since inflammation has an important role in the development and progression of atherosclerosis, acute phase proteins such as PTX3 can be deployed in determining the prognosis of CAD. Nevertheless, thus far in ACS patients, particularly those who have been undergoing primary PCI, useful biomarkers have not been set clinically for the prognosis and outcome of PCI. Accordingly, the objective of this study was to evaluate the PTX3 level and its related factors in patients undergoing primary PCI.

### Materials and Methods

In this cross-sectional study, 100 patients with typical chest pain with ST-elevation on their electrocardiogram (ECG), were considered as the research statistical population who referred to Shahid Modarres Hospital, Tehran, Iran, from September 2014 to September 2015. After initial assessments, patients who underwent angiography and angioplasty were included in the study. PTX3

level was measured upon arrival (within 20 minutes of admission) in this population and patients were divided into two groups based on the level of PTX3 (with and without normal level, normal level is less than 3.1 and abnormal level is more or equal to 3.1), and then during hospitalization the associated factors according to the variables were evaluated. The variables of the questioner were age, gender, high blood pressure, hyperlipidemia, diabetes, history of PCI, coronary artery bypass grafting (CABG), MI, cigarette smoking and global registry of acute coronary events (GRACE) score. GRACE scoring system includes parameters such as age, history of congestive heart failure (CHF), history of MI, heart rate during rest, systolic blood pressure at the time of hospital admission, ST-segment deviation captured in ECG, initial serum creatinine level, and elevated cardiac enzyme level in the hospital, place of lesions on angiography, number of involved vessels, contrast nephropathy, in-hospital death, PCI candidate, and left ventricle ejection fraction (LVEF).

All patients with chest pain who had ST-elevation on ECG and underwent angiography and angioplasty were enrolled in the study and those persons who did not fill their information completely, candidates for CABG, patients with cardiogenic shock, positive cancer history, chronic kidney disease (CKD), psoriasis, treated by statins drugs, and patients without any intervention programs after triage in hospital were excluded and not used in the final analysis.

In this cross-sectional study, a check list was filled by cardiology resident for each patient. Cardiovascular assessments were also recorded and the location of the MI lesion was determined based on the ECG detected features. PTX3 was measured for all patients before PCI. The sampled blood was approximately 10 cc. Sample was measured by laboratory expert with human PTX3 enzyme linked immunosorbent assay (ELISA) kit (Cusabio Biotech Co., Japan). During hospitalization, patients were evaluated in terms of associated factors.

Categorical data are reported as number (percentage). Continuous variables are presented as mean [standard deviation (SD)]. The Shapiro-Wilks test was used to examine the normality assumption of quantitative variables. To compare demographic and baseline characteristics of groups, independent samples t-test was used for continuous variables and Pearson chi-square tests were conducted for nominal variables. P-values less than 0.05 were considered statistically significant. All tests were two-sided. The SPSS for Windows (version 18.0, SPSS Inc., Chicago, IL, USA) was used for all data analyses.

Informed written consent was obtained from all participants and the ethical committee of Shahid Beheshti University of Medical Sciences, Iran, (ethical code: IR.SBMU.RAM.REC 13940229) approved this study in 2015. The study protocol conforms to the ethical guidelines of the 2008 Declaration of Helsinki.

## Results

In this study, patients were divided into two groups based on the level of PTX3 (with and without normal level of this factor). Cut-off for normal value was 3.1 ngr/ml, based on the manufacturer kit. Accordingly, 30 persons had normal levels of PTX3 and 70 persons had abnormal levels.

In this project, 100 patients with acute ST-elevated MI (STEMI) who were candidate for primary PCI were enrolled, with the mean age of 58.7 (11.4) years. Eighty-nine (89%) participants were male which shows that male sex was a significant and meaningful risk factor. Smoking

cigarette 42 (60%) ( $P = 0.660$ ), high blood pressure 29 (41.4%) ( $P = 0.110$ ), diabetes 20 (28.6%) ( $P = 0.460$ ) and hyperlipidemia 3 (4.3%) ( $P = 0.360$ ) were other statistically significant risk factors in group with abnormal PTX3 (Table1). The distribution of cases with and without normal level of PTX3 is shown in table 1 based on quantitative variables. It was found that the difference between the groups with and without normal PTX3 level (Table 1) in terms of heart rate ( $P = 0.002$ ), GRACE score ( $P = 0.008$ ), lactate dehydrogenase ( $P = 0.026$ ), total creatine phosphokinase ( $P = 0.013$ ), duration of hospitalization ( $P = 0.004$ ), platelet count ( $P = 0.018$ ), platelet/lymphocyte ratio ( $P = 0.001$ ) has been statistically significant.

