East African Medical Journal Vol. 85 No. 1 January 2008
PREVALENCE OF DYSLIPIDEMIA AND DYSGLYCAEMIA IN HIV INFECTED PATIENTS
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E. M. MANUTHU, M. D. JOSHI, G. N. LULE and E. KARARI

ABSTRACT

Background: **Highly active antiretroviral therapy (HAART) has dramatically reduced AIDS morbidity and mortality, however long-term metabolic consequences including dysglycaemia and dyslipidemia have raised concern regarding accelerated cardiovascular disease risk.**
Objective: **To determine the period prevalence of dyslipidemia and dysglycaemia in HIV-infected patients.**
Design: **Cross-sectional comparative group study.**
Setting: **Kenyatta National Hospital, a tertiary HIV dedicated out-patient facility.**
Subjects: **Consecutive HIV- positive adult patients.**
Main outcome measures. **Dyslipidemia: presence of raised total or LDL cholesterol or low HDL cholesterol, or raised triglycerides. Dysglycaemia: presence of impaired fasting glucose or impaired glucose tolerance, or diabetes mellitus.**
Results: **Between January and April 2006, out of 342 screened patients, 295 were recruited and 58% were females. One hundred and thirty four (45%) were on HAART, 82% of whom were on stavudine, lamivudine and either nevirapine or efavirenz. Overall prevalence of dyslipidemia was 63.1% and dysglycaemia was 20.7%. High total cholesterol occurred in 39.2% of HAART and 10.0% HAART naive patients (p<0.0001, OR 5.18, CI 3.11-10.86), whereas high LDL cholesterol occurred in 40.8% and in 11.2% respectively (p<0.0001, OR 5.43, Cl 2.973-9.917). HDL levels were low in 14.6% and 51.3% among HAART and HAART naive patients, respectively, (p<0.0001, OR 0.16, Cl 0.091-0.29) while high triglycerides occurred in 25.6% and 22.5% respectively (p=0.541 OR 1.184 Cl 0.688-2.037). Among patients on HAART compared to HAART naive patients, diabetes was found in 1.5% against 1.2% (p=0.85), impaired fasting in 2.2% against 0.6% (p=0.30) and impaired glucose tolerance in 16.4% against 21.1% (p=0.22), respectively.**
Conclusions: **HIV- infected patients demonstrated a high prevalence of dyslipidemia. HAART use was associated with high levels of total, and LDL cholesterol and high triglyceride levels, an established athrogenic lipid profile. However, HAART was not associated with low HDL cholesterol and had no significant effect on dysglycaemia**

INTRODUCTION

The introduction of highly active antiretroviral therapy (HAART) has drastically reduced the morbidity and mortality associated with AIDS(1,2) and patients now enjoy longer and healthier lives. HAART however is not a cure for AIDS and thus has to be administered on a permanent and continuous

fashion for maximum benefit to be obtained (3). Concern is now rising regarding the long-term metabolic complications associated with HAART, including insulin resistance and type 2 diabetes mellitus, dyslipidemia, increased levels of plasminogen activator inhibitor type 1, apolipoprotein B and high-sensitivity C-reactive protein (4). Antiretroviral drugs are also associated with abnormal fat distribution and greater visceral adiposity (4). These metabolic changes have been shown to increase the risk of developing cardiovascular disease independent of HAART, which strongly suggests that patients on HAART potentially have a disproportionately elevated risk.

In untreated HIV-infected individuals, lipid abnormalities are common. These include elevated plasma levels of triglycerides, decrease in total and LDL-cholesterol levels while HDL-cholesterol levels decrease or remain unchanged (5,6).

Increased prevalence of HAART associated dyslipidemia has been consistently reported in the literature (7,8). Friis-Moller *et al* in a large cross-sectional study, reported hypercholesterolemia (>240mg/dl;6.3mmol/l) of 27%, 23%,10% and 8% among patients receiving PI, NNRTI, NRTI based antiretroviral regimens, and previously untreated patients respectively. Corresponding levels of hypertriglyceridemia (>200mg/dl, >2.3mmol/1) were 40%, 32%, 23%, and 15%; and low HDL (<35mg/dl, 0.9 mmol/1) were 27%, 19%, 25% and with 26% (8,9).

