CD4⁺ T Cell Response to Lamivudine, Stavudine and Nevirapine in *Human Immunodeficiency Virus* Infected Antiretroviral-Naive Men in Nigeria

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Abstract Increase of ($\geq$) 50 CD4⁺ T cells/ μ l in post-commencement of highly active antiretroviral therapy (HAART) is acceptable as indicator of therapeutic success (TS). We therefore hypothesized that median change in CD4 count of the TS and therapeutic failure (TF) groups were comparable after 3 months; and that no associations existed between HAART outcome and adherence to therapy. One hundred *Human immunodeficiency virus* (HIV) infected antiretroviral (ARV) naive men on lamivudine + stavudine + nevirapine at Federal Medical Centre (FMC), Lokoja, Kogi State, Nigeria were studied. Data of the men were obtained with interviewer-administered questionnaire forms. Their ethylene diamine tetra acetic acid (EDTA)-treated whole blood samples were analysed with Partec CyFlow[®] Counter for pre-HAART and follow-up CD4 counts. Adherence to the ARV regimen was recorded for each patient as self-reported. We used Mann–Whitney U test, Kruskal–Wallis, Wilcoxon’s matched pair and CHI² statistical tests for analyses. Overall adherence rate was 95.0%. Though the median follow-up CD4 count was higher ($P = 0.001$) than the pre-HAART value; only 85% of the men attained TS (increase of ≥ 50 cells/ μ l) at follow-up. Median change in CD4 count (+104.0 cells/ μ l; $n = 85$) of the TS was higher ($P = 0.001$) than that (-8.0 cells/ μ l;

$n = 15$) of TF group; the two groups were however, comparable in age ($P = 0.17$) and body weight ($P = 0.96$). Only adherence and pre-HAART CD4 counts were associated ($P = 0.001$) with HAART outcome; while only age apparently influenced ($P = 0.01$) adherence rate. Eighty-five percent of the men benefited from the HAART. The success was apparently due to adherence and less than or ($\leq$) 200 pre-HAART CD4 counts; while age ≥ 40 years apparently reduced adherence level.

Keywords Men · Antiretroviral therapy · CD4 count · Adherence · Nigeria

Introduction

Human immunodeficiency virus (HIV) has been described as a global disaster. This is not only due to the facts that it has led to more than 20 million deaths globally and more than 33 million people infected; but to the projection that by 2010, more than 40 million children will have one or both parents dead from acquired immunodeficiency syndromes (AIDS). In addition, HIV infects both male and female individuals of all age groups, predominantly affecting those in the productive age groups thereby reducing gross national product (GNP) [25].

Following invasion of human body, HIV targets and infects human cells bearing on their surfaces CD4 and CXCR4 or CCR5 molecules (<http://www.niaid.nih.gov/>). Though other cells with these molecules are susceptible, the CD4⁺ T helper cell types 1 and 2 are specific target of the virus. The CD4⁺ T cells are lysed following productive intracellular replication of HIV. Release of infectious HIV progeny from such lysed cells results in infection of more susceptible cells which culminates in massive destruction of

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the T helper cells. As the infectious virus number increases in the plasma of HIV-infected, the CD4⁺ T cells decline in number [4]. In addition to decimation of these cells, HIV-mediated disruption of CD4⁺ T cell functions also occurs thereby impacting on the classical pathways of immune function by altering the activation and production of specific effector T cells and immunoglobulins [12, 19]. In HIV-infected, assessment of functional status of CD4⁺ T cells is good but this is said to be notoriously difficult as a laboratory routine [19]; hence, among others, the blood CD4⁺ T cell count and HIV RNA copy number (plasma infectious HIV particles) have become major markers of AIDS-defining illnesses and response to clinical management of HIV and AIDS. However, Plasma viral load measurement is not necessary before initiating antiretroviral therapy (ART) as it rarely informs the clinical decision regarding when ART should begin if both CD4 testing and the assessment of clinical staging are performed [26]. Early in the course of HIV infection, human body positively responds by replenishing the lost CD4⁺ T cells (<http://www.niaid.nih.gov/>) but, at a certain point, the body's ability to buffer the loss becomes inadequate. This is the point the otherwise asymptomatic infected becomes symptomatic with diverse clinical signs (i.e. clinical syndrome); and in the absence of effective ARV therapy, the victim progresses to immunodeficiency-mediated syndrome known as AIDS. Successful management of HIV infection and AIDS-defining illnesses is achievable with more than twenty approved ARVs belonging to six mechanistic classes. These classes include nucleoside reverse transcriptase inhibitors (NRTIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs), protease inhibitors (PIs), fusion inhibitor (FI), CCR5 antagonists and integrase inhibitors within which different combination regimens can be designed (<http://www.aidsinfo.nih.gov/ContentFiles/AdultandAdolescentGL.pdf>). Lamivudine (3TC) and stavudine (d4T), as NRTIs, are competitive inhibitors of HIV-1 reverse transcriptase (RT) which act as terminator of synthesis of new HIV-1 DNA chain. A good example of NNRTIs is nevirapine (NVP) which acts as non-competitive inhibitor of HIV RT. As effective as these ARVs are, their clinical use as monotherapies is however bedeviled and therefore discouraged by the incidence of ARV drug resistance [26]. This resistance is engendered by the generation of genetically diverse progenies of HIV following productive intracellular replication. Research was however, able to get around this by the discovery that drug resistance was less of a problem for combination of ARVs; and more importantly, ARV combination into one pill improves adherence of patients to ARV regimen and its success [26]. The drug combination is referred to as highly active antiretroviral therapy (HAART), i.e. the treatment regimen of using three or more ARVs to reduce the viral load of individuals infected with HIV

