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Added Value of Modified Anderson–Wilkins Acuteness Score in Prognostication of Patients with Acute Myocardial Infarction

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Abstract

BACKGROUND: Electrocardiogram (ECG) signs on admission can serve as a prognostic marker in patients treated for myocardial infarction (MI).

AIM: The aim of the study was to determine the predictive role of modified Anderson–Wilkins (MAW) ECG score of acuteness on the extent of myocardial injury, left ventricular (LV) remodeling, and clinical outcome in patients with acute MI.

METHODS: Prospective, observational cohort study on patients treated for MI at the University Clinic for Cardiology. Subjects were analyzed for their demographic, clinical, ECG, LV functional, angiographic variables, course of treatment, and in-hospital outcome. MAW score was calculated for each patient. Patients were comparatively analyzed divided in two groups (score <3 and ≥3).

RESULTS: One hundred fifty patients (70% males and 30% females), aged 60.9 years were included in the study. Sixty-eight patients had MAW score <3 (mean 1.7), and 82 had score ≥3 (mean 3.5), p>0.001. Patients with ST-segment elevation MI had OR 2.1 (p>0.000), and patients with multiple locations (excluding anterior) had OR 2.1 (p > 0.000) of having MAW score ≥3. They received mechanical reperfusion 1.9 (p = 0.032) times more often. High MAW score was associated with stress hyperglycemia (OR 2.1; p = 0.032); low potassium (OR 2.8; p = 0.032), lower creatinine (p = 0.050), and higher NT-proBNP (OR 2.5; p = 0.050). High MAW score was associated with decreased LV function and increased LV dimensions on the follow-up echocardiography (p = 0.050 and 0.012, respectively).

CONCLUSION: ECG is an important prognostic tool in MI patients. ECG-derived MAW score demonstrates a strong correlation with stress hyperglycemia, potassium, creatinine, and natriuretic peptides level and can serve as an early marker of LV remodeling after MI.

Introduction

Acute myocardial infarction (AMI) is one of the leading causes of death worldwide, with cardiovascular diseases (CVD) being responsible for 31.8% of all deaths [1]. With a longer duration of myocardial ischemia, the size of irreversibly injured and necrotic myocardium is increasing, and the extent of myocardial salvage is decreasing. Nevertheless, the speed of necrosis progression depends on the vulnerability of the myocardium to ischemia and demonstrates great variations among patients. Myocardial ischemia is related to the amount of collateral blood flow of the coronary artery and metabolic preconditioning of the myocardium [2], [3]. The progression of myocardial ischemia to myocardial necrosis translates into myocardial segmental wall motion abnormalities estimated by electrocardiography [4].

The rapid revascularization of the acutely occluded coronary artery, either with the primary percutaneous coronary intervention (pPCI) or thrombolytic therapy, is of paramount significance for myocardial salvage, risk of subsequent heart failure, and survival [5], [6], [7].

Yet, estimation of symptom onset is very biased by sometimes inaccurate patient recollection or pre-existing conditions leading to clinically “silent” myocardial ischemia. Here, we stress again the very important role of electrocardiogram (ECG) and ECG signs of acuteness and severity of the myocardial injury. The Anderson–Wilkins acuteness score (AW score) quantifies the acuteness of myocardial ischemia from the ECG, and according to published literature on this subject, it is superior to historical timing and treatment delay (time from symptom onset to wire) in predicting myocardial infarct size, salvage, and mortality [8], [9], [10].

The Anderson–Wilkins acuteness score evaluates the acuteness of myocardial ischemia in patients presenting with acute thrombotic occlusion of coronary artery, based on a 12-lead standard ECG. [11] Briefly, an acuteness phase is assigned based on the presence or absence of a hyperacute T-wave or an abnormal Q-wave [12]. According to the AW score,

there are four phases, starting with the most acute one: Phase 1A: Hyperacute T-wave and no abnormal Q-wave; phase 1B: Positive T-wave and no abnormal Q-wave; phase 2A: Tall T-wave and abnormal Q-wave; and phase 2B: Positive T-wave and abnormal Q-wave. The AW score ranges from 1 to 4, with 1 being the least acute and 4 being most acute. Acute ischemia is defined as ECG acute score ≥3 and non-acute ischemia as ECG acuteness score <3 [13], [14]. The AW score may indicate an electrophysiological estimate of the viability of the myocardium, independently of the patient-reported symptoms onset, and may subsequently be used as a predictor of achievable myocardial salvage [11], [12], [13].

Echocardiography is the most frequently used non-invasive diagnostic technique which provides information regarding ventricular function and presence or absence of wall motion abnormalities [15]. Left ventricular ejection fraction (LVEF) and volumes are well-known predictors of prognosis in patients with AMI. Lower EF and larger volumes result in worse clinical outcomes [16]. According to authors White *et al*. [17] and Møller *et al*. [18], post-MI patients with EF <40% and end-systolic volume >130 cm^3 have a 5-year survival rate of 65% and 52%, respectively.

In this study, we aimed to determine if there is a place for modified Anderson–Wilkins (MAW) ECG score of acuteness in the prediction of the extent of myocardial injury, as measured with biochemical and echocardiography-gained parameters. Our secondary goal was to find out if this score can serve as a predictor of early in-hospital outcome in MI patients.

