Impact of the metabolic syndrome on high-sensitivity C reactive protein levels in patients with acute coronary syndrome

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A B S T R A C T

Objective: Underlying predisposition for a heightened inflammatory response is postulated as one of the mechanisms for elevated high-sensitivity C reactive protein (hs-CRP) levels in patients with acute coronary syndrome (ACS). It is unclear whether metabolic syndrome (MetS) may cause a predisposition for heightened hs-CRP response in patients with ACS. The aim of this study is to investigate the interaction between hs-CRP levels and presence of MetS in patients with and without ACS.

Methods: Two hundred and seventy-three consecutive patients presenting with a first ACS event and 261 MetS patients without any ACS event were included in the study. The study participants were divided into three groups as MetS (+) ACS (−) [n = 261], MetS (−) ACS (+) [n = 110], and MetS (+) ACS (+) [n = 163]. Median levels of hs-CRP were compared between and within the three groups.

Factors associated with hs-CRP levels were troponin elevation, presence of ACS, body mass index (BMI), and presence of MetS (R² = 0.26, p < 0.01). Predictors of elevated hs-CRP levels (>0.3 mg/dl) were the presence of ACS (OR = 3.6, 95% CI = 1.9–6.5, p < 0.01), presence of MetS (OR = 2.1, 95% CI = 1.0–4.0, p = 0.02), troponin elevation (OR = 5.7, 95% CI = 2.8–11.5, p < 0.01) and BMI (OR = 1.1, 95% CI = 1.0–1.1, p < 0.01).

Conclusions: The presence of MetS had an impact on the increase in hs-CRP levels observed with an ACS event in the study population. These findings suggested that a heightened baseline inflammatory status of MetS may predispose ACS patients to an augmented hs-CRP response.

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1. Introduction

The recognition of inflammation as a central phenomenon in athero-thrombosis has forced clinicians to evaluate the relationship between circulating inflammatory biomarkers, cardiovascular risk, and the progression of atherosclerosis [1–4]. High-sensitivity C reactive protein (hs-CRP) has emerged as a robust inflammatory marker and a reliable predictor of future cardiovascular events in healthy individuals, patients with metabolic syndrome (MetS) and in those with acute coronary syndromes (ACS) [2,5–7].

A clear relationship between hs-CRP and future adverse events was described in patients with ACS; however, the source and mechanisms of generation of elevated hs-CRP levels in ACS patients remain elusive [2,6–9]. Myocardial necrosis following AMI, reperfusion therapy, extent of the atherosclerosis, inflammatory burden of coronary vascular bed, plaque rupture, thrombosis phenomena and concomitant coronary risk factors with ACS were potentially accused mechanisms [2,10,11]. It has also been concluded that the magnitude of hs-CRP response may vary according to both clinical condition and from person to person but the highest CRP values during ACS are likely to be observed in patients with already-elevated CRP values at baseline. Emerging work also shows the association of such genetic variants in CRP not only to CRP levels, but also to variation of CRP levels in the acute phase response [2,8,9].

In this regard, whether some ACS patients are predisposed to develop an enhanced inflammatory response and whether this ability is acquired or genetic remain matters of constant debate [8,9]. A number of studies in patients with ACS showed a strong positive correlation between the baseline levels of hs-CRP and its augmentation by various inflammatory stimuli. These results demonstrated that patients with elevated baseline hs-CRP values have an enhanced augmentation of this biomarker in response to different inflammatory factors [11–13].
Given that patients with MetS have usually higher baseline hs-CRP levels [14,15], we hypothesized that these patients are likely to have an augmented hs-CRP response (i.e., inflammatory hyperresponsiveness) to inflammatory stimuli such as plaque disruption or myocardial necrosis. Thus, we aimed to investigate the impact of the presence or absence of MetS on hs-CRP levels in patients with and without first ACS event. We corroborated our findings using various definitions of MetS and in the subgroups of patients with and without troponin elevation and among the various ACS subgroups such as unstable angina, non-ST segment elevation ACS and ST segment elevation ACS.

