The importance of two metabolic syndrome diagnostic criteria and body fat distribution in predicting clinical severity and prognosis of acute myocardial infarction

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Abstract

Introduction: The interrelation between metabolic syndrome (MetS) (the revised National Cholesterol Education Program-Adult Treatment Panel III (NCEP-ATP III) and International Diabetes Federation (IDF)) and obesity indices in predicting clinical severity and prognosis of acute ST-elevation myocardial infarction (STEMI) is insufficiently known.

Material and methods: This prospective study included 250 acute STEMI patients treated with primary percutaneous coronary intervention. The patients with/without MetS were analyzed by baseline (medical history, demography and obesity indices: overall – body mass index (BMI) vs. central – body adiposity index (BAI), conicity index (Cindex), visceral adiposity index (VAI), waist circumference (WC), waist-to-hip (WHR) and waist-to-height ratio (WHtR)), severity (clinical presentation, laboratory, echocardiography, coronary angiography and in-hospital complications) and prognostic parameters (major adverse cardiovascular events and sick leave duration during 12-month follow-up).

Results: There were 136 (54.4%) and 147 (58.8%) patients with MetS (NCEP-ATP III) and MetS (IDF), respectively. MetS (NCEP-ATP III) increased the risk of > 1 significantly stenosed coronary artery (CA), very high BAI increased the risk of dyspnea, Cindex > 1.25/1.18 increased the risk of total in-hospital complications, increased VAI increased the risk of coronary segment 3 significant stenosis, WHR ≥ 0.90/0.85 increased the risk of proximal/middle coronary segments (especially of segment 1) significant stenosis, WHtR ≥ 63/58 increased the risk of heart failure, and the number of significantly stenosed CAs increased the risk of total MACE (p < 0.05).

Conclusions: MetS (NCEP-ATP III) and several central obesity indices are superior to BMI in predicting acute STEMI severity (clinical presentation, in-hospital complications, severity of coronary disease), while WC and MetS (IDF) have no influence on it. They all have no influence on prognosis.

Key words: anthropometry, metabolic syndrome, myocardial infarction, obesity, percutaneous coronary intervention.

Introduction

Metabolic syndrome (MetS) is defined as a group of interrelated factors (hyperglycemia, abdominal obesity, atherogenic dyslipidemia, hy-
pertension, prothrombotic and proinflammatory states), which significantly increases the risk of coronary artery disease (CAD) and other forms of atherosclerotic cardiovascular diseases (CVD), impaired fasting glucose and diabetes mellitus type 2 (DMT2), and cardiovascular and all-cause mortality [1, 2]. MetS is a worldwide problem with rapid growth. It could be explained by the parallel rise of obesity prevalence. Approximately one-quarter of adult Europeans have MetS, depending on geographic location, age and characteristics of the study population [3, 4]. Its prevalence increases with age, markedly from age 30, and peaks around age 60–75, but generally with no gender differences [5–7].

The revised National Cholesterol Education Program – Adult Treatment Panel III (NCEP-ATP III) definition is the most widely accepted and cited in the literature because it provides a relatively simple approach for the diagnosis of MetS [1]. According to the NCEP-ATP III and thereafter published International Diabetes Federation (IDF) diagnostic criteria, one of the MetS constitutive parameters is central obesity [1]. It correlates with excessive visceral fat, which is directly associated with insulin resistance and compensatory hyperinsulinemia, dyslipidemia and inflammatory states that synergistically lead to smooth muscle cell proliferation, calcium and cholesterol ester deposition in the artery, and finally to atherosclerotic vascular disease [8]. Thus, it is not surprising that central obesity indices, i.e. waist circumference (WC), waist-to-hip (WHR) and especially waist-to-height ratio (WHtR), are reported as stronger predictors of CVD risk than body mass index (BMI), which is a measure of overall obesity [9–11].

MetS is common among patients with CAD. Moreover, it is highly prevalent among patients with acute ST-elevation myocardial infarction (STEMI) [12–14].

The main objective of this study, performed on patients with acute STEMI treated with primary percutaneous coronary intervention (PCI), was to investigate the interrelation between MetS (diagnosed by using the revised NCEP-ATP III and IDF diagnostic criteria) and various obesity indices in predicting clinical severity and prognosis of acute STEMI.

Material and methods

We prospectively analyzed 250 consecutive patients with acute STEMI treated with primary PCI at the Department of Cardiology, Sestre milosrdnice University Hospital Center, Zagreb, Croatia (September 2011 – September 2012). The study was approved by the Sestre milosrdnice University Hospital Center Ethics Committee. The inclusion criteria were as follows: presenting within 12 h from the onset of symptoms (history of chest pain/discomfort lasting for 10–20 min or more, not responding fully to nitroglycerine), persistent ST-segment elevation on electrocardiography (ECG) in at least two consecutive leads or (presumed) new left bundle branch block (LBBB), and elevated cardiac laboratory biomarkers (cardiac troponin T (cTnT) and creatine kinase (CK)). The diagnosis of acute STEMI was established and primary PCI performed using the European Society of Cardiology criteria [15, 16]. After primary PCI, patients were classified into two groups (with/without MetS) which were analyzed by baseline, as well severity and prognostic parameters of acute STEMI.

Diagnosis of MetS and its components

MetS (IDF) was diagnosed in the presence of central obesity (WC ≥ 94/80 cm) and at least two of the next four parameters [1], as follows:

1) hypertriglyceridemia: triglycerides (TG) ≥ 150 mg/dl (1.7 mmol/l), or on medication for elevated TG;
2) low high-density lipoprotein (HDL) cholesterol: HDL < 40 mg/dl (1.04 mmol/l) in males or HDL < 50 mg/dl (1.29 mmol/l) in females, or on medication for low HDL;
3) arterial hypertension: blood pressure ≥ 130/85 mm Hg, or on medication for hypertension;
4) hyperglycemia: fasting plasma glucose ≥ 100 mg/dl (5.5 mmol/l), or on medication for hyperglycemia.

MetS (NCEP-ATP III) was diagnosed in the presence of any of three or more of the following five parameters: central obesity (WC ≥ 102/88 cm), hypertriglyceridemia, low HDL, arterial hypertension and hyperglycemia [1].

Baseline parameters

Baseline demographic and medical history parameters included gender, age, smoking, known family history of cardiovascular events (MI, stroke), previous MI, previous PCI and coronary artery bypass grafting (CABG). Data concerning long-term therapy before and after admission, including aspirin, β-blockers, calcium-channel blockers, angiotensin-converting enzyme inhibitors (ACEIs), angiotensin II receptor blockers (ARBs), diuretics and statins, were also collected. According to current guidelines, all patients with acute STEMI and who had undergone PCI received dual antiaggregation therapy (aspirin and clopidogrel) and statin.