(www.anecca.org). The use of one NNRTI or a PI (with or without ritonavir boosting) and dual NRTI backbone is the most extensively studied ARV regimens for ART-naïve patients (<http://www.aidsinfo.nih.gov/ContentFiles/AdultandAdolescentGL.pdf>). Four NNRTIs (etravirine, delavirdine, efavirenz and nevirapine) are Food and Drug Administration (FDA) approved (www.fda.gov). From all these, various combinations have been reported, e.g. efavirenz with zidovudine and lamivudine; nelfinavir with zidovudine and lamivudine; efavirenz with stavudine and didanosine. WHO recommended generic NVP with 3TC and d4T, among others, as first-line ART and it has been widely used with effectiveness [8, 11, 22, 24, 26]. Specifically, NVP was recommended as alternative NNRTI in adult HIV-infected males with CD4 count of less than or ($\leq$) 400 cells/ μ l (<http://www.aidsinfo.nih.gov/ContentFiles/AdultandAdolescentGL.pdf>). Since its introduction, management of HIV and AIDS with HAART has become effective at decreasing HIV-related sickness and deaths as it brings about HAART-mediated immune reconstitution [2, 5, 21, 23]. This is due to reduction of plasma viral load, thereby allowing the victim's immune system achieve rebound of otherwise decimated CD4⁺ T cells, as well as, restoration of their functions. The CD4⁺ T cell rebound is said to be accelerated in the first 3 months post-ART [14]. Though the early rebound of these cells following ART has been attributed to re-distribution/release of CD4⁺ T cells from lymphoid tissues into blood circulation [17]. Many reports indicated that greater than or ($\geq$) 50 CD4⁺ T cells/ μ l is a marker of HAART-mediated immunologic success [4, 15, 16, 27]. The immunologic success had been reported to be dependent on adherence to ART [16, 27]. In addition to its effectiveness, when appropriately used, HAART markedly reduces the risk of heterosexual and vertical HIV transmission (<http://www.aidsinfo.nih.gov/ContentFiles/AdultandAdolescentGL.pdf>). Therefore, HAART has become essential to all HIV victims. Many authors have provided information regarding the decision to initiate HAART by demonstrating that adverse clinical outcomes are less likely to arise in those who initiate HAART with CD4⁺ T cell counts of greater than 0.200×10^9 cells/L; HIV-1 RNA levels of less than 50,000 copies/mL and an absence of a clinical AIDS-defining event [13, 20].

In Nigeria, like many other African countries, 3TC, d4T and NVP as HAART is still offered to ART-naïve HIV and AIDS patients in government and private-owned health facilities where voluntary confidential counselling and testing for HIV infection, as well as, HIV and AIDS management are done. Though, many authors have reported the effectiveness of this drug combination, reliable information on the efficacy is still continually desired; as ART remains a rapidly evolving and challenging area of HIV medicine [28]. Many studies [1, 3, 7, 9] have reported the therapeutic benefit

of these drugs to people living with HIV and AIDS in Nigeria and different parts of the world.

To the best of our knowledge, Kogi State, Nigeria has paucity of information on the immunologic benefit of generic 3TC, d4T and NVP to adult HIV-infected males. This study was therefore conducted to assess the CD4⁺ T cell response of ARV-naïve, HIV-infected men to the HAART after 3 months.

