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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The association of plasma fibrinogen with the extent and complexity of coronary lesions in patients with acute coronary syndrome

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Abstract

Background: High Syntax score (SXscore) is associated with more serious disease and worse prognosis in patients with acute coronary syndrome (ACS). Plasma fibrinogen levels are associated with poor cardiovascular outcomes.

Aim: To investigate the relation of admission fibrinogen levels with intermediate-high SXscore in patients with ACS.

Methods: A total of 752 patients (61.6 ± 12.8 years, 67.3% men) with ACS, who underwent urgent coronary angiography (CAG) were enrolled. Laboratory data including fibrinogen and high sensitivity C-reactive protein were obtained before CAG. Syntax scores of all patients were calculated from baseline CAG. The patients were divided into two groups: low SXscore (≤ 22) and intermediate-high SXscore (≥ 23).

Results: Admission fibrinogen levels were significantly higher in the SXscore ≥ 23 group when compared with the SXscore ≤ 22 group (median 492 mg/dL, interquartile range 428–581 mg/dL vs. median 370 mg/dL, interquartile range 309–428 mg/dL, respectively; $p < 0.001$). In multivariate analysis, the independent predictors of intermediate-high SXscore were fibrinogen (OR 1.008, 95% CI 1.005–1.010, $p < 0.001$), left ventricular ejection fraction (OR 0.935, $p < 0.001$), and age (OR 1.029, $p = 0.041$). A level of fibrinogen > 417 mg/dL had an 80.0% sensitivity and 71.3% specificity in predicting intermediate-high SXscore.

Conclusions: Increased fibrinogen levels are independently associated with intermediate-high SXscore in patients with ACS.

Key words: plasma fibrinogen, Syntax score, acute coronary syndrome

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INTRODUCTION

The Syntax score (SXscore), an angiographic classification system that aims to evaluate coronary anatomy regarding the extent and complexity of coronary lesions [1]. Higher SXscore are associated with more serious disease and worse prognosis in patients with acute coronary syndromes (ACS) [2, 3]. It is well established that underlying processes such as inflammation, enhanced coagulant activity, and endothelial dysfunction are closely related to the initiation and progression of atherosclerosis [4, 5].

Fibrinogen level is a readily measurable systemic inflammatory marker that is also an acute phase reactant, which is a response to acute exacerbation of chronic inflammation [6, 7]. Increased fibrinogen plasmatic levels are directly related with the risk of ACS [8]. It takes part in the atherosclerosis process inducing plaque growth, stimulating adhesion of platelets and white blood cells to the vessels wall cells, endothelial dysfunction, and promoting muscle cell proliferation and migration [9, 10]. Plasma fibrinogen levels have been found to be higher in patients with ACS compared to those of patients with stable coronary artery disease or healthy controls, and higher plasma fibrinogen levels may be predictors of poor long-term prognosis [11–14]. The association of the extent and complexity of atherosclerosis in ACS with fibrinogen levels has not been adequately studied.

Thus, we sought to investigate the relation of plasma fibrinogen levels with the extent and complexity of coronary

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artery disease assessed by SXscore in patients with ACS undergoing urgent coronary angiography (CAG).

METHODS

Patient population

The present study included patients with ACS, who underwent urgent CAG between January 2013 and May 2014 in our hospital.

We excluded individuals who had a history of coronary artery bypass grafting, in whom the SXscore could not be calculated. Patients with angina of secondary aetiology, active infection, or chronic inflammatory diseases, severe hepatic or renal dysfunction, all kinds of haematological disease including bleeding disorders, leukaemia, and history of malignancy and those on anticoagulation treatment were also excluded. Severe renal insufficiency was defined as pre-dialysed patients with an estimated glomerular filtration rate (eGFR) < 30 mL/min/1.73 m^2. The criteria for severe liver impairment were alanine-aminotransferase > 50 U/L, plasma albumin < 2.5 g/dL, and/or prothrombin time > 20 s.