Anthropometric baseline data included body adiposity index (BAI), BMI, conicity index (Cindex), visceral adiposity index (VAI), WC, WHR and WHtR [1, 9, 17–22]. Body mass index was calculated by dividing body weight in kilograms by the square
of body height in meters (kg/m²) and classified as BMI < 25.0 (normal weight), BMI 25.0–29.9 kg/m² (overweight) and BMI ≥ 30.0 kg/m² (obesity). The WC was measured in the standing position at the midpoint between the lowest rib and the iliac crest. The cutoff values were ≥ 102/88 cm (central obesity) for males/females, respectively. The hip circumference was measured in the standing position between both major femoral trochanters. The WHR was calculated by dividing WC by hip circumference. The cutoff values were ≥ 0.90/0.85 (central obesity) for males/females, respectively. The VAI was calculated as follows: VAI (males) = (WC/39.68 + (1.88 × BMI)) × (TG/1.03) × (1.89 × BMI) and VAI (females) = (WC/36.58 + (1.89 × BMI)) × (TG/0.81) × (1.52/HDL) [19]. The cut-off points for normal (normal weight) and increased VAI (central obesity) are ≥ 0.90/0.85 (central obesity) for males/females, respectively. Each principal vascular segment was assigned a multiplier in accordance with the functional significance of the myocardial area supplied by that segment: the left main coronary artery × 5; the proximal segment of the LAD × 2.5; the proximal segment of the LCX × 2.5; the mid-segment of the LCX × 2.5; the mid-segment of the LAD × 1.5; the RCA, the distal segment of the LAD, the OM artery × 1; the posterolateral artery, and the OM artery × 0.5. Severe CAD was defined as having a Gscore of 20 or more [24]. Secondary parameters

Severities of acute STEMI were estimated by clinical presentation (angina pectoris, dyspnea, and length of hospital stay), in-hospital complications (arrhythmias, heart failure, cardiogenic shock, cardiac arrest, mechanical ventilation, reinfarction, repeated PCI, mortality, and total in-hospital complications), coronary angiography, laboratory (maximal cTnT and CK) and echocardiography (left ventricular ejection fraction, LVEF) findings.

Coronary angiography was performed by applying a monoplane system (Axiom Artis, Siemens, Erlangen, Germany) using a common technique as recommended in the current guidelines [16]. Patients received 70 IE/kg of unfractionated heparin, 300 mg of aspirin, a loading dose of 600 mg of clopidogrel, and a GPIIb/IIIa inhibitor according to the judgment of the interventional cardiologist. Coronary arteries (CAs) stenosis of more than 50% was considered clinically significant. It was measured with the system software at all patients. The WHR was calculated by dividing WC by hip circumference. The cutoff values were ≥ 1.25 and 1.18 (central obesity) for males/females, respectively. The BAI was calculated using the equation suggested by Bergman et al.: BAI = (hip circumference/body height^1.5) – 18 [19]. The cut-off values of overweight for males/females are 21/33% (20–39 years), 23/35% (40–59 years) and 25/38% (60–79 years), while cut-off values of central obesity are 26/39% (20–39 years), 29/41% (40–59 years) and 31/43% (60–79 years) [20]. The VAI was calculated by using the following formula: VAI (males) = (WC/39.68 + (1.88 × BMI)) × (TG/1.03) × (1.89 × BMI) and VAI (females) = (WC/36.58 + (1.89 × BMI)) × (TG/0.81) × (1.52/HDL) [21]. The cut-off points for normal (normal weight) and increased VAI (central obesity) are 2.52 (age < 30 years), 2.23 (age ≥ 30 and < 42 years), 1.92 (age ≥ 42 and < 52 years), 1.93 (age ≥ 52 and ≥ 66 years) and 2.00 (age ≥ 66 years) [22].

Severity parameters

The severities of acute STEMI were estimated by clinical presentation (angina pectoris, dyspnea, and length of hospital stay), in-hospital complications (arrhythmias, heart failure, cardiogenic shock, cardiac arrest, mechanical ventilation, reinfarction, repeated PCI, mortality, and total in-hospital complications), coronary angiography, laboratory (maximal cTnT and CK) and echocardiography (left ventricular ejection fraction, LVEF) findings. The Gensini score (Gscore) was computed by assigning a severity score to each coronary stenosis according to the degree of luminal narrowing and its geographic importance [24]. Reduction in the lumen diameter and the roentgenographic appearance of concentric lesions and eccentric plaques were evaluated (reductions of 25%, 50%, 75%, 90%, 99%, and complete occlusion, were given Gscores of 1, 2, 4, 8, 16, and 32, respectively). Each principal vascular segment was assigned a multiplier in accordance with the functional significance of the myocardial area supplied by that segment: the left main coronary artery × 5; the proximal segment of the LAD × 2.5; the proximal segment of the LCX × 2.5; the mid-segment of the LAD × 1.5; the RCA, the distal segment of the LAD, the postero-lateral artery, and the OM artery × 1; and others × 0.5. Severe CAD was defined as having a Gscore of 20 or more [24]. Serum CK activity was measured by spectrophotometry (Olympus 680, Beckman Coulter Inc., California, USA). Serum cTnT levels were measured by electrochemiluminescence (ECL) assay (Cobas e411, Roche Diagnostics, Sussex, UK). During hospitalization, echocardiography was performed in all patients (Acuson Sequoia 512, Siemens, Munich, Germany) according to clinical standards and current echocardiography guidelines [25].

Prognostic parameters

During hospitalization, 19 (7.6%) patients died and 231 (92.4%) patients were included in the follow-up of 12 (3–12) months. The prognosis of acute STEMI was estimated using major adverse cardiovascular events (MACE) parameters (reinfarction, CAs restenosis and new stenosis, cardiac and non-cardiac rehospitalization, stroke, urgent CABG, mortality, total MACE). Data were collected by medical examination, checking medical doc-
umentation, or telephone contact with patients, family members or home physicians. Also, during the same follow-up period, we collected data on sick leave duration (SLD) in the working population.

Statistical analysis
Qualitative data were presented in absolute number and percentage. We used the χ² test with Yates correction. Quantitative data were presented as median and corresponding interquartile range. Differences between the two groups were tested by Mann-Whitney U test. The χ² test and univariate or multivariate logistic regression analysis were used to investigate the relationship between one dependent and one or several independent variables (after their adjustment) that may influence or predict the value of the dependent variable. The level of statistical significance was set at p < 0.05. Processing was done using the Statistica 6.0 for Windows software.

Results
Patients with acute STEMI (aged 62 (25–92) years) were more frequently male (73.2% vs. 26.8%, p < 0.05) and had high rates of central obesity (WHR ≥ 0.90/0.85 (88.8%), WC ≥ 94/80 cm (83.6%), Cindex > 1.25/1.18 (80.8%), WC ≥ 102/88 cm (59.6%), increased VAI (51.8%), WHTR ≥ 63/58 (32.4%) and very high BAI (10.2%), dyslipidemia (76.0%) and hypertension (72.4%), as well as lower rates of overall obesity (BMI ≥ 30.0 kg/m² (28.8%)) and hyperglycemia (24.4%) (Table I).