Methods

A longitudinal, prospective observational cohort study was undertaken on patients hospitalized at the University Clinic of Cardiology over the period of September 2018–March 2019 for acute MI. Inclusion criteria: Patients (all incomers) hospitalized for AMI over the aforementioned period who were willing to participate in the study and gave signed informed consent. Exclusion criteria: Patients who were not consented to participate in the study and patients who suffered in-hospital mortality over the index hospitalization.

Data were collected on demographics, CV risk factors, comorbidities, ECG-signs of myocardial injury, biomarkers of myocardial injury, LV function, angiographic distribution of the disease, MI treatment, and medications used and early in-hospital outcome.

At the study entry, to collect variables of interest, every patient underwent: taking medical history; physical examination; 12-lead ECG recording; blood sampling for: Hemogram, lipid (non-fasting), and glycemic profile, markers of myocardial injury: Highly sensitive troponin T/I (hsTn) and brain natriuretic peptide (NTpro-BNP), biochemical parameters; coronary angiography and echocardiography (2-D transthoracic echocardiography [2D TTE]).

The first post-hospital evaluation was performed in the time-frame period of 3–6 months after the index event. Medical history, physical examination, 12-lead ECG, and 2D TTE were undertaken.

Stratification of patients according to the severity and acuteness of ischemia was made from the admission electrocardiogram (12-lead pre-hospital or first in-hospital ECG recording with good quality 25 mm/s, 10 mm/mV, and 150 Hz). For the purposes of our study, we applied the calculation of the modified Anderson–Wilkins (MAW) ECG score of acuteness, in details described by Hedén *et al*. [13], according to the following formula:

$$\text{Accuteness score} = \frac{\begin{array}{c}4(\#\text{leads 1A}) + 3(\#\text{leads 1B}) \\ +2(\#\text{leads 2A}) + 1(\#\text{leads 2B})\end{array}}{\sum \#\text{leads with 1A, 1B, 2A, 2B}}$$

Patients were divided in two groups: Group A: MAW ECG score of acuteness = 0 (<3) and Group B: MAW ECG score of acuteness = 1 (≥3).

The study was approved by the ethics committee of the University Clinic of Cardiology and was conducted in accordance with the Helsinki Declaration. Informed consent was obtained from all patients before their inclusion in the study.

IBM SPSS statistical software version 22 was used for statistical analysis. Descriptive and comparative statistical methods were applied. Continuous variables were presented as means, while categorical as frequencies and percentages. Comparative statistic tests: Chi-square test for variables with dichotomous distribution, t-test, and one-way ANOVA for continuous variables with two or more categories were applied. Risk ratios with a 95% confidence interval were calculated, and the significance was determined using Cochran and Mantel-Haenszel test. Receiver operating characteristic (ROC) curves (receiver operator characteristic curves) were used for prediction capability. Correlations, uni- and multivariate linear, and logistic regression analysis were undertaken to identify significantly associated variables. Significance was determined at the level of 0.05.

Results

Patients treated for AMI at the University Clinic of Cardiology over the period of September 2018-March 2019, were 150 in total, with 70% males and 30% females, at mean age of 61 years were subjected to analysis.

No statistically significant differences were observed in age and gender distribution (Figure 1) and CV risk profile, except for hyperlipidemia and arterial hypertension being more frequent in patients with MAW score <3 (OR 1.5 and 1.6, p=0.038 and p=ns, respectively) (Table 1).

SC	148 (98.7%)	2 (1.3%)	0	ns	
Aces site					
Radial	144 (97.3%)	65 (98.2%)	74 (96.3%)	ns	
other	4 (2.7%)	1 (1.8%)	3 (3.7%)		
NO of diseased vessels	1.9 ± 0.9	1.8 ± 1.0	1.9 ± 0.8	ns	
SINTAX score	15.3 ± 7.1	14.9 ± 8.6	15.6 ± 5.7	ns	
NO of treated vessels	0.9 ± 0.3	0.9 ± 0.4	1.1 ± 0.2	0.003	
Type of treatment					
PCI (including POBA and thromboaspiration)	137 (91.3%)	55 (80.9%)	82 (100%)		1.9 (1.1–3.2)
Medical treatment	9 (6%)	9 (13.2%)	0	0.000	0.032
Urgent CABG	4 (2.7%)	4 (5.9%)	0		
Reperfusion					
NO	5 (7.4%)	1 (1.2%)	6 (4%)	0.067	
YES	63 (92.6%)	81 (98.8%)	144 (96%)		
Medical treatment					
ASA	146 (97.3%)	64 (94.1%)	82 (100%)	0.040	
$P2Y_{12}$	148 (98.7%)	66 (97.1%)	82 (100%)	ns	
BB	90 (60%)	37 (54.4%)	53 (64.6%)	ns	
RAAS	131 (87.3%)	58 (85.3%)	73 (89%)	ns	
MRA	36 (24%)	12 (17.6%)	24 (29.3%)	0.070	1.5 (0.9-2.5); ns
Loop diuretics	60 (40%)	22 (32.4%)	38 (46.3%)	0.057	1.4 (0.9-2.1); ns
DM treatment	31 (20.7%)	13 (19.1%)	18 (22%)	ns	
Hospitalization	5.2±2.3	5.1±2.4	5.4±2.2	ns	
In-hospital morbidity	31 (20.7%)	13 (19.1%)	18 (22%)	ns	1.1 (.7-1.7); ns
In-hospital mortality	4 (2.7%)	2 (2.9%)	2 (2.4)	ns	

