Research Article

PREVALENCE OF THE METABOLIC SYNDROME AND ITS IMPACT ON HOSPITAL OUTCOMES IN PATIENTS WITH ACUTE ST ELEVATION MYOCARDIAL INFARCTION

Kushmanendra Parashar *, Anoop Jain, Neeraj Chaturvedi, Manish Ruhela
Department of Cardiology, SMS Medical College & Hospital, Jaipur, Rajasthan, India

Correspondence should be addressed to Kushmendra Parashar

Received February 20, 2015; Accepted March 5, 2015; Published March 9, 2015;
Copyright: © 2015 Kushmendra Parashar et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.


ABSTRACT

Aims and Objectives
To ascertain the prevalence of metabolic syndrome in patients of acute STEMI and to study the impact of the metabolic syndrome on hospital outcomes.

Methods and Result
Among the 324 patients with acute STEMI who were followed up till discharge from hospital, there were 122 patients with MS (group I) and 202 patients without MS (group II). Prevalence of the metabolic syndrome in our study was 37.65%. Prevalence of metabolic syndrome was significantly higher in female (59.45%) than male (31.2%) (*P* <0.05). Also, they showed higher proportion central obesity (*P* <0.001), hypertension (*P* <0.001), DM (*P* <0.001) and family history of coronary artery disease (*P* <0.001). but no significant difference in Left ventricular function, Killip class and in hospital mortality relative to group II (*P* >0.05). Whereas in patients without MS frequency of smokers was higher than MS group. Among the 5 components of MS blood pressure had the highest positive predictive value for acute MI followed by waist circumference, TG, HDL and impaired fasting glucose.

Conclusion
As MS is highly prevalent in the patients with acute STEMI such individuals could identify higher risk on the occurrence of ACSs, so awareness and preventative measures are important in hopes of improving outcomes in these patients.

KEY WORDS: STEMI, ACS, MS

INTRODUCTION

Coronary artery disease (CAD) is one of the commonest causes of death in developing and developed world. Various studies have revealed a consistent increase in prevalence of CAD in urban and rural population [1].

It has been known for several decades that risk factors for atherosclerotic cardiovascular disease often cluster together. At its simplest, the metabolic syndrome is a
Metabolic syndrome (MS) is characterized by the clustering of risk factors related to insulin resistance (IR) and is associated with an increased risk of cardiovascular disease [2][3][4][5][6]. In 2001, the National Cholesterol Education Program-Adult Treatment Panel (NCEP-ATP) III provided a definition for MS, and this is based on simple clinical criteria and is considered as a prognostic indicator of vascular risk in patients with no overt coronary artery disease[2][3][4][5]. In 2005, American Heart Association/National Heart Lung and Blood Institute (AHA/NHLBI) presented new criteria modified from NCEP-ATP III criteria[7]. Many studies in India have reported high prevalence of metabolic syndrome[8][9][10].

Furthermore, a recent study in patients with established symptomatic vascular disease (e.g. coronary artery disease, stroke, or peripheral arterial disease) showed that the prevalence of MS correlated with the extent of vascular damage.[11]

Little is known about the prevalence and the impact of the metabolic syndrome on hospital outcomes after presentation for an acute myocardial infarction (AMI) in South-East Asians, therefore we attempted to assess clinical characteristics, the prevalence of metabolic syndrome, its component and impact of metabolic syndrome on hospital outcomes in patients with acute ST elevation myocardial infarction.

