Comparison of pentraxin-3 and ischemia-modified albumin with troponin in early diagnosis of acute coronary syndrome

Demir MT¹, Baydin A², Amanvermez R³, Erenler AK⁴, Güzel M¹, Yücel O⁵

Ondokuz Mayis University, Faculty of Medicine, Department of Emergency Medicine, Samsun, Turkey.

ahmetbaydn@yahoo.com

ABSTRACT
INTRODUCTION: In this study, our aim was to evaluate clinical utilities of Pentraxin 3 (PTX3) and Ischemia-modified Albumin (IMA) in diagnosis of acute coronary syndrome (ACS) and compare these two biomarkers with a conventional diagnostic marker, cardiac troponin I (cTnI).

MATERIALS AND METHODS: Sixty adult patients with ACS diagnosis were involved into this prospective study. Additionally, 20 healthy subjects were determined as control group (Group IV). Patients were divided into 3 groups as follows: Patients with Acute Myocardial Infarction (STEMI Group, n=20, Group I), patients without ST elevation but with elevated cTnI levels (NSTEMI Group, n=20, Group II), and patients with unstable angina pectoris (USAP Group, n=20, Group III). Blood measurements were obtained for each marker at admission and in the 4th hour.

RESULTS: Troponin level was significantly different between groups I and II at both admission and in the 4th hour. Additionally, PTX 3 level was significantly different at admission and 4th hour between groups II and III.

CONCLUSION: This study revealed that cTnI is the most sensitive test in ACS diagnosis at the admission to Emergency Department. Our results also revealed that PTX 3 may be a useful diagnostic tool for ACS at admission, however, IMA alone cannot be used for diagnosis of ACS. Similarly, in the 4th hour, cTnI was found to be the most useful marker in ACS diagnosis, however, PTX 3 and IMA were found to be inadequate for diagnosis of ACS (Tab. 3, Ref. 19).

KEY WORDS: acute coronary syndrome, cardiac troponin i, ischemia-modified albumin, pentraxin 3, emergency department.

Introduction

Acute coronary syndrome (ACS) involves a broad spectrum of complaints from angina pectoris to irreversible myocardial damage resulting in acute myocardial infarction (AMI) (1). Recently, many potential biomarkers have been studied for the early and appropriate diagnosis of AMI. Cardiac troponin I (cTnI) and troponin T are commonly used for determining the extent of cardiac muscle injury in AMI. However, studies for novel early diagnostic biomarkers are still ongoing (2).

Ischemia-modified albumin (IMA) is a marker formed after damage in the N-terminal region of albumin. The causes of the increases in IMA have been shown to be endothelial or extracellular hypoxia, acidosis, and free oxygen radicals (34).

Similar to IMA, Pentraxin 3 (PTX 3) is known to be released as a specific response to vascular damage and PTX 3 levels are likely to be strongly related to later stages of atherosclerosis (5). In this article, our aim was to investigate the usefulness of IMA and PTX 3 in the early diagnosis of AMI and compare their utility with conventional marker, cTnI.

Materials and methods

After ethical approval form Local Ethics Committee, 60 adult patients with ACS diagnosis admitted to Coronary Intensive Care Unit were involved into this prospective study. Additionally, 20 healthy subjects formed a control group. Patients with ACS were divided into 3 groups as follows: Patients with AMI confirmed by ST elevation on serial electrocardiograms (ECGs) and cTnI measurements (STEMI Group, n = 20, Group I), patients without ST elevation but with elevated cTnI levels (NSTEMI Group, n = 20, Group II), and patients with unstable angina pectoris (USAP Group, n = 20, Group III). Control group was named as Group IV.

Patients with chronic obstructive pulmonary disease, renal failure, collagen tissue disease, vasculitis, depression, somatization disorder and patients with a medical history of coronary angiography, coronary by-pass and open heart surgery were excluded in order to avoid false positive cTnI levels.
Blood samples of the patients were evaluated at admission and in the 4th hour of follow-up. Blood samples for IMA and PTX 3 were taken at admission and in the 4th and were centrifuged at 3500 rpm for 15 minutes, and serum samples were stored at –80 °C until analysis.

