Correlation of Viral Load to Intestinal Parasitosis in HIV Seropositive Patients Attending U.I.T.H., Ilorin

[1]AA Joseph and Ano-Edward GH[2]

1. Department of Medical Microbiology and Parasitology, College of health sciences, Bowen University, Iwo, Nigeria
2. Department of Anatomic Pathology, College of health sciences, Bowen University, Iwo, Nigeria

Abstract

Background: *Intestinal parasitosis is endemic in sub-Saharan Africa. Many researchers have defined the spectrum of intestinal parasites in HIV seropositive Nigerians at various levels of CD4+ counts.There is paucity of information on the relationship between intestinal parasitosis and viral load among Nigerians living with HIV/AIDS.*

Objective: *To correlate the pattern and density of intestinal parasitosis in HIV seropositive individuals, to the degree of their viraemia.*

Methodology: *A descriptive, cross-sectional study involving 500 participants comprising 250 HIV positive patients as test and 250 HIV negative patients as control. Participants were from the Highly Active Antiretroviral Therapy (HAART) clinic and GOPD of University of Ilorin Teaching Hospital (UITH), Ilorin. Collected blood and stool samples were screened for HIV infection, CD4+ cell count and HIV viral load estimation and gastrointestinal parasites.*

Result: *Intestinal parasitosis of 60.8% in HIV positive patients was significantly higher than 16.4% in HIV negative controls ($p< 0.05$). Single intestinal parasitosis is commoner (48.8%) than multiple parasitosis (12.0%) in the HIV positive test group. Parasites identified from test subjects were Ascaris lumbricoides (10.4%), Hookworm (3.6%), Strongyloides stercoralis larva (2%) and the Coccidian parasites (55.6%). The mean CD4+ count of the HIV patients was 232.1±189.3 (range 1-882 cells/µl) while the mean viral load was 372,306±824150.7 (range 20 – 7,369,327 copies/ml). Intestinal parasitosis was seen at CD4 count $\leq$ 207cells/µl and a viral load >7085 copies/ml.*

Conclusion: *In this study, intestinal parasitosis occurs in HIV infected with markedly depressed immunity evident by a low CD4+ cell count and high viral load.*

Keywords: HAART, HIV, viraemia, intestinal parasitosis

Correspondence to: Dr Joseph AA, Department of Medical Microbiology and Parasitology, College of Health Sciences, Bowen University Teaching Hospital, Ogbomoso, Oyo State.
Email: adejokejoseph2012@gmail.com

No conflicts of interest have been declared by the authors

Introduction

Intestinal parasitic infections are among the most common infections worldwide. It is estimated that some 3.5 billion people around the world are affected.[1] Parasitic infection of all types remain a common cause of morbidity and mortality in populations living in tropical and subtropical regions of the world, suggesting that anti-parasitic immunity is inefficient in these regions[2]. However when immune system is severely depressed as seen in HIV infection, opportunistic parasitic infections become more frequent and can be life threatening implying that in immunologically normal individuals, prevalence and severity of infection are well controlled.[2]

The prevalence and burden of intestinal parasitic infections remain high in developing countries as a result of inadequate safe water supply, poor environmental hygiene and sanitation.[3,4] Intestinal parasites are frequently transmitted by unhygienic habit such as direct transfer of ova or cysts from anal region to mouth, eating with unwashed hands or eating and drinking of contaminated food and drink.[3]

HIV has become one of the world's most serious health challenges.[5] Nearly 30 million people have died of AIDS-related causes and there are approximately 34 million people currently living with HIV[5]. While cases have been reported in all regions of the world, 97% of those living with HIV reside in sub-Saharan Africa.[5.] One of the major health problems among HIV seropositive patients is superimposed infection due to the defect of immunity. Intestinal parasitic infection, which is also one of the health problems in sub-Saharan Africa, is common in this patients.[6] Harms and Feldmeier postulated that HIV and parasitic infections may interact and mutually affect one another. Although the natural history of parasitic disease may be altered by co-infection with HIV, parasitosis may in turn facilitate the progression from asymptomatic infection to AIDS.[7]

Gastrointestinal involvement in HIV/AIDS is universal with significant disease occurring in 50.0- 90.0% of patients. Several species of protozoa have been associated with gastrointestinal involvement in HIV diseases.[8] The prevalence of intestinal parasitosis of 87.8% among HIV seropositives and 74.0% among seronegative controls were reported in Ilorin,[9] 23.3% among HIV positives and 33.8% in HIV negatives in Lagos[10], and 24.7% in HIV positives in Abuja[11].

Reports from Nigeria have described the relationship between HIV and intestinal parasites especially the opportunistic intestinal coccidian parasites such as *Cryptosporidium, Cyclospora* and *Isospora* species.[12,13] These studies however have limited information on the influence of the level of viraemia on the types of intestinal parasites present and on the parasite density. Thus, we set out to define the spectrum of gastrointestinal parasites in HIV infected individuals at various levels of viraemia in patients attending HAART Clinic of the UITH, Ilorin.

Objective: To correlate intestinal parasite density to CD4+ cell count and the viral load of HIV seropositives attending the HAART clinic of UITH, Ilorin, Nigeria.