ST-segment elevation myocardial infarction (STEMI) patients were defined as those presenting with ischaemic symptoms > 20 min with ST-segment elevation of > 2 mm in two contiguous precordial leads or > 1 mm in two or more contiguous leads, new (or presumably new) left bundle branch block, or true posterior myocardial infarction on admission electrocardiogram. Patients with unstable angina/non-ST-segment elevation myocardial infarction (NSTEACS) were required to have unstable ischaemic symptoms and electrocardiographic changes compatible with new ischaemia or increased cardiac biomarkers.

Hypercholesterolaemia was defined as a total cholesterol level ≥ 220 mg/dL or previous or ongoing treatment with lipid lowering agents. Arterial hypertension was defined when a patient was under active treatment with antihypertensive drugs or if the systolic blood pressure was ≥ 140 mm Hg or the diastolic blood pressure was ≥ 90 mm Hg on two or more separate occasions. Current smokers were those with regular smoking in the previous six months. The diagnosis of diabetes mellitus (DM) required active treatment with insulin or an oral hypoglycaemic agent, abnormal fasting blood glucose (≥ 126 mg/dL), blood glucose > 200 mg/dL at any time, or abnormal glucose tolerance according to World Health Organisation criteria. The eGFR values were calculated using the Modification of Diet in Renal Disease (MDRD) formula [15] and utilising the creatinine level measured on admission.

The study was approved by the Local Ethical Committee. All patients gave informed written consent.

Laboratory measurements

Baseline demographic information and laboratory data including complete blood count, complete lipid panel, high sensitivity C-reactive protein (hs-CRP) and fibrinogen, and cardiac markers of myocardial ischaemia/necrosis (creatine kinase-myocardial band [CK-MB], troponin T) were obtained in all patients. Plasma fibrinogen levels were measured using a Beckman Coulter ACL-TOP analyser (Beckman Coulter Life Sciences, Indianapolis, Indiana) with Instrumentation Laboratory reagents (Instrumentation Laboratory, Lexington, Massachusetts) from venous blood samples taken on admission (using citrate as the anticoagulant) immediately after first contact of patients with the hospital. Cardiac markers of ischaemic injury were measured every 8 h following CAG until hospital discharge or up to 24 h to assess the degree of myocardial injury.

Transthoracic echocardiography was performed for all patients (Vivid 3; GE Medical System, Horten, Norway) within 48 h of the hospitalisation. The left ventricular ejection fraction (LVEF) was determined using modified Simpson's method.

Coronary angiography and Syntax score

All patients were pretreated with aspirin, clopidogrel, and intravenous/subcutaneous heparin at the time of diagnosis after arrival at the emergency service. Diagnostic CAG was performed using standard techniques (Siemens Axiom Artis zee 2011; Siemens Healthcare, Erlangen, Germany). All of the interventions were performed with standard femoral approach with a 6-French guiding catheter (Launcher; Medtronic, Minneapolis, Minnesota, USA). Multivessel disease (MVD) was defined as the presence of a stenosis greater than 50% in three major epicardial coronary arteries based on CAG. Preprocedural SXscore for each patient was calculated by a team of two interventional cardiologists. All coronary lesions that produced a luminal narrowing ≥ 50% in vessels ≥ 1.5 mm were separately scored using the SXscore calculator, and then summed to provide an overall SXscore. The online latest updated version (2.11) was used for the calculation of the SXscore (http://www.syntaxscore.com) [1]. A low SXscore was defined as ≤ 22, intermediate as 23–32, and high as ≥ 33. The patients were divided into two groups: those with low SXscore (≤ 22), and those with intermediate-high SXscore (≥ 23).

