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Myeloperoxidase and global longitudinal strain in prognostication of clinical events after ST segment elevation myocardial infarction

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Elevated levels of several inflammatory cytokines and chemokines, such as myeloperoxidase (MPO) and some characteristics of damaged myocardium such as myocardial deformation (global longitudinal strain — GLS) seem to be promising biomarkers for acute coronary syndrome, but their predictive ability for clinical outcomes amongst ST segment elevation myocardial infarction (STEMI) patients having obesity remained unclear.

The aim of the study was to to determine the impact of MPO and GLS on prediction of 1-year combined clinical events in STEMI patients successfully treated with primary percutaneous coronary intervention (PCI) depending on abdominal obesity presentation.

Materials and methods. We prospectively enrolled 102 individuals with STEMI who were successfully treated with primary PCI. All patients gave their consent to participate in the study. The biomarkers (MPO, cardiac troponins), echocardiographic and Doppler parameters including global longitudinal strain (GLS) were evaluated at the baseline.

Results. The MPO level in the entire population of patients with STEMI was 129.45 [48.48—179.70] ng/ml. The group was divided into two cohorts depending on the median level of MPO (i 98.34 ng/ml and < 98.34 ng/ml). We observed 26 combined endpoints (10 and 17 in patients with MPO ≥ 98.34 ng/ml and < 98.34 ng/ml, respectively (F test = 0.064285; $\varepsilon^2 = 4.29$; p = 0.046). Multivariate linear regression showed that the only GLS and MPO remained independent predictors for the clinical outcomes.

Conclusions. Global longitudinal strain was the best predictor for 1-year combined clinical outcomes in STEMI patients who were undergone successful primary PCI. Body fat accumulation seems to show borderline significance when compared with GLS, while it was sufficiently better than MPO.

Keywords:
STEMI, myeloperoxidase, visceral fat accumulation, global longitudinal strain, prognosis.

Acute ST segment elevation myocardial infarction (STEMI) remains challenging clinical condition with strong contribution to significant morbidity and mortality worldwide [43]. STEMI requires urgent reperfusion mainly percutaneous coronary intervention (PCI), but during COVID-19 pandemic the delay in reperfusion was found in the majority of countries due to both patient-related (predominantly coexisting conditions and comorbidities) and systemic medical care factors [17]. All these make in turn the diagnosis of STEMI and short- and long-term prognosis amongst STEMI patients after PCI highly responsible and thoroughly evaluated. Conventional predictive scores, such as Thrombolysis in Myocardial Infarction (TIMI) or Global Registry of Acute Cardiac Events (GRACE), seem not to be extrapolated to STEMI patients with a higher burden of abdominal obesity, diabetes mellitus, hypertension and chronic renal disease [4].

Inflammation playing a pivotal role in accelerating atherosclerosis deals metabolic comorbidities including abdominal obesity with atherothrombotic

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complications. Indeed, adipose tissue produces and releases a large spectrum of adipokines, which are considered to be multifunctional in terms of modulating inflammation and immune response and having direct actions on remote tissues including myocardium and vasculature [12]. Interestingly that not just ectopic expansion of adipose tissue mainly perivascular or pericardial localization, but also fat quality was noticed to be associated with all-cause mortality and non-cardiovascular death [7, 45, 46], whereas visceral fat accumulation was strongly associated with increased cardiovascular (CV) risk [28, 31, 38, 48].

Myeloperoxidase (MPO) is a member of chemokine superfamily called hemeperoxidases that are mainly stored in circulating leukocytes and having by far lower expression in tissue monocytes/macrophages [36, 40]. There is strong evidence regarding the fact that MPO was widely associated with oxidative stress and inflammation of adipose tissue [34, 50]. The chemokine is produced by activated neutrophils, monocytes/macrophages, which are recruited from circulation into the adipose tissues and where they intervene a release of pro-inflammatory cytokines [33]. Myeloperoxidase is the most abundant protein in human neutrophils that being a physiological antagonist of nitric oxide plays a crucial role in mitochondrial oxidative stress, lipoprotein oxidation, and endothelial dysfunction [20, 39]. Therefore, increased circulating levels of MPO were found to be a powerful trigger for plaque rupture and microvascular inflammation and obstruction in STEMI patients [3, 55]. Although elevated levels of MPO was noticed as a promising biomarker with discriminative potency for persistent chest pain due to acute coronary syndrome, its predictive ability for clinical outcomes amongst STEMI patients having obesity remained unclear [5,11,24]. The aim of the study was to determine the impact of MPO on prediction of 1-year clinical events in STEMI patients successfully treated with primary PCI depending on abdominal obesity presentation.

Materials and methods

Ethical Declaration

All procedures performed due to the ethical standards and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards and approved by the local ethics committee (Protocol #6, 30.05.2017). All the patients signed the inform consent.

