

## Original Article

# The clinical characteristics of metabolic syndrome in patients with acute myocardial infarction in the real world

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Received September 12, 2017; Accepted March 28, 2018; Epub June 15, 2018; Published June 30, 2018

**Abstract:** Objective: This study aimed to evaluate the clinical significance of metabolic syndrome (MetS) and each of its components in patients with acute myocardial infarction (AMI). Method: In this research, 1,311 eligible subjects selected from 2,712 AMI patients were divided into a group with MetS and a group without MetS. The age, sex and other basic clinical characteristics, myocardial infarction size estimated by the peak concentration of blood cardiac-specific enzymes, left ventricular functions gauged by echocardiography, and major adverse cardiovascular events (MACE) in the hospital were compared between the two groups, as well as among sub-groups of each MetS component in AMI patients with MetS. Result: The MetS prevalence rate was 47.22% (619/1311) in AMI populations. The AMI patients with MetS were younger than the patients without MetS, with increased platelet volume distribution width (PDW) and mean platelet volume (MPV), decreased total bilirubin, and a higher coronary lesion number; for the AMI patients with MetS, the sub-populations of increased fasting glucose, higher blood pressure upon hospital admission, and normal triglyceride (TG) and high-density lipoprotein cholesterol (HDL-c) patients had a higher peak concentration of cardiac-specific enzymes. Conclusion: The patients with MetS had an earlier AMI onset age, more severe platelet activation and thrombotic event risks, lower anti-oxidant ability, and a more severe degree of coronary lesion than the AMI patients without MetS; for the AMI patients with MetS, the subpopulations of increased fasting glucose, higher blood pressure upon admission, and normal TG and HDL-c tended to have a larger estimated myocardial infarction size and should be afforded more attention.

**Keywords:** Metabolic syndrome (MetS), acute myocardial infarction (AMI), estimated infarction size, major adverse cardiovascular events (MACE)

## Introduction

Metabolic syndrome (MetS) is defined as a clustering of interrelated atherosclerotic risk factors, including impaired blood glucose, high blood pressure, a low level of high-density lipoprotein cholesterol (HDL-c), a high triglyceride (TG) level, and abdominal obesity [1]. The concept of MetS provides an understanding of the underlying pathophysiology that all of the components combined have in common in the real world. It affected about 15.4% of adults in China [2] and more than 20% of the population in the Western world [3]. MetS is related to increased risks of cardiovascular and diabetes associated morbidity and mortality [4]. It is also a significant risk factor for acute myocardial infarction (AMI) [5]; individuals with MetS have a higher prevalence of ST-segment elevation myocardial infarction (STEMI) [6], though it can

be effectively managed by lifestyle therapies in the early stages. The clinical manifestation and consequences of MetS in the general population are well known, while the population of AMI patients is still not fully understood. This study aimed to investigate the clinical significance of MetS and each of its components in patients with AMI. The main contents of this study include an analysis of basic clinical characteristics, a comparison of myocardial infarct size as estimated by the peak concentration of cardiac-specific enzymes, including creatine kinase-MB fraction (CKMB), myoglobin (Myo), and troponin I (TnI) [4], major adverse cardiovascular events (MACE) that occurred at the hospital, including stroke, recurrent myocardial infarction, heart failure, and cardiovascular death, and the left ventricular functions gauged by echocardiography for AMI patients with and without MetS. This study also further explored

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differences in the above items between sub-groups of each MetS component in AMI patients with MetS. It was expected this study would provide some useful hints for current clinical practice and future in-depth research.

### Patients and methods

#### *Study population*

This study reviewed a consecutive series of 2,712 patients with AMI who were admitted to the cardiac care unit (CCU) at Beijing Friendship Hospital from April 2013 to April 2017. A total of 1,311 AMI patients were eligible for this study according to the inclusion and exclusion criteria on presenting with STEMI or non-ST-segment elevation myocardial infarction (NSTEMI). The STEMI patients underwent percutaneous coronary intervention as part of reperfusion therapy within 12 hours of the onset of symptoms. For NSTEMI patients, initial antithrombotic therapy was instituted and subsequent coronary angiography was performed within the first week. This study was approved by the local ethics committee and conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from all the study participants.

