LIPID PROFILE AND FATTY-ACID COMPOSITION OF HUMAN SERUM IN HYPERTENSION PATIENTS

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The aim of this study was designed to determine the effect of hypertension on the level of lipid fractions and percentage of fatty acids in serum. The study included 50 patients with hypertension, blood was taken after 10-12 h fasting. The age of patients was between 60 and 80 years. Blood samples from (50) normal subject with the same age were collected as control. The patients samples collection were from the medical word. A number of biochemical parameters were measured using enzymatic kits methods also the analysis and the measurement of percentage of fatty acids in fatty component of serum (cholesterol ester (CE), phospholipids (PL) and triglyceride (TG)) separated by thin layer chromatography(TLC) followed by transmethylation of fatty acids and measurement of fatty acids percentage using Capillary Gas Chromatography (CGC). The result of this study showed that there is a significant differences in the level of studied biochemical parameters and fatty acids percentage in hypertension patients compared with the control group. The results of this study also showed that a significant increase in level of (TG) in serum of hypertension patients. The result showed that a significant increase in percentage of (PUFA) in (PL) part.

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Introduction

Dietary fatty acid intake may affect blood pressure. Some studies have found that saturated fats increase blood pressure and that polyunsaturated and monounsaturated fats decrease blood pressure. Many of the studies that have examined the relation between fatty acids and blood pressure have measured or manipulated the dietary intake of fatty acids. Because the methodologies that assess dietary intake are imprecise, alternative methods of estimating fatty acid intake have been proposed. Dietary-derived essential fatty acids that may be associated with blood pressure can be precisely measured in the cholesterol esters and phospholipids of serum lipoproteins.[1] The nonessential fatty acid composition of cholesterol esters and phospholipids reflect dietary consumption as well as fatty acid synthesis and metabolism and therefore are less reliable indicators of dietary intake. Nevertheless, the associations with blood pressure of individual saturated fatty acids, such as myristic acid (14:0), palmitic acid (16:0), and stearic acid (18:0), which are highly correlated in the diet may be examined with the use of serum fatty acid levels.[2]

To examine the association between serum fatty acids and blood pressure, we conducted a cross-sectional study of men enrolled in the Multiple Risk Factor Intervention Trial (MRFIT).[3] Using stored frozen serum samples that were collected at the outset of the study, we measured the serum fatty acid levels in fifty patients and fifty control subjects. We have performed stepwise multivariate analyses to determine whether serum fatty acids were independently associated with blood pressure.

Experimental

Collection and treatment of blood samples

In this study the blood samples were collected from hypertension patients and control subjects over a period between 9/12/2008 and 23/2/2009, after fasting for 10-12 h. Blood (5 mL) was collected from each subject and then the serum was separated. Serum was divided into two parts. From one part, total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), triglyceride (TG), low density lipoprotein cholesterol (LDL-C) were measured by enzymatic methods using kits.[4,5] Very low lipoprotein cholesterol (VLDL-C) was evaluated theoretically.[6] The second part was stored at -18 °C for the measurement of fatty acids.

Serum samples were treated with methanol and chloroform to extract lipids.[7] Lipids extract was separated into three parts cholesterol ester (CE), TG and phospholipids (PL) by TLC.[8] Analysis and re-esterification of fatty acids were achieved using boron trifluoride(BF_3)(16 %) in methanol.[9] Determination of fatty acids in the three lipid fractions was performed by Capillary Gas Chromatography (CGC) Shimadzu 2010, column type TR-WAX, and length 30 m.

Statistical analysis of results from biochemical parameters and percentage of fatty acids was performed using T-test, $p \leq 0.05$ was considered significant.[10]

Results

Lipid Fraction

The results presented in Table 1 showed significant increase in TC ($p<0.05$), LDL-C ($p<0.001$), TG ($p<0.001$) and VLDL-C ($p<0.001$) in the patient group in comparison

with those of the control group. On the other hand, the results showed significant decrease in HDL-C.

Percentage of fatty acids

The percentage of fatty acids was measured using CGC through comparison of results with standard sample composed of twelve fatty acids. Retention time (RT) of the standard fatty acids is given Table 2.

Table 1. Serum lipids from patient and control groups.

Lipid fraction mmol L^{-1}	Control $n = 50$	Hypertension patient $n = 50$	*P* value
TC	4.80±0.31	5.01±0.80	≤0.05
HDL-C	1.48±0.10	0.80±0.10	≤0.05
LDL-C	2.80±0.25	3.21±0.23	≤0.001
TG	1.19±0.10	3.40±0.46	≤0.001
VLDL-C	0.28±0.03	1.20±0.35	≤0.001

Table 2. Retention time of standard fatty acids.

Standard fatty acids	Symbol	Retention time (min)
Capric acid	C10:0	4.900
Lauric acid	C12:0	5.138
Myristic acid	C14:0	8.500
Palmitic acid	C16:0	10.08
Palmitoleic acid	C16:1	16.74
Stearic acid	C18:0	19.09
Oleic acid	C18:1	19.48
Linoleic acid	C18:2	20.12
Linolenic acid	C18:3	22.20
Arachidonic acid	C20:4	23.46
Eicosapentaenoic acid	C20:5	25.12
Docosahexaenoic acid	C22:6	26.68

The results of the determination of fatty acids in the patient and control groups showed that there is no significant increase in percentage of total saturated fatty acids (SFA), whereas significant increase in total percentage of monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) in the patient group in comparison with control group in the CE part. Also the results showed that a significant decrease in total of SFA and MUFA and a significant increase in total of PUFA in the patient group in comparison with control group in the PL part. The result presented in Table 3 showed that a significant increase in total of SFA and MUFA, and significant decrease in total of PUFA in the patient group in comparison with the control group in TG.

