

Ischemia-modified albumin in differential diagnosis of acute coronary syndrome without ST elevation and unstable angina pectoris

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Abstract

Background: Ischemia modified albumin (IMA) was registered by the United States Food and Drug Administration as a marker of myocardial ischemia.

Aim: To assess the usefulness of IMA measurement for differentiating patients with acute coronary syndrome (ACS) with no ST elevation and patients with unstable angina pectoris.

Methods: The study group consisted of 121 patients (mean age 63 ± 12 years, 84 males), who were admitted to our department with retrosternal chest pain occurring at rest and lasting more than 20 minutes. The patients had laboratory tests performed including aspartate aminotransferase, isoenzyme of creatine kinase activity, troponin T, N-terminal pro-brain natriuretic peptide (NT-proBNP), C-reactive protein, IMA concentration and creatinine clearance. Coronary angiography was also performed. All study patients were divided into 2 groups: group I with elevated troponin concentration (58 patients) and group II with troponin concentration below reference value (63 patients).

Results: The IMA concentration in the serum did not differ significantly between group I (troponin positive) and group II (troponin negative) (95.2 ± 12.8 U/mL vs 94.0 ± 17.9 U/mL, NS). The percentage of patients with elevated IMA values (cut off point of 85 U/mL) did not differ significantly between group I and group II patients (76.6% vs 76.2%, NS). In patients from group I an upward trend was noted, whereas in patients from group II a downward trend was associated with the duration of ischemic chest pain. In group I the correlation between the IMA concentration and the NT-proBNP concentration was positive ($R = 0.2957$; $p < 0.0316$). The parameters differentiating patients from group I and group II were: left ventricular ejection fraction, leukocytosis, serum glucose concentration and creatinine clearance.

Conclusions: 1. The IMA concentration does not differentiate ACS patients without ST segment elevation myocardial infarction from patients with unstable angina. 2. The upward trend of IMA concentration was associated with the duration of chest pain in patients with ACS, whereas the opposite trend was found in patients with unstable angina pectoris.

Key words: IMA, ACS with no ST elevation, unstable angina pectoris

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INTRODUCTION

Acute coronary syndrome (ACS) is a sequence of pathophysiologic processes in coronary arteries, where inflammatory cytokines play an important role causing plaque's destabilization and rupture, myocardial ischemia and infarction [1].

A gold standard in diagnosis of the necrosis of myocardium is troponin due to its high sensitivity and specificity. However, troponins are not very early markers, and up to 4 hours from the onset of symptoms their sensitivity does not exceed 50% [2]. Importantly, the high sensitivity (90%) of protein binding

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fatty acids has been confirmed as an early (within 3 hours from the onset of symptoms) marker of myocardial necrosis [3, 4].

Diagnosis of ACS is not difficult to be made if a patient presents with a typical chest pain, ECG changes and elevated markers of myocardial infarction. However, the diagnosis becomes problematic when the ECG changes are atypical and biomarker levels are not elevated. The question remains whether we face reversible ischemia or maybe myocardial necrosis is present.

During acute ischemia of myocardium, the ability of binding ions such as copper, zinc and cobalt is decreased, therefore a form of albumin is produced, which is described as ischemia modified albumin (IMA). The IMA was registered by the United States Food and Drug Administration as a marker of myocardial ischemia. The test is based on the decreased ability of albumin to bind cobalt due to the structural change of NH₂ which develops in the ischemic environment. It is important to emphasise that the decreased ability of albumins to bind cobalt occurs in hypoxia, acidosis, sodium and calcium pump malfunctions, and tissue damage caused by free radicals [5, 6]. It has been shown that the concentration of IMA increases within a couple of minutes from the onset of ischemia, remains elevated until 6 to 12 hours and returns to its normal range after 24 hours [7].

The aim of the study was to examine the usefulness of IMA in differentiation between patients presenting with myocardial infarction without ST elevation (NSTEMI) and unstable angina.

METHODS

Study group

The study was performed on 121 patients, aged between 26 and 100 years (mean 63.2 ± 11.5 years), of whom 84 (69.4%) were males, admitted to hospital with prolonged (> 20 min) retrosternal chest pain radiating to the neck and/or left shoulder [79 (62.3%) patients] or with stable angina that progressed to the CCS class III [35 (28.9%) patients].

The study was approved by the Bioethics Committee (RNN/576/07/KB — 11 XII 2007).

The study population was divided into two groups: group I and II. Group I comprised 58 patients with elevated troponin T (TnT > 0.010 ng/mL) at the admission and following increase of > 0.03 ng/mL (NSTEMI) during hospitalisation. Group II consisted of 63 patients with first troponin levels, measured at the hospital admission, within normal range (< 0.010 ng/mL), which did not increase during the hospitalisation (unstable angina).

