IgG, IgM and inflammatory markers serum concentration in patients with acute coronary syndrome: a pilot study

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Abstract

Background: A number of biomarkers have been found that might help to predict the risk of acute coronary syndrome (ACS) in adults.

Aim: To analyse the association between immunoglobulins concentration and other inflammatory markers such as C-reactive protein (CRP) and fibrinogen that show correlation with the risk of ACS.

Methods: The study population consists of 52 consecutive patients with ST segment elevation myocardial infarction (STEMI) or unstable angina/non-STEMI. Concentrations of total protein, albumin, alpha-1 globulin, beta globulin, gamma protein, immunoglobulin in class A (IgA), G (IgG), M (IgM) and E (IgE), creatinine kinase (CK), creatinine kinase MB (CK-MB), CRP and fibrinogen were quantified.

Results: In the ACS patients, there was a significant increase in gamma globulin, CRP and fibrinogen. IgG was elevated only in the STEMI group and correlated with fibrinogen (R = 0.48, p < 0.01).

Conclusions: 1. IgG appears to be the only immunoglobulin associated with ACS in the STEMI group. 2. Fibrinogen reveals features of a reactive biomarker of ACS. 3. CRP appears to be closely related to the causative process in coronary artery disease patients.

Key words: acute coronary syndrome, markers, immunoglobulin, IgG

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INTRODUCTION

Although a significant decrease in cardiac mortality has been reported in industrialised countries, acute coronary syndrome (ACS) still remains the major cause of death in adults [1, 2]. Among the risk factors predisposing to ACS are a number of biomarkers reflecting the inflammatory process, e.g. leukocytosis [3], elevated C-reactive protein (CRP) [4], high-sensitivity C-reactive protein (hs-CRP) [5], lipoprotein-associated phospholipase A2 [6], interleukin-2 [7], interleukin-6, tumour necrosis factor [8] and procalcitonin [9]. An association of biomarkers with different patterns of coronary atherosclerosis, as quantified by coronary computed tomography angiography or optical coherence tomography, has been recently

noted [10, 11]. Likewise, there are antibodies that seem to play an important role in the acute phase of ACS [12, 13]. However, there has been limited research published describing early change in the immunoglobulin (Ig) profile in ACS patients [14]. Therefore, we designed the present study to examine the differences of Ig dynamics between distinct types of ACS and to establish the relations between Ig and inflammatory markers in the course of ACS.

METHODS

We observed 52 consecutive patients who presented with symptoms suggesting ACS, defined as chest pain and dynamic changes in electrocardiogram (ECG) or elevated tropo-

nin I (TnI), and who were aged under 60. Otherwise, there were no specific exclusion criteria. The patients were included over a period of three months. All patients were admitted to the cardiology ward and treated according to the European Society of Cardiology Guidelines valid at the time of the study [15]. Based on the clinical presentation, ECG changes, and changes in the cardiac markers, two types of ACS groups were distinguished, namely myocardial infarction (MI) with ST segment elevation (STEMI), and unstable angina or MI with no ST segment elevation (UA/NSTEMI).

As per study protocol, an informed consent was obtained from each patient before the first sample of 5 mL of blood was taken on admission to the ward (sample 1). Consecutive samples were drawn between days 10 and 12 (sample 2) and three months following hospitalisation (sample 3). These time periods were chosen mainly to assess the short-term (1–2 weeks) and longer-term (three months) dynamics. The serum was separated and stored frozen at –72°C for laboratory concentration measurements of total protein, albumin, alpha1 globulin, beta globulin, gamma protein, immunoglobulin in class A (IgA), G (IgG), M (IgM) and E (IgE). Additionally, creatinine kinase (CK), creatinine kinase MB (CK-MB), CRP and fibrinogen levels were quantified [Roche Diagnostics assay].

Statistical analysis was performed using SPSS software and included t-Student's test and non-parametric tests i.e. U-Mann-Whitney, Kolmogorov-Smirnov for independent samples and Wilcoxon for dependent samples. Statistical significance was assumed for p value of < 0.05. The results are presented as means ± SD.

A quantitative measurement of CRP serum concentrations was performed only for concentrations > 3 mg/dL. Statistical analysis of samples with concentration < 3 mg/dL assumed a continuous uniform distribution in this study group.

