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

Aleksandar Serafimov1*, Hajber Taravari2, Enes Shehu2, Darko Kitanoski2, Visar Miftari2, Ljubica Georgievska-Ismail2, Saso Kedev2, Marija Vavlukis3

1Department of Cardiology, Clinical Hospital Shtip, Faculty of Medical Sciences, University Goce Delchev, Shtip, Republic of Macedonia; 2Department of Cardiology, University Clinic for Cardiology, Medical Faculty, Skopje, Republic of Macedonia; 3Department of Neurology, Clinical Hospital Tetovo, Faculty of Medical Sciences, University of Tetovo, Tetovo, Republic of Macedonia

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 preexisting 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 cm³ 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 (hsTnI) 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:

\[
\text{Acuteness score} = \frac{4(\#\text{leads 1A}) + 3(\#\text{leads 1B}) + 2(\#\text{leads 2A}) + 1(\#\text{leads 2B})}{\#\text{leads with 1A, 1B, 2A, 2B}}
\]

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).

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).

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. OR (for categorical variables) (95% CI) |
|----------|---------------|---------------------|---------------------|-------------------------------------------|
| EF (%)   | 51.9 ± 8.7    | 53.4 ± 6.3          | 50.9 ± 9.8          | ns                                         |
| EF (%)   | 50.6 ± 8.4    | 51.5 ± 7.7          | 50.1 ± 8.9          | ns                                         |
| LVES (%) | 51.5 ± 5.3    | 53.1 ± 5.6          | 53.7 ± 4.9          | ns                                         |
| LVES (%) | 51.8 ± 5.8    | 53.7 ± 5.5          | 53.6 ± 4.9          | ns                                         |
| LA (%)   | 52.6 ± 7.5    | 53.6 ± 6.7          | 53.5 ± 4.5          | ns                                         |
| LA (%)   | 52.1 ± 7.9    | 53.3 ± 7.4          | 53.4 ± 4.7          | ns                                         |
| IVS (%)  | 48.9 ± 5.9    | 50.2 ± 6.8          | 51.0 ± 6.2          | ns                                         |
| IVS (%)  | 48.8 ± 7.2    | 50.3 ± 6.9          | 51.1 ± 6.3          | ns                                         |

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 LVEDV dimension; however, LVES dimension was significantly higher on the first and on the second 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 on the first measurement