Study design

All study patients had ECG performed. The blood was drawn on the admission to measure troponin T (TnT), aspartate aminotransferase (AspAT), alanin aminotransferase (AlAT) and creatinine kinase MB (CK-MB). If the first troponin T measurement was below 0.010 ng/mL, the second measurement

was repeated at 6 hours and 12 hours in some patients. The following tests were also performed: 1) complete blood count; 2) serum glucose, urea, creatinine, electrolytes such as sodium and potassium, total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, IMA, NT-proBNP and C-reactive protein (CRP), 3) fibrinogen. Twenty-three (19.2%) patients underwent echocardiography on admission and all the remaining patients had the echocardiography performed on day 4–5 of hospital stay. All patients underwent coronary angiography.

The weight of patients was measured and body mass index (BMI) was calculated. Creatinine clearance was calculated based on the Cockcroft-Gault formula [8].

The IMA concentration was measured using Roche Cobas Integra 400 plus platform. The test used is also known as ACB™ (Albumin Kobalt Binding). The troponin T concentration and NT-proBNP were measured via electroluminometric immunoassay (ECLIA) method using Roche Elecsys 2010 platform.

Statistical analysis

The results are presented as means \pm standard deviation or numbers and percentages. The statistical significance of the differences between the study groups was assessed using student t test (for parametric variables) and U Mann-Whitney test (for non-parametric variables). In order to test for normal distribution the Shapiro-Wilk test was performed. We used χ^2 and χ^2 with Yates correction to compare categorical variables. The interrelationship of the variables was also assessed using Spearman's rank correlation coefficient. We adopted a p value of < 0.05 as statistically significant. All calculations were performed using STATISTICA PL 7.1.

RESULTS

The longest time from the onset of ischemia to the first blood draw and assessment of myocardial necrosis biomarkers was 14 hours in group I and 22 hours in group II. Baseline characteristics of the study groups are depicted in Table 1.

Compared to the group II patients, patients in group I had higher WBC count, higher serum glucose, creatinine, CRP, AspAT and lower creatinine clearance (Table 1). The two study groups, group I (troponin positive) and group II (troponin negative), did not differ in respect to the IMA concentrations. However, CK-MB and NT-proBNP levels were significantly higher in group I when compared to group II patients (Table 2).

The percentage of patients with elevated IMA concentration in blood serum (cut off point of 85 U/mL — per manufacture's manual) did not differ between group I and II patients, and accounted for 77.6% and 76.2%, respectively. The percentage of patients with elevated IMA concentration within 3 hours from the onset of ischemia was 12.1% in group I and 17.5% in group II. The 3–6 hour IMA and beyond 6 hours IMA levels for group I and group II patients respectively, were as follows: 24.1% and 12.7%, 41.4% and 46.0% (NS). The mean

Table 1. Baseline characteristics of the study groups

Variables	Group I (troponin-positive) n = 58	Group II (troponin-negative) n = 63	p
Age [years]	64.9 ± 11.6	61.7 ± 11.2	NS
Height [cm]	168 ± 6.8	169 ± 7.8	NS
Males, n (%)	45 (77.6)	39 (61.9)	NS
Females, n (%)	13 (22.4)	24 (38.1)	NS
Weight [kg]	78 ± 13.4	82 ± 14.1	NS
Body mass index [kg/m ²]	27.0 ± 4.1	28.7 ± 4.2	NS
Hypertension, n (%)	43 (74.1)	39 (61.9)	NS
Diabetes, n (%)	14 (24.1)	11 (17.5)	NS
Smoking, n (%)	19 (32.8)	16 (25.4)	NS
Peripheral artery disease, n (%)	6 (10.3)	2 (3.2)	NS
ECG changes, n (%):			
Left bundle branch block, n (%)	3 (5.2)	2 (3.2)	NS
Atrial fibrillation, n (%)	4 (6.9)	5 (7.9)	NS
ST segment depression and/or T inversion, n (%)	49 (84.5)	47 (74.6)	NS
Normal ECG, n (%)	2 (3.5)	9 (14.3)	NS
Time from the onset of symptoms to hospital admission [hours]	7.0 ± 4.2	9.2 ± 7.3	NS
Heart rate [beats/minute]	78.0 ± 13.4	73.9 ± 11.8	NS
Systolic blood pressure [mm Hg]	127 ± 17.2	126 ± 17.9	NS
Diastolic blood pressure [mm Hg]	79 ± 8.6	77 ± 12.5	NS
Left ventricular ejection fraction [%]	52.0 ± 11.3	59.5 ± 11.3	NS
Laboratory tests:			
White blood cell count [thousands/mL]	9.0 ± 2.8	7.7 ± 2.6	< 0.0039
Glucose [mg/dL]	130.0 ± 55.7	111.4 ± 33.1	< 0.008
Creatinine [mg/dL]	1.0 ± 0.3	0.9 ± 0.3	NS
C-reactive protein [mg/L]	7.4 ± 8.1	4.1 ± 4.1	NS
AspAT [U/L]	70.8 ± 93.1	24.8 ± 9.3	< 0.0001
Creatinine clearance [mL/min]	89.3 ± 36.9	108.7 ± 45.5	NS