Statistical analysis

Categorical variables are presented as counts and percentages, whereas continuous variables are presented as mean ± standard deviation (SD) or as median and interquartile ranges (IQR) depending on the normality of distribution assessed with the use of a Kolmogorov–Smirnov test. Comparison of parametric values between the two groups was performed by means of independent samples t test. Comparisons of nonparametric values between the two groups were performed by Mann–Whitney U test. Categorical variables were compared by the χ^2 test. Spearman's rank test was used for correlation between fibrinogen and hs-CRP levels. In addition, we used multiple regression and logistic regression to observe the association between intermediate-high SXscore and the impact of the principal

Table 1. Baseline demographics and prior medications of the study population

Characteristic	Syntax score		P
	≤ 22 (n = 551)	≥ 23 (n = 205)	
Age [years]	60 ± 12	66 ± 12	< 0.001
Women	167 (30.3%)	80 (39%)	0.023
Body mass index [kg/m^2]	28.2 ± 4.7	27.2 ± 4.5	0.015
Hypertension	243 (44.1%)	92 (44.9%)	0.848
Diabetes mellitus	150 (27.2%)	90 (43.9%)	< 0.001
Current smoker	261 (47.4%)	65 (31.7%)	< 0.001
Hypercholesterolaemia	327 (59.3%)	116 (56.6%)	0.493
Family history of coronary artery disease	158 (28.7%)	57 (27.8%)	0.813
Prior myocardial infarction	36 (6.5%)	21 (10.2%)	0.086
Prior stroke	12 (2.2%)	9 (4.4%)	0.100
Systolic blood pressure [mm Hg]	132 ± 25	129 ± 24	0.136
Diastolic blood pressure [mm Hg]	79 ± 14	78 ± 14	0.220
Heart rate [bpm]	78 ± 14	84 ± 15	< 0.001
Clinical presentation:			0.319
ST-elevation myocardial infarction	267 (48.5%)	91 (44.4%)	
Non-ST-elevation acute coronary syndrome	284 (51.5%)	114 (55.6%)	
Time between pain onset and first contact [min]	165 (120–270)	180 (120–240)	0.610
Left ventricular ejection fraction [%]	51 ± 10	44 ± 11	< 0.001
Previous medications:			
Aspirin	102 (18.5%)	28 (13.6%)	0.305
Clopidogrel	24 (4.4%)	12 (5.7%)	0.534
Beta-blocker	74 (13.4%)	30 (14.8%)	0.758
Renin-angiotensin-aldosterone antagonists	140 (25.5%)	52 (25.3%)	0.975
Statin	61 (11.1%)	16 (7.9%)	0.158

variable (fibrinogen) and of other variables that probably acted as confounders (age, gender, body mass index [BMI], current smoker, DM, LVEF, heart rate, haemoglobin, eGFR, glucose, neutrophil-to-lymphocyte ratio (NLR), mean platelet volume (MPV), hs-CRP, and ferritin). Receiver-operating characteristic (ROC) curve analyse was performed to determine the best fibrinogen cutoff value for the prediction of intermediate-high SXscore while maximising sensitivity and specificity. Two-tailed p values were considered as significant at < 0.05. All statistical analyses were performed using SPSS V 18.0 for Windows (version 18.0, SPSS, Chicago, Illinois).

RESULTS

A total of 756 ACS patients were enrolled in our study. There were 551 patients (mean age 60 ± 12 years, 30.3% women) in the low SXscore (≤ 22) group and 205 patients (mean age 66 ± 12 years, 39% women) in the intermediate-high SXscore (≥ 23) group.

Baseline clinical characteristics of patients are listed in Table 1. Intermediate-high SXscore was significantly associated with older age, female gender, DM, heart rate and inversely associated with BMI, current smoking, and LVEF. Prior medications were similar between the groups.