Materials and Methods

Study Location

This study was carried out between May and October, 2008 at Federal Medical Center (FMC), Lokoja, Kogi state, Nigeria. Lokoja, the capital of Kogi State with population of 60,579, houses three major ethnic groups i.e. Igala, Ebira and Okun (Yoruba). Other minor groups include Kankanda, Kupa, Ogori-Mangongo, Nupe, Bassa-Komo, Bassa-Nge and Gwari; as well as, people from other Nigerian states and non-nationals. Kogi state is the most centrally located state in Nigeria and bordered by nine other states. The study participants were laboratory confirmed HIV-infected men attending HIV voluntary counselling and testing [VCT]/ART section of FMC, Lokoja.

Study Design

This is a prospective study. The aim of the study was explained to the Management of FMC, Lokoja and permission to undertake the study was subsequently granted. A health officer explained the objectives and details of the study to HIV-positive, ART-naïve men attending the medical centre's HIV VCT/ART section. Criteria for including each participant in the study are (1) male gender ≥ 18 years; (2) laboratory confirmed HIV infection; (3) ART-naïveté prior to start of HAART and known date of ARV initiation; (4) clinically documented baseline CD4 count and willingness to do follow-up CD4 count; (5) eligibility to take ARVs; (6) understanding ART; (7) readiness to start and adhere to 3TC, d4T and NVP regimen; and (8) consent to enroll in the study. Exclusion criteria include: male less than ($<$) 18 years; feminine gender; ART-experience; being on ARVs other than 3TC, d4T and NVP; no interest to enroll in the study.

Subsequently, men were consecutively recruited to enroll in the study. Participant's confidentiality was maintained by assigning identification number (ID) number to each; the responses of the men at interview were documented into self-constructed questionnaire forms. Other pertinent socio-demographic data used in the study were as documented in each patient's medical record. The baseline

CD4 counts of the men were used to categorize them into three pre-HAART groups viz: ≤ 200 ; 201–350 and ≥ 351 CD4⁺ T cells/ μ l. The men were requested to report in the hospital for regular medical check-up, especially at 3 months to assess follow-up CD4 count. The independent variables collected about the men include educational status; marital status; age; body weight (BW); presence of opportunistic infections (OIs) and information on adherence to ARV regimen. "Adherence" to the ARV regimen was documented for each patient as self-reported at interview; and any deviation from the ARV regimen was recorded as "non-adherence". HAART was defined as the combination of 3TC, d4T and NVP at 150 mg, 30/40 mg and 200 mg respectively on twice-daily regimen. Main outcome measures were the three-month post-HAART CD4 count and adherence rate. Therapeutic success (TS) and failure (TF) were defined as ≥ 50 CD4⁺ T cells/ μ l and < 50 CD4⁺ T cells/ μ l respectively at 3-month follow-up.

Blood Sample Collection and Laboratory CD4 Count

About 5 ml whole blood was aseptically collected by venepuncture from each patient. The blood was collected in EDTA-treated blood sample tubes. CD4 count of each blood sample was done using Partec CyFlow[®] Counter (Partec GmbH, Otto-Hahn-Straße 32, D-48161 Münster, Germany). The CD4 count was carried out according to the instructions of the Flow Cytometer manufacturer. The CD4 count results were recorded into respective questionnaire forms.

Data Analysis

The results of this study were presented with descriptive statistics. Median values of the parameters were presented with range. We used Mann–Whitney *U* test, Kruskal–Wallis, Wilcoxon's matched pair and χ^2 statistical tests to establish statistical difference/association or lack thereof between patients' variables. Two-tailed hypothesis was used with $P \leq 0.05$ as indicator of statistical significance. Statistical Package for the Social Sciences (SPSS) 15.0 for Windows[®] was used to perform the analyses.

Results

Demographic Characteristics

One hundred HIV-infected, ARV-naïve men aged 24–60 years (median: 39.5 years) enrolled in the study and initiated HAART at FMC, Lokoja, Kogi State, Nigeria. All the participants completed the 3-month period. Participants' variables with respect to HAART outcome i.e. TS and TF are as shown (Fig. 1).

The men had median BW of 61.0 kg ($n = 100$; range: 33–80 kg). The overall ARV adherence rate was 95% (Fig. 1; Table 1). Of the latter, 88.4% attained TS, while only 20.0% achieved TS among the 5 HIV-infected men that self-reported non-adherence (Table 1); the remaining 4 experienced decline in their CD4 count ranging from –86 to –8 CD4⁺ T cells/ μ l (data not shown).