Materials and Methods

Study area and study design

The study was a descriptive cross-sectional, hospital-based study in which treatment naïve HIV positive patients were used as test participants, while control participants were HIV negative. Self and interviewer administered questionnaire were filled for all subjects at recruitment. Venous blood sample and stool samples were collected from participants at the HAART Clinic of UITH, Ilorin, Nigeria. The total number of enrollee as at time of study was 4431 (1383 adult males, 2674 adult females, 179 pediatric males and 195 pediatric female patients). Of the adult enrollees, 1241 have been commenced on antiretroviral drug.

Subject selection

Study participants consisted of newly diagnosed HIV positive individuals yet to commence HAART (treatment naiive). Purposive sampling was done to select participants into the study. An interviewer-administered questionnaire was completed at recruitment for all participants and stool and venous blood samples were also collected from every selected participant. HIV seropositive patients who have been commenced on HAART and HAART-naïve HIV positives who have being on antimicrobials in the last two (2) weeks prior to sample collection were exempted from the study. Equal number of apparently healthy age and sex matched patients who were HIV seronegative and attending the GOPD of the hospital were recruited as control.

Laboratory Procedure

Specimen procedure

Stool was collected in a clean, wide mouthed container with tight-fitting lid and about 12 mls of venous blood was collected aseptically with the use of a vacutainer needle into three (3) Ethylene DiaminoTetra-Acetic Acid (EDTA) containers, 4mls in each bottle.

Specimen processing

Each stool sample was first examined macroscopically and the findings noted. It was then divided into three (3) parts; one part was processed fresh, one part preserved in 10% formalin and the third part preserved in absolute ethanol. From the unpreserved part, direct smears were made for saline and iodine wet preparation. Formalin-preserved portion was concentrated using the Formol Ether sedimentation method while the third part was stored frozen for future use. From the concentrated stool sample, three (3) smears were made on three separate glass slides for Modified Ziehl-Neelsen stain, Trichrome staining and Fluorochrome staining using the Auramine stain. Intestinal parasites were identified using diagrams in the image library of Division of Parasitic Diseases of Center for Disease Control and Prevention, USA (DPDx CD). A positive result by any two of the three staining techniques is considered as truly positive. Where oocysts are seen, it was reported as positive. Parasite density was estimated for Coccidian parasites using the guide below, adapted from reporting Ziehl Neelsen staining for acid fast bacilli[71]:

3+ if >10 oocysts were seen per High Power Field.

2+ if 1-10 oocysts were seen per High Power Field.

1+ if 10-100 oocysts were seen per 100 High Power Fields.

Scanty if 1-9 oocysts were seen per 100 High Power Fields.

CD4+ count was estimated from whole blood within 6 hours of collection while plasma, prepared by spinning the anticoagulated blood in a centrifuge was kept frozen at-70^{o}C till analysis for viral load. Estimating the number of HIV RNA copies was done using COBAS AmpliPrep/COBAS® TaqMan®HIV-1 Test, version 2.0, the result of which was expressed in copies/ml[25]

Data Analysis

Clinical findings and laboratory results were recorded in the study questionnaire. The entered data were analyzed using the Statistical Package for Social Sciences (SPSS) Version 19, licensed to IBM Company since 2009. Results were presented in tabular forms and figures as found applicable. Categorical variables were compared by Chi square test and continuous variables were described by means ± SD and compared by the Student's T-test. Statistical significance was tested at predetermined p-value of <0.05.

Other statistical tests used were Spearman Correlation; Receiver Operating characteristic (ROC) curve which was drawn using the MedCalc® software and used to determine a cutoff value for the viral load and CD4+ cell count and Analysis of Variance (ANOVA) to test the differences between group means for statistical significance. Least Significant Difference (LSD) test was used when ANOVA is statistically significant to determine the smallest significant result between two means, enabling direct comparism of two means from two groups. Any difference larger than the LSD was considered significant. Pearson Correlation which measures the strength of a linear association between two variables was also used. The Pearson Correlation Coefficient ranges from + 1 to -1, a value of 0 indicating no association, >0 indicating positive association,< 0 indicating negative association. Positive association denotes that both variables are increasing. Results were presented in tabular forms and figures as found applicable.

Results

Sociodemographic characteristics of respondents

A total of 500 participants comprising of 250 HIV positive (test group) and 250 HIV negative (control group) were recruited for the study. The sociodemographic characteristics of the

Table 1: Socio-demographic variables of study population

Socio–demographic	Subject N (%)	Control N (%)
Mean age ± SD	38.8 ± 11.3	39.4 ± 12.8
Age group		
15 – 24	18 (7.2)	24 (9.6)
25 – 34	81 (32.4)	77 (30.8)
35 – 44	86 (34.4)	70 (28.0)
45 – 54	37 (14.8)	43 (17.2)
55 – 64	21 (8.4)	26 (10.4)
≥ 65	7 (2.8)	10 (4.0)
Sex		
Male	102 (40.8)	117 (46.8)
Female	148 (59.2)	133 (53.2)
Level of Education		
Primary	41 (16.4)	73 (29.2)
Secondary	110 (44.0)	74 (29.6)
Tertiary	87 (34.8)	55 (22.0)
Others	12 (4.8)	48 (19.2)
Religion		
Islam	154 (61.6)	170 (68.0)
Christianity	96 (38.4)	77 (30.8)
Others	0 (0.0)	3 (1.2)
Tribe		
Yoruba	180 (72.0)	185 (74.0)
Hausa	18 (7.2)	22 (8.8)
Igbo	25 (10.0)	26 (10.4)
Others	27 (10.8)	17 (6.8)

participants as presented in table1 shows that the age ranged from 15 to above 65 years, with a mean age of 38.8±11.3 years for test group and 39.4±12.8 years for the control group.Median age for test group was 37years, 37.5 years for control group and 37years for the total population.

