C-REACTIVE PROTEIN RESPONSE IN ANGINA PECTORIS

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ABSTRACT

C-reactive protein (CRP) response was studied in 44 patients with acute angina pectoris admitted to the coronary care unit. 71% of patients with acute myocardial infarction (MI) had positive CRP test while 73% of patients presumed to have unstable angina pectoris had negative CRP test. This test can be helpful in differentiating between acute MI and unstable angina pectoris.


INTRODUCTION

It has been demonstrated that C-reactive protein (CRP) appears in the serum of the patients with acute injury disorders.1 Among these, myocardial infarction (MI) is of prime importance.

In this study CRP was detected in the serum of 44 patients admitted to the coronary care unit (CCU) because of angina pectoris, and the results were compared between the two groups of acute MI and unstable angina.

PATIENTS AND METHODS

44 patients with presumed unstable angina pectoris and/or myocardial infarction were studied at the cardiovascular unit Shariati Medical Center. Blood samples were taken in the CCU for routine clinical laboratory tests, including erythrocyte sedimentation rate (ESR), and serum enzyme concentration on admission and on three consecutive days. Agglutination test for CRP was performed according to the method of Siger, et al,2 on the first day of admission. In this method, one part of 1/20 diluted serum is mixed with one part of latex reagent over the slide, and after two minutes macroscopic agglutination is observed under reflected light, and according to the World Health Organization (WHO) criteria,3 21 patients were found to have definite acute MI and 23 patients who did not meet the criteria of MI were classified as unstable angina pectoris.

Our criteria for acute inflammatory disorder other than acute MI on admission were fever, evidence of organ infection and increased ESR (>10 mm in one hour)4 by the Westergren method. Thereby we excluded the above mentioned patients.

RESULTS

CRP test was positive in 15 patients with acute MI and was unresponsive in the remaining six patients. Among the patients with positive test and acute MI, 11 had anterior wall, and four had inferior or inferoposterior wall MI. CRP was +4 positive in eight patients with

Table I. CRP response in patients with acute MI and unstable angina

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>CRP (p=0.005)</th>
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<tbody>
<tr>
<td>Acute MI</td>
<td>15 (71%)</td>
<td>Positive (**)</td>
</tr>
<tr>
<td></td>
<td>6(29%)</td>
<td>Negative (**)</td>
</tr>
<tr>
<td>Unstable Angina</td>
<td>17(23%)</td>
<td>Negative (**)</td>
</tr>
<tr>
<td></td>
<td>6(27%)</td>
<td>Positive (**)</td>
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* C-reactive protein response. Sensitivity = 70%, specificity = 80%.

anterior and one patient with inferior wall MI, +3 positive in two patients with anterior and one patient with inferior wall MI, +2 positive in one patient with anterior and one patient with inferior wall MI, and +1 positive in one patient with inferoposterior wall MI. From among six patients with negative CRP test, four had anterior and 2 had inferior wall MI.

CRP test was negative in 17 and positive in six patients with unstable angina pectoris. Occasionally, patients with positive CRP had prolonged chest pain and/or criteria of old MI in their electrocardiograms. In summary, as shown in Table I, 71% of patients with acute MI had positive CRP test and 73% of patients with unstable angina pectoris had negative CRP test. Although CRP was measured qualitatively, there is no strong correlation between the degree of agglutination and extension of MI in our patients.

**DISCUSSION**

C-reactive protein is an acute phase protein which is formed by the liver. It can be detected in normal human serum, but may increase significantly during diseases associated with active inflammation or tissue injury. Myocardial infarction is one of the settings which stimulates formation of CRP in the liver and elevates its concentration in the serum early after onset of angina pectoris. In our study CRP was reactive in the serum of 15 patients with acute MI, while in 17 patients presumed to have unstable angina pectoris CRP was non-reactive. Sluggish hepatic response in formation of CRP in some patients may explain why six patients with acute MI had negative CRP tests. CRP was positive in six patients with unstable angina pectoris. It may be that, these latter patients actually had mild myocardial necrosis, although this could not be confirmed by the clinical criteria employed.

In conclusion, we found CRP a valuable and simple test in early detection of patients with acute MI and distinguishing them from those with unstable angina pectoris. These findings were consistent with the previous studies concerning the role of CRP during acute MI.

**REFERENCES**