Serum Selenium Levels in Cardiovascular Diseases
Kalp-Damar Hastalıklarında Serum Selenyum Düzeýleri

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SUMMARY

Various epidemiological and clinical researches have been carried out in order to find out the relationship between selenium deficiency and other diseases, mainly cardiovascular diseases and certain types of cancer. In this study, we have aimed at determining serum selenium levels of the patients with acute myocardial infarction (A.M.I.), coronary artery disease (C.A.D), and the healthy control group, and investigating whether there are any significant differences between the serum selenium levels of the healthy control group and the patients with A.M.I, and C.A.D. Mean serum selenium levels were found to be 38.59 ± 15.25 \( \mu g / L \) in the patients (n=22) with acute myocardial infarction, 37.20 ± 11.44 \( \mu g / L \) in the patients (n=27) with coronary artery disease, and 63.66 ± 11.71 \( \mu g / L \) in the healthy control group (n = 21). There was no significant difference between mean serum selenium levels of the patients with A.M.I, and C.A.D (p>0.05), but significant differences have been found between the mean serum selenium levels of the healthy control group and the other two groups mentioned above. (p<0.01)

ÖZET

Selenyum eksikliği ile başta kardiovasküler hastalıklar ve kanser olmak üzere çeşitli hastalıklar arasındaki iliþkiiyi ortaya koyan çeşitli epidemiyolojik ve klinik çalışmalar bulunmaktadır.

Bu çalışmada akut miyokard infarktüslü ve koroner arter hastalığı olan hastalarda serum selenyum düzeylerinin tayini ve sağlıklı kont-
Serum Selenium Levels in Cardiovascular Diseases

Since the discovery of selenium as an essential element for mammals (1) and its role in glutatione peroxidase activity (2), there has been an increased interest in this element. Initially interest in selenium was caused by its potential toxicity(3), but subsequently this has been changed because of some significant observations. First of all, selenium acts as an anticancer agent in chemically or virally induced tumor formation in experimental animals. (4, 5) Several experiments carried out with fish and mammals revealed another beneficial effect of this element in acting as an antagonist against various toxic metals like, arsenic, cadmium, copper, lead, inorganic mercury and methylmercury compounds. (6-11) Finally, several epidemiological, clinical and experimental studies support the concept that selenium and other antioxidants have roles in the development of human cardiovascular diseases. (12-20)

Selenium deficiency has been shown to be associated with cardiomyopathy. (21-23) Keshan disease, an endemic cardiomyopathy, was the first human disease related to selenium deficiency which occurred in China in the regions where dietary selenium intake was very low. (24, 25).

Selenium is part of the enzyme glutatione peroxidase that protects the tissue from lipid peroxidation. (26) It was found out that this enzyme was effective in all tissues and in the vascular system.(27) Selenium mainly provides a means of defence against the build-up of lipid peroxides and free radicals that damage all membranes and macromolecules including deoxyriboonucleic acid (DNA). It is claimed that cardioprotective effect of selenium may be caused by its function in glutatione peroxidase. (20)
In this study, we have aimed at determining the serum selenium levels of the patients with acute myocardial infarction (A.M.I), coronary artery disease (C.A.D) and the healthy control group and investigating whether there are any significant differences between the serum selenium levels of the healthy control group and those of the patients with A.M.I and C.A.D.

EXPERIMENTAL

Subjects

Serum samples were obtained from the patients admitted to the Cardiology Department of Yüksek İhtisas Hospital. Serum selenium levels were measured in twenty-two patients (4 females aged 50 to 78 years and 18 males aged 41 to 91 years) with acute myocardial infarction verified by typical chest pain and electrocardiographic changes or by the appearance of a Q wave on electrocardiogram and in twenty-seven patients (7 females aged 49 to 64 years and 20 males aged 33 to 63 years) with coronary artery disease verified by coronary angiography. Twenty-one healthy subjects matched with age and sex were served as the control group. None of the healthy subjects mentioned above had been inpatient or outpatient. The serum samples from the control and patient groups were taken concurrently and analysed in the same laboratory.

Method

Whole blood samples were taken from the superficial arm vein with a great care in order to avoid contamination. Each blood sample was kept in an acid cleaned plastic tube. Serums were obtained by centrifugation at 3,000 rpm for 10 min and kept -20°C until their analysis. The serum samples were digested by a mixture of nitric, sulphuric and perchloric acids (2:1:0.4). In order to digestion, a furnace with a temperature controller was developed. After hydride generation using a sodium borohydride method(28), all samples were analysed. A Varian Model Spectra AA 30 / 40 atomic absorption spectrometer equipped with a Varian VGA-76 vapor generation accessory was used.

By use of statistical techniques, mean values and standard deviations were calculated. The mean values for the patients and the control group were compared by Student's t test.


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RESULTS

The results obtained from this study are summarized in Table I and Table II.