ASA: Acetylsalicylic acid, BB: Beta-blockers, BMI: Body mass index, BSA: Body surface area, COPD: Chronic obstructive pulmonary disease, CABG: Coronary artery bypass grafting, DM: Diabetes mellitus, EF: Ejection fraction, GIT: Gastrointestinal tract, HLP: Hyperlipidemia, HTA: Hypertension, MI: Myocardial infarction, PCI: Percutaneous coronary intervention, STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non-ST-segment elevation myocardial infarction, SC: Selective coronarography, NO: Number, SINTAX score: Synergy between PCI with Taxus and cardiac surgery score, PCI: Percutaneous coronary intervention, POBA: Plain old balloon angioplasty, $P2Y_{12}$: Antiplatelet drug – $P2Y_{12}$ inhibitors, RAAS: Renin-angiotensin-aldosterone system blockers, MRA: Aldosterone receptor antagonists.

From 150 MI patients, 71.3% had STEMI and an OR of 2.1 (p > 0.000), having MWA score ≥3. A statistically significant difference for MAW score was observed in patients according to the MI location. In patients with anterior MIs, there was an equal distribution of MAW score, while MAW score < 3 predominated in patients with inferior MIs, and score ≥3 predominated in patients with multiple locations excluding anterior MI, OR 2.1 (p = 0.016). Furthermore, Group B patients had bigger ST-segment elevation (p > 0.000), while no difference was observed with respect to the transmural distribution of MI (as expressed through the presence ofQ sequela). No significant differences in the MAW score were observed with respect to the extent and severity of the disease, as assessed angiographically (Table 1).

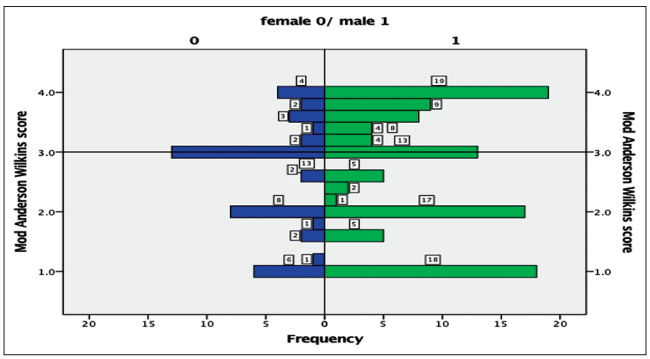

Figure 1: Population plot – distribution of MAW score across genders, with horizontal reference line set at value of 3

Table 1: Clinical characteristics of the study group and in the two comparator groups

Variable	Total (No./%)	Group A (Score <3)%	Group B (Score ≥3)%	Sig.	OR (for categorical variables) (95% CI)
Gender	150 (100)	68 (45.3)	82 (54.7)		
Female	45 (30)	20 (44.4)	25 (55.6)	ns	
Male	105 (70)	48 (45.7)	57 (54.3)		
Age (years)	60.9 ± 11.9	60.8 ± 11.9	61.0 ± 12.0	ns	
BMI (kg/m)	28.4 ± 4.9	28.8 ± 4.1	28.2 ± 5.3	ns	
BSA ($kg/1m^2$)	1.9 ± 0.2	1.9±0.1	1.9±0.2	ns	
Obesity (BMI >30)	20 (13.3)	6 (8.8)	14 (17.1)	ns	
HLP	137 (91.3)	66 (97.4)	71 (86.6)	0.021*	1.6 (1.2–2.2); 0.038
Family history	87 (58)	40 (58.8)	47 (57.3)	ns	
Smoking	98 (65.2)	42 (61.8)	56 (68.3)	ns	
HTA	133 (88.7)	64 (94.1)	69 (84.1)	0.046*	1.5 (1.0–2.0); ns
DM	44 (29.3)	19 (27.9)	25 (30.5)	ns	
Previous MI	21 (14)	11 (16.2)	10 (12.2)	ns	
Previous PCI	20 (13.3)	10 (14.7)	10 (12.2)	ns	
Preexisting EF <50%	8 (5.3)	4 (5.9)	4 (4.9)	ns	
COPD	18 (5.3)	6 (8.8)	12 (14.6)	ns	
Anemia	11 (7.3)	6 (8.8)	5 (6.1)	ns	
GIT disease	10 (6.7)	5 (7.4)	5 (6.1)	ns	

EF: Ejection fraction, PCI: Percutaneous coronary intervention, BMI: Body mass index, BSA: Body surface area, HLP: Hyperlipidemia, HTA: Hypertension, MI: Myocardial infarction, COPD: Chronic obstructive pulmonary disease, DM: Diabetes mellitus, GIT: Gastrointestinal tract.

However, there were significant differences in MAW score and therapeutic treatment approach. Patients with MAW score ≥3 had OR of 1.9 (p = 0.033) for receiving primary PCI (pPCI) treatment as compared to patients from Group A. Furthermore, patients from Group B more frequently received revascularization on more than one "culprit" coronary artery (p=0.003). As for medical therapy, patients from Group B had OR of 1.5 and 1.4 (p = ns for both) to receive mineralocorticoid receptor antagonists and loop diuretics. No significant differences for in-hospital morbidity or mortality were observed, although they were statistically insignificantly higher in Group B patients (Table 1).

