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Original Scientific Paper

NOMOGRAM CONTAINING SIMPLE ROUTINE CLINICAL AND BIOCHEMICAL PARAMETERS CAN PREDICT PATHOLOGIC VENTRICULAR REMODELING IN STEMI PATIENTS

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SUMMARY – Heart failure is the leading cause of morbidity and mortality worldwide, with ischemic heart disease being one of the most important etiologic factors. Heart failure develops due to ventricular remodeling, which leads to increases in left ventricular end-systolic and end-diastolic volumes. In this prospective observational study, we included 101 patients with first episode of ST-segment elevation myocardial infarction in whom percutaneous coronary intervention was conducted within 12 h and Thrombolysis in Myocardial Infarction III flow was achieved. The aim was to determine which clinical and biochemical parameters can help predict pathologic ventricular remodeling 1 year after myocardial infarction. We created a nomogram based on routinely used blood tests and vital parameters which showed highest correlation with pathologic ventricular remodeling. The nomogram included NTproBNP value 12 h after reperfusion, aspartate transaminase value 12 h after reperfusion, systolic blood pressure value on admission, and culprit coronary artery. We performed ROC analysis which yielded great predictive value of the nomogram. The area under curve was 0.907 (95% CI 0.842-0.973). The nomogram value of -3.54 had 91.4% sensitivity and 74.0% specificity. We believe that this nomogram, once validated, could offer a widely available, low-cost option that would help identify patients at risk of developing pathologic left ventricular remodeling and achieve this at a very early stage of myocardial infarction (12 h after reperfusion has been achieved).

Key words: *Acute myocardial infarction; Left ventricular remodeling; End-diastolic volume; End-systolic volume*

Introduction

Heart failure is the leading cause of morbidity and mortality worldwide. Atherosclerotic coronary artery disease is the most important promoting factor of myocardial infarction. Due to advances in treatment, the number of patients who survive myocardial infarction is continually increasing, some of whom eventually develop heart failure.

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Development of heart failure occurs due to ventricular remodeling, which includes structural, functional, cellular, and molecular changes involving cardiac myocytes and the interstitial collagen matrix. The processes described are initially compensatory and driven to preserve cardiac output. Pathologic remodeling may occur with pressure overload or volume overload, or following cardiac injury (e.g., myocardial infarction). Following myocardial infarction, remodeling leads to ventricular dilatation and increased oxygen demand[1,2]. As dilatation progresses, it leads to further hemodynamic consequences. Consequent alterations in geometry lead to mitral regurgitation[3], which also contributes to the development of ischemic cardiomy-

opathy. The changes in left ventricular geometry and function described above take place a couple of weeks to months after myocardial infarction. Even though the mechanism of pathologic left ventricular remodeling is somewhat understood, there is no apparent, clear definition of left ventricular remodeling. In a study performed by White *et al*. using contrast ventriculography[4], it was found that left ventricular end systolic volume (LVESV) has a greater predictive value in patient survival compared to end diastolic volume (EDV) and ejection fraction (EF). In another study, which included patients with myocardial infarction who underwent primary percutaneous coronary intervention (pPCI), pathologic ventricular remodeling was defined as a 20% increase in EDV during follow up. The study found the increase in EDV to lead to higher morbidity and mortality[5]. In a study performed by Yoshida *et al*., a model was created showing that LVESV index (LVESVI) directly caused increases in plasma B-type natriuretic peptide (BNP) levels and that LVEDVI adaptably reduced plasma BNP levels[6].

Conclusion that can be derived from the studies, irrespective of the definition used for pathologic ventricular remodeling, is that increase in the left ventricular volume is correlated to worse clinical outcomes[2].

Multiple therapeutic targets exist to stop adverse remodeling and promote reverse remodeling, including early revascularization, optimal medical therapy with neurohormonal antagonists, and cardiac resynchronization therapy in appropriately selected patients.

The aim of this study was to find which parameters could predict the risk of pathologic left ventricular remodeling in the early stage of myocardial infarction.

