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Research Article

Plasma Fat Status is Associated with the Prevalence of Opportunistic Infections and may Predict Disease Progression in HIV/AIDS

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Abstract

Objective: To establish the association between plasma fat status and the prevalence of opportunistic infections in HIV/AIDS patients.

Design and Methods: A Cross Sectional and Quantitative study involving 174 adult HIV/AIDS patients recruited over a period of 18 months at a University Teaching Hospital in Ndola, Zambia. Participants were subjected to clinical assessments with anthropometry, $CD4^+$ count, viral load and plasma fat measurements at baseline and repeated on a follow-up visit. The Wilcoxon rank sum test for continuous variables and the Chi square test for categorical variables were applied to compare the study population by gender. The main research question was addressed by establishing the association between Plasma fat and Opportunistic infections, adjusted for potential confounders using the multiple linear regression model.

Results: The plasma fat status revealed a higher median total cholesterol of [3.86(3.02,4.62) mmol/l], median triglyceride [1.19(0.87,1.51) mmol/l] and LDL-c [2.31(1.58,2.90) mmol/l] for females than that for the males [3.53(3.06,4.61) mmol/l], [0.96(0.71,1.60) mmol/l] and [1.86(1.36,2.80) mmol/l] individually. In contrast, median HDL-c concentration were higher in the male [1.4(1.21,1.55) mmol/l] than the female gender [1.33(1.13,1.51) mmol/l]. The results per clinical status revealed relatively more males [17(27%)] than females [15(15.2%)], p=0.06 presenting with opportunistic infections. Results in both the overall and by

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gender regression analyses suggested a consistent inverse interaction involving most of the plasma fat types and BMI with opportunistic infections.

Conclusion: Plasma fat status was not significantly predictive of the prevalence of opportunistic infections. However, the consistent inverse association reported between plasma fat and opportunistic infections, may suggest a potential role of plasma fat in immune mechanisms or viral suppression and corresponding clinical outcomes.

Keywords: Malnutrition; Opportunistic infection; Plasma fat status; HIV/AIDS; Disease.

Introduction

Background to the problem

HIV/AIDS remains a major cause of morbidity and mortality in Zambia [1], despite significant progress achieved in the provision of anti-retroviral therapy and accompanying support services over the past 3 decades. There is still evidently a high HIV prevalence of 11.1% [2], coupled with a high burden of malnutrition [3], among the HIV infected adults presenting for care in Zambia. The vicious interaction between HIV/AIDS and malnutrition can have a devastating impact on clinical outcomes resulting in worsening morbidity and mortality rates. HIV/AIDS often presents with a wide range of opportunistic infections and co-morbidities which if left unattended can result in mortality. This should underscore the importance of taking a holistic approach than focusing on the part of ART alone in the management of HIV/AIDS. In prior studies low CD4 counts, advanced WHO clinical stage, anemia, immune reconstitution inflammatory syndrome and malnutrition have been associated with high early mortality in the course of treatment [4-7].

Prior research on micronutrients has quite extensively covered the role of multiple vitamins and minerals [8-11], but the role of macronutrients such as fats in immune mechanisms involving $CD4^+$ counts and viral load suppression in HIV/AIDS patients in resource limited settings like Zambia is not well known.

Fat is defined chemically as triglycerides; trimesters of glycerol with several fatty acids [12,13]. Triglycerides, cholesterol and other essential fatty acids, store energy, insulate us and protect our vital organs. They start chemical reactions involved in growth, immune function, reproduction and other aspects of basic metabolism. Fats help the body stockpile certain nutrients as well. The so-called "fat-soluble" vitamins-A, D, E and K are stored in the liver and in fatty tissues [14].

An essential fatty acid is a polyunsaturated fatty acid required by the body that is synthesized by plants however, not by the human body and is thus a dietary requirement [15]. In this study, we sought to examine the potential role of plasma fat towards disease progression by establishing its association with the prevalence of opportunistic infections in HIV/AIDS.

Literature review

The Global Nutrition Report shows that 44% of countries with data available (57 out of 129 countries), now experience very

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serious levels of both under nutrition and, adult overweight and obesity. The report further shares that despite good progress in some countries, the world is off track to reduce and reverse the trend [16]. In the setting of the HIV/AIDS population malnutrition has been shown to worsen the effects of HIV by weakening the immune system and HIV also in turn promoting nutritional deficiencies [17]. A number of studies conducted globally have demonstrated the important role fat plays in health when consumed within the recommended dietary allowance. Overall cholesterol can be used as essential biomarker since lipids have a part in viral entry, uncoating, replication, protein synthesis, assembly, budding and infectivity [18-20]. Further, studies have shown that, cells of the immune system in person with hypercholesterolemia had greater phagocytic activity, added total T cells, more $CD8^+$ T cells, extra circulating lymphocytes, more proliferation and differentiation, more immunoglobulin production, and migration of lymphocytes than from individuals with lower cholesterol levels [21,22].