Baseline laboratory and angiographic characteristics of patients in relation to SXscore are presented in Table 2. Patients with SXscore ≥ 23 had increased MPV, NLR, glucose, creatinine, ferritin, and hs-CRP, and decreased haemoglobin and eGFR at admission. Peak troponin T levels were also higher in this group. The plasma levels of fibrinogen were higher in the patients with SXscore ≥ 23 (median 492 mg/dL, IQR 428–581) than in the patients with SXscore ≤ 22 (371 mg/dL, IQR 309–428; $p < 0.001$) (Table 2, Fig. 1). In addition, it was determined that there was a correlation between fibrinogen and hs CRP levels in the whole study population ($r = 0.54$, $p < 0.001$). In subgroups according to SXscore as low and intermediate-high SXscore there were also correlations between these two parameters ($r = 0.48$, $r = 0.39$, $p < 0.001$, respectively). Culprit lesions in patients with SXscore ≥ 23 were more likely to be located in the left anterior descending coronary artery, whereas in patients with SXscore ≤ 22 they were more

Table 2. Laboratory measurements and angiographic findings of the study population

Characteristic	Syntax score		P
	≤ 22 (n = 551)	≥ 23 (n = 205)	
White blood cell count [×10⁹/L]	10.3 ± 3.3	10.8 ± 3.8	0.071
Platelet count [×10⁹/L]	236 ± 67	245 ± 74	0.138
Mean platelet volume [fL]	8.67 ± 1.04	8.93 ± 1.01	0.002
Neutrophil-to-lymphocyte ratio	2.86 (1.77–4.92)	4.21 (2.59–6.90)	< 0.001
Haemoglobin [g/dL]	14.2 ± 1.8	13.3 ± 1.9	< 0.001
Glucose [mg/dL]	117 (96–156)	148 (109–214)	< 0.001
Creatinine [mg/dL]	1.07 ± 0.30	1.15 ± 0.32	0.001
Estimated glomerular filtration rate [mL/min/1.73 m²]	74 ± 20	64 ± 20	< 0.001
Alanine-aminotransferase [U/dL]	23 (17–37)	24 (16–37)	0.840
Fibrinogen [mg/dL]	370 (309–428)	492 (428–581)	< 0.001
Ferritin [ng/mL]	79 (44–134)	105 (51–196)	0.001
Peak troponin-T [ng/mL]	565 (159–1715)	1182 (338–4852)	< 0.001
Total cholesterol [mg/dL]	195 ± 48	180 ± 50	0.226
High-density lipoprotein cholesterol [mg/dL]	41 ± 10	40 ± 10	0.508
Low-density lipoprotein cholesterol [mg/dL]	124 ± 41	120 ± 41	0.217
Triglyceride [mg/dL]	137 (91–191)	133 (91–193)	0.517
High-sensitivity C-reactive protein [mg/dL]	6.02 ± 2.84	8.87 ± 2.99	< 0.001
Anterior myocardial infarction	97 (17.6%)	56 (27.3%)	0.003
Number of diseased vessel	1.59 ± 0.72	2.55 ± 0.67	< 0.001
Multivessel disease	251 (45.6%)	184 (89.8%)	< 0.001
Number of narrowed coronary arteries:			< 0.001
1	303 (55%)	21 (10.2%)	
2	171 (31%)	51 (24.9%)	
3	77 (14%)	133 (64.9%)	
Acute coronary syndrome-related artery:			0.001
Left main	3 (0.5%)	6 (2.9%)	
Left anterior descending	243 (44.1%)	112 (54.6%)	
Left circumflex	141 (25.6%)	42 (20.5%)	
Right	164 (29.8%)	45 (22%)	
Chronic total occlusion	50 (9.1%)	88 (42.9%)	< 0.001
Procedure:			< 0.001
Balloon angioplasty	17 (3.1%)	8 (3.9%)	
Balloon angioplasty with stent implantation	158 (28.7%)	73 (35.6%)	
Direct stenting	329 (59.7%)	45 (22%)	
Coronary artery bypass grafting surgery	31 (5.6%)	71 (34.6%)	

commonly located in the left circumflex coronary artery and the right coronary artery. The number of diseased vessels was significantly higher in patients with intermediate-high SXscore compared with the low SXscore (2.55 ± 0.67 vs. 1.59 ± 0.72, $p < 0.001$). MVD, anterior STEMI, and chronic total occlusion were also more often in patients with SXscore ≥ 23. The rate of patients who underwent stent implantation was higher in the SXscore ≤ 22 group, while patients who underwent coronary artery bypass grafting surgery was higher in the SXscore ≥ 23 group.