2. Materials and methods

2.1. Study patients

The study group consisted of 534 patients with first ACS event (n = 273) and without any ACS event (n = 261). The ACS group consisted of consecutive patients presenting to our tertiary center within 6 h after the onset of chest pain and admitted to our coronary care unit from November 2005 to December 2007. The aforementioned diagnoses were made according to the Joint European Society of Cardiology/American College of Cardiology (ESC/ACC) criteria and the 2004 American College of Cardiology/American Heart Association (ACC/AHA) guidelines [16,17]. Patients without ACS were recruited from subjects whom were already diagnosed as MetS by National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria and presenting in the same time period to our center for an annual or 6 months clinical and laboratory evaluation (n = 261) [18]. Exclusion criteria were as follows: subjects who experienced ACS with symptom onset of ≥6 h, prior diagnosis of myocardial infarction, unstable angina or revascularization at any time, cardiogenic shock, congestive heart failure, active infection, thyroid dysfunction, neoplastic disease, loss of consciousness, pregnancy, chronic renal disease (creatinine >1.5 mg/dl), degenerative or rheumatic valvular disease, prior valvular or any other prosthetic material or intracardiac defibrillator replacement, and prior any surgical intervention in the preceding 6 months. The study was approved by the local ethics committee, and all patients gave written informed consent to participate.

2.2. Definition of MetS in ACS patients

Given that acute metabolic derangements occur in association with ACS, all MetS diagnosis was established in the stable phase in patients with ACS. Prior diagnosis of hypertension was carefully recorded and the blood pressure values used were those collected on the day before discharge. Since the presence of fasting glycaemia at days 4 and 5 of an ACS accurately predicts glucose metabolism assessed at 3 months and represents a valid early marker of individuals at high risk of abnormal glucose metabolism, fasting glycaemia was determined from the mean blood glucose values at days 4 and 5 [19,20]. As a gradual decrease in mean HDL cholesterol and triglyceride levels during the in-hospital stay has been reported but is only minor during the first 24 h, blood samples for lipid values were drawn at the first following morning after admission [19,21,22].

2.3. Distribution of patients according to presence and absence of MetS or ACS

The study participants were divided into three groups as MetS (+) ACS (−) [Group I, n = 261], MetS (−) ACS (+) [Group II, n = 110], and MetS (+) ACS (+) [Group III, n = 163]. Subsequent to usage of NCEP criteria, every participant’s data were reevaluated for the diagnosis of MetS by using National Heart, Lung and Blood Institute/American Heart Association (NHLBI/AHA) and International Diabetes Federation (IDF) definitions [23,24]. Similar to creation of the three subgroups by using NCEP criteria, different subgroups were also generated according to each special definition of MetS.

2.4. Laboratory analysis

Since serum levels of CRP within 6 h after the onset of acute myocardial infarction (AMI) merely reflect a chronic and persistent inflammatory process without being affected by myocardial necrosis after AMI, venous blood samples for hs-CRP measurements were taken within the initial 6 h after the onset of chest pain in ACS patients [25,26]. Serum hs-CRP levels were measured by using an immunoassay with a validated, high sensitivity method with an autoanalyzer (IMMAGE Immunochemistry Systems, Beckman Coulter, California). The intra- and inter-assay coefficients for hs-CRP were approximately 5%. This assay has been validated against the Dade Behring hs-CRP method and has an inter- and intra-assay coefficient of variation (CV) of <8%. Among patients with ACS, bedside quantitative measurements of troponin I, CK-MB and myoglobin levels were analyzed by using Triage Cardiac Panel (Biosite Diagnostics). Hemogram, electrolyte levels, glucose, urea, creatinine, uric acid, lipid profile and all other biochemistry measurements of the whole study group were carried out by the analytical unit of the Biochemistry Department of our institution using standard methods.