Prevalence of Intestinal Parasitosis and the Pattern

Gastrointestinal parasitosis was reported in One hundred and fifty-two (60.8%) HIV positive test subjects and 41 (16.4%) HIV negative controls (Table 2). One hundred and twenty-two (48.8%) of these test group with parasitosis had a single form of intestinal parasitosis while 30 (12.0%) had multiple intestinal parasitosis. Only one (1) participant in the control group had more than one type of parasite present. Eighty-three point six percent (83.6%) of the HIV negative controls had no parasites present.

Spectrum of parasite identified in the test group with intestinal parasitosis included *Ascaris lumbricoides* (10.4%), Hookworm (3.6%), *Strongyloides stercoralis* (2%), *Cryptosporidium parvum* (33.6%), *Cyclospora cayetanensis* (20.8%) and *Isospora belli* (1.2%). Similar parasites were seen in the control group though at differing frequency. Parasites identified in the controls

Table2: Prevalence of single and multiple intestinal parasitosis

HIV Patients	Subject N (%)	Control N (%)	x^2	p Value
Parasite Present	152 (60.8)	41 (16.4)	63.839	< 0.001*
Single parasite	*122 (48.8)*	*40 (16.0)*	*41.506*	*< 0.001**
Multiple parasite	*30 (12.0)*	*1 (0.4)*	*27.129*	*<0.001**
Parasite Absent	98 (39.2)	209 (83.6)	40.134	< 0.001*

*x^2: Chi square; * P value less than 0.05 (i.e. statistically significant)*

Table 3: Prevalence of intestinal parasitosis in HIV positives

Parasite	Subject N (%)	Control N (%)	x^2	p Value
Ascaris lumbricoides	26 (10.4)	15 (6.0)	2.951	0.086
Balatidium coli	0 (0.0)	1 (0.4)	0.000	1.000^Y
Entamoeba histolytica	0 (0.0)	1 (0.4)	0.000	1.000^Y
Fasciola buski	0 (0.0)	1 (0.4)	0.000	1.000^Y
Hook worm	9 (3.6)	6 (2.4)	0.600	0.439
Schistosoma mansoni	0 (0.0)	1 (0.4)	0.000	1.000^Y
Strongyloides stercoralis	5 (2.0)	0 (0.0)	3.200	0.074^Y
Coccidian	139 (55.6)	17 (6.8)	95.410	0.001*
Cryptosporidium	84 (33.6)	11 (4.4)	56.095	<0.001*
Cyclospora	52 (20.8)	5 (2.0)	38.754	<0.001*
Isospora	3 (1.2)	1 (0.4)	0.250	0.617^Y
No parasite	98 (39.2)	209 (83.6)	40.134	<0.001*

***NB:** Multiple parasites present in some patients; x^2: Chi square; Y: Yates' chi square (when 20% of expected count is less than 5); * P value less than 0.05 (i.e. statistically significant)*

were *Ascaris lumbricoides* (6.0%), *Balantidium coli* (0.4%), hookworm (2.4%), *Schistosoma mansoni* (0.4%), *Cryptosporidium parvum* (4.4%), *Cyclospora cayetanensis* (2.0%), *Isospora belli* (0.4%). *Strongyloides stercoralis* was not seen in any control subject (Table 3).

Table 4: Parasite density

0	135 (35.8)	242 (64.2)	377 (100.0)		
1+	78 (92.9)	6 (7.1)	84 (100.0)		
2+	17 (100.0)	0 (0.0)	17 (100.0)		
3+	12 (100.0)	0 (0.0)	12 (100.0)		
Scanty	8 (80.0)	2 (20.0)	10 (100.0)	124.638	0.001

Parasite Density

One hundred and thirty-five (35.8%) test subjects and 242(64.2%) control respondent had a parasite density of 0, 8(80%) of test and 2(20%) of control had scanty parasites, 78(92.9%) of test subjects and 6(7.1%) of control subjects had a density of 1+ while 17(100%) and 12(100%) of test subjects had a density of 2+ and 3+ respectively. (Table 4)

Mean CD4+ / Viral Load

The range of CD4+ T cell count of test group is 1 to 882 cells/mm^3, with a median count of 202.5cells/mm^3. One hundred and nineteen HIV positive subjects (47.6%) had a CD4+