Pathogenetic mechanisms involved in causing dyslipidemia include increased apolipoprotein B levels, increased dense LDL 2 levels, and increased hepatic synthesis of triglyceride-rich very-low-density-lipoproteins with decreased clearance of triglycerides. Circulating cytokines, acute-phase reactants and viral infections may also play a role (10).

Insulin resistance is frequently found in HIV-infected patients and is commonly associated with subcutaneous lipoatrophy, buffalo hump accumulation of fat and central obesity (11). Although it is not entirely clear how insulin resistance comes about in patients on HAART, the abnormal distribution of fat (in particular increase in visceral fat and decrease in subcutaneous fat) appears to contribute to this indirectly. Visceral adiposity may increase the levels of circulating substrates including free fatty acids thereby causing abnormal insulin signaling (12,13).

Compelling data now emerging seem to strongly suggest associated increasing cardiovascular and cerebrovascular morbidity and mortality. The DAD (Data collection on Adverse events of anti-HIV drugs) study group, an observational study investigating a total of 23,468 HIV infected patients in 11 cohorts across 21 countries in Europe, USA and Australia, reported a 26% increase in risk in the frequency of myocardial infarction per year of exposure to HAART (p for trend <0.001) (14). Furthermore, recent data from the same group now show an increase in other cardiovascular and cerebrovascular events (CCVE) other than myocardial infarction in patients on HAART. These include invasive cardiovascular procedures (coronary angioplasty or stenting, coronary artery bypass surgery, and carotid endarterectomy), stroke and death from other CCVEs. The incidence of first CCVE was 5.7 per 1000 person years and increased with longer exposure to HAART (15).

These results lend credibility to the hypothesis that atherosclerosis is a side effect of HAART. As the weight of evidence accumulates, there is increasing need to determine conclusively whether the HAART associated risk translates into increased cardiovascular events. This study aims to document the prevalence dyslipidemia and dysglycaemia.

MATERIALS AND METHODS

This was a cross-scctional comparative study conducted, after appropriate institutional ethical approval, between January and April 2006 at the Comprehensive Care Centre, a daily dedicated HIV out-patient clinic at the Kenyatta National Hospital, a tertiary referral and teaching hospital in Kenya. Normally, new patients are clinically evaluated by the attending medical team, and after a comprehensive history, physical examination and laboratory investigations, patients are given a scheduled appointment, usually a week later, when they are to be considered for commencement of antiretroviral medication. The study screened consecutive HAART naive patients in the interim period between this first visit and commencement of ARVs, whereas those on HAART were screened during their monthly visit when they normally returned for their supply of drugs. All patients had their fasting glucose and lipid profiles done on their subsequent scheduled visit. Consecutive adult HIV positive patients (as documented in their clinic records) receiving highly active antiretroviral therapy (HAART) for at least four weeks, who gave no history of having diabetes, and had not been on treatment with lipid lowering agents were enrolled in the study. Patients with a familial dyslipidemias and those who had changed their antiretroviral regimens in the last one year were also excluded. Similarly, HIV positive patients, eligible for, but not yet receiving HAART were also enrolled to serve as the comparative group. All patients gave informed written consent.

After a complete history was obtained, both groups of patients were examined, had their blood pressure taken according to WHO criteria, after which an investigator administered questionnaire

was completed. On subsequent scheduled visit, and having fasted for 9-12 hours, patients had blood samples taken for fasting blood sugar and lipid profiles and 2 hours later for post prandial blood sugar following administration of a glucose solution containing 75g of glucose in 300ml water. Blood glucose was measured with Medisense Precision QID machine that uses dry oxidation method. Lipid profiles were done using the AEROSET SYSTEM machine that utilises a direct method to measure HDL and LDL cholesterol and enzymatic hydrolysis to assay total cholesterol and triglycerides.