2.5. Statistical analysis

SPSS 13.0 and MedCalc 8.1.0.0 statistical software packages were used for statistical analyses of the study. Results are presented as median and inter-quartile ranges or mean ± SD or as percentages and numbers for categorical data. Normality tests were used for all variables. Anthropometric measurements and metabolic risk factors were compared in the three groups with one-way analysis of variance (ANOVA). Homogeneity of variances was tested for all variables with Levene’s test. If equal variances were assumed post hoc Tukey HSD, if not Tamhane’s T2 tests were used to compare the parameters within the groups. Natural log transformation of the hs-CRP data achieved a normal distribution, so log-transformed hs-CRP values were used. For the comparison of hs-CRP levels and non-normally distributed variables between and within the groups, Kruskal–Wallis test and Mann–Whitney U tests were used. Categorical data and proportions were analyzed using Chi-square ($\chi^2$) or Fisher’s exact test where appropriate.

Multiple linear regression analysis was conducted with stepwise method by using log(hs-CRP) as a dependent variable to determine the relative contributions made by each variable (age, gender, body mass index, presence of ACS, troponin elevation and presence of NCEP ATP III defined MetS) to the outcome variable. Linear regression analysis was conducted in troponin-positive and troponin-negative cases separately to eliminate the potential confounding effect of myocardial necrosis on hs-CRP levels. The same analysis was repeated by using components of MetS instead of the presence of MetS as independent variables. Adjusted parameters in the model were age, gender, body mass index, presence of ACS, presence of troponin elevation, presence of abdominal obesity (≥102 in men, ≥88 in women), high fasting glucose (≥110 mg/dl), high triglyceride (≥150 mg/dl), hypertension criteria (≥130/80 mmHg or prior history of hypertension) and low HDL (≤40 mg/dl in men, ≤50 mg/dl in women).

Binary logistic regression analysis was performed by using forward Wald method for determining predictors of hs-CRP elevation which was defined as either >0.3 or >1 mg/dl. This analysis was performed in a sense if the presence of MetS has any effect on
these clinically important cut-off values identified from large scale randomized trials [27,28]. Hosmer–Lemeshow test was used to assess goodness-of-fit of the model. A p value over than 0.05 was considered as statistically significant in this test. Adjusted parameters in the model were age, gender, body mass index, presence of ACS, troponin elevation, and presence of NCEP ATP III defined MetS. Logistic regression analysis was also repeated after excluding troponin-positive cases. A p value less than 0.05 was considered as statistically significant.

3. Results

Mean age of the study population was 57 ± 10 years, 52% of subjects were male and 213 subjects (40%) were current smokers. The frequency of known hypertension was 72% and 182 patients (34%) had known diabetes mellitus. Median value of hs-CRP in the study population was 0.58 ± 22.9 mg/dl and ranged from 0.01 to 22.9 mg/dl.

Patients with ACS consisted of 121 patients with the diagnosis of unstable angina (USAP), 84 subjects with the diagnosis of Non-ST elevation myocardial infarction (NSTEMI) and 68 patients with the diagnosis of ST elevation myocardial infarction (STEMI). Baseline clinical and laboratory characteristics of patients with and without ACS were given in Table 1. Except mean values of fasting blood glucose, both metabolic and lipid parameters were significantly different between the two groups. Patients with ACS had significantly higher median levels of hs-CRP than patients without ACS.

3.1. Metabolic parameters and hs-CRP levels of patients with and without MetS or ACS

Table 2 shows the comparison of the metabolic characteristics of the three subgroups. Body mass index (BMI), hip circumference, systolic and diastolic blood pressure, fasting blood glucose, total cholesterol, HDL cholesterol and non-HDL cholesterol values of MetS (+) ACS (−) patients were signiﬁcantly higher than MetS (−) ACS (+) and MetS (+) ACS (+) patients.

Median value of hs-CRP was 0.44 mg/dl in MetS (+) ACS (−) group whereas 0.76 mg/dl in MetS (−) ACS (+) group and 1.19 mg/dl in MetS (+) ACS (+) group (Fig. 1A, p < 0.001). The presence of first ever attack of an ACS caused significant elevations and the presence of ACS with MetS caused markedly elevations in hs-CRP levels. This result was consistent with different analyses according to different definitions of MetS. No statistically significant difference was found about the effect of different definitions of MetS on hs-CRP levels in patients with and without ACS.