| Variable | Total (No./%) | Group A (Score <3%) | Group B (Score ≥3%) | Sig. (2-tailed) between groups |
|----------|---------------|---------------------|---------------------|-----------------------------|
| EF (%)   | 51.9 ± 8.7    | 53.4 ± 6.3          | 50.9 ± 9.8          | ns                                         |
| EF (%)   | 50.6 ± 8.4    | 51.5 ± 7.7          | 50.1 ± 8.9          | ns                                         |
| LVES (%) | 51.5 ± 5.3    | 53.1 ± 5.6          | 53.7 ± 4.9          | ns                                         |
| LVES (%) | 51.8 ± 5.8    | 53.6 ± 5.8          | 53.7 ± 6.7          | ns                                         |
| LA (%)   | 52.6 ± 7.5    | 53.6 ± 6.7          | 53.5 ± 4.9          | ns                                         |
| LA (%)   | 52.1 ± 7.9    | 53.3 ± 6.8          | 53.7 ± 4.7          | ns                                         |
| IVS (%)  | 48.9 ± 5.9    | 50.2 ± 6.8          | 51.0 ± 6.2          | ns                                         |
| IVS (%)  | 48.8 ± 7.2    | 50.3 ± 6.9          | 51.1 ± 6.3          | ns                                         |
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 (x10³)                         | 4.7 ± 0.6     | 4.7 ± 0.6           | 4.8 ± 0.6           |     |             |
| Hgb (g/L)                         | 141 ± 17.8    | 140.4 ± 18.7        | 141.7 ± 17.1        |     |             |
| Hct (%)                           | 41.4 ± 4.5    | 41.3 ± 4.6          | 41.4 ± 4.4          |     |             |
| Le (+/−9)                         | 11.3 ± 3.5    | 10.9 ± 3.5          | 11.6 ± 3.6          |     |             |
| PLT (+/−6)                        | 247 ± 17.0    | 256.7 ± 85.2        | 239.1 ± 54.3        |     |             |
| Glycemic status                   |               |                     |                     |     |             |
| Stress glycemia (mmol/L)          | 9.44 ± 6.3    | 8.3 ± 3.7           | 10.2 ± 5.1          | 0.011 |             |
| Stress hyperglycemia              | 92 (61.7)     | 35 (52.2)           | 57 (69.5)           | 0.023 |             |
| HbA1c (%)                         | 6.3 ± 1.5     | 6.3 ± 1.4           | 6.3 ± 1.5           |     |             |
| Glico regulation                  |               |                     |                     |     |             |
| >4–<10 mmol/L                     | 112 (74.7)    | 54 (79.4)           | 58 (70.7)           | 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           |     |             |
| LDL-C (mmol/L)                    | 3.5 ± 1.1     | 3.4 ± 1.2           | 3.6 ± 1.1           |     |             |
| HDL-C (mmol/L)                    | 1.2 ± 0.3     | 1.2 ± 0.4           | 1.2 ± 0.3           |     |             |
| TG (mmol/L)                       | 2.0 ± 1.6     | 1.9 ± 1.4           | 2.1 ± 1.8           |     |             |
| Biochemical variables             |               |                     |                     |     |             |
| BUN (mmol/L)                      | 6.4 ± 3.2     | 6.7 ± 3.1           | 6.1 ± 3.3           |     |             |
| 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         |     |             |
| 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 |             |
| Cardiac biomarkers                |               |                     |                     |     |             |
| hsTn (ng/L)                       | 9737.5 ± 32148.5 | 11815.1 ± 40916.2 | 8525.5 ± 22509.6 |     |             |
| NTpro-BNP (pg/ml)                 | 3171.8 ± 5378.7 | 2875.2 ± 5230.5 | 3417.8 ± 5504.0 |     |             |
| NTpro-BNP >125 pg/ml              | 255 (80.9)    | 75 (91.5)           | 255 (80.9)          | 0.049 | 2.5 (0.9-6.7); p=0.009 |

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 Gl >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 were 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.

Table 5: Variables associated with MWA score in univariate analysis

| Variable                          | beta  | Exp(Q (95% CI)) / Mantel– Haenszel OR | Sig.  |
|-----------------------------------|-------|---------------------------------------|-------|
| HLP                               | −0.178| 1.121 (1.020–1.232) p=0.038           | 0.021 |
| HTA                               | −0.179| 1.188 (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                       |       |                                      |       |
| Superior versus anterior           | 0.834 | 2.303 (0.957–5.544) p=0.003           | 0.019 |
| Inferior versus anterior           | −1.367| 0.255 (0.088–0.661) p=0.005           | 0.019 |
| NO of treated vessels              | 0.283 |                                      | 0.000 |
| NTpro-BNP >125µg/ml               | OR 2.3 (0.916–6.765) p=0.064          | 0.080 |
| Stress hyperglycemia (mmol/L)     | 0.147 | 0.777 (0.187–4.348) p=0.028           | 0.026 |
| Glico-regulation                  | 0.043 | 0.165                                 |       |
| WBC (+10³)                        | 0.010 | 0.777                                 |       |
| IVS (mm)                          | −0.271| 0.591                                 | 0.001 |
| Diuretics treatment               |       | 1.806 (0.926–3.523) p=0.083           | 0.700 |

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 MWA score and biochemical variables, as measured in the acute phase of MI.
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 evolution 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) |
|--------------------------|--------|-------|------|--------|-------------------|
| STEMI/STEMI (multiple versus) | -1.194 | 4.514 | 0.034 | 0.303  | 0.101 - 0.912     |
| 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 hyperglycaemia    | 0.111  | 1.431 | 0.242 | 1.118  | 1.004 - 1.245     |
| Creatinine (mmol/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)                 | -0.398 | 5.423 | 0.002 | 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 | 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 8: Lead specific criteria for abnormal Q-waves and tall T-waves

| Lead       | Abnormal Q-wave | Tall T-wave |
|------------|-----------------|-------------|
| I          | ≥20 ms          | ≥0.50 mV    |
| II         | ≥30 ms          | ≥0.50 mV    |
| III        | ≥30 ms and abnormal Q in aVF | ≥0.25 mV |
| aVR        | ≥30 ms          | ≥0.50 mV    |
| 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 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.

