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ABSTRACT

Context: Lipid metabolism is altered in leprosy. Whether this is associated with risk of cardiovascular disease is yet to be ascertained, especially in our environment where there is paucity of literature. Aims and objectives: This study aims to determine the total plasma lipids and cholesterol levels in Nigerian male leprosy patients. Patients and methods: Study new and untreated patients of Oji River leprosy settlement (LP) in Enugu State of Nigeria were consecutively recruited and matched with sixty healthy individuals (LF) who were randomly selected by a lucky dip of yes or no. Results: This revealed that the mean plasma total lipid and mean plasma total cholesterol levels of LP (4.27±1.39g/l and 128.12±8.52mg/dl) respectively were significantly lower (P=0.006) than those of leprosy free (LF) (6.89±1.23g/l and 185.21±12.14mg/dl) respectively. Conclusion: These results indicate that leprosy leads to alteration in lipid metabolism. The resultant effect of this significant lowering of serum plasma lipid may be cardioprotective when the levels of other lipid subfractions are taken into consideration.

Keywords: Total lipids, total cholesterol, leprosy, atherosclerosis.

INTRODUCTION

Leprosy is a chronic infectious disease caused by mycobacterium leprae. This organism is transmitted between humans and affects more than 11 millions individuals world wide (Fene 1982). Besides the immunological approach to the problem, researchers have also attempted to study the biochemical alterations including the study of lipid metabolism as a guide to early diagnosis. Lipid composition of various mycobacteria has not been extensively studied. Although, it was initially thought that vacuolisation of foam cells in leprosy was a hydropic change, it was soon established that the contents are made up of lipids (Gupta et al 2002). The ability to synthesize different lipid moieties and their distribution through plasma to all body tissues seems to be disturbed in leprosy (Ahale and Gilbert 1992). It has also been reported that HDL – cholesterol estimation is relevant for early diagnosis of lepromatous leprosy and is also an index for differentiating between paucibacillary and multibacillary leprosy (Kumar et al 1988). Investigation of lipid metabolism is of additional interest as atherosclerosis and myocardial infarction are rare in leprosy (Desikan and Job 1968). Abnormal lipid metabolism may be responsible for some vascular abnormalities seen in leprosy patients. Various studies have been done on other parts of the world, on the alteration of lipid metabolism in leprosy (Misra et al 1964, Srinivasa et al 1979, Khem et al 1983 and Ahale et al 1992). However, there is paucity of literature in our environment. This prompted us to determine the total cholesterol and lipid levels in leprosy in other to stimulate interest in future research on the relationship between various lipid subfractions and histological subtypes of leprosy.

PATIENTS AND METHODS

Study Area:

This study was carried out in Oji River leprosy settlement at Oji River town of Enugu State. Enugu State lies within the forest savannah zone of Nigeria. The average annual temperature is between 22 -1°C and 31°C with a rainfall of 1520 to 2030mm. There are two major seasons, rainy season (April to October) and dry season (November to February). The settlement has an approximate population of about 300 people made up of the leprosy patients attending the clinic, attendants who are free of leprosy and other staffs who are all resident in the settlement.
Serum Lipid Profile of Male Leprosy Patients in Enugu State, South East Nigeria

Study Design
After obtaining informed consent and ethical clearance from the relevant authority, sixty newly diagnosed male leprosy patients (LP, n = 60) were consecutively recruited into the study. Sixty normal, leprosy-free (NF LP n = 60) males resident in the Oji River. They had no clinical or laboratory evidence of any other ailment. Exclusion criteria include: diabetic mellitus, obesity, smoking, chronic alcoholism, hormonal treatment, liver and renal diseases, acute or chronic inflammatory, and overt vascular disease (aortic, transient ischemic attack).

Clinical examination: The diagnosis of leprosy was made by history, physical examination, and laboratory investigations including skin smear examination.

Laboratory methods: Blood samples (5ml) were collected after an overnight fast from the subjects by venepuncture, into a 10ml capacity polyethylene sample bottles containing 0.5% (W/V) ethylenediaminetetraacetic acid (EDTA) as anticoagulant. The blood was thoroughly mixed with the anticoagulant by gentle rocking and then centrifuged as 3000 x g for 10 minutes using a Wisperferalt bench centrifuge (3-todl BS4). The yellowish tip liquid layer was collected with a dropping pipette. The plasma was either used immediately for assays or stored in deep freezer (-20°C) in aliquots of 200µl until used. All the entire chemicals used in this study were of analytical grade. Total lipid assay was determined by the method of (Shirou and Makarova, 1989). Total Cholesterol was measured using Cholesterol enzymatic endpoint method (Stein, 1986).