3.2. Levels of hs-CRP in various subgroups of ACS and among patients with and without troponin elevation

Median value of hs-CRP was 0.49 mg/dl in the USAP group whereas 1.45 mg/dl in NSTEMI and 1.63 mg/dl in STEMI groups (p < 0.001). In patients with the diagnosis of ACS (n = 223), patients with elevated troponin levels (n = 152) had significantly higher hs-CRP levels than patients without troponin elevation (1.51 mg/dl vs. 0.49 mg/dl, p < 0.001). When we exclude troponin-positive patients,

### Table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ACS (−) (n = 261)</th>
<th>ACS (+) (n = 273)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>54.5 ± 8.8</td>
<td>59.8 ± 10.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>80 (31%)</td>
<td>197 (72%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>32.6 ± 5.9</td>
<td>27.6 ± 4.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>103.0 ± 10.4</td>
<td>97.6 ± 11.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hip circumference, cm</td>
<td>109.9 ± 11.5</td>
<td>102.2 ± 11.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Waist to hip ratio</td>
<td>0.94 ± 0.07</td>
<td>1.33 ± 6.17</td>
<td>0.3</td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>146.2 ± 23.4</td>
<td>132.3 ± 25.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic blood pressure, mmHg</td>
<td>87.3 ± 10.2</td>
<td>78.8 ± 13.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting blood glucose, mg/dl</td>
<td>121.5 ± 32.7</td>
<td>100.2 ± 35.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total cholesterol, mg/dl</td>
<td>184.3 ± 78.5</td>
<td>178.5 ± 43.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triglyceride, mg/dl</td>
<td>43.6 ± 8.0</td>
<td>38.1 ± 11.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL cholesterol, mg/dl</td>
<td>129.3 ± 33.8</td>
<td>121.6 ± 38.3</td>
<td>0.02</td>
</tr>
<tr>
<td>Non-HDL cholesterol, mg/dl</td>
<td>169.0 ± 39.0</td>
<td>151.8 ± 42.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total cholesterol/HDL</td>
<td>4.9 ± 0.9</td>
<td>5.6 ± 1.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>hs-CRP, mg/dl</td>
<td>0.44</td>
<td>0.96</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

ACS: acute coronary syndrome.
the presence of first ACS event did not cause an elevation whereas the presence of MetS with ACS caused a statistically significant elevation in levels of hs-CRP. Median value of hs-CRP was 0.37 mg/dl in MetS (-) ACS (+) group and 0.63 mg/dl in MetS (+) ACS (+) group in this analysis (Fig. 1B, p < 0.001). Overall evaluation of the data revealed that presence of MetS further increased hs-CRP levels in addition to both the presence of ACS and troponin elevation (Fig. 2).

3.3. Distribution of subjects according to quartiles of hs-CRP

Frequency of patients with the lowest quartile of hs-CRP was highest in the MetS (+) ACS (-) group, whereas lowest in the MetS (+) ACS (+) group. Great amount of the MetS (+) ACS (+) group consisted of patients with the highest quartile of hs-CRP. These results were similar if patients were classified according to previously identified risky cut-off values of hs-CRP (Table 3).

3.4. Factors associated with levels of hs-CRP in the entire study group and among patients without troponin elevation

In all the study population, stepwise linear regression analysis showed that log(hs-CRP) levels were independently associated with elevated troponin levels (β = 0.4, 95% CI = 0.31–0.54, p < 0.01), presence of ACS (β = 0.3, 95% CI = 0.21–0.45, p < 0.01), and BMI (β = 0.02, 95% CI = 0.02–0.03, p < 0.01). R and R² values in the model were, 0.51 and 0.26, respectively.

When we exclude troponin-positive cases from the study population, factors independently associated with log(hs-CRP) levels were found as presence of ACS (β = 0.4, 95% CI = 0.30–0.54, p < 0.01), presence of MetS (β = 0.4, 95% CI = 0.26–0.59, p < 0.01) and BMI (β = 0.02, 95% CI = 0.02–0.03, p < 0.01). R and R² values in the model were, 0.42 and 0.18, respectively. When the same analysis was repeated by using individual components of MetS instead of presence of MetS as independent variables, none of the components were independent predictors of hs-CRP alone.

