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ORIGINAL ARTICLE

Medicine Science International Medical Journal

Medicine Science 2022;11(3):1098-104

Levels of serum platelet-derived growth factor-β (PDGF-β) and factors affecting mortality in patients with acute myocardial infarction

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Received 08 March 2022; Accepted 04 April 2022
Available online 20.07.2022 with doi: 10.5455/medscience.2022.02.047

Abstract

Acute myocardial infarction (AMI) is the necrosis in the heart tissue resulting from obstruction of the coronary arteries. The main phenomenon in coronary artery disease and the formation of acute coronary syndromes is atherosclerosis which develops following the formation of coronary plaque. Platelet-derived growth factors (PDGF) that are produced by platelets, macrophages and endothelial cells are actively involved in atherosclerosis. The present study is, therefore, intended to investigate the diagnostic value of PDGF-β, as well as the factors affecting mortality in patients with acute myocardial infarction. According to the comparison of the study groups in terms of their PDGF-β levels, the NSTEMI group was found to have the highest level of PDGF-β. In the intergroup comparison of the subgroups of the patient group, the NSTEMI group was found to have significantly higher levels of PDGF-β than the STEMI group had (Post-Hoc test p=0.001). In the analyses of the values measured in the blood samples collected at the time of admission to the ED, PDGF-β was found to have a moderate positive linear relationship with WBC, troponin and lactate values. According to the analyses of the subgroups of the patient group, only in the NSTEMI group was there a moderate positive linear relationship between troponin and PDGF-β. We investigated whether serum PDGF-β is the new prognostic biomarker for AMI, serum PDGF-β levels were significantly higher, especially in patients with NSTEMI. However, in order for serum PDGF-β levels to be used as a marker for predicting prognosis, further comprehensive studies that focus on this subject are needed.

Keywords: Serum platelet-derived growth factor-β (PDGF-β), acute myocardial infarction, mortality

Introduction

Acute myocardial infarction (AMI) is the necrosis in the heart tissue resulting from obstruction of the coronary arteries, which leads to inadequate blood supply to the cardiac muscles since these arteries feed the heart. Acute myocardial infarction is accompanied by atherosclerosis as the underlying disease and has high rates of mortality despite improved treatment methods [1,2]. Acute myocardial infarction is divided into two subgroups, which are ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) [1,3]. Rapid diagnosis of patients with acute myocardial infarction bears significant importance for predicting and preventing various complications that develop subsequent to AMI. AMI is diagnosed in reference to patient history, ECG, and biomarkers. Since laboratory examinations that are used for diagnostic purposes such as those of troponin levels can detect elevations due to secondary reasons, there are ongoing efforts to find other biomarkers with improved specificity for AMI diagnosis [1,4,5].

The main phenomenon in the coronary artery disease and the formation of acute coronary syndromes is atherosclerosis that develops following the formation of coronary plaque [1,6]. Platelet-derived growth factors (PDGF) that are produced by platelets, macrophages and endothelial cells are actively involved in atherosclerosis [7-9]. In the situation of lower oxygen pressure, the level of PDGF–β and thrombin increases and it plays an important role in the formation of atherosclerosis. PDGF-β has a chemoatractant effect on plateletes and monocytes and as a result, there is a formation of plaque. Finally the overexpression of PDGF-β results with growing of atherosclerotic plaque.

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The present study is, therefore, intended to investigate the diagnostic value of serum platelet-derived growth factor-β (PDGF-β), as well as the factors affecting mortality in patients with acute myocardial infarction.

Materials and Methods

The current study is a prospective clinical trial conducted in the emergency department of a tertiary university hospital between 03.03.2021 - 01.09.2021. The study was conducted in accordance with the World Medical Association's Declaration of Helsinki for studies involving human subjects. Before starting the study, the study approval was obtained from our university's Scientific Research Ethics Committee (protocol no: TÜTF-BAEK 05/01 dated 01/03/2021). Our study included 50 patients (patient group) diagnosed with Acute Myocardial Infarction in the emergency department and 25 healthy volunteers (control group) who did not have any chronic diseases. The exclusion criteria herein included being under the age of 18, having congenital or acquired thrombocytopenia/thrombophilia, pregnant women, as well as those who are on estrogen replacement therapy or using oral contraceptive pills, those with active cancer diagnosis over the last five years, those with unidentified patient outcome, and those not interested in participating in the study. Each patient was then given a phone call for follow-up purposes on the 30th day of the infarction, following their discharge from the hospital.