MATERIALS AND METHODS

Study subjects

The current study was a observational descriptive study. 324 Patients with acute STEMI were enrolled in this study. A final diagnosis of MI was made in the presence of serial increases in serum biochemical markers of cardiac necrosis, associated with typical electrocardiographic changes and/or typical symptoms [12]. Patients with ST-segment elevation $\geq 1$ mm in $\geq 2$ extremity electrocardiographic leads or $\geq 2$ mm in $\geq 2$ contiguous precordial leads or new left bundle branch block on the admission electrocardiogram were defined as having STEMI. We analyzed baseline demographic and clinical characteristics, and relevant laboratory results. Echocardiography was performed in all patients. All patients were followed until hospital discharge for following end points:

i. Killip class at admission
ii. Occurrence of PMIA/reinfarction
iii. Severity of left ventricular dysfunction
iv. All-cause mortality/cardiac mortality Re-infarction was defined as the recurrence of symptoms or electrocardiographic changes in association with a rise in cardiac enzymes above the upper limit of normal.

For the diagnosis of MS at baseline, we used the NCEP-ATP III criteria. Merely, central obesity was defined as waist circumference $>90$ cm in men or $>80$ cm in women by modified ATP III guideline that WHO-Western Pacific Region (WPR) and International Association for the Study of Obesity (IASO) presented for Asian populations in 2000. ATP III definitions were based on the association of factors with subsequent coronary heart disease in Caucasian cohorts. As Indians have higher body fat content than their western counterparts for the same BMI, lower cut-offs of waist circumference were used as suggested by Asia-Pacific guidelines [19]. Waist circumference (WC) cut-offs were taken as $>90$ cm for males and $>80$ cm for females to define overweight.[13]. The presence of MS was analyzed considering the presence of the following criteria:

i. Central obesity: waist circumference $>90$ cm (men), $>80$ cm (women)
ii. A fasting triglyceride level $\geq 150$ mg/Dl
iii. Reduced high density lipoprotein (HDL) cholesterol: $<40$ mg/dL (men), $<50$ mg/dL (women)
iv. Hypertension: blood pressure $\geq 130/85$ mmHg or taking antihypertensive medication
v. Impaired fasting glucose (IFG): fasting glucose $\geq 100$ mg/dl or taking medication for past history of type 2 DM [14]. Patients were considered to have MS in the presence of $\geq 3$ of criteria, according to the definition proposed by the AHA/NHLBI [7].

Statistical analysis

The results were reported as mean $\pm$ standard deviation for the quantitative variables and percentages for the categorical variables. The groups were compared using the Student’s t-test for the continuous variables and the Chi-square test for the dichotomous variables. $P<0.05$ were considered as statistically significant. All calculation done by IBM SPSS PASW 18 software.

RESULTS

Baseline clinical characteristics of the study population

Among the 324 patients with acute STEMI who were followed up till discharge from hospital, there were 122 patients with MS and 202 patients without MS. Prevalence of the metabolic syndrome in our study was 37.65%. Prevalence of metabolic syndrome was significantly higher in females (59.45%) than males (31.2%) ($P<0.05$). Among the 5 components of MetS blood pressure had the highest positive predictive value for acute MI followed by waist circumference, TG, HDL and impaired fasting glucose (Figure 1).
### Table 1

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>NON MS(202)</th>
<th>MS(122)</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>SEX(MALES)</td>
<td>172 85</td>
<td>78 64</td>
<td>.001</td>
</tr>
<tr>
<td>CAD</td>
<td>11 5</td>
<td>4 3</td>
<td>.427</td>
</tr>
<tr>
<td>DM</td>
<td>26 13</td>
<td>58 48</td>
<td>.001</td>
</tr>
<tr>
<td>HTN</td>
<td>64 32</td>
<td>87 71</td>
<td>.001</td>
</tr>
<tr>
<td>WC</td>
<td>48 24</td>
<td>78 64</td>
<td>.001</td>
</tr>
<tr>
<td>SMOKING</td>
<td>104 51</td>
<td>52 43</td>
<td>.314</td>
</tr>
<tr>
<td>F/H</td>
<td>37 18</td>
<td>36 30</td>
<td>.024</td>
</tr>
<tr>
<td>KILLIP- 1</td>
<td>148 74</td>
<td>85 70</td>
<td>.42</td>
</tr>
<tr>
<td>KILLIP- 2</td>
<td>45 22</td>
<td>22 18</td>
<td>.351</td>
</tr>
<tr>
<td>KILLIP- 3</td>
<td>5 2</td>
<td>12 10</td>
<td>.11</td>
</tr>
<tr>
<td>KILLIP- 4</td>
<td>4 2</td>
<td>3 2</td>
<td>.997</td>
</tr>
<tr>
<td>PMIA_REINFARCT</td>
<td>49 24</td>
<td>36 30</td>
<td>.304</td>
</tr>
<tr>
<td>MORTALITY</td>
<td>18 9</td>
<td>18 15</td>
<td>.123</td>
</tr>
<tr>
<td>AWMI</td>
<td>115 57</td>
<td>63 51</td>
<td>.326</td>
</tr>
<tr>
<td>NON-AWMI</td>
<td>87 43</td>
<td>49</td>
<td>.354</td>
</tr>
</tbody>
</table>

