Biochemical changes in adiponectin and myeloperoxidase in acute myocardial infarction
Raafat R. Mohammed, Hussein Abdel-Maksoud, Yasser M. Abdel-Nabi

Background
Nitric oxide is a common mediator for the action of adiponectin and myeloperoxidase (MPO). Its importance is as a predictor of acute myocardial infarction (AMI) severity.

Aim
The aim of the present study was to find a relationship between plasma adiponectin level, MPO activity, lipid profile, serum nitrite/nitrate, and severity of AMI disease.

Patients and methods
To achieve this aim, 30 patients with AMI with age ranged from 35 to older than 70 years and 10 clinically healthy participants as control were subjected to this study.

Results
The result of this study showed a significant association between the occurrence of AMI and low adiponectin level, high MPO activity, low nitrite level, high total cholesterol level, high triacylglycerol level, high low-density lipoprotein-cholesterol level, low high-density lipoprotein-cholesterol level, and high low-density lipoprotein-cholesterol/high-density lipoprotein-cholesterol ratio. These parameters may all be regarded as predictors or risk factors for AMI.

Conclusion
The findings of the present study suggest that hyperlipidemia, vascular inflammation, and oxidative stress are primary interacting mediators in the pathogenesis of AMI.

Keywords: acute myocardial infarction, adiponectin, myeloperoxidase

Introduction
Adipose tissue secretes many proatherogenic and anti-atherogenic adipokines such as leptin and adiponectin that play an important role in the pathogenesis of atherosclerosis and coronary artery disease [1]. The adipokines, leptin and adiponectin, are considered as a potentially important link between systemic inflammatory processes and cardiovascular disease [2,3].

Myeloperoxidase (MPO) is a leukocyte-derived proinflammatory and proatherogenic enzyme that participates in the processes of development, progression, and complication of coronary artery disease through various mechanisms [4]. MPO converts low-density lipoprotein (LDL) into an atherogenic form, oxidized LDL; provides oxidative stress condition; and decreases nitric oxide (NO) bioavailability, all leading to atherosclerosis [5–8]. Thus, adiponectin was hypothesized to play a role in acute myocardial infarction (AMI) [9], and MPO also plays a pathophysiologic role in AMI [10].

NO has a role in both acute and chronic inflammation and was proposed to play a role in AMI atherogenesis [11].

This study was aimed to find a relationship among plasma adiponectin level, MPO activity, lipid profile, and serum NO metabolites (nitrite/nitrate) in patients with AMI.
(3) Group III consists of 10 patients with AMI from 51 to 70 years.

(4) Group IV consists of 10 patients with AMI with age more than 70 years.

A cardiologist in the coronary care unit finally established the diagnosis of AMI guided by the WHO criteria.

Sampling
During coronary angiography, using a cardiac catheter, 10 ml of blood was withdrawn and collected from every patient and control healthy participant after overnight fasting. The blood samples were divided into two parts.

The first one was evacuated into tubes with 5% EDTA. Plasma samples were collected after centrifugation and used freshly for determination of adiponectin concentration [13]. The remaining granulocyte/erythrocyte pellets were further processed for separation of neutrophils to assess MPO activity [14,15].

The remaining 5 ml of blood is evacuated in tubes without an anticoagulant and then allowed to clot and centrifuged for isolation of serum, which was used freshly for determination of NO metabolites (nitrate/nitrite) [16], total cholesterol [17], triacylglycerol (TG) [18], high-density lipoprotein-cholesterol (HDL-C), LDL-C[19], and very low-density lipoprotein-cholesterol (VLDL-C) concentrations [20].

Results
The data presented revealed that AMI is accompanied by significant decrease (*P* < 0.05) in the mean values of plasma adiponectin level, serum nitrite, serum nitrate, and serum HDL-C and a significant increase (*P* < 0.05) in the activity of MPO, serum total cholesterol, TG, LDL-C, and VLDL-C in comparison with the mean values recorded in the control healthy individual group (Tables 1 and 2).

Discussion
Myocardial infarction is exhibited in 10–15% of patients presented to the hospitals with chest pain annually [21].

Cardiovascular biomarkers including MPO, adiponectin, interleukins, and chemokines are under intensive validation and research [22].

The present study showed that plasma adiponectin level was significantly lower in patients with AMI (groups II, III, and IV) compared with the control group, which was in agreement with previous studies [23–25], and this might be owing to a strong inflammatory activity in the atheromatous plaque [26].

The reported significant negative correlation between adiponectin level and age in patients with AMI could be owing to a possible disturbed adipokines synthesis or secretion in old-age individuals, an explanation that might support the concept of old age being a risk factor [27].