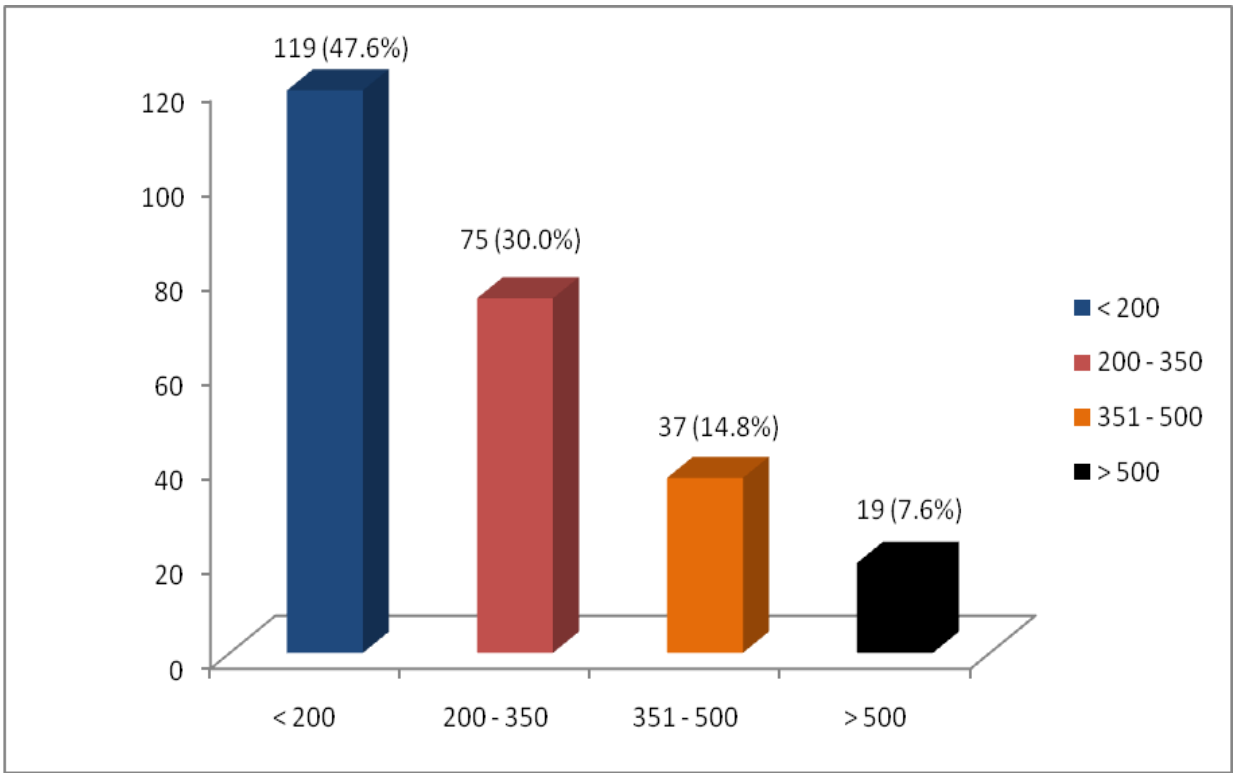

Fig. 1: CD4+ CELL Count Range of HIV Positive Participants

count of less than 200cells/mm^{3}while 19 (7.6%) had a count greater than 500 cells/mm^3(Figure 1).Viral load of subjects ranged between 20 and 7,369,327copies/ml with a median value of 70523.0 copies/ml. 43(18.8%) of respondent had a viral load of <1000 copies/ml, 40(16.0%) had a load of between 1000-10,000, 48(19.2%) had a load > 10,000 but less than 100,000 while 115(46.0%) had a load of > 100,000 copies/ml.(Figure 2)