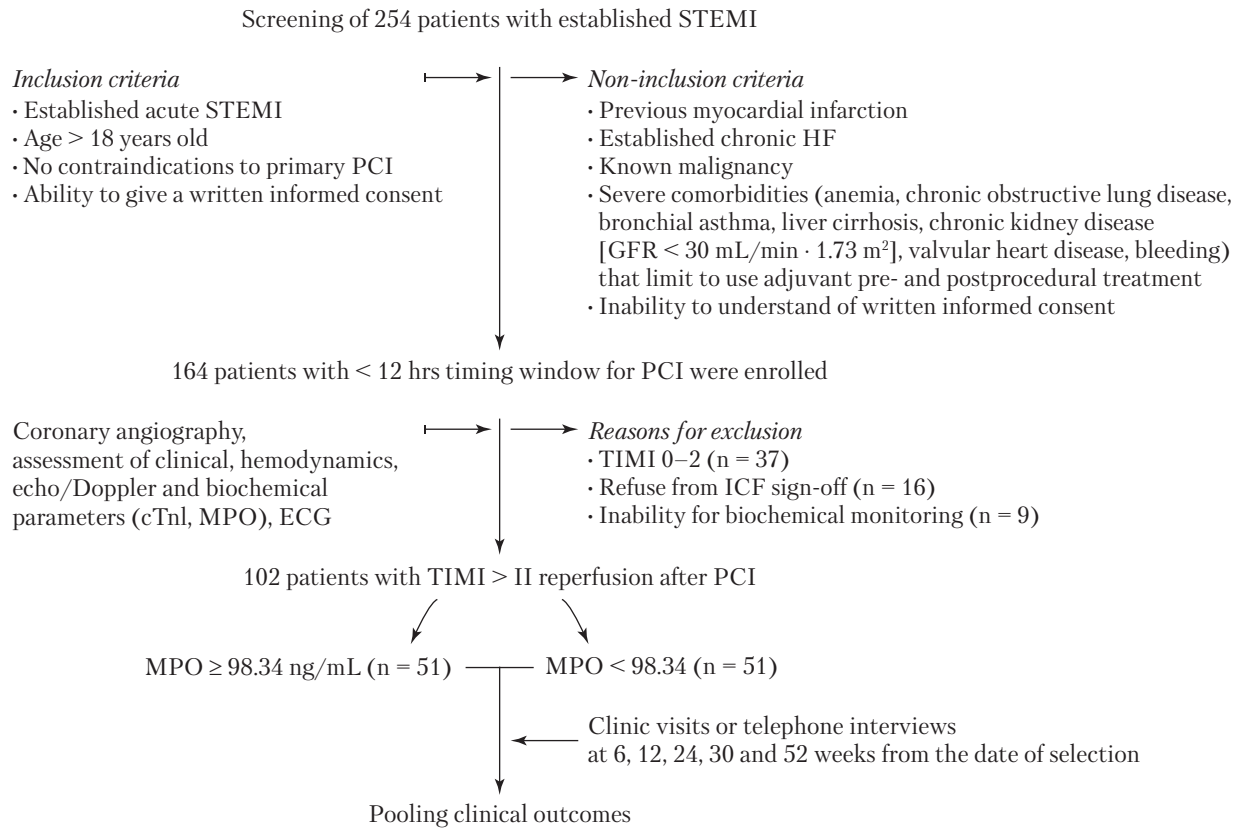

Figure 1. The design of the study: STEMI, ST segment elevation myocardial infarction; PCI, percutaneous coronary intervention; TIMI, Thrombolysis in Myocardial Infarction; COPD, chronic obstructive pulmonary disease; GFR, glomerular filtration rate; MPO, myeloperoxidase

Study population

Two hundred and fifty four patients with acute STEMI were prospectively screened with the aim of participation in the study according to inclusion criteria (acute STEMI, age > 18 years old, and a lack of contraindications to PCI). These patients were urgently admitted to the intensive care unit (ICU) of GI «L.T. Mala National Therapy Institute of NAMS of Ukraine» within a given period from January 2018 to February 2021. Figure 1 represents a flow chart with clear inclusion/non-inclusion criteria.

Exclusion criteria were previous myocardial infarction, established chronic heart failure, known malignancy, severe comorbidities (anemia, chronic obstructive lung disease, bronchial asthma, liver cirrhosis, chronic kidney disease, valvular heart disease, bleeding), and inability to understand of written informed consent. Taking into account non-inclusion criteria we finally enrolled 102 individuals who and completely met inclusion/exclusion criteria. All patients with STEMI who were in ICU during COVID-19 pandemic had been demonstrated twice-negative PCR COVID-19 tests before hospitalization. We diagnosed acute STEMI according to ECS Guidelines (2017) [15].

Coronary angiography and PCI

Coronary angiography was performed immediately after admission of the patients to the hospital using Digital X-Ray system «Integris Allura» (Philips Healthcare, Best, The Netherlands) and managed by radial or femoral vascular access. Coronary arteries were visualized with two-to-three orthogonal projections per conventional protocol. We used automatic contrast injector in order to support procedure with the contrast «Ultravist-370» (Baier Pharma GmbH, Germany). Primary PCI with bare-metal stent RebelTM (Platinum Chromium Coronary Stent System, Boston Scientific, USA) implantation in culprit artery was performed.