### Table 2

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>GROUP</th>
<th>MEAN</th>
<th>S.D.</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>WC</td>
<td>NON MS</td>
<td>80.97</td>
<td>5.675</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>MS</td>
<td>91.26</td>
<td>9.009</td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>NON MS</td>
<td>123.81</td>
<td>19.068</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>MS</td>
<td>138.02</td>
<td>23.420</td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>NON MS</td>
<td>79.27</td>
<td>11.390</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>MS</td>
<td>87.25</td>
<td>14.385</td>
<td></td>
</tr>
<tr>
<td>FBG</td>
<td>NON MS</td>
<td>89.52</td>
<td>22.918</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>MS</td>
<td>121.66</td>
<td>39.459</td>
<td></td>
</tr>
<tr>
<td>LDL</td>
<td>NON MS</td>
<td>104.29</td>
<td>23.351</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>MS</td>
<td>117.22</td>
<td>27.635</td>
<td></td>
</tr>
<tr>
<td>TG</td>
<td>NON MS</td>
<td>119.00</td>
<td>36.516</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>MS</td>
<td>153.09</td>
<td>52.217</td>
<td></td>
</tr>
<tr>
<td>HDL</td>
<td>NON MS</td>
<td>45.81</td>
<td>5.763</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>MS</td>
<td>41.65</td>
<td>6.892</td>
<td></td>
</tr>
<tr>
<td>LV</td>
<td>NON MS</td>
<td>40.25</td>
<td>8.270</td>
<td>.577</td>
</tr>
<tr>
<td></td>
<td>MS</td>
<td>40.79</td>
<td>8.343</td>
<td></td>
</tr>
<tr>
<td>WP(hrs)</td>
<td>NON MS</td>
<td>6.10</td>
<td>4.116</td>
<td>.782</td>
</tr>
<tr>
<td></td>
<td>MS</td>
<td>6.23</td>
<td>4.527</td>
<td></td>
</tr>
</tbody>
</table>
In-hospital outcome

Estimated in-hospital mortality from present study was 15% in the group I and 9% in the group II ($P=0.123$). In our study relation of in hospital mortality with age, HTN, dyslipidemia, Killip class at admission, post MI angina, left ventricular ejection fraction and window period were statistically significant.

DISCUSSION

The results of previous studies suggested that MS was very common among patients with coronary artery disease, because almost a half of patients had MS and that it was associated with advanced vascular damage [15][16]. In contrast, recently, some experts have raised concerns about the clinical validity of MS [17][18], and its clinical significance remained controversial. Our study, based on an unselected population of patients hospitalized with MI, confirmed the high prevalence of MS in patients with acute STEMI. More advanced vascular damage has been associated with the presence of MS in patients with manifest vascular disease, which may worsen the prognosis [16]. MS represents a cluster of several risk factors, each of which may be involved in this poor outcome.