In table 2 the statistically significant and meaningful items were, type of MI ( $P = 0.050$ ), the culprit vessel ( $P = 0.030$ ), and the other terms like Killip class ( $P = 0.010$ ), stage PCI requirement ( $P = 0.005$ ) and left ventricular function ( $P = 0.007$ ) were statistically significant.

**Table 1.** Baseline characteristics of the participants

Characteristics	Normal PTX3 (n = 30)	Abnormal PTX3 (n = 70)	P
Male [n (%)]*	27 (90.0)	62 (88.6)	> 0.990
Smoking (yes) [n (%)]*	16 (53.3)	42 (60.0)	0.660
Hypertension (yes) [n (%)]*	7 (23.3)	29 (48.4)	0.110
Diabetes (yes) [n (%)]*	6 (20.0)	20 (28.6)	0.460
Affected with HLP (yes) [n (%)]*	3 (10.0)	3 (4.3)	0.360
Familial history of CAD (yes) [n (%)]*	14 (46.7)	28 (40.0)	0.660
Age (year)	55.4 ± 12.5 (40.0-91.0)	60.1 ± 10.7 (44.0-89.0)	0.070
Triglyceride (mg)	148.9 ± 58.0 (38.0-266.0)	130.3 ± 86.4 (37.0-552.0)	0.210
Cholesterol total (mg/dl)	177.0 ± 34.7 (114.0-249.0)	180.8 ± 32.4 (110.0-284.0)	0.630
Low density lipoprotein(mg/dl)	105.6 ± 32.4 (49.0-160.0)	103.7 ± 30.0 (50.0-190.0)	0.780
High density lipoprotein (mg/dl)	40.7 ± 12.3 (5.1-69.0)	43.4 ± 12.5 (23.0-79.0)	0.310
Uric acid (mg/dl)	5.5 ± 1.4 (2.2-7.9)	6.8 ± 11.6 (2.5-100.0)	0.360
Door to balloon (min)	65.5 ± 28.7 (30.0-180.0)	77.3 ± 35.7 (30.0-240.0)	0.080
Symptom to balloon (min)	241.5 ± 171.1 (72.0-2656.0)	274.1 ± 346.7 (90.0-2880.0)	0.530
Stents diameter (cm)	2.9 ± 0.4 (2.2-3.5)	3.0 ± 0.4 (2.5-4.0)	0.260
Creatinine (mg/dl)	1.1 ± 0.1 (0.8-1.3)	1.1 ± 0.2 (0.09-1.5)	0.300
Hemoglobin (Hb) (mg/d/l)	13.4 ± 1.4 (8.9-16.3)	12.7 ± 2.2 (7.3-16.8)	0.058
Systolic blood pressure (mmHg)	124.0 ± 20.1 (95.0-185.0)	124.1 ± 17.7 (90.0-165.0)	0.980
Heart rate	74.6 ± 10.5 (50.0-93.0)	82.9 ± 13.8 (45.0-119.0)	0.002
GRACE score	148.0 ± 25.2 (98.0-215.0)	177.0 ± 80.1 (120.0-798.0)	0.008
Lactate dehydrogenase (LDH)(u/l)	551.0 ± 249.0 (284.0-1153.0)	745.1 ± 611.4 (268.0-3561.0)	0.026
Creatine phosphokinase-MB (IU/l)	71.6 ± 124.4 (5.0-542.0)	127.4 ± 138.3 (10.0-550.0)	0.052
Total creatine phosphokinase (IU/l)	491.5 ± 720.7 (32.0-2800.0)	994.2 ± 1239.9 (53.0-5250.0)	0.013
Hemoglobin A1c (mmol/mol)	6.2 ± 1.9 (4.3-13.8)	6.1 ± 1.4 (4.0-11.8)	0.079
Duration of hospitalization (d)	6.2 ± 2.6 (4.0-18.0)	8.2 ± 3.7 (5.0-26.0)	0.004
Platelet count (10 <sup>9</sup> /l)	191.0 ± 27.0 (122.0-250.0)	216.1 ± 76.4 (117.0-666.0)	0.018
Platelet/lymphocyte ratio	84.6 ± 26.3 (30.0-139.0)	136.8 ± 67.0 (11.8-3561.0)	0.001
Troponin (ng/ml)	66.1 ± 37.5 (3.0-172.0)	74.5 ± 60.9 (2.0-193.0)	0.120