Medications

All enrolled patients received standard adjuvant treatment in accordance to current ESC recommendation on acute STEMI [15]. A 300-mg loading dose of clopidogrel and aspirin along with 80 mg of atorvastatin were routinely applied for these patients immediately before the interventional procedures. Administration of aspirin (100 mg/d) indefinitely and a P2Y12 receptor antagonist (clopidogrel [75 mg/day] or ticagrelor [180 mg/daily]) for 12 months after PCI were strongly encouraged in these patients. Adjusted daily doses of low molecular heparin/unfractionated heparin were used for perioperative anticoagulation. We ascertained patients with prescribed guideline-directed medications post discharge, including angiotensin-converting enzyme inhibitor (ACEI) or angiotensin II receptor blockers (ARBs), beta-blockers, and statins. The glycemic control target in patients with established diabetes mellitus was defined as fasting plasma glucose < 6.1 mmol/L, and prandial plasma glucose < 8.0 mmol/L. We used initial insulin dose for short time in adjusted every day doses depending on the values from the seven-point glucose profile. After discharge type 2 diabetes mellitus (T2DM) patients were treated with combination of diet and metformin and or SGLT2 inhibitor when needed.

Determination of clinical events

Prospective follow-ups were by conducted by a medical doctor as clinic visits or telephone interviews at 6, 12, 24, 30 and 52 weeks from the date of selection. The primary endpoint was combined event (follow-up major adverse cardiac events — MACEs) that occurred during 1-year after discharge from the hospital. MACEs were defined as the composite of CV death, recurrent MI, newly diagnosed heart failure, rhythm and conduction disturbances. CV death was ascertained by personal or phone contacting the family doctor or the hospital where the patient died. The diagnosis of recurrent myocardial infarction required the presence of clinical signs/symptoms or electrocardiographic changes, identification of local contractility dysfunction by echocardiography and elevation of biomarkers of necrosis, i.e. creatinine kinase isoenzyme-MB (CK-MB) and troponin T [15]. Severity of heart failure was assessed according to the Killip classification. The diagnosis of heart failure (HF) has been established according to ESC clinical guideline and based on appearance of clinical sign and symptoms of HF, determination of lowered left ventricular (LV) ejection fraction (EF) < 50 %, structural alterations (left atrial volume index [LAVI] > 34 mL/m^2 or a LV mass index (LVMI) ≥ 115 g/m^2 for males and ≥ 95 g/m^2 for females, E/e′ ≥ 13), increasing NT-fragment pronatriuretic peptide > 125 pg/mL [27].

Concomitant diseases

Dyslipidemia was diagnosed if total cholesterol level was above 5.2 mmol/L, and/or low density lipoprotein cholesterol level was above 3.0 mmol/L, and/or level of triglycerides (TG) was above 1.7 mmol/L according to with European Cardiology Society dyslipidemia guideline (2019) [25]. Hypertension was diagnosed if systolic blood pressure was > 140 mm Hg, and/or diastolic blood pressure > 90 mm Hg according to European guideline on diagnostics and treatment of arterial hypertension (2018) [56]. Type 2 diabetes mellitus

was determined according to new ADA statement (2019) [8] or having diabetes treatment records. Scales OMRON BF511 was used to determine of body parameters: mass, visceral fat content (%), daily metabolism (Kcal), body mass index (kg/m^2), skeletal muscle (%). Body mass index was calculated using the ratio of body weight in kilograms and the square of the height in meters. Estimated glomerular filtration rate (eGFR) was calculated using the chronic kidney disease epidemiology collaboration (CKD-EPI) [23].

Echocardiography and Doppler examination

Echocardiography and Doppler were carried out on ultrasound machine «Aplio 500» (TUS-A500) Toshiba Medical Systems Corporation (Japan) with 3.5 MHz phase probe at discharge from the ICU. LV end diastolic volume (EDV),LV end systolic volume (ESV), LV EF measuring were performed according to Simpson's method [30]. Longitudinal global strain was evaluated in accordance to the conventional method [32].

Sample size

Sample size was calculated by using single population proportion formula taking into consideration the following assumptions: 50 % prevalence, 95 % confidence level of significance alpha 0.05 = 1.96, and 5 % margin of error, 7 % in-hospital mortality, 13 % out-hospital mortality. The final sample size was 102 patients.

