Original article:

“Oxidants & Antioxidants in Coronary Heart Disease.”

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ABSTRACT:

Background: Coronary heart disease (CHD) is the number one killer in the world. Thus heart disease is the cause of the total mortality. Recently adequate evidence have been accumulated which point out the central crucial role played by free radical process known as lipid peroxidation in pathogenesis of atherosclerosis which increase with advancing age and is reported to be involved in cardiac damage. An imbalanced oxidants and antioxidants can increase the risk of coronary heart disease.

Material and Methods: In this study 100 subjects were included above 20 years in age. 50 clinically diagnosed CHD patients and 50 normal healthy subject were recruited as control. Serum lipid peroxide were measured by ‘Kei Satoch ’ method, serum Total and HDL cholesterol were measured by CHOD-POD method and serum VLDL cholesterol was measured by ‘Precipitation’ method, while serum vitamin E was measured by ‘Baker and Frank method.

Statistical Analysis: Data were analysed using ‘t’ test for their level of significance

Result: The level of lipid peroxide and LDL cholesterol in CHD were significantly increased than the controls. And the level of vitamin E was significantly lower in CHD patients as compared to control groups.

Conclusion: High levels of oxidants and low levels of antioxidants might be contributing to the increased susceptibility for the development of CHD.

Key Words: Lipid peroxide, LDL cholesterol, Vitamin E, CHD.

INTRODUCTION:

Coronary heart disease(CHD) is a leading cause of morbidity & mortality in developed countries & is emerging as an epidemic in developing countries. (1) It is predicted that there will be an increase of 111% in cardiovascular deaths in India by the year 2020 when compared to the year 1990. (2) This is much higher than that predicted to any other region both in Asia as well as outside Asia. In India, the prevalence of CHD is much higher in South when compared to North India.(3,4) This high prevalence warrants probing into the presence of various risk factors & their association with CHD. Traditional risk factors like serum cholesterol, blood pressure & smoking account for not more than 50% difference in mortality of CHD (5).

Oxidized LDL cholesterol has been identified in atherosclerotic lesions.(6) This has prompted the study of antioxidants in the prevention of the ignition & progression of cardiovascular disease,(7) Case-control studies (8,9,10) have shown low levels of serum antioxidants in CHD patients when compared to controls & suggested the role of vitamin E in prevention of CHD.

MATERIAL AND METHODS:

The present study was carried out in the Department of Biochemistry ,Dr. V. M. Medical college, Solapur. The patient selected for the present study were I P D
from Department of Medicine, Shree Chatrapati Shivaji Maharaj General Hospital Solapur. The diagnosis of the patients was done by senior physicians on the basis of detailed clinical history, clinical examination, electrocardiographically & relevant biochemical investigations such as SGOT, CPK etc.

In the study total number of 100 subject were included. The distribution of the above subjects is done as follows.

- Control group: 50 subjects.
- Coronary heart disease: 50 cases.

All above subjects were of both sexes in the age group of 20-80 years. The control subjects selected were healthy & showed no abnormality on clinical examinations.

Collection of blood samples:
Total 5 ml of venous blood sample was collected in the morning after an overnight fast. The blood sample was collected under aseptic precautions in dry centrifuge tubes. After retraction of clot, samples were centrifuged at 3000 rpm for ten minutes within two hours of collection. The serum was separated into others stoppered clean glass bulbs & following parameters were estimated.

- Serum Lipid peroxide by ‘Kei Sathoh’ method. (11)
- Serum LDL cholesterol.
- Serum Vitamin E by ’Baker & Frank’ method. (14)

Statistical Analysis: All statistical analyses were performed using Minitab Software. The data of patients and controls was analysed by students ‘t’ test. P values < 0.001 were considered significant.

TABLE No. 1: Shows serum Lipid peroxide levels in control subjects of different age groups and in CHD patients.

<table>
<thead>
<tr>
<th>Sr. No</th>
<th>Age Group</th>
<th>n</th>
<th>Control n mol/ml mean ± S.D.</th>
<th>CHD n mol/ml mean ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20-35</td>
<td>10</td>
<td>2.06 ± 0.19</td>
<td>2.92 ± 0.34**</td>
</tr>
<tr>
<td>2</td>
<td>36-50</td>
<td>10</td>
<td>2.10 ± 0.30</td>
<td>3.50 ± 0.32**</td>
</tr>
<tr>
<td>3</td>
<td>51-65</td>
<td>15</td>
<td>2.85 ± 0.35</td>
<td>3.97 ± 0.23**</td>
</tr>
<tr>
<td>4</td>
<td>66-80</td>
<td>15</td>
<td>3.06 ± 0.64</td>
<td>4.92 ± 0.32**</td>
</tr>
</tbody>
</table>

** p<0.001, n - Number of cases
TABLE No.2: Shows serum LDL cholesterol level in control subjects of different age groups and in CHD patients.