#### *Inclusion and exclusion criteria*

Patients were included if they met the universal definition of AMI [7] and had no documented history of other cardiovascular diseases (valvular heart diseases, left ventricular dysfunction, left ventricular hypertrophy, atrial fibrillation), respiratory diseases (pneumonia, chronic obstructive pulmonary disease, asthma, interstitial lung disease, pulmonary hypertension, pulmonary embolism), kidney diseases (glomerular nephritis, nephropathy syndrome, chronic renal failure, dialysis), infectious diseases (tuberculosis, hepatitis B, active infective endocarditis), endocrine diseases (hyperthyroidism and hypothyroidism), rheumatic disease (systemic lupus erythematosus, rheumatoid arthritis, vasculitis), hematological diseases (neutropenia, anemia, leukemia, lymphoma, disseminated intravascular coagulation), and varieties of neoplastic diseases.

#### *Diagnostic criteria for metabolic syndrome*

Eligible AMI patients were classified according to the Chinese Diabetes Society's (CDS) 2004

criteria for metabolic syndrome into two groups [8], AMI patients with MetS and without MetS. According to the criteria, MetS was diagnosed when at least three of the following risk factors were present: 1) A body mass index (BMI)  $\geq 25$  kg/m<sup>2</sup>, 2) Systolic blood pressure  $\geq 140$  mmHg or diastolic blood pressure  $\geq 90$  mmHg, or taking anti-hypertension drugs, 3) A documented history of diabetes or fasting glucose  $\geq 6.1$  mmol/L, and 4) TG  $\geq 1.7$  mmol/L, or HDL-c  $\leq 0.9$  mmol/L in men or HDL-c  $\leq 1.0$  mmol/L in women.

#### *The basic clinical characteristics data*

The hospital medical records were detailed and intact. Most of the data in this research was extracted from the medical records, including demographic data (age and sex), disease history (CHD, diabetes, and other diseases), the presence of smoking and drinking, family histories (hypertension, diabetes, and CHD), and medications taken before admission. Body mass index (BMI) was calculated by dividing weight in kilograms by height in meters squared (kg/m<sup>2</sup>).

#### *The echocardiography and coronary angiogram analysis*

The transthoracic echocardiography was performed at a median of five days after AMI. All images were analyzed by a single investigator, who was blind to all clinical data. The coronary angiograms were performed via a radial artery approach, and each coronary angiogram was interpreted by two independent cardiologists.

#### *Biochemical analysis*

The blood concentrations of cTnI, Myo, CKMB, and N-terminal pro-brain natriuretic peptide (NT-proBNP) were measured during admission and at 12-hour intervals during the first 5 days, following the presentation of acute myocardial infarction (from symptom onset). Serum peak concentrations of TnI, Myo, and CKMB levels were used for estimations of myocardial infarct size. After 12 hours of fasting, blood samples were taken from an antecubital vein to measure total cholesterol (TC), TG, HDL-c, LDL-c, and fasting plasma glucose levels.

#### *Statistical analysis*

This research was a case-control study conducted by reviewing the medical data from con-

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**Table 1.** Baseline characteristics of patients with metabolic Syndrome