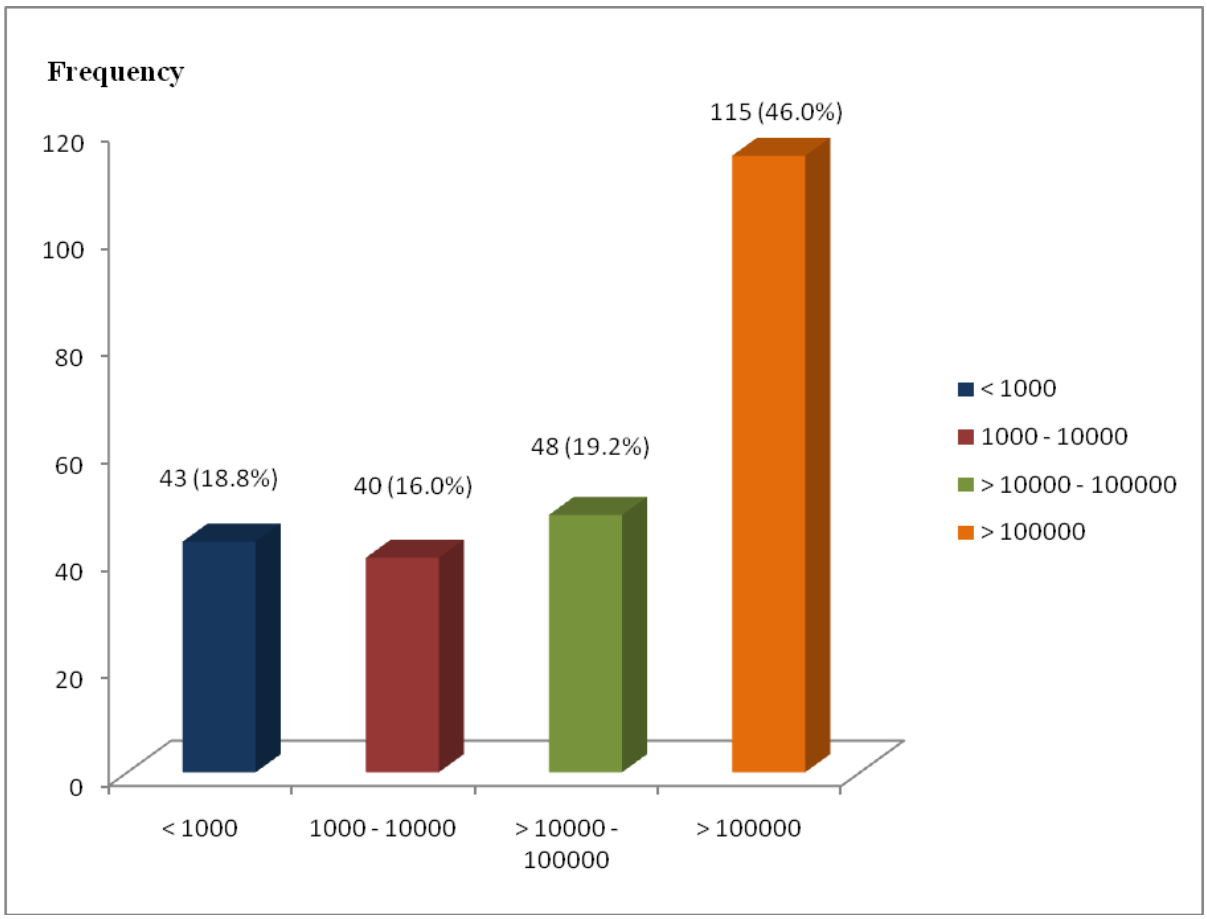

Fig. 2: Viral load range of HIV positive participants

Relating the Mean CD4 T+ Cell Count to Parasite Density

Table 5 presents the mean CD 4+ count at various parasite densities. An inverse relationship was noted between the CD4 cell count and the parasite density that is as the density increases, the count decreases. Receiver Operational Curve (ROC) drawn to relate CD 4+ T cell count and intestinal parasitosis shows that generally, intestinal parasitosis occurs at CD4+ cell count of 207cells/µl, while parasitosis with the coccidian group of parasites occurs at CD4+ cell count of 148 cells/µl.

Correlation of Viral Load to Parasite Density

Table 6 presents the mean viral load at various parasite densities.A directly proportional relationship was noted, with increasing parasite densities as the viral load increases. Receiver

Table 5: Correlating the mean CD4+ count to parasite density

Parasite Density	CD4+ Count				
	N(%)	Mean ±SD	F	R	P
0	135 (54)	281.68 ± 202.81[a]			
Scanty	8 (3.2)	180.00 ± 118.04			
1+	78 (31.2)	194.58 ± 166.78[b]			
2+	17 (6.8)	139.41 ± 116.99[cd]			
3+	12 (4.8)	83.83 ± 82.70[c]	6.648	-0.326	0.001*

*F: ANOVA (Analysis of Variance); *: p value is < 0.05 (i.e. statistically significant); Means with different alphabets indicates significant difference using the Least Significant Difference (LSD), (N.B: Means without alphabets are not significantly different from those with alphabets)*
R: Spearman coefficient of correlation

Table 6: Correlating the mean viral load to parasite density

Parasite Density	Viral Load				
	N(%)	Mean ($x10^5$) ± SD	F	R	p value
0	135 (54.0)	202465.12 ± 597789.37[a]			
Scanty	8 (3.2)	311378.63 ± 370658.16			
1+	78 (31.2)	589594.01 ± 997396.60[b]			
2+	17 (6.8)	771280.94 ± 315602.97			
3+	12 (4.8)	928939.67 ± 654763.06[b]	4.413	0.440	0.002*

*F: ANOVA (Analysis of Variance); *: P value is < 0.05 (i.e. statistically significant); Means with different alphabets indicates significant difference using the Least Significant Difference (LSD), (N.B: Means without alphabets are not significantly different from those with alphabets)*
*R: Spearman coefficient of correlation; *: P value < 0.05 (i.e. statistically significant)*

Table 7: Correlation of CD4+ T cell count with viral load

Tests	**Mean ± SD**	**R**	**p value**
Viral load	372,306.0x10^5 ± 824,150.7	**- 0.214**	**0.001***
CD4 count	232.1 ± 189.3		

*R: Pearson coefficient of correlation; *: P value < 0.05 (i.e. statistically significant)*

Operational Curve (ROC) for viral load and intestinal parasitosis shows that the lowest viral load at which intestinal parasitosis occurswas 7085copies/ml whileparasitosis with the coccidian parasites occursat a viral load level of 14, 565copies/ml and above.

Correlation of CD4+ Count to Viral Load

CD4+T cell count and viral load were correlated using Pearson's correlation (Table 7). An inverse correlation between these two variables was noted, that is, an increase in viral load results in a decrease CD4+ T cell count.