HAART was defined as any combination of at least three drugs from the three classes of antiretroviral drugs i.e protease inhibitors (PIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs) and nucleoside reverse transcriptase inhibitors (NRTIs), one of which was a PI or an NNRTI, or a triple combination of NRTIs, with or without the addition of a fusion inhibitor. This was further subcategorised into PI-containing regimens, PI-sparing regimens, and PI and NNRTI sparing regimens.

Dyslipidemia was defined as presence of any of the following: high total or LDL cholesterol, high triglycerides or low HDL cholesterol according to NCEP, ATP III categorisation (16). Dysglycaemia was defined as presence of diabetes, impaired fasting glucose or impaired glucose tolerance according to the World Health Organization criteria (17).

Data were analysed using the SPSS version 11.5 statistical program and described as frequency distribution, means, and percentages and was then presented as pie-charts, bar charts, and cross tabulations. The student t-test was used to compare continuous data and the chi square test was used for categorical data.

RESULTS

A total of 342 consecutive HIV-positive patients were screened, 47 (13.7%) were excluded; five declined consent, 33 failed to turn up for blood sample collection, three blood samples were unsuitable for analysis, and six HAART treated patients had been on more than one regimen. Two hundred and ninety five were thus enrolled, 134 (52%) of whom were on HAART. The characteristics of the excluded patients did not differ appreciably from those of enrolled patients.

The demographic and cardiovascular risk factor profiles of study participants, stratified on basis of HAART category, are depicted in Table 1. Females predominated in both categories of patients; 59.7% HAART and 56.5% in non-HAART (p=0.582). Majority of patients were married and 99% were residents of Nairobi and its suburbs. Age ranged between 24 and 69 years in patients on HAART and 20 and 75 years among patients not on HAART. Corresponding mean and median HAART ages were 39.4 years and 36.5 years; and non-HAART 36.5 years and 36 years, respectively. In both groups of patients, females were younger.

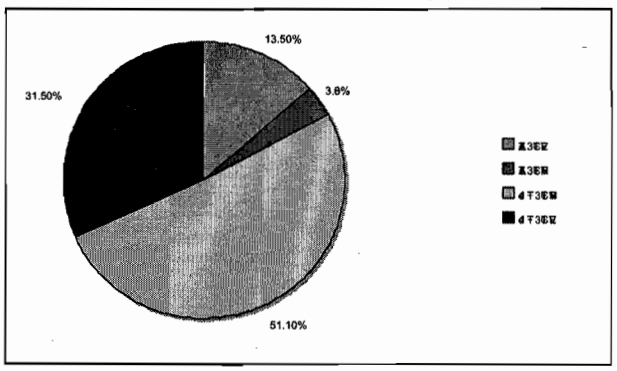
Table 1
Study population characteristics by HAART status

Characteristic		HAART				Total	
		Yes		No			
		No.	(%)	No.	(%)	No.	(%)
Sex	Male	54	40.3	70	43.5	124	42.0
	Female	80	59.7	91	56.5	171	58.0
Age(years)	20-30	21	15.7	46	28.6	67	22.7
	31-40	62	46.3	67	41.6	129	43.7
	41-50	31	23.1	36	22.4	67	22.7
	≥51	20	14.9	12	7.5	32	10.8
Cigarette	Current	7	5.2	20	12.4	27	9.2
smoking	Former	21	15.7	25	15.5	46	15.6
	Never	106	79.1	116	72.0	222	75.3
Hypertension		18	13.4	24	14.9	42	14.2
Family history on CAD		5	3.7	3	1.9	8	2.7

Duration from the date of first diagnosis of HIV in study subjects ranged from one to 144 months with a median of 12 months. Majority (88%) of patients not receiving HAART had been diagnosed with HIV within the preceding one year, compared to 44% among those on HAART (p<0.0001).

Thirty two percent of patients on HAART had been on treatment for 4 to 24 weeks, 26.1% for 24 to 48 weeks and 41.8% for more than 48 weeks. All patients on HAART treatment, except one, were on protease inhibitor-sparing regimens. Majority (82.7%) were on stavudine-based regimens with 51.1% on a combination of stavudine, lamivudine and nevirapine (d4T+3TC+NVP) and 31.6% receiving stavudine, lamivudine, and efavirenz (d4T+3TC+EFV). The remaining 17.3% were receiving zidovudine-based regimens with 13.5% on zidovudine, lamivudine and efavirenz (AZT+3TC+EFV) regimen and only 3.8% on zidovudine, lamivudine and nevirapine (AZT+3TC+NVP). The single protease inhibitor-based regimen composed of zidovudine, lamivudine and ritonavir-boosted lopinavir (AZT+3TC+LPVr) (Figure 1).

