Study on persistent bacterial and viral infections as risk factors for myocardial infarction

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ABSTRACT

Hypertension, smoking, and hyperlipidemia are main causes of Myocardial Infarction. In recent years other factors such as chronic and persistent bacterial and viral infections are considered as causes of chronic inflammatory reaction in blood vessels leading to atherosclerosis. One hundred sera of patients with Myocardial Infarction and 76 sera of controls is examined by commercial ELISA kits to measure IgG to cytomegalovirus, Chlamydia pneumonia, Mycoplasma pneumonia, and Helicobacter pylori. C-reactive protein, highly sensitive-C-Reactive Protein, and ASO are also measured by commercial kits. Seropositivity to cytomegalovirus, Chlamydia pneumonia, Mycoplasma pneumonia, and Helicobacter pylori showed significant relationship to occurrence of myocardial infarction but not for Mycoplasma pneumonia. C-reactive protein and highly sensitive-C-Reactive Protein also showed significant relationship with myocardial infarction but not ASO. Some chronic and persistent infections other than common risk factors of Myocardial Infarction can induce inflammatory changes leading to atherosclerosis in coronary arteries and promote occurrence of Myocardial Infarction.

Key Words: Myocardial Infarction, Persistent Infections, Inflammatory response

INTRODUCTION

Myocardial Infarction (MI) is an important health problem worldwide and is a common cause of morbidity and mortality especially in elderly and middle aged individuals living in urban areas of developed countries[1]. Complete or incomplete obstruction of coronary arteries by blood clots, vascular spasm, and atherosclerotic plaque of inflamed vessels are main causes of MI [1-3]. Disorders in lipids metabolism, changes in concentration of lipoproteins such as cholesterol and leptin, genetic factors, diabetes, hypertony, obesity, stress, and hypertension are common known pathologic causes of MI [1]. In recent years, it has been proposed that chronic inflammatory response in blood vessels is a cause of atherosclerosis, hence predisposing factor for MI [3-7]. Several studies showed a causative role for persistent bacterial and viral infections in inducing long term inflammatory reactions in vascular endothelium leading to atherosclerosis [7-10].

Our investigation is a descriptive retrospective study for detecting indexes of persistent bacterial and viral infections (cytomegalovirus, C. pneumonia, M. pneumonia, and H. pylori) which are regarded as most microbial causes of chronic inflammatory changes in vascular endothelium that can promote atherosclerosis in blood vessels. We also investigated two indexes of inflammatory response (CRP & hsCRP) in patients with MI.
MATERIALS AND METHODS

One hundred Patients with history of heart attack admitted to Heshmat cardiovascular diseases hospital, Rasht, Iran, who have had MI according to clinical presentation, ECG, and echocardiography, are included in this study. Patients' demographic data are recorded by asking from patients attendants and filling the questionnaire. First day sera of patients were stored for the study. Seventy six healthy individuals, who were referred to Pars clinical diagnostic laboratory, Rasht, Iran, for periodic routine checkup, were selected as control group. Experimental and control groups were matched according to sex, age, jobs.

IgG for cytomegalovirus, Chlamydia pneumonia, Mycoplasma pneumonia, and Helicobacter pylori were measured in all sera using commercial ELISA kits (Euroimmune, Germany). In addition, C - reactive protein (CRP), highly sensitive- C-Reactive Protein (hsCRP), and Antistreptolysin O (ASO) are also measured using commercial kits (Hunman, Germany). Data were analyzed by Qui Square statistical test.

RESULTS AND DISCUSSION

Nineteen percentage of patients were younger than 50 years old, 25% were between 51-60 years old and 56% were older than 60. About 55% of cases were living in urban areas and 45.0% were villager. Seventeen percentage of patients were businessman, 6.0% were driver, 18.0% were farmer, 4.0% were worker, 25.0% were housekeeper, 5.0% were employee, and 25.0% were unemployed. Six percentage of patients were seropositive for ASO, 62.0% were seropositive for CRP and 100.0% were seroposive for hsCRP. Frequency of all studied parameters are depicted in Table 1.

Table 1: Frequency of seropositivity rates for measured indexes in two groups

<table>
<thead>
<tr>
<th>Cases indexes</th>
<th>IgG to (%)</th>
<th>CRP (%)</th>
<th>hsCRP (%)</th>
<th>ASO (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>cytomegalovirus</td>
<td>C. pneumonia</td>
<td>M. pneumonia</td>
<td>H. pylori</td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>82.0</td>
<td>93.7</td>
<td>49.2</td>
<td>85.5</td>
</tr>
<tr>
<td>control</td>
<td>71.6</td>
<td>86.4</td>
<td>52.3</td>
<td>73.1</td>
</tr>
</tbody>
</table>

Mean titer of hsCRP in patients were 23.2 mg/L and in control group was 6.3 mg/L. There was significant relationship between occurrence of MI and seropositivity to cytomegalovirus (p=0.004), H.pylori (p=0.039), and C. pneumonia (p=0.007) but not with M. pneumonia (p>0.05). Serpositivity to CRP and hsCRP showed also significant relationship with MI occurrence but not with ASO (Table 2). Other data for nonmicrobial studied parameters is showed in Tabal 3.

