Plasma Ascorbic Acid, Lipid and Lipoproteins levels in HIV Infected Patients

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Abstract: Human Immunodeficiency Virus (HIV) induces a wide array of biochemical and immunological alterations resulting in the progressive impairment of cellular functions, several metabolic abnormalities and eventual development of acquired immune deficiency syndrome (AIDS). There is paucity of information on the plasma ascorbic acid concentration in HIV patients on antiretroviral therapy in University College Hospital This study was designed to assess the interplay of ascorbic acid, lipids and lipoproteins in HIV positive patients on antiretroviral therapy. One hundred subjects consisting of fifty HIV positive patients with mean age of 34.02 ± 0.95 years and fifty controls with mean age of 35.06 ± 0.98 years were selected for this study. Plasma ascorbic acid, lipids and lipoproteins were estimated using spectrophotometric methods. The anthropometric indices were measured using standard methods. The result showed significant reductions in plasma high density lipoprotein cholesterol (HDLC) (p<0.01) and body weight (p<0.05) when compared with the controls. There were significant increases in the plasma total cholesterol (TC) and low density lipoprotein cholesterol LDLC (p<0.01) when compared with corresponding control values. There were significant correlations between TC and HDLC (r=0.427, p<0.01) and LDLC (r=0.975, p<01).TC/HDLC ratio was negatively correlated with TC/LDLC (r=-0.782, p<0.01) and HDLC/LDLC (r=-0.742, p<0.01). Plasma ascorbic acid, lipids and lipoproteins were altered in our human immunodeficiency virus. Infection patients on antiretroviral therapy. There were interplays in the plsma lipids and liporoteins in HIV patients.

[Ebesunun Maria, Finebone Patience, Adetunji Kehinde, Umahion Kingsley. **Plasma Ascorbic Acid, Lipid and Lipoproteins levels in HIV Infected Patients**. New York Science Journal 2011;4(5):78-81]. (ISSN: 1554-0200). http://www.sciencepub.net/newyork.

Keywords: Ascorbic acid, cholesterol, High density lipoprotein, human immunodeficiency virus

1. Introduction

Human Immunodeficiency Virus (HIV) is a retrovirus that attacks a specific type of white blood cells known as T-Lymphocytes, which is important in the formation of antibodies. CD+T-Lymphocytes are the primary targets of HIV infection because of the affinity to the CD+4 surface markers. Infection with HIV leads to a progressive impairment of cellular functions characterized by a gradual decline in peripheral blood CD+4 T-Lymphocyte levels. This results in an increasing susceptibility to a wide variety of opportunistic viral, bacterial, protozoan and fungal infections (Khiangte et al.2007, Ball and Fowke, 2003)

Lipid, an ubiquitous compound has many key biological functions, such as acting as structural components of cell membranes, serving as energy storage and participating in signaling pathways (Fashy and Brown, 2005). An increased level of triglyceride could also block blood vessels and cause complications such as abdormal pain, pancreatitis and atherosclerosis (Fashy and Brown, 2005). Study has associated HIV infection with increased plasma triglyceride levels by decreasing the clearance of circulating lipoproteins; a process considered to be due either to reduced lipoprotein lipase or by stimulating hepatic synthesis or re-esterification of fatty acids derived from lipolysis (Obirikorang et al, 2011,Grunfeld and Kotler 1991).

The Ascorbic acid, an antioxidant vitamin, serves as an aqueous phase free radical scavenger preventing lipid peroxidation and protects DNA from free radical damage (Graby and Singh 1991).

Ascorbic acid assists the immune system to rid the body of foreign invaders. It accomplishes these by stimulating the production of white blood cells, primarily neutrophils; which attack foreign antigens such as bacteria and viruses (Moses and Jack 1998). It plays significant role in the synthesis of neurotransmitter, nor-epinephrine and carnithine (Allard and Chau 1998).

Reports showed that excessive free_radicals are produced in HIV infection; which progressively overwhelms the body antioxidant nutrients resulting from oxidative stress and eventually tissue damage (Papadopulos-Eleopulos et al, 1995).