Figure 1
Categories of HAART among study population



Majority of patients in both groups were lifetime non-smokers; 79.1% in patients on HAART against 75.3% among those not on HAART. Former smokers accounted for 15 % in both groups and current smokers for 5% and 12% among HAART and non-HAART patients, respectively.

Hypertension was present in 18(13.4%) of patients receiving HAART and 24(14.9%) among patients not on HAART (p=0.425). Only eight patients in the study population reported a family history of coronary artery disease. Data on body weight were available for 289 patients. The mean weight for patients on HAART was found to be 65.2kg with a median of 66kg and was significantly higher than that of patients not on HAART whose mean weight was 58.1 kg with a median weight of 57kg (p<0.0001).

Dyslipidemia: Among the patients on HAART, the prevalence of hypercholesterolemia (total cholesterol (TC) ≥5.17mmol/1) was 39.2% compared to 10.0% in patients not on HAART (p<0.0001).The probability for high TC in patients on HAART was five fold that of non-HAART patients (OR 5.81, 95% CI 3.11-10.86 (Table 2). Degree of TC elevation in this HAART category of patients was evenly spread between borderline high (22%) and moderate- severe high (16.9%).

Table 2
Prevalence of hypercholesterolemia among patients on HAART and among HAART naive patients

Hypercho-lesterolemia	HAART Yes	(%)	HAART No	(%)	Total No.	(%)
Abnormal	51	39.2	16	10.0	67	22.7
Normal	79	60.8	144	90	223	77.8
Total	130		160		290	100

OR 5.8 (95% CI 3.1-10.9) p-value < 0.0001
Hypercholesterolemia defined as TC≥5.17 mmol/1

In HAART and non-HAART patients the prevalence of elevated LDL-c was 40% and 11% respectively (p<0.0001). Patients on HAART were five more likely to have an elevated LDL-c relative to no HAART (OR 5.43, 95% CI 2.973-9.917) (Table 3). Borderline high LDL-c occurred in 27.7%, high LDL-c in 9.2% and very high LDL-c in 3.8% of patients.

Table 3
Prevalence of high LDL cholesterol among patients on HAART and those not on HAART

High LDL Cholesterol	HAART Yes	(%)	HAART No	(%)	TOTAL No.	(%)
Abnormal	53	40.8	18	11.3	71	24.1
Normal	77	59.2	142	88.8	219	75.9
Total	130		160		290	100

OR was 5.43 (49% CI 2.93-9.92), p-value < 0.0001
High LDL defined as LDL ≥ 3.34 mmol/l.

The prevalence of a low HDL-c was 14.6% in HAART and 51.3% non HAART (p<0.000I). Being on HAART was not found to confer a higher likelihood for having low HDL-c (OR 0.163, 95% CI 0.091-0.290) (Table 4).

Table 4
Prevalence of low HDL cholesterol among patients on HAART against those who were HAART naive

Low HDL cholesterol	HAART Yes (%)	HAART No (%)	Total No. (%)
Abnormal	19 14.6	82 51.3	101 34.2
Normal	111 85.4	78 48.8	189 65.8
Total	130	160	290 100

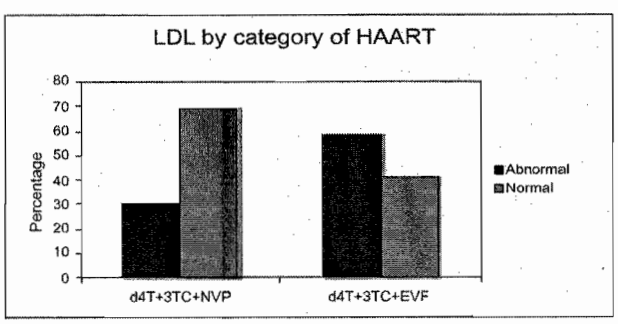
OR was 0.16 (95% CI 0.09-0.29), p-value < 0.0001. Low HDL defined as < 1.03 mmol/l

Prevalence of a high TG was 25.6% and 22.5% in HAART and non-HAART patients respectively. HAART did not confer a higher probability of an elevated TG level (OR 1.184, 95% CI 0.688-2.037).