Table 2: Statistical relationship of studied Microbial and inflammatory parameters with occurrence of MI

<table>
<thead>
<tr>
<th>studied parameters</th>
<th>IgG to (%)</th>
<th>CRP (%)</th>
<th>hsCRP (%)</th>
<th>ASO (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>cytomegalovirus</td>
<td>C. pneumonia</td>
<td>M. pneumonia</td>
<td>H. pylori</td>
<td></td>
</tr>
<tr>
<td>P value</td>
<td>0.004</td>
<td>0.007</td>
<td>0.094</td>
<td>0.039</td>
</tr>
</tbody>
</table>

Table 3: Statistical relationship of studied no microbial parameters with occurrence of MI comparing two groups

<table>
<thead>
<tr>
<th>parameters</th>
<th>smoking</th>
<th>Age (over 60)</th>
<th>Gender(male)</th>
<th>Chronic heart disease</th>
<th>Living in urban area</th>
<th>occupation</th>
<th>hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>P Valu</td>
<td>0.001</td>
<td>0.002</td>
<td>0.001</td>
<td>0.001</td>
<td>0.000</td>
<td>0.450</td>
<td>0.029</td>
</tr>
</tbody>
</table>

In recent years several investigators have studied the role of inflammation in promotion of atherosclerosis process [3-5,11,12]. Several studies showed that inflammation is an important inducer factor for atherosclerosis [6,7,11]. Although prmotive factors of inflammatory changes in vascular tissue are not well known [3-7,12]. Results of our study are in consistence with study of Danesh [6] and Roivainen [7] showed that main inflammatory indexes increase in first day of MI. Infectious agents might be important inducers of pathologic inflammatory events in blood [7-10]. It is well known that groups of people show variable reaction to infections. It may cause aterogenic complications [8, 9]. Some researcher showed that persistent infections are inducer of long term inflammation in blood vessels and a well known cause of atherosclerosis [8-10].

Our study showed significant relationship between occurrence of MI with persistent infection of cytomegalovirus. This virus can affect endothelium of blood vessels and causes functional disorders promoting atherosclerosis [13]. Several epidemiological studies have suggested a role for persistent cytomegalovirus infection for promotion of atherosclerosis [13,14] but Adler and coworker(1998) did not find this relationship [15]. Rothemucher and coworker (1999) reported that risk factor of coronary disease for patients with previous cytomegalovirus infection is two times more than no infected patients [16].

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We also found significant relationship between occurrences of MI with prior history of C.pneumonia infection. The bacterium is an initially respiratory pathogen that is able to cause other diseases such as asthma, arthritis, and atherosclerosis. Saiku (1988) and Danes (2000) reported an inducing and promoting role for C.pneumonia in atherosclerosis [17,18]. Kahler (2001) showed its role in ischemic heart disease [19] and Liuba (2003) reported that it is an inducer for aortic valve disease [20] but this role is not confirmed in other studies. Nobel (1999) and Altmann (1999) did not find significant role for C.pneumonia in acute coronary diseases [21,22]. These discrepancies might be due to criteria for selecting patients or methodological differences.

Role of prior M.pneumonia infection in incidence of MI is investigated by many researchers. In most of cases, common role of M.pneumonia and C.pneumonia is studied and showed positive relationship [23-27], we did not find such relationship but our study showed significant relationship between persistent infection of H.pylori and MI that is in consistent of similar studies [28-31].

In recent years, several researchers are investigating the role of inflammation in pectoralis angina and acute myocardial infarction by measuring some important inflammatory indexes such as CRP and IL-6 [11,12,32-34]. Several studies have shown increased level of CRP in patients with pectoralis angina, and in 20-50 percent of patients with MI during first 6 hours of the attack [35-37]. We also found increased seropositivity to CRP and hsCRP and increased hsCRP titer in patients with MI. As, other studies have shown, these two inflammatory indexes can be considered as reliable diagnostic marker for MI [38-40]. In our study, ASO did not show significant relationship with the frequency of MI occurrence indicating that prior streptococcal infection is not a predisposing factor for MI.

CONCLUSION

We found that hsCRP is more sensitive than CRP for diagnosis and follow up management of MI.

Acknowledgment

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REFERENCES