	Non-MetS n=692	MetS n=619	P value
Age, years	62 (55-72)	60 (53-71)	0.007
Sex, male	509 (73.55%)	476 (76.90%)	0.179
STEMI, n	386 (55.78%)	316 (51.05%)	0.096
Family history of CHD, n	201 (29.05%)	191 (30.86)	0.506
Family history of diabetes, n	67 (9.68%)	89 (14.38%)	0.010
Family history of hypertension, n	159 (22.98%)	189 (30.53%)	0.002
Smoking, n	417 (60.26%)	398 (64.30%)	0.138
Alcohol, n	240 (34.68%)	234 (37.80%)	0.250
History of CHD, n	192 (27.75%)	211 (34.09%)	0.014
Previous AMI, n	57 (8.24%)	45 (7.27%)	0.537
Previous PCI, n	44 (6.36%)	56 (9.05%)	0.076
Previous cerebral infarction, n	28 (4.05%)	42 (6.79%)	0.036
Hypertension, n	316 (45.66%)	498 (80.45%)	0.000
SBP, mmHg	124 (110-137)	134 (119-148)	0.000
DBP, mmHg	71 (64-80)	76 (68-85)	0.000
Diabetes, n	113 (16.33%)	280 (45.23%)	0.000
FPG, mmol/L	5.23 (4.69-5.91)	6.65 (5.54-8.52)	0.000
HbA1c, %	5.70 (5.40-6.10)	6.40 (5.70-7.90)	0.000
TC, mmol/L	4.39 (3.78-5.00)	4.55 (3.86-5.17)	0.024
LDL-c, mmol/L	2.51 (2.05-3.01)	2.62 (2.18-3.08)	0.012
HDL-c, mmol/L	1.10 (0.96-1.26)	0.95 (0.84-1.10)	0.000
TG, mmol/L	1.26 (0.93-1.63)	1.88 (1.29-2.56)	0.000
BMI, kg/m <sup>2</sup>	24.03 (22.03-25.95)	27.04 (25.54-29.07)	0.000
Leucocyte, 10 <sup>9</sup> /L	7.70 (6.38-9.69)	8.10 (6.50-10.10)	0.048
Neutrophil count, 10 <sup>9</sup> /L	5.41 (4.08-7.19)	5.59 (4.22-7.36)	0.128
Lymphocyte count, 10 <sup>9</sup> /L	1.67 (1.28-2.13)	1.72 (1.34-2.15)	0.085
Monocyte count, 10 <sup>9</sup> /L	0.28 (0.18-0.41)	0.30 (0.19-0.42)	0.113
N/L ratio	3.23 (2.18-5.00)	3.17 (2.37-4.71)	0.973
RDW, %	12.70 (11.90-13.50)	12.80 (12.00-13.60)	0.173
Platelet count, 10 <sup>12</sup> /L	218 (182-259)	219 (180-259)	0.641
PDW, %	13.50 (12.00-15.30)	14.00 (12.20-15.80)	0.011
MPV, fL	8.10 (7.40-8.94)	8.30 (7.60-9.10)	0.004
hs-CRP, mg/L	5.38 (1.99-13.43)	6.48 (2.54-14.86)	0.019
ESR, mm/hour	12.00 (7.00-21.00)	13.00 (7.00-24.00)	0.144
ALT, U/L	24.00 (15.00-38.00)	25.00 (17.00-40.50)	0.016
AST, U/L	39.00 (22.00-104.50)	39.00 (21.30-87.50)	0.440
Total bilirubin, umol/L	2.00 (1.21-3.58)	1.67 (1.00-3.12)	0.001
Urea nitrogen, mmol/L	5.00 (3.98-6.44)	5.22 (4.14-6.38)	0.192
Serum creatinine, umol/L	80.80 (72.45-90.60)	82.60 (72.80-93.05)	0.081

P values for comparisons between the two groups. Significance level was 0.05. MetS, metabolic syndrome; STEMI, acute ST segment elevation myocardial infarction; CHD, coronary heart disease; AMI, acute myocardial infarction; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; HbA1c, hemoglobin A1c; TC, total cholesterol; TG, triglyceride; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; BMI, body mass index; N/L ratio, neutrophil to lymphocyte ratio; RDW, red cell volume distribution width; PDW, platelet volume distribution width; MPV, mean platelet volume; hs-CRP, high-sensitivity C reactive protein; ESR, erythrocyte sedimentation rate; ALT, glutamic pyruvic transaminase; AST, glutamic-oxaloacetic transaminase.

secutive series AMI patients. All analyses were conducted with Statistical Package for Social

Sciences (SPSS) software version 26. All data were initially analyzed using the Kolmogorov-

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**Table 2.** The estimated infarction size, MACE and left ventricular function of metabolic syndrome in the real world

	Non-MetS n=692	Met-S n=619	P value
Number of stenotic vessels	3.00 (2.00-4.00)	3.00 (2.00-5.00)	0.000
LM, n	64 (9.25%)	55 (8.89%)	0.848
LAD, n	470 (67.92%)	445 (71.89%)	0.132
DB, n	291 (42.05%)	288 (46.53%)	0.106
LCX, n	360 (52.02%)	375 (60.58%)	0.002
OR, n	222 (32.08%)	223 (36.03%)	0.144
RCA, n	393 (56.79%)	385 (62.20%)	0.049
PD, n	141 (20.38%)	167 (26.98)	0.005
PB, n	114 (16.47%)	144 (23.26%)	0.002
Peak value of NT-proBNP, ng/L	1151 (429-2984)	941 (366-2522)	0.039
Peak value of CKMB, ng/ml	52.10 (10.60-15600)	48.00 (12.00-148.00)	0.886
Peak value of Myo, U/L	63.70 (30.00-189.00)	56.50 (27.80-183.00)	0.464
Peak value of Tnl, ng/ml	5.00 (1.30-15.62)	4.51 (1.44-14.15)	0.992
Peak value of NT-proBNP, ng/L	1151 (429-2984)	941 (366-2522)	0.039
Peak value of CKMB, ng/ml	52.10 (10.60-15600)	48.00 (12.00-148.00)	0.886
LVEF, %	62 (54-67)	62 (55-66)	0.713
E/A	0.87 (0.72-1.21)	0.86 (0.70-1.18)	0.108
MACE, n	22 (3.18%)	17 (2.75%)	0.750
Cardiac death, n	12 (1.73%)	4 (0.65%)	0.080
Target vascular reconstruction, n	0 (0.00%)	0 (0.00%)	
Acute stent thrombosis, n	0 (0.00%)	0 (0.00%)	
Malignant arrhythmia, n	11 (1.59%)	14 (2.26%)	0.420
Recurrent myocardial infarction, n	0 (0.00%)	0 (0.00%)	
Cerebral infarction, n	3 (0.43%)	2 (0.32%)	1.000
Cerebral hemorrhage, n	4 (0.58)	3 (0.48%)	1.000