Before admission, the most frequently prescribed drugs were ACEIs/ARBs (38.8%) and calcium channel blockers (CCBs) (22.4%), then β-blockers (21.2%) and diuretics (6.8%). After primary PCI and during follow-up, all patients were taking dual antiaggregations therapy and statins, while the most frequently prescribed drugs were ACEIs/ARBs (72.1%) and β-blockers (59.7%), followed by diuretics (13.9%) and CCBs (8.7%). Higher rates of drug consumption were recorded in patients with MetS (p < 0.05).

Furthermore, we obtained the following results:
1) Among the total of 250 patients, there were 147 (58.8%) and 103 (41.2%) patients with and without MetS, respectively. MetS (IDF) patients were more frequently male and had higher rates of arterial hypertension, dyslipidemia, hyperglycemia and WC ≥ 94/80 cm, which was expected as they are diagnostic criteria for MetS. Furthermore, they had higher rates of overall (BMI ≥ 30.0 kg/m²) and central obesity (very high BAI, Cindex > 1.25/1.18, increased VAI, WHTR ≥ 63/58), as well as wider stent diameter (Tables I–IV). There were no other significant differences in baseline or severity parameters, or in all prognostic parameters (MACE and SLD), between the two groups.
2) The univariate and multivariate logistic regression analysis for investigating the influence of MetS (NCEP-ATP III and IDF), its constitutive parameters and obesity indices on clinical severity and prognosis of acute STEMI led to the following conclusions (Tables V–VII):

- MetS (NCEP-ATP III) independently increased the risk of >1 significantly stenosed CAs (odds ratio (OR) = 1.72, 95% confidence interval (CI): 1.04–2.84, p = 0.034) (n = 250 (100%) patients);
- Very high BAI adjusted for BMI < 25.0 kg/m² and BMI 25.0–29.9 kg/m² increased the risk of dyspnea (OR = 3.06, 95% CI: 1.13–8.27, p = 0.027) (n = 225 (90.0%) patients);
- Cindex > 1.25/1.18 adjusted for MetS (NCEP-ATP III) and WHTR ≥ 63/58 increased the risk of total in-hospital complications (OR = 2.64, 95% CI: 1.20–5.77, p = 0.016) (n = 250 (100%) patients);
- WHR ≥ 0.90/0.85 independently increased the risk of significant stenosis of the coronary segment 1 (OR = 3.34, 95% CI: 1.12–9.96, p = 0.031) and proximal/middle CAs segments (OR = 4.27, 95% CI: 1.58–11.56, p = 0.004) (n = 249 (99.6%) patients);
- WHTR ≥ 63/58 adjusted for hyperglycemia increased the risk of heart failure (OR = 2.05, 95% CI: 1.13–3.71, p = 0.017) (n = 250 (100%) patients);
- Increased VAI and WHTR 53/49–62/57 increased (OR = 2.69, 95% CI: 1.01–7.16, p = 0.047) and reduced (OR = 0.40, 95% CI: 0.17–0.95, p = 0.037) the risk of coronary segment 3 significant stenosis, respectively. After adjustment, we found that increased VAI increases the risk of the coronary segment 3 significant stenosis (OR = 2.66, 95% CI: 1.00–7.10, p = 0.049) (n = 227 (90.8%) patients); and
- MetS and obesity indices had no influence on prognosis (MACE and SLD). But, the number of significantly stenosed CAs adjusted for LVEF and distal coronary segments stenosis increased the risk of total MACE (OR = 1.79, 95% CI: 1.17–2.77, p = 0.008) during 12-month follow-up (n = 228 (91.2%) patients).

Discussion
This prospective study investigated the importance of MetS (NCEP-ATP III, IDF) and various obesity indices (BAI, BMI, Cindex, VAI, WC, WHR and WHTR) in predicting clinical severity and prognosis of acute STEMI urgently treated with primary PCI. Among them, Cindex, BAI, VAI and WHTR were used for the first time.
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Several studies have reported that MetS (NCEP-ATP III) increases the risk of heart failure, in-hospital mortality and total in-hospital complications, but not of target vessel revascularization and MACE during 12-month follow-up after primary PCI [13, 14, 26–30]. Zeller et al. concluded that hyperglycemia (among MetS components) was the main correlate of the risk of development of severe heart failure during AMI [14, 31].

Table I. Baseline characteristics of patients with acute STEMI

| Findings | Parameters | MetS (IDF) (n = 147) | No MetS (IDF) (n = 103) | Total (n = 250) | P-value† |
|----------|------------|----------------------|-------------------------|-----------------|----------|
| Demographic, medical history and anthropometric data | Men, n (%)² | 103 (70.1) | 80 (77.7) | 183 (73.2) | 0.234 |
| | Women, n (%)² | 44 (29.9) | 23 (22.3) | 67 (26.8) | 0.234 |
| | Age (years)³ | 63 (25–92) | 60 (39–91) | 62 (25–92) | 0.233 |
| | Hypertension, n (%)² | 139 (94.6) | 42 (40.8) | 181 (72.4) | < 0.001 |
| | Dyslipidemia, n (%)² | 133 (90.5) | 57 (55.3) | 190 (76.0) | < 0.001 |
| | Hyperglycemia, n (%)² | 52 (35.4) | 9 (8.7) | 61 (24.4) | < 0.001 |
| | Smoking, n (%)² | 76 (51.7) | 53 (51.5) | 129 (51.6) | 0.928 |
| | Family history (MI/stroke), n (%)³ | 69 (46.9) | 38 (36.9) | 107 (42.8) | 0.147 |
| | Previous MI, n (%)³ | 17 (11.6) | 5 (4.9) | 22 (8.8) | 0.106 |
| | Previous PCI, n (%)³ | 17 (11.6) | 6 (5.8) | 23 (9.2) | 0.186 |
| | Previous CABG, n (%)³ | 1 (0.7) | 0 (0) | 1 (0.4) | – |
| | WC ≥ 94/80 (IDF) cm, n (%)² | 147 (100) | 62 (60.2) | 209 (83.6) | < 0.001 |
| | WC ≥ 102/88 (ATP) cm, n (%)² | 117 (79.6) | 32 (31.1) | 149 (59.6) | < 0.001 |
| | MetS (ATP), n (%)² | 131 (89.1) | 5 (4.9) | 136 (54.4) | < 0.001 |
| | BMI < 25.0 kg/m², n (%)² | 20 (13.6) | 40 (38.8) | 60 (24.0) | < 0.001 |
| | BMI 25.0–29.9 kg/m², n (%)² | 64 (43.5) | 54 (52.4) | 118 (47.2) | 0.209 |
| | BMI ≥ 30.0 kg/m², n (%)² | 63 (42.9) | 9 (8.7) | 72 (28.8) | < 0.001 |
| | WHR ≥ 0.90/0.85, n (%)² | 135 (91.8) | 87 (84.5) | 222 (88.8) | 0.106 |
| | WHtR < 53/49, n (%)² | 5 (3.4) | 37 (35.9) | 42 (16.8) | < 0.001 |
| | WHtR 53/49–62/57, n (%)² | 78 (53.1) | 49 (47.6) | 127 (50.8) | 0.468 |
| | WHtR ≥ 63/58, n (%)² | 64 (43.5) | 17 (16.5) | 81 (32.4) | < 0.001 |
| | Cindex > 1.25/1.18, n (%)² | 132 (89.8) | 70 (68.0) | 202 (80.8) | < 0.001 |
| | BAI (normal), n (%)² | 60 (45.1) | 54 (58.7) | 114 (50.7) | 0.062 |
| | BAI (high), n (%)² | 54 (40.6) | 34 (37.0) | 88 (39.1) | 0.680 |
| | BAI (very high), n (%)² | 19 (14.3) | 4 (4.3) | 23 (10.2) | 0.028 |
| | Normal VAI, n (%)² | 56 (41.2) | 54 (58.7) | 110 (46.2) | 0.014 |
| | Increased VAI, n (%)² | 80 (58.8) | 38 (41.3) | 118 (51.8) | 0.014 |