Table 8: Factors associated with intestinal parasiosis

	Univariate Analysis			Multivariate Analsis		
Variable	**B**	***p* value**	**OR (95% CI)**	**B**	***p* value**	**OR (95% CI)**
Age	-0.005	0.480	0.995 (0.98-1.01)	NA		
Sex	0.016	0.931	1.016 (0.71-1.46)	NA		
Level of Education						
Primary	0.129	0.836	1.14 (0.34-3.86)	NA		
Secondary	0.239	0.536	1.270 (0.60-2.71)	NA		
Tertiary	0.431	0.143	1.539 (0.87-2.74)	NA		
Religion						
Islam	0.163	0.895	1.176 (0.11-3.11)	NA		
Christianity	0.355	0.774	1.426 (0.13-6.02)	NA		
Tribe						
Yoruba	0.395	0.256	1.48 (0.75-2.93)	NA		
Hausa	0.567	0.218	1.763 (0.72-4.31)	NA		
Igbo	0.672	0.121	1.959 (0.84-4.59)	NA		
CD4count	-0.003	0.000*	1.0 (0.996-0.999)	-0.002	0.002*	0.998(0.996-0.999)
Viral load	0.001	0.007*	1.001(1.00-1.002)	0.001	0.033*	1.001(1.000-1.002)

*B: Coefficient of binary logistic regression; OR(95% CI): Odds ratio (95% Confidence Interval); *: p value < 0.05 (statistically significant)*

Factors Associated with Intestinal Parasitosis

Table 8 represents the various factors influencing presence of gastrointestinal parasitosis in HIV positives. Using univariate and multivariate analyses, age, sex, religion, level of education, and tribe had no significant influence on the presence or otherwise of intestinal parasites in patients. CD4 count and viral load were however found to have statistically significant influence on presence of intestinal parasitosis, with CD4 cell count having a greater influence.

Discussion

Gastrointestinal parasite infection is common in the tropics and Ilorin is not an exception. The prevalence of intestinal parasitosis in HIV positives and negative control reported in this study is 60.8% and 16.4% respectively, lower

than the report by Babatunde and Salami in Ilorin in 2010 where a rate of 87.8% in HIV positives and 74% in HIV negatives was reported.[9] The differences in rate between the two studies may be due to level of immunosuppression between the two groups of patients, high sensitivity but low specificity and negative predictive value of the technique used in the previous study, where yeast and other spherical objects staining red can be difficult to discriminate from acid fast oocysts.[15] Compared with HIV negative control, these values are statistically significant ($p<0.001$).

Polyparasitism, was reported in 31 participants, 30 of which were HIV positive patients with CD4+ count below 200 cells/mm^3. This was also noted in a previous study in Ilorin by Babatunde *et al.*[9] The severely depressed immunity at this level of CD4 count could be the reason for this, particularly as this situation reduces the capacity of the patient's defense system to clear the body of unwanted parasitic colonization.[16]Spectrum of parasites reported in both test and controls was similar, though at differing frequencies. Exception to this is the larva of *Strongyloides stercoralis* which was reported only in test group, and *Balantidium coli*, *Entamoeba histolytica*, *Schistosoma mansoni* reported only in control subjects. The prevalence for each of the reported parasites was higher in HIV positives than negatives, probably due to the defective T cell mediated immunity seen in HIV infection. The most prevalent parasite reported in this study was the coccidian group of parasites (55.6%), *Cryptosporidium parvum*, *Isospora belli* and *Cyclospora cayetanensis*, followed by *Ascaris lumbricoides* (10.4%), and Hookworm (3.6%). Looking at the spectrum of parasites seen, there was no statistically significant difference between subjects and control when protozoa parasites are concerned ($p>0.05$) with the exception of the coccidian parasites whose prevalence was significantly different between test and control ($p=0.001$). This is in keeping with previous studies done at UITH, Ilorin[9].

This study also reported *Cryptosporidium parvum* in healthy controls (6.8%), consistent with the finding of Ikeh *et al*.[17] In support of this finding is the fact that *Cryptosporidium parvum* has been found to contaminate drinking water and food and also transmitted via contact with infected animal. Even though associated with diarrhea in immunocompromised patients, it has been reported in immunocompetent hosts as well.[18,19]

The CD4+ T cell count of subjects ranged between 1 and 882 with a median of 202.5cells/µl with the majority having a count less than 200 cells/µl as at time of first presentation at the clinic. Adult apparently healthy residents of Ilorin have been seen to have significantly lower absolute mean CD4+ count compared to the national reference value.[19]This could explain the extremely low counts in some patients at presentation. HIV positive patients in this environment also tend to seek medical attention late as indicated by the low mean CD4 cell count at presentation. Probable factors which may be likely contributors to delayed presentation at healthcare facility, though scientifically unproven, may include financial constraint, ignorance, cultural practices and belief, attendance of religious homes, and fear of stigmatization . Compared with resource rich countries like United States and Canada where mean entry CD4 count is 336 cells/mm^3, this CD4+ cell count at entry into care is very low, referred to as a late entry.[20]

CD4+ count at which intestinal parasitosis was first reported in this study was 207cells/µl; coccidian parasites were seen at a CD4+ count of 148 cells/µl and lower. This is similar to the finding in India where coccidian parasites were seen at a CD4 count of 186cells/mm^3.[21] An inverse relationship was noted between the CD4 count and parasite density ($R= - 0.269$, $p= 0.002$). The mean CD4 count at which no parasite(0) was seen and at which Scanty parasites were seen is not significantly different.