P values for comparisons between the two groups. Significance level was 0.05. MetS, metabolic syndrome; NT-proBNP, N-terminal pro-brain natriuretic peptide; CKMB, creatine kinase-MB fraction; Myo, myoglobin; Tnl, troponin I; LM, left main coronary artery; LAD, left anterior descending coronary artery; DB, diagonals branch; LCX, left circumflex coronary artery; OR, obtuse round branch; RCA, right coronary artery; PD, posterior descending branch; PB, posterior branch of left ventricle. LVEF, left ventricular ejection fraction; E/A, ratio of early to late ventricular filling velocities; MACE, major adverse cardiovascular events.

Smirnov test to assess for normality. Continuous data were presented as mean  $\pm$  SD while normally distributed and median with interquartile range (IQR) when non-Gaussian in distribution. Unpaired t-tests and Mann-Whitney-U rank sum tests were used for bivariate analyses of normally and non-normally distributed continuous data, respectively. Non-parametric data characteristics were assessed as percentages (%) and compared between groups using the chi-square test or Fisher's exact tests when appropriate.

### Results

#### *Baseline characteristics of patients with metabolic syndrome*

In this study, 1,311 medical documents selected from 2,712 AMI patients were analyzed. The 6165

MetS prevalence rate for the 1,311 patients was 47.22% (619/1311), while it was 49.75% (303/609) for the NSTEMI subgroup and 45.01% (316/702) for the STEMI subgroup. But if not for excluding the ineligible patients, the above items would be 48.63% (1,319/2,712), 23.76% (339/1,427) and 76.26% (980/1,285), respectively, for the 2,712 patients.

As shown in **Table 1**, the median AMI onset age for MetS patients in the hospital was lower than for non-MetS patients, and their rates of family CHD and diabetes histories were higher than for non-MetS patients. The MetS diagnostic criteria-associated items, including blood pressure, diabetes rate, TC, LDL-c, and BMI, were higher in MetS patients, and those results supported our MetS classification in this study. Other basic characteristics, including sex,

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**Table 3.** The treatment situation of metabolic syndrome before hospital admission in the real world

	Non-MetS n=692	Met-S n=619	P value
Antiplatelet agents, n	160 (23.12%)	148 (23.91%)	0.393
Nitrate ester, n	117 (16.91%)	115 (18.58%)	0.236
Proprietary Chinese Medicine, n	132 (19.08%)	130 (21.00%)	0.211
Antihypertensive treatment, n	243 (35.2%)	392 (63.33%)	0.000
ACEI/ARB, n	108 (15.61%)	184 (29.73%)	0.000
Beta receptor blocker, n	49 (7.08%)	68 (10.99%)	0.009
CCB, n	123 (17.77%)	230 (37.16%)	0.000
Diuretic agents, n	14 (2.02%)	15 (2.42%)	0.380
Other antihypertension treatments, n	24 (3.47%)	36 (5.82%)	0.029
Blood sugar lowering treatment, n	91 (13.15%)	228 (36.83%)	0.000
Biguanides, n	11 (1.59%)	61 (9.85%)	0.000
Alpha glucosidase inhibitor, n	47 (6.79%)	116 (18.74%)	0.000
Insulin sensitizer, n	11 (1.59%)	15 (2.42%)	0.171
Insulin, n	22 (3.18%)	36 (5.82%)	0.014
Statin, n	59 (8.53%)	82 (13.25%)	0.004

P values for comparisons between the two groups. Significance level was 0.05. MetS, metabolic syndrome; ACEI/ARB, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers; CCB, calcium channel antagonists.

smoking, alcohol usage and so on, showed no significant differences between the two groups.