Pre-HAART CD4 Count

The baseline CD4 count obtained ranged from 121 to 584 cells/ μ l ($n = 100$; median: 186.5 cells/ μ l). For the HIV-infected men with TS and TF, their median CD4 counts are as shown (Fig. 2); their respective ranges of pre-HAART CD4 count were 121–584 and 210–463 cells/ μ l.

The baseline CD4 count of the patients in ≤ 200 CD4 count/ μ l category ranged from 121 to 192 cells/ μ l ($n = 53$). The corresponding values for the 201–350 and ≥ 351 CD4⁺ T cells/ μ l groups were 210–343 cells/ μ l ($n = 31$) and 351–584 cells/ μ l ($n = 16$) respectively. Figure 3 shows their median CD4 counts.

Follow-up CD4 Count

None of the 100 HIV-infected men who initiated HAART had zero (i.e. no change) CD4 count response at the 3-month post-therapy. The overall follow-up CD4 count ranged from 177.0 to 833.0 cells/ μ l ($n = 100$; median: 285.0 cells/ μ l, data not shown); the median change in CD4 count was +99.0

cells/ μ l (range: –135 to +283 cells/ μ l, $n = 100$). Those with TS and TF respectively had median follow-up CD4 counts ranging from 215 to 833 cells/ μ l ($n = 85$) and 177 to 330 cells/ μ l ($n = 15$). Their respective median CD4 counts are as shown (Fig. 2). Eight (53.3%) of the 15 that attained TF experienced decline in CD4 counts ranging from –135 to –8 cells/ μ l at follow-up (data not shown).

The median follow-up CD4 counts of the three pre-HAART CD4 count categories ranged from 215 to 377 cells/ μ l for the ≤ 200 cells/ μ l group; 177.0 to 466.0 cells/ μ l for the 201–350 cells/ μ l and 310.0 to 833.0 cells/ μ l for the ≥ 351 cells/ μ l group. Their respective median CD4 counts over the 3-month period are as shown (Fig. 3).

The median change in CD4 count per month for the 100 HIV-positive men was 33.0 cells/ μ l/month (range: –45 to 94.3 cells/ μ l/month). The corresponding values were 34.7 cells/ μ l/month (17.7–94.3 cells/ μ l/month) and –2.7 cells/ μ l/month (–45 to 15.3 cells/ μ l/month) for the 85 and 15 men that attained TS and TF respectively.

Monthly changes of 33.0 CD4⁺ T cells/ μ l/month, 32.3 CD4⁺ T cells/ μ l/month and 47.2 CD4⁺ T cells/ μ l/month were documented for the ≤ 200 , 201–350 and ≥ 351 cells/ μ l pre-HAART groups respectively. The proportional changes in the HIV-infected men comprising each of these groups are as shown (Fig. 4).

Based on the increase in CD4⁺ T cell of ≥ 50 cells/ μ l as the indicator of HAART benefit at follow-up, 85% of the men attained TS (Fig. 1). The demographic data of the 15% that attained TF is reflected in Fig. 1. The ARV

Fig. 1 Demographic data of Human immunodeficiency virus (HIV) infected men who initiated highly active antiretroviral therapy (HAART) with respect to immunologic outcome at federal Medical Centre (FMC), Lokoja, Kogi state, Nigeria. *Legends:* *OI* Opportunistic infection, *BW* Body weight (Kg), *Blue bar* ≥ 50 CD4 count/ μ l. *Red bar* < 50 CD4 count/ μ l

Table 1 Association of patients' variables with therapeutic outcome and adherence rate among men who initiated HAART at FMC, Lokoja, Kogi State, Nigeria