ATP – NCEP-ATP III diagnostic criteria, BAI – body adiposity index (cut-off values of overweight for males/females are 21/33% (age 20–39 years), 23/35% (age 40–59 years) and 25/38% (age 60–79 years); cut-off values of central obesity are 26/39% (age 20–39 years), 29/41% (age 40–59 years) and 31/43% (age 60–79 years)), BMI – body mass index, CABG – coronary artery bypass graft, Cindex – conicity index, IDF – IDF diagnostic criteria, MetS – metabolic syndrome, MI – myocardial infarction, PCI – percutaneous coronary intervention, STEMI – ST-elevation myocardial infarction, VAI – visceral adiposity index (cut-off points are 2.52 (age < 30 years), 2.23 (age 30–41 years), 1.93 (age 42–51 years), 1.92 (age 52–65 years) and 2.00 (age ≥ 66 years)), WC – waist circumference, WHR – waist-to-hip ratio, WHtR – waist-to-height ratio. †Statistical significance defined as p < 0.05. ²Data are expressed as absolute number and percentage (%), compared with χ² test. ³Data are expressed as median and range, compared with Mann-Whitney U test.

In our previous study [32], MetS (NCEP-ATP III) patients had longer hospitalization and severe CAD. While MetS increased the risk of > 1 significantly stenosed CAs and total in-hospital complications, none of the MetS components per se (except hyperglycemia, which increased the risk of heart failure) had a significant influence on clinical severity or prognosis. Our results confirmed the most important fact that MetS (NCEP-ATP III)
Table II. Severity of acute STEMI

| Findings | Parameters | MetS (IDF) (n = 147) | No MetS (IDF) (n = 103) | Total (n = 250) | P-value† |
|----------|------------|----------------------|-------------------------|-----------------|----------|
| Clinical presentation | Angina pectoris, n (%)‡ | 144 (98.0) | 101 (98.1) | 245 (98.0) | 0.686 |
| | Dyspnea, n (%)‡ | 43 (29.3) | 32 (31.1) | 75 (30.0) | 0.866 |
| | Hospital stay [days]§ | 9 (1–31) | 8 (1–32) | 9 (1–32) | 0.196 |
| In-hospital complications | Arrhythmias, n (%)‡ | 29 (19.7) | 14 (13.6) | 43 (17.2) | 0.274 |
| | Heart failure, n (%)‡ | 40 (27.2) | 24 (23.3) | 64 (25.6) | 0.582 |
| | Cardiogenic shock, n (%)‡ | 9 (6.1) | 9 (6.7) | 18 (7.2) | 0.590 |
| | Cardiac arrest, n (%)‡ | 22 (15.0) | 14 (13.6) | 36 (14.4) | 0.903 |
| | Mechanical ventilation, n (%)‡ | 6 (4.1) | 4 (3.9) | 10 (4.0) | 0.803 |
| | Reinfarction, n (%)‡ | 1 (0.7) | 0 (0) | 1 (0.4) | – |
| | Repeated PCI, n (%)‡ | 3 (2.0) | 1 (1.0) | 4 (1.6) | 0.880 |
| | Mortality, n (%)‡ | 9 (6.1) | 10 (9.7) | 19 (7.6) | 0.418 |
| | Total, n (%)‡ | 66 (44.9) | 38 (36.9) | 104 (41.6) | 0.257 |
| Laboratory | Maximal cTnT [ng/ml]§ | 2.92 (0.02–10.0) | 3.21 (0.02–10.0) | 3.11 (0.02–10.0) | 0.414 |
| | Maximal CK [U/l]§ | 1815 (25–14094) | 1914 (70–15617) | 1867 (25–15617) | 0.420 |
| ECHO | LVEF (%)§ | 50 (25–70) | 52 (30–76) | 50 (25–76) | 0.904 |
| Coronary angiography | Number of stenosed CAs† | 2 (1–4) | 1 (1–4) | 2 (1–4) | 0.278 |
| | > 1 stenosed CAs, n (%)‡ | 80 (54.4) | 48 (46.6) | 128 (51.2) | 0.276 |
| | Number of stents† | 1 (1–3) | 1 (1–4) | 1 (1–4) | 0.145 |
| | Diameter of stents [mm]§ | 3.5 (2.3–4.0) | 3.3 (2.8–4.0) | 3.5 (2.3–4.0) | 0.035 |
| | Length of stents [mm]§ | 20 (12–38) | 20 (8–38) | 20 (8–38) | 0.819 |
| | Proximal/middle CSS, n (%)‡ | 134 (91.2) | 92 (90.2) | 226 (90.8) | 0.972 |
| | Distal CSS, n (%)‡ | 61 (41.5) | 36 (35.3) | 97 (39.0) | 0.393 |
| | Gscore ≥ 20, n (%)‡ | 129 (87.8) | 89 (86.4) | 218 (87.2) | 0.903 |

CAs – coronary arteries, CK – creatine kinase, CSS – coronary segment stenosis, cTnT – cardiac troponin T, ECHO – echocardiography, Gscore – Gensini score, IDF – IDF diagnostic criteria, LVEF – left ventricular ejection fraction, MetS – metabolic syndrome, PCI – percutaneous coronary intervention, STEMI – ST-elevation myocardial infarction. †Statistical significance defined as p < 0.05. ‡Data are expressed as absolute number and percentage (%), compared with c² test. §Data are expressed as median and range, compared with Mann-Whitney U test.

as a pathophysiological concept is relevant and superior to its components in risk prediction of patients with acute STEMI urgently treated with primary PCI.

In this study, MetS (NCEP-ATP III) still increased the risk of > 1 significantly stenosed CAs, but Cindex was a stronger predictor of total in-hospital complications.