RESULTS
Population characteristics
The mean age of the study population was 51.1 years. There were 34 cases of confirmed STEMI, 12 cases of NSTEMI and six cases of UA. The latter two subgroups were combined and analysed as one UA/NSTEMI subgroup. There were more females (38.9% vs. 14.7%, p = 0.043) and more cases of hypertension (72.2% vs. 38.2%, p = 0.047) in the UA/NSTEMI group. In the STEMI group, more people smoked (82.3% vs. 61.1%, p = 0.049). The markers of heart ischaemia (CK and CK-MB) were higher in the STEMI group. Apart from the above, the groups did not differ in terms of cardiovascular risk factors i.e. previous MI, diabetes, hyperlipidaemia or family history of cardiac disease (Table 1). There were no chronic inflammatory or neoplastic diseases recorded in the study population. In terms of antihypertensive treatment, the patients received beta-blockers, diuretics and angiotensin converting enzyme (ACE) inhibitors. There were no differences between the groups regarding treatment mode.

Table 1. Baseline characteristics of the patients included in the study — comparison in respect of the type of acute coronary syndrome

	STEMI (n = 34)	UA/NSTEMI (n = 18)	P
Age [years]	51.23 ± 4.81	51 ± 6.12	NS
Gender (female)	5 (14.7%)	7 (38.9%)	0.043
Previous MI	5 (14.7%)	3 (16.7%)	NS
Hypertension	13 (38.2%)	13 (72.2%)	0.047
Diabetes type 2	5 (14.7%)	2 (11.1%)	NS
Active smoker	28 (82.3%)	11 (61.1%)	0.049
Hyperlipidaemia	21 (61.7%)	10 (55.5%)	NS
Family history of IHD/SCD	10 (29.4%)	9 (50%)	NS
CK max	1,919.79	451.67	< 0.001
CK-MB max	146	33	< 0.001

STEMI — ST segment elevation myocardial infarction; UA — unstable angina; NSTEMI — non-ST segment elevation myocardial infarction; MI — myocardial infarction; IHD — ischaemic heart disease; SCD — sudden cardiac death; CK — creatinine kinase; CK-MB — creatinine kinase MB

In terms of pharmacological treatment, there were no differences between the two groups. Acetylsalicylic acid, beta-adrenolytic, ACE inhibitors, statins and nitrates were administered to selected patients. All patients received a low molecular heparin, either enoxaparin or nadroparin. All STEMI patients were treated with fibrinolysis (streptokinase) — the standard treatment at the time of the study.

Protein electrophoresis
We found significant differences between the STEMI and UA/NSTEMI groups in the serum concentrations of immunoglobulins and in protein electrophoresis on admission. IgM, IgG, total protein and gamma protein were significantly elevated in the UA/NSTEMI group in the acute phase of ACS (sample 1). No significant differences were found in regard to any other proteins between the groups (Table 2).

The concentrations of IgG, total protein and gamma protein, but not IgM, rose significantly in the STEMI group between admission and the second week (Fig. 1). Among the UA/NSTEMI patients, only gamma globulin increased significantly in the corresponding period.

Long term follow-up (second week vs. third month) showed a significant elevation of the serum concentration of total protein and albumins in the STEMI group. No variations were found in the UA/NSTEMI group (Table 3).

Inflammatory markers
As measured on admission, the serum concentration of CRP in the acute phase of MI was elevated in all ACS patients. It was significantly higher in the STEMI than in the UA/NSTEMI group (44.24 ± 46.10 vs. 20.91 ± 28.81, p = 0.032). After the first two weeks, it decreased significantly in both groups

Table 2. On admission: laboratory results of immunoglobulin classes and protein electrophoresis in STEMI and UA/NSTEMI patients (mean ± SD)

	STEMI (n = 34)	UA/NSTEMI (n = 18)	P
Total protein [g/dL]	6.29 ± 0.49	6.67 ± 0.49	0.007
Albumins [g/dL]	3.85 ± 0.31	2.97 ± 0.98	NS
Globulins [g/dL]:			
Fraction α-1-	0.25 ± 0.07	0.23 ± 0.06	NS
Fraction α-2-	0.61 ± 0.17	0.64 ± 0.17	NS
Fraction β-	0.84 ± 0.15	0.92 ± 0.23	NS
Fraction χ-	0.74 ± 0.16	0.91 ± 0.24	0.003
IgA [mg/dL]	223.87 ± 83.7	245.07 ± 95.19	NS
IgG [mg/dL]	862.07 ± 166.15	1016.87 ± 205.04	0.005
IgM [mg/dL]	93.97 ± 48.85	148.2 ± 132.99	0.026
IgE [IU]	59.57 ± 49.48	79.79 ± 76.23	NS
CRP [mg/dL]	44.24 ± 46.10	20.91 ± 28.81	0.032