Blood samples

Blood samples were drawn before PCI and were centrifuged, serum was isolated and stored in plastic tubes until being shipped to the laboratory of immune-chemical and molecular-genetic researches of GI «L. T. Mala National Therapy Institute of NAMS of Ukraine». Whole blood was collected into tubes, and were centrifuged per protocol at 4 °C, 3000 rpm for 10 minutes and the supernatant (serum or plasma) was cryopreserved at –70 °C for testing. The assay of the biomarkers was performed by MPO Instant Elisa kit, Invitrogen, Austria. Troponin I (Tn I) level detected with chemo luminescent immunoassay (Humalyser 2000, Mannheim, Germany). The TnI level average was 0.5—50 ng/mL. Total cholesterol, low density lipoprotein cholesterol, high density lipoprotein (HDL) cholesterol and triglycerides (TG) were measured direct enzymatic method (Roche P800 analyzer, Basel, Switzerland). The intra-assay and inter-assay coefficients of variation were < 5 %. Fasting glucose level was measured by a double-antibody sandwich immunoassay (Elecsys 1010 analyzer, F. Hoffmann-La Roche Diagnostics, Mannheim, Germany). The intra-assay and inter-assay coefficients of variation were < 5 %. N-terminal fragment of brain natriuretic peptide (NT-proBNP) was measured by commercially available standard kit (R&D Systems GmbH, Wiesbaden-Nordenstadt, Germany). The NT-proBNP level average was 10—12000 pg/mL.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows version 21 (IBM Corp, Armonk, New York). The distribution pattern of the variables was analyzed using the Kolmogorov-Smirnov test. Continuous variables are presented as mean ± standard deviation (SD) when normally distributed. Categorical variables are presented as frequencies and percentages. The Student t test was used to compare parametric continuous variables, and the Mann-Whitney U test was used to compare nonparametric continuous variables. Categorical variables were compared using the χ^2 test and Fisher exact test, the results of which were presented as percentages. We used the Kendall rank correlation coefficient (τ) to describe associations amongst circulating levels of biomarkers, body fat accumulation and cardiac hemodynamics performances. Univariate and multivariate linear regressions with stepwise forward selection process were consequently performed to identify independent risk factors for end-point in STEMI patients (MACE). We calculated beta coefficient, standard deviation (SD), odds ratio (OR), 95 % confidence interval (CI) for each factor. The risk curves of the different groups were mapped, and receiver operating characteristic (ROC) curves were constructed to show optimum cut-off points of MPO with Youden test and using the intersection point by plotting sensitivity against specificity. Predictive models were compared by maximum likelihood ratio test (nested models). Validity conditions of the multiple linear regression model were checked. All differences were considered statistically significant with 2-tailed $p < 0.05$.

Results and discussion

The patient's population consisted of 102 persons mainly men with the age of 60.8 ± 9.70 years having several comorbidities and conventional CV risk factors including hypertension (82 %), smoking (52 %), type 2 diabetes mellitus (25 %), obesity (28 %), and hypercholesterolaemia (32 %). Primary PCI was successful and all included patients had TIMI > 2.

The MPO level in the total population of patients with STEMI was 129.45 [48.48—179.70] ng/mL. Entire group of STEMI patients was divided into two cohorts depending on the median level of MPO (≥ 98.34 and < 98.34 ng/mL). Clinical characteristics of STEMI patients' cohorts presents in Table 1.

Table 1. Patient characteristic, risk factors depending on median myeloperoxidase level

Indicator	Entire group (n = 102)	MPO ≥ 98.34 ng/mL (n = 51)	MPO < 98.34 ng/mL (n = 51)	p
Age	60.8 ± 9.70	59.98 ± 9.69	60.78 ± 9.78	0.678
Men	76 (74.5 %)	37 (72.5 %)	39 (76.5 %)	0.650
Hypertension	84 (82.4 %)	44 (86.3 %)	40 (78.4 %)	0.299
T2DM	26 (25.5 %)	11 (21.6 %)	15 (29.4 %)	0.364
Smoking	53 (52.0 %)	27 (52.9 %)	26 (51.0 %)	0.843
Heredity coronary artery disease	35 (34.1 %)	16 (31.4 %)	19 (37.3 %)	0.532
Hypercholesterolemia	33 (32.4 %)	16 (31.4 %)	17 (33.3 %)	0.664
BMI > 30 kg/m^2	28 (27.5 %)	20 (39.2 %)	10 (19.6 %)	0.030
Skeletal muscle, %	29.56 ± 6,08	29.63 ± 5.72	29.49 ± 6.49	0.911
Total fat, %	32.04 ± 9.27	31.67 ± 8.70	32.42 ± 9.91	0.689
Visceral fat, %	13.65 ± 5.84	14.80 ± 6.47	12.45 ± 4.88	0.043
Heart rate, per 1 minute	76.53 ± 12.57	77.06 ± 11.53	76.02 ± 13.60	0.591
Systolic blood pressure, mm Hg	134.51 ± 27.27	136.82 ± 32.18	132.25 ± 21.49	0.057
Diastolic blood pressure, mm Hg	83.49 ± 13.38	86.30 ± 15.61	80.73 ± 10.18	0.025
LV EDV, mL	124.98 ± 24.77	121.19 ± 27.56	129.11 ± 20.86	0.104
LV ESV, mL	63.35 ± 17.30	60.16 ± 20.07	66.82 ± 13.01	0.046
iLAV, sm	12.30 ± 9.73	18.61 ± 5.71	19.16 ± 4.88	0.412
LV EF, %	46.76 ± 5.91	47.39 ± 6.44	46.12 ± 5.32	0.150
E/e′	14.13 ± 4.47	13.11 ± 4.56	15.37 ± 4.07	0.033
GLS, %	–10.29 ± 2.36	–10.00 ± 2.47	–10.54 ± 2.28	0.416
Total cholesterol, mmol/L	4.72 ± 1.17	4.68 ± 1.18	4.76 ± 1.16	0.750
High density cholesterol, mmol/L	1.04 ± 0.27	1.00 ± 0,20	1.08 ± 0,32	0.144
Low density cholesterol, mmol/L	2.64 ± 1.03	2.54 ± 1,06	2.74 ± 0,99	0.345
Peak troponin I, ng/mL	9.19 ± 4.20	9.92 ± 2.93	7.98 ± 4.02	0.040
NT-proBNP, pg/mL	186.80 ± 27.90	179.50 ± 25.30	194.20 ± 22.70	0.248