Univariate and multivariate logistic regression analysis of the association between the multiple characteristics and intermediate-high SXscore are shown in Table 3. The univariate screen-identified significant predictors were assessed by multivariate analysis. In multivariate analysis, the independent predictors of intermediate-high SXscore were fi-

brinogen (odds ratio [OR] 1.008, 95% confidence interval [CI] 1.005–1.010, p < 0.001) as well as LVEF (OR 0.935, 95% CI 0.912–0.959, p < 0.001) and age (OR 1.029, 95% CI 1.001–1.058, p = 0.041).

The ROC curve analysis showed that fibrinogen predicted intermediate-high SXscore with an area under the curve of 0.812 (95% CI 0.778–0.846) (Fig. 2). The best cutoff value of fibrinogen for the prediction of intermediate-high SXscore while maximising sensitivity (80.0%) and specificity (71.3%) was 417 mg/dL. Using this value, patients were divided into two groups: those with fibrinogen > 417 mg/dL (n = 434) and those with fibrinogen ≤ 417 mg/dL (n = 322). Patients with a fibrinogen level > 417 mg/dL were significantly older with higher prevalence of female gender, hypertension, DM, but lower frequency of current smoking and lower LVEF

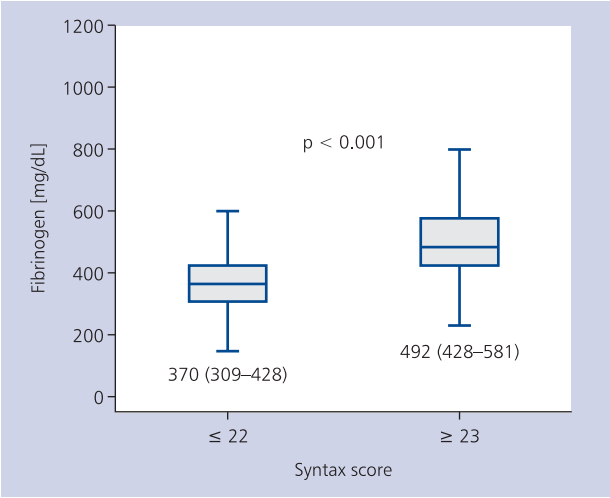

Figure 1. Comparison of plasma fibrinogen levels between groups

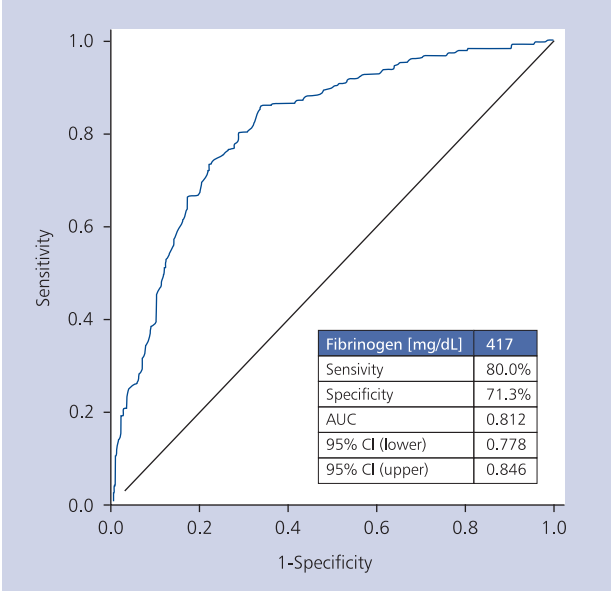

Figure 2. Receiver operating characteristic curve analysis of plasma fibrinogen data for intermediate-high Syntax score