PTX3: Pentraxin-3; HLP: Hyperlipidemia; CAD: Coronary artery disease; GRACE: Global registry of acute coronary events; SD: Standard deviation

Data are expressed as mean ± SD (range) and P-values are based on independent Student's t-test unless otherwise stated

\* P-values based on Fisher's exact test

**Table 2.** Characteristics of heart disease in two groups of patients with normal and abnormal level of pentraxin-3

Variables	Normal PTX3 (n = 30)	Abnormal PTX3 (n = 70)	P*
Type of myocardial infarction [n (%)]			0.050
Anterior	7 (23.3)	35 (60.0)	
Lateral	4 (12.3)	1 (1.4)	
Inferior	9 (30.0)	17 (24.3)	
Posterior	0 (0)	1 (1.4)	
RV Inferior	8 (26.7)	13 (18.6)	
Inferoposterior	2 (6.7)	3 (4.3)	
Culprit [n (%)]			0.030
LAD	7 (23.3)	38 (54.3)	
RCA	18 (60.0)	23 (32.9)	
LCX	5 (16.7)	8 (11.4)	
LM	0 (0)	1 (1.4)	
Pre-dilation [n (%)]			> 0.990
Yes	7 (23.3)	17 (24.3)	
No	23 (76.7)	53 (75.7)	
Post-dilation [n (%)]			0.180
Yes	9 (30.0)	32 (45.7)	
No	21 (70.0)	38 (54.3)	
Thrombectomy [n (%)]			0.080
Yes	22 (73.3)	62 (88.6)	
No	8 (26.7)	8 (11.4)	
Killip class [n (%)]			0.010
I	29 (96.0)	35 (50.0)	
II	1 (4.0)	33 (47.1)	
III	0 (0)	2 (2.9)	
Need to repeat PCI [n (%)]			0.005
Yes	7 (23.3)	38 (54.3)	
No	23 (76.7)	32 (45.7)	
Left ventricle function [n (%)]			0.007
Normal	15 (50.0)	13 (18.6)	
Mild dysfunction	6 (20.0)	17 (35.7)	
Moderate dysfunction	7 (23.3)	25 (35.7)	
Severe dysfunction	2 (6.7)	15 (21.4)	
Quantitation of mitral regurgitation [n (%)]			0.070
Without problem	11 (36.7)	10 (14.3)	
Trivial	5 (16.7)	19 (27.1)	
Mild	7 (23.3)	23 (32.9)	
Moderate	7 (23.3)	18 (25.7)	

PTX3: Pentraxin-3; RV: Right ventricle; LAD: Left anterior descending; RCA: Right coronary artery; LCX: Left circumflex artery; LM: Left marginal; PCI: percutaneous coronary intervention

\* P-values based on chi-square test except for pre-/post-dilation, thrombectomy and need to repeat PCI which were tested with Fisher's exact test

## Discussion

Inflammatory mediators are related to a cascade of events leading to the beginning, progression, and rupture of atherosclerotic plaques. The results of our study were in agreement with those of Inoue et al.,<sup>5</sup> however, it seems necessary to conduct more studies on larger populations. Inoue et al. examined 52 patients with stable angina, averring that PTX3 does not have any association with coronary risk factors such as hypertension, diabetes mellitus, and

hyperlipidemia. PTX3 levels in unstable angina were clearly higher than those in the stable angina.<sup>5</sup> Furthermore, they suggested that PTX3 levels in patients with arterial inflammation, especially unstable angina, were increased and originated from atherosclerotic plaques. Thus, this reflects the active atherosclerosis process, indicating PTX3 as a useful predicting factor for the unstable angina.<sup>5</sup> On the contrary, Salio et al. studied the rat model and reported that PTX3 plays a crucial role as a

regulator and cardio-protector and carries out this role by regulating the complement cascade.<sup>10</sup>

In our study, GRACE score was higher in the group with abnormal PTX3 levels. GRACE risk score is “a multinational registry covering all forms of ACS, including STEMI, non-ST elevated myocardial infarction (NSTEMI), and unstable angina which consists of clinical parameters concerning patients’ mortality when at hospital and six months after discharge. GRACE scoring system includes parameters such as age, history of CHF, history of MI, heart rate during rest, systolic blood pressure at the time of admission to hospital, ST-segment depression, initial serum creatinine, and elevated cardiac enzyme level in the hospital.<sup>11</sup> Recently, Latini *et al.* reported that PTX3 as an “acute phase protein” is a 3-month mortality predictor after adjusting for major risk factors and other prognostic markers in the acute phase.<sup>7</sup>

In our study, the number of the involved vessels, MI type, stent length and lesion culprit, and the need to have PCI all had a significant relation with abnormal PTX3 levels, representing the severity of coronary artery disease in patients with abnormal levels of PTX3.