STEMI — ST segment elevation myocardial infarction; UA — unstable angina; NSTEMI — non-ST segment elevation myocardial infarction; IgA — immunoglobulin A; IgG — immunoglobulin G; IgM — immunoglobulin M; CRP — C-reactive protein; NS — not significant (p ≥ 0.05)
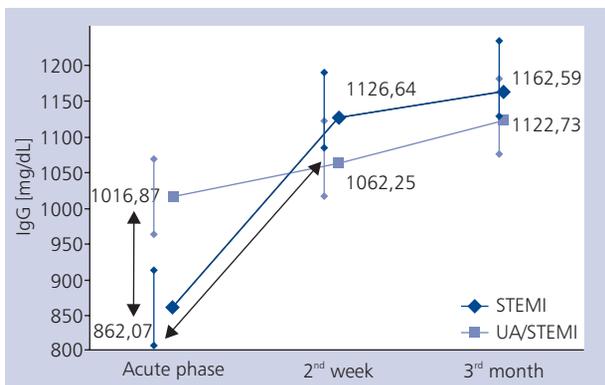

Figure 1. Change in immunoglobulin G (IgG) mean serum concentration in ST segment elevation myocardial infarction (STEMI) and unstable angina or non-ST segment elevation myocardial infarction (UA/NSTEMI) patients over three month follow-up

Figure 2. Change in the concentration of C-reactive protein (CRP) in ST segment elevation myocardial infarction (STEMI) and unstable angina or non-ST segment elevation myocardial infarction (UA/NSTEMI) patients over three month follow-up
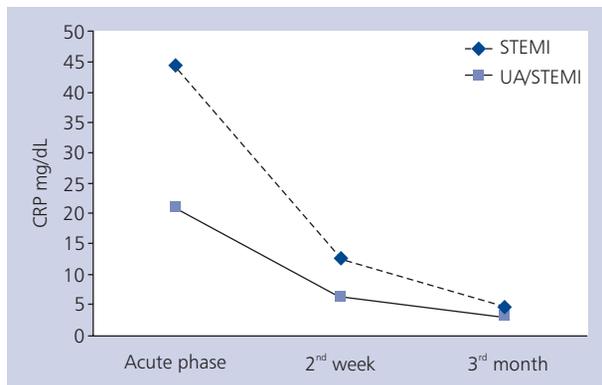
and levelled off at comparable concentrations (12.53 ± 20.86 vs. 6.30 ± 7.53, p ≥ 0.05). The concentrations remained similar in the third month between the two groups (4.48 ± ± 7.91 vs. 3.01 ± 5.57, p ≥ 0.05) (Fig. 2, Table 3).

In both groups, serum concentration of fibrinogen increased significantly in the first two weeks, reaching its peak on day 14. It had declined to its starting point by the third month. Fibrinogen concentrations tended to be higher among STEMI patients compared to other ACS patients; however, the difference was statistically significant only for measurements evaluated in week 2 (sample 2) (Table 3).

The serum concentration of fibrinogen in the third month after ACS correlated with the serum concentration of IgG (R = 0.48, p < 0.01). Correlation analysis of other immunoglobulins with inflammatory markers did not show any significance.

DISCUSSION

Our findings confirmed differences in the protein electrophoresis profile between STEMI and UA/NSTEMI patients in the acute phase of myocardial ischaemia. We found an increased serum concentration of IgM, IgG gamma globulin and total protein in the UA/NSTEMI group on admission. These values, except for gamma globulin, remained raised through the entire period of the three month follow-up. On the other hand, we found that in the STEMI group there was a marked increase of IgG, gamma globulin and total protein measured in the second week that remained elevated until three months following the onset of ACS. Additionally, we demonstrated an increase of albumins three months post ACS.

These findings suggest a diverse underlying pathogenesis of ACS between STEMI and UA/NSTEMI patients. We could assume that an average UA/NSTEMI patient would have

Table 3. Second week: laboratory results of immunoglobulin classes and protein electrophoresis in STEMI and UA/NSTEMI patients (mean ± SD)