Polyunsaturated fatty acids on the other hand are considered protective against degenerative pathologies in a balanced ratio of n-6: n-3 close to 1. Many diseases such as; cardiovascular diseases, cancer, diabetes, obesity and metabolic syndrome related disease are associated with high ratio [23]. Therefore, proper amounts of dietary n-6 and n-3 fatty acids should be considered in making dietary choices and recommendations. N-3 fatty acids are known to modulate biomarkers such as C-reactive protein (CRP) and CD4 count which play a key role in the progression of HIV disease and other inflammatory conditions [24,25], and therefore have the potential to enhance clinical outcomes in HIV patients. No important changes in interleukin-6 (IL6), interleukin-1 beta (IL1-beta) and tumor necrosis factor-alpha (TNF-alpha) serum concentrations were observed with fish oil supplements for 12 weeks as revealed in one study by the contrary findings. In that study, the objective was to estimate the result of a low dose of marine omega-3 fatty acids on inflammatory marker concentrations in HIV-infected subjects under antiretroviral treatment [26].

Research design and methods

The study was a cross sectional design in which quantitative methods were referred to determine the relation between independent and outcome variables with the primary goal being to analyze and represent the interaction through statistical analyses. In this analysis, the independent variable is Plasma fat status while the main outcome variable is absolute Opportunistic infection. The target populations were adult HIV/AIDS patients receiving care from the HIV clinic, who qualified and consented to take part in the study. A total of 174 participants who were selected through a simple random sampling technique were recruited and followed up for the study. The Zambian Ministry of Health certified and standardized questionnaires in HIV care were adopted and applied to derive information on history taking, physical assessments and anthropometric measurements. The FacsCount machine was used for CD4 count measurement.

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The baseline characteristics of participants by gender were analyzed using Wilcoxon rank sum assessment for continuous variables and chi-square assessment for categorical variables.

The main research question was addressed by establishing the association between Plasma fat and Opportunistic infections, adjusted for potential confounders using the multiple linear regression model. The statistical software package used to analyze the data was STATA version 12. Ethical approval was sought and granted by the TDRC Research Ethics Committee in Ndola and the National Health Research Authority in Lusaka, Zambia.

Results

The total number of subjects whose results were available for analysis in the study was 174. Of the 174 subjects, 107(61%) were female and 67(39%) were males. The study population were generally young, with males being older [40(37.5,42.4) years] than females [3 (34.5,38.7) years], p=0.02 (Table 1).

Variable	Female N=107(61%)	Male N=67(39%)	P
Age (years)	37(34.5,38.7)	40(37.5,42.4)	0.02
BMI (kg/m²)	22.9(20.4,27.5)	21(18.8,23.9)	0.01
$CD4^+$count (cells/ul)	357(231,543)	245.5(167.5,407.5)	0.002
Viral load (copies/ml)	355(20,6770)	254(23,2694)	0.84
Total cholesterol (mmol/l)	3.86(3.02,4.62)	3.53(3.06,4.61)	0.65
Triglyceride (mmol/l)	1.19(0.87,1.51)	0.96(0.71,1.60)	0.25
LDL-c (mmol/l)	2.31(1.58,2.90)	1.86(1.36,2.80)	0.19
HDL-c (mmol/l)	1.33(1.13,1.51)	1.4(1.21,1.55)	0.15
Opportunistic infection			
Present	15(15.2%)	17(27%)	0.06
Co-morbidity			
Present	14(14.1%)	2(3.23%)	0.02
Smoke status			
Yes	2(3.08%)	9(23.1%)	0.001
Alcohol status			
Yes	16(16.2%)	23(37.1%)	0.003

Table 1: Baseline characteristics. Values are median (interquartile range) unless otherwise stated, BMI: Body Mass Index; $CD4^+$: Cluster Differential; LDL: Low Density Lipoprotein-cholesterol; HDL: High Density Lipoprotein-cholesterol; TC: Total cholesterol.

The plasma fat status revealed a higher median total cholesterol of [3.86(3.02,4.62) mmol/l], median triglyceride [1.19(0.87,1.51) mmol/l] and LDL-c [2.31(1.58,2.90) mmol/l] for females than that for the males [3.53 (3.06,4.61) mmol/l], [0.96(0.71,1.60) mmol/l] and [1.86(1.36,2.80) mmol/l] independently. In contrast, the median HDL-c concentration were higher in the male [1.4(1.21,1.55) mmol/l] than the female gender [1.33(1.13,1.51) mmol/l]. The results per clinical status revealed relatively more males [17(27%)] than females [15(15.2%)], p=0.06 presenting with opportunistic infections. Results in both the overall and by gender (especially male) regression analyses

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suggested a consistent inverse interaction involving most of the plasma fat types and BMI with opportunistic infections [Tables 2 and 3].