This study was designed to determine the plasma ascorbic acid, lipids and lipoproteins in HIV infected patients on antiretroviral therapy.

2. Materials and Methods

Subjects: Fifty HIV positive patients on treatment with pre highly active antiretroviral therapy consisting of 20 males and 30 females, aged 25-45 years were randomly selected for this study. Fifty apparently healthy HIV negative volunteers within the same age range (33 males and 17 females) served as controls. The diagnosis of HIV was based on laboratory and clinical assessments. Ethical approval for the study was obtained from the University of Ibadan/University College Hospital Ethical Review Committee. Inform consent was obtained from each participant before the commencement of the study.

Anthropometric Measurement: Bodyweight and height of all subjects were measured using standard methods and body mass index (BMI) calculated as body weight (kg)/height (m²)

Sample Collection and Methods of Analysis: Venous fasting blood sample was collected from each patient and control into ethylene diamine tetra acetic acid bottle. Samples were centrifuged within one hour of collection and the plasma separated into clean dry specimen tubes. The plasma was immediately analyzed for the ascorbic acid content (Kyaw 1978). while the remaining was stored at -20° C until analyzed for total cholesterol, high density lipoprotein cholesterol after precipitation (Allain et al. 1973) and triglyceride (Buccolo and David 1973). Low density lipoprotein cholesterol was calculated using Friedewald formula (Friedewald et al, 1972).

Accuracy and precision of biochemical tests were monitored by including commercial quality control samples within each batch of test assay.

Statistical Analysis

Means of results were analyzed using statistical package of social sciences (SPSS). Student's-test was used to compare two means and Cox correlation coefficient was used to assess association between biochemical and biophysical parameters. Differences in means were regarded as significant at p<0.05.

3. Results

Table 1showedthebiophysicaland biochemical parameters of all the subjects. The age, height and body mass index were not significantly different from the corresponding control values. The body weight was significantly reduced in the human immunodeficiency virus positive patients compared with the control value (p<0.05). There were significant increases in the plasma total cholesterol and low density lipoprotein cholesterol (p<0.01) when compared with the corresponding control value. Although the plasma triglycerides were slightly higher in the human immunodeficiency virus positive patients, this increase was however not statistically significant when compared with the corresponding control values. There was a significant decrease in the plasma high density lipoprotein cholesterol (p<0.01) in human immunodeficiency virus positive patients when compared with the corresponding control value. The mean plasma ascorbic acid was slightly lower in the human immunodeficiency virus positive patients. This increase was however not statistically significant.

Table 1: Biophysical and biochemical parameters of all subjects (Mean ± SEM)
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Variables	Patients $n = 50$	Controls $n = 50$	t-value	p-value	
Age (Yrs)	34.02 ± 0.95	35.06 ± 0.98	-0.76	NS	
Weight (Kg)	57.32 ± 1.70	70.38 ± 5.90	-2.12	< 0.05	
Height (m)	1.61 ± 0.01	1.63 ± 0.01	-1.72	NS	
BMI (Kg/m ²)	22.15 ± 0.58	26.61 ± 2.44	-1.78	NS	
Ascorbic acid (mg/dl)	0.40 ± 0.03	0.49 ± 0.02	-1.22	NS	
TC(mg/dl)	187.60 ± 13.80	134.74 ± 4.60	3.64	P<0.01	
TG(mg/dl)	140.04 ± 10.70	129.84 ± 8.40	0.75	ns	
HDLC(mg/dl)	38.68 ± 2.60	58.18 ± 2.90	-5.04	P<0.01	
LDLC(mg/dl)	120.97 ± 12.60	50.59 ± 4.40	5.26	P<0.01	
TC/HDLC	0.57 ± 0.03	0.36 ± 0.02	5.67	P<0.01	
HDLC/LDLC	3.75 ± 0.50	0.36 ± 0.02	5.14	P<0.01	