	On admission (sample 1)	Second week (sample 2)	Third month (sample 3)	P (sample 1 vs. 2)	P (sample 2 vs. 3)
STEMI group					
Total protein [g/dL]:	6.29	6.91	7.12	< 0.01	< 0.01
Albumins [g/dL]	3.85	4.02	4.58	NS	< 0.05
Globulins [g/dL]:					
Fraction α-1-	0.25	0.24	0.23	NS	NS
Fraction β-	0.83	0.97	0.97	NS	NS
Fraction χ-	0.744	0.957	1.01	< 0.01	NS
IgA [mg/dL]	223.87	255.96	224.85	NS	NS
IgG [mg/dL]	862.06	1,126.64	1,162.59	< 0.01	NS
IgM [mg/dL]	93.97	120.96	96.85	NS	NS
CRP [mg/dL]	44.24	12.53	4.48	< 0.05	< 0.05
Fibrinogen [mg/dL]	415.68	480.13	368.14	< 0.05	< 0.05
UA/NSTEMI group					
Total protein [g/dL]	6.67	6.93	7.29	NS	NS
Albumins [g/dL]	3.97	4.08	4.42	NS	NS
Globulins [g/dL]:					
Fraction α-1-	0.23	0.22	0.24	NS	NS
Fraction β-	0.92	0.94	0.97	NS	NS
Fraction χ-	0.908	0.979	1.055	< 0.01	NS
IgA [mg/dL]	245.07	264.44	258.81	NS	NS
IgG [mg/dL]	1,016.87	1,062.25	1,122.73	NS	NS
IgM [mg/dL]	148.2	155.25	138.94	NS	NS
CRP [mg/dL]	20.91	6.3	3.01	< 0.05	< 0.05
Fibrinogen [mg/dL]	366.5	413.06	314.33	< 0.05	< 0.05

STEMI — ST segment elevation myocardial infarction; UA — unstable angina; NSTEMI — non-ST segment elevation myocardial infarction; IgA — immunoglobulin A; IgG — immunoglobulin G; IgM — immunoglobulin M; NS — not significant (p ≥ 0.05)

a continuously raised immunoglobulin level, suggesting a mild but constant inflammatory activation of the immune system. Whereas, in the STEMI patient, the inflammatory proteins, which initially remained low, dramatically increased in response to an acute phase of ACS (Table 3). These findings are consistent with the very limited evidence from the literature published in the 20th century. Logacheva et al. [16] reported a marked immune reaction to ACS associated also with increased circulating immune complexes and cardiolipin antibodies. The mean values of M and G immunoglobulins were found to be increased in the acute, subacute, and post-infarction periods, but no distinction was made with regard to the type of ACS (STEMI vs. UA/NSTEMI) [17].

Interestingly, Kristensen et al. [18] reported an association of increased concentration of IgG and the prevalence of cardiovascular risk factors i.e. hypertension. This supports our hypothesis that high risk patients, who suffer from multiple comorbidities, might have a continuous activation of their immune system, expressed by increased serum concentration levels of selected immunoglobulin classes.

CRP concentration was markedly elevated irrespective of the type of ACS, and decreased over the first couple of weeks following the incidence. Although in the STEMI group, the baseline concentration of CRP was significantly higher compared to the UA/NSTEMI group, the respective standard deviations were relatively wide (44.24 ± 46.10 vs. 20.91 ± ± 28.81). This would suggest that not all STEMI patients have elevated CRP in the acute phase of ischaemia. These findings are consistent with reports found in the literature. Liuzzo et al. [19] established that in a substantial proportion of STEMI cases, CRP concentration is not elevated more than in other types of ACS. They also found that CRP was elevated more if the patient had been diagnosed with an ischaemic heart disease in the past [20]. This could suggest the existence of an inflammatory component that would trigger plaque destabilisation in this subgroup of ACS patients. A recently published study that compared hs-CRP with plaque morphology assessed using optical coherent tomography revealed that inflammatory marker concentration is significantly higher among patients with plaque rupture and those with thin-cap fibroatheroma [11]. According to the authors, a cut-off level of 4.5 mg/L for hs-CRP could detect a ruptured plaque with a sensitivity of 91.7% and a specificity of 77.8%. This confirms a potential role for the measurement of inflammatory markers in patients at risk. On the other hand, CRP did not show additional predictive value of significant atherosclerotic stenosis when added to traditional cardiovascular risk factors [21]. The inflammatory markers could be elevated for non-cardiac

reasons. However, as reported by Czerniuk et al. [22] in an ACS population, with a concomitant periodontal disease (PD), the hs-CRP decrease after MI is analogous to that of non-PD patients.

The change in CRP values over time differed from the fibrinogen dynamics. The serum concentrations of the later one appeared to be significantly higher at the second week post ACS when compared to the values on admission, and those measured after the third month. The pattern of a peak in fibrinogen concentration was identified in the previous century when fibrinogen measurements were done routinely in all MI patients [23]. Those precise reports delivered daily changes in its concentration, indicating that the highest values appear around day 5 after ACS. However, a recent large study by De Luca et al. [24] revealed that there is a significant association for extremely high levels of fibrinogen (5th percentile) and coronary artery disease with at least one lesion of more than 50% stenosis ($p < 0.0001$). This might shed a new light on the role of fibrinogen in identifying groups at high risk for ischaemic heart disease.