Table 2. Concentrations of cardiac necrosis/ischemia biomarkers

Variables	Group I (troponin-positive), n = 58	Group II (troponin-negative), n = 63	p
IMA [U/mL]	95.2 ± 12.8, n = 58	94.0 ± 17.9, n = 63	NS
CK-MB [U/L]	79.7 ± 127.1, n = 51	21.1 ± 10.2, n = 50	0.0001
NT-proBNP [pg/mL]	2451.1 ± 5878.9, n = 53	464.1 ± 694.8, n = 46	0.0002

IMA — ischemia modified albumin; CK-MB — creatinine kinase-MB; NT-proBNP — N-terminal prohormone brain natriuretic peptide

IMA concentrations in respect to the time measured from the onset of ischemia are presented in Figure 1. The mean albumin concentrations of the study patients were within the reference range (3.8 g/dL to 5.1 g/dL).

Coronary angiography revealed more severe atherosclerotic changes in major coronary vessels in group I, than in group II patients [55 (94.8%) vs 49 (77.8%), respectively p < 0.0149].

Left main coronary artery stenosis > 70% was present in 4 (6.9%) patients from group I and 1 (1.6%) patient in group II (NS). A three-vessel coronary artery disease (CAD) in group I and group II was recognized in 9 (15.5%) patients and 5 (7.9%; NS) patients, respectively. The 2-vessel CAD accounted for 43.1% (25 patients) in group I, and 31.7% (20 patients) in group II. The non-obstructive CAD was detected only in 11.1%

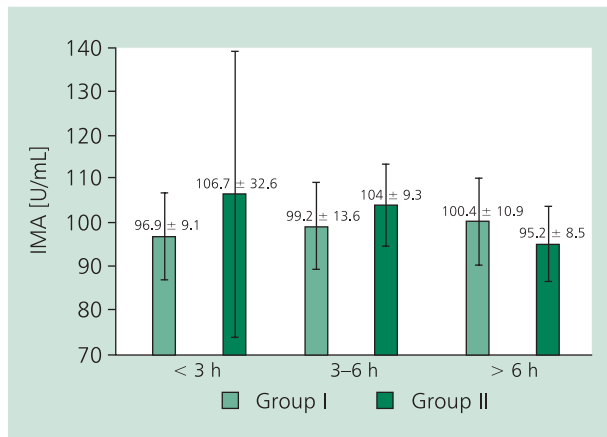


Figure 1. Mean ischemia modified albumin (IMA) concentrations in respect to the time from onset of ischemia in group I (troponin-positive) and group II (troponin-negative); $p > 0.05$

of patients in group II. There was no significant difference in percentage of patients with IMA concentration levels above the cut off point (85 U/mL) in patients with severe stenoses in major coronary arteries between the two study groups (95.6% in group I vs 79.2% in group II, NS).

The correlation analysis between the IMA concentration and the time from the onset of ischemia to the blood draw, leukocytosis, CK-MB, AspAT, troponin T concentration (only in group I patients), fibrinogen, CRP, left ventricular ejection fraction (LVEF), creatinine clearance, total cholesterol, HDL-cholesterol and triglycerides confirmed positive relationship between IMA and NT-proBNP in group I (troponin positive) ($R = 0.2957$; $p < 0.316$). There was no significant correlation between the IMA concentration and the other factors in group II and all the remaining study patients.

Variables differentiating patients with NSTEMI from patients with unstable angina pectoris included: leukocytosis, elevated NT-proBNP, increased CK-MB and AspAT, elevated serum glucose, decreased LVEF and decreased creatinine

clearance. Probability scores for NSTEMI detection by using some tested variables are presented in Table 3.

DISCUSSION

The IMA measurement as a marker of myocardial ischemia without myocardial necrosis and/or preceding myocardial necrosis has introduced the hope for improved diagnosis in patients with ACS without or with non-specific ECG changes.