Table 3. Univariate and multivariate predictors of Syntax score ≥ 23 in patients with acute coronary syndrome

Characteristic	Univariate analysis		Multivariate analysis	
	Odds ratio, 95% confidence interval	P	Odds ratio, 95% confidence interval	P
Age	1.040 (1.026–1.054)	< 0.001	1.029 (1.001–1.058)	0.041
Women	1.472 (1.053–2.056)	0.024	1.426 (0.768–2.645	0.261
Body mass index	0.953 (0.917–0.991)	0.016	0.992 (0.942–1.046)	0.777
Current smoker	0.515 (0.367–0.723)	< 0.001	0.738 (0.387–1.407)	0.356
Diabetes mellitus	2.092 (1.499–2.923)	< 0.001	1.040 (0.558–1.939)	0.901
Left ventricular ejection fraction	0.933 (0.917–0.950)	< 0.001	0.935 (0.912–0.959)	< 0.001
Heart rate	1.027 (1.016–1.038)	< 0.001	1.001 (0.985–1.017)	0.921
Haemoglobin	0.773 (0.708–0.843)	< 0.001	0.886 (0.760–1.033)	0.122
White blood cell	1.042 (0.996–1.090)	0.072		
Estimated glomerular filtration rate	0.977 (0.969–0.985)	< 0.001	0.995 (0.980–1.010)	0.516
Glucose	1.005 (1.003–1.007)	< 0.001	1.002 (0.999–1.006)	0.223
Total cholesterol	0.998 (0.995–1.001)	0.261		
Neutrophil-to-lymphocyte ratio	1.064 (1.027–1.102)	0.001	1.049 (0.986–1.116)	0.129
Mean platelet volume	1.268 (1.090–1.475)	0.002	1.202 (0.952–1.518)	0.122
High-sensitivity C-reactive protein	1.253 (1.188–1.322)	< 0.001	1.035 (0.956–1.120)	0.399
Fibrinogen	1.010 (1.008–1.012)	< 0.001	1.008 (1.005–1.010)	< 0.001
Ferritin	1.003 (1.001–1.004)	< 0.001	1.001 (1.000–1.003)	0.109

Table 4. Clinical characteristics of the study patients according to fibrinogen cutoff level

Characteristic	Fibrinogen [mg/dL]		P
	≤ 417 (n = 434)	> 417 (n = 322)	
Age [years]	59 ± 12	66 ± 12	< 0.001
Women	109 (25.1%)	138 (42.9%)	< 0.001
Body mass index	28.1 ± 4.46	27.8 ± 5.0	0.430
Hypertension	174 (40.1%)	161 (50%)	0.007
Diabetes mellitus	107 (24.7%)	133 (41.3%)	< 0.001
Current smoker	217 (50%)	109 (33.9%)	< 0.001
Hypercholesterolemia	260 (59.9%)	183 (56.8%)	0.396
Left ventricular ejection fraction [%]	50 ± 10	47 ± 10	0.001
Serum glucose [mg/dL]	139 ± 72	165 ± 85	< 0.001
Serum creatinine [mg/dL]	1.05 ± 0.26	1.15 ± 0.35	< 0.001
Estimated glomerular filtration rate [mL/min/1.73 m^2]	76 ± 19	64 ± 20	< 0.001
Haemoglobin [g/dL]	14.3 ± 1.7	13.4 ± 1.9	< 0.001
Platelet count	230 ± 60	250 ± 79	< 0.001
Neutrophil-to-lymphocyte ratio	2.80 (1.71–4.67)	3.79 (2.32–6.44)	< 0.001
High-sensitivity C-reactive protein [mg/dL]	5.11 ± 3.64	9.03 ± 2.84	< 0.001
Multivessel disease	200 (46.1%)	235 (73%)	< 0.001
Chronic total occlusion	48 (11.1%)	90 (28%)	< 0.001
Syntax score	13 ± 7	20 ± 10	< 0.001
In-hospital mortality	12 (2.8%)	21 (6.5%)	0.012

