Peak CRP Levels are Higher in Patients with ST Elevation Than Non-ST Elevation Acute Coronary Syndrome

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INTRODUCTION

- A large body of evidence suggests that inflammation plays a key role in the pathogenesis of atherosclerosis.
- The chronic inflammatory process can develop into an acute clinical event by the induction of plaque rupture, leading to acute coronary syndromes.
- CRP levels increase after acute myocardial infarction (AMI) but their changes in the process of an acute ischemic attack has been studied mainly in patients with non-ST elevation AMI.

CRP is locally generated from Endothelium also

Cytokines
- IL-1β & IL-6
- TNF α

Phospholipase A2
ICAM
VCAM
E & P Selectin
Trop T
CKMB
Adipose Tissue

CRP
Fibrinogen
SAA

Rader. NEJM. 2000; 343(16):1179
OBJECTIVE

To study the differences in hsCRP levels in patients with two clinical forms of ACS of non-ST elevation myocardial infarction compared to ST myocardial infarction.
METHODS

Acute Coronary Syndrome

No ST Elevation
- NSTEMI
- Unstable Angina

ST Elevation
- Myocardial Infarction
- NQMI
- QwMI
Recruited N = 94

FINALLY SELECTED = 89

CONTROL
N=29

♀ 11 ♂ 18

NSTEMI
N=28

♀ 7 ♂ 21

STEMI
N=32

♀ 10 ♂ 22

HS-CRP AT BASELINE PEAK AND FOLLOW UP LIPOPROTEIN(a)
**INCLUSION CRITERIA**

**NSTEMI:** Patients with NSTEMI were required to have angina-like chest pain at rest in the last 24 h lasting ≥ 5 min, with associated ST-segment depression of ≥ 0.1 mV in ≥ 2 contiguous leads upon presentation.

**STEMI:** (1) continuous chest pain upon presentation, refractory to nitrates, and lasting ≥ 30 min; (2) ST-segment elevation of ≥ 0.2 mV in ≥ 2 contiguous precordial leads, or ≥ 0.1 mV in ≥ 2 contiguous limb leads, or new (or presumably new) left bundle branch block on admission electrocardiogram; (3) presentation within the first 12 h from index pain.
(1) angina of secondary etiology,
(2) recent surgery,
(3) active infection, or chronic inflammatory diseases
(4) significant hepatic or renal dysfunction, and
(5) malignancy, were not included as well as
(6) individuals with body temperature of >37.8° C at admission,
(7) coronary or cerebral event in that same period, those with complete left bundle block, those with pacemaker rhythm, and those with serious aortic valve disease, obstructive hypertrophic cardiomyopathy, and subjects who were critically ill or with ongoing or recent (< 1 month) infectious diseases
HSCRP and Lp(a) were measured by TIA on Hitachi-911 with kits manufactured by BIOKIT Spain a Standardized Assay.
<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>NSTEMI</th>
<th>STEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N</strong></td>
<td>29</td>
<td>28</td>
<td>32</td>
</tr>
<tr>
<td><strong>Gender M/F</strong></td>
<td>18/11</td>
<td>21/7</td>
<td>22/10</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>54.62 ± 10.60</td>
<td>59.22 ± 13.12</td>
<td>55.57 ± 11.44</td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td>26.12 ± 6.08</td>
<td>25.24 ± 7.44</td>
<td>29.23 ± 4.73**</td>
</tr>
<tr>
<td><strong>BP Systolic</strong></td>
<td>129.93 ± 19.07</td>
<td>136.37 ± 23.68*</td>
<td>130.41 ± 16.88</td>
</tr>
<tr>
<td><strong>BP Diastolic</strong></td>
<td>75.83 ± 12.26</td>
<td>79.56 ± 18.32</td>
<td>76.72 ± 11.84</td>
</tr>
</tbody>
</table>

Data is expressed as Mean ± SD

*p<0.05 versus STEMI & Control

**p<0.01 versus NSTEMI & Control
Table I: Lipid and Lp(a) Profile ACS patients with STEMI compared to NSTEMI.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>STEMI</th>
<th>NSTEMI</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC mmol/L</td>
<td>4.38 ± 0.50</td>
<td>4.49 ± 1.66</td>
<td>4.22 ± 1.37</td>
</tr>
<tr>
<td>TG mmol/L</td>
<td>1.11 ± 0.49</td>
<td>2.02 ± 1.62</td>
<td>1.77 ± 0.84</td>
</tr>
<tr>
<td>LDL mmol/L</td>
<td>2.71 ± 0.53</td>
<td>2.72 ± 1.31</td>
<td>2.69 ± 1.03</td>
</tr>
<tr>
<td>HDL mmol/L</td>
<td>1.07 ± 0.32</td>
<td>0.69 ± 0.30</td>
<td>0.70 ± 0.20</td>
</tr>
<tr>
<td>Lp(a) mg/dl</td>
<td>14.57 ± 11.81++</td>
<td>31.92 ± 37.34</td>
<td>22.05 ± 18.66</td>
</tr>
</tbody>
</table>

Systolic blood pressure (SBP), Diastolic Blood pressure (DBP), Total cholesterol (TC), Triglycerides (TG), Low density Lipoprotein (LDL) and High density lipoprotein (HDL) and Lipoprotein(a) [Lp(a)]. Differences were studied by Kruskal–Wallis -test for Lp(a) and ANOVA for other parameters.

## p<0.01 versus NSTEMI & STEMI
**Table I: Lipid and Lp(a) Profile ACS patients with STEMI compared to NSTEMI.**

<table>
<thead>
<tr>
<th>Cardiac Enzymes</th>
<th>NSTEMI</th>
<th>STEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin T</td>
<td>0.95 ± 1.51</td>
<td>2.61 ± 2.76</td>
</tr>
<tr>
<td>CKMB</td>
<td>111.75 ± 44.33</td>
<td>205.39 ± 152.15*</td>
</tr>
<tr>
<td>AST</td>
<td>38.00 ± 31.13</td>
<td>95.00 ± 72.66*</td>
</tr>
<tr>
<td>LDH</td>
<td>188.38 ± 92.63</td>
<td>309.00 ± 213.02</td>
</tr>
</tbody>
</table>

Creatine kinase myocardial bound (CKMB), Aspartate aminotransferase (AST), Lactate dehydrogenase (LDH)

Differences were studied by Mann–Whitney test

Data is expressed as Mean ± SD

*p<0.05 versus NSTEMI
Figure 1 - Comparison of mean CRP levels at baseline, peak and at 4-6 weeks of follow up in all ACS, NSTEMI and STEMI patients
• In our study, the difference in CRP levels was significant between STEMI and NSTEMI patients at peak levels only. This suggests that it might be influenced by the degree of early myocardial tissue necrosis. Therefore, this variation in CRP kinetics should be taken into consideration when designing future studies.

• The higher the maximum CRP recorded, the more severe the infarction suffered, the greater the likelihood of ventricular remodeling, the lower the ejection fraction, and the greater the risk of heart failure, heart rupture, and death.

• The results of the present study expand upon previous reports that demonstrated non-significant differences in CRP levels at baseline in patients with acute coronary syndromes, which tended to be higher in successive samples.

Sánchez PL. Rev Esp Cardiol. 2006; 59 (5): 441-7
Conclusions

- STEMI patients have significantly higher peak CRP levels compared to NSTEMI patients.
- These data suggest that inflammatory processes play an independent role in the pathogenesis of myocardial infarction.
- CRP assessment may assist in risk stratification after myocardial infarction.
THANKS