HAART regimen and dyslipidemia: Eighty two percent of HAART patients in the study were on stavudine based regimens, namely d4T and 3TC plus either EFV or NVP, hence subsequent comparisons were restricted to these two regimens. Efavirenz regimen (d4T+3TC+EFV) had the higher prevalence of dyslipidemia compared to nevirapine regimens (d4T+3TC+NVP) of 71.4% and 52.9% respectively, however this did not achive statistical significance (P=0.182).

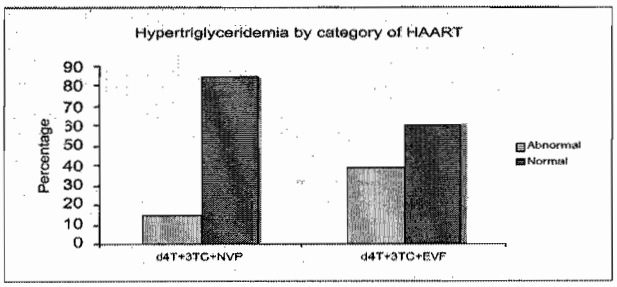
The prevalence of elevated LDL-c was significantly higher in those receiving efavirenz compared to nevaripine. (58.5%; 30.3%; p= 0.004), (Figure 2).

Figure 2
Prevalence of high LDL levels between stavudine based regimens (OR 3.25, CI 1.34-77.97, p=0.004)



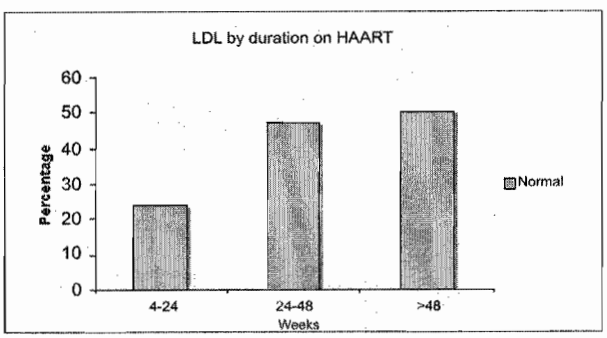
Prevalence of low HDL-C was not statistically different in the two regimens. 15.2%,17.1% p=0.79). Significantly higher levels of TGs were found in efavirenz treated patients. (39.0% vs 15.4% P=0.006) (Figure 3).

Figure 3
Prevalence of hypertriglyceridemia between stavudine based regimens (OR 3.52, CI 1.28-9.81, p=0.006)



Dyslipidemia and duration of HAART: There was a statistically non-significant trend towards an increase in prevalence of dyslipidemia with duration of any HAART treatment; duration of 4-24 weeks, 24-48 weeks and >48 weeks was associated with dyslipidemia prevalence of 22.9%, 29.2% and 48%, respectively. (p=0.235). Prevalence of elevated TC also showed a trend towards increase with duration of HAART, with corresponding prevalence of 28.6%, 47% and 42%, respectively (p=0.202). Elevated LDL-c mirrored TC, peaking at 24 to 48 weeks and the difference were statistically significant (23.8%, 47.1% and 50.8%, respectively; p=0.024) (Figure 4).

Figure 4
Prevalence of high LDL cholesterol according to duration of treatment (p=0.024)



HDL-c levels showed no trend with duration of HAART therapy (16.7%, 14.7% and 13% respectively; p =0.211).The prevalence of elevated TG levels also rose non-significantly (22%, 17.6% and 33.3%; p=0.212).