The leucocyte count, red cell volume distribution width (RDW), platelet volume distribution width (PDW), mean platelet volume (MPV), high-sensitivity C-reactive protein (hs-CRP), and total bilirubin were higher in the MetS group.

From **Table 2**, it can be found that the coronary lesions were more severe in the MetS group, but the estimated myocardial infarction size, MACE, and the left ventricular functions in the MetS group did not differ from those of non-MetS AMI patients.

The MetS-associated treatments included anti-hypertensive treatment, blood sugar lowering treatment, and statins. From **Table 3**, it can be found that the rates of MetS-associated treatments were higher in the MetS group before admission to the hospital.

### *The impacts of each components of metabolic syndrome on AMI*

The blood sugar abnormal patients could be divided into three subgroups: the group with

increased fasting glucose, the previously diagnosed diabetes group, and the group with both increased fasting glucose and diagnosed diabetes. **Table 4** showed that the STEMI rate and estimated infarction size in the group with increased fasting glucose were the highest among the four sub-groups. The MACE and left ventricular functions showed no obvious differences.

The patients with abnormal blood pressure could be divided into three subgroups: the group with higher blood pressure upon admission, the group with previously diagnosed hypertension, and the group with both higher blood

pressure upon admission and previously diagnosed hypertension. **Table 5** showed that estimated infarction size in the group with higher blood pressure upon hospital admission was the highest among the four groups. The MACE and left ventricular functions showed no obvious differences.

The patients with abnormal blood lipids could be divided into three subgroups, including the abnormal LDL-c group, the abnormal TG group, and the group with both abnormal LDL-c and abnormal TG. As shown in **Table 6**, the estimated infarction size in the normal HDL-c and TG group was the highest among the four groups. MACE showed no obvious differences among the four groups.

The MetS patients could be divided into two groups by BMI, according to the CDS 2004 criteria for MetS. There were no significant differences between the sub-group of BMIs below 25 kg/m and the sub-group of BMIs above or equal to 25 kg/m<sup>2</sup> (**Table 7**).

### **Discussion**

In this study, 1,311 eligible AMI subjects selected from 2,712 AMI patients without serious interference factors were evaluated. The CDS 2004 criteria for MetS was employed [2]. The

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**Table 4.** The comparison of clinical outcomes among subgroups of blood sugar abnormal patients

	Normal blood sugar n=153	Fasting glucose increased n=186	Diabetes n=71	Both diabetes and fasting glucose increased n=209	P value
Age, years	58 (51-66)	59 (53-67)	67 (57-75)	63 (54-72)	0.000
Sex, male	130 (84.97%)	152 (81.72%)	53 (74.65%)	141 (67.46%)	0.000
History of CHD, n	50 (32.68%)	47 (25.27%)	32 (45.07%)	82 (39.23%)	0.005
STEMI, n	73 (47.71%)	110 (59.14%)	22 (30.99)	111 (53.11%)	0.001
Peak value of NT-proBNP, ng/L	845 (287-1891)	865 (360-2505)	1006 (333-3613)	1180 (432-2838)	0.035
Peak value of CKMB, ng/ml	42.5 (11.05-155.50)	80.30 (23.05-220.50)	33.40 (10.30-65.20)	32.25 (8.92-121.50)	0.000
Peak value of Myo, U/L	50.35 (23.95-145.50)	98.05 (39.30-315.50)	43.30 (25.50-126.00)	51.55 (27.18-182.50)	0.000
Peak value of Tnl, ng/ml	4.15 (1.37-13.95)	7.20 (2.46-25.00)	2.20 (1.24-8.18)	4.48 (0.95-14.10)	0.002
Number of stenotic vessels	3.00 (2.00-4.00)	3.00 (2.00-4.00)	4.00 (2.00-5.00)	4.00 (2.00-5.00)	0.232
MACE, n	2 (1.31%)	0 (0.00%)	0 (1.61%)	9 (4.31%)	0.206
LVEF, %	63 (56-67)	61 (53-66)	62 (57-66)	61 (57-66)	0.087
E/A	0.88 (0.71-1.21)	0.87 (0.69-1.15)	0.82 (0.70-1.25)	0.85 (0.69-1.16)	0.819

P values for comparisons between the two groups. Significance level was 0.05. CHD, coronary heart disease; STEMI, acute ST segment elevation myocardial infarction; MACE, major adverse cardiovascular events; NT-proBNP, N-terminal pro-brain natriuretic peptide; CKMB, creatine kinase-MB fraction; Myo, myoglobin; Tnl, troponin I; LVEF, left ventricular ejection fraction; E/A, ratio of early to late ventricular filling velocities.