CK-MB and cTnI levels were measured in serum by Simens ADVIA Centaur Cp analyzers in emergency laboratory.

Serum PTX 3 level was measured by ELISA using a kit (USCN Life Science Inc. Cloud-Clone Corp. Houston, USA, Lot No: W22169285). For this measurement, sandwich immunoassay technique was used.

Statistical analysis

For statistical analyses, SPSS 17.0 programme was used. Data are presented as arithmetic mean ± standard deviation and median (minimum–maximum) values. For evaluation of normality, Shapiro–Wilk test was used. In comparison of groups, One Way ANOVA was used for normal distribution and Kruskal–Wallis variance analysis was used for non-normal distribution. For comparison of homogenous two groups, Tukey was used for homogenous variance and Tamhane test was used for heterogeneous variance. For comparison of two normally distributed groups, Bonferroni corrected Mann Whitney U test was used. Pearson correlation test was performed to test correlation between normally distributed data and Spearman’s correlation test was used for non-normally distributed data. Diagnostic values of the markers were evaluated by Receiver Operating Characteristic (ROC) Analysis and Area Under Curve (AUC). p < 0.05 was considered statistically significant.

Results

Demographical and laboratory findings of the groups are summarized in Tables 1 and 2. Troponin level was significantly different in groups I and II at both admission and the 4th hour when

Tab. 1. Groups according to demographic findings.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Gender</th>
<th>Age±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>17 (85%)</td>
<td>3 (15%)</td>
</tr>
<tr>
<td>Group II</td>
<td>15 (75%)</td>
<td>5 (25%)</td>
</tr>
<tr>
<td>Group III</td>
<td>17 (85%)</td>
<td>3 (15%)</td>
</tr>
<tr>
<td>Group IV</td>
<td>14 (70%)</td>
<td>6 (30%)</td>
</tr>
</tbody>
</table>

For comparison of two normally distributed groups, Bonferroni corrected Mann Whitney U test was used. Pearson correlation test was performed to test correlation between normally distributed data and Spearman’s correlation test was used for non-normally distributed data. Diagnostic values of the markers were evaluated by Receiver Operating Characteristic (ROC) Analysis and Area Under Curve (AUC). p < 0.05 was considered statistically significant.

Tab. 2. Laboratory findings of the groups.