Mean CD4 at which a density of 0 (no parasite) was seen was however significantly different from 1+, 2+, 3+ density. With worsening immunosuppression evident by decreasing CD4+ count, parasite density was increasing. This explains the role played by Cell Mediated Immunity in host defense against intestinal parasites and the rate of clearance in intestinal parasitic colonization. Heavy parasite load and delayed clearance of parasite in individual with HIV induced immunosuppression is usual, due to defective CMI.[22] For the viral load however, there was a direct proportionality between the load and parasite density and at viral load of 7,085copies/ml and above, intestinal parasitosis was reported. Coccidian parasites appear later in the disease than other parasites, seen at a viral load of 14,525copies/ml and above. There is no documented value in this environment to compare this finding with, but was found to be in deviant to what was reported in a study carried out in Brazil where no clear association between the level of the absolute CD4+ count or the viral load and a specific parasitic infection was established.[23]

Factors predisposing to parasitic infection includes level of hygiene, poor nutrition, lack of toilet facilities in the home and lack of portable water.[24] factors identified to be responsible for the continued persistence of intestinal parasites infection include poor sanitary conditions, unhygienic practices, absence of portable water, poor housing and poverty.[25,26] In this study however, these factors did not significantly influence the presence of intestinal parasites in HIV positives compared to their HIV negative controls (p=0.986; 0.156).This is probably due to the fact that diarrhea in HIV patients occurs irrespective of the geographical location of the individual and has been said to be due to alteration in structure and function of enterocytes caused by the pathogen (HIV).[27]

Sociodemographic variables such as age, sex, level of education, religion and tribe also did not influence the presence of intestinal parasites in HIV positives in this study. This was however in deviant with what was reported in a study carried out in Benin City, Nigeria, where male gender, level of education, and source of water were significant influences on prevalence of intestinal parasitosis.[28] Identified factors associated with intestinal parasitosis in this study were hence CD4+ count and viral load. On multivariate analysis of the influence of both variables, CD4 count was found to have greater influence on the presence or otherwise of intestinal parasites (p= 0.002) compared to viral load (p=0.033). This probably explains the concept of discordant viral load and CD4+, mismatch between an undetectable viral load and the absence of immune reconstitution evidenced by a low CD4 count which can be confusing to both patient and healthcare provider. This finding is also a confirmation that CD4 count is the strongest predictor of disease progression, survival, and risk of death.

In conclusion, this study has shown a high prevalence of intestinal parasitosis in HIV infected adults with a low CD4 + count and high level of HIV viraemia. The type of parasites reported in the seropositives were similar to those found in seronegative controls, the prevalence however differs, being higher in the HIV positives. The coccidian group of parasites accounts for the majority of cases. Polyparasitism was also commoner in the HIV seropositive. Identified factors influencing the presence of parasitosis were the CD4 cell count and viral load. Level of education and other socioeconomic factors did not significantly influence the presence of parasites in subjects. Gastrointestinal parasitosis in the HIV seropositive is hence a function of reduced or reducing immune status of the patient.

We recommend that screening for parasitic infection and viral load estimation should form part of the baseline investigations when enrolling clients into HIV care program and antiparasitic agents should be commenced prophylactically at CD4+ cell count less than 207 cells/µl and at viral load less than 7,085 copies/ml. Antiparasitic agent should also be included in the treatment of HAART- naïve patients with diarrhea.

