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Full Length Research Article

Evaluation of Plasma Lipids and Lipoproteins in Nigerians Suffering From Depressive Illness

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ABSTRACT

There are conflicting reports on the role of plasma lipids in depressive illness. Very little is known about the lipid and lipoprotein status in Nigerian adults suffering from depression. One hundred subjects consisting of sixty (60) depressed patients with mean age (40.3±12.3 yrs) and forty (40) apparently healthy controls (40.1±10.1 yrs) were selected for the study. All subjects were free from medication at least one month prior to the start of the experiment. The anthropometric indices were also determined. There was a significant increase in plasma triglyceride concentration in depressed subjects when compared to control values ($p<0.01$). However, the plasma total cholesterol, low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C) and body mass index (BMI) did not significantly differ from control values. The mean plasma triglyceride was significantly increased in female patients in comparison to corresponding female controls. Plasma lipid and lipoprotein levels did not demonstrate any definite pattern with increasing level of depression in patients. In conclusion, plasma lipid levels could play a significant role in depressive illness in Nigerians

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INTRODUCTION

Depression is a growing health problem and one of

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the least understood diseases. It is differentiated from normal mood changes by the extent of its severity, the symptoms and the duration of the disorder (Murray and Lopez, 1996). The exact cause is not known (Baldwin, 2000). Untreated depression may lead to suicide, which is one of the leading causes of death in the western parts of the world (Klebba, 1979).

Among Africans, earlier studies suggested that depression was rare (Carothers, 1947) but later studies have revealed an increasing prevalence of this condition in many African communities (Leighton et al, 1963, Wintrob, 1967). Evidence suggesting any possible relationship between plasma lipids and mental illness has been conflicting. Boston et al (1996) reported that significant changes in serum lipids might be linked with mental health. Some studies have found that cholesterol is important for the uptake of serotonin, a neurotransmitter which when present at low levels in circulation, is associated with depression (Engelberg, 1992, Maes et al, 1995). In a large Swedish population, Suarez et al (1999) noted that a low serum cholesterol level was associated with a higher prevalence of depression in middle-aged women. Also several studies have reported low total plasma cholesterol concentrations in depression (Glueck et al, 1994, Chen et al, 2000) while others did not show any relationship between circulating levels of cholesterol and incidence of depression (McCallom et al, 1994). However, in some earlier studies (van Doornen and van Blockland, 1987, Shizuka and Yambe, 2001) significantly elevated plasma lipid concentrations were associated with persistent depressive feelings.

Lipids and some elemental micronutrients constitute major parts of the human brain. Therefore, it is speculated that factors affecting cerebral lipid could have profound effects on normal brain function (McLoughlin and Clarke, 1989).

Depressive illness is a major cause of morbidity and mortality especially in the elderly (Baldwin, 2000). Age, gender, apolipoprotein E phenotypes, condition of fraility and inflammation states are important factors that can induce significant modification in lipid profile in the elderly (Etukudo et al, 1999). Also, hypocholesterolaemia is associated with diseases like malnutrition and some chronic diseases which are common in the elderly in different populations (Siemianowiwicz et al, 2000, Zuliani et al, 2001). The presence of all such confounding factors had previously made it difficult to confirm a close link between low serum cholesterol and depression in the elderly populations. Recent interest in biological basis of neurological disorders has attracted much interest worldwide, but there is scanty information on systematic lipid studies in Nigerian Africans. The present study was designed to evaluate the lipid and lipoprotein profiles in adult Nigerians suffering from varying degrees of depression, to assess the confounding effects of gender and the tendency to commit suicide on the possible association between circulating plasma lipid and depression in the Nigerian African.

MATERIALS AND METHODS

Subjects: One hundred (100) subjects were selected for this study after ethical committee approval by the UI/UCH joint ethical committee. The study group was made up of sixty (60) depressed subjects (38 females, 22 males) mean age (40.3±12.3) yrs and forty normal controls (21 females, 19 males) mean age (40.1±10.1) yrs. All the subjects were free from medication for at least one month prior to the start of the experiment. All were normotensive, non-diabetic, without renal or liver dysfunction and with a body mass index (BMI) of less than 30kg/m^2. None of the patients had received lithium therapy. Subjects who frequently took alcohol or smoked or those on a special diet were excluded from the study.

Diagnosis of depression was made using the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria at the Psychiatry Department of the University College Hospital Ibadan by the attendant Consultant Psychiatrist. Other scales used in the study are Hamilton Depression Rating Scale (HDRS) which allows a clinician to rate the severity of depressive symptoms and Center for Epidemiological Studies on Depression Scale (CESD), a self-report questionnaire used mainly in primary care. Normal controls were required to score below 16 on application of CESD (Zich and Attkisson, 1990) and none of them reported any past history of past

history or family history of psychiatric illness. Patients were subsequently classified into clinical subgroups as mild (HDRS 16-25), moderate (HDRS 26-34) and severe depression (HDRS > 34).