If we analyse the dynamics of CRP and fibrinogen concentrations over time, we should recognise two important points. Firstly, both inflammatory markers demonstrate a statistically significant peak of serum concentration after ACS. This confirms a significant relation between the clinical presentation of symptoms and the biochemical response of the body.

But secondly, the time from the onset to the peak is different for each of the two markers. CRP would be elevated at a very early stage of the syndrome and gradually diminish over the subsequent three months. Whereas fibrinogen would slowly rise from relatively normal values on admission to its maximum peak concentration, as measured in our study, at week 2, and then reduce back to its starting point by the third month (Table 3). The above observations suggest diverse roles for these two biomarkers in the pathogenesis of an acute ischaemic event. CRP would act like an early protein of the triggering inflammatory process associated with the acute phase of ACS, while fibrinogen presents as a reactive biomarker to the syndrome. This finding is indirectly supported by evidence from the literature confirming that hs-CRP can be a potential therapeutic target in coronary artery disease patients if started on statin therapy [25, 26]. Furthermore, the results of the JUPITER trial by Ridker et al. [27] show that diminution of the inflammatory process, described as a decrease of hs-CRP in statins treated patients, is correlated with a significant absolute reduction of MI and death in a one-year follow-up.

Some of the patients included in the study who suffered from STEMI underwent fibrinolysis. This type of treatment may potentially influence the concentrations of inflammatory parameters. In fact, we have not recorded these differences. However, this might have occurred due to the relatively small sample analysed in the study.

Our study has important limitations. Firstly, it was performed in a relatively small number of patients (34 cases of confirmed STEMI, 12 cases of NSTEMI and six cases of UA) and thus its results require verification in a larger-scale study. Secondly, it was not assessed actively if the patients had any associated chronic inflammatory or neoplastic disease that could influence the results. Thirdly, there was no control group of healthy individuals that could bring more insights into the characteristics of the biochemical changes. Lastly, there was no control group included in the study, which would have given a more objective view on the dynamics of the measured factors.

CONCLUSIONS

To reiterate the main points, biomarker profiles differ between distinct types of ACS. STEMI patients present with an increased immune reaction in the early phase of acute myocardial ischaemia. IgG appeared as the only immunoglobulin closely associated with ACS in the STEMI group. Fibrinogen reveals features of a reactive biomarker of ACS with a delayed rise in serum concentration, whereas CRP appears to be closely related to the causative process in these patients. There is a potential correlation between immunoglobulins and inflammatory markers.

Conflict of interest: *none declared*

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Stężenie IgG, IgM i markerów stanu zapalnego w surowicy krwi u chorych z ostrym zespołem wieńcowym: badania pilotażowe

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Streszczenie

Wstęp: Na podstawie przeprowadzanych badań zidentyfikowano wiele biomarkerów, które mogą potencjalne służyć predykcji ryzyka ostrego zespołu wieńcowego (OZW) u osób dorosłych.

Cel: Celem pracy była ocena związku między stężeniem immunoglobulin a stężeniem innych markerów stanu zapalnego korelujących z ryzykiem OZW, takich jak białko C-reaktywne (CRP) i fibrynogen.

Metody: Do badania włączono 52 kolejnych chorych z zawałem serca z przetrwałym uniesieniem odcinka ST (STEMI) lub niestabilną chorobą wieńcową/zawałem serca bez uniesienia odcinka ST (UA/NSTEMI). Wykonano pomiary stężeń dla białka całkowitego, albumin, alafa-1-globuliny, białka beta, białka gamma, immunoglobulin w klasie A (IgA), G (IgG), M (IgM) i E (IgE), kinazy kreatyninowej (CK), kinazy kreatyninowej MB (CK-MB), CRP i fibrynogenu.

Wyniki: U chorych z OZW zaobserwowano istotny wzrost stężenia gamma globuliny, CRP i fibrynogenu. Stężenie IgG było podwyższone wyłącznie w grupie STEMI i korelowało ze stężeniem fibrynogenu (R = 0,48; p < 0,01).

Wnioski: 1. IgG wydaje się być jedyną immunoglobuliną, której stężenie zmienia się u chorych ze STEMI. 2. Stężenia fibrynogenu wskazują na reaktywne zachowanie tego parametru w odpowiedzi na OZW. 3. Dynamika stężenia CRP wydaje się związana z przyczynowym procesem prowadzącym do OZW.

Słowa kluczowe: ostry zespół wieńcowy, markery, immunoglobuliny, IgG

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