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ORIGINAL ARTICLE

Wiktoria Smyła[1], Sebastian Krych[1], Michał Jurkiewicz[1], Zenon Czuba[2], Michał Skrzypek[3], Patrycja Piłat[1], Mariusz Gąsior[4], Bożena Szyguła-Jurkiewicz[4]

[1]Student's Scientific Association, 3rd Department of Cardiology, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice
[2]Department of Microbiology and Immunology, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice
[3]Department of Biostatistics, Faculty of Health Sciences in Bytom, Medical University of Silesia, Katowice
[4]3rd Department of Cardiology, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice

Serum pentraxin and E-selectin levels are associated with outcomes in patients with acute myocardial infarction who undergo percutaneous coronary intervention

Corresponding author:

Bożena Szyguła-Jurkiewicz
3rd Department of Cardiology,
Faculty of Medical Sciences in Zabrze,
Medical University of Silesia, Katowice,
Marii Skłodowskiej-Curie 9 St.,
41–800 Zabrze, Poland;
email: bjurkiewicz@sum.edu.pl

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ABSTRACT

Introduction: Despite reperfusion by primary percutaneous coronary intervention (PCI) in acute myocardial infarction (AMI) some patients develop left ventricular dysfunction. The extent of cardiac injury is associated with the inflammation and the reperfusion damage. An overly long inflammatory phase can cause sustained myocardial damage and improper healing, leading to remodelling and ventricular dilatation. We sought to elucidate whether proinflammatory proteins, cytokines and adhesion molecules that participate in the processes of inflammation, ischaemia-reperfusion injury and postmyocardial left ventricular remodelling are associated with the major adverse cardiovascular events (MACE) (all-cause mortality or AMI) during the two-year follow-up.

Material and methods: It is a prospective analysis of patients with AMI admitted to the cardiology ward who underwent PCI between December 2018 and April 2019. The Luminex Multiplex Assay was used to measure serum biomarker concentrations (Bio-Plex System 200, Bio-Rad, USA), and the data were analysed using Bio-Plex Manager Software.

Results: The median age was 66 (58–74), and 31.8% were women. The end-point was reached in 53 (37.9%) patients. Multivariate analysis found that serum pentraxin-3 [odds ratio (OR) 1.295 (1.175–1.443), $p < 0.001$] and E-selectin [OR 1.539 (1.151–2.265), $p = 0.03$] concentrations were associated with MACE.

Conclusions: Higher pentraxin and E-selectin serum concentrations are independently associated with all-cause mortality or myocardial infarction in the analysed population during the two-year follow-up.

Keywords: percutaneous coronary intervention, myocardial infarction, biomarkers, PTX-3 protein, E-selectin

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Introduction

Acute myocardial infarction (AMI) is defined as myocardial cell death due to prolonged ischaemia, leading to progressive deterioration of cardiac pump function [1]. The most common cause of AMI, referred to as type 1 myocardial infarction in the Fourth Universal Definition of Myocardial Infarction, is sudden coronary artery occlusion due to unstable atherosclerotic plaque rupture and thrombus formation [1]. Early and successful percutaneous coronary intervention (PCI) can effectively decrease the infarct size and improve clinical

outcomes but triggers an injury of the myocardium called ischaemia-reperfusion injury [2].

Endothelial dysfunction, immune activation and the inflammatory response play important roles in the induction and development of ischaemia-reperfusion injury [3]. Following AMI, the left ventricle (LV) undergoes a series of repair and wound-healing responses that include inflammation and scar formation; the combined constituents are collectively termed LV remodelling. The extent of LV remodelling after MI determines long-term outcomes, which include LV structure and function and survival rates.

Given the close relationship between revascularization, reperfusion, ischaemia–reperfusion injury, inflammation and cardiac remodelling during and after AMI, it was speculated that biomarkers of these processes, such as pentraxin-3, CD40 ligand, interleukin (IL)-10, IL-6, epidermal growth factor (EGF), fibroblast growth factor (FGF), E-selectin, cathepsin-S, endothelin-1, and Granulocyte-Macrophage Colony Stimulating Factor (GM-CSF), might be associated with outcomes in AMI patients. Pentraxin-3 and CD-40 ligands are sensitive and specific biomarkers for unstable and vulnerable plaques and reflect the extent of myocardial damage done by AMI [4, 5]. IL-10 is an anti-inflammatory cytokine that inhibits the release of proinflammatory mediators [6]. Il-6 suppresses innate immune signals to prevent the negative consequences of uncontrolled inflammation on cardiac geometry and function [7]. EGF is closely related to the proliferation of cardiac fibroblasts as an autocrine and paracrine factor [8]. FGF exerts cardioprotective effects in myocardial infarction and inhibits inflammation and myocardial fibrosis [9]. Elevation of E-selectin is considered a specific marker of endothelial activation [10]. Cathepsins regulate scar formation in the infarcted myocardium and participate in post-AMI remodelling [11]. Endothelin-1 is the vasoconstrictor synthesized by vascular endothelium in small-resistance coronary arteries; hence, enhanced release of Endothelin-1 from ischaemia–reperfusion–injured endothelium may result in intense and sustained microvascular constriction [12]. GM-CSF plays an important role in LV remodelling after AMI [13].