Moreover, the reported significant positive correlation between adiponectin and NO metabolites (nitrite/nitrate) levels could be explained by the assumption that

Table 1 Mean values±SE of plasma adiponectin (ng/ml), myeloperoxidase activity (unit/mg protein), serum nitrite (μmol/l), and serum nitrate (μmol/l) in control healthy and individuals with AMI

<table>
<thead>
<tr>
<th>Group</th>
<th>Plasma adiponectin (ng/ml)</th>
<th>MPO activity (unit/mg protein)</th>
<th>Serum nitrite (μmol/l)</th>
<th>Serum nitrate (μmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>6408.80±80.75</td>
<td>2.35±0.71</td>
<td>6.70±0.33</td>
<td>10.78±1.11</td>
</tr>
<tr>
<td>II</td>
<td>2730.90±96.50*</td>
<td>5.99±0.89*</td>
<td>4.25±0.29</td>
<td>6.84±0.97</td>
</tr>
<tr>
<td>III</td>
<td>2580.53±82.73*</td>
<td>9.13±0.77**</td>
<td>3.38±0.39*</td>
<td>6.01±0.89*</td>
</tr>
<tr>
<td>IV</td>
<td>2004.80±71.50**</td>
<td>14.57±1.11**</td>
<td>2.17±0.91**</td>
<td>4.75±0.38**</td>
</tr>
</tbody>
</table>

MPO, myeloperoxidase. *P* > 0.05, significant. **P > 0.01, significant.

Table 2 Mean values±SE of serum total cholesterol (mg/dl), triacylglycerol (mg/dl), HDL-cholesterol (mg/dl), LDL-cholesterol (mg/dl), and VLDL-cholesterol (mg/dl) in healthy control and individuals with acute myocardial infarction

<table>
<thead>
<tr>
<th>Group</th>
<th>Serum total cholesterol (mg/dl)</th>
<th>Serum triacylglycerol (mg/dl)</th>
<th>Serum HDL-cholesterol (mg/dl)</th>
<th>Serum LDL-cholesterol (mg/dl)</th>
<th>Serum VLDL-cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>138.60±3.40</td>
<td>97.25±2.11</td>
<td>49.81±2.13</td>
<td>70.10±3.15</td>
<td>19.45±0.42</td>
</tr>
<tr>
<td>II</td>
<td>198.75±3.91</td>
<td>174.28±3.09*</td>
<td>32.40±2.11</td>
<td>130.61±4.11*</td>
<td>34.86±0.62*</td>
</tr>
<tr>
<td>III</td>
<td>236.66±5.01*</td>
<td>200.81±3.27**</td>
<td>33.92±2.75</td>
<td>162.59±4.19**</td>
<td>40.03±0.66**</td>
</tr>
<tr>
<td>IV</td>
<td>265.35±5.90*</td>
<td>250.13±4.17**</td>
<td>24.16±2.25</td>
<td>191.31±4.89***</td>
<td>50.13±0.84**</td>
</tr>
</tbody>
</table>

HDL, high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very low-density lipoprotein. *P > 0.05, significant. **P > 0.01, significant. ***P > 0.001, significant.
Adiponectin increases NO production by promoting the activity of endothelial nitric oxide synthase or by ameliorating the suppression of endothelial nitric oxide synthase activity by oxidized LDL [28].

It was found that MPO activities serve as a strong and independent predictor of endothelial dysfunction in humans, giving a mechanistic link between oxidation, inflammation, and cardiovascular disease [29]. Moreover, MPO causes modification of HDL in AMI and generates dysfunctional HDL [30]. Studies have shown that MPO levels are a predictor of death after chest pain and AMI. MPO has recently been shown to contribute to microvascular obstruction in patients with AMI. During myocardial ischemia-reperfusion sequences, microvascular function is associated with recruitment of polymorphonuclear neutrophils and has been attributed to decreased bioavailability of NO. Endothelium-dependent microvascular function is a multifactorial process involving endothelial injury or dysfunction, neutrophil accumulation, overproduction of reactive oxygen species, thrombus embolization, and activation of the coagulation cascade [31].

The present study showed a significant increase in MPO activity in patients with AMI (groups I, II, and III) compared with the controls. This might be related to its secretion from activated leukocytes under inflammatory conditions [32].

Similarly, MPO has been shown to activate metalloproteinases and to promote destabilization and rupture of atherosclerotic plaque surface, thus MPO could be related to the future risk of AMI events [29].

A significant negative correlation was shown in this study between MPO activity and NO metabolites (nitrite/nitrate) levels, due to the uptake of MPO by endothelial cells through transcytotic process to accumulate within the subendothelial space, and to consume NO, thus interfering with the atheroprotective effect of NO [33].

Estimated serum levels of both NO metabolites (nitrite and nitrate) were significantly lower in patients with AMI compared with controls. Increasing number of cardiovascular risk factors was correlated with the degree of decrease in nitrite level. There were high levels of NO metabolites in both acute and chronic inflammatory conditions including atherosclerosis [34].

The levels of lipid profile were significantly changed in patients with AMI, which is in agreement with a study that had significantly higher levels of total cholesterol, LDL-C, VLDL-C, triglycerides, LDL-C/HDL-C, total cholesterol/HDL-C, and lower levels of HDL-C as compared with the controls [35].

In the present study, a significant correlation between lipid profile and adiponectin and MPO was shown. In addition, a significant correlation was found between lipid profile and NO metabolites as mentioned before [36].

**Conclusion**

In conclusion, AMI occurs accompanied by low levels of adiponectin, nitrite, nitrate, and HDL-C and high levels of MPO activity, total cholesterol, TG, and LDL-C ratio. These may all be regarded as risk factors and could be used as diagnostic tools for AMI.

The present study findings show the importance of NO as a predictor of AMI severity, a common mediator for the action of adiponectin and MPO, besides its possible interaction with dyslipidemia and hypertension. These findings point to the importance of NO in diagnosis and treatment of AMI.

**Financial statement and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**

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