EFFECT OF THE LIPID PROFILE DIFFERS WITH TUBERCULOSIS SUBJECT INNOVATION TO ELIMINATE – A REVIEW

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ABSTRACT

The lipid profile was strongly affected by pulmonary tuberculosis in the Serum levels of total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and triglycerides (TG). Serum cholesterol in pulmonary TB patients is lower than healthy controls, if it is a risk factor or a consequence of the disease itself. To detect the differences in lipid profile between Karaikal district, TB patients, and controls and to test and find whether there is changes after treatment or not aiming to prove if the difference is a risk factor for tuberculosis or a consequence of the disease itself. From the recorded data of a medical college laboratory, we analysed fasting serum lipid profile of serum cholesterol (Chol), triglycerides (TG), low density lipoproteins (LDL) and high density lipoproteins (HDL). Then a follow up of lipid profile was done. We confirmed from the chief of laboratory that the Samples for lipid profile from healthy controls were also drawn and recorded. We distinguished 30 new TB patients, 14 women and 16 men with a mean age (SD) of 33.4 ± 13.25 years. There were 16 pulmonary TB and 14 other forms as pleural (5), TB lymphadenitis (5), TB peritonitis (4) plus 15 controls. Regarding the whole studied group, only serum triglyceride was significantly lower before treatment than control group (P<0.01) while both serum cholesterol and HDL showed a significant increase after treatment than before it (P<0.01 for both). Regarding pulmonary tuberculosis patients, both serum cholesterol and triglycerides were significantly lower on diagnosis than healthy controls (P<0.05 for both) and only serum cholesterol increased significantly after treatment than before it (P<0.01). Conclusions and recommendations: Hypercholesterolemia in Karaikal, district patients with pulmonary TB is present at the time of diagnosis. However, it proved to be a consequence of the disease rather than a risk factor as serum cholesterol significantly increased in both pulmonary TB and in the whole group after treatment.

Keywords: Lipid Profile BMI, Tuberculosis, Infection, CHD,

Objectives: compare and assess the lipid profile with TB subjects

Introduction

There are multiple abnormalities in lipid metabolism were reported in TB infected patients. Changes in blood lipids occur naturally during the course of Tuberculosis infection, with decreases in early phase of the infection in both total cholesterol (TC) and high-density lipoprotein (HDL) cholesterol, and increases later in

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triglycerides (TGs). Infection with TB is known to increase plasma TG levels by decreasing the number of circulating lipoproteins, a process considered to be the result of reduced lipoprotein lipase or by stimulating hepatic lipid synthesis through an increase in hepatic fatty acid synthesis or an increase in re-esterification of fatty acid derived from lipolysis. However, highly active also leads to lipid changes with increases in both TGs and TC. While increases in TC during therapy may represent a return to pre-infection levels associated with an increased risk of CVD such as myocardial infarction. Few studies has studied lipid profiles of Tuberculosis subjects. But they were either of short duration or based on a small number of patients (less than 100). Therefore, present study was designed to evaluate lipid profiles.

Materials and Methods

The present study was analysed from the recorded data of Tuberculosis infected subjects attending anti-retroviral drug from VMMC Hospital, Karaikal District South India. For the purpose 35 recorded data of subjects aged from 21 years involved in the study. Biochemical and Lipid profile and other infected diseases analyses of data were recorded at the beginning of the study and after twelve months. Fasting blood glucose, serum TC, TGs, HDLs, low-density lipoproteins (LDLs), urea, and creatinine serum enzyme alanine aminotransferase (ALT) were estimated by an automated clinical chemistry auto analyzer. Blood was drawn after a 12-hour fast for the measurement of plasma lipids. Diagnosis of dyslipidemia was based on cholesterol levels 4.54 mmol/L and TG levels 1.29 mmol/L. Subjects data were computerized data base for the later statistical analysis. Out of total 189 subjects included in this study, 23 did not complete the programmed follow-up. There were four tuberculosis related deaths; the rest defaulted follow-up for reasons unknown to us. The mean age of the study subjects was 41.2 ± 7.2 males being older than females in general (P = 0.02). The majority of subjects had a heterosexual source of exposure to the virus; almost half of the participants had TB either by clinical or immunological parameters and one out of every ten subjects was overweight (BMI, 27 kg/m²).