Dysglycaemia: Dysglycaemia in the entire study population was detected in 61 patients constituting a prevalence of 20.7%; 42.6% were male and 60.7% were aged below 40 years. Prevelence in HAART and non-HAART was 17.9% and 22.9% respectively (p=0.284; OR 0.731, 95%CI 0.412-1.298). Diabetes was detected in only four patients; impaired glucose tolerance (IGT) was the most common dysglycaemic abnormality occurring in 19% of study subjects with no difference between treatment groups.

Out of the 24 patients with dysglycaemia and on HAART, 17 (70.9%) were on stavudine based regimens with seven patients 29.2% receiving nevirapine (d4T+3TC+NVP), and 10 patients 41.7% on efavirenz (d4T+3TC+EFV).The rest were on zidovudine based regimens with six getting efavirenz (AZT+3TC+EFV) and only one patient on nevirapine (AZT+3TC+NVP). This difference was not statistically significant (p= 0.087).

Among the 24 patients on HAART and who had dysglycaemia, seven (29.2%) had been on treatment for the duration between 4 and 24 weeks, four (16.7%) were on treatment between 24 and 48 weeks, and 13 (54.2%) patients for more than 48 weeks. This difference did not attain statistical significance (p=0.341).

DISCUSSION

This study, conducted between January and April 2006 at the Kenyatta National Hospital, a national referral and teaching institution in Kenya, comprised 58% female patients, majority of whom were aged below 40 years. This is in keeping with the national estimates of higher HIV prevalence in young adults, and particularly in females (18). The mean weight of patients on HAART was significantly higher, a probable reflection of the documented improvement in morbidity and mortality occasioned by antiretroviral treatment (1,2).

Risk factors for cardiovascular disease in the study population remained low and were similar between the two patients groups. Current cigarette smoking stood at 9%, hypertension at 14.2%, and family history of coronary artery disease at 2.7%.

Majority (65.1%) of the HIV infected individuals enrolled in the study had been diagnosed within the last 12 months. Increasing awareness of HIV/AIDS, VCT centres, reduction in stigma and widespread availability and accessibility of antiretroviral therapy may explain the higher number of recently diagnosed individuals in our study.

Majority of the patients (82.2%) on HAART had been on regimens containing nucleoside reverse transcripts inhibitors (NRTI), mainly stavudine and lamivudine along with a non-nucleoside reverse transcriptase inhibitor (NRTI) backbone of either efaviren or nevirapine, as recommended by the national guidelines on initiation of antiretroviral therapy (19).

The overall prevalence of dyslipidemia in our HIV study population was found to be 63.1%. In comparison, two local studies in HIV negative hypertensive patients, one performed more than 10 years ago and the other more recent, report a dyslipidemia prevalence of 28.3% and 69.9%, respectively (20,21). The lower prevalence in the older patients may be a reflection of the use of cut-off value much higher than the current NCEP, ATP III guidelines, as utilised in our study. The latter study, although reporting similar prevalence to ours, was done on a highly select population of patients with other CVD risk factors, whereas our patients were fairly young with few other CVD risk factors. Viewed in this perspective, therefore, the prevalence in our study is disproportionately high.

We report a prevalence of elevated total and LDL cholesterol of 39% and 40%, and that these dyslipidemias were four fold more likely in patients receiving HAART than in patients who were HAART naive. This is consistent with findings in other studies (9,22): Thiebaut, *et al* (22) reporting on the Aquitaine cohort of HIV infected patients receiving HAART showed a prevalence of high total cholesterol of 32.7% (TC >5.5mmol/l); and in the DAD study,(9) high total cholesterol, of $\geq$ 6.2mmol/l, was found in 22% of individuals on treatment with NNRTI-containing regimens. In our study we found a slightly lower prevalence of 16.9% (at the same cut-off value of $\geq$6.2mmol/1), but still much higher than that of 2.5% found in HAART naive patients. When we included borderline high cholesterol ($\geq$5.17mmol/l) the prevalence reached 39.2%. Furthermore the median levels of LDL cholesterol were found to be higher by 0.7mmol/l in patients receiving HAART (data not shown). It has been estimated from epidemiological studies that cardiovascular risk increases twofold per every 1mmol/l elevation in LDL cholesterol; the additional risk conferred to patients on HAART by elevated LDL in our study therefore translates approximately to 1.5 fold increase in risk, relative to naïve patients.