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].

![Figure 3: Receiver operating characteristic curve for modified Anderson–Wilkins score and biochemical variables](https://example.com/f3.png)
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 (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.

Acknowledgment

We would like to acknowledge the collaboration and support of the colleges and nurses in the ICCU at the University Clinic for Cardiology for their help and support over the course of this study that is a part of broader work on my doctoral thesis.

References

1. Ioacara S, Popescu AC, Tenenbaum J, Dimulescu DR, Popescu MR, Sirbu A, et al. Acute myocardial infarction mortality rates and trends in Romania between 1994 and 2017. Int J Environ Res Public Health. 2019;17(1):285. https://doi.org/10.3390/ijerph17010285
PMID:31906114
2. Denktas AE, Anderson HV, McCarthy J, Smalling RW. Total ischemic time: The correct focus of attention for optimal ST-segment elevation myocardial infarction care. JACC Cardiovasc Interv. 2011;4(6):599-604. https://doi.org/10.1016/j.jcin.2011.02.012
PMID:21700244
3. Floyd JS, Maynard C, Weston P, Johanson P, Jennings RB, Wagner GS. Effects of ischemic preconditioning and arterial collateral flow on ST-segment elevation and QRS complex prolongation in a canine model of acute coronary occlusion. J Electrocardiol. 2009;42:19-26. https://doi.org/10.1016/j.jelectrocard.2008.09.006
PMID:19070706
4. Fakhri Y, Ersemb L, Keber L, Hassager C, Hessefeld R, Steinmetz J, et al. Pre-hospital electrocardiographic severity and acuteness scores predict left ventricular function in patients with ST elevation myocardial infarction. J Electrocardiol. 2016;49(3):284-91. https://doi.org/10.1016/j.jelectrocard.2016.02.012
PMID:26962019
5. Lønborg J, Schoos MM, Kelbaek H, Holmvang L, Steinmetz J, Vejlstrup N, et al. Impact of system delay on infarct size, myocardial salvage index, and left ventricular function in patients with ST-segment elevation myocardial infarction. Am Heart J. 2012;164(4):538-46. https://doi.org/10.1016/j.ahj.2012.07.021
PMID:23067912
6. Steg PG, James SK, Atar D, Badano LP, Blomstrom-Lundqvist C, Borger MA, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment...
The impact of glyco-metabolic status in myocardial infarction. Eur Heart J. 2012;33(20):2569-619. PMid:22922416

7. Terkelsen CJ, Sørensen JT, Maeng M, Jensen LO, Tilsted HH, Trautner S, et al. System delay and mortality among patients with STEMI treated with primary percutaneous coronary intervention. JAMA. 2010;304(7):763-71. https://doi.org/10.1001/jama.2010.1139 PMid:20716739

8. Corey KE, Maynard C, Pahlm O, Wilkins ML, Anderson ST, Cerqueira MD, et al. Combined historical and electrocardiographic timing of acute anterior and inferior myocardial infarcts for prediction of reperfusion achievable size limitation. Am J Cardiol. 1999;83(6):626-31. https://doi.org/10.1016/s0002-9149(99)01042-x PMid:10190393

9. Englbohm H, Strauss DG, Heden B, Hedström E, Jovinge S, Götberg M, et al. The evaluation of an electrocardiographic myocardial ischemia acuteness score to predict the amount of myocardial salvage achieved by early percutaneous coronary intervention clinical validation with myocardial perfusion single photon emission computed tomography and cardiac magnetic resonance. J Electrocardiol. 2011;44(5):525-32. https://doi.org/10.1016/j.jelectrocard.2011.03.008 PMid:21658711

10. Sejersten M, Ripa RS, Maynard C, Grande P, Andersen HR, Wagner GS, et al. Timing of ischemic onset estimated from the electrocardiogram is better than historical timing for predicting outcome after reperfusion therapy for acute anterior myocardial infarction: A DANish trial in acute myocardial infarction. 2 (DANAM-II) substudy. Am Heart J. 2007;154(1):61. https://doi.org/10.1016/j.ahj.2007.04.003 PMid:17584552