Serum PDGF-β analysis

The Human PDGF-β Elisa kit with the catalogue number 201-12-2119 from the brand SunRed was used to determine the levels of serum platelet-derived growth factor-beta. The measurements were carried out in the Medical Biochemistry Department Laboratory installed within our university, using the μQuant™ model device with the serial number 218731 from the brand BioTek Instruments.

Statistical Analysis

To assess continuous variables for their distribution normality, the Shapiro-Wilk test was applied. The data obtained was compared using the Student t test (Mann-Whitney U test for non-normally distributed data) between the two groups. To compare the data among three or more groups, One-Way Analysis of Variance was performed (Kruskal Wallis test for non-normally distributed data (Dunn's Post-Hoc test)). The relation between two categorical variables was examined using Pearson's Chi-square test and Fisher's Exact test. The data were expressed as median, minimum and maximum values, while the categorical variables were expressed in percentage. The association between two continuous variables was evaluated using Spearman's rank-order correlation coefficient. The statistical software SPSS version 23 (SPSS Inc., Armonk, NY) was used for all the analyses conducted herein. The significance level was considered $p<0.05$.

Results

Demographics

The control group had a mean age of 61.8±10.5 years, while the group of patients with NSTEMI had a mean age of 63.8±12.8 and those with STEMI had a mean age of 63.2±14.3 years. The study groups were compared and found not to have any statistically significant difference in terms of age and gender (p=0.847 and p=0.761, respectively) (Table 1).

Table 1. Demographic characteristics and vital findings of the study groups

	Total	Control Group	NSTEMI	STEMI	p
Age, (year)					
Mean±SD Med (min-max)	62.9±12.5 63 (38-94)	61.8±10.5 62 (40-83)	63.8±12.8 63 (45-90)	63.2±14.3 67 (38-94)	0.847[a]
Gender n (%)					
Female	55 (73.3)	17 (68)	19 (76)	19 (76)	0.761[b]
Male	20 (26.7)	8 (32)	6 (24)	6 (24)	
Vital findings Mean±SD, Med (min-max)					
Systolic Blood Pressure (mmHg)	125.9±21.3 127 (49-170)	129.6±6.9 130 (114-143)	130.4±26.4 132 (70-170)	117.8±23.3 124 (49-150)	0.036
Diastolic Blood Pressure (mmHg)	125.9±21.3 127 (49-170)	61.4±4.2 61 (54-68)	62.2±12.6 65 (30-89)	59.6±14.1 62 (21-90)	0.640
Apex beat (/min)	61.1±11.1 62 (21-90)	74±6.1 74 (64-83)	88.8±19.6 84 (65-134)	74±14.1 73 (40-111)	0.004
Axillary body temperature (°C)	79±15.8 75 (40-134)	36.5±0.3 36.5 (35.9-37.2)	36.6±0.3 36.7 (36-37.1)	36.5±0.3 36.5 (36.1-37)	0.446[c]
Respiratory rate (/min)	36.6±0.3 16 (12-37)	15.1±1.9 15 (12-18)	17.5±4.1 16 (13-30)	18.4±6.1 16 (13-38)	0.037

	p[c]		
	Control - NSTEMI	**Control - STEMI**	**NSTEMI - STEMI**
Systolic Blood Pressure (mmHg)	0.738	0.040	0.017
Apex beat (/min)	0.006	0.755	0.002
Respiratory rate (/min)	0.038	0.019	0.780

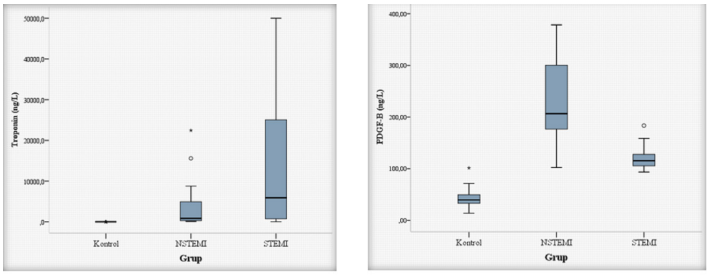
Mean±SD: mean ± standard deviation, Med (min-max): median (minimum-maximum).
a: Student t test, **b:** Pearson Chi-square test, **c:** Dunn's Post-Hoc test

Vital Findings

When examined for vital findings, the study groups were found to differ in terms of systolic blood pressure, apex beat and respiratory rate (p=0.036, p=0.004 and p=0.037, respectively). In the intergroup comparisons of multiple groups in terms of their vital findings, there were no significant difference found between the NSTEMI and STEMI groups in terms of respiratory rate (Post-Hoc test p=0.780) (Table 1).