compared with patients with fibrinogen levels ≤ 417 mg/dL. Additionally, platelet count, NLR, creatinine, glucose, and hs-CRP levels and SXscore were significantly higher, but haemoglobin and eGFR values were significantly lower in the fibrinogen > 417 mg/dL group. Chronic total occlusion and MVD were more frequent in the higher level of fibrinogen group. In-hospital mortality was higher in the higher fibrinogen group compared with lower fibrinogen group (21 [6.5%] vs. 12 [2.8%], respectively, p = 0.012) (Table 4).

DISCUSSION

The main finding of our study is that plasma fibrinogen levels on admission are independently associated with the extent and complexity of coronary atherosclerosis in ACS. An increased level of fibrinogen predicts intermediate-high SXscore in these patients.

The SXscore is widely accepted as an independent prognostic marker in patients with ACS [2, 3]. Palmerini et al. [2] showed that the SXscore is an independent predictor of the one-year rates of death, cardiac death, myocardial infarction, and target vessel revascularisation in patients with NSTEACS undergoing percutaneous coronary intervention. Magro et al. [3] demonstrated that the SXscore during primary percutaneous coronary intervention is a useful tool that provides additional risk stratification to known risk factors of long-term mortality and major adverse cardiovascular events in patients with STEMI.

Fibrinogen is a coagulation/inflammation biomarker, which is strongly associated with atherogenesis [6]. An array of mechanisms by which fibrinogen may promote atherosclerosis and/or atherothrombosis [16, 17]. Several studies have shown that fibrinogen levels are related to major adverse cardiovascular events [18, 19]. Elevated fibrinogen levels are directly related with the risk of ACS [8]. Plasma fibrinogen levels have been found to be higher in patients with ACS, and higher plasma fibrinogen levels may be predictors of poor prognosis [12]. Similarly, our study suggested that in-hospital mortality was higher in patients with higher (> 417 mg/dL) fibrinogen levels when compared to those with lower (≤ 417 mg/dL) fibrinogen levels in our study population.

In our study we found that higher admission fibrinogen levels were associated with the extent and complexity of coronary atherosclerosis in patients who underwent CAG for ACS. The exact mechanisms for the coagulation-dependent mechanisms of atherogenesis are still obscure. It is well known that the pathogenesis of atherosclerosis involves a number of local inflammatory mechanisms, including endothelial dysfunction, leukocyte migration, extracellular matrix degradation, and platelet activation [5], and these mechanisms mediate many of the stages of the development of the atheromatous plaque, from initial leukocyte recruitment to eventual rupture of the unstable atherosclerotic plaque [20]. Fibrinogen may play an important role in all stages of these

events. Fibrinogen is a marker for inflammation because it is induced by interleukin-6, a cytokine that also induces CRP [7]. In our study, we found that well known inflammatory markers that reflect vascular inflammation such as fibrinogen, hs-CRP and NLR levels were higher in patients with intermediate-high SXscore. In addition, fibrinogen contributes to blood viscosity, platelet aggregation, and fibrin formation and modulates subsequent coagulation activation [21]. Fibrinogen has been shown to promote smooth muscle cell proliferation and induce monocyte chemotaxis [22]. Smooth muscle cells and monocyte/macrophages are the major cellular components of the atheromatous plaques associated with coronary atherosclerosis. Moreover, fibrinogen can bind to endothelial cell receptors and trigger the release of vasoactive mediators [23]. Fibrinogen and its degradation products modulate endothelial cell permeability and promote endothelial cell migration. As a result of these interreactions, fibrinogen leads to endothelial dysfunction [24]. It is well known that impaired endothelial function represents a major factor for atherogenesis. Furthermore, there is evidence that hyperfibrinogenaemia predisposes to the formation of denser fibrin clots displaying impaired fibrinolysis, which might enhance atherosclerosis [25]. So, the elevated fibrinogen levels in our study population consisting of ACS may the result of acute complication of vascular disease by serious events including acute thrombosis and enhanced coagulant activity because of impaired fibrinolysis. Consequently, hyperfibrinogenaemia could be a result of more complex coronary atherosclerosis.