Surprisingly, no significant differences were found for LV function between the groups, not only on the first in-hospital echocardiography but also on the 3–6-month follow-up test (after the period of LV remodeling). In the patients from Group B, statistically significant thinner IVS was observed (0.009), which can be discussed accompanied with Q-wave sign of transmural MI that was more frequent in the same patients (Table 2).

Table 2: LV morphologic and functional variables as measured by 2-D TTE of the study group and in the two comparator groups on the first and second study

Variable	Total (No./%)	Group A (Score <3)%	Group B (Score ≥3)%	Sig.
First measurement				
LVEDd (mm)	51.8 ± 5.1	52.1 ± 5.2	51.6 ± 5.0	ns
LVESd (mm)	36.5 ± 5.7	36.5 ± 5.7	36.5 ± 5.7	ns
EF (%)	52.4 ± 9.6	52.7 ± 9.1	52.2 ± 10.1	ns
LA (mm)	38.2 ± 4.4	38.6 ± 3.8	37.8 ± 4.8	ns
IVS (mm)	11.5 ± 1.7	11.9 ± 1.6	11.2 ± 1.8	0.009
Diastolic dysfunction	59 (40.4)	26 (39.4)	33 (41.3)	ns
LV dysfunction				
EF <50%	70 (46.7)	32 (47.8)	38 (47.5)	ns
EF >50%	77 (51.3)	35 (52.2)	42 (52.5)	
Second measurement				
LVEDd (mm)	53.5 ± 5.1	53.1 ± 5.6	53.7 ± 4.9	ns
LVESd (mm)	38.4 ± 5.2	37.9 ± 5.2	38.7 ± 5.3	ns
EF (%)	50.6 ± 8.4	51.5 ± 7.7	50.1 ± 8.9	ns
LA (mm)	39.3 ± 3.9	39.6 ± 4.0	39.2 ± 3.9	ns
IVS (mm)	11.0 ± 2.2	10.9 ± 2.6	11.1 ± 2.0	ns
Diastolic dysfunction	29 (70.7)	11 (78.6)	18 (66.7)	ns
LV dysfunction				
EF <50%	24 (37.5)	9 (37.5)	15 (37.5)	ns
EF >50%	77 (62.5)	15 (62.5)	25 (62.5)	

2-D TTE: 2-dimensional transthoracic echocardiography, LVEDd: Left ventricular end-diastolic dimension, LVEDs: Left ventricular end-systolic dimension, EF: Ejection fraction, LA: Left atrium; IVS: Interventricular septum

We performed a paired samples analysis for each patient on the first and second echocardiography study, and also after dividing the patients according to their MAW score (Table 3). We found no statistically significant difference in EF; however, a significant increase in LV dimensions was observed (p = 0.002 and p = 0.013, respectively). A comparative analysis between groups demonstrated that even in the absence of difference in mean EF on the first study, on the second study, patients from Group B had statistically significant lower EF. We found no significant differences in LVED dimension; however, LVES dimension was significantly higher on the first and on the second 2 TTE study in patients from Group B (p = 0.012 and 0.050, respectively).

Table 3: Left-ventricular parameters of the study group and in the two comparator groups

Variable	Total	Group A (Score <3)	Group B (Score ≥3)	Sig (2-tailed) between groups
EF(%) first	51.9 ± 8.7	53.4 ± 6.3	50.9 ± 9.8	ns
EF(%) second	50.6 ± 8.4	51.5 ± 7.7	50.1 ± 8.9	0.051
Sig. f vs. s	ns	0.049	ns	
LVEDd (mm) first	51.4 ± 5.2	50.8 ± 5.5	51.8 ± 5.0	ns
LVEDd (mm) second	53.5 ± 5.1	53.1 ± 5.6	53.7 ± 4.9	ns
Sig. f versus s	0.002	0.024	0.033	
LVEDs (mm) first	36.7 ± 4.7	36.2 ± 5.0	37.2 ± 4.5	0.012
LVEDs (mm) second	38.4 ± 5.2	37.9 ± 5.2	38.6 ± 5.3	0.050
Sig. f versus s	0.013	ns	ns	

EFf-EF: From the first study; EFs-EF: From the second study, LVEDdf-LVEDd: From the first study, LVEDds-LVEDd: From the second study, LVESdf-LVESd: From the first study, LVESds-LVESd: From the second study.

Out of all analyzed biochemical variables, stress glycemia was significantly higher in Group B patients (p = 0.011), who also demonstrated worse glycemic control over the course of hospital treatment, with observed episodes of GI >10 mmol/L (OR 1.6, p = ns). Potassium level was found to be lower in Group B (p = 0.012), while creatinine level was higher in Group A patients (p = 0.050). Interestingly, patients from Group B had 2.6 times higher relative risk of elevated NTpro-BNP levels (p = 0.060) (Table 4).

Variables that we found to be significantly associated with the MWA score were subjected to univariate binary logistic regression analysis to confirm significant associations. Hyperlipidemia and arterial hypertension were found to be negatively associated with MWA score 1, as were low serum potassium and creatinine levels. STEMI patients had 5.6 times higher risk to have MWA score >3 and MIs with multiple locations, as compared to anterior MIs (OR 2.3), while the lowest OR was found for inferior MIs (OR 0.2). Positive association with MWA score ≥3 (Group B) was found for number of treated vessels. Stress glycemia was higher in Group B patients, who had 2.3 times higher risk of having unsatisfactory glycemic control. Borderline significance was detected for IVS thickness and the use of diuretics over the hospital course of treatment (Table 5).