**Table 5.** The comparison of clinical outcomes among subgroups of blood pressure abnormal patients

	Normal pressure n=72	High blood pressure in hospital n=49	Previous hypertension n=283	Both previous hypertension and high blood pressure in hospital n=215	P value
Age, years	56 (53-64)	59 (51-67)	61 (54-71)	62 (53-72)	0.023
Sex, male	67 (93.06%)	42 (85.71%)	215 (75.97%)	152 (70.70%)	0.001
History of CHD, n	17 (23.61%)	9 (18.37%)	107 (37.81%)	78 (36.28%)	0.011
STEMI, n	41 (56.94%)	28 (57.14%)	149 (52.65%)	98 (45.58%)	0.204
Peak value of NT-proBNP, ng/L	566 (308-1662)	1126 (360-2470)	991 (415-2648)	950 (355-2625)	0.137
Peak value of CKMB, ng/ml	51.70 (15.45-196.50)	74.6 (12.98-193.25)	60.1 (13.40-162.00)	34.9 (9.72-88.70)	0.042
Peak value of Myo, U/L	59.9 (27.30-237.50)	72.50 (36.63-331.75)	55.90 (28.60-165.00)	49.80 (26.90-170.50)	0.255
Peak value of Tnl, ng/ml	5.16 (1.90-15.00)	6.83 (1.89-22.10)	5.73 (1.67-19.80)	3.49 (1.00-9.00)	0.015
Number of stenotic vessels	3.00 (2.00-4.00)	4.00 (3.00-4.50)	3.00 (2.00-5.00)	4.00 (2.00-5.00)	0.221
MACE, n	0 (0.00%)	0 (0.00%)	13 (4.59%)	4 (1.86%)	0.053
LVEF, %	62 (57-66)	61 (58-67)	61 (54-66)	62 (56-66)	0.855
E/A	0.93 (0.75-1.25)	0.84 (0.72-1.30)	0.87 (0.70-1.17)	0.81 (0.68-1.18)	0.263

P values for comparisons between the two groups. Significance level was 0.05. CHD, coronary heart disease; STEMI, acute ST segment elevation myocardial infarction; MACE, major adverse cardiovascular events; NT-proBNP, N-terminal pro-Brain Natriuretic Peptide; CKMB, creatine kinase-MB fraction; Myo, myoglobin; Tnl, troponin I; LVEF, left ventricular ejection fraction; E/A, ratio of early to late ventricular filling velocities.

main findings were that: 1) For AMI patients in the hospital, the MetS subgroup was younger at the age of AMI onset, higher in family history rates of CHD and diabetes, and higher in previous prevalence history of CHD and cerebral diseases; 2) The platelet function was up-regulated in the MetS subgroup, as reflected by the higher PDW and MPV, and the total bilirubin, an anti-oxidant component [9], was lower in the MetS group; 3) The coronary lesion degree was higher in the MetS group, but the estimated infarction size, MACE, and left ventricular functions during hospitalization showed no obvious differences; and 4) The four components of MetS were divided further to explore their

impacts on the clinical outcomes of AMI. The results showed that the subgroup of increased fasting glucose, the subgroup of higher blood pressure upon admission, and the subgroup of normal TG and HDL-c tended to have larger estimated infarction sizes after AMI.

Regarding the basic characteristics, it makes sense that AMI patients with MetS were younger at AMI onset, higher in family history rates of CHD and diabetes, and more severe in coronary lesion because all four components of MetS comprised the risk factors for CHD and AMI [10]. In this study, the PDW and MPV, which were the platelet activation markers [11], were

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**Table 6.** The comparison of clinical outcomes among subgroups of blood lipoprotein abnormal patients