Discussion

Lipid Fractions

The results showed that there is a significant increase in TC in the patient group as compared with that of control group (Table 1). This may well be due to an increase in TC synthesis as a result of insulin resistance in patients.[11] The results also showed that a significant decrease in HDL-C in the patient group in comparison with the control group, which may be due to a close relationship to the elevated activity of plasma cholesterol ester transfer protein which promotes the lipoprotein cholesterol of HDL to be transferred to other lipoprotein in patients.[12] The observed significant increase in LDL-C may be due to a defect in hepatic receptor, Apo B100, which plays an important role in increasing LDL-C through decreasing transport of LDL-C to hepatic tissue.[13]

The result of this study indicate that there is a significantly higher level of TG and very low density lipoprotein cholesterol in the patient group as compared those in the control group. It may be either due to decrease in lipoprotein lipase activity in hypertension patients which leads to decrease in TG clearance from blood[14] or due to insulin resistance in hypertension patients which causes abnormality in metabolism of lipids.[15]

Fatty acids in CE fraction

The results of this study indicate that there was no significant difference in the percentage of saturated fatty acids between that in the patient and control groups. Further, there is a significant increase in the total percentage of monounsaturated and polyunsaturated fatty acids. This may be due to an abnormality in action of lipoprotein lipase which leads to a defect in metabolism of TG and lead to increase percentage of fatty acids.[16]

Fatty acids in PL fraction

The results of this study showed that there is a significantly lower percentage of total SFA in the patient group as compared that in the control group. It may due to an increased ingestion of some type of food which leads to increase the in the risk factor in the patient group in comparison with control group.[17] This study also showed a significantly lower percentage of total MUFA and a significantly higher percentage of total PUFA for the patient group as compared to those of the the control group. It may be due to insulin resistance in hypertension patients and cardiac disease in general which leads to a big defect in enzymes action specially lipoprotein lipase and also defect in action for enzymes of desaturase class and elongation and oxidation process of fatty acids.[1]

Fatty acids in TG fraction

The result of this study indicate that there is a significant increase in the percentage of total SFA in the TG fraction. It may be due to transport of Acetyl-CoA from different metabolism pathways to the pathway causing anabolism of SFA.[18]

The results showed that there was a significant increase in percentage of total MUFA and a significant decrease in percentage of total PUFA. It may be due to a defect in the action of desaturation enzymes (Δ9), (Δ6), (Δ5) and elongation enzymes in stroke patients.[19]

Table 3. Percentage of fatty acids composition of CE, PL and TG in the patient and control groups.

Fatty acid	CE		PL		TG	
N	Control 10	Patients 10	Control 10	Patients 10	Control 10	Patients 10
			SFA			
10:0	1.2±0.23	0.98±0.05	0.80±0.01	0.10±0.08	0.55±0.01	0.9±0.08
12:0	0.88±0.31	1.22±0.02	1.2±0.02	1.55±0.05	0.78±0.20	1.88±0.5
14:0	0.72±0.20	0.78±0.10	0.25±0.1	0.28±0.10	2.0±0.05	2.0±0.08
16:0	11.45±1.5	11.51±1.0	26.5±1.5	22.5±1.89	25.5±0.60	27.0±1.8
18:0	1.35±0.27	1.56±0.35	15.8±1.2	12.8±1.52	6.80±0.24	8.0±0.63
Total	15.5±2.50	15.82±1.5	44.05±2.8	37.13±3.6*	35.53±1.0	39.3±3.1*
			MUFA			
16:1	3.88±2.24	4.21±1.24*	1.20±0.5	0.8±0.45*	2.25±0.15	4.1±1.0*
18:1	19.20±3.0	21.58±2.2*	9.50±1.2	7.21±0.97*	30.26±1.2	33.5±1.5*
Total	23.08±5.2	25.79±3.5*	10.7±1.70	8.01±1.42*	32.5±1.36	37.6±2.5*
			PUFA			
18:2 n-6	38.0±1.0	49.23±4.2*	28.0±1.8	32.02±3.2*	18.0±2.0	14.0±.51*
18:3 n-3	0.45±0.12	0.40±0.15	1.8±0.56	1.95±0.23	2.10±0.45	2.89±.90
20:4 n-6	8.5±1.80	10.15±1.7*	10.62±2.1	12.75±2.0*	1.85±0.05	2.06±.30*
20:5 n-3	1.20±0.2	1.58±0.01	1.56±0.62	1.24±0.25	2.00±0.12	1.05±.01*
22:6 n-3	0.50±0.02	0.85±0.12	4.0±1.10	6.2±1.10*	3.25±0.95	1.20±.20*
Total	50.65±3.1	58.21±6.2*	39.98±6.2	48.16±6.7*	27.2±3.57	21.2±1.9*
n-3	2.15±0.34	2.83±0.28	7.36±2.28	9.39±1.58*	7.35±1.52	5.14±1.1*
n-6	48.5±2.80	55.38±5.9*	32.6±3.90	38.77±5.2*	19.85±2.0	16.06±.8*

*P value ≤ 0.05

Conclusions

High level of triglyceride is linked to atherosclerosis. Condition in which cholesterol and other substances from plaque fragment or blood clots that can bloke the flow of blood in an artery supplying either to the heart, which could cause a heart attack, or to brain, which could cause a stroke. The results of this study showed that there is a in decrease proportion of total SFA and MUFA, and an increase in the proportion of total PUFA. This indicates that there may be an increase in the lipids peroxidation in hypertension patients also an increase in the metabolism of MUFA to PUFA, which considered as a risk factor for stroke because it increases the amount of free radicals.

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