Materials and Methods

Study population

The study included patients aged 18-79 years diagnosed with first episode of ST-elevation acute myocardial infarction (STEMI) in whom PCI was performed within 12 hours of the onset of chest pain, with Thrombolysis in Myocardial Infarction III (TIMI III) flow achieved in final coronarogram. The inclusion period was from July 1, 2017 to March 1, 2018. We excluded patients with known cardiomyopathy (regardless of etiology), chronic renal insufficiency stage IV and V, and intolerance of lipophilic angiotensin converting enzyme inhibitors (ACEI) or angiotensin receptor blocker (ARB) or beta blockers. Additionally, patients who had reinfarction within the follow up period were excluded. All included patients received standard therapy for myocardial infarction (ACEI/ARB, beta-blocker, statin, acetylsalicylic acid, clopidogrel/ticagrelor). Acute myocardial infarction was defined according to the European Society of Cardiology guidelines[7]. The study was approved and overseen by the hospital Ethics Committee. The study was performed in accordance with the guidelines set in Declaration of Helsinki and its amendments or comparable ethical standards. All patients were informed about study details by one of the authors and signed a written consent form which was approved and reviewed by the institutional Ethics Committee.

Biochemical and biomarker analysis

All blood tests and vital parameters were collected at baseline, then at 6 and 12 hours following PCI. N-terminal pro-B-type natriuretic peptide (NTproBNP) was also collected at 1 year following PCI.

Echocardiography

Transthoracic echocardiography was performed within 5 days of myocardial infarction and 1 year later. At 1 year follow up, cardiac function and chamber size assessment was performed using Simpson triplane method. Pathologic ventricular remodeling was defined as left ventricular end-diastolic volume index (EDVi) >79 mL/m^2 for men and EDVi >71 mL/m^2 for women, and/or left ventricular end-systolic volume index (ESVI) >32 mL/m^2 for men and ESVI >28 mL/m^2 for women, according to the normal reference ranges for cardiac chamber size from the NORRE study[8].

Coronary angiography

Coronary angiography was performed within 12 hours from the onset of chest pain. All procedures were performed with transradial approach. Patients in whom TIMI III was achieved (as *per* operator assessment) were included in the study. Only culprit lesion was revascularized during pPCI, whereas other major lesions (evaluated by the operator who performed pPCI) were revascularized within 3 months.

Statistical analysis

Final analysis was performed on 101 patients who had full biochemical, bioassay and echocardiography parameters completed (out of 160 patients included during the study period, 28 patients had suboptimal echocardiography imaging, which prevented appropriate Simpson triplane analysis; 19 patients had incomplete blood tests or these were not taken within the set time frame; three patients had myocardial reinfarction during follow up; one patient died during follow up period; and eight patients failed to present for scheduled follow up examination).

The covariates included were age, systolic blood pressure on admission, diastolic blood pressure on admission, Killip class on admission, heart rate on admission, culprit coronary artery, number of arteries with significant stenosis (defined as >70%), SYNTAX score, pain to balloon time, diabetes, weight, body mass index, dyslipidemia, smoking status, previous medical therapy, medical therapy at discharge, hypertension, prior cerebrovascular disease, creatine kinase (CK) level at admission, 6 h post PCI and 12 h post PCI; high-sensitivity troponin I (hsTnI) at admission, 6 h post PCI and 12 h post PCI; aspartate transaminase (AST) level at admission, 6 h post PCI and 12 h post PCI; alanine transaminase (ALT) level at admission, 6 h post PCI and 12 h post PCI; lactate dehydrogenase (LDH) level at admission, 6 h post PCI and 12 h post PCI; glucose plasma level at admission, 6 h post PCI and 12 h post PCI; hemoglobin A1c during hospital stay; and NT-proBNP level at admission, 6 h post PCI and 12 h post PCI, and 1 year after myocardial infarction.

Descriptive statistics was initially performed. Data distribution was analyzed by Kolmogorov-Smirnov test. Most of the variables did not follow normal distribution. Continuous variables were expressed as mean and standard deviation, but were analyzed by nonparametric tests. Independent samples were compared by Kruskal-Wallis or Mann-Whitney test. Dependent continuous variables were analyzed by Wilcoxon test. Categorical variables were compared by χ^2-test with Yates correction. Correlation between individual parameters was analyzed by Spearman correlation and binary logistic regression. Binary logistic regression was also used to analyze the interrelationship of the variables and to correct the correlation for disruptive factors. When performing multivariate analysis, a model of backward stepwise conditional regression was used. The aim of this model was to find out which independent variables were independently related to the dependent variable, and the same was used in all forms of regression. ESVI was used as a dependent variable on multivariate analysis, and a maximum of 6 variables were used in each model, which is appropriate to the sample size. Only variables that showed a statistically significant difference on univariate analysis were included in multivariate analysis. Since variables that did not follow normal distribution were used in multivariate analysis, all analyses were repeated on logarithmically transformed data. Since the same results were obtained, only the results obtained by the analysis of 'raw' data are shown. In order to relate the predictive value of each parameter that proved to be an independent predictive factor, a regression equation composed of non-standardized coefficients of multivariate analysis for each individual predictive factor was constructed. Finally, the reciever operating characteristic (ROC) analysis was used to analyze the predictive value of an individual parameter, or regression equation in the prediction of elevated ESVI.

