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

Marko Mornar Jelavic[1], Zdravko Babic[2], Hrvoje Pintaric[3]

[1]Department of Internal Medicine and Dialysis, Zagreb – East Health Center, Zagreb, Croatia
[2]Coronary Care Unit, Sestre Milosrdnice University Hospital Center, Zagreb, Croatia
[3]Cardiac Catheterization Laboratory, Sestre Milosrdnice University Hospital Center, Zagreb, Croatia

Submitted: 5 January 2016
Accepted: 15 March 2016

Arch Med Sci 2017; 13, 4: 795–806
DOI: https://doi.org/10.5114/aoms.2016.59703

Corresponding author:
Marko Mornar Jelavic MD, PhD
Department of Internal Medicine and Dialysis
Zagreb – East Health Center
Ninska 10
HR-10000 Zagreb, Croatia
Phone: +385(91)7826135
E-mail: mjelavic@yahoo.com

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/m^2) and classified as BMI < 25.0 (normal weight), BMI 25.0–29.9 kg/m^2 (overweight) and BMI ≥ 30.0 kg/m^2 (overall 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. Cindex was calculated as follows: Cindex = WC/(0.109 × (weight/height)$^{1.5}$) [18]. 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) × (1.31/HDL) 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 severity of acute STEMI was 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. We analyzed the number of significantly narrowed CAs, number, length and diameter of used stents. Additionally, for the first time, we analyzed significantly stenosed segments of CAs. For that purpose, and according to the modified American Heart Association classification [23], CAs were divided into 16 segments. Segments were classified into two groups, as follows:

A) proximal and middle CAs segments: segment 1 (right coronary artery (RCA), proximal), segment 2 (RCA, mid), segment 5 (main stem), segment 6 (left anterior descending coronary artery (LAD), proximal), segment 7 (LAD, mid), segment 9 (first diagonal, D1), segment 11 (left circumflex artery (LCX), proximal), segment 12 (obtuse marginal – OM); and
B) distal CAs segments: segment 3 (RCA, distal), segment 4 (right posterior descending), segment 8 (LAD, distal), segment 10 (second diagonal, D2), segment 13 (LCX, distal), segment 14 (LCX, posterolateral branch), segment 15 (LCX, posterior descending branch), segment 16 (RCA, posterolateral branch).

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 posterolateral 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 χ^2 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 χ^2 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/m^2 (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 antiaggregation 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/m^2) 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/m^2 and BMI 25.0–29.9 kg/m^2 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.

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* (%)‡	103 (70.1)	80 (77.7)	183 (73.2)	0.234
	Women, *n* (%)‡	44 (29.9)	23 (22.3)	67 (26.8)	0.234
	Age [years]§	63 (25–92)	60 (39–91)	62 (25–92)	0.233
	Hypertension, *n* (%)‡	139 (94.6)	42 (40.8)	181 (72.4)	< 0.001
	Dyslipidemia, *n* (%)‡	133 (90.5)	57 (55.3)	190 (76.0)	< 0.001
	Hyperglycemia, *n* (%)‡	52 (35.4)	9 (8.7)	61 (24.4)	< 0.001
	Smoking, *n* (%)‡	76 (51.7)	53 (51.5)	129 (51.6)	0.928
	Family history (MI/stroke), *n* (%)‡	69 (46.9)	38 (36.9)	107 (42.8)	0.147
	Previous MI, *n* (%)‡	17 (11.6)	5 (4.9)	22 (8.8)	0.106
	Previous PCI, *n* (%)‡	17 (11.6)	6 (5.8)	23 (9.2)	0.186
	Previous CABG, *n* (%)‡	1 (0.7)	0 (0)	1 (0.4)	–
	WC ≥ 94/80 (IDF) cm, *n* (%)‡	147 (100)	62 (60.2)	209 (83.6)	< 0.001
	WC ≥ 102/88 (ATP) cm, *n* (%)‡	117 (79.6)	32 (31.1)	149 (59.6)	< 0.001
	MetS (ATP), *n* (%)‡	131 (89.1)	5 (4.9)	136 (54.4)	< 0.001
	BMI < 25.0 kg/m^2, *n* (%)‡	20 (13.6)	40 (38.8)	60 (24.0)	< 0.001
	BMI 25.0–29.9 kg/m^2, *n* (%)‡	64 (43.5)	54 (52.4)	118 (47.2)	0.209
	BMI ≥ 30.0 kg/m^2, *n* (%)‡	63 (42.9)	9 (8.7)	72 (28.8)	< 0.001
	WHR ≥ 0.90/0.85, *n* (%)‡	135 (91.8)	87 (84.5)	222 (88.8)	0.106
	WHtR < 53/49, *n* (%)‡	5 (3.4)	37 (35.9)	42 (16.8)	< 0.001
	WHtR 53/49–62/57, *n* (%)‡	78 (53.1)	49 (47.6)	127 (50.8)	0.468
	WHtR ≥ 63/58, *n* (%)‡	64 (43.5)	17 (16.5)	81 (32.4)	< 0.001
	Cindex > 1.25/1.18, *n* (%)‡	132 (89.8)	70 (68.0)	202 (80.8)	< 0.001
	BAI (normal), *n* (%)‡	60 (45.1)	54 (58.7)	114 (50.7)	0.062
	BAI (high), *n* (%)‡	54 (40.6)	34 (37.0)	88 (39.1)	0.680
	BAI (very high), *n* (%)‡	19 (14.3)	4 (4.3)	23 (10.2)	0.028
	Normal VAI, *n* (%)‡	56 (41.2)	54 (58.7)	110 (48.2)	0.014
	Increased VAI, *n* (%)‡	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), 1.92 (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. ‡Data are expressed as absolute number and percentage (%), compared with χ^2 test. §Data are expressed as median and range, compared with Mann-Whitney U test.