Laboratory results

According to the comparison between the study groups in terms of Hb, RBC, and PLT counts, there were no statistically significant difference found (p>0.05). In the study, the NSTEMI and STEMI groups were found to have significantly higher levels of WBC than that of the control group (p<0.001). In the intergroup comparison of the subgroups of the patient group, they were found not to have any statistically significant difference in terms of WBC (Post-Hoc test p=0.358) (Table 2).



(a) (b)

Figure 1. Troponin (a) and PDGF-β (b) values by study groups

According to the comparison of the study groups in terms of their troponin levels, the STEMI group was found to have the highest level of troponin (p<0.001). In the intergroup comparison of the subgroups of the patient group, there was no significant difference between the NSTEMI and STEMI groups (Post-Hoc test p=0.141) (Table 2, Figure 1).

Table 2. Laboratory results of the study groups

Laboratory results Mean±SD, Med (min-max)					
	Total	**Control Group**	**NSTEMI**	**STEMI**	**p a**
WBC (10^3/ µL)	10.6±4.9 9.8 (3.8-31.2)	6.5±1.4 6.5 (3.8-10.5)	11.8±4.2 11 (6-21.4)	13.4±5.3 12.6 (4.59-31.2)	<0.001
RBC (10^6/ µL)	4.5±0.6 4.6 (2.7-5.8)	4.4±0.6 4.5 (3-5.4)	4.6±0.7 4.6 (3.1-5.8)	4.5±0.6 4.6 (2.7-5.3)	0.428
HB (g/dL)	13.8±2 14.5 (7.9-16.5)	14.7±0.9 14.6 (13.1-16.4)	13.6±1.8 13.8 (9.9-16.5)	13.2±2.7 14.5 (7.9-16.1)	0.109
PLT (10^3/ µL)	268.6±91.2 251 (90-606)	269.4±83.3 256 (156-476)	273.2±91.6 243 (182-606)	263.3±101.4 251 (90-592)	0.908
APTT (/sec)	28.1±14.4 23.7 (18.2-120)	23±1.3 23.4 (20.7-26.4)	25.6±7.2 23.7 (18.2-55.4)	35.7±22.1 28.2 (18.6-120)	<0.001
INR	1±0.2 1 (0.76-1.8)	0.9±0.1 1 (0.76-1.2)	1±0.1 1 (0.87-1.47)	1.1±0.2 1.1 (0.93-1.8)	<0.001
C-reactive protein (mg/L)	2.5±5.6 0.4 (0.2-28)	0.4±0.2 0.3 (0.3-1.1)	2.4±4.7 0.8 (0.2-20.6)	4.6±8.1 0.8 (0.3-28)	0.011
GFR	88±25.9 97 (14-123)	100.8±8.9 100 (85-123)	83.3±24.3 90 (14-119)	80±33.9 96 (16-118)	0.031
Troponin (ng/L)	6635.1±12725.7 369 (1.5-50000)	1.8±0.6 1.5 (1.5-3.6)	3561.9±5443.7 843 (100-22451)	16341.5±17773 5922 (19-50000)	<0.001
PDGF-β (ng/L)	129.2±88.7 114.4 (13.8-378.4)	43.1±17.7 39.4 (13.8-101.4)	225.7±77.6 206.5 (102.4-378.4)	118.8±20.1 115.5 (93.5-183.5)	<0.001
Lactate (mg/dL)	14.3±15.5 9 (1.2-106)	6±1.3 6 (4-8)	19.6±19.7 13 (7-106)	17.4±15.4 13 (1.2-71)	<0.001

pb			
	Control - NSTEMI	**Control - STEMI**	**NSTEMI - STEMI**
WBC (10^3/ µL)	<0.001	<0.001	0.358
APTT (/sec)	0.197	<0.001	0.003
INR	0.024	<0.001	0.042
C-reactive protein (mg/L)	0.010	0.010	0.997
GFR	0.013	0.043	0.645
Troponin (ng/L)	<0.001	<0.001	0.141
PDGF-β (ng/L)	<0.001	<0.001	0.001
Lactate (mg/dL)	<0.001	<0.001	0.387

Mean±SD: mean ± standard deviation, Med (min-max): median (minimum-maximum)
a: Kruskal Wallis test, **b:** Dunn's Post-Hoc test

According to the comparison of the study groups in terms of their PDGF-β levels, the NSTEMI group was found to have the highest level of PDGF-β (p<0.001). In the intergroup comparison of the subgroups of the patient group, the NSTEMI group was found to have significantly higher levels of PDGF-β than the STEMI group had (Post-Hoc test p=0.001) (Table 2, Figure 1).

According to the comparison of the study groups in terms of their lactate levels, the NSTEMI group was found to have the highest lactate level (p<0.001). In the intergroup comparison of the subgroups of the patient group, there was no significant difference between the NSTEMI and STEMI groups (Post-Hoc test p=0.387) (Table 2).

According to the comparison of the study groups in terms of their APTT values, the STEMI group was found to have the highest APTT value (p<0.001). In the intergroup comparison of the subgroups of the patient group, the STEMI group was found to have significantly higher values of APTT than the NSTEMI group had (Post-Hoc test p=0.003) (Table 2).

PDGF-ß levels of the study groups

The results from the analyses on PDGF-β levels of the study groups and the findings from the intergroup comparisons of multiple groups are shown in Table 2.

Relationship between demographic characteristics and PDGF-ß levels

In the study groups, the PDGF-ß levels were found to have no significant linear relationship with age and gender (Table 3).

Relationship between vital findings and PDGF-ß levels

The PDGF-ß levels were found to have no significant linear relationship with the systolic blood pressure, diastolic blood pressure, respiratory rate and axillary body temperature measured at the time of admission to the emergency room (p>0.05). Of all the study groups examined here, only in the STEMI group was there a minor positive linear relationship between PDGF-β level and the apex beat measured at the time of admission (r=0.406, p=0.044) (Table 3).

Table 3. Relationship of PDGF-ß level with demographic characteristics, vital findings and laboratory results

		Total	Control Group	NSTEMI	STEMI
Age	p	0.884	0.550	0.621	0.519
Gender	p	0.578	0.582	0.977	0.475
Vital findings					
Systolic Blood Pressure (mmHg)	r	0.032	-0.116	-0.104	0.139
	p	0.787	0.582	0.621	0.508
Diastolic Blood Pressure (mmHg)	r	0.102	-0.192	0.020	-0.051
	p	0.384	0.358	0.924	0.808
Apex beat (/min)	r	0.282	-0.175	-0.110	0.406
	p	0.014	0.402	0.600	0.044
Axillary body temperature (°C)	r	0.085	-0.116	0.105	-0.265
	p	0.471	0.581	0.618	0.201
Respiratory rate (/min)	r	0.195	-0.226	-0.190	0.274
	p	0.093	0.276	0.362	0.185
Laboratory results					
WBC (10^3/ µL)	r	0.524	-0.075	0.367	-0.160
	p	<0.001	0.721	0.071	0.446
Troponin (ng/L)	r	0.665	0.054	**0.635**	0.145
	p	<0.001	0.796	**0.001**	0.490
Lactate (mg/dL)	r	0.593	0.142	-0.054	0.065
	p	<0.001	0.497	0.796	0.759

r: Spearman's rank-order correlation coefficient

Relationship between laboratory results and PDGF-ß levels

In the analyses on the values measured in the blood samples collected at the time of admission to the emergency room, PDGF-β was found to have a moderate positive linear relationship with WBC, troponin and lactate values (r=0.524, p<0.001, r=0.665, p<0.001, r=0.593 and p<0.001, respectively). According to the analyses on the subgroups of the patient group, only in the NSTEMI group was there a moderate positive linear relationship between troponin and PDGF-β (r=0.635, p=0.001) (Table 3).

Mortality analysis

The mortality rate in the group of patients with acute myocardial infarction in the first 30-day period was 12% (n=6). 2 (8%) patients died in the NSTEMI group and 4 (16%) died in the STEMI group. There was no significant difference between the subgroups pf the patient group in terms of mortality (p=0.155).

According to the analyses performed to identify the demographic factors that affected mortality in the patient group, there was no significant correlation between gender and mortality (p=0.621) but the group of deceased patients had a higher mean age than that of the surviving group (p=0.013) (Table 4).

As for the relationship between mortality and the vital findings in the group of patients diagnosed with acute myocardial infarction, the group of deceased patients was found to have significantly lower levels of systolic blood pressure and diastolic blood pressure,

while the respiratory rate per minute was found to be higher in the surviving group (p<0.001). In the patient group, mortality was found not to have any significant correlation with apex beat and axillary body temperature (p=0.105 and p=0.356, respectively). The results from the analysis on mortality and vital findings are as shown in Table 4.

According to the analysis on the relationship between mortality and laboratory results in the group of patients with acute myocardial infarction, the group of deceased patients had significantly higher level of INR and lactate (compared to the group of surviving patients), while their RBC, Hemoglobin and GFR values were found to be lower (again compared to the surviving group). The two groups of deceased and surviving patients were found not to have any significant differences in terms of WBC, PLT, CRP, APTT and troponin levels. Since the deceased patients (the number of observations) were not adequately distributed in the study groups, the relationship between mortality and PDGF-β level could not studied.

In the group of patients with acute myocardial infarction, 13.3% (n=6) of the 45 patients with wall-motion abnormalities detected in transthoracic echocardiography died within the first 30 days, while none of the 5 patients with no wall-motion abnormality died (p=1.000) (Table 4).

When the patient group was examined for accompanying comorbidities, chronic hypertension was found to be the most common disease. The levels of serum PDGF-β were significantly higher in the patients with hypertension than in those without hypertension. According to the study of the relationship between hypertension and mortality, the mortality rate was 20.8% (n=5) in the presence of hypertension, while it was 3.8% (n=1) in the group without hypertension.

Table 4. Analyses on mortality

Demographics (Patient group)		**Survived (n=44)**	**Deceased (n=6)**	**p[a]**
Age (year)		61.6±12.4	77.3±13.6	0.013
Gender (n.%)	Female	34 (89.5)	4 (10.5)	0,621
	Male	10 (83.3)	2 (16.7)	
Vital findings (Mean±SD)				**p [b]**
Systolic Blood Pressure (mmHg)		130.9±17.7	74±15.5	<0.001
Diastolic Blood Pressure (mmHg)		64.3±9.5	36.3±11.6	<0.001
Apex beat (/min)		79.9±16.5	92.8±28.5	0.105
Axillary body temperature (°C)		36.6±0.3	36.7±0.3	0.356
Respiratory rate (/min)		16.4±2.6	29±6.2	<0.001
Laboratory results (Mean±SD)				**p [b]**
WBC (10^3/ µL)		11.9±3.4	17.8±9.2	0.081
RBC (10^6/ µL)		4.7±0.5	3.8±0.8	0.013
HB (g/dL)		13.8±2	10.8±2.5	0.009
PLT (10^3/ µL)		266.3±82.8	282.3±175.1	0.873
APTT (/sec)		31.1±18.1	27.4±4.7	0.694
INR		1.1±0.2	1.2±0.2	0.019
C-reactive protein (mg/L)		2.6±5.5	10.4±10.7	0.189
GFR		86.2±27.1	48.7±23.6	0.002
Troponin (ng/L)		9224±13916	15284±19037	0.199
Lactate (mg/dL)		14.2±7.3	50±34.5	0.006
Findings on transthoracic echocardiography				
Any wall-motion abnormality detected	Yes	39 (86.7)	6 (13.3)	1.000[a]
	No	5 (100)	-	
		PDGF-ß (ng/L)(Mean±SD)		
	Control Group	**NSTEMI**	**STEMI**	
Survived (n=44)	43.1±17.7	226±72.6	119±21.8	
Deceased (n=6)	-	222.1±169.3	117.6±8.5	

[a]: Fisher's exact test, **[b]:** Mann-Whitney U test

Discussion

The main phenomenon in the coronary artery disease and the formation of acute coronary syndromes is atherosclerosis that develops following the formation of coronary plaque. Acute coronary syndrome is a life-threatening symptom of atherosclerosis [1]. Platelets play an important role in the pathogenesis of atherosclerosis and in the process of thrombus formation around plaque following a plaque rupture in coronary arteries [10]. Platelet-derived growth factors that are produced particularly by platelets, as well as by macrophages and endothelial cells, are actively involved in atherosclerosis [7-9]. PGDF consists of two homologous polypeptide chains (A and B). In cases of exposure to low oxygen pressure and excessive thrombin, such as acute coronary syndromes, the synthesis and release of PDGF-β increases significantly, particularly in endothelium and platelets [7,8,11,12]. Therefore, PDGF-β is considered a key mediator in the formation of thrombosis. According to current studies, concentrations of PDGF-β that are found to be over a standard level in healthy or normal people are indicative of the risk or presence of thrombosis

[7,9]. The present study is therefore intended to investigate whether serum PDGF-β level has diagnostic value for cases of acute myocardial infarction and is correlated with mortality.

Endothelial dysfunction plays a very important role in the progression of atherosclerosis in the coronary arteries. After endothelial dysfunction, inflammation and atherosclerotic plaque forms. Therefore, atherosclerosis is a process of multifactorial inflammation [1,6]. Given the fact that atherosclerosis is actively involved in the pathogenesis of many cardiovascular diseases, it is clearly understood that acute phase reactants (C-reactive protein, WBC) are closely associated with the presence of atherosclerosis. In the current study, this part addresses the values of acute phase reactants measured in the study groups and compares these measurements with the levels of serum PDGF-β detected. There are studies in the literature that focus on this topic and investigate the diagnostic value of acute phase reactants in acute coronary syndromes, as well as their predictive value for the severity and prognosis of diseases. The correlation between the elevation in systemic indicators of inflammation such as CRP and WBC and the clinical characteristics of acute coronary syndromes has been demonstrated in many studies. In patients with acute myocardial infarction, high levels of CRP are reported to be closely related to mortality and to remain a useful marker for predicting the width of the infarction area and possible cardiac rupture [13,14]. Similarly, a study by Açıkel et al. [13]. stated that the high CRP and WBC values measured at the time of admission are associated with an increased risk of heart failure and higher mortality. In acute myocardial infarction, increased white blood cell count is reported to be accompanied by decreased epicardial blood flow, myocardial perfusion abnormality, treatment-resistant thrombus and poor clinical outcomes [15,16]. On the contrary, another study carried out by Topal et al. [15] in Turkey reported that increased serum CRP levels in acute MI patients are not correlated with the type and width of infarction.

To summarize the data obtained in our study, there were no difference detected between the group of patients with acute myocardial infarction and the control group in terms of CRP values. Again, there was no difference in terms of the type of acute myocardial infarction and the separation of subgroups (NSTEMI-STEMI). Another acute phase, WBC values were analyzed, and the WBC values measured in patients with acute myocardial infarction were found to be higher than that of the control group, while there were not any difference between the subgroups of the patient group with myocardial infarction. It was additionally another finding that the two groups of surviving and deceased patients did not differ in terms of their CRP and WBC values measured. In the light of the data obtained herein the present study, we concluded that high levels of acute phase reactants are not useful in identifying the type of myocardial infarction and predicting mortality. Additionally, increased levels of CRP were not significantly correlated with serum PDGF-β values, while there was a positive linear relationship between WBC and serum PDGF-β. Since the deceased patients (the number of observations) were not adequately distributed in the study groups, we were unable to conduct an analysis to find out whether increased levels of serum PDGF-β can be used to predict mortality in patients with acute myocardial infarction.

Anemia leads to increased chance of acute coronary syndrome to develop and negatively affects the prognosis as it elevates myocardial ischemia. In a study by Sabatine et al. [17] where 39922 patients were included, the researchers found that anemia is an independent and important risk factor for the occurrence of undesired cardiovascular events in patients with ACS. According to this study, the risk of recurrent ischemia and death was increased in patients with non-ST elevation myocardial infarction (NSTEMI) and anemia. In a prospective cohort study by Dauerman et al. that included 5378 patients [18], anemic patients with acute myocardial infarction were also found to have an increased risk of mortality. Another similar study was conducted by Gül et al. [19] on 570 patients in the Coronary Intensive Care Unit for AMI diagnosis. In this study, the mortality rate in anemic cases was significantly higher than in non-anemic patients. In the CADILLAC study by Nikolsky et al. [20], the researchers found that anemia increased mortality and the frequency of unwanted cardiac events to occur. Similarly in our study as well, the group of deceased patients were found to have lower RBC and Hemoglobin values than the survivors did. Another finding we present herein is that serum PDGF-β was not significantly correlated with RBC and Hemoglobin values.

Blood lactate levels are often used in clinical practice to determine the severity of the disease and to assess the response to therapeutic interventions [21]. In patients with acute heart failure following acute myocardial infarction, blood lactate levels elevate as a result of decreased oxygen transmission due to low heart flow and decreased tissue perfusion. In these patients, serious irreversible organ dysfunctions may develop due to the common impact that congestion and hypoxia impose. This factor is involved in poor prognosis [22]. In a study conducted by Gjesdal et al. [23], the rate of 30-day mortality was found to be significantly higher in the group of patients suffering from acute myocardial infarction who do not present with cardiogenic shock and who suffer from moderate heart failure with a lactate level of ≥2.5 mmol/L. In another study by Liang et al. [24], the rates of 30-day and 180-day mortality were significantly higher in the group of patients with acute coronary syndrome whose blood lactate levels were measured as ≥2.7 mmol/L at the time of admission.

Similarly, in our study, the group of deceased patients had higher lactate values than the survivors did. We found a positive linear relationship between lactate value and serum PDGF-β value.

Cardiac troponins are routinely used in the diagnosis of acute myocardial infarction. Blood concentration generally increases due to myocardial damage. Each increase in the concentration of troponins is associated with the severity and poor prognosis of ACS (4). In our study, unlike the available literature, there was no significant difference between the groups of deceased and surviving patients in terms of troponin levels. In addition, a moderately linear relationship was detected between serum PDGF-β levels and troponin values. Although our study found a positive linear relationship between serum PDGF-β level and troponin value, there is still need for further prospective studies including larger numbers of patients where troponin and serum PDGF-β levels are evaluated together focusing on their predictive value for mortality in patients with acute myocardial infarction.

Conclusion

In light of the data obtained in this study, where we investigated whether serum PDGF-β is the new prognostic biomarker for

AMI, serum PDGF-β levels were significantly higher especially in patients with NSTEMI. We think that; it could be related to non-complete obstruction in the situation of NSTEMI and the slow formation of plaque in the vessel. Because the cases of STEMI occurs more acutely and there is no time for the elevation of PDGF-β. So in the diagnosis of NSTEMI, combination of PDGF-β levels with other cardiac markers could be more helpful for clinicians. However, in order for serum PDGF-β levels to be used as a marker for predicting prognosis, further comprehensive studies that focus on this subject are needed.

Limitations of the study

The main limitation of our study is that it is single-centered and included a small number of patients. Additionally, among other limitation are the facts that the patient group consists only of patients diagnosed with MI, patients admitted to the emergency service for chest pain and then given non-ACS diagnoses were excluded from the study, the patients who died were not adequately distributed among the study groups, and the serum PDGF-β level has only been studied once in each patient.

Footnote

This study is the speciality thesis in Emergency Medicine of Mehmet Selim Özbilen.

Conflict of interests

The authors declare that there is no conflict of interest in the study.

Financial Disclosure

The authors declare that they have received no financial support for the study.

Ethical approval

The study approval was obtained from our university's Scientific Research Ethics Committee (protocol no: TÜTF-BAEK 05/01 dated 01/03/2021).

References

1. Solati Z, Ravandi A. Lipidomics of Bioactive Lipids in Acute Coronary Syndromes. Int J Mol Sci. 2019;20:1051.
2. Onat A, Karakoyun S, Akbaş T, et al. Turkish adult risk factor survey 2014: Overall mortality and coronary disease incidence in Turkey's geographic regions. Turk Kardiyol Derg Arşivi. 2015;43:326-32.
3. Anderson JL, Morrow DA. Acute Myocardial Infarction. N Engl J Med. 2017;376:2053-64.
4. Ustündağ M, Orak M, Güloğlu C, et al. Comparative diagnostic accuracy of serum levels of neutrophil activating peptide-2 and pentraxin-3 versus troponin-I in acute coronary syndrome. Anadolu Kardiyol Derg. 2011;11:588-94.
5. Berezin AE, Berezin AA. Adverse Cardiac Remodelling after Acute Myocardial Infarction: Old and New Biomarkers. Dis Markers. 2020; 2020:1215802.
6. Mallika V,Goswami B,Rajappa M. Atherosclerosis pathophysiology and the role of novel risk factors:a clinicobiochemical perspective. Angiology. 2007;58:513-22.
7. Sakçi D, Sayhan MB, Salt Ö, et al. A Novel Diagnostic Marker in Pulmonary Thromboembolism? Platelet-Derived Growth Factor –β. Dicle Medical Journal. 2022;1;1-11.
8. Bruzelius M, Iglesias MJ, Hong MG et al. PDGFB, a new candidate plasma biomarker for venous thromboembolism: Results from the VEREMA affinity proteomics study. Blood. 2016;128:e59-66.
9. Alhabibi AM, Eldewi DM, Wahab MAA, et al. Platelet-derived growth factor-beta as a new marker of deep venous thrombosis. J Res Med Sci. 2019;22:48.
10. Bath PM, Butterworth RJ. Platelet size: measurement, physiology and vascular disease. Blood Coagul Fibrinolysis. 1996;7:157-61.
11. Tannenberg P, Chang YT, Muhl L, et al. Extracellular retention of PDGF-B directs vascular remodeling in mouse hypoxia-induced pulmonary hypertension. Am J Physiol Lung Cell Mol Physiol. 2018;314:L593-605.
12. Das M, Dempsey EC, Bouchey D, et al. Chronic hypoxia induces aggregated growth responses in pulmonary arterial adventitial fibroblast. Am J Respir Crit Care Med. 2000;22:15-25.
13. Açıkel M, Erol MK, Yılmaz M, et al. The effect of increased acute phase reactants on early phase prognosis in acute myocardial infarction. MJAU. 2001;33:17-21.
14. Horne BD, Anderson JL, John JM, et al. Intermountain heart collaborative study group. Which white blood cell subtypes predict increased cardiovascular risk? J Am Coll Cardiol. 2005;45:1638–43.
15. Topal K, Sandıkçı S, Demirhindi H, et al. Relationship Between Elevated Serum CRP Level and Diabetes Mellitus In Acute Myocardial Infarction. Marmara Medical Journal. 2006;19;58-64.
16. Barron H, Cannon C, Murphy S, et al. Association between white blood cell count, epicardial blood flow, myocardial perfusion, and clinical outcomes in the setting of acute myocardial infarction thrombolysis in myocardial infarction 10 sub study. Circulation. 2000; 102:2329–34.
17. Sabatine MS, Morrow DA, Cannon CP, et al. Relationship between baseline white blood cell count and degree of coronary artery disease and mortality in patients with acute coronary syndromes: a TACTICS-TIMI 18 (Treat Angina with Aggrastat and determine Cost of Therapy with an Invasive or Conservative Strategy–Thrombolysis In Myocardial Infarction 18 trial substudy. J Am Coll Cardiol. 2002;40:1761–8.
18. Dauerman HL, Lessard D, Yarzebski J, et al. Bleeding complications in patients with anemia and acute myocardial infarction. Am J Cardiol. 2005;96:1379-83.
19. Gül R, Alibaz Öner F, Yurdakul S, et al. The effect of anemia on mortality in patients with acute coronary syndromes. Nobel Med. 2011;7:78-81.
20. Nikolsky E, Aymong ED, Halkin A, et all. Impact of anemia in patients with acute myocardial infarction undergoing primary percutaneous coronary intervention: Analysis from the Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications (CADILLAC) Trial. J Am Coll Cardiol. 2004;44:547-55.
21. Logeart D, Thabut G, Jourdain P et al. Predischarge B-type natriuretic peptide assay for identifying patients at high risk of re-admission after decompensated heart failure. J Am Coll Cardiol. 2004; 43:635-41.
22. Zymlinski R, Biegus J, Sokolski M et al. Increased blood lactate is prevalent and identifies poor prognosis in patients with acute heart failure without overt peripheral hypoperfusion. European Journal of Heart Failure. 2018; 20:1011–8.
23. Gjesdal G, Braun CÖ, Smith JG, et al. Blood lactate is a predictor of short-term mortality in patients with myocardial infarction complicated by heart failure but without cardiogenic shock. BMC Cardiovasc Disorder. 2018;18:8.
24. Liang D, Zhou X, Hong X et al. Association between admission lactate levels and mortality in patients with acute coronary syndrome: a retrospective cohort study. Coron Artery Dis. 2019;30:26–32.