HAART outcome		
Variables	Total number (% of TS)	P-value
Adherence to ARV regimen		
Yes	95 (88.4)	0.001**
No	5 (20.0)	
Age (years)		
<40	64 (89.3)	0.18
≥40	44 (79.5)	
BW (Kg)		
<61	49 (83.7)	0.72
≥61	51 (86.3)	
Educational status		
Primary	19 (84.2)	0.93
Secondary	31 (87.1)	
Tertiary	50 (84.0)	
Presence of OIs		
Yes	80 (85.0)	1.00
No	20 (85.0)	
Marital status		
Married	52 (76.9)	0.11
Single	36 (91.7)	
Divorced	9 (100.0)	
Widower	3 (100.0)	
Pre-HAART CD4 count/μl		
≤200	53 (100.0)	0.001**
201–350	31 (67.7)	
≥351	16 (68.8)	
Adherence outcome		
	Total number (adherence %)	
Age (years)		
<40	56 (100.0)	0.01*
≥40	44 (88.6)	
Educational status		
Primary	19 (89.5)	0.47
Secondary	31 (96.8)	
Tertiary	50 (96.0)	
Presence of OI		
Yes	80 (95.0)	1.00
No	20 (95.0)	
Marital status		
Married	52 (94.2)	0.73
Single	36 (97.2)	
Divorced	9 (88.9)	
Widower	3 (100.0)	
Pre-HAART CD4 count/μl		
≤200	53 (98.1)	0.21
201–350	31 (93.5)	
≥351	16 (87.5)	

TS Therapeutic success, OI opportunistic infection

** Highly significant association

* Significant association

Fig. 2 Comparative median CD4 counts of therapeutic success (TS) and therapeutic failure (TF) groups of the HIV-infected men who initiated HAART at FMC, Lokoja, Kogi state, Nigeria. *Legends: Blue bar TS group, Red bar TF group. Statistics: Red bars Pre-HAART, follow-up statistically similar, Blue bars Pre-HAART, follow-up significantly different. Blue and red bars at pre-HAART significantly different. Blue and red bars at follow-up statistically similar. Blue and red bars at change in CD4 count significantly different* **Fig. 3** Comparative median CD4 counts of the three categories of HIV-infected men who initiated HAART at FMC, Lokoja, Kogi state, Nigeria. *Legends: Blue bar ≤200 CD4 count/μl. Red bar 201–350 CD4 count/μl. Green bar ≥351 CD4 count/μl. Statistics: Blue, Red and Green bars at Pre-HAART significantly different. Blue, Red and Green bars at follow-up significantly different. Blue, Red and Green bars at change in CD4 count statistically similar*

adherence rates for the 85 and 15 men with TS and TF were 98.8% (84 men) and 73.3% (11 men) respectively. The adherence rates for the three pre-HAART categories: ≤200; 201–350 and ≥351 cells/μl are shown (Table 1).

Discussion

This study was carried out to assess immunologic (CD4 count) benefit of HAART (3TC, d4T and NVP) to HIV-infected, ARV-naïve men attending HIV and AIDS clinic of FMC, Lokoja, Kogi State, Nigeria. Of the 100 men studied, 95% adhered to ARV regimen. Whether or not

Fig. 4 Proportional trend in the three categories of HIV-infected men who initiated HAART with respect to pre- and post-HAART CD4 counts at FMC, Lokoja, Kogi state, Nigeria. *Legends: Blue bar Pre-HAART, Red bar Follow-up*

these men would maintain or exceed this rate remained speculative. Though the sample size was small, the 95% adherence rate recorded in this study was close to >95% adherence rates reported by Paterson et al. [18] and Gordillo et al. [10] as necessary requirement in order to maximize benefits of ART. The 95% adherence rate among these men was higher than 58.1% reported by Erah and Arute [6] in Benin city, Nigeria for males (36) and females (66) HIV and AIDS patients though. A study (Kilaru et al. [15] reported similar observation that 69.6% HIV and AIDS patients in a developing Caribbean country had adherence rate of 91–100%.

Eighty-five per cent attained TS (i.e. increase of ≥ 50 CD4 count) after 3-month HAART (Fig. 1). The increase of ≥ 50 CD4 count/μl after 6 months on HAART as marker of immunologic success had earlier been reported by Kilaru et al. [15] in a developing Caribbean country. In their study, immunologic success rate of 77.5% among males ($n = 89$) was documented; this rate was less than 85% TS observed in this study.

Overall, the median follow-up CD4 count was significantly ($P = 0.001$; $n = 100$) higher than the corresponding pre-HAART value. Having observed that the HIV-infected men that attained TS and TF were comparable in age ($P = 0.17$) and BW ($P = 0.96$), we attempted comparison of their CD4 count profiles. The median follow-up and pre-HAART CD4 counts of those men with TF were not significantly ($P = 0.19$) different (Fig. 2), but the median follow-up CD4 count of those with TS was significantly ($P = 0.001$) higher than their corresponding baseline value. These two groups differed significantly in median pre-HAART CD4 counts ($P = 0.001$) and in median change in CD4 counts ($P = 0.001$). They were however, comparable ($P = 0.86$) in median follow-up CD4 counts. The reason for the latter observation was not apparent to us vis-a-vis the increase of ≥ 50 CD4 count/μl as indicator of TS among the HIV-infected men. We therefore, reaffirmed

change in CD4 count, rather than the follow-up count, as better indicator of HAART-mediated immunologic success [4, 15, 16, 27]. We also observed that 98.1% of the men changed from the range of severe immunodeficiency for the better CD4 count after 3-month HAART (Fig. 4).