Blood Samples: Ten (10) ml of blood was obtained from each patient and control after an overnight fast. Aliquot of samples were drawn into sodium EDTA for lipid analysis and in heparin containers for determination of other biochemical parameters. Blood samples were centrifuged and plasma separated and stored below -25^0C until analysis was carried out.

Physical Parameters: Weights and heights were measured in kilogram and meters respectively. The BMI was then calculated as BMI = kg/m^2.

Methods: Enzymatic colorimetric methods were employed for the estimation of plasma total cholesterol (Allain et al, 1974) and triglyceride (Buccolo and David, 1973) HDL-cholesterol was also determined using enzymatic colorimetric methods after separation from other lipoproteins using a mixture of phosphotungstic acid and magnesium chloride. LDL-cholesterol was calculated using the formular of Friedwald et al (1972) while activities of liver enzymes (aspartate and alanine transferases) and plasma albumin levels were determined using the methods of Bergmeyer et al (1986) and Doumas and Watson (1971) respectively.

Statistical Analysis: Results were expressed as mean ± standard deviation. Comparison of means was made using the student-t- test and $p<0.05$ regarded as significant. Group comparisons were carried out using one-way analysis of variance (ANOVA). Pearson correlation coefficient (r) was used to determine the relationship between variables.

RESULTS

The slight increase in the mean values of total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C) observed in all depressed patients were not statistically significant ($p>0.05$) when compared with the corresponding values in the control subjects (Table 2). Similarly, the reduction in mean high-density lipoprotein cholesterol (HDL) in depression was not statistically significant. On the other hand, the increase in mean plasma Triglyceride concentration in depressed patients when compared with the concentration in the control group was significant ($p<0.01$).

TABLE 1: Age, Body Mass Index (BMI), Weight and Height in Patients and controls.

Physical Parameters	Depression (N=60)	Control (N=40)	t-value	P
AGE (Yrs)	40.3 ± 12.3	40.1 ± 10.1	0.1069	N.S
BMI (Kg/m^2)	21.9 ± 3.4	21.6 ± 2.7	0.353	N.S
Weight (Kg)	61.8 ± 13.2	57.2 ± 16.5	1.476	N.S
Height (m)	I.68 ± 0.2	1.63 ± 0.5	0.601	N.S

N = number of subjects; N.S = Not significant

TABLE 2: Plasma Cholesterol, Triglyceride, HDL And LDL Levels in Patients and Controls

Parameters	Depression (N=60)	Control (N=40)	t- value	P
Total cholesterol (mg/dl)	172 ± 33	171 ± 35	0.114	N.S
Triglyceride (mg/dl)	87 ± 26	70 ± 19	3.561	P<0.01
HDL-c (mg/dl)	48 ± 16	53 ± 12	1.8998	N.S
LDL-c (mg/dl)	107 ± 29	101 ± 38	0.788	N.S
AST (I U/L)	23 ± 11.6	25 ± 8.8	1.196	N.S
ALT (IU/L)	15 ± 12.1	16 ± 6.3	0.536	N.S
Albumin (g/dl)	4.2 ± 0.50	4.3 ± 0.4	0.939	N.S

N = Number of Subjects; HDLc = High density lipoprotein cholesterol; LDLc = Low density lipoprotein cholesterol; N.S = Non-significant

TABLE 3: Plasma Lipids, Lipoprotein Cholesterol Concentrations in Male and Female Patients

Parameters		Depression	Control	D Vs C
Cholesterol (mg/dl)	Female	171 ± 31 (n = 38)	F 180 ± 31 (n = 21)	M vs M^{NS} F vs F^{NS}
	Male	174 ± 38^{NS} (n = 22)	M 162 ± 37 (n = 19)	
Triglyceride (mg/dl)	Female	84 ± 22 (n = 38)	F 62 ± 11 (n = 21)	M vs M^{NS} F vs F, $p < 0.01$
	Male	91 ± 32^{NS} (n = 22)	M 78 ± 23 (n = 19)	
HDL-c (mg/dl)	Female	50 ± 15 (n = 38)	F 52 ± 14 (n = 21)	M vs M, $p < 0.01$ F vs F^{NS}
	Male	44 ± 12^{NS} (n = 22)	M 54 ± 9 (n = 19)	
LDL-c (mg/dl)	Female	105 ± 28 (n = 38)	F 110 ± 40 (n = 21)	M vs M^{NS} F vs F^{NS}
	Male	111 ± 31^{NS} (n = 22)	M 92 ± 33 (n = 19)	

n = number of subjects; D Vs C = Depression Vs Control; HDL-c = High-density lipoprotein cholesterol NS = Non-significant; LDL-c = Low-density lipoprotein cholesterol; p>0.05 = NS; M = Male; F = Female

Table 4: Plasma Lipids, Lipoprotein Cholesterol Concentrations and LDL-C/HDL-C Ratio in Mild, Moderate and Severe Depression (Mean ± SD)