We identified seven independently associated identifiers with a high MAW score using multivariate logistic regression analysis (backward conditional) and a model with a Chi-square test (test statistic 50.856; p = 0.000; percent correct prediction 74.1%). The identifiers were: STEMI, STEMI with multiple locations (excluding anterior), presence of stress hyperglycemia, creatinine and potassium, thin IVS, and treatment with diuretics during a hospital stay (Table 6).

We also analyzed correlations between MAW score and various disease variables and significant correlation coefficients are displayed in Table 7 and Figure 2.

Predictive functions of various biochemical variables (hsTn, NTpro-BNP, stress glycemia, WBC, creatinine, and potassium) were subjected to ROC curve analysis (Figure 3 with accompanying table). Only two biochemical variables demonstrated statistically significant discriminatory function for MWA score (≥3): High-stress glycemia and low serum potassium (area under the curve .618, and .371, p=0.013 and 0.007, respectively).

Table 4: Biochemical variables of the study group, and in the two comparator groups

Variable	Total (No./%)	Group A (Score <3)%	Group B (Score ≥3)%	Sig.	OR (95% CI)
Hemogram					
Er (×10^9)	4.7 ± 0.6	4.7±0.6	4.8±0.6	ns	
Hgb (g/L)	141.1 ± 17.8	140.4 ± 18.7	141.7 ± 17.1	ns	
Hct (%)	41.4 ± 4.5	41.3 ±4.8	41.4 ± 4.4	ns	
Le (×109)	11.3 ± 3.5	10.9 ± 3.5	11.6 ± 3.6	ns	
PLT (×106)	247.1 ± 70.3	256.7 ± 85.2	239.1 ± 54.3	ns	
Glycemic status					
Stress glycemia (mmol/L)	9.4±4.6	8.3±3.7	10.2±5.1	0.011	
Stress hyperglycemia	92 (61.7)	35 (52.2)	57 (69.5)	0.023	2.1 (1.1–4.1) p=0.032
HbA1c (%)	6.3 ± 1.5	6.3 ± 1.4	6.3 ± 1.5	ns	
Glico regulation					
>4–<10 mmol/L	112 (74.7)	54 (79.4)	58 (70.7)	ns	1.6 (0.7-3.4)
>10 mmol/L	38 (25.4)	14 (20.6)	24 (29.3)		
Lipoproteins					
Cholesterol (mmol/L)	5.7 ± 1.3	5.6 ± 1.3	5.9 ± 1.2	ns	
LDL-C (mmol/L)	3.5 ± 1.1	3.4 ± 1.2	3.6 ± 1.1	ns	
HDL-C (mmol/L)	1.2 ± 0.3	1.2 ± 0.4	1.2 ± 0.3	ns	
TG (mmol/L)	2.0 ± 1.6	1.9 ± 1.4	2.1 ± 1.8	ns	
Biochemical variables					
BUN (mmol/L)	6.4 ± 3.2	6.7 ± 3.1	6.1 ± 3.3	ns	
Creatinine (µmol/L)	87.3 ± 26.6	91.7 ± 27.7	83.6 ± 25.3	0.050	
Sodium (mmol/L)	138.3 ± 3.4	138.1 ± 3.5	138.6 ± 3.3	ns	
Potassium (mmol/L)	4.2 ± 0.5	4.3 ± 0.6	4.1 ± 0.5	0.012	
Potassium <3.5 mmol/L	27 (18.1)	7 (10.4)	20 (24.4%)	0.012	2.8 (1.1–7.0); p=0.032
Cardiac biomarkers					
hsTn (ng/L)	9737.5 ± 32148.5	11181.2 ± 40916.2	8525.5 ± 22509.6	ns	
NTpro-BNP (pg/ml)	3171.8 ± 5378.7	2875.2 ± 5230.5	3417.8 ± 5504.0	ns	
NTpro-BNP >125 pg/ml	130 (86.7)	55 (80.9)	75 (91.5)	0.049	2.5 (0.9–6.7); p=0.050

Er: Erythrocytes, Hgb: Hemoglobin, Hct: Hematocrit, Le: Leukocytes, PLT: Platelets, HbA1c: Glycated hemoglobin, LDL-C: Low-density lipoproteins, HDL: High-density lipoproteins, TG: Triglycerides, BUN: Blood urea nitrogen, hsTn: High sensitive troponin, NTpro-BNP: N-terminal prohormone of brain natriuretic peptide.

Discussion

We investigated the predictive value of the MAW score of acuteness as compared with biochemical, LV functional parameters, extent and severity of CAD, and in-hospital clinical outcome. Our general finding is a high degree of agreement between MAW score and biochemical variables, as measured in the acute phase of MI.