So far, clinical studies have shown an increase of IMA in blood serum in patients with stable CAD who underwent percutaneous coronary intervention [9], in patients with arrhythmia after ablation, [10] and in patients with atrial fibrillation after cardioversion [11]. The IMA concentration remains unchanged in patients with positive and negative stress tests. [12, 13].

In the study by Roy et al. [14] IMA measured within 3 hours from the onset of symptoms, in patients with retrosternal chest pain with no or atypical ECG changes and normal troponin levels, has been shown to be an independent predictor of ACS. Consuegra-Sanchez et al. [15] showed that IMA measured within 3 hours from the onset of symptoms was an independent predictor of adverse outcome associated with the more frequent occurrence of combined endpoint (death, myocardial infarction and refractory ischemia), as well as with the total 30-day mortality. On the contrary, Worster et al. [16] did not find any association between elevated IMA, measured within 6 hours from the onset of ischemic symptoms, and adverse cardiac outcomes measured during the next 72 hours.

In our study, similarly to reports from Roy et al. [14], the mean IMA concentrations in blood serum and the percentage of patients with elevated IMA levels did not differentiate patients with NSTEMI from patients with unstable angina.

The mechanism of IMA increase is not well understood. It has been pointed out that active oxygen forms may play a role in the albumin modification process. The oxidative stress, associated with ischemia and reperfusion of myo-

Table 3. Odds ratio (OR) and 95% confidence intervals (CI) of variables increasing the probability of myocardial infarction diagnosis: activity of creatinine kinase-MB (CK-MB), activity of aspartate aminotransferase (AspAT), N-terminal prohormone brain natriuretic peptide serum concentration (NT-proBNP), left ventricular ejection fraction (LVEF), white blood cell count, serum glucose concentration and creatinine clearance

Diagnostic test	OR	-95% CI	+95% CI
CK-MB ≥ 20.50 [U/L]	5.45	2.34	12.71
AspAT ≥ 24.5 [U/L]	4.96	2.23	11.02
NT-proBNP ≥ 247.85 [pg/mL]	4.79	2.07	11.07
LVEF ≤ 59 (%)	3.82	1.81	8.07
WBC [thousands/mL] ≥ 7.55	3.12	1.50	6.51
Glucose ≥ 109.5 [mg/dL]	2.85	1.37	5.90
Creatinine clearance ≤ 93.92 [mL/min]	2.39	1.14	4.98

cardium, leads to the reduction of cobalt ion binding by human albumins [17].

It has been confirmed by Roy et al. [18], that active oxygen forms, in particular hydroxyl radicals, might modify chemically human albumin and produce IMA.

The experimental studies indicated that IMA might be produced through an adaptive response of human body to developing hypoxia. It is well known, that hypoxia activates multiple genes, which are responsible for cell and tissue adaptation to the hypoxic environment [19]. Endogenous myocardial mechanisms prevent cells from dying, which are considered to be an integral part of the process known as “the late phase of preconditioning” [20]. Xi et al., [21] proved that the induced NO (iNOS) synthesis played an important role in the discussed late cardioprotective process. In the following studies by Xi et al. [22], these authors reported that cobalt chloride through its activation of hypoxia-inducible factor 1 (HIF-1) and activator protein 1 (AP-1) was responsible for the cardioprotective mechanism, and along with iNOS causes the late phase conditioning.

Since 1992, when HIF-1 [19] was discovered, there have been approximately 60 genes described, which are associated with different cells' functions (proliferation, survival, apoptosis, glucose metabolism and angiogenesis) which also induce the synthesis of HIF-1 [23]. The synthesis of HIF-1 might be also induced in the hypoxic environment by growth factors, cytokines, angiogenic hormones and viral proteins. HIF-1 consists of two sub-units: alpha and beta. The beta sub-unit exists inside the cells in every condition, but the presence of alpha sub-unit, which is the main functional protein, is associated with an extra stimulus, such as hypoxia. During hypoxia the degradation of HIF alpha is inhibited by proteasomes. It has been shown, in the experimental studies, that HIF-1 alpha plasmid inoculation into the post infarction necrotic myocardium was associated with the shrinking of infarcted area and the increase of myocardial perfusion [24]. In the light of the above mentioned data, the increase of IMA might be interpreted as the endogenous response mechanism to hypoxia, that prevents or decreases the necrosis of myocardium.