Parameters	Mild M1 (n=25)	Moderate M2 (n=25)	Severe M3 (n=10)	F	P
Total Cholesterol (mg/dl)	172±38	169±29	180±36	.422	NS
Triglyceride (mg/dl)	87±27	86±23	89±31	.033	NS
LDL-c (mg/dl)	108±28	102±25	115±42	.648	NS
HDL-c (mg/dl)	50±17	49±14	48±21	.061	NS
LDLc/HDLc	2.4±0.9	2.2±.8	3.1±2.2	1.9	NS

n=number of subjects; LDL-c=low-density lipoprotein cholesterol; HDL-c=High-density lipoprotein cholesterol

TABLE 5: Correlation Coefficients for Lipids, Lipoproteins and Depression Rating Scores in Depression

	TC	TG	HDL-C	LDL-C	HDRS
TC	1.00	.153	.428[b]	.890[b]	-.253
TG	.153	1.00	-.204	.112	-.302
HDL-C	.428[b]	-.204	1.00	-.005	-.061
LDL-C	.890[b]	.112	-.005	1.00	-.182
HDRS	-.253	-.302	-.061	-.182	1.00

a= P<0.05; b = p<0.01

When the subjects were classified according to gender, (Table 3) compared with the corresponding values in male control subjects, there were no significant differences between the plasma total cholesterol, triglyceride and LDL-cholesterol in male depressed patients. On the other hand, only the mean plasma HDL- cholesterol level was significantly reduced in male patients when compared with the corresponding value in the male control subjects (p<0.01). In female depressed patients, the mean plasma total cholesterol, HDL-cholesterol and LDL-cholesterol levels were not statistically different from the corresponding mean values in the female control group. However, the mean concentration of plasma triglyceride in female depressed patients was significantly increased when compared with the concentrations in the female control group (P<0.01). As shown in table 4, there were no significant differences in the plasma levels of total cholesterol, triglyceride, HDL-cholesterol and LDL-cholesterol between mild, moderate and severe depression. Similarly, plasma lipid and

lipoprotein concentrations did not correlate with Hamilton Depression scores (HDRS) (Table 5)

DISCUSSION

The results of this study suggest that plasma total cholesterol was not associated with depression. Similarly, the changes in mean LDL-C and HDL-C concentrations were not significantly altered when compared to the respective values in the control group. These results in middle aged Nigerians of varying socio-economic classes agree with those of other studies (McCallom et al, 1994) but they are at variance with others who showed that the mean total cholesterol was either reduced (Chen et al, 2000) or elevated (Shizuka and Yambe, 2001) in depression. Mechanisms of action adduced by these workers were the speculated direct relationship between low cholesterol levels and poor serotonin uptake and lipid-induced increased viscosity leading to low cerebral perfusion respectively.

Inconsistencies among these various reports appear to suggest that the link between plasma total cholesterol and occurrence of depression is less straightforward than it might seem, probably, due to factors like age, other clinical conditions, nutritional factors or other life style factors that modulate plasma lipids. For instance, the significant inverse relationship between cholesterol and depression in a previous study were limited to elderly persons and some of the studies only made use of self-reporting questionnaires. Thus, it could not be ascertained what proportion of reported depression was clinically significant.

The most striking change in lipid profile in this study is the significant increase in plasma triglyceride in depression, irrespective of the severity of disease, mood (suicide tendency or not) or level of plasma total cholesterol. It was also noted that the change was more pronounced in female patients when compared with their male counterparts. The possible role of triglyceride metabolism in the aetiology of depression is largely unknown but Fowkes et al (Fowkes et al, 1992) in an earlier study indicated a positive relationship between serum circulating triglyceride concentration and personality trait in depression. Similarly, a previous study (Rogers et al, 1989) had reported that hypertriglyceridemia is associated with peripheral neuropathy and dementia especially in young subjects and the clinical conditions were reversed when a reduction in the grossly elevated lipids was achieved by drug or dietary therapy. It has been speculated that elevated lipids could enhance atherogenesis of the extracranial and intracranial arteries causing an increase in blood viscosity, which in turn decrease cerebral perfusion and impair cognitive performance (Fowkes et al, 1992). However, the finding in the present study is at variance with some others who could not confirm any association between high triglyceride values and depression or psychological symptoms.

Engelberg (1992) and Glueck et al (1994) had shown that low serum cholesterol concentration is associated with suicide. However, plasma lipid profiles in the subgroup of patients that had suicidal tendency or made an attempt to commit suicide in this study did not reveal an striking changes in lipid concentration when compared with the corresponding levels in other depressed subjects who had no tendency for suicide. The lack of statistically significant differences between the two groups included in the present study may be due to the small number of

patients with suicidal tendency, a finding consistent with the suggestion (Asuni, 1962) that suicides appear to be uncommon in Nigerians. It was important to determine the liver function status in all the subjects studied, since neurological complications in liver diseases may often manifest in the form of intermittent confusion and abnormal behavior including tremor of the hands (Walton, 1984) The mean plasma albumin, a sensitive index of protein energy malnutrition as well as a measure of the synthetic ability of the liver and the activities of the aminotransferases were within normal range in the patients suffering from mild, moderate or severe depression.

In conclusion, this study suggests that altered triglyceride levels could contribute to depressive illness probably via increased blood viscosity leading to lowered cerebral perfusion.

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