	Normal HDL-c and TG n=83	Abnormal HDL-c n=175	Abnormal TG n=171	Both abnormal HDL-c and TG n=190	P value
Age, years	66 (56-75)	66 (57-73)	60 (54-67)	56 (50-63)	0.000
Sex, male	46 (55.42%)	156 (89.14%)	98 (57.31%)	176 (92.63%)	0.000
History of CHD, n	28 (33.73%)	62 (35.43%)	50 (29.24%)	71 (37.37%)	0.416
STEMI, n	53 (63.86%)	87 (49.71%)	85 (49.70%)	91 (47.89%)	0.092
Peak value of NT-proBNP, ng/L	1862 (668-4899)	1251 (519-3040)	933 (355-2708)	551 (261-1282)	0.000
Peak value of CKMB, ng/ml	79.80 (24.33-200.75)	45.35 (11.78-155.50)	46.10 (12.53-126.00)	36.10 (10.00-116.00)	0.034
Peak value of Myo, U/L	92.00 (37.20-288.50)	59.60 (32.25-197.50)	52.00 (26.93-221.75)	50.10 (4.70-146.00)	0.007
Peak value of Tnl, ng.ml	7.75 (2.06-21.93)	6.16 (1.76-15.95)	4.52 (1.19-13.35)	3.94 (1.04-11.30)	0.101
Number of stenotic vessels	3.00 (3.00-4.00)	3.00 (2.00-5.00)	3.00 (2.00-4.00)	3.00 (2.00-5.00)	0.765
MACE, n	4 (4.82%)	2 (2.29%)	7 (4.09%)	2 (1.05%)	0.197
LVEF, %	66 (54-64)	60 (51-65)	63 (58-67)	63 (58-67)	0.000
E/A	0.79 (0.64-1.06)	0.83 (0.70-1.13)	0.80 (0.69-1.14)	1.00 (0.75-1.25)	0.000

P values for comparisons between the two groups. Significance level was 0.05. TG, triglyceride; HDL-c, high-density lipoprotein cholesterol; CHD, coronary heart disease; STEMI, acute ST segment elevation myocardial infarction; MACE, major adverse cardiovascular events; NT-proBNP, N-terminal pro-brain natriuretic peptide; CKMB, creatine kinase-MB fraction; Myo, myoglobin; Tnl, troponin I; LVEF, left ventricular ejection fraction; E/A, ratio of early to late ventricular filling velocities.

**Table 7.** The comparison of clinical outcomes between BMI below and above or equal to 25

	BMI under 25 n=104	BMI equal and above 25 n=515	P value
Age, years	64 (56-76)	60 (53-69)	0.003
Sex, male	72 (69.23%)	404 (78.45%)	0.055
History of CHD, n	38 (36.54%)	173 (33.59%)	0.572
STEMI, n	52 (50.00%)	264 (51.26%)	0.830
Peak value of NT-proBNP, ng/L	1239 (458-4435)	909 (345-2227)	0.003
Peak value of CKMB, ng/ml	41.35 (12.85-116.50)	49.00 (11.95-161.00)	0.570
Peak value of Myo, U/L	60.65 (26.55-204.75)	55.90 (28.50-171.00)	0.528
Peak value of Tnl, ng.ml	6.02 (1.73-16.20)	4.28 (1.43-13.80)	0.394
Number of stenotic vessels	4.00 (2.00-5.00)	3.00 (2.00-5.00)	0.436
MACE, n	2 (1.92%)	15 (2.91%)	0.751
LVEF, %	62 (57-67)	62 (55-66)	0.251
E/A	0.87 (0.71-1.14)	0.86 (0.69-1.18)	0.993

P values for comparisons between the two groups. Significance level was 0.05. BMI, body mass index; CHD, coronary heart disease; STEMI, acute ST segment elevation myocardial infarction; MACE, major adverse cardiovascular events; NT-proBNP, N-terminal pro-brain natriuretic peptide; CKMB, creatine kinase-MB fraction; Myo, myoglobin; Tnl, troponin I; LVEF, left ventricular ejection fraction; E/A, ratio of early to late ventricular filling velocities.

increased in the MetS group, suggesting that the MetS population was in a more severe thrombotic event condition. Total bilirubin was decreased in the MetS patients, with lower total bilirubin indicating decreased anti-oxidant ability [12, 13], and decreased serum levels of total bilirubin may be an important factor in coronary lipid plaque formation, while serum total bilirubin levels were found to be inversely associated with vulnerability to coronary plaque [14]. Thus, theoretically, the clinical outcome would be worse for MetS populations, but in this study, the estimated infarction size, MACE,

and left ventricular function in the MetS group did not differ from those of the no-MetS group. It was speculated that MetS-associated treatments, including anti-hypertension, lowering glucose, and regulating blood lipids, might improve the clinical outcome of MetS populations to some degree, and also that the observational period during the in-hospital stay was not adequate to draw a clear conclusion.