<table>
<thead>
<tr>
<th>Sr. No</th>
<th>Age Group</th>
<th>n</th>
<th>Control n mol/ml mean ± S.D.</th>
<th>CHD n mol/ml mean ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20-35</td>
<td>10</td>
<td>102.8 ± 15.3</td>
<td>130.6 ± 5.28**</td>
</tr>
<tr>
<td>2</td>
<td>36-50</td>
<td>10</td>
<td>106 ± 7.3</td>
<td>135 ± 4.8**</td>
</tr>
<tr>
<td>3</td>
<td>51-65</td>
<td>15</td>
<td>112 ± 8.3</td>
<td>145 ± 6.3**</td>
</tr>
<tr>
<td>4</td>
<td>66-80</td>
<td>15</td>
<td>116 ± 5.7</td>
<td>148 ± 4.5**</td>
</tr>
</tbody>
</table>

** p<0.001, n - Number of cases

TABLE No.3: Shows serum vitamin E level in control subjects of different age groups and in CHD patients.

<table>
<thead>
<tr>
<th>Sr. No</th>
<th>Age Group</th>
<th>n</th>
<th>Control n mol/ml mean ± S.D.</th>
<th>CHD n mol/ml mean ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20-35</td>
<td>10</td>
<td>15.33 ± 0.38</td>
<td>7.9 ± 0.28**</td>
</tr>
<tr>
<td>2</td>
<td>36-50</td>
<td>10</td>
<td>14.15 ± 0.10</td>
<td>7.1 ± 0.42**</td>
</tr>
<tr>
<td>3</td>
<td>51-65</td>
<td>15</td>
<td>13.09 ± 0.15</td>
<td>6.0 ± 0.16**</td>
</tr>
<tr>
<td>4</td>
<td>66-80</td>
<td>15</td>
<td>12.0 ± 0.16</td>
<td>5.0 ± 0.19**</td>
</tr>
</tbody>
</table>

** p<0.001, n - Number of cases
DISCUSSION: Table 1 shows serum lipid peroxide levels in control subjects of different age groups and in CHD patients. Table 2 shows serum LDL cholesterol levels in control subjects of different age groups and in CHD patients. Table 3 shows serum vitamin E levels in control subjects of different age groups and in CHD patients. Total cholesterol, VLDL cholesterol, LDL cholesterol were significantly higher (p<0.001) & HDL cholesterol (p<0.002) levels were significantly lower in patients when compared to controls. The serum lipid peroxide & LDL cholesterol were significantly higher in the patients than the control (p<0.001). The level of vitamin E was significantly lower (p<0.001) in CHD than the control subjects. An important implication of the oxidative modification hypothesis of atherosclerosis is that antioxidants may inhibit atherogenesis, through mechanisms like protection of LDL against oxidative modification. Antioxidant enzymes are present in small amounts only in plasma and other external fluids. Besides these, antioxidants like Vitamin E are of major importance (15). Increased prevalence of coronary heart disease necessitated the intervention that can reduce the risk of CHD. Hence, correspondingly greater attention is being focused on prevention of CHD. In this area antioxidants may serve an important role (15).

First the development of atheroma is partly dependent on incorporation of oxidized cholesterol into monocytes and macrophages within the arterial wall. Secondly platelet adhesion is so important in thrombosis and it is more likely when the antioxidant status is low. Thirdly the arterial endothelium is protected against damage in primates by dietary vitamin E and fourthly unopposed free radical attack within the ischemic myocardium can impair its function and repair. Hypercholesterolemia, in particular high levels of LDL is a well established risk factor for development of CHD. The oxidative modification of LDL is a key process because these modifications render LDL particles more atherogenic, specifically in contrast to native LDL. The oxidized LDL may include vascular inflammation & even gives rise to autoimmune reactions in the vascular wall, all of which have been implicated in atherogenesis (16).

Reduced levels of vitamin E may also be due to the increased requirement of vitamin E in pro-oxidant milieu with enhanced free radical status, leading to the increased lipid peroxidation, a resultant depletion of free radical scavenger and antioxidant reserves of the body.(17) Esterbauer et al (18) have reported that endogenous antioxidants, mainly tocopherol, contained in LDL particles are rapidly consumed after induced oxidation and propagation of the oxidative process doesn't begin until antioxidant molecules are largely exhausted. Low levels of vitamin E was associated with increased risk of CHD.(19) Significant inverse association of vitamin E and CHD was observed in studies conducted in India and abroad (9,20). In our study also we have observed significant inverse relation of serum vitamin E with CHD.

CONCLUSION:
Increased oxidants & decreased antioxidants status positively correlates with severity of coronary heart disease. Therefore determination of levels of serum oxidants & antioxidants may be used as an index for the assessment of coronary heart disease, as well as a preventive measure. Increasing the antioxidant status and lowering the oxidant levels by the therapeutic measures may also contribute for preventing the cardiovascular disease.
REFERENCES