Note. BMI, body mass index; T2DM, type 2 diabetes mellitus; MPO, myeloperoxidase; STEMI, ST segment elevation myocardial infarction; GLS, global longitudinal strain, iLAV, index of left atrium volume.

There were not significant differences between both cohorts in age, gender, comorbidities and presentation of CV risk factors apart from obesity. We found that obesity had been determined frequently in patients having MPO ≥ 98.34 ng/mL. In addition, the percentage of visceral fat in these patients was also higher when compared with another. Yet, peak troponin I levels were noticed to be significantly higher in STEMI patients with MPO ≥ 98.34 ng/mL than those who had MPO < 98.34 ng/mL. Diastolic blood pressure were significantly higher and left ventricular end systolic volume was much lower in the patients having MPO ≥ 98.34 ng/mL in comparison with individuals with MPO < 98.34 ng/mL. No significant differences in angiographically culprit lesion of coronary arteries in both patients' cohorts were found (Table 2).

Table 3 illustrates the number of clinical events during in-hospital period out of the ICU and 1-year observation. We found significant differences in clinical events between both cohorts in Killip class > II acute HF ($F = 0.029623$; $\varepsilon^2 = 4.99$; $p = 0.044$) and the total number of combined end-point ($F = 0.064285$; $\varepsilon^2 = 4.29$; $p = 0.046$).

Correlations of circulating levels of biomarkers, body fat accumulation and cardiac hemodynamics performances

We found positive correlations between serum levels of MPO and body fat accumulation ($\tau = 0.28$; $p = 0.036$), NT-proBNP ($\tau = 0.31$; $p = 0.046$), and inverse correlation with diastolic BP ($\tau = –0.14$; $p = 0.038$). There were no significant associations of MPO with other cardiac parameters, such as heart rate, LV EF, GLS, iLAV, LV EDV, and LV ESV.

Table 2. Coronary angiography findings depending on median levels of MPO

Indicator	Entire group (n = 102)	MPO ≥ 98.34 ng/mL (n = 51)	MPO < 98.34 ng/mL (n = 51)	p
STEMI localization				
Anterior	53 (51.9 %)	24 (47.1 %)	29 (56.9 %)	0.322
Posterior	28 (27.5 %)	15 (29.4 %)	13 (25.5 %)	0.657
Other	21 (20.6 %)	12 (23.5 %)	9 (17.6 %)	0.463
Stenotic coronary arteries				
One	26 (25.5 %)	11 (21.6 %)	15 (29.4 %)	0.364
Two and more	65 (63.7 %)	37 (72.5 %)	28 (54.9 %)	0.064
Left artery descending	81 (79.4 %)	39 (76.5 %)	42 (82.4 %)	0.463
Right coronary artery	69 (67.6 %)	32 (62.7 %)	37 (72.5 %)	0.290
Circumflex	43 (42.2 %)	26 (51.0 %)	17 (33.3 %)	0.071
Left main	16 (15.7 %)	5 (9.8 %)	11 (21.6 %)	0.086

Note. STEMI, ST segment elevation myocardial infarction, MPO, myeloperoxidase.

Table 3. Clinical events depending on median myeloperoxidase level

Indicator	Entire group (n = 102)	MPO ≥ 98.34 ng/mL (n = 51)	MPO < 98.34 ng/mL (n = 51)	p
During period of hospitalization				
The total number of complications	23 (9.8 %)	8 (15.7 %)	15 (29.4 %)	0.155
Killip class > II acute HF	9 (0.98 %)	2 (3.9 %)	9 (17.6 %)	0.044
Life threatening cardiac rhythm and conduction abnormalities	14 (13.7 %)	6 (11.8 %)	8 (15.7 %)	0.774
1-year events				
Chronic HF	17 (16.7 %)	8 (15.7 %)	9 (17.6 %)	0.791
Rhythm and conduction abnormalities	1 (0.98 %)	0	1 (1.96 %)	0.500
Repeated MI	4 (3.9 %)	2 (3.9 %)	2 (3.9 %)	0.691
Death	3 (2.9 %)	0	3 (5.9 %)	0.121
Combined end-point	27 (26.4 %)	10 (19.6 %)	17 (33.3 %)	0.046

Note. MI, myocardial infarction, STEMI, ST-segment elevation myocardial infarction; HF, heart failure.