References

1. World Health Organization. Control of tropical diseases. World Health Organization Geneva, 1998; 1: 1-5
2. Wakelin D. Parasites and the immune system. Bioscience 1997; 47(1):32-40.
3. Adegbola R.A., Demba E.., Deveer G and Todd F. Cryptosporidium infection in Gambian children less than 5 years of age. J Trop Med Hyg. 1994; 97:103-107.
4. Cegilski J.B., Ortega Y.R., McKee S., *et al.*Cryptosporidium, Enterocytozoon, and Cyclospora infection in paediatric and adult patients with diarrhoea in Tanzania. C. Infectious Disease 1999; 28: 314-321.
5. The Henry J. Kaiser Family Foundation: The Global HIV/AIDS Epidemic. UNAIDS. Fact Sheet: The Global AIDS Epidemic; 2012;1:1-3. Available at http://kff.org/global-healthpolicy/factsheet/theglobalhivaids-epidemic. Accessed 05.03.13.
6. UNAIDS. Epidemiological Core Slides-GAP report: Global Summary of the AIDS Epidemic; 2012.
7. Harms G. Feldmeier H. HIV Infection and Tropical Parasitic Diseases: Deleterious interactions in both directions. Trop Med Int Health 2002; 7: 479-488.
8. Veazey RS, DeMaria M, Chalifoux LV, Shvetz DE, Pauley DR, Knight HL,*et. al.*Gastrointestinal tract as a major site of CD4+ T cell depletion and viral replication in SIV infection. Science. 1998; 280(5362): 427-431.
9. Babatunde SK., Salami AK., Fabiyi JP., Agbede OO and Desalu OO. Prevalence of intestinal parasitic infestation in HIV seropositive and seronegative patients in Ilorin, Nigeria. Ann Afr Med 2010; 9: 123-128.
10. Sanyaolu AO,Oyinbo WA.,Fagbenro-Beyioku AF, Gbadagesin AH and Iriemenam NC. Comparative study of entero-parasitic infections among HIV sero-positive and sero-negative patients in Lagos, Nigeria.Acta Trop. 2011; 120(3): 268-272.
11. Abaver DT,Nwobegahay JM, Goon DT, Iweriebor BC and Anye DN. Prevalence of Intestinal Parasitic Infection among HIV/AIDS patient from two health institutions in Abuja,Nigeria. Afr Health Sci. 2011; 11(1): 24-27.
12. Okodua M, Adeyeba OA,Tatfeng YM, and Okpala HO. Age and Sex Distribution of Intestinal Parasitic Infection Among HIV Infected in Abeokuta, Nigeria. Online Journal of Health and Allied Sciences. 2003; 2 (4): 1-2
13. Guptal S., Narang S., Nunavath V and Singh, S. Chronic Diarrhea in HIV Patients: Prevalence of Coccidian Parasites. Indian Journal of Medical Microbiology. 26(1): 172-175.
14. Araoye M,. Research Methodology and Biostatistics for students and health sciences, 1st edition, 2001;6:115-123
15. Khurana S,Sharma P.,Sharma A and Malla N.Evaluation of Ziehl Neelsen staining, Auramine Phenol staining, Antigen detection enzyme linked immunosorbent assay and polymerase chain reaction, for the diagnosis of intestinal cryptosporidiosis. Trop Parasitol. 2012; 2: 20-23.
16. Rodrigues V., Cordeiro-da-Silva A, Laforge M, Ouaissi A, Akharid K,et al. impairment of Tcell function in parasitic infections.2014.PLoS Negl Trop Dis,8(2): e2567.
17. Ikeh EI, Obadofin MO,Brindeiro B and Baugher,C. Intestinal Parasitism in Magama Gumau rural village and Jos township in North Central Nigeria. Nigerian Postgraduate Medical Journal.2007; 14(4): 290-295
18. Ikeh EI, Obadofin MO, Brindeiro B; Baugher C, Frost F, Vanderjaght D and Glead R. Intestinal Parasitism in rural and urban areas of north central Nigeria: an update. Internet journal of microbiology. 2005. 2,(1): 1-3

19. Afolabi JK., Fadeyi A., Desalu OO, Durotoye IA, Fawibe AE, Adeboye MA., *et al.* Normal CD4 Count Range among Healthy Nigerian Population in Ilorin. J Int Assoc Provid AIDS Care .2014. Available at http://www.ncbi.nlm.nih.gov/pubmed/24842948. Accessed on 30.06.14
20. Micheal Carter. CD4 cell count at time of entry to HIV care did not increase significantly between 1992 and 2011.Major implications for debates about treatment as prevention and when to start therapy. Available at www.aidsmap.com/page/2726275. Accessed on 01.08.14.
21. Gupta S, Narang S, Nunavath V and Singh S. Chronic diarrhea in HIV patients: prevalence of coccidian parasites. Indian J Med. Micobiol, 2008; 26: 172-175.
22. Zelalem Teklemariam, Degu Abate, Habtamu Mitiku, and Yadeta Dessie, "Prevalence of Intestinal Parasitic Infection among HIV Positive Persons Who Are Naive and on Antiretroviral Treatment in Hiwot Fana Specialized University Hospital, Eastern Ethiopia," ISRN AIDS, 2013, Article ID 324329, 6 pages, 2013. doi:10.1155/2013/324329.
23. FEITOSA, Giovana *et al.* High prevalence of giardiasis and strongyloidiasis among HIV-infected patients in Bahia, Brazil.Braz J Infect Dis, 2001; 5.6.Available from http://www.scielo.br/scielo.php. Accessed on 02.02.15.
24. Jean R., Christopher B.,Ingrid A.,Corey C., Eliane L.,Clarke J., *et al.* Discordant CD4+ cell count and viral load responses to antiretroviral therapy. 2004. HIV web study case-based modules.
25. Akinsegun A, Adedoyin D and Adewumi A.CD4 count pattern and demographic distribution of treatment-naïve HIV patients in Lagos, Nigeria. AIDS Research and Treatment, 2012. 2012, article ID 352753:6.
26. Fredrick Olusegun Akinbo. Prevalence of intestinal parasitic infections among HIV positive patients in Benin City, Nigeria.Libyan Journal of Medicine, 5(0), 2010; 1-6
27. Edungbola D and Obi A. A review of human intestinal parasites in Nigeria; challenges and prospects for integrated control. Nigerian Journal of Parasitology, 1992. 13: 27 – 37.
28. WHO. Action for the control of soil transmitted helminthiasis in Nigeria. Proceeding of an International workshop on strategies for the control of soil transmitted helminthiasis in Nigeria. Ile-Ife, Nigeria 7th- 9th May, 1991