Comparisons among sub-groups of each component of MetS in AMI patients with MetS were further applied in this study. It was found that

the subgroup of the increased fasting glucose group, the subgroup of higher blood pressure upon admission, as well as the subgroup of normal TG and HDL-c, tended to have larger estimated myocardial infarction sizes. Myocardial infarct size is an important surrogate end point for early and late mortality after AMI [15]. In theory, those patients would have more serious outcomes with MACE and worse ventricular functions, but actually, due to the short time span of the observational period in this study, the differences in MACE and ventricular functions were not remarkable for those in-hospital patients. Thus, when MetS patients with AMI are at admissions, those three subgroups should be afforded more attention. Why did those three subgroup populations have a larger estimated myocardial infarction size? It could be deduced that: 1) The increased fasting glucose group and the higher blood pressure upon admission group had a recent onset of abnormal blood sugar and blood pressure, thus they would not have received appropriate treatment before admission; 2) The recent onset of abnormal blood sugar and lipoprotein would be related to the stress and inflammation status of the inner environment hemostasis [16, 17], as in this study, where the hs-CRP and leucocyte counts were higher in the MetS group, along with the decreased total bilirubin level; and 3) Due to the definition of MetS, if the patients had at least three risk factors, including abnormal blood sugar, abnormal blood lipids, abnormal blood pressure, and abnormal BMI, the normal lipoprotein subgroup would have had abnormal blood sugar. Further research is needed to explore the underlying mechanism for those clinical phenomena.

As for the diagnostic criteria for MetS, the five widely accepted versions are: the World Health Organization (WHO) 2004 version [18, 19], the National Cholesterol Education Program Adult Treatment, Panel III (NCEP-ATPIII) version [20], the revised NCEP (NCEP-R) version, the International Diabetes Federation (IDF) version [21], and the American Association of Clinical Endocrinologist (AACE) version [22]. The cutoff value of BMI in the WHO version was 30 kg/m<sup>2</sup>, while the waist cutoff values in NCEP-ATPIII and NCEP-R were 102 cm (male) and 88 cm (female), respectively, which were not suitable for the Chinese population, as MetS would be underestimated by the former two cutoff values

in practice [23]. In addition, Chinese people do not measure their waist value regularly during inpatient or outpatient therapies, while the BMI value was the relatively intact data used for evaluating obesity in China. As for the IDF and AACE versions, the hypertension cutoff value was 130/80 mmHg, while the Chinese guidelines recommend a hypertension cutoff value of 140/90 mmHg [24]. Many Chinese people had no habit of undergoing regular health examinations in their routine lives, thus leading to the omission of a considerable number of patients without health records. Therefore, for pragmatic reasons concerning the reality of Chinese patients and Chinese medical practice, the Chinese Diabetes Society's (CDS) 2004 criteria for MetS was employed.

### Limitations

Some limitations of our study deserve mentioning. First, the diagnostic criterion for MetS was based on the Chinese Diabetes Society's (CDS) 2004 criteria. The international criteria were not employed in this study because of discrepancies in Chinese patients and Chinese medical practices, which would not be a good conventional fit for international criteria. Second, follow-up observations were not employed in this study, and differences in MACE and left ventricular function between AMI patients with MetS and without MetS could not be fully observed in such a short time span of in-hospital treatment. Third, the assessments of blood pressure, glucose metabolic state, and lipids were applied during the peri-myocardial infarct period, meaning those results were acquired under conditions of inflammation and stress, and might not reflect the real conditions of blood sugar metabolism and blood pressure regulation of the body in terms of physiological status.

### Conclusion

The MetS patients were younger at AMI onset age, with higher rates of CHD and diabetes in their family history, and previous prevalence history of CHD and cerebral diseases; they had more severe platelet activation and thrombotic events risk, lower anti-oxidant ability, and a higher coronary lesion degree and lesion vessel number. For AMI patients with MetS, the subgroup of increased fasting glucose, the subgroup of higher blood pressure upon admis-

sion, and the subgroup of normal TG and HDL-c tended to have larger myocardial infarction sizes and should be afforded more attention.

### Acknowledgements

This study was supported by the National Natural Science Foundation of China (Grant No. 81600276), and we are also thankful for the support from Miss Guoliang Zhao and the Cardiovascular Center Beijing Friendship Hospital Database Bank (CBD bank) work group at Beijing Friendship Hospital.

### Disclosure of conflict of interest

None.

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