Table 4. Predictors for1-year clinical evens after STEMI. Depending variable: combined end point

Variable	Univariate linear regression				Multivariate linear regression			
	β-coefficient	OR	95 % CI	p	β-coefficient	OR	95 % CI	p
Body fat accumulation	0.42675	1.5323	1.0012—2.3686	0.0500	0.27237	1.3131	0.9832—1.7536	0.0650
BMI > 30 kg/m^2	0.31819	0.7275	0.3562—1.4856	0.3824	–	–	–	–
Multivessel injury	0.24324	1.2754	0.1241—13.1019	0.8378	–	–	–	–
E/e′	0.12950	1.1383	0.7005—1.8495	0.6011	–	–	–	–
MPO	0.15541	1.0157	1.0022—1.0373	0.0448	0.012745	1.0128	1.0068—1.0231	0.0176
GLS, %	1.47756	4.3822	1.1660—16.4695	0.0287	1.26363	3.5383	1.3264—9.4388	0.0116
Peak troponin I	0.083705	13.1759	0.1729—10.1924	0.7860	–	–	–	–
Hypercholesterolemia	2.57839	0.8848	0.6638—1.1255	0.2435	–	–	–	–

Note. BMI, body mass index; GLS, global longitudinal strain; MPO, myeloperoxidase; STEMI, ST segment elevation myocardial infarction.

Univariate and multivariate linear regression analysis

Univariate linear regression shows that body fat accumulation had a borderline impact on the depending variable, whereas global longitudinal strain (GLS) and MPO demonstrated significant influence on combined end-point (Table 4).

Using multivariate linear regression, we noticed that the only GLS and MPO remained independent predictors for this depending variable.

ROC curve analysis

ROC curve analysis has shown that all three predictive models based on body fat accumulation (AUC = 0.908. $p < 0.0001$), MPO (AUC = 0.721. $p < 0.0049$) and GLS (AUC = 0.925; $p < 0.0001$) significantly distinguished from the basal model and allowed to identify the patients at risk of clinical outcomes in STEMI patients after successful primary PCI (Fig. 2—4).

Comparison of the predictive models

We compared predictive values of the models using maximum likelihood ratio test and found that GLS was the best predictor for poor clinical outcomes in STEMI patients, whereas MPO, on contrary, had the highest negative likelihood ratio amongst others (Table 5).

Body fat accumulation seems to show borderline significance when compared with GLS, while it was sufficiently better than MPO.

The results of the study has revealed that circulating levels of MPO and GLS were the most powerful biomarkers for combined clinical outcomes in STEMI patients who had successful reperfusion of TIMI > II, whereas body fat accumulation did not demonstrate significant influence on combined end-point in multivariate regression. Therefore, we did not confirm that elevated levels of MPO were associated with by far more profound post-STEMI adverse cardiac remodeling than lower ones. Yet, body fat accumulation, on contrary of our previous expectations, has demonstrated a borderline significance

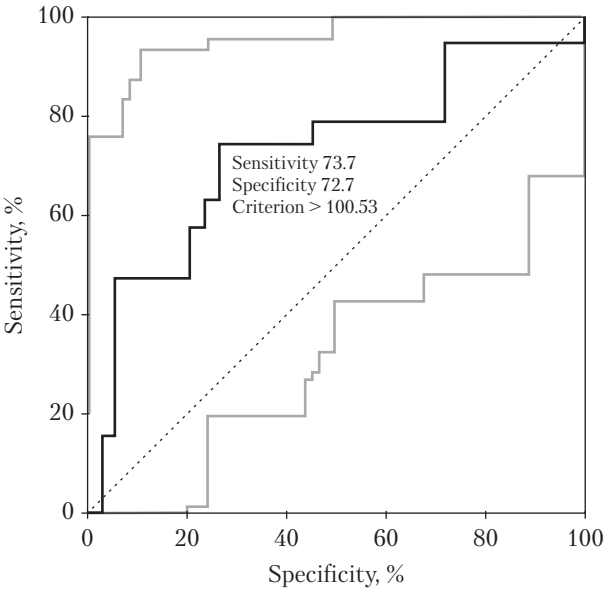

Figure 3. Predictive model of clinical outcomes in STEMI patients after successful primary PCI based on myeloperoxidase