BMI was calculated as follows.

\[
\text{BMI} = \frac{\text{weight in kilograms}}{\text{Height in meters}^2}
\]

.BMI is usually expressed in kilograms per square meter, resulting when weight is measured in kilograms and height in meters.

Statistical Analysis

Statistical analysis was done using suitable statistical tool. Data was estimated on excel sheet and analysed statistically. Quantitative data was summarized in the form of MEAN ± SD and differences in mean of both the groups were analyzed using Student’s unpaired t-test. The P value <.05 was taken as significant. Association was found by Pearson’s Correlation.

Results

The mean percentage changes of total TG, TC, HDL and LDL between baseline (month 0) and month X after start of treatment were determined for each individual patient using the formula by Van Leth and colleagues.
Increase ($\%) = \frac{C_X - C_0}{C_0} \times 100$

Where, $C_X$ = Concentration at month X (X is the study end point at which follow up takes place - 12 months) after the start of treatment; $C_0$ = Concentration at month 0 (baseline or month 0). A body mass index (BMI) above 25 kg/m$^2$ was considered to be high.

Clinical Parameters of the Subjects: Out of total 229 subjects included in this study, 17 did not complete the programmed follow-up. There were four TUBERCULOSIS-related deaths; the rest defaulted follow-up for reasons unknown to us. The mean age of the study population was 46.3 ± 9.3, males being older than females in general ($P = 0.03$). The majority of subjects had a heterosexual source of exposure to the virus; almost half of the participants had TUBERCULOSIS either by clinical or immunological parameters and one out of every five subjects was overweight (BMI, 25 kg/m$^2$).

Baseline association between lipid profiles and TB-associated variables:

The female gender was associated with statistically significant elevation of all serum lipids at baseline, while absent difference was observed between lipid profiles of hypertensive and cohorts. Diabetes mellitus was associated with a significant elevation in mean LDL. Overweight/obese subjects had high TG and low HDL cholesterol. The presence of tuberculosis defines the illnesses or immunological tuberculosis.

Changes in the level of serum lipids and some biochemical parameters after 12 months:

Table 2 shows the changes in lipid profiles after 12 months as shown, there were significant increase in TC, TG, HDL and LDL. Fasting blood glucose also increased. There was a significant reduce in creatinine, but ALT remained unchanged throughout the study period. The weight gain increase in BMI from 25.1± 2 to 28.1± 3. Younger subjects 35 years had a significant mean change in their lipid parameters in comparison to older participants 50 years after 12 months.

Changes in lipid profiles in some metabolic and TB-associated variables after 12 months

Taking gender and some metabolic and TB-associated variables into account, renal impairment was associated with significantly low increase in mean HDL and high increase in TG. Subjects with metabolic and TB-associated variables had similar changes in lipid parameters to other cohorts. This was associated with a statistically significant elevation in mean LDL and TC, as depicted in Table 1.

Table 1: Baseline association between lipid profiles and TB-associated variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>LDL</th>
<th>HDL</th>
<th>TG</th>
<th>TC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>2.79 ± 1.35</td>
<td>1.53 ± 0.76</td>
<td>1.46 ± 0.20</td>
<td>4.78 ± 1.31</td>
</tr>
<tr>
<td>Male</td>
<td>2.42 ± 1.16</td>
<td>1.31 ± 0.76</td>
<td>1.12 ± 0.22</td>
<td>4.28 ± 1.42</td>
</tr>
<tr>
<td>$P$-value</td>
<td>0.027</td>
<td>0.026</td>
<td>0.000</td>
<td>0.006</td>
</tr>
<tr>
<td>Present</td>
<td>2.66 ± 1.20</td>
<td>1.46 ± 0.74</td>
<td>1.35 ± 0.19</td>
<td>4.62 ± 1.31</td>
</tr>
</tbody>
</table>
Table 2: After 12 months, the percentage mean change of the lipid parameters