11. Fakhri Y, Schoos MM, Clemmensen P, Sejersten M. Clinical use of the combined Sclarovsky Birnbaum Severity and Anderson Wilkins acuteness scores from the pre-hospital ECG in ST-segment elevation myocardial infarction. J Electrocardiol. 2014;47(4):566-70. https://doi.org/10.1016/j.jelectrocard.2014.03.009 PMid:24792905

12. Wilkins ML, P pryor AD, Maynard C, Wagner NB, Elias WJ, Litwin PE, et al. An electrocardiographic acuteness score for quantifying the timing of a myocardial infarction to guide decisions regarding reperfusion therapy. Am J Cardiol. 1995;75(5):617-20. https://doi.org/10.1016/s0002-9149(99)08629-8 PMid:887390

13. Hedén B, Ripa R, Persson E, Song Q, Maynard C, Leibrandt P, et al. A modified Anderson-Wilkins electrocardiographic acuteness score for anterior or inferior myocardial infarction. Am Heart J. 2003;146(5):797-803. https://doi.org/10.1016/s0002-7870(03)00404-6 PMid:14597927

14. Fakhri Y, Sejersten M, Schoos MM, Hansen HS, Dubois-Rande JL, Hall TS, et al. Electrocardiographic scores of severity and acuteness of myocardial ischemia predict myocardial salvage in patients with anterior ST-segment elevation myocardial infarction. J Electrocardiol. 2017;51(2):195-202. https://doi.org/10.1016/j.jelectrocard.2017.12.010 PMid:29174706

15. Esmaeilzadeh M, Parsaei M, Maleki M. The role of echocardiography in coronary artery disease and acute myocardial infarction. J Tehran Heart Cent. 2013;8(1):1-13. PMid:23646042

16. Sia YT, O'Meara E, Ducharme A. Role of echocardiography in acute myocardial infarction. Curr Heart Fail Rep. 2008;5(4):189-96. https://doi.org/10.1007/s11897-008-0029-6 PMid:19032913

17. White HD, Norris RM, Brown MA, Brandt PW, Whitlock RM, Wild Cj. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation. 1987;76(1):44-51. https://doi.org/10.1161/01.cir.76.1.44 PMid:3594774

18. Meller JE, Hillis GS, Oh JK, Reeder GS, Gersh BJ, Pellikka PA. Wall motion score index and ejection fraction for risk stratification after acute myocardial infarction. Am J Heart. 2006;151(2):419-25. https://doi.org/10.1016/j.ahj.2005.03.042 PMid:16442909

19. Gupta D, Agrawal P, Nohria S, Singh A. To study the correlation of location of myocardial infarction according to ECG and echocardiography. IOSR J Dent Med Sci. 2015;14(8):12-5.

20. Pandit A. Hypertension and myocardial infarction: A study and review. J Cardiol Clin Res. 2017;5(6):1118-23.

21. Pedrinelli R, Ballo P, Fiorentini C, Denti S, Galdersini M, Gianau A, et al. Hypertension and acute myocardial infarction: An overview. J Cardiovasc Med. 2012;13(3):194-202. https://doi.org/10.2459/jcm.0b013e32834158e2 PMid:22317927

22. Konstantinou K, Tsiovfis C, Kouvellis A, Mantzouranis M, Kasiaikogias A, Doumas M, et al. Hypertension and patients with acute coronary syndrome: Putting blood pressure levels into perspective. J Clin Hypertens (Greenwich). 2019;21(8):1135-43. https://doi.org/10.1111/jch.13622 PMid:31301119

23. Mach F, Baignet C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: Lipid modification to reduce cardiovascular risk. Eur Heart J. 2020;41(1):111-88. https://doi.org/10.15889/1560-4071-2020-3826 PMid:31504418

24. Klimchak AC, Patel MY, Iorga SR, Kulkarni N, Wong LD. Lipid treatment and goal attainment characteristics among persons with atherosclerotic cardiovascular disease in the United States. Am J Prev Cardiol. 2020;1:100010. https://doi.org/10.1016/j.ajpc.2020.100010

25. Ballarino P, Cervellin G, Trucchi C, Almonte F, Bertini A, Bonfanti L, et al. An Italian registry of chest pain patients in the emergency department; Clinical predictors of acute coronary syndrome. Minerva Med. 2020;111(2):120-32. https://doi.org/10.23736/s0026-4806.20.06472-1 PMid:32338841

26. Backer GD, Jankowski P, Kotseva K, Mirrakhimov E, Reiner Ž, Wladimirova A, et al. Atherosclerotic cardiovascular disease in the United States. Circulation. 2018;138(20):2230-51. https://doi.org/10.1161/circulationaha.118.035238 PMid:30154483

27. Vanlukis M, Zafirovska B, Antova E, Pocesta B, Shehu E, Taravari H, et al. Hypertension and acute myocardial infarction: A study and review. J Cardiol Clin Res. 2017;5(6):1118-23.