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].

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 (8.7)	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 χ^2 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.

Ekmekci *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

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 χ^2 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 χ^2 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/m^2	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/m^2	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/m^2	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
WHtR < 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
WHtR 53/49–62/57	0.85 (0.50–1.46)	0.562	0.58 (0.32–1.03)	0.061	0.78 (0.47–1.29)	0.326
WHtR ≥ 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/m^2	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/m^2	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/m^2	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
WHtR < 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
WHtR 53/49–62/57	0.82 (0.50–1.36)	0.444	0.77 (0.32–1.83)	0.552	1.16 (0.69–1.96)	0.591	0.40 (0.17–0.95)	0.037
WHtR ≥ 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).

Table VII. Influence of MetS, its components and obesity indices on prognosis (MACE) in patients with acute STEMI

Parameter	MACE	*P*-value†
MetS (NCEP-ATP III)	1.05 (0.55–2.00)†	0.881
MetS (IDF)	1.12 (0.58–2.16)	0.738
Hypertension	1.63 (0.76–3.51)	0.210
Dyslipidemia	0.78 (0.38–1.61)	0.500
Hyperglycemia	0.85 (0.40–1.86)	0.690
WC ≥ 94/80 cm (IDF)	1.29 (0.50–3.31)	0.601
WC ≥ 102/88 cm (ATP)	1.07 (0.55–2.07)	0.842
BMI < 25.0 kg/m^2	1.03 (0.48–2.19)	0.948
BMI 25.0–29.9 kg/m^2	0.78 (0.41–1.49)	0.457
BMI ≥ 30.0 kg/m^2	1.30 (0.66–2.58)	0.452
WHR ≥ 0.90/0.85	0.70 (0.27–1.77)	0.453
WHtR < 53/49	0.56 (0.21–1.53)	0.260
WHtR 53/49–62/57	1.56 (0.81–2.98)	0.183
WHtR ≥ 63/58	0.82 (0.41–1.66)	0.595
Cindex > 1.25/1.18	1.97 (0.78–4.97)	0.150
BAI (normal)	0.82 (0.42–1.63)	0.579
BAI (high)	1.00 (0.50–2.01)	0.993
BAI (very high)	1.56 (0.57–4.25)	0.382
Increased VAI	1.07 (0.55–2.06)	0.849
Number of significantly stenosed CAs	2.11 (1.40–3.17)	< 0.001
LVEF	0.96 (0.93–0.99)	0.045
Distal CA segment stenosis	2.18 (1.14–4.17)	0.019

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, CAs – coronary arteries, Cindex – conicity index, IDF – IDF diagnostic criteria, LVEF – left ventricular ejection fraction, MACE – major adverse cardiovascular events, 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 ≥ 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 ≥ 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 ≥ 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.

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