Table 5: Variables associated with MWA score in univariate analysis

Variable	beta	ExpB (95% CI) / Mantel–Haenszel OR	Sig.
HLP	−0.178	1.121 (1.020–1.232) p=0.038	0.021
HTA	−0.179	1.118 (1.001–1.250) p=0.065	0.046
STEMI	1.736	5.676 (1.448–22.247) p=0.013	0.000
ST-seg elevation	0.404		0.000
MI location			
Multiple versus anterior	0.834	2.303 (0.957–5.544) p=0.063	0.019
Inferior versus anterior	−1.367	0.255 (0.098–0.661) p=0.005	
NO of treated vessels	0.283		0.000
NTpro-BNP >125pg/ml	0.929	OR 2.532 (0.961–6.765) p=0.064	0.080
Stress hyperglycemia (mmol/L)	.158		0.054
Glico-regulation	0.777	2.174 (1.087–4.348) p=0.028	0.026
WBC (×10^6)	0.147		0.063
Creatinine (μmol/L)	0.165		0.043
Potassium (mmol/L)	−0.209		0.010
IVS (mm)	−0.271		0.001
Diuretics treatment	0.591	1.806 (0.926–3.523) p=0.083	0.070

HLP: Hyperlipidemia, HTA: Hypertension, STEMI: ST-segment elevation myocardial infarction, MI: Myocardial infarction, NO: Number, NTpro-BNP: N-terminal prohormone of brain natriuretic peptide, WBC: White blood cells, IVS: Interventricular septum.

Electrocardiography (ECG), since its invention, is the main diagnostic tool for MI diagnosis, in the same time localizing the site of infarction, with different leads representing the specific myocardial areas [19]. The evolution of MI begins early after an acute coronary artery occlusion and the subsequent ECG changes are caused by the development of ischemia and later on necrosis. Initial ECG changes - tall T-waves are very often undetected, as they develop within minutes after acute occlusion. They are followed by the evolvement of ST-segment elevation, abnormal Q waves, T-wave inversion, and lastly, resolution of the ST-segment elevation [14].

Table 6: Multivariate logistic regression analysis

Variables	B	Wald	Sig.	Exp(B)	95% CI for EXP(B)	
					Lower	Upper
STEMI/NSTEMI	−1.194	4.514	0.034*	0.303	0.101	0.912
MI location (multiple versus)		4.906	0.086			
MI anterior	−0.987	2.821	0.093	0.373	0.118	1.179
MI inferior	−1.305	4.733	0.030*	0.271	0.084	0.879
Number of treated CA	1.316	2.526	0.112	3.727	0.736	18.882
Stress hyperglycemia (mmol/L)	0.111	4.131	0.042*	1.118	1.004	1.245
Creatinine (μmol/L)	−0.017	3.905	0.048*	0.983	0.966	1.000
Potassium (mmol/L)	−1.357	6.076	0.014*	0.257	0.087	0.757
IVS (mm)	−0.308	5.423	0.020*	0.735	0.567	0.952
Diuretics treatment	1.212	6.093	0.014*	3.360	1.283	8.798
Constant	11.735	12.305	0.000	124844.073		

STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non-ST-segment elevation myocardial infarction, MI: Myocardial infarction, IVS: Interventricular septum.

The original Anderson–Wilkins acuteness score was developed for MI with anterior localization and could not be used when inferior MI was present [12]. The problem is the abnormal Q-wave criterion (≥30 ms duration), that is rarely met in the inferior leads, where predominantly positive QRS complex is expected [13]. The MAW score loosens up the strict criteria and adjusts the Q-wave criterion from ≥30 ms to ≥20 ms in inferior leads, and thus the MAW score predicts myocardial salvage equally well for anterior and inferior AMI [8], [13], (Table 8).

Table 7: Significant correlations with MAW score

Variable	Correlation (r)	Sig (p)
Length of hospitalization	0.157	0.056
HLP	−0.178	0.029
HTA	−0.201	0.014
Type of MI (STEMI)	−0.413**	0.000
ST-seg. elevation	0.423**	0.000
NO of treated CA	0.276**	0.000
Stress glycemia	0.169	0.039
WBC	0.166	0.042
Potassium (low)	−0.206	0.012
IVS (thin)	−0.305**	0.000

HLP: Hyperlipidemia, HTA: Hypertension, MI: Myocardial infarction, NO: Number, CA: Coronary artery, WBC: White blood, MAW: Modified Anderson–Wilkins

Test result variable(s)	Area	Std. Error	Asymptotic Sig	Asymptotic 95% CI	
				Lower bound	Upper bound
hsTn (ng/L)	0.474	0.048	0.583	0.380	0.567
NTpro-BNP (pg/ml)	0.561	0.048	0.203	0.467	0.654
Stress GI (mmol/L)	0.618	0.046	0.013*	0.528	0.709
WBC (×10^9)	0.567	0.047	0.160	0.474	0.660
Creatinine (μmole/L)	0.410	0.047	0.060	0.318	0.503
Potassium (mmol/L)	0.371	0.046	0.007*	0.281	0.460

hsTn: High-sensitive troponin, NTpro-BNP: N-terminal prohormone of brain natriuretic peptide, WBC: White blood cells.

According to the first study addressing the prognostic value of the MAW acuteness score, when stratifying patients by MAW acuteness score, the initial difference in myocardial salvage results in a long-term difference in mortality, while the incidence of re-infarction is independent of the MAW score [10].

Table 8: Lead specific criteria for abnormal Q-waves and tall T-waves

Lead	Abnormal Q-wave	Tall T-wave
I	≥30 ms	≥0.50 mV
II	≥30 ms	≥0.50 mV
III	≥30 ms and abnormal Q in aVF	≥0.25 mV
aVR	-	-
aVL	≥30 ms	≥0.25 mV
aVF	≥30 ms	≥0.50 mV
V1	Any Q-wave	≥0.50 mV
V2	Any Q-wave	≥1.0 mV
V3	Any Q-wave	≥1.0 mV
V4	≥30 ms	≥1.0 mV
V5	≥30 ms	≥0.75 mV
V6	≥30 ms	≥0.50 mV

Ms: Milliseconds, mV: Millivolts.