Statistical analysis was performed with SPSS software version 20.0. All tests were considered statistically significant if $p<0.05$.

Results

Statistical analysis was performed on 101 patients who had complete clinical, biochemical, biomarker and echocardiographic parameters. In our study, all patients who had ESVI elevated, also had EDVI elevated, therefore tables show only variable relation to ESVI.

Univariate analysis

Univariate analysis of continuous variables (Table 1) showed good correlation of pathologic remodeling (in our study defined by enlarged ESVI and EDVI) with higher levels of several biochemical markers that depict the area of infarction (hsTnI, CK, AST, ALT, LDH) 12 hours after the PCI. Especially strong correlation was found with the 'old' biochemical markers of CK, AST, ALT and LDH, the levels of which 6 hours after the PCI also had statistically significant

Table 1. Results of univariate analysis of continuous variables

	ESVI				p
	Normal		Elevated		
	Mean	SD	Mean	SD	
0 h Troponin I (ng/L)	1872	5990	1006	2411	0.581
6 h Troponin I (ng/L)	27462	20516	35685	21277	0.053
12 h Troponin I (ng/L)	32187	19128	42054	16445	0.008
0 h CK (U/L)	227	328	240	217	0.228
6 h CK (U/L)	1414	1166	2727	2167	0.003
12 h CK (U/L)	1375	941	2830	1769	<0.001
0 h ALT (U/L)	26	13	32	22	0.066
6 h ALT (U/L)	39	21	61	37	0.001
12 h ALT (U/L)	41	21	71	36	<0.001
0 h AST (U/L)	32	24	36	35	0.307
6 h AST (U/L)	128	95	245	184	0.002
12 h AST (U/L)	139	91	283	165	<0.001
0 h BG (mmol/L)	8.5	3.4	9.6	6.1	0.376
6 h BG (mmol/L)	7.5	2.8	9.3	5.7	0.232
12 h BG (mmol/L)	7.1	2.9	9.0	4.7	0.042
0 h LDH (U/L)	224	85	232	74	0.410
6h LDH (U/L)	413	224	651	442	0.018
12 h LDH (U/L)	463	247	848	474	<0.001
HbA1c (%)	5.9	1.2	6.0	1.2	0.948
Age (years)	63	10	61	11	0.437
Systolic blood pressure on admission (mm Hg)	134	23	124	20	0.043
Diastolic blood pressure on admission (mm Hg)	79	11	78	12	0.703
Frequency on admission (beats/min)	73	12	80	17	0.014
Pain to balloon (min)	283	168	291	171	0.576
SYNTAX	12.4	8.2	18.1	7.4	<0.001
Body surface area (m^2)	2.0	.2	2.0	.2	0.947
NTproBNP (0 h) (pmol/L)	27.8	57.5	70.5	76.6	0.013
NTproBNP (12 h) (pmol/L)	49.3	71.1	78.8	55.1	0.032
NTproBNP (1 year) (pmol/L)	18.8	25.4	65.7	81.3	0.002

ESVI = end-systolic volume index; SD = standard deviation; CK = creatine kinase; ALT = alanine transaminase; AST = aspartate transaminase; BG = blood glucose; LDH = lactate dehydrogenase; NTproBNP = N-terminal pro-B-type natriuretic peptide

correlation with pathologic remodeling. The lower sensitivity of hsTnI l can perhaps be explained by the assay used (Abbott Architect, Abbott Park, Illinois, USA), where the maximum level that can be measured is 50000 ng/L, which limited the hsTnI discriminatory value in mid-sized/large myocardial infarctions. In the clinical variable category, age was not found to have any correlation with pathologic remodeling. Also, somewhat surprising, the pain to balloon time (representing total ischemia time) was not found to correlate with development of pathologic ventricular remodeling. SYNTAX score, systolic blood pressure and heart frequency at admission also showed correlation with remodeling.