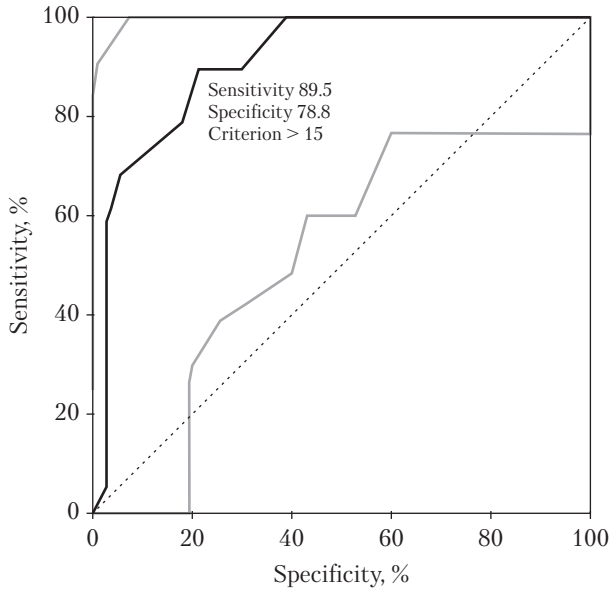

Figure 2. Predictive model of clinical outcomes in STEMI patients after successful primary PCI based on body fat accumulation

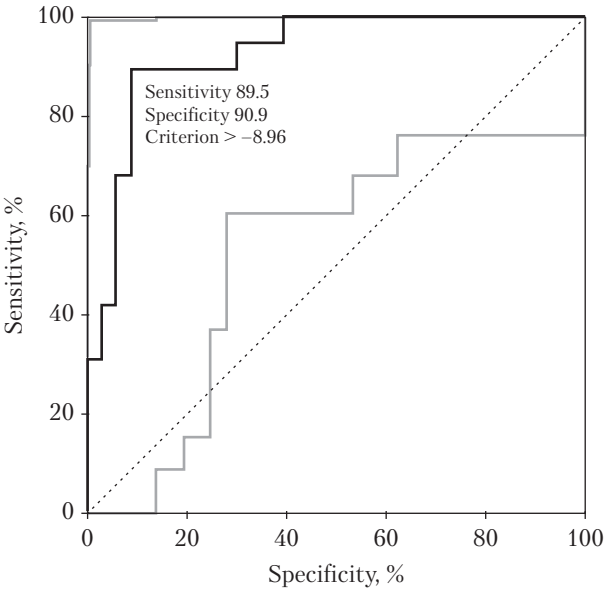

Figure 4. Predictive model of clinical outcomes in STEMI patients after successful primary PCI based on global longitudinal strain

Table 5. Predictive values of the models

Variable	AUC	95 % CI	Sensitivity, %	Specificity, %	LR(+)	LR(-)	p
MPO	0.721	0.579—0.836	73.7	72.7	1.08	0.43	0.046
Body fat accumulation	0.908	0.795—0.971	89.5	78.8	4.22	0.13	0.044
GLS	0.925	0.817—0.980	89.5	90.9	9.84	0.12	0.002

Note. LR(+) positive likelihood ratio; LR(–), negative likelihood ratio; MPO, myeloperoxidase; GLS, global longitudinal strain.
Significance value was computed with the likelihood ratio test.

for cumulative clinical outcomes. These findings are not a common sight on inflammatory biomarkers including MPO, which had previously revealed a solid relation to severe microvascular obstruction, severity of atherosclerosis, number of culprit lesions, and myocardial infarct size [51, 55]. Perhaps, there was a sufficient difference between the patients' study populations. Indeed, the studies mentioned above have been enrolled STEMI patients with microvascular obstruction and post-PCI TIMI—II, whereas our investigation was based on an enrollment of STEMI patients with complete reperfusion. However, MPO remaining its predictive ability for post-PCI MACEs [7, 52] have been found as having a strong discriminative potency for in-hospital mortality and fatal MI on long-term follow-up [9, 52]. Moreover, there was solid evidence regarding the fact that MPO was independently associated with a nearly two-fold higher risk of non-fatal MI and recurrent ACS [19, 30, 44].

We also hypothesized that MPO being activated in adipose tissue resulting its inflammation contributes a clinical course of post-PCI STEMI patients. Indeed, positive association between the levels of MPO and body fat accumulation, as well as evidence of predictive ability of body fat accumulation for 1-year cumulative clinical outcomes confirm this supposition. In addition, these findings obtained by us in the investigation well correspond to the results of other studies [21]. We also incline toward an idea that MPO might incorporate in a risk stratification with the aim of developing a guide therapy of post-PCI STEMI patients. Although there was a hypothesis toward MPO may be significantly better predictor than NT-proBNP level in STEMI patients regardless of T2DM [18], MPO exerted its independent predictive potency on prognosis much better in T2DM patients than in non-T2DM individuals [37]. We did not find a strict confirmation of this assumption in the study, in spite of having numerous data received in animal models and clinical observations and clarifying that MPO potentiates visceral adipose tissue inflammation in T2DM. On the other hand, a reduction of circulating levels of MPO with AZM198 have not improved cardiac function and reduced cardiac hypertrophy and fibrosis, whereas the levels of lipid peroxidation biomarkers were found to be significantly attenuated [53].