Limitations of the study

Our study has several limitations. First, the assessment of CAG findings was limited to the visual interpretation, and CAG detects only major coronary arterial lesions. Second, fibrinogen was based on a single measurement. It would be interesting to know if the fibrinogen changes over time and if the fibrinogen in the subsequent tests is still a predictor of extent and complexity of coronary disease. Finally, several disorders could increase fibrinogen levels, so the impact of higher fibrinogen levels may be recommended as a minor risk factor for atherosclerosis.

CONCLUSIONS

In conclusion, plasma fibrinogen levels are significantly associated with the extent and complexity of coronary atherosclerosis in patients with ACS. A higher fibrinogen level is an independent predictor of intermediate-high SXscore.

Conflict of interest: *none declared*

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Zależność między stężeniem fibrynogenu w osoczu a zaawansowaniem i rozległością zmian w naczyniach wieńcowych u pacjentów z ostrym zespołem wieńcowym

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Streszczenie

Wstęp: Wysoka punktacja w skali Syntax (SXscore) wiąże się z bardziej zaawansowaną chorobą i gorszym rokowaniem u pacjentów z ostrym zespołem wieńcowym (ACS). Większe stężenie fibrynogenu koreluje ze zwiększonym ryzykiem zdarzeń sercowo-naczyniowych.

Cel: Celem pracy było zbadanie zależności między stężeniem fibrynogenu przy przyjęciu do szpitala a pośrednimi i wysokimi wartościami SXscore u pacjentów z ACS.

Metody: Do badania włączono ogółem 752 chorych (61,6 ± 12,8 roku; 67,3% mężczyzn) z ACS, u których wykonano w trybie pilnym koronarografię (CAG). Przed CAG przeprowadzono badania laboratoryjne, w tym zmierzono stężenia fibrynogenu i białka C-reaktywnego oznaczonego metodą wysokoczułą. U wszystkich chorych obliczono SXscore na podstawie wyjściowej CAG. Chorych podzielono na dwie grupy: osoby z niską punktacją SXscore (≤ 22) i osoby z pośrednią/wysoką punktacją SXscore (≥ 23).

Wyniki: Stężenie fibrynogenu przy przyjęciu do szpitala było istotnie wyższe w grupie z punktacją SXscore ≥ 23 niż w grupie, w której punktacja SXscore wynosiła ≤ 22 (odpowiednio: mediana 492 mg/dl, zakres międzykwartylowy 428–581 mg/dl vs. mediana 370 mg/dl, zakres międzykwartylowy 309–428 mg/dl; $p < 0,001$). W analizie wieloczynnikowej niezależnymi czynnikami predykcyjnymi pośredniej/wysokiej punktacji SXscore były: stężenie fibrynogenu (OR 1,008; 95% CI 1,005–1,010; $p < 0,001$), frakcja wyrzutowa lewej komory (OR 0,935; $p < 0,001$) i wiek (OR 1,029; $p = 0,041$). Stężenie fibrynogenu > 417 mg/dl pozwalało prognozować pośrednią/wysoką punktację SXscore z czułością wynoszącą 80,0% i swoistością równą 71,3%.

Wnioski: Zwiększone stężenia fibrynogenu są niezależnie związane z pośrednimi/wysokimi wartościami SXscore u pacjentów z ACS.

Słowa kluczowe: stężenie fibrynogenu w osoczu, skala Syntax, ostry zespół wieńcowy

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