3.5. Predictors of hs-CRP elevation

By using a cut-off value of hs-CRP > 1 mg/dl as a dependent variable, binary logistic regression analysis model found that predictors of hs-CRP elevation were: presence of ACS (OR = 4.6, 95% CI = 2.6–8.2, p < 0.01) and troponin elevation (OR = 2.8, 95% CI = 1.7–4.6, p < 0.01) and BMI (OR = 1.9, 95% CI = 1.0–1.1, p < 0.01). When we exclude troponin-positive cases, repeated logistic regression analysis showed that predictors of hs-CRP elevation were: presence of ACS (OR = 6.1, 95% CI = 3.3–11.4, p < 0.01) and presence of MetS (OR = 2.4, 95% CI = 1.0–5.7, p = 0.05) and BMI (OR = 1.1, 95% CI = 1.0–1.1, p < 0.01). p values of Hosmer–Lemeshow goodness-of-fit tests were >0.05 in the two models.

By using a cut-off value of hs-CRP > 0.3 mg/dl as a dependent variable, binary logistic regression analysis showed that predictors of hs-CRP elevation were: presence of ACS (OR = 3.6, 95% CI = 1.9–6.5, p < 0.01), presence of MetS (OR = 2.1, 95% CI = 1.0–4.0, p = 0.02), troponin elevation (OR = 5.7, 95% CI = 2.8–11.5, p < 0.01) and BMI (OR = 1.1, 95% CI = 1.0–1.1, p < 0.01). When we exclude troponin-positive cases, predictors of hs-CRP elevation were: presence of ACS (OR = 4.9, 95% CI = 2.5–9.6, p < 0.01) and presence of MetS (OR = 3.0, 95% CI = 1.3–7.0, p < 0.01) and BMI (OR = 1.1, 95% CI = 1.0–1.2, p < 0.01). p values of Hosmer–Lemeshow goodness-of-fit tests were >0.05 in the two models.

4. Discussion

Recent observations suggest that baseline hs-CRP levels prior to ACS may contribute increased hs-CRP levels measured after ACS [11–13]. Although a clear relationship between hs-CRP and each component of MetS were shown, it is unclear whether MetS may...
cause a predisposition for heightened hs-CRP response in patients
with ACS [29–31]. The present study showed that MetS had a sig-
nificant impact on hs-CRP elevation among patients with first ACS
event. This result was confirmed by using various definitions of
MetS and validated in patients with and without troponin eleva-
tion.

Studies that incorporate MetS and ACS are mostly focused on the
prevalence of MetS in ACS or the impact of the presence of MetS
on outcomes [19,32–39]. However, these studies did not directly
examine the effect of the presence of MetS on hs-CRP levels and
did not explore the possible link between hs-CRP levels and adverse
events. Thus, potential association between elevated hs-CRP levels
among patients with MetS and ACS is unclear. Zeller and colleagues
found that MetS (+) ACS (+) patients had higher median hs-CRP lev-
els than MetS (−) ACS (+) patients. However, no relationship was
found between hs-CRP levels and in-hospital outcomes despite a
strong relation between the components of MetS and in-hospital
heart failure was established [19]. Boulon et al. examined the preva-
ce of MetS in ACS and validated in patients with and without troponin
elevation.

The different diagnostic components in different definitions of
MetS may also affect levels of hs-CRP [42,43]. This issue was

Table 3
Distribution of patients according to quartiles of hs-CRP and its previously identified risky cut-off values.