Table 1. The mean serum selenium levels in the patients with clinical diagnosis of acute myocardial infarction (A.M.I), coronary artery disease (C.A.D) and the healthy control group.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Number of Subjects</th>
<th>Serum Selenium Levels (µg/ L) Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with A.M.I.</td>
<td>22</td>
<td>38.59a,b ± 15.25</td>
<td>18.50-56.22</td>
</tr>
<tr>
<td>Patients with C.A.D.</td>
<td>27</td>
<td>37.20a ± 11.44</td>
<td>17.20-78.84</td>
</tr>
<tr>
<td>Healthy Control Group</td>
<td>21</td>
<td>63.66 ± 11.71</td>
<td>36.40-87.40</td>
</tr>
</tbody>
</table>

a) Difference from the mean serum selenium levels of healthy control group (p <0.01)-Student's t-test.

b) Difference from the mean serum selenium levels of patients with C.A.D (p>0.05)- Student's t-test.

Table II. Distribution of serum selenium levels of the patients with clinical diagnosis of coronary artery diseases and the healthy control group according to sex and age.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Number of Subjects</th>
<th>Serum Selenium Levels (µg/ L) Mean ± SD</th>
<th>Range</th>
<th>p Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female;</td>
<td>11</td>
<td>36.27±7.71</td>
<td>22.20—48.24</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Male</td>
<td>38</td>
<td>37.55±13.76</td>
<td>17.20—78.84</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Age &lt;60</td>
<td>33</td>
<td>38.62±14.23</td>
<td>17.20—78.84</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>&gt;60 Healthy Female</td>
<td>16</td>
<td>34.00±8.26</td>
<td>23.20—56.22</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Male</td>
<td>15</td>
<td>62.08±11.56</td>
<td>36.40—86.00</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Age &lt;60</td>
<td>9</td>
<td>63.21±12.40</td>
<td>52.10—79.00</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>&gt;60</td>
<td>12</td>
<td>61.78±13.29</td>
<td>55.00—89.00</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>
The mean serum, selenium levels were found to be 38.59 ± 15.25 μg/ L in the patients with A.M.I 37.20 ± 11.44 μg/ L in the patients with C.A.D and 63.66 ± 11.71 μg/ L in the healthy control group. There was no significant difference between the mean serum selenium levels of the patients with A.M.I and those with C.A.D. (p>0.05) However, serum selenium concentrations in the patients with A.M.I, and C.A.D were found to be significantly lower than the healthy control group. (p<0.01)

The mean serum selenium, levels of the female and male patients with clinical diagnosis of coronary artery disease were found to be 36.27 ± 7.71 μg/ L and 37.55 ± 13.76 μg/ L respectively. The mean serum selenium levels with coronary artery disease were found to be 34.00 ± 8.26 μg/ L for the. ages over 60 years and 38.62 ± 14.23 μg/ L for the ages below 60 years Ni significant differences were found in the serum selenium levels when compared on the basis of age and sex. (p>0.05)

DISCUSSION

A number of factors are found to be associated with increased risk for cardiovascular diseases (C.V.D). Among these factors the trace element selenium is suggested to be associated with C.V.D. Selenium is only a contributory secondary cause. Its deficiency affects several cellular mechanisms that have been implicated in the pathogenesis of atherosclerotic vascular disease. (12, 14, 16, 18, 20) Selenium depletion is accompanied by a decrease in the activity of glutathione peroxidase, a selenium-containing enzyme present in several tissues, including platelets and arterial walls. This enzyme has important functions in the removal of hydrogen peroxide and organic hydroperoxides possibly protecting the coronary epitelium from oxidative damage. (26, 27)

Reportedly, patients with coronary atherosclerosis, myocardial infarction or cardiomyopathy have a significant lower selenium concentration in their serum than do healthy control groups. (15, 22, 23, 25, 27) in a prospective epidemiological study from Finland it was found that serum selenium levels below 45 μg / L was associated with an increased risk of coronary heart disease (15). Another Finnish study found no correlation between serum selenium and development of clinical manifestations of coronary heart disease (17) and a British
study claim that there is no correlation between glutathione peroxide activities and the risk factors for coronary heart disease. (19)

The results of our study showed that serum selenium concentrations were significantly lower in the patients with A.M.I and C.A.D than in the healthy control group. No significant differences were found in the serum selenium levels when compared on the basis of age and sex. Therefore, we presented the results on a single table Table II. The serum selenium levels in the patients with A.M.I and C.A.D observed in the present study are in conformity with the observations made by other study groups. (13-16, 27)

We believe that this study is one of the first studies associating cardiovascular disease to selenium deficiency in Turkey. According to published literature, the evidence concerning the relationship between the serum selenium concentration and the risk of C.V.D is inconclusive. New epidemiological studies on the role of selenium in C.V.D are needed to confirm or negate the previous findings. In order to better understand the mechanisms through which selenium deficiency could increase the risk of C.V.D further investigation should be conducted on the experimental animals. These studies should take into consideration confounding with other risk factors.

ACKNOWLEDGEMENTS

This study was partly supported by a grant from Ankara University Research Foundation (Project No: 90-300019)

REFERENCES