In our study MetS and its components have consistently been associated with ACS developed, MetS did not correlate with in-hospital mortality. Which is similar to finding in study done by Zeller M et al [19] and Pandey et al. [20]

Among patients who have a history of acute MI, MS was recently shown to be associated with a higher rate of all-cause death and the composite of cardiovascular death, non-fatal stroke, and non-fatal MI [21]. MS has also been shown to be associated with a higher incidence of severe

Table 3

<table>
<thead>
<tr>
<th>PREDICTORS</th>
<th>B</th>
<th>S.E.</th>
<th>P VALUE</th>
<th>Exp(B)</th>
<th>95% C.I. for EXP(B)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lower</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Upper</td>
</tr>
<tr>
<td>AGE</td>
<td>0.076</td>
<td>0.032</td>
<td>0.017</td>
<td>1.079</td>
<td>1.014</td>
</tr>
<tr>
<td>HTN</td>
<td>2.845</td>
<td>0.862</td>
<td>0.001</td>
<td>17.205</td>
<td>3.175</td>
</tr>
<tr>
<td>DYSLIPIDEMIA</td>
<td>2.821</td>
<td>0.866</td>
<td>0.001</td>
<td>16.800</td>
<td>91.681</td>
</tr>
<tr>
<td>KILLIP</td>
<td>2.374</td>
<td>0.635</td>
<td>0.001</td>
<td>10.746</td>
<td>37.270</td>
</tr>
<tr>
<td>PMIA_REINFARCT</td>
<td>2.120</td>
<td>0.734</td>
<td>0.004</td>
<td>8.330</td>
<td>35.117</td>
</tr>
<tr>
<td>LV</td>
<td>-0.102</td>
<td>0.049</td>
<td>0.037</td>
<td>0.903</td>
<td>0.821</td>
</tr>
<tr>
<td>WP_HRS</td>
<td>0.195</td>
<td>0.085</td>
<td>0.022</td>
<td>1.216</td>
<td>1.029</td>
</tr>
</tbody>
</table>

Figure 1: comparison of MS criteria for both MS and Non-MS patients
heart failure following acute MI [19]. Previous study showed that MS was a strong predictor of late-onset DM in post-MI and the risk of death among patients with MS was mainly associated with the transformation in DM [21]. If more prolonged follow-up period is fulfilled, it would be possible that long-term outcome would be meaningfully increased in patient with MS relative to patient without MS. There are a number of reasons why MS could predispose to short-term mortality. Its components, such as central obesity, insulin resistance, dyslipidemia, hypertension, are all risk factors for endothelial dysfunction, which is an important factor in the pathophysiology of atherosclerosis and acute coronary syndromes. Hallmarks of the MS include a prothrombotic proinflammatory state and markers of inflammation have been found to correlate with the presence of MS in survivors of acute MI [7][22][23]. Patients with impaired glucose tolerance found during admission for acute MI have an increased rate of non-fatal stroke, non-fatal MI, severe heart failure, and cardiovascular death [24].

The association between MS and poor prognosis highlights the clinical relevance of this syndrome, especially given the high prevalence among patients presenting with acute MI. Patients with MS should be identified and cared appropriately, given the increased prevalence of MS in patients with acute STEMI. Although there are currently no specific treatments directed at the MS as a whole, treating the individual components via lifestyle modification and lipid correcting agents have shown to slow the progression of MS and reduce the risk of cardiovascular disease [21][25].

This study has several limitations. First, our study is single center prospective study, and it was not a randomized, controlled study. Thus there was probably a selection bias when enrolling patients into both study groups. Second, although we assessed risk factors at the time of the index event, we could not reliably measure how long the risk factors had been present before the MI. Finally, the period of our study is relatively short, because our study is a comparison of the in hospital outcome.

In conclusion, Presence of MS was not a predictor of in-hospital death events in patients with STEMI . As MS is highly prevalent in the patients with acute STEMI such individuals could identify higher risk on the occurrence of ACSs, so awareness and preventative measures are important in hopes of improving outcomes in these patients.

REFERENCES