Statistical Analysis: This was done using the statistical package for social sciences (SPSS) version 11.

Values were recorded as mean and standard deviation. Test for significance was done using the Z-test at 95% confidence interval. Values of p < 0.05 are regarded as statistically significant. Pearson's correlation was used to determine whether there was any association between total lipids and cholesterol.

RESULTS:
The table below shows that the mean plasma cholesterol for LP was 128.12±8.52mg/dl. This was significantly lower than that of the LP group, 185.21±12.14mg/dl (Z-test = 29.89, P=0.000). Furthermore, total lipids for LP was 4.27±1.50g/dl and this was also significantly lower than LP control, 6.80±1.23g/dl (Z-test=10.14, P=0.000). These are also illustrated in figures 1 and 2. There was no significant correlation between total lipid concentration and total cholesterol.

Total plasma cholesterol and lipids (test and control)

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<th>Control</th>
<th>Z-test</th>
<th>P-value</th>
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<td>Total cholesterol (mg/dl)</td>
<td>128.12±8.52</td>
<td>185.21±12.14</td>
<td>29.89</td>
<td>0.000</td>
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<tr>
<td>Total lipids (g/dl)</td>
<td>4.27±1.50</td>
<td>6.80±1.23</td>
<td>10.14</td>
<td>0.000</td>
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Pearson's Correlation: 1.000

Correlation is significant at the 0.01 level (2-tailed) while the Z-test is significant at P<0.05.

Figure 1: Total plasma cholesterol (test and control)
DISCUSSION

The significant decrease in serum lipid levels in this study has been reported by various authors (Alley et al. 1992, Minea and Verma 1964, Sridharan et al. 1979, Kher et al. 1983, Kumar et al. 1980, Chung et al. 1986, and Hurlarte, 1994.). This result is also corroborated and consistent with reports of substantial reduction in plasma polyunsaturated fatty acids in plasma cholesterol infected patients (Demark-Bevers et al. 1995). Hypocholesterolemia found in this study has been in consonance with others reports (Gupta et al. 2002 and Basak et al. 1997). The marked hypocholesterolemia observed in leprosy patients has also been demonstrated in other chronic granulomatous diseases like tuberculosis, rheumatoid arthritis, myeloproliferative disorders, systemic lupus erythematosus and sarcoidosis (Salazar et al., 2001, Ezzat et al. 2005). The decrease in total lipids may be due to decreased lipogenic activity as a result of reported hepatic cellular dysfunction (Molhe 1969). Lipid peroxidation in lepromatous state could also have led to the significant depletion of total lipids, which may be responsible for the weight loss associated with leprosy. Furthermore, it has also been observed that the ability to synthesize different lipid molecules and their distribution through plasma to all the body tissues seem to be altered in leprosy. The observation of low level of cholesterol in lepromatous patients in this study may further support the hypothesis that cell damage causes more cholesterol to be released into the blood stream. However, the observed reduction of cholesterol in lepromatous state could be due to partial inhibition of cholesterol synthesis as was observed in depression of cisarachid and prostaglandin E production by essential fatty acid deficiency under a variety of conditions (Linda et al. 1997). The low incidence of hypertension and coronary heart disease in patients of leprosy may be related to reduction of total cholesterol and elevation of HDL cholesterol subfraction (Gupta et al. 2002, Desikan & Job, 1998). Other factors that may be responsible for the decrease in plasma lipid and cholesterol levels in leprosy are increase in the levels of cytokines such as tumour necrosis factors (TNF) which lowers the HDL level. Decreased activity of lipogenic and glycolytic enzymes (Sridharan et al. 1979), genotoxic factor and ageing (Wyler 1982). The non correlation between total lipid and total cholestrol confirms that both lipid derivatives have the same effect during the leprosy. Little work has been done on lipid metabolism in leprosy in our environment.
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Thus this work serves as a stepping stone for further research. As this study was funded with personal resources, it was not possible to assay the various cholesterol substractions. Further research should take this into consideration while focusing on lipid metabolism and perturbation of the various clinical subtypes of leprosy. Furthermore, long term monitoring of leprosy patients to determine the actual risk of developing atherosclerosis should not be overemphasized.

REFERENCES