<table>
<thead>
<tr>
<th>Laboratory Findings (On admission)</th>
<th>Group I (n = 20)</th>
<th>Group II (n = 20)</th>
<th>Group III (n = 20)</th>
<th>Group IV (Controls) (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>White blood cell (3.7–9.7 thousand/uL)</td>
<td>12±3.5 (10.7–17.5)</td>
<td>10.6±3.2 (10.7–18.4)</td>
<td>9.2±2.5 (4.6–13.6)</td>
<td>7.1±1.5 (4.9–10.9)</td>
</tr>
<tr>
<td>Haemoglobin (13.3–17.2 g/dL)</td>
<td>14.36±2.4 (14.9–17.2)</td>
<td>13.9±2.0 (14.4–17.1)</td>
<td>14.4±1.5 (12.0–17.6)</td>
<td>13.8±1.6 (11.4–16.5)</td>
</tr>
<tr>
<td>Platelet (179–373 thousand/uL)</td>
<td>238.9±50.2 (239.5–346.0)</td>
<td>240.8±66.2 (232.5–394.0)</td>
<td>246.0±112.7 (224.5–538.0)</td>
<td>249.5±48.4 (244.0–342.0)</td>
</tr>
<tr>
<td>Glucose (70–110 mg/dL)</td>
<td>176.2±84.8 (136.0–401.0)</td>
<td>155.4±71.9 (130.5–380.0)</td>
<td>159.7±86.3 (122.0–396.0)</td>
<td>94.9±12.2 (72.0–121.0)</td>
</tr>
<tr>
<td>BUN (5–24 mg/dL)</td>
<td>17.8±7.2 (9.0–44.0)</td>
<td>18.0±4.7 (10.0–27.0)</td>
<td>19.4±12.3 (16.0–63.0)</td>
<td>10.9±3.3 (4.0–19.0)</td>
</tr>
<tr>
<td>Creatine (0.4–1.4 mg/dL)</td>
<td>0.9±0.2 (0.9–1.5)</td>
<td>0.9±0.2 (0.9–1.5)</td>
<td>0.9±0.2 (0.9–1.5)</td>
<td>0.9±0.2 (0.9–1.5)</td>
</tr>
<tr>
<td>AST (8–46 UL)</td>
<td>48.5±37.8 (34.0–143.0)</td>
<td>56.30±48.2 (47.00–233.0)</td>
<td>25.7±12.8 (22.00–57.00)</td>
<td>18.95±3.25 (18.0–26.0)</td>
</tr>
<tr>
<td>ALT (7–46 UL)</td>
<td>34.4±14.5 (32.0–67.0)</td>
<td>30.6±12.6 (28.5–64.0)</td>
<td>28.9±21.6 (22.5–96.0)</td>
<td>19.7±8.8 (18.5–43.0)</td>
</tr>
<tr>
<td>Creatin Kinase (35–195 UL)</td>
<td>339.6±358.7 (139.0–1239.0)</td>
<td>428.9±526.9 (328.5–2456.0)</td>
<td>218.3±345.5 (101.0–1484.0)</td>
<td>171.2±154.2 (137.5–64.0–796.0)</td>
</tr>
<tr>
<td>CK-MB (0–10 mg/dL)</td>
<td>48.3±42.1 (37.0–200.0)</td>
<td>67.3±99.8 (40.0–479.0)</td>
<td>25.6±13.0 (20.5–51.0)</td>
<td>14.4±1.31 (12.0–18.0)</td>
</tr>
<tr>
<td>Calcium (8.6–10.0 mg/dL)</td>
<td>9.4±0.7 (9.5–10.0)</td>
<td>9.5±0.5 (8.3–10.3)</td>
<td>9.8±0.7 (8.8–11.6)</td>
<td>9.9±0.6 (9.0–11.8)</td>
</tr>
<tr>
<td>Sodium (135–145 mEq/L)</td>
<td>138.8±3.7 (139.5–145.0)</td>
<td>139.3±2.8 (139.0–145.0)</td>
<td>139.6±2.9 (139.5–140.0)</td>
<td>140.1±1.5 (136.0–142.0)</td>
</tr>
<tr>
<td>Potassium (3.5–5.5 mEq/L)</td>
<td>4.0±0.5 (3.90–4.3)</td>
<td>4.3±0.5 (4.2–4.4)</td>
<td>4.3±0.4 (4.2–4.3)</td>
<td>4.3±0.34 (3.8–5.0)</td>
</tr>
<tr>
<td>Chlorine (99–110 mEq/L)</td>
<td>104.0±4.2 (96.0–113.0)</td>
<td>105.0±3.9 (97.0–115.0)</td>
<td>105.4±3.9 (97.0–112.0)</td>
<td>104.0±1.8 (101.0–107.0)</td>
</tr>
</tbody>
</table>
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conventional markers of AMI such as CK-MB and troponin levels. They concluded that the use of IMA alone was not adequate as a marker in diagnosis of ACS and should be used in combination with other markers in order to increase its specificity (16). In our study, IMA was found to be useful neither in ACS diagnosis on admission nor in the 4th hour.

Our results revealed that IMA level correlated with creatine level compatible with the fact that IMA level is affected by renal function. In end-stage renal disease (ESRD) patients, serum IMA levels were found to be high because of anemia, leading to generalized hypoxia. In another study, Türedi et al. reported that IMA levels of ESRD patients, both pre and post-hemodialysis, were significantly higher than those of the control group (17, 18). However, another result of our study, correlation of WBC with IMA and PTX needs to be clarified. Previously, Baydin et al. reported that in patients with carbon monoxide poisoning, when hypoxic condition is causing damage to oxygen dependent tissues such as heart and brain, PTX3 and IMA were not superior to cTnI (19). Their results are in compliance with ours that these two markers cannot be used as independent predictors of ACS.

Conclusion

Superiority of troponins, particularly cTnI, in the diagnosis of ACS is still accepted. Serum PTX 3 and IMA measurements alone do not reflect cardiovascular status of patients. PTX 3 may differentiate patients with NSTEMI and USAP. However, cTnI has advantage of being readily available in most of the EDs.

References


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