Ekmecki et al. reported MetS (IDF) prevalence of 45.1% in patients with acute STEMI [33]. There are insufficient literature data about the influence of MetS (IDF) on in-hospital outcomes and prognosis in patients with acute coronary syndrome, especially with acute STEMI. The presence of MetS (IDF) or any of the MetS components is not an independent predictor of in-hospital adverse cardiovascular events in acute STEMI treated with primary PCI [33]. Al Suwaidi et al. performed an analysis of the Gulf Registry of Acute Coronary Events (Gulf RACE) and concluded that STEMI patients with MetS (IDF) have double the risk of stroke, recurrent myocardial ischemia and MI. But, they emphasized that a long-term follow-up period is needed to confirm their findings [34]. We had 58.8% of patients with MetS (IDF), who only had wider stents in comparison with controls. MetS (IDF) and its constitutive parameters had no independent influence on clinical severity and prognosis during 12-month follow-up. This could be explained by the fact that increased WC
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### Table III. Analysis of CA segments with significant stenosis in patients with acute STEMI

| Findings                  | Parameters                | MetS (IDF) (n = 147) | No MetS (IDF) (n = 103) | Total (n = 250) | P-value† |
|---------------------------|---------------------------|-----------------------|-------------------------|-----------------|----------|
| Proximal/middle CA segments | Segment 1, n (%)§         | 51 (34.7)             | 32 (31.4)               | 83 (33.3)       | 0.682    |
|                           | Segment 2, n (%)§         | 34 (23.1)             | 21 (20.6)               | 55 (22.1)       | 0.749    |
|                           | Segment 5, n (%)§         | 5 (3.4)               | 5 (4.9)                 | 10 (4.0)        | 0.791    |
|                           | Segment 6, n (%)§         | 55 (37.4)             | 45 (44.1)               | 100 (40.2)      | 0.353    |
|                           | Segment 7, n (%)§         | 37 (25.2)             | 25 (24.5)               | 62 (24.9)       | 0.976    |
|                           | Segment 9, n (%)§         | 12 (8.2)              | 6 (5.9)                 | 18 (7.2)        | 0.664    |
|                           | Segment 11, n (%)§        | 36 (24.5)             | 23 (22.5)               | 59 (23.7)       | 0.839    |
|                           | Segment 12, n (%)§        | 19 (12.9)             | 7 (6.9)                 | 26 (10.4)       | 0.184    |
|                           | Total, n (%)§             | 134 (91.2)            | 92 (90.2)               | 226 (90.8)      | 0.972    |
| Distal CA segments        | Segment 3, n (%)§         | 13 (8.8)              | 13 (12.7)               | 26 (10.4)       | 0.436    |
|                           | Segment 4, n (%)§         | 6 (4.1)               | 6 (5.9)                 | 12 (4.8)        | 0.725    |
|                           | Segment 8, n (%)§         | 18 (12.2)             | 6 (5.9)                 | 24 (9.6)        | 0.146    |
|                           | Segment 10, n (%)§        | 5 (3.4)               | 1 (1.0)                 | 6 (2.4)         | 0.421    |
|                           | Segment 13, n (%)§        | 10 (6.8)              | 6 (5.9)                 | 16 (6.4)        | 0.977    |
|                           | Segment 14, n (%)§        | 6 (4.1)               | 3 (2.9)                 | 9 (3.6)         | 0.897    |
|                           | Segment 15, n (%)§        | 13 (8.8)              | 8 (7.8)                 | 21 (8.4)        | 0.962    |
|                           | Segment 16, n (%)§        | 6 (4.1)               | 5 (4.9)                 | 11 (4.4)        | 0.997    |
|                           | Total, n (%)§             | 61 (41.5)             | 36 (35.3)               | 97 (39.0)       | 0.393    |

CA – coronary artery, IDF – IDF diagnostic criteria, STEMI – ST-elevation myocardial infarction. †Statistical significance defined as p < 0.05. §Data are expressed as absolute number and percentage (%), compared with χ² test.

### Table IV. Prognosis of acute STEMI

| Findings                  | Parameters                | MetS (IDF) (n = 138) | No MetS (IDF) (n = 93) | Total (n = 231) | P-value† |
|---------------------------|---------------------------|-----------------------|-------------------------|-----------------|----------|
| Follow-up (months)§       |                           | 12 (3–12)             | 12 (4–12)               | 12 (3–12)       | 0.725    |
| MACE                      | Reinfarction, n (%)§      | 1 (0.7)               | 1 (1.1)                 | 2 (0.9)         | –        |
|                           | Restenosis, n (%)§        | 4 (2.9)               | 3 (3.2)                 | 7 (3.0)         | 0.796    |
|                           | New stenosis, n (%)§      | 5 (3.6)               | 2 (2.2)                 | 7 (3.0)         | 0.796    |
|                           | Cardiac rehospitalization, n (%)§ | 22 (15.9)     | 15 (16.1)               | 37 (16.1)       | 0.866    |
|                           | Non-cardiac rehospitalization, n (%)§ | 7 (5.1)       | 2 (2.2)                 | 9 (3.9)         | 0.430    |
|                           | Stroke, n (%)§            | 1 (0.7)               | 0 (0)                   | 1 (0.4)         | –        |
|                           | Urgent CABG, n (%)§       | 5 (3.6)               | 1 (1.1)                 | 6 (2.6)         | 0.435    |
|                           | Mortality, n (%)§         | 2 (1.5)               | 2 (2.2)                 | 4 (1.7)         | 0.904    |
|                           | Total, n (%)§             | 29 (21.0)             | 18 (19.4)               | 47 (20.4)       | 0.867    |
| Other                     | SLD [weeks]§              | 12 (2–52)             | 11 (1–48)               | 12 (1–52)       | 0.074    |

CABG – coronary artery bypass graft, IDF – IDF diagnostic criteria, MACE – major adverse cardiovascular events, MetS – metabolic syndrome, SLD – sick leave duration, STEMI – ST-elevation myocardial infarction. †Statistical significance defined as p < 0.05. ‡Data are expressed as median and range, compared with Mann-Whitney U test. §Data are expressed as absolute number and percentage (%), compared with χ² test.
### Table V. The influence of MetS, its components and obesity indices on clinical presentation (dyspnea, heart failure and total in-hospital complications) in patients with acute STEMI