28. Kurt NG, Orak M, Ustundag M. Relation between stress hyperglycemia and mortality in patients with acute myocardial infarction. An Italian registry of chest pain patients in the emergency department; Clinical predictors of acute coronary syndrome. Minerva Med. 2020;111(2):120-32. https://doi.org/10.23736/s0026-4806.20.06472-1 PMid:32338841

29. Simjuru N, Núñez J, Blasco ML, Miñanab G, Martínez-Maicasa H, Carbonell N, et al. Prognostic implications of stress hyperglycaemia in acute ST elevation myocardial infarction. Prospective observational study. Rev Esp Cardiol. 2011;64(3):201-7. https://doi.org/10.1016/j.rec.2010.08.005
Serafimov et al. Modified Anderson-Wilkins Acuteness Score in Prognostication of Patients with Acute Myocardial Infarction

30. Marfella R, Siniscalchi M, Esposito K, Sellitto A, De Fanis U, Romano C, et al. Effects of stress hyperglycemia on acute myocardial infarction: Role of inflammatory immune process in functional cardiac outcome. Diabetes Care. 2003;26(11):3129-35. https://doi.org/10.2337/diacare.26.11.3129

31. Cinar H, Avci A, Gulen M, Avci BS, Comertpay E, Satar S. Does stress hyperglycemia affect mortality? Acute myocardial infarction-case control study. Arch Med Sci Atheroscler Dis. 2019;4(1):e201-7. https://doi.org/10.5114/amsad.2019.87303

PMid:31538125

32. Dixon DL, Abbate A. Potassium levels in acute myocardial infarction: Definitely worth paying attention to. Eur Heart J Cardiovasc Pharmaco. 2015;1(4):252-3. https://doi.org/10.1093/ehjcvp/pvz029

PMid:27532448

33. Madias JE, Shah B, Chintalapally G, Chalavarya G, Madias NE. Admission serum potassium in patients with acute myocardial infarction: Its correlates and value as a determinant of in-hospital outcome. Chest. 2000;118(4):904-13. https://doi.org/10.1378/chest.118.4.904

PMid:11035655

34. Goyal A, Spertus JA, Gosch K, Venkitachalam L, Jones PG, Van den Berghe G, et al. Serum potassium levels and mortality in acute myocardial infarction. JAMA. 2012;307(2):157-64. https://doi.org/10.1001/jama.2011.1967

PMid:22235086

35. Jose P, Skali H, Anavekar N, Tomson C, Krumholz HM, Rouleau JL, et al. Increase in creatinine and cardiovascular risk in patients with systolic dysfunction after myocardial infarction. J Am Soc Nephrol. 2006;17(10):2886-91. https://doi.org/10.1681/asn.2006010063

PMid:16928807

36. Cakar MA, Gunduz H, Vatan MB, Kocayigit I, Akdemir R. The effect of admission creatinine levels on one-year mortality in acute myocardial infarction. Sci World J. 2012;2012:186495. https://doi.org/10.1100/2012/186495

PMid:22619619

37. Cambou JP, Simon T, Mulak G, Bataille V, Danchin N. The French registry of acute ST elevation or non-ST-elevation myocardial Infarction (FAST-MI): Study design and baseline characteristics. Arch Mal Coeur Vaiss. 2007;100(6-7):524-34. https://doi.org/10.1136/heartjnl-2012-301700

PMid:17893635

38. Fakhr Y, Schoos MM, Sejersten M, Ersbøll M, Valeur N, Keber L, et al. Prehospital electrocardiographic acuteness score of ischemia is inversely associated with neurohormonal activation in STEMI patients with severe ischemia. J Electrocardiol. 2017;50(1):90-6. https://doi.org/10.1016/j.jelectrocard.2016.11.002

PMid:27887720