Association of common comorbidities and risk factors for AMI and MAW score

Our data showed that hyperlipidemia and arterial hypertension were more frequent in patients with MAW score <3 (less acute and less severe).

Arterial hypertension is a well-known major risk factor for CAD and MI [20]. Over 90% of MI victims bear many risk factors for coronary atherosclerosis besides HTN, smoking habit, obesity, dyslipidemia, etc. HTN is an independent risk factor over other risk factors that may coexist. There is a linear increase in the risk of MI with an increase of blood pressure. HTN particularly raises the risk of MI in people under 65 years [21]. Along with HTA, the other major risk factor for CVD is hyperlipidemia or dyslipidemia [22], [23], [24], [25]. According to Ballarino *et al*., age, gender, receiving therapy for CVD, smoking, hypertension, hypercholesterolemia, and increased BMI are all predictive of the acute coronary syndrome [26]. In the FAST MI registry, patients with Q wave tend to be younger, males, with a history of smoking and family

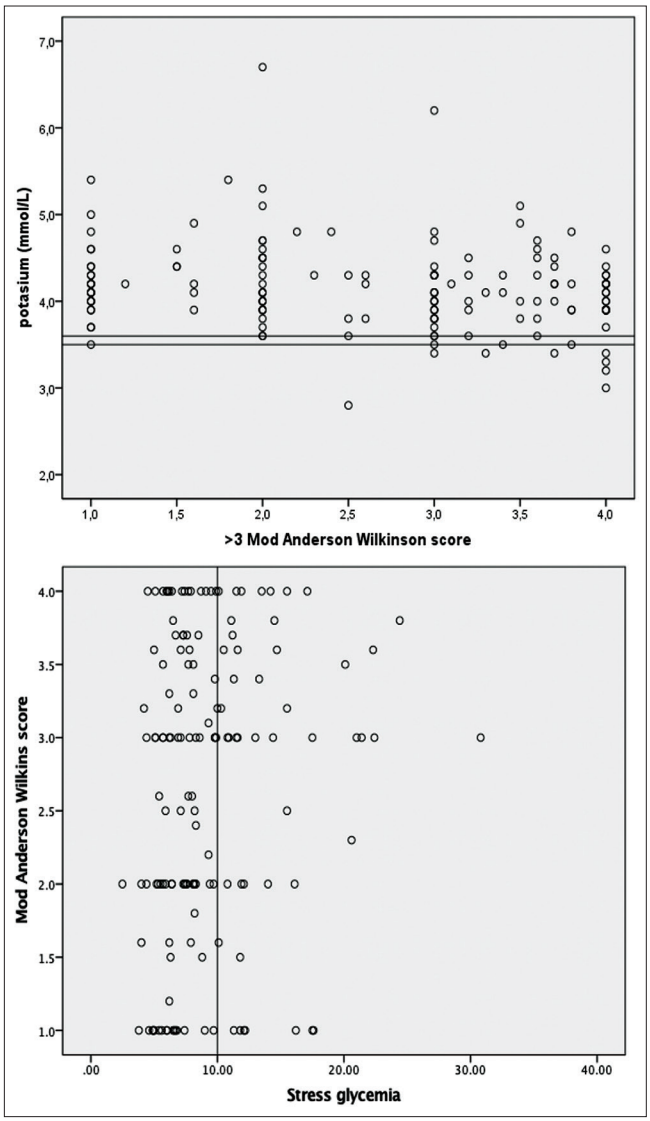

Figure 2: Correlation coefficient matrix of modified Anderson–Wilkins score with potassium and stress glycemia

history. Patients with non-Q wave MIs were heavily burdened with risk factors and had worse baseline demographic characteristics [37].

This might be the explanation why in our study population, patients with lower acuteness severity index were more often patients with hypertension and hyperlipidemia.

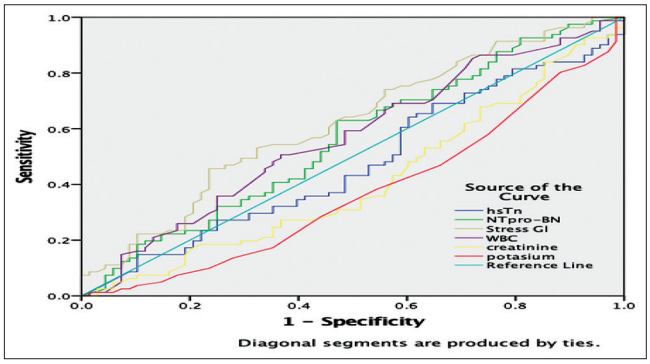

Figure 3: Receiver operating characteristic curve for modified Anderson–Wilkins score and biochemical variables

Association of biochemical variables with MAW

We found that stress hyperglycemia, serum levels of potassium, and creatinine are statistically significant associated with the MAW score of acuteness, as well as natriuretic peptide (NT-proBNP).