Prevalence of high triglycerides in this study was similar in both groups of patients; this contrasts with findings in other studies (9,22) which observed high TGs particularly in patients receiving protease inhibitor-based therapy. Although absence of protease inhibitors in our study may account for this finding, this explanation can only be partial as studies done before the era of HAART indicated that triglycerides and free fatty acids are high in HIV infected patients who are not on ARVs (5). The increase in triglycerides is thought to be mediated by high circulating levels of cytokines, particularly interferon (IFN)-α, found in HIV infection which reduce the activity of lipases and slow the clearance of triglycerides.

Thus, the most common types of dyslipidemia found in association with HAART were raised total cholesterol and LDL cholesterol. Majority of the patients had borderline elevation; 13% of patients on HAART had high and very high levels of LDL cholesterol. This lipid profile is atherogenic, and it offers sufficient grounds for concern, particularly because it is primarily affecting a rapidly increasing population of fairly young individuals whose

treatment with HAART is life long, and thus their continued exposure to the risk of cardiovascular disease. Already there is accumulating evidence that this dyslipidemia may translate to cardiovascular disease. Friis *et al* (9) showed a 26% annual relative risk increase in the incidence of myocardial infarction in patients on treatment with HAART (14). Subsequent studies also point to an increase in other cardiovascular and cerebrovascular events other than myocardial infarction in this population of patients (15).

In our study correlation of dyslipidemia with the type of HAART was hampered by the fact that majority of the patients (>82%) were on only two regimens: stavudine, lamivudine with either nevirapine or efavirenz. Nevertheless, in patients receiving these two regimens, treatment with efavirenz showed increase in prevalence of high LDL cholesterol and triglycerides. This study did not replicate findings in other studies that indicated that nevirapine administered with two NRTIs raised HDL cholesterol (23,24). Taking into consideration that regimens containing efavirenz are part of the first-line antiretroviral drugs, a considerable proportion of patients receiving HAART may therefore already be exposed to therapy causing lipid abnormalities. It is therefore advisable that during subsequent revision of first line antiretroviral therapy, consideration be given to drugs causing less dyslipidemia

Duration of HAART therapy positively impacted on the the prevelance of dyslipidemia. The prevalence of high LDL cholesterol significantly increased with longer duration of therapy, with levels peaking at the period between 24 and 48 weeks and subsequently leveling out. This trend was observed also in total cholesterol and triglycerides although it did not reach statistical significance. Data from another study (24) showed increase in total and LDL cholesterol by week six of treatment peaking at week 24 and stabilising thereafter. This finding suggests that dyslipidemia develops with cumulative duration of exposure of HAART.

The overall prevalence of dysglycaemia in the study population was 20.7% and the rates were similar in both HAART and HAART naive patients (17.9% against 22%). Diabetes was found in only 1.5%, consistent with findings in other studies (9,25) while impaired fasting glucose was found in 1.4%. Meaningful associations between HAART regimens and duration of treatment could not be drawn because the number of patients was too small. The majority of the patients with dysglycaemia had impaired glucose tolerance, this is an important finding as it reinforces the utility of performing glucose tolerance tests whenever possible in order to detect dysglycaemia. It is well established that impaired glucose tolerance, apart trom predicting occurrence of type 2 diabetes, is also a risk factor for CVD (26,27).

A minor limitation in this study was that we could not verify conclusively that patients were adherent to HAART therapy. However, noting that the drop-out rate was less than 10%, it is apparent that these were highly motivated patients and the likelihood of non-adherence is deemed as low. We also used the same cut-off value of low HDL for both male and female patients which could have led to an underestimation of low HDL in female patients.

In conclusion therefore, use of HAART was associated with a lipid profile that is likely to increase the risk of cardiovascular disease, however it was not associated with a higher prevalence of dysglycaemia. We recommend that lipid profiles should be performed at baseline prior to initiation of antiretroviral therapy and again after six months to monitor the rising trends. It is also necessary that patients should undergo global cardiovascular risk stratification in order to institute timely and appropriate primary intervention measures .

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