High density lipoprotein cholesterol (HDL-C) analysis in leprosy patients

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Summary A study was undertaken with an aim to find out the levels of circulating high density lipoprotein (HDL) cholesterol and ratio of this fraction to total blood cholesterol in lepromatous leprosy patients before and after drug therapy. The healthy contacts of these patients were considered as control subjects of the study. The subjects were distributed in three groups on the basis of their age. HDL-cholesterol to total cholesterol ratio was significantly raised in both treated and untreated patients in all three groups compared to the healthy controls. The data may explain the low risk of myocardial infarction due to atherosclerosis in leprosy patients.

Introduction

A strong inverse relation exists between plasma levels of high density lipoprotein (HDL) and mortality from coronary heart disease. The role of HDL as a risk-preventing factor and the importance of this cholesterol-carrying protein in patients who are likely to develop risk of coronary heart disease have been of great importance to both clinicians and biochemists. High levels of serum HDL-C are associated with a low incidence of the complications of atherosclerosis, whereas low levels are associated with an increased incidence. Various factors are known to influence the plasma levels of HDL-C.7-13 There seems to be a direct correlation between the low-density lipoprotein cholesterol (LDL-C) and coronary heart disease (CHD) but it is said to be less consistent compared to inverse correlation existing between HDL-C and CHD.4 There is definitive alteration in the lipid metabolism of leprosy patients as has been shown by many workers.14-21 Atherosclerosis and CHD appear to be very uncommon in leprosy patients.26-28 Although there have been many reports regarding general serum
lipid status of leprosy patients, no one has so far attempted to find out the HDL-C which can offer a better explanation of the hitherto unexplained difference in CHD prevalence and incident rates in leprosy patients. The present study is the continuation of our earlier work.\textsuperscript{20}

**Materials and methods**

Ninety-three cases were included in this study. All were male and none of them had any previous clinical record of cardiovascular involvement. Factors known to affect lipid metabolism like alcohol, smoking, diet, etc., were carefully taken into account when selecting the cases. They were clinically diagnosed and divided into three groups. Group I. Twenty-five cases of untreated lepromatous leprosy, Group II had 46 cases of treated lepromatous leprosy, and Group III consisted of 22 cases of healthy contacts of lepromatous leprosy. All of them belonged to the same socio-economic status. Each group was subdivided into three age groups: (a) 30–39 years, (b) 40–49 years, and (c) 50–60 years. Treatment included administration of dapsone, clofazimine or rifampicin but none of the patients were given steroids or any other drug known to affect lipid metabolism.

Fasting blood samples were collected and fresh sera were used for analysis. Separation of HDL fraction and estimation of cholesterol in that fraction were carried out according to the method of Vikari.\textsuperscript{22} Twelve per cent (w/v) PEG-6000 was used as a precipitating agent. But instead of adding equal volume of the precipitating agent to the serum, double the volume was added, i.e. to one part of serum two parts of 12\% PEG-6000 were added in order to get maximum precipitation of LDL-fraction. Total cholesterol was estimated by the method of Zak \textit{et al.}\textsuperscript{23}

**Results and discussion**

It was found that the HDL-C was significantly higher in group Ic compared to control group III (\(P<0.05\)), even though all other age groups tended to show a marginal increase in values. After treatment, again the 50–60 years group (IIc) presented significantly higher value of HDL-C (\(P<0.05\)).

After treatment, in group IIc there was an increase in HDL-C compared to that of group IIIc but there was not any marked change from the untreated patients of the same age group. The statistical analysis and comparison of groups I and II show that no importance could be attributed to the effect of therapy per se.

The values of the ratio of HDL-C to total cholesterol were significantly higher in all subgroups of groups I and II compared to the corresponding results of control group III. The results of the present study are given in Table I.
Table 1. High density lipoprotein cholesterol analysis in leprosy patients and healthy controls