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Baseline</th>
<th>After 12 Months</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Cholesterol (mmol/L)</td>
<td>4.54</td>
<td>5.16</td>
<td>0.0000</td>
</tr>
<tr>
<td>Triglycerides (TG)</td>
<td>1.29</td>
<td>1.59</td>
<td>0.000</td>
</tr>
<tr>
<td>HDL (mmol/L)</td>
<td>1.42</td>
<td>1.54</td>
<td>0.0440</td>
</tr>
<tr>
<td>LDL</td>
<td>2.63</td>
<td>3.18</td>
<td>0.000</td>
</tr>
<tr>
<td>ALT</td>
<td>33.89</td>
<td>32.03</td>
<td>0.466</td>
</tr>
<tr>
<td>Glucose</td>
<td>4.88</td>
<td>5.34</td>
<td>0.000</td>
</tr>
<tr>
<td>Creatinine</td>
<td>88.13</td>
<td>83.08</td>
<td>0.020</td>
</tr>
<tr>
<td>Urea</td>
<td>4.19</td>
<td>4.83</td>
<td>0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.27 ± 4.21</td>
<td>25.61 ± 3.89</td>
<td>0.001</td>
</tr>
</tbody>
</table>

The mean TG changed by 23.3% over the study period, while LDL, HDL and TC changed by 20.9%, 8.5% and 13.7% respectively.
Discussion

Previous reports have demonstrated that TB-infected subjects exhibit multiple abnormalities in lipid metabolism\(^5\) and especially with the use of protease inhibitors (PIs)\(^{19-21}\). Although the mechanisms that induce lipid abnormalities associated with potent antiretroviral therapy remain elusive, 19 – 21 several path physiologic models have been proposed to explain the development of dyslipidemia in TB-infected subjects, involving several proposed interactions between the virus, antiretroviral therapies, and host factors.\(^6\) Hyperlipidaemia, defined as an increase in TG and TC levels, was observed in this study, consistent with a previous report.\(^7\) The baseline hypercholesterolemia was similar to findings However after 12 months, the prevalence of hypercholesterolemia increased to 32.3 %, which is comparable to 49% on a PI based regimen.\(^{27}\). Subjects who demonstrate elevated TC and/or TG levels should be treated appropriately, in order to prevent the development and progression of atherosclerotic heart disease, stroke, and pancreatitis. The mean HDL determined before commencement in this study was 1.27±0.81 mmol/L which is higher than other reports.\(^8\)

Decreased levels of HDL occur at an early stage of TB infection. Low HDL is a well-recognized independent risk factor for adverse cardiovascular outcomes and this has even been shown to be true in TB-infected individuals, irrespective of other risk factors.\(^9\) Several studies have shown that an HDL increase is associated with a significant decrease in mortality from coronary heart disease (CHD), independent of changes in LDL.\(^10\)

Changes in blood lipids occur naturally during the course of TB infection, with decreases early in the infection in both TC and HDL cholesterol and increases later in TGs.\(^{11}\) our cohort also leads to lipid changes, with increases in both TC\(^{11}\) and TGs\(^{10}\) as previously documented. While increases in TC during therapy may represent a return to pre-infection levels to some degree\(^{12}\), the increased incidence of hypercholesterolemia after 12 months in our study may suggest the associated with an increased risk of myocardial infarction. The strengths of our study include the longitudinal design and 12 month follow-up, combined with our exclusion of pre-treated subjects, and data once subjects switched from one type of therapy to another. The absence of any association between any of the lipid parameters and hypertension may be related to the duration of the hypertension, the degree of blood pressure control or the degree of immune suppression. Even in previously diagnosed hypertensive, who have been on medication or who have a positive family history of cardiovascular events in a first degree relative, there were absent significant correlations between hypertension and abnormal lipid profiles, especially low HDL.\(^{31}\) This suggests that TB infection constitutes an additional and independent cardiovascular risk in hypertensive subjects. Studies have reported elevated TG was shown to positively correlate with interferon alpha, in subjects with advanced disease/opportunistic infection. Markedly reduced immunity had to delayed clearance of plasma lipids due to reduced lipoprotein lipase activity.\(^{13}\) We found absent significant association between hepatitis serological status and abnormalities of the lipid profiles. Other studies have reported low LDL, low TC and low HDL in TB-positive subjects. In particular, those co-infected with hepatitis C and with advanced stages of liver disease tended to have low TC.\(^{14}\) Though the findings were not significant, absolute values of these parameters were lower in those with hepatitis infection 36. TB is a common opportunistic infection in TB/TUBERCULOSIS subjects and causes high mortality if not treated.

Conclusion

Subjects with TB infection should be screened for lipid disorders given their high prevalence as observed in this study, because of its potential for morbidity and mortality to the subjects.
References