| Parameters                        | Dyspnea  | P-value† | Heart failure | P-value† | Total in-hospital complications | P-value† |
|-----------------------------------|----------|----------|---------------|----------|----------------------------------|----------|
| MetS (NCEP-ATP III)              | 1.38 (0.80–2.40) | 0.245    | 1.71 (0.95–3.07) | 0.072    | 1.76 (1.05–2.94) | 0.031    |
| MetS (IDF)                        | 0.92 (0.53–1.59) | 0.758    | 1.23 (0.69–2.21) | 0.486    | 1.40 (0.83–2.33) | 0.207    |
| Hypertension                      | 1.60 (0.85–3.04) | 0.147    | 1.20 (0.62–2.28) | 0.590    | 1.49 (0.84–2.64) | 0.178    |
| Dyslipidemia                      | 0.81 (0.44–1.52) | 0.518    | 0.75 (0.39–1.42) | 0.370    | 0.70 (0.39–1.25) | 0.226    |
| Hyperglycemia                     | 1.31 (0.71–2.43) | 0.386    | 1.97 (1.05–3.70) | 0.033    | 1.64 (0.92–2.94) | 0.094    |
| WC ≥ 94/80 cm (IDF)              | 0.70 (0.35–1.41) | 0.316    | 1.08 (0.50–2.34) | 0.846    | 1.14 (0.57–2.25) | 0.715    |
| WC ≥ 102/88 cm (ATP)             | 1.30 (0.75–2.28) | 0.354    | 1.69 (0.93–3.10) | 0.086    | 1.52 (0.90–2.55) | 0.117    |
| BMI < 25.0 kg/m²                  | 2.00 (1.10–3.67) | **0.025** | 1.20 (0.63–2.31) | 0.578    | 1.20 (0.67–2.16) | 0.540    |
| BMI 25.0–29.9 kg/m²              | 0.51 (0.29–0.90) | **0.020** | 0.64 (0.36–1.14) | 0.132    | 0.76 (0.46–1.26) | 0.294    |
| BMI ≥ 30.0 kg/m²                 | 1.14 (0.63–2.05) | 0.670    | 1.42 (0.77–2.62) | 0.255    | 1.18 (0.68–2.05) | 0.562    |
| WHR ≥ 0.90/0.85                  | 0.75 (0.33–1.70) | 0.485    | 0.70 (0.30–1.63) | 0.402    | 1.32 (0.58–2.99) | 0.504    |
| WHR < 53/49                      | 1.37 (0.68–2.76) | 0.377    | 0.76 (0.34–1.69) | 0.498    | 0.50 (0.24–1.04) | 0.064    |
| WHR ≥ 53/49                      | 0.85 (0.50–1.46) | 0.562    | 0.58 (0.32–1.03) | 0.061    | 0.78 (0.47–1.29) | 0.326    |
| WHR ≥ 63/58                      | 0.97 (0.55–1.74) | 0.930    | 2.14 (1.19–3.84) | 0.011    | 2.00 (1.17–3.43) | 0.011    |
| Cindex > 1.25/1.18               | 1.19 (0.59–2.41) | 0.624    | 2.30 (0.98–5.43) | 0.057    | 3.30 (1.56–7.00) | 0.002    |
| BAI (normal)                     | 0.96 (0.54–1.73) | 0.902    | 1.18 (0.63–2.20) | 0.601    | 1.15 (0.67–1.98) | 0.599    |
| BAI (high)                       | 0.67 (0.36–1.23) | 0.195    | 0.62 (0.32–1.21) | 0.162    | 0.67 (0.38–1.17) | 0.159    |
| BAI (very high)                  | 2.71 (1.13–6.53) | 0.026    | 1.92 (0.76–4.81) | 0.167    | 1.85 (0.78–4.40) | 0.165    |
| Increased VAI                    | 1.08 (0.61–1.93) | 0.796    | 0.79 (0.43–1.45) | 0.446    | 0.86 (0.50–1.46) | 0.571    |

**ATP – NCEP-ATP III diagnostic criteria, BAI – body adiposity index (cut-off values of overweight for males/females are 21/33% (age 20–39 years), 23/35% (age 40–59 years) and 25/38% (age 60–79 years); cut-off values of central obesity are 26/39% (age 20–39 years), 29/41% (age 40–59 years) and 31/43% (age 60–79 years); BMI – body mass index, Cindex – conicity index, IDF – IDF diagnostic criteria, MetS – metabolic syndrome, STEMI – ST-elevation myocardial infarction, VAI – visceral adiposity index (cut-off points are 2.52 (age < 30 years), 2.23 (age 30–41 years), 1.92 (age 42–51 years), 1.93 (age 52–65 years) and 2.00 (age ≥ 66 years)), WC – waist circumference, WHR – waist-to-hip ratio, WHtR – waist-to-height ratio. †Statistical significance defined as p < 0.05. †Univariate logistic regression analysis – odds ratio [confidence interval].**

is an obligatory component in the IDF worldwide accepted definition of MetS [1]. Increased WC may have no role in development of angiographically significant CAD, which has been termed the central ‘obesity paradox’ [35]. The subcutaneous fat component, with a lower influence on atherogenesis, is probably mainly responsible for the paradoxical protective effect of abdominal obesity, whereas visceral fat has an opposite effect and increases the risk of significant angiographic CAD [14]. Visceral fat is associated with increased adipocytokine production, proinflammatory activity, deterioration of insulin sensitivity, increased risk of developing diabetes, dyslipidemia, hypertension, atherosclerosis, and higher mortality rate [36]. Zeller et al. did not find that WC reliably predicts outcomes in acute MI [14]. The measurement of WC does not add prognostic information for prediction of 6-month mortality or myocardial reinfarction in patients with acute MI [37]. Regarding the fact that WC alone does not help in distinguishing between subcutaneous and visceral (both omental and mesenteric) fat mass, the investigators have recently developed a novel sex-specific index based on measurement of WC, BMI, TG and HDL levels, and termed it VAI. It is a mathematical model and simple indicator of visceral adipose mass strongly associated with the severity of CAD [21]. In addition, VAI has been proposed as a useful tool for early detection of a condition of cardiometabolic risk before it develops into an overt MetS. In our study, we found that increased VAI increases the risk of coronary segment 3 significant stenosis.
### Table VI. Influence of MetS, its components and obesity indices on severity of coronary artery disease in patients with acute STEMI