HDLC = High Density Lipoprotein Cholesterol Yrs = Years BMI = Body Mass Index NS = Not Significant SEM = Standard Error of Mean TC = Total Cholesterol NS = Not Significant TG = Triglyceride **Table 2** shows Cox's correlation coefficient of all parameters in human immunodeficiency virus positive patients. Age was significantly correlated with body weight (r=0.332, p<0.05) and BMI(r=0.296, p<0.05), while body weight was significantly correlated with height (r=0.432, p<0.01) BMI(r=0.296, p<0.05).TC was significantly correlated

with HDLC (r=0.427, p<0.01) and LDLC (r=0.975, p<0.01) respectively. TC/HDLC was negatively correlated with TC/LDLC (r=-0782, p<0.01) and HDLC/LDLC (r=-0.742, p<0.01).No significant correlations were obtained in the other parameters.

Table 2: Cox's correlation coefficient of physical and biochemical parameters in	n HIV positive patients.
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	Age (Yrs)	Weight (Kg)	Height (m²)	TC (mg/dl)	HDLC (mg/dl)	LDLC (mg/dl)	BMI (kg/m²)	TC/HDLC	TC/LDLC	HDLC/LDLC
Age (Yrs)		.332*					.296*			
Weight (Kg)	.332*		.432**				.863**			
Height (m ²)	.432*									
TC (mg/dl)					.427**	.975**				
HDLC (mg/dl)				.427*						
BMI (kg/m²)	.296*	.863**								
LDLC (mg/dl)				.975**						
TC/HDLC									782	742
TC/LDLC HDLC/LDLC									782	.644**
TIDEC/EDEC									.762	

TC = Total Cholesterol

HDLC = High Density Lipoprotein Cholesterol

LDLC = Low Density Lipoprotein Cholesterol

BMI=Body mass index

Yrs = Years

4. Discussion

The body weight was significantly reduced in the human immunodeficiency virus positive patients. This change may be attributed to viremia; which could have contributed to decrease in immune response and this invariably may have accounted for the reduced body weight. This finding is similar with a previous report (Behrens et al. 2000) which showed apparent reduction in body weight in HIV patients due to loss of subcutaneous fat.

Although, there was a decrease in the mean plasma ascorbic acid value, this decrease was however not significant. This slight decrease may suggest a gradual depletion of cellular antioxidant in these patients that in part regulates the free radicals generated during oxidative stress which expectedly could be overwhelming in these patients. It could be speculated that the insignificant increase in plasma ascorbic acid, may be due to increase awareness among our patients on the essentials of dietary supplementation of ascorbic acid. The plasma total cholesterol and low density lipoprotein cholesterol were significantly different from the controls, the positive correlation between total cholesterol and low density lipoprotein cholesterol indicates that increase in low density lipoprotein can lead to corresponding increase in total cholesterol. This may suggest predisposition of HIV patients to cardiovascular disease and peripheral disease risk; since increase in low density lipoprotein cholesterol is a possible index of atherosclerosis risk (Fashy and Brown 2005). The TC/LDLC was significantly higher in the patients than the controls. An earlier study has shown that increase level of this is a strong marker of atherogenic index (Behrens et al, 2000).

The decreased in the mean HDLC of HIV patients as obtained in this study is an indication of potential risk of CVD in these patients. An earlier report by Grunfeld et al, (Grunfeld and Kotler 1991) showed reduced level of plasma HDLC in HIV infected patients. Also previous study by Goldbount et al,1998, showed that for every 1mg decreased in plasma HDLC increases the the risk of CVD by 2.5%. This suggests that these patients are more likely prone to developing premature cardiovascular disease in which reduced plasma HDLC is a part. Plasma triglyceride, a known independent risk factor for cardiovascular disease was not significantly increased in our HIV positive patients when compared with the controls. This is at variance with previous report in increase plama triglyceride was obtained (Fashy and Brown, 2005).

5. Conclusion

Plasma ascorbic acid, lipids and lipoproteins were altered in our human immunodeficiency virus patients on antiretroviral therapy. There were interplays in plasma TC, LDLC and HDLC in our patients. However, further study is warranted.

Acknowledgement

We wish to acknowledge the co-operation of the patients and the control volunteers for their willingness to take part in this study.

There are no conflicts of interest among the authors.

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