<table>
<thead>
<tr>
<th>Clinical condition</th>
<th>Age group (all males)</th>
<th>Serum total cholesterol (TC) mg% †</th>
<th>HDL-cholesterol (HDL-C) mg% †</th>
<th>HDL-C/TC</th>
</tr>
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<tr>
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<tr>
<td>1 Untreated lepromatous</td>
<td>(a) 30–39 (14)</td>
<td>113 ± 22.8</td>
<td>29.48 ± 10.59*</td>
<td>0.258 ± 0.061 (P &lt; 0.05)</td>
</tr>
<tr>
<td>leprosy</td>
<td>(b) 40–49 (7)</td>
<td>120 ± 18.6</td>
<td>24.11 ± 4.87*</td>
<td>0.203 ± 0.007 (P &lt; 0.05)</td>
</tr>
<tr>
<td></td>
<td>(c) 50–60 (4)</td>
<td>138 ± 16.4</td>
<td>60.25 ± 4.92 (P &lt; 0.05)</td>
<td>0.436 ± 0.014 (P &lt; 0.02)</td>
</tr>
<tr>
<td>2 Treated lepromatous</td>
<td>(a) 30–39 (26)</td>
<td>118 ± 21.8</td>
<td>30.60 ± 7.00*</td>
<td>0.250 ± 0.014 (P &lt; 0.05)</td>
</tr>
<tr>
<td>leprosy</td>
<td>(b) 40–49 (12)</td>
<td>123 ± 22.4</td>
<td>42.75 ± 8.52 (P &lt; 0.01)</td>
<td>0.346 ± 0.006 (P &lt; 0.02)</td>
</tr>
<tr>
<td></td>
<td>(c) 50–60 (6)</td>
<td>150 ± 17.8</td>
<td>68.88 ± 6.64 (P &lt; 0.05)</td>
<td>0.440 ± 0.009 (P &lt; 0.02)</td>
</tr>
<tr>
<td>3 Healthy contacts of</td>
<td>(a) 30–39 (11)</td>
<td>140 ± 16.8</td>
<td>23.46 ± 3.38</td>
<td>0.177 ± 0.014</td>
</tr>
<tr>
<td>lepromatous leprosy</td>
<td>(b) 40–49 (4)</td>
<td>150 ± 17.5</td>
<td>20.06 ± 9.16</td>
<td>0.123 ± 0.044</td>
</tr>
<tr>
<td>(control cases)</td>
<td>(c) 50–60 (7)</td>
<td>158 ± 24.8</td>
<td>40.62 ± 3.95</td>
<td>0.255 ± 0.015</td>
</tr>
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</table>

* Statistically not significant.
† Standard deviation of the mean values. Numbers in parentheses indicate number of cases studied.

It is well known that compared to a normal population the prevalence of CHD due to atherosclerosis is much less in leprosy patients. This might be due to their favourable HDL-C/Total cholesterol ratio and higher HDL-C levels than the controls as of our study. Heart, large blood vessels and gall bladder are somehow not affected by leprosy. There are no references in the literature regarding the involvement of heart and the formation of atheroma in big vessels. Documented reports for the involvement of cardiovascular system are (a) specific endocarditis due to rapidly growing chromogenic mycobacteria, and (b) specific allergic vasculitis and phlebitis, both cited. However, peripheral arteries and veins reveal sclerosis. Two cases of definitive cardiac involvement in lepromatous leprosy have recently been presented where again the biochemical findings are not complete and that no data are presented on the cholesterol and lipoprotein levels. They speculated that leprosy might have affected the coronary vessels giving rise to CHD. Recent studies have shown that HDL is an independent risk-lowering factor. Higher levels of HDL-C may be protective against the development of atherosclerosis and hence CHD. The following explanations have been provided to explain the role of HDL-C as a protective factor.

(a) Higher concentrations of HDL-C prevent deposition of cholesterol from LDL by blocking the latter’s receptors and hence its uptake by endothelial cells.
(b) HDL provides a carrier to transport cholesterol from peripheral tissues to the liver where it is catabolized and excreted.
(c) HDL prevents the accumulation of cholesterol esters inside cells, especially those of the reticulo-endothelial system which phagocytose red blood cells and other dying cells whose membranes contain cholesterol.

So far as our understanding goes, there have been no reports on the effects of dapsone, clofazimine or rifampicin affecting the lipid metabolism in leprosy patients. Our present study has not shown any significant change in HDL-C levels after the patients have commenced taking these drugs. Even though group Ic showed higher values for HDL-C the number of cases we could investigate was only four and this very small number makes our interpretation difficult. More information can be obtained if a controlled study is carried out to find the effect of various anti-leprosy drugs on the lipid metabolism of the patient. It would be useful to carry out an indepth epidemiological study to compare the prevalence of CHD and the usefulness of HDL-C in leprosy patients.

Acknowledgment

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References

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29 Geracôte et al ???