Univariate analysis of categorical variables is shown in Table 2. We found good correlation of pathologic ventricular remodeling with patient sex (male sex being the one at risk) and Killip class. Also, left anterior

Table 2. Results of univariate analysis of categorical variables

		ESVI				p
		Normal		Elevated		
		n	%	n	%	
Killip class	1	60	100.0	34	85.0	
	2	0	0.0	5	12.5	
	4	0	0.0	1	2.5	0.008
Heart rhythm	Nodal/AV block	0	0.0	2	5.0	
	Atrial fibrillation	3	5.0	3	7.5	
	Sinus rhythm	57	95.0	35	87.5	0.183
Coronary artery	RCA	26	43.3	9	22.5	
	ACx	16	26.7	2	5.0	
	LAD	18	30.0	29	72.5	<0.001
Number of diseased arteries	1	33	55.0	19	47.5	
	2	18	30.0	11	27.5	
	3	9	15.0	10	25.0	0.455
Sex	Female	24	39.3	6	15.0	
	Male	37	60.7	34	85.0	0.014
Arterial hypertension	No	24	39.3	13	32.5	
	Yes	37	60.7	27	67.5	0.532
Diabetes mellitus	No	54	88.5	29	72.5	
	Yes	7	11.5	11	27.5	0.061
Dyslipidemia	No	27	44.3	24	60.0	
	Yes	34	55.7	16	40.0	0.155
Smoking	No	29	47.5	23	57.5	
	Yes	32	52.5	17	42.5	0.416

ESVI = end-systolic volume index; SD = standard deviation; RCA = right coronary artery; ACx = circumflex artery; LAD = left anterior descending artery

descending artery (LAD) as a culprit coronary artery in myocardial infarction was found to be a predictor with strong statistical correlation to pathologic ventricular remodeling.

Only variables that showed a statistically significant difference on univariate analysis were included in multivariate analysis (Tables 3 and 4).

Multivariate analysis

In the first models of multivariate analysis, clinical, anthropometric and anatomic parameters were used (Table 3). Model 1 included frequency on admission, systolic blood pressure on admission, and patient sex. All three parameters were shown to be independent predictors of remodeling. In model 2, SYNTAX score was added to the 3 prior parameters; analysis showed only frequency (higher value) and SYNTAX score to be independent predictors. In model 3, culprit coronary artery was added as an additional parameter to the 3 parameters used in model 1, and this model showed systolic blood pressure (lower value) and culprit coronary artery (LAD as culprit) to be independent predictors of remodeling. Model 4 included the 3 original parameters from model 1, as well as parameters from models 2 and 3, which showed only culprit coronary artery to be an independent predictor. Model 5 as the last model presented in Table 3 included 2 parameters for which previous analyses revealed to yield the greatest independent prediction, i.e. SYNTAX and culprit coronary artery, and showed that

Table 3. Multivariate analysis of clinical, anthropometric and anatomic parameters that showed a statistically significant difference in univariate analysis (except for Killip class due to unfavorable distribution)

	B	SE	p	OR	95% CI	
Model 1 (sex, SBP, HR)						
Male sex	1.179	0.549	0.032	3.252	1.109	9.534
SBP	-0.026	0.012	0.027	0.974	0.952	0.997
HR	0.043	0.017	0.010	1.044	1.010	1.078
Model 2 (model 1 + SYNTAX)						
HR	0.039	0.017	0.022	1.040	1.006	1.075
SYNTAX	0.068	0.030	0.025	1.070	1.009	1.135
Model 3 (model 1 + artery)						
SBP (mm Hg)	-0.024	0.012	0.039	0.976	0.954	0.999
Coronary artery						
RCA *vs*. LAD	-1.482	0.513	0.004	0.227	0.083	0.621
ACx *vs*. LAD	-2.390	0.820	0.004	0.092	0.018	0.457
Model 4 (model 1 + SYNTAX + artery)						
Coronary artery			0.011			
RCA *vs*. LAD	-1.256	0.531	0.018	0.285	0.101	0.806
ACx *vs*. LAD	-2.006	0.845	0.018	0.135	0.026	0.705
Model 5 (SYNTAX + artery)						
Coronary artery						
RCA *vs*. LAD	-1.248	0.515	0.015	0.287	0.105	0.789
ACx *vs*. LAD	-2.051	0.832	0.014	0.129	0.025	0.657
SYNTAX	0.063	0.029	0.032	1.065	1.005	1.128

B = non-standardized coefficient; SE = standard error; OR = odds ratio; CI = confidence interval; SBP = systolic blood pressure; HR = heart rate; RCA = right coronary artery; ACx = circumflex artery; LAD = left anterior descending artery

both of them were independent predictors of pathologic ventricular remodeling (higher SYNTAX score and LAD as culprit artery).