| Parameters                                      | > 1 significantly stenosed CA | P-value | Significant proximal/ middle CSS | P-value | Significant stenosis of segment 1 | P-value | Significant stenosis of segment 3 | P-value |
|------------------------------------------------|-------------------------------|---------|----------------------------------|---------|----------------------------------|---------|----------------------------------|---------|
| MetS (NCEP-ATP III)                            | 1.72 (1.04–2.84)              | 0.034   | 2.45 (0.99–6.01)                 | 0.051   | 1.22 (0.71–2.07)                 | 0.472   | 1.15 (0.51–2.61)                 | 0.740   |
| MetS (IDF)                                      | 1.37 (0.83–2.27)              | 0.224   | 1.12 (0.47–2.66)                 | 0.797   | 1.16 (0.68–2.00)                 | 0.585   | 0.66 (0.29–1.50)                 | 0.325   |
| Hypertension                                   | 1.42 (0.81–2.47)              | 0.221   | 1.44 (0.58–3.57)                 | 0.428   | 0.91 (0.51–1.64)                 | 0.764   | 0.70 (0.29–1.64)                 | 0.408   |
| Dyslipidemia                                    | 1.39 (0.77–2.48)              | 0.271   | 1.15 (0.43–3.07)                 | 0.777   | 1.83 (0.94–3.58)                 | 0.076   | 2.57 (0.74–8.89)                 | 0.136   |
| Hyperglycemia                                   | 1.39 (0.77–2.49)              | 0.268   | 1.19 (0.42–3.34)                 | 0.747   | 0.79 (0.42–1.48)                 | 0.466   | 1.15 (0.46–2.89)                 | 0.761   |
| WC ≥ 94/80 cm (IDF)                            | 0.79 (0.40–1.55)              | 0.493   | 0.74 (0.21–2.62)                 | 0.643   | 0.84 (0.42–1.70)                 | 0.629   | 0.48 (0.19–1.25)                 | 0.135   |
| WC ≥ 102/88 cm (ATP)                           | 1.28 (0.77–2.12)              | 0.339   | 1.71 (0.72–4.05)                 | 0.222   | 1.19 (0.69–2.05)                 | 0.523   | 1.08 (0.47–2.49)                 | 0.852   |
| BMI < 25.0 kg/m²                                | 1.22 (0.68–2.19)              | 0.500   | 0.89 (0.33–2.37)                 | 0.815   | 1.21 (0.66–2.23)                 | 0.530   | 1.79 (0.75–4.25)                 | 0.190   |
| BMI 25.0–29.9 kg/m²                             | 0.97 (0.59–1.60)              | 0.916   | 0.96 (0.41–2.27)                 | 0.933   | 0.93 (0.55–1.58)                 | 0.788   | 0.96 (0.43–2.18)                 | 0.928   |
| BMI ≥ 30.0 kg/m²                                | 0.86 (0.50–1.50)              | 0.603   | 1.17 (0.44–3.09)                 | 0.754   | 0.92 (0.51–1.64)                 | 0.767   | 0.55 (0.21–1.53)                 | 0.255   |
| WHR > 0.90/0.85                                 | 1.06 (0.48–2.31)              | 0.893   | 4.27 (1.58–11.56)                | 0.004   | 3.34 (1.12–9.96)                 | 0.031   | 0.66 (0.22–2.09)                 | 0.483   |
| WHR ≤ 53/49                                     | 0.94 (0.49–1.83)              | 0.865   | 0.70 (0.25–2.02)                 | 0.514   | 0.87 (0.43–1.80)                 | 0.720   | 1.98 (0.77–5.06)                 | 0.154   |
| WHR ≥ 53/69–62/57                               | 0.82 (0.50–1.36)              | 0.444   | 0.77 (0.32–1.38)                 | 0.552   | 1.16 (0.69–1.96)                 | 0.591   | 0.40 (0.17–0.95)                 | 0.037   |
| WHR > 63/58                                     | 1.30 (0.76–2.20)              | 0.340   | 1.82 (0.65–5.10)                 | 0.252   | 0.92 (0.52–1.62)                 | 0.774   | 1.60 (0.70–3.67)                 | 0.264   |
| Cindex > 1.25/1.18                              | 1.30 (0.69–2.45)              | 0.409   | 0.87 (0.28–2.69)                 | 0.810   | 1.44 (0.71–2.90)                 | 0.308   | 1.00 (0.36–2.81)                 | 0.995   |
| BAI (normal)                                    | 1.56 (0.93–2.64)              | 0.096   | 0.85 (0.35–2.06)                 | 0.718   | 1.42 (0.81–2.49)                 | 0.218   | 1.93 (0.78–4.76)                 | 0.152   |
| BAI (high)                                      | 0.65 (0.37–1.11)              | 0.114   | 0.91 (0.37–2.23)                 | 0.834   | 0.86 (0.48–1.53)                 | 0.612   | 0.52 (0.20–1.38)                 | 0.191   |
| BAI (very high)                                 | 0.92 (0.39–2.17)              | 0.843   | 2.57 (0.33–20.03)                | 0.369   | 0.53 (0.19–1.49)                 | 0.230   | 0.82 (0.18–3.73)                 | 0.794   |
| Increased VAI                                   | 0.78 (0.46–1.31)              | 0.347   | 0.97 (0.38–2.49)                 | 0.953   | 0.92 (0.53–1.60)                 | 0.773   | 2.69 (1.01–7.16)                 | 0.047   |

ATP – NCEP-ATP III diagnostic criteria, BAI – body adiposity index (cut-off values of overweight for males/females are 21/33% (age 20–39 years), 23/35% (age 40–59 years) and 25/38% (age 60–79 years); cut-off values of central obesity are 26/39% (age 20–39 years), 29/41% (age 40–59 years) and 31/43% (age 60–79 years)), BMI – body mass index, CA – coronary artery, Cindex – conicity index, CSS – coronary segments stenosis, IDF – IDF diagnostic criteria, MetS – metabolic syndrome, STEMI – ST-elevation myocardial infarction, VAI – visceral adiposity index (cut-off points are 2.52 (age < 30 years), 2.23 (age 30–41 years), 1.92 (age 42–51 years), 1.93 (age 52–65 years) and 2.00 (age ≥ 66 years)), WC – waist circumference, WHR – waist-to-hip ratio, WHtR – waist-to-height ratio. *Statistical significance defined as p < 0.05. †Univariate logistic regression analysis – odds ratio (confidence interval).
Firstly, according to all the above facts, we can conclude that the measurement of VAI instead of WC is more reliable for predicting clinical severity of acute STEMI, simply because VAI reflects the amount of visceral adipose tissue which pathophysiologically has one of the major roles in the atherosclerotic process. Secondly, the optional presence of increased WC in MetS (NCEP-ATP III) and obligatory presence in MetS (IDF) may explain the role of MetS (NCEP-ATP III) in predicting clinical severity of acute STEMI.

The presence of increased WHR is associated with significant CAs stenosis, but not with the number of significantly stenosed CAs [38]. Patients with WHR \(\geq 0.90/0.85\) have higher rates of heart failure and mortality in acute STEMI; increased WHR is an independent predictor of 6-month mortality [39]. We found that WHR \(\geq 0.90/0.85\) is an independent predictor of severity of CAD (significant stenosis of proximal/middle CAs segments (especially of the coronary segment 1)), without an influence on other parameters of severity and prognosis of acute STEMI.

Studies have reported a paradoxical clinical effect of BMI on outcomes after PCI in patients with acute MI. The association between elevated BMI and improved survival has been termed the overall 'obesity paradox' [40, 41]. Furthermore, Babic et al. used the American Association of Clinical Endocrinologists/American College of Endocrinology (AACE/ACE) definition of MetS in patients with acute STEMI and found no statistically significant differences in severity or prognosis between groups of patients with and without MetS [42]. The authors concluded that, among other problems, anthropometry (use of BMI) was the most important reason for that. We also found no significant influence of BMI on clinical severity and prognosis of acute STEMI, which could be explained by the previously mentioned overall 'obesity paradox' [40, 41].

As we already mentioned, Cindex, BAI, VAI and WHtR were used for the first time in patients with acute STEMI and primary PCI. We found that Cindex \(>1.25/1.18\), very high BAI and WHtR \(\geq 63/58\) increased the risk of total in-hospital complications, dyspnea and heart failure, respectively. Finally, the number of significantly stenosed CAs increased the risk of total MACE, which is consistent with the literature data [43].

In conclusion, MetS (NCEP-ATP III) and several central obesity indices are superior to overall obesity (BMI) in predicting acute STEMI severity (clinical presentation, in-hospital complications and severity of CAD), while WC and MetS (IDF) have no influence on it. Finally, MetS (NCEP-ATP III, IDF) and obesity indices have no influence on prognosis (MACE and SLD).