From the foregoing, based on median follow-up CD4 count on one hand, it could be generally inferred that all the HIV-infected men benefited immunologically from the HAART. On the other hand, based on the increase of ≥ 50 CD4 count/μl after 3 months on HAART, only 85% had immunologic benefit. The latter further corroborated change in CD4 count as a better means of evaluating immunologic benefit of HAART among the HIV-infected men in Nigeria.

We also endeavour to assess immunologic outcome of HAART among three categories of the HIV-infected men (≤ 200 ; 201–350 and ≥ 351 CD4 count groups). The three groups were comparable in age ($P = 0.36$) and body weight ($P = 0.99$), as well as, in change in CD4 counts ($P = 0.06$) but, differed significantly in pre-HAART ($P = 0.001$) and follow-up CD4 counts ($P = 0.001$) (Fig. 3). Highest and lowest median follow-up CD4 counts observed for the ≥ 351 and ≤ 200 groups respectively after 3 months appeared similar in trend to mean follow-up CD4 counts reported by Erhabor et al. [7] for males (53) and females (47) at 12 months on 3TC, d4T and NVP at University of Port-Harcourt, Nigeria.

In order to determine independent variables of the HIV-infected men that influence HAART outcome on one hand and adherence on the other, we analysed statistical associations between the variables and the two outcome measures. Overall, only adherence to ARV regimen appeared significantly ($P = 0.001$) associated with HAART outcome (Table 1); in addition, among the TS and TF groups, those that self-reported adherence to therapy had 4.42 folds greater likelihood of attaining therapeutic success (data not shown). Age ($P = 0.18$); BW ($P = 0.72$); educational status ($P = 0.93$); presence of opportunistic infections ($P = 1.00$); and marital status ($P = 0.11$) had no significant associations with outcome of HAART among the men (Table 1). The observation that age was not associated with TS among these men was similar to report of Kilaru et al. [15].

However, as regards adherence, only age of the men had significant ($P = 0.01$) association with adherence rate. Marital status; educational status and presence of opportunistic infections ($P = 0.73$; $P = 0.47$ and $P = 1.00$ respectively) were not significantly associated with adherence rate among these men (Table 1). These showed that adherence to ARV regimen influenced HAART outcome, while only age influenced adherence to therapy. Adherence had equally been cited as a strong factor influencing ART success [6, 10, 26, 27].

Analyses showed that among the three pre-HAART groups, baseline CD4 count was significantly ($P = 0.001$) associated with HAART outcome with those in ≤ 200 CD4 count group having 100% TS (Table 1). Kilaru et al. [15] who categorized males and females HIV and AIDS patients they studied into <50 ; 50–199 and ≥ 200 pre-HAART CD4 count groups made a contrary observation that baseline CD4 count did not appear to influence immunologic success after 6-month HAART. The difference in the observations might be due to differences in gender studied; criteria of baseline CD4 count categorization; ARVs used and longer HAART duration. In the present study, pre-HAART CD4 count was however, not significantly ($P = 0.21$) associated with adherence despite the observation that adherence rate was highest (98.1%; $n = 53$) among the ≤ 200 CD4 count/ μl group (Table 1).

The median monthly change in CD4 count of 34.7 cells/ μl /month for those that attained TS was significantly ($P = 0.001$) higher than -2.7 cells/ μl /month observed for the HIV-infected men having TF. However, statistical comparability was observed for the three pre-HAART CD4 count categories ($P = 0.12$) regarding monthly change in CD4 count (data not shown).

A limitation of this study was low sample size; a future study of larger sample size was therefore suggested. Also, benefit of 3TC, d4T and NVP to HIV-infected men having baseline ≥ 351 CD4 cells/ μl needs further assessment with larger sample size as this study could not ascertain such.

In conclusion, 85% of the HIV-infected men benefited immunologically from the HAART. The success was apparently due to adherence and ≤ 200 pre-HAART CD4 counts; while age ≥ 40 years appeared to reduce adherence level among the men.

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