Table 4 shows multivariate analysis of biochemical parameters and biomarker (NTproBNP). Model 6 included NTproBNP, hsTnI, CK, AST, ALT and LDH (levels measured 12 hours after reperfusion), and showed NTproBNP and AST to be the variables that had independent correlation to remodeling. Model 7 was created by including 2 variables showing independent prediction and adding values from model 1 (systolic blood pressure on admission, heart frequency on admission, and patient sex) and found NTproBNP, AST level 12 hours after reperfusion and systolic blood pressure to have statistical correlation as independent predictors.

Then, the next model 8 was formed by adding SYNTAX score to the variables analyzed in model 7; again, NTproBNP and AST (12 h after reperfusion) and systolic blood pressure on admission were found to be independent predictors. The last model 9 was made by using the biochemical parameters and biomarker used in model 6 and analyzing them with SYNTAX score and culprit coronary artery. Model 9 showed NTproBNP and AST values measured 12 hours after reperfusion to be independent predictors of pathologic remodeling, as well as systolic blood pressure on admission and culprit coronary artery (Table 4).

In order to unify the predictive value of each parameter derived from multivariate analysis, a regression equation was made using non-standardized coefficients for each parameter:
(NTproBNP_12 × 0.017 + AST_12 × 0.008 – SBP × 0.035) – 2.005 (for RCA) or – 4.949 (for ACx)

We performed ROC analysis which found great predictive value of the nomogram. Area under the

Table 4. Multivariate analysis of laboratory parameters that had strongest predictive value in univariate analysis and their combination with previous clinical, anthropometric and anatomic parameters

	B	SE	p	OR	95% CI	
Model 6 (NTproBNP, hsTnI, CK, AST, ALT, LDH 12 hours after reperfusion)						
NTproBNP_12	0.011	0.005	0.023	1.011	1.001	1.020
AST_12	0.009	0.002	<0.001	1.009	1.004	1.013
Model 7 (NTproBNP_12, AST_12 + sex, SBP, HR)						
NTproBNP_12	0.012	0.005	0.020	1.012	1.002	1.021
AST_12	0.007	0.002	0.002	1.007	1.003	1.012
SBP	-0.033	0.016	0.043	0.967	0.937	0.999
Model 8 (model 7 + SYNTAX)						
NTproBNP_12	0.012	0.005	0.021	1.012	1.002	1.023
AST_12	0.007	0.002	0.005	1.007	1.002	1.011
SBP	-0.034	0.016	0.039	0.966	0.936	0.998
Model 9 (model 6 + SYNTAX + artery)						
NTproBNP_12	0.017	0.006	0.002	1.018	1.006	1.029
AST_12	0.008	0.003	0.007	1.008	1.002	1.014
SBP	-0.035	0.018	0.047	0.965	0.932	1.000
Coronary artery						
RCA *vs.* LAD	-2.005	0.705	0.004	0.135	0.034	0.536
ACx *vs.* LAD	-4.949	1.876	0.008	0.007	<0.001	0.280

B = non-standardized coefficient; SE = standard error; OR = odds ratio; CI = confidence interval; SBP = systolic blood pressure; HR = heart rate; NTproBNP = N-terminal pro-B-type natriuretic peptide; hsTnI = high-sensitivity troponin I; CK = creatine kinase; AST = aspartate transaminase; ALT = alanine transaminase; LDH = lactate dehydrogenase; RCA = right coronary artery; ACx = circumflex artery; LAD = left anterior descending artery

curve (AUC) was 0.907 (95% CI 0.842-0.973). Nomogram value of -3.54 had 91.4% sensitivity and 74.0% specificity (Fig. 1).

Discussion

The nomogram we created is based on routinely used blood tests and vital parameters which showed highest correlation with pathologic ventricular remodeling. It included NTproBNP value 12 hours after reperfusion, AST value 12 hours after reperfusion, systolic blood pressure value on admission, and culprit coronary artery. ROC analysis found great predictive value of the nomogram.