Thus, MPO seems not to be a factor directly influenced MACE. We suggested that the effect of MPO maybe mediated by impaired relaxation of myocardium and supported by body fat accumulation. Indeed, the univariate and multivariate regression analysis have revealed that this assumption had confirmed. However, there is a challenging speculation whether MPO and obesity might improve conventional STEMI predictive scores. In fact, add-on MPO to the GRACE score enables more accurate prediction of cardiovascular events compared with GRACE risc score alone in patients with STEMI, whereas obesity did not improve the score [26, 29]. At the same time, epicardial adipose tissue thickness rather total body fat composition showed independent prediction for no-reflow phenomenon and CV mortality in STEMI patients [10, 54].

Finally, we received the data that GLS was the most important predictor for MACEs in post-PCI STEMI patients, but lowered levels of MPO may be more accurate for favorable prognosis after successful PCI. Indeed, GLS can predict remodeling and adverse clinical events in STEMI patients [41, 42] and it exhibited the superiority before LV EF and a size of MI [42, 49]. Although numerous studies have shown no predictive ability of GLS on adverse cardiac remodelling following STEMI in patients with normal or near normal LV EF [16, 35, 47], there are several alternative findings [1, 6, 13, 14, 22]. We suggest that GLS as a powerful echocardiographic parameter related to development of post-PCI adverse cardiac remodeling seems to be combined with MPO measure and body fat accumulation assessment to significantly improve conventional risk models in successfully reperfused STEMI patients. This requires a large clinical investigation in the future.

Study limitation

This study has several limitations. First, a small sample size and single center design. Indeed, more results from large clinical studies need to obtain more information regarding MPO as a predictor

for clinical outcomes. However, we organized the study so that patients were transported to the ICU without a delay and all treatment procedures would performed according to actual local requirements. Secondary, COVID-19 pandemic requires performing PCR COVID-19 tests prior hospital admission that potentially declined a number of eligible patients. We minimized a variety of the data received by strong logistic chain during patients' transfer. Finally, we suggest that the data can be extrapolate to other patients with STEMI and allow us to interpret the findings as they are.

Conclusions

GLS was the best predictor for 1-year combined clinical outcomes in STEMI patients who were undergone successful primary PCI. Body fat accumulation seems to show borderline significance when compared with GLS, while it was sufficiently better than MPO.

The study is a fragment of the research project: «To study the biochemical, genetic mechanisms of reperfusion damage of the myocardium and to assess the cardioprotective effect of antiplatelet therapy in acute myocardial infarction», State Registration No. 0117U003028/Ukraine

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Мієлопероксидаза та глобальний повздовжній стрейн у прогнозуванні клінічних подій після гострого інфаркту міокарда з елевацією сегмента ST

Підвищені рівні деяких запальних цитокінів та хемокінів, зокрема мієлопероксидази (МПО), і такі характеристики ушкодженого міокарда, як його деформація (глобальний повздовжній стрейн (ГПС)) є перспективними біомаркерами для гострого коронарного синдрому, але його предикторна здатність для наступних клінічних подій у пацієнтів з гострим інфарктом міокарда (ГІМ) з елевацією сегмента ST та ожирінням не визначена.

Мета — визначити вміст МПО та ГПС, які можуть бути предикторами комбінованої кінцевої точки протягом 1 року після ГІМ з елевацією сегмента ST у пацієнтів, яких успішно пролікували з використанням черезшкірного коронарного втручання (ЧКВ), залежно від наявності абдомінального ожиріння.

Матеріали та методи. Проспективно залучили у дослідження 102 пацієнти з ГІМ з елевацією сегмента ST, яким було проведено успішне ЧКВ. Усі пацієнти надали добровільну згоду на участь у дослідженні. При надходженні до стаціонару забирали кров для дослідження біомаркерів (МПО, кардіальний тропонін) та проводили ехокардіографію з визначенням допплер-параметрів (ГПС).

Результати. Рівень МПО в загальній групі пацієнтів становив 129,45 [48,48—179,70] нг/мл. Загальну групу розподілили на дві когорти залежно від рівня МПО за медіаною (≥ 98,34 нг/мл та < 98,34 нг/мл). Досліджували 26 комбінованих кінцевих точок (10 у пацієнтів з МПО і 98,34 нг/мл та 17 у пацієнтів з МПО < 98,34 нг/мл ($F = 0{,}064285$; $\varepsilon^2 = 4{,}29$; $p = 0{,}046$). Мультиваріантний лінійний регресійний аналіз показав, що лише ГПС та МПО були незалежними предикторами клінічних подій.

Висновки. Глобальний повздовжній стрейн був найкращим предиктором щодо виникнення комбінованої кінцевої точки протягом 1 року після ГІМ з елевацією сегмента ST у пацієнтів, яким проведене успішне первинне ЧКВ. Акумуляція вісцерального жиру мала граничне значення порівняно з ГПС, але була значно кращим предиктором, ніж МПО.

Ключові слова: гострий інфаркт міокарда з елевацією сегмента ST, мієлопероксидаза, акумуляція вісцерального жиру, глобальний повздовжній стрейн, прогноз.

ДЛЯ ЦИТУВАННЯ

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