Independent variables	Unadjusted estimates (95% CI)	P-value	Adjusted estimates (95% CI)	P-value
tChol2	0.00(-0.05 0.04)	0.88	0.24(-0.50 0.99)	0.52
Trig2	0.04(-0.04 0.12)	0.32	-0.10(-0.31 0.11)	0.34
HDLc2	0.10(-0.24 0.44)	0.58	-0.35(-1.20 0.51)	0.42
LDLc2	-0.01(-0.01 0.04)	0.67	-0.27(-1.01 0.48)	0.48
lnVL2	0.01(-0.01 0.04)	0.19	0.01(-0.02 0.04)	0.64
lnCD4v2	-0.17(-0.25 -0.09)	0	-0.09(-0.21 0.02)	0.11
BMI	-0.01(-0.03 0.00)	0.04	-0.01(-0.03 0.01)	0.45
Age_n	0.00(-0.01 0.00)	0.48	0.00(-0.01 0.01)	0.72
gender	-0.12(-0.25 0.00)	0.06	-0.12(-0.33 0.09)	0.27
SmoSt	-0.10(-0.30 0.10)	0.32	-0.24(-0.57 0.09)	0.15
AlSt	0.04(-0.11 0.19)	0.58	0.12(-0.12 0.36)	0.32

Table 2: Multiple linear regression taking OpIn as the dependent variable. Using multiple linear regression, adjusted for age, gender, BMI, smoke status and alcohol status by visit 2. $CD4^+$: Cluster differentiation 4; tChol: otal Cholesterol (mmol/l); Trig: Triglyceride (mmol/l); LDL: Low density lipoprotein-cholesterol (mmol/l); HDL: High density lipoprotein-cholesterol (mmol/l); VL: Viral load (copies/ml); BMI: Body mass index (kg/m^2); SmoSt: Smoke status; AlSt: Alcohol status; OpIn: Opportunistic infection.

Independent variables	Adjusted estimates (95% CI)	P-value
tChol2	-0.06(-1.12 1.01)	0.91
Trig2	-0.14(-0.60 0.33)	0.55
HDLc2	-1.21(-2.74 0.31)	0.11
LDLc2	0.12(-0.95 1.20)	0.81
lnCD4v2	-0.08(-0.38 0.23)	0.61
lnVL2	-0.01(-0.10 0.07)	0.77
BMI	-0.01(-0.07 0.06)	0.77
Age_n	0.02(-0.01 0.04)	0.13
SmoSt	-0.12(-0.65 0.41)	0.64
AlSt	-0.20(-0.73 0.32)	0.43

Table 3: Multiple linear regression taking OpIn as the dependent variable by male gender. Using multiple linear regression, adjusted for age, BMI, smoke status and alcohol status by visit 2. $CD4^+$: Cluster differentiation 4; tChol: total Cholesterol (mmol/l); Trig: Triglyceride (mmol/l); LDL: Low density lipoprotein-cholesterol (mmol/l); HDL: High density lipoprotein-cholesterol (mmol/l); VL: Viral load (copies/ml); BMI: Body mass index (kg/m^2); SmoSt: Smoke status; AlSt: Alcohol status; OpIn: Opportunistic infection.

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Discussion

Plasma fat status

Findings showing improved plasma fat status from visit 1 to visit 2 are in keeping with revelations from previous studies suggesting increased nutrient uptake associated with enhanced appetite and resolving HIV enteropathy following the initiation of antiretroviral therapy [27]. The mechanisms underscore the synergistic roles of anti-retroviral therapy and plasma fat towards improving the immune system, promoting viral suppression and prevention of opportunistic infections.

The inverse association between plasma fat and opportunistic infections reflects findings in a clinical trial were n-3 unsaturated fatty acid supplementation was related with decreasing viral load trend in the beneficiaries and an increase in the control group [28].

Similarly, polyunsaturated fatty acids have been shown to enhance humoral immune responses [29] and existence in mice [30]. In vitro, n-6 fatty acids such as arachidonic acid confer resistance via infection by both bacteria and viruses [31]. In an in vitro study beta-estradiol has been shown to inhibit HIV-1 replication in human peripheral blood lymphocytes [20,32], inhibiting target cell infection that involves cell-entry through higher expression of chemokines [33]. Studies have further revealed that hypocholesterolemia impairs HAART effectiveness in HIV infected patients [34].

Regression of plasma fat with opportunistic infections

There was no statistically significant association between plasma fat types and opportunistic infections. However, in both the overall and the gender-based regression analyses, our study revealed a consistent inverse association involving most of the plasma fat types with opportunistic infections. This finding may suggest a potential role of plasma fat or BMI in immune mechanisms or viral suppression and corresponding clinical outcomes, a subject for further exploration.

Study limitations

Due to attrition following the lost to follow-up of some subjects by visit 2, the power of the study could potentially be limited to detect a statistically significant association for the interaction where significance may not have been suggested. The study was potentially prone to reporting bias which may arise from failure by respondents to report the truth. Recall bias could also have resulted from failure to recall or report clearly, responses toward the clinical assessments. The study could also be prone to measurement error which may arise during the testing process of the samples for the variables of interest.

Conclusion

Plasma fat status was not significantly predictive of the prevalence of opportunistic infections. However, there was an overall and by gender consistent inverse association reported between plasma fat and opportunistic infections, which finding may suggest a potential role of plasma fat in immune mechanisms or

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viral suppression and corresponding clinical outcomes.

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Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of the manuscript.

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Author contributions

The concept, design, data acquisition, analyses and interpretation of study findings were conducted by Christopher Nyirenda. All coauthors contributed towards the content, review and ultimate write-up of the manuscript.

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