<table>
<thead>
<tr>
<th>Quartiles of hsCRP, mg/dl</th>
<th>MetS (+) ACS (−) (n = 261)</th>
<th>MetS (−) ACS (+) (n = 110)</th>
<th>MetS (+) ACS (+) (n = 163)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.26 (25th percentile)</td>
<td>87 (33%)</td>
<td>22 (20%)</td>
<td>24 (15%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>0.26–0.58 (25th to 50th percentile)</td>
<td>81 (31%)</td>
<td>26 (24%)</td>
<td>27 (17%)</td>
<td></td>
</tr>
<tr>
<td>0.58–1.46 (50th to 75th percentile)</td>
<td>68 (26%)</td>
<td>24 (22%)</td>
<td>42 (26%)</td>
<td></td>
</tr>
<tr>
<td>&gt;1.46 (75th percentile)</td>
<td>25 (10%)</td>
<td>38 (34%)</td>
<td>70 (42%)</td>
<td></td>
</tr>
</tbody>
</table>

Risky cut-off values of hsCRP, mg/dl (previously identified)

| <0.1 | 24 (9%) |
| 0.1–0.3 | 71 (27%) |
| >0.3 | 166 (64%) |
| 0.3–1 | 123 (47%) |
| >1  | 43 (17%) |

| p   |
| <0.01|

MetS: NCEP/ATP III defined metabolic syndrome; ACS: acute coronary syndrome.

trolling for its component risk factors [40]. These findings suggested
that MetS can be considered as a specific risk factor beyond its com-
ponents [40,41]. In the present study, although MetS was a stronger
predictor of hs-CRP levels, none of the individual components of
the MetS were the predictors of hs-CRP alone. Since elevated hs-
CRP clearly reflects increased cardiovascular risk, such speculation
should be the risk conferred by the MetS as a specific entity can also
be associated with the incremental effect of the MetS on hs-CRP
levels. Thus, further studies are required to explore this synergistic
effect.

The different diagnostic components in different definitions of
MetS may also affect levels of hs-CRP [42,43]. This issue was
addressed in a limited number of studies. Lin et al. compared the
IDF and NCEP ATP III definitions in Chinese people without ACS
and found that IDF definition of MetS had a stronger relationship
with hs-CRP levels than NCEP ATP III definition in men but not in
women [40]. Hirso et al. investigated whether the IDF, NCEP ATP III
and World Health Organization definitions of MetS differ regarding
the strength of their association with elevated hs-CRP (>0.3 mg/dl)
among a Finnish population. The investigators found a weaker asso-
ciation between IDF definition and hs-CRP levels, especially among
men [41]. In the present study, we found no statistically significant
difference between the effects of three different definitions on lev-
eels of hs-CRP. Nevertheless, whether the most important definition
of MetS related to levels of hs-CRP and outcomes in ACS patients
need further prospective investigation.

4.1. Limitations of the study

It is a single-center case–control study with a relatively limited
number of cases from a tertiary-hospital based population. Due to
its cross sectional methodology, prognostic conclusions and clini-
cal implication of elevated levels of hs-CRP in MetS (+) ACS (+)
patients cannot be drawn. The risk factors of ACS patients were
evaluated at the time of the index event. Therefore, how long
the risk factors had been present before the ACS event also could
not be reliably measured. Although the exact time of symptom
onset was carefully recorded in ACS patients, potentially inaccurate
duration from onset of chest pain to blood sample could also be
present. Hence, the effect of myocardial damage on serum hs-CRP
would not be completely eliminated in the present study. How-
ever, multivariate analyses after excluding troponin-positive cases
strengthened the hypothesis that MetS had an impact on hs-CRP
levels in patients with ACS in this cohort of the study popula-

5. Conclusions

In conclusion, the presence of the MetS had an impact on the
increase in hs-CRP levels observed with an ACS event in this cohort

Another controversial area of investigation is whether the risk
attributed to MetS is directly related to the presence of the MetS
itself as a specific entity or associated with the clustering of the
components that comprise the syndrome [40,41]. In a recent meta-
analysis, Gami and colleagues examined the cardiovascular risk in
the studies that simultaneously adjusted for MetS and its compo-
nents into multivariable models. An increased risk of cardiovascular
disease or death in patients with MetS was observed, even after con-

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of the study population. These findings suggested that MetS can be evaluated as an underlying predisposition toward greater inflammatory responsiveness that may mediate variation in the levels of hs-CRP and outcomes in patients with ACS. Further studies are required for the interaction between heightened hs-CRP response related to presence of MetS and outcomes in patients with ACS.

Conflict of interest

None.

References