The NTproBNP value is considered to be the most valuable and reliable biomarker for diagnosing heart failure and cardiac dysfunction. It is released to the bloodstream by myocytes undergoing wall tension due to volumetric or pressure overload[9]. It is well known

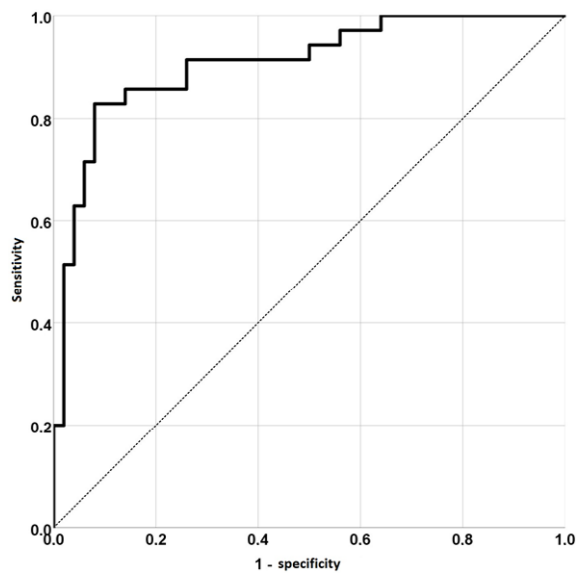

Fig. 1. ROC curve showing diagnostic accuracy of the nomogram in predicting increased end-systolic volume index.

that acute ischemic heart disease is associated with elevation of BNP levels, which might reflect the severity of LV dysfunction[10]. NTproBNP together with systolic blood pressure on admission could represent the Killip classification system, which is using physical examination and development of heart failure in order to predict and stratify the risk of mortality after acute myocardial infarction. AST and ALT elevations are common in myocardial infarction. Both markers are correlated with CK-MB area under curve but independently associated with worse mortality and clinical outcomes[11]. Perhaps the biggest surprise in our nomogram was better correlation of pathologic ventricular remodeling present 1 year after myocardial infarction with AST than with CK (when CK was included instead of AST, the predictive value dropped slightly, to approximately 88%). This can be attributed to statistical 'glitch' due to small patient sample, but we believe that, since AST proved to be elevated after ischemic cell death of several other tissues including kidney, skeletal muscle and brain, better correlation of AST was due to it, being a marker of ischemic end organ damage in acute coronary syndrome rather than a marker of myocardial lesion[12-16]. It is also noted that myocardial infarction affecting LAD coronary artery is associated with the highest risk of adverse clinical outcomes because of the large amount of myocardial territory supplied by the LAD compared to other coronary arteries[17].

All the parameters of the nomogram are routinely used and they add no extra cost. Their correlation has great diagnostic accuracy in predicting left ventricular remodeling.

In our study, we used absolute values of ESVI and EDVI to define pathologic ventricular remodeling. A prospective study conducted by Farah *et al.* included 66 patients with anterior infarction, with the aim to analyze left ventricular remodeling at 6-month follow up. Ventricular remodeling was defined as a 10% increase in end-systolic or end-diastolic diameter[18]. In that study, 58% of patients presented with ventricular remodeling, which is more than in our study (39.6%). It can be explained by the fact that the above-mentioned study included only patients with anterior myocardial infarction, who are at the highest risk of developing pathologic ventricular remodeling. When analyzing the subgroup of patients with anterior infarction from our study, the results are similar (61.7% of patients with anterior infarction developed pathologic ventricular remodeling), despite the fact that the same criteria were not used to define pathologic ventricular remodeling. In a study by Zaliaduonyte-Peksiene *et al.*, remodeling was defined as 20% increase in EDV within 6 months of myocardial infarction[19]. The prevalence of pathologic ventricular remodeling was 34.7%, which is similar to our results. Another study assessed galectin 3 plasma levels in STEMI patients as a predictor of remodeling, defining remodeling as 20% increase in ESV from baseline in a 6-month period. Using these criteria, the incidence of remodeling was 38.6%, which is very similair to our study[20]. Also, in a study by Park *et al.*, which included 50 patients with anterior-wall acute myocardial infarction to test whether longitudinal strain could be a useful predictor of left ventricular remodeling after reperfusion therapy in acute myocardial infarction, a 15% increase in end-diastolic diameter was used to define pathologic remodeling[21]. However, the prevalence of left ventricular remodeling was similar to that observed in our study. Thus, it can be concluded that the use of an absolute value (EDVI >79 mL/m^2 for men and EDVI >71 mL/m^2 for women, and/or ESVI >32 mL/m^2 for men and ESVI >28 mL/m^2 for women) is equally valuable and sensitive for assessing the occurrence of pathologic ventricular remodeling, as well as the use of dynamic value (increase in EDV at follow up). In our study, all patients with an increased ESVI also had an increased EDVI, suggesting that ESVI is a better parameter for predicting pathologic ventricular remodeling compared to EDVI, which is supported in other studies. Saito *et al.* analyzed data on 266 STEMI patients who underwent successful percutaneous coronary intervention[22]. Patients were divided into 4 groups according to LVEDP and LVEF value (LVEDP ≥21 mm Hg and LVEF ≥55%). The study found a statistically significant difference in ESVI in the group with elevated LVEDP and decreased EF compared to the group with normal LVEDP and decreased LVEF. Patients in the group with elevated LVEDP had an increased risk of major adverse cardiovascular events and re-hospitalization, emphasizing the value and importance of ESVI in the prognosis of patients with myocardial infarction.