### Conflict of interests

The authors declare no conflict of interest.

### References

1. Huang PL. A comprehensive definition for metabolic syndrome. Dis Model Mech 2009; 2: 231-7.
The importance of two metabolic syndrome diagnostic criteria and body fat distribution in predicting clinical severity and prognosis of acute myocardial infarction

2. Kassi E, Pervanidou P, Kalttis G, Chrousos G. Metabolic syndrome: definitions and controversies. BMC Med 2011; 9: 48.

3. Batsis JA, Nieto-Martinez RE, Lopez-Jimenez F. Metabolic syndrome: from global epidemiology to individualized medicine. Clin Pharmacol Ther 2007; 82: 509-24.

4. Grundy SM. Metabolic syndrome pandemic. Arterioscler Thromb Vasc Biol 2008; 28: 629-36.

5. Qiao Q, Tuomilehto J, Balkau B, Borch-Johnsen K, Heine R, Wareham NJ; DECODE Study Group. Are insulin resistance, impaired fasting glucose and impaired glucose tolerance all equally strongly related to age? Diabet Med 2005; 22: 1476-81.

6. Hu G, Qiao Q, Tuomilehto J, Balkau B, Borch-Johnsen K, Pyorälä K; DECODE Study Group. Prevalence of the metabolic syndrome and its relation to all-cause and cardiovascular mortality in nondiabetic European men and women. Arch Intern Med 2004; 164: 1066-76.

7. Hu G, Qiao Q, Tuomilehto J, Eliasson M, Feskens EJ, Pyörälä K; DECODE Insulin Study Group. Plasma insulin and cardiovascular mortality in non-diabetic European men and women: a meta-analysis of data from eleven prospective studies. Diabetologia 2004; 47: 1245-56.

8. Lee CD, Jacobs DR Jr, Schreiner PJ, Iribarren C, Hong WY; Alameda Project. Waist circumference and waist-to-height ratio as screening tools for cardiovascular disease and diabetes: 0·5 to 2·5 standard deviations from the mean. Diabetologia 2007; 50: 155-61.

9. Coutinho T, Goel K, Corrêa de Sá D, et al. Central obesity and survival in subjects with coronary artery disease: a systematic review of the literature and collaborative analysis with individual subject data. J Am Coll Cardiol 2011; 57: 1877-86.

10. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0-5 could be a suitable global boundary value. Nutr Res Rev 2010; 23: 247-69.

11. Zhu J, Su X, Li G, Chen J, Tang B, Yang Y. The incidence of acute myocardial infarction in relation to overweight and obesity: a meta-analysis. Arch Med Sci 2014; 10: 855-62.

12. Feinberg MS, Schwartz R, Behar S. Impact of metabolic syndrome in patients with acute coronary syndrome. Adv Cardiol 2008; 45: 114-26.

13. Lee MG, Jeong MH, Ahn Y, et al. Impact of the metabolic syndrome on the clinical outcome of patients with acute ST-elevation myocardial infarction. J Korean Med Sci 2010; 25: 1456-61.

14. Zeller M, Steg PG, Ravisij J, et al.; Observatoire des Infections de Côte-d’Or Survey Working Group. Prevalence and impact of metabolic syndrome on hospital outcomes in acute myocardial infarction. Arch Intern Med 2005; 165: 1192-8.

15. Van de Werf F, Bax J, Betriu A, et al.; ESC Committee for Practice Guidelines (CPG). Management of acute myocardial infarction in patients presenting with persistent ST-segment elevation: the Task Force on the Management of ST-Segment Elevation Acute Myocardial Infarction of the European Society of Cardiology. Eur Heart J 2008; 29: 2909-45.

16. Task Force on Myocardial Revascularization of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS); European Association for Percutaneous Cardiovascular Interventions (EAPCI), Wijns W, Kolh P, Danchin N, et al. Guidelines on myocardial revascularization. Eur Heart J 2010; 31: 2501-55.
32. Mornar Jelavic M, Babic Z, Pintaric H. Metabolic syndrome: influence on clinical severity and prognosis in patients with acute ST-elevation myocardial infarction treated with primary percutaneous coronary intervention. Acta Cardiol 2015; 70: 149-56.
33. Ekmekci A, Gungor B, Uluganyan M, et al. Presence of metabolic syndrome is not an independent predictor of in-hospital adverse events in patients with ST elevation myocardial infarction that underwent primary percutaneous coronary intervention. Endocrinol Metab Synd 2013; 2: 112.
34. Al Suwaidi J, Zubaid M, El-Menyar AA, et al. Prevalence of the metabolic syndrome in patients with acute coronary syndrome in six middle eastern countries. J Clin Hypertens (Greenwich) 2010; 12: 890-9.
35. Bechlioulis A, Vakalis K, Naka KK, et al. Paradoxical protective effect of central obesity in patients with suspected stable coronary artery disease. Obesity (Silver Spring) 2013; 21: E314-21.
36. Amato MC, Giordano C, Galia M, et al.; AlkaMeSy Study Group. Visceral Adiposity Index: a reliable indicator of visceral fat function associated with cardiometabolic risk. Diabetes Care 2010; 33: 920-2.
37. Martins A, Ribeiro S, Gonçalves R Correia A. Role of central obesity in risk stratification after an acute coronary event: does central obesity add prognostic value to the Global Registry of Acute Coronary Events (GRACE) risk score in patients with acute coronary syndrome? Rev Port Cardiol 2013; 32: 769-76.
38. Zen V, Fuchs FD, Wainstein MV, et al. Neck circumference and central obesity are independent predictors of coronary artery disease in patients undergoing coronary angiography. Am J Cardiovasc Dis 2012; 2: 323-30.
39. Lee SH, Park JS, Kim W, et al.; Korean Acute Myocardial Infarction Registry Investigators. Impact of body mass index and waist-to-hip ratio on clinical outcomes in patients with ST-segment elevation acute myocardial infarction (from the Korean Acute Myocardial Infarction Registry). Am J Cardiol 2008; 102: 957-65.
40. Kang WY, Jeong MH, Ahn YK, et al.; Korea Acute Myocardial Infarction Registry Investigators. Obesity paradox in Korean patients undergoing primary percutaneous coronary intervention in ST-segment elevation myocardial infarction. J Cardiol 2010; 55: 84-91.
41. Mehta L, Devlin W, McCullough PA, et al. Impact of body mass index on outcomes after percutaneous coronary intervention in patients with acute myocardial infarction. Am J Cardiol 2007; 99: 906-10.
42. Babic Z, Pavlov M, Bulj N, et al. Metabolic syndrome and outcome in patients with acute myocardial infarction. Acta Clin Croat 2011; 50: 193-9.
43. Sorajja P, Gersh BJ, Cox DA, et al. Impact of multivessel disease on reperfusion success and clinical outcomes in patients undergoing primary percutaneous coronary intervention for acute myocardial infarction. Eur Heart J 2007; 28: 1709-16.