In the study by Namazi *et al.*, increased levels of PTX3 were high differentiation indices for high SYNTAX score, and PTX3 levels higher than 0.29 ng/dl had high specificity for the detection of coronary stenosis complex.<sup>12</sup> In the study by Karakas *et al.* carried out on patients with chronic stable angina, PTX3 was related to complexity with hs-CRP and severity of coronary artery disease and was an independent predictor for higher SYNTAX score.<sup>13</sup>

In our study, the increased levels of PTX3 received higher Killip class, lower ejection fraction, and higher GRACE score. To evaluate the role of PTX3 in the pathology of CHF, more interventions in larger populations are required as multicenter trials. Thus far, several studies have suggested that plasma PTX3 levels increase in patients with heart failure and are independently associated with the increasing risk of cardiac events. Suzuki *et al.* showed that plasma PTX3 levels in patients with heart failure were significantly higher than those in the control group, and by improving New York Heart Association (NYHA) class, particularly in patients with severe heart failure, the functional classes of III and IV increase.<sup>14</sup>

In our study, the group with abnormal PTX3 had a significant difference in platelet counts and platelet cells in comparison with the group with the normal level of PTX3. By using Pearson correlation,

a weak correlation ( $r = 0.4$ ) was found between the level of PTX3 and TIMI frame count; moreover, a weak correlation ( $r = 0.4$ ) was discovered between platelets and lymphocytes ratio and the level of PTX3. In both cases, the calculated correlation was significant ( $P < 0.001$ ). Furthermore, previous studies have indicated that higher amounts of platelets and lymphocyte counts were less associated with adverse cardiovascular outcomes. Currently, platelet-lymphocyte ratio (PLR) has been proposed as a new negative predictor of cardiovascular consequences.<sup>15</sup> Azab *et al.* demonstrated that higher levels of PLR act as long-term mortality markers in patients with NSTEMI.<sup>16</sup> Moreover, Balta and Ozturk stated that PLR may be a useful inflammatory indicator in clinical practice.<sup>17</sup> A strong correlation has been found between acute phase reactants, pro-inflammatory proteins such as hs-CRP, tumor necrosis factor (TNF), interleukin-1 (IL-1), and IL-6, and increased platelet counts in non-specific inflammatory conditions.<sup>18</sup> Increased platelet count may reflect a basic inflammatory marker by which various inflammatory mediators stimulate the proliferation of megakaryocytes, thus leading to relative thrombocytosis. However, studies have shown that in patients with coronary atherosclerosis, the increased levels of platelets and monocytes accumulate in the peripheral blood, which is associated with plaques sustainability. Duygu *et al.* showed that the average size of plaques as an index of platelet activation is associated with the reduced coronary flow after the coronary intervention.<sup>19</sup> Higher platelet counts are markers of the pre-thrombotic situation. On the other hand, higher platelet counts may indicate a higher proportion of platelet-rich thrombi in atherosclerotic plaques, resulting in worse outcomes.<sup>18</sup>

There are some limitations to this study that should be acknowledged. Our study was conducted in one medical center. Our recommendation is to conduct a multi-central study so that it can be more comprehensive. The sample size was small and we could investigate a single marker. Future studies should have a bigger sample size and other markers should be measured in order to get a comprehensive result and conclusion.

## Conclusion

Currently, the biomarkers are extremely important in the field of cardiovascular diseases. The diagnostic and prognostic importance of PTX3 as a new marker has been underscored in recent studies, and it can play a significant role to triage patients

with MI. Differentiating between high-risk patients with acute cardiac infarction and low-risk ones through their clinical signs has been difficult. In this study, we found that people with a higher PTX3 level are in greater urgency for hospitalization and treatment compared with normal PTX3 level.

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### Conflict of Interests

Authors have no conflict of interests.

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