Limitations of our study were several, the biggest one being a relatively small patient sample from a single centre. Also, since coronary disease severity assessment was performed by the operator performing

pPCI, there certanly was a degree of intraobserver variance in the assessment of coronary artery disease in our patients.

In a recent study, Rodriguez-Palomares *et al*. concluded that assessment of left ventricular remodeling at 6 months did not increase the prognostic value of the principal cardiac magnetic resonance (CMR) derived variables provided by early CMR[23]. Since access to CMR is limited, we believe that this nomogram, once validated, could offer a widely available, low-cost alternative to predict patients at risk of developing pathologic left ventricular remodeling and we are doing so in a very early stage of myocardial infarction (12 hours after reperfusion has been achieved) despite administering optimal medical therapy and revascularization[24].

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Sažetak

NOMOGRAM S JEDNOSTAVNIM I RUTINSKIM KLINIČKIM I BIOKEMIJSKIM PARAMETRIMA MOŽE BITI PREDIKTOR PATOLOŠKOG REMODELIRANJA VENTRIKULA U BOLESNIKA SA STEMI-JEM

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Zatajivanje srca je vodeći uzrok pobola i smrtnosti u svijetu, a ishemijska bolest srca je njegov najvažniji etiološki čimbenik. Zatajivanje srca nastaje posljedično remodeliranju lijevog ventrikula, koje uzrokuje povećanje njegova volumena na kraju sistole i dijastole. U ovom prospektivnom opservacijskom istraživanju uključili smo 101 bolesnika koji su imali prvu epizodu STEMI i kojima je učinjena perkutana koronarna intervencija 12 sati od početka bolova uz postignuti protok TIMI III. Cilj istraživanja bio je utvrditi koji klinički i biokemijski parametri mogu pomoći u predviđanju nastupanja patološkog ventrikulskog remodeliranja godinu dana nakon preboljelog infarkta. Na temelju rezultata istraživanja stvoren je nomogram koji je uključivao vitalne parametre i rutinske biokemijske nalaze koji su pokazali najbolju korelaciju s pojavom patološkog ventrikulskog remodeliranja. Nomogram uključuje vrijednost NTproBNP-a 12 sati nakon postignute reperfuzije, vrijednost AST-a 12 sati nakon reperfuzije, vrijednost sistoličkog tlaka kod prijma te koronarnu arteriju okluzija koje je odgovorna za nastanak infarkta miokarda. Učinjena je ROC analiza koja je pokazala izvrsnu prediktivnu vrijednost nomograma. Površina ispod krivulje (AUC) je bila 0,907 (95% CI 0,842-0,973). Vrijednost nomograma od -3,54 imala je osjetljivost od 91,4% i specifičnost od 74,0%. Mišljenja smo da bi ovaj nomogram, jednom validiran, mogao ponuditi jeftinu i široko primjenjivu metodu za rano prepoznavanje bolesnika koji će razviti patološko ventrikulsko remodeliranje nakon preboljelog infarkta miokarda i to omogućiti već u vrlo ranoj fazi bolesti odnosno 12 sati nakon postignute reperfuzije.

Ključne riječi: *Akutni infarkt miokarda; Remodeliranje lijevog ventrikula; Tlak na kraju sistole; Tlak na kraju dijastole*