The above suggestions seem to support the studies in stable CAD patients [12], in whom the positive correlation, at rest, has been reported between the estimated indicator of energy consumption (heart rate multiplied by systolic blood pressure) and the concentration of IMA in blood serum. The negative correlation discovered immediately after exertion, in the same population, may suggest insufficient mechanisms of cardiac protection during ischemia. The highest IMA concentration in patients with ACS has been reported within 3 hours from the onset of ischemic symptoms. The lower IMA concentration has been described in the ACS patients with onset of symptoms between 3 and 6 hours, and the lowest in patients with time from the onset of symptoms beyond 6 hours. The opposite relationship has been described in patients with unstable angina.

Furthermore, our own studies indicated a significant positive correlation in ACS patients without ST elevation between the IMA concentration and the NT-proBNP in blood serum, which indicates the dysfunction of the myocardium [25]. Contrary to the analysis reported by Dominiguez-Rodriguez et al. [26], no correlation has been found between the LVEF and the IMA concentration.

The IMA concentration depends on the function of kidneys and the total blood serum concentration of albumin. There has been correlation reported between the creatinine concentration and the IMA concentration in blood serum, which decreased after the procedure, in patients undergoing dialysis [27]. In our patients who had normal or slightly impaired kidney function, we did not find any correlation between the serum creatinine concentration and the serum IMA concentration. In patients with hypalbuminemia, the serum IMA concentration increases (less cobalt is bound). In patients with normal albumin serum concentration, the correlation between the albumin concentration and the serum IMA concentration is within referenced range [28]; in our studies the serum albumin concentration was within the referenced range.

CONCLUSIONS

1. IMA concentrations do not differentiate patients with ACS and NSTEMI from patients with unstable angina.
2. There was a positive correlation between IMA concentration and the duration of chest pain in patients with ACS, whereas a negative correlation was found in patients with unstable angina.

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Albumina modyfikowana niedokrwieniem w diagnostyce różnicowej zawału serca bez przetrwałego uniesienia odcinka ST i niestabilnej duszniczy bolesnej

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Streszczenie

Wstęp: Albumina modyfikowana niedokrwieniem (IMA) uzyskała rejestrację *Food and Drug Administration* jako marker niedokrwienia mięśnia sercowego.

Cel: Ocena przydatności oznaczania stężenia IMA w surowicy, w różnicowaniu chorych z zawałem serca bez przetrwałego uniesienia odcinka ST oraz pacjentów z niestabilną dusznicą bolesną.

Metody: Badania przeprowadzono u 121 osób przyjętych do Kliniki z powodu przedłużającego się (> 20 min) spoczynkowego bólu zamostkowego. U badanych wykonano podstawowe badania laboratoryjne oraz oznaczono w surowicy stężenie troponiny T (TnT), aktywność aminotransferazy asparaginianowej (AspAT), izoenzymu kinazy kreatynowej (CK-MB), końcowego fragmentu peptydu natriuretycznego (NT-proBNP), białka C-reaktywnego (CRP), albuminy modyfikowanej niedokrwieniem oraz obliczono klirens kreatyniny i wykonano badanie koronarograficzne. Badanych podzielono na dwie grupy — I: z podwyższonym stężeniem troponiny (n = 58) i II: ze stężeniem troponiny poniżej wartości referencyjnych (n = 63).

Wyniki: Stężenie IMA w surowicy nie różniło się istotnie między grupą I (troponino-dodatnią) i grupą II (troponino-ujemną) ($95,2 \pm 12,8$ U/ml v. $94,0 \pm 17,9$ U/ml). Również odsetek badanych z podwyższonymi wartościami IMA (punkt odcięcia 85 U/ml) nie różnił się istotnie między grupą I i II (76,6% v. 76,2%). U chorych z grupy I obserwowano trend wzrostowy, zaś u osób z grupy II trend malejący stężenia IMA związany z czasem trwania bólu wieńcowego. W grupie I stwierdzono dodatnią korelację między stężeniem IMA w surowicy i stężeniem NT-proBNP ($R = 0,2957$; $p < 0,0316$). Zmiennymi mierzalnymi różnicującymi chorych z grupy I i II były: CK-MB, AspAT, NT-proBNP, frakcja wyrzutowa lewej komory, leukocytoza, stężenie glukozy w surowicy i klirens kreatyniny.

Wnioski: 1. Stężenia albuminy modyfikowanej niedokrwieniem nie różnicują chorych z zawałem serca bez przetrwałego uniesienia odcinka ST i pacjentów z niestabilną dusznicą bolesną. 2. Trend wzrostowy stężenia IMA w surowicy był związany z czasem trwania bólu wieńcowego u chorych z ostrym zespołem wieńcowym, zaś malejący u osób z niestabilną dusznicą bolesną.

Słowa kluczowe: IMA, zawał serca bez przetrwałego uniesienia ST, niestabilna dusznicza bolesna

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