According to the definition of The American Diabetes Association, stress hyperglycemia is an elevation of fasting glucose ≥7 mmol/L, or 2-h postprandial glucose ≥11 mmol/L, in a patient without previous diabetes mellitus. Distinction on whether a patient has stress glycemia or previously undiagnosed diabetes is made by glycated hemoglobin (HbA1c). HbA1c value ≥ 6.5% indicates preexisting unrecognized diabetes, while value ≤6.5% indicates stress hyperglycemia [27]. Studies show that stress hyperglycemia is present in one of four hospitalized ACS patients, and it is found in 41% of elderly patients. Stress hyperglycemia can be used as a prognostic indicator in patients with AMI [28], [29] According to Marfella *et al*. [30], patients with hyperglycemia had a larger infarct size compared with normoglycemic patients, and also, there is evidence that supports an association between ventricular desynchrony and blood glucose levels in patients with MI, and an association with early in-hospital mortality in patients with AMI [31].

Our data is in concordance with such findings. In our study, patients from Group B had OR of 2.1 (p = 0.023) of having stress hyperglycemia, which also demonstrated a very good discriminatory function as a continuous variable with ROC analysis (area under the curve .618, p = 0.013).

Normal serum potassium level ranges from 3.5 to 5.1 mmol/L in adults [32], while low potassium level is described in approximately 8% of patients with MI [33]. According to Goyal *et al*., a higher mortality rate can be observed in AMI patients with potassium levels below 3.5 and above 4.5 mmol/L. The “so-called” safe window is levels 3.5–4.5 mmol/L [34].

In our study, we found that serum potassium was statistically significantly lower in Group B (p = 0.012), with an OR of 2.8 (p = 0.032), as compared to Group A, to have serum potassium <3.5 mmol/L. No significant association with in-hospital morbidity was observed.

Renal dysfunction is a strong independent predictor of cardiovascular outcome after MI [35]. Cakar *et al*. found that the presence of elevated creatinine on admission in STEMI patients is associated with increased 1-year mortality, independent of other conventional risk factors [36].

We found that patients from Group A had higher serum creatinine level (p = 0.048), which is in accordance with the literature, as patients with renal dysfunction tend more often to be late presenters.

Natriuretic peptides are not only biomarkers for the diagnosis of heart failure but even more important prognostic markers. In MI patients, natriuretic peptides correlate with the presence and degree of heart failure in the early phase, but also, they are powerful prognosticators of LV remodeling, LV dysfunction, and early and late cardiac morbidity and mortality. Fakhri *et al*. demonstrated the association of MAW score with increasing NT-proBNP levels. In STEMI patients with severe ischemia, neurohormonal activation is inversely associated with ECG patterns of acute myocardial ischemia [38].

We found high levels of natriuretic peptides (NT-proBNP was measured), without inter-group statistically significant difference. However, patients from Group B had OR 2.5 (p = 0.050), to have NT-proBNP level above 125 pg/ml.

LV functional parameters and MAW score

LVEF is one of the well-known predictors of prognosis in patients with AMI, the lower the EF, the worse the mortality and morbidity will be [16]. The SAVE echocardiography substudy reported that larger infarct size correlates with LV shape distortion and predicts progressive LV dilatation, LV dysfunction, and cardiac death [16], [18].

Simple comparative statistics of LV morphological and functional parameters measured with 2-D TTE on the first and second measurements found no statistically significant differences between the two groups. However, paired sample statistics revealed a completely different situation when comparing LV morphology after the 3 months period. This is a period of LV remodeling after MI, and we observed that there was no significant change in global LV function as measured through EF (%). However, signs of LV remodeling can be observed through statistically significant increases of LV dimensions (end-diastolic and end-systolic, p = 0.002 and p = 0.013, respectively) that were taken as surrogate markers for LV volumes. That said, comparing the process of LV remodeling in the patients from the two groups, we observed a statistically significant decrease of EF on the follow-up study in Group B patients, while patients from both groups had increased LVES dimensions. It is our conclusion that, even in such a small study population, a more pronounced LV remodeling can be predicted with a more severe MAW score of acuteness.

Fakhri *et al*. used global longitudinal strain analysis (GLS) and reported that pre-hospital risk stratification by ECG identifies patients with acute and severe ischemia who are at increased risk for reduced ventricular function (assessed by GLS) after STEMI. Optimizing reperfusion delays in these patients can, therefore, be of particular benefit in improving clinical outcome after STEMI [4].

Limitations

One of the biggest limitations of this study is the number of study subjects, bearing in mind the prevalence of the disease, which may, in some way, affect the results that we received.

Furthermore, another limitation of the study is that we only applied 2-D TTE early and after 3 months of MI, no imaging modality that can distinct myocardium at risk and final area of necrosis was applied.

However, this is the first study done with an analysis of NTpro-BNP in a cohort of patients with acute MI for prognostication purposes. Even though this is a biomarker known for several years, it was not widely available in our country, and to our knowledge, this is the first study that analyzes the role of natriuretic peptides in the prognostication of MI patients.

Conclusion

ECG is still an irreplaceable tool in diagnosis and prognosis of MI patients. ECG-derived scores, as MAW score, are better surrogate markers of “times” in MI patients. MAW score demonstrates a strong correlation with biochemical variables: Stress hyperglycemia, serum potassium and creatinine level, and natriuretic peptides. MAW score can serve as an early marker of LV remodeling, as demonstrated by the correlation with LV parameters.

However, we were unable to demonstrate a significant association of MAW score with in-hospital morbidity nor mortality. Larger scale studies are needed to draw such conclusions.

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