Therefore, in the following study, the authors sought to elucidate whether proinflammatory proteins, cytokines and adhesion molecules that participate in the process of inflammation, ischaemia-reperfusion injury and post-AMI left ventricular remodelling are associated with MACE (all-cause mortality or AMI) during the two-year follow-up. Furthermore, other factors associated with outcomes in the study group were analysed.

Material and methods

Patients with AMI admitted to the coronary care unit between December 2018 and April 2019 were included in the study if they met the following criteria: admission to the coronary care unit within 3 hours of the onset of chest pain; no history of previous AMI; acute myocardial injury with clinical evidence of acute myocardial ischaemia and with detection of a rise and/or fall of troponin values with at least 1 value above the 99th percentile of the upper reference limit; new ischaemic ECG changes in at least two leads; and no history or signs of acute or chronic inflammatory disorders. AMI was diagnosed based on clinical presentation, coronary angiography, and additional test results, in accordance with current European Society of Cardiology (ESC) guidelines [2]. Diagnostic and therapeutic strategies, including pharmacologic and interventional treatment, were used in accordance with the current ESC guidelines [2]. Each patient underwent coronary angiography. The data on long-term follow-up were obtained from the official registry of the National Health Fund, which ensured complete data collection. Follow-up data were available for all patients. Blood samples were taken immediately before PCI. The samples were immediately centrifuged, and the aliquoted plasma and serum were stored in microcentrifuge tubes at –80°C until assayed. The concentrations of biomarkers in serum were evaluated using the Magnetic Luminex Assay (R&D Systems, Minneapolis, MN, USA) according to the manufacturer's instructions. The assay was performed in a 96-well microplate. In brief, 50 μL serum/well was mixed with 50 μL/well of antibody-coupled beads, incubated and washed. Next, a 50 μL/well mixture of biotinylated secondary antibodies was added, and after incubation and washing, the antibodies were conjugated with streptavidin-phycoerythrin (50 μL/well). Washed and resuspended beads were read on a Luminex System (Bio-Plex System 200, Bio-Rad, USA), and the data were analysed using Bio-Plex Manager Software [14].

The study was approved by the institutional ethics committee (specific ethics code — KNW/0022/KB1/36/18, date of approval: 15 May 2018) and was conducted in accordance with the ethical standards laid down in the Declaration of Helsinki.

The statistical analysis was performed using SAS software, version 9.4 (SAS Institute Inc., Cary, NC, USA). Descriptive statistics are reported as mean ± standard deviation (SD) or median with upper and lower quartiles for continuous variables and as frequency (percentage) for categorical variables, as appropriate. Differences between the study groups were assessed using Student's t-test, the Mann–Whitney test or the χ^2 test, respectively.

An univariable logistic regression analysis was employed to select the potential end-point factors for inclusion in the multivariable analysis. The univariable end-point factors with $p \leq 0.05$ or less were entered into the multivariable logistic regression model by stepwise selection. The correlation between the explanatory variables was checked, and multicollinearity was evaluated by means of the tolerance and variance inflation factor. The results are presented as odds ratios (ORs) with their 95% confidence intervals (CIs). A p-value < 0.05 was considered statistically significant.

Results

The median age of the patients was 66 (58–74), and 31.8% of them were women. The baseline characteristics of the patients in the general population as well as the major adverse cardiovascular events (MACE) and non-MACE groups are presented in Table 1. The MACE during the two-year follow-up in 53 (37.9%) patients was observed. The multivariate logistic regression analysis confirmed that pentraxin and E-selectin serum

Table 1. Baseline characteristics of the study population

	General population (n = 148)	Group without MACE (n = 92)	Group with MACE (n = 56)	p
Age [years]	66.00 (58.00–74.00)	64.46 (11.58)	66.57 (11.73)	0.2853
Women, n [%]	47 (31.8)	31 (33.7)	16 (28.6)	0.5869
Hypertension, n [%]	126 (85.1)	79 (85.9)	47 (83.9)	0.7475
Type 2 diabetes, n [%]	63 (42.6)	39 (42.4)	24 (42.9)	0.9557
Hypercholesterolaemia, n [%]	107 (72.3)	63 (68.5)	44 (78.6)	0.1833
Leukocytes, $\times 10^9$/L	8.4 (7.2–10.9)	8.2 (7.0–10.6)	8.9 (7.4–11.9)	0.0368
Haemoglobin [mmol/L]	8.8 (8.2–9.5)	8.8 (8.2–9.6)	8.9 (7.8–9.4)	0.6105
Creatinine [μmol/L]	85.0 (73.0–98.0)	85.0 (73.0–95.0)	87.0 (73.0–107.0)	0.4536
Total bilirubin [μmol/L]	7.7 (5.4–11.1)	7.2 (5.3–10.2)	8.5 (6.0–12.4)	0.172
Uric acid [μmol/L]	344.0 (291.0–412.0)	337.0 (291.0–407.0)	352.0 (292.0–417.0)	0.3899
hs-CRP [mg/L]	2.4 (1.2–10.1)	2.4 (1.2–10.0)	2.3 (1.1–11.3)	0.5052
Procalcitonin [pg/mL]	0.2 (0.1–0.9)	0.4 (0.1–0.9)	0.2 (0.1–0.6)	1
Troponin [μg/L]	0.08 (0.02–0.35)	0.04 (0.02–0.15)	0.19 (0.04–0.59)	0.001
CK-MB	6.3 (3.2–20.03)	5.2 (2.9–12.0)	11.9 (4.0–42.2)	0.026
Cathepsin S [pg/mL]	4393.2 (3182.3–6281.7)	4386.4 (2827.0–5936.7)	4420.8 (3370.1–6685.1)	0.2956
EGF [pg/mL]	14.4 (7.2–33.7)	11.9 (4.9–18.5)	24.5 (10.9–56.7)	< 0.0001
FGF [pg/mL]	165.3 (133.7–210.4)	165.2 (131.4–205.3)	169.4 (133.7–228.0)	0.5005
IL-10 [pg/mL]	3.2 (1.6–4.9)	3.5 (1.4–4.7)	2.9 (1.7–6.3)	0.7287
Pentraxin 3 [pg/mL]	993.1 (642.5–1392.3)	779.8 (574.4–1097.0)	1570.6 (978.9–1968.9)	< 0.001
E-selectin [pg/mL]	22996.9 (18649.6–31457.8)	21835.2 (17934.7–27983.9)	26593.7 (19488.2–34537.3)	0.0126
CD40 Ligand [pg/mL]	2646.1 (1881.5–5453.6)	2264.5 (1661.5–4216.1)	3570.6 (2360.9–6505.1)	0.0004
Endothelin-1 [pg/mL]	11.1 (9.3–12.8)	10.9 (9.1–12.4)	11.4 (9.6–14.2)	0.2056
GM-CSF [pg/mL]	7.1 (4.3–11.6)	7.1 (4.3–14.9)	7.1 (4.1–9.2)	0.3933
IL-6 [pg/mL]	5.4 (3.1–11.6)	5.2 (3.6–11.0)	5.8 (3.1–13.6)	0.3933
LVEF [%]	48.0 (40.0–50.0)	48.0 (40.5–53.0)	45.0 (38.0–50.0)	0.0204

CK-MB — creatinine kinase myocardial band; hs-CRP — high sensitivity C-reactive protein; EGF — epidermal growth factor; FGF — fibroblast growth factor; GM-CSF — granulocyte-macrophage — colony-stimulating factor; Il-10 — interleukin-10; LVEF — left ventricle ejection fraction; MACE — major adverse cardiovascular events

Table 2. Univariable and multivariable analysis

Parameters	Univariable predictors		Multivariable predictors	
	OR	p	OR	p
Pentraxin 3	1.003 (1.002–1.004)	< 0.0001	1.295 (1.175–1.443)	< 0.0001
E-selectin	1.007 (1.005–1.008)	0.0053	1.539 (1.051–2.261)	0.03
EGF	1.018 (1.007–1.029)	0.0015		
CD40 Ligand	1.002 (1.001–1.003)	0.0372		
Endothelin-1	1.088 (1.013–1.169)	0.0213		
Leukocytes	1.127 (1.012–1.255)	0.0295		
Troponin	1.735 (1.123–2.680)	0.0130		
CK-MB	1.008 (1.001–1.016)	0.0241		

CK-MB — creatinine kinase myocardial band; EGF — epidermal growth factor; OR — odds ratio

concentrations were associated with MACE during the two-year follow-up. The univariable and multivariable logistic regression analyses are presented in Table 2.

Discussion

The present study demonstrated that pentraxin-3 and E-selectin serum concentrations were associated with two-year outcomes (death or AMI) in the analysed group of patients.

Several studies have shown that increased pentraxin-3 in serum was associated with worse outcomes of acute coronary syndrome (ACS). A meta-analysis of 8775 patients with ACS showed that elevated serum pentraxin-3 level was connected with an increased risk of mortality and cardiac events. Notably, the aforementioned association between higher pentraxin-3 and cardiac events in the meta-analysis was stronger among patients with STEMI than among patients with non-ST-elevation acute myocardial infarction (NSTEMI) [15]. It should also be emphasized that serum pentraxin-3 concentration was more closely associated with angiographic outcomes than serum C-reactive protein (CRP) [16]. A prognostic role of pentraxin-3 was also found in the Lipid Assessment Trial Italian Network (LATIN) study [17]. Among the biomarkers analysed in this study, only serum pentraxin-3 measured on the first day after AMI was independently associated with the mortality rate [17]. Measurement of pentraxin-3 levels may also be useful for evaluating stent-induced inflammation after PCI. Kotooka et al. [18] showed that patients with restenosis had higher pentraxin-3 than patients without restenosis.

Another independent predictor of outcomes in the present study population was the serum E-selectin concentration. E-selectin is a cell adhesion molecule that mediates leukocyte adhesion to vessel walls in response to inflammation. E-selectin enables rolling in monocytes, neutrophils, effector T cells, B cells and natural killer cells [19]. Recruiting these cells, especially monocytes, begins the formation of atherosclerotic plaques. E-selectin is expressed on the vascular endothelium and is responsible for the adhesion and transendothelial migration of circulating leukocytes. E-selectin is important and unique because it is expressed only by endothelial cells of the intima associated with atherosclerotic lesions. The circulating level of the soluble form of E-selectin reflects its endothelial expression and indicates the presence of systemic inflammation and endothelial activation [19, 20]. In the present study, E-selectin was tested as a prognostic biomarker because its expression by cultured endothelial cells and its release into the supernatant occurs after stimulation of endothelial cells by a variety of proinflammatory cytokines. Elevation of E-selectin is considered an apparently specific marker of endothelial activation and dysfunction [19–22]. Galvani et al. [23] showed that E-selectin was high in the serum of patients with clinically significant atherosclerosis, but they did not find a significant difference between patients with chronic coronary syndrome and ACS. Wenzel et al. [24] showed that the serine/arginine-128 polymorphism in E-selectin was associated with a greater risk for early severe atherosclerosis and was the reason for incorrect recognition of heparin. A role for E-selectin in atherosclerosis was supported by Hwang et al. [25]. They concluded that soluble E-selectin and ICAM-1 are

molecular markers of atherosclerosis and subclinical coronary heart disease. Weil et al. [26] have shown that the slow rolling of neutrophils due to E-selectin adhesion plays a significant role in the direct phase after myocardial infarction; this process allows neutrophils to phagocytize dead cardiomyocytes, remove the extracellular matrix and unleash the healing process.

Several limitations of this study should be noted. First, the study was done in a single centre and was therefore subject to selection bias. The second limitation was the relatively small number of patients, which calls for the results to be interpreted with caution. To overcome these limitations, multicentre studies with more patients are required to validate the clinical value of the analysed parameters and indices.

Conclusions

Higher pentraxin-3 and E-selectin serum concentrations are independently associated with all-cause mortality or myocardial infarction in the analysed population of patients during the two-year follow-up.

Article information

Data availability statement: *Consecutive patients with AMI admitted to the coronary care unit of III Department of Cardiology of Medical University of Silesia between December 2018 and April 2019 were included in the study if they met inclusion criteria. The data on long-term follow-up were obtained from the official registry of the National Health Fund, which ensured complete data collection. Follow-up data were available for all patients.*
Ethics statement: *The study was approved by the institutional ethics committee (specific ethics code — KNW/0022/KB1/36/18, date of approval: 15 May 2018) and was conducted in accordance with the ethical standards laid down in the Declaration of Helsinki.*
Author contributions: *WS — was involved in data collection, analyzed the data and was involved in drafting the manuscript; SK, MJ and PP — were involved in data collection, analyzed the data and were involved in drafting the manuscript; ZC — was involved in data analysis and drafting the manuscript; BS-J contributed to the study concept and design, data analysis and interpretation, drafting and revision of the manuscript and was responsible for the critical revision of the manuscript for intellectual content; MS — contributed to the data analysis and interpretation and performed statistical analysis; MG — was involved in data collection and was responsible for the critical revision of the manuscript